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OF THE
OPHTHALMOLOGICAL SOCIETY
OF THE
UNITED KINGDOM
VOL. XXIX
SESSION 1908-1909

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With this volume is enclosed a list of Additions to the Library.

LONDON
J. & A. CHURCHILL
7. GREAT MARLBOROUGH STREET
1909
TRANSACTIONS

OF THE

OPHTHALMOLOGICAL SOCIETY

OF THE

UNITED KINGDOM

VOL. XXIX

SESSION 1908–1909

WITH

LIST OF OFFICERS, MEMBERS, ETC.

LONDON

J. & A. CHURCHILL

7, GREAT MARLBOROUGH STREET

1909
IN EXCHANGE.

Annales d'Oculistique.
Annali di Ottalmologia.
Annals of Ophthalmology, St. Louis, Mo., U.S.A.
Archiv für Ophthalmologie, Von Graef.
Archives de Neurologie, Charcot.
Archives d'Ophthalmologie, Lapersonne, Landolt, Gayet, et Badal.
Archives of Ophthalmology, Knapp and Schweigger.
Bericht der ophthalmologischen Gesellschaft, Heidelberg.
Brain.
Bulletins de la Société française d'ophtalmologie.
Centralblatt für praktische Augenheilkunde, Hirschberg.
Klinische Monatsblätter für Augenheilkunde, Axenfeld u.
Uhthoff.
Ophthalmic Record, Chicago.
Recueil d'Ophthalmologie.
Revue générale d'Ophthalmologie.
Royal London Ophthalmic Hospital Reports.
Transactions of the American Ophthalmological Society.
NOTICE.

The present volume comprises the proceedings of the Ophthalmological Society of the United Kingdom during its Twenty-ninth Session, October, 1908, to July, 1909.

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.

11, Chandos Street,
Cavendish Square, W.

October, 1909.
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OFFICERS AND COUNCIL
OF THE
Ophthalmological Society of the United Kingdom
ELECTED AT

President.
G. A. BERRY.

Vice-Presidents.
W. H. H. JESSOP.
R. A. Reeve (Toronto).
Priestley Smith (Birmingham).
Gustavus Hartridge.
Joseph Nelson (Belfast).
James Taylor, M.D.
J. C. Uthhoff (Brighton).
R. Marcus Gunn.
J. Tatham Thompson (Cardiff).

Treasurer.
J. B. Lawford.

Librarian.
W. T. Holmes Spicer.

Council.

HAROLD GRIMSDALE.
W. T. Lister.
C. Devereux Marshall.
Cyril H. Walker (Bristol).
Arnold Lawson.
William G. Laws (Nottingham).
A. Maitland Ramsay (Glasgow).
F. E. Batten, M.D.
E. Brewerton.
J. H. Parsons.
C. H. Usher (Aberdeen).
W. W. Sinclair (Ipswich).

Secretaries.
J. Herbert Fisher.
E. Farquhar Buzzard, M.D.
PRESIDENTS OF THE SOCIETY
(FROM ITS FORMATION).

ELECTED
1880 SIR WILLIAM BOWMAN, Bart., LL.D., F.R.S.
1883 SIR JONATHAN HUTCHINSON, F.R.S.
1886 JOHN WHITAKER HULKE, F.R.S.
1889 JOHN HUGHLINGS JACKSON, M.D., LL.D., F.R.S.
1890 HENRY POWER.
1893 D. ARGYLL ROBERTSON, M.D.
1895 EDWARD NETTLESHIP.
1897 SIR HENRY R. SWANZY.
1899 SIR ANDERSON CRITCHETT, Bart., C.V.O.
1901 DAVID LITTLE, M.D.
1903 SIR JOHN TWEEDY.
1905 PRIESTLEY SMITH.
1907 MARCUS GUNN.
1909 G. A. BERRY, M.D.

TRUSTEES FOR THE SOCIETY.
JOHN ABERCROMBIE, M.D.
W. A. BRAILEY.
R. MARCUS GUNN.
EDWARD NETTLESHIP.
SEYMOUR J. SHARKEY, M.D.

TRUSTEES OF THE NETTLESHIP PRIZE FUND.
JOHN ABERCROMBIE, M.D.
R. MARCUS GUNN.
J. B. LAWFORD.

BOWMAN LECTURERS.

ELECTED
1884 SIR JONATHAN HUTCHINSON, F.R.S.
1885 J. HUGHLINGS JACKSON, M.D., LL.D., F.R.S.
1886 PROF. ZEHENDER (Rostock).
1887 HENRY POWER.
1888 SIR HENRY R. SWANZY.
1889 PROF. HANSEN GRUT (Copenhagen).
1890 J. W. HULKE, F.R.S.
1892 PROF. LEBER (Heidelberg).
1893 T. PRIDGIN TEALE, F.R.S.
1895 SIR W. R GOWERS, M.D., F.R.S.
1896 PROF. SNELEN (Utrecht).
1898 PRIESTLEY SMITH.
1900 R. MARCUS GUNN.
1902 PROF. E. FUCHS (Vienna).
1904 F. W. MOTT, M.D., F.R.S.
1907 PROF. SATTLER (Leipsig).
1909 EDWARD NETTLESHIP.

EDWARD NETTLESHIP PRIZE.
1904 PRIESTLEY SMITH.
1907 JOHN HERBERT PARSONS.
1909 EDWARD NETTLESHIP.
LIST OF MEMBERS OF THE SOCIETY.

Honorary Members.

Professor Zehender, Rostock.
Professor Leber, Heidelberg.
Professor Fuchs, Vienna.
Professor Sattler, Leipzig.

EXPLANATION OF ABBREVIATIONS.

O.M.—Original Member.
Pres.—President.
V.-P.—Vice-President.
T.—Treasurer.
*.—Denotes Resident Life Members.
†.—Denotes Non-Resident Life Members.

GENERAL LIST OF MEMBERS.

ELECTED
1905 Allen, R., 5, Queen's Elms, Belfast.
1907 Allport, Wilfred, M.B., 133, Edmund Street, Birmingham.
1899 †Amenabar, Julio Daniel, Guayacan, Coquimbo, Chili.
1887 *Anderson, Tempest, M.D., 17, Stone-gate, York.
1903 Anderson, William, 11, Upper Brook Street, W.
1883 Andrews, A. G., Carlton House, Moss Lane East, Manchester.

1907 Archer, Major S. A. Arthur, R.A.M.C., Jullundur, Punjab, India.
O.M. Archer, T. Brittin, 29, Nottingham Place, W.

1891 Armstrong, Hugh, Tamworth, New South Wales.

1895 Attlee, John, M.B., 65, Grosvenor Street, W.

1903 Ballantyne, A. J., 11, Sandyford Place, Glasgow.

1907 Bardi, Dosabhai Rastamji, Tardeo, Albert Building Fort, Bombay, India.

1907 Bardsley, Percy C., M.B., 36, Dean Road, Willesden Park, N.W.

1900 Barker, A. J. Glanville, P.M.O., Kuching, Sarawak, Borneo.
O.M. *Barlow, Sir Thomas, Bart., K.C.V.O., M.D., 10, Wimpole Street, W. (C. 1880-81. V.-P. 1894-7.)

1889 *Barrett, James W., M.D., 34, Collins Street East, Melbourne, Australia.

1883 Barton, J. Kingston, 14, Ashburn Place, Courtfield Road, S.W.


1892 Batten, Rayner D., M.D., 9, Wimpole Street, W. (C. 1905-8.)

1888 *Beaumont, W. M., 4, Gay Street, Bath. (C. 1901-4.)

1909 Bell, T. Herbert, Canadian Government Office, 17, Victoria Street, S.W.

1891 *Bennett, Alfred H., North Terrace (opposite Government House), Adelaide, South Australia.

1897 Bennett, H. Percy, 12, Victoria Square, Newcastle-on-Tyne.

1905 Bennett, F. D., M.D., 20, St. James' Place, S.W.
O.M. Benson, A. H., M.D., 42, Fitzwilliam Square, Dublin. (C. 1888-91. V.-P. 1901-3.)

1905 Bergin, W. M., M.B., B.S., 53, Devonshire Street, Portland Place, W.
<table>
<thead>
<tr>
<th>Year</th>
<th>Name</th>
<th>Address</th>
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<td>1899</td>
<td>Bickerton, R. E.</td>
<td>137, Harley Street, W.</td>
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<tr>
<td>1881</td>
<td>*Bickerton, T. H.</td>
<td>88, Rodney Street, Liverpool</td>
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<td>1905</td>
<td>Bird, J. W.</td>
<td>15, Downside Crescent, Hampstead, N.W.</td>
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<td>1892</td>
<td>Black, John Wilson</td>
<td>46, Academy Street, Inverness</td>
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<td>1898</td>
<td>Blair, Charles Samuel</td>
<td>14, Stratford Place, W.</td>
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<td>1895</td>
<td>*Boxar, Thomson, M.D.</td>
<td>114, Via del Babuino, Piazz di Spagna, Rome</td>
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<td>1907</td>
<td>Bond, C. Shaw</td>
<td>7, Broadlands Road, Highgate</td>
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<td>1885</td>
<td>Bower, Ernest Dykes</td>
<td>Elton House, Gloucester</td>
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<td>1897</td>
<td>Bower, John A., M.B.</td>
<td>18, Royal Crescent, Cheltenham</td>
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<td>1903</td>
<td>Bradburne, A. A.</td>
<td>61, Hoghton Road, Southport</td>
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<tr>
<td>1905</td>
<td>Brailey, A. R.</td>
<td>11, Old Burlington Street, W.</td>
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<tr>
<td>O.M.</td>
<td>Brailey, W. A., M.D.</td>
<td>11, Old Burlington Street, W.</td>
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<td>1897</td>
<td>Breuer, August, M.D.</td>
<td>10, Finsbury Circus, E.C.</td>
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<td>1899</td>
<td>Brewerton, Elmore</td>
<td>84, Wimpole Street, W.</td>
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<tr>
<td>1903</td>
<td>Brinton, A. G., Box 5852</td>
<td>Johannesburg, South Africa</td>
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<td>1891</td>
<td>Bristowe, Hubert Carpenter</td>
<td>Wrinton, Somerset</td>
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<td>1886</td>
<td>Bronner, Adolph, M.D.</td>
<td>33, Manor Row, Bradford</td>
</tr>
<tr>
<td>1901</td>
<td>*Brooks, R. Philip</td>
<td>24, Wimpole Street, W.</td>
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<tr>
<td>1909</td>
<td>Brouchá, B. B., care of Thomas Cook and Sons</td>
<td>Ludgate Circus</td>
</tr>
<tr>
<td>O.M.</td>
<td>Browne, Edgar A., M.D.</td>
<td>39, Rodney Street, Liverpool</td>
</tr>
<tr>
<td>1898</td>
<td>Browne, James M., M.B.</td>
<td>22, St. Patrick's Hill, Cork</td>
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<tr>
<td>1895</td>
<td>Browne, J. Walton, M.D.</td>
<td>10, College Square North, Belfast</td>
</tr>
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</table>
ELECTED

1901 Buchanan, Leslie, 17, Sandyford Place, Glasgow.
1907 Buckland, Francis, M.D., Moorland Court, Poole Road, Bournemouth.
1889 Bullar, John F., M.B., 7, Carlton Crescent, Southampton.
1907 Burdon-Cooper, J., M.D., 22, The Circus, Bath.
O.M. Burnham, G. H., M.B., 157, Simcoe Street, Toronto, Canada.
1909 Butler, Thomas H., M.D., B.Ch., 2, The Quadrant, Coventry.
1902 Buzzard, E. Farquhar, M.D. (S.) 78, Wimpole Street, W. (C. 1908-9.)
O.M. Buzzard, Thomas, M.D., 74, Grosvenor Street, W. (V.-P. 1888-91. C. 1881-2.)
1897 Byers, W. Gordon M., M.D., 346, Mountain Street, Montreal.
1892 Caiger, Herbert, M.B., Burghersdorp, Cape Colony.
1905 Campbell, C. A., 93, Bloor Street W., Toronto.
1891 Campbell, E. Kenneth, M.B., 23, Wimpole Street, W.
1887 Cant, W. E., M.D., British Ophthalmic Hospital, Jerusalem.
1882 *Cant, W. J., White Cross, Lincoln. (C. 1898-1901.)
1891 Cargill, L. Vernon, 31, Harley Street, W. (C. 1905-8.)
1905 Carpenter, George, M.D., 12, Welbeck Street, W.
1905 Carruthers, J. F., 2, Clifton, Guernsey.
1895 Cartwright, E. H., M.D., Myskyns, Ticehurst, Sussex.
1885 Chesshire, Arthur Edwin, 55, Darlington Street, Wolverhampton.
1885 Clarke, Ernest, M.D., B.S., 3, Chaudois Street, Cavendish Square, W. (C. 1893-6.)
1905 Clements, E. C., 3, Lindum Road, Lincoln.
1902 Coats, George, M.D., 2, Harley Place, W.
1907 Cobbledick, Arthur Stanley, M.D., B.S.Lond., 368, Brixton Road, S.W.
Elected

1901 Cole-Baker, Lyster, M.D., Bayfield, Kent Road, Southsea.


1886 Collins, Sir W. J., M.S., M.D., B.Sc., 1, Albert Terrace, Gloucester Gate, Regent's Park, N.W.

1901 Cooke, Arthur, 69, Bridge Street, Cambridge.

1901 Cooper, Ludford, 19, Victoria Street, Rochester, Kent.

1909 Coory, Edward A., M.D., 39, Woburn Place, W.C.

1894 Coote, Patrick, M.D., 73, St. Ann Street, Quebec, Canada.

1904 Corbett, W J., 12, Cambridge Gardens, Kilburn, N.W.

1898 Coulter, Robert J., M.B., 11, Clytha Park Road, Newport, Mon.

O.M. Couper, John, 80, Grosvenor Street, W. (C. 1881-2. V.-P. 1895-8.)

1895 Courtenay, J. D., M.D., Ottawa, Canada.

1909 Craig, James A., M.B., 11, University Square, Belfast.

1901 Crawley, F. C., M.D., 41, Lower Baggot Street, Dublin.

1907 Cree, Robert Ewart, M.D., 40, Jesmond Road, Newcastle-on-Tyne.

1899 Cresswell, B. P. Skeffington, M.B., B.S., 24, Windsor Place, Cardiff.

1904 Cridland, A. Bernard, 11, Waterloo Road, Wolverhampton.


1898 Critchley, Harry G., M.D., 89, Park Lane, Croydon.

1906 Crocket, A. Pierce, 50, King's Square, St. John's, New Brunswick, Canada.

1900 *Cropper, John, Mount Ballan, Chepstow (Mon.).
ELECTED

1881 Cross, F. R., Worcester House, Clifton, Bristol. (V.-P. 1898-1901. C. 1891-4.)
1902 Cruise, R. R., 85, Harley Street, W.
1904 Cunningham, H. H. B., 69, University Road, Belfast.
1905 Cunningham, J. F., Sec Tor, Axminster, Devon.
1909 Daggan, J. N., C.S. Ophthalmic Hospital, Byculla, Bombay.
1905 Dani, R. W., 37, Samuel Street, Wadagadi, Mandoi, Bombay.
1907 Davidson, Alex. Dyce, 25, Hanover House, Regent’s Park, N.W.
1884 Davidson, James McKenzie, M.B., 76, Portland Place, W. (C. 1892-5.)
1903 Davies, David, 18, Mount Sion, Tunbridge Wells.
1908 Davies, D. L., M.S., M.D., 124, Newport Road, Cardiff.
1889 Davis, G. C., 3, Hyde Park Terrace, 173, Liverpool Street, Sydney, New South Wales.
1897 Dawnay, Arch. Hugh Payan, 126, Harley Street, W.
1903 Dean, C. W., Lindow Square, Lancaster.
O.M. Dent, Clinton Thomas, 61, Brook Street, W.
1903 Dickson, R. H., Nelson Place, Newcastle-under-Lyne, Staffordshire.
1881 Dixon, W. E., Oulton Lodge, Oulton Broad, Lowestoft.
1907 Dombrain, Ernest A., 205, Macquarie Street, Sydney, N.S.W.
1905 Dorrell, E. A., 1, Lyncroft Gardens, West Hampstead, N.W.
1887 Doyne, Robert W., 30, Cavendish Square, W.; and 53, Broad Street, Oxford. (C. 1892-5. V.-P. 1904-7).
Elected

1881 *Drake-Brockman, E. F., 18a, Hanover Square, W. (C. 1895-8.)

1889 †Drake-Brockman, Herbert E., I.M.S., c/o Thomas Cook and Son, Ludgate Circus, E.C.

1895 Du Boulay, H. H., 2, Royal Terrace, Weymouth.

1886 Dunn, Hugh Percy, 54, Wimpole Street, Cavendish Square, W.

O.M. Eales, Henry, 7, Newhall Street, Birmingham. (C. 1890-3. V.-P. 1900.)

1900 Eason, Herbert L., M.B., Guy’s Hospital, S.E.

O.M. *Edmunds, Walter, M.D., 2, Devonshire Place, Portland Place, W. (C. 1885-8.)

1902 †Elliot, Robert Henry, Major I.M.S., M.D., Sc.D., c/o Messrs. Grindlay, 54, Parliament Street, S.W.

1903 Ellis, W. F., c/o Messrs. Holt, 3, Whitehall Place, S.W.

1908 Emmerson, Herbert H., 275, Glossop Road, Sheffield.

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

1887 Ensor, Henry C., 23, Windsor Place, Cardiff.

1900 Evans, John Jameson, M.B., 85, Edmund Street, Birmingham.

1900 †Evans, Thomas, 211, Macquarie Street, Sydney, New South Wales.

1902 Evershed, A. R. F., 13, Harley Street, W.


1888 Fergus, A. Freeland, M.B., 22, Blythswood Square, Glasgow. (C. 1905-8.)

O.M. †Ferguson, H. L., Dunedin, New Zealand.

1894 †Fischer, E. C., M.D., Cairo, Egypt.

1904 Fisher, Major J., I.M.S., c/o Messrs. Grindlay and Co., 54, Parliament Street, S.W.

Elected

O.M. Fitzgerald, C. E., M.D., 27, Upper Merrion Street, Dublin. (V.P. 1882-5. C. 1880-1.)


1889 Flemming, Percy, 31, Wimpole Street, W. (C. 1901-4.)

1892 Folker, Herbert Henry, Hanley, Staffordshire.

1886 Ford, A. Vernon, South View Lodge, Kent Road, Southsea.


1895 Galloway, A. Rudolf, M.B., 250, Union Street, Aberdeen, N.B.

1885 †Gardner, John J., M.D., 128, Bishop Street, Montreal, Canada.

1889 Gibbs, Alfred N. G., 52, Whiteladies Road, Clifton.

1902 †Gibson, J. Lockhart, M.D., Wickham Terrace, Brisbane, Australia.

O.M. Glascott, C. E., M.D., 21, St. John Street, Manchester. (C. 1896-9. V.-P. 1902-5.)

1908 Glenny, Elliott Thornton, M.B., B.S., 61, Effingham Road, Bristol.

1885 †Godfrey, Alfred Charles, St. Heliers House, St. Heliers, Jersey.

1899 Goldsmith, G. Harvey, M.B., Bedford.

1907 Goulden, Charles Bernard, 9, Richmond Park Road, Clifton.

1904 Gowans, Thos., M.B., 4, Abbotsford Terrace, Newcastle-on-Tyne.


1897 Granger, F. M., 18, Nicholas Street, Chester.

1895 †Grant, H. Y., M.D., 414, Delaware Avenue, Buffalo, U.S.

1895 †Gray, James, M.D., 119, Robert Street, Toronto, Canada.

1887 Green, Edwin Collier, 27, Friar Gate, Derby. (C. 1903-4.)
Elected

1900 Green, F. W. Edridge, M.D., Hendon Grove, Hendon, N.W.
1903 Greene, Arthur, 4, Theatre Street, Norwich.
1896 Grey-Edwards, Henry, M.D., Bank Place, Bangor, N. Wales.
1895 Griffin, W. Watson, M.B., 68, Brunswick Place, Hove, Brighton.
1885 *Griffith, A. Hill, M.D., 17, St. John Street, Manchester. (V.-P. 1905-8. C. 1893-6.)
1894 Grimsdale, Harold, M.B., (C.), 3, Harley Place, W. O.M.
1899 Grossmann, K. A., 70, Rodney Street, Liverpool.
1908 Gunning, C. J. H., 13, Thurloe Place, South Kensington, S.W.
1898 Guthrie, Leonard G., M.D., 15, Upper Berkeley Street, W.
1887 *Habershon, Samuel Herbert, M.D., 88, Harley Street, W. (S. 1894-7. C. 1897-1900.)
1889 Hailes, C. D. G., M.D., Hawkesdale, 27, Alma Road, Clifton.
1885 †Haines, Humphrey, Auckland, New Zealand.
1902 †Halliday, J. C., Macquarie Street, Sydney, N.S.W.
1900 Hallidie, Andrew, M.B., Avondale, Chesterfield Road, Eastbourne, and 6, Warrior Square Terrace, St. Leonards-on-Sea.
1896 Hamilton, Robert J., 82, Rodney Street, Liverpool.
1878 †Hamilton, T. K., M.D., Wakefield Street, Adelaide, South Australia.
1901 Hancock, Edw. D., 10, Brock Street, Bath.
1899 Hancock, W. Ilbert, 27, Queen Anne Street, W.
1908 Hanna, Henry, 57, University Road, Belfast.
1901 Hanson, Reginald E., 5, Harley Street, W.
1900 Harman, N. B., M.B., 108, Harley Street, W.
ELECTED

1902 Harries-Jones, E., 16, Castilian Street, Northampton.
1886 Hartley, Robert N., M.B., 2, Clarendon Road, Leeds.
1882 *Hartridge, Gustavus (V.-P.), 12, Wimpole Street, W.
  (C. 1889-92, 1895-8. S. 1892-5.)
1905 †Harty, G. W., M.B., Ch.B., Wellington, New Zealand.
1899 Hawkes, C. S., Glencairn, Wickham Terrace, Brisbane, Queensland.
1901 Hawthorne, C. O., 63, Harley Street, W.
1908 Hay, Percival J., 3, Northumberland Road, Sheffield.
1892 Haydon, Frank, Apothecaries' Hall, Water Lane, Blackfriars, E.C.
1898 Hayes, George C., 24, Park Square, Leeds.
1899 Henderson, Edward E., M.B., 6, Lower Berkeley Street, Portman Square, W.
1896 Henry, R. Wallace, M.D., 6, Market Street, Leicester.
1905 Hepburn, Malcolm Langton, M.D., B.S., F.R.C.S., 66, Wimpole Street, W.
1897 Herbert, Lt.-Col. Herbert, I.M.S., Castle Grove, Nottingham.
1887 *Hern, John, M.D., Summercote, Darlington.
1905 Hewkley, F., 19, Lower Seymour Street, Portman Square, W.
1895 Hickman, H. R. Belcher, M.B., 5, Harley Street, W.
1895 O. M. Higgen, Charles, 52, Brook Street, W. (C. 1880-3.
  V.-P. 1901-4.)
1903 Hill, Francis R., 62, Warwick Road, Carlisle.
1888 *Hinell, J. S., M.B., 62, Garland Street, Bury St. Edmunds.
1907 Hinshelwood, James, 26, Woodside Place, Glasgow.
1905 Hird, R. B., M.B., 81, Edmund Street, Birmingham.
1899 Hodday, James, M.B., Beverley House, Colney Hatch Lane, N.
1886 †Hodge, Rev. Sydney Rupert, The Wesleyan Mission Men's Hospital, Hankow, China.
ELECTED

1897 Hogg, G. H., M.D., 95, George Street, Launceston, Tasmania.

1889 Holthouse, Edwin H., M.B., 6, Devonshire Street, Portland Place, W.

1907 Hormasjee, Jehangir, c/o Thos. Cook and Son, Ludgate Circus, E.C., and Khelwadi, 7th Street, Bombay.

1905 Hosford, J. S., 20, St. James' Place, St. James' Street, S.W.

1893 †Howe, Lucien, 183, Delaware Avenue, Buffalo.

1908 Hudson, A. C., M.D., Royal London Ophthalmic Hospital, City Road, E.C.

1884 †Hudson, Ernest, Central Gaol, Benares, N.W.P., India.

1889 †Hughes, Samuel II., 169, Macquarie Street, Sydney, New South Wales.

1893 †Hughes, Wilfrid Kent, M.B., 102, Collins Street, Melbourne.

1908 Hunter, George, 31, Bridge Street, Inverness.


1901 Inman, Wm., M.B., 55, Elm Grove, Southsea.

1906 Irving, Robert J., Stocksfield-on-Tyne.

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

O.M. Jackson, J. Hughlings, M.D., F.R.S., 3, Manchester Square, W. (Pres. 1889-90. V.-P. 1880-2, 1890-3.)

1898 James, George Brookbank, 5, Harley Street, W.

1888 James, J. T., M.D., 108, Harley Street, W.

1908 James, Robert Ruston, 119, Oxford Street, W.

1905 Jaques, R., 7, North Parade, Penzance.

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

1908 Jeremy, H. Rowe, 8, Chingford Road, Walthamstow, Essex.
ELECTED

1882  Johnson, G. Lindsay, M.B., 55, Queen Anne Street, Cavendish Square, W.
1888  Johnston, Geo. D., Georgia Street, Vancouver, British Columbia.

O.M.    Jones, Evan, Ty-mawr, Aberdare, Glamorganshire.
1898  *Jones, George, M.B., 8, Church Terrace, Lee, S.E.
O.M.    Jones, H. Macnaughton, M.D., 131, Harley Street, W.
1897  Jones, Hugh E., 19, Rodney Street, Liverpool.
1894  *Jones, R. H., M.B., B.S., 209, Macquarie Street, Sydney, New South Wales.
1905  Juler, F. A., 24, Cavendish Square, W.
1899  Keeling, G. S., M.D., Attleborough, Norfolk.
1900  *Kelsall, H. T., M.D., 1, Devonshire Terrace, Perth, W. Australia.
1888  *Kenny, Augustus Leo, M.B., 87, Collins Street, Melbourne, Victoria, Australia.
1904  Killick, Chas., 3, Marsham Street, Maidstone.
1895  Knaggs, Robert Lawford, M.D., 27, Park Square, Leeds.
1881  *Knaggs, S. T., M.D., 1, Lyons Terrace, Hyde Park, Sydney, N.S.W.
Elected


1896 Lawson, Arnold, M.D., (C.), 12, Harley Street, W.

1895 †Lea, J. Augustus, M.B., Grahamstown, Cape Colony, South Africa.

1885 †Le Cronier, Hardwick, St. Heliers, Jersey.

O.M. Lediard, H. A., M.D., 26, Lowther Street, Carlisle. (C. 1900-1.)

1885 Lee, Charles G., 11, Princes Avenue, Liverpool.

1904 †Lee, W. A., Lt.-Col. I.M.S., c/o Mrs. Lee, Heaton Hall, Newcastle-on-Tyne.

1903 Levy, A., M.D., 67, Wimpole Street, W.

1907 Lindsay, W. J., 84, Herne Hill, S.E.

1896 Lister, W. T., M.B., (C.), 24, Devonshire Place, W.

1902 Little, Andrew, 114, Manningham Lane, Bradford, Yorks.

1892 Lodge, Samuel, jun., M.D., 28, Manor Road, Bradford.

1903 †Lückhoff, James, M.D., Rhodes Buildings, St. George's Street, Capetown.

1883 Lunn, J. R., Resident Medical Officer, New Marylebone Infirmary, Rackham Street, Ladbroke Grove Road, W. (C. 1892-5.)

1899 Lyle, H. Willoughby, M.D., Eversley, Elmfield Road, Bromley, Kent; and 39, Hertford Street, Mayfair, W.

1900 MacCallan, Arthur Ferguson, M.B., Turf Club, Cairo, Egypt.

1905 Macdonald, J. G., Imperial Chambers, Dee Street, Invercargill, New Zealand.

1902 Mackay, D. Matheson, M.D., 48, Beverley Road, Hull.


1889 †Mackenzie, F. Wallace, M.B., 139, Upper Willis Street, Wellington, New Zealand.
1889 MacLehose, Norman M., M.B., 18, Harley Street, Cavendish Square, W. (C. 1902-5.)
1897 †MacLennan, Duncan N., M.D., 126, Bloor Street West, Toronto.
1892 †MacLeod, Charles G., M.B., 157, Macquarie Street, Sydney, N.S.W.
1881 †Maconachie, G. A., Brigade Surgeon Lieutenant-Colonel, M.D., 33, Queen's Road, Aberdeen.
1899 Maddox, Ernest E., M.D., Glenartney, Poole Road, Bournemouth. (C. 1904-6.)
1883 †Maher, W. Odillo, M.D., Craignish, 185, Macquarie Street, Sydney, N.S.W.
1899 †Marché, Charles, B.A., M.D., 4, Piazza Regina, Valletta, Malta.
1901 †Manning, Leslie S., Christchurch, New Zealand.
1909 Marchant, E. Lachlan, Paddington Green Children's Hospital, London, W.
1904 Markus, Charles, M.D., 28, Wimpole Street, W.
1883 †Marlow, Frank William, M.D., 401, Montgomery Street, Syracuse, New York State, U.S.A.
1907 Marshall, James Cole, M.D., 36, Albion Street, Hyde Park, W.
1888 †Martin, Albert, M.D., Wellington, New Zealand.
1905 Massey, A. Yale, M.D., 28, Queen Road, St. John's, Newfoundland.
1884 Maxwell, Patrick William, M.D., 19, Lower Baggot Street, Dublin. (C. 1900-2.)
1904 May, H. J., M.B., B.C., Naini Tal, College Place, Southampton.
Elected

1902 Mayou, Stephen, 46, Weymouth Street, W.
1890 *McGillivray, Angus, M.D., 23, South Tay Street, Dundee, N.B. (C. 1902-5.)
O.M. McHardy, M. M., 5, Savile Row, W. (C. 1887-90.)
1895 **McIntosh, J. R., M.D., 40, Coburg Street, St. John, New Brunswick, Canada.
1895 McKenzie, H. V., M.D., Elmbank, Abbey Road, Torquay.
1902 McMullen, W. H., 4, Chandos Street, Cavendish Square, W.
1904 McNab, Angus, 31, New Cavendish Street, W.
O.M. Meighan, T. S., M.D., 37, Elmbank Crescent, Glasgow.
1901 Menzies, J. A., 9, Castle Hill Avenue, Folkestone.
1897 Miller, G. Victor, M.B., 2, Barrington Crescent, Stockton-on-Tees.
1899 Miller, Herbert Percy, M.D., 100, Stoke Newington Road, N.
1881 **Milles, W. Jennings, care of Drs. Henderson and Macleod, Shanghai, China.
1897 **Minnes, Robert Stanley, M.D., 127, Metcalfe Street, Ottawa, Ontario.
1901 Montgomery, Robert J., M.B., 28, Upper Fitzwilliam Street, Dublin.
1896 Mooney, Herbert C., M.B., 22, Lower Baggot Street, Dublin.
1908 Moore, Robert F., M.B., St. Bartholomew's Hospital, E.C.
1904 Morrice, G. G., 17, Royal Terrace, Weymouth.
1898 Mott, F. W., M.D., F.R.S. 25, Nottingham Place, W. (V.-P. 1904-7.)
1890 Mowat, Daniel, M.D., 123, Stamford Hill, N.
1908 Moxon, Frank, Cunningham Park Road, Wealdstone. Middlesex.
Elected

1891 Myddelton-Gavey, Edward Herbert, Stanton Prior Meads, Eastbourne.


1909 Nell, Andreas, Victoria Eye and Ear Hospital, Colombo, Ceylon.

O.M. *Nelson, Joseph, M.D., (V.-P.), 29, Wellington Place, Belfast. (C. 1893-5.)


1881 Nicholson, A., 30, Brunswick Square, Brighton.

1895 *Ogilvie, F. Menteith, M.B., The Shrubbery, 72, Woodstock Road, Oxford.

1895 Ogilvy, Alexander, M.B., Lemy, Clifton Park, Clifton, Bristol.

1889 †O'Kinealy, Frederick, Major, I.M.S., care of Messrs. King, Hamilton and Co., 7, Hare Street, Calcutta.

1899 Oldmeadow, Lloyd J. H., Kineton, Warwickshire.

1899 Ormond, Arthur Wm., 7, Devonshire Place, W.


1890 †Osborne, A. B., M.D., 46, McNab Street South, Hamilton, Canada.

O.M. Owen, D. C. Lloyd, 41, Newhall Street, Birmingham. (V.-P. 1891-4.)

1905 Owen, S. A., M.B., 50, Harrington Gardens, S.W.

1890 †Palmer, L. Loran, M.D., 42, St. George Street, Toronto, Ontario, Canada.

1894 *Parker, Herbert, 7, Bloomsbury Place, W.C.

1899 Parker, Herbert George, 13, Chorley New Road, Bolton.

1900 Parsons, John Herbert (C.), 27, Wimpole Street, W.

1887 †Patel, D. H., Bai Hirabai, B. J. Charitable Dispensary, Tardeo, Bombay.
1907  Paterson, James V., 5, Castle Street, Edinburgh.

1900  Patkar, Bhagvant Sakharam, Carnac Road, Kalkadeir Post, Bombay, India.

1902  Paton, Leslie, 1, Spanish Place, Manchester Square, W.

1907  Pearson, Maurice G., M.B., Cleveland Moore Road, Durban, Natal.

1888  Percival, Archibald Stanley, M.B., B.Ch., 25, Ellison Place, Newcastle-on-Tyne. (C. 1906-9.)

1891  Perry, A., Surgeon-Major, Principal Civil Medical Officer, Ceylon.

1889  *Perry, Francis F., Lahore Medical College, Punjab, India.

1889  Phillips, T., 126, Harley Street, W.

1895  Pickard, Ransom, M.D., 31, East Southernhay, Exeter.

1909  Pike, Norman H., 10, York Terrace, St. George’s Road, Cheltenham.

1900  *Pockley, Francis Antill, M.B., 227, Macquarie Street, Sydney, N.S.W.

1903  Pollock, W. B. Inglis, 276, Bath Street, Glasgow.

1896  *Pooley, G. H., 89, Priory Road, Hampstead, N.W.

1894  *Pope, R. J., M.D., Box 407, G.P.O., Sydney, N.S.W.

1900  Pope, Thomas Henry, M.D., B.Sc., Blairmore, Northdown Avenue, Cliftonville, Margate.

1902  Potter, Bernard E., 5S, Park Street, W.

1903  *Potts, George, County Ophthalmic Hospital, Maidstone.


1899  Price, Henry J., Maldon, Essex.

1882  Prichard, Arthur William, 6, Rodney Place, Clifton.

1908  Pridmore, Walter G., Major I.M.S., c/o Grindlay and Co., 54, Parliament Street, S.W.

1903  Pritchard, Eric L., M.D., 70, Fairhazel Gardens, N.W.
Elected

1892 Pronger, Charles Ernest, Litchdon, Harrogate.
1909 Prowse S. Willis, 706, Union Bank Buildings, Winnipeg, Canada.
O.M. Purves, W. Laidlaw, 20, Stratford Place, Oxford Street, W.
1905 Ramage, J., M.D., 6, Gloucester Road, Regent’s Park, N.W.
1889 Ramsay, A. Maitland, M.D., (C.), 15, Woodside Place, Glasgow.
1899 Read, E. I., Government Medical Officer, Trinidad, West Indies.
1881 †Reeve, R. A., M.D. (V.-P.), 22, Shuter Street, Toronto, Canada.
O.M. Reid, Thomas, M.D., 11, Elmbank Street, Glasgow. (V.-P. 1884-7.)
1885 Renton, James Crawford, M.D., 1, Woodside Terrace, Glasgow.
1891 Reynolds, Austin Edward, Highcroft, Shepherd’s Hill, Highgate, N.
1897 Richmond, R., M.D., 29, Lingsfield Road, Wimbledon, S.W.
1907 Ridge, E. Manners, New River House, Church Street, Enfield.
1892 Ridley, Nicholas C., M.B., 27, Horse Fair Street, Leicester.
1900 Riseley, Stanley, 387, Glossop Road, Sheffield.
1897 Rivers, W. H. R., M.D., 11, Queen Victoria Street, E.C.
1885 *Roberts, Edward, 23, St. John Street, Deansgate, Manchester.
1891 Robertson-Fullarton, Archibald Louis, M.B., C.M., 201, Bath Street, Glasgow.
O.M. Rockliffe, W. C., M.D., 17, Charlotte Street, Hull. (C. 1892-5. V.-P. 1900-3.)
Elected

1898 Roe, Arthur Legge, 43, Pryme Street, Hull.
1898 Roll, G. W., M.B., B.C., 7, Upper Wimpole Street, W.
1890 Rolston, John R., 14, The Crescent, Plymouth.
1882 †Roth, Reuter E., 42, College Street, Hyde Park, Sydney, New South Wales.
1893 *Rowan, John, M.B., 10, Woodside Crescent, Charing Cross, Glasgow, N.B.
1899 *Roxburgh, A. B., M.B., 7, Henrietta Street, W.
1881 †Rudall, J. T., 61, Spring Street, Melbourne, Australia.
1903 Rutherford, A. Freer, 19, Abbey Road, Barrow-in-Furness.
1888 †Sanders, Richard C., M.D., Loppington Hall, nr. Wem, Salop.
1909 Saunders, Edward H., 16, Torrington Square, W.C.
O.M. Savage, G. H., M.D., 26, Devonshire Place, W. (C. 1892-4.)
1900 †Sager, D. S., M.D., Brantford, Ontario, Canada.
1901 Scott, G. Melmoth, 16, Palace Chambers, Kalgoorlie, Australia.
1888 Scott, Kenneth, M.D., 7, Manchester Square, W.
1900 Scott-Heyliger, E. D., S9, Preston New Road, Blackburn.
1892 Shannon, John Rowlands, M.D., 25, West 36th Street, New York.
1894 Shaw, Cecil E., M.D., 29, University Square, Belfast. (C. 1906-9.)
Elected

1883 Shears, Charles H., 19, Upper Duke Street, Rodney Street, Liverpool. (C. 1900-3.)


1891 Sinclair, Walter William (C.), 3, Arcade Street, Ipswich.

1889 Smith, John, M.D., Brycehall, Kirkcaldy, N.B.

O. M. Smith, Priestley (V.-P.), 95, Cornwall Street, Birmingham. (V.-P. 1887-90; 1898-1901. C. 1883-6. Pres. 1905-7.)

1907 Smith, W. Harvey, M.D., Room 26, Canada Life Buildings, Winnipeg, Canada.

1903 Smyth, Ernest J., Maythorne, Epsom Road, Guildford.

1901 Snowball, Thomas, M.B., 83, Bank Parade, Burnley.

1889 Spencer, Matthew H., M.B., B.Ch., 92, Oxford Gardens, North Kensington, W.

1889 Spicer, Wm. T. Holmes, M.B. (L.), 5, Wimpole Street, Cavendish Square, W. (C. 1900-2.)

1905 Sprawson, F. C., M.B., 7, Imperial Terrace, Claremont Park, Blackpool.

1897 Square, James Elliot, 22, Portland Square, Plymouth.

1895 †Stamberg, A. C., M.B., 5, Windsor Crescent, St. Heliers, Jersey, Channel Islands.


1896 Stevenson, Edgar, M.D., 39, Rodney Street, Liverpool.

1904 Stevenson, John Simpson, Royal Exchange Buildings, Cathedral Square, Christchurch, New Zealand.

1905 Steward, E. S., 10, Princes’ Square, Harrogate.

1893 Stirling, Alexander Williamson, M.D., Atlanta, Georgia.

1887 †Stirling, J. W., M.B., 255, Mountain Street, Montreal, Canada.
ELECTED

O.M. Story, J. B., 6, Merrion Square North, Dublin. (C. 1885-8. V.-P. 1894-7.)

O.M. †Sturge, W. A., M.D., Icklingham Hall, Mildenhall, Suffolk.


1904 Sykes, Walter, 20, Fox Street, Fishergate, Preston.

1888 *Sym, William George, M.D. 12, Alva Street, Edinburgh. (C. 1906-9.)

1883 †Symons, Mark Johnston, M.D., North Terrace, Adelaide, South Australia.

1907 Tampi, K. R., Trivandrum, South India.


1903 Taylor, H. H., 36, Brunswick Square, Hove, Brighton.

1902 Taylor, Henry J., 44, St. George’s Road, Bolton.

1899 Taylor, Inglis, M.B., 16, Harley Street, W.


1889 Taylor, S. J., M.B., 44, Prince of Wales’ Road, Norwich. (C. 1904-7.)

1900 Teale, Michael A., 4, Park Square, Leeds.

1900 Thomas, Frank G., M.B., 22, Walter Road, Swansea.

1903 Thomas, R. Russell, 26, Windsor Place, Cardiff.

1895 Thompson, A. Hugh, M.D., 36, Weymouth Street, W.

1903 Thompson, A. Henry, 294, Romford Road, Forest Gate, E.

1885 Thompson, C. Sinclair, The Quay, Bideford, Devon.

1895 Thompson, George W., M.B., 80, Harley Street, W.

1888 Thompson, John Tatham, M.B. (V.-P.), 24, Windsor Place, Cardiff. (C. 1901-4.)

1895 Thompson, Robert, M.D., B.S., Bunya Bunya, Wickham Terrace, Brisbane, Queensland.
ELECTED

1905 Thomson, H. Wright, M.D., 3, Sandyford Place, Glasgow.
1898 Thomson, W. Ernest, M.D., 2, Somerset Place, Glasgow.
1900 Tomlinson, John H., "Belmont," Vicarage Road, Egham.
1904 †Tooke, Frederick T., M.D., 1, MacGregor Street, Montreal, Canada.
O.M. Tosswill, L. H., 34, West Southernhay, Exeter.  (C. 1896-9.)
1907 Tosswill, Leonard R., 34, West Southernhay, Exeter.
1904 Townsend, T. H. D., M.B., 14, St. Patrick’s Hill, Cork.
1905 Trench, F. P., M.B., 16, Bury Street, St. James’s, S.W.
1902 †Trilokekar, V. S., 308, Lohar Street, Bombay, India.
1890 Turner, William Aldren, M.D., 18, Harley Street, W.  (C. 1897-1900, 1903-6.  S. 1900-3.)
1898 Tyrrell, F. Astley Cooper, M.B., 27, New Cavendish Street, W.
1905 Tyson, W. J., 10, Langhorne Gardens, Folkestone.
1883 Unthoff, J. C., M.D. (V.-P.), Wavertree House, Furze Hill, Brighton.  (C. 1905-S.)
1888 Walker, Cyril H., M.B., (C.), 8, Oakfield Road, Clifton, Bristol.
O.M. Walker, G. E., 45, Rodney Street, Liverpool.
1892 Walker, H. Secker, 45, Park Square, Leeds.  (C. 1903-6.)
Elected.

1900 Wardale, John D., M.B., Carlton Villa West, Jesmond Road, Newcastle-on-Tyne.

1893 Warren, H. Guy S., 201, Macquarie Street, Sydney.

1907 Webster, G. F. G., Woodward Street, Wellington, New Zealand.

1893 †Weekes, Charles Jones, Lithgow, New South Wales.


1885 Werner, Louis, M.B., 31, Merrion Square North, Dublin. (C. 1902-5.)

O.M. West, Samuel, M.D., 15, Wimpole Street, W. (C. 1888-91.)

1908 Wharton, John, M.D., 21, St. John Street, Manchester.


1905 Williams, J. T., M.B., 31, Nicholas Street, Chester.

O.M. Williams, R., Fronbenlog, Bangor, North Wales. (V.-P. 1896-9.)

1894 Williams, W. E., M.B., Portmadoc, Carnarvonshire.

1888 †Willis, C. Fancourt, M.D., Satara, Bombay Presidency.

1903 Wood, Arthur, M.B., Fernleigh, Bodenham Road, Hereford.

1900 Wood, C. G. Russ, Hardwick House, Shrewsbury.

1889 Wood, David J., M.B., Cape Town, South Africa.

1903 Wood, Percival, The Grange, Crawley.

1909 Wood, R. Moorson, 46, Harley Street, W.

O.M. *Woodhead, G. Sims, M.D., 6, Scrope Terrace, Cambridge. (C. 1894-5. V.-P. 1905-8.)

1899 *Worth, Claud, 138, Harley Street, W.

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ELECTED.

1890 *Wray, Chas., Bank Chambers, North End, Croydon.

1898 †Wright, Edward Wm., M.D., 115, Montague Street, Brooklyn, New York.

1908 Wright, F. R. Elliston, M.B., Braunton, North Devon.

1896 Yarr, M. T., Lt.-Col., R.A.M.C., Army and Navy Club, Pall Mall, S.W.
RULES.

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

2. Constitution.—The Society shall consist of Ordinary and Honorary members. All registered medical practitioners of the United Kingdom, and all legally qualified medical practitioners in India and the colonies, whose qualifications are satisfactory to the Council of the Society, 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. Ordinary 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 for election as Honorary members men of dis-
tinguished eminence in Ophthalmology, or in the sciences bearing upon it. They shall be elected by ballot in the same manner as Ordinary members at the next meeting. The Honorary members shall not exceed ten in number.

7. Resignation of Members.—Any member may retire from the Society after giving notice in writing to the Secretaries and paying any contribution due.

8. Re-admission of Members.—Any member who has retired from the Society and wishes to rejoin it, must be proposed, balloted for, and admitted in accordance with Rules 4 and 5.

9. Expulsion of Members.—A member can be expelled only at a General Meeting especially 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.

10. Subscriptions.—The Annual Subscription shall be One Guinea, payable in advance in the month of October. 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 such member shall not be required to pay a Subscription during the next Session.

11. Arrears.—Any member whose Subscription is six months in arrear shall be reminded of the same by one of the Secretaries, and shall cease to be a member if it be not paid within the current year.

12. Composition Fee for Resident Members.—Any member may on entrance pay a Composition Fee of Twenty Guineas, and be thereby exempted from paying any further Subscriptions, such member enjoying all the rights and privileges of a Subscribing member. After entrance, the Composition Fee will be according to the following scale:—After five payments Seventeen Guineas, after ten payments Fifteen Guineas, after fifteen payments Twelve Guineas, after twenty payments Ten Guineas, after twenty-five payments Eight and a Half Guineas, after thirty payments Seven Guineas, after thirty-five payments Five Guineas, after forty payments Two and a Half Guineas, and after forty-five payments nil.
13. Composition Fees for Non-Resident Members.—Any member resident out of the United Kingdom may pay a Composition Fee of Eleven Guineas instead of the Annual Subscription, and will then be entitled to receive, post free, a copy of the Society's 'Transactions' each year; but if at any time such member subsequently become a Resident member of the Society, payment of the Annual Subscription shall be resumed at the end of six years from the date of election, or if more than six years have elapsed, from the date of return to residence. The question of further composition shall be decided by the Council. N.B.—The Composition Fee will be held to include the Entrance Fee if paid any time during the First Session.

14. Election of Officers.—The Officers of the Society shall be elected yearly by Ballot at the Annual Meeting. A Balloting list of the names recommended by the Council for election shall be sent to each Resident member, together with the notice of the Annual Meeting. In the event of any name being substituted for any recommendation of the Council, the election shall not be valid unless effected by a majority of those present at the meeting. The President shall not hold office for a period of more than two consecutive years. No other member of the Council, except the Treasurer and the Librarian for the time being, shall hold the same office for more than three consecutive years. Any ordinary member of Council who has been absent from all the meetings of the Council for one session shall not be eligible for election to the Council for the ensuing session.

15. Scrutineers.—Two Scrutineers appointed by the President at the commencement of the Annual Meeting shall receive the lists during the first hour, and report the result to the President, who shall have a casting vote.

16. 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.
17. **Secretaries.**—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.'

18. **Treasurer.**—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.

19. **Librarian.**—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.

20. **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.

21. **Meetings of Council.**—The Council shall meet immediately after the meetings in October, January, May, and June, 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. The Council shall decide upon all questions relating to the reception of communications, and to their publication in the Society's 'Transactions.'

22. **Vacancies of Officers.**—The Council 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.

23. 'Transactions.'—A copy of the 'Transactions' shall be sent to each Ordinary member of the Society whose subscription is not in arrear, and a copy of the 'Transactions' shall be sent to each Honorary member of the Society.

24. Meetings.—These shall consist of Ordinary, Clinical, Special, and General Meetings.

Ordinary Meetings.—The business during the first half-hour shall consist of the discussion of cases and card specimens, after which papers shall be read and discussed. Nothing relating to the laws or management of the Society shall be considered.

Clinical Meetings.—One or more of the meetings in each session may be devoted exclusively to the exhibition and discussion of cases and specimens. The number and dates of such meetings shall be arranged by the President and Secretaries.

The bye-laws and regulations relating to Ordinary and Clinical Meetings will be found on page xlv.

Special Meetings.—At the discretion of the Council an extra meeting may be arranged in order to hold a discussion upon some subject, of which due notice shall be given, or one of the Ordinary Meetings of the Society may be devoted to this purpose.

General Meetings.—The Annual General Meeting, of which every resident member shall receive one week's notice, shall be convened by special summons; ten shall form a quorum. It shall be held immediately after the Ordinary Meeting in July. The business shall consist in the election of Officers, the presentation and adoption of the Annual Report, and the discussion of any proposed alteration of the Rules, notice of which shall be given in the summons convening the meeting. No alteration in the Rules shall be adopted unless four fifths of the votes are given in its favour.

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, or on the requisition of fifteen members of the Society. The nature of the business to be transacted at such meetings
shall be specified in the summons sent to each member of the Society, and no other business shall be considered.

Dates and Hours of Meetings.—The dates of meetings shall be, unless otherwise determined by the Council, the third Thursday in October, the second Thursday in November and second Thursday in December, the last Thursday in January, the second Thursday in February, the second Thursday in March, the first Thursday in May, the second Thursday in June, and the second Friday in July. All but the General, Clinical, and Special Meetings shall commence at 8.30, and shall not be prolonged after 10 p.m., except on the proposal of a member, duly seconded, and carried by a show of hands. The hour of all other meetings shall be determined by the President and Secretaries.

25. Visitors.—Each member may introduce as visitors two members of the medical profession to all but the General Meetings. They shall sign their names in the attendance book, opposite to the name of the member introducing them.

26. Order of Communications.—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.

27. Publication of Papers and Discussion.—No paper shall be read before the Society unless a copy of it has been sent to the Secretaries at least four weeks before the meeting, together with an abstract suitable for immediate publication in the journals. When possible, notice should be given relating to card cases, etc. All papers read before the Society shall be deemed the exclusive property of the Society, and if published elsewhere by the author without the express permission of the Council shall thereby be disqualified for admission into the Society’s ‘Transactions.’ No report of the meetings of the Society may be published by members or others without the sanction of the Council.

28. Committee of Reference.—Six members of the Society shall be chosen annually by the Council to form with the President and Secretaries a Committee of Reference. No
communication to the Society shall be rejected unless it has been referred by the President and Secretaries to two or more members of such Committee of Reference for the purpose of considering the fitness of the communication in question to be read before the Society, or to be published in the Society's 'Transactions.'

LIBRARY RULES.

1. The Library shall be open at the same hours as that of the Medical Society, viz. from 9 a.m. to 6 p.m. daily, except on Saturdays, when it will be closed at 2 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 book 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.
ADAMS FROST COLLECTION OF LANTERN SLIDES.

1. The slides shall be in the custody of the Honorary Librarian.

2. They may be lent, for the purpose of teaching, to teachers in any recognised Medical School on application to the Honorary Librarian. The Council shall also have the power to lend them on any special occasion.

3. The regulations under which the slides may be borrowed, together with a list of the slides, shall be printed (a) in the Transactions; (b) in the Catalogue of the Library; (c) on leaflets which may be sent to enquirers.

4. Other slides may be added to the collection, but the numbering (in so far as it coincides with that of the figures in Frost's Fundus Oculi) shall be retained.

5. That the following be the printed regulations under which the slides may be borrowed, which may, however, be modified by the Council on special occasions.

Regulations for the Loan of Lantern Slides.

1. Not more than 20 slides may be borrowed at one time.

2. Not less than three days' notice of the wish to borrow slides shall be given, and a list giving the numbers of the slides required shall be sent to the Honorary Librarian.

3. The borrower shall sign a receipt for the slides, which will be given back to him on their return.

4. The slides must be returned within three days, no other slides can be lent to the same borrower till this has been done.

5. Any slides broken, lost, or damaged shall be replaced or repaired at the expense of the borrower.
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."

EDWARD NETTLESHIP PRIZE.

On the occasion of Mr. Nettleship's retirement from practice in 1901 a fund was inaugurated by his friends and pupils, with the object of founding an Edward Nettleship Prize for the Encouragement of Scientific Ophthalmic Work, and at a meeting of the General Committee of the above fund on July 2nd, 1902, the following resolutions were adopted:

1. That the prize should be a Gold Medal for the encouragement of scientific ophthalmic work.

2. That the Council of the Ophthalmological Society of the United Kingdom be asked to undertake the custody of the Prize and Fund, and to appoint Trustees.

3. The award of the Medal shall be entrusted to three members of the Ophthalmological Society appointed by the Council, such members to be changed after each award.

4. That the Medal shall be awarded at such intervals as shall be determined upon.

5. That only British subjects be eligible to receive this Prize.
6. That, subject to the discretion of the said Council, the Prize shall be awarded—

(a) For the most valuable contribution to Ophthalmology during the three years immediately preceding or since the last award, or

(b) For the best work done on any subject previously selected and announced by the Council of the Ophthalmological Society.

7. That in the event of no work being found of sufficient merit an award shall not be made.


"It shall be lawful for the Trustees, with the consent of the Council of the Society, to expend any surplus arising from the non-award of the Medal in any one year, or accumulated during several years, in the purchase of works for the Library of the said Society, such works to be inscribed 'Purchased by the Nettleship Prize Fund'; or, with the consent of the said Council, to expend such surplus in any manner calculated to promote the objects of the Society as defined in the Rules."

BYE-LAWS CONCERNING COMMUNICATIONS.

1. The 'Transactions' shall consist of such communications made to the Society by or through members, as may be deemed by the Council suitable for publication. Also of discussions of importance or interest arising out of such communications.

2. No communication to the Society shall occupy more than twenty minutes, and in the subsequent discussion of it no member shall speak more than once, or for more than ten minutes, without the special permission of the Chairman.

3. All communications accepted by the Society become the property of the Society.

4. Communications are admissible which may have been read elsewhere, provided they have not been published, and are not
intended to be published, in whole or in abstract, through another channel.

5. The cost of illustrations shall be borne by the Society so far as, in the opinion of the Council, is consistent with the state of its funds.

6. Reprints of papers may be obtained by authors at their own expense, by arrangement with the printer.

REGULATIONS CONCERNING THE EXHIBITION OF PATIENTS AND OF PATHOLOGICAL SPECIMENS BY CARD.

A. Patients must attend not later than 8 p.m., and will be allowed to leave at 9.30. A card, provided by the Society, must be placed conveniently near the patient (unless it is undesirable that it should be read by the patient or friends), and on it must be clearly written an account of the case, comprising all the particulars intended for publication. The title only of the case will be announced by the President to the meeting, but the Exhibitor (or his representative) must be present at the meeting, and be willing to read the case and furnish additional details if called upon to do so; the length of such oral communications not to exceed five minutes.

The narration and discussion of Card Specimens shall not occupy more than the first half-hour of the meeting.

B. Pathological Specimens may, at the discretion of the Exhibitor, be shown by card, and will then be subject to the above regulations. It is particularly to be noted that the description on the card must comprise all the particulars intended for publication.

REGULATIONS FOR CLINICAL EVENINGS.

1. Notice of cases or specimens should be sent in to the Surgical Secretary as early as possible, but not later than the Thursday preceding the meeting.
2. Cases and specimens may be shown without previous notice if there is time, but not until all on the printed list have been disposed of.

3. The number of cases to be shown by any member, and the total number on one evening to be left to the discretion of the President and Secretaries.

4. A list of the cases and specimens to be sent before the meeting to every member in the kingdom.

5. Particulars of each case to be fully written out before the meeting, and given to the Secretaries at the close of the meeting.

6. A short abstract of the case to be written on the card provided for the purpose, and placed by the patient.

7. The narration of a case shall not occupy more than five minutes, and in the subsequent discussion no member shall speak more than once or for more than five minutes.

8. Patients and specimens to be in place and ready by 8 p.m., one of the Secretaries being in attendance.

9. That the meeting for the narration and discussion of cases shall commence at 9 p.m.

10. If the exhibitor or a representative be not present the case cannot be taken.

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ON SOME HEREDITARY DISEASES OF THE EYE.

BEING

THE BOWMAN LECTURE.

Delivered on Thursday, June 10th, 1909,

BY

E. NETTLESHIP.

INTRODUCTORY.

I have chosen the heredity of certain diseases of the eye as my subject, because whilst possessing great attractions, it furnishes a theme upon which one who is to some extent out of touch with the newest clinical ophthalmology may still, perhaps, hope to speak without presumption. I believe also that all here who had, like myself, the great privilege of acquaintance with Sir William Bowman, of experiencing the charm of his voice and diction, and of seeing him at work, will agree that had he been alive to-day he would, with the keen but discerning enthusiasm that he always brought to bear upon new scientific problems, have recognised that the study of heredity confronts us with subjects of absorbing interest, the right interpretation of which must have important consequences for the future of our race. As a matter of fact Bowman actually communicated to Charles Darwin some of the earliest generalised observations upon the heredity of cataract, and, as we shall see further on, later work has but confirmed his statements.

Taking a few of the principal ophthalmic diseases, the hereditary transmission of which is now recognised, I propose to-day to consider them from that point of view.

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and at the same time to indicate some of the directions in which further research into their nature is most needed. Many, such as the heredity of errors of refraction and of the musculature of the eye, I cannot touch. We could all cite plenty of examples showing the family prevalence of both these classes of defect, but I do not think much has yet been done in the direction most suitable for clinical observers—the careful record and analysis of individual pedigrees. The elaborate statistical enquiry upon the inheritance of ametropia lately brought out by Professor Karl Pearson and Miss Amy Barrington* will help to elucidate the ever-present problem of environment versus heredity in the causation of myopia, although the imperfection of the data (data derived from ophthalmological examinations, be it confessed) often detracts from their value to the biometrical statistician.

I shall say but little on the theoretical side of my subject, being, as I am, quite unable to deal with the biological and mathematical complexities in which the modern student of heredity finds himself involved. As one who must be content with a very modest share of spade work I am grateful that in the medical domain there is still virgin ground where the tasks of excavating, collecting and recording may be safely undertaken by those who enjoy them. And here I wish to express my deep indebtedness and cordial gratitude to the many colleagues and friends who have generously furnished me with cases and numerical records bearing upon heredity, and have, often at much tedious trouble to themselves, aided me in the collection and disentanglement of genealogical details. I could have done next to nothing without such help.

Before getting to close quarters with individual diseases I must ask your indulgence whilst, in order to avoid needless repetition, I refer to certain generalities.

One of the first questions generally raised when heredity is under discussion is the influence of consanguinity in the parents or ancestors. The belief that kinship between parents is a source of disease or degeneracy in the children is widely spread and has its roots deep in the past; and yet we meet every day with marked differences of opinion and practice in regard to the matter, between one house or family and another. The real question is this: Can the marriage of blood relations produce disease of which neither the parents nor ancestors showed any trace, or does the consanguinity operate simply by increasing the likelihood that both of a pair of parents will contain the seeds of the same undesirable, or it may be desirable, character? If the former be true no cousin-marriage can be said to be safe. But if the latter be the correct position—and the results of all modern research appear to point that way—the outcome of the consanguineous union will depend entirely upon whether the particular disease, or other heritable character, is carried by both parents, by only one of them, or by neither; the consanguinity will be operative only if it increase the chance that both parents are tainted. If the transmissible condition be one that is very common there may be as much chance of its presence in both of an unrelated pair as in both of a pair of cousins; but any comparatively rare disease is more likely to be present in two cousins than in two unrelated persons.

Accordingly we find a general belief in the medical profession that in diseases so relatively infrequent as retinitis pigmentosa and deaf-mutism consanguinity of the parents plays an important part. And the same is true of some other conditions where, as in the diseases just named, both sexes are liable to suffer from, and both liable to transmit, the disease.

But in sex-limited conditions, such as Leber's disease and congenital colour-blindness, where only the males suffer, though the disease is carried down by (apparently) normal females, consanguinity of parents is known to be infrequent. If we start with a colour-blind male we know
that all his children will, as a rule, have normal colour-perception; that if his sons, who neither exhibit nor carry the defect, have issue, that issue too will be normal; but that some of the sons of some of his daughters will show the defect, whilst the other daughters, who we presume do not carry it, will have all normal children. If one of these normal children of a normal daughter marries a cousin,

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<td>- female</td>
</tr>
<tr>
<td>Twins.</td>
</tr>
<tr>
<td>Died young.</td>
</tr>
<tr>
<td>With or without sex signs; Two or more persons, the number sometimes shown by numeral.</td>
</tr>
<tr>
<td>Other Diseases than the one illustrated by the Order of birth unknown</td>
</tr>
<tr>
<td>Parents Consanguineous</td>
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</table>

the issue of one of the normal sons, the result, as regards colour-perception, will be the same as if two unrelated normals marry. In fact, if the sex-limitation were invariable in colour-blindness and other sex-limited conditions, only one kind of cousin-marriage out of the several possible kinds shown in Figs. 1 to 5 (I speak of first cousinship throughout) would be attended by special risk, viz., when the mothers of the parents are sisters who,
although not manifestly colour-blind, both carry the defect (Fig. 1, II). If one of these two grandmothers of the male IV, I is free from taint (as in II, Figs. 2 and 3), it is impossible to understand that she can have any more influence than if she came from a different stock; and the same will be true if one or both of the two grandparents (parents of the cousins) be male and unaffected (II, Figs. 4 and 5).

In this connection Fig. 48 (Leber’s disease, a sex-limited affection; Klopfer’s case, 1898), is instructive.

Effect of Cousin-Marriage in Sex-limited Disease.

The disease appeared only in the last two of the eight or nine known generations, in three childships cousins to each other, and, according to rule, affected males only.*

Each of these childships was the issue of a consanguineous marriage; but, as the lines show, these parental cousinships all came from grandfathers who were unaffected, and therefore, on the hypothesis, did not contain the disease. If the normal rule obtained, as seems to have been the case, the disease must have followed the thick, black line to an affected male

* Some particulars of this pedigree will be found at p. cviii.
ascendant of V, 23, in which case the cousinships do not count, for V, 23 was from an outside stock.

But limitation of the disease to the males and transmission through normal females is not invariable. For

though I believe that an unaffected male never carries colour-blindness, exceptions are found to the other part of the rule. Thus an affected male sometimes transmits to his son, and colour-blindness is sometimes seen in females. The influence, whatever it is, that usually prevents the colour-blind male from passing the defect on to, or through, his sons and compels him to transmit it only
through daughters who do not themselves show it, is sometimes lacking.

The effect of consanguinity and the facts as to sex-limitation can both be considered in relation to the Mendelian theory. According to this theory of inheritance, a character and its opposite or absence is represented in the gametes by particles; this at least applies to certain characters, pathological as well as physiological. These particles occur in even numbers or pairs in each gamete. The members of each pair may be similar (both representing presence of the character, or both representing its absence), or may be dissimilar (each pair containing one of each kind). In the fertilised germ the pairs derived from the gametes, so to speak, change partners. The constitution of the particles in the resulting zygote depends upon that in the original gametes; the zygote may contain only pairs representing the character, only those for its complement or absence, or a hybrid between the two. For convenience, one of the characters (or its particulate representative) is called “dominant” (D.D., Fig. 6 a and b), because when it unites with the other, or “recessive” (R.R., Fig. 6 b), the resulting hybrid shows only the former character, although carrying both (D. R., Fig. 6 b). The other character, the “recessive,” although potentially present, is undeveloped and does not show. In some cases, however, the “recessive” factor does show, and then the visible result is an intermediate form.

As I understand the matter, the vital point in Mendel’s interpretation of the facts of heredity is the separate, and numerically equal, particulate representation of qualities in the gametes. Visible dominance is not an essential part of the theory, because intermediates occur showing both the constituent qualities. The terms “dominant” and “recessive” are convenient and useful, but must be dissociated from any conception of what may be either “good” or “bad,” “desirable” or “undesirable,” “strong” or “weak”; indeed, as we shall see presently, in some cases the disease or defect behaves as what Mendel
called a dominant over the normal condition, in other cases as a recessive. But since the victims of the same hereditary disease, be it purely dominant or purely recessive, scarcely ever intermarry, no trace of pure disease is established.

In most cases, using Mendelian terms, an hereditary disease is transmitted by the mating of an impure, or hybrid, dominant (D.R.) with a recessive (R.R., Fig. 6 e),

**Mendelian Inheritance**

<table>
<thead>
<tr>
<th>Fig. 6</th>
<th>A</th>
<th>B</th>
<th>C</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>DD × DD</td>
<td>DD × RR</td>
<td>DD × DR</td>
</tr>
<tr>
<td>II</td>
<td>DD·DD</td>
<td>DR·DR</td>
<td>DD·DR</td>
</tr>
<tr>
<td>Rare in Human Disease</td>
<td>Common when Disease Recessive</td>
<td>All the Offspring Normal</td>
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**Theoretical results of First Matings.**

<table>
<thead>
<tr>
<th>D</th>
<th>E</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>DR × DR</td>
</tr>
<tr>
<td>II</td>
<td>DD·DR·DR·RR</td>
</tr>
<tr>
<td>Common when Disease is Recessive</td>
<td>1/4 of Offspring Diseased</td>
</tr>
<tr>
<td>Commonest form: Disease either Dominant or Recessive</td>
<td>1/2 of Offspring Diseased in either case</td>
</tr>
</tbody>
</table>

and if sufficiently large numbers be taken half of the resulting offspring should be normal and half diseased, whether the disease be the dominant or the recessive partner.

If the disease be dominant it is of course rare for mating to occur between two persons suffering from it. When such union does occur all the offspring should be diseased if the dominance in one parent or both be pure (Fig. 6 a, b, and c), three quarters if both parents be hybrid dominants (Fig. 6 d).
If the disease be recessive the matings shown in c and d will also be frequent, for then either one parent or both will appear normal; mating c will give only normals, but half of them should carry the disease; in mating d one quarter of the offspring should show the disease if sufficiently large numbers be taken, and one half should carry it invisible or potential; in mating e, as just stated, the disease should appear in half and be carried by the other half, if sufficient numbers be taken.

Therefore if the simple Mendelian theory be applicable to any human disease or defect we shall expect that, in most cases, either one quarter or one half of the offspring will show the condition (Fig. 6 d and e).

The assumption is that dominance and recessiveness are constant for the same character in all stocks and families; that the same character or disease cannot be dominant in one pedigree and recessive in another. But when a given character is linked with sex in such a way as to be manifest only in one sex (the male), although carried in an incompletely and invisible state by the other (female), the fact has been explained in Mendelian terms by Professor Bateson on the assumption that the character, although dominant in the male, becomes recessive in the female. This hypothesis appears to explain some otherwise difficult cases. For example it can be made to account for the clinical fact—invariable as far as we yet know—that a colour-blind woman transmits her defect to all her sons, and that she herself has always had a colour-blind father. But on the other hand it does not explain the ordinary experience that a colour-blind father very seldom has colour-blind sons.

And in many other cases the experimental breeding of plants and animals has given results which, in order to bring them within the four corners of the Mendelian theory, require the assumption of various modifying or controlling influences. But this is not the time, nor am I the person, to discuss the hypotheses dealing with such subjects as "dihybridism," "gametic coupling," "rever-
sion on crossing;" "epistatic" and "hypostatic" factors, and the complex results that flow from them, results in which the actual and the expected numbers are often so strikingly near.

Whether the Mendelian theory, or any one of the current doctrines of heredity, contains the whole truth is perhaps doubtful; but we may rest assured that sooner or later a ground will be discovered upon which the advocates of the various theories can meet in common. Meantime, I conceive that our contribution to the problem, as students of the natural history of living man, should consist in the collection, classification and analysis of fresh pedigrees of disease or defect wherever we can find them.

The Mendelian theory in its simple form is so precise, and in regard to a number of unit characters in certain plants and animals its expectation has been found to fit so nearly with experimental results, that no surprise can be felt at the attraction it has for workers in human heredity. But, founded as the theory is upon a strictly quantitative conception, it would certainly never have been formulated from data afforded by human disease alone, and this for several reasons. Thus it is difficult and often impossible to get a record of all the maternal conceptions; and even then we do not know how many of the miscarriages and stillbirths, and but rarely how many of those born alive but dying in infancy, would have been affected.* Again, when dealing with a condition that comes on many years after birth the record is incomplete unless all can be followed quite up to the susceptible age. Then it is, to say the least, probable that in some cases the disease which exists potentially may never appear for lack of some agent or influence—called for want of a better name an excitant or stimulant—that is necessary to complete it, e.g. Fig. 38, retinitis pigmentosa probably brought out

* We can at present only assume that had these immaturities and early deaths survived they would have suffered in the same proportion as those who lived. This assumption, however, may be unwarranted.
by haemorrhage. Further, there can be little doubt that in certain cases we have to deal with equivalent, substitute, or heteromorphic diseases—cases in which the same cause produces disease of one part, e.g. the retina, in one person and of another part, e.g. the organ of hearing, in another member of the same genealogy. In popular no less than medical belief such heteromorphism is sufficiently notorious in the case of gout, though the evidence is somewhat lacking in precision. Lastly, can we regard it as certain that single births, occurring at comparatively long intervals, always follow the same laws of transmission as frequently recurring multiple broods?

Amongst normal human characters the colour of the iris has been investigated, and Hurst has shown that pigmentation is in Mendelian terms dominant to lack of pigment, i.e. the brown or otherwise pigmented iris is dominant to the pure blue or grey iris. Captain Hurst* was good enough to let me see, on May 17th last, at the Village School at Burbage, his home in Leicestershire, a considerable sample (thirty-eight) of the persons upon the colour of whose irides his paper was based. I wished particularly to know whether entire lack of visible pigment meant the same thing to myself as to Mr. Hurst. Mr. Hurst's method is to examine the iris with a magnifier out of doors in good daylight. The ones I saw were all children attending the school and we examined them in the open yard outside at about 2 o'clock. In those that Mr. Hurst had recorded as "simplex," i.e. entirely free from visible stroma pigment, I could find not the least evidence of pigment in any, except a doubtful slight trace at one part of one iris in one child, so slight that I thought the appearance might perhaps be due to the colour of an unusually large blood-vessel. In the slightly and partially pigmented ones Mr. Hurst's observations and mine were also in complete agreement; in many of this class the pigment, although very evident on careful scrutiny.

at close quarters, was invisible to casual inspection and the colour of such irides would undoubtedly have been passed as "blue" or "grey," meaning "devoid of stroma pigment," if examined without a lens, or at a distance of twelve or more inches, or in a not very well lighted room.

In regard to human diseases and defects I consider that, in spite of, or allowing for, numerical discrepancies that must occur from such causes as have been mentioned, many pedigrees are, in their broad features, consistent with Mendelian theory. I purposely use no stronger term; for although, as I have said, human pedigrees do not, and cannot, prove the theory, we may well be interested in finding that some of them are at least compatible with it so far as they go. Pedigrees abound in which the rule, "once free always free," required by Mendelism for a dominant disease is found to hold good; and others occur in which the frequency of consanguineous marriage and of discontinuity in transmission are consistent with a recessive. It is when we come to quantities that the relative numbers of diseased and normal are often found to be wide of the mark, sometimes far too many, sometimes not nearly enough, being affected. In regard to such discrepancies we may remember, besides the hindrances to complete knowledge above mentioned, that at present we know very little about the indications and measure of inherited liability or soil as distinguished from actual disease, e.g. liability to tubercle or to mental disease; nor do we know whether in certain cases death in infancy may not itself take the place of the disease that is to appear later in life in the survivors.* Then, again, granting exact numerical segregation of unit characters, it seems reasonable to expect, for man and the higher animals,

* For a case in which D.R. × D.R. gave, in self-fertilised variegated antirrhinum (snapdragon) 2 instead of 3 D. to 1 R., because the remaining fraction died for want of chlorophyll during germination, see Baur, quoted by Bateson in his Mendel's Principles of Heredity, 1909, p. 253, where, under the heading "Departures from Numerical Expectation," other facts and suggestions bearing on the subject will be found.
complexities due to interaction far more intricate than any yet dealt with in experimental biology. Finally the particular representatives of hereditary disease must often, if not always, be far less ancient in origin than those representing normal characters, and therefore presumably more easily modified by disturbing influences during embryonic life.

Even the term "unit" needs to be defined, for just as hardly any two persons are exactly alike even in a single normal feature, and as in cases of family disease or defect minor differences can often, perhaps generally, be observed between the morbid appearances in one or another of the affected members, so we can safely take it for granted that the germinal representatives differ slightly amongst themselves in some of their attributes. The alternative would be to suppose that all slight variations of inherited condition were due to environmental causes either before or after birth.

I propose, nevertheless, to give, for what they may be considered to be worth, the numbers of affected and normal actually found in the collected pedigrees of a few of the diseases we are concerned with to-day, for comparison with Mendelian expectation.

Only those sibships (childships) were used that were probably complete, and either contained a case or cases of the disease or were the offspring of an affected parent. Early deaths, stillbirths and miscarriages have been omitted, as well as all sibships that were certainly, or even probably, incomplete. When the disease was discontinuous the intervening (free) generation was not counted. All these omissions, though making for accuracy, entail large deductions from the total.*

I. Acquired or post-natal cataract at all ages. Descent continuous: total 440 (100), affected 177 (40), normal 263 (60), numbers that are quite wide of Mendelian requirements. But we may assume without the least hesitation that had every member been examined

* The data used are given in Appendix I.
incipient senile cataract would have been found in some, perhaps a fair number, of those who, judging only from report, have been entered as normal; the 40 per cent. is therefore too low, though we cannot say by how much.

II. Congenital cataract of all kinds. Descent continuous: total 566 (100), affected 260 (46), normal 306 (54), a bad approximation to the equality required by the Mendelian scheme, Fig. 6 e, if the pedigrees used are as complete as they are supposed to be.

III. Retinitis pigmentosa; pedigrees showing continuous descent only: total 387 (100), affected 198 (51), normal 189 (49). Practical equality as in Fig. 6 e.

IV. Congenital night-blindness with continuous descent. For quantitative purposes the great Cunier pedigree is too inexact for this purpose. Other data give: total 63, affected 33, normal 30. Not far from equality, as required by Fig. 6 e.

V. Leber's disease. Descent discontinuous, all cases, female as well as male, being counted: total 547 (100), affected 245 (45), normal 302 (55). A poor approach to equality.

It must, however, be mentioned that in this disease the proportion of diseased to normal is influenced by sex.

(a) In families where the disease affects males exclusively the numbers are—total, both sexes, 402 (100), affected males only, 165 (41), normal, both sexes, 237 (59).

(b) In families where the disease affects some females as well as males the numbers are—totals, both sexes, 145 (100), affected, both sexes, 80 (55), normal, both sexes, 65 (45).

VI. Retinitis pigmentosa; pedigrees showing invariably discontinuous descent.

The numbers in this group can be interpreted in Mendelian terms on the assumption that the disease is dominant in some sibships and generations, recessive in others.

The totals are small, but may, for the present, be analysed into three sub-groups as follows:
(a) Seventeen completed sibships containing—total 117 (100), affected 55 (48), normal 62 (52).

(b) Ten similar sibships containing—total 58 (100), affected 15 (26), normal 43 (74).

(c) Three similar sibships, containing—total 13, affected 11, normal 2.

It will be noticed that in these three small sub-groups, where in all cases the parents were normal, only the second (b) fits the Mendelian expectation of Fig. 6 n, where two impure dominants carry the disease as recessive and throw one quarter of their offspring diseased. Both (a) and (c) require dominant to have changed place with recessive in the second generation in order to bring them into the theory at all. (Fig. 6 a and c.)

The numbers I have just quoted are the outcome of careful examination and the exclusion as far as possible of incomplete examples; I hope, therefore, that they will not be without interest at the present time. I may say that I was quite unprepared for such a near approximation to halves and quarters as are shown by certain of these groups.

Allusion has been made to the change of dominance supposed to occur in sex-limited disease. I believe there is clinical ground for suspecting that dominance, if we use the term, may sometimes change, or rather may be different, for the same disease in different families irrespective of sex; and if this be true, the factor causing the alternation of dominance in the sex-limited cases may be, not sex itself, but something else, usually, but not invariably, associated with sex. Retinitis pigmentosa, for instance, appears to be recessive in many families, but in the largest recorded pedigrees it behaves as a dominant, and yet it is the same disease in both instances. If such change can occur at all, we need go only a step further in order to explain the first appearance of a dominant disease. A condition that has for want of meeting with another similar gamete been propagated for generations as recessive in an impure dominant would at
once become apparent, *i.e.*, dominant, if it came under the action of the supposed transforming influence.

I now leave these crude speculations and come to the safer ground of observation.

Anticipation in hereditary disease means the manifestation of the morbid change at an earlier age in each successor, either in members of each succeeding generation as a whole, or in successively born children of one parentage. Bowman was one of the first to notice anticipation in successive generations in hereditary acquired cataract,* and examples of the phenomenon will be quoted later. It is only seen in some of the families, and we do not yet know in what proportion of them.

Anticipation in generations is also a marked feature in hereditary glaucoma, but the material hitherto collected is smaller than for cataract.

Anticipation is also seen fairly well marked in connection with Leber's disease, both in successive generations and successively born siblings.

Anticipation is not known to occur in retinitis pigmentosa, and I believe has not been proved in the now well-known hereditary reticular and nodular keratitis.

This anticipation in heredity is by no means peculiar to diseases affecting the eye. It appears to occur in phthisis,*† and is certainly sometimes met with in hereditary diabetes (Fig. 7),‡ and hereditary jaundice with enlarged spleen (Figs. 8 and 9)§; also in at least one

* Bowman communicated these observations to Darwin, who incorporated them in his chapters on "Inheritance" in Animals and Plants under Domestication, i, p. 453, and ii, p. 56 (1868). They do not seem to have been published in any other form.

† Pollock, J. E., Medical Handbook of Life Assurance, 4th edition, 1895.


§ The ages at death are given in Fig. 7 (unpublished case, E. N.), but they could not be conveniently inserted in Figs. 8, 9 and 10.

§ Figs. 8 and 9 are constructed from the paper, "Some Cases showing Hereditary Enlargement of the Spleen," by Claude Wilson, Clin. Soc. Trans., xxiii, 162 (1890), and xxvi, 163 (1893).
extensive pedigree of hereditary ataxy* (Fig. 10), and possibly in some of the other chronic diseases of cord and brain. When a disease tends always to occur at a later age in one sex than the other, the comparison as to anticipation in hereditary examples must of course be made between members of the same sex.

It appears to me that the subject of anticipation deserves much more attention than it has received in relation to theories of heredity and to the origin and extinction of heritable conditions.

We may note that the reverse process, appearance of the disease at a later age in the later born, though


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sometimes taking place irregularly in successive siblings, does not seem to occur in successive generations.*

Two different heritable conditions may be met with in the same pedigree, and care is then necessary to distinguish coincidence from correlation or equivalence. Thus in Fig. 11 we have congenital lamellar cataract, congenital ptosis, and progressive goitre; and it is seen that, whatever may have been the source of the ptosis, the goitre undoubtedly came in from an entirely distinct stock. Many analogous cases might be quoted.

Reference has been made to cases in which a heritable condition, though apparently limited to one anatomical or physiological system, may invade different parts of that system in different persons. The best of the well-defined cases is seen in the trio—retinitis pigmentosa, progressive nerve deafness, and feeble-mindedness or idiocy, diseases that seem capable of acting as mutual equivalents or substitutes; some correlation also seems to exist between Leber's disease and epilepsy; and of course the neuropathic constitution may show itself in several different forms of mental disease. Albinism is also complicated with defects of the nervous system in a disproportionate number of cases, and the association must therefore be looked upon as more than a coincidence. The possibility that early death may in certain cases represent a substitute form of a heritable disease has already been mentioned.

It appears that the individuals affected by hereditary imperfections and disease are very often members of unusually large sibships. This has been mentioned by Dr. James Taylor† in relation to hereditary ataxy, whilst Karl Pearson‡ concludes that both tuberculous and deaf-mute stocks are quite as fertile as, and probably more fertile

* Darwin makes a general statement to the same effect (Animals and Plants under Domestication, ii, p. 56).
† James Taylor, T.O.S., xvii, 1897, p. 63.
‡ A First Study of the Statistics of Pulmonary Tuberculosis, 1907, p. 20.
than, normal stocks of the same social class. In regard to more general signs of inferiority we are told by Mr. Heron that, at any rate for the London districts, "there is a very close relationship between undesirable social status and a high birth-rate." We shall see presently that the same is apparently true for retinitis pigmentosa and other eye diseases. Some caution, however, is needful in concluding that large birth-rate and disease are as closely connected as they appear to be; for some pedigrees of disease have been selected for investigation just because they contain large numbers of accessible members, and the prevalence in them of large childships may be only what is normal to the particular population, class or stock.

I will refer next to the question of sex liability in some of the hereditary eye-conditions.

We have first the sex-limited group—ordinary colour-blindness, Leber's disease of the optic nerves, and one form of congenital stationary night-blindness. In these so large a majority of the affected persons are males that affected females are regarded as rare exceptions; and this rule holds in general terms for each separate family as well as for the aggregate.

Next come diseases that have no special correlation with sex; the lump sum of males and females is about equal, or at most not widely different, although separate families often display marked departures from the rule, one having a great excess of males, another of females. The best examples are all forms of post-natal cataract, glaucoma (so far as we yet know), and a second form of congenital stationary night-blindness. Probably other diseases will be added to this group.

In the third group—containing all forms of congenital

* "On the Relation of Fertility in Man to Social Status and on the Changes in this Relation that have taken place during the last Fifty Years," David Heron, 1903, Drapers' Company Research Memoirs: Studies in National Deterioration, p. 21.
cataract, retinitis pigmentosa, albinism, and probably some of the less frequent affections, such as congenital daylight-blindness—we still find great discrepancies as to sex numbers in individual families, but when large numbers are taken a fairly uniform, though not extreme, preponderance of males.

As the sex-inequality in this last class cannot be accounted for by any obvious cause it is probably the expression of some general law, and the following facts seem to support this view: (1) Although more boys than girls are born (about 104 boys to every 100 girls in England and Wales in 1907)* the inequality is more than redressed by the higher general death-rate for males so that the total living population shows a deficit of males (about 93 males to every 100 females in England and Wales in 1907). (2) The males die in excess chiefly (a) between birth and five years of age, (b) between fifteen and sixty-five; between five and fifteen the sexes die in nearly equal numbers (about fifty-one females to forty-nine males). The higher mortality of males under five, which alone concerns us now, is due chiefly to deaths from causes classed by the Registrar-General collectively as "immaturity," i.e. premature birth, congenital defects, teething and congenital hydrocephalus. In 1907 out of every hundred children dying from these causes under five years old fifty-six were males, forty-four females. (3) There appears to be a similar excess of boys over girls with various "defects of development," principally of the sense-organs and intelligence, such as Dr. Francis Warner described in 1894 in children at the elementary schools.† Dr. Warner's statistics show that if the number of boys and girls examined by him had been equal there would have been sixty defective boys to forty defective girls in every hundred of those selected by him as showing deficiencies.‡

* The data from which these and the succeeding statements are drawn may be found in the Report of the Registrar-General.
‡ Since the above was written Mr. Alan Barlow has supplied me with
Archibald Garrod* states that the rare heritable conditions, alkaptonuria, cystinuria and pentosuria are all more frequent in males than females. In 157 subjects of these three diseases he finds no less than 113 males.

Now it is not a little remarkable, as regards the third group of eye diseases just mentioned, that in retinitis pigmentosa (1381 cases) and lamellar cataract (1793 cases), 62 per cent. of those affected are male and 38 female; whilst in other forms of congenital cataract (335 cases) and in albinism (upwards of 1000 cases) the proportions are not very different—about 55 per cent. males and 45 per cent. female. In day-blindness there is a considerable excess of males.

It is said also that there is a marked excess of males over females amongst deaf-mutes.

I feel sure that stores of information as to the relative liability of the sexes to hereditary disease must exist. But meanwhile the few facts now brought forward favour the view that, in man, the male is on the whole more liable than the female to many innate defects and diseases, and perhaps especially to such as affect the organs of sense and intelligence.

It is extremely important to know whether the inheritance of an imperfection influences the longevity of the affected who survive; either by the direct effect of the disease upon vitality as in diabetes or haemophilia, or by some figures from the Education Office, which, although probably needing correction in certain particulars, appear to point in the same direction. These figures are taken from the *Statistics of Public Education, 1906-7-8*, and refer to the number of children between the ages of five and sixteen attending schools for the defective and epileptic in England. The average number of children attending about 160 such schools in each of the three years mentioned was 16,161, of whom 6019 were boys and 4445 girls. The numbers are vitiated to some extent by the facts that *(a)* they include a certain number with physical rather than mental defects, and *(b)* boys tend to leave the schools at an earlier age than girls; but these two sources of error may not improbably tend to cancel each other, and in any case would not be likely to account for nearly all the difference between 57 and 43 per cent. shown by the above numbers.

its indirect power of lowering the resistance to hurtful influences. The purely hereditary diseases of the eye do not seem to have any relation to length of life, at any rate a good many old persons are found in pedigrees of cataract, glaucoma, retinitis pigmentosa, Leber's disease, and albinism. But the subject has not yet been at all adequately looked into; and attention may suitably be called to the importance of recording everything we can about age in every member of a morbid pedigree; age of parents at marriage; age at onset of the disease in those affected; age at death, especially when the disease has "anticipated."

Every effort should also always be made to get the order of all the births, or rather of all the conceptions, and the intervals between them. Only in that way can we find out whether a disease tends to affect the earlier or the later births to excess. Karl Pearson's studies of the statistics of phthisis, insanity and crime lead him to believe that the earlier born children are more frequently predisposed to those conditions than the later ones.* Laqueur considered that the first and second born were decidedly less likely to suffer from hereditary diseases of the eye than the third and later births; but his remarks were based on only forty-eight families, containing in all no more than 244 children.† Berry has pointed out that in a particular pedigree of cataract (Fig. 24) the eldest born girl of each sibship invariably had the disease.

**CATARACT.**

(Figs. 11 to 27.)

It is well known that cataract often runs in families, sometimes appearing in several generations. This has been ascertained beyond doubt for several of the best-

* A First Study of the Statistics of Pulmonary Tuberculosis, 1907, p. 25. Boyle Lecture. Also The Problem of Practical Eugenics, 1909, p. 19, etc.
† Laqueur, Zeitschrift f. Praktische Aerzte, 1897, No. 21, p. 8.
‡ For the abbreviated titles of periodical publications referred to in this or subsequent sections see Appendix IX.
marked varieties, and will probably be found true for all as opportunities for investigation occur. When cataract occurs at birth, or early in life, in brothers and sisters, both parents being free and no history obtainable of ancestral or collateral cases, when it is, in fact, what is called *familial* without proof of heredity, there may be grounds for attributing it to some defect of intra-uterine nutrition. But this explanation, unlikely even when the mother is affected, is impossible when the father, not the mother, suffers, for in this case there must be a germinal cause. That the germ-cell, whether male or female, should be able to transmit a well-defined and often almost identical imperfection limited to so small a part of the body as the lens, and often to only a small portion even of it, shows how inconceivably minute the morbid germinal representation may be, and this whether we think of the lens itself or the parts upon which it depends for nourishment at different stages of its growth. From Priestley Smith's researches* we may take it that the weight of the normal human lens at between 20 and 30 years of age is about 175 mgrm. or roughly three millionths of the ordinary body-weight at that time of life.† Yet even this is too much. The opacity in a typical case of discoid (or "Coppock") cataract occupies only a small fraction of the entire lens, possibly one twentieth or even less. The malign germinal influence, whatever it is, presumably acts upon the lens only at its earliest stage, possibly even before the closure of the lens cup, and even then is so limited in its range as to damage no other part of the epiblast; or if another interpretation be preferred, affects no other part of the mesoblast than the minute portion concerned in the nutrition of the rudimentary lens.

In hereditary lamellar cataract the dimensions of the opacity are not so extremely minute, but it also, like the

† Average body-weight of 7 + 9 at 20 to 25 about 130 lb. or say 59 kilogrammes = 59,000,000 milligrammes ÷ 175 = 337,154, or, say, one third of a million.
discoid form, must be due to the influence of the male parent in many cases (e. g. Fig. 11). Some fairly large pedigrees have now been collected, and one of them seems to show conclusively that the discoid or "Coppock" form and ordinary lamellar cataract are essentially the same, and not, as we at first thought, independent forms; so that the two names, discoid and lamellar, should be used only when convenient for descriptive purposes. (In the pedigree furnishing Fig. 12 both forms occurred.) The discoid is probably only the smallest possible form of lamellar, so small that the two layers are united or indistinguishable. The position of the disc or flattened lamella at a deeper level than the nucleus of the normal lens, but in front of the posterior capsule, still awaits satisfactory explanation, though perhaps related to displacement of the nucleus backwards from some developmental cause.*

Opinions have differed for many years as to whether lamellar cataract of ordinary sizes is always congenital, i. e. actually formed before birth, or sometimes postnatal. I think the evidence is conclusive that it may be either one or the other according to the diameter of the opaque shell, but that in most of the hereditary cases the process occurs towards the end of foetal life. The diameter of the human lens at the fourth month of foetal life is about 3·3 mm., at the sixth month 4·5 mm., at the seventh month 5 mm., and at birth 5·75 mm.‡ Between birth and one year old the diameter is about 7·4 mm.‡ If shrinkage of the nucleus is the first stage in the formation of the opaque peri-nuclear layer the dimensions of the opacity may be a trifle less than the dimensions of the clear cortex from which the opacity was formed;

* According to Treacher Collins displacement of the nucleus backwards may occur in the foetal lens as a consequence of faulty backward growth of the lateral lens-fibres. "Developmental Deformities of the Crystalline Lens," The Ophthalmoscope, 1908.

‡ Treacher Collins, Researches into the Anatomy and Pathology of Eye, 1896, p. 5.

‡ Dub, quoted by Parsons in his Pathology of the Eye, ii, 1905, p. 405.
thus if a lamellar opacity measures 6 mm. across, the lens
must have had at least that diameter, or a little more, say
7 mm., before the opacity formed, and in such a case we
should probably be right in concluding that the cataract
developed shortly after birth.* Now the largest lamellar
opacity that has been measured after extraction of the
lens had a diameter of 6 mm.; the ordinary size is from 5
to 5.5 mm. In some it is much less, down to, say, 3.5 and
4 mm., and in these cases of small-sized opacity we should
be justified in assuming that the morbid process had
begun and ended before birth, even if there were no
clinical evidence to that effect. There is, however, enough
of such evidence to be convincing. We have first the
observation attributed by Hulke† to Bowman about the
year 1846, of lamellar cataract found in a kitten a few
days old. Of later observations Hosch in 1897 pub-
lished a case in which a mother had seen cataract in her
baby’s eyes, at its birth, the diagnosis of lamellar being
made by Professor Horner when the child was six weeks
old and the opacity measuring 4 mm. across at the
age of six years. The same woman detected the opacity
in another of her children two days after birth.‡ Mr.
Fisher has given me the case of a female baby (Fig. 14;
IV, 2), in whom he diagnosed dense lamellar cataract at

* Collins, however, concludes that the opacity must always be ante-
natal if the part affected is, as is assumed, the most peripheral layer; or
that if post-natal the part affected is not the most peripheral.

† Hulke (T.O.S., vii, 1887, p. 27), defending in his Bowman Lecture
(in 1886) the pre-natal formation of lamellar cataract, writes as follows:

... “the first distinct recognition of lamellar and zonular cataract
based on dissection was, so far as I know, made by Mr. Bowman, the
subject being a kitten, killed and prepared for lecture in the physio-
logical laboratory in King’s College. The date of this was, so far as
my recollection serves me, 1846, but it might have been slightly
later.” Mr. Hulke, who, as he tells us in another part of the same
Address, was about this time one of Mr. Bowman’s dressers, states that
he (Hulke) wrote down at the time a description of the appearances
although he was unable to find it at the date of the above occasion — 1886.
The kitten was only a few days old.

‡ Quoted as Case 69 in my paper upon “Heredity in Cataract.” R.I.O.H.,
xvi, p. 229.
three months of age, the history being perfectly clear that the opacity had been seen by the parents at fourteen days old, the child not having opened its eyes until then.

In another family of lamellar cataracts, one of the mothers (Fig. 11; Gen. III, 3), told me that she had seen the cataract within an hour or two of birth in more than one of her children, and as in some of them there was a conspicuous white opacity at the anterior pole of the front layer, almost filling the pupil, I have no doubt she was correct in her observation.

After writing the above I had the opportunity of seeing the newly born male infant of a cataractous brother of the above woman (Fig. 11; Gen. IV, 14a), and found the usual small, perfectly well defined lamellar cataract, of about 4-5 mm., in both eyes exactly a week after birth (child born April 19th, eyes examined under mydriatic on the 26th); the cortex was clear so far as a moderately exhaustive examination in the mother's bedroom allowed one to see. Here also there was a dense anterior polar opacity which had been seen by the nurse and mother as soon as the baby's face was cleaned after birth.* In another case of typical small, dense, lamellar opacity (Dearsley) the clear testimony was that the opacity had been seen the day after birth. Lamellar cataract has doubtless been seen repeatedly at less than one year old† Some of the small lamellar opacities have no doubt been described as congenital nuclear or perinuclear cataract.

The condition of the enamel of the permanent teeth in a patient with lamellar cataract helps us indirectly to decide the time at which the opacity was formed. It is

* Later still, on July 3rd, I examined IV, 19 in the same pedigree, a female born on June 19th, i.e. at 14 days, and found precisely similar, small, lamellar cataracts. On the same occasion I was told that IV, 15a, born in September, 1908 (after my original visit, which was in August), was certainly affected; it was a feeble baby and died in May; my informants were the mother, III, 10 and III, 11, who live in the same village, and may both be counted as skilled observers for this purpose.—E. X., July 11th, 1909.

† See R. L. O. H., xvi, p. 228, Case 65, for such an example.
chiefly with the larger specimens of lamellar cataract that the well-known and characteristic deficiency of enamel in the permanent incisors and first molars is found, and Mr. Norman G. Bennett, after careful consideration of the evidence in connection with the date of formation of the enamel, has come to the conclusion that the cause of the deficiency is active from shortly after birth until about two years of age;* and that the correlated lenticular change is probably not ante-natal. He points out that the epiblastic elements of both lens and enamel become isolated within mesoblastic tissue, and that both might therefore not improbably be affected by a common cause of malnutrition.

Now I have myself often noticed that in cases of unusually small lamellar cataract (as well as in its minimal discoid variety), there is usually no defect of the enamel of the permanent teeth. This fact comes out strongly in all the extensive pedigrees of lamellar cataract hitherto published, for in these the opacity is almost invariably small and the teeth good.†

The conclusion, therefore, is that when lamellar cataract is hereditary the small size of the lenticular opacity, and the absence of dental deformity, both point to the cataractous change having occurred during intra-uterine life. It has been assumed that the visible results —lack of enamel for the permanent teeth and lamellar opacity in the lens—mark the commencement of the morbid process, but this can hardly be true, at least for the lens; something is probably wrong both in the lens and the uncalcified enamel before we can detect any

† Exceptions are, of course, seen, but I believe they are not very frequent or very well marked. See R.L.O.H., xvi, p. 231, Case 74 and Case 75, 1 (Elizabeth). On the contrary, for confirmation of the general statement see Cases 72, 74 (mother), and 76, 5 (Louisa). The point is also illustrated in Fig. 12 (from T.O.S., xxviii, p. 226), where the only one (IV, 102) with large lamellar cataract had the characteristic teeth, whilst the teeth were normal in those with small-sized cataract.
change, and thus the number of cases that should be classed as intra-uterine is increased rather than diminished.

\[ \text{Fig. II} \]

\[ \text{Fig. 12} \]

The degree and manner in which small lamellar and discoid cataract may be heritable is shown in Figs. 11, 12, 13, 14, 15, and in sundry other pedigrees not exhibited.
to-day.* Several new pedigrees are, I know, being worked out at the present time by members of this Society.

The descent of lamellar cataract appears to be always continuous, and there are hardly any consanguineous marriages. Lamellar cataract, whether hereditary or sporadic, is, I need hardly say, not confined to either sex:

but whereas in senile and presenile cataract as a whole there are more females than males,* the reverse occurs in lamellar cataract. I have been able to collect, through the kindness of several friends in various parts of the United Kingdom, with the assistance of Mr. J. F. Cunningham at Moorfields, and from published sources, the particulars as to sex in 1887 subjects of lamellar cataract; and find 1166 males to 721 females. Although the excess of males varies greatly in different batches it is present, little or much, in practically every separate return; in a few lots the sex numbers are equal, or nearly so, and in only one is there an excess, and that merely nominal, of females.‡

Isolated cases of lamellar cataract, usually of larger size than in the hereditary cases, are of course common enough, and the same is true of other forms of so-called congenital cataract. Although we may feel sure that some of these would have furnished pedigrees if they could have been followed up, there is at present little doubt that such single specimens may often arise independently of hereditary influence, and be due to some nutritional defect confined to the individual.

I will refer next to the form of hereditary cataract that Mr. Gunn§ has named "coralliform," in which the principal opacities radiate forwards from the central part of the lens, ending anteriorly in expansions that appear to be tubular, and remind one of the separate "mouths" of a madrepore coral. Mr. Treacher Collins conjectures that these tube-like opacities lie in the planes of suture between the lens-fibres.|| I published a large pedigree of this form of cataract in 1905; another

* Nettleship, R.I.O.H., xvi.
† See Appendix II.
‡ The numbers in the separate returns are given in Appendix II.
§ Gunn, T.O.S., xv, p. 119.
* Nettleship, R.I.O.H., xiv, p. 218, Case 58 (Betts).
(Fig. 16), shown at a recent meeting here, I owe to the kindness of Mr. Gunn and Mr. Leslie Paton; for a third, shown at the same meeting, I am indebted to Mr. Treacher Collins (Tomes family), and I know of others. The mode of descent is the same as in lamellar cataract. Although coralliform cataract is probably not very rare it has been apt to escape differential observation, its features not being prominent, whilst the characteristic trumpet-like or tube-like opacities are often intermingled with a number of discrete dots and spots of opacity. It is generally looked upon as congenital because it has been seen several times in children, and only progresses with extreme tardiness; a middle-aged subject of the disease calls himself "short-sighted," and cannot remember ever seeing better; in old age nuclear haze is apt to increase the difficulty. We have, however, no record of coralliform cataract having been seen before the age of eighteen months.† Moreover, the average number and size of the

† In the Betts' pedigree (R.L.O.H., xvi, p. 218, Case 58) Gen. IV, 23 was operated upon for the cataract at two years of age, his brother, IV, 22, at three years, and another brother, IV, 21 at about five. V, 12 was also operated upon at the age of five. IV 11, who died at eighteen months of age, was reported by other members of the family to have had cataract like the others.
characteristic opacities has seemed to me decidedly less in the young than in middle-aged and old subjects, and I am therefore inclined to think that in these people the lens may be clear at birth and for some months afterwards. But few of those affected have had anything done, and not much can be said about the outcome of operations; but there have certainly been several poor results. In the 68 known cases 36, or rather more than half, were females, 29 males, the pedigrees containing them showing a total of 167 persons—males 73, females 75, sex not recorded 19. These numbers are much too small for finality as to sex distribution; they may easily be upset by fuller data, as may be evident when I say that in one large pedigree (Betts) there were 20 affected males to 11 affected females—a great excess of males—whilst in the other five pedigrees the females were in such large majority that, in the whole six, the male excess was more than neutralised, leaving, as just stated, a definite majority of females.

We find similar examples of extreme difference between one pedigree and another in the proportion of affected males to females in many conditions besides cataract; precisely as in normal families where the offspring of some parents may be nearly all male, of others female.* No conclusion as to sex incidence of an hereditary disease, except it be a really sex-limited one, is worth anything unless based on very considerable numbers.

Of other distinct varieties hitherto included under the general title of "congenital cataract" accurate pedigrees will no doubt be forthcoming in future, and several incomplete ones might be quoted. In the best that I am acquainted with, given by Zirm and Bergmeister under the title "congenital stellate" cataract† (Fig. 17), at

* Cf. R.L.O.H., xvi, p. 188, for further facts as to sex-incidence in family cataract.
† Given in R.L.O.H., p. 100, Fig. 54. The four younger generations appear to be completely recorded to date and contain fifteen cases of cataract in about forty persons; but the sixth generation, consisting of young children, may have increased since. Several other interesting pedigrees of cataract are to be found in the same paper.
least sixteen cases of cataract occurred in six generations, the disease as usual behaving like a "dominant."

It is much to be hoped that someone will collect information methodically about the minute vacuoles or dots of opacity so often seen in the lenses of the young; are they congenital, do they run in families, do they lead to cataract, and do they occasion, or only happen to accompany, the asthenopic symptoms from which their owners so often suffer?

Such minute changes have been noticed in several members of congenital cataract pedigrees who were themselves free from the definite family complaint (e. g. in the families shown in Figs. 12 and 13). For the present it is uncertain whether such slight alterations are related to the family cataract or are merely accidental.

Post-natal or acquired cataract (Figs. 18 to 27), is often hereditary, and quite a number of pedigrees have been collected by many observers. A considerable number of these—I do not know what true proportion—show anticipation in generations and sometimes in successive siblings.

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and the pedigrees I have chosen for illustrating the heredity of senile cataract to-day (Figs. 18 to 25), all illustrate this phenomenon in a greater or less degree.

It is impossible to make the family record as full in hereditary senile cataract as in the congenital forms; the older members are scattered and may die with incipient cataract undetected. But we already know enough to say that senile, pre-senile and juvenile
cataract may be transmitted through several generations, that, as in the congenital forms, either sex may pass it on to either sex or to both sexes, and that the descent is, so far as we know, practically always continuous.* Although either sex may transmit, the tendency is, however, in fact, passed on oftener by women than men, and

* Apparent discontinuity, however, is seen in one or two places in a few pedigrees, viz., in R.I.O.H., xvi, p. 390 (Fig. 46); ibid., p. 208, etc. Cases 40, 80, 92, 100. The generation marked as normal in these pedigrees may, however, have contained some individuals with incipient cataract.
seen, hereditary cataract often occurs at an earlier age in the children than in the parents, whilst in those of the same generation it frequently begins at about the same age in each. Fuchs remarks that when senile cataract is a family disease it often comes on unusually early.

This earlier incidence in each generation—"anticipation"—is not known to be accompanied by disease or early degeneracy of other parts of the body; but more data are much needed upon this important point.

Of postponement—onset later in the next generation—there is next to no evidence in cataract, but occasionally when cataract begins at the same age in near relatives it may progress at very different rates in each of them.†

* For good published pedigrees showing anticipation see R.L.O.H., xvi. p. 179, et seq., Cases 2, 4, 5, 7, 8, 15, 46 and 390; Dr. Green's Case Fig. 18 in present lecture, the numbers on which are from Dr. Green's figure and do not represent ages; R.L.O.H., loc. cit., Cases 40, 41 (p. 208); a case published in T.O.S., xxviii, p. 220 (present Fig. 25); another published in T.O.S., xxix, p. 209 (present Fig. 24, giving some of the ages). Also Figs. 21 (Westly, Mr. Fisher's case) and 23 (Helyer, Dr. E. J. Smyth's case) now recorded for the first time, and giving the ages of onset. Figs. 19 (Sichel's), 20 (Louis Stricker), and 22 (unpublished case of my own) all show the same feature and some of the ages are indicated.

† Fuchs, Text-Book of Ophthalmology.

Cases are met with that may be called "hereditary infantile senile cataract," general opacity of the lens coming on quite early in life (Figs. 26, Tatham Thompson, and 27, Berry). In the latter family the author states that
the opaque lenses were much harder than normal lenses of corresponding age.

**Glaucoma.**

*(Figs. and descriptions 28 to 34 in Appendix III.)*

About glaucoma as a hereditary disease I need not say very much, since the known cases (some twenty-four families) have been quite lately collected by Mr. Lawford. So far the most striking features are the strong tendency to anticipation in the younger generation and the continuous descent.* Probably many of us have seen one or two cases of typical primary glaucoma in children, and it will be of extreme interest in future to investigate the family history of these very rare cases.

In some of the glaucoma families there seems to be a possible relationship between this disease and myopia, and an attempt might well be made to ascertain whether, in such families, the two conditions can in any degree replace one another.

The prognosis for operation is another point whose importance needs only to be mentioned; in the members of some pedigrees the prospect is as good as possible, but I am inclined to suspect there are other families in which operation is generally unfavourable.

These and other considerations show how urgently we need the collection of much more material relating to the hereditv of glaucoma.

**Retinitis Pigmentosa.**

*(Figs. 35 and 36 in text; 37, 38 and 39 in Appendix IV.)*

This malady, which, especially since Liebreich’s observations in 1861, has been a rich source of material to those interested in the influence of heredity and of consanguinity in family disease, has lately been dealt with at some length

* Lawford, *R.L.O.H.*, xvii, 1907, p. 57. Anticipation is shown in Cases 1, 3, 6, 7, 8, 10, 11, 13, 14, 15, 16, 17 and 24 of the series. Only one. Case 2, shows discontinuity of descent.
elsewhere,* and to-day I need only allude to some of the principal points and ask attention to some of the unsettled problems in the natural history of this disease and its equivalents. I will keep almost clear of numbers, but may mention that the paper referred to was based upon notes of nearly 1000 families (strictly 976) containing an average of close upon two ascertained cases of the disease in each. There was proof of heredity in one quarter of the families and of consanguinity of parents or ancestors of those affected in another quarter (I use round numbers, the

**Fig. 35**

exact figures are given in the original paper). In the remaining half, where no history of either consanguinity or heredity was recorded, the notes were often very imperfect, and there can be no doubt that such a history would often have been forthcoming had more pains been taken.

In the largest pedigrees† of retinitis pigmentosa the descent of the disease is continuous from parent to child, no healthy member ever producing affected offspring (Fig. 35).

* Nettleship, R.L.O.H., xvii.
† To the fully recorded pedigrees quoted in the paper above referred to Snell has since added another in T.O.S., xxvii, 1907, p. 217.
But we meet with some in which the direct line of transmission is without doubt interrupted by a healthy generation (Figs. 36 and 37).* I say "without doubt" advisedly, because the statement so often made by a subject of this disease, that his or her parents had perfect sight and were not related by blood, though usually correct, does certainly need revision in some instances.

* Figs. 37, 38 and 39, not previously published, are fully described in Appendix IV.
A similar caution is necessary as to the families in which only a single example of the disease can be discovered (Figs. 38 and 39); in some of these the disease may have existed latent in several members, but become
manifest only in the solitary one who was reached by some efficient determining cause. In others deafness or mental defect, easily omitted from the record unless directly sought for, may take the place of the eye disease (as in Fig. 39), and thus partly or wholly restore the continuity.

In respect to consanguinity we have to confess that but few of the records tell us the source and kind of cousinship, an omission that may very much lower their value. In future when taking the family histories of persons whose parents were cousins, it will usually be easy to record whether they both belonged to the affected side of the genealogy or not, and whether they were children of two sisters, or of two brothers, or of a brother and sister, or of a sister and brother.

Of the persons seen at all ages with retinitis pigmentosa a considerable majority are males (at least sixty males to rather less than forty females). This fact may, as suggested before, be an expression of some wider law; but two other interpretations suggest themselves for the time being, viz., either that the females in these families die in excess before they are old enough to show the disease, or that the malady occurs most in the families that contain an excess of male births. It may be of interest to note that a marked excess of males is also seen in the chronic renal diseases, and in diabetes, whilst the reverse is found in the interstitial keratitis of congenital syphilis.

Next to heredity and consanguinity comes the influence of ill-health in bringing out a liability to retinitis pigmentosa where, but for such an exciting cause, it might have remained latent. Probably such an influence may sometimes explain the solitary cases. Of such determining causes some of the acute exanthemata seem to be the commonest, but probably tubercle and syphilis and in rare cases even severe loss of blood may have the same effect (Fig. 38). We may suppose that anything capable of damaging the arterioles might determine the onset of
retinitis pigmentosa in a choroid and retina predisposed to the disease. This part of the subject is well worth more attention.

Retinitis pigmentosa may set in very early in life or even before birth; and on the other hand there is reason to believe that its advent is sometimes delayed until quite an advanced age. The amount and distribution of the pigment varies a great deal, but the extreme periphery of the retina is usually free even in cases of long standing; when visible vessels are ensheathed in pigment such vessels are, in my experience, always veins, i.e. the pigment travels in the direction of the blood-current. Retinitis pigmentosa sine pigmento is nearly always merely retinitis pigmentosa at an early stage before the pigment has accumulated in the superficial retinal layers and become ophthalmoscopically visible; but in rare cases, although the retinal atrophy progresses, pigment does not travel inwards in any quantity, and then the term sine pigmento may be appropriate even at a later stage.* There does not seem to be any correlation between the quantity of pigment as judged by the ophthalmoscope and the colour of the patient's hair, irides and choroid. Retinitis pigmentosa does not hinder fertility; the subjects of the disease often have very many brothers and sisters, whilst if they themselves marry they frequently produce many children; whether the average fertility is above

* A case which may throw an important side-light on the seat and nature of the early changes in retinitis pigmentosa has lately been published by Bordley (Johns Hopkins Hosp. Bull., September, 1908). In a negro pedigree night-blindness occurred during five generations, and progressed through gradual constriction of fields to total blindness; in the older members there were ophthalmoscopic signs of pronounced arterio-sclerosis, but even in them no other changes and no pigmentation. In the pedigree of forty-three individuals thirty-four are marked as night-blind. There are some improbabilities in the record, since it is stated that there is no record of any normal-sighted member having had children, and that all eight children of one night-blind parent were affected. The occurrence of night-blindness in relation to disease of the liver is the subject of an interesting section in Parsons's Pathology of the Eye, iv. p. 1292.
the normal can perhaps hardly yet be either asserted or
denied. Until more, and more precise, data are collected,
we cannot tell whether the order of the birth of the
children, or the age of the parents at marriage, have any
influence in determining the disease. The relative fre-
quency with which the same fundamental cause produces
retinitis pigmentosa in one, deafness in another, and
mental inferiority in a third member of the same pedi-
gree, has not yet been worked out; but we find on the
other hand that certain stocks produce only retinitis pig-
mentosa and others only the equivalent deafness. It is
especially noteworthy that the largest pedigrees of the
retinitis are quite free from the other degeneracies, and
the survival of such families is probably due to this cir-
cumstance.

I should like to return for a few moments to the two
kinds of descent, continuous and discontinuous, met with
in this disease. As I said in my introductory remarks,
continuous descent in Mendelian terminology usually
means "dominance," and interrupted descent, except in
sex-limited conditions, means "recessiveness." Retinitis
pigmentosa, although more frequent in the male, cannot be
put into the sex-limited class with colour-blindness, Leber's
disease and others in which women very seldom suffer.
Therefore since, as Figs. 36 and 37 show, pedigrees exist
in which a healthy generation always comes in the direct
line between two that contain retinitis pigmentosa, or
one of its equivalents, and since the normal "carrier"
may be of either sex, the disease must then, in Mendel's
terms, be recessive.* And yet in the largest pedigrees the
descent, as I have already said, is always continuous and
the disease therefore dominant.

This is as far as we can go at present. In the discon-
tinuous pedigrees we can make sure that the intervening
generation has neither eye disease, deafness nor mental
defect; but there may perhaps be other morbid states,
other equivalents of retinitis pigmentosa, that give no con-

* For the data see Appendix I d.
spicuous signs, and at present, therefore, escape detection. This is mere speculation for future work; the arteriole disease leading to retinitis pigmentosa, or to deafness or mental deficiency, may possibly in some cases affect an entirely different region, e.g., the arterioles of kidneys or liver or even of the hands or feet. But at present, if we are to test our data for retinitis pigmentosa by the Mendelian scheme, we must assume that change in mode of descent means change of dominance, however improbable this may appear. We were formerly content to say that a given disease or character could become latent for a generation or more and then re-appear, either capriciously, or perhaps when re-inforced by a marriage between cousins. But the Mendelian conception of pairs of complementary characters, one of which, in virtue of some attribute, dominates or prevents the appearance of the other, does not in its simple form allow the dominant to lose dominance or the recessive to gain it. But if the members of a pair representing a given character, say retinitis pigmentosa and its absence, could, without losing their affinity, become linked with, and influenced by, a pair representing some other character, a change of dominance in the original pair might conceivably be brought about, the second or linked character not necessarily attracting attention.* This is only the crudest possible indication of the ingenious hypothesis of "coupling," by which some of the complex and unexpected results obtained in experimental breeding are explained, and which appears to have been verified by control experiments in certain cases.

Of the varieties of retinitis pigmentosa, retinitis punctata albescens has, so far as I know, never been seen in a well-marked form in more than one generation, and if it is not a new departure, a "mutation," it must, in some cases, have skipped several generations.

Hereditary Night-Blindness.

(Figs. 40 to 43 in text; 44 in Appendix V.)

Two sorts of hereditary night-blindness are met with which may be conveniently taken next, although they are, so far as we can tell, absolutely distinct from retinitis pigmentosa, and probably also from each other. Both are, so far as can be ascertained, present from birth, stationary, and not associated with any other defects or degeneracies. In one of them the defect (it seems hardly right to say disease) affects both sexes almost equally, descends continuously through either parent, and is not connected with any other peculiarity of the eyes or sight, nor with any unnatural appearances at the fundus. Besides the now well-known genealogy originally published by Cunier, there are only about half a dozen recorded pedigrees of this abnormality (Figs. 40 and 41 show two of them). Probably, however, the condition is less rare than

*A list of the cases, and the pedigrees of some of them given in Appendices I, 6 and ni, and V.
we suppose, and now that attention has been drawn to it we may hope soon to hear of more cases. There has been no opportunity for anatomical examination, and nothing is known of the intimate nature of the night-blindness; we cannot even be sure whether its seat is retinal or cerebral.

In the other group of hereditary night-blindness shown in Figs. 42, 43, and 44 (Appendix V), the leading features are limitation to males with descent through normal-sighted females and myopic refraction, but visual acuity with cor-

rection often subnormal. Slight changes are sometimes found at the fundus, but even when present they are not constant either in character or situation. Considerable myopia has certainly been present in childhood in some of them, and perhaps in all; 3-5 D. to 9 D. are the usual figures, 11 D. the maximum recorded. No case has been found with steadily progressive myopia or severe myopic changes at the fundus. Colour-vision was normal in such as were tested. Nystagmus has been noticed in a few. Nothing is known of the nature of this condition; but the association of early myopia, frequently defective central
vision and occasional nystagmus, and the occurrence in some cases of various slight ophthalmoscopic changes, suggest that in this group the night-blindness, although congenital, is truly pathological, the result of a limited intra-uterine elongation of the eyeball interfering with the development of the choroid and outer layers of the retina. But why the condition should usually be limited to males is as great a mystery as in other sex-limited conditions. It is to be noted that ordinary myopes sometimes complain of seeing worse in twilight, but the significance of the symptom in such patients is open to more than one interpretation, and its proper analysis is full of difficulties, as I believe some of my friends who are investigating the subject have found. About a dozen fairly good pedigrees of this sex-limited myopic night-blindness are known, and may be found in my paper already mentioned. Consanguinity of the parents was present in at least three of them.

Since the publication of the paper in which all the above cases are given I have obtained the new and quite characteristic, although small, pedigree of this sex-limited myopic night-blindness, fully described with its Fig. 44 in Appendix V. There was no consanguinity.

Another case (44a), seen at the same time as the above, was less thoroughly examined, and is given in the same Appendix for what it is worth. In this case two of the three affected siblings were girls; the parents were first cousins.

I owe these two new examples to the kindness of Mr. W. J. Cant and Mr. Clements, of Lincoln, who courteously allowed me and Mr. C. H. Usher to examine the affected members for ourselves last autumn. To the Rev. C. N. Usher, of Wellingore, I am indebted for the kindness and trouble he took in arranging for our meetings with the patients at his house.

Some few small pedigrees of night-blindness are found in which, though descent is discontinuous, the disease affects both sexes; myopia appears to be common in the
affected members, but nothing like universal, and V. corrected is also apt to be subnormal. The relations of this group will have to be worked out by future observers.

**Leber's Disease.**

(*Figs. 45 to 52 in text, 88 being inserted at p. lxii.*)

The hereditary optic neuritis, or, as it is often called, optic atrophy, described by Leber, is so well known that I need dwell only upon certain points that call for further study.

Although nearly always symmetrical and usually simultaneous in onset, it is sometimes unequal in intensity in the two eyes even to the degree of occasionally leaving one eye untouched, as in a case by Johnson Taylor,* or but slightly affected, as in Norris’s case (Fig. 49, IV, 13), whilst an interval of weeks between one eye and the other is not very rare, and even years occasionally intervene (see Fig. 51). After an acute or subacute onset the climax is generally reached in a few weeks or months and no further change takes place, the leading permanent feature being a central or nearly central scotoma that varies in size and density in different cases. Peripheral loss of field is much less common. Total blindness is said to ensue in rare instances, but I believe this has generally rested on lay testimony. The usual age of onset is about 20 years. The subjects are males in a large majority of cases, but descent nearly always takes place through the unaffected mother. Consanguinity of parents is but seldom met with. In only a few cases do we find a history of other neuroses (most often epilepsy) in the patient or his relations. The following are good illustrative pedigrees of Leber's disease:†

* T.O.S., xii, p. 146, Case 3. June 12th, 1909: Mr. Johnson Taylor has kindly re-examined the members of this genealogy quite recently and brought the history down to date. See Fig. 110, Appendix VI, a.
† For the other data on Leber's disease see Appendices I, I, and VI.
1893. (Fig. 45.) Gould (George M.), Pan-American Congress, Section Ophthalmology, and Annals of Ophthalmology and Otology, ii, p. 303.

I, 1, blind rather late in life. II, 1 age at onset unknown, II, 2 at 40. III, 1 affected, died at 86; III, 2 affected at 28; III, 4 doubtful case, died at 40; III, 3 died at 62; III, 5 died at 74 and was blind in old age, cause unknown.

IV, 7 died at 40. V, 3 affected at 23, V, 4 at 28, V, 5 at 33, V, 8 at 52, V, 15 at 34, V, 16 at 28, V, 17 at 23, VI, 5 at 27. Author observes that the disease is dying out for want of child-bearing daughters in the later generations.

1893. (Fig. 46.) Gould. Ibid. Author’s Cases 3 and 4.

III, 2 affected at 25; his 4 siblings all living, 27 to 12. III, 5 affected at 24, seen at 36; III, 10 failed at 21, seen soon after. All very severe cases. Ages of III, 5 to 11, from 36 to 18; III, 12 and 13 died of “croup” at 3 and 2 years. II, 3 sight bad after smallpox at 40;
lived to 65. All in I and II lived to good age, and I, 3 was alive at 93.

1908. (Fig. 47). Hancock, R.L.O.H., xvii, p. 167.

Twelve cases, all males, in 5 generations; 6 recovered practically full V. in from twelve to eighteen months, *viz.*, I, 1; IV, 5, 8, 9, 13, 14; and III, 5 improved enough to resume business. III, 1, 7, 8; IV, 7 and V, 2 did not recover. IV, 9 very epileptic since 16; aged 26 at record. The following were intemperate in alcohol and tobacco: III, I, III, 7; IV, 7, IV, 13, IV, 14. The following were very moderate in alcohol and tobacco, III, 5; IV, 5, IV, 8, IV, 9 (total abstainer from alcohol,
small smoker, epileptic, takes bromide). The age of onset was as follows: I, 1 about 25; 25 in III, 1, 5, 8, IV, 7 and 9; 26 in IV, 13; 30 in III, 8; 31 in IV, 14; 17 in V, 2; 41 in IV, 5. No early deaths. Descent strictly according to rule. Of the 12, 8 had no children and apparently did not marry; the 4 who married had at least 20 children between them. As to present age, IV, 14 is now (1909) 42; IV, 13, 41; IV, 9, 29. I, 1 died at a ripe age, about 100 years ago. No consanguinity.

1898. (Fig. 48, inserted at p. lxii, supra.) Klopfer, Inaug. Dissert. Tubingen.

The 8 in VII (author's Case 5) reported to have had the same disease. In VIII (counting from the left), No. 1 (author's Case 4) was affected at or about 20. VIII, 4 (author's Case 2) affected in 24th year, seen soon after; three years later, no recovery; V. fingers. VII, 5 (author's Case 1) affected at 21, seen six months later; five years later, no recovery. VII, 6 (author's Case 3) affected in 20th year, seen three years later. Much consanguinity, but disease probably derived through VI, 23.

1884. (Fig. 49.) Norris, T.A.O.S., iii, p. 662.

Four generations, 14 cases. I, 1 female, no details, but family records seemed trustworthy. II, 1 affected at 14, died 45; his first-born (III, 1), 45 at date of record, normal, had two children (IV, 1 and 2) of whose sight nothing known; second son, III, 2 affected at 18, died at 22. II, 2, normal, five children; III, 3 affected at 15, died childless at 50; III, 4 affected at 35, living, aged 50 at date; III, 5, 48 at date, and her husband, examined and found quite normal; they were not consanguineous; III, 7 normal, childless at 40; III, 8 affected at 19, aged 40 at date; two sons (IV, 14 and 15), aged 6 and 3 at date and normal. IV, 3, affected at 18, died at 30; IV, 4, affected at 14, died at 18; IV, 5, normal, 20; IV, 6, 18, no information to be got. IV, 7 to 13, issue of III, 5 and 6, all affected and all examined (author's Cases 1 to 7) — IV, 7 aged 22 at date (author's Case 5), failed at 14, stationary 3 years, then improved so that she could sew, and at date
V. 6/8, IV, 8 (author’s Case 6) affected at 19, no recovery in a year; V. 2/6, IV, 9 (author’s Case 7) affected at 18, under care at date; IV, 10 (author’s Case 1), affected at 14, V. 6/60, no recovery in nine months; IV, 13 (author’s Case 4), affected at 7, seen at 8, V. of R. much worse than L., marked neuritic appearances in both, R. 6/60, L. 6/7; IV, 12 (author’s Case 2), affected at 8 1/2, seen at 10, V. 6/18, slight neuritic and atrophic changes; IV, 11 (author’s Case 3) affected at 8, seen at 12 with V. 6/18 to 6/4 and O.Ds. pale.

1901. (Fig. 51.) Mathieu (Jules), *These de Paris*, No. 117, p. 51.

[Diagram]

II, 4 failed at 50, no recovery, lived to 69; her first child (III, 1) born several years before marriage, affected at 40 (author’s Case 1). III, 2 born several years after III, 1, affected at about 40 (author’s Case 3); III, 3 married twice; children by both husbands, but the two paternities not separated on pedigree; III, 4 affected at 22 (author’s Case 2), living at record, not married; III, 5 died at two days; III, 6 died at 34, sight was "beginning to decrease." III, 5 had two illegitimate sons, III, 7 and 8 (who assert that they had different fathers), both affected at 28 and 32 respectively (author’s Cases 4 and 5). IV, 1 to 6, 14 children of III, 1; IV, 1 (author’s Case 6) affected between 16 and 19; IV, 4 (author’s Case 7) attacked at same period of life, recovered completely; IV,
3 all died young, and IV, 6 all dead at record; IV, 9 (author's Case 8) affected about same age as IV, 1 and 4, seen at 25, improved very decidedly 2 to 3 years after onset. In III, 1 there was interval of 8 years between the two eyes, R. before L.; and in III, 8 between 2 and 3 years, R. before L. All the five affected ones in III had pterygium.

In respect to prognosis, the chance of recovery has, I think, been put too low—how much too low it is impossible to say. There do not seem to be any signs by which we can forecast the future for a given attack; but

with our present knowledge I am sure we not only may, but should, speak hopefully about any case seen within a couple of years from the onset or even longer. I find records of at least 25 affected persons (22 males, 3 females), in 16 genealogies who recovered either perfect or quite useful central vision; and minor degrees of improvement are probably rather common.* Most of these recoveries took place between the ages of 20 and 30, viz., at the period when the disease is most frequent; but 2 were in children. In the same genealogy and even in the same sibship some may recover and others not; thus in Hancock's recent remarkable case (Fig. 47), 6 recovered out of 12 attacked; in

* Appendix VI, b.
one of Leber's earliest cases (1871), 3 siblings were attacked and all recovered; and in a case of my own 2 cousins recovered out of 4 attacked. In the recovered cases many different lines of treatment had been tried and we cannot be sure that any of them had much effect. A very important feature in these cases is the length of time that may elapse before noticeable improvement of sight begins, often 12 or 18 months, and, in one case, if we can believe the history, as much as 3 years. This possibility of considerable delay in recovery should lead to a more hopeful prognosis being given in future cases; one can, indeed, hardly doubt that the list of favourable results would have been longer had cases been more frequently followed up. Probably some of the "astonishing cures" of long-standing "blindness" of which we hear from time to time may have been examples of delayed recovery from this disease.

We shall probably be right in attributing certain cases that individually resemble the type but are without family history of the disease to the same essential cause, whatever that may be. Such cases, sometimes diagnosed as tobacco amblyopia, do not improve on ceasing to smoke and sometimes show contraction of fields as well as central defect. Interesting communications on such, possibly borderland, cases have been made by Lawford, and Edgar Brown.*

There is a tendency to anticipation in Leber's disease, both in successive generations and to a less marked degree in successive births in the same sibship; but the phenomenon is not so pronounced as it is in successive generations affected by glaucoma or by senile cataract.

Anticipation in successive generations was shown in 14 pedigrees out of 31 that gave the necessary information, the difference between ages of onset in the elder and younger generation being from 15 to 25 years. In 11

* T.O.S., x, 1890, p. 166.
others the age of onset was practically the same in both generations. In only 3 cases was there evidence that the disease began later in the second generation than the first.* In the rare cases where a mother is affected the onset of the disease at an earlier age in her sons than in herself can hardly be called "anticipation," because the disease usually appears earlier in all males than in all females.

Anticipation in successively born brothers or sisters is not so frequent, being found in only 29 out of 82 completed sibships containing 2 or more cases of the disease. In 14 others the disease began at a later age in each successive birth, in 16 at practically the same age in each case, and in 23 the ages of the successive siblings when attacked varied irregularly. The differences of age-onset are of course much less between successive siblings than between successive generations, the age of onset in the junior sibling being usually about 3 years less than in the senior, and seldom as much as 5 years.†

It is of interest to inquire whether when a mother suffers from the disease her children will have it in greater numbers or with a different sex-distribution than if she had merely carried it as a potential in the usual manner?

In the corresponding case of congenital colour-blindness, Professor Bateson finds, as already mentioned, that if a woman be colour-blind the history always shows that her father was so, and that if she have sons they will all be colour-blind; whereas we know that in the ordinary case, where the mother is unaffected but carries the defect, only a proportion of her sons will have it.‡ Careful examination of the corresponding data for Leber's disease shows that it does not conform to this rule§:—I. A man with Leber's disease who has children seldom transmits it;

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* Norris, *Amer. Ophth. Soc.*, iii, p. 673, Cases 58, 75 and 93 in Appendix VI, b.
† For the data relating to anticipation see Appendix VI, b.
§ The data for what follows are given in Appendix VI, b.
in 11 pedigrees 23 affected males became fathers and had 100 children who lived long enough to have the disease, the males and females being in about equal numbers; only 6 of the 100 became affected, 4 males and 2 females. II. An affected female has generally, like an affected male, had both parents normal; it is rare to find that she had an affected father or an affected mother. III. An affected female may transmit the disease to her children of either sex, but of her sons some usually escape. It is not clear why there should be this difference between colour-blindness and Leber's disease in the transmission to and by an affected female. We may note, however, that the one condition is an actually innate physiological defect, the other a disease of which in the vast majority of cases we cannot say more than that the liability to it is innate.

But although a woman suffering from Leber's disease does not, as a rule, give the disease to all her sons, she does give it to a larger proportion of her total issue than she would do if she only carried it incomplete or latent in the ordinary way:—I. In 12 completed sibships, where the mothers were affected but the fathers normal and the siblings of the necessary age normal, 64 children survived and 33 had the disease, viz., 21 males and 12 females. II. In 38 similar sibships where the mothers were normal, but carried the disease (the fathers also being normal), there were 215 eligible children of whom 65 got the disease, 64 males and 1 female. In the first case one half, and in the second case rather less than one third of the children suffer, and the difference is almost entirely due to the excess of affected daughters in the first group, viz., the group where the mother had the disease.

In one extraordinary case (Case 49, supra) all 7 children (4 male, 3 female) of the normal and unrelated parents (III, 5 and 6) had the disease, and had it unusually early in life.

The number of children born in the childships containing cases of Leber's disease is seldom less than the
normal, and is sometimes very large. In 44 completed childships, each containing one or more affected, with both parents normal, 28 contained 7 children or more, 10 of these having from 10 to 14 each, and one had 16. In 19 similar affected childships where one parent had the disease, 8 had 7 or more children, 2 of these having 10 and 2 having 14, 11 contained 6 or less. It is noticeable that in both these sets of sibships (44 and 19), those in which females as well as males were diseased averaged rather larger than those with only males diseased, as 8·25 to 7. The normal branches of affected stocks are seldom fully recorded, but in such of them as seem complete we find several containing 9 and 10 children each. So it is clear, on the whole, that the stocks in which Leber's disease is found are quite up to the normal in fertility, that the sibships in which the disease occurs are larger than normal, and frequently very large; and further, that the affected ones who marry often beget full families.

But if the births are too many the early mortality is large, sometimes very large, especially among the male children. This has been pointed out by several writers, notably by Gould. We find that in the sibships that have been reduced by a high early death-rate, the proportion of the survivors who get the disease is larger than in those sibships where few or none have died; almost half of the former became affected, including several females, but where no early deaths took place the proportion affected is one third.

One naturally suspects that a disproportionate number of those who died early would have suffered from the disease had they lived long enough, and that thus early deaths may contribute to the extinction of the disease; but this, of course, is at present a mere guess.

In several families there has been a high mortality from phthisis, but the number of such families is too small to justify any inference.

The characters of the disease are usually the same in
each individual at whatever age the attack occurs. In pedigrees where it comes on early, i.e., before the fourteenth year, some females are usually affected; the reverse is true of the pedigrees where the onset is deferred until 30 or later, female cases being found in only a few of these. There seems, therefore, to be some connection between early age of onset and the female sex.

We have not much information about the longevity and causes of death of those who are afflicted, but the records show that a fair number were alive at 50 and later, up to 75; one died at 69, a pair of brothers at 72 and 73 respectively, and one man at 86. I have lately (thanks to the kindness of Mr. Doyne) seen a woman, now 71 (Fig. 52, II, 2), who has had the disease since birth or infancy; whilst in another woman, seen at 75 by Mr. Sym, the disease did not set in till she was 51. The subject is worth following up.

Case and Fig. 52, 1898 to 1909, unpublished, kindly communicated by Mr. Doyne, is a very important one.

I, 3 had some defect of sight, but not so bad as daughter (II, 2), and could do needlework and read; it may have been only myopia. Had 14 siblings (I, 2); her first child (II, 2) illegitimate, aged about 71 when seen (1909), sight failed in childhood, and has remained same ever since; symptoms and appearances characteristic in her and the other cases, all of which have been seen; has been very deaf for many years. I, 2 afterwards married 1, 4, and had by him 2 sons and 4 daughters (II, 3 and 4), who all saw well. II, 2 married apparently after 30. Husband (II, 1) of about same age, afterwards lost both eyes from a boiler accident. They have had 4 children, who are all living; III, 2, aged 31 (1909), who has one living child, IV, 2, aged 8, normal, and one who died (IV, 1); III, 3, aged 32, normal, has 3 children (IV, 3, 4, 5), aged 5 years to 10 months, all normal; III, 4 seen by Mr. Doyne at 20 and by E. N. at 30 (1909), "born with the sight as it is now," is married to III, 1, who was examined and found normal; III, 5,
aged 29 (1909), single. IV, 6 to 10, issue of III, 1 and 4. IV, 6 believed to have seen well till about 3 years old, when he began to have to look about for things; when about 8 Mr. Doyne found myopia from 7 to 8 D. in each eye, with 3 D. of As. in R., V. fingers, O.Ds. pale; ordered − 7 D. In March, 1909, I (E. N.) found IV, 6 about the same; could read 3 or 4 J. slowly, held very close. IV, 7 saw well till about 4; at 5 Mr. Doyne found O.Ds. pale, V. fingers at 3 ft., refraction H. 2 D., no As.; in March, 1909, I (E. N.) found her in the same state. IV, 8 died at nine months with good sight;

IV, 9, miscarriage. IV, 10 noticed by mother to see badly when two months old, or even earlier; at six months Mr. Doyne found irregular nystagmic movements, and noted that child did not follow a light; in March, 1909, at 1 1/2 years, I (E. N.) found the O.D. decidedly pale on Y.S. side, and the mother said the child saw so badly that he would run against the table or chair, and "has to look under the light to see." IV, 6-10 all suckled, intelligent, and show no other degeneracies; same general remarks apply to III, 4 and 5. Circumstances prevented proper examination of Fs. in any of these subjects, but it was quite evident from the manner in which they looked at objects that sight was best towards periphery; central
vision very bad in all, and they all have more or less nystagmus; O.Ds. much alike in all, pale all over in the adults, but especially so on Y.S. side; in the children nasal side is fairly coloured, but Y.S. side quite pale. None of those affected, from the grandmother (II, 2) to the youngest (IV, 10) are getting either worse or better. No consanguinity.

Apart from early deaths, we do not meet with any very prevalent morbid tendencies in these families. The most frequent seem to be epilepsy and aggravated hysteria; such conditions are recorded in several families, sometimes in the subject of the Leber's disease, sometimes in a sibling or a maternal relative. In one case Basedow's disease occurred in the sister of a man with Leber's disease.* Insanity or mental defect is recorded in three affected brothers, and in one other affected male, whose affected brother was epileptic. Diabetes is mentioned in two or three cases. At least two males, one affected, the other normal, and perhaps a third affected male, were congenitally colour-blind†—probably a normal coincidence; likewise, the association of retinitis pigmentosa with Leber's disease, observed by Wider, Coppez, and H. Schmidt, appears to have been purely accidental.

As to sex, I find about 60 females against about 300 males.‡ It has been said that in females the disease tends to come on with special frequency at about the climacteric, but little evidence of this can be found. Of the 57 affected females the disease came on before the age of 13 in 13 cases, almost equally spread over the

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* Case mentioned (on Liebreich's authority) by A. Terson in his article "Maladies de l'Œil" in the Traité de Chirurgie of Dentu-Debet, T.V., 1897, p. 198.
† Cases 106, 117, 147.
‡ The exact number depends upon the inclusion or exclusion of a few doubtful cases. There are probably more than 300 males to 60 females; small pedigrees with only males affected are relatively common and not always recorded, but cases in females have been more generally published on account of their rarity. Perhaps some few of the cases in females that I have included would be rejected as atypical by others.
25 years between 14 and 39 in 26 cases, in the critical years between 40 and 50 in only 10 cases.

We commonly notice that the age of onset is almost the same for all cases in the same genealogy—all very early in one, all unusually late in another. But marked exceptions are seen, as in a case (Fig 103) where one of two brothers lost his sight at 21, the other not until 60. The latter was diabetic at about the same period, and one cannot help suspecting that had he not been so he might have escaped the disease of his optic nerves.

Consanguinity of parents is seldom met with; I find at present only 6 or possibly 8 cases. In Arnold Knapp's case, given fully below, a woman with Leber's disease (Fig. 50; III, 3), whose father was also affected, married her normal first cousin, the son of a normal brother of the affected father. In Gunn's case* a woman with the optic nerve disease from childhood married her healthy first cousin and has so far two children, female and male, both affected at 3 or 4 years of age.

1904. (Fig. 50.) Knapp (Arnold), A. of O., xxxiii, p. 383 (1904, and further information, March, 1909).

I, 1 and 2 normal. II, 1 failed at 24, recovered slowly enough to read, is now 67 (1904); had 2 brothers and 3 sisters, 1 of whom died at 17, 1 at 30, 1 at 67 and 2 still living; none affected. III, 1 to 4 all failed during their early school years. I, 1 has 2 sons, normal (1909). III, 2 lately married (1909). III, 4 married, no issue (1909). III, 3 married son of her father's brother; 6 children, of whom IV, 2, 4 and 5 all failed at early school age. IV, 3 and 6 remain normal, ages 15 and 9 in 1909. IV, 7 died at 4, with good sight.

This scarcity of consanguineous parentage in the history of Leber's disease is what we expect in a sex-limited affection. In such a disease, as already shown (Figs. 1 to 5), when two unaffected cousins marry only one, the wife, can possibly carry the disease, since the husband will so far as we know always show it at the proper age if he contains

* Fig. 45, Appendix VI, a.
it*; here, therefore, the cousinship does not increase the risk to the children. Next, if the cousinship comes through the wife's unaffected father she will not, any more than her unaffected husband, contain the disease, and might have been unrelated so far as risk of this particular disease is concerned. The only case in which a cousin marriage increases the risk is when the man is affected with the disease and his cousin wife carries it latent, deriving it either from her affected father or through her mother from an affected male of an earlier generation.

The differential diagnosis of Leber's disease, generally easy, may now and then be difficult when we have to distinguish it from familial optic atrophy associated with tower-skull and other cranial deformities.† I have provisionally included the cases by Rampoldi and Suckling, which, although probably genuine, presented some unusual features and are not described in sufficient detail; and one of Higgen's cases is also included, although the author seems inclined to think that syphilis in the mother may have taken a share in causing the optic atrophy that

* But compare the suggestion on last page as to the possible influence of diabetes or other agencies in exciting a latent tendency to the disease.

† Patry, "Contribution à l'étude des Lésions Oculaires dans les Malformations Craniennes," Thèse de Paris, 1904. Several cases of tower-skull or oxycephaly will be found in the T.O.S. and elsewhere in British literature.
occurred in three of her children; (these three are Figs. 181a, 181b and 181c, Appendix VIa).

Cases of family or hereditary congenital optic atrophy have been described as if forming a group in some way distinct from Leber’s disease. I believe that most of these are true Leber’s disease setting in very early in life or perhaps sometimes before birth. Certainly several of those given in Appendix VIa present the classical symptoms; and I doubt whether we have at present sufficient evidence to justify us in setting up any of these family infantile cases as a distinct group.

There does not seem to be any connection between juvenile Leber’s disease and the cases of progressive failure of sight with slight macular and papillary changes, and coincident mental degeneracy, in children, described by F. E. Batten, Mayou, Sydney Stephenson and others.*

As outlying cases the following may be mentioned:

I have once seen double chronic stationary central amblyopia with partial optic atrophy coming on in an old man at about the age of 76, and ordinary acute retrobulbar neuritis, first in one eye, and after a year’s interval in the other, in his daughter aged 23–24 who had symptoms suspiciously like early disseminated sclerosis (P. 49, 62 and 52, 146).

I also saw in 1881–1882 retrobulbar neuritis limited to one eye and following an attack of diplopia due to paresis of one of the rotators, in a man æt. 50 years,† whose daughter was under Mr. Holmes Spicer’s care 20 years later, æt. 33 years, for retrobulbar neuritis of left eye, which relapsed slightly 4 years later (1906), when she also had threatenings of disseminated sclerosis.

Acute retrobulbar neuritis has also been seen in two sisters in more than one instance.

* T.O.S., xxiii, 1903, p. 386, and xxiv, 1904, p. 142, et seq.
† Ibid., iv, p. 210, Case 17.
Hereditary Nystagmus (Sections \(a\) and \(b\)).

Without attempting a thorough search, I have found about twenty-five pedigrees in literature under the title of "Hereditary or Family Congenital Nystagmus," and have added a few of my own. Generally speaking, more attention has been paid to the oscillation than to its causes, so that we are often unable to classify the cases in any natural order. One is almost reminded of the time when every case of obscure blindness was called "amaurosis."

It may safely be asserted that infantile nystagmus, as a family affection, is in the vast majority of cases a symptom of defective sight, and not due to a primary central cause. Our business is to find out the nature of the amblyopia, and to arrange the cases accordingly. The defect of sight is always dated either from birth or early infancy; it is often due to some affection that causes little or no ophthalmoscopic change; and as the oscillation, especially in a baby or young child, often renders a refined ophthalmoscopic examination impossible, we may be unable to make an exact diagnosis until the patient is old enough to answer questions and have his visual functions tested. In some cases, however, we can come to a conclusion before that period.

Perhaps the first thing to note is that nystagmus in general is more easily produced in some persons than others; this is evident enough in cases where it follows blindness; and I am told that coal-miners and others do not all acquire it with equal facility under like conditions of work.* The same surely must be true for nystagmus produced in early infancy by defective sight; some infants will learn steady fixation sooner, more readily and with less perfect vision than others; those who have most

* The last letter I received from Mr. Simeon Snell, written a few weeks before his death, was in reply to a question I had asked him on this subject; it was to the effect that some coal-miners are more susceptible than others.
difficulty are likely, other things being equal, to develop nystagmus. Therefore, from time to time we meet with nystagmus dated from birth, or soon after, with only slight defect of visual acuity, such as may perhaps be due to nothing more than a moderate degree of astigmatism; but as a rule the defect of acuity required to produce the oscillation is considerable.

Nystagmus dating from early life often becomes less marked in later years; it may even cease entirely, although such complete cure seems to be rare. Nystagmus is often less marked in some one position of the eyes, a position constant for the same person, but not the same for different persons; it also varies much in the rapidity and range, and also the direction, of the movements.

We can seldom be sure of the precise date at which the nystagmus begins in albinos and others with congenitally defective vision. In some albinos, however, the oscillation has certainly not been noticed until the child was many weeks or even some months old, and the movements are slower and perhaps less rhythmical at first than they become afterwards. Albinotic infants not infrequently keep their eyelids closed for weeks after birth, and this has sometimes led to the report that albinos were born blind; but when such infants have been seen it has been found that, with the eyelids held open, they evidently perceived the difference between light and shade, and that the pupils responded to light.

There must be several different intra-uterine, or very early infantile, diseases or defects of retina, choroid, or optic nerve that, running in families, cause hereditary nystagmus; but for the present the two that stand out as best known are albinism of various degrees and the affection called, for want of a better name, “total colour-

* I am not yet convinced that astigmatism alone can produce nystagmus, because the frequency of astigmatism in albinos suggests a correlation between deficient pigmentation and the corneal deformity, and in cases of nystagmus apparently due to astigmatism only we are not yet in a position to exclude a defect in the retinal epithelial pigment.
blindness” or “day-blindness.” A large unclassed residue remains, but in all probability had attention been paid in these cases to the colour-sense and to the pigmentation of the eye some of them would have been placed in one or other of the two categories just named.

I have nine or ten pedigrees illustrating nystagmus in families affected by what I look upon as incomplete albinism limited to the eyes, ten of nystagmus with day-blindness or total colour-blindness, and fifteen of unclassed nystagmus.

(a) Albinism.

(Figs. 54 and 55 in text; 56, and 59 to 60 in Appendix VII a.)

What little I have time to say about albinism must be in connection with the incomplete or partial cases that, as I believe, have often hitherto been entered as hereditary nystagmus. Pedigrees of ordinary conspicuous albinism with and without consanguinity, with continuous or discontinuous descent, and with, as well as without, other correlated or coincident disease, are seen in Figs. 56, 57, 58, 59, 60 in Appendix VII.* A typical case of the slight degree of albinism in which the deficiency of pigment falls mainly upon the eye is seen in Fig. 53 (Appendix V) (Mr. Jameson Evans's case). Here a child at 15 months (Gen. IV, 1), had nystagmus, slight pink reflex from the pupils, grey irides, and nearly white hair; whilst its brother, when seen at six weeks old, had steady eyes, black pupils, grey-blue irides and yellow hair, “not so light as the elder one at fifteen months old.” The one marked III, 9, at 8 years, with slightly pink pupils, grey irides, decided lack of pigment in choroid, nystagmus, from 2 to 3 D. of astigmatism and V., corrected, $\frac{6}{24}$, had hair of a light shade of dull-brown which had formerly been lighter. The prevalent hair-colour in the others was dull brown and the irides grey. In this family with nystagmus, had

* From a forthcoming memoir upon albinism by Professor Karl Pearson, Mr. Usher, and the writer.
the evidences of albinism afforded by the eyes of the two children above described (IV, 1 and III, 9) not been forthcoming, the true bearings of the case could hardly have been discovered.

In a case of my own (Albinism Memoir, Fig. 402)*, a similar state of affairs existed, except that there was no nystagmus and V., with the moderate myopia and slight astigmatism corrected, was 6/9. I have myself no doubt that Lloyd Owen's well-known case, Fig. 54 (Albinism Memoir, Fig. 449), was really albinism limited to the eyes.

* This lecture, Fig. 186, Appendix VII.
and incomplete even in them. I put the same interpretation on Fig. 55 (Albinism Memoir, Fig. 410, my own case, Mansfield), and Figs. 187 and 188, Appendix VII.*

The note of all these cases is the blue or grey iris, hair now brown, but with the history that it was very fair or even "white" in early childhood, and a more or less albinotic fundus; almost all have nystagmus and marked amblyopia; when, as in a few of the cases, sight is good and the eyes steady, we must suppose that the retinal epithelium, at least at the yellow-spot region, is sufficiently pigmented, however lacking in pigment the choroid may be.†

* The suggestion that the cases just mentioned, and others like them were of albinotic nature was made, so far as I am aware, for the first time by myself in R.L.O.H., xv, p. 110, 1902. It is evident that the idea of albinism was present to the minds both of Mr. Lloyd Owen in connection with Fig. 54 in 1882 and myself in relation to Fig. 55 in 1887, but it was mentioned by each of us at those dates, only to be dismissed.

† The hypothesis is that the imperfect sight, and with it the nystagmus, is caused by deficiency of pigment in the retinal epithelium; that this want may vary in degree, and may even, perhaps, affect only a part—say the central region—of the fundus; and lastly, that such relative or absolute lack of pigment in the epithelium is not recognisable with any certainty by ophthalmoscopic examination, the different depths of tint at the fundus depending far more upon differences of pigmentation of the choroid than of the hexagonal epithelium. In support of this speculation we may say (1) that in albinism with quite translucent iris, i.e. no pigment in the retinal layer, stroma pigment is occasionally present in sufficient quantity to give the iris an ordinary brown colour; (2) that microscopical examination of the choroid of normal European eyes shows—in the comparatively small number of specimens where attention has been carefully directed to the point—that the quantity of pigment in the retinal epithelium appears to be sensibly the same in eyes with pigmented iris and choroid as in those with iris and choroid almost, or quite, devoid of stroma pigment. Whether this position will be maintained when a larger number have been examined remains to be seen. Also we must be careful, for the present at least, to allow for probable differences in the kind of pigment in the eyes of European and of dark races; the eye-pigment of a Negro may be darker than that of a Scandinavian although the quantity be the same in the two. These are nice, but important, problems for future determination, and I have reason to believe that work is already in progress upon them. Examination of partly albinotic eyes, the so-called "wall eyes" or piebald eyes they might be called, of dogs and horses by Mr. Coats and Mr.
There are all sorts and degrees of albinism between these cases which I have ventured to include and the well-marked general albino whom we all know. It may be hoped that in describing future cases of hereditary nystagmus attention will be bestowed upon the present and past colour of hair, eyebrows, eyelashes and iris, aspect of fundus, colour-vision and refraction.*

In their heredity these partial cases appear to be almost perfectly sex-limited; of forty-three affected persons, forty were males, and the descent was through the mother in every case; no affected male ever had an affected child.† In these characters, the group I am calling incomplete ocular albinism differs from general albinism. It is true that in general albinism the descent is usually discontinuous, but the normal parent who acts as carrier is by no means always the mother; again, although there is a decided excess of males with general albinism over females it is much less than in the small Usher within the last year have shown that—apart from the tapetum in those animals—every possible combination of pigment deficiency in the retinal epithelium and choroid or iris may be present, a result supporting, so far as it goes, the above contention. I believe we do not know anything positive about increase of pigment in the hexagonal epithelium after birth; but even if such increase were proved to occur in cases of incomplete albinism, it does not follow that visual acuity would be improved; the pigment might come too late for the otherwise developed retina to benefit by it; we know as a fact that improvement of visual acuteness in albinos, although by no means unknown, is decidedly rare. A great puzzle is the frequency and high grade of the ametropia and especially of astigmatism in nearly all recognised albinos, and the same problem meets us for these cases of blue-eyed nystagmus, and appears to furnish another link between the two groups.

* I do not suggest that everyone with blue eyes, nystagmus, and amblyopia is albinotic in any degree; but some certainly are so, and many others probably; whilst if the essential feature of an albinotic eye is lack of pigment in the hexagonal retinal epithelium, we are not yet in a position to deny the possibly albinotic nature of any clinical case where no more reasonable explanation of the nystagmus and defective acuity can be found.

† These small numbers are given for what they are worth. But even if a few other pedigrees of nystagmus are included where the evidence for albinism is even less than in the above, the excess of males affected over females affected remains very large—3 or 4 to 1.
series mentioned above, being about fifty-five males to forty-five females.*

In the small series available (ten families) consanguinity has not been recorded in any, but I am not sure that inquiry was always made, and even if it had been we could not attach importance to the absence of consanguinity in so small a number. In general albinism consanguinity of parents is common. This fact together with the very great frequency of discontinuous descent in human albinism point to its being a Mendelian recessive. But apart from the question of correct numerical proportions, the infinite varieties both of degree and distribution of albinism in man, i.e. the frequency of intermediates, appears to militate against the applicability of the theory. This leads to the remark that in speaking of albinism we need a definition, and, without going into controversial matters with which, in the present case, I am not fitted to deal, I may at once say that, whatever may be true for such of the lower animals as have been fully examined, it is quite clear that for man we cannot limit the term to persons whose skin, hair and eye tissues are perfectly devoid of pigment. In the first, or last, place you cannot tell without microscopic examination whether a given skin or hair or eye contains a little pigment in certain places or none at all anywhere; and therefore if we refuse the term "albinism" when any trace of pigment is present we must refuse to diagnose albinism in man at all until someone has examined a human eye thoroughly and found it absolutely free from pigment. So far as I know this has not yet been done—not because such eyes do not exist, but because in themselves they are rare and the opportunity of getting them for anatomical examination enormously more so. Clinically we all know that every degree of defective pigmentation occurs in skin or hair or eyes, or in all together, to which we cannot refuse the term "albinism," qualified when

* In upwards of 1000 albinos of all races and various degrees, the excess of males is found not only in the aggregate, but in each separate group used in the summation.
necessary by such terms as "partial" or "incomplete." The problem for human medical observers is, not whether degrees of human albinism, either general or localised, exist, but how far we may carry our subdivisions—what are the smallest tracts or lowest degrees of deficient pigmentation that may be included in the species. It is, I think, likely, although we cannot as yet either prove or disprove the point, that perfect albinism of any one part does not occur without perfect albinism of the whole body. But as we have already seen for the eye, and as is well known also for the skin and hair, we find short of albinism perfect dissimilar degrees of it in the same individual, irregularities of distribution, and differences in the same tissue or organ at different periods of life.

(b) Day-Blindness with Total Colour-Blindness.

(Figs. 61 to 64.)

This interesting but rare hereditary disease is always accompanied by amblyopia, often of high degree, due to defect at the centre of the field; the fundus may appear normal, or slight changes about the macular region and at the disc may be present, not, however, such as would lead one to expect any serious defect of sight. There is always colour-blindness, and in the severer cases it is, as the title indicates, total, but in some milder cases the want of colour sense is less pronounced. No special kind or degree of ametropia is found. Nystagmus is a usual but not invariable symptom. The condition is always said to date "from birth," and it gets neither worse nor better with age. The patients almost always say they see best in a dull light, and sometimes put it that they are "blind" in bright daylight; this, the ordinary condition in toxic amblyopia, retrobulbar neuritis and central retinal disease, is often much more strongly marked in the condition I am describing. The disease often occurs in several siblings, but has, I believe, not yet been seen in parent and child; it is, however, known to have occurred
in two sibships of cousins and once or twice in an uncle. The total number of affected persons I have been able to find recorded, including the fifty-two collected by Grunert in 1903,* is eighty-four, including single cases without family history. Whether we consider this grand total, or only the instances of family prevalence, we find considerably more males than females with the disease with no corresponding excess of males amongst the healthy. There is, in the small series collected hitherto, a decided prevalence of consanguinity of parents. Mental defects have been relatively frequent either in the subjects of the disease or in collaterals. In these broad general characters the disease reminds us of retinitis pigmentosa—indeed, in one of my cases typical pigmentation of the retina was actually present;† and another case‡ one was tempted to interpret as transitional between the two conditions; but the non-progressive character of the present affection appears to constitute an absolute difference.

No case has been examined post-mortem; Galeczowski, who published one of the earliest of the modern cases (1868), conjectured that the seat of the disease lay in the cones, and Grunert, working on much larger clinical material and by improved methods, also sums up in favour of cone-blindness. It is interesting to note in this connection that Stock believes he has microscopical evidence that the bacillary layer is the first part to undergo visible change in retinitis pigmentosa.§

My first case (shown in Fig. 61), was so striking that as the disease seems still to be but little known, I venture to quote it from the paper in which it appeared almost thirty years ago.|| This patient, an intelligent, fairly educated woman, aet. 25 years, came to St. Thomas's Hospital in 1879, with one of her sisters, who was affected like herself.

* Grunert, A./f./O., 1903.
† Fig. 185, Appendix VII.
‡ Miss A—T. O. S., xxviii, p. 86, Case 9.
|| St. Thomas's Hosp. Repts., 1880.
The chief complaint was that she could not see by day and could not tell colours. She said that in the daytime her sight was so bad that she was afraid to cross the street though she could do it with ease in the evening, and that she could read small print by a light so dull that other people had to put away their books. She was so colour-blind that she always dressed in black and white to avoid making absurd mistakes. Her refraction was very slightly H., and V. \( \frac{2.0}{20.0} \) and J. 6 held very close in daylight; constant slight lateral nystagmus. She saw worse after eserine had contracted the pupils. She sorted Holmgren's wools entirely according to their brightness, yellow looking the brightest. Disc and retinal vessels of healthy appearance, but a slight whitish haze of doubtful meaning about the Y.S. A sister, aet. 20 years, who came with her had exactly the same defects of sight, and the spectrum to her was a band or stripe of one colour, brightest in the middle and darker at each end. I afterwards saw a brother, aet. 22 years, who was affected in the same way. They were members of a childship of 11, of whom 6 were living. The parents were said to have perfect sight and no colour defect, but an uncle was said to be colour-blind.

I afterwards saw a still more interesting family (Pike-Channon), (Fig. 62), in which two sets of cousins were affected, two of the victims being idiotic and quite blind. The colour-blindness of the brothers III. 4 and 5 was carefully examined by Captain, now Sir William Abney,
several years later and recorded in his work on colour-vision.*

This case is given as two separate cases by Grunert, one attributed to me, the other to Abney. Fig. 63 shows another case carefully gone into by Mr. Holmes Spicer and myself several years ago, in which the father probably

had the same disease. The last case I know of (Fig. 64), has been given to me recently by Mr. Holmes Spicer, with very careful and detailed notes taken by Dr. W. C. Souter in 1908. One girl and two boys, age from 8 to 6 years (IV, 4, 5, 6), are affected in a sibship of 8; only one other, a

boy, aged 3 years, has lived, and he appears normal; two died at 3 months, one at birth, and one was a miscarriage; no reason to suspect syphilis; parents second cousins through their mothers, who were first cousins. No other cases known in a fairly extensive pedigree. Family from Dorsetshire.

I have fourteen pedigrees of nystagmus not recorded fully enough to allow of their being assigned with certainty to either of the two classes we have just considered, or to any other recognised disease, although several of them are very extensive. Eleven of them are published, the latest being the one by Dr. W. H. Dudley.* Though these pedigrees are inconclusive as to the nature of the disease and probably in some cases not quite accurate in details, they are readily divisible to sub-groups, showing: (1) Continuous descent, seven pedigrees containing about 50 cases of nystagmus with defective sight, one of the pedigrees alone containing 25 cases; in two of these there was consanguinity, in the remaining five it was not mentioned. (2) Discontinuous descent; four pedigrees with more than 20 cases, only 4 being females; descent proved to be through unaffected mother in several instances; apparently no consanguinity in any. Mr. Ernest Clarke's case belongs to this little group, but is so extraordinary that I have counted it separately; this pedigree shows 22 males affected out of 23, and every one of the 20 females escaping; Mr. Clarke has been unable to see the recorder again in order to verify the particulars. (3) In three pedigrees the descent was

* Dudley, W. H., A. O. 0., 1908.
continuous in some instances, discontinuous in others in the same genealogy; two of these (Audeoud, 1895, and Burton-Fanning, the same year), have evidently been drawn up with much care in all respects except as to the ocular details. A list of these pedigrees is given at the end of Appendix VII.

**Choroid.**

Data as to the family prevalence and heredity of various diseases of the choroid and of the cornea are beginning to accumulate.

(1) We know that central senile choroiditis is apt to occur in several brothers and sisters.*

(2) It is of course sometimes difficult to say whether a case should be classed *clinically* as choroidal or retinal. I have for convenience placed several family cases to which this doubt applies at the end of my recent paper on retinitis pigmentosa, *viz.*, one such group called atrophia gyrata choroidea et retinæ by Fuchs, the other (rather paradoxically) a small series in which the choroid is congenitally absent except over a small area at the macular region.† Putting these two little groups together provisionally, we have 23 affected persons of whom 17 or perhaps 18 were male and 5 female. Five were, judging from the records, single cases; the other eighteen occurred in seven families, most of them in siblings only, but once in father and son and once in great-uncle and nephew. In one family the choroidal disease was associated with dullness of intellect, undergrowth of body, and arrest of sexual development. It is unnecessary to dwell longer on this group. The cases are evidently very rare, and in future examples the family history should be inquired into much more fully.


† The best known cases of these allied conditions are given in abstract in *R.L.O.H.*, xvi, pp. 369-377, 1908.
(3) In another group of cases a multitude of small, round, whitish dots or spots of what appears to be superficial disease of the choroid occupy the central area of the fundus, and in some examples pigmentary changes are described more peripherally. Mr. Doyne recorded the first definitely hereditary case of this variety in 1899,* using the term "honey-comb choroiditis." His patient was one of several siblings similarly affected, and the disease had occurred in the ascendants for three generations; I believe that Mr. Doyne has not hitherto published the genealogy of this remarkable family in full. A case probably of the same sort in a brother and sister had been published by Mr. Lang† in 1885, and a single case perhaps of the same type, without record of others in the patient's family, by Mr. Juler in 1893,‡ whilst in 1897 Mr. R. D. Batten and Mr. Holthouse§ recorded another single case, agreeing with the description of honey-comb choroiditis in a woman, aged 25 years, the last born of 24 children, 20 of whom had died young from some obscure cerebral disease. A case, probably similar, with coloured illustration, was published by Mr. Reginald E. Bickerton in 1900.|| In 1901 Mr. Hugh Thompson* put on record the case of a woman, aged 57 years, with extensive superficial choroidal changes around the discs which had caused no symptoms; but her father and three brothers who were his grandsons, i.e. were nephews of the patient, were night-blind, and one of them who was seen had the appearances of atypical retinitis pigmentosa.

The case given by Liebrecht** as retinitis punctata albeescens in 1895 does not agree in all respects with that

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† Lang, T.O.S., v, 1885, pp. 140 and 141.
‡ Juler, T.O.S., xiii, 1893, p. 143.
§ E. H. Holthouse and R. D. Batten, T.O.S., xvii, 1897, p. 62, and xx, p. 95, with Plate III, fig. 2.
* Reginald E. Bickerton, T.O.S., xx, p. 93, with Plate III, fig. 1.
* Hugh Thompson, T.O.S., xxi, 1901, p. 66.
condition, and may, I think, have been of the same character as Deyne's "honey-comb" cases.

(4) In 1872, Mr. Cowell* published in much detail a peculiar case of destructive irido-choroiditis in a father and two of his children, and detachment of retina in a third child, but as the author thought that the whole affair was very probably syphilitic the case cannot be quoted with confidence as an example of heredity.

(5) I find notes of two sisters, aet. 20 and 21 years, seen at St. Thomas's Hospital in 1883, with identical extensive superficial choroidal atrophy which appeared to have come on at the age of 8 in one and 12 in the other, and was apparently not progressing; there was no pigmentation of retina and no evidence of hereditary syphilis. Both were myopic and had V. $\frac{20}{200}$ with correction. The cases may have been of the same kind as that of Miss A—, referred to in the Section on Day-blindness.

(6) I am indebted to Mr. Fisher for the notes of a case in which two sisters and a brother, aet. from 39 to 33 years in 1908, have changes similar to the last case (5) and dating, as in that case, from late childhood; the refraction hypermetropic. There was not the slightest facial or dental evidence of syphilis. They were the third, fourth and fifth born in a sibship of nine. The mother, who died at 53, had had poor sight for many years but was not blind.

(7) Two cases of family choroiditis have also been recorded by Hutchinson,† but in at least one of them there was a strong suspicion of syphilis in the father

**Cornea.**

*(Figs. 65 to 69 in Appendix VIII.)*

(1) "Nodular" and "Reticular" Opacity of the Cornea.

Of these conditions, classing them together as probably

† Hutchinson, Archives of Surgery, xi, 1900, p. 122; and R.I.O.H., v, 1866, p. 324.
of essentially similar nature, we have now at least eight pedigrees showing the disease in from two to four generations, quite half a dozen others of "familial" prevalence, and perhaps a dozen other single cases.

In the pedigree and familial cases the sexes are about equal, descent continuous with one exception, and from either sex to the same or to the opposite sex.

We shall no doubt have larger numbers to deal with before long.

Descriptions of the most extensive pedigrees (Holmes Spicer, Freund,* Doyne and Stephenson, and Folker) are given with Figs. 65 to 69 in Appendix VIII.

(2) Several other affections of the cornea are known to occur as family diseases from time to time.

In February, 1905, Mr. Jessop wrote to me that he had then lately seen conical cornea in a lady of about 50, who stated that her mother had gone blind from conical cornea. In June, 1906, I heard from Mr. Laws that he had just seen the case of a young woman with conical cornea, whose mother stated that the daughter's eyes had been like they now were from birth, and that three more of her children were affected in the same way; she had had eleven children, most of whom died in childhood; one was in an asylum; the parents were first cousins.

Buphthalmos has been seen in several brothers and sisters, and it is not unlikely that the case published by Crompton in 1840† as congenital opacity of the cornea in two siblings out of ten and the earlier one by Farar‡ in 1790, in three siblings, were of that nature.

* Freund's Case 2 (Bienert) has been brought up to date by the author in courteous reply to inquiry (June, 1909), and is now correctly shown by Fig. 67.
† S. Crompton, London Medical Gazette, xxvii, 1840, p. 432.
APPENDICES.

The following appendices will enable the reader to verify the more important statements made in the Lecture, especially those as to the numbers of diseased to normal, the relative liability of the two sexes to be affected by each of the diseases in question, and the occurrence of anticipation. In some cases the data themselves are given, in others specific references are made to papers I have lately published which contain the necessary information.

The subject of Leber’s disease is so important that I have thought it well to make a short abstract of every case published and unpublished that I could lay hands upon, and to insert figures of the pedigrees of a large number; this collection is based primarily upon Hormuth’s Dissertation,* published in 1900, towards which the considerable series published by Habershon in our Transactions (vol. viii, 1888) furnished an important contingent. A number of other cases, published and unpublished, have been added to Hormuth’s series.

The illustrative cases and figures are numbered serially from 1 to 188. Of these, 47 are inserted in the text of the Lecture, the remainder appear in the appendices in connection with their respective diseases. Although this plan will cause some inconvenience to the reader, it is preferable to the alternative of having two separate series of numbers, one for the Lecture, the other for the Appendices.

The following are the Appendices:

1. Illustrating the introductory section of the lecture.

Frequency tables for:

(a) Cataract, post-natal.
(b) " congenital, lamellar and discoid.
(c) " other forms.
(d) Retinitis pigmentosa, continuous descent.
(e) " discontinuous descent.
(f) Diseases allied to retinitis pigmentosa.
(g) Night-blindness, continuous descent.
(h) " discontinuous descent.
(i) Leber’s disease.
(j) Proportion of females carrying disease in certain sex-limited afflictions.

II. Relative numbers of males and females affected by lamellar cataract and other forms of congenital cataract.

III. Glaucoma, Case-figs. 28-34.

IV. Retinitis pigmentosa, Case-figs. 37, 38, 39.

V. Night-blindness without changes.

(a) References.
(b) Mr. W. J. Cants’ new case of congenital night-blindness, Case and Fig. 44; also Case 44a.


Vol. XXIX.
VI. Leber’s disease.

(a) Abstracts of all the cases with figures, except Case-figs. 45-52 placed in the text of the lecture.

(b) References to cases or figures in (a), illustrating various features of the disease discussed in the lecture.

VII. Nystagmus.

(a) Albinism section: (1) Case-figs. 53, 56, 57, 58, 59, 60.

(2) Various references to published cases and the following new cases, Figs. 182, 183, 184, and 188.

(b) Day-blindness section: Various references to cases and Case-fig. 185.

(c) Unclassed nystagmus; references to places of publication.


IX. Abbreviations for titles of periodical publications.

APPENDIX I.

Frequency Tables.

Data upon which the Statements at p. 150 et seq. of the Lecture as to the Relative Numbers of Normal and Diseased are based.

Only those sibships (childships) have been used that were either known or judged on good grounds to be complete as to numbers and sex record; only those in which (with one single exception) the youngest member was old enough to be susceptible to the disease in question; and only those in which either one or more of the siblings or one of the parents of the sibship was affected. Therefore, from the childships selected all members are excluded who died before the usually vulnerable age and all still-births and miscarriages. I am well aware that the omission of these items might lead to inferences that in the present state of our knowledge are unwarranted; for whether the numbers here given from human data do or do not agree with Mendelian requirements we are certainly not at present entitled either to affirm or deny that the proportions of normal and diseased would have been the same if all the conceptions had lived to the susceptible age. My object has been only to ascertain, on a somewhat larger scale than has been attempted before, how far the available numbers, as they stand, do or do not fit with Mendelian expectation as based upon experimental breeding.

In such diseases as post-natal cataract, very small lamellar or discoid cataract, and even retinitis pigmentosa, the proportion of affected to normal is almost certain to be more or less too low, for in these diseases, and especially post-natal in cataract, the earliest stages of the malady may pass undiscovered unless the eyes of every member be examined—a condition that can seldom be fulfilled.

When descent is continuous every completed sibship in which the disease occurs is counted, and every sibship of which either parent is affected, whether any of the children are so or not. When descent is discontinuous only the sibships showing the disease can be used.
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### Individual Case Data

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#### Additional Notes
- **Cataract:** Post-nasal or Acquired Cataract, all Ayes, Refer.
(b) Lamellar and Discoid Cataract.

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(c) Congenital Cataract other than Lamellar: Coralliform, Stellate, and Undescribed Forms.

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*P.S.—If we add two pedigrees showed at this Society by Mr. Bishop Harman at the July meeting (his Cases 1 and 2) the numbers are 306 (54 per cent.) normal, 206 (44 per cent.) cataract, total 566 (100).

(d) and (e) Retinitis Pigmentosa.

In compiling the following tables of retinitis pigmentosa I have omitted 9 childships—containing an aggregate of 70 children, the smallest having 6—each of which contains only one case of the disease, viz., R.L.O.H., xvii, Cases 43, 81, 83a, 83b, 83c, 83d, 83e, 83f, 83i.

In the following 5 childships used in the tables either deafness or idiocy has been taken as equivalent to retinitis pigmentosa, viz., R.L.O.H., xvii, Cases 32, 2 r.p. + 1 idiot = 3; 33, 3 r.p. + 1 idiot = 4; 84, 3 r.p. + 1 deaf = 4; 119, 1 r.p. + 1 idiot = 2; this lecture Case—fig. 39, r.p. + 1 deaf = 2.
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<th>Persons counted affected individual</th>
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<td>T.O.S., xxvii, p. 217 (Snell).</td>
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</table>
(E) Retinitis Pigmentosa—Discontinuous Descent, i.e. Parents Normal; Both Sexes Affected.

---

The above 22 cases contain 31 sibships available for the foregoing tables. Thirty of these sibships may be classed into three groups, showing respectively (c) almost every individual affected, (a) nearly one half, (b) nearly one quarter. Only one, Fig. 36, IIIa, with 4 affected out of 11, is widely inconsistent.
(c) Nearly all affected.

*R.L.O.H., xvii, p. 17, ct seq.*

<table>
<thead>
<tr>
<th>Case</th>
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</tr>
<tr>
<td>15b. III</td>
<td>8</td>
<td>7</td>
</tr>
<tr>
<td>36. Vc</td>
<td>4</td>
<td>3</td>
</tr>
<tr>
<td><strong>Totals</strong></td>
<td><strong>13</strong></td>
<td><strong>11</strong></td>
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</tbody>
</table>

(a) Nearly one half affected.


<table>
<thead>
<tr>
<th>Case</th>
<th>Total</th>
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<tbody>
<tr>
<td>15. IVa</td>
<td>7</td>
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<tr>
<td>15a</td>
<td>4</td>
<td>2</td>
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<tr>
<td>16</td>
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<td>3</td>
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<tr>
<td>18</td>
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</tr>
<tr>
<td>20 IIIa + b, a = 8 and 3; b = 13 and 6</td>
<td>21</td>
<td>9</td>
</tr>
<tr>
<td>25</td>
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<td>3</td>
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<tr>
<td>26</td>
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<td>4</td>
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<tr>
<td>27</td>
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<tr>
<td><strong>Totals</strong></td>
<td><strong>38</strong></td>
<td><strong>28</strong></td>
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<table>
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<td>33</td>
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<tr>
<td>This Lecture, 37, IIIa</td>
<td>8</td>
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<tr>
<td>Liebreich, <em>Arch. Gen. de Med.</em>, 1861, Case 2, IIIa</td>
<td>6</td>
<td>3</td>
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<td>Family D. II</td>
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<td>Webster, <em>T.A.O.S.</em>, ii, 504, Cases 20 to 33, II</td>
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<td>Coleman, <em>Amer. Pract.</em>, 1889, p. 49, II</td>
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<tr>
<td><em>R.L.O.H., xvii, p. 366, Case 118, IIIb.</em></td>
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<tr>
<td><strong>Totals</strong></td>
<td><strong>59</strong></td>
<td><strong>27</strong></td>
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</table>

(b) Nearly one quarter affected.

*R.L.O.H., ibid., Fig. 38* 40 4 1

This Lecture, Fig. 37, IIIb 4 1

36. IVa, Va, b and d.

all same proportions 23 6

This Lecture, Fig. 39, V. 7 2

**Total** 42 11

*R.L.O.H., ibid., p. 365, Case 118, IIIa* 8 2

vol. ix, p. 172, 2, IVa 8 23 3

**Totals** 16 4 or 5

Total 58 15 or 16
### Diseases Allied to Retinitis Pigmentosa.

#### Atrophy gyrata choroidae et Retinæ (Fuchs).

<table>
<thead>
<tr>
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<th>Persons counted and affected until normal +O</th>
<th>Number affected</th>
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<tbody>
<tr>
<td>Ibid. &quot; 127</td>
<td>(2 ob. inf'cy)</td>
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<tr>
<td>&quot; 129</td>
<td>II 10–1 = 9</td>
<td>(1 ob. inf'cy)</td>
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#### Congenital Absence of Choroid.

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<tbody>
<tr>
<td>Ibid. &quot; 133</td>
<td></td>
<td>8</td>
<td>3</td>
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<tr>
<td>&quot; 134</td>
<td>11–3 = 8</td>
<td>(3 ob. inf'cy)</td>
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</table>

### Retinitis punctata albescens.

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<tbody>
<tr>
<td>Ibid. &quot; 142</td>
<td></td>
<td>(4 died inf'cy)</td>
<td>3</td>
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<tr>
<td>&quot; 143</td>
<td>Ile</td>
<td>7–1 = 6</td>
<td>2</td>
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<tr>
<td>&quot; 144</td>
<td>IV</td>
<td>(1 ob. inf'cy)</td>
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<tr>
<td>&quot; 145</td>
<td>VI</td>
<td>8–6 = 2</td>
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<tr>
<td>&quot; 146</td>
<td>(6 ob. inf'cy)</td>
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#### Liebrecht’s case: diagnosis doubtful.

### Summary.

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### (a) Congenital Night-blindness without Changes—Continuous Descent; Both Sexes Affected. Cuvier Group.

#### Reference.

<table>
<thead>
<tr>
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<tr>
<td>c</td>
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### (b) Reference.

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<td>&quot; 156</td>
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### Summary.

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(n) Congenital Night-blindness without Changes—Discontinuous Descent.

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<td>Ila</td>
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<tr>
<td>&quot;</td>
<td>161</td>
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Notes: (a) young, (b) living, (c) sex, (d) infancy, (e) ob.
Leber’s Disease (only Males Affected)—continued.

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### Leber's Disease—Males and Females Affected.

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**Summary of both Tables.**

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(4) Proportion of Females Carrying Disease in Certain Sex-limited Affections.

In a disease transmitted only by unaffected females the number of sisters in any childhood who carry it can, with our present knowledge, be known only if they all have children. The following are the only examples I have been able to find, and even in them the evidence must be regarded as incomplete for such of the sisters as had very few children:

(1) Discontinuous retinitis pigmentosa: Fig. 36, IVb,* contains 5 sisters, 1 of whom dies single; of the others 2 have 12 children in all, some of whom have the disease; the other 2 have 4 children in all (only 3 of these are shown in the Figure), none of whom have the disease.

(2) Discontinuous night-blindness: Fig. 42, IVa, contains 4 sisters; 2 of them have 9 children in all, some affected; the other 2 have 4 in all, all normal.

(3) Discontinuous night-blindness (R.L.O.H., xvii, p. 419; Fig. 175), II, contains 4 sisters; 2 of them have in all 5 children, some affected; the other 2 have in all 3 children, all normal.

(4) Discontinuous night-blindness (ibid., xvii, p. 422; Fig. 178, IIIb), contains 2 sisters; 1 has 4 children, some affected; the other 2 children both normal.

(5) Leber's disease (this Appendix: Fig. 108, IIIa), contains 2 sisters, both of whom had affected issue. Note that of their 7 brothers 6 had the disease.

(6) Leber's disease (ibid.: Fig. 94), the two small childships IIa and b, contain 3 females, 2 of whom married, and both had some affected children.

(7) Leber's disease (ibid.): Fig. 143, II, contains 2 sisters, of whom one certainly bore affected issue.

(8) Leber's disease (ibid.): Fig. 166, IIIa, contains 2 sisters, of whom one bore three children, one of them affected; the other had an only child who was affected.

In these eight instances we have 24 sisters, of whom 21 certainly had children (1 died childless and 2 others appear to have been unmarried at date of record). Of these 21, 13 had amongst them rather more than 51 children (exact number in one case not given), containing 18 affected. The remaining 8 had amongst them only 14 children, all normal.

* IVb.—The letter b signifies the second eligible childhood in Gen. IV counting from the left; the first is a. These letters are not marked on the Figure. The same explanation applies to the other relevant Figures.
**APPENDIX II.**

*Sex in Lamellar Cataract, whether Hereditary or Sporadic (including Discoid Cataract).*

The following returns have been kindly supplied to me by various colleagues and friends.

<table>
<thead>
<tr>
<th>Source</th>
<th>Males</th>
<th>Females</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Moorfields Hospital, over 10 years</td>
<td>405</td>
<td>195</td>
<td>600</td>
</tr>
<tr>
<td>about ten to fifteen years</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>St. Thomas's Hospital, since 1878</td>
<td>60</td>
<td>30</td>
<td>90</td>
</tr>
<tr>
<td>St. Bartholomew's Hospital, twenty-four years</td>
<td>113</td>
<td>65</td>
<td>178</td>
</tr>
<tr>
<td>St. George's Hospital, about twenty years</td>
<td>29</td>
<td>18</td>
<td>47</td>
</tr>
<tr>
<td>London Hospital, the last few years</td>
<td>18</td>
<td>10</td>
<td>28</td>
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<tr>
<td>Dublin (Sir H. R. Swanzy), ten years</td>
<td>45</td>
<td>18</td>
<td>63</td>
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<tr>
<td>Aberdeen (Mr. C. H. Usher)</td>
<td>32</td>
<td>33</td>
<td>65</td>
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<tr>
<td>Manchester (Mr. Hill Griffith)</td>
<td>58</td>
<td>41</td>
<td>99</td>
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<table>
<thead>
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<th>Total</th>
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<tr>
<td>Oxford (Mr. Doyne)</td>
<td>36</td>
<td>29</td>
<td>65</td>
</tr>
<tr>
<td>Birmingham, Queen's Hospital (Mr. Priestley Smith)</td>
<td>57</td>
<td>38</td>
<td>95</td>
</tr>
<tr>
<td>Birmingham Eye Hospital, forty and a half years</td>
<td>18</td>
<td>9</td>
<td>27</td>
</tr>
<tr>
<td>Cases from forty, published and unpublished</td>
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<td>130</td>
<td>270</td>
</tr>
<tr>
<td>Cases seen in private practice, seven separate returns</td>
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<td>57</td>
<td>115</td>
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<td>Moorfields Hospital (partial return only)</td>
<td>4</td>
<td>2</td>
<td>6</td>
</tr>
<tr>
<td>St. Bartholomew's Hospital, twenty-four years</td>
<td>6</td>
<td>5</td>
<td>11</td>
</tr>
<tr>
<td>Dublin (Sir H. R. Swanzy), ten years</td>
<td>12</td>
<td>7</td>
<td>19</td>
</tr>
<tr>
<td>Aberdeen (Mr. C. H. Usher)</td>
<td>28</td>
<td>5</td>
<td>33</td>
</tr>
<tr>
<td>Manchester (Mr. Hill Griffith)</td>
<td>39</td>
<td>23</td>
<td>62</td>
</tr>
<tr>
<td>Totals</td>
<td>1166</td>
<td>721</td>
<td>1887</td>
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</table>

*Sex in Congenital Cataracts other than Lamellar.*

<table>
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<th>Source</th>
<th>Males</th>
<th>Females</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Moorfields Hospital (partial return only)</td>
<td>58</td>
<td>38</td>
<td>96</td>
</tr>
<tr>
<td>St. Bartholomew's Hospital, twenty-four years</td>
<td>22</td>
<td>18</td>
<td>40</td>
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<tr>
<td>Dublin (Sir H. R. Swanzy), ten years</td>
<td>18</td>
<td>8</td>
<td>26</td>
</tr>
<tr>
<td>Birmingham, Queen's Hospital (Mr. Priestley Smith)</td>
<td>25</td>
<td>20</td>
<td>45</td>
</tr>
<tr>
<td>Birmingham Eye Hospital (Mr. Edles), five years</td>
<td>45</td>
<td>55</td>
<td>100</td>
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<tr>
<td>Cases from various published pedigrees</td>
<td>82</td>
<td>82</td>
<td>164</td>
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</tbody>
</table>
APPENDIX III.

GLAUCOMA.

The paper by Lawford in R.L.O.H., xvii, p. 57 (1907), "Examples of Hereditary Primary Glaucoma," contains particulars of twenty-four families in which the disease prevailed, and a list of nineteen references to the literature. Six of the cases are new, eighteen had been published before.

Anticipation was well marked in at least half of the series, viz., Cases 1, 3, 6, 7, 8, 10, 11, 13, 14, 16, 17, 24, taking them serially as they come in Lawford's paper. The following seven of these were shown in pedigree form at the lecture.

Fig. 28 (Lawford, Case 7, from Lucien Howe): I, 4 affected at about 40, and became blind; II, 2 affected at 28; III, 1 at 28; III, 3 probably about the same age; III, 5 at 17; III, 6 at 26; III, 8 at 19.

Fig. 29 (Lawford, Case 1): I, 1 blind from "amaurosis" (? glaucoma simplex) for some years before death at 85; II, 6, double, quiet glaucoma at 66, operated upon, died at 71; his wife (II, 14) died at 63; 3 of his siblings died in infancy; 2 others (II, 5 and 9) died as adults, all the others living and reported to see well; III, 1 to 7 issue of II, 6 and 14, aged, at record, from 53 to 36, and all except III, 1 examined by author; III, 3 and 5 got glaucoma simplex at 48 and 40 respectively, and III, 2 had, at 52, signs of the incipient disease. In IV all are reported to see well, the eldest of IV, 1 being 27, and of IV, 3, 29.

Fig. 30 (Lawford, Case 8: Nettleship, The Ophthalmoscope, September and October, 1906). I, 1 affected at about 71; II, 1 at 45; III, 1 at 23.

Fig. 31 (Lawford, Case 3): I, 1 blind at 60, almost certainly from glaucoma, died at 68; II, 1 attacked at 58; II, two years younger, attacked at 47; II, 3, 6 other living siblings who see well; II, 4, 4 who died.

Fig. 32 (Lawford, Case 14): I, 2 glaucoma at 47; of his 2 children by first wife, II, 2 had glaucoma at 26, and of the two by third wife, II, 4
glaucoma at 13; second wife (1, 3) childless. This case also shows coincidence of high myopia and glaucoma in II, 4.

Fig. 33 (Lawford, Case 15, Mules, O.R., ii, p. 48, 1883): 1, 1 glaucoma simplex at 49; II, 1 the same at 18; II, 2 at 16; II, 3 at 15 had increased tension, but no other signs.

Fig. 34 (Lawford, Case 24; Jacobson, A.J.O., 1886, iii, p. 96): 1, 1 glaucoma simplex at 70; II, 1 at 45, and II, 2 at 40.

APPENDIX IV.

Retinitis Pigmentosa.

The principal pedigrees upon which I base what is said at p. xcvi et seq. of the text as to continuous and discontinuous descent in different families may be found in R.L.O.H., xvii, pp. 7-17 and 360 for continuous descent, and at pp. 18-24 and 26-31 for discontinuous descent. I have gone over these again carefully, and find no errors except that in Fig. 33 Gen. I should be omitted, there being no information.

Discontinuous descent of retinitis pigmentosa side by side with continuous descent of lamellar cataract is shown in a pedigree published in T.O.S., xxviii, p. 226 (1908). In the text of the lecture the two parts of this genealogy were treated separately, the part containing the cataract cases being shown in Fig. 12, and that containing retinitis pigmentosa in Fig. 36.

The details of the cases shown by Figs. 37, 38, and 39 are as follows:

Fig. 37 (p. xcvi), sent by Mr. C. H. Usher (Rowand family), 1909. I, 3 was invalided from the Army as a young man for "moon-blindness," and was told it would get worse; could see well in the day, but not in the evening; got steadily worse, was quite blind at 50, and died at 70. I, 1, 2, and 4 all good sight. II, 1 and 5 both good sight. III, 8, act. 36 years, typical advanced retinitis pigmentosa; has one child with good sight (IV, 4). III, 7, act. 38 years, conditions much like III, 8; hearing very quick; has four sons, all living, and one daughter, who died lately, all with good vision (IV, 2 and 3). III, 9, act. 34 years, conditions as in the other two; married fourteen years, no issue. III, 12, act. 50 years, nearly blind of retinitis pigmentosa, a drinker, and has been under care for delirium tremens; twice married; no issue by first wife, but by
second has five or six children, in one or two of whom sight is bad both
day and night. III, 4 died in infancy. III, 5 at 14. III, 6 is 40; has
good sight and three normal children. III, 10 and 11, and the child,
IV, 5, all see well.

Fig. 38 (p. xcvii), from Mr. Lawford and Mr. E. Collier Green (Paynter
family). A single case in a large childship; possible influence of severe
loss of blood.

III, 3. Mr. Lawford's patient at Moorfields Hospital in the spring of
1909 for typical retinitis pigmentosa. He is at 38 years. From his
account, confirmed by personal investigation of his family history and
examination of his mother and several siblings by Mr. E. C. Green, of
Derby, it may be considered certain that no other cases of bad sight or of
degeneracies are known in his generation or the next. He considers
his sight to have been failing ten or twelve years, but can give no
precise date, and did not himself connect it with the haemorrhages
of which he gives a history. When 26, a railway porter, he bled
violently from the nose one day from 9 a.m. till noon, and was
plugged at St. Bartholomew's Hospital. When 33 (five years ago) was
operated at the Great Northern Hospital for "appendicitis," and says
that about two weeks after the operation he vomited a quantity of blood.
Again a year ago he was in bed for six weeks, and passed blood both
from the bowel and bladder. The history of the epistaxis is clear enough,
but his statements as to the internal haemorrhages are, of course, of less
value. Has not had typhoid or other infectious illnesses to his know-
ledge, and denies venereal disease of any kind, and shows no signs of
congenital syphilis. Married ten years; two children, of which IV, 2
died at 13 months and would now be 8; IV, 2 living, at 27 years.
Mother (II, 2), at 60 years, examined by Mr. Green, and found normal;
by first husband (II, 3), who died at 52 from an accident, sixteen concep-
tions (III, 1 to 16), of whom III, 1, at 40 years, and III, 9 and 10 (the
latter the youngest living, at 23 years) have been examined by Mr.
Green and found normal. III, 2, 6 and 11 to 15 miscarriages (seven in
all), and III, 16 died of measles at 9 months. IV, 1, 8 children (3 boys,
5 girls) of III, 1, at from 17 to 3 years; 7 are living, and were examined
by Mr. Green and found normal; the boys are the first, eighth, and fifth;
the latter died of "brain fever" after an accident three years ago;
the girls are Nos. 2, 3, 4, 6, and 7. The other sixteen (IV, 4 and 5)
and their parents are scattered, and could not be seen, but all are
confidently reported to see well. II, 2 had no issue by her second
husband. Her brother (II, 1) married, but had no issue. I, 1 living;
I, 2 dead; sight good in both. No consanguinity.

Fig. 39 (p. xcvii), from Mr. Herbert Fisher. The family records have
been accurately kept for many generations. The figure shows only
the parts of the family tree that bear upon the occurrence of retinitis
pigmentosa and deafness.

V, 5 at 45 years, well-marked typical retinitis pigmentosa, and is
moderately deaf. V, 8 deaf, but good sight; V, 1 died of phthisis as
a young man; V, 3, 6, 7 and 9 normal. No other cases known of bad
sight like V, 5, but a vague history of defective sight in IV, 12 and III, 10. Clear history of deafness from early life in II, 6, IV, 7 and 11, V, 8 and 10; the deafness has varied in severity in the different persons, very bad in II, 6 and IV, 11, moderate in the others. VI, 2 has had attacks of mania; her brother, a medical man, says she resembles her aunt, V, 5, closely in some respects. VI, 6 died in infancy "from some defect in the larynx," and VI, 7, the two children of V, 8, also died in infancy. IV, 7 and his wife, IV, 3 were second cousins, but the consanguinity was from another stock in which there are no known cases of blindness or deafness.

APPENDIX V.

Night-Blindness without Changes.

(a) The cases used, being all I have been able to find either in the literature or amongst my own notes, are given in R.L.O.II, xvii, pp. 401 to 426, and there numbered Cases 151 to 190 (1908).

(b) Mr. W. J. Cant's case, Case and Fig. 44.—Night-blindness without visible changes affecting myopic males.

In this small pedigree only three or four cases are known. Two of them have been seen and examined with care, and I therefore record the case fully.

I, 1 was a woman who lived to be 90; she became blind in her old age, but there is nothing to show that she had night-blindness.

She had at least one daughter, II, 1, but whether there were other children is not known. II, 1 had three children, no more.
The first-born, III, 2, is reported by his daughter to have been "very short-sighted and night-blind, and had always to be led about after dusk all his life"; as he lived to be 76, and is never known to have worn reading spectacles, he was probably myopic. He had a sister and a brother (III, 4 and 6) who had perfect vision until they died; both left children and grandchildren. Of the latter, two had some affection of sight, but no one knows whether they were night-blind or not (see below); all their other descendants in IV and V are normal.

He (III, 2) married twice, there being no consanguinity between him and either wife or between the wives. By the first he had an only child 7 (IV, 2), with good sight, who had an only child (V, 1, illegitimate), now aged thirty-five and night-blind; his only child (VI, 1) is normal. By his second wife, III, 2 had 5 children (IV, 3 to 7) all with good sight, three ♂, two ♀. One of the daughters (IV, 4), who died in 1908, left a son who is night-blind (V, 4) and a daughter (V, 3) with normal eyes. Of the numerous other grandchildren of III, 2, none are night-blind, but it is noteworthy that the other female who might transmit (IV, 5), has had only one child.

Description of the Cases.

III, 2 a compositor, who died at 76, is reputed to have been always unable to see at night but to have had no defect in the day; his daughter, IV, 4, remembers (speaking in 1907) having often in former years had to lead her father home by the arm at night; he never wore any spectacles, and was therefore probably myopic in some degree.

V, 1, at 35 years, a clerk at Doncaster, was examined by Mr. C. H. Usher and myself at the house of Mr. Usher's brother in Lincolnshire in 1908. Has been short-sighted and unable to see at night as long as he can remember; as a small boy when first at school he could not see the blackboard; the night-blindness has got no worse. Has never had glasses, and the progress or otherwise of the myopia therefore cannot be ascertained; refraction now, estimated about 7 D. and 10 D. in K. and L. at posterior pole, decidedly less at periphery; fundus of medium complexity and normal in every particular except for moderate myopic crescents. Black hair, colour of irides not noted. Married eight years, one child only. The tests as to light sense defect were necessarily inexact but were made with much care, and in all cases were compared with our own sight under the same conditions of light, but his myopia was not corrected.

As to reading:—with a considerably lowered light, he was unable to read print with his myopia uncorrected, which I (E.N.) could read perfectly with + 4 or + 5 D.; but in good lamp-light he read J. 1 easily. When shown a screen and a bed-cover, each with large patterns of different but somewhat sombre colours, he required much more light than we did to recognise the pattern, and this at his own normal far point. No defect of F's could be discovered, but we noticed that when seeking to see an object beyond his far point, viz., a picture, or the pattern on the
screen, he always looked considerably above the object, i.e., he used the retina a little above the Y.S. He was then tested in the garden in brilliant moonlight, and the following notes made: he could not see a white handkerchief on the grass only a foot from him, although it was visible to all of us at many yards off; he could not see the white cuff of his own shirt when his coat sleeve was drawn up; he was quite unable to see a number of flowers that were easily visible to the rest of us; finally he was quite unable to find his way about the lawn (a flat one) without a guide, and on turning to the house said that the lighted window was the only thing by which he could at all guide himself; and it was obvious that if there had been any pit or obstacle on the ground he would have walked into it unless guided. The whole result was very striking, and certainly not to be explained merely by the uncorrected myopia.

Married eight years, one child, act. 8 years (VI, 1), seen and found to have H. 2 D, with normal fundus, perfect sight, and no night-blindness.

V. began glasses at four years old, and was first seen by Mr. W. J. Cant in 1889, i.e., when from five to six years of age, and found to be using -9 D. spectacles; Mr. Cant gave him -6 D. In April, 1904 (act. about 10 years) the R. was found to be divergent and somewhat amblyopic, V. with correction being only $\frac{9}{2}$ against $\frac{9}{12}$ with the L., and he had difficulty in maintaining fixation with R.; it was probably then that his present full correction was ordered. There is no further record until the early part of 1908 (act. 14 years), when Mr. E. C. Clements and Mr. Cant made a careful examination, with the following results: R. -10 D. $\frac{9}{12}$; L. -9 D. sph. with -1.5 D. cyl. $\frac{9}{2}$ partly; is already wearing glasses of this strength. When the illumination is reduced to half light, V. with correction equals only $\frac{9}{20}$, and with one quarter illumination only $\frac{9}{60}$, and with the window-blind drawn still further down he was unable to see a piece of white paper 10 in. (25 cm.) square at 6 ft., even when it was moved about. Fields for white; L. normal in full light, much reduced in the same half and quarter light; when the blind was drawn more than three quarters down the fixation object was invisible to him even when $\frac{1}{2}$ in. square; R. (amblyopic eye) smaller for full light than L., and shows similar further contraction in lowered light. Fundus perfectly healthy in appearance in every detail, except for ordinary sharply defined crescents from one quarter to one third the width of O.D.; especially, retinal vessels of full size and no trace of retinal pigmentation.

His mother noticed that his sight was not right before he was a year old, and that when between two and three he could not see his toys if the light was at all bad, but had to grope for them.

When Mr. C. H. Usher and I saw him (October 3rd, 1908) we found him a tall, narrow-chested boy, 5 ft. 6 in. high, of between 11 and 15 years, with, as already noted, perfectly normal fundi; by estimation the myopia was much less at the periphery than at posterior pole of globe; choroids rather fair. Various comparative qualitative tests applied whilst wearing his correction, such as a square of white paper several inches in the side viewed under different degrees of illumination, showed repeatedly.
that his light-minimum was much higher than ours, and that he required longer than we did in order to see the object even when the light was enough (viz., slow adaptation); and with light below his minimum a slight increase at once made the object visible to him (this also shows slow adaptation). Fields to rough hand tests did not show contraction. He always takes his sister's arm when out with her in the dusk. At home he often knocks against a certain door-prop which his sister says no one else would do.

V, 3, only sister of the last, act. about 20 years, a school teacher, not myopic, fundus perfectly normal.

V, 9, said to have been "very short-sighted, held his book very close," and his aunt, IV, 4, who knew him, wrote that "she thought he was afflicted in much the same way as her son," V, 4; he did not use spectacles; died at 25. He had about six siblings, who all saw quite well.

V, 11 said never to have had good sight, and eventually went quite blind, but no particulars are known. She is dead. She was one of the seven or eight children. She did not wear glasses.

V, 13 known to have good sight.

IV, 4, who died during 1908, had perfect sight, as has her husband, IV, 14.

III, 4 and 6 had perfect vision.

No consanguinity between IV, 4 and 14.

Case 44e (no figure).—By a curious coincidence another family with the same complaint lives in the next village to V, 4 of the case just narrated. The two families are not related in any way on either side; the former came from a distance in recent years, the latter has been settled as farmers at or near Navenby for a long time.

This second family could not be fully searched out; the information obtained is given for what it is worth.

III, 1 and 2 were first cousins and I, 1 was the grandfather of one of them and he became blind, probably from cataract, in old age, and died recently (1907 or 1908) at 80. II, 1 is living and sees well; her husband, who also had good sight, died in middle age. They have 5 children, and I believe there were no more.

III, 1, act. 24 years, very poorly educated on account of his bad sight, is said by his mother to have been very short-sighted and night-blind since early childhood. When examined (October 3rd, 1908) we estimated his myopia at about 10 D. by direct ophthalmoscopic measurement, and found the retinal vessels normal and no fundus changes, except moderate crescents. With his spectacles on his sight was conspicuously defective for objects 4m. to 5m. off in a dim light (partially darkened passage in his own house); without glasses he only read J. 4 word for word, but as he was almost illiterate this test was inconclusive; the Fs. to rough hand test seemed full. His mother said that he always had to be led home from church after evening service.

III, 2, act. 17 years, now has about 3 D. of myopia, and did not show any shortness of sight till she was about 13. Nothing was said about night-blindness in her; fundus normal.
III, 5, aged 13 years, is just like III, 1 in his sight; has been very short-sighted and blind at night since early childhood; now has about 10 D. of myopia, with moderate crescents, but no other fundus changes; read 1, 3 and 4 fluently both with and without her glasses. Like her brother, III, 1, she has always to be led home from evening church. The other two have no defect. There is a history of "blindness" in a half-cousin, but no particulars were to be had.

APPENDIX VI.

Leber's Disease.

(a) Abstracts of Published Cases in Chronological Order beginning with v. Graefe's case, 1858, to which are added some hitherto unpublished Cases communicated by friends or taken from my own note-books.

The references down to 1899 are taken chiefly from Habershon's paper in T.O.S., viii (1888), and Hormuth's Dissertation published in 1900 (title given on p. cxxxvii). The particulars of each case are also in many instances taken from Hormuth's tables. When, however, his abstract of a case seemed unsatisfactory reference was made to the original; but such reference has seldom led to any correction of Hormuth's rendering. All cases consulted in the original are marked with a star (*).

Male, onset at twenty years of age; failure progressed three months, both eyes: recovered to reading small print in four weeks; no ophthalmoscopic note. His brother, onset at 20 years, J. 20, no improvement; fundus normal three years later. Third brother, also attacked in same way at nineteen. Parents normal.

Usually quoted as Leber's disease, but the coincident family paralysis

![Figure 71](image)

and absence of ophthalmoscopic examination exclude precise diagnosis. 1, 1 blind from "amaurosis" at about 55. II, 1 amaurotic both eyes at 56; 2, living, aged 63 years, good eyes; 3, died paralysed but good vision.
at 56: 4, living at 60, paralysed, good sight; 5, living, act. 56 years, good sight, no mention of paralysis: 6, amaurotic, both eyes, at 48, died paralysed; 7, left eye amaurotic at 43; 8, amaurotic both eyes at 42, died, but age and cause not given; 9, normal, place in childhood not noted; 10, normal, act. 38 years.

Two brothers, both attacked at 23, in both eyes; central scotoma, moderate optic atrophy in one patient, no note in other; no recovery. Family history negative.

Three brothers and an uncle, loss of central V. with slight neuroretinitic appearances. Ibid., three brothers; ibid., two brothers; ibid., two brothers. All attacked between 18 and 23. Some improvement in early period of treatment in all.

1871. Case 77. Leber, A.f.O., xvii, 2, p. 249, family II. Two brothers and a sister affected out of six. Parents normal. II, 1 affected at 17, seen at 30. II, 3 affected at 28, and II, 6 at 19, both seen at early stage. All three recovered after some months from finger V. to J, 1

1871. Case 78. Ibid.

1871. Case 79. Ibid.
Family I. Five brothers in a sibship of six, and two maternal uncles. Parents normal. No recovery. II, 1 affected at 20; II, 2 at 13; II, 3 at 28; II, 4 at 13; II, 5 at 21; II, 6, sister, place in childhood not
stated, escaped. I, 1 and 2, two brothers of mother affected, ages at onset not given.

Mother, I, 2, affected at 43, her son and her nephew (II, 2 and I) as young adults (exact ages not given). No note as to sex of I, 1 or siblings of II, 1 and 2.


1, 3 affected at 21; II, 1 affected at 26; II, 2 at 24; II, 3 at 21; II, 4 at 27; II, 5 at 21. The three mothers, I, 1, 2, 4, normal.

Two brothers; elder affected in twentieth year, R, beginning six months before L, no recovery; younger brother (age not stated) affected in exactly same way.

Two brothers; ibid., two other brothers; ibid., three brothers. Ages of onset not given. One of them with V, reduced to J, 15 improved to reading J, 1 in eighteen months.

I, 1 age of onset not given; did not recover. II, 1–3, sons of sister of I, 1. II, 1 affected at 29; II, 2 at 23; II, 3 at 20; all attacked in same year. No recovery. No other cases in family. (Sex signs omitted in II, by oversight.)

I, 1 affected in his youth, and improved enough to read writing. II, 1 affected at 21; II, 2 at 27, “temporary improvement” in both; II, 3 affected, no particulars. The three sisters and parents normal.

I, 1 and 2 affected at 20; I, 3 not until 57; II, 1 at 19; II, 2 at 17. No improvement. Parents normal.
In a childhood of eight, first-born (♂) and four sisters normal. The
other three brothers (II, 2, 3, and 4) affected at 32, 25, and 22 respectively. Parents normal.

1879.* Case 90. Ibid.
I, 1 and 2 affected at 21; I, 1, at 59 years when seen; I, 3 to 6 normal sisters, one of whom (6) had five sons and one daughter by normal husband. The five sons (II, 1, 2, 3, 5, 6) all affected, II, 3 at 33, the other four about 20-21. No recovery.

1879. Case 91. Ibid.
Three brothers: eldest affected at 52, next at 49, last at 48. No recovery.

Father, I, 2, died at 74, 1878, good sight, his last child then quite young. Mother, I, 1, living in 1880, good sight, had fourteen conceptions, of which two miscarried; five boys and two girls (II, 6 and 7) died young of measles or whooping-cough; five living at date of record, viz., II, 1 affected at 16, II, 2 at 15, watched till 19, no recovery, severe case; II, 3, age at onset not given, but apparently younger than 1 and 2; II, 4 and 5 quite young at date of record.

Five generations; transmission by affected males. No consanguinity. I, II, and III, 1, all said to have been affected. IV, 1 affected, but after
being "blind" recovered enough sight to resume ordinary occupations; his son (V, 1) affected, no details. IV, 2 normal, had nine children (V, 2-10), of whom first (V, 2) was affected at 48 and seen at 49, and fourth (V, 5) seen at 43, age at onset not given. The two daughters (V, 3 and 8) normal at 46 and 33 respectively. The other five died in infancy.

sexes not stated. IV, 3 affected, age of onset not recorded; her son (V, 11) affected at 49, seen at 50. "Most became affected between 25 and 40."

Family I. 1, 1 and 2 normal sisters. II, 1 affected at 25; II, 4, 5, 6 all affected at 20. III, 1 at 20; III, 3 and 4 between 17 and 20. No consanguinity.

1882. Case 95. Ibid. Family II.
1, 1 affected at 20; his two sisters, 1, 2 and 3, normal. II, 1 affected
at 39, seen at 44; II, 2 affected at 12, seen at 33; II, 4 affected at 25, died at 28 of diabetes; II, 5 and 6, brothers, affected at 20. No consanguinity. No record of any sisters in II. (Numerals omitted from Figure, in error.)


I, 1 affected at 20; I, 2, age at onset not stated; I, 3 normal. Of her four children the youngest, II, 4, affected at 11 and seen soon after; eldest, II, 1, act. 24 years, and the two daughters, normal. I, 4 an affected female cousin of I, 3. No consanguinity.


Four brothers affected at 20, 30, 32, and 37; order of birth not recorded. Also a male nephew at 19; presumably his mother was sister to the four affected brothers, but this not stated.


In a sibship of 8, 4 brothers and 4 sisters, the eldest brother affected before 23, and died of epilepsy at 23; second brother affected at 21, died of phthisis, and was alcoholic; third brother affected as a young man, and died in an asylum; fourth brother affected at 30, was 45 at date of record, and had had attacks of insanity. Of the sisters, the third, affected at 40, was seen in the early stage at about same date as the fourth brother; her two older sisters (places in sibship not recorded) very excitable; youngest sister and eighth born normal.


Notwithstanding the title the cases read like typical examples of

Leber's disease. Parents (I, 1 and 2) and grandparents normal; II, 1, act. 28 years, normal; II, 2 affected at 22, seen at 23 (author's Case 3); II, 3 affected at 18, and seen at same age and until 20 (author's Case 1); II, 4, act. 18 years, normal; II, 5, affected at 15, seen nearly a year later (author's Case 2). No improvement of II, 3 in two years.


Two brothers; one failed at 22 rapidly, seen at 31, had not improved; Myr. 1-5 D., V. J. 19; married at 19, and had two children before eyes failed, who are living and well. Other brother, five years younger, failed
at 17, seen at 26 (same time as elder brother), and again at 34 (1895), no improvement; V. J. 16, close to and fingers 2 m., My. 6 f). There are several other brothers all normal (no mention of sisters); parents saw well; father died of cancer, mother phthisis.

Three brothers: one affected at 31, and seen at 32; the other two failed in exactly same way, but ages not given.

I, 1 a weakly woman; I, 2 died of cancer at 67; II, 1 not much information, but no cases known on her side; II, 2 was one of a very large sibship, of whom only three lived to grow up, all males; he died at 55; no consanguineous marriages; no recoveries; III, 1 affected at 16, seen at 45; married, no issue; III, 4 affected at 15, seen at 39, some children; III, 7 affected at 17, at 32 had some children; III, 9 affected at 12, act. 27 years when III, 1 was 45; III, 2, 3 and 10 died of phthisis between 17 and 22; III, 5 of disease of spine at 24; III, 6, 8 and 11 (act. 24 years) normal.

I, 1 and 2 brothers, normal; II, 6 affected at 21, act. 50 years at date
(1884): II, 7 not affected until 60, and became diabetic later; III, 2 affected at 34, no recovery, living aged 55 (1906); III, 3, age of onset not known; III, 4 affected at 16; II, 3 all died of phthisis, normal sight; II, 8, 9 and her two adult sons III, 5 and 6, and II, 10, single, all normal.


Two brothers; the elder attacked at 22, and seen soon after; the younger attacked at 19, seen at intervals for four years, and V. remained about \(\frac{5}{6}\). The brothers failed within about four months of each other. A brother of their mother (Boxall) has had bad sight many years.


Two brothers and, perhaps, a sister. Elder brother affected at 23, act. 30 years at record, no recovery; other affected brother failed at 19 when elder brother was 30. A sister had sight and wears glasses, not seen, and has fits (epileptic). One other brother and two other sisters normal; the brother died of phthisis. Parents normal sight; father died of phthisis.


II, 1 attacked at 23, died unrecovered at 40; II, 2 married at 23, and had eight children and one miscarriage in eighteen years, the eldest act. 17 years; the last child, act. 11 months, born when she was 40; all suckled. Her sight failed at 40 when suckling last child.


Three brothers; II, 1, firstborn, attacked at 27, seen at 40; II, 2 attacked at 32, seen very soon after; III, 3 seen soon after onset, age not given. The two sisters, places in sibship not given, normal.


I, 1 and 2 normal, but an indefinite history of bad sight in relations of I, 2. II, 1 normal, and II, 2 attacked at 48; II, 3 at 9; II, 4 at 21; II, 5 at 14. III, 2 attacked at 27; III, 5 at 33; III, 7 at about 20; III, 8 at 29; III, 9 at 18, act. 37 years at record; III, 10 at 20. IV, 4, age at attack
not given; IV, 9 at 17. Four of those affected were seen; onset rapid in all, then stationary at V, about fingers, with atrophic discs when seen about 15 to 20 years after onset. In March, 1909, Dr. Haswell was unable to supplement the notes.


Six brothers, and two of their maternal uncles. Age of onset 21 in one of the six brothers, not given for the others.


Pedigree of four generations: I, 1 became blind or nearly so at 40, eyes looking natural. II, 1 and 2 had respectively 10 and 5 normal children. II, 3 married 1, both normal and not consanguineous; issue, 11 children, all grew up, and one miscarriage; of the 5 sons, 1 affected. III, 3 at 25, seen at 27, died of lung inflammation at 37, leaving one son as soldier in
1909, with good sight), and 5 daughters, all good sight; III, 4 affected at 21, seen at 25, living in 1909 and has 5 normal children; III, 7, one eye only affected at 6, and still same in 1909; III, 10 affected at 6, seen at 13, and heard of in 1909 as in same state at 30, unmarried. The 3 daughters, III, 5, 6, and 8, aged in 1909 about 40, 39, and 35 years, have 19 children, all normal.

I, 2 affected at 51, living at 75; her ascendants not known. II, 1 affected at 26; II, 3 affected soon after severe cupping for yellow fever at 20, now 47 (1891); II, 4 at 33; II, 5 at 25, now 36, has two sons of 8 and 6 (1891) (III, 3 and 4). III, 1 and 2, act. 18 to 12 years, all normal.

I, 1 to 4 all normal. II, 3, first wife of 4, issue normal; 1, 2, and 4 all normal; II, 4 died at 62, alcoholic; II, 5, second wife of 4, died at 58; of her siblings, II, 7 failed in sight at 50, cause unknown, died at 52. III, 3 affected at 26, 41 at record; her "eldest son," (IV, 1) affected at 20, papillitis chiefly L. V. ⅓ in L., normal in R., contracted. III, 4 at 30; III, 5 at 31, married at 27; III, 6 at 32, married at 28; III, 7 doubtful, slight case, at 30. No consanguinity. (Cf. Case 110.)

Vague history of similar blindness in ascendants of 1, 1 and 2. II, 2 affected at 34, II, 3 at 28, II, 5 at 18, seen by author; II, 6 and his ascendants normal. III, 1 "blind," III, 2 very amblyopic but exact data wanting. III, 3 affected at 19, seen by author.

A man, act. 37 years, R. affected about two months before L., typical, except for a small hemorrhage in L. retina near O.D. in early stage when neuritic appearances were present. A brother of his mother was "blind" from "disease of optic nerves."

Three brothers in childship of 16: II, 4 affected at 24; II, 9 at 27; II,
13 at 22. Eight others died in infancy and 2 miscarried. III, 1 to 10, act. from 6 years to a few months, the twins (III, 2) and III, 6 died or still-born. 1, 6 and 7 normal; one of I, 5, and also II, 11, had hysterical fits. In II, 9 L. eye improved from $\frac{a}{10}$ to $\frac{a}{2}$ in between two and three months.


I, 1 and 2 and collaterals normal. II, 1 affected at 11, seen at 51, no improvement; II, 2 affected at 10, died at 33; II, 3 at 12, seen at 48 with V. $\frac{a}{10}$, age at marriage not stated; has had only 4 children, 3 of them affected; III, 1 died act. 3 years; III, 2 affected at 11. V. with H. 45 D. corrected $\frac{a}{12}$, four months later $\frac{a}{10}$, a year after second note $\frac{a}{6}$; III, 3 failed down to $\frac{a}{10}$ at act. 9 years, with pale O.Ds.; III, 4 failed at 8. with H. 3 and slight As. corrected $\frac{a}{6}$, O.Ds. much congested; two years later only $\frac{a}{12}$ (1898). No consanguinity.

1897.* Case 117. Snell, T.O.S., xvii, p. 60. Author's Case 1.

I, 1 to 4, and II, 1 to 3 all known to have had good sight. In III, 1 act. 32 years: 4, 29; 5, 27; 7, 24; and 8, 21, all severe amblyopia from their earliest recollection with pale O.Ds. and no other changes, no scotoma (but V. "better at night") and no contraction of Fs. V. from $\frac{a}{10}$ in III, 1 to $\frac{a}{12}$ in III, 8. III, 2 doubtful, is colour-blind like the rest, but V. R. $\frac{a}{6}$, L. $\frac{a}{12}$, and no note of condition of O.D. IV, 1 and 2, young children, 1 act. 3 years examined and normal.

1897.* Case 118. Ibid. Author's Case 2.

Two brothers, both affected in same way at 17 years. No consanguinity and no other cases known in relations.

1897.* Case 119. Ibid. Author's Case 5.

III, 1, 3 and 5 all affected at 13, seen at same date at 10, 32, and 26 respectively. III, 2 at 36; 4 at 29; 6 at 23, normal. III, 1 and 3 married some years, no issue; III, 5 has three normal children. All in I and II lived to good age with good sight. No consanguinity. II, 1, 63, and 2, 64 at record, and were about 22 and 23 at marriage.
1897.* Case 120. Ibid. Author’s Case 6.
II. 1 “blind” in middle age and never recovered; II, 2 lived to 85, good sight; II, 3 lived to 67, he and 1, 1 good sight; III, 1 affected at 52, seen again at 62, has three sons all normal; III, 2 at 57, seen same time, six children all normal, subject to fits from age of 24 to 40; III, 4 affected at 36, seen at 44, married, no issue. III, 5, act. 43 years; III, 6 and 7 died in infancy. No consanguinity.

Three brothers affected, II, 1 at 19, II, 2 at 25, II, 3 at 20, act. 24 years at record; several others died young. Parents normal.


I, 1 affected at 20, and improved in about a year. II, 1 at 27, seen soon after; II, 2, six years younger than II, 1, affected at 20; II, 3 epileptic and subject to migraine. Parents normal and not consanguineous.

1900. Case 123. (First of Leber’s 9 new unpublished cases) Hormuth’s text, p. 16, and his Tables, p. 114.

Four males, sons of three sisters, ages not given; first case (II, 1) seen a year after onset.


Two brothers; elder affected at 39, younger at 18. Family history not given.


Two brothers affected at 20 and 27; an uncle, brother of their mother, had same disease at 24.


Two male cousins affected at 21 and 30; their mothers were sisters. A brother of the two mothers, maternal uncle of the other two cases, also affected at about 18 or 20.


In a childhood of 5, the 2 brothers affected at 25 and 17, and of the 3 sisters, 1, much younger than the brothers, affected at 42; the other 2 sisters normal. Parents normal.


Two brothers, affected at 24 and 32. History incomplete.


In a sibship of 8, 2 brothers affected at or about 20; the elder now 40, the other quite recent, at 20 years, at date of record. Parents normal.


Two brothers affected at 18. Nothing else recorded.


Sibship of 3; 2 brothers, the elder affected at 27, the other, 9 years younger, at 18; one sister between them normal. Both recovered, the elder to being able to read, the other to being able to resume his painting.


1, 3 affected, but age of onset not noted. II, 1 and 2 each affected at about 23, 1 being two years older than 2. One normal sister, age not given.

Two brothers; one affected at 14, seen at 40, V. /₁₀₀₀ or ⅓₀₀₀; the other about two years younger, not affected till 40, seen soon after. No other cases known in family.


Nephew and uncle; nephew attacked at 31 and seen at 37; age of onset in mother's brother not recorded.


I, 1 and her husband, 2, a physician, normal. II, 1, 2, 3, three normal daughters; no record of any other children. III, 1 affected at about 55, seen some months later, and improved definitely in four months; III, 3 affected at 22; III, 8 at 40; age at onset in III, 4 not given.


Two brothers; one failed at about 48, seen two and a half years later, aged 51; other brother affected at 27, present age not given. Parents, good eyes.


I, 1 said to have had the disease. II, 1 affected at 25, seen at 58 (1897), typical central defect with also concentric contraction of Fs. III, 1 affected at 24, seen at 36 (1897); III, 2 at 25, seen at 35. Age of onset in III, 3, 4, and 5 not given. One of these three epileptic, and others mentally affected.


Five generations. I, 1 and 2 normal, had one normal daughter, II, 3, who transmitted the disease to her sons by both her husbands, and 2 sons, II, 4 affected at 25, and II, 5 at 23; all their descendants in III and 4 and 5 to date, normal. III, 2 affected at 20; III, 4 and 5 both at 19. IV, 1 at 21; IV, 3 at 22; IV, 4 at 17; IV, 5 at 19. Connecting line between II, 4 and his children III, 6 and 7, also between II, 5 and his children III, 8 and 9, accidentally omitted in the Figure.

1895. Case 140. E. N., unpublished, St. Thomas's Hospital, 1890-91. (Pitt, Barrett, and Wilson.)
I, 1 and 2, no information. II, 2 to 6, their issue. II, 2 had bad sight and married a first cousin with bad sight, but no particulars of the disease or of kind of cousinship, nor of sight of their 12 children (III, 1). II, 3 had 10 children, of whom a son and daughter (III, 2 and 3) had some defect of sight, but no details. II, 3 also some unknown affection of sight; II, 5 affected at 14, and went to Moorfields then; living, act. 61, in 1891, sight not improved; II, 6 living, with good sight. III, 5 (Mrs. Pitt) failed at 36, married at 18, no children; III, 8 (Mrs. Barrett) failed at 26, has had children, too young to show the disease (IV, 1 and 2); III, 9 (Lynham) failed at about 22, seen nine months later; III, 10 (Wilson) failed at 22½ after influenza, seen six months later; III, 12 (fifth born), fits.

1895. Case 141. E. N., unpublished, St. Thomas's Hospital, 1885 and 1893. (Donovan.)

I, 1 and 2 had good eyes. II, 1, Hodgkins, of Birmingham, had a son (III, 1) affected at about 10; II, 3 married Jones and had son (III, 3) affected so early that he never learned to read, act. 30 years in 1893; II, 4 married Donovan II, 5 and had issue. III, 4 (J. Donovan) affected at 30, seen at 36 and again at 43; III, 5 (Mrs. Leonard) married at 22, affected at 33, and seen soon after; III, 8, 10 who died quite young. IV, 1 affected in early life, could never see his work properly; IV, 2, five children of III, 4, two dying early; IV, 3, four children of III, 5, two dying early.

1895. Case 142. E. N., unpublished, Moorfields Hospital, 1896. (Laxford.)

I, 1 believed to have had bad sight; had two sons undoubtedly affected like the rest (II, 1 and 2); I, 2 certainly affected; had one normal daughter (II, 3) with normal children (III, 1, 2, and 3); one affected daughter (II, 4) and two affected sons (II, 5 and 6); no record of other
children. II, 5 and 6 apparently s.p., but II, 4 had seven children, viz., III, 4 died in infancy; III, 5 died unaffected at 60, probably heart failure; III, 6, act. 60 years in 1896, no issue, believed to be affected;

III, 7 affected, act. 58 years in 1896, had then had four children, IV, 1 died of influenza, and 3 living; III, 8 probably affected, died suddenly at 30; III, 9, patient, failed at 26, seen at 58 (1896), has normal children (IV, 5 and 6). IV, 8 to 11, 9 children of III, 10, 6 of whom died young.

1895. Case 143. E. N. (unpublished), Moorfields Hospital, 1897. (Philbrick.)

I, 2 was affected; II, 1 affected at 14, recovered sufficiently to be able to read; II, 2 affected at 33; II, 3 and 4 each at 25; II, 5 and 6 are the last born of the childship; III, 1 patient, act. 22 years.

1895. Case 144. E. N. (unpublished), Moorfields Hospital, 1890. (Haile and Drudge.)

I, 2 reported to have had the family blindness; II, 1 to 6, order of birth not known; II, 1 and 2 affected; II, 1 had 16 children, of whom first born, III, 1, was affected at 22 and seen at 29, unmarried; III, 2 and
3 unmarried; III, 4 affected at 17, and seen then; III, 5, 12 who died young. No consanguinity.

1895. Case 145. E. N. (unpublished), Moorfields Hospital, 1891. (Booty.)

I, 1 affected, "nearly blind," has children, but no details; I, 2 similarly affected, and has an affected son (II, 2); II, 7 affected at 12, seen at 49, no recovery, married, no issue; II, 8 also affected, unmarried; II, 9 unmarried. No consanguinity.

1907.* Case 146. Gunn (R. Marcus), T.O.S., xxvii, p. 221.

Incomplete, and cannot be completed. I, 1 affected in childhood, married a first cousin (kind of cousinship not recorded), and had (up to 1907) two children; II, 1 affected at 5, seen at 8; II, 2 affected at 3, seen at 4.

1887.* Case 147. Lawford, St. Thomas's Hospital Reports, xvii, p. 158. Author's Case 1.

I, 1 and 2 sisters; I, 1 had at least three children, of whom II, 1 certainly got the affection at 19, and 2 and 3 probably suffered; I, 2 had seven children, of whom all the sons suffered; II, 7 at 31, seen at 32; II, 8 (4 years younger than 7) at about 18; and II, 9 (3 years younger than 8) at 19; eldest, II, 4, at 29 years, and youngest, II, 10, 22 at record; II, 7 was also congenitally colour-blind.

1887.* Case 148. Ibid. Author's Case 2.

I, 1 good sight, but epileptic fits, husband good sight; II, 1 affected at 18; II, 3 living and normal; II, 2 died at 2, "consumptive bowels"; II, 4 still-born; II, 5 at 1 year; II, 6 and 7 at 1 year of diarrhoea.


I, 2 married twice, by first husband (I, 1) three sons; II, 1 affected at 14, seen at 38; II, 2 affected at 10, seen at 36; II, 3 began at 29, seen at 34; by second husband (I, 3) 2 sons; II, 4 affected in eleventh year, 26 at record; II, 5 affected in twentieth year, 24 at record; one daughter II, 6, who at 20 became extremely amblyopic of both eyes (fingers 12 in.) with contracted Fs. but no ophthalmoscopic changes, and recovered perfectly; no note about her pupillary reaction; probably hysterical amblyopia. No positive information about vision in parents, nor as to consanguinity.
Three brothers attacked at 32, 29 and 25, and a nephew at 36; no female suffered.

Mother and daughter. Mother, at 26 years, sight bad as now from earliest recollection; fingers 8 feet, scotoma, O.Ds. white, retinal vessels normal; very undergrown, but well-proportioned, teeth and skull normal; has 4 living siblings, one of whom (♀) is, like her, very small; one other died at 2°; her parents normal sight. Daughter, at 3 ½ years, sight failing 2 years, sees large objects; O.Ds. white with some surrounding haze, no choroiditis; had many convulsions at about 18 months old; teething and walking both delayed; rather undergrown; skull normal; no note of any siblings.

I, 1 and 2 normal; II, 1 affected at 23, II, 3 at 24, II, 4 at 25; III, 1 normal, III, 2 affected at 25, III, 3 at 23, III, 4 at 24.

I, 1 and 2 both normal; II, 1 affected at 39, II, 4 at 25, III, 5 at 13, III, 7 at 22, III, 13 at 20.

I, 1 died at 60 and I, 2 at 72, both with good V. II, 1 (author's Case 3)
failed at 30. II, 2 had glaucoma; married, no issue. II, 8, 3 who died “young” but with good sight. III, 2 (author’s Case 2) failed at 24, has one child of 14 (IV, 1). III, 7 “bad sight” without further details. IV, 2 (Author’s Case 1) failed at 25; IV, 3 two young children, good sight. Married, but without issue, II, 3; III, 1, 5, 9, 10, and 12.

1898.* Case 155. Leitner, second paper. Ibid., No. 3. Author’s Case 1.

I, both normal; II, 9 sisters and 3 brothers all normal and all having

I

II

III

children. III, 1 affected at 18, III, 2 at 24, III, 3 at 29; III, 4, three normal sisters; III, 15 at 18, act. 21 years at record. III, 14 normal at 32; III, 24, 11 siblings, eldest 21; III, 25, another 11, eldest 24; III, 26 act. 9 years. No consanguinity, no early deaths.

1898.* Case 156. Ibid. Author’s Case 2.

I, 1, 2, and 3, normal. II, 1, act. 20 years, and 2, act. 18 years, also 3 and 4, all normal. II, 5 affected at 15, II, 6 at 12, both seen four years later, no recovery. No consanguinity.

1898.* Case 157. Ibid. Author’s Case 3.

Single case in a coming on at 16, seen at 17; V, fingers 0.65m., symptoms typical; has 2 sisters, normal. No other details. No consanguinity.


Male twins; one affected about a year before other, at about 16 and 17 respectively. R. eye failed before L. in both. Parents, good sight, but a brother of the mother had the same disease at about 20.

1901.* Case 159. Gallemarets, Policlinique, Bruxelles, April 1st. Author’s Cases 3 and 4.

I, 3 reported affected like the others, and all his 5 children (II, 6) said to have bad sight of the same kind; II, 1 affected at 21, act. 33 years and unmarried at record (author’s Case 4); II, 2 affected at 17, seen soon after (author’s Case 3).

1901.* Case 160. Stood (Dr. W., of Barmen), K.M.j.L., 39, i. p. 238.
Fourteen members of a family affected. Two of them, a young man whose case at first looked hopeless, and his sister, recovered V. $\frac{5}{6}$.


I, 1 died insane. III, 3 affected at 21, now 30 (1902). IV, 1 affected at 21, now 27; IV, 2 and 3, both at 21, now 44 and 29 with V. from $\frac{5}{6}$ to $\frac{1}{6}$. Numerous other descendants of I, 1, but no other cases. No known consanguinity.


I, 1, father of either II, 1 or II, 2, was "blind"; no details. II, 1 to 4 all saw well to end of life; III, 1 died at 53 of liver disease; III, 2 lived to 73, good sight. IV, 1, probably firstborn, about 44 (1902), IV, 6, 37 and IV, 7, 36; two of these three sisters have several children, the eldest 12. IV, 2 affected at 22, now 43 (1902); IV, 3 at 22, now 42; IV, 4 at 27; IV, 5 at 22; IV, 8, patient, at 30, seen soon after, final result not known. None of the four affected brothers recovered; IV, 9 died at 13, IV, 11 at 11, IV, 10 at 13, the other three at a few months.


I and II all said to have been normal; III, 1 affected at 20, 29 at record; III, 3 at 20, 24 at record; III, 5 at 20, seen soon after; skull normal.


I, 1 and 2 affected at 20, one of them died of "cardiac dropsy"; I, 3 also died of "dropsy" at 60; I, 4 at 70 of "stroke"; II, 1 said to have had "weak sight"; II, 2 affected at 21, no recovery, 47 at record; skull normal; II, 3, patient, at about 27, no recovery, 41 at record.


I, 1 affected when in Army 1871, now about 55 (1905), no recovery.
I, 1 affected at 29, died of dropsy at 32 (1889), would be 48 in 1905, no recovery; II, 2 affected at 25, seen at 35 (1905), no recovery. Married at 28, no issue; II, 4 affected at 21 (1905), seen soon after, skull unsymmetrical, slight proptosis on both sides from shallowness of orbits.


I, 1 and 2, first cousins, but kind of cousinship not given. II, 1 died of diabetes at 43; II, 2 living, 86 at record; II, 3 became rapidly blind at 30, probably optic atrophy, died at 49. III, 1 died at 43, cerebral tumour; III, 3 at 51, cerebral softening; they had a son, IV, 4 (author's Case 2), affected by typical Leber's disease at 26, seen at 38, no recovery. Syphilis two years before failure of V. III, 1 affected at 24, seen at 53, said to have remained the same for 20 years, and then improved to reading largish letters; for years could only with difficulty see to go about (author's Case 3). IV, 3 affected at 22, seen at 26, somewhat improved. V, 5 at record (author's Case 1). (Indicating numerals to IV omitted in error.)

I, 1 gouty. III, 3 affected at 24, seen soon after (author’s Case 4); III, 1 affected at 23, seen at 46, when he was absolutely blind and had paralysis agitans (author’s Case 5). II, 1, 2, and 4, all suffered from same disease at almost same age as the other two. II, 3, 5, 6, died young; II, 7 died at 18.


Four cases with typical symptoms and mode of descent; consanguinity of paternal ancestors of one case, but no cases of the disease on that side. Narrow base of skull in some of the affected ones, but this still more marked in III, 2 from unaffected division. No miscarriages, and apparently no early deaths. Ages in IV: 1 was 27 in 1907, 3 was 17, 6 was 16. III, 6 married at 19; only two children, IV, 1 born five years, and IV, 2 ten years after marriage; both labours natural, no forceps. III, 4 affected at 23, 58 at record (1907), unmarried (author’s Case 2); III, 7 affected at

35, 48 in 1907 (author’s Case 3); III, 8 affected at 48, 53 in 1907 (author’s Case 4). IV, 2 affected at 2½, seen six months later. III, 9 had gross central choroiditis in both when seen at 12 in 1907, with V, much reduced and My. 2 D.


I, 1 to 4 normal. II, 2 affected, her sister normal. III, 1 normal, her three brothers affected, two with reduction of V. to fingers at 2 m., the other to V. ⅔ in R., ⅔ in L. IV, 1 affected at 15, seen three months after.


Single case in boy coming on at 8 in September, 1908, with slight neuritic appearances. V. went down to R. ⅔, L. ⅔, with F’s, much reduced, then, at end of December, began to improve rapidly, and by end of January, 1909, V. was ⅔ in each, R. better than L. No other cases known, but mother very ignorant of the family history.

I, 1 affected. II, 3 affected. III, 1 affected at about 20, patient of Mr. Doyne at Oxford, now 33 years, his six siblings all normal; III, 9 affected at 16, seen 1917 and again 1909 when at 24 years, V. $^a_0$; III, 13 affected at 10 (March, 1904), V. down to $^a_0$, in July began to improve, and by March, 1905, had recovered to $^a_0$ each eye, and remained same in February, 1909 (act. 15 years); O.D.'s became somewhat pale some months after onset. III, 12 died act. 3 years; III, 14 also died in childhood. No miscarriages. III, 8 act. 27 years. II, 9 died at 21; II, 10 married, no issue; II, 1 died at 40.

![Fig. 171](image1)

![Fig. 172](image2)

Case 172 seen by Leber, 1871; Magers, 1898; Vossius,* 1899-90.

I, 1 affected at 20, no recovery, lived to 72; I, 2 at 21, no recovery, lived to 73; I, 3 normal, lived to 83. II, 3 affected in 17th year in 1866, and seen by v. Graefe then and by Ewers in 1869; married later and in 1899 had two sons and two daughters (erroneously marked as one of each), aged from 24 to 16 years. III, 2 affected at 23 (1894), improved, and in 1897 could read newspaper with a magnifier; had variola, whooping-cough, scarlet fever, and diphtheria in childhood, with nephritis and dropsy, and later paralysis of right arm and leg, and later of left leg, then good health till 16, when he had pneumonia, now (1897, act. 28 years) healthy. III, 3 affected at 22, act. 27 in 1897; III, 1 act. 29 years, and III, 6 act. 22 years (1897); III, 7 affected at 19, when he was seen by Magers two years later (1898) with V. fingers 5 m.


I, 1 failed in sight after a slight accident, and did not recover; I, 3 and 4 living, but no record of their sight, presumably both normal. II, 1 a blind and idiotic daughter of I, 2; she cannot walk. II, 2 to 12, 11 siblings, of whom 12 died at 5, the other ten living and aged (in 1909) from 41 to 20 years. II, 3 affected at 27 (1896, Moorfields, under care of E. N., seen by Mr. Batten, 1909); no recovery. II, 4 affected at about 37, attending Mr. Worth (Moorfields). II, 5 affected at about 22-23, now 36, and still attending Mr. Batten (Western Ophthalmic Hospital). II,
6 affected at 22 (1897), and still under Mr. Batten's care, at 34 years. Question of lead poisoning was raised, as at least three of the affected brothers were plumbers, but there was no decided evidence of plumbism.

1906. Case 174. Mr. C. H. Usher. Cases of Leber's disease in a pedigree drawn up to illustrate albinism. (Forthcoming Albinism Memoir, Fig. 130.)

III, 10 and 11 Leber's disease set in at about 30; age of onset in III, 12 not recorded. In IV, 4 Leber's disease present at 30, and in IV, 5 at 25. In II, 6 sight failed in old age, and also in two of her brothers, but the nature of the failure not known. I, 1 also said to have failed in sight as an old man. The albinos were offspring of two mothers by same father, the father (III, 12) having Leber's disease, the two mothers almost certainly unrelated to each other. No consanguinity.

I, 2 good sight at 86; no information about 1, 3 and 4; II, 1 first wife

of II, 2 had only one child (III, 1), who in her turn had five normal children (IV, 1); II, 3, second wife of II, 2, had five children (III, 2 to 6), of whom III, 2 failed at 30 and was seen at 31 (author's Case 2); and
III, 5 failed at 19, and was seen three months later (author’s Case 1): IV, 1, 2 and 3 all normal, and no miscarriages or early deaths: IV, 4, a seven months child, paralysis of lower limbs.

1809.* Case 176. Ibid. Author’s Cases 3 and 4.
1. I died of phthisis at 35; II, 2 died blind and paralysed at 50, no details of the blindness; II, 1 progressive failure of V. at 48 and optic atrophy found, at 50 sudden hemiplegia; II, 3 died from heart disease, blind, at 58, no details of the blindness; either she or another sister (II, 2) had a son (III, 6) who went “blind” at 30, no details; III, 1 affected at 31, seen at 33, no recovery (author’s Case 4), has 2 children; 8 and 7 years (IV, 1 and 2), and no miscarriages; III, 2 affected during military service and recovered; III, 3 affected at 27, seen soon after (author’s Case 3), has one child who died young; III, 4, five who died young; III, 5, two stillborn.

1. I, 1 and 2 first cousins, but kind of cousinship not noted; II, 1 died at 4 months; II, 2 said to have never seen, at 1 could only see large objects and O.Ds. very atrophied (author’s Case 2); III, 3 thought to have seen well till 6 months old; at 1½ years sees large objects, O.Ds. very white. Both children intelligent and good tempered. Syphilis not mentioned. No other history of blindness in family. This case has sometimes been quoted as perhaps being an instance of Leber’s congenital retinal atrophy without pigmentation.

1. I affected at 14, if not earlier; L. much worse than R., typical scotoma in each; now 31 (1907). II, 1 now 10 with typical scotoma in each, affected since early infancy. II, 2 age not stated, sees quite well. At date of record there had been no more births.

Male affected in 28th year, perfect recovery in a year or year and a half. His brother, ten years younger, attacked at 25; final result not known. Two males, first cousins, had the disease at 28 and 35 years respectively; the latter is said to have recovered perfectly, the former did not. These two pairs of brothers were sons of two sisters.

1894.* Case 180. König (Hornamh, p. 94).
Man, 22 years, no recovery in one year. A brother of his mother (maternal uncle) and a cousin also on mother’s side were blind of optic
atrophy; the uncle improved. Also a great uncle on mother's side was affected. Four cases, all males.

Case 181. E. N. (Littlechild). St. Thomas's Hospital (Out-Patient's Book; v. p. 133), February to May, 1890.

Blindness from birth with optic neuritis and large skull in three siblings. Probably not a true case of Leber's disease. Parents, first cousins, had 8 children and no miscarriages.

No history of blindness in rest of family, except, perhaps, in a male, "second cousin" of I, 3 said to have been blind all his life. I, 1 and 2 good health. No history of syphilis.

II, 1 and 2 good sight and health; one of them squints; 6 and the next born, which by mistake is not shown and should be 7, also healthy and see well. II, 4 died at 7 weeks, but could see.

II, 3, quite blind from birth; taken to Middlesex Hospital when a baby and told "the nerve was inflamed." February 28th, 1890, act. 7 years; Ps. motionless before mydriatic, but dilate widely after its use; L.O.D. seen with difficulty; it is hazy, and one vein decidedly enlarged, but no swelling and no visible atrophy; R. not seen; shadows some H., but degree not measured. Cranium rather large, forehead broad and prominent, the eyebrows overhanging the orbits very much, so that the eyes are extremely sunken and look small, although really of normal size; nose short; face well formed; speaks well, and seems intelligent.

II, 5 was blind from birth; died at 15 months of age; no particulars.

II, 8 (erroneously marked 7) brought in February, 1890, act. 7 months. Appeared to have no p.I., and mother said she was certain the child had never seen. Her seeing children had all noticed the light very soon after birth; this one never did so at all. February 28th, 1890: Pupils small, equal and motionless to light; irregular slow nystagmic movements and frequent strong convergence of eyes. O.Ds. swollen and very hazy, and veins tortuous. Head large and square, fontanelle open, frontal eminences square; ribs slightly beaded; spleen 1½ in. below costal margin; for some weeks past head sweating; suckled, but for the last two months some bread and oatmeal in addition; has had no illness and no fits. Though quite blind the child screws up her eyes in sunlight, but takes no notice of lamplight. Last seen in May, 1890, in status quo.

Case 181a.* Rampoldi, Anna, di Ott., xii, pp. 269-271.

I, 1 blind of "gutta serena" at 35, dead at date of record; II, 1 good sight; II, 2 became blind at 35 and H, 3 between 35 and 40, also of "gutta serena"; III, 3, optic atrophy came on in R. soon followed by L. early in 1883, act. 67 years, R. going to complete blindness, L. not so severe; had an attack of gastro-enteritis with some loss of blood two or three years before eyes failed. III, 4 living, good sight; III, 5 living but blind, probably of same disease; III, 6, act. 73 years at record and quite blind; sight failed from the same disease at 65. IV, 1 act. 33 years, good sight; IV, 3, act. 34 years, nearly blind, age of onset not stated; is married.
Optic atrophy without other changes coming on in II, 4 at about 1½ and reducing V. to finger-counting in a few months; in II, 5 at 1½, and in II, 8 at 10. The disease set in in all three during about the first half of 1881. Mother showed evident signs of syphilis shortly before birth of II, 4; she also had four or five miscarriages (not marked on the diagram, Fig. 181b) between II, 4 and 5; also II, 3 died at 14 months and II, 7 at 1 day old. II, 1, 2, 9, and 10 reported healthy. [If syphilis were the cause of the optic atrophy in these three siblings, why did the disease set in at approximately the same date (1881) in all of them? Was there some additional cause, such as lead poisoning or influenza? — E.N.]

Case 181c.* Suckling, Lancet, 1887, ii, p. 1271.
I, 1 became blind at 50. II, 1 went completely blind from double optic atrophy which came on gradually when he was 50; his sister, II, 2, was blind, and a female cousin (II, 3) on his mother's side is also quite blind (sex of I, 2 not given). No history of syphilis or disease of nervous system, and no signs of locomotor ataxy.

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**Fig. 181a**

II

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**Fig. 181b**

II

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**Fig. 181c**

II

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**Fig. 181d**

Unpublished. Case communicated by Mr. Jameson Evans (Birmingham).
No information about the ascendants of Gen. I. I, 1 to 7, five brothers, of whom three were affected, and two sisters, both of whom married and had children; one of them (I, 3) carried the disease, the other (I, 6), had only daughters, and therefore the point could not be determined. II, 4 to 12, nine children of I, 3, six male, of whom three are affected, three female of whom one (II, 8) died at 2; one of the survivors (II, 7) carries the disease, and the other (II, 6) has children of both sexes, none of whom have suffered hitherto (but their ages not stated). III, 11 and 12, twins, died at three months; III, 4 still-born; III, 5 aged 14 years, affected; III, 6, 7, 8 have not reached the usual vulnerable age.

(b) References to Cases of Leber's Disease Illustrating Various Statements in the Lecture.

Recovery or marked improvement of sight is to be found in affected members of the following pedigrees: 77; one of either 83, 84, or 85; 87, 93, 49, 115, 116, 122, 132, 136, 141, 144, 160, 170, 171, 172; and in a few others.

"Anticipation" in Leber's Disease.

(i) In successive generations, the phenomenon is well shown in the following cases: 80, 49, 96, 108, 111, 112, 113, 50, 153, 154, 51, 168, 174.

Case 112, Despagnet, shows the anticipation in three generations, and in the case occurring in the third generation one eye recovered whilst the other passed into atrophy of the optic nerve.


Transmission by Affected Males.

In six pedigrees containing only male cases of the disease 13 of the affected men became fathers, and had from 48 to 56 children, not one of which suffered from the disease, viz., 45 omitting Gens. I and II, 120, 47, 168, 174, 167.

In five pedigrees containing cases of the disease in both males and females, 10 affected men became fathers and had 44 children, of whom 4 ♂ and 2 ♀, six in all, had the disease, viz., 93, 49, 108, 142, 50.

Adding the two series together we have 23 affected males, who had between them from 92 to 100 children, of whom only 6 became affected, and these six occurred exclusively in pedigrees containing some affected females as well as males.

Condition of the Parents of Affected Females.

Both parents normal: 77, 78, 49, 95, 99, 105, 106, 112, 116, 119, 120, 141, 123. Father affected, 50 and 93 IV, 1; mother affected, 93 and 49.
Condition of the Children of Affected Females.

All the sons of an affected mother were affected in Cases 111, 138, and 1.

Some of the sons of an affected mother escaped in Cases 108, 142, 50, and 52.

Proportion of affected to normal in the total surviving issue of an affected mother and normal father, shown in 12 sibships of the following 9 pedigrees: 93, 49, 108, 116, 140, 50, 52, 51, 176.

Proportion of affected to normal in the total surviving issue of normal : and mother normal but carrying the disease, shown in 38 sibships following 19 pedigrees: 92, 102, 105, 45, 115, 46, 120, 130, 144, 47, 54, 48, 155, 168, 171, 174, 167.

Effect of Early Deaths upon the Proportion of the Survivors who suffer, in Childships originally consisting of Seven Children or more.

In the following 10 cases there were 12 sibships of 7 or more, with few, if any, early deaths, and one third of the individuals suffered from the disease.

Total births, 102, of whom 33 males and 3 females got the disease (36 in all): 110, 117, 130, 138, 142, 143, 47, 147, 158, 174.

In the following 16 cases, containing 18 sibships of 7 or more, a number of the children died early, and one half of the survivors suffered from the disease: 92, 93, 45, 115, 119, 120, 121, 140, 141, 144, 148, 154, 51, 162, 170, 167.

These 18 childships produced 195 children, of whom 96 died in infancy; of the 99 survivors 30 males and 7 females got the disease (46 in all).

Longevity of those Affected.

The following cases contain affected persons who lived to be 50 or more. Cases in which the disease set in after 40 in males are excluded. All cases in females who lived to 50 are counted, at whatever age the disease set in, as it has been supposed that the disease is especially likely to occur during the climacteric in women: 90, 103, 49, 111, 45, 116, 120, 140, 142, 50, 52, 154, 51, 161, 166, 168, 172, 176.

Early and Late Age of Onset.

(i) Cases in which the disease occurred early in life, i.e. from earliest childhood up to 13 years old: 92, 96, 95, 110, 116, 119, 141, 145, 50, 52, 149, 151, 153, 156, 171, 177, 178, 146; also two atypical cases, 181, 181b.

(ii) Cases in which the disease set in late, i.e. from 30 years old upwards.

A. Pedigrees showing examples of late onset in males:

(1) In the following only males were affected: 89, 90, 103, 107, 45, 46, 120, 136, 143, 47, 147, 168, 175.

(2) The following contained cases in both males and females: 93, 49, 111, 112, 141.

B. Pedigrees showing late onset in females: 90, 49, 95, 98, 106, 108, 110, 111, 113, 140, 141, 153, 51, 162, 176; also atypical cases, 181a, 181c.
Other affections, chiefly of the nervous system, in the stocks containing Leber's disease:—Epilepsy, Case 77, 98, 105, 120, 122, 138, 148; insanity or idiocy, 95, 173; mental defect, 45, in the only female (VI, 2) whose female ascendants went back to male with Leber's disease; severe hysteria, 115; diabetes, 95, 103; phthisis prevalent in 102, 98, 103; congenital colour-blindness, 106, 117, 147.

The affected females are to be found in the following cases: 77, 78, 80, 93, 49, 95, 99, 105, 106, 108, 110, 111, 112, 113, 116, 117, 119, 138, 140, 141, 142, 145, 50, 52, 123, 131, 153, 159, 51, 162, 165, 169, 176, 177, 178, 53; and in the following atypical cases: 181, 181a, 181b, 181c.

APPENDIX VII.

Nystagmus.

(a) Albinism.

(1) Under "Hereditary Nystagmus," allusion is made at p. cxxii et seq. of the Lecture to Albinism. With the exception of certain cases of nystagmus that I regard as a form of partial albinism and have already written about as such, the problem of albinism in general was not discussed in the Lecture since it forms the subject of a long and elaborate memoir planned and initiated some five years ago by Professor Karl Pearson, who has taken by far the largest share in its execution, although in certain sections Mr. C. H. Usher and I have been mainly responsible. This memoir is now very near completion, and may appear either a little before or a little after the present writing; although, therefore, any discussion of albinism as a whole in my Lecture would have been out of place, there is no impropriety in using a few of the pedigrees (somewhat condensed to save space) that will appear in the memoir to illustrate certain clinical features. Of course no general conclusions are to be drawn from these samples.

Fig. 53 illustrates the condition of incomplete albinism affecting chiefly the eyes, and is described at p. cxxiii of the Lecture. It is to be taken in
conjunction with Figs. 54 and 55, which, having been published, are inserted in the Lecture (p. exxiv). (Fig. 53 is Fig. 205 in the forthcoming memoir upon albinism in man above mentioned, and is from a case sent by Mr. Jameson Evans, of Birmingham.)

Fig. 56. General albinism with both discontinuous and continuous inheritance, the latter occurring where an albinotic woman marrying a normal first cousin of the same stock has albinotic children. Bisexual twins occur twice, and in one of them one member is an albino, the other normal. (Forthcoming memoir upon albinism in man, Fig. 27, Mr. C. H. Usher.)

Fig. 57. Discontinuous and continuous descent of albinism. No consanguinity. (Ibid., Fig. 28, Mr. C. H. Usher.)

Fig. 58. Continuous and discontinuous descent. No consanguinity. A normal man of the albinotic stock marries twice, both wives being

![Diagram](image)

from unrelated stocks; he has albinotic children by one wife, all normal children by the other. (Ibid., Fig. 226, Dr. Schoute, Amsterdam).

Fig. 59. Discontinuous descent. Albinism and deaf-mutism in different members of same sibship. No history of deaf-mutism in any ascendants on either parental side (father’s side not shown but inquiry made). No consanguinity. (Ibid., Fig. 211, Mr. Wherry.)

Fig. 60. Marriage between two albinotic stocks that are believed to be unrelated, and between one of them and a third stock containing insanity and epilepsy, but no albinism. (Ibid., Fig. 30, Mr. C. H. Usher.)
(2) The cases upon which the remarks on Hereditary Nystagmus in relation to Albinism at p. cx xv of the text of the lecture are based are as follows:

(1) Lloyd Owen, O.R., i, p. 239 (1882), Fig. 54 in present Lecture. (Fig. 449 in forthcoming Memoir on Albinism.)

(2) Lawford, St. Thomas's Hospital Reports, xvii, p. 166. Case 1. Fig. 187 in present Lecture. (Fig. 68 in Memoir on Albinism.)

(3) E. Nettleship, B.L.O.H., ii, p. 366 (1887). Fig. 55 in Lecture. (Fig. 410 in Memoir on Albinism.)

(4) McGillivray, O.R., xiv, p. 260 (1895). Case B. Gorrie family. Fig. 188 in present Lecture. (Fig. 448 in Memoir on Albinism.)

(5) E. Nettleship (1897). Fig. 186 in present Lecture. (Fig. 402 Memoir as above.)

(6) Caspar, C.f.A., 1908, p. 199. Fig. 182 below.

In III are four childships, the first containing 4 males all affected and 4 females all free; second, 2 males both affected, 5 females free; third, 5 females free; fourth, 1 male and 5 females all free. Dr. Caspar has been unable to send any further information (February, 1909).
(7) E. N., unpublished, St. Thomas's Hospital out-patient, October 24th. 1881 (Simpson). (Fig. 183.)

III, 1, aet. 3½ years, fair complexion, choroid pale around O.D., but fundus normal, marked lateral nystagmus; healthy; did not have ophthalmia neonatorum; is thought by mother to be "short-sighted." Refraction not recorded. III, 2 treated for ophthalmia neonatorum at St. Thomas's; saw well, and had steady eyes; died of convulsions; no note of sex or colour of hair and eyes. II, 6 mother, aet. 22 years, normal eyes, some H., colour of hair and eyes not noted. Her 3 brothers and II, 1, a son of a sibling (I, 1) of her mother (I, 2) had moving eyes like III, 1; her sisters (II, 2), number not given, had steady eyes.

(8) Jameson Evans, Fig. 53 above described.

(9) Dr. R. J. Smyth and E. Nettleship (1907). (Fig. 184.)

I, 1 and 2 lived to 70 and 65, not consanguineous; II, 1 operated by E. N. for glaucoma when 35; II, 2 died at 35; II, 3, 4 and 5 steady eyes; II, 6, 7 and 8 aet. 35, 32, and 30 years, nystagmus and more or less A., with defective vision ($\frac{6}{27}$ to $\frac{6}{12}$ corrected); irides of these 3 grey with pigment at sphincter circle; fundus not suggestive of albinism in any; II, 7 has reddish-brown hair which was lighter formerly. There are about 20 children in III, all said to have good sight and steady eyes (not shown in Figure).

The above 9 pedigrees contain 43 cases of nystagmus, 40 males, 3 females; and in the same childships about 65 to 70 normals, viz., 20 males and 45 to 50 females, total 109 to 114.

(b) Day-blindness with Colour-blindness.

The family cases known to me are the following:

(1) Nettleship, St. Thomas's Hospital Reports, x, 1880. (Family 1, Foster.) Quoted in text of Lecture, p. cxxix, with Fig. 61.
(2) Nettleship, *ibid.* (Families 3 and 4, Pike, Channon.) *Ibid.*, with Fig. 62.

(3) Nettleship, *ibid.* (Family 6, Gould.) (Fig. 185.)

I, 1 and 2 brothers, reported to have seen well. III, 1 who was liable to melancholic attacks, but had good sight, married II, 2, her first cousin once removed, who also saw well, and had 9 children, of whom 8 were living at date of record, IV, 5 having died; IV, 1, at. 34 years, not seen said to be affected in same way as her two brothers, IV, 6, at. 25 years, and IV, 7, at. 23 years; both of these, with clear media, saw better in dull than in bright light, and were afraid of summer days and preferred to hold the head down to shade the eyes, in spite of having much contracted Fs. and retinitis pigmentosa; both colour-blind. They had nystagmus, and their sight, according to their own and their mother's account, had been in exactly the same state since early childhood. All the others said to have very good sight.

(4) Nettleship, *R.L.O.H.*, xi, p. 373 (Case 27), 1887. (Mr. Waren Tay's case.)

In a childship of 5, the first-born male, the other 4 female, the 2 elder girls (Nos. 2 and 3 born) totally colour-blind, day-blind, and amblyopic, V. with H. 3 D. corrected about $\frac{3}{2}$. Parents first cousins, but exactly how is not stated.


In a childship of 7, 1 of the 3 males and 1 of the 4 females affected; quite typically. H. 3 to 4 D. V. corrected $\frac{3}{4}$. No consanguinity.


In a childship of 4, Nos. 1 and 2, both female, typically affected; No. 3 female and No. 4 male, normal. Parents first cousins, but kind of cousinship not noted.


Parents normal, and not related by blood. Patient is second born of 8, all living, at. from 24 to 2 years. His case is typical. The first born, male, 24, said to be similarly affected, and the youngest, female, 2,
thought to have same defect. Nos. 3 and 6 (males) and 4, 5 and 6 (females) all good sight. Interval of 8 years between No. 2 (patient, act. 22 years) and No. 3 (act. 14 years).


Nystagmus with total colour-blindness and V. about 1 2 in a brother and sister of 14 and 12. Not albinotic. Very incompletely reported.

(9) Netleship and Holmes Spicer, T.O.S., xxviii, p. 83 (1908), with Fig. 63 in Lecture, p. cxxxi.

(10) Mr. Holmes Spicer and Dr. Souter, unpublished (1908), with Fig. 64, ibid.

The above 10 pedigrees contain 34 cases of this day-blindness with colour-blindness: 18 males, 15 females, and 1 sex not recorded. The same childships contain at least 45 (probably more) normals: 17 male, 22 female, and 6 or more sex unrecorded, total about 80 to 85.

The total of 84 cases mentioned at p. cxxix of the lecture is made up of my own and Grunert’s series, including some single cases of mine not given above, but useful in relation to sex prevalence, and three others published since the appearance of Grunert’s paper by Wehrli, 1903 (Abstract in Nagel’s Jahresbericht, xxxix, p. 92); Bjerrum, 1904 (Abstracted, ibid., xxxvi, p. 105, and again p. 205); Ronne, 1906 (abstracted—ibid., xxxvii, p. 78, and original reproduced in full in K.M.f.A. (Beilage, heft), xlv, p. 193. In Bjerrum’s case, two brothers of the (male) patient were also affected.

(c) Nystagmus, Unclassed.

The references to the fourteen unclassed cases of hereditary or family nystagmus, spoken of at p. cxxvii of the lecture, are given in chronological order below.

Published—
1893.* Bolland, Rec. d’Opth., p. 569. This is an abstract by Rolland from the Echo Médicale, which appears to have copied from the original in the Limousin Médical of unspecified date.
1895.* Burton-Fanning (F. W.), The Lancet, ii, p. 1497.
1903.* Clarke (Ernest), The Ophthalmoscope, i, p. 86.
1903.* Sinclair (M. McIntyre), ibid., May 23rd.
1908.* Dudley (W. H.), A. of O., xxxvii, 565.

Unpublished—
1905. Case communicated by Dr. Angus MacGillivray (Dundee).
1906. Case communicated by Mr. Lawford (London).
1908. Case communicated by Dr. Vilhelm Magnus (Christiania).
APPENDIX VIII.

Cornea.

Reticular and Nodular Keratitis.
Description of Figs. 65-69.

Fig. 65. Holmes Spicer, T.O.S., xxiv, p. 42 (1904), and later information.

I, 1 believed to have had good eyes; I, 2 lived to 101, and is known to have had perfect sight to the end. II, 3, second wife of II, 2, and her brothers, II, 4 (number not recorded), said to have suffered in same way as III, 4 and his daughter. II, 2 and his first wife, II, 1, and her children, all had perfect eyes. III, 2, at. 65 years at record, probably normal; III, 3 probably affected, sight “peculiar” in same way as III, 4, and an opera glass was useless to her; III, 4 seen by author, at. 50 years, typical changes, eyes have been troublesome all his life; III, 5 had symptoms like those in III, 4, and on trying to enter the Navy failed to pass the sight test, he died at 30; III, 6 has never had any trouble with his eyes. IV, 1, only child, seen by author at 23, characteristic changes, no severe symptoms, and V. with slight M. As. corrected § and § in R. and L., and appears to have been same for many years.

Fig. 66. Freund, A.f.O., lvii, p. 377 (1904), and Wien. klin. Woch., xix, No. 5, 1906. Family 2 (Hermann).

All marked “+” were examined by the author. The only ones believed, or known, to have the family disease are II, 5, who died many years before the record; III, 1, who died at 23, and is said to have had “scrofulous inflammation of the eyes”; and IV, 2 and 3, at. 10 and 6 years at record, and definitely stated by the author to have been free from the disease at that time; though not starred they were probably
examined. The ages of the affected ones when seen were: II, 1 (author's Case 8) 61, II, 2 (Case 9) 56, III, 2 (Case 10) 26, III, 3 (Case 12) 39, III, 4 (Case 14) 38, IV, 1 (Case 13) 13, III, 5 (not seen, Case 15), 26. In II, 3 (Case 11), age not stated, the eye disease had existed more than twenty years. II, 4 not seen or described, but stated to be affected; was 46 at date of record. Nothing said about early deaths.

Fig. 67. Freund, *ibid.*, Family 1, Bienert.

In this genealogy the ages, unless otherwise stated, are as given in the author's earlier publication (A.J.O., lvii), and refer apparently to 1902, or sometimes perhaps rather earlier. The pedigree now presented in Fig. 67 is the result of collating the published ones of 1904 and 1906, and adding important new information that Dr. Freund has with the greatest courtesy supplied to me in reply to questions.

Dr. Freund's latest reply, dated June 14th, 1909, three days after the delivery of the lecture, gives the result of his examination of the eight children IV, 10 to 18; whilst in a letter of May 3rd he gave the present condition of the four siblings, V, 1 to 4, who were all normal seven years ago, whilst three of them now show the typical condition. I reproduce the names of all the members as given by Dr. Freund in order to facilitate reference if still further information should be forthcoming in future. I, 1 died young, had good eyes. I, 2, Wenzel Bienert, husband of I, 1, also died young, between 1860 and 1870; is reported to have had the family disease. II, 1, 2, 3, order of birth not recorded, died before Karl (II, 4); all three had bad eyes, the eldest being quite blind, no other details. II, 4, Karl Bienert (the elder), died in 1889, age not given; reported to have had the family disease; his place in

* Gen. VI, 1909, should be V. The same childship was examined in 1902 and again in 1909.—E. N.
the childhood not given. II, 6, aet. 84 years at date of first record, and still living (May, 1909); eyes affected for more than fifty years; the cornea are densely opaque and scarred, and it cannot now be proved that she has the family disease; her only child, Daniel, aet. 56 years (III, 14) has normal eyes. II, 5, Karoline Wolf, aet. 72 years and still living (May, 1909), has had the family disease all her life. III, 1, Karl Bienert (the younger), 63, has had the corneal disease thirty years. III, 2, Ferdinand Bienert (the elder), died at 36 in 1876; had the family disease and his sight was very bad. III, 3, Josef Bienert, 53, cornea clear but iris shows remains of fetal pupillary membrane. III, 4, Edward Bienert, died at 30 in 1879, believed to have had good sight. III, 5, Antoine Bartosch, 49, and III, 6, Johann Bienert, 48, both typically affected. III, 7, Wenzel Wolf, 50, affected, but sight still relatively good. III, 8, Ant. Wolf, 48, affected and sight very bad. III, 9, Karoline Beer, died at 43 between 1890 and 1900, was affected by the family disease, but is said to have still seen well. III, 10, Berta Jung, 45, affected and sight very bad. III, 11, Leopold Wollmann, 41, affected and sight very bad. III, 12, Matilda Rösler, 39, affected, but sight still good. III, 13, Marie Wolf, 17 (?) 37), affected, and sight very bad. III, 14, see II, 6. IV, 1, Emil Bienert, 39, affected. IV, 2, Fran Engelfeld, about one year younger than IV, 1, reported to be normal, as also her six children, but could not be seen (May, 1909). IV, 3, Karl Bienert (the third), examined at 13 (?) 1900), high myopia but no corneal disease. IV, 4, Karl Bienert (the fourth), 31, and his sister, IV, 5, Augusta, 29, both affected. IV, 6, Ferdinand Bienert (the younger), 26, moderately high myopia, no corneal changes. IV, 7 and 8 examined and normal; IV, 9 died at 24 nearly blind, but believed not to have had the “Bienert disease.” IV, 10, Hedwig Bartosch, 23, affected. IV, 11 to 18, eight children of III, 6, examined, June 1909; IV, 17, Max Bienert, 5, “already shows small, spotted, sub-epithelial opacity of both corneas; it extends to the periphery of the cornea, and the corneal surface is at present smooth; it is not altogether identical with the family disease.” The other seven, IV, 11, Marie, 20; 12, Hans, 19; 13, Eleonore, 17; 14, Margarete, 11; 15, Walter, 9; 16, Curt, 8; and 18, Gerda, 1 year, are normal. IV, 19 to 21, Karoline Jung, 26, Emma, 11, and their siblings, no information obtainable. V, 1 to 4, examined in 1902 and again in May, 1909: V, 1, Mathilde Bienert, 14, normal, and is still normal in May, 1909, aet. 21 years; V, 2, first seen at 10 with normal corneas; when re-examined at 17 (May 1st, 1909) characteristic changes in the cornea; the same is true of V, 3, normal when seen at 8, the same corneal changes at 15; and of V, 4, normal at 4 and characteristically diseased at 11. In the figure Gen. VI, 1909, should have been V.

Fig. 68. Doyne and Stephenson, The Ophthalmoscope, iii, 213 (1905).

I, 1, eyes had from youth, and towards end of life sight so bad that she had to be led about; died at 65. II, 2 seen at 48, with very advanced opacity of R., and less of L.; age of onset 39, or perhaps earlier. II, 4, a sister, now dead, said to have had the family disease.
III, 1, act. 24 years, eyes began to fail at about 16, but with interval of three years between R. and L.; now almost universal dense opacity: III, 2 began at 9, now 22, and as bad as III, 1; III, 4 disease began at 7 and has steadily got worse, and now, at 15, is nearly as bad as III, 2, and has much severe acne on face.*; III, 5 began at about 11, seen at 12; chief part of opacity showed much resemblance to "transverse calcareous film," and, as is common in that condition, showed numerous small, clear holes. No consanguinity.

Case 68a. A new case has been given to me this year by Mr. Herbert Fisher, but as there has not yet been an opportunity for examining all the available members of the family I withhold it; at least two sisters are affected, and probably two or three of their siblings.

Fig. 69. Folker, T.O.S., xxix, p. 42 (1909).
1, 1 now 92, history of first failure when about 50; about ten years later operated for cataract in both eyes; wife living, has had 13 or 14 children. 7 still living, and no miscarriages. II, 3, now 50, sight "always" been defective; 11 children, 8 living, 3 died under 2 years; II, 5, act. 46 years, sight "always" been defective; has 9 children. III, 1, act. 30 years, sight defective as long as she can remember, and apparently getting steadily worse after each confinement; has had 6 children in 8 years, one dying in infancy, 5 living; III, 2, act. 28 years, sight defective all his life, now V. 1/; III, 4, act. 21 years, sight defective as long as she can remember, now V. 1/., married, 1 child, act. 10 weeks; III, 5, act. 18 years, no definite history of commencement, but is getting worse, V. 1/; III, 9, act. 21 years, has never noticed any defect of sight, and has now 2/ in R., 3/ in L., but central area of each cornea shows 20 to 30 small scattered spots; III, 13, act. 12 years, no symptoms, and V. 3/ with each eye, but has a few small dots of corneal opacity like his brother.

A general review of the disease illustrated by these pedigrees leaves one in no doubt that it is often, if not always, progressive, that in an early stage sight may be so little affected that nothing short of careful

* Severe scar-leaving acne was observed by Marcus Gumm in one of his cases: T.O.S., xix, p. 97.
examination of the cornea can be taken as conclusive, and that practical
blindness may ensue from gradual extension of the area and increase
in the density of the opacity. Careful inquiry in many of the cases has
shown that there is no reason whatever for thinking that syphilis takes
any part in causing the disease.

The following is a list of the principal papers upon nodular and
reticular opacity of the cornea. * Most important. Some others may be
found at end of the article by Doyne and Stephenson.

*1890. Groenouw, A. of O., xix, p. 245.
1891. Chevallereau, France Médicale, May 2nd.
1892. Oliver (C. A.), Amer. Journ. of Ophth., p. 234; also given in
O.R., ii, p. 319 (same year).
*1899. Dimmer, ibid., ii.
xix, No. 5.
1904. Fehr, C.f.A., xxviii, January and June.
*1905. Doyne and Stephenson, The Ophthalmoscope, iii, p. 213.
*1908. Folker (H. H.), T.O.S., xxix, p. 42.

APPENDIX IX.
ABBREVIATIONS OF TITLES OF PERIODICAL PUBLICATIONS.
A.f.O.—Von Graefe's Archiv für Ophthalmologie.
R.L.O.H.—Royal London (Moorfields) Ophthalmic Hospital Reports.
T.O.S.—Transactions of the Ophthalmological Society of the United
Kingdom.
C.f.A.—Hirschberg's Centralblatt für praktische Augenheilkunde.
A. of O.—Knapp's Archives of Ophthalmology.
O.R.—Ophthalmic Review.
1. A spectroscopic test of colour vision.

By Dr. A. Maitland Ramsay.

Although there is considerable difference of opinion regarding the value and trustworthiness of the various methods of testing the colour sense, there is absolute agreement as to the desirability of using for the purpose, as far as possible, the pure colours of the natural spectrum. Their employment in this way in exact scientific experiment has been invariable, but the corresponding clinical application has always been attended by serious difficulties. Several years ago Dr. Thomas Reid showed me a diffraction grating, the dispersion power of which is greater than that of an ordinary prism, and it occurred to me that the use of a diffraction grating might overcome many of these obstacles. In carrying on the necessary experiments, and in working out the problem, I have been greatly assisted by Mr. Trotter, optician, Glasgow, and have to thank him for the skill and ingenuity he has shown in the construction of the apparatus.

The instrument which has been devised (Fig. 1) consists of a rectangular brass box, 12½ in. long, 3 in. broad, and 1½ in. deep. This is mounted on a double metal support resting on a wooden base, and is inclined at an
angle convenient for ordinary vision. At the upper end of the box is an eye-piece (I, Fig. 2) magnifying about ten times, in the focus of which are two diaphragms (A and A\textsuperscript{1}), with slits which permit only one thin line of colour to be visible in each. At the lower end light enters the instrument through two slits (B and B\textsuperscript{1}), which are protected from dust by a slip of ground glass, and the width of which can be altered by graduated adjusting screws (D and D\textsuperscript{1}). In this way the brilliancy of the colour in the spectrum band can be diminished or increased at will, the differences being measured by the numbers on the graduated screw-heads, which roughly indicate tenths of a millimetre of difference in the size of the slits. The interior of the box is carefully blackened, and in the middle is placed a diffraction grating (with 14,438 lines to the inch), on either side of which is a collimating lens. The result of the passage of a ray of light through the lower slits, the grating, and the lenses, is that two spectra of considerable dispersion are formed in the focus of the eye-piece, the one above the other, the light from the lower slit B\textsuperscript{1} controlling the colour seen through the upper diaphragm A\textsuperscript{1}, and vice vers\'a. Either or both of the spectra can be moved from side to side by moving the shutters containing the lower slits (B and B\textsuperscript{1}) by means of screws (not seen in the figures), one on each side underneath the lower end of the box, and thus any desired portion of the spectrum may be brought opposite the slits. The movements of the shutters are noted by the movements of the indices on the dials C and C\textsubscript{1}, which are roughly graduated in wave lengths, and on which are also marked the principal lines of the spectrum—lines C, D, E, and F. At the upper end of the box, on the left-hand side facing the eye-piece, is a handle (G) by means of which the diaphragm A\textsuperscript{1} can be displaced at will, and the whole spectrum rendered visible (Fig. 3). At the upper end of the box is a rotary diaphragm (II) perforated by three minute circular apertures, which can be turned into position in place of
A SPECTROSCOPIC TEST OF COLOUR VISION.

Figs. 1-4.
A SPECTROSCOPIC TEST OF COLOUR VISION.

When seen through the eye-piece the diameters of the circles are equal to 1, 2, and 4 mm. respectively; and this arrangement, which permits only a single circular spot of colour to be seen (Fig. 4), enables the correct naming of colours to be tested as well as the correct matching. If a double-image prism be adjusted over the eye-piece, this spot of colour is doubled, and by the rotation of the prism the second (or excentric) colour-spot can be made to revolve round the stationary central one, like a satellite round a planet.

The instrument is convenient in size and so light that it can be easily carried or moved about. It is simple in construction, and has no part liable to get out of order or to be injured by use or by ordinary change of temperature, so that the apparatus could be readily used on board modern steamships, or in railway works—wherever, indeed, an electric current can be obtained. The lamp can be connected with the electric main by an ordinary plug, care being, of course, taken that the lamp is of a voltage suitable for the strength of the current.

The apparatus is used in the following way: The examiner, having, by means of the right-hand screw underneath the lower end of the box (not shown in any of the figures), brought any portion of the spectrum he pleases opposite the lower slit, as a test, the patient is seated in front of the eye-piece, and asked to look through the lens and to turn the left-hand screw underneath till the colour seen through the upper slit is exactly the same as that visible through the lower. The examiner can tell what the patient is doing by watching the movements of the index on the left-hand dial; and, if he write down the registered index figures on both dials, he can keep a written record of the examination. To begin with, the whole upper spectrum may be exposed to view by turning the handle, G; and, indeed, in the early models of the instrument this was always so, the diaphragm A1 being added on the suggestion of Mr. Edridge Green.

The whole upper spectrum being exposed, the person
whose colour-sense is being tested is asked to bring the corresponding portion of it immediately above that shown in the lower slit. When the diaphragm is in position the patient can be asked not only to match the colour seen in the lower slit, but also to turn the left-hand screw underneath, and to say at once whenever he detects the slightest change in colour, in either direction. The test colours can be varied at will, and each eye ought to be tried separately.

Lastly, the upper slit diaphragm should be again displaced, and the rotary one brought into use. A spot of colour 1, 2, or 4 millimetres across is now in the line of central vision, and can be varied as the examiner pleases, the patient being asked to name the colours just as would be done if the lantern test were being employed. The instrument may thus be used, with very trustworthy results, to detect colour scotoma in toxic amblyopia, the patient being unable to recognise the colour (mostly red or green) in the central spot. If the double-image prism be placed over the eye-piece the test becomes still more delicate; for the patient, while he cannot see the central stationary spot, can, by peripheral vision, see the excentric one as it revolves when the prism is rotated. By diminishing the amount of light and noting the reading of the graduated screw-heads, it is possible to form an approximate estimate of the acuity of the patient's colour light-perception—for example, how quickly and readily the patient could recognise the colour of a railway signal, or of a ship's light in foggy weather.

(October 15th, 1908.)

Mr. C. D. Marshall said he thought it was generally recognised that the spectroscope was the final test for defects of colour vision, and was the one which gave the most accurate results. He wished to congratulate Dr. Maitland Ramsay on his most ingenious apparatus, which combined most of the essential points. Dr. Edridge Green was still experimenting, with the help of a well-known optician, with a view of perfecting an apparatus
with which one would be able to test candidates in that manner. He had only just seen Dr. Ramsay's apparatus, but it seemed one of the best contrivances yet evolved in connection with that important subject.

2. A case of unilateral ptosis treated by the Motais operation very slightly modified.

By Sydney Stephenson.

The patient, a little girl then at 2\frac{1}{2} years, was first seen at the Evelina Hospital, London, on September 3rd, 1907. She was affected with incomplete ptosis of the left eye, as shown by the photograph (Fig. 5). Movements of eyeball good. No synkinesis noted. No associated deformities.

Patient youngest of nine children (four males and five females). No heredity.

On September 24th, 1907, under chloroform, left superior rectus divided into two parts, the outer of which was stitched into the upper lid between the skin and orbicularis muscle, on the one hand, and the tarsus, on the other. Sutures removed seven days after operation.

Present condition of patient, upwards of a year after operation, is shown in the accompanying photograph (Fig. 6). No squint is present. Child can close eye naturally.

(October 15th, 1908.)

The President said that the result with both eyes open was very satisfactory. A hypothetical objection to this operation existed in as far as the normal upward rotation of the cornea during sleep, due to the action of the superior rectus, would here presumably be accompanied by elevation of the upper lid. This non-closure of the lids was, of course, a difficulty in all operations for ptosis, but in Motais' operation one would expect it to be even more
pronounced than usual. He should be glad to know whether or not this objection applied to the present case.

Mr. E. Treacher Collins asked whether there was much hyperphoria produced by the operation.

Mr. Stephenson, in reply, said the eyelids of the eye which had been operated upon were not entirely closed during sleep, but he was not aware that the child had taken any harm from that. So far as he knew, after any successful operation for ptosis the eyelids were not quite closed, whether it was done by Motais' operation or not. In answer to Mr. Collins, there was some squint for some weeks after the operation, but that had now disappeared, as members would have assured themselves.

3. Microscopic section from a case of tuberculous irido-cyclitis in a man, æt. 75 years.

By A. Ogilvy.

Patient, first seen at Bristol Royal Infirmary in November, 1906, complained of severe pain in left eye, radiating over forehead (on that side) and vertex.

Pain had been present for three weeks, during which time vision was lost. When first seen at Bristol Royal Infirmary eye was blind.

Patient had always had good health, and up to three weeks before had never had any trouble with either eye; eye first became inflamed during an asthmatic attack, which lasted a week.

Eye was inflamed; anterior chamber full of greenish-yellow material looking like pus; iris much retracted.

During observation tension alternated, − and +. Pain not very marked unless when tension was high.

After three weeks eye was enucleated for pain and glaucoma.

Sections were made by Dr. J. Walker Hall. Drawings
were shown illustrating the tuberculous nature of the inflammation. (Card specimen. October 15th, 1908.)

The President asked whether in Dr. Ogilvy's view the eye was the primary seat of tubercle in this patient. It was certainly most unusual to meet with tubercle developing in the eye at that age for the first time.

Mr. Parsons asked whether the other eye was all right.

Dr. Ogilvy, in reply, said he thought it was the primary seat. The fellow eye was normal.


By J. B. Lawford.

Geo. P,—, aet. 40 years, shepherd, admitted to St. Thomas's Hospital October 1st, 1908. His left eye was injured three years ago by a chestnut which was thrown at him, and has since been defective. Four months ago his right eye was injured; he was pulling a nail from a piece of wood, and "jabbed" his thumb between the eyeball and the upper orbital margin. The sight of this eye failed immediately; one week later he "could not see across a field"; vision subsequently improved.

On admission; V. = R. $\frac{6}{12}$ and 4 J., not improved by lenses. L. $\frac{6}{18}$ and 14 J., not improved by lenses.

R. Field of vision shows a small defect at the upper nasal periphery.

L. Field of full extent.

Ophthalmoscopic examination.—R. eye (recent injury): Choroido-retinal changes in region of O.D. and macula. A horizontal streak of choroidal atrophy extends from O.D. outwards beyond the macula. It is bordered by pigment accumulation above and below, and immediately below it is some blood extravasation in the retina. Above this streak is a smaller streak of choroidal atrophy, and
around the O.D. are several small spots and patches of partial atrophy of choroid, with some pigment accumulation in connection with most of them.

L. eye (old injury): Occupying the region of the disc and yellow spot are areas of choroido-retinal atrophy, with abundant pigmentation at their margins. There is one narrow streak of atrophy (rupture of choroid) between the macula and the disc. The extent of the choroidal disease is larger than in the right eye, and all the lesions appear old and quiescent.

The patient is a strong, healthy man, and describes the blow upon each eye as very trifling. His ocular tissues appear to be unusually vulnerable, and he is an example of a somewhat rare condition, viz., permanent damage to the sight of both eyes by similar, but not simultaneous, injuries. *(Card specimen. October 15th, 1908.)*

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5. *A case of oil-globules in the anterior chamber.*

By A. C. Hudson.

Ernest M—, aet. 27 years, was admitted to the Royal London Ophthalmic Hospital on May 29th, 1908, under Mr. Devereux Marshall, having been struck in the right eye a few hours previously by a rivet head the size of a farthing. Castor-oil had been instilled into the conjunctival sac immediately after the accident. The patient was suffering from a central, horizontal, perforating wound of the cornea, about 5 mm. in length, to which the iris was adherent. Atropine drops were instilled, the eye was bandaged, and the patient was put to bed. On the following morning the anterior chamber was of normal depth. The iris had become free from the wound, but on its surface at each extremity of the central horizontal meridian were several globular bodies presenting an
appearance suggestive of frog-spawn. These were surmised to be droplets of oil, which had made their way into the anterior chamber from the conjunctival sac. The iris in contact with the inner group, one of the members of which had a minute pigmented body on its surface, presented evident signs of inflammation. The globules retained their fixed positions until fifteen days after the accident, when they began to become free, and to coalesce into larger droplets, which floated to the top of the anterior chamber when the patient held his head erect. Three globules can now be seen floating freely in the aqueous humour in accordance with the laws of gravity. The small pigmented body which was originally observed on the surface of one of the globules now rests on the upper and inner part of the iris. The eye has been somewhat slow to quiet, but is now free from signs of inflammation, and the vision with \(-75\) D. cyl. 73° in = \(\frac{6}{5}\) and J 1. The tension of the eye is normal, and has never presented any remarkable features.

(October 15th, 1908.)

6. A case of reticular keratitis.

By A. C. Hudson.

Isabella H—, aet. 56 years, in-patient at the Royal London Ophthalmic Hospital under Mr. Stanford Morton, October 8th, 1908.

The patient’s grandmothers were sisters, her father and mother were first cousins. She has had three male and eight female children. She knows of no eye disease in her family, except in the case of her mother, who used to say that she had had a spider’s web over her left eye from the age of fifty.

The patient had good health until one day in February, 1900, when she suddenly lost consciousness. On recovery
after a few moments she became aware of weakness in the right arm and leg, which, however, passed off in about six weeks, leaving a sensation of numbness in the right leg without pain or loss of sensation, and an occasional difficulty in articulation; both symptoms have persisted. The near vision in each eye was noticed to be defective immediately after the fit, but subsequently improved. When the patient attended as an out-patient at the Royal London Ophthalmic Hospital in July, 1900, she was noted to have a condition of both corneae similar to that which is now present. R.V. = $\frac{6}{6}$; L.V. = $\frac{6}{6}$; R. and L., T +. There was a defect in the right upper quadrant of each visual field. Dr. James Taylor diagnosed a lesion in either the occipital lobe or the posterior part of the internal capsule of the left side. During the last five months there has been a gradually progressing failure of vision for all purposes, without pain until two months ago since when there has been some pain. The right eye was very painful for a few days a fortnight ago, owing to the formation of a small ulcer at the lower and outer corneal margin.

The patient is healthy and robust. The pulse is rather high tension, and there is some hypertrophy of the left ventricle. The urine is healthy.

The central two thirds of each cornea is occupied by a grey mycelium-like striation, the members of which branch and occasionally anastomose. The lines are of varying breadth, the broadest presenting a somewhat craggy outline. They appear to lie for the most part nearer the anterior than the posterior surface of the cornea, but occupy different levels, inasmuch as they may be seen at some points to cross one another on different planes; occasionally there is developed in the course of one of the lines a fissure-like formation involving a considerable thickness of the cornea. Under oblique illumination the lines have the apparent consistence of half-melted snow; to transmitted light they are dark. In the left eye there are to be seen, in addition to the
PLATE I.

Fig. 1 illustrates Mr. A. C. Hudson's case of Reticular Keratitis (p. 11).

Fig. 2 illustrates Mr. R. R. James' case of Linear Opacity of Cornea, following Birth Injury (p. 33).
above appearances, throughout the central part of the cornea innumerable intensely fine wavy grey lines, conforming in many places to an irregularly parallel arrangement, so as to produce an appearance somewhat suggestive of a very fine cross grain in wood; this appearance is also present to a much less extent in the right eye. In the right eye there is, apparently just beneath the corneal epithelium, a central, very faint, obliquely disposed brown streak, which fades imperceptibly into a surrounding diffuse mottled grey opacity. In the left eye there is some softly outlined, faintly mottled grey opacity near the anterior surface of the centre of the cornea, and some more clearly defined, central grey opacity near the posterior surface. The peripheral zone of each cornea is quite free from opacity, except for the presence in the right eye, just inside the lower and outer corneal margin, of a small grey, slightly depressed ulcer-scar, in close relation to which lies the attenuated extremity of one of the main grey lines (Pl. I, fig. 1). The sensation of both corneas is unimpaired. The vision of each eye can be improved to $\frac{6}{50}$ with +2 D.s. The right optic disc, seen with difficulty, presents no appearance of pathological cupping; the left disc cannot be seen. The tension in each eye is full normal. Each visual field for white shows a defect in the right upper quadrant, which is almost complete in the case of the left eye, but rather less extensive in the case of the right.

The first description of cases of this kind appears to have been given by Treacher Collins and by Haab in 1899 (October 15th, 1908.)

Mr. Herbert Fisher asked whether the patient had hemianæsthesia as well as hemiplegia.

Mr. Hudson said he could not reply definitely to Mr. Fisher's question. The patient said the right leg felt numb and rather heavy, but there was no loss of sensation, and there had been no pain.
7. A case of dipterous larva in the anterior chamber.

By F. G. Thomas and J. Herbert Parsons.

We venture to bring this case to your notice, since we believe it to be one of extremely rare parasitic intra-ocular infection.

W. E—, male, æt. 2 years and 9 months, was sent to one of us (F. G. T—) on March 18th, 1908, by Dr. Vincent Morgan, of Narberth, Pembrokeshire.

History.—For some months previously the parents had noticed what they thought a laughable new habit in the boy; he would start as if he saw something, and make a movement with his hand as if to brush it away from before the left eye.

Three and a half weeks before, his mother thought that "the middle of the left eye looked somehow different," and the next day it became somewhat red, and photophobia with lacrimation developed. Some days the child was very fretful, holding his hand to the eye as if in pain, but this was not so always. He would not allow the eye to be opened.

There was no history of any injury or of any affection of the eyes. There had never been any discharge from the nose, and his general health was remarkably good. I made careful inquiry as to any symptoms suggestive of intestinal worms, but there were none, except that the mother said he had been picking his nose a great deal lately.

On examination the boy was well nourished and healthy looking. He did not appear to be in any pain, but kept the left eye constantly closed. He resisted any attempt to open the eye, which on pressure appeared to be tender.

A glimpse showed that an unusual condition was present, so I administered an anaesthetic, and was able to examine the eye at leisure. There was a slight degree of ciliary injection; the cornea was perfectly clear, the
anterior chamber somewhat deepened, and the aqueous faintly turbid.

The iris was altered in colour from blue to a dull, dirty grey, with the pattern rather fogged. The pupil, irregularly quadrilateral, was firmly bound down to the lens by pigmented lymph, which was also deposited on the anterior capsule.

Lying on the iris was what I then took to be a small round worm. The head was lying at the periphery of the iris in the lower and outer quadrant, while its tail disappeared in the angle of the chamber in its upper and inner quadrant. The body, of a greyish-yellow colour, lying curved below the pupil between these two points, measured, as far as I could estimate it, 12 or 13 mm. in length with a thickness at the middle of about 2 mm.

It consisted of eight clearly-defined segments, including the head. The under-surface of the latter, where in contact with the iris, appeared to be pigmented and as if partially embedded in the iris tissue.

With a binocular loupe and oblique illumination a very delicate network of grey material, like a cobweb, could be made out covering the parasite, and reflected on to the surface of the iris. No movements could be observed.

With the ophthalmoscope no reflex could be obtained through the occluded pupil. The tension of the eye was about – 1. The right eye was in all respects healthy. I made a drawing of the left anterior chamber whilst the patient was under the anaesthetic, representing as faithfully as I was able the appearance and position of the parasite.

Enucleation was performed the same day, the bulbar conjunctiva being found firmly adherent to the globe.

As I was under the impression that the parasite was a small intestinal worm, I gave the patient powders containing calomel, scammony and santonin, and instructed the mother to carefully search the stools each morning. This was done, with no result.
The patient made the usual uneventful recovery, and has remained perfectly well up to the present time.

The eye, hardened in 10 per cent. formalin, was sent to Mr. Parsons, for whose kindness in undertaking its examination and for the pathological portion of this paper I wish to express my sincere thanks.

Sections of the eye, with the parasite in situ, and a separate preparation containing its head, were sent to Dr. Shipley, of Cambridge, who kindly undertook to examine them. His report is as follows:

"I examined the head of your fly, and came to the conclusion that it was the maggot of a blow-fly."

Subsequently he added:

"The larvae of flies are very difficult to differentiate. My opinion is that the one in question is probably the larva of the blow-fly, but it may be it is the larva of one of the species of Sarcophaga. Both Sarcophaga carnaria and Sarcophaga magnifica larvae are found in the nasal cavities, and in other channels leading from the outside into man. The former has been found on the conjunctiva. They are not uncommon in ulcers, especially in Russia."

Poitchiuski reports an epidemic which occasioned much disease and death at Mohilew in Russia, due to infection of the inhabitants by the larvae of Sarcophaga magnifica.

We are indebted to Dr. Gordon-Hewitt, of the Department of Economic Zoology, Manchester University, for some notes on the varieties of Diptera, whose larva may be the one in question, and for suggestions as to its mode of entry.

There are several species of flies that lay their eggs in the nostrils of man, and occasionally in the eyes if there is a purulent discharge. They are the blow-fly (Calliphora erythrocephala [Meijen]) and Lucilia caesar (Linneus), of which the former would be the more likely in this case.

Some species are viviparous, such as the Sarcophajidae,
and deposit living larvae, the eggs having hatched in the vagina of the fly.

By the kindness of Dr. Gordon-Hewitt I am able to show a specimen of *Sarcophaga carnaria* in alcohol, with a full batch of recently-deposited larvae.

These larvae may be deposited like the ova in the nasal passages in suppurative conditions, in the roof of the mouth, in the intestines, or any other passages opening outwardly in man.

Dr. Gordon-Hewitt states, however, that he has looked through all his literature on the occurrence of Dipterous larvae in man, but failed to find a single case comparable to the one we are bringing to your notice. By what path the larva found its way into the anterior chamber is difficult to understand. The following possibilities are suggested:

(1) The larva may have been deposited in the nostril, and it may have worked its way through the nasal duct to the lacrimal artery, from which it could travel to the interior of the eye by way of the central artery of the retina, or of one of the ciliary arteries.

(2) Having been deposited as before, it may have worked through the sclerotic from the lacrimal region, or, if deposited in the conjunctival sac, the same mode of entry might be possible.

The second conjecture is less likely on account of the nature of the sclerotic tissue, since the larva, finding itself in the orbit, would not be likely to select this route in the presence of less resistant and more satisfying substances. Finding itself, however, by accident in a blood-vessel, such as the lacrimal artery, it might work its way along the vessel to an artery entering the eyeball.

It is unlikely that an ovum could be carried by the blood-stream to the interior of the eye, since neither that of the blow-fly nor the *Sarcophaga carnaria* measures less than 1 mm. in length, and is devoid of the activity of the larva.

*Sarcophaga magnifica* is unlikely to be the one under vol. xxix.
consideration, as, though a European, it is not a British species. The full-grown larva of *Sarcophaga carnaria* measures about 16 mm., so that this, if of that species, would not be full-grown.

The possibility of this being the larva of another species of Diptera must not be overlooked, since two cases of infection of the eye of a child by the larva of *Hypoderma lineata*, or the warble fly of the ox, are on record.

In Neumann's *Parasitic Diseases of Domesticated Animals* is mentioned a case in which van Setten, a veterinary surgeon at Onderdenham, Holland, extracted from the anterior chamber of a horse a larva, which was subsequently identified as that of *Hypoderma bovis*.

The essential characteristic of the *Oestridae* or Hypoderma is the necessary parasitism of their larvæ. The femalé lays her eggs on the bodies of Mammalia in order to be hatched. The larva is found most frequently in the subcutaneous tissues, but also may invade the frontal sinuses, or pharynx, the stomach or intestines. At the end of its first stage it is about 13 mm. long and about 2·2 mm. thick. It is white, except at the mouth and posterior stigmata, where it is brown.

By the kindness of Mr. Goodall, veterinary surgeon, of Christchurch, Hampshire, I am able to show a specimen of a newly hatched larva of Hypoderma. In this, at the lines of demarcation of the different segments, are a number of spines.

Mr. Goodall states that a larva he recently removed from the skin of a horse was just the shape of the drawing I sent to him, whilst Neumann describes the Hypoderma of the horse as being smaller than that of the ox, the segments very marked and destitute of spines, and the mouth unarmed.

*Pathological examination.*—The eye was fixed in formol. Cornea hazy. A.C. deep. Iris discoloured; pupil displaced upwards and irregular. There is an elongated cylindrical yellow body lying across the anterior chamber in a direction from up and in to down and out.
The eye was divided, after freezing, by an oblique section from up and out to down and in, i.e., at right angles to the foreign body. The A.C. was found to contain exudate in front of the iris, tying down the cylindrical mass to it. The lens was somewhat shrunken. The vitreous was shrunken and contained a coagulum. The retina showed umbrella-shaped detachment with a sub-retinal coagulum. The choroid was in situ.

Microscopical examination.—We are indebted to Mr. G. Coats for kindly preparing the sections for microscopical examination.

The cornea shows some infiltration at the periphery. There are leucocytes adherent to Descemet’s membrane, entangled in the coagulum which pervades the A.C.

A.C. is deep, and contains homogeneous coagulum with multitudes of leucocytes. The angles are widely open, but are densely occupied by leucocytes, which also infiltrate the tissues around the canal of Schlemm. Lying upon the surface of the iris is a circular body bounded by a hyaline membrane which is clearly an animal parasite, seen in transverse section.

The iris shows very intense iritis. It is oedematous, and packed with leucocytes, which are aggregated in places into nodules. The iris is also covered with leucocytes, which surround the parasite. Aggregations of leucocytes are also present on the back of the iris, and over the surface of the ciliary processes.

The ciliary body is densely infiltrated with leucocytes, especially in the inner part, including the processes. It is covered by a fibrinous coagulum, which extends round the lens and contains leucocytes.

The lens is distorted, and shows cataractous changes in the posterior part close to the posterior capsule.

The retina is detached and infiltrated with leucocytes. The sub-retinal coagulum shows the usual characteristics.

The choroid is in situ, and very little changed from normal. There is comparatively little infiltration.

One section showed the head of the parasite very
beautifully. It has unfortunately been lost in transmission through the post. Mr. Shipley was, however, able to examine this and the other sections.

Remarks.—Three cases only of Dipterous larvae within the eye have previously been recorded, and in each case the parasite was in the anterior chamber. Animals appear to be as little subject to the disease as men. On the other hand, irritation caused by the presence of Dipterous larvae in the conjunctival sac is not an uncommon complaint in certain districts, so-called *myiasis* being well known in parts of Russia, and in Mexico where it is known as *mal de ojo*.

In all four cases of intra-ocular larvae children were affected, the ages being 9, 5, 5½ and 23/4 years. In the first two cases the larva is definitely stated to have been that of the *Hypoderma bovis*. In Ewetzky's case the worm showed signs of imperfect development, probably due to its unnatural environment, and Prof. von Kennel was unable to determine the species precisely.

It is obvious that the deposition of the eggs must occur during the period of activity of the flies, *i.e.*, from about June to September, and the histories bear this out in showing that the first signs of conjunctival irritation occurred within this time. In the normal life-history of the flies, *Hypoderma bovis* for example, the eggs are laid in the hairs of the ox, the larva penetrates beneath the skin of the animal, and passes about nine months of incubation there, finally becoming extruded and falling to the ground. According to some the larva wanders from the skin into other parts of the body, returning to the surface before extrusion. According to others some larvae pass into the alimentary canal. It is almost certain that in those cases in which the larvae pass into the eye they do so from the conjunctival sac by penetrating the walls of the globe.

We are very deeply indebted to Mr. A. E. Shipley, F.R.S., and to Dr. Gordon-Hewitt, for the interest they have shown in the case, and for the trouble they have so
kindly taken, and also to Mr. G. Coats for preparing the sections.

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(October 15th, 1908.)

Mr. Herbert Parsons said it only remained for him to add the pathological description, and the notes of previous cases. With regard to the pathological examination, there was intense irido-cyclitis set up, and the retina was detached. Apart from that, there was nothing very special to note. A section had been placed under the microscope showing the parasite in the anterior chamber. The Society was indebted for the sections to Mr. Coats. With regard to the literature of the subject, he had found records of three previous cases of Dipteron larva in the anterior chamber of human beings. In two of them they were described definitely as larva of Hypoderma bovis; in the third it was carefully examined by an entomologist, von Kennel, and the case was published by Ewetzky, but he was unable to determine the species. Von Kennel gave a long description, and thought the larva had undergone changes and arrest of development owing to its unusual environment. The fourth case was published by Stählberg, and of that he had only been able to see a short abstract. There were no other recorded cases of intra-ocular larvae; there was a description of a fairly common disease called "myiasis," due to Dipteron larva in the conjunctival sac, and the same condition occurred in Mexico, but in none of them did the larva become intra-ocular. He favoured the idea that it was conjunctival to start with,
and that it made its way through the sclerotic. All the cases on record occurred in young children, the oldest æt. 9 years, and the others æt. 5½ years, 5 years, and the present one 2½ years. The sclerotic was not so hard in children as in adults, and that might explain its becoming intra-ocular in children. It was difficult to explain the presence of the parasite invariably in the anterior chamber in the cases recorded if the idea was correct that it got into the eye by the blood-stream.

The President said a most interesting account had just been given them of what was evidently a very rare condition. So far as he knew, it was the first time it had been met with in this country. He had looked up the subject in MacKenzie's Diseases of the Eye—an old store-house of information—and he found there mentioned one case where a man in a state of intoxication had the larvæ of a fly deposited in several of the natural openings of his body, including both conjunctival sacs. From there larvæ had penetrated deeply, and both eyes were destroyed. He believed that this occurrence took place in France.

Dr. George Mackay said that he had recently a somewhat similar case under observation, and was now waiting for the pathologist's report upon it. A few months ago a little girl was brought to him at the Eye Wards of the Royal Infirmary, Edinburgh, with what looked like a small threadworm in the right anterior chamber. The eye had lately been showing signs of irritation. There was slight ciliary injection, the pupil was clear. The supposed worm was situated in the lower part of the anterior chamber. It looked as if it had entered at the base of the iris about the five o'clock position, and coming forward had ascended the lower half of the posterior surface of the cornea, then turning backwards towards the iris and again meeting resistance had hung its head and given up the struggle. It had thus a slight spiral twist. Though watched for some days it showed no signs of vitality, but the parents thought it had been growing lately. With the pupil dilated nothing abnormal was
detected posterior to the iris with the ophthalmoscope. No detachment of the retina. Vision about 6/18. A section made with a keratome introduced beside the filament at the corneal margin enabled him to remove it with iris forceps. It was brittle, and broke into two pieces so soon as grasped. The suggestion that it might be a Dipterous larva was new to him. As regards previously recorded cases, Dr. Argyll Robertson had reported something similar to the International Ophthalmic Congress at New York, on September 14th, 1877 (see Compte-Rendu, p. 103). Dr. Mackay hoped to make a further communication upon his case at some future date.

Major R. H. Elliot, I.M.S., said that in India worm in the eye was commonly met with in horses. It was very common in the Madras Presidency, and he had taken out many such worms from horses' eyes for veterinary surgeons. The symptoms were different from those just described. Seen in the early stage the eye was clear, but a small white worm could be seen, with a lashing tail, moving rapidly from one end to the other of the anterior chamber. He had seen the worm pass from the anterior to the posterior division of the aqueous chamber, and back again. The veterinary surgeon would sometimes say that the worm had disappeared from the eye, but on manipulation of the eye the worm would appear again from behind the iris. The usual method of operating was very crude; the veterinary surgeon threw the horse, seized the eye with the conjunctival forceps, waited until the worm came to the part which was most convenient, and then gave a sudden jab with a keratome through the cornea; with the gush of aqueous the worm came away. In Madras he had taught them to make an incision with a sharp keratome, "stand by" with a blunt curette until the worm came near the incision and then press the posterior lip, when the worm popped out. If there was delay for a few days the cornea became more and more opaque, the opacity apparently starting from the posterior surface, and might become so dense as to make the horse practically
CASE OF DIPTEROUS LARVA IN THE ANTERIOR CHAMBER.

blind. In all the cases which he had seen the eye had cleared up with the removal of the worm. One case, of great interest to him, was where a child came into hospital with what he took to be worm in the anterior chamber. He was a little too enthusiastic about it, and the parents suspected that he had some motive of his own, and removed the child from him. He could not give a classification of the worm, but veterinary surgeons told him that the original parasite was supposed to be taken with water into the alimentary canal. It was often followed by what they called "kamri" (the waist), and the disease meant paralysis of the hind legs, due, he was told, to meningitis or myelitis, or both. It was said that the eggs of the worm found their way into the spinal theca, or into the cord, and set up inflammation. In one case in which he operated on a horse for worm in the eye, the horse died soon after of kamri. He lost a horse of his own from that disease, and had met with worm in the eye in yet another of his horses, so they would gather from his personal experience how very common the parasite was in Madras.

Mr. Hewkley asked which was the head of the worm exhibited. There seemed to be a very sharp business end, which gave colour to the view that it might be an active motor end. A very interesting article was written by Prof. Sir Ray Lankester on the question of the bookworm. He showed that it would go through a whole series of things, and would not even stop at lead. So it did not seem necessary to suppose that the mucous membrane of the nose was a source of entrance; the conjunctival sac seemed a natural place for the attack.

Mr. Ormond said Dr. Argyll-Robertson had recorded, on two occasions, Filaria loa, which was commonly found on the West Coast of Africa, under the conjunctiva.

Mr. G. Coats said that in the eyelid of a mouse he found a worm, probably a Strongylus. One of the ankle-joints was also diseased. The case was under investigation.
Mr. G. H. Poole suggested that possibly the horses died of trypanosomiasis, and the insect which carried it might also deposit such larvae as were under discussion.

Mr. Thomas, in reply, said that particulars of most of the things which had been discussed could be found in Neumann's *Parasitic Diseases of Domesticated Animals*. In his own specimen, he regarded the thick end of the maggot as the head, and the pathological examination bore that out. Certain larvae had spiked or armoured heads, by means of which they bored through hard substances. He believed the Hypoderma of the ox had such an end, so that it could pierce the skin and live in the subcutaneous tissues. There were several species of flies which had penetrating organs, known as ovipositors. By means of them they could even penetrate tough substances such as the bark of trees, and deposit their ova in that fashion. He showed a specimen of such a fly, the Crane fly. But the fly of which his case was the larva had no ovipositor, so that the larva itself must have worked its way into the eye by some other means.

8. *A study in heredity—six generations of piebalds.*

By N. Bishop Harman.

At a time when the study of heredity is being diligently pursued, the details of the inheritance of a well-marked and easily determined physical character in a human family may present features of interest.

The family is English. The earliest known member lived in a large south-western cathedral city. Now the family is scattered over the southern counties and in several distant suburbs of London. Particular inquiries have been made regarding marriages; there has been no case of in-breeding, and on no occasion has a consort been marked by any peculiarity of the kind borne by the family.
The inherited physical character is a sort of partial albinism: certain parts of an otherwise normal body are devoid of pigment. For the most part the characteristic markings are these: A white forelock, a white patch of skin spreading from beneath this white hair down the centre of the forehead like a "flare." The position is distinctly median and corresponds to the area of skin supply of both supra-trochlear nerves. In some subjects patches of white skin about the median line of the trunk, or on the inner sides of the calves of the legs. The white tissues are appreciably more delicate than the normal skin and hair. The colour of the ordinary hair of the family ranges from light brown to a deep brown, almost black. The eyes are quite normal; in no case have I seen any parti-coloured irides, or any whitening of the eyelashes. In one case only there were white tufts in the eyebrows (see photo). There have been no other physical anomalies found. There is a distinct tendency to an early "senile" whitening of the hair of the head, particularly in those whose hair is darkest, but the white lock is always distinguishable by its superior delicacy. The members of the family are long-lived, robust, and intelligent.

Six generations have been traced; of these four are now living, and have been seen. The generations comprise thirty-four childships and about 138 individuals. The piebald marking continues in a direct line of descent in these generations in nine childships and twenty-four individuals.

In the chart the lines of the family-tree are shown in detail. Some notes may be given of individual cases.

*Generation I.*—A Mrs. Safe (1750–1835); she is remembered by No. 7 in Gen. III, who vouches for the presence of the white forelock. Members of Gen. IV, living in widely separated places, preserve the tradition of their great-grandmother. Nothing can be discovered of her ancestry or connections.

*Generation II.*—Mrs. Safe had two children; one a girl, with the white forelock, the other a boy, not marked.
I Generation

II

III

IV

V

VI

\( \bullet \) = piebald, \( \bigcirc \) = not piebald.

\( \bot \) = died at birth, \( \downarrow \) = married, no child.

NB.—Unaffected collaterals are put in small size, and to save space the total number in a childhood is given: \( 2c \) = two children.
The latter had children and grandchildren, none of whom were marked.

*Generation III.*—No. 1 of Gen. II bore 14 children. Two died at birth, of whom nothing can be said. There were nine girls, of whom five had the white forelock; and three boys, none of whom were marked. Five of those who were not marked (three girls, two boys) married and had children and grandchildren, none of whom are marked. Of the five marked girls two only married; one only had children, a childship of six; this mother is still alive, at 83 years, in strikingly good health mentally and physically; the white forelock is distinct in spite of her grey hair.

*Generation IV.*—Only one childship of six, the children of Gen. III, No. 7, need be considered.

No. 1, Male, at. 56 years; has a large white forelock, a small patch of white skin beneath it and a large patch of white skin on the left leg. He has one child and one grandchild slightly marked, and others unmarked.

No. 2. Male, drowned, at. 23 years. Photograph shows a large white forelock.

No. 3. Female, at. 50 years, has a large white forelock and a large white patch of skin around her navel. Married, but no children.

No. 4. Male, at. 48 years. White forelock of small size. Has children by two wives, most of whom are strongly marked.

No. 5. Female, at. 46 years. As a child is said to have had a white patch on each knee; the woman says she cannot find the patches now. Her children are unmarked.

No. 6. Male, at. 44 years. White forelock. Has two children, one marked.

*Generation V.*—Nos. 1 and 2 (children of Gen. IV, No. 1). The girl is not marked. The boy has a tuft of white hair over the occiput, and a patch of white skin over the sacrum. He has children; one is slightly marked.
Photograph of I. H. --, at 5 years (Gen. V, No. 11). The central white forelock is of about average size; the "flare" is larger than usual. There is a small white tuft in the middle of each eyebrow. The spot on the left cheek is a pigmented mole.
Nos. 3 to 11. A man (Gen. IV, No. 4) married twice. He bore no blood relationship to either wife, nor were the wives related to each other. At the first marriage husband and wife were each aged 21 years; at second marriage husband was aged 30 years, wife 27 years. In the first childship of four, the two eldest girls have well-marked white forelocks; No. 5, the boy, has a few white hairs in his forelock (he has dark red hair) and white patches on his calves; No. 6 died at birth. In the second childship of five all are piebald; Nos. 7, 8, 9, and 11 have white forelocks and white flares of large size; No. 11 has also a small tuft of white hairs in the middle of each eyebrow (see photo); No. 10 has a small, white flare on the forehead, and a large white patch on the belly abutting on the middle line. None of the children are married yet.

No. 14. The daughter of Gen. IV, No. 6, has a well-marked white forelock. Her brother has no mark.

Generation VI.—Four children in one childship. Nos. 1, 2, and 4 have no marks. No. 3, a boy, has a narrow, white stripe in the lumbar furrow.

Remarks.

The most outstanding features of the pedigree are these:

(1) The piebald is conveyed by direct descent. Mother or father hands it down to the child.

(2) Once the piebald has been lost there is no evidence of a revival of the marking, so that an unmarked parent has an unmarked child.

* The following have been seen by me († Shown to Ophthal. Soc.). Gen. III, 7 (and her husband); Gen. IV, 1 (and wife) 3†, 4 (and second wife), 6. Letter received from No. 5. Gen. V, 2 (and wife), 4†, 5, 7†, 8†, 10, 11†, 12†; Gen. VI, 1, 3, 4. Photographs have been seen of Gen. II, 1 (not very distinctive owing to cap worn), Gen. IV, 2, and Gen. V, 9, both distinct. Letters have been received from children of Gen. III, 3, 4, and 8; one of these, a mother of six children, has been seen.
(3) The children are not marked in proportion to the extent of the marking of the parent. Gen. IV, No. 1, is very broadly marked, yet the marking seems to be dying out in his grandchildren. Gen. IV, No. 4, is only fairly marked, but his children by two wives are very broadly marked.

(4) Sex inheritance. In Generations I, II, and III the piebalds are all females, and the inheritance is through them only. This rule is broken in Generations IV, V, and VI, where males and females are affected in equal numbers, and the inheritance is through the males only.

**Mendel’s Law.**

It is a matter of difficulty to determine how far the pedigree of a human family fits in with the law of inheritance as propounded by Mendel. Thomson, in his recent work (*Heredity*, 1908), states that it is doubtful how far, if at all, Mendel’s law applies to humans. Bateson, in the discussion on Mr. Nettleship’s paper ("A History of Congenital Stationary Night-Blindness in Nine Consecutive Generations," *Trans. Ophth. Soc.*, 1908), gave two tests as an indication of Mendelian inheritance.

Mendel’s law can be best stated in the terms of his simplest experiment: Tall and short peas were crossed; the progeny were all tall, the tall variety was dominant. Subsequent in-breeding showed the short type was not lost, for the next generation gave tall, short, and "tweenies" in equal proportions. Now since in the pedigree of the piebalds there is no in-breeding, each childship is the result of a fresh cross, so one would expect all the children of a childship to be either piebald or not piebald. This is not the case. A complete childship of piebalds as dominants occurs three times in nine piebald childships: Gen. IV, Nos. 1 to 6 (and No. 5 is a little doubtful), and Gen. V, Nos. 3 to 6, and Nos. 7 to 11.
And the odd thing is that the last two groups are two childships by the same father.

Bateson's tests are these: "A Mendelian dominant might be tested in two different ways: (1) By the simple evidence that it was transmitted only by the affected. (2) If the case were of the simplest Mendelian kind, then the offspring of the affected, married with an unaffected person, should be affected and unaffected in equal numbers." Test 1 is true of this pedigree, the inheritance is only by the affected. Test 2 is true in Gen. II. Nearly true in Gen. III (7 unaffected, 5 affected, 2 dead at birth.) Not true in Gen. IV, for all are affected. True in Gen. V, Nos. 1 and 2. Not true of Gen. V, 3 to 6 (3 affected, 1 dead at birth). Not true of Gen. V, 7 to 11, for all are affected. True in Gen. V, 14, 15. Not true in Gen. VI.

The elaborations of Mendel's law are very difficult to follow and to bring into line; knowledge is at present very partial. But there are certain difficulties that want solution:

(1) Bateson says in effect that to him that hath marks, to him shall be given children with marks, and to no one else. How, then, did the original ancestor of these piebald, white-forelock people get his marks? Mendel got "tweenies" in his in-breedings, which kept on breeding dominants, "tweenies," and recessives. Thomson, in Heredity, allows for "discontinuous sports," or jumps in variation, and cites cases of this kind known to Darwin. Unless our favoured law of inheritance can admit the possibility of the transmission of characteristics (acquired or originating as sports), then we are forced to the conclusion that Adam was a piebald, and we, the self-coloured majority of the population, are recessives to these original dominant piebalds!

(2) Punnet, following Bateson on Mr. Nettleship's paper, said that a physical feature "apparently would never disappear," and "could only be got rid of by the death of those who carried it." Now in this pedigree five piebalds got rid of the mark in some of their progeny,
LINEAR OPACITY OF CORNEA, FOLLOWING BIRTH INJURY. 33

and one got rid of it completely; and from these unmarked children have proceeded seventeen childships quite free from piebald. It has been got rid of! Therefore it would appear that an affected stock need not necessarily carry on the inheritance.

Telegony.

It goes without saying that the family has a legend to account for the occurrence of the white forelock. It is their tradition that the mother of Mrs. Safe (Gen. I) was frightened by a piebald horse when she was "carrying" the child. I had this from two branches of the family. From another branch I had the more mysterious story that old Mrs. Safe promised to tell her grandchildren the reason for their being so marked when they were old enough to understand, but she died with the story untold! (November 12th, 1908.)

9. Linear opacity of cornea, following birth injury.

By R. R. James.

(Introduced by J. Herbert Parsons.)

Grace N—, aet. 13 years, attended the Royal London Ophthalmic Hospital, under the care of Mr. Parsons, a fortnight ago, complaining of mistiness of the right eye, which she had noticed for three weeks.

Her vision was R. \(\frac{3}{6}^{\text{o}}\) no H.m., words of J. 1 at 3 in. L. \(\frac{6}{6}\), H.m. 0·5, J. 1 at 12 in.

The pupillary reactions were normal, and the tension was normal.

Under atropine her refraction was as follows:

\[
\begin{align*}
R. & \quad -100 \quad -60 \text{sph.} \\
& \quad -50 \quad -50 \text{ cyl.} \quad \text{H.} = \frac{5}{6} \text{ a.} \\
L. & \quad +2 \\
& \quad +2 + 10 \text{ sph.} = \frac{3}{6} \text{ a.}
\end{align*}
\]
The left eye was found to be normal.

In the right eye there was found a linear opacity, situated deeply in the cornea, traversing the whole length of the cornea from above downwards, and situated just to the right of the middle line. This opacity measures less than 1 mm. across, and is vertical in position. Besides the main opacity there are others as figured (Pl. I, fig. 2).

The right fundus showed a small temporal crescent, and nothing else abnormal.

Corneal measurements: R.: Transverse, 13 mm.; vertical, 11·6 mm. L.: Transverse, 12 mm.; vertical, 11·6 mm.

Seen a week later, R.V. under correction not improved, i.e., still $\frac{6}{3\frac{6}{6}}$.

The corneal astigmatism was measured, and found to be 5 D.

The mother gives the following history: All her confinements have been difficult. This child, the last of thirteen, was delivered by forceps. Next day it was noticed that the eye looked crushed, and that the "sight of the eye" was like a bit of liver. There was a slight scratch on the skin over the right external angular process of the frontal bone. The mother is not certain about any bruising of the lids being present, and is rather inclined to contradict herself. It took six weeks for the "sight" of the right eye to clear up. (Card specimen. November 12th, 1908.)

10. Vascular changes in "albuminuric retinitis."

By Angus Macnab.

A. H. B.—. July 8th, 1908: The refraction was investigated; he complained of difficulty in reading. R.V., $-75$ cyl. $\oplus 80^\circ = \frac{6}{6};$ L.V. $= \frac{6}{6}$. Reading glasses were ordered.

August 22nd, 1908: Sudden failure of left eye. Fundus changes showed a recent obstruction of the central artery. Cherry-red spot at macula. There was no P.L. in left.
September 5th, 1908: Retinitis, with plastic exudation and haemorrhage in the other eye. Admitted for treatment of albuminuria.

At the first visit the patient was considered to be an ordinary presbyope with astigmatism. When the second eye became affected there was a considerable amount of albumen in the urine; this diminished very much under treatment, and the last record reports a "trace."

The interesting appearance of the retinal arteries of the left eye is worthy of note. Immediately after leaving the disc the inferior temporal artery becomes opaque, white, and "frosted" for a length of about one disc's diameter; beyond this the vessel wall again becomes transparent, with opaque lengths alternating to the periphery.

The same condition, less marked, is found in the upper temporal vessel.

There are retinal haemorrhages, many of which show degenerative changes.

Present vision.—L.V. = no P.L.; R.V. = $\frac{6}{18}$.

The heart has been examined, and no gross lesion discovered.

The case appears to be one of arterio-sclerosis, producing thrombosis and plugging of the central artery on the left side. On the right side the vessel walls are degenerate, but the circulation is still intact.

(Card specimen. November 12th, 1908.)

11. Acute optic neuritis in one eye with changes at the macula, in a girl without any evidence of constitutional disease.

By Rayner D. Batten.

Annie H—, aet. 16 years. On October 22nd, 1908, the sight failed suddenly in the left eye. V. = R. $\frac{6}{12}$. L. $\frac{6}{12}$.
(In June, 1908, the vision was equal in the two eyes.)

The patient was first seen on October 29th. She then had intense optic neuritis in the left eye. Swelling of her O.D. estimated at 4 D. The retinal vessels hidden inœdema; numerous fine radiate changes at the macula.

On November 9th the neuritis was subsiding. The swelling estimated at 2 D. The macular changes were still well marked. V. improving \( \frac{6}{3} \). The field is somewhat restricted, and there is a central scotoma for red and blue.

The patient appears well and healthy in every way. No evidence can be found of any constitutional disease.

There has been no pain, or tenderness on pressure over the eye.

The right fundus is normal. There are no myopic changes. (Card specimen. November 12th, 1908.)

The President said he remembered a case of his own which was very similar to Mr. Batten's, and occurred also in a girl of about the same age. Nothing was found amiss in her general condition to account for it. He believed Mr. Jessop showed a similar case before the Society. He presumed that such cases were instances of very severe retro-ocular neuritis occurring close behind the papilla, and consequently associated with particularly marked ophthalmoscopic changes. In his own case the papillitis gradually disappeared and the macular figure faded, but central vision was never perfectly restored.

Mr. Whitehead said it would be interesting to have a report on the case twelve months hence. In some similar cases which he had seen the optic neuritis became double, and symptoms of intra-cranial lesion manifested themselves.

Mr. Johnson Taylor asked whether the nose was examined in this case, to find out whether there was disease of the sphenoidal or other sinuses. Sometimes such a condition was due to extension from a sinus.
Subsequent note.—December 10th, 1908: V.L. = \( \frac{6}{36} \). The left O.D. appears normal. The macular area appears as a dark circle and some of the white retinal streaks persist.

January 12th, 1909: V.L. = \( \frac{6}{18} \). The left fundus appears normal. No trace of the previous neuritis or retinal changes discoverable.

12. Case of posterior cataract commencing subsequent to prolonged exposure to X rays.

By Leslie Paton.

Miss M. N—, act. 32 years. A little over six years ago patient had good sight in both eyes. She could see well in the distance ("better than most people"), and could do the finest needlework. At that time she was suffering from lupus on both cheeks and underwent treatment with X rays, the old form of unprotected tubes being used. She had twenty exposures on the right side and eighteen on the left side, the eyes being protected with rubber. During the time of the exposures the only trouble she experienced in her eyes was some swelling of the lids and a feeling of grittiness with slight inflammation. Occasionally her eyes became bloodshot. Nine months after this period her sight had failed very distinctly, and two years after she could see nothing with her right eye, and badly with her left eye. In January of this year she was under treatment at St. Mary's Hospital for lupus, and Sir Almroth Wright sent her to the Ophthalmic Department.

Condition in January, 1908: R.V. = fingers at 1 yd.; L.V. = fingers at \( 1\frac{1}{2} \) yds.

The right eye could not be completely closed owing to the cicatrisation of the lower lid. The ocular conjunctiva of each eye showed numerous clusters of little curling
varicose vessels, more numerous immediately round cornea, and in the R.E. more than in the L.E. On dilating the pupils the main opacity was found to be in the form of a dense, greyish-white granular plaque, lying in the posterior part of the lens well behind the nucleus, and probably against the posterior capsule. The rest of the lens showed a granular opacity, more in the cortical portions and somewhat denser at the periphery. Both lenses were similar in appearance. Mydriasis did not produce any definite improvement in vision.

The right lens has been removed after preliminary iridectomy, and the vision in the R.E. with +11 D. = 6/6, with +14 D. = J. 1.

The fundus is normal.

In the left eye a preliminary iridectomy has been done. (November 12th, 1908.)

The President asked whether there were any evidences of associated disease in the fundus which might account for posterior cataract. A good deal of attention was at present being directed to glass-workers' cataract, and it would be of great importance if members generally would bring forward any personal observations bearing upon this subject, particularly with regard to opacities in the lens resulting from exposure to heat, bright light, X rays, or to radium. The present general appearance of the opacity in Mr. Paton's case was very similar to that occurring in glass-workers' cataract, and it was certainly suggestive that this seemed to have formed after exposure to X rays.

Mr. Paton replied that there was no evidence of disturbance in the fundus. The patient had 6/6 in the eye which had been operated upon, J. 1, and a good field. There was no choroidal disturbance, and no history pointing to cyclitis at any period. The only external change was in the form of curious little clusters of vessels scattered over the fundus, but they were less numerous since the iridectomies had been done.
13. A case of orbital abscess following retinal embolism.

By A. L. Whitehead.

Mary L.—æt. 42 years, first came under my care on March 22nd, 1908.

She was married, and had five healthy children, the last having been born three years previously.

Menstruation had ceased about twelve months ago. On March 7th she commenced to suffer from pain in the right shoulder, with rapid rise of temperature and shivering. The elevation of temperature, with pain and some swelling in the right shoulder, and general malaise continued for ten days.

In the early morning of March 17th some dimness of vision was noticed in the left eye, at first a general cloudiness which rapidly became worse, so that in a few hours she was practically blind. Towards evening the eye became congested and painful; during the next three days the inflammatory symptoms persisted and increased, and a progressive proptosis was noticed.

When seen on March 22nd, that is, five days after the onset of the blindness, there was extreme proptosis, with diffuse swelling of the orbital tissues.

The cornea was steamy, there was a small hypopyon, iritis was present, and the pupil was covered with exudation; vision was nil.

There was some pain on moving the right shoulder, but no marked tenderness or swelling, and no redness of the skin.

There was a soft systolic mitral bruit. No focus of suppuration could be found in any part of the body, and the pelvic organs were healthy. The temperature was 102·2° F., and the pulse-rate 120. The original condition having been diagnosed as acute rheumatism, sodium salicylate and aspirin had been given, with marked relief from the pain.
Two incisions were made into the orbit and some pus was evacuated; the eyeball was subsequently excised, and pus was found to be escaping from a small opening in the outer wall through a necrosed area of sclerotic.

Four days later pain was complained of in the right arm, and the brachial and axillary veins were found to be thrombosed; the thrombus subsequently broke down, and an abscess formed in the axilla. This was opened and drained. The axilla and orbit healed rapidly and uneventfully.

When she returned to her home in the country on April 25th the systolic bruit could be heard only at intervals, the right shoulder was quite mobile and free from pain, and her general health was satisfactory.

Although endogenous panophthalmitis of embolic origin was apparently common in the pre-antiseptic days, especially in puerperal cases, it has become comparatively rare at present, and from the cases recorded perforation of the sclerotic and orbital cellulitis would seem to be the less frequent termination.

An infective endocarditis associated with acute rheumatism is, of course, well known, but in this case the general constitutional disturbance was not so severe as would be expected in such a condition. It is difficult, however, to find any other cause for what the symptoms would lead one to diagnose as an infective embolic retinitis.

(November 12th, 1908.)

Mr. Herbert Parsons asked whether any bacteriological examination had been made of the blood or tissues. The behaviour of the various organisms was very different. As an example, he had recently seen at Great Ormond Street Children's Hospital a very interesting case, in which the child was admitted for mastoid trouble and middle-ear disease. It was operated upon, and the sinuses were washed out repeatedly. But the child became worse. She developed left metastatic iritis, with hypopyon, and it went on to panophthalmitis. But only a few days
before death was there optic neuritis in the other eye. Post-mortem, there was found to be an abscess in the right uncus, on the side opposite to the middle-ear disease. In that case the blood contained streptococci during life, and they were also recovered from the sections of the eye which Mr. Coats examined with him. Records of cases showed that different organisms had a great tendency to selection of site when metastatic infections started in the eye. Another point was the different actions of those organisms by endogenous as compared with exogenous infection; they were often less virulent when reaching the spot by the blood-stream than when they came from the outside. It was for those reasons he asked the nature of the organism in the present case. In reply to the President, it was very difficult to say whether there was any particular organism more likely than another to affect the retina and optic nerve. But the pneumococcus seemed to start in the retina rather than in the uveal tract. It would be of great interest to know whether the Diplococcus rheumaticus was in the blood. Also whether the organism was Gram positive or Gram negative.

Mr. Whitehead said examination was made of the blood, and of the pus from the eye and from the abscess. In the first two cases it was sterile, but in the axillary abscess there were some diplococci. He would be glad to find out the Gram reactions.

Mr. MacNab said that many of the cases where there was a localised abscess were pneumococcal, and that, in later stages, the abscesses were sterile. When encapsulated the pneumococcus rapidly dies out. So that this case, being subsequently sterile, would probably be pneumococcal.

By Herbert H. Folke.

On September 21st Harriet R—, æt. 21 years, a married woman, was sent to me by Dr. Carter, of Tunstall, on account of defective vision and a peculiar form of keratitis (Pl. II, fig. 1).

History.—Has noticed a defect in her vision as long as she can remember. She can only recall two attacks of inflammation, one three years ago, the other last year, each attack being slight and lasting about a fortnight. Has a baby ten weeks old, and since the confinement the vision has become worse. No indication of syphilis or history of rheumatism. She is one of eleven children.

Examination.—With focal illumination both corneas show irregular opaque patches confined to the centre, the periphery being clear. They appear to be confined to the anterior layers of the substantia propria, and differ in degree of intensity, some of the opacities having the appearance of small funnel-shaped depressions. The epithelial surface is quite smooth and free from any signs of nebulae. Fluorescein gives no reaction. Pupils react well; T.n. R.V. 6/65, L.V. 6/65, not improved with glasses.

States that her mother and some of her brothers and sisters have defective sight. This statement led to an inquiry, with the result that I was enabled to examine most of the other members of the family through the courtesy of her doctor, to whom I am deeply indebted for the trouble he has taken in bringing them to light.

George P—, æt. 92 years. First noticed his vision failing forty-two years ago. Says he underwent operation for double cataract in 1868. He is a wonderful old man for his age, enjoying very fair health. He worked for nearly fifty years as a forgeman. There are no signs of syphilis; his wife is living, and they have had a family of
Fig. 8.

Pierpoint Pedigree

- : Female
- : Male
\(\) : Not Sexured

\(\) : Examine, not optically

\(\) : Obliquity present

I II III
thirteen children, seven of whom are now living. There were no miscarriages.

Both eyes have had iridectomy inwards performed, and there are traces of opaque capsule filling in the coloboma. The centres of both cornea are occupied by a mass of irregular white opacities, which have the appearance of a dense leucoma; the periphery is fairly clear; the epithelium is quite smooth and free from opacity. V. = fingers at one metre; no conjunctival injection; T.n. (Pl. II, fig. 2).

Mary Ann H,—, aet. 50 years, daughter of George P.—. Says her vision has always been defective; remembers her eyes being inflamed when quite a child. No signs of syphilis or rheumatism. Says she has always been a strong, healthy woman; has been married twice; no family by the second husband; eleven children by the first, eight of whom are living; two died at twelve months and one at eighteen months (two after scarlet fever and one after bronchitis) (Pl. II, fig. 4).

Examination.—Both cornea have dense irregular opacities situated in the central area interspersed with fine white lines, which invade the periphery; they appear beneath the epithelium and in the anterior layers of the substantia propria. The epithelium is quite smooth. Details of the fundi cannot be made out owing to the opacities; T.n. R.V. 6; L.V. 6; pupils react sluggishly.

William P,—, son of George, aet. 46 years. A collier by trade. Says his vision has always been defective. Has had occasional attacks of inflammation, but they have always been slight (Pl. II, fig. 3).

Examination.—There are two or three small nebulae on each cornea, the result of foreign bodies, otherwise the epithelial surface is quite smooth. Beneath the epithelium are dense, irregular patches, with fine white lines interspersed similarly to the last case. The opacities are confined to the central two thirds, whilst the periphery is practically clear. No signs of syphilis. Has a family of nine.
PLATE II.

Illustrates Mr. Herbert H. Folker's paper on Nodular Opacity of the Cornea in Three Generations (p. 42).
Fig. 1.—Harriet R—.

Fig. 2.—George P—.

Fig. 3.—William P—.

Fig. 4.—Mary W—.
NODULAR OPACITY OF CORNEA IN THREE GENERATIONS.

Sarah, aet. 38 years; Hannah, aet. 36 years. Both clear.

Elizabeth W — , aet. 30 years. Is the eldest daughter of Mary Ann II — . Has been married eight years, had six children, five of whom are living. One died when five weeks old from convulsions. She has always noticed a defect in her vision, and the sight has been worse after each confinement, recovering in a slight degree, but never attaining the condition it was prior to parturition. Has had frequent attacks of inflammation, but never severe enough to prevent her doing her ordinary work.

Examination.—Slight conjunctival injection. Pupils react well. T.n. The centre of the cornea in each eye is occupied by irregular opacities, situated in the anterior layers of the substantia propria, of a fair size verging off to smaller ones as they approach the periphery. The epithelial layer is unaffected. The fundi can only be seen indifferently, but they appear quite normal. R.V. $\frac{6}{24}$, L.V. $\frac{6}{36}$.

William W — , aet. 28 years. Has always noticed a defect in his vision. Has had occasional attacks of inflammation, but they have never been of a severe type.

Examination.—The cornea in this case presents a different appearance to the others, the spots having coalesced into one large opacity, occupying chiefly the lower part of the central area. There is a smaller mass above and more central. The density appears fairly uniform. The periphery is clear. R.V. $\frac{6}{18}$, L.V. $\frac{6}{18}$.

Frank W — , aet. 24 years. Corneas clear. V. $\frac{6}{6}$, both eyes.

Annie W — , aet. 18 years; single. Has had intermittent attacks of inflammation, which are increasing in frequency and severity. Notices that her sight is gradually getting worse.

Examination.—Slight conjunctival injection. Pupils react well. T.n. Slight irregular-shaped dots, occupying the centre of the cornea, distributed in the form of a circle. The peripheral portion is quite clear. The
epithelial layer is not involved. R.V. \( \frac{6}{12} \), L.V. \( \frac{6}{12} \) nearly.

Thomas, æt. 16 years. Corneæ clear. V. \( \frac{6}{6} \), both eyes.
Minnie, æt. 14 years. Corneæ clear. V. \( \frac{6}{6} \), both eyes.
Ethel, æt. 12 years. Corneæ clear. V. \( \frac{6}{6} \), both eyes.

George Thomas W—, æt. 21 years. Eldest son of William P—. Says he has never noticed any defect in his vision. Has had occasional "redness" of his eyes.

Examination.—The corneæ of both eyes have some twenty or thirty small dots, irregular in distribution and confined to the central area, the periphery being clear. They are beneath the epithelium, which is not involved. R.V. \( \frac{6}{6} \), L.V. \( \frac{6}{6} \).

Louisa, æt. 19 years. Corneæ clear. V. \( \frac{6}{6} \) in both eyes.
Sarah Jane, æt. 18 years. Corneæ clear. V. \( \frac{6}{6} \) in both eyes.
Hannah, æt. 14 years. Corneæ clear. V. \( \frac{6}{6} \) in both eyes.

William P—, æt. 12 years. Fifth child of William P—; is at school and experiences no difficulty with his lessons. Has not been troubled with redness of his eyes. V. \( \frac{6}{6} \) in both. The condition of this case is exactly similar to his brother, the opacities being a few small dots.

Polly, æt. 9 years. Corneæ clear.
Hilda, æt. 7 years. Corneæ clear.
Ethel, æt. 4 years. Corneæ clear.
Harold, æt. 3. Corneæ clear.

Remarks.—From the foregoing cases there are one or two points which, I think, deserve notice:
(1) That the condition is undoubtedly hereditary.
(2) The absence of any unevenness or elevation of the epithelium corresponding to the opacities, which Fuchs mentions as being present in cases of nodular opacities.
(3) The presence in two of the cases of fine lattice-work lines, which is allied to reticular opacity.

The aetiology of this disease seems to be unknown, and
from the literature I have read on the subject there seems to be a difference of opinion as to the pathology, according to different authorities. Groenouw, who first described these cases, found hyaline deposits. Chevaliereau found deposits of crystals of sodium urate in his case. Fuchs found four kinds of changes in the opacities, which were limited to the superficial layers. He describes the altered superficial layers corresponding to the opacities as due to the epithelium being raised by the nodules, but in some cases this was counteracted by diminution in the number of layers, the cells of which showed the degenerative changes usual under such circumstances.

Parsons says it is due to some general agent, since it always affects both eyes, develops very slowly, and sometimes occurs in several members of the same family.

The question occurs to me, Is there a limit to the life of the disease? Can it wear itself out in successive generations? and What treatment, if any, is the best to aid the attainment of such a desirable end? Personally, I have been unable to find any work which deals with the treatment, and shall be grateful to receive enlightenment on the subject from any member present.

References.

Parsons.—The Pathology of the Eye, vol. i.
Fuchs.—Text Book of Ophthalmology.
   (November 12th, 1908.)

The President congratulated Mr. Folker on the manner in which he had traced the inheritance of the affection in this family. In looking back on the work brought before the Society during the last few years, it was very noticeable how carefully this question of heredity had come to be considered. This was, he felt sure, particularly gratifying to one member who had done so much in this direction. He (the President) had only seen two families showing nodular opacities of the cornea with
NODULAR OPAcity OF CORNEA IN THREE GENERATIONS.

raising of the epithelium. One of these he had brought before the Society several years ago.

Mr. G. Coats said he had examined a shaving from Mr. Macnab's case. The nodules were due to a substance which, in its appearance and staining reactions, exactly resembled the hyalin material found in degenerate corneal scars. It was present in Bowman's membrane and in the immediately subjacent layers, and in places projected among the deepest layers of the epithelium. Small tracks of cells surrounded by hyalin material ran through the membrane; these might represent the "funnels" which had been mentioned.

Mr. Macnab, in reference to the case mentioned by Mr. Coats, said he saw all the available members of the family—father, mother, and the three cases with nodular opacity shown before the Society. A brother had posterior polar cataract, while a sister was quite normal. He had tried shaving off the anterior layers of the cornea, the opacity thereby being considerably reduced. Some observers believed that tubercle was a factor in the cause, but there was no evidence of tubercle in any of his cases. Calmette's reaction was negative. He had seen a case at Heidelberg; there the question of tubercle had been gone into. The patient seen in Germany had tubercular lesions, but his brother, seen in London, and having opacities, had not. Shaving the cornea was only a partial success.

Mr. J. H. Fisher referred to notes of a case of nodular keratitis which he had seen three years ago. The clinical appearances corresponded closely with those in the cases now brought. He had described the spots as not unlike snowflakes, and had noted that they caused no elevation of the corneal epithelium. His case seemed to some extent to bear out the hereditary nature of the disease. The patient was a woman, aged 47 years; she gave a history of having had slight attacks of inflammation of her eyes eight years ago. She was the oldest of her family. There was no evidence of syphilis in the case. A sister,
the fourth child, had "ulcers on the sight" when between twenty and thirty years of age, and the sight was imperfect. Another sister and three brothers had good eyes. Her father's father went blind in old age. Her father's brother had bad sight; he lost the sight of one eye and the other was defective. Her mother died of consumption at the age of forty-one. In one case brought forward by Mr. Treacher Collins there was a history of tubercle.

Mr. Holmes Spicer said that such cases were commoner than was usually thought; after seeing the varieties which had been shown at the Society one could now frequently recognise them in the early stages. Frequently there was progression, which could be recognised after a period of months; he had one case under his care now, of which he had made maps from time to time, showing a gradual extension of the opaque areas. In this case the opacity was uniform, extensive, and not broken up into nodules. With regard to treatment, he had scraped one case with marked benefit, and the scraping showed hyaline change with calcareous deposits. It seemed to be not very different from the transverse calcareous band, and he thought there might turn out to be a close affinity between the two conditions.

Mr. R. W. Doyne said he thought there were two distinct types of the condition. Mr. Stephenson and he had shown four generations of those in whom, at an early age, there was a very dense opacity. He agreed with Mr. Spicer as to their similarity to transverse calcareous films. They had little holes in them, and if there were not a good background they looked like spots of pigment. Other points seemed to show that nodular and reticular opacity were the same thing. In all those cases the epithelium had been raised, and the cases he had seen had never been followed by deep opacity and the obvious foreign-body appearance which existed in the cases shown by Mr. Stephenson, Mr. Folker, and himself. The nature of those he had seen was similar to scars in the tissues. In the cases described as nodular or reticular
opacity the affection was much more central, leaving the periphery clear. Mr. Spicer would remember a clergyman whom both of them saw, and he had a similar case, which developed into nodular opacity of the central part of the cornea; he did iridectomy to give him vision. The success of scraping in the cases of transverse films was marked in one case, and he suggested it might have been tried in the present cases.

Mr. Sydney Stephenson said that, apart from the tuberculous history which had been suggested, some of the cases were believed to be connected with dystrophy of the thyroid gland. He did not know whether Mr. Folker had inquired into that point, or whether, indeed, it was worth inquiring into.

Mr. Bishop Harman asked whether anyone had tried thiosinamine or fibrolysin, which were held to be resolvents of scar tissue. He had not tried those drugs, as they had to be injected, but he had tried allyl oil, which was the basis of such substances, for interstitial keratitis, and with benefit. He had used allyl oil in one case of nodular opacities of the cornea: A man, who had been the round of the hospitals, had previously been treated by scraping and trephining. One eye was practically blind, V. 1/60. Having warned the man of the painfulness of the application, he began by instilling drops of a strength of 0·5 minim of the oil to the ounce of castor oil, gradually increasing the strength to 2 minimis to the ounce. A cold pack was put on the lids immediately after the instillation; the pain lasted about five minutes and the reaction was brisk. The man had continued to come for the treatment for some time. He could not as yet say that it had resulted in benefit, but certainly the condition was no worse.

Mr. Nettleship expressed his appreciation of the pedigree shown by Mr. Folker. With regard to any analogy between the cases of nodular or reticular opacity and the previously described cases of calcareous film, he stated that in the latter disease the condition did not by any
means always begin at the centre, but often at the sides, the patches sometimes, but not always, coalescing at the centre. When beginning at the centre the patch was oval, not round, fitting very well with the fissure left when the eyelids were half opened. He thought there must be two conditions; the transverse calcareous film, which could sometimes be chipped off like an eggshell, could scarcely be the same as the condition described by Mr. Folker, in which the opacities were in the superficial layers of the substance of the cornea. He took the opportunity of appealing for the standardisation of pedigrees of disease by the use of the same symbols in all cases; much trouble and confusion would thereby be saved and greater accuracy ensured.

Mr. Treacher Collins said that he had very definite evidence of the tendency of these cases to progress. Some years ago he showed before the Society a typical case of nodular opacity of the cornea, a drawing of which appeared in the Transactions. He had seen the case at intervals several times since, and had been able, by comparison with the drawing, to watch the gradual extension of the opacities. The patient was a driver of a cart, but owing to the increasing failure of his sight had had to give up that occupation.

Major Elliot said he had not seen such cases as had now been described, but had seen a number of instances of transverse film in India, where it seemed mostly to be a matter of nutrition. The two classes of cases were quite distinct in his opinion. It was difficult to trace the ultimate result, because the patients were very irregular in attendance, but he thought it was bad. It was a common condition in children, which was naturally attributed to the fact that in times of famine it was the children who suffered most.

Mr. J. H. Parsons considered that although the pathology of the two conditions was probably similar, clinically they were different. Mr. Nettleship's remarks seemed very much to the point. The transverse calcareous
film appeared in different types. There was the rare type, specially referred to on the present occasion, in which vision was good if the film was removed, and there was the common pathological condition in shrunken eyes, where it developed as a result of drying of the cornea. He asked whether the rare form occurred in young patients; certainly the nodular and reticular forms did.

Mr. A. Hugh Thompson asked whether the condition was binocular in every case, and if so, whether the development was the same in both eyes.

Mr. G. Coats said that pathologically there was a difference between nodular opacity and transverse calcareous film. In nodular opacity the membrane of Bowman between the nodules was free from deposits; in band opacity scattered granules occurred uninterruptedly over a considerable area. Nodular opacity was a hyalin change; band opacity was usually mainly calcareous.

Mr. Folker, in reply, said the cases were not examined for tubercle, but he thought the ages mentioned would exclude it; ninety-two was a good age for a tubercular patient, and the smallest family was one of eight. He did not think what was seen were holes in the opacities; they were funnel-shaped. In all the cases except the old man there was a very fair margin of periphery of cornea absolutely clear. He did not know of any examination of the thyroid gland having been made. The condition was binocular, and there was no difference in the degree of opacity, but there were little differences in visual acuity.

15. An improved perimeter.

By Priestley Smith.

The original model of this perimeter was shown to the Society twenty-five years ago (Transactions, 1883). I have lately improved it in several respects:
The base is hinged at one end so that an adjustment for height can be made rapidly by a single movement, all the parts remaining in the same relative position. The slope of the instrument when raised has no disadvantage but rather the reverse.

The rest for the face is simplified. The ivory button against which the cheek rests is one inch in diameter. Its upper margin should stand about one inch below the pupil.

The arc, instead of presenting a mere edge, now presents a surface four inches wide. It is covered with black woolen cloth, so as to afford a uniform dull background for the fixation object. It is cut short at $85^\circ$, instead of at $90^\circ$ as heretofore.

The test-object, if the operator desires to use the tangential method, may be a white or coloured knob on the end of a wire, *e.g.*, a hat pin, which is held in the hand. In the great majority of cases the circular method is preferable, and the test object is a square of paper gummed upon a cloth-covered carrier, which is placed upon the arc. The test-object I usually employ is a 2 mm. square of white paper. Others of 1 mm. and 5 mm. are provided.

The chart is placed upon the back of the hand-wheel as before, and, if the tangential method be employed, it is marked by the help of a scale in the same manner as before. For the circular method the manner of marking has been simplified. The scale is supplemented by a series of short tubes corresponding in position with the circles on the chart. A small lead pencil placed in one of the tubes marks the corresponding circle on the chart when pressed by the finger. For example, the test-object being placed at the 10 circle, the pencil is placed in the corresponding tube; the hand-wheel is then rotated, and if the test-object disappears at any point the pencil is pressed against the chart until a point is reached at which the test-object reappears. For faintness of the test-object over any part of the circle intermittent pressure may be made, so as to make a dotted rather than a continuous
line upon the chart. By dealing in the same manner with successive circles we rapidly obtain an accurate chart.

The condition in which the perimeter gives the most important and the most necessary help is, I think, simple chronic glaucoma. In this disorder the shape of the field, and especially any change occurring between one examination and the next, can be determined with much greater precision by the circular method than by the tangential method. My present practice is in all cases where there is the slightest ground for suspecting glaucoma to test in the first instance with the scotometer. If no defect be found either in the 10 or in the 25 circle the question is settled. If a defect be found I usually resort at once to the perimeter, for the reason that it will test the entire field and give a graphic record. The circular method is also better than the tangential method for all varieties of hemiopic defect, and for most other conditions.

Messrs. Curry and Paxton, the makers of the original model, supply the improved instrument.

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By N. Bishop Harman.

Recently the editor of the Ophthalmic Review asked me to study the several papers written by Dr. Rémy, of Dijon, on the subject of the "Diploscope," an instrument devised by him for the demonstration of the existence and varieties of binocular vision. After the examination of his numerous papers I constructed a model of his instrument, a description of which I have given recently in the Ophthalmic Review.* It was evident that the "diploscope" was constructed on the principle of the well-known "bar-reading test" of Javal. Whilst I was experimenting

with these tests it occurred to me that a very useful, simple, and convenient instrument could be produced by reversing the plan of Javal—by substituting a diaphragm for an obstruction. By Javal's plan the patient looks, as it appears to him, through a pencil or bar; by the plan I propose the patient looks through a hole in a card and perceives what is behind the card without being aware that the objects he sees through the hole are not seen by both his eyes.

By a little experimenting and arrangement of the mutual relations of test object, diaphragm, and patient, I found it possible to obtain at one and the same time true binocular vision for an object fixed by both the eyes, and monocular vision of a separate object by each of the eyes, and all this without the patient being aware that his vision was being dissected in this fashion. Such conditions of vision may seem somewhat unnatural, yet they occur to us almost every day in our common experience, and we are so used to them that we take little note of the occurrence.

The test can be demonstrated with no other apparatus than one's own hands, thus: Stand facing a window; then hold up both hands, palms towards you, six inches from your face and on a level with the eyes; let the hands be separated so that the little fingers are distant from each other about three fingers' breadth. It is quite obvious that the hands cover the eyes, yet a perfectly clear perception of the view out of the window is obtained. But if one eye be closed half of the view is lost, for the right hand obscures the right eye, and the left hand the left eye; and the eyes see crosswise through the space between the hands.

The Apparatus.

To use the test in practice some apparatus is necessary, and I show you a very simple construction with which a great variety of very subtle and interesting tests can be
made. The instrument consists of a length of wood like a flat ruler, 44 cm. long, with a rack at one end to receive the test cards, and a clip at 11 cm. from the rack to receive the diaphragm.

The diaphragm is cut in a piece of mill-board, measuring 9 by 6 cm., there are two forms of diaphragm, one square, 1·7 by 1·7 cm., the other round, of 1·7 cm. diameter. Behind the diaphragm is fixed a fine wire, arranged so that it can be turned down into the opening as desired. There is a convenient handle fixed beneath the base board. In use the patient takes hold of the handle with both hands, and places the free end of the rule against the upper lip, just beneath the nose. (In testing a suspected malingeringer it is well to cover the free end of the rule with a piece of lint, and direct the patient to seize the end between his front teeth; this secures steadiness of the instrument.) When the instrument is in position the patient is asked to look either through the hole, or at the pointer projecting into it, according to the test desired, and to tell what is seen through the hole.

Tests.

1. Reading. A paragraph of print (J. 6, bourgeois in heavy-faced clarendon type serves best) is set in the test rack. The patient with normal binocular vision looking through the square diaphragm can read the whole of it
quite easily,* A careful observer will note the following appearances:

Fig. 11.

A large selection of Screens in Chippendale, Sheraton, Louis XV. and XVI. and other styles, also Lattice Screens, Japanese Screens, Cheval and Wing Screens: An example of value, the Four-Fold

Paragraph of print as seen through square diaphragm by subject with normal binocular vision. The dotted lines indicate the overlapping of the crossed images of the diaphragm. Both eyes see the narrow median columns between the dotted lines, the R. eye the remaining left portion and the L. eye the right portion.

That the hole, instead of being square, seems twice as broad from side to side as it is from above downwards, also that he can sometimes detect the over-lapping borders of the crossed images of the hole running across the middle of the print somewhat after the fashion of the dotted lines in Fig. 11. Now, if one eye, say the right, be covered, it will be found that the left half of the print is no longer seen. It is evident that, when the hole is looked clear through, it is seen by crossed diplopia, and the obstructing sides of the hole, which block the

* This test is very useful in determining equality of visual acuity in the two eyes; a variation of 0.25 D. is readily detected.
vision of the eye opposite to it, is negotiated by the other eye. The lines of the light rays are given in Fig. 12.

Any number of reading tests can be used, either whole paragraphs of print or lines of letters or figures. Simple tests of two large letters or figures can be employed. Or one large letter, one half of which is seen with each other one; for this test the letter W is particularly useful, for when seen whole it is promptly named, but the half of it is a V; the ornamental form of the letter \( \hat{W} \) is very good for this test. For children common objects such as separate triangles, apex to apex, or a large central cross are useful; some pictures are very successful, such as

\[ \text{Fig. 13.} \]

\[
\begin{array}{c}
\begin{array}{c}
\square \\
\square \\
\square \\
\end{array} \\
\begin{array}{c}
\square \\
\square \\
\end{array}
\end{array}
\]

A test card. Red \( \square \), green \( \square \).

that of Punch and Judy, joined as on the old-fashioned, crutch-handled sticks.

\textit{A most useful Test-card.}

There is another test which is of great value, and which serves several purposes; in fact, this test and a paragraph of print are quite enough for all practical purposes. On the test-card are set small squares of coloured paper; the squares on the one half of the card are set on a different level to those on the other half of the card (Fig. 13). The number and colour of the squares may be varied at pleasure; red and green are the best contrast, and one or three squares on each side the most convenient number. When the test is put in its place and examined by the
patent, it is seen quite distinctly as though it were laid on the table before him, yet the red squares are seen by the right eye and the green by the left.

Now turn down the wire pointer into the diaphragm and direct the eyes to that; there is binocular fixation of and vision of the pointer, yet at the same time we see slightly out of focus the red and green squares set above each other as in Fig. 14. In this test we get three separate factors: binocular and simultaneous fixation and perception of the pointer; and monocular perception of the red square by the right eye and the green by the left. The squares appear shifted so as to range one above the other, because fixation of the pointer has produced convergence, and consequently homonymous diplopia and apparent approximation of the more distant-coloured squares.

In general I find the tests and diaphragms may be varied as we wish to demonstrate the following:

*For Determining the Presence of Binocular Vision.*

To catch out a suspected malingerer it is best to use the square diaphragm; the crossed images of the square meet in an oblong with an even upper and lower border, and suspicion is not likely to be aroused, as might be the case when two interlocking circles are seen. I find it best to start such a patient with the coloured squares and
the pointer projecting into the hole. The man is told to look at the pointer, and his eyes are watched by the examiner; when all is ready the test-card is unveiled, and he is asked to state what he sees behind the hole. It would be a remarkably clever, "artful dodger," who would detect the manner in which his vision was being detected by that test. And the pitfalls are so many that an expert would hesitate to attempt a false answer.

For Determining the Absence or Weakness of Binocular Vision.

For this purpose the reading-test with the square diaphragm is good. There is every stimulus to obtain binocular vision. As I have shown in Fig. II, a narrow, middle section of the print is seen by both eyes. Yet those with weak powers fail to keep it steady; they either tend to converge and fuse the two halves, and so shorten the middle syllables, or they tend to diverge and so allow the two halves to separate, with a result that they get reduplication of the middle column of letters.

With these patients the coloured-squares test can be used with the circular diaphragm. To those who converge excessively the circular hole is an irresistible temptation, and they cannot look clear through it to the squares, consequently they can only see the squares ranged one above the other. To those who tend to diverge it is difficult to fix the hole even when the pointer is turned down; they can only see clear through the hole, and the double images of the hole tend to separate more and more, and one may glide away and the image of it be suppressed.

The Physiological Inconstancy of Fixation.

There is one test with which I obtain very curious results. On one half of a test-card is drawn a cross of St. George, on the other half is drawn a cross of St. Andrew; the crosses are placed in such relations that when the
test is in position and the pointer is turned down and fixed by the two eyes the images of the crosses are superposed or fused. Now for a moment the fused images of the crosses present the appearance of a star of eight points; but it is found that this appearance is not constant for the whole time of observation; there appears to be an alternation in the distinctness with which the images of the right and left eyes are perceived by the brain, so that, as though by an electric-flashing sign, the crosses of St. George and St. Andrew pulsate on the screen. The effect is very curious, and the seemingly

![Fig. 15.]

The test of the fusion of the crosses of St. George and St. Andrew.

definite rhythm of the alternation suggests some reason for the phenomenon. The only explanation I can think of is that fixation is not constant, and that unconsciously it falls off, and the fixation is renewed when a fading of the images occurs. The peculiar alternation of the images suggest that first one and then the other eye takes up the duty of fixation. That fixation is never absolutely stable is well known from the appearance of the "after-sun-spots," when the eyes are closed after a momentary glance at the sun's disc.

I suggest that this "diaphragm-test" is likely to be useful to ophthalmologists for several purposes, and that it is not unworthy to be considered one of the tests for the experimental demonstration of binocular vision.

A serviceable form of instrument for the "diaphragm-test" is made by Geo. Culver, Ltd., London, X., at the cost of a few shillings. (December 10th, 1908.)

The President said he had tried the apparatus, and he was satisfied that it was a good and useful test.
17. Some observations on cataract extraction.

By Major R. H. Elliot, I.M.S.

I feel that some explanation, if not apology, would seem to be called for from one who ventures to bring before such an assembly as this a topic so often discussed as the operation for cataract extraction.

Twenty years ago, and shortly after I had qualified as a medical practitioner, it was my good fortune to commence the study of ophthalmology under our President of this evening. Very shortly afterwards I found myself in India—a land, if I may be permitted the expression, flowing with cataract and ophthalmia.

Whilst nearly all forms of eye-disease are rife there, cataract is one of a small group which, for various reasons, presses itself daily on the attention of all medical men who practise in the East. It is not, therefore, surprising that it should "bulk large" in the view of the student of ophthalmology in India. The Government Eye Hospital of Madras is the only special institution of the kind for a population of over forty million people, and its records show an average of about 1000 extractions of cataract a year, all performed by the Superintendent, or, in his absence on leave, by his locum tenens. Naturally, under such circumstances our interest in perfecting the operation for cataract is very great. A great opportunity presents itself to "try all things," and to endeavour to "hold fast that which is good." This, in common with many other Anglo-Indian surgeons, I have steadily endeavoured to do. In the course of some 7000 extractions I have tried and rejected many measures which have occurred to me, either in the course of reading the work of others or as a result of my own reflections.

Time does not permit me to quote extensive statistics, or to take up many branches of the subject, but I desire to bring before you my convictions as to the value of certain measures which appear to me to stand out as more
excellent than the rest. At the same time I do this with every deference for the opinions of other ophthalmic surgeons, and especially for those of the members of this Society. It has been my good fortune to see the operation for cataract performed in nearly every country in Europe, and to discuss its technique with ophthalmologists from America, the Colonies, and elsewhere. The wide divergence of opinion expressed by men whose results are all so splendid makes one feel the need for a very large measure of humility, and if I appear to put the case at all strongly to-night, I would ask you to believe that while I speak strongly of matters on which I feel strongly, I am fully conscious that my great interest in the subject may have more than tinted my views.

Brushing aside all matters of lesser importance, I desire to invite your attention to the following:

1. The antisepsis of the conjunctival sac before operation.
2. The value of lacerating the capsule with a Bowman's needle before making the incision.
3. The dangers of vitreous loss.
4. The value of free irrigation of the capsule as a means of cleansing the chamber, and of replacing the iris, and preventing the impaction of tags of iris.

(1) Lieut.-Col. Herbert, I.M.S. (retd.), has for many years advocated the antiseptic treatment of the conjunctiva before operation. At one time I gave this method a trial, but unfortunately dropped it, discouraged by a few bad results. I therefore confined myself to a careful scrubbing of the conjunctiva by means of cotton-wool swabs mounted on small sticks, and carefully sterilised in the steam steriliser. Even with the greatest care I found that at times I failed to exclude sepsis, and I was therefore induced to give Herbert's method another trial. It is as follows: Some ten minutes before operation the everted lids are exposed for from one to two minutes to a stream of perchloride lotion (1 in 3000). To quote Herbert's own words in a letter he wrote to me: "The perchloride im-
prisons the organisms in the mucus, whose secretion it excites; . . . all mucus and filmy exudation is removed by movement of the lids under a stream of saline fluid, and by a touch with gauze if necessary." It at once occurred to me to combine Herbert's method with my own, and this I now invariably do. An assistant applies the perchloride according to Herbert's directions; the patient comes on the table, and there I swab out the conjunctival sac to its farthest recesses with sterilised wool swabs under a stream of boiled water, poured out of a boiled irrigator. The results have been most gratifying, as may be gathered from the following facts:

The records of the hospital show that in 1897, out of 1161 cataracts extracted there were 98 failures. The figures are taken from my predecessor's notes in the operation register of the hospital:

<table>
<thead>
<tr>
<th>Total No. of cases</th>
<th>Suppuration and panophthalmitis</th>
<th>Suppurative iritis and keratitis not ending in panophthalmitis</th>
<th>Non-suppurative iritis</th>
<th>Unclassified failures</th>
</tr>
</thead>
<tbody>
<tr>
<td>1161</td>
<td>18, or 1.55 per cent.</td>
<td>15, or 1.29 per cent.</td>
<td>38, or 3.27 per cent.</td>
<td>11, or 0.94 per cent.</td>
</tr>
</tbody>
</table>

The remaining 16 are attributed to causes other than sepsis.

In 1902 I published in the *Lancet* the results of 750 cataract extractions, performed in the hospital in 1901 and 1902 (June, 1901, to February, 1902); in these cases swabbing of the conjunctiva was alone relied on for its antisepsis. The following table shows the results so far as sepsis was met with:

<table>
<thead>
<tr>
<th>Total No. of cases</th>
<th>Panophthalmitis supervened in</th>
<th>Suppurative iritis and keratitis not ending in panophthalmitis in</th>
<th>Non-suppurative iritis in</th>
</tr>
</thead>
<tbody>
<tr>
<td>750</td>
<td>3, or 0.4 per cent.</td>
<td>16, or 2.13 per cent.</td>
<td>15, or 2 per cent.</td>
</tr>
</tbody>
</table>

In 1907 I commenced to use Herbert's method in combination with my own. At first a few cases were selected for it out of each batch, but later the combined method was adopted as a routine for all cases, and is so to-day.

From October, 1906, to October, 1907, I extracted 725
somes observations on cataract extraction. 65
cataracts, of which the eyes had been prepared for opera-
tion in this way. The results are as follows:

<table>
<thead>
<tr>
<th>Total No. of cases.</th>
<th>Panophthalmitis supervened in</th>
<th>Suppurative iritis in</th>
<th>Supacuta iritis in</th>
</tr>
</thead>
<tbody>
<tr>
<td>725</td>
<td>1, or 0.13 per cent.</td>
<td>1, or 0.13 per cent.</td>
<td>6, or 0.8 per cent.</td>
</tr>
</tbody>
</table>

In the case of panophthalmitis, the lacrimal sac of the
same side had been submitted to operation by another
surgeon. It was unfortunately assumed that the extirpa-
tion had been complete, an assumption which a careful
examination subsequent to the disaster proved to have
been ill-founded. Of the cases in which I have myself
extirpated the sac and subsequently removed the lens, all
have done well, and it was a confidence so engendered
which led me to assume all was well in this case. I cannot
sufficiently regret that I was not more sceptical. Subse-
quent to the 725 cases we are considering, I was able, on
February 20th, 1908, shortly before leaving India, to
complete a tale of one thousand consecutive cases operated
on for cataract after treatment by the combined method
of cleaning the conjunctival sac. In this series there had
been only one case of suppuration of the eyeball, the one
already spoken of, and 691 consecutive cases of extraction
had been performed (since the one case of panophthal-
mitis) without a failure.

One cannot but reflect with pity and regret that even
that one failure might so easily have been avoided, and
that it was in no sense to be debited to the methods
employed.

The case of suppurative iritis ended in loss of vision.
The six cases of subacute iritis were of a mild type, as
may be gathered from the final vision of five of them,
which was respectively $\frac{3}{30}$, $\frac{5}{20}$, $\frac{5}{20}$, $\frac{5}{15}$, and $\frac{6}{5}$. The sixth
case occurred in a young man suffering from congenital
cataract, and the final vision was only hand movements. It
seems likely that this result was due more to antecedent
fundus changes than to the inflammatory condition. In any
case, it will be observed that only in 3 (0.42 per cent.) at
the outside can sepsis be held accountable for loss of vision,

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though it may have lowered the final result attained in our others.

It will be observed that the percentage index of septic incidence after extraction, which stood at 7 in 1897, fell to 4.53 in 1902, and to 1.06 in 1907. If the index of sepsis destructive to vision (i.e., responsible for a vision of less than \( \frac{5}{30} \)) be taken for 1907, it stands at from 0.275 per cent. to 0.42 per cent., according as we count in the doubtful case above alluded to or not. I lay some stress on these figures, because the claim has been urged that the extraction of a lens in its capsule removes the element of danger from inflammation, which we must encounter if we leave the capsule behind. I hold most strongly that deep-seated inflammation of an eye after extraction is due to sepsis, and that to take any other view of the case is a mere "burying of one's head in the sand." I would wish to be clearly understood that in so saying I make no reflection on the operation of extraction within the capsule, towards which operation I have always kept an open mind, and which I leave with confidence to the test of time and experience.

I would wish to add that my locum tenens, Capt. Kirkpatrick, who follows my method closely, writes to tell me that he has had only one suppuration since I left India in his extraction practice. I should greatly like an expression of opinion from this Society on the subject. To my mind, the method of operating is of little consequence. You may do a combined or a simple operation. You may tear your capsule with forceps, with sickles, with a knife, or with a needle; you may remove the lens in its capsule or not; these and many others are secondary matters, but the hub of your wheel of success is your attention to asepsis and antisepsis.

I shall not weary you by discussing matters on which we shall all probably be in agreement, such as the sterilisation of instruments, solutions, dressings, etc., or the treatment of the lid-edges and of the surrounding skin.
As to my second point:
(2) Nowhere out of India have I seen the capsule lacerated with a Bowman's needle before the incision is undertaken, and yet for precision and safety in effecting the needling, for the easy sterilisation of the instrument used, and for the valuable information thus acquired as to the consistency of the cataract, and the size of its nucleus (enabling us to graduate our size of incision accordingly), this method has to my mind no equal.

During the past four years a great deal of trouble has been taken in the Madras Ophthalmic Hospital to study the appearances of cataractous lenses before operation, and to compare these observations with the physical properties of the same lenses after extraction. As a result, we have been able to define a certain number of classes of lens, and we can in many instances foretell with confidence the class of lens we are going to meet with, and the special treatment it will require for its complete extraction. At the present time we are making a systematic effort to render this classification more reliable and more precise. In this scheme the needle plays an important part, being used practically as a probe as well as a lacerator of the capsule. If one enters one's knife point in the needle puncture it will slip through more easily and fill up the hole at the same time, thus avoiding aqueous leakage.

For tearing the capsule one should choose a needle (Bowman's), the shank of which is at least as big as the blade. Too large a blade means too large a cut, and consequent leakage alongside the needle during laceration. Such an accident need never occur with a suitable needle.

I emphasise this point because I have heard the leakage urged as an objection to needling. It is entirely preventable. But should the chamber empty during needling, owing to the blade of the needle being too large, or to the knife having been introduced with its edge the wrong way, or to any other cause, the chamber may
be easily refilled, through the small puncture already made, by pressing the nozzle of an irrigator against the puncture, and turning on the stream. One is at once placed again in the position of working with a full chamber.

Again, I have heard surgeons object to the early needling in the operation for Morgagnian cataract on the ground that such a procedure makes it impossible to see the knife-point. In Madras, where patients wait years after blindness has supervened, Morgagnian cataract is so common that I sometimes operate on four or five cases on one Saturday morning. I always use the early needling, and never find any difficulty in directing my knife-point, which can quite easily be seen at the edge of the chamber.

It is curious that this class of cases should have been brought under objection, for it is precisely the one in which I find needling more than usually valuable, and that for two reasons: (1) The fluid escaping from the lens naturally deepens the chamber, which in Morgagnian cataract is often very shallow, and so gives lots of room for the knife to pass across without entangling itself with the iris or cornea; and (2) the size of the nucleus can be easily gauged by noting the amount of the fluid which escapes, and a very shrewd estimate can thus be made of the size of the incision necessary for its delivery. It is a dictum with me that "too large an incision is a mistake, but too small an incision is a crime against the eye," and I always endeavour to so graduate the size of my incision that the lens will just escape without difficulty, and no more. There is more to be said in favour of needling. It enables the operator to take his time with the laceration of the capsule. There is no fear of his instrument becoming entangled with the iris. He is working on an eye with normal tension, and with the normal supports of the organ still uninterfered with by the section. He can cut out a piece of the capsule if he fancies such a procedure. He is working with a cutting edge, and not with the point alone (I emphasise this), and in the case
of a hard lens he can utilise that edge to the full by sliding his point in under the capsule and then cutting up, and so avoiding all pressure on the suspensory apparatus of the lens. With any moderate care and skill he runs no risk of dislocating his lens in the way that a surgeon working with capsule forceps appears to me to do. I have freely tried laceration of the capsule with the knife in crossing the chamber, and I think the preliminary needling vastly superior to it, inasmuch as it is more deliberate, more accurate, and therefore, to my mind, more scientific.

My third point is:

(3) The danger arising from loss of vitreous occurring during the operation for extraction of a cataract.

You are possibly aware that a somewhat animated controversy has recently raged round this point in the columns of the *Indian Medical Gazette*. It arose over the question of the advisability of removing all, or at least the majority of cataracts within the unbroken capsule. This method has been looked on with suspicion by a large number of Anglo-Indian surgeons, including the three Presidency ophthalmic surgeons of the time, *viz.*, Lt.-Col. Herbert, Lt.-Col. Maynard and myself. My own view, which I believe is shared by those two surgeons, and by many others, was expressed in the following words in the *Indian Medical Gazette* of December, 1906:

"Our opponents may urge, and with some show of reason, that since we so greatly dread the consequences of vitreous escape, we ought to produce our own statistics as a justification for our fears, and to prove therefrom that our apprehensions of the ill-consequences arising from vitreous escapes are well founded. I would answer this objection in a two-fold manner—(1) positively and (2) negatively. To take them in this order: (1) We know that the European ophthalmologist has a very decided dread of the after-consequences of vitreous escape. Unless we have a very strong case to go on, we feel that it would be injudicious to go against what we believe
to be a widespread and deeply rooted ophthalmological opinion. (2) Negatively we have to acknowledge that up to date our own statistics give us too little to go on, for the simple reason that with our present methods the percentage of vitreous escapes is so very low that there are comparatively few of such cases to return and be seen again. It is not even as if one could count on one's failures invariably coming back to the original operator. I see other people's failures from all parts of India, and I do not doubt that others see mine. It is only natural that this should happen. Seeing, then, that our vitreous escapes are few in number; that probably only a percentage of them culminate in blindness (we will presumably all concede this); and that of these only a percentage are likely to come back to the surgeon who did the original operation, it is obvious that our difficulties in the way of settling the question are great. At the same time it is henceforth our obvious duty to lose no opportunity of carefully investigating the cause of failure in each poor or blind eye we meet with after extraction."

A further light will be cast on this subject by a later quotation from the same paper. I there say:

"In my last paper I quoted the vitreous loss in 200 cases. In 170 cases the capsule was left untouched, with a percentage of vitreous loss of a little under 3 per cent. In 30 cases, where the tags of floating capsule were removed, the loss rose to five, or 16.6 per cent. (on the 30).

I then reviewed the results of some well-known and able Anglo-Indian surgeons, who had tried intra-capsular extraction, and showed how greatly their figures for vitreous loss whilst using that method exceeded mine, in the course of the ordinary method of extraction. I closed this part of my subject with these words:

"It is to be borne in mind that my figures represent a percentage within a percentage. They do not show my vitreous loss on the 200 cases, which, even with this element thrown in, was only 5 per cent., but on the thirty cases out of that 200, in which it was found
necessary or advisable to remove floating fragments of capsule after extraction. It is, however, the series of thirty cases which comes within the arena of this argument. Even so, a comparison of my figures with those of skilled surgeons, such as Major Birdwood or Captain Oxley, appears to support my contention, which was, that, 'Though this manœuvre (the removal of obvious floating pieces of capsule after extraction of the lens in selected cases) has thus doubled my vitreous loss, it is to be taken into account that it is a much less dangerous proceeding than the expression of a lens in its capsule.'

I have brought this subject forward this evening with a view to obtaining the views of the members of this Society as to the gravity of the risks incurred by losses of vitreous during extraction. We know well that in a large proportion of cases the early results are good. Will members give us their personal experience of the later course of such cases? Though I took up the cudgels against the advocacy of intra-capsular extraction, I did so, not against the operation itself, but against the methods of its advocacy. If it is really a safe operation in the long run, there is, indeed, much to be said for it. My own data are insufficient, and I think Herbert and Maynard will say their's are too. I doubt not that you can shed much light on this question, and I shall be most sincerely grateful if you will do so.

I now come to my fourth point, viz.:

(4) The value of irrigation in the toilet of the capsule and iris. For the removal of cortex M'Keown's method of intra-ocular irrigation has given me far the best results of any method I have tried, and I have had ample opportunities of using those commonly advocated. It is safe, clean, and effective, provided that the most ordinary care is taken, and after using the method on over 5000 cataracts it appears to me to be the one important step in advance made recently in the treatment of cataract. Be the nature of the cataract what it may, irrigation is always of value. In the case of lenses with soft cortex
the chamber is washed clear in a few seconds. Brittle edges and doughy masses are washed out clean and unbroken, sticky cortex becomes quickly manageable, and can be removed in large part at least, whilst in not a few cases the whole lens (cortex and nucleus), can be washed out without the need of using any other instrument than the irrigator, once the iridectomy has been performed. Blood and débris can be washed out, leaving a clear chamber and an easy view of details. The iris and any capsule tags can be washed back into place, and all this by the means of one instrument, which can be safely and easily sterilised, viz., a silver nozzle carrying a stream of sterile normal saline solution. There need be no introduction far into the chamber of hard instruments, which threaten the integrity of the eye should the patient make a sudden move. The even, steady, hydrostatic power sweeps away all removable matter and gently completes the toilet of the eye. Even if you pass the nozzle well into the chamber, you have still little to fear from a sudden movement, for the chamber is distended with fluid, and your instrument point lies free in this fluid in the middle of the chamber, and is not in actual contact with the neighbouring membranes of the eye.

I am aware that some surgeons have had very bad results whilst giving irrigation a trial. Sepsis has freely supervened, and they have blamed the method. Is this fair? I have shown you that in my last 1000 cases I had one suppuration (clearly traceable to an overlooked suppurating sac), and that the percentage index of loss of vision due to sepsis worked out in the first 725 cases of that series was from 0·275 to 0·4 per cent., according as we admit or exclude one doubtful case, whilst the total percentage index of septic incidence was 1·06 per cent. I have told you that Captain Kirkpatrick, closely following my methods, has had almost as good a series, and I believe you will now readily accept my contention that there is strong reason to believe that the method of intra-ocular irrigation, which I advocate, does not introduce any
element of danger of sepsis into the operation. If operators are dirty or careless, let them attribute the evils which follow to their dirt or carelessness, but not to this method, which deserves no such treatment at their hands.

I shall next deal with the methods of using the irrigator, which are many and various. In so doing I shall describe also some manoeuvres I at times employ in connection with irrigation.

(1) Irrigation under the lower margin of the iris, by depression of the nozzle of the instrument after its introduction within the chamber. This serves to wash out débris of lens from beneath the membrane, and for the latter purpose is a regular step in every operation. The iris bellies out, and is often carried bodily towards the lower part of the chamber, and away from the incision. Not infrequently, however, the opposite effect is produced, and the iris is washed out with the cortex, so that its edges prolapse in the wound.

It is not necessary to introduce the nozzle far into the chamber in the great majority of cases. Where, however, there are doughy casts or sticky masses which do not quickly yield to the fluid, I do not hesitate to introduce the nozzle freely and in various directions till I find how best to dislodge the recalcitrant masses. If necessary I employ a strong stream, directed close on to the masses, for the purpose. It is not to be supposed, however, that many cases require such treatment. They do not. Such a manoeuvre is the exception and not the rule.

(2) If the above manoeuvre fails to result in the return of the iris, the nozzle of the irrigator should be withdrawn 2 or 3 mm. length from the incision, and a gentle stream of fluid should be directed on the lips of the wound from outside. The chamber will often fill at once, and the iris fall into place almost with a snap.

If reposition is still unaffected, it may be safely assumed that one of two things has happened. Either the iris is caught in the angles of the wound, or (and more commonly still) the membrane has been folded on itself and
fixed there. If the hold of the iris-forceps on the membrane is a very light one, and if the iridectomy is quickly and cleanly made, it will very rarely happen that the pillars of the coloboma become impacted in the angles of the wound. The folding of the iris on itself is a much commoner complication, and one which it is often difficult to avoid. What happens is that the iris becomes thrown into folds during the exit of the lens, and that these folds become, as it were, gummed together by the sticky lens substance, or by some other means more difficult to explain.

I frequently demonstrate to medical visitors and to students the unfolding of those iris creases under the influence of irrigation. One can watch the folds open and the iris returning to its place with an action that suggests the unfolding of a fan, or that of a theatre curtain as it is dropped. This leads me to the third method, which consists of—

(3) Irrigating with the nozzle within the chamber, the stream being directed over the anterior surface of the iris, instead of below it. One can reinforce this method by at the same time gently stroking the iris in the required direction with the flat of the irrigator nozzle. It will sometimes happen that an iris will tend to prolapse on the use of even the gentlest stream. This seems to be due to want of tone, and often yields to the steady flow of the fluid, the tone appearing to be regained. If this is not quickly the case, I proceed to the next method, viz.:

(4) The reposition of the membrane with a curette, after seeing the chamber has been washed clear. If all these means fail, and it is but seldom that they do so if properly used, there remains yet a very valuable method, viz.:

(5) The replacement of the iris edges by seizing each in turn with carefully introduced iris-forceps, and pulling them in the required direction. A sharp key-hole pupil at once succeeds to the bow-like curve of the iris, or to the upward displacement of the pupil, either or both of which indicate a failure in proper replacement of the membrane.

It will seldom be difficult to replace the iris satisfactorily.
SOME OBSERVATIONS ON CATARACT EXTRACTION.

if one is careful during iridectomy (1) not to pinch the portion one picks up in the forceps, and (2) not to draw the membrane tightly into the sclero-corneal section. If the iris is pinched the patient experiences pain, and in consequence, shrinks away, and by so doing draws the iris tight into the corners of the section. My routine practice is to perform an iridectomy in every cataract extraction; a small piece of the pupillary margin of the iris is seized gently and withdrawn from the chamber without tugging on it at all; a section is then made with scissors at right angles to the wound, a small segment being cut off cleanly with one cut. If the membrane does not at once fall back into the chamber in its proper position, I wash it back with the irrigator before commencing extraction; this saves such a case from impaction of the iris in the wound-angles. If the iris does not readily return, I use the nozzle of the irrigator as a curette to replace it, or introduce a curette for the purpose. In some cases after delivery of the lens, it may be found that the iris will not wash back; on carefully examining the wound, the cause of this may be at once revealed in the impaction of a mass of cortex under the scleral lip of the corneo-scleral wound. Such a mass will most often be left behind when we are dealing with lenses, in which a firm nucleus is surrounded by a layer of cortex of a doughy or cheesy consistency. As the lens dislocates upwards, and tilts its upper edge forward for delivery, it may happen that the edge of the nucleus more nearly corresponds with the gap of the incision than does the actual upper edge of the lens. A fracture takes place in the lens at the level of junction of the nucleus with cortex above, and the upper margin of the nucleus becomes the leading point of the delivering lens, whilst the brittle cortex strips off above and remains impacted under the scleral edge of the sclero-corneal wound. I think I am right in saying that the capsule is invariably left behind at the same time. Very often when the latter cannot at first be seen, it is at once obvious on filling the chamber
with fluid from the irrigator. The mass of impacted lens-matter may in very many cases be easily got rid of, and the iris at once replaced by the following manœuvre:

(6) The irrigator nozzle is turned round so that the stream is directed backwards, or even upwards and backwards, thus playing direct on the impacted mass. Very often this at once suffices to wash out the obstruction, and the iris easily slides into position, as soon as the stream is again directed over its surface in the usual direction. If this is not the case we may next proceed to another manœuvre, which, however, requires considerable skill.

(7) A pair of iris forceps being introduced into the chamber, one seizes a portion of the capsule, which can be seen hanging down into the chamber, and draws it towards the centre of the pupil. By so doing, one everts the shallow bag formed by the upper cul-de-sac of the capsule, and thereby empties its contents (cortical matter) free and loose into the chamber, from which they can be easily washed out. At once the impaction of the iris is relieved, and the membrane can be easily returned into position. Very often, when the impacted capsule cannot easily be seen, one can, as already stated, render it patent by gently irrigating the chamber; the flapping fragments of the torn capsule at once become obvious, and are easily seized. In recent extractions I have been resorting to this manœuvre with increasing frequency.

When dealing with Morgagnian cataracts, or with cataracts in which the cortical matter is abundant and soft, it is my practice to wash out the whole lens (nucleus and cortex) with the irrigator. As soon as the iridectomy has been made, all instruments save the irrigator (the speculum excepted) are laid aside, and a stream of fluid is directed into the chamber behind the lens. At once the whole cataract washes out (nucleus and cortex) with the least possible disturbance to the eye, for the even hydrostatic pressure reduces the risks of delivery to a minimum. It is a most fascinating manœuvre to perform or to watch.
Irrigation as a Routine Step in Iridectomy.

For whatever purpose an iridectomy is undertaken I invariably use the irrigator as a matter of routine. As soon as the iris section has been made, the nozzle of the instrument is introduced and a gentle stream is poured into the chamber. At once the iris falls back into place, and blood is easily and quickly washed out. Moreover, as in cataract extraction with the use of the instrument, the operation closes on a full chamber, and enables the surgeon to see that his section edges are in good apposition. It is no small advantage to substitute the even hydrostatic pressure of a sterilised normal saline solution for the introduction into the chamber of various instruments. Need I dilate on the advantage of working with a clear chamber, which permits every detail to be easily seen, instead of groping to replace an iris which is hidden by a quantity of blood?

Cleansing a Cloudy Chamber.

It not infrequently happens that during the course of an extraction lens débris or blood escapes into the chamber, and renders the field of operation obscure. If the lens presents easily, this is not a matter of much consequence, as the disturbing matter is usually expelled with the cataract; unfortunately this clouding of the chamber is most likely to occur when, for one reason or another, the lens is not easily expelled. It is one thing to have to decide what it is best to do when the details are thus obscured, and quite another when, by means of a few seconds' irrigation, the whole field lies plain before one. It can at once be seen, for instance, that the section has been made too small, or that the lens has dislocated upwards and laterally, and these defects can at once be remedied, with all the advantages attendant on clear vision. It seems hardly necessary to point out that if the
lens happens to have dislocated upwards and inwards, or upwards and outwards, the direction we will have to apply our pressure to replace it, before delivery can be effected, will be different in the two cases; to attempt to replace it in the wrong direction will only lead to disaster, whereas a few moments suffice with properly applied pressure (personally I usually use a needle for the purpose) to right the dislocation, and clear the path for easy delivery.

The longer I use irrigation the more new ways of using it do I find. The charm of it never lessens, and the pleasure with which one watches the cortex clearing away from the chamber and leaving a clear black pupil behind is ever new. It often reminds one of the way in which a mist clears before sunlight. (December 10th, 1908)

The President said the Society was much obliged to the author for having brought forward those observations. No temerity need be felt in doing so, as members were always glad to hear remarks on the subject, particularly from those who had had such a large experience in India. Major Elliott had divided his paper practically into four parts: (1) The means of preparation of the conjunctival sac; (2) the method of previous cystotomy; (3) the question of the loss of vitreous; (4) the use of irrigation. He had asked whether there were any experiences as to the late results of cases where vitreous had been lost. He remembered one case in which a good deal of vitreous was lost: it was about the first operation he did in private. The eye was very myopic, and immediately after making the incision the lens disappeared. The iridectomy was made with the vitreous presenting. He recovered the lens easily with the scoop, fortunately, as it was practically the woman's only eye. She lived for about twenty years afterwards, and retained perfect vision to the end. There was no damage. It was not often one had an opportunity in out-patient work of following up cases of loss of vitreous. When he was house-surgeon it used to be taught that many such cases got retinal
detachment later, and that others were liable to get optic nerve atrophy afterwards. But he, like Major Elliott, felt he did not know enough about the ultimate results of loss of vitreous. Some of the best results as regards vision were seen in those who lost a little vitreous. If the loss of vitreous was due to much pressure on the eye, either by the surgeon or by the patient, he thought the outlook was bad, but if it were lost in a comparatively passive way he did not think bad results commonly followed.

Mr. Jessop asked whether Major Elliot put his needle horizontally through the cornea, or at the periphery, or between the periphery and the centre?

Mr. Johnson Taylor asked whether Major Elliott dilated the pupil beforehand. Colonel Drake Brockman, at the Lucerne International Congress, told him privately that he always, in connection with cataract extraction, dilated the pupil, and made a preliminary capsulotomy with the needle.

Mr. Holmes Spicer said he thought that the question of damage from loss of vitreous depended largely on whether the vitreous was healthy or not. It was serious to lose healthy vitreous, but not so serious to lose vitreous which was not healthy. That was probably another way of saying what the President had stated on the subject. The reason, he thought, was that if the vitreous was quite fluid nothing remained attached to the wound; but, if healthy, it was a definite structure, which remained in the wound, and caused all the evil consequences of anterior adhesion.

Mr. Lang asked what was the condition of the conjunctiva after the cleaning process, i.e. in regard to the epithelium. If stained with fluorescein, would the surface be intact everywhere? Also, did he anaesthetise by cocaine?

Major Elliot, in reply to the questions, said any kind of capsule incision fancied could be made, and in any desired position. There was plenty of time as the aqueous
remained in. The needling could be done without leakage, if the shank of the needle were not smaller than its blade. He used a very fine needle with a cutting blade. He needled at the point where he was going to enter his knife afterwards. As a routine measure he put in a drop of atropine forty-eight hours before. But if there was any suspicion of raised tension he omitted that, putting in homatropine an hour before. In reply to Dr. Bronner, he now invariably performed an iridectomy. It was not the case that irrigation was impossible if iridectomy was not done, though with it the irrigation was rendered easier. He had tested the question of visual result by carrying out the combined operation on one eye of the patient, and the simple on the other eye in a series of cases, and the result had been very slightly in favour of iridectomy. He made a very narrow iridectomy at right angles to the incision. He always felt sceptical when he heard about "a small loss" of vitreous. The intra-capsular operation was a magnificent one if one could exclude danger from the greatly increased percentage of loss of vitreous. Major Smith had contended that the risk of sepsis was diminished by the intra-capsular operation, though his own view was it would be greatly increased. In reply to Mr. Lang, he said the condition of the conjunctiva for a day or two after operation would sometimes frighten the operator. On rare occasions the exudation made the lid and ocular conjunctiva stick together. Lieut.-Col. Herbert's advice to him in such cases was to leave it alone, and he had done so, with the result that in a few days it had absorbed and done no harm. He pointed out that in Madras the operations were carried out in a land of sepsis and dirt, and the patients were very apt to put their fingers to their eyes just before operation, and in face of that he thought it remarkable that 1000 cases had been done with one (avoidable) suppuration. As a result of the perchloride irrigation and swabbing the cornea was not infrequently abraded, and probably would stain with
fluorescein, and for a few days afterwards it was sometimes cloudy, but in none of his 1000 cases could he trace permanent corneal opacities to the method. He irrigated the eye first, then used cocaine, which had been boiled, and which was dropped in at intervals for five minutes. He used the 4 per cent. solution. He did not use adrenalin.

18. Iridectomy in cases of acute iritis in which the pupil cannot be dilated.

By Adolph Bronner.

It has, I believe, always been taught that it is dangerous, under any circumstances, to perform iridectomy during an attack of iritis, as long as there is any inflammation present.

Jessop (text-book) says: In recurrent iritis with posterior synechiae iridectomy may be performed, but should never be performed during an attack.

Fuchs.—Iridectomy is not performed while inflammation still exists, save in very exceptional cases, since if we perform it, then we should have reason to fear that the newly-made pupil would be closed up again owing to a continuance of the exudative process.

Berry.—Iridectomy, when performed for iritis, should be done at a time when all symptoms of inflammation have subsided.

Nettleship is, I believe, the only author who advocates iridectomy in these cases: "Iridectomy is needed for cases of severe iritis, even when there is no increase of tension." Up to two years ago I quite believed in the old theory. I had then, however, seen many cases of iritis of my own, and those of other surgeons, in which extensive adhesions had remained, the anterior lens capsule become opaque, and the vision impaired by increased tension, and I felt
sure that in many of these cases useful vision could have been retained if iridectomy had been performed during the attack of iritis.

(1) In July, 1906, I was called to see a case of iritis in a man, æt. 45 years. He had had severe pain in the right eye for ten days, and had been treated in the usual way with atropine and salicylate of soda. There was severe pain in eyeball and forehead. Much ciliary congestion. The iris was discoloured and thickened, and the pupil only partially dilated. Aqueous slightly hazy. Vision about $\frac{6}{6}$. Much against my convictions I decided to try iridectomy. To my great surprise the next day there was less pain, in three days the pupil was widely dilated, and in ten days the vision was $\frac{6}{6}$. He had been using atropine, and taking iodide of potassium since the operation.

(2) In December, 1906, I saw a woman, æt. about 48 years. She had had severe pain in the right eye for three days. The family doctor thought it was glaucoma, and gave eserine. It was a typical case of iritis. I ordered atropine ointment, 2 per cent. every two hours, dionine, leeches, and salicylate of soda. In three days the pupil was only partially dilated, and there was much pain and redness. I performed iridectomy. The next day there was some blood in the anterior chamber, and still much pain. On the second day the blood was partially absorbed, and there was less pain, and to my great surprise I saw that the pupil was nearly fully dilated. There was an uninterrupted recovery.

(3) In November, 1907, I saw a man, æt. 25 years. He had had pain in the left eye for six to seven days. It was a typical case of iritis, with contracted pupil. He had not been under treatment. There was a specific history. I ordered atropine ointment, 2 per cent., potassium iodide gr. xx, and mercurial inunctions. In four days the pupil had not dilated. I then performed iridectomy. In this case there was severe pain for three days after the operation, and much chemosis. The
aqueous was hazy, and there was much lymph in the coloboma. The symptoms then rapidly subsided, and in five days the pupil was three-quarters dilated, with one or two small synchiae. The reason why in this case the pain continued for a few days was, I believe, entirely due to the fact that the patient had never undergone prolonged anti-specific treatment.

(4) Man, æt. 50 years, was seen February, 1908. Pain in right eye for ten days. Last three days patient had been using atropine. The pupil was not dilated. The iris was discoloured and thickened. Atropine ointment was used every two hours, hot fomentations, leeches, aspirin. After six days the pupil was only half dilated; vision was worse, 6/6. The tension was slightly increased. I performed iridectomy, and pulled out the iris as far as possible to break down adhesions. There was pain for two days and some hyphæma. Afterwards an uninterrupted recovery. There was a rheumatic history in this case.

(5) In June, 1907, I saw a woman, æt. 30 years. She had had severe pain and redness of left eye for two weeks, and had been using atropine. The pupil was only slightly dilated, the iris thickened and discoloured. Vision about 6/21. I performed iridectomy. There was much pain for a few days, the aqueous was hazy, and there was a good deal of lymph covering the coloboma. I found that there was a well-marked, specific history. I ordered large doses of potassium iodide and mercurial inunctions. In five days there was decided improvement. The lymph eventually disappeared, and the vision improved. This case shows that in all these cases general treatment must be most carefully and energetically carried out if we want to secure good results.

(6) In July, 1908, I saw a man, æt. 50 years. He had had severe pain and redness of the left eye for two weeks, with much ciliary congestion. The iris was thickened and the pupil small. There was well marked eczema of both lids, and much pain and photophobia. It was
evidently a case of iritis and atropine eczema. I ordered 1 per cent. duboisine drops. In four days the pupil was partially dilated, and there was still much pain and photophobia. Vision, $\frac{6}{6}$. I performed iridectomy. In two days there was marked improvement, and the pupil eventually became fairly well dilated.

I have often noticed that in cases of atropine dermatitis the pupil does not dilate, as if the atropine did not act as a mydriatic at all, or at least not as powerfully as in ordinary cases. On this subject I should like to hear the opinion of the meeting.

I should suggest that iridectomy be performed in all cases of ordinary iritis, in which, under the usual treatment—2 per cent. atropine ointment every hour, dionine, hot fomentations, leeches—the pupil does not dilate in four to five days, and the aqueous becomes hazy, or there is the slightest increase of tension, and much dimness of vision. Also in all cases of iritis with atropine eczema. It does not matter if there is any inflammation present, or not. There is often a specific history, in which cases large doses of potassium iodide should be given, and mercurial inunctions used, if possible before, or at least at the same time as, the operation. If there is a rheumatic history aspirin and potassium iodide should be given freely. If this is not done you get well marked exudation, such as you get if you perform iridectomy in cases of serous iritis and sympathetic opthalmia.

The iridectomy causes decrease of inflammation and pain, often breaks down fresh adhesions, prevents secondary glaucoma, and opacities of the anterior capsule. It also, in many cases, prevents recurrence of iritis, with subsequent impaired vision. I, of course, exclude all cases of serous iritis and sympathetic opthalmia, as we know that in most of these cases you get excessive exudation, and often cyclitis, after an iridectomy.

The cases I refer to are not very common, and in a large number the patient refuses operation, so that the every-day experience is not very large. I have brought these
cases before you in the hope of hearing the experience of others. The subject seems to me to be one of very great practical importance. Opinions evidently differ very widely, and a definitive decision, whether it is right or wrong to perform iridectomy during an attack of iritis, can only be arrived at by the experience of many surgeons. 

(December 10th, 1908).

Major Elliot said he had been profoundly dissatisfied with the operative treatment of iritis, so much so that he had looked round for some other expedient, and he had found it. He had had admirable results from free sub-conjunctival injections of normal saline fluid. They had produced dilatation of the pupil and abatement of the symptoms, when he was at his wits' ends to know what to do. He repeated these injections twice a week, or in severe cases daily at first.

Mr. C. Wray said that before resorting to operative treatment in iritis, one must carefully consider what the painful iritis was due to. He believed a very painful iritis was due to the action of toxins in the anterior chamber. It was equally true that the effect of toxins on epithelial tissues was to make them swell. If there was a swelling of epithelial cells in Schlemm's canal, obviously the circulation of aqueous must be interfered with. Normally it was very slow, and it should be a part of the treatment to make up for that slow movement. In hospital his rule was to first try with atropine and cocaine. He gave the patients printed directions, of which he handed round copies, telling them to drink large quantities of water containing acetozone, and take a good deal of exercise. Three weeks ago he saw an officer of artillery, who had been treated by the recognised methods, and was brought to him by his brother, a medical man. The pupil had refused to dilate. He came on Thursday; on Saturday his pupil was well dilated, and he was apparently well. He went back to his regiment in nine days. About a week later, after he had given up acetoz-
zone, he had a recurrence in the other eye. He was again put on treatment, and he as quickly recovered. Tubercle could be excluded, and there was no history of syphilis.
APPENDIX.

The following cases and communications have also been brought before the Society:

2. Crateriform Hole in the Optic Disc, S. Stephenson.
3. (a) Drawings illustrating Connections between Circular Iridis Major and Schlemm's Canal, traced in serial sections. (b) Colour Micro-photographs (Lumière process) illustrating the same connections, Thomson Henderson.
4. Discoid, or "Coppock" Cataract, L. Paton.
19. **On the operative treatment of strabismus.**

By G. Brooksbank James.

Ophthalmologists seem to be fairly well agreed at the present day as to the advantages of early orthoptic treatment in cases of strabismus, and recognise that a considerable proportion of the subjects of this peculiar anomaly can be cured by this means alone. But even when such cases are seen at a very early age, and when every care is taken with their treatment, a certain number remain intractable to such palliative measures, and require the further aid of surgery for their cure. With regard to the further questions, *i.e.* the age at which operation should be undertaken and the kind of operation which should be performed, the verdict of the profession is not unanimous. Many surgeons still prefer the single or the double tenotomy in the minor and medium degrees of convergence, and perform in addition an advancement of the external rectus of the deviating eye when the squint exceeds a certain degree. So far as I am aware, the single or the double advancement operation in convergent strabismus, though strongly advocated by a few continental surgeons, has been received with little enthusiasm in this country, where surgical practice with regard to this matter has remained much the same during the last twenty-five years. For some time past I have devoted a good deal of attention to this subject, and have been led by reflection and experience largely to abandon the operation of tenotomy, as I fully recognise the objections which there are to it when practised by the ordinary subconjunctival method and followed by the usual careless after-treatment. I recognise that it is advisable and, indeed, necessary in certain cases to divide a tendon, but I think greater care should be exercised in this proceeding, and that it should preferably be done by the open method when the tendon
can be permitted to retract as far as safety may suggest, and then be attached to the sclera in the new position by a stitch. Both eyes should then be bandaged, and the patient kept in bed for six days. This seems a formidable after-treatment for so simple an operation, but the results would be more certain and more permanent, and would be more in keeping with the principles of orthopaedic surgery. In the following remarks I shall refer more particularly to the advancement of the external recti in cases of convergent strabismus.

I have now performed this operation in a considerable number of cases, and as I possess full notes of twenty which are immediately available I purpose to analyse them. These twenty cases were treated by a single advancement of the external rectus of the deviating eye, and show what can be done by this operation alone in many cases of squint. The patients had all been submitted to orthoptic treatment for some months or years prior to operation, and the quality of their binocular vision had been accurately ascertained. The ages ranged from five to seventeen years; only two were below the age of seven. Of these twenty cases 6, or 30 per cent., may be considered complete cures. Two of the six are of particular interest. In the first case, a girl, set. 17 years, there was a convergence of the right eye of 20° combined with 7° of right hyperphoria. The history of the patient and photographs showed that the squint had developed much later than is usually the case, it having existed for only seven years. No definite cause could be assigned. The lateral movements were good; both the convergence and hyperphoria were concomitant. The visual acuity in each eye = $6/6$, and the patient was wearing and had worn for six years glasses which accurately corrected her hypermetropia, i.e., $+3$ D. sph. in each eye. Tested with the stereoscope with the objects suitably arranged for fusion, she showed no power of relaxing her convergence in the interests of binocular vision, nor any appreciation of the third dimension. Advancement of
the right external rectus was performed. The effect two weeks later as regards the convergence anomaly was excellent, but the hyperphoria remained exactly the same, i.e., 7°. I found it impossible to procure fusion at varying distance by means of prisms, and hit on the following expedient: A long white stick was placed upright in front of a dull background and the patient was instructed to look steadily at the centre of the stick and practise fusion at varying distances without taking any notice of the extremities of the object. After about a fortnight's practice she was able to correct a considerable part of the hyperphoria, and within another ten days the vertical diplopia which had troubled her disappeared and I found that she possessed perfect binocular vision for all distances. She has since been able to read, etc., with perfect comfort.

The second case was that of a boy who came under observation, five years prior to operation, for interstitial keratitis. The attack fell chiefly on his right eye, and for a time greatly impaired its vision. Convergence of 20° ensued in this eye and persisted, despite every effort made to improve the visual acuity and restore binocular vision. The visual acuity improved to \( \frac{6}{12} \), but the convergence remained the same for four years, when I finally decided to advance the external rectus of the affected eye. Cure soon ensued, and he now enjoys full binocular vision with accurate appreciation of the parallax at all distances.

These cases are somewhat anomalous, but they show that the convergence innervation may be upset at a fairly late age, and even in the presence of an accurately corrected refractive error and at a time when binocular vision has been thoroughly established. There is no question of paresis here; the boy suppressed the false image with the same facility as is shown by the ordinary squinter, while the girl was only troubled by diplopia on account of the associated hyperphoria. Of the remaining four cases in this first group who may be considered cures two are patients in whom the visual acuity in each eye = \( \frac{6}{6} \) or \( \frac{6}{9} \), while in
two cases the originally deviating eye remains amblyopic with vision of $\frac{6}{24}$ and $\frac{6}{36}$. In three of these four cases the lateral movements are excellent and the convergence normal. In one of the amblyopic cases the convergence is slightly below normal, but is improving with training. All the cases possess stereoscopic vision according to the degree of their visual acuity, and besides passing the ordinary tests with geometrical figures in the stereoscope they all bar read with ease, and appreciate the parallax at all distances when examined with the distance stereoscope which I showed at the Society last year. I consider that this stereoscope affords the most simple and accurate test for binocular vision with which I am acquainted. It is superior to the well-known Hering test in being a quantitative and not merely a qualitative test, and it enables the observer to estimate with great accuracy the grade of stereoscopic vision possessed by a patient. A full description of it appeared in the Lancet for July last.

Of the remaining fourteen cases, three are very nearly cured. The angle of the squint prior to operation varied from $18^\circ$ to $24^\circ$. The result cosmetically appears perfect, and in the stereoscope they possess fusion with good amplitude. They can bar read with a weak $+\text{ lens}$ added to their distance correction, and two of them can appreciate the parallax in the distance stereoscope when approached to within 3 mètres of the apparatus, and provided with a weak $+\text{ lens}$ in addition to their distance correction. They are not as yet able, therefore, to harmonise their accommodation and convergence at all distances in ordinary life. Orthoptic treatment will probably bring about their cure without further operation. In two of these cases the visual acuity in each eye $= \frac{6}{6}$. In one case V. in the deviating eye $= \frac{6}{18}$. One of the two cases with normal vision, while passing the plane fusion tests and possessing great amplitude of fusion in the stereoscope, has no perception of the third dimension. I have observed this curious fact in several other cases of squint. The third
group of cases consists of four. The angle of the squint varied from $28^\circ$ to $35^\circ$. Three have good visual acuity in each eye. One case is amblyopic: $V. = \frac{6}{36}$ in the deviating eye. They all fuse in the stereoscope, and possess some power of relaxing their convergence in the interests of binocular vision. Bar reading is by alternation even when convex lenses are worn in addition to their distance correction. They will all require a second advancement operation. The fourth group consists of three cases: in two the visual acuity is normal in each eye, while in the third case the $V.$ in the amblyopic eye $= \frac{6}{60}$. The angle of the squint varied from $30^\circ$ to $50^\circ$. They all possess plane fusion with some slight power of relaxing convergence. Bar reading is by alternation. The effect of the operation, i.e., a strong advancement with resection of the tendinous extremity of the muscle, has been to reduce the convergence by about $20^\circ$ to $25^\circ$, but another strong advancement will be necessary. The last group of four cases consists of those in whom a cosmetic result alone was possible, the deviating eye having no fixation either from the long duration of the squint or the presence of opacities in the cornea or lens. In all a very satisfactory result has been obtained; the convergence before operation ranged from $15^\circ$ to $25^\circ$.

I will now describe my method of operating. It is extremely important that a full view should be obtained of the different structures during the operation, in order that the attachment of the stitches, both to muscle and sclera, should be firm enough to withstand the strain of the powerful internal recti until union by new tissue has taken place. Most text-books at the present day advise that the threads which are attached near the cornea should be passed underneath the conjunctiva and through the episcleral tissue. I have found, however, by experience that the tissue which lies between the conjunctiva and true scleral fibres is of no use at all in holding a stitch, one or both threads tearing through soon after their insertion, and where a firm hold has been obtained by this method the
operator has invariably, I believe, gone deeper than the episcleral tissue, and has passed his needle through the true sclera, a very slight hold on which will suffice. Now, my point is that it is impossible to do this with accuracy or safety unless the sclera is thoroughly exposed. With regard to the muscular attachment of the suture it is equally important that this should not slip, and that the muscle itself should not be damaged or strangled in the process. The method by which I have obtained the most satisfactory results is as follows:

A vertical fold of conjunctiva, including Tenon's capsule, is picked up with forceps 4 mm. from the corneal margin and snipped off with scissors. The conjunctiva and underlying Tenon's capsule are then carefully dissected off the sclera up to the corneal margin, great care being taken to expose the true scleral fibres. The strabismus hook is then slipped underneath the tendon and followed by the clamp forceps, which are securely closed on the end of the tendon, the conjunctiva being excluded. The tendon is then divided close to the sclera and the conjunctiva carefully dissected from its outer surface and allowed to retract very fully. This is an important matter, as it prevents any subsequent drawing out of the outer canthus or caruncle as the case may be. This proceeding freely exposes the lateral expansions of the muscle, which it is necessary to see, and prevents the conjunctiva from subsequently interfering with the free movement of the advanced muscle, which it does, I think, to some extent when brought forward in the usual way. A short, straight, flat, double-edged needle, specially made for the purpose, and exceedingly sharp, is now threaded with No. 0 white silk and passed through the retracted conjunctiva close to its free margin, then through the lateral expansion of the muscle just below the lower border of the latter at the point A (Fig. 16, 1), which may, of course, be a variable distance behind the attachment of the clamp forceps, according to the effect desired. In high degrees of squint the needle should be entered as far back as possible.
The needle is then carried forward and is passed vertically through the superficial fibres of the sclera at the point \( b \). The eyeball is steadied meanwhile by grasping the fold of conjunctiva, which has been turned forwards over the corneal margin. This will suffice if the needle is sharp enough, but it is better if there is a difficulty in the passage of the needle to lay hold of the stump of the divided tendon, as the conjunctiva is apt to lacerate in the process. It is advisable, however, to have several needles ready threaded, and in the event of the first not readily biting the sclera to immediately discard it for another. This attachment to the sclera is all important, and should be very carefully done. The thickness of this coat at the site of operation is only \( \frac{1}{10} \) mm., and on this account I prefer a straight needle, over which the surgeon has full command. The needle is now carried backwards parallel to its original

Fig. 16.

1. Operation as performed on the right external rectus.

2. Operation as performed on the left internal rectus.
course, and emerges at c through muscle capsule and conjunctiva about $2\frac{1}{2}$ mm. above the point of entry. A second thread is passed through the conjunctiva and upper margin of the muscle opposite to the first at the point d. It is then passed through the sclera at e and emerges on its return through the lateral expansion g of the muscle, and then through the margin of the conjunctiva at f. There are now four threads hanging from the side of the eyeball. The assistant holds the globe in the middle line by means of forceps applied to the conjunctiva on the opposite side close to the cornea, while at the same time he draws the muscle forwards with the clamp forceps so that it lies flat against the sclera. The operator now ties the threads in the manner shown in the figure. The forceps are then removed from the muscle, and a variable portion of its tendinous extremity in front of the sutures is cut off obliquely so as to leave a raw surface in contact with the sclera. It will be noticed that in cases of high convergence, where the stitches have been inserted far back, the free extremity of the muscle comes well over the cornea, and in some cases I have removed as much as 6 mm. or even more. The conjunctival wound is now closed with a fine continuous stitch, which is not tied, and the free extremities of which are carried underneath that membrane away from the corneal margin.

The following advantages may, I think, be claimed for this technique:

(1) By a complete exposure of the various structures the operator knows precisely what he has done.

(2) It will be observed that only the margin of the muscle with a piece of the adjoining expansion is included in the stitch, so that the muscle itself is not strangled or injured to any degree.

(3) The knots lie at some distance from the corneal margin, and so do not cause irritation of the latter.

(4) The attachment of the stitches close to the corneal margin, while it increases the rotatory movement of the globe does not produce enophthalmos.
(5) The hold on both eyeball and muscle is firm.

(6) The stitches are easy to remove, the conjunctival stitch being simply drawn out while the others frequently come away by themselves about the ninth or tenth day, and if not the loop can readily be snipped.

(7) Cosmetically the result is very satisfactory, there being only slight indications of surgical interference after some months have elapsed. It is important in this regard to be neat and careful in suturing the conjunctiva, etc.—more especially in operations on the internal recti.

A slight modification of technique is occasionally necessary when operating on the internal recti in high divergence, as there is some difficulty in manipulating a straight needle in the confined space towards the inner canthus. The scleral stitch may first be inserted (Fig. 16, 2, b), and the extremities of the thread carried underneath the muscle and other structures by means of curved needles as shown in the second figure. The proceeding takes little extra time and is often unnecessary. The dosage must be regulated chiefly by the experience of the operator and his knowledge of the angle of the squint. Nearly all my operations were done under cocaine, which can be used successfully with children of seven years and upwards.

An important paper by Landolt appeared in the May number of the French Archives of Ophthalmology, in which he recounts the results of the double advancement operation in twenty-five cases of strabismus. The results are remarkable, and may lead to renewed interest in the subject by ophthalmic surgeons in this country. Landolt operates on both eyes at one sitting, a method which I have so far not adopted and to which I think there are objections. His published results, however, appear certainly to justify the practice. I may mention that in one or two cases of heterophoria where operation has been necessary, I have contented myself with including the expansions only of the tendon in the loop of the thread, and have drawn the muscle forward by this without passing the thread through the muscle itself, and I think that this
will be found sufficient in other similar cases. Since writing the above paper two cases in Group 2 have been finally cured by orthoptic training without further operation, and two cases in Group 3 have been cured by an advancement of the other external rectus muscle. These are the only cases so far which have been submitted to further operation. (January 28th, 1909.)

20. Tubercular mass in choroid (?).

By Arthur W. Ormond.

S. H—, female, ast. 39 years, attended the Out-patient Department of Guy's Hospital on October 5th, 1908, complaining of defective sight in the left eye. This had been noticed since July.

V.: R.E., $6 \text{ c} + .5 \text{ D. cyl. } 120^\circ = \frac{6}{6}$; L.E., $\frac{6}{24}$, not improved.

On examination of the left eye a large solid detachment of the retina was seen situated below and on the outer side of the optic disc. The inferior temporal vessels passed over it. The detachment was circular and dome-shaped, with a white soft-looking surface, edged by numerous small haemorrhages. The top of the swelling was seen with a $+ 7 \text{ D. sph.}$

On medical examination the patient was found to be quite healthy and nothing was discovered which threw any light on the nature of the mass.

Patient was admitted to the ward and kept in bed, the temperature being taken every four hours for three days. It was quite normal.

On October 15th 005 c.c. T.O.A. was injected, and twenty-eight hours later the temperature was 99.4°F.

Four days later, after the temperature had returned to normal, a second injection was made, but double the dose was used (01 c.c.), and in twelve hours the temperature rose to 103.8°F.
The patient was subsequently treated with Koch's new tuberculin (T.R.) .0001 mgrm. being given at first, and this dose or an enlarged one has been given at intervals of fourteen days since.

Note.—The mass in the choroid has now been under observation for about four months and under treatment for three months.

There is no doubt that the swelling is smaller in circumference, and although the top can still be seen with a +7·0 D. lens, after the injections of T.O.A. the swelling was larger and seen with a +10 D. lens.

Note.—March 31st, 1909: The condition may be said to remain unchanged; it is neither better nor worse. The injection of tuberculin is being continued at intervals of about fourteen days. She has recently had influenza. Vision remains as before.

(Card specimen, January 28th, 1909.)

Mr. Ormond said he brought the case in the hope that he would obtain the opinion of members upon it. He might be able to show the case again later on, and then members would be interested in noting the differences.

The President said it would be important to see the case again. He had not been in the habit of finding so much detachment in the cases where he had diagnosed tubercle of the choroid. He understood that the temperature rose as the result of the treatment, but in spite of that there might be reason subsequently for thinking it was not tubercle.

Mr. Ormond replied that the temperature rose on the first occasion, after a small dose, to 99·8° F. within twenty-eight hours. Four or five days elapsed, and then the second dose was given, of double the strength. Then the temperature rose within twelve hours to 103·8° F.

By R. W. Doyne.

A. J. B—, aet. 64 years. Patient first seen by me June, 1905. The condition then was much the same as it is now, except that the lenses have more opacities. Patient has known that he is diabetic nearly two years. Fitted in 1896 with R., $-4\frac{5}{5}$ Ds. $-2$ cyl. ax. 100; L., $-5\frac{5}{5}$ Ds. $-1$ cyl. ax. 65, and was told then he had some cataract, but the patient does not recall that anything was said about the cornea.

His vision in 1905, with correction, R. $\frac{6}{12}$ partly, L. $\frac{6}{12}$ partly. To-day, January 28th, 1909, with some correction, R. $\frac{6}{18}$, L. $\frac{6}{3}$.

*Description in 1905.*—Cornea: In both, but especially the left, and occupying the central part, touching the limbus on each side is a haze very sharply defined at the upper and lower margins, but fading towards the centre, where it is practically transparent. No structure can be made out in it, nor does it at all resemble the condition known as transverse calcarceous film; at the densest parts the structure of the iris is marked (this will not be noted to-night because the pupils are dilated with homatropine).

It does not correspond to the palpebral fissure, and has rather the disposition of a congenitally opaque cornea. This description corresponds to the appearance at the present time.

*Lenses in 1905.*—There is in the centre of each lens, especially the right, near the surface, some greenish-white dots like those seen sometimes as congenital flecks; and more usually seen scattered at the periphery of the lens, but these are more agglomerated and "scratchy." In the right lens there are a few very small vacuoles.

To-day, January 28th, 1909.—I think the condition of the right lens is more gross than formerly; the opacities correspond somewhat to the structure of the lens.

*(January 28th, 1909.)*
The President said he understood Mr. Doyne to say that the opacity had increased both in density and area since it was first observed by him. When Mr. Doyne kindly told him that he was going to show this case he (Mr. Gunn) looked up his notes about the patient. He had seen him nine years previously to Mr. Doyne's first notes, namely in 1896, and he had made no note in his case-book of anything wrong with the cornea at that time. He had, however, noted that there were opacities of the lens, showing that he had examined the case with focal light, and he did not think he would have overlooked corneal opacity if such had been present. The vision at that time was very good, and therefore the condition now shown had almost certainly developed since the date when he saw the patient himself. The opacity was of a peculiar nature, and its causation seemed to be obscure.

Mr. Doyne replied that the interest of the case was great because of the slow progress. He was satisfied that the condition was not present when Mr. Gunn saw him, but the President was very modest about it.

22. Congenital cataract; a pedigree of five generations.

By N. Bishop Harman.

M. H—, a girl, aet. 7 years, was seen by me in a blind school a year ago. She had a dense lamellar cataract filling three fifths of the lens area. The opacity was clean and sharp edged, and there were no outlying riders. The responses to all the usual tests to ascertain the condition of the retina were good; the periphery of the fundus could be seen to be of good colour. The teeth were good. The parents were notified that an operation was advisable. The advice was neglected for a time, but by the good offices of a school manager the attendance of the girl at
hospital was secured. Then it was found that the mother had congenital cataract also, and on investigating the family history I obtained the pedigree given here.

The investigation has been carried through five generations; members of four generations are alive now. Seventeen childships are represented, with 63 persons in direct descent;* 8 died in infancy or at birth. Of the remaining 55 it is known that 19 had cataract in early life.

There has been no instance of in-breeding, and in no case can I trace any instance of marriage into a family affected with congenital cataract, or with a tendency to senile cataract. The members of the family are of fair intelligence; in one childship a male is mentally defective; in another childship one girl has become insane in later life.

Notes on Individuals.

[* Seen by the author. † Condition reported by good authority.]

Generation I.—1, male, dead. He is reported by the wife of Generation II, 3, as habitually holding things he wished to see close to his eyes; she had heard that the "inside of his eyes" were like her husband's, who has cataract. Generation III, 2, knew him well, and described the manner in which he shaded his eyes and screwed up his eyelids in attempting to see distant objects. His wife is stated to have had excellent sight.

Generation II.—1, female, died a year ago; had cataract for which she had operated in childhood.

2, female, died many years ago; "had good sight."

3,† male, had congenital cataract of small size; died last Christmas just after I had investigated his case.

4, female; three branches of the family state that she had cataract from childhood.

5, female; eyes said to be good; the son states his mother can read the finest print with ease.

* Consorts are not included.
Generation III.—Nos. 1 to 5, childship of II, 1; father cataract; mother normal.

1, male, dead, was operated upon for cataract at a London hospital, name forgotten.

2†, male, dead; stated to have been operated upon at old St. Thomas's Hospital. Mr. Lawford kindly caused inquiries to be made, and the case was traced: "F. J.—æt. 6 years, admitted October 14th, 1863; discharged November 22nd, 1863. Congenital cataracts; under Mr. Simon."

3,* male, æt. 49 years. He said his sight was perfect. On examination, small congenital opacities were found in the central region of each lens just anterior to the nucleus. V. = J. 1 with + 1 sph. Teeth normal. This man has married twice, wives not related to him or to each other; both are dead, but are reported to have had good sight. A daughter by the second wife with a former husband has been seen; her lenses are normal.

4,* female, æt. 46 years. The woman said her eyes were perfect. On examination I found small congenital opacities in the anterior layers of the central area of the lenses. V. = J. 2 without glasses. Teeth normal.

5,* female, æt. 43 years; eyes and teeth examined and found normal.

Nos. 6 to 12, childship of II, 2; parents dead; father had good sight—he was a mounted police sergeant; mother said to have had good sight.

6,* female, æt. 50 years; eyes examined, found normal.

7, female, died in infancy.

8, male, died in infancy.

9, female, died of phthisis many years ago. The master of the school where she was a resident says that, so far as he knows, her sight was good.

10, male, died at birth.

11,* male, æt. 40 years, an engraver; "sight perfect; can engrave the Lord's Prayer on a three-penny piece"; eyes examined, lenses normal.

12,+ male, æt. 28 years, an inmate of Colney Hatch
Fig. 17.

CONGENITAL CATARACT: PEDIGREE OF FIVE GENERATIONS.

Male: ☐ female: ☐ congenital cataract; ☐ died in infancy.

(Consorts are not marked in the pedigree, but they are duly noted in the text.)
Asylum. Her eyes have been examined by the Medical Superintendent, who reports—"lenses free from any trace of cataract."

Nos. 13 to 20, childship of II, 3; father cataracts; mother normal.

13,* female, had both eyes operated on for congenital cataract at Moorfields as a child. Teeth good.
14,* male, also operated on at Moorfields; teeth good.
15,* female; has well-marked, hard-edged, congenital nuclear cataract; optical iridectomies have been made.

V. = $\frac{6}{1.4}$ Teeth good.
16, female, and 17, twin males, died in infancy.
18, female, said to have a perfect sight; lives in Madagascar.

19,* male, twin with 20; eyes and teeth quite normal.
20,* male, twin with 19; 
21 and 22, males, children of III, 4, cannot be traced; mother had cataracts as a child.
23,* male, child of III, 5; parents, normal sight; son's eyes examined and found normal.

Generation IV.—Nos. 1 to 5 first childship of III, 3; father has cataracts; mother dead, said to have had perfect sight.

1,* male, aet. 26 years, mentally defective; minute central opacities just in front of the lens nucleus each eye. Teeth good.
2,* male, aet. 24 years; both lenses removed at Moorfields as a child. Teeth good.
3, female, died, aet. 17 years, of phthisis; sight said to have been good.
4,* female, aet. 15 years; eyes and teeth good.
5,* male, aet. 4 years, child of III, 3, by second wife; eyes examined and found normal.

6 to 10, childship of III, 5; parents said to have perfect sight.
6, male, aet. 27 years, living in S. Africa; "sight perfect."
7,* female, aet. 26 years; ) eyes examined, found normal.
8,* female, aet. 23 years; ) Teeth good.
9, male, aet. 21 years, not seen; in Government service as postman; eyes said to be perfect.
10, male, aet. 19 years; eyes said to be perfect.

Nos. 11 to 15, childship of III, 6; mother seen, normal; father dead, said to have been normal.
11,* male, aet. 20 years; 12,* male, aet. 18 years; 13,* female, aet. 15 years; 14* and 15,* males, aet. 12 and 8 years all seen; eyes and teeth found normal.

Nos. 16 and 17, childship of III, 11; parents' eyes normal.
16,* male, aet. 12 years; ) eyes examined, lenses per-
17,* female, aet. 7 years; ) feet. Teeth good.

Nos. 18 and 19, childship of III, 13; mother cataracts, father normal.
18, male, died in infancy.
19,* male, aet. 15 years; both lenses removed for cataract at a London hospital. Teeth good.

Nos. 20 to 22, childship of III, 14; father cataracts; mother normal.
20,* female, aet. 13 years; operated on at Guy's Hospital for congenital cataract. Teeth good.
21,* male, aet. 10 years; ) eyes and teeth examined and
22,* female, aet. 5 years; ) found normal.

Nos. 23 to 29, childship of III, 15; mother cataracts, father normal. It is worth noting that all of the childship were said to have perfect sight save No. 26.
23,* female, aet. 13 years, V. = $\frac{6}{9}$, II. 5 D.; lenses clear. Teeth normal.
24,* female, aet. 11 years, V. = $\frac{6}{12}$ R. and L. refraction
CONGENITAL CATARACT: PEDIGREE OF FIVE GENERATIONS.

+ 2 D. sph. + 1 D. cyl.; small opacities anterior to nucleus of each lens, Fuch's colobomata of discs. Teeth slightly ridged.

25,* female, aet. 9 years; V. = $\frac{6}{18}$, refraction R. = 1 D. sph. - 1·5 D. cyl., L. = 5 sph - 2 D. cyl.; small opacities anterior to nucleus of each lens, R. Y-shaped, L. X-shaped. Teeth normal.

26,* female, aet. 6 years; dense lamellar cataracts occupying the middle three fifths of each lens; removal at the Belgrave Hospital with good results. Teeth normal.

27,* female, aet. 5 years; R. and L., H. 3 D., lenses normal.

28,* male, aet. 3 years; both lenses have delicate lamellar defect and central stellate opacities.

29,* female, aet. 6 months; lenses quite clear.

Nos. 30 to 33, childhood of III, 18, migrated to Madagascar; parents said to have perfect sight. Nos. 30 and 32 died at birth; 31 and 33, females, are said to have perfect sight.

Generation V.—Only one member as yet. A boy,* aet. 3 years, child of IV, 7; parents normal eyed; the child was examined, the lenses were perfect.

Remarks.

Character of cataracts.—In every case the opacity is of such a size that it is evident that the disturbance of lens nutrition which resulted in the "fault" of the lens growth took place before birth. In many cases the opacities are quite small, close to the nuclear region, and take such a shape as indicates the fault lies in the union of the anterior ends of the lens fibres as they grew from behind forwards. In those cases where a definite lamellar structure is found the opacity is small in relation to the total size of the lens, and is sharply defined and free from riders.
There is no evidence of an increase in the opacities during life.

Teeth.—The teeth are good. Only one case was found with ridged teeth, and then of slight degree only, and in this case there were small lens opacities close to the nucleus.

Inheritance.—The affected persons are always born of affected parents; no cataracts have been found in childships born to normal parents. Males and females transmit to their children. In one line the transmission is through male, female, male; in another line it is through male, male, female. It is therefore clearly a condition of germ inheritance.

Numerical proportions.—Of fifty-five persons nineteen were affected. This falls short of the Mendelian requirement, as it is propounded by Bateson, by eight. But it is difficult to be sure of the condition of eyes in cases such as these failing personal examination, for individuals reported to have good sight may really be affected by lens opacities of small degree. Indeed, in this pedigree one childship of seven members only owned to one cataractous member; on examination a total of four was found; in another childship of five, cataract was acknowledged in two members, but examination showed four were affected. Of the fifty-five individuals I have examined thirty-six myself, and have certain, or what amounts to certain, information of five more, making forty-one; amongst these there were nineteen cases of cataract. In the medical history of these childships there were no special features other than those named at the outset of the paper.

(January 28th, 1909.)
23. An unusually rapid development of complete cataract in a boy.

By N. Bishop Harman.

The boy who is the subject of this note was brought to me at the Belgrave Hospital for Children in September, 1908; he had been referred for examination previous to consideration of special teaching in a blind school.

His mother stated that the boy could see quite well nine months previously, and that his sight had rapidly got bad until the present time, when he was blind. She said that he had suffered no injury, nor any illness since measles and scarlet fever at the age of two years.

The woman's manner was voluble and not a little irrelevant, so that little weight was attached to her statement, but inquiry proved she told the truth.

C. H,—, ast. 9 3/4 years, a healthy looking, well developed lad; answers questions well, but gives the general impression of being "mazed." He does not walk like the child who has been blind for years, but gropes about very slowly and with uncertainty, stumbles frequently, and shows a total absence of that sense of location which is so noticeable a possession of the blind.

Examination of the eyes showed that the lens of each was completely cataractous. When the pupils were widely dilated by a mydriatic each lens was seen to possess no clear substance; all the lens matter was opaque right up to the capsule and as far to the equator as could be seen. The lenses had the appearance of "soft total cataracts." The opacity had the semi-translucent appearance of milk and water; by oblique illumination the filament bundles could be clearly seen as radiate striations of slightly greater density, but no hard, densely white particles were present in any part.

No fundus reflex could be obtained, even in the extreme periphery.
The lenses appeared to be of the normal contour and size, and the anterior chambers were normal in depth and in clearness of contents. All the visible parts of the eye were healthy—lids, cornea, sclera, and iris; there were no evidences of previous illness or injury, no K.P., and no scars. The reflexes were good. Both pupils reacted well to light, directly and consensually.

The boy had perception of light; he could locate the movement of an object passed between the light and his eyes, but he could not determine its shape nor distinguish fingers. The fields taken by projection of light were full. Testing the maculae by approximating and separating two lighted matches gave uncertain results; his answers were rarely correct.

His teeth were good; the urine normal (sp. gr. 1012, acid reaction, no sugar, no albumen); the physician reported his bodily condition excellent, and heart, lungs, and abdominal organs normal; there was no evidence of any exposure to chemical poisons.

In fine, the boy's lenses presented the appearance of typical senile cataracts in a stage of perfect maturity, and in just the condition which one would exhibit to students as cataractous lenses ripe for extraction. But there was no evidence to show how he had come by this condition, and the appearance of such cataracts at so early an age made one wonder what operative interference would bring forth.

He was taken into hospital, and each lens removed by discission and curette evacuation.

First day, R. lens needled freely.
Eighth day, R. lens evacuated.
Fifteenth day, R. lens posterior capsule needled; L. lens needled freely.
Twenty-second day, L. lens evacuated.
Twenty-ninth day, L. lens posterior capsule needled.
Thirty-sixth day, discharged with +12 D. sph. R. and L. V. = \( \frac{6}{18} \).

Three months later vision with glasses = \( \frac{6}{6} \).
The work took just under six weeks. A general anaesthetic was given at each operation and taken without difficulty. The lenses reacted perfectly to the interference; their consistency was uniform; each swelled up, filling the anterior chamber with flocculent masses, which were evacuated so easily that the pupils were clear two or three days after the curette evacuation, and at no time did any inflammatory reaction appear. The only point of an exceptional nature was that at each operation, when the anterior chamber was entered, blood was seen to ooze—"spurt" might almost be written—from the root of the iris all round its circumference, so that the blue iris was tinged red; the effusion was not serious, it did not well up so as to float freely in the anterior chamber, and it was speedily absorbed.

Examination of the eyes subsequent to operation revealed no abnormality in the posterior chamber or fundus of the eyes. Retinoscopy gave his refraction as +11.5 D. sph., with +1.5 D. astigmatism in each eye. With +12 D. sph. before each eye the boy straightway read $\frac{6}{18}$ and one letter of $\frac{6}{12}$. No better proof could be made that he had been a sighted child within quite recent date.

At this date he reads $\frac{6}{6}$ easily with his glasses.

**School and Family History.**

While the child was in hospital these matters were investigated:

The father has disappeared.

The mother’s eyes are healthy; she has the presbyopia natural to her years.

The childship consists of six; all are living.

1, female, aet. 21 years, not seen; stated to have perfect vision.

2, male, aet. 15 years, not seen; stated to have perfect vision.
3, female, æt. 13 years, not seen; stated to have perfect vision.

4, male, æt. 10½ years; R. and L. V. $\frac{6}{9}$ each. Retinoscopy R. and L. $-1$ D. cyl. = $\frac{6}{9}$.

5, male, æt. 9 years, C. H—; cataract.

6, female, æt. 8 years; R. and L. V. $\frac{6}{6}$ each. Retinoscopy R. and L. $+1$ D. sph.

Of seven, the eyes of three—mother and two children—are healthy and show no lens opacities; their teeth are good. The three eldest children could not be seen.

School.—At the age of 7½ he entered an L.C.C. elementary school. The headmaster writes: "There is no record of his vision as he left the week before the test was taken, but at the examination held March, 1907, his marks were: Standard O, reading $\frac{8}{10}$, writing $\frac{8}{10}$, spelling $\frac{5}{10}$, freehand drawing $\frac{7}{10}$.”

In September, 1907, he was transferred to another L.C.C. school, and his record is the following:

"Vision test, September, 1907, D. = 18.

"Vision test, September, 1908, can see nothing on test-card.

"His sight has grown rapidly worse since May, 1908, when his mother was notified. Writing and reading were stopped, but at the examination of July 15th he was allowed to do a paper of writing. This paper is forwarded, together with one done on September 3rd.”

The July paper proves that the boy could see comparatively well; the writing is fair, and runs evenly between the ruled blue lines, which are 4 mm. apart. The writing of September is just that which a boy would do blindfold; it is irregular, letters are inverted, duplicated, or missing, and there is no alignment. It is evident from these data than in the space of fifty days vision was completely obscured.

In the course of these inquiries I learnt that the boy had been taken to St. Thomas's Hospital earlier in the year. Mr. J. H. Fisher has kindly furnished me with the following notes of his examination:
"March 23rd, 1908: Complaints from school about eyesight six months; holds book close to eyes and is backward.

"Opacities at posterior part of each lens; fair view of R. fundus obtained, no view of L.

"No family history of cataract.

"March 27th, 1908: V. under atropine $c + 0.75 D. sph. = \frac{6}{36}$ partly R. and L. Recommended for admission for needling, but failed to attend when sent for."

**Remarks.**

There is no evidence on which to form any judgment of the boy's eyes before the time when his vision was known to be failing. There may possibly have been lamellar cataracts of developmental or rachitic origin, which later progressed and matured with such rapidity, but the boy's general state, his mental capacity, his teeth, and the state of his elder brother and younger sister, who were examined, give no support to the supposition. There only remains the supposition that some unknown or unnoticed changes in his general health affected the blood supply of his ciliary bodies and indirectly of the lens epithelium. It is known that the family were in straitened circumstances just previous to the failure of the boy's vision owing to the delinquency of the father, but these circumstances have not affected the two children who most nearly approach the boy's age. There appears to be evidence of an unusual condition of the blood-vessels of the ciliary regions of the boy's eyes in the readiness with which they allow extravasation of blood on the diminution of the internal pressure of the eye consequent on the penetration of the anterior chamber. Further, the condition of the lenses in March last—opacities in the posterior part of each lens—as noted by Mr. Fisher, indicated a failure of the lens epithelium to maintain the nutrition of the crystalline fibres.

*(January 28th, 1909,)*
Mr. Herbert Fisher’s notes of the condition when the boy was in St. Thomas’s Hospital were read by Mr. Harman.

The President said the papers were both very interesting. Mr. Harman had given a valuable genealogical tree of lamellar cataract. He asked Mr. Harman whether he had found noted in literature any instances of such a rapid development of cataract as was recorded in his second paper. He believed that Becker had described a case of very rapid development of cataract in one or both eyes, and he (Mr. Gunn) had had one case of his own. It occurred in a patient with high myopia, for which she had been under treatment for some time at the Moorfields Hospital. The myopic condition was progressive, and was associated with much choroidal disturbance and pain. Between two visits, which were on successive weeks, a cataract developed to maturity in one eye; the cataract was complete or nearly so, yet no opacity had been seen the previous week. There had been no injury to the eye. From the nature of the case the vascularity of the choroid was here also abnormal.

Mr. Harman, in reply, said he had looked up the literature, and found no case of the kind with healthy fundus appearances and without history of injury or body disease. Norris and Oliver, and Lagrange and Valude refer to cases occurring in young children, the latter under the term “Phacomalacia.” But it was said the contents of the lens were usually fluid with calcareous particles.
A CASE OF RETINAL EXUDATIONS.


By David J. Wood (Cape Town).

(With coloured ophthalmoscopic drawing [Plate III].)

In vol. xii, p. 143 (1892) of the Transactions is a drawing with a brief description of a case of retinal detachment with distended and tortuous vessels. In vol. xxv, p. 96 (1905) I described, though without a drawing, a second case, and recently I have, through some curious luck, been able to examine a third example of what I believe to be the same rare condition.

I saw the patient first in 1902 when I ordered glasses for her. No complaint was made of bad sight then, but she said she had once had an attack of dim vision and I examined her fundi. As my second instance had not at that time impressed upon me the interest attaching to these cases, I only made a note that there were two large blood-vessels running up to a patch at the upper outer part of the fundus, which I thought might have been the result of a former haemorrhage, the same having been the cause of her attack of bad sight.

She returned to me in November, 1907, being then about fifty years of age, and told me that a few days previously her right eye had failed suddenly, so that she could only discern large objects. I found this to be due to diffuse opacity in the vitreous doubtless caused by a haemorrhage, and no fundus details could be made out. The left fundus was normal, but a small amount of opacity of the lens was present.

As the right vitreous began to clear there was made out by degrees the picture which, with the help of Mr. Donald Gunn, I am able to show the Society.

The first thing visible was the white area above, and a little later the enormously dilated blood-vessels which can
be seen running up from the disc to it. The picture shows to some extent their size and their twisting in the plane of the retina, but one could not well indicate the tortuosity in the opposite plane which, by producing large parallactic movements, was at least as striking. At first we called these vessels arteries, later, veins, and at last it became pretty certain that their colour was really intermediate and perhaps variable. Several vessels elsewhere seemed to alter their colour in different parts of their course; nearly all were difficult to trace out, and at least two arose from one branch and ended in another.

A third peculiarity, which is brought out in the drawing, was that from one of the main trunks (the left) a vessel springs which is obviously arterial in colour, while one as obviously venous springs from the other main trunk.

The size of the large trunks was from eight to ten times the breadth of what we thought would represent a normal vessel near the disc, but, as can be seen, they varied greatly. Both trunks run up and out and are lost in the white patch already mentioned. The highest part of this can be seen with a +7 D. or +8 D.; the surface is stippled and gives one the impression of blood-vessels seen through intervening tissue.

This is not shown well in the drawing, nor have we attempted to indicate the extensive fine choroido-retinal changes which cover the inner part of the fundus.

The question arose early as to the nature of the growth, and for a time I was inclined to think of sarcoma, a view which was also held by a colleague, who advised excision.

I was, however, so strongly impressed by the family likeness to my two earlier cases that I held my hand, and meanwhile I came across, in vol. xiv of the Transactions, Mr. Treacher Collins' full and careful description of the pathological conditions found in the eyes of my first patient and his sister.

That this supplies the key to these cases I can hardly doubt, and though detachment of the retina prevented me from making out any growth in my second case, it is at
PLATE III.

Illustrates Mr. David J. Wood's case of Retinal Exudations with Extreme Distension of Vessels, and perhaps Arterio-venous Anastomoses (p. 115).
least very probable that the growth in the present instance is, as Mr. Collins believes, angiomatous. In this way the distension and the abnormal colour of the large vessels is explicable, but the communicating branches are not so easily explained, unless, indeed, they are only lesser developments of what formed ultimately the angiomatous mass. So far no detachment has occurred in this case, and, as my second case suggested, this is probably a late change which may perhaps be started in these cases by the increasing twists in the blood-vessels.

In the picture are shown two rings of pigment on one of the large vessels; another has since appeared, and is so far the only certain alteration observed in the fundus. With the clearing up of the vitreous there was, of course, great improvement in vision, but that it should have reached \( \frac{4}{5} \) is more than one would have expected, the more so that the macula lies near the lower white patch and the curly vessel to the left of the disc. (It is necessary to tilt the picture to the left to obtain the correct orientation of the drawing.) I regret that inquiries have not enabled me to hear more of my second case, but I hope to keep this one under my observation so as to report further on it. (January 28th, 1909.)

Mr. Treacher Collins said the cases of angioma of the retina described by Mr. Wood were exceedingly rare. He had only seen the two which had been referred to, in a brother and sister, and he had only heard of one other in which a pathological examination of the eyeball had been made, and that was referred to in a recent paper by Mr. Coats. In the two cases which he (Mr. Collins) examined pathologically, the clinical appearances were very remarkable. In one they were very similar to that in the picture shown this evening. In the other, the sister, who came under observation later, both eyes were involved and the vascular growth had spread forward to the anterior part, so as to invade the cornea. Both corneae were staphylomatous, and both eyes had to be excised on
account of the pain. He had not seen anything resembling the appearance shown clinically or microscopically in these two cases before or since.

Mr. G. Coats said he had recently had occasion to look into the literature of such cases (Royal Lond. Ophth. Hosp. Reports, vol. xvii, pt. 3, p. 496). Leaving out of account an incomplete record by Panas and Remy (1879), Fuchs gave the first clear description in 1882, and in all some eighteen cases had been reported. Mr. Wood’s first case was the third in the series. The cases were all alike and formed quite a group by themselves. They occurred in young people, the average age being under twenty-five. Very frequently the disease was bilateral, and Mr. Wood’s first case was especially interesting in this respect, because a brother and sister were both attacked, and in each case the disease was bilateral. The rarity of all family or personal stigmata of disease was one of the most remarkable and puzzling features of the affection; such as had been found in individual cases were so varied that no aetiological importance could be ascribed to them. The most characteristic ophthalmoscopic feature was the enormous enlargement of certain vessels. These were probably always an artery and a vein, though in most of the recorded cases they looked much alike—either both light or both dark. Very frequently, although the main trunks were indistinguishable, the branches preserved their arterial or venous characters. Mr. Wood had noted this point. When they could be followed to their termination the vessels ended in a rounded yellow or reddish area. There was usually a good deal of retinal exudation. If watched over a prolonged period the cases always progressed; detachment and sometimes vitreous haemorrhage occurred, new anastomoses formed in different parts of the fundus, and finally, perhaps in ten to fifteen years, the eye was almost inevitably lost from iritis and secondary glaucoma. As to the pathology, omitting a very incomplete record by Panas and Remy, Mr. Collins made the first examination on Mr. Wood’s first case, and
on the sister of the same patient. Mr. Collins attributed the condition to a capillary angioma of the retina, and recently Czermak had come to a similar conclusion without knowledge of Mr. Collins' work. Apart from this independent agreement there were other points in favour of that hypothesis; the youth of the patients and the slow course of the disease were in accordance with it, while its tendency to be bilateral was more favourable to that explanation than to an inflammatory origin. Moreover, its gradual invasion of the surrounding tissues, as described by Mr. Collins, suggested active growth. At the same time it was undoubted that the affection might appear in an area of retina where it had not been present before, and that was unlike the behaviour of an angioma. It must be remembered also that if an artery and vein became enlarged for any reason their terminal capillaries would also become enlarged, and would come to resemble angiomatous tissue. The cases which he had himself examined pathologically were possibly allied, but were not exactly of the same nature.

Mr. Johnson Taylor asked whether the cases referred to had any similar condition in any other part of the body, such as cirrroid aneurysm or dilatation of vessels.

Mr. Coats, replying to Mr. Taylor, said that in no case had there been such condition as he inquired about.


By Archibald Stanley Percival.

I first saw Benham's top some ten years ago, and it was only after reading about Mr. Stewart's experiments on
Talbot's law* some months afterwards that I got any hint of an explanation of the colours produced on rotating it. As far as I understand the present theory of colour vision my explanation is no longer tenable. My object in bringing before you this physiological experiment is to obtain some reasonable explanation of it according to the present view, and if that is not obtainable to submit that there is still something to be said in favour of the discredited Young-Helmholtz theory.

In 1888 Mr. G. N. Stewart made some experiments on Talbot's law with reference to the sense of appreciation of light. A parallel beam of light was allowed to fall upon a rotating mirror in a darkened room, and in this way a series of very short flashes of light were received by the eye of the observer. Fusion of the flashes was obtained by increasing the speed of rotation of the mirror. He found that for the shortest stimuli he was able to use there was no noticeable change of intensity of the sensation, once complete fusion had been reached. Even for the faintest light no definite variation appeared, however rapidly the mirror was rotated, i.e., there was no appreciable departure from Talbot's law even when the stimulus only lasted \( \frac{1}{100} \) sec.

In the course of this investigation Mr. Stewart found that when the mirror was rotated slowly but with gradually increasing speed a series of colour changes was seen in the light reflected to the eye, especially towards the edges of the light band. As the result of numerous experiments it was found that for any given intensity of light there is a rate of revolution of the mirror with which violet preponderates, with a higher speed, green, and with a still higher speed, red. A decrease of illumination puts the whole phenomenon further forwards and corresponds

* Talbot's law: Once complete fusion has been reached, no alteration in the intensity of the resulting impression produced by a series of flashes takes place, however short the duration of each flash may be, provided that the number of the flashes in a given time and the duration of each flash be always kept inversely proportional. Complete fusion of stimuli is here analogous to complete tetanus of muscle.
to an increase of speed, while an increase of illumination puts the phenomenon to an earlier stage corresponding to a decrease of speed.

Now all these changes take place about or below the speed necessary for complete and steady fusion of the separate flashes. The idea at once suggests itself that

Fig. 18.

the phenomena are connected with the different course of the curves representing the excitation of the three-colour sensations of the Young-Helmholtz theory.

The adjoining figure (Fig. 18) represents the curves given by Mr. Stewart for the red, green and violet sensations. The time during which the stimulus acts is measured along the horizontal axis, and the intensity of excitation is denoted by the height of the ordinate at that point. Suppose that the stimulus considered be that of white light; if it be of the comparatively long duration
indicated by $O_A$, the excitation of each of the primary visual sensations will be equal, as is seen by the equal height of the ordinates, consequently the resulting sensation will be white. But if the stimulus lasts for a shorter time, as $Oa_3$, the violet sensation will be the most excited; if the duration of the stimulus be very short as $Oa_1$, the resulting sensation will be red. So far

Fig. 19.

I have been practically only quoting from Mr. Stewart's paper (*Proc. Roy. Soc. Edin.*, 1888). I will now give my explanation of the colours seen on this rotating disc, which depends on Mr. Stewart's figure (Fig. 18) of the excitation curves of the three primary colour sensations.

Consider a disc half white and half black, the white part being marked through a quadrant with a curved black line. If the disc be now rotated counter-clockwise and the eye be fixed, the line if thin will appear red. If the line be thick, the margins only of the line will
INTERMITTENT STIMULATION WITH LIGHT. 123

appear red. This shows us that the phenomenon depends on the irradiation of the surrounding white area over the edges of the black line. We have here nothing to do with the duration of the stimulus which was the important factor in Mr. Stewart's experiments, for it is found that an alteration in the size of the white sector, i.e., an alteration in the length of the curved black line, makes little difference in the tone of the colour seen. We must regard the eye after its period of rest while viewing the black area as suddenly stimulated by white light, with an irradiation effect extending over the margins of the black line. Now, considering this irradiation we are dealing with a rise of stimulation, and we see from Fig. 18 that the red sensation rises most abruptly, and therefore a red colour will be seen about the black line. Now on reversing the rotation of the disc the line appears blue. To explain this we must consider the course of the decline of the sensations. Helmholtz, Fechner and others have described a series of colours in the positive after-image of a white object. The after-image goes quickly out of the original white, through blue, into a rose colour. It is probable, therefore, that in their decline the curves follow a similar "grape-vine" course as during their rise; and, indeed, the colours of this rotating disc will be clearly explained if we regard them as precisely the same. When the disc is rotated clockwise, the eye may be considered as moving counter-clockwise round the diagram (Fig. 19). After a stimulus of white light the black line is encountered and an irradiation over its margins occurs. But as this involves a diminution of the stimulus of white light, we have only to follow the curve in Fig. 18 backwards and we see that at $Oa_3$ the violet blue line has the highest ordinate, and that therefore a blue colour will be seen. When the curved line occupies an intermediate position there will be more or less white stimulus, as the case may be, before the black line is encountered, and therefore an intermediate colour will be seen. Consequently when a disc with four sets of lines as this (Fig. 20) is rotated, four
colours will appear—red, stone-yellow, dirty green, and blue. Several erroneous conclusions have been drawn from these colour phenomena in physical experiments. I might instance the elaborate series of experiments carried out by Forbes and Young on the velocity of light published in the *Philosophical Transactions*, 1882. The method adopted was a modification of that of Fizeau, the details of which it is unnecessary now to describe. They found that when the brightness of the point of light was increasing its colour was red, but that when it was fading its colour was blue. From this they inferred that blue light travelled in air about 1.8 per cent. faster than red light. This conclusion was obviously erroneous, and Mr. Stewart shows that the phenomenon was really a physiological one; it depends, like Benham's top, upon an intermittent stimulation of the retina with white light, a
red colour being seen when the stimulus is rising, and a blue colour when it is falling.

(January 28th, 1909.)

26. Note on some rhythmic oscillations of the pupil.

By Archibald Stanley Percival.

We are all familiar with the rhythmic oscillations of the pupil that are seen when light is concentrated on the pupil of the eye in a case of retro-bulbar neuritis. In some cases, after perhaps one passing contraction, the pupil dilates and remains dilated for two seconds or more. In such cases I have been in the habit of regarding the prognosis as particularly grave. I am very anxious to hear the opinions of the members present on this point, as well as their explanation of the cause of the rhythmic oscillations that are so frequently seen. It seems obvious that the cause is to be found in some condition of the optic nerve, but to glibly assign the presumed diminished conductivity of the nerve as a sufficient reason is not a very satisfactory explanation.

Now it seems to me that the peculiar oscillatory discharge of a Leyden jar that occurs under certain conditions may throw light on the subject. It is found that if a Leyden jar be discharged slowly through a conductor of high resistance, such as a nearly dry linen thread, the charge simply dies away, the current increasing at first in strength and then gradually fading away. If, however, the discharge is made through a coil of wire of low resistance the effect is wholly different, for then the discharge consists of a number of excessively rapid oscillations or surgings.
SOME RHYTHMIC OSCILLATIONS OF THE PUPIL.

We owe to Lord Kelvin,* who investigated these oscillations, the mathematical explanation of the phenomenon. He showed that if a jar of capacity $K$ were discharged through a wire of resistance $R$ and of inductance $L$, the discharge would be aperiodic, i.e., all in one direction, if \[ \frac{1}{4} R^2 > \frac{L}{K}, \] and that the discharge would be oscillatory if \[ \frac{1}{4} R^2 < \frac{L}{K}. \] It is very remarkable that the oscillation of the discharge should depend merely on the relation between the resistance and the inductance of the conducting wire. If we regard the nerve-cell and the axis cylinder of the nerve as analogous to the Leyden jar and the conducting wire, some pathological explanation of the oscillatory action of the pupil might be attained, if one could say what property in a nerve corresponds with the inductance of a wire. I would suggest irradiation as analogous to inductance.

In the electrical instance we note that a diminution of the resistance is what is required to start the oscillatory discharge. In the case of retro-bulbar neuritis I would imagine that the resistance to the nervous current was similarly lessened owing to the destruction of the insulating sheath, for that I presume is the function of the medullary sheath of Schwann. In the early stages of

* Lord Kelvin's explanation of the phenomenon simply depends on the solution of the differential equation \[ \frac{d^2 Q}{dt^2} + \frac{R}{L} \frac{dQ}{dt} + \frac{Q}{KL} = 0, \] where $Q$ denotes the amount of electricity with which the jar is charged. The solution of this equation depends on the relative values of $R$ and $\frac{L}{K}$.

If \[ \frac{1}{4} R^2 > \frac{L}{K}, \] $Q = Ae^{\lambda_1 t} + Be^{\lambda_2 t}$, where $\lambda_1$ and $\lambda_2$ are the roots of the equation $\lambda^2 + \frac{R}{L} \lambda + \frac{1}{KL} = 0$. In this case the discharge is aperiodic.

If, however, \[ \frac{1}{4} R^2 < \frac{L}{K}, \] $Q = (A \sin q t + B \cos q t) e^{-\frac{R^2}{2L}}$, and the discharge is oscillatory with a frequency of \[ \frac{1}{2\pi} \sqrt{\frac{1}{KL} - \frac{R^2}{4L^2}}. \]
retro-bulbar neuritis the nerve-fibres are strangled and the medullary sheaths break up; consequently several uninsulated axis cylinders will come in contact, thus opening up several different paths for the nervous current—in other words the resistance will be decreased. When the axis cylinders themselves are strangled the resistance will presumably become greater, and the frequency of the oscillations therefore less. Can any member more versed in the intricacies of electricity and physiology than myself throw any light on this analogy, and explain "the inductance of a nerve" and the condition on which it depends?

I would regard the movement-tremors of disseminated sclerosis as an almost exact parallel to the oscillatory discharge of a Leyden jar. In this disease the chief alteration found in the diseased areas is the narrowing and breaking up of the insulating medullary sheath, and therefore the resistance to the passage of a nervous impulse will be diminished, as several different paths are opened up owing to the defect of insulation.

It may be objected that the irregularity of these tremors is their chief characteristic, and that therefore my analogy with the rhythmic oscillations of a Leyden jar breaks down. To this I would reply that as the disease is disseminated, the innervation of any one muscle may be obtained by various routes in the spinal cord of different lengths, and that therefore irregularity in the movements is just what one would expect.

(January 28th, 1909.)

The President, commenting on the second paper, said all must have noticed the fact that the pupil did not act well to light in retro-ocular neuritis, and that under those circumstances it occasionally showed those marked more or less rhythmic contractions and dilatations. But oscillations of a slighter degree were the normal condition in every pupil; under sufficient magnification it would be seen that there was no such thing as a restful pupil in the
normal eye. In some cases, quite apart from the light reflex circle, these oscillations were much exaggerated, possibly due to an excitable state of the sympathetic system. We must also recollect that changes in the size of the pupil were brought about by variations in the vascularity of the iris; in certain conditions, e.g., the pupil moved consensually with the respirations. The explanation which Mr. Percival brought forward he, of course, only meant to be applicable to certain of these rhythmic phenomena.

Mr. R. W. Doyne said he did not offer any criticism on the paper, but there was a fact in connection with innervation in cases of retro-bulbar neuritis which was not generally known, and which he, Mr. Doyne, had not published, namely, that the latent period of reaction of the pupil was reduced by about one-tenth in cases of retro-bulbar neuritis which had gone on to atrophy. Old cases of retro-bulbar neuritis, with loss of central vision, instead of having a latent period of four-tenths of a second, which was the normal, had the period reduced to three-tenths of a second. When people spoke about the pupil being "sluggish" it had an indirect meaning. The fact was that its reaction to light was quicker than normal, but the degree was less. He had measured by photometry many pupils in a series of retro-bulbar cases, and the only certain difference he found was, that while the reaction was much less in degree, the latent period, before reaction set in, was reduced. He had no doubt that the latent period was distinctly reduced by one-tenth of a second. He was not so sure about the rate of reaction, because if the reaction was very small the curve spread over a long distance was so very slight that there was great risk of error of observation. The normal pupillary oscillation varied very much.

Mr. Percival, in reply, said he had been very interested in Mr. Doyne's remarks about the latent period, but his suggestion only referred to that peculiar oscillatory action of the pupil on exposure to light which was known in
Tobacco Amblyopia—A Substitute for Smoking.

27. Tobacco amblyopia—a substitute for smoking.

By N. Bishop Harman.

Everyone knows that the only cure for tobacco amblyopia is to stop the use of tobacco. Patients do not always do as they are told; some assert the habit too strong to be broken, others promise and do not perform. On occasions circumstances make the pipe almost a necessity, and it was one of these cases that made me seek some substitute for smoking.

An out-door night-watchman suffered a severe attack of tobacco amblyopia. When told he must cease smoking and chewing tobacco, he asked: "What was he to do through a solitary night-watch of eight long hours with nothing to do?"

Substitutes for tobacco, mixtures of the petals of various flowers and the leaves of sweet-smelling plants, are sold by herbalists, but the man found them disgusting. Remembering that men engaged in powder factories or soldiers on sentry duty chew tobacco, since smoking is prohibited, I cast about for something which would make a satisfactory "chew" and provide a satisfying substitute for smoking.

Bitter flavours seemed the best, and quassia, gentian, cinchona and other bitter woods were tried. Quassia was undoubtedly the most favoured. I have given men suffering from this form of poisoning quassia chip to chew for more than a year with very helpful results; they agree that something to chew and the bitter flavour checks the retro-bulbar neuritis. It was different from the minute oscillation to which the President had referred—respiratory or circulatory oscillation. Hippus he had seen in neurotic girls, but he thought that was due to cortical excitement, and it had no bearing on his present theory at all.
desire to smoke. One and all agree that the bitter is a great appetiser.

Recently I have had the quassia chips ground into fine powder and mixed intimately with a "chewing-gum" mass.

A rough and ready confection can be obtained by melting down hard paraffin wax and stirring in the powdered quassia; this "gum" is very friable when first chewed, but it speedily becomes plastic.

Two formulae for chewing-gum bases are given in the National Druggist, 1907, No. 37, p. 20:

(1) Gum chicle, 25; hard paraffin, 8; balsam of tolu, 1; sugar, 96; water, 24; flavour as desired. Dissolve the sugar in the water with heat; melt the chicle, paraffin, and tolu together; pour the syrup on to an oiled slab; add the melted gum mass to it, and mix by kneading.

(2) Gum chicle, 60; white beeswax, 16; sugar, 160; liquid glucose, 32; water, 48; Peruvian balsam, 1; flavour as desired. Mix as above.

Messrs. Fuller, the well-known confectioners of Regent Street, London, have made up a box of chewing gum containing quassia in the proportion of one to twenty of the mass. It is made and put up with their usual elegance, and is fit for the most fastidious patient. Half a pound of this should last the most energetic chewer for three months, so the cost would work out at less than an equal weight of cheap shag.

(Febuary 11th, 1909.)


By A. Levy.

(With Plate IV, fig. 1.)

E. A—, aged 22 years, came to Moorfields on January 27th complaining that six days previously the vision in the right eye had suddenly failed.
PLATE IV.

Fig. 1 illustrates Mr. A. Levy's case of Obstruction of Cilio-retinal artery (p. 130).

Fig. 2 illustrates Mr. E. Nettleship's black-and-white drawing of Fundus, showing scattered Spots of Past Choroiditis, which are peculiar in being, with one or two exceptions, placed exactly beneath Retinal Vessels, the Vessels being invariably Veins (p. 134).
She had been a patient previously at Moorfields, having had a squint in the left eye, and the vision in the right at that time was a full \( \frac{6}{6} \).

On examination the pupils reacted well, but not quite so fully in the right as in the left. V. : R. = \( \frac{6}{6} \); L. = \( \frac{6}{18} \).

*Ophthalmoscopically.*—R.: Stretching from the outer and lower side of the disc for three to four disc diameters outwards there is a broad white patch of exudation; this patch coincides with the distribution of a vessel beginning at the outer margin of the disc, and which has been taken to be a cilio-retinal artery of perhaps somewhat larger size than usual. This artery is apparently obstructed just as it leaves the disc, and here there are two or three small haemorrhages.

The band of exudate is very much narrowed at one spot, the nutrition of this part being apparently maintained by a branch of the central artery which crosses here. The band of exudate is sharply limited above and below, but at its temporal edge it gradually fades into the surrounding healthy tissue.

The branches of the central artery are somewhat thickened, nipping the veins where they cross, and a similar condition of the vessels was noted in the other eye.

Nothing could be found in the patient's general condition to account for this eye trouble.

*(Card specimen. February 11th, 1909.)*

29. *A case of aniridia, with a family history of the condition in four generations.*

By J. F. Cunningham.

E. B—, a girl, æt. 10 years, first attended Mr. Lawford's out-patient department at St. Thomas's Hospital last summer. She was brought up on account of defective
vision. When questioned about the absence of iris, the mother said it was common in the family on the father’s side. The eyes were said to look like sloes. In some of the members affected the absence of iris was not so marked.

The patient was ordered the following correction:

R.: 3·0 D. sph. − 2·75 D. cyl. ax. horiz.
L.: 8·0 D. sph. − 2·0 D. cyl. ax. horiz.

Vision was not improved beyond $-2$ partly in each eye.

Fig. 21.

There was some iris tissue, up and in, in each eye, and this was more extensive in the right. There were also some dotted opacities on the anterior lens capsule, which were larger in the right eye than in the left.

The accompanying pedigree is from information supplied by the patient’s mother. I have not been able to examine any other members of the family yet. The mother and father were both from Oxfordshire, though in no way related. There was no family history of any eye affection on the mother’s side, and nothing suggestive in the history of glaucoma on the father’s side.

The patient is the fifth member of Generation IV.
PLATE V.

Illustrates Mr. E. Nettleship's coloured drawing of Fundus in a case of Active Syphilitic Choroiditis Disseminata with Hazy Vitreous, and probably some Deep Haze of Retina (p. 133).
Mr. Doyne has kindly furnished me with notes of two cases, one from Oxfordshire and the other from Buckinghamshire, but at present no relationship can be traced between the families and this one.

(Card specimen. February 11th, 1909.)

30. Coloured drawing of fundus in a case of active syphilitic choroiditis disseminata with hazy vitreous, and probably some deep haze of retina.

By E. Nettleship.

(With Plate V.)

The special feature is the arrangement of certain of the choroidal foci in chains, following exactly the course of retinal veins. Three such well-marked chains are seen in the upper half of the drawing (inverted image); there are no chains in relation to retinal arteries.

The patient, a woman, aged 45 years, had had syphilis a year or year and a half previously, and iritis had occurred in the eye here represented; she still had mucous tubercles and other symptoms.

Vision of this eye, fingers at 3′; a month later, after mercurial inunction, \( \frac{9}{36} \) and 10 J. No further improvement owing to the severe damage at Y.S.

In connection with this and the drawing next described it is of interest to note that in retinitis pigmentosa it is the veins—never the arteries—that become pigmented.

(February 11th, 1909.)
31. Black-and-white drawing of fundus, showing scattered spots of past choroiditis, which are peculiar in being, with one or two exceptions, placed exactly beneath retinal vessels, the vessels being invariably veins.

By E. Nettleship.

(With Plate IV, fig. 2.)

The patient was a man, æt. 26 years, who was at Moorfields Hospital in February, 1891.

The notes of the case have, unfortunately, been lost.

(Febuary 11th, 1909.)

32. Ophthalmoscopic drawing to show: Hæmorrhages, probably choroidal, at macular region of a highly myopic eye, apparently caused by a blow on the corresponding eyebrow. There was also hæmorrhage into the vitreous and around the perforating anterior ciliary arteries.

By E. Nettleship.

(With Plate VI, figs. 1 and 2.)

Sarah W—, æt. 53 years, at Moorfields Hospital August, 1889, for recent failure of R. (the better) eye, noticed since a fall two weeks previously, in which she struck the R. brow; no proof that the eye itself was struck. V. with −15 or 16 D. $\frac{6}{36}$ barely; much opacity in vitreous. Drawing made at this date to show central region of fundus more than half covered by well-defined, rounded blood-patches of various sizes; some of the very small ones are darkest at centre, but the others are uniform in tint. Retinal vessels pass unobscured in front of the hæmorrhages and there is no trace of exudation or retinal haze. The large, mainly central, area of partially atrophied choroid is sharply bounded on its temporal side by a curved border of healthy-looking choroid, suggesting the edge of a true posterior staphyloma (Pl. VI, fig. 2). The hæmorrhages have probably come from small choroidal
PLATE VI.

Illustrates Mr. E. Nettleship's ophthalmoscopic drawing to show: Hæmorrhages, probably Choroidal, at Macular Region of a Highly Myopic Eye apparently caused by a blow on the corresponding Eyebrow. There was also Hæmorrhage into the Vitreous and around the Perforating Anterior Ciliary Arteries (p. 134).
HEMORRHAGES, PROBABLY CHOROIDAL.

vessels on the macular portion of the atrophic area. The L. eye, with 16 D. of My., was very amblyopic (less than $\frac{6}{6}$ with correction) from gross atrophic changes at Y.S., but presented no haemorrhages.

In the ciliary region of the R. were eight or ten perforating anterior ciliary arteries, each one surrounded by a narrow zone of blood-staining where it entered the sclera. A drawing of this was also made and is shown (Pl. VI, fig. 1).

She was examined again a year and a half later (January, 1891). The haemorrhages at R. fundus had quite disappeared, leaving much choroidal atrophy, partly in the linear form, and some irregular pigmentation at Y.S. Some opacities still seen in vitreous; V. about the same as before. (February 11th, 1909.)

Mr. J. B. Lawford asked whether the pigmentation along the track of the veins was seen in old degenerate eyes, or in true retinitis pigmentosa. Mr. Lawford said the reason he had asked the question was that years ago he believed in a particular specimen both arteries and veins were sheathed by pigment.

Mr. W. T. Lister said that in an old case of retinitis pigmentosa which he examined one could see that the large vessels were pigmented, but he could not remember whether they were arteries or veins especially. He would look the matter up.

Mr. Doyne said that in many cases of retinitis pigmentosa there was no pigmentation at the extreme periphery of the retina, and that would not bear out the view which Mr. Nettleship had advanced.

Mr. Nettleship, in reply, said that in retinitis pigmentosa the extreme periphery was usually free from visible disease, and was often functionally good, but he did not know that that necessarily negatived the notion, because pigment started from the capillaries. It was not necessary to assume that pigment started from the extreme periphery of the fundus.
33. *Pituitary neoplasm with ocular symptoms in a child, æt. 10 years.*

By Ilbert Hancock.

(With Plate VII.)

Emily S—, æt. 10 years, first came to Moorfields under Mr. Treacher Collins on May 4th, 1908, with a history of defective sight in the left eye, noticed fourteen days. The child for about a month had complained of headache, and her mother had noticed that her face swelled at times, more especially on the left side. No history of vomiting, vertigo or fits. Patient was admitted into Moorfields on the first day of her attendance at the Hospital.

*In-patient notes.*—Patient is one of a family of seven, all the others being quite healthy. No history of tubercle or syphilis in the family. General condition good. Heart and lungs normal. Urine: sp. gr. 1010, and no sugar, no albumen, no polyuria. Mouth very septic, several carious stumps.

R. V. = \( \frac{6}{24} \), J. 12. Fundus normal. Pupil, sluggish reaction to light; reacts to convergence. Fields not taken.

L. V. = No P.L.; pupil inactive to direct stimulation—reacts consensually; no local pain or tenderness on pressure upon globe. Temporal half of disc pale; movements of both eyes perfect. Treatment, hyd. čret.

May 11th, 1908.—R. \( \frac{6}{18} \), L. = faint P.L. Several carious stumps extracted.

May 29th, 1908.—R. \( \frac{6}{15} \), 2 letters; L. fingers in temporal field. Pupil: Slow reaction to direct stimulation, but badly maintained; reacts to convergence and consensually; pallor of temporal half of disc; no sign of neuritis.

May 30th, 1908.—Discharged.

January 14th, 1909.—R. V. = \( \frac{6}{5} \), J. 1. Disc pale all over, vessels normal; no evidence of neuritis. Field: Temporal hemianopsia (see Chart); pupil reacts briskly to
direct stimulation; doubtful Wernicke reaction. L.V. = no P.L.; disc atrophic, vessels good. Pupil inactive to light, reacts to convergence and consensually.

General condition.—Weight: 6 stone. Mental: Has been quite bright at school, passed fifth standard; mother has noticed no deterioration.

Physical condition.—Head large, but no special enlarge-

![Diagram](image)

Emily S—, February 8th, 1909.

ment of supra-orbital ridges. Face: Left side distinctly more puffy than right; no enlargement of lower jaw. Teeth well separated from each other. Nose rather large and broad.

Body.—Thorax broad and deep. Slight antero-posterior curve in cervico-dorsal, and lordosis in lumbar region. Bones: No definite enlargement of clavicles, sternum, or ribs. Pelvis well formed, perhaps rather large. Extremities: Arms large, hands large and broad; legs large, feet large and broad (mother states that during the last nine months the child has grown out of three sizes in vol. xxix.
boots, and now wears women's No. 1). Hair natural, no pubic hair. Skin: Subcutaneous tissues, slightly thickened. Well marked excessive development of adipose tissue all over body, not more marked on one side than the other. No excessive perspiration. No enlargement of lips or tongue. Nails: normal. Abdomen pendulous. There is a definite difference in the size of the two halves of the body. The left side is larger than the right (mother states left side of body often swells). This enlargement is both in the direction of length and breadth, and is present in face, chest, arms and hands, legs and feet. Child is left-handed. Thyroid: No enlargement. Thymus, no evidence of enlargement. Voice deeper than normal for her age. Respiration abdominal in type, chest healthy. Pulse 80. Heart-sounds normal.

Nervous system.—Taste, smell, and hearing good. Cranial nerves nil, except optic nerves. Motor power good, nothing abnormal.

Sensory.—No change. Reflexes, superficial and deep, normal. Organic reflexes normal.

During her stay in the hospital it will be noted that the R.V. improved from \( \frac{6}{24} \) to \( \frac{6}{2} \) and is now \( \frac{6}{9} \) full, and L.V. from no P.L. to fingers in temporal field. On the discovery of temporal hemianopsia in the right eye pressure on the chiasma at once suggested itself, and an X-ray photograph kindly taken by Mr. Mackenzie Davidson shows the presence of a growth in the region of the sella turcica. It is difficult to explain the recovery of vision in the temporal field of L. eye whilst in the hospital. The general aspect is of a stunted, heavily built child older than ten years, but when the age is considered and the child more carefully examined there can be no doubt there is a distinct over-growth. The general features have a superficial resemblance to myxœdema, but the enlargement of the bones, especially of the hands and feet, the mental condition and ocular changes are against this diagnosis. Although by no
means typical of acromegaly, the visual changes, the enlargement of hands and feet, and change in spine and thorax suggest that the case is an early one of that disease. The excessive development of fat is very marked, a clinical feature frequently noted in association with tumour in and around the pituitary body.

References.

Unthoff.—Bericht. über die 34 vers der Ophth. Ges., Heidelberg, 1907.
Mr. Mackenzie Davidson's report.—Several X-ray photographs of the head taken laterally all show a small roundish body situated in the brain a little in front of the "sella turcica," and about half an inch above the base of the skull; it is a little to the left of the middle line. Its situation can be well seen in the stereoscopic transparencies shown in the revolving stereoscope. X-ray photographs of the hands and feet do not appear to show anything abnormal, except perhaps their size.

(Card specimen. February 11th, 1909.)

Mr. Mackenzie Davidson said the patient was sent to him to see if X-rays would show anything abnormal in the brain. He had often tried in such cases, but without success. But in this case there was something very definite in the region where a lesion was suspected. The picture had been taken stereoscopically, and measurement showed the spot to be in the brain substance about half an inch above the base of the brain, and a little in front of the sella turcica, so possibly some interference with the pituitary body was confirmed by the appearance. It was very small and very dense, therefore must contain lime, because the degree of opacity to X rays was in proportion to the atomic weight of the substance. Nothing normal to the brain itself could give such a shadow as that. It might be only the densest part of a larger area. He had taken many negatives of the brain to try to detect lesions there, but had never seen such an appearance before.

Mr. Leslie Paton said he had seen a case of similar nature. He had seen the patient at intervals during the last six years. That boy developed primary optic atrophy in both eyes, evidently due to pressure on the chiasma. He had been in the National Hospital at various times, and at one time he developed so much adipose
PLATE VII.

Illustrates Dr. Ilbert Hancock's case of Pituitary Neoplasm with Ocular Symptoms in a Child æt. 10 years (p. 136).
tissue that one of the house-physicians there suggested it was feminism associated with optic atrophy. The boy had pubes like a female, and well-developed breasts, and general feministic traits well marked. Later the patient became very irritable and lost his temper on slight provocation, and developed symptoms showing involvement of the frontal lobes. He now had a large tumour involving both frontal lobes from the pituitary region. He was now eighteen, and he believed was an inmate of an asylum.

Mr. Macnab: The case before us seems to present very distinctly the associated signs of optic atrophy and adiposity. The association of these two signs is a distinct indication of a tumour in the region of the chiasma. A great many sections are available which prove this conclusively. The first recorded is by Mohr in 1841. The question was discussed in this Society in 1887, and before the Ophthalmological Society in Heidelberg in 1907. The cases belong to a well defined clinical class, the chief features being disturbance of growth, cryptorchism, adiposity, and optic atrophy. The lesion is probably not necessarily one of the hypophysis itself, as tumours of the infundibulum and parts around have given rise to the same symptoms. The cause of the adiposity appears to be pressure on the basal centres in the neighbourhood. It is not due to the associated cryptorchism, as there is a case on record in which the sign occurred in a wound of the base not affecting the hypophysis, and where the sexual organs were normal. The present case shows adiposity more on one side than the other; this was also the case in Nieden's patient, in which the left side was first affected, but later the increase in fat became general. It would be a mistake to suppose that every tumour of the hypophysis caused a bi-temporal hemianopsia; in 22 cases in which the diagnosis was confirmed post-mortem, 23 per cent. were bi-temporal, 23 per cent. unilateral temporal, 22 per cent. concentric, 13 per cent. central scotoma, 9 per cent. homonymous, 9 per cent. only sector remaining, 4 per cent. irregular. There are no figures available to show
what percentage of tumours in this region cause this adiposity, but it seems to be quite as frequent as the 23 per cent. of bi-temporal hemianopia.

The Chairman (Mr. Nettleship) said an interesting point concerned the occurrence of scotoma in such cases in quite an early stage. He had been puzzled by it in the early stage of more than one case. It turned out to be the beginning of what eventuated in bi-temporal hemianopia, beginning from near the centre (Transactions, vol. xvii, p. 277).

Mr. J. H. Fisher, referring to Mr. Macnab's remarks that there was a similar train of symptoms, whether the lesion was one of the pituitary body or in the region adjacent thereto, asked, was it possible, by an X-ray picture, to determine whether there was an enlargement of the bony sella turcica? When the pituitary body lying in the sella turcica beneath the diaphragma sellae enlarged, it caused enlargement of the bony cavity. If the X-ray pictures enabled one to say there was an enlargement of the sella turcica, one would infer the case was one of pituitary body tumour and not an extra-pituitary growth pressing on the pituitary body and the adjacent neighbourhood. He raised the point because he had treated a case of bi-temporal hemianopia by thyroid extract, with remarkable improvement, both in direct vision and in the field in the only eye with which the patient was seeing at the time he came for advice. Another case was recorded by Mr. Richardson Cross, which also terminated favourably by that method of treatment. These two cases were presumably cases of hypertrophy of the pituitary gland. The practical importance of being able to decide whether the lesion was primarily pituitary or extra-pituitary was therefore obvious. In his own case the improvement had been maintained for years by occasional courses of thyroid extract, and the patient had been rendered able to support himself and his wife by constant work.

Mr. MacKenzie Davidson said he could give approximately the size of that sella turcica, but he could not say what
Cyst of the Iris.

By E. W. Brewerton.

M. S—, æt. 28 years. The right eye was excised by the late Mr. John Griffiths in January, 1898. The eye was blind and painful from long-continued inflammation, which was believed to be due to tubercle.

In 1903 the left eye was affected with recurrent plastic irido-cyclitis. The pupil became blocked with lymph and the lower half of the anterior chamber filled with organised exudation. The vision sank to fingers at ten inches.

In April, 1906, as there had been no inflammation for more than six months, an optical iridectomy upwards was performed. The vision later reached \( \frac{5}{60} \).

In February, 1907 (ten months later), a small black spot appeared in the lower half of the anterior chamber. Tension + 1. From this time slow general enlargement of the eye has taken place; the black spot has increased in size, and now measures 10 mm. horizontally by 5 mm. vertically. The tension has remained high since the spot appeared. During the last two months pigmentary staining of the neighbouring sclerotic has shown itself. Vision, now, only hand-movements.

By transillumination the dark areas are found to transmit light readily.

*Family history.*—Mother and father both died of phthisis. One sister has had haemoptysis, another sister died of "consumption of the throat."

(*Card specimen. February 11th, 1909.*)
35. Two cases of hereditary optic atrophy in a family, with recovery in one case.

By Rayner Batten.

Albert S—, aged 10 years (1904), attended at the Western Ophthalmic Hospital, complaining of rapid failure of sight which his mother had only noticed two days, but the patient thought he had not seen well for about a month. V. : R. = $\frac{6}{6}$, J. 14 at 6 ; L. = $<\frac{6}{6}$, J. 19.

At the time of his first attendance I failed to obtain any family history, and the correct history was only obtained subsequently.

Ophthalmoscopic examination did not reveal any sufficient cause for the defective vision, the only changes being—R. O.D. rather pale at outer margin; retinal vessels not diminished; retina dotted with fine dots, "red pepper." The macula is somewhat oedematous showing fine light rings, "water silk retina." Left fundus a similar condition. O.D. appears normal.

In September, 1904, after the vision had begun to improve the O.D.s. were noted as being rather pale and
TWO CASES OF HEREDITARY OPTIC ATROPHY.

Fig. 26.

RIGHT

Albert S—, June, 1904.

Fig. 27.

LEFT

Albert S—, June, 1904.
TWO CASES OF HEREDITARY OPTIC ATROPHY.

Fig. 28.

RIGHT.

Albert S—, September 26th, 1904.

Fig. 29.

LEFT.

Albert S—, September 26th, 1904.
TWO CASES OF HEREDITARY OPTIC ATROPHY.

Fig. 30.
RIGHT.

Albert S—, March, 1905.

Fig. 31.
LEFT.

Albert S—, March, 1905.
TWO CASES OF HEREDITARY OPTIC ATROPHY.

Fig. 32.

RIGHT

Albert S—, February 1st, 1909.

Fig. 33.

LEFT

Albert S—, February 1st, 1909.
excavated. The maculae showed slight pigment change, but no evidence of any retinal oedema.

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He was treated with potassium iodide and arsenic.

Harold S—, aet. 22 years (1907). His sight began to fail when about sixteen years old. Failed gradually. The failure was not associated with any illness.

*Fundus examination.*—O.D. pale, atrophic; large retinal vessels not much reduced in size.

1909 V. = R. $\frac{6}{6}$. J. 14 at 5 in.; L. $\frac{6}{6}$. J. 20 at 6 in.

There is no material alteration in his condition since first seen in 1907, except that the visual fields appear slightly more constricted.

A cousin of these two cases, Arthur G—, aet. 20 years (1895), attended under Mr. Doyne at Oxford, who has kindly sent me the notes of his case.

He was then described as a case of retro-bulbar neuritis with symmetrical retinal haemorrhages at the margins of the optic discs and water-silk undulations of the macula.

The sight began to fail three weeks previous to his attendance and was reduced to R. and L. $<\frac{6}{6}$.

He was under treatment till 1900, but without improvement. The haemorrhages absorbed and the O.D.s. became very white. There was a central scotoma for white and colours.

In March, 1900, he developed nervous symptoms, cramps in both feet, numbness of tips of fingers, tremors of lips and chin. The knee-jerks were absent. The pupils reacted well. (*Card specimen. February 11th, 1909.*)

Mr. Doyne alluded to one of the cases in the family tree, that of Gibbens, in connection with whose case there were points of great interest, for with the onset of symptoms there was a good deal of flame-shaped haemorrhage accompanying the vessels from each disc but with no
definite evidence of swelling. The case was again seen some years later, when other symptoms had developed, i.e., loss of knee-jerks and tremor of the mouth.

36. Case of supposed congenital lack of separation of M. levator palpebrae superioris and M. rectus superior; unilateral.

By N. Bishop Harman.

D. S——, a healthy, well-developed girl, æt. 10 years, attended Mr. Treacher Collins' clinic at Moorfields, February 4th, 1909.

The father said she did not appear to open the eye properly for the first three days after birth. No instruments had been used in delivery. The eyes had always been odd.

On examination the position of the eyes and the eyelids were found to be asymmetrical. There was an alternating convergent and vertical squint, and the right palpebral fissure was wider from above down than the left.

The child could fix with either eye, but the left was most often employed. The appearances differ according as right or left eye fixed.

(1) Right eye fixation.—Right eye and lid look quite normal. Left upper lid droops 3 mm., and left eye turns slightly down and in.

(2) Left eye fixation.—Left eye and lid look quite normal. Right eye turns up and in; the lid is retracted upwards, but the inner part more so than the outer part.

(3) Concomitant movements.—The movements of the left eye are perfect and full in all directions. The movements of the right eye are imperfect in extreme elevation; it fails to follow the left eye by 3 mm.

(4) Vision.—R. = 6/6 partly, H.m. 1 D.; L. = 6/6 perfectly, H.m. 1 D. Pupils equal, reactions normal.

Remarks.—The defect appears to be with the right eye.
The history given by the father that the left eye was irregular for a few days after birth would appear to be a coincidence and have no bearing on the condition seen now.

The lack of full movement upwards of the right eye fixes the defect in the elevator musculature of that eye. On the other hand, there is the singular feature of a higher level of rest, or an upward and internal displacement of the right eye and lid when the left eye fixes.

The defect of the right elevator musculature is not a simple insufficiency. The musculature is in some positions too effective, in other positions it is insufficient.

The only reasonable explanation I can bring forward to meet these phenomena is that there has been a failure in the separation of the original muscle mass which goes to form the two muscles known as the levator palpebrae superioris and rectus superior. The levator is really split off from the original rectus mass. The state of separation is complete in man, so that in most subjects the two muscles can work independently. This is the only case
in man I have seen that suggested an incomplete separation. Incomplete separation is very common in lower vertebrates where there are attempts at the development of lid retractors; in the Orhagaviscus mola, the big sun-fish, the rectus superior passes under the obliquus superior muscle, and the homologue of the levator of the lid passes superficial to the oblique muscle, though in its origin still forming part of the original rectus mass. Similar but lesser separations to form lid retractors occur in the internal and inferior recti and the inferior oblique, but there is no division of the external rectus.

Such an incomplete division in this girl might be held to explain the phenomena seen. In the horizontal line too much work would be done, and both eye and lid would turn up and in. In extreme elevation the rectus superior would be hampered by its bed-fellow, and fail to make the right eye follow the movement of the more highly equipped left eye. *(February 11th, 1909.)*

37. Quinine amblyopia from a single dose of quin. *sulph.* 5j.

By L. V. Cargill.

For the notes of this case I am indebted to Dr. T. Herbert Bell, late House Surgeon Royal Eye Hospital.

E. M—, female, aet. 28 years; married. First seen December 3rd, 1908.

Sight had failed nine days previously—the morning after taking three pennyworth of quinine, which amount proved to weigh 5j.

For one day she could not find her way about the room. Had severe tinnitus and was very deaf and dizzy.

Vision has gradually improved, though she cannot distinguish colours.
38. Rodent ulcer of right eyelid and nose, part treated by zinc ionisation and part by X rays.

By L. V. Cargill.

G. E—, aet. 80 years. Ulcer on right side of nose noticed quite fifteen years ago, that involving the eyelid about five years.

Has had no treatment up to his being sent to the Electrical Department at King's College Hospital on October 29th, 1908.

The ulcers presented the usual typical appearances, the one on the nose being about the size of a threepenny-
piece. The right lower eyelid was markedly drawn down and everted.

The treatment carried out by Mr. Gilbert Scott was as follows:

October 29th, 1908.—The nasal ulcer was ionised with

\[ \text{ZnSO}_4 \] 2 per cent., the positive electrode about 2 m.a. being used for seven minutes. This was sufficient to loosen the scab.

Two days later, the scab having come off and the ulcer being clean, it was again ionised as before for twenty-five minutes, using about 3 m.a. When finished the ulcer was pearly white in appearance.
The ulcer was quite healed in nineteen days.
On December 4th the scar was ionised for twenty minutes on account of a slight ridge noticed in it. No application since.
The ulcer involving the eyelid is being treated by

**Fig. 36.**

X rays, and up to date has received twenty sittings, averaging seven minutes twice a week until lately.

The interest in this case lies not so much in the treatment by ionisation, which is by no means new, as in the relative rapidity of healing. One ionisation—the first sitting being for the purpose of removing the scab—was sufficient to heal the one ulcer in nineteen days, whilst
with twenty applications of X rays, extending over nine weeks, healing is not yet complete.

The treatment by ionisation was first introduced by Leduc in a paper read before the International Congress of Electro-Biology in Paris, entitled, "Introduction des Substances Medicamentueuses dans le Profundens des Tissus par le Courant Electrique." In 1903 Leduc published a case of rodent ulcer cured by zinc ionisation. In this country Dr. Lewis Jones published other cases in 1905-06, and in February, 1908, he read a paper before the Royal Society of Medicine on "The Principles of Ionic Medication," which was a complete exposition and resumé of the subject.

(Card specimen. February 11th, 1909.)

39. Optic atrophy possibly due to haemorrhage into optic nerve sheath.

By W. Lang.

E. F. B—, aged 42 years, was seen on February 6th, 1909, the day he arrived from Johannesburg, where, when working in a stooping position at the bottom of the shaft of a gold mine on November 2nd, 1908, a bucket full of wood, weighing one and a half tons, descended on his back and flattened him out. It rested there for one and a half minutes before it could be raised again. He was rendered unconscious for a short time. His blood was pressed out of his body into his head, causing his eyes to protrude and blood to issue from his nose and mouth. He recovered consciousness very soon, but was blind for fourteen days. He left the hospital at the end of a month, seeing a little with the R. and nothing with the L.

Present condition.—R.: ¾ not improved; field contracted; lower part of disc pale; large vessels good size. L.: no P. L.; P. reacts consensually only; disc atrophic;
main vessels large; cannot converge, but no other loss of power of ocular movements.

It is probable that the blood was pressed out of the abdomen into the head and face. He remembers feeling his eyes bulging out before he lost consciousness. The

![Diagram](image)

E. F. B—, February 6th, 1909.

skin of the face was black for some time, and it is probable that haemorrhage took place into the optic nerve sheaths.

(Card specimen. February 11th, 1909.)

40. Specimen of oxycephaly (crâne en forme de tour—steeple skull) of an infant, wt. 8 weeks.

By Dr. George Carpenter.

The head has been split vertically in the middle line, the section passing through the spine and displaying on
one side the spinal cord intact. Viewed in front the face and skull combined are diamond shaped—vide pictures of child during life.* A side view shows the skull to be compressed antero-posteriorly; the occipital bone is dented inwards, the frontal bone is flattened and thrust backwards. The parietal bones are narrowed at the vertex, and the skull is carried upwards is the shape of a truncated cone. The orbits are very shallow, and elongated in the vertical diameter; their margins are egg-shaped, set slightly obliquely, and their long diameters, if projected upwards, would meet at a point in the middle line one inch above their superior margins.

The brain, which is in situ, is bent on itself so that the frontal and occipital lobes are only about one and a half inches apart, the cerebrum being curled in the shape of a loop and fitting into the distorted skull. Laterally during life the brain bulged over the sites of the anterior lateral fontanelles, which were very large, covered by membrane, and measured three inches by one and three quarter inches.

The eyes during life were protuberant and readily dislocated. There were no changes in the fundus oculi.

The spinal canal shows a haemorrhage. The haemorrhage is apparently outside the dura mater.

The child was shown during life at the Section for the Study of Disease in Children of the Royal Society of Medicine in December, 1908. It had six toes on each foot, and the middle and third fingers of each hand were webbed. There was a ventral hernia above the umbilicus, and just before death it developed a large inguinal hernia.

Two sisters, since dead, were shown eight years ago to the Society for the Study of Disease in Children (vide Reports, vol. i, p. 110). Both had malformed skulls, though not to the extent of the exhibit; they also had malformations of the fingers and toes. Parents both healthy—no syphilis. The third child, act. 7 years,

photo shown, is now alive and quite normal. The fourth child was premature, and is stated to have looked like the two former children. The fifth child is the present exhibit.

(Card specimen. March 11th, 1909.)

The Chairman said it was a typical example of the condition, and he believed Mr. Sydney Stephenson had already shown a case.

41. A case of unilateral optic neuritis with white spots in the retina.

By J. Herbert Parsons.

Rose A——, act. 16 years, tailoress, was admitted to the Royal London Ophthalmic Hospital as an out-patient under me on January 30th, 1909. She complained of not being able to see with the left eye, a condition noticed one week previously. The loss of vision came on suddenly when out walking. She had not been feeling well for some time and had been treated for anaemia. Three weeks previously she had nausea in the morning, accompanied by frontal headache which lasted all day. There was no actual vomiting except on one occasion. She has had measles, scarlet fever and anaemia; no rheumatism, no nasal discharge.

Family history.—Father suffers from asthma; mother is said to have weak sight. Seven brothers and sisters are all healthy; there is no history of tubercle in the family.


There is a rhythmic action of the pupils on each side to light; both react well both to light and convergence.

The left disc shows marked oedematous swelling (+ 3 D.); the outlines are blurred and the oedema extends into the surrounding retina, the macular region
being involved. The vessels are quite lost to view on the disc, appearing at the upper and lower margins. The veins are tortuous and somewhat dilated, but not to any extreme degree. The arteries appear to be disproportionately small; where they cross the veins the latter are constricted. There are a few small streaky haemorrhages on the nasal side and below the disc. The periphery of the fundus looks normal.

The right fundus shows shot-silk reflexes, but the disc, vessels, etc., appear to be quite normal.

The right field is normal. The left field, not very reliable, is contracted.

February 6th.—The left vision is now counting fingers at two feet. Since the last visit bright white spots have appeared around the fovea, forming an incomplete macular star resembling that usually associated with albuminuric retinitis.

The urine is acid, of sp. gr. 1010, containing no albumen or sugar.

For more thorough observation and investigation of the general condition the patient was transferred to University College Hospital. On February 11th she was admitted an in-patient under Dr. J. Rose Bradford, to whom I am indebted for the medical notes.

Present state.—Examination of the chest, heart, abdomen and nervous system fails to show any physical signs of disease. The temperature is slightly subnormal. Urine normal.

Blood-count.—Red corpuscles, 3,650,000 (slight vacuolation); haemoglobin, 70 per cent.; colour index, 0.9; white corpuscles, 35,000; polymorphonuclear neutrophiles, 43 per cent.; polymorphonuclear oxyphiles, 8 per cent.; large lymphocytes, 32 per cent.; small lymphocytes, 14 per cent.; hyaline, 2 per cent.; transitional, 1 per cent.

The blood-count shows some peculiarities, especially in the excess of white corpuscles, but it is not indicative of anything more than a slight anaemia.

February 15th.—Left fundus much the same as on
February 6th, but there are a few spots around the disc and close to it. They are white and sharply defined, and are most numerous above the disc. They are about the diameter of a retinal vessel or slightly larger. The vessels pass superficial to them.

February 22nd.—The neuritis is subsiding. There are more white spots around the disc. The spots at the macula remain the same. The fovea appears as a reddish-grey area rather sharply defined.

L. V. = fingers at 2 in.

March 11th.—Left fundus: Media clear, the temporal and nasal edges of the disc are sharply defined, and no swelling can be made out. The physiological cup is filled in, and the vessels on the disc are obscured. Extending below the disc for 1 1/4 p.d. is a whitish patch, which obscures the lower vessels and tails off along the lower temporal artery. The vessels look about normal in calibre and show no signs of disease. At the macula there is an imperfect macular star, the small white spots being mostly above the fovea. Around the disc, especially above it, are numerous whitish round spots of various sizes up to about twice the diameter of a vessel. They all lie at a lower level than the vessels, which pass over some of them. They show a slight mottling and are dull, not glistening. Towards the periphery below, probably near the equator of the eye, are innumerable glistening, small white spots peppered about over a considerable area. They are mostly intermediate in size between the largest and smallest spots near the disc. They differ from them in being quite uniform in colour over their whole surface, denser in appearance, and very bright, but resemble them in being at a deeper level than the retinal vessels.

Remarks.—The cause of the condition is extremely difficult to conjecture. The case shows some similarity to two previously shown at the Society by me (vol. xxvii, p. 121; vol. xxviii, p. 161). There is less foundation for a diagnosis of partial thrombosis of the central vein in this case than in the first of those referred to. There is
perhaps more evidence of metastatic bacterial invasion than in the second. It is, however, founded on very slender data. Cases have been described of white spots in the retina under the designation retinitis septica (Roth). Roth’s paper was published in 1872, and although the condition, arising from a septic cause, has been confirmed by various observers since that time, considerable doubt as to the identity of the condition must be admitted. Similar white spots have been described in various other diseases, notably various forms of anaemia. They are not, however, usually accompanied by such an extreme degree of papillœdema. That the spots are probably retinal exudates or the result of retinal exudates deeply seated in the retina, and not of choroidal origin, is indicated by the fact that all are sharply defined from their first appearance. There is no evidence whatever in the patient of tubercle—nor, indeed, of any septic focus. The spots differ in appearance and distribution from those of retinitis punctata albescens, which is entirely negatived, too, by the absence of other characteristic signs of that disease. 

(March 11th, 1909.)

The Chairman asked for the history of the case and whether there were any vitreous opacities, also what Mr. Parsons thought it was. He thought it looked more like tubercle owing to the very little pigment and the absence of vitreous opacities. He asked whether Mr. Parsons found any œdema of the retina in connection with the upper spots. His own impression was that they were rather deep down, perhaps in the choroid, as they had a flat appearance, and might be the remains of solitary tubercles. He had himself a case somewhat analogous.

Mr. Herbert Parsons, in elaboration of the case, said that he saw the patient first at the end of January immediately after she had noticed the loss or almost complete loss of vision in that eye. She had unilateral optic neuritis; there was 3 D. swelling and very little disturbance of the retina. During the next week or two the white spots developed in
the neighbourhood of the disc, and a star similar to what was now seen developed at the macula. He could not say exactly when the peripheral spots appeared; he only noticed them at a comparatively late stage. There were no vitreous opacities at the beginning, and he thought there were none now. He did not know the cause of the condition. Some time ago he showed a case, which was reported in the Society's Transactions, of a girl who had had the sudden onset of neuritis in the same way, with much oedema of the retina, a few haemorrhages and very dilated veins. She had nothing the matter with her that he could make out except that she missed a few menstrual periods. She was carefully examined by a physician with negative results. He attributed the condition to partial thrombosis of the central vein. The present girl appeared to be healthy, except that she had slight anaemia. Dr. Bradford did not consider that she had signs of any well-recognised form of anaemia. If it were thrombosis at all the obstruction could not be complete. A group of cases had been recorded under the designation "retinitis septica," described originally by Roth, the characteristic features of which were white spots dotted about the fundus. It was difficult to know from the literature what the ophthalmoscopic picture of retinitis septica was. Subsequently similar white spots had been described in leukaemia, various anaemias, diabetes, and other conditions. There was no doubt white spots formed in some forms of metastatic invasion of the retina, and some of those spots had been examined microscopically and the actual organisms found in them. It was characteristic of organisms in the eye coming by endogenous means that they were liable to die out comparatively early, and that must be taken into consideration in reflecting whether here one had to deal with a metastatic septic partial thrombosis. The girl had no sign of tubercle; she had had the von Pirquet's test applied and it did not come off. He did not know whether it was of any use; probably not. One other case of optic neuritis was shown in a woman,
aet. 50 years, who developed a star of the macula, and she also was peculiarly healthy. That quieted down and there were no white spots in the case. He did not think with Mr. Jessop that the lesion referred to could be in the choroid, because the spots appeared at first very much as they were now. Had they been there he would have expected to see an appearance similar to that of choroidal tubercle, with yellowish colouration and an infiltrated retina over it. There was an extraordinary difference in the appearance of the spots at the disc compared with those at the periphery.

Dr. F. E. Batten said Mr. Parsons appeared to question the value of the von Pirquet reaction. He believed the reaction to be one of considerable value as establishing the negative, but he admitted that a positive reaction was of little value, since many apparently healthy adults gave a positive reaction.

Mr. Parsons rejoined that he had not criticised the reaction so much as expressed his ignorance of it.

Mr. Bishop Harman said that he had recently seen a case which was considered clinically to be typical of tubercular irido-cyclitis; there were the usual heavy precipitates on the cornea, and the granulomata at the base of the iris in a girl, aet. 18 years. The reaction of von Pirquet was tried twice and on each occasion with negative result. It is true no diagnosis can be certain where no tubercle bacilli are obtained, but from the clinical appearances of the case one would have no hesitation in saying it was one of tubercular iritis.

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42. Optic atrophy (retro-bulbar type) with cerebellar symptoms in a child.

By L. Guthrie and M. S. Mayou.

William S—, male, aet. 9 years, was admitted to Paddington Green Children's Hospital on January 14th,
1908, complaining of loss of sight, headache, and unsteadiness in the limbs.

Family history.—Father and mother alive and well; one other child, healthy; one stillborn; one miscarriage.

Past history.—Patient has always been a healthy child, but has had whooping-cough, chickenpox, scarlet fever, measles, and diphtheria.

History of present attack.—The patient was in perfect health until three months ago, when he began to complain of constant frontal headache. No vomiting. The day before his admission he became suddenly blind in his right eye, and later in the day the sight of the left eye also became defective.

On admission.—Eyes: R.V., can barely count fingers; L.V., no perception of light. Pupils: The left is almost inactive to light; the right sluggish. Both discs show slight pallor, which especially affects the macular bundle. The retinal vessels are normal in size. No nystagmus or ocular paralysis.

Speech is very slow and deliberate, the boy taking a long time to answer questions, and the memory seems poor.

General examination.—The lower limbs show marked spasticity, with great exaggeration of the knee-jerks and well-marked knee and ankle clonus. Babinski’s sign is not present. Abdominal reflexes are present. There is no alteration in the sensations. The gait is staggering, with a strong tendency to fall towards the left. There is no dragging of the toes or Rombergism. The heart and lung sounds are normal, and nothing can be felt in the abdomen.

Present condition.—Since admission the child has improved gradually. He can now walk fairly well without aid, except that there is a tendency for him to lean, and even at times to fall, towards the left side.

Eyes.—Vision, R. and L., can count fingers at six feet. Pupils still show a sluggish reaction. The peripheral fields, to hand movements, are good, but there is evidently
a large scotoma present. Both discs show a marked pallor (atrophy) in the temporal half.

_March 11th, 1909._

Dr. F. E. Batten asked whether lumbar puncture had been performed in the case, as this in the acute stage of the disease might have helped the diagnosis. The history of the case would scarcely fit in with encephalitis, seeing that the boy had had symptoms three months before the acute onset of blindness, he thought it accorded much more with hydrocephalus. He would at once negative the diagnosis insular sclerosis, since there was no pathological proof that that disease occurred in young children.

Dr. Guthrie replied that lumbar puncture was not performed nor had the von Pirquet reaction been taken, but he would try the latter. Though he was not quite prepared to believe it was hydrocephalus, he thought it highly probable.

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43. **Tumours of the ciliary portion of the iris in both eyes.**

By Walter H. Jessop.

Mary Ann O—, aet. 17 years, was admitted in the wards of St. Bartholomew's Hospital on March 2nd, 1909. She had been attending as an out-patient, under Mr. Spicer, since January 25th, 1909. She had erythema nodosum and an attack of left facial paralysis in November, 1908. The facial paralysis lasted about six weeks, the treatment being salicylates and aspirin.

There are no marks of congenital syphilis. Family history _nil._

The note of January 25th says: Right eye: ciliary congestion; many large, fleshy remains of posterior synechiae; pupil does not dilate well below; pigment on anterior capsule. Ophthalmoscopically, fundi normal.
V. : R. $\frac{6}{9}$; L. $\frac{6}{6}$. Treated with mercury and iodide of potassium, and atropine locally.

For the last fortnight the left eye has been inflamed, and she has not been able to open it.

Present condition.—Right eye: Very slight conjunctival congestion; cornea, old K.P. below; pupil under mydriatic irregular. There are two round, pink-grey, gelatinous-looking tumours about the size of a very small pea at periphery of iris in iridic angle; the surface is smooth, and the one below, at six o'clock, has blood-vessels on it. V. $\frac{6}{12}$. Fundus normal. Left eye: Scarcely any conjunctival congestion; cornea, below many "mutton-fat" spots of K.P.; pupil irregular. As in the right eye, there are three similar small tumours at periphery of iris below. V. $\frac{6}{24}$. Fundus normal.

The arm was scratched with 4 per cent. tuberculo-toxin in water in four places, but no positive reaction.

Did the tumour spring from the ciliary body or from the ciliary edge of the iris? Was it tuberculous or syphilitic? The von Pirquet reaction was tried in six smears, but gave negative results.

(Card specimen. March 11th, 1909.)

Mr. E. Nettleship said that a girl was under his care at St. Thomas's Hospital fifteen or more years ago, having previously been at Moorfields. She was kept in St. Thomas's for a long time, where she had relapses of what was called "erythema nodosum," and a progressive subacute irido-cyclitis. He did not remember the actual appearance of her anterior chambers, but he was sure that she had masses in the angle as in Mr. Jessop's case. Eventually she got secondary cataract, and an unfavourable prognosis was given. There was no history of any constitutional cause but the tuberculin tests were not then in current use. Some years afterwards, when grown up, she came under Mr. Spicer's care at St. Bartholomew's Hospital, had the lens removed by him from one eye and obtained an extremely good visual result. This was a lesson
against giving up a case even when apparently hopeless. He remembered another somewhat but not exactly similar case published in vol. i of the Society's Transactions in a thin under-grown girl, æt. 14 years. She had no signs of inherited syphilis, and the history was negative. In one eye she had a large vascular mass, which half filled the anterior chamber, and had the appearance of sarcoma of the iris, for which he took it. He excised the eye and examined it, and it was decided that it had the structure of a gumma. Subsequently the other eye became bad in the same way, and in a couple of years recovered perfectly. Anti-syphilitic treatment did not seem to have any influence, and when seen in her twentieth year she remained well. There was no proof whatever of either tubercle or syphilis, and he suggested that in some of these cases there might be some third condition not yet recognised.

Mr. Doyné said that a strong point was, that so far as he could recall, tubercle nearly always affected one eye only, whereas in syphilitic affections it was common to get both eyes involved. The appearance of the spot in the present case was much like that of gumma, and if so, it would probably clear up under the administration of mercury.

Mr. Jessop, in reply, had never seen tumours like these, and thought the case might have been tubercle, but the vascularity of one of the masses in the right eye and the negative result of the tuberculin reaction contra-indicated tubercle.
44. *Congenital anterior staphyloma.*

By E. Treacher Collins.

(With Plates VIII, IX, and X.)

The child who is the subject of this communication was first brought to me when a month old on August 14th, 1907. The doctor who attended the mother in her confinement stated that directly after birth he noticed the cornea of the right eye was opaque and unduly prominent, also that there was a slight haze of the centre of the cornea in the left eye.

The child was born at full term. No instruments were used. The mother had not had any unnatural vaginal discharge during pregnancy, and except for a slight trace of mucus there had been no discharge from the child's eyes.

The mother was quite unable to account for the defect; she had one other healthy child, and there was no history of any congenital eye affections or other malformations in her's or her husband's families. During the third month of her pregnancy she had had a poisoned finger from a wound with a holly-bush; an abscess formed and was opened; at that time she had felt feverish and indisposed, otherwise her health had been quite good.

The appearance of the eyes when I first saw the child was said not to have altered since birth. I found the cornea of the right eye enlarged, opaque, vascular and staphylomatous. It projected forward through the palpebral aperture, but the eyelids could be closed over it. The density of the opacity varied in different parts, being greatest in the centre. As far as could be made out the iris lay in contact with the back of the cornea throughout its entire extent, there being no anterior chamber. In the left eye there was a small faint central nebula, but in other respects it appeared to be normal.

I saw the child at intervals during the next fourteen months. The right cornea became gradually a little more prominent; at one time a dry horny scab formed on...
its most exposed part, evidently composed of cornified epithelium. This separated, and afterwards the surface was kept moist with ointments and lotions. Up to the time of the operation the eyelids continued to close over the surface of the globe in sleep, though when the child was awake it formed a most unsightly protuberance through the palpebral fissure. The nebula of the left cornea became gradually fainter and less conspicuous.

On October 14th, 1908, when the child was fifteen months old, I performed an evisceration operation on the right eye. An elliptical piece of the front of the eye including the whole cornea was first removed; to it the iris and lens adhered. The rest of the contents of the globe, including the vitreous, choroid and retina, all of which appeared healthy, were then turned out. A glass globe was inserted into the empty sclerotic and stitched in. The child made an uninterrupted recovery from the operation.

Pathological Examination of the Elliptical Piece removed from the Front Part of the Eye.

Macroscopical appearances.—The margins of the cornea were very ill-defined; laterally it measured about 17 mm. It was opaque and had blood-vessels coursing through it. The anterior ciliary processes were included in the specimen, also the lens. The measurements of this latter after it had been hardened in formalin solution were—laterally 8 mm., anterio-posteriorly 2 mm. It was clear except at the anterior pole. When it had been separated from the back of the cornea there was seen to be a conical projection at the anterior pole, which stood out 0·8 mm. from the rest of its surface.

The posterior surface of the cornea was covered with uveal pigment except over an area corresponding to the pupil, where there appeared to be some projection backwards of the corneal substance.

On transverse section of the cornea it was seen to be much thicker than normal, and much thicker in the centre
PLATE VIII.

Illustrates Mr. E. Treacher Collins's case of Congenital Anterior Staphyloma (p. 169).

(For the micro-photographs from which the figures on this plate are reproduced the writer is indebted to Mr. E. Collier Green.)

Fig. 1 shows a Section through the centre of the Staphylomatous Cornea.

Fig. 2 shows the Epithelium on the Anterior Surface of the Staphylomatous Cornea.

Fig. 3 shows the Posterior Surface of the Cornea, with the Pigment Epithelium of the Iris in contact with it. Note complete absence of Descemet's Membrane, Ligamentum Pectinatum, and Stroma of Iris.
than at the two sides (Pl. VIII, fig. 1). It measured anteroposteriorly in the centre 4·5 mm. and at the sides 2 mm.

Microscopical appearances.—The epithelium on the surface of the cornea is thicker than normal, and, except at the periphery, presents everywhere well-defined superficial horny layers devoid of nuclei. The base line of the epithelium, though showing some undulations, is everywhere very regular, there being no process of epithelial cells extending back into the fibrous tissue beneath (Pl. VIII, fig. 2).

There is no anterior limiting membrane, the epithelium resting directly on the laminated fibrous tissue composing the substance of the cornea. This latter is much thicker than normal. A little piece of the sclerotic which is shown at the margin of the cornea on each side of the sections has the normal width. Proceeding from it the cornea gradually expands in width, but more rapidly on one side than the other so that the thickest part is not quite in the centre (Pl. VIII, fig. 1).

The layers of fibrous tissue composing the cornea are not as regularly arranged as they are in the normal cornea, and have more cells distributed between them. Everywhere throughout it blood-vessels are to be seen cut in various directions. They seem to be derived partly from the ciliary vessels in the sclera and partly from those in the conjunctiva.

No trace of Descemet's membrane is to be detected, neither its elastic layer nor lining endothelium. There is no ligamentum pectinatum (Pl. VIII, fig. 3, and Pl. IX, fig. 4).

The posterior surface of the cornea is lined by the pigment epithelium of the iris, which, from the way it has become separated in places in some of the sections, seems not to have been intimately adherent to the fibrous tissue in front of it, only pressed into close contact with it.

The space corresponding to the pupillary area is not situated centrally, the pigment epithelial layers on the back of the cornea being of greater length on one side of the sections than on the other.

Over a considerable extent of the posterior surface of the
cornea nothing separates the two pigment epithelial layers from the fibrous tissue of the former. Here and there, however, in front of the anterior of the two epithelial layers and adherent to it, are collections of spindle-shaped cells; they are seen cut in some cases longitudinally and in others transversely; they resemble the cells of the unstriated muscle-tissue of the iris (Pl. X, fig. 6).

Bleached sections show the two layers of pigment epithelial cells continuous at the margin of the gap corresponding to the pupillary area, much as they are continuous at the margin of a normal pupil. The two layers of pigment cells do not lie everywhere in contact. On the side of the section where they are shortest the posterior layer is separated for a considerable space from the anterior, and is convoluted as though there was an excessive amount of it (Pl. VIII, fig. 3). On the opposite side of the sections no space is left between the two layers of pigment epithelium, but little processes are formed by projection backwards of folds in the posterior layer.

In the space corresponding to the pupillary area there is some projection backwards of the fibrous tissue composing the cornea (Pl. IX, fig. 5).

The anterior of the ciliary processes are situated abnormally far forwards, and seem to protrude from the root of the iris.

The extent of the specimen does not allow of much being made out as to the condition of the ciliary muscle. A small piece of its anterior extremity is all that is to be seen. It seems to arise from the fibrous tissue of the sclerotic, there being no ligamentum pectinatum for it to be attached to (Pl. IX, fig. 5).

A circular channel cut transversely and containing blood is seen in all the sections in the posterior part of the fibrous tissue at the sclero-corneal margin. It may correspond to Schlemm's canal or the circular arteriosus iridis major (Pl. IX, fig. 5).

Sections of the lens, which were cut separately from the rest of the specimen, show at its anterior pole a conical
PLATE IX.

Illustrates Mr. E. Treacher Collins's case of Congenital Anterior Staphyloma (p. 169).

(For the micro-photographs from which the figures on this plate are reproduced the writer is indebted to Mr. E. Collier Green.)

Fig. 4 shows the Corneo-sclerotic Margin. The most anterior of the Ciliary Processes is seen coming off from the Posterior Surface of the Iris. The Ciliary Muscle is arising directly from the Sclerotic, there being no Ligamentum Pectinatum.

Fig. 5 shows the Termination of the Pigment Epithelium at the Pupillary Margin on each side. The Fibrous Tissue comprising the Cornea protrudes backwards through the opening corresponding to the Pupil.
projection produced by a bending forwards of the cortical layers in that locality (Pl. X, fig. 7). The most prominent part of this projection is covered by the hyaline capsule and a single row of epithelial cells as in the normal lens. On each side of the conical projection in the angles which it forms at its junction with the surface of the lens there are masses of tissue like those met with in capsular cataract. These masses are composed of layers of fibres with epithelial cells scattered between them; on one side there is also some calcareous material deeply staining with haematoxylin. The capsular cataracts are covered on the anterior surface with the hyaline capsule without any cells lining it. On the posterior surface—that towards the lens—there is a single layer of epithelial cells continuous with those lining the capsule elsewhere. So that at the margin of a laminated mass the hyaline capsule and its lining epithelium appear to part company, the former going in front and the latter behind.

The lens fibres protruding forwards into the conical projection have some deeply staining granular degenerative material amongst them.

Cells line a considerable part of the posterior capsule, there being only a small area at the posterior pole devoid of them. Immediately in contact with the cellular lining of the capsule, both posteriorly and laterally, there are numerous large globular nucleated cells or fibres.

Congenital anterior staphyloma is a rare affection, and only a few cases in which the condition has been examined pathologically have been described.

Most of those who have written on the subject have been of opinion that the changes found were the outcome of intra-uterine ulceration and perforation of the cornea. To this conclusion Parsons* and Coats,† who have recently recorded cases in this Society's Transactions, likewise arrived.

The microscopical appearances found in some of the

* Vol. xxiv, 1904, p. 17.
† Vol. xxvi, 1906, p. 36.
cases closely resembled those which are met with where an anterior staphyloma has resulted from a perforating ulcer of the cornea occurring in infancy. There was a central defect in Descemet's membrane, the peripheral parts remaining intact. The bulging part of the cornea was composed of irregularly arranged laminae of vascularised fibrous tissue, resembling scar-tissue more than normal cornea, and its posterior surface was lined by the uveal pigment of the iris.

Parsons, who, besides describing a case, summarised the literature on the subject, discusses the way in which ulceration of the cornea in utero may be brought about. He points out that endogenous infection by bacteria through the blood-stream of an avascular structure, such as the cornea, is out of the question. Though he mentions endogenous infection by toxins entering the lymph-stream as possible, he considers exogenous infection of the cornea from the surrounding amniotic fluid as more probable.

Infants are sometimes born with ophthalmia, and in others the symptoms of ophthalmia follow so shortly after birth that it seems necessary to assume that infection took place in utero. Stephenson and Rosa Ford* have collected and analysed thirty-five recorded cases and added seventeen new cases of this so-called ante-partum ophthalmia. About half (44.5 per cent.) they consider satisfactorily accounted for by a premature rupture of the membranes, allowing access of micro-organisms to the baby's conjunctival sac. In the remainder (55.5 per cent.) they think "a slight injury to the membranes may determine access of micro-organisms, or infection through the uninjured membranes must be assumed to have taken place." In support of the possibility of infection occurring through the membranes, they refer to a case quoted by Armaignac, in which the Bacillus coli communis and other anaerobic microbes were found in liquor amnii drawn off before rupture of the membranes.

PLATE X.

Illustrates Mr. E. Treacher Collins's case of Congenital Anterior Staphyloma (p. 169).

(For the micro-photographs from which the figures on this plate are reproduced the writer is indebted to Mr. E. Collier Green.)

Fig. 6 shows the Posterior Surface of the Cornea and the Pigment Epithelium of the Iris; between them are some Unstriated Muscle-fibres. Note complete absence of Descemet's Membrane and Stroma of Iris.

Fig. 7 shows the Front Part of an Antero-posterior Section through the Lens. Note the Conical Projection forwards at the Anterior Pole, with a Mass of Capsular Cataract on each side of it.
In cases of perforating ulcer of the cornea which occur in infancy, where the resulting appearances are comparable to those met with in the recorded cases of congenital anterior staphyloma, the course of events is as follows: A large portion of the whole thickness of the cornea is destroyed by ulceration, its extreme periphery alone remaining. At the seat of the perforation inflamed iris is left exposed, from the surface of which granulation tissue grows out and fills the gap. By the conversion of this granulation tissue into fibrous tissue a new pseudo-cornea is developed; it is, of course, lined posteriorly by pigment epithelium, whilst over its anterior surface epithelium spreads from the undestroyed cornea at the sides. The usual channels of exit for the aqueous humour in such an eye being closed, it is unable to escape and the tension becomes increased. Before this increase of tension the newly-formed soft pseudo-cornea expands and becomes staphylomatous.

The above series of changes resulting in the formation of the staphyloma necessarily take some time to eventuate, usually some weeks. It seems unlikely that there would be time for them to occur in utero, and for the child to be born with a fully developed staphyloma from an ophthalmia due to the entrance of micro-organisms through a laceration in the membranes.

Apparently, then, we must regard congenital anterior staphyloma either as the result of ulceration of the cornea from an ophthalmia produced by amniotic fluid, infected through the intact membranes some weeks before birth, or abandon the view that it is inflammatory in origin, and find an explanation for it in some perversion of development.

Evidence as to whether a congenital defect is due to intra-uterine inflammation or to mal-development is sometimes to be found in the presence and character of other congenital defects or in the family history.

In the epitome of the recorded cases of congenital anterior staphyloma given by Parsons I do not find
mention made of any other defects apart from those of the eyes. In some there was a defect noted in the fellow eye to the one with the staphyloma. Thus its condition was described in Krückow's case as "microphthalmia"; in Hirschberg's and Birnbacher's as "phthisis bulbi"; in one of Steinheim's as "small with cystic ectasis"; in Westhoff's as "shrunken with opaque cornea"; in Lawson's and Coats's as "microphthalmic" with opacities of the cornea. In Crampton's two cases there was opacity of the cornea in the fellow eye. In the case recorded in this paper there was a nebula of the cornea of the fellow eye. In two of Steinheim's cases the anterior staphyloma was bilateral.

Parsons suggests that the comparative frequency with which the two eyes are involved favours the view that there has been exogenous infective inflammation of the eyes through the amniotic fluid.

The bilateral occurrence of developmental defects in the eyes which are certainly not inflammatory in origin is, however, of very frequent occurrence, and there is considerable evidence to show that microphthalmia is sometimes met with apart from all signs of inflammation. In none of the recorded cases has a pathological examination of the affected fellow eye been made.

In none of the recorded cases of congenital anterior staphyloma has the condition been met with in members of more than one generation. Crampton describes the affection as met with in two brothers. Steinheim gives the remarkable history of a family of five children with the following conditions: (1) Both eyes staphylomatous; (2) eyes normal; (3) right eye total staphyloma, left eye small with cystic ectasis; (4) both eyes total staphyloma; (5) both eyes white and shrunken. It is difficult to believe that in four out of five children in one family exogenous inflammation of the eyes through the amniotic fluid should occur, and in all of them result in perforating ulcers of both corneas.

In two recorded cases the anterior staphyloma was
composed of a mass of dermoid tissue. The first of these was in a case of v. Graefe's, described by Swanzy.* A large congenital dermoid growth projected from the front of the eye, its base occupying nearly the entire cornea. It was composed of two segments, an anterior the size of a large cherry and a posterior less than half that size; they were connected by a narrow band. The colour of the growth was that of the ordinary integument, and on magnification a few fine hairs were discerned. Microscopically a cutis-like layer with hair-follicles and sudoriferous glands was found covering the growth with ordinary subcutaneous fatty tissue beneath it. A thin layer of fibrous tissue occupied the normal position of the cornea with a rudimentary iris lying in contact with it, so that there could not have been any anterior chamber. No statement is made as to the condition of Descemet's membrane. At the operation for the removal of the growth there was considerable escape of vitreous and no lens was found; it is inferred that it was congenitally absent, but as the globe itself was not excised and examined pathologically, only the staphylomatous growth, this is by no means certain.

In the other case recorded by Bernheimer† the clinical appearances were very similar. The anterior part of the staphylomatous cornea was composed of skin-like tissue. The epithelium was horny on the surface and had many down growths. There were hair-follicles, hairs and sebaceous glands. The fibrous tissue beneath contained capillaries and groups of round cells; towards the periphery, where its tissue was of a somewhat looser texture, there were masses of adipose tissue. Descemet's membrane was absent over an area in the centre. Adherent over the back of the cornea was atrophic iris with its pigment epithelium. The lens was in situ.

It would seem impossible to explain the presence of the dermoid tissue covering the front of the staphylomatous

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* Dublin Quarterly Journal of Medical Science, May, 1871.
† Arch. f. Augenheilk., Bd. xviii, 1887, S. 171.
cornea in these two cases as the outcome of ulceration. Parsons speaks of it as a teratoid development. I think it may be accounted for by supposing an anterior staphyloma of the cornea to have occurred (as a defect of development in the way to be described later), the projection of which through the palpebral fissure prevented the union of the lids over the front of it, and the formation of the conjunctiva for a time into a closed sac. The surface of the projecting cornea would then be left exposed to the same influences as other superficial parts of the body, and like them would develop a skin-like surface.

A similar process is seen to take place when a portion of an eyelid fails to form and the surface of the eye opposite the defect remains exposed. We then have a dermoid growth on the surface of the eye associated with a coloboma of the eyelid. When no eyelids develop as in cryptophthalmos the whole surface of the eye becomes covered with skin.

The pedunculated character of a portion of the growth in Swanzy’s case was probably due to its being nipped between the margins of the eyelids. A congenital dermoid of the conjunctiva which I examined microscopically became pedunculated in this way, and has been recorded by Snell.*

In cases of ulceration of the cornea occurring in infancy the destructive process never extends outward sufficiently far to destroy the whole of Descemet’s membrane. It never reaches the ligamentum pectinatum. In the case recorded in this paper, therefore, where the whole of Descemet’s membrane and the ligamentum pectinatum were absent, it seems impossible to explain the condition as the outcome of a perforating ulcer of the cornea, and we are forced to consider how it might have arisen as the result of some defect in development.

The substantia propria of the cornea, the hyaline layer of Descemet’s membrane, its lining endothelium, and the

anterior fibro-vascular sheath of the lens, are all products of the mesoblastic tissue which grows in from the sides in front of the lens vesicle to separate it from the surface epiblast.

At one time this mesoblastic tissue is entirely composed of cells of a uniform type. Then it becomes differentiated into two parts, an anterior which thickens quickly and where the cells show a tendency to become elongated, and a posterior in which the cells are round and arranged in two rows. Very soon these two parts become separated by a thin hyaline membrane, which gradually thickens and forms the hyaline layer of Descemet's membrane. It is probably produced as a kind of secretion from the row of cells on its posterior surface which develop into its lining endothelium. The part of the mesoblast in front of the hyaline layer remains devoid of blood vessels and is gradually transformed into the laminated fibrous tissue of the substantia propria. In the posterior of the two layers of rounded cells behind the hyaline membrane blood-vessels extend from the anterior ciliary arteries, it becoming the anterior fibro-vascular sheath of the lens.

The abnormality in the specimen which is here described can, I suggest, be adequately explained as due to a failure of the mesoblast which intrudes between the lens vesicle and the surface epiblast to become differentiated into its several layers, i.e., an atypical development of this mesoblast. No differentiation of it into two parts took place and no hyaline layer formed. There was, therefore, no Descemet's membrane and no fibro-vascular sheath, only a thick mass of fibrous tissue. Blood-vessels, instead of being restricted to its hindernost part, the fibro-vascular sheath, permeated in various directions, and so tended to alter its appearances considerably from that of the normal substantia propria.

The anterior fibro-vascular sheath plays a very important part in the development of both the iris and the lens. The anterior layers of the iris and stroma are mainly produced from it. Beneath it the two layers of
the secondary optic vesicle, which become the posterior pigment epithelial layers of the iris, extend inwards, and from them, according to the researches of Forsmark* and others, the muscular tissue of the iris is developed.

The lens, during the most active period of its growth in foetal life, before the formation of the anterior chamber and the secretion of the aqueous humour, receives a large part of its nutrient supply from the anterior fibro-vascular sheath.

If, then, as I have suggested in the case here recorded, the anterior fibro-vascular sheath failed to become differentiated off from the mesoblastic tissue which intrudes between the lens and the surface epithelium, we should expect to find defective development of the stroma of the iris and of the lens. What we do find is that the pigment epithelial layers of the iris are lying in contact with the vascularised fibrous tissue which represents the cornea, but that nowhere can any tissue corresponding to iris stroma be recognised in front of them. Here and there patches of spindle-shaped cells similar to those of the unstriated muscular tissue of the iris are to be seen in front of the pigment epithelium, but no stroma. The pupillary area is clearly distinguishable.

The lens is small, much flatter than usual from before backwards, and besides other abnormal appearances in the arrangement of its cells and fibres, presents a most peculiar condition at the anterior pole. There is a conical projection forwards of the lens fibres, which apparently is encircled by a ring of capsular cataract having the usual characteristics of capsular cataracts at the anterior pole of the lens.

Coats, in the pathological examination of an eye with congenital anterior staphyloma, recorded by him and Arnold Lawson, describes a forward dislocation of the inner layers of the anterior part of the uvea. The ciliary processes arose further forward than the ending of the

membrane of Descemet; the pars plana was on a level with the ligamentum pectinatum, and the ora serrata was in front of the ciliary muscle.

Such a condition, he points out, is one of extreme rarity; he could only find one other recorded instance of it; that was in a case of congenital anterior staphyloma described by Krückow in 1875.

It is very probable that in the specimen the subject of this paper a somewhat similar condition was present; its extent, however, did not permit of a complete examination of the relations of the ciliary body. All that can be said is that the ciliary processes were abnormally far forwards, the most anterior projecting out from the back of the root of the iris.

From the pathological point of view I regret I did not remove the whole eye instead of eviscerating and inserting a Mules' globe. From the clinical point of view, however, I have achieved a more satisfactory result by the evisceration than would have been produced by enucleation.

Coats naturally infers that some connection must exist between congenital anterior staphyloma and dislocation of the uvea, and is led to ask how it is that the latter is not also met with in connection with post-natal anterior staphyloma, which is of much commoner occurrence.

Coats, who believes the pre-natal, like post-natal anterior staphyloma, is the outcome of perforating ulcer of the cornea, suggests that the difference is due to the looser connection between the uvea and the structures external to it (including the ciliary muscle) during foetal life, so that when stretched it is capable of being displaced bodily forwards. On the other hand, after birth, the connection having become more intimate, the stretching is only capable of causing elongation and atrophy.

I would suggest that this difference in the arrangement of the anterior part of the uvea in cases of pre-natal and post-natal anterior staphyloma may be taken as evidence in favour of a different origin of the staphyloma in the two classes of cases. In the pre-natal anterior staphyloma
there would be a natural tendency for the anterior part of the secondary optic vesicle to extend forwards over the inner surface of the expanded anterior part of the globe, so leading to a forward displacement of the ciliary processes, *pars plana* and *ora serrata*. In the post-natal anterior staphyloma, the uveal, having first developed in the normal way, would subsequently have acquired adhesion to the cornea, the bulging of which would cause parts of it to become stretched and atrophied.

In conclusion I wish to summarise briefly the different malformations which may arise from aberrations in development of the mesoblast, which intrudes between the surface epithelium and the lens vesicle, examples of which I have been able to record from time to time in the *Transactions* of this Society or elsewhere.

(a) Complete failure in the formation of Descemet's membrane, the ligamentum pectinatum, and the anterior fibro-vascular sheath, resulting, as in the case here described, in a vascularised thickened cornea, absence of stroma of iris, apposition of its pigment epithelium to the posterior surface of the cornea, absence of anterior chamber, increase of tension, and anterior staphyloma.

(b) Complete failure in the formation of Descemet's membrane, with adhesion of the anterior capsule of the lens to the fibrous tissue of the cornea, resulting in obstruction to the growth inwards of the iris between them, and microphthalmia.

This condition was met with in a chick's eye described by Parsons and myself in vol. xxii of our *Transactions*; there was a congenital absence of the eye on the other side (anophthalmia).

(c) Failure in development of a large central area of Descemet's membrane, resulting in an extensive anterior synechia of the iris, absence of anterior chamber, increase of tension, anterior staphyloma, and often extreme thinning of part of the cornea.

In some of these cases, in which the staphylomatous condition occurred early and prevented the union of the
eyelids over the front of the eye for a portion of foetal life, the protuberant part has developed a skin-like covering, as in the cases recorded by Swanzy and Bernheimer.

(d) Failure in development of a small piece of Descemet's membrane, with an adhesion of the iris or persistent pupillary membrane to the *substantia propria* of the cornea. The cornea in all other respects normal, and the extent of the adhesion not sufficient to give rise to increase of tension.

An example of this condition is recorded by von Hippel,* and I have described it as present in the eye of a cat.†

(e) A complete development of the hyaline layer of Descemet's membrane, but a failure over a small area of the cells lining it to become differentiated and separated from the anterior fibro-vascular sheath, resulting in an anterior synechia of the iris or pupillary membrane, the cornea in all other respects being normal. I have described this condition as being present in an eye with a persistent and patent central hyaloid artery.‡

A case presenting similar appearances has also been recorded by Ballantyne.§

(f) A normal development of Descemet's membrane but a complete failure in the formation of the ligamentum pectinatum. There being no channels for the exit of aqueous humour, increase of tension and buphthalmos result.

I have described this condition as present in a buphthalmic eye removed from a boy, *et. 14 years.*|| Descemet's membrane, instead of splitting up into a number of fibres at the angle of the anterior chamber, continued round it, extending for a short distance along the anterior surface

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* Arch. f. Ophth., Bd. ix, Heft 3, S. 444.
of the iris and terminating rather abruptly. Externally to the angle of the chamber in this eye there was a broad adhesion of the root of the iris to the sclerotic, to which also the ciliary muscle was attached, but neither fibres of the ligamentum pectinatum nor a canal of Schlemm could be detected.

I have also met with complete congenital absence of the ligamentum pectinatum in two buphthalmic eyes which occurred in connection with plexiform neuroma of the eyelids. One is described in a paper on that subject by Snell in vol. xxiii of our Transactions, and the other in a paper by Rayner Batten and myself in vol. xxv.

(g) A normal development of the Descemet's membrane and the ligamentum pectinatum, but an imperfect separation from the back of the latter of the peripheral part of the anterior fibro-vascular sheath, which becomes the anterior layers of the iris. The angle of the anterior chamber is then imperfectly prolonged outwards and a condition exists which is normally present in some of the lower animals, i.e., a ligamentum pectinatum with an outer laminated zone and an inner cavernous zone. The latter should disappear before birth, leaving only the former. I have met with a persistent cavernous zone in several microphthalmic eyes without any increase of tension.* I have also met with it in congenitally buphthalmic eyes in which presumably its presence offered an impediment to the exit of fluid from the eye, and so was the cause of the increase of tension and enlargement of the globe.

(March 11th, 1909.)

The Chairman said the Society was much obliged to Mr. Collins for this excellent piece of work, and for the way in which he had joined together his different remarks connected with the arrest of development of Descemet's membrane and the ligamentum pectinatum. It would be interesting to hear what Mr. Parsons and Mr. Coats had to say on the subject.

Mr. Parsons said there was not time to discuss the paper properly. Mr. Collins had kindly sent him the paper so that he might have the opportunity of criticising it, but there was not time to deal with so large a question. He still thought that the inflammatory theory was the simplest and most probable, and that did not put out of court arrest of development following and resulting from inflammatory change. When one saw an anterior staphyloma which had been caused after birth in the ordinary way one did not expect to find the ligamentum pectinatum, but one found Descemet's membrane, and he thought it was very difficult to explain the total absence of Descemet's membrane as the result of an early inflammatory condition. The case which was most difficult to explain, on the developmental theory, was the one which Messrs. Coats and Lawson published. He had recently had another case which he took to be a condition resembling anterior staphyloma, that of a child who was brought to him at Great Ormond Street at ten months of age. He excised the eye, and it was now being examined. He thought it supported Mr. Collins's views better than any case which had been published. There was very little staphyloma, although the eye was scarcely enlarged, and there was much cupping of the disc. But there was a widely dilated pupil and the iris was in contact with the cornea. He thought it difficult to explain that case on the same theory as ordinary congenital staphyloma. When he published this recent case he proposed to discuss the question more fully.

Mr. G. Coats said that the chief peculiarity of his case was a forward displacement of the inner layers of the uveal tract, so that the ciliary processes were in front of the angle of the anterior chamber and the ora serrata in front of the ciliary muscle. Mr. Collins accounted for this displacement by an extension forward of the secondary optic vesicle. But there was much more than this, for the underlying mesoblast was also displaced forwards; the ciliary muscle, for instance, was lined...
by choroid, in which a distinct membrane of Bruch and chorio-capillaris were visible. It seemed impossible to account for that condition by any mere lack of differentiation. Again, in this case differentiation was by no means very imperfect. In the periphery the corneal stroma, the membrane of Descemet, the endothelium, the ligamentum pectinatum and the spaces of Fontana were all well formed, and there was an anterior chamber. The iris and ciliary processes were also quite well formed, but displaced forward relative to these other structures. The strongest point in favour of corneal perforation and against imperfect differentiation was, however, the finding of lens remains among the fibres of the pseudo-cornea. It was true that there had been a post-natal rupture of the globe, but it was only two days old when the eye was excised, and inasmuch as the lens substance was embedded in the midst of cicatricial tissue, and was found quite apart from the perforation, it was quite evident that the evacuation of the lens had not taken place at the time of the post-natal rupture. On Mr. Collins's hypothesis this condition could only be explained by supposing that the lens vesicle had never formed properly. But in that case the eye could not possibly have formed so well as it had. Moreover there was definite evidence that the lens vesicle had developed, since considerable pieces of well-formed capsule were found. There was therefore complete proof that the lens at least, and presumably the iris as well, were in their proper positions during a large part of foetal life, and that the lens subsequently became displaced forwards and evacuated in consequence of a corneal perforation, just as occurs in post-natal ulceration. The displacement of the inner layers of the uveal tract he regarded as due to dragging from the expansion of the globe, the connection between the inner and outer layers being looser in foetal than in post-natal life. At the same time he did not mean to deny the great importance of defective differentiation, especially in connection with the abnormalities about the angle of the anterior chamber.
which seemed to be at the bottom of the pathology of buphthalmos. Mr. Collins had mentioned dermoids of the cornea, but it should be remembered that the histological characters of these tumours were quite different from those of congenital anterior staphyloma. It was difficult to accept the theory that mere exposure, even in foetal life, was sufficient to transform mucous membrane, and *à fortiori* corneal stroma into skin. One might perhaps expect cornification of the epithelium, but not the development of hairs, and still less the development of adipose tissue in the deeper structures. Moreover no such process occurred in the ordinary form of congenital anterior staphyloma, or in cases of congenital exophthalmos from oxycephaly, etc., nor could ordinary dermoids of the cornea or conjunctiva with normal lids be so explained, and the dermoids with gaps in the eyelids were probably better accounted for by an adhesion of the foetal eyelid, which subsequently separated, leaving a piece behind. This accounted both for the dermoid and for the gap.

Mr. Collins, in reply, said, with regard to the condition of the lens in Mr. Coats's case, there was in it a perforating ulcer which led to excision of the eye when the child was sixteen weeks old, so according to Mr. Coats's hypothesis there was a perforating ulcer in infancy resulting in anterior staphyloma, and then later a second perforating ulcer. It was very rare for a child who had staphyloma from perforating ulcer after birth to later get a second perforating ulcer. He suggested that at the perforation when sixteen weeks old the lens escaped, and that in that way the appearance of the lens capsule described by Mr. Coats might be accounted for.
45. Seven new pedigrees of hereditary cataract.

By E. Nettleship.

Case 1 (Fig. 38).—Lamellar cataract in four generations; the opacity of small diameter in all cases, and vision improved by iridectomy in several. Excess of the cataractous over normal members—twenty-three with cataract to about twelve normals in the affected divisions. No history of fits, and few, if any, defects of dental enamel. No early deaths recorded. The cataract found in one case at the age of 3 (IV, 18), and proved by the mother (III, 3) to have been visible within an hour or two of birth in IV, 9 and some of her other children. She (III, 3) told me that she had been able to see the cataract by opening the baby's eyes almost as soon as the nurse had washed it immediately after birth. She is an intelligent and observant woman, disconcerted by having borne so many children with imperfect sight (six affected out of nine, all living), and very solicitous to have normal ones; she had therefore looked at the eyes of her younger children critically immediately after birth. IV, 11, who died at 11 months, was also undoubtedly affected, and IV, 14 showed the anterior polar opacity at the age of 3 months (see description of cases below).* No consanguinity of any parents. Congenital ptosis in two of those with cataract.

This lamellar cataract stock, family name Everett, came from Wantage, in Berkshire, where I, 4, the earliest member about whose sight anything is known and his parents resided. I, 4 died at 70 about twenty years ago. Some of his collaterals are said to have stayed at Wantage, and one brother (I, 3) went to Newbury in the same county. I have made some inquiries at both places; they seem not to be known now at Newbury, and only a single family of the name is at Wantage and it contains no one with defective sight. A large contingent is at Swindon (I, 1 and her numerous descendants in II and

* For additional case, IV, 14a, seen at one week old, see note to p. 194.
III: all these are reported normal, and as they live side by side with two affected members (III, 8 and 9), who also reside there, the report may be taken as correct. I have found that the affected members of this genealogy are always right in their diagnosis, that they make no attempt to conceal the defect in those who have it, and that those whom they report as free have always turned out on examination to be so.

I obtained access to this genealogy through Mr. Fisher, who has one of the children (IV, 14) under care. I have had much assistance from Mr. Cyril Walker (Bristol) with the children of III, 3, and from Major F. M. Mangin, Ophthalmologist to the R.A.M.C., Aldershot Command, who kindly examined III, 7 and his child for me. Two or three of the affected ones have been under the care of Mr. Lawford, Mr. Doyne, and others. I have seen the majority of both affected and normal members myself and
found that most of those with the family cataract have got on well enough to satisfy themselves without advice or treatment. The two patients (four eyes) in whom optical iridectomy has been performed (III, 2, both eyes; III, 4, both eyes) have benefited decidedly, and probably most of the others might have had this operation done with advantage. In only three (III, 9, and IV, 15, and II, 6) has the lens been needled, and circumstances have prevented either of these from being finished. Thus only five of the twenty-four had undergone operation when I saw them. Many of the family now live on the borders of Gloucestershire and Oxfordshire, others at Swindon and Bristol.

The cataract is uniform with slight variations in all those affected; the shell of opacity dense and seldom transmitting any red light with an undilated pupil,* sharply defined and circular, except in one or two, where it was bluntly triangular, sometimes with a few projecting spokes, but never with any separate riders or other evidence of a second layer of opacity, and measuring 3·75 to 4·5 or 5 mm. in diameter. The cortex clear, but noted as slightly dim in one case, and in that one the circumlental space unusually wide as if the lens were small (III, 4). It was not possible to dilate the pupils in any of the cases, and hence details of the fundus and refraction could not be ascertained, and V was not accurately tested in any of them; needlework is difficult, but they can all see to read pretty well by management; they cannot face the light, and they see best towards evening and on a cloudy day, and one said "it is curious we see best in the dark." None of those seen were fair, and most of them had decidedly dark hair and brown irides.

One of the girls with cataract, aged 18 years (IV, 10), has a well-marked goitre, which has been growing for several years; her mother (III, 18), an only child, has a larger one, and the grandmother (II, 3) is reported to

* But all the examinations were made at the homes of the affected persons, in very imperfectly darkened rooms and sometimes with a poor light.
have a very large one. It will be seen that II, 3 and III, 18 are not related by blood to the cataractous stock. II, 3 lives at Bampton and presumably III, 18 was there as a child, but IV, 10 has always lived several miles from that place.

Description of the Cases.

I, 4 was a sack-weaver at Wantage; sight was always defective so that he could never earn full wages, but he did not go blind; died at 70 about twenty years ago. His eldest son (II, 2) remembers him perfectly, and is sure that his sight was just like his own. I, 4 was one of about eleven; one (I, 3) is remembered; neither II, 2 nor II, 4 can recollect hearing that any of I, 1, 2, or 3 had bad sight.

I, 4 married twice; by first wife he had only one child (II, 2). After many years he married again and had three more children (II, 1, 5, 6), II, 4 being twenty years younger than II, 2. Nothing is known as to sight of parents of I, 1 to 4. No consanguinity between the two wives or between either of them and I, 4. I, 5 was from Wantage; I, 6 from Suffolk, maiden name Stally.

II, 2. Henry Everett, at 70 years (seen August 30th, 1908), farm labourer; has been in the same place forty-nine years. A fine, rather tall man, with better features than most of his children. Sight defective all his life, and he says getting slowly worse (probably senile rigidity of pupils); learnt to read pretty well as a child. Small dense lamellar cataracts with a few spokes; the "shell," which entirely stops light from mirror, is scarcely larger than the 4 mm. pupil. He says sight of L. is worse than R. By focal light the cataract is uniformly grey. Has lost all his front teeth. His wife, II, 1, not consanguineous with him, died two years ago.

II, 4. George, eldest ½ sibling of II, 2, born in 1858, now at 50 years. Cataracts almost exactly similar in size and density to II, 2. Ps. more active than in II, 2. For many years has used + 5 D., and with them reads news-
paper type fairly well with his back to the light. Teeth good as to enamel of incisors. Is a gardener and keeps a small shop.

II, 5. John, had perfect sight; was born in 1861, three years after II, 4, and died at 42. Had five children (III, 13-17), the eldest æt. 19 years, all of whom I have seen since and found normal as well as their mother.

II, 6. Elizabeth, went abroad in service about “twenty” years ago. Not very long before she left England and when “in her teens” was operated upon for cataracts at Reading (testimony of II, 4). She has been lost sight of.

III, 2-11.—Ten children of II, 2; all living and aged from 48 to 27.

III, 2. Henry Everett, æt. 48 years, at the railway works at Swindon. Seen August 13th, 1908. Had optical iridectomy on each eye down and in by Mr. Lawford at Moorfields in 1892 or 1893, and sees much better in consequence; wears no glasses. Small lamellar cataracts with wide clear cortex; refraction Em. or H. in L., not tested in R. Teeth good. Has decided congenital ptosis. Married, no children.

III, 3. Polly, Mrs. Cleverley, has nine children (IV, 1-9), of whom six have the cataract. She is the mother referred to at p. 188 as having detected the opacity in some of her children immediately after birth.

III, 4. William, æt. 43 years, farm labourer and preacher, Lechlade. Had optical iridectomy downwards on each eye by Mr. Doyne seventeen years ago and is well satisfied. Circular sharply-defined lamellar cataracts of about 4 to 4.5 mm. diameter; no spokes, cortex shows as a slight general opalescence in comparison with the rather wide circumlental space; a sharply defined white Y-shaped opacity at anterior pole of each. Lower incisors good enamel, upper incisors gone. Married, no children.

III, 5. Charles, æt. 42 years, farm labourer, Kelmscott. Dark hair, brown irides, teeth quite good. Cataracts
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decidedly smaller than the previous cases, 3:5 or 3:75 mm., barely as large as lightly shaded pupil; no spokes. A broad-limbed Y of dense white on anterior surface of opaque zone, the intermediate parts allowing some red light to pass. Slight congenital ptosis of both upper lids.

III, 18, wife of III, 5, has a large symmetrical goitre of many years' standing; her aspect, manner and hair do not suggest myxoedema. She is an only child, and her mother (II, 3), a native of Bampton and still living there, is said to have a still larger goitre. They have three children (IV, 10, 11, 12).

IV, 10. Dolly, aet. 18 years, dark hair, teeth very good; has a large goitre like her mother's, and causing obvious obstruction in breathing; nothing suggestive of myxoedema. Cataracts rather larger than her father's, 4 to 4.5 mm., with quite white anterior polar spot and some spokes at equator. On first examining with mirror there was slight, quick nystagmus, but it passed off during the examination.

IV, 11. Died at 11 months of bronchitis during whooping-cough. Mother is quite sure, from the way in which he used to look at objects, that he had the family cataract; "he looked sideways like the others."

IV, 12. Francis Charles, aet. 6 years. Small cataracts of same size as father's, and with Y-shaped anterior polar dense opacity, as in him.


III, 7. Thomas, soldier at Aldershot. Examined, together with his only child, IV, 13, aet. 9 years, by Major F. M. Mangin, R.A.M.C.; lenses quite clear in both. The child has considerable H.

III, 8. George, aet. 35 years, employed at the Swindon Railway Works. Seen August 13th, 1908. Small, dense, clean-edged lamellar cataracts; rather wider than small pupil, i.e., about 3.5 to 4 mm. Sees well enough for his
work (labourer). Passed into the Army and served his seven years satisfactorily. Reads easily. Lower incisors show slight deficiency of enamel near cutting edge, but upper ones have good enamel. Married ten years; only one child (IV, 14)—Mr. Fisher's patient at Moorfields.*

IV, 14, aet. 10 years, has similar cataracts, 4.5 mm. in diameter, and subtriangular in outline rather than perfectly circular, and with dense anterior polar opacity. No operation has been done. Irides dark brown, hair black. His mother was ill for many weeks after his birth, and unable to notice the boy's eyes, but as soon as she was well enough, when he was about three months old, she noticed the white speck in each eye.

III, 9. John, aet. 32 years, farm labourer, unmarried, living with his father (II, 2). R.: Needled once three and a half years ago; P. blocked by white lenticular residue requiring another needling. L.: Small, dense, but slightly translucent lamellar opacity, with some faint spokes. Teeth good.

III, 10. Mrs. Annie Osman, aet. 30 years; married five years. Small, dense lamellar cataracts, about 3.5 to 4 mm. No spokes seen. Teeth normal. Is just expecting fourth baby. Her three children are—

IV, 15. Alice, aet. 6 years. R.: Cataract needled three times by Mr. Doyne, and is clear. L.: Appears to have been needled once, the whole lens being opaque, with a specially dense, projecting spot near anterior pole. Permanent lower incisors just up and normal.

IV, 16. George, aet. 3½ years. Lenses perfect, all well seen.

IV, 17. Albert, aet. 2 years. R.: Fairly seen with

* On April 19th, 1909, the wife of III, 8 was confined with her second child, a boy, and a speck was seen in its pupils, both by the nurse and the mother, immediately. On the eighth day (April 26th) I examined this baby's eyes under a mydriatic (pupils about 7 mm.) and found a typical lamellar cataract in each eye; diameter not quite 5 mm., dense white opacity at anterior pole, cortex clear. Healthy, well grown; hair quite dark. Should appear as IV, 14a on the pedigree between 14 and 15. There were no recognised conceptions between IV, 14 and 14a.
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+ 10 D., and no opacity. L.: Not seen so well, but good red reflex all over pupil.

III, 11. Daughter, act. 27 years, living with her father (II, 2). Small, dense lamellar cataracts, not more than 3·5 or 4 mm. diameter, and with a very dense, white, circular dot at each anterior pole. No spokes seen. Slight nystagmus in L. when light was thrown on eye, but it soon ceased. Teeth very good. Has one child (IV, 18), born out of wedlock, act. 3½ years when seen: small zonular cataracts (3·5 to 4 mm.), with a dense white spot exactly at anterior pole, precisely like her mother's. Father a man of same class as mother, but act. at least 60 years.

**Case 2 (Fig. 39).—Coralliform** cataract in four generations (Perrin family, near Harlow, Essex). For parts of this pedigree I am indebted to Mr. Marcus Gunn and Mr. Leslie Paton; and have also to thank Dr. F. N. Day (Harlow), Dr. W. E. Dring (Buckhurst Hill), and Dr. L. W. Dryland (Kettering) for much assistance, and Dr. Reeve of Toronto, for taking much trouble to follow and examine III, 7 in Canada.

The pedigree shows twenty-two cases of cataract, twenty of whom are living, and eighteen of these have been examined. Nearly all the non-affected members of the cataractous branches have also been examined, as shown by the X placed against each on the diagram. In every case of cataract the condition is alike in both eyes. The

* This term was first used by Marcus Gunn in describing the case of this form of cataract recorded and illustrated in these Transactions, vol. xv, p. 119 (1895). Long, straight, apparently tubular opacities springing from the central part of the lens pass radially forwards and become progressively larger towards their anterior extremities, which, being seen at full or three-quarter face, may be compared to the open mouths of a madreporic coral, or equally well to a tubular corolla or a short straight horn. The term "ampulliform" has also been used in describing the appearance of the expanded end, but is inapplicable, as the appearance is that of an open mouth, not a flask. Knies has compared these peculiar opacities to the sails of a windmill. Separate clumps and dots of opacity are also found.
Fig. 39.

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ages of those affected and examined vary from 72, 66 and 62 in Generation II, to 8, 9 and 10 in IV; III, 16, now aged 33 years, was also seen and described by the late Mr. Jas. E. Adams, at Moorfields, when she was 13. Several babies in IV have been examined with negative result, but of these only IV, 11, aged 1 year; 23, aged 1 year and 2 months; and 26, aged 9 months, had a cataractous parent. No other inferiority or degeneration has occurred in the stock, the members of which are of the labouring and artisan class. There is no doubt, from the appearances in cases at various ages, and from the statements of those affected, that the total amount of opacity very slowly increases, and that whilst V. of $\frac{6}{12}$, or even $\frac{6}{9}$, has been found in early life, and $\frac{6}{18}$ or $\frac{6}{24}$ in the forties, some of the old people see so badly as to be almost incapacitated. I could not feel sure that this result had been due to anything more than slow nuclear opacity of the ordinary senile type, although from a review of all the cases, the impression left certainly is that the typical radial tubular and dotted changes are decidedly more marked in the middle-aged than in the children. This, together with the fact that hitherto no cases have been discovered younger than eight years old, makes it doubtful whether the cataract in this pedigree is present at birth.* Not a single one of those affected has been operated upon. The teeth showed no abnormality of enamel. There has been a notable scarcity of early deaths, still-births, and miscarriages. Only one consanguineous marriage (between half first cousins) has taken place (II, 3a and 3), and it is too far back to assist in settling the source of the cataract in I, 1 and 2.

At the right-hand end of the pedigree are shown three cases of total blindness, probably due to glaucoma or some form of irido-cyclitis; only one of this stock, and he himself with healthy eyes (II, 8), married into the cataractous genealogy, and no bad effect has ensued.

* But in a pedigree of this form of cataract in 1905 several cases occurred in children between the ages of one and a half and five years. Nettleship, Roy. Lond. Ophth. Hosp. Reports, xvi, pp. 222-223.—E. N.
The following are the essential details of the pedigree:

Generation I, 1 and 2, a brother and sister, who according to the testimony of II, 3, 4 and 5, were all their lives affected with just the same defect of sight as the latter. I, 2 lived to be 86; his wife (1, 3) died of consumption at 35, after bearing him five children. I, 1 and 2 were two of nine or ten children of the same mother by two different fathers; it is not known how many belonged to each father.

II, 4 (misplaced in error, should precede 3), aet. 72 years, has radio-axial opacities and nuclear haze, complicated by corneal nebulae; sight very bad.

II, 3, aet. 66 years, labourer. Characteristic opacities with nuclear haze in addition. Could see to read until 40; sight now very bad. His wife, 3a, who was his half first cousin, as indicated on the pedigree, is dead.

II, 5, aet. 62 years. Two or three years ago Mr. Leslie Paton noted V. with $-1\ D. \frac{6}{3_6}$ with each; typical opacities (sketch made).

III, 1—4. Four children of II, 3 and 3a, all grown up, aged from about 35 to 25; eldest and youngest affected. III, 1 has V. $\frac{6}{1_2}$ with her H.m. 2 D. corrected; her teeth very good. No stillbirths or miscarriages.

III, 5—12, eight children of II, 4, of ages between 48 and 30. There were no stillbirths, etc. The four affected ones all have typical opacities. III, 5, aet. 48 years, has V.: R. $\frac{6}{2_4}$, L. $\frac{6}{1_8}$. III, 8, aet. 43 years, was seen some years ago by Mr. Treacher Collins, who found "fine dotted opacities at centre of each lens," and V. with $-6\ D.\ sph.\ -2.5\ cyl.\ \frac{6}{1_8}$ each eye. The opacities are not now all dotted, some being of the classical form.

III, 10, aet. 35 years; married some years but no pregnancies. Cannot remember sight ever being other than "short," and thinks it is very slowly getting worse; teeth good.

III, 12, aet. 30 years. Typical opacities radiating from a central patch or clump. Has one child (IV, 11) aet. 1 year; examined and lenses showed no opacity.

III, 13—20, eight children of II, 5, all living; no
history of still-births or miscarriages. Of the five affected three were examined; the other two refused to be looked at, but their near relatives were all agreed that they were as bad as, or worse than, any of the others.

III, 13 was examined in May, 1908, by Dr. L. W. Dryland, who found coarse striae.

III, 16, now 33 years, is interesting, as she was seen and the condition recorded by the late Mr. J. E. Adams at Moorfields when she was thirteen, the note then being—"Central opacities at the posterior surface of each lens, fundus normal. V.: R., + 1 D. sph., with −3 5 D. cyl. axis vertical \( \frac{20}{30} \) well; L., + 1 D. sph., with −3 D. cyl. axis oblique \( \frac{40}{10} \)." She married at 17, and has five children aged from 15 to 1 \( \frac{1}{2} \) (IV, 19–23), of whom one (IV, 21), aged 10 years, has typical radiating grey opacities, very good teeth, and brown irides; his two younger sisters (IV, 22 and 23) have blue and grey-brown irides respectively; the second (IV, 20) died at 11 months, the first (IV, 19) is 15 years and has perfect lenses.

III, 20, act. 22 years, has slight myopia; has always been "short-sighted" as long as he can remember; has the typical opacities and good teeth. He thinks his sight is now a little worse than when at school.

IV, 1, a cook, has been examined by Dr. Dring, who tells me she has opacities in the lenses; she declined to let me see her. Her next sister (IV, 2) has been examined by Mr. Leslie Paton, who found the usual appearances. In IV, 6, act. 10 years, I found small axial opacities; teeth good.

IV, 5, act. 16 years, has a single elongated dot of opacity a little upwards and inwards of posterior pole in R. lens; L. lens perfect.

IV, 7–9, three children of III, 8. Eldest (IV, 7), act. 8 years, typical opacities, but not very abundant; fundus Em. or slight H.; does pretty well at school. IV, 8 was stillborn, and 9 died at 8 months.

IV, 13, act. 9 years, son of III, 13, found by Dr. L. W. Dryland in May, 1908, to have radiating opacities like his
father, but less dense. Dr. Dryland found his elder sister (IV, 12), aet. 13 years, to have clear lenses.

IV, 19–23. See under III, 16.

II, 6 and 7, both dead. The latter left six children, all reported to be quite free from the family defect; they are scattered in various distant places and could not be seen. The former (II, 6) had nine children (III, 21–24), all free from the defect. Six died of consumption at about 20; two (III, 21 and 22) I have seen and found their lenses quite normal; the other living one (III, 23) was unable to come for inspection.* III, 21, aet. 53 years, however, has very bad vision from extensive old choroido-retinitis, with some shrinking of retinal arteries involving Y.S. region of both eyes, which seems to be in the same condition now as when she was a patient under the care of the late Mr. Streatfeild twenty-four years ago. The attack came when she was 29, three or four years after marriage. Was it tubercular or syphilitic? She has had no children.

Case 3 (Fig. 40).—Coralliform cataract: a small pedigree of perfectly typical cases living at Woking, named Tomes; family originally from Buckinghamshire.

III, 7, Nellie T—, Mr. Treacher Collins's patient at the Moorfields Hospital in June, 1900, then aet. 13 years. Sight was not considered to be bad by her family until the school teachers complained of it. Mr. Collins found V. 6/8 in each eye with and without correction of her high compound II. As. (L., H. 2.5 D. vertical, 5 D. horizontal; R., 2.25 D. and 4 D.). Characteristic tube- or trumpet-shaped opacities projecting radially forwards from axial part of lens, with separate dots of opacity between. I saw her at her home in January of the present year (1909), aet. 15 years; the eyes were no worse; dark hair, blue irides, good teeth. No history of fits or illness. Her father (II, 4)† was also seen by Mr. Collins in 1906, and his lenses found to be in almost exactly the same

* Marked by mistake with x as if he had been examined.
† Numeral omitted in pedigree by mistake.
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state as III, 7; his sight and that of two other daughters had always been "short" and somewhat defective as now. I saw him, aet. 53 years, at home at the same time as III, 7, and found the lenticular opacities no worse; irides blue, hair dark originally. At the same time I saw the other two daughters and their three small children: III, 2, aet. 30 years, blue irides, hair dark, but was lighter when a

child; opacities typical, rather more abundant in R. than L., refraction low H.; her only child (IV, 1), aet. 3 months, examined carefully and lenses found quite clear; III, 4, aet. 25 years, has opacities of the same kind and in same position, but the radii are longer and less crowded than in III, 2; refraction slight H.; her two children, IV, 2 and 3, aet. 4½ and 1½ years, examined and their lenses found to be quite clear. III, 4 has had eight children, one (III, 6) stillborn, the other seven all living, the eldest (III, 1) aet. 31 years, the youngest (III, 8) aet. 12 years. I saw III, 8, and found his lenses perfect; III, 1, 3, and 5 were

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not seen, but were reported to have perfectly good sight, quite different from the affected sisters. I, 3, a maternal aunt of II, 4, and personally known to him, who died at 60 "with every tooth in her head perfect," is well known to have had defective "short" sight all her life, and II, 4, from his description of it, evidently thinks it was like his own; she had an only child (II, 2), living, and now upwards of 60 and known to have perfect sight. All the others in I, II, and III are known to see or have seen perfectly well (except II, 7 and III, 11, who died in infancy too early for the state of sight to be known). There appear to be no special constitutional diseases, defects or degeneracies in the genealogy, except the cataract, and all whom I saw are intelligent well-developed people. All the children and grandchildren of II, 4 have either blue or grey eyes, and I did not hear of any brown eyes in the pedigree. II, 4 was 21 and his wife, now aet. 52 years, was 20 at marriage. II, 3 is aet. 58 years and II, 5, aet. 46 years. There has been no consanguineous marriage.

Case 4 (Fig. 41).—Senile cataract in husband and wife and some of their children; earlier incidence of the cataract in the second generation.

III, 5, was found by Mr. Fisher to have striae in the lower inner part of his L. lens when 50, and had noticed defect of this eye a year before that; in February, 1909 (aet. 52 years), I found no increase of the opacity. He is the fifth born in a childship of eight (III, 1 to 8), of whom III, 2, 7 and 8 died in infancy, and III, 1, aet. 60 years, 4, aet. 54 years, and 6, aet. 47 years, have perfect sight. III, 3, now aet. 57 years, married many years, but childless, had her L. eye operated for cataract at the Great Northern Hospital by Mr. Stanford Morton when she was 46, and the R. six months later (1897-98); result excellent in both. The mother (II, 4) had cataract removed from her L. eye by Mr. Morton at the age of 71, and lived to be 78: fairly good result, R. eye not operated. Her father
(I, 2) died an old man with good sight; her mother (I, 1) died at 30 after amputation of leg for diseased bone. The father (II, 5) became blind from cataract between 65 and 70, but being hemiplegic from more than one apoplectic attack was advised not to undergo operation; the father of II, 5 (not shown), lived to 78, and saw well to the end; but the paternal grandfather of II, 5 was blind for some years as an old man, cause unknown. One of his brothers (II, 6), still living and aet. 70 years, has cataract, and has been advised to have it removed, but declines. It is believed that none of the siblings of II, 4 or of II, 5 and 6 had cataract, at any rate they all died with good sight. The numerous descendants of II, 7 and 8 and the less numerous offspring of II, 1, 2, 3, are believed to have good eyes.

III, 5 has had four children, of whom two (IV, 2 and 5) died in infancy; IV, 3, aet. 26 years, has a single conspicuous vacuole a little below the posterior pole of her L. lens, and some As. and defect, the R. lens and V. being perfect. IV, 1 (only child of III, 4) wears glasses for short sight, and IV, 2, aet. 22 years, has perfect sight;
these two have not been examined. No consanguinity of any parents. Note that the L. lens has led as regards opacity in II, 4, III, 3 and 5, and IV, 3.

Case 5 (Fig. 42).—Inheritance of presenile anticipating cataract from father and of mental instability from mother. No consanguinity. II, 2, a man named Hiblen, whose R. eye was operated upon for cataract when he was 40, in June 1869, by Mr. George Critchett, at Moorfields; the cataract was complete and is said to have formed in eight months; good result. The notes state "L. commencing," but it is not clear whether this L. eye was ever operated; he died in 1885, at 56 years. This man was twice married; no consanguinity between him and either wife, nor between the two wives; eight children, all living, three by first wife, he being 33 at birth of eldest (III, 1); five by second wife, he being 51 at birth of youngest (III, 8); interval of seven years between the two childships. Two of his children, the first and last born, have had cataract. III, 1, now (1908) at 46 years, unmarried, had her R. eye operated for cataract.
(needling and suction) by Mr. Couper in 1881, when she was about 21, and got a good result; the L. was operated upon at another hospital in 1889, and did not turn out well; the R. cataract was, according to the description, of a speckly or spotted variety and V. with \(-2\) D. \(\frac{20}{100}\) at the time of operation. III, 8 came under Mr. Fisher's care (to whom I am indebted for the case) in September, 1908, aet. 28 years, single, for failure of L. eye of eighteen months' duration; lens opaque with coarse flakes and some small grey and green dots; no keratitis punctata: R. \(\frac{6}{6}\) partly; dots of grey opacity of various sizes scattered throughout the lens, none of them green, no keratitis punctata; iris dark brown, cornea normal, hair nearly black; incisor teeth show absence of enamel exactly as in lamellar cataract, the only surviving first molar normal; linear extraction of L. lens, result excellent. She considered her R. eye to be perfect, although its lens was full of dots; it is therefore quite possible that this dotted cataract, both in herself and her sister, was congenital. III, 2, aet. 43 years, single, and III, 3, aet. 40 years, have good sight and health; the six children (IV, 1-6) aged from 19 to 5, all see quite well. Of the five children of the second wife (III, 4-8), 4 and 5 are twins, and all except III, 8 have good sight; III, 5 and 7 are in an asylum, and the cataractous one (III, 8) is subject to mental depression; their mother (II, 3) is subject to delusions and has been "paralysed" for some years, and her father (I, 1) was alcoholic and violent. The two children, IV, 7 and 8, aet. 4 and 2 years, see well. I have not been able to examine any of this genealogy except III, 1 and 8.

Case 6 (Fig. 43).—(i) Presenile cataract in two or three generations; anticipation in some instances; (ii) lamellar cataract in a stock, perhaps related to (i).

(i) V, 8, Grace Deasley. Came under the care of Mr. Parsons, who kindly allows me to use the case, in May, 1907, aet. 12 years. V. then \(\frac{6}{36}\) or \(\frac{6}{60}\); had been
failing for two years. The lenses showed posterior polar opacity with striae running forwards from the nucleus towards the neighbourhood of the anterior pole, but the whole appearance was not that of typical coralliform cataract. All the incisors and all four first molars show deficiency of enamel, just as in lamellar cataract. Both lenses needled with very good final result. Her eldest sibling (V, 1), æt. 28 years (1907), was operated for cataract at Moorfields when he was 8, and has seen well ever since. Of her other six siblings, V, 2, 3, and 5 died young or in infancy, two of them from measles; V, 4, æt. 22 years, V, 6, æt. 17 years, and V, 7, æt. 14 years, see quite well. Their father (F. J. Deasley, IV, 8) began to notice failure of sight at 36; was operated upon for cataract at Moorfields by Mr., now Sir John, Tweedy in 1898 at the age of 44, and died in 1908, at 54. He (IV, 8) was the second of eight (all living) when the notes were made (1907), four having good sight and four (three besides himself) being either known or believed to have cataract. IV, 12 and 14 live at a distance, and it has not yet been possible to see them. It is notable that the normal and cataractous siblings alternate; the ages are from 54 (IV, 7) to 33 (IV, 14). IV, 10, now æt. 48 years, was operated for cataract when she was 18, and sees well. She has had one miscarriage (V, 12) and six children, who are all living, aged from 26 (V, 9) to 3 (V, 15); one of these (V, 10) began to complain of her sight at 12, and was operated at 19 for cataract, and has again recently been in under Mr. Flemming’s care. The children of IV, 12 and 14 have not been seen, but are believed to see well. Going upwards, III, 3 (John Deasley), father of IV, 7–14, was operated for cataract at St. Bartholomew’s a good many years ago, and died aged about 70 in 1903; his age when operated upon is not known. His father also is known to have had cataract, and it is not improbable that William Darsley (II, 3) was he, although the point cannot be decided. This William Darsley was operated for cataract at the age of 63 at Moorfields in May, 1857, by Mr.
Critchet (his residence is not given); he would be thirty-nine or forty years older than III, 3.

At the left-hand end of Fig. 43 is shown a small pedigree, which may possibly be related to the Deasley portion, the name of this smaller family being Dearsley; but although both families have made inquiries a common origin cannot be proved. It is noteworthy, however, that
I, II, I and 2, and III, 3 all came from the same district (Colchester, Clare in Suffolk, and Newmarket) and that the father of III, 3, whether represented by William Darsley (II, 3) or not, was from Bury St. Edmunds.

The sisters, IV, 2 and 4, are two in a childship of six, of whom two are living and normal (IV, 1 and 5), and two died at 13 and 5 months respectively (IV, 3 and 6).

I first saw IV, 2 at St. Thomas's Hospital in 1883, when she was 22. The history was that the cataracts had been seen in her eyes within one day of birth, and that repeated needle operations were done by Mr. Nunn and the late Mr. Rouse between the ages of 11 months and 6 years; the result had been unsatisfactory, and when I saw her there was much opacity of cornea, especially in R., with bare P.L. in that eye and no P.L. in the other. In 1908 I saw her again, æt. 37 years; there was no change. Her teeth have no defect of enamel. IV, 4, æt. 18 years when seen in 1893, and 32 in 1908, had iridectomy down and out in each eye by Mr. Bader at 5 years of age for small, very dense lamellar cataracts (4 to 5 mm. diameter). She can read J. 6 held very close with the L., and this was the same in 1893. In 1898 I removed the lens from the R. with good surgical result, but she prefers the L. for use, probably owing to the nystagmus, which negatives the use of the correcting lens for the aphakic eye, though with + 10 D. she sees 6/60. Teeth quite good.

The parents (III, 1 and 2) are first cousins in the way shown; the father (III, 2) was an only child, and his parents were not consanguineous; was barely 20 at marriage, is now 60. The mother (III, 1) was 24 at marriage, now 65; is fourth in a childship of seven, of whom Nos. 6 and 7 died under a year old; No. 3 died at 40; and the other three besides III, 1, viz., Nos. 1, 2, and 5, are living, aged from 72 to 63. These four siblings of III, 1, have had about twenty-five children, most of whom are living, and none have had cataract. The marriage between III, 1 and 2 is the only consanguineous one known.
Case 7 (Fig. 44).—Senile cataract passing by anticipation into presenile. Family of Oldfield and Woodcock; home at Whittlesea, Cambridgeshire.

I, J, James Woodcock, was operated upon for cataract at Moorfields on May 19th, 1841, by Mr. Tyrrell; the eye (L) suppurated and was lost; he died at 85 in 1873,
never having had the other eye operated. He had seven children; the firstborn (II, 4) died at 47 of cancer of pylorus; four of the remaining six died between the ages of 64 and 79; the other two are believed to have died young; all kept good sight to the end. II, 3, wife of II, 4, also had good sight all her life; she and her husband not related. III, 1 and 3 to 8, seven children of II, 3 and 4. The firstborn (III, 1) died at 40 with good sight, leaving six children all with very good sight; the last (III, 8) died at 3 weeks. The other five are living, four of them having perfect sight: one (III, 3), the second-born, has had cataract; she had good sight till after the birth of her first child when she was about 26; she was operated upon for cataract by Mr. Bader when about 29, did well, is now 60 and might be improved by a needle operation. She has had sixteen conceptions, four ending in miscarriage; twelve born alive, of whom three (IV, 7, 9 and 11) died at 8 months, 18 months, and 5 weeks; nine are living, and four of them certainly, perhaps five, have double cataract.* The cases are: IV, 2, James Oldfield, now aet. 31 years (1908); was operated for cataract when 22 by Mr. Spicer at Moorfields (1899). IV, 3, Alice, now aet. 30 years; sight began to fail at about 28; operated at Moorfields by Mr. Morton, June, 1908. IV, 8, William, a twin, now aet. 23 years; cataract began at 20, and when first seen the opacity was posterior polar with spokes, each of which had enlargement on its anterior end; operated upon by Mr. Parsons (to whom I am indebted for the opportunity of working out the pedigree) in November, 1908. IV, 12, Arthur, now aet. 19 years; cataract begun at 14, and was almost complete at 15, when he was operated upon (1905) and got V. 6. All have done well and regained good sight after operation. IV, 16, aet. 17 years, a cousin of IV, 1—12, had cataract removed from her L. eye by the late Mr. Bell Taylor at the age of 4 (paper seen). Dr. Kirkwood, of Peterborough, has been kind enough to examine her R.

* The doubtful one is IV, 5; he was living at a distance and I could not see him; his mother replied rather dubiously about his sight.
lens at my request lately, and finds it perfectly clear; Dr. Kirkwood also examined several of her brothers and sisters and found no trace of cataract in any; probably the cataract in IV, 16 was not of the same variety as in the others. III, 2, the father of the cataractous siblings, is now at 59 years with perfectly good sight; he was one of a childship of five brothers and two sisters, of whom one brother and one sister died at 35; the other four lived to over 60, all with good sight (not shown in Fig. 44): his father, II, 1, died with good sight at 59 years old; had two brothers and four sisters, all with good eyes, and one brother (II, 2), James Oldfield, who was operated upon for cataract at Moorfields when about 58, dying at 63. No consanguineous marriages. The whole stock on both sides appears to be of ordinary soundness, and no one disease has been prevalent except ague, from which III, 3 and all her siblings suffered badly when they were children. Her children (IV, 1–12) were all born at full time and all suckled; they are decidedly above the average of their class in stature and intelligence; the teeth are good in all; irides grey-blue in all whom I saw, cataractous and normal alike. There have been at least four twin-births in the pedigree—IV, 5 and 6, 8 and 9, and twice amongst the eight children of III, 6, seven of whom died young.

(March 11th, 1909.)

46. Some observations on so-called artificially produced temporary colour-blindness.


An elaborate and painstaking investigation on this subject was made by Prof. George J. Burch of Oxford, and a paper on it was published in the Philosophical Transactions of the Royal Society in 1898. The experiments were mostly made upon himself, but in addition he examined the colour sensations of 109 people.
For the purpose of producing artificial colour-blindness upon himself he used a horizontal beam of sunlight from a heliostat, and passed it through a series of prisms. About twenty feet away he placed a convex lens of three inches focus in the path of the refracted rays, adjusting its position until, on examining the light collected by it with a hand spectroscope it was found to include only that portion of the spectrum which he desired to use. He then exposed his eye for a few seconds to the extremely bright light in the focus of the lens, and immediately afterward looked through a single prism spectroscope illuminated by the light of the sky, and compared the result seen with the other eye which had not been so treated. In this way he was able to saturate the eye with any colour he wished to investigate, and could in rotation expose the eye to all the spectral colours.

He found that all direct sensation of the colour used for fatiguing the eye was practically lost; that the temporary abolition of any one primary colour sensation was without effect on the intensity of the remaining colour sensations.

He believes that there are four primary colour sensations, *viz.*, red, green, blue and violet, and each of these colours could be washed out of the spectrum seen with an eye which had previously been exhausted by the same spectral colour.

He considers that there is no special colour sensation for yellow, because "yellow results either from the simultaneous excitation of the green and red sensations, or from the action of a weak red light during the positive after-effect of green." "Blue is not a mixture of green and violet, for blue and violet are both visible during green blindness, and green and blue are both visible during violet blindness. Violet is not a mixture of blue and red, for the violet is unaltered during red blindness. "Light from D produces blindness to both red and green; light from F produces blindness to both green and blue; light from G produces blindness to both blue and violet."
He concludes from his experiments that his results are unfavourable to the theory of Hering, but that they confirm that of Young and Helmholtz, but indicate the existence of a fourth colour sensation, viz., blue.

It is these experiments and conclusions which we thought well to investigate, and we wished to see whether we could detect any error, either in the method of experimenting or with his conclusions.

We do not desire in this communication to discuss Prof. Burch's paper in detail—that would take far too long—but we wish to criticise one or two points.

In the first place let us consider his method. In order to fatigue his retina he used not only rays of direct sunlight as they emerged from a spectroscope, but actually employed a convex lens of 3 inches focus to concentrate the light on his retina. It is true that this would produce most effectual fatigue of the retina, and although probably none of us would object to run a certain amount of risk in the pursuit of scientific knowledge, yet I fancy we should have to look far before we could find an ophthalmic surgeon doing anything so unwise as to concentrate direct sunlight on his own retina, that is if he ever expected or wished to use his eyes again. We have probably all of us seen quite enough cases of eclipse blindness to deter us from offering our own eyes upon such an altar of sacrifice as this.

In consequence of the manifest disadvantages of using such a powerful light, light which would at least temporarily, and probably permanently, impair the observer's eye and vitiate his observations, we determined to investigate the matter with lights which would produce all the fatigue necessary, but which would not either endanger our own eyes, or produce a scotoma from which it would take minutes or hours to recover. In addition we considered that with our eyes not more fatigued than would be consistent with health, that our judgment would be far more reliable. Quite apart from the fact that Prof. Burch used light so strong that he was unable read large print for a
considerable time afterwards, he seems to have overlooked the fact of the great changes brought about by luminosity contrast. He first blinded his eyes with direct sunlight and then used a direct vision spectroscope illuminated by the sky only. The effect of this may be illustrated by the following experiment: If two pieces of the same grey paper are laid, one on a white surface and one on a black surface, the one on the white looks darker, and the one on the black lighter than it really is. Therefore, as yellow is such a luminous colour, the other colours of the spectrum, which are not so luminous, are diminished on being subsequently viewed.

The one point which struck us more than any other was the statement that light from D (orange-yellow or the band of the sodium flame) produced blindness to both red and green.

The way to test this safely was to produce fatigue by using a sodium flame and staring at it for some minutes. We used both pure sodium and sodium chloride, and obtained a perfectly pure orange-yellow light. A spectroscope was alongside in which a spectrum was visible from an electric lamp, and which gave a good yellow band. The lamp was screened so that it could be burnt in the darkened room in which we were working without a ray being visible except through the spectroscope.

On exposure of the eye to the sodium flame for from three to fifteen minutes and then looking at the spectrum we found the yellow entirely obliterated, and only a faint band of orange separated the red and green, but if the eye were still further fatigued this also was obliterated, and the red and the green met without anything whatever intervening. The red looked rather more purple, and the green was slightly tinged with blue. The blue and violet at first appeared greatly diminished in intensity, but not changed in character. There was no shortening whatever of the red end of the spectrum; it terminated at precisely the same place as it did previous to the eye being fatigued with the yellow light. It also produced no trace of a
scotoma. If one eye only were exposed the other remained perfectly normal and could be used as a control. This is absolutely opposed to the observations of Prof. Burch, who found red and green had disappeared, and it was due to this experiment more than any other that he held he had established the complex nature of yellow light, and in which he upholds the Young-Helmholtz theory.

We next tried exhausting our eyes with different colours, and for this purpose we took a piece of pure red glass, which on spectroscopic analysis we found was transparent to the red rays only. With our eye protected with this we looked intently into an incandescent electric lamp, and on looking into the spectroscope we found the red obliterated as red. It was shortened to from one half to three quarters of its extent, and here we were unable to see any light at all; the part we could see was changed to orange. The true orange now became yellow, and the yellow yellowish-green. Fatiguing the eye with green light caused the yellow and blue to encroach on the green. If the eye were saturated with blue light the blue disappeared and the violet and green met. If saturated with violet light the violet became indistinguishable from the blue and the real blue became greenish.

These experiments add additional weight to the psycho-physical theory (Edridge-Green theory) of colour-perception, and it is impossible to explain them on either the Young-Helmholtz or Hering theory.

Further experiments are required in order to measure exactly in wave-lengths the alterations noticed, but in this communication it was thought unnecessary to do other than explain the main facts.

In experiments of this kind the following factors have to be taken into consideration:

1. The luminosity of the primary light which is used to fatigue the eye.

2. The luminosity of the light which is subsequently viewed.

The relation between these two is important, and it is
SO-CALLED ARTIFICIALLY PRODUCED COLOUR-BLINDNESS.

best to use a primary light which is slightly stronger than the secondary light. When the lights are very unequal the element of luminosity contrast greatly influences the result, as in viewing a piece of ordinary white paper after having looked at pure snow, while everyone is familiar with the dirty appearance of an ordinary white blind or curtain when it is backed up by freshly fallen snow.

(3) The duration of the primary stimulus.
(4) The effects of simultaneous contrast.
(5) The wave-length of the primary and secondary stimulus.

(6) The purity of both lights, that is, their freedom from white light or light of other wave-lengths.

A careful consideration of the facts given will show that they may be classed under two heads:

(1) Those due to psycho-physical perception.
(2) Those due to colour-fatigue of the retina and brain. The first class includes all those changes in which the colour appears to be shifted to a higher or lower point in the colour scale. The difference between the colours is exaggerated.

When the colour-perceiving centre in the brain has been fatigued by impulses caused by light of a certain wave-length, it and the retina are less responsive to rays of that kind, and objects subsequently viewed appear to be deficient in the corresponding colour.

(March 11th, 1909.)
APPENDIX.

The following cases and communications have also been brought before the Society:

1. Flattening of both Corneæ after Phlyctenular Keratitis, A. H. Thompson and F. Hewkley.

2. Retinal Changes, the result of Concussion Injury, R. E. Bickerton.

3. Annular Corneal Arcus in a Man, act. 27 years, Arnold Lawson.


5. Stereoscopic Colour Photographs (Lumière process), J. Rowan.

47. Exhibit of three instruments: (a) an optic nerve hook for use in the operation of optico-ciliary neurectomy; (b) an exsiccation spoon; (c) an elevator for use in extirpation of the lacrimal sac.

By Major R. H. Elliot, F.M.S.

Optico-ciliary Neurectomy Hook.

The blade of the hook is 5 cm. long, and the handle 10 cm. long. The large curve of the blade has been moulded on the antero-posterior curve of an average eye, and corresponds to a little less than half the circumference of such an eye.

The extremity of the hook is bent back 4 mm. in the smaller curve, the opening of which has been so graduated that it will admit and hold firmly an optic nerve. The instrument shown was made for me by Messrs. Arnold and Sons.

After division of the internal rectus muscle the hook is slid round the eyeball till the insertion of the optic nerve is passed; a slight rotation of the instrument on its main axis then engages the nerve. The instrument is gently withdrawn till the nerve is tense, and the section is made in the usual way with scissors. It is easy to cut the nerve 10 to 12 mm. behind the globe. The eyeball can now be easily rotated (till the nerve presents in the

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wound) by a suitable movement of the instrument, which never slips from its hold on the nerve during this manœuvre.

With a little practice the whole operation becomes quite easy.

**Evisceration Spoon.**

Easy to use and to sterilise. Removes the contents of the eye quickly and easily, whilst it does not damage the sclerotic coat.

**Elevator for Lacrimal Sac Operations.**

This is simply an ordinary periosteal elevator reduced in size for the purposes of the above operation.

Both instruments were made by Messrs. Arnold and Sons, from models made in an Indian bazaar at a cost of a few pence each. *(May 6th, 1909.)*

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48. *A case of congenital anterior staphyloma.*

By **Sydney Stephenson.**

Thomas H—, born March 23rd, 1909, admitted to the Queen's Hospital for Children, London, on March 31st,
CASE OF CONGENITAL ANTERIOR STAPHYLOMA.

1909, under the care of Dr. George Carpenter, who was good enough to transfer the baby to me.

History.—Patient is the first and only child. He was born after a non-instrumental labour, the first stage of which lasted a little over twenty-four hours. The membranes ruptured twenty minutes before the head was born. When the mother was three months pregnant some boiling bacon fat splashed into her eyes. In consequence of the accident she was unable to open her eyes for four or five days, and attended St. Bartholomew's Hospital for about three weeks. The child came into the world with eyes in the condition they now are.

State on admission.—A small baby, weighing $4\frac{1}{2}$ lbs. Each palpebral fissure is occupied by a globular, translucent, fleshy mass, in which no trace of cornea can be recognised. The mass protrudes from the eyelids, and is larger on the left side than on the right, where it is about the size of a small marble. The masses move in conjunction with the globes, which can be seen to be present in the orbits. The eyelids cannot be closed so as to cover the fleshy protuberances, which accordingly are always exposed to view whether the baby is asleep or awake. Each palpebral fissure is extended by a slit-like prolongation beyond what should be the outer canthus, thereby recalling the congenital anomaly known as macrostoma or congenital enlargement of the mouth. There are no evidences of recent inflammation as regards the palpebral conjunctiva, which is smooth, thin, and merely reddened. In particular, there is nothing in the condition of the conjunctiva to recall a past ophthalmia. No discharge is present from the conjunctiva.

Other congenital malformations are present, as follows:

1. The head is spherical in shape, measuring $11\frac{3}{4}$ inches in circumference (microcephalus).
2. The ears are normal as regards shape, but from the posterior surface and the helix there grows forward a fringe of long, soft, dark hair.
3. The right testicle cannot be felt in the scrotum, although the left one is in place (monorchism).
4. The
little finger is small and incurved—it is, in fact, of Mongolian type. (5) The lower extremities show several deformities: (a) no movement of the hip-joint can be elicited; (b) the patellæ appear to be absent; (c) the knees cannot be flexed, but extension is possible to the extent of a right angle on both sides (genu recurvatum).

Remarks.—The appearance of the eyes is more suggestive of maldevelopment than of inflammation. The association of the staphyloma with a macrostoma-like condition of the eyelids, microcephalus, monorchism, genu recurvatum, and absence of the patellæ appears to point in the same direction.

(May 6th, 1909.)

49. An unusual appearance of the optic disc, the result of a previous papillitis.

By G. Winfield Roll.

K. W,—, æt. 6 years. Defective sight in the left eye believed to have existed since birth with inward squint.

The child never had brain fever or convulsions; has always been healthy with the exception of a severe attack of measles at Christmas time, 1908.

The mother had a right convergent strabismus which she has outgrown. There is defective vision in the mother’s right eye, but the optic disc presents a normal appearance. Her first child only survived thirty-six hours; three other children without defect of sight.

The left disc gives an impression of large size, with shallow depression of surface and a greyish colour. The centre of the disc is occupied by a greyish-white mass of irregular outline. There is a ring of atrophy around the disc with well-defined limits. The vessels of the retina are arranged around the disc hooking over the edge. One or two reach near the centre appearing below,
the connective-tissue mass in the centre. A group of vessels passing up and in present white lines. A few vessels branch after passing the edge of the disc; many are devoid of branches. Some areas of choroidal disturbance, notably above the disc, with pigmentation.

Fig. 48.

V. R. \( \frac{9}{6} \); fundus normal.  

(May 6th, 1909.)

50. A case of nodular leprosy affecting the eyes.

By E. Treacher Collins.

(With Plate XI, fig. 2.)

The patient, æt. 15 years, and his father, both suffering from leprosy, were brought to me by Dr. McMahon, of Cork, on April 22nd, 1909.
The father, aged 40 years, is a Pole who has resided in Ireland for twenty years; he came there from Riga. He says the first symptoms of the disease appeared in him five years ago, and he thinks he contracted it from a woman who lodged in the same house with him, and who was suffering from it. She had come recently from Riga. He has three children, and the boy shown this evening is the only one affected; he was born in Ireland and has always lived there. The disease commenced in him two years ago. The condition of the father is said to have somewhat improved of late, but that of the son has steadily become worse; so that the disease appears to be now in a more advanced stage in the son than in the father. The boy has been treated by a doctor in Cork with injections of "Nastin B.O." but is stated to have become worse after them. He has also had injections of fibrolysin.

The boy's manner is dull and apathetic. He does not readily reply to questions; formerly he used to be bright and intelligent. The bridge of his nose is very depressed; at one time it was well formed. Early in the disease he had attacks of epistaxis and discharge from the nose. His upper lip is swollen and protuberant. His cheeks are thickened and face expressionless. The skin of his face is mottled with reddish patches, and numerous hard nodules are to be felt in various parts of it. There is one very prominent nodule in the centre of the right upper lid.

Hair is absent from the right eyebrow on the inner side, and the right upper lid is devoid of eyelashes.

There are numerous nodules to be felt in the skin of his arms, the calves of his legs, his buttocks and back. The nodules about his elbows are beginning to break down and ulcerate.

A very definite nodule can be felt involving his right ulnar nerve. A comparison of his two hands and forearms shows wasting of the muscles on the left side. There is anaesthesia over the distribution of the ulnar nerves,
Fig. 1.—An illustration from Dr. George Carpenter’s paper, "A further Clinical Contribution to the Study of Tubercle of the Choroid" (p. 228).

Fig. 2 illustrates Mr. E. Treacher Collins’s case of Nodular Leprosy affecting the Eyes (p. 223).

Fig. 3 illustrates Mr. E. Treacher Collins’s case of Symmetrical Circumferential Encroachment on the Cornea by the Limbus of the Conjunctiva in the two Eyes, with an Arcus Senilis inside the Limbus, the patient being also the subject of Progressive Ptosis (p. 225).
most marked on the left; also over areas in his legs below the knees.

In his right eye there are two large yellowish swellings with blood-vessels on them, at the upper and lower margin of the cornea. Each extends some distance into the cornea, and only a narrow strip of semi-opaque cornea is left uninvolved between them.

V. = Counting fingers at one metre only.

In his left eye a similar yellowish swelling is seen at the lower margin of the cornea extending some distance into it. There is more clear cornea in this eye than in the right. The iris is discoloured and has some small yellowish nodules in it near the pupillary border up and out. There are numerous posterior synechiae and an inflammatory membrane fills the pupil.

V. = doubtful P.L.

Dr. Wedd made smears and sections from a nodule on the leg, and found the typical appearances of leprosy with enormous numbers of acid-fast bacilli.

(May 6th, 1909.)

In reply to the President, Mr. Collins said there was nothing in the dietary which he knew of to throw light on the case, but he had not investigated that point.

51. Symmetrical circumferential encroachment on the cornea by the limbus of the conjunctiva in the two eyes, with an arcus senilis inside the limbus, the patient being also the subject of progressive ptosis.

By E. Treacher Collins.

(With Plate XI, fig. 3.)

Mrs. M. B—, æt. 69 years, attended at Moorfields Hospital on April 29th, 1909, complaining of defect of sight and inability to open her eyes.

On examination it is found that she has very marked
ptosis on both sides, slightly more on the left than the right. She is only able to see beneath her upper lids by throwing back her head and contracting the skin of her forehead with her occipito-frontalis muscle.

There is considerable sinking in of the skin immediately beneath the upper margin of the orbits, showing that there has been much absorption of orbital fat.

She states that she has never been able to open her eyes as wide as most people, and that her difficulty in doing so has very much increased of recent years. These statements are borne out by the examination of two photographs of her, and the comparison of them with her present condition. A photograph taken thirty years ago shows her with rather narrow palpebral fissures, but apparently making no effort to raise her lids by means of her occipito-frontalis muscle, and not throwing her head back. In a photograph taken ten years ago the palpebral fissures are narrower and unequal, the left being narrower than the right, but neither so narrow as they are at present. She is seen to have been contracting the skin of her forehead to some extent, but held her head straight.

On raising her upper lids and examining her corneas, only an oval central portion in each, with its long axis horizontal, is seen to present its normal transparency. The outline of the normal cornea can be made out, but the peripheral portion of it in its entire circumference is opaque, the band of opacity being broader above and below than at the sides. The opaque area is of a greyish colour, not densely white like the sclerotic. Blood-vessels continuous with those of the conjunctiva, and about as numerous, extend into it.

The appearance of the opacity, which is exactly symmetrical in the two eyes, suggests that it is due to the limbus of the conjunctiva having become prolonged for some distance over the surface of the cornea, instead of terminating in the usual position at its margin.

In the peripheral part of the oval area enclosed by the
circumferential opacity is an opaque ring, similar in appearance to an arcus senilis. It is separated from the circumferential opacity by a semi-opaque zone in the same way as a normally situated arcus senilis is separated from the limbus.

The pupils are circular and normally active. Only the pupillary margin of the iris is visible through the oval central transparent portion of the cornea. When the pupil is dilated with a mydriatic the whole of the iris becomes hidden from view.

There are some cortical lens opacities. The optic discs and fundi show no changes.

Vision: R. = 6/21, c — 1.75 cyl, axis 50° down and out 6/12; L. = 6/12 partly, unimproved.

The patient is a strong, healthy woman for her age, and gives no history of any constitutional disease or local inflammation likely to account for the condition of her eyes.

Remarks.—It is probable that in this case the ptosis was partly congenital but that it has increased in amount of late years so as to be almost complete. Cases of progressive bilateral ptosis of a similar character have been recorded by Fuchs under the heading "Isolated Bilateral Ptosis" (Arch. f. Ophth., xxxvi, 1, p. 234). All his five cases were females. In three no ptosis appeared until late in life. In the other two, as in my patient, the ptosis was either congenital or developed in early infancy and subsequently progressed to completeness. In one of his cases he removed a portion of the levator palpebrae muscle, and found on microscopical examination that its muscle-fibres had undergone extensive degeneration, that there was an increase in its connective tissue but no fatty changes.

I can only recall seeing one case with a circumferential opacity of the cornea having any resemblance to that in this patient. It is recorded and figured in the Transactions of this Society (vol. xxiv, p. 45). The patient was a man, aet. 21 years, and, as in this case, the condition had come
on without any known cause and was symmetrical in the two eyes, leaving only a central oval area of each cornea unaffected. Unlike the present case, however, there were no blood-vessels detected in the opaque areas and there was no arcus senilis.

The formation of an arcus senilis so much nearer the centre of the cornea than usual, and yet at the same relative distance from the limbus which it generally occupies, is a feature of interest in this case. (May 6th, 1909.)

Mr. Holmes Spicer said the condition of the cornea might be congenital. One saw not uncommonly a certain amount of difference in the encroachment of the limbus on the cornea. He had seen it marked in children, but not so marked as in the present case. He thought it was usually congenital; it occurred in many cases in which there had been no history of inflammation or other disease in the eye. And taking that with the fact that to some extent the ptosis dated from birth he suggested that the whole condition might be congenital. He supposed the ptosis in its advancing condition was closely allied to that seen occasionally in cases of myasthenia gravis.

Mr. Collins replied that there were no other evidences of myasthenia in the patient.

52. A further clinical contribution to the study of tubercle of the choroid.

By George Carpenter, M.D.

(With Plate XI, fig. 1.)

It was my intention, and I hoped to have been able, to exhibit a case of multiple miliary tuberculosis of the choroid in both eyes this evening, but unfortunately the child in the interim has become rapidly worse, which has prevented her removal from hospital.

This patient is a girl, aet. 7 years, under the care of my
colleague, Dr. Porter Parkinson, at the Queen's Hospital for Children. She has been getting thinner for some five weeks, and was admitted into the wards with physical signs at the right apex in front and behind. She also gave a history of abdominal pains, but nothing could be discovered in her abdomen by palpation. For the last few days she has shown signs of drowsiness, and the most noticeable features at present are extreme wasting of comparatively sudden onset, and she is becoming comatose.

In the fundus of the left eye there are six choroidal tubercles. One just above the macular region is a small fawn-coloured object of irregular triangular outline. The other tubercles are large and four of them are arranged in pairs. They are situated above and to the outside of the macular region, and about the same distance from the macula as the macula is from the optic papilla. The sixth tubercle is on a level with the lower margin of, and to the inside of, and some little distance from, the disc.

By indirect examination the tubercles are of oval outline, of a whitish-yellow colour, and have flecks of retinal pigment scattered over them. The retinal bloom appears to have been partially rubbed off, leaving this underlying discoloration described. At the periphery of the tubercles the retinal pigment seems to be a trifle darkened—an effect which is possibly due to contrast. Three of the tubercles are crossed by retinal vessels in perfect outline.

Viewed by the direct method they are not so contrastive. Light, fawn-coloured objects, they gradually merge into the red reflex. Covering them is the most delicate veil possible of very fine retinal pigment granules; every here and there the stippling becoming somewhat denser. Even the tubercle at the yellow spot which appears clear fawn-colour in its centre by indirect examination has very fine retinal pigment granules covering it.

In the right eye there are two small tubercles, one on the disc side at the periphery of the macular zone, the
other above and two discs' diameter outside the optic papilla. The tubercle at the yellow spot region is light fawn-coloured, and has a fine veil of retinal pigment completely covering it and is not sharply bounded. The second tubercle is identical in appearance with those shown in the drawing.

The other cases of the complaint that I am about to bring to your notice unfortunately died a short time after the discovery of the lesions in the eyes, and I am only able to exhibit pictures of the fundus oculi accompanied by a brief clinical account.

The next on my list, Gladys A. M. T,—a girl, aet. 2 years, had been ailing for six weeks and had been suffering from sickness and headache for five days when she was admitted under my care at the Queen's Hospital for Children on February 10th, 1909. When I saw her she was suffering from meningitis, and she died on the sixth day after her admission.

The right fundus oculi was quite normal; no tubercles or optic neuritis could be seen there, but on the left side there was obvious, though slight, optic papillitis. Close to the optic papilla and to its upper and inner side was a tubercle in the choroid of considerable size, of circular outline and a trifle smaller than the swollen papilla. In colour it was whitish with a shade of yellow mingled with it. Though a well-defined ophthalmoscopic object it was not sharply cut, and it passed imperceptibly into the red reflex, the transition being quite gradual. Several blood-vessels, arteries and veins, crossed its face in perfect outline.

The retinal arteries were somewhat small but there was nothing very noticeable about the retinal veins, in spite of the fact that the surface vessels of the brain were found to be intensely congested. Death was sudden: without warning she became cyanosed and asphyxiated, and died immediately. The autopsy verified the diagnosis of tuberculous meningitis and extensive general tuberculosis was also found.
The third case is that of a boy, George A—, aet. 17 months, whom I first saw on March 1st, 1909. He had been ailing for ten days, and when admitted into hospital he had double otorrhoea, and some moist sounds were detected over the base of the right lung. Two or three days afterwards he became drowsy and displayed signs of meningitis, and a lumbar puncture was made at a later date, which corroborated the diagnosis of tuberculous meningitis which had been pronounced on the evidence of the eye examination.

Owing to the great difficulties experienced in examining this child's eyes, the drawing of the right fundus oculi here shown had to be made by the indirect method.

Just below and to the outside of the macula and nearly as far from that region as the macula itself is from the optic papilla was a tubercle, white in colour with a shade of yellow in it, and somewhat smaller than the optic papilla (Pl. XI, fig. 1). Its lower margin had a toothed edge, and the intensity of the retinal pigment was somewhat heightened at this edge. Its upper margin was crossed by a retinal vein, and was shaded off into the red reflex just above that vessel. A small artery also crossed it, and both artery and vein were in perfect outline. There was no optic neuritis, and the tubercle described was the only one seen.

The child was under observation for six more days but nothing happened to the tubercle in the interim, nor were there any fresh tuberculous deposits. At the post-mortem examination tuberculous meningitis and general tuberculous were found.

The fourth case is that of a boy, George H—, aet. 2 years, who had had a bad cough for five weeks, and was admitted under my care on March 9th, 1909, with consolidation of both bases, and he was thought to be suffering from simple broncho-pneumonia on the evidence of the physical signs in his chest, but five days later he was sick. I then became suspicious and examined the fundus oculi.
Just above the yellow spot a small, oval, fawn-coloured tubercle was detected. It was crossed by a small retinal vein at its lower margin, and the tiny body was thrust through, as it were, the retinal pigment, which in this region was of some density. The retinal pigment in its periphery appeared to be minutely fissured in various and strictly limited situations. There was also a small amount of optic papillitis in both eyes. He died twelve days after admission, no changes in the fundus oculi having taken place in the meanwhile. At the autopsy the lungs and bronchial glands were in an advanced stage of tuberculosis, he had tuberculous meningitis, and tubercles were found in other organs of his body.

My fifth case is that of a girl, H. M—, æt. 5 years, and is perhaps the most interesting of the series; it is certainly the most extensive and varied. She was admitted under my care on January 11th, 1909. She had been wasting since whooping-cough four months previously, and had been short of breath and drowsy for a week. When I saw her she had signs of patchy broncho-pneumonia, her right heart was markedly dilated, her spleen and liver enlarged, and she was cyanosed.

An examination of her blood-corpuscles showed—red 5,500,000 per c.mm., white 23,000 per c.mm., and haemoglobin 85 per cent. She was in hospital for nine days and during that time had slight fever. Cyanosis increased and she was also delirious. Slight dropsy of her feet occurred one day before her death. The urine was free from albumin during her illness. She died suddenly.

The nature of her illness was determined by the ophthalmoscopic examination which I made when I first saw her, and her eyes were frequently under observation during the whole course of her illness.

In the left fundus oculi there were two tubercles, and in the right I detected ten scattered in various parts, four of them being near the macula. They were mostly oval in outline, and varied in extent from the apparent size of
a hemp-seed to a body which was more than half that of the optic papilla.

One tubercle crossed by an artery and situated near the disc was elongated, and another in the periphery was of irregular shape. Retinal arteries and veins, when crossing the tubercles, presented natural outlines. The largest tubercle, a very prominent ophthalmoscopic object, was very pale in the centre. Half way round and bounding this inner pale zone was a fringe of irregularly shaped grey pigment-granules. The outer zone was not so pale; it was dotted with red and gradually merged into the surrounding red reflex.

At the macular region was a small, very bright, almost white tubercle which was well defined. Two other tubercles just above it were also bright, but not so bright. The fundus was stippled over with grey pigment-dots which extended over two of the tubercles. The other tubercles were pale fawn-coloured appearances of the red reflex, as though the colour of this region had been altered by some chemical application, and they were flecked with red. The tubercle of irregular shape in the periphery was light in tone and presented a sharply defined edge.

The arteries and veins were markedly tortuous and the veins full of dark blood. The brain in this case, when viewed at the autopsy, was not congested. The child, as I have already mentioned, had recently had whooping-cough, and I think the condition of the retinal veins and arteries may possibly be ascribed to that complaint, and for the following reason: In the discussion on whooping-cough at the Royal Society of Medicine in January last, I drew attention to just such a condition of retinal blood-vessels as that now shown which is found in certain cases of whooping-cough, though in many it is certainly absent. My observations in this direction have been fitful rather than continuous, but I think my impressions warrant that this matter should be more closely inquired into. At the autopsy extensive general tuberculosis was found through-
out the body. A few tubercles were seen at the apex of the cerebellum, but there was no meningitis.

The choroidal tubercles in these cases conformed to the types which are usual in this infection, and to which I have called attention in other contributions on this subject during the past twenty years, both independently and in association with my friend Mr. S. Stephenson. Viewed by the indirect method of examination they are much more contrastive than when seen by the direct.

Departures from the usual oval or rounded forms occurred in only two of the tubercles described, one of them being of triangular outline, the other displaying an irregular figure. Pigmentation in gross masses is not a characteristic of young choroidal tubercles, though flecks of fine retinal pigment covering or splashed upon them are common enough. Pronounced pigmentation does not occur in choroidal tubercles until they enter the chronic stage, and during their passage from that to obsolescence.

One of the cases narrated did show pigmentation to some slight extent in one of the lesions, this doubtless being the oldest deposit in that particular case.

Just as scarring of the skin indicates a one-time destruction and subsequent repair of the derma, so does atrophy and pigmentation of the choroid denote the destruction and subsequent repair to which that structure has been formerly subjected. Coarse pigmentation of choroidal tubercles therefore denotes chronicity and obsolescence. It will, I think, be readily granted that there is nothing distinctive in the ophthalmoscopic appearances of young choroidal tubercles.

Just as the grosser syphilitic and tuberculous lesions of the fundus oculi are indistinguishable in their early stages, and it is then only by the combination of signs and symptoms in association with them that they are correctly diagnosed, so in like manner it is impossible to separate the syphilitic from the tuberculous in the atrophic stages by their ophthalmoscopic appearances alone.

Although acute choroidal tubercles in comparison with
corresponding syphilitic deposits are quite common ophthalmoscopic objects, that is due to the routine use of the ophthalmoscope in cases of medical tuberculosis. Of course their appearances can scarcely be very familiar to those who practise ophthalmology pure and simple, since they produce no visual disturbance and are not likely to be seen at an ophthalmic hospital.

In both syphilis and tubercle patches of atrophy follow the corresponding choroidal deposit if the patient survives, and in both disorders pigmented disturbance must necessarily be a common sequence. There is nothing pathognomonic about choroidal pigmentation. Pigmentation in this situation simply denotes the reaction of the living choroid towards a variety of injures, such as trauma, syphilis, and haemorrhage, to mention well-recognised conditions, and arises from a proliferation of the pigment epithelial cells as well as from an increase in their contained pigment-granules. This pigmentation is apt to environ and be deposited in old specific lesions, and possibly may be essentially protective in design—the reaction of the living choroid pigment-cells to microbic invasion.

It has hitherto been customary to look upon certain atrophic pigmented areas in the choroid as conclusive evidence of syphilis, notwithstanding the patient's protestations to the contrary. There are no more valid reasons, I believe, for stoutly asserting that certain solitary or multiple atrophic lesions of the choroid are necessarily syphilitic because of their pigmented structure, than there are for strenuously denying their tuberculous origin for a like reason, and pathological examination is as difficult to obtain in the one case as it is in the other.

The early stages of both are identical in appearance, and the later and final changes are probably quite indistinguishable the one from the other, as no doubt will be proved to be the case at some future date.

If such chronic choroidal changes be associated with chronic tubercle in other parts of the body, or with a
history of past tuberculous infection, and if syphilis can be excluded, surely the weight of the clinical evidence is in favour of their tuberculous origin.

The future will, I think, prove the contentions of Mr. S. Stephenson and myself as to the similarity in appearance of chronic syphilitic and tuberculous choroiditis, and that not only will pigmented atrophic patches in the choroid be regarded as a definite pathological sequence of the active stages of a tuberculous deposit, but that in some cases they possibly denote a limited and exclusive tuberculous invasion.

Advances along this line of research are more likely to be made by recognising tuberculosis as an important and not uncommon factor in the causation of chronic choroiditis than by adhering to the conventional view that such a condition must necessarily be syphilitic, no matter what the evidence may be to the contrary.

Another point of interest displayed by the cases I have related is the occurrence in one of them of unilateral optic neuritis of meningitic type. This is an unusual appearance, which I have observed before in tuberculous meningitis.

These cases are also valuable testimony to the diagnostic worth of a routine use of the ophthalmoscope in the medical practice of children’s diseases.

The use of this instrument often makes it possible to come to a rapid and correct decision in doubtful cases, and renders unnecessary, in some cases at least, the more laborious spinal punctures with leucocyte counts and the application of tuberculin, whether dropped into the eye or inoculated into or injected under the skin, and which takes some time to react. The use of the ophthalmoscope should therefore precede these diagnostic measures.

My conclusions are as follows:

(1) That acute miliary tuberculosis of the choroid can be commonly found by those who take the trouble to look for it in suitable cases.

(2) That the presence of choroidal tubercles by no means invariably indicates the existence of a generalised tuberculous infection.
(3) That fine retinal pigment is a common ophthalmoscopic appearance over the surface of recent tubercles.

(4) That it is not in my experience a fact that miliary tubercles grow perceptibly larger, or that there are in these cases fresh deposits occurring within a few days as claimed by Professor Fuchs in his text-book.

(5) That miliary tubercle is generally situated in the central region of the fundus oculi; as a rule it is of small size, solitary, and is limited to one eye.

(6) That chronic tuberculous choroiditis cannot be distinguished from similar syphilitic choroiditis by ophthalmoscopic examination alone, and that the advocates of the syphilitic origin of this complaint will find it just as difficult to uphold their contention on pathological observations, as it is ours (Mr. S. Stephenson and myself) to prove by the same methods that large, more or less pigmented areas in or about the central region of the fundus, and with or without satellites, are tuberculous.

(7) Lastly, I repeat, in the words of Mr. S. Stephenson and myself writing on this subject in June, 1901, "that tubercle of the choroid may be met with in any form of tuberculosis, whether acute, chronic, or obsolescent."

(May 6th, 1909.)

Dr. F. E. Batten asked in what percentage of cases of tubercle occurring in the hospital with which Dr. Carpenter was connected was he able to find tubercle of the choroid. From an experience at Great Ormond Street Children's Hospital, both clinically and examining eyes pathologically, he was convinced that tubercle of the choroid was not of great frequency of occurrence. Some observations on the point had been made by Dr. Priestley at that hospital, and he found tubercles in the choroid in 7 per cent. of cases dying of tuberculosis. In the cases which he himself examined the proportion was about 10 per cent. It was well recognised that tubercle of the choroid occurred in the cases of acute miliary tuberculosis much more frequently than in other cases. He agreed
with what Dr. Carpenter said about the pigmentation met with around the old chronic tubercle. The author had insisted on the great value of the ophthalmoscope in the diagnosis of tubercle as distinct from syphilis, but he had also stated that it was difficult to distinguish the appearances in the two diseases. Therefore he, Dr. Batten, would have thought that the examination of the cerebro-spinal fluid was of still greater diagnostic value; it was particularly valuable in tuberculous meningitis, for in 70 to 80 per cent. of the cases the tubercle bacilli could be found in that fluid.

Dr. George Carpenter, in reply, said he thought the number of cases in which tubercle of the choroid was found depended on one's energy and experience. He had no recent observations on which to answer Dr. Batten's question concerning the frequency of tubercle of the choroid in cases of tuberculosis. But he referred Dr. Batten to vol. i of the 'Reports of the Society for the Study of Disease in Children,' to a paper read by Mr. Sydney Stephenson and himself at the provincial meeting of that Society held at Liverpool in June, 1900. It was present in 50 per cent. of their cases. But percentages were somewhat unreliable, and in hospital practice cases of all varieties were apt to come in batches. Perhaps the fact that he had seen no less than five cases of tubercle of the choroid during the last five months might be due to some such occurrence. To collect reliable statistics it would be necessary to go through large numbers of cases of acute tuberculosis which had been examined with the ophthalmoscope by a competent observer. He agreed that spinal puncture was a very reliable mode of examination, but it took time and trouble; and the ophthalmoscopic examination was done quickly, and it was especially useful in consultation practice where unerring clinical penetration and prompt diagnosis were always expected of the specialist, no matter how great the difficulties. In children's complaints he had found the use of the ophthalmoscope of the greatest possible service.
53. A case of spring catarrh treated and cured by radium.

By Jas. Mackenzie Davidson and Arnold Lawson.

History of case.—The patient was a well-nourished, healthy boy, aged 12 years, the son of well-to-do parents. I (A. L.) first saw the boy in April, 1905, when he was brought to me with a history of chronic photophobia accompanied by lacrimation and slight conjunctival discharge. This condition of things had been going on for nearly a year. The family doctor had applied the usual remedies for conjunctivitis, but had met with no success, and the boy had been rendered incapable of any prolonged use of his eyes for all close work for some months. The onset of symptoms could not be traced to any exciting cause. The boy at the commencement of the trouble was in good general health, and the latter had not been in any way affected during the past year.

Examination.—Everting the upper lids, both tarsi were seen to be entirely covered with dense, hard, flat-topped excrescences or papules of irregular shape, closely aggregated together and separated by deep, narrow, interpapillary fissures.

The usual smooth level surface of the tarsi was roughened though the general level was but little disturbed.

There was a good deal of reddening and swelling of the conjunctiva itself, and the retrotarsal tissue was very markedly swollen and hypertrophied. A slight viscid discharge was uniformly spread over the conjunctival surface. The bulbar conjunctiva was perfectly healthy, and there was no pannus nor any disturbance of the corneal surface.

The lower conjunctival sacs were quite healthy. Both eyes were affected to about the same degree.

The case, in short, presented the appearance of an extremely severe example of spring catarrh. This dia-
agnosis was later on confirmed by examination of the discharge, which contained the usual eosinophile bodies in large numbers.

The examination of the lids brought on much lacrimation and general pinkiness of the eyes, and these symptoms were always induced when any attempt was made to use the eyes for close work. The irritability of the eyes was otherwise chiefly marked in the morning, when he was usually unable to stand ordinary daylight for a couple of hours after waking.

**Treatment.**— Complete rest from close work was ordered, with dark glasses to be worn out of doors or in bright light, and a trial was at first made of the yellow mercuric oxide ointment of a 4 per cent. strength, which was to be used twice daily.

The refraction was examined, and a hypermetropic error of 2 D. was corrected.

This treatment was a complete failure as it caused too much pain, and a week later he was put on copper sulphate drops, of a strength of one grain to the ounce. With these drops he, for a time, showed very marked improvement in a general way. The photophobia and lacrimation were very much less; the conjunctival redness and the swelling of the follicular tissue had largely disappeared, and the papules certainly seemed smaller and less prominent.

The apparent improvement in the condition of the lid outgrowths was, however, I think, only due to the reduction in the inflammatory condition of the overlying conjunctival epithelium.

The treatment with copper drops was continued, and the strength of the drops increased to two grains to the ounce.

During June and July the boy got on fairly well, not showing any decided alteration in the lid growths themselves, but with his subjective trouble largely controlled by the copper.

In August, four months after his first visit to me, he
was taken to the seaside against my advice, and there the left eye became very bad. On his return the tarsi were inflamed, and the papules in the left were larger and much thicker than before. The boy's general health continued to be excellent.

I did not see the boy again, after his visit on his return from the seaside at the end of August, until the middle of January, 1906. During the remaining months of 1905 he was fairly free from subjective trouble, and used a lotion of bicarbonate of soda with a little opium and hydrocyanic acid, which seemed to give him considerable relief. Weak copper drops, as before, were also used from time to time.

About the commencement of 1906 the left eye became very irritable again, and when I saw him the condition of the left tarsus was, if anything, worse than it had ever been, whilst the right was more or less quiescent.

Nine months had now elapsed, and all that treatment had effected was the partial, though great, relief afforded to the general symptoms of irritation. The right tarsus, though no worse, was certainly no better, whilst the papules on the left tarsus, on the other hand, were very decidedly larger and more dense.

Previous experience has shown the utter futility in these cases of grattage, attempts at expression, etc. The boy had had no education for over a year, and at his age this was a serious matter—the more so because I could hold out no hope to the parents of being able to effect a cure within a reasonable time. Under these circumstances I determined to ask Mr. Mackenzie Davidson if he saw any objection to trying the effects of radium applied directly to the tarsal surface of the lids. Mr. Davidson was of opinion that it could be applied cautiously without any risk. So having told the parents that it was purely experimental, that neither Mr. Davidson nor myself had any experience of radium in this disease, the boy was referred to Mr. Davidson, who undertook the treatment.
We decided to use the radium with extreme caution, commencing only with the left, which was much the worse lid, and not repeating the application until all danger of reactionary inflammation should have disappeared.

The following notes contain Mr. Mackenzie Davidson's précis of the treatment and the results obtained.

The treatment by radium was carried on during the whole of 1906, during which eight sittings were given. The last sitting was on January 9th, 1907, so that roughly the treatment extended over a period of twelve months. Each eye was treated seven times. The appended notes will speak for themselves.

"February 9th, 1906.—Left upper eyelid was everted, and tubes containing 39 mgrm. of radium bromide were held in contact with the granulations for fifteen minutes.

"February 19th.—Left eye improved since treatment. Right (not treated) is worse than it was last visit. Applied tubes with 44 mgrm. of radium bromide to left eye for ten minutes and to right eye for fourteen minutes.

"May 8th.—Right eye much better. Left eye improved a little. Applied to it tubes with 44 mgrm. of radium bromide for fifteen minutes.

"June 6th.—Much better.

"June 25th.—Greatly improved. Applied radium as before to each upper everted eyelid for fifteen minutes.

"July 24th.—Greatly improved.

"October 22nd.—Almost quite well. Granulations nearly gone; no scar. A few remnants treated with three tubes for two minutes at each place—both eyelids.

"January 9th, 1907.—Left eye quite well, with smooth and natural appearance of conjunctiva. Some small granular spots still on the right upper eyelid. Treated with tube of 29 mgrm. of radium applied at each place for five minutes and at one spot for one minute."

During 1906 I kept in touch with the case, seeing the
SPRING CATARRH TREATED AND CURED BY RADIIUM. 243

boy now and then to observe for myself the results, both immediate and remote, of the effects of the radium.

Practically the immediate results were nil, except after one sitting when there was a little redness of the eye with some increase of photophobia and lacrimation for a day or two.

No pain of any kind attended the applications, either at the time or afterwards.

The remote effects were simply that the papules seemed to gradually melt away without any obvious alteration in their contour or appearance whilst so doing.

The improvement in the lessening of the irritability of the eyes began to be manifested within six weeks of the commencement of the radium treatment, so that the boy was able to attend a day-school during the greater part of 1907, and could do his lessons without suffering any inconvenience.

I saw the boy a few days after the eighth radium sitting, and I noted that he was to all appearances quite cured; the lids being quite smooth and all traces of the disease completely vanished. We thereupon decided to suspend treatment of all sorts and simply watch the result.

I last saw the boy on January 12th of this year. He had kept perfectly well for twelve months, and there is not the slightest trace of recurrence. The only noticeable feature about the upper tarsi is that the uniform smooth redness of the normal conjunctiva is replaced by a very irregular coloration. There is nothing in the least suggestive of cicatricial contraction, but over considerable areas the tarsus looks yellowish-white and bloodless, except that here and there these areas are traversed by a few blood-vessels.

We have deferred bringing forward this case until it was absolutely certain that the cure was a genuine and a permanent one. There seems already to be far too many cases of so-called cures by radium in which a sufficient time has not elapsed to warrant the term being used.

It is possible, and, indeed, probable, that in future a
Cure of this disease might be effected far more rapidly than in our own case, but we were hindered in that we had no experience to guide us as to the possibility of reactionary inflammation, and, so far as we could find out, there was no reported case of the treatment of spring catarrh by this method, added to which the case was an exceedingly severe one.

That the treatment was cautiously carried out may be gathered from the fact that the eight sittings were spread over a period of twelve months.

A point of interest is the amount of radium employed. The large dose of 44 mgrm. was applied in this case. I think that this has probably a large and direct bearing on the successful result obtained. I mean that more frequent sittings with a weak dose of radium are not so likely to effect so good a result in the long run as a few sittings in which a large dose is employed; nor will the length of the sitting make up for the weakness of the dose.

I have, during the last twelve months, tried the effect of frequent applications of radium—the dose being less than half the amount employed in our case to-night—in another severe example of spring catarrh. The result has been only partially satisfactory. The papules are much smaller and the subjective symptoms have been relieved, but the case is far from cured. We now propose to submit this case to a far larger quantity of radium, and I believe that an increased rapidity in reaction to treatment, which is in a sense proportionate to the dosage employed, will be observed.

(May 6th, 1909.)

Mr. Mackenzie Davidson said, in answer to the President, that they had applied radium with great success when the eyeball itself was affected. He believed that radium was going to render immense service to ophthalmology. At present it was new, and there were so many exaggerated reports of it that its use had fallen a little into discredit. But he thought many conditions
which were incurable by other means would yield to this, and that even conditions curable by other means would be more rapidly cured by radium. The difficulty was to get enough of it. It was most important to get the right dosage, which was easily measured. Many of the tubes sold, even by reputable firms, did not contain anything like the amount they were said to. Another important matter was the filtering off of the rays not required for the treatment. If rodent ulcer or epithelioma was treated with the rays filtered through thick glass or lead the case could be cured.

54. The abuse of atropin in refraction work.

By R. R. Cruise.

The title I have chosen is sufficiently explicit to introduce the subject matter of the paper I am bringing before you this evening.

It is a difficult task to attempt to dislodge a conviction, especially such a universal ophthalmic conviction as the one in question, namely, the absolute supremacy of atropin as a cycloplegic.

I think I may take it for granted that the only excuse for its supremacy lies in its alleged unqualified efficiency, and therefore that is the point that I wish to free from the overwhelming—I might almost say paralysing—weight of authority. Homatropin and cocaine, in English clinics at any rate, is the almost universal cycloplegic for adolescents and adults—I think I may say almost as universal for those periods of life as atropin is for children, and I class as children all patients up to the age of sixteen.

Of course atropin is frequently used "to ensure complete cycloplegia" in patients over sixteen, but this paper deals entirely with patients below that age whose
range and power of accommodation is at its maximum, and consequently whose resistance to cycloplegics and whose capacity for concealing the total hypermetropia is at its highest. And it is in this class of case that I have made the following investigations to show the equality, if not superiority, of homatropin and cocaine as a cycloplegic for the general clinical purposes of refraction work. My attention was drawn to the subject accidentally; I had homatropised (and in every subsequent use of this term I mean a solution of homatropin and cocaine) a patient with persistent pupillary membrane for purposes of demonstration to some students; on the same case I proceeded to demonstrate retinoscopy and noted the result 4 D. of hypermetropia in each eye. I then ordered atropin gr. iv, ad 3j, and explained that next time I would expect roughly another half to one dioptre of hypermetropia to be manifested.

I was surprised to find the atropin result identical with the homatropin. Since then I have similarly examined 140 eyes, first under homatropin and cocaine and then atropin, and the results of that series of examinations I propose to tabulate and describe. Throughout the series the atropin used was in the form of a 1 per cent. ointment. The homatropin and cocaine was always used in the form of an aqueous solution. In the first seventy eyes the strength was 1 per cent. homatropin and 2 per cent. cocaine, and in the second seventy a 2 per cent. homatropin and a 2 per cent. cocaine. The latter strength, as will be seen presently, is undoubtedly more efficacious, and is the one I invariably make use of in my own work.

The routine procedure was as follows:

(1) The vision taken.
(2) Homatropin and cocaine instilled four times at intervals of fifteen minutes.
(3) Between three-quarters of an hour and an hour from time of first instillation retinoscopy performed, and the highest + glass (or weakest —) which just reversed the shadow noted in the usual way.
(4) Subjective test performed and result registered, if patient appeared capable of reliable answers.

(5) Atropin 1 per cent. prescribed to be used three times a day for three days.

(6) At the next visit the same retinoscopy and subjective test performed and registered, without comparison with the previous examination.

The retinoscopy results are tabulated as follows:

\[
\begin{array}{cccc}
\text{Atropin,} & \text{Homatropin and cocaine, cocaine +} & \text{Homatropin and cocain, cocain +} & \text{Atropin same result.} \\
\text{No. of eyes.} & \text{70} & \text{52} & \text{4} & \text{14} \\
\end{array}
\]

Series I:

<table>
<thead>
<tr>
<th>Homatropin 1 per cent.</th>
<th>70</th>
<th>52</th>
<th>4</th>
<th>14</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cocaine 2 per cent.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Series II:

<table>
<thead>
<tr>
<th>Homatropin 2 per cent.</th>
<th>70</th>
<th>45</th>
<th>22</th>
<th>3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cocaine 2 per cent.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

\(N.B.—+ = \) more hypermetropia or less myopia revealed under.

So that out of 140 eyes examined—necessitating 280 retinoscopies—97 gave identical results.

In twenty-six eyes more H. was revealed under homatropin and cocaine than under atropin, while in seventeen eyes atropin revealed more H. than homatropin and cocaine; but, whereas in the 1 per cent. homatropin solution the proportion was in favour of atropin by 4 to 1, in the 2 per cent. homatropin solution the proportion was in favour of homatropin and cocaine by 22 to 3.

The number of cases in which dissimilar results were arrived at may at first sight appear large, but when it is remembered that:

(1) Each retinoscopy under atropin was registered quite independently of what had previously been noted under homatropin and cocaine;

(2) Every slight degree of difference, such as 0.25 sph.,
was duly allotted to the column giving supremacy to one or other drug;

(3) The maximum difference was 1 D.—an amount which was only observed on three occasions;

(4) That 280 retinoscopies were done; the discrepancies do not materially minimise the value of the results arrived at.

With reference to the three cases where a difference of 1 D. was noted, two showed atropin to be the more efficacious, one homatropin and cocaine. The two first, which had originally been examined under homatropin 1 per cent., I re-tested after an interval of some weeks with homatropin 2 per cent., having first retained their spectacles for a week to allow the ciliary muscle to return to its previous condition of contraction or unrest, and in both cases the second examination gave results identical with the atropin cycloplegia.

In the other case the patient was obviously not thoroughly under atropin.

In twenty cases, representing forty tests, selected on account of their comparative intelligence, in addition to the above examination I measured the distance of the near point, first under homatropin and cocaine and then under atropin. For this purpose I used a flat bar of wood marked off in centimetres, carrying a sliding rider in which was gripped a card of Jaeger type; the butt end of the bar was placed across the bridge of the nose and the rider moved closer and closer to the eyes till the Jaeger 1 type could no longer be distinguished.

A + 2 D. spherical glass placed as close to the eye as possible was added to the far correction to bring the near point within measurable distance, and the results read off. Now it is obvious that should the homatropised eye still have some accommodative power left, it would utilise it under a strong voluntary stimulus and so read the type at a distance nearer to the eye than the atropised eye, in which presumably there was no such power.

In no single instance was this the case, and as a
matter of actual experiment the near point was frequently further away under homatropin and cocaine than under atropin.

To give a concrete instance:

F. F.—, age 44 years, under homatropin and cocaine—

R. and L. c + 6 D. sph. = J. 1 at 21 cm.

under atropin—

R. and L. c + 6 D. sph. = J. 1 at 18 cm.

This series of estimations of the near point affords in my opinion the strongest confirmatory evidence of the efficiency of homatropin and cocaine as a cycloplegic as compared with atropin. The test used, though not of extreme accuracy for determining the absolute near point, is sufficiently delicate for the purpose under consideration, namely, the relative distance of the near point under the two drugs.

Parenthetically, and apart from the subject of the paper, I was surprised to find a constant approximation to the eye of the nearest point at which J. 1 type could be read out of proportion to the strength of the glass used for bringing the near point within measurable distance; for in the instance quoted before—


\[ \text{c + 6 D. sph. = J. 1 at 21 cm. under homatropin and cocaine.} \]
\[ \text{= J. 1 at 18 cm. under atropin.} \]

the effect of a + 2 D. sph. glass ought in an eye with no accommodative power to bring the near point to 50 cm., whereas the actual near point was 21 cm. and 18 cm. under homatropin and cocaine and atropin respectively.

This error was constant in all the cases where the near point was examined. To give a further instance, where two fine parallel lines drawn close to each other were substituted for the Jaeger type on the card:
THE ABUSE OF ATROPIN IN REFRACTION WORK.

F.B. Retinoscopy + 2.75
Homatropin and cocaine:

\[
\begin{align*}
V. & : \bar{c} + 1.74 \text{ S.} = \frac{6}{6} \\
& \bar{c} + 3 \text{ S.} = \text{p.p.} - 36 \text{ cm.;}
\end{align*}
\]

Atropin:

\[
\begin{align*}
V. & : \bar{c} + 1.75 \text{ S.} = \frac{6}{6} \\
& + 3 = \text{p.p.} - 33 \text{ cm.,}
\end{align*}
\]
p.p. being the point at which the two lines blurred into one.

Here the focus of the + 1.25 S. would be at 80 cm., and the patient's p.p. was 36 and 33 cm. under homatropin and cocaine and atropin respectively.

This suggests either that the additional glass, though placed as close to the eyes as possible, still had a value out of proportion to its focal length in dioptres, or—and this seems the more reasonable—that with neither drug is all the accommodative power lost.

As stated before, in no single instance was the near point closer to the eye under homatropin and cocaine than under atropin, but the differences when present were slight, and may readily be accounted for by imperfect application of atropin by the patient's parents.

This last statement leads naturally to the strongest argument in favour of the use of homatropin and cocaine in preference to atropin from the clinical point of view, namely, one visit and the application of homatropin and cocaine by the surgeon yields a paralysis of the ciliary muscle with a certainty not to be attained by ordering atropin for home use. This is especially the case with hospital patients, where the child will come up time after time with its frontal, nasal and malar regions liberally smeared with ointment, and its pupils as delicately reactive as Nature intended. Between this extreme and cycloplegia there must necessarily be all shades of degree militating against the result desired—the estimation of the total refraction of the patient.

It would be the merest waste of your time for me to enumerate the advantages of a cycloplegic such as
The above series of comparisons of the two drugs has, in my mind at any rate, left no further room for doubt on the subject, and if there should be any among you who are not infected with my conviction, the sceptic has only to subject a few cases to the tests above enumerated to share my views on the equal, if not superior, efficacy of homatropin and cocaine over atropin.

Messrs. Lang and Barrett, in the Moorfields reports of 1886, showed "that for ophthalmoscopic and similar purposes the application of homatropin and cocaine to paralyse the sphinciter pupillae and ciliary muscles produced better results than those produced by homatropin alone and in a shorter period," but make no statement as to the variability or certainty of the amount of paralysis produced as compared with atropin.

I have been unable to find any literature making a series of comparisons such as I have done to show the effects of homatropin and cocaine and atropin on the ciliary muscle, and it was with the idea of removing the uncertainty attaching to the use of the former drug that I was induced to carry out a reasonable number of observations on patients without any discrimination as to their respective refraction. Since writing the above I have seen a paper by Alex. Duane, of New York, on a very similar subject and with almost identical conclusions, but whereas at the end of the paper he states: "The cases in which homatropin proves inefficient are few, they are marked by varying vision . . . . in such cases atropin should be used," in the body of the paper he asserts, "but even then," referring to the unsatisfactory cases of changeable
results, "it is not often that the findings with atropin differ from those with homatropin . . . and even with atropin there may be in these cases a residuum of accommodative power left just as there was with homatropin," a statement entirely in accordance with my own experience, but not an argument in favour of any superiority possessed by atropin.

To summarise, may I insist that the sole aim and endeavour of this paper has been, by using atropin as a standard for comparison, to prove the efficacy of homatropin and cocaine where, and where only, it is desired to estimate the total refraction of the patient with the most certain and satisfactory drug or combination of drugs, in our present knowledge, and to establish their reliability on a sound cycloplegic basis.

(May 6th, 1909.)

Mr. Sydney Stephenson said the methods of applying the two drugs did not seem to have been identical. Apparently atropine had been given for home use, whereas Mr. Cruise applied the homatropin himself. That might have introduced a source of fallacy and afforded an explanation of the differences in the results.

Mr. C. Devereux Marshall said that he was speaking to Mr. Lang that morning on the subject of the paper, on which he had done much work. As a result of not only Mr. Lang's own observations but also of what he did with a colleague some years ago, he found that the effect was largely a question of how the mydriatic was put in; he found that homatropin gave certain results after having been instilled two or three times, and that then if one carefully put in atropine one probably found $\frac{1}{2}$ D. more as the result of the atropine. But for practical purposes the results of the two were identical.

Mr. Bishop Harman said he agreed with Mr. Cruise as to the use of homatropin and cocaine. In 1901 he examined 100 eyes, using in comparison on the same patients—(1) homatropin and cocaine 2 per cent., watery solution four times in the hour; (2) homatropin and
cocaïne 2 per cent. of the alkaloids dissolved by heat in castor oil, used only once in the hour; (3) atropine ointment 1 per cent. four times daily for a week, and care was taken to see that it was used.

He found the homatropin and cocaïne was reliable, and that the one instillation of the oily solution was of equal efficiency to the four instillations of the watery solution. His practice in cases of children was to lightly bandage the eyes after the instillation, and only remove it a few minutes before retinoscopy. He used this method constantly in school work. Last year he examined a series of eyes of young adults at fifteen, thirty, forty-five and sixty minutes after the use of the oil; in almost all cases the eyes were ready in thirty minutes. The oily solution had the advantage of being exceedingly economical—a very little went a long way; and it kept good indefinitely.

Mr. Ernest Clarke said the Society should be very grateful to Mr. Cruise for his paper, but it would have been more valuable if he had mentioned the astigmatism in those cases. His own experience was that with a low astigmatism the muscle was found in a state of spasm in young people, which was not present in other conditions; and homatropin was not sufficient to cause complete paralysis in patients under twenty years of age. He had used Burroughs Wellcome’s tabloid “W,” put in, and then repeated half an hour later, in cases of children who were brought at the last moment and had to return the next day. But he only did it under protest, telling the parents or guardians that on the first opportunity they should come up for atropine examination. As a result of twenty years’ experience he could say that atropine was the final “Court of Appeal” on the matter for young people in low cases of astigmatism with headache. The physician sent the case to the ophthalmic surgeon to eliminate the eye condition, and if the latter did not use atropine in young people his report was valueless.

Major Elliott suggested that in hospital practice some
account must be taken of the relative cost of the drugs before atropine could be lightly abandoned.

55. The disappearance of the iris from the pupillary area following injury.

By S. Mayou.

The disappearance of the iris from the pupillary area following trauma, with or without a solution in continuity of the globe, is due to a variety of causes, most of which are well known. The one to which I particularly wish to draw attention is retraction of the iris into the angle of the anterior chamber by the organisation of fibrous tissue on its surface, as I do not think it is generally recognised as a cause of apparent aniridia. Before passing to this it may be of interest to the Society to enumerate the other causes of apparent aniridia.

Incarceration of the iris in a scleral wound apart from operation usually occurs in cases of rupture of the globe, the escaping aqueous or lens carrying the iris into the wound.

Avulsion of the iris.—Occasionally in rupture of the globe the forcible extrusion of the aqueous and lens may carry the iris with it bodily, tearing it completely away from its attachment to the ciliary body. That the forcible expulsion of the aqueous alone is probably sufficient to tear away the iris from its attachment is suggested by a few cases where complete expulsion of the iris has occurred without displacement of the lens.

Recovery with a fair amount of vision after these accidents has been recorded, and it seems more frequently to take place in those cases in which complete avulsion of the iris has occurred; in the cases in which the iris is incarcerated in the wound subsequent cyclitis is more likely to occur on account of the iris
forming a track by which inflammation may spread into the globe, or along which the epithelium from the conjunctiva may spread and so form a fistulous opening.

Retro-flexion of the iris, that is to say the doubling back of the iris on the ciliary body from the pupillary area, is an extremely rare condition and is always associated with forward dislocation of the lens. It is probable that most of the recorded cases are cases of rupture of the ligamentum pectinatum or retraction of the iris, since pathological examination is absent from nearly all the records.

Rupture of the ligamentum pectinatum may also give rise to the disappearance of the iris from the pupillary area. The first case of this condition was shown before this Society in 1892 by Mr. Treacher Collins. Cases have also been recorded by Buchanan and Muller. Most of the cases have ended in enucleation soon after the injury. In the following case the eye was not removed for glaucoma until forty-eight years after the original injury. In it the whole ciliary body, carrying with it the iris, had slipped back on the sclerotic to near the equator of the globe, so that in most situations the pupillary margin of the iris only just reached forward to the corneo-sclerotic junction. The iris and ciliary body showed comparatively little change considering that the anterior ciliary vessels must have ruptured at the time of the injury. The ciliary body had regained a fresh attachment to the sclerotic, which was situated much further back than in most of the recorded cases.

Case.—R. D.—, male, aet. 67 years. Admitted to the Central London Ophthalmic Hospital on October 24th, 1908, under Mr. T. B. Archer, to whom I am indebted for the notes of the case. Forty-eight years ago the patient was hit in the left eye by the catch of a door. During the last fortnight the eye had become painful. On the upper and outer side of the left eye in the ciliary region was the scar of an old wound in the sclerotic. Down-
wards and inwards in the anterior chamber was seen a white mass, apparently the semi-calcareous remains of the lens. In the cornea directly over it there was some opacity which was situated in its deeper layers. There was practically no anterior chamber. The iris on the outer side was pulled over to the inner side, where it was adherent to the calcareous lens. T. + 1. No perception of light. As the eye was painful it was enucleated, hardened in formol and divided in antero-posterior horizontal section. Sections were made in celloidin.

The cornea in the situation of the mass in the anterior chamber showed a gap in Descemet's membrane with some loss of substantia propria, which was probably the result of atrophy due to the prolonged contact with the dislocated calcareous lens.

The white mass in the anterior chamber was seen to be made up of the remains of the calcareous lens with a certain amount of fibrous tissue.

The iris on the outer side was adherent to this mass and in a condition of iris bombé, the anterior surface being closely applied to the posterior surface of the cornea. The iris tissue was stretched, thin and atrophic.

The ciliary body on the outer side was in its normal position. On the inner side the ciliary body had become retracted to near the equator of the globe. It was slightly atrophic; the ciliary processes were present, but short and covered by a layer of fibrous tissue which sent a prolongation forward to the mass in the anterior chamber; the ciliary muscle had largely disappeared but there was a considerable increase in the interstitial fibrous tissue. The iris attached to this portion of the ciliary body was somewhat atrophic, but not nearly so markedly so as on the outer side of the globe where it was bombé; it reached forward so that the pupillary margin was opposite the corneo-sclerotic junction.

The choroid was atrophic, and in the anterior half of the globe showed very marked pigmentation.

The retina was in its normal position, but atrophic.
The disc was cupped.

Retraction of the iris.—This condition is, I think, not usually recognised as a cause of disappearance of the iris after an injury. Apart from injury I have seen it in connection with the late stages of cyclitis, accompanying interstitial keratitis, and in pseudoglioma of metastatic origin. The pathological condition of, at any rate, two of the eyes described below, was somewhat akin to that found in pseudo-glioma. In the three cases recorded the eyes had all been the subject of penetrating injuries, and in two the foreign body was subsequently found within the globe. The injury in each instance had evidently resulted in acute cyclitis, the subsequent organisation of the exudate on the surface of the iris accounting for the condition of retraction found. In two of the cases and probably in the third the lens was partly dislocated, its want of support to the iris probably playing an important part in the disappearance of the iris, since adhesions could not form between the iris and the anterior capsule so that the iris was drawn back into the angle of the anterior chamber. Where the iris was supported by the lens adhesions were present, and the iris had not disappeared although there was a thick mass of fibrous tissue on its surface.

Clinically, the condition may be difficult to distinguish from the other causes of disappearance of the iris, since, in the early stages of all the conditions which lead to apparent aniridia, the anterior chamber may be filled with blood, and therefore difficulty may arise in finding out exactly what has occurred. If the iris does not disappear till some time after the injury one may be practically certain that it is due to this condition of retracted iris. Further, the history of the accident together with the presence of a foreign body in a shrinking globe are all points which help in the diagnosis.

Another point of interest in these cases is that although the angle of the anterior chamber is so completely occluded there is no tension in the eye. This, I think, is
probably due to the opening up of the supra choroidal lymph space by the separation of the choroid in that region associated with the gap in the suspensory ligament, following the dislocation of the lens, allowing a free filtration of the intra-ocular fluid into the anterior ciliary veins.

Case 1. — L. T., male, æt. 3 years.

Admitted into the Central London Ophthalmic Hospital under Mr. W. I. Hancock in May, 1908.

Four months previous to admission his right eye was injured by a flying piece of broken glass. On examination no scar could be seen in the globe. The cornea was clear; no keratitis punctata was present. The lens was clear but seemed dislocated downwards and inwards. Behind the lens could be seen a white mass, on the surface of which a few vessels could be made out. T. — 1.

The iris had completely disappeared with the exception of a small tag present downwards and inwards, which was adherent to the anterior surface of the lens capsule. No ciliary processes could be seen.

On June 5th the eye was enucleated, hardened in formol, and divided in antero-posterior horizontal section.

The cornea was normal.

The anterior chamber was very deep owing to the displacement of the lens backwards and inwards. The angles of the chamber were completely occluded both by the iris and by the organisation of fibrous tissue in the angle of the chamber. This fibrous tissue was more conspicuous in the situation where the iris was not so markedly retracted.

The iris itself was atrophic, retracted and thrown into folds into the angle of the chamber, the retraction evidently being brought about by the organisation of exudate on its anterior surface. It showed a marked ectropion, the sphincter muscle being everted. The pigment-cells from the posterior surface had spread right
Case 1.—Retraction of the iris showing the displaced lens, with the detached cystic retina.

Fig. 50.

Case 1.—Showing the retraction and folding of the iris. The pigment epithelial cells have grown over the anterior surface of the iris up to the posterior surface of the cornea.
up to the anterior surface of the cornea, evidently giving rise to the condition of aniridia when seen clinically.

The ciliary body was somewhat atrophic, the ciliary processes being slightly pulled inwards by the mass of the exudate on its surface and behind the lens.

The lens was displaced downwards and inwards, but was otherwise normal.

The retina was completely detached and showed marked cystic changes.

The choroid, in the anterior part of the eye in the neighbourhood of the supra-choroidal lymph space was detached. Towards the posterior pole of the globe it was in its normal position, but was atrophic. No piece of glass was found within the globe.

Case 2.—W. C—, male, æt. 15 years.
Admitted into the Central London Ophthalmic Hospital under Mr. T. B. Archer on May 25th, 1908.

Four months previous to admission the patient was shot on the right side of the face and body. The right eye escaped but the left was injured.

The condition of the left eye on admission was as follows:

It was evidently a shrinking globe. The anterior chamber was shallow; below there was a small hyphæma.

The iris had completely disappeared above, but below was adherent to the anterior capsule of the lens.

The lens was clear, but its position could not be determined. A whitish mass could be seen behind the lens.

The X-ray photograph showed multiple shot scattered about the orbit, two fragments being localised in the globe.

On May 28th the eye was enucleated and an old wound in the sclerotic was found about the insertion of the external rectus. The eye was hardened in formol and divided in an antero-posterior vertical section.

The cornea was normal.

The lens was displaced downwards and inwards.
Case 2.—Retraction of the iris showing the displacement of the lens and the detached cystic retina.

Fig. 52.

Case 2.—Showing the fibrous tissue on the anterior surface of the iris, where the latter is adherent to the lens capsule.
The angles of the anterior chamber were completely occluded by the iris and by the organisation of fibrous tissue on its surface.

The iris was atrophic. Above, where the apparent aniridia was present, the iris was thrown into folds, and there was some organisation on its outer surface. It showed marked ectropion, and the pigment-cells from the posterior surface had covered the anterior surface up to the cornea. Below, where the iris was adherent to the lens capsule, was a thick, white mass of fibrous tissue.

The ciliary body was atrophic, and the ciliary processes were pulled inwards by the mass of organising lymph on the surface of the ciliary body and behind the lens.

The retina was completely detached and showed marked cystic changes.

The choroid in the anterior part of the globe was detached in the region of the supra-choroidal lymph space. In the posterior part of the globe it was in its normal position, but showed atrophic changes. Two pieces of lead shot were found embedded in the sclerotic near the equator of the globe.

Case 3.—For the notes and sections of a similar case I am indebted to Mr. E. Treacher Collins.

A man, æt. 35 years, was, in November, 1890, admitted to the Royal London Ophthalmic Hospital under Mr. Hulke, having been struck in the right eye by a chip of steel. There was a perforating wound of the sclerotic to the outer side, from which a small piece of steel was removed. Another foreign body was seen in the globe. The cornea was clear, but posterior synechiae were present and there was blood in the vitreous. The eye was subsequently enucleated and a piece of steel found near the optic disc. No note of the condition of the iris before enucleation was made. The sections, which were of the anterior half of the eye only, showed a marked retraction, ectropion, and folding of the iris, with complete occlusion of the angle of the chamber exactly similar to the other two cases which
have already been described. The condition of the lens and retina could not be told as they were not present in the section.

(May 6th, 1909.)

56. A case of bilateral congenital glaucoma treated by iridectomy after the method of Lagrange.

By Arnold Lawson.

The patient, a man, aged 34 years, was first seen in December, 1908. He stated that the left eye had always been weak, but the sight had lately been getting much worse. He had no special complaint with regard to the right eye, which he believed to be sound.

L.V. = Fingers at two feet.

Both eyes are above the average in size, with large corneas and deep anterior chambers. The pupils are somewhat dilated and almost immobile, the left more so than the right. The horizontal median line of each cornea is traversed by a faint, deep grey striping or band of the character not uncommonly seen in buphthalmic eyes. The intra-ocular tension is very high (T. +2 or more) in the left, and also much increased, though to a lesser degree, in the right. Both discs exhibit typical glaucoma cupping, which in the case of the left is very deep and accompanied by considerable venous engorgement.

The right visual field shows considerable contraction over the inner, upper, and outer segments. The left field is reduced to a minute area about two degrees in diameter round the fixation point.

There are, and have been, no marked subjective symptoms of any sort.

It was obvious that the right eye was slowly following the lead of the left, and that blindness would in course of
time result if nothing radical was done. Eserine, though contracting both pupils, had no effect on the tension. Iridectomy by the usual method did not seem to offer, in this instance, the smallest chance of success. The most favourable procedure appeared to be either sclerotomy or iridectomy after the method of Lagrange. Cyclo-dialysis was rejected as the known results of this operation are few and its value doubtful. It was elected to try the effect of Lagrange's operation on the left—the worse eye, and the operation was performed on December 14th, 1901. The healing was uncomplicated, except that there was some swelling about the wound for the first ten days. Firm flat healing was in time obtained, the patient being kept in bed for nearly a fortnight. The intra-ocular tension two months after operation was still above normal, though less than before. It has since been constantly falling in a gradual manner, and is now normal. The right eye

Fig. 53.

RIGHT

Field for white, 10 mm. disc, six weeks after iridectomy.
was treated in the same way eight weeks later, and the healing was equally easy and uncomplicated. The intraocular tension in this eye has also gradually come down and is now normal.

Comparison between the fields taken within the last few days and those before the operation shows an apparent gain in the right eye over the nasal segment, whilst that on the left side has also apparently increased to a slight extent. With correcting glasses R.V. = \( \frac{6}{18} \) full; L.V. = \( \frac{2}{60} \).

Patient has been using weak eserine (\( \frac{1}{4} \) per cent.) since the operations, but this will now be omitted. He states that so far as the left eye is concerned it feels much stronger, and he has lost the sensation of weakness and discomfort which brought him in the first instance to seek advice. 

(Card specimen. June 10th, 1909.)
Dr. Weeks (Philadelphia) said the result in this case was excellent at the present time. He had himself not been impressed with the reports of Lagrange in regard to the results of the operation; he had hoped an operation to produce permanent lowering of the tension had been devised. He, Dr. Weeks, had done thirty or forty operations of the kind, but his anticipations had not been entirely realised. The operation enabled one to do an excellent iridectomy, to retard healing of the wound, and maintain filtration for some time, but eventually cicatrisation took place, connective tissue filled the gap, and at the end of three to nine months there was a cicatrix very much like that obtained after ordinary iridectomy. Many attempts had been made to produce a filtering scar, and in that connection the work of Major Herbert was known to most of them. He thought one could not expect to produce a filtration scar in operations which did not enclose something between the lips of the fibrous coat, and that had been attempted by inclusion of a piece of iris, or turning in a conjunctival flap. In Mr. Lawson's case he thought the reduction of tension was due in some measure to incarceration of the iris; in the left eye there was incarceration of the iris at the angle, though small in degree. It seemed possible that a recently devised operation, in which there was subconjunctival incarceration of iris, might produce a filtration scar. He did not propose to follow the operation of Lagrange at the present time.

Mr. Brooksbank James said he had not done Lagrange's operation, but he had recently operated upon several cases by turning down a large flap of conjunctiva and making an incision from without, 1 mm. from the limbus. He thought there were some advantages in doing that, especially in chronic glaucoma, where the iris was adherent to the back of the cornea. It prolapsed readily into the wound. The escape of the aqueous could be exactly regulated, and one could remove as much iris as required and as much sclera, the latter being more easily
removed by a punch than with scissors. He thought that a number of cases done by that method would give a better notion of the actual benefit derived from a sclerectomy, and then one would learn how large a section in the sclera to make, and whether a large or a small iridectomy was necessary. There seemed to be much difference of opinion as to whether the filtration subsequent to the operation went on through the iris, or whether a filtration cicatrix was formed. He had done six cases so far, and four were cases of glaucoma simplex, in one of which the field was contracted nearly to fixation point, and the vision was at present \( \frac{6}{18} \) in each eye since the operation. Very slight astigmatism had been produced, and he thought that the amount of astigmatism produced by a large section by the ordinary method was a point to consider. Moreover, if it were desired to draw a small piece of iris tissue into the angle of the wound the large conjunctival flap was a great safeguard.

Mr. Treacher Collins said he had never performed Lagrange's operation, for theoretical reasons; the object of the operation seemed to be a faulty one. He agreed with Dr. Weeks that it was impossible to produce a real filtration scar unless there was an entanglement of a fold of iris which lined the two edges of the wound, so forming a gap in the wall of the globe through which the aqueous could subsequently escape. In Lagrange's operation half the thickness of the sclerotic was only removed, and that would not allow the formation of a fistula in the cicatrix. He thought it was not a desirable thing in most cases of glaucoma to try to produce a filtration scar, i.e. one in which a piece of the iris was incarcerated in the wound and was adherent to the conjunctiva. It was dangerous to do so, because with the iris left adherent to the conjunctiva any inflammation of the conjunctiva which might arise was very likely to spread to the iris and cause intra-ocular inflammation. In eyes with cystoid cicatrices which contracted purulent ophthalmia, suppurative pan-ophthalmitis had
arisen from infection passing into the eye through the filtration scar. If suppurative inflammation could extend in that way through a cystoid scar, it was also possible that an inflammation capable of causing sympathetic ophthalmitis in the other eye might gain entrance in a similar way. He did not think they should regard as an ideal operation for glaucoma one which caused a cystoid scar, but rather aim at opening up the normal channels of filtration. It seemed to him that the operation of cyclodialysis, which had recently been introduced, would prove to be founded on a better principle than Lagrange’s operation.

Mr. Odillo Maher said that for the past ten or twelve years he had been in the habit of performing an operation for chronic glaucoma which gave a small and satisfactory cystoid cicatrix. He operated by making the usual section, drawing out the iris, cutting it at one angle of the wound, detaching it from its base corresponding to the section by dragging on it with the iris forceps, and finally leaving prolapsed at the other angle of the wound a tag of iris several millimetres in length. A week later he cut off with a scissors the prolapsed iris level with the surface of the sclerotic, leaving an entanglement of iris with a cystoid cicatrix. One case was lost from irido-cyclitis following the operation, but all the other cases which he had been able to trace had been much more satisfactory than those in which an ordinary iridectomy had been done. With regard to losing the other eye from sympathetic ophthalmia and also the operated eye from infection one recognised that these were dangers, but his experience was that such misfortunes were few and far between, and he had caused many cystoid cicatrices with entanglement of the iris. He thought that the benefits derived from such a cicatrix quite outweighed the risks of sympathetic ophthalmia and septic infection. In the early stage of chronic glaucoma before adhesions had taken place at the filtration angle it was better to do an ordinary iridectomy, but cases were
seldom seen sufficiently early. When, however, the
disease had lasted for some time, and it was no longer
possible to re-open the filtration angle, the aim should be
to produce a filtering cicatrix. Many of his cases he
had seen years afterwards with normal or slightly sub-
normal tension and good vision.

Mr. Cross said one could not wish for a better result
than Mr. Lawson had shown in this case. He agreed
that if a leaking scar could be obtained and the leakage
was not too great that was the ideal operation, espe-
cially in a simple case of glaucoma. There might be good
reasons for having a prolapsing iris; as a rule it should
be avoided, but he agreed with the last speaker that the
result in many cases of simple glaucoma was so disastrous
that one was justified in running the risk of a prolapsing
iris. He had no particular confidence in any special
forms of procedure. In his own earlier cases the filtra-
tion scars had been to some extent a matter of luck
rather than of preconceived ideas. The new departure
by removing a piece of sclerotic and aiming more defi-
nitely at a filtration scar was the right way to attack
such cases. He did not favour leaving a prolapsed iris
if it could be avoided.

57. A case of unusual cataracts, bilateral, in a girl.

By J. Herbert Fisher.

A. M. S—, female, aet. 12 years when first seen in
October, 1907. Is the middle child of a family of five;
one of the other four are defective in sight as far as the
parents know.

Under atropin, after retinoscopy, the right eye took
\(-0.5\) D. sph,
\(-2.75\) D. cyl. \(-50^\circ\) and saw \(\frac{5}{2}\) partly; the left eye \(\tilde{c} -
3.0\) D. cyl. \(35^\circ\), saw \(\frac{5}{2}\), two letters. Each lens showed a
CASE OF UNUSUAL CATARACTS.

circular dot of opacity at the posterior pole, with a little want of general clearness, and about six radiating cortical striae at symmetrical intervals.

When seen again a year later rather higher cylinders were chosen, and the best vision obtained was $\frac{6}{24}$ badly in the right eye and $\frac{6}{18}$ in the left eye. There were no lamellar teeth. Apart from the cortical striae the more posterior changes were not unlike the discoid type of cataract.

It is evident that a progressive development of the lens opacities has taken place. At her second visit the discovery had been made that, in school, on closing the left eye she could not see the blackboard with her right; the acuity had been practically equal in the two eyes when she first came under observation. When seen in May of this year the report was that she had been able to read before Christmas, 1908, but not since, and school lessons had had to be suspended entirely. The right eye now gets, with correction, $\frac{6}{24}$, 2 letters, the left not $\frac{6}{60}$ with any glasses. At the present time there appears in each lens a boat-shaped or elliptical shell of opacity, lying somewhat obliquely, with the long axis approximating to the vertical; the free margin is sharply cut as a hard line. The cortical striae alluded to originally are still present, and while not showing the open mouths are strongly suggestive of the trumpet-shaped opacities seen in coralliform cataracts; they appear slightly whorled or moniliform. The convex side of the boat rests on the posterior pole of each lens, and is most dense at this point, where some glistening reflecting particles are suggestive of crystals; from this disc in the left lens some opacity can be faintly seen coming forwards in the axis of the lens as if a spindle might be expected to develop.

I am inclined to regard the cataracts as a variety of the coralliform type. I have examined her father's lenses; they are quite normal.

(Card specimen. June 10th, 1909.)
58. *Bilateral ptosis and ophthalmoplegia externa.*

By Walter H. Jessop.

Norah H—, aet. 5 years, was admitted into St. Bartholomew's Hospital on April 28th, 1909. The mother says the eyes were perfectly natural till six weeks ago, and this statement is corroborated by a photograph. On March 15th the mother noticed drooping of the right upper lid and external squint; on March 21st she noted for the first time drooping of the left upper lid.

*Past history.*—The child has had no previous illness; the tonsils and adenoids were removed a year ago.

*Family history.*—The maternal grandfather died of phthisis. The mother has lost two daughters of wasting disease, one at thirteen months old and the other at eleven months.

*Present condition.*—The child is a healthy-looking child with adenoid facies. No enlarged lymphatic glands. The tongue is protruded straight; the soft palate moves on phonation; pharynx natural; larynx normal.

*Eyes.*—There is complete paralysis of both levator palpebrae muscles; the palpebral fissures are kept slightly open by tonic contraction of the occipito-frontalis, and the head is thrown backwards. The left palpebral fissure is more open than the right. In the right eye there is an external strabismus of 5°; the movements of the eyeball are greatly limited upwards, downwards and inwards.

*Left eye.*—The movements of the eyeball are slightly defective upwards and outwards.

The pupils are equal and react normally; the fundi are normal. Vision difficult to estimate, but apparently normal. Patient was examined by Dr. Ormerod, who thought it might possibly be a tumour in the region of the corpora quadrigemina.
May 10th.—Von Pirquet’s skin reaction gave positive result. A control being made with normal saline solution, injection of tuberculin T.R. 0002 mgm. was given.

May 18th.—Ptosis the same; no movement can be obtained in either eye.

May 25th.—Right upper lid seems to be on slightly higher level. No movements of the eyeball can be obtained; the pupils equal and act normally.

June 4th.—Left eye—slight circum-corneal zone. Small whitish corneal ulcer, about size of pin’s head, in lower part of cornea near centre, which stains with fluoresceine.

June 10th.—She has had altogether three injections of tuberculin up to 0008 mgm.

*Present condition.*—No external movements of eyeballs.

Right upper eyelid has some movement in it, and has been more raised for the last three days.

Left upper eyelid the same.

Left eye.—Ulcer has healed; pupil dilated by atropin.

The temperature has never been above normal.

*Card specimen. June 10th, 1909.*

Dr. F. E. Battex asked whether the onset in Mr. Jessop’s case was acute or gradual; that would make an essential difference to the diagnosis, and also as to the prognosis. If the onset was an acute one, it would seem in favour of the condition being a polio-encephalitis which had picked out the cranial nuclei. It was almost certain that not only was the third nucleus affected, but also the sixth and seventh, so that the lesion had been a fairly extensive one. A gradual onset, he thought, would be much in favour of a growth, and the partial recovery which had taken place was occasionally met with in intra-cranial growths, especially tuberculous ones. Without the history and other facts it was very difficult to say what the nature of the case was; but the fact that the third, sixth, seventh and fourth nuclei were all affected made it probable that there was a tuberculous condition affecting the corpora quadrigemina, and extending back-
wards to involve the sixth and seventh. He had recently had under care an exactly similar case in a child, who developed an external squint with ptosis, and finally complete paralysis of the muscles supplied by the third nerve with right hemiplegia. The condition was diagnosed as tuberculosis on the ground that the child gave a definite von Pirquet reaction, the spinal fluid had definite lymphocytosis, and tubercle bacilli were present in the cerebro-spinal fluid. The autopsy showed a tuberculous tumour in the region of the corpora quadrigemina. He suggested that the present case was either a tuberculous growth, or possibly polio-encephalitis, but the diagnosis depended on the mode of onset and the nature of the cerebro-spinal fluid.

Mr. Jessop, in reply, said that Dr. Ormerod, who saw the case, thought it might be a tumour in the region of the corpora quadrigemina, and the von Pirquet reaction was obtained. Three months ago the patient had the photograph taken, and the condition began in one eye five weeks ago. The onset all happened in five days, and it gradually spread from the different muscles, starting in the right and gradually working to the left. In ten days every muscle was paralysed.

Note.—June 14th: Absence of corneal reflex and no reflex reaction of the orbicularis palpebrarum muscles.

The child looks ill, and on two occasions she has had attacks of choking and cyanosis; there is a marked nasal tone to her speech, which is very indistinct.

Examination by Mr. Harmer.—Soft palate moves with phonation, but not vigorously. Pharynx: food remains in lower part; on swallowing fluid patient choked feebly and in an abnormal manner, suggesting that the sensation of the opening of larynx (internal laryngeal) is defective.

Whether there is paralysis of the pharyngeal muscles is uncertain. No adenoids. Examination of larynx impossible without general anaesthesia.

Dr. Lewis Jones's report.—The orbicularis palpebrarum
reacts normally but weakly to induction coil currents on both sides; to continuous current the response is quite quick and not sluggish.

The other face muscles respond similarly. The levator palpebrae was examined specially for response; something like it was observed in the left eye only. This muscle is said to show a visible contraction to testing when in a state of R. D. only.

June 16th.—The child was taken out of the Hospital, and the same night had a fit of dyspnoea and was seen by Dr. Francis.

June 17th.—Dr. Francis was called to see the child and found her dead. Post-mortem examination was refused.

59. (1) A family with congenital displacement of lenses;
   (2) A family with congenital opacities of lenses.

By P. H. Adams.

Numerous instances of families with congenital dislocation of the lens running through several generations have been recorded, notably by Morton (1), Dixon (2), Mules (3), Tiffany and Miles (4). The family that I now record is one of a mother and nine children, seven of whom are affected. I could obtain no history of any other of their relations suffering in the same way. The explanation given of its occurrence in the mother was that her mother fell down a flight of stairs the night before her child was born.

The eldest boy was seen in 1896 by Mr. Doyne, who noted that the right lens was displaced downwards, leaving the upper third of the pupil clear, whilst in the left eye it was completely displaced, no lens being visible in the pupil at all. Correcting glasses, R. +12, L. +10, were ordered.
Second child, a girl, æt. 16 years.  
The right lens inwards, left lens up and inwards.  
She was wearing convex lenses obtained elsewhere.  
Third child a girl, æt. 14 years.  Lenses in the normal position.  Refraction normal.  
Fourth child, a girl, æt. 12 years.  Both lenses downwards, the left one a little inwards as well.  She was highly myopic.  V.: R. - 20 D. sph. = < 6/60; L. - 20 D. sph. = < 6/36.  
Fifth child, a girl, aged 11 years, with lenses in normal position.  Refraction normal.  
Sixth child, a girl, æt. 10 years.  Both lenses inwards.  V.: R. + 7 D. sph. = 6/36 < 6/24; L. + 8 D. sph. = > 6/18.  
Seventh child, a boy, æt. 8 years.  Right lens upwards, left up and out.  V.: R. - 20 D. sph. = < 6/60; L. - 20 D. sph. = 6/60.  
Eighth child, a girl, æt. 3 years.  Right lens downwards, left inwards.  
Ninth child, a girl, æt. 17 months.  Both lenses upwards.  Numbers 3 and 5 were certainly brighter and more intelligent-looking children and obviously different from the others, who were all very much alike, but one could not make out that they resembled the father more than the others.  The interesting point about this series of cases lies in the unusual situation of the displaced lens.  It is usually stated that in congenital dislocation of the lens, the lens is displaced in an upward direction, rarely if ever downwards, the deficiency in the suspensory ligament normally occurring opposite to the foetal cleft, while in three of these cases the lenses are definitely displaced downwards, and in the eldest boy the lens of the left eye is completely dislocated downwards.  All of them, of course, showed tremulousness of the iris, and in none of them could one get the pupils more than semi-dilated, even after atropin freely used for a week.  The fundus was normal in all the cases and there were no other congenital abnormalities.
(2) The second family is an example of hereditary stellar cataract. The opacities outlined the sectors of the lens on the posterior surface, and in some cases the anterior surface also, though to a much less extent. The eldest member that I examined was the great-grandfather, aet. 83 years. In him the changes had progressed chiefly in the anterior part of the lens, the opacity taking a rather tri-lobed shape, as though it had spread from an original Y-shaped mark. One could see numerous fine spokes at the periphery of the lens, which was comparatively clear; these were situated on the posterior surface. He gave a definite history of his father and his two sisters having always had similar defective vision as himself. I did not see his daughter, but her sight has also been always defective. Her two children were both affected. The son was seen by Mr. Doyne in 1892, when he showed Y-shaped markings on the anterior surface of both lenses and three primary rays, each branching into two on the posterior surface. I saw him again last week and he
now has fairly dense opacities in the front of both lenses, chiefly three small circular opacities, and also numerous rays on the posterior surface, but it was not possible to make out their definite arrangement. Of the children the eldest girl shows the posterior lines very sharply marked, seven rays in the right eye and six primary ones in the left eye, with some secondary branches. She had myopic astigmatism: R. - 6 D. sph. - 4 D. cyl. ax. horiz.
The second child has perfectly normal eyes with a low degree of hypermetropia.

The third, a boy, aet. 5 years, in addition to posterior markings had very faint Y-shaped marks on the anterior surface. He had about 2 D. of hypermetropic astigmatism.

The fourth, a girl, aet. 3½ years, showed four primary rays in the right eye, branching at the end of two of them, and three in the left eye, almost immediately dividing into two. The youngest child, aet. 7 months, showed an inverted Y on the posterior surface just branching into two at the end of the limbs with nothing visible on the front of the lens.

In none of them were the lenses perfectly transparent; they seemed to have a faintly dotted appearance and the fundus looked blurred, but normal.

The lines themselves were as if they had been drawn with a fine BBB pencil on rather rough paper, but were very sharply defined in the children. In the father, who is now aet. 36 years, the posterior lines appeared slightly broader and not so sharply marked.

The condition appears to be very slowly progressive, the opacity increasing most in the anterior part of the lens. It is rather remarkable that the great-grandfather, aet. 83 years, was able to read large print up to about three years ago, though he had never found any glasses to help him.

As to the situation of the opacity Hasner placed it in the interfibrillar material. Tweedy (5), however, who described the intersectional lines visible in the normal lens many years ago, placed the opacity at the ends of the lens-fibres themselves. Zirm (6) described a somewhat similar family to this present one.

References.

(2) Ibid., vol. i, p. 54.
(3) Ophthalmic Review, 1883.
60. A case of massive exudation of the retina with (?) arterio-venous communication.

By A. Hill Griffith and A. W. Ormond.

(With Plates XII and XIII.)

A. J—, female, aged 31 years, came to the out-patient department of the Royal Eye Hospital on May 5th, 1906, having been sent by Dr. Cummin Air, of Norwood.

The patient, a small, thin woman with a frail, anxious appearance, speaks in a tired, uncertain manner; she is deaf and has been so since she was nine years old. Her left eye had been excised ten years previously by Dr. Hill Griffith, of Manchester.

She complained of pain and failing sight in her right eye, also that she constantly saw "specks" floating in front of her. As she only had the one eye she feared lest she should go blind, and her constant anxiety may in some degree account for her frail condition. On examination her vision was found to be 6/9, and her manifest hypermetropia equal to 3 D. On ophthalmoscopic examination it was noticed that the optic disc was very indistinct at its inner margin, and gave the appearance of being inflamed; the retinal veins were very much dilated and tortuous, and where an artery crossed over a vein it appeared to divide it. The appearance of the fundus has undergone very little change since I (A. W. O.) first saw her; there were no floating vitreous opacities, but a single thread of transparent material could be seen coming from the region
of the optic disc to within a short distance of the back of
the lens, simulating a persistent hyaloid artery; the rest
of the media was quite clear. The optic disc had a very
indistinct inner margin and was covered by a thin film of
connective tissue, but there was no swelling. The upper
branch of the central retinal vein divided above the disc
into superior nasal and temporal veins; both of these were
very large, tortuous, and pale in colour; the superior
nasal vein could not be traced to the periphery of the
fundus, but as far as it could be seen it remained un-
altered. On the other hand, the superior temporal vein
could be traced upwards and outwards, and ended in a
large, white, waxy-looking mass, only the edge of which
could be seen. The arteries showed marked changes;
several were mammilliform, especially one passing directly
inwards from the optic disc. The superior temporal
artery was enlarged and tortuous and could be seen to
enter the mass in the extreme temporal part of the field.
There were also in the retina one or two small patches of
exudation situated beneath the retinal vessels. The in-
ferior temporal and nasal vessels show some enlargement
and tortuosity near the optic disc, but are quite normal
when seen in the extreme periphery (Pl. XII).

I wrote to Dr. Hill Griffith, who removed this patient's
left eye in 1896, and in his answer he referred me to
vol. iii of Norris and Oliver's System, and he said: "I have
the eye, also her sister's, and both are cases of the same
sort of fibro-plastic choroidal exudation with increased
tension."

The patient continued to attend my out-patients until
the end of June, 1906, and seemed to improve in general
health under tonic treatment; after that I did not see
her until February, 1907, when I made the following note:
"The patient says that on February 2nd, in the afternoon,
she noticed what looked like a cobweb, which broke and
separated into a number of pieces; some part of it looked
like smoke of a brownish-green colour; the piece persisted
and continually moved about, and there was a veil of
PLATE XII.

Illustrates Mr. A. Hill Griffith and Mr. A. W. Ormond's case of Massive Exudation of the Retina with (?) Arterio-venous Communication (p. 279).
spots which interfered with vision, but through which she could see."

On examining her I found that her vision at that time was $\frac{10}{12}$, and there were extensive vitreous opacities, but beyond that not much change.

The patient was given iodide of potassium and she continued to come and see me during 1907; throughout that time there was no marked alteration in her condition.

In order to thoroughly examine her medical condition, and if possible to ascertain the aetiological factor present in this case, I took her into the ward at Guy's Hospital.

She was examined on several occasions in order to ascertain if her medical condition were sound, and the heart, lungs, kidneys, etc., were found to be quite healthy; her ears and nose were also examined, but nothing was discovered to account for the ocular condition. Her opsonic index was normal and she did not react to Koch's tuberculin test. The field of vision was full, and nothing was discovered by transilluminating the eye.

Beyond recognising that the changes in the fundus of the right eye were possibly due in some way to vascular disease, I was unable to classify this case, but after hearing Mr. Coats' account of similar cases, which he described in a paper read at Oxford last year, it was possible to account for some points which had been obscure before.

The patient went home quite early in the present year, but she has continued to come and see me regularly at my out-patients since, and there has been no noteworthy change in her condition. I have examined the eyes of one brother and one sister, but found them quite normal, also the remaining eye of the sister operated on by Dr. Hill Griffith, which is healthy.

The following are the notes made by Dr. Hill Griffith in September, 1896:

The patient is æt. 22 years, and is married. Vision of the R. eye, tension normal, fundus normal. L. eye, no P.L.; tension plus, slight injection of the globe; anterior
chamber shallow; pupils slightly dilated, round, no adhesions; reacts consensually to light, but not directly. Lens clear; no red reflex; retina can be seen applied closely to posterior surface of lens. The retina and its apparently normal vessels are easily seen by oblique illumination; show sulci in form of a Y-shaped figure; the pupil shows a pigment ring as one gets in glaucoma, but it is also present in the other eye; there is a history of general loss of sight for four years with latterly some pain; the eye was removed as it was feared that an intra-ocular growth was present.

Examination of eyeball after removal.—Antero-posterior measurement and also transverse is 23 mm., the vertical 22.5 mm.; cornea, transverse, 12 mm., vertical 11 mm.; optic-nerve cut 11 mm. long. Total detachment of retina from disc to back of lens; anterior part of retina is roughened by presence of little nodules on its outer surface. A fine strand of yellow lymph stretches from a triangular elevation on the choroid close to the position of the macula lutea, going from this to the stalk of the detached retina. The space between the choroid and retina is entirely filled by a straw-coloured glue-like mass with a quantity of fine flocculent brown or rusty-like material, chiefly aggregated along and adherent to the choroid.

Dr. Hill Griffith sent me (A. W. O.) some microscopical sections which were taken from a horizontal slab and which show the following points. The triangular elevation on the choroid, from the apex of which stretches a fine strand of yellow lymph, shows the choroid passing entirely below it. The choroid cannot be said to be absolutely distinct from it, because in some of the sections there is undoubtedly no mark of demarcation between the choroid and the mass, but on the other hand some sections appear to show an entire separation between the two. Large spaces filled with blood can be seen in its periphery superficial to the choroid (Pl. XIII, fig. 1, e). "Ghost" cells surround the elevation where it is in contact with the exudation;
PLATE XIII.

Illustrates Mr. A. Hill Griffith and Mr. A. W. Ormond's case of Massive Exudation of Retina, with Arterio-venous Communication (p. 279).

Fig. 1.—A. Sub-retinal exudation.
  b. Small-celled inflammatory mass with ghost-cells at its edge.
  c. Fibrous capsule.
  d. Cavity filled with calcareous débris.
  e. Hæmorrhage.
deep to this is a small-celled inflammatory mass, (Pl. XIII, fig. 1, v) and it is a prolongation of this that stretches forward to the retina and macroscopically was described as a small strand of yellow lymph. In the centre of the elevation there is a cavity (Pl. XIII, fig. 1, v) partially filled with calceareous débris, and surrounding it a fibrous capsule (Pl. XIII, 1, c) with numerous cholesterin spaces present in it. There are no giant cells to be seen.

Notes on the condition of A. J—'s sister:

J. W—, æt. 14 years and 11 months, had lost the sight of the left eye certainly for six months, and probably it had been failing for about eighteen months. At the date of enucleation there was no perception of light; tension + 3, cornea hazy, pupil dilated ad max.; only a very narrow rim of iris to be seen covered with pigment on its anterior surface. There was total, or nearly total separation of the retina, forming a funnel-shaped cavity extending from the disc to the ciliary attachment.

The detached retina was thickened and discoloured, and did not wave about; splashes of blood were seen in places.

During the few days the case was under observation the haziness of the media increased, and the diagnosis of intra-ocular growth having been arrived at the eye was removed.

The globe was divided into an upper and a lower half. The sagittal diameter measures 25 mm., the transverse 24·5 mm. The angle of the anterior chamber is closed by advance of the root of the iris, otherwise the anterior parts of the eye are normal.

The detached retina appears as a rounded cord running straight forward from the optic disc. It is evidently much thickened, especially at its posterior attachment, where it forms a pinkish-grey fusiform swelling, whose base is closely adherent to the choroid at the temporal side of the disc.

Longitudinal sections of the optic nerve, with the retina and other parts of the back of the eye, show the follow-
ing points: The optic nerve is normal, or practically normal; the choroid is normal except at the temporal side of the disc, where it is much thickened and engorged with blood, and shows embedded in its substance several plates of young bone. Its structure is here quite continuous with the fusiform swelling formed by the detached retina.

In the posterior part of the swelling one can readily enough make out the thickened nerve-fibre layer of the opposed halves of the retina, and trace these backwards to the lamina cribrosa, which is distinctly depressed. The other layers of the retina cannot be made out separately, but are all much proliferated and contain a good number of blood-vessels and a moderate amount of pigment, which, being confined to the parts adjacent to the choroid, is evidently derived from the hexagonal pigment-cells. The case was thought at the time of excision to be one of new growth, but afterwards to be a localised chronic inflammation of the choroid and retina.

We have here three eyes to consider—two from a pathological standpoint and one from a clinical, the three being obtained from two sisters. The clinical aspect of A. J—'s right eye is that of a chronic progressive change affecting the retina. There is nothing suggesting choroiditis. The marked enlargement of the retinal vessels, the similarity in colour of the arteries and veins, their tortuosity and the white en-sheathing, as well as the varicosity of at least two vessels, point to their being pathologically affected. The mass in the extreme upper and outer periphery from which an enlarged artery and vein can be traced is deep to the retinal vessels, but is non-pigmented and is raised above the retinal level; the two large vessels running from it can be differentiated, but in colour and size they are less easily distinguished than a normal artery or vein would be, and they probably communicate within the mass. In addition there are one or two small white areas of exudation elsewhere in the fundus, and they are also beneath the vessels.
This clinical picture is similar to Group 3 of Mr. Coats' collection of cases published in the Moorfields Reports, vol. xvii, Part III, and can be best explained on the hypothesis that the mass is due to an old haemorrhage in the outer layers of the retina, which has since organised and has opened up a communication between an arterial and a venous channel.

The two pathological specimens, on the other hand, were both excised in a glaucomatous condition, and were not examined clinically until the detachment of the retina was complete, and then they appeared to be cases of secondary glaucoma due to intra-ocular growth. The detachment, however, in each case was due to a fluid, not a solid source, and the cause of this rapid and extensive exudation is not clear, but might be due to a "metastasis of a little virulent organism in the capillaries of the outer reticular layer," that is to say, to a chronic exudative choroido-retinitis. Between the choroid and retina, however, in both cases there was a mass which was undoubtedly due to an organising haemorrhage, but if a localised haemorrhage alone was the primary defect it is difficult to explain the extensive exudation and the secondary changes in the eye. May not all the conditions be explained better by the assumption that we are dealing with a general disease of the terminal branches of the retinal vessels supplying the outer reticular layer and that the ophthalmoscopic changes, massive exudation, vascular dilatation, etc., are results secondary to long-continued capillary disease?

(June 10th, 1909.)

Mr. G. Coats said that the case was a very illustrative example of a little known disease, of which the first case was reported by Fuchs in 1882. Among the characteristic features which the present case showed were the good general health of the patient, the bilaterality, the extremely chronic course, and the final loss of one eye. He had recently collected examples of this and allied forms
of disease, and added some pathological examinations. Among his pathological material, however, there was no instance belonging to the present group—that with arterio-venous communication. This group he had been inclined to separate, at least in many respects, from other cases of retinal disease with massive exudation. He was therefore extremely interested when Mr. Ormond showed him first the case itself, and afterwards sections from the other eye. The sections dated from a long time back, when practically nothing was known of the disease, and unfortunately they did not show well the most important point of all, namely, the state of the peripheral retinal vessels in the region of their communication. Apart from that, however, the fibrous mass between the retina and choroid, with calcification of the centre and cholesterin crystals, closely resembled the changes found in the cases which he had examined pathologically. This might indicate that the groups with and without arterio-venous communication were more nearly allied than he had supposed, but he would express himself with caution on that point till there were more pathological data.

Mr. Treacher Collins said the Society was very fortunate in having another example of this rare disease brought before it again so soon. Only two meetings ago Mr. Wood, of Cape Town, submitted a paper on this subject. In the first case shown before the Society by Mr. Wood some years ago, he, Mr. Collins, had the opportunity of making a pathological examination of the eye, and also of the two eyes of the sister of that patient, who had the same condition. In both cases he had found an angioid growth from the retinal vessels, which had resulted in detachment of retina and the development of glaucoma. The mass of exudation on the outer surface of the choroid in Mr. Ormond’s case he thought might well be explained as the result of haemorrhage secondary to an angioid growth. It was interesting that Mr. Ormond should have found the disease in two members of the same family, as Mr. Wood and he had done.
Mr. Ormond, in reply, said it was true that the specimens were made ten years ago. He could not show a specimen of the sister's eye, as it had undergone some change during the time, and it was impossible to reproduce it as a lantern slide.

61. An epithelial filament in the anterior chamber simulating a threadworm.

By George Mackay, M.D., F.R.C.S.E.

(With Plate XIV.)

At the meeting of the Society held on October 15th, 1908, Messrs. F. G. Thomas and J. Herbert Parsons reported a case of dipterous larva in the anterior chamber of a little boy.* In the discussion which followed that communication I referred to a case recently under my observation which appeared to be of a somewhat similar nature, but the diagnosis was in doubt as I had not at that time received a satisfactory pathological report upon the specimen.

The brief communication which I lay before you tonight is in fulfilment of the promise then made to complete my report, and I am glad that I left the diagnosis open to modification, because further investigation proves that while the appearance was certainly suggestive of a parasite in the anterior chamber the case was not of that nature.

Clinical history.—A little girl (Maggie M—), age 7 years, was referred to me by Dr. Bowman of Cardenden, Fife, on June 6th, 1908, on account of a slightly irritable condition of the right eye, associated with an appearance of a thread-like structure in the anterior chamber. The

* Transactions, vol. xxix, p. 11.
child had always enjoyed good health, had no special illness, had never suffered from worms and had not complained much about the eye. The filament had first been noticed when she was eight months old. No independent movement had ever been detected in it, but the parents were under the impression that it was slowly increasing in size. The appearance on admission is well seen in the accompanying illustration (Fig. 57). There was a little occasional lacrimation and a trace of ciliary injection. The pupil was approximately circular, dilated well under atropin, but not ad maximum, perhaps a little more fully above than below. There were no visible synechia. It responded freely to light and accommodation. The tension was normal, the media transparent, the fundus could be readily seen. No trace of the filament (even with the pupil dilated) could be detected with the ophthalmoscope behind the plane of the iris. The most remarkable feature was the thread-like foreign body in the lower part of the anterior chamber. At the periphery of the iris, close to the angle of the anterior chamber,
between the 5 and 6 o'clock meridians, a buffy-grey filament about a millimetre in diameter emerged from between the fibres of the iris, ascended in close relation to the posterior aspect of the cornea for three or four millimetres, then bent backwards towards the lower border of the pupil, and crossing the vertical meridian of the anterior chamber, appeared to rest lightly in contact with the lower part of the sphincter surface of the iris. Thereafter it bent downwards, following the plane of the iris nearly in the 7 o'clock meridian. The structure terminated in a slightly bulbous free extremity which did not re-enter the iris nor extend to the angle of the anterior chamber. The whole filament had thus a slight spiral twist and, as I said last October, it looked exactly "as if a small thread-worm had entered the anterior chamber from behind, ascended the posterior surface of the cornea for a short distance, turned backwards, traversing the anterior chamber antero-posteriorly, but meeting the resistance of the iris, had hung its head downwards and given up the struggle."

The refraction of the eyes was about emmetropic. The vision of the right eye $\frac{6}{12}$ fairly and the left $\frac{6}{6}$.

In view of the parents' statement, and fearing lest the retention of this foreign body might jeopardise the future usefulness of the eye by setting up further irritation, I determined to try to remove the thread. The child was put under chloroform, and with a bent triangular keratome an incision was made at the corneo-scleral junction a little to the nasal side of the filament, which was then grasped with iris forceps at its point of emergence from the iris below. It proved to be of somewhat brittle consistence and snapped at the junction of the ascending and descending limb, requiring a second introduction of the forceps for the removal of the distal fragment. This was happily accomplished and healing took place without further incident.

On inquiry lately Dr. Bowman informs me that the eye appears now quite normal and the child is well.
Pathological examination.—The specimen was placed in a weak solution of formalin and submitted to Dr. Theodore Shennan, of Edinburgh, for examination. It was first placed in a glycerine and water solution (about 30 per cent.). The strength of the glycerine was increased and the specimen examined in pure glycerine, but no structure could be made out. The glycerine was then washed out, the specimen gradually dehydrated in alcohol, cleared in cedar oil, washed out in xylol and examined in balsam, but still no structure could be determined. The balsam was slowly removed with xylol and the specimen brought back again through alcohol into water. What I have called the "distal fragment" or "descending limb" was broken during Dr. Shennan's manipulation.

The fragments were now sent to Mr. A. E. Shipley, F.R.S., at Cambridge, who had examined the specimen previously referred to for Messrs. Thomas and Parsons. Mr. Shipley was good enough to make serial sections of one fragment. They are exhibited this evening under the microscope, and one section is illustrated in Pl. XIV, figs. 1 and 2.

Mr. Shipley reports that the tissue is not well preserved, and that the sections seem to point to the specimen being an epithelial growth. It shows no trace of being a nematode or other parasite, and Dr. A. K. Anderson, who had kindly examined it along with him, thinks that there is little doubt that its nature is epithelial. Dr. Anderson recalls having met with somewhat similar growths in the eye of a cat from the margin of the iris.

The main mass of the section consists obviously of stratified epithelium with unstained nuclei. On the surface of the filament, presumably that next to the iris, there is an appearance of some loose connective tissue, hyaline in parts with small mononuclear cells in the meshes. At another part nuclei of fibrous tissue are to be seen, and throughout the texture on this aspect of the structure are granules of melaninc pigment. Some of the sections show a similar isolated piece of epithelium
PLATE XIV.

Illustrates Dr. George Mackay's case of an Epithelial Filament in the Anterior Chamber simulating a Threadworm (p. 287).
towards the presumably corneal surface of the filament, and one is illustrated in Pl. XIV.

Remarks.—Such an appearance as this case presented appears to be extremely uncommon, and the only thing at all like it which I have been able to discover is the case reported to the International Ophthalmic Congress at New York on September 14th, 1877, by the late Dr. Argyll Robertson, and recorded in the Compte-Rendu of the Congress, p. 103, as a filamentous body in the anterior chamber of the eye of a man, aged 39 years. The filament in this case entered the anterior chamber from the base of the iris at the nasal side, and following the corneal curvature, advanced more than halfway across the pupil and terminated in an abrupt curve like a crochet hook. In the erect position it appeared to rest lightly against the cornea, but if the eye was vigorously moved, and especially if the head was thrown well back, the filament could be made to oscillate. There was apparently a little haze at the corneo-scleral junction in nearest relation to the point of origin of the thread, and though it was not removed, nor was its precise nature determined, Dr. Robertson concluded that it was not a parasite on account of its long duration, the absence of inflammation occasioned by it, and the want of spontaneous movement. He also put aside the idea that it was an eyelash because there was neither trace of a cicatrix nor history of a penetrating wound.

The same conclusions appear to be justified in the present case, further confirmed by the pathological report. In the case of Maggie M—, it seems to me that the epithelium has been derived rather from the iris than from the cornea. In Fig. 57 between the pillars of the spiral filament there is an area in which the epithelial layer of the iris is thinned, suggesting that there may possibly have been in embryonic life a separation of some cells which have gone to form this abnormal epithelial thread, much in the same way as a dermoid on the conjunctiva may be associated with a coloboma of the lid.
If any member of the Society has had the opportunity of examining the structure of the threads which go to form a congenital capsulo-pupillary membrane it would be interesting to know whether there is any resemblance to the microscopic structure shown in the present case. It only remains for me to thank Dr. Shennan and Mr. Shipley for their kind assistance in this inquiry.

(June 10th, 1909.)

Mr. Herbert Parsons said he saw those sections some time ago, and they did not show the structure very well. In appearance they resembled stratified epithelium; but it was difficult to understand how, without a wound or without the possibility of the downgrowth of epithelium or an implantation cyst, epithelium of a stratified nature could get into the anterior chamber. He thought it possible from the appearances under the microscope, that it might be endothelial, and, if so, be derived from the endothelium on the surface of the iris.

62. The report of the International Commission on the determination of visual acuity and of the notation of the meridians of astigmatism.

By Walter H. Jessop.

I have brought this subject before the meeting to-night in order to show the Society the findings of the Commission appointed by the International Ophthalmological Congress of 1904. This Commission consisted of Messrs. Charpentier, Dimmer, Eperon, Hess, Jessop, Nucl and Reymond.

(1) Determination of Visual Acuity.

The Commission at the commencement of its labours
agreed that theoretical speculations with regard to visual acuity, such as the dimension of a cone, should be laid aside, and that the subject should be approached in a thoroughly practical manner.

Visual acuity can not be determined by one point, but must have a simple figure as a test object.

The unit of measure of the acuity of vision remains the angle of one minute (*minimum separabile*).

For test-types the objects employed should be either the broken rings of Landolt or Arabic numbers, and the number of objects in each line of the types should be limited to three or four.

Two arrangements of test-types are published with the report, consisting of numerals (1, 4, 7, 0) and the broken rings.

The standard distance at which the test-types are to be used is five metres.

The objects in the types are to be arranged in lines, and as to size in arithmetical progression from 1.1 to 2.

The different lines on the test-types are to be numbered in gradation, 1, 2, 3, 4, 5, 6, 7, 8, 9, 1, 1.5, 2.

The lighting of the types is to be by diffused daylight, when possible, the source of the light to be opposite to the type and not placed laterally.

(2) Unification of Notation of the Meridians of Astigmatism.

The Commission carefully considered the eleven different methods in use for denoting the axes of astigmatism. This diversity of opinion as to the numbering of the axes has prevented hitherto any satisfactory conclusion being arrived at, although the subject has been dealt with by two committees and many oculists.

The results of the findings of the Commission, which were agreed to by the Congress, were the following:

(a) That the axes of cylindrical glasses should be
measured and represented as the observer looks at the patient's face.

(b) That the method adopted should be as simple as possible, avoiding all extra letters or signs, and that the same number should correspond to symmetrical meridians. To insure these points the Commission consider that the numbering of the axes should start from the middle line of the face in each eye and proceed upwards and temporalwards. The zero of the scale would therefore lie at the nasal end of the superior semi-circle, and 180° at the temporal end; 90° would be above and midway between these points. (June 10th, 1909.)

![Diagram](Fig. 58)

Mr. Adams Frost asked what was the advantage of the decimal notation over the old vulgar fraction. Under the present system \( \frac{5}{2} \) showed the distance at which the patient stood, as well as the type read. If one said \( 5 \) it showed that the vision was half the normal. He thought the arrangement which had been decided on was a backward move.

Mr. Holmes Spicer asked what was the test which the Committee used for astigmatism under those conditions. The disadvantage of the "C" types against the letters now used in England was that they were circular, and did not provide lines which ran in various directions. With the ordinary Snellen's letters it was not necessary to use a clock, and the letters themselves were a sufficient test of astigmatism. He asked whether the present system gave a corresponding test. The figures in the other test only gave lines running in certain directions and not in all directions.
Mr. Arnold Lawson asked whether the old ideas about twenty feet were to be upset. One great advantage about the rings was that the testee could not learn them off by heart, as he was sure some did the present series. Where the attempt was made to get the exact visual acuity the letters were not altogether satisfactory. Patients recognised certain blurs, and identified them as certain letters.

Mr. Ernest Clarke asked whether the decisions of the Committee were to be regarded as final. If so, there seemed little use in discussing them. It would be interesting to hear what was meant by "diffuse daylight." "Diffuse daylight" that afternoon would be worth 6/12, whereas on the previous day 6/6. Those who were practising in London found it better to have artificial light, of a definite standard, so that the same illumination was obtained each time.

Mr. Treacher Collins asked how it was proposed to read the type on the left side of the board.

Mr. Jessop replied that he brought up the diagrams and report to show what was the finding of the Committee. It was suggested that 4, 7, 1, 0 should be the four used, and those were measured so that they would represent in the square the same amount of white and black, and at the same time they were measured up to the angle of one minute. It must be remembered that there were many analphabetics about, especially on the Continent, and it was necessary to adopt something which would suit them. But there would be no objection to having letters also. The findings were settled, and he had brought the matter up to show how it worked out.

Mr. Lawford asked whether the types now shown were measured at the five-metre distance, and were numbered for that distance, so that they must be used at that distance or less.

Mr. Jessop replied in the affirmative.
63. *New pedigrees of cataract—posterior polar, anterior polar and microphthalmia, and lamellar.*

By N. Bishop Harman.

**Posterior Polar Cataract (Fig. 59).**

Pedigree of W-T—family.

This family came under inquiry through the members of Generation III, 10-12, two of whom had been under my care in London County Council blind schools.

The type of the cataract scarcely varies amongst eight of the ten affected individuals, so one description may stand for all.

The eyes of the affected are rather small; the lids and general surroundings of the eye are normal. Cornea and iris are normal. The affected lens has a deep sheen, which indicates an opacity behind or deep in its substance; when the iris is dilated by a mydriatic the pupil opens about half way, 6 to 8 mm.; then the opacity is easily seen. It affects the central half of the posterior part of the capsule, and also the lens-fibres immediately anterior to the capsule to a greater or less degree. There can be no doubt that the lens-fibres are affected in all cases, and the variation in the extent of this affection can be best seen by stating the conditions found in one childhood of three girls (III, 10-12), all of whom are affected. No. 10 has the usual type of cataract. In No. 11 the opacity advances so as to include the nucleus. In No. 12 the opacity projects clear through the pupillary opening of each eye, like a tuft of white coral. Indeed, when I first saw the girl I thought the lenses had been needled for cataract, and that opaque lens matter was projecting into the anterior chamber, but when the pupils were dilated I found the projecting matter was part of a true "axial cataract," which had its apex in the projection into the anterior chamber and a broad base at the posterior capsule. When these lenses were looked at with oblique
focal illumination the cataractous matter in the surrounding clear lens substance gave the appearance of a rock submerged in a clear pool of water whose crest was raised clear of the surface.

Fig. 59.

(W-T-family)

Posterior polar cataract.

Hypermetropic astigmatism and Fuchs' colobomata of optic discs.

From the similarity in the appearances of these cases to others where the persistence of the embryonic hyaloid artery has been demonstrated, I am inclined to think that such an artery persists in all these cases.
Vision of the affected members.—All of them have internal squint of one or other eye, and all have constant nystagmus. They can get about quite well; they count fingers at about one metre; one man (III, 3) can read the newspaper slowly when it is held at the distance of 5 cm. from his better eye. III, 11 and 12 are the most nearly blind, for in these the cataract affects a great part of the lens.

In only one of the ten affected persons has an attempt been made to remove a lens: III, 6 had her right eye operated on at a London children's hospital over twenty years ago. The hospital secretary kindly sent me the following reply to my inquiry:

"J. W—, in-patient three times. December, 1886, right lens needled, but no disturbance or absorption followed. April, 1887, iridectomy on right eye. August, 1887, right eye needled; suppurated; evisceration. Left eye cataract. No mention is made in the notes of a 'hyaloid artery.'"

Of the general characters of the family.—They are all stunted or undersized, particularly the affected members. Intelligence is below the average. The enamel of the teeth is good, in no case was pitted or honeycombed enamel found even when there was gross rickets of the bony skeleton (IV, 6—9). Marriages: There has been no in-breeding.

The pedigree covers about fifty individuals. In the affected branches there are twenty-two individuals; of these ten are affected in three generations; I have seen each of these. The inheritance is always continuous; in one branch the line is through male—male to the third generation, in another line the female carried it on. Where the defect arose originally cannot certainly be said, for all inquiries tend to show that the parents derived from the two unrelated childships shown in Generation I were normal eyed.
Description of Cases.

**Generation I.**

The male progenitor (3) came from Hereford; he and his brothers and sisters were working tailors. It is said that they all had good sight. That they "worked at the needle all day" would seem fair evidence that they were not affected with the cataract that appeared in the next generation.

The man married a London woman (6), the eldest of three children. All her family are said to have had good sight. The woman lived to the age of eighty-nine years, and only died two years ago. Four people who knew her and know her children are sure her eyes were quite right and not like those of her blind children; "she could read well with her glasses, and her sight only failed in the last two years of her long life."

**Generation II.**

1, female, died æt. 27 years; married, no children; eyes "quite right."

2, male, æt. 67 years; posterior polar cataract; married a woman with normal eyes; five children, three of whom are affected.

3, female, died young; eyes "quite right."

4, female; died after birth of second child; her eyes were normal, as also are those of her two sons; one is a soldier, the other a bootmaker (II, 8 and 9).

5, female, æt. 50 years; posterior polar cataract; married a man with normal eyes; has three girls, all affected.

6, male; cannot be traced. His relatives say his eyes are quite right, and that when last seen he had several children with good eyes.
Generation III.—Nos. 3 to 7, children of II, 2; father affected; mother normal.

3, male, aet. 36 years; baker's assistant; posterior polar cataract. He married a normal woman and had by her eight children; two had the cataract and four have hypermetropic astigmatism and Fuchs' colobomata of the optic discs. It is noteworthy that his wife had previous to her marriage borne a child to another man; this boy, aet. 14 years (IV, 1), has been seen; his eyes are normal.

2, female, aet. 34 years; eyes said to be quite normal; married, has six normal children. The father of this woman will not permit communication with her on the subject of eyes.

5, female, aet. 30 years; normal; married, no children.

6, female, aet. 33 years; posterior polar cataract in left eye; right has been eviscerated, vide supra.

5, female, aet. 30 years; posterior polar cataract both eyes.

Nos. 10 to 12 children of III, 5; mother affected; father, normal eyes.

10, female, aet. 22 years; posterior polar cataract.

11, female, aet. 19 years; cataract involves the nucleus and posterior pole.

12, female, aet. 14 years; cataract involves axis of lens, vide supra; now attends blind school. (This family is in a miserable state of destitution; the father has disappeared.)

Generation IV.—Nos. 2 to 9 children of III, 3; father affected; mother normal.

2, male, aet. 14 years; lenses normal; high H. As. V. with glasses 6/9.
3, female, aet. 11 years; lenses normal, V. = 6/9.
4, female, died aet. 4 years, of meningitis; "had the cataract like her father."
5, female, aet. 9 years; not seen; schoolmaster replied to inquiry that vision was 6/6 this year.
6, female, aet. 8 years; V. = 6/12, H. As. 2 D.; lenses normal; rickets.
7, male, aet. 6 years; compound H. As. 4 D.; lenses normal; rickets.
8, female, aet. 5 years; H. As. 3 D.; lenses normal; rickets.
9, male, aet. 2½ years; posterior polar cataract involving nucleus; rickets.
Nos. 2, 6, 7 and 8 have Fuchs' colobomata of the optic discs. (All the irides of this family are blue.) The rickets noted in the last four children appears to have no relation to the eye conditions, but is associated with poverty and mal-nutrition.

**Microphthalmia and Anterior Polar Cataract (Fig. 69).**

Pedigree of S—, P—, and T— family.

This family came under inquiry through Generation III, 8, who is an inmate of a London County Council residential blind school.

The defect that has appeared in four members of this family, in two generations and three childships, constitutes a serious and irremediable form of blindness.

The globe is very small, the corneal diameter measures 7 to 7·5 mm., the iris is poorly developed, the pupil eccentric, and on the front of the lens is a plaque of dense white tissue. Both eyes are equally affected. There is
marked internal squint and constant nystagmus. Mentally the affected ones are below average. One died of paralysis in an asylum, but an unaffected sister made a similar end also.

The vision of the affected members is necessarily very poor, but they can see enough to find their way about. Generation III, 3 is the worst; she has only perception of light. III, 8 is the best, for she can read Jaeger 12 at 5 cm.
NEW PEDIGREES OF CATARACT. 303

Description of Cases.

Generation I:

The pedigree begins with the S— family. Two are remembered, but nothing can now be discovered about the state of their eyes.

I, 1 had two children (II, 1 and 2), who were seen within recent years; their eyes were normal. 1, 3 married twice; nothing is now known of the eyes of herself or of her husbands. It would seem that the initial defect, either actual or potential, lay with her, for in each of the childships by her two husbands she had one affected child.

Nos. 5 to 7 represent in their proper order the childship from which the consort of the progenitor of the third generation sprang. She is a healthy old lady, at 73 years, the eldest of ten, of whom eight are yet alive; their eyes are all normal.

Generation II.—First childship of I, 3.

3, male, dead, eyes normal.
4, female, dead. "She had tiny eyes and a terrible squint."

Neither of these married.

Second childship of I, 3.

5, 6 and 7, females; normal eyes; never married.
8, female, normal eyes, died in a lunatic asylum.
9, male, died, at 40 years, of paralysis in an asylum. His wife (10) has normal eyes; she is fairly robust and healthy generally.

Generation III.—Childship of II, 9 and 10; father affected; mother normal.

1, female, at 24 years, normal, married to a normal man, and has one child, a normal girl, at 2 years (IV, 1).
2, female, aet. 20 years, affected; V. = P.L. only; inmate of a blind asylum.
3, female, aet. 19 years, normal, laundymaid.
4, female, aet. 16 years, normal, housemaid.
6, male, died, aet. 1 year and 10 months; "eyes were perfect."
7, male, died, aet. 8 weeks; "eyes were perfect."
8, female, aet. 12 years, affected; in London County Council blind school. She has a high-arched palate and badly placed teeth; enamel normal.

Lamellar Cataracts (Fig. 61).

Pedigree of M—family.
The affected member of the third generation attended Mr. Treacher Collins' clinic at Moorfields, and was operated upon for lamellar cataract. Inquiry elicited the information that the father had a similar defect, but no other member of his family or of his connections were affected.

Generation I.
Nos. 1 to 5 form one childship. All are said to have normal eyes. All married; 1 to 4 have several children each. 5 was a tailor and had "perfect sight." He married the sixth of a childship of eight; this woman is still alive, aet. 80 years. Of the rest 6 and 10 had normal families; 7, 9, and 13 died unmarried; 8 and 12 died young.

Generation II.
No. 2, male, aet. 49 years, has lamellar cataract, worse in the right eye; teeth badly honeycombed; he had fits very badly as a baby.
Nos. 3, 4, 5, 6 are all normal; they married late in life so have small families, except 5, who has no issue. The children are normal. No. 6 is a very shrewd woman, and
from her most of the information concerning the family was derived.

*Generation III.*—Nos. 1 to 5, childship of II, 2, father affected, mother normal.

Fig. 61.

1, male, died æt. 2 years; eyes believed to be good; did not have fits.

2 and 3 died shortly after birth.

4, male, æt. 14 years; sight was only found to be defective one year ago. R.V. = $\frac{6}{9}$, fine lamellar opacity in lens; L.V. = $\frac{3}{6}$, with difficulty; well-marked lamellar of large size. Central incisors and first molars badly honeycombed; he had fits in infancy.

5, male, died shortly after birth.

The occurrence of the same order of defect of the eyes and teeth in father and son is very suggestive of a direct germ inheritance. In favour of some defect of the paternal germ-plasm may be cited the feeble viability of the progeny.
of the man: only one child survives out of five, and that one has grave defects of eyes and teeth. Against this supposition must be set the fact that there appear to be no defects in the other members of a fairly wide family connection. The conclusion must be indeterminate unless the affected son marries and has children.

(July 9th, 1909.)

64. A preliminary note on the treatment of eye disease by radium.

By Arnold Lawson and Mackenzie Davidson.

During the last four months Mr. Mackenzie Davidson and myself have been trying the effects of radium on various external diseases of the eye. The cases have been taken from my outpatient clinic at Moorfields Hospital, and have all been selected as instances of maladies which usually prove obstinate or react slowly to the usual forms of treatment, or which—such as hypopyon ulcer—are of an active virulent nature, and often require some drastic remedial measure.

The reason of this selection was that we felt that the value of radium could best be shown by demonstrating, if possible, its utility in what may be described as difficult or severe cases, and consequently we passed over for a time all cases of a simple or straightforward character which are readily amenable to ordinary forms of treatment.

The number of cases which we bring forward to-night is but a small one, numbering seventeen in all; but in every case a result from the exposure of radium has followed which may not in each instance be conclusive as to the value of this method of treatment, but which, in several of them at any rate, shows that radium is a very powerful and successful agent in the treatment of eye diseases.

In other words, the results we have so far obtained are
extremely encouraging, and it is with the hope that others may be induced to experiment further with this new method that we venture to submit the following abstract of cases as a preliminary note to more dogmatic statements later on, which must necessarily require a much larger experience, a more extended period of time, and a vastly greater record of cases.

Of the seventeen cases, ten are examples of corneal ulceration, four are concerned with non-ulcerative diseases of the cornea, whilst there are two cases of inflamed pterygium and one case of recurrent episcleritis.

The ten cases of corneal ulceration included three cases of hypopyon ulcer. In two of these the results of exposure of the infected area to radium were very extraordinary.

One was the case of a woman, aged 30 years, who came to the hospital with a large, grey, deeply infiltrating ulcer near the centre of the cornea and a small hypopyon. There was great redness of the whole bulbar conjunctiva with the usual symptoms of photophobia and lacrimation, and there was also some iritis. The patient gave a history of inflammation of the eye of about a week's duration. A report by Dr. Wedd, the hospital bacteriologist, stated that diplococci were present in scrapings but were not absolutely identifiable without cultivation. The same day 25 mgrm. of radium were applied for five minutes. The next day the patient was already better. There was still a small hypopyon with great congestion of the globe, but the patient expressed herself as much more comfortable, and all subjective symptoms were less marked. The patient did not trouble to attend the hospital again for a fortnight, and when she came the eye was quite well, the conjunctiva white, and only a faint nebula showed where the ulcer had been. At her last visit she had been given boric lotion and atropine and told to keep the eye bandaged, and she had got on so well that she had not troubled to come.

The second example of hypopyon ulcer was the case of
a man, aged 35 years, who gave a history of injury to the right eye four days previously. There was a deep ulcer of the cornea 2 mm. in length and 1 mm. in breadth, surrounding a grey infiltrated area 4 mm. in diameter. The pupil was sluggish, with marked ciliary injection, and there was a large hypopyon. The same day 29 mgrm. of radium were applied for five minutes. Five days later the injection was subsiding, the ulcer had healed over, and the hypopyon had disappeared. On that day 29 mgrm. of radium were for a second time applied for three minutes. Two days later, or eight days after admission, the man was discharged from the hospital, the eye being then not quite white, but for all practical purposes quite well.

The third case of hypopyon ulcer was a little different to the other two, because a paracentesis of the anterior chamber was performed twelve days before radium was used. The case was an extremely bad one. There was a large, dirty-yellow spreading ulcer with a large hypopyon, and although the paracentesis did much good, the lower third of the cornea, at the date when radium was used, was densely infiltrated and sodden, and was bulging considerably. Forty-nine mgrm. of radium were applied for five minutes. No pain or reaction followed, and improvement rapidly ensued, so that six days later healing was well advanced, and the sodden area was less than two thirds of the former size. A second exposure to 59 mgrm. of radium was given for five minutes on this day, and no reaction or pain followed. From this time up to his discharge from the hospital the patient made steady progress, the corneal lesion healed with a dense, flat scar, which was very much smaller than seemed probable when the radium was first employed, and vision had improved from "fingers at one foot" to $\frac{4}{60}$.

A third exposure of 39 mgrm. was given a week before the patient left the hospital. After the first exposure to radium no other treatment was adopted beyond bandage, atropine and boracic irrigation.
Two cases were good examples of that troublesome form of corneal ulceration generally known as "recurrent vascular ulcer of the cornea." In both there was a history of repeated attacks of corneal inflammation, and in both the cornea exhibited an old nebula stretching from the limbus over a considerable area of the corneal superficies; there was a point of active ulceration at one part; superficial pannus was present, with a large leash of engorged vessels at the limbus. In both cases it was determined to try the effects of radium applied in very small quantity, and an exposure of five minutes to 5 mgrm. was given. In one of these cases the ulcerated point did not stain with fluorescein on the following day, and three days later the pannus had almost disappeared, and the subjective symptoms, which had been severe, were much less. Another exposure of the same duration and with the same dose was given, and nine days later, when the patient was next seen, he was quite well. In the other case, bandaging, boracic irrigations and yellow ointment were tried for four days before using radium, but the patient was no better at all at the end of this time. One exposure of five minutes to 5 mgrm. of radium was then given, and the patient was much better three days later, and quite well ten days after the exposure. As in the other cases, the only treatment when once radium was employed was boracic lotion and atropine.

Another case was of an extremely interesting nature. The patient, a woman, aged 24 years, had been attending hospital since January, 1908, with constantly recurring punctate erosions of one or other eye. No sooner was one eye well than the other would become affected, and as a result for over fifteen months she had been in almost constant discomfort and pain, and quite unable to depend on her eyes from one day to another. All sorts of different methods of treatment were tried, but none proved of any lasting service, and each attack caused complete disablement for a week or two, or sometimes longer. In March this year, when the left eye was affected, 59 mgrm.
of radium were applied for five minutes. The cornea showed numerous punctate nebulae, some of which stained with fluorescine, together with superficial pannus, and there was marked pain and photophobia. Three days after the exposure the photophobia and pain had gone, and the eye was much whiter. All traces of pannus had disappeared a week after exposure, and the eye then kept quite free from further symptoms for six weeks. At the end of this time one small fresh erosion appeared on the left cornea, and an attack of the usual character had commenced in the right cornea, which exhibited three small staining areas. Radium, 49 mgrm., was applied for five minutes to the left and for three minutes to the right. Two days later the right cornea did not stain and the eye was white, and all irritability had disappeared. The left did not show such good progress, and this eye was still irritable though better, but a few days later it seemed quite well. The exposures were followed by no reaction or pain. Since this time the right eye has again given a little trouble, and radium has again been used, so that the disease has not been subdued; but at the same time it has been extremely interesting to note how that each time radium was employed rapid painless improvement in all the local symptoms immediately followed, with very great relief to the patient.

Another interesting case was that of a woman, aged 30 years, who had suffered from vesicular keratitis in the left eye for six months. There was a linear infiltrating ulcer extending over more than a half of the vertical corneal diameter and many tiny vesicles, which ruptured on irrigating the eye, were scattered over the corneal surface, so that after irrigation quite two thirds of the cornea stained with fluorescine. Radium, 5 mgrm., was applied for five minutes and atropine boracic lotion and bandage ordered. Four days later the linear ulcer only stained over about one sixth of its former extent and staining was confined elsewhere to a few minute specks.
A similar exposure of radium was again given and the cornea was quite healed nine days after the first application. Whether or not this will be a cure remains to be seen, but, as in the immediately preceding case, the rapid improvement after the radium exposure was curious and interesting.

With regard to the remaining three cases of corneal ulceration one was that of a woman who gave a history of pain and photophobia on and off for five months. There was a white infiltrating ulcer in the upper outer quadrant of the cornea of a sluggish, non-infective character. Radium, 29 mgrm., was applied for five minutes, and three days later the ulcer did not stain with fluoresceine, the cornea was brighter, and the patient had lost all her subjective symptoms. A fortnight later it was only by the closest inspection that one could discern the faint nebula which marked the site of the former ulcer.

I will not take up the Society's time further this evening with narrating the other two cases, both of which responded to radium in a marked way but which presented no specially interesting points beyond those associated with all the others in common, viz., a cessation at once of the spread of the ulcerating surface and a tendency to rapid healing.

With regard to the four cases of corneal disease of a non-ulcerative nature one was a case of very severe old-standing trachomatous pannus. Radium in this case, though tried three or four times, seemed to do no good at all, and after one exposure the patient had a good deal of pain and increased vascularity. Another case was one of old specific interstitial inflammation in a child, where the disease had left dense central corneal opacity. The child certainly improved very much after the radium was used, but we feel that one instance of this kind is of little value in a disease where the usual course is for improvement to follow merely the lapse of time and attention to general health. The third and fourth cases were also examples of interstitial inflammation. One of them was that of a girl who had been attending the
hospital for five months with little benefit. The result of radium was astonishing, but not till three exposures had been given. After the first exposure of 49 mgrm. for five minutes there was increased pannus and photophobia with some pain for a few hours after the application, but this soon quieted down and a week later she was already better. The second and third applications were not followed by any reaction, and ten days after the third application, or three weeks after the first exposure, all active symptoms of inflammation had gone, the pannus and photophobia had disappeared, and a patchy corneal haze was alone left. So that radium in this case effected in about three weeks what treatment on the usual lines had not commenced to effect in five months.

The effects of radium in the remaining interstitial case was doubtful and no value can be attached. Improvement followed, but it may well have been due to several causes.

Two cases on our list were examples of inflamed pterygium. One had had six operations of transplantation performed by one of my colleagues during the last four years without any lasting success.

After each operation the condition generally reasserted itself within six months. When radium was first tried the pterygium was causing marked subjective symptoms. It was very red and inflamed and the corneal part of it swollen and juicy. The patient has had several applications of radium and the improvement has been steady and progressive. All subjective symptoms rapidly disappeared, so that the patient said that the eye felt better than it had done for years. At the present time after three months there is still a little redness, but it is vastly less than it was and the improvement is extremely marked.

In the other case of pterygium there was a persistent inflammation of the conjunctiva following a successful transplantation in each eye. The irritation had gone on for several months. Two exposures of radium were followed by great relief to the subjective symptoms.
Lastly, one case was an example of recurrent episcleritis, and here the radium was very successful, and quickly effected a cure when the patient had been suffering continually for over two months without any improvement. There was much swelling of the sclera with violet injection and a considerable degree of sclerosing keratitis. Much pain, lasting about twenty hours, followed the first application of 59 mgrm., which was employed for ten minutes—a much longer exposure than we have been accustomed to use—and the pain was also accompanied by flashes of light appearing to proceed from the opposite side to that on which the radium was applied; but after this the progress was rapid. An attack of influenza caused a slight recrudescence of inflammation a few weeks later, but this quickly passed away and the patient has since remained well. Only one exposure was given in this case.

A glance at a summary of these cases will show that the dosage of radium employed was very variable. In more than one instance of corneal ulceration a very small dose, such as 5 mgrm., seems to have produced as rapid an improvement as could have been effected by a much larger dose. It is at the present time quite impossible to make any statement with regard to any relation that may exist between the size of the dose and the effect produced.

Nor can much be said as to the length of exposure that should be employed. In nearly all our cases a uniform exposure of five minutes has been used. An exposure of this kind seldom if ever causes inflammatory reaction or pain. On the other hand where exposures of ten minutes or longer have been employed with a large dosage pain has been the rule, accompanied by increase of the subjective symptoms of the disease.

The pain has usually been described as of a numbing and throbbing character, and in one instance, already mentioned, it was accompanied by definite signs of retinal irritation. Both its severity and its duration have been very variable.
The method of exposure has in every instance been to cocainise the eye and to hold the radium enclosed in glass tubes applied to the affected area.

Lastly, we would like to call attention to the fact that in each instance all treatment was dropped after starting radium, except simple boric acid bathing together with atropin when necessary. (July 9th, 1909.)

Mr. Adams Frost asked whether the radium was screened in any way.

Mr. Ernest Clarke asked what was the guide as to the amount of radium to use.

Mr. Hewkley asked whether an attack had been made by means of radium upon the calcareous leucomata. Also whether lesions of a tubercular origin had satisfactorily responded to radium treatment.

Mr. Mackenzie Davidson replied that the radium was applied in sealed glass tubes, the glass cutting off the Alpha rays and permitting the passage to the lesion of only the Beta and Gamma rays. Occasionally he had used a screen of tin foil, or lead foil, but not for eye diseases, because as they wanted to get some data the conditions were varied as little as possible. Therefore an exposure in sealed glass tubes was carried out for five minutes. The eye was cocainised and the lids held open with the fingers, and the radium held close to the eye. If the surface to be treated was fairly large the radium was moved about somewhat. They had not yet any guide as to what was best to use. All his tubes had 5 mgrm. each of radium, excepting one containing 29 mgrm. The treatment at present was tentative. They were feeling their way, but had already had some good results. He could not answer Mr. Hewkley's questions, but the calcareous and tuberculous deposits were worth trying by the method, judging by his experience in the case of keloids. There had been a suggestion to try the method on old corneal opacities. He could not say what was its special beneficial effect on tubercular lesions; but where-
as he could cure epithelioma of the tongue and rodent ulcers and epitheliomata of the skin with radium, he had the greatest difficulty in causing a good effect on lupus. One of the reasons for bringing forward the communication was that it might induce others who possess radium to use it in their ophthalmic practice and get evidence of what could and what could not be done by it.

APPENDIX.

The following cases and communications have also been brought before the Society:

1. A Case operated upon for Paralytic Ptosis by his Method of Resection, Dr. A. Freeland Fergus. (The operation is described in the Transactions, vol. xxviii, p. 184.)

2. An Unusual Adhesion between Cornea and Iris, J. Herbert Bell.


4. Pemphigus of Conjunctiva, C. Blair.
REPORT OF THE COUNCIL,

_read at the Annual General Meeting of the Society,
July 9th, 1909._

The Council is happy to report the continued prosperity of the Society.

During the session now ending twenty-four new members have been elected, and five members have resigned.

Three of the original members of the Society have died during the past session, all of whom had given valuable service to the Society; Dr. Argyll Robertson, a former President, Dr. C. E. Beevor, a former Secretary and Vice-President, and Mr. Simeon Snell, a former Member of Council and Vice-President. Dr. Bell Taylor, a member since 1882, has also died.

The membership of the Society at present numbers 478.

The Treasurer's report and statement of accounts are submitted and show the condition of the finances of the Society.

During the past year the Council has carefully considered the question of amalgamation of the Society with the Royal Society of Medicine. A memorandum regarding this question was prepared by the Council and circulated to all members of the Society resident in the United Kingdom, pointing out the advantages and disadvantages of such an amalgamation.
The Society devoted an evening to the discussion of the proposal and the following resolution was proposed:

"That this meeting, specially convened to consider the question, is of opinion that amalgamation of the Ophthalmological Society of the United Kingdom with the Royal Society of Medicine is desirable, and hereby authorise the steps necessary to such amalgamation being taken in due course."

To this the following amendment was moved:

"That any further consideration of the amalgamation of the Ophthalmological Society with the Royal Society of Medicine be postponed until every member of the former Society has had the opportunity of expressing his opinion for or against such amalgamation, and that a special committee of the Society be appointed to draw up a statement or statements to be sent to each member with a voting paper, setting forth the alleged advantages and disadvantages of the proposed amalgamation."

The amendment was carried by 53 votes to 42, and being put as a substantive motion was passed by a large majority, the actual figures not being taken.

A committee was appointed to carry out this resolution, and by means of a circular will obtain the views of non-resident as well as resident members.

The Bowman Lecture was delivered by Mr. Edward Nettleship on June 10th, the subject being "Some Diseases of the Eye illustrating Heredity."

The three members of the Society entrusted by the Council with the award of the Nettleship Medal selected for the honour Mr. Edward Nettleship for his work on "Heredity in Diseases of the Eye."

The thanks of the Society are due to Mr. Nettleship and Mr. Sydney Stephenson for their contributions to the library during the past year.
ACCOUNT OF THE RECEIPTS AND PAYMENTS
OF THE
OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM.

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- £20 B Annuities, East India Railway.
- £500 Ramsgate Corporation 3% Stock.
- £200 2 1/2% Consols.
- £300 L.C.C. 3% Stock (Nettleship Prize Fund).

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Balance available for ordinary expenditure | £86 13 0 |

J. B. LAWFORD, Hon. Treasurer.
June 21st, 1909.

Examined, compared with vouchers, and found correct.

RAYNER D. BATTEN, Auditors.

ARTHUR W. ORMOND.
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NOTICE.

September, 1902.

Members desirous of using abbreviations in their communications to the Society are requested to confine themselves to those included in the following official list.*

ABBREVIATIONS.

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<th>Abbreviation</th>
<th>Description</th>
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<tr>
<td>Ace.</td>
<td>Accommodation</td>
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<td>Aq.</td>
<td>Aqueous humour</td>
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<td>As.</td>
<td>Astigmatism</td>
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<td>A.C.</td>
<td>Anterior chamber</td>
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<tr>
<td>C.</td>
<td>Cornea</td>
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<tr>
<td>Ch.</td>
<td>Choroid</td>
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<tr>
<td>cm.</td>
<td>Centimetre</td>
</tr>
<tr>
<td>Cyl.</td>
<td>Cylindrical lens</td>
</tr>
<tr>
<td>D.</td>
<td>Dioptre or dioptic</td>
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<tr>
<td>E.</td>
<td>Emmetropia</td>
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<tr>
<td>F.</td>
<td>Field of vision</td>
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<tr>
<td>H.</td>
<td>Hypermetropia</td>
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<tr>
<td>H.l.</td>
<td>Latent hypermetropia</td>
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<tr>
<td>H.m.</td>
<td>Manifest hypermetropia</td>
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<tr>
<td>I.</td>
<td>Iris</td>
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<tr>
<td>L.</td>
<td>Left eye; and R., right eye</td>
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<tr>
<td>m.</td>
<td>Metre</td>
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<tr>
<td>mm.</td>
<td>Millimetre</td>
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<tr>
<td>My.</td>
<td>Myopia</td>
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<tr>
<td>M.L.</td>
<td>Macula lutea; and Y.S., yellow spot</td>
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<tr>
<td>Oph.</td>
<td>Ophthalmoscope, ophthalmoscopic examination, ophthalmoscopic appearances</td>
</tr>
<tr>
<td>O.D.</td>
<td>Optic disc</td>
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<tr>
<td>O.P.</td>
<td>Optic papilla</td>
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<tr>
<td>P.</td>
<td>Pupil</td>
</tr>
<tr>
<td>Pr.</td>
<td>Presbyopia</td>
</tr>
<tr>
<td>P.L.</td>
<td>Perception of light</td>
</tr>
<tr>
<td>p.p.</td>
<td>Punctum proximum</td>
</tr>
<tr>
<td>p.r.</td>
<td>Punctum remotissimum</td>
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<tr>
<td>R.</td>
<td>Right eye; and L., left eye</td>
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<tr>
<td>Ret.</td>
<td>Retina</td>
</tr>
<tr>
<td>Scl.</td>
<td>Sclerotic</td>
</tr>
<tr>
<td>Sph.</td>
<td>Spherical lens</td>
</tr>
<tr>
<td>T.</td>
<td>Tension of the eyeball</td>
</tr>
<tr>
<td>T.n.</td>
<td>Tension normal</td>
</tr>
<tr>
<td>T. + 1, T. + 2, T. + 3, T. - 1, T. - 2, T. - 3,</td>
<td>degrees of increase and decrease of tension</td>
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<tr>
<td>Vit.</td>
<td>Vitreous humour</td>
</tr>
<tr>
<td>Y.S.</td>
<td>Yellow spot; and M.L., macula lutea</td>
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<td>V.</td>
<td>Visus, acuteness of sight, power of distinguishing form</td>
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SYMBOLS.

+ Symbol for a convex lens.
- Symbol for a concave lens.

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