Presented to
The Library
of the
University of Toronto
by
THE ALEXANDER MACDONALD FOUNDATION
OPHTHALMOLOGICAL TRANSACTIONS.

VOL. V.
TRANSACTIONS
OF THE
OPHTHALMOLOGICAL SOCIETY
OF THE
UNITED KINGDOM.

VOL. V.
SESSION 1884-85.

WITH
LIST OF OFFICERS, MEMBERS, ETC.

LONDON
J. & A. CHURCHILL
11, NEW BURLINGTON STREET
1885
IN EXCHANGE.

Archives d'Ophtalmologie, Panas, Landolt and Gayet.
Centralblatt für praktische Augenheilkunde, J. Hirschberg.
Klinische Monatsblätter, Augenheilkunde, W. Zehender.
Récuei d'Ophtalmologie, Galezowski and Cuignet.
Récuei générale d'Ophtalmologie, Dor and E. Meyer.
Transactions of the American Ophthalmological Society.
Récuei des Sciences Médicales, Hayem.
Archives of Ophthalmology. Dr. H. Knapp, 25, West 24th Street, New York.
Graefe's Archivs für Ophthalmologie. Prof. H. Leber, Gottingen.
NOTICE.

The present volume comprises the proceedings of the Ophthalmological Society of the United Kingdom, during its fifth Session, October, 1884, to July, 1885.

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.
# TABLE OF CONTENTS

## OF VOLUME V.

<table>
<thead>
<tr>
<th>Subject</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Officers and Council</td>
<td>IX</td>
</tr>
<tr>
<td>List of Members</td>
<td>XI</td>
</tr>
<tr>
<td>Rules</td>
<td>XXVII</td>
</tr>
<tr>
<td>List of Communications made during the Session 1884-85</td>
<td>XXXIII</td>
</tr>
<tr>
<td>List of Plates</td>
<td>XL</td>
</tr>
<tr>
<td>List of Woodcuts</td>
<td>XL</td>
</tr>
<tr>
<td>Bowman Lecture</td>
<td>1</td>
</tr>
<tr>
<td>Prevention of Blindness from Ophthalmia Neonatorum</td>
<td>31</td>
</tr>
<tr>
<td>Diseases of Eyelids and Conjunctiva</td>
<td>41</td>
</tr>
<tr>
<td>Intra-ocular Tumours, &amp;c.</td>
<td>47</td>
</tr>
<tr>
<td>Diseases of the Iris</td>
<td>66</td>
</tr>
<tr>
<td>Sympathetic Ophthalmitis</td>
<td>71</td>
</tr>
<tr>
<td>Panophthalmitis</td>
<td>101</td>
</tr>
<tr>
<td>Variations of Tension</td>
<td>106</td>
</tr>
<tr>
<td>Diseases of the Lens and Capsule</td>
<td>107</td>
</tr>
<tr>
<td>Diseases of the Retina</td>
<td>113</td>
</tr>
<tr>
<td>Diseases of the Choroid</td>
<td>136</td>
</tr>
<tr>
<td>Diseases of the Optic Nerve</td>
<td>149</td>
</tr>
<tr>
<td>Functional Diseases</td>
<td>193</td>
</tr>
<tr>
<td>Injuries and Operations</td>
<td>199</td>
</tr>
<tr>
<td>Congenital Defects</td>
<td>207</td>
</tr>
<tr>
<td>Instruments</td>
<td>209</td>
</tr>
<tr>
<td>On Cocain</td>
<td>211</td>
</tr>
<tr>
<td>Report of the Council</td>
<td>253</td>
</tr>
</tbody>
</table>
OFFICERS AND COUNCIL

OF THE

Ophthalmological Society of the United Kingdom,

ELECTED AT

THE ANNUAL GENERAL MEETING, JULY 3rd, 1885.

President.

JONATHAN HUTCHINSON, F.R.S.

Vice-Presidents.

SIR WILLIAM BOWMAN, BART., F.R.S.
GEORGE JOHNSON, M.D., F.R.S.
THOMAS REID, M.D. (Glasgow).
T. SYMPSON (Lincoln).
D. ARGYLL ROBERTSON, M.D. (Edinburgh).
J. C. WORDSWORTH.

Treasurer.

J. F. STREATFEILD.

Librarian.

W. ADAMS FROST.

Council.

JOHN ABERCROMBIE, M.D. | PRIESTLEY SMITH (Birmingham).
SIDNEY COULAND, M.D. | SIMEON SNELL (Sheffield).
GEORGE COWELL. | J. B. STORY (Dublin).
G. A. CRITCHETT. | JOHN TWEEDY.
WALTER EDMUNDS. | W. SPENCER WATSON.
A. STANFORD MORTON. | E. NETTLESHIP.

Secretaries.

W. A. BRAILEY, M.D.
SEYMOUR J. SHARKEY, M.B.
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. L.—Librarian.
Pres.—President. S.—Secretary.
V.-P.—Vice-President. C.—Member of Council.
T.—Treasurer.
*.—Denotes Resident Life Members.
†.—Denotes Non-Resident Life Members.

GENERAL LIST OF MEMBERS.

ELECTED

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

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

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


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

O.M. Andrew, Edwyn, M.D., Surgeon to the Shropshire Eye and Ear Hospital; Hardwick House, St. John’s Hill, Shrewsbury. (C. 1881-4.)

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

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

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

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

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

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

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

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

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

1881 Bickerton, T. H., 1, St. James Road, Liverpool.

1885 Blumer, W. P., Honorary Surgeon to the Sunderland and North Durham Infirmary; 12, North Bridge Street, Sunderland.

1884 Bond, Charles J., The Infirmary, Leicester.

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

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

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., Senior Surgeon to the Liverpool Eye and Ear Infirmary; 39, Rodney Street, Liverpool.

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

1885 Browne, Oswald A., 25, Bernard Street, Russell Square, W.C.

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.
Elected

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.

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

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

1885 Clarke, Ernest, Surgeon to the Miller Memorial Hospital, and Assistant Surgeon to the Central London Ophthalmic Hospital.

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

O.M. Cooper, William White, Consulting Ophthalmic Surgeon to St. Mary’s Hospital; 19, Berkeley Square, W.

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

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

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

O.M. Cowell, George (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.
Elected

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

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

1884 Davidson, James Mackenzie, Aberdeen University.

O.M. Denby, T. 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.

1883 Dew, Henry, Berkeley Square, Bristol.

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

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


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

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

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

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

O.M. Ferguson, H. L., Ophthalmic Surgeon to the Dunedin Hospital; Dunedin, New Zealand.

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

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. (L.), Assistant Ophthalmic Surgeon to St. George's Hospital; 77, Wimpole Street, W.
1883  †Da Gama, Jerminio Accacio, Khoja Moola, Bombay.
1885  Gay, William, 111, Disraeli Road, Putney, S.W.
1883  Gibbons, R. A., M.D., Physician to the Grosvenor Hospital for Women and Children; 32, Cadogan Place, S.W.
O.M.  Glascott, C. E., M.D., Surgeon to the Manchester Royal Eye Hospital; 11, St. John Street, Manchester.
1885  †Godfray, Alfred Charles, St. Helier's House, St. Helier's, Jersey.
O.M.  Gowers, W. R., M.D., Assistant Professor of Clinical Medicine at, and Assistant Physician to, University College Hospital; 50, Queen Anne Street, W. (C. 1880-3.)
O.M.  Greenfield, W. S., M.D., Professor of Pathology University of Edinburgh; Heriot Row, Edinburgh.
1885  Griffith, A. H., M.D., Assistant Surgeon, Royal Eye Hospital; 17, St. John Street, Manchester.
O.M.  Grossman, K. A., Ophthalmic Surgeon Stanley Hospital, Liverpool; 70, Rodney Street, Liverpool.
1881  Gulliver, George, M.B., Assistant Physician to St. Thomas's Hospital, and to the London Fever Hospital; 16, Welbeck Street, W.
O.M.  Gunn, R. Marcus, Assistant Surgeon to the Royal London Ophthalmic Hospital, Moorfields, Ophthalmic Surgeon to the Hospital for Sick Children, Great Ormond Street; 54, Queen Anne Street, W. (C. 1882-5.)
1882  *Hartridge, Gustavus, Consulting Ophthalmic Surgeon to St. Bartholomew's Hospital, Chatham, and Assistant Surgeon to the Royal Westminster Ophthalmic Hospital; 47, Kensington Park Gardens, W.
1882  †Henderson, W. H., M.D., Kingston, Ontario, Canada.
ELECTED


O.M. Higgins, 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.

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

1884 Hudson, Ernest, Royal London Ophthalmic Hospital, Moorfields, E.C.

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.)

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

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

1883 †Jenkins, E. J., M.D., Nepean Towers, Douglass Park, Sydney, N.S.W., Australia.
1883 Jessop, W. H. H., Senior Assistant Surgeon to the Central London Ophthalmic Hospital; Ophthalmic Surgeon to the Paddington Green Children's Hospital; 73, Harley Street, W.

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

1882 Johnson, G. L., M.B., 14, Stratford Place, Oxford Street, W.

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

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

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

O.M. Lawford, J. B., M.D., Curator and Librarian to the Royal London Ophthalmic Hospital, Moorfields; Royal London Ophthalmic Hospital, Moorfields, E.C.

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

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

O.M. Lediard, H. A., M.D., Surgeon to the Cumberland Infirmary; 43, Lowther Street, Carlisle.
1885  Lee, Charles E., Assistant Surgeon, Eye and Ear Infirmary, Liverpool; 84, Bedford Street, South Abercromby Square, Liverpool.

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

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

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

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

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

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

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

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

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

1883 †Mauer, W. O., M.D., 20, College Street, Hyde Park, Sydney, N.S.W.

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

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

1884 Maxwell, Patrick William, M.B., 10, Lower Mount Street, Dublin.
ELECTED

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

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

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

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

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

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

O.M. Morton, A. Stanford, Senior Assistant Surgeon to the Royal South London Ophthalmic Hospital; 26, Weymouth Street, W.

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

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.

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

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.
XXI

Elected

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, Consulting Surgeon to the Sussex Eye Hospital; 7, Brunswick Place, Brighton.

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

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

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

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

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. (V.-P.), Surgeon to the Glasgow Eye Infirmary, and Lecturer on Ophthalmic Medicine, University of Glasgow; 11, Elmbank Street, Glasgow.

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

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.

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

1885 Rose, James, Ophthalmic Surgeon, Bootle Hospital; Clinical Assistant, Eye and Ear Hospital, Liverpool; 61, Hope Street, Liverpool.

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

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

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

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

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

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

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

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

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

1883 Silcock, A. Q., M.D., 5, Graham Road, Dalston, E.

1883 Skinner, D. S., M.D., 1, Bedford Gardens, Campden Hill, W.
ELECTED

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

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

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

O.M. Snell, Simeon (C.), Ophthalmic Surgeon to the Sheffield General Infirmary; 17, Eyre Street, 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; 14, Portland Square, Plymouth.

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

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.


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

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

1883 *Symons, Mark Johnston, M.D., Port Adelaide, South Australia.

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

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

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


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

1885 THOMPSON, C. S., Bideford, Devon.

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

1883 TOBIN, WILLIAM, 31, Hollis Street, Halifax, Nova Scotia, Canada.

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

O.M. TOSSWILL, L. H., Surgeon to the West of England Eye Infirmary, 49, Magdalen Street, Exeter.

O.M. TWEEDY, JOHN (C.), Assistant Ophthalmic Surgeon to University College Hospital, and Professor of Ophthalmic Medicine and Surgery at, University College; Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 24, Harley Street, W.

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

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

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

1885 WERNER, LOUIS, 5, Church Avenue, Rathmines, Dublin.

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


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

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

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

O.M. Wordsworth, J. C. (V.-P.), Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 20, Harley Street, W. [Re-elected 1883.]
RULES.

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

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

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

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

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

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

7. Expulsion of Members.—A member can be expelled only at a General Meeting specially called for that purpose, and of which a written notice shall have been sent to every member at least fourteen days previously. At least ten votes must be recorded, and four fifths shall carry the expulsion.
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. Two Scrutineers appointed by the Chairman at the commencement of the Annual Meeting shall receive the lists during the first hour, and report the result to the Chairman. 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. The Librarian shall have entire charge and control of the Library. He shall purchase books for the Library as opportunities arise at his discretion out of the grant previously voted for this purpose by the Council. He shall see that all books belonging to the Society are duly entered in the Catalogue, and that the periodicals and pamphlets are from time to time, as occasion may require, suitably bound. It will be his duty to see that the Library Rules are not infringed.

15. 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.

16. The Council shall meet half an hour before the meeting in October, January and May, and half an hour before the Annual General 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.'

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

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

19. Visitors.—Each Member may introduce two visitors on writing their names in the attendance book.
20. The business at Ordinary Meetings shall consist in the reading and discussion of papers, which may be illustrated by specimens, drawings, &c. When patients are to be shown they should attend half an hour before the meeting.

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

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

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

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

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

LIBRARY RULES.

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

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

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

4. A book must be returned at the expiration of a fortnight if
wanted by any other Member. The Librarian will in such a case write to the Member in whose name the book was taken out.

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

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

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

THE BOWMAN LECTURE.

Resolution of Council, September 18th, 1883.

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

BROUGHT BEFORE THE SOCIETY DURING THE SESSION 1884-5.

On the relation of certain diseases of the eye to gout.
Being the Bowman Lecture, delivered Thursday, November 13th, 1884.
By Jonathan Hutchinson, F.R.S. 1

Communication with the Government on the prevention of blindness from ophthalmia neonatorum 31

I. DISEASES OF EYELIDS AND CONJUNCTIVA.

1. Primary lupus of the conjunctiva.
   By Arthur H. Benson 41

2. Rapidly extending isolated patch of favus on adult upper eyelid, treated by excision.
   By M. M. McHardy 42

3. Fat developed in both upper eyelids.
   By M. M. McHardy 44

4. Obstinate case of ectropion successfully treated by Argyll Robertson's operation. By M. M. McHardy 45

5. Vesicle of cornea.
   By Anderson Critchett and Henry Juler 46

VOL. V.
II. INTRA-OCULAR TUMOURS, ETC.

1. Tuberculosis (?) of the cornea and iris. By Arthur H. Benson 47
2. Tubercle of the iris and choroid; cultivation of the bacilli in blood serum. By W. H. Jessop 55
3. An intra-ocular gumma in a child the subject of inherited syphilis. By W. Spencer Watson 56
4. Double retinal glioma resulting in the shrinking of one eye and the perforation of the other. By W. A. Brailey 61
5. Glioma of both retinae. By W. Lang 64
6. A dark-coloured lobulated growth over the ciliary region. By M. M. McHardy 65

---

III. DISEASES OF THE IRIS.

1. New formation of pigment on front of iris. (With Plate I). By E. Nettleship 66
2. Cystic tumour of iris. By F. R. Cross 67
3. Cyclo-choroiditis with vitreous hæmorrhages, subsequently involving the iris, with hyphæma, varying tension, and later, enormously deep anterior chamber By W. A. Brailey 68
4. Sequel to a case of granular-looking body on iris. By Frank H. Hodges 70

---

IV. SYMPATHETIC OPHTHALMITIS.

1. On a blood theory in explanation of reflex ophthalmitis. By Jonathan Hutchinson, F.R.S. 71
2. On the condition of the ciliary nerves in certain diseases of the eye. By W. A. Brailey 93
3. Microscopical specimens showing an exceptional condition of the ciliary nerves in three cases of uveitis. By W. A. Brailey 98
4. Microscopical specimens showing the condition of the ciliary nerve in a case of sympathetic disease. By W. A. Brailey 99
5. Sympathetic disease of one week's standing, indicated by rigidity of the pupil and a history of two attacks of swelling of the lid, with pain in and about the eye. By W. A. Brailey 99

V. PANOPHTHALMITIS.

1. Purulent irido-cyclitis with opaque vitreous in a young child suffering from a febrile illness,—? Pyæmia,—? Meningitis.—Death in six weeks;—Cerebro-spinal meningitis from purulent disease of the middle ear. —The meningitis and eye disease probably pyæmic. —Examination of the eye after death. By E. Nettleship 101

VI. VARIATIONS OF TENSION.

1. Persistent diminished tension after blow with nail five months ago. By W. A. Brailey 106

VII. DISEASES OF THE LENS AND CAPSULE.

1. Note on the spontaneous disappearance of diabetic cataract. By E. Nettleship 107
XXXVI

2. Case of black cataract. By Henry Power 111
3. Symmetrical dislocation of lenses upwards; congenital. By F. R. Cross 111

VIII. DISEASES OF THE RETINA.

1. Remarks on three recent cases of detachment of the retina. By W. A. Brailey 113
2. Two cases of night blindness from exposure to a bright light. By W. Adams Frost 123
3. A case of supposed unilateral albuminuric retinitis. By Henry Eales 126
4. Visual function retained by a retina which has almost certainly been detached for several years. By E. Nettleship 133
5. Detached retina in the yellow spot region. By W. Lang 135

IX. DISEASES OF THE CHOROID.

1. Naevus of left side of face; naevus of choroid, sub-retinal haemorrhage, and detached retina in left eye. (With Plate II.) By J. B. Lawford 136
2. Central choroiditis with disseminated patches in remainder of fundus. By W. Lang 140
3. Central choroiditis. By W. Lang 141
4. Bands of connective-tissue growth in vitreous with choroidal atrophy. By W. Lang 141
5. Atrophy of choroid. (With Plate III.) By A. Stanford Morton 142
6. Uniocular irido-choroiditis with new formation of connective tissue in the vitreous. By R. Marcus Gunn 144
7. Severe central choroiditis with almost perfect acuteness of vision; visual field defective at periphery. (With Plate IV.)

By E. Nettleship 147

X. DISEASES OF THE OPTIC NERVE.

1. A case of amblyopia with partial optic atrophy and general nervous depression and emaciation, caused by the vapour of bisulphide of carbon and chloride of sulphur. Partial recovery. By E. Nettleship 149

2. A case of amblyopia with slight neuritis followed by pallor of the discs, caused by the vapour of bisulphide of carbon and chloride of sulphur; severe nervous depression, emaciation, and muscular wasting. Recovery. (Communicated by E. Nettleship.)

By Ernest Fuchs 152


157

4. Case of symmetrical coloboma to the temporal side of each optic disc. (With Plate V, fig. 1.)

By W. H. Jessop 176

5. A case of opaque nerve-fibres. (With Plate V, fig. 2.)

By G. Hartridge 177

6. Optic neuritis, increased tension, nasal polypi, numbness of face, slight hemiplegia and albuminuria.

By W. A. Brailey 178

7. Symmetrical enlargement of upper part of face, with double proptosis and optic atrophy, complete anosmia and paralysis of left portio dura.

By R. Marcus Gunn 180

8. Further observations on the condition of the optic nerves in intracranial disease. (With Plate VI.)

By Walter Edmunds and J. B. Lawford 184
XI. FUNCTIONAL DISEASES.

1. Recurrent paralysis of third nerve in association with migraine.
   By Simeon Snell 193
2. Case of conjugate deviation of the eyes, down and to the right.
   By W. Adams Frost 197

XII. INJURIES AND OPERATIONS.

1. Rupture of the eyeball to outer side in February, 1884, now showing rupture of choroid; recovery with useful vision.
   By Walter H. Jessop 199
2. Evisceration of the globe.
   By P. H. Mules 200

XIII. CONGENITAL DEFECTS.

   By W. Lang 207
2. Peculiarly shaped eyeball.
   By R. Marcus Gunn 207

XIV. INSTRUMENTS.

1. Ophthalmic models for teaching.
   By Priestley Smith 209
XV. ON COCAIN.

2. On the use of cocain in ophthalmic practice. By E. Nettleship 217
3. The cocainised eye. By Walter H. Jessop 240

Report of the Council 253
LIST OF PLATES.

I. New formation of pigment on front of iris.
   E. Nettleship 66

II. Nævus of choroid.
    J. B. Lawford 138

III. Atrophy of choroid.
     A. Stanford Morton 144

IV. Severe central choroiditis, with almost perfect acuteness of central vision.
    E. Nettleship 147

V. Fig. 1. Coloboma of optic disc.
    W. H. Jessop 176

   Fig. 2. Opaque nerve-fibres.
          G. Hartridge

VI. Figs. 1 and 2. Illustrating the condition of the optic nerves in intra-cranial disease.
   Walter Edmunds and J. B. Lawford 184

LIST OF WOODCUTS.

Charts of fields of vision in detachment of retina (W. A. Brailey's paper) . 114, 115, 117, 118, 120, 121

hart of field of vision in atrophy of choroid (A. Stanford Morton's case) . 143

Charts of fields of vision in amblyopia from bisulphide of carbon, &c. (Professor Fuchs' case) . 154—156

Tracing of pulse in chronic Bright's disease, with optic neuritis (W. A. Brailey's case) . 179
ON THE RELATION

OF

CERTAIN DISEASES OF THE EYE TO GOUT.

BEING

THE BOWMAN LECTURE,

Delivered Thursday, November 13th, 1884,

By Jonathan Hutchinson, F.R.S.

Gentlemen,—At the request of your Council I have the honour this evening to deliver the first Bowman Lecture. This lecture has been instituted with the design of gratefully commemorating the services of Sir William Bowman to science, and more particularly those which he has rendered to this Society in his capacity as its first President.

It is intended that the lecturer shall on each occasion undertake the investigation of some special subject in connection with Ophthalmology.

I do not think that any more appropriate method could have been devised by which to pay respect to one of our very foremost workers. For myself, I can only say that I feel deeply the responsibility of the yet very agreeable duty which has devolved upon me, and could heartily wish that your Council had selected some one more capable of doing it justice. Having made this personal apology, I feel, however, that I shall best honour the name of Bowman by trying to imitate the example of his life, and
proceeding at once, to the best of my ability, to deal with the subject in hand.

I take for the topic of my lecture the relations which exist between certain Diseases of the Eye and Gout. We shall have to classify several different forms of disease—for the most part, but not exclusively, inflammations of the iris—to describe their peculiarities and to examine the evidence which is forthcoming as to their real connection with gout. The inquiry is an important one, for a gout taint is of extremely common occurrence in a large proportion of the British population. The investigation is also in many of its ramifications one of great intricacy and difficulty. Certain facts we shall, I trust, be able to establish with clearness, but respecting many others we shall not be able to get further than the stage of more or less confident inference. I had at one time intended to bring before you a good deal in the form of attempted proof—that is, of statistical or tabular statements of fact. I have found, however, in the preparation of the lecture that the time at my disposal will not permit of more than a brief sketch of the whole subject, and I shall therefore, on compulsion, spare you much of the detail. It is, however, only just, both to you and to myself, to state that my arguments and assertions this evening will be based upon the careful examination of a lengthy series of recorded cases. A large part of these will be found given in detail in vols. vii and viii of the Ophthalmic Hospital Reports, and the remainder I have in type before me, although not yet published.

Definitions of Gout and Rheumatism.

Before we proceed to inquire whether gout may be deemed to be the cause in some instances of such diseases as haemorrhagic retinitis, recurring iritis, glaucoma, and others, it may be well to state that I wish to keep a clear distinction between gout and rheumatism. This distinction
is in practice often very difficult and sometimes quite impossible to draw, but for all purposes of accurate clinical pathology we must insist upon its existence. By gout I shall mean all states of health which are, whether directly or remotely, connected with the accumulation of lithate of soda in the blood, as the result of overfeeding or defective assimilation.

Under the laws of inheritance of morbid tendency it may easily be the fact that a person may feel the results of this condition in his parents, who may yet never himself exhibit it. We may have a form of inherited gout which is not associated in the patient with either lithiiasis or lithæmia. Hence, as I shall have to show presently, some very peculiar forms of morbid action.

Thus it may perhaps be conveniently laid down that there are two fundamental conceptions of what gout is. They are not antagonistic, but rather in mutual co-existence. In practice the two methods of influence constantly modify each other.

What I may call the primary conception of gout regards it as due to a state of the blood induced in part by errors in feeding, and in part by defects in assimilation and depuration. It has regard to the food, the digestive power, the habits as regards exercise, and the integrity of the kidneys. In this form of dietetic, or humoral, gout the stage of lithæmia is essential, and the final product of deposit of urates in the tissues almost equally so. We must, however, seek a wider vision than this. As an introductory stage let us imagine the condition of a man who many years ago was subjected for a long period to the ordinary causes of primitive or humoral gout, but who for a considerable series of years has carefully avoided them. In such a person it is easy to see that although his blood may have been long kept free from excess of urates, yet, as the consequence of conditions of long duration in the past, his tissues may have received modification, and may be prone still to suffer in a peculiar manner when exposed to the ordinary exciting causes of
disease. In particular they may be liable to grow old on a peculiar type. Not only are his tissues in general modified, but those of his nervous and vascular system have probably been especially influenced. Hence the power of control over any process of inflammation, by whatever cause it may have been initiated, is peculiar and defective. If such a man sprains a joint it will not recover in the way it would have done had he never had gout, yet the joint may nevertheless be very far from assuming a typical gouty state. There may be no tendency whatever to the deposit of urates. Now let us take another step in the attempt to give reality to our conceptions of the hidden influences under which the every-day phenomena about us are produced. Let us imagine that such a person as we have supposed, one who has suffered severely, and through many years, from humoral gout, becomes a parent. His offspring will inherit his tissues, his tendencies as regards digestion, kidney excretion, skin elimination, and all the rest. These will, no doubt, give a greatly increased proclivity to common typical gout should the causes of it come into play. They may, however, do much more than this. They may render their subject liable, quite independently of exciting causes, or with but little help from them, to forms of inflammation or of degraded nutrition, which are not unequivocal gout, but which are yet the direct consequence of it in predecessors. The modification which is here inherited is, I repeat, of the tissues and not of the blood, though it is obviously very easy that the two may complicate each other. The subject of tissue modification from inherited gout may easily become the victim of humoral gout as well.

Inherited Gout.

It is clear from this argument that we must be prepared to see inherited gout assume very different forms from those in which we are so well accustomed to recognise the
primitive type. Permit me now to suggest that it is the clinical, or rather the social, fact that a not inconsiderable proportion of the well-to-do classes of the British population are born under the conditions which I have been supposing. The number of those who are the descendants of parents who have suffered from humoral gout is very great indeed, and, especially in our large towns, it is by no means restricted to the wealthy classes. We must be prepared, then, to encounter, not unfrequently, peculiar types of morbid action which derive their peculiarities from this association. It is the duty of the clinical investigator to carefully examine the evidence which is forthcoming as regards each before he admits its claim. Certain diseases of the eye for which this claim is with more or less confidence put forward will form part of our topics for discussion this evening. It may, perhaps, facilitate matters if I express at this stage the belief that most of the varieties of what is called "rheumatic gout" stand in this relation to true gout. The list would include lumbago, sciatica, neuralgia, crippling rheumatism, arthritis deformans, osteo-arthritis, many forms of iritis, and many cases of glaucoma. I by no means wish to suggest my belief that any one of these is, in all cases, in some degree dependent upon the inheritance of gout. Some of them are doubtless in not a few instances purely rheumatic, and this admission renders it desirable that I should say two or three words as to what our conception of rheumatism ought for the most part to be. Let me hint that from our primary idea of rheumatism I should like to exclude all causes having reference to food or assimilation, and to count only those which regard climate and weather, and especially exposure to cold and damp. The rheumatic diathesis, like that of gout, may become ingrained, and at the same time modified, by hereditary transmission, and in this connection it is easy to overlook its original causes and even to believe that it may begin spontaneously, or in consequence of other influences than the true ones. The hereditary diathesis of rheumatism
(arthritic susceptibility to weather) may easily co-exist with that of gout (arthritic susceptibility to diet), and each of them or both together may combine in endless variety with other forms of morbid tendency. It follows that we have to study many hybrid, or mongrel, types of disease in more or less close connection with them.

"Hot Eye," Irritable Eyes, Quiet Gout, &c.

There is a condition which, for want of a better name, I have for long been in the habit of recognising as Hot Eye. It is one of the many curious phenomena which attend quiet gout. I use the term quiet gout as distinct from acutely paroxysmal gout. In families liable to gout, for one person who becomes the subject of acute attacks of podagra—what is known as "unequivocal gout"—there are often half-a-dozen who are the subjects of minor symptoms which denote a similar tendency. In them the tendency never rises to sufficient height to induce a severe and characteristic paroxysm. They are nevertheless liable, after taking beer or wine injudiciously, as to quantity or quality, to experience slight pricking pains in joints, attended by lithic acid in the urine, and other unmistakable signs of the diathesis. In many, these symptoms occur in such invariable association with the causes suggested that it is not possible to doubt as to their nature. The liability varies with the weather and time of year, and it often ends, unless precautions are taken, in a sharp attack in the great toe. The terms "suppressed gout," or better, "undeclared gout," are often used in reference to it, but since it is simply a minor form of the disease, not in any way suppressed, and certainly not to trained observers undeclared, I prefer the term quiet. Amongst the frequent occurrences which denote its presence are repeated short attacks of congestion of the eye. Usually one only is affected, but sometimes both. The conjunctiva becomes red, and the eyeball feels hot, and pricks, as if sand were
in it. The attack may come on within half an hour of the meal which has disagreed, and it may last a few hours, or a day or two. Sometimes, owing to interference with the ciliary muscle, vision is slightly dim, and all attempts at accommodation are usually painful. Those who are liable to "hot eye" not unfrequently in the end suffer from iritis; indeed it is not unfrequently an introductory stage to that disease. On the other hand, many are liable for years to very frequent attacks of it without apparently any risk of its assuming more serious proportions. In the intervals of the attacks, the eye is usually quite well, and it is indeed its definitely paroxysmal character, its sudden development, and very complete and rapid disappearance, which especially mark its arthritic relationship.

In connection with this subject it may be noted, that not unfrequently those who suffer from unequivocal gout, experience shoots of sharp pain in the eyeballs, the nature of which they well understand. A man, William B—, aet. 51, who had suffered much from gout, and whose father and four of his father's brothers had all had it, complained much of this kind of pain, yet he had never had iritis. When he was gouty, he said he frequently had "darts of pain through the middle of each eyeball," and occasionally across the bridge of the nose.

A further practical note I must ask leave to make. It is to the effect that many cases of irritable, hyperaesthetic and easily tired eyes in young persons are in association with inherited gout. I feel sure that in some of these cases we are in the present day in danger of pushing the recommendation of spectacles to an excess. Some slight degree of hypermetropia may be detected, and it is assumed to explain the irritability of the eye. Yet glasses do no good, and in fact only increase the irritation. In such cases, very often, the real malady is inherited gout.
ON THE RELATION OF CERTAIN

Transverse Calcareous Bands of the Cornea.

I must admit, in the commencement of our investigation, that little or no evidence exists as to the deposit of urate of soda in any of the structures of the eye. I doubt whether it has ever been proved in any single case, although several observers have suggested its presence. I trust, however that we are long past the stage of belief which regards such deposit as essential to a gouty inflammation. The nearest approach to proof of gout deposit has been made probably in the case of what are known as the transverse calcareous bands in the cornea. Sir William Bowman, in the year 1848, was, I believe, the first to publish cases of this kind, but the earliest case had been observed by Mr. Dixon. Valuable information respecting the disease has recently been collected and studied by Mr. Nettleship. I believe I may fairly sum up the present state of our knowledge by saying that no one has yet proved that the salt deposited is that of gout. It is usually calcareous. The disease occurs in persons very likely to suffer from gout, and sometimes with a gouty history, but we are not as yet justified in holding that this earthy deposit is in direct connection with that diathesis.

Arthritic Iritis.

It will be admitted, without dispute from anyone, that there are several forms of Iritis which are arthritic in their origin. In other words, they occur to those who are liable to attacks of inflammation of joints, and the inflammations of the eye occur under conditions similar to those which excite the inflammations of the joint. Both are in some cases prone to occur in an acute but transitory attack. Both are very apt to recur after periods of complete immunity, whilst in other cases both may assume a
chronic, persisting, and destructive form. With such features of similarity we are justified in assuming, as indeed is done by all authors, that they are due to the same causes. The problem which I have to consider tonight is whether these causes belong to gout or to the rheumatic class; whether they are sometimes the one and sometimes the other; and lastly, whether they are not in some cases of a hybrid or mixed nature. Permit me briefly to state some of the peculiarities which are to be observed in different types of arthritic iritis; and to ask respecting each what evidence we possess which would connect them with the tendency to gout. Let us take first the common form in which the iritis occurs in acute transitory paroxysms. Of this, good typical examples will be in the memory of us all. Its subjects are usually men, and often of vigorous health, and past middle life. The iritis scarcely ever affects both eyes at the same time, but occurs first to one and then to the other, sometimes keeping to the same eye during several successive attacks and then leaving it, to assail its fellow. The attacks are often acute, beginning very suddenly, and attended with great pain. When they subside they subside completely, and leave the eye without the slightest degree of irritability. The duration of the severity of an attack may vary greatly at different times in the same patient. The tendency to form adhesions is extremely great. It is very remarkable how, in most cases of this class, the disease restricts itself to the iris and shows no tendency to disorganise the globe. Thus a patient in whom, in consequence of repeated attacks, the pupils may be occluded, with the exception of mere pin-hole apertures, may yet continue to enjoy very fair sight. I by no means wish to imply that this is usually the case. The case of Dr. Curry, as recorded by himself in the 'Medico-Chirurgical Transactions,' is a good type-form of this disease. Dr. Curry had suffered much from true gout. I may cite, however, as another good type-illustration of this class, that of a gentleman whose friendship I had the privilege to enjoy during a long period of
my early life. He was a florid, fair-complexioned man, of vigorous habits, a large eater, and through life accustomed to drink beer. He inherited rheumatism, but there was no known history of gout. He had suffered in boyhood in consequence, as he believed, of too free bathing when hot, from rheumatic fever. When I knew him first he was about five and forty, and from that time onwards to his death, at the age of sixty, he had a variety of arthritic complaints—lumbago, sciatica, chronic arthritis of the knees and other joints, and recurrent iritis. Both his pupils were closed with the exception of very small apertures, through which, however, he still continued to see enough to follow an occupation in which everything depended on good sight. I attended this gentleman year after year, usually in the early part of February, for most severe attacks of iritis, never affecting both eyes at once. He was benefited by large doses of iodide of potassium, and the free use of blisters. At length his attacks ceased to occur, and during the ten years preceding his death I believe he had not had any. I made a post-mortem examination of his body, and found no proof of gout. His knees contained loose cartilages, and there were patches where the articular cartilage was wearing away, but there was no urate of soda.

I have the record of a considerable number of cases in which the liability to these recurrent forms of iritis occur in those who were the subjects of unequivocal gout. But there were a far larger number in whom the concomitant symptoms were of rheumatic arthritis and not of true gout. The question remains, however, whether this form of rheumatic gout, although unattended by lithate of soda deposit, be not in reality a hybrid disease possessing, in addition to an unquestionable share of rheumatism, an admixture also of gout tendency. The infrequency of the disease in women, the age, the habit of body, and the mode of life of those who are usually its subjects, seem to me to strongly support this view. So does also the extreme severity of the attacks, and their rapid and
complete subsidence when once the point is turned. When this form of iritis does occur to women it is never, I think, so acute or so paroxysmal as in men; and it almost always happens to those whose relatives have suffered from gout.

**V**arious **F**orms of **A**rthritic **I**ritis.

Next to this form of recurrent iritis, and including perhaps some of its examples, I have to mention the iritis which occurs in connection with gonorrhœal rheumatism. All observers will admit, I think, that iritis associated with ordinary rheumatic fever is infinitely rare. So also is it in connection with that type of arthritis to which the name of crippling rheumatism may be given. In other words, iritis in association with either acute or chronic rheumatism is a thing that we scarcely ever observe. In men, however, the rheumatic affections, whether acute or chronic, which are induced by gonorrhœa are not unfrequently accompanied by iritis. This iritis is less distinctly paroxysmal and much more liable to persist and be destructive than is the case in the form which I have just described. My explanation of this is easy. The reason why gonorrhœal rheumatism so often causes iritis is because it occurs usually to the subjects of inherited gout.

A third form of arthritic iritis is one which affects women more frequently than men, which not unfrequently attacks both eyes at once, which is apt to spread to the ciliary region and choroid; to persist and to prove destructive. In a very considerable proportion of the women who suffer from this form there is a history of gout in former generations.

Another group of arthritic iritis might be constituted of cases in which the malady happens to young patients, and proves but slightly, if at all, liable to recur. These single attacks of iritis without history of gonorrhœa or syphilis, and occurring in young persons, usually, I think,
affect the male sex and almost always there is the history of gout in relatives.

I must just mention, in order to complete my classification, although it is but of little clinical importance, the group of cases in which iritis occurs in association with glycosuria. In these, according to my experience, the patient is almost always the subject of gout also.

To sum up then, I would say, that although in many individual cases of arthritic iritis there may be no proof of liability to gout in either the patient or his relatives, yet the tenor of the evidence in general is in favour of the conclusion that when iritis occurs there is in reality some gout complication. The more purely and definitely is the case one of rheumatism the less the probability that iritis will happen. When iritis occurs, the complications are almost invariably those which suggest what we call rheumatic gout, rather than rheumatism pure and simple; thus the smaller joints are often affected and nodi digitorum present, whilst sciatica, lumbago, and neuralgia are frequent complications. I have elsewhere tried to prove that gonorrhoeal rheumatism occurs, in nine cases out of ten, to those who inherit a gouty constitution and that it is chiefly this inheritance that gives the proclivity to it. I cannot stop now to recapitulate the evidence on which this belief rests, but if it be trustworthy it offers an explanation of the fact that iritis is so frequently met with in association with this type of rheumatism.

In certain cases of iritis with arthritic associations a very peculiar condition is observed. I allude to the filling of the anterior chamber with a soft, gelatinous, jelly-like mass. This material, which produces an appearance most alarming to the uninitiated, concealing the pupil and suggesting entire opacity of the cornea, is susceptible of very rapid absorption, and may, in the course of a very few hours, clear right away. I believe that in some cases of syphilitic iritis this peculiar form of effusion has been noticed, but if I were to speak from my own experience I should cite it as a symptom very strongly indicative
DISEASES OF THE EYE TO GOUT. 13

of gout. I would make the same remark, though with less confidence, respecting the rare cases of iritis complicated with haemorrhage.

Statistics as regards Arthritic Iritis.

My report on arthritic iritis, published in the 'Ophthalmic Journal' of 1872–74, contains the narrative of 104 cases which are suitable for our present purpose; for various reasons I omit a few on account of doubts as to their nature or imperfections as to the details. The report comprises cases of all varieties in which either rheumatism or gout, or the two together, were supposed to have been the causes of inflammation of the eye. They were collected from my note-books, both from hospital and private practice, up to the date mentioned. It is right to state that at that time my opinions as to the influence of true gout were less definite than they are at present, and that my knowledge as to the means of recognising it was also less. The notes of many of the cases had been taken some years before they were published, and thus certain inquiries which I should now carefully make were unfortunately omitted. On this account I have no doubt that we must consider that the report does not show the influence of gout so strongly as it would have done had the cases been taken with better knowledge. Out of the 104 cases I find a positive history of gout having occurred in the patient in only eighteen cases, and only in twenty-nine was there the history of positive gout in near relatives. It is to be observed, however, that these numbers take cognizance only of what has been termed "unequivocal gout," namely, of those in which an unmistakable attack of inflammation in the great toe or other single joint had unquestionably occurred. It omits altogether the much larger number in which the gouty tendency had remained undeclared, or had been manifested only in what we may
ON THE RELATION OF CERTAIN

term its quiet form. Anyone who will examine for himself the narratives to which I refer, will, I feel sure, agree with me in this opinion. In a very large proportion there was, I think, good reason for believing that a taint of gout existed where the narrative mentions rheumatism only; the arthritis had constantly been of the chronic type, and had very frequently presented exceptional conditions. If we may count such maladies as sciatica, lumbago, neuralgia, nodi digitorum, gonorrhoeal rheumatism, rheumatic gout when affecting the smaller joints, as being for the most part indications of gout rather than of pure rheumatism, we shall then find that facts at any rate suspicious of gout are present in nearly all the cases. It is, I think, decidedly exceptional for rheumatism pure and uncomplicated to show any tendency to attack the tissues of the eye. Iritis in conjunction with rheumatic fever of the ordinary form is almost unknown, and even in cases in which patients suffer from repeated attacks, as is sometimes seen, the eyes usually remain free. So soon, however, as changes of type occur, the disease showing a tendency to attack the smaller joints, or to affect only single joints, or to pass into the chronic form; then we get the liability on the part of the eye to suffer. On this point my own experience is quite in accordance with that previously expressed by an acute and zealous clinical observer, the late Dr. Fuller. Dr. Fuller in his first edition, speaking of rheumatic gout, stated that in 11 out of 101 cases which he had tabulated when Medical Registrar to St. George's Hospital, the eye had suffered more or less severely, and in 14 out of 193 cases which had subsequently been under his own care in the hospital. In a second edition he stated that he had so far modified or developed that opinion that he had come to believe that many, if not all, of the cases in which this complication occurred, were examples of obscure gout or else of gonorrhoeal rheumatism, adding, "This at least is certain, that since my attention has been specially directed to this
question, I have been enabled to find a "gouty or venereal (meaning, I think, gonorrhoeal) taint in every case in which the eye has been inflamed in connection with presumed rheumatic gout." This is strong testimony, stronger certainly than I am prepared to give, but I doubt whether it much exceeds the truth. Among other authors who have distinctly recognised gout as a cause of diseases of the eye, and appear to have referred a considerable proportion of those diseases, when arthritic, to gout rather than to rheumatism, I may mention Dr. Jacob, Mr. Weller, Mr. Middlemore, Dr. Copland, and perhaps Mr. Wardrop. Mr. Middlemore goes so far as to think that he has seen in one or two cases the pupil occupied by calcareous concretion of a gouty nature. None of these observers have, I think, recognised any other special disease as connected with gout than recurrent iritis. Almost invariably I find scleroticitis put down as rheumatic.

**Insidious and Destructive Iritis from Inherited Gout.**

I must now enter upon one of the most important divisions of my lecture. It is the attempt to prove that there is a very peculiar form of destructive iritis, occurring for the most part in young persons, which stands in all cases in direct relation with the inheritance of a gouty constitution. It does not occur to those who themselves suffer from attacks of gout, but to their descendants.

It was in 1863 that I first saw, at Moorfields, a girl named Mabey. She was then about eighteen, tall, well-formed and florid. She was too florid, and the circumscribed areas of colour in her cheeks varied in tint from bright red to slightly livid, according to the coldness of the day. She was the subject at the same time of a most peculiar form of arthritis of the last joints of all her
fingers, and of double iritis. Her right eye was lost and painful and I accordingly excised it. Her left was saved only by repeated iridectomies and finally by extraction of the lens. After the last operation she continued for five years to enjoy good sight and was free from relapses. Her fingers also got well. At the age of twenty-six she became the subject of phthisis and died, I believe, at about thirty. I showed this patient at the Hunterian Society, and drew attention to the unusual features of her disease. Someone present, looking at her hands, remarked, "Surely this is gout." I took the hint, inquired into her family history and found that in all probability it was gout. Her father had suffered repeatedly from that malady in an unequivocal form, and he had tophi in his ears. I show you drawings of the state of the girl's fingers. In order to ascertain whether there were urate deposits in connection with the swellings, I cut into one of the largest and obtained only a soft jelly-like substance. When, after some years, the swelling subsided, the terminal joints were all left disorganised and the last phalanges were more or less displaced. There appeared reason to believe that a feeble state of the circulation combined in this case with the gout inheritance.

I have repeatedly mentioned Mabey's case in public and with it others which came under my notice subsequently and I must content myself on the present occasion with a short summary of the facts. In 1872 I gave a lecture at Moorfields on this disease and was able to cite four well-marked examples of it. In 1880 I gave, at the London Hospital, a second lecture, in which I adduced seven additional cases, making eleven in all. Of these eleven patients four were females and seven males, but it happened that the most typical and severe forms of disease occurred in females, a fact which my subsequent experience has fully confirmed. In all cases both eyes ultimately suffered, but only in one were both affected simultaneously. In three instances "last-joint arthritis,"
that is to say destructive inflammation of the last joints of the digits, occurred. In all the cases vitreous opacities as well as iritic adhesions were developed. In most of the cases the eye which was last attacked suffered most, and a decided tendency was shown for the disease to come to an end as the patient advanced in life. In several cases the eye was saved by repeated iridectomies, and in one or two change of climate seemed to be very beneficial.

In all the cases but one the family history of gout was clear and strong, and in the exception the patient was the son of a brewer’s man, and his father had died early, so that it was very possible that his proclivities had not declared themselves. In several of the cases the patient had been reduced almost to blindness. In three one eye had been excised and in one both were quite lost. In all excepting two the disease had begun between puberty and the age of twenty-five, and it would appear to be the fact that the earlier it begins the more severe it is. Such then was the state of my facts in 1882 when I put my last lecture into print. I think you will allow that I was justified in avowing a strong belief that inherited gout was the real cause of this peculiar form of iritis.

That I may add a little more colour to my picture I will ask your permission to state briefly the facts of three cases which have come under my notice since the date referred to. They are all cases of great interest and two of them illustrate a fact which I had previously only once or twice noticed, namely, that there is a tendency to the formation of cataract.

The last case which I have seen is that of a Miss D—, æt. 30, the daughter of a surgeon in the country. She was brought to me with the left eye quite destroyed, and the right nearly so. In the left an opaque lens had undergone spontaneous absorption and the iris was everywhere adherent to the opaque capsule. In the other the lens was half opaque and the iris extensively adherent. I was told that I had myself seen this patient ten years
before and had then recognised only commencing cataract in both. The patient's father, an intelligent medical man, was astonished when I told him there had been extensive iritis, and assured me that his daughter had never had any attacks of inflammation. The patient herself said that she had never noticed more than that the eyes had sometimes been red and hot and would prick a little. We have here then a good instance of the very insidious course of the disease. In the left eye there was no perception of light, and very probably the vitreous was affected. I found on inquiry that in Miss D—'s family there had been much gout. She herself appeared to be in good health.

Mrs. O— is a young married lady who has borne three children, and during lactation in each instance suffered from a chronic form of almost painless iritis. Both pupils are almost excluded. Her father and several other near relatives have suffered from gout. She is of feeble circulation and liable to chilblains.

I have kept one of my best cases to the last. Miss L—, at. 22, is the youngest of a family of eight, and was born after the death of a father who had suffered much from "chalk gout." Her eldest brother has had both gout and rheumatism, and there is rheumatism also on her mother's side. Miss L— began to suffer in her eyes at the age of twelve, and at first it was simply redness and irritability with "black specks," "balls," and mist. At the age of fourteen she had a severe attack in both and was several months under Mr. L—'s care "almost blind." From this she recovered, but at eighteen a surgeon in Liverpool performed an iridectomy on the left on account of exclusion of the pupil. A little later Mr. C— did an iridectomy in the right. Neither of these operations resulted in any improvement of sight. In 1880 a surgeon in Leeds removed a soft cataract. The eye was lost and Mr. C— excised it a few months later. Miss L—'s present condition is that with the one remaining eye she can just puzzle out $\frac{20}{200}$. 
DISEASES OF THE EYE TO GOUT.

Could I possibly produce before you a more marked example of an insidious destructive disease, progressing in spite of the most highly skilled assistance to its melancholy end? Yet it is only a fair example of about half those included in my series.

Relapsing Cyclitis.

There is a peculiar form of chronic inflammation of certain parts of the eyeball which clinical observers have recognised under the name of *Relapsing Cyclitis*. It is a cyclo-kerato-iritis involving the ciliary region of the sclerotic, the adjacent part of the cornea, and the iris. Sometimes one of these structures suffers more than another, but usually all are involved. It seldom damages the pupil itself much, seldom invades the centre of the cornea, or only very late in the disease, and it does not show much tendency to involve the choroid or vitreous. It usually begins in one eye and only affects the other after a long interval, and to the last one usually suffers more severely than the other. It may begin in early life, but often does not do so till middle periods. When once it has begun it never wholly leaves its victim, but continues either to persist with slow chronicity or recurs over and over again after intervals of health. It causes scars in the ciliary part of the cornea, thinning and discolouration of the ciliary region of the sclerotic, and ends either by inducing staphyloma or by making the whole cornea dull. It may occasionally become needful to excise the eyeball on account of the persisting irritability combined with great impairment. It affects, I think, women more frequently than men. It is a very peculiar type of disease, and it would not be difficult to place side by side a group of examples of it all exactly alike. I know of no treatment short of a complete change of climate which does much to benefit it. In this respect it is much like the form of iritis which I have been striving to prove to be a
direct result of inherited gout. Yet I am by no means in a position to produce before you, respecting relapsing cyclitis, such evidence in reference to its gout origin as that which we have been discussing. I dare not, indeed, do more than suggest that it is very probable that in some cases the constitutional cause of this destructive and persisting disease may be gout. I have found a gout history in some of my cases, but in others it has been absent. The last case which I have seen has been the one in which this suspicion was the strongest.

Miss D— is the daughter of a medical friend. She is now forty, and she has suffered all her life from her eyes. She was treated when two years old by Mr. McMurdo for what was called strumous ophthalmia, and which proved very intractable. Since then she has had numberless relapses, and in both eyes the cornea at its edges has become extensively opaque. Her left eye is the worst, and has recently been so troublesome that I was inclined to recommend its excision. It is by no means a blind eye, but the cornea is so hazy that it is of little use. Now, say that we have here a case in which strumous ophthalmia has persisted through life, let us ask what is the constitutional peculiarity which has conduced to this exceptional result. The lady has been well cared for all her life, she shows no other signs of struma, nor are such present in any of her relatives. Her father is a robust man. Mark that both eyes have suffered, and that in both it is the peripheral parts of the cornea which have been chiefly involved. Now, this lady's maternal grandfather and two of her maternal uncles suffered severely from gout, her mother had an attack in her great toe, and even she herself has on one occasion had an attack of it. This strong gout history is certainly by far the most definite fact which I can obtain as explaining the peculiarities of her eye disease.
Can Gout cause Neuritis?

Although from time to time strong suspicions have been expressed as to the occurrence of gouty affections of the nervous system, I believe the question has seldom been definitely put, "Does gout cause neuritis?" We know that gout can cause the tissues of a joint to inflame, and that òedematous effusion with cell proliferation, in fact the ordinary phenomena of inflammation, are its results. I shall be much disappointed also if it is not regarded as proved that it can cause the tissue of the iris to inflame in a precisely similar manner and with like results. Can it attack any of the structures of which the nervous system is composed? Can it cause inflammation of ganglia, of nerve-trunks, or of their investments, or may it indeed attack the central organs? Without venturing to suggest that any of these occurrences are common, it is yet difficult to deny the possibility or even probability in exceptional cases. We can understand the influences that are likely to localise gout; its exciting causes may come into play far less frequently in reference to the nervous system than to the joints; or even to such an apparently exposed part of the organism as the eye. Still, if we admit that, in the gouty condition, inflammation may attack the cellular tissue in any part of the body, it is difficult to suppose that the cell elements which enter into the formation of nerve-trunks, for instance, will always escape. I am glad to note in the progress of neuro-pathology of the last few years that there seems an increasing tendency to recognise the possibility that not a few nervous affections may be due not to central disease, but to primary inflammation of the connecting trunks or of the peripheral organs. We know for certain that chronic neuritis, probably beginning peripherally, is a part of leprosy. It is highly probable that it is often a part of syphilis and quite possible that it occurs also in locomotor ataxy. How else explain many of the transitory forms of ptosis, &c., which occur in the two latter diseases?
GOUTY NEURITIS OF THE OPTIC NERVE.

The question which I now wish to put definitely is this: Are there any cases of inflammation of the optic nerve, or of any of the motor nerve-trunks of the eyeballs, the direct cause of which is the existence of a gouty constitution? The decision is of importance not only in reference to diseases of the eye, but because it would throw much light on certain other obscure affections met with in this disease. Is sciatica really a neuritis of the sciatic trunk or its sheath? Are any of the forms of gouty neuralgia really produced by gouty neuritis? If the optic nerve can inflame from gout why not the pneumogastric or the phrenic? I am sorry to say that I have exceedingly little evidence to bring forward in answer to these important questions. I will mention briefly one or two facts which are suggestive, and which I hope will prove sufficiently so to induce a more careful investigation of the subject. In two or three cases I have attended young ladies, of families in which gout had been prevalent, who suffered from attacks of ptosis, with the other group of symptoms referable to paralysis of the third nerve, for which I could find no other more plausible explanation than that they were caused by gouty neuritis. In one of these the attacks were transitory and recurrent, several having occurred within a period of a few years. I must not venture to trouble you with details of these cases, but feel myself obliged to speak rather more at length concerning one in which the optic nerve itself was inflamed, there being, I think, fair reason to suspect that the cause was inherited gout. A young lady of sixteen, was brought to me from Ipswich, on account of blindness of one eye, in July, 1879. She had no other symptoms whatever, was florid and appeared to be in good health. The ophthalmoscope showed nothing but what was quite normal, and had it not been that her pupil dilated more widely when the other was covered, I might have suspected
DISEASES OF THE EYE TO GOUT.

that she was feigning. A week later the changes were conspicuous, the disc being much swollen and its margin concealed. Under treatment by mercury combined with quinine, in the course of a few weeks the attack had passed away, but the disc was left pale. Four years later I saw this patient again with similar symptoms in the other eye, and I was now told that she had several times suffered from transitory attacks which always occurred during spring. Her health remained perfect. There was a strong history of gout in the family, both her parents having suffered repeatedly. It seems to me not at all improbable that these recurrent attacks of transitory neuritis have been really of a gouty type. In saying this, I have regard to the entire absence of the more ordinary causes and concomitants of optic neuritis, to the recurrent nature of the affection, its want of symmetry, and the exceptionally strong family history, both parents and many other near relatives having suffered. I cannot call to mind any other case of recurring optic neuritis, with gout history, which I could at all fairly place side by side with this one. I have, however, several times seen optic neuritis occur in women who were of gouty family without any of its ordinary accompaniments and clear away entirely under treatment. In none of the cases of which I am thinking did any recurrence take place, and in most of them both eyes were simultaneously attacked.

Other Forms of Gouty Neuritis.

Before quite taking leave of this important question as to the possible occurrence of gouty neuritis, I must trouble you with yet one other case. A lady of fifty-six came under my observation in whom for several weeks the left facial nerve had been quite paralysed. I observed at once that she had had two large iridectomies done downwards and outwards, and on inquiry found that she was nearly blind. It was twenty-three years
since she had first noticed cobwebs before her sight, and fifteen since she had last been able to read. The iridectomies had been done six years, with what object I do not know, for although she said that she had once had rheumatic inflammation of her eyes, there did not appear to be any adhesions. In each eye the lens was partially opaque, and in each there were extensive choroido-retinal changes with pigment deposits and waxy atrophy of the discs (a peculiar form of retinitis pigmentosa). Mrs. H—told me that she had a sister who also had "amaurosis." I persuaded her to bring her sister, and may now state the family history which belongs to them both. One of their brothers suffers not unfrequently from attacks of unequivocal gout, and a sister is crippled by rheumatism, but their parents died aged, and are not known to have suffered. The elder of the sisters, aged sixty-nine, has herself had gout, and describes her great toes graphically as having been swollen and "red like tomatoes." This sister after her first confinement, aged twenty-seven, had an attack in her eyes which lasted several years attended by great pain and intolerance of light. Her recovery from it was very gradual, but finally it was almost perfect, and I could now find no material changes. For several years she has been very deaf and she has suffered severely from neuralgia and chronic arthritis of wrists and fingers. Once she was told by Mr. Critchett that she had "gout in the eyes." The younger sister describes ten years ago an attack of shoulder neuralgia attended by torticollis which kept her in bed six weeks and was accompanied by agonising pain, as if her arm were in the fire. She also has suffered most severely from neuralgia on many occasions.

Here then is a family so definitely gouty that a brother and sister have each had true gout and several other sisters are crippled by rheumatism; one sister has become almost blind with neuro-retinitis, and has suffered from neuralgia, torticollis, facial paralysis and an attack which was probably neuritis of the brachial plexus. Another sister is
deaf, has been all her life liable to neuralgia, had a several years' attack of pain in her eyes which was called "amaurosis," and in later life one which was named "gout in the eye." It seems highly probable that we have here an instance of gout affecting, at different periods of life in each sister, different parts of the nervous system, and attended by true neuritis of various nerve structures.

**Glaucoma in Relation with Gout.**

I had intended to have said a good deal as to the nature of glaucoma and the evidence as to its connection with gout. The subject is, however, too important to be discussed hurriedly, and I have not time to attempt to do it justice. I must therefore content myself with the mere expression of opinion that in the production of this extraordinary disease a gouty tendency often takes a large share. More particularly would I suggest this in the exceptional cases in which glaucoma shows itself in early life, and I could, if time permitted, bring before you some important cases illustrating this point.

**Retinitis Hæmorrhagica and its Connection with Gout.**

There is yet another definite and well-characterised affection of the eye which claims conspicuous mention in connection with our subject.

*Retinitis hæmorrhagica* is very rarely indeed seen excepting in those who are themselves gouty. It is a disease of middle life or of advancing years, and in its more typical forms is never seen in the young. It seldom happens to both eyes at once. It is attended by great swelling of the disc and adjacent parts of retina and by such turgidity of the central vein that I was at one time tempted to believe that it depended primarily upon throm-
botic phlebitis of that vessel. It is possible, however, that such is not its true pathology, and that the venous distension and stasis, perhaps in some instances thrombosis, are really secondary to the neuritis. If this be the fact and neuro-retinitis be the primary condition, then we have in retinitis haemorrhagica an instance in proof that acquired or humoral gout may become the cause of neuritis. It is not, I think, ever seen in association with the inheritance only of a gouty constitution, but is found usually with lithæmia, in free livers who have experienced unequivocal attacks. The case which Jaeger took for his beautiful plate in illustration of the disease, was that of a tavern keeper of plethoric habit and bloated appearance. Nothing is said as to gout, but it is fair, in such a man, to assume that there was a tendency to it. In order to determine statistically, as far as practicable, the association of retinitis haemorrhagica with gout, I have tabulated twenty-four cases—with the exception of the one of Jaeger's, all from my own note-books. In twelve of these, exactly one half, the patient had suffered definite attacks of gout, and in five others there was strong presumptive evidence of a gouty constitution. In seven there was no proof of gout. This last group comprises two in which diabetes was present; one in which albuminuria existed, and two in which the retinitis was not very well characterised as of the haemorrhagic group. If I had kept more closely to the type of cases illustrated by Jaeger's portrait, I should have been able to make yet stronger statements as to the almost invariable association of the disease with gout. It may be of interest to state a few other facts deduced from my table. Thirteen of the patients were men and eleven women. The youngest was forty-five. In seventeen cases only one eye was affected, and in seven both. In some cases there were haemorrhages only, with little, if any, evidence of neuro-retinitis.
SUMMARY.

I must now bring my lecture to a close, and in doing so may, I think, venture to assume that it has been shown to be probable that there are many different forms of inflammation of the eye, or of parts of it, which are in connection with gout. Some of these are very peculiar and specialised types of disease, and have already been accorded distinctive clinical names; others quite as distinct are not as yet so well known, and of others we may say that they are to be distinguished from other inflammations of the same structures not so much by their features as by their cause. Of all we may assert that they are infrequent; some, if we confine ourselves to well-marked types, are distinctly rare. We have divided these different affections into two groups: (1) those who go with acquired, humoral, or renal gout, and (2) those which depend upon the inheritance of structures damaged, or at any rate specialised, by gout in predecessors. It is needless to repeat that in almost all cases of acquired gout there is inheritance also, and that in many in which the disease is chiefly caused by inheritance, some modification and increase may have been derived from personal habits. Still, the difference between the two classes of affections is very marked. In the one attacks of a transitory nature are the rule, and these attacks are often acute and attended by much pain. In the second group, although a tendency to temporary recovery and recurrence is often observed, yet there is a great proneness to chronicity and persistence. The invasion is often insidious, but the disease is usually in the end destructive. In the former group we have placed hot eye, scleritis, recurrent iritis, and retinitis haemorrhagica. All these are diseases of adult life. In the second group we have insidious disorganising iritis, relapsing cyclitis, certain forms of soft cataract, and perhaps some of primary optic neuritis. Not only are there clearly marked clinical differences between the two
classes of affections, but the difference in treatment is equally marked. In the first, the well-known measures against gout must be taken, a restricted regimen, alkalies, colchicum, aconite, and liberal counter-irritation. In the second we must use tonics, and although counter-irritants are here also often valuable, we cannot trust to any measure as really curative short of complete change of climate.

**On the Proofs of Gout.**

It may be, perhaps, convenient to say a few words as to the kind of evidence which justifies a diagnosis of gout as the cause of any particular disease of the eye. In the case of humoral or acquired gout there ought to be the history of one or more definite attacks of joint inflammation usually of an acute character, and attended by redness and oedema, and followed by peeling; usually the great toe will have been the joint affected. Such patients will often state that they are very susceptible to the influence of beer and wine, and that malt liquor and some wines almost always cause indigestion, and make the urine muddy. These dietetic disturbances, to which as a test of gout attention was, I think, first claimed by Sir James Paget, are very important and valuable. If tophi are present in the ears or elsewhere they are of course conclusive. In a few cases we are justified in assuming the existence of humoral gout, although no paroxysm has ever occurred. If the dyspepsia be there, if the joints ache and prick after beer or wine, and if there be gout in relatives, we may confidently believe that it is present, although not yet declared. As regards the inherited form, we may take it as highly probable whenever parents or grandparents, or any one of them are known to have suffered definitely. If even uncles, aunts, brothers, or sisters, or cousins have suffered from true gout in early life, the belief that a family taint exists becomes very probable. The evidence must always be carefully sifted.
It will not do to take the statement of the patient without first carefully informing him as to the scope of the inquiry. Patients will often confess to gout who do not know what the word means, and a far more numerous class will hastily deny its history, although the facts, when correctly obtained, may be most conclusive. If, however, proper care be taken, and the patient, after being instructed, be allowed time for consideration—above all, if the inquiry be repeated after an interval, or if several relatives be interrogated, then I believe that in most cases truthful data will be obtainable.

It may be inquired as to the value of certain affections which may be considered to belong both to rheumatism and gout, as symptoms of the latter. Permit me very briefly to repeat my creed. I believe that the subjects of gonorrheal rheumatism are in a very large majority of instances the inheritors of a gouty constitution, and that all the conditions usually classed as rheumatic gout are really, in most instances, dependent in a large degree upon like inheritance. Thus, if a patient has had sciatica or lumbago, if he shows nodi digitorum (osseous, not tophi), if he has suffered from chronic rheumatism affecting the smaller joints, I should think it fair to allow considerable weight to these facts as pointing to a taint of gout. Even the strictly rheumatic disorders, acute rheumatism itself, if it happens to the relatives of those who have had gout, lends support to the theory of family tendency to gout. It is an observation as old as the days of Heberden, and confirmed I am sure by daily experience, that the children of the gouty are more liable than others to attacks of rheumatic fever. In this we see another proof of the inheritance of structural proclivities, rather, I think, than of blood disorder or tendency to it.

Conclusion.

And now, gentlemen, as my last word, I do not know whether I have to defend myself in the eyes of any of
you from the charge of "seeing gout in everything." I am well aware that this diagnosis is a very easy one, and seductively ready at hand for the idle prescriber. I submit, however, that it has not been exactly in that temper that I have brought before you the statements which I have made this evening. My desire has been to state the issues explicitly, and to keep close to facts. Where statistics were admissible and obtainable I have had recourse to them. I may assert that I have said nothing but what has been based not only upon clinical observation, but upon clinical note taking, and the subsequent collation of cases. Nor, I contend, have I made any very sweeping statements. So far from my having exceeded the truth, my conviction is that when our clinical pathology shall be more advanced, and diseases more minutely classified, we shall in all probability recognise as gouty yet other maladies, and perhaps not a few beyond what I have claimed. It is a subject upon which scepticism is as irrational as credulity. That the gouty constitution exists, and is very common in our English population, that it is potent in the production of disease, and that it is remarkably hereditary, are facts which no one will doubt. In relation to the multiform diseases of the eye it must have a domain, and that an important one. To discover some of the extensions and limits of that domain has been the object of my best endeavours to-night.
COMMUNICATION WITH THE GOVERNMENT

ON THE

PREVENTION OF BLINDNESS FROM
OPHTHALMIA NEONATORUM.

The first of the resolutions adopted at the meeting of the Society in June, 1884, reads as follows, being almost precisely identical with that originally proposed by Dr. D. McKeown—

"That the purulent ophthalmia of newborn infants being the cause of a vast amount of blindness, mainly because of the ignorance of the public regarding its dangerous character, and the consequent neglect to apply for timely medical aid, it is desirable to instruct those in charge of newborn children by a card, in substance as follows:

"Instructions regarding newborn infants.—If the child's eyelids become red and swollen, or begin to run with matter, within a few days after birth, it is to be taken without a day's delay to a doctor. The disease is very dangerous, and, if not at once treated, may destroy the sight of both eyes.

This card to be distributed through the medium of the Poor-law and Birth Registration organisations of the United Kingdom. In England the Relieving Officer, and in Scotland the Inspector of the Poor should, in every case
of labour under the Poor-law system, read to and leave with the person obtaining the order for medical aid, or the persons in charge of the patient, a copy of the card. In Ireland the card should be attached to the order for medical aid in such cases, and the person who gives the order and card should, before doing so, read the card to the applicant. The Registrar of births should read and hand to each person registering a birth a copy of the card—

This resolution was forwarded to the respective Presidents of the Local Government Board of England and Ireland, and of the Board of Supervisors in Scotland.

In reply to it the following communication addressed from the Local Government Board of Ireland to the Secretaries of the Society, was read at the October Meeting, 1884:

"Dublin Castle,
25th September, 1884.

Gentlemen,—With reference to your letters of the 18th July last, and accompanying resolution adopted by the Ophthalmological Society, I am directed by the Lord-Lieutenant to transmit to you herewith, for the information of the Society, copies of circulars which have been issued by the Local Government Board in Ireland on the subject of ophthalmia of newborn infants.

I am, Gentlemen,
Your obedient servant,
The Hon. Secretaries of the Ophthalmological Society,
16, Orchard Street,
London."

The circular sent to the medical officer of each dispensary district is as follows:

"Local Government Board, Dublin,
16th September, 1884.

Sir,—The Local Government Board for Ireland desire to acquaint you that a Committee of the Ophthalmological
Society have recently reported, as the result of inquiries they have made, that there is reason to suppose, so far as they have been able to ascertain, that 30 per cent. of all blind persons in the United Kingdom have lost their sight from the results of the ophthalmia of newborn infants.

The Local Government Board wish to call your attention to the subject, and to observe that this species of ophthalmia appears to be caused by acrid discharges coming into contact with the conjunctiva at the time of birth, or subsequently to result from the exposure of the newborn infant to cold.

The Local Government Board suggest that when you are in attendance on a woman in childbirth, you should be careful to see that the child’s eyes are carefully washed immediately after birth, and that you should give directions that the child shall be protected, as far as is practicable, from currents of cold air; it would also be desirable that the parents should be instructed that on the slightest appearance of inflammation indicated by red or swollen eyelids, the child ought to be brought to you for treatment without a moment’s delay, as the disease is most dangerous, and if proper treatment be not adopted the sight of one or both eyes may be lost.

By Order of the Board,

To W. D. Wodsworth, Secretary.

The Medical Officer of each Dispensary District.”

A copy of this circular was also enclosed to each midwife, together with the enclosed letter:

"Local Government Board, Dublin, 16th September, 1884.

Madam,—The Local Government Board for Ireland forward herewith for your information a copy of a circular which they have addressed to the Medical Officers of the several Dispensary Districts in Ireland on the subject of ophthalmia of newborn infants; and the Board request..."
that your attention may be given to the observance of the instructions contained in the circular.

By Order of the Board,
W. D. Wodsworth,
Secretary.
To
Each Midwife.''

Similarly each Board of Guardians had one enclosed to it with the following:

"Local Government Board, Dublin,
16th September, 1884.

Sir,—I am directed by the Local Government Board for Ireland to forward herewith for the information of the Board of Guardians, copies of circulars which they have addressed to the Medical Officers and Midwives of the several Dispensary Districts in Ireland on the subject of ophthalmia of newborn infants.

By Order of the Board,
To
W. D. Wodsworth,
Secretary of each Union.''

The Local Government Board in England subsequently sent the following reply, which was read to the Society at its January meeting, 1885:

"Local Government Board, Whitehall, S.W.,
15th December, 1884.

Gentlemen,—I am directed by the Local Government Board to advert to your letter in which, on behalf of the Ophthalmological Society, you submit certain suggestions with the view of checking ophthalmia in infants.

I am directed to state that the Board have been in communication with the Registrar-General as regards the proposal which concerns the Registrars of Births, and have received from him a reply, a copy of which is transmitted herewith for the information of the Ophthalmological Society.
With respect to the suggestion as to the Relieving Officers, I am directed to state that the Board, while fully recognising the importance of the object which the Society have in view, do not consider that they can impose on those officers the duty which the Society propose should be assigned to them.

I am directed at the same time to express the regret of the Board that they are unable to adopt the suggestion of the Society.

I am, Gentlemen,
Your obedient servant,

To W. A. Brailey, Esq.,
John Abercrombie, Esq.,
The Honorary Secretaries of the
Ophthalmological Society,
16, Orchard Street,
Portman Square, W.''

The enclosed copy of the Registrar-General's letter therein referred to, read as follows:

"July 24th, 1884.

Sir,—I beg to acknowledge the receipt of your letter of the 11th instant (62,327a), enclosing a copy of a resolution passed by the Ophthalmological Society, and requesting me to forward any observations I may have to make on the practicability of the suggestions made by that Society.

The only point in the resolution passed by the Society which at all concerns the Registrar-General is the recommendation that the Registrars of Births "should read and hand to each person registering a birth a copy of the card," which that Society wishes to have printed and distributed.

This would give considerable trouble to the Registrars; and they would most certainly, and not unreasonably, demand to be paid for the service which does not form part of their recognised duties. Say that the Registrar was paid no more than twopence for each card which he
read and delivered, the cost in payment to Registrars would nevertheless amount to no less than £7334 a year, as there are some 880,000 births annually registered.

Again, the person who comes to register a birth at the Registrar's office is by no means necessarily the mother, who will afterwards be responsible for the infant's management. The Registrar would therefore frequently read the card to a person who will have nothing to do with the care of the child's health.

Neither must it be forgotten that purulent ophthalmia is by no means the only serious affection to which children are liable to fall victims owing to the ignorance of their mothers. If the Registrar of Births be employed to give directions as to the mode of avoiding this disease, there seems no reason why he should not equally be called on to lead out and to distribute directions as to all other ailments to which infants are liable from their mothers' ignorance or carelessness, and in short to give each mother a discourse on the proper management of a child's health.

The above considerations lead me to the conclusion that the proposal made by the Ophthalmological Society is not a practicable one.

I am, Sir,

Your obedient servant,

(Sd.) Brydges P. Henniker,

To The Secretary,

Registrar-General.
Local Government Board, W."

Thereupon the Society appointed the deputation nominated at the meeting on June 5th, 1884, as a special committee to consider and report upon the above communication. Their report was read at the March meeting, 1885, as follows:

"That, as in the opinion of the Registrar-General the reading over of a printed form by the Registrars of Births to the parents would entail a considerable expense, this may be dispensed with; and that, in place of this reading,
the following notice be printed on all official documents issued to parents in relation to the birth, registration, and vaccination of children, namely:

**Instructions regarding Newborn Infants.**

If the child’s eyelids become red and swollen, or begin to run with matter within a few days after birth, it is to be taken, without a day’s delay, to a doctor. The disease is very dangerous and, if not at once treated, may destroy the sight of both eyes.

(Signed) Jonathan Hutchinson.
John Tweedy.
W. A. Brailey.
John Abercrombie."

This report was unanimously adopted by the Society, and ordered to be presented to the Local Government Board by means of the deputation already appointed.

Consequently, on May 15th, 1885, Mr. Jonathan Hutchinson, F.R.S., Sir Wm. Bowman, Bart., F.R.S., Mr. Tweedy, Dr. D. McKeown, and Drs. Brailey and Abercrombie (Secretaries), interviewed Mr. George Russell, M.P., by appointment, Dr. Buchanan and the Registrar-General being also present.

The subject was introduced by the President of the Society, following whom Sir Wm. Bowman pointed out in a short but earnest and impressive speech that this was a disease which attacked infants during the first few days of life, and which, if taken in time, was almost invariably curable, whereas, when left to run its course, it led, in a large number of cases, to complete and irremediable blindness, and in many more to permanent damage of one or impairment of both eyes. The circumstances of its coming on in infants of so tender an age seemed to the Society to justify the Government in giving State aid to prevent a disease which caused so much suffering and harm to a large section of the community. It had been ascertained that about 30 per cent. of the inmates of the
blind asylums had lost their sight in this way, and from an approximate computation it had been ascertained that there were at least 7000 persons in the United Kingdom blind from this cause at present, and to these an equal number might be added to represent those suffering from the effects of the malady in less severe forms. In answer to a suggestion from Mr. Russell, that if the Government interfered in regard to one disease of infancy they might be subsequently called upon to adopt measures against others, Sir William Bowman pointed out that this disease ought to be ranked quite apart from all others owing to the peculiar circumstances of its origin, and he did not think that any other disease could be legitimately compared with it in reference to its claim for State aid. Mr. Hutchinson followed, pleading the very great anxiety the Society felt that some measures should be taken to prevent the dire effects of this eminently remediable complaint. As the disease was one which occurred mainly amongst the poor, the Society thought that an attempt might be made to check it through the agency of the Poor-law organisations throughout the kingdom. With this object they suggested that to every applicant for parish relief at the time of her confinement, with the order issued by the Relieving Officer for medical attendance in childbirth there should be given a printed slip containing the following words:—"If the child’s lids become red and begin to run with matter within a few days after birth it is to be taken without a day’s delay to a doctor. The disease is very dangerous, and if not at once treated may destroy the sight of both eyes." This might be given as a separate slip or printed on the actual order for the attendance of the doctor. This slip should also be given to every midwife attending parish cases, and a notice that such slips had been distributed should be sent to the Poor-law medical officers. But, inasmuch as these measures would only reach a small section of the poor owing to the fact that many are attended in their confinement by midwives not connected with the parish authori-
ties, and inasmuch as there was no system of certification of such midwives in existence, it was desirable that some further steps should be taken. The Society therefore suggested that the Registrars of Births should, when giving the certificate of vaccination to the person who registered the birth of a child, at the same time give a printed slip containing the above notice. The warning would be received too late the Society knew for that particular child, but the parents might bear it in mind in the case of any subsequent child, and at any rate a knowledge of the first symptoms and dangerous character of the disease would thus be gradually diffused throughout the country, which could not but tend to have a beneficial effect. The Society did not wish to do more than make these general suggestions, and would be glad to leave the details in the hands of those more competent to deal with such matters.

The Registrar-General, who was present, said he could not see his way to advising that the suggestions of the Society, so far as his own department was concerned, should be carried into effect. It was proposed to throw some additional work upon the Registrars of Births, but these officers already had their duties very clearly defined by Act of Parliament, and were paid accordingly; this work would therefore be distinctly an addition to their ordinary duties, for which there would be no remuneration; and seeing that the information was only to benefit some possible child in the future, he did not hold out any hope of giving effect to the proposals of the Society. Mr. Russell said that he would communicate the result of the interview to Sir Charles Dilke, and without consulting him he was unable to make any definite promise, but he stated that he was personally impressed with the desirability of taking some such steps as those suggested by the Society. He reminded the deputation, in concluding, that the Local Government Board had no power to order the Boards of Guardians to do anything, though they had always found that those bodies showed great readiness in
carrying out suggestions emanating from the Local Government Board for the benefit of the public.

The matter rests thus so far as the Society is concerned, no further steps having been taken, so far as they are aware, by the Local Government Board.
REPORTS.

I. DISEASES OF EYELIDS AND CONJUNCTIVA.

1. *Primary lupus of the conjunctiva.*

By ARTHUR H. BENSON, F.R.C.S.I. (Dublin).

The ten drawings exhibited were taken from six patients suffering from lupus of the conjunctiva. In three cases it was *primary*, there being no other patch of lupus over the body. In the others the lupus appeared in the eye as a separate patch of disease, but subsequently to the appearance of lupus of the mucous membrane of the mouth or nose; and in two of the cases there was also lupus ulceration of the external skin. In nearly all the cases there were evidences of a strumous diathesis, such as enlarged lymphatics in the neck and scars from suppurated glands, &c. In five of the cases the conjunctiva of the lower lid was affected; in two that of the upper lid, and in one the ocular conjunctiva, as well as that of the upper and lower eyelids, was affected with lupus.

The diagnosis was arrived at by a consideration of:

1. Its similarity of appearance to lupus of the gums and other mucous surfaces.
2. Its softness, its tendency to bleed, and its friability.
3. Its very slow growth (from one to five years).
4. Its well-defined limits and elevated, uneven surface.
5. Its very slight tendency to produce pannus or ulceration of the cornea.
6. Its being confined to one eye and usually to part of one eyelid.

7. The effect of treatment (scraping with the spoon and cauterisation with solid nitrate of silver).

Note.—In those cases where the disease was quite localised to one eyelid or to small patches on both, the treatment, after a variable time, proved successful; in the other cases relapses occurred.

(March 12th, 1885.)

2. Rapidly extending isolated patch of favus on adult upper eyelid, treated by excision.

By M. M. McHardy.

The patient, a woman, æt. 37, who was suckling an infant four months old, presented her left upper eyelid for treatment on February 3rd, 1885, on account of a very painful, dry, not bleeding, fast increasing scabbed ulcer, about 5 mm. across, at the external end of the upper lid some 4 or 5 mm. above the ciliary edge. There was neither induration nor specific history nor any enlargement of glands. No clue to the causation of the local trouble was obtained. The malady was stated to have been noticed as a fine, white speck of scab some two or three weeks previous.

Its appearance, notwithstanding its brief history, made me favour the opinion that it was an exceptionally behaving rodent ulcer.

Six days later, February 9th, the patch had doubled in area, was covered by a very thick, deeply pitted, dry, palish, yellowish-brown scab, and caused the patient so much pain that she slept very little.

During the next three days the patch increased 80 per cent. and did not alter in character. It was seen by
an eminent dermatologist, who shared my view that it ought to be thoroughly removed, and that it was an exceptional rodent ulcer.

It was seen by Sir Joseph Lister, who believed it to be favus. As it had now reached the edge of the lid I did not delay its removal, which was effected by first removing the entire scab, then disinfecting the surrounding skin, next excising the entire area of deeply ulcerated skin, and then using the actual cautery freely over the raw surface. The slough soon separated and uninterrupted cicatrization followed. Immediate relief was afforded by the operation, and no troublesome distortion of the lid has resulted.

The microscope immediately demonstrated that the disease was favus, hence that Sir Joseph Lister's diagnosis was correct.

Inquiry elicited that the patient's cat had died of an illness, that her house was over-run by mice, that dead mice had been found about the house with their heads covered with white scab, and that her boy had remarked, "Why, mother, you have on your eye the same as those dead mice."

The following questions are suggested by this case; viz:

Would any other treatment have been so effective and safe as extirpation, and if so, what other treatment, when the disease has so nearly reached the conjunctiva?

Is anything known of the behaviour and amenability to treatment of favus when it attacks the conjunctiva of human beings?

Was the exceptionally rapid extension of this single patch due to the tissue on which it was planted, and also to the then debilitated condition of the underfed, recently confined, suckling mother?

What other examples of single patches of favus confined to the eyelid are on record?

(\textit{Living specimen. March 12th, 1885.})
3. Fat developed in both upper eyelids.

By M. M. McHardy.

F. S—, a servant girl, aet. 16, of very singular facial appearance, but in excellent health, sought treatment on account of her upper eyelids, each of which, the left more than the right, was abnormally full, looked puffy, and posed (at its best) with a fold of skin drooping below the external two thirds of the ciliary margin.

The local condition strikingly resembled that due to oedema. The abnormal swelling was not painful; it was in no way tense, was very elastic, conveyed no sense of lobulation, and felt like emphysematous cellular tissue without the "crackling."

The urine was normal. The lacrimal gland could not be detected.

I cut down upon the worse (left) swollen upper eyelid, removed an elliptical fold of skin, some 5 cm. long, in the direction of the natural creasing of the normal lid, and excised a quantity of immediately underlying subcutaneous loose yellow fat, with a satisfactory cosmetic effect. I propose to do a similar operation on the right side. I am anxious to learn the experience of others regarding such an unusually placed development of fat.

History.—It is stated that the affection was not noticed until the child was ten years old; that it subsequently increased as she developed; that the prominence, weight, drooping, and inconvenience of the upper eyelids varied in degree, being worst after crying, which at times caused a complete obstruction of the vision. Occasionally a marked aggravation of the trouble was noticeable during menstruation.

(Living specimen. May 14th, 1885.)
4. Obstinate case of ectropion successfully treated by Argyll Robertson's operation.

By M. M. McHardy.

The patient is a carman, aet. 39, for very many years subject to relapsing ulcerative keratitis with conjunctivitis, and habitually exposed to the weather of London. For upwards of four years the right lower lid had been persistently everted. During upwards of seven weeks' in-patient treatment and prolonged out-patient care every effort short of operating had failed to remedy the ectropion.

On December 31st, 1884, I operated to cure this ectropion in the manner recommended by Dr. D. Argyll Robertson, 'Edinburgh Clinical and Pathological Journal,' December, 1883.

I tied a bunch of three thicknesses of india-rubber drainage-tube, of the size of No. 6 urethral catheter, under the external loops of suture, and was able to keep the lead splint and suture in position until the thirteenth day, when the distortion was quite completely remedied. The benefit has continued. A more severe test for the operation has not come in my way these last two years, and can hardly be imagined by me. This and other experience of the operation induces me to recommend it in the most unreserved manner for suitable cases.

Dr. Robertson's experience has led him, like me, but quite independently, to place a bunch of drainage-tube under the loops of suture instead of a single thickness as at first advised.

(Living specimen. March 12th, 1885.)
5. Vesicle of cornea.

By Anderson Critchett and Henry Juler.

John G—, æt. 54, labourer, came to St. Mary's Hospital on the 5th December, 1884, suffering from a well-marked vesicle on the central part of the right cornea. It was nearly hemispherical in shape, its base being about 4 mm. in diameter, and its forward projection about 3 mm. Its contents were almost transparent and colourless. There was considerable pain in the right eye and brow, and much photophobia and lacrymation, but there was only slight inflammatory redness. The patient stated that this condition had existed for five months, and that he believed it originated in a cold. There were no signs of herpes in other parts of the body, nor could we ascertain any history of such. His general health had been fairly good; had never suffered from any thing but chronic rheumatism. He now complains of feeling somewhat weak.

The tension of this and of the left eye was normal. A light compress was applied to the closed eyelids, and on presenting himself to-day it was found that the vesicle had ruptured, but was again refilling. This, the patient believes, is what it has done all along since its first appearance.

(Living specimen. December 11th, 1884.)

In answer to some suggestions as to treatment a trial of arsenic internally was suggested by Mr. McHardy.
II. INTRA-OCULAR TUMOURS, ETC.

1. Tuberculosis (?) of the cornea and iris.

By Arthur H. Benson.

I suppose it may be assumed that, in the present state of knowledge no growth in which the specific bacillus has not been demonstrated can be regarded as undoubted tubercle. On the other hand, few will refuse to allow great probability to attach to a diagnosis based wholly on the anatomical and clinical characteristics which, until recently, would have been regarded as irrefragable proofs. We can, however, hardly go so far as Mr. Mules in his recent article in the 'Ophthalmic Review,' in which he says, "The non-detection of the tubercle bacillus in supposed miliary tuberculosis need in nowise invalidate the accuracy of the diagnosis." It would seem rather that in such a case the verdict of "not proven" is the only one that can be passed. For, if the bacillus is the only indisputable proof we possess of the tubercular nature of a growth, no evidence short of its presence can be regarded as convincing. If this be so, the case which I wish to bring before the Society to-night must be regarded as of doubtful nature, but, as further evidence may be forthcoming, and the case is, at least, a rare one, I shall venture to give some particulars regarding it.

Case.—Mary E—, æt. 18, was admitted into St. Mark's Ophthalmic Hospital December 6th, 1884.

History.—She stated that until five months ago her sight in both eyes was perfect. The left eye then got itchy, and subsequently bloodshot, to relieve which she was leeched, and afterwards caustic was used to the eye-
The sight of the left eye steadily deteriorated, but the sight of the right eye remained perfect until about a fortnight before she came under my care.

She had only once menstruated in her life, and that was about the time that the first eye became affected, eighteen months before admission, and she had suffered from a pain in the left side pretty constantly for a considerable time. An eruption of psoriasis occurred on arms and thighs about the time of her first menstruation. No evidence whatever of syphilis could be obtained.

**Family history.**—Her father died of “decline” a year ago. Her sister is dying of the same thing, and has a cough and spitting of blood. A brother is “delicate” and has a cough.

**Condition on admission.**—She is fair haired with bright complexion. Has several enlarged glands in the neck under the jaw; she has no cough, but suffers from a “stitch” in the left side, due apparently to a very slight attack of pleuritis. The condition of the chest organs was subsequently, on two occasions, most carefully examined without eliciting any evidence of disease.

**The eyes.**—The left cornea was flattened somewhat and for the most part rendered opaque by masses of yellowish grey, translucent material, several of which had, in places, coalesced. The masses were apparently free from blood-vessels, but there was a zone of vascularity surrounding their bases, most marked in the masses nearest the periphery of the cornea. The smaller spots in the cornea had no vascular zones. The anterior chamber was shallow and there were small nodules of yellowish colour on the iris also; and at the angle of the anterior chamber the masses seemed to fill the space between the iris and cornea. There was deep circum-corneal vascularity. The fundus could not be illuminated. Vision = p. 1. The tension, normal at first, soon fell to — 2.

The right eye had only been a fortnight affected. There was considerable zonular vascularity; the cornea was transparent except in five or six places where it was
rendered opaque by the presence of masses similar in appearance to those in the left eye. There was one nodule visible on the inner side of the pupil border, and the whole iris looked congested and infiltrated, and acted but sluggishly and imperfectly to atropine. There were some small grey punctiform masses on the anterior capsule of the lens. The tension was normal; the vision was $\frac{5}{60}$. In the conjunctiva covering the sclerotic to the outer side of the cornea there were three small, elevated, yellowish masses, not unlike what are at times seen in the commencement of an attack of episcleritis. They were moveable with the conjunctiva, though partly implicating the tissue beneath it.

There was no ulceration or abrasion of either cornea, but, corresponding with the position of one or two of the nodules, there was a slight anterior elevation. In some few of the corneal nodules a superficial conjunctival vessel seemed to run, but it broke up into a zone of small vessels and supplied the cornea around the base of the growth. Some of the nodules seemed to be situated almost on the inner surface of the cornea, whilst others occupied a considerable portion of the corneal thickness.

In both eyes the cornea has been all along the structure most affected, and it seems probable that it was that first attacked, though of this there is no proof.

The opinion that the case was probably one of tuberculosis was hazarded, and constitutional and local treatment based on that supposition was adopted. However, the eyes seemed little influenced by treatment and steadily deteriorated in vision, tension, and transparency of cornea. To endeavour to arrive at a more satisfactory diagnosis I enucleated one of the nodules from the conjunctiva of the right eye, but when hardened it was too small to manipulate, and its examination yielded only negative results. The nodule, however, did not re-form, and the others diminished in size. As the sight of the left eye was almost wholly gone, and its tension was $-2$, I enucleated it, and hardened it in alcohol to examine for tubercle
bacilli. Dr. Denning, our house surgeon, kindly undertook to make the microscopic examination for me under the supervision of Professor Purser.

Microscopical examination.—Sections through the cornea show that the nodules are situated external to the posterior elastic lamina, which, though extensively displaced and raised up by the mass, seems for the most part to have resisted disintegration; and the layer of endothelial cells on its aqueous surface remains intact.

The neighbouring cornea is infiltrated and its structure partly destroyed, the lamellae being separated by masses of cell growth.

The nodules consist of an agglomeration of round nucleated cells, in the centre of which a caseating area is seen and epithelioid cells. In places there is an appearance as if several masses of cells had coalesced, the peripheral portions of each mass stain much better than the central. There are no distinct giant-cells visible.

The nodules on the iris show a very similar arrangement; they, however, for the most part project from the uveal surface into the posterior chamber. The tissue of the iris is infiltrated and its thickness considerably increased.

The patient's condition, as regards general health, seems improving. Since her admission to hospital, the glands in the neck are much less marked, and the pain in the side has gone. Menstruation (again accompanied as at the first, by a very scaly psoriasis rash) has returned, and she is looking better and healthier than when admitted.

It is possible that the removal of one focus of infection, the left eye, has had something to do with this, but probably also the good feeding and the cod-liver oil and iron help. The condition of the right eye still deteriorates. Tension is now — 2, and \( V. = 1\frac{5}{6} \). The cornea is more clouded, and the nodules everywhere are larger and more numerous than on admission. The only point of improvement is the conjunctiva where the yellow nodule was removed.

Is tuberculosis of the conjunctiva identical with what is
better known as lupus of the conjunctiva, or are the affections different?

The description of tubercle of the conjunctiva given by Horner (Vide Mules's paper, 'Oph. Rev.,' January, 1885, p. 3) would equally well suit cases which I have been in the habit of calling lupus. He says, "It is a manifestation of a coexistent and similar affection of the lymphatic system of the same side, and is generally found upon the conjunctiva of the lid, rarely of the globe; the lid appears thickened; the conjunctiva is converted into a greyish-red mucous mass, granulating and discharging a yellowish secretion. The disease always lasts a long time. Decay slowly proceeds while new granulations spring up; marginal ulcers and pannus of the cornea may finally develop. The prognosis is not always bad; sometimes the disease disappears, leaving but a few scars."

Wishing to demonstrate, if possible, the tubercular nature of such a case, portions of the living granulation were inserted rapidly into the anterior chambers of two rabbits.

Some conjunctivitis was present for a day or two, this, however, soon subsided, and the small pieces of transferred tissue could be dimly seen behind the cornea. For a week the rabbits showed no signs of indisposition, but then one of them refused its food, and died in two days.

Numerous dark patches of submucous haemorrhages were found in the stomach, but nothing else to account for death. No tubercles were observed in any of the viscera. The eyes were examined by Mr. Abraham, who has kindly furnished me with the following report. "On section the eye exhibited a granulation in the anterior chamber close to the point of inoculation. It consisted of cells of various sizes and shapes, but showed no signs of caseation. The line of cicatrix was well seen, and in its neighbourhood the corneal fibres were densely infiltrated with inflammatory cells, which likewise spread into the conjunctiva and Descemet's membrane near by; the latter seemed to be raised up, and its proliferating
cells became incorporated with the granulations within the chamber. Some of the granulations present a papillary arrangement.

"Sections were examined for tubercle bacilli by Gibbes' method, but without result. The other rabbit flourished; the inoculated mass diminished in size and seemed almost totally to disappear after a couple of weeks. About a month or five weeks after the inoculation a greyish-yellow nodule was observed on the iris, this enlarged, and others appeared and coalesced. The corneal wound was somewhat bulged out, and there was considerable zonular vascularity around the cornea. The anterior chamber rapidly filled with the new growth, but the cornea for the most part remained transparent except around the inoculation puncture.

"This rabbit lost its life by chloroform two months after inoculation. Its anterior chamber seemed to be immensely enlarged and entirely filled by a new growth, which on section was seen to be made up of similar irregular cells, with much surrounding inflammation, and the same papillary arrangement as was observed in the eye of the other rabbit. This granulation showed some evidence of caseation, and some doubtful giant-cells were also to be made out, but no bacilli could be found."

The question for which we thus sought an answer has been, I suppose, settled by recent observers, notably Pagenstecher, who found the bacillus of tubercle in the growth produced by the inoculation of lupus granulations into the anterior chamber.

It seems however, open to some doubt whether such a demonstration is an absolute proof of the identity of the affections, for other experimenters have produced like results by inoculation with various substances having no claim to specific organisms; so that until after frequent confirmation by exceptionally careful experiment, all such demonstration must be regarded as no more than strong presumptive evidence.

If, however, it be true that the affections are identical,
TUBERCULOSIS (?) OF THE CORNEA AND IRIS.

it is worth noting that in the case which forms the text of this paper, the appearances of the growths in the anterior chamber resembled most markedly the appearances seen in the rabbit's eye a month after inoculation, with only this difference, that in the rabbit the iris was the most obviously affected, whilst in my case the cornea was far and away the most extensively, as probably it was the earliest, implicated structure.

The diagnosis is therefore not indisputable at present. It is based upon the patient's strong tubercular antecedents; on the strumous glands in the neck and other evidences in her of a tubercular disposition; on the clinical history, the rapid spreading, and the formation of distinct foci; on the utter failure of treatment to stay the progress of the disease; on the microscopic similarity which the disease presents to cases which have been acknowledged to be tubercular; on the anatomical disposition and formation of the growths under the microscope; and, lastly, on the difficulty of assigning it to any other cause.

By a process of exclusion we come to tubercle.

Into the literature of the subject I need not now enter, as it is familiar to all present. If this case is really one of tubercle it presents the peculiarity that in both eyes the cornea appears in all probability to have been the structure first affected, and it was certainly the one which suffered most from the disease.

In conclusion, I beg to express a hope that the Society will permit the specimens to be referred to the Committee of Reference to report upon at the next meeting.

(March 12th, 1885.)

Report of the Committee on Mr. Benson's Case of Growths on the Cornea and Iris.—We have examined Mr. Benson's sections of the cornea and iris of this case, and have also cut and examined other sections of the cornea, iris, and choroid of the affected eye.

We find that the nodules on the cornea are chiefly
situated external to the posterior elastic lamina, which in some sections completely surrounds the nodule, but in other sections has given way, and the iris has become adherent to the cornea, and infiltrated by the same growth.

The nodules consist of round nucleated cells staining deeply, and in some sections small granular spots may be seen, as if the cells might have broken down; but this condition is not constant, and is the only evidence we find of anything approaching caseation. On the contrary, in most of the nodules the cells, especially towards the centre, become elongated and spindle-shaped. There are no giant-cells to be found anywhere, and there is an absence of any reticulated structure as is found generally in tubercle. Several sections were stained on two separate occasions by the Weigert Ehrlich method for bacilli, but none were found.

The sections of the choroid only showed it to be healthy.

Mr. Benson tells us that the growths on the cornea and iris of the infected rabbit were exactly of the same character as in the eye we examined.

Therefore, from the absence of giant-cells, reticulated structure, bacilli, and the doubtful existence of caseation, we should say that this is not a case of tuberculosis. This opinion is fortified by the inoculation experiments into the anterior chamber of rabbits producing no evidence of general tuberculosis.

WALTER H. JESSOP.
J. B. LAWFORD.

(June 4th, 1885.)

Mr. Jessop having read the report on Mr. Benson's preparation.

Mr. Swanzy inquired what Mr. Jessop considered the nature of the disease process to have been.

Mr. Jessop having replied, that he would regard it as a granuloma—
Mr. Swanzy further inquired whether Mr. Jessop was of opinion that a distinction between granuloma and tubercle should be made.

Mr. Jessop answered that tubercle is an infective granuloma as also are lupus, syphilis, glanders, &c., and that it could only be diagnosed by the sum of the characters appertaining to it, viz. giant-cells, caseation, reticular structure, distinctive bacilli, and general tuberculosis as a result of experimental injections. These factors being absent, he certainly pronounces the growth to be non-tuberculous. Further than this negative result the Committee could not go as the microscope alone could not diagnose a syphilitic granuloma.

2. Tubercle of the iris and choroid; cultivation of the bacilli in blood serum.

By W. H. Jessop.

The author showed sections of eye of a rabbit artificially inoculated with tubercle by Mr. Watson Cheyne during his experiments on the relation of micro-organisms to tuberculosis, recorded in the 'Practitioner' of April, 1883.

The bacilli of tubercle were cultivated in serum, and some of the cultivation product was injected into the anterior chamber of the eye. The eye in three weeks showed well-marked tubercular deposits on the iris and soon became converted into a caseous mass, the animal living sixty-six days. The whole body showed signs of general tuberculosis.

The sections of the eye were stained by the Weigert Ehrlich method, by fuchsiu, and methylin blue. They show bacilli in great quantities, pervading the tissue in all
parts, especially in the choroid and iris, and there are several giant-cells to be seen.

A tube was exhibited showing the cultivation from which the inoculation was done. It exhibits the characteristic growth of the bacilli in blood serum, forming whitish irregular crusts on the surface. The specimen is the twenty-third cultivation from the lung of a patient who had died of phthisis.

(Card specimen. January 8th, 1885.)

3. An intra-ocular gumma in a child the subject of inherited syphilis.

By W. Spencer Watson.

The following case illustrates the difficulties of diagnosis sometimes experienced in dealing with uncommon cases. At an early stage the case seemed one of hypopyon and onyx, later on it took the aspect of intra-ocular abscess or choroidal tubercle, and at last it assumed its ultimate form which I venture to call an intra-ocular gumma. This may seem, even if a correct, to be a somewhat vague diagnosis; but my reasons for avoiding greater precision and being content with a somewhat unsatisfactory diagnosis as regards the particular tissues affected, will be apparent in the course of the relation of the case.

Eunice S—, at. 6, a fairly well-nourished child with good facial development, good bridge to nose and fair complexion, has enlarged lymphatic glands in the neck and scars of abscesses. The aspect, therefore, suggesting scrofula rather than syphilis. First came under observation at the South London Ophthalmic Hospital on Dec. 1, 1884, with what was at first thought to be scrofulous keratitis of the right eye of about fourteen days' dura-
The child had a hoarse whispering voice and a discharge from the right nostril dating from her birth; and the account given by the mother made it almost certain that she was the subject of inherited syphilis. The family history in detail was, that the father had been attending Brompton Hospital and is at the present time suffering from cough and night-sweats. The mother has been married eight years, and has had three confinements; the first child, now seven years of age, is healthy with the exception of having enlarged glands in the neck; the second child is Eunice (the patient of this report); the third was born dead five years ago; there have been no miscarriages. The mother first noticed a rash and sore throat after the birth of the second child; the tongue then became sore on the edges and her hair fell off. She has now psoriasis at the bend of the elbows. Previously to the birth of the second child she had no symptoms of syphilis and never had a primary sore. This account is taken from the notes made by Mr. Strugnell, house surgeon of the Great Northern Hospital.

Later on an ulcer of a serpiginous character formed on the left thigh.

The affection of the eye was treated at first by simple local remedies, with syrup of iodide of iron internally, but, about January 5th, 1885, the centre of the cornea became dull and nebulous and a small hypopyon formed. On January 12th the aqueous had become clearer and the opacity in the cornea passed off, but a yellowish deposit now occupied the pupillary urea, and at the same time a prominence of the size of a small split pea had formed on the upper and outer ciliary region of the sclerotic. There was also much pain and photophobia.

The sclerotic prominence having the aspect of an abscess about to burst, cocain was applied and the centre of the pointing part incised. Yellow, cheesy, semi-solid substance presented in the wound but no pus. The eyeball did not collapse, vitreous and aqueous remaining in their normal situations. No pain was felt by the child at the time of
the puncture. The cocain therefore answered admirably. A boracic acid lotion was now applied and the other medicines continued. On January 16th (four days later) another puncture was made with a similar result. About this time an ulcer on the left thigh was noticed. It had begun as a more or less circular group of pustules which subsequently ulcerated into a serpiginous ulcer, healing at one edge and spreading along the opposite margin.

About this date the child was admitted into the Great Northern Hospital, January 25th. She was now put on a course of mercury, at first by inunction and afterwards by the internal administration of grey powder. Iodoform in the form of powder was applied to the ulcer on the thigh, and an ointment of iodoform and vaseline was used to the nostril. The hoarseness of voice still continued, and my colleague, Dr. Clifford Beale, was kind enough to examine the larynx. He found both vocal cords inflamed and thickened so that they could not be completely abducted or adducted, though still capable of limited movement, to the extent of perhaps one eighth of an inch. When approximated as closely as possible a chink remained between them. Dr. Beale therefore regards the case as one of chronic laryngitis.

Hitherto the intensity of the photophobia had prevented any ophthalmoscopic examination, but at length (on January 31st), having first applied cocain freely, the photophobia was so far relieved that I succeeded in getting a view of the retina and optic disc of the left eye. The disc was somewhat pale but distinctly outlined, and the retinal veins somewhat distended, but there were no changes in the choroid.

After treatment by mercurial inunction for five days, the following was the condition on January 31st: The prominence of the sclerotic region of the right eye had nearly subsided, but its position was marked by a minute pigmented scar. Vision remained nil. The ulcers on the thigh had scabbed over; and on February 4th photophobia was gone. The pupil now appears black; there
is no inflammatory opacity of the cornea nor vascularity of the eyeball.

On February 18th the voice was still hoarse but more sonorous than before. The affected eye is evidently shrinking; there is no prominence in the sclerotic but a flat pigmented scar only. The general health improved and the ulcer of the thigh quite healed.

The points of this case to which I wish to direct attention more particularly are: 1. The difficulty of diagnosis in the early stages. 2. The results of treatment as bearing upon the diagnosis. 3. The rarity of the lesion in connection with inherited syphilis.

1. Difficulty of diagnosis. For a few days at the early period of the case the appearances were those of hypopyon ulcer, then the yellow deposit formed in the pupil, and the case resembled one of intra-ocular abscess, the matter pointing at the prominent part of the sclerotic; then later on the prominence was punctured, and, no pus escaping, it was evident that the deposit was of a semi-solid nature, and as this subsided under the influence of a course of mercury, I think we may conclude that there was here an instance of intra-ocular gumma.

2. The results of treatment by mercury were so marked that they must necessarily tend to confirm our diagnosis as to syphilis, for had it been a case of abscess or retinal glioma or choroidal tubercle we should have scarcely expected mercury to be beneficial. The diagnosis, therefore, was confirmed partly by the absence of pus when the swelling was punctured and partly by the undoubtedly good effects of mercury, and the treatment thus materially aided us in arriving at a conclusion.

3. The rarity of this affection must be great, but I cannot help thinking that some of the cases of glioma or supposed glioma of the retina, which have shrunk away spontaneously, may have been instances of the same kind as the one related. Gliomata seem to be in some cases highly infective and in others closely resembling them as to symptoms; though no operative treatment is adopted the
eyeball shrinks and we hear no more of any gliomatous disease in it or any other part of the body. The obvious conclusion is that we are here concerned with two different diseases, or perhaps with three or more, in all of which the initial symptoms may be somewhat similar. In any such cases the family as well as the personal history should be carefully investigated, and it may turn out that some of the so-called gliomata are tubercles, some abscesses, and some syphilitic gummata.

Having this suggestion in mind it may be useful to avoid too hasty a diagnosis as to glioma of the retina, and we may thus avoid the discredit which attaches to mistaken prognosis and treatment.

Two cases bearing on the diagnosis are related by Dr. Gowers. One, a case of tubercle of the choroid which perforated the sclerotic and appeared on the exterior of the eye, reported from Dr. Manz, and a case of Dr. Barlow's, in which an intra-ocular growth threatened to perforate the sclerotic but which, on extirpation of the eyeball, was found to be a growth resembling tubercle (confluent) lying between the choroid and sclerotic in the lower half of the eyeball. In this last case death occurred a year later, and on post-mortem examination tubercular growths were found in the cerebrum and cerebellum.

As, in my case, only a few months have elapsed, it is possible that further symptoms may develop, and it is therefore well to keep the child under observation. This I shall do and report any further particulars that may seem of interest.

May 14th, 1885.—The patient, who has been laid up three weeks with pleurisy, presented herself at the hospital and can now (she says) see a little with her right eye. When tested, however, vision, if any, is the merest perception of light.*

(June 4th, 1885.)

* Sept., 1885.—Since the above notes interstitial keratitis of the left eye has come on, and was running its usual protracted course when the child was last seen.
4. Double retinal glioma resulting in the shrinking of one eye and the perforation of the other.

By W. A. Brailey, M.D.

A. D,—, aged 16 months, was brought to my house in October, 1882. The parents had some two weeks previously noticed something peculiar about the left eye, and they had taken the child to Mr. Vernon, whose opinion it was that it was suffering from retinal glioma. My notes say the left shows a yellowish-red, vessel-bearing, flattened surface in the posterior part of the vitreous chamber. It nowhere extends as far as the lens, and is much less conspicuous towards the inner side. The tension is doubtfully full. The ant. chamber is shallow, the iris periphery is not retracted. The pupil is of medium size and round; it dilates evenly but not fully to atropine. There appears to be no perception of light and no pain. There are no enlarged glands.

The right shows a greyish reflex from the lower part of the fundus, looking rather like a retinal detachment. The vision seems eccentric as well as defective.

The appearances warrant a clear diagnosis of glioma in the left eye, where the entire retinal thickness is implicated, and in the right eye a strong probability of glioma, commencing and developing in the external retinal layers (glioma exophytum). Information kindly furnished by Mr. Vernon and by Dr. Brock of Tooting, shows that in August, 1883, i.e. ten months after I saw her, the right (the second affected eye) was the subject of an acute suppurative panophthalmitis. The upper lid was inflamed, swollen, and closed. The eyeball was said to be swollen. Discharge of a pale yellowish colour escaped from between the lids. There was at first great pain, after which the child, who was treated for meningitis, lay in a state of semi-stupor for three or four days. Recovery seemed doubtful at first, but as the eye gradually shrunk, the
child regained its previous state of health. In three months there was merely an atrophied and painless stump as at present.

In February, 1885, i.e. about two and a half years after the patient was first seen and one year and seven months after shrinking of the right eye, a small, flattish, circular, wartlike growth made its appearance through the sclerotic at the upper and inner ciliary region of the left eye, and thenceforward grew very rapidly. The mother thinks that a second point of perforation developed in the sclerotic of the lower and outer ciliary region. When on April 28th, 1885, I saw the child for the second time, a rounded mass, some half inch in thickness, projected for some three quarters of an inch from beneath the red and much thickened left upper lid. It moved together with the shrunken stump of the right side. It looked very vascular, but its surface was smooth and rather shiny. It was not ulcerated or scabbed at any point.

No glandular enlargements are visible. The child's memory is perfect. Its intelligence is remarkable, and it appears extremely sensitive to its deformity. It is very pallid and rather thin. The only other things to be noticed are periodic attacks of severe pain, each lasting several days. Latterly they have recurred about every two weeks. The pain appears to be in the orbit and in the front part of the head. Two months later the child was still alive, but the growth was rapidly increasing and frequently bleeding.

The points of interest in the case are:

(1) Its long duration.
(2) The shrinking of the second eye from acute suppurative panophthalmitis.
(3) The extension of the disease from the eyeball.

In connection with the first point, I may state that it has long been an opinion of mine that retinal glioma runs a much slower course than is commonly supposed. Indeed, I fancy that it always takes its rise within the period of intra-uterine life.
Mr. Snell, in the 'Transactions' of this Society for 1884, records a case in which one gliomatous eye shrunk while the tumour went on developing in its fellow, ultimately perforating and forming a large mass as here. But this process of shrinking, which I suppose we may regard as a spontaneous cure, does not appear to have been in his case the result of a suppurative panophthalmitis. I quoted at the same meeting a case somewhat akin to his.

As to the mode of extension of the disease, I gather that Mr. Snell, from the result of his post-mortem examination, is of opinion that the tumour cells implicated the optic nerves and tract and so were the cause of the disease of the second eye. I have not so far had the opportunity of similarly examining this case. But I may refer to a case reported by me in vol. x of the 'Ophthalmic Hospital Reports,' where the gliomatous right eye of a child, aged two months, was excised. The nerve, as far back as the lamina cribrosa, was distinctly invaded by tumour cells, and it was very evidently hypernucleated beyond this limit. At that time the other eye was sound, but thirteen months later it too was found to contain a glioma. It was excised two and a half years later than the other.

The optic nerve of this eye was vastly thickened up to where it was divided by the scissors. The inter-sheath space was closely packed by glioma cells of rather faint outline. Similar cells were visible within the hypertrophied fibrous trabeculae of the nerve close to the eye, but a little further back these interspaces were simply filled by a nearly translucent substance, which appears to be the product of compressed and degenerated glioma cells. The fibrillation of the fibrous tissue was much less distinct than normal, and blood-vessels were rare in it. I have no further notes of the progress of the case, but in it the disease appears to have spread along the nerve from one eye to the other without any recurrence in the socket of the first eye, and without up to that period any implication of the meninges of the brain.
I am therefore disposed to infer that in my present case the disease spread from the left eye to the right by direct continuity along the nerve, and that the shrinking of the first eye is due to degeneration of the glioma cells, either directly from the compression produced by the suppurative inflammation, or indirectly from diminution of their blood-supply from the same cause.

For glioma cells are very prone to degeneration, as may be observed in almost every microscopical preparation of intra-ocular glioma, where each capillary is immediately surrounded by a zone which stains deeply with logwood, its cells being well-defined, whereas those outside this zone have little staining capacity and little sharpness of outline but look finely granular or fatty. Such changes are commonly well seen in the optic nerve after its morbid thickening has endured for some time.

(June 4th, 1885.)

Note, September 12th, 1885.—The protruding mass still grows, though more slowly. There is constant discharge of a thin, reddish fluid, but no considerable haemorrhage. Secondary growths have made their appearance in the parotid and submaxillary regions of the same side. The right arm and leg are numbed, and that side generally appears to have lost power.

Mr. W. Adams Frost asked if there were any cases recorded in which life had actually been preserved by the excision of both eyes. If, as Dr. Brailey suggested, the second eye becomes affected in consequence of a direct extension of the glioma cells along the optic nerves and chiasma, removal of the globes must be futile since the intervening infected tract remains. If cases did exist in which double excision had proved successful they would go far to prove that the second eye was not, at any rate always, affected by direct extension.
5. Glioma of both retinae.

By W. Lang.

The patient was first seen when sixteen months old in March, 1882, with a glioma of the right retina for which the eye was enucleated. The history as to the length of duration was not very definite, but something strange—a redness in the eye—was noticed after vaccination, i.e. when the patient was three months old.

At the time of the enucleation no abnormal change was noticed in the left eye. The growth was confined to the retina, did not extend along the optic nerve, and occupied about the posterior two thirds of the globe.

In January, 1884, the patient was again brought with a growth in the left eye. It was a very small growth in the front part of the retina, at the inner side close to the ciliary processes. The vision then seemed normal and the mother says the child could see well till within three months.

The pupil still acts slightly to light, though the fundus is nearly filled by the growth. There is now a small secondary growth in the episternal notch.

(Card specimen. July 3rd, 1885.)

6. A dark coloured lobulated growth over the ciliary region.

By M. M. McHardy.

S. J—, a healthy woman, aet. 54. A large growth of two years standing, was amputated in July, 1884. It had a thin, wide pedicle, which was touched with the actual cautery and caused no trouble, till the present small single globular growth developed at its upper extremity. There is a flat pigmented patch over the ciliary region above the cornea; this staining has been noticed six years, and is increasing. The vision is not impaired.

(Card specimen. May 14th, 1885.)
III. DISEASES OF THE IRIS.

1. New formation of pigment on the front of the iris.

By E. Nettleship.

(With Plate I.)

The greater part of the front of the iris is covered by a layer of dark, reddish-brown pigment. The pigment entirely covers the inner circle of the iris; peripherally its outline is irregular and presents numerous broad processes, none of which, however, reach quite to the ciliary border. This pigment layer is everywhere sharply defined; it has a quite appreciable thickness and is in some parts separated from the iris beneath by a space sufficiently deep to allow of its casting a shadow. It has every appearance of being a new formation of pigment. The iris itself is of a grey colour.

The patient, Roderick M—, aet. 43, with very dark hair, had a cataract removed from the eye (represented in the Plate) at the age of twenty-one; the eye was very badly inflamed for two months afterwards, and has been blind ever since. There is now extensive and very deep detachment of the retina; p. clear and round and of the same size as the other, its indirect reflex action brisk and of fair range; a. c. of fair depth; T - 2; no p. l. At one examination a translucent membranous substance was present in the pupil. The other eye is perfect, and its iris of the same grey colour as the true iris of the blind eye.

(Card specimen. October 9th, 1884.)
DESCRIPTION OF PLATE I.

Plate I illustrates Mr. Nettleship's case of New Pigment formed on the front of the Iris (p. 66).

(From a drawing by Miss Boole.)
2. Cystic tumour of iris.

By F. R. Cross.

E. B—, female, æt. 30. Left eye normal. V. = $\frac{20}{25}$, and J. 1. Right eye V. = $\frac{20}{15}$, and J. 4. A dark smooth body protrudes into the anterior chamber, pushing up the iris at its periphery and splitting its fibres, and occupying the lower one sixth of the iris ring. The lower edge of pupil is quite free but is pushed up by growth, thus probably causing the impairment of vision.

Two years and nine months ago the patient, while walking, felt as though something had blown into the eye. When she got home the eye was red, and continued so, with a sensation of some foreign body being in it for three days.

At the end of three days the irritation entirely ceased, but a black spot, as large as a pin’s head, was noticed at the worried spot, where the tumour now is. No interference with sight has been noticed. The spot enlarged without any pain for a year, reaching its present size, and then subsided to the size of a pin’s head, being scarcely noticed. In May, 1884, it again enlarged with redness, pain, and symptoms of inflammation.

On September 11th, 1884, I first saw the patient. The tumour was then, as now, the size of a pea, but the eyeball was hyperæmic and painful, but not glaucomatous. This pain continued a month or so. For the last two months the eye has been perfectly quiet, except a slight conjunctival catarrh, which the patient thinks relieves the eye.

(Card specimen. December 8th, 1884.)
3. **Cyclo-choroiditis with vitreous hæmorrhages, subsequently involving the iris, with hyphæma, varying tension, and later, enormously deep anterior chamber.**

By W. A. Bailey, M.D.

Setonia W—, æt. 55, a married German woman living in London, has been under observation for nineteen months. She first complained of severe pain over both brows, especially the left, and this eye had large black-looking floating opacities in the vitreous, and vision = 6/60 only, and still failing; iris flat and of good colour. T. n. The right eye had the pupil excluded and occluded, and the iris bulged; p. 1. only; — T. 1. She gives a history of attending Moorfields Hospital two and a half years ago for the right eye, which was attacked with severe pain and gradually failing sight, the course of the complaint being exactly similar to that of the left now, so far as she could judge. She became gradually worse till twelve months later when the sight of that eye was entirely gone. She came to Guy’s soon after she noticed that the left was affected. There is no history of syphilis, gout or rheumatism. She has had six children in all; three are alive, the eldest æt. 26, and also the fifth- and sixth-born, æt. 14 and 12 respectively. This last is a perfectly healthy looking girl. The second-born died æt. seventeen days, and the third æt. eleven months. These were twins. The fourth-born died of fever at the age of five years.

The husband treats and feeds the patient very badly. She was treated in hospital successively with iodide of potassium, quinine and iron, salicylate of sodium, alkalis, &c., without material benefit. Indeed she became decidedly worse with the iodide. She attended on and off till September, 1884, when blood made its appearance in the a. c. of the left eye. About three weeks later the tension was observed to be in excess, and the blood gradually reached the pupillary edge. Since then it has varied, on the whole gradually diminishing. The tension has varied from T. 1 to — T. 1, but it has
borne no relation to the amount of blood in the a. c. Nothing relieves the pain but very temporarily.

On November 20th both supra-trochlear nerves were stretched to rupture as a last resort for the relief of the pain. This subsided and the tension kept pretty normal till November 28th when the left eye had T. 1.

December 4th.—Pain was complained of round both eyes. 5th.—The iris of the left was discoloured and the pupillary margin irregular.

15th.—The pain was worse over the left eye which had — T. 1 with ciliary injection and rather deep a. c. On the 18th the left a. c. was vastly more deep, the eye tender to pressure, with much ciliary injection and pain. The right was also slightly injected. Its a. c. was deepened; at its margin six anterior synechiae are visible, from which ridges extend radially towards the excluded pupil.

21st.—These ridges are even more prominent, and the a. c. is deeper. T. slightly diminished.

The left has a. c. deeper than ever; the aqueous clear, and the iris much discoloured. There is a hazy reflection from behind the pupil; p. l. still exists; the blood looks much less, but this is probably simply because the a. c. is of such enormous depth.

On January 1st, 1885, the left is much the same, but the right a. c. is now shallow as it was three months ago, and the synechiae and ridges are not visible at all.

From this date the inflammation gradually subsided in the left eye also. The author asked the Society, what is the nature of the inflammation? and was its exacerbation in any way related to the nerve stretching?

(Card specimen. January 8th, 1885.)

In May, 1885, an artificial pupil down and in was made upon the right eye, since the iris here was of fairly good colour as compared with the left. Much thin yellow fluid escaped, and then apparently some thin vitreous. The lens was found opaque, and no fundus reflex was obtained, though the eye recovered well from the iridectomy.
4. *Sequel to a case of granular-looking body on iris.*

By **Frank H. Hodges** (Leicester).

The patient was exhibited 13th March, 1884 (*vide* "Transactions," vol. iv, p. 59). The growth was shaved off and examined under the microscope, and found to be composed of round cells. On the suggestion of Sir William Bowman a compress bandage was kept on the eye for several weeks, during which absorption of the growth or exudation slowly took place. When examined on August 19th, 1884, a slightly raised and somewhat vascular leucoma marked the site of the former growth. The curvature of the globe was good, \( T - 1, V. = \frac{6}{5} \). Patient has not presented herself for examination since.
IV. SYMPATHETIC OPHTHALMITIS.

1. On a blood theory in explanation of reflex ophthalmitis.

By Jonathan Hutchinson, F.R.S., President of the Society.

I hardly know whether I ought not to offer some apology for bringing before the Society nothing better than a question of speculative pathology. If such is needed I will seek it in the fact that the interpretation of the mode of production of "reflex" or "sympathetic" ophthalmitis, is one of exceeding difficulty, and at the same time of great importance.

Six years ago, in a short course of lectures at the College of Surgeons on the influence of the nervous system in the production of disease, I discussed in some detail the facts as to this most peculiar affection and their modes of explanation. When I began the preparation of these lectures I certainly believed that, in reflex ophthalmitis, we had one of the best instances that could be adduced in proof, that it is possible for a destructive inflammation to be initiated directly through the influence of nerves. More close examination of the subject, and comparison of its phenomena with those of herpes and other cognate maladies, led me, however, to the belief that the nerves did not initiate the inflammation of the second eye by any exercise of functional power, but that they were probably themselves structurally involved in it. Thus, my hypothesis was that the inflammatory action spread along nerve filaments to the central nucleus, and there effected a crossing-over and implication of those
of the opposite side. This idea got rid of any suggestion of trophic power in nerves, but it must be admitted that it was not much more easy of belief.

I wish this evening to put forward for consideration another and wholly different suggestion. Is it not possible after all that reflex ophthalmitis is not reflex or sympathetic in any sense, and has nothing whatever to do with the nervous system? May we not find parallel phenomena under conditions which require that we shall suspect the circulating fluids rather than the nerves? I know that this thought is not by any means original to myself, and that of late years several observers have arrived at somewhat similar conclusions. Whether, indeed, even the details of what I am now going to say contain anything that is new or not I must leave to the decision of others.

In another course of lectures delivered subsequently in the same place, I discussed the modes of spreading or rather of multiplying of various other maladies, and arrived at the general conclusion that not only malignant newgrowths, but all forms of common inflammation, have the power of so infecting the blood as to induce similar processes elsewhere. Further than this it also seemed probable that there is another law of selective affinity, underlying the general one of blood contamination, which renders it likely that the secondary processes will be set up in certain definite parts, to the exclusion of all others. The best example which I can offer in illustration of this occurs in the case of multiple periostitis. It is one which I have often before quoted, but it is necessary to adduce it again, in order to make my meaning clear.

If a child in good health becomes the subject of acute periostitis of one tibia (or other bone), in consequence of a blow, there is a considerable probability that other bones will be affected in the same way. Thus multiple periostitis may result, to be followed by multiple necrosis; the condition to which Mr. Simon many years ago gave the name "necrosial fever." It is quite certain that in these cases the bones secondarily affected suffer, not on account of
previous ill-health in the patient, but that they are directly influenced by the inflammation of the first. They never begin simultaneously with the first but after an interval, and it is often beyond all doubt that the bone first affected suffered in consequence of an injury, and not in connection with any special constitutional state. It is also quite certain that there is no structural continuity between the bones primarily and those secondarily affected. Neither lymphatic nor nerves can, I think, be plausibly called in to our aid in this instance. The most probable explanation is, that certain elements, cells or nuclei, are shed into the blood-current, from the primarily inflamed tissues, which possess the power of inducing similar inflammation in similar tissue wherever they may meet with it. Thus often precisely the fellow-bone in the opposite side is the one to suffer, and almost always if a long bone starts the process it is a long bone which suffers secondarily. Thus apparent symmetry may be produced, but it is never simultaneous. If it is necessary to give examples I may say that I have many times seen both tibiae suffer and more than once both tibiae and one humerus. Nor is it uncommon for a bone to be threatened and the process to be arrested without actual suppuration. Transitory pains in the other bones, with local tenderness, is, I believe, a common condition during acute periostitis.

The application of similar reasoning to the case of the eye is not difficult, and it appears to me to cover fairly well the phenomena of sympathetic ophthalmitis. From the eye first injured elements are given off into the blood, which find, in the course of the circulation, their most suitable home in tissues of precisely similar character in the fellow-organ. It is a case of migration, in which the emigrant, cast forth from his home, finds for himself a distant country in which the conditions of life are similar to those amidst which he was born. There he reproduces new surroundings as like as possible to those which he has left. It will be seen that this colonisation-process is something wholly different from the extension of inflam-
mation by continuity of tissue. Of the latter we have our best example in orchitis when consequent on gonorrhoea, in which there can be no doubt that the inflammatory action spreads along the vas to the epididymis. On the other hand, the orchitis which follows mumps is very probably a colonisation process by wandering cells, and the same hypothesis may help us to explain a great number of the facts as to pathogenesis when one local process of disease appears to follow another. In the instance under consideration it well fits with the facts that there is always an interval after the injury and inflammation of the first before the second suffers, and also that it is quite possible for the second to suffer even when the first was excised some days before any symptoms were present. It may also perhaps explain the very exact symmetry, as regards the special parts affected in the second eye, which we sometimes see realized.

It will be observed that the explanation which I offer rests upon two probabilities; first, that an inflamed part does shed into the blood elements which may prove infective elsewhere; and, secondly, that the parallel structures on the opposite half of the body may exercise, so to speak, an attraction for those elements. If the theory be true for traumatic ophthalmitis it is probably true, though perhaps in a less conspicuous manner, for other forms of acute inflammation and over a wide dominion of general pathology. Pneumonia, for instance, usually begins on one side and frequently ends by being double. It may be that one lung infects the other. So also in many cases of inflammatory disease in double organs in which symmetry of development occurs after an interval.

The theory which comes the nearest to that which I have put forward this evening, is that given by Berlin, in a lecture published in 1880 in Volkmann's series. It is, indeed, in so far as it relies on the circulation of the blood as the means of transference, anticipatory of my own, and it has been adopted and ably advocated by other observers in this country. He supposes that germ
elements from the atmosphere infect the wound in the injured eye,—that they are taken up by the blood and transferred to the corresponding organ. This would clearly restrict the process to recent wounds, whereas I am desirous to suggest that one and the same law probably applies to many forms of inflammatory action, some of them having no connection with injuries, and not probably attended by any atmospheric infection. I should liken it to the process which occurs in the multiplication of malignant new growths, through the blood elements, rather than to that of bacillar septicæmia. It would indeed take a midway position between the two, the infective agents being cells produced, or at any rate modified by, the part itself, and not due to any infection from without. Still they are the products of inflammation, and in this respect differ in degree from those shed by neoplasms.

(May 14th, 1885.)

The discussion on the foregoing paper having been adjourned I thought it convenient at the next meeting to state briefly and categorically the different theories at present before us in reference to the remarkable pathological phenomenon under debate. This was attempted in the following propositions:

That the second eye becomes infected owing to disturbance produced in the hypothetical centre for nutrition of the eyes.

That an inflammatory process travels along the nerves, in their substance or in their sheaths, and thus by uninterrupted continuity of progress reaches the second eye. This may be supposed possible in reference to a the optic nerves, b the ciliary nerves (i.e. the third, fifth, and vasomotor, one or all). According to this the process is a form of travelling neuritis.

These theories suppose the nervous structures or lymph spaces in connection with them to be the paths by which the morbid action is conveyed from one eye to the other. The one is excitation of disease through "trophic nerves,"
the other its conveyance along nerve-trunks. In the one
the nerves take an active share; in the other they are
passive, and submit to disease rather than excite it.

The next hypotheses dismiss the nervous system
altogether, and suppose that the circulation of the blood is
the means of transference of infective material from one
eye to the other. They differ chiefly as to the nature of
the infective material.

Prof. Berlin supposes the infection of the wound in the
injured eye by atmospheric germs to be the first stage.
These germs multiply and circulate with the blood, finding
a suitable nidus for further development in the other eye.
He supposes that the smallness of the capillaries and the
influence of light may perhaps conduce to their infective
efficiency in that particular organ. We may conveniently
speak of this as "secondary septic invasion."

We have another form of secondary septic invasion
which may be spoken of as neuro-lymphatic since the
micro-organisms spread by direct continuity from eye to
eye in the perineural lymph-spaces. This is, I believe,
the theory of Leber and Snellen, and is supported by
facts observed by Max Knies and by able arguments
adduced by Dr. Mules.

The last hypothesis would suggest that the explanation
of reflex ophthalmitis comes under the general law that
all inflammations are infective, and that the selective infec-
tion of the opposite eye is due simply to sameness of
tissue. This theory would also suggest that in many other
diseases of double organs, in which the second suffers after
an interval, there is infection by the blood as the direct
cause of implication of the second. Without either
assuming or denying the invariable existence of germs in
connection with inflammatory action it would exclude the
restriction to wounds which Berlin suggests.

Mr. Power, in discussing the views of the President of
the Society, which he had no doubt were founded on large
clinical experience, ventured to think that there were many clinical facts opposed to the theory advanced by him. In the first place it afforded no explanation of what he believed would be in the experience of all, the greater frequency of the disease in children than in adults. This was in perfect consonance with the theory of inflammation extending along the nerves, a condition that was eminently likely to result from the restlessness of young patients, and the impossibility, on the score of health, of keeping them shut up in a dark room. A second difficulty of the blood-contamination hypothesis was the regularity, or at least frequency, with which sympathetic inflammation supervened at the end of about a month or six weeks. Were the noxious and malign elements chiefly produced about that period, or did they take that time to discover the opposite and symmetrical region on which they fastened? Again, why, when they had once attacked the symmetrical region, did the symptoms in many cases subside, to light up again on exposure to bright lights or to cold? He had an instance in his knowledge of a man who was injured with a fragment of iron, and who repeatedly declined to have the injured eye removed. This man had repeated attacks of sympathetic irritation or inflammation, but rest and warmth stopped each attack, till at length twelve years afterwards one attack became so severe that he submitted to removal of the injured eye. The enucleation was accomplished with difficulty owing to the optic nerve being the seat of a large fragment of iron. In such a case why did not the noxious elements affect permanently and seriously the opposite eye? Again, how many cases presented themselves in which long intervals of time elapsed between, not the original injury only, but the enucleation? Where in such cases was the seat of origin of the malign particles? Had they been in the system through the whole period, but been unable to discover the symmetrical spot? Lastly, there are some cases in which sympathetic ophthalmia occurs without wound. What explanation can be afforded of them?
Mr. Spencer Watson.—The President's theory seems to me to be open to the following, among other objections: It does not sufficiently take into account the fact that (as far as we know at present) the sight organ is the only one of the sense organs subject to sympathetic inflammation. We have no experience of "sympathetic otitis" or of sympathetic inflammation of the nose or of the organs of touch or taste. But if sympathetic ophthalmitis be a septicæmia in which the specific poison is set free in the blood but only attacks the fellow half of the organ by reason of that part alone affording a suitable nidus or home for its development, why should not similar processes go on in the other sense organs? On the other hand, if we adopt the theory that sympathetic inflammation in the eye is due to an extension of mischief, whether by means of bacilli or otherwise along the lymph-channels or spaces in the optic nerves, there is an obvious reason for sympathetic inflammation being peculiar to the sight organ and not being a known affection of the other sense organs. The sight organ is the only sense organ in which the two halves are intimately blended into unity by a "chiasma," and nothing corresponding to this unity exists in regard to the ear, the nose, or the organs of touch and taste. And the fact that this anatomical unity carries with it a corresponding physiological unity makes it more marked as an essential condition of the perfect sight organ and emphasises the distinction between it and the remaining sense organs. That this distinction in the normal and healthy condition should carry with it a corresponding distinction as regards pathological processes is only what might be reasonably anticipated. I incline, therefore, to the opinion that the extension of inflammation from one eye to the other in ordinary sympathetic ophthalmitis is in some way due to this anatomical and functional feature of the visual apparatus and that the readiest means of conveyance of inflammation is across the bridge provided by the chiasma of the optic nerve. My objection to the President's theory is that it ignores this anatomical and functional peculiarity of the sight.
organ as compared with the other sense organs, in which, if his theory were true, it would be very difficult to account for the absence of sympathetic inflammations analogous to that known as sympathetic ophthalmitis. In reference to Mr. Power's statement that young children are more liable to be affected with sympathetic ophthalmia than adults, my own experience inclines me to a directly opposite opinion.

Henry D. Noyes, M.D. (of New York).—Mr. Chairman,—In rising to comply with your very courteous invitation to take part in this discussion, I labour under two disadvantages, first, that I am a stranger, and second, that the topic is one of the most difficult in ophthalmic surgery. You have with admirable completeness stated the several theories which are invoked to explain sympathetic ophthalmia. For myself, while invited to express my opinion on the question of its mode of production, I am not ashamed to avow that in the existing condition of the subject, and with the lack of sufficient and decisive facts and experiments, I hold my judgment in suspense. While having a certain bias I have not a positive conviction. We all make the distinction between a reflex or sympathetic neurosis and a true sympathetic inflammation. It is generally held that the neurosis does not develop into the inflammatory condition. Can this be asserted with certainty? Has anyone observed a case of neurosis for so long a period that he can declare that time enough, say weeks or months, was allowed for inflammatory lesions to appear and that they did not appear? For myself, I have had no experience of this nature, and the ophthalmic surgeons to whom I have put this inquiry have not been able to give me an affirmative answer. The reason is obvious, viz. that for both conditions enucleation is the remedy employed, and further observation is thus prevented. I need not dwell on the different symptomatology of the two classes of cases, nor do I purpose to speak of the dissimilar ways in which sympathetic inflammation appears,
in one case announcing itself by iritic adhesions with vitreous and corneal opacities, in another case appearing primarily as a neuritis optica. I do not discuss the relations of these morbid phenomena to each other. I venture to bring forward another view of the subject, which is purely clinical, and to call attention to the variety of lesions which may evoke sympathetic trouble, whether this be the neuritic or the inflammatory form. I think we have failed of sufficient precision in noting what has been the causative mischief and what the resulting conditions, and in seeking to learn what lesions might be traced to certain kinds of injury. In fact such an inquiry is beset with very great difficulties, and when one fears imminent dangers to vision under the form of uveitis acuta, or neuritis optica, or panophthalmitis, enucleation is universally and incontinently employed as a prophylactic, and I do not see how we are to make any advance in knowledge and discriminating judgment.

A brief enumeration of the exciting causes of sympathetic ophthalmia, whether neuritic or inflammatory, may begin with the conjunctiva. I have seen it caused by the wearing of an artificial eye, and by a burn by lime, with symblepharon. In the former case, disuse of the shell removed the symptoms; in the case of burn, after waiting a week, enucleation gave relief. Concretions upon the cornea which occur in eyes the seat of chronic staphyloma, &c., sometimes awaken sympathetic symptoms, and removal of the lime salt gives comfort. Wounds with prolapse of the iris, and especially wounds in the ciliary region, are the causes most frequently met with; and as rivals in potency for evil are foreign bodies lodged within the eyeball. Among these cases may be mentioned not a few of sympathetic ophthalmia following extraction of cataract, and I venture the assertion that not less than thirty can be found recorded, and the unrecorded will be manifold more. If now we criticise the classes of cases thus far mentioned, how enormous is the number of not perfectly normal cataract operations unattended by the
result we are discussing. I have now under observation a man who for nineteen years has carried a piece of iron in plain view upon his iris and suffers no detriment. I do not propose to attempt its removal. I have known a bit of copper cap remain imprisoned within the lens for seven years with no harm to the opposite eye and then after absorption of the lens, enucleation became needful. There was total detachment of the retina and absorption of the vitreous. I have seen a foreign body suspended in the middle of the vitreous, where it had remained for three years and caused extremely little harm to this eye and none to the fellow. Numerous cases in literature can be adduced to the same effect. Again, the son of a physician lost one eye by the penetration of a fragment of a copper percussion-cap. After a few months severe iritis attacked the opposite eye, but the eye recovered with unimpaired sight. A second similar attack took place a few months later and with the same innocuous result. Was or was not this inflammation sympathetic? The eye at first damaged was not removed despite my remonstrances. On the other hand, I need not say that I have seen multitudes of cases where my conviction that a foreign body would cause the dreaded mischief or did cause it has led me to remove the offending eye. Again, with regard to wounds which involve prolapse of the iris, especially at its periphery, or which enter the ciliary region, I have seen many which did not cause trouble to the fellow and also many which did cause such trouble.

We are obliged rather frequently to remove stumps in which bony growths exist; an ossific change which comes from old inflammatory products in the choroid. We recognise these cases by the hard feel upon squeezing the stump and the pain which pinching produces. These cases are to me puzzling, because if we test the shrunken eye in the case of many persons who wear a shell upon it we shall find marked sensitiveness on pressure.

Again, a shrinking globe, painful on pressure, is by some held to be unconditionally doomed to enucleation in...
the interest of the remaining eye. Marked subtension belongs to many morbid states from which the eye may recover, and in many of these cases it is very painful on pressure. These two symptoms are not warrant sufficient for capital surgery.

Not a few times have I, in face of wounded eyes which still enjoyed a measure of sight, stood out against a proposal for enucleation and been justified in my opposition by the event. Again, I have in two cases done enucleation to arrest the progress of an inflammation in the other eye thought to be sympathetic and gained no advantage; then finding marked tenderness at the apex of the orbit when my finger was deeply pushed in, I have dissected out the remaining piece of the optic nerve or eviscerated the orbit, and the result was the cessation of the sympathetic symptoms. This has also once been the experience of Dr. Hasket Derby, of Boston.

Again, I have published a case of herpes zoster ophthalmicus which destroyed the affected eye by keratitis, and also caused sympathetic inflammation in the other, both eyes becoming blind. A similar but less severe case has recently been under my notice at Moorfields Hospital. In glancing over the various kinds of cases enumerated it seems to be difficult to classify them all under one head; the last-named, beginning as a pure neuralgia eventuates not in a neurosis of the second eye, but in the lesions which are recognised as truly inflammatory. Is this, therefore, an argument in favour of transmission by nerve channels? If at first sight it so appear the answer is prompt that the ulceration of the cornea of the first eye may have caused germ infection and thus germ propagation. But how rarely does this follow in cases known to be microbiotic either as primary keratitis or when following paralysis of the fifth nerve?

I therefore come back to my original statement, that I am not able to hold a decided opinion on the question of how the sympathetic mischief is propagated. Doubtless the theory by transmission of germs along the lymph
channels of the optic nerves is the most enticing, but it lacks conclusive experimental proof, and hints which I have given and other considerations make it difficult of full adoption.

I may not detain you longer, Mr. Chairman. From you and from your colleagues of twenty-five years ago I am glad to acknowledge that I acquired much of the knowledge which has served me since that time. Then, and now also, the removal of the eye was and is practised with great freedom and often most justly. To this proceeding I have contributed no inconsiderable share. But I resort to this operation with much more reluctance and after more critical trial of other methods than formerly.

Wounds with prolapse of iris may be so managed in numerous instances by suitable forceps and methods as to do away with the conditions of danger. I resort to this practice in many cases. A broad and clean iridectomy will sometimes put an irritable eye at rest and protect its fellow. I have by the aid of Wecker's scissors done an iridotomy across the entire iris in a case of traumatic cataract during the stage when the eye was shrinking and have arrested the process and re-established the faulty nutrition and saved the little boy from mutilation. The extraction by suitable measures of a luxated lens will often arrest cyclitis, preserve some sight, and avoid the need of enucleation. In dealing with foreign bodies we are bound, when they are of iron, to resort to the magnet before excision, and to do it under conditions which Hirschberg has shown to be essential to its successful employment. If the magnet is unavailable because we have some other foreign substance than iron, we need not as a matter of routine refuse to attempt its removal, because if we take advantage of all the aseptic precautions which may be employed and whose neglect I may declare to be culpable, we can use surgical measures to remove the offending body which will give a fair chance of success. I may not go further in my remarks. I plead strongly for conservation of the eye. I do not take so lightly as do some
surgeons the being deprived of an eye. Nor am I so enamoured of artificial eyes as some would seem to be. With the experiments of Mr. Mules I have had no experience. But even with the best that can be offered as a substitute, a totally blinded and not seriously disfigured eye which has been rendered harmless is far better than one of glass. The latter causes annoying secretion, must be attended to, must be repeatedly removed, is expensive, and too often can be worn for only a few years because of conjunctival irritation and thickening.

The social position, the age, the sex, the occupation of a patient, bear an important part in making up the decision of the surgeon in a particular case. To save an eye will cost far more time than to recover from the operation of excision. In doubtful cases the intelligence of a patient, the promptness with which he can get competent help in case trouble threatens the second eye, and the likelihood or possibility of his seeking it, all these points must be weighed in deciding what is to be done. But, leaving these considerations to the judgment of the prudent surgeon, I bring before you the other considerations of a purely surgical nature as my argument in favour both of saving eyes and of putting us in the way of a better understanding of the true nature of the process which we have this evening been discussing.

Mr. Nettleship.—I suppose that we may set aside any theory ascribing sympathetic ophthalmitis to the influence of nerve currents passing from the exciting to the sympathising eye, without continuous tissue change (inflammation); such nervous influence can produce no more than sympathetic "irritation."

Of the various other theories, that of blood infection is certainly simple and accounts for some of the phenomena of the disease more easily than any others that have been advanced. Thus it enables us to understand the simultaneous appearance of changes, such as neuro-retinitis and
iritis, in widely different parts of the eyeball. "Sympathetic" inflammation conveyed from one eye to the other by the blood would furnish the best possible proof of the discriminative powers of infective germs; whether, with Mr. Hutchinson, we suppose that these be home-grown, or assume with Professor Berlin that they are always imported and that a perforating lesion of the "exciting" eye is therefore necessary.

But, though most comprehensive and attractive, the blood theory presents some difficulties. Thus if sympathetic disease be carried by the blood it is hard to understand how the incubation period can ever be so very long as we know that it sometimes is. In one case of my own, severe sympathetic iritis came on nine years after an accidental wound of the other eye, and seven and a half years after an iridectomy upon it for the relief of subsequent symptoms. Again, there is difficulty in understanding how the disease can break out in the sympathising eye, as it occasionally does, at a considerable interval after the removal of the "exciting," or rather the "infecting" eye. I find that, in no less than seven of fourteen recorded cases of this kind, a period of from twenty-two to thirty-two days intervened between the enucleation and the first symptoms of sympathetic inflammation.*

* Nine of these cases are given in a paper by the writer in the 'Transactions of the Clinical Society,' vol. xiii, p. 206, and are as follows:

Case 1. Interval between excision and first symptoms in sympathising eye,

22 days.

" 2. Ditto, 23 days.
" 3. Ditto, 20 to 25 days.
" 4. (Hugo Müller), ditto, 5 days.
" 5. (Colsmann), ditto, "a few days."
" 6. (Colsmann), ditto, 6 months; a doubtful case; neuro-retinitis only.
" 7. (Pagenstecher), ditto, 9 days.
" 8. (H. Schmidt), ditto, 4 days.
" 9 (McHardy), irritation 6 days; no iritis 38 days after excision; marked iritis 3 days later (41 days after excision).

The other five cases are:

Case 10. (Snell, 'Trans. Ophth. Soc.,' ii, 19), interval 32 days.
Two other questions may be asked, though they may perhaps be used in evidence for, as well as against, the theory in question. How can the exceptional, but well-attested, cases be explained, in which parts outside the eye, such as the eyelashes, undergo organic change in the course of sympathetic inflammation? And how is it that the exciting eye may be but slightly diseased though the sympathising eye suffer profoundly, even to total blindness? The severity in the sympathising eye must, ceteris paribus, depend on the number of germs which it contains. If these be bred in the exciting eye why does it not always suffer in proportion? On the other hand, if they multiply in, and are not simply carried by, the blood, how, in a severe case, can the other tissues of the body, or at least the blood itself, escape serious change? Whilst if we suppose that the multiplication goes on in the sympathising eye itself, why should it not always take place to the same degree in the exciting eye?

Passing from the blood, as the carrier, to the tissues,—from general infection to local infection or transmission,—we also, however, find difficulty in accepting any of the possible paths along which the disease has been supposed to travel.

The optic nerves, from their large size and their connection at the chiasma, furnish the most obvious route. Deutschmann* has lately proved that if inflammation be excited in the eye of the rabbit by injecting septic material (cocci) into the vitreous, ascending optic neuritis occurs

Case 11. (Frost, ibid., p. 21), interval 22 days.

12. Milles (ibid., vol. iii, p. 61), interval 22 days.

13. Frost (ibid., vol. iv, p. 80), interval 5 days.

Several others with an interval of a few days have been recorded, and probably such are not rare.

Case 14. Ayres gives a case coming on 12 months after excision, but there was reason to suspect that it was set up by changes in the orbital tissues (‘Knapp’s Archives,’ xi, 199). I have excluded this case and Case 6 from the above statement, the seven examples referred to being Cases 1, 2, 3, 9, 10, 11 and 12.

* ‘Ophth. Review,’ ii, 22; iii, 305; iv, 12.
followed by neuritis of the other eye, and that this neuritis is itself followed by choroiditis and disease of the vitreous. The latter phenomena, so far as they go, resemble those of sympathetic inflammation. Finding that inflammation was present along the whole extent of both optic nerves, including the chiasma, and that there was, at least in some cases, meningitis over the chiasma, Deutschmann concluded in favour of the transmission of septic inflammation by way of the optic nerve. There is a certain amount of clinical evidence in favour of this, the earliest, view; the patient sometimes notices failure of sight before any other symptom, and the surgeon sometimes finds papillitis or papillo-retinitis as almost the first recognisable change.*

* Alt gives details of the following seven cases of "Sympathetic Neuro-Retinitis" in the 'Transactions of the International Ophthalmological Congress,' 1876, p. 37.

In Case 1 (v. Graefe) serous iritis was proved within a few days of the onset of the failure of sight and its absence earlier cannot be assumed.

In Case 2 (v. Graefe) there was retinitis and disease of vitreous in a young man whose other eye had been long blind from total detachment of retina, but there is no proof that the disease was sympathetic at all.

In Cases 3 and 4 (Pooley) neuro-retinitis merely accompanied the ordinary irido-choroiditis of sympathetic disease.

In Case 5 (Alt) a man, act. 35, had lost his left eye from suppuration of the cornea fourteen years before, and had worn a glass eye for years over the stump. Changes just like those of albuminuric retinitis were found in the right eye, and three weeks after this diagnosis had been made, severe irido-choroiditis had set in with + T., and the eye eventually became blind and soft in spite of iridectomy. There is no note of the state of the urine.

In Case 6 (Alt) iritis was present at the same time as the venous congestion of the disc and retina. The eye was subsequently lost by violent inflammation ending in staphyloma and perforation.

In Case 7 (Alt), a negro, act. 38, had repeated attacks of inflammation in the right eye for twelve years, ending in "total staphyloma." He came for failure of the left eye of six days' duration, going to nearly total blindness; there was diffuse neuro-retinitis and a few haemorrhages, with no opacity of the media. The right eye was excised, the patient treated with corrosive sublimate; and the left rapidly recovered. The urine contained no albumen but there is no note as to sugar. Syphilis is not mentioned.

**Other Cases.**

**Case 8.—Benson ('Ophth. Review,' ii, p. 140) gives the case of a girl, act. 8, in whom sympathetic inflammation of the left eye commenced about a
It is also a fact that inflammation does ascend the human optic nerve, for a certain distance at any rate, in some cases of traumatic inflammation of the eyeball; such ascending neuritis is well shown in the drawing I have round, and Becker has published a case in point.*—But it should be observed that iritis, or at the least keratitis punctata, has been present in nearly all the cases which have shown early retinal or neural changes. Now, failure of vision, papillo-retinitis, and keratitis punctata, may readily be accounted for by inflammation of the choroid, whether this be general, or be confined to the posterior region of the eye, as we may well suppose that

month after a wound of the right with prolapse of iris, in the ciliary region. When first seen, a few days after the mother had noticed irritability of both eyes, the left showed some ciliary congestion and discoloration of iris, and slight diffuse muddiness of the vitreous, but no synechia and no keratitis punctata; the disc was somewhat swollen and hazy, and the veins enlarged and too tortuous.

Case 9.—Nettleship ('Clin. Soc. Trans.,' xiii, 206). Man, at. 20; sympathetic inflammation of left, terminating in blindness, began (twenty-two days after excision of right) with rapidly-increasing dimness; he was not seen till a week later, when there was haze of o. d., engorgement of retinal veins, ciliary injection and commencing plastic iritis.

In the same paper two other cases bearing on this point are quoted from H. Müller and Colsmann.

Case 10.—Frost, 'Ophth. Soc. Trans.,' vol. iv, p. 88, mentions a case of sympathetic ophthalmitis which was under the care of Mr. Tay, in which the changes in the anterior part of the uveal tract were comparatively slight, and in which perfect recovery of vision took place, but in which optic neuritis was present, the swelling of the disc persisting long after all other symptoms had disappeared.

Case 11.—Brailly has recorded a case in which failure of sight with slight papillitis, followed by partial atrophy but considerable improvement of vision, came on about two weeks after a complicated wound of the other eye; the media were perfectly clear and the iris healthy from beginning to end. The case reads, so far as the "sympathising" eye is concerned, like one of the ordinary cases of acute "retro-bulbar neuritis" ('Trans. Ophth. Soc.,' iv, p. 87).

Case 12.—Deutschmann gives the case of a boy, at. 15, in whom sympathetic inflammation appeared first as congestion of the disc with dilated tortuous vessels and diffuse retinitis; the vitreous and iris were normal, but there was extensive keratitis punctata ('Ophth. Review,' iv, p. 17).

*See 'Ophth. Review,' ii, p. 22.
it sometimes is. I think, therefore, that the clinical proof of transmission along the optic nerve must rest upon the production of cases in which changes appear at the disc some time before any other changes in the eye whatever, and I should expect that failure of sight in such cases might precede the visible alterations.—Again, if sympathetic disease were transmitted along the optic nerve or along the lymphatics,* or blood-vessels† we should expect often to meet with clinical evidence of basic meningitis. Snellen,* indeed, has met with one such case, but as the patient recovered the diagnosis could not be verified; Mooren‡ is said to have seen similar cases. Then we should expect that in mild cases the disease would sometimes be limited to the optic nerve, but I believe that this has not been noticed except in Brailey’s single case.§ And it may have been mentioned that we have no clinical ground for supposing that inflammation which begins independently in the orbital part of one optic nerve (“retro-bulbar neuritis’) ever spreads upwards to the other, though we do know that it goes downwards to the optic disc of the affected eye.

The chief difficulty to my mind in believing that sympathetic inflammation is transmitted along the filaments of the fifth, or of the sympathetic nerve, is that the path is so long and so very narrow. The mere fact that histological changes have not often been found in the ciliary nerves is not of much value, for these nerve-twigs are so small, and such short portions of them are available for examination, that slight inflammatory changes might easily escape detection—might, indeed, be almost impossible to detect.

Dr. Mules pointed out the necessity for careful discrimination between “sympathetic irritation” and

† Samuelsohn, ibid., p. 45.
‡ Mooren, see 'Ophth. Review,' iii, 305.
§ Brailey, loc. cit.
"sympathetic inflammation." The latter he holds to be essentially bacterial, the former a true reflex neurosis. These two have nothing in common except that sympathetic irritation may, and probably does, dilate the perineural lymph spaces, thus facilitating the movements of the bacterioid bodies. He suggested that the differences in time from the exciting cause to the secondary outbreak depended on the movements of these bacterioid bodies which might be deferred indefinitely. Having no knowledge of the life-history of these organisms it was impossible to forecast whether or not recurrent attacks would take place under injudicious irritation. In reference to the President's view, if the bacterial theory be the correct one, obviously that introduced by the President cannot be, but Dr. Mules hopes that in evisceration we shall find a means for the prevention of sympathetic inflammation and that a good deal of the mystery surrounding it may be cleared up.

Dr. Brailey thought that all that was required for the purposes of the present discussion was that he should combat the President's view without being under the obligation of championing a rival one. The fact that tumour cells were sometimes carried from the eye through the blood to distant parts, as, for example, in choroidal sarcoma where secondary deposits in the liver were not infrequent was so far in favour of it. But, on the other hand, he knew of no case where a choroidal tumour had secondarily infected the uveal tract of the opposite eye, though transmission along the optic nerve was possible and even probable in retinal glioma. In the same way a suppurative choroiditis might produce localized patches of pus on the meninges of the brain without any evidence of direct continuity, but he had never known suppurative changes to be produced in the eye of the opposite side. To compare the President's view with one, perhaps the most commonly accepted and plausible, that of direct transmission along the optic nerve or its intersheath
space; there was some little physical evidence in favour of this, for example: (1), the case observed and recorded by Snellen, where an injured eye produced not only a severe sympathetic ophthalmitis but also total deafness with all the symptoms of an acute meningitis; (2), the experiments of Deutschmann, which showed a papillitis of one eye after injections into the vitreous of the other; and last but not least, the very numerous pathological examinations of excised eyes, both sympathizing and exciting, which have demonstrated inflammatory cells in the intersheath space of the optic nerve and within the nerve itself. Against this evidence there was positively nothing in the way of physical evidence to be advanced in favour of the plausible, comprehensive, and attractive theory which formed the subject of the President's paper.

Mr. Hutchinson.—In reply I have to thank those who have taken part in the interesting discussion. With those who are cautious in accepting any definite theory I am fully in accord. Our facts do not as yet warrant a decided creed. I am sure, however, that we shall be helped towards arriving at such by clear comprehension of the possibilities before us. For myself, in spite of the criticisms which it has received, I see no reason to regard less favourably than I did at the first, the explanation which I ventured to suggest. Mr. Power has asked me how it explains the more frequent occurrence of reflex ophthalmia in children. Well, it so happens that it is precisely in children that we witness the most common examples of multiple periostitis, the malady with which I attempted to establish an analogy. It is in children that cell-growth processes are most rapid and cell infection most easy. It has been objected also, that in some cases the interval is very long. To this I would reply, that the same irregularity of interval is witnessed in the case of tumours. Without our knowing why, it is certainly the fact that some tumours infect distant parts speedily and others very slowly. It is probable that such infection is a matter to
some extent of accident. It may easily be the fact that many abortive infections take place and that many eyes are temporarily threatened, in which the process never rises to any height. So also it is not difficult to meet the suggested difficulty (Dr. Noyes, I think), that cases sometimes recover for a time and then, on some undue exposure, relapse. This is what we ought to expect, for an organ in which once an inflammatory process is set going is very likely to submit to some extent to remedies, to exclusion of irritants, and to relapse when treatment is suspended. The occasional benefit and the frequent failure of the excision of the exciting eye are also to be explained, on this hypothesis at least, as easily as on any other. If we remove the exciting eye after the process has begun in the fellow we take away the sources of infective material and thus cut off further supplies and in this way benefit the second, but, supposing the process to be well established, we are clearly too late to influence it in any other way. Against the older theories that the process of inflammation travels by continuity in the sheath of optic or other nerves which several speakers have been inclined to fall back upon, we have an objection which is, I think, strong. How comes it that an inflammation which proves so virulent and disastrous when it reaches the eye never produces any effects, irritative or otherwise, in the long tract of structures which it passes through on its way? Why do we never get a basal arachnitis? This basal arachnitis is very common, let us remember, when, in consequence of fractures of the petrous bone, inflammation originates and travels along the seventh pair of nerves. In the able speech which was made by Dr. Noyes I confess I thought that he forgot for the moment to draw a sufficiently clear distinction between reflex irritation and reflex inflammation. All that we observe as to irritation without cell effusion is easily explicable by reference to the fifth nerve. Even perhaps some dilatation of blood-vessels and some slight changes in structure may be so explained. But the case is quite different when we come
2. On the condition of the ciliary nerves in certain diseases of the eye.

By W. A. Brailey, M.D.

These examinations, comprehending ninety different eyes, were made primarily with a view to ascertain whether structural changes of the ciliary nerves had anything to do with the transmission of sympathetic disease. But certain other morbid conditions are also embraced in the inquiry, such as wounds and ulcers of the cornea, iritis, suppuration...
of the globe, and tumours. In addition, notes have been made as to the condition of the ciliary arteries and other blood-vessels traversing or nourishing the sclerotic. It must be remarked that, in every case examined, the morbid process, whatever it may be, has been sufficiently severe or advanced to have caused the removal of the eyeball.

In about half the cases of primary glaucoma the nerve cannot be declared other than normal in structure. But in some (22 per cent.) there is slight evidence of neuritis. This, however, being slight, is not incontestable, as will be readily understood by anyone experienced in the microscopic examination of nervous tissues, since, in the normal condition, a slight increase in the thickness of the section will bring into view a much increased number of nuclei.

Leaving then this point not perfectly decided, we pass on to remark the very considerable size of the nerve, which often prevails in primary glaucoma. In about two thirds of such cases the size much exceeds the normal mean. But in a few no excess can be remarked, and, on the other hand, rare cases of various forms of disease associated with normal tension are observed, in which the nerve is as large as in any glaucomatous eye. The cause and significance of this enlargement are not apparent. In many cases the axis cylinders, and indeed the nerve-fibrils generally, appear large, though in about half the cases without the slightest indication of neuritis. Indeed neuritis alone cannot be its cause as this condition exists without enlargement in many of the eyes with normal tension. Possibly it may be due to an infiltration of the nerve with serum and consequent swelling of its fibrils. The enlargement is indicated on the exterior of the sclerotic, especially in the case of the inner long ciliary nerve, by a wide dark-blue streak where the sclerotic appears, and is, indeed, in reality, thinner than elsewhere. The enlargement of the accompanying artery, usually associated with glaucoma, will also share in causing the thinning.
There are certainly many cases of absolute glaucoma where the nerve, whether enlarged or not, shows degeneration of its structure, the axis cylinders being large and indistinct, or not visible at all. This is probably the cause of the corneal anesthesia noticed in advanced cases of glaucoma. It is in all probability merely a very late result of the pressure.

In eyes which have excited sympathetic disease the nerve is usually normal in structure, though it may show neuritis of much the same degree and probably in about the same proportion of cases as in glaucoma.

But inflammation around it, clearly derived from that round its accompanying artery, where it is more marked, is of very common occurrence. Such inflammation is seen, not only in the immediate neighbourhood of the long ciliary artery and nerve, but round the other blood-vessels nourishing or perforating the sclerotic; and the sclerotic itself is, in the great majority of cases, a little inflamed. From certain cases in which this last has been wanting, it would appear that the inflammation spreads from the blood-vessels to the sclerotic rather than the reverse way.

As to the sympathetically inflamed eye, we find, in two out of the three cases examined, a little inflammation round the vessels and a trifling amount, less than in the exciting eye, in the sclerotic itself. The third case presents no trace of such changes.

In serous iritis, not sympathetically excited, inflammation is found round the blood-vessels in a marked degree in each of the two well-marked cases examined, but there is little or none in the sclerotic generally.

In suppurative panophthalmitis where inflammation round the arteries and also in the sclera itself reaches its maximum, we still find the former vastly more marked than the latter. Both, as has just been implied, are more marked here than in eyes exciting sympathetic disease. The outermost layers of the sclerotic are those most affected.

In wounds or ulcers of the cornea the scleral tissue,
together with its contained blood-vessels, participates in the inflammatory changes, and this more especially in its outer layers. In such cases there may be inflammatory cells in the optic nerve itself and still more on the loosely arranged fibres between its sheath, while the vitreous is still normal and no inflammatory cells are found on the surface or round the arteries of the optic disc. It therefore appears as if the inflammation had in such cases travelled from the cornea backwards along the scleral tissue, rather than by the aqueous humour and iris, to and along the vitreous. But in the more severe and more advanced cases which form the great majority of those coming under examination, the vitreous, from the physical and microscopical changes which it presents, constitutes the more obvious route. And the presence of cells in the swollen disc and on it strengthens this supposition. It is likely that the inflammation taking the scleral route, though comparatively small in amount, is the earliest to arrive at the optic nerve and produce an optic neuritis. It is not improbable, however, that the coats of the contained blood-vessels may be as much or more a factor than the proper tissue of the sclerotic itself.

Concerning iritis, comprising under this head inflammation of the whole uveal tract which are neither suppurative nor causing sympathetic disease nor produced sympathetically, it is impossible to lay down a universal rule. It would appear that inflammation of the nerves themselves is here more common than either in glaucoma or in eyes concerned in sympathetic disease. Sometimes, as in the two specimens shown, one half the nerve is much inflamed, and the other half perfectly normal. In each of these cases the inflammation has clearly spread to the nerve from its companion artery.

But inflammation round the artery and nerve, though in some cases particularly well marked, as in the two microscopical preparations exhibited at a former meeting, is on the whole less common than in the other conditions mentioned.
In intra-ocular tumours the occurrence of inflammatory changes at parts not in the immediate neighbourhood of the tumour, for example, in the optic nerve and iris, round and in the ciliary nerves, and in the space between the optic nerve-sheaths, is in some cases, notably those of choroidal sarcoma, very striking. It may be said that these changes are not of an inflammatory kind, but merely stages in the spread of the tumour to the regions in question. It is, however, clear that as far as the iris is concerned this is not always so, since we have frequent ocular demonstration by the presence of synechiae, discolouration of the iris and ciliary injection, that a tumour may in reality produce a true iritis.

To sum up our results as regards sympathetic disease: inflammation of the ciliary nerves is not an essential factor in its production. Indeed, it is doubtful whether it can ever be a cause, since it is not infrequently entirely absent, both in the exciting and sympathetically affected eye (see the microscopic specimens exhibited), and since, moreover, it is markedly present in some cases of irido-choroiditis (see others similarly exhibited) which neither have produced nor, judging from their microscopical characters, are likely to produce sympathetic disease.

Inflammation round the ciliary nerves, though more often present in both the exciting and sympathising eye, is not an essential cause, since it is usually less marked than the inflammation round the accompanying artery from which it has evidently spread.

Inflammation round the long ciliary arteries, though a more probable cause than that round or in the nerve, being more marked and more constant in addition to offering a shorter and more direct route from one eye to the other, whether passing by the arterial sheaths throughout, or from them to the loose tissue between the sheaths of the optic nerve, have still little or nothing to do with the transmission for the following reasons:—It may be wanting entirely (see specimens). It is at least as well seen in recent cases of suppurative panophthalmitis, a
condition remarkably free from liability to sympathetic complications.

It may also be noted that inflammation of the sheaths of the arteries, very striking both as to extent and severity, occurs in the disease known as iritis serosa, one little liable, as far as we know, to affect the fellow-eye sympathetically.

3. Microscopical specimens showing an exceptional condition of the ciliary nerves in three cases of uveitis.

By W. A. Brailey, M.D.

A. Inflammation round internal long ciliary artery extending into the contiguous portion of the corresponding long ciliary nerve. Eye excised on account of iritis, with recurring tension one month after the extraction of cataract. The eye was glaucomatous prior to the extraction. The other eye shows very doubtful slight sympathetic irritation.

b. Specimen very similar to the above, only the inflammation is less severe. Iritis following on iridectomy nine months ago, intended to be preliminary to cataract extraction. Other eye, sympathetic irritation.

c. Severe neuritis of long internal ciliary in an eye excised on account of severe irido-choroiditis of spontaneous origin. Other eye normal.

(Card specimen. January 8th, 1885.)
4. Microscopical specimens showing the condition of the ciliary nerve in a case of sympathetic disease.

By W. A. Brailey, M.D.

D. The sympathising eye (left) excised on account of loss of sight, severe inflammation and pain. History:—The right (exciting) eye was operated on for cataract eleven months ago. Four months later an opaque pupillary membrane was needled. The left remained perfectly free from inflammation. Five months later (two months ago) the left was found to be quite blind from iritis, presumably sympathetic. The long ciliary nerve and artery are perfectly normal.

E. Perfectly normal long ciliary nerve and artery of an eye, which has been excised on account of exciting sympathetic uveitis. It was injured thirteen weeks ago. The adjacent choroid is in a state of severe inflammation. (Card specimen. January 8th, 1885.)

5. Sympathetic disease of one week's standing indicated by rigidity of the pupil and a history of two attacks of swelling of the lid, with pain in and about the eye.

By W. A. Brailey, M.D.


The right became red a few days after the other, but it caused him no anxiety till one week ago, when
swelling of the lid came on with pain in and about the eye. The temple was sore, as if it had been bruised. This feeling subsided, but in two days recurred. Now: Pupil rigid, does not dilate fully to atropine. At its edge the crenated uveal pigment is distinctly visible. There is considerable conjunctival injection on both the inner and outer sides of the cornea, apparently of the nature of pterygium. He has been abroad. $V = \frac{6}{5}$.

December 19th.—Went home. The pupil will now not yield at all to 1 per cent. atropine solution applied thrice daily for some days. Both eyes are considerably better as regards injection.

Under treatment with local abstraction of blood, exclusion of light and mercury, the left eye became perfectly quiet, and the corneal opacity became much thinner. Concurrently with these changes, the symptoms in the right gradually subsided and the pupil gradually yielded fully to atropine, leaving no synechiae.

(Living specimen. December 11th, 1884.)
V. PANOPHTHALMITIS.

1. **Purulent irido-cyclitis with opaque vitreous in a young child suffering from a febrile illness,—? Pyæmia,—? Meningitis.—Death in six weeks;—Cerebro-spinal meningitis from purulent disease of the middle ear.—The meningitis and eye disease probably pyæmic.—Examination of the eye after death.**

By E. Nettleship.

In a paper which I * read before this Society during the last session but one, upon cases of destructive ophthalmitis in young children simulating glioma, attention was asked to the frequency with which such eye attacks coincided with some general illness, an illness often accompanied by cerebral, and sometimes by pyæmic symptoms. The case narrated below is interesting because it was watched from its commencement to its fatal termination and was examined carefully after death. The child died of cerebro-spinal meningitis, which no doubt was caused by purulent otitis. The ear-disease was not recognised during life, and, partly on this account, the nature of the major disease remained doubtful until the post-mortem. It seems most probable that the eye-disease, which was purulent irido-cyclitis with suppuration of the vitreous, was pyæmic; and the meningitis itself was perhaps of the same nature, at least in the beginning, for no continuity could be traced between it and the disease of the ear. Viewed in this way the case perhaps throws light on the rare cases in which death occurs from meningitis after excision of an acutely inflamed eyeball, for in these again there is, I believe, seldom any evidence of pus passing along the

* Vol. iii, p. 36.
nerves from the orbit to the brain. Both may perhaps be called local pyæmia, or spreading phlebitis; the eye or the inflamed orbital structures being the source of infection in the cases just referred to, as the ear was in the present instance. If, however, pseudo-glioma in children is often a result of pyæmic inflammation of the eye, or of suppurative phlebitis of ocular veins, we must assume that recovery from pyæmia is comparatively common in very early life; adults with pyæmic inflammation of the eye very seldom live long enough for the eye to pass into the corresponding state.

Ethel E—, æt. 2½, was brought to the Eye Department at St. Thomas's Hospital, on the 5th of last March (1884), with some haze of the l. cornea, purulent infiltration of its lower part and semitransparent white lymph blocking the pupil and lying in flocculi in the anterior chamber. There was slight chemosis and t. was thought to be increased; the eye was not tender. The temperature was 101·5° and the child, being ill, was taken in. The previous history was negative. The present illness was attributed, but without reason, to a slight blow on the face or head from a fall whilst running about on February 27th; on March 2nd the child shivered, vomited, was thirsty, and had some diarrhoea; she was delirious for the next day or two, but there was no cough or rash; the eye was first noticed to change on the 3rd, and she was brought to the hospital (as stated above) on the 5th.

On the day after admission (March 6) she lay usually on her r. side and cried when touched. Temp. 102·2° to 103·6°; cheeks flushed; there seemed to be tenderness about the knee-joints but no swelling; no rash except a few ill-defined papules (not varicella). Urine sp. gr. 1028, contained some albumen. Chest and abdomen normal, no fits and no vomiting. Later in the day a small fairly-defined swelling was found over the head of the r. fibula; it moved with the skin and seemed to be very tender. No other swellings.

7th. Some crepitation over the root of the r. lung.
Temp. 102°4 to 104°4; urine, no albumen. Both knees seem slightly swollen and tender. Eye unchanged except that the lymph has partly subsided, leaving the iris more visible.

8th.—Irritable and drowsy; still lies usually on r. side with hand under head; no otorrhœa.* Takes milk well. Swelling over right fibula smaller. L. eye: lymph disappearing from anterior chamber and pupil nearly clear; no reflex from fundus. R. eye (under atropine) examined for first time, now shows marked papillitis; veins distended, disc swollen and hazy, no hæmorrhages. In the opinion of Dr. Percy Smith, the Resident Assistant Physician, who watched the case carefully with me throughout, this papillitis was the only symptom pointing strongly to meningitis.

11th.—Child much brighter, now quite conscious. Some diarrhœa for last three days (three to four motions daily), but not like typhoid. No spots. R. disc less hazy. Still swelling over r. fibula, but now no tenderness.

13th.—Restless; still diarrhœa; no spots; moves legs well and has no swelling of joints. Lymph has almost disappeared from l. anterior chamber; pupil round and half dilated; t. n.

27th and 29th.—L. eye quiet; anterior chamber of good depth and clear; by focal illumination a whitish reflex from the vitreous. Child is wasting and takes very little food; very fretful, especially if moved, but does not speak. Well-marked tache cérébrale. No diarrhœa for some days. The swelling over r. fibula has quite gone. Some dulness at base of l. lung with somewhat tubular breathing; no crepitation. Has vomited to-day after food.

April 6th.—L. eye, T. — 2; beginning to shrink, but still shows congestion of sclerotic and yellow reflex.

11th.—Vomiting after all food. Much weaker. Inunction of cod-liver oil.

14th.—Keeps food down well. More lively.

* The ear, however, was never thoroughly examined.
15th.—Temperature has been normal for a week past, having been previously from 100° to 104°, but child not improving. L. eye shrinking rapidly; examination difficult, but iris does not appear retracted at periphery.

The child gradually sank, the vomiting having returned, and died on the 18th, about six weeks after admission. Temperature went up to 100·6° the day before and at the time of death.

No definite diagnosis was ever made. The occurrence of papillitis in the other eye of course pointed strongly to meningitis, and if the disease of ear had been recognised this suspicion would have been immensely strengthened. There were, however, no convulsions or screaming and no definite retraction of the head, although for a day or two the head was turned to one side and the child resisted attempts to put it straight.

The suspicion of pyæmia aroused by the tenderness of knees, and especially by the swelling over the r. fibula, was not confirmed by any other symptoms.

There was nothing either in the temperature or symptoms characteristic of typhoid or of any other exanthem, nor of general tuberculosis. On the whole we were most prepared to find tubercular meningitis.

The post-mortem examination was made the day after death by Dr. Hadden. The l. eye was removed, frozen, cut in two horizontally, and a rough sketch made of the section. The drawing now shown was made by Miss Boole after hardening in Miller's fluid.

Acute cerebro-spinal meningitis; Disease of ears; ? Local thickenings of capsule of liver.

Body emaciated. Calvaria healthy. Dura mater healthy. Sinuses contain only non-adherent clot. No meningitis at vertex. Decided excess of subarachnoid fluid, both on surface and in ventricles. The crura cerebri, optic tracts, nerves, and chiasma, pons, and fore part of cerebellum, are covered with thick yellowish-white
lymph, more abundant on the right side. No lymph in fissures of Sylvius, and no decided tubercles here or elsewhere in the brain. Walls of lateral ventricles softened. No tubercular mass in the brain.

On removing dura mater at base the roof of the middle ear on right side has a slightly yellowish-white appearance, and is somewhat softened. The membrana tympani is soft and yellow, partially detached and perforated in the centre. The small bones are not diseased. The middle ear and mastoid cells contain a good deal of thick pus. There is no caries of the temporal bone superficially, and the dura mater over it is quite healthy in appearance. The lateral and cavernous sinuses are also healthy.

Right eye.—Generally shrunken; in front the vitreous is purulent. The retina is extensively detached, much thickened, and opaque.

Heart.—Healthy. No pericarditis or pleurisy. No tubercles on serous membranes.

Lungs.—Some interlobular emphysema. No tubercles. Organs otherwise healthy.

Liver.—There are numerous small, white, dot-like bodies in the capsule and extending a little way below. They are not raised. They are apparently not tubercles, new growths, or abscesses; ? local thickenings of the capsule.

Kidneys.—Healthy.

Intestines.—No ulceration.

Spinal cord.—On opening the theca the whole posterior half of the cord from the upper dorsal region downwards is covered with thick yellowish-white lymph. The cervical part is not affected. No lymph seen anteriorly. No tubercles visible. No obvious change in cord itself.

The left ear was also examined. The membrana and small bones were healthy, but there was some pus in the middle ear and in the mastoid cells. No superficial caries.

(October 9th, 1884.)
VI. VARIATIONS OF TENSION.

1. *Persistent diminished tension after blow with nail five months ago.*

By W. A. Brailey, M.D.

Daniel S—, aet. 50, plasterer, received a blow with a nail on the left eye five months ago. Next day he could see nothing plainly, and could not bear the light. These symptoms persisting, he was brought to Guy's Hospital, by Dr. Long, of Deptford, six weeks later. Then he had T = 1; a. c. shallow. Good fundus reflex, but no details visible. V = 6/60 doubtfully. Slight photophobia.

Since then his vision has slowly improved, being now 6/60 clearly. T. still —1. There is a superficial opacity near the outer corneal margin. In the iris corresponding to the upper edge of this, is a brown spot, apparently a congenital pigment spot only. The disc is now fairly visible. There is a thin patch of opacity, about 2 m.m. square, on the posterior lens capsule.

*(Living specimen. January 5th, 1885.)*

July 9th.—V. = 6/60 doubtfully; a.c. extremely shallow. T. = 1. The lens now presents a small central superficial opacity. Two months later the condition was just the same.
VIIT. DISEASES OF THE LENS AND CAPSULE.

1. Note on the spontaneous disappearance of diabetic cataract.

By E. Nettleship.

My attention was drawn to this subject by the publication, by Dr. Tannahill, of a case of spontaneous disappearance of diabetic cataract, in the 'British Medical Journal' of January 31st last, p. 226. Dr. Tannahill's communication was brief, but he has very kindly placed at my disposal some further particulars, which add so much to the interest and importance of the case that I venture to lay their substance before the Society.

It would seem that the spontaneous disappearance of opacity of the lens (not the spontaneous absorption of an opaque lens) in persons with diabetes, although not unknown, is, so far as published records show, very rare. Two cases detailed in Dr. J. Seegen's 'Diabetes Mellitus,' &c., published in 1870, and alluded to by Becker in his chapter on the lens, and by Förster in his chapter on general diseases, in 'Graefe und Saemisch's Handbuch,' are in fact the only ones that I have been able to find; no cases are recorded in 'Nagel's Jahresbericht d. Ophthalmologie,' from its commencement in 1870 to the present time. As the subject is evidently one of pathological interest, I propose to quote the principal facts of the three cases (Seegen's two, and Tannahill's one), although I have no fresh material of my own to place with them.

Seegen's first case (l. c. Case 40, p. 212) is as follows:—The patient, a man aet. 39, was first seen in July, 1863, about six months after symptoms of diabetes had
been first noticed. He was then much emaciated and very feeble; the skin dry, the urine (6500 ccm. daily) containing 7 per cent. of sugar; lungs healthy. The lenses were cataractous and hazy, and for some weeks he had seen all objects as if through a mist. Under treatment at Carlsbad the urine quickly diminished in quantity, and the crystalline lenses cleared, V. beginning to improve eight or ten days after admission. About the middle of August, that is after about one month's treatment, the quantity of urine was reduced to 3000 ccm.; the L. lens was perfectly clear, and in the R. only a slight trace of opacity remained, and V. was quite clear. Although the quantity of urine was much reduced, the percentage of sugar remained the same as on admission. Nine months later, May, 1864, Dr. Seegen found the lenses quite clear, but the patient still much emaciated, and passing much sugar. The following winter his general state became worse, V. again became impaired, and before he died in the spring of 1865, he had become blind; although Dr. Seegen does not actually state that the lenses again became opaque, there is no doubt from the context that he intends this to be understood.

Seegen's second case (No. 113) is not recorded in the same detail. The patient was a woman whose symptoms of diabetes began in the autumn of 1867, when she was 55 years old; at Christmas her sight began to fail and became misty, and after a time it became so bad that she could no longer read. She first came under medical care in February, 1868, when Professor Gerhardt diagnosed diabetes. Under his treatment the sugar disappeared from the urine, though the sp. gr. kept high, and at the same time the opacity of the lenses diminished. In the following May Dr. Seegen found still some opacity of the lenses, though the woman could now read newspaper print. At this time she was very thin, but the urine free from sugar, and sugar did not reappear when she was allowed starchy food. In this case we have no description of the state of the lenses when V. was at its
worst, and can only infer that they were at first opaque from the statement that they afterwards cleared.

The following is the substance of Dr. Tannahill's case, taken chiefly from the additional notes with which he has supplied me since the case was published:

The patient, a coalminer, began to be very thirsty, and to pass more water than usual in April, 1882, when he was 43 years of age. About the same time his sight began to fail and continued to get slowly worse, but for several months he was able to read fairly well without the help of glasses. He left off working in the coalpit soon after his symptoms began. On the 28th February, 1883, he was committed to the Wakefield prison for fourteen days for an assault, and it was there that Dr. Tannahill saw him. Both lenses were cataractous (symmetrical nuclear cataract), the L. being more opaque than the R., the opacity being easily noticeable from a distance of several feet when the man faced the light; his sight was so defective that he could not recognise people or large objects, and could not read ordinary print, either with or without glasses, but he had good perception of light. He weighed 117 lb., his weight when in health being about 150 lb.; he was much emaciated and his skin dry; he had had boils. The urine was highly saccharine, but the quantity of sugar fell to almost one half very soon after the commencement of proper diet and opium. He was under care only fourteen days. During that time his lenses gradually cleared and sight improved, and when he was discharged, on March 13th, "no trace of haziness or opacity could be detected in the lenses, and he was able to read very small print with the naked eye;" his general health had also improved. He stated that a brother had died of "diabetes" at the age of 24, but he had no affection of sight.

I append Dr Tannahill's tabular statement of the diet, and of the quantity of urine and sugar passed daily between March 3rd and 13th; the sugar was estimated by Pavy's method.
I believe that no satisfactory explanation has yet been given of the manner in which diabetes produces cataract; even as to the comparatively simple question whether the lens itself contains sugar in these cases, different observers have obtained contradictory results. The theory that the cataract is due to permanent degeneration of the lens fibres, owing to the general cachexia, certainly seems not to be borne out by the cases I have quoted. The disappearance of the opacity from the lens, and of the sugar from the urine at the same time, and at least in one case (Case I) the reappearance of the cataract when the diabetes again increased, seems to support the theory which ascribes diabetic cataract either to the presence of sugar in the lens, or to the abstraction of water from the lens fibres owing to increased density of the blood.

(May 14th, 1885.)

* Opium 2 grains daily began.
2. Case of black cataract.

By Henry Power.

Caroline H—, æt. 43, admitted into St. Bartholomew's Hospital on May 29th, 1885, for cataract. She has always been myopic. She thinks she was struck on the right eye sixteen years ago. It gradually became dim. Vision of left eye became obscure seven years ago.

Both eyeballs are very prominent. There is a very dark striated opacity in right eye; pupil dilated, slight external squint. The fundus cannot be illuminated. Left eye shows general haze in lens.

June 1st.—The lens was removed without iridectomy. Lens quite black.

July, 1885.—R. p. l. good. L. counts fingers.

(Card specimen. June 4th, 1885.)

Note, July, 1885.—The second eye was operated on since, also without iridectomy. The lens, which was of a very deep brown tint, escaped on slight pressure and was followed by fluid vitreous. Some hours after the operation the patient had a violent attack of retching which led to haemorrhage into the vitreous and loss of the eye. After the attack of vomiting the patient stated that she was liable to periodical attacks of retching, a circumstance which she had not previously considered requisite to communicate.

3. Symmetrical dislocation of lenses upwards; congenital.

By F. R. Cross.

Ada L—, æt. 8. Each eye virtually similar. Tremulous iris. Lower edge of lens just below upper edge of pupil. Six months ago V. = \( \frac{2}{6} \), J. 1, p. r. \( \frac{2}{2} \) inches.
DISEASES OF THE LENS AND CAPSULE.

January 5th, 1885. — \(\frac{20}{200}\), \(\circ + 10\) D. \(\frac{20}{100}\) and J. 1, p. r. 2½.

*Under atropine sight better*, \(\circ + 11\) D. \(\frac{20}{60}\). Edge of lens distinctly crosses pupil. Lens moveable.

Ophthalmoscope shows a double disc image. No distinct diplopia (monocular).

As a child her attention was not readily drawn to objects. Sees best on a bright day; equally well by gas or daylight, and by day and evening.

*Atropine improved vision* somewhat. The sight (or judgment of external objects) is better than six months ago, the parents say. She is among children of her own age at school.

No *family history* towards dislocated lens. Parents see well. A sister is myopic.

(Card specimen. January 8th, 1885.)
VIII. DISEASES OF THE RETINA.

1. Remarks on three recent cases of detachment of the retina.

By W. A. Brailey, M.D.

As the subject of retinal detachment has been one of widely differing experience on the part of different observers, I venture to give a brief account of the last three cases that have come under my own care.

As will be seen, the general results are decidedly favourable, but we cannot on that account blind ourselves to the fact that the vast majority of cases encountered are of a widely different character.

In each of my cases the detachment occurred rather suddenly and its duration is comparatively short.

Anyone's observation of excised eyes will not fail to convince him that the vast majority of detachments are incapable of any remedy. For in many the vitreous is shrunken to a small size and is of tough structure. Its connections with the retina are, moreover, very firm. And, even in those where the retinal cavity is empty, or merely contains traces either of vitreous itself or of any substance allied to or derived from it, the retina is for the most part disposed in folds so firm and rigid as to offer considerable resistance even to mechanical post-mortem reapplication. The cases in which the detachment is local and dependent on an accumulation of fluid between retina and choroid, in association with a healthy-looking structure of the vitreous, are decidedly rare if we exclude those in which the lesion is the direct result of intra-ocular sarcoma.

Case 1. George Robinson, æt. 43, driver of a cart. He came to the Eye Department, Guy's Hospital, as an in vol. v.
out-patient on Friday, July 18th, 1884. For the last month he has been attending Dr. Goodhart as out-patient for a severe cough.

Fourteen days ago he went to bed in his usual condition but during the night he had an exceptionally violent fit of coughing. He did not strike a light, but when he awoke in the morning, he found he could see nothing at all with his right eye. He is sure that he could not even see the light. Gradually a little sight returned till, when I first saw him, he could count fingers at a foot. His field of vision was wanting on the upper and inner side, the defect
extending nearly up to the fixation point (see Chart 1). This defect corresponded to a large, moderately sharply defined, detachment of the lower and outer part of the retina. The other ophthalmoscopic appearances and also the tension were normal in the two eyes, and the pupils were large and sluggish.

**Chart 2. July 24, 1884.**

He was told to come on operation day, six days later, for treatment by scleral puncture in association with rest in bed and the internal use of jaborandi. He accordingly presented himself on July 24th, 1884, having been at his usual occupation in the meantime, but
said that he was so far better that he was doubtful whether the operation was necessary. And indeed he was much better. The vision of the eye, with detached retina, was now $\frac{6}{14}$, and the field, though still contracted on the upper and inner side, was considerably larger than before (see Chart 2). No retinal detachment could now be made out, but, besides a couple of ill-defined dark patches near the seat of the puncture, and some few small, well-defined opacities, floating freely in various parts of the therefore rather fluid vitreous, there was nothing morbid to be seen. The disc was normal and tolerably well defined.

The left had throughout $V. = \frac{6}{6}$. Field perfect. The eye looked quite normal.

October 9th, 1884.—His wife reports that the improvement is still maintained.

**Case 2.** Master M—, æt. 12, has of late years been noticed by his parents to be rather shortsighted. Three months ago he received a blow from a stone on the outer side of the right eye. It was not a very severe blow, and it is questionable whether it struck the globe itself or merely the edge of the orbit.

Nothing was found out to be wrong with the eye itself till about two months later when the vision was very defective.

He came under Mr. Snell's care at Sheffield, and he diagnosed a large detachment of the upper and outer part of the retina. As this did not subside under the use of internal remedies, Mr. Snell wished to resort to puncture but hesitated owing to the fact that the detachment was in several folds, more or less separated from each other.

When I saw him some weeks later there was a large detachment occupying the upper and outer part of the globe. Its lower margin was well-defined, rising somewhat steeply from the level of the retina below and extending inwards up to about the macula. The rest of it shelved off gradually above. Corresponding to the
detachment the lower and inner part of the field of vision was wanting, the defect involving the fixation point (see Chart 3). Other opthalmoscopic appearances were normal.

V. = fingers at about 6'. Both eyes had T. normal and the pupils medium-sized and sluggish.

The left eye had V. = $\frac{6}{18}$ only. It was the subject of considerable mixed astigmatism, + 2 D. correcting one meridian, and − 2 D. the other. With full correction the vision equalled $\frac{6}{6}$.

As the fluid was now collected into one pouch, I punctured the eye under the approval of Mr. Snell with a Graefe
knife one week later. A second puncture was made, since from the very defective light at the time of operation there was some doubt as to the quantity and character of the escaping fluid.

In the end considerable fluid, apparently of a serous nature, was evacuated. The boy was put to bed and jaborandi was given internally in hourly doses, just large enough to excite moderate diaphoresis.

Ten days later, on uncovering the eye, it was found by the ophthalmoscope that, though a flattened opaque fold of retina could be still seen extending from in front of
the outer equator up to and beyond the macula, the rest of the retina had reapplied itself. This part, however, was still not perfectly transparent, nor had—as indeed appears to be always the case—it regained its normal visual acuteness. It was found by estimation with the fingers that there was a blind track or process corresponding to the fold, extending from the middle of the inner border of the field up to and beyond the fixation point which it included. The lower and inner part of the field, though still limited, was considerably larger than before (see Chart 4).

There was clearly distinguishable in the fold nearer to its yellow-spot end, and at what was about its summit, a dark gap, corresponding in size to the puncture of a Graefe knife, which was evidently the aperture of incision through the retina.

Ten days later the field had shrunk nearly to its size previous to the puncture.

**Case 3.** Thomas S—, aged 40, chairmaker, High Wycombe, was sent to Guy's Hospital by Dr. L. W. Reynolds, on account of a detachment of the retina at the lower and outer part of his left eye.

Up to March, 1884, this eye appeared quite as good as the other, though he had had a severe blow on it with a piece of wood ten years before.

He was suffering from a severe cold and cough at the time he first noticed the impairment of vision. The fits of coughing were very violent, but he cannot connect any particular fit with the deterioration of sight. Things seemed duller towards his left side, and this got gradually worse till admission, when nearly the entire inner half of the field of vision was wanting, inclusive of the fixation point, but not right up to the middle line below (see Chart 5, p. 120).

\[ V = \text{fingers at about 8 feet.} \]

*Right eye* \[ V = \frac{6}{6}, \text{no H.m., T. n. Field complete.} \]

*On admission,* September 25th, 1884, the sclerotic was punctured near the lower and outer equator, a bent iridec-
tomy knife, 5 mm. broad, being used. The knife went in about 5 mm. Abundant syrupy fluid escaped. After eight days in bed, and treatment by compress bandage, atropine, and jaborandi (for the first half of the time, however, in ineffective doses), the field was found conspicuously enlarged on the defective side. No chart was taken till the thirteenth day when the field seemed to him and to us to have again diminished slightly. Still the fixation point was free while the field was very considerably enlarged below the horizontal meridian. \( V = \frac{6}{6} \) (see Chart 6, p. 121).
No detachment is now visible with the ophthalmoscope, but numerous opacities, some of them very large, are seen floating freely in the lower and outer part of the vitreous. Indeed, the vitreous is generally hazy and the disc is but dimly visible.

The patient is an intelligent man and has been in the habit of reading a good deal. He takes but little exercise of any sort, and certainly none of a violent character.

Since in two out of these three cases the other eye was found healthy and no defect had been previously noticed in its fellow, it is reasonable to suppose that the detach-
ment had occurred in association with a healthy vitreous. In one of these two cases there is fair evidence that a fit of coughing was the immediate cause of the detachment, and there is a reasonable probability of the same in the other case.

I find it impossible to conceive how, with a normal vitreous, the retina can be detached without its rupture. In that respect I am in accord with the observations of Leber, though I account for the rupture—which, however, it is proper to say I failed to make out ophthalmoscopically—in a different manner. It is not possible to imagine how the tunics of a globe can be so pressed, even in the most violent muscular efforts, whether respiratory or other, against the scarcely yielding vitreous as to rupture them. I incline to the opinion that the sudden recoil after such compression is the cause of the phenomenon.

With regard to treatment, the first case might be taken as indicating that no treatment is necessary. But the third case, in which the affection steadily progressed up to the time of operation and was then alleviated, points to a different conclusion.

As to the relative value of the three agents employed, viz. puncture, rest in bed, and jaborandi, I am unable to form a decided opinion. I rather incline against jaborandi. Certainly it is a disagreeable drug, both to the taste and in its effects. Rest is, I think, of real benefit; if so, this result must be simply due to the effect of gravity acting mechanically on the displaced part and favouring the re-application on the slightest tendency to the re-absorption of fluid. Perhaps the patient should accordingly lie as much as possible on that side.

When a retinal detachment disappears the subretinal fluid must pass either into the vitreous or be absorbed by choroid or retina. With an unruptured retina I judge that its course is along the sheaths of the retinal vessels and so into the optic nerve, which in health appears to be the route of the waste fluid from the choro-capillaris.
NIGHT BLINDNESS FROM EXPOSURE TO A BRIGHT LIGHT. 123

I may remark that two of the cases are, as far as can be inferred, decidedly in opposition to the doctrine of Galezowski, (1) that in retinal detachment the vitreous is the subject of a previous softening, (2) that with healthy vitreous detachments do not occur as the immediate results of injury.

It is scarcely necessary to add in the face of these two cases that I think a simple puncture, sufficiently large and in a safe position, quite as effective as any more elaborate proceeding, with its dissection away of conjunctiva, &c. Indeed, the greater need of an anaesthetic furnishes a strong reason against any such complicated procedures. I have usually punctured the retina into the vitreous, but I am not convinced of the necessity of thrusting in the knife so far.

In conclusion, I ask whether during operations the retina is detached in all cases of loss of vitreous. If so, it is capable of reapplying itself and that speedily, otherwise the adhesion between normal vitreous and retina must be extremely slight.

(October 9th, 1884.)

2. Two cases of night blindness from exposure to a bright light.

By W. Adams Frost.

Case 1. Alfred E—, æt. 34, a painter by occupation, had never had colic, and had never been out of England.

He first came on May 24th, 1880, to St. George's Hospital as an out-patient, stating that since ten days previously he had found himself unable to see at dusk or in a dull light in the daytime. He had been engaged for some weeks in painting the exterior of white houses. The weather had been very bright, and he had felt the glare very much. V. was $\frac{20}{30}$ in each eye. Discs slightly hyperæmic.
He stated that in the preceding year about the same time he had been similarly affected. On that occasion also he was engaged in outdoor work, but he believed that the weather was dull. He was transferred to indoor work and recovered completely in about a fortnight.

On the present occasion he was admitted into the hospital. Leeches were applied to the temples, and he was kept in the dark for twenty-four hours. A good deal of photophobia succeeded, and eserine was used with the object of diminishing the size of the pupils. In a fortnight after admission he had completely recovered.

On May 21st, 1881, exactly a year after his first visit, he returned with similar symptoms caused under identical circumstances. He was treated as an out-patient and recovered in a fortnight.

In July, 1882, he had another attack, coming on this time, however, in dull weather, but in other respects it was similar to the preceding ones.

In May, 1883, he was attacked for the fifth time, after painting in bright weather; he was, however, already recovering when I saw him, and I have no note of any second visit.

On May 24th, 1884, he again appeared. The weather for many weeks had been excessively bright. On this occasion he was examined more thoroughly. Vision, as on every other occasion, was \( \frac{20}{30} \). The visual field was tested and found to be normal. Thinking it probable that we might obtain evidence of fatigue of the retina, I passed the test object round a second time but did not obtain a "spiral" chart. I also tested the light-sense with Förster's photometer, using No. 1 Jaeger as the test object, and found that he required a much larger amount of light than a normal subject. The difference was sufficient to remove all doubt as to its reality, but I found great differences in the amount of light required by normal eyes; and in testing about a dozen students, found that an aperture varying from 2·0 to 5·5 mm. was required.
The patient, however, could never read with an aperture of less than 12 mm.

He was treated with iron, quinine, and eserine, and recovered in about a fortnight.

**Case 2.** James Wheatley, æt. 19, lamplighter by occupation. I first saw him on June 6th, 1884. For three weeks he had had great difficulty in seeing at dusk. For six weeks before this he had been employed in painting the lamp-posts stone colour on the country roads about Woolwich and Clapton. The weather during the whole time was very bright, and he felt the glare very much. After three weeks of painting there was much aching pain and lachrymation, but he kept at his work. When seen V. was normal (\( \frac{6}{6} \) and J. 1). I had no opportunity of testing him with the photometer, but trying him in the dark room by gaslight, I did not find that he required a stronger light than I did myself. The visual field was normal. He was treated with eserine and discontinuance of painting. He had nearly recovered five weeks after the commencement of the attack, and a fortnight later he was said to have completely recovered.

August 25th.—He returned stating that he had remained quite well until two days previously, when he noticed a failure of vision at dusk. He had not on this occasion been engaged in painting, and could give no cause for the attack. As atropine had been put in elsewhere the visual field was not tested, and I have no note of any subsequent visit.

The fact that this patient's vision did not perceptibly deteriorate on diminishing the artificial illumination, puzzled me at the time, as I was not then acquainted with the explanation given by M. Fontan ('Rec. d'Oph.,' Oct., 1882). Gradually diminishing the illumination he found that the minimum amount of light necessary for normal acuteness of vision was the same for the normal eye and for the patient affected with night-blindness, but that with the minimum illumination the pupil of the patient with night-blindness was double the diameter of that of
the normal subject, and further found that by placing in front of the eye of the night-blind patient a diaphragm whose aperture was equal to that of the pupil of the normal eye, V. at once became lessened.

(October 9th, 1884.)

3. A case of supposed unilateral albuminuric retinitis.

By Henry Eales.

The case which I have the privilege of bringing before this Society to-night is one of a very unusual character, in which the most diffuse and destructive retinitis of the albuminuric type occurred in one eye only (the left), the other eye showing no traces of disease whatever throughout a period of nearly four months, during which time the patient suffered from albuminuria.

All authorities are agreed, and it is the almost universal experience of ophthalmologists, that this form of affection invariably affects both eyes, if not simultaneously, at least within a very short period of one another. Amongst recent writers I may mention that Juler in his 'Handbook of Ophthalmic Science and Practice,' says (p. 189), "Both eyes are always affected;" that Swanzy also, in his 'Handbook of Diseases of the Eye,' says (page 321) "Both eyes are affected." There has recently been reported, however, a very remarkable case by Yvert in the 'Recueil d'Ophthalmologie' for March, 1883, to which I shall specially allude, because it appears to me to bear great resemblance to my own in its clinical features, and because the death of the patient enabled a post-mortem to be made which revealed such a very unusual state of things as to suggest to the writer in explanation of the retinitis a theory which, while new, seemed most capable of accounting for all the facts.

I have been unable to obtain the actual paper, but have
SUPPOSED UNILATERAL ALBUMINURIC RETINITIS. 127

come across several abstracts of it during my researches, the best of which is perhaps that in the 'Ophthalmic Review' for July, 1883.*

The writer of this review, in speaking of this exceptional case, says, "Yvert, commenting on the foregoing case, calls attention to the extreme rarity of a unilateral albuminuric retinitis. Amongst about eighty cases examined by himself both eyes were affected in all. He also quotes the writings of Warlomont and Abadie to the same effect, and to this we may add that Leber speaks of the disease as being almost without exception a bilateral one; the only exception known to himself being one in which there was an unusual ring-like defect in the visual field, but no well-marked or characteristic ophthalmoscopic change (Graefe and Saemisch, vol. v, page 584).

The following is the history of my case:—F. B.,æt. 25, single, by occupation a striker, has never had scarlet fever or any other illness in his life till the present attack, his general health always being very good. No history of syphilis, which he denies having ever had. For the last seven years he has been a very heavy drinker, getting drunk two or three times a week, and frequently drinking continuously from Friday night to Monday, a statement supported by his appearance, notably the distension of his facial capillaries. As a rule he drinks nothing but ale, but he would sometimes have whiskey. Two years ago he suffered from a skin affection which occurred chiefly on the face and neck and the backs of the hands, and was accompanied by a weeping condition of skin.

One doctor said it was the itch. Three other doctors said his "blood was out of order," and ordered him medicine and ointment, but in spite of treatment it continued for ten months to trouble him, and he found that "he could not get cured while he was drinking," but got well in a week or two on leaving off his beer. Has sometimes had pain

* Since reading this paper, I have had an opportunity of perusing Yvert's original communication in the 'Recueil D'Ophthalmologie,' and can confirm the correctness of the statements herein made.
and stiffness in the shoulder, but not in the great toe or any other joint. He is not a large meat eater. Ten years ago, on November 5th, he received a severe blow with a heavy stick on the left eye, which gave him a black eye for some time, but he does not think that this in any way affected his sight.

On Saturday August 2nd, 1884, he was in London with his brothers, who are in the army, and got very drunk, and was so severely affected with vomiting, that though against the rules, he was kept in barracks for the night. On this occasion he fell down three or four stairs in the barracks and severely wrenched and injured his left side in the loins, but did not injure his eye or his head; of this he feels confident, as he had no black eye, nor bruises, nor scratches about his head the next day; moreover, he remembers the falling. On waking about 5 a.m. the next morning, he found that "the upper part of the sight of his left eye was cloudy," and the pain in his left loin was so intense that he could not stand upright. This pain was of an aching character, and persisted for quite a fortnight, during which time he noticed nothing unusual about his urine. As his sight got worse, on Wednesday, August 6th, he went to an eye hospital where the doctor told him that "the nerve of the eye was decayed at the back and that he would never be any better or any worse in his eye," and advised him to go to work and not bother about it; but, however, ordered him some medicine, which on future examination of his prescription paper, I found to be iodide of potassium and bichloride of mercury. There was no diagnosis on the prescription paper, but a note as follows: "Left eye just counts fingers." The medicine was repeated on August 13th and again on August 20th.

According to the doctor's advice, he tried to go to work on the following day, August 7th (Thursday), but his back was still so bad that he had to give it up. He tried to work again the following Monday, nine days after his fall, but still was unable on account of his left side, so, as his sight continued to get worse, he came to the Birmingham
SUPPOSED UNILATERAL ALBUMINURIC RETINITIS.

Eye Hospital on August 26th for the first time, and in my absence was seen by my colleague Dr. White (then our house surgeon), who made the following note as to his condition: "Right eye normal. Left eye, neuritis; disc very pale, considerable proliferation; several spots in retina round macula of albuminuric appearance; one large hæmorrhage near disc. V. = \(\frac{20}{5}\). Urine sp. gr. 1014; albumen in slight quantity."

On September 1st I first saw him, and entirely confirmed the above note as to the state of his eyes and urine. He stated that he had not suffered from headache since his sight began to fail three weeks previously.

On September 15th, his eye having got worse rather than better in spite of treatment by Liq. Ferri Perchlor. internally, with a purge of elaterium every other day, I persuaded him to come into the hospital. At that time the retina was extensively covered all round the disc (which was not much swollen however), and macula with dense patches of yellowish white exudation, which was specially dense in the lower quadrant of his retina, and mostly beneath the vessels. Just above the disc a large hæmorrhage, before noted, evidently mostly in the deep retinal layers, was found. V. = shadows only. He could not count fingers at two feet. The right eye was quite normal in appearance. V. = \(\frac{5}{5}\). He presented slight evidence of anaemia. He was put on a milk diet, with, however, a little fish for dinner, and ordered a mixture containing Liq. Ferri Perchlor., to which after a few days Tr. Digitalis was added; and he was ordered a purge of elaterium and a hot bath every other day. Under this treatment he expressed himself as feeling better, and the condition of his eye rapidly improved. The urine, which was measured daily, amounted to from sixty-six to seventy-six ounces, with an average sp. gr. of 1020, while for the most part the albumen got less daily. He had to get up at night to make water, a symptom which had not previously troubled him. On September 28th the following note was made as to his eye by the
house surgeon, "Proliferation round disc has almost quite gone, leaving disc very pale." On this day jalap was substituted for elaterium purge. On October 3rd it was noted "that there was no albumen in the urine." October 10th no albumen found in urine. He was allowed chicken for dinner in place of fish. October 20th he was discharged. About this time I carefully examined his urine as also did Dr. Saundby kindly for me, and we could neither of us find any albumen or casts. Dr. Saundby also reported that he showed no signs of cardiac disease, that his apex-beat was normal in position, but that his pulse denoted some excess of arterial tension. I regret that casts were not previously looked for. Several future attempts failed to find casts or albumen, but a week later, on being put on ordinary meat diet, though no albumen was as a rule found in the urine, the faintest trace (opalescence) could be found about one hour after breakfast only.

At this time I found that the disc was quite atrophic, that the vessels, especially the arteries, were reduced to threads, but partially emptied, and pulsed on the disc on pressure being made on the globe; that there were several minute glistening white patches all round the macula, where previously large whitish patches were seen. V. = 3/60. Two large patches of atrophy were seen in the choroid below, one irregular with pigmented edges, the other round and sharply circumscribed.

He said he could see much more now than he could a week or two previously.

October 30th.—He was readmitted as he did not feel so well, and was unable to work. His eye was much as before but improved, the patches round the macula being smaller and less visible. Urine showed a trace of albumen only after breakfast; no casts.

November 27th, he was again discharged, his eye being very little improved and his condition being as at present. Right eye normal, V. = 7/6. Left eye, disc atrophic, vessels reduced to threads; several slight glistening white
patches around the macula; a large patch of atrophy of choroid below. V. = \frac{3}{6}.

Remarks.—Before proceeding to comment on my own case, I will draw attention to the main features of Yvert's, to which, in many points, mine bears a striking resemblance.

The patient, a male, suffering from albuminuria, was affected with most diffuse and intense retinitis in the left eye of a mixed inflammatory and degenerative type, ending in almost complete loss of sight. The right eye remained normal throughout. After being under observation seventy-two days, he died. At the autopsy the right kidney was found to be entirely absent, there being no trace of ureter, artery, or vein. The left kidney showed well-marked parenchymatous nephritis. Yvert quotes as analogous to his case five cases published by Potain in the 'Gazette des Hôpitaux,' February 17th, 1883, in which there was anasarca of one side, or at least more marked on one side, in consequence of contusions of one kidney; in explaining which Potain considers an abnormal action of the sympathetic nerve of one side of great importance.

Yvert also explains one-sided retinitis albuminurica in his case, by assuming an irritation of the sympathetic nerve of one side due to a unilateral affection of the kidney.

Bearing in mind in my case the history of an injury to the left loin, in a man prone by alcoholic habits to nephritis, and at a time when he was intoxicated, one can hardly help feeling that the kidney affection is possibly in this case confined to the injured side, and of a traumatic, and possibly temporary nature, a fact supported by the temporary character of the albuminuria; while the rapid onset of the eye symptoms (within a few hours) after the injury seems to point to it as the cause of the eye affection; and Yvert's view of "irritation of the sympathetic" seems to be a most plausible explanation of so rapid, and so completely unilateral, though intense, retinitis on the same side; at any rate I cannot see my way to any better explanation, for the man is quite confident there was no
injury to the eye or even to his head to induce the local affection. As the night was very warm on which his symptoms began, and he remained in barracks, he is sure that he suffered in no way from cold or exposure.

To some it may seem that there is a mistake in the diagnosis. It may be urged that there were no casts in the urine, nor any cardiac hypertrophy. To this I would reply that while the presence of casts is strong evidence in support of a diagnosis of kidney disease, their absence would not exclude its existence. Moreover, I would allude to the fact that no casts were looked for during the existence of the albuminuria, when they were most likely to be present, which omission I now much regret. Finally, as an ophthalmic specialist I would decline to be dragged into the apparently interminable discussion as to whether albuminuria is necessarily pathological or not. I would also quote Gowers to show that cardiac hypertrophy is not essential. He says, in his ‘Medical Ophthalmoscopy,’ page 103, ‘It is not probable that there is any necessary connection between the retinal and the cardiac change.’ Then it may be said that the atrophic patch in the choroid suggests that the disease was choroidal. To this I would say, I have a case under my care now in which both albumen and casts have been present for two years, and in which the choroid has been similarly but not so extensively affected opposite spots of effusion into the retina, and I would draw attention to the fact that the retina was most affected where now the patches are seen in the choroid.

Moreover, I would ask, would so limited a choroiditis account for such fairly typical peri-macular changes as are seen in this case, and for such a characteristic condition of the disc, and such extensive retinal degeneration, and, moreover, without effusion in the vitreous, of which there has never been any evidence? And, further, I would remind my hearers of the history of a blow on this eye ten years ago, as a possible explanation of the condition of the choroid.
Then, it may be suggested that it was embolism, or thrombosis of the retinal artery or vein, and the albuminuria only a coincidence. To this I would say that neither the appearances, nor the history of the eye affection coincide with this view. Neither embolism nor thrombosis of the artery is, so far as I know, usually followed by a return of circulation in the vessel, which is certainly present in this case; nor do either lead to large patches of exudation in the retina, as occurred here, and which are only feebly represented now by the minute spots around the macula, and the disturbance of the choroid around the disc, together with loss of definition of its margin; while thrombosis of the vein would surely have led to hæmorrhages which, with one exception, were absent in my case. Moreover, the retinitis was most typically albuminuric of the mixed inflammatory and degenerative type; and, further, the eye condition, and the kidney condition improved simultaneously.

I have drawn attention to the administration of mercury to this case, for I felt it a duty not to conceal it, because I think it may in some measure account for the severity of the eye affection. I have myself twice seen a similar severe mixed inflammatory and degenerative retinitis supervene at once on the administration of mercury, on rule of thumb principles, in cases of slight neuritis occurring in patients suffering from albuminuria (not I am happy to say in my own practice), and I have before alluded to the same.

(Living specimen. December 11th, 1884.)

4. Visual function retained by a retina which has almost certainly been detached for several years.

By E. Nettleship.

Patient's L. eye, which diverges widely, shows universal detachment of the retina from the edge of O. D. forwards
in all directions as far as can be seen. In the lower part the detachment is very deep, forming a large rounded prominence, but in most other parts it is comparatively shallow and, although undulating, free from deep folds. The greater part of the retina remains fairly, if not quite, transparent, so that the red of the choroid is fairly visible in most parts, and can be made out even behind the prominent, deeply detached portions. In several places, however, especially near the Y. S., portions of the retina show whitish haze. The physiological pit of the O. D. is E. or M. 0.5 D.; the detachment begins at the very margin of the O. D., and at the fovea centralis, the situation of which can be recognised by the arrangement of the small blood-vessels, the surface of the retina is H. 7 or 8 D. Between the Y. S. and O. D. there is a very red spot bordered by a crescentic white edge, perhaps a small rent. The refraction of the retina at the upper part of the fundus is H. about 13 D. P. 5.5 mm. acts directly to l.t. He has good P. L. and good projection, except in upper part of F., and seems to see best with the inner and lower parts of F.; without a glass he cannot count fingers but with various glasses from + 8 to + 18 D. he counts fingers at 1', sees letters in 20 J., and even makes out a few in 18 J.; the letters in 19 J. are too crowded to be distinguishable. In looking at an object he appears to place it in the centre of F. With the other (R.) eye V. = V. 6/6 and 1 J. refraction E. He is an intelligent lad of seventeen, a teacher. Neither he nor his mother can give any conclusive evidence as to the exact date of the failure of L.; the divergent squint, however, has been noticed for about eight years, and the patient can never remember seeing better with the eye than he does now. At the age of nine he had a blow on the head and soon afterwards he had scarlet fever; it is thought that the squint began soon after the fever.

(Living specimen. January 8th, 1884.)
5. Detached retina in the yellow spot region.

By W. Lang.

Eliz. L—, æt. 27, domestic servant, states that she received a blow on the L. eye eighteen months ago owing to the accidental slipping of her own hand when shaking up bedclothes.

She experienced considerable pain, which lasted about half an hour, but she cannot tell whether the sight failed immediately after or within a few days of receiving this blow.

No history of other injury or eye disease.

Family history good.

Present condition.—V. \( \frac{6}{2} \); with Cyl. + 1.5 V. = \( \frac{6}{1.2} \) and J. 1.

L. Counts fingers excentrically.

In yellow spot region of this eye there is a detachment of retina about ten times the area of the disc.

(Living specimen. March 12th, 1885.)
IX. DISEASES OF THE CHOROID.

1. Nevus of left side of face; nevus of choroid, subretinal hemorrhage, and detached retina in left eye.

By J B. Lawford.

(With Plate II.)

Rosa B—, æt. 8, a fairhaired child, was admitted to the Royal London Ophthalmic Hospital on May 15th, 1884, under the care of Mr. Lawson, to whom I am indebted for permission to bring the case before the Society.

In October 1882, the child had been to the hospital as an out-patient, and a note then made ran as follows:

"Left eye, cataract, ? secondary; iris bulged forwards; T + 1 or 2, difficult to estimate, but certainly more than that of right eye."

Her mother stated at that time that the eye had only recently failed. She was not seen again till the day on which she was admitted, eighteen months later. On May 2nd the child was struck on the left eye by one of her playmates, and soon after complained of severe pain. Her mother then noticed, apparently for the first time, "a yellow colour" in the eye.

On admission the following notes were made:

Right eye, V. = $\frac{20}{20}$, media clear; fundus healthy.

Left eye, slight ciliary injection; cornea clear; no vessels visible on it; a. c. rather shallow; pupil dilated and irregular. Lens opaque, yellow, no p. l. T. + 2.

The left eye does not seem enlarged; in fact it looks, if anything, rather smaller than the right, but this is probably only apparent, and due to the condition of the eyelids.
On the left side of face there is a large dull red capillary nævus, extending from the forehead to the chin, crossing the median line for about an inch in the latter situation, but not elsewhere. The left eyelids are involved and slightly thickened; the ocular conjunctiva is not affected. There is a small similar patch immediately in front of right ear. No nævi in other parts of the body.

The child is healthy in other respects.

May 16th.—Left eye excised. No unusual bleeding, nor any evidence of nävoid condition of the orbital tissues.

20th.—Patient discharged.

The eyeball was preserved in Müller's fluid, then divided while frozen by a vertical antero-posterior section. The result of the examination of the different structures is as follows:

**Cornea.**—Anterior epithelium undisturbed, corneal tissue fairly normal, but some proliferation of cell elements; several newly formed vessels are spreading inwards from the margins for a short distance; these are distributed throughout the thickness of the tunic, and are not more numerous in deeper than in superficial layers. The posterior elastic lamina and epithelium are normal. The subconjunctival tissue at margins of cornea is considerably increased in amount and contains numerous vessels, and a few small extravasations of blood. Schlemm's canal is patent.

**Sclerotic** is healthy in appearance. Anterior ciliary vessels are full and large; the posterior ciliary vessels, where they pierce the tunic, show an increased number of nuclei in their coats. The ciliary nerves in section appear healthy.

**Ciliary processes and iris.**—The ciliary processes are much atrophied, and appear to have been forced against peripheral part of cornea, carrying the root of iris in front of them; they encroach upon and indent the cornea for a short distance anterior to the canal of Schlemm. The "iris-angle" is thus blocked. The iris is atrophied and
thin. There is no dilatation of vessels in iris or ciliary processes.

*Lens* shrunken and opaque. The capsule is entire; a small amount of uveal pigment adheres to it anteriorly. Immediately beneath this portion of the capsule is a thick layer of indistinctly fibrous structure, with a few oval nuclei which stain with logwood, and some calcareous looking spherules but nothing like lens fibres. In the deeper portion of lens the fibres can generally be made out, but are indistinct and irregular. Throughout are a large number of oval or elongated, slightly granular, cellular-looking structures, which do not stain; they are larger than the nuclei of lens fibres, and are probably products of degeneration.

*Retina* is detached from choroid everywhere except at O. D. and ciliary processes, and extends forwards in a double fold to the posterior surface of lens with which it is in contact. It has undergone much degeneration, and its several layers are to a great extent indistinguishable, the outer granules being most easily made out. Close to the posterior surface of the detached retina is a layer of colloid material in semitranslucent spherules. The space between retina and choroid is filled by a soft, gelatinous exudation, which contains colloid material. Near the choroid posteriorly is a large recent haemorrhage.

*Choroid.*—This tunic, surrounding the optic nerve for a distance of 5 mm. to 7 mm., is considerably thickened, measuring 0.5 mm. to 1 mm. close to the disc, and gradually thinning off towards the periphery. This thickened area has a spongy appearance to the naked eye, and is brownish in colour. Under the microscope this portion is seen to be channelled with vascular spaces of varying size. The greater number of these are dilated capillaries, some of very large size, but in many no endothelial wall can be distinguished, and they appear to be bounded by the connective-tissue elements of the choroid which are considerably increased in quantity. Ciliary vessels enter this area through the sclerotic. There
DESCRIPTION OF PLATE II.

Plate II illustrates Mr. Lawford’s paper on Nævus of Choroid (p. 136).

Section of a portion of the choroid in the area involved by the nævus. The blood-channels are seen throughout the whole thickness of the tunic. In many of the spaces towards inner surface of choroid the blood-cells have been washed out during preparation, in which process also the hexagonal pigment layer became detached. The section is through the thickest part of the nævus.

(From a drawing by M. H. Lapidge.)
is less pigment to be seen in the choroid coat in this area than is usually present. The hexagonal retinal pigment layer is adherent to the choroid in greater part, but is disturbed in two places by nodules of colloid growth, and in another situation by some products of inflammation (an indistinctly fibrous layer) on the inner surface of the choroid.

Beyond the limits of the nævus the choroid is thin, but presents no abnormal appearances. The bloodclot spoken of above lies immediately anterior to, but does not encroach on, the choroid.

Optic nerve.—The portion close to the eyeball shows a considerable increase of leucocytes throughout; the fibres appear to have undergone little if any change. The nerve is extensively excavated at its entrance to the eyeball; the lamina cribrosa is deeply concave anteriorly; the cup is filled by degenerated retina and some effused blood.

This appears to be a cavernous angioma of the choroid, involving only a portion of that tunic. As far as I am aware, it is an example of a rare or at least of a rarely observed condition. I have been unable to find records of any cases (with the single exception of that shown by Mr. Milles last year) in which microscopic examination has demonstrated the existence of nævus of the choroid; but I should add that my search has not been as exhaustive as I should like. Perrin and Poncet, in their 'Atlas of Path. Anat. of the Eye,' record a case, the description of which suggests that it may have been a nævus; they call it "Congestion of the choroid." And yet a priori one feels inclined to say that the choroid, with its unusually vascular structure, would be a not unlikely situation for the development of nævus.

(May 14th, 1885.)
2. Central choroiditis with disseminated patches in remainder of fundus.

By W. Lang.

Ellen S—, æt. 30, married four years, has had two healthy children. The eldest suffers from convergent strabismus with H., eyes otherwise normal; the other has not been examined. She is the eldest of a family of three. The mother, æt. 70, is alive and well, and sees to read the newspaper with glasses. The father died, æt. 41, from the effects of a horse-bite which caused "mortification." He had been previously married and had two children, one a daughter who is alive, and a son who died a soldier in India. There is no family history of gout, rheumatism, or phthisis.

The patient noticed that her sight was failing six years ago, and it has gradually got worse since then, but without causing any discomfort except when suckling each child, and then the pain was so great that she had to wean them. The vision was R. = \frac{6}{36} with \text{cyl. } \frac{1}{2} and J. 2 slowly; L. J. 2, words and \frac{6}{0}, not improved with glasses. Her health is uniformly good except that she has occasional headaches.

The patient's brother is similarly affected (vide case following).

The following description applies to each eye of both patients.

In the y. s. region there is considerable disturbance of the retinal pigment with increased pigmentation in one or two places. Discs normal.

In the posterior part of fundus there are numerous small round, or oval yellowish-white patches scattered in every direction; they do not assume any regular distribution, but the oval ones generally have their long axis at right angles to the retinal vessels. They are apparently situated in the superficial part of the choroid.

(Card specimen. March 12th, 1885.)
3. Central choroiditis.

By W. Lang.

Walter V—, single, æt. 28 (brother of Ellen S—, who is similarly affected (see case preceding). He first noticed that his sight was failing twelve years ago, but could see to read the newspaper till eighteen months ago. His vision R. and L. now = J. 6 and $\frac{6}{6}$, but he complains of not being able to see faces well, although he has no difficulty in following his occupation as gardener. His colour vision is good. His general health has always been good. He had smallpox six years ago but no other illness. Urine sp. gr. 1011, no albumen or sugar. No history of syphilis. The preceding description of the changes in his sister's eyes applies to his also. He has a twin sister whose vision = $\frac{6}{6}$ and whose fundus in each eye is normal.

(Card specimen. March 12th, 1885.)

4. Bands of connective-tissue growth in vitreous with choroidal atrophy.

By W. Lang.

The patient is a well-developed man of 21. He has always known that the right eye was defective, but of late it has watered on exposure to sunlight.

The left eye has no change in the fundus, and the vision is $\frac{6}{6}$, with H. m. = 0.75 D.

The vision in the R. = J. 16 badly and with $-7$ D. = $\frac{6}{6}$. With the ophthalmoscope a large band of connective tissue is seen running forwards from the centre of the disc. About a quarter of the way through the
DISEASES OF THE CHOROID.

vitreous it divides into three branches. One continues on to the centre of the back of the lens capsule where it is adherent, a second passes directly above this nearly to the upper edge of the back of the lens, whilst the third runs horizontally forwards and inwards, but does not get much beyond the equator of the globe. All these bands appear white by direct examination, but by oblique examination the one that follows the course of the hyaloid artery is brownish in colour. Around the disc there is a considerable amount of choroidal atrophy, and white bands border most of the vessels. The portion of the disc that is not covered by the connective tissue bands is of a dark colour.

At the upper and inner part of the fundus there is some choroidal pigmentation with atrophy.

(Card specimen. June 4th, 1885.)

5. Atrophy of choroid.

By A. Stanford Morton.

(With Plate III.)

The patient, John J—, aged 55, painter, is under the care of Mr. Tay at Moorfields and gives the following history of himself:—At about the age of twelve years he had "gastric fever," and suffered from fits till about the age of fourteen, they then ceased until ten years ago when they recommenced, and he has one occasionally. While following his occupation abroad he had yellow and intermittent fevers, and was twice insensible with sun-stroke. Since following his occupation at home he has twice had lead colic for which he was treated at St. Bartholomew's, and he was also in that hospital suffering from injuries received to back and ribs. He had what he describes as gout in the great toe in 1862, and suffers at times from swelling of knees, ankles, wrists, and elbows.
DESCRIPTION OF PLATE III.

Plate III illustrates Mr. Stanford Morton's case of Atrophy of the Choroid (p. 142).

(From a drawing by A. Stanford Morton.)
ATROPHY OF CHOROID.

His father was gouty. There is no history of syphilis, but his wife had, he thinks, two miscarriages; there is no family. The patient's sister and her child have had their eyes examined and were found perfectly normal. The first defect in the patient's sight was noticed five or six years ago when he commenced to stumble against objects as soon as it was dusk. This defect is now the most prominent symptom, and even in the day, though his central V., as taken by Mr. Hudson, is quite normal, his field is contracted to within an average of about 10° (see Chart). The fundus shows in a most remarkable manner the whole
of the choroidal distribution of vessels. There seems, except in the region of the macula, to be almost complete atrophy of the pigment layer of the retina and of the chorio-capillaris. The larger choroidal vessels retain their normal appearance towards the periphery, but, on approaching the disc, the lumen is almost obliterated and they appear as white bands traversed in the centre by a fine red streak, so small as to be discernible only by direct examination. The interspaces are filled with dark pigment. Chiefly towards the periphery, among the terminal retinal vessels, there exist several areas in which are seen fine reticulations of pigment together with some larger, irregular, and stellate masses. Whatever may be assigned as the cause of these appearances, they are at any rate very unusual. If syphilis were supposed to have produced them, it would be strange to have the central vision perfectly good when such extensive peripheral changes had occurred, and also that there should not be deeper extension with patches of exposed sclerotic. On the other hand, though the normal central vision co-existing with the very contracted fields and night-blindness is suggestive of retinitis pigmentosa, there is very little pigment. What there is, however, resembles in many places the pigmentation of this disease, and some cases are recorded in which the pigment was remarkable rather by its absence.

(Card specimen. March 12th, 1885.)

6. Uniocular irido-choroiditis with new formation of connective tissue in the vitreous.

By R. Marcus Gunn.

Edith D—, æt. 8, attended at the Great Northern Central Hospital three days ago on account of dimness of sight in the right eye.
During early childhood she was quite healthy with the exception of "mumps" when two years old. No history of snuffles or thrush. She was always a very bright child until two years ago, when she had an attack of what was called "typhoid fever." This commenced with a convulsive seizure affecting the arms, and the doctor attending her observed that "the pupil of the right eye was larger than the left." About the second week of the illness her mother found that the right eye was nearly, if not quite, blind. There was at this time frequent sickness, unconsciousness, and delirium. No headaches were complained of. The right arm now seemed useless, the condition of the lower limbs was not observed. During the illness there was much feverishness. The bowels were constipated and no rash was observed on the body. She was very deaf at this time, and the right ear has remained so ever since. The mother seems sure that the right eye saw well previously to this attack. Ever since this illness she has never been like the same child as before; she is occasionally very absent-minded and dull.

Family history.—The mother had syphilis before the birth of this child. The family were as follows:

1. F., died, aet. 6 weeks, of hooping-cough.
2. M., aet. 18, in good health, had a fit when about six years old from "congestion." When he was about a month old the mother had well-marked secondary symptoms.
3. F., aet. 16, history of thrush. Eruption over body at eighteen months. Has had palmar psoriasis lately. General health fairly good.

Miscarriage.
4. M., snuffles. Died at five months.
5. F., living, scarring at left angle of mouth. Had very severe teething fits.
6. M., died of bronchitis at sixteen months.
7. Patient.
8. M., aet. 6, very delicate.
9. F., died at two years of measles.
10. M., æt. 2½, healthy; no hereditary taint.

Next followed two miscarriages.

Present condition.—No sign of inherited syphilis in face or teeth. Right eye slightly divergent, pupil smaller than left; acts feebly to light, well with convergence; T. n., counts fingers at five inches. Left eye: pupil active; V. = \( \frac{2}{2} \). (Atropine in R.). Right eye: Numerous posterior synechiae and dots of uvea on anterior capsule, but centre of pupil clear. The optic disc is not seen, but its position can be determined by the large branches of the central blood-vessels. Between it and the Y. S. is a large horizontally oval patch of choroidal atrophy, and there are numerous small round spots of the same in other parts of the fundus, but chiefly near the periphery (choroiditis disseminata). The disc itself is occluded by a dense light grey mass, with well defined edges, which protrudes into the vitreous. Inferiorly it is prolonged into one or two highly refracting, white, tail-like bands, which are situated more anteriorly than the bulk of the mass. Vessels can be easily traced on its surface for a short distance, but these do not seem to belong to the proper retinal circulation. Stretching horizontally inwards from the body of this new formation is a narrow detachment of the retina, and there is another small linear detachment near the macula. Left eye: Ophth. shows no fundus change.

Remarks.—The first point of interest in the case is the somewhat unusual condition of well-marked disseminated choroiditis confined entirely to one eye. Next, we note that the ocular symptoms first were observed during an attack of what was apparently meningitis. Is it likely that there was any connection between the eye affection and the meningitis? What is the probable nature of the growth in the vitreous? Its position and appearance suggest its possible origin in an exudation occurring in association with a past papillitis. It is now evidently shrinking and beginning to pull on the retina as small detachments have occurred. (December 11th, 1884.)
DESCRIPTION OF PLATE IV.

Plate IV illustrates Mr. Nettleship's case of Severe Central Choroiditis with good acuteness of Vision (p. 147).

(From a drawing by Miss Boole.)
Additional note, March 19th, 1885.—The mother reports that the right suddenly became blind two days ago, the child herself remarking that "the eyelid was surely closed over it." She had been reading closely, stooping by the firelight. V. = bare hand-reflex. There is now almost total detachment of the retina. March 30, the iris is slightly discoloured of a greenish hue, — T. 3; barest perception of light. Left eye: V. = $\frac{2}{20}$.

7. Severe central choroiditis with almost perfect acuteness of vision; visual field defective at periphery.

By E. Nettleship.

(From notes by Mr. Marlow and Mr. Jessop.)

(With Plate IV.)

The drawing is from the erect image of the R. eye. The choroid over the whole of the Y. S. region is superficially atrophied and to a great extent covered by large, densely black patches of pigment of irregular shapes; some of the pigment is reticulated; some separate outliers of choroidal disease are seen beyond the Y. S. region; retinal vessels normal; O. D. healthy, but its choroidal ring strongly marked. V. $\frac{2}{20}$ fairly, and 1 J.; p. p. 8"; F. qualitatively defective over a broad zone in outer, upper and lower parts, of full quality inwards; no ring scotoma could be made out; fixation not eccentric. The appearances are those of superficial choroiditis with great proliferation of the pigment epithelium and infiltration of some of the pigment into the retina. Such changes involving, as they appear to do in this case, the fovea centralis, would be incompatible with such excellent vision as this eye possesses. We may suppose either (1) that a slightly eccentric fixation had been acquired and the function of
the corresponding spot of retina improved by practice, or (2) that the choroiditis in reality took place in the deepest layers of the choroid (lamina fusca) and did not affect the capillary layer at all; but the appearances are not in favour of this view, for the pigment masses are seen to lie \textit{in front} of the large choroidal vessels, not between or behind them.

The patient (Hy. Schroeder, 29) is a tall, muscular, black-haired German, intelligent and fairly educated (Moorfields Hospital, May 17th, 1884, 'Note-book,' p. 149) who came complaining of a "dark ring with a clear centre" in the middle of the R. field of vision. This appearance had, he said, come on suddenly one day five years before and had not altered since. He was in Australia at the time "prospecting" for gold. On the morning of the day in question he had, according to custom, drunk a large quantity of new milk. Shortly afterwards he had a heavy meal of mutton and potatoes, and this was followed by general severe headache. He went out walking, and noticing something wrong with his sight, covered each eye separately and found the defect to be in the R. Had been reading the newspaper the same morning and had not noticed anything amiss with the sight. Had no pain in eye. Not liable to epistaxis. No arthritic or syphilitic history. The sun used to affect his eyes (photophobia), but he had no ophthalmia or other disease of eyes. Complains of muscae lately. With the other (L.) eye V. \(\frac{2}{20}\) and 1 J.; H. m. 1 D.; Oph. normal.

This case seems to be best explained by assuming that a large extravasation of blood took place, at the date referred to, from a choroidal vessel at the centre of the fundus, and that some inflammation set up by the haemorrhage was followed by partial absorption of the choroidal structures.

\textit{Card specimen.} (October 9th, 1884.)
X. DISEASES OF THE OPTIC NERVE.

1. A case of amblyopia with partial optic atrophy and general nervous depression and emaciation, caused by the vapour of bisulphide of carbon and chloride of sulphur. Partial recovery.

By E. Nettleship.

James Wm. M—, æt. 20, came to the Eye Department at St. Thomas's Hospital on August 2nd, 1884, with the statement that his sight had failed for about three weeks. He was extremely pale, very thin, and showed marked general muscular feebleness; pulse 56, tongue flabby. He seemed dull and stupid.

About three weeks previously (on July 12th), whilst riding on an omnibus, he found that he could not see the people on the pavement. The next day he was still able to read the newspaper with difficulty, but since then the sight had got worse. He noticed that he saw worst in bright daylight. Objects did not appear coloured.

State of eyes on admission (August 2nd): V. with each eye separately \( \frac{5}{7} \), and reads 12 J. badly at 6'' (16 cm.); not improved by glasses; colour-perception perfect for blue and yellow; quite absent for red and green (confuses rose with blue, and has no idea whatever of any reds or greens). Unfortunately there is no written note of the ophthalmoscopic appearances, but I feel quite sure that I examined him and that there were no conspicuous changes. The fields taken three days after admission (August 4th) by Dr. S. W. Sutton, with a white square 10 mm. in the side, were of full extent, but acuteness and colour
perception were so defective that no test for scotoma could be applied.

He attributed the failure of his sight to working in the "curing room" of an india-rubber factory; he had been in this "curing room" for the past ten months, but before that date he had always worked in the open air. That his sight had previously been good is shown by his having passed as a recruit for the army about two years before I saw him. He married just before going to the india-rubber factory. His duty in the "curing room" was to pass india-rubber articles singly through an open bath consisting of bisulphide of carbon (32 parts) and chloride of sulphur (1 part). Much vapour was given off.

He did this work for four hours a day on an average, but just lately as much as five hours. He said that this part of the work was known to be unhealthy.* When he had been doing this "curing" for three or four months (i.e. six months before he came to me) his health began to fail with general weakness in all the limbs, and liability to nausea, but no actual vomiting. Some weeks before admission he began to suffer from headache "as if his head was opening and shutting"; on one occasion he had free epistaxis. He continued to sleep well, without dreaming, and his memory was not affected; but he became nervous. He had no numbness or pins and needles, and had not noticed any coldness of limbs.

He was taken into the ward a couple of days later, put upon generous diet, and kept in a subdued light. No evidence of disease of any internal organs was found, and

* It may be noted that his clothes and person generally had a strong pungent smell allied to the odour given off by hard vulcanite when rubbed or heated. This smell hung about his outer clothes all the time he was in the ward. As to the vapours of bisulphide of carbon being hurtful he stated that he knew another young man (sect. 22) who had worked at the same process in another factory, and had become so blind that he could not see to count his wages; he also had lost much flesh. The patient's fellow-workman in the same room as himself was a man of forty, and had been at the "curing" for six years; he had not suffered in his sight but was extremely thin.
the urine was natural. He thought that he began to see better about ten days after admission; on August 21st there was decided improvement, V. of R. being $\frac{20}{50}$, and of L. $\frac{10}{50}$. At that date the O. Ds. were somewhat pale all over, more so on the temporal side; there was some filmy haze over them, and the neighbouring retina showed the "watered silk" appearance; the large vessels, both arteries and veins, were very tortuous.

On 25th (three weeks after admission) he was so much better in health and so anxious to go home that he was discharged. Professor Fuchs of Liége, and Dr. Nelson of Belfast, who were at the hospital on that day, examined him and both thought that the retina at the Y. S. region was too visible and stippled.

He took no medicine whilst in the ward; but for the last two weeks of his stay the constant current was used, at first daily, then on alternate days only, as the daily application sometimes caused faintness. There seemed no reason for attributing the improvement of vision to this treatment. He has since attended as an out-patient and taken some strychnia.

On October 6th I found his sight a good deal improved; (V. = $\frac{20}{50}$ and words of 6 Jaeger slowly with each eye); colour-perception was much better and he could now recognise red and green fairly well, if the tests used were large. The colour of a red square of 10 mm. in the side was not recognised in any part of the F., but on using a much larger red test object the colour was seen at the fixation point, and still better over the inner part of the F.; but though outwards from the fixation point it looked colourless or "brown," and in the upper and lower parts it was only badly recognised. The distribution of this area of defective perception of red in the outer upper and lower parts of the F. was exactly symmetrical in the two eyes; there was in fact a large and ill-defined scotoma for red a little to the outer side of the centre in each field. The O. Ds. were now somewhat paler and clearer than at the previous note.
He was at this date well and strong; and his manner was much brighter than before. He still worked at the same factory but not in the "curing room," and was not now exposed to the same noxious vapour as before.

This man married a month or two before beginning to work in the rubber factory, and sexual exhaustion may possibly have predisposed him to the influence of the poison.

As possible exciting causes of the attack may be mentioned the hot weather and bright sunlight, for the failure took place in July and August; more vapour would be given off in such weather, and the bright light may have contributed to set up the subacute inflammatory process which no doubt took place in his optic nerves, just as exposure to bright light sometimes seems to excite an acute attack of tobacco amblyopia.

(October 9th, 1884.)

2. A case of amblyopia with slight neuritis followed by pallor of the discs, caused by the vapour of bisulphide of carbon and chloride of sulphur; severe nervous depression, emaciation, and muscular wasting. Recovery.

By Ernest Fuchs, M.D.,
Professor of Ophthalmology in the University of Liège.

(Communicated by E. Netteship.)

Elizabeth M—, æt. 23, was admitted at the Ophthalmic Department of the Liége Hospital under Dr. Fuchs on July 30th, 1883. For about six weeks she had been unable to read. On examination V. of R. was = fingers at 2 m. (about \( \frac{2}{10} \)), and of L. = fingers at 4 m. (\( \frac{4}{10} \)); Ps. of medium size and sluggish; Oph.: slight neuritis in each eye, O. Ds. pale and hazy, but not swollen, the
haze extending for a disc's breadth into the retina, veins engorged, arteries too small.

She was pale, thin, and weak; particularly, the muscles forming the thenar eminence of the thumb were markedly atrophied, just as in progressive muscular atrophy; the interossei also were similarly atrophied.

The patient stated that she had for several years past been working in an india-rubber factory, where she was constantly exposed to the fumes of bisulphide of carbon. Since taking to this work her health had become impaired, the most marked symptoms being weakness of limbs, particularly of the legs, coldness, and frequent formication. She also became subject to headache and giddiness, and lost her appetite.

Previous history.—Had convulsions between six and seven years of age, and again at the age of 10; they do not appear to have been epileptic. Was subject to headache and neuralgia till she was fifteen, when the catamenia began, and since which time they have been regular and copious. Her health was better for some time after puberty until she went to the india-rubber work.

She was taken into the ward on July 30th, and treated by subcutaneous injections of strychnia on alternate days, with iodide of iron internally and good food and wine.

On September 14th, V. in each eye was still only $\frac{5}{60}$.

On the 22nd, Dr. Fuchs, on returning from his holiday, took the fields of vision and found a small but well-defined central scotoma for red (Chart 8), none for white, but no limitation of field for white. He observes that the scotoma had probably been larger at an earlier period in the case.

By the end of this month (September) she had become much stouter and ceased to complain of weakness and formication, and the muscles of the thumb had become much more plump.

On October 21st, the field still showed a scotoma of the same size as before for red, and some doubtful contraction of the field for red (Chart 9).

November 17th.—V., both eyes together, $\frac{5}{18}$. 

AMBLYOPIA FROM BISULPHIDE OF CARBON. 153
A white object 1 centimetre square was used for the determination of the outer limits of the field, indicated by outermost white line. With a white object, or with a red one, each of 1 cm. square, no central scotoma is found. But with a red square of \(\frac{1}{2}\) cm. square the minute central scotoma for red, bounded by the innermost white line, is detected.

December 21st.—R., V. \(\frac{5}{18}\); L., V. \(\frac{5}{22}\).

February 23rd, 1884.—R., V. \(\frac{5}{12}\); L., V. \(\frac{5}{6}\); no scotoma.

(Chart 10.)

April 13th.—R., V. \(\frac{5}{9}\); L., V. \(\frac{5}{6}\). O. Ds. paler than normal, but sharply defined; retinal arteries a little diminished. No remains of atrophy of the interossei and thumb muscles.
Amblyopia from Bisulphide of Carbon.


I. Minute central scotoma for red.
II. Zone in which the red square (½ cm. side) is recognised as red.
III. Zone in which the red square (½ cm. side) is not recognised as red.

Remarks on the above cases.—In the ‘Edinburgh Medical Journal’ for May last (1884) Dr. Alexander Bruce has recorded three cases of chronic poisoning by bisulphide of carbon, and in one of these the patient, a man aged 49, lost his sight rapidly after a longer exposure than usual to the vapour in the ‘curing house.’ As in my own and Dr. Fuchs’s cases, the amblyopia did not come on until the man’s muscular and nervous systems had suffered severely for several months, and as in them his
The field was ascertained by means of a white object 1 cm. square. Its margin is indicated by the dotted line. No scotoma could be detected either with white or red test objects of any size.

sight and general state gradually and steadily improved, so that in three or four months vision had become perfect and his health restored; there were no ophthalmoscopic changes. The details of the case, which are very interesting, differed in some particulars from Dr. Fuchs's and mine. Unlike our patients, this man had delusions and frightful dreams, and for a time he had coloured vision, sometimes green, sometimes red; he rapidly lost all sexual desire and power.
AMBLYOPIA FROM BISULPHIDE OF CARBON.

Dr. Bruce's other two patients had the same general symptoms,—extreme muscular weakness and depression of the whole nervous system, but no failure of sight; one was 49, the other 39.

The case of Dr. Bruce and the two narrated this evening, together with the one, if we may assume it to have been one, in the friend of my patient (Footnote to p. 150), are, so far as I know, the only ones recorded in which damage to sight has been caused by the vapours of bisulphide of carbon and chloride of sulphur, and although such cases are fortunately never likely to be common, they are of great interest as illustrating in a new way the peculiar liability of the optic nerves, as compared with the other nerves of special sense, to be damaged by influences which depress the nervous system. (October 9th, 1884.)

P. S.—I knew nothing of the French literature on this subject until the Committee (named below) took it up.—E. N.


Poisoning by bisulphide of carbon has been known for many years to occur in a certain branch of the india-rubber manufacture, namely that in which the rubber is "vulcanised" by immersion in a solution of chloride of sulphur in bisulphide of carbon,* and partial blindness is also well known to be one of the symptoms of this poisoning. Two elaborate mémoires were written on this subject by Dr. A. Delpech in Paris in 18561 and 1863,2 and several inaugural theses have also been published there (References 3 to 6).

The symptoms, so far as we know, occur only from exposure to the above-mentioned fumes in the caoutchouc

* Known as the "Parkes process" or "cold process," and introduced in 1846 by Mr. Parkes, of Birmingham.
manufacture. The fluid employed consists of about two parts of chloride of sulphur to ninety-eight of bisulphide of carbon.* The chloride of sulphur seems to be used for scarcely any other trade purpose†; but bisulphide of carbon is largely employed for many other uses, such as the extraction of fats and oils, the preparation of varnishes, &c. In order to ascertain which of the two substances was to blame Delpech made numerous experiments on rabbits; he found that the action of the two was similar, but that the bisulphide of carbon was the more powerful, and that a mixture of the bisulphide of carbon and the chloride of sulphur did not produce a greater effect than the former alone. The action of bisulphide of carbon when inhaled by animals has also been more recently studied by Poincaré and Lewin,¹⁰ and this substance is now known to produce a definite train of symptoms both in man and the lower animals. It was used a few times as a general anaesthetic, side by side with chloroform, by Simpson and Snow, but was abandoned on account of its inconveniences.‡ It has also been used in the form of atomised vapour as a local anaesthetic.§

* The exact proportion is said to vary a little in different factories, each manufacturer keeping the precise composition of his own vulcanizing fluid secret. One informant mentioned 1 in 60 (or 1¾ per cent.) as the composition he was familiar with.

† Chloride of sulphur is used in refining sugar by "Eastes' process," from 2 to 8 ounces of it being mixed with 100 gallons of the raw sugar liquor in order to throw down the albuminous matter. Probably but little of the chloride escapes as vapour in this process. Poisoning by the bisulphide seems to occur in some trades where this substance is used without the chloride of sulphur. We are indebted to Dr. C. Davidson, of South Hackney, for information as to two cases, in which men engaged in a mill where the bisulphide of carbon is used for the extraction of palm-nut oil, suffered for a time from nervous depression, weakness, and symptoms in some respects like those of locomotor ataxy, though without noticeable failure of sight; they recovered and resumed work. Dr. Davidson's enquiries led him to believe that the cases he saw were not the only ones that had happened.

‡ Simpson says that it produces rapid and powerful anaesthesia, but sometimes causes depressing and disagreeable visions, and is followed by headache, giddiness, and vomiting (Pereira ¹⁰).

§ See Appendix, p. 167.
AMBLYOPIA FROM BISULPHIDE OF CARBON. 159

We may probably conclude from the above statements, that the fluid used in the "cold" or "Parkes" process of vulcanising owes most of its bad effects upon the health to the bisulphide of carbon of which it mainly consists; and this notwithstanding the belief among many of the French workers that their symptoms arose from the chloride of sulphur, a belief possibly founded on its having a more pungent odour than the bisulphide. The exemption from poisoning which those seem to enjoy who work with bisulphide of carbon in other industries may no doubt be explained by the fact that most of the oil-extracting, and similar processes in which it is used, are carried on in carefully closed vessels.*

In the cold vulcanizing process the workman's hands usually come into contact with the vulcanizing fluid, and local irritation arises from this; but that the toxic symptoms are due to the vapour is shown by the following evidence: (1) the rapidity of onset and the severity of the symptoms vary directly with the amount of vapour in the workshops; (2) a few cases have occurred in foremen who, though passing several hours daily in the "curing room," where the process is carried out, have not actually come into contact with the fluid; (3) in one case a decided improvement took place when an apparatus was used which prevented the inhalation of gases, although the man's hands continued to be immersed in the vulcanizing fluid for several hours a day.

The following is a short résumé of the usual course and symptoms of chronic poisoning by bisulphide of carbon as described by M. Delpech, to whose account more recent observations have not added anything of importance. Usually two stages can be distinguished: (1) exaltation; (2) depression or collapse. The first may, however, not occur, or not be indicated by well-marked symptoms; and the two are occasionally mixed, exaltation of some functions coinciding with depression of others.

The earliest and most constant symptom is a severe

* See Appendix, p. 167.
heavy pain in the forehead or temples, the head feeling as if compressed in a vice or supporting a heavy weight. Many persons begin to have this pain on the very day they enter the factory, and it often precedes the other symptoms by months or even years. It is attributed by the patients themselves to the offensive smell of the vulcanizing fluid, and in support of this belief is the significant fact that the only one of Delpech’s patients (No. xxiii) in whom this symptom was wanting, suffered from congenital absence of the sense of smell. Probably, therefore, this compressive headache forms no necessary part of the true toxic phenomena.

The earliest symptoms of the stage of exaltation are loquacity, vertigo, and a feeling as of drunkenness on going into the open air from the workshop. The temper becomes exceedingly irritable and the spirits variable, unusual hilarity alternating with deep dejection. The appetite is often enormously increased and excessive sexual excitement is not unusual. Vision sometimes becomes affected in this stage, the patients stating that all objects seem veiled in a mist; this symptom in some instances is only temporary, occurring towards the end of the day’s work, and in one case it came on only during fasting and was relieved by taking food. Some in whom there was vertigo complained of constantly seeing an open hole close to them into which they were afraid of falling. The hearing is impaired more frequently than the sight. General hyperaesthesia of the integument is sometimes present.

In the stage of depression the appetite usually fails, there is insomnia, or the sleep is disturbed by distressing dreams, the spirits become greatly dejected, and patients, who previously were active and intellectual, become utterly indifferent to surrounding circumstances and passing events. Failure of memory is an almost constant symptom and usually first shows itself as a forgetfulness of words in talking. The hyperaesthesia of the skin gives place to a loss of sensation in the limbs often reaching up to the groins and to the insertion of the deltoids, but not
affecting the trunk. Painful cramps and great muscular weakness, affecting especially the lower limbs, with loss or impairment of sexual desire are all very frequent symptoms in this stage, and complete anaphrodisia is not uncommon. The fingers become stiff, awkward, and numb; this may, however, be in part due to the direct action of the fluid. Visual troubles are more frequent in this stage than in the former, and consist in the appearance of a fog before the eyes and a dimness which is most marked in broad daylight. The pupils are usually abnormally large but retain their activity. The visual field is stated to have been normal in all the cases in which it was examined, but it probably was not tested for central scotoma. In some cases slight pallor of the discs, and some loss of definition of the margins, have been noted.

As long as the patient remains exposed to the fumes the symptoms continue to increase in severity; the loss of memory becomes absolute, and the muscular weakness so great that the patient is unable to stand without support. Improvement generally commences as soon as the patient is removed from the influence of the poison, but it is very gradual. Most of the cases were treated for some months with small doses of phosphorus, and in a large number recovery from the more important symptoms took place. The most intractable symptoms were the anaphrodisia and the weakness of the lower limbs. In every instance a relapse took place when work was resumed.

Previous to 1863 (the date of Delpech's second mémoire) these cases were exceedingly numerous, and Delpech states that all those employed suffered considerably in health. This was no doubt due to the fact that many of the men worked for long hours in small, heated, ill-ventilated rooms, in which the quantity of vapour was often sufficient to form a perceptible cloud near the floor, which could sometimes be ignited. Delpech also states that when the industry commenced, an unnecessary amount of vapour was produced owing to defects in the process which were afterwards remedied. Although a few cases did occur in
those who were working in open sheds, in these the onset was more gradual and the symptoms were less severe. Several, who had worked for years in open sheds without marked symptoms, suffered much on being transferred to close workrooms.

As to the frequency of these cases at the present time we cannot make any precise statement; the absence of any mention of the subject by Leber and Förster in Graefe and Saemisch's 'Handbuch'—published as recently as 1877—is remarkable, and would seem to indicate that they occur but rarely in Germany. The first cases published in England are, we believe, those by Dr. Alexander Bruce, (8) referred to in Mr. Nettleship's communication at the October Meeting of the Society.* Perhaps the frequency of the affection in France is chiefly due to the larger use of this process there than elsewhere.

We have written to fifteen of the leading india-rubber manufacturers in London and have obtained information from a few elsewhere. All the manufacturers are evidently aware of the unhealthiness of the Parkes' process, and special precautions seem to be taken in every manu-
factory in which it is employed. The shed in which it is carried on is either entirely open on one side, or very freely ventilated; and in several of the replies it is specially mentioned that the men are not allowed to work in the curing room for many hours consecutively. Only one of the replies (that from Messrs. George Angus and Co.) mentions in any detail the ill-effects of the process; they say that persons constantly employed in curing india-
rubber by the process in question, suffer from "failure of sight, loss of muscular energy, very defective appetite, and become in a few years old men." They add that the other processes of india-rubber making are not thought, by those employed, to be more unhealthy than the gene-
rality of confined occupations.

This process of curing is not largely used in Britain

* We wish here to acknowledge our indebtedness to Dr. Bruce for additional information which he has been kind enough to send us on the subject.
AMBLYOPIA FROM BISULPHIDE OF CARBON. 163

except by a few firms, being only necessary for the smaller and finer kinds of goods. Hence no doubt the meagreness of most of the replies we have received.

The only factory in London where we had the opportunity of seeing the process carried out is that of Mr. Henry Bell, in York Road, Lambeth, where the case read at the October Meeting of the Society occurred. Mr. Bell's factory is, we understand, one of the few in London where this process is largely employed. When we were there, on November 20th, a man was engaged in the curing room, in dipping small india-rubber goods such as teats, enema bottles, &c. The liquid was in an open earthenware trough over which he was obliged to hold his head as he dipped each article in succession. The article is immersed for a few seconds, during which the bisulphide permeates the texture of the india-rubber, carrying with it the chloride of sulphur which is the vulcanising agent. On being taken out it is allowed to drain over the bath for a few seconds, and is then placed on one side to dry, and owing to the extreme volatility of the bisulphide this occurs very quickly. Although the room was cold and several large skylights were freely open, there was a strong odour, of a heavy and sickening character, arising from the combined vapours.* Mr. Bell told us that he had not had any cases of serious illness caused by working in the curing room; one man lived to sixty-five after working there for many years, and another, under the same circumstances, lived to be an old man. Recently, however, the case of the young man M—, that was read at the October meeting (p. 149) had occurred, and another much older man (referred to in the footnote to p. 150) had been moderately affected and had gone into St. Thomas's Hospital, though his sight had

* The pure chloride of sulphur is a sherry-coloured liquid, which gave off visible fumes when the stopper was taken out of the bottle, smelling somewhat like volatilised sulphur. The commercial bisulphide of carbon has a very disagreeable odour, but according to the 'Pharmaceut. Journ.' (ser. ii, p. 641, 1869-70) it has, when quite pure, a smell allied to that of chloroform.
not suffered. In Mr. Bell’s factory, as in the other English ones, already referred to, the men never work for long at a stretch in the curing room.

No means have hitherto been devised which have succeeded in entirely preventing the occurrence of such cases, although they are probably less frequent than formerly. The vapour of the bisulphide is so inflammable that the creation of draughts by fire, in any form, is quite out of the question. Ordinary respirators are often provided for use, and the man we saw at work was using one; but unless it can be provided with some substance capable of chemically separating the deleterious vapour from the inspired air, it can be of little or no value. Perhaps a face-piece and inspiration tube communicating directly with the outer air, and provided with a valve for expiration, might be practicable.

As the subject is a comparatively new one in this country, we have thought it well to add a tabular statement of all the cases of amblyopia from bisulphide of carbon which we could find, viz. fifteen from Delpech and nine from other sources. Of the frequency of amblyopia in chronic poisoning from bisulphide of carbon, some idea may be formed from the fact that no less than fifteen of the series of thirty-three cases reported by Delpech in his two mémoires were so affected.

Conclusions.—We have tabulated twenty-four cases, being all that we could find in which vision was affected. Of these, twenty-two were in men; but two well-marked cases occurred in women, and the excess of males is probably accidental.

The age when the patients were seen varied from 15 to 52; of these no less than ten were under 25, and seventeen were under 40.

The prognosis is usually good, or at least fair, if the sufferer can entirely give up the curing work. Thus, of the twenty-four cases, eight (Nos. 1, 2, 11, 13, 17, 19, 20, 23) are noted as having recovered perfect or very good vision, and seven others (Nos. 5, 8, 12, 14, 15, 18,
AMBLYOPIA FROM BISULPHIDE OF CARBON. 165

21) as having improved more or less. In five (Nos. 4, 6, 10, 16, 24) there was little or no improvement, but one of these (No. 16) was a doubtful case, and another (No. 10) resumed work after temporary improvement. In four cases (Nos. 3, 7, 9, 22) the notes were incomplete.

No conclusion can be drawn as to the length of exposure to the noxious influence that is necessary for the production of amblyopia; the sources of error being too many, and the notes frequently imperfect. Slight toxic symptoms are often noticed within a few days of the first exposure, but the date of onset of the more severe symptoms, including the failure of vision, seems very variable.

The amblyopia is never an isolated symptom, it never occurs without well-marked general toxic symptoms. In most of the cases which were seen by oculists decided changes were found at the optic discs; in the earlier period haziness and other signs of chronic neuritis, in the later stages some degree of atrophy or pallor. In some, central defect of the visual field has been found, but as a rule the field was not carefully examined.

Several of the patients are noted as being smokers; and probably many others, where this is not stated, were so. None are stated to have been drinkers; one (No. 5) was a total abstainer, and several are noted particularly as being temperate.

W. ADAMS FROST,
R. MARCUS GUNN,
E. NETTLESHP.

(Presented January 8th, 1885.)

List of References.

(1) DELPECH, A. 'Mémoire sur les accidents que développe chez les ouvriers en caoutchouc l'inhalation du sulfure de Carbone en Vapeur,' Paris, 1856.
DISEASES OF THE OPTIC NERVE.


(4) Gourdon, P. ‘ De l’intoxication par le Sulfure de Carbone’ (Thesis), Paris, 1867.


(7) Galezowski. ‘Rec. d’Ophthalmologie,’ 1877, ii, 121. Two new cases, but most of the paper is a reproduction of Delpech’s ‘Mémoire.’


(9) Information on the uses of Bisulphide of Carbon and Chloride of Sulphur in various trades will be found in:—Payen: ‘Industrial Chemistry’ (Paul’s translation, 1878).


‘Cooley’s Cyclopaedia,’ 1880, p. 404.


Lewin: ‘Virchow’s Arch.,’ vol. 78, p. 113.
Appendix.

Apparatus designed by Delpech's patient, No. xix (Case 11 in the following tables), for avoiding inhalation of the fumes.

In a chamber, which can be freely ventilated through its two extremities, a horizontal board is fixed to the sides by its two ends; from its anterior edge a board descends to the floor of the chamber; from its posterior rises a vertical plank, fourteen inches high; this is pierced by three pairs of apertures so arranged as conveniently to give passage to the hands and forearms of three workmen. From the upper border of this vertical plank a sheet of glass passes upwards and forwards, and allows the workers to see their hands. The closure of the chamber is completed anteriorly, so that the only communication between it and the remainder of the room is through the six circular apertures, these are protected by impermeable and supple india-rubber, which fits closely to the wrists of the workers by means of bracelets. The workmen sit with their legs beneath the horizontal table. All the operations are carried on within the chamber. It is stated that no odour was perceptible, and that although the operations were slightly retarded they were not so to any inconvenient extent.
Abstracts of Cases of Amblyopia caused by the Fumes of Bisulphide of Carbon. All the Cases, except No. 19, have been published.

<table>
<thead>
<tr>
<th>Author</th>
<th>Age</th>
<th>Sex</th>
<th>Time employed in work</th>
<th>Previous general health, &amp;c.</th>
<th>Eye symptoms</th>
<th>Other symptoms</th>
<th>Result, remarks, &amp;c.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Delpech (II) 1</td>
<td>24</td>
<td>M.</td>
<td>2 years</td>
<td>Good; temperate habits</td>
<td>Headache from first day. in a few weeks pains in limbs, and became very excitable; dimness of V., objects appearing as if seen through a fog; this would disappear shortly after leaving work. Later, loss of memory and muscular fatigue, exaggerated appetite, sexual excitement. After a year’s work left it for 4 months,—abnormal appetite and sexual excitement disappeared. On resuming work they returned, then drowsiness, progressive muscular weakness and loss of memory, then anaphrodisia.</td>
<td>Left work and recovered.</td>
<td></td>
</tr>
<tr>
<td>Ibid. (III) 2</td>
<td>21</td>
<td>M.</td>
<td>9 years, with interval</td>
<td>Good</td>
<td>Headache and feeling of intoxication from first day. Worked 6 to 8 hours a day. Soon insomnia, nausea, occasional deafness. In 6 months memory had failed, walking difficult. Left work.—recovered. 5 years later resumed work, all symptoms returned. V. became less clear, objects veiled in fog, unable to read by artificial light, hearing dull. 18 months later great weakness of lower limbs. Left work and improved. Resumed work,—sexual excitement followed by anaphrodisia. After a year sudden loss of consciousness, lasting three hours, from excess of vapour; became worse; pallor, emancipation; pupils dilated, active. Left work.—2 months later, V. clear to 15 cm., beyond this “fog”; pupils dilated and sluggish, slight dilatation of retinal veins.</td>
<td>Relapse of general symptoms occurred on resuming work. Almost complete recovery.</td>
<td></td>
</tr>
<tr>
<td>Ibid. (IV) 3</td>
<td>30</td>
<td>M.</td>
<td>5 yrs. (?) Good; intelligent; habits temperate</td>
<td></td>
<td>Worked in small room—much vapour. At one time he passed 19 days and nights in workshop, sleeping only for very short periods. Feeling of intoxication on leaving work, later headache; sleep disturbed, became irritable, dejected, Not stated.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
indifferent to surroundings; great loquacity, but frequent loss of memory for words; excessive appetite, then anorexia; stiffness of lower limbs; anaphrodisia. V. affected at age of 26, "fog" veiling all objects; near V. not improved by a convex lens; fog most perceptible when fasting; after a meal could often read; pupils dilated, active. After working 2 years all symptoms aggravated. Left work and improved, except V., which remained stationary. 18 months later resumed work cautiously, but lost all he had gained,—relapse of excitement, loquacity, and loss of memory returned. After a year V. 7 J. with difficulty; visual fields normal; pupils moderately dilated, active; slight pallor of inner part of O. D.; cupping more marked than normal; some loss of clearness of disc margins (sa transparence est minoindre). Is now a moderate smoker, formerly spent 30 to 40 centimes a day on tobacco.

<table>
<thead>
<tr>
<th>Ref.</th>
<th>Age</th>
<th>Gender</th>
<th>Duration</th>
<th>Occupation</th>
<th>Symptoms</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ibid. (VI) 4</td>
<td>45 M.</td>
<td>10 mos. (?)</td>
<td>Habits temperate</td>
<td>He came into actual contact with the vulcanising fluid but little. Headache from commencement; anorexia; diarrhoea. Soon vision seriously affected. Unable to read small print (presbyopia not excluded); quite deaf with left ear, hearing dull in right; memory failed; great irritability; difficulty in talking; malaise; general weakness; pains in lower limbs; difficulty in walking; partial anaphrodisia. Sleep disturbed by distressing dreams; vertigo; a sensation as if he saw a hole close by, into which he was in danger of falling; V. and memory became worse. Left off vulcanising; but continued in the factory.</td>
<td>V. almost completely lost; hearing improved; meniscal feebleness and anaphrodisia persisted.</td>
<td></td>
</tr>
<tr>
<td>Ibid. (IX) 5</td>
<td>21 M.</td>
<td>3 years, including an interval</td>
<td>A total abstainer</td>
<td>Worked at first in ill-ventilated room. Severe headache from commencement. Excitement; great impairment of V.; diplopia; ardent thirst; painful twitchings in limbs. Became worse, and after a year left work and improved greatly. Resumed work in an open shed,—became drowsy and dejected; vertigo; sexual excitement, followed by anaphrodisia.</td>
<td>Relapse of general symptoms on resuming work. Final result not stated.</td>
<td></td>
</tr>
<tr>
<td>Author.</td>
<td>Age</td>
<td>Sex</td>
<td>Time employed in work</td>
<td>Previous general health, &amp;c.</td>
<td>Eye symptoms.</td>
<td>Other symptoms.</td>
</tr>
<tr>
<td>---------</td>
<td>-----</td>
<td>-----</td>
<td>-----------------------</td>
<td>--------------------------------</td>
<td>---------------</td>
<td>-----------------</td>
</tr>
<tr>
<td>Delpech (X)</td>
<td>6</td>
<td>M.</td>
<td>2 years, including an interval</td>
<td>—</td>
<td>Headache from commencement. Exaggerated appetite; sexual excitement; sleep deep and prolonged; loss of memory; deafness when at work; V. much impaired, and said to have been more so earlier.</td>
<td>—</td>
</tr>
<tr>
<td>Ibid. (XIV)</td>
<td>7</td>
<td>M.</td>
<td>—</td>
<td>—</td>
<td>At first worked at “cutting,” then no symptoms; immediately on commencing “curing,” pain in head. Soon muscular weakness; anorexia; loss of sleep; impairment of memory; dejection of spirits; delusions—heard voices, and believed that he had committed a robbery. Treated and nearly recovered. Resumed work,—relapsed. No anaphrodisia; hearing impaired. 3 months after resuming work permanent and progressive impairment of V.; numeae. V. better in twilight; pupils regular, contracted, and quite inactive to light, but dilated under atropine. V. 15 J.; optic papilla congested.</td>
<td>—</td>
</tr>
<tr>
<td>Ibid. (XV)</td>
<td>8</td>
<td>M.</td>
<td>4 years Habits temperate</td>
<td>—</td>
<td>Worked 6 to 9 hours daily in close, ill-ventilated room—much vapour. First day, headache and feeling of intoxication on leaving workshop; gradually became worse. 4 months after commencement memory very defective; difficulty in talking. Became exceeding irritable, then much dejected. V. became impaired, everything appearing as if seen through a fog; pupils normal; hearing dull; considerable sexual excitation, which continued; walked with difficulty; easily fatigued; became cachectic and aged; had several attacks of loss of consciousness within a few days. Workshop ill-ventilated. Headache since first day. Insatiable appetite; pains in limbs and joints. V.—All objects veiled by a fog; appearance as of a spider’s web before eyes; obliged to blink to see clearly; made mistakes as to the form of objects; saw obstacles in front of him which did not exist. Naturally quick-tempered, he became exceeding</td>
<td>—</td>
</tr>
<tr>
<td>Ibid. (XVII)</td>
<td>9</td>
<td>M.</td>
<td>4 years</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Reference</td>
<td>Age</td>
<td>Duration</td>
<td>Recovery</td>
<td>Description</td>
<td></td>
<td></td>
</tr>
<tr>
<td>-----------</td>
<td>-----</td>
<td>----------</td>
<td>----------</td>
<td>-------------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ibid. (XVIII) 10</td>
<td>52 M.</td>
<td>2 years</td>
<td>Good (interval)</td>
<td>Worked 11 hours a day in abundant vapour. Soon headache at first transient, then constant, often necessitating 2 to 3 days rest; anorexia. V. soon became affected, objects appearing as if seen through a fog; vision. Hearing dull; several attacks of semi-unconsciousness with convulsive movements of the limbs, lasting half a minute; progressive loss of memory; speech difficult; anaphrodisia without previous excitation; at first became very irritable, then dejected; gradual loss of sensation in R. hand. After 22 months sudden loss of consciousness, lasting 7 days, and complete right hemiplegia; total blindness of R. eye, great impairment of L. eye. In 2 months partial recovery of motion, and complete of sensation; memory improved; V. still defective but could read. Resumed work,—hemiplegia returned; V. became worse; total anaphrodisia. A year later pupils sluggis, dilated, especially L.; R. eye retinal vessels diminished, fine floating filaments and specks in vitreous. Treated by rest, tonics, and phosphorus.</td>
<td>Relapse of general symptoms on resuming work. Headache became less intense, intermittent, and disappeared. Hemiplegia recovered, and partial recovery from the anaphrodisia. Memory remained defective, but speech became normal. Vision not stated.</td>
<td></td>
</tr>
<tr>
<td>Ibid. (XIX) 11</td>
<td>34 M.</td>
<td>5 years</td>
<td>—</td>
<td>Sometimes worked 12 hours a day in much vapour. Headache since first day in temples and occiput; became very irritable; two attacks resembling hysterical fits, with convulsive movements of the face; frequent vertigo; laughter without cause; weakness of legs, numbness of fingers, and inability to hold small objects. Three years after commencement, from stress of work, exposed for several days to excess of vapour; V. now became affected; unable to read, everything veiled in fog; pupils active; voracious appetite; partial anaphrodisia; memory impaired. Constructed an apparatus which enabled him to work without inspiring the vapour; began to improve. Some months later pupils dilated but active; V. P. normal. V. = 10 J. with difficulty, O. D. pale, “deeply cupped,” less transparent than normal. (Patient a moderate smoker.) Treated with phosphorus.</td>
<td>While continuing work with special apparatus improved in most respects; anaphrodisia disappeared; V. remained defective. Left work,—5 months later could read a letter.</td>
<td></td>
</tr>
<tr>
<td>Author</td>
<td>Age</td>
<td>Sex</td>
<td>Time employed in work</td>
<td>Previous general health, &amp;c.</td>
<td>Eye symptoms. Other symptoms.</td>
<td>Result, remarks, &amp;c.</td>
</tr>
<tr>
<td>--------------</td>
<td>-----</td>
<td>-----</td>
<td>-----------------------</td>
<td>-----------------------------</td>
<td>-------------------------------</td>
<td>---------------------</td>
</tr>
<tr>
<td>Ibid. (XX)</td>
<td>31</td>
<td>M</td>
<td>12 years</td>
<td>—</td>
<td>Acted as a foreman, did not work himself. Suffered from headache but no other symptoms. Two years after commencement accidentally exposed to more abundant vapour than usual; lost consciousness. Since then loss of appetite and fatigue. He passed most of his time in an office apart from workshop, but to which smell of sulphur penetrated. Irritability; loss of memory; disturbed sleep with painful dreams; vertigo; pains in limbs; V. obscured by a &quot;fog;&quot; sexual excitement. On being exposed continuously to more vapour became worse and V. more defective.</td>
<td>Left work improved.</td>
</tr>
<tr>
<td>Ibid. (XXI)</td>
<td>21</td>
<td>M</td>
<td>6 years</td>
<td>—</td>
<td>Worked occasionally for 4 or 5 days at a time vulcanising, room well ventilated (in the factory cutting caoutchouc for 4 preceding years). Headache; vertigo; muscular weakness; loss of memory; irritability, later dejection; V. obscured by &quot;fog;&quot; pupils dilated, sluggish (R. eye defective from injury). At 15 sexual excitation, at 18 almost total anaphrodisia.</td>
<td>Relapse; phosphorus and tonics. Left work; recovered. Subsequent temporary relapse after sleeping with a workman who was thoroughly impregnated with sulphur.</td>
</tr>
<tr>
<td>Ibid. (XXIV)</td>
<td>34</td>
<td>M</td>
<td>3 years</td>
<td>A strong athletic man</td>
<td>Worked 6 hours daily in well-ventilated workshop. Headache since first day, specks and flashes before eyes. After some time excessive loquacity, irritability, insomnia, exaggerated appetite, and great sexual excitement; stage of excitement lasted 2 years; then removed to ill-ventilated room; headache worse, vertigo, loss of memory, difficulty in walking, dejection and loss of energy; V. obscured by &quot;fog;&quot; pupils perhaps somewhat dilated but active. A smoker, but not to excess. Slight anaphrodisia.</td>
<td>Recovered almost entirely in 5 weeks. V. improved, but remained defective; O. Ds. atrophic, especially L.</td>
</tr>
<tr>
<td>Name</td>
<td>Sex</td>
<td>Age (years)</td>
<td>Duration</td>
<td>Condition</td>
<td>Description</td>
<td></td>
</tr>
<tr>
<td>--------------</td>
<td>-----</td>
<td>-------------</td>
<td>----------</td>
<td>------------------------------------------------</td>
<td>-------------------------------------------------------------------------------------------------</td>
<td></td>
</tr>
<tr>
<td>Galezowski,</td>
<td>M</td>
<td>2 (?12 yrs.)</td>
<td></td>
<td>Amblyopia</td>
<td>For several years only slight headaches towards end of afternoon; definite symptoms about 2 years (at. 41); vomiting, pains in belly; painful cramps, followed by such muscular weakness that he could not stand; all symptoms improving after a few days rest, but returning whenever work was resumed. V. affected between 1 and 2 years later (4 to 5 months before G—saw him); V. <em>2</em> better than R., V., F. n., colour V. n., constant flashes and spots before his eyes, dazzled by bright light; O. D. pale, especially outer part, and margin very hazy (“perineuritis”).</td>
<td></td>
</tr>
<tr>
<td>Rec. d'Ophth.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1877, p. 130</td>
<td></td>
<td>15</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ibid.</td>
<td>M</td>
<td>Several</td>
<td></td>
<td>Amblyopia</td>
<td>Suffered from symptoms of bisulphide poisoning after having worked several years in the curing room. He lost his sight and both discs passed into atrophy. (N.B.—This case is possibly the same as Case 4 in this series, but the details are insufficient to decide this question.)</td>
<td></td>
</tr>
<tr>
<td>pp. 131-2</td>
<td></td>
<td>16</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bruce, A.</td>
<td>M</td>
<td>4 years</td>
<td></td>
<td>Amblyopia</td>
<td>Had been in same factory for some time before going to the “curing” work. After this no severe symptoms for about 2 years, when, from a change of process, the vapour became more concentrated. He then became frequently sick and easily fatigued; some incontinence of urine came on; alternated between mental excitement and depression, and had frightful dreams; sexual power entirely disappeared. Then, in about 3 months, legs so weak he had to give up work and was in bed 6 weeks; L. leg almost paralysed; had numbness, cramps, and coldness of feet and legs. Then returned to work better. Though the symptoms returned he kept at work 3 months, when V. began to get rather misty. One day when very bad he vomited, and then found on going out of doors, to get refreshed, that V. was much worse; could not distinguish objects, and everything was bluish-green. At a hospital it was found he could only see the largest test letters held very close. The day after atropine had been used he had an attack of erythropsia (“rose-coloured” vision).</td>
<td></td>
</tr>
<tr>
<td>Case 1</td>
<td></td>
<td>17</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**OBSERVATIONS ON AMBLYOPIA FROM BISULPHIDE OF CARBON.**

After 2 months abstinence from work, with treatment by phosphorus, decided improvement of sight and general condition; discs clearer but white. Then lost sight of.

General symptoms soon improved on his quitting the work; “sight also seemed to become stronger whilst he remained under care.”

After the above bad attack he went into the country, and V. improved; erythropsia disappeared in 7 weeks, and in 3 to 4 months he could read small print again easily. The fundus of each eye was perfectly healthy when examined after recovery. Other symptoms entirely disappeared.
<table>
<thead>
<tr>
<th>Author, Case</th>
<th>Age</th>
<th>Sex</th>
<th>Time employed in work</th>
<th>Previous general health, &amp;c.</th>
<th>Eye symptoms</th>
<th>Other symptoms</th>
<th>Result, remarks, &amp;c.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nettleship 18</td>
<td>20</td>
<td>M.</td>
<td>10 mos.</td>
<td>Fair. Married just before beginning India-rubber work. Had been at out-door work</td>
<td>General muscular weakness, headaches, pallor; no loss of sexual power; V. ( \frac{5}{6} ) and 12 J. with each, no contraction of F., central defect for red, O. Ds. pale and slightly hazy.</td>
<td>Partial recovery of V. and improvement on leaving curing work.</td>
<td></td>
</tr>
<tr>
<td>(unpublished) 19</td>
<td>23</td>
<td>M.</td>
<td>—</td>
<td>—</td>
<td>Attended at Moorfields for failure of V., which he attributed to working in &quot;curing room,&quot; but which I at the time diagnosed as probably due to tobacco. This case was under care as a case of &quot;Tobacco amblyopia at an unusually early age&quot; before I knew anything of amblyopia from bisulphide of carbon. Mr. J. Hutchinson, jun., who took careful notes of the case, drew my attention to the man's own statement that he believed the bisulphide had done him harm, and I have to thank Mr. Hutchinson for now reminding me of the case. Unfortunately the notes were on the man's letter and were not kept at the hospital, but there is no doubt about his recovery as stated in the next column.—E. N.</td>
<td>Recovered vision quite well on leaving his work, and afterwards went back to the same occupation. No doubt he was told also to leave off smoking.</td>
<td></td>
</tr>
<tr>
<td>Fuchs 20</td>
<td>23</td>
<td>F.</td>
<td>&quot;Several years&quot;</td>
<td>—</td>
<td>V. ( \frac{4}{6} ), O. D.'s pale and hazy; muscular weakness, pallor, loss of flesh, wasting of interosseous muscles.</td>
<td>Almost complete recovery of V. and of health 8 months after leaving work. During the course of the case a well-defined central scotoma was found in each F.</td>
<td></td>
</tr>
<tr>
<td>Huguin, Case 1 21</td>
<td>15</td>
<td>M.</td>
<td>8 months</td>
<td>—</td>
<td>At first daily vomiting; afternoon headache and alcohol-like intoxication. After 4 months of exposure, pale, muscular</td>
<td>13 months after beginning the</td>
<td></td>
</tr>
<tr>
<td>Author</td>
<td>Case</td>
<td>Sex</td>
<td>Age</td>
<td>Duration</td>
<td>Symptoms</td>
<td>Treatment</td>
<td></td>
</tr>
<tr>
<td>-----------------</td>
<td>------</td>
<td>-----</td>
<td>------</td>
<td>----------</td>
<td>--------------------------------------------------------------------------</td>
<td>---------------------------------------------------------------------------</td>
<td></td>
</tr>
<tr>
<td>Ibid., Case 11</td>
<td>22</td>
<td>F.</td>
<td>15</td>
<td>2 years</td>
<td>—</td>
<td>Work, and after some months of rest and treatment, V. had improved but was still weak, and muscular power still deficient.</td>
<td></td>
</tr>
<tr>
<td>Ibid., Case 14</td>
<td>23</td>
<td>M.</td>
<td>20</td>
<td>20 years</td>
<td>Acute symptoms soon came on, with afternoon intoxication; V. failed, objects seeming green, then red, and foggy; became excitable and had headache.</td>
<td>Left the work. General state had improved a month later, but no note of V.</td>
<td></td>
</tr>
<tr>
<td>Delpech (11), 1856; Delacroix</td>
<td>24</td>
<td>M.</td>
<td>25</td>
<td>5 years</td>
<td>No symptoms for 5 or 6 years</td>
<td>Treated with phosphorus and recovered.</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>After 3 years work changed, and he was exposed to more of the fumes. Then nausea, cold sweats, and general weakness came on, and V. failed &quot;suddenly,&quot; with a sudden access of vertigo, which lasted only an hour. Continued his work; vertigo returned, and V. got worse, and memory failed. Now ceased work.</td>
<td>Months after leaving the work V. still very dim; could not recognize people at 2 inches; muscular weakness still remained, and partial anaphrodisia was present. Memory had improved.</td>
<td></td>
</tr>
</tbody>
</table>
4. Case of symmetrical coloboma to the temporal side of each optic disc.

By W. H. Jessop.

(With Plate V. fig. 1.)

T. L—, æt. 17, came to the Central London Ophthalmic Hospital on March 24th, 1885, as he found that suddenly there was a thick fog before the left eye. He had always had bad sight in both eyes, but never remembered his eyes being red or inflamed. He is the third child, the others being healthy.

There are the cicatrices of old cracks at the corners of his mouth, and the teeth are a little suspicious of hereditary syphilis.

Right eye V. = \( \frac{6}{0} \); J. 16 at 22 cm. H. m. 4 D. \( \frac{6}{36} \). Cornea clear; iris apparently healthy, no sign of coloboma of the iris or ciliary body.

Lens two irregular capsular opacities, and a few striae. Fundus, from external side of disc leading outwards is a large oval, well-defined patch of white sclerotic, having on it a few small patches of pigment. Some retinal vessels pass across it, but the main branches are at its upper and lower borders; a short distance from the outer margins of the large patch of sclerotic are several posterior ciliary vessels. The lower edge of the atrophied patch is the better defined, and deeply pigmented; the upper edge shades off gradually into the retina.

The hypermetropia at the disc is about 7 D., but varies in parts especially towards the lower edge. There is a well-marked scotoma for white.

Left eye V. = \( \frac{6}{0} \), J. 19 at 22 cm.; not improved. Cornea and iris as in right eye. Lens numerous striae, which prevent the fundus from being well seen. From the outer part of the disc, and surrounding the fundus more than in the right eye, there is a large patch of white sclerotic to
DESCRIPTION OF PLATE V.

Fig. 1 illustrates Mr. Jessop's case of Symmetrical Coloboma to the temporal side of each Optic Disc (p. 176).

Fig. 2 illustrates Mr. Hartridge's case of Opaque Nerve Fibres (p. 177).

(From drawings by A. Stanford Morton.)
be seen, but the edges are not so well defined apparently; some retinal vessels pass across it, and the posterior ciliary vessels also emerge from it as in the right eye. There is more pigment to be seen on the atrophied area, and one large boss of pigment above lies outside the atrophied part.

From the above description this case seems to agree with two cases reported by De Wecker* and Nuel† as posterior temporal colobomata of the optic nerve. One point of great interest is that it occurs in a hypermetrope, whereas the other two cases were both myopes, and also that in this case there is a symmetrical coloboma in each eye. The sudden failure of sight for which this patient came was apparently due to the increase in his lenticular trouble; the lenticular opacities, and pigment in the left eye may have been due to hereditary syphilis, which his general aspect rather denoted. That the large symmetrical atrophied areas should be due to choroiditis alone is very improbable, as there is so very little evidence of pigmentary disturbance.

(May 14th, 1885.)

5. A case of opaque nerve-fibres.

By G. Hartridge.

(With Plate V. fig. 2.)

Mary H—, æt. 38, married, came under my care at the Royal Westminster Ophthalmic Hospital for asthenopia. The sight in the left eye she said was good, but the right was blind and had "turned out" ever since she could remember.

Vision \[
\begin{align*}
\text{R. hand reflex only.} \\
\text{L. 6/6 no H. m. J. 1.}
\end{align*}
\]

* Graefe und Saemisch, 'Handb.', t. iv, p. 595.
† 'Annales d'Oculistique,' Mars-Avril, 1885, p. 174.
Right divergent strabismus. Left fundus normal. Right fundus, opaque nerve-fibres present to a very unusual extent, they completely encircle the disc, and extend for a long distance over the fundus, as shown in the drawing by Mr. Morton. This eye was highly myopic (—16 D.).

(January 8th, 1885.)

6. Optic neuritis, increased tension, nasal polypi, numbness of face, slight hemiplegia and albuminuria.

By W. A. Brailey.

Charles W—, æt. 42, bricklayer, admitted into Guy's Hospital from Dr. T. A. Richardson, of Croydon, on September 3rd, 1884.

Patient is a short, thickset and rather torpid-looking man, with hesitating speech and slight left hemiplegia. He is somewhat puffy below the eyes; pupils slightly dilated and very sluggish. On admission, L. eye had T. 2 and R. T. 1.

R. has a point-like posterior synechia below, and a second near to it on the inner side. The O. D. is decidedly red, also ill-defined and swollen somewhat, so that the vessels, especially those running upwards, dip down and are partially obscured at its edge. Beyond this they are tortuous. V. = 1/2, but varies from time to time. Cornea and face of this side have slightly diminished sensibility. L. has no synechiae. The O. D. is ill defined, less red than R., possibly very slightly swollen; beyond its edge the vessels, though of good size, are tortuous. V. = not p. 1. The cornea and face of this side are less sensitive than normal, but the difference is not so marked as on the other side.

His urine contains a small quantity of albumen; it
OPTIC NEURITIS.

is acid, sp. gr. = 1012. Dr. Mahomed reports that the patient has probably an early stage of chronic Bright's disease with granular kidney, for his pulse, though it gives the impression of being small and of low pressure, is really one of contracted arteries with decidedly increased arterial pressure.


History.—Hard sore twenty-four years ago; four years later left side became rather suddenly hemiplegic. The attack began with a loss of consciousness lasting several days. Five years ago he had a fit, not followed, however, by any fresh paralysis. Soon after this he had obstruction in his nostrils and several polypi were removed. Two years ago he had dreadful neuralgic pain in his head, for which he had thirteen teeth extracted; the pain continued off and on till three months ago. One year ago the left side of the face, corresponding to the distribution of the superior maxillary nerve, became numb. Several more polypi were now removed from the left nostril. Two months ago the L. eye began to fail and got gradually worse till three weeks ago, when p. l. ceased. Five weeks ago the right side of the face became numb. This numbness was preceded by a few days by a failure of vision of the corresponding eye.

His left patellar reflex is decidedly stronger than the right and may be exaggerated, as is the ankle clonus of this side. His blood preserves its fluidity for long, and flows very freely.

His eldest child has the typically notched teeth of
inherited syphilis. The marriage took place twelve years ago. There are five children.

After admission the tension soon fell to normal under eserine. The vision of the R., which was gradually failing, ceased to deteriorate after the removal of polypi from each nostril. These, about nine in number, each some $\frac{1}{2}$ inch in diameter, presented the ordinary gelatinous appearance of nasal polypi. They were not sufficient to block the nares and had no appearance of compressing or being compressed forcibly. Now, however, the O. D. of the right side ceased to get more pale and the numbness of corneae and face became less marked. Indeed it was difficult to trace some ten days later except over the right cornea. The albumen also became less and was now only appreciable by the fine tests of Dr. Mahomed.

The vision remained stationary, but he regained no power in his side.

(Living specimen. October 9th, 1884).

7. Symmetrical enlargement of upper part of face, with double proptosis and optic atrophy, complete anosmia and paralysis of left portio dura.

By R. Marcus Gunn.

Susan E—, æt. 22, married, attended at Moorfields Eye Hospital on 6th June, 1885.

The patient is one of two children, both of whom had thrush severely on mouth and bottom soon after birth. There is no further suspicion of inherited syphilis. There is no family history of phthisis, or of importance in other respects. Until the age of 16 her health was good, but for nine months preceding the onset of her catamenia at seventeen years she suffered almost daily from severe neuralgia in both temples, and says that at this time the whole
of her face was swollen, and she lost her smell partially. There was no further ailment until eleven months ago, when she began to vomit daily and often, being at the time two months pregnant with her first child. The vomiting continued for three months, and then only recurred at long intervals. Seven and a half months ago she again began to suffer from severe neuralgia, pain in both temples, worse in the left. This continued until her confinement in February last. Two months before her confinement the sight of the left eye began to get dim and became gradually worse, vision failing first at the inner part of the field and at the centre, the outer part remaining longest unaffected. Two days before the birth of the baby she became quite blind in this eye and so remained for one month. The vision then gradually improved until June 5th, when she again became suddenly blind in this eye. The day after the baby's birth she suddenly became blind in the right eye and continued so for a fortnight. Since then the vision of this eye has steadily improved. In the right eye the sight went "altogether" and suddenly, and did not, she thinks, fail more in one part than in another. Contemporaneously with the initial failure of vision, viz. two months before her confinement, she first noticed her face to be slightly swollen, especially her nose and eyelids, and her eyeballs to be prominent; these symptoms considerably increased until her confinement, since when they have not been quite so marked. The facial swelling has been sometimes greater, sometimes less, the patient remarking that she considered that the swelling varied directly with the pain. During the last four months she has been at times under treatment for ulcerated tongue. For the past six weeks she has objected to seeing anything of a blue colour; if she saw even a blue book she would ask that it might be removed. Confinement natural; child alive and healthy but not suckled by mother. No history of fits and no obtainable history of syphilis from husband or patient. Since confinement frequent neuralgia, but not so severe
as previously; no further vomiting. She says that she has been losing flesh for the last twelve months. Her sense of smell was never acute since her former illness, but it has recently failed entirely—before the failure of vision.

Note made on June 6th.—Patient very pale and puffy, considerable prominence of both eyeballs and swelling of upper part of face, very marked in the eyelids. No aneurismal bruit; movements of eyeballs normal. Bilateral symmetrical deformity of face. There is apparently some thickening and marked tenderness over the whole of both superior maxillary bones and over the ramus of the jaw on both sides, and especially near the zygomatic processes. Considerable thickening and increased breadth of nasal bridge, and air does not enter either nostril very freely, but she breathes with mouth closed. She does not breathe quite so freely through the right nostril as through the left. No discharge from nose and nothing visible anteriorly; complete anosmia. She cannot widely open her mouth on account of pain. Teeth normal; soft palate normal. Throat normal. The dorsum of the tongue has the appearance of having its epithelium denuded in circinate patches of varying size, these being situated at its anterior part and especially at the tip and sides. The denuded patches are paler than other parts, their edge definite and slightly red; the filiform papillae are prominent. Movements of eyeballs normal; pupils of medium size, right active to light, left barely so. T. n. in both. R. V. = fingers at 4 feet. L. V. = bare perception of light.

Ophthalmoscopic examination.—Media clear; atrophy of both optic discs; margin well defined, veins large and rather tortuous. There is still slight capillary redness at the inner part of each disc. No change in fundus elsewhere. Heart normal; urine sp. gr. 1007, acid, no trace of albumen (it has been repeatedly examined with similar result). No swelling of ankles. Patellar reflex normal. Ordered Ol. Morr. 5ij, twice daily. Mist: Quin., ver. c Pot. Iodid. gr. v, thrice daily.

June 13th.—Cachectic paleness more marked. Ordered
HYD. ĉ. Cretâ gr. j; Ferr. Redact. gr. iss, thrice daily. The dose of iodide of potass. to be 10 grs., thrice daily.

17th.—Has complained since last visit of almost constant neuralgic pain over both eyes. No sickness. R. pupil active but oscillatory. Counts fingers at 6 feet. L. pupil inactive, no perception of light.

24th.—Face very pale, and skin of face somewhat cold. Urine 1008, slight phosphates, no trace of albumen.

27th.—R. field of vision is limited on inner side and below almost quite up to fixation point; above and outwards field good. To continue mercurials and iodide of potass. To take also Tinct. Ferr. Perchlor. mX, thrice daily.

29th.—Dr. Felix Semon kindly examined the patient, and reports: "No tumour in naso-pharyngeal cavity. Slight deviation of septum nasi (congenital). Slightly increased vascularity of mucous membrane, but not sufficient to account for the loss of smell."


This evening (at the Society) the patient appears much more ill, and has now paralysis of the left portio dura (this was first observed by her friends yesterday morning). Neuralgia in both temples much increased. She says that the sight of the R. eye became dimmer four days ago, and again much worse this morning. There has been very severe pain in the left jaw and behind the left ear for the last four days.

Additional notes.—5th.—Paralysis of portio dura as before; the soft palate is not involved. Pupils equal, semi-dilated, inactive to light. R. V. = ? barest perception of light; L. no p. l.; no loss of sensation in area of fifth nerve, but rather slight hyperesthesia. There is less pain than on last examination, but her sight has become worse since then. Her intelligence remains perfectly good.
15th.—Condition unchanged. There is an apparent increase of bony hardness over both superior maxillary regions.


Aug. 5th.—Her general health has improved considerably lately, the pain is easier, and she is in better spirits. There is less paralysis of the left portio dura. Absolutely no p. l. in either eye. R. pupil $8\frac{1}{2}$ mm.; L. pupil $6\frac{1}{2}$ mm. The level of the two eyes is not quite the same, the right being directed slightly downwards. Taste appears to be less acute on the left side of the tongue anteriorly than on the right. To continue her medicines.

For the notes of this case I am largely indebted to Dr. Oswald Brown.

(July 3rd, 1885.)

8. Further observations on the condition of the optic nerves in intracranial disease.

By Walter Edmunds and J. B. Lawford.

Two years ago we made a communication to the Society on the nature of the changes the optic nerves undergo in those forms of intracranial disease in which they are affected.*

The conclusions we arrived at were, that the optic neuritis which occurs not unfrequently in the course of intracranial disease is due to the direct propagation of inflammation from the meninges at the base of the brain to the meninges of the optic nerve and to the nerve itself, thus causing the papillitis which manifests itself to the ophthalmoscope.

* 'Transactions,' vol. iii, p. 138.
DESCRIPTION OF PLATE VI.

Figs. 1 and 2 illustrate Messrs. Edmunds and Lawford's paper on the Condition of the Optic Nerves in Intra-cranial Disease (p. 184).

Fig. 1 is the peripheral part of transverse section of the posterior part of the left optic nerve of Florence S— (Case 10). It shows inflammation in the sheath-space (perineuritis), and also increase in number of nuclei in the nerve itself. Magnified 55 diameters.

Fig. 2 is a transverse section of optic nerve immediately behind eyeball (Case 5). It shows:—(1) Edema spaces round nerve-fibre bundles. In these spaces delicate bands may be seen with a high power. (2) Increase of nuclei throughout nerve. (3) Inflammatory effusion in the sheath-space. Magnified 120 diameters.

(From drawings by M. H. Lapidge.)
With the view of placing these conclusions on a broader basis, we have, with the kind permission of the physicians and surgeons to St. Thomas's Hospital, continued to observe such cases, and, where possible, have obtained and examined microscopically the optic nerves.

Altogether there are thirteen cases in addition to the twenty-two in the former paper. They are as follows:

Case 1.—George D—, æt. 9, admitted March 15th, 1885, discharged June 3rd, 1885. When playing in street run over by carriage and pair, the horses of which had bolted.

On admission.—Unconscious, bleeding from R. ear, eyelids bruised and swollen, subconjunctival haemorrhage of L. eye, R. mastoid process bruised, paralysis of R. facial nerve. On inquiry it is found that the patient has had "all his life" discharge from the ears.

March 18th.—Quite sensible. Ophthalmoscopically, much swelling of R. optic disc. L. eye cannot be examined owing to oedema of L. eyelids, and L. side of forehead.

28th.—Paralysis of R. external rectus muscle.

April 4th.—Abscess has formed behind R. ear and is opened. Deafness of R. ear. Purulent discharge from ears and nostrils.

17th.—Ophthalmoscopic examination (no atropine, and is very restless): R. optic disc pale, little if any swelling, but outlines indistinct; (?) small haemorrhage at apparent upper edge. Vessels very slightly tortuous. L. not seen. R. pupil oval, longest diameter vertical. R. pupil is much less brisk to light than L. and is always larger than L. Media of both eyes clear. "Sister" says his sight is bad, said to have had strabismus of R. eye.

May 18th.—Discharged nearly well.

Case 2.—C—, æt. 31. Fourteen days before admission met with an accident on his bicycle. He was stunned
but could be brought home same night by train. Gradually became worse till admission, June 24th, 1885.

On admission.—Delirious, especially at nights, when noisy and requires to have a porter in attendance. Pupils large but act to light. Gradually got better while in Hospital. Discharged nearly well July 10th, 1885.

Ophthalmoscopic examination, July 9th.—R. eye: Very marked white lines bordering large branches of both veins and arteries on the disc, and extending some distance along the main upward and downward divisions. Inner edge of disc indistinct, no swelling, no tortuosity of vessels. L. eye: Retina immediately around disc streaky, no other changes. Probably has had slight neuritis which is now passing off. No albuminuria.

August 3rd.—Comes up to be seen. Right eye: White lines bordering vessels while on disc are very marked, but beyond disc they can now only be traced on one large vein which runs upwards and outwards. Left eye normal.

September 2nd.—Loss of smell and taste.

Case 3.—Henry H—, aet. 9, admitted March 14th, 1885. Run over by carriage.

On admission.—Quite unconscious. Hæmorrhage from both ears. Only survived two hours in hospital; survived accident five and a half hours.


Microscopically.—Considerable increase in the number of nuclei in the optic nerve especially in proximal part.

Case 4.—Ada J—, aet. 7 months, admitted November 15th, 1884.

On admission.—Coma, vomiting, and fits.

November 22nd.—Examined ophthalmoscopically, "not a trace of optic neuritis."

Post-mortem.—Meningitis with much lymph over the whole of the base of brain, small cerebellar abscess.
Microscopically.—There is a decided increase of nuclei throughout the optic nerves, and too much material in sheath space. No sections of the optic disc itself were made, but the inflammatory signs in the nerve are well marked as far forward as its entrance into eyeball.

Case 5.—Alfred J—, æt. 11, admitted November 19th, 1884, died January 7th, 1885. Fifteen months before admission blow on head with piece of iron; the symptoms commenced next day and he had been gradually getting worse, with fits and paralysis till admission.

On admission.—Pain in the head, great drowsiness, occasional vomiting, and is nearly blind. Before death he became quite blind. He was only once examined ophthalmoscopically and the discs were said to be atrophied. He died January 8th, 1885.

Post-mortem.—Chronic cerebro-spinal meningitis. Both surfaces of the cerebellum were covered with a white material one to four millimetres thick, which under the microscope was seen to be organized lymph. There was also much thickening and opacity of the pia mater about the optic tracts and chiasma, also internal hydrocephalus. The specimen was exhibited at the Pathological Society by Dr. Hadden May 5th, 1885.

Microscopic examination of the right optic nerve. There is a very large increase of nuclei throughout the nerve bundles, more noticeable in them than in the trabeculae. A considerable amount of new material between the sheaths; the trabeculae are not appreciably thickened. (Vide Plate, VI, fig. 1). The O. D. is not swollen but there is an unusual number of nuclei in the nerve at its entrance into eye and in front of lamina cribrosa. There are small oedema spaces in transverse sections.

Case 6.—Louisa G—, æt. 6, admitted May 28th, 1885.

On admission.—Pain on movement of head, well-marked strabismus, calls out with pain in head.

May 30th.—Much worse. Examined ophthalmoscopically; no optic neuritis. 3 p. m. died.
Post-mortem.—Purulent meningitis at base. Meninges full of pus, chiasma surrounded by it. Suppuration in middle ear; the ear disease is probably the cause of the meningitis. It was a very acute case.

Microscopically.—Slight inflammation of optic nerve in posterior part. In sections near the eyeball, inflammatory signs are unevenly distributed, more evident at periphery of nerve than in central part. Papilla swollen and inflamed.

Case 7.—Alexis P—, aet. 14, admitted June 14th, died June 21st, 1884. Six years ago had scarlet fever. Discharge from left ear ever since. Eight days before admission ill with earache and vomiting.

On admission was delirious, had fever and rigors.

Ophthalmoscopic examination was difficult, owing to delirium, but optic neuritis was detected.

Post-mortem.—Disease in both temporal bones, with suppuration in mastoid cells, worse on R. side. In R. temporo-sphenoidal lobe an abscess; acute basal meningitis; acute spinal meningitis.

Microscopically.—R. O. N., great increase of nuclei and tissue in sheath space (perineuritis); throughout O. N. increase of nuclei in trabeculae and bundles; this is most marked in proximal part of O. N. where it is great. L. O. N. same as R.

Case 8.—Thomas T—, aet. 20, admitted 27th December, 1884, died 15th January, 1885. One month before admission pain in left side of occiput and round to left eye; seventeen days before admission dimness of left eye, and three days later total blindness of left eye.

On admission.—Pain and tenderness on left side in mastoid region; firm mass can be felt in neighbourhood of styloid process. Paresis of all divisions of the left fifth nerve. Impairment of movements of L. eye. No p. l. in left eye. No ophthalmoscopic changes in either eye.

January 6th.—Severe pain in head; unconscious. Temp. 103·1°.

13th.—Ophthalmoscopically.—Left disc normal; the
edges of the right disc are not so clearly defined as those of left; slight blurring, but nothing more (examined only by indirect method).

15th.—Died.

Post-mortem.—Growth starting from base of skull growing into nostrils.

Brain.—Meninges at convexity congested, at base well-marked meningitis, the inflammatory products surrounding the optic and other nerves. No growth could be found pressing on the left optic nerve. Aortic valves incompetent.

Microscopically.—There is very marked inflammation of the left optic nerve, especially in that part of it near the optic foramen; the inflammation is not uniform throughout the section, but most marked in a patch near the centre; there is some degeneration of nerve-fibres which have lost their sharpness of outline, and have a more granular appearance than normal.

In the anterior part of the nerve the inflammation is still distinct but less marked. The O. D. is swollen. In the R. O. N. in the posterior part of the nerve there is much inflammation of the fibres in sheath space, and some increase of nuclei in the nerve itself; the inflammation is also present in the anterior part of the nerve, but is not so well marked there.

Case 9.—Alec. —, âet. 14,* comes of delicate family.

On admission.—Cold abscess over L. parietal bone, weakness of R. arm; has had one fit recently.

Ophthalmoscopically, both discs slightly swollen and their margins blurred; the veins kinked, white lines along the vessels. No haemorrhages.

The abscess was incised under ether, at its base was found a circular piece of bone three quarters of an inch in diameter, almost completely detached. It was lifted up with the elevator and removed; the whole thickness of the skull was involved. Before leaving hospital the optic

* Previously published in 'Brain,' April, 1885, "Case of Perforating Tuberculosis of Skull," by W. Edmunds.
neuritis disappeared, and the opening in the skull partially closed.

Case 10.—Florence S—aet. 18, admitted April 28th, 1884. Taken ill five weeks before admission with vomiting and headache on right side of vertex. Gradually got worse with vomiting and drowsiness.

May 29th.—Commencing neuritis in left.

June 20th.—L. O. D. hazy, edge of disc cannot be seen; no tortuosity of veins; no haemorrhages; white lines along some of the arteries. R. O. D. much clearer than L. and probably normal. Pupils very large and do not contract. Died July 5th, 1884.

Post-mortem.—Convolutions flattened and sticky at surface. Base of brain has a tendency to adhere to skull but no lymph at base. In R. hemisphere of cerebellum tumour size of a large cherry.

Microscopically.—L. O. D. much swelling; increase of nuclei in sheath space throughout (perineuritis) (see Plate VI, fig. 2); marked increase of nuclei in proximal part of nerve, only slight in anterior part. R. O. D. swollen; O. N., some increase of nuclei in sheath space, proximal sections show increase of nuclei.

Case 11.—John Hart P—aet. 51, admitted July 21st, 1884. This case is published by Dr. Bristowe, under whose care he was, in 'Brain,' October, 1884, vol. vii, p. 327. Chief symptom was drowsiness, from which he could only be roused with difficulty.

July 24th.—Examined under atropine, both fundi normal. Died July 31st, 1884.

Post-mortem.—Tumour of corpus callosum, membranes at base slightly opaque but contain no lymph.

Microscopically.—R. O. D. swollen. R. O. N. increase of nuclei nerve and in sheath space both in anterior and posterior sections. L. O. N. not examined.

Case 12.—Ellen T—aet. 17, admitted November 19th, 1884. Four months before admission underwent ovariotomy. Since then has had "hysterical attacks."
On admission.—Great enlargement of liver, shortness of breath and cough. Died suddenly day of admission; not ophthalmoscoped.

Post-mortem.—Sarcomatous tumours in liver, lungs, pleura, heart, abdominal glands and brain, the tumour in the brain was in white substance under second right frontal convolution. A tendency to adhesion between brain and base of skull.

Microscopically.—R. O. N. inflamed. Effusion between sheaths not very large in amount. Increase of nuclei throughout nerve very markedly shown close behind lamina cribrosa, where there are some aggregations of them. Papilla much swollen. L. O. N. very like R., but signs generally better marked, and in some sections the changes are not uniformly distributed throughout the thickness of the O. N. L. O. D. same as R. O. D. R. O. tract (?) any changes; some small meningeal vessels close to tract are evidently inflamed.

Case 13.—Eliza T,—, æt. 10, admitted February 27th, 1885, with symptoms of ulcerative endocarditis.

March 10th.—Hemiplegia suddenly came on.

16th.—She died.

Post-mortem.—Heart.—In left auricle vegetations as large as a bean.

Spleen.—Several infarcts.

Kidneys.—Several infarcts.

Brain.—Left middle cerebral artery completely plugged, and softening of the whole corpus striatum, and caudate and lenticular nuclei on left side, and the convolutions of the island of Reil. There were several small infarcts, giving rise to haemorrhage in, and local inflammation of, the meninges; the left vertebral artery was also plugged. (The specimens were exhibited by Dr. Sharkey at the Pathological Society on March 17th, 1885.)

Microscopically.—L. optic disc markedly swollen. Trabeculae in nerve in both longitudinal and transverse sections show considerable increase of nuclei. Some
œdema spaces between nerve bundles and trabeculae. Well-marked inflammation of tissue in sheath space.

These thirteen cases may be classified as follows:

(i) Head injuries, three cases (1, 2, 3).
(ii) Meningitis, four cases (4, 5, 6, 7).
(iii) Diseases of skull, two cases (8, 9).
(iv) Tumour of cerebellum, one case (10).
(v) Tumour of cerebrum, two cases (11, 12).
(vi) Cerebral embolism with softening, one case (13). Taken as a whole these cases we consider confirm the conclusions arrived at in the former paper, but there are two cases (Nos. 4 and 6) which at first sight seem to tell strongly against these views; they are cases of meningitis, and in neither of them was any optic neuritis to be seen with the ophthalmoscope, although at the post-mortem in each case a large quantity of lymph was found over the chiasma and adjacent parts at the base of the brain. Microscopically, however, inflammation was found in the optic nerves, and in one case also slight swelling of the optic disc, though perhaps not enough to be recognisable with the ophthalmoscope in a delirious child.

The absence of well-marked optic neuritis with so much inflammation at the base of the brain is probably to be explained by the fact that such severe basal meningitis must be so rapidly fatal as not to allow time for the optic neuritis to reach the optic disc.

Case 1 is interesting as an instance of a fracture of the base of the skull occurring in a boy who was already the subject of chronic otorrhœa; optic neuritis appeared, but he recovered; possibly the occurrence of the optic neuritis may be in some way related to an irritable condition of the meninges caused by the ear disease, for optic neuritis is not very common in fractures of the skull.

In Case 5, too, the symptoms dated from a head injury, and it seems not improbable that this should be regarded as a case in which injury in a tuberculous subject started meningitis.
XI. FUNCTIONAL DISEASES.

1. Recurrent paralysis of third nerve in association with migraine.

By Simeon Snell.

Ethel T—first came among my out-patients at the Sheffield General Infirmary on November 30th, 1883, on account of drooping of the left eyelid. The mother was unable to attend with the little girl, but sent a letter graphically describing a good portion of the history of the case. I give a part of what she said: “Ethel was eight years old last month (October, 1883), and has had the failing in left eye since she was eighteen months old. It first came on when she was cutting her corner teeth; she was not born so. When her stomach is out of order the eyelid droops once in about six months, and remains down about six weeks. She has great pain in the eye whilst closing or until it has become quite closed. Then it is easier and (the eye) gradually opens, but it is never so strong or so straight as the right eye.” A note made at this time says, “There is complete ptosis, divergence of eyeball, and general third nerve paralysis; the pupil is not dilated and it acts well to atropine; the fundus oculi is normal; refraction hypermetropic, about 1.5 D.” The right eye is normal.

She was just over the severe part of one of her attacks. Improvement gradually took place.

On January 18th, 1884, a note states, “She is as well now, mother says, as ever she is; she fancies the eye is straighter than usual. The eyelid can be raised almost to the full, but there is a degree of divergence.”
The little patient seems to have suffered from several of these attacks during her life and to have recovered from them in a manner similar to that described.

In this instance the mother thought recovery was somewhat quicker than usual. Still further improvement took place after the above entry in January, and except for the persistent divergence the eyes did not appear so very dissimilar, yet the left eyelid never recovered to be quite like the other.

On June 6th, in the night, she was seized with sickness and cried very much with pain over the left eyebrow. The next day she ran about as usual, but in the night again complained of the pain, and the following day (8th) was on the sofa all day; all this time she was vomiting; everything she took returned. The eyelid commenced to droop this day, and by night the eye was completely closed. There is constipation when these attacks come on. No worms have at any time been noticed, nor has there been any trouble with the teeth since she cut them.

On the 10th she came to the infirmary; there was then complete ptosis, but it was possible to uncover a very little of the eye by means of the occipito-frontalis muscle; all the third nerve muscles were affected; pupil not dilated; divergence of eyeball. Recovery again soon commenced.

On July 18th, a note says that "the eye is opening." August 18th, "It is wide open, divergence still remains; the pupil is the same size as right one."

1885, January 20th.—She appeared to day with the eyelid partially drooped; it commenced yesterday.

21st.—I had desired word to be sent to me when an "attack" came on that I might see her in one, and today my colleague, Dr. Banham, physician to the Sheffield General Infirmary, was good enough to accompany me. We found the little girl lying on the couch, dreading to be disturbed, with flushed face, giddiness on attempting to stand, vomiting, and dirty-looking tongue; great pain in the head, confined chiefly to left side and especially in
the forehead and over eyebrow. There was complete ptosis, but by the aid of the occipito-frontalis muscle it was possible to raise the eyelid a very little; the pupil was a little larger than the right, and appeared to be inactive to light and accommodation; the other third nerve muscles were affected as in previous attacks.

23rd.—She came to the infirmary to-day. Sickness and head symptoms have passed off; the eyelid is still closed.

Recovery from the ocular paralysis took place as it had done on previous occasions.

On April 30th she is reported as well, and the condition of the left is as follows: the eye is open, and the levator palpebræ possesses a great amount of power, still the eye is not so widely open as the right; the appearance of the eye is distinctly divergent, but there is on effort fair power of convergence; the upward and downward movements are almost nil. The pupil, about the same size as its fellow, acts very little to light or accommodation, the difference between the two eyes being very distinct. The fundus oculi is normal, but there is a considerable degree of amblyopia; $V. + \frac{1}{2} = \frac{10}{20}$, and only J. 16 is read.

The family history is not without interest. Our patient is the second among four girls, no boys. There is a distinct neurotic history. The father suffers from "tic." The mother is now aged 34; she commenced to have headaches a year ago; they were at first slight, are becoming worse and quite recently has had a very distinct sick headache. The father's mother has suffered from "sick headaches" for very many years, a brother as well as a sister also suffer.

The case here related is an unusual one and the connection of the recurrent paralysis with the migraine has been observed too often to admit of doubt. Many curious ocular and other nerve troubles have been described in association with megrim. Ptosis is met with. Anstie, in his work on 'Neuralgia' (p. 92), quotes the complications
met with in an analysis of 128 cases of trigeminal neuralgia made by M. Notta.*

In this series "paralysis affected the motor oculi causing prolapse of the eyelid in six cases; in half of these there was also outward squint."

Two cases, very similar to the one there described, have been recorded † by Dr. Saundby, of Birmingham. In one the third nerve paralysis completely passed off, whilst in the other the "superior rectus remained permanently paralysed and the other recti enfeebled." One of Dr. Saundby's cases was a boy, aged 7, and it would seem that the attack described was a first one, and this was the case in which the ocular symptoms completely passed off. The other was a young woman aged 19 who had suffered from similar attacks from the age of twelve; here the left eye was affected; in the boy it was the right.

With respect to my own case the early age at which the migraine and the ocular symptoms commenced is worthy of note. The left eye has been the one affected and it will have been noticed that a permanently paralytic condition of the superior and inferior recti has resulted as well as enfeeblement of the power of the other muscles and the pupil. I think there is little doubt that the accommodation is affected at the time of the "attacks," but the degree of amblyopia present, and the unreliable answers of the little patient, make it difficult to state its condition as carefully as could be wished. The amblyopia would appear to depend only on exclusion from use. Dr. Saundby mentions amblyopia as present in his elder case.

I may mention also the following case to which Dr. Saundby has kindly drawn my attention, and of which he has become aware since the publication of his own cases. It is recorded by Dr. Buzzard in his 'Clinical Lectures on Diseases of the Nervous System,' ‡ and is as follows: "Anstie has drawn attention to the paralysis of the

* 'Archives Générales de Medicine,' 1854.
‡ Page 164.
culo-motor nerve which occasionally occurs in neuralgia of the fifth. We had a case in the hospital the other day. Jane H—, æt. 30, has been subject for years to paroxysms of facial tic, recurring about every fortnight and concentrated more or less definitely in the ophthalmic division of the fifth nerve.

"Within the last year or two, each attack has been followed by partial paralysis of the oculo-motor, the right eyeball being turned outwards and the eyelid dropped. This paralytic condition will last for a few days. We had the opportunity of observing one of these attacks during the patient's short stay in hospital."

(May 14th, 1885.)

2. Case of conjugate deviation of the eyes, down and to the right.

By W. Adams Frost.

Annie T—, æt. 25, single, dressmaker, attending the Royal London Ophthalmic Hospital under the care of Mr. Waren Tay, by whose permission the case is published.

Present condition.—Both eyes directed downwards and to the right. The L. appears to be on a slightly lower level than R. (a line drawn horizontally from lower border of right cornea cutting centre of left). Both upper eyelids droop, but not more than corresponds with the position of the eyes.

Patient professes to be unable to move the eyes in any direction except further downwards and to the right, and to be unable to raise the eyelids. On being told to raise the lids no action of the elevator muscles can be perceived, but a slight contraction of the occipito-frontalis takes place, which has no influence on the lids.

If one eye is covered the other can generally (but not
always) be made to follow the finger in all directions, and the eyelid then moves normally. The L. eye more frequently fails in this than the R.

On several occasions when the covered eye was exposed unexpectedly the eyes remained in the position in which they were at the moment, but usually they returned to the "down and right" position.

There is no diplopia. V., R. \( \frac{2}{5} \)°, L. \( \frac{2}{5} \)°. H. m. 1 D.

Fundus of each eye normal.

History.—Dates were given very differently on different occasions, but the following is the most consistent account. One afternoon three years ago, while singing, she suddenly fell down unconscious and remained so twenty minutes. Next day she vomited and went to bed, on the third day she again became unconscious, and remained so for eight or ten days; she then found that right leg dragged. In the autumn of last year she was in St. George's Hospital for symptoms of hip disease, and was examined under ether several times, but no disease found. She now wears a high-heeled boot, but there is no actual shortening. The foot is everted and dragged in walking; she can walk backwarks without falling. The date given for the appearance of the deviation of the eyes varies too much to have any value.

(December 11th, 1884.)
XII. INJURIES AND OPERATIONS.

1. Rupture of the eyeball to outer side in February, 1884, now showing rupture of choroid; recovery with useful vision.

By Walter H. Jessop.

Maud P—, æt. 8.
February 18th, 1884.—Fell and hit her eye against the bedpost. Taken into Paddington Green Children’s Hospital.

19th.—L. E.: lids much swollen, on outer side rupture of conjunctiva and V = p. I., some vitreous exuding; cornea normal; anterior chamber shallow and small hyphæma. Pupil dilated, oval, does not act to light. On iris above is small hemorrhage. No reflex. T. — 3. The eye washed well with carbolic lotion. Vitreous snipped off with scissors under chloroform, and conjunctiva sewn over wound. Treatment: ice compress.

21st.—Pain and slight redness of conjunctiva; anterior chamber deep, muddy; T. — 1; no reflex; iris greenish. Attack of iritis. (Hirudines ij, Hyd. e. Cretà gr. iss, b. d., Gutt. Atrop. gr. ij.)

March 6th.—Eye looks quieter; anterior chamber normal; pupil dilated, regular, slight red reflex below; T. — ½. Still great photophobia and lachrymation.

April 10th.—Going out. Eye quiet but irritable to light; pupil still dilated but regular; vitreous hazy and opacities; disc apparently healthy; T. n. Counts fingers.

P.S.—Eye looks same as other, but left pupil slightly oval; media clear. Disc, edges indistinct, old papillitis; to the inner side of disc about two discs breadth away is rupture of the choroid extending upwards along an artery;
to the outer side of disc, near y. s., is another rupture, more pigmented. V.: L. 3/6, J. 14 at 22 cm., not improved. V.: R. 6/6, J. 1 at 22 cm.

(May 14th, 1885.)

2. Evisceration of the globe, with artificial vitreous.

By P. H. Mules.

The opportunity afforded me of introducing the subject of "evisceration of the globe" as an alternative to enucleation is one of which I am glad to take advantage; the operation is devised for all cases requiring removal of the eyeball save those of malignant disease, and it may not be out of place if I touch upon the retrospective literature of the subject. The removal of certain portions of altered contents of an eyeball, such as bony fragments of the choroid, has long been practised by ophthalmic surgeons, but the emptying of the contents of the globe with the intent to prevent secondary infections is of very recent date, and has risen upon the newer, and to many of us more correct pathology of so-called "sympathetic disease." It is possible that some of you may have seen in 'Knapp's Archives' for June, 1884, a paper entitled, "Secondary Septic Ophthalmitis," in which after discussing the researches of Leber, Snellen, Brailey* and others, I ventured to prophesy that the outcome of these labours would be to establish the preventive treatment of sympathetic ophthalmia upon rational and intelligible bases, and being in perfect accord with these views, I hoped that a clearance of the interior of the globe under aseptic conditions before infecting particles of the specific uveitis had begun to travel, would not only prevent "sympathetic ophthalmitis," but leave a firm healthy support, or stump,

* Dr. Brailey appears to have since modified his views.
in every way superior to that ordinarily seen after enucleation, and I further hoped that whilst the sclera would tolerate a foreign body in the shape of an irregular bony choroid with the ciliary nerves in situ, so after the severance of such nervous connections, I might introduce a light hollow glass sphere or artificial vitreous into the cavity of the denuded sclera which, whilst preserving the shape of the globe and causing no irritation, would perfect the stump for the adaptation of an artificial eye.

Great difficulty was found in obtaining these little spheres of the original design, and this delayed my first operation of "evisceration" for some months, indeed till October of last year, 1884, since which time we have operated on nine cases, the last being in the present month. The man whom you have seen this evening wearing his artificial vitreous is an example of the method and opens possibilities for the treatment of this sad deformity which is pleasant to contemplate. During these operations I wrote to Mr. Nettleship asking for suggestions as to the prevention of certain complications that had occurred in some of the cases, and which were traced to defective asepticity. Carbolic solution 1 to 20 had been used and failed, and a solution of corrosive sublimate 1 to 1000 was prepared for the next case, when I learned that Professor Graefe, of Halle, had performed the operation of "evisceration" or, as he terms, it "exenteration," earlier in the year than I had done, his object, stated by himself, being to prevent purulent meningitis following the enucleation of a suppurating globe (happily in the practice of most surgeons, a very far off contingency). His experience, published in December last, extends to forty cases, and is favourable. I communicated with him, and he sent me a pamphlet reviewing the operation, whence it appears that the first recorded case is by Fröhlich in 1881, who accidentally emptied the globe of all its contents in an endeavour to extract a foreign body, and leaving the denuded sclera to the unaided efforts of nature, was so delighted at the patient’s recovery that he evolved the
term "excochleation," or scooping out, but carried his researches no further. Dr. Daubenton relates in the same year as Graefe's and my cases that Müller experimented on the cadaver, and on rabbits, but not on living human beings intending to extend his treatment as preventive of sympathetic disease.

This then, so far as we know, is the history of the operation! Fröhlich, one accidental case in 1881 to extract a foreign body. Müller, rabbits and cadaver in 1884. Graefe in 1884 for prevention of purulent meningitis. Mules in 1884 for prevention of sympathetic ophthalmitis, the last named differing from the others in the introduction of a hollow glass sphere, when feasible, into the cavity of the denuded sclera, the cosmetic importance of which, if it remains innocuous, is so obvious that it needs no further explanation. It must ever remain an extraordinary coincidence that Graefe, Müller, and I should have followed, in some respects, the same path almost at the same time without the slightest foreknowledge of the others' views.

The operation, though tedious, is performed as follows:

Operation.

1. Anaesthetise the patient.
2. Use hand spray and thoroughly cleanse and disinfect the appendages with 1 to 1000 corrosive sublimate solution.
3. Transfix and remove the front of the eye with a sharp knife at the corneo-scleral junction; it is better, I think, not to cut the conjunctiva.
4. Empty the contents of the globe in any way that is convenient, taking special care to remove the ciliary body and choroid, leaving only a clean white sclera.
5. With a thin india-rubber tube used syphonwise, run the sublimate solution into the eye the whole time the operation is being performed; to make sure I use the hand spray also, and continue this till the bleeding ceases or nearly does so.
6. Select the size of glass sphere best suited to the
EVISCERATION OF THE GLOBE.

1. EVISCERATION OF THE GLOBE.

203

3. EVISCERATION OF THE GLOBE.

4. EVISCERATION OF THE GLOBE.

5. EVISCERATION OF THE GLOBE.

6. EVISCERATION OF THE GLOBE.

7. EVISCERATION OF THE GLOBE.

8. EVISCERATION OF THE GLOBE.

9. EVISCERATION OF THE GLOBE.

case. Slit the sclera vertically until the glass sphere will with difficulty enter the cavity. Understand this difficulty only refers to getting the globe in; when inside it should fit so that the sclera unites easily over it and without leaving any awkward angles.

7. Sew up the sclera along the cut edge with prepared catgut, taking care to obtain good apposition.

8. Spread a layer of finely-powdered iodiform over the whole conjunctiva, and dress with wood wool in a double layer of Lister's gauze.

9. Keep patient, as a precautionary measure, in bed for three days, and dress all the time under spray.

Should you succeed in keeping the wound aseptic, the reaction is comparatively trivial. The man you have seen had some pain, but quite bearable, for two days. If suppuration ensues, the pain and distress is severe, the orbit is infiltrated, and the sclera in my fourth case partly sloughed away. I cannot lay too much stress upon perfect asepticity, and the operation should never be performed without full precautions for its attainment; in any case it is wise to warn the patient that there may be some orbital pain for two or three days.

I possess full notes of these cases, with which I will not now trouble you. Of the nine, the first six gave me some anxiety, for they all suppurated more or less, though finally, with one exception, doing well; these six were performed, as I have previously mentioned, under the protection of carbolised solution. The last three have been everything that could be desired under the protection of "corrosive sublimate" solution. Fearing that the glass sphere caused the irritation, I removed it from the second, third, fifth, and sixth. Now I know that it did not do so I proceed with perfect confidence to complete the operation as originally designed. The man you have see this evening was operated upon four weeks ago by Dr. Griffith, our assistant surgeon, for me during my absence from home, and is an example of the "completed operation."
I would point out that I am fully aware of the objections that can be urged against an untried operation. By untried, I mean any operation that has not stood the test of time, so I invite your co-operation that we may establish or condemn this treatment. The question of breakage need not enter into our calculations.

Any direct violence that would rupture a glass globe embedded in soft and yielding tissues would rupture the eye in its normal state, but the wearer should be warned, and if such an accident occur, enucleation is imperative. I do not think removal of the fragments should be attempted.

In its relation to the pathology of "sympathetic ophthalmitis," the case before you claims an interest specially its own, and must, I think, help to substantiate the accuracy of the deductions drawn by the observers before mentioned. The wound causing the loss of the eye was corneal; extensive hypopion being present, corneal section was performed, irido-choroiditis supervened, and the sound eye assumed all the characters of "sympathetic irritation." You can judge for yourselves the present condition of the sound eye and the eviscerated globe. A comparison of the operation of enucleation and evisceration leaves a large balance in favour of evisceration. Evisceration appears less formidable to the patient. The normal relation of parts outside the sclera are not disturbed, and the tendency to the formation of cicatrical bands is avoided. Add to these the certainty of fair movement, even without the artificial vitreous and the probability that it will be undertaken at an early date as a preventive treatment, and we have enough to justify the assumption that evisceration will take its place amongst the important operations of the eye. That we shall not hear of any mischances is too much to hope, but I can confidently assert, from the extended experience of Dr. Graefe and my own more limited, that if the precautions laid down in this paper are adopted, there is no real cause for apprehension. In conclusion, I would emphasise that
the artificial vitreous is no part of the operation of evisceration, but is designed solely in the interest of our cosmetic perceptions.

*(Living specimen. March 12th, 1885.)*

Mr. Adams Frost thought that the operation of "evisceration" was somewhat analogous to that of abscission, which had been abandoned by many on account of its risks. Accidental evacuation of the contents of the globe, and the older cases in which that proceeding had been purposely adopted, were hardly comparable with the present proceeding of carefully cleaning the sclerotic with antiseptic precautions. The paper seemed to open two questions for discussion:—1. Is the operation of evisceration preferable to that of enucleation? 2. If so, is it desirable to introduce an artificial vitreous? Unless every particle of the uveal tract could be removed, the risk of sympathetic trouble would be great. Is it not very difficult to ensure the removal of the whole of the ciliary body from a cavity filled with a mixture of blood and antiseptic solution? As to the employment of the artificial vitreous, the risk of fracture of the glass sphere did not seem so problematical as Mr. Mules seemed to think, and fracture might easily cause laceration of the stumps of the ciliary nerves. The movements of the glass eye exhibited did not appear to be more free than those often obtained by the ordinary method (this remark applied to the eye, not to the stump, which had not then been seen). Altogether the proceeding did not seem to offer advantages sufficiently great to counterbalance the extra risks.

Mr. Higgens said that he had more than once seen evisceration performed accidentally during an operation. He asked what evidence the author had that the operation had any advantage whatever over enucleation in the prevention of sympathetic ophthalmitis.

Mr. Hartridge asked Dr. Mules if he had tried any material other than glass of which to make these small
globes that were to take the place of the vitreous. Would not celluloid answer the purpose as well and be in less danger of breakage?

Mr. Juler desired to know what form of instrument was employed for the purpose of clearing the sclerotic. He thought some modification of Volkmann’s scoop might be useful.

Note.—October, 1885. Up to the present time all the cases permanently fitted with the artificial vitreous are perfectly well. One case only that left the hospital with the scleral wound slightly gaping has had trouble from decomposition of retained secretion. It appears necessary to insure perfect healing of the wound at the outset. I would suggest that the scleral wound be sewn up vertically, and that over it the conjunctiva be drawn and united at right angles.

The small glass spheres can be obtained from Messrs. Armstrong Brothers, opticians, Deansgate, Manchester.
XIII. CONGENITAL DEFECTS.


By W. Lang.

The mother of the elder patient says she was severely frightened when pregnant; and after the birth of the child the eyes were noticed to be defective.

She has now lateral nystagmus; absence of iris in each eye, and a lamellar cataract in R. The left cataract was operated on, but not successfully.

This patient has been married six years, and has had three children. The eldest, a girl, is living, has no defect, but the second and third children, a boy and girl, have had similar defects in each eye.

The second child died, and the youngest is the present patient. It has lateral nystagmus, absence of iris, and striae in each lens.

There is no history of similar defects in any other member of the family.

(Living specimen. July 3rd, 1885.)

2. Peculiarly shaped eyeball.

By R. Marcus Gunn.

Girl aged 8. Right eye. On examination by the direct method the inner side of the optic disc is seen with a + 3 D. lens, showing in this part a hypermetropia of that amount. The same condition holds good for the
entire inner part of the globe. Just outside the optic disc, however, a — glass becomes necessary so as to get accurate details, and the yellow spot shows a myopia of 4 D. Thus the difference in distance from the cornea between the inner part of the disc and the yellow spot respectively is 7·8 D., or about 2·1 mm. There is no pathological change in either fundus.  

(Living specimen. October 9th, 1884.)
XIV. INSTRUMENTS.

1. *Ophthalmic models for teaching.*

By Priestley Smith.

The models formed a series of simple construction designed and used by himself for class demonstration.

1. A large diagram of the eye in section, the figure white upon a ground of black millboard. The one half slides upon the other, so as to shorten or lengthen the globe in imitation of hypermetropia and myopia.

2. A spherical wooden model, with glass cornea, iris, a moveable lens to simulate accommodative changes, and a ground glass retina. The globe can be shortened or lengthened. It exhibits the condition of the retinal picture in emmetropia, hypermetropia, and myopia, with and without accommodation, with and without glasses.

3. A lens and moveable painted retina, mounted on a block, used for the same purposes as the foregoing, and to demonstrate the "fundus-image" test, &c., to a class without artificial illumination.

4. A simple artificial eye for beginners with the ophthalmoscope. A cylindrical block of wood, hollowed, carrying a small lens with the iris painted on it in front, a moveable card-retina behind. It may be hung upon the wall of the dark room. Various degrees of refraction, and right or left eye, can be imitated.

5. A "cornea" of india rubber, cut from the side of a large ball. Pressed upon at the edges it assumes unequal curvatures in different meridians and represents corneal astigmatism. A large wooden "cylinder" to explain the optical correction of the same.
6. The mechanism of the accommodation. A strip of thin elastic steel bent to the proper shape represents the outline of the lens in transverse section. Silk ribands attached, one on each side, to the equator of the lens, and ending externally in spiral steel springs, represent the suspensory ligament kept in a state of tension by the elasticity of the choroid. Other ribands attached to these anteriorly represent the ciliary muscles. When the latter are pulled the traction of the suspensory ligament upon the lens is lessened, and the lens becomes more convex by its own elasticity.

7. An apparatus for demonstrating operations with pigs' eyes. Two short cylindrical pillars cupped at the top receive the eyes; over these slide tubular caps, open at the top, which hold the eyes in place and bring them to the necessary state of tension. A gutta-percha mask with apertures at the eyes is laid over the whole.

8. A model illustrating the causation of diplopia and its correction by prisms. Two cardboard cylinders, the eyes, rotate about their vertical axes; a convex lens in the side of each represents the refracting media; a piece of thin paper stretched across an opening at the opposite side, the retina. In the centre of each retina a small area, rendered more translucent by a drop of paraffin, represents the yellow spot; the portion of the image falling on it is clear and brilliant. The fixation object is a lighted candle. The model demonstrates the relation which exists in all ocular deviations between the deviation of the cornea and the apparent deviation of the image.*

(December 11th, 1884.)

* These models were left, by request of members of the Society, with Messrs. Pickard and Curry, who have made others like them.

By Arthur H. Benson.

Cocain has been for some time in use amongst laryngologists as a local anaesthetic, but it is quite recently that its value in ophthalmic practice has been set forth.

Dr. Koller, of Vienna, appears to have first discovered its qualities, and at the Ophthalmological Congress at Heidelberg held last month a paper was read by Brettaufer extolling its value to the ophthalmologist.

My friend and colleague Dr. Story heard this paper (and saw the use of the drug demonstrated) at the Congress, and wrote informing me of the facts stated therein as follows:—"A drop or two of a 2 per cent. solution of hydrochlorate of cocain in water inserted into the conjunctival sac produces complete corneal and conjunctival insensibility in less than ten minutes, and the effect lasts about ten minutes more. You can scratch the cornea, rub, scrape, cut it, use a fixation forceps and speculum, without causing pain. The sensibility of the iris and deeper parts, however, persists. It slightly dilates the pupil and paralyses accommodation, but eserine and light act as usual." I at once ordered some of the hydrochlorate of cocain, which was obtained for me from Messrs. Morson and Co., of London, and had a 2 per cent. and a 4 per cent. solution made with water.

I introduced two drops of the 2 per cent. solution into the conjunctival sac of a man affected with cataract. It did not seem to pain him at all. After a few minutes I
tested the corneal sensibility in each eye, and found only a very slight difference of corneal and conjunctival reflex sensibility, and no subjective appreciation of difference between the two eyes.

The sensibility of the cornea and conjunctiva was tested in this and all subsequent cases by means of a fine point of cotton-wool or of soft paper, which was applied alternately to the affected and the non-affected eye so as to compare the impressions produced in each. Wishing to observe the subjective sensations produced I introduced a drop of the 4 per cent. solution into my left conjunctival cul-de-sac. It at once produced a sensation of smarting very similar to that caused by soap. This passed off in about half a minute. In one minute there was well-marked anaesthesia of the cornea and conjunctiva, but even then touching them gave some pain. Soon a sensation of tightness or drooping of the lids was experienced as if it required some effort to keep them open. In about ten minutes this had nearly passed off, and with it most of the anaesthesia. In seven minutes the pupil began to dilate, and there was very slight paresis of accommodation, and corneal and conjunctival sensibility had to a considerable extent returned. In ten minutes the pupil had reached its maximum dilatation (about one third), and anaesthesia had wholly disappeared, but the corneal reflex was perhaps less sudden. In fifteen minutes the paresis of accommodation had given way to spasm, and the pupil in five minutes more began to diminish in size. In half an hour it was smaller than the normal pupil, and the contraction reached its maximum in about three quarters of an hour. The spasm of the ciliary muscle apparently reached its maximum before this, for in a quarter of an hour more, that is, within one hour from the instillation of the cocain, the accommodation had undergone paresis and spasm, and had returned to its original normal standard, whilst the contraction of the pupil lasted in some degree for a considerably longer time.
A second drop of the same solution, introduced an hour and a quarter after the first, produced exactly the same smarting and anaesthesia, but did not at any time dilate the pupil or paralyse it. The pupil, which had not yet fully recovered from the myosis produced by the previous drop in a few minutes, became more myotic without previous dilatation, and the spasm of it increased. These symptoms passed off in about the same time as the previous series.

At the same time, I applied a single drop of the 4 per cent. solution to my friend Dr. Nevill's eye, and almost an identical series of phenomena occurred. In him the dilatation of the pupil and paresis of A. were very slight, but having naturally very large pupils, the subsequent contraction was most striking, the pupil becoming little more than half the diameter of the normal one, whilst the spasm of accommodation was also well marked for half an hour or so.

The pupils at all stages acted well to light.

I have also applied the alkaloid to the eye of Dr. Keane, house surgeon at St. Mark's Hospital, and to several of the students attending the clinique, and in each case some temporary anaesthesia was produced, with subsequent dilatation of the pupil and paresis of A., followed by myosis and spasm.

I have, moreover, also applied the cocain to about twenty other cases, including several normal eyes, also a case of irido-choroiditis, sclerotico-choroiditis anterior, ulcer of the cornea, strabismus, traumatic cataract, &c., and have extracted cataract, done iridectomy, and performed several minor operations on the eye whilst under its influence, but in each case without producing a degree of anaesthesia which seemed likely to prove of much practical use in operations.

From what I have seen of the hydrochlorate of cocain I fear it would not supply the desired anaesthetic, but if it is to be used it must be applied very shortly before the operation in which it is intended to produce the anaes-
anesthesia, for the greatest anaesthetic effect occurs within a few minutes of its instillation, and passes off with great rapidity.

In all the operations which I performed with its aid the fixing of the speculum and forceps and the use of the knife or scissors caused apparently just as much pain as is usually caused, and in the case of the cataract extraction (I had previously operated on the patient’s other cataract without cocaine) he said that he could find no difference in his sensations on each occasion.

It seemed, therefore, that whilst for the slighter operations, requiring no fixation, &c., cocaine might be of use (for it did undoubtedly cause some decided anaesthesia), still in the more formidable and painful proceedings it gave hardly any appreciable relief.

The very remarkable oscillation in the condition of the iris and ciliary body which I noticed in myself and in Dr. Neville and the others, had not, as far as I was aware, been observed by others. It was a phenomenon so peculiar that it could scarcely have escaped observation, and it was so well marked in us as to challenge attention.

I was not aware of any other drug which produced at all the same series of actions upon the eye. I therefore undertook a series of further observations and found that the myosis and spasm of accommodation which occurred after a very temporary dilatation of the pupils and paresis was probably produced by the presence of a trace of eserine in the specimen of cocaine with which those experiments were conducted, for subsequently I obtained, through the kindness of my friend Mr. Swanzey, a pure specimen of cocaine which he had procured from Merck, of Darmstadt. Experimenting with this, I found that no contraction of the pupil or spasm of accommodation followed, but that the pupil remained partially dilated for several hours. If, however, the very slightest trace of eserine were added, then the dilatation soon gave place to contraction, and the paresis to spasm. I therefore had a fresh solution prepared from the original salt obtained
and found that it too produced no contraction. It seems therefore evident that the first solution was contaminated.

In experimenting further on myself I found that the anaesthesia produced reached its maximum in two or three minutes and was extremely local. I could, by applying a small drop to the inferior *cul-de-sac*, produce marked anaesthesia of the lower half of my cornea and conjunctiva whilst the upper half retained perfect sensation, but that this result was not obtained if I winked my eyelid after introducing the drop.

I further found that the degree of anaesthesia depended, within limits, upon the amount of the drug employed. Three or four applications of a 2 per cent. solution at intervals of half a minute produced such complete anaesthesia in most cases that I was able to use a fixation forceps and speculum, make the corneal incision for iridectomy, cataract extraction, discission, &c., without the patient complaining of the pain. And the removal of foreign bodies from the cornea has been by the aid of cocaine rendered easy and painless.

In most cases the sensibility of the iris seemed to be slightly, if at all, diminished. Probably, by using a stronger solution and for a longer time the effect might be so increased as to penetrate to the deeper structures. After applying a drop to my eye, I always experienced within from half an hour to an hour a peculiar discomfort which was not relieved by closing the eyelids: this gave rise to a slight frontal or supra-orbital ache of a very dull indefinite kind. On testing the tension I found that it was definitely raised in the drugged eye. Testing the tension in a number of normal eyes treated with cocaine, I found that in nearly all cases (only one or two excepted) the tension rose perceptibly when the dilatation of the pupil took place, usually reaching to + 1, and diminished and disappeared before the pupil recovered its normal size. This result I obtained more certainly and manifestly when experimenting with Merck’s cocaine. The effect on the
tension of pathological eyes I have not attempted to determine.

It seemed reasonable to expect that photophobia should be benefited (at least, if due to irritation of the corneal or conjunctival nerves) by applications of the drug. I therefore applied it in cases of interstitial keratitis (specific), strumous keratitis, ulceration of cornea, and acute catarrhal conjunctivitis, and with the expected good result. In some cases I was enabled to examine the cornea fully and without apparent pain to the child, where I had utterly failed on several previous occasions to obtain a view of the parts. In other cases the patients volunteered the information that the drops took away the pain caused by the light.

I applied cocain to the tongue and lips but failed to obtained marked results, only a slight dulness of sensation being produced, but my experiments were not extensive. I also used it in removing a large polypus growing from the middle ear, and with apparently good results, the pain was not entirely destroyed but mitigated.

In no case I have observed any imperfection in the healing of wounds where cocain had been used before the operation.

In several cases where there was much existing inflammation in the cornea or conjunctiva the cocain produced much less effect than on the healthy tissues; in one case notably, though it was applied abundantly, no appreciable anaesthesia seemed to be produced. This was in a case of traumatic cataract and iritis on which I performed iridectomy whilst considerable zonular vascularity remained. In most cases, however, the drug gives to the cornea and conjunctiva total immunity from pain, but to obtain this result it is necessary to apply it freely to every part which it is desired to affect, and this must be done very shortly before the operation is undertaken.

In cocain, I think we possess one of the most valuable drugs in the ophthalmologist's pharmacopœia.

(October 9th, and December 11th, 1884.)
2. On the use of cocaine in ophthalmic practice.

By E. Nettleship.

It seems probable that cocaine may before long be employed with four different objects in ophthalmic practice; as an anaesthetic in operations, as a means of relieving intolerance of light, as a mydriatic for ophthalmoscopic examination, and as a means of diminishing congestion of the eyeball. Like most other observers I have so far been occupied chiefly with its use as a local anaesthetic in operations upon the eye, and I shall not have much to say on the other three heads.

I first heard of cocaine from Mr. Gunn at Moorfields very early in October, Dr. Pinto, of Heidelberg, having brought the news of Dr. Koller's discovery direct from the meeting of the Heidelberg Society. I first operated on eyes under its influence on October 10th:—a woman aged 72 had preliminary iridectomy performed in the L. eye followed immediately by extraction of cataract with iridectomy in the R.; a man aged 65, with central corneal opacities had double iridectomy for artificial pupil; and a woman aged 51 had iridectomy after extraction in the L. followed immediately by extraction with iridectomy in the R. The two first patients behaved perfectly well although they felt during the iridectomy. The third was nervous, unruly, and deaf, and although she felt neither the speculum, fixation forceps nor knife, she winced very much during the iridectomy and lost a good deal of fluid vitreous from the eye in which the secondary iridectomy was being made. Since then I have operated on more than seventy eyes under cocaine and I have no doubt that it will to a large extent take the place of the general anaesthetics in ophthalmic operations.

I think its chief disadvantages are that it only acts for a short time and that it does not destroy sensation in the iris. Full anaesthesia of the conjunctiva and cornea comes on so quickly after the application, and begins to go off so
soon, that we have either to time the case accurately lest we miss the brief period of maximum effect, or to make repeated applications every five minutes; and if several patients have to be operated upon still more care is needed that each shall be exactly ready when his turn comes. The second objection to cocain, that it does not destroy the sensibility of the iris, is not often a serious one; if, just before seizing the iris with the forceps the operator warns the patient that a little pain will be felt, usually no trouble is experienced, but if no warning be given the patient is apt to start or wince much more at this stage than he would have done had the previous stages of the operation also been felt.

It is of course possible that there may be other drawbacks. I have thought that in one or two nervous patients who behaved perfectly well during the operation there was more pain than usual an hour or two afterwards, and in two cases of cataract extraction I suspected that some bleeding which took place into the anterior chamber a few days after the operation might be the result of vascular dilatation secondary to the contraction which is one of the normal effects of cocain. But we need further experience on these and other points. I suspected, after some of the earlier trials, that the anaesthetic action was less certain and complete in old than in young persons, but later experience has shown that no such difference exists.

Returning for a moment to the iris, I have tried in several cases the effect of applying cocain every five minutes for an hour, and in others for half an hour, immediately before operating, but in none has the iris been anaesthetic nor could I in any case feel sure that its sensibility was even lowered; but I have not made any comparative experiments on the two eyes of the same patient, and that alone could settle this point.

In the earlier operations I used a watery solution containing 2 per cent. of hydrochlorate of cocain (about 9 grs. to fl. 3j), and in a few cases a 4 per cent. (18 grs. to fl. 3j). Mr. Martindale then made for me solutions in vaseline and
in castor-oil each containing 2.5 per cent., and Messrs. Savory and Moore made me gelatine discs each of which contains $\frac{1}{200}$ gr. of the alkaloid.*

I think that, on the whole, the gelatine discs are the most convenient for operations on the eyeball, although all the preparations named are quite efficient; of the watery solutions the 2 per cent. solution seems as good as the 4 per cent. I have not tried solutions weaker than 2 per cent., but it would be quite worth while to do this and also to have discs containing less than $\frac{1}{200}$ gr. Some little inconvenience arises from the gelatine of the discs sticking about the lashes if the discs be used repeatedly with the idea, e.g. of destroying sensibility in the iris, but the gelatine may be washed away just before commencing the operation. Latterly, however, I have relinquished the attempt to influence the sensibility of the iris and have used only two or three discs, one being inserted beneath the upper lid about three minutes before operating, and the others from two to three minutes later, or about one minute before beginning to operate.

It is best to try the conjunctiva with the fixation forceps before inserting the speculum, and this trial should be made in two or three places, for unless the preparation used has spread itself equally, sensation is sometimes found to remain in one part or another of the conjunctiva. Simply touching the eyeball with a probe or finger is not a sufficient test of complete anaesthesia. The parts at the free border of the eyelids seem not to get completely numbed, and the slight sensation due to the contact of the speculum here no doubt accounts for the continuance of reflex action of the orbicularis palpebrarum in certain patients, especially where there is a photophobic habit. Even when the superficial parts are quite anaesthetic any sudden movements of the eye or eye-

* Owing to the salt of cocain being very hygroscopic it is impossible to make the discs much stronger than the above, as they cannot be dried. Even those containing $\frac{1}{200}$ th of a grain are not perfectly dry, and often stick together a little.
lids are perceptible to the patient and are apt to make him start or wince. Hence it is very necessary to finish all large sections very gradually; the fixation forceps should also be loosened without the slightest jerk, and the speculum removed quietly. These various little points, although they look troublesome on paper, are readily attended to in practice.

I have not hitherto had good success with the palpebral conjunctiva. In several cases of granular conjunctivitis the ointment or the castor-oil solution has been freely smeared over the surface of the everted lid before using the actual cauterery. Some parts of the mucous membrane have been rendered anaesthetic, but others have seemed to feel as much as ever. The part nearest the free border is naturally much more sensitive than the rest, but this does not explain the result mentioned, for complete anaesthesia of this part has been produced, whilst portions further back and normally less sensitive have remained sensitive. It is no doubt necessary that the cocain should come into contact with every part of the palpebral conjunctiva that is to be deadened, and this does not occur when the greasy preparations are used, nor can it be depended on even if discs are inserted or watery solutions dropped into the sac. Probably the best plan will be to paint the whole surface of the everted lid once with a much stronger solution, say 20 per cent., a method already employed in operations on the larynx by Dr. Semon and others.

The following is a short statement of the operations which I have performed upon eyes under the influence of cocain, including the six alluded to at the beginning of the paper.

Extraction of senile cataract 18 (eyes); preliminary iridectomy had been done in 5 of these, in the remaining 13 an ordinary upward extraction with iridectomy was performed. 17 of the 18 did well, only 1 having troublesome iritis. One eye was lost by suppuration, the patient, a woman aged 68, was gouty and in
bad health, but her urine free from albumen; she was nervous, but the operation (upward extraction with iridectomy) was perfectly satisfactory. Although I had had no case of suppuration after extraction at St. Thomas's for the previous eighteen months, there seems no reason for attributing the disaster in the present case to the influence of the cocain. In one other case already mentioned, a woman aged 51 lost fluid vitreous during iridectomy for closed pupil after extraction in one eye, though a satisfactory extraction with iridectomy was immediately afterwards done in the other eye. Without cocain, chloroform or ether would have been given to about twelve of these eighteen patients.

Removal of soft cataract by curette, through small incision without iridectomy, 3 eyes; all behaved well, and the eyes progressed well; 1 had some iritis.

Extraction of floating opaque lens, 1 eye. The lens was perfectly free, and lay in the lower part of the vitreous chamber when the patient lay on his back, and often when he simply sat or stood upright, but came quite into the a. c. if he stooped forward. The patient, a man aged 33, was very deaf, and the eye was not fully anaesthetie. The lens having been brought into the a. c. by making the man bend his head forwards, was fixed with a needle and removed by a spoon through an upward incision, the patient sitting in a chair. He became unruly, a large quantity of perfectly watery "vitreous" was lost and the eye collapsed and had to be removed a few days later.

Operations upon the iris, 20 eyes, in fifteen patients, as follows:—Preliminary iridectomy 3 eyes; two behaved quite well, one, an old man of 70 lay perfectly quiet until the iris was pinched when he became suddenly so uncontrollable that chloroform had to be administered; this was the only case of the whole number in which ether or chloroform had to be given.

Simple glaucoma, with good a. c., 2 eyes of the same patient; no difficulty.
For closed pupil after extraction 1 eye; unruly and lost vitreous (case already mentioned).

For central corneal opacities, 6 eyes in three patients; two behaved perfectly, the third, a very fidgety young man still suffering from some photophobia after chronic corneal ulcers, gave some trouble, but the operations were properly finished; it would have been impossible for me to operate on this man without ether or chloroform, unless cocain had been used.

For synechiae or closed pupil after iritis, 3 eyes in two patients; both behaved well.

Removal of iris prolapsed through recent wound of cornea, (iris freely drawn out and cut off), 2 eyes; one was a boy, very frightened and fidgety, and ether would certainly have been necessary but for the cocain.

Iridotomy after extraction of cataract 2 eyes.

Separation of anterior synechia by cutting needle after operation for conical cornea, 1 eye. The apex of the cone had been excised, under cocain, without the least sensation three weeks before.—Without cocain ether or chloroform would have been given to nearly all of these fifteen patients.

Discission, 8 eyes, 5 of them for capsule or membrane after removal of lens, 3 for soft cataract; the youngest was aged eight. Probably all of these would have been done without anaesthesia even had cocain not been available.

Removal of opaque capsule with forceps, 1 eye; traction on the iris when the membrane was pulled out was felt.

Scraping serpiginous ulcers of cornea, 3 eyes; these would probably have been done without anaesthesia in the absence of cocain, but with a good deal of pain from the fixation of the congested conjunctiva; as it was the operation was scarcely felt.

Tenotomy, 18 eyes; internal rectus 16, external rectus 2; Liebreich's operation was chosen in 7, the Moorfields operation in the remaining 11; one
child was aged 7½, another 8, and many of the others were between 10 and 12. There is very seldom any difficulty even in the longer and more violent procedure of Liebreich if the hook and scissors be handled gently and steadily without sudden movements. Ether would have been given in about half these cases without the aid of cocain. Two or three other squints were operated on under cocain, but no note was made. There is always some sensation, occasionally decided pain, when the tendon is put on the stretch by the hook and scissors, but it has, I think, never been enough to make the patient cry, even when quite a child. Repeated applications have not seemed to affect this sensibility of the fascia and muscle.

Cocain has also been used almost daily for some weeks in cases of foreign body on the cornea, and with complete success.

I have not yet excised the globe or operated for glaucoma (with the exception of the simple case already mentioned) under cocain. The great importance of removing the iris freely and deliberately in iridectomy for glaucoma would seem to militate against anything like a general abandonment of chloroform in favour of cocain for this particular operation.

In respect to the second use of cocain, the relief of photophobia, I have found, in common with many other observers, that very marked relief is generally given within a few minutes of applying the solution in cases of corneal ulcer. I believe it will be found, however, that a larger quantity is required to produce complete anaesthesia of the conjunctiva and sclerotic, if not of the cornea, in severely congested eyes (from all causes) than in health, though in a few trials that I have made there have been considerable differences. In a boy aged 11, with small corneal ulcer and severe ciliary congestion and photophobia, a single application of 2·5 per cent. ointment gave complete anaesthesia of cornea and conjunctiva in three minutes; four minutes later and after a second application there was scarcely any photophobia and the p. was beginning
to dilate; a third piece of ointment was now put in, and half an hour later the p. was wide (9 mm.), and the eye free from photophobia, but sensibility had returned fully. This eye reacted quite normally to cocaine.

In a little boy aged 8, with a moderate attack of interstitial keratitis and ciliary congestion, one cocaine gelatine disc had produced only partial anaesthesia in three minutes, but nine minutes after a second application (two discs), (or twelve minutes after the first disc) anaesthesia was complete even to deep pressure on both ciliary region and cornea.

In a lad aged 19, with ulcer and moderate photophobia in L. eye, a single disc to each eye gave complete anaesthesia in three minutes in the sound eye, but not quite so complete in the inflamed eye.

In a man aged 40, with relapsing iritis of L., and liability to attacks of severe pain; a single disc to the sound eye caused perfect anaesthesia of cornea and conjunctiva in six minutes or less; but a second and even a third disc to the inflamed eye, though destroying all feeling in the cornea, still left the conjunctiva (or sclera) only partly anaesthetic, deep pressure being decidedly disagreeable.

In a girl aged 13, with severe catarrho-pustular ophthalmia of L. eye, the vaseline ointment, 2.5 per cent., gave perfect anaesthesia of cornea in four minutes, and the ocular conjunctiva appeared to be anaesthetic if very lightly touched; but even a second application did not much alter this condition, anything more than the most superficial pressure still causing pain.

In a case of troublesome photophobia and constant blinking due apparently to overuse of the eyes, in an engine-fitter, and uncomplicated by any inflammation or congestion, cocaine ointment gave immediate relief for a short time.

The relief of photophobia by cocaine shows how important is hyperesthesia of the fifth nerve as a factor in what is commonly spoken of as "intolerance of light."

As a mydriatic cocaine will probably be used for ophthalm-
moscopic examination. But it will always be less convenient for this purpose than the atropine compounds, because a pupil dilated by cocaine still contracts a good deal when brightly lighted. Its very slight effect on Acc., however, is a great convenience.

We have yet to learn whether cocaine can produce contraction of the superficial blood-vessels of an inflamed eye, as it certainly does in most healthy eyes. I say "superficial" blood-vessels, not knowing whether any observations have been made as to its effects on the retinal circulation. I have tried repeated applications of the discs and ointment, and of watery solutions of 2 per cent., and even of 20 per cent. to several eyes, inflamed and congested in various ways, but without being able to satisfy myself, or others who were present, that the redness of the eyes was at all diminished, and this although the p. often became widely dilated.

The subject is so new that I have ventured to add to this paper a few observations as a contribution to our knowledge of the physiological actions of cocaine upon the human eye. These have been made partly on myself and friends, and partly upon patients, and have been made for me, to a great extent, by Dr. S. W. Sutton, Mr. W. G. Mackenzie, and Mr. Lawford.

In observations on ten healthy persons anæsthesia is noted to be complete (after a single disc had been used) in from 2.5 to 10 minutes; from observations on operation cases I should say that from three to five minutes is the usual time required for maximum effect. It remains complete for from three to about six minutes, then rapidly declines; but in many cases some slight insensibility remains for twenty to thirty minutes and sometimes longer. As the numbness passes off a slight feeling of coldness is sometimes experienced, whether from deprivation of arterial blood, or from a slight degree of hyperæsthesia due to a nervous reaction, has to be determined.

A decided retraction of the upper lid usually comes on in about three to five minutes, and lasts from twenty to
forty minutes, i.e. considerably longer than the anesthesia; the lower lid does not alter in position. I have not observed whether the natural descent of the upper lid with the eye in looking down is interfered with as it is in Graves's disease.

About the same time as this retraction of lid comes on the staring appearance of the eye is usually still further increased by a marked whitening of the conjunctiva and sclerotic; I am not sure how long this usually lasts, but certainly for half an hour.

The pupil seems sometimes to contract a little about three minutes after the application (Dr. Sutton notes this in two of six cases). It begins to dilate in from five to ten or twelve minutes, and reaches its maximum in from twelve to thirty-five minutes; the rate of dilatation varies, it may e.g. begin rather late and reach its maximum early. The dilatation is often, but not always, as great as that produced by homatropine and even by atropine; but as the reflex and associated actions of the pupil remain, to some degree at least, the cocain maximum will probably be found to depend very much upon the illumination; very likely also it will vary with the condition of the heart's action and with the state of the peripheral arteries. The mydriasis begins to lessen very soon, but usually takes nearly twenty-four hours to pass off entirely.

The accommodation is scarcely influenced by a single application of cocain; p. p. is not usually definitely removed, although some slight "micropsia" or difficulty in acc. will be felt.

A single application of an extremely small quantity of eserine a few minutes after maximum mydriasis, produces miosis and ciliary spasm as quickly and completely as if no cocain had been used. In my own case the quantity was \( \frac{1}{8000} \) grain.

A few careful observations have been made (chiefly by Mr. Mackenzie and Mr. Lawford) on the p. and acc., when cocain is used, often at short intervals, e.g. every five minutes for an hour. When this is done the acc. is
almost completely destroyed after about eight to ten discs have been used, p. p. being removed to about 60 cm.; complete cycloplegia, however, only lasts a very short time, beginning to pass off in about a quarter of an hour after the last disc, and being entirely gone in an hour, although the p. continues dilated for many hours. We find further that even these repeated doses of cocain have no more power of resisting eserine than the single application. Neither does the anæsthesia seem to last longer, at any rate not much longer, than after one or two applications.

The same seems to hold good even when a 20 per cent. solution is used every five minutes for an hour; the p. p. recovered, in such a case tried yesterday, from 57 cm. to 25 cm. within thirty minutes of the last application, and eserine being then used, acted as on a natural eye. T. was not sensibly altered by the cocain.

Thus it seems clear that cocain does not dilate the p. or affect the acc. by acting directly on the intra-ocular branches of the third nerve; its mode of action on the pupil, together with the retraction of lid and whitening of eyeball, seem to point to spasm of muscular fibres supplied by the sympathetic. It seems a fair suggestion that the paralysis of acc. may be due to temporary contraction of the perforating branches of the anterior ciliary arteries and consequent temporary bloodlessness of the ciliary muscle; or possibly a corresponding condition of the intra-ocular motor nerve-fibres and ganglion cells may afford a better explanation.

(December 11th, 1884.)

<table>
<thead>
<tr>
<th>Time</th>
<th>Anaesthesia</th>
<th>Retraction of lid</th>
<th>Pupil</th>
<th>Acc.</th>
<th>Blanching of eye</th>
</tr>
</thead>
<tbody>
<tr>
<td>11:25</td>
<td>Cocain</td>
<td>...</td>
<td>...</td>
<td>9 mm.</td>
<td>...</td>
</tr>
<tr>
<td>11:27</td>
<td>Cocain</td>
<td>...</td>
<td>...</td>
<td>9 mm.</td>
<td>...</td>
</tr>
<tr>
<td>11:30</td>
<td>Cocain</td>
<td>...</td>
<td>...</td>
<td>95 mm.</td>
<td>...</td>
</tr>
<tr>
<td>11:32</td>
<td>Cocain</td>
<td>...</td>
<td>...</td>
<td>9 mm.</td>
<td>...</td>
</tr>
<tr>
<td>11:37</td>
<td>Cocain</td>
<td>...</td>
<td>...</td>
<td>95 mm.</td>
<td>...</td>
</tr>
<tr>
<td>11:40</td>
<td>Cocain</td>
<td>...</td>
<td>...</td>
<td>11 cm., but only 2 J.</td>
<td>...</td>
</tr>
<tr>
<td>11:42</td>
<td>Cocain</td>
<td>...</td>
<td>...</td>
<td>51 cm. (2 J.)</td>
<td>...</td>
</tr>
<tr>
<td>11:44</td>
<td>Cocain</td>
<td>...</td>
<td>...</td>
<td>57 cm. (2 J.)</td>
<td>...</td>
</tr>
<tr>
<td>11:47</td>
<td>Cocain</td>
<td>...</td>
<td>...</td>
<td>57 cm. (2 J.): 8 c. -5 D.</td>
<td>...</td>
</tr>
<tr>
<td>11:52</td>
<td>Cocain</td>
<td>...</td>
<td>...</td>
<td>47 cm., but only 4 J.</td>
<td>...</td>
</tr>
<tr>
<td>11:55</td>
<td>Cocain</td>
<td>...</td>
<td>...</td>
<td>9 mm.</td>
<td>...</td>
</tr>
<tr>
<td>11:57</td>
<td>Cocain</td>
<td>...</td>
<td>...</td>
<td>95 mm.</td>
<td>...</td>
</tr>
</tbody>
</table>

**Eserine case.**

Observations by Mr. W. G. MacKenzie.

1 drop of 20 per cent. solution of cocain put in every 5 minutes from 11.2 to 11.57 (11 applications in all).

T. tried repeatedly at intervals, and always the same as other eye (= n.).
The patient was a healthy clerk, about seventeen, well nourished, with good colour. V. n.

J. B. L— Right eye.

<table>
<thead>
<tr>
<th>Time</th>
<th>Disc Description</th>
<th>Distance</th>
<th>Magnification</th>
<th>Observations</th>
</tr>
</thead>
<tbody>
<tr>
<td>12.37</td>
<td>Disc (5 minutes after 1st disc)</td>
<td>Partial Complete</td>
<td>4.5 mm. P. p. 14 cm.</td>
<td>Ciliary injection Nov. 30th, 1884, 1 disc (3 gr.) put into R. at 12.32 noon, and every 5 minutes till 1.27 (12 discs).</td>
</tr>
<tr>
<td>12.39</td>
<td>Disc.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12.42</td>
<td>Disc.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12.45</td>
<td>Disc.</td>
<td></td>
<td>P. p. 15 cm.</td>
<td></td>
</tr>
<tr>
<td>12.47</td>
<td>Disc.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12.50</td>
<td>Disc.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12.52</td>
<td>Disc.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12.55</td>
<td>Disc.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12.57</td>
<td>Disc.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1.0</td>
<td>Disc.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1.2</td>
<td>Disc.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1.5</td>
<td>Disc.</td>
<td></td>
<td>Wide 22.5 cm. with effort (L. = 14 cm.)</td>
<td></td>
</tr>
<tr>
<td>1.7</td>
<td>Disc.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1.10</td>
<td>Disc.</td>
<td></td>
<td>7.5 mm.</td>
<td></td>
</tr>
<tr>
<td>1.12</td>
<td>Disc.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1.15</td>
<td>Disc.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1.17</td>
<td>Disc.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1.20</td>
<td>Disc.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1.22</td>
<td>Disc.</td>
<td>Noticeable (not looked for before)</td>
<td>8.5 mm. 53 cm.</td>
<td></td>
</tr>
<tr>
<td>1.25</td>
<td>Disc.</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

USE OF COCAIN IN OPHTHALMIC PRACTICE.
<table>
<thead>
<tr>
<th>Time</th>
<th>Anesthesia</th>
<th>Retraction of lid</th>
<th>Pupil</th>
<th>Aec.</th>
<th>Blanching of eye</th>
</tr>
</thead>
<tbody>
<tr>
<td>1:27</td>
<td>Disc</td>
<td>...</td>
<td>...</td>
<td>8.5 mm.</td>
<td>58 cm., reads 12 J., +4 D. = 1 J. p. p. 20 cm.</td>
</tr>
<tr>
<td>1:30</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>Still injected</td>
</tr>
<tr>
<td>1:35</td>
<td>...</td>
<td>...</td>
<td>Still complete</td>
<td>...</td>
<td>53 cm.</td>
</tr>
<tr>
<td>1:40</td>
<td>...</td>
<td>Slight sensibility</td>
<td>...</td>
<td>...</td>
<td>53 cm.</td>
</tr>
<tr>
<td>1:45</td>
<td>...</td>
<td>...</td>
<td>8.5 mm.</td>
<td>45 cm.</td>
<td></td>
</tr>
<tr>
<td>1:50</td>
<td>...</td>
<td>...</td>
<td>8.5 mm.</td>
<td>48 cm.</td>
<td></td>
</tr>
<tr>
<td>1:55</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>24 cm.</td>
<td></td>
</tr>
<tr>
<td>2:0</td>
<td>Surveillance</td>
<td>...</td>
<td>...</td>
<td>Still injected</td>
<td></td>
</tr>
<tr>
<td>2:10</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>Still injected</td>
</tr>
<tr>
<td>2:15</td>
<td>...</td>
<td>...</td>
<td>Still slight numbness</td>
<td>...</td>
<td>21 cm.</td>
</tr>
<tr>
<td>2:20</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>20 cm.</td>
<td></td>
</tr>
<tr>
<td>2:25</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>15 cm.</td>
<td></td>
</tr>
<tr>
<td>2:30</td>
<td>...</td>
<td>...</td>
<td>Dilated</td>
<td>14 cm.</td>
<td>No bleaching</td>
</tr>
</tbody>
</table>

P. still larger than other twenty-four hours later. Discs caused smarting even when there was complete anaesthesia to touch.

Boy, Crocker, at 16 (Moorfields).

<table>
<thead>
<tr>
<th>Time</th>
<th>Before experiment</th>
<th>1st disc.</th>
<th>2nd (5 minutes later)</th>
<th>3rd</th>
<th>4th</th>
<th>5th</th>
<th>6th</th>
<th>7th</th>
<th>Eserine case</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>Partial</td>
<td>...</td>
<td>9 cm.</td>
<td>...</td>
<td>Observations by Mr. W. G. MacKenzie.</td>
</tr>
<tr>
<td></td>
<td>10 mm.</td>
<td>7.5 mm.</td>
<td>8.5 mm.</td>
<td>9 mm.</td>
<td>10 mm.</td>
<td>10 cm.</td>
<td>14 cm.</td>
<td>...</td>
<td>Nov. 29th, 1884,</td>
</tr>
<tr>
<td>S. No.</td>
<td>Complete</td>
<td>...</td>
<td>...</td>
<td>44 cm., but only 10 J.</td>
<td>44 cm. (10 J.)</td>
<td>57 cm. (10 J.)</td>
<td>57 cm. (10 J.), No. II.</td>
<td>5 minutes for an hour to one eye.</td>
<td></td>
</tr>
<tr>
<td>--------</td>
<td>----------</td>
<td>-----</td>
<td>-----</td>
<td>----------------------</td>
<td>----------------</td>
<td>----------------</td>
<td>----------------------------</td>
<td>----------------------------------</td>
<td></td>
</tr>
<tr>
<td>9th</td>
<td></td>
<td></td>
<td></td>
<td>10 mm.</td>
<td>48 cm. (10 J.)</td>
<td>60 cm or 55 cm. (10 J.)</td>
<td>Healthy, emmetropic.</td>
<td>Anesthesia and retraction of lid not noted systematically.</td>
<td></td>
</tr>
<tr>
<td>10th</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11th</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12th</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5 mins. after eserine</td>
<td>Complete</td>
<td>...</td>
<td>...</td>
<td>6 mm.</td>
<td>30 cm. (6 J.)</td>
<td>20 cm. (1 J.)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>18 &quot; cocain</td>
<td></td>
<td></td>
<td></td>
<td>1.5 mm.</td>
<td>7 cm.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>15 &quot; eserine</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>28 &quot; cocain</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>90 &quot; eserine</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>103 &quot; cocain</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

E. W—, æt. 34.

Disc inserted 11.6 p.m. 3 minutes later. Complete. 0 Retracted. Dilating. 1 disc only (1/10 gr.) inside lower lid.

Nov. 22nd, 1844. Experiments begin 11.6 p.m. R. eye. 1 disc going off at top and centre; still complete below.
<table>
<thead>
<tr>
<th>Time</th>
<th>Anesthesia</th>
<th>Retraction of lid</th>
<th>Pupil</th>
<th>Acc.</th>
<th>Blanching of eye</th>
</tr>
</thead>
<tbody>
<tr>
<td>31 minutes later</td>
<td>...</td>
<td>Still retracted</td>
<td>...</td>
<td></td>
<td></td>
</tr>
<tr>
<td>38</td>
<td>...</td>
<td>Still retracted</td>
<td>...</td>
<td>...</td>
<td>Eye whiter than other</td>
</tr>
<tr>
<td>44</td>
<td>...</td>
<td>&quot;</td>
<td>Same</td>
<td>? Sl. weakening of Acc.</td>
<td></td>
</tr>
</tbody>
</table>

**Lovell P—, wt. 38.**

<table>
<thead>
<tr>
<th>Time</th>
<th>Anesthesia</th>
<th>Retraction of lid</th>
<th>Pupil</th>
<th>Acc.</th>
<th>Blanching of eye</th>
</tr>
</thead>
<tbody>
<tr>
<td>Disc inserted 11.6 p.m.</td>
<td>Complete</td>
<td>...</td>
<td>No dilatation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4 minutes</td>
<td>Complete Ocular conjunctiva. Complete</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>Complete Cornea.—Anaesthesia going off at centre and top; still complete below</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>Anaesthesia going off Anesthesia very nearly gone</td>
<td>...</td>
<td>Still larger Same</td>
<td>...</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>...</td>
<td>No retraction</td>
<td>...</td>
<td>...</td>
<td>Other p. also acts slowly</td>
</tr>
<tr>
<td>19</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td></td>
</tr>
<tr>
<td>29</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>Eye whiter than other</td>
</tr>
<tr>
<td>37</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td></td>
</tr>
<tr>
<td>Before experiment</td>
<td>2 minutes after cocaine</td>
<td>5-10 &quot; &quot;</td>
<td>12 &quot; &quot;</td>
<td>14 &quot; &quot;</td>
<td>45 &quot; &quot;</td>
</tr>
<tr>
<td>-------------------</td>
<td>------------------------</td>
<td>-----------</td>
<td>--------</td>
<td>--------</td>
<td>--------</td>
</tr>
<tr>
<td></td>
<td>...</td>
<td>Partial</td>
<td>...</td>
<td>...</td>
<td>Noticeable</td>
</tr>
<tr>
<td>F. S—, F., at 19. Left eye. V. 6 6 6 + .75 D. c.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Before experiment</td>
<td>2 minutes after cocaine</td>
<td>3½ &quot; &quot;</td>
<td>8 &quot; &quot;</td>
<td>9-11 &quot; &quot;</td>
<td>12 &quot; &quot;</td>
</tr>
<tr>
<td></td>
<td>...</td>
<td>Partial</td>
<td>None</td>
<td>Complete</td>
<td>&quot; &quot;</td>
</tr>
<tr>
<td>K. W—, F., at 20.</td>
<td>3 minutes after cocaine</td>
<td>4 &quot; &quot;</td>
<td>5 &quot; &quot;</td>
<td>10-15 &quot; &quot;</td>
<td>20 &quot; &quot;</td>
</tr>
<tr>
<td>Time</td>
<td>Effect</td>
<td>Anesthesia</td>
<td>Retraction of lid</td>
<td>Pupil</td>
<td>Acc.</td>
</tr>
<tr>
<td>-------</td>
<td>---------------------------------------</td>
<td>------------------------------------</td>
<td>-------------------</td>
<td>---------</td>
<td>--------</td>
</tr>
<tr>
<td>Before experiment</td>
<td></td>
<td>Palpebral fissure</td>
<td>R. 11 mm</td>
<td>R. 3 mm</td>
<td>P. p. 7&quot;</td>
</tr>
<tr>
<td>11.27 a.m., one disc to R. lower conjunct. sac.</td>
<td>2½ minutes</td>
<td><em>Ocular conjunct.</em>—Anæsthesia lower part, not of upper part.</td>
<td>L. 11 or 10½</td>
<td>L. 3 mm</td>
<td>P. p. 7&quot;</td>
</tr>
<tr>
<td></td>
<td>3½ &quot;</td>
<td><em>Ocular conjunct.</em>—Upper part still feels.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>4 &quot;</td>
<td><em>Ocular conjunct.</em>—Upper part scarcely feels.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>7 &quot;</td>
<td><em>Ocular conjunct.</em>—Anæsthesia nearly gone</td>
<td>Retracted (12 mm. at least)</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td></td>
<td>9 &quot;</td>
<td><em>Ocular conjunct.</em>—Anæsthesia going</td>
<td>12 mm.</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td></td>
<td>11 &quot;</td>
<td>Still slight anæsthesia</td>
<td></td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td></td>
<td>12 &quot;</td>
<td></td>
<td></td>
<td>4 mm.</td>
<td>...</td>
</tr>
<tr>
<td></td>
<td>15 &quot;</td>
<td></td>
<td></td>
<td>4½ mm.</td>
<td>7½&quot;</td>
</tr>
<tr>
<td></td>
<td>20 &quot;</td>
<td><em>Ocular conjunct.</em>—Still very slight anæsthesia</td>
<td>12-13 mm.</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

---

*Eserine case.*

Nov. 23rd, 1884.

1 disc only ($\frac{3}{4}$ gr.). R. eye.
USE OF COCAIJN IN OPHTHALMIC PRACTICE.

<table>
<thead>
<tr>
<th>Time</th>
<th>Condition</th>
<th>Pressure</th>
<th>Eye Movement</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>27</td>
<td>Cold douche less felt than by L eye; eye, however, still feels colder than L.</td>
<td>...</td>
<td>65 mm.</td>
<td>7 1/4&quot;, but Acc. seems more difficult than before (micropsia)</td>
</tr>
<tr>
<td>47 (12.14)</td>
<td>Eye feels subjectively warmer than L. (Retraction nearly gone)</td>
<td>11 mm.</td>
<td>&quot;</td>
<td>7 3/4&quot;</td>
</tr>
<tr>
<td>12.14 (47 min. after cocaine)</td>
<td>eseine put in (1/4 of a disc containing 1/2000 = 1/5000 gr.)</td>
<td>...</td>
<td>...</td>
<td>L. fissure 10. Retraction of R. nearly gone; but voluntary effort still exposes sclerotic above R. cornea much sooner than L.</td>
</tr>
<tr>
<td>3 min. after eseine</td>
<td>Twitching of inner end of lower lid.</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>6</td>
<td>Twitching of both lids; lower lid, inner end; upper lid, outer end.</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>9</td>
<td>Still twitching 6 or 6.5 mm.</td>
<td>14</td>
<td>P. p. 7 1/2&quot;</td>
<td>...</td>
</tr>
<tr>
<td>14</td>
<td>Still twitching 5.5 mm.</td>
<td>16</td>
<td>P. p. 6 1/2&quot;</td>
<td>...</td>
</tr>
<tr>
<td>19</td>
<td>Still twitching 4 mm.</td>
<td>21</td>
<td>P. p. 5 1/2&quot;</td>
<td>...</td>
</tr>
<tr>
<td>21</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
</tbody>
</table>

Slight aching of eye. Continuous slow aching at upper part of eye. Much aching during strong Acc. R. p. = L. = 3 mm.
E. N., æt. 39—continued.

<table>
<thead>
<tr>
<th>Time</th>
<th>Amnesia</th>
<th>Retraction of lid</th>
<th>Pupil.</th>
<th>Acc.</th>
<th>Blanching of eye</th>
</tr>
</thead>
<tbody>
<tr>
<td>26 minutes after eserine</td>
<td>...</td>
<td>No spasm for some minutes</td>
<td>2.6 mm.</td>
<td>P. p. 5/4&quot;</td>
<td>...</td>
</tr>
<tr>
<td>28 &quot; &quot;</td>
<td>...</td>
<td>...</td>
<td>2.3 mm.</td>
<td>...</td>
<td>Whitening has gone off.</td>
</tr>
<tr>
<td>36 &quot; &quot;</td>
<td>...</td>
<td>...</td>
<td>2 mm.</td>
<td>P. p. 4/4&quot;</td>
<td>...</td>
</tr>
<tr>
<td>46 &quot; &quot;</td>
<td>...</td>
<td>...</td>
<td>1.6 mm.</td>
<td>P. p. 4&quot;</td>
<td>...</td>
</tr>
<tr>
<td>56 &quot; &quot;</td>
<td>...</td>
<td>...</td>
<td>&quot;</td>
<td>&quot;</td>
<td>Elevation of lids still same as before.</td>
</tr>
<tr>
<td>1 h. 15 m. &quot;</td>
<td>...</td>
<td>...</td>
<td>&quot;</td>
<td>&quot;</td>
<td>Voluntary elevation of lids now equal on the two sides.</td>
</tr>
<tr>
<td>2 hours &quot;</td>
<td>...</td>
<td>...</td>
<td>&quot;</td>
<td>P. p. 5/4&quot;</td>
<td></td>
</tr>
<tr>
<td>6 &quot; &quot;</td>
<td>...</td>
<td>...</td>
<td>3 mm.</td>
<td>P. p. 7/4&quot;</td>
<td></td>
</tr>
</tbody>
</table>

Left eye.

<table>
<thead>
<tr>
<th>Time</th>
<th>Retraction well marked</th>
<th>Pupil</th>
<th>Acc.</th>
<th>Blanching of eye</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.30 p.m. 1 cocain disc 5 minutes</td>
<td>...</td>
<td>6 mm.</td>
<td>3.5 or 4 mm. (dull daylight)</td>
<td></td>
</tr>
<tr>
<td>4 hours</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
<tr>
<td>19 &quot;</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
</tbody>
</table>

Nov. 23rd, 1884, 1 cocain disc (1/60 gr.) 2.30 p.m.
<table>
<thead>
<tr>
<th>Before experiment</th>
<th>1st disc.</th>
<th>2nd disc.</th>
<th>Partial (?)</th>
<th>3rd disc.</th>
<th>4th disc.</th>
<th>5th disc.</th>
<th>6th disc.</th>
<th>7th disc.</th>
<th>8th disc.</th>
<th>Complete</th>
<th>9th disc.</th>
<th>10th disc.</th>
<th>11th disc.</th>
<th>12th disc.</th>
<th>5 min after last disc</th>
<th>5 min later</th>
<th>1 drop of eserine now put in.</th>
<th>17 min after last cocaine</th>
<th>10 min.</th>
<th>100 min.</th>
<th>21 cm, but only 8 J.</th>
<th>32 cm, (6 J.) cm.</th>
<th>12 cm, (6 J.)</th>
<th>21 cm, (4 J.)</th>
<th>8 cm.</th>
<th>85 mm.</th>
<th>6 mm.</th>
<th>1.5 mm.</th>
</tr>
</thead>
</table>

Observations by Mr. W. G. Mackenzie, Nov. 29th, 1884.
1 cocaine disc (1/10 grain) every 5 minutes for an hour.
Healthy emmetropic.
Anesthesia and lid retraction not specially noted.

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>3 minutes after cocain</td>
<td>...</td>
<td>...</td>
<td>3·5 mm.</td>
<td>P. p. 7·5 mm.</td>
<td>Opposite P. under homatrop. = 10 mm.</td>
</tr>
<tr>
<td>5 '' ''</td>
<td>Begins</td>
<td>...</td>
<td>No miosis</td>
<td></td>
<td>Smarting just after cocain put in.</td>
</tr>
<tr>
<td>8 '' ''</td>
<td>...</td>
<td>Noticeable</td>
<td>4·5 mm.</td>
<td></td>
<td>5 minutes later “lid feels very thick.”</td>
</tr>
<tr>
<td>12 '' ''</td>
<td>...</td>
<td>...</td>
<td>6 mm.</td>
<td></td>
<td>35 minutes later “drawing feeling at teno-oculi.”</td>
</tr>
<tr>
<td>20 '' ''</td>
<td>...</td>
<td>None</td>
<td>...</td>
<td>P. p. 12·5 mm.</td>
<td></td>
</tr>
<tr>
<td>32 '' ''</td>
<td>Quite gone off</td>
<td>...</td>
<td>6 mm.</td>
<td>P. p. 7·5 mm.</td>
<td></td>
</tr>
<tr>
<td>to 60 '' ''</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>5 minutes after cocain</td>
<td>...</td>
<td>...</td>
<td>3 mm.</td>
<td>P. p. 11 cm. (1 J.)</td>
<td>Immediate smarting.</td>
</tr>
<tr>
<td>8 '' ''</td>
<td>Begins</td>
<td>...</td>
<td>3·5 mm.</td>
<td></td>
<td>No after-sensation.</td>
</tr>
<tr>
<td>10 '' ''</td>
<td>Complete, but only at point of application</td>
<td>None</td>
<td>No miosis</td>
<td>P. p. not altered</td>
<td></td>
</tr>
<tr>
<td>25 '' ''</td>
<td>Quite gone off</td>
<td>...</td>
<td>6·5 mm.</td>
<td></td>
<td>P. of opposite eye (congested after operation) under atropine = 6 mm.</td>
</tr>
<tr>
<td>80 '' ''</td>
<td>...</td>
<td>...</td>
<td>3 mm.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>30 hours ''</td>
<td>...</td>
<td>...</td>
<td>...</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Left eye (congested, and some iritis after curette extraction. P. under atropine = 6 mm.).

<table>
<thead>
<tr>
<th>1 application of cocain</th>
<th>Anesthesia.</th>
<th>Retraction of lid.</th>
<th>Pupil.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Never complete anywhere</td>
<td>...</td>
<td>...</td>
<td>Remained at 6 mm.</td>
</tr>
</tbody>
</table>

No after-sensation.
Use of Cocain in Ophthalmic Practice.

<table>
<thead>
<tr>
<th>Before experiment. 1 application of cocain</th>
<th>2 minutes after cocain</th>
<th>...</th>
<th>4.5 mm.</th>
<th>P. p. 11 cm.</th>
<th>Opposite P. under homatrl. = 7.5 mm. Immediate prickling for a minute.</th>
</tr>
</thead>
<tbody>
<tr>
<td>10 &quot; &quot;</td>
<td>Begins</td>
<td>...</td>
<td>...</td>
<td>P. p. 18 cm.</td>
<td></td>
</tr>
<tr>
<td>30 &quot; &quot;</td>
<td>Complete</td>
<td>...</td>
<td>10 mm. or more</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

E. B—, F., at. 12. Second observation on right eye.

<table>
<thead>
<tr>
<th>After 12 applications of castor-oil cocain in one hour</th>
<th>5 minutes after last cocain 1 drop of eserine gr. iv to 3</th>
<th>9 or 9.5 mm.</th>
<th>12 cm. 1 J. and 6/12 partly Hm. 5</th>
<th>Castor-oil. sol. of cocain (2½ per cent.) every 5 minutes for an hour; then eserine (1 per cent.) once; then 1 cocain disc (200 gr.).</th>
</tr>
</thead>
<tbody>
<tr>
<td>10 minutes later</td>
<td>35 minutes after eserine 1 cocain disc</td>
<td>1.75 mm.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3 minutes later</td>
<td>Complete anaesthesia</td>
<td>...</td>
<td>P. same 1.75 mm.</td>
<td></td>
</tr>
<tr>
<td>6 hours after cocain, and 6½ hours after eserine</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
3. The cocainised eye.

By Walter H. Jessop.

The object of this paper is to examine in detail the phenomena presented by an eye under the local influence of cocain.

The facts adduced have been obtained by applying to human eyes the hydrochlorate of cocain in solutions of 2, 4, and 20 per cent. I have discussed the points in the following order:

I. The local anaesthesia has been so well described by Koller and others that it is hardly necessary to say much. It is excessively local, and by careful attention to its application, such as drying the surface to be anaesthetised, it may be limited to the extent required. In the eye apparently it spreads more than in other parts owing to the movements of the lids; this diffusion may also be seen in loose cellular tissues such as the eyelids. The anaesthesia does not last long, and in muscular structures is accompanied by pain like cramp.

The anaesthesia is accompanied by a feeling of coldness in the eye, followed by dryness. The superficial anaesthesia may be followed by loss of sensibility in the deeper structures by several applications, the drug thus sinking into the tissues; this is also produced by injections beneath the superficial parts. That the peripheral terminations of the sensory nerves are the parts affected may be demonstrated by the pain produced by cutting beneath the parts in direct contact with the drug.

II. Enlargement of the palpebral fissure is often the first sign noticeable, especially when strong solutions are used. It is due to raising of the upper lid and depression of the lower, thus giving rise to appearance of prominence of the eye as the lids retract. On noticing the change in my own eye, I thought it could not be due, as Koller asserted, to the want of sensibility in the eyeball, and on
looking carefully I found that the lower lid was depressed somewhat spoon-like to the outer part, and that the length between the inner canthus and the punctum was increased, the latter being slightly inverted, and so giving rise to slight lachrymation.

Here, then, was a condition quite different to facial palsy, in which the lid tending to be depressed towards the middle speedily everts both the edge of the lid and the punctum.

Dr. Steavenson very kindly faradised my eye twice when under the influence of cocain, with the effect that there was increased action of the orbicularis of the cocainised eye to the faradaic current, and apparently the whole muscle, including the ciliary portion, responded to the current.

To explain this increased faradaic action of the orbicularis, the following three causes suggested themselves:

1. That paralysis of some of the fibres of the orbicularis might act as a slight weight to the muscle, and so increase the action of the remaining fibres.

2. That it might be due to the orbicularis acting against the increased action of another muscle.

3. That direct irritation of the muscular fibres of the orbicularis by the drug might account for it.

Luckily I found a patient with complete facial palsy, and no action of the orbicularis palpebrarum to the faradaic current; his lower lid presented the ordinary everted condition. Before Dr. Steavenson and others, a few drops of the 20 per cent. solution were applied to the lower lid; in five minutes there was distinct difference in the eversion of the lid, the punctum being slightly elevated, and after another application the lid and punctum were still more raised with a tendency to inversion.

From this I argued, the action of the orbicularis being out of the question, that the cause was irritation of the unstriped muscular fibres of the lower lid by the drug.

This contraction of the unstriped muscular fibres of the lid, acting in the opposite direction to the orbicularis
palpebrarum, would account for the increased faradaic excitability of the latter muscle in a cocainised eye.

The lids of an eye under cocain may be both affected if the drug is brought into contact with their surface, and therefore the one usually most so is the lower.

The enlargement of the palpebral aperture can therefore be explained by irritation of the unstriped muscular fibres of the lids supplied by the sympathetic.

III. The dilatation of the pupil of an eye under cocain I have observed now in over a hundred cases, and the chief points are its large size and its action to light and the movements of accommodation.

The larger the mydriasis the less perceptible is the contraction of the pupil to light and accommodation, and at first in extreme cases I thought the iris did not act to light, but on careful observation with a pair of spectacles + 4 D. on my own eyes I found in all cases a slight and quick contraction followed instantaneously by a swing back to dilatation. The action being slight, is, I presume, due to the sphincter pupillae being so stretched as to have little power to contract.

The contraction of the iris to light may also be induced by throwing light into the opposite eye even when it is under atropine.

The cocainised pupil is easily affected by pressure from without, and may thus be pushed into any shape, also it commences to dilate at the place where the drug is applied.

The continuance of the mydriasis is comparatively short, the pupil attaining its normal size in from twelve to twenty-four hours.

The following cases show the main points in the mydriasis of cocain, and that the stronger the solution the quicker the initial, and ad maximum dilatation.

H. C—, set. 15. Pupils 6·5 mm.; cocain 4 per cent. on right eye at 2.5 p.m.; at 2.25 p.m., pupil 10 mm., and not further increased. The pupil had resumed its normal size twelve hours afterwards.
THE COCAINISED EYE.

W. H—, æt. 30. Pupils 5 mm.; at 10.20 a.m., cocain 20 per cent. in left eye; in seventeen minutes pupil 8 mm.; and other instillations did not increase its size. Pupil regained normal size ten hours afterwards.

E. W—, æt. 31. Pupils 5 mm.; at 11.20 a.m. cocain 2 per cent.; at 11.30 a.m., cocain 2 per cent.; and at 11 a.m., pupils 8.5 mm. Fourteen hours afterwards pupils normal size.

To consider the reason of the action of cocain on the pupil we may best, I think, enumerate briefly the physiological action of the iris as usually taught.

The muscular fibre is arranged in two sets—radiating fibres supplied by the sympathetic nerve, and circular fibres supplied by the third nerve. This muscular system is normally in a state of tonus, and not of excited antagonistic action, and therefore on section of either nerve we get either a slight contraction or a partial dilatation, but irritation of either nerve gives rise in one case to extreme myosis, and in the other to extreme mydriasis.

The circular muscular fibres are also better and stronger developed than the radiating fibres, hence the greater tendency always to contraction of the pupil.

Section of the third nerve always give rise to absence of the movements of the pupil to light and accommodation.

Now, considering the action of the following drugs applied locally, namely atropine, pilocarpine, and eserine.* Atropine (including homatropine, belladonna, &c.) acts first by paralysing the endings of the oculo-motor nerves in the sphincter, and then if the drug is strong the mydriasis is increased by irritation of the sympathetic or dilator fibres, shown by atropine increasing the mydriasis produced by cutting the third nerve, and also by the dilatation of the pupil produced by atropine being increased on irritation of the cervical sympathetic.

Pilocarpine acts by stimulating the peripheral terminations of the oculo-motor nerve in the sphincter producing

* 'Pharmacology, Therapeutics, and Materia Medica,' 1885; T. Lauder Brunton, M.D., F.R.S., pp. 186—189.
myosis, and therefore cannot act in a fully atropinised eye, as the endings of the third nerve are palsied by atropine, and pilocarpine has no effect on the dilator system.

Eserine acts by stimulating the muscular fibres of the iris directly, and hence the stronger sphincter prevails over the weaker radiating fibres, and myosis ensues.

In order to try and account for the mydriasis of cocain, I next tried the effect of the drug in connection with atropine, pilocarpine, and eserine.

Taking a fully dilated cocainised pupil, atropine did not increase the size of the pupil, but stopped the action of the iris to light and accommodation.

F. M—, æt. 25. Pupils 5 mm., two instillations of cocain, 2 per cent. in right eye; pupil became 9·5 mm., acting to light and accommodation; Atrop. Sulp. \( \frac{1}{560} \) gr. added, no increase in size of pupil, although atropine put in three times; the pupil, however, did not act to light or accommodation.

The mydriasis produced by atropine and homatropine was increased by the addition of one drop of cocain 4 per cent. solution.

A. G—, æt. 16. Under atropine (4 grs. to the ounce three times a day for ten days), both pupils 9 mm., and equal, not acting to light or the movements of accommodation. In left eye cocain 4 per cent., and in fifteen minutes the pupil was 10 mm.

The increase of the atropinised pupil by cocain is best seen perhaps in a presbyope, in whom atropine generally does not exercise so much power.

C. H—, æt. 67. Pupils (under atropine for seven days) 5·5 mm., equal, and do not act to light or accommodation. Cocain 4 per cent. once in left eye, and pupil became 7·5 mm.

Pilocarpine reduces the cocainised pupil after several applications to the ordinary myosis of pilocarpine.

W. V—, æt. 3½. Pupils 6·5 mm., and equal; to left eye six applications of cocain 2 per cent., and pupil became 11
mm., after three applications of pilocarpine (2 per cent. solution), pupil became 2 mm.

By taking 2 per cent. solutions of pilocarpine and cocain I found that the pupil dilated by cocain was reduced to the normal size, on taking the drugs in the proportion of one of pilocarpine to four of cocain.

Eserine very easily reduces the cocainised pupil to a condition of myosis.

T. C—, aged 4. Pupils 6 mm., equal, after 8 instillations of cocain (2 per cent. solution), on right eye pupil was 9 mm.; two instillations of eserine (2 per cent. solution), and pupil became 1 mm. In the same manner as in the pilocarpine experiments I found the proportion to reduce the cocainised pupil to the normal size to be one of eserine to twenty seven of cocain (2 per cent. solutions being used of each).

Considering then the facts we have elicited it is necessary to try and state the exact cause of the mydriasis induced by cocain.

Mydriasis may be induced by paralysis of the sphincter pupillae or its nerve, and also by excitation of the dilating mechanism of the eye, or again by a combination of these causes.

That the cocainised pupil is not produced by palsy of the third nerve, seems certain from the facts that the mydriasis is larger than that of section of the third nerve, the pupil acts always to light and accommodation, and pilocarpine (which acts on the endings of the oculo-motor nerve alone) produces extreme myosis. Experiments also on other parts show that the drug has no effect on the cerebro-spinal motor nerves.

That the sphincter pupillæ muscle is not paralysed is shown by its action to light and accommodation, and also by the fact that eserine, which stimulates directly the muscular fibre, easily produces myosis. Therefore we have left for consideration the dilating mechanism, and must see if the several parts can be reconciled by that alone.

The pupil produced by irritation of the sympathetic is
exactly like that of cocain, being very large and capable of acting to light and accommodation. On this theory also, all the facts with regard to the action of the different drugs may be explained, eserine and pilocarpine producing myosis of the cocainised eye because they act on the sphincter pupillae or the third nerve, the increased dilatation by cocain of the atropinised pupil being due to an extra stimulation of the dilator mechanism.

That the dilatation is due rather to the stimulation of the endings of the mydriatic nerve than of the radiating muscular fibres, is, I think, shown by the great mydriasis produced, as both circular and radiating fibres being unstriped muscle the irritation of both sets would produce a resultant inducing contraction of the pupil.*

IV. Paralysis of accommodation occurs always in a fully cocainised eye, and reaches its climax soon after the pupil is fully dilated, when there may be absolute loss of accommodation as in the following case:—

Dr. R. T—, act. 30, consulted me about his eyesight. Owing to the smallness of his pupils, and his being amblyopic in one eye, I did not like to use atropine or even homatropine, so he consented to the application of a 20 per cent. solution of cocain to the left eye.

In ten minutes the pupil increased from 2 mm. to 8.5 mm., and the whole of his accommodation was paralysed, enabling me to make an easy estimation of his hypermetropic astigmatism. In several experiments on my own eyes, I found that four or five applications of the 4 per cent. solution of cocain took away four dioptries of accommodation.

The only point I would suggest is whether astigmatism may be produced owing to the alteration of the cornea by becoming flaccid.

I am tabulating the numerous cases I have collected

* In a paper on "Cocaine Mydriasis," read before the Royal Society on June, 1885, I showed, by experiments on animals, &c., that the dilatation of the pupil was due to direct irritation of the endings of the mydriatic nerve in the eye ("Proceedings of the Roy. Soc.," vol. xxxviii, p. 432).
to find out the effects on myopic and hypermetropic eyes, but the facts are not yet ready to lay before you.

The paralysis of accommodation lasts from about half an hour to an hour and passes off before the pupil regains its normal size.

Mr. Nettleship has suggested that this paralysis of accommodation may be due to anaemia of the ciliary body by constriction of its vessels; this would be explained by cocaine acting on the vessels through the sympathetic.

V. On applying the drug to the normal eye there is marked pallor of the vessels of the conjunctiva, and this may also be noticed in other parts, such as the lips, &c.

On vessels dilated by inflammation it seems to have little effect.

This action can be explained by its acting as a local irritant to the vaso-constrictor nerves from the sympathetic, and the changes in inflammation would prevent the muscular coat of the vessels contracting.

VI. One marked feature seems harder to explain, namely, the flaccidity of the cornea attended by alteration of tension.

In an eye well under the influence of cocaine one can, by pressing on the cornea, indent it easily, and the tension of the anterior part of the eye is apparently diminished. I have noticed in several cases that after the application of the drug there was a slight increase of tension posteriorly; which, however, soon disappeared, the tension returning to normal or even being diminished. This increase of the tension in the posterior chamber, and at the same time apparent diminution of tension of the anterior chamber, has been noticed by Benson and others.

To try and explain these phenomena one might imagine that the sympathetic irritation producing dilatation of the pupil acting against the intact sphincter muscle and dilating it keeps the pupillary border close to the anterior part of the capsule of the lens, and thus cuts off the filtration from the posterior chamber; the constriction of the iritic vessels and the tucking up of the iris into the iritic
angle would enlarge the anterior chamber temporarily without any increase of fluid to fill it up, and hence the cornea would be unsupported towards the centre and become flaccid, and also the tension in the anterior chamber would be diminished. During this time the ciliary body being full of blood, filtration would take place into the posterior chamber and so increase its tension.*

VII. I have made several investigations on the circulation of the retina, but could see no constriction or alteration in either veins or arteries. The sensibility of the retina tried for colours and fields of vision seems normal both in bright and dull lights.

From the foregoing considerations we can, I think, explain the anaesthesia by the drug paralysing the peripheral sensory endings of the cerebro-spinal nerves, and all the other symptoms by its irritating the endings of the sympathetic nerves.

Passing now from the physiological action of the drug to its uses in ophthalmic surgery, I will briefly mention some operations I have done on patients under its influence.

The operations comprise twenty-two, namely, two extractions for senile cataract, three needlings for soft cataract, three iridectomies (one for glaucoma), two tenotomies for squint, three removals of Meibomian cysts, slitting up three caniculi, opening periosteal abscess of orbit, five removals of foreign bodies from the cornea. To these may be added the passage of probes down the nasal duct six times.

The first extraction case was done on a woman of 65; two instillations of 4 per cent. solution of cocain on the cornea at five minutes' interval; the cornea was perfectly anaesthetic, but she suffered great pain on seizing the iris with the forceps.

The second case was a man of 79, and the strength of

* Since writing this, I have made some experiments on animals, and have always proved diminution of tension of the eye under cocaine, and have not been able to demonstrate any increase of tension.
the solution used was 20 per cent.; six instillations at five minutes' interval were placed as nearly as possible over the seat of section of the cornea; he experienced not the slightest pain even on cutting the iris, although I had warned him it would be painful, and asked him to say the exact time when it was so.

The needling operations were apparently quite painless, though in children. The iridectomy cases all experienced pain, especially the glaucoma case, which I put down to the heightened tension + 3 preventing absorption.

In my second case of squint, after rendering the conjunctiva over the seat of incision anaesthetic, I injected apparently into the internal rectus, beneath the capsule of Tenon, two minims of the 4 per cent. solution, and all the steps of the operation were painless.

My first case was simply by conjunctival instillation, and here pain was felt on lifting the rectus.

In the same way in one of the Meibomian cyst cases an injection beneath the conjunctiva, and into the cyst, rendered the operation painless, but in my first cases mere conjunctival instillation was accompanied by pain.

In my last case of slitting up the canaliculus, the operation was painless owing to the steps taken. The patient was a nervous boy, and it was with some difficulty I could get him on the operating table. I dropped some 20 per cent. solution on the inner canthus, and then after five minutes introduced a small cannula, made by Messrs. Arnold, into the punctum, and by means of a small syringe attached to the cannula I injected some of the solution along the canaliculus. After five minutes the canaliculus was slit up without any pain, and a piece from the internal wall cut off.

As the boy might have had a low degree of sensitivity I tried to perform the operation on the opposite side without the anaesthetic, but soon had to desist, and it was only on promising to do the operation in the same manner as the first, that I got him back again on the table, and met with the same success as to absence of pain.
In removal of foreign bodies from the cornea the anaesthetic properties of the drug render the operation quite painless, but care must be taken as the flaccidity of the cornea due to cocain increases often the difficulty of removing the body, and leads therefore to great scratching and destruction of the corneal epithelial surface.

For the passage of probes the canaliculus and nasal duct may be rendered quite anaesthetic by cocain, and an ordinary sized probe passed down without pain, but the passage of a very large one giving rise to tension induces pain.

Since making this list I have done an iridectomy by inducing first of all local anaesthesia of the cornea by conjunctival instillations, and then injected a minim of the 4 per cent. solution into the anterior chamber by means of a bored needle attached to a syringe. Five minutes after the injection a perfectly painless iridectomy was done.

This last iridectomy, and the one done in the second cataract case recorded above, were the only painless operations in which the iris was involved.

The reason of this is that care was taken to apply the drug over the seat elected for operation, and then either by direct application or by imbibition, the solution finds its way to the iris and anaesthetises it.

Looking at the result of these operations and also of many others I have either assisted at or watched, I should make the following suggestions.

That the part to be operated on should be thoroughly dried, the solution of the drug applied exactly over the site selected for operation, and if the deeper parts, such as the iris, are to be anaesthetised let it be used five or six times at about five minutes' interval. Deep structures may also be treated after superficial anaesthesia has been produced by injections beneath the skin, fascia, or into the anterior chamber. This latter operation had been done with perfect success on rabbits before I tried it on the patient mentioned above. As to the strength of the solution to be used it matters apparently little, remembering
that the weaker the solution used the more often and frequent the applications necessary.

In cases of corneal ulcer with extreme photophobia, the patient may be induced to open his eye and spontaneously exhibit it, thus doing away with the struggles often witnessed in trying to get a glimpse at the cornea.

My observation of the increase of the atropinised pupil by cocain led me to apply it in cases of iritis with posterior synechia, which atropine had failed to break down, and in cases in which the adhesions are not very strong it is undoubtedly very valuable, giving rise to relief from pain and apparently diminished tension.

The following are the brief notes of a case:

E. G—, aet. 42, rheumatic iritis in left eye, has had guttae atropine (gr. iv to the 3j) for three weeks.

*Left eye.*—Marked circumcorneal zone; pupil 5 mm., dilated and irregular; posterior synechiae only to be seen on inner and under aspect; very little uveal pigment to be seen on the lens.

Cocain 2 per cent. was dropped into the left eye, the pupil increasing in size almost directly; after three applications the circumcorneal zone was diminished, and the pupil 8.5 mm.; numerous other and smaller adhesions could be seen all round, and fine shreds of uveal pigment within the pupillary border of the iris. Patient spontaneously said his eye felt much easier.

Four days after this he came again, and the notes are:

*Left eye.*—No circumcorneal zone; pupil 8 mm., round and apparently regular. On adding 2 per cent. solution of cocain pupil dilated to 8.5 mm., showing one small adhesion upwards and inwards; uveal pigment as before on capsule of lens, but no other synechiae.

The effect of the drug then in iritis cases seems to be an increased action of the dilator system of the pupil, the mydriasis induced breaking down synechiae which are not affected by atropine.

Besides the mere stretching of the synechiae by dilatation of the pupil, the action of cocain in constricting the small
blood-vessels may also help in breaking down the adhesions by cutting off their blood supply and literally starving them.
REPORT OF THE COUNCIL.

The Council have again to congratulate the Society upon its continued prosperity. During the Session eighteen new members have been elected, so that the total number in now 202, including 25 non-resident members.

Since the last annual meeting the Society has lost two members by death, viz. Drs. Buchanan Baxter and F. A. Mahomed. One resident and three non-resident members have ceased to belong.

The Council recall with satisfaction that early in the Session Sir William Bowman was elected unanimously an honorary member of the Society.

The first Bowman lecture was delivered by the President in November, and at the request of the Council Dr. Hughlings Jackson, F.R.S., has consented to deliver the second lecture.

The Library now contains considerably more than a hundred volumes, nearly all of which have been presented to the Society. The Council recommend the appointment of a special officer as librarian to take charge of it in the future.

A deputation of the Society, consisting of the President, Sir William Bowman, Bart., F.R.S., Mr. John Tweedy, Dr. D. McKeown, Dr. Brailey and Dr. Abercrombie waited on Mr. G. Russell, M.P., at the Local Government Board office on May 8th, to urge upon that Board the necessity for adopting some measures for the prevention of blindness from ophthalmia neonatorum. The deputation was very well received, and though no action has as
yet been taken, there is reason to believe that some good may result.

During the session a considerable number of papers have been communicated, and the meetings have been well attended.

The Committee appointed to consider some points in connection with sympathetic ophthalmia has sifted a large mass of evidence, and it hopes shortly to be in a position to present a report.
ACCOUNT OF RECEIPTS AND PAYMENTS
OF THE
OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM.

<table>
<thead>
<tr>
<th>Receipts</th>
<th>£ s. d.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Balance from 1884</td>
<td>461 6 7</td>
</tr>
<tr>
<td>Subscriptions</td>
<td>150 3 0</td>
</tr>
<tr>
<td>Composition Fees</td>
<td>59 17 0</td>
</tr>
<tr>
<td>Admission Fees</td>
<td>29 8 0</td>
</tr>
<tr>
<td>Sir Wm. Bowman, Bart.</td>
<td>50 0 0</td>
</tr>
<tr>
<td>By Sale of ‘Transactions’</td>
<td>16 19 0</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Payments</th>
<th>£ s. d.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bankers’ Charges</td>
<td>0 9 9</td>
</tr>
<tr>
<td>Mr. Adlard, for Printing Vol. IV of ‘Transactions’ &amp;c.</td>
<td>129 0 0</td>
</tr>
<tr>
<td>Illustrations for Vol. IV</td>
<td>66 1 6</td>
</tr>
<tr>
<td>Rent (from July, 1883, to March, 1885)</td>
<td>63 15 0</td>
</tr>
<tr>
<td>Mr. Poole, for Refreshments</td>
<td>12 12 6</td>
</tr>
<tr>
<td>Publishers’ Fees</td>
<td>2 2 0</td>
</tr>
<tr>
<td>Law Charges</td>
<td>3 11 0</td>
</tr>
<tr>
<td>Secretaries’ Expenses</td>
<td>10 8 9</td>
</tr>
<tr>
<td>Library Fittings, &amp;c.</td>
<td>100 0 0</td>
</tr>
<tr>
<td>Balance</td>
<td>379 13 1</td>
</tr>
</tbody>
</table>

£767 13 7

J. F. STREATFEILD, Treasurer.
Audited and found correct, June 27th, 1885.

J. A. ORMEROD, A. S. MORTON, Auditors.
INDEX.

Aniridia, congenital (W. Lang) . . . . 207

Benson (Arthur H.), cocain . . . . 211
   — cornea and iris . . . . 47
   — lupus of conjunctiva . . . . 41
Bisulphide of carbon, amblyopia from (E. Nettleship) 149
   — — (Ernest Fuchs) . . . . 152
   — Report of Committee on . . . . 157
Bowman lecture by Mr. J. Hutchinson, F.R.S. . 1
Braley (W. A.), on detachment of retina . . . . 113
   — ciliary nerves in eye disease . . . . 93, 98
   — persistent diminished tension . . . . 106
   — double retinal glioma . . . . 61
   — cyclo-choroiditis . . . . 68
   — sympathetic disease . . . . 99
   — optic neuritis, &c. . . . . 178

Cataract, black (H. Power) . . . . 111
   — diabetic, spontaneous disappearance of (E. Nettleship) . 107
Choroiditis, central, with good vision (E. Nettleship) . . . . 147
   — central (W. Lang) . . . . 140, 141
Choroid, atrophy of (A. Stanford Morton) . . . . 142
   — and iris, tubercle of (W. H. Jessop) . . . . 55
Ciliary region, growth over (M. M. McHardy) . . . . 65
   — nerves in eye disease (W. A. Braley) . . . . 93, 98
Cocain (E. Nettleship) . . . . 217
   — (Arthur H. Benson) . . . . 211
   — (Walter H. Jessop) . . . . 240
Coloboma of each optic disc (W. H. Jessop) . . . . 176
Conjunctiva, lupus of (Arthur H. Benson) . . . . 41
Conjugate deviation of eyes (W. Adams Frost) . . . . 197

Vol. V. 17
Index.

Cornea and iris, tuberculosis of, Report of Committee on 53
—— vesicle of (Anderson Critchett and H. E. Juler) 46
Council, Report of 253
Critchett (Anderson and H. E. Juler), vesicle of cornea 46
Cross (F. R.), congenital dislocation of lenses 111
—— cystic tumours of iris 67
Cyclo-choroiditis, with haemorrhage (W. A. Brailey) 68
Cystic tumour of iris (F. R. Cross) 67

Eales (Henry), retinitis albuminurica 126
Ectropion, case of (M. M. McHardy) 45
Edmunds (Walter), optic nerves in intra-cranial disease 184
Evisceration of globe (P. H. Mules) 200
Eyeball, peculiarly shaped (R. Marcus Gunn) 207

Fat in eyelids (M. M. McHardy) 44
Favus of eyelid (M. M. McHardy) 42
Frost (W. A.), night blindness 123
—— conjugate deviation of eyes 197
Fuchs (Ernest), amblyopia from bisulphide of carbon 142

Glioma of both retinae (W. Lang) 64
—— double retinal (W. A. Brailey) 61
Gout in relation to eye diseases (J. Hutchinson) 1
Gumma, intra-ocular, in inherited syphilis (W. Spencer Watson) 56
Gunn (R. Marcus), unicocular irido-choroiditis 144
—— peculiarly shaped eyeball 207
—— proptosis, optic atrophy, and anosmia 180

Hartridge (G.), opaque nerve fibres 177
Hodges (F. H.), granular-looking body on iris 70
Hutchinson (Jonathan), on reflex ophthalmitis 71
—— the Bowman lecture 1

Irido-choroiditis, unicocular (R. Marcus Gunn) 144
Irido-cyclitis with meningitis in a child (E. Nettleship) 101
Iris, new formation of pigment on (E. Nettleship) 66
—— cystic tumour of (F. R. Cross) 67
—— and choroid, tubercle of (W. A. Jessop) 55
—— granular-looking body on (F. H. Hodges) 70
INDEX.

JESSOP (Walter H.), cocain rupture of eyeball . . . . . 259
—— tubercle of choroid and iris . . . . . 199
—— coloboma of each optic disc . . . . . 55
—— coloboma of each optic disc . . . . . 176
JULER (H. E., and Anderson Critchett), vesicle of cornea . . . . . 46

LANG (W.), congenital aniridia . . . . . 259
—— detachment of retina . . . . . 135
—— glioma of both retinæ . . . . . 64
—— connective tissue in vitreous . . . . . 141
—— central choroiditis . . . . . 140, 141

LAWFORD (J. B.), naevus of choroid . . . . . 207
—— detachment of retina . . . . . 135
—— glioma of both retinæ . . . . . 64
—— connective tissue in vitreous . . . . . 141
—— central choroiditis . . . . . 140, 141

MCKEOWN (D.), on the prevention of blindness . . . . . 31

MORTON (A. Stanford), atrophy of choroid . . . . . 142
MULES (P. H.), evisceration of globe . . . . . 209

Nævus of choroid (J. B. Lawford) . . . . . 135
—— spontaneous disappearance of diabetic cataract . . . . . 135
—— detachment of retina with retention of vision . . . . . 147
—— amblyopia from bisulphide of carbon . . . . . 149
—— cocaine . . . . . 217
—— irido-cyclitis with meningitis in a child . . . . . 101

Night blindness (W. A. Frost) . . . . . 123

Opacity nerve fibres (G. Hartridge) . . . . . 177
Ophthalmia neonatorum, deputation with regard to . . . . . 31
Ophthalmic models (Priestley Smith) . . . . . 209
Ophthalmitis, reflex (J. Hutchinson, F.R.S.) . . . . . 71
—— discussion on (H. Power) . . . . . 76
—— ——— (Spencer Watson) . . . . . 78
—— ——— (Henry D. Noyes) . . . . . 79
—— ——— (E. Nettleship) . . . . . 84
—— ——— (P. H. Mules) . . . . . 89
—— ——— (W. A. Brailey) . . . . . 90
<table>
<thead>
<tr>
<th>Condition/Procedure</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ophthalmitis, reflex, President's reply</td>
<td>91</td>
</tr>
<tr>
<td>Optic nerves in intra-cranial disease (Walter Edmunds and J. B. Lawford)</td>
<td>184</td>
</tr>
<tr>
<td>—— neuritis, &amp;c. (W. A. Brailey)</td>
<td>178</td>
</tr>
<tr>
<td>Paralysis of third nerve, with migraine (Simeon Snell)</td>
<td>193</td>
</tr>
<tr>
<td>Power (H.), black cataract</td>
<td>111</td>
</tr>
<tr>
<td>Proptosis, optic atrophy, and anosmia (R. Marcus Gunn)</td>
<td>180</td>
</tr>
<tr>
<td>Retina, detachment of (W. Lang)</td>
<td>135</td>
</tr>
<tr>
<td>—— —— (W. A. Brailey)</td>
<td>113</td>
</tr>
<tr>
<td>—— —— with retention of vision (E. Nettleship)</td>
<td>133</td>
</tr>
<tr>
<td>Retinitis albuminurica (Henry Eales)</td>
<td>126</td>
</tr>
<tr>
<td>Rupture of eyeball (W. H. Jessop)</td>
<td>199</td>
</tr>
<tr>
<td>Smith (Priestley), ophthalmic models</td>
<td>209</td>
</tr>
<tr>
<td>Snell (Simeon), paralysis of third nerve, with migraine</td>
<td>193</td>
</tr>
<tr>
<td>Sympathetic disease (W. A. Brailey)</td>
<td>99</td>
</tr>
<tr>
<td>Tension, persistent diminished (W. A. Brailey)</td>
<td>106</td>
</tr>
<tr>
<td>Tubercle of iris and choroid (W. H. Jessop)</td>
<td>55</td>
</tr>
<tr>
<td>Tuberculosis of cornea and iris (A. H. Benson)</td>
<td>47</td>
</tr>
<tr>
<td>Vitreous, connective tissue in (W. Lang)</td>
<td>141</td>
</tr>
<tr>
<td>Watson (W. Spencer), intra-ocular gumma in inherited syphilis</td>
<td>56</td>
</tr>
</tbody>
</table>