OPHTHALMOLOGICAL TRANSACTIONS.

VOL. III.

TRANSACTIONS

OF THE

OPHTHALMOLOGICAL SOCIETY

OF THE

UNITED KINGDOM.

VOL. III.

SESSION 1882-83.

WITH

LIST OF OFFICERS, MEMBERS, ETC.

LONDON:
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1883.

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NOTICE.

THE present volume comprises the proceedings of the Ophthalmological Society of the United Kingdom, during its third Session, October, 1882, to July, 1883.

The Society does not hold itself responsible for the statements, reasonings, or opinions expressed in the communications which the Council has deemed suitable for publication.

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OFFICERS AND COUNCIL

OF THE

Ophthalmological Society of the United Kingdom,

ELECTED AT

THE ANNUAL GENERAL MEETING, JULY 6TH, 1883.

President.

JONATHAN HUTCHINSON, F.R.S.

Vice-Presidents.

WILLIAM BOWMAN, F.R.S.
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W. SPENCER WATSON

Secretaries.

JOHN ABERCROMBIE, M.D.
W. A. BRAILEY, M.D.

*** *Members are requested to communicate with the Secretaries when corrections are necessary.*

LIST OF MEMBERS OF THE SOCIETY.

Honorary Members.

Professor Donders, Utrecht.
Professor Helmholtz, Berlin.
Professor Stokes, Cambridge.
E. Williams, M D., Cincinnati, United States.

EXPLANATION OF ABBREVIATIONS.

O.M.—Original Member.
Pres.—President.
T.—Treasurer.
V.-P.—Vice-President.
S.—Secretary.
C.—Member of Council.
†.—Denotes Non-Resident Life Members who have paid the Composition Fee.

GENERAL LIST OF MEMBERS.

ELECTED

O.M. Abercrombie, John, M.D. (S.), Assistant Physician to the Charing Cross Hospital; 39, Welbeck Street, W.

O.M. Adams, James E., Surgeon and Ophthalmic Surgeon to, and Lecturer on Surgery at, the London Hospital; Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 17, Finsbury Circus, E.C. (C. 1880-3.)

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

O.M. Andrew, Edwyn, M.D. (C.), Surgeon to the Shropshire Eye and Ear Hospital; Hardwick House, St. John's Hill, Shrewsbury.

ELECTED

1883 Andrews, A. G., London Hospital, Whitechapel Road, E.

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., Assistant Physician to, and Assistant Teacher of Clinical Medicine at, University College Hospital; 10, Montague Street, Russell Square, W.C. (C. 1880-81.)

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

O.M. Baxter, E. Buchanan, M.D., Assistant Physician to King's College Hospital; Professor of Materia Medica and Therapeutics, King's College; 28, Weymouth Street, Portland Place, W.

O.M. Beevor, C. E., M.B., Assistant Physician to the National Hospital for the Paralysed and Epileptic; 129, Harley Street, W.

O.M. Benson, A. H., M.B., Lecturer on Ophthalmic and Aural Surgery, Ledwich School of Medicine, Dublin; 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., 2, Mount Street, Rodney Street, Liverpool.

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

O.M. Bowman, W., 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.)

ELECTED

O.M. Brailey, W. A., M.D. (S.), Ophthalmic Assistant Surgeon to Guy's Hospital; Ophthalmic Surgeon to the Evelina Hospital for Children; 16, Orchard Street, Portman Square, W. (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.

O.M. Bronner, Edward, M.D., Surgeon to the Bradford Eye and Ear Hospital; 33, Manor Row, Bradford.

1882 Brown, George A., Tredegar, Monmouthshire.

O.M. Browne, Edgar A., Surgeon to the Liverpool Eye and Ear Infirmary; 86, Bedford Street, Abercromby Square, Liverpool.

1882 Browne, John Walton, M.D., 10, College Square North, Belfast.

O.M. Bruce, S. N., 43, Kensington Gardens Square, W.

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

1883 †Buller, Frank, M.D., 1351, St. Catherine 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.)

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.

ELECTED

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 Hospital; 63, Grosvenor Street, W.

1881 Coles, Donald A., M.D

O.M. Cooper, William White, Consulting Ophthalmic Surgeon to St. Mary's Hospital; 19, Berkeley Square, 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., Physician to, and Lecturer on Pathological Anatomy at, the Middlesex Hospital; 14, Weymouth Street, Portland Place, W.

O.M. Cowell, George (C.), 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.

O.M. Critchett, G. Anderson (C.), Ophthalmic Surgeon to St. Mary's Hospital; 21, Harley Street, W.

1881 Cross, F. R., Honorary Ophthalmic Surgeon to the Bristol Dispensary; Surgeon to the Bristol Royal Infirmary; 5, The Mall, Clifton, Bristol.

O.M. Davidson, A. Deas, 11, Somerset Place, Swansea.

O.M. Davidson, Alex. Dyce, Lecturer on Ophthalmic Surgery, University of Aberdeen; Ophthalmic Surgeon to the Royal Infirmary, Aberdeen; 224, Union Street, Aberdeen.

1882 Deane, Andrew, M.D., Bengal Army, Naini Tal, N.W.P. India.

O.M. Denby, S. C., Assistant Surgeon to the Bradford Eye and Ear Hospital; 1, Camden Terrace, Bradford, Yorkshire.

O.M. Dent, Clinton Thomas, Assistant Surgeon to St. George's Hospital; 19, Savile Row, W.

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

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

ELECTED

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

O.M. Eales, Henry, Surgeon to the Birmingham and Midland Eye Hospital; 40, Newhall Street, Birmingham.

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

1883 Emrys-Jones, A., M.D., 10, St. John Street, Manchester.

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., Dunedin, New Zealand.

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

O.M. Fitz-Gerald, W. A, M.D., 9, Ely Place, Dublin.

1882 Fox, Arthur E. W., M.B., 16, Gay Street, Bath.

O.M. Frost, W. A., Assistant Ophthalmic Surgeon to St. George's Hospital; 77, Wimpole Street, W.

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

1883 Gibbons, R. A., M.D., 32, Cadogan Place, S.W.

O.M. Glascott, C. E., M.D., Surgeon to the Manchester Royal Eye Hospital; 11, St. John's Street, Manchester.

1882 Gosse, Charles, M.D., Adelaide, South Australia.

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.)

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

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

ELECTED

1881 Gulliver, George, M.B., Assistant Physician to St. Thomas's Hospital, and to the London Fever Hospital; 75, Lambeth Palace Road, S.E.

O.M. Gunn, R. Marcus (C.), Assistant Surgeon to the Royal London Ophthalmic Hospital, Moorfields, Ophthalmic Surgeon to the Hospital for Sick Children, Great Ormond Street; 108, Park Street, Grosvenor Square, W.

1882 Hartridge, Gustavus, 47, Kensington Park Gardens, W.

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

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

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

O.M. Horrocks, Peter, M.D., Assistant Obstetric Physician to, and Demonstrator of Practical Obstetrics at, Guy's Hospital; 9, St. Thomas's Street, S.E.

1882 Hudson, R. S., M.D., Redruth, Cornwall.

O.M. Hulke, J. W., F.R.S., 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. (*Pres.*), Consulting Surgeon to the London Hospital, and to the Royal London Ophthalmic Hospital; 15, Cavendish Square, W. (V.-P. 1880-1.)

O.M. Irwin, H. R., Surgeon to the Darlington Eye and Ear Hospital; Coniscliffe Road, Darlington.

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

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

1883 Jessop, W. H. H., Ophthalmic Surgeon to the North-West London Hospital; 70, Harley Street, W.

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

1882 Johnson, G. L., M.B., Fern Lea, Highfield Hill, Upper Norwood, S.E.

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

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

O.M. Juler, H. E., 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 King, Louis, Rivers House, Bath.

1881 Knaggs, S. T., M.D., Newcastle, New South Wales.

O.M. Lang, William, Ophthalmic Surgeon to the Middlesex Hospital; 26, Upper Wimpole Street, W.

1881 Langdon, J. Winkley, Winkley Square, Preston.

O.M. Lawford, J. B., M.D., Royal London Ophthalmic Hospital, Moorfields, E.C.

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

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

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

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

ELECTED

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

O.M. Mackenzie, F. M., 10, Hans Place, S.W.

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

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

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

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

1883 Maher, W. O., M.D., Royal London Ophthalmic Hospital, Moorfields, E.C.

1883 Mahomed, F. A., M.D., Assistant Physician to Guy's Hospital; 12, St. Thomas's Street, S.E.

1883 Marlow, Frank William, St. Thomas's Hospital, S.E.

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

O.M. McHardy, M. M., Ophthalmic Surgeon to King's College Hospital; Professor of Ophthalmology, King's College; 5, Savile Row, W.

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

1881 Milles, W. Jennings, Curator and Librarian to the Royal London Ophthalmic Hospital, Moorfields, and Ophthalmic Surgeon to the Great Northern Hospital; 45, Devonshire Street, Portland Place, W.

O.M. Morton, A. Stanford, Senior Assistant Surgeon to the Royal South London Ophthalmic Hospital; 57, Welbeck Street, W.

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

1881 Myers, A. B. R., Surgeon to the Coldstream Guards, Vincent Square, S.W.

ELECTED

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

O.M. Nettleship, Edward (C.), 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.)

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

1881 Oram, A. M., M.D., Sydney, Australia.

O.M. Ord, W. M., M.D., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; 7, 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.

O.M. Penfold, Henry, Senior Surgeon to the Sussex and Brighton Infirmary for Diseases of the Eye; 7, Brunswick Place, Brighton.

O.M. Power, Henry (V.-P.), 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. (C. 1880-2.)

1882 Prichard Arthur William, 31, Victoria Place, Clifton.

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

1882 Pringle, J. J., M.B., Medical Registrar to the Middlesex Hospital; 35, Bruton Street, W.

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.

ELECTED

O.M. Pye, Walter, Surgeon to, and Lecturer on Physiology at, St. Mary's Hospital; 4, Sackville Street, Piccadilly, 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.

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

O.M Rockliffe, W. C., 9, Charlotte Street, Hull.

O.M. Rogers, G. H., 3, Clifford Street, W.

1882 Roth, Reuter E., 61, Botany Street, Sydney, New South Wales.

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

O.M. Ryerson, G. S., M.D., Lecturer on Ophthalmic and Aural Surgery at Trinity Medical School, and Ophthalmic and Aural Surgeon to the General and Sick Children's Hospitals, Toronto; 317, Church Street, Toronto.

1881 Ryley, James, M.B., 91, Regent Road, Great Yarmouth.

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

1881 Sansom, A. E., M.D., Assistant 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.

O.M. Sharkey, S. J., M.D., Assistant Physician to, and Joint Lecturer on Physiology and Demonstrator of Morbid Anatomy at, St. Thomas's Hospital; 77, Lambeth Palace Road, S.E.

ELECTED

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

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; Glen View, 304, Western Bank, Sheffield.

O.M. Solomon, J. Vose, Surgeon to the Birmingham Eye Hospital; Professor of Ophthalmic Surgery, Queen's College, Birmingham; 22, Newhall Street, Birmingham. (C. 1880-3.)

O.M. Square, W., Surgeon to the Plymouth Royal Eye Infirmary; 4, Portland Square, Plymouth.

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

O.M. Streatfeild, J. F. (T.), Senior Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Ophthalmic Surgeon to University College Hospital; and Professor of Clinical Ophthalmic Surgery at University College, London; 15, Upper Brook Street, W.

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

1883 Sutton, S. W., M.B., St. Thomas's Hospital, S.E.

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., Royal London Ophthalmic Hospital, Moorfields, E.C.

O.M. Sympson, Thomas, Surgeon to the Lincoln County Hospital; 2 and 3, James Street, Lincoln.

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, 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. If any candidate, who is legally qualified to practise in India or the Colonies, be not personally acquainted with three members of the Society, the signatures, from personal knowledge, of teachers in the Medical School at which he was educated shall be accepted instead of the signatures of the same number of members of the Society.

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 four 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.

8. *Subscriptions.*—The Annual Subscription shall be One Guinea, payable in advance at the date of the Annual General Meeting. Each member on election shall pay an Entrance Fee of One Guinea in addition to the Subscription, but in the case of a member elected at a meeting of the Session subsequent to Easter he shall not be required to pay a Subscription during the next Session. Any member whose Subscription is six months in arrear shall be reminded of the same by one of the Secretaries, and if it be not paid within the current year he shall cease to be a member. Any member may, at any time, pay a Composition Fee of Fifteen Guineas and be thereby exempted from paying any further Subscriptions, such member enjoying all the same rights and privileges as if he were a Subscribing member. Any member resident out of the United Kingdom may pay a Composition Fee of Five Guineas instead of the Annual Subscription, and will then be entitled to receive, post free, a copy of the Society's 'Transactions' each year, and to have his name printed in the list of members; but if at any time he subsequently become a Resident member of the Society, the question of further payment by him shall be decided by the Council. N.B.—The Composition Fee in either instance will include the Entrance Fee.

9. The Officers of the Society shall be elected yearly by Ballot at the Annual Meeting, to which all the Ordinary members shall be summoned one week before. No gentleman shall hold the same office for more than three consecutive years. Balloting lists of the names recommended by the Council for election shall be sent to each Ordinary member, together with the notice of the Annual Meeting.

10. *The Secretaries* shall receive the lists during the first hour of the Annual Meeting. At the end of the hour they shall be delivered by the Chairman to two Scrutineers who shall report the result. In the event of equality of suffrage the Chairman shall determine.

11. *The President and Vice-Presidents.*—The President shall regulate all the proceedings of the Society and Council, state and put questions, interpret the application of the Laws, and decide any doubtful points. He shall check irregularities and enforce the observance of the Laws. He shall sign the minutes of General and Council Meetings. In the absence of the President one of the Vice-Presidents, the Treasurer, or some other member chosen by the Meeting, shall perform his duties.

12. *The Secretaries* shall manage all correspondence, shall attend every meeting of the Society and Council, and take minutes, which shall be read at the following meeting. They shall notify to new Members their election. They shall arrange with the President the order of proceedings at all the meetings. They shall have charge of, and keep a register of, all papers communicated, and shall be the Editors of the 'Transactions.'

13. *The Treasurer* shall receive all moneys due to the Society, and make all payments ordered by the Council, keeping an account of all such receipts and payments. He shall keep a printed receipt book for the subscriptions, and every receipt shall be signed by himself and countersigned by one of the Secretaries. He shall present to the Annual Meeting a written Report of the financial state of the Society, signed by himself and by two members of the Audit Committee.

14. *Audit Committee.*—The President, one of the Secretaries, and two Members of the Society nominated by the President at some meeting of the Society previous to the Annual Meeting, shall form a Committee to audit the Treasurer's accounts.

15. The Council shall meet half an hour before every meeting, and at such other times as they may be specially convened. Three shall form a quorum. The Council shall determine questions by show of hands (or by Ballot if demanded), the President having in both cases a casting vote in addition to his ordinary vote. They shall have the power of filling up any vacancies which may occur in any of the offices of the Society between one Annual Meeting and another. They shall decide upon all questions relating to the reception of communications and to their publication in the Society's 'Transactions.'

16. '*Transactions.*'—A copy of the 'Transactions' shall be sent to each Member of the Society.

17. The Ordinary Meetings shall be held from 8.30 to 10 p.m. on the second Thursday in October, December, January, March, and May, and on the first Thursday in June, and the Annual General Meeting on the Friday after the first Thursday in July.

18. *Visitors.*—Each Member may introduce two visitors on writing their names in the attendance book.

19. *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.

20. 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.

21. 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.

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

23. 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.

24. 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.

LIST OF COMMUNICATIONS

BROUGHT BEFORE THE SOCIETY DURING THE SESSION 1882-3.

I. DISEASES OF EYELIDS AND CONJUNCTIVA.

II. DISEASES AND TUMOURS IN THE ORBIT.

III. INTRA-OCULAR TUMOURS.

IV. PANOPHTHALMITIS.

V. SYMPATHETIC OPHTHALMITIS.

VI. DISEASES OF CRYSTALLINE LENS AND CAPSULE.

VII. DISEASES OF RETINA AND CHOROID.

VIII. DISEASES OF OPTIC NERVE.

IX. FUNCTIONAL AFFECTIONS.

DISCUSSION ON EYE SYMPTOMS IN DISEASES OF THE SPINAL CORD.

X. AFFECTIONS OF MUSCULAR AND NERVOUS SYSTEMS.

XI. NEW INSTRUMENTS.

LIST OF PLATES.

LIST OF WOODCUTS.

DESCRIPTION OF PLATE I.

Illustrating Messrs. Critchett and Juler's case of Chronic Membranous Conjunctivitis (p. 1).

FIG. 1 shows the appearance of the left eye about five months after the commencement of the disease. The lower lid is well everted and drawn down in order to show the thick deposit on its inner surface, which is separated from that on the globe by the red line of thickened tissue of the lower *cul de sac*.

FIG. 2 illustrates the condition of the right eye of the same case, taken ten days after the onset of the affection.

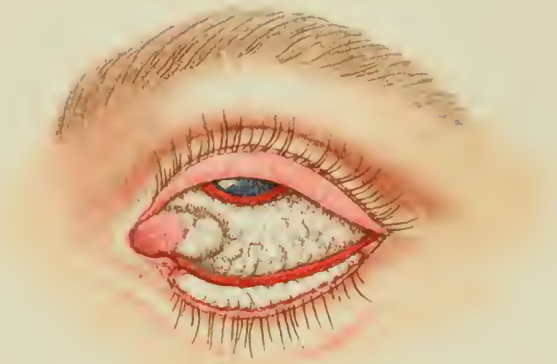

Fig 1

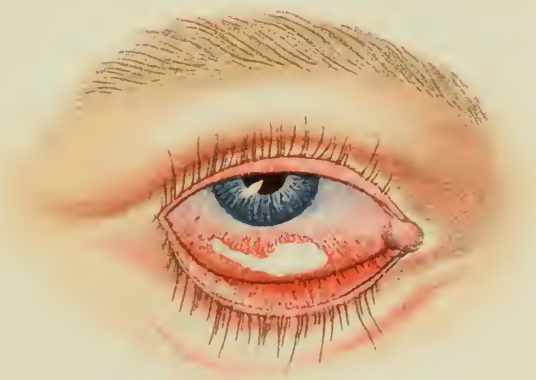

Fig 2

Lebon & Co

REPORTS.

I. DISEASES OF EYELIDS AND CONJUNCTIVA.

1. *Case of chronic membranous conjunctivitis.*

By Anderson Critchett and Henry Juler.

(With Plate I.)

Agnes H—, æt. 17, healthy-looking. In January and February, 1882, she was in-patient at the Lock Hospital under the care of Mr. Shillitoe, when she had numerous suppurating (soft) sores over the vulva and anus. There was no constitutional syphilis. No mercury was given. She was discharged cured. In May 1882, whilst under the care of Messrs. Bishop and Whitcombe at the Lock Asylum, she had an attack of simple conjunctivitis, which was treated from the first with lotions of alum and zinc; this was chiefly ocular, and had almost disappeared when a diphtheritic-looking patch was noticed occupying the lower cul-de-sac and the lower part of the globe of the left eye. This growth steadily increased, in spite of all treatment until she was shown to the Society on October 12th 1882. At that time (see Plate I, Fig. 1) all the lower part of the ocular conjunctiva, and all the inside of the lower lid were occupied by a thick, white, membranous

deposit, which was constantly being cast off in shreds. The white substance could not be completely peeled off without great difficulty and some bleeding; under the microscope it presented nothing but amorphous matter, no pus, no bacilli. The conjunctiva beneath and immediately around the deposit was thickened, and highly vascular. Cornea clear; no great pain. Various mild local remedies, and plain water dressing, had been duly tried in rotation before she was exhibited to the Society. Shortly after this a portion of the affected conjunctiva was excised. Microscopic sections were made of this, but presented nothing beyond dense infiltration of the tissue with leucocytes. Strong nitrate of silver was applied to the cut surface, and to the surrounding affected parts. This was followed by slight improvement, but the false membrane reappeared within ten days.

Sodium ethylate and other remedies were afterwards applied, but in vain; the white substance continued to appear, and to be cast off day by day.

During October the other (right) eye became similarly but less severely affected. Its appearance ten days after the onset is represented in Plate I, fig. 2. Only the lower part of the ocular conjunctiva was attacked; this was intensely red, and presented an irregular, transverse patch of tough, white, diphtheritic looking membrane midway between the cornea and the lower *cul-de-sac*. It was treated by the daily application of the crayon of lapis divinus, and was cured in a few weeks.

With the right eye cured, and the left eye unimproved, she was sent from St. Mary's to the Moorfields Hospital, where Mr. Tweedy undertook the case, and has kindly supplied us with the following notes.

"Jan. 26th, 1883.—The deposit matter was removed by scraping and washing with ether, leaving a red, rough, raw surface of thickened conjunctiva. A Buller's shield was applied.

"27th.—Deposit re-forming.

"28th.—The Buller's shield was seen to have the same

white deposit on its *outer* surface, and the glass was separated from the mackintosh part of the shield.

"30th.—The patient was suspected of applying the deposit to the lid (malingering), and for the next week the deposit was daily stained with strong aniline dye, which showed that the deposit was not applied from the outside but renewed from the conjunctival surface. Lotion of iodide of potassium (five grains to the ounce) was constantly applied.

"Feb. 14th.—The deposit matter separated, the conjunctiva was red, rough, and swollen; there was symblepharon. Carbolic oil applied instead of the iodide.

"March 3rd.—Discharged practically cured.

"April 4th.—Readmitted with the lid granular, traces of the deposit, and symblepharon. The deposit varies almost daily, but is always very small in amount. It appears to be best kept down by boracic acid powder.

"June 18th.—Discharged with a slight trace of deposit upon the symblepharon."

July 2nd, 1883.—We have again seen the patient. The girl is in excellent health, she is not hysterical, nor have we personally been able to detect any malingering. The right eye has continued quite well since it was cured in October last. The left eye is still affected with the same kind of deposit, and is far from well. The conjunctiva of the lower lid is red and rough, but is free from infiltration; there is slight symblepharon; the ocular conjunctiva just below the cornea is occupied by a circular patch of creamy-looking deposit, with thick reddened edges.

The extremely chronic nature of the affection of the left eye in this case exceeds everything we have previously experienced.

In an interesting paper entitled "Diphtheritic Ophthalmia," by Mr. Nettleship, published in 'St. Thomas's Hospital Reports,' vol. x, 1880, several cases of a chronic membranous nature, some of them lasting as long as our own case, are mentioned, and the following authors who have recorded others of a similar nature, are there quoted.

1. Mason, in 'Oph. Hosp. Reports,' vii, 164, 1871. 2. Hulme, 'Med. Times,' 1863, ii, 452. 3. Businelli, 'Gaz. Med p. le prov. Venete,' 1872, No. 16.

(*Living specimen. October 12th and December 14th*, 1882.)

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2. *Chancre on the mucous membrane of the upper lid with marked adenopathy, of three weeks' duration.*

By James E. Adams.

The sore appears on the inner surface of the upper lid as a small projection with a flattened surface equal in area to the size of a threepenny piece, resembling the appearance of a suppurating chalazion, but puncture proved the absence of pus.

No history of contagion could be obtained and a further period must elapse before its true nature can be decided.

(*Living specimen. December 14th*, 1882.)

P.S.—This patient has been lost sight of. The character of the induration, however, and the glandular swelling were such as to leave little doubt about the specific origin.

A case of hard chancre at the inner canthus.

By Simeon Snell (Sheffield).

Clara G—, æt. 21, a nurse, came to the Sheffield General Infirmary on August 15th, 1882, in consequence of a sore at the inner corner of the left eye. For thirteen months she had lived as nurse in a family. The youngest

child, a baby of five months, had had, and then had, a rash on its body, also thrush which "had gone through it," and a sore nose and snuffles. The patient was in the habit of sleeping with this child and kissing and fondling it a great deal. It is thought that there had been miscarriages before the birth of this baby, and there was evidence of indifferent health in the mother.

The sore was situated at the left inner canthus, involving the integument at the commissure and both eyelids as far as the puncta, the lower being more affected than the upper. The ulceration affected also the caruncle and the conjunctiva in its immediate neighbourhood. There was a good deal of chemosis and swelling of the lids. The sore was about the size of a shilling, the surface greyish and covered with a scanty, hardly purulent, secretion; the edges well marked, raised, and surrounded by much induration, though not cartilaginous. The gland in front of the ear and the submaxillary glands were indurated.

The patient was uncertain as to when she had first noticed anything wrong with the eye. The sore was quite small when observed (a pimple ?) and she fancied that it must have begun six or seven weeks previously. On her face and chest there was a scanty, coppery, papular rash; the throat had been sore for a week or two, and there was slight ulceration of the left tonsil; the hair was falling out. She complained of malaise.

The appearance and characters of the sore, and the induration of præ-auricular and submaxillary glands, not often, I believe, met with in non-specific ocular ulcerations, at once suggested the nature of the sore in this case to be a Hunterian chancre. The child under her care was from the history most probably capable of acting as a source of infection and careful inquiry disposes, I think, of any other means of infection. I was, however, not allowed to see the child. The patient was quite unable to fix any definite period at which a scratch or other injury was inflicted.

On August 15th she was put upon perchloride of

mercury, and a lotion of boracic acid, 5 grains to the ounce, was ordered for local application.

29th.—The sore is in about the same condition, but the enlargement of præ-auricular and submaxillary glands has increased, giving a very full appearance to the left side of the face, and interfering with the movements of the jaw.

Blue pill (4 grains) night and morning, was substituted for the perchloride, and a lotion of borax and glycerine prescribed in place of the boracic acid.

Sept. 1st.—Chancre is noted as improved.

12th.—Healing fast.

22nd.—Sore nearly healed. Both tonsils ulcerated, especially left. Chlorate of potash gargle prescribed; to repeat the mercurial pill and lotion.

25th.—One pill only to be taken daily.

Oct. 3rd.—Throat still ulcerated, though better; back of tongue is affected also; induration of glands has subsided a great deal.

13th.—Mucous tubercles on vulva; calomel ordered for dusting. The chancre is healed and disappearing and hardly any induration remains; all the glandular enlargements have gone.

At the time of writing (middle of November, 1882) although the situation of the chancre is readily recognised, it bids fair to leave the most trifling deformity. Indeed it is now chiefly noticeable from the remaining redness. The eyelids and puncta have subsided to their normal positions, so that the lachrymal apparatus is hardly likely to be permanently interfered with.

A point of interest and value from a diagnostic point of view was the induration of glands, a condition seldom existing with eyelid affections. Mr. Wherry alludes to this, in his case of chancre of conjunctiva recorded in the last volume of the Society's 'Transactions,' and it has received notice at the hands of other writers. As regards treatment, I would merely remark on the apparent benefit derived from the borax and glycerine lotion. Dr. G. Thin, in the 'Lancet' of May 27th, last, advocated its use

in chancres, and the theory on which the treatment was conceived was that of keeping the surface of the sore saturated with a solution that would effectually prevent the development of organisms.

(*December 14th*, 1882.)

II. DISEASES AND TUMOURS IN THE ORBIT.

A case of rapidly growing sarcoma of the orbit, in a child.

By Anderson Critchett and Henry Juler.

Emily E—, æt. 6 years. About the 17th of last November—less than one month ago—the mother noticed a slight prominence of the child's right eye. This increased so rapidly as to cause some anxiety, and she was brought to St. Mary's Hospital on November 24th. At that time there was distinct proptosis of the right eye, this being about 1½ centimetres in advance of the left, which was quite normal. There was no pain, or tenderness. The fundus oculi was normal in appearance and the vision good. The conjunctiva beneath the upper cul-de-sac was œdematous.

An exploratory incision was made by Mr. Anderson Critchett through the upper lid, the scalpel being plunged deeply into the orbit above the globe of the eye. The skin incision was sufficiently large to admit the end of the little finger which was passed into the orbit, but no solid tumour could be felt, nor was there any but a sero-sanguineous discharge. The wound was closed with silk sutures, and a light compress applied.

On December 1st—one week later—the wound in the eyelid had healed, but the proptosis had increased to at least 2 cm. Chemosis was also greater, being now abundant all around the globe. Exploratory punctures were now made by Mr. Juler with a hydrocele trocar and cannula, which was thrust deeply into the orbit at the upper, lower and outer sides of the globe. Only a slight discharge of blood took place.

The proptosis and chemosis have steadily progressed till the present time. There is now (Dec. 14) excessive bulging of the globe, and of the orbital contents to the extent of 3 to 4 cm. No pulsation or bruit can be detected. There is no pain, the child only complains of itching of the protruded conjunctiva, which is beginning to ulcerate. The skin of the lids and surrounding region is extremely tense and dusky.

The child has always been fairly healthy. There is no family history of syphilis.

(*Card specimen. December 14th,* 1882.)

P.S.—On Dec. 15 the globe was enucleated, and the orbit found to be completely filled by pale, pulpy substance somewhat resembling fatty tissue. This involved all the structures of the orbit with the exception of the globe itself, which was quite flaccid. Upon microscopic examination the new growth presented the characters of a rapidly growing, round-celled sarcoma. The whole of the contents of the orbit were as far as possible removed; chloride of zinc paste was then applied to the exposed surfaces. The disease, however, gradually extended to the surrounding parts. She was removed from the hospital on Jan. 3, 1883, and died shortly afterwards.

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2. *On a case of double pulsating exophthalmos, with observations.*

By W. Adams Frost.

Thomas L—, æt. 38, when ten years old, being then in good health, was run over by an empty timber waggon. One wheel passed over his left shoulder and the left side of his head. He immediately became unconscious, and remained so for two or three weeks, blood flowed in con-

siderable quantity from his nose, mouth, and one or both ears, and it is said that the left eye was forced out upon his cheek.

Ever since the accident there has been a pulsating swelling above the left eye, and he has heard a drumming noise in the head on the left side, at first constantly, but of late years only when lying down, and not always then. Until within the last few years the left eye was very prominent; now, it is not more so than normal, or than the right. Ever since the accident the left eye has been rotated inwards, but he does not remember ever to have had diplopia.

He has followed the craft of a wheelwright, and has suffered no serious inconvenience from the orbital affection; indeed, he came to me in December last, not as a patient, but in order to bring his boy.

His condition then was as follows:—a short, thick-set man, in good general health. His left eye was rotated inwards about 25°, and could be rotated outwards not quite to the middle line. Immediately beneath the *left* eyebrow, covered by the skin of the upper lid, was an oval swelling the size of a filbert, its long diameter transverse and its inner extremity situated 5 mm. from the inner canthus; further to the inner side was a smaller, more globular swelling, which was divided by a depression, corresponding to the tendo oculi, from a flatter and more diffuse swelling on the side of the nose and in the angle between the nose and the eye. In the skin of the upper eyelid were a few dilated and tortuous veins. The swellings were soft and compressible, and pulsated in a gently distensile manner; over the nasal portion a distinct vibratory thrill was perceptible. A systolic bruit, loud, but devoid of roughness, could be heard over the whole swelling, and was conducted in all directions with gradually decreasing distinctness.

In the *right* orbit beneath the junction of the middle with the inner third of the eyebrow, was a prominent globular swelling rather larger than a pea, it was soft and reducible, and pulsated feebly. A faint bruit was audible

over it, but from its low intensity this might have been conducted from the other orbit. The movements and position of this eye were normal. He was not aware, until it was pointed out to him, that there was anything wrong in this orbit. There was no undue prominence of either eye. Vision: right, $\frac{20}{70}$; left, $\frac{20}{50}$, $c + 1$ D $= \frac{20}{30}$; so that the sight of the displaced eye was practically unaffected. Ophthalmoscopic appearances normal in both eyes. The visual field of the right eye was slightly smaller than normal, that of the left constricted on the inner side. The common carotid arteries could be efficiently compressed only with difficulty, and the pressure was not well borne, in doing this I had the assistance of Mr. Juler. After several trials we came to the conclusion that pulsation could be stopped in both orbits by pressure on the left carotid. On one occasion we thought that by pressure on the right carotid the pulsation in the right orbit was controlled, but on every other occasion the pressure on this artery had no effect on the pulsation in either orbit.* For about ten days at the end of January and the beginning of February, he suffered from very severe pain of a throbbing character in the left forehead, and loud subjective noises in the left ear, which he compared to the beating of a tambourine. These symptoms subsided after rest, low diet, and five-minim doses of tincture of digitalis, but ever since there has been marked venous injection of the right conjunctiva. With these exceptions there has been no change in the condition since I first saw him.

I have called this case one of "Pulsating exophthalmos," although there is now no protrusion of either eyeball, for it obviously belongs to the group of cases of which pulsation and exophthalmos are the most prominent symptoms.

I suppose I may say, without fear of contradiction, that

* Mr. Adams and Mr. Higgens examined the patient as to this point on the evening on which he was exhibited, and confirmed our conclusion as regards the effect of compression of the left carotid, but thought that the pulsation in the right was controlled by pressure on the right carotid. The pulsation in the right orbit was very feeble, and could only be felt when the finger rested very lightly on the swelling.

all those who, in recent years, have paid any attention to the subject are agreed that the cause of the symptoms in the majority of these cases is the existence of a communication between the internal carotid artery and the cavernous sinus, and consequent distension of the orbital veins which form the pulsating swelling in the orbit. In this case there can, I think, be no doubt that such a communication was established by a fracture of the base of the skull crossing the artery.

This view of the pathology of the affection is, however, a comparatively recent one, and it is interesting very briefly to glance at the stages by which it has been reached.

Commencing then with Travers' case ([1]) in 1809, we may say that from that date to 1859 none of the authors who, in this country, recorded cases of the kind, seem to have suspected that the symptoms might be due to any other cause than to an aneurysmal affection within the orbit, although it was disputed whether this was a true aneurysm, an aneurysm by anastomosis, or an aneurysmal dilatation of the ophthalmic artery in its whole course. In favour of the first hypothesis was the fact that true aneurysms of the ophthalmic artery in the orbit had been found post-mortem in two instances. One of these, recorded by Carron du Villards ([2]), was not seen during life and nothing is known of the symptoms. The other case (Guthrie's) ([3]) is exceptional from the fact that no tumour was perceptible during life; an aneurysm, the size of a filbert, was found on each ophthalmic artery, and the orbital veins were much distended owing, it is said, to obstruction at their exit from the orbit, but the cavernous sinus was not examined ([4]).

The symptom which, more than any other, led to the diagnosis of aneurysm by anastomosis, was the extension of the tumour along the supra-orbital and angular vessels, but this is equally explicable on the supposition that the varicosity of the orbital veins extends to these veins. There can be no doubt, however, that some of the recorded cases were pulsating angiomata. Thus, in a case of Morton's ([5]) the orbital symptoms were noticed soon after

birth, and the varicose aneurysmal dilatation involved the whole side of the head, and the corresponding half of the tongue was hypertrophied. In a case of Warren's ([6]) the pulsation in the tumour could be controlled by pressure on the facial artery, and ceased on its division. In two other cases ([7]) the contents of the orbit were removed and in each instance the growth proved to be of this nature. Haynes Walton's case ([8]), from the fact that it occurred in a young child, is also generally considered to have been of this nature.

The only evidence in favour of aneurysmal dilatation of the ophthalmic artery having been the cause of the symptoms is, I believe, that afforded by one of Nunneley's ([9]) cases. The characteristic symptoms were present and the common carotid was tied. The patient died and the post-mortem was made hurriedly and in the presence of a relative; a ruptured aneurysm of the carotid was found in the cavernous sinus, and in the orbit a dilated and tortuous vessel which Nunneley believed to be the ophthalmic artery, but which there is every reason now to believe was the vein.

In 1859 a new light was thrown upon the pathology of this affection by the publication by Mr. Hulke ([10]) of a case which had been under the treatment of Mr. Bowman. This case is so well-known and has been so often quoted that I will only mention its leading features.

A woman, æt. 40, received a blow which fractured the lower margin of the orbit. Soon afterwards the characteristic symptoms of pulsating exophthalmos made their appearance. The common carotid was tied and the symptoms disappeared, but the patient died from the effects of secondary hæmorrhage on the eighteenth day. At the post-mortem no arterial lesion was found, but the orbital veins were dilated and the cavernous sinus and its tributaries filled with puriform clot.

It has often been suggested, that these changes were recent and consequent on the operation, and that the original lesion was a small fissure in the artery which was over-

looked, but the possibility of this being the case has been denied by Mr. Hulke* and in a case recorded by Aubry ([11]) no such explanation seems admissible. The symptoms came on after an attack of enteric fever, and persisted until the patient's death from an independent cause four years later. The cavernous sinus was found to end posteriorly in a *cul-de-sac* and no communication between it and the inferior petrosal sinus could be discovered. It was found that by injecting fluid into either carotid artery the pulsation and bruit which had been present during life could be closely imitated, and yet none of the fluid was found in the sinus. However we may explain the occurrence of pulsation in these two cases, there can be no doubt as to the effect that the first one has had in modifying the views of the pathology of pulsatile exophthalmos held in this country. This is shown by the following quotation from Mr. Nunneley's ([12]) paper read before The Royal Medical and Chirurgical Society in 1865.

"In the great majority of cases of protrusion of the eyeball there is no disease whatever in the orbit, the seat of it is most commonly intra-cranial, the protrusion of the eyeball is passive, and the other symptoms are secondary and depend on obstruction to the return of blood through the ophthalmic vein."

Here we have a distinct advance in the pathology but not the whole truth, and in 1870 the missing link was supplied by Delens ([13]) who in a most able monograph showed that, in addition to what may be termed the passive obstruction to the return of blood from the orbit through the veins, there is in most cases an arterial propulsion of blood *into* the veins, owing to the existence of a communication between the internal carotid artery and the cavernous sinus; a conclusion which the papers of Rivington ([14]) in 1875, Schlaefke in 1879, and Sattler ([15]) in 1880, have confirmed.

* *Vide* Mr. Holmes' Lecture on Aneurysm at the Royal College of Surgeons.

The idea was not really a new one; as long ago as 1835, M. Baron ([16]) had shown at the Société Anatomique a specimen of an aneurysm of the internal carotid which had ruptured in the cavernous sinus, and had asked "Is it not to this cause that we must attribute the varicose dilatation of the orbital veins, which had caused a considerable exophthalmos?" but the case seems to have excited little interest at the time, and it took thirty-five years to return an unhesitating answer that, not only in that case but in the great majority of others with similar symptoms, an arterio-venous communication was the essential lesion.

To examine critically all the evidence that can be adduced in support of this theory, would carry me far beyond the limits of this paper, and it has already been most ably done by the authors I have quoted. I shall merely refer briefly to what may be called the direct evidence afforded by post-mortem examinations.

These, as far as I can ascertain, are nineteen in number, and while they shew that the symptoms may be produced by a variety of causes, they also prove that an arterio-venous communication was present in a large proportion of cases, that its presence might be reasonably suspected in others, and could be disproved in very few.

In three cases an orbital tumour was found. In two of these, Nunneley's and Lenoir's ([16]) the tumour was malignant and had extended beyond the orbit. The third (Oettingen's) was a fibrous tumour associated with a meningocele ([17]).

In two cases, Bowman's ([10]) and Aubry's ([11]) already referred to, an affection of the cavernous sinus only was observed.

In two, Guthrie's ([3]) and Carron du Villards ([2]), a true aneurysm of the intra-orbital portion of the ophthalmic artery was found, in one of these the condition was present on both sides and the orbital veins were dilated, the other was not seen during life.

In the remaining twelve cases an arterio-venous communication certainly existed in seven, and almost certainly

in eight, while in the other four its presence was not disproved.

Those in which this lesion was certainly present are the following.

1. *Baron* ([16]) (already referred to) in which an aneurysm of the internal carotid ruptured in the sinus.

2. *Gendrin* ([18]) rupture of the carotid in the sinus.

3. *Nélaton* ([19]) wound of left carotid in the sinus by a rib of an umbrella thrust into the opposite orbit.

4. *Hirschfeld* ([20]) rupture of an atheromatous carotid in the sinus.

5. *Nunneley* ([9]) rupture of an aneurysm in the sinus.

6. *Nélaton* ([21]) fracture of the petrous portion of the temporal bone, the carotid in the sinus wounded by a splinter.

7. *Schlaefke* ([22]) wound of artery in the sinus by a pistol shot which had entered through the mouth.

In *Blessig's* case ([23]) there was a fracture of the base of the skull, crossing the petrous portion of the temporal bone, and the orbital vein was much dilated; unfortunately the carotid was too much damaged in the dissection for it to be ascertained whether there had been a fissure in its wall or not.

Four cases remain.

Nunneley ([24]).—The examination was not made till more than four years after the cure of the symptoms by ligature of the common carotid. A cured aneurysm of the ophthalmic artery at its origin was found, it lay partly in the sella turcica, and had compressed the ophthalmic vein. The internal carotid is said to have been healthy.*

Oettingen ([25]).—The symptoms came on suddenly in a debilitated woman, æt. 64, and disappeared under treatment by local pressure. A post-mortem examination was made two years later; the ophthalmic veins were found filled with coagulum. The carotid in the sinus was not specially examined.

Wecker ([26]).—The symptoms came on suddenly in a

* It is possible that in this case the communication was between the intracranial portion of the ophthalmic artery and vein.

woman, æt. 63; she died fifty-two hours after ligation of the carotid. The orbital veins were found greatly distended. The carotid was very atheromatous, but it does not appear to have been examined in the sinus.

Morton ([27]).—No pulsation had been present during life. The symptoms, which consisted in subjective noises in the head and protrusion of the globe, had come on after a severe blow upon the head. The patient died after ligature of the carotid. An incomplete examination was first made; twenty-four hours later Morton re-opened the head, he found no arterial lesion, but the orbital veins were dilated. The examination was made under difficulties, for the friends were present, and no part was allowed to be removed from the coffin in which the body lay.

In all the cases except those of orbital tumour, and Carron du Villards' case of aneurysm, the orbital veins were dilated, and in these where pulsation was present, the pulsating swelling was formed by the distended vein, except perhaps in Guthrie's case of aneurysm.

Deducting the cases of orbital tumour we may say that out of sixteen autopsies a communication between the internal carotid artery and the cavernous sinus existed in eight; that no such lesion was present in Aubry's, Carron du Villards', and perhaps in Bowman's case; that it may have been present in the remaining five, Guthrie ([3]), Nunneley ([24]), Oettingen ([25]), Wecker ([26]), and Morton ([27]).

Returning to my own case, the most interesting features in it seem to be the clear connection of the symptoms with the injury, the fact that both orbits are involved and the length of time—twenty-eight years—during which the symptoms have existed.

There can I think be no doubt that there was an extensive fracture of the base of the skull, and my theory is that this crossed the left internal carotid artery and so established a communication between it and the cavernous sinus, which led to varicose dilatation of the orbital veins, and that this has now extended by the transverse and circular sinuses to the venous channels on the opposite side.

A fracture of the base seems to have been a very frequent starting-point of the symptoms. Thus out of 110 cases ([15]), of which I have been able to find a record (and this includes the present case) 65 were of traumatic origin; of these, 21 presented well-marked symptoms of fracture of the base, and in 15 others lengthened periods of unconsciousness followed the accident. The other 29 traumatic cases were as follows:—

In 8 there was a wound, caused in 5 by sharp bodies thrust into the orbit ([19, 33-36]), in 2 by the entrance of shot ([22] and [37]), and in 1 ([38]) by a fragment of a burst soda-water bottle.

In the 21 remaining cases in which accidents of various kinds occurred, it is impossible to estimate the amount of violence inflicted.

The appearance of pulsation in the second orbit in my case gives strong confirmation to the theory that the lesion causing the symptoms is an arterio-venous communication. In an idiopathic case the affection of the second orbit is explicable on the hypothesis of an independent but similar lesion in the two orbits, as in Guthrie's case of double aneurysm; but if the one orbit is affected as the direct consequence of an injury and similar symptoms appear in the other orbit after an interval, it is only reasonable to conclude that there has been a direct extension from the one to the other; now the venous channels alone form a vascular connection between the orbits (for the anterior extremity of the circle of Willis is beyond the origin of the ophthalmic arteries and therefore out of the question) and these venous channels have been found dilated in nearly all the cases examined. So that we may conclude that the pulsating tumour in the orbit which was first affected—the left—is formed by the distended veins, and that the dilatation has extended by the circular and transverse sinuses to the right orbit. If I am right as to the effect of compression of the carotids, viz. that compression of the left carotid stops pulsation in both orbits whilst compression of the right controls neither, the only conclusion

possible is that there is a lesion of the left carotid causing first distension of the venous channels connected with the left orbit, and subsequently extending to those of the right; and what other lesion but an arterio-venous communication will fulfil all these conditions?

I can find only one analogous case—that of Velpeau ([28]). The patient was a man, aged thirty, in whom the symptoms came on after a blow on the nape of the neck. Six months after this the right eye was much protruded and its vision impaired, and there was a pulsating swelling above it in which a loud bruit was audible. The left eye was not protruded, but there was a similar swelling to that present in the other orbit. Pressure on the right carotid controlled pulsation in both orbits, pressure on the left affected the pulsation in the left orbit only. The right carotid was tied and the symptoms disappeared; a relapse, however, occurred in the right orbit, and it was now found that pulsation in this could be controlled by pressure on the left carotid.

There are six other cases of pulsating exophthalmos which I have found published, in which symptoms were present in both orbits, but with the exception of Guthrie's ([3]) already sufficiently referred to, and Maklakoff's ([33]) in which the details, in the only report I have seen, are very scanty, pulsation was present in the one orbit only (vide Nos. [29-32]).

I did not intend to adopt any active treatment, as the patient has for years suffered no inconvenience from the affection. A few weeks ago, however, there was a decided increase in the symptoms and some venous congestion of the right eye has persisted, and this has to some extent re-opened the question in my mind as to whether any active measures should be undertaken. Compression of the carotid would certainly not be endured, and the choice would therefore seem to lie between ligature and inducing coagulation by other means.

The injection of styptics would be fraught with some danger, although in the nasal portion it might perhaps be

done without much risk. Rest, low diet, and the internal administration of digitalis, would be free from risk and might prove effectual; they certainly influenced his symptoms when these were aggravated. If these measures failed, ligature of the carotid would have to be taken into consideration. The statistics of ligature for the cure of this affection are certainly very favourable ([39]). Out of sixty-three cases of ligature, of which I have been able to find a record, thirty-one were cured at once and, as far as is known, permanently; three were partially cured; in thirteen a relapse occurred, but a cure was subsequently effected by other means (in two by ligature of the other carotid). In one no effect was produced, and in Velpeau's case ([28]), in which both orbits were affected, a relapse occurred in one. Fourteen cases were fatal; seven from the effects of the operation; one after galvano-puncture performed for a relapse; the remainder from causes unconnected with the operation.

(1) *Travers*, 'Med.-Chir. Trans.,' ii, p. 6.

(2) *Carron du Villards*, 'Guide pract. pour l'Etude et le Traitm. des Mal. des Yeux.'

(3) *Guthrie*, 'Operative Surgery,' 1823, p. 158.

(4) For arguments in favour of intra-orbital aneurysm, see case and paper of *Busk*, 'Med.-Chir. Trans.,' xxii, p. 124.

(5) *Morton*, 'Amer. Journ. Med. Sc.,' lx, p. 44.

(6) *Warren*, 'Surg. Obs. on Tumours,' quoted by Sattler, 'Graefe Saemisch Handb.,' vi, xi.

(7) *Frothingham*, 'Amer. Journ. Med. Sc.,' lxxiii, p. 97; *Hansen*, mentioned by Sattler, in 'Graefe Saemisch Handb.,' vi, xi, p. 844.

(8) *Walton*, *Haynes*, 'Surg. Dis. of the Eye,' 3rd Ed., p. 318.

(9) *Nunneley*, 3rd case in 1st paper "Aneurism of or within the orbit," 'Med.-Chir. Trans.,' xlii.

(10) *Bowman* (Hulke), 'Ophth. Hosp. Reports,' 1859.

(11) *Aubry*, 'Gaz. des Hôpit.,' xliii, p. 171.

(12) *Nunneley* second paper, "Vascular Protrusion of Eyeball," 'Med.-Chir. Trans.,' xlviii.

(13) *Delens*, 'De la Communication de la Carot. Int. et du Sinus Cavern.,' Paris, 1870.

(14) *Rivington*, 'Med.-Chir. Trans.,' lviii.

(15) *Sattler*, "Pulsirender Exophthalmos." A list of 106 cases, which includes all the cases in Rivington's paper except Freer's, and all the cases

referred to in this communication except one by Martin, 'Journ. de Med. de Bordeaux,' 1881, one by Dr. Wolfe, 'Lancet,' 1881, ii, 946, and the author's case now published for the first time.

(16) *Baron*, quoted by Delens, op. cit.; *Nunneley*, 3rd case in 2nd paper, 'Med.-Chir. Trans.,' xlviii; *Lenoir*, 'Bull. de la Soc. de Chirurg.,' ii, 61, quoted by Sattler, loc. cit.

(17) *Oettingen*, 'St. Petersb. Med. Zeitsch.,' xii, 45, xxiv, 345, quoted by Sattler, loc. cit.

(18) *Gendrin*, quoted by Dermarquay, 'Tumeur de l'Orbite.'

(19) *Nélaton*, Delens, loc cit.

(20) *Hirschfeld*, 'Gaz. des Hôpit.,' 1859, p. 57.

(21) *Nélaton*, Delens, loc. cit.

(22) *Schlaefke*, 'A. f. O.,' xxv, iv, 112.

(23) *Blessig*, 'St. Petersb. med. Wochenschr.,' xxi, 269, quoted by Sattler, loc. cit.

(24) *Nunneley*, 4th case in 1st paper, post-mortem in 2nd paper, 'Med. Chir. Trans.,' xlii and xlviii.

(25) *Oettingen*, 'St. Petersb. med. Zeitschr.,' xi, p. 1, quoted by Sattler, loc. cit.

(26) *Wecker*, 'Annal. d'Oculist,' 1869, p. 186.

(27) *Morton*, 'Amer. Journ. Med. Sc.,' lxxi, p. 334.

(28) *Velpeau*, 'Bull. de Thérapeut.,' xvii, p. 128, and 'Leçons Orales,' iii, 437. There is a discrepancy in the two accounts as to the effect on compression of the carotids; *vide* also Sattler, loc. cit.

(29) *Grüning*, 'Knapp's Arch.,' 1876.

(30) *Halstead*, 'New York Med. Journal,' 1869, p. 665, and Sattler, loc. cit.

(31) *Harlan*, 'Amer. Journ. Med. Sc.,' lx, p. 45; *Galezowski*, 'Trait. des Mal. des Yeux,' and Sattler, loc. cit.

(32) *Maklakoff*, "Nagel Jahresber. ueber d. Liest. u. Forteschr," in 'Geb. des Oph.,' vi (1877), p. 444. Sattler, loc. cit.

(33) *Hart*, 'Lancet,' 1862, i, 272.

(34) *France*, 'Guy's Hosp. Rep.,' ser. iii, vol. i, p. 38.

(35) *Lawson*, 'Brit. Med. Journ.,' 1869, ii, 631.

(36) *Passavant*, quoted by Sattler, loc. cit., No. 46.

(37) *Holmes*, 'Amer. Journ. Med. Sc.,' 1864, p. 46.

(38) *Landsdown*, 'Brit. Med. Journ.,' 1875, i, 736, 771, and 846.

(39) These statistics are compiled from Sattler's List (15), but include Wolfe's case.

(*March 9th*, 1883.)

A case of hydatid cyst in the orbit.

By P. H. Mules, M.D. (Manchester).

This case occurred in a boy, æt. 6, the patient of Mr. Ring, of Keswick, by whom he was sent to me for examination.

The history of the case is as follows :—

In June, 1882, the boy's friends noticed that the right eye protruded, and sent him to Mr. Ring. Mr. Ring punctured the most prominent part of the swelling, no fluid escaped, and he sent the patient to me.

Four weeks after the appearance of the disease I saw the boy, the protrusion of the globe was remarkable and the movements of the eye impeded in every direction, the action of the external rectus being specially defective. His vision was bad with the affected eye, scarcely reaching 16 J ; the ophthalmoscope showed only engorged retinal veins. Behind the globe and rather to the outer side, was a firm, lobulated swelling which, considering the rapidity of its development and the absence of marked constitutional symptoms, suggested the diagnosis of a cyst. The boy was chloroformed, and after dividing the outer canthus I carefully cut down to the growth, between the superior and external recti muscles. A gush of clear fluid followed the incision, the eye receded and for a time all was well, the vision recovering to 1 J. and $\frac{6}{6}$; at the end of ten days the eye began again to protrude and two days later I found the disc choked and swelled to the extent of 1 mm. The lad was a second time chloroformed and the cyst exposed, but not opened. The external rectus was found closely adherent to the cyst, and being carefully dissected off, was saved from injury, and the cyst was traced to the apex of the orbit. I thought it wiser not to cut it away but to open it freely and introduce two drainage tubes. Suppuration with slight febrile symptoms followed, and after a week the cyst, measuring

$1\frac{3}{4}''$ in its long diameter and $1''$ in its shorter, was discharged; the wound rapidly healed up and with the exception of weakness of the external rectus, which necessitated slight tenotomy of its opponent, the eye resumed its normal appearance, and vision again returned to $\frac{6}{6}$ and 1 J., but has now fallen off considerably, and the disc has become very white. The cyst consists of from eighteen to twenty layers of structureless material and resembles in every way an hydatid. The first fluid was not caught but on examination of the second, which contained no albumen, we were unable to detect any hooklets.

The desirability of removing the cyst at the first operation would naturally strike those who have met with similar cases, but the extreme difficulty of doing so, without cutting the muscles through at their bony insertion, was apparent to those who assisted at the operation; and the subsequent spontaneous escape of the cyst gave a satisfactory result.

The diagnosis was founded on the microscopical examination of the wall only of the cyst, which was apparently barren. The structure as detailed above is typical of hydatid cysts and is observed in no other condition.

(*December 14th*, 1882.)

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4. *Necrosis and spontaneous separation of a large ivory exostosis of the orbit.*

By H. A. Lediard, M.D. (Carlisle).

I am indebted to Mr. H. R. Hughes, of Bangor, for permission to bring forward this rare specimen this evening, and also for the notes of the case which I now read.

"William O—, æt. 33, a sailor, was admitted into the Carnarvonshire and Anglesey Infirmary under the care of Mr. H. R. Hughes, on June 14th, 1870. The patient was informed by his parents that the tumour was about

the size of a pea at his birth, that it commenced to grow half way between the left upper eyelid and eyebrow, that it gradually increased in size, growing downwards and outwards, obstructing, by degrees, his sight. When about nine years of age the pressure upon the eyeball was so great that the globe gave way and its contents escaped; this was the only occasion on which he has suffered any pain whatsoever.

The tumour continued to enlarge up to his twenty-fifth year when growth ceased, and two years later the skin which had hitherto covered it suddenly gave way, some falling in strips, the remainder receding towards the left side where it, together with a portion of the lower eyelid, remains, forming an hypertrophied cartilage-like mass.

The condition when I first saw him was as follows:— From the cavity of the left orbit, apparently, arises a tumour 4½ inches long, 5 inches broad, of the colour of cheese, of stony hardness, irregularly pyramidal in shape, ivory-like, nodulated and botryoidal in appearance. The tumour admits of considerable movement in every direction and the nose is pressed by it to the right.

The neck is much enlarged, the anterior and lateral portions being of stony hardness, the left side being much larger than the right. The girth of the neck is 17½ inches, 10 inches of which are of stony hardness; the patient states that his throat was in this condition when he was born.

Whilst under observation the tumour gradually became looser, and an offensive discharge issued from the base.

On July 15th, at 1.50 p.m., whilst the patient was walking in the garden, the tumour suddenly fell and struck his boot, without either associated pain or hæmorrhage.

The site of the tumour presented a pedicle slightly forked at its summit and covered with granulations arising from the floor of the orbit, and corresponding to the cavity in the tumour, the two forming a kind of ball and socket joint."

A few days later Mr. Hughes trimmed up the orbit, removing a hypertrophied mass of hardened cutaneous

structures hanging down at the external margin of the orbit, and brought the integuments together with a suture.

The weight of the tumour is $9\frac{1}{4}$ oz.

It will be interesting if I add that, on the 18th of October of this year, I received a letter from the wife of William O—, she says: "My husband is alive, well, and happy, he is at present a master of a vessel, his eye is well, he did not feel anything from it since it was cured."

In 'Guy's Hospital Reports,' for 1836, Mr. Hilton recorded a case very similar to the present one, a drawing of the tumour and its site being given. The man was thirty-six years of age, the tumour commenced to grow "just under the eye close to the nose" when he was thirteen. The eyeball ruptured at nineteen, and when he reached thirty the tumour began to loosen; at thirty-four it was exposed and held only by bands of integument, and when these had ulcerated the tumour fell.

The weight was $14\frac{3}{4}$ oz.

Mr. Hutchinson, in his 'Illustrations of Clinical Surgery,' gives a portrait of a case, that of a woman aged fifty, who came under the care of Mr. Borlase Childs in 1859. A large nodular mass of ivory-like bone filled the orbit, and the eye was destroyed. The tumour was believed to spring from the frontal sinus, and in this case also necrosis and separation followed, although this was considered to be possibly due to violence inflicted during attempted removal a year before.

I allude to one other large ivory exostosis, well-known probably, inasmuch as Sir James Paget mentions it in his Surgical Pathology. An excellent photograph of it has been kindly sent me by Professor Humphry, of Cambridge, the specimen being in the University Museum.

In this case there was no tendency to spontaneous separation, and the mass projected both outside and inside the skull.

Boyer* alludes to the occasional spontaneous necrosis of these tumours as forming the best possible termination of the cases. *December 14th*, 1882.

* 'Traité des Maladies Chirurgicales,' cinquième edition, t. iii, p. 484.

III. INTRA-OCULAR TUMOURS.

1. *Epithelial growth from an eyelash in the anterior chamber.*

By W. C. Rockliffe (Hull).

(With Plate II, Figs. 1, 2 and 3.)

Joseph B—, æt. 23. About the first week in August, 1881, he was struck in the left eye by an iron staple, receiving a vertical lacerated wound in the outer third of the cornea. Prolapse of the iris followed, but retracted under atropine, and all inflammation subsided.

The patient first came under my observation on September 12th, 1881, (about six weeks after the accident) with supposed eyelash in the anterior chamber.

On careful examination a scar was found on the lower lid, and there was an absence of one or two eyelashes at a point corresponding with the corneal cicatrix. The patient remembered that the lid was slightly injured at the time of the accident.

The supposed eyelash was visible as a narrow dark line lying transversely, one end in the angle of the anterior chamber at the nasal end of its transverse diameter, passing somewhat obliquely downwards and outwards below the pupil, the other end hidden from view by the corneal cicatrix (Pl. II, fig. 1). There was a slight pink zone round the cornea and traumatic cataract.

With transmitted light there was some doubt whether the dark line was an eyelash or the remains of a hæmorrhage. But it was decided to open the anterior chamber and try to remove it. The attempt failed, the eyelash being firmly attached, from one end to the other, to the iris.

DESCRIPTION OF PLATE II.

Figs. 1, 2, and 3 illustrate Mr. Rockliffe's case of Epithelial Tumour of the Iris (p. 26). Half diagrammatic drawings by Miss Boole, from sketches by Mr. Rockliffe.

FIG. 1 shows the eyelash stretching from the scar of the corneal wound to the opposite side of the anterior chamber, before the growth had begun.

FIG. 2 shows the size, position, and appearance of the growth about one year after the accident.

FIG. 3 shows the appearances immediately before operation. The position of the eyelash, where covered by the tumour, is shown by the interrupted line.

Figs. 4 and 5 illustrate Dr. Stephen Mackenzie's case of Tubercle of the Choroid and Brain (p. 119).

FIG. 4.—Tubercle of choroid.

a. Tubercle.
b. Multinucleated giant cell.
c. Choroid.
d. Sclerotic.

FIG. 5.—Edge of one of the masses of confluent tubercle in the brain, more highly magnified.

a. Necrotic centre.
b. Multinucleated giant cell.

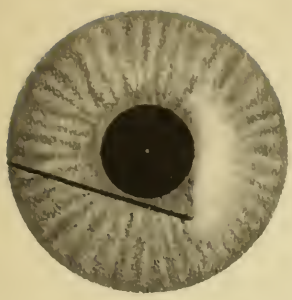


Fig. 1

Fig. 2.

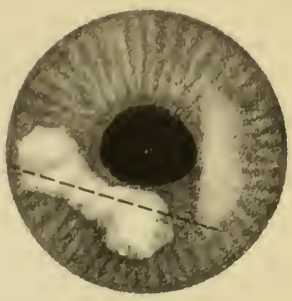


Fig 3

Fig. 4.

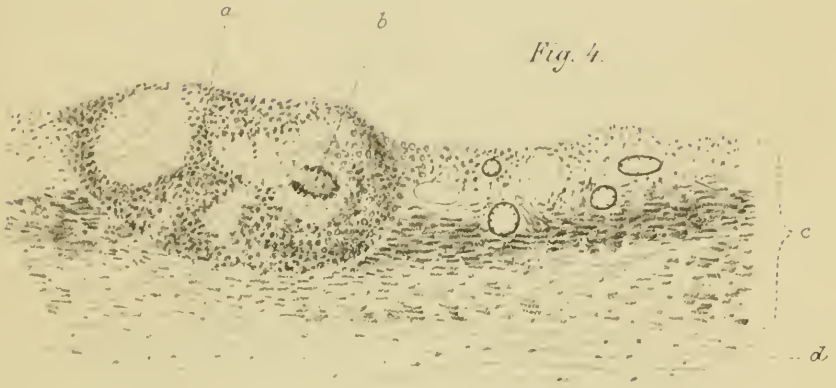
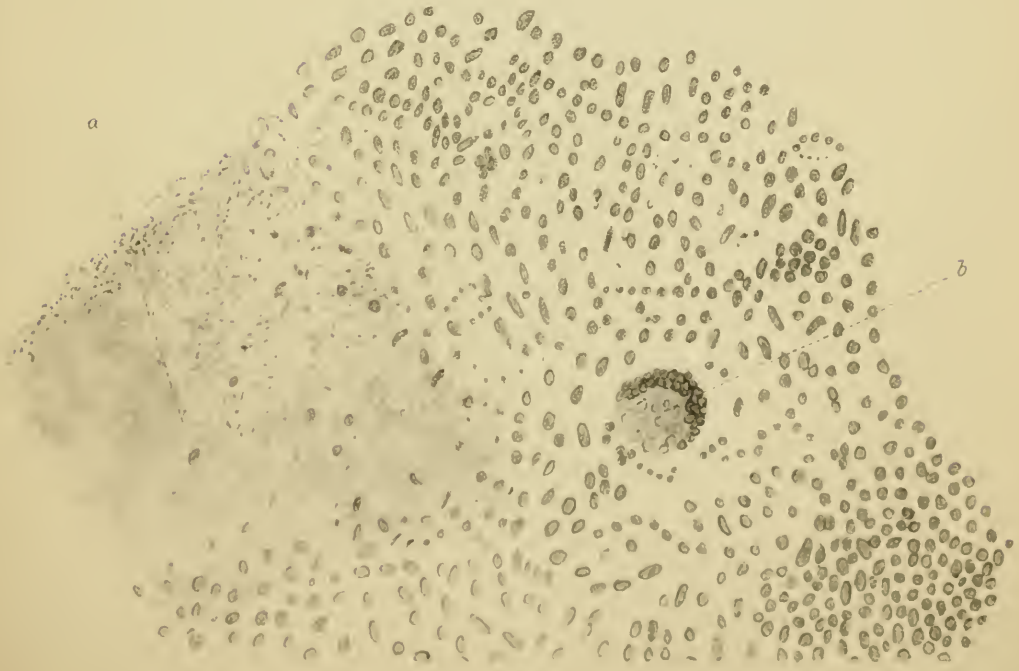


Fig. 5.



In a week all irritation and injection of sclerotic had disappeared; there was slight adhesion of iris, and the dark thread across the iris remained.

Vision at this date was: *Left,* counts fingers at 9′; cataract; *Right* = $\frac{20}{200}$, spells 6 J.; cannot read. T n. F. of V. free. No tenderness or irritation.

On inquiry I found he never had seen very well with the right eye, but there was nothing abnormal in the fundus to be detected, and his vision was not improved by glasses.

March, 1882.—No irritation. Vision same in right, rather improved in left. Cataract clearing.

September, 1882 (one year since accident).—Came with acute inflammation of left eye of fourteen days' duration. The corneal cicatrix vascular at its upper part.

Over the inner extremity of the supposed eyelash was a peculiar, white, woolly-looking growth resembling disintegrated lens matter, measuring about two millimetres in diameter and hemispherical in shape (Pl. II, fig. 2). This was taken to be a growth from the hair, and it rapidly increased in size.

The healthy (right) eye showed more symptoms of irritation. Soon afterwards, before deciding on further treatment, I sent him to Mr. Nettleship who wrote "probably a sebaceous (epithelial) tumour growing from the tissues of the eyelash," and recommended the removal of the growth by a large iridectomy. At this time the tumour had grown to such an extent, that the eyelash was entirely hidden from view (Pl. II, fig. 3). The growth was lying in apposition with the posterior surface of the cornea and pressing the iris backward.

October 18th.—Vision: *Left,* spells 22 J.; *Right,* spells 12 J.

Under ether I opened the lower and inner fifth of the anterior chamber, intending to make an iridectomy, when the tumour with the eyelash firmly adherent to its inner extremity was carried out with the rush of aqueous.

As there was an anterior synechia and considerable

inflammation a corresponding iridectomy was made. The wound healed without a bad symptom, all trace of inflammation having disappeared by November 8th when vision was:—*Left*, spells 17 J.; *Right* = $\frac{20}{100}$, spells 5 J.

Since this date I have not seen the patient, but hear that his vision is much improved in both eyes.

On examination the tumour measured 6 millimetres in length, 2 millimetres in width, 2 millimetres in depth.

Its anterior surface was smooth and moulded to the cornea, and the posterior surface and edges lobulated and more or less pigmented along that portion which had been in apposition with the eyelash. The eyelash stood at right angles to the posterior surface of the growth and was firmly adherent to it, so that the whole mass could be suspended by the eyelash without becoming detached.

I forwarded the specimen to Dr. Brailey who had kindly undertaken its examination and reported as follows:—"The mass consists of flattened epithelium cells exactly like the more superficial cells of the conjunctiva, and their nuclei stain very distinctly. It looks as if the cells of the root-sheath of the eyelash had proliferated in the anterior chamber."

The rare occurrence of such tumours is I hope sufficient apology for bringing the case before the Society.

(*January* 11*th*, 1883.)

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2. *Case of round-celled sarcoma of the iris; successful removal of the tumour.*

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

(With Plate III.)

The case I wish to bring before you, is that of a girl æt. 20, who was first seen by me in June, 1880. She stated that sixteen months previously, while in the act of

DESCRIPTION OF PLATE III.

Illustrating Dr. David Little's case of Sarcoma of the Iris From drawings by A. H. Young, M.B. (p. 28).

Fig. 1.—Eye, showing new growth *in situ* (natural size).

Fig. 2.—Section of tumour, under low power (Zeiss, obj. A × 4). Shows cellular elements interspersed with strands of more fully formed connective tissue. Sections of embryonic vessels are also seen.

Fig. 3.—Portion of same section, under higher power (Zeiss, E × 4).

Fig. 4.—Isolated cells (Zeiss, obj. E × 4).

Fig. 1.

Fig. 2.

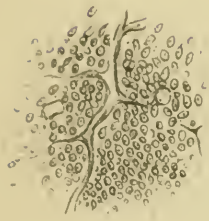

Fig. 4.

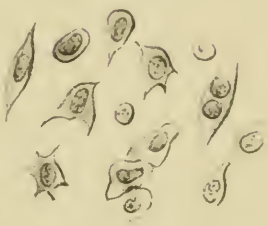

Fig. 3.

stooping, her left eye became suddenly blind, and remained so for nearly a week. On recovering sight she observed for the first time, a spot on the coloured part of the eye. During those sixteen months she had three or four attacks of dimness of vision at irregular intervals, but the sight of the eye on each occasion returned.

These attacks probably depended on hæmorrhage into the anterior chamber.

She has always had good health and there was no evidence or suspicion of syphilis. Her family history was good.

On examining the eye, I observed a pale, brownish mass the size of a small pea on the outer and lower quadrant of the iris. It extended from the pupillary margin to the periphery of the iris. It was somewhat irregular in shape and portions of it came in contact with the posterior part of the cornea; its surface presented some minute blood-vessels, and numerous red points. The eye was free from all irritation, the iris and fundus were healthy, and the vision normal. I had no doubt at the time the growth was sarcomatous, but as there appeared to be nothing urgent in the case, I determined to watch it for a time.

At the end of four months, the tumour had visibly increased, particularly towards the peripheral region; in all other respects the eye was the same as when I first saw it. Fearing a further extension of the growth towards the ciliary body, I proposed to remove it.

On the 30th November of the same year, the patient was put under ether, and a linear incision was made with a Graëfe's knife, close to the corneo-scleral junction, very similar to that made in cataract extraction. The tumour and corresponding piece of iris were removed with forceps without difficulty, no bleeding occurred in the anterior chamber. In about three or four weeks the eye had perfectly recovered and the vision was $\frac{20}{20}$.

I handed the specimen to my colleague Dr. Dreschfeld, professor of pathology in Owens College, for microscopical examination. He reported as follows:—

"The mass consisted entirely of round cells; these had the size of leucocytes, but contained a very large round nucleus, filling up nearly the whole of the cell, and showing in its centre one or more highly refractive nucleoli. Besides these round cells, there were found free nuclei, oval cells with large nuclei, and a few spindle cells with nuclei. Many of these cells were not pigmented; a few however, in the neighbourhood of the bloodvessels contained brown pigment both in the nucleus and cell protoplasm. The blood-vessels were all of the embryonic type, their walls consisting of one or more rows of cells arranged longitudinally, having large and well defined nuclei. This examination shows the tumour to be a pigmented round-celled sarcoma" (Plate III).*

It is now over two years since I removed this tumour and when I last saw the patient three or four months ago there was no evidence of recurrence and the sight was normal.

Although sarcoma of the iris, must be considered a rare disease, yet there are several well authenticated cases on record, and I propose to give a short summary of those to which I have been able to refer.

In a paper by Dr. Kipp of Newark, N.J., in Knapp's "Archives of Ophthalmology," vol. v, p. 34, (1876) giving particulars of a case under his own care, I learn that the first case on record is one which occurred in the practice of Von Graëfe in 1868 and was published by Hirschberg in the Archiv. f. Ophthalmologie Bd. xiv, Abtheilung 3, p. 235. The patient was a man æt. 38, with a tumour of a dark brown colour, on the lower part of the iris, nearly filling the lower two-thirds of the anterior chamber. Central and eccentric vision were unimpaired. Graëfe diagnosed malignant disease and enucleated the eye. Hirschberg made a microscopical examination and showed the tumour to be a pigmented spindle-celled sarcoma.

* For the drawings from which the Plate was taken I am indebted to my friend, Dr. A. H. Young, Lecturer on Surgical Pathology, Owens College.

Five months after the operation, there was no return of the disease.

In the same 'Archives,' for 1874 (vol. iii, p. 106) Drs. Argyll Robertson and Knapp publish a case. The patient was a healthy woman, twenty-four years of age, presenting a small light brown tumour on the right iris, at its upper and outer ciliary margin. The growth increased in size, there was deep glaucomatous excavation (T. + 2) and the vision was reduced to faint perception of light. The eye was enucleated. Dr. Knapp made a microscopical examination and pronounced the tumour a melanotic sarcoma. Two years after the operation there was no recurrence of disease.

The third case occurred in my own practice in 1871, and is recorded by Dr. Dreschfeld in the 'Lancet,' January 16th, 1875, p. 82.

The patient was a lady, æt. 53, subject to rheumatism, and altogether of indifferent health. A reddish grey tumour developed in about two and a half years in the lower part of the anterior chamber of the left eye; its growth was attended by repeated hæmorrhages in the anterior chamber. For about two years the tumour had increased very little, the fundus continued healthy, and the vision good; ultimately hæmorrhage became more frequent and extensive, the growth rapidly increased, filling nearly the lower half of the anterior chamber, the eye became painful and glaucomatous, and vision rapidly deteriorated. In July, 1874, I performed enucleation. Dr. Dreschfeld made a careful microscopical examination and pronounced the tumour a spindle-celled sarcoma. I had an opportunity of seeing this patient several years afterwards and the orbit remained healthy.

Dr. Kipp's own case, in his paper referred to, is the first in which a sarcoma of the iris has been excised successfully with preservation of sight. It occurred to a healthy man, æt. 36, the tumour occupied the inner and lower quadrant of the right iris, extending from the pupillary to the ciliary margin. It had developed very

slowly, having been first noticed twelve years before. The eye, in all other respects, was healthy and the vision normal; excision of the growth with the corresponding portion of iris, was performed. Vision afterwards was $\frac{20}{20}$. Microscopic examination showed it to be a spindle-celled sarcoma. Eighteen months after operation the eye was free from disease.

The case resembles the one I now report almost in every particular.

Mr. Brudenell Carter gives particulars of a case of sarcomata of both irides, in a boy, æt. 15 (vide 'Clinical Soc. Trans.,' vol. vii, 1874). In the left eye the tumour was of the size and colour of a split pea on the lower inner quadrant of iris; it was of three months' duration. Vision reduced to 8 J. In the right eye there were two small tumours the size of pins' heads. Vision was unaffected. The growth in the left eye was excised and was proved to be a round-celled sarcoma; ultimately a fresh tumour appeared in the outer portion of the iris. The growths in the right eye steadily increased. About nine months later Mr. Carter had an opportunity of examining the patient, when there was very little visible increase of the growths, but vision even of the right eye was limited to the perception of large objects.

Dr. Knapp, of New York, reports three cases of successful removal of sarcoma of the iris (vide Knapp's 'Archives of Ophthalmology,' vol. viii, p. 82, 1879). In the first case, a man, æt. 36, the tumour occupied fully one third of the iris. V. $= \frac{20}{70}$. The mass was excised and after the eye had recovered, sight was $\frac{20}{200}$; twelve months after operation there was no return of the disease. On examination the growth was found to be a spindle-celled sarcoma.

His second case was a Frenchwoman, æt. 35, with a blackish tumour, the size of a small pea in the lower part of her iris, it had existed for many years and its presence had given rise to repeated attacks of iritis, leaving posterior synechiæ. Vision $\frac{20}{100}$. The tumour was removed and was found to be a melanotic spindle-celled sarcoma.

The sight after the operation, was as good as before. There was apparently no return of the disease three years afterwards.

The third case occurred in a man, æt. 22, with a yellowish-red tumour on the lower and outer portion of the iris, the eye otherwise was normal. The tumour was excised and found to be a sarcoma. The result is not given as the case was recent when Dr. Knapp wrote his report.

In the same paper Dr. Knapp refers to a case by Lebrun,* of rapid growth; in this case the globe was removed; and also to one observed by Dr. B. St. John Roosa, but not operated upon.

The last case I find on record is one published in the 'Lancet,' April 12th, 1879, p. 511, by Mr. J. E. Adams, of London. The patient was a girl, æt. 13. On the lower and outer part of the left iris there was a round brownish tumour of the size of two pins' heads, extending to the ciliary region; the eye showed slight signs of a former iritis with faint corneal dotted opacities. Vision was $\frac{20}{50}$. Enucleation was performed, and the tumour was found to be a round-celled sarcoma. It was shown, by examination of the excised eye, that any attempt to save the organ would have been worse than useless.

Reference may also be made to Mr. Nettleship's case of multiple growths from the irides in the first volume of our 'Transactions.'

(*March 8th*, 1883.)

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3. *Case of cysticercus in the vitreous humour.*

By J. W. Hulke, F.R.S.

Annie R—, æt. 6, admitted into the Royal London Ophthalmic Hospital on March 14th, 1883, under Mr. Hulke's

* 'Annal. d'Ocul.,' vol. lx, p. 209.

care. The mother complained that the child could not see with the right eye, and that the sight had been failing for two months, though there had been a "cast" for eighteen months.

The family history is unimportant.

The patient has always been a nervous and excitable child, and during the last two years the mother has noticed that on an average of once a week the child would become suddenly restless in her sleep, moaning and groaning and rolling her eyes for two or three minutes, then awaking suddenly in a state of free perspiration and with an expression of surprise. The child otherwise enjoys good health, but is rather anæmic.

No signs of the presence of a tapeworm have been seen.

Right eye.—Vision = shadows, no field, T. full. With the ophthalmoscope a large, white or bluish-white cyst is seen, nearly circular in form, to the outer side of and overlying about one half of the disc (drawing exhibited). The cyst appears to be in front of the retina and projecting into the vitreous; a retinal vessel is seen through the cyst running downwards and outwards. The cyst looks to be five or six times the size of the disc. Several small hæmorrhages surround the cyst. Downwards and to the inner side of the cyst is a dark patch rather smaller than the disc in size, it is situated behind the retina and is probably an old hæmorrhage; in front of this patch the vitreous is rather hazy. At about the distance of the breadth of the cyst from its margin is seen in all directions a shimmer of light, which moves with the movement of the mirror and is apparently due to a reflex of light from the surface of the cyst. In the centre of the cyst is a part, more markedly white than the rest, which may be taken for the head of a cysticercus invaginated into its cyst, but no movement could be made out.

The child was removed by the parents, and was afterwards in St. Bartholomew's Hospital under Mr. Vernon,

who has recorded the case in the 'Lancet' for May 26th, p. 904. The case had, however, been previously entered on the list of communications to the Society, but deferred for want of time.

(*Card specimen. July 6th*, 1883.)

IV.—PANOPHTHALMITIS.

1. *On certain cases of destructive ophthalmitis, simulating glioma, in children.*

By E. Nettleship.

The cases referred to in the above title have not, I believe, received the attention that their pathological interest and clinical importance demand. In many of them we are called on to decide whether or not the disease is glioma of the retina, and the term "pseudo-glioma" in common use for certain of the cases under notice points clearly enough to their clinical resemblances. But it is especially in reference to their morbid anatomy and their clinical and pathological relationships that further examination is needed. We must look chiefly to those engaged with diseases of children to fill up many of the gaps in our knowledge of the somewhat miscellaneous group of cases included in this paper. I propose to do little more than to record such as have been under my own care or placed at my disposal by friends, and to give the most important points in such published cases as I have been able to find, no large number indeed, for it is only now and then that a case seems to have been thought worth putting on record. Published cases in which the account given leaves it doubtful whether the changes were malignant are not included. Nor have I included those known to be examples of tubercular inflammation, or large solitary tubercle, of the structures of the eyeball.

The chief clinical differences between "medullary cancer

of the retina" in young children and cases of deep-seated inflammatory deposits behind the lens were clearly drawn many years ago; in 1829 Mr. Travers* drew attention to cases in which "a splendid yellow tint" was reflected from deep in the eye, and where from the previous history and the absence of general symptoms he had decided against malignant disease and had abstained from operating; his diagnosis being confirmed in several such instances by the subsequent dwindling of the eyeball.

One of the most important distinctions between real and spurious glioma is found in the state of the iris and anterior chamber. Mackenzie† in his 'Treatise' observes that in some cases of non-malignant deposits occupying the place of the vitreous humour "the ciliary edge of the iris appears wrinkled, the larger circle is drawn somewhat backwards, while the smaller circle projects forwards and is broader than usual; the pupil is in a middle state of dilatation, and its edge is fringed with uvea." To this we may add that in some cases synechiæ from previous iritis are present, though the periphery of the iris is not retracted. Mackenzie had also noticed that the colour of the deposit or growth was not the same in all instances, and that it was "not so defined as in the malignant cases." I am under the impression, though I may very probably be mistaken, that these points have often not been sufficiently attended to in diagnosis.

Increase of tension in cases of the class under notice is almost always proof that the disease is glioma.‡ Marked and persistent lowering of tension is a still more certain sign that the disease is innocent. Even when the tension is normal, as it often is in the early stage of glioma, and as it sometimes is in cases simulating glioma, the state of the

* Travers, "Observations on the Local Diseases termed Malignant," 'Med.-Chir. Trans.,' xv, 235, &c.

† Mackenzie, 'Treatise,' 4th ed., 1854, p. 709.

‡ For exceptions, see "A Case of Chronic Suppuration of the Vitreous in a child, æt. 7," by Vernon, 'Ophth. Hosp. Rep.,' vi, 293; and Brailey, 'Guy's Hosp. Rep.,' xl, p. 500 (1881).

iris and pupil of the affected eye is nearly always distinctive. The presence of blood-vessels visible on the growth or mass is by no means proof of glioma, though always a suspicious circumstance.

The pathological changes found in the eyeball of course vary much according to the acuteness and severity of the case and the stage of the attack at which the specimen was obtained. There seem, however, to be two principal types, one with, the other without, eventual detachment of the retina.—In many, probably in most, of the cases an acute puro-plastic, or a chronic plastic, irido-cyclitis causes exudation into the vitreous humour, especially into its anterior part; exudation may also occur between the choroid and retina. By the subsequent organisation and shrinking of the lymph in the vitreous, the ciliary processes and periphery of the iris are dragged backwards and inwards, and the retina separated from the choroid. Sometimes a large collection of blood (Cases 6, 14) or bloody fluid (Cases 12, 13) is found in the sub-retinal space, no doubt the result, not the cause, of the retinal detachment.—In other cases we find inflammation of the outer layers of the vitreous, and of its axial portion, associated with diffuse hyperplastic or fibrous retinitis, the retina, however, remaining in position (Cases 7, 8).

Raab* has described essentially similar changes; in three eyes enucleated for supposed glioma he found membranes in the vitreous and fibroid thickening of the retina; in two others sero-purulent exudation into the vitreous from irido-choroiditis, causing detachment of the retina.

Two cases, each illustrating one of the above types, have been lately published by Mr. Higgens† with Dr. Brailey's report.

Brailey‡ has recently stated that masses of connective tissue occasionally grow from the optic disc; and he adds

* Raab, Graefe's 'Archiv f. Ophth.,' xxiv, iii, 163 (1878).

† Higgens, 'Lancet,' 1882, i, 860.

‡ Brailey, 'Guy's Hosp. Rep.,' loc. cit.

"spontaneous suppurative hyalitis" and "spontaneous diffuse uveitis" to the list of primary morbid changes which sometimes occur.

It is of even more importance to inquire into the causes of the various states which may simulate glioma, and it is especially here that physicians can help us, for they more often see the cases at their commencement. In the cases recorded below it will be found that whilst in some the eye-changes were not accompanied by any symptoms of general disturbance, in the greater number the eye attack was preceded or accompanied by an attack of one of the infectious diseases, especially hooping-cough or measles, or by an acute illness suggestive of meningitis, or that it occurred in a syphilitic child. Further, whilst in some the eye is lost with violent inflammatory symptoms the lids being "swollen" or "projected out," in others, all remains quiet from beginning to end. It is sometimes in these quiet cases that the greatest difficulty in the diagnosis from glioma is found (Case 17).

In some cases perforation of the cornea takes place, probably as a consequence of suppurative action transmitted from the deeper structures. Dr. Barlow tells me that he has seen more than one case of this kind in connection with some general illness, and a case has been recorded by Hutchinson.*

The acute cases are seldom brought to the surgeon until the child's health is restored and the eye comparatively quiet, and we therefore, as a rule, have only the history to guide us.

In regard to the acute cases with febrile symptoms, or associated with one or other of the exanthemata, three suppositions readily occur as possibly serving to explain the connection between the local symptoms and the constitutional disturbance. When the illness is of an undefined character it may be that the general symptoms are entirely due to the irritation of the inflamed eyeball, being analogous to the symptoms caused by teething. A

* Hutchinson, 'Ophth. Hosp. Rep.,' vi, 219.

slight examination of the cases will show, I think, that this explanation can hold good in but very few.

The convulsions, screaming and unconsciousness, of which a history is often obtained, might point to some form of meningitis of which the ocular disease was an occasional consequence. I must confess that in a certain number of the cases, where the history of cerebral disturbance has been clear, this explanation has seemed to me at the time very probable. It is supported by the well-known fact that purulent irido-choroiditis occurs in some cases of epidemic cerebro-spinal meningitis. Prof. Förster, in his valuable chapter in Graefe and Saemisch's handbook on the relation between ophthalmic and general diseases, states positively that sporadic meningitis, especially in children under five, is a common cause of the eye-changes we are considering; he does not, however, give any details. This point, of course, can be decided only by physicians; we want clinical records of the cases in their early stages, and especially post-mortem examinations, if indeed the children who suffer from these eye-changes ever die during their quasi-cerebral attacks. Should meningitis be proved we shall still have to ask why and how does it sometimes cause inflammation of the uveal tract instead of optic neuritis?, how is the eye mischief set up?, and why is it so much more common in children than adults?

A third suggestion, and one in favour of which I am prepared to hear a good deal of testimony, would connect the eye disease in nearly every case with some one or other of the exanthemata. Should the frequency of the association between these eye affections and the acute exanthemata receive confirmation, there will still be the question whether the ophthalmitis forms a part of the constitutional malady itself, as iritis does of secondary syphilis, or is to be looked upon as the result of some condition, allied perhaps to pyæmia, occasionally following the specific fever. The possibility that some of the cases are

pyæmic was suggested by Hutchinson* some years ago in connection with two cases of destructive eye disease in young children. The first was a case (No. 10 of my series) of double destructive panophthalmitis with opaque vitreous, and ultimately secondary cataracts, in a pale flabby child, a year and nine months old, suffering at the time from an eruption which Mr. Hutchinson thought was chicken pox. The second (No. 11) was in a male infant of eighteen months who lost both eyes by suppurative inflammation, with destruction of the cornea, just after a severe febrile illness in which an abscess formed on one hip. After discussing the possibility of pyæmia in these two cases Mr. Hutchinson adds that he believes varicella to be "much more than any other (exanthem) liable to be followed by severe complications of a pseudo-pyæmic nature." Dr. Barlow informs me that he has often suspected that the cases of "pseudo-glioma" which he has seen might be pyæmic; recovery from a pyæmic condition being, he thinks, not very uncommon in children. The same point was raised in a case which I published a short time since,† unfortunately without making reference to Mr. Hutchinson's earlier observations.

Before drawing any conclusions as to the above or any other points, I will give the details of such cases as have come under my own notice, and the outlines of the more important British published cases.

Case 1.—Julia B—, æt. 3½. When I saw her she was healthy looking, well grown, and intelligent.

A year previously she had had bronchitis, followed in a few months by hooping cough. She had also had eczema of the head and face. She was described as having been always delicate and easily upset. There was not much reason to think her syphilitic.

About a month before admission she began to have pain in the head, and it seemed to be chiefly in the left frontal region; twelve hours later convulsions set in, she

* Hutchinson, 'Ophth. Hosp. Rep.,' vi, 146 and 219 (1868).

† Nettleship, 'Med. Times and Gazette,' 1880, i, p. 63.

became unconscious, and remained so for a week. On the third day of the illness the left eye was swollen and inflamed, and the iris, as the medical attendant, Dr. Soper, informed me, much discoloured. The eyeball continued very painful for a week, after which the child recovered its health. When I saw her on March 11th, 1881, the left eye and left side of the head seemed still to be painful; the eye was soft (T.−2), the periphery of the iris retracted, its inner circle pushed forwards into contact with the cornea, the cornea and lens clear, and a yellowish-red reflex, devoid of vessels, came from behind the lens. The other eye was healthy. I did not excise the affected eye.

Case 2.—Dr. B— brought me his little girl, eighteen months old, in March, 1881. The left eye was congested and very soft (T.−3), pupil small from iritis, the iris touching the cornea, and a bright yellowish reflex from behind the clear lens. The eye being intolerant of light and the child fretful, a good examination was impossible.

Just three months previously, after a drive in the park, the child was one day seized with quick breathing, rapidly followed by unconsciousness, with dilated pupils. There were never any convulsions. The next day the left eye was noticed to be red; soon iritis set in, and was followed by a "fiery" appearance deep in the pupil. There was, however, no proptosis. The child remained very ill for nearly three months, *i.e.* till shortly before I saw her, and the temperature for the first fortnight was often 103° and 104° F. She was seen by an eminent authority on diseases of the nervous system, and was several times so ill that she was expected to die.

Soon after the beginning of the illness hooping-cough developed, and it is perhaps open to question whether the whole attack may not have been a masked form of that disease.

I saw the child again some months later, and as the

eye was still somewhat troublesome I advised excision, but the parents did not consent.

Case 3.—Jane F—. At the age of ten months had measles, not very badly. Either during or soon after the measles her mother noticed a yellowness in the pupil of the right eye, and thought the eye was blind. The eye seems to have given no trouble till more than a year later, when the eyelids swelled up, and it was about two months after this occurred that the child was brought to the Hospital for Sick Children. She was then (Sept., 1882) two years and four months old; she was fretful, but sleepy, was suffering from cough, and said to be losing flesh.

The right eye was somewhat shrunken, soft, and much congested. The pupil dilated, and filled by a bright yellow mass, probably the altered lens; the iris either bloodstained or covered by dark blood. Often put its hand up to the blind eye, but did not cry as if in pain. Excision was advised, but declined.

Case 4, under my care at St. Thomas's Hospital, was a case of acute destructive irido-cyclitis with lymph on the iris, accompanying a slight feverish attack with effusion into the joints. It was, in my opinion, a question at the time whether the attack was allied to pyæmia. The patient was a boy of four years who had had measles six months before.*

Case 5 was briefly as follows:—Richard B—, had "bronchitis and low fever" when about eight months old, and after being ill three or four weeks was not expected to recover. During the illness the left eyelids swelled and the eye became very red; there was no discharge. I saw him at ten months of age; the eye was soft (T.−2) and rather small, centre of iris pushed forward, pupil excluded, a bright pinkish-yellow reflex,

* Published in full in 'Med. Times and Gaz.,' 1880, i, 638.

without blood-vessels, was seen from behind the clear lens. The case was watched for some months but nothing was done.*

Case 6.—James B—, æt. 16 months. A few weeks ago the child was very ill for a time with screaming attacks and other apparently cerebral symptoms. During or just after the illness a reddish appearance was seen in one eye. When admitted the eye was soft, there were iritic adhesions and a yellowish-red deep reflex. After excision by Dr. W. A. Duncan, the retina was found detached and shrunken, and the subretinal space filled by blood.†

Case 7.—Emma G—,‡ æt. 5. The eye was not excised till it had been blind more than four years. Here I found the vitreous traversed from before backwards by fibrous cords in one of which were blood-vessels (probably persistent hyaloid vessels), whilst the cortex of the vitreous was also converted into a fibrillated tissue. The retina was not detached. Probably a severe chronic retinitis was the primary change. This patient was born prematurely and was always small and weakly but there was no evidence or history of syphilis. She was much weakened by measles at six months of age, and about this time the eye (*i.e.* pupil) "turned colour," but there was neither pain nor inflammation. She was still puny and weak with prominent forehead and large veins, when Mr. Hutchinson removed the globe.

Case 8.—Brailey§ has described what was probably an earlier stage of the same condition, in the eye of a boy, two and a half years old; the outer layer of the vitreous was opaque, corpuscular, and fibrillated, and there were thick white streaks along the retinal vessels near the disc; no detachment of retina; clinical history imperfect.

* Published, ibid.

† Published in 'Path. Soc. Trans.' for 1880, p. 253.

‡ Nettleship, 'Ophth. Hosp. Rep.,' vii, 632 (1873).

§ Brailey, 'Ophth. Hosp. Rep.,' viii, 537 (1876).

Case 9.—In another specimen from a girl, æt. 3½, Brailey* found a fibrous cord traversing the axis of the vitreous, expanding into a thin membrane behind the lens which was reflected so as to line the inner surface of the ciliary body. There were iritic adhesions. The lens was clear and clinically the case resembled detachment of the retina. At about eighteen months of age this child, a girl, was very ill for three months with cough, severe diarrhœa and sickness. At about the end of the illness the right eye became blood-shot and "prominent" but the mother does not seem to have noticed the whiteness in the pupil till several months later, when the child was between two and three years old, when also she found that the eye was blind. The cough had continued all this time and is said to have been like hooping-cough, and two other children in the house had hooping-cough about the same time. The child was brought to Mr. Hutchinson at three and a half years of age, when the eye had been blind for probably more than a year.

Cases 10 and 11 are those of Hutchinson already briefly given† (p. 41).

Case 12.—In the right eye of a boy, æt. 18 months, Brailey‡ found detachment and shrinking of the retina, the remains of the vitreous firm, tough, and opaque, and bloody fluid between retina and choroid. Here there was no history of inflammatory symptoms.

Case 13. — Mr. Critchett§ excised the eye of a female child about two months after the onset of severe iritis followed by a deep-seated yellow reflex. Malignant disease was suspected, but on dissection the vitreous was found shrunken, its anterior part converted into fibrous tissue, and the retina separated from the choroid by

* Brailey, ibid., p. 543.

† Hutchinson, 'Ophth. Hosp. Rep.,' vi, 146 and 219 (1868).

‡ Brailey, 'Ophth. Hosp. Rep.,' viii, 538 (1876).

§ Critchett, 'Lancet,' 1854, i, 242. The specimen is described, with some additional details, in Mackenzie's 'Treatise,' p. 712.

grumous, bloody fluid. The child had been pale, thin weak, and restless, but soon improved after the operation ; the fontanelle was widely open and the forehead "indicating some tendency to hydrocephalus." There is no mention of syphilis.

Case 14.—In a case by Mr. Hulke* a child of ten had acute inflammation of the left eye for three weeks ; before excision a yellow gleam was apparent in the pupil. The retina was detached, and probably drawn forwards by the contraction of the shrunken, yellowish vitreous ; the subretinal space was filled by bloody fluid and fresh blood ; the choroid œdematous, the lens clear. This eye had been long defective and presented a scar on the cornea with anterior synechia the result, Mr. Hulke states, of perforating ulcer. Mr. Hulke does not discuss the question of injury ; but we may suggest that the conditions found were more like those following a small perforating wound penetrating to the vitreous, than a corneal ulcer.†

Case 15. *Detachment of retina, &c., in both eyes coming on with symptoms of subacute meningitis ; hereditary syphilis.*—Isabel Anne H— was admitted under Dr. Barlow's care at Great Ormond Street in October, 1880, and whilst there I had an opportunity of seeing her, and have to thank Dr. Barlow for permission to use his notes. She was quite blind of both eyes from what appeared to be detachment of the retinæ, but the reflex was brighter than in ordinary detachments as if there were something pink or yellowish deep in the eyes.

Nine weeks before admission she had been taken ill with vomiting and convulsions, followed by an apathetic (? unconscious) state which lasted a few days. The vomiting continued a week. She became blind during the illness, but no account was obtained of any inflammatory symptoms in the eyes.

* Hulke, 'Ophth. Hosp. Rep.,' iii, 274.

† For a case by the author bearing on this question, see 'Lancet,' 1875, ii, 278 (Case 2).

For a time after admission there were symptoms which, together with the history, were thought to indicate subacute meningitis, probably cerebro-spinal, the chief symptoms being retraction of the head, inability to sit up, and that she always cried when moved.

No convulsions occurred after admission. She was not febrile. She got fatter whilst in the ward, but did not improve in other respects. Fontanelle still open, but child not otherwise rickety.

The previous history pointed strongly to hereditary syphilis; in early infancy the child had thrush and well-marked eruption about the buttocks, and the only previous pregnancy had resulted in a miscarriage. The child had had no infectious diseases. She was suckled for fourteen months.

Case 16. *Loss of one eye from irido-choroiditis during an illness (? meningitis); other eye slightly affected; hereditary syphilis.*—Georgina S— was sent to me by Dr. Barlow, and considered by him, from the history, to be syphilitic. She was pretty well for some months and had begun to talk. When about nine months old she became ill; she used to scream and vomit, and is said to have had fits at intervals for more than a year. Early in the illness both eyes inflamed, but were not swollen; the right recovered, but the left was blind. She remained ill for several months.

I saw her when she was three years old; the left eye was then somewhat shrunken, T.−; iris in contact with cornea; lens opaque. Three years later (May, 1882) there was a transversely oval patch of superficial haze on the cornea, but the eye was otherwise unaltered and quite quiet. The left was examined ophthalmoscopically both times, and found quite healthy.

She lost her power of talking during the illness, and has become idiotic and bad tempered, though she is well grown.

In this case the eye was not seen until secondary

cataract had developed, but in other respects the case agrees with most of the others.

Case 17.—Thomas C—, sent by Dr. Barlow. In infancy was treated for well-marked congenital syphilis by Dr. Read. Could see well till the age of about six months, when he became ill with bronchitis, and about that time his eyes began to "work," and his mother thought he could not see well. The eyes were not inflamed. No history of injury. He had no fits. Has not had hooping-cough, measles, or any other children's complaints. Was suckled till seven months old. He is the fourth born and eldest living of the family, the three elder children having died at birth, at five months, and at three weeks of age respectively. Father had some venereal disease about a year before marriage.

On admission (September 26th, 1882) he was aged 15 months, well grown and good tempered, was beginning to speak, and could nearly stand alone. There was small, quick, horizontal nystagmus of both eyes. His father, who brought him, thought he could not see at all with the right, and it was evident that he saw very badly even with the left.

Condition of right eye.—T.n.; anterior chamber uniformly shallow; pupil shows delicate pointed posterior synechiæ; behind the clear lens is a whitish flocculent-looking opacity, on some parts of which vessels are clearly visible; no chalky-looking spots or nodules as in many cases of glioma. No red reflex. *Left eye.*—T.n.; anterior chamber shallow; pupil measures about 5 mm., dilates well to atropine, and shows no traces of iritis; lens and vitreous clear; the whole choroid shows slight superficial changes (fine disturbance of epithelium); disc very pale and rather hazy, retinal vessels can only be traced a short distance.

Though the presence of iritic adhesions in the right, and of ordinary syphilitic choroido-retinitis in the left,

were strongly against the diagnosis of glioma retinæ in the former eye, the appearances were so equivocal that I advised excision, and performed it on October 3rd. On opening the fresh eyeball the vitreous was found much shrunken and tough, the retina detached in the usual manner in deep folds, some of which were in contact by their inner surfaces, and others irregularly constricted; the sub-retinal fluid was muddy and reddish yellow, but on the choroid at the inner side was a thin layer of white curdy-looking substance, easily separable from the choroid. The choroid itself was patchy, but showed no distinct signs of atrophy.

Case 18.—Martha W— was under the care of Mr. Owen and Dr. Barlow at Great Ormond Street for congenital syphilis from the age of seven weeks till she was two years and a half old; the chief symptoms were enlargement of one tibia and fibula and of the ends of the bones forming both elbow-joints, and slight snuffles. There was no history of symptoms in the mother. "Keratitis" was noted on the out-patient paper at about the age of six months, but no details given. The child was brought to St. Thomas's when two years and seven months old (September 3rd, 1879) for an opinion as to its eyes. The mother then said that the left had been blind "from birth," and that the right was "going."

In the left eye I found old iritic adhesions with complete cataract, and the iris in contact with the cornea. In the right the pupil, though small and inactive to light, dilated fairly to atropine, and showed no evidence of iritis; a large quantity of thin membranous-looking opacity was seen in the vitreous, chiefly at the periphery as if lining the ciliary body, but extending, though less densely, behind the whole surface of the lens; disc easily seen and pale, but no other decided changes made out. The child could see large objects. There was now no evidence of the previous bone disease.

Case 19. *Severe hereditary syphilis, with choroiditis, iritis, detachment of retina, and a large membrane in the vitreous in both eyes.*—E. B—, æt. 2, was the second born of three children, the first and third being still-born. She was very ill and had bad thrush soon after birth; no snuffles and no fits. Father had "ulcerated throat" shortly before marriage; mother no symptoms. The left eye is said to have looked "peculiar" a few months after birth, but the whitish appearance had only been noticed a month. Latterly she had been very fond of putting her hand up over this eye but there was no history of inflammation of the eyes.

When admitted into St. Thomas's Hospital in December 1879, at the age of two years, she was fairly well grown, intelligent, and could speak a little, but was weak on her legs; frontal eminences prominent; temporo-parietal suture ridged. Dr. Barlow who examined her reported that she was rather cyanotic, and that there was dulness over the base of the left lung, but no evidence of disease or malformation of the heart. In both eyes there were long slender posterior synechiæ, opacities in the vitreous, detachment of retina and choroiditis. In the right the retinal detachment was confined to the upper-inner part, and there was much choroiditis near it; there was a semi-transparent cord (? hyaloid canal) stretching forwards from the optic disc. In the left a tremulous mottled membrane causing a conspicuous, whitish pupillary reflex, was present at the inner half of the eye; no vessels were visible on it; very little choroiditis was visible. It is doubtful whether the appearance described was due to the retina or to a præretinal membrane.

Case 20.—L. P—, æt. 13 months. Two months ago she was ill with what was called "water on the brain," and the doctor thought the case serious. The peculiar appearance in the left pupil has been noticed only about two weeks. The left eye is now soft (T−2), and slightly congested, the anterior chamber very shallow, the pupil irregular from synechiæ and showing a deeply-seated

bright reflex, partly yellow, partly red. There is a history of ulcerated mouth and sore buttocks, at two months old; the forehead is prominent and rounded, and the head rather large. The child is therefore probably syphilitic.

Case 21. *Hereditary syphilis; choroido-retinitis; detachment of retina with sub-retinal tough membrane in one eye; membrane in vitreous of other eye.*—T. H—, æt. 11, was under the care of Drs. Gee and Barlow, at Great Ormond Street, for a long time in 1878-79. Her eyes were repeatedly examined by several very good observers; in the left there was extensive old choroido-retinitis pigmentosa, in the right appearances which some translated as due to ordinary detachment of retina, others as indicating some solid exudation between retina and choroid. She died in January 1880, and I obtained the back of each eyeball.—In the right the retina was detached in the usual way, its layers thickened, and almost adherent by their inner surfaces, and the whole retina displaced towards the lower part of the eye where it was still in contact with the choroid over a narrow tract. In other parts, especially near the disc, were several strings of adhesion stretching between the retina and choroid, evidently formed before the detachment occurred. Between the retina and choroid, towards the lower part of the globe, was a perfectly distinct membrane of fibrous tissue which in the fresh specimen was not attached to either tissue, but, after hardening, came to be slightly adherent to the choroid in parts. There was abundant evidence of previous superficial choroiditis.—In the left there was besides extensive choroiditis a very distinct membrane in the vitreous, broad and folded in front, tapering to a narrow stalk by which its hinder extremity was attached to the disc. No microscopical examination was made.

There was wasting of one crus cerebri and old disease of pia mater. The right optic nerve was wasted but the tracts appeared equal.*

* For further details of this case, *vide* 'St. Barth. Hosp. Rep.' for 1880, vol. xvi, p. 38.

Case 22. *Choroido-retinitis in hereditary syphilis; fibrous membrane between retina and choroid.*—This is, like the last, little more than the description of a post-mortem specimen (No. 142). William G—, was under the care of Dr. Henry Humphreys, for old hereditary syphilis, at the Children's Hospital, Pendlebury, and died in March, 1879; the back of one eye was kindly forwarded to me through Dr. Barlow.

The retina was loosened but apparently only from post-mortem shaking. Between the retina and choroid was a thin tough translucent membrane of considerable extent, broad in front, tapering behind to a narrow string, which was attached at or close to the sclero-choroidal foramen. There was old superficial choroiditis with pigmentation of retina, the pigment being (in the opacified retina) more visible from the outer than from the inner surface; the epithelium of the choroid was almost entirely absent. Microscopically, the membrane was dotted over on its choroidal surface with fine granules of brown pigment, sometimes grouped in patches. Towards its retinal surface were scattered nuclei, usually round and never tailed or vacuolated. The membrane itself had a faintly fibrous structure. There was much pigment around the small vessels of the retina.

Case 23. *Early disease of choroid and vitreous in hereditary syphilis; subsequently fibrous changes in retina and vitreous, &c., suggestive of new growth.*—Hutchinson* gives a case in which a female syphilitic infant lost the left eye with dilatation of pupil and haze of lens (and probably of vitreous also) at the age of a few weeks. The lens became opaque and subsequently dislocated backwards. When the the child was twelve the eye, being irritable, was removed and the opaque lens found imbedded in dense fibrous structure in the vitreous chamber, the retina and choroid being adherent and disorganised. Before excision these changes produced as a yellow reflex

* Hutchinson, 'Ophth. Hosp. Rep.,' vii, 42.

through the widely dilated pupil, but the lens was identified. The child was under care for interstitial keratitis of the other eye when the left was excised.

Case 24.—Vernon* describes a case of double destructive panophthalmitis in a very emaciated male syphilitic infant, seven months old. There was recent inflammation of iris, ciliary processes and choroid, with exudation of a large quantity of whitish puriform substance into the vitreous and between the retina and choroid. The lens was opaque, and thus the case could never have simulated closely a case of glioma.

Case 25. *Both eyes blind, with appearance somewhat like glioma; no cause assigned.*—Charles O—, æt. 18 months. Believed never to have had any p.l., was brought to Mr. Liebreich, at St. Thomas's Hospital, when six months old. Has never shown symptoms of pain. In April, 1878, I found the right eye shrunken, cornea small, yellowish, and semi-opaque; the left eye, no a.c., cornea and lens clear, a yellowish reflex from deep behind lens.

Case 26†.—Chronic plastic choroiditis with degeneration of retina, dense epichoroidal membrane, and masses of cholesterine in the vitreous; lens cataractous. History incomplete; "cataract" was noticed at nine months; the eye was excised because irritable at three years; it was soft with very deep anterior chamber, iritic adhesions and opaque lens. No history of injury.

Case 27 is an instance of buphthalmos but the history of the mode of onset of the eye symptoms seems to ally it with some of the foregoing.

E. S—, the youngest of five children, all of whom are living and reported healthy; there was a miscarriage between numbers two and three. The mother was well whilst pregnant with the patient. He was well till nine weeks old and had learnt to take notice. Then he got a cold and had "bronchitis" and this was soon followed by

* Vernon, 'Ophth. Hosp. Rep.,' vi, 292 (1869).

† Nettleship, 'Path. Trans.' for 1880, xxxi, 253.

convulsions and fits lasting for about three months, during which he would lie as if dead. As he got better it was noticed that he kept his eyes shut in the light, only opening them when shaded. For a time they were blood-shot but there was never any discharge. Gradually the eyes enlarged and he was found to be blind. Was not vaccinated till six months old, *i.e.* not till after the illness. I saw the child when eleven months old (September 1877). There was great enlargement of the cornea with some diffuse haziness, deepening of the anterior chamber, a moderately dilated pupil free from adhesions; the fundus was well-lighted but, owing probably to the corneal haze, no details were visible. T. slightly +. The sclerotic looked somewhat distended. Both eyes equally affected. There was doubtful p. l.

A few months later (Feb. 1878) there was no change. The child was well-grown, could stand and was beginning to talk; head rather nodular, and fontanelle not closed; no rickets; intelligent, but temper bad. No history or symptoms of syphilis. The mother thinks he has difficulty in swallowing from the bottle. She says that when at the breast he would never suck for long together, but liked to hang his head back and cry; and also that he liked to lie on his belly.

In September 1880, when four years old I saw him again. He had become very fond of music, but was still bad tempered. An unsuccessful iridectomy was performed on one eye, vitreous being lost and the globe shrinking.

A short analysis of the foregoing cases gives the following results.

The two *sexes* seem about equally liable to these deep-seated forms of disease. Of twenty-four cases whose sex is mentioned eleven were males, thirteen females.

Symmetry.—Of twenty-six cases one eye only was affected in fifteen, both eyes in eleven, but of the latter number the disease was by no means always of the same type in both eyes.

Age at onset.—The patients varied from three months or rather less to about four years, at the time of onset of the eye changes; in one (No. 25) the child was said to have been blind from birth. Of 24 cases 10 were under one year, and 10 between 1 and 2 years old at the onset; 3 cases were between 2 and 4 years old.

Cause.—Of the entire series no less than 10 patients were the subjects of hereditary syphilis (Nos. 15 to 24, both cases included). In several of these (Nos. 15, 17, 20, 21 and 23) the appearances were quite near enough to those of glioma to require great care in diagnosis. In the others (Nos. 16, 18, 19, 22 and 24) the disease was either a violent panophthalmitis (Case 24) or mainly a very severe type of the more usual syphilitic choroido-retinal affections, and did not simulate intraocular growth. Cases 21 and 22 are remarkable for the formation of very distinct tough membranes between the retina and choroid and in the vitreous, and in one (No. 17) a peculiar, white, muddy, or curdy deposit lay on the choroid. All the syphilitic patients were under two years old when the eye disease began (except perhaps Nos. 21 and 22 where this point is not noted). Of the 10 syphilitic cases both eyes were affected in greater or less degree in no less than 7; but in several of these the disease took the form of an ordinary syphilitic choroido-retinitis in one eye, and in one case (No. 17) this was a great help to the diagnosis in the other eye.

In the cases entered as occurring in non-syphilitic children only 4 were symmetrical (Nos. 10, 11, 25 and 27), and of these the history is incomplete in No. 25, whilst No. 27 does not closely resemble any of the others.

In cases of "pseudo-glioma" of the retina, we may therefore say that precise symmetry of the eye disease or the presence of choroido-retinitis in the other eye, not only makes glioma improbable but points very strongly to inherited syphilis as the cause.

These very severe and disorganising forms of syphilitic eye disease are certainly in my experience much commoner

in the inherited than the acquired disease, and it is worth noting that in several of the cases given above, there were either symptoms or post-mortem proofs of chronic meningeal and cerebral change, of types which are not usual in acquired syphilitis. I am inclined to think that very severe choroiditis early in inherited syphilis is very often accompanied by chronic cerebral disease.

Of the 17 non-syphilitic cases we find that in no less than 11 an acute illness of some kind had occurred shortly before, or with, the eye disease. Of the 6 in which no illness is noted the account is incomplete in 5 (Nos. 8, 12, 13, 25 and 26), and the remaining case (14) is very probably one of injury. Of the 11 cases with a history of illness, (No. 27, and Nos. 1 to 11, omitting No. 8) 5 were in males and 5 in females (sex not noted in No. 10). The age at onset seems not characteristic; in all but 2 (Nos. 1 and 4), the disease began under 18 months of age.

In regard to the nature of the illness with which the ocular changes were associated in these 11 cases, we find considerable variation. Measles was the associated or causing malady in Nos. 3 and 7; in case 7 the patient was premature and the measles occurred as early as 6 months, and in No. 3 the patient was only 10 months old. Bronchitis, with hooping cough in No. 1, with "low fever" in No. 5, with diarrhœa in No. 9, with convulsions in No. 27, preceded the eye symptoms by a longer or shorter time. Symptoms, perhaps pointing to acute cerebral disease, were present in Nos. 2 and 6, and pyæmia was probable in Nos. 4, 10, and 11.

In regard to the cases with decided head symptoms, both in this group and in the syphilitic group, the clinical account is in several instances too incomplete to warrant a certain diagnosis. It is at least remarkable that of the cases with apparently head symptoms (Nos. 1, 2, 6, 15, 16, 20, 21, 27), all but 2 (Nos. 6 and 27) were known to be suffering from some infectious blood-disease at the same time; Nos. 1 and 2 developed hooping-cough and the

other 4 were syphilitic. There seems therefore at present little or no proof that the eye diseases forming the subject of this paper are caused by meningitis, but there is strong reason for connecting them in nearly every case with one or other of the infectious blood-diseases.

(*October* 12*th*, 1882.)

P.S.—In a paper on "Glioma of the Retina," by Dr. U. Vetsch, in Knapp's 'Archives' for last March, some very instructive cases and remarks will be found bearing on the subject of this communication.

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2. *A case illustrating the development of the condition commonly known as pseudo-glioma.*

By W. A. Brailey, M.D.

Lilian B—, æt. 16 months, a pale but tolerably well-nourished child, was brought to the Evelina Hospital on Friday, March 16th, 1883. The right eye was protruding and both lids were swollen and red. The iris was dull, and the pupil fixed and rather small. The tension could not be ascertained with precision. The mother said that on the previous Monday the child was well, but that on the next day it appeared to be in pain, and the eye looked red and swollen. Nine days after the onset of the symptoms the inflammation and proptosis were subsiding. A yellowish reflex from behind the clear lens was visible through the smallish, excentric, and somewhat irregular pupil. The tension was thought to be normal. A week later the appearances were exactly those most commonly recognised in pseudo-glioma, except that the lids remained a little thick and red. That is to say, the eye was a little soft, and looked a trifle smaller than the other; the iris-periphery was retracted, while its pupillary part was pushed forward by the clear lens.

The child has a cough, and remains, even now, feverish at nights, though there are no physical signs of any grave chest affection. The patient was treated when five weeks old for diarrhœa with swollen abdomen; the attendance lasted eight months, and the malady was called consumption of the bowels. Her mother is pale and thin, and suffers through every winter from cough. I think the case to be essentially a spontaneous suppurative hyalitis.

(*May 10th*, 1883.)

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3. *Rapid purulent infiltration of the eyeball through a cystoid cicatrix of two years' standing.*

By P. H. Mules, M.D. (Manchester).

This example of destructive inflammation in which the eye was lost within twelve hours of infection, occurred in a young man, æt. 22, in whom, glaucoma supervening upon myopia, both eyes were iridectomised upwards in March, 1880. No unusual incident occurred during the healing process, and he left the hospital on the ninth day, the cicatrix in the left eye being level, that in the right slightly cystoid. In July, 1882, two years and a quarter after the operation, his sight having improved meanwhile, and his work continuing without interruption, the right eye became hot and painful at bedtime. I saw him the next morning and found a slight purulent discharge from this eye (the one with cystoid cicatrix), the iris was greenish, the cornea bright and clear, but the whole organ evidently in a most critical state. He was at once taken into the hospital and put to bed; in an hour the anterior chamber was full of pus, the conjunctiva chemosed, and the eye was destroyed by panophthalmitis within twenty-four hours of the first symptom of purulent infection.

We meet with cases where a cystoid cicatrix, having long been a source of danger to the eye by repeated inflammatory attacks, eventually causes destruction by a suppurative process; but this is by no means an analogous case, as the eye under consideration had never been uneasy since the operation. I can only look upon it as septic absorption through a cystoid cicatrix. I can offer no explanation why the germs should enter the globe, nor am I aware that the probability of such a contingency has been discussed.

(*December 14th*, 1882.)

V. SYMPATHETIC OPHTHALMITIS.

1. *Five cases of recovery from mild sympathetic ophthalmitis.*

By W. Jennings Milles.

I have grouped together the following cases of sympathetic serous iritis, from the fact that there was a marked similarity in their clinical characters, that the attack of sympathetic inflammation was of a very mild type, and also from their bearing upon the important, and still undecided, subject of treatment.

Case 1. *Wound of cornea and ciliary region; sympathetic serous iritis in twenty-eight days; excision of exciting eye; recovery of sympathising eye.*—Ellen G—, æt. 24, admitted into Moorfields Hospital on May 2nd, 1882. The right eye had been injured the previous day by the bursting of a soda-water bottle. There was a long vertical wound of the cornea, the upper end reaching into the ciliary region. The iris was prolapsed into the whole extent of the wound; the prolapse was at once freely cut away by operation, not more than half the iris being left behind. The left eye was in a normal condition.

On May 16th the corneal wound was completely healed, and there was no entanglement of iris visible.

On the 23rd there was slight hypopyon which disappeared in two or three days.

On the 29th the patient was about to be discharged

from the hospital, as all symptoms of inflammation of the right eye had disappeared, but on examination of the left eye, in the ordinary course of routine, I found serous iritis, as shown by numerous small dots on the posterior surface of the cornea. The pupil was active, dilating fully to atropine. There was slight ciliary congestion, some photophobia, but no pain. Fundus was normal and tension normal.

On being questioned the patient said that she had noticed a slight uneasiness, and a few flashes of light occurring in this eye during the last twenty-four hours.

Excision of the right or exciting eye was performed immediately the sympathetic inflammation became manifest. In the sympathising eye the dots persisted for ten days, and then slowly disappeared. The iris never became visibly affected, and the media and optic disc remained normal. Vision was also normal with correction of a slight error of refraction. She was discharged in about three weeks' time, having been kept during this period in a darkened ward.

I saw this patient again in March of this year (nine months after excision), and found no traces of the preceding inflammation.

Unluckily I have been unable to obtain full notes of the condition of the excised eye. The following were the naked eye appearances. "Sclerotic extremely thin. Vitreous clear. The lens is absent, but the media in the front of the eye are transparent. In the optic disc the vessels running upwards are extremely small, indeed scarcely visible. Those running downwards are also small. The ciliary processes look normal." There are no notes of microscopical examination.

Case 2. *Wound of cornea; prolapse of iris; sympathetic serous iritis in eight weeks; excision of exciting eye; recovery of sympathising eye.*—Frederick R—, æt. 16, a ginger-beer maker, admitted into Moorfields on September 20th, 1882. Ten days previously his right eye and upper lid had been

wounded by the bursting of a soda-water bottle. There was a wound of the cornea passing from the upper sclero-corneal margin downwards and inwards, with a large prolapse of iris. The latter was covered by a layer of lymph. There was also some iritis. The wound of the upper lid, which was of considerable size, was healing.

The left eye was normal; V. = $\frac{20}{20}$ and J. 1.

The prolapsed iris was cut off and freed from entanglement in the wound, with the exception of a small tag at the outer part. The eye remained red and irritable for some time; vision was J. 6.

On November 15th slight ciliary injection of the left or sympathising eye was noticed for the first time. There was a small posterior synechia which yielded easily to atropine. There were also several small dots on the posterior surface of the cornea. Fundus normal and tension normal.

As it was considered a case of sympathetic inflammation the right eye was excised. Four or five days later there was slight blurring of the optic disc, a slight neuro-retinitis, and the keratitis punctata became more marked. The pupil remained well dilated with atropine, no pain was complained of, and there was only slight ciliary congestion. The patient unluckily struck his eye rather severely on December 10th, so that recovery was somewhat retarded. However, by December 24th all symptoms had disappeared and vision, which had never been diminished below $\frac{20}{40}$, was now $\frac{20}{30}$. Treatment consisted in the frequent application of atropine, and rigid seclusion in a dark room with a dark bandage over the eye.

I saw this patient again on February 19th of this year, (three months after excision) and found the eye in every respect in a normal state.

The condition of the excised eye was as follows :—The corneal wound extended some distance into the ciliary region with entanglement of iris. There was a good a. c. An equatorial section of the globe showed that the fundus was apparently normal. Lens clear, fixed at the wound

by a little lymph. Vitreous firm and clear. Slight cloudiness over the ciliary body.

On *microscopical examination* I found that there was marked irido-choroiditis. The iris was much swollen and infiltrated with cells, grouped chiefly round the blood-vessels. In the ciliary body, just beneath the pigmentary layer which was greatly disturbed, were numerous small punctate patches of more acute inflammation, as shown by the excessive accumulation of cells, so numerous in some parts that probably the stage of suppuration had been reached. In the choroid were similar changes not quite so marked and chiefly around the optic disc. The retina was also implicated in these changes, and here and there was a patch of exudation from its internal surface. These changes were scarcely visible in the optic disc or optic nerve.

Case 3. *Wound of cornea; entanglement of iris; sympathetic serous iritis in seven weeks; recovery of sympathising eye previous to excision of exciting eye.*—William C—, æt. 31, an engineer, was injured in the right eye by a piece of steel on May 1st, 1882, returned to work in a few days, but was compelled to give it up in seven weeks' time owing to irritation of both eyes. The first time he presented himself for examination was on July 27th, twelve weeks after the injury.

There was a scar on the right cornea, triangular in shape, not involving the sclerotic, but having a small piece of the iris entangled in it. V. was $\frac{20}{200}$; with +2·5 D. $\frac{20}{70}$. Lens slightly cataractous. T. n.

In the left eye there was well-marked keratitis punctata chiefly in the lower part of the cornea. Pupil active, dilating well to atropine. Media clear. Fundus normal. T. n. V.=$\frac{20}{70}$; with −1·5 D. cyl. $\frac{20}{30}$ slowly.

The patient was kept in the dark, and atropine used for both eyes; at the end of six weeks all the dots had disappeared, both eyes were quiet, and the man was allowed to attend as an out-patient. I have no note

stating the condition of the fundus at the date of discharge.

He was readmitted in December, when the following notes were made.

Right pupil dilates partly to atropine. Striæ in upper part of lens. Anterior synechia as before. With −3 D. cyl. sees $\frac{20}{200}$; irregular astigmatism. T. n. Eye quiet.

Left (sympathising eye) quiet. No remains of dots; with −1·5 D. cyl., axis horizontal, $\frac{20}{20}$. T. n.

As he came a long distance and could not return, it was thought advisable to attempt the removal of the incarcerated piece of iris. The lens was wounded during the operation, and, as the eye remained irritable, even after the swollen lens had been as far as possible removed on a subsequent occasion, enucleation was performed in January, 1883.

Shortly the changes in the excised eye consisted in an entanglement of the iris and some atrophy of the ciliary folds. The vitreous was healthy, as well as the retina and optic disc. Microscopical examination showed that the iris was acutely inflamed and adherent at one point to the cornea, and the lens-capsule was folded up and fixed to the iris. No changes in retina and optic disc.

June, 1883.—Patient seen again. Left eye remains sound.

Case 4. *Puncture of globe; traumatic iritis and cataract; partial extraction ten months later; loss of fluid vitreous; commencing shrinking of globe; excision of eye; sympathetic serous iritis three weeks after excision.*—Edith S—, æt. 17. In the early part of April, 1882, she "pricked the pupil of the right eye" with a needle. For the following fortnight she suffered a good deal of pain. The eye, however, gave her no trouble afterwards.

On admission into Moorfields on January 18th, 1883, the condition of the right eye was as follows.—Good a. c., pupil acts, but best upwards, some very fine posterior synechiæ; traumatic cataract. V.=hand reflex. T.n. Left eye: V.= J. 1 at 12″, with −3d. $\frac{20}{20}$, p. a., T.n.

January 19th.—Modified Graefe's extraction performed on the right, followed by the loss of a considerable quantity of fluid vitreous.

26th.—The anterior chamber filled with blood. Later on the tension of the eye began to diminish. Excision was performed on February 3rd, fifteen days after extraction. At this date the left eye was perfectly quiet.

She was readmitted on March 3rd with a history of irritation and some pain in the left eye which had begun thirteen days previously, that is thirty-five days after the extraction and twenty-two days after the excision.

In the left (sympathising eye) there was keratitis punctata. The dots were very small; the pupil was active, dilating well to atropine; fundus normal; media clear. V. $= \frac{20}{20}$ with $-$ 3·50 D. The symptoms did not become more severe, but slowly diminished, till at the end of three weeks all the dots had disappeared. Treatment consisted in keeping the patient in a dark room, and in the constant application of atropine to the eye.

The excised eye showed the following changes:—The globe was commencing to shrink. T. -1. The lips of the corneal section were not healed superficially. No other scar visible in the cornea. Iris much discoloured; a. c. shallow; pupil blocked with exudation and slightly drawn up. On making an equatorial section of the globe, the vitreous was found to be fluid, the ciliary processes were normal above, but covered below by a fan-shaped cicatricial mass. The retina reaching backwards from this mass was detached and puckered. The optic disc looked normal.

The microscopical appearances were as follows:—A marked proliferation of epithelial cells in the superficial part of the corneal wound. The cells spread in columns into the cornea, producing an appearance not unlike papillæ. The iris was greatly thickened and its whole substance was crowded with cells. The cells were arranged in isolated groups in the ciliary body and also, but not so markedly, in the choroid. The retina was

greatly swollen, especially in the region of the optic disc. The swelling was chiefly due to accumulations of cells in the middle layers.

Case 5. *Perforating ulcer of cornea; shrinking of globe; sympathetic serous iritis two years afterwards; excision of exciting eye; recovery of sympathising eye.*—Eliza M—, æt. 29, admitted into Moorfields Eye Hospital on August 18th, 1882. Five years previously had been laid up with a severe attack of rheumatic fever. Two years ago suffered from inflammation of the left eye. No history of a blow. She had since had no pain or uneasiness in either eye till three weeks ago, when the right eye began to become dim.

The condition of her eyes on admission was as follows:—

Left eye shrunken, tender on pressure, especially at the outer side. Cornea flattened and vascular; pupil dilated and fixed; lens cataractous. T.−2. No p. l.

Right (sympathising) eye: keratitis punctata; pupil active, dilating to atropine; well-marked optic neuritis. T.n.

The left eye was enucleated.

On August 31st the condition of the right eye had not materially altered.

On September 7th the keratitis punctata had diminished, but optic neuritis was still present.

The vision was not noted.

She was discharged at the latter end of September with the eye quiet, but still with some optic neuritis.

She presented herself again in May, 1883 (nine months after excision), having used atropine almost continuously since leaving the hospital. She had had no return of inflammation of the eye, which was in a perfectly normal condition, with active pupil, normal acuteness of vision, and no appearance of optic neuritis.

Were the symptoms in these cases sufficiently distinct to include them under sympathetic ophthalmitis, or was

the serous iritis simply a coincidence? These questions are naturally obvious ones. I can only say that I inquired very carefully into all the histories, and could find no account of previous affections of the eyes. The patients were all healthy individuals, with no special diathesis, and with good family histories; except in Case 2, where the father had died probably of phthisis, and two sisters during childhood, one of some bone disease. There are other arguments in favour of sympathetic inflammation. First, that in all the cases there was a wound in a position known to be specially liable to produce the disease, and secondly that the serous iritis occurred only in one eye, and that the unwounded one.

In Case 1 the dots on the cornea were so very minute as to be scarcely visible even by most accurately focussing them with oblique illumination, and I was only able to see them distinctly by direct examination with a high convex glass (+24 D.) behind the ophthalmoscope. The dots then stood out as distinct black specks on the background of the illuminated fundus.

From the mildness of the general attack, from the fact that in Case 1 no complaint whatever was made and that the disease was discovered almost by accident, I think it is not at all unlikely that cases similar to this one may be passed over unnoticed.

They also illustrate some points in the treatment in sympathetic inflammation. Mauthner in his treatise on sympathetic diseases lays down the law that *we must not excise in serous iritis,* his reason being that excision of the exciting eye converts a serous into a plastic iritis.

My first two cases, however, show that excision in serous iritis, at all events does no harm, as the patients made an uninterrupted and rapid recovery. It might be even argued that by the very act of excision, the sympathetic inflammation was arrested or greatly modified, thus accounting for the mildness of the symptoms.

But Case 3 is undoubtedly opposed to this last view of the matter; for here excision was not performed, and yet

the sympathising eye completely recovered. In this case excision was eventually had recourse to, but many months after all appearances of sympathetic inflammation had disappeared, and on account of failure of an operation on the exciting eye. It seems to me therefore, that arguing from these cases, excision is justifiable in sympathetic serous iritis, but that this proceeding will have no effect in modifying or arresting the progress of the disease. I think, however, that the final result will be much more favourable, if by the constant use of atropine and by keeping the patient rigidly in the dark, all sources of irritation are withdrawn from the eyes.

No. 4 is another addition to the series of cases of sympathetic inflammation occurring after excision collected by Mr. Nettleship. It is also interesting from another point of view. Excision of the eye was performed 15 days after the extraction, on account of failure of the operation. Sympathetic inflammation was noticed 22 days after the excision, or 35 days after the extraction, and ten months after the original wound. These intervals show with very little doubt that the exciting cause was the operation of extraction, and illustrate the short period that may intervene between an injury and the first changes that ultimately lead to sympathetic ophthalmitis.

In conclusion, I should say, that I am indebted to the members of the surgical staff at the Moorfields Hospital for allowing me to make use of their cases for the purposes of this paper.

(Micro-photographs were exhibited showing the principal pathological changes in the excised eyes.)

(*May* 10*th*, 1883).

2. *A case of severe sympathetic ophthalmia completely recovered from.*

By W. Bowman, F.R.S.

On 12th October, 1868, Louisa F—, a healthy child of six years, fell while carrying a plate, which being broken, cut the forehead and eyelid slightly. The eye itself was not thought of. No medical man was called in for two weeks, there being no pain. My friend Mr. R. W. Wilcox, of Aylesbury, then saw her, and found a horizontal cut through the cornea and ciliary region of the right eye, with the iris largely protruding. He removed this prolapse, but when, in a few days, the aqueous was restored, further prolapse occurred. After three more weeks, viz. on the 17th November, Mr. Wilcox, not having seen her for a week, found the other, or left, eye inflamed, and sent her to me. On the 18th I found the wound bulging; there was moderate congestion of the globe, though the cornea and aqueous were clear, and the iris was nearly natural in aspect. Pupil small, motionless, drawn behind the cicatrix, −T1? V.=J20. The left eye was generally injected, with large vessels near the cornea; the anterior chamber was rather deep, the diameter of the pupil varied, under changes of light, between 4 and 6 mm., there being no synechia. −T1. V.=J16. I excised the wounded eye the same afternoon, and ordered continual exclusion of all light from the left.

The next day, the 19th, eighteen hours after the excision of the right, the child was evidently relieved and merry. Tn., and continued to be Tn. on each of many following days.

On the 23rd, the conjunctiva and sclerotica were perfectly free from injection, but on slight exposure to light for purposes of examination the ciliary vessels, especially

below, became a little injected, and the eye watered. V. =J2. Atropine was now used regularly, but the pupil dilated only to a diameter of 4 mm., being slightly held to the lens by effusion.

On the 24th, pupil free, 7 mm. in diameter. Tn.

25th.—Pupil 9 mm. V.=J2.

28th.—Atropine having been intermitted pupil contracted to 4 mm. V.=J1. But there was rather more ciliary congestion, especially of the perforating anterior ciliary arteries. Atropine was now constantly used.

30th.—Ciliary region redder, especially along a circle about 3 mm. from the cornea, and the conjunctiva reddened generally, with lacrimation, when the eye was exposed to light. It was thought better not to test the vision during the ensuing weeks.

On December 2nd, the pupil was adherent at its lower edge, and the lower half of the cornea was finely dotted on its posterior surface.

3rd.—Less redness. Continue the atropine.

12th.—Till to-day injection has been lessening and Tn. uniformly, but now more general redness, and −T1. Also more haze of cornea and chamber. Treatment: darkness and atropine (gr. ij—℥j) more frequently.

21st.—Many synechiæ all round; pupil 4 mm. Tn.

24th.—Yesterday more injection without obvious cause; now lower half of the ciliary region injected, the chief redness being at a little distance from the cornea. The two lower perforating arteries dilated and tortuous (atropine gr. iv—℥j).

29th.—For two days much better. The ciliary vessels much emptier, and now not very obvious.

January 4th, 1869. Improving. Dull reflex by the ophthalmoscope. Iris thrown into plaits below (see Fig.). Anterior chamber otherwise normal; aqueous clear; dots on the cornea less apparent. With $+\frac{1}{9}$ V.=J14.

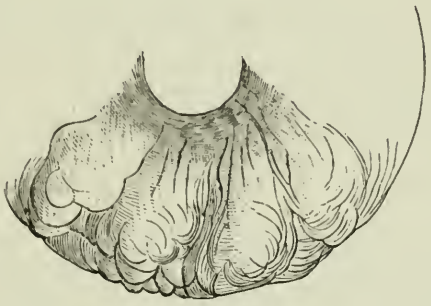
14th.—Continued absence of vascularity. Only the two perforating ciliary arteries rather too full; corneal dots fainter. The iris is now, in all its circle, and from

its great circumference half way to the pupil, or rather more, thrown into folds bulging towards the anterior chamber, and showing accumulation of fluid behind it in closed pockets formed by partial uveal adhesions to the lens-capsule. Atropine gr. iv—ʒj, ter die.

21st.—Improving.

27th.—Progressing slowly. With $+\frac{1}{9}$ V. = J6.

February 4th.—Sclerotica now nearly white, its vessels



Iris thrown into bulging folds by fluid collected behind it.

hardly seen. Sight said to be "much clearer."

11th.—The eye has reddened for some hours after violent exercise in running about the park, and perhaps some exposure to light.

25th.—Corneal specks less.

March 4th.—Cornea clearer. With $+\frac{1}{9}$ V. = J8.

11th.—Reflex still dull, but better than on January 4th. An irregular circle, 4 mm. diameter, of fine pigment dots on lens. Diameter of pupil, 5 mm.

April 1st.— Tn. No redness. Only a few fine brown dots on back of cornea. No puckering of the iris now. A faint circle of pigment on capsule within the line of the dilated pupil.

May 3rd.—With $+\frac{1}{9}$ V. = J4 at 5″. Iris in its normal plane; only the faintest remains of specks on cornea.

August 9th.—No spots on cornea, only a faint haze seen by lateral illumination. The iris flat, except at one spot below at the great circumference, which possibly escaped observation in April and May. Pupil dilated to

to 5·5 mm. Apparent uveal adhesions at its margin. Sclerotic and ciliary vessels are quite normal. With $+\frac{1}{9}$ V.=J2 at 5″. In May had scarlatina, and eye became temporarily redder, but immediately yielded to atropine, which has been continued to the present time. Continue atropine once a fortnight.

November 24th.—Pupil under atropine dilates to 6·5 mm. Iris looks perfectly natural. No appearance of any synechia, and none of pigment on the lens. The fundus normal. Repeat atropine once a month.

1870, May 18th.—Tn., pupil active. V.=J2 at 12″.

October 29th.—Eye seems well.

1872, November 9th.—Eye reddens a little. Some myopia coming on When corrected with $-\frac{1}{28}$ V=J18 at 15′.

1873, March 17th.—No redness. Pupil free and active. Artificial eye ordered. M.=$\frac{1}{20}$, and V.=J18 at 15′.

1874, July 22nd.—Well. V.=J1 at 10″, and with $-\frac{1}{18}$ V.=J19 at 15′.

1881, March 23rd.—Eye perfectly comfortable and well; pupil active. On the front of the lens, up to the anterior pole, chiefly on the lower and outer part, a very fine dusting of brown pigment, not in patches, could be seen by careful examination under lateral illumination, but not by transmitted light. V.=J1 at 10″: with $-\frac{1}{28}$ V.=J2 at 14″, and with $-\frac{1}{9}$ V.=J18 at 15′. No astigmatism. Ordered $-\frac{1}{28}$ for reading, and $-\frac{1}{9}$ for distance, in reversible frame.

(*May 10th*, 1883.)

P.S.—1883, July 10th.—Mr. Wilcox replies to my inquiry:—"Eye in as good condition as one could wish. Iris healthy in appearance. Pupil round and free. After reading and doing fine needlework there is a feeling of fulness at the back of the eye, and that is the only complaint that I could induce her to make."

3. *A case of sympathetic ophthalmitis; good result; exciting eye not excised.*

By W. Adams Frost.

Louisa S—, æt. 10, admitted into the Royal London Ophthalmic Hospital under the care of Mr. Waren Tay (by whose permission I bring forward the case) November 27th, 1881. Thirteen days previously (November 11th) the right eye had been wounded by a thrust from a pair of scissors.

On admission, R. E., wound of the inner and lower quadrant of the cornea, reaching to, but not beyond, the sclero-corneal junction. Iris prolapsed into the wound. Lens opaque and swollen at lower part. No operation was performed and the patient was discharged in about four weeks.

Seven weeks after the accident the lens was to a great extent absorbed; the pupil was pear-shaped and displaced towards the cicatrix, to which the iris adhered; no conjunctival injection; no symptoms in the other eye.

Eight weeks and a half after the accident the father noticed slight injection of the sound eye, and dimness of vision was complained of. On the following day the patient was readmitted. Her condition was then as follows:—Right eye as above, except that on the cornea were "several well-marked dots,"* and there was a very slight ciliary blush in the region of the wound. No tenderness. L. E.: faint circum-corneal blush, numerous fine opacities on the lower half of the cornea; the pupil dilated well to atropine; the media were slightly hazy, and the margins of the disc obscured, "great hyperæmia of disc scarcely amounting to optic neuritis."* Treated by

* The words in inverted commas are quoted from the notes of Mr. W. Jennings Milles, who was house surgeon at the time.

exclusion of light and the use of atropine. The right eye was not excised.

Thirteenth week, left cornea clearer. Well-marked papillitis. V. $=\frac{20}{50}$.

Seventeenth week, made an out-patient, but atropine continued.

Twentieth week.—Atropine has been used continuously since the appearance of the symptoms. Pupil dilated fully; cornea quite clear; papillitis unchanged.

V. with $\frac{+1{\cdot}5 \text{ cyl.}}{+2 \text{ sph.}} = \frac{20}{20}$. Atropine discontinued.

Thirtieth week.—Has not been seen since last note. Numerous tough adhesions are now present all round the left pupil. Cornea clear; there is no conjunctival injection, and none has been noticed by the father, who is an intelligent man; disc as before. Atropine was now used continuously for four weeks without any effect on the synechiæ, and the disc remained unchanged. During this stage in the case a fresh crop of opacities appeared on the cornea, but these again disappeared a few weeks later.

Fifteen months after the accident, when all treatment had been abandoned for seven months, the following was the condition.

R. (wounded) eye: no pain, tenderness, or conjunctival injection; cornea clear; pupil partially occluded by lens-matter and capsule; V. with +15 D. = 16 J.

L. (sympathising) eye: no conjunctival injection; a very small number of dots on the cornea; pupil as before; margins of optic disc very blurred, veins distended and tortuous. V. with glasses $\frac{20}{30}$ and one letter of $\frac{20}{20}$.

(*May 10th*, 1883.)

P.S.—August 20th, 1883.—There is slight improvement in the vision of the left (sympathising) eye; the other conditions remain unchanged.

4. *A case of sympathetic ophthalmitis; operation on the exciting organ; recovery of both eyes with nearly perfect sight.*

By Simeon Snell (Sheffield).

William S—, æt. 36, was engaged on October 5th, 1882, mending a "sleeve" (*i. e.* a bag through which wheat is shot in a mill), when the band broke and the packing-needle ran into his left eye. He went to a medical man, who five days after the accident commenced to caustic the eye.

On October 20th he came under my care at the Sheffield General Infirmary. It was evident that the packing-needle had penetrated the sclero-corneal junction, at its lower and inner part, and that the wound almost encroached upon the ciliary region. There was a fair sized prolapse of iris, the pupil being altered as if an iridectomy had been performed. The lens was uninjured. There was no precise note made as to the vision of the wounded (left) eye, but the patient could see fairly well with it. No complaint was made respecting the right eye, except that after each application of the caustic it had seemed "misty." Atropine was prescribed to the left (injured) eye and a compressive bandage.

Nov. 3rd.—The left eye is about the same; perhaps the prolapse may be a little smaller. Complaint was made at his last visit, two or three days ago, that the right eye was somewhat "misty," and to-day he states that the "mist" in front of the eye is denser. The ocular surface is reddened, the iris has lost its lustre, and the pupil dilates hardly at all to atropine; plastic iritis. He was admitted an in-patient, and atropine was ordered to be used for both eyes.

5th.—Right eye worse; the pupil has dilated hardly at all, and there is more evidence of effusion round the

pupillary margin. The left eye is unaltered. The following day the right eye was reported as being again worse.

The condition presented in this case appeared very grave. Sympathetic iritis was undoubtedly set up, and was apparently increasing in severity. A consideration of the case suggested the propriety of attempting to release the prolapsed iris from its entanglement, and to reduce the tension on that membrane and the ciliary nerves. With this object on November 6th the prolapse was punctured with a Graefe's cataract knife; the instrument having been entered at one side was carried straight across it, completely severing in this manner the thin covering over the displaced and entangled iris. As much of this membrane as could be seized with forceps was then withdrawn and excised. The iris was, however, so incorporated with the other tissues that only a very little could be removed, and this it was merely possible to do piece meal. The incision was then prolonged in the sclerotic on either side, just behind the sclero-corneal junction (sclerotomy), the aqueous chamber being freely opened. No anæsthetic was administered. The atropine was continued for the right eye. The next day the patient himself observed on the improved condition of his right eye, and it was soon noticed that the pupil dilated evenly and well. There was no return of the prolapse in the left eye.

13th.—Vision of the right $=\frac{20}{40}$; left (injured eye)$=\frac{20}{100}$. The media in the right are clear; the pupil dilates well and a concentric layer of pigmented dots are visible on the lens-capsule, marking the old iritic adhesions. There is no redness of the ocular surface.

19th.—Vision of right$=\frac{20}{30}$; left$=\frac{20}{50}$. The pupil of the right eye dilates to-day a little more irregularly. Perchloride of mercury in a mixture was prescribed, and hyoscyamine drops were ordered for the right eye. The prolapse of iris in the left eye has completely subsided.

December 3rd.—The pupil is widely dilated and there are no iritic adhesions.

During the next few weeks he was kept under observation and then allowed to return to his employment.

April 13th, 1883.—He states he has been enabled to follow his employment as well as formerly. With either eye V.$=\frac{20}{20}$ fairly. The tension of both eyes is good.

The case just related presents features of interest and points worthy of remark. Firstly, may be mentioned the excellent vision recovered in the exciting eye, and the subsidence and disappearance of the disease in the sympathising organ, with the return of practically perfect sight. The importance of being on guard, especially when in a case of sympathetic ophthalmia the exciting eye still retains useful vision, has frequently been pointed out, as well as the fact that the injured globe may possibly eventually become the better organ of the two.

Authors discuss fully the various means of treating the sympathetically affected eye and dilate upon the necessity of enucleating the exciting eye, or in other cases of performing ciliary or optico-ciliary neurotomy. Little mention is, I fancy, generally made of the practicability, in certain cases, of any procedure, having for its object the relief of the sympathetic disease, and at the same time the retention of vision in the injured eye.

In Mauthner's monograph on sympathetic diseases,* however, the following passage occurs :—" Are we to perform iridectomy on the eye which causes sympathy? Under one circumstance only ; when the iris (the eye being otherwise unharmed) has become incarcerated in the peripheral wound in the cornea after an injury or operation, as well as after spontaneous perforation of the cornea. In such cases we may have neuralgia of the eye first affected, or sympathetic inflammation of the second eye. Iridectomy is then of great benefit, for by this operation we can abscise the imprisoned bit of iris as well as the crushed ciliary nerves, and succeed in saving both eyes from danger. But when the incarceration of the iris has already induced

* 'The Sympathetic Diseases of the Eye,' American translation, by Dr. W. Webster and Dr. Spalding, p. 189.

irido-cyclitis, or when the latter affection has originated from any cause whatever, iridectomy is of no avail and cannot in any respect be advantageously resorted to as a substitute for enucleation."

At the time the case I have related came under my care, I had not read Mauthner's opinion just quoted. But one naturally hesitated to entertain the question of enucleating a globe possessing good vision. Without entering into the theories, as to the causation of sympathetic disease, in this case the direct origin of the affection of the second eye seemed to lie in the prolapse of iris, with the entanglement of its nerve filaments in the wound.

To seek a relief to these conditions appeared reasonable and the effect of the operation was immediate and unmistakeable. Conclusions cannot, of course, be drawn from one such case. It may, however, be regarded as instructive and encouraging.

As an instance of recovery from sympathetic ophthalmia the case may perhaps be considered of interest. The disease had not, however, assumed the malignant type (irido-cyclitis) from which recovery is rare.

(*July 8th*, 1883).

VI. DISEASES OF CRYSTALLINE LENS AND CAPSULE.

1. *On the growth of the crystalline lens.*

By Priestley Smith (Birmingham).

The present position of knowledge concerning the growth of the crystalline lens cannot, I think, be stated more concisely, or on better authority, than by quoting a passage from the chapter by Otto Becker in the Handbook of Gräefe and Saemisch.

"In the new-born child the lens is smaller than in the adult, and of a more globular form; it is clear as water, very soft, and nearly equally soft in all its layers. The comparison as to size is true, however, only of the equatorial diameter, for according to Jaeger the antero-posterior diameter is equal, or very nearly so, to that of the adult lens. The changes which take place during life are referable to two opposing processes.

"So long as the whole individual grows, that is up till about the twenty-fifth year, new lens-fibres are laid down at the equator and apply themselves, as they further develop, to the anterior and posterior surfaces, each fibre bending round the equator. Hence the lens continually increases in its transverse diameter, and gradually loses its spherical form. The addition of the new fibres at the equator has little influence on the thickness of the lens, because it is only when the fibres have attained their maximum length that they reach to the poles. Thus the surfaces of the lens assume a flatter curvature and the dioptric power diminishes.

"The slight increase in thickness which would never-

theless occur is nullified to a great extent by the peculiar process of sclerosis which affects the fibres of the lens progressively from earliest youth until old age, and which ceases, unless pathological processes intervene, only with the death of the individual.

"The sclerosis of the lens-fibres is the analogue of the hardening of the cuticle, only that while in the cuticle the older cells lie on the external surface, in the lens they are constantly driven inwards towards the centre by those more newly formed. In other respects the analogy is complete. The younger lens fibres are thicker, softer, richer in water, freer from colour; the older they grow the more they give up their water and become flat, closely compacted, and amber-coloured. And since the centrally situated fibres are for the most part the oldest, the sclerosis begins at the centre of the lens and progresses thence towards the surface."

To this description of the life history of the lens I am able, as the result of an investigation carried out during the last two years, to add another fact, namely that *the growth of the lens does not cease with that of the rest of the body, but is continuous, unless morbid processes intervene, throughout the whole period of life.*

The first intimation of this fact was given in a paper read at the Cambridge meeting of the British Medical Association.* The data then obtained were too few to justify any general conclusion, and a systematic investigation was commenced shortly afterwards.

156 lenses removed after death from the eyes of 91 persons have been examined. Each lens was accurately weighed, and immediately afterwards measured as to its volume by means of an apparatus specially devised for the purpose. I am indebted to Prof. Donders for the suggestion which led me to weigh the lenses. Weighing affords an easier and a more accurate means of estimating quantity than does linear measurement, but in the case of bodies like the lens, the specific gravity of which is not

* Published in the 'Roy. Lond. Ophth. Hosp. Reports,' vol. x, part i.

constant, it does not suffice to indicate the volume. In addition to weighing, it was necessary for my purpose either to ascertain the specific gravity of each lens, or to measure the volume by some other method. I chose to measure the volume directly rather than to calculate it from the specific gravity, because in this way each lens would be examined by two entirely independent processes.

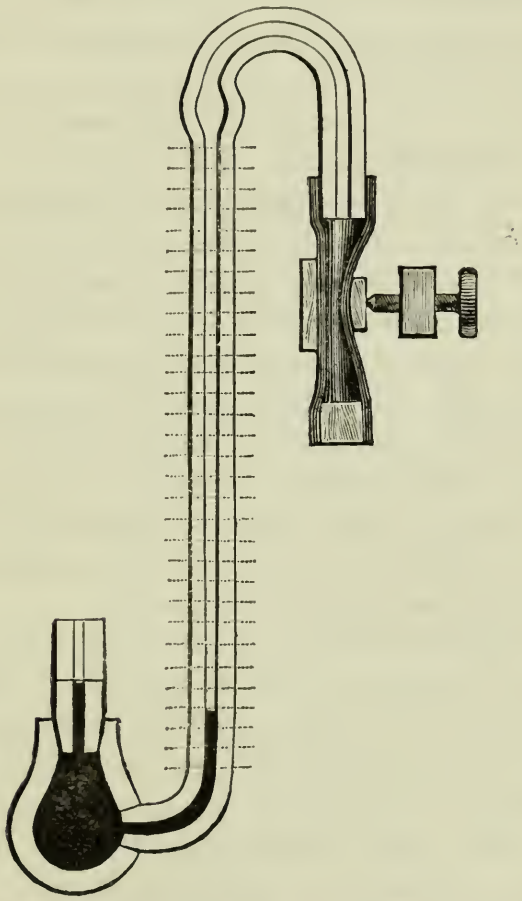


FIG. 1.—Apparatus for measuring the volume of the crystalline lens.

The measurement was effected by displacement of fluid along a graduated glass tube. This tube (see fig. 1) terminates at its lower end in a bulb closed by a perforated glass stopper very accurately ground in; its upper end is connected with a simple suction apparatus consisting of a

piece of india-rubber tube plugged at its free extremity and compressed by means of a screw.

The instrument contains rectified oil of turpentine sufficient to fill the bulb and the lower part of the graduated tube. By means of the suction apparatus the fluid can be drawn from the bulb into the vertical tube or driven in the opposite direction at pleasure. A bulbous expansion in the glass tube at its upper end obviates the danger of drawing the fluid over into the descending arm, and increases the amount which can be withdrawn from the bulb. The cubical content of the graduated tube, as ascertained by experiment is, 2·24 cubic millimetres for each mm. of length. A measurement is made as follows :—The fluid is driven exactly up to a fine line on the glass stopper, and the height of the column in the graduated tube is noted down. The screw is then reversed and the fluid drawn up the tube so as to clear the stopper and the upper part of the bulb. The stopper is then removed, and the lens dropped into the bulb ; the stopper is replaced, the fluid is again driven exactly up to the mark on the stopper, and the height of the column is again noted down. The difference between the present height and the former height, in millimetres, multiplied by 2·24, equals the volume of the lens in cubic millimetres.

Several possible sources of error have been taken into account and overcome as far as possible. Thus, changes of temperature occurring during the making of a measurement would vitiate the result by altering the volume of the turpentine. A thermometer is attached to the instrument in order that the absence of such changes may be definitely ascertained. Again, adhesion of the fluid to the internal surface of the graduated tube causes a considerable error if the column be lowered rapidly and irregularly. To obviate this the head of the screw is divided round its circumference by eight equi-distant notches and is turned in all cases at the rate of one notch in a second. The instrument has been crucially tested, with the help of Professor Poynting of the Mason Science College, by

measuring a series of small bodies of known volumes. The average error was about ·5 cubic mm., the maximum was less than 2 cubic mm. For the purpose in hand, errors of this amount are of little importance.

Immediately after each lens had been measured in the way described, it was laid in a shallow vessel of water and at once measured as to its transverse diameter by means of finely pointed spring compasses. When, as happened several times, the diameter was unequal in different meridians, the mean was stated. In my first few observations this measurement was omitted. I knew that it could not be relied on to accurately represent the diameter of the living lens, but being advised by Mr. Bowman to make it notwithstanding, I did so in all subsequent cases, and the results have a very decided value.

The interval between the removal of the lens from the eye and its examination was as short as possible, rarely more than an hour, generally much less, and special precautions were taken to prevent loss or gain of bulk by evaporation or absorption of moisture. Shreds of the suspensory ligament were removed as cleanly as possible. When the capsule ruptured, and this happened frequently in the earlier cases, the specimen was discarded.

From the age of 20 up to that of 70 I have examined more than twenty perfectly transparent lenses in each period of ten years. Beyond 70 years of age my opportunities have been less frequent, and a large proportion of the lenses examined have been imperfectly transparent, so that the desired number of twenty clear lenses in each decade has not yet been reached beyond the age of 70. The whole of the observations are recorded in the tables appended to this paper. In every case in which any opacity was observed in the lens the fact is stated in a foot-note; the corresponding figures are given in a different type, and are not included in the averages. I now ask attention to Table I, which presents the averages only (see p. 92).

Column A shows that the *average weight* of the lens

increases considerably in each decade. Between 20 and 29 years of age it is 174 mgr.; between 60 and 69 it is 240 mgr. This is a gain of 66 mgr. in forty years, and as the rate of gain is not very unequal in the different decades, we may say, speaking roughly, that the weight of the lens increases at the rate of about 1·5 mgr. each year.

The fact that the lens becomes heavier with the advance of life is, I believe, not new. Bader makes the statement that the lens of a person aged 70 is nearly as heavy again as that of a person aged 20.* But the increase appears to have been attributed to a change of density rather than of size. This, however, is incorrect, as the next column in the table proves.

Column B shows that the *average volume* increases in nearly the same proportion as the weight, viz. from 163 cub. mm. in the first decade, counting from the twentieth year, to 225 in the fifth. This is an increase of 62 cub. mm. in the forty years, so that, speaking roughly, as before, we may say that the volume of the lens increases at the rate of about 1·5 cub. mm. each year. In order to present this fact in a graphic form I have prepared a chart (see p. 85) which shows the volume of every lens examined, together with the age of the subject from which it was obtained. The dots represent lenses which were perfectly transparent; the circles represent those in which more or less opacity was observed. Omitting these latter from present consideration, it is obvious that the individual volumes vary considerably in each decade, but that the whole group moves steadily upwards even to extreme old age.

Column C shows the average *specific gravity* in each decade. It must be remembered that the figures do not represent direct measurements of the specific gravity, but are obtained by calculation from the weights and volumes shown in the preceding columns; their accuracy is there-

* 'The Human Eye,' &c., p. 268.

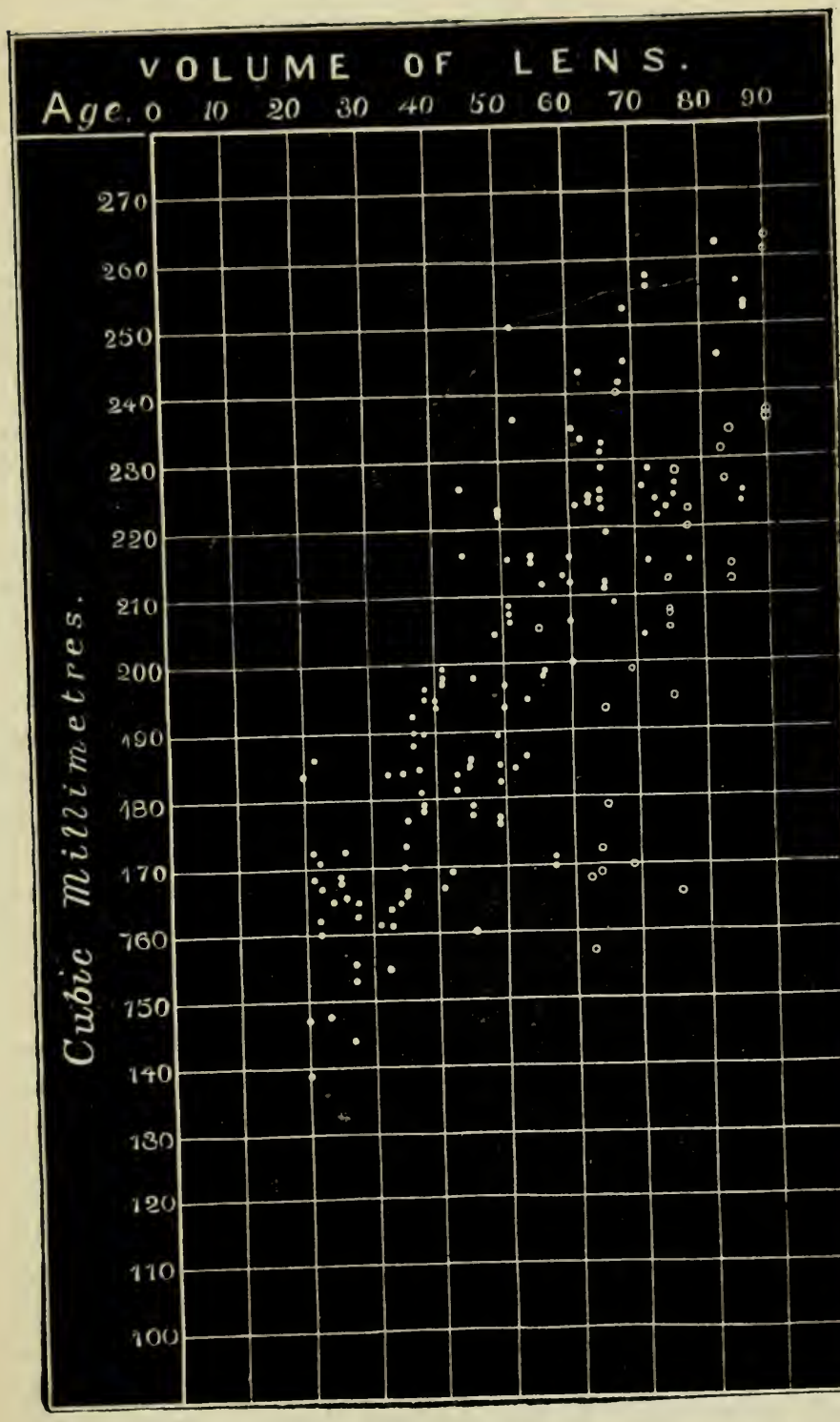


Chart showing volume in cub. mm. of each lens examined, and age of person from whom it was taken.

fore dependent upon the accuracy of these latter. The specific gravity of each lens was calculated separately, and is recorded separately in the tables. There is a considerable difference in different lenses, but in this respect age appears to exert no constant influence, for while there are lenses of high specific gravity and lenses of low specific gravity in each decade of life the average remains nearly constant throughout. The figures show a slight rise from 20 to 40, and a slight fall from 40 to 60, but the changes are too small to be accepted as positive; a very slight alteration in the corresponding weights or volumes would reverse them. The only inference which can be safely drawn from this column is that the specific gravity of the lens is, on the average, nearly the same at different periods of adult life. The continual increase in the weight of the lens is clearly not due to a change of density, but to an increase of volume.

I have not been able to find many published statements concerning the specific gravity of the human lens. Nunneley ascertained it in a few cases, and the average was 1112, but this can only be taken as a very rough estimate, for the observations were only four in number, and there were considerable differences even among these. Chevenix, as quoted by Nunneley, puts the average at 1079; this accords very closely with my own results.

Column D shows the *average diameter* in each decade. Like the weight and the volume it continually increases. With regard to this column a special source of error has to be noted. When the lens is freed from the traction of the suspensory ligament it undoubtedly alters its form to some extent, the alteration being towards the spherical, and being greater or less according to the elasticity of the lens. It is probable, therefore, that the diameters here given are slightly too small for the living eye, and that the error is greater in the earlier decades than in the later. If, therefore, something must be added to each, and rather more to the younger than to the older, the real increase in transverse diameter during the forty

years will be rather less than that indicated by the figures.

The antero-posterior diameter was not systematically measured in my investigations; the measurement is more difficult to make in a trustworthy manner than that of the transverse. The impression which I derived from mere inspection was very decidedly that the older lenses were larger in all ways than the younger, and in several instances in which I was able to compare a young and an old lens side by side, there was a more obvious increase in the anteroposterior diameter than in the transverse. Fortunately it is not necessary to rely upon this impression, for the data now before us afford some evidence on the point.

The volumes of spheres vary as the cubes of their diameters. If the crystalline lens were a sphere of constant specific gravity we could calculate its diameter, at each period of life, from the ascertained volume. The same is true of bodies which, like the lens, are not spherical, provided the *form* is alike in all, *i.e.* provided the several diameters vary in equal proportions. Now, it is quite certain that the form of the lens is not alike in all cases; there are individual variations, as in all living structures, and the average form probably is different at different periods of life. But, for the sake of experiment, I have supposed for a moment that the form remains unaltered throughout life, and I have calculated, in the series before us, what the increase of transverse diameter would then be in each decade. These calculated diameters are set down in column E. On comparing columns D and E it appears that the actual increase, obtained by measurement, is on the whole not greater than it would be if the lens enlarged in equal proportion in all diameters. This is a proof that the increase is not limited to the transverse diameter.

Figure 2 shows in a diagrammatic way the relative sizes of two average lenses, aged 25 and 65 respectively. It is not to be supposed that it represents the exact form

of the average lens at either age. It simply presents to the eye transverse sections of two imaginary lenses identical in form, but differing in volume, in the proportion of 163 to 225. The diameters of the figures vary as the cube-roots of these volumes, viz. as 8·67 to 9·65. For the sake of comparison the outline of the smaller lens is inscribed within the larger, the zone external to this line thus represents the addition which is made during the forty years. The volume of the added part equals rather more than one third of the smaller lens.

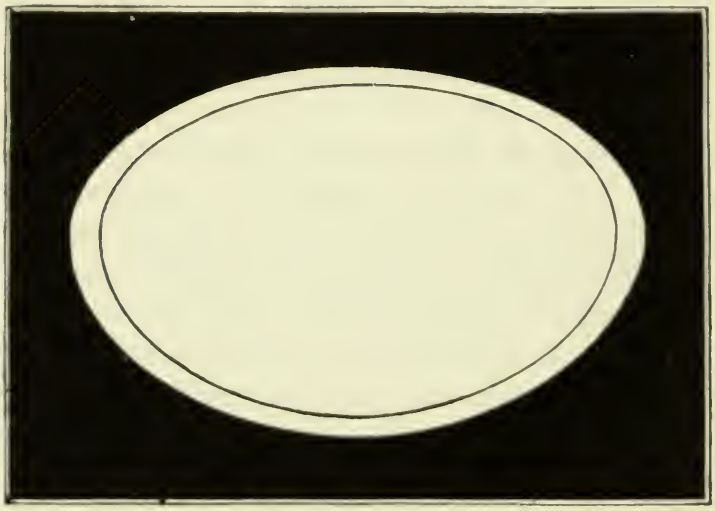


FIG. 2.—Diagram showing size of crystalline lens at the age of 25 (inner line) and 65 (outer boundary).

If we look to the statements which have been published concerning the dimensions of the adult lens, we find very considerable discrepancies. In Merkel's table in the Handbook of Graefe and Saemisch, which places side by side measurements of the several parts of the eye made by many observers, the transverse diameter of the lens is given variously from 8·1 mm. to 10·3 mm., average 9·3; the antero-posterior diameter from 3·6 mm. to 5·5, average 4·25. These discrepancies are now intelligible enough in view of the continual enlargement of the lens; the averages have little value, as they are not assignable to any particular period of adult life.

I have not included the last two decades in the foregoing analysis, because the observations are too few to afford trustworthy averages ; it will be seen, nevertheless, that the increase in weight and volume is continuous in these also. In the last decade but one the increment is small by comparison with that belonging to every other period ; a reference to Table VII shows that the lenses in question belong almost exclusively to the earlier years of the decade.

The continuous growth of the lens being thus established as an anatomical fact, it is interesting to regard the matter from a physiological point of view. The lens is derived by development from the cuticular epiblast ; its growth, as pointed out in the foregoing quotation from Otto Becker, is closely analogous to that of the cuticle. But its cells, unlike those of the cuticle, have no free surface, and are not cast off as they grow old. They multiply within a closed capsule, and are laid down layer upon layer in such a way that the older are surrounded by the younger. This unique structural arrangement seems to afford a natural explanation of the continuous enlargement. With regard to both characteristics the lens probably stands alone among all the organs of the body.

Certain of the physiological peculiarities of the senile eye may perhaps find their explanation in the continuous growth of the lens. For example, *hypermetropia acquisita*. The emmetropic eye tends when middle life is past gradually to become hypermetropic, and in old age often acquires a considerable degree of hypermetropia. Donders (p. 206) ascribes the change partly to a more uniform density throughout the layers of the lens, partly to a flattening of its refracting surfaces. Now it is obvious that a symmetrical enlargement like that indicated by the figures would involve some flattening of the surfaces, and it is not unlikely that the enlargement is not as a rule symmetrical, but that the new material is added in rather larger proportion near to the equator than at the poles, for the formation of new fibres seems to occur chiefly in the

equatorial region. If the refracting surfaces are built up more rapidly in proportion at the periphery than at the centre, a still further flattening will occur. This may perhaps prove to be the cause of the hypermetropia of old age.

Again, with regard to the shallowness of the anterior chamber, which is commonly observable in advanced life. This has been attributed to an advance of the whole lens towards the cornea (Donders, p. 206). No explanation of this supposed advance has been forthcoming, however, and it is not easily reconcilable with the actual change of refraction, for an advance of the lens produces *per se* not hypermetropia but myopia. I have little doubt that the diminished depth of the anterior chamber in the healthy senile eye is simply an expression of the increased thickness of the senile lens.

Apart from the main object and result of this research, one or two points which were observed incidentally remain to be noticed.

The close relation of cataract to senility comes out in a striking manner. Between the ages of 20 and 49 no single instance of any opacity was met with among the sixty-six lenses examined. Between 50 and 59 two lenses* out of twenty-two, *i.e.* 9 per cent., presented the earliest signs of cataract in the form of slight cortical opacities at the equator. Between 60 and 69 nine out of thirty-two, *i.e.* 28 per cent., were affected similarly or to a greater extent. Between 70 and 90 thirty-four lenses were examined, and of these no less than sixteen, *i.e.* nearly 50 per cent. were affected in like manner. Thus there were altogether twenty-seven lenses which were more or less cataractous. Two of them were completely cataractous, four presented nuclear opacity, the remaining twenty-one all showed cortical opacities which appeared to be very near to the capsule, and which in nearly all cases were limited to the equatorial zone. In many of these

* These were both from the same individual; one was damaged in extraction, and does not appear in detail in the table.

the opacities would, I think, from their position, have been hardly discoverable with the ophthalmoscope.

As already stated, the cataractous lenses are shown on the chart by circles, and it is interesting to note that, as a rule, the circles stand at a decidedly lower level than the dots in the same decade; in other words, the lenses in which cataract was beginning were as a rule smaller than transparent lenses of the same age. According to the latest researches of Becker the commencement of senile cataract is due to the separation from each other of certain of the layers of lens-fibres in consequence of advancing sclerosis and shrinkage, the separation occurring first just where the capsule and subjacent fibres are most affected by the traction of the suspensory ligament, viz. at the equator. My observations are entirely favourable to this view. The subnormal size of the cataractous lenses, which was observable even in those which presented only very slight cortical opacities, suggests that the formation of cataract is perhaps preceded by a period in which the rate of growth gradually falls below the normal.

In two instances* I was able to compare a completely cataractous lens with a fellow lens which presented only some slight cortical opacities at the equator. In both cases the opaque lens was very much smaller and lighter than its fellow. Here there was evidently a shrinking or shrivelling as the result of the degenerative process.

Finally, I may mention that, in the removal of this series of lenses from the eyes of dead subjects, I obtained constant experience of a fact which has been pointed out by Pagenstecher and others, namely, that the attachment of the lens to its suspensory ligament, and especially to the hyaloid membrane of the vitreous at its posterior surface, is very much weaker in the senile than in the youthful eye.

* Table VI, No. 67, and Table VII, No. 31.

Table I.—*Averages.*

Age.	A Weight. mgr.	B Volume. cub. mm.	C Spec. grav.	D Diameter. mm.	E Diameters proportionate to volumes.
20 to 29	174	163	1067	8·67	8·67
30 to 39	192	177	1085	8·96	8·91
40 to 49	204	188	1085	9·09	9·10
50 to 59	221	205	1078	9·44	9·36
60 to 69	240	225	1067	9·49	9·65
70 to 79	(245)	(227)	(1079)	(9·64)	—
80 to 90	(266)	(244)	(1090)	(9·62)	—

N.B.—Above the age of 69 the number of transparent lenses examined was much smaller than in the earlier decades; the averages are given in brackets, and must be taken as less certain than those belonging to the earlier periods. For details see the following tables.

Table II.—*Ages 20 to 29.*

No. in Register.	Age.	Sex.	Weight.	Volume.	Sp. gr.	Diameter.
47	20	M.	159	147	1081	8·5
			—	—	—	—
48	20	M.	—	—	—	—
			195	183	1066	9·
66	20	M.	—	—	—	—
			151	139	1086	8·75
35	21	M.	178	168	1060	9·
			180	172	1046	9·
46	21	F.	167	160	1044	8·5
			180	162	1049	8·5
60	21	M.	—	—	—	—
			195	186	1048	9·
44	22	F.	180	171	1053	8·25
			175	167	1048	8·25
29	23	F.	—	—	—	—
			152	148	1048	8·25
43	24	M.	175	165	1060	9·
			—	—	—	—
41	25	M.	176	168	1048	8·75
			178	168	1060	8·75
68	26	F.	190	172	1105	8·75
			184	166	1108	8·75
4	27	M.	—	—	—	—
			153	144	1062	—
58	27	M.	162	153	1059	8·5
			166	156	1064	8·5
70	28	M.	181	164	1104	8·75
			180	162	1111	8·75
Average			3648÷21 = 174	3419÷21 = 163	1067	173·5÷20 = 8·67

In this and the following tables the upper figures in each bracket represent the right eye, the lower figures the left eye. Where no figures are given, it is to be understood that the lens-capsule was broken during extraction and the specimen thereby rendered useless.

Table III.—*Ages* 30 *to* 39.

No. in Register.	Age.	Sex.	Weight.	Volume.	Sp. gr.	Diameter.
39	32	M.	167	155	1077	8·75
			172	161	1068	8·75
80	33	M.	177	164	1079	8·75
			173	161	1074	8·75
26	34	M.	195	184	1060	9·
			177	165	1073	9·
3	35	F.	197	173	1139	—
			—	—	—	—
7	35	F.	180	170	1059	9·
			—	—	—	—
18	35	M.	183	166	1102	—
			184	166	1108	—
51	36	M.	192	184	1043	9·
			192	177	1085	9·
34	37	M.	196	192	1021	8·75
			—	—	—	—
76	37	F.	206	188	1096	9·
			206	190	1084	9·
42	38	M.	199	184	1081	8·75
			201	190	1058	8·75
8	38	M.	199	179	1111	9·
			—	—	—	—
75	38	M.	197	181	1088	9·
			195	179	1089	9·
72	39	M.	215	196	1097	9·5
			216	195	1108	9·5
	Average		4219÷22 = 192	3905÷22 = 177	1085	170·25÷19 = 8·96

TABLE IV.—*Ages 40 to 49.*

No. in Register	Age.	Sex.	Weight.	Volume.	Sp. gr.	Diameter.
71	40	M.	217	194	1118	9·
			216	195	1108	9·
33	41	M.	178	166	1072	8·75
			—	—	—	—
86	42	M.	220	198	1111	9·
			—	—	—	—
38	42	F.	217	199	1090	9·5
			215	198	1086	9·5
85	42	M.	189	169	1118	9·
			—	—	—	—
77	43	M.	196	183	1071	9·25
			194	181	1071	9·25
87	44	M.	238	226	1053	9·
			230	217	1060	9·
83	45	F.	194	178	1090	8·75
			191	160	1194	8·5
16	45	M.	—	—	—	—
			194	185	1049	9·5
5	45	M.	204	185	1103	—
			198	179	1106	—
25	46	M.	211	198	1066	—
			—	—	—	—
20	49	M.	199	189	1053	9·5
			—	—	—	—
45	49	M.	193	185	1043	9·
			189	177	1068	9·
1	49	M.	203	183	1126	—
			196	177	1107	—
6	49	F.	214	204	1049	—
			—	—	—	—
		Average . .	4696÷23 = 204	4326÷23 = 188	1085	154·5÷17 = 9·09

Table V.—*Ages 50 to 59.*

No. in Register.	Age.	Sex.	Weight.	Volume.	Sp. gr.	Diameter.
24	50	F.	208	197	1056	9·5
			213	194	1091	9·5
32	50	M.	240	222	1081	10·
			242	222	1090	10·
13	51	M.	226	208	1086	9·
			222	206	1078	9·
22	51	M.	227	214	1060	10·
			226	207	1091	10·
19	52	M.	—	—	—	—
			191	184	1038	—
64	52	M.	251	237	1069	9·5
			257	250	1029	9·5
23	53	F.	204	186	1097	9·5
			209	196	1066	9·5
11	54	M.	227	214	1060	9·
			227	214	1060	9·
49	55	M.	220*	205	1073	10·
			—*	—	—	—
10	56	M.	—	—	—	—
			234	211	1109	9·5
17	56	M.	219	198	1106	9·25
			217	199	1090	9·25
65	57	M.	186	170	1094	9·
			188	171	1099	9·
50	59	M.	225	213	1056	9·75
			—	—	—	—
Average			4639÷21 = 221	4313÷21 = 205	1078	188·75÷20 = 9·44

N.B.—The *thick figures* in this and subsequent tables represent lenses which were not perfectly transparent; they are not included in the averages.

* Slight cortical opacities at the equator.

TABLE VI.—*Ages 60 to 69.*

No. in Register.	Age.	Sex.	Weight.	Volume.	Sp. gr.	Diameter.
40	60	M.	215	200	1075	9·5
			219	206	1063	9·5
88	60	F.	239	216	1106	9·
			236	213	1108	9·
61	61	M.	248	235	1055	9·5
			242	223	1085	9·5
15	62	F.	171*	157	1090	—
			179†	168	1065	—
30	62	M.	251	233	1077	9·5
			252	243	1037	9·5
52	63	M.	239	224	1067	9·75
			240	224	1071	9·75
84	63	M.	190‡	172	1105	9·
			183‡	169	1083	9·
28	65	M.	231	225	1027	9·
			223	211	1057	—
36	65	F.	236	224	1054	9·5
			228	211	1080	9·5
53	65	M.	245	228	1075	9·25
			237	223	1063	9·25
54	65	M.	247	231	1069	9·5
			247	232	1065	9·5
59	65	F.	199‡	179	1111	8·5
			200‡	193	1031	8·5
63	66	M.	226	209	1081	9·5
			236	219	1078	9·5
90	68	M.	263§	240	1096	9·75
			263	242	1087	9·75
56	69	F.	264	252	1044	10·
			260	245	1061	10·
67	69	M.	184‖	170	1082	9 5
			216‡	199	1085	9·75
	Average . .		5524÷23 = 240	5169÷23 = 225	1067	208·75÷22 = 9·49

* Nuclear opacity, with striæ radiating from it.
† Slight nuclear opacity.
‡ Slight cortical opacities at equator.
§ One flake of opacity deep in cortex.
‖ Completely cataractous.

Table VII.—*Ages 70 to 79.*

No. in Register	Age.	Sex.	Weight.	Volume.	Sp. gr.	Diameter.
12	71	M.	237	226	1049	—
			—	—	—	—
91	71	M.	—	—	—	—
			220	204	1078	9·25
69	72	M.	249	228	1092	9·75
			237	215	1102	9·5
92	72	M.	282	256	1100	9·5
			284	258	1100	9·5
2	73	M.	244	225	1084	—
			243	222	1095	—
55	75	M.	218*	205	1063	9·25
			212*	195	1087	9·25
57	75	F.	229†	213	1075	9·75
			231	222	1041	10·
81	75	F.	225†	207	1087	9·5
			222†	207	1072	9·5
31	76	M.	252†	228	1105	10·
			175‡	166	1054	—
93	76	M.	239	224	1067	10·
			242	226	1071	10·
9	78	F.	230	215	1070	9·25
			—	—	—	—
74	78	F.	241†	220	1095	9·75
			244†	223	1094	9·75
	Average		2938÷12 = 245	2721÷12 = 227	1079	86·75÷9 = 9·64

* Slight nuclear opacity.

† Slight cortical opacities at equator.

‡ Completely cataractous; cortex shrivelled.

TABLE VIII.—*Ages* 80 *to* 89 (and 90).

No. in Register.	Age.	Sex.	Weight.	Volume.	Sp. gr.	Diameter.
73	82	M.	270	245	1102	10·
			283	262	1080	9·75
82	83	F.	247*	231	1069	9·5
			245*	227	1079	9·5
95	83	M.	235*	213	1103	9·
			234*	215	1088	9·
89	84	F.	254*	234	1085	9·75
			—	—	—	—
94	86	F.	249	224	1112	9·5
			249	226	1102	9·5
78	87	M.	273	253	1079	10·
			273	253	1079	10·
79	90	M.	252†	237	1063	9·75
			254†	237	1072	9·75
62	90	M.	277*	263	1053	10·
			277*	261	1061	10·
	Average		1597÷6 = 266	1463÷6 = 244	1090	57·75÷6 = 9·62

* Slight cortical opacities at equator.

† Considerable cortical opacities at equator.

(*January* 11*th*, 1883.)

P.S.—August, 1883. Fourteen lenses, belonging chiefly to the later decades, have been examined since this paper was read. The results are incorporated in the tables as now given.

2. *Posterior dislocation of the lens of twelve years standing, following a blow.*

By Samuel West, M.D.

About twelve years ago, the patient, an old woman, knocked her right eye against the corner of a table. "The eye bled." She was not under treatment but poulticed it for a week.

She had a little pain at the time but none since. The sight was lost at once, but she thinks it has remained the same. She can now count fingers at twelve feet. The right pupil is of maximum size. The iris is reduced to a narrow rim, which is notched at the upper left side, and this notch corresponds with a linear scar extending about $\frac{1}{8}''$ from the sclerotic on to the cornea. Lying free in the vitreous is the lens, which forms a yellow opaque body. It moves freely as the eye moves, being probably attached only at its lower margin.

Ophthalmoscopic examination shows large patches of choroidal atrophy and atrophy of the disc.

(*Card specimen. March 8th,* 1883.)

DESCRIPTION OF PLATE III BIS.

This Plate illustrates Dr. Stephen Mackenzie's case of Tortuous Retinal Vessels in a case of Emphysema (p. 101).

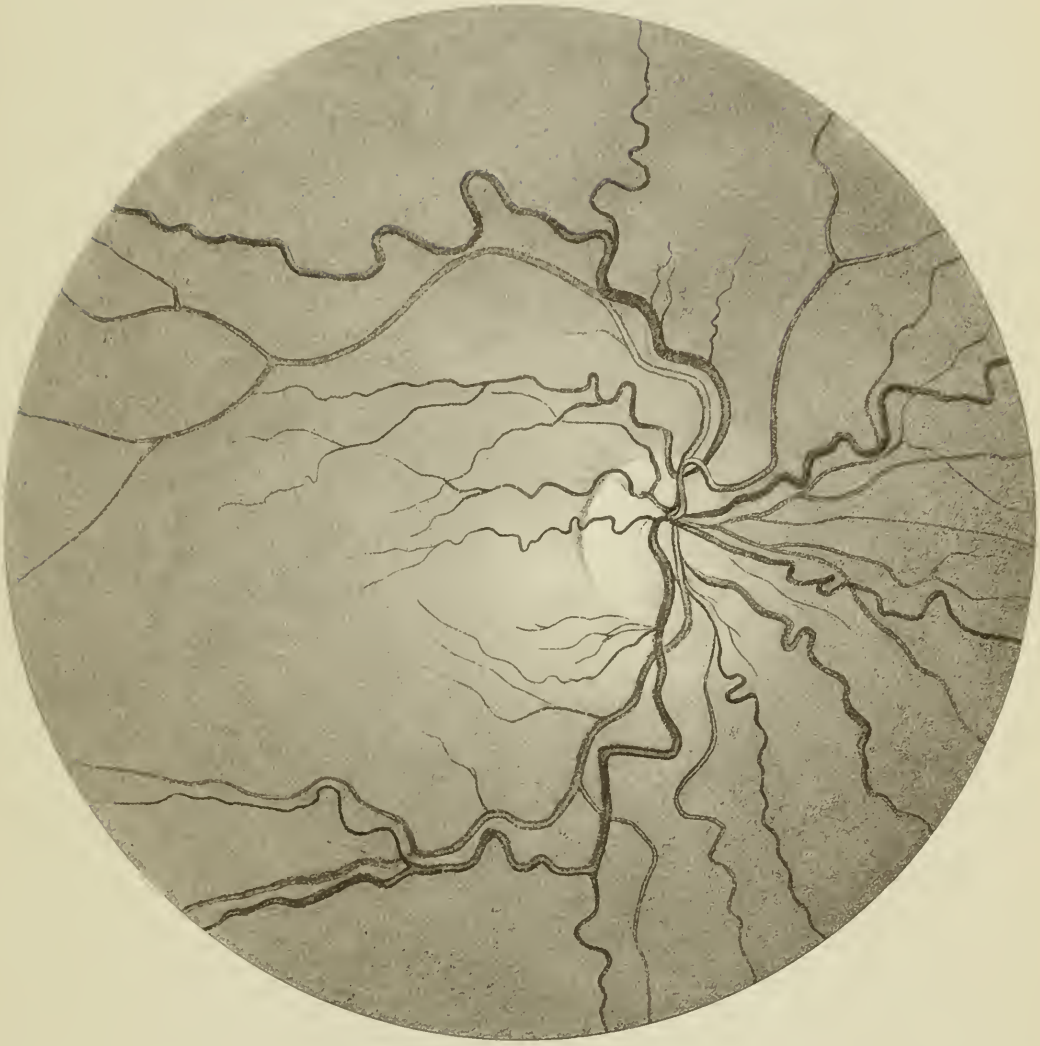


Fig. 1.

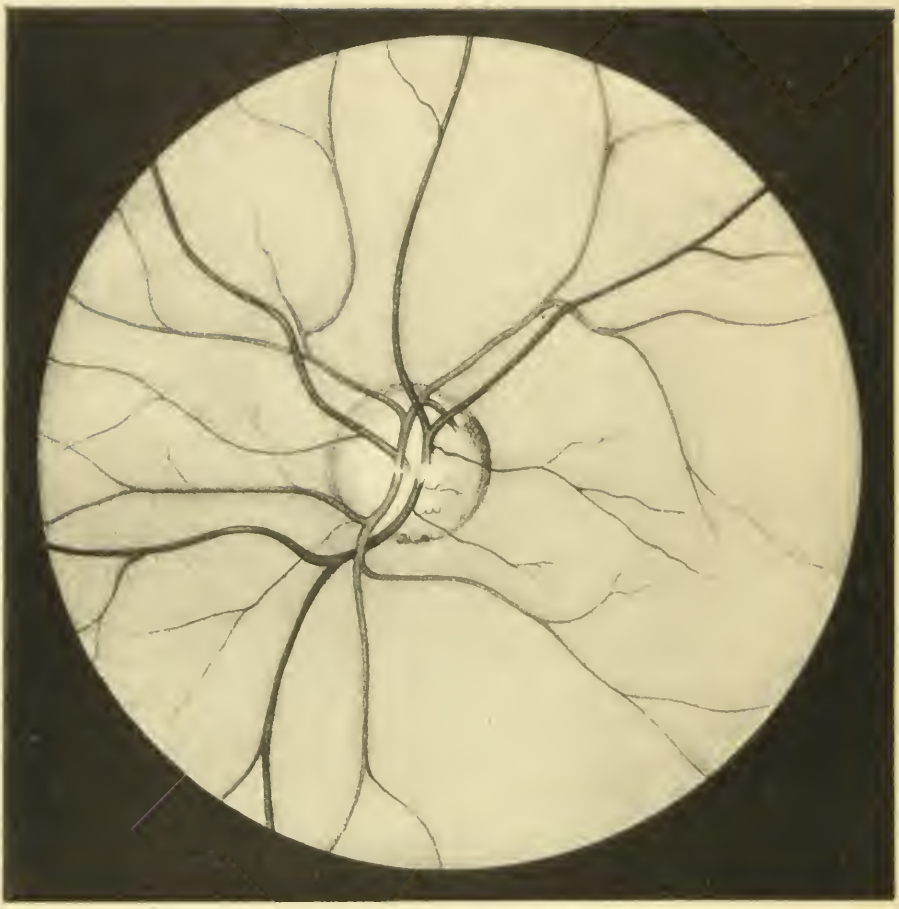
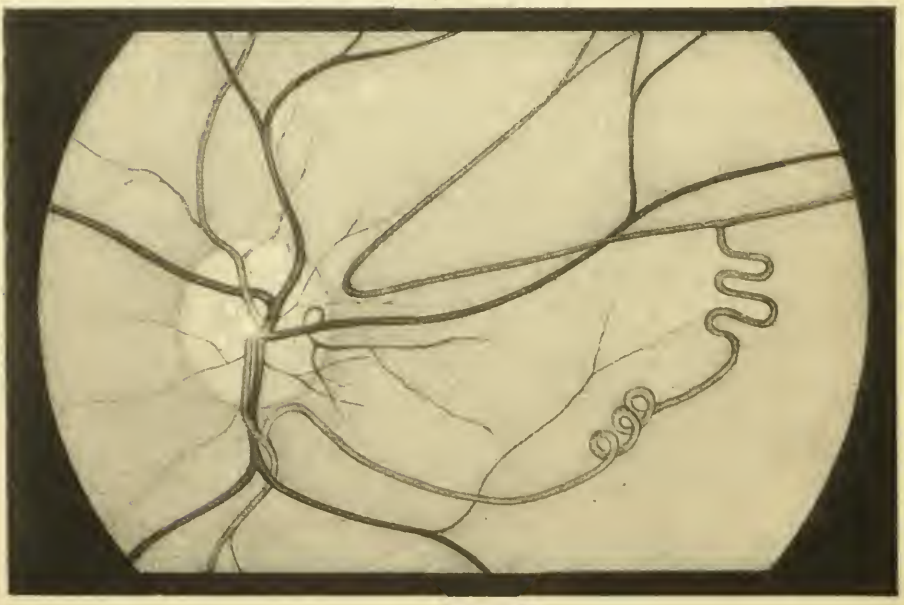


Fig. 2.



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DESCRIPTION OF PLATE IV.

FIG. 1 shows the ophthalmoscopic appearances in Mr. Benson's case of Retino-ciliary Artery (p. 101). From a drawing by himself.

FIG. 2 shows the ophthalmoscopic appearances in Mr. Story's case of Anomalous Distribution of Retinal Arteries (p. 102). From a drawing by Mr. Benson, after a sketch by Mr. Story.

VII. RETINA AND CHOROID.

1. *Ophthalmoscopic drawing showing great tortuosity of retinal vessels.*

By Stephen Mackenzie, M.D.

(With Plate III *bis*.)

H. T—, æt. 20, suffering from severe vesicular emphysema, œdema of legs, and lividity. Vision normal; pupils large. In both eyes the retinal veins were extremely tortuous, somewhat dilated, and dark (in the drawing from which the Plate is taken the veins are scarcely represented large or dark enough); arteries only slightly tortuous. A condition of extreme tortuosity of the retinal veins and arteries, as has been shown by Benson, and of retinal veins as has been shown by Nettleship ('Ophth. Soc. Trans.,' vol. ii, pl. iii, p. 56), may occur in otherwise healthy eyes. The condition in the present instance might be ascribed to the venous obstruction consequent on the emphysema, but the two cases recorded by Nettleship where, as in this patient, the change was limited to the veins, make this more than doubtful. It is thought desirable to place the case on record to draw attention to the point raised.

(*Card specimen. December* 14*th*, 1882.)

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2. *Drawing showing an unusual course taken by a branch of the central artery on the disc.*

By Arthur H. Benson (Dublin).

(With Plate IV, fig. 1.)

The figure shows an anomalous branch of the central artery of the retina. From the large artery running from

the disc upwards and outwards, a branch of the second magnitude is given off just at the disc border (Plate IV, fig. 1). This branch runs downwards on the disc surface, under the large vein, and seems to sink into the substance of the disc near its margin. It does not again become visible anywhere on the retina.

It seems as if this branch were the reverse of a cilio-retinal artery, *i.e.* that instead of one of the ciliary arteries sending a branch to supply some portion of the retina, the retinal artery sends a branch to take the place of one of the ciliary. We may perhaps call it a retino-ciliary artery, as distinguished from such cilio-retinal arteries, as are described by Nettleship in the 'Roy. Lond. Ophth. Hosp. Reports,' vol. ix, p. 161.

(*Card specimen. March 8th*, 1883.)

On an anomalous distribution of the retinal arteries.

By John B. Story (Dublin).

(With Plate IV, fig. 2.)

The remarkable peculiarity in the arrangement of the retinal arteries exhibited in Plate IV, fig. 2, occurred in the right eye of a young gentleman who consulted me upon the subject of spectacles in May, 1881. For two years he had been using $-3{\cdot}5$, D., not for distant objects alone but for reading also, in spite of the fact, which was at once observed on ophthalmoscopic examination, that both eyes were hypermetropic. After the use of atropine the right eye took + 2·25 D. and the left +1·75 D. for distant vision, and I ordered him somewhat weaker convex glasses for reading when the effects of the atropine had passed off. Some astigmatism appeared to be present, but I could not definitely measure it, and I failed to get any better vision with cylindrical than with spherical lenses. In both eyes vision was only $\frac{5}{10}$ after correcting the refraction by glasses. During the existence of the

atropine mydriasis I had ample opportunity of accurately examining the remarkable vascular anomaly, of which I took a rough sketch at the time, from which sketch my friend, Mr. Arthur Benson, has kindly made the picture which is reproduced in the Plate.

I noticed when I first examined the fundus of the right eye that the superior nasal artery appeared near the edge of the disc as a free loop, whose two branches passed upwards and inwards, and horizontally inwards, respectively. This loop had no apparent origin, being neither connected with the superior temporal nor with any other artery on the disc, and did not in any wise resemble a cilio-retinal vessel. I concluded that it probably had some junction with the central artery lower down in the disc beneath the nerve fibres, and did not discover the true state of affairs till after the pupil was dilated and I was able to carefully follow out the branches of the various arteries. I then discovered that the upper branch of this loop represented in its course and distribution the normal superior nasal splitting up, as I followed it to the periphery of the fundus, into smaller and smaller branches, which eventually became so small that I could observe them no further.

The lower of the two branches behaved in a very different manner. It ran horizontally inwards from the upper and inner edge of the disc, without giving off any branch at all, until it arrived at a point distant about two diameters of the disc, where a large branch left it running vertically downwards. The horizontal prolongation then began to behave in the ordinary way, it was itself smaller than its apparently down-running branch, and it soon split up into smaller branches, and was lost by degrees as I followed it towards the periphery. I then followed the downwards branch, and found that it began at once to form two or three large lateral curves, which sank deeper into the retinal tissue in some parts of their sweeps, it then ran downwards again and a little more outwards, and then performed a series of corkscrew bends,

after which it ran with a gentle curve to the lower margin of the disc, and again took a sharp turn downwards before it ended in the inferior artery of the disc, above the point where the latter divided into the inferior nasal and the inferior temporal arteries. The artery did not alter much in size during this course from the upper edge of the disc to its junction with the inferior artery, but whatever change there was took the direction of increase, and not of diminution, of calibre, and I had no difficulty in following out accurately every minute bend and twist of the vessel, although I confess that I was not able to reproduce them accurately in my sketch, so that although I am able confidently to assert the general fidelity of the drawing to what I observed, I cannot assert that each bend and twist is perfectly true to nature. There cannot be a shadow of doubt that the course of the blood-stream in this patient's retina was from the inferior artery of the disc by a gentle curve downwards and inwards to the corkscrew bends, then upwards through the wide lateral sweeps to the horizontal portion, where it divided into two streams, one running horizontally inwards to supply the capillaries of the neighbourhood, and the other passing horizontally outwards again to the disc, where it sent off the artery which performed the function of the superior nasal. I searched most carefully and could find no evidence of any second communication between this arterial system and the central artery; at the bend at the upper and inner margin of the disc I am certain none existed, and though I thought that at times I could detect a whitish line passing towards the disc from the bend, at others I could not see it. I found it impossible to make out any such whitish line as a distinct object; it was more that I could not persuade myself that it ought not to be there.

This eye also presented other abnormal appearances. There were white perivascular lines on several of the vessels, and the disc contained a somewhat excessive amount of white opaque tissue. These appearances

existed also in the left eye, but the right disc had besides two greyish-white circular spots in its temporal half rather near its centre.

I have not seen any similar spots except in one case under the treatment of Mr. Arthur Benson at St. Mark's Hospital. In his case, however, they covered the whole disc, and extended also over its edge at several points, and were slightly raised over the surface. In mine they seemed to be flat. I have written to my patient lately to ask him to let me have a second opportunity of examining his eye, but I have not received any answer to my communication.

My friend, Professor Purser, has suggested to me that the cause of this anomalous arterial arrangement has been a blocking of the superior nasal artery at its exit from the disc in very early life—perhaps during fœtal life. There are, as we all know, no arterial anastomoses between the retinal vessels, but capillary anastomoses must exist, which at the period of life at which embolus of the central artery usually occurs are useless for the purpose of re-establishing the circulation, but there is no reason why in the young subject these capillary anastomoses may not be capable of developing sufficiently to effect a good collateral circulation, and that is what I believe occurred in my case.

There were other evidences of some past pathological process in the retina, the white opaque tissue in the disc, the peri-vascular lines on the vessels (not reproduced in the drawing), and the two whitish-grey spots in the disc, all point to some antecedent diseased state, and I think from all these facts taken together I am justified in concluding that my patient had in very early life an embolus, or thrombus, in the superior nasal artery of the retina, which completely obliterated that vessel at the point where it left the disc, and that a collateral circulation was established by the dilatation of capillary vessels connected with the inferior retinal artery.

I should add that the very remarkable bends and

twists in the anastomosing vessel lend additional weight to the theory I have advanced. I regret that I did not measure the field of vision in this case, but the perfectly normal size and appearance of the arteries made me forget this point.

No matter what our views may be as to the etiology of the peculiar vascular arrangement I have described, the case is I consider worth putting on record as an instance which is, I believe, unique, where three fourths of the retina received its blood-supply from the inferior artery, and only one fourth from the superior artery of the disc.

(*July 6th*, 1883.)

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4. *A case of facial and ocular nævus.*

By P. Horrocks, M.D.

(With Plate V, fig. 1.)

E. A—, æt. 9, has been subject to fits since birth. The fits are of an epileptic nature and cause clonic spasms of the left side of the trunk and limbs. She is hemiplegic on the left side, the left arm being nearly useless, and in a permanent state of flexion from rigidity. She can use the left leg in progression even without crutches, which she has never worn, but she limps considerably. There is little or no wasting of the limb, nor is the left side colder than the right. There is no loss of sensation. She has the fits almost every day, often two or three a day, although she is taking fifteen grains of bromide of potassium three times a day. The right side of her face, including the skin of the eyelids and forehead, is covered with a nævus, giving a port-wine stain appearance; it does not cross the median line at any point. The front of the eyeball on the same side is also affected, showing a network of vessels on each side of the cornea over the sclerotic, whilst on the left side the conjunctiva is quite normal. On examination with the ophthalmoscope,

DESCRIPTION OF PLATE V.

FIG. 1 shows the ophthalmoscopic appearances in Dr. Horrocks's case of Facial and Ocular Nævus (p. 106). From a drawing by Miss Boole.

FIG. 2 shows some of the ophthalmoscopic appearances in Mr. Benson's case of Retinal Aneurysms (p. 108). From a drawing by himself.

Fig. 1.

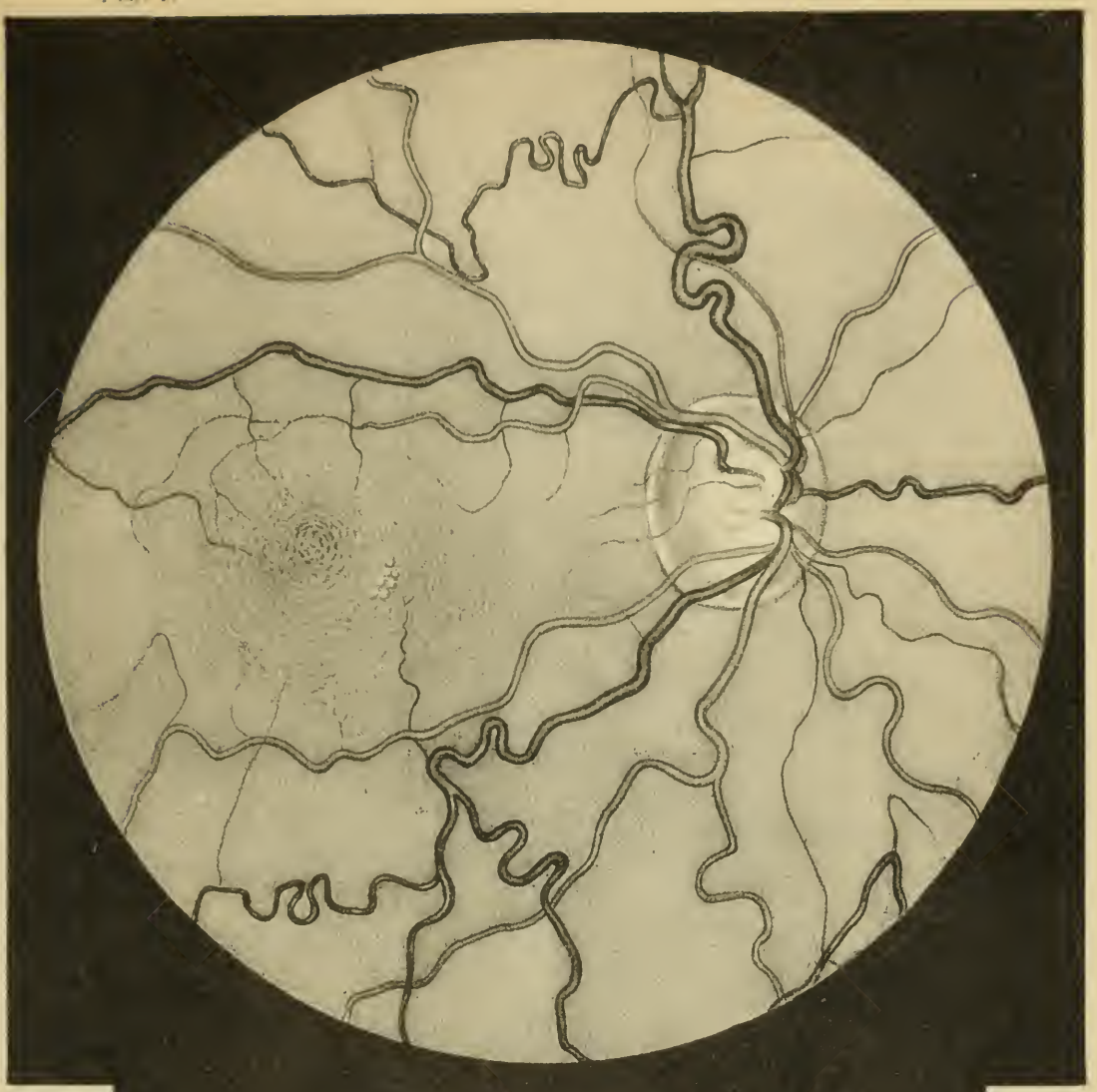

Fig 2.

the retinal veins of the right eye are seen to be exceedingly tortuous (Plate V, fig. 1), contrasting strongly with those in the left eye, which appear quite normal. The choroidal vessels are unaffected, nor does the right choroid appear at all darker than the left. The sclerotic is unaffected. The pupils are large, and act readily to accommodation and light. The girl has never been to school, and her intellect has suffered considerably through the fits. Hence, she scarcely understands questions put to her, and nothing satisfactory can be elicited as to her field of vision, acuteness of vision, or colour-sense; but as far as can be made out, she sees quite well and equally with both eyes.

An interesting point in the case is that, although all blood-vessels are derived from the mesoblast, yet only those appear to have been implicated which are distributed to structures developed from epiblast (skin, conjunctiva, retina). From this one naturally asks, may not possibly the vessels of the pia mater on the right side, supplying the brain (also derived from epiblast) be similarly affected? and may this have something to do with the fits which convulse her left side?

Without the facial and conjunctival nævus, the case would probably be set down as one of great tortuosity of the veins; but any one who sees this case will admit that it is certainly associated with, and due to, the same causes as the facial nævus. And perhaps many cases of tortuosity of retinal veins are due to the same disturbances of development as those producing nævi.

Mr. Nettleship reports to me as follows on the condition of the eye:—

"The cornea of the affected (R.) eye is 14 mm. wide, being about 2 mm. wider than the other, and the pupil is usually larger. Refraction very slightly myopic. The disc shows a very large and deep physiological cup, the bottom of which is not seen clearly with less than − 6 D. There is no evidence of undue vascularity of the choroid. The enlarged vessels on the front of the eyeball are certain

groups of the anterior ciliary veins (not conjunctival), chiefly at the inner, lower and lower-outer parts of the sclerotic; the vessels emerge as usual from the sclerotic close to the cornea, and divide into twigs as they pass backwards. The anterior ciliary arteries (with one exception) seem to be of natural size. There is no affection of the capillaries of the sclerotic."

(*Living specimen. July 6th*, 1883.)

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5. *Aneurysms on retinal vessels in a peculiar case of retinitis.*

By J. B. Story and A. H. Benson (Dublin).

(With Plate V, fig. 2.)

W. J—, æt. 20, has been attending St. Mark's Ophthalmic Hospital, under the care of Mr. Story, at intervals since 1881, in consequence of a peculiar form of retinitis, associated with considerable fibrous proliferation, and perivasculitis.

At a meeting of the Dublin Ophthalmological Club in March, 1883, Mr. Story (being unable to attend) asked me to exhibit the case for him as one of peculiar retinitis. On examining the patient for that purpose I discovered, for the first time, that the vessels in the outer periphery of the right eye were dilated in places.

Three bright red, well defined, cherry-like, globular protrusions of one of the walls of the artery running above the macula were observed, and the other appearances shown in Plate V, fig. 2 (which is taken from a drawing I made next day) were noted, viz. a cylindrical dilatation on the trunk of the artery, which so suddenly and evenly became enlarged as to have quite the appearance of an intussusception.

There was also much white fibrous thickening along the arteries, so that in places the blood column could hardly be seen.

On the small vein near this artery was observed a three headed swelling, and a little further on a single swelling of the same vein.

The remainder of the fundus was carefully searched for any similar appearances, but none were visible. There were in many places prominent masses of fibrous tissue in the retina, in some of which blood-vessels ran, but nothing of the nature of an aneurysmal dilatation was found further than those shown in the Plate.

These appearances were demonstrated to the members of the club, and all had an opportunity of examining the case.

In June, 1883 (*i.e.* three months after the drawing was taken), I again saw the patient. Considerable changes had occurred in the appearance of the fundus, and another drawing * was made, representing a small part of the right fundus situated to the inner side of the disc; a portion of the fundus which, three months before, was perfectly free from aneurysms. On the small branches of a vein and an artery running close together were seen several well-defined dilatations, but apparently differing from those seen in the first drawing (Plate III), in that they were no longer the lateral swellings, but seemed to occupy the whole circumference of the vessels in the affected parts. They were for the most part surrounded by an area of thickened retina, and they had not the bright, sharply-defined outline that those in the first drawing had. There were one or two other dilatations of the vessels not copied in either of the drawings. On comparing the first drawing made three months before, with the region which it represented, it was found that very considerable changes had now taken place. The aneurysms had got much smaller, and had lost their bright cherry-like appearance, and had become duller, more hazy, less-filled looking, and surrounded with more fibrous thickening of the retina, so that they approximated somewhat to the appearances

* This drawing has not been reproduced, but was shown at the Meeting of the Society.

shown in the second drawing. The venous dilatations were less changed than the arterial.

It seemed as if nature were effecting a cure, constricting the vessels by the fibrous tissue developed about them, and thus narrowing or obliterating their lumen, and allowing coagulation and subsequent shrinking to occur in the aneurysms.

The history of the case has most unfortunately been lost. Thinking that all the notes taken at the hospital could be found I omitted to test heart, urine, vision, &c., till the end, and then the patient suddenly left for England without fulfilling his promise of returning for these final investigations to be made.

He was a tall, dark, slight, delicate-looking man, with a tendency to marginal blepharitis. He did not complain of ill health; headaches he had at times. T. was always normal. The case is, so far as I am aware, unique.

(*July 6th*, 1883.)

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6. *Peculiar appearance in the retina in the vicinity of the optic disc occurring in several members of the same family.*

By R. Marcus Gunn.

Sarah C— attended the Western Ophthalmic Hospital on September 30th, 1882, complaining of headaches and burning pain in the eyelids. There is no failure of vision, but "when the lids feel very hot the sight seems hazy."

She is a very intelligent, healthy-looking girl, æt. 16. When between two and three years old she fell and struck her head on the hearthstone, and was unconscious for some time; but she then seemed to recover perfectly. About three years ago she had a bad ulcerated throat, but it was probably not syphilitic, although there is a

history of a slight eruption following it (possibly scarlatina). Her health generally has been good, but recently she has been subject to severe headaches, coming on at all times of the day, often before breakfast just after rising out of bed. Once or twice she was sick, and vomited at this time. Since the headaches began there has been great drowsiness; her mother says she is constantly falling asleep. Menstruation regular, sometimes excessive in amount. She complains of palpitation and shortness of breath on slight exertion, but does not look anæmic. Her eyes are normal in appearance. Pupils active, V. $= \frac{20}{20}$, fairly with each, Hm. 0·5 D. After using atropine drops for a fortnight, each eye sees $\frac{20}{20}$ with + 1·25 D. On examining now with the ophthalmoscope by the direct method the following condition is found.—In the *R. eye* there are very minute, yellowish white, shining dots in the retina for some distance around the disc, especially to the nasal side and below; in distribution these dots are remarkably equidistant from one another, and are situated anteriorly to the largest retinal blood-vessels, each being less than one fifth of the diameter of a large vessel; the outline of the disc is rather indistinct, the large veins full and somewhat tortuous. The *L. eye* shows a similar condition, but the disc outline is more blurred than in the right. This appearance is most easily seen when the light is thrown somewhat obliquely on the part of the retina to be examined; the dots will then be seen to stand out well near the border of the image of the flame.

Family history.—There is a history of rheumatism on the father's side, and several members of his family have had heart disease. No consanguinity of parents. No evidence of hereditary syphilis. The patient's mother and several of her brothers and sisters are subject to "weak eyes." About twenty years ago, after a severe sorethroat during a household epidemic of scarlet fever, the mother's right eye became inflamed; for years afterwards it used to be red and irritable at intervals of from two to four weeks,

but for several years past she has been quite free from these attacks. R. eye, V. = $\frac{20}{30}$, Hm. 1·5 D., but V. not $\frac{20}{20}$; L. eye, V. = $\frac{20}{50}$, Hm. 2 D, V. = $\frac{20}{30}$, with cylinder added = $\frac{20}{20}$ partly. *Ophthalmoscope.*—R. eye: a few minute dots similar to those described above are visible beside the vessels going upwards and outwards from the disc. L. eye: no dots seen in any part of fundus.

Her family is as follows:

1. Daughter, æt. 27, suffers from "weak heart." Is married and has two children, aged six and five years. The same appearance of dots as that described above is visible in each retina, but not so distinctly as in the patient. Her children were both examined also: in neither was there a trace of this appearance.

Next there was a miscarriage.

2. Son, æt. 25, in good health. Resides abroad. Not examined.

3. Daughter, æt. 24. Her eyes have occasionally given her trouble; she wore glasses for a time when about seventeen years old. The ophthalmoscope shows the dotted appearance very well marked in both eyes.

4. Son, æt. 23. About age of seventeen his eyes were weak. Not examined.

Next a miscarriage.

5. Daughter, æt. 21. General health good, but her eyes are sometimes inflamed. She was troubled with styes when a child, and the eyes still smart when reading much. V. = $\frac{20}{20}$, Hm. 0·75 D. in each eye. The dots are visible in both eyes, especially well marked above the disc.

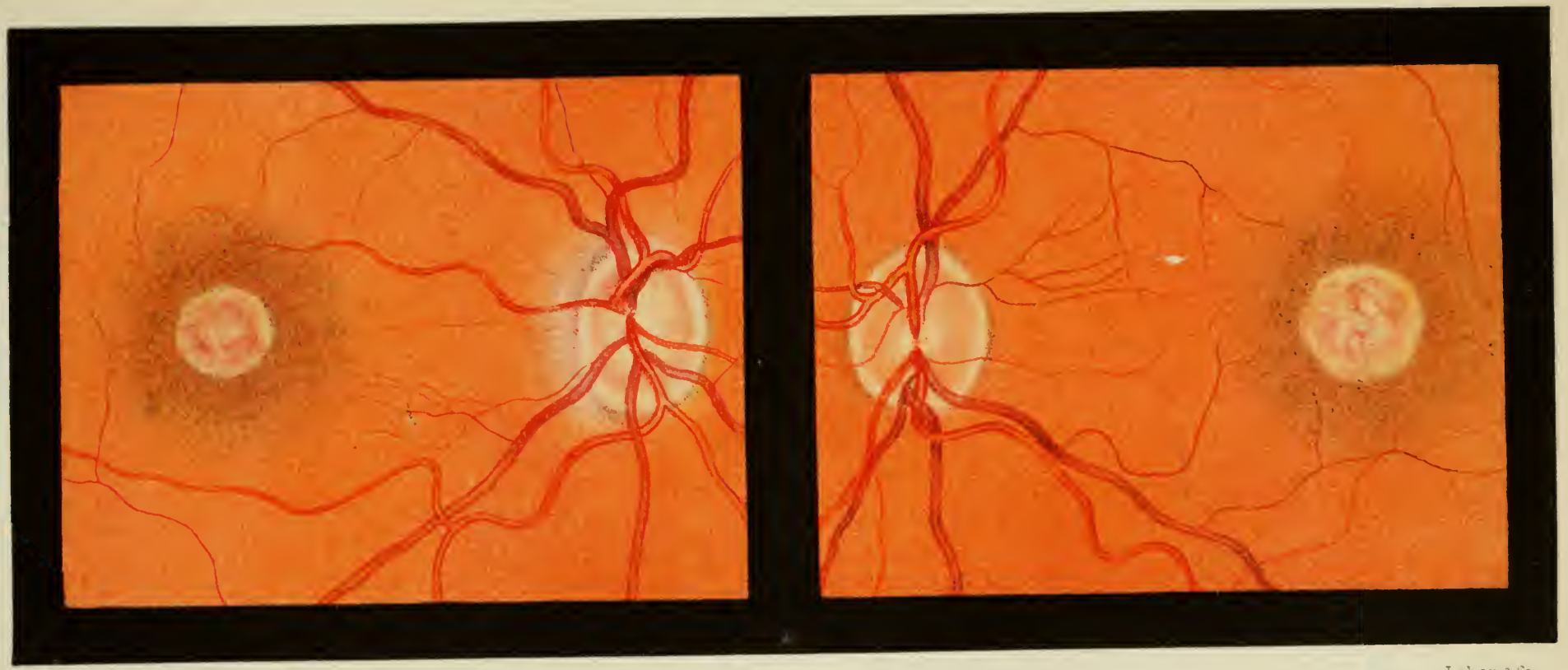
6. Patient.

7. Son, æt. 15. General health not very good. His eyes are often inflamed and weak. R. eye, V. = $\frac{20}{20}$ well, Hm. 0·5 D.; L. eye, V. = $\frac{20}{20}$ partly, with 0·50 D. = $\frac{20}{20}$. *Ophthalmoscope.*—The dotted appearance of retina is well marked in both eyes.

8. Son, æt. 12. General health good. His eyes have not given much trouble, but occasionally they become

DESCRIPTION OF PLATE VI.

Plate VI shows the ophthalmoscopic appearances in the erect image in Mr. Adams's case of Peculiar Appearances at the Yellow Spot in each Eye (p. 113). From drawings by Miss Boole.



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bloodshot. R. eye, V. = $\frac{20}{20}$, no Hm.; L. eye, V. = $\frac{20}{20}$ partly, with + 0·50 D. = $\frac{20}{20}$. *Ophthalmoscope.*—Same punctate appearance well marked in both eyes.

Since the birth of this son the mother has had four miscarriages, but no living child.

Thus in every member of this family that has been examined the same ophthalmoscopic appearance is found.

(*Living specimen. December 14th,* 1882.)

P.S.—July, 1883.—Since bringing this paper before the Society, I have carefully examined a large number of patients with reference to the occurrence of the described condition of retina. Altogether I have found the same appearance in four cases.

1. Female, æt. 25, exophthalmic goitre.

2. Female, æt. 26, complains of aching when she reads. V. = $\frac{20}{20}$, in each eye no Hm. Asthenopia disappeared under quinine and iron tonic.

3. Female, æt. 16. Ever since an attack of "the blight" at the age of seven, her eyes have troubled her: occasional dimness on close work, &c. R. eye, V. = $\frac{20}{30}$, Hm. 0·75 D.; L. eye, V. = $\frac{20}{20}$ fairly, Hm. 0·5 D. *Ophthalmoscope.*—Opaque nerve fibres in both. In the right eye the dots described above are well seen.

4. Female, æt. 21. Her eyes have always been weak. R. eye, V. = $\frac{20}{20}$ fairly; L. eye, V. = $\frac{20}{50}$ fairly. *Ophthalmoscope.*—Dots seen in both eyes. Myopic astigmatism.

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7. *Case showing peculiar changes in the macula.*

By Jas. E. Adams.

(With Plate VI.)

The patient, a woman, æt. 37, from whom the drawings reproduced in Plate VI were taken, presented herself at Moorfields, complaining of pains in the head, espe-

cially on the left side and in the occipital region, and she had discovered that her sight had deteriorated in the left eye.

On examination it was found that the peculiar change shown in the Plate occupied the region of the macula, and it was assumed that it was the cause of the diminished visual acuteness (viz. $\frac{20}{50}$ and Sn. $1\frac{1}{2}$ in left), but an almost precisely similar condition was discovered in the right eye, in which the vision was absolutely normal. It is, of course, quite possible that the change in the left is somewhat more advanced, and that the sight of the right will get worse, but as the change is an unfamiliar one I thought it worthy of publication in its present stage, as the patient will in all probability remain under observation. The patches are not raised, but appear simply as alterations in texture and colour, and concerning the exact nature of them one can do little more than guess.

The eyes have been examined several times during three months, but no alteration has taken place either locally or with regard to the vision.

Patient now reads :—R. eye = $\frac{20}{20}$ and with + 1 D. Sn. $1\frac{1}{2}$; L. eye = $\frac{20}{50}$ and two letters of $\frac{20}{40}$, and with + 1 D. Sn. $1\frac{1}{2}$, but not fluently.

(*July 6th*, 1883.)

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8. *A case of embolism of the central artery of each retina occurring in the right eye nearly twelve years ago, in the left eighteen months ago, with degenerative changes at the macula in each eye.*

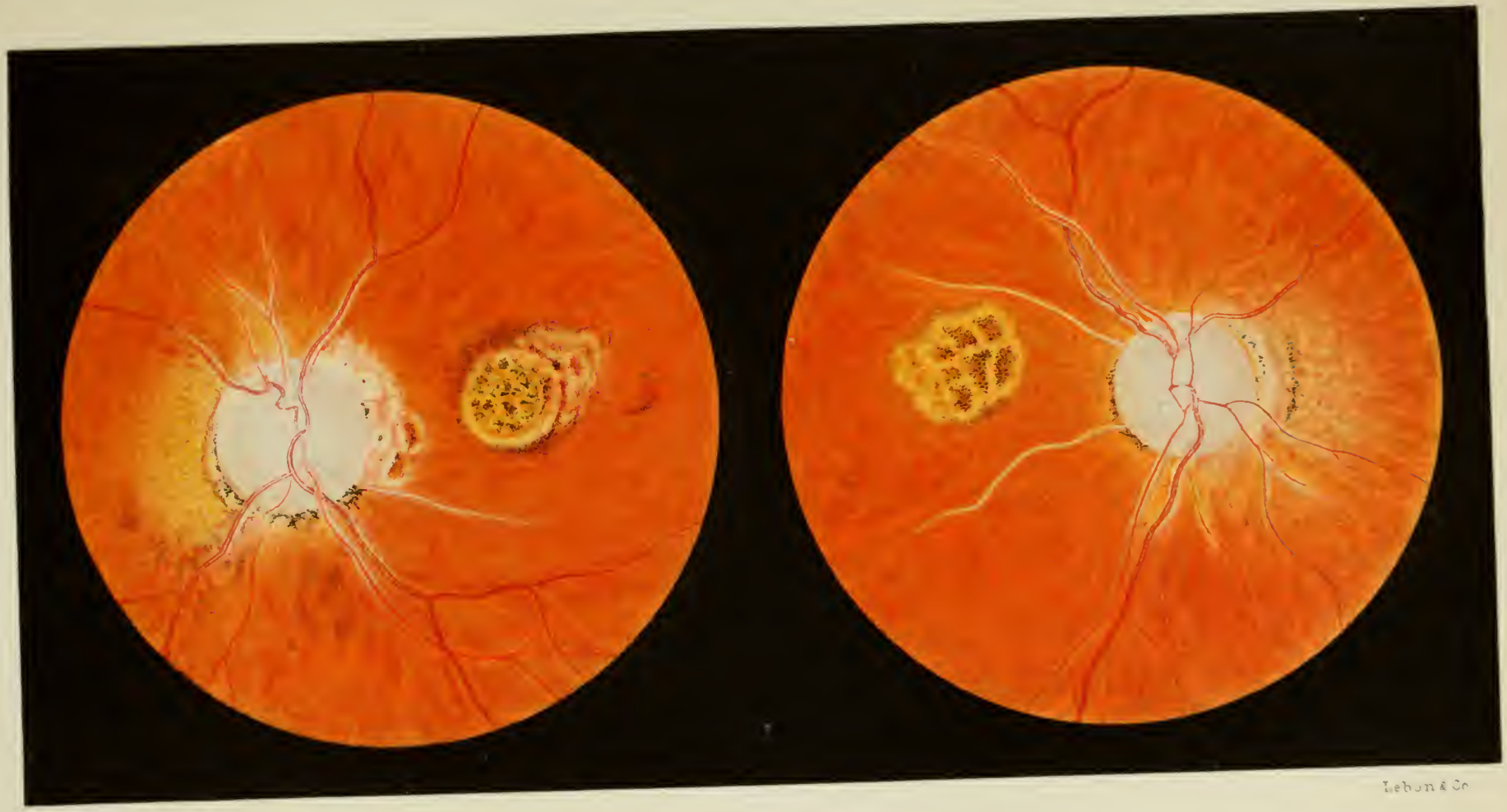
By James E. Adams.

(With Plate VII.)

The case is interesting only as showing certain patches of choroido-retinitis in the maculæ after embolism, which suggests the probability that similar changes may be rather

DESCRIPTION OF PLATE VII.

Plate VII shows the ophthalmoscopic appearances in the inverted image in Mr. Adams's case of Embolism of both Retinal Arteries (p. 114). From drawings by Miss Boole.



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frequent in cases where patients survive the occurrence of the blocking of the vessel for a sufficient length of time, a supposition that will lead us to examine our old cases of embolism carefully, after long intervals.

Joseph S—, a miner, æt. 61. The right eye became suddenly and totally blind on July 5th, 1871, and has remained so ever since, the left following in a precisely similar manner on August 24th, 1881.

Present state.—Pupils act very slightly. External appearances normal. Media clear. He has no p. l. No cardiac murmur. History of slight rheumatic pains. No gout or syphilis. The retinal arteries in each eye contain scarcely any blood, and many of them are quite threadlike (see Plate VII). Here and there the margins of the veins present a distinct white band (old inflammatory changes), and one larger trunk in the left presents a beaded appearance. There are marked traces of old neuro-retinitis at the disc margins, and each macula is occupied by a well defined circumscribed patch of choroido-retinitis, the changes being more defined, circumscribed, and advanced in the right.

(*March 8th*, 1883.)

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9. *Foreign body (a piece of steel) embedded at the fundus near the macula with preservation of almost perfect vision.*

By James E. Adams.

(With Plate VIII, fig. 2.)

Robert H—, a smith by trade, was wounded in the left eye in November 1881, by a small triangular chip of steel which flew off the anvil upon which he was striking. The sclerotic, choroid and retina were penetrated by the fragment about 7 mm. from the corneal margin, and it was seen lying at the fundus at the spot indicated in the Plate, partly concealed by blood and lymph, on the day

after the injury. There was also a peculiar appearance in the vitreous near the wound, resembling a large air-bubble as seen under the microscope.

For some hours after the injury vision was reduced to p. l. (doubtful), but the sight steadily improved until on June 19th, 1882, he was reported to see 1 J. and $\frac{20}{30}$, which now (May 1883) he does perfectly. The point of penetration is barely visible externally, but is indicated within by a large white patch with irregularly pigmented borders. No attempt at extraction of the foreign body, beyond holding the large electro-magnet near the wound, was made. That the dark mass shown in the drawing is the foreign body covered by inflammatory products, there can, I think, be no doubt, taking the history and the appearance immediately after the injury into account, and the patient's positive statement that the fragment broken off was very small.

The patient was not under my observation at the time, and it is much to be regretted that the notes are very incomplete, and especially the absence of a perimetric chart is to be deplored. Taken, however, as it is, the case may be usefully considered in connection with the series of similar ones collated by H. Knapp ('Archives of Ophthalmology,' for 1882, p. 221), and the very excellent, comprehensive remarks appended thereto. In the absence of the much desired documents the illustration (the original of which was perfectly accurate) may afford some information as to the probable size of the body, and the character of the surrounding changes.

This case, and those recorded by Knapp, cannot fail, I think, to compel us to reflect, whether we have not in times past been somewhat hasty in advising enucleation, and possibly too energetic in endeavouring to extract foreign bodies; at any rate, I am sure that for my own part I shall in future take much more interest in watching and treating cases of this kind than heretofore. One point especially alluded to by Knapp, namely, absolute rest in bed and on the back, with both eyes bandaged and atropised, I am

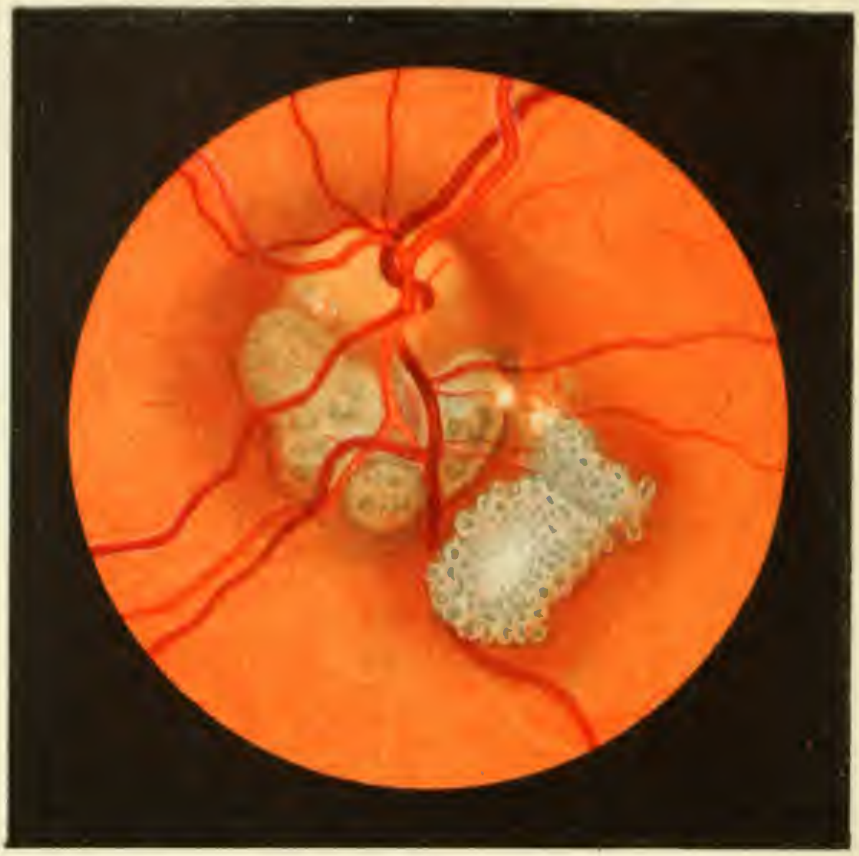

Fig 1

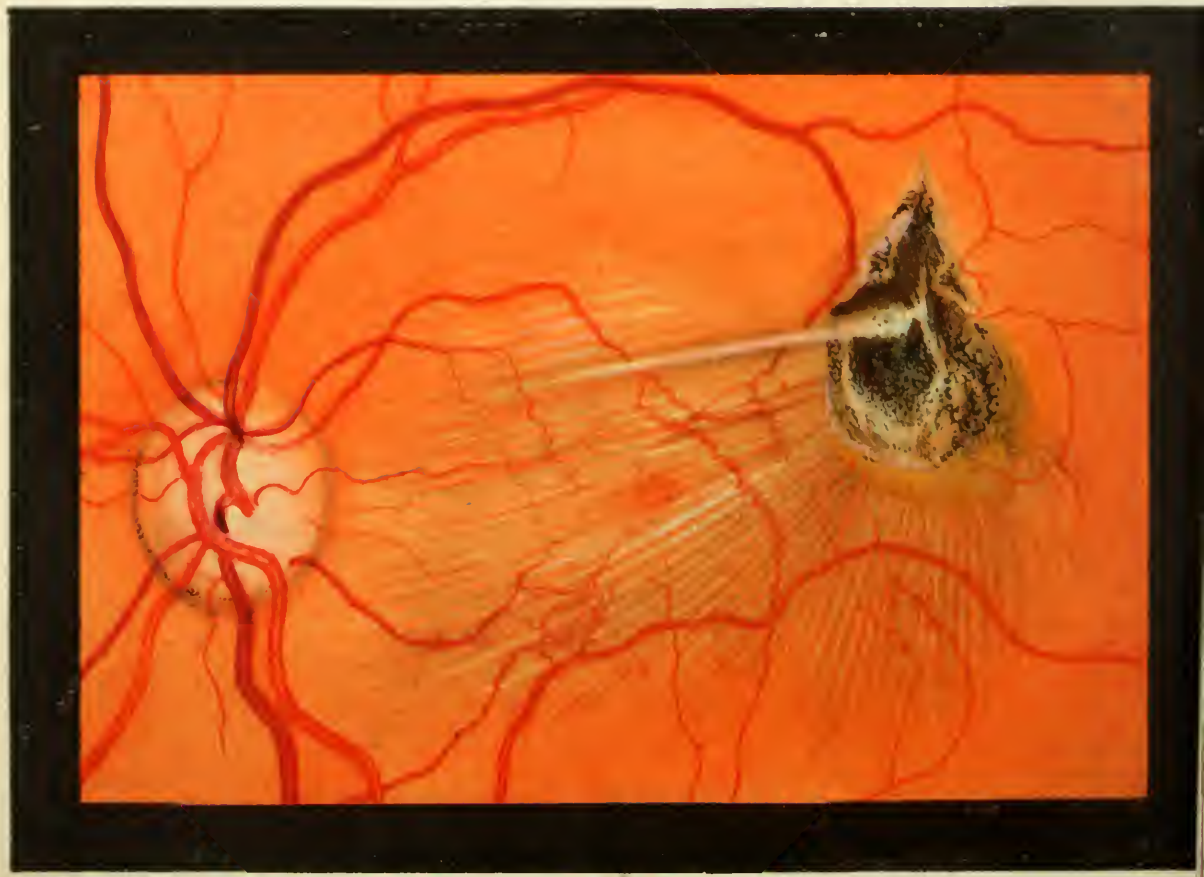

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Fig 11

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DESCRIPTION OF PLATE VIII.

FIG. 1 shows the ophthalmoscopic appearances in the right eye in Mr. Lawson's case of Bead-like Growths in Syphilitic Choroiditis. Erect image (p. 117). From a drawing by Miss Boole.

FIG. 2 shows the ophthalmoscopic appearances in Mr. Adams's case of Foreign Body embedded near the Yellow Spot in the left eye. Erect image (p. 115). From a drawing by Miss Boole.

sure has not been sufficiently carried out. It is, however, still to be feared that the number of recoveries with useful sight after these injuries will constitute a very small proportion of the whole.

(*July 6th*, 1883).

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10. *Case showing a rent in a detached retina.*

By Arthur H. Benson (Dublin).

The drawing exhibited showed a recent spontaneous detachment of the retina, with a well-defined rent in its structure. The case is of interest in regard of Leber's theory of the mechanism of spontaneous detachment of the retina. According to him the first thing to occur is a hyalitis, cicatricial contraction of the vitreous follows and produces the rent in the retina, behind which the fluid and diseased vitreous forces its way and produces the detachment. In this case the history is of a very sudden blindness coming on without injury.

(*Card specimen. March 8th*, 1883.)

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11. *Syphilitic choroido-retinitis with peculiar growths at the fundus.*

By George Lawson.

(With Plate VIII, fig. 1.)

William Smith, æt. 23, farm servant. Twenty months ago the patient noticed his sight failing gradually and equally in both eyes. One month later he received a blow with the fist on the left eye (no black eye), after which the left failed more rapidly than the right. Thirty

months ago he had two sores on the glans penis, beginning as pimples, gradually spreading, but never becoming deep, they lasted for three months. Two months later he had an eruption between the thighs, lasting for a fortnight, and twelve months later an eruption over the scalp lasting three weeks. Has been subject to sore throat, but there is no ulceration now. Had inflammation of lungs twelve years ago. *Family history.*—Father, mother, one brother, and two sisters all healthy, and all have good vision.

Vision: R. = 10 J. and $\frac{20}{200}$; L. = 16 J. and $\frac{20}{200}$. Vision is markedly defective at night-time.

Right eye.—Punctate opacities of lens; large floating opacities in vitreous; disseminated choroido-retinitis; diffuse haze of retina, with patches of pigmented choroiditis chiefly at the outer periphery. Upwards and outwards from the disc is a large, white, translucent patch, composed of very numerous confluent bead-like bodies, looking like the grains of a psammoma (see Plate VIII, fig. 1); on some parts of the surface of the mass are distinct golden spangles of cholesterine. The mass projects forwards as is shown by its overhanging a large retinal vein, and proved also by ophthalmoscopic measurement, the refraction at the most prominent part of the mass being H. 2·5 D., at the neighbouring parts Em. The margin of the disc is about half surrounded by a somewhat similar deposit, translucent but not beaded.

In the *left eye* two patches of the confluent beads are present, also near the disc, but the patches are smaller. The larger is seen with +3 D. There is diffuse choroido-retinitis, chiefly at the outer periphery, with much pigmentation of the choroidal patches. Near the disc is a small linear hæmorrhage in the retina.

The patches have undergone no visible change during the many weeks that the patient has been under observation. He is a healthy-looking man, a native of Devonshire.

(*Living specimen. July 6th*, 1883.)

12. *On a case of chronic tubercle of the choroid and brain.*

By Stephen Mackenzie, M.D.

(With Plate II, figs. 4 and 5.)

The disease occurred in a female child aged four years.

The patient's parents were alive and strong, and the mother's parents living. Besides patient there were three other children who enjoyed good health; two have died: one at 13 months from so-called "consumption of the bowels" (chronic gastro-enteritis), and the other at 18 months, from bronchitis. No history of phthisis in the family.

The patient had chicken-pox when a year old. Eighteen months before coming under treatment she had hooping-cough, since which time she had been weak and ailing. During this period she had suffered from frontal headache and drowsiness, and often had feverish attacks and frequent diarrhœa, with occasional vomiting in the morning. She lost appetite and flesh, and did not care to join in any games. At the end of the preceding summer she had swelling of the right knee, for which she was treated at a children's hospital with Scott's dressing for two months, but at the end of that time there was no improvement. On the bank holiday (Aug. 2nd, 1880) she went out for a walk with her sisters, and on coming home she said she was tired, and lay down. When asked to come to tea she was noticed to grope about, and she complained of headache. She was soon put to bed, and the next morning on getting up was thought to be worse. Her mother now tried her sight by asking her to pick up things from the floor, but found she had a difficulty in seeing them; she also "seemed queer," and cared for nothing but lying down. One week later she was taken to the Eye Hospital, Moorfields, and after attending three times was advised to go to the London Hospital. For some time past she had been getting pale

and thin, and used to mumble and groan in her sleep. When two years old had a bad fall down ten or eleven stairs, but up to the time of the hooping-cough enjoyed very good health and was very stout and strong.

She was admitted under the care of my colleague, Mr. Waren Tay, who subsequently transferred her to my care.

When coming under my observation on Aug. 21st, 1880, she was a fairly well-nourished child, with light brown hair and grey irides. She was a little flushed on both cheeks, and had a heavy look. She lay remarkably quiet all day. The abdomen was distended, everywhere resonant; bowels relaxed. There was slight tenderness in both flanks and opposite the ends of the eleventh rib on each side. No abnormal signs or sounds in chest, which was well made. Pulse 120. Temp. 101° F. She complained, when asked, of frontal headache. Vertigo was inquired for, but she could not be made to understand what was meant. She slept well. There was no vomiting, paralysis, or tremor. Smell and taste good; no impairment of hearing. Sight was *nil*. She could not distinguish light from darkness, nor see a lamp held within half a foot of her eyes.

Ophthalmoscopic appearances.—Left eye: Papilla prominent, margins indistinct and œdematous. Veins tortuous, not very large, arteries of medium size. In the region of the yellow spot was a patch of choroidal disease. It was larger than the disc in diameter. Its centre was yellowish-white and somewhat lustrous, surrounded by an irregular thick zone of black pigment, and at the circumference was another zone of pigment much less in amount than the internal one, marginating most of the patch. Below and slightly to the outer side of the disc was a circular white patch with tolerably defined borders, slightly pigmented at the outer margin. This patch was about the diameter of the disc, and crossed by a retinal artery and vein which did not appear displaced or distorted. The patches projected little, if at all.

Right eye: Papilla swollen and veins tortuous. There

was a small white patch in the upper-outer quadrant, circular in outline and very much smaller than the patches in the other eye.

Urine sp. gr. 1012, one tenth albumen. Urine and fæces passed involuntarily the first two or three days.

She was ordered a mixture of iron, bismuth, and chalk.

She remained with much the above symptoms for about ten days, the temperature being about 100·5° F. on the average.

On Sept. 13th it was noted "for the last three days she had complained of pain across the forehead. She would not sit up, and did not care for food. On two occasions she had vomited, and twice passed her urine under her. The diarrhœa had ceased. Her skin was hot. Temp. 101·2° F. Cheeks flushed. An ice-bag was applied to the head, and 10-grain doses of bromide of potassium given.

14th.—Seemed better. Had taken more to eat, and only passed urine in bed once, calling the nurse two or three times when she wanted to micturate or defæcate. Said she had no headache.

15th.—Very fretful. Could not be got to sit up in bed or to take anything. When asked, said she had no headache. No sickness. Slept a good deal, and passed urine and fæces under her. No fresh ophthalmoscopic signs. Still well-marked papillitis. Ordered one grain of iodide of potassium every three hours.

18th.—Lay perfectly quiet, with eyes almost closed, and was with difficulty roused. Knew her mother yesterday, but took very little notice of her. If moved became fretful and cried. Vomited repeatedly yesterday. Skin hot. Morn. temp. 100·5°, even. 102·4°. No *taches cérébrales*. Complained of headache.

19th.—Had several convulsions in the night. The nurse said they were chiefly right-sided, and not very violent. Some of the later ones were on both sides. This morning she lay in a state of stupor, knew no one

and did not speak. Was very sick in the morning. Temp., morn. 101°, even. 99°; respiration natural; pulse good.

20th.—Takes no notice of anything. Face much flushed, and lies with eyelids partially open. Temp., morn. 99·2°, even. 100°.

22nd.—Yesterday morning the temperature was 101·5°, and in the evening 104°, the highest since admission. She was sponged several times, but the temperature had a tendency to rise rapidly. She was not so flushed, seemed better, could answer questions, and put out her tongue, which was covered with a white fur through which the papillæ showed. Urine and fæces still passed involuntarily. No fresh changes in fundi.

23rd.—During the night she was sponged every two hours, her temperature being reduced to 100°, and rising betweenwhiles each time to 103°; once it reached 104·8°. Slight diarrhœa. She lay on her back with her eyelids open, and the globes directed a little upwards. There were constant movements of the lips, tongue, and jaw, of a co-ordinated character. There was a slight tremor or twitch of the limbs, chiefly left-sided, especially during inspiration. *Taches cérébrales* well marked.

24th.—Right-sided convulsion in night. Breathing slightly cerebral (Stokes-Cheyne respiration). Movements of lips, tongue, and jaw not present, but the mouth was a little more open, and the lip more raised on the right side than on the left. Tremor of left arm much less. On being asked to put out her tongue opened her mouth, but did not protrude tongue. Temperature at 9 a.m. 104·2°, in spite of sponging every two hours; at 5.30 p.m. 103·9°. To take Pot. Iod. gr. iij every two hours.

25th.—Convulsion in night. Much tremor of left side; slight of right. Lay with eyelids open, globes directed upwards. *Taches cérébrales* marked. The greater part of the skin had a red mottled appearance, disappearing on pressure. No fresh changes seen in fundi. Temperature at 8 a.m. 105°, at 10 p.m. 102°.

26th.—Patient died at noon, comatose and bathed in profuse perspiration. The temperature at 2 and 6 a.m. was 104°, and two hours before death 103·5°.

Necropsy.—Body slightly wasted. Head only allowed to be examined.

The brain when removed was very soft. At the base, a quantity of opaque, yellow lymph covered in the central parts, and extended into the fissure of Sylvius. On opening up the latter, small grey granulations (miliary tubercles) were visible to the naked eye. The membranes at the posterior part of the base of the brain, were opaque and thickened, but there was no lymph at this part, the lymph being confined to quite a central position. In the left ascending parietal convolution was an irregular curdy-looking mass, slightly raised above the surrounding convexity of the brain. It felt hard, and on cutting into it, it was found to extend irregularly into the brain beneath, the greatest depth being about a quarter of an inch. In the other parts of the convexity on both sides were isolated yellow nodules, rather smaller than a hemp seed, from ten to twenty in number. On the surface of the cerebellum was a nodule about the size of a kidney-bean, which when cut into was found to extend for a considerable distance into the right lobe, and probably involved the middle lobe. In the right corpus striatum was a small nodule, about the size of a pea, and in the posterior hemisphere was a similar sized nodule. The lateral ventricles were slightly enlarged, and the interior of the brain was very soft. The pons appeared healthy. The medulla contained a small tubercular nodule about the size of a millet-seed, rather above the left olivary body.

These masses, wherever situated, were firm, cut hard, and with a sharp edge; and the section presented an appearance resembling raw potato. None were breaking down.

The backs of the eyeballs were removed. In the left a white patch surrounded by a black border was seen, to the outer side of the papilla, and in the right fundus there was a small white nodule.

Microscopic examination of the masses from various parts of the brain show the characteristic appearances of aggregated tubercle. In each the centre is necrotic, fibro-granular, and more or less free from cells that stain with hæmatoxylin; whilst the circumference is made up of clustered masses of cells, some irregular in shape, large, with one or more distinct nuclei, others small and round, and scattered amongst these are multi-nucleated giant-cells (Plate II, fig. 5). The pia mater is found everywhere occupied with tubercular infiltration, surrounding the vessels, and dipping into the subjacent cerebral tissue, and the vessels penetrating into all parts are surrounded by small round cells. This is very conspicuous in the medulla and pons.

The trunk of the optic nerve is infiltrated with round cells throughout the whole length examined (about one inch). In one place a slight effusion of blood has occurred. The inter-vaginal space contains a large number of exudation cells, in some places aggregated into heaps, but there is no appearance of tubercular formation at any part examined.

The retina is slightly swollen, the vessels surrounded by collections of round cells, and in places there is a protruding column of cells from the subjacent choroid.

The superficial layer of the choroid is thickened, owing to the presence of a number of small round cells, and in places tubercles containing giant-cells are present. The tubercles appear to involve the upper layer especially, slightly pushing up the retina, and are surrounded by dense granular pigment (Plate II, fig. 4). In one place is a small hæmorrhage into the choroid. Thin sections of the growing edge of the aggregated tubercles of the brain, and of the diseased choroid, have been examined for bacilli by Ehrlich's process, and by that of Dr. Heneage Gibbes. The latter has also himself examined some sections of the former. No bacilli have been found.

I am indebted to Dr. Walter Edmunds for making some of the sections.

Remarks.—Choroidal tuberculosis is sufficiently uncommon in medical practice to merit the record of individual cases.

In the present instance, the detection of the tubercular disease in the choroid was of some value, aiding the diagnosis as to the nature of a coexisting encephalic lesion, but unfortunately the kind of disease was such that this knowledge was of little service in treatment.

That some intracranial disease was present was indicated by the double papillitis, headache, occasional vomiting, drowsiness, &c.; and that it was probably tubercular by the age of the patient, the feverish attacks, pallor and wasting, and "white swelling," but these indications were strengthened by the appearances observed in the choroid. The few and isolated changes in the choroid, and their lack of resemblance to the ordinary forms of choroidal disease, led to the inference of their tubercular character.

Deutschmann, in a series of experiments, has shown that, in artificially excited tuberculosis of the membranes and brain in rabbits, a double-sided papillitis, and tuberculosis of the vitreous and choroid supervened. "The cause of the ophthalmoscopic changes was proved, by post-mortem examination, to be, in all cases, a tuberculous affection of the sheaths of the optic nerves, derived from the inoculated tuberculosis of the brain and its membranes, not by immediate propagation *in continuo*, but by metastasis. For as the first tubercles deposited in the sheaths were found at the entrance of the optic nerve into the eye, *and only there*, it would seem that the germs must have passed from the tuberculous foci in the brain into the sub-vaginal space, and have been carried along this space, meeting with no resistance until they were stopped at the scleral foramen, where they then set up the infective process. The infection was found to have taken place sometimes on the inner, sometimes on the outer sheath."*

* Deutschmann, "Miliary tuberculosis of the brain and its membranes, and

Deutschmann also found in a child, who died from tubercular meningitis, after twenty-three days illness, small round nodules in the inner sheath of the optic nerve, which he believed to be tubercles.

The sheaths of the optic nerves in the present case have been examined with care and interest on this account, but though inflammatory changes of a pronounced type are present, there is no evidence of the existence of tubercle there. Whilst it is important not to overlook this mode of propagation from the brain to the eye or *vice versâ*, of course it is not necessary to assume this mode of extension, as tubercle appears in parts widely apart, and without the means of extension open to it from brain to eye.

In conclusion, the case is interesting from the similarity of the changes in the brain and eye, illustrating that the appearances in the latter are not only useful in diagnosis, but valuable in showing us pathological processes. In the brain and membranes were the aggregated tubercle and inflammatory changes, and in the eye, the tubercle and optic neuritis.

(*October* 12*th*, 1882.)

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13. *A case of tubercles in the choroid in general miliary tuberculosis, with no meningitis.*

By Francis Warner, M.D.

(Communicated by Stephen Mackenzie, M.D.)

Florence E—, æt. 9, was admitted into the London Hospital, July 20th, 1880, complaining of abdominal pain with headache.

She had not been well for three months, and this was attributed to grief at the loss of a sister from consumption.

the connection of this disease with affections of the eye: an experimental study" (Von Graefe's 'Archiv,' xxvii, 1, 1881), Review in 'Ophth. Rev.,' vol. i, No. 1, p. 13.

The girl was intelligent, rather thin, slightly pale with a malar flush. Respiration was markedly diaphragmatic, 80 to the minute; pulse 120; temp. 99·2° F. A few crepitations were heard over the apex of the right lung, but there was no dulness. No enlarged glands were found except a few under the jaw. The child continued in much the same state, weak, ill, and feverish; she complained of abdominal pain; the bowels were constipated.

July 28th.—Temperature between 102 and 103° F.; there were more crepitations over the right lung, but there was no dulness; respirations 84; pulse 132, small and weak. Mr. Lloyd Francis, my house physician, reported that he had seen in the eyes some light coloured, cloudy spots.

I examined each eye under atropine and took the following notes:

Left eye: Disc appeared quite healthy, and the vessels could be distinctly traced over it. At a little distance from the disc two or three small spots were seen near one another, varying in size, of a light whitish colour, they appeared to be raised; at one point a retinal vessel appeared to have been pushed aside in its course, and here it became slightly indistinct, though clear on either side of the projection.

In the right eye the disc was reddish, but the vessels were not at all obscured, the light streak on the arteries was very distinct. A distinct rounded spot was seen in the choroid with a fairly defined margin, it was darker in colour than the spot in the left eye but was surrounded by a lighter zone. There was also another ill-defined spot, light in colour and apparently raised. Next day several light shining choroidal tubercles were seen, over one an artery could be traced, around another there was some pigmentary disturbance. There was no vomiting, no paralysis, no convulsion, and the child was quite sensible, in fact there were no signs of brain disease.

She died August 3rd.

At the *post-mortem* miliary tubercles crowded the lungs, others were found on the surface of the liver and spleen,

and also in the substance of the kidneys. There was a small ulcerated patch in the bladder about the size of a pea and apparently tubercular. There was no cerebral meningitis and no increase of the cerebro-spinal fluid. Over the third left frontal convolution, immediately beneath the pia mater, was a dull, greyish-white, vascular patch the size of a pea, and a similar mass was found in the left optic thalamus.

The posterior portion of each eyeball was removed and examined both by myself and by Mr. Nettleship. We have no doubt, as far as naked-eye appearances go, that the choroidal spots are miliary tubercles, more abundant in the right eye than in the left. No microscopical examination was made.

This case illustrates the association of miliary tuberculosis of the choroid and of the lungs without meningitis, this being probably a more common association than that of the choroid and meninges.

Dr. Garlick * has tabulated twenty-six cases of tubercular meningitis, and concludes by saying, "I have only once seen tubercles in the choroid."

Dr. Gowers, in his work on medical ophthalmoscopy states that "practically choroidal tubercle is confined to cases where tubercle is widely distributed, its presence is evidence in a febrile case of probably widely-spread tubercle. Choroidal tubercle is more common in cases of tuberculosis without meningitis, than in cases with meningitis." The case given will illustrate these remarks.

De Wecker in his work, 'Ocular Therapeutics,' on the contrary, says, p. 238, "If in miliary tuberculosis the meninges are attacked, there will generally be a corresponding deposit in the choroid." Both eyes are usually affected; the probability of tubercle being found in the choroid is in proportion to the rapidity of its evolution. De Wecker recognises, however, that choroidal tubercle is a sign of tuberculosis rather than of meningitis.

(*October* 12*th*, 1882.)

* 'Med.-Chir. Trans.,' vol. lxii (1869).

14. *Case of tubercle of the eye resembling in some of its clinical aspects a retinal glioma.*

By W. A. Brailey, M.D.

John H—, æt. 2, under the care of Mr. Couper at Moorfields in March, 1882, was a stout, healthy-looking, hearty child, who had had a purulent discharge from the left ear since infancy.

At birth the parents thought the right eye somewhat the smaller, but during the last three months it has grown rapidly larger. Now, together with the general increase of the globe, there is a more than proportionate enlargement of the cornea. The anterior chamber is deepened, and the iris is much stretched from numerous point-like posterior synechiæ, leaving a medium-sized, clear, but very irregular pupil. Through the clear lens, can be discerned a greyish, vessel-bearing layer, made apparently of two folds, separated by a horizontal crease, the effect being as if the retina, while still retaining its connections at the optic disc and ora serrata, were bulged forwards in two principal folds. The tension is increased. The *left eye* seems perfectly normal.

On March 17th, 1882, the *right* was excised. The sclera is much stretched and thinned. The optic nerve is not thickened. An equatorial section shows that the greyish layer is the retina which is bulged forwards and detached from the optic disc by abundant, clear, yellowish, subretinal fluid. On the external retinal surface is a loose greyish web, possibly the remains of blood-clot. Projecting from the region of the papilla and immediately surrounding choroid, is a mass the size of a large pea, which reaches forwards nearly as far as the posterior part of the detached retina, from which, however, it appears to be free. *Microscopic sections* show that its main thick-

ness is in the region of the papilla, where it pushes backward the lamina cribrosa, though not extending into the nerve beyond this. The immediately adjacent choroid is much thickened by a mass exactly like that at the papilla. In some sections the two structures are directly continuous, but in others there is a deep crease partially separating the two. A very few isolated patches of similar nature are found in the adjacent sclera and even on its deeper surface.

Histologically the new formation is composed of staining nuclei, each surrounded by a zone of unstaining matter. The majority of the bodies thus constituted are round, but there are many tracts in which their general shape is oval. A few thin-walled, distended blood-vessels are visible in the mass, but generally it is little vascular. Many small areas of apparent degeneration can be traced in it. The most central part of these may be either finely granular, representing probably the stage of caseous degeneration, or structureless with some embedded, pale, brightly refracting nuclei, the whole constituting in this case a so-called giant cell. The outermost part of each area is constituted by a zone which stains rather more than the mass generally, but when examined minutely its cell elements are seen to be smaller, less defined, and more granular than those of the intervening tissue. They even stain less, the higher colour of the zone being due to their more close aggregation.

The child's father is said to be very healthy and strong. Its mother was taken ill with consumption fifteen months, and died much wasted six months, before the child was seen. The child looked then very thin, but had picked up very much at the time of the operation. Owing to the migratory habits of the poor, or to the custom of giving false addresses at hospitals, I have, I regret to say, obtained no answer to my inquiries as to the after-history of the case.

The patient is the second child. The eldest, a girl,

is two years older. Before her birth there was a miscarriage at the fourth month.

It appears impossible to say whether this new formation, which has the structure of tubercle, originated in the choroid, or in the nerve tissue of the papilla. Judging, however, from our experience of other cases we shall probably decide for the former, notwithstanding that the thickening is less where the choroid is undoubtedly affected, than over the papilla, and that the thickened choroid tapers off very rapidly into the adjacent unaffected choroidal tissue.

(*October* 12*th*, 1882.)

Dr. Coupland.—Speaking solely from my experience in the post-mortem room at the Middlesex Hospital, I may say that, although I have never met with choroidal tubercle apart from tubercular meningitis, yet in each instance (six cases in all) the tuberculosis was more or less general. The meningitis in these cases conformed to the general rule in being but one manifestation of the tuberculosis—and in this respect the choroidal affection stands in the same category. Thus I should prefer to say that there is no necessary connection between the choroidal and meningeal conditions, either may be present (or absent) as part of the general infection; and their association in any case is purely accidental. Nor is it difficult to explain the apparent infrequency of choroidal tubercle apart from tuberculosis of the pia mater, since it is not customary to examine the eyes either during life or after death except in cases that have shown cerebral symptoms. This explanation was long since borne out by the researches of Cohnheim,* who was one of the first to study the pathological aspect of choroidal tubercle. Examining after death the eyes of subjects of tubercular

* Referred to in a paper I communicated to the Pathological Society in 1873 (' Path. Trans.,' vol. xxv, p. 215). See ' Virchow's Archiv,' Bd. xxxvi, p. 448, and also a paper by A. von Graefe and Th. Leber, 'Archiv für Ophthalmologie,' Bd. xiv, p. 183.

disease (many being cases of pulmonary phthisis), quite irrespective of the presence of meningitis, he found choroidal tubercle to be very frequent, and I regret not to have seized what opportunities I have enjoyed in verifying his statements, which are certainly remarkable. I may add that choroidal tubercle is often caseous, and I have recorded in the 'Lancet' (1879, ii, p. 277) a case where a solitary tubercular nodule much resembling that described to-night by Dr. Mackenzie occurred in the choroid of one eye.

Dr. E. B. Baxter said that it was most desirable to make out what precisely was the common association of tubercle of the choroid. During the last twelve years, he had examined the eyes in a large number of cases of tubercular meningitis, but had never seen choroidal tubercle, with the ophthalmoscope, in any of them. Papillitis more or less decided was usually observed. He had only seen tubercles in the choroid on two occasions; both patients succumbed to miliary tuberculosis of the lungs and other viscera, without meningitis.

Dr. Barlow said:—It appears to me, sir, with respect to the frequency of the co-existence of choroidal tubercles and tubercular meningitis, that it is imperative that the question should be brought to the test of post-mortem examination. Everybody who has been in the habit of examining, during life, the eyes of children the subjects of tubercular meningitis, must be aware of the enormous difficulty of asserting a negative, as far as choroidal tubercle is concerned, and, with the greatest respect for ophthalmoscopic observations, I should decline to accept any statistics in which a negative was not verified by removing the backs of the eyes after death and carefully examining them. Out of 16 cases of choroidal tubercle of which I have notes, in 13 tubercular meningitis was present, whilst in 3 there was tubercle in other organs, but none appreciable in the meninges. There appears to be in the minds

of some a latent doctrine that for the growth of tubercle of the choroid a longer time must be allowed than is represented by the ordinary duration of a case of tubercular meningitis.

I think it would be very difficult to put any limit on the possible rapidity with which tubercle may grow. I can certainly testify to having seen a tubercle of the choroid during two or three days show a distinct change, viz. that a minute opaque spot appeared in the centre of it like a nucleus, and that after post-mortem examination microscopic examination of this tubercle presented in the centre a granular, ground-glass, degeneration which, I presume, corresponded with the appearance of the central opacity during life.

I will also mention that in at least three cases of tubercular meningitis there was during life a very fine, dappled appearance to be seen in the fundus with the ophthalmoscope, and that post mortem there were some minute, white, raised dots, along with larger and undoubted choroidal tubercles. I have no doubt that these dots were commencing tubercle and that they might very justly be compared with what Barthez and Rilliet have called "tubercular dust" in the liver.

I cannot resist the conviction that minute tubercles of the choroid would be found, if carefully looked for, in many cases of tubercular meningitis. On the other hand, it seems to me quite as important to keep one's mind open to the occasional long duration of choroidal tubercle, as illustrated by Dr. Mackenzie's important case.

In two, at least, of my cases the tubercle, besides occurring in discrete nodules, has been present in what might be called a small botryoidal mass, obviously formed by the partial coalescence of adjacent nodules. Also, just as I believe there is evidence that choroidal tubercle may remain latent for a considerable period, so I do not doubt (though it is difficult actually to prove) that a local group of miliary tubercular nodules may remain latent in the pia mater for a considerable period.

I may, perhaps, refer to a more diffuse, massive, and chronic form of tubercle of the choroid, a case of which came under my observation about six years ago.

A boy, eight years old, was brought to me at Great Ormond Street on account of occipital headache, which had lasted on and off for six months, and vomiting which had repeatedly recurred for one month. Three months after this, when seen again, he had the barest perception of light with the right eye. I found an opaque greyish reflex in the lower part of the right fundus with branching retinal vessels in the upper part. There was scarcely any increase of tension and the mobility of the eyeball was not affected. Both eyeballs had always been prominent; perhaps the right was a shade more prominent that the left. In the left fundus there was very marked optic neuritis. It appeared probable that the boy had some form of cerebral tumour which had given rise to his headache, vomiting, and optic neuritis, but whether secondary to, or concomitant with, or subsequent to the growth which was suspected to exist in the back of the right eye seemed indeterminate. There was no reason to think that the growth in the right eye gave the boy any trouble, but as two months later there appeared much vascularity of the conjunctiva in the lower part, and a considerable swelling of or beneath the sclerotic, my colleague, Mr. Howard Marsh, consented to excise the eyeball. There was found a large hemispherical mass growing from the choroid, extending from the optic disc to the ora serrata, at one small spot below it had perforated the sclerotic. The cortical portion was semi-gelatinous; the central part caseous. On microscopic examination by my friend, Mr. Nettleship, it was found to present the characters of confluent tubercles. The wound healed and there was no recurrence of the growth. The optic neuritis in the other eye progressed to atrophy. The vomiting and headache varied considerably, and after a time ceased, but the boy gradually developed hydrocephalus and suffered much from pains in the back and limbs.

When he died, thirteen months after the operation and rather more than two years from the first onset of symptoms, I found, on post-mortem examination, caseous bronchial glands, several caseous scrofulous tumours in the brain, viz. one in the middle lobe of the left hemisphere, one in each temporo-sphenoidal lobe, and one in the middle of the cerebellum, with much internal hydrocephalus, but no other tubercle in connection with the remaining eyeball, nor indeed, any small nodules of miliary tubercle anywhere.

If I may be permitted to sum up the gist of my observations it is this :—

(1) Cohnheim's generalisation that tubercle of the choroid exists more commonly in cases of general tuberculosis than in tubercular meningitis, ought not to be taken as final, for (*a*) tubercular meningitis, is often only a part of general tuberculosis, and the distinction therefore is not a good one, and (*b*) of the cases which I have given in thirteen out of sixteen, tubercle of the choroid co-existed with tubercular meningitis.

(2) No statistics on the absence of choroidal tubercle in cases of tubercular meningitis should be accepted without a post-mortem examination of the backs of the eyes because (*a*) of the general difficulty of accurate ophthalmoscopy in such patients, and (*b*) the special difficulty of excluding very minute tubercles, which I have ventured to compare with the tubercular dust of Barthez and Rilliet, and which certainly sometimes co-exist with unquestionable choroidal tubercular nodules.

(3) That in the choroid we ought to be prepared for considerable variety of tubercles, viz. (*a*) minute nodules in which we may see changes from day to day, (*b*) semi-confluent nodules, (*c*) diffuse massive deposits caseating in the centre, quite comparable with scrofulous tumours of the brain, and perhaps comparable with *a* and *b* in the same way as some caseous lobular pneumonia is with miliary tubercles of the lung.

VIII. DISEASES OF OPTIC NERVE.

1. *Sequel of a case of optic neuritis, with numerous, sudden, short attacks of complete blindness.**

By Samuel West, M.D.

Edith K—, æt. 19, was a patient under Mr. Morton in January, 1881. Her father died, æt. 51, of "chest disease"; mother died of cancer of the throat, æt. 55. In October, 1880, the patient suffered from neuralgia in the forehead, off and on, with singing in the ears. She was under her own doctor, and was treated for chlorosis.

In January, 1881, she consulted Mr. Morton, complaining of a "shadow coming over her eyes." She had then singing in her ears, and was chlorotic. Both discs at that time were greatly swollen, although the vision was perfect, 1 J. and $\frac{20}{20}$.

In February, 1881, I saw her. She was in the same general condition, the vision being perfect. Her only complaint was that at times suddenly, without any known cause, "everything became dark" to her. "It was," she said, "as if I had shut my eyes." This temporary blindness lasted a few seconds only, perhaps a minute. It came on under many different circumstances—in the street, at work, reading, &c., and several times she had walked into people. She said the headache which she had previously had was "not like headache," it felt as if it was "inside the head."

In June, 1881, the vision began to fail in the right eye, being 2 J. and $\frac{20}{30}$. The field in this eye was much contracted towards the inner and upper half. The discs were still greatly swollen, and there were numerous

* Reported in Vol. I, p. 121, by Mr. A. Stanford Morton.

glistening irregular patches. The pains in the head were worse, and the noise in the ears, chiefly the left, was very troublesome; pressure behind the ear would stop it.

At the end of July, 1881, the vision in the right eye was completely gone, and in the left was defective. Shortly before this, her sister said she had squinted with the right eye.

In September, 1881, she became an in-patient in the Royal Free Hospital, and was there treated very actively with iodide of potassium and mercury, though these remedies had been pushed at various times throughout her illness, and she had been, I think, three times salivated. She was now completely blind of both eyes, and had lost even perception of light, though occasionally she thought she could still see people's feet as they passed her. This was doubtful. The swelling of the discs still remained, and the patches were large. She was under constant supervision, but no change occurred, except in the gradual appearance of atrophy in both discs as the effusion subsided.

In June, 1882, she came complaining of twitchings in her sleep, but these subsided entirely in a short time with a little bromide of potassium, and were the only symptoms of the kind which she ever had.

On August 30th, 1882, the effusion had completely disappeared, and the discs had become white and extremely atrophic, both arteries and veins being reduced to mere threads.

On November 2nd, 1882, the condition was as follows: —No perception of light. Pupils do not react to light, but freely to movements of eyeball. The eyes diverge, but can fix fairly well. The discs as above.

The patient has visited many of the ophthalmologists of London, as well as several physicians. I have received letters from some about her, and the opinion of others I have heard; and there seems to be a general concurrence of opinion in favour of a tumour in the brain, possibly, it

has been suggested, a tubercular tumour, which has become stationary or perhaps regressive.

The points of interest in the case are :—

1. The long persistence of perfect vision with extreme double optic neuritis (five months).

2. The attacks of temporary, complete blindness which formed the first symptom.

3. The rapidity with which the failure of vision became absolute when once it commenced.

4. The entire absence of any other physical signs than the eye changes.

The diagnosis is a very difficult question, for with the exception of headache and singing in the ears at the time when the patient was very chlorotic, which disappeared as she became stronger, and the slight twitchings which came on for a few days only, about eighteen months after the commencement of her illness, there were absolutely no symptoms pointing to any cerebral lesion; and yet it seems difficult to believe that so severe consequences could be the result of simple neuritis.

(*December 14th*, 1882.)

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2. *Examination of the optic nerves in cases of intra-cranial disease, with remarks on the immediate causation of optic neuritis.*

By Walter Edmunds, M.D., and J. B. Lawford, M.D.

(With Plates X, XI, XII.)

It is now, we believe, generally admitted that the optic neuritis which occurs in connection with intra-cranial disease is a descending inflammation of the nerves. In cases of cerebral tumour, however, it is still a question by what path the inflammation descends from the tumour to the optic nerves. The two explanations which suggest them-

DESCRIPTION OF PLATE X.

Illustrating the paper on the Immediate Causation of Optic Neuritis, by Drs. Edmunds and Lawford (p. 138). From drawings by Mr. M. H. Lapidge.

Fig. 1 shows a longitudinal section of the optic nerve and disc from the left eye of Case No. 17 (cerebellar tumour, p. 147). It shows swelling of the disc and inflammation of the nerve. × 24 diameters.

Fig. 2.—Transverse section of the same optic nerve, immediately behind the part represented in Fig. 1. It shows inflammation of the tissue between the two sheaths and of the nerve, the inflammation of the nerve being greater at the circumference than at the centre. × 24 diameters.

Fig. 1.

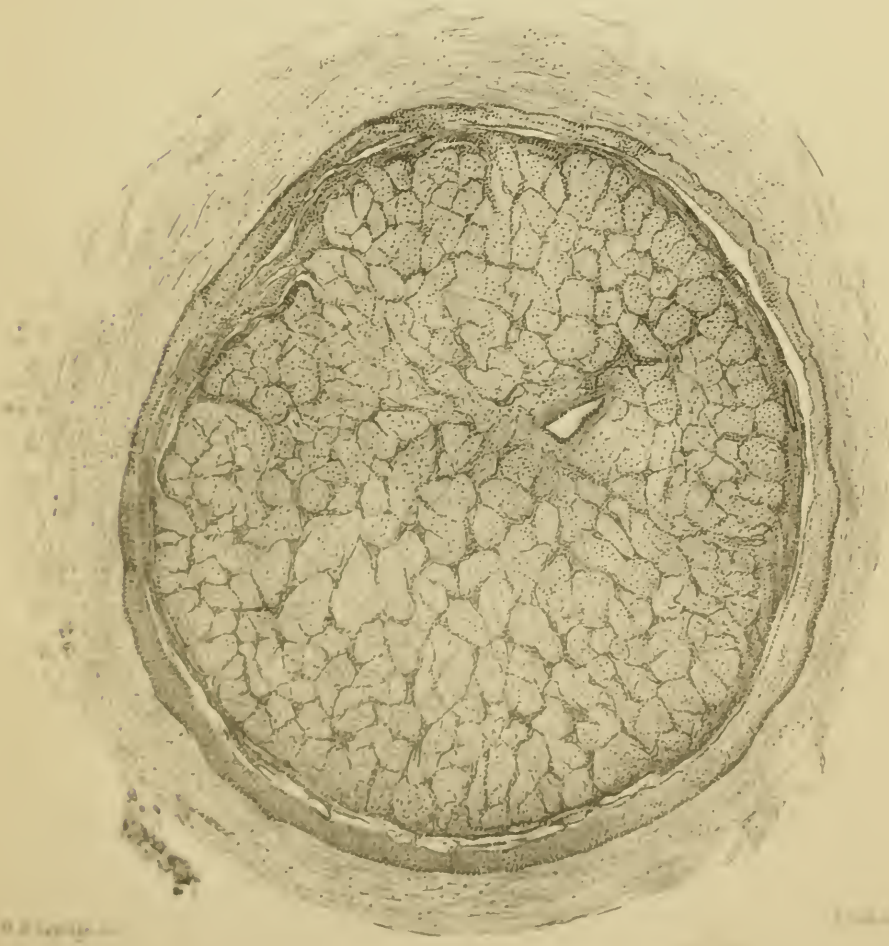


Fig. 2.

DESCRIPTION OF PLATE XI.

Illustrating the paper on the Immediate Causation of Optic Neuritis, by Drs. Edmunds and Lawford (p. 138). From drawings by Mr. M. H. Lapidge.

Transverse sections of the optic nerve, from the same case as the preceding plate (Case No. 17, p. 147, cerebellar tumour).

FIG. 1 shows a part of the circumference of the nerve with the two sheaths and sheath tissue.

FIG. 2 shows the centre of the same transverse section. The two figures taken together show how much greater the inflammation is at the circumference than at the centre of the nerve.

Each is magnified 122 diameters.

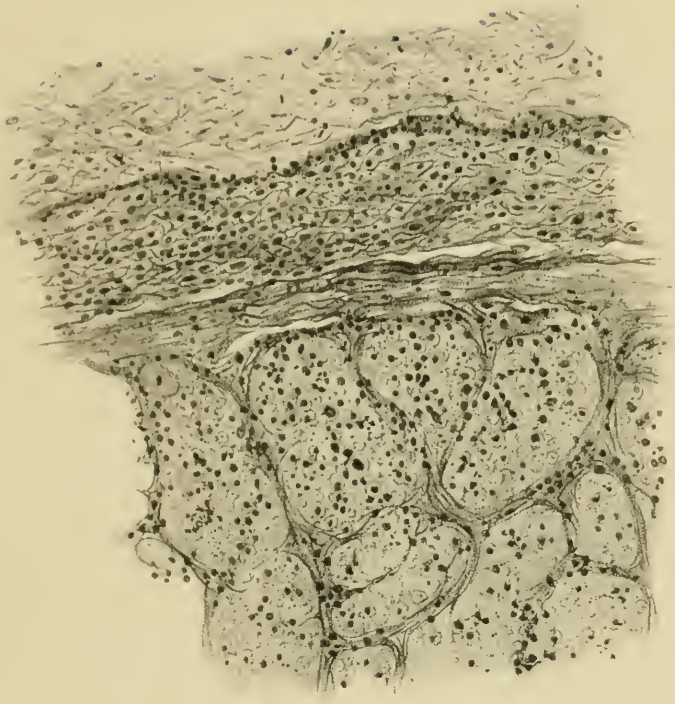


Fig. 1.

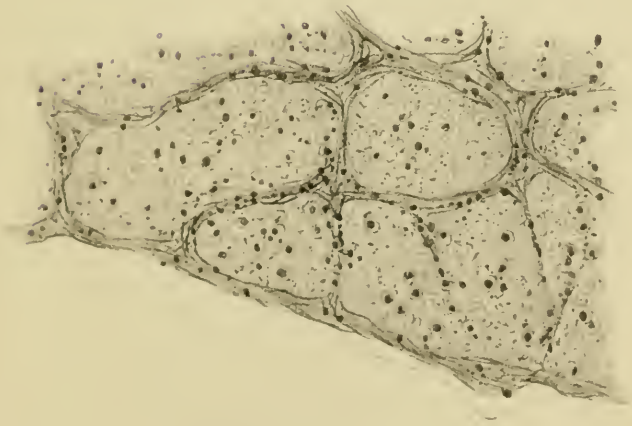


Fig. 2.

M. & N. Hanhart lith.

F. Huth Lith.r Edin.r

DESCRIPTION OF PLATE XII.

Illustrating the paper on the Immediate Causation of Optic Neuritis, by Drs. Edmunds and Lawford (p. 138). From drawings by Mr. M. H Lapidge.

The two drawings are from a transverse section of the optic nerve of Case No. 18 (cerebellar cyst), p. 148.

FIG. 1 shows the circumference of the nerve.

FIG. 2 the centre of the same section.

They show how much greater the inflammation is at the periphery than the centre of the nerve

Each is magnified 122 diameters.

Fig. 1.

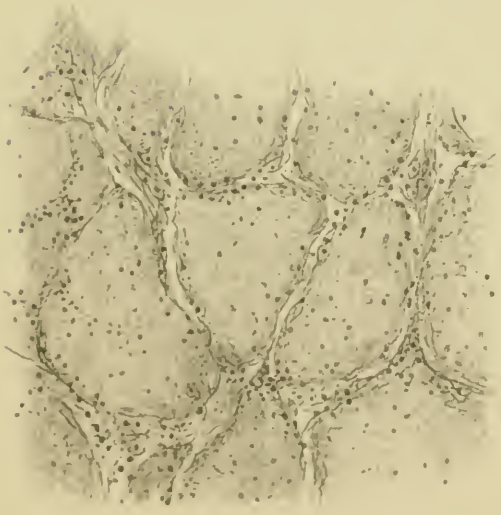


Fig. 2.

selves as most probable are, either that the inflammation extends along the nerve tissue directly, "descending cerebritis," or that it spreads from the tumour to, or is excited by the tumour in, the meninges, and thus reaches the optic nerves indirectly through their sheaths.

At the discussion on optic neuritis in relation to intracranial disease at this Society in March, 1881, Dr. Edmunds concluded his remarks as follows:*—"I submit that the connection between intracranial disease and optic neuritis is to be found in an inflammation extending down the connective tissue and blood-vessels of the nerve; and, conversely, that double optic neuritis with cerebral symptoms means meningitis about the base of the brain, which may be either primary, or secondary to a tumour, or some other disease, of the brain-substance itself."

The object of the present communication is to bring forward further evidence in favour of this view, and to offer some criticism on the objections to it.

We have here very briefly recorded twenty-two cases of head injury and intracranial disease, of all the fatal cases of which, twenty-one in number, we have notes of the post-mortem examination, and of the microscopic examination of the optic nerves and discs in twenty, and in some also of the chiasma and optic tracts. One of the cases (No. 20) has been already recorded by Mr. Nettleship and Dr. Sharkey,† another (No. 1) by Dr. Edmunds.‡ The remaining twenty have all occurred recently at St. Thomas's Hospital, and we desire to express our thanks to the physicians and surgeons under whose care the patients were, for permission to make use of them. The cases were not selected, but taken as they came. It may be well to add that whenever microscopic examination has been made, vertical sections of the optic papilla, and transverse sections of the nerve immediately behind the eyeball and again about the situation of the optic foramen, were examined; also

* 'Trans. Ophthal. Soc,' vol. i, 115.

† 'Trans. Path. Soc.,' vols. xxxi and xxxiii.

‡ 'St. Thos. Hosp. Rep.,' vol. xi.

in some of the cases sections of the commissure and of the optic tracts, close to it, and more posteriorly, where they lie on the crura cerebri. The eyeballs were usually frozen in the orbits, by a mixture of salt and ice, and their posterior halves removed, this method of proceeding causing less disturbance of the structures at the back of the eyeball, than cutting through them when soft. All the specimens were hardened, cut, and mounted in exactly the same way.

Case 1.—Kate C—, æt. 8. (Reported, as already mentioned, in 'St. Thos. Hosp. Rep.,' vol. xi.) Run over by a horse and cart. Died twenty-four hours after injury. No ophthalmoscopic examination.

Post-mortem.—A large laceration of liver, with extravasation of blood. Rupture of the right kidney. Fracture of base of skull in middle fossa. Some basilar meningitis. Optic nerves of a reddish colour and distension of the sheath immediately behind the eye.

Microscopically.—Some swelling of optic papilla from œdema between and in the nerve bundles. The central pit is however not obliterated. Inflamed capillaries are seen passing through the papilla. Distension of retinal vessels with clot. The space between the two sheaths is seen to terminate anteriorly in the usual acute angle showing that there has been no considerable fluid pressure between the sheaths. On examining transverse sections of the nerves there is seen in the outer sheath an increased number of staining nuclei of the tissue, and inflammation of the small blood-vessels and capillaries of the part. The space between the two sheaths, in which there are usually only a few trabecular fibres, was distended by inflammatory products consisting of densely packed cells with some fibres and blood-vessels among them. The inner sheath of the nerve showed less sign of inflammation. The nerve itself was slightly affected; there was inflammation of the blood-vessels passing along the trabeculæ, and this was more marked at the periphery

than towards the centre of the nerve; the blood-vessels in the nerve were full of blood clot.

Case 2.—Emma Mary F—, æt. 2, fell from second story window on to her head. On admission: unconscious, hæmorrhage into scalp. Had two convulsive fits. Died ten hours after injury. No ophthalmoscopic examination.

Post mortem.—Blood effused into and beneath scalp. Fracture extending from left temporal region across vertex to great wing of sphenoid on right side. Hæmorrhage between dura mater and brain, and severe laceration of brain at vertex. No basal meningitis to naked eye. Optic nerves dark coloured from effusion of blood between the two sheaths.

Microscopically.—Optic nerves not inflamed; hæmorrhage between sheaths.

Case 3.—John F—, æt. 23, fell from hayloft. On admission unconscious; stertorous breathing. Bleeding from right ear, nose, and mouth. Died four days after injury, never having recovered consciousness. Note of ophthalmoscopic examination: "? slight changes."

Post mortem.—Blood effused into scalp. Fracture of base of skull along anterior margin of right petrous bone, extending across ethmoid bone. Extravasated blood between dura mater and bone in course of right middle meningeal artery. Some laceration of left temporo-sphenoidal lobe of brain. No note of meningitis.

Microscopically.—Optic nerves exhibit slight inflammation. An excess of staining nuclei chiefly at periphery of nerve, and in intersheath space.

Case 4.—Mary Ann B—, æt. 37, fell downstairs. On admission: unconscious, oozing of blood and serum from right ear. Died two days after injury. No ophthalmoscopic examination.

Post-mortem.—Fracture extending from left frontal bone across base of skull into foramen magnum. Effused

blood between brain and dura mater over left frontal lobe, and brain here contused. No basilar meningitis to naked eye.

Microscopically.—Optic nerves apparently normal.

Case 5.—Henry C—, æt. 23, kicked by a horse. On admission partially conscious, bleeding from left ear. Behind left ear was a compound comminuted depressed fracture of skull, with laceration of dura mater and brain. Elevation performed; died three days after injury. No ophthalmoscopic examination.

Post mortem.—Fracture extended from parietal, across temporal fossa, to middle of sphenoid bone. No basilar meningitis to naked eye.

Microscopically.—Optic nerves apparently normal.

Case 6.—Walter P—, æt. 1½. Eight days before admission one prong of a toasting-fork was stuck three quarters of an inch into his head. No symptoms ensued for four days, except discharge of a watery fluid from the wound; he was then brought to hospital. On admission there was a fine hole into the skull through the left parietal bone, near the situation of the middle meningeal artery; a probe could be passed through this opening into the brain substance. There was paralysis of the right side, and convulsions of that side had occurred.

Ophthalmoscopic examination. — Optic discs normal. Trephining was performed, and a small quantity of pus let out from beneath dura mater. Patient died the next day.

Post mortem.—Whole of vertex of left hemisphere covered with a whitish exudation, which lies on the outer side of the visceral layer of the arachnoid (arachnitis of Hutchinson). No basal meningitis to naked eye.

The optic nerves were obtained for microscopical examination, but were unfortunately lost.

Case 7.—Walter B—, æt. 16, fell from a scaffolding twelve feet high. On admission was almost unconscious,

in a state of cerebral irritation. Paralysis of one external rectus. Died two days after injury. No ophthalmoscopic examination could be made, owing to his extreme restlessness.

Post mortem.—Extravasation of blood beneath scalp over right temporal bone. Fracture on right side, extending from root of zygoma forwards along the squamous suture, and backwards along the parietal and occipital bones. The petro-sphenoidal suture on this side was started. Extravasated blood on right side between dura mater and brain. A branch of the right middle cerebral artery near the Sylvian fissure had ruptured on the surface of the brain. Optic nerves of bluish colour on removal.

Microscopically.—Optic nerves : extravasation of blood into external sheath, and between the two sheaths. Slight increase in the number of staining nuclei in the nerves and trabeculæ. Trabeculæ not thickened. Disc slightly swollen.

Case 8.—Wm. L—, æt. 37.

March 13th, 1883.—Fell off a cart on to the back of his head. Became unconscious and restless. There was bleeding from mouth and left ear.

28th.—The discharge from ear became serous the day after the accident, and the serous discharge continues. Became conscious two days ago, but dull.

Ophthalmoscopically.—Discs slightly swollen ; margins blurred ; vessels bend slightly at margin ; well-marked white lines along veins ; no hæmorrhages ; neuritis passing off (?).

April 21st.—Swelling and tenderness behind left ear. Incision. Trephining ; one drachm of pus let out.

May 2nd.—Much better in every way.

9th.—Now up every day ; optic discs of nearly normal appearance. Reads small print with each eye.

Case 9.—John P—, æt. 11, died twenty-four hours after injury. No ophthalmoscopic examination.

Post-mortem.—Fracture extending through left temporal bone to base of skull reaching nearly to spheno-occipital articulation.

Microscopically.—The optic discs seem normal. Sections of optic nerve close to disc present very slight, if any changes, but sections of the nerve at the optic foramen show very noticeable increase of nuclei confined to the outer part of the nerve. The optic tract appears normal, but there is evidence of inflammation in the small vessels of the pia mater on its surface.

Case 10.—Henry B—, æt. 16. Admitted June 1st, 1883, with compound comminuted fracture of vault of skull; right hemiplegia; double optic neuritis. Died June 24th of pyæmia.

Post-mortem.—Large fracture of vault of skull on left side and radiating fractures extending from this in various directions. Great excess of fluid at base of brain; no obvious inflammation of meninges; no abscess in brain.

Microscopically.—Optic discs swollen and inflamed; optic nerves near discs show inflammation which is more marked at the periphery than in the central part of the nerves. Sheath space distended. Sections of nerves near optic foramen show the signs of inflammation distributed fairly evenly throughout the nerves.

Case 11.—John H. B—, æt. 10. On admission, June 3rd, 1882, moaning with pain in head. Dulness at apex of left lung.

June 6.—Commencing optic neuritis in right eye; left disc normal.

7th.—Right eye, well-marked optic neuritis, advanced since yesterday. Left disc probably healthy. One vein tortuous, but this was thought to be physiological.

9th.—Right eye as on 7th. Left eye, now undoubted slight neuritis.

12th.—Died.

Post mortem.—Tubercular meningitis. Much fluid in

ventricles. Miliary tubercles in both Sylvian fissures, more noticeable in right.

Microscopically.—Great swelling of right optic disc. Vessels in disc full of blood-clot. Optic nerves: transverse sections close to globe show signs of inflammation in the sheath-space and in the trabeculæ and nerve bundles. In the sections from the proximal end of the nerve, near the optic foramen, these signs are much more marked. There is inflammation of the chiasma. The inflammatory changes appear not to differ materially in degree in the two nerves. The left optic disc was unfortunately lost.

Case 12.—Rosina C—, æt. 9. Admitted April 9th, 1883. Drowsy, irritable; hydrocephalic cry; tache cérébrale; paralysis of both external recti, and of right superior rectus and levator palpebræ; both pupils dilated, right most. No ophthalmoscopic examination.

April 11th.—Died.

Post-mortem.—Tubercular meningitis at base. Excess of cerebro-spinal fluid. Pia mater and arachnoid too adherent, peel off badly at base of brain.

Microscopically.—Optic disc slightly, but quite appreciably, swollen. Vessels in disc full. Optic nerve: very distinct inflammation of the sheath; sheath-space contains new inflammatory material. Considerable increase of staining nuclei in peripheral part of nerve, chiefly in the trabeculæ; in central portion of nerve increase much less noticeable.

Case 13.—Wm. H. B—, æt. 13. Admitted May 12th, 1883, with intense headache, giddiness, and defective sight.

Ophthalmoscopically.—Double optic neuritis, more intense in the left eye in which the swelling extends some distance beyond the disc. White patches are arranged around the base of the swollen disc, not at the yellow spot as in renal cases. The changes are of many weeks' duration.

No albuminuria. Died May 18th, 1883.

Post-mortem.—Tubercular meningitis with lumps of tubercle in cerebellum.

Microscopically.—Well-marked inflammation of optic nerves most pronounced near surface of nerve. Increase of tissue in the sheath-space.

Case 14.—Wm. B—, æt. 3. Admitted March 24th, 1883, for cachexia, distended abdomen, and history of fits.

May 20th.—Drowsy with headache.

21st.—Unconscious.

Ophthalmoscopically.—Doubtful haze of margins of discs.

28th.—Died.

Post-mortem.—Gross tubercular meningitis with caseating tubercular masses in cerebellum. Excess of fluid in ventricles. Old tubercular peritonitis.

Microscopically.—Nuclei of optic nerves considerably increased in number, this increase most noticeable peripherally. Increase of tissue between inner and outer sheaths, and very abundant nuclei. Moderate swelling of optic discs. Angle between nerve and sheath close to eyeball is distended. Optic tracts show very marked excess of nuclei at the periphery; their number towards the central portion of the tract appears to be but slightly above the normal.

Case 15.—Frederick B—, æt. 7. Admitted June 9th, 1881, for hip disease. It was noticed then that he had a peculiar vacant stare.

On September 17th he had tremors in hands, nystagmus, was awkward in all his movements, answered questions slowly and deliberately.

Ophthalmoscopic examination at this date by Mr. Nettleship: "Optic discs in third stage of papillitis, swollen, woolly. Large constellation of small, bright dots at yellow spot. Changes all more intense in right eye. Pupils wide."

November 24th.—Almost blind. Quite conscious.

December 22nd.—Died.

Post-mortem.—On the dura mater at the base of brain there are two masses, each about the size of a walnut, which on section are homogeneous, pale yellow, and slightly rough, without doubt tubercular.

Microscopically. — Optic nerves: great increase of nuclei in nerve bundles and trabeculæ, which latter are much thickened. Considerable disorganisation of nerve-tissue, bundles shrunken, leaving œdema spaces round them. In sheath-space large amount of newly formed tissue, containing many staining nuclei. Discs much swollen, enormous number of nuclei, considerable destruction of nerve tissue, vessels inflamed.

Case 16.—Rebecca P—, æt. 48. Admitted for spontaneous fracture of several of the long bones; she had also scirrhus of right breast and multiple secondary tumours. Several years previously had a tumour removed from the left breast; she was quite blind, and the optic discs were in a late stage of neuritis, becoming woolly in appearance. She was quite deaf.

Post-mortem.—New growths were found in ribs, both humeri, both femora, bones of skull, also in the liver and right breast; and nodules in the skin in various situations. The dura mater in left anterior fossa and right middle fossa was much thickened, and unusually adherent to the base of skull. The inner surface of calvarium showed numerous bony thickenings.

Microscopically. — Very marked increase of nuclei throughout nerve bundles, also in trabeculæ, which are thickened. There are many more small vessels throughout the optic nerves than in normal specimens. Nerves shrunken.

Case 17.—Daniel L—, æt. 16. Admitted November 29th, 1882. Pains in head, constant vomiting, occasional diplopia.

December 1st.—Double optic neuritis, some defect of vision.

January 1st, 1883.—Died.

Post-mortem.—Basilar meningitis. Excess of sub-arachnoid fluid. Wasting of grey matter of cerebrum in left hemisphere. In left lobe of cerebellum a tumour, size of a hazel nut, partially on surface, partially embedded. A larger tumour in central lobe of cerebellum.

Microscopically.—(Plates X and XI).—Sections through papilla show great swelling of disc. Sections of the optic nerve close behind globe show new inflammatory tissue in the sheath-space. In the nerve itself there is great increase of nuclei, both in the nerve-bundles and trabeculæ, the latter are not much thickened. The small vessels in the sheaths are inflamed. The evidences of inflammation in the nerve near the globe are all more marked towards the periphery, whereas in the sections of the nerve at the optic foramen they are evenly distributed throughout the nerve. There is distinct increase of staining nuclei in the optic tracts.

Case 18.—Caroline S—, æt. 23. Admitted April 10th, 1883. Headache and vomiting, staggering gait.

Ophthalmoscopically.—Great swelling of both discs, with radiating hæmorrhages on all sides of discs. Eye-sight defective.

19th.—Now quite blind.

20th.—Died, comatose.

Post-mortem.—Effusion of some dirty coloured fluid at base of brain. Ventricles distended with fluid. In right lobe of cerebellum a large simple cyst (see St. Thomas's Hospital Museum). Membranes at base opaque and abnormally adherent to brain. Some effusion in optic sheaths close to eyeballs.

Microscopically.—(Plate XII).—Very marked swelling of optic disc, with several hæmorrhages in and near it. Central retinal vessels not full. Lamina cribrosa convex anteriorly. Transverse sections of nerve show considerable, newly-formed, inflammatory tissue in the sheath-space. The small vessels in the nerve sheaths are

inflamed. In the outer part of the nerve there is in parts considerable increase in number of the staining nuclei; this is most noticeable in the trabeculæ, but is also seen in the nerve bundles at the periphery.

Case 19.—Wm. F—, æt. 10. Admitted December 12th, 1882, with intermittent headache and vomiting. No paralysis. Double optic neuritis; sight good.

January 24th, 1883.—Quite blind. Papillitis passing into atrophy.

March 8th.—Died.

Post-mortem.—Two tubercular nodules in cerebellum at posterior edge. Distension of ventricles by fluid. No basal meningitis to naked eye.

Microscopically.—Distinct evidence of inflammation of the meninges at base of brain.

Optic nerves.—In transverse sections there is seen to be increase of connective tissue in sheath-space, with great increase of nuclei.

There is enormous increase of nuclei throughout the nerve bundles, and also in the trabeculæ, which are thickened. All the small vessels cut transversely show inflammatory changes. Optic disc much swollen; great increase of nuclei. Vessels inflamed, and full of blood corpuscles. The changes in the optic nerves are evenly distributed, not noticeably greater at the periphery.

Case 20.—John T—, æt. 22.* On admission, October 25th, 1878, severe headache. Well-marked double optic neuritis. Vision good, and remained good ($\frac{20}{50}$, and 1 J badly) two or three days before death.

November 5th.—Died comatose.

Post-mortem.—In right lobe of cerebellum a simple cyst. Lateral ventricles distended with serum. No note as to meningitis.

Microscopically.—Optic nerves: marked swelling of discs. Great increase of nuclei, especially in trabeculæ. "This

* Previously recorded by Mr. Nettleship and Dr. Sharkey, 'Trans. Path. Soc.,' vol. xxxi, p. 252; vol. xxxiii, p. 8.

increase of nuclei is also noticeable in greater or less degree throughout the whole orbital portion of the nerve, *especially in the circumferential part*" [(Nettleship). Italics ours]. By the kindness of Mr. Nettleship we have had an opportunity of examining the sections of the optic nerves of this case, and we fully agree with the description given by him, which points out that the intensity of the inflammation is greater in the peripheral part of the nerve.

Case 21.—Joseph McK—, æt. 41. Admitted April 19th, 1882, with aphasia, right hemiplegia, headache, and history of vomiting. Sight thought to be good; no ocular paralysis; pupils equal. Hearing defective, right ear worst.

Ophthalmoscopic examination (April 24th).—Right eye: optic disc hazy, outline ill defined, white lines along several vessels, veins slightly enlarged, disc rather woolly, probably in a late stage of papillitis. Left eye: opaque nerve fibres, otherwise healthy.

17th.—Right eye in same condition, no hæmorrhage. Left eye: optic disc not quite clear, and by direct examination at least three of the vessels bend abruptly at margin of disc; margin striated; slight papillitis.

29th.—Died comatose.

Post-mortem.—Dura mater appeared to be normal. Little, if any, excess of fluid either subarachnoid or in ventricles. No basal meningitis to naked eye. In the anterior part of each hemisphere was a new growth in the white substance, but on the left side involving the grey matter.

Microscopically.—Right optic nerve: sheath-space somewhat distended; trabeculæ markedly thickened and containing many nuclei; increase of nuclei throughout nerve bundles. Disc greatly swollen, marked increase of nuclei, vessels full. Left optic nerve: slight signs of inflammation chiefly in sheath-space and peripheral part of nerve, where there are distinct increase of nuclei and signs of inflammation about the small vessels. Disc swollen, but to a much less extent than that of right eye.

Here, as in the nerve behind the eye, there is some increase in the number of nuclei.

Case 22.—Edward B—, æt. 7. Admitted August 2nd, 1882. On admission drowsy, weakness and tremors of arms and legs, ptosis of both eyelids, weakness of right internal rectus. No optic neuritis.

October 20th—More drowsy, headache.

November 30th.—Died comatose.

His eyes were frequently examined, and there were no changes seen till November 26th (four days before death), when slight optic neuritis was found.

Post-mortem.—Tubercular tumour in corpora quadrigemina pressing on crura. Tubercular meningitis at base with effusion of some fluid. Sheath of optic nerves immediately behind eye contained a drop or two of serum.

Microscopically.—Optic disc considerably swollen, inflammation in sheath-space. Optic nerve: marked swelling of trabeculæ, and very noticeable increase of nuclei in them, as also throughout nerve-bundles. New inflammatory tissue, with many nuclei between sheaths.*

The foregoing cases may be classified into five groups:

I. *Cases of head injury,* 10 (Nos. 1—10).—Of these nine were fatal. In only one of the nine was there obvious basal meningitis post mortem (No. 1), and in this case there was well-marked inflammation of the optic nerves. In four other cases (Nos. 3, 7, 9, and 10) there was microscopically inflammation of the optic nerves. In all these cases fracture of the base of the skull had resulted from the injury. In the remaining four cases of this group (Nos. 2, 4, 5, and 6) there was no inflammation of the optic nerve, although injury of various parts of the brain, so severe as to prove fatal, had been sustained.

The case in which recovery is taking place (No. 8) was one of undoubted fracture of the base of the skull, and there was ophthalmoscopically double optic neuritis.

* This case has been published in full by Dr. Bristowe, under whose care the patient was, in a paper in 'Brain,' July, 1883.

The view that the optic neuritis in such cases is secondary to basal meningitis is supported by two cases referred to by Mr. Hutchinson* in his lectures on head injuries; one where inflammation of the subarachnoid spaces at the base of the brain occurred, and in which there was optic neuritis, the other a case of arachnitis, in which, as in our case of arachnitis (No. 6), optic neuritis did not occur. Mr. Waren Tay† has recorded two cases of optic neuritis in head injuries, and the view to which he inclined was, "that some inflammatory process had occurred at the base of the brain." In one of his cases there was undoubtedly fracture of the base of the skull.

II. *Cases of tubercular meningitis*, 4 (No. 11, 12, 13, 14).—In all these cases there was inflammation of the optic nerves.

III. *Cases of growth in dura mater at base of skull*, 2 (Nos. 15 and 16).—In both there was double optic neuritis, which was passing into atrophy at the time of death. It is to be expected that a tumour of dura mater at the base of the skull would very soon give rise to inflammation of the meninges around it, and thus cause optic neuritis, which would have time to pass into atrophy before the disease proved fatal, as in the above two cases.

IV. *Cases of tumour of cerebellum*, 4 (Nos. 17, 18, 19, 20), in all of which optic neuritis was present. In two (Nos. 18 and 20) there was a simple cyst of cerebellum, in one (No. 19) a tubercular, and in one (No. 17) a gliomatous growth. In all there was post mortem excess of cerebro-spinal fluid distending the ventricles. In two (Nos 17 and 18) there was basal meningitis visible to the naked eye, in one (No. 19) inflammation of the meninges at the base of the brain was found by microscopic examination; in the other (No. 20) no note was made as to meningitis.

It is to be observed that in all these four cases of

* 'Med. Times and Gazette,' 1875.

† 'Trans. Ophthal. Soc.,' vol. ii, p. 66.

cerebellar tumour optic neuritis was present when they came under observation. This can be readily understood on the meningeal theory of the origin of optic neuritis; the cerebellum being in close proximity to the meninges of the base of the brain, a tumour in the cerebellum would at an early period of its existence set up basal meningitis, and extension of the inflammation to the optic nerve-sheaths would soon occur.

V. *Cases of tumour of cerebrum,* 2 (Nos. 21 and 22).—In both there was optic neuritis. In one of these cases (No. 22) there was a tumour in the corpora quadrigemina; the patient was in the hospital for many weeks, and was repeatedly examined ophthalmoscopically, but no optic neuritis was found. An attack of tubercular meningitis, fatal in a few days, at last supervened, and optic neuritis then occurred.

In the other case (No. 21) there was a tumour in each frontal lobe; no basal meningitis to naked-eye examination. No microscopic examination of the meninges was made.

It will, we think, be allowed that in Case 22, in which the optic neuritis did not appear till symptoms of the fatal meningitis came on three or four days before death, the optic neuritis was due to the meningitis and not to the tumour; and this, although the tumour was situated in the corpora quadrigemina, a portion of the brain closely connected with the sense of sight. This case is also an example of what frequently occurs, viz. symptoms of intracranial tumour existing for a long time without optic neuritis and then, often with an aggravation of symptoms, optic neuritis appears. The occurrence of a secondary basal meningitis affords a ready explanation of this.

Looking at all these cases together it is to be noticed—(1) that in all those in which there was obvious meningitis at the base there was optic neuritis; (2) that in one case (No. 19) in which it was noted at the post-mortem examination that there was no meningitis—the meninges, when examined microscopically, were found to be inflamed; (3) that there is no case with optic neuritis in

which the meninges at the base were shown by microscopic examination to be free from inflammation.

The microscopic examination of the optic nerves of these cases affords further evidence in favour of the meningeal origin of the neuritis.

In the illustrations (Pls. X, XI, XII), and in all specimens of recent cases of optic neuritis examined, whether there was obvious meningitis or not, it can be seen that at one or other part of the nerve, according to the duration of the inflammation, the inflammatory process is distinctly more marked at the peripheral portion of the nerves than at their central portion. Plate XII shows very well that the inflammation (evidenced chiefly by the increase of staining corpuscles) is spreading from the sheath inwards along the trabeculæ, and had at the time of death scarcely reached the nerve-tissue proper.

In all the cases there was found to be inflammation of the loose tissue between the outer and inner sheaths of the optic nerves, and in many of the specimens there was a considerable amount of new inflammatory material in this sheath-space.

In no specimens examined have we found inflammatory changes more marked at the central than at the peripheral part of the nerve.

These facts seem to us to point strongly to the theory that the inflammation extends from the meninges to the sheaths of the optic nerves, down which it spreads, invading the nerve-tissue by extension inwards along its fibrous framework.

It will, we suppose, be admitted that in cases of basal meningitis the inflammation of the optic nerve has extended to them from the inflamed meninges, and the same for cases (as Nos. 15 and 16) of tumour of the dura mater at the base of the skull. In cases of cerebral and cerebellar tumour, however, the following objections may be raised to this theory :—

(*a*). That cases occur in which there is well marked optic neuritis, but no basal meningitis is found at the

post-mortem examination. To this objection we would reply that the test of a naked-eye examination of the meninges is not conclusive; the inflammation of the optic nerve itself is usually unrecognisable, unless looked for with the microscope (see case 19).

(*b*). That although basal meningitis may be found at the post-mortem, the optic neuritis was of much earlier date. It does not, however, follow that because the case was brought to a fatal termination by a severe attack of meningitis, there was no earlier slight inflammation of the meninges. Moreover, the optic neuritis is not always antecedent to the fatal meningitis; for in Case 22 the optic neuritis only appeared when the fatal attack of meningitis supervened.

(*c*). That cases are reported of basal meningitis without optic neuritis. No such case has come under our notice.

Dr. Garlick,* in twenty-six fatal cases of tubercular meningitis, found only three in which optic neuritis could not be recognised by ophthalmoscopic examination, and in one of these three he notes that the meningitis was confined to the convexity of the brain.

(*d*). That there are certain cases of cerebral tumour in which the optic neuritis is single, and usually on the side opposite to the lesion. These cases are rare, and we have only found five recorded in this country. Dr. Hughlings-Jackson† mentions two cases. In one there was right-sided hemiplegia, with marked optic neuritis in right eye. Left eye normal. Paralysis of left external rectus. Post-mortem: there was found a glioma of left hemisphere. No note seems to have been made as to meningitis. The optic nerves were examined by Dr. Pagenstecher, who found well-marked inflammation of the right nerve and its sheath. The left optic nerve was apparently normal. Nothing was found to account for the paralysis of left sixth nerve.

* 'Med.-Chir. Trans.,' vol. lxii, p. 441.

† 'Ophth. Hosp. Rep.,' vol. vii, Nov., 1871.

Dr. Jackson's other case, so far as we have been able to ascertain, is not reported at length. Dr. Gowers* records one case, probably on the side opposite the lesion, which did not terminate fatally.

Dr. Field† records one case. In this the neuritis was on the side opposite the tumour. It was a very exceptional case, the neuritis disappearing completely while the disease progressed to a fatal termination.

Mr. Nettleship, in a paper read before the British Medical Association in August, 1882,‡ refers to a case in which there was single optic neuritis on the side (R.) opposite the lesion, with, however, atrophy of the optic nerve on the same side (L.) as the lesion. Post mortem there was found to be softening of the under surface of the left frontal lobe, which was adherent to the dura mater, a condition which is fair evidence of previous inflammation.

Closely resembling such cases are those in which the optic neuritis, though double, is of unequal severity in the two eyes, or takes place in one eye long before the other. One such case is recorded by Mr. Nettleship,§ in which, however, it is to be noted that the neuritis was most severe in the eye on the same side as the lesion. Dr. Buzzard‖ has recorded a case of tumour of the left hemisphere of the cerebellum, in which the optic neuritis, though double, was most advanced in the eye on the same side as the tumour. In this case there was basal meningitis and a small tumour in the right half of medulla.¶

In two of our cases (Nos. 11 and 21) the optic neuritis appeared in one eye before the other, and was of unequal

* 'Med. Ophthalmosc.,' 2nd edit., p. 292.

† 'Brain,' July, 1881, p. 247.

‡ 'Brit. Med. Journ.,' Dec. 2, 1882.

§ Loc. cit.

‖ 'Trans. Clin. Soc.,' 1874, p. 165.

¶ Dr. S. Mackenzie records, in 'Brain,' July, 1883, a case of tumour of brain in which the optic neuritis appeared first in the eye on the same side as the tumour.

severity, as seen by ophthalmoscopic examination shortly before death.

One of these (No. 21) was a case with a tumour in each frontal lobe, the tumour on the opposite side to the optic nerve first inflamed being the larger of the two. The other case (No. 11) was one of tubercular meningitis.

The theory of a descending cerebritis travelling down the optic nerves themselves, by extension from the brain substance, is supported by two cases, one recorded by Dr. Stephen Mackenzie,* "A Case of Double Optic Neuritis without Gross Cerebral Lesion;" the other by Dr. Hughlings-Jackson,† "A Case of Double Optic Neuritis without Cerebral Tumour." In the post-mortem report of the first of these cases it is noted that "the pia mater is milky looking and semi-opaque," and in a sketch of the microscopical appearances of the brain appended, an inflamed blood-vessel is very prominent; which vessel, we presume, came from the meninges. It is therefore probable that there was some inflammation of the meninges in this case. The meninges of the nerves are described as inflamed.

In the second of these cases the post-mortem report by Dr. Sutton is, in part, as follows:—"Scalp normal; dura mater healthy; arachnoid healthy. Vessels of pia mater enormously distended, and of a blackish colour. It is rare to see the vessels of pia mater so very much engorged."

Microscopically there were some changes in the cell arrangement of the second and third layers of the grey matter of the cortex. Little or no change in the capillaries.

The optic nerves were examined by Dr. Gowers, who, in his description of them, says:—"The connective tissue in the intervaginal space is very abundant, the trabeculæ being numerous and thick."

* 'Brain,' vol. ii, p. 257.

† 'Ophth. Hosp. Rep.,' vol. viii, p. 445, and Gowers' 'Med. Ophthalmosc., 2nd edit., p. 305.

Dr. Silk* has recorded a case of optic neuritis "secondary to some obscure cerebral lesion (? cerebritis)." Microscopical examination of sections of optic nerves and papillæ showed that multiplication of nuclei had occurred, but "was mainly confined to the nerve-sheaths."

There are, however, objections to this theory. It is specially claimed for it that it alone satisfactorily explains the occurrence of single optic neuritis on the side opposite the lesion in cases of cerebral tumour, but it seems reasonable to suppose that if inflammation were started in one optic tract by a tumour it would, on reaching the chiasma in its descent, extend down both optic nerves in consequence of the semi-decussation of the fibres. Again, if this theory be correct in cases of cerebral tumour, why is the optic neuritis not always single and an early symptom? Further, if this descending cerebritis be the true explanation, we should expect optic neuritis to occur especially in cases of tumour or other lesion in or near the centres of vision or their connections with the optic nerves. In one of our cases, however (No. 22), there was a tumour of the corpora quadrigemina; no optic neuritis occurred till within a few days of death, when a fatal attack of tubercular meningitis came on; while, on the other hand, in all the four cases of cerebellar tumour, the new growths being situated some distance from the origins of the optic nerves, optic neuritis was present.

In Dr. Ferrier's experiments on monkeys optic neuritis never occurred after excision of the angular gyrus or gyri, but primary atrophy of the optic nerves followed the destruction of these gyri when the animals were kept alive long enough.†

(*May 10th*, 1883.)

* 'Brit. Med. Journal,' May 26, 1883.

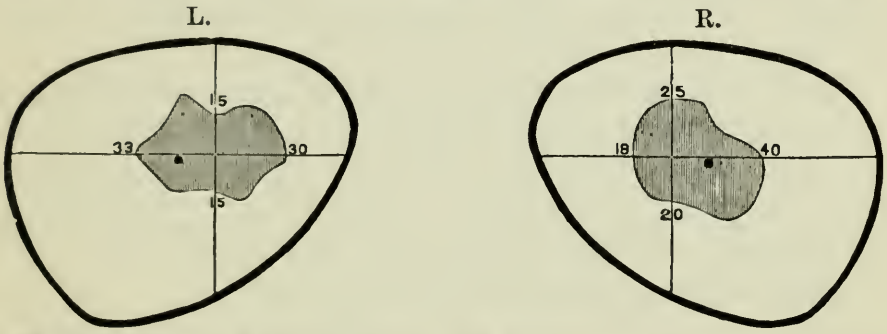
† 'Brain,' Jan., 1881.

3. *Case of central amblyopia in a smoker suffering from diabetes.*

By A. Stanford Morton.

Samuel H—, æt. 34, a railway-ticket collector, was admitted at the South London Ophthalmic Hospital on January 9th last (1882). His sight had been failing decidedly for six weeks, but he had not been able to read the newspaper well for eight weeks.

On admission he read 16 J. with both eyes open, but could not see 200 Sn. at twenty feet. His colour-vision for wools was perfect, but I found a well-marked area of large size in the central part of each field on which red and green were not recognised (*see* Fig.). In conse-



Fields of vision.—R. right, L. left. For description see figure on p. 162.

quence of this central defect he had a difficulty in seeing the signals ;—" if I want to see the arm of the signal I must look to one side of it." With the ophthalmoscope I found the discs hazy and rather congested, and in the left eye near the yellow spot one or two bright spots surrounded by very slight grey haze.

This man was a smoker, but only to the extent of an ounce and a half of shag tobacco weekly. He drank whisky, not beer. He said that he had formerly had diabetes, but " had none now."

At the next visit, February 2nd, his urine was tested and found to contain a good deal of sugar. He said he was passing a large quantity of urine.

On February 15th the urine still contained sugar; sp. gr. 1025. I now thought there was a very faint whitish haze around the yellow spot in both eyes.

March 1st (seven weeks after admission).—Urine contains plenty of sugar; sp. gr. 1032; is passing from four to five pints a day (twenty-four hours); is taking a modified diet. For several weeks past he has begun to smoke again a little, and he admits that even when he ceased smoking for a time after he first came under care he chewed instead. V. has not improved (reads 16 J. as on admission); there is still a well-marked central scotoma for red and green, *not for blue*.

April 3rd.—Sees 10 J. barely and $\frac{20}{200}$.

24th.—Has not been so well; is lame, and the ankles are swelled; is not passing more urine, and is not thirsty. V. seems worse again (16 J. and $\frac{20}{200}$). Discs now pale on outer side.

May 22nd (four months and a half after admission).—Has been feeling languid and ill; quantity of urine not greater, and no thirst. V. R. not $\frac{20}{200}$. Scotoma for red and green still present. L. 16 J. and $\frac{20}{200}$ by fixing eccentrically, scotoma well-marked and large.

July.—Patient has not been seen since.

(*December 14th*, 1882.)

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4. *Examination of optic nerve from a case of amblyopia in diabetes.*

By Walter Edmunds, M.D., and J. B. Lawford, M.D.

(With Plate XIII.)

George Y—, æt. 29 years, was admitted into St. Thomas's Hospital (under Dr. Ord's care) on December 16th, 1881, and died on the 21st of the same month.

History.—A year before admission he was passing a

DESCRIPTION OF PLATE XIII.

Illustrating a case of Disease of the Optic Nerves in Diabetes, by Drs. Edmunds and Lawford (p. 160). From drawings by Miss Alice Boole.

FIG. 1 is from a transverse section of the optic nerve between the globe and the point of entrance of the central vessels. The drawing includes rather more than half the width of the nerve, and extends beyond the limit of the disease. The outer sheath is absent. The diseased area, involving about a quarter of the section, and extending, where widest, from the central artery to the inner sheath, shows an increase of nuclei, chiefly in the nerve-fibre bundles, but also to some extent in the fibrous septa; considerable disorganisation of the trabeculæ, and thickening of the portions remaining; increase in thickness of the inner sheath of the nerve where it skirts the diseased patch. × 21.

FIG. 2.—Part of the diseased area. The nerve-fibres are greatly disorganised; in many of the bundles only a few, which can be recognised as fibres, remain. There is considerable increase of fibrous tissue surrounding the small vessels (*v*, *v*). In that portion of the nerve intervening between the patch of disease and the inner sheath the fibrous septa are somewhat thickened, though the nerve bundles appear normal. × 84.

s. Inner sheath.
v, *v*. Small vessels.
a and *b* point to corresponding parts in the two figures.

Fig. 1.

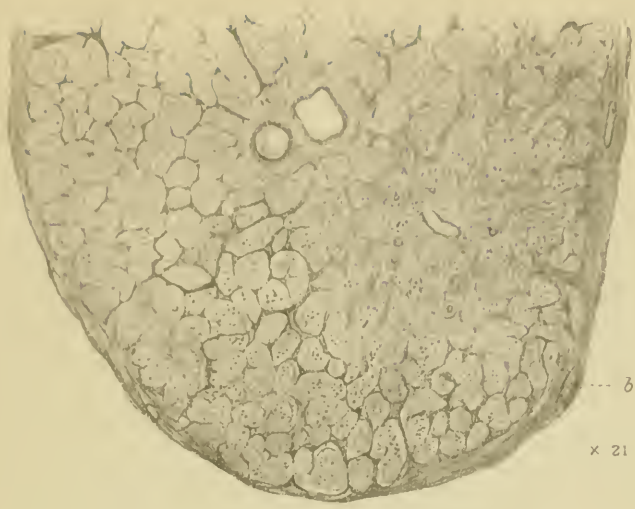
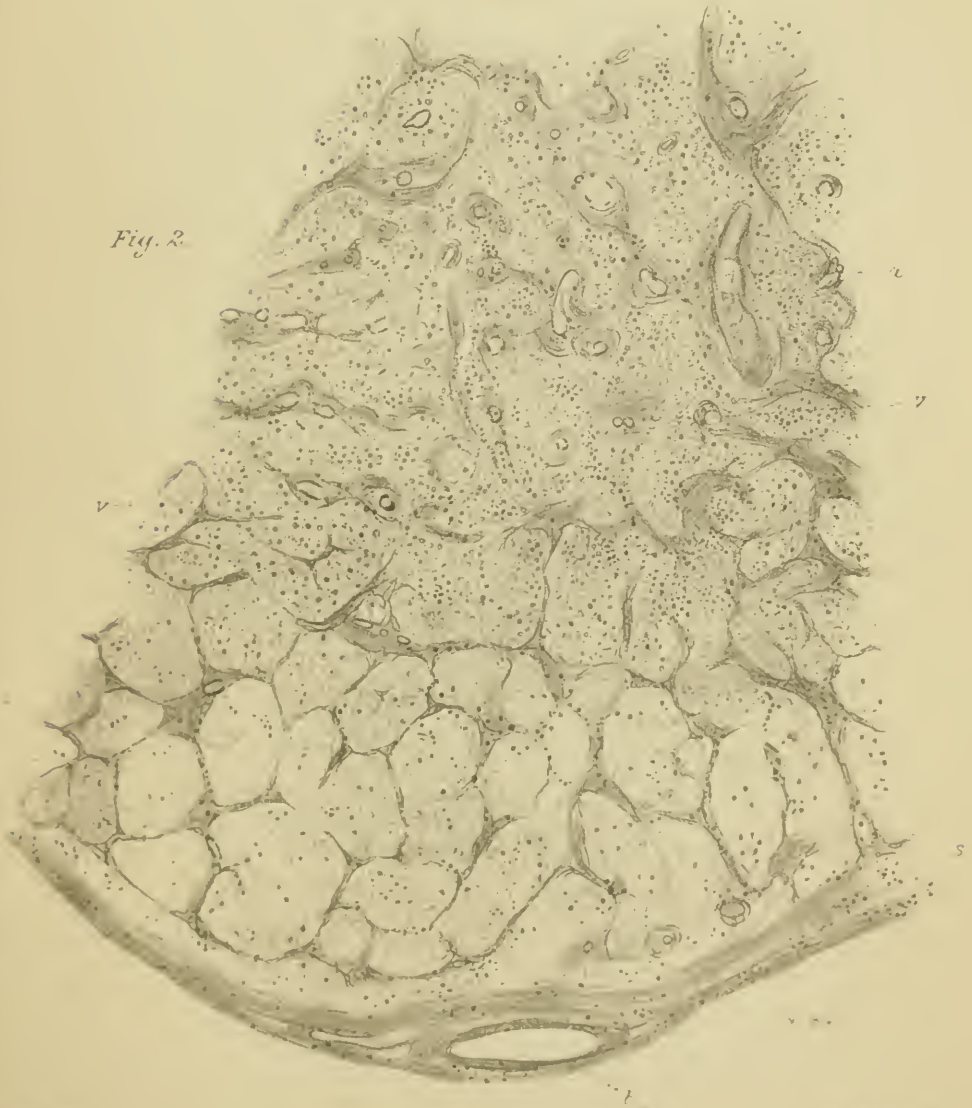


Fig. 2.



large quantity of urine. His sight began to fail four months before admission, and was now very bad. Used to smoke an ounce of shag a day.

State on admission.—Extremely emaciated and weak; great thirst. Passes eight pints of urine in twenty-four hours of sp. gr. 1036; urine contains a large quantity of sugar and a trace of albumen. Left pupil larger than right. Complains of pains in the limbs and great weariness.

The eyes were examined ophthalmoscopically and found normal. Owing to his very weak condition a prolonged examination could not be made. Total absence of both patellar reflexes. Thirty-six hours before death diabetic coma came on.

Post-mortem examination.—Liver large, 4 lbs. ½ oz. Spinal cord appeared to the naked eye normal. Back of right eye and right optic nerve kept for further examination.

Examination of the optic nerve shows no abnormal appearance to the naked eye.—*Microscopically,* marked changes were found in a group of the nerve-bundles throughout the whole length of the orbital part of the nerve; the portion of the nerve posterior to the optic foramen was not examined.

In the diseased area (Plate XIII) there is seen to be a great thickening of the trabeculæ and of the walls of the nutrient blood-vessels there; the bundles of nerve-fibres seem to have the fibres in them destroyed, and they seem to be replaced by an irregularly granular structure in which there is a large number of staining nuclei. This diseased area does not at any part reach quite to the surface of the nerve, nor is it anywhere centrally placed; but it cannot be determined, from the sections, on which side of the axis of the nerve the altered area lies. The left optic nerve was, unfortunately, not kept for examination.

Remarks.—The absence of knee-jerk and the occurrence of pains in the limbs would seem to indicate that the optic nerves were not the only part of the nervous system affected.

The changes found in the nerve appear to us to be too great to be secondary.

A comparison of these drawings with those exhibited by Mr. Nettleship and Dr. Edmunds, at a meeting of this Society in 1881 (see 'Transactions,' vol. i, pl. iv) seems to show that the disease in our case had reached a later stage, and that the inflammatory had been succeeded by degenerative processes, causing loss of considerable portions of the trabeculæ and of many of the nerve-fibres.

(*December* 14*th*, 1882.)

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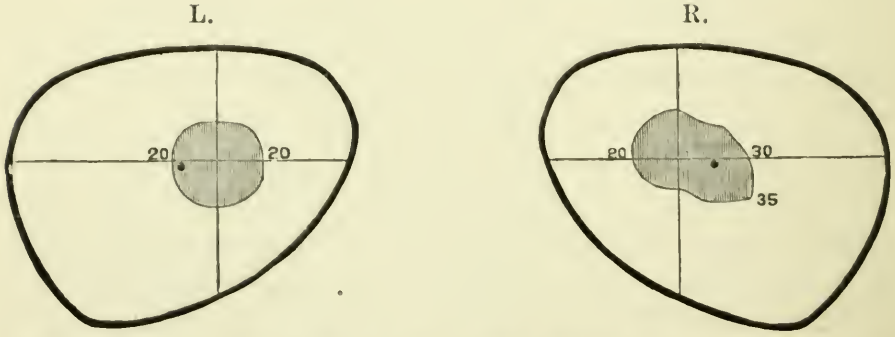
5. *A case of central amblyopia in a smoker with diabetes.*

By W. Lang.

Henry S—, æt. 28, attended at Moorfields on March 2nd, 1882. He gave the following history:—Previous to February, 1881, his health had been very good, but about that time he was seized with an unquenchable thirst, passed large quantities of urine, and gradually lost strength and flesh. In June he attended at St. Peter's Hospital and



Fields of Vision: R. right, L. left. The shaded area in each shows the scotoma. The numbers indicate the size of the scotoma in degrees, measured from the fixation point at the crossing point of the vertical and horizontal meridians. The black dot is the blind spot.

was under the care of Mr. Heycock, who tells me there

was a large quantity of sugar in the patient's urine. Four months ago, he first noticed that people's faces were dim when they were coming towards him, and about this time he had to give up reading. He had been in the habit of smoking half an ounce of shag tobacco daily for many years. His vision with each eye was $\frac{8}{200}$, and 16 J. There were no marked changes in either fundus. The fields of vision were normal in extent, but presented a complete central scotoma for red and green. (*See* Fig.)

Some few weeks after this one attendance at Moorfields the patient died.

I am indebted to Mr. Adams, under whose care he was, for being allowed to bring this case before your notice.

(*December 14th*, 1882.)

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6. *A case of stationary tobacco amblyopia in a man subsequently affected by diabetes.*

By J. B. Lawford, M.D.

C. F—, male, æt. 46, a waiter, was admitted into St. Thomas's Hospital in September, 1882, under Dr. Stone, who has kindly allowed me to make use of the case.

There is nothing of importance in the family history. The patient has always enjoyed good health. No gout or rheumatism.

He has been a heavy drinker, chiefly of spirits, though during the last few years his habits have been more moderate. He has, since he was a lad, smoked largely, "cigars and shag tobacco."

Seven years ago, while at his work as a waiter, he found that he "mistook half sovereigns for sixpences," and that "he could not see the colour of a soldier's coat in the distance," and he went to the South London Oph-

thalmic Hospital on account of these symptoms.* There he was told, to use his own expression, "that he was colour-blind from tobacco," and was urged to give up smoking. This advice he did not follow, but has continued to smoke to the same extent since then, without any further deterioration of sight.

Six months before admission to St. Thomas's he had a sharp attack of diarrhœa, followed by rapidly increasing weakness, thirst, and frequency of micturition.

When admitted he was greatly emaciated, suffering much from thirst. Skin very dry. He was passing, on an average, 120 to 150 oz. of urine a day, sp. gr. 1030 to 1035, and containing a considerable amount of sugar, but no albumen. Complains slightly of headache; no vomiting. No sciatica or other neuralgia. Knee-jerk normal. No disease of organs revealed by physical examination. He says that his sight has not become worse in the last six months.

V. R., $\frac{20}{100}$ and 10 J. with correcting glasses. Refraction H.
L., $\frac{20}{100}$ and 10 J. with correcting glasses. Refraction H.

Ophthalmoscopic examination.—Both discs look pale, more so on the y. s. side; choroids dark. Examination with Holmgren's wools revealed no defect of colour vision, but on trying with a 5 mm. red spot there is found to be a large absolute central scotoma for red.

The chief points of interest in this case are:

1. That the central amblyopia has existed for about seven years, and has remained the same in degree during that period, though the use of tobacco has been continued.

2. That the defect of vision has not increased since the date at which the diabetes is believed to have begun; and consequently the amblyopia should in all probability be attributed entirely to the tobacco.

(*December 14th*, 1882.)

* The patient had forgotten under whose care he then was, and had lost the card by which his notes might have been identified.

7. *On central amblyopia in diabetes, especially as to its dependence on, or independence of, tobacco smoking.*

By E. Nettleship and Walter Edmunds, M.D.

Since the occurrence of the cases of central amblyopia in patients suffering from diabetes, which were brought before the Society by us in July, 1881, four more cases have come under our notice. Several cases of amblyopia in diabetics without ophthalmoscopic changes are also to be found in various publications, and we propose to add short abstracts of most of these to the present communication before commenting on the entire group.

Case 1.—George B—, engraver, æt. 40, married, came under Mr. Nettleship's care in November, 1881. He was a small, pale, thin man, and looked older than he stated himself to be. He was at the same time attending the hospital under Mr. Clutton for stricture of the urethra.

The sight had been failing in both eyes alike for about a month,* without variations. There had been no other symptoms.

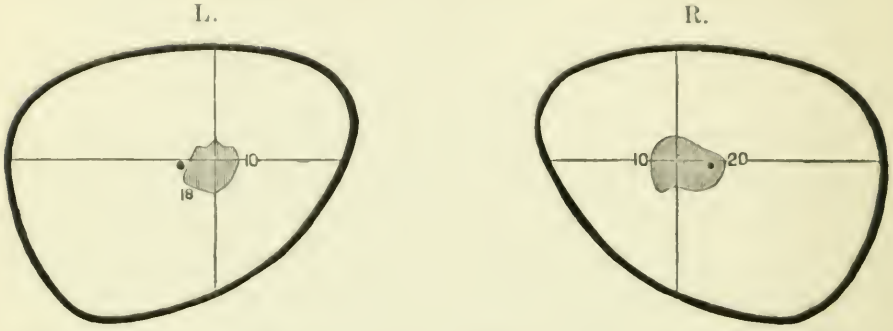
V. R., $\frac{20}{100}$, and letters 18 J., with + 4 D. letters of 14 J.
L., $\frac{20}{100}$, and letters of 18 J., with + 3 D. letters of 14 J.

A well-marked central scotoma for red was easily found. He was in the habit of smoking half an ounce of tobacco a day, but said he drank very little beer and no spirits. He was told to leave off smoking.

He did not attend again for four months (March 28th, 1882), when his sight had, if anything, become rather worse (*viz.*: 16 J. with + 2 D., but not $\frac{20}{200}$). He had gone on smoking all the time though not quite so much as before. The pupils were natural. Ophthalmoscopic appearances normal, except a slight haze across one vessel

* A later note states that the failure had been more gradual.

at the disc in the left eye; media clear. Very well marked central scotoma in each eye (*see* Fig.), but no



Fields of Vision: R. right, L. left. For description *see* previous figure (p. 162).

contraction of the field of vision. No colour-blindness could be detected by the wools, but when tried with an imitation signal-lamp he was quite unable to name the green, and only occasionally and doubtfully the red.

He had been getting thinner and weaker, chiefly no doubt from being half starved, as he could not see to work. He had noticed an increase in the quantity of his urine for several months, and was troubled by constipation and thirst. The urine was clear, acid, free from albumen, but gave marked reactions to the sugar-tests; sp. gr. 1045. The average quantity daily for the next few days after admission, before special diet or treatment, was from 150 to 180 ounces (7½ to 9 pints).

On April 15th, a few days after admission to the ward, special diet was begun, and on 24th codeia was given, at first in half-grain doses, gradually increased to one and a half grains three times a day. He remained in the ward till the end of May (six or seven weeks), and the quantity of urine, which was measured daily, steadily decreased, with occasional fluctuations, from 160 to 70 ounces a day, and he gained eight pounds in weight. Vision remained just the same as on first admission, $\frac{20}{200}$, and with + 3 D. some words of 14 J. He would, however, persist in taking tobacco secretly, both by chewing and smoking,

and was therefore sent out of the hospital and has not been seen since. It is important to observe that the amblyopia did not increase during the six months of observation, though smoking was continued.

Case 2.—The Rev. F. L. I—, æt. 38, was admitted into St. Thomas's Home, under Dr. Edmunds, in January, 1882. He had been suffering from diabetes for three or four years previously, but it was not severe. He was, on admission, passing five pints of urine a day, of sp. gr. 1028, giving an abundant sugar reaction.

For many years the patient had been in the habit of smoking half ounce of "golden Virginia" a day, a tobacco which, according to him, is nearly as strong as shag. He said it made him tremulous, but it did not make him feel "limp" or less fit for his work. He never confined himself for any length of time, either in food or drink, to the prescribed diet. He was accustomed to take champagne somewhat freely. His brothers smoked and had not suffered in their sight. His sight was perfect till early in the autumn of 1881 (about five months before he came under observation).

In August (1881) he could see to shoot grouse, but on September 1st he could not shoot partridges; he could, he said, see them rise but when he prepared to aim he lost them. By the end of September he had entirely lost the power of reading and could not see people's faces. He thought his sight remained stationary after about a month's failure.

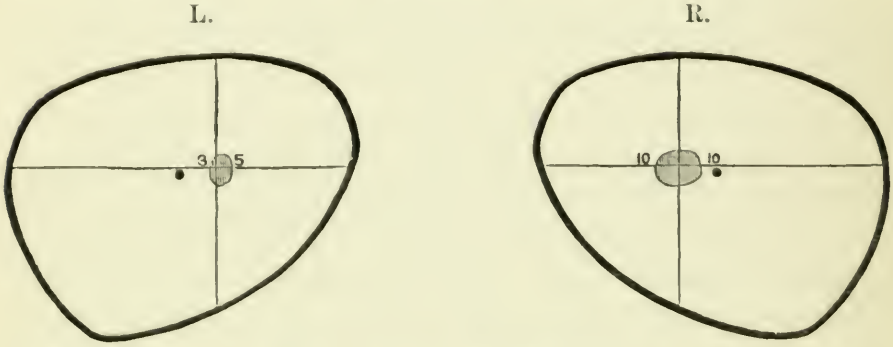
He saw a well-known ophthalmic surgeon in the north of England about this time; diabetic amblyopia was diagnosed, and he was told not to smoke so much, but appears not to have acted on the advice.

We examined his eyes, with Mr. Lawford's assistance, early in February and found as follows:

V. R., 19 J., sometimes words of 14 J. at 4″, and $\frac{5}{200}$.
L., 20 J.; at another examination 14 J. barely at 4″, and less than $\frac{5}{200}$.

The left eye had been operated on for convergent squint

twelve years ago and was thus probably somewhat defective before. Pupils of ordinary size and act well to light. There was some colour-blindness, he confused green and grey wools, but did not confuse pinks and reds with any others. The visual fields were of full size for white, and the boundary of the field for red was also of about the normal size, but at the centre was a scotoma on which the red could not be recognised. A green spot was not recognised in any part of the field, being always called "white," *i.e.* the central scotoma was apparently as large as the natural field for green. In the figure the shaded



Fields of Vision: R. right, L. left. For description see figure, p. 162.

central area shows the part on which red was not recognised. In all probability perception of red was lowered, though not quite abolished, over a considerably larger area, as is usual when acuteness of vision in central amblyopia is as low as it was in this case. The centre of his field, indeed, was so defective that he had quite a difficulty in going about in a strange room, and hence had a much more "amaurotic aspect" than is usual with these cases. As the fields were only taken once and the patient was somewhat irritable, too much stress must not be laid on the difference of size of the central defect in the right and left as shown in the figure. The patient died in the country two or three months after the above notes were made.

Case 3.—James H—, æt. 48, married, the father of

five healthy children, a harness-maker, came under Mr. Nettleship's care on account of his sight in February, 1882. He said that his vision had been perfect till five weeks before, about which time he found he could not see to read the newspaper. So far as he knew both eyes failed at once, but his account was meagre. At the time his sight failed he was in a workhouse infirmary for "diabetes," and had been under treatment for the disease for twenty-one months altogether. Till then he had always been healthy. He had smoked since he was sixteen, usually three quarters of an ounce of shag a day, and never found it disagree with him. He had also been a heavy rum drinker.

With the left eye he saw $\frac{20}{100}$ and 16 J., improved by + 2 D. to seeing words of 8 J. The media were clear, and the fundus normal, except that the disc had a brownish tinge.

With the right eye he could only count fingers. This difference was sufficiently accounted for by the presence of satiny-looking striæ in the cortex of the right lens; the fundus showed no changes. The fields of vision were only roughly tested, but enough to prove the presence of a nearly central scotoma extending from the fixation point outwards. The colours of a red and green spot were, as usual in these cases, best seen just to the nasal side of the fixation point.

He was admitted into the hospital, but in a few days had to be sent out for bad conduct. Whilst in he passed from eleven to twelve pints of urine daily. It was pale, of sp. gr. 1035, free from albumen, but gave a marked reaction for sugar.

Case 4.—Mr. J. A. G—, now æt. 58, a rather excitable but healthy-looking man, has been in business for upwards of thirty years in Calcutta. He once, long ago, had an attack of "fever and liver," but since then has scarcely ever been ill. He has, he considers, always been very moderate in stimulants, usually taking beer, and "never

drinking anything before twelve." He has never been a great smoker; when in India his allowance was two or three large cheeroots a day; since coming home, on account of his sight, he has smoked only a smaller sort. There is no history or evidence of syphilis.

In March, 1881, his sight, till then perfect, quickly failed in both eyes alike. In less than a month it got to its worst, and, he thinks, remained at a standstill from that time onwards. He was seen by several specialists, but seems not to have been prohibited from smoking by any of them.

When the failure began he had pain across the forehead and round the back of the head; this pain, he says, was severe. There was no vomiting or other brain symptom. On inquiry he admitted that for some time before his sight was affected he had been sleeping badly on account of business anxiety.

He came to Mr. Nettleship on November 15th, 1882. With each eye he saw $\frac{20}{200}$ and 19 J., improved to 16 J. with his +3·5 D. glasses; the pupils were rather large but acted very briskly both to light and accommodation. The optic discs were decidedly pale all over, more so than is usual in tobacco cases, even of such long standing and occurring at such an advanced time of life as in this patient.

Sight, he said, was worst in bright light, best in the evening: he had, indeed, a sort of photophobia, and liked to wear blue glasses in the daytime, as without them he got his old frontal pain back again. He was aware that he could not see red "except a little in certain positions," and he had often mistaken gold coins for silver. The fields of vision were of full extent, but at the centre and extending horizontally outwards was an area of red-blindness exactly as in typical cases of tobacco amblyopia.

He did not think his water was particularly free. Mr. Nettleship sent him to Dr. Gowers for his opinion, mentioning the suspicion that it might be a diabetic case. Dr. Gowers found no indication of disease of the nervous system (other than the optic nerves), but reported that the

urine contained sugar to the extent of fifteen grains to the ounce. No albumen.

December 12th.—Two other samples of urine examined to-day gave good reaction for sugar with Fehling's solution. Their sp. gr. = 1020.

Jan. 25, 1883.—V. with each eye alike, $\frac{20}{200}$, improved to $\frac{20}{100}$ with + ·5 D., and 16 J. with his own glasses.

The quick onset and stationary character of the amblyopia, with the unusual pallor of discs in a rather small smoker, contributed to suggest some unusual factor in this case, and from previous experience diabetes seemed not improbable.

A good deal has been written since the invention of the ophthalmoscope on amblyopia in diabetes, without cataract and without retinal hæmorrhages or other visible lesions.

Some of the cases are imperfectly recorded, others are probably examples of the rapidly progressing failure of accommodation, or premature presbyopia to which Trousseau* and other authors have called prominent attention.

We have selected only such of these published cases and references as may, with fair probability, be grouped together with the cases we have heard this evening; they are as follows:

1. A hospital sister seen by Desmarres and Mialhe, age not stated. Formerly strong and stout, now emaciated and feeble, passing a considerable quantity of sugar. Sight bad, became so bad that she could no longer read and made frequent mistakes.†

2. M—, æt. 25, passing a very large quantity of sugar and became very thin and debilitated. Vision failed so that he could neither read nor write even with glasses.

After several months' treatment for diabetes his health had greatly improved, and his sight was so much

* 'Trousseau's Lectures,' vol. iii (N. S. Soc's. translation), p. 505. Rapidly increasing failure of accommodation is "one of the most common as well as one of the most remarkable of the symptoms of saccharine diabetes." He does not seem aware of any other form of amblyopia.

† Desmarres, 'Traité,' 1858, t. iii, 521.

better that he could easily read with the aid of No. 8 glasses.*

Dr. Warburton Begbie,† writing in 1861, stated that he was familiar with a form of "diabetic amaurosis" coming on earlier in the disease than cataract, advancing very gradually, and sometimes, after remaining stationary for a time, partially disappearing; it was accompanied by pain or uneasiness in the eyes or head. Begbie gives no cases.

Later on in the same year Lecorché‡ wrote a careful article on "Diabetic Amblyopia," in which he drew attention to a slight and a severe form. Though he does not distinguish clearly enough between failure due to rapid presbyopia and true optic nerve amblyopia, it is evident that he is well aware of these two forms of visual failure and also of the occurrence of cases due to retinal hæmorrhage.

After a careful criticism of previous writers, he states his belief that in the severer forms of glycosuric amblyopia without ophthalmoscopic changes, the cause will probably be found to be a partial or general atrophy of the optic nerve or retina occurring independently of changes in any part of the brain, the fourth ventricle for example. He says that diabetic amblyopia is very rare under twenty-five, being most common between twenty-five and forty, that it attacks men much more often than women (diabetes itself being, as he immediately remarks, commonest in males), is symmetrical though unequal in the two eyes, and seldom goes on to absolute blindness.

The suggestion that some cases of diabetic amblyopia were due to a cerebral cause had been previously made by Von Graefe,§ who had also recorded several examples of various forms of eye disease in diabetes.

* Desmarres, loc. cit.

† Begbie, 'Edin. Med. Journ.,' 1861, p. 1105 (June).

‡ Lecorché, 'Gazette Hebd.,' 1861, pp. 717 and 749 (November).

§ Von Graefe, 'Arch. f. Ophth.,' iv, ii, 230, 1858. In this paper seven cases are given, four being cataract, one choroiditis, and only two disease of

3. M—, æt. 38, formerly obese but now emaciated and passing much sugar. Vision failed to $\frac{5}{6}$ in left and $\frac{2}{3}$ in right, with no changes (examination by Cohn, of Breslau). Vision improved under treatment and did not relapse when sugar subsequently again increased.*

4. M—, æt. 42, one eye nearly blind from old injury, diabetic symptoms for about six months, now passing more than a pound of sugar daily. Vision failing about one month, disc somewhat pale, but no other changes; exact vision and refraction not stated.†

5. M—, æt. 43, passing much sugar but no albumen; not a drinker, but an excessive smoker.

Slight amblyopia of right (V. about $\frac{1}{2}$ and 1 J. slowly), field normal, colours normal. At same time very high amblyopia of left (shadows at centre of field, fingers at 5′ in outer part; inner half of field almost absent). Final recovery of both with improvement of the diabetes. In 1875 (four years after admission), V. quite good.

During recovery of the left in this case the defect passed off in such a way as to leave a scotoma for white and colours at the central, and outer central, part of the field. There were no ophthalmoscopic changes.‡

6. M—, æt. 50, heavy smoker and drinker. May, 1875, equal double amblyopia with central scotoma for colours, but no contraction of fields. V. $\frac{20}{70}$ and 5 J. badly, with correcting glasses. Ophthalmoscope normal. Treated as a tobacco and alcohol case, and V. improved in a few weeks to $\frac{20}{30}$ and 2 J. badly.

Two months later retrogression of right eye only to $\frac{20}{100}$ and 16 J. after patient had returned to business. Central scotoma still present in both, though very slight

optic nerves, viz.: one case of homonymous lateral hemianopia, and another of incomplete atrophy of the optic nerves, with concentric contraction of the visual fields; both of these were in young patients.

* Seegen, 'Der Diabetes Mellitus,' Leipzig, 1870 (Case 114).

† C. E. Fitzgerald, 'Dublin Quart. Journal Med. Science,' vol. 50, p. 226 (1870).

‡ Leber, Graefe, 'Arch. f. Ophth.,' xxi, 3, 206.

in the left (better) eye. Urine now found to contain sugar occasionally. Hitherto only morning specimens had been examined, and sugar had never been present. Now, under treatment for the diabetes, V. improved again very markedly, and remained good at date of last note, October, 1875.*

Leber gives a third case of sudden, high, double amblyopia passing into homonymous hemianopia, with good central vision but irregular contraction of the remaining halves of the visual fields. The patient was a man, aged seventy-three, passing a good deal of sugar. The case does not belong to the present category.

Förster gives the following case also not quite of the same group. 7. F—, æt. 62, in good health, but her urine containing a large quantity of sugar. Came with a central defect of vision in the right eye on April 30th. Next day the eye was quite blind, and remained so, the pupil losing all direct reflex action. The disc, for a time quite normal, ultimately became pale. The left eye was normal both in appearance and visual functions (including an exhaustive examination of the visual field), till the beginning of June, when the disc began to show signs of atrophy.†

8. Colonel in French army, æt. 48. Diabetes (150 grains of sugar a day) for six years before sight began to fail, then vision got gradually worse for two and a half months, when it became stationary ; periphery of field of vision normal. Sight so bad that he could not recognise faces at three yards. No ophthalmoscopic changes. Subsequently some improvement with diminution of glycosuria. Ultimately albumen as well as sugar in urine. No note as to tobacco.‡

9. M—, a clergyman, æt. 44, healthy, but subject to articular gout. Failure of both eyes rather quickly down to V. $\frac{1}{6}$ in June, 1878. Colour perception normal, " visual

* Leber, loc. cit.

† Förster, Graefe, und Saemisch, ' Handbuch,' vii, 187.

‡ Galezowski, ' Recueil d'Ophthalmologie,' 1878, Jan., p. 84.

field intact." No ophthalmoscopic changes. Urine contained a good deal of sugar. No note as to tobacco.*

This case, very imperfectly reported, is given in a short paper on glycosuric amblyopia without ophthalmoscopic changes. This paper is especially interesting as it is the first in which, as far as we know, attention is definitely drawn to the resemblance between this affection and "alcoholic" (*i.e.* tobacco) amblyopia. According to Galezowski,† the differential diagnosis of the two diseases is often very difficult, the most important distinction drawn by him being that whereas "alcoholic" amblyopia always affects both eyes, in the diabetic disease one eye may escape.

10. A case is reported by Mr. Eales, of Birmingham, of central amblyopia (scotoma for red, green, *and blue*) without ophthalmoscopic changes, in a diabetic man, æt. 50, who probably did not smoke. The patient died not long after Mr. Eales saw him.‡

11. Bresgen records the following case :—M—, æt. 24, first seen by Bresgen in March, 1879, six years after onset of diabetes mellitus; symmetrical amblyopia (V. $\frac{20}{200}$ and 4 J. at 5″—10″) with central scotoma for red and green, not for blue; no contraction of field; no ophthalmoscopic changes; refraction Em. Fifteen months later (summer, 1880) vision worse ($\frac{2}{200}$ and 18 J.), scotoma larger, no contraction of fields; ophthalmoscope still normal; quantity of sugar greater. Patient stated that vision improved when he abstained from starchy food. No mention of tobacco.§

Bresgen comments on other cases: his case and others like it are to be classed with the "intoxication" amblyopias, whilst those with defects in the periphery of the field, hemiopia, &c., are probably due to hæmorrhage into

* Galezowski, Ibid., 1879, p. 75.

† Galezowski, loc. cit.

‡ 'Lancet,' 1881, vol. ii, p. 200. Mr. Eales has kindly furnished some further particulars.

§ H. Bresgen, 'Hirschberg's Centralbl. f. Augenheilkunde,' 1881, 31.

the nerves or brain; his own case he compares with Leber's Case 3 (No. 6 above), which was at first diagnosed as "alcohol-tobacco" amblyopia.

In all the cases of amblyopia in which we have ourselves found glycosuria the defect of vision has been double and equal, the fields of vision have been of full size, but over an area extending from the centre outwards to, and usually beyond, the blindspot, acuteness of vision has been much lowered, whilst over the same area perception of red and green has been much diminished or absent.

The failure, which in every case has begun simultaneously in the two eyes, seems to have made progress for about a month and then to have become almost or quite stationary.

All the four patients have been smokers, and except that the failure has been rather unusually rapid and that it quickly came to a standstill, the cases differed in no one particular from the common cases of tobacco amblyopia.

Together with these four cases, we have now five others of the same kind in smokers, viz. one case by Leber (Case 6 above) and the four cases narrated to-night by Messrs. Lawford, Lang, and Morton, in addition to the one already published by us, or ten cases of double central amblyopia in smokers suffering from diabetes.

Cases 2 (Desmarres), 3 (Seegen), 4 (Fitzgerald), and 8 (Galezowski) in men may also be put down as probably belonging to the same group.

Of double equal amblyopia in non-smoking diabetics, no indisputable case seems to have been put on record, for in our own case in a diabetic woman, published in vol. i of the Society's 'Transactions,' the defect of vision was but slight, and only one examination of the patient was made. This case was, however, sufficiently marked to draw attention to the necessity for examining diabetic women more carefully in future as to amblyopia.

Mr. Eales's case, too (9), is on this point not quite conclusive.*

If to these cases we add the opinions already quoted of Begbie, Lecorché, and Galezowski, and the cases mentioned, but not detailed, by Mooren† (three cases of double amblyopia without changes), and Steffan‡ (two cases of double amblyopia without changes and without contraction of the visual fields), and several earlier and imperfectly recorded cases by Testelin § and others, we may probably conclude that amblyopia due to impairment of the centre of the field of vision is not uncommon in diabetes, at least in male sufferers from that disease.

Only future inquiry can decide whether this form of amblyopia (depending, as has been shown, on changes in those nerve-bundles which are central at the hinder end of the nerve, but lie on its temporal side close to the eyeball), is the usual one in diabetes, or whether some other affections of the optic nerve, either of one or of both eyes, may not with equal frequency be so caused. Some cases on record, *e.g.* one of Leber's cases (5), Förster's case (7), and others, show at least the coincidence between diabetes and disease affecting the two optic nerves very unequally; whilst in others hemianopia, doubtless of cerebral origin, has occurred.

Should it eventually appear that, apart from tobacco-smoking, there is a proclivity in diabetes to the particular form of partial disease of the optic nerves, of which we have heard cases this evening, the fact will not be without interest in regard to the changes found in other parts and organs, in this disease.

We may here point out that there seems to be an analogy between the condition we are discussing and the symmetrical neuralgia which occurs in diabetes.

* Mr. Eales tells us he believes his patient did not smoke, but is not absolutely certain.

† Mooren, 'Ophth. Beobacht.,' 1867; 'Ophth. Mittheil.,' 1873.

‡ Steffan, 'Jahresbericht d. Augenheil.-Anst.,' 1872-3.

§ Testelin's two cases referred to by Leber, loc. cit.

Cases of this have been recorded by Worms* (affecting in one case both sciatic nerves, in another both inferior dental nerves), and by Buzzard† (affecting both sciatic nerves). These cases were remarkable by their obstinacy, not yielding to any of the ordinary remedies for neuralgia, but Worms' cases improved with a diminution of the glycosuria, in which they resembled Nos. 2, 3, 5, 6, and 8 in our summary of cases of amblyopia.

It may, however, eventually appear that diabetes acts only as one of many other influences in predisposing those who smoke to that particular disease of the optic nerves, of which central amblyopia is the clinical expression. Should this be true, it will still be very interesting to follow up a number of cases, in order to learn whether the prospect of sight improving is as good if tobacco be relinquished by a smoker who has diabetes, as we know it to be in a smoker who has not diabetes.

Cases of tobacco amblyopia in which vision only improves a little or not at all, even after prolonged disuse or at least permanent reduction of tobacco, are probably known to all ophthalmic surgeons, and the case read by Mr. Lawford this evening, and our own Case 4, open the question as to how many such occur in patients who are also glycosuric?

If both tobacco and sugar have the same specific effect on certain nerves, especially the optic nerves, such cases as these would cease to surprise us.

(*December 14th*, 1882.)

* Jules Worms, 'Gaz. Hebd.,' 1880.

† Buzzard, 'Lancet,' 1882, vol. i, p. 302.

IX. FUNCTIONAL AFFECTIONS.

1. *On the connection between diseases of the eye and affections of the genital organs in females.*

By C. E. Fitzgerald, M.D. (Dublin).

Dr. Mooren's paper on "Disturbances of Vision and Uterine Diseases," which appeared in the September number of the 'Archives of Ophthalmology,' has brought before ophthalmologists a subject which demands more attention from them than it has hitherto received. The field of inquiry which this matter opens up, though surrounded with considerable difficulties, is, I think, suggestive of very important results. The connection between cerebral and renal diseases and affections of the eye has been thoroughly recognised ever since the discovery of the ophthalmoscope, but if we come to look into the literature of the subject of the present paper, we find that it is extremely scanty. That there is a connection between diseases of the eye and affections of the female genital organs will, I suppose, be pretty generally admitted, and yet it is almost impossible to obtain any definite information on the subject. In Dr. Allbutt's admirable work 'On the Use of the Ophthalmoscope,' the first systematic work on the application of ophthalmoscopy to medicine, the matter is disposed of in a short chapter on "The Effects of Menstrual Disorders upon the Optic Nerves." The author himself seems to have been quite unable, after most careful investigations, to establish any connection between the menstrual and visual disorders.

The subject received more recognition in the article by Professor Förster, in the 'Graefe and Saemisch Hand-

book,' in an interesting chapter "On the Connection between Affections of the Genital Organs and the Eye." This certainly placed the whole question on a much surer basis, and established beyond yea or nay the existence of such a connection, but if we omit from this chapter the portions which deal with the symptoms met with in that condition of the uterus, first described by Professor Freund, and termed by him chronic atrophic parametritis, the symptoms in Graves' disease and the disorders of vision occurring during pregnancy and lactation; if, I say, we omit these, the remainder of the chapter is as meagre as the bibliography appended to it. Dr. Gowers in his valuable work, the latest addition to medical ophthalmoscopy, vouchsafes but half a page to the consideration of the influence of diseases of the sexual organs on the eye.

Dr. Mooren's article deals much more largely with the question, though he evidently regards it himself more in the light of a preliminary and suggestive communication. In answer to the objection which has frequently been raised, viz. that there are an untold number of women who have disturbances of circulation from abnormal conditions of menstruation without any corresponding disturbances of vision, he says:

"Of course this is an undeniable fact, but still it does not prove anything positive, since how many people have contracted severe colds without having had pneumonia or rheumatism; and how many workmen lie down to rest on the damp ground without contracting myelitis. We are not to ask the question, why does not the same cause always produce the same effect in the system? It would be much more important to know to what extent any local morbid process can effect the one or other part of the general system."

Dr. Mooren's statistics show in a very marked manner the predisposition of women to the various diseases of the eye, for while 32½ per cent. of those affected were men, 67 per cent. were women. He points out very ably and

suggestively the way in which the various morbid conditions of the uterus, &c., may affect the visual organs through the circulation. When we consider the serious consequences to the general economy which so frequently follow the sudden cessation of the normal menstrual flow, it is not surprising that such a delicate organ as the eye should sometimes be very dangerously implicated when this occurs. The following case illustrates this :

Case 1.—Last January a young lady consulted me on account of sudden impairment of vision of the right eye which had occurred a week previously. The day before it happened she had suffered from a very severe pain at the back of the eye which had lasted for about ten minutes. Her menses had ceased some time before and she had been under the care of a medical man who had prescribed for her with the view of bringing them on again. Her visual acuteness was reduced to being merely able to count fingers at 0·5 m. The ophthalmoscope showed the most intense neuro-retinitis I think I have ever seen. I placed her at once under treatment (the application of the artificial leech, hot mustard baths and the iodide of potassium) and gradually the vision began to improve. Then her menses reappeared and she made a rapid recovery. I saw her the other day and her vision has now risen to some letters of $\frac{6}{12}$ Sn. and 0·5 Sn. with difficulty.

I have had a lady under my care for a long time suffering from disseminated choroiditis with floating opacities in the vitreous. Latterly she had not made the progress I expected, so I requested her to visit a gynæcologist as I found her menses had for a long time been very excessive. The gentleman she consulted informed me that she had multiple fibroid tumours of the uterus. It has occurred to me that possibly the presence of such tumours may very seriously affect the circulation and react in this way on the delicate vascular tissue of the eye.

In proof that even an irritation or inflammation of the mucous membrane of the vagina may have an influence in

producing retinal hyperæsthesia or accommodative asthenopia, Dr. Mooren alludes to the subject of masturbation, a habit much more common amongst women, I believe, than is generally supposed. Whilst quite admitting that the subject is an unsavoury one I cannot understand why, in this country, it seems to be universally agreed upon all hands to taboo all discussion or even mention of it. Gynæcological authors though enumerating it amongst the causes of various maladies with which they have to deal, nevertheless, with but few exceptions, devote little or no space in their treatises to a description of the signs and symptoms which show that the habit is practised. I really feel that it is quite a difficult subject to speak about because it seems to be generally agreed upon that the individual who broaches it, is either a person of a morbidly unhealthy, not to say nasty mind, or the pitiable victim of a hobby. The remarkable paper by Professor Cohn on "Eye Diseases from Masturbation" in the last number of the 'Archives of Ophthalmology,' has brought the matter prominently before our branch of the profession and emboldens me to introduce the subject at our Society.

I shall merely mention two or three cases which have come under my own observation with reference to this subject:

Case 2.—A few years ago a young lady, who brought a letter from the practitioner under whose care she had been in the country, consulted me with reference to her sight, which had been failing for some time previously. She had been seen by the late Mr. Wilson, and one of the most distinguished physicians in our city, but no treatment appeared to have benefitted her. She presented various well marked so-called hysterical symptoms. The country doctor said that there was no uterine or ovarian disease. Beyond a slight haziness of one of the optic discs and pallor of both, there were no ophthalmoscopic signs. The impairment of vision was very considerable, and there was a remarkable dilatation of both pupils, though they

responded to the action of light. She remained under treatment for a long time, and at one period a most hopeful improvement took place, but subsequently there was a relapse, and gradually, but steadily, the vision began to deteriorate, and atrophy of both optic nerves declared itself. It was then that, in spite of the country doctor's assurances, I insisted on her undergoing a thorough examination by a gynæcologist, and, as she consented, I sought the advice of Dr. Macan, the present Master of our Rotunda Hospital. He found what was quite sufficient to satisfy us both that the patient practised masturbation. She commenced attending him, and shortly afterwards he taxed her with carrying on this practice, and informed her that it was useless her coming to him unless she was prepared to give it up. She indignantly denied the allegation, and left him greatly offended. She called upon me some days afterwards, and bitterly complained of what had taken place. I reasoned with her for some time, and finished up by telling her that I quite concurred in the doctor's opinion. After some hesitation, she admitted that it was perfectly true, and that she had practised the habit for about two years previously. Complete atrophy of both optic nerves subsequently ensued, and she is now a blind woman. I have dwelt on these particulars of this case because I think they illustrate one of the difficulties which surround the subject, namely, the course to be adopted by the practitioner when he is satisfied that the patient practises the vice. I do not, by any means, wish to insist that in this case masturbation was the sole cause of the atrophy. I think it is quite possible the morbid process had some other source, but that this unfortunate habit played an important rôle in hastening it, is surely not unreasonable to suppose.

Case 3.—A young lady, lately under my care, consulted me some time ago for asthenopic symptoms. These were greatly mitigated, indeed for a time removed, by the correction of a simple error of refraction in one eye, and

a slight amount of hypermetropic astigmatism in the other. Then the symptoms returned with considerable pain at the back of the right eye. On examination, the disc was found to be distinctly hyperæmic when compared with the other, and there was a slight haziness at its margin. The application of the artificial leech and small doses of the iodide of potassium gave some relief, but it was merely temporary, and at last I insisted on her visiting a gynæcologist. She accordingly consulted a well-known gentleman in our city, who informed me that he found a decided enlargement of the uterus and left ovary, and a considerable amount of irritation in the vagina. In a conversation I had with him subsequently, he told me that he had little doubt she had been practising masturbation. Though quite a young girl, he said the vagina was so large that it readily admitted the speculum. He also told me that he had seen quite a number of cases and that he believed the habit was very extensively practised by females in all classes.

This opinion quite coincides with that expressed to me by one of our most distinguished physicians, who tells me that he has seen a large number of cases in his extensive practice.

I ought to have mentioned that the gentleman under whose care the last case was, one day applied a rather powerful caustic to the vagina, which rendered the parts very tender. For some days after this there was a marked improvement, the patient being able to read a good deal every day without fatigue, the reason being, I conclude, that she was unable to carry on the practice she was addicted to.

I have met with another most pitiable case in quite a young and most attractive girl. My suspicions were aroused some time ago about what was going on, and I have since found them confirmed. I believe she acquired the habit at school. She is now a complete neurasthenic wreck.

One of the great difficulties we have to contend with in this matter is the objection, and a very proper one, which our gynæcological brethren have to making a physical examination in the case of young unmarried women. I quite sympathise with them, and it certainly should not be lightly undertaken.

I fear I have very inadequately brought this subject before the Society, but if I have succeeded in directing attention to a matter which I believe is of grave importance, I shall be satisfied that I have done some good work.

(*March 8th*, 1883.)

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2. *Case of Hemiachromatopsia.*

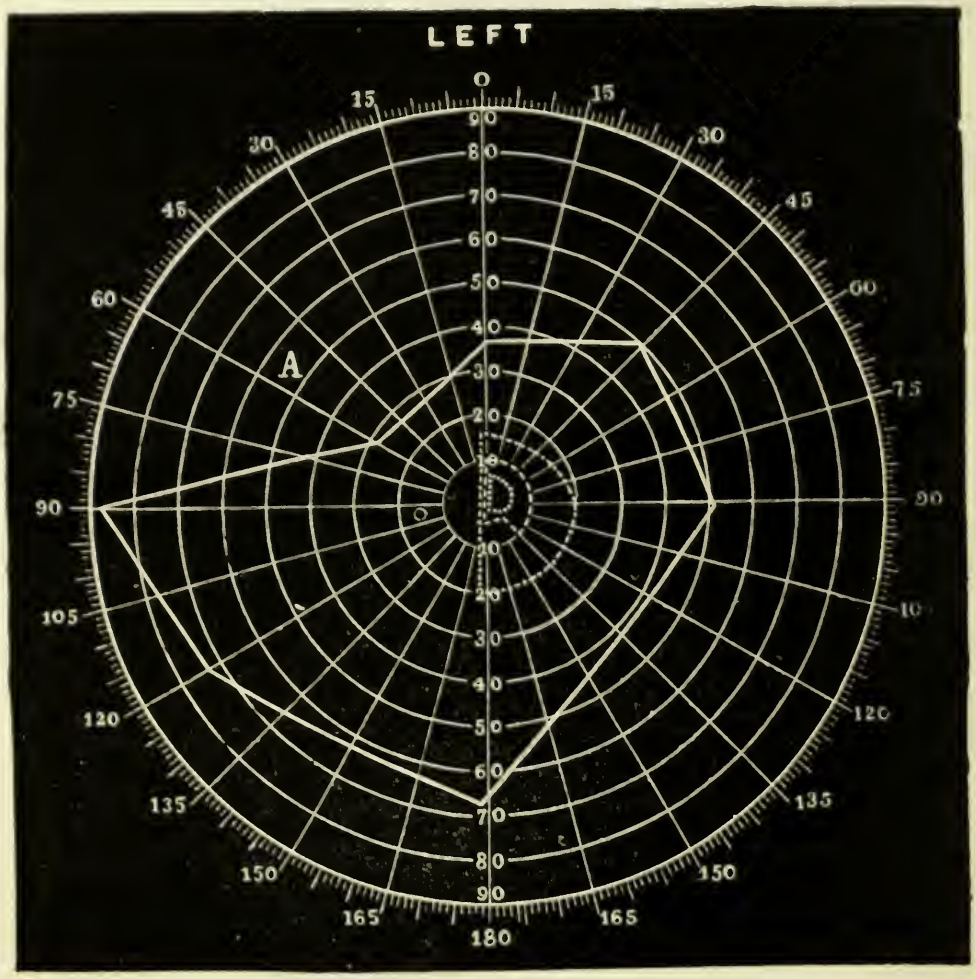
By H. R. Swanzy, F.R.C.S.I. (Dublin).

In the month of November last, a clergyman, æt. 77, on rising in the morning found his head somewhat light and was obliged to make his way back to bed again. He soon became unconscious and remained so until evening. Next day he was much better and in a few days more he had regained his usual good health, except that he at once complained of defective eyesight, and of confusion of ideas if he made any unwonted mental effort. There was not, even for a short time, any hemiplegia, affection of speech, or other paralysis.

Five months later he consulted me, his chief complaint being a difficulty in recognising people even when near to them; and this he referred to defective eyesight. I found in each eye H=1·5 D. and V=$\frac{6}{12}$. In the left eye there was some slight peripheral opacity of the lens, but in other respects the eyes were organically sound. The defect in vision, as estimated by means of the test types, I referred to senile changes in the media and retina, but this comparatively slight defect was not sufficient to

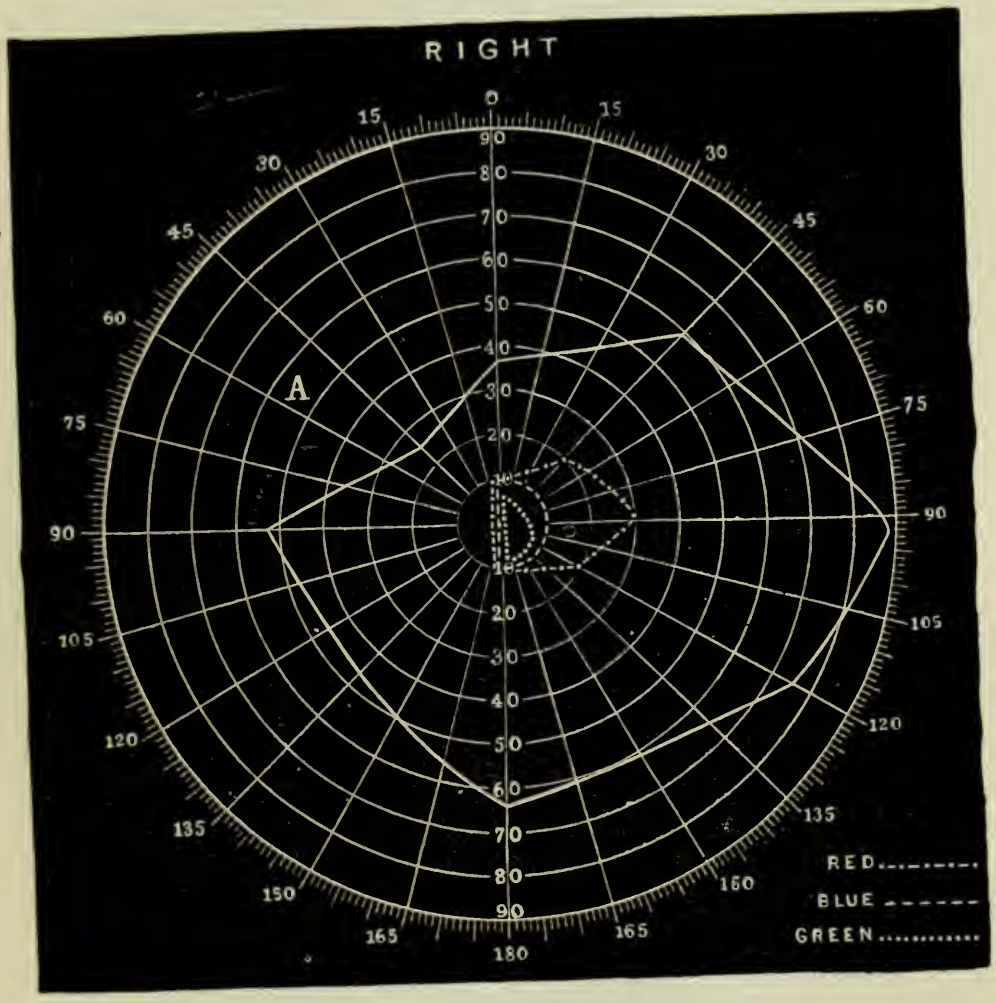
account for his difficulty in recognizing people, and I am inclined to regard this symptom simply as a disturbance of a cerebral function.

I found the fields of vision normal when examined with the perimeter, except for a homonymous defect in the right upper quadrant (A in charts) of each field. When I was



about to test his colour vision by Holmgren's method he said he had found it most difficult to distinguish colours since the attack in November. Nevertheless he performed Holmgren's tests with accuracy. I then examined his fields of vision with coloured wools, and ascertained that the left side of the field in each eye was colour blind,

while with the right side colours could be distinguished, although in an area more restricted than normally (vide chart). I took much trouble to ascertain whether the line of demarcation for colour perception ran through the fixation point, or circumvented that point as does the analogous line in cases of ordinary hemianopsia, and I was able to



satisfy myself that the former arrangement existed. In the accompanying chart the boundary for blue alone is made to pass along the median line, because it was not possible to represent in the diagram the actual condition, in which the median boundary line for every colour was coincident. Red, blue, and green, were the only colours with which

the patient was tested, for I did not wish to lengthen the examination for him by testing with the other and less important colours.

Five other cases of chromatic hemianopsia have been observed. 1. By Landolt (' Charpentier's Thèse,' 1878). A young man with a cerebral affection, particulars of which, and of the other symptoms, I cannot obtain. 2. By Samelsohn of Cologne (' Centralblatt für die Med. Wisssensch.,' 1881, No. 47). The patient was a man æt. 63 whose impairment of vision dated from an apoplectic attack, attended with right hemiplegia, occurring nine months before Dr. Samelsohn was consulted. Sensibility had by that time quite returned and power of motion partially. Vision was compatible with the age and optic media of the patient. The right superior rectus was slightly paralysed. The extent of the fields of vision was normal as tested with squares of white paper of various sizes. Coloured papers, however, showed complete colour blindness of the left half of each field with normal power of colour perception in the right halves up to the normal boundary. 3. By Bjerrum (Hospitals Tidende R. 2 B. 8, p. 51, vide paper by Dr. Berry ' Edin. Med. Journ.,' Feb. 1880). No particulars obtainable. 4 and 5. By Noyes ; (two cases, ' Archives of Ophthalmology,' xi, 217).

The first three cases of chromatic hemianopsia which I have quoted, and the one I now bring under the notice of the Society, all clearly dependent upon a cerebral lesion and without retinal or optic nerve changes, go to prove that the centre for colour perception is situated in the brain and not in the eye or peripheral parts of the optic nerve, and, moreover, that in the brain it is a separate centre distinct from that for the form sense and for ordinary light perception. Of these four cases I think mine is probably the most interesting, as the lesion seems to be so very accurately localised in the chromatic centre of the right cerebral hemisphere.

Noyes' two cases were not I think cases in point, although the author publishes them as such, for in each

there were ophthalmoscopic alterations, and one of them was probably a case of glaucoma, while the other was complicated with spinal symptoms.

In my case the circumstances that red and not blue is the colour with the largest field, and that the fields for colour are concentrically contracted on the right side in each eye are peculiar.

(*July 6th*, 1883).

DISCUSSION

ON

EYE SYMPTOMS IN DISEASES OF THE SPINAL CORD.

June 7th and 8th.

Dr. W. R. Gowers, in opening the debate, said: In the memoranda which, at the request of the Council, I have drawn up to serve as a guide to the discussion this evening, I have mentioned certain points regarding two classes of eye symptoms met with in spinal disease, to which, it seems to me, the attention of the Society might with advantage be chiefly directed. These are "optic nerve atrophy" and "internal ocular paralysis." These topics do not comprehend the whole of the subject announced for discussion. There are many others, such as optic neuritis, nystagmus, and palsy of the extra-ocular muscles, which equally deserve attention. But optic neuritis is extremely rare in association with spinal disease; the meaning of nystagmus is uncertain; and the subject of the palsies of the external muscles is so large, that, while we shall, I am sure, welcome any new facts regarding these points which may be brought forward, it seems better to reserve their special discussion for a future day, and to consider to-night those topics on which we have all had some experience, and about which our knowledge is sufficient to define our ignorance, to indicate the new facts we most need, and the directions in which they must be chiefly sought.

The two subjects to be considered accord conveniently

with the composition of our Society. Optic-nerve atrophy, affecting vision, falls chiefly under the notice of the ophthalmic surgeon; while the most common intra-ocular palsy, that of the iris, causing no symptoms of which the patient is aware, comes as an isolated eye-symptom under the attention of the physician. At the same time, the ophthalmic surgeon, with his exact study of all the morbid elements in the eye, is able to correct and supplement the observations of the physician on intra-ocular palsies, and the medical observer can perhaps supply facts regarding some points connected with atrophy which rarely come under the ophthalmic surgeon's notice.

Two general facts respecting these symptoms deserve attention. The first is that we must regard them as associations, and not effects, of the spinal lesion. The evidence of this is: (1) That disease of any nature may exist in any part of the spinal cord without the occurrence of ocular symptoms, if we except the very rare paralysis of the dilators of the pupil in disease of the sympathetic tract in the cervical region. (2) The ocular symptoms, which may be absent when the cord disease is advanced, may exist in extreme degree when such disease is in a very early stage. (3) With the single exception of the sympathetic symptoms just mentioned, we know of no anatomical connection or functional mechanism by which the spinal-cord disease can produce the ocular symptoms.

The second general fact is that these associated ocular symptoms are always the result of degenerative processes, and their presence shows that the cord disease is also essentially degenerative in its nature. In many cases we do not need the eye-symptom to tell us this. The slow, progressive character of the spinal symptoms is sufficient evidence of the fact. But it is not always so. Degenerative processes of the cord sometimes present sudden exacerbations which may conceal the nature of the underlying process. In these cases the eye symptoms give us information of the highest importance. Their significance

in this respect is not confined to spinal disease, and it is difficult to exaggerate the practical value of indications which, so readily ascertained, at once put us on the track of the morbid tendency from which a patient is suffering.

It may seem surprising, numerous as are the degenerative diseases of the spinal cord, that the chief association of eye-symptoms should be with one of them alone, with locomotor ataxy, tabes dorsalis. But recent pathological discoveries, if they have not penetrated the mystery, at least enable us to comprehend it. As long as ataxy was believed to be a disease limited to the posterior columns of the spinal cord, the association with it of a peripheral degeneration in the optic nerve was an anomaly. But the brilliant researches of Pierret, confirmed as they have been in part by Déjerine, and anticipated in a slight degree by Westphal, have enlarged, and by enlarging altered, our conception of the malady. Pierret has shown that the degeneration in the optic nerve is not the only peripheral lesion, and that that in the cord is not the only central change in this remarkable disease. He has demonstrated that there is, often at least, an independent degeneration in the cutaneous nerves, commencing in their extremities, to which the optic-nerve change is strictly analogous. He has shown, moreover, that there may be a degeneration at the central termination of the optic as well as of other cranial nerves, analogous to that in the posterior columns of the cord. He has thus enlarged our conception of the affection from a limited disease of the spinal cord to a "wide sensory neurosis," as he aptly terms it, in which the optic-nerve atrophy falls into its definite place. The relation which the intraocular palsy bears to the other symptoms of the disease is a point to which I shall return presently. To what extent the degeneration ordinarily preponderates in the central or in the peripheral sensory tracts, only future observation can show. At present we have no means of ascertaining during life the occurrence of the peripheral spinal, or the central optic, changes. We

must still therefore in our clinical study content ourselves with observing the relation between the two which we can recognise.

What proportion of cases of primary atrophy of the optic nerves are connected with spinal disease? So rarely is atrophy associated with other lesions, that the question may practically stand, in what proportion of the cases of atrophy are there indications of ataxy? As the earliest of these, the loss of the knee-jerk may be conveniently taken without risk of more than the most trifling error. The answers which have been given to the question have varied between wide limits. It is only from ophthalmic surgeons that an answer can be looked for, and the combined experience of the members of this Society would certainly yield a result very near the truth. For my own part I will only venture on one very general statement. It is certain that atrophy may precede the symptoms of ataxy which bring the patient under the physician's care. I have seen one case in which the atrophy existed for twenty and another in which it existed for fifteen years previous to the locomotor symptoms. But we know that the loss of the knee-jerk may exist for an almost indefinite time before locomotion suffers. It has not, I think, yet been proved that atrophy ever precedes the loss of the knee-jerk or lightning pains in the limbs, and we are not justified, in an inquiry into facts, in assuming that any case will be followed by spinal symptoms in which none are present at the time of observation.

The converse question—In what proportion of cases of tabes does optic-nerve atrophy occur?—is one to which an answer is difficult to obtain. It is difficult to obtain because the optic and spinal symptoms tend to separate the patients, and often to keep them separate. My own statistics yield about 20 per cent. of atrophy, but I think it is probable that this has been increased by accidental circumstances, and that the estimate, which I gave some years ago, of 15 per cent. will be found to be not far from the truth.

The period of the disease at which atrophy commences

is a point of great practical importance. For the purpose of ascertaining facts, we may conveniently divide the course of the disease into three stages—First, in which there is no affection of the patient's gait; the chief symptoms are the loss of the knee-jerk, rheumatoid and lightning pains, and often unsteadiness in standing with bare feet, toes and heels together, and closed eyes. Second, in which there is distinct ataxic gait, but the patient is able to walk alone or with the aid of a stick. Third, in which the patient is no longer able to walk except with the assistance of another person. There is no doubt that it is common for atrophy not only to commence, but to advance to a considerable degree, in the first stage of the disease. In the cases which have come under my own observation atrophy has commenced twice as frequently in the first as in the second stage, and very rarely in the third; but I think it will be found from a comparison of the experience of ophthalmic surgeons and of physicians that the real excess in the first stage is still greater than this. Moreover, I think that there is a peculiar relation between the symptoms, which increases the difficulty of arriving at a conclusion from partial statistics. It is that when atrophy commences in the early stage the progressive tendency of the atrophy is often strong and that of the spinal disease is slight, and conversely when the atrophy commences later in the disease, after the alteration of the gait has become well marked, the tendency of the atrophy to progress is much less marked. Of course, exceptions are not rare, but I have seen many cases which illustrate the statement. In some early cases atrophy has progressed to complete loss of sight, and for two, three, and even five years there has been no increase in the spinal symptoms, and no affection of gait, even after the steadying influence of vision has been withdrawn. On the other hand, in several cases, amblyopia, coming on during the second stage, has remained stationary for one or two years, and in some has even improved. For instance, a man came under

treatment with the second stage well developed, but still able to walk. Vision had lately failed. There was no affection of accommodation, no distinct limitation of the fields, but acuity was reduced to $\frac{1}{7}$ right and $\frac{1}{10}$ left. Two years later the spinal symptoms had increased, so that he was scarcely able to walk without help, but vision had improved to right 1 : $3\frac{1}{2}$ and left 1 : $4\frac{1}{2}$.

I have not succeeded in tracing any relation between the character of the spinal symptoms and the occurrence of atrophy. It is well known that the atrophy usually commences in one eye before the other, and sometimes reaches a considerable degree before the second eye suffers. I have not met with any case in which sight was entirely lost in one eye while the other was unaffected. Which eye usually suffers first can only be decided by the collection of cases on an extensive scale. The symptoms of the atrophy, the characters of the impairment of vision, their uniformity or diversity, constitute a problem of some practical, and still greater pathological, interest. It is frequently said that colour vision becomes impaired, and the field limited, before central vision fails. Certainly, however, the field for white does not always become limited before acuity of vision is reduced. I have seen several cases in which, when roughly tested with a small object, no peripheral limitation of the field could be found, although acuity and colour vision were considerably impaired. I may mention one case, in the first stage of tabes, with grey atrophy of both discs, apparently equally advanced in the two. There was a considerable difference not only in the degree, but in the mode in which vision was affected in the two eyes. In the right there was only qualitative perception of light, and even this was limited to a narrow zone around the fixing point. With the left eye only No. 16 Jäger could be read at eighteen inches. No green field could be found. That for red was much limited. Those for yellow, blue, and white appeared to be normal. Carefully tested with a white object, half a centimetre square, the patient's field for white was the same

as my own. A year later acuity had failed still further. He could only read No. 50 Jäger. Colour vision was nearly the same as on the previous occasion. The field for white presented only a slight peripheral limitation at the inner and lower part. There was no central loss. I suspect that a careful examination of the mode of failure would show that it presents many variations, just as does the loss of sensation in the legs. Pain may be lost before touch, or touch before pain. The loss may be confined to the soles of the feet, or the soles may alone retain sensibility, which is impaired elsewhere.

Irregular defects in the fields of vision are of considerable interest in connection with another question. Do the symptoms ever suggest that the process which impairs vision is greater in extent and intensity behind the eyeball than at the visible extremity of the nerve? Two cases of ataxy have been recorded, in which there was temporal hemiopia, one of them I showed to this Society, and I have recorded another in which there was a defect in each inner and lower quadrant—viz., partial nasal hemiopia, These cases suggest that the damage to the nerves reached its chief degree at the chiasma. In connection with this question of retro-ocular damage another condition deserves notice, the occurrence of amblyopia without any change in the appearance of the optic discs, or of much greater loss of sight in one eye than the other when the ophthalmoscopic appearances are slight and equal in the two. I have more than once seen this. One patient, for instance, who had no affection of accommodation, and whose discs had a perfectly normal appearance, could only read with one eye No. 2, and with the other No. 10 test type. These cases certainly deserve study.

The impairment of vision sometimes increases very rapidly. A similar rapid increase is occasionally observed in the spinal symptoms. I have more than once known an ataxic patient who could walk fairly well lose in a day or two all power of locomotion. Do the cases with sudden failure of sight, or of rapid but steady course, present any differ-

ences, in the aspect of their discs, from those in which the downward progress is slow? In some patients the disc is clear and excavated; in others it appears to be occupied by a grey, soft, gelatinous-looking tissue, and the vessels are sometimes slightly narrowed. Does any difference in course correspond to this difference in aspect? In the patient with temporal hemiopia the loss came on very rapidly, and the gelatinous aspect of the disc was very conspicuous. Is the colloid-looking tissue seen between the fasciculi in sections of the nerve more abundant in these cases than in others?

Regarding the connection of optic nerve atrophy with other spinal diseases, I have little to say. I have only twice seen it in cases of slight lateral sclerosis, and three times in insular sclerosis, never in progressive muscular atrophy or myelitis. Doubtless the slight frequency with which it occurs in these affections, compared with ataxy, is to be associated with the fact that they for the most part affect the motor, and tabes the sensory, nervous tracts. In general paralysis of the insane, atrophy is somewhat more common, and may be an early symptom; but the question of its frequency, and the spinal symptoms with which it is associated in this disease, I will leave to those who have had larger opportunities of watching the course of the affection.

I pass next to the second part of the subject—the conditions of the internal muscles of the eyeball, of the iris and ciliary muscle, which are met with in spinal disease. These conditions and their probable mechanism have been more than once brought before the notice of the members of this Society. Of the four muscular actions—contraction of the ciliary muscle on accommodation, contraction of the sphincter iridis on stimulation of the optic nerve, contraction of the sphincter iridis in association with that of the ciliary muscle and internal recti, and contraction of the dilator fibres of the iris on stimulation of the skin—of these actions, some or all may be lost in association with spinal disease. They depend on at

least three centres capable of separate action and liable to separate disease, all of which probably lie in a tract beneath the aqueduct of Sylvius, below the front part of the corpora quadrigemina. The experiments of Hensen and Voelckers make it probable that the anterior portion of the tract governs accommodation, and the centre next behind it the reflex contraction of the iris. To the outer side of the latter is a centre on which depends the reflex sensory dilatation of the iris. The efferent paths of the two former are through the third nerve. We as yet know little as to the centre and path for the contraction of the iris which is associated with accommodation. We do not know whether the nucleus for the ciliary muscle is connected with the mechanism for contraction of the pupil at the centre, or in the lenticular ganglion, or in the ganglionic mechanism within the eye. The latter is, on the whole, unlikely, and it is not improbable that the connection is established in the lenticular ganglion. If the connection were at the centre—that is, if the centre for the ciliary muscle caused contraction of the pupil by acting on the adjacent centre for reflex contraction,—the pupil ought not to preserve its action on accommodation, when it no longer acts to light. But, as is well known, the light-reflex is often lost when the associated action is preserved. The path by which stimulation of the skin causes reflex dilatation of the iris is more circuitous. The afferent impulse reaches the centre by the cervical part of the spinal cord when the skin of the neck is stimulated, and the efferent impulse descends the cervical cord, probably passes through a subsidiary centre in the lower part of the cervical enlargement, thence to the superior thoracic ganglion of the sympathetic, and then ascends the sympathetic to the eye.

Like atrophy of the optic nerve, paralyses of the internal muscles of the eyeball are as frequent in locomotor ataxy as they are rare in other diseases of the spinal cord. The most frequent condition is loss of reflex action to light, while the pupil still contracts on an effort at accommoda-

tion, reflex iridoplegia, the "Argyll Robertson phenomenon" as it is termed. With this is often associated, as Erb first pointed out, a loss of the dilatation on stimulation of the skin. How far the association is invariable will be presently considered. Next in frequency, but very much less common, is paralysis of all the muscles within the eye, both cycloplegia and iridoplegia, the "ophthalmoplegia interna" of Hutchinson. The rarest of all is loss of accommodation, cycloplegia, without loss of reflex action. How frequently are these conditions met with in ataxy? Of seventy-two cases of primary degenerative ataxy of which I have notes, the internal muscles of the eyeball were normal in only six; some defect existed in sixty-six, or 92 per cent. Loss of reflex action to light was the only condition in forty-eight; but to these should probably be added six others, in which action to light was very slight, although just recognisable, and one in which the loss existed in one eye only. Thus there was total loss of the light reflex in about two-thirds, and either total or partial loss in about three-quarters of the whole number of cases. In the remaining eleven cases (15 per cent. of the whole) the pupil did not contract on an effort at accommodation, and in most of these it was clear that accommodation itself was also lost. In six there was total loss of accommodation and of the light-reflex, ophthalmoplegia interna. In two cases accommodation was lost in one eye, and action to light in both. In ten cases accommodation was lost in both eyes, and the light-reflex in one only. In two cases accommodation was lost, but the action to light was perfect.

It is well known that these intra-ocular paralyses often occur early in the course of tabes, but the point deserves more exact examination. Of the seventy-two cases twenty-five were in the first stage, twenty-nine in the second, and eighteen in the third. The percentage of the cases with intra-ocular palsy was in the first stage 84, in the second stage 93, and in the third 100. Thus in no case in the third stage, were they absent. These facts

show that in the majority of cases (four-fifths) these ocular complications occur early, but also that cases which escape in the early stage usually suffer during the subsequent course of the disease.

Do these paralyses precede the earliest symptoms of ataxy—occur before even the loss of the knee-jerk? To prove this it is not enough to find, for instance, the loss of light-reflex without spinal symptoms, because we are not justified in assuming, any more than in the case of optic nerve atrophy, that the spinal symptoms will follow. Proof of the sequence can only be supplied by the demonstration of its occurrence, by the observation, for example, of a case in which loss of the light-reflex, at first existing alone, was succeeded by the loss of the knee-jerk. Such a case has not come under my own observation. The nearest approximation to it was supplied by two cases in which there was total loss of the light-reflex and an unequal knee-jerk, and in one of these, two months later, the knee-jerk could no longer be obtained on the side on which it was, at first, the slighter.

When the light-reflex is lost the pupils are often, but not invariably, small, a point to which attention has been directed by Mr. Hutchinson. In two-thirds of the cases they were below 2½ mm. in diameter. In the remainder they were larger, 3 mm. or 3½ mm., and occasionally 4 mm., 4½ mm., or 5 mm. When there is loss of accommodation they are rarely very small, rarely less than 2½ mm., and they are often 4 mm. or 5 mm. in diameter. Inequality in size is common in both conditions, and so also is slight irregularity in shape. I have not succeeded, as a rule, in tracing any relation between the size of the pupil and the other symptoms of the disease. One exceptional case deserves mention. In this, on the side on which the pupil was the smaller, there was unilateral sweating over the head and face; this may be taken as evidence of lessened action of the sympathetic, which, it will be remembered, supplies also the dilator fibres of the iris. In the conditions in which we meet with loss of the light-reflex

this is sometimes modified in a peculiar manner. The pupil contracts under the influence of light, but immediately dilates again to its previous size, and this is maintained, often after a few conspicuous oscillations. Does this condition pass into total loss of the reflex?

The reflex dilatation of the pupil when the skin is stimulated has risen into importance since Erb pointed out that its loss is associated with the loss of the light-reflex. It still remains rather a matter of curiosity than of practical value, but certainly deserves further study. It is a phenomenon closely allied to the contraction of arteries which may be produced in animals by the stimulus of pain. The dilatation of the pupil may be obtained by stimulation of the skin of the face as well as of the neck, and also from other parts, but the neck is the most convenient place. Erb employed the faradaic brush, but the phenomenon can be obtained with equal readiness by any other painful stimulation. The point of a quill pen, for instance, answers well. Like the light-reflex, it is double; stimulation of one side causes dilatation of both pupils. It is not always easy to recognise, and is certainly sometimes absent under normal conditions, especially in persons beyond middle life. In testing for it care must be taken that the pupils are not contracted either under the influence of a strong light or of accommodation. Without doubt the statement of Erb, that this skin-reflex is usually absent when the light-reflex is lost, is true of the majority of cases, but it is not true of all. I have seen several cases in which there was no contraction to light, but well-marked dilatation on stimulating the skin. In these cases the pupils were large; but the skin-reflex may be lost even when the pupils are not below middle size. The skin-reflex may be preserved when the accommodation is lost. Thus, in one patient there was loss of accommodation and no contraction on an attempt to accommodate, in either eye. The right pupil, 4 mm. in diameter, did not contract to light, while the left, 2 mm. wide, did contract. Both pupils dilated when the skin was stimulated.

A peculiar interest attaches to the early occurrence of these intra-ocular palsies in tabes. Almost all the other symptoms are on the side of the sensory system. Here only have we an early lesion in motor structures. It is true the loss of reflex action may be regarded as due to a lesion on the sensory side of the reflex mechanism. But this is improbable, because the loss of accommodation sometimes associated with it can only be due to a degeneration in motor structures. It is extremely probable that the loss of the skin-reflex is of the same nature. Hence, while recognising the peculiar interest which, as Dr. Buzzard has pointed out, attaches to the coincidence of the loss of the knee-jerk and of the light-reflex early in the course of tabes, we must also recognise the difference which almost certainly exists between them, the one being due to a sensory, the other to a motor, lesion.

I have met with this affection of the intra-ocular muscles in no other disease limited to the spinal cord but ataxy. In lateral sclerosis, insular sclerosis, spinal muscular atrophy, and various forms of myelitis it has not been present. Even in cases of combined lateral and posterior sclerosis, in which there is ataxy of gait, inability to stand with the eyes closed, weakness and excessive knee-jerk, these pupil states are absent ; evidence, with other differences, that the process of posterior sclerosis is not the same in site or character as in ordinary tabes.

On the other hand, in general paralysis of the insane, these ocular conditions are frequent, at least in hospital cases. The examples of this disease which are most frequent among hospital out-patients present certain apparent peculiarities. The physical symptoms are the same as in the classical form, indications of spinal degeneration, especially of lateral sclerosis, are frequent, but the mental symptoms are less characteristic. There is usually some mental failure, loss of memory, &c., but there are rarely the optimism, delusions, and expansive delirium, so common in asylums. It may be said, and perhaps it is true, that these are merely cases of the classical form in an

early stage, but certainly their progressive character is little marked. I have watched a few of these cases for a year and a half or two years, without being able to observe any considerable increase in the mental symptoms. In this form the affection of the pupils is almost as frequent as in tabes. Of nine cases, in all of which tabetic symptoms were absent, in six the light-reflex was lost, and in one other it was lessened. In no instance was accommodation lost. Of the frequency of these symptoms in asylum cases of general paralysis I hope we shall hear more to-night.

Intraocular palsy may occur without spinal disease (as Mr. Hutchinson has shown), and such cases deserve careful study. I have notes of fifteen, in eleven of which there was loss of reflex action to light, accommodation being preserved. In four accommodation was also lost. Two patients were the subjects of epileptoid attacks, with some indication of brain failure. In four there had been slight hemiplegia, and, in two of these there was also some mental change. In two there was optic nerve atrophy, unequal in the two eyes, and slight enough in one eye to establish the independence of the reflex loss. In two there was no other nervous symptom, and in the rest only slight and indefinite symptoms. Mr. Hutchinson has shown how frequently syphilis is to be traced in the past history of the subjects of ophthalmoplegia interna and the same lesson is taught by the cases now under consideration. In no less than seven of the fifteen there was a history of constitutional syphilis; two others had had suspicious sores, and in another syphilis, although not proved, was in a high degree probable. I may mention a striking instance of this relation. A woman whose husband had had constitional syphilis came under treatment for an attack of hemiplegia, followed by chronic mental disturbance and weakness of one third nerve. In each eye there was total ophthalmoplegia interna, with large pupils. The husband, who presented no nervous symptoms, had very small pupils, with loss of the light-reflex. The relation of these symptoms to syphilis is of special

interest on account of their frequency in ataxy, and of the much disputed relation of ataxy and syphilis. Like tabes, the pupil symptoms are doubtless due to a degenerative process; but we are on that account as little justified in denying as we should be in affirming a causal relationship. It is a question of fact, and not of theory as to what syphilis can or cannot cause, or at least only so far of theory as it may be necessary to modify our theories in harmony with the facts.

I regret that I am unable to offer any fresh contribution to our knowledge of the pathological anatomy of these affections; but I trust that the deficiency may be supplemented by others. I have thought it better to keep to the region of clinical observation, than to venture into the fair but infertile field of speculation as to the causes which determine the association of the symptoms. For these we must still wait for facts, and we may reasonably anticipate that they will come. In few departments of medical ophthalmology has progress been more rapid, and, it may be added, in few has it been more rich in its practical applications.

On ocular symptoms occurring in general paralysis of the insane. By W. Bevan Lewis (Wakefield).

(Communicated by E. Nettleship.)

In collecting the following notes relative to oculo-motor disturbances occurring during the course of general paralysis, I have been prompted by a desire to extend and confirm certain views advanced by me in the sixth volume of the 'West Riding Asylum Reports' for the year 1876. Mr. Jonathan Hutchinson, in commenting upon these views, erroneously attributed them to my late colleague, Dr. Robert Lawson, and whilst now assuming the full responsibility for such views, I would wish to state that my belief in their accuracy has not been diminished by subsequent observation. The mental anomalies of the general paralytic interfere at times very materially with the full

examination demanded by such cases, the difficulties presented by very advanced cases being quite insurmountable; but by the very valuable assistance offered me by my colleague, Dr. Lloyd Francis, I have been enabled, in prosecuting these inquiries, to obtain fairly extensive and accurate results.

The weak points indicated by Mr. Hutchinson in my former article I have endeavoured to compensate for in these notes, each case being examined with a view to the following points:

1. Actual size, equality, marginal irregularity, and mobile condition of the pupils.
2. Reflex excitability of the pupils to light (diffused and focal).
3. Sympathetic action of the one eye on the other.
4. Reflex dilatation of pupils to a painful cutaneous stimulation.
5. Movements attendant upon accommodation.
6. Visual acuity and accommodative capacity.
7. Appreciation of colour.
8. Spinal conditions as ascertained by deep and superficial reflexes, gait, and general muscular co-ordination.

Loss of reaction to light.—Of the 60 patients examined, 47 cases or 78 per cent. showed a more or less inactive state of the pupils to light, viz. complete immobility to light, diffuse or focal, in one or both eyes in 28 cases, a less complete loss of this reaction in 19 patients. The eyes were tested separately and simultaneously by overshading and by exposure, and the sympathetic contraction of one pupil noted when the other eye was illuminated. In two cases where the right pupil was rigid to light and the movements of accommodation were lost, the *left* pupil, contracting very sluggishly to light, was stimulated to active and extensive range of movement upon illuminating the *right* eye. In the tables appended, the actual size of pupils is given in each case, so that the implication of the sympathetic or third nerve or of both may be decided. The "sympathetic reflex" was completely lost in 24

cases (40 per cent.), and was more or less completely abolished in 18 patients.

Movements on accommodation (associated iridoplegia).—A very large proportion were more or less completely devoid of the movements attendant upon near and distant vision, viz. 26 cases, or a percentage of 43. A perfectly rigid pupil during distant vision and strong convergence occurred in eight cases, both eyes being equally involved; whilst, in several others, these movements were totally abolished in one eye only. In eleven cases (18 per cent.) either the contraction during convergence or dilatation during distant vision were respectively impaired in activity and range.

Reflex dilatation to cutaneous stimulation.—The impairment of this reaction was the most frequent anomaly presented; in 38 cases (63 per cent.) this dilatation of the pupil could not be detected in the slightest degree in either eye and in seven other cases it was completely absent in the one eye. An impairment in the activity and range of this reflex was also noted in 10 cases (16 per cent). In the article already alluded to out of 40 general paralytics examined, this reflex dilatation was completely abolished in 35·8 per cent., whilst it was impaired in activity and range in 30 per cent. more. The much larger proportion of cases showing this anomaly in the present series is accounted for by the fact that few cases were available for examination in the early stage of general paralysis, whilst, in the former series one half the patients examined were in the early stage of the disease.

Loss of sympathetic reflex.—The term used here, indicative of the sympathetic action of one pupil during shading or illumination of the other eye is objectionable but is used for the present for want of a better.* In 24 cases (40 per cent.) this reflex was completely abolished in both eyes and in 6 patients this movement was totally suppressed in one eye only. As to the association of these

* Does not "indirect" express the meaning better? "direct" reflex action of a pupil being that which follows stimulation of the retina of the same eye.—Eds.

anomalous conditions in individual cases, examination of the tables reveals the fact that reflex iridoplegia (inactivity to light, was an *almost invariable accompaniment* of the loss of movements on accommodation; thus, *absolute loss of movements on accommodation* associated with reflex iridoplegia (inactivity to light) occurred in 14 cases or 23 per cent. In all cases alike, when the movements on accommodation were impaired to a minor extent, or wholly abolished, *no reflex dilatation could be produced by a painful cutaneous stimulation.*

Argyll Robertson symptom.—Complete fixity of the pupil (even to focal illumination) without impairment of the movements on accommodation was found in 9 cases *i.e.* 15 per cent. but an incomplete and presumably an early stage of this reflex iridoplegia without implication of accommodation movements was seen in 17 other cases (28 per cent.). Out of the 60 patients examined, but one case was seen where, the pupils being fixed to light, normal dilatation to cutaneous stimulation occurred, and this one case therefore forms an exception to the rule established by Erb, that reflex iridoplegia is *invariably associated* with the loss of this reflex dilatation.

Ophthalmoplegia interna (*Hutchinson*).—As above stated 15 cases (24 per cent.) exhibited a perfectly rigid state of one or both pupils during efforts at accommodation, and in at least four of these patients there was a greater or less degree of cycloplegia; so that paralysis of the whole internal ocular musculature obtained in nearly 7 per cent., though it was rarely complete as regards the ciliary muscle. In several cases when accommodation movements were abolished the actual range of accommodation could not be ascertained owing to the advanced dementia or attendant excitement displayed by the patient. As most of the paralytics were advanced in the last stages of their disease it is quite possible therefore, that the frequency with which ophthalmoplegia interna occurs in the affection may have been under-estimated here. It is however, certain that accommodation is unaffected even, in advanced stages of

general paralysis, in a large proportion of cases, as is evident from the fact that over 28 per cent. could read No. 1 Jäger, fluently and without glasses.

Does ophthalmoplegia interna begin as reflex iridoplegia? I can quote but a single case coming under my observation which seems definitely to indicate this course. Upon admission the pupils were noted as normal; a few weeks subsequently they were dilated and fixed to light but no loss of the movement to accommodation was observed. A few weeks later a very anomalous condition was apparent. When the patient was told to look at a distant object the pupils *contracted,* when made to converge the eyes strongly they distinctly and widely *dilated.* This peculiar condition was associated with a "dimness of vision," a failure of accommodation for near objects (type, &c.), and a failure in the appreciation of colours—red was invariably called blue, blue was termed 'peach,' green and yellow were always recognised correctly. The peculiar motor anomalies were watched carefully and repeatedly by myself and others. At the present time accommodation is still more enfeebled and the movements normally accompanying this adjustment are quite abolished. In this case it appears that ophthalmoplegia interna commenced as reflex iridoplegia, proceeded to a peculiar derangement of the ciliary apparatus, and resulted in nearly complete paralysis of its nervous supply. A large proportion of cases exhibited phenomena which have been suggested as possibly the incipient symptoms of reflex iridoplegia; during focal illumination there was an active initial contraction, then a notable oscillation of the pupil and subsequently a dilatation to its full extent *under sustained illumination.* This feature was witnessed in 17 cases or a percentage of 28.

From the tables appended it will be seen that this initial contraction and secondary dilatation during focal illumination occurs as one of the earlier phenomena of ocular troubles, in general paralysis, the condition of the accommodation movements being usually noted as normal.

In examining the reflex dilatation which accompanies a painful cutaneous stimulation the following precautions should be taken, otherwise an erroneous opinion may readily be arrived at.

1. The eye should be shaded from a glare of light.

2. Voluntary movements on the part of the patient should be completely in abeyance during the test.

3. Strong coffee appears to have the power of inhibiting this reflex (it is well known that caffeine, theine, and allied principles have the power of contracting the pupil).

As regards the latter fact I have frequently failed to obtain this reflex dilatation as the result of the administration of strong coffee shortly prior to the test. The test applied by myself has lately been the pricking of the hand by a sharp needle, and I have wholly discarded the faradaic brush; for this simple procedure appears to me far more satisfactory and efficient. As regards the second indication alluded to above, I have already drawn attention to the fact, that certain *psycho-motor discharges are invariably accompanied by a wide dilatation of the pupil.* If the eyes are carefully shaded from a glare of light, and the pupils carefully watched, they will be perceived steadily to dilate upon raising a leg off the ground; if now the body be poised upon the toes of the other foot, so as to call more complex balancing efforts into play, the pupils dilate to a still greater extent.

Similar actions of the pupil accompany rapid movements of the arms and the same rule may be formulated for masticatory efforts. In fact for the extensive series of movements involved in locomotion, prehension, equilibration, and mastication, this dilatation of the pupil appears to be an invariable accompaniment. I draw attention more particularly to this fact because in testing one's self for reflex dilatation upon sensory stimulation, it is of course important that no muscular efforts be made; and in like manner with a fractious patient a source of error is introduced, which would seriously interfere with the accuracy of our results.

Tabulated results of the Examination of Ocular Anomalie

	Size of pupils in millimetres.	Reaction to light.	Reaction to focal illumination	Sympathetic reflex.	Reflex dilatation.	Movements with accommodation.	Visual acuity.
C. B.	R. 2·25 L. 1·75	R. = Immobile L. = Very slight initial contraction, then dilatation	R. = Immobile L. = Very slight initial contraction, then full dilatation	Abolished	Abolished	Active and equal	Sn. $\frac{d}{D} = \frac{6}{1}$
J. B.	3·25 3·25	Initial contraction, then gradual dilatation to 3·25 mm., with slight oscillations	Similar condition, but the initial contraction more marked	Abolished	Absent	Active and equal	$\frac{d}{D} = \frac{6}{1}$
J. W. M	4·75 3	R. = Nearly immobile, then slight dilatation L. = Slight contraction, followed by similar dilatation	R. = Slight contraction, then slight dilatation L. = Fair contraction, followed by slight dilatation	Normal	Absent	Active and equal	$\frac{d}{D} = \frac{6}{6}$
J. W.	3 3	Very slight contraction	Both contract fairly L. = Oscillates after initial contraction	Normal	Normal	R. = Fairly good L. = Very sluggish	$\frac{d}{D} = \frac{6}{1}$
A. H.	2 3	Both immobile	R. = Immobile L. = *Very* slight contraction	R. = Fixed L. = Slight contraction	R. = Immobile L. = *Very slight* dilatation	R. = Immobile L. = Slight reaction	$\frac{d}{D} = \frac{6}{6}$
J. C. C.	6·5 6	Both immobile	Both immobile	Absent	Absent	Both immobile	$\frac{d}{D} = \frac{6}{6}$
S. S.	2 2	Normal	Normal	Moderate	Normal	Normal	$\frac{d}{D} = \frac{6}{3}$
J. P.	3·25 3·25	Good initial contraction, followed by oscillation and dilatation to former size	Good initial contraction, followed by oscillation and dilatation to former size	Active and equal	Absent	Normal	$\frac{d}{D} = \frac{6}{1}$
S. A. L.	1·5 1·5	Immobile	Immobile	R. = Absent L. = Very slight	Absent	Normal	?
J. M.	2·25 2	Slight initial contraction, then oscillation and dilatation to former size	Slight initial contraction, then oscillation and dilatation	Absent	Absent	Normal	$\frac{d}{D} = \frac{6}{1}$
B. K.	3·5 3·25	Immobile	Immobile	Absent	Absent	Both immobile	$\frac{d}{D} = \frac{6}{5}$
W. T. S.	4·5 4·25	R. = Sluggish L. = Very sluggish	R. = Fairly active L. = *Very* sluggish contraction, then oscillation and dilatation to former size	R. = Active L. = Sluggish	Extremely sluggish or absent	R. = Active L. = Sluggish, and of more limited range	$\frac{d}{D} =$
J. W.	1·75 1·75	Immobile	Both contract, but to *extremely* limited range	Absent	Absent	Extremely sluggish, and of very limited range	$\frac{d}{D} = \frac{6}{1}$

Sixty Cases of General Paralysis of the Insane.

Power of accommodation.	Colour appreciation.	Patellar reflex, &c.	Plantar reflex.	Gait, &c.	Age.	Duration of disease.
$\frac{0\,0}{2\,4}$ = 4 D (roughly)	Normal	Exaggerated No clonus	Diminished	Stiff; no ataxia	46	2 years.
$\frac{0\,0}{2\,1}$ = 5 D (nearly)	Normal	R. = Exaggerated L. = Absent No clonus	Normal	Dragging of right leg; diminished grasp; contractures	47	11 years.
$\frac{0\,0}{2\,3}$ = 4 D (nearly)	Normal	Normal	Normal	Normal; no ataxia	29	1½ years.
$\frac{0\,0}{2\,5}$ = 4 D	Blue = "Green" Other colours normal	Exaggerated	Normal	Impaired locomotion and equilibration; marked ataxic gait	37	6 years.
$\frac{0\,0}{3\,4}$ = 3 D	Normal	Absent	Normal	Stiff, swaying, and unsafe; no marked ataxia	48	7 years.
Too excited)	Normal	Nearly absent	Normal	Brisk, but somewhat stiff; no ataxia	33	4 years.
$\frac{0\,0}{1\,6}$ = 6 D	Normal	Exaggerated No clonus	Exaggerated	Stiff, feeble, tottering; no marked ataxia	32	2 years.
$\frac{0\,0}{2\,5}$ = 4 D	Blue = "Puce" Yellow = "Pink" Others normal	Exaggerated No clonus	Exaggerated	Brisk, stiff, and a little tottering; no ataxia	38	1½ years.
Too excited)	Yellow & green = "Gold" Other colours correct	Absent	Exaggerated	Brisk, fairly elastic; no ataxia	45	4½ years.
$\frac{0\,0}{1\,9}$ = 5 D	Yellow = "Crimson" Other colours correct	Exaggerated No clonus	Exaggerated	Steady, good; no ataxia	33	1½ years.
Too excited)	Red = "Puce" Other colours correct	Exaggerated No clonus	Sluggish	Stiff, slow; drags left leg; no ataxia	46	7 years.
$\frac{0\,0}{2\,5}$ = 4 D	Normal	Absent	Exaggerated	Brisk and elastic; no ataxia	41	4 years.
$\frac{0\,0}{3\,0}$ = over 3 D	Red = "Pink" Yellow = "Purple" Others correctly	R. = Excessive L. = Normal	R. = Normal L. = Exaggerated	Slow, heavy, stiff, and leans to left side	41	3 years.

	Size of pupils in millimetres.	Reaction to light.	Reaction to focal illumination.	Sympathetic reflex.	Reflex dilatation.	Movements with accommodation	Visual acuity.
W. S.	$\frac{4{\cdot}25}{3{\cdot}75}$	Sluggish in both	Initial contraction active, then oscillation and dilatation to former size	Sluggish in both	R. = Almost absent L. = Immobile	Normal	Sn. $\frac{d}{D} = \frac{6}{30}$
C. J. C.	$\frac{4{\cdot}25}{4{\cdot}50}$	Immobile	Immobile	Immobile	Absent	R. = Immobile L. = Normal	$\frac{d}{D} = \frac{6}{15}$
J. B.	$\frac{4{\cdot}5}{4{\cdot}5}$	Slight initial contraction, then slight dilatation	R. = Slight initial contraction, then secondary dilatation L. = Strong initial contraction, then very slight dilatation	Present, but of limited range	Normal	Normal	—
J. H. W.	$\frac{2{\cdot}75}{2{\cdot}25}$	Immobile	Immobile	R. = Slight contraction and dilatation L. = Immobile	Absent	Active and equal	$\frac{d}{D} = \frac{6}{5}$
J. A.	$\frac{3{\cdot}5}{4{\cdot}75}$	R. = Very slight contraction, followed by dilatation L. = Immobile	Immobile	Absent	Absent	?	?
J. M.	$\frac{7}{5}$	Immobile	Immobile	Absent	Absent	Immobile	?
J. L.	$\frac{2{\cdot}5}{3}$	Both react over *very limited* range L. = Least active	Contract actively, but through a very limited range	Present in both; most active in right	Sluggish	Normal	$\frac{d}{D} = \frac{6}{12}$
L. B.	$\frac{3{\cdot}25}{2{\cdot}5}$	Active and equal	Active initial contraction, then oscillation in both, and in the right wide dilatation	Normal	Normal	Normal	$\frac{d}{D} = \frac{6}{10}$
F. S.	$\frac{4}{4}$	Active and equal	Very active and equal	Normal	R. = Active L. = Less active	Normal	$\frac{d}{D} = \frac{6}{12}$
J. T.	$\frac{4}{4{\cdot}25}$	Both very sluggish and to a very limited range	Both very sluggish and to a limited range. Left most active of the two	Neither contract below 4 mm. Both dilate, but sluggishly	R. = Absent L. = Sluggish	Present in both; the right dilates *very actively* for distance, and over a wide range	?
H. M.	$\frac{3}{2{\cdot}75}$	R. = Sluggish L. = Immobile	R. = Fair contraction L. = Immobile	R. = Active L. = Immobile	Absent	R. = Dilates actively and contracts very slightly beyond 3 mm. L. = Immobile	?
A. S.	$\frac{4}{4}$	R. = Active L. = Sluggish	R. = Active, but then oscillates L. = Sluggish, and then dilates widely	Absent	Absent	Normal	$\frac{d}{D} = \frac{6}{18}$
J. H.	$\frac{3{\cdot}5}{3{\cdot}5}$	Both *very* sluggish R. = More active Very slight dilatation on shading eyes	Both very sluggish, then oscillate and tend to dilate to former size	Contraction slight; dilatation active	Normal, but especially active in right eye	Normal	?

Power of accommodation.	Colour appreciation.	Patellar reflex, &c.	Plantar reflex.	Gait, &c.	Age.	Duration of disease.
$\frac{0\,0}{2\,5}$=4 D	Normal	Very sluggish	Normal	Brisk, active, and elastic; no ataxia	40	9 months.
oo excited)	Normal	R.=Normal L.=Exaggerated	Normal	Stiff, leans to left; no ataxia	40	9 months.
oo excited)	—	Exaggerated	Sluggish	Cannot stand or walk	37	16 months.
$\frac{0\,0}{2\,6}$=about 4 D	Normal	Exaggerated	Sluggish	Stiff, but no ataxia	42	3½ years.
oo excited)	?	Absent	Normal	Stiff, somewhat waddling; no ataxia	38	3 years.
oo excited)	Green="Yellow" Other colours correct	Exaggerated No clonus	Sluggish	Cannot stand; contractures	42	6 months.
$\frac{0\,0}{2\,5}$=4 D	Blue="Puce" Yellow="Orange" Green="Puce"? Red="Red"	Exaggerated	Normal	Firm, steady, elastic; no ataxia	45	?
$\frac{0\,0}{2\,5}$=4 D	Normal	Normal	Sluggish	Slow, stiff, heavy, and unsteady; no ataxia	38	3 years.
$\frac{0\,0}{2\,7}$=nearly 4 D	Green="Blue" Other colours correct	Sluggish	Active	Brisk and good	42	6 months.
oo excited)	?	Exaggerated	Absent	Quick, stiff, and somewhat unsteady; no ataxia	25	12 months.
oo excited)	?	?	?	Stooping, legs bent, shuffling, and unsafe; no ataxia	50	7 years.
$\frac{0\,0}{2\,7}$=nearly 4 D	Red="Yellow" Yellow="Green" Green="Crimson" Blue="Blue"	R.=Sluggish L.=Normal	Exaggerated	Stooping, slovenly, and insecure; no ataxia	55	4 years.
oo excited)	?	Normal	Sluggish	Heavy and somewhat tottering; no ataxia	43	12 months.

	Size of pupils in millimetres.	Reaction to light.	Reaction to focal illumination.	Sympathetic reflex.	Reflex dilatation.	Movements with accommodation.	Visual acuity.
							Sn.
K. T.	$\frac{3{\cdot}25}{3}$	Active and equal	Fairly active	Contraction active; dilatation in both *very sluggish*	Extremely sluggish	Normal	$\frac{d}{D}=\frac{6}{10}$
B. H.	$\frac{4}{3}$	Slight contraction and dilatation in both, but equal	Slight in both, but equal	Slight reaction	Absent	Normal	$\frac{d}{D}=\frac{6}{5}$
J. C.	$\frac{2{\cdot}5}{2{\cdot}5}$	Immobile	Immobile	Absent	Absent	Normal	$\frac{d}{D}=\frac{4}{30}$
M. B.	$\frac{3}{3}$	Active and equal	Active	Active	Absent	Normal	$\frac{d}{D}=\frac{6}{5}$
T W. H.	$\frac{2{\cdot}75}{3}$	Immobile ?	Immobile ?	Absent ?	Absent	Extremely active movements	$\frac{d}{D}=\frac{6}{5}$
J. J.	$\frac{4{\cdot}5}{4}$	Immobile	Immobile	Absent	Absent	Sluggish	$\frac{d}{D}=\frac{6}{6}$
J. B.	$\frac{3{\cdot}25}{3}$	R. = *Very* sluggish reaction, good range L. = Immobile	R. = *Very* sluggish, but of good range L. = Immobile	Absent	R. = Active L. = Immobile	Contraction active in both, but left does not dilate for distance beyond 3 mm.	?
M. K.	$\frac{2}{2{\cdot}75}$	R. = Immobile L. = Very sluggish, but of very limited range	R. = Immobile L.—Very sluggish, of very limited range	R. = Absent L. = Active	Absent in both	R. = Immobile L. = Over a very limited raage	$\frac{6}{6}$
E. A. W.	$\frac{4{\cdot}5}{4{\cdot}5}$	Immobile	R. = Immobile L. = *Very slight*	Active	Absent	Do not contract, but dilate somewhat for distance	?
S. L.	$\frac{2{\cdot}5}{2{\cdot}5}$	Both react to a *very limited range ;* do not dilate on shading	Both contract over very limited range; do not dilate on shading	Sluggish	Absent	Absent	?
A. D.	$\frac{4{\cdot}25}{4{\cdot}5}$	Immobile	Immobile	Absent	Absent	R. = Absent L. = Extremely slight and sluggish	$\frac{d}{D}=\frac{6}{12}$
H. M.	$\frac{3{\cdot}5}{3}$	Immobile	R. = Extremely slight dilatation, but prolonged by shading L. = Decided dilatation, followed by contraction	Absent	R. = Very slight L. = Immobile	Extremely slight dilatation ; no contraction beyond $\frac{3{\cdot}5}{3}$ upon convergence	$\frac{d}{D}=\frac{6}{10}$
M. J. R.	$\frac{4{\cdot}5}{4}$	Active and equal	Active and equal	R. = Very sluggish L. = Normal	Scarcely appreciable	Normal	$\frac{d}{D}=\frac{6}{10}$
E. J. E.	$\frac{2{\cdot}75}{2{\cdot}75}$	Active and equal	Active and equal	Apparently absent	Absent	Normal	?
M. H.	$\frac{4{\cdot}5}{3{\cdot}75}$	Active	R. = Contracts actively, but to limited range, then dilates fully L. = Contracts actively, and to small size, and remains so	Absent	R. = Active L. = Slight	Normal	$\frac{d}{D}=\frac{6}{8}$

Power of accommodation.	Colour appreciation.	Patellar reflex, &c.	Plantar reflex.	Gait, &c.	Age.	Duration of disease.
)o excited)	Normal	Very sluggish	Very sluggish	Swaying and tottering, leans to right side; no ataxia	49	12 months.
$\frac{0\,0}{2}$ = 2·5 D	Normal	Exaggerated	Normal	Stiff and slow; no ataxia	45	12 months.
$\frac{0\,0}{0}$ = 5 D	Green = "Yellow" Other colours correct	Absent	Normal	Stiff, slovenly; no ataxia	37	6 months.
$\frac{0\,0}{8}$ = 5·5 D	Normal	Normal	Sluggish	Brisk and good	32	18 months.
$\frac{0\,0}{0}$ = 5 D	?	R. = Slight L. = Absent	Normal	Stiff; no ataxia	42	12 months.
ubtful	Green = Not known Other colours correct	R. = Normal L. = Exaggerated	Normal	Somewhat stiff; no ataxia	38	3 years.
?	Red and blue = correct Other colours doubtful	R. = Exaggerated L. = Normal No clonus	Exaggerated	Heavy, swaying, and slovenly; no ataxia	52	9 years.
ads none of Snellen ıp to 4	?	Excessive No clonus	Exaggerated	Swaying, unsafe; no ataxia	32	2½ years.
?	?	Exaggerated No clonus	Normal	Cannot stand; no ataxia	43	4 years.
	?	Normal No clonus	R. = Defective L. = Normal	Cannot stand; no ataxia	49	3 years.
ellen 3, near point 0 cm.	Normal	Normal	Diminished	Cannot stand; no ataxia	39	3 years.
ellen 1, near point 5 cm.	Red and blue = correct Yellow and green = correct, but dubious	R. = Normal L. = Sluggish	Exaggerated	Good	42	12 months.
ads No. 2 J. fluntly; Snellen ·5 at 0 cm.	Normal	Exaggerated No clonus	Normal	An unsteady jog-trot; no ataxia	37	2 years
?	Normal	Exaggerated No clonus	Nearly absent	Slow, but steady; no ataxia	43	3½ years.
?	Normal	Absent	Normal	Stooping and slovenly; no ataxia	44	8 months.

	Size of pupils in millimetres.	Reaction to light.	Reaction to focal illumination.	Sympathetic reflex.	Reflex dilatation.	Movements with accommodation.	Visual acuity
E. A. W.	$\frac{5}{5{\cdot}25}$	Active	Active and equal	Normal	Absent	Dilate for distance; no contraction beyond $\frac{5}{5{\cdot}25}$ for convergence	Sn. $\frac{d}{D}=\frac{6}{1}$
A. F.	$\frac{2}{3{\cdot}5}$	Active, but of limited range	Active, but of limited range	Absent	Absent	Normal	$\frac{d}{D}=\frac{6}{3}$
J. G.	$\frac{4}{3}$	Immobile	Immobile	Absent	R. = Absent L. = Active	Absent	$\frac{d}{D}=\frac{6}{2}$
L. R.	$\frac{3{\cdot}5}{3{\cdot}5}$	Immobile	Immobile	Normal	Normal	Normal	?
E. B.	$\frac{4{\cdot}5}{5}$	Active, but the right contracts to smaller size	Both very active, but left recovers former size under full glare of light	Normal	Absent	Normal	$\frac{6}{12}$
E. C.	$\frac{3}{3}$	Active, but left has a more extensive range	Active, but the left has a more extensive range	Normal	Normal	Contraction upon convergence *very sluggish*; dilatation active	$\frac{d}{D}=\frac{6}{1}$
S. C.	$\frac{4{\cdot}5}{6}$	Very active contraction, followed in left by oscillation and dilatation to former size (6mm.)	Both = Active contraction L. = Then oscillates and dilates again to 6 mm.	R. = Very sluggish L. = Very active	Absent	Absent	R. read Sn. $\frac{2{\cdot}5}{15}$ L. read Sn. $\frac{2{\cdot}5}{1}$
J. H.	$\frac{4}{3}$	Immobile	Immobile	Immobile	Both immobile	R. = Immobile L. = Active	R. $\frac{d}{D}=\frac{6}{6}$ L. $\frac{d}{D}=\frac{6}{6}$
G. B.	$\frac{2}{2{\cdot}25}$	Immobile	Immobile	Absent	Absent	Active and equal	$\frac{d}{D}=\frac{6}{6}$
J. D.	$\frac{4}{3{\cdot}25}$	Sluggish reaction	Contracts to very limited range, then oscillates and dilates again	Normal	Normal	Fairly active L. = More sluggish dilatation than right	R. $\frac{d}{D}=\frac{6}{1}$ L. $\frac{d}{D}=\frac{6}{2}$
F. B.	$\frac{3}{4}$	Immobile	Immobile	R. = Active L. = Immobile	Absent	R. = Normal L. = Almost nil	R. $\frac{d}{D}=\frac{6}{2}$ L. $\frac{d}{D}=\frac{6}{6}$
U. P.	$\frac{2{\cdot}45}{4}$	Normal	Normal	Normal	Absent	Normal	$\frac{d}{D}=\frac{6}{1}$
A. S.	$\frac{3{\cdot}5}{4{\cdot}5}$	Normal	Normal	Slight and sluggish	Absent	Of good range, but sluggish	$\frac{d}{D}=\frac{6}{6}$
J. O. O.	$\frac{3}{4}$	R. = Slight reaction L. = Strong reaction	R. = Slight reaction L. = Strong reaction	Normal	Absent	Normal	$\frac{d}{D}=\frac{6}{1}$

Power of accommodation.	Colour appreciation.	Patellar reflex, &c.	Plantar reflex.	Gait, &c.	Age.	Duration of disease.
ds No. 1 J. flu-itly	Normal	Absent	Acute	Fairly good; no ataxia	32	7 years.
?	Normal	Absent	?	Firm and steady; no ataxia	37	2 years.
not read small type	Normal	Normal	Exaggerated	Swaying and slovenly; no ataxia	37	?
?	Green = dubious All others correctly	R. = Normal L. = Exaggerated No clonus	Exaggerated	Good, steady; no ataxia	45	?
ds No. 1 J. flu-itly	Normal	Normal	Normal	Good; no ataxia	33	12 months.
ds No. 1 J. flu-itly	Normal	Normal	Normal	Good; no ataxia	37	?
not read Nos. 1 to J.	Normal	R. = Sluggish L. = Absent	Very sluggish	Slow, stiff; no ataxia	36	2½ years.
btful	Normal	Absent	Exaggerated	Brisk, elastic; no ataxia	47	3½ years.
$\frac{0}{0}$ = 5 D	Normal	Slightly exaggerated	R. = Normal L. = Sluggish	Quick, elastic; no ataxia	35	6 months.
$\frac{0}{8}$ = 3·5 D	Yellow = "Purple," but corrected when purple is shown Other colours correct	Sluggish, especially left	Normal	Quick, elastic; no ataxia	51	2½ years.
$\frac{0}{5}$ = 4 D	Normal	Normal	Sluggish	Steady and firm; no ataxia	37	6 years.
$\frac{0}{2}$ = over 4 D	Normal	Exaggerated No clonus	Normal	Good; no ataxia	35	4 months.
$\frac{0}{3}$ = over 4 D	Normal	Slightly exaggerated	Normal	Brisk and elastic; no ataxia	39	12 months.
$\frac{0}{4}$ = 4 D	Normal	Exaggerated	Normal	Brisk, but somewhat stiff	41	12 months.

	Size of pupils in millimetres.	Reaction to light.	Reaction to focal illumination.	Sympathetic reflex.	Reflex dilatation.	Movements with accommodation	Visu acuit
							Sn.
C. G.	$\frac{6}{6}$	Extremely sluggish	Active contraction, then oscillation and dilatation in both	R. = Very sluggish L. = Still more so	R. = Sluggish L. = Absent	Present in both, but very sluggish	$\frac{d}{D} = \frac{1}{4}$
T. T.	$\frac{4}{4}$	Very sluggish, and to very limited range	R. = Active L. = Very sluggish, and limited range	Sluggish	Slight in both	Normal	$\frac{d}{D} = \frac{1}{2}$
J. L.	$\frac{4\cdot5}{4\cdot5}$	Immobile	Immobile	Immobile	Absent	Immobile	?
W. M.	$\frac{3}{3}$	Immobile	Immobile	Immobile	Absent	Absent	R. $\frac{d}{D} =$ L. $\frac{d}{D} = \frac{1}{1}$
J. H. L.	$\frac{4}{3\cdot75}$	Active and normal	Active and equal	Active and equal	Normal	Normal	$\frac{d}{D} =$

To summarise:

1. A loss of reflex dilatation to sensory stimulation (as high as the fifth nerve) occurs in a very large proportion of cases of general paralysis of the insane.

2. Reflex iridoplegia (loss of action to light), next to this condition, is the more frequent accompaniment of the disease.

3. Complete loss of movements on accommodation occurred in fifteen out of sixty cases (25 per cent.) in one or both eyes.

4. Cycloplegia, more or less complete, was associated with the latter in a few cases (at least four).

5. This condition of imperfect ophthalmoplegia interna was present only in the most advanced stages of the disease, and in one case appeared to commence as reflex iridoplegia.

6. Reflex iridoplegia, except in one case, was always present when the movements on accommodation were impaired or lost.

7. As seen from the tables, spinal symptoms (absence of patellar reflex, &c.) were by no means specially associated

Power of commodation.	Colour appreciation.	Patellar reflex, &c.	Plantar reflex.	Gait, &c.	Age.	Duration of disease.
=over 5 D	Green = uncertain Other colours correct	Normal	Normal	Elastic and brisk; no ataxia	39	9 months.
=nearly 6 D	Normal	Absent	Normal	Brisk; left leg somewhat stiff; no ataxia	34	2 years.
tful	Normal for red and blue Other colours doubtful	Greatly exaggerated Clonus in both	Extremely sluggish	Quick and elastic; no ataxia	36	6 years.
=5 D	Normal	Doubtful	Normal	Good; no ataxia	38	5½ years.
=5 D	Normal	Normal	Normal	Brisk, firm, elastic; no ataxia	41	12 months.

with the more grave ocular troubles in general paralysis, and appeared to bear no decided relationship to the latter.

8. Judging from the nature and progress of the disease, its duration, the history of cases in the earlier stage, and the condition of the more advanced paralytics, it would seem that the sequence of morbid phenomena occurring in the iris in this disease observed the following course:

(*a*) Paralysis of reflex dilatation to cutaneous stimulation.

(*b*) Reflex iridoplegia (loss of action to light), probably shown at first by an initial contraction followed by dilatation under full focal light, and passing into a later stage of immobility, the pupils varying much in size in individual cases.

(*c*) Partial, and occasionally complete, ophthalmoplegia interna.

Summary of Individual and Associated Anomalies met with in Sixty cases Examined.

Loss of reaction to light.

Complete in both eyes . . .	23 cases	(38 per cent.)
„ one eye only . .	5 „	(8 „)
Incomplete in both eyes . . .	15 „	(25 „)
„ one eye only . .	4 „	(6 „)

Loss of "sympathetic reflex."

Complete in both eyes . . .	24 cases	(40 per cent.)
„ one only	6 „	(10 „)
Incomplete in both eyes . . .	8 „	(13 „)
„ one only	4 „	(6 „)

Loss of "Reflex dilatation to cutaneous stimulation."

Complete in both eyes . . .	38 cases	(63 per cent.)
„ one only	7 „	(11 „)
Incomplete in both eyes . . .	8 „	(13 „)
„ one only	2 „	(3 „)

Loss of movements accompanying accommodation.

Complete in both eyes . . .	8 cases	(13 per cent.)
„ one only . . .	7 „	(11 „)
Incomplete in both eyes . . .	7 „	(11 „)
„ one only . . .	4 „	(6 „)

Associated symptoms.

Impairment of light-reflex and reflex dilatation to cutaneous stimulation	36 cases	(60 per cent.)
Impairment of light-reflex and sympathetic reaction of the one pupil on illumination of its fellow	„	„

Impairment of light-reflex associated with loss of movements of accommodation :

DESCRIPTION OF PLATE XIV.

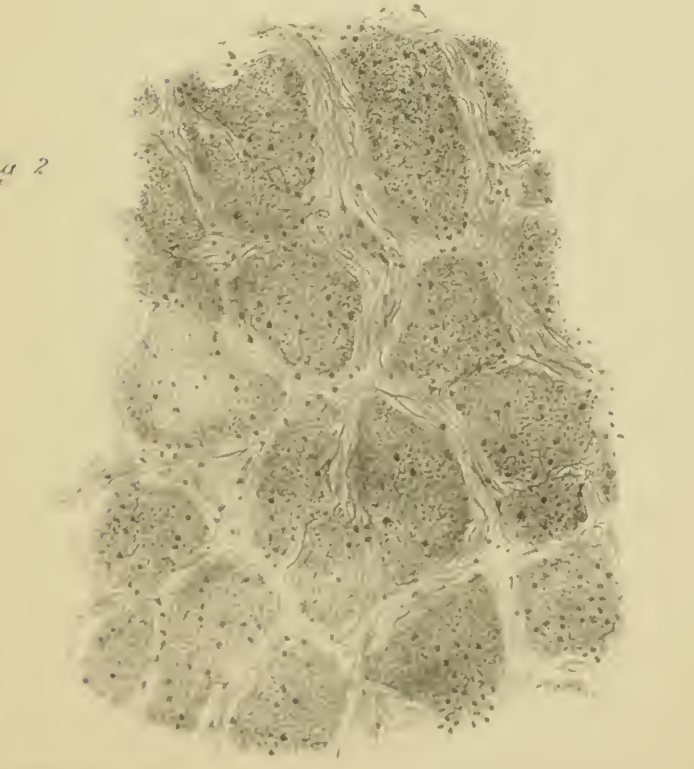
Illustrating Mr. Lawford's cases of General Paralysis of the Insane with Atrophy of the Optic Nerves (p. 221). From drawings by Mr. M. H. Lapidge.

FIG. 1 (low power) and FIG. 2 (high power).—Transverse section of optic nerve (from Case 3) immediately behind globe. The portion of nerve shown is close to the central vessels. The trabeculæ are greatly thickened, and have acquired a wavy, somewhat translucent appearance, contrasting markedly with the straight, regular trabeculæ of a healthy nerve (see Gowers's 'Med. Ophthal.,' pl. xvi, fig. 6). But few staining nuclei are to be seen in these trabeculæ; the number of nuclei scattered through the nerve-bundles is greater than in healthy specimens. The nerve-fibres do not appear to have undergone much degeneration, though many of the bundles are undoubtedly diminished in size, and in some parts of the sections it could be seen that one or two bundles of nerve-fibres had almost entirely disappeared. There is considerable increase of fibrous tissue surrounding the central vessels and some of the smaller arteries. The coats of the vessels are slightly, but not markedly, thickened, and no obliterated vessels were found. These changes are very unequally distributed, the appearance of the section in some parts differing very slightly from that of a healthy nerve. The inner sheath of the nerve is thickened, and the continuity of the trabeculæ with it is unusually well shown. To the naked eye the optic nerve appeared of about normal size, but too white.

Fig. 1.

One Millimetre
× 24.

Fig. 2.



Tenths & Hundredths of a Millimetre
× 122

1st. With absolute loss of accommodation movements in both eyes	7 cases	(11 per cent.)	
2nd. With absolute loss of accommodation movements in one eye only . . .	7 „	(11 „	)
3rd. With more or less impaired accommodation movements	21 „	(35 „	)
Immobility to light with normal accommodation movements (Argyll Robertson symptom) .	9 „	(15 „	)
Simple impairment of reaction to light, with normal accommodation movements.	17 „	(28 „	)
Colour anomalies	14 „	(23 „	)

Spinal reflexes.

Patellar reflex exaggerated in	20	cases.
„ abolished	„ 13	„
Ankle clonus occurred	„ 1	case.

Seven cases of general paralysis of the insane with atrophy of the optic nerves. By J. B. Lawford, M.D.

(With Plate XIV.)

Case. 1—Ralph M—, æt. 47, married, six children. Had been a sober man. Admitted February 16th, 1878; died December 1st, 1878. Well-marked ataxic gait. Total loss of patellar reflex. Paralysis of sphincter ani. Epileptiform fits.

April 11th, 1878.—Optic discs certainly atrophied.

July 6th.—Note by Mr. Nettleship: "Both optic discs pale and somewhat indistinct, especially on y. s. side. Vessels about normal. Right disc paler than left, and changes in it marked. Vision fairly good."

October 4th.—Now nearly blind. Optic discs white.

Post-mortem.—Sclerosis of posterior columns of spinal cord was found on microscopical examination.

Case 2.—Clarence E. V—, æt. 37, married, one child. Had been intemperate. Admitted November 4th, 1872; died June 28th, 1877. Had an epileptic fit some years previously, and had epileptiform convulsions of right side soon after admission.

February, 1877.—Commencing contraction of lower limbs.

March.—Lower limbs contracting rigidly; it is difficult in consequence to keep him in a chair.

April.—Reflexes of legs much exaggerated. Lower limbs cannot be straightened. Right arm wasted and contracted. Right optic disc pale and atrophied, vessels small. Seems to see well.

June.—Both optic discs atrophied.

Case 3.—Edwin M—, æt. 47, married, twelve children. Had been a sober man. Admitted September 6th, 1880; died April 26th, 1883. On admission atrophy of optic discs was noted. There was great exaggeration of patellar and other deep reflexes; and excessive irritability of all muscles to induced current. His limbs soon became contracted rigidly and could not be straightened.

November 8th, 1881.—Both optic discs pale, right more so than left.

December, 1882.—Both optic discs very pale and white, edges sharply defined. Lamina cribrosa exposed and very distinct. Vessels normal or (?) slightly diminished. Pupils rather wide, act badly to light. Vision cannot be tested but patient is not blind. (Plate XIV is taken from sections of the optic nerves in this case.)

Case 4.—Francis R—, æt. 30, single, intemperate. Admitted April 26th, 1881; died March 27th, 1883.

October 1st, 1881.—Marked exaggeration of patellar reflex.

November 10th.—Reads 2 N. with each eye at $5\frac{1}{2}''$; R. p. = $3\frac{1}{2}$ mm.; L. p. = $2\frac{1}{2}$ mm. Both act to accommodation; badly to light. Ophthalmoscopic appearances normal.

November, 1882.—Limbs now greatly contracted. Reflexes all excessive.

January 16th, 1883.—Pupils equal, measuring $2\frac{1}{4}$ mm., action to light very slight, though appreciable. Right optic disc very pale, atrophied, edges sharply defined. Lamina cribrosa exposed. Left optic disc atrophied, not so pale as right, slight indistinctness of edges. (Examination difficult). Vision very defective, but patient not blind. Examined microscopically, the optic nerves show changes similar to those in the nerves of Case 3 (see Plate XIV) but of slighter degree.

Case 5.—Henry W—, æt. 37, married, three children, a sober man. Admitted October 18th, 1881, at present date is still in hospital. Has a somewhat spastic gait and exaggeration of patellar reflex. Has had epileptiform fits.

November, 1881.—Reads 2 N. Ophthalmoscopic appearances normal.

February 16th, 1883.—Left optic disc pale, edges too sharply defined. Vessels normal size. Right optic disc very white, edges very sharply defined, lamina cribrosa too plainly visible; vessels about normal. Vision = 8 N. badly, refraction Em.

P.S.—Sept. 20th.—The patient is now so blind that he cannot see to walk about alone. The atrophy of the discs has progressed considerably.

Case 6.—Eliza M. B—, æt. 40, married, no children, intemperate. Was in Bethlem Hospital from March, 1879, to December, 1880, and was diagnosed as a case of general paralysis of the insane.

In October, 1881, she attended as an out-patient at St.

Thomas's Hospital under Mr. Nettleship, who has kindly given me the following note which he then made :—" Left eye no p. l. Right eye sees hand correctly. Both optic discs in an advanced stage of atrophy, yellowish white; edges quite abrupt and sharp, except above and below; arteries considerably diminished, and along some there are white lines. Appearances consistent with, but not conclusive of, previous neuritis."

There are no notes of spinal symptoms. She died in March, 1882, " quite blind."

Case 7.—John A—, æt. 38, single, sober. Admitted June 27th, 1880. Died March 10th, 1881. No notes of spinal symptoms. Had epileptiform convulsions, chiefly unilateral.

July 9th, 1881.—Patient says he can only distinguish light from darkness with the right eye; this eye diverges, the pupil is larger than the left, and inactive to light; optic disc pale all over, and arteries diminished. Left eye apparently normal in all respects.

The foregoing cases occurred in Bethlem Hospital. I am indebted to Dr. Savage, the resident physician, for permission to use them. I have copied from the case-books only such notes as refer to the condition of the optic nerves, or to spinal symptoms. I regret that they are not more complete.

In five of the seven cases there were spinal symptoms; in one posterior sclerotic, and in four lateral sclerotic, symptoms. In this connection, and having in view Dr. Gowers' question, " When eye-symptoms occur in general paralysis of the insane, is the case more likely to be complicated with spinal symptoms ?"—I may mention that in the last seven months I have examined at Bethlem Hospital twenty-two cases of general paralysis, in three of which I found atrophy of the optic nerves; these three are Cases 3, 4, and 5 above-mentioned, and in all three there were symptoms indicative of changes in the lateral

columns of the spinal cord. In reference to the changes in the pupils, I am disposed to think from my small experience that they occur as frequently, if not more frequently, in cases free from spinal symptoms; I have examined too few cases to be able to form a more definite opinion on this point.

Double optic neuritis in the early stage of disseminated sclerosis; incomplete atrophy of optic discs. By Seymour J. Sharkey, M.D.

Amelia F—, æt. 34, was lately an out-patient under my care at St. Thomas's Hospital and then for a short time an in-patient under Dr. Bristowe, who kindly allows me to use his additional notes of the case.

Her symptoms seem to have begun about five years ago (1878) with giddiness, thickness of speech, trembling, especially on the right side of the body, and severe headache. After about three months the sight of the left and afterwards of the right eye failed (details of her state at that time are given further on). A year and a half ago, after her last confinement, she became weak, especially losing power in the legs, and became unable to walk without help.

Condition on admission, early in 1883.—Mental condition good; special senses, except sight, natural. Sight dim in both eyes (V. about $\frac{20}{100}$, not improved by glasses, reads 12 J.). Pupils act well to light and accommodation; neither nystagmus nor ocular palsy. Incomplete atrophy of both optic discs; discs pale, their margins clearly defined, and the central vessels of normal size. Sometimes has tingling in right half of face and tongue, and in right arm. Rhythmical tremors of arms on exertion, becoming worse in proportion to the effort made, and worse in the left than the right. Legs weak, she totters very much in walking; tremors on using the legs. No affection of lips or tongue; no paralysis of any part,

no rigidity, and no wasting; no affection of sphincters. Patellar reflex much exaggerated, especially on right side; wrist reflexes exaggerated; ankle clonus on right side, not on left. All the viscera normal; urine contains no albumen.

Family history.—Patient's mother died of "general decline." Patient's father is living; he was once insane for a short time, but has recovered. Patient had two aunts who became insane; one of whom died in that condition, the other recovered, and is now well. A sister is very subject to nervous headaches. Patient has had three children (all in good health) and two miscarriages. Previous health good.

In October, 1878, she was under the care of Mr. Hulke at Moorfields for optic neuritis and defective sight in the left eye. She was then twenty-nine, and had been suffering for about three months from the nervous symptoms already mentioned. For three weeks the sight of her left eye had been getting dim; Mr. Hulke (who kindly allows me to make use of his notes) found the disc much swollen, its contour hidden, and the retinal veins very turgid, vision reduced to counting fingers at a few inches. She remained under treatment, taking iodide of potassium, and subsequently strychnia, for five months, and the sight improved so that in December (two months after admission) she read 10 J. with the defective eye. Mr. Hulke's current notes mention, at various dates, complaints of drowsiness, a sensation of coldness and pinching in the right leg and in the tongue, tingling in the right arm, jerking of the left leg when the foot touched the ground with consequent difficulty in walking, and trembling of the limbs. Mr. Hulke's notes make no mention of neuritis in the right eye, but the present state of its disc, and the patient's statement that vision failed in this eye after the left, make it almost certain that the neuritis was double.

Amblyopia with changes indicative of slight chronic neuritis, in disseminated sclerosis. By E. NETTLESHIP.

WM. S—, æt. 42, was under Dr. Sharkey's care in February, 1880, with symptoms of disseminated sclerosis, which had been present for some time. I had the opportunity of examining his eyes several times. Unfortunately the notes of his spinal symptoms have been lost; I recollect, however, perfectly well that Dr. Sharkey brought the man to my out-patient room as an example of optic nerve disease in disseminated sclerosis.

He said that his sight had been failing for the last twelve months; till then he had always been near-sighted, but could read the smallest print. Besides dimness of sight he complained that the letters "jumped into one another" when he tried to read. His vision on February 11th, 1880, was $\frac{20}{70}$ with each eye when his myopic astigmatism of 2 D. was corrected, and he read words of from 8 J. to 14 J. slowly. No colour defect. The discs were slightly pale and misty all over, the deep tissue being opaque and obscuring the lamina cribrosa, and the borders being nowhere quite clear; the central vessels were normal. Four months later (June 11th) vision had improved to $\frac{20}{40}$ partly, and words of 6 J. with each eye; the fields were normal for white, but the red field was too small (no scotoma); no colour-blindness, but he sometimes hesitated between green and blue.

Rapid failure of sight with slight papillitis and pains in the limbs; later, incomplete atrophy of discs and colour-blindness with symptoms of disseminated sclerosis. By SEYMOUR J. SHARKEY, M.D., and E. NETTLESHIP.

FRED. J. H—, has been under care at intervals since November, 1878, sometimes on the medical side, sometimes

in the eye-department. The first notes refer to his eyes in November, 1878, when he was twenty-two years old. The left eye was defective from old corneal opacity. The right had, he said, been failing for a fortnight, and with it he could read only 18 J. and not 200 Sn. at 20 ; and he saw better in a dull light ; the disc was hazy and slightly œdematous, and there were white lines along the vessels, but no gross papillitis. The pupils were too large, but acted freely to light.

Some months previously he had had "rheumatism" in the left hand and right leg (from knee to ankle) ; there was, however, no swelling and very little pain. He was laid up by this attack for four months, and afterwards suffered from "swimming in the head." He was a carman, and attributed the symptoms to getting wet in his occupation. He had been married two years when the symptoms began.

He had had measles and hooping-cough, not scarlet fever, and when a baby had "water on the brain." Denied all history of venereal disease. He was one of seven children, all living. His mother died of bronchitis ; his father was living.

He only attended twice, and was not seen again for more than a year (January 1st, 1880), when he was admitted into the eye ward. The sight of the right eye had become a little worse (20 J. badly), and the disc had passed into grey atrophy, with atrophic cupping, exposure of the lamina cribrosa, and some diminution of the vessels ; the left disc, seen through the nebulous cornea, seemed of a better colour. The visual fields were of full size, and there was no scotoma (perimetric examination). Well-marked red-green blindness (confusion of green with grey, and red with brown). Still preferred dull light, "bright light dazzles me." Knee reflex normal, perhaps diminished on left side. Had sometimes lately had a "numbness from the armpit down the side and leg," with "a tight feeling" round that part of the body and leg.

He was in for a month, and had daily a subcutaneous injection of strychnia, increased from one sixtieth to one twelfth of a grain without benefit. He was forbidden to smoke.

He has lately (spring of 1883) been under Dr. Sharkey's care on the medical side, with characteristic symptoms of disseminated sclerosis.

Dr. Hughlings Jackson, after remarking on the great excellence of the paper, spoke of the complexity of tabes dorsalis. The symptoms were most various; joint-affections, gastric crises, several very different morbid affections of the eyes, bladder symptoms, &c. Of the so-called typical symptoms, one or more might be absent; ataxy was often absent; with ataxy the knee-jerks might be present. In one case, of seventeen years' duration, the Argyll Robertson symptom was not found. Some of the so-called typical symptoms were discovered in other diseases. The commonest pupillary condition was seen in some cases of general paresis. The knee-jerks were absent in many different morbid conditions, one of the most important of them being diphtherial paralysis, a disease which, superficially regarded, had sometimes a great likeness to locomotor ataxy. There were degrees of some, at least, of the symptoms; no ataxy, degrees of ataxy, and, so to speak, a degree beyond ataxy, and inability to walk at all. If there were not degrees of pupillary conditions, there were various such conditions. The width of the symptomatology was exceedingly different in different cases. There might be the Argyll Robertson phenomenon with no other definite nervous symptoms, and when this was so, the nature of the case, beyond perhaps the vague diagnosis of nerve-degeneration, could not be concluded upon. A case of tabes without ataxy might present a far wider symptomatology than one with ataxy. Whilst admitting the frequency of eye-symptoms with disease of the spinal cord, he knew of none from lesion

of it, excepting when that lesion was in the cilio-spinal region, as, for example, in a section of half the cord from a stab with a knife (contraction of the pupil on the same side, and narrowing of the ocular aperture). To statistics of optic atrophy, in its relation to other tabetic symptoms, he could offer nothing definite. The Argyll Robertson condition was often found in cases of optic atrophy (tabetic), not only when sight was slightly impaired, but when there was bare perception of light; in one case he had found it when the loss of sight was absolute. The pupils enlarged when the patient "made believe" to look at the clouds, and contracted when he "made believe" to look at his fingers held near him. On the other hand, with considerable impairment of sight, the pupils might remain contractile to light. To illustrate the varying width of association of optic atrophy with other tabetic symptoms, he mentioned five cases: (1) atrophy of one optic nerve and then of the other (green appearing grey, and red reddish brown), pupils acting to light, gait good, knee-jerks present; in short, no other symptoms except lightning pains; (2) optic atrophy with the Argyll Robertson condition, and without pains; and for the rest (saying nothing, however, of colour perception, which was not tested) like the former case; (3) like 2, but with pains also; (4) optic atrophy (blind eight years), Argyll Robertson condition, gait good, no knee-jerks; (5) a much more rapid case; pains one year; blindness complete, except for bare perception of light, in six months; could only just stand (loss of sight, no doubt, contributing to this disability); no knee-jerks. Dr. Hughlings Jackson then spoke of cases of double optic neuritis, with absent knee-jerks. A woman had bare perception of light, reeling gait, no knee-jerks; after mercurial inunction and iodide of potassium she recovered, except that when last seen she had no return of the knee-jerks. A girl, seen with Mr. Bowman, had double optic neuritis, reeling gait, no knee-jerks then or afterwards; later, right hemiplegia and aphasia; there was no necropsy. A man, who died with tumour of the left

cerebral hemisphere, had aphasia and right hemiplegia; both knee-jerks were present at first, both lost later; no morbid changes were found in the spinal cord by Dr. James Anderson. He had also seen double optic neuritis with absent knee-jerks, and no, or at any rate, no other, localising symptoms. He had nothing to say as to the nature of any associations of optic neuritis with any morbid conditions answering to those of the knee-jerks. The knee-jerks and double optic neuritis were present in some cases of tumour of the cerebellum, in one case of a lateral lobe, in another of the middle lobe (necropsies). He then spoke of diphtherial paralysis. He had been correctly reported to have said that this disease was owing to a morbid affection of the sympathetic system. What he ought to have said—all that he really held—was, that the ocular, the palatal, and the rarer circulatory symptoms (great slowness of pulse) of this disease, were morbid affections of parts supplied through ganglia of the sympathetic; he believed the spinal cord, as well as higher parts of the nervous system, to be morbidly affected in this disease. He had not seen a case of so-called diphtherial amaurosis in a stage when the paralysis of the ciliary muscle was complete; in some cases, where accommodation was only weak, he thought the pupils acted well to light, whilst action of them during accommodation was at least imperfect. In one case, the knee-jerks did not reappear until one year after all the symptoms of diphtherial paralysis had gone. Dr. Hughlings Jackson then went on to speak, by stating cases, of the very different, abnormal, intra-ocular motor conditions met with in tabes, or existing along with one or more of the so-called typical symptoms of this affection. (1) In one case there was sudden and complete loss of both pupillary activities, and of accommodation on but one side; gait good, no knee-jerks; there had been lightning pains four or five years. The other eye, carefully examined for the Argyll Robertson condition, was normal. (2) The same condition as in the last case, except that

the so-called good eye presented the Argyll Robertson symptom. This patient, a healthy-looking, intelligent sea-captain, had no other symptoms, mental or physical; hence the nature of his case could only be guessed at. (3) Argyll Robertson condition on but one side; ataxy, lightning pains, no knee-jerks. (4) A woman. Loss of action of one pupil to light and during accommodation, accommodation itself being absolutely perfect (examined by Mr. Couper); the pupil of the other eye was normal; that eye had all her life been slightly defective; no other symptoms of any sort were discoverable, except the most significant one of absent knee-jerks. (5) The same ocular condition on both sides (examined by Mr. Couper); ataxy, lightning-pains, no knee-jerks. (6) The same ocular conditions, except that accommodation was slightly weaker than usual at the patient's age (examined by Mr. Nettleship); gastric crises, ataxy, lightning pains, no knee-jerks. (7) Both pupils acting in no way, accommodation of each eye good; ophthalmoplegia externa. The only further tabetic symptom was absence of one knee-jerk and partial loss of the other. Dr. Hughlings Jackson expressed his belief that Dr. Gowers' able paper would help greatly in the precise and methodical investigation of tabes dorsalis.

Dr. Savage was not prepared to bring forward any statistics bearing on the question. He believed that the examination of the optic discs would lead to a considerable increase in our knowledge with regard to general paralysis of the insane. When, now a good many years ago, he had first begun to examine patients suffering from this disease with the ophthalmoscope, the results had been negative or unsatisfactory. In conjunction with Mr. Henry Power, he had made careful observations on a large number of these patients with the sphygmograph and the ophthalmoscope, but the results had been so purely negative, that it had not appeared worth while to make

any permanent record of them. In recent years, however, he had gradually come to appreciate certain changes which occurred in the optic discs, not of all, but of a considerable number of patients suffering from general paralysis. In one class of these patients, tabetic symptoms were prominent, and sometimes preceded the other symptoms of the disease by many years; for instance, in one case tabes had been in existence for ten years before the development of general paralysis led to the admission of the patient into Bethlem Hospital. In such cases as these, changes in the optic discs were commonly found; he had, however, only recently learnt that these changes were not confined to the patients who presented tabetic symptoms, but that they occurred also in another class of patients—those who presented symptoms of lateral sclerosis. In this connection, the question arose whether this lateral sclerosis was secondary to degeneration of the motor tracts in the brain. He had met with a few cases of general paralysis of the insane occurring in young single men of steady habits, where lateral sclerosis developed secondarily to intellectual symptoms, and in these cases there were changes in the optic discs. It was now generally recognised that "general paralysis" was a wide term, embracing a number of separable conditions; in making this subdivision, a careful attention to alterations in the optic discs and in the reflex phenomena would be of great assistance.

Dr. Walter Edmunds narrated a case of tabes dorsalis with temporary complete failure of vision.

The patient was a medical man, æt. 57. About nine years ago he had a sore on his finger from attending a midwifery case, this was followed by a roseolous rash, but no other symptoms of syphilis. He was well till three years ago, when his horse fell with him; he hurt his neck and had considerable pain in it for some time. He had another fall from his horse three months later, this

being due, he thinks, to loss of consciousness. Two years ago he had an attack of loss of vision, he was sitting at a table when suddenly he lost his sight. He had no vomiting, no headache, no giddiness. He was led to a house about a quarter of a mile away, and soon after getting there his sight suddenly returned. This was followed by headache. He has been subject to severe attacks of vomiting (gastric crises), and while in hospital he had one of these attacks. He has had lightning pains; his gait is awkward but not obviously ataxic. He can stand with feet together and eyes shut. There is no knee-jerk and no elbow-jerk. Vision good, no squint, has never seen double; pupils do not act to light; left pupil acts well to accommodation, right only very slightly. Optic discs normal; no albuminuria. The temporary loss of vision in this case resembles that which occurs occasionally in double optic neuritis; it was clearly not megrim.

Mr. Marcus Gunn.—I propose taking *seriatim* a few of the questions propounded in the memoranda, and seeing how far the statistics at my command tend to throw light upon them.

1. In what proportion of cases of atrophy of the optic nerves can the signs of locomotor ataxy be detected? This is eminently a question for an ophthalmic surgeon.

Of eighteen consecutive cases of optic atrophy admitted into Moorfields, there were well-marked signs of locomotor ataxy in two cases, while in three others there were present symptoms suspicious of this affection, but these latter were not uncomplicated cases of the disease.

2. When does atrophy of the optic nerve usually commence in the course of ataxy?

Altogether I have notes of nine cases of true locomotor ataxy. In seven of these there was optic-nerve atrophy, and in all it occurred in the first stage of the affection. There is, of course, an obvious source of error in a conclusion

such as this, drawn from the experience of an Eye hospital alone. Patients come there as long as the loss of sight is the most prominent symptom. Should the gait be decidedly affected first they will seek advice elsewhere, any subsequent failure of vision will then probably be explained to them as a part of the more general nerve disease, and as not requiring special treatment.

3. In what proportion of cases does tabetic atrophy affect one eye before and more than the other, and which eye is most frequently affected first?

Of the seven atrophic cases the right eye was affected most in two, the left eye in five cases. The *priority* of affection was only noted three times, and in all these instances the left eye was affected first.

4. Does the atrophy always progress to total blindness? Can any instances of considerable and permanent improvement of sight in tabetic atrophy be brought forward?

The cases were not sufficiently long under observation to afford any very definite answer to this question, but this much can be said, that there may be a temporary arrest, and even slight improvement. In one case the vision of one eye improved from ability to count fingers at five feet, to counting fingers at eight feet; in another case from counting fingers at one foot, to counting them at three feet; in a third the vision of one eye improved from ability to count fingers at 14″ to reading 16 J.

Two of the cases are of sufficient interest to merit a short separate description. I have to thank Mr. Couper for permission to bring them forward now.

Case 1. *Optic atrophy, with signs of locomotor ataxy without marked deterioration of vision.*—The patient is a well-built man, æt. 39, serjeant-major in the Hussars, who attended Moorfields April 20th, 1883. He says that recently he has had a difficulty in "dressing" his men on parade, and that he often cannot distinguish their features at a little distance so well as he used to do. In 1878 he had "heat apoplexy" in India.

Pupils react imperfectly to light, well with accommodation. Knee-jerk entirely absent on both sides. Suffers from shooting pains in the legs.

Vision: L. $\frac{20}{30}$ barely, not improved by glasses. Field good. R. $\frac{20}{30}$ barely and 1 J. Field good. Colour perception markedly defective in both eyes.

Ophthalmoscopic examination.—Media clear. Discs very pale; larger vessels of normal size. In the right there is a patch of double-contoured nerve-fibres.

On observation to-day (June 8) the vision of right has deteriorated to $\frac{20}{300}$, and that of left to $\frac{20}{40}$.

Case 2. *Well-marked symptoms of locomotor ataxy in an old case of Graves' disease, with other nerve lesions, in a woman, æt.* 49.—She gave a history of rheumatic fever at age of twenty-seven, followed six months later by small-pox and "intermittent fever." Soon afterwards, when aged twenty-eight, paralysis occurred in the left foot, then in the right hand, and she was shortly afterwards paralysed "all over" for two years and a half.

On August 27th, 1872, she attended Moorfields on account of prominence of the eyeballs. Diagnosis made "by ophthalmoscope, Graves' disease." Treated for some time without improvement, and then ceased attending.

On 12th July, 1879, she returned. Ordered iron internally. Since then the prominence of the eyes has gradually got less, and now there is very little. About October, 1877, she had a fit for the first time, evidently epileptic.

September 28th, 1880.—Pupils very small, inactive to light, but act on accommodation. Vision: R. $=\frac{20}{200}$, Hm. 0·5 D. L. $=\frac{20}{200}$, Hm. 0·5 D.

December 27th, 1881.—Homonymous diplopia dependent on paresis of the right external rectus. Pupils as before. Vision as before. Knee-jerk much impaired.

February 7th, 1882.—Slight ptosis of left upper eyelids. The movements of this eye are somewhat interfered with

in the directions upwards, downwards, and outwards. The left pupil is smaller than the right. Knee-jerk defective and much delayed. Pupils do not act at all to light, but well on accommodation. There is diplopia on looking upwards, downwards, and to the left. She states that this paralysis came on quite suddenly.

For the last four or five months she has had a feeling of tightness round the waist. Has been subject to severe pain in the legs ever since she was first paralysed. Stands well with heels and toes together and eyes closed. Her skin is getting much pigmented lately. The epileptic fits have been much milder and less frequent since taking bromide of potassium (ordered in February, 1881). At first she used to have two in a week. She has not had one now for the last four months.

June, 1883.—The paresis of left third and (?) sixth nerves recovered slowly. Has had one or two epileptic fits since last note. Knee-jerk quite absent. Does not complain of her eyesight, and she went away without her vision being tested. This case seems to be of importance as affording evidence in favour of the central localisation of Graves' disease.

Dr. Mahomed exhibited two patients with somewhat obscure nerve disorders, and read the following notes regarding them :

Case 1. L. W. R—, æt. 41, admitted into Guy's Hospital Feb. 7th, 1882, discharged April 13th, 1882. Has one aunt an idiot. An uncle whose sight failed in same way as his own at the age of 30, is supposed to have died afterwards of diabetes.

He joined the Horse Artillery in 1860 (aged 17) and went to Madras. Was laid up with fever for sixteen days, otherwise had good health. He has drunk hard, smoked immoderately while in India, and indulged in great sexual excesses. No history or evidence of syphilis. Has had gonorrhœa seven times. In 1873 had a severe attack

of jaundice. Came home from India in 1876. Health was perfectly good till June, 1882, when suddenly seized with severe pain in the head "as if something had given way." More or less constant pain in head ever since; during this period he has noticed great increase of urine and constant thirst, often drinking two quarts of water at night and five quarts of lemonade and ginger beer and soda water during the day. He passed his water every half hour. No excessive appetite. Has smoked immoderately. These symptoms, with more or less pain in the head and weakness, persisted, and six weeks before Christmas he became an out-patient at Charing Cross Hospital, where he says he was treated for diabetes. One week before Christmas, 1882, he came as an out-patient to Guy's Hospital under Mr. Higgens in consequence of the failure of his sight; he attended for about a month, his sight gradually becoming worse.

On admission.—He is quite blind with his right eye and can only see a little with his left. For some weeks he has had shooting pains in his arms and legs, a dull aching pain in his forehead, and a sharp pain in the back of his head. The pains seem to have got worse since his eyes became bad. He has had no fits. He has suffered singular sensations in the skin of his legs and forearm which prevented him sleeping, and were of an indescribable character, tearing small pieces out, crawlings, &c. He has them now but only occasionally. He is a pale, fairly well-nourished man, with light complexion and light brown and scanty hair; expression somewhat anxious; skin hot, dry, and white, rather harsh. Slight yellow tinge of conjunctivæ. Scars of old buboes in groin, a scar on the penis, and some small indurated glands felt in neck.

Nervous system.—Speech and hearing perfect. His movements are perfectly normal, no paralysis or loss of co-ordination. No loss of sensation. No muscular wasting. His reflex reactions are all normal, except that the knee-jerk is decidedly excessive. No ankle clonus. Oc-

casional pain in head, the forehead and back part being its seat. He suffers from what he calls "electric shocks" in his limbs, these are worse at night and often disturb his sleep.

He is completely blind with his right eye. Left eye has lost vision in about the inner third of the field (chart exhibited); his vision in the remainder of the field is indistinct, and he has no appreciation of *red* or *green*.

Ophthalmoscopic examination.—The discs are white, but are still traversed by vessels. In the right disc the veins are large and full, the arteries very small, scarcely visible. Left disc veins smaller but arteries not so small. The vessels appear broken here and there.

Electrical reactions.—The muscles of upper and lower extremities act normally to both currents. No qualitative changes.

Urine.—Very marked polyuria. From February 9th to 28th the quantity of urine varied from 178 to 96 oz. in the twenty-four hours, and averaged 120 oz. in the twenty-four hours. Its sp. gr. varied from 1004 to 1008 during the same period and averaged 1005.

During the month of March the condition of his urine improved, and varied from 140 to 49 oz. per diem, it averaged 86 oz. During the same period its specific gravity varied from 1004 to 1010, and averaged 1007.

During the first twelve days of April it still further improved, the quantity varying from 104 to 32 oz., and averaging 64 oz. while the specific gravity varied from 1008 to 1018, and averaged 1010.

His urine was tested daily, and was always found free from albumen and sugar, except on one occasion when a slight trace of albumen was detected. It was always found to be acid when its reaction was tested.

His appetite is good, he has some thirst. Since June he has occasionally suffered from nausea on an empty stomach and has sometimes been sick. Respiratory system normal.

Circulatory system.—Apex beat a little diffused, but not

displaced. No increase of cardiac dulness. Heart sounds rather distant. No bruit, second sound accentuated at base. Pulse firm, a little long and persistent; the sphygmograph shows that the arterial pressure is rather above the normal. A tracing taken on February 24th shows the vessels more lax and the heart's action more excited than one taken on April 4th.

On February 20th it is noted that he has lost his "electric shocks" and feels better in his health, but his sight is becoming worse.

March 9th.—Pupils normal, react to light. On the 14th his sight was thought to be improving a little.

April 5th.—The following note is made:—"Patient feels quite well. No pains in his limbs. Pupils act to accommodation but not to light. His sexual feelings are entirely in abeyance. He complains of pain, which changes about from part to part, *e.g.* from right arm to left, which is worse at night, though he is free from it to-day. He can still appreciate the fact that he is looking out of window, but can see nothing. Patellar reflex exaggerated, elbow reflex lessened. He complains of numbness in the thumb, index, and middle fingers, this apparently varies, being worse some days than others."

He left the hospital on April 13th to go to a convalescent home. His general health had improved, and he had gained a stone in weight, but his sight had steadily diminished. On leaving he was totally blind with right eye and could only appreciate light very slightly with his left.

Treatment produced no definite effect. From February 17th to March 8th he took Liq. Hyd. Perchlor. ʒj and Pot. Iodid. gr. x, ter die. From March 8th till March 20th he had ɱij of 1 per-cent. solution of nitro-glycerine, three times a day; during this time his sight appeared to improve slightly, but it increased his headache. From March 20th till he went out he took Liq. Sodæ Arseniat. ɱviij, Liq. Strychniæ ɱv, Inf. Gent. co. ʒj, ter die.

I am indebted to my clinical clerks, Mr. G. A. Johnson

and Mr. Walker, for their careful report of this case. It caused us much perplexity. The case was sent up to the medical ward as one of "smoker's atrophy" of the optic nerve and diabetes insipidus; but as this appeared an unusual and unexplained association, I was not inclined to accept the diagnosis. Locomotor ataxy immediately suggested itself, but this did not explain the polyuria, and the "knee-jerk" was rather increased than diminished; so for the time we put aside this diagnosis, till Mr. Walker worked it out more completely, bringing into prominence the neuralgic symptoms and lightning pains, the perception of blue and yellow, and loss of appreciation of red and green, together with the optic atrophy; but the diagnosis still remains uncertain. I am rather inclined to regard the case as one of cerebral tumour.

The polyuria is a remarkable symptom and well worthy of attention. Mr. Page recently brought a case before the Clinical Society, which he had also exhibited at the International Congress, in which a patient with tabes dorsalis and severe arthropathies, had also intermittent hæmaturia; Mr. Page stated that Raynaud had described attacks of nephritic colic in tabes; this case may lend some evidence in support of a renal neurosis in this disease; the more common urinary symptom is retention, owing to loss of vesical reflex, and this is illustrated in my second case.

June 3rd.—To-day he came to my house. He says that he is not nearly so well. His sight is still worse. He cannot tell where the window is. Soon after leaving hospital he woke up one night and found he had to some extent lost his speech; he stammered and hesitated, and could scarcely get a word out. This has since much improved. Sensation normal everywhere, but the thumb and two first fingers of the left hand feel very numb. He can pick up a pin and co-ordinate his movements perfectly.

Case 2.—Alfred P—, æt. 47. Admitted May 14th, 1883, for pain in the epigastrium. He pulls a fish-

barrow and has been a sailor. His mother died from consumption he thinks. Out of ten brothers and sisters only three are living. One brother died from consumption, causes of death in the other cases unknown.

He is said to have had ague when twelve years old, and another attack in 1870, when in America, followed by some dropsy in the legs. For several years he has suffered from rheumatic pains after exposure to wet or cold. Has always been a moderate drinker. No history of syphilis can be obtained. Has had gonorrhœa once.

For the last four or five years he has occasionally suffered more or less from indigestion, having an attack of pain after food. Three months ago, one morning, he had a sudden attack of sharp pain in the epigastrium which continued all day. He vomited all the food he took that day, since then the attacks of pain have become more frequent and occasionally he has been sick. Now he has pain after every meal.

On admission.—He is a restless, excitable, eccentric sort of man; of a tough and wiry build, fairly nourished, bright complexion, black hair turning to grey, grey eyes. Rather below the average height.

Alimentary system.—He has lost most of his teeth and those that remain are decayed. Appetite is not good. Occasionally feels sick, but has not vomited since admission. After taking food he complains of a sense of weight in the epigastrium; he has occasionally sharp pain here, which seems to have a distinct relation to food; it comes on directly after eating, it is relieved by vomiting, but often returns again. There is some epigastric pulsation, but no tumour. Liver dulness extends from seventh rib to two inches below margin of thorax. He has a double inguinal hernia, for which he wears a truss.

Circulatory and *respiratory* systems normal.

Urine of a dark yellow colour, sp. gr. 1022, contains a little albumen. No sugar.

Nervous system.—There is no loss of power in any of his limbs, and no want of co-ordination. He can stand

with his eyes shut, walk along a plank, turn quickly, button his shirt collar, and touch any given point of his face with one finger when his eyes are closed. He has the sensation of a band, about 4 in. broad, tied round the lower part of his thorax; he complains of a sense of numbness over the same area; there is also a sense of numbness of his back from the band round his body up to the level of the spine of the scapula, and along the extensor surfaces of both arms to the hands. There is numbness of the face and scalp, which is most marked on the forehead and below the mouth; his hands and feet are often numb. There is slight diminution of tactile sensation over the part of the back where numbness is complained of, and marked anæsthesia over the extensor surfaces of the arms, especially of the forearms. Sensation of the legs is not impaired.

During the last three months he has suffered from attacks of severe pain across the front of his head, his head sometimes feeling "as if it were going to burst." He has these attacks of pain three or four times a week, and they last from half an hour to three hours. For the last four months he has had shooting pains in both legs; he describes them as beginning in his toes and running up his legs; these pains frequently come on at night, after he has been in bed for a short time.

Reflexes.—Patellar reflex is quite absent on both sides. There is no ankle clonus. Good extensor muscle tap contraction, no contraction to tendon tap, or to Achilles tendon tap. In the soles of the feet, though sensation is perfect, tickling produces very little discomfort, and the feet are not withdrawn. There is slight cremasteric reflex on the left side. The epigastric reflex is normal.

Eyes.—There is paralysis of the external rectus of the right eye, with double vision for objects held on the right side of his head. The pupils are unequal, right rather larger than the left. They do not react to light, nor to accommodation for near objects. There is some intolerance of light and he prefers to wear a shade.

Ophthalmoscopic examination.—Right eye, outer part of disc white; deep central pit, with lamina cribrosa unduly visible. Well marked venous pulsation. Veins rather full; arteries small. Vision $\frac{6}{18}$. Left eye; disc white near outer part. Deep central pit. Well marked venous pulsation. Vision $\frac{6}{12}$. The field of vision is entire. Colour perception normal.

Since May 26th he has retention of urine, partly due to loss of bladder reflex, but also to loss of power in bladder. He has been taught to pass a soft catheter for himself, and has to use it three times a day.

He has been treated with Mist. Bismuthi Sed. and Liq. Strychniæ ♏ v three times a day, and his diet restricted to milk, with the addition latterly of fish and farinaceous food. Under this, his symptoms have greatly improved. His nervous symptoms have remained unaltered, with the exception, that yesterday (June 7th) he developed suddenly some paralysis of the extensors of his right forearm, which appears to have been due to pressure on his musculo-spiral nerve, caused by the position in which he usually sits, his arm resting on the back of a chair.

Dr. Mahomed went on to say: None of us can have listened to Dr. Gowers' most admirable paper without feeling a debt of gratitude to him for his careful and laborious work, for the able manner in which he has placed it before us, and for the great advances that have been made in diagnosis by means of his observations and those of his fellow workers. Although I cannot claim any special knowledge on the subject there are some points which present themselves to me which appear of importance, and I would claim the indulgence of the Society for a few minutes that I may bring them before its members. Dr. Gowers has dealt with the frequency of certain symptoms in certain diseases. I would direct your attention to the occurrence of these symptoms without the diseases. It is well that we should remember the continuity of structure and of function of the whole nervous system: in

dealing with its diseases we are dealing with disease of *one organ*, and of *one tissue* (though containing various elements). Like the vascular system it is everywhere continuous, and its diseases are in many cases similarly continuous, that is, are uniformly distributed throughout it. I do not mean, of course, that there are no *local* diseases, but I would point out that possibly many diseases which we regard as diseases of *parts*, are in reality diseases of the whole, and that any grave local disease may transmit its influence very widely. As in the cardio-vascular system the disturbances of function may be widely diffused, and may lead to structural change throughout the system, so I hold that in the nervous system disturbance of function may be widely diffused and lead to structural changes throughout it. Again, it is well known to every pathologist that similar tissues in the body, although disconnected both in position and function, sympathise with each other or are subject to the same diseases. I need only mention the way in which all the bones, or all the periosteum, in the body may become liable to the same form of new growth, no other tissue being affected. I have even seen a new growth confined to several of the unstriped muscles, spreading widely through them but affecting no other tissues. The disorders which pervade the skin and subcutaneous tissues are, of course, other examples.

We must remember then that, in watching the changes in the optic nerve, we are only watching changes in one of the most sensitive parts of the whole nervous system, and one which is fortunately exposed to our examination, and it may be true that similar changes are taking place, though perhaps in a less degree, in all the nerves of the body. And when we test the peculiarly delicate and complicated intra-ocular reflex mechanism, we are only trying to discover disorders in the most susceptible, which may exist in a lesser degree in all the coarser reflex actions. Indeed, in tabes, this would appear to be the case.

If we had a small artery similarly exposed and could watch its disordered states, its varying tensions, its muscular hypertrophy and the degenerations of its coats, we should learn a similar amount about the condition of the vascular system as a whole; indeed, to some slight extent we can do this in the optic disc itself, as Dr. Gowers has especially pointed out.

I believe that the tendency of the moment is to attach too much importance to individual symptoms and to attribute to them a greater pathognomonic significance than they deserve; in saying this I believe I am only following the opinions expressed, perhaps more reservedly, by Dr. Hughlings Jackson and Dr. Savage last night.

I would draw your attention to the fact that these individual symptoms derive their importance from *clinical* observations, which in the great majority of the cases have not been subjected to correction in the dead house; this at the best, is frail foundation.

I would especially ask how much we can rely on such symptoms as variations in knee-jerk, clonus, nystagmus, intra-ocular reflexes, optic atrophy and neuritis, as localising symptoms, or even symptoms of organic, as distinguished from functional, disease, if I may be pardoned for grouping these together, although not all immediately concerned in the subject of discussion. I would remark that I have frequently seen as, I suppose, we all have, increase and diminution of knee-jerk associated with purely physiological conditions; and I have seen it absent in persons who had not at the time, nor have since developed, any symptoms of nerve disease. I am told that it has been found to be absent in two per cent of all persons examined, though at the moment I cannot give a reference to these observations.

With regard to ankle-clonus it is known to be produced under physiological conditions, and I have now repeatedly seen it in cases of hysterical paraplegia which have completely recovered. Nystagmus, again, is not unfrequently a congenital and permanent phenomenon in persons of

weak nervous systems. I had a remarkable instance in the case of an out-patient who came under my care two years ago.

N. H—, æt. 16, a rather delicate-looking girl, with dark hair and eyes, came under observation March 28th, 1881. She had three brothers, two of them have nystagmus the eldest æt. 25, the youngest 7. Four sisters, all well. Father died in an asylum, was out of his mind for four months. Mother had a fit the previous Christmas, was paralysed on left side, was then recovering. Maternal grandmother was bed-ridden nine years with hemiplegia. Her father and mother were cousins. The patient has had nystagmus from birth and sometimes sees double. She came under treatment for large tonsils. She also suffered occasionally from severe frontal headache. The catamenia were regular. She knew her letters but could read but very little; she could not write; her eyes did not allow her to do so. She suffered from typical, and at the time severe nystagmus. It varied in severity. The optic discs were normal, as far as I could discover. I have no note of her intra-ocular reflexes, I fear the observation was not made. I sent her to see my colleague Mr. Higgens, who informed me that he had seen similar cases.

The intra-ocular reflexes are notoriously liable to variations with sympathetic disturbance and physiological conditions. I had a most remarkable example last week under my care in the hospital; unfortunately the case is not complete, though it is highly suggestive.

A. F—, a nervous, delicate-looking, spare and tall man, æt. 37, came to my out-patients on May 28th for headache, from which he had suffered on and off, more or less severely, since Christmas, and for renal colic from which he had suffered for two days. While waiting in the out-patient room he had a very severe attack of renal colic with agonising pain, retraction of right testicle and of abdominal wall on right side, accompanied by vomiting. I sent him into the ward, under my own care. On the fol-

lowing day, the renal colic having been relieved, I noticed that his pupils were unequal; both rather contracted, but right more so than left. The right did not react at all to light reflex, the left scarcely at all, a momentary contraction being immediately followed by dilatation to the previous extent. They both reacted to accommodation. Both eyes rather intolerant of light. We then found that his patellar reflex was absent. His superficial reflexes were normal.

These symptoms may have been due either to the morphia, which he had taken rather less than twenty-four hours before, or to some sympathetic disturbance; the inequality of the pupils suggested the latter. On June 2nd (four days later), the light-reflex was still sluggish, though present. The knee-jerk was still absent. The optic discs were normal. There were no symptoms of loss of co-ordination. He went out by his own desire and unknown to me, on the 6th, without opportunity being afforded for further observation.

Atrophy of the optic nerve is said by some authors to occur in association with various conditions of deteriorated health, perhaps as a local, perhaps as part of a general change. So also with neuritis, of which I had a case in a young woman of about sixteen a short time ago, who came for catamenial irregularity, debility, and some headache. I sent her to see Mr. Higgens, who reported that she had double optic neuritis, and that he had frequently seen it before in association with catamenial disorders. She remained under observation for some time and never developed any other symptoms of organic nerve disease.

These cases and remarks may be trite, but they suggest themselves to me in consequence of the increasing reliance that appears to be placed on these symptoms as evidence of organic or well-localised disease. On the other hand, it appears to me that they may be produced by conditions of nervous exhaustion, of sympathetic excitation, by reflex trophic disorders, and other remote

or diffused conditions. If this be so, it is clear that we should never pronounce a grave diagnosis on the evidence afforded by such symptoms as these alone. Indeed, the evidence brought before the Society last night shows how frequently some of these symptoms are found in other and more general diseases of the nervous system. They may mean functional disorder, they may mean very general structural changes, although no doubt it is true that they are most frequently observed in connection with localised disease. We want to know how frequently and under what circumstances they exist without it.

Mr. Nettleship said : Dr. Gowers has stated his belief that the optic nerve lesion in spinal diseases is probably not continuous with the changes in the cord. Apart from pathological proofs of this, to which Dr. Gowers has referred, I think we have good clinical evidence in favour of the belief. In the earlier periods of progressive optic atrophy in locomotor ataxy the disc is not merely pale : its edge is frequently rather softened owing to slight haze of the deeper layers, which just overlaps but scarcely extends beyond the choroidal rim ; and the area of the disc itself loses its transparency, assuming a nearly opaque appearance like blanc mange, or a dead white chalky aspect, by which in many cases the lamina cribrosa is concealed from view, although the opacity is always beneath the large vessels. Such appearances are not seen in atrophy following injury or other destructive lesions of the nerve far behind the globe.

These appearances, of an active, although slow, inflammatory process in the deeper parts of the papilla, are usually well marked before the patient makes much complaint of his sight. It must have happened to all of us to see patients who say that one eye is very bad but that there is nothing amiss with the other, and in whom we nevertheless find the well-known appearances of progressive atrophy in both discs ; and although in nearly all such

instances careful examination will show some contraction of the visual field in the "good eye" which has as yet escaped the patient's notice, we occasionally meet with cases where the visible changes at the disc precede any functional failure whatever.

Thus, Richard W—, æt. 46, had "pallor of both discs alike;" left eye failing two months, V. $\frac{20}{50}$, blindness for green, loss of upper and outer part of field; right eye V. $\frac{20}{20}$, colour perception and field normal. In others the earliest ophthalmoscopic changes seem to agree in time with the failure of visual acuteness; thus Mr. H—, æt. 40, had pronounced atrophy of his left with V. $\frac{20}{200}$ and 16 J., and colour-blindness; the right disc being normal, V. $\frac{20}{30}$ and 1 J., and colour vision full. Six months later the right vision had sunk to $\frac{20}{50}$ and 10 J. with red-green blindness and concentric contraction of the field, and the disc was now grey-white like its fellow.

If the disc atrophy were secondary to morbid changes beginning high up in the optic nerves, or in the optic tracts, we should often meet with amblyopia or even blindness, coming on some time, probably considering the slowness of the cases many weeks, before any changes were apparent in the fundus of the eye,* as we well know is true in some cases of retro-ocular neuritis. Such pre-atrophic amblyopia is, I believe, almost unknown in cases which afterwards pass on into progressive atrophy and blindness. In this connection the following case is instructive:—A railway inspector, an Irishman, fell or jumped from the foot-board of a train going at the rate of ten miles an hour; he was badly shaken and suffered for months from nervousness and inability to do any work. His symptoms were so threatening that for a time, whilst he was an in-patient under Dr. Sharkey's care, the onset of general paralysis was suspected, and the more so perhaps as progressive failure of sight in both eyes had come

* In cases of destructive injury to the optic nerve from fracture involving the optic canal, from two to four weeks usually elapse before any pallor of the disc can be detected. *See* 'St. Thomas's Hospital Reports,' xi, 113.

on since the accident. The eye symptoms were characteristic of tobacco amblyopia, there were no ophthalmoscopic changes, and the man was a smoker. He was made to give up smoking, and as his general symptoms improved, he recovered perfect sight. His was tobacco amblyopia excited, as this disease often is, by some cause of general enfeeblement.

Dr. Gowers asks, "In what proportion of cases of atrophy of the optic nerves can the signs of locomotor ataxy be detected?" And further on, "How does the optic atrophy of tabes differ from that sometimes met with in other forms of chronic spinal cord disease." In order to contribute towards a reply to these questions I have tabulated all the cases of progressive atrophy of which I have useful notes, and from the tables have made the subjoined abstract. From these tables all cases were excluded of post-papillitic atrophy, atrophy after hæmorrhage, tobacco amblyopia, and atrophy, whether rapid or slow, due to local causes in the orbit or eye.

TABLE *showing the frequency of symptoms of chronic spinal-cord disease in cases of progressive optic atrophy.*

Total number of cases of optic atrophy	76

Simple loss of knee reflex is counted as diagnostic of tabes in cases of double optic atrophy. If the knee reflex was present or its absence not noted, any two other common symptoms are accepted, *e.g.* pains, ataxic gait, numbness, impotence or satyriasis, incontinence or retention of urine, reflex iridoplegia. Mere reflex iridoplegia with optic atrophy is not accepted as proof of tabes (see Group *d*). In the great majority several symptoms were present.

GROUP *a*.—With undoubted locomotor ataxy . .	38
GROUP *b*.—With mixed symptoms of spinal and cerebral disease, usually most like general paralysis of the insane . .	11
GROUP *c*.—With other forms of chronic spinal-cord disease not ataxy, and without signs of brain disease	9

GROUP *d*.—With reflex iridoplegia, but no other symptoms of spinal or cerebral disease 3
GROUP *e*.—Other cases 15

Of this total, 38, or just one half, were therefore the subjects of locomotor ataxy, whilst 20 others (Groups *b* and *c*) were suffering from some other form of chronic spinal disease or from mixed cerebral and spinal symptoms, leaving only 18 (about 25 per cent. of the whole) in which it cannot be affirmed that disease of the spinal cord existed. Amongst this residuum of 18 cases there are, however, 3 with the typical Argyll Robertson pupil, a symptom which alone is very suspicious, although it cannot at present be taken as pathognomonic, of threatened tabes dorsalis. Further, of the 15 cases in Group *e* not more than 7 can be said to be quite uncomplicated, for the remaining 8 all presented some one symptom pointing to the probability of spinal disease, such as pains, weakness of legs, impotence, &c.

I have for long been struck with the great frequency of symptoms of nervous disease in progressive optic atrophy, but I confess I was not prepared to find that out of 76 cases only 8 (scarcely more than 1 in 10) would be free from ascertainable complications. It is however probable that the proportion with nervous complications is somewhat larger in my series than it would be if I were not attached to a general hospital; for at St. Thomas's Hospital my medical colleagues have sent me several cases of spinal disease with optic atrophy which would not otherwise have come to my notice; on the other hand, some of these very cases had previously been under ophthalmic treatment elsewhere.

Among the 7 apparently uncomplicated cases, one (Wm. B—) was a man whose sight failed rapidly after a severe fist-blow between his eyes fifteen years before I saw him, and had remained stationary, at perception of light only, ever since.

One (John H—) was a lad of eighteen in whom there

was nothing whatever to account for the failure; he is now about twenty-two, intelligent, in perfect health, and doing well as an organist in a blind school.

Another (Mrs. R—) was a woman whose sight failed simultaneously and equally in the two eyes during her fifth pregnancy, at the age of thirty-three, in 1879, and has very slowly got a little worse during the last four years; her vision is now about $\frac{1}{10}$ with normal fields, normal colour perception, and pupils acting well both to light and accommodation; very careful inquiries and examinations have failed to throw any light on the cause of the failure. Another case (John L—, æt. 39) is complicated with double ophthalmoplegia, externa and interna, but whether due to sclerosis of centres or to a cerebral tumour has not been decided. The other three cases occurred in men between the ages of thirty and forty, none of whom remained under care long enough to make their cases of much value (Jas. R—, John L—, George K—).

In respect to Group *b*, it will be convenient here to reply, so far as the cases permit, to the question whether *when failure of sight occurs in insanity, is the case more likely to be complicated with spinal symptoms?*

Among the eleven cases in Group *b* two showed decided symptoms, and three others threatenings, of general paralysis of the insane; all of these had reflex iridoplegia and other spinal symptoms. Another man (L—) lately become insane, also has "spinal" pupils and no knee reflex; his cousin is insane. Two patients were senile old men, apparently becoming demented, without spinal symptoms. One man (H—) died out of his mind, æt. 53, after a few days' illness; he had long before had fits; he had had an attack of nervous derangement; his pupils were "spinal." Another man, æt. 33 (M—), became extremely depressed, tremulous, and nervous, with nystagmus. One patient was a lad, æt. 18, subject to recurring attacks of insanity. In several patients in this group the failure of sight was exactly equal, and began at the same time, in the two eyes, and it is worth noting that these very patients

showed no symptoms of tabes or general paralysis. In the cases resembling general paralysis the eye failure followed the same rule as in ataxy, the atrophy in one eye leading, and keeping in advance of, that in its fellow. I ought to add that, in the examination of this group, I have often had the great advantage of the opinion of Dr. G. H. Savage, or of gentlemen who had worked under him as assistants in Bethlem Hospital.

In Group *c* are placed six cases, diagnosed by one or other of my medical colleagues as either certainly, or probably, disseminated sclerosis. Three of these are given in detail above (pp. 224-6); two others may here be mentioned.

Horace N—, æt. 28. Left eye failed "suddenly" four years ago, and has since improved a little; V. = 14 J.; no colour-blindness. Right eye reads 2 J.; no colour-blindness; discs pale, especially on y. s. side, the left showing the more change. For two years has had weakness of legs and tremors of hands; knee reflex is much exaggerated, and there is nystagmus, especially in extreme movements to his right. Pupils act to light.

George S—, æt. 40, with quick nystagmus, twitchings of legs, and giddiness, with normal pupillary movements, has moderate amblyopia with pallor and slight haze of disc in one eye only; field normal and no colour defect.

In a sixth case the diagnosis is much more doubtful, since the atrophy of nerves has gone on to complete blindness, and all the symptoms have been present for many years (Alfred P—, æt. 42).

We may say, I think, from the cases given here and at pp. 224-6, that if the optic nerves suffer in disseminated sclerosis they seldom suffer profoundly, and often recover partially; and that the process is more acute and, to the ophthalmoscope, more inflammatory than in tabetic atrophy.

The remaining cases in my Group *c* call for no special remark. Two of them were considered by Dr. Gowers to be lateral, or mixed lateral and posterior sclerosis, (Mr. C—, Edward S—).

As to the frequency of precise symmetry of the optic nerve disease in locomotor ataxy;—I believe the disease almost always begins in one eye before the other, usually some months before, and the eye which leads always keeps the lead, *i.e.* always remains the worse and becomes blind before its fellow. In thirty-seven cases where the point is noted, I find only four where the failure of sight was observed at the same time in each eye, and in these the disease progressed equally in each eye as long as they were under notice. Of the remainder, the right eye failed first in eighteen, the left in fifteen cases.

The second eye nearly always begins to fail before the sight of the first has become very bad; but in one instance (C—) the right had been absolutely blind (no p. l.) for six months before the man (a letter carrier) began to notice any defect of his left eye. William R—, æt. 56, now very ataxic, lost sight in his right eye entirely in twelve months, the left remaining, in his opinion, perfect for nearly eighteen months after the right had become blind (see also the case of Mr. S—, mentioned below). In these two cases the blindness came on rather quickly in each eye.

Commonly the interval noticed by the patient between the commencement of symptoms in the two eyes is from two to about eight months; but the interval was three years or more in the case of Henry R—, suffering from well-marked ataxy; two years and a half in William R—, also very ataxic; and at least four years in Mary B—, whose ataxic symptoms dated back five years. An interval of from one to two years is noted in several.

Mode of failure.—One of the most striking points is the exactly symmetrical invasion of the fields of vision often noticed, although the loss of field is usually, as would be expected from what has been said, of larger extent in one than the other. This symmetry must depend upon an equally precise similarity in the seat and distribution of the morbid process in each optic nerve, a point perhaps of some interest in the pathology of the major disease. In

at least three cases the loss has been of the lower and inner part of each field; in two other cases this part of the field, on the other hand, was the last to remain. More commonly the outer and lower part is the last to remain. When a large sector is lost up to the centre of the field we may sometimes think there is a central scotoma until a careful examination has been made. After going carefully over all my cases I cannot find a single instance of proved central scotoma in spinal optic atrophy; in two cases where the fields were not carefully taken the symptoms seemed to point to its existence, but it so happens that in neither of these were the spinal symptoms well-marked, and I only saw the patients once (John B—, Mr. H—). In one case, which I mention for what it is worth, homonymous and very complete lateral hemianopia came on, quite suddenly, in an ataxic man at about the time when he began to be troubled in walking; but whether it was anything more than a coincidence I do not pretend to decide (John T—, æt. 53).

Marked colour-blindness is almost invariable in tabetic atrophy, but there are some real or apparent exceptions, cases with more or less loss of acuteness, often great contraction of the field, and yet little or no defect in colour perception. At the other end of the scale are cases with almost normal acuteness, with the field contracted as in the former group, and pronounced lowering of perception for red and green. It is possible that, after all, these apparent anomalies are largely accounted for by an originally high standard of colour vision in the first group and a low standard in the second. At any rate, though the explanation is not yet forthcoming I must modify a statement made three years ago "that I had never seen atrophy of the optic nerves in locomotor ataxy without colour-blindness."* I should say now, that in locomotor ataxy there may, in rare cases, be considerable lowering of visual sharpness with only the slightest possible lowering of colour-perception. The following

* 'Brit. Med. Journ.,' 1880, ii, 779.

cases in point are all from Group *a* (atrophy with symptoms of locomotor ataxy):

Mr. S—, æt. 65, had had no perception of light with his right for some months; the left was just beginning to fail, and though he could still manage to read 1 J., there was considerable contraction of the upper part of the field, and the disc was very pale all over. He passed the wool test perfectly. He had been suffering from tabes for five years, and was unable to walk, but had not much pain. Reflex iridoplegia and small pupils.

In the case of William T—, æt. 43, there was the most extreme contraction of the fields that I have ever seen in progressive atrophy, with nearly normal acuteness, $\frac{20}{30}$ and 1 J., and not the slightest colour-blindness.

Daniel R—, æt. 51, with V. $\frac{20}{70}$ and 6 J. in the better eye, and loss of the upper-inner part of the field, showed only very slight defect for green and red.

James A—, æt. 39, with V. $\frac{20}{50}$ and 10 J. in the better eye, and a much contracted field, has only very slight lowering of green perception.

John G— (an Irishman), æt. 37, had been under Dr. Hughlings Jackson's care at the London Hospital for locomotor ataxy for about eighteen months before he came to me at St. Thomas's Hospital for his eyes (June, 1881). He had been able to read well till a month before, but his sight had been failing a little for longer than that. Vision of right 16 J. at 8″ and $\frac{15}{100}$ partly; left 19 J. at 8″ and less than $\frac{15}{200}$; signs of past iritis in the left; visual fields entirely absent, except a narrow sector at the nasal side of each, the symmetry of which was exact. Discs grey, arteries rather small. With the wools he showed a very slightly defective perception of the pale green, but no defect whatever for any other colours. Pupils 4 mm., motionless both to light and accommodation. Probably had syphilis sixteen years ago. Is very ataxic. He became quite blind in about a month after I saw him. It is worth noting that this man's father had been under me for symmetrical amblyopia, with very well-

marked central scotomata of the form met with in tobacco amblyopia, and no contraction of fields. It was a case of about two years' standing, the patient being seventy years old, the discs were pale, but his sight improved from $\frac{20}{100}$ and 14 J. to $\frac{20}{70}$ and 10 J. in eighteen months, when he cut down his allowance of tobacco. His pupils acted well to light and on efforts at accommodation, and he had on symptoms of ataxy.

On the other hand, Daniel D—, æt. 45, has very marked red-green blindness, though with the better eye he sees $\frac{20}{50}$ and 1 J. well; he has lost most of his field, retaining only the downward and outer part in each eye. He is drowsy, his speech slow and tremulous, and he has tremors of the head.

James T. D—, æt. 42, with V. $\frac{20}{20}$ slowly and 1 J. in the better eye, and a field much and uniformly contracted, has fairly well-marked red-green blindness.

William B—, æt. 32, with incomplete reflex iridoplegia and threatened general paralysis, saw $\frac{20}{30}$ and 1 J. with his better eye, the field being much contracted; there was well-marked, though not complete, red-green blindness.

In the case of Mr. W—, æt. 37, ataxic and liable to delusions, vision in the better eye was $\frac{12}{12}$ badly and words of 1 J. The field was much contracted, and there was already slight, but decided, lowering of perception of green and red, yellow and blue being recognised perfectly.

Mr. Brudenell Carter said that the papers read had added much to the wealth of the material placed before the Society on the previous evening, but they served also to show the great complexity of the problems involved. It had not been his intention to take part in the discussion, but he would venture to say that all practitioners would be grateful to those who could give a clue to the value of the symptoms in the class of cases under consideration. It often happened that a patient with some impairment of walking power, and some optic atrophy, desired a prognosis; but it was most difficult to know

whether the case was one of progressive spinal disease or not. He had attached some importance to the knee-reflex as a guide in this matter, but had not always found it trustworthy. Again, some cases came with optic atrophy and no other symptoms, as in the case of a sergeant who had been nineteen years in the army and had enjoyed perfect health, but for some months had been rapidly losing his sight, which improved markedly under hypodermic injection of strychnine. Would that man become ataxic? The occurrence of such cases made one grateful for any hint which could be utilised in daily work.

Dr. GOWERS said that the very interesting papers were valuable contributions to our knowledge of the subject, but the discussion had been so slightly controversial that it left him but little to reply to. Mr. Gunn's case of Graves's disease was one of great interest and complexity; it was also interesting as an instance of complete loss of the reflex action of the pupils to light preceding loss of the knee-jerk. Dr. Mahomed's general principles were unquestionably of great importance, and should be borne in view; but he (Dr. Gowers) had often been impressed with the opposite fact, viz. the remarkable way in which the nervous system suffers in part. Nothing, for instance, is more remarkable than that the posterior columns of the cord should be absolutely destroyed, whilst other parts exhibit no visible change. A definite diagnosis was hardly possible in Dr. Mahomed's case of optic atrophy with polyuria; the only symptom of tabes present in that case was pain. The occurrence of severe headache at the time of onset of the loss of sight suggested the possibility of the case being one of tumour just above the chiasma, a situation in which he remembered a tumour occurring that ran a very chronic course, and one in which polyuria was, he thought, also present. At the same time that would not explain the pains. It certainly was very unusual to have pains in the limbs from tabes of so long duration, and not to have the knee-jerk reduced.

Dr. Mahomed had alluded to the occasional absence of knee-jerk under normal conditions. Berger found it so absent in about 2 per cent.; and he (Dr. Gowers) mentioned several cases in which he could not detect it in his paper read before the Royal Medical and Chirurgical Society in 1879. But from that time to this he had never found it absent in health. There was a great source of error in regard to the failure to elicit the knee-jerk; it very often could not be obtained because the patient did not perfectly relax the flexors of the knee-joint, and if these muscles were not relaxed the jerk did not occur. This was particularly liable to arise in hysterical subjects, and hence the statement that the jerk was absent in hysterical paraplegia. This condition could be detected by placing the fingers on the flexor tendons or obviated by causing the patient to sit upon the edge of a table. There was also an error to guard against in testing the pupillary reaction to light, owing to the patient involuntarily accommodating to the light. He had been told of a case where the pupils did not react to artificial light, but did to daylight; probably this was due to the patient accommodating to the former. Dr. Sharkey's case of disseminated sclerosis was very interesting. Sight was sometimes lost, not because the optic nerve was affected, but because of sclerosis in the optic tract. Mr. Nettleship's statistics were of great value, being the most carefully compiled facts upon the question that had yet been made, and it was to be hoped that other ophthalmic surgeons would be stimulated to a similar research. Such facts bearing on the relation of optic atrophy to spinal diseases, and to spinal symptoms, should go far to settle the question. In the case read by Mr. Nettleship, he thought that the homonymous hemiopia was certainly accidental, and not connected with the morbid process of ataxy.

X. AFFECTIONS OF MUSCULAR AND NERVOUS SYSTEMS.

1. *On ocular movements, with vertigo, produced by pressure on a diseased ear.*

By J. Hughlings Jackson, M.D., F.R.S.

In the last volume of our 'Transactions' reference is given to some observations I made on a patient during his paroxysm of auditory vertigo; during it the eyes jerked to the right, and at the same time external objects seemed to him to move in that direction. There is in that volume a communication by Donders on the subject of movements of the eyes in relation to apparent movements of objects. In the case I have to relate to-night no paroxysm was seen, but pressure on part of a diseased ear was invariably followed by ocular movements; the patient had at the same time apparent displacement of objects; perhaps we may say that slight paroxysms of auditory vertigo were in this case artificially produced, as they seem to be in some people, by syringing the ear. Schwalbach has recorded an essentially similar case; a summary of his observations by Clarence Blake is reproduced in 'Brain,' for April, 1879. I have to thank Mr. Laidlaw Purves and Mr. Couper for great help in the investigation of the case. I now proceed to detail.

A woman, æt. 49, consulted me November 23rd, 1882, for giddiness and irregular walking; these symptoms had existed for about three months. She had had disease of the right ear since childhood; at no time had she had

noise in it, but had had some in the left ear, which was, except for this, apparently healthy. She had had severe "bilious" attacks, clearly, by her account, paroxysms of auditory vertigo; she had also occasionally very slight and transient paroxysms of giddiness without sickness. There was also a chronic condition, since the so-called bilious attacks she often walked as if drunk, and was therefore ashamed to walk in the street. She would sometimes, when washing the right ear, stumble to the left. I could not ascertain that her general vigour had lessened before her illness began. She had missed one menstrual period.

She told me that pressing on a certain part of the ear (the tragus) made her giddy, and produced a disagreeable feeling in her head, "something coming over the brain." When I pressed on the tragus I saw her eyes move from side to side, and she told me that objects "went" from right to left; she did not see them coming back again. She also felt a falling towards the left side. On stopping the pressure the eyes were again still. I could make them move whenever I liked. The two movements, one to the left, which was the first, and one back, were not so different in rate as in the case, already alluded to, of the man who had a paroxysm of auditory vertigo. In the woman's case, however, the eyes passed more slowly from right to left than they came back—that is, they came back to the right more jerkingly. The apparent movement of objects to the left coincided with the slower movement to the left, not with the jerk to the right. I sent the patient to Mr. Laidlaw Purves, who confirmed my observation as to the ocular movements. He reported also on the state of the ear. The right meatus was full of pus. The membrana tympani could not be seen. The watch could not be heard on the right side. The tuning-fork was heard one third less when placed on the mastoid; pressure on the pus, as well as on the tragus, caused the ocular movements. Mr. Purves reported, later, "There is a swelling on the upper wall of the meatus, caused almost certainly by some osseous affection from

old otorrhœa of childhood. The ear is, however, so sensitive to interference, and the meatus is so closed, that the actual condition is as yet not determinable."

December 4th.—She was better; ocular movements were less easily producible. Mr. Purves gathered from the patient that she now saw the objects coming back, and he thought the jerk back (to the right) was slower. I gathered from her that she did not see the objects coming back. I regret this discrepancy of opinion.

About the beginning of January, 1883, Mr. Couper saw the patient and reported as follows (January 4th): "I find the jerks of the eyes most readily evoked by pressing back the tragus, as though to shut it down on the external auditory meatus, with at the same time an upward pressure against the root of the zygoma. The jerks are always horizontal towards her right, and equal as well as simultaneous in both eyes. Generally the sharp jerk to the right is preceded by a slower preliminary movement to her left. This is always so when her eyes happen to be directed either straight in front, or horizontally to her left. The jerks of largest excursion are preceded by proportionally large movements to the left. Movements of widest excursion are obtained when she fixes a distant object above the level of her head. The jerks are shorter when she looks horizontally in front of her, and shortest of all when the axes converge as she looks down at a near object below the level of her head, and, in this last position, some rotatory movements (*i.e.* jerks of upper edge of vertical meridian plane to her right on an antero-posterior axis) are observed. In no other conditions are rotatory jerks present, and when these are well marked the lateral jerks are proportionately absent. No spasmodic or other abnormal movements of her pupils are detected. Both pupils react fairly well both to light and during accommodation. There is $H\frac{1}{48}$ manifest with normal acuteness. For reading I gave $+\frac{1}{16}$ spherical."

I saw her again to-day, January 11. She felt much better, and now she said that to produce the giddiness and

movement of external objects she had to put the finger in the ear, "the nerve seemed to have gone further in." I produced no movement by pressing as before, nor by pressing as Mr. Couper did; but she, by putting her finger in her ear, produced movements of the kind already described but of very slight excursion, about three millimetres. There was a little apparent displacement of objects. Mr. Purves reported to me that the discharge was almost gone. The remedies used were syringing the ear, and quinine in doses of from two to three grains.

It is needless with regard to this case to do more than mention the now old, but very important, experiments of Flourens, on the semi-circular canals. The case, like that recorded by Schwalbach illustrates very well Cyon's experiments on the semicircular canals of rabbits. A summary of Cyon's researches is given by Ferrier in the second number of 'Brain,' and by Croom Robertson in 'Mind,' October, 1878. The researches of Crum-Brown, Mach, and Goltz, are very important in the interpretation of cases of giddiness with ear disease. The observations and conclusions of Menière are well known. I suppose that in my patient the pressure made on the tragus was transmitted by the chain of bones to the contents of the semicircular canals; probably the contents of one or more of these canals was abnormal also. To reach the ocular muscles there must have been propagation of changes from the canals through some part of the central nervous system, cerebrum or cerebellum, or possibly through both. The case is valuable as giving further evidence, if further evidence be needed, that ear disease is one cause of, or factor in producing, vertigo. If such procedures provoke vertigo in a deaf patient who has quasi-spontaneous attacks of giddiness we may convince him that ear disease is the chief factor in his ailment, and in difficult cases they may make the diagnosis more clear to us. They will, I suppose, give valuable evidence, as to the progress of cases. This case supports the opinion of Dr. James Taylor, of Chester, expressed as regards the parox-

ysms of auditory vertigo in the man whose case I recorded ('Brain,') July, 1879, in so far as that the apparent displacement of objects is concomitant with the slower movement and tends to disprove the opinion I then stated, which was, that the displacement of objects is in the direction of the jerks. In the woman's case the displacement was not in the direction of the jerks to the right, but in that of the slower movements to the left.

I may be permitted to mention some recent important observations. Dr. James, of Boston, has pointed out ('American Journal of Otology') that deaf mutes are much less liable to vertigo on rapid rotation than the healthy: many deaf mutes are not made giddy at all by this means. He thinks from his observations that they have a comparative immunity from sea-sickness. The horrible depression occurring in paroxysms of auditory vertigo is likened by some of the patients to that in sea-sickness. It has occurred to me that disturbances of these canals may be in part to blame for sea-sickness.

(*January* 11*th*, 1883.)

P.S.—I saw her last February 27th. She looked well, felt perfectly well, and had gone back to her professional duties.

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2. *A case of paralysis of some of the ocular muscles, &c., after diphtheria; with remarks on the pathology of the affection.*

By Arthur H. Benson (Dublin).

Mary F—, æt. 11 years, was sent to me on Dec. 22nd, 1882, in consequence of a rather sudden failure of sight which had manifested itself two weeks previously.

History.—Her mother stated that she had been subject

to attacks of tonsillitis for some years past, that about seven weeks previously she had had another attack, for which she was treated by the physician who sent her to me, and who diagnosed a diphtheritic inflammation of the tonsils and pharynx.

In a month she was quite well, the attack not having been a very severe one. In about a week after her recovery was complete *i.e.* five weeks from the onset of the disease, the child noticed that the "sight was scattered." This symptom progressed gradually till she was unable to read or see objects distinctly at any distance.

She never had pain in the eyes, but complained of supra-orbital pain varying in degree, and made worse by using the eyes. There was no specific history obtainable.

Physical examination.—The irides were unaffected. Both pupils were of medium normal size, and acted freely, both to light and to convergence.

The *ciliary muscles* were both paralysed though not completely. R., V. = $\frac{6}{36}$, and 14 J., from 17 cm. to 22 cm. with + 2·75 D., V. = $\frac{6}{6}$ with + 9 D., V. = 1 J. from 15 cm. to 28 cm. L., V. = $\frac{6}{36}$, 13 J. from 15 to 26 cm. with + 2·75 D., V. = $\frac{6}{6}$ with + 9 D., V. = 1 J. from 15 cm. to 28 cm. Ac. therefore = 3 D., about.

The other ocular muscles seemed normal, the fundus of the eye presented no ophthalmoscopic changes.

There was *paresis of the soft palate and fauces* and consequent dysphagia. Fluids regurgitated through the nose and rendered feeding very difficult. This symptom only manifested itself two days before she came to me.

The *voice* had the peculiar nasal timbre to which Donders* drew attention many years ago.

She was *deaf* to some extent also, but not markedly; her mother called it stupidity at first. The deafness was most marked when the palatal paresis was at its worst. She had no cold in the head, or local inflammation or obstruction in the external meatus to account for the deaf-

* New Sydenham Society's translation, 1864, p. 601.

ness. The paresis of the palate and the regurgitation lasted about a fortnight.

January 1st, 1883.—There was slight drooping of both upper eyelids; she could raise them fairly well with an effort, but they drooped again, giving her a sleepy look. Her mother noticed that they drooped more at home when she was sitting quiet and not minding. V. = $\frac{6}{24}$. (The ptosis lasted about one week.)

On January 3rd she returned in a fright, saying that she was much worse, that she now "saw everything double." There was paresis of both external recti muscles, with slight convergent strabismus, causing the diplopia. The excursion outwards of each eye was diminished, that of the right being most restricted. The internal motion of each eye was not restricted, it was not therefore a defect of associated movement, but of the action of the recti externi muscles themselves. When one eye was covered, and an object at 30 cm. distance fixed by the other, the covered eye deviated inwards about 7 mm. There was homonymous diplopia and the images were erect. R., V. = $\frac{6}{18}$; L., V. = $\frac{6}{18}$. With both eyes together everything was so indistinct that she could not see $\frac{6}{60}$. This diplopia lasted only four days, but there was weakness of the external recti for some time longer.

15th.—She now complains of feeling her knees very weak; she could not run, and in walking she stumbled over every slight obstruction; the ground felt "queer" under her, and she could not properly feel where she was treading. She had, moreover, a feeling as of pins and needles in her feet. The upper extremities were unaffected. No tendon-reflex was obtainable. The weakness in the knees, &c., lasted for about three weeks.

28th.— $\left.\begin{matrix}\text{R.,}\\ \text{L.,}\end{matrix}\right\}$ V. = $\frac{6}{6}$, 1 J. from 10 cm. to 50 cm., A. = 13 D. about. All paretic symptoms had disappeared, and she considered herself as well as ever she had been, but the patellar tendon-reflex was still absent. The urine

had, unfortunately, not been tested during the earlier stages of the affection; but when tested later on no albumen could be discovered in it.

To recapitulate:

The *primary throat affection* lasted	4 weeks.
The *ciliary muscles* were affected in the	5th week,
and continued so for about	7 weeks.
The *soft palate* was affected in the	6th week,
and remained so for about	2 weeks.
The *hearing* was affected in the	6th week,
and remained so for about	1 week.
The *levatores palpebrarum* were affected in the	9th week,
and continued so for about	1 week.
The *recti externi* muscles were affected in the	9th week
(two days after the levatores palpebrarum),	
and remained so for about	3 weeks.
The *convergent strabismus* and *diplopia* were	
present during the	10th week,
and lasted for about	4 days.
The weakness of the lower extremities began	
in the	10th week,
and lasted for about	3 weeks.
Numbness and tingling in the feet began in	
the	10th week,
and lasted for about the same time as the	
weakness, *i.e.*	3 weeks.

The above history is by no means an uncommon one, but it is none the less remarkable for that.

Here we have an affection of the throat, primarily a local affection according to Oertel*, which, after producing considerable constitutional disturbance, lasting for four weeks, apparently passed off. After an interval of a week, in which the patient seemed perfectly convalescent, there followed with considerable rapidity, a series of isolated, bilateral paralyses, both of motor and sensory nerves, from which recovery took place with varying rapidity.

The patient, all through, experienced no inconvenience

* Ziemssen, 'Cyclop.,' 1875, vol. i, p. 581.

or ill health further than that which resulted from inability to use the paralysed muscle, or the perverted sense.

No other paralyses are so various in their distribution, so evanescent, and so harmless as these, except, perhaps, hysterical paralyses, concerning which so much has been written and so little is known. There is, however, this essential difference between them, that whereas the possibility of imposture is ever present to the physician in attendance upon a hysterical patient, no such idea need be entertained regarding the sufferer from the sequelæ of diphtheria.

The following statements may, I think, be regarded as proved :

1. The degree of subsequent paralysis is not proportionate to the severity of the primary affection.*†

2. The ciliary muscle is one of the muscles most frequently affected.‡

3. Paralysis of it is seldom complete, but is usually bilateral.

4. Paralysis of the abducens oculi is as rare as that of the ciliary muscle is frequent (Pagenstecher).†

5. Usually the pupil is unaffected and its motions unimpaired.†

6. The muscles of the palate are often, though not always, the first to suffer.

7. Paralysis of the palate and of other parts may occur after wound diphtheria (Oertel).§

8. Throat diphtheria may produce paralysis elsewhere without implication of the palate muscles (Scheby-Buch).

Paralysis of the ciliary muscle without alteration of the condition of the iris seems to be by far the most frequent affection of the intrinsic muscles of the eye. Donders,‖

* Althaus, ' Diseases of the Nervous System,' 1877.

† ' Schweigger's Handbuch,' p. 73.

‡ ' Nagel's Jahresberich,' 1871, Manz. Of 90 cases recorded by Maingault in 1860, 39 had amblyopia, and 10 had strabismus.

§ Ziemssen, ' Cyclop.,' vol. i, p. 621.

‖ Op. cit.

however, noticed mydriasis in most of his cases; and Abercrombie, in a paper on the subject read before the International Medical Congress in 1881, stated that of eighteen cases recorded in his paper, all had mydriasis. "The pupils," he says, "are always *dilated* and *sluggish*" (the italics are his).

On the other hand, Manz,* Jacobson, Classen, Setweitzer, Maingault, and most modern observers, regard affections of the pupil as relatively rare; whilst Pagenstecher* never, and Scheby-Buch only once, saw a case in which the action of the iris was affected.

In endeavouring to solve the pathological problems involved in the case, the inquiry naturally divides itself under two heads.

I. What is the seat of the lesion?

II. What is the nature of the lesion?

I. Where, then, is the seat of the lesion causing this remarkable, isolated, bilateral paralysis of accommodation?

Dr. Hughlings Jackson, in his address on "Ophthalmology in its Relation to General Medicine,"† seems to be in favour of the idea that the sympathetic ganglia are at fault:—"The peculiarity," he says, "of diphtherial amaurosis is that the paralysis is of parts supplied through a ganglion of the sympathetic chain; there is not paralysis, or paresis of the third nerve, but of parts of it which are supplied through the lenticular ganglion. In other regions we see the same thing. The defective articulation is owing to paralysis of muscles of the palate supplied through Meckel's and the otic ganglia. The very slow pulse which, as Jenner and Greenhow have pointed out, is found in some cases, may be explained by affection of the cervical ganglia of the sympathetic." He saw, however, the difficulty of a sympathetic theory, and added: "I grant fully that I cannot show that a similar state of things applies to the rest of diphtherial paralysis."

Dr. Jackson in the above quotation appears to regard

* Nagel, 1871, p. 197.

† 'Brit. Med. Jour.,' 1877, vol. i, p. 577.

disease of the lenticular ganglion as a probable and sufficient explanation of the ciliary paralysis. But even if disease of this ganglion were found—and Dr. Jackson does not say that such ever has been found after diphtheria—even if it were found it would not explain the clinical facts; for, as we have seen, the occurrence of oculo-pupillary changes, invariable, as I believe, in lesions of the lenticular ganglion, is, in diphtherial paralysis relatively rare.

The validity of Mr. Hutchinson's theory of the causation of ophthalmoplegia interna depends entirely upon the invariable association of oculo-pupillary as well as ciliary paralysis, with disease of the lenticular ganglion.

The sympathetic theory—at least as far as this particular muscle is concerned—seems untenable; moreover, other signs of sympathetic implication, vaso-motor disturbances, &c., are not recorded as occurring with anything like constancy. The researches of Hensen and Voelcker have shown that a centre exists in the hinder part of the floor of the third ventricle which governs the ciliary muscles, and that this centre is independent of that governing the circular fibres of the iris, and also of that which presides over the radiating fibres of the iris.

These three centres presiding over these bilaterally co-ordinated movements, may each be affected separately. If the first alone is affected we have the accommodation alone affected, as in diphtherial paralysis.*

Is it not, therefore, reasonable to refer the paralysis of accommodation to lesion of this cerebral centre?

Dr. Jackson has further elaborated his sympathetic theory so as to account for the occasional occurrence of deafness, by implication, he thinks, of the otic ganglion producing paralysis of the tensor tympani muscle. Such an affection may occur, but it seems much more probable that the deafness is the result of paresis of the palatal muscles than that the nervous supply of the tensor tym-

* For an admirable *résumé* of the Physiology of Ophthalmoplegia see paper by Dr. Allen Sturge. 'Ophth. Soc. Transac.,' vol. i, p. 165.

pani is interfered with. This supposition is further supported by the fact observed in my case, that the deafness was only present whilst the palatal paresis was at its worst. The symmetry of the affection of the eye and throat is also against the sympathetic theory.

The occurrence of paralysis of the muscles involved in other single co-ordinated actions would still further help to localise the lesion in the higher centres in the brain Such we find do occur. Thus in my case there was paresis of both levatores palpebrarum alone, the other muscles supplied by the third nerve being unaffected.

Ferrier* found that "at the base of the first frontal, and extending partly into the second frontal convolution in the brain of the monkey, there is an area, irritation of which causes elevation of the eyelids." . . . "There are clinical cases on record tending to show that there must be a distinct centre for the levator palpebræ superioris, inasmuch as paralysis may occur, limited to this muscle without affecting the other muscles supplied by the third nerve; an occurrence difficult to explain by peripheral affection of this nerve. Some such cases have been observed in connection with disease of the cortex, and attempts have been made, but not, I think, successfully, or in accordance with experimental lesions, to localise this centre in the angular gyrus.† If such a centre really exists, I should be inclined to look for it in the region of which I am speaking."

Here, then, we have another reason for regarding the affection as central not peripheral, as cerebral rather than sympathetic.

Simultaneous paralysis of both external recti muscles, again, would point to lesion of the nuclei of the sixth nerves in the upper part of the floor of the fourth ventricle, rather than to peripheral affection of the nerves themselves.

* Gulstonian Lectures on the Localisation of Cerebral Disease. 'British Med. Journ.,' April 6th, 1878, p. 474.

† Landonzy, "Blépharoptose Cérébrale;" 'Archiv. Gén. de Méd.,' August, 1877.

Leber,* however, has endeavoured to explain bilateral paralysis (or paresis) of the sixth nerves by supposing that the increased intra-cranial pressure (in cerebral disease) causes these nerve-trunks to be pressed against the internal carotid arteries where they lie in contact with them in their passage through the cavernous sinuses. The pulsation of the arteries is supposed to cause a pressure-atrophy of the nerves, or at any rate to interfere with their functions. This does not seem a possible explanation in my case.

Absence of tendon-reflex has been noticed by Rumpf† in a girl, æt. 9, suffering from other ataxic symptoms which came on after diphtheria. The paralytic symptoms commenced by loss of acommodation and paresis of the internal recti muscles. Abercrombie‡ mentioned that there was absence of patellar tendon-reflex in all the cases, nine in number, tested by him. In my case the knee phenomena were on several occasions tested without any reflex being found, the difficulty, however, of testing this point in children is considerable.

If subsequent experience confirms the observations above mentioned, and it is found that absence of tendon-reflex is the rule, it would still further strengthen the idea of a central lesion.

As regards the situation of the lesion various theories have been suggested. The following structures have each been regarded by some as the primary seat of the affection.

1. *The muscles themselves.*—Against this may be urged the fact that:

(1) The ciliary muscles in these cases act to eserine.

(2) The atrophic changes in the muscles suggest a secondary, not a primary, metamorphosis.

(3) The frequent implication of sensory nerves.

2. *The peripheral nerves.*— Voelckers's explanation, adopted by Förster, is rendered improbable by the fact

* 'Arch. f. Ophth.,' xiv, Abth. 2, p. 333, and 'Gräfe-Saemisch,' vi, p. 60.

† Nagel, 1877, p. 381. ‡ Loc. cit.

that the seat of the primary affection seems to have little influence in determining the nature and position of the subsequent paralysis, as is shown by the sequelæ of wound diphtheria.*

3. *The nerve-trunks.*—This is disproved by the occurrence of paralysis of a single muscle supplied by the third nerve, as, for example, the levator palpebræ, without implication of the other muscles supplied by the same nerve. Nor is it the short root of the ciliary ganglion that is affected, for the pupil continues to act to light.

4. *The sympathetic system.*—This theory seems incompatible with the fact of—

(1) The isolated paralysis of the ciliary muscle without implication of the pupil.

(2) The absence of vaso-motor disturbances or other signs of sympathetic affection.

(3) The occurrence of paralysis of the extremities.

(4) The presence of sensory-nerve phenomena.

5. *The brain and spinal cord.*—All the results of modern research seem to point to the great nervous centres, the brain and spinal cord, as the seat of the lesion, whatever it may be, which produces the paralytic sequelæ of diphtheria.

II. As to the *nature of the lesion,* but little can be said with certainty.

Donders considered that the "altered blood crasis" produced a secondary morbid process in the central organ.†

Bretonneau‡ looked upon the paralysis as a secondary symptom of the morbid poisoning to be compared with the secondary phenomena of syphilis.

Trousseau regarded albuminuria as the cause of the paralysis.

Althaus‡ thought the paralysis was produced by "migrating neuritis."

* Ziemssen, 'Cyclop.,' loc. cit.

† Donders, op. cit., p. 608.

‡ Op. cit., p. 180.

It is with difficulty and by slow degrees that pathological facts are beginning to take the place of mere hypotheses in the explanation of diphtherial paralysis, but it may not be without profit to analyse the results of post-mortem observations as far as they have at present gone.

Diphtherial paralysis is so rarely fatal that the opportunities for microscopical examination of the affected structures are very limited, and it is only in the most severe cases that any opportunity occurs. Even in these the results of examination had until lately been purely negative.

Dr. Greenhow* in 1860 wrote on the morbid anatomy of diphtheria, and drew attention to the frequency with which extravasations of blood, minute or larger, were found in the various tissues of the body, and quoted cases recorded by Dr. Bristowe, Dr. Gull, and Mr. Simon, where hæmorrhages of various sizes were found freely dispersed throughout the brain and spinal cord as well as in the skin. In most, if not all the cases, albumen was present in the urine, and the post-mortem examinations showed extensive disease of the kidneys.

Buhl† found in one case that the spinal nerves were thickened at their roots, that hæmorrhages had taken place, and that the sheaths of the nerves, and here and there, also, the strips of connective tissue running between the bundles of nerve-fibres and the ganglion-cells of the sensitive roots, were filled with lymphoid nuclei and cells. "These cells," he thinks, "either disappear unnoticed, or are followed by a perceptible thickening of the connective tissue, which by contracting squeezes the nerves. The nerve symptoms appear when the constricting effect of the thickening of the connective tissue begins, they persist while it lasts, and finally disappear when the thickening diminishes."‡

* Greenhow, 'On Diphtheria,' 1860, chap. xi, p. 235.

† Nagel, 1871.

‡ Ziemssen, vol. i, p. 657.

Oertel* found capillary extravasations of blood in the brain, the spinal cord and the nerves issuing from it. There was also a considerable increase of nuclei in the grey matter which, to the naked eye, appeared free from hæmorrhages and softened spots. These changes were most noticeable in the anterior horns. He further noticed a large quantity of the smallest variety of micrococci both in the blood and in the hæmorrhages of the membranes of the brain and spinal cord, and in the tissues. There was also extreme atrophy and fatty degeneration of the muscles in one case. According to the results of his investigations, Oertel says ;:—"We shall then have to explain the functional disturbances in the different muscles, from simple paresis of individual groups up to complete paralysis of the same, or of the whole muscular system, by the alterations first in the muscle itself, then in the peripheral nervous system, and finally in the central organs, brain, and spinal cord."

Schweitzer† found lymph cells, dilated vessels, thrombi, and larger and smaller hæmorrhagic foci in the connective tissue surrounding the nervous structures of the brain, sympathetic ganglia, and the vagus.

Abercrombie‡ examined the spinal cord and medulla oblongata in seven cases. The only pathological changes he was able to detect were in the grey matter of the anterior cornua, and they consisted in a swollen condition of the large motor cells. The margins of these were very ill defined, and the processes had, in most instances, entirely disappeared. In some places the cells appeared shrunken rather than swollen. No blood extravasations were seen. Albumen is mentioned as occurring in four out of his eighteen cases, but particulars are not given to enable us to say in which cases the albumen was found.

The examination of the medulla oblongata yielded negative results, with one single exception, where some of

* 'Graefe u. Saemisch Handbuch,' Band vii, p. 176.

† Nagel, 1871.

‡ Op. cit.

the outermost cells of the vagus nucleus were found to be rounded and more or less completely deprived of their processes. M. Déjérine had found similar changes in three cases.

Dr. Percy Kidd* lately read a paper before the Royal Medical and Chirurgical Society in which he described the appearance of the cord in a case he had examined. The changes were similar to those described by Vulpian, Déjérine, and Abercrombie, but he also found a numerical atrophy of the motor nerve-cells in the anterior cornua. The disease might be described, he thought, as a "poliomyelitis anterior."

In the discussion to which Dr. Kidd's paper gave rise, the pertinent question was asked, "What is the pathology of those cases which recover?" Physiologists have not determined what amount of regeneration is possible in the nervous structures of the brain and cord. But it seems hardly possible that recovery could take place with the rapidity and completeness with which it does in most cases, if there were anything like the degree of degeneration of the ganglionic cells which has been described as found in the post-mortem examinations.

It seems to me that the explanation may possibly be found in the occurrence, even in the milder forms of the disease, of minute capillary hæmorrhages, such as have been observed post mortem. If this could be proved it would explain all the symptoms; such small hæmorrhages might easily be absorbed in the time during which the paralysis usually lasts, leaving the nerve structures uninjured. Larger or smaller hæmorrhages would account for the varying duration and degree of the paralysis, as also for the hemiplegic and other grave forms which are met with at times.

Further research of a more systematic and complete nature is required before anything can, with certainty, be stated regarding the pathology of this strange affection; but cases of fugitive paralysis of various degrees have

* 'Brit. Med. Journ.,' Jan. 13th, 1883, p. 57.

from time to time been recorded in the 'Transactions'* of this Society, as elsewhere, the only explanation offered being hæmorrhage.

There are known affections which seem to predispose to hæmorrhage into various organs, as for instance, the occurrence of hæmatoma auris amongst lunatics. May not diphtheria predispose to hæmorrhages into the brain and spinal cord, the position, size, and frequency of the extravasations determining the position, duration, and curability of the resulting paralysis?

I have to thank Dr. W. A. FitzGerald for the kind assistance he has given me in collecting the materials for this paper.

(*March 8th*, 1883.)

3. *Case of paralysis of the third nerve, in a child, with cerebral symptoms, leaving ophthalmoplegia interna.*

By David B. Lees, M.D.

(Communicated by John Abercrombie, M.D.)

Alice A—, æt. 6½, was brought to the Hospital for Sick Children, Great Ormond Street, November 10th, 1881, on account of a squint in the right eye and shaking of the left arm and leg. The squint had been observed for three months, and for the same length of time the right pupil had been larger than the left. Two weeks after the onset of the eye symptoms the left arm and leg began to shake, and this shaking had gradually increased. The girl had three fits five years ago, but there was no fit at the commencement of this illness. There had been slight headache over the right eye for the last fortnight only. In other respects the health was good.

* Lang and FitzGerald, 'Ophth. Soc. Transac.,' vol. ii, p. 231.

On examination it was found that there was marked paralysis of the right third nerve. The right pupil was quite twice as large as the left, and did not respond to light, dilate further when shaded, or contract with accommodation. Divergent strabismus was present. The eye could be moved outwards, upwards, or inwards and slightly upwards, but it could not be brought into the inner canthus, and it could not be moved downwards. The eyelid drooped slightly; this drooping was said by the mother to be more obvious when the child was tired. Both optic discs were found to be normal. No affection of the fifth or seventh nerves could be discovered.

The shaking movements of the left upper limb consisted partly in slight forwards and backwards movements of the whole limb in a vertical plane, and partly of alternate short flexions and extensions of the wrist, without any pronation or supination. They were rhythmical and uniform; they occurred while the limb was not used.

Similar but less decided movements affected the lower limb. The mother stated that they did not quite cease even in sleep, but that they were worse when the child was excited. She also said that the girl did not use her left arm much, but allowed it to hang by her side, and that she complained of numbness in it. There was, however, no distinct paralysis of the limb.

The child had previously suffered from hooping cough, measles, and varicella, but no history of congenital syphilis could be obtained. It was thought right to try the effect of iodide of potassium. Four-grain doses three times daily were ordered. Improvement followed at once. After six weeks (December 19th, 1881) it was noted that the shaking of the left limbs was much less, in fact hardly to be seen. The mother stated that it was more obvious after excitement. The right eye was more moveable, some downward motion could be effected, and the cornea could be brought nearly to the inner canthus. The pupil remained enlarged and motionless.

She continued to take the iodide regularly. On the 4th May, 1882 (after six months' treatment), the dose was raised to five grains four times a day, and this quantity she has continued to take till the present time. On this date a baby brother, aged three months, was brought for inspection. He was found to have snuffled since his birth, and to have had some difficulty in sucking; some coryza still existed. The mother said that he had had some rash on his buttocks, but none was then present. There were no other symptoms of congenital syphilis.

On the 20th November, 1882, after treatment for twelve months, it was noted that no shaking of the limbs could be detected. The mother, however, stated that some occurred when the child was awakened from sleep. The squint had entirely disappeared, leaving only a little weakness of the (right) internal rectus. The cornea could be brought well into the inner canthus, but it went a little upwards at the same time. The pupil, however, remained dilated and motionless; it measured 6 millimetres in diameter, whilst the left measured only $2\frac{1}{2}$.* The left pupil responded to light, and contracted slightly with accommodation, but the right remained absolutely motionless. The power of accommodation in the right eye seemed lost, and if the left were closed she could read only large capitals. On testing her with Snellen's types, it was found that the left eye could read that headed D. $=0{\cdot}5$ at a distance of about a foot, but that the right

* Mr. Nettleship has given me the following more exact report on the state of the eye early in October, 1882:—Right eye: Lateral movements are of full extent, but some weakness of the internal rectus is still present, for convergence power is very defective, the eye usually wandering outwards when she is told to look at a near object; no ptosis; the eye looks rather more prominent than the left. Pupil = 6 mm. when looking at a distant object; no associated action, but there is some reflex action, a slight increase in diameter taking place when both eyes are well shaded and *slow* contraction to former size when uncovered V. $\frac{15}{70}$ and 3 Sn.; with + 3·5 D. = $\frac{15}{30}$; with + 6 D. reads 0·5 Sn. Left eye:—Pupil when looking at distant object = 2·5 mm., associated action slight, reflex action brisk. V. $\frac{15}{40}$, and reads 0·5 Sn. easily. Ophthalmoscopic appearances natural in each eye.

could read nothing less than D.=4. By placing, however, a convex lens of 6 dioptrics in front of the right eye she could read D.=0·5.

This condition remains unaltered. She still has a dilated and absolutely motionless pupil, and a loss of the power of accommodation, a group of symptoms to which Mr. Hutchinson has given the name of ophthalmoplegia interna, and which he believes to be associated with disease of the lenticular ganglion.

The present case seems rather to support the theory advanced by Dr. Gowers, that the group of symptoms depends upon a cerebral lesion, the site of which is probably near the nucleus of the third nerve, below the aqueduct of Sylvius.

(*January* 11*th*, 1883.)

P.S.—In the discussion on this case Mr. Hutchinson pointed out that in the condition which he had described under the title of ophthalmoplegia interna the pupil was not dilated; it was motionless, but of normal size. He was therefore of opinion that this case did not quite conform to that type.

It was afterwards found that the instillation of eserine into the girl's eye caused contraction of the pupil to the normal size. It still remained motionless when shaded or illuminated, and during an effort to accommodate.

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4. *Case of paralysis of the left sixth nerve, with choreiform movements affecting mainly the right side of the face.*

By David B. Lees, M.D.

(Communicated by John Abercrombie, M.D.)

William W—, æt. 11½ years, was brought to Great Ormond Street Dec. 2nd 1882, on account of a convergent

squint in the left eye, and occasional twitching contortions of the face.

The mother stated that the squint had been present since he was three years old, and had first appeared whilst he was suffering from hooping-cough. On examination it was found that the left eye could not be brought to the outer side of the median position. There was no other affection of the ocular movements, except that there seemed some difficulty in getting the right cornea into the inner canthus, this could only be effected with effort and the eye soon moved outwards again. The pupils were equal, each of a diameter of three and a half millimetres for distant vision; they both acted normally in all respects. The optic discs were normal, except that each was partly surrounded by a crescent.

Every now and then, at irregular intervals, the boy's face was curiously contorted by a kind of spasmodic contraction of the facial muscles. The right side of the face was mainly affected, but the left eyebrow was also implicated. The left cheek appeared to be unaffected. The mother said that sometimes the right arm also was involved, but her account was not very definite, and this did not occur under my observation.

The boy had been at school in the country for four years. He stated that his sight was not good and that when at school he had to sit facing the light. The facial movements had been present, he thought, about two years; they were worse when his attention was directed to them. For the last few months he had suffered from headache, otherwise his health was good, though he was pale. He had never had rheumatism, and the heart was normal. There was no paralysis of the left seventh nerve, the facial muscles responding to Faradism quite as well on the left side as on the right.

It seems probable that the choreiform movements and the headache are both dependent upon abnormal refraction, for both eyes are hypermetropic and astigmatic, and for this condition spectacles were ordered. One sees

occasionally semi-involuntary facial movements of this kind in anæmic nervous children, but these are usually symmetrically bilateral.

(*January 11th*, 1883).

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5. *Congenital ptosis with peculiar associated movements of the affected lid.*

By R. Marcus Gunn.

Florence J—, æt. 15, attended the North-West London Hospital on June 1st, 1883. Complains of drooping of the left eyelid, and of the fact that, while eating, and occasionally while speaking, the lid is rapidly jerked upwards. This movement of the lid is quite involuntary, indeed, she is unconscious of its occurrence except when she observes herself in the mirror; but it is a source of annoyance to her friends.

Family history.—Her parents are both rather delicate; her mother is very nervous and excitable. They were not related before marriage. There is no family history of mental peculiarity or marked deformity. Her paternal grandfather and all his family have had a slight want of symmetry in the upper eyelids. Her father and his sister were alone examined; in both there is a very slight extra fulness of the right upper lid, but no real drooping; there is no inequality of the pupils and no associated movement of the eyelids except those normally occurring with upward and downward movements of the globe. The only other member of the patient's family is a boy aged nine, who is in very delicate health (bronchitis), but his lids are quite natural. There have been no deaths.

The patient is a well-grown, intelligent girl, not at all nervous in manner. Her health has never been very strong, and she is now somewhat anæmic. Her complexion is rather dark, her hair dark brown, and her

irides grey. When about five weeks old it was noticed that when she was sucking the breast her left upper eyelid "nearly went up out of sight." There was rather more drooping of the lid then than now when at rest. Since then, especially during the last two or three years, there has been a gradual improvement in this condition, the lid not jerking upwards so much as formerly.

There is a slight want of symmetry between the two sides of the face, more evident on smiling, when a deep dimple appears on the left cheek about an inch outside the angle of the mouth, while on the right side the the dimple is not so deep, and is placed high on the cheek, about an inch from the right ala nasi. When she looks straight forwards, the left upper lid droops so as to cover the upper one fourth of the pupil. The fold of this lid does not run parallel to the free border as in the normal condition, but meets it at an acute angle near the inner canthus, thus giving an appearance of obliquity. This pupil is smaller than the right, its reaction to light is good both alone and consentaneously, and it contracts well with accommodation. The upper lid follows the upward and downward movements of the globe, though it is not thus raised quite to the same extent as on the right side. On shutting her eyes gently the left upper lid does not close quite so perfectly as the right. On being asked to raise her eyelids while looking straight forwards, she endeavoured to do so with the aid of the occipito-frontalis, and only succeeded in raising the left to a very slight extent. When asked to do so without wrinkling her brows, she succeeds on the right side but the left remains motionless.*

On lateral movement of her jaw to the right side (left external pterygoid) the left upper lid is raised quickly and powerfully, and this position of the lid is maintained as long as the jaw is kept drawn to the right. The associated action of the lid is best marked when the jaw is moved while she is looking downwards. The mouth

* The aunt could do this well. The father was not tried.

can be opened gently without any lid movement, but on the lower jaw being projected forward there is immediately a jerking of the left eyelid upward. The same movement of the lid occurs in a slight degree occasionally while speaking, especially, as the patient has herself observed (while looking in the mirror), in pronouncing words containing an "s" or "x." If the jaw be moved to the right while the lids are kept closed, the contraction of the left levator palpebræ superioris can still be determined from the skin being thrown into folds over its insertion. During all such movements of the lid the pupil remains quite stationary, and there is no observable movement of the eye itself. The right upper lid is quite normal and all excursions of both globes are well performed.

R., V. $=\frac{20}{20}$, no Hm. Reads 1 Sn. with a near point of three inches.

L., V. $=\frac{20}{20}$, Hm. 0·5 D. Reads 1 Sn. with a near point of two and a half inches.

Ophthalmoscopic examination.—Deep physiological cup with marked venous pulsation in each. By direct examination fundus of right eye seen with + 1·5 D. and left eye with + 2 D.

Size of pupils.

In medium light	R.	pupil measures		5·5	m.m	in diam.
	L.	,,	,,	4	,,	,,
40 minutes after a drop of homatropine (gr. 4 and ℥j)	R.	,,	,,	7·5	,,	,,
	L.	,,	,,	6·5	,,	,,
Under eserine (gr. 2 and ℥j)	R.	,,	,,	2	,,	,,
	L.	,,	,,	1·5	,,	,,

There is well-marked dilatation of each pupil on stimulation of the corresponding sympathetic nerve in the neck by means of the faradaic brush.

No one-sided perspiration or blushing has ever been observed.

(*July 6th*, 1883).

A Committee was appointed to examine Mr. Gunn's Case of "Congenital ptosis," and reported as follows :—

The case thus presents :

(1) Contraction of the levator palpebræ in association with the external pterygoid.

(2) Diminished action of the levator (tonic and active) in its association with other muscles supplied by the third nerve.

(3) Imperfect relaxation of the levator when the eyelids are gently closed.

(4) Diminished size of the pupil.

The latter symptom raises the question whether there is any defective action of the sympathetic, to which the slight ptosis may also be due. But there is no other indication of sympathetic defect. The pupil dilates, when the skin is stimulated, as readily as does that of the other eye. The smaller size of the pupil can be equally well explained by an increase in the tonic contraction of the sphincter, and the lower position of the eyelid, by lessened innervation from the third nerve.

The prominent fact of the case is that the levator contracts when the external pterygoid is put in action, while the latter does not contract when the levator is put in action. There must be an abnormal connection between the central mechanism for the external pterygoid and the levator muscle. The simplest explanation is that the levator is innervated both from the nucleus of the third nerve and from the external pterygoid portion of the nucleus of the fifth nerve. Since the innervation from the third nerve nucleus is defective, we may assume that some of the fibres of the levator palpebræ portion of the third nerve arise, not from the nucleus of the third, but from that of the fifth. Thus we can understand both the action of the levator in association with the external pterygoid, and the lower position of the eyelid at rest, and on movement of the eye. The fact that the levator does not relax perfectly when the eyelids are closed, may

perhaps be accounted for by its partial innervation from the fifth nucleus.

It is conceivable that the smaller size of the pupil, presumably due to increased tone of the sphincter, is due to the abnormal connection of the levator fibres.

The levator receives less, the sphincter iridis more than the normal influence from the third nucleus. If the third nerve nucleus contains the normal number of nerve-cells it is evident that some, which should act on the levator, are not connected with it, and it may be the influence of these cells, which determines, in some way, the smaller size of the pupil.

The difference observed in the movement of the two sides of the face appears to be due simply to a difference in the connection of the muscles and the skin.

W. R. Gowers.
Stephen Mackenzie.
William Lang.
John Abercrombie.

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6. *A case of muscular asthenopia in a child.*

By W. A. Brailey, M.D.

William C. C—, æt. 7, a thin, pallid child with large and sluggish pupils, was brought to Guy's Hospital, having suffered for several years from severe headaches, especially affecting the frontal region. These have not been constant, but have always come on after very short use of the eyes in reading. Even after five minutes so employed his head aches, and his eyes pain and water. The headaches are worse towards evening. His visual acuity is normal. There is no hypermetropia. The eyes diverge when the object is approached to a distance of

five inches. The internal recti overcome a prism of 8° and the external one of 1° only.

Atropine used thrice daily gives no relief to the headache, nor do spherical glasses, but prisms of 2° bases inwards give immediate and lasting relief. His mother has, however, been instructed that they should be worn only for short periods at a time. Iron is administered internally.

The patient had smallpox one and a half years ago, and suffers always from a cough. The parents have lost two children, one from bronchitis and the other from operation for a nævus.

It is remarkable that such marked relief should be given by prisms with the bases inwards, considering that the internal recti appear when tested with prisms even unduly strong in comparison with the external. It must be remembered, however, that even the internal are weaker than normal, and that the ordinary methods of testing with prisms are liable to be fallacious. The fact of his immediate and total relief by the prisms, however, remains.

(*May 10th*, 1883.)

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7. *Case of asthenopia relieved by the use of a prism placed vertically.*

By W. A. Brailey, M.D.

G. C— (f.), æt. 16. The right eye is constantly somewhat exposed, even in sleep, by the contraction of an old cicatrix just above the upper lid. She complains of occasional double vision and of aching in near work. On approaching an object to within 14″ the *right* diverges conspicuously in an outward and upward direction. Its vision is 2 J. at 15 cm. and $\frac{6}{36}$; with + 1·5 D. sph. + ·75 D. cyl., axis up and out, it equals $\frac{6}{9}$. This is nearly the

total measure of its hypermetropia. The *left* reads 1 J. from 17 to 40 cm. and has distant V. = $\frac{6}{6}$ poorly. It is somewhat improved by + 5 D. cyl., axis up and out.

With prisms the internal recti overcome 9° only, and the external 3° only. When at 8′ the images are separated by stronger prisms, that of the right (the false image) stands about six inches lower than that of the left. Under ordinary circumstances she has diplopia at $2\frac{1}{2}$ metres when looking down, and also when looking up, or up and to the left. The vertical meridian of the false image is usually straight but is sometimes slightly inclined to the left, especially when looking directly upwards. When the images are separated laterally, they are brought to a level by a prism of 3° apex down before the left. Then the internal recti are found to act more powerfully, overcoming 12° while the external remain as before.

Her full correction with sphericals and cylinders usually gives relief, but sometimes they render the diplopia even more distressing.

She finds great benefit by the occasional use, in near vision, of spectacles, in which a prism of 3° apex down before the left eye is also combined.

This case bears a striking resemblance, both as to symptoms and results of treatment, to one which I brought before the Society in 1881 (‘Transactions,’ vol. i, p. 188).

(*May 10th*, 1883.)

XI.—NEW INSTRUMENTS.

1. *On a new method of determining the relation between convergence and accommodation.*

By Ernest E. Maddox.

(Communicated by Prof. W. S. Greenfield, M.D.)

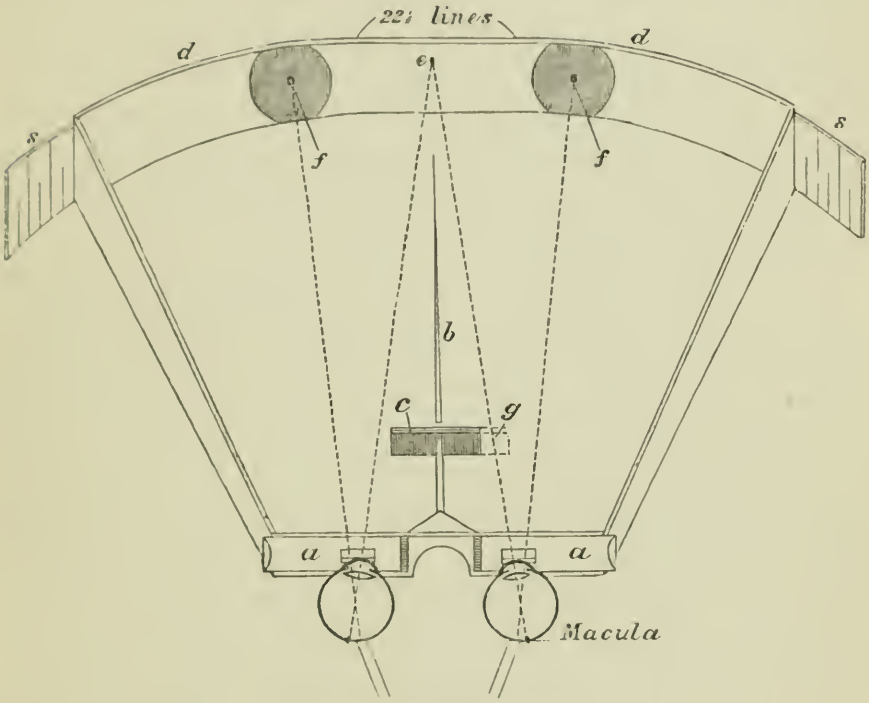
That, within limits, we can modify the ordinary relation between convergence and accommodation, by changing the nature of the afferent impulses from the retina, is a fact daily illustrated by the use of prisms and lenses, in which the appeal from double images or diffusion circles meets with a speedy response.

A question of some interest is: What part do such impulses play in the perfect harmony of normal binocular vision? Is the connection between the brain centres complete in itself and normally independent of their aid, or is it incomplete and supplemented by them?

It is the purpose of the instrument about to be described to estimate the effect of retinal impulses in each eye, and, by eliminating them in turn, to measure the efficiency of the co-ordinating apparatus without them. If one eye be placed completely in the dark, and its fellow be made to fix some near object, it is required to investigate the exact position of the benighted eye without the perception of a ray of light. To solve this problem the blind spot has been utilised.

The method is illustrated in the accompanying diagram, which represents a light wooden box, measuring about a foot from side to side, and eight and a half inches from before backwards. It is one inch deep along the curved

border, and inclines gradually to the depth of half an inch at the narrow end. The latter is provided with two visual apertures pierced through slides (*a*), which permit of mutual approximation or the reverse, and between them is a groove for the nose. A median partition (*b*) extends to within two inches of the middle of the curved border, and is crossed by a small transverse obstructive (*c*), which slides through a slit in the roof. The curved end of the box consists chiefly of two arcs (*d*), each



Camera with roof removed.

having its centre identical with the centre of rotation of the observer's corresponding eye, taking that point as 13 mm. behind the anterior surface of the cornea. They are united by a straight piece 22½ lines long, in the centre of which is a *small fixed aperture* (*e*). On either side is a *moveable luminous point* (*f*), one red the other blue, pierced through brass slides (*s*), which travel in grooves and allow each lateral aperture to sweep the whole corresponding arc. These luminous points are on a lower level

than the central aperture, since the optic disc is on a higher level than the retinal extremity of the visual axis.

The box is constructed on such a scale that every 4 mm. of the curve subtends an angle of one degree, with its apex at the centre of rotation of the eye on the same side. The brass slides are marked accordingly in degrees, which indicate in each the distance of the luminous point they bear from the centre aperture. This relation is easy to remember in association with the fact that 1 mm. of the retina subtends an angle of four degrees at the principal focus of the dioptric apparatus. One of the slides can carry a slip of paper, marked with figures or letters, which pass, one by one, before the central aperture. Since corresponding ones are marked upon the slide outside, the surgeon can check the observer's statements and ensure complete fixation.

With the *obstructive in the middle*, the observer holds the box up to the window and fixes the central aperture with both eyes. It is well to commence with the two moveable coloured points on the blind areas, the projection of the blind spot at the distance of the curve being about an inch wide, and subtending an angle of six degrees in most cases. One of the slides is then pushed inwards till the point it bears becomes just visible, when it is again withdrawn, thus registering the exact position of the inner border of the blind area when the visual axis is directed towards the central aperture. The diaphragm or obstructive is then pushed to the same side, say the right, as indicated in dotted outline *g*; thus obstructing the pencils of light from the *central* aperture to the right eye. The mind, as Donders says, cannot tell whether an object is seen with one or both eyes, and although the fixation aperture (*e*) looks a little dimmer, the observer does not know what has been done. Now, however, the right eye is absolutely in the dark subjectively, since the central aperture is hidden by the obstructive, and the image of the coloured point impinges on the optic disc, without producing any impression. In

nearly all eyes, after a moment's hesitation, the right coloured point springs into view, showing that the eye has rotated, and that the blind area has been moved to another part of the curve. Its inner border may now again be noted, and the deviation recorded in degrees.

We are therefore led to conclude that the co-ordination between the converging centres, unlike that between the accommodating centres, is incomplete, and the perfect harmony of ordinary binocular vision is contributed to by afferent impulses unknown to the mind, cut off in this case by the obstructive, which appeal either directly or indirectly to the nervous converging mechanism.

In every case, however, the idle eye maintains a considerable degree of convergence. To what extent this is due to co-ordination with the accommodating centres, and to what extent with the fellow converging centre, it is easy to investigate.

The observer, instead of fixing the central aperture itself, may gaze through it at some distant object. Accommodation then becomes negative in both eyes, and its effect upon convergence is eliminated. Again, if it is desired to note the effect of changes in the direction of the visual axis of the *active eye*, the central aperture may be blocked, and one of the moveable apertures used in its place. Observations may then be taken with either near or distant vision, and with the fixation point at varying distances from the median line.

There are many other ways in which the instrument may be used physiologically, but, before any classified results can be obtained from its use, the refraction of every eye experimented upon must be determined. It might possibly be of service in the measurement and analysis of squints ; and by determining the size of the blind area in optic neuritis, staphyloma posticum, and various conditions of the optic nerve, it would give information as to progress, and prognosis. For clinical purposes a different form of instrument might be useful, consisting of four brass tubes, like the letter W, capable of vertical as

well as lateral movement, to register the position and size of scotomata too small for the perimeter.

It would be interesting to illuminate the central aperture by lights of different colour and intensity, and notice whether the degree of convergence of the idle eye is affected by the nature or strength of impressions upon the retina of the active one.

In central amblyopia, by placing two Nicol's prisms before the central aperture in a rotating apparatus, a point might be reached in turning it round, when one eye would rotate outwards and the hitherto hidden coloured point spring into view, thus measuring the *degree* of amblyopia by a method more objective in principle than those involving the patient's judgment.

All these points, however, I must defer, but shall be glad to lay them before the Society at a later meeting if the subject proves deserving of further attention.

(*January 11th*, 1883.)

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2. *A new registering perimeter.*

By Priestley Smith (Birmingham).

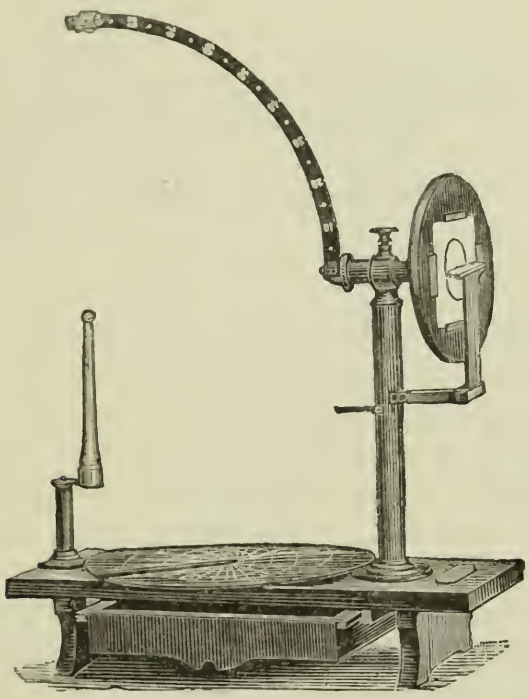
This is an improved form of the instrument described in the 'Ophthalmic Review' for November, 1882.

The quadrant is rotated by a wooden hand-wheel attached to its axis, and is balanced by a weight upon the hand-wheel, so that it will stand in any position without being fixed.

The chart is placed upon the hinder surface of the hand-wheel, and a horizontal scale is fixed immediately behind it, the divisions of which correspond with the circles on the chart. As the quadrant rotates the chart rotates with it, and in whatever position the quadrant stands the corresponding meridian lies against the scale.

This arrangement enables the operator to finish off his observations with the greatest ease, and has the further advantage that the chart is constantly under inspection, so that any part of the field can be immediately brought under examination or re-examination at any time, by turning the corresponding point on the chart round to the scale.

The test object is a square of paper gummed upon a light vulcanite wand; this the operator holds in his left



hand, while with the right he rotates the hand-wheel and pricks the readings upon the chart.

The charts are of two kinds, A and B, corresponding respectively to the whole visual field, and to its central area on a larger scale. The scale of the perimeter is graduated accordingly on its two sides; the A side is used with the A charts, the B side with the B charts.

There are cases in which the field is mapped more accurately by moving the test object in circles concentric with the fixation point than by moving it in meridians,

e.g. hemianopic, sector-like, and all other defects in which the boundary line runs in a meridional direction. In such cases the test object should be placed in the clip which slides upon the quadrant, and carried round the field in successive circles, every point at which it enters or leaves the sentient field being pricked upon the chart.

(*May 10th*, 1883.)

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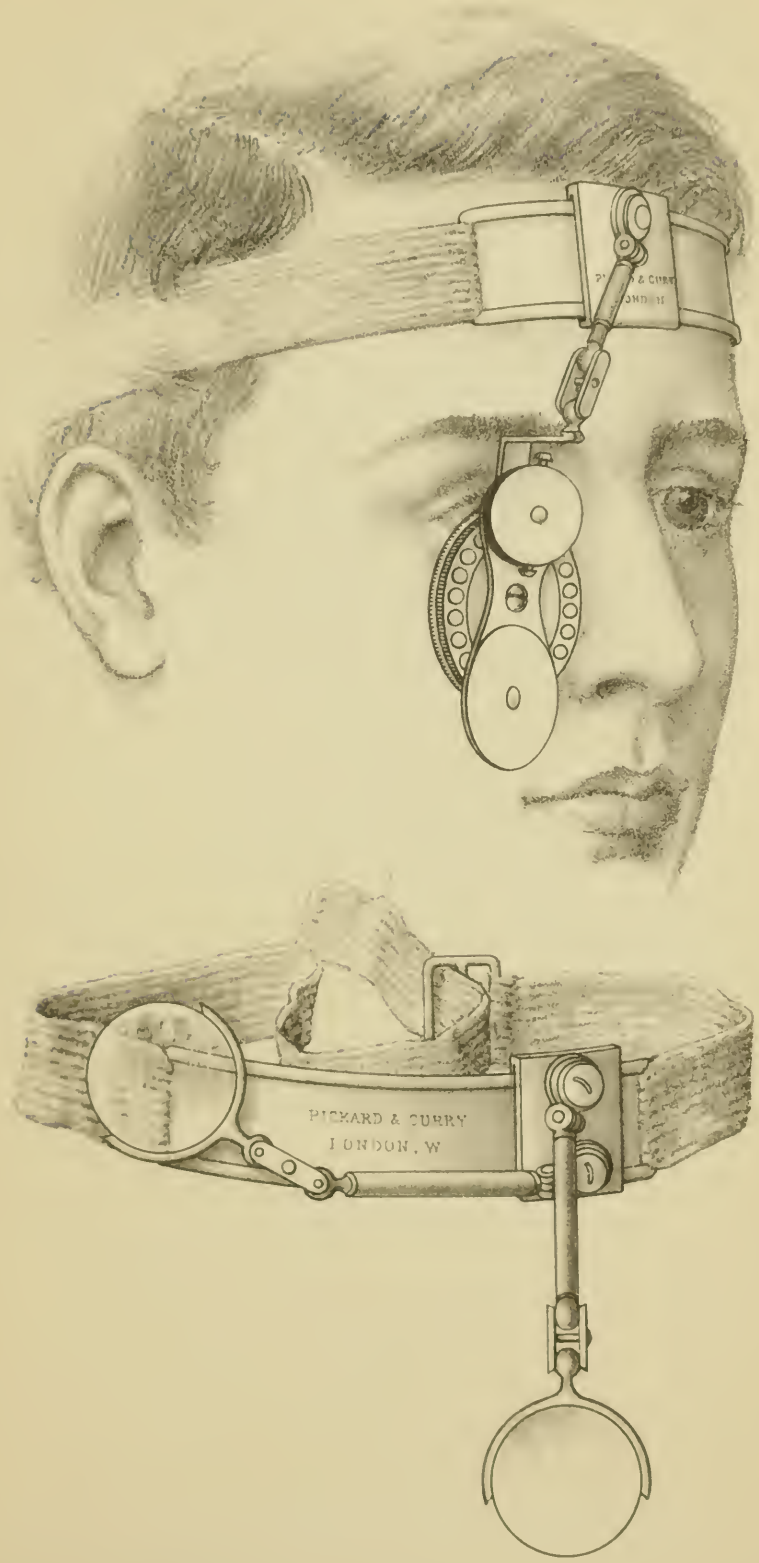
3. *Description of a new ophthalmoscope, designed especially for the use of artists.*

By James E. Adams.

(With Plate XV.)

The desirability of being able to illuminate all parts of the eye by means of lenses and mirrors, and of yet leaving the hands free, occurred to me some years ago on a certain occasion, when I had, without help and in a bad light, to remove a minute fragment of steel that was embedded in the cornea. For this purpose I devised that portion of the present contrivance which I call the "cornea illuminator," consisting simply of a metal band to be fixed on the patient's forehead, carrying two convex lenses on limbs with universal joints, one for focal illumination and the other to magnify the object. This instrument has proved of great use, and has enabled me to operate many times by artificial light without any extra assistance, which in urgent cases, such as injuries and glaucoma, may be of some importance. I have also frequently used it for examining the fundus in the inverted image.

It subsequently occurred to me that those engaged in making drawings of the fundus would find it a great help to have their hands free, and not to have to put down the ophthalmoscope to take up the brush, and *vice versâ ;* and I


PICKARD & CURRY
LONDON, W

also thought that perhaps, even for ordinary examinations, we might be glad to save ourselves the trouble of holding the instrument. Furthermore, Mr. McHardy pointed out that the drawings being frequently made by ladies, whose muscular systems may not be of the strongest, there was a probability of the hand being so far fatigued as to interfere with those finer touches of the brush so essential for accurate ophthalmoscopic drawing; and there is the possibility that the continuance of the combined effort of holding the ophthalmoscope and brush may produce a condition analogous to writer's cramp. The Plate illustrates sufficiently the principle on which the instrument is constructed. The difficulty in contriving the instrument was to devise any arrangement to allow of the mirror and lenses being placed as close to the eye as it would be if held in the hand. Messrs. Pickard and Curry have, I think, quite got over this difficulty, and we can now have an instrument with any number of lenses placed in any position we may desire.

For those who do not wish quite so large a series of lenses they have made a smaller instrument, consisting of one mirror and one disc, and this will be found sufficient for most people.

There are some objections to the band encircling the head, and I am now experimenting to see whether any practicable arrangement can be made whereby the mirror may be carried on a frame, like an ordinary frame made for the trial cases of lenses.

(*July 6th*, 1883.)

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4. *A magazine refraction ophthalmoscope.*

By John Couper.

The inconveniences of discs in the refraction ophthalmoscope as at present arranged, are obvious enough. In

order to obtain a sufficient number of glasses, we must either use numerous discs and submit to the trouble of changing them frequently, or we must use a pair of superposed discs and must add lens to lens, or subtract lens from lens, to obtain the higher glasses. The latter alternative involves the addition of an index wheel of a certain complexity, showing the focal length of the combined lenses, and it is further objectionable in that two masses of glass and four refracting surfaces are interposed precisely in the very cases where such interposition is undesirable. Discs, being close to the mirror, make the upper end of the instrument bulky, and bring the finger that works them close to, and frequently in contact with, the patient's face.

In the ophthalmoscope which I am about to describe, it is proposed to obviate these disadvantages by the total abolition of discs and the substitution of a new plan of construction. The handle of the instrument is converted into a magazine, containing a series of separate lenses, each of which can be brought in succession to the sight-hole of the mirror by means of a direct-action driving wheel. The mechanism is of the very simplest kind, and will be readily understood from the accompanying woodcuts.

In Fig. 1 the lid of the magazine is omitted so as to show the internal arrangement of the latter, and the method of direct propulsion. Fig. 2 shows the complete instrument in its more recent and improved form. The magazine consists of a shallow metal box, three quarters of an inch broad by ten inches long, containing seventy-two lenses. A central rib or projection divides the box lengthways into two parallel grooves which join at the ends, thus forming a continuous circuit for the lenses. The latter touch each other edgeways, but are not articulated as in a chain. The focal length of each glass is engraved on the metal disc which bears it, and can be read off at the sight-hole. Within the box, and on the spindle which carries the driving wheel, is a smaller wheel with six

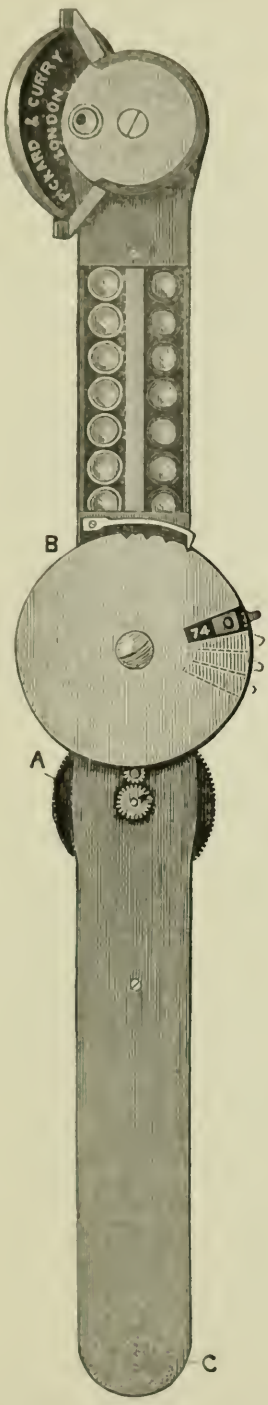

Fig. 2.

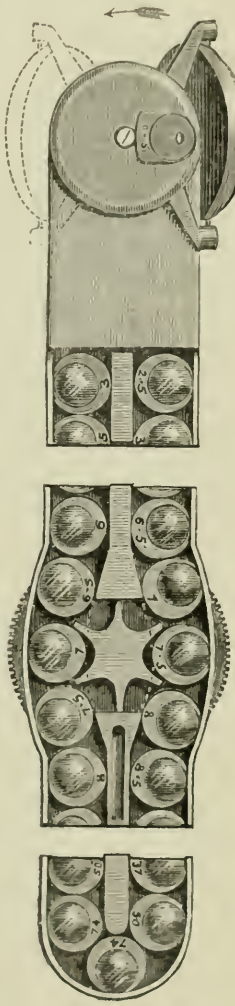

Fig. 1.

teeth. The lenses, being caught by the teeth, are propelled round the box. The back of the driving wheel is also notched at suitable intervals, and the point of a metal spring, falling into a notch, centres each lens as it arrives at the sight-hole.

The swing mirror of my former instrument has been retained. The axial line of its hinge is as nearly as practicable a tangent to the back of the mirror at a point close to the sight-hole on the one hand, and on the other to the edge of each lens as it becomes centred at the sight-hole.

By this arrangement the upper end of the instrument, being unencumbered by discs, is conveniently flat and narrow, and lies well under cover of the observer's brow. There is no lateral projection to touch the patient's face and prevent close approximation of eye to eye. The driving wheel is below the level of the patient's chin. The mirror can be so tilted as to reflect light into the eye under examination without inclining the lens, and with a minimum separation of lens from sight-hole.

The utmost proximity of lens to sight-hole is *desirable* (when lenses of five millimetres diameter are employed) as giving a wide angular aperture to the instrument, and is *necessary* for direct examination in high grades of myopia, in order to bring the eye of the ophthalmoscopist nearer to that of the patient than the far point of the latter.

The series of seventy-two is divided equally between minus and plus lenses, and in each section ranges from 0·5 D. to 74 D. I have chosen seventy-two merely as affording a very ample series, and not by any means as the furthest limits of number. On the contrary, in the instrument shown in Fig. 2 it is as easy to employ a larger series by lengthening the magazine, as it is to curtail the series by shortening the magazine. The effective diameter of each lens is five millimetres.

If the ophthalmoscope is to be used as an optometer it must obviously contain at least as many lenses as are usually found in clinical trial boxes. It must supply the

measure not merely of any possible ametropia in the patient, but also of that which may happen to be present in the observer's eye, and when the ametropia of examiner and examinee is the same in kind, the instrument should supply a measure of the sum of these quantities.

It is but a poor argument against this easily attainable completeness to say that the higher lenses of the series are but seldom needed. Whoever takes the trouble to make daily use of an instrument containing a lens series commensurate with the whole range of ametropia, will speedily discover its practical convenience. Let me quote one common example. The treatment of myopia often presents this difficult question—Is the myopia progressive, and is the amblyopia present a result of central hyperæmia, or other such nutritional disturbance which is amenable to medical treatment, or is it an incurable result of old textural change? It is unnecessary to insist on the importance of finding a true answer to this question, and on the light to be thrown on it by a good direct view of the fundus, displaying as it does in the higher grades of myopia a much amplified image of the fine pigmentary and other changes at the macula. These changes are only too easily overlooked in the inverted image, owing to its small size. The higher the grade of myopia the greater is the difficulty of obtaining a satisfactory direct view; each case is measured by one particular lens, and that lens alone gives the best image. In the highest grade, direct inspection is impracticable without a lens of adequate power, and even with it, if placed too far distant from the sight-hole. It is in such cases that the proximity of the lens to the sight-hole comes to tell. With a minus glass of three quarters of an inch focal length I have obtained a satisfactory direct view of the fundus of a myope, using for full correction a glass of one and a third inches. The far point of this eye can have been little more than two inches from the cornea.

In the ophthalmoscope shown in Fig. 1, the metal ring in which each lens is framed has margin enough at the inner

side to carry a figure denoting the power of the lens, which becomes visible at the sight-hole. This arrangement, although extremely simple, has certain drawbacks :

1. The large size of the frame of each lens adds to the length of the magazine.

2. In order that the figures may present in proper position at the sight-hole the broader part of each metal ring must point inward. Free rotation, therefore, of one ring on another is inadmissible. A facet ground on the outer edge of each, while it secures a due position of the number, at the same time increases friction by hindering the mutual free play of contiguous rings. This increase is an inappreciable quantity with a magazine of forty or even fifty lenses. It begins to tell, however, with seventy-two lenses.

To meet these difficulties I had constructed the instrument represented in Fig. 2, which answers its purpose admirably. It will be seen from the woodcut that the metal frames are reduced to rings of no greater thickness than suffices to protect the lenses. They carry no figures, and, being absolutely circular, each is free to roll on its neighbours. The mechanism resembles in principle the ball bearings of a bicycle wheel, the design of which is to minimise friction. In the present instance this object is fully attained, as is shown by the ease with which the whole series of seventy-two is propelled round the box. The finger on the driving wheel is not sensible of more resistance than in the ordinary disc instrument. With the lessened size of the individual pieces a smaller driving wheel (A. Fig. 2) and a shorter stroke suffice for propulsion.

The numbers are transferred to an index wheel (B. Fig. 2) which, being geared to the driving wheel, keeps pace therewith and with the lens series. Minus glasses have their rings coloured white, and are denoted by white figures on the wheel. Plus glasses have dark rings and are indicated by red figures. To economise space the white and red figures are arranged concentrically on the wheel. In

order to show at any given moment which way the driving wheel must turn to bring up plus or minus lenses to the sight-hole, a portion of the lid of the box is made of glass. Through this window the relative position of the plus and minus series is seen at a glance owing to their difference of colour.

Messrs. Pickard and Curry, the makers of the instrument, have taken incredible trouble in embodying my ideas. I desire to thank especially Mr. Paxon of that firm, without whose ingenuity and skill as a mechanician success would not have been possible.

(*May 10th*, 1883.)

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5. *Appliance for carrying ophthalmic ointments.*

By J. B. Story (Dublin).

The specimen consisted of a small German-silver cylindrical case about three inches long and one third of an inch in diameter, containing two collapsible tubes such as are used for moist colours, one tube being filled with an ointment of eserine, four grains to one ounce of vaseline, and the other being filled with an atropia ointment of the same strength.

(*Appliance shown. July 6th*, 1883.)

PRESIDENT'S ADDRESS

AT THE

THIRD ANNUAL GENERAL MEETING, JULY 6TH, 1883.

By WM. BOWMAN, LL.D., F.R.S.

THE Society has passed through its third session, and is in all respects in a satisfactory state. The expenses during the present session have not been heavy; and, as the Treasurer's Report will show, we have a good balance in hand.

During the session twenty-two new members have been elected, fourteen of these being metropolitan, three from colonies or dependencies, and five non-metropolitan but within the kingdom. There have been two withdrawals, and the Society has lost two members by death, viz. Mr. Critchett and Mr. R. W. Lyell. The Society has already given expression to its sense of the great loss it has sustained in the demise last autumn of our eminent and esteemed colleague Mr. Critchett, and this is not the time to enlarge upon it. Mr. Lyell was a young surgeon and ophthalmologist of brilliant attainments and great promise, and his early death has deprived the Society of a member whom we had hoped would add much to the value of its records.

Our gain *in number* during the session is therefore eighteen; and our total number of ordinary members is now 176.

The number of meetings has been the same as last year, viz. eight; seven of these being fixed and one an extra meeting for the adjourned discussion on Eye Symptoms in Spinal Disease.

Owing to the building operations which have been

going on nearly all the session, several of the meetings, especially in the cold weather, were held in considerable discomfort, despite the best efforts of the officials of the Medical Society, to whom we are always much indebted. We may therefore, I think, feel well satisfied that the average attendance of members has been very nearly as good as in the two previous sessions.

The time of the Society has at every meeting, without exception, been very fully occupied with communications and the discussions arising out of them. The total number of communications made this session is slightly less than last session, being sixty-six instead of seventy-one; we have, however, had several papers of much scientific value; and there have also been several communications of considerable pathological and clinical importance. The exhibition of living specimens has been much hampered by the state of the premises, but this important feature of our meetings will no doubt again assume larger proportions in future sessions.

The special subject opened by Dr. Gowers at the June meeting gave rise to a fair discussion, and to the contribution of a considerable mass of facts. This discussion is of much value as it stands, but its greatest use will (as in similar instances before) doubtless lie in the stimulus it will give to more exact clinical and pathological research in regard to certain specified points and problems.

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