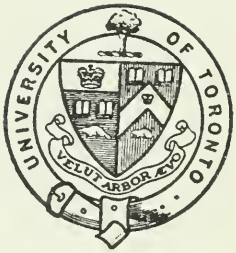

OPHTHALMOLOGICAL TRANSACTIONS

VOL. XXXV

TRANSACTIONS

OF THE

OPHTHALMOLOGICAL SOCIETY

OF THE

UNITED KINGDOM

VOL. XXXV

SESSION 1915

WITH

LIST OF OFFICERS, MEMBERS, ETC.

LONDON
J. & A. CHURCHILL
7, GREAT MARLBOROUGH STREET

1915

ADLARD AND SON, IMPR., LONDON AND DORKING.

IN EXCHANGE.

American Journal of Ophthalmology.
Annales d'Oculistique.
Annali di Ottalmologia.
Annals of Ophthalmology.
Archiv für Ophthalmologie.
Archives d'Ophtalmologie.
Archives of Ophthalmology.
Bericht der ophthalmologischen Gesellschaft, Heidelberg.
Bulletin de la Société française d'ophtalmologie.
Centralblatt für praktische Augenheilkunde.
Klinische Monatsblätter für Augenheilkunde.
Ophthalmic Record, Chicago.
Ophthalmology.
Revue générale d'Ophtalmologie.
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 Thirty-fifth Session, April, 1915.

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

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OFFICERS AND COUNCIL

OF THE

Ophthalmological Society of the United Kingdom

ELECTED AT

THE ANNUAL GENERAL MEETING, APRIL 22ND, 1915.

President.

W. H. H. JESSOP.

Vice-Presidents.

J. B. LAWFORD.

F. RICHARDSON CROSS (Bristol).

A. MAITLAND RAMSAY (Glasgow).

SYDNEY STEPHENSON.

Treasurer.

JAMES TAYLOR, M.D.

Council.

ELMORE BREWERTON.

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GORDON HOLMES, M.D.

J. CRAIG (Belfast).

H. L. EASON.

Secretaries.

GEORGE COATS (*Surgical; Congress Business, Transactions, etc.*).

S. A. K. WILSON (*Medical; Council Business, List of Members, etc.*).

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, F.R.S.
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.
1911	J. B. LAWFORD.
1913	F. RICHARDSON CROSS.
1915	W. H. H. JESSOP.

TRUSTEES FOR THE SOCIETY.

J. B. LAWFORD.
SEYMOUR J. SHARKEY, M.D.

TRUSTEES OF THE NETTLESHIP PRIZE FUND.

J. B. LAWFORD.
W. T. HOLMES SPICER.

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. SNELLEN (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.
1911	E. LANDOLT, M.D. (Paris).
1914	PROF. W. UHTHOFF (Breslau).

EDWARD NETTLESHIP PRIZE.

1904	PRIESTLEY SMITH.
1907	JOHN HERBERT PARSONS.
1909	EDWARD NETTLESHIP.
1912	GEORGE COATS.
1915	EDWARD TREACHER COLLINS.

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

LIST OF MEMBERS OF THE SOCIETY.

Honorary Members.

Professor Zehender, Rostock.
Professor Leber, Heidelberg.
Professor Fuchs, Vienna.
Professor Sattler, Leipsig.
Thomas Buzzard, M.D., 74, Grosvenor Street, W.
E. Landolt, M.D., 8, Rue de Berri, Paris.
Sir William R. Gowers, M.D., F.R.S., 34, Ladbroke Sq., W.

EXPLANATION OF ABBREVIATIONS.

O.M.—Original Member.
Pres.—President.
V.-P.—Vice-President.
T.—Treasurer.
L.—Librarian.
S.—Secretary.
C.—Member of Council.
F.—Foreign Member.
*.—Denotes Resident Life Members.
†.—Denotes Non-Resident Life Members.

GENERAL LIST OF MEMBERS.

ELECTED

1905 Adams, P. E. H., 53, Broad Street, Oxford.
1912 Alexander, G. F., M.B., 35, Prince of Wales Terrace, Scarborough.
1907 Allport, Wilfred, M.B., 133, Edmund Street, Birmingham.
1899 †Amenabar, Daniel, Casiila 699, Santiago, Chili.
1910 Angelucci, Prof. (F.), 2, Piazza N. Amore, Naples.
1913 Anklesaria, M. Dhanjishaw, Ahmedabad, India.
1891 †Armstrong, Hugh, Tamworth, New South Wales.
1895 Attlee, John, M.B., 65, Grosvenor Street, W.
1910 Axenfeld, Prof. (F.), Freiburg, i.B. Germany.

ELECTED

1911 Baker, George Wiston, M.D. (Address uncommunicated.)

1903 Ballantyne, A. J. (C.), 11, Sandyford Place, Glasgow.

1907 Bardsley, Percy C., M.B., 67, Wimpole St., W.

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. (V.P. 1912–15. C. 1901-4.)

1912 Beedham, Henry Wm., 15, Crediton Hill, West Hampstead, N.W.

1909 Bell, T. Herbert, 801, Boyd Building, Winnipeg, Canada.

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. Berry, G. A., M.D., 31, Drumsheugh Gardens, Edinburgh. (*Pres.* 1909-11. C. 1889-92. V.-P. 1895-8. 1912.)

1899 Bickerton, R. E., M.B., 137, Harley Street, W.

1881 *Bickerton, T. H., 88, Rodney Street, Liverpool. (C. 1895-8. V.-P. 1910-12.)

1905 Bird, J. W., 21, Great St. Andrew St., Shaftesbury Avenue, W.C.

1892 Black, John Wilson, M.D.Edin., 46, Academy Street, Inverness.

1898 Blair, Charles Samuel, M.D., 14, Welbeck Street, W.

1895 †Bonar, Thomson, M.D., Elm Lodge, Staplegrove, Taunton, Somerset.

ELECTED

1907 Bond, C. Shaw, Highcroft, Shepherd's Well, Highgate.

1911 Boucher, R. B., M.D., Vancouver, B.C.

1885 Bower, Ernest Dykes, Elton House, Gloucester.

1897 Bower, John A., M.B., 18, Royal Crescent, Cheltenham.

1905 Brailey, A. R., 11, Old Burlington Street, W.

1902 *Brailey, Wm. Herbert, 21, Lansdowne Place, Hove, Sussex.

1911 Brearley, E. A., M.B., University Club, Sydney, N.S.W.

1911 Brennard, H. T. W., University Club, Sydney, N.S.W.

1897 Breuer, August, M.D., 30, Finsbury Square, E.C.

1899 Brewerton, Elmore (C.), 84, Wimpole Street, W. (C. 1909-10. S. 1910-13.)

1903 Brinton, A. G., Box 5852, Johannesburg, South Africa.

1909 Broachâ, R. B., Karachi Eye Hospital, Karachi, India.

1886 Bronner, Adolph, M.D., 33, Manor Row, Bradford. (C. 1900-3.)

1901 *Brooks, R. Philip, 14, Welbeck Street, W.

O.M. Browne, Edgar A., 39, Rodney Street, Liverpool. (C. 1887-90. V.-P. 1893-6.)

1895 Browne, J. Walton, M.D., 10, College Square North, Belfast.

1901 Buchanan, Leslie, 8, Royal Crescent, West, Glasgow.

1907 Buckland, Francis, M.D., Moorland Court, Poole Road, Bournemouth.

1907 Burdon-Cooper, J., M.D., 12, 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., 78, Wimpole Street, W. (C. 1908-9. S. 1909-12.)

1897 †Byers, W. Gordon M., M.D., 346, Mountain Street, Montreal.

1892 †Caiger, Herbert, M.B., Burghersdorp, Cape Colony.

1905 Campbell, C. A., 172, Bloor Street E., Toronto.

1891 Campbell, E. Kenneth, M.B., 23, Wimpole Street, W.

ELECTED

1882 *Cant, W. J., White Cross, Lincoln. (C. 1898-1901.)

1891 Cargill, L. Vernon, 35, Cavendish Square, W. (C. 1905-8.)

1912 Carr, Edward, Laround, Colne, Lancs.

1895 Cartwright, E. H., M.D., Myskyns, Ticehurst, Sussex.

1911 Chambers, W. J., 212, VII Avenue W., Calgary, Alberta, Canada.

1885 Clarke, Ernest, M.D., B.S., 3, Chandos Street, Cavendish Square, W. (C. 1893-6. V.-P. 1910-12).

1911 Clegg, John Gray, M.D., 22, St. John Street, Manchester.

1905 Clements, E. C., 3, Lindum Road, Lincoln.

1902 Coats, George, M.D., (S.), 50, Queen Anne Street, W. (C.)

1907 Cobbledick, Arthur Stanley, M.D., B.S.Lond., 368, Brixton Road, S.W.

1885 Collins, E. Treacher, 17, Queen Anne Street, Cavendish Square, W. (V.-P. 1905-8. C. 1889-92. 1901-4. S. 1898-1901.)

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 Coorey, Edward A., M.D., Bolton, Bainbalapitiya, Ceylon.

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

1914 Coppez, Henri, M.D., 24 Avenue des Arts, Brussels.

1904 Corbett, W. J., 18, Weymouth Street, 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.

ELECTED

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

1901 Crawley, F. C., M.D., 5, Fitzwilliam Place, Dublin.

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

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

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

O.M. Critchett, Sir G. Anderson, Bart., C.V.O., 21, Harley Street, W. (*Pres.* 1899-1901. C. 1883-6. V.-P. 1894-7, and 1902-5.)

1898 Critchley, Harry G., M.D., 83, Park Lane, Croydon.

1906 Crocket, A. Pierce. (Address uncommunicated.)

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

1881 Cross, F. R. (V.P.), Worcester House, Clifton, Bristol. (*Pres.* 1913–15. V.-P. 1898-1901. C. 1891-4.)

1902 Cruise, R. R., 85, Harley Street, W.

1912 Cummins, J. D., M.B., Clare Street, Dublin.

1904 Cunningham, H. H. B., 30, University Square, Belfast.

1905 Cunningham, J. F., 27, Weymouth Street, W.

1909 Daggan, J. N., Sir J. C. Ophthalmic Hospital, Girguina, Bombay.

1907 Davidson, Alex. Dyce, "Hollydene," Cholmeley Park, Highgate, N.

1884 Davidson, Sir James McKenzie, M.B., 26, Park Crescent, W. (C. 1892-5.)

1903 Davies, David, 8, Lonsdale Gardens, Tunbridge Wells.

1908 Davies, D. L., M.S., M.D., 31, 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.

1909 Dé, Chandra Mohan (Rai Bahadur), Med. School, Agra, U.P., India.

1914 De Lapersonne, Prof., M.D., 30 Rue de Lisbonne, Paris.

1903 Dean, C. W., Lindow Square, Lancaster.

ELECTED

1903 Dickson, R. H., Nelson Place, Newcastle-under-Lyne, Staffordshire.

1910 Dighton, Chas. A. Adair, M.B., 73, Rodney Street, Liverpool.

1910 Dimmer, Prof. (F.) 15 Reichsratstrasse, Vienna.

1881 Dixon, W. E., Oulton Lodge, Oulton Broad, Lowestoft.

1889 Dodd, Henry Work, 136, Harley Street, W. (C. 1902-5).

1907 Dombrain, Ernest A., 205, Macquarie Street, Sydney, N.S.W.

1905 Dorrell, E. A., 45, Welbeck Street, W.

1899 Douglas, J. Sholto, M.B., Mara Berbice, British Guiana.

1887 Doyne, Robert W., 30, Cavendish Square, W. (C. 1892-5. V.-P. 1904-7.)

1881 *Drake-Brockman, E. F., 18, Hanover Street, 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, Percy, 54, Wimpole Street, W.

1900 Eason, Herbert L., M.B. (C.), 62, Portland Place, W.

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

1900 Edridge-Green, F. W., M.D., 99, Walm Lane, Willesden Green, N.W.

1902 †Elliot, Robert Henry, Lt.-Col. I.M.S., M.D., Sc.D., 143, Harley Street, 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.

1902 Evershed, A. R. F., 22, Harley Street, W.

1907 Fenton, Capt. A., M.B., I.M.S., c/o Messrs. Thos. Cook, and Son, Rangoon.

1911 Fenwick, George, Auckland Club, Auckland, N.Z.

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

ELECTED

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.

1895 Fisher, J. Herbert, M.B. (C.), 83, Wimpole Street, Cavendish Square, W. (C. 1904-7. S. 1907-10.)

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

1912 Flaxman, Samuel C. R. (Address uncommunicated.)

1889 Flemming, Percy, 70, Harley Street, W. (V.-P. 1911–13; C. 1901-4.)

1892 Folker, Herbert Henry, Hanley, Staffordshire.

1886 Ford, A. Vernon. (Address uncommunicated.)

O.M. Frost, W. Adams, Shepherd's Well, Forest Row, Sussex. (L. 1900-6. V.-P. 1906-9.)

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

1887 †Gardner, John J., M.D., 128, Bishop Street, Montreal, Canada.

1913 Ghaswala, K. S., 3, Churchgate Street, Fort Bombay, India; and c/o Sir Cowasji Jehangir Ophthalmic Hospital, Byculla, Bombay.

1912 Gibb, H. P., 17, Bentinck Street, W.

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., Rosemullion, Budleigh Salterton, Devon. (C. 1896-9. V.-P. 1902-5.)

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

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

1907 Goulden, Charles Bernard, 5, Union Street, Oldham.

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

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

ELECTED

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

1895 †Gray, James, M.D., Helena, Montana, U.S.A.

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

1903 Greene, Arthur, 4, Theatre Street, Norwich.

1911 Greeves, Reginald A., 14, Harley Street, W.

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.)

1910 Griffith, Arthur Donald, M.B., 30, Welbeck Street, W.

1894 Grimsdale, Harold, M.B., 3, Harley Place, Marylebone Road, N.W. (C. 1907-10.)

O.M. Grossmann, K. A., 70, Rodney Street, Liverpool.

1910 Grósz, Prof. E. von (F.), 5, Reviczhy Ter., Buda-Pest, Hungary.

1899 Gruber, Rudolph, M.D., 81, Harley Street, W.

1908 Gunning, C. J. H., 6, Hertford Street, Mayfair, W.

1898 Guthrie, Leonard G., M.D., 15, Upper Berkeley Street, W.

1910 Haab, Prof. O. (F.), 41, Pelikanstrasse, Zurich, Switzerland.

1889 Hailes, C. D. G., M.D., Hawkesdale, 27, Alma Road, Clifton, Bristol.

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.

1912 Hanafin, Capt. P. J., R.A.M.C. (Address uncommunicated.)

ELECTED

1901 Hancock, Edw. D., Merrow Grange, Guildford.

1908 Hanna, Henry, 57, University Road, Belfast.

1901 Hanson, Reginald E., 20, Kensington Park Gardens, W.

1900 Harman, N. B., M.B., 108, Harley Street, W.

1902 Harries-Jones, E., 16, Castilian Street, Northampton.

1910 Harston, George M., M.D., Hong Kong.

1882 *Hartridge, Gustavus, 12, Wimpole Street, W. (C. 1889-92, 1895-8. S. 1892-5. V.-P. 1910-11.)

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.

1899 Henderson, Edward E., M.B., 6, Lower Berkeley Street, Portman Square, W.

1903 Henderson, Thomson, 15, The Ropewalk, Nottingham.

1896 Henry, R. Wallace, M.D., 6, Market Street, Leicester.

1905 Hepburn, Malcolm Langton, M.D., 111, Harley Street, W.

1897 Herbert, Lt.-Col. Herbert, I.M.S., Castle Grove, Nottingham. (C. 1911–13.)

1887 *Hern, John, M.D., Summercote, Darlington.

1910 Heron, David, R.A.M.C. Mess, Grosvenor Road, S.W.

1910 von Hess, Prof. Carl (F.), 1, Pleichenglacisstrasse. Würzburg, Bavaria.

1905 Hewkley, F., 19, Lower Seymour Street, Portman Square, W.

1900 Heyliger, E. D. Scott, M.B., C.M., 89, Preston New Road, Blackburn.

1895 Hickman, H. R. Belcher, M.B., 5, Harley Street, W.

O.M. Higgens, Charles, 52, Brook Street, W. (C. 1880-3, V.-P. 1901-4.)

ELECTED

1913 Hill, Rowland, M.D., 7, University Road, Belfast.

1888 *Hinnell, J. S., M.B., 62, Garland Street, Bury St. Edmunds.

1905 Hird, R. B., M.B., 52, Newhall Street, Birmingham.

1899 Hobday, James, M.B., Beverley House, Colney Hatch Lane, N.

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

1911 Holmes, Gordon M., M.D. (C.), 58, Harley Street, W. (S. 1912–15.)

1889 Holthouse, Edwin H., M.B., 35, Nottingham Place, W.

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

1893 †Howe, Lucien, 183, Delaware Avenue, Buffalo, U.S.A.

1908 Hudson, A. C., M.D., 50, Queen Anne Street, W.

1884 †Hudson, Lt.-Col. Ernest, I.M.S. (Address uncommunicated.)

1889 †Hughes, Samuel H., 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.

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

1898 James, George Brooksbank, 5, Harley Street, W. (C. 1911–13.)

1908 James, Robert Ruston, 6, Lower Berkeley Street, W.

1905 Jaques, R., 20, Athenæum Street, Plymouth.

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

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

1883 Jessop, W. H. H. (*Pres.*), 73, Harley Street, W. (C. 1889-92; V.P. 1907-10, 1914–15.)

ELECTED

1910 Jewell, William H., M.D., 27, Queen Anne Street, W.

1882 Johnson, G. Lindsay, M.B., 5 and 6, Castle Mansions, Eloff Street, Johannesburg, S.A.

1888 †Johnston, Geo. D., Georgia Street, Vancouver, British Columbia.

1898 *Jones, George, M.B., 8, Church Terrace, Lee, S.E.

1897 Jones, Hugh E., 19, Rodney Street, Liverpool.

1894 †Jones, R. H., M.B., B.S., 209, Macquarie Street, Sydney, New South Wales.

1914 Joseph, Hugh Percival, M.R.C.S., The Hospital Bungalow, Gallé, Ceylon.

1905 Juler, F. A., 24, Cavendish Square, W.

O.M. Juler, H. E., 23, Cavendish Square, W. (C. 1886-9. V.-P. 1900-2.)

1914 Karani, N. N., Eye Clinic, 18, Kalbadevi Road, near Edward Theatre, Bombay.

1899 Keeling, G. S., M.D., Attleborough, Norfolk.

1900 †Kelsall, H. T., M.D., 1, Devonshire Terrace, Perth, W. Australia.

1898 †Kendall, H. W. Martindale, Wellington, New Zealand.

1888 †Kenny, Augustus Leo, M.B., 87, Collins Street, Melbourne, Victoria, Australia.

1913 Killen, W. M., M.D., 9, Clifton Street, Belfast.

1904 Killick, Chas., 3, Marsham Street, Maidstone.

1906 Kilkelly, Major P. P., I.M.S., c/o Messrs. H. S. King and Co., Pall Mall.

1895 Knaggs, Robert Lawford, M.D., 27, Park Square, Leeds.

1881 †Knaggs, S. T., M.D., Wellington, Bondi Road, Bondi, Sydney, N.S.W.

1910 Knapp, Dr. Arnold (F.), 10, East 54th Street, New York, U.S.A.

1911 Lang, B. T., B.C., 22, Cavendish Square, W.

ELECTED

O.M. Lang, William, 22, Cavendish Square, W. (C. 1886-9. V.-P. 1900-3. (*Acting Pres.* 1903.)

O.M. Lawford, J. B. (V.-P.), 99, Harley Street, Cavendish Square, W. (C. 1886-9; 1898-1901. S. 1895-8. V.-P. 1905. T. 1905-11. *Pres.* 1911-13.)

1889. *Laws, Wm. G., M.B., 3, East Circus Street, Nottingham. (C. 1909-11.)

1896. Lawson, Arnold, M.D., 12, Harley Street, W. (C. 1909-1911.)

1895 †Lea, J. Augustus, M.B., Grahamstown, Cape Province, S. Africa.

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

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

1912 Lee, H., F.R.C.S., 6, Park Square, Leeds.

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

1911 Letchworth, T. W., 68, Claremont Road, Surbiton.

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

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

1896 Lister, W. T., M.B., 24, Devonshire Place, W. (C. 1907-10.)

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

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

1911 Loosely, Alfred E. A., 25, New Cavendish Street, W.

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

1883 Lunn, J. R., The Haven, Mill Road, Worthing. (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., Public Health Department, Cairo, Egypt.

ELECTED

1913 McCready, W., M.B., 2, University Square, Belfast.

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

1890 *MacGillivray, Angus, M.D., 23, South Tay Street, Dundee, N.B. (C. 1902-5.)

1895 †McIntosh, J. R., M.D., 40, Coburg Street, St. John, New Brunswick, Canada.

1902 Mackay, D. Matheson, M.D., 15, Albion Street, Hull.

1888 *Mackay, George, M.D., 20, Drumsheugh Gardens, Edinburgh. (C. 1900-2. V.-P. 1906-9.)

1889 †Mackenzie, F. Wallace, M.B., 139, Upper Willis Street, Wellington, New Zealand.

1895 McKenzie, H. V., M.D., Elmbank, Abbey Road, Torquay.

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.

1902 McMullen, W. H. (C.), 4, Chandos Street, Cavendish Square, W.

1904 McNab, Angus, 118, Harley Street, W.

1911 McNabb, Henry H., M.D., 8a, St. John Street, Manchester.

1912 McPherson, Major, I.M.S., c/o Ophthalmic Hospital, Bombay, India.

1911 MacQueen, J. F., 12, Royal Terrace, Southend-on-Sea.

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 †Manché, Charles, B.A., M.D., 33, S^da. Alessandro, Valletta, Malta.

1901 †Manning, Leslie S., Christchurch, New Zealand.

1911 Manson, W. H., M.D., 5, Clifton Place, Glasgow, W.

1904 Markus, Charles, M.D., 28, Wimpole Street, W.

ELECTED

1883 †Marlow, Frank William, M.D., 200, Highland Street, Syracuse, New York State, U.S.A.

1892 Marshall, Charles Devereux, 112, Harley Street, W. (C. 1903-4. S. 1904-7. C. 1907-10.)

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., 24, Tavistock Square, W.C.

1884 Maxwell, Patrick William, M.D., 19, Lower Baggot Street, Dublin. (V.P. 1912–15. C. 1900-2.)

1914. Maxwell, Miss E. M., M.B., 19, Lower Baggot Street, Dublin.

1904 May, H. J., M.B., B.C., Naini Tal, College Place, Southampton.

1893 Maynard, Frederic Pinsent, Lt.-Col., Indian Medical Service, 13, Harington Street, Calcutta.

1902 Mayou, Stephen, 30, Cavendish Square, W. (C. 1912–15.)

1912 Mead, John Clarke, 55, High Street, Lowestoft.

1901 Menzies, J. A., 9, Castle Hill Avenue, Folkestone.

1897 Miller, G. Victor, M.B., 2, Barrington Crescent, Stockton-on-Tees.

1881 †Milles, W. Jennings. (Address uncommunicated.)

1897 †Minnes, Robert Stanley, M.D., 127, Metcalfe Street, Ottawa, Ontario.

1911 Mitchell, Leonard J. C., 4, Collins Street, Melbourne.

1896 Mooney, Herbert C., M.B., 22, Lower Baggot Street, Dublin.

1908 Moore, Robert F., M.B., 17, Bentinck Street, W.

1909 Morrell, Reginald A., 13, Harley Street, W.

O.M. Morton, A. Stanford, 133, Harley Street, W. (C. 1886-9. V.-P. 1902-5.)

1896 Mott, F. W., M.D., F.R.S., 25, Nottingham Place, W. (V.-P. 1904–7.)

ELECTED

1910 Mould, Major G. T., I.M.S., 4, Edith Road, W. Kensington, W.

1908 Moxon, Frank, M.B., B.S., 56, Seymour St., W.

1909 Naylor, W. Roebuck, 14, Carr Road, Nelson, Lancs.

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

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

1910 Nordenson, Dr. Erik (F.), 10, Wasagatan, Stockholm, Sweden.

1912 Norman, Alfred Clarence, M.D., Durham County and Sunderland Eye Infirmary, Sunderland.

1895 *Ogilvie, F. Menteith, M.B., The Shrubbery, 72, Woodstock Road, Oxford. (C. 1910-12.)

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

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

1911 Oliver, M. W. B., M.B., 67, Wimpole Street, W.

1914 Oliver, George H., D.O.Oxon., 284, Manningham Lane, Bradford.

1899 Ormond, Arthur Wm. 7, Devonshire Place, W. (C. 1910-12).

1892 †Orr, Andrew William, M.D., 71, Wickham Terrace, Brisbane.

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.D., 15, Queen Anne Street, W.

1890 †Palmer, L. Loran, M.D. (Address uncommunicated.)

1894 *Parker, Herbert, Saragossa House, New Street, Henley-on-Thames.

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

1900 Parsons, John Herbert, 54, Queen Anne Street, W. (C. 1909-12).

ELECTED

1914. Pascheff, Constantin, M.D., 151, Rue Rakowsky, Sofia, Bulgaria.

1887 †Patel, D. H., Bai Hirabai, B. J. Charitable Dispensary Tardeo, Bombay.

1907 Paterson, James V., 11, Melville Street, Edinburgh.

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

1902 Paton, Leslie, 29, Harley Street, W. (C. 1912–15.)

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

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

1889 †Perry, Francis F. (Address uncommunicated.)

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

1910 Pisani, Lt.-Col. L. J., I.M.S., 49, Wimpole Street, W.

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

1903 Pollock, W. B. Inglis, 21, Woodside Place, Charing Cross, Glasgow.

1896 *Pooley, G. H., 70, Hanover Street, Glossop Road, Sheffield.

1894 †Pope, R. J., M.D., Box 497, G.P.O., Sydney, N.S.W.

1900 Pope, Thomas Henry, M.D., B.Sc., Price's Avenue, Margate.

1902 Potter, Bernard E., 58, Park Street, W.

1903 *Potts, George, Bower Cottage, Tonbridge Road, Maidstone.

1899 Price, Henry J., Maldon, Essex.

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

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.

1892 Pronger, Charles Ernest, Litchdon, Harrogate.

ELECTED

1909 Prowse, S. Willis, 706, Union Bank Buildings, Winnipeg, Canada.

O.M. Purves, W. Laidlaw, 20, Stratford Place, Oxford Street, W.

1911 Quick, Hamilton E., M.B., Northampton Place, Swansea.

1889 Ramsay, A. Maitland M.D. (V.-P.), 15, Woodside Place, Glasgow. (C. 1909-11.)

1899 Read, E. I., Government Medical Officer, 21, Queen's Park, W., Port of Spain, Trinidad, West Indies.

1881 †Reeve, R. A., M.D., 22, Shuter Street, Toronto, Canada. (V.-P. 1907-10.)

1891 Reynolds, Austin Edward, 15, Finsbury Circus, E.C.

1913 Riad, M., Kasr el Aino Hospital, Cairo, Egypt.

1897 Richmond, R., M.D., 29, Lingfield 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.

1914 Roberts, Miss Adeline Mary, M.D., 4, Nottingham Place, W.

1885 *Roberts, Edward, 23, St. John Street, Deansgate, Manchester.

1896 †Roberts, J. R., Major, M.B., care of Messrs. King, King and Co., Bombay.

1891 Robertson-Fullarton, Archibald Louis, M.B., C.M., Kilmichael, Brodrick, Isle of Arran, N.B.

O.M. Rockliffe, W. C., M.D., 17, Charlotte Street, Hull. (C. 1892-5. V.-P. 1900–3.)

1912 Rodger, Douglas, M.B., Dept. of Public Instruction, Brisbane, Queensland.

1898 Roe, Arthur Legge, 43, Pryme Street, Hull.

1898 Roll, G. W., M.B., B.C., 7, Upper Wimpole Street, W. (C. 1910-12.)

ELECTED

1890 Rolston, John R., 14, The Crescent, Plymouth.

1891 Roper, Arthur Charles, The Shrubbery, Exeter.

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., 3, Manchester Square, W.

1881 †Rudall, J. T., 61, Spring Street, Melbourne, Australia.

1895 Russell, J. S. Risien, M.D., 44, Wimpole Street, W. (C. 1900–3. S. 1903-6. C. 1906-9.)

1903 Rutherford, A. Freer, 19, Abbey Road, Barrow-in-Furness.

1884 *Sandford, Arthur W., M.D., 13, St. Patrick's Place, Cork. (C. 1896-9. V.-P. 1902-5.)

1900 †Sager, D. S., M.D., Brantford, Ontario, Canada.

1907 Scott, G. Affleck, M.B., Kingsland House, Ballarat, Victoria.

1888 Scott, Kenneth, M.D., 7, Manchester Square, W.

1892 Shannon, John Rowlands, M.D., 17, East 38th Street, New York.

O.M. Sharkey, Sir S. J., M.D., 24a, Portland Place, W. (S. 1885-8. C. 1888-91. V.-P. 1900-2.)

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

1911 Sheedy, Thos., 114, Lower Richmond Road, Putney, S.W.

1899 Sinclair, A. H. H., M.B., 5, Walker Street, Edinburgh.

1891 Sinclair, Walter William, 3, Arcade Street, Ipswich. (C. 1909-1912.)

1912 Smith, D. Priestley, M.B., 52, Frederick Road, Edgbaston, Birmingham.

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

O.M. Smith, Priestley, 95, Cornwall Street, Birmingham. (V.-P. 1887-90; 1898-1901; 1907-10. C. 1883-6. *Pres.* 1905-7.)

ELECTED

1907 Smith, W. Harvey, M.D., 173, Portege Ave., E., Winnipeg, Canada.

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

1911 Snell, H. Cecil, Moor Lodge, Sheffield.

1910 Snellen, Prof. H. (F.), Biltstraat, Utrecht, Holland.

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, Manchester Square, W. (C. 1900-2. V.-P. 1910-12.)

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.

1886 Stephenson, Sydney, M.B. (V.-P.), 33, Welbeck Street, W. (C. 1898-1901, 1904-7. S. 1901-4.)

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.

1913 Stewart, John Barbour, M.B., 20, Charing Cross Mansions, Glasgow.

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

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

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

1910 Straub, Prof. M. (F.), Van Baerlestraat, 2, Amsterdam.

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

1910 Sulzer, Dr. (F.), 3, Rue Troyon, Place de l'Etoile, Paris.

1904 Sykes, Walter, 31, Winckley Square, Preston.

ELECTED

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.

O.M. Tay, Waren, 61, Oakfield Road, West Croydon. (C. 1880-2.)

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

1899 Taylor, Inglis, M.B., 31, Cavendish Square, W.

1891 Taylor, James, M.D., (T.), 49, Welbeck Street, W. (V.-P. 1910-11. C. 1894-7, 1900-3. S. 1897-1900.)

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

1914 Terson, Albert, M.D., 47, Boulevard des Invalides, Paris.

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

1903 Thomas, R. Russell, 40, St. Andrew's Crescent, Cardiff.

1895 Thompson, A. Hugh, M.D., 36, Weymouth Street, W. (C. 1910-12.)

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

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

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

1911 Thomson, Eric A., M.B., British Ophthalmic Hospital, Jerusalem.

1905 Thomson, H. Wright, M.D., 3, Sandyford Place, Glasgow.

1898 Thomson, W. Ernest, M.D., 2, Somerset Place, Glasgow.

1883 †Tobin, William, c/o Messrs. John Tobin and Co., Halifax, Nova Scotia, Canada.

1900 Tomlinson, John H., "Belmont," Vicarage Road, Egham.

1904 †Tooke, Frederick T., M.D., 1, MacGregor Street, Montreal, Canada.

ELECTED

1895 Toombs, Herbert George, Cromwell Mansions, 193, Cromwell Road, S.W.

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.

1912 Traquair, H. Moss, M.D., 46, Melville Street, Edinburgh.

1905 Trench, F. P., M.B., 48, Chester Terrace, Eaton Square, S.W.

1902 †Trilokekar, V. S., 308, Lohar Street, Bombay, India.

O.M. Tweedy, Sir John, 100, Harley Street, W. (*Pres.* 1903-5. C. 1884-7. V.-P. 1891-4, 1905-8.)

1898 Tyrrell, F. Astley Cooper, M.B., 27, Queen Anne Street, W.

1883 Uhthoff, J. C., M.D., Wavertree House, Furze Hill. Brighton. (C. 1905-8. V.-P. 1908-12.)

1910 Uhthoff, Prof. W. (F.), 16 ap. Schweidnitzer Stadtgraben, Breslau.

1894 Usher, C. H., M.B. 3, Bon Accord Square, Aberdeen. (C. 1909-1912.)

1913 Viner, Geoffrey, M.D., 15, Devonshire Place, W.

1910 Wagenmann, Prof. (F.), Bergstrasse 80, Heidelberg, Germany.

1888 Walker, Cyril H., M.B., 8, Oakfield Road, Clifton, Bristol. (C. 1907-10),

1892 Walker, H. Secker, 45, Park Square, Leeds. (C. 1903-6.)

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

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

1914 Weeks, John Elmer, M.D., D.Sc., 46, East 57th Street, New York City, U.S.A.

ELECTED

1910 †Weihen, A. Wallace, M.D. (Address uncommunicated.)

1887 Wells, Arthur P. L., M.B., 83, Harley Street, W. (C. 1896-9.)

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.

O.M. *Wherry, G. E., M.B., Corpus Buildings, Cambridge. (C. 1897-1900.)

1895 Whitehead, Arthur Longley, M.B., 31, Park Square, Leeds. (C. 1912–15.)

O.M. Williams, R., 125, High Street, Bangor, North Wales. (V.-P. 1896-9.)

1888 †Willis, C. Fancourt, C.B., M.D., Col., I.M.S., Frith Manor, East Grinstead.

1914 Wilson, James Alexander, 4, Central Avenue, Cambuslang, nr. Glasgow.

1911 Wilson, S. A. K., M.D. (S.), 14, Harley Street, W.

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, 32, Beaumont Street, W.

O.M.* Woodhead, G. Sims, M.D., Dysart House, Luard Road, Cambridge. (C. 1894-5. V.-P. 1905-8.)

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

1910 Worton, A. S., M.D., 71, Harley Street, W.

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.

ELECTED.

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

1910 Yearsley, J. Herbert, "Giffnock," Christchurch Road, Bournemouth.

1912 Zorab, Arthur, 7, Carlton Crescent, Southampton.

RULES.

1. Object of the Society.—The object of the Society is the cultivation and promotion of Ophthalmology.

2. Meetings.—The Society shall hold a meeting each year; the sittings shall be held on two or more consecutive days. On each day there shall be usually two sittings. The date and place of meeting shall be chosen by the Council.

3. Constitution.—The Society shall consist of Ordinary and Honorary members. All medical practitioners whose qualifications are satisfactory to the Council of the Society shall be eligible as Ordinary members.

4. Officers of the Society.—The officers of the Society shall consist of a President, four Vice-Presidents, a Treasurer, two Secretaries and six other members, who together shall form the Council and manage the Society's affairs.

5. Election of Members.—Every applicant for admission to the Society shall send to one of the Secretaries a form signed by at least three members, who have personal knowledge of the applicant. Such application shall be submitted to the Council, and if approved the applicant shall forthwith be admitted a member.

6. Honorary Members.—The Council shall have the power of electing as Honorary members men of distinguished eminence in Ophthalmology, or in the sciences bearing upon it.

7. Subscriptions.—The Annual Subscription shall be 12*s.* 6*d.* payable in the month of October. No member whose subscription is in arrear shall be entitled to attend the meeting. Any member whose subscription is twelve months in arrear shall cease to be a member of the Society.

8. Election of Officers.—The Officers of the Society shall be elected yearly by Ballot at a Business sitting held during the meeting. A balloting list of the names recommended by

the Council for election shall be posted to each member two weeks previously. At the Business sitting twelve shall form a quorum.

9. Duration of Office.—The President shall not hold office for a period of more than two consecutive years. No other member of Council, except the Treasurer, shall hold the same office for more than three consecutive years.

10. 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.

11. Secretaries.—The Secretaries shall manage all correspondence, and shall keep the Minutes of the Society and Council. They shall notify to new members their election. They shall arrange with the President the order of proceedings at the meetings. They shall have charge of and shall keep a register of all papers communicated, and shall be the Editors of the 'Transactions.'

12. 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. The accounts shall be audited once a year and presented to the Society at the Annual Business Meeting. Two members of the Society shall be nominated by the President to audit the Treasurer's accounts.

13. Meetings of Council.—The Council shall meet as frequently as necessary during the Annual Meeting and at other times if specially convened. Five shall form a quorum. The President shall have a casting vote in addition to his ordinary vote. The Council shall decide upon all questions relating to the reception of communications, and shall have power to submit any paper to referees.

14. Vacancies of Offices.—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 the next.

15. 'Transactions.'—The Society shall publish a yearly volume of its 'Transactions' as soon as possible after the Annual Meeting. There shall be an Editorial Committee consisting of the President, the Secretaries and two other members of Council. A copy of the 'Transactions' shall be sent to every member whose subscription has been duly paid; and to each Honorary member.

16. Publication of Papers and Discussion.—No paper shall be accepted if it has been published elsewhere. 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, and a short abstract of all papers to be read before the Society shall be distributed to all members before the meeting. No report of the meetings of the Society may be published by members or others without the sanction of the Council.

17. Notice of Meeting.—Three months before the date of the Annual Meeting a preliminary notice of the meeting shall be sent to every member.

18. Visitors.—Any member of the medical profession may attend a meeting of the Society (except the Annual Business Meeting) on being introduced by a member of the Society.

The Library has now been transferred to the premises of the Royal Society of Medicine, 1, Wimpole Street.

LIBRARY RULES.

1. The Library shall be open at the same hours as that of the Royal Society of Medicine, viz. from 11 a.m. to 6.30 p.m. daily, except Sundays.

2. Members will be entitled to read the books at 1, Wimpole Street, between these hours, or to take them out on signing a receipt provided for that purpose.

3. A large number of periodicals will be accessible to members in the Library. The current number may not 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 by special permission.

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

8. Members of the Ophthalmological Society who are also Members of the Section of Ophthalmology of the Royal Society of Medicine can have the full use of the Fellows' Library on payment of an additional guinea instead of the usual subscription of two guineas.

ADAMS FROST COLLECTION OF LANTERN SLIDES.

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

2. They may be lent, for the purpose of teaching, to teachers in any recognised Medical School on application to the Librarian.

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. The following are the regulations under which the slides may be borrowed:

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 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.

Resolution of Council, January 25th, 1906.

"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.

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. 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.

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.*

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REPORTS.

DISCUSSION ON OPHTHALMIC INJURIES IN WARFARE.

Opening Paper.

By Walter H. H. Jessop.

In opening this discussion on Ophthalmic Injuries in Warfare I have confined my remarks chiefly to those seen in connection with the present war on land. It is my good fortune to have associated with me in this discussion Col. W. T. Lister, who, in his position as Consulting Ophthalmic Surgeon to the Forces in France, has had unrivalled opportunities of seeing and observing the cases at the front.

At this early period of the war it is impossible to do more than chronicle cases of interest and suggest lines for further inquiry.

My own observations have been chiefly gathered from the wounded soldiers in St. Bartholomew's Hospital and also at Base Hospital No. 1 at Camberwell. My thanks are due especially for valuable information from Mr. Oliver, of No. 13 General Hospital in France.

The only statistics I have been able satisfactorily to collect are some in connection with the frequency of papillœdema in injuries to the skull.

Missiles.—It will perhaps be useful to consider first the

missiles and substances producing the wounds in the present form of warfare.

(1) Conical shaped lead bullets, which have mantles at the end of steel or cupro-nickel. This mantle may often be chipped, producing the small steel or nickel foreign bodies which may perforate the cornea or even embed themselves in its substance. The bullets travel as a rule at a high velocity, but may lose this by ricochet from a stone, which at times may break the bullet into pieces. The ricochet wounds are more serious than the direct.

(2) The shrapnel shell is a hollow metal cylinder filled with spherical iron, lead, or "mixed metal" bullets. It is composed of a body with a separable head—the time or percussion fuse. The wounds are produced by the bullets and by splinters of the casing. The splinters may be large or quite small, and may quickly lose their velocity so as not even to penetrate the cornea. The bullets have not nearly the velocity of a rifle bullet.

(3) The common shell is a hollow cast-iron or forged steel cylinder exploded by a time fuse made of copper or lately of aluminium, and filled with a charge of lyddite, guncotton, melinite, or gunpowder. It bursts into twenty to fifty pieces, and the wounds inflicted are, as a rule, very septic and very serious.

(4) Hand grenades—small explosive shells of iron, annealed glass, or even jam-pots filled with powder, bits of iron, pebbles, etc. They are only very dangerous at short range.

(5) Stones, earth, pieces of glass from the trenches, pieces of clothes, iron, copper, powder, are not at all uncommon and produce the same injury as any ordinary foreign body.

(6) Boiling pitch, petrol, etc.

Environment.—Next, as to the environment and its effect on the wounds caused by the missiles. This war on land has, up to now, been fought under somewhat new circumstances owing to the chief part of the fighting being waged in trenches.

This has resulted in a greater proportion of head and face injuries than in any other war owing to exposure of the upper part of the body.

The wounds have been very septic even from the commencement, owing to contact with the soil in the trenches —the fragments of a burst shell carrying with them soil, as may also a ricochet bullet.

Orbit.—Projectiles as a rule enter the orbit from in front, but may pass transversely or from below. If from above, the man is generally in the prone position. The wound caused by a bullet travelling at moderate or high velocity entering the orbit from in front is always serious, and in nearly every case causes destruction of the eyeball and perforates the wall of the orbit, frequently passing through the cranial cavity.

There are a few instances of spent bullets entering the orbit and remaining there, without bursting the eyeball, but these are probably ricochet. These cases often show intra-ocular hæmorrhages, choroido-retinal changes and detachment of the retina, which is generally complete. The only case I have seen where a bullet did damage to the orbit without affecting the interior of the eyeball is the following: The bullet had passed through the right superior maxillary bone and back of nose and lay in the nose, the apex of the bullet pushing in the inner wall of the left orbit near the superior internal angle without penetrating the bone. The only ocular damage was interference with the pulley of the superior oblique and consequent diplopia. Mr. Rose extracted the bullet from the nose, but there is still homonymous altitudinal diplopia.

Perhaps the most distressing and terrible ocular injuries, caused generally by enfilading fire along the trenches or from a sniper, are the transverse bullet wounds passing through both orbits, and, as a rule, destroying the vision of both eyes. In this war these cases have been very prevalent and must total already at least fifty or more. At Waterloo, Dr. Thomson observed eight to ten cases,

and in the Franco-German War of 1870 the Germans give twenty-eight. These transverse orbital wounds seem to bear a high proportion to the antero-posterior orbital wounds. This is probably only due to the fact that they do not pass through the brain and are rarely fatal. A bullet passing through the orbit from the front and entering the cranial cavity may often cause immediate death.

Of course, in orbital injuries from bullet, shrapnel, or shell, a skiagram should be taken to find out or localise any foreign body present. It is, however, better not to probe or investigate a bullet wound, even if it has perforated the cranial cavity. If a wound has been made in the orbital roof by a bullet the brain substance will tend to fill it up, and as one cannot prevent sepsis it is better to leave well alone.

Bullet and shell wounds of the eyeball are always serious, and practically in every case cause complete destruction of the eye—the sclera being torn to ribbons and the eyeball completely disorganised. Owing to the damage done to the orbit and eyeball there is generally profuse intra-orbital hæmorrhage producing proptosis, and the tissues quickly become very œdematous and septic. In such cases it is best to foment the wound and keep it as clean as possible with sodium chloride solution, and remove the eyeball when the conditions are not so inflamed, always bearing in mind the risk of sympathetic ophthalmitis if the other eye is a good one. In the Franco-German War no less than forty-two out of ninety-seven cases of sympathetic ophthalmitis were due to a badly damaged eyeball.

If both eyes are destroyed it is generally best to wait till the danger of sepsis is over or much reduced before removing the destroyed eyeballs for fear of meningitis.

In treating these cases of ruptured globe from a bullet, etc., it must be remembered that the contents of the orbit are generally septic, and care must be taken in excising the eyeball or the remnants of the eye. Whether the

optic nerve, with a portion of sclerotic, is left, or the nerve is divided, a drainage tube should be inserted for at least forty-eight hours, and no great pressure should be put on by cotton-wool and bandage. Col. Lister has wisely suggested an operation in which a portion of the sclerotic round the nerve is left, and by that means the nerve sheath is not opened. The details of the operation are :

(1) Eviscerate the contents and if possible pack the globe with gauze.

(2) Dissect back the conjunctiva and Tenon's capsule as in an ordinary excision and remove the gauze. Then remove the eye by dividing the sclera in front of the optic nerve so as to leave a mushroom-shaped piece of sclera.

After excision of the damaged globe the conjunctiva and contents of the orbit often remain very œdematous and septic for weeks or months, preventing the introduction of an artificial eye.

I have never seen damage to the eyeball by "windage," though such cases have been described.

Eyelid injuries are very common, and are almost always accompanied by injuries of the eyeball. As a rule the lids become very œdematous and swollen, and, in consequence, care should be taken not to put in sutures.

Conjunctivitis has not been very prevalent in this war, and I have not seen or heard of any special form.

Cornea.—Perforating wounds of the cornea are frequently met with, produced by minute pieces of metal casing, etc., and a skiagram should be taken as soon as possible, and the foreign body accurately localised by Mackenzie Davidson's method. If of iron or steel they may be extracted by the magnet, but the ones I have seen have been nickel from bullet mantle or shrapnel covering.

The following case is one in point :—

A private was hit by a bit of shell or spent bullet on the left side of the face and head, but did not know his eye had been struck. Five days afterwards he complained

of discomfort in the eye, and on examining it I found a very small grey spot on the cornea suspicious of a perforating wound. A skiagram was taken and showed a minute foreign body in the eye in the ciliary region. On the eighth day after the accident the ciliary congestion increased greatly ; there was great pain in the eye which prevented his sleeping. The eyeball was excised on the tenth day after accident. A small piece (1·3 mg.) of non-magnetic bright metal, probably nickel, was found in the ciliary body.

In these cases it is often exceedingly difficult to decide whether the eye should be removed or not. Luckily one may safely wait six or seven days without running any risk of sympathetic ophthalmitis. Occasionally, although there is a mark on the cornea suggesting perforation, no foreign body can be found by the skiagram. These foreign bodies are probably grains of sand, etc., and in most cases the lens has been injured. The treatment is expectant, with perhaps removal of lens later on.

Spicules of metal, powder, dirt, etc., are often found on the cornea. If the foreign body is a splinter and penetrates deeply into the substance of the cornea it is sometimes very difficult to remove, and great care must be taken to operate under a general anæsthetic and to use a knife rather than a needle, as there is great danger of penetration into the anterior chamber.

Powder may be removed by fomentations, but one should not try to pick out small pieces such as dynamite grains.

Sympathetic ophthalmitis must always be borne in mind in gunshot injuries of the eyeball, and in consequence great discretion is needed in trying to save an eyeball. As the disease does not occur before seven or eight days, and very rarely before three weeks, there is always time to consider the case after arrival at a base hospital.

As evidence of its prevalence in such injuries the German official returns for the Franco-German War gave the high percentage of 55–56 in all cases of injury to the

eyeball. Of the ninety-seven cases of sympathetic ophthalmitis, fifty-two occurred within one year of the injury, and of these twelve occurred within three months. No less than forty-two out of the ninety-seven followed complete destruction of the eyeball, and only four were associated with a foreign body in the eye.

In the American Civil War out of 254 cases of destruction of the eyeball there were forty-one cases of sympathetic ophthalmitis, 16·14 per cent.

In this war as yet I have not seen or heard of a single case, and this must be due to the great care and discrimination in diagnosis and treatment, especially in the early enucleation of a ruptured globe.

Nerves.—I have only seen one case of wound by bullet implicating the ocular nerves. I append the notes, as it is a remarkable case and difficult to explain.

A corporal, under the care of Mr. Eccles, was shot on December 28th, 1914, by a ricochet bullet, which entered just above the left zygoma, about $\frac{3}{4}$ in. in front of pinna, causing considerable extravasation of blood into the tissues of the left parotid gland. There was a small perforating hole in the squamous portion of the temporal bone.

On admission to the 1st London General Hospital about seventy-two hours after being wounded, he was unable to elevate the left upper lid. When this lid was raised the eyeball was found to be rotated outwards, and could not be moved inwards, upwards, or downwards. The pupil was dilated and inactive to the reflexes. There was crossed diplopia; vision both eyes $\frac{6}{6}$. Ophthalmoscope: Optic discs, etc., normal. There was anæsthesia over the distribution of the ophthalmic division of the left fifth cranial nerve, including the cornea. He had therefore complete paralysis of the left third, fourth, and superior division of the fifth nerve.

A skiagram was taken, and showed six pieces of metal lying apparently near the sphenoidal fissure close to the cavernous sinus.

On March 21st, 1915: He can now raise his eyelid a

little by the levator palpebræ, and there is fair movement of the superior rectus, the pupil is still dilated and immovable, but sensation has come back to the cornea and over the area of distribution of the superior division of the fifth nerve.

Papillœdema.—Bullet and shrapnel wounds of the vault of the skull are generally depressed fractures, and I have been surprised at the frequent occurrence of papillœdema in such cases.

The papillœdema is, as a rule, not characterised by much swelling of the optic disc, though in some cases there has been 3 D. to 5 D. The œdema and swelling is practically limited to the optic disc and a narrow ring of effusion round it. The retina beyond this ring is apparently not affected. There are, as a rule, no hæmorrhages and no white masses of effusion and the vessel walls are clear and not hidden. The physiological cup may be narrowed, normal, or in some cases filled up. The colour of the papilla is a little pink but not very red. The vision, as a rule, is unaffected and the colour-sense and fields are normal. The whole appearances are those of œdema due to pressure and not those of inflammation.

I have myself notes of fifteen cases of this condition. In most cases, after decompression or trephining, the swelling of the disc subsided in five to six days, and in fourteen to twenty-one days all traces of the condition had disappeared. I have never seen any cases go on to atrophic changes, and the colour of the optic disc afterwards has not been markedly pale.

The one case in which the papillœdema lasted for two and a half months was a depressed fracture at the back of the skull, from a bullet wound on October 28th, 1914. Decompression was performed by Major Rawling on November 1st, when the left optic disc had 1 mm. of swelling, whilst the right disc on the nasal side had 1 mm. of swelling, and on the temporal side no swelling at all; this is evidently due to a dense band of connective tissue in the lamina cribrosa from the neighbourhood of the

central vessels towards the outer edge of the disc (Paton and Holmes, *Brain*, xxxiii, p. 394). Two other slight operations for removal of bone had to be done, and in consequence the papillœdema persisted till January, 1915, but on January 28th both optic discs had normal appearance. The vision was throughout $\frac{6}{6}$ in each eye.

In none of these cases could I find any relation between the site of the injury and the papillœdema as to its commencement or the amount of swelling.

Papillœdema is probably present in all cases of vault injuries, with increased intra-cranial pressure, and, as a rule, is not present without this pressure. On decompression or trephining the papillœdema quickly disappears unless there is some cause keeping up the irritation.

On the question whether a gunshot injury of the scalp with simple furrowing of the skull is accompanied by papillœdema I have no direct evidence. The impact of small bore bullets on the skull ever so lightly almost always produces a fracture.

Mr. Oliver kindly sent me three different times the percentage of cases of vault fracture in which he had observed papillœdema. At first the percentage was 75 in vault fractures; next, in thirty cases he found sixteen, or 53·3 per cent.; and, lastly, in forty-two cases the number was twenty-two, or 52·4 per cent. Of course these numbers are too few for statistical purposes. I think the percentage must be higher than 52·4, as changes are often so slight and evanescent that they can be easily missed.

In Mr. Oliver's twenty-two cases three had the regular classical "choked disc" appearance with great swelling of the disc, accompanied by many new vessels, white patches of effusion, and many hæmorrhages—the retina around the disc being very œdematous. These were not like the papillœdema of the others, but rather a true inflammatory condition with œdema—*papillitis*. Of the three cases, two were cerebellar abscess and died after operation, and the third one, too ill for any operation, and died without post-mortem examination.

These cases of vault injury show, I think, conclusively, that papillœdema is produced by intra-cranial pressure, as in such cases it is always present with this pressure. Relief of pressure by decompression or trephining is quickly followed by subsidence of the swelling and return of the optic nerve head to the normal.

Occipital lesions.—Fractures of the skull in the neighbourhood of the occipital lobes are often accompanied by hemianopsia, and if on the left side are associated with other visual defect symptoms. The number of head injuries in this war and the presence at the front of our Secretary, Dr. Gordon Holmes, and Col. Lister, leads one to hope great things from them on the subject of accurate localisation.

In the Russo-Japanese War, Dr. Tatsuji Inouye took the fields of 28 cases of occipital bullet wounds and found amongst them: 4 typical and 2 atypical right or left hemianopsia; 4 typical and 1 atypical inferior hemianopsia; 3 typical and 1 atypical superior quadantial hemianopsia; and 2 superior hemianopsia.

In vol. xxi of our *Transactions* there are notes of four cases of occipital injury with field of vision changes from the South African War—one case by Sir Anderson Critchett (p. 123) and three cases by Mr. Fisher (p. 127).

Sir Victor Horsley, at the Medical Society last February, detailed notes of another case.

The great point I want to emphasise is that, in all these cases, as in the two following, there was complete blindness followed by permanent field of vision changes more or less symmetrical in each eye.

Case 1.—A private, æt. 24 years, was in the 1st London General Hospital from October 9th to December 10th, 1914, under the care of Mr. Harmer. On admission he was semi-conscious, but twelve hours later his condition was almost normal except for a tendency to drowsiness. He was completely blind and very deaf. There was a small septic gutter fracture in the left quamo-parietal region above the base of the mastoid process.

On October 20th his condition had improved and the wound was explored under an anæsthetic and several small fragments of bone removed. It was then found that he had an intra-cranial abscess with local laceration of the meninges and brain substance. Afterwards the wound healed quickly and the general condition steadily improved.

The blindness lasted for ten days, and on passing off was followed by complete right hemianopsia. The vision returned to $\frac{6}{6}$ in each eye.

With the ophthalmoscope there was papillœdema of both optic discs, the swelling being 3 D. in each eye, but this has now completely cleared up.

Case 2.—A private with gunshot injury of head received on February 14th, 1915. Admitted to field hospital in Boulogne on February 16th: semi-comatose, complete paralysis left arm and leg, quite blind, could not see the flash of a match; both pupils equal. There was a large ragged bullet wound of skull in the parietal region and a wound not explored in the occipital region. He was trephined, two inch diameter bone removed; a great deal of blood clot and brain *débris* was exposed; the dura mater was freely incised and blood clot and blood stained fluid evacuated. Two drainage tubes were inserted. On March 3rd, he began to recover use of left arm. On March 7th, he was admitted to No. 1 London Base Hospital. The trephine hole is occupied by soft swelling.

Eyes: Ocular movements are good, pupils are equal and act normally. Ophthalmoscope: There are no marked signs of papillœdema, but both optic discs are a little obscured as to the edges.

Vision: Right eye $\frac{6}{9}$; left eye $\frac{6}{9}$.

Fields of vision: There is left symmetrical inferior quadrantal hemianopsia.

Ears: Is deaf to the ticking of a watch held quite close to right ear.

These cases of blindness after injury to the occipital region of the skull followed by hemianopsia, complete or

quadrantal, go far to prove v. Monakow's last theory of diaschisis.

The injury produces diaschisis or shock in these cases on the systems and groups of neurons anatomically or functionally correlated with the cortical neurons damaged or destroyed.

In these occipital cases the "initial" phase of the diaschisis is blindness due to the fact that the shock of the injury to the occipital area of the brain on the side of the lesion is transmitted by commissural fibres to the correlated occipital area on the other side. As in these cases each area affected presides over half the field of vision in each eye, the effect of shock on the areas on both sides is blindness.

After a time, depending on the condition of the brain, the shock tends to pass off, and the "regressive" phase is entered on. The correlated centre on the opposite side of the brain to the lesion recovers generally completely, and we are left with the "residual" phase. This in the above cases is "hemianopsia" complete or incomplete, and the resultant is generally permanent. In my cases the recovery from deafness also illustrates this theory.

Temporary blindness.—At the beginning of the war there were many cases of complete blindness following severe explosions, shell flash, especially from the "Jack Johnson" shells. The patients remained blind for different periods of time, rarely more than a week. The pupils were active to light and the condition was always bilateral. The ophthalmoscopic appearances were normal.

Some cases were deaf, and others, more rarely, dumb. In all my patients the vision returned completely. In one case on recovery of vision there was a well-marked spiral field for a time.

Three men had extreme blepharospasm, and were unable to open their eyes for days; in one patient, a Belgian, the condition continued for three weeks.

These cases were all due to shock, and in some cases

to neurasthenia, and even "subconscious" malingering. I feel sure, however, from carefully watching four cases, that there must have been some definite cerebral affection.

In the occipital lesion cases lately mentioned I called in to explain them von Monakow's theory of "diaschisis." In these cases there was no actual localised lesion of brain substance, but yet there was definite shock from the shells, which in some cases fell so close to the patients that they were knocked over by the explosion and rendered unconscious. Here we have a condition which may be "psychic" shock from violent emotion or "commotio cerebri" from mechanical causes as "windage."

The "initial" shock in these cases is loss of consciousness, which, passing off, leaves as "regressive" phase loss of sight, deafness, or even loss of speech, followed in most cases by complete recovery.

Opening Paper.

By W. T. Lister.

In reviewing the ophthalmic injuries in warfare, the outstanding features are their severity and the impossibility, in almost every case, of employing conservative surgery. If the eye is touched it is spoilt. On the other hand, there is a great deal of work to be done. Ophthalmic injuries are all too common, and even after considerable efforts have been made to concentrate them, many cases remain scattered, and visiting occupies a large part of one's time. Some experience of diseases of the eye is of use in deciding which eyes are dangerous and which are innocuous from the point of view of sympathetic ophthalmia; the detection of optic neuritis and the estimation of the amount of swelling is of assistance to those who have to operate on head injuries, while the careful taking of fields in occipital cases may lead to a

widening of our knowledge on the cerebral representation of the fundus. In addition, there are a great number of "walking cases," who are seen as out-patients, suffering from conjunctivitis, ulcers, refractive errors, or who are tired of their present mode of life and hope that a sufficiently imposing ocular complaint may lead them to more congenial surroundings.

One may group the cases as follows :

(1) Contusions.
(2) Wounds.
(3) Cases with a foreign body in the eye.
(4) Functional cases.
(5) Head cases.

(1) *Contusions of the eye.*—In concussion cases, so far as I have noticed, if the bullet does not go through the orbit itself, there are no concussion changes in the fundus; the orbital walls may be cracked or broken to some extent, so that hæmorrhage into the orbital tissues may occur and some orbital cellulitis supervene, yet the fundus shows no ophthalmoscopic change ; but if the bullet passes through the orbit, the globe not being ruptured, the usual dramatic concussion picture is seen, viz. large glistening white areas with gross hæmorrhages like blood-red clouds, retinal or sub-hyaloid, with usually some other smaller extravasations in the retina. Ruptures of the choroid are fairly common, and they may be star-like or arranged in definite groups, some concentric with the disc, others radiating from it.

The rest of the retina shows some pallor due to commotio retinæ. I do not know if it has been suggested before that commotio retinæ and concussion of the brain are comparable. The retina is, of course, originally part of the brain, and just as after concussion, quite apart from penetrating injury and inflammation, the brain swells, so, as the result of concussion, the retina swells and undergoes a change which is visible with the ophthalmoscope, and which we recognise as commotio retinæ. I think this is a great opportunity for investigating the

exact nature of the white retinal patches in these concussion cases, and for getting definite records of the restrictions of the fields, which are usually considerable, and for discovering whether such restrictions have a choroidal, retinal, or nervous distribution. During the short time available for watching these cases before they are sent to England, I have seen very little improvement take place in the fields. As the commotio retinæ clears up and any hæmorrhage into the vitreous is absorbed, so the vision improves, but so far as I have seen the loss of field corresponding with the severe changes is permanent.

It is curious that one comes across detachment of the retina so uncommonly, considering how frequently blows on the front of the eye cause this condition in civil life.

When the bullet travels nearer the globe, rupture takes place, and, as in civil practice, on the opposite side to that of the injury. One sees many cases of the cornea turned down as a flap, from the bullet passing through the side or back of the orbit.

An interesting form of concussion injury occurred in one case at the front of the eye. The bullet passed through the root of the nose and grazed the lids, but did not touch the cornea. In this case there was an area of very marked striate keratitis towards the outer side of the cornea, and opposite this area the pupil refused for several days to dilate. I am inclined to look upon the case as one of concussion of the eye, caused by the cushion of air in front of the bullet.

(2) *Wounds of the eye.*—If the globe is penetrated by the bullet or shrapnel it is ripped up from before backwards into numerous leaves, so that when removed the sclerotic resembles the corolla of a very much faded flower. The contents are often so completely eviscerated that the sclerotic may contain no trace of vitreous, retina, or choroid. When removing these ruptured eyes an obviously simple plan is to take hold of each petal of sclerotic with pressure forceps and to dissect off the muscular attachments after the fragment has been drawn

forward and made taut, otherwise some difficulty is liable to occur.

If there is orbital cellulitis or panophthalmitis I have found it a good plan to remove the cornea first, where this is not already ruptured, and to scrape out the contents of the globe, and, having divided the muscles, to cut the sclerotic far back, leaving a frill of about four or five millimetres in radius round the intact optic nerve. This procedure has the advantages that there is no danger of septic meningitis as the result of the operation, there is no shock due to dividing the nerve, bleeding is less than in the ordinary method, free drainage is afforded for the cellulitis, and healing is much more rapid than if the whole sclerotic is left, as after simple evisceration. I have sent a short note of this slight modification of the usual mode of enucleation to the *British Medical Journal*.

The great amount of laceration of the lids and conjunctiva, as well as deep infiltration of the tissues in many of the cases of septic bullet wounds of the orbit, must lead to a great amount of scarring, together with considerable contraction if not complete obliteration of the conjunctival sac. There will be ample scope in these cases for ingenuity in devising plastic operations to fashion new lids and to allow the wearing of a glass eye.

(3) *Penetration of the eye with small foreign bodies* gives rise to one of the most unsatisfactory groups of cases with which one has to deal. Many of the foreign bodies do not give a visible shadow with X-rays, and even if they do, practically all are non-magnetic; in my own experience I have not come across one which *was* magnetic. The chance, therefore, of removal, and of thus saving a useful eye by operation, is (so far as I know) only very remote. Information of any success in removing such foreign bodies, and the methods of obtaining it, would be very valuable, and welcomed by many surgeons.

The two chief factors which make the success of an operation doubtful are :

(1) The frequent presence of more foreign bodies than have been diagnosed.

(2) The foreign bodies are often friable and are liable to be broken when caught in the forceps, and portions may remain behind.

When a foreign body is proved by the X-rays to be present, or at any rate strongly suspected, *and the sight is bad*, one's course is clear ; but the dilemma occurs when *the sight is still useful ;* are we to sacrifice at the altar of the demon of sympathetic ophthalmia, or can we leave the eye so long as the sight lasts? What is the risk of sympathetic ophthalmia due to a foreign body in the eye? From personal experience and from conversation with others I am inclined to believe that even if the eye contains a foreign body, provided there is no prolapse or entanglement of uvea or lens capsule, the risk of sympathetic is *nil*, in spite of the teaching of text-books and tradition.

Though the eye will probably degenerate, yet useful vision may remain for a long time. I would therefore rather wait and see, or wait till the eye does *not* see, before enucleating, provided the congestion and irritation subside. Of course, if the vision deteriorates and signs of irritation do not abate, the eye must be removed. The complication of prolapse of uvea is a serious one, but if the prolapse can be removed freely and cleanly, so that there is no entanglement, again I am inclined to wait. On the other hand, if any entanglement remains which cannot be removed the eye should be sacrificed.

One feels very helpless when foreign bodies have penetrated both eyes ; here, our obvious course is to leave them alone while the present technique of extracting them is so imperfect.

The above views are put forward tentatively, this subject being one on which there is bound to be considerable divergence of opinion, and I feel that full discussion would be most valuable and may pave the way towards a commonly accepted line of treatment.

Foreign bodies in the cornea are often curiously trouble-

some to remove, and one comes across a great variety of substances driven into the substantia propria; from the cornea of one man I removed two small pieces of metal, a hair, and a portion of his neighbour's skull. I have found a Beer's knife the most useful instrument when the foreign body lies deeply embedded; a needle is not stiff enough. A definite cut down to the level of the foreign body is made with the knife, and then by turning the blade sideways it can be removed; the linear cut leaves less nebula than simply scratching with a needle. Where the foreign bodies are very numerous and there is no irritation one may surely leave them alone and treat them as if the cornea had been tattooed; the opacities caused by treatment will be greater than those due to the foreign bodies. These cases, though apparently so simple, need careful investigation. I have several times been mistaken in thinking that eyes were not severely wounded on first examination, for the iris may be regular, the photophobia and congestion may be slight, and only one or two grains may be seen in the cornea, and it is not till after full dilatation of the pupil with atropine that one finds that some minute grains of sand have been driven into the eye.

(4) *Functional cases.*—There has been considerable discussion over that interesting group of cases with impairment of sight resulting from shock due to shell explosions. Such effects may be produced whether the man be subjected to the fierce glare and heat of the explosion, whether he be buried by the earth hurled over him, or whether he be exposed merely to intense atmospheric disturbance, or even perhaps overwhelming mental strain. As is well known, the effects of such shock are wide spreading, affecting not only the special senses, but the whole nervous mechanism. So far as the eye is concerned, a variety of phenomena may be met with; *e. g.*, blepharo-spasm with or without loss of sight; blindness more or less complete; strange colour sensations, such as yellow mists, red fringes to objects, etc. A very common complaint is some degree of night blindness.

The ordinary functional defects associated with neurasthenia and hysteria may occasionally be found; but after the appalling experiences of so many it is indeed remarkable that more purely neurasthenic cases do not occur. The following example of nervous shock affecting vision may serve. I am indebted to Dr. Myers for the following graphic notes on a case I saw with him at Le Touquet:

"J. A. P—, while retreating after an advance, was kneeling down and trying to get under wire entanglements. He got hooked in the back and could move neither forwards nor backwards. Three shells burst close to him. One burst just in front and cut his haversack away and bruised his side, and he thinks it was this one that took his sight away. The one behind gave him a great shock; it was like a punch on the head without any subsequent pain. After the shells had burst he managed to free himself, but he could only see as in a mist. It hurt his eyes to open them, and they burnt him when closed. He opened his eyes occasionally to see where he was going. He was crying all the time and worrying about going blind, and kept thinking about all that had passed between the advance and the bursting of the shells, and even when admitted to the hospital he shivered almost constantly."

One heard that in some of these functional cases œdema of the retina has been diagnosed, and in others optic neuritis, but, so far, I have come across no cases with any external or ophthalmoscopic sign of disease or injury.

The patients in this group require specially firm, though sympathetic, treatment, for it is very important to remember that sufferers from real functional defect may degenerate into malingerers and that not very uncommonly.

(5) *Head injuries.*—The data given under this heading are derived mainly from the excellent notes taken by Dr. Gordon Holmes, for whose co-operation and stimulating enthusiasm I cannot be sufficiently grateful, and

in these cases the condition of the fundus was noted either by himself or by me.

When a bullet strikes or penetrates the head there is often papillœdema, and this may occur within a few days of the injury or it may come on later, according to its causation. Taking papillœdema in the broader sense to include not only those cases with swelling of the nerve, but also those with congestion of the disc, as shown by blurring and striation of the edge and fulness of the veins, we found that papillœdema occurred in slightly over 50 per cent. of the cases; of these two-thirds had merely blurring or swelling of less than 1 D.; the remaining one-third had swelling of more than 1 D.; these numbers closely correspond with the observations of Mr. Oliver, who is now working at No. 13 General Hospital at Boulogne. The cases in which the swelling was greatest happened to be those of cerebellar abscess, but more data are necessary before one is able to say whether marked swelling is of localising significance, but the above coincidence is worthy of note.

Hæmorrhages or exudations in these cases do not of themselves appear to have any diagnostic value; they are not commonly present, nor are they necessarily associated with the higher degrees of swelling, and they may even occur when the neuritis is subsiding. I would suggest, therefore, that their presence or absence depends more upon some local condition than on the degree of venous congestion or intra-cranial pressure. The papillœdema is due to increased intra-cranial pressure, and this pressure is caused by: (1) Swelling of the brain due to concussion, (2) swelling of the brain due to inflammation, (3) hæmorrhage on the surface of the cortex, (4) abscess, or possibly (5) serous exudation from the meninges. That the papillœdema may follow concussion of the brain, quite apart from inflammation of the brain, is shown by the fact that swelling up to 1 or 2 D. may exist with grazing wounds when neither the bone nor the dura have been penetrated, and this may subside without any operation being done.

From this important fact it is obvious that the presence of papilloedema does not point to the immediate necessity of operation, but it is increasing swelling, pointing to increasing intra-cranial pressure, which is of practical significance. For this reason frequent ophthalmoscopic observations should be made and recorded in these cases.

I have come across two cases where the papilloedema was asymmetrical, and it has happened that the greater swelling was on the same side as the greater headache, and, as the operation showed, on the same side as a large blood-clot pressing on the cortex. In view of the free communication of the cerebro-spinal fluid on the two sides, it is difficult to understand how a large blood-clot on one side should cause greater pressure down one optic nerve-sheath than down the other; there is also the fact that in the case of cerebral neoplasm the side of the tumour does by no means always coincide with that of the greater swelling of the disc; one does not, therefore, want to lay stress on the above coincidence, I merely mention the point in case further data should prove its clinical significance. It is striking how quickly and completely the swelling of the nerve may subside after the pressure has been relieved.

The last group to which I wish to draw attention is that of cases of *occipital injury*. These are of very great interest, and the study of them may throw important light on the cortical representation of the fundus, for surely, never before have we had such an opportunity of studying the effect of experiments on the cerebral cortex, though some criticism of the coarseness of the lesion is allowable. Dr. Gordon Holmes and I have been taking the fields of vision in a number of these cases. The facts are comparatively simple: (1) When the lesion occurs over the occipital cortex of only one side the result is either hemianopsia or homonymous quadrantic defect on the opposite side; (2) when the bullet passes through *both* occipital lobes there is at first complete blindness, as one might expect, but in several cases this gradually clears centripetally after an interval varying in different cases

from hours to days, giving rise to a more or less central scotoma with a full peripheral field. In others one may either get hemianopsia with partial homonymous quadrantic defect on the opposite side combined with a central scotoma, or a central scotoma with a marked tendency to hemianopsia. It would be expected that a one-sided lesion would cause hemianopsia, but with the present views of cortical representation it is difficult to understand why a bilateral lesion should cause a central scotoma and no loss of peripheral fields, especially when one considers that the central region of the field has a double cortical representation, while the periphery of the field has only a single representation. It seems impossible to theorise usefully on the subject at the present time, but it is conceivable that some modification of the present views of cortical representation may have to be made.

I feel that I must apologise for the superficial and scattered nature of these notes, and some of the views expressed are tentative and may require modification. I must plead as an excuse that one's time is very fully occupied, and owing to the cases necessarily being sent off to England at the earliest possible moment, observations on them can only be made for a short period.

Dr. Landolt

said this discussion had been most fruitful, and they were much indebted to their good friends, Mr. Jessop and Colonel Lister, for their excellent papers. He had been particularly interested in Colonel Lister's comparison between commotion of the retina and commotion of the brain, especially in view of the fact that the retina must be regarded as a part of the brain. The most interesting part of the papers, from his own standpoint, was that which dealt with lesions of the occipital lobe, and what might be learned from these cases concerning the intimate physiology of the brain. He would quote the

very sad case of one of the most esteemed of their officers. When a lead bullet struck a hard body it became deformed, and if it entered the tissues afterwards it did so with a jagged cutting edge. Such a bullet struck this officer, destroying the whole left half of the face and the left eye, tearing off skin and muscles. At the same time, so severe an infection was produced that the other eye would have to be sacrificed owing to the onset of sympathetic ophthalmitis.

Prof. DE LAPERSONNE:

I shall only mention the following observations:

(1) *Hemianopic scotoma.*—Bullet wound of the occipito-parietal region a little on the left of the median line; the soldier was lying down; he was trephined and had secondary hæmorrhages. When he recovered, complete blindness lasted several days; little by little the vision returned, and on January 20th, 1915, four months after the wound, the peripheral field of vision was limited and a hemianopic scotoma persisted.

(2) *Inferior quadrantal hemianopia.*—Lieutenant C—, wounded on September 8th (Marne battle). The bullet passed through the posterior portion of the skull, from the left occipital region to the right parietal region. Loss of consciousness during several days; when he came to himself he noticed that he could not see objects on the right side. In the course of the first examination a complete symmetrical hemianopia was observed; later the field of vision gradually extended, and in two months the constriction was localised in the inferior right portion of the two fields of vision; since then there has been no change.

(3) *Syndroma of Claude-Bernard-Horner.*—I have observed a very interesting instance of lesion of the cervical sympathetic. A soldier was injured by a bullet in the neighbourhood of the right posterior region of the neck. The following permanent symptoms were observed: Paralysis of the right median and cubital nerves; on the

same side, ptosis, enophthalmos, miosis; neuritis optica, with reduction of the vision. A skiagram was taken and showed a fragment of bullet on the level of the right lamina of the sixth cervical vertebra. It is therefore a case of paralysis of the right sympathetic due to a lesion of *rami communicantes*. Removal of the bullet and laminectomy of the sixth cervical vertebra were performed by my friend, Dr. de Martel; a few hours later the sympathetic paralysis symptoms had disappeared. The vision improved and the optic neuritis diminished.

Such are the few observations I would have had the pleasure of presenting at your meeting.

Capt. P. H. Adams:

My experience has been gained at the 3rd Southern General Hospital, Oxford.

The worst cases of injury from bullet wounds that have come under my observation have been those in which the bullet has entered the face on one side and come out through the eye on the opposite side, completely destroying it, whilst the eye on the same side as the entrance-wound showed in one case a rupture of the choroid, in another some hæmorrhage into the vitreous below, whilst in other cases it had escaped altogether.

But perhaps the most remarkable case of all was a young private who was shot through the right temple, the bullet emerging at the other temple. This man was only unconscious for about ten minutes after the wound was received. He suffered no pain or headache afterwards and the only symptom left was a hole at the macula of the left eye with corresponding small central scotoma. In one case of traumatic cataract with foreign body in the eye localised by Mackenzie Davidson's method, I was able to remove the lens and then a small piece of metal with a magnet; but in the majority of such cases the number of the foreign bodies is very numerous, the eyes being simply peppered with fine particles. These eyes

have kept irritable and practically sightless, and I have accordingly enucleated them.

I have seen several cases that have had their eyes peppered with fine particles from explosion of shells, many having penetrated deeply into the cornea, sclerotic, and in the episcleral tissue and lids, but without perforation of the eye. I have been struck with the quietness and freedom from pain and inflammation in these eyes. If any of the particles have been superficial I have removed them, otherwise have left them alone, and the eyes have quieted down completely, though the fine particles in the cornea give rise to a slight amount of dazzling in bright light and want of fine definition in seeing.

The case mentioned by Col. Lister of the man shot through the nose has been under my care since and is only now almost well. The epithelium over the damaged area of cornea remained very bullous, but has practically flattened down now.

What has struck me most, though, is the effect of shell explosion and constant strain in the trenches on the hypermetropic eye. Even the small errors became totally manifest, so that one gets vision such as $\frac{6}{12}$ or $\frac{6}{9}$ brought up to $\frac{6}{6}$ by + 0·5 D. sphere, and on testing them under a mydriatic one finds that this is their total hypermetropia. Many men with high errors, for example one with + 7 D., are practically helpless. I have found this condition in very many patients who have complained that their sight, which was previously perfect, has deteriorated since being at the front.

This question may also be one of importance as shown by the following case. The patient was a man, æt. 27 years, sent back from the front with the diagnosis "cataract," after having been knocked down by a shell. On inquiry he said his sight had never been perfect, but he had just managed to pass the test. On testing the vision I found it to be R. $< \frac{6}{9}$, L. $< \frac{6}{18}$, and after retinoscopy found he had some mixed astigmatism which, when corrected, brought him up to R. $\frac{6}{9}$ and L. $\frac{6}{12}$. The changes were

central radiating lines with a surrounding ring of dots, and were suggestive of congenital changes to me, but I should like to ask if anyone has observed lens changes following exposure to shell explosion, as I was in doubt whether the diminution in the sight was due to recent lens change or to the fact of his refractive error becoming manifest.

In shooting, too, I have had many complaints from hypermetropes, both from men back from the front and amongst men in the new army, three battalions of which have been quartered in Oxford. It is always the same story, the longer they aim the worse they shoot. If they can get on to the target quickly and snap off at once they are all right, otherwise they lose the sights and the target becomes blurred.

With regard to shell blindness, Mr. Jessop tells me that after the first concussion no lesion is to be found. I should like to know if these cases of blindness are due to shell concussion simply, and if the blindness is permanent; or is it only temporary, and, if so, how long does it last, and is there any way of distinguishing it? I have been puzzled by a man who, I am convinced in my own mind, was largely malingering. He stated that one eye was blind in consequence of a shell concussion. Yet the eye was absolutely normal in appearance, and the pupil reactions were normal. He could not be tested satisfactorily for binocular vision, because he had spasm and photophobia in the other eye. I am in doubt whether the alleged blindness is real or not. The blinking in the left eye had only come on recently. He kept his left eye tightly closed on attempts being made to examine it, though he permitted the right eye, the "blind" one, to be examined.

Capt. G. H. Pooley:

I have listened with the greatest interest to the very able opening papers, and have gained much useful in-

formation from them. Col. Lister's paper, containing the results of his invaluable experience with the forces overseas, particularly invites discussion. Those of us who, like myself, are attached to the big Base hospitals in this country, see more of the walking cases and less of the recent acute injuries. We see a great many trivial cases; these include those who would like to escape from the bonds of discipline by practising the art of Ananias, while others wish to make a very small complaint go a very long way. One man came all the way back from France because he had broken his glasses, which were for $\frac{1}{4}$ dioptre of astigmatism only. In some cases, old injuries of the eye, received before enlistment or when on the reserve, have required to be diagnosed from injuries due to service. One of these had an old ruptured choroid, due to a shot from an air-gun. Other cases have escaped the vigilance of the examining medical officer, such as one man who had had both lenses removed, and another who had large dense lamellar cataracts in each eye, in spite of which he had been promoted corporal—his vision was $\frac{6}{36}$, each eye.

Miner's nystagmus has proved a stumbling-block, as those who have it at all severely cannot see sufficiently well at night.

I have met with at least one case in which a very definite attack of iritis, with posterior synechiæ and much exudate on the lens, was attributed, justly, I think, to a non-penetrating blow on the eye by fragments of sand violently thrown by the impact of a bullet.

In at least two other cases, traumatic cataracts were caused by similar non-penetrating injuries.

I now propose to discuss Col. Lister's paper and follow his grouping.

(1) *Contusions.*—In most of these cases the bullet goes through the orbit, and the slower-moving shrapnel produces as much disintegration as the more rapid bullet, the least disturbance being caused by bullets at extremely long ranges. The changes have been so well described by

Col. Lister that there is no need for me to re-describe them. In one case, in which the bullet was lodged deep in the temporal fossa without, so far I could judge, having passed through the orbit, there was a typical patch at the macula, about twice the size of the disc. The change in the retina is similar to the changes seen in the spinal cord at an autopsy, after a bullet has struck the vertebral column without penetrating the cord. I saw more than one such case in South Africa, with a transverse area, changed in colour and consistence, full of petechial hæmorrhages. The cause is probably that the bullet, travelling at high velocity, communicates its velocity in the form of vibratory waves of equally high velocity to the surrounding tissues; these are sufficiently intense to cause molecular disintegration of tissues, particularly such as the brain and spinal cord.

These cases do not remain long enough in the Base hospitals to allow us to note the alterations in the shape of the fields taken from time to time. In such cases as I have seen some time after the injury I have noted the following changes: The retinal arteries are small; grey areas, speckled with branched pigment, are much more common, and have probably taken the place of what were formerly glistening white areas. I have never seen detachment of the retina in these cases.

(2) *Wounds of the eye.*—Col. Lister's description of these cases reminds me of a class of case I see in Sheffield, from the penetration of large fragments of steel, and associated with much bruising and swelling of the tissues of the orbit. My habit in these cases is to turn down a conjunctival flap nearly as far as the insertion of the ocular muscles, which are not exposed. I then divide the sclerotic just behind the ciliary body, and thus remove the cornea, iris, ciliary body, lens, etc., with a strip of sclera. I then carefully scrape out all traces of choroid and retina. In this way Tenon's capsule is not opened; there is excellent drainage, and the stump moves admirably; there is very little freshly-cut tissue to become

affected. In suitable cases the conjunctiva can be loosely held together by a fishing-gut suture, which is put in at the inner side, taken two or three times through the conjunctiva like a post-mortem suture, and brought out at the outer side, the ends tied loosely together. It is easily removed by pulling on one end—it can be left quite loose. I find this a most useful procedure, and now use it more often than any other operation of the kind. Patients can usually leave hospital within a week of operation.

When the velocity of the bullet is much reduced, the eyeball is not necessarily penetrated any more than it is by a pellet of shot in civil life. It is not easy to be sure that penetration has taken place, as is shown by the two following cases : In the first of these, a bullet struck the eyeball, did not penetrate it, and lodged in the antrum of Highmore, from which it was subsequently removed. In the second case, there was a grazing wound of the eye apparently without penetration, the cornea was hazy, and there was a moderate amount of circum-corneal injection. I removed this eye. A few days after its removal, there was a sharp attack of iridocyclitis in the other eye which completely recovered after treatment, which included neo-salvarsan. This was possibly a mild case of sympathetic ophthalmia.

(3) *Penetration of the eye with small foreign bodies.*— In this class of case I quite agree with what Colonel Lister has said. I have seen similar cases due to the bursting of blasting cartridges in civil life. Where the foreign bodies are very small, and non-magnetic, attempts at removal are most unsatisfactory ; the eye is far more tolerant than is commonly supposed of small foreign bodies, which I believe become encapsuled. I know of at least three cases in which magnetic foreign bodies have been retained in an eye for periods of nineteen years or more, at the end of which time I have successfully removed them. I have also known a large sharp piece of glass to be retained in an eye for many years ; two corners of it were protruding when I saw the patient. In

none of the cases was there the least suspicion of sympathetic ophthalmia. The ciliary body seems as tolerant of a foreign body as any other part of the eye. My practice now is carefully to clean the point of entry of any foreign body, using spirits of iodine to the sclera after cutting away all bruised conjunctiva and then to cover the wound with a conjunctival flap. If the eye is becoming quiet, and the tension not unduly low, by the end of three weeks, I leave the eye alone, otherwise I remove the eye.

I think I remove a smaller percentage of injured eyes than I used to do—that is, of those which are not disorganised. Several particles, so small as to escape detection at the routine X-ray examination, have occasioned attacks of circum-corneal injection a few months later. I have been able to remove most of those foreign bodies which were magnetic. If I was unable to remove the foreign body I should feel inclined to remove even an eye with good sight, the other having good sight, if these attacks were repeated at short intervals, were severe, or associated with K. P., otherwise I should not remove it.

Where the wound involves the cornea the main thing is to get accurate apposition of its edges, by removing any iris or lens capsule which prevents this. I regard the prognosis as less favourable when there is a leaking wound in the cornea than when the wound is in the sclera and involves the ciliary body and particularly when the lens is wounded and swells up. I believe the reason to be that the corneal wound has less power of resistance to late infection from the conjunctival sac than the scleral wound, when covered by a flap of conjunctiva, which resembles peritoneum in its power of resisting infection. In one case a man was struck on the inner side of his cornea by a fragment of nickel casing from a bullet. The fragment did not injure the lens, and became encapsuled in the sclera below, leaving a white spot on the retina, his vision returned to normal. I find stereoscopic photographs taken with a lead glass artificial

eye in the conjunctival sac, a most useful way of localising small foreign bodies.

Foreign bodies in the cornea.—I have found an old Graefe knife the most useful instrument for removing these, when deeply embedded, particularly when they are projecting through into the anterior chamber. I also make a definite incision.

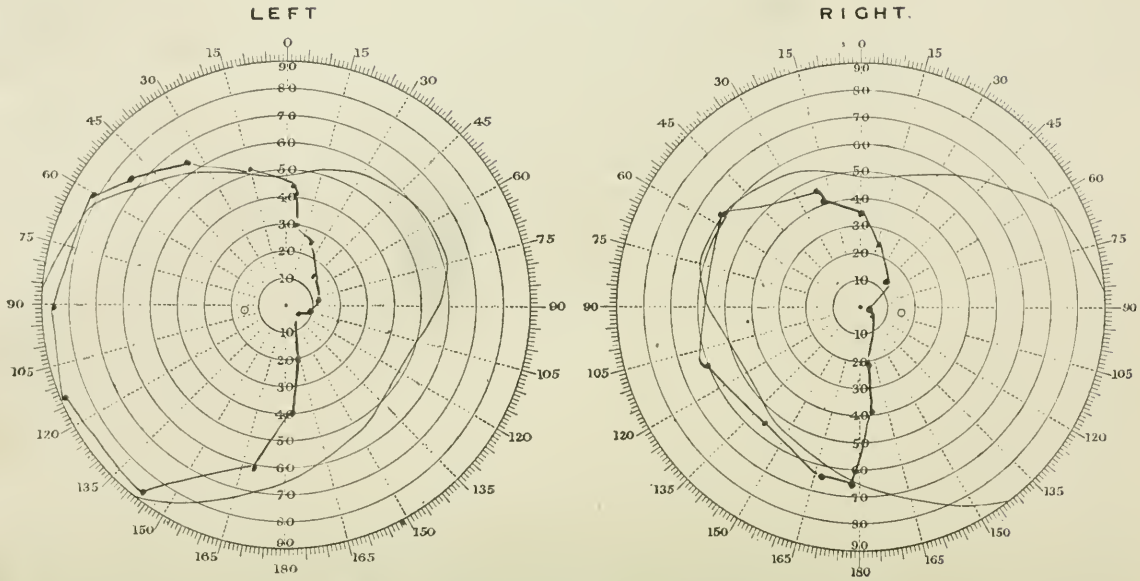
(4) *Functional cases.*—A large group of these cases closely resembles the cases of traumatic neurasthenia so frequently met with after industrial accidents. The most severe I have come across was in a man who had been blown out of a trench into a tree by an explosion.

Cases associated with blepharospasm and photophobia are the most liable to recur. One such case, who had been under the care of Mr. Jessop at St. Bartholomew's Hospital, after a shell fright in October last, came to see me in December, wearing glasses of an appalling tint of blue not supplied by Mr. Jessop's order. He has been on the borderland of melancholia. At first his vision was $\frac{6}{36}$, but he had no colour scotoma. He dosed himself with tobacco and produced a fine scotoma a little later. This has now disappeared again, and his vision is $\frac{6}{6}$ partly in one eye and $\frac{6}{12}$ in the other. He is still unduly depressed. This man was commended for his conduct and is a sergeant, to whom a commission has been offered. I do not think he will ever be fit for active service again. There has never been the slightest change in his retinæ.

The other cases have all recovered completely, and most of them have returned to duty.

In this class of case, does the subconscious mind gain an ascendency over the higher centres of control, in some cases more permanently and with a greater amount of self-suggestion, in other cases only temporarily, allowing the normal aspect towards life to be speedily regained? They may be divided into three groups: (1) Borderland insanity cases, who, although they realise that their ailments are entirely home made, and greatly wish to

FIG. 1.



Private C—, 20 mm. white, good light, April 12th, 1915.

Fig. 2.

Private S. T—, 20 mm. white, good light, April 19th, 1915.

recover, cannot cast off their brooding for more than a a little while.

(2) Cases, severe at first, which, in cheerful surroundings and with firm, but kindly treatment, quickly throw off their self-suggestion and recover their self-control.

(3) Cases in which the desire to recover is very slight or absent. These are usually not so severe at first, but will last indefinitely, so long as their surroundings are more comfortable than those to which they are not very keen to return.

After these, comes the group of wilful malingerers.

(5) *Head injuries.*—Not many of these have come under my notice owing to the distance from the port of disembarkation. In one, an extensive wound of both occipital lobes, with the removal of a good deal of bone from the back of the skull, caused temporary total blindness; he has now central vision varying from $\frac{6}{36}$ to $\frac{6}{18}$, not being quite constant from day to day, with homonymous hemianopia, a greater part being taken symmetrically from the lower than from the upper part of the fields, the seeing area forming a bulge above the macula in each eye (Fig. 1).

The other case received a wound on the left of the middle line, between the two parietal eminences. When I first saw him, his optic discs were normal; with the Maddox rod he had thirty degrees of convergence and two of hyperphoria. A fortnight later, when a yard of gauze had been removed from the cranial cavity through a small hole in the apparently healed wound, the convergence was reduced to six degrees, and the hyperphoria was gone. On taking his fields I found a symmetrical loss of the lower parts of both fields (Fig. 2).

Mr. Pooley added that he would like to know at what range the bullet was fired which passed through both orbits without destroying the sight. From what he had heard he would expect to learn that it was at very long range, the bullet having lost much of its initial velocity, so that it did not impart so much velocity to the tissues

struck, and there was very little vibratory motion. If any tissue took up the motion imparted by a swiftly moving projectile there was disorganisation of that tissue, with petechial hæmorrhages. He saw that kind of thing in the South African War, in injuries to the spinal cord. If a bullet struck any portion of the vertebral column at anything like short range, there was a transverse myelitis 1½ in. in extent opposite the seat of injury. At the autopsy, which many of those cases reached, the spinal cord was seen to be a greyish yellow, was altered in consistency and was full of small petechial hæmorrhages. He had not seen during this war the cases of lens concussion with fine changes such as Capt. Adams mentioned, but he saw one in civil practice following a very sharp blow. He was doubtful whether it was congenital; certainly it had not changed. In cases of "shell fright" he had found that the pupil reactions were normal. Malingering was a big subject, and he believed it would be even bigger yet.

Dr. George Mackay

related the following case as an illustration of an extraordinary escape from serious damage following a bullet injury.

Lce.-Cpl. G—, æt. 33 years, of the 2nd Battalion, 93rd Sutherland Highlanders, went out with his regiment to France and was wounded on October 23rd, 1914, at some place between Soissons and La Bassée. He was on the road-side when a shrapnel shell burst somewhere to his left and slightly behind him. One of the bullets struck his left temple. He was taken to a hospital forty or fifty miles from the firing line, and says that by the time he got there he was blind in both eyes. From the further history of the case I suspect that what he regarded as blindness was merely the shutting off of external objects by the swollen state of his eyelids. The swelling round the R.E. began to go down in about a week, and when

the lids were forcibly separated he could see with that eye fairly well. The L.E. did not open until after an operation was performed on November 23rd at the Military Hospital, Tidworth, by Dr. Sidney Boyd, who gave him the following certificate:

"This man was wounded by a shrapnel bullet in the left temporal region. The bullet was lodged partly in the orbit and partly in the left frontal sinus. It was removed from this situation by one incision along the supra-orbital ridge. The diplopia which was present before the operation has persisted."

(Signed) Sidney Boyd,
M.D., F.R.C.S., R.A.M.C.

The patient complained of slight deafness in his right ear, but had no other injuries.

I did not see him until January 20th, 1915, when, having returned to Scotland on furlough, he was referred to me by my friend, Colonel C. H. Melville, R.A.M.C., for advice as to the diplopia. By that time there was little to be seen at fault with the eye externally. Only a small scar in the left temple indicated the place of entrance of the shrapnel bullet. The extraction wound was well disguised by the eyebrow, and would not have attracted any attention. The left upper lid showed a trace of ptosis, but this was possibly accentuated by the fact that he was still wearing a patch over the eye to avoid diplopia. The lids could be voluntarily raised and firmly closed. There was no appearance of scarring either within or without the eyeball. The media were clear; the fundus was undamaged; the pupil and accommodation were unaffected. The vision of the injured eye was $\frac{6}{6}$ partly, while that of the uninjured R.E. was $\frac{6}{5}$. A slight failure in the upward movement of the L.E. was perceptible. In other directions the movements were good.

On looking directly forwards two images were seen, one a little above and in front of the other. The false image seemed to be the higher and further away. On looking to the left the false image moved further to the left.

With the rod test before the L.E. at 5 m., the streak appeared to be about 5 in. above the source of light, equivalent to about 1° of displacement. A prism of 1°, edge up before the R.E., or edge down before the L., practically corrected the diplopia in the horizontal plane. Tested with a candle flame the separation of the images increased as the flame went up, decreased as the flame was lowered. Separation was greatest when the light was held to the patient's left.

The conclusion appeared to be that the structure mainly involved was the *left superior rectus*. Of course, in an injury of this sort, the damage can scarcely be confined to one particular muscle or its nerve supply. The patient admitted that the diplopia was appreciably lessening, and I gave a pretty confident prognosis that it would entirely pass away. If it annoyed him he was to wear a 1° prism, edge down, before L.E. I have not heard of him since.

Capt. R. R. Cruise:

The use of the telephone in the localisation and extraction of metallic particles from any part of the body is only tardily being recognised by surgeons in general, although thirty-two years ago Graham Bell introduced this electrical appliance to our knowledge.

Sir James Mackenzie Davidson, to whom ophthalmic surgeons are so deeply and particularly indebted for his work on the exact localisation of foreign bodies in the eye by X-rays, has identified himself with the improvement and practical application of the telephone, in more recent times.

For a detailed account of the physics and other information on the subject I would refer you to his article in the *Lancet* of January 30th last; for the present it is sufficient if I state that a telephone of low resistance is used, one terminal wire is attached to a carbon plate, the other to a clip which can be attached to any metal instru-

ment desired, needle, probe or forceps. The carbon plate is strapped to the arm of the patient and the connection is thoroughly moistened with saline solution to ensure good contact. The needle searches in the tissues for the metallic foreign body, when contact is established a microphonic rattle is distinctly heard by the surgeon who has the receivers on his ears.

The first case I operated on was a soldier at the 3rd London Base Hospital, Wandsworth; Sir James very kindly came down, lent me his instrument and gave me invaluable assistance.

A piece of shrapnel had been located far back in the upper and inner part of the left orbit, the appliance was adjusted, and I plunged the needle into the orbit in the direction intimated by the X-ray plate; presently I heard the unmistakable rattle, I moved the needle about and estimated the size by the rattle area.

Col. Sir Alfred Pearce Gould who was watching, took up the receivers and his facial expression unmistakably revealed satisfaction at his auditory stimulation.

I then made a small ¾ in. incision in the upper lid which was very swollen and œdematous, substituting a sinus forceps for the needle and attaching it to the terminal of the telephone, I passed it into the orbit till contact was heard; with a little manipulation and guided entirely by auditory stimuli, I extracted a fair sized piece of shrapnel.

The result was extremely satisfactory, the orbital infection which is always a marked feature of shrapnel injuries rapidly subsided, and the patient left the hospital with his eye in his head—a damaged eye, but his own, not an artificial one.

This case was a very striking contrast to an almost identical one which had been referred to me by Sir John Bland Sutton, before I had had any experience of the telephone. As before, a piece of shrapnel had been located at the back of the orbit; the orbital contents were infected and œdematous. I made a fair sized

incision below the eyebrow and probed and searched with my finger, to no avail. Whether the particle had got beneath the periosteum or into the ethmoidal cells I do not know, but I never found it and Sir John counselled against too determined a search. The orbital swelling increased and the eye itself became infected, so that I had to remove it, and even then I did not find the shrapnel. The inflammation took a long time to subside and the patient now has a contracting socket. I have no doubt that had I been familiar with the telephone I could have discovered that infective piece of shrapnel, and credited myself with a better surgical result.

I have used the telephone on several occasions with complete satisfaction, in one case removing seven fragments of shrapnel from an orbit, and the patient retained useful vision in the eye, a result undoubtedly contributed to by the early removal of the infecting particles.

My experience hitherto has been limited to wounds in and around the orbit, I mean in contra-distinction to intra-ocular foreign bodies, but in a suitable case I should certainly attempt removal in the latter class of injury.

Sir James Mackenzie Davidson and myself performed numerous experiments on excised pigs' eyes by inserting metallic particles into the vitreous and attempting their removal through a scleral incision. In the case of minute particles the results were disappointing—firm contact is essential to produce a distinct rattle, and the fragments slipped away before the forceps; the vitreous also forms an insulating layer and prevents true contact, but in cases where a foreign body, the size of a No. 5 shot, is present in an eye, where panophthalmitis is not threatened, and where there is a reasonable chance of preserving some sight or even the eye itself, an operation for attempted removal by the aid of the telephone attachment to forceps is, in my opinion, justified.

An important point, as Sir James points out in his paper, is to have the forceps insulated except at the tip and the inner surface of the blades, otherwise one hears

the rattle when the outer side of the forceps is in contact with the foreign body, and grasping efforts are unsuccessful.

I do not know if many present are familiar with this appliance, but to such as are not I would like to emphasise the fact that in my own experience the telephone is no mere electrical toy, but a practical efficacious aid to surgery in general, and to conservative ophthalmic surgery in particular, and that by its guidance the surgeon can locate and remove, with the maximum of accuracy and the minimum of tissue disturbance, any metallic object concealed anywhere, from the proverbial needle in the bundle of hay—soaked in saline—to the foreign body lurking in the depth of the orbit and possibly the vitreous.

Mr. Jessop had referred to an instance of hemianopia or quadrantic loss of vision in a case of injury to the occiput. I have had two cases which have been extremely interesting; one was English, the other German. In the case of the Englishman the bullet entered at the right side of the parietal eminence and travelled backwards, emerging somewhere in the right occipital region. When I first saw the man in the 3rd London General Hospital, he had perception of light in the outer and upper field. That suggested that the lower and inner portion of his right retina, and the lower and outer of the left, were functionating. He could see hand movements. Sir Victor Horsley operated upon him, and opened up a gutter wound in the posterior cortex. There was, however, an infection from a gas organism, which produced cellulitis. I saw the man again later, and he had improved definitely, and could see the hand distinctly in the upper and outer quadrant. There is now some central vision, and I think the patient will go on improving. His cerebral condition has changed; he has become foolish, and he does not give good answers to questions. There is no malingering, but a subconscious deceitful condition. Last time he was examined he could see over a larger area and there was partial return of central vision.

The case of the German was more pronounced. His statement was that he was blind; his injury had been in the occipital region. He went through a phase of improvement. When the patient was last seen there was an exactly defined quadrantic loss of the lower and outer field to the left. He got $\frac{6}{6}$ vision, but the quadrantic defect was to be regarded as permanent.

It would be useful to hear the opinion of members who had had cases of miner's nystagmus, as to whether a man who had nystagmus was thereby debarred from service in the army? I have had three or four cases in which there was no nystagmus present, though the men declared they had had the condition. Some had vision of $\frac{6}{6}$. I have not had much experience of the condition, but I understand that in the case of a miner with nystagmus, if the man is taken from the mine and set to work above ground the nystagmus disappears. When the men I refer to looked straight in front of them the eyes were quiet, but nystagmus could occasionally be elicited by simulating the posture adopted in "getting" the coal in a shaft.

L. Vernon Cargill:

Comparing my experiences as ophthalmic surgeon to the Imperial Yeomanry Hospitals in the South African War with those obtained in this war from No. 4 General Military Hospital at Denmark Hill, the Wrest Hospital, the Dreadnought Hospital, and the Royal Eye Hospital, Southwark, the following differences are noteworthy. The proportion of ophthalmic injuries is much higher in this war in correspondence with the great preponderance of wounds of the head and face due to the type of trench and siege warfare.

As regards the missiles causing injury, in the South African War by far the greater number of wounds were caused by bullets, and comparatively few by shells, whereas in this war the very large number of eye injuries, as of other injuries, caused by shell fire has been an outstanding

feature. Again, in South Africa machine gun wounds were conspicuously absent. The dome-shaped or ogival tipped Mauser bullets in South Africa made much cleaner wounds of entry and exit, and in the majority of cases did less relative injury to the parts than the pointed German bullets of lower trajectory in this war.

The pointed bullets are more unstable in their flight and show a greater tendency to turn over or rotate on a transverse axis if they do not hit square, and this turning over has caused more serious injury to orbits and their contents, whether they have been involved either directly or indirectly by the bullet in its transit. The soft parts have been more contused, torn, and ruptured, and the bones more damaged. The bullet wounds of the South African War often healed extraordinarily rapidly and left trivial scars, and even bone penetration frequently left little evidence of bone injury, and they often did very little damage even when in fairly close proximity to the orbit. They did not show the same septic tendency as many have shown in this campaign, and probably less than 20 per cent. of bullet wounds in South Africa suppurated.

In the Boer War the range of rifle fire varied usually from 1000 to 2000 yards, and at that distance, although the velocity and spin of the bullet were considerably diminished, the velocity was still generally sufficient to maintain flight and prevent lodgment. The greater severity of present bullet wounds is due not only, as already mentioned, to their different shape, but also to the much shorter range of 100 to 200 yards, or less, at which range they have a greater expansive or explosive effect, as it is called, in passing through the tissues. Another difference is in the number of injuries from ricochet bullets, which were comparatively infrequent in South Africa, whereas to-day they are common. The ricochet bullet, becoming jagged or mushroomed before hitting, causes a serious wound, such as destruction of the eyelids and parts around to a greater or less extent; or if

the bullet is broken up, fragments of nickle mantle or lead core become scattered in the cornea, under the conjunctiva, into the sclera, and into, or even through, the eyeball.

In South Africa I saw very few cases of shell wounds implicating the eyes. In some of those where a man had been struck by a shell fragment about the region of the head, there was a history of loss of sight for half an hour or more after the blow, and I looked upon these cases as instances of concussion or commotio. The worst case I had of shell wound was where a fragment of a 5 lb. shell destroyed both eyes and caused considerable deformity of the face, the appearance of which was subsequently improved by suitable plastic operations.

In South Africa I did not meet with any example of the psychological condition of shell shock or traumatic neurosis with ambylopia, which has been occurring so frequently in this war. The cases of temporary blindness were of short duration, and had all received some head injury from a bullet or piece of shell, and they were, therefore, instances of concussion and not of traumatic hysteria or neurasthenia.

In neither war have I seen a case of arterio-venous aneurysm or pulsating exophthalmos. In South Africa I saw two cases of paralysis of the cervical sympathetic from gunshot wounds implicating the brachial plexus, but I have not seen any such case so far arising from this war.

With regard to the utility of the electro-magnet, I did not find it of any service in South Africa, and since the beginning of this war I have only had one case in which a fragment lodged in an eye was magnetic, and that was a comparatively large piece of shell lying at the back of the eyeball which had done such damage that nothing but enucleation was to be contemplated. In most cases the foreign bodies have consisted of fragments of bullet, either core or mantle.

I should like to take this opportunity of renewing my tribute to Sir James Mackenzie Davidson's cross thread

method of localising foreign bodies, which, as in South Africa, so in this war, has left nothing to be desired in the matter of accuracy when properly and carefully carried out.

Referring to the question of latent hypermetropia becoming rapidly manifest, I saw several cases in the South African War where good distance vision had, as the result of strain and stress, become much worse, the power of accommodation having temporarily failed.

As showing the mistakes which are made in passing recruits, I came across a man with a right amblyopic squinting eye, having vision of only $\frac{6}{36}$. He shot from the right shoulder and aimed at the object with the left eye by getting the foresight of the rifle in line with the object, so that the flight of his bullet would be well to the left of the object he intended to hit.

In the last few months I have sent back as quite unfit for service two or three cases of miner's nystagmus and one case with a complete detachment of the right retina.

I remember one unusual case in South Africa of concussion injury to the front of an eye, caused by a grazing bullet, where the pupil only recovered activity between five and six weeks after the accident. There was no permanent eye damage, and I think that Lister is correct in ascribing such an effect to the cushion of air in front of the bullet.

Capt. A. W. Ormond :

During the last eight months it has been my privilege to see a large number of men who have either returned from the fighting line or are training to go there. These have been seen either at St. Mark's College, Chelsea (No. 2 General Hospital), Guy's Hospital, or the Queen Mary Hospital, Southend. The total number has been nearly 1000, and I have selected some of these cases, those that appear to me to be of special interest, to recount to you.

I have seen three cases of traumatic amblyopia following

the explosion of shells. (1) H. S—, æt. 31 years, private, was present at the retreat from Mons, and after three days' marching and whilst under fire from the German guns he suddenly felt giddy and fell. He is very vague as to what occurred immediately before and for some time after, and was sent home in a more or less dazed condition. He told me that he only became fully conscious of his defective sight two days before I saw him, which was on September 18th, and that then everything appeared to be of a red colour. He was sent to me by his regimental doctor. On examination I found his vision to be only equal to the counting of fingers at a distance of a yard, and there was marked limitation of his fields of vision. Objectively I could find nothing whatever the matter; he had no wound of any kind, the pupils reacted to light, the optic discs and fundus oculi were quite healthy in appearance, and if it had not been that the man displayed great eagerness to be cured and to be allowed to rejoin his regiment I should have suspected him of malingering, having noticed among similar cases a marked disinclination to entertain the idea of returning to the front, and an effort to accentuate any existing defect. The patient was admitted to the ward and instructions were given that he should remain in bed for half of each day, and should have all the food he could take; injections of strychnine were administered. The sister and nurses were all quite convinced that his sight was defective: he, however, never injured himself moving about or ran into objects, although his fields of vision were not more than fifteen to twenty degrees in any direction. He had no error of refraction and Wassermann reaction was negative. He gradually improved and left the hospital in October, quite recovered, with full vision and full fields.

The second case, whom I only saw after he had recovered his sight, was sent to me as a case of concussion blindness with only partial recovery. On November 8th, at Zillebeke, he was knocked down and rendered unconscious by a high explosive shell, but received no direct injury in

the form of a wound ; after he had been collected by the ambulance it was discovered that he was quite blind and he remained so for seven days, after which his sight gradually returned. I saw him first in February ; his vision was then only $\frac{6}{24}$ with either eye ; he appeared to me to be quite healthy and I could find nothing to account for his defective sight until I noticed that he had a high degree of hypermetropia. With + 4 D. sph., his visual acuity was improved to $\frac{6}{6}$.

The third case is of interest, as up to the present time complete recovery has not taken place. G. L—, æt. 29 years, had been marched with his company to the top of a hill and told to rest there ; shortly afterwards the enemy found the range and a shell burst close to him, killing several of his companions. The man in relating his history was always, and not unnaturally, very indignant at what he thought was the inexperience displayed by his officer in allowing his men to rest on the top of a hill silhouetted against the sky, and his mental attitude was always one of dissatisfaction with his officer. I mention this because it has perhaps some influence on the progress of the case. Immediately after the accident his sight failed, but he was never totally blind. He was sent home on account of defective vision, and I saw him at St. Mark's about a week after the occurrence. The vision of his right eye was $\frac{6}{18}$, and the left $\frac{6}{60}$. His fields of vision were contracted, especially on the temporal side ; the pupils were equal and active and his knee-jerks were present ; the only objective sign I could obtain was a marked pallor of his discs. Although under observation for six weeks he did not improve at all, and it still remains a question whether his defective sight was really the result of the explosion or whether some atrophy of the optic nerve was threatening the patient irrespective of the explosion. I could never prove by any tests that his vision was better than he represented it to be, but he stated on one occasion that he thought his right eye had begun to fail before he went to the front, although later on he could not be got to endorse this.

I should like here to thank Mr. Parsons for his very erudite paper on this subject which he read before the Neurological Society last month, and I quite agree with him that these cases must be considered very carefully from the psychological point of view, as it is the mental attitude of the patient which alone enables the surgeon to distinguish these cases from those of malingering. In passing I might mention a case of a territorial whose life was probably saved by his glasses. On January 2nd, at Armentieres, he was standing talking in the trenches when a bullet travelling from right to left struck the left lens of his glasses, splintering the glass and grazing the skin of his face beyond the external canthus. The outer half of his left cornea had numerous scars, the result of the broken glass, and the mark on the skin could be distinctly seen. Had he not been wearing glasses the bullet must have entered his orbit and would probably have passed backwards to the brain.

The frequency of "explosion injuries," either from hand grenade, shrapnel, or high explosive shells, may be remarked. Small fragments of metal, gunpowder, sand and mud are scattered widely and with such force that many small foreign bodies are driven through the tunics of the eye. In some of the slighter cases these small wounds heal and in two or three weeks time the incident has been forgotten. At the beginning of the war, I saw two cases at St. Mark's of men sent back from the front as cases of iritis, which they undoubtedly were, but on investigation the character of the inflammation, the absence of any syphilitic history, and the inability to procure dilatation of the pupil with atropine, suggested that they might be cases of sympathetic trouble resulting from injury. Both eyes on X-ray examination revealed the presence of foreign bodies and the globes were of course at once removed. I do not doubt that had these cases been overlooked, sympathetic inflammation of the other eye would have occurred.

Another case, that of a Belgian soldier whom I saw at Southampton, might be recorded here. He had lost the

sight of his right eye owing to a bullet having passed through the orbit; there was no evidence of any damage to the sclerotic, but considerable disorganisation of the interior of the eye had occurred. The left eye appeared to have escaped all damage. Some weeks later he was sent to my friend Dr. Zorab on account of mistiness in his left eye, the vision of which was $\frac{6}{9}$. Dr. Zorab found slight vitreous haze and circumcorneal injection and the man undoubtedly had sympathetic trouble; he at once removed the right eye and a small punctured wound of the sclerotic close to the optic nerve was discovered, with a small bead of pus in connection with it. Dr. Zorab thinks the condition was due to a splinter of bone having penetrated the sclerotic and having been driven inwards by the bullet, as several sequestra have since come away from the sinus. With regard to the question of removal of an eye containing a foreign body I would strongly recommend that no general dogmatic statement be made on this very debatable point, as it seems to me that every case must be considered individually. For instance I saw a case at Southend on Sunday last in which a foreign body could be seen with the ophthalmoscope in the lens; this case had been seen by Col. Lister some weeks before and he had then decided not to remove the foreign body, and with his decision I cordially agreed because the man had useful vision. On the other hand, I have just removed an eye in which three foreign bodies were present although the initial inflammation had entirely subsided and the globe seemed to be free from any active trouble, but in this case the man had no useful vision and as his remaining eye was perfectly sound, it seemed to me that the risk to the man was too great to permit his keeping so dangerous an organ. If there should be any question at all of the presence of a foreign body I would not rely solely on the finding of the X-ray examination, because in several of my cases the foreign body was not discovered until a second or third photograph had been taken and sometimes not even then. I think that the history and the

nature of the inflammation as well as the amount of vision present should be considered when one is making one's decision. It is quite probable that during the next twelve months a large proportion of cases of sympathetic ophthalmia may be seen. A soldier in the fighting line with double buphthalmos must be, I think, a rather rare condition. A Belgian soldier whom I saw in the Queen Mary Hospital at Southend had been wounded in the foot, but the unusual appearance of his eyes caused the medical officer to ask me to see him although the man himself had not complained of his eyes. His vision was $\frac{6}{60}$ in each eye and was not improved by glasses; there was some irregular astigmatism due to slight opacities in the cornea; the anterior chamber was deep, the cornea 15 mm. in diameter, but the media were quite clear and there was no cupping of his discs. He has since rejoined the Belgian army.

Owing to reasons of administration a large number of those soldiers blinded in the war have passed through No. 2 General Hospital. I have records of twenty-eight such cases, short notes of which I have had printed and distributed. Thirteen of these men have no perception of light at all, and the sight of those who have vision ranges from a mere perception of light to the counting of fingers in one part of their field. Eighteen were the victims of bullet wounds, which for the most part traversed the front part of the head from side to side; four were wounded by shrapnel, one by a hand grenade, and the rest by explosion, the exact nature of which the men themselves are unable to give; one, however, was due to a bullet hitting the magazine of the man's own rifle which exploded in his face. One remarks the extraordinary malignancy of these indirect bullet wounds of the eyes; several of the men when looked at, now appear to be absolutely fit, often with both eyes intact and having a natural appearance, and the scar of the entrance and exit wounds are almost impossible to locate. Sir Victor Horsley states that the severity of bullet wounds is proportional to: (1) the sectional area

of the bullet, (2) its velocity, (3) the amount of fluid present in the medium through which the bullet travels; this last factor, I think, accounts for the extreme disorganisation of the globe found in many cases in which the eye has not been hit directly at all; the greater part of the vitreous chamber being occupied with soft glistening white masses of organised blood clot; in part showing also some red coloration and often entirely masking all details of the fundus. In a few cases the optic nerves themselves have been severed and in others definite rupture of the choroid and the retina can be made out.

I cannot perhaps do better than quote here some remarks made by Mr. Nettleship and reported in vol. xxi of the *Transactions*. "These indirect lesions are, it seems, to be explained by assuming that, at any rate, in the case of the soft tissues which during life are fluid, the force of the projectile is partly changed into vibrations of the tissue particles, which vibrations radiate in lines from the long axis of the bullet track so forcibly that, as Professor Stevenson of Netley puts it, 'they act as secondary missiles.' We must suppose that such lines of intense vibration impinging on the outer surface of the eye-ball, though unable to cause disintegration of the dense sclerotic, can pass through it with sufficient force to produce rupture of the much less firmly knit choroid and retina beneath."

Several men have lost the sense of smell in addition to their sight, and others have had extensive injuries to the jaw, maxillary antrum, nose, or skin of the face.

Of all the cases of injuries to the eyes that I have seen, including a large number in which one eye is only damaged, I have only had one case in which detachment of the retina was probably present, and that is in Case 2 on the list, so my experience coincides with that of Colonel Lister. I have also had only one case of cataract, which is, I think, remarkable considering the number of cases of injury to the eye both direct and indirect which I have seen. To those of us who are working in Base Hospitals it is a matter of the greatest regret that we are unable to

do anything to improve the vision of these blinded men, the damage is so extensive and of such a nature that it precludes any hope of improvement; two of the cases, 8 and 17, have improved slightly, to the point of seeing shadows, due, probably, to the effect of the absorption of the blood; on the other hand, two cases have lost the little sight they had, probably due to secondary changes, the result of contraction of the organised clot. A great deal of work has of course been expended in cleaning up the wounds, and curing septic discharges, which in some cases were very profuse, but the main work of the surgeon is now to devise various plastic operations to diminish the disfigurement and to enable the men to wear artificial eyes.

The psychology of these men who have been blinded is very interesting; with only two exceptions, and of these one is probably the result of damage to the frontal lobe, all of them exhibit extraordinary cheerfulness and contentment; one man to whom I expressed the opinion that he was very unlucky, as his eyes alone seemed to have suffered any damage and he had escaped all other injury, seemed only to be occupied in congratulating himself on being still alive, his remark being, "I think I'm very lucky, sir, as I might have lost my head," and another who not only lost both eyes but had as well extensive wounds of his face involving the whole of the maxillary antrum on one side, always replies in the most cheery voice in answer to a query as to how he is with the expression: "Oh, I'm in the pink, sir." I am quite sure that the arrangement by which these men are kept together from the start has been a great asset in combating the melancholia and depression which one would have expected to find. How long this mental phase will last one cannot say, but the main utility of the scheme which is being organised by Mr. Arthur Pearson at St. Dunstan's, Regent's Park, lies in the fact that before there has been time for a period of depression to supervene, they will have been rendered less dependent on external assistance

Short notes on twenty-eight cases of soldiers blinded during the present war, 1914–15.

By Arthur W. Ormond, F.R.C.S., Captain R.A.M.C.(T.).

Initials and No.	Age	Rank.	Regiment.	Nature of wound.	Place.	Vision.	Remarks.
1, S—	28	Sergeant	2nd Royal Scots Fusiliers	Transverse bullet wound through lower part of both orbits	Ypres, Oct. 14th, 1914	P. L. ?	Both globes are shrunken. Cornea contracted T-3 Anosmia, ptosis.
2, M. T—	18	Private	Irish Guards	Transverse bullet wound behind both globes	La Bassée, Dec. 30th, 1914	P.L. with right eye. *Nil* with left eye	Both eyes *in situ*. Retina and choroid disorganised. Vitreous chamber full of blood. Some retinal vessels seen $\bar{c}$ + 12 D. Left optic nerve severed. Left external rectus paralysed. Pupils inactive.
3, F—	36	Lance-Corporal	Leicesters	Transverse bullet wound through orbits	La Bassée, Jan. 27th, 1915	No P.L.	Right globe removed in France. Left optic nerve severed. Interior of globe disorganised.
4, H—	30?	Private	?	Bullet wound. Destroyed both eyes	—	No P.L.	Patient was very depressed, gloomy, and suspicious, and refused all offers of assistance.
5, R. O—	20	Private	Royal Fusiliers	Bullet entered right orbit and crossed to left side above the zygoma	Armentieres, Dec. 16th, 1914	No P.L.	Right eye removed at the American Hospital at Paignton. Left eye *in situ*. Optic disc white. Large rupture of choroid and retina at macula. Anosmia.
6, G. A—	20	Private	1st Chesters	Bullet traversed lower part of left orbit and behind root of nose to a point just in front of the right ear	Ypres, Feb. 8th, 1915	No P.L.	Extensive wound of the right maxillary antrum, right eye destroyed. Left eye has a hole at its lower part to which the lower lid is adherent.
7, G. C—	25	Private	?	Shrapnel	France	No P.L.	Was under Mr. Harrison Butler at Birmingham. The right lid was adherent to the shrunken globe. Left eye removed. Had extensive injuries to the root of his nose and face.

8, K—	23	Private	Royal Munster Fusiliers	Shrapnel burst in his face and bespattered him with small metallic pieces	Etreux, August 27th, 1914	Right eye P.L. Left eye can count fingers	No foreign bodies found in globes by X-rays. Both eyes *in situ*. Small prolapse of iris in left eye, which was removed. Vision slightly improved the last two months. Was seen by Col. Lister in France. Extensive scars on face.
9, K—	32	Private	1st Royal West Surrey	Transverse wound by bullet through both orbits	Ypres, Oct. 31st, 1914	No P.L.	Both eyes entirely destroyed. Typhoid fever developed after his return.
10, B—	29	Sergeant	1st Rifle Brigade	Bullet entered on left side 1 in. behind external angular process and crossed to right orbit, leaving at right external angular process	Ligny, August 27th, 1914	Can see light on outer side with left eye only	Was unconscious for seven days after injury, and does not know when right eye was removed. Left globe *in situ*. Large mass of organised blood on the inner side. Ptosis of left lid.
11. H—	20	Lance-Corporal	Sherwood Foresters	Shrapnel burst in his face	Lille, Dec. 10th, 1914	Shadows at 2 ft.	Left eye excised at Namur. Extensive wound right side of face as far as the ear, but the right eye remains *in situ*. Pupil reacts slightly. Large white mass in fundus.
12, J. B—	23	Private	3rd Royal Sussex	Shrapnel burst in front of him as they were preparing to charge with bayonets	Ypres, Oct. 30th, 1914	P.L. with right eye	Both globes are shrunken and have deep contracted scars. Jaw also fractured and tip of tongue lost.
13, H. D—	31	Private	3rd Rifle Brigade	Bullet hit the magazine of his own rifle and the whole thing exploded in his face	Lille, Oct. 29th, 1914	P.L. with left eye	Was under care of Mr. Hudson at Moorfields. The right eye was removed on account of pain. Numerous foreign bodies in shrunken left eye.
14, H—	25	Private	1st Wiltshires	Bullet entered left orbit and smashed through the root of nose into right orbit	La Bassée, Oct. 19th, 1914	*Nil*	Both eyes removed at Aldershot.

Initials and No.	Age.	Rank.	Regiment.	Nature of wound.	Place.	Vision.	Remarks.
15, L—	31	Private	Munster Fusiliers	Bullet entered occipital region, and he was operated on at the front, but no records came with him and he can remember nothing. Eyes are quite normal	? About Jan. 26th, 1915	Right eye hemianopia. Can see the outline of objects only	Patient was quite blind when first seen, Feb. 12th, 1915, he has since recovered some vision. Has extensive scars on the scalp all over the occipital region. His mental condition is slow and deliberate, but he is improving.
16, W—	19	Private	Scots Guards	Bullet entered the left orbit, passed through root of nose into right orbit	La Bassée, Jan. 21st, 1915	Counts fingers at 1 yd.	The left eye remains *in situ*, but has a corneal scar on inner side; the fundus of this eye shows rupture of choroid. Right eye destroyed. Smell not affected.
17, I—	—	Private	Rifle Brigade, 3rd Batt.	Bullet entered right orbit and left through maxillary antrum on left side. Jaw also damaged	Dixmude, Jan., 1915	Objects at 20 yds. with left eye	Right eye destroyed. Left optic nerve atrophic. Large masss of organised blood clot in vitreous chamber. Sight has improved a little lately. Anosmia.
18, L—	40	Private	Essex	Hand grenade exploded in his face. Whole face discoloured by gunpowder, etc.	Bizet, Feb. 16th, 1915	P.L. only	Right eye destroyed. Left cornea riddled with small pieces of metal. No red reflex obtained. Jaw broken and teeth knocked out.
19, W. G—	24	Gunner	R.G.A.	4·7 lyddite shell exploded owing to heat of gun just as he was going to fire it	Neuve L'Eglise, Mar. 10th, 1915	*Nil*	Both eyes entirely destroyed. Face very much burnt. Was blown into the air by the explosion.
20, T. G—	26	Private	King's Own Rifles	Bullet entered on left side, passing through orbit and above right orbit, probably damaging frontal lobe of brain. Had a depressed fracture also. Trephined.	—	Moving objects at 3 yds. below	Left eye destroyed. Compound comminuted fracture of skull on left side. Mental condition altered; very restless and irritable. Right ptosis. Vitreous chamber full of blood; red reflex above only.

21, C. V—	25	Belgian soldier	?	Bullet through orbits, which entered on the left side over the left brow and passed out in front of right ear	Dixmude	*Nil*	Left eye removed at No. 4 Base Hospital (King's College). Right eye *in situ*. Pupil fixed and inactive. Large mass of organised blood on outer and upper side of fundus; on inner side the choroid can be seen to be ruptured. Nerve probably severed.
22, G. S—	25	Private	North-amptons	Explosion? The man himself talks of an explosive bullet	Neuve Chapelle, Nov. 15th, 1914	Right eye can distinguish contrasts at 2–3 yds.; left eye P.L.?	Was operated on at Aberdeen, probably for removal of metal from the lids and orbit. Both eyes *in situ*. Right eye has hæmorrhage in the vitreous chamber. Sight of right eye is worse than it was. Right pupil reacts to light.
23, T. W—	32	Private	1st Hants	Bullet entered left eye, passing horizontally through the bridge of nose behind the right eye, the exit wound being 1½ in. behind external angular process on right side	La Bassée, Feb. 2nd, 1915	Doubtful P.L.	Smell not affected. Both eyes shrunken. Was under Mr. Jessop in No. 1 Base Hospital.
24, L—	31	Drummer	West Yorks	Bullet entered on the left side at level of apex of orbit and passed obliquely forward through right orbit	At the Aisne, Sept. 20th, 1914	Bare P.L.	Was under Dr. Clegg, of Manchester. Right eye removed in France. Left eye *in situ*. Pupil reacts; optic disc white. Extensive pigmentary changes at macular. Rupture of choroid and retina.
25, G. D—	26	Private	2nd Dorsets	Bullet entered on left side below orbit and passed obliquely upwards and outwards	Basra, Persian Gulf, Sept. 17th, 1914	Right eye sees shadows and contrasts in lower part of field	Both eyes *in situ*. Right eye, white mass of organised blood covering over the optic disc; otherwise media are clear. Left eye, vitreous chamber full of organised blood clot.

Initials and No.	Age.	Rank.	Regiment.	Nature of wound.	Place.	Vision.	Remarks.
26, E. C—	31	Private	King's Own Yorkshire Light Infantry	Bullet entered the right side of head above the brow and passed through to the left external angular process	Ypres, Dec. 21st, 1914	No P.L.	Both eyes were destroyed. Ptosis of left lid. Smell not affected.
27, N. P—	23	Lance-Corporal	1st King's Rifles	Bullet entered near malar bone on left side and traversed apices of orbits, leaving close to right external angular process	Aisne, Sept. 19th, 1914	Left eye can tell the time by a watch; right eye blind	Both eyes *in situ*. Right pupil dilated and fixed, but reacts consensually. Rupture of choroid at posterior pole with large mass of organised blood clot coming forward into vitreous. Left eye, fan-shaped rupture of choroid below and on outer side of optic disc. Has a para-central scotoma; sees best on outer side.
28, F. H—	32	Private	2nd Welsh	Was sniping and using field-glasses which he thinks heliographed his position, as he was shot by bullet which came through loophole and struck the glasses, driving fragments of metal into both eyes and nose.	Neuve Chapelle, April 1st, 1915	Counts fingers at 1 ft. with right eye	Large hole in nose going through to the nasal cavities. Right eye *in situ*. Pupil widely dilated, but reacts slightly. Vitreous full of blood. No details of fundus visible. Left eye deeply scarred and shrunken.

by being taught to be self-supporting, and to a greater or less extent self-dependent.

Leaving the subject of bullet wounds, I should like to mention a case of severe ulceration in the cornea which occurred in an officer. He had been in the trenches continuously for eight days without any opportunity of washing; he returned from the trenches to the head-quarters of his regiment and washed his face and hands, using a towel which had been in general requisition. He returned to the trenches again next morning after twenty-four hours, and returned again to headquarters to attend a court-martial. He did not wash then as he was rather late, but he complained at that time of his eyes smarting. He was seen by the medical officer, who sent him on an ambulance train to the hospital, where he was attended to by Lieut. Macrae, R.A.M.C., to whom I am indebted for the following notes: "Right upper eyelid was markedly inflamed, œdematous, and reddened; complained of photophobia. There was marked purulent discharge, the left eye was not protected in any way, and he was using the same handkerchief to both eyes. On syringing and opening the lids intense pain was experienced, and some ulceration at the inner edge of the cornea was noticed. The conjunctiva was very swollen around the cornea, which showed a slight general haze. The pupil was dilated with atropine, and a microscopical examination of the discharge showed the presence of gonococci, streptococci, and staphylococci. There was no history of any urethritis, and an examination of the genitals showed absolutely no trace of gonorrhœa. No pus-cells or gonococci were found on milking the urethra or centrifugalising the urine. Forty-eight hours later the cornea perforated and there was considerable relief from pain."

When I saw him at the end of January there was profuse purulent discharge and a small perforation of the cornea. This has since healed, and at the present time a large area of the cornea is clear. I propose shortly to perform an iridectomy, and I hope he will get some useful

vision. Another case from the same locality has since occurred, but fortunately it was suspected early, and seems not to have affected the cornea.

Amongst the cases that have been sent to me from among the men training in and around London, I have seen about half a dozen instances of miner's nystagmus, which had apparently been relieved, but which had re-developed under the strain of training; these men had all been considered unfit for foreign service. Two cases of retinitis pigmentosa with well-marked pigmentary changes in the fundus have also come to me; these are quite unfit for either home or foreign service.

I have only had one case of deliberate malingering, which was very easily detected, but a large number of men are inclined to exaggerate existing defects and require some reassuring that matters are not as serious as they would like one to believe. On the other hand it is pleasant to record that many men have given in their ages as anything from thirty-five to thirty-nine, whom I have found from their inability to see to read, notwithstanding the excellence of their distant vision, to be at least fifty years of age, but whose keenness and fitness make them in every way desirable soldiers. I have not enlightened the authorities on these facts.

Capt. Frank Thomas

said that what had struck him most in examining soldiers from the front was the condition resembling concussion of the retina. These cases showed all the symptoms of neurasthenic amblyopia which one met with in civil life. He had found it very difficult to do anything for them; they were tedious to test, and there was great difficulty in distinguishing them from malingerers. He was uncertain whether they were cases of neurasthenic amblyopia or were the results of concussion with retinal microscopic damage to the retina. He thought that in many the latter was the case. In the case of a man within five

yards of whom a "Black Maria" had exploded the retinæ showed superficial and deep hæmorrhages, and as they cleared up they left precisely this condition resembling neurasthenic amblyopia, *i. e.* extreme headache, contraction of fields, and diminished central vision. He saw one man whose eye and its muscles completely escaped injury, in whom the bullet entered the orbit, was located, and removed through the nose. That man exhibited the same conditions, contracted field and diminished central vision, but there appeared to be no anatomical damage in the retina. He had had under his care one man blinded immediately by a transverse wound from temple to temple. He never lost consciousness at the time he was wounded; he was shot and suddenly went blind; and he reported that he had had very little pain. He was at present being taught in Mr. Pearson's school in London, and was proving himself a useful picture-frame maker. Both optic nerves were severed.

Replying to Captain Cruise's remarks, he would not like to estimate the proportion of men he saw—but it was a large proportion—who had been recruited from among the miners of South Wales and who were the subjects of miner's nystagmus. His opinion was that they were absolutely useless for soldiering. Some of them could shoot well if they were standing upright, but immediately they shot from the recumbent posture or assumed a strained position they were useless. Moreover, in the dark many of them were blind. He did not agree with Mr. Pooley that all these cases were night-blind. A certain proportion found that they could not see in the twilight, but were better in the dark. He felt sure that even the men who appeared to have recovered and exhibited no oscillations were useless as soldiers; for the stress and strain at the front would result in their developing nystagmus again and their being sent home as unfit for active service.

Lieut. T. H. Townsend

said he had had some cases of miner's nystagmus, and they had invariably been referred to him, not by the medical officer of the unit to which they belonged, but by the sergeant-instructor on the range, as they were found to be useless for getting on to the target. But he suggested that there was a certain proportion of men in each battalion who need not necessarily be shots. Sixty-seven men in each regiment were detailed to do "donkey" work; it should not, therefore, be assumed that all men with nystagmus should be out of the service. In the Dover district, to which he was attached, the instructions were that no man should be sent out of the service for eye trouble unless his vision was less than $\frac{6}{24}$ in both eyes. They could be reserved for sanitary squads.

Lieut. Townsend desired to ask Mr. Jessop whether he considered that the amblyopia in shell concussion cases was possibly permanent, or only temporary. If a man were under his care for five months, suffering from concussion with supposed loss of sight, and if there were no fundus changes and the retinoscope showed no error of refraction, would Mr. Jessop be inclined to regard such a man as a malingerer?

Mr. A. L. Whitehead

said that working, as he was, in the 2nd Northern Hospital, he naturally saw many cases of nystagmus. He would rule definitely—and that ruling had been accepted by the War Office—that men in whom nystagmus could be elicited, as by making them stoop and look upwards, were not fit to be on active service in the firing-line. They were fit for home service and should be sent to their depôt.

With regard to traumatic neurasthenia, he had had one case following the explosion of a shell a short distance

away. That man developed very considerable spasm of his accommodation. He had, apparently, 5 to 6 D. of myopia, which, under homatropine, was reduced to 3 D., and entirely passed off under atropine.

In reference to the action of bullets, it had been pointed out to him by an expert in the matter that the German bullet was peculiar in that it had three distinct motions; an ordinary forward motion, a rotatory motion, and in addition, for a considerable part of its flight, a curious spiral motion. This last became spent after travelling a certain distance, so that thereafter the bullet went straight. If it struck in the earlier part of its flight it was this spiral motion which caused so much destruction of tissue. At long range the bullet would probably go straight through from one side to the other. It had been pointed out to him by his colleague, Sir Berkeley Moynihan, that when a bullet struck anything it imparted its momentum to all the tissues in the vicinity, so that when striking a bone, every splinter became in reality a projectile; hence there was a sort of "explosive" action.

Mr. Priestley Smith

showed what he supposed to be now an obsolete instrument—Nélaton's probe. He had himself used it very frequently and with advantage while working as a surgical assistant among German and French wounded in the Franco-German War. The probe was tipped with a small ball of rough white china. A bullet or fragment of lead at the bottom of a suppurating sinus declared its presence very clearly by the black mark which it made on the china.

Mr. Arthur Greene

said that in Norwich they were out of the area for miner's nystagmus, but he regarded the question of concussion amblyopia as one of great importance, because

what had been said showed that all these cases were not malingerers. In one instance there were voluminous notes written at "the front," and the pronouncement was that the case was one either of syphilis or malingering—the pupils did not react. There was contraction of the fields, and the man said his vision was getting better. He also saw two cases which were injured by the bomb explosions at King's Lynn. They had no concussion symptoms, but the injuries were by glass in the eyes.

Everyone did not seem to appreciate the importance of having an X-ray picture taken when there was a bullet injury, especially by a ricochet bullet. He saw a case which had passed through hospital without it having been recognised that there was anything wrong with the eye. The man was sent to him for an opinion as to whether he was fit to return to the front. There was a detached retina, and the man was completely blind in one eye. A skiagram was then taken for the first time, and he was found to have two foreign bodies in the eye. The eye being blind it was removed without anxiety.

A couple of days ago he saw the case of a man who had been hit in the nose by a ricochet bullet. The surgeon who sent him the case for "conjunctivitis" said he did not think there was anything in the eyes. But he removed a piece of lead which was sticking in the sclerotic, and an X-ray report showed there was also a foreign body in one eye.

He asked for a diagnosis in the following case. It was not one in which the bullet passed through the back of the orbit. The man had several head injuries caused by shrapnel. In the right eye he had a white atrophic-looking optic nerve, yet he could see well in the lower part of his field. In the left eye he had marked swelling of his optic nerve—papillœdema—but the vision and field were not much reduced. His own suggestion was that the man had fallen and sustained a fracture about the sphenoid, interfering with the nerves.

Colonel R. H. Elliot

said he had had over twenty years' experience with soldiers, and had constantly had injured men in the Madras Hospital (in the small wars that took place near the Frontier). He knew nothing more irritating than to have a man under observation whom one suspected of feigning disease; one had the feeling that one did not want to be got the better of by him, or that he should succeed in getting out of the Army on false pretences. Yet, with all the time one had to spend over such cases, he felt it was better that twenty men, if need be, should go out of the Army on false pretences rather than that a slur should be cast on one good man. A previous speaker had spoken of punishment for malingerers. In his, the speaker's, early days, he got two men a long sentence for malingering, and they admitted the justice of the conviction, but added that they did not think the trouble would have been taken to track them down. Nevertheless, the longer he went on with the work the more reluctant did he become to accuse a soldier of feigning disease. To bring punishment or disgrace to an innocent man would be a terrible thing to do.

Mr. Gray Clegg

said he saw one of the cases mentioned by Capt. Ormond in his paper. During several weeks the fundus was practically normal; then pigmented dots appeared at the macula and gradually increased in number so that eventually a very large pigmented area was observable. Possibly, therefore, microscopic changes took place in the retina from shock, and these might eventually be revealed by the ophthalmoscope. This might explain some of the cases which were now regarded as traumatic neurasthenia.

Mr. F. Richardson Cross

expressed the thanks of the Society to Mr. Jessop and Colonel Lister for the admirable papers they had con-

tributed, and which had been printed so that members might study them before coming to the meeting. He thanked Mr. Jessop particularly for the interesting way in which he had submitted the subject to the meeting.

The discussion had been very full and valuable.

Referring to the present discussion, he had been much struck with the number of men who had faulty right eyes, but could see well with the left. Something should be done with them. They could not shoot from the right shoulder, therefore they could not carry out squad drill under the sergeant; but all such men could be placed together and made a left-handed squad.

A practical point was that when one had a case of very serious shrapnel wound at the front, that case should be dealt with at once by the most careful antiseptic precautions and rigid cleanliness. It should not be left for three or four days, but should be immediately sent on for special treatment. No doubt many of the cases were lost owing to the dirt which was driven in from the trenches by the projectile. Hot fomentations brought to the area a great accession of leucocytes, which assisted the healing.

With regard to papilloedema, there were certain cases in which there was little more than slight œdema of the optic nerve-sheath. It was practically a normal nerve, but round it there was some swelling or pallor. Where there was a gross head lesion, especially with pressure signs, there would probably be papillitis with hemianopsia. The field of vision depended very much upon where the injury was. Hemianopsia might occur from damage to the optic tract or towards the occipital lobe. When the injury was at the calcarine fissure there was typical hemianopsia. If the damage occurred above there would be defect in the lower field, and if below the calcarine fissure the defect would be in the upper field. Such cases had clear central vision. When there were lesions of the occipital lobe further back, towards the angular gyrus, the case might be somewhat hemianopic, but there was loss of macular vision. These cases could not be put

into four or five classes—there were gradations. One of the most interesting class of cases was that in which there was absolute loss of vision, and he thought that was due to shock or damage to the occipital lobe far back. As Mr. Parsons was not present, he would mention a case of his which he, Mr. Cross, saw. An officer was shooting when a bullet struck his bayonet, or the end of his rifle, and destroyed one eye and injured the other, which became filled with blood. The first eye was removed. The other eye he saw later on with one of Sir James Mackenzie Davidson's pictures, and the whole orbit was stippled with tiny bodies; it was difficult to say whether they were in the eye or not; he believed they were not. He did not remember what the vision was, but the choroid was torn at several places. The opinion they both gave was that the man would retain useful vision in that eye. In the early stages of these cases the eye was full of blood, and later on it cleared up. Serious damage to the choroid might recover, provided the foreign body that caused it remained in or outside the sclerotic. He had known gunshot cut the sclera, cause severe intraocular hæmorrhages, remain imbedded on the outer surface of the sclera, leaving a white scar within the eyeball, while the sight returned almost to the normal.

Mr. Cross added that he had received a message from Surgeon-General Keogh to the effect, "Don't send us any cases of nystagmus." In action they might not only shoot badly, but to one side, and thus damage their comrades instead of the enemy.

As he had already mentioned, there were many men who had good vision in the left eye but not in the right eye. In drilling they had to use the right shoulder and eye for the rifle, but the right eye was no good for sighting. He believed several battalions could be formed of left-eyed men who could be taught to use the rifle with the left hand.

Reply by Opener.

Mr. Walter H. Jessop, in reply, said somebody had asked whether one or two horsehair drains would benefit the cellulitis by flooding the tissues with opsonines; also, was there any connection between Tenon's capsule and the œdema? He thought that in most of these cases there was such a connection, and that drainage was indicated, but he did not know whether opsonines would play a great part in the process. The best measure in these bad orbital cases was continual fomentation and frequent irrigation with sodium chloride solution. He agreed with Sir Almroth Wright that it was a mistake to put into such septic cavities antiseptics such as carbolic acid, which could not stop the septic process. The great object should be to increase the flow of lymph. Strong antiseptics had never been greatly in favour with oculists, who had long preferred saline solutions as lotions.

With regard to how long shell-blindness might last, he could not say definitely; some of his cases had lasted two months, but all had recovered. As regards other special senses, he might mention his own case. Ten years ago he had a motor accident, resulting in an injury to the back of his head. This was followed by absolute loss of smell and taste, which persisted for two years. He now had those senses as keen as ever.

Many of these cases of shell-blindness he believed to be due to concussion, and they could be explained on Von Monakow's diaschisis theory. Reports had been received of people having died from the concussion produced by the "Jack Johnson" shells without sustaining any external injury.

With regard to Mr. Pooley's case of iritis, he thought it was probably due to a septic fragment of bullet mantle or particle of sand or stone which had perforated the cornea Careful examination after the inflammatory symptoms had subsided would probably disclose some in the lens, too.

These cases of small particles were most difficult to treat, and often X rays afforded no help. In his own case, though the man had no eye symptoms at first, the foreign body was luckily found by the X rays.

Sir James Mackenzie Davidson's methods of localisation had been of the greatest use to oculists, and his adaptation of the telephone probe would greatly aid operative measures.

Mr. Cargill's remarks on the difference between the injuries met with in the Boer War and those seen at the present time were most interesting.

Mr. Ormond's reference to twenty-eight cases of blinded soldiers was very interesting. A case lately seen just escaped by extraordinary luck from being added to this list. A bullet passed through the zygoma on the right side, one inch behind the malar tubercle, and came out on the left side through the os planum of the ethmoid and the great wing of the sphenoid. The only ophthalmic injury was loss of vision in the upper part of the field of vision of the left eye.

With regard to the distance at which the bullet was fired in this particular case, he could not say definitely, but the Germans were only fifty yards away.

In reference to nystagmus, he had one case of a miner, apparently cured, who enlisted in the Army Service Corps. When driving a restive pair of horses the nystagmus returned, with the result that a fatal accident nearly occurred.

He was very much interested in Mr. Whitehead's remark about the spiral motion of the German bullets. Sir Victor Horsley had done many experiments on the physical effects produced by high velocity bullets, and he said that it made no difference whether they entered the tissues in the ordinary way or after having been reversed, as the German's were said to have done. But he, the speaker, thought that though the result was proportional to the sectional area there might be a difference, owing to the business end being nickel, whereas the other end was lead,

and if when reversed the lead split a dum-dum effect would be produced.

With regard to papillœdema in vault fractures, he had hoped to hear something from Mr. Paton. The papillœdema was present very frequently and was due to pressure from fluid in the nerve sheath. It might last only two or three days and then disappear. It vanished as soon as a decompression or trephining had been done if the intracranial pressure had been relieved. He had never known an injury of the scalp with only grazing of the external table to be followed by papillœdema.

In the fracture cases involving the occipital lobe there might be all sorts of changes in the field of vision—scotoma, hemianopsia, etc. He had hoped that a very interesting collection of fields would have been shown by Mr. Lister, but these would be published later.

He could not close his remarks without saying how much he regretted the absolutely unavoidable absence of Mr. Lister, who had contributed such an important part to the discussion. A letter received from him yesterday says: "The work here is pouring in. I pulled out a veritable 'beam' from a brother's eye yesterday, a piece of wood about one and a half inches long and about half an inch in diameter. In another case a big chunk of 'casing' from the back of an orbit, attacking it through the original opening at the side. I turned down a flap over the temple and chipped away the bone—a sort of modified Krönlein—and I think he is going to do well."

II. DISCUSSION ON DETACHMENT OF THE RETINA.

Opening Paper.

By A. Maitland Ramsay.

Time does not permit of my giving a *resumé* of the extensive literature dealing with the causation and treatment of detachment of the retina, but even were it otherwise such a detailed account is unnecessary, as the subject has been already fully dealt with both by Ernest Thomson (1) and by Casey Wood (2). For the purpose of this discussion, however, I shall in the first place briefly refer to the principal literary landmarks in the history of the disease, and afterwards give my personal experience of its treatment.

The older ophthalmic surgeons were quite familiar with the appearance of separation of the retina in the eyes they examined after enucleation, but it was only after the introduction of the ophthalmoscope that it became possible to recognise the existence of the disease during the lifetime of the patient. Very soon after that instrument was introduced into practice Von Graefe (3) gave a clinical picture of detachment as he saw it through the pupil, and his description, published in 1854, is so full and accurate that later observers have found little to add or to correct. Once the disease became known, clinicians attempted to classify the cases. They observed that it often followed an injury to the eye, that it was an early sign of an intra-ocular growth, and that it occasionally appeared in the course of acute general disease, more particularly in the nephritis accompanying pregnancy and scarlet-fever. These cases they separated from others,

which occurred for the most part in the short-sighted, but whose ætiology was obscure, designating the latter as idiopathic, and placing them in a large group by themselves. We now know, however, that detachment of the retina is not a clinical entity, but that it is always secondary or symptomatic, and although we still use the term idiopathic, we employ it only because our methods of diagnosis are not yet sufficiently perfect to enable us to discover the antecedent cause of the disease.

The ætiology of detachment of the retina has been the subject of much laborious research, but up till the present time no single theory is sufficient to explain all the clinical facts. This part of the discussion has fortunately been entrusted to those far better qualified to deal with it than I am, so I shall just say in passing that the different theories can all be classified into three main groups—the retraction theory of Leber (4) and Nordenson (5), the diffusion theory of Raehlmann (6), and the exudation theory of Arlt (7) and Von Graefe (8). To these there must also be added the new theory put forward by Vail (9), that there is a paralysis of the secretory function of the ciliary processes.

In every case, however, the cause underlying the detachment is disease of the uveal tract and of the pigment epithelium (Kümmel) (10), which in some instances amounts to nothing more than senile degeneration, while in others it is a more or less acute inflammation of the ciliary body and choroid. In this connection I would like to refer to a recent paper by Hertell on "The Dependence of the Intra-ocular Pressure on the Composition of the Blood," because I believe that as methods of investigation improve, research in that direction will enable us to arrive at a better understanding of the pathogenesis of idiopathic detachment of the retina.

Rational treatment of a morbid state invariably depends upon accurate knowledge of its cause, for it is only by a proper understanding of the pathological process underlying a disease that we can arrest its progress or effect its cure. Until the whole pathology of a disease is thoroughly

understood many different remedies will be advocated, or the most diverse surgical operations tried; and this is exactly what has happened in the treatment of detachment of the retina, for, unfortunately, as yet we seem to be a long way from knowing the primary cause of spontaneous separation. What we see with the ophthalmoscope, or find on an examination of an ordinary pathological specimen, is the result of a morbid process rather than the process itself; and it is, accordingly, only these results that can be dealt with by all surgical procedures for the cure of detachment.

Whenever it was known that there was a collection of fluid between the choroid and the retina, the most natural thing to occur to the mind of a surgeon was to evacuate it by puncturing the sclerotic. This, the oldest of all the operations, was first performed by Ware (12); but not till fifty years after was it definitely introduced into ophthalmic practice by Sichel (13). Since then many modifications of simple scleral puncture have been suggested. Wolfe (14) elaborated the technique of the operation and laid stress on the necessity of getting rid of all the subretinal fluid. Pagenstecher (15) advocated multiform puncture of the sclera with a steel needle. Galezowski (16) drew off the liquid with an aspirating syringe, and quite recently Parker (17) and Curtin (18) used a trephine to remove a disc of sclerotic in the immediate neighbourhood of the detachment.

The use of the ophthalmoscope revealed that in some cases of separation the retina was torn, and as those cases were supposed to follow a more favourable course than others in which rupture did not occur Von Graefe (19), who did not approve of scleral puncture, devised a method of tearing the retina with a needle, passed through the sclerotic on the side opposite to the detachment, thus making a way of escape into the vitreous for the subretinal fluid. With the same end in view Bowman (20) made a rent in the retina with two needles, employing them one against the other in a manner similar to that

in which he afterwards operated on the lens capsule. The Graefe-Bowman operation was on the whole unsatisfactory, and as it was sometimes followed by destructive inflammation of the eye it was abandoned. It has, however, been revived in a modified form in recent years by Lang (21), who evacuates the fluid in the ordinary way by puncture through the sclerotic and subsequently passes the point of the knife through the retina.

Unfortunately the satisfactory results that frequently followed immediately after scleral puncture were not lasting. The fluid reaccumulated and the separation recurred. In an endeavour to obviate that, De Wecker (22) tried to drain the subretinal space first by a strand of gold wire and afterwards through a small gold cannula. Galezowski (23) attempted to keep the retina in position by stitching it to the subjacent structures with a catgut suture, while Schöler (24), Abadie (25), and others sought to obliterate the subretinal space by injecting a solution of iodine in order to set up adhesive inflammation between the retina and the choroid. Both De Wecker and Galezowski soon adopted much simpler methods, but strange as it may appear the iodine treatment was practised for several years. Needless to say it ended in failure, and in at least one instance, reported by Gelpke (26), purulent choroiditis, meningitis, and death followed the intra-ocular injection of three drops of tincture of iodine.

Although the iodine treatment was ultimately abandoned other means to excite adhesive inflammation between choroid and retina were at once suggested. Abadie (27) punctured the sclerotic and choroid with a thin galvano-cautery, burning, instead of cutting, his way into the subretinal space, while De Wecker (28) and Dor (29) cauterised the bare sclerotic at several points and repeated the operation at regular intervals, taking great care not to perforate the sclera.

Electrolysis was advocated later on by Abadie (30), Terson (31), and others, and was practised with varying

success for a considerable time. In this country Snell (32) was the chief exponent of this method, and reported several cases he had treated. Iridectomy, too, a favourite method of treatment in many different diseases of the eye, had for a time a vogue, but it was ultimately abandoned, some of its former advocates turning so much against it as to say that it did more harm than good.

In 1895, Deutschmann's (33) classical paper was published, in which he described and strongly advocated the two operations now known by his name, and at the present time probably the most popular of all the operations for detachment of the retina. Although the technique is altogether different, the aim of the bisection operation is similar to the Graefe-Bowman procedure. Deutschmann not only makes a double communication through the retina between the subretinal space and the vitreous humour, but also divides the vitreous freely in order to cut any bands which attach it to the retina, the contraction of which, according to Leber, is the principal cause of detachment. After the operation, the patient is kept in bed for seven or eight days, and after the first day only the eye operated on is covered. Deutschmann emphasises the importance of repeating the operation over and over again. The second operation is reserved for cases in which the intra-ocular tension is greatly reduced owing to diminution in the volume of the vitreous humour. In those cases in addition to bisection, Deutschmann injects with a special syringe sterile vitreous prepared from the eyes of calves. The idea of adding to the bulk of the vitreous in this way had already been suggested by Grossmann (34), who injected sterile saline solution, and by Weber (35), who aspirated the subretinal fluid with a syringe and afterwards injected it into the vitreous humour. These operations can be performed with little risk provided aseptic precautions are strictly observed, but as absorption of the material injected soon takes place, the bulk of the vitreous can only be increased temporarily,

and any good effect it may have had in causing the retina to fall into place must be of correspondingly brief duration.

Müller (36) endeavoured to attack the disease by reducing the size of the eyeball. Iwanoff (37) had observed that in high myopia the vitreous body did not increase in volume in proportion to the distension of the chamber and became in part detached from the retina, the space thus formed being filled by serum. Müller, after having gained access to the temporal side of the eyeball by cutting through the outer wall of the orbit, resects a long strip of sclerotic at the equator of the globe, closes the wound with sutures and thereby reduces the size of the eyeball. He claims several successes, but I fear there are not many ophthalmic surgeons who would, with a light heart, advise and carry out such heroic measures.

Quite recently, Lagrange (38) has suggested superficial cauterisation of the sclera around the limbus corneæ in order to obtain after cicatrisation a band of fibrous tissue, which, by contraction, will hinder the escape of the intra-ocular fluids. He argues that diminished intra-ocular tension is as harmful in detachment of the retina as increased pressure is in glaucoma. He reports several cases in which he has performed the operation and records satisfactory results. The time of observation is as yet, however, too brief to permit of any definite judgment, but the procedure commends itself by its simplicity, and if the operation be carried out carefully it might quite well turn the balance in favour of recovery in a case of detachment in which there was great reduction of the intra-ocular tension.

From this rapid and necessarily incomplete summary it will be seen that the operations for the relief of detachment may be readily divided into groups according as their primary aim is to evacuate the subretinal fluid, to open a communication between the subretinal space and the vitreous humour and to divide any bands of connec-

tive-tissue which are pulling on the retina, to excite adhesive inflammation between the retina and the choroid, to increase the volume of the vitreous humour, to diminish the size of the eyeball, or to increase intra-ocular tension. In none is any attempt made to reach the source of the disease, and many of them aim at establishing a condition of matters that is quite abnormal. That applies specially to those in which an attempt is made to seal the retina to the underlying choroid. Apart from the fact that Scheffels' (39) experiments on rabbits prove that the application of the galvano-cautery to the sclerotic causes detachment of the retina, the procedure is unsupported by what is known either of physiology or of pathology. In the normal condition there is no organic union between the retina and the underlying structures except at the optic nerve entrance and the ora serrata; at all other parts it is kept in position by the pressure of the vitreous. It is true that the rods and cones dip into and are partially sheathed by the filamentous processes of the hexagonal pigment cells, but the union is of the slightest and when separation occurs the retina strips from the pigment layer, which remains closely attached to the choroid. From the pathological side, on the other hand, it has been demonstrated both by Elschnig (40) and by Treacher Collins (41) that a patch of adhesive choroido-retinitis does not prevent the occurrence of detachment, and that when the two conditions co-exist the former is the probable cause of rupture of the retina.

Though the originators of all these different operative procedures, as well as many of their immediate followers, have reported successful results, yet, in looking over the published statistics, the marked contrast between the optimism of some operators and the pessimism of others is very striking. The special manipulative skill of the particular surgeon accounts for a great deal of his success, but it does not satisfactorily explain the marked discrepancy in the reported results, and on the whole the general trend of opinion seems to be that purely operative measures

promise little help. After having operated on a considerable number of patients and having tried many different procedures, I cannot, in my personal experience, recall a case where operation alone did any permanent good, and I have to admit that in several instances it appeared to be distinctly harmful.

Leber, trusting in his own views of its ætiology, has said that detachment of the retina is generally incurable, and sooner or later ends in blindness, and Vail (42), after consulting a large number of American Ophthalmologists, concludes that with the ordinary methods of treatment the prospect of cure is less than 1 in 1000 cases. Pessimism can hardly be carried further than that, but it seems to me that both Leber and Vail over-state the case, for although doubtless many examples can be cited in support of their view, yet there are other cases in which the prognosis is much more hopeful. The majority of clinicians have seen patients in whom the disease remained stationary and some vision was retained and others in whom it took a favourable course and terminated in spontaneous cure. The number of such cases is admittedly small, but the very fact that they occur is an encouragement to try to find out how they have come about. Recovery may take place suddenly: in one case reported by Higgens (43), the patient woke up from sleep seeing as well as ever, and in another described by Post (44), sight returned while the patient was lying in bed waiting for a cataract operation on the other eye. At other times recovery has followed an accident, a circumstance that suggests that the immediate exciting cause was rupture of the retina. Poncet (45) has pointed out that provided the layer of rods and cones has not been injured by disease of the choroid the detached retina may retain its function for a considerable time, and as vision only remains in abeyance, it is at once restored whenever the retina again comes in contact with the layer of pigment cells.

There are, however, other cases in which the function of the detached retina speedily becomes impaired and

recovery does not take place when it becomes re-applied. At the time of writing I have a patient under my care whom I first saw in 1909. At that time she was suffering from extensive detachment of the retina in both eyes. No active treatment was advised and the patient was seen very occasionally. In 1911, a cataract formed in the left eye, and two years later the same eye suffered from a severe attack of irido-cyclitis. Ultimately it made a good recovery, but it retained only a bare perception of light. For a long time the sight of the right eye did not grow worse, but towards the end of 1914 it began to fail, and when the patient consulted me in January 1915, she complained that she had great difficulty in seeing to guide herself about the house. When I examined the eye with the ophthalmoscope, I found, to my surprise, that the detachment had completely disappeared. The case may be regarded as a spontaneous cure of the separation of the retina, but the patient's condition is no better than it was when the detachment was present.

On the other hand, every clinician is familiar with the fact that a patient's vision may improve decidedly even although a considerable separation of the retina exists. Such improvement is due in some measure to increased transparency of the media, but the site and the extent of the detachment are the factors which chiefly influence sight. A separation, which at first implicates the central area of the retina, may afterwards settle down and allow the macula to resume its functional activity, and only reveal its presence to the patient by a peripheral contraction of the field of vision from which little inconvenience may arise. In neither case is the word cure strictly applicable, but from the patient's standpoint an improvement in sight is much more to be desired than an anatomical replacement of a retina whose function has become seriously impaired.

Even although he knows that his efforts may not result in cure the clinician seeks to ameliorate the patient's condition, and in applying that idea to the treatment of

detachment of the retina I have availed myself of all that I considered best in the different methods that have been recommended, and have sought to apply the remedies in such a way that if I did the patient no good I would at least do him no harm.

The most important factor in the treatment is absolute rest in bed. Patients often volunteer the statement that they see best in the morning after a good night's rest, but that sight fails as the day advances. If, however, the patient is to derive the utmost benefit from rest he must be encouraged to keep as still as possible, to refrain from any exertion, and to avoid straining at stool, sneezing, or coughing. The time necessary for treatment is from four to six weeks, and grave danger attends any attempt to shorten it. Relapse is ever prone to occur, but it takes place much more readily in those who have remained in bed only a short time. In some cases the irksomeness of the period of rest may be reduced by allowing the patient to occupy one bed during the day and another at night, or he may be lifted from bed to a couch for a few hours each day. It is most important to make him feel restful and comfortable. Every effort must also be made to improve his general well-being. He must be suitably dieted, and special care taken to ensure the regular and efficient action of the bowels, kidneys, and skin. His whole physical condition and environment ought to be considered with the most minute attention, and the surgeon must be quick to observe and ready to correct any transient departure from health. In detachment of the retina, as in so many other diseases of the eye, the ophthalmologist can never dissociate himself from the general treatment of the patient.

Next in order of importance comes the pressure bandage. Both eyes should be covered, and the pressure of the bandage must be distinctly felt, but ought to cause no pain. It must be changed night and morning to allow the eyes to be bathed and a drop of 1 per cent. atropine solution to be instilled into the one affected. Sattler (46),

Freytag (47), and some others disapprove of the pressure bandage because its application tends to diminish intra-ocular pressure, and from that they argue that it will do harm rather than good. Such theoretical objections are, however, unsupported by the results of Wessely's (47) experiments in the eyes of cats and dogs, and my personal clinical experience does not confirm them. On the contrary, I regard the pressure bandage as a very valuable means of treatment. It happens very occasionally, however, that a patient cannot bear the bandage. He complains that it causes pain, and when it is removed the eye is found to be red and irritable. In such circumstances its use must be discontinued, because if it be persevered in irido-cyclitis will supervene. My experience is that the cases which are intolerant of the bandage are of bad prognosis.

The third place in my practice is occupied by sub-conjunctival injections. Their use is sanctioned by the experience of De Wecker (49), Dor (50), Darier (51), and many others. The patient is kept in bed with his eyes bandaged for forty-eight hours before the injection treatment is begun. He is then examined with an electric ophthalmoscope, and if the site and the character of the detachment appear favourable, but only in these circumstances, the subretinal fluid is evacuated by scleral puncture. If this operation be performed with strict aseptic precautions it is, in my experience, perfectly harmless, and the careful withdrawal of the fluid permits the retina to fall into place with the least possible delay. Local anæsthesia is employed, the lids are held apart by a speculum, and the patient is asked to turn the eye as far as he can to the right or to left side according to the eye to be operated on. The conjunctiva covering the lower and outer aspect of the globe is grasped with forceps and pulled towards the cornea, and a puncture is made through the sclerotic as far back as possible between the inferior and external recti muscles. Formerly I employed a broad needle for this operation, but latterly I have been using a large Deutschmann's

knife, which is pushed right across the vitreous chamber until its point comes in contact with the opposite side of the globe. It is then partially withdrawn and rotated until the lips of the wound in the sclerotic gape sufficiently to allow the subretinal fluid to escape. The knife is then withdrawn, and the wound is at once covered by allowing the conjunctiva to fall into place. Whenever the subretinal fluid is evacuated, and before the speculum separating the eyelids has been removed, a subconjunctival injection is given. Five to twenty drops of a solution containing 1 per cent. dionine and 8 per cent. chloride of sodium in 1 in 2000 bicyanide of mercury is injected. An all-glass hypodermic syringe, armed with a Dor's curved needle, is employed. A fold of conjunctiva is picked up with fixation forceps about 10 mm. from the corneo-scleral margin between the insertions of the recti muscles, the needle is pushed well backwards, avoiding Tenon's capsule, and the fluid is very slowly injected. The ocular conjunctiva rises in a bleb, but this soon disappears, and is replaced by a diffuse œdema.

The great drawback to the use of subconjunctival injections is the severe pain which they occasion, but that can be greatly mitigated by using chemically pure chloride of sodium, by adding a few drops of 1 per cent. acoine or 2 per cent. alypin solution to the injection fluid immediately before use, by heating the injection fluid, and by applying a fomentation to the eye immediately after the injection is made. If these precautions are taken the suffering is rarely acute and is of short duration. On the day following the injection there is marked increase in the intra-ocular tension, and the media are distinctly hazy, but this loss of transparency does not last long, and, as a rule, within forty-eight hours of the first operation the patient says that his sight is clearer. In about a week the conjunctival œdema has for the most part disappeared, and the eye is ready for another injection. The average number given is from four to six. A marked chemosis of the conjunctiva is essential to the success of the treat-

ment, the improvement in vision seeming to go *pari passu* with the degree of reaction following the injection.

The solution just mentioned seems to answer the purpose, and if the results are watched and the dose carefully graduated a satisfactory chemosis of the conjunctiva can be obtained in any patient without the agonising pain and sickness which so frequently accompany the use of solutions containing a high percentage of saline constituents. Not only, too, are such concentrated solutions very painful, but they are also strongly irritant and cause the conjunctiva to become firmly adherent to the episcleral tissues, so that it is difficult to seize it with forceps or even to give another injection. Citrate of sodium, 25 gr. to 1 fluid oz. of water, has been recommended by Savage (52), and solutions of other substances—sugar, gelatine, etc.—have been recommended by others, but on the whole they do not appear to possess any special advantages. Various opinions are held regarding the *modus operandi* of the injections. From what I have observed at the bedside I have formed the opinion that the injected fluid acts simply as a local counter-irritant, and that belief is so far supported by the experimental work of Wessely (53) and J. H. M'Ilroy (54), both of whom found that an injection beneath the conjunctiva is followed by a marked increase of albumen in the aqueous humour.

Diaphoresis is such a common method of treatment in many different diseases that one cannot wonder that its use has been strongly recommended in detachment of the retina. Of all diaphoretics pilocarpine in doses of $\frac{1}{8}$ gr. to $\frac{1}{4}$ gr. administered hypodermically is the best, and its effect is at times so striking as to raise the question how far internal secretions may influence the nutrition of the eye. I only employ it in cases in which after a fortnight's trial little improvement has been brought about by rest, pressure bandage, and subconjunctival injections. In some cases the first free diaphoresis marks the beginning of improvement which afterwards steadily progresses. In

such cases the use of pilocarpine is alternated with subconjunctival injections, and if the dose be carefully regulated there is little fear of unpleasant symptoms arising, and these may be further guarded against by the addition of $\frac{1}{60}$ gr. of strychnine to each hypodermic injection of pilocarpine.

It is very desirable that as soon as possible after the patient is able to be out of bed he should be sent to the country for a few weeks in order that the general health and strength be brought up to the highest possible standard. Relapse follows much more certainly and speedily in those whose circumstances oblige them to resume their ordinary duties immediately after the treatment is completed. Cases of cure after a visit to a spa are on record, and I have often observed considerable improvement take place during the period of convalescence. The patient should be advised to rest as much as possible in the recumbent position and warned to avoid all stooping or straining movements. One of my most successful cases relapsed suddenly while the patient was sitting in a railway carriage and feeling that he was being much shaken by the vibration of the train. Since then I have always advised those who have to go home by rail immediately after treatment to lie down on the seat of the compartment with the head resting on a pillow and to maintain that position as long as the train is in motion. With the same object in view—the reduction of jarring motion as much as possible—Maddox advises patients to wear rubber heels to their shoes. Sometimes constipation plays an important part in determining relapse, consequently it is important to give particular instruction regarding the action of the bowels.

It is well that at a meeting like this the chief interest is in the discussion and not in the introductory paper, because the combined method of treatment just outlined contains nothing new, and can claim no brilliant results, yet the measure of success that has attended it in my hands encourages me to persevere. It is not easy to give

statistics that are trustworthy, because patients who suffer from retinal detachment go from clinic to clinic and from one consultant to another, and consequently it is very difficult to keep in touch with them, but as far as I can judge nearly half my patients have received benefit, and in about 10 per cent. the improvement has been decided and as far as I know has lasted.

Sattler (53) has said that the smaller the detachment and the more recent the case the more encouraging the prospect, but that must not be interpreted to mean that when the separation is large and of long duration the prognosis is necessarily unfavourable. Some of my most successful results have been in patients in whom the retina had been separated from six to nine months and even longer. It is very difficult to say beforehand what cases are favourable for treatment: it is easier to select those that are unfavourable. Failure is almost certain when the choroid is extensively diseased, when the pupils are sluggish in their response to mydriatics, when intra-ocular tension is greatly reduced, and when little or no improvement is manifest after treatment has had a fair trial for two or three weeks.

There are some who say that no treatment is of any use, and that those cases which improve are examples of spontaneous cure. I cannot subscribe to that belief, for I have seen cases which prove the contrary, and, moreover, the improvement that follows the first subconjunctival injection is frequently so striking that it is difficult to regard the result as otherwise than that of cause and effect.

When so little can be done for the cure of detachment of the retina one naturally wonders if nothing can be done to prevent it. Myopia is associated with detachment so frequently that the latter condition must be looked upon as in some way dependent upon the former. Galezowski thought the fact of the myopia was of more importance than the degree, and every clinician knows that in a myopic eye a trivial injury or a fit of coughing fre-

quently and quite suddenly determines separation of the retina. The best prophylaxis, therefore, is the wise care of the eyes of children and young adults who suffer from progressive myopia. In many instances the onset of detachment is so sudden that no warning symptoms herald its approach, but in other cases an observant patient will describe visual troubles, which had they been appraised at their real value would have given timely warning of the danger of the onset of separation. If a patient suffering from myopia complains of a sensation as if water was flowing in front of the eye, of phosphenes or of brightly coloured lights, of metamorphopsia, of floating bodies, or of seeing a grey or a black curtain in front of the eyes, he ought to be treated at once, and every effort made to get rid of the symptoms. Their timely recognition and proper interpretation may save the patient from much more serious trouble.

Lastly, what ought to be done with patients who develop cataract in an eye which has previously suffered from detachment, and whose other eye is of little or no use? Clinical experience has proved that many of those cases are not nearly so hopeless as one would naturally imagine, because after the lens loses its transparency the retina in some instances becomes replaced so far that the extraction of the cataract gives the patient useful vision. These eyes, however, are very vulnerable. They bear operation badly, and the gradual onset of irido-cyclitis, it may be months afterwards, takes away the sight that has been restored. In such cases my practice is to perform simple extraction and to remove the opaque lens beneath a large bridge of conjunctiva. By that method the minimum of traumatism is secured and healing takes place with great rapidity.

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Opening Paper.

By Leslie Paton.

In medicine there are some diseases which invariably produce a feeling of deep pessimism when they come under discussion. The majority of these will be found to have one common feature—an obscure pathology. The first step towards the cure and extirpation of cancer will have been taken when its causation is discovered. The attitude of medicine towards tuberculosis has been completely changed by the finding of the tubercle bacillus, and a similar disappearance of pessimism is taking place in the subject of syphilis since the spirochæte has been

tracked to his pallid couch. In any disease, so long as the cause is unknown and the pathology obscure, the treatment must of necessity be empirical and very frequently ineffectual. Of few diseases can this be said more truly than of detached retina. It is in some ways the most depressing ocular lesion that we can meet with. It belongs to the rare category of cases like Mooren's ulcer and hæmorrhagic glaucoma that one would rather see in the hands of one's colleagues than deal with oneself. Yet it is not by a mere *non-possumus* that we shall ever do any good. The problem has to be faced and some day it will be solved. Meantime a free discussion of its nature may be of assistance.

The reader of an opening paper in a discussion may regard his function in various ways. He may seek to cover the whole ground of the subject and deal with it in a comprehensive fashion guarding his whole line against the attacks wherever he can anticipate them. In other words, he may regard his audience as an enemy lying in wait to attack his propositions. This method has given rise to many valuable contributions to medical literature and much credit to the readers of such opening papers.

I have found it impossible to deal with the subject in such a way. I can lay down no thesis and support it with an array of facts. I have found no text round which to write. I have found only the mists of confusion and conflict of opinions—conflict of opinion as to ætiology, as to treatment, and as to results, and the only certainty that emerges from all the accounts I have read is that in many cases where the surgeon reports a cure, the unfortunate patient is still in the dark.

I think we must first realise that we are not dealing with a clinical entity in the way of disease. Retinal detachment is an end result, and it is a result which may be brought about by varying and essentially different causes. It may clear the ground a little if we consider firstly some of the cases which can be explained without having recourse to any doubtful hypothesis.

In cases of tumour of the choroid detachment may legitimately be regarded as due to the accumulation of œdematous fluid behind the retina produced by the irritation of the growth. As Parsons has shown, there is often evidence of inflammatory reaction at the edges of the growth. He also called attention to the fact that the detachment may be away from the tumour, as, when the tumour is in the upper segment of the eye, the fluid tends to gravitate to the lower segment.

A second type of case where there is little doubt as to the causation is where there has been a perforating wound of the sclera. In August, 1905, I saw W. H—, a railway porter, æt. 31 years. Two months before he had been knocked off a van on to the railway bank and had injured the left side of his head and his left knee. He did not know of any injury to his eye, but about a month later he noticed that with his left eye things were looking far away and distorted, and at times he could only see the floor and nothing above the floor level. On examination vitreous showed numerous floating opacities and in the lower part a bluish grey opacity looking like a foreign body. The lower part of the retina was detached. X-rays showed a small foreign body in the lower outer quadrant fairly far back and near the periphery. Vision was $\frac{6}{36}$. There was no indication from the history that the foreign body was steel or iron, but on inserting the small magnet through a scleral incision it came out at once. The immediate result of the operation was a considerable increase in the area of the detachment, but later on this disappeared, and when I last saw him in January, 1912, six and a half years later there was no trace of detachment; his field was full, his vision was $\frac{6}{6}$, and the only sign left was the large white pigmented scar of the operation in the lower outer quadrant of the left eye.

In such a case I think the assumption is justified that the perforating body after piercing the sclera and choroid carries the looser retina in front of it before tearing through

into the vitreous, and the separation thus set up is kept up by the resulting hæmorrhage and exudation from the injured choroid.

In a third class of case the detachment of the retina may be explained by the rapid exudation of lymph and serum into the subretinal space in a severe choroiditis or general uveitis. W. C—, æt. 18 years, was admitted to St. Mary's Hospital on May 6th, 1904, for detachment of left retina. There was a history of a cut on the forehead seven weeks before for which the patient was admitted to Guildford Hospital, and, according to his statement, his sight was then affected. He went back to work, but six weeks after the accident his left eye became inflamed and very painful. He became drowsy and vomited after food, lost his appetite, and began to lose weight and became giddy. On admission left eye was congested, the cornea was hazy, the tension was raised, the iris was discoloured and congested, the anterior chamber was deep, the vitreous was hazy, and only a very small area of red reflex was seen in the lower part of the fundus. From the rest of the fundus the reflex was greenish. No history of rheumatism, gonorrhœa, or syphilis.

The right eye was emmetropic with vision $\frac{6}{6}$. The left eye was excised and was found to have a large detachment of the retina due to a large subretinal effusion of lymph and serum from general uveitis.

Similarly subretinal hæmorrhage may be a cause of detachment. F. H—, æt. 47 years, seen on August 7th, 1907. History: On the morning of July 28th wakened up with a feeling that something was wrong with right eye. On testing it he found he could only see large objects very dimly. No pain in the eye, but since onset he can see dimly in the morning, but as day goes on his sight becomes worse. He had rheumatic fever in 1904. He has never had syphilis. He is married and has four healthy children. When first seen he could count fingers with the right eye. There was a large detachment of retina below with vessels seen on the surface. The

tension was normal. After three weeks' treatment with rest in bed the condition was much worse. Ophthalmoscopically there was a large black mass involving most of the field except a small area above which gave a red reflex. No details were now visible. I asked Mr. Fisher and Mr. Parsons to see him at Moorfields, and they both agreed that it was advisable to enucleate. Examination of the eye showed no trace of a growth, but a large hæmorrhage behind and in front of the retina. It was obvious from the history that the primary hæmorrhage must have been behind the retina, and that the bursting through into the vitreous took place between the time he was first seen on the August 7th and the date of his admission to St. Mary's Hospital a week later. As to the source of the bleeding in this case, whether from retinal or choroidal veins, I do not feel able to give a definite opinion.

In these four classes the main effective force in detachment operates from outside the retina and shoves it away from its apposition to the choroid. The next type of case I have to mention illustrates the reverse condition of the retina being pulled away from inside.

A. M—, æt. 28 years, first seen in November, 1905. Had been the subject of recurrent vitreous hæmorrhages, The first one occurred in the right eye in March, 1904. and in the interval there had been several in each eye. The clinical history of the case presents many points of very great interest, but as they have no direct bearing on the subject of detachment I omit them. But the vitreous hæmorrhages had given rise to much vitreous degeneration and the formation of organised bands in the vitreous (proliferating retinitis), one of which could be traced down to a large detachment in the lower part of the field, evidently of some standing, as it was greyish white in colour. There were cicatricial bands running in the retina itself, and at one point where the bands crossed, forming a T, there was a distinct tear. In such a case the evidence favours the hypothesis that the detachment is due to the

cicatricial bands in the vitreous forming retinal attachments and then by their contraction pulling it away from its apposition.

But there is still another way in which I think it is possible that detachment may occur, and that is by actual cicatricial contraction in the retina itself. Some of you may remember a case of a small boy, whom I showed at the Ophthalmic Section of the Royal Society of Medicine, with a very curious hole at the macula and a most extensive star of œdema all round the macular area. Later on there developed a shallow detachment over the œdematous area. Now in albuminuric and diabetic retinitis, especially in late and severe cases, detachment may occur. Of course here there may be two or three different factors at work, but one factor that must not be overlooked is the cicatrisation in the retina itself, causing it to contract and straighten, and so pull away from the choroid. In the following case, indeed, this seemed to be the main factor in the detachment.

Mrs. H—, æt. 52 years, first seen April, 1906. She had noticed some dimness in reading for three or four months, and in distance for some time longer. R.V. $\bar{c} - 1{\cdot}5$ D. Sph. $= \frac{6}{30}$. L.V. $\bar{c} - 1{\cdot}25$ D. Sph. with $- {\cdot}5$ D. Cyl. ax. V. $= \frac{6}{12}$. Lenses showed early cataracts.

Rt. Fundus.—The whole macular and perimacular area was œdematous, and there was a circle of blot hæmorrhages round the macula. A similar but less marked condition was found in the left eye. Examination of her urine showed 1·5 per cent. of sugar, but no albumen. In February, 1907, the œdema in the right eye was less, there was a small fresh hæmorrhage near the macula, and another on the disc. There had been a small hæmorrhage into vitreous. Vision was reduced to $\frac{6}{60}$. In the early autumn a detachment took place over the whole macula, and spread into the upper part of the retina. Meantime the vision in the left eye was deteriorating slowly, with œdema and constant small hæmorrhages in the perimacular area. And in February, 1909, I have a note of con-

siderable œdema and several hæmorrhages with white lines of old exudate (?) showing in the retina. Vision reduced to $\frac{6}{60}$. I think that if death had not intervened the next stage would have been a similar detachment in her left eye, brought about by contraction of these cicatricial bands in the retina itself.

So far the types I have been dealing with belong to what have been termed by Abadie symptomatic detachments, and here also must be included traumatic detachment in normal eyes. I have already mentioned one form of traumatic detachment where the injury is a perforating wound of the sclera, but the ordinary form may be called more simply concussion detachment. To deal fully with this part of the subject would necessitate an analysis of the various results on different portions of the eyeball, which may result from a blow, a task which in itself would require a text-book. Wagenmann, in Graefe-Saemisch's handbook, devotes over one hundred and fifty pages to the injuries due to simple contusion. It must suffice for my purpose to-day to say that here, again, we get evidence that the detachment may be produced in different ways: (*a*) Rupture of the choroid and hæmorrhage from the torn vessels lifting up the retina; (*b*) hæmorrhage into the vitreous, with subsequent vitreous degeneration and pull on the retina; and (*c*) injury to and rupture of the retina itself, without obvious choroidal rupture or bleeding and without obvious intra-vitreous hæmorrhage or vitreous degeneration. In this third class I think will be found many of these cases where the evidence of detachment only manifests itself some time after the blow. The following case illustrates this point. In January, 1910, I examined the eyes of a gentleman, H. C—, æt. 34 years. His visual acuity in each eye was $\frac{6}{5}$. His fundi were normal. He had + ·5 D. of normal astigmatism in his right eye, and his left eye had + ·25 D. of hypermetropia. He had slight chronic angular conjunctivitis, and I think his only reason for consulting me was that he came in with his wife and thought he might

as well let me have a look at his eyes. In February of 1911, while hunting, he was hit under the right eye by the branch of a tree. I gathered that he had a fairly bad black eye, but that after the swelling had gone down he did not notice much wrong with his sight, nor did it cause him any worry until June or July, when he found some difficulty in playing tennis and taking a ball high up on his right hand. He did not consult me till the shooting season commenced, when he found a difficulty in judging pace and distance, and that he could not shoot with a rifle because of the blurring of the foresight. By this time, the middle of September, he noticed that on looking at anyone with the right eye he saw the legs better than the head. Examination showed a large detachment in the lower part of the right retina. The vitreous was clear, but on the surface of the detachment at one part there was an elongated, horizontally lying, whitish area with cholesterine on the surface of it. In this case rupture of the choroid, and hæmorrhage from it or hæmorrhage into the vitreous at the time of injury, would have caused more interference with sight at the time, but a slight tear in the retina, with a little subretinal hæmorrhage, might have passed unnoticed until the increase of the detachment forced it on the patient's attention. In another case, E. F—, æt. 60 years, the injury was due to the loose end of a rein, thrown over a horse's back, catching him on the left eye. It caused a good deal of pain for some hours, but he noticed nothing wrong with his sight for ten weeks, when he became conscious of a darkness in the lower part of his left field, which spread in the course of three weeks so as to obscure the whole field of the left eye except a small portion above. He was emmetropic with $\frac{6}{5}$ vision in the right eye, and had had as good sight in the left eye before the accident. The detachment, when I saw him, was in the upper part of his retina, spreading down over the macula. The vitreous was clear, but no tear was seen in the retina. In another case, C. H—, the interval between the blow and any noticeable affection of

vision was two months. Again this was an emmetropic with no obvious vitreous change.

From the examination of these different types of case I think we may conclude that symptomatic detachment may arise either from (1) a push from behind, (2) a pull from inside, (3) a contraction in the retina itself, or (4) a combination of any or of all these causes.

Having to some extent cleared our feet we may now turn to the consideration of so-called simple detachment, though the use of the term "simple" seems, to put it mildly, somewhat sarcastic. I do not wish to trespass on Mr. Mayou's province, but it is necessary for me to refer briefly to the two main hypotheses which have been advanced to explain simple detachment. Raehlmann in 1876 definitely formulated the diffusion hypotheses. We may regard his work as the codification of many preceding indeterminate suggestions. A subretinal exudate is a colloidal fluid with a relatively high isotonic coefficient, which to some extent is due to its non-diffusible proteid constituents. On the other side is a fluid vitreous with a lower isotonic coefficient, which for practical purposes we may assume as equal to or little higher than that part of the isotonic coefficient of the subretinal fluid due to its crystalloid constituents. There are thus present on two sides of a membrane the retina, two fluids of different tonicity, and, presuming the living retina to act as a passive dialysing membrane, fluid must pass from the vitreous into the subretinal space and raise the retina until its pressure causes a rupture. This hypothesis of Raehlmann's makes three assumptions—(1) a degenerate fluid vitreous, (2) some choroidal or retinal lesion which will give rise to a subretinal exudate, and (3) the passivity of the retina.

Leber in 1882 definitely formulated the hypothesis of vitreous traction which, owing to the excellent work of Nordenson, is now usually known as the Leber-Nordenson theory. The vitreous loses its normal jelly consistency, becomes coarsely fibrillar with fluid in the meshes of the

fibrillæ. These fibrillæ shrink. Normally the vitreous is attached to the retina only at the optic disc and anteriorly at the ora serrata, but exudates into the vitreous may cause it to form false attachments. The shrinking of the vitreous fibrillæ pulling on the retina either at its normal attachments or at pathological attachments tears the retina, and the fluid vitreous thus gets access to the subretinal space and detachment results. Leber and Nordenson's main assumptions then are: (1) Degeneration of vitreous with coarse fibrillation, (2) contraction of these fibrillæ pulling on retinal attachments, and (3) a tear in the retina.

Vitreous degeneration and fluidity are the features which are common to these two hypotheses, and it may be accepted as a justifiable assumption in the majority in cases. But I should like to make here a distinction of the way in which we word this assumption. Symptomatic cases excluded, detachment does not occur so long as the vitreous humour retains its normal consistency and occupies its relative normal space in the eyeball. Wording it in this way brings out one point. In those cases where the scleral cavity increases in cubic capacity we get definite evidence that there is not a corresponding growth in superficial area of the choroid and retina, and the deficit is partly made up by stretching of these coats and partly by uncovering of a portion of the scleral area round the disc. At the same time there is not a corresponding growth in normal vitreous to fill this pathologically increased space, and the deficiency has to be filled with fluid which will secondarily bring about a degeneration in the vitreous. There are then two classes of case in which a vitreous does not adequately fill its space: (1) where there is shrinking and degeneration of the vitreous, and (2) where there is increase of the space to be filled without corresponding increase of the vitreous.

The assumption of a fluid vitreous as a precedent of detachment does not get us very much further. The condition of the vitreous is dependent mainly on the con-

dition of the ciliary body. It may be affected also in retinal and choroidal lesions. Consequently vitreous exudates and vitreous degeneration are mostly secondary to disorders of the ciliary body and to a less extent of the choroid and retina. Occasionally there is very definite evidence of involvement of the uveal tract. I have already cited one case of severe iridocyclitis amongst the symptomatic cases and comparatively recently I have seen a mild iridocyclitis with a few adhesions and slight K.P. developing in a case while under treatment and observation.

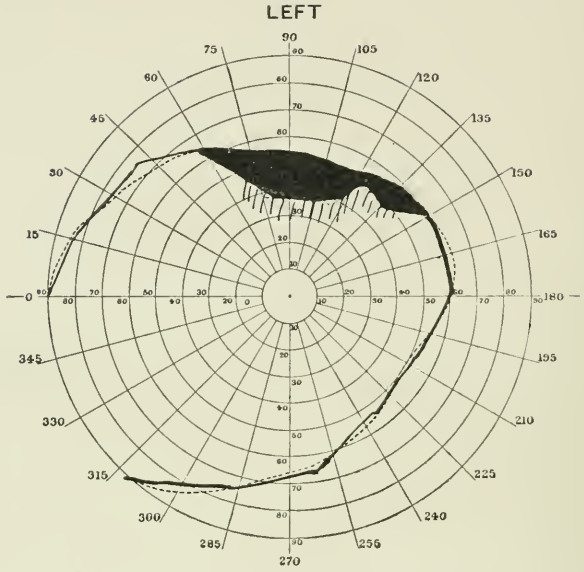
The one undoubted clinical fact about simple detachment is that the great majority of cases occur in myopes. Galezowski found that out of 1158 cases 918 were myopic, 79 per cent. Other authors give lower percentages than this, but I think most surgeons will agree that the figure is nearer 75 per cent. than 50 per cent. The degree of myopia does not seem to be of importance. It would be of great interest if some one could give us statistics of the rate of progress of the myopia preceding detachment as an aid to determining whether a progressive myopia is more likely to be followed by a detachment than a non-progressive. Looking over my notes I find it occurring in all degrees of myopia from − 2 D. to − 16 D. This of course does not represent an upper limit, but I think that the greater proportion of cases are under 12 D. of myopia and that from 5 to 10 D. represents the peak of the danger curve.

I have already spoken of the increased cubic capacity of the scleral cavity in myopia. There remain for discussion the other changes associated with myopia which may conduce to detachment. The most obvious change is the shrinking away of the inner coats from the edge of the disc, the development of the myopic crescent. But other signs of the strain put on these coats may be seen. Sattler, in his Bowman Lecture, refers to a more diffuse clearing of the posterior part of the fundus which renders the choroidal vessels more visible, and

to the appearance of zig-zag lines of venous sclerosis. He ascribes the development of the patches of myopic atrophy of the choroid to rents in the membrane of Bruch owing to the stretching. Now if a tear in the membrane of Bruch takes place rather suddenly and is more extensive than usual, I think it is quite likely to give rise to detachment. One of the cases which I hope to be able to show at the clinical meeting will demonstrate better than any words of mine can the possibility of this factor.

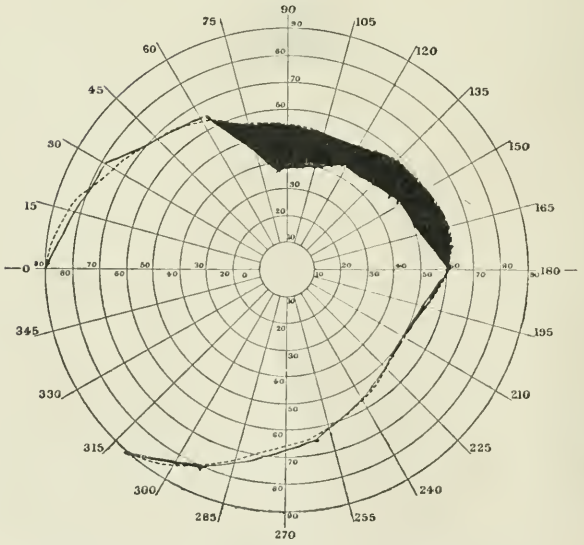
Miss D—, æt. 30 years, was sent to consult me in July, 1908. She had always been short-sighted, and had worn glasses for twelve years. She was wearing − 3·5 D. sph. E.E. Her full correction was − 5·5 D. sph. E.E. She complained that for some time, on using her left eye, there had been a blurring of objects up and in, lines did not seem to be straight, and she was always conscious of a kind of greenish light. Her pupils were active and her tension normal. Corrected vision in right eye = $\frac{6}{5}$; in left eye = $\frac{6}{9}$. Left visual field *v.* chart. Ophthalmoscopic examination of left eye showed a large detachment in the lower temporal segment sloping up towards the macula, the highest point quite clear with + 8 D. Some blackish looking striæ on surface of the highest part of detachment. In the course of three months' treatment the detachment subsided completely. Vision in the left eye improved to $\frac{6}{5}$, and, though the actual absolute loss of field remained the same, there was recovery of the area partially lost. Now, after more than six years, there has been no recurrence of the detachment, but over the site of the old detached area there are numerous spots and blotches of pigment, much of it without any obvious choroidal disturbance. Some distance below the disc and towards the upper edge of the area of pigmentary disturbance there runs a long line edged with black pigment, and with three or four larger white areas strung on it like beads on a string. This may mark the site of a line of rupture of the membrane of Bruch. This patient at no time showed

FIG. 3.



Miss K. D—. 10 mm. white. July 3rd, 1908.

FIG. 4.



Miss K. D—. 10 mm. white. February 20th, 1915.

obvious vitreous disturbance, nor could I ever see any tear in her retina, though it was a very easily seen detachment. I think there is, in this case, some justification for the assumption that the sequence of events was first a long tear in the membrane of Bruch and a consequent injury to the chorio-capillaris with hæmorrhage into the subretinal space, and that the pigmentary disturbance now obvious is mainly due to disturbance of the retinal pigment while the detachment lasted.

Another case, which I also hope to be able to show at the clinical meeting, gave evidence which favoured the hypothesis of vitreous traction.

Miss P—, æt. 41 years, was sent to me in August, 1907, with a large detachment in her right eye. The sight of the left eye had been lost for eighteen years before from detachment. When I saw her in August it was owing to the absence from London of all the other surgeons who had been in charge of the case, but she had been under observation then for over ten weeks, and the detachment has been spreading. I found a large greyish detachment involving the whole nasal half of the right retina reaching up to the edge of the disc and the upper part of the retina extending over the temporal side. Her vision in the early stages of the detachment had been $\frac{6}{9}$ with − 16 D. sph., but when I saw her it had fallen to $\frac{6}{60}$. On August 13th I operated on her by post equatorial galvano-puncture of the sclerotic over the detached area. The result of the operation was that the detachment went back in position on the nasal side, but an almost equally large detachment appeared on the temporal side. On September 9th I repeated the operation. At the end of September when I, in turn, left town, her vision had very much improved, but she still remained in the nursing home. Her subsequent history is that in November, 1907, her R.V. c 16 D. = $\frac{6}{30}$, and there was still some detachment at the outer side, *i. e.* away from the original detachment. In 1908 vision improved to $\frac{6}{18}$; in 1912 to $\frac{6}{9}$ and J. 1; and on March 19th,

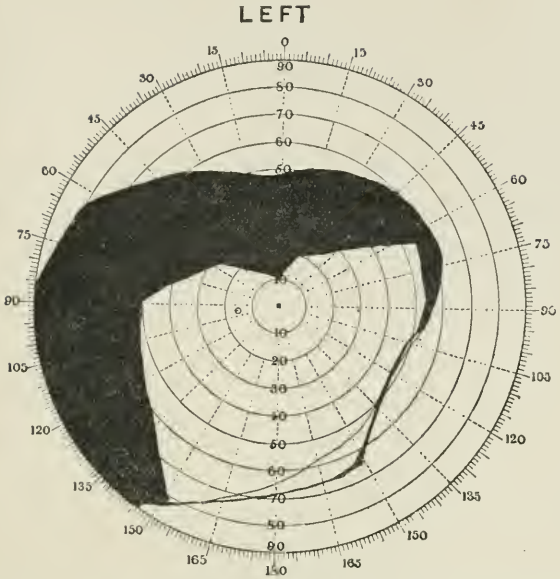
1914. Right vision $\bar{c} \frac{-16 \text{ D. sph.}}{-\cdot 75 \text{ D. cyl.}}$ ax. 100 $\frac{6}{9}$ (5) and $\bar{c}$ — 13 D. sph. cyl. = J. 1, and no detachment is visible. In this case, then, the immediate result of pulling the retina back into position at one side was to detach it at the opposite pole, and the most probable cause of this was the presence of vitreous bands running from side to side.

This point is well illustrated by the visual fields of another case G. B— (male, æt. 43 years), which I show. I much regret that his story has not such a happy ending as Miss P—'s. But it illustrates the same point that replacement of the detachment at one pole was accompanied by detachment at the opposite pole. And this was also definitely observed ophthalmoscopically in G. B—'s case, and I have seen similar happenings in other operation cases. It is not a strong point, but so far as it goes it is in favour of the vitreous traction hypothesis.

A point on which Vail, of Cincinnati, lays great stress is that in myopes generally the circular ciliary muscle is atrophied from non-use. He considers that this may be associated with a more generalised atrophy of the ciliary body with, as consequences, diminution of aqueous secretion and so lowering of ocular tension, and, if I take his meaning aright, also degeneration of the vitreous. The general lowering of the vitreous tension causes passive hyperæmia of the blood vessels of the vascular tunics, and consequently serous transudation into the subretinal space, and so, by Raehlmann's hypothesis, detachment. Vail's hypothesis, apart from this one point about myopia, seeks to explain all cases of detachment on the basis of a diminution of the secretory function of the ciliary body, and, as I have already pointed out, the previous hypotheses lead us only as far as the ciliary body if not alone at least as part of the uveal tract, so that I think he must be considered as working on the right lines in directing his attention mainly to the ciliary body.

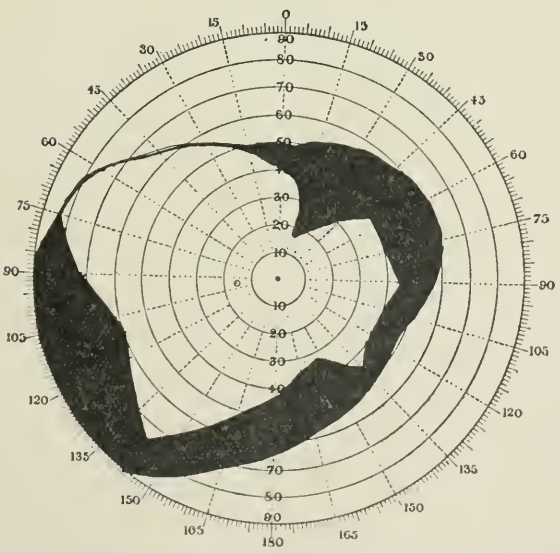
The possibility of sympathetic detachment was suggested by Galezowski in 1884, and again by Wicherkiewicz

Fig. 5.



G. B—. Before operation. 10 mm. white. January 1st, 1912.

Fig. 6.



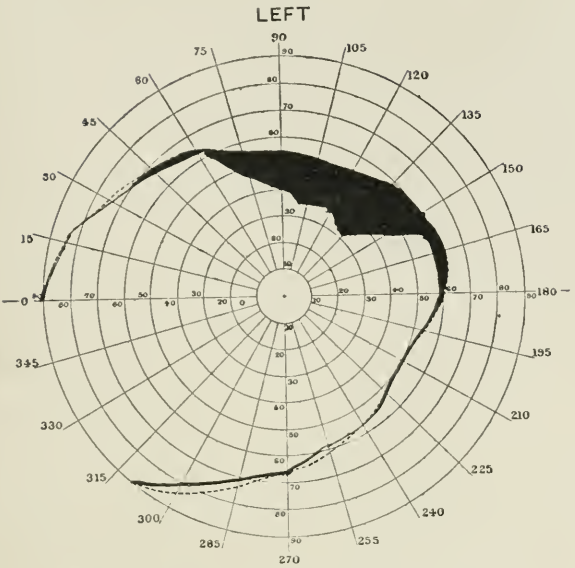
G. B—. After operation. 10 mm. white. March 13th, 1912.

in 1900. Whether it is a direct sympathising, or whether the affected eye acts by setting up some form of chronic cyclitis or uveitis in the sympathising eye, is a moot point, but I have on my records a case where some such connection seems the only possible explanation of the detachment in the second eye.

W. P—, æt. 53 years, gamekeeper, was admitted to the National Hospital, Queen Square, in July, 1907, and later on transferred to St. Mary's. Thirty-three years before (November 5th, 1874) he lost the sight of his right eye from detachment of the retina caused by a firework exploding in his face. I think it must be sufficient evidence of the healthy condition of his left eye that he was and is a very valued head gamekeeper in a big shoot in the south of England. In July of 1906 the vision in his left eye began to fail, and by April, 1907, he was unable to read. At the time of his admission to the National Hospital he could see fingers at 1 ft. with his right eye. With his left eye his vis. ac. $= \frac{3}{60}$, though occasionally he could get a glimpse of 36 metre Snellen type held at the foot of his bed. There was a large old standing detachment of the right retina. The left fundus showed a detachment involving the temporal half of his retina. There was some old choroidal (?) change at the extreme nasal side of his retina in this eye. The case is reported in vol. xxviii of the Society's *Transactions*, but as I hope to show him at the clinical meeting I may briefly record his subsequent history. He had in all three post-equatorial galvano-punctures. By July, 1908, he could read $\frac{6}{12}$ with his left eye. August, 1910, vision was $\frac{6}{9}$. He shot a rabbit at forty-five yards. December, 1910, shot a right and left brace of partridges. September, 1911, vision $\frac{6}{6}$ and field full, with reading correction J. 1. When last tested in February of this year he could not get full $\frac{6}{6}$, but he got Jaeger 1 and $\frac{6}{9}$ easily.

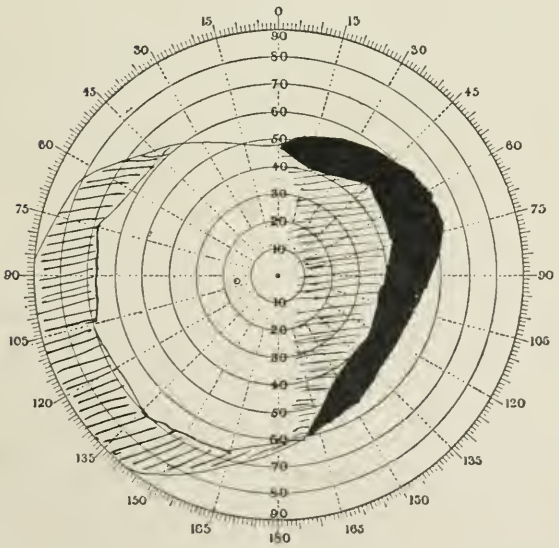
Now in this case, unless the detachment of the right retina and consequent degenerative condition of the inner eye coats had set up some sympathetic changes in the uveal

FIG. 7.



W. P—. 10 mm. white. February 25th, 1910.

FIG. 8.



W. P—. 10 mm. white. July 25th, 1907.

tract of his left eye which led to the subsequent retinal detachment of that eye, I am quite unable to explain how the detachment arose. As I have mentioned there was doubtful evidence of choroidal mischief in the extreme periphery of the fundus away from the detachment.

I have not gone into statistics of my own cases, and such as I have cited have been mainly to illustrate points I wished to lay stress on. I have had some cases of cure —*i.e.*, not only reposition of the retina, but also restoration of function—and while I would like to think that these cures are the result of my treatment, and while in my own heart I may really believe that they are so, there must always lurk behind that thought the knowledge that spontaneous cure occasionally takes place in this condition, and the suspicion that these might have been cases which if left alone would have got better. I can only say that having been left alone they had not got better, and that after treatment they did get better. All the published statistics on this question seem to me to be vitiated by using the term "cure" for simple reposition of the retina, and I do not wish to add my quota to these misleading numbers.

It is a very important practical point that we constantly have to decide what we are to say to a patient who comes with detached retina. I think the only plan we can adopt is to put the whole circumstances and probabilities as fully and fairly as possible before the patient, and if he decides to have nothing done then, like Dogberry, "take no note of him, but let him go and thank God you are rid of a knave."

I am only too conscious of the many lacunæ which my paper presents. I have found that in the attempt to compress what I have to say within the prescribed limits I have had to cut short arguments, and leave many frayed edges showing. Even so there are many points which I have not touched upon at all. But I hope I have made sufficiently clear my own belief that detachment of the retina may be the end result of many different processes, and that in the present state of our knowledge

there is no hypothesis which can have a general application. Nor are we in a position to prognosticate which cases may have a favourable ending if we do decide to intervene actively in treatment. To talk of reposition of the retina without restoration of vision as a cure is nonsense. The displacement of the retina is certainly the result of the disease, but it is not the whole disease, nor is its reposition the whole cure.

The retina is primitively a vesicle, and even in its full development much of the vesicle condition still persists. The outer wall of this vesicle forms part of the membrane of Bruch, and so is brought into fairly firm connection with the subjacent choroid, while the inner wall, over the greater part of its extent, is only held loosely in apposition with the outer wall by the presence of the vitreous in the posterior chamber. A potential space is present, and it requires no great disturbance of these relations to convert it into an actual space. Indeed, when we consider the anatomical conditions and the frequency of fluid vitreous we may think ourselves fortunate that detachment is not a great deal more common than it seems to be.

I must, in concluding my paper, acknowledge my indebtedness to Dr. Ernest Thomson's very full review and bibliography of this subject which appeared in vols. vii and viii of the *Ophthalmoscope*. It has been of constant aid to me in hunting up information on various points.

Additional Remarks by Mr. Paton.

With regard to operation, you are not dealing with the causes of the disease, neither are you when you remove a cataract; you are removing the effects of the disease, not dealing with the primary cause. So in trying to get a detachment back into position you are not dealing with the disease but with the serious result from the patient's standpoint. I do not think that, in all cases, operation is desirable. It is not desirable if there is a good eye. But

if one eye has been lost, and there is detachment in the other, and you do not do anything, you are leaving that patient in a dreadful position. If you have a success you have always the lurking suspicion that the case might have got well in any case, for you know that cases do get well spontaneously. In some cases I have obtained good results after operation, even good results in cases which have been left alone for a long time, and I have seen many bad results in cases which have been left alone. I think that in some cases operation is desirable and necessary. In doing the operation it is desirable to try to get reposition of the retina with as little bleeding as possible, and I do not think there is any way of doing that so easily as with the cautery. I have tried trephining the sclera far back, but in going through you are bound to get bleeding from the choroidal vessels if you use a sharp instrument. The cautery should be used at a very dull red heat, and used slowly, with the idea of cauterising the vessels as you go through. In the case of the old gamekeeper, after cauterising above and below the external rectus I perforated the retina with a Graefe knife and tried to pull the retina into the wounds. In a case which I did some months afterwards I got through the choroid without causing any bleeding. This you can tell, because unstained sub-retinal fluid comes out. But in perforating the retina I evidently cut through a retinal vessel; this bled freely, and the result was disastrous. I do not think a sharp instrument should be used, because there may be a relapse of the condition if there is blood about. I always try to get as far behind the equator as I can. I catch up the appropriate rectus on a strabismus hook, rotate the eye well round, so as to get as far back in the sclera as possible, and use the cautery at a low heat, trenching the sclera instead of making a hole, and aspirating with the lacrymal syringe after perforating the choroid.

Opening Paper.

By Stephen Mayou.

(With Plate I.)

It is impossible in the short time at my disposal to discuss all the various pathological conditions which give rise to detached retina, which is only a sequel to other intra-ocular diseases. I therefore propose to divide this paper into two parts, firstly, to say something of the physical phenomena with which it is associated, and secondly to deal with some of the forms of detachment which I have had a special opportunity of studying, *e. g.* congenital detachment. Like so many other terms in ophthalmology the term "detached retina" is pathologically speaking not correct. The condition is one of separation of the retina between the pigment cell layer and the rods and cones—that is to say, in the situation where the two layers of the optic cup come together during fœtal life, and which is subsequently represented as a lymph space.

The disease is essentially a cystic condition which may be congenital or acquired. There are three main theories which account for it :

(1) Which was advanced by Von Graefe, that the condition is due to an exudation of fluid in the inter-retinal space.

(2) Which was advanced by Leber, that the condition is due to the pulling off of the retina by the contraction of the vitreous.

(3) Which was advanced by Raehlmann, that the condition is produced by the diffusion of fluid from the vitreous into the inter-retinal space. Other theories are combinations of these in some shape or form.

Although it is probable that all cases of detachment cannot be explained on one theory since the condition is a

sign rather than a disease, the author is a strong adherent of the first, namely, that the cause is exudative in origin and that it is impossible for a detachment to occur without fluid being effused from the choroidal vessels, and that this fluid must have a higher specific gravity than the fluid contained in the vitreous chamber, shrinkage of the vitreous only predisposing to the condition.

The part played by diffusion can be neglected on the premises that the amount of saline constituents of the vitreous and the blood plasma or lymph is about equal, although the fluids on either side of the separated retina may vary in the amount of albumin they contain; albumin plays no part in osmosis of fluid through an animal membrane, and it is only the difference in the amount of the saline constituents which produces the passage of fluid from one side to the other. In the normal eye the inner layers of the retina are attached to the walls of the globe at the ora serrata and around the optic disc. The remainder of the inner layers of the retina under normal conditions do little more than lie in contact with the pigment cell layer and it is probable that there is no true tissue adhesion between the rods and cones and pigment cell layer, or if there be any adhesion, it is so slight that it is a negligible quantity in keeping the retina in its place. This potential space is filled with lymph, so that the rapid tissue change associated with the bleaching and regeneration of the visual purple can take place.

The outer layers of the retina are held in contact with the pigment cell layer lying on the membrane Bruch (1) by the vitreous enclosed in the hyaline membrane pushing the retina against the outer wall of the globe, (2) by the co-adhesion of the membrane between it and its pigment cell layer.

In the normal eye *distension of the vitreous in its hyaloid* is by fluid derived from the ciliary process; this probably plays an important factor in holding the retina in its place—that is to say, that the vitreous exerts a pressure by having a higher density (containing more

PLATE I.

Illustrates Mr. Stephen Mayou's paper on Detachment of the Retina (p. 107).

Fig. 1.—Detached retina ? due to thrombosis of venæ vorticosæ, showing retinal and choroidal hæmorrhages. Retina detached by coagulated albuminous fluid.

Fig. 2.—Section through the walls of the globe. *a*. Albuminous fluid in the inter-retinal space in which are strands of fibrous tissue. *b*. Choroidal hæmorrhages. *c*. Sclerosed posterior ciliary vessels. *d*. Hæmorrhages in the retina.

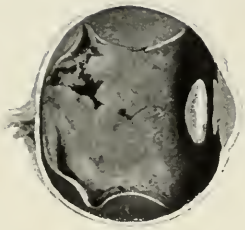


Fig. 1.

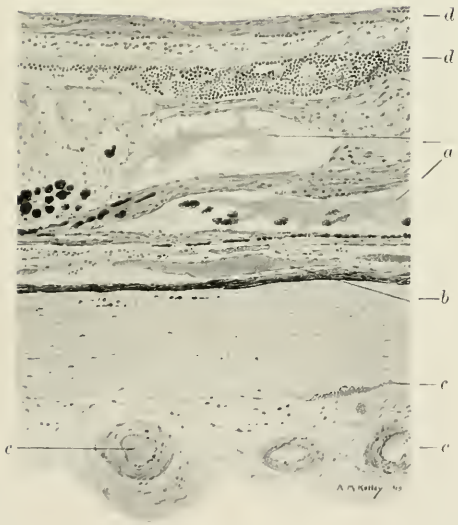


Fig. 2.

Adlard & Son, Impr.

albumin) than the lymph which is present in the inter-retinal space.

In many diseases, especially those associated with a detached retina, the vitreous undergoes degeneration and becomes more fluid than normal, or more commonly the supporting framework of the vitreous shrinks together with the hyaloid, and the space between the hyaloid and the retina is occupied by free fluid. Both conditions are clinically known as fluid vitreous, but, pathologically speaking, the latter is not strictly of this nature, although the fluid is probably derived from the ciliary process. The author is inclined to think that the latter condition is by far the most frequent.

When such degeneration of the vitreous has occurred the density of the fluids on the two sides of the membrane very nearly approximates, and a small increase in the density of the fluid in the inter-retinal space will cause a detachment to take place, the fluid vitreous being pushed forward into the anterior chamber and excreted from the eye at the filtration angle.

In investigating such cases the author would point out that it is necessary to use some fixing agent which coagulates albumin and which does not destroy it for microscopical purposes. For this purpose he has found that boiling is the most satisfactory; the eye, after its removal, being placed in hot water and then brought to the boil. Very good fixation of the vitreous and the tissues is thus obtained.

The underlying cause of shrinkage of the vitreous is usually very difficult to ascertain. It is due to deficient absorption of fluid into the vitreous, possibly from some change in the osmotic properties of the hyaloid membrane, or more probably a deficient secretion from the ciliary body, the latter condition being the result of atrophy, either from previous inflammation, increased intra-ocular tension, or stretching of the globe (myopia) (Vale [1]).

The cohesion between the retina and its pigment cell layer.

When the surfaces of two membranes are in contact, with a thin layer of fluid between them they cannot be pulled apart without the space being filled either with fluid or with air. As, for example, in the body we have the pleural cavity where the two surfaces of the pleural membrane are always kept in contact by this force. When there is an effusion of fluid into the pleural cavity the two layers are separated, causing detachment of the inner layer of the pleura. In the same way, detachment of the retina occurs when a larger pressure is exerted outside the membrane than there is within—that is to say, when fluid is exuded into the inter-retinal space of a higher specific gravity than the fluid in the vitreous chamber, detachment of the retina must occur.

In the normal eye the lymph which occupies the interval between the rods and cones and pigment cell layer is much below the specific gravity of the normal vitreous, and therefore there is no tendency to separation between the layers. If, on the other hand, the vitreous be degenerated or shrunken, and the fluid which takes its place approximates more nearly to the specific gravity of the normal lymph, the tendency to detachment is much greater, and any additional rise in the specific gravity of the fluid in the inter-retinal space, such as might be produced by venous obstruction in the choroid, would lead to detachment of the retina by pushing the retina inwards and forcing some of the fluid vitreous forward into the anterior chamber. It is obvious that if the specific gravity of the fluid in the inter-retinal space falls as the result of the relief of the pressure on the veins the fluid may be reabsorbed and the detachment go back into its place. This has been experimentally proved by Sinclair (2), who injected saline solution into the inter-retinal space; it was found that, although it caused a temporary detachment, this rapidly disappeared and the retina became re-

attached, but if serum were substituted for saline solution the detachment was permanent and tended to increase.

Having examined the factors which hold the retina in its place, and the predisposing and exciting causes of detachment, it is now necessary to go to the individual disease, which causes it to illustrate the theories which have been advanced, starting first with those in which a normal vitreous is primarily present.

Detachment can occur with a normal vitreous, as in the case of a *growth in the choroid.* The growth is usually surrounded with a very highly albuminous fluid approximating to the consistency of blood serum, which is of a much higher specific gravity than the normal vitreous. It is probably produced either by the compression of the veins in the choroid, or by the irritation caused by the growth. As the result of this effusion of fluid, the retina is pushed inwards, and a certain amount of fluid is squeezed out of the connective tissue meshes, of which the vitreous is composed, and passes forward into the anterior chamber. This compression of the vitreous may go on until the vitreous is nothing more than a shrunken mass behind the lens, and the whole retina is detached. On the other hand glaucoma may supervene before this takes place.

The time of onset of the glaucoma does not depend on the size of the growth, but rather to the rapidity of the effusion of the highly albuminous fluid behind the detachment. Thus one may find a small growth, with a small amount of fluid, causing glaucoma, due to the vitreous being unable to lose its fluid rapidly, and so pushing the lens forward, obstructing the angle of the anterior chamber. In other cases glaucoma does not supervene until the inter-retinal space is filled with growth or with fluid, and the retina is completely detached and the vitreous represented by a shrunken mass behind the lens. In this case there is a more gradual pressure on the vitreous which has caused it to lose its fluid entirely, and glaucoma does not supervene until the lens is pushed forward by the subretinal fluid or the growth itself.

The detached retina associated with a growth in the choroid is, therefore, undoubtedly exudative in character although it may cause secondary changes in the vitreous.

Retinal detachment in children.—The most common cause of the exudation of albuminous fluid of a higher density than the vitreous is probably venous obstruction in the choroid. This may be due either to a sudden thrombosis of the venæ vorticosæ or obstruction at the exit from the eye. The following is a case of probable thrombosis in a child, and is of such rarity as to warrant a detailed report:

F. L—, male, æt. 8 years. There was a doubtful history of a boy putting his finger in his left eye five weeks before admission to the hospital. The eye was only red for a short time, and there was no further trouble from it. Healthy boy, urine no sugar or albumin. Blood coagulation time normal. Four days before admission on February 10th, 1910, the left eye became inflamed and painful. The left eye showed conjunctival and ciliary injection, T. + 2, pupil dilated, slight hyphæma, some new vessels seen on the iris. No details of the fundus could be seen, except that there was a whitish reflex deep in the vitreous. The patient was placed in bed, eserin instilled into the eye and hot fomentations applied. Under the treatment the eye improved rapidly. In one week after admission a large retinal detachment could be seen, of greenish-white colour, irregular in depth, dotted over with dark retinal hæmorrhages. In one situation a hole in the retina could be seen. The eye was removed under a general anæsthetic and fixed in formalin. An anterior posterior section was made; the angles of the anterior chamber were occluded. The whole retina was detached by a peculiar yellow-green coagulated albuminous fluid of a much higher density than the vitreous. Near the posterior pole beneath the retina—that is to say, in the choroid—there were four darkly pigmented patches of old hæmorrhage in the situation of the point of exit of the venæ vorticose, which were intensely engorged and filled

with soft blood clot. There were several smaller hæmorrhages both in the retina and the choroid (Pl. I, fig. 1). In some situations the retina was adherent to the choroid, whilst in others it had evidently been torn off, leaving small areas behind attached to the choroid, thus accounting for the holes. The sections show extensive massive subretinal and intra-retinal exudation, with hæmorrhages and enormously engorged retinal veins. The choroid shows intense engorgement of the venæ vorticosæ, which were plugged with soft clot; in other parts the choroidal vessels were engorged, but for the most part the choroid was atrophic. In one situation there was a partly organised nodular mass in the exudation (Pl. I, fig. 2).

The author has under his care at the present time a boy with retinal detachment which varies in its extent from time to time for which no apparent cause can be found. Mr. Levy (3) has also reported a similar case.

Batten (4) has recorded a case in a boy, æt. 14 years, with albuminuria. These cases seem to form a distinct group about which comparatively little is known; but from the anatomical investigations of the above cases and from the fact that in Batten's case there was albuminuria, they seem to be due to vascular disease, most probably causing thrombosis or obstruction of the emissary veins of the eye. Four (5) other cases have been reported as "congenital detachment of the retina" in children, but probably belong to this group.

Retinal detachment associated with albuminuria also belongs to the group caused by venous obstruction. The subretinal fluid being of a higher density than the vitreous, it is probable that the vascular sclerosis causes pressure on the veins at their exit through the rigid sclera, causing an effusion of a highly albuminous fluid into the inter-retinal space. The vitreous in these cases is usually normal, or more highly albuminous than normal. If the venous obstruction is relieved the fluid becomes less dense and may be absorbed, the retina then going back into its place, as not infrequently happens in these

cases. On the other hand glaucoma may subsequently intervene.

In myopia, which is usually associated with a fluid vitreous, a fluid exuded into the inter-retinal space of a comparatively low density would cause detachment—possibly this may be due to venous obstruction associated with the stretching of the globe. This is somewhat borne out by the ophthalmoscopic appearance of the veins in the choroid. Some time ago the author collected a number of cases of myopia, and found that detachment of the retina was more frequent in the degree of − 6 to − 14 diopters than in the very high degree of twenty or more. This the author attributed to the fact that in the later stages the sclera had become so thinned as not to cause pressure on the veins of the choroid. If the stretching of the outer covering of the globe were the cause alone one would expect more frequent detachment in the higher degrees of myopia, which is not the case.

Inflammatory exudation from the choroid may cause detachment if it be of a serous nature rather than plastic, but this is necessarily rare, as inflammation of the choroid is usually of the latter nature. A case of sympathetic ophthalmia has been recorded by Von Hippel (6) in which a detachment occurred, with a normal vitreous, from inflammatory exudation. Other pathologists have also attributed detachment to this cause. *Experimentally* a typical retinal detachment has been caused by applying severe cauterisation to the sclerotic.

Detachment after injuries.—This may occur either with or without perforation of the globe. Blows on the eye, without penetration, may either cause detachment by a direct trauma, as has been described in the above experiment, or more commonly probably, by causing venous obstruction following the swelling of the tissues as the result of the injury. After penetrating injuries with loss of vitreous, detachment may occur at the time of the injury from an effusion of fluid or blood into the inter-retinal space, and in this case, when the exudate absorbs and the

vitreous becomes filled with fluid, the retina may go back again into position. This sometimes occurs after operations, especially for glaucoma.

In a case under the care of the author, on which trephining was performed, the patient complained of intense pain following the removal of the disc of sclera, this was followed by a rush of aqueous, and subsequently vitreous appeared in the wound and escaped in considerable quantity. After the operation there was for a week an escape of blood-stained serum through the trephine hole, which was seen to be filled with blood-clot. Although the vision was $\frac{6}{9}$ before the operation, the patient only had perception of light for two weeks subsequently. On examination with the opthalmoscope a large anterior detachment could be seen involving the outer, inner, and inferior portion of the retina. Subsequently the blood was absorbed, and the retinal detachment disappeared, and the patient regained her original vision, namely $\frac{6}{9}$. Another similar case has been recorded.

Detachment of the retina may also occur some time after injury with loss of vitreous, owing to the normal vitreous being replaced by fluid.

There are a number of phenomena, which, personally, the author thinks play only a subsidiary part in the detachment of the retina.

Much attention has been given to the presence of *fibrous bands in the vitreous* as the cause of the detachment by cicatricial contraction. They are not at all infrequent in vascular sclerosis with or without albuminuria (retinitis proliferans), severe inflammatory changes in the ciliary body, and densest of all in congenital detachment. Although these bands are most obvious, clinically and pathologically, they very rarely cause detachment by contraction, but may predispose to it by causing a shrinkage and fluidity of the vitreous. Even in inflammatory cases where the retina is matted up by fibrous tissue, the primary detachment is probably caused by the

effusion of fluid from the choroid. To suppose that fibrous bands can cause detachment one must, firstly, have an adhesion of the retina to the hyaloid. Secondly, they must have such a tremendous pull on the retina to cause an exudation of fluid from the vessels as would tear the retina to pieces, unless there is previously a hole in the retina, so that fluid vitreous can pass into the subretinal space, and in that instance the density and fluid on both sides of the membrane would be the cause, which is not the case. Thirdly, in the congenital cases where these fibrous bands are most marked, the author hopes to be able to show that the condition is purely one of distension of the inter-retinal space with fluid, and that the bands are formed by a typical development of the vitreous and play no part in causing the detachment.

Holes in the detached retina are not infrequent. Much importance has been attributed to them in that the fluid from the vitreous is supposed to pass through them and strip up the retina. As a rule the holes are small, not constant, and most frequently situated in the anterior part of the detachment. They have been attributed to the pull of the bands in the vitreous. This the author does not think is correct; firstly, because the bands are not found with pieces of retina attached; secondly, there are usually no bands near the holes; thirdly, in four cases (7) which have been examined, and in the case described above, the portions of retina which were missing have been found adherent to the choroid. The adherence of these portions of the retina is probably due to the results of old choroido-retinitis, which is more common in the anterior part of the globe; the retina being then stripped up by the exudation it has remained adherent to its pigment cell layer and a hole in the retina is thereby produced.

The colour of the detachment depends upon the fluid behind it. If it be highly albuminous and opalescent, the detachment is white; if clear, the red reflex from the choroid can be seen through it.

Gravitation of the fluid along the lymph space to the bottom of the globe occurs if the primary detachment is in its upper part; especially is this likely to take place if the percentage of albumin is high in the inter-retinal fluid. This is most noticeable in the case of a growth in this region, two detachments occurring, one in the upper part of the globe and one at the bottom. It is not necessarily associated with that condition, however, since the author has seen two cases of two separate detachments, in both of which the eyes were removed, but no growth was found to be present.

Cystic changes in the detached retina are not at all infrequent, and are due to œdema in the inter-nuclear layer probably as the result of pressure on the retinal veins.

Cystic changes do occur in the retina apart from detachment and the author has one specimen in which there is a shrunken vitreous, in which the retina is separated along the inter-nuclear layers, half remaining attached to the pigment cell layer, probably the result of old inflammation, and half remaining attached to the shrunken vitreous.

Cysts in the retina in its normal position may reach a considerable size, so much so that they bulge forward into the vitreous and cause detachment of the retina on either side of them.

The author thinks it is possible that some of the cures of detached retina which have been recorded after operation have really been due to the letting out of the fluid from these cysts which allows it to collapse and the retina to go back into its normal position.

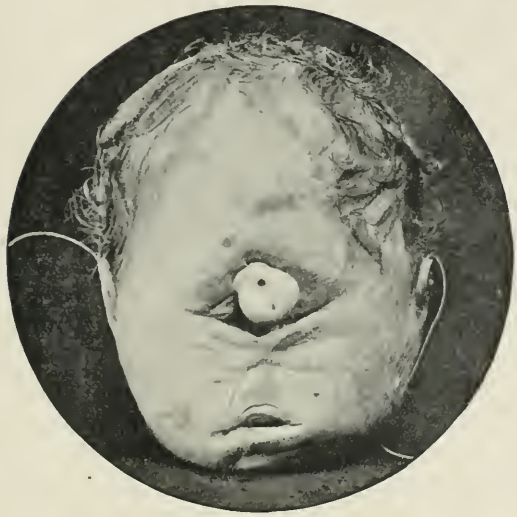
Rapidity of the detachment.—Much discussion has arisen as to the rapid failure of sight with detachment. The most usually accepted theory is that a small detachment occurs in the periphery unnoticed by the patient and that as a result of gravitation or great increase in the amount of fluid the more important part of the retina near the macula is involved, and so the attention of the patient is drawn to it.

Recovery of vision after re-attachment.—The most satisfactory theory is that advanced by Eason (8); it is that the function of the retina only remains in abeyance whilst it is detached, its nutrition being supplied by the retinal vessels and subretinal fluid, and that for its function to be restored the rods and cones must be in contact with the pigment cell layer which provides the visual purple and without which the retinal elements are unable to obtain a satisfactory image.

Congenital detachment of the retina.—This condition is one which is not usually recognised, as it occurs in microphthalmic eyes and is not to be seen clinically in such conditions; other cases of retinal detachment in children have been reported as congenital in origin, but probably belong to the group described on p. 112. The condition also lends strong support to the theory that the detachment of the retina is primarily due to the effusion of fluid into the inter-retinal space. Although the author has used the term "detachment" it is strictly speaking not a detachment of the retina, since the layers of the optic vesicle have never come into apposition.

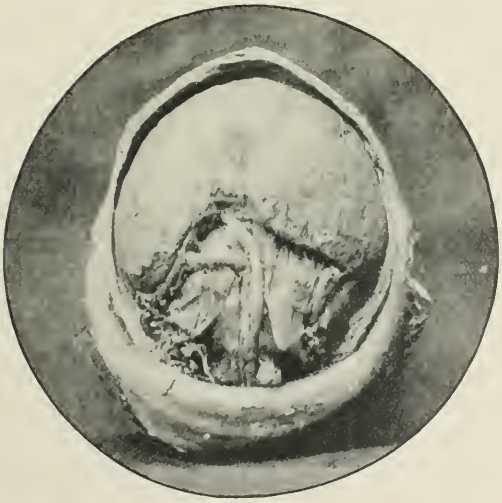
In very early fœtal life when the optic vesicle buds out from the anterior end of the fore brain, it contains a hollow cavity filled with fluid, which communicates and is of exactly the same description as the hollow cavities in the rest of the fœtal neural system. Many portions of these rudimentary neural canals are liable to undergo pathological distension by the fluid which they contain; thus distension of the anterior end of the neural canal will produce a condition which is known as cyclops (Figs. 9 and 10). In this condition the ventricles of the brain together with the optic vesicles undergo such gross distension that the optic vesicles are forced on to the ventral surface of the embryo, pushing forward the fronto-nasal process and themselves uniting together in varying degrees along the line of the fœtal ocular cleft, resulting in various degrees of microphthalmos (Fig. 11). Similar distension, less extensive in character, may take place in other parts of the neural

FIG. 9.



Human cyclops with microphthalmos.

FIG. 10.



Brain from Fig. 9, showing large cerebral cyst as the result of distension of the anterior end of the neural tube.

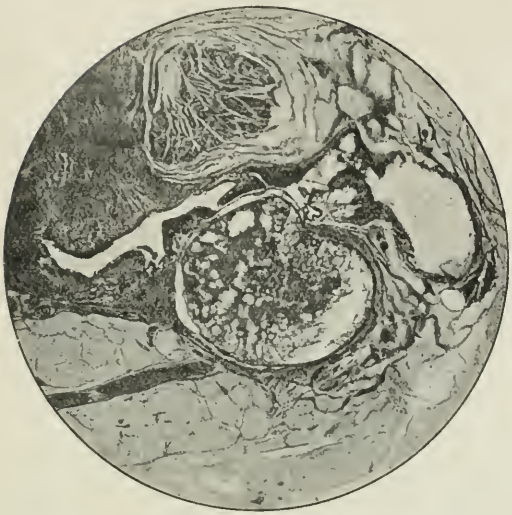
canal. When the brain is more fully formed the ventricles may be affected, especially the posterior horn of the lateral ventricles, which give rise to large cysts occupying the situation of the occipital lobe; these children are born blind (Turnbull [9]).

Similar distension of the lower end of the neural canal gives rise to spina bifida. The amount of deformity to the brain and the eye which arises from these causes depends on the stage of fœtal life at which the distension occurs. Thus the earlier the distension takes place the greater the amount of deformity produced. It has already been pointed out that the distension of the primary optic vesicle with fluid may be associated with distension of the forebrain, but it may also occur apart from this condition, and similarly, as in the brain, it may produce varying degrees of deformity of the eye depending on the amount of fluid which is present and on the stage at which the distension of the primary optic vesicle occurs. The condition which it produces is known clinically as anophthalmos or microphthalmos. If the distension takes place in the early stages with a large amount of fluid there may be no eye apparent in the orbit, but cystic changes are found which may be so large as to form a mass protruding from between the eyelids. Such a case has been examined by the author and shown before this Society (10) (Fig. 11).

In these cases one finds the representatives of the outer and inner layer of the optic vesicle separated from each other by a space containing fluid. In other words, the two layers have never come in contact with each other. The condition is also characterised by a very considerable overgrowth of the pseudo-retinal elements and also of the mesoblastic tissue which goes to form the vitreous. This fibrous tissue can play no part in the separation of the layers of the optic vesicle since no walls to the globe are formed.

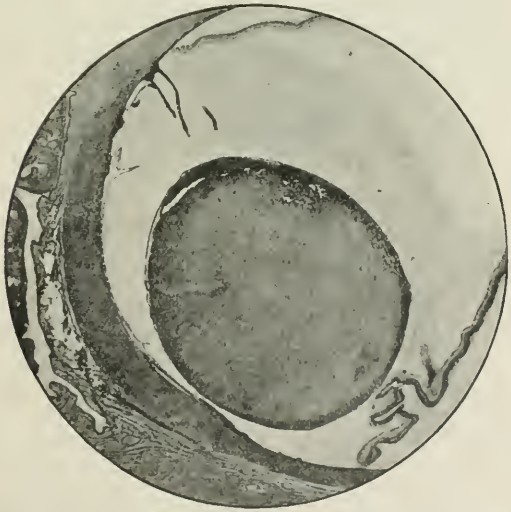
In a less marked stage, where the mesoblastic covering of the globe has been formed, the distension of the primary

FIG. 11.



Microphthalmos, the result of distension of the primary optic vesicle without the formation of a globe.

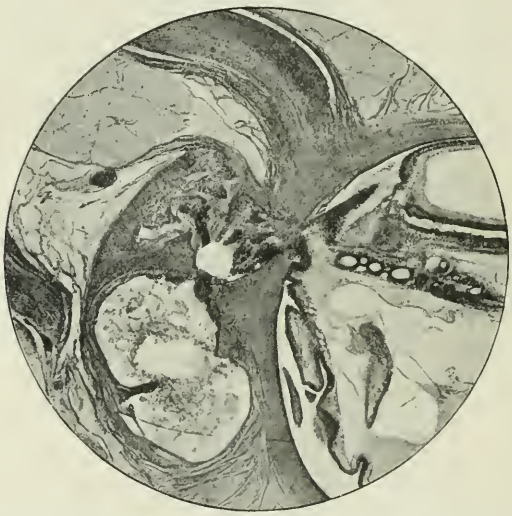
FIG. 12.



Microphthalmos (with cystic globe), showing the separated layers of the optic vesicle, the inner of which is thrown into folds. The lens is congenitally dislocated.

optic vesicle and its external mesoblastic covering may yield locally along the line of the fœtal ocular cleft with the formation of a local cyst, usually situated towards the posterior pole of the globe. Such a case is described by the author in the *Transactions* of the Society (10), and is a common form of microphthalmos (Fig. 13). The inner layer of the optic cup is thrown into multiple folds, never coming

Fig. 13.

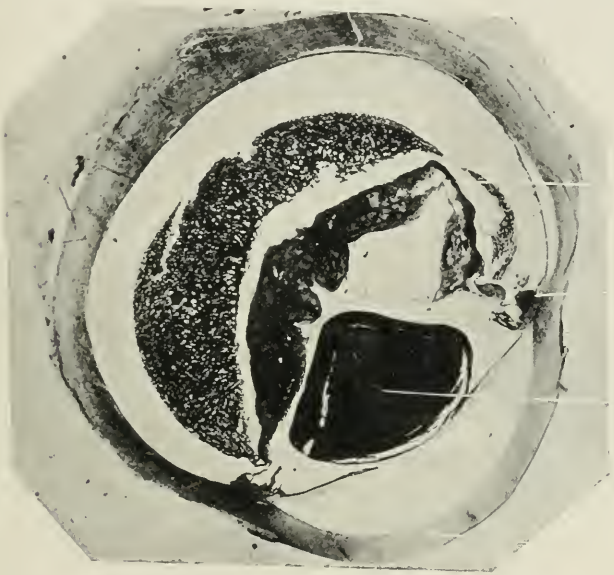


The posterior part of the same globe, showing the cyst and fibrous vitreous.

in contact with its outer pigment cell layer in the posterior part of the globe. The mesoblastic tissue which would form the vitreous is developed as fibrous bands which, unless the condition is considered as a whole, might be thought to cause the retinal separation due to its being held out of place by the bands of mesoblastic tissue, but, as has already been shown, in the case where there is no globe similar fibrous tissues occur. These latter are really developed secondarily as the result of the inner layers of the optic cup being pushed inwards by its distension of fluid, no proper space being allowed for the develop-

ment of the mesoblast into the vitreous. For if the detachment were due to the pull of these bands a negative pressure would be present in the inter-retinal space, and there would be no reason for the formation of cystic protrusion in the posterior part of the globe (Fig. 13).

Fig. 14.



Microphthalmos in a fully formed globe, showing a congenital detachment of the retina. The darkly-stained material in the inter-retinal space is blood-clot formed at the time of enucleation.

Where the globe is still better formed the eye, although slightly microphthalmic, may be a completely enclosed cavity showing no external cysts; but still the inner layer of the optic vesicle is not in contact with the outer. Such a case has been shown before this Society by the author in April, 1914 (Fig. 14).

In this instance there was comparatively little fibrous vitreous, and the anterior portion of the primary optic

vesicle had united together, forming the iris and ciliary body, whilst the posterior part of the retina was separated from its pigment cell layer by fluid. At the line of the union anteriorly there was an overgrowth of the outer pigmented layer to form a congenital pigmented tumour of the ciliary body. No ora serrata was present, the condition being similar to that found during fœtal life.

The author has tried to show here that the essential feature in congenital separation between the layers of the retina, and incidentally of most cases of microphthalmos, is the distension of the primary optic vesicle with fluid, and that the fibrosis of the vitreous is of secondary development and not the cause of failure of union between the inner and outer layer. In other words the condition is one of congenital cystic distension of the primary optic vesicle.

Literature.

Thomson, E.—*Ophthalmoscope*, 1909, p. 605, 1910. This paper is an excellent review of the subject and contains all the antecedent literature.

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(2) Sinclair.—*Journ. Path. and Bact.*, June, 1901, p. 291.

(3) Levy, A.—*Trans. Oph. Soc.*, xxxiv, p. 182.

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(5) Fernandez.—*Report of Spanish-American Oph. Soc. Annal Ocul.*, 1905, t. cxx, xiv, p. 136.

(5) Sulzer.—A doubtful case in a child aged 12, probably as other cases. *Annal. Ocul.*, 1895, t. cxiv, p. 22. This group probably belongs to the retinal detachment in children, not to congenital detachment.

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(7) Elschnig.—*Klin. Mon.*, August, 1892, s. 416.

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(8) Eason, H. L.—*Lancet*, January 2nd, 1915.

(9) Turnbull, H. M.—*Brain*, 1904, xxvii, p. 209, "Bilateral Loss of Post-central Cortex, apparently Congenital, in an Adult."

(10) Mayou, M. S.—"Cyclops," *Trans. Oph. Soc.*, vol. xxvi, p. 281; "Microphthalmos," *ibid.*, vol. xxiv, pp. 341, 348; *ibid.*, vol. xxviii, p. 107.

Mr. Gustavus Hartridge:

I have read with much interest the opening papers on this subject. The essential point to my mind is carefully to separate the various forms of detachment which result from such very different pathological conditions.

The traumatic variety may be divided into two kinds: The first form, which occurs in a healthy eye at the time of a blow or immediately afterwards; this is usually caused by the effusion of blood which pushes forward the retina; sometimes the choroid and retina are both pushed away from the sclerotic; these detachments which are hæmorrhagic in character are always dark coloured; we have all seen cases of this kind undergo spontaneous recovery as absorption of the blood gradually takes place.

The second traumatic variety which has not been referred to in this discussion, occurs in cases where a metallic foreign body has entered the eye and has been successfully removed by the magnet. The patient appears to make an excellent recovery and leaves the hospital with perhaps almost normal vision; months later the vision of the eye gradually fails, and on examination a large detachment is found. I have seen dozens of such cases, and would venture to state that the majority of cases in which a foreign body is removed from the back

of the eye through the vitreous end in this way. No doubt this is the result of changes taking place in the vitreous, causing cicatricial contraction and shrinking, pulling the retina away from the choroid, as fluid is gradually secreted behind the retina to take the place of the diminishing vitreous. No treatment is of any avail in these cases, and improvement seldom, if ever, takes place; personally I have never seen a case of this kind recover.

Most of us have seen cases of detachment of the retina in albuminuria recover with rest and suitable treatment.

The above cases are comparatively rare; the majority of cases of detachment of the retina occur in medium and high degrees of myopia; here the conditions and changes in the eyeball are quite different from the varieties above mentioned. The changes which lead up to and produce the separation of the retina are, the gradually increasing size of the eyeball, which the vitreous is unable to fill, so that the lymph space, between the layer of rods and cones and the epithelial pigment layer of the retina gradually enlarges, being filled by a serous fluid; the connective frame work of the vitreous breaks down, the nutrition of this tissue becomes impaired by the disturbance of the lymph channels through it, leading to increased fluidity and vitreous opacities; the effusion of fluid is necessary to fill up the additional space and gradually causes a separation of the retina; once started the detachment is usually progressive in character; any slight additional accident, as a blow, a fit of coughing or sneezing, or lifting any heavy object, may start the separation. I agree with Dr. Maitland Ramsay that rest in bed, cutting off the patient's fluids, gentle purging and diaphoretics with a pressure bandage, give the sufferer the best chance of improvement which, however, is usually very slight.

With regard to operative interference, I have never seen any good result from it, and, as patients with detachment usually go from one ophthalmic surgeon to another, many cases have come before me which have been operated on by others, and almost always they express

great disappointment at the result of the operation from which they expected so much.

Occasionally one meets with a very optimistic patient who considers he is much improved by operative treatment, as the following case well illustrates.

Two years ago, Mr. X—, æt. 45 years, consulted me for a rather sudden failure of sight in the right eye. His vision was:

R. $\frac{3}{60}$ − 7 D. = $\frac{6}{24}$ T.n.
L. $\frac{4}{60}$ − 7 D. = $\frac{6}{9}$ T.n.

There was a loss of the upper and outer part of the field in the right eye; with the ophthalmoscope a shallow detachment was seen down and in; the detached portion was quite transparent.

He was advised to rest in bed for three weeks, the fluids he was taking were diminished, hot-air baths in bed were given every second day, a dose of sulphate of soda was prescribed every morning, so as to produce a free evacuation, and a pressure bandage was applied at night; this treatment was very carefully and rigidly carried out. At the end of the three weeks his vision in the right eye was $\frac{6}{12}$, the field of vision was about the same, and the detachment seemed less. After this the patient was allowed to get up a little every afternoon, and at the end of a further two weeks he began to resume his natural life. From this time the vision began gradually to decrease, the field showed still further loss, and when last seen his vision was down to $\frac{6}{36}$. The patient was most anxious to know if anything further could be done; My reply was that some surgeons might advise an operation, but personally I would not recommend it. The next news of the patient was that he had gone abroad and had an operation done. Some three months later, hearing that he had returned to London, I wrote and invited him to pay me a visit; he very kindly came to see me, and said he considered he was distinctly better, in fact did not feel much inconvenience now. His vision with a correcting glass was $\frac{6}{60}$; the field had further diminished,

the detachment was certainly no less, and the retina was beginning to lose its transparency; he had evidently become accustomed to his partial loss of the right field and was adapting himself to his new conditions.

I should never advise a patient to submit to an operation for detached retina in myopia; as far as my experience goes no benefit from surgical interference ever takes place.

Mr. E. Treacher Collins:

My object in joining in this discussion is to put on record two cases which I think I am entitled to describe as cures of detached retina after operative procedures.

Case 1.—Mr. L. R—, æt. 37 years, came to see me first on May 22nd, 1913. He began to wear glasses for short-sight when he was seventeen years old, and he had them changed in strength from time to time. In September, 1911, the sight of his right eye failed; he says "he lost the field in it." He was operated on by Dr. Wood in Cape Town, and regained the sight for a time; it again failed, and a second operation was performed in May, 1912. In November, 1912, after an operation on his nose and throat, detachment of the retina occurred in his left eye also. An operation and ten injections were made in it at Johannesburg.

When he came to me he was wearing, in his right eye -3 sph. and -4 cyl. axis down and out; in his left eye $-2{\cdot}5$ sph. and -4 cyl. axis down and out. With these glasses he saw, in the right $\frac{3}{36}$, and in the left $\frac{3}{24}$. The upper half of each field of vision was considerably contracted, rather more on the left side than on the right. Ophthalmoscopic examination showed extensive detachment of the lower part of each retina. There were numerous floating opacities in the vitreous, but no bands could be seen. No hole could be discovered in either retina, and nothing indicating any cicatricial change. After having had my advice he consulted Sir John Tweedy, Mr. Lister, Mr. Paton, and Mr. Knox Shaw, all of whom

confirmed the diagnosis and advised operation. He also went to see an optician, who offered to cure him with glasses.

On July 4th, 1914, I punctured the sclerotic and choroid downwards and outwards with the galvano-cautery, having first turned back a flap of conjunctiva and drawn the eye as far as possible upwards with a strabismus hook inserted beneath the inferior rectus muscle. After the operation the patient was kept constantly on his back in bed with his head low. A week after the operation, though improvement of sight had taken place, some detachment of retina could still be seen. On July 15th a similar procedure with the galvano-cautery was carried out, but this time on the inner side of the inferior rectus. He was still kept in bed on his back. After this second operation the patient at once became aware of a marked improvement in his sight. On ophthalmoscopic examination on August 5th the retina appeared to be everywhere in position, the tension was normal, and with his glasses he could see to read the newspaper. He was then, for the first time since the first operation, allowed to sit up in bed. A few days later he got up altogether, and gradually moved more and more about. On October 2nd I tested his sight in my rooms, and found that the left eye saw with $-$ 4·5 sph. and $-$ 3·5 cyl., 165° down and in $\frac{6}{12}$ partly. His field of vision was quite full. The retina was everywhere in position, the vitreous had become much clearer and nearly free from floating opacities.

In November, 1913, I treated his right eye in a similar way, making two punctures of the sclerotic and choroid with the galvano-cautery, at intervals of a week, one on each side of the inferior rectus. His field of vision in this eye became somewhat enlarged, but at the conclusion of the treatment a shallow detachment was still present.

In June, 1914, his left eye with $-$ 5 sph. and $-$ 3·5 cyl. saw $\frac{6}{9}$, the field was full, and there was no detachment to be seen.

He then returned to South Africa, and I have heard from

him so lately as March 25th, that is, a year and seven months after the operation; his sight still remains quite good.

Case 2.—Henry H—, æt. 33 years, came first to Moorfields Hospital on June 14th, 1909. He said he had always been short-sighted, and that his left eye had been blind as long as he could remember. A week before his visit he had noticed sudden failure of sight in his right eye. He was wearing − 4 sph. and − 3 cyl. axis vertical in the right eye, and with it could see $\frac{1}{60}$. With his left eye he could only distinguish hand movement. The tension of both eyes was normal. He had no nystagmus. Ophthalmoscopically, in the right eye, an extensive detachment of the retina was seen down and out. Its presence was noted by my assistant on the out-patient letter, and by the Senior House Surgeon in the in-patient notes. There were also some opacities at the posterior pole of the lens. In his left eye there was a detachment of retina evidently of long standing.

He was admitted to the hospital, and on June 17th, 1909, a scleral puncture was performed on the right eye at the seat of the detachment down and out. He was kept in bed afterwards for a month, and on July 19th, 1909, left the hospital; the detachment was noted as shallower and the tension as low normal.

The following December I found that vision in his right eye, with − 9 sph. and − 2·5 cyl. 70° down and in, was $\frac{6}{24}$, his field of vision was full, and no detachment could be seen. The condition of his eye has remained the same up to the present time.

From these two cases I have arrived at the following conclusions:

(1) That the detachment of the retina is not an incurable affection.

(2) That it may be cured as the direct result of treatment.

(3) The treatment consisted in prolonged rest in the recumbent position, and the evacuation of subretinal fluid.

(4) That in the first case the operation was an essential feature in the treatment is shown by the improvement of sight having followed directly after it, and by the fact that, though there was detachment in both eyes, the retina only went back into position in the one operated on.

(5) That a cure may be effected, and a full field of vision regained, after a detachment has existed for seven months.

(6) That the presence of floating opacities in the vitreous humour does not prevent a cure being effected.

(7) That by collecting, critically examining, and collating a number of cases which have been reported as cured, further points of interest and importance may be elicited.

Such an inquiry might well be carried out by a committee of this Society.

(A Committee, with this reference, consisting of Messrs. Collins, Ramsay, Paton, Mayou, Story, and Greeves, was accordingly nominated by the President.)

Sir Anderson Critchett, Bart.,

said he would like, in the first place, to express his great gratification that this subject had been brought before the Society. He believed he was right in saying—if not, perhaps his friend, Prof. Landolt, would correct him—that a few years ago it formed the subject of debate at the Société Française d'Ophtalmologie. But for ophthalmologists in England, hitherto, it had been a sort of Bluebeard's Chamber, which they seemed almost afraid to enter. He was glad that now, however, the door was open.

He thought the Society ought to congratulate the three gentlemen who had brought the matter forward, and who had exercised such special care, knowledge, and skill in doing so. It was, perhaps, in obedience to the law of compensation that their friend, Dr. Maitland Ramsay, had been selected to open the discussion, because rather more than a generation ago it was from the second largest city

of the Empire that there came forth a series of results of operations for detachment of the retina, which were so —perhaps he might use the word—Utopian as to excite the wonder and envy of other workers. Several members, himself included, carried out the operation exactly as it was detailed by Dr. Wolfe, but, he regretted to say, their results were not equally satisfactory. A general practitioner, on one occasion, brought a case to him which he did not regard as a suitable one for operation, and he told him so. The doctor then asked him if he had read the wonderful statistics of Dr. Wolfe, of Glasgow. He replied that he had, and that he had followed his method. The practitioner then said: "Then, how on earth can you hesitate for a moment to operate on this case?" To that he replied that the only possible explanation he could give was that there seemed to be an unjustifiable favouritism on the part of the River Tweed, because cases seemed to do so much better north than they did south of that river.

With regard to the question of treatment of detached retina, he believed he had, in succession, adopted every suggested form of treatment, and with varying results. Seating himself on the stool of confession, he had to regret—and doubtless many others would be able to utter the same on their own behalf—that sufficiently careful notes and data of his cases had not been kept. He was confirmed in that view by hearing Mr. Collins' description of his two cases; such precise particulars and full histories were exactly what was required.

When it was known that this subject was to be debated a colleague asked him whether he thought any possible good could result from it. He replied that he thought good would certainly come of it if everybody would tell the truth. There must, for the elucidation of the subject, be the pure, naked, unadulterated truth from all, with as much detail as could possibly be given; only thus could they help each other, and those who would come after them, to achieve the best results, for the disease under discussion was a terrible one.

He had just said he believed he had adopted every known method. But there was one method which he had not adopted, but which was brought very forcibly before him about two years ago, and he felt he would be wanting in his duty if he were to withhold details of it from his colleagues.

He was attending a young lady, of about seventeen years of age, who had had a very extensive detachment, with holes in the retina, as the result of an accidental blow with a lawn-tennis racket. He had her kept in the recumbent posture for some weeks, and was using atropine. He had the advantage of the approval and co-operation of Mr. Lister in the case. At the end of about five weeks he failed to see her one day, and next time he came he found that a foreign oculist had been called in. What that practitioner had done would be best expressed by saying he had grafted a different treatment on to his own. He had stopped the atropine and ordered eserine, and in addition had applied a little mercurial ointment over the lid. Then, in the presence of Sir Anderson, having sat the patient up in an arm-chair, he fired three syphons of water at her eye, begging her to keep her eye open as much as possible while he did so! There was one point which, in justice, he ought not to omit to state, namely, that the water was of a certain degree of warmth, and that it contained a 1 per cent. of chloride of sodium. Having watched this procedure with some amazement—to apply no stronger term—he told the mother of the patient, in the foreigner's presence, that he must withdraw from the case. She expressed her regret, but added that as she had received from her aristocratic friends the assurance that this gentleman was the one authority on the subject in Europe, she would be doing an injustice to her daughter if she did not place herself unreservedly in his hands. As they were descending the stairs the mother asked him what he thought might be the result of the treatment which was to be adopted. He replied that he could speak with no certainty as to that, but that judging from the

vigour with which these three applications had been squirted at the young lady's eye, he doubted very much whether the lens would remain transparent. Somewhat more than a year afterwards the penitent mother brought the girl into his consulting room with a particularly white, glistening lens, and he wished then that he had proved to be a false prophet, instead of a true one, as her unhappiness was very evident.

In conclusion, he wished only to say that if at any time they had felt inclined to be a little proud of themselves on the increase of their armamentarium, and the improvement of the methods of dealing with disease, these cases came along to humble them. He fully endorsed Mr. Treacher Collins' hope that a committee would be appointed, and that the evidence which would come before that committee would be of the most truthful character.

He added that he had operated upon three cases with the galvano-cautery ; in one with an eminently satisfactory result, in a second with a moderate result, while that in the third was absolutely negative.

Mr. J. B. Lawford :

My contribution to this discussion is of microscopic proportions. I wish to bring to your notice three cases of detachment of retina in which the retina became replaced and was retained in position for a number of years. This replacement was accompanied in each case by recovery of partial, but useful vision. No active treatment was adopted ; in all three cases prolonged rest was advised and carried out more or less strictly.

These are the only instances of non-traumatic detachment, under my personal observation, in which non-operative treatment has resulted in a recovery of proved duration.

The notes of all my cases are unfortunately imperfect, and are lacking in some important details, but the broad facts are reliable, and, I think, are worthy of record.

Case 1.—*High myopia; detachment of retina in left eye; treatment by prolonged rest in bed; recovery and retention of good vision; no detachment fifteen years later.*

Miss M—, æt. 39 years (vol. xxiv, p. 27), came under my observation in December, 1907. She had a high degree of myopia and astigmatism, and had worn glasses constantly for many years. Her parents were not "short-sighted," but one grandmother and some other near relatives were short-sighted.

She was an intelligent and highly educated woman, and I felt justified in relying upon her statements concerning her ocular ailments.

The history obtained from the patient was briefly as follows:

She had been a patient of Mr. John Couper for many years, and he had corrected her refractive errors with his usual care.

Fifteen years before (1892), vision failed in her left eye, and she was told she had detachment of the retina. The defect of sight apparently passed off, or at least became less obvious in a short time, but six months later recurred in greater severity, and detachment was again diagnosed. At this time she consulted several well-known ophthalmic surgeons, among them Mr. Couper and Mr. (now Sir) John Tweedy. A discouraging prognosis was given, and prolonged rest in bed was advised. Her statement to me was that when she inquired how long she was to remain in bed, Mr. Couper said (perhaps not quite seriously) "for a year."

She followed this advice quite literally, and was in bed for the greater part of twelve months. At the end of that period the sight of the left eye had undergone decided improvement, and this has been maintained.

Unfortunately, I have been unable to obtain any record of the vision at the time when the detachment was first noted. The earliest note I have is one by Mr. Couper in 1896. Vision at that time was, with correction, R. $\frac{6}{12}$, L. $\frac{6}{9}$.

Eight years later, in 1904, Mr. Couper saw her and noted her vision, with correction, as R. $\frac{6}{18}$; L. $\frac{6}{5}$.

On December 11th, 1907, Miss M— consulted me, and I made the following record:

Patient is wearing glasses, R. $\frac{-14\text{ D. sph.}}{-3\text{ D. cyl.}}$;

L. $\frac{-11\text{ D. sph.}}{-3\text{ D. cyl.}}$

With these, V. is R. $\frac{6}{36}$; Sn. 0·5, slowly at 10 cm.

L. $\frac{6}{18}$ barely; Sn. 0·5, at 20 cm.

Vision in the right eye cannot be improved by other lenses; in the left it is raised to $\frac{6}{12}$ partly, by $\frac{-12\text{ D. sph.}}{-3\text{ D. cyl.}}$

Ophthalmoscopic examination.—R. large myopic crescent; opacities in vitreous; fundus details very imperfectly seen, and condition of macular region could not be ascertained.

L. O.D. pale grey colour, some choroidal atrophy adjoining disc. A large band of pigmentation in retina and choroid runs almost horizontally across upper part of fundus; no white areas or streaks. In the lower part of the fundus is a large, oval, pigmented patch. There is no detachment of the retina in any part of the ophthalmoscopic field.

This patient has been engaged for several years in secretarial and literary work, and has found her eyesight quite equal to the demands made upon it.

She was going abroad shortly after her visit to me, and I have not seen her again.

Case 2.—*Myopia of moderate degree; detachment of retina in left eye; spontaneous recovery some time during the five years following its onset; no recurrence twelve years later.*

Miss A. C—, æt. 36 years (vol. xviii, p. 195) consulted me in May, 1904, on account of headache after prolonged reading. She had a moderate degree of myopia, with some astigmatism, and had worn glasses for distant vision

for many years. She gave a history of failure of vision in her left eye, due to detachment of retina some years previously; at that time she had been under the care of Mr. (now Sir) Anderson Critchett and the late Mr. Nettleship. I have been able to obtain the notes of her case, made by Mr. Nettleship, on several occasions during a period of five years, and these, together with my examination, furnish an intermittent record extending from 1892 to 1904.

Mr. Nettleship's notes, copied *verbatim*, are as follows:

Defect of lower part of L. field in September, '91; got better under medicinal treatment; in November of same year defect came in centre of field; got better about January, '92.

March 22nd, 1892.—R. — 4 D. $\frac{6}{6}$ partly.

L. — 3 to 4 D. $\frac{6}{24}$, letters distorted.

L. widespread detachment of retina, in upper nasal part, reaching down to y.s., refraction + 0·5 D. or less just above y.s., + 2 D. at upper inner periphery.

April 16th.—After two weeks in bed, no appreciable change.

May 3rd, 1892.—L. — 4 or 4·5 D. = $\frac{6}{18}$ correctly, letters still crooked.

July 28th, 1892.—Has been at Kissingen, and is in better health.

R. — 4 to 4·5 D. = $\frac{6}{5}$ partly.

L. no lens now gives quite $\frac{6}{36}$.

Oph. L. Detachment of retina down-out; it is still clear.

June 14th, 1897.—R. $\frac{-5.\text{ D. sph.}}{-0{\cdot}5\text{ D. cyl.}} = \frac{6}{5}$ partly.

L. — 5. D. = $\frac{6}{9}$ partly.

Left field charted and found of full extent.

Oph. (after mydriatic) L. No trace of detachment anywhere, but two long narrow bands of partially atrophied choroid, one curving from outer edge of O.D. up and out, free from pigment and apparently ending above the equator; another much longer, but less curved, from

O.D. up and in, and going beyond the ophthalmoscopic limit towards periphery; it widens a little towards periphery, and is pigmented in some places. Retinal vessels pass perfectly level with both these bands.

Mr. Nettleship's notes end here.

May 5th, 1904.—R. $\frac{-5.\text{ D. sph.}}{-0{\cdot}5\text{ D. cyl.}} = \frac{6}{5}$.

L. − 4·5 D. $\frac{6}{12}$ partly; letters appear distorted and their outlines are uneven to left eye.

Oph. R. Choroid generally thin; no localised atrophy; no detachment. L. No detachment of retina discoverable in any part of fundus; choroidal atrophy much as described in 1897, but there appears to be some cicatricial tissue in choroid and retina along the bands extending from the O.D.

Case 3.—*High myopia; detachment of retina in right eye; recovery during prolonged rest; no recurrence three years later.*

Mr. P. T. R—, æt. 50 years (vol. xxxii, p. 18), consulted me on April 10th, 1912.

Three weeks previously he had noticed flashes of light before his right eye, followed by an appearance which he described as "a mirage effect."

On the morning of the 9th inst. the sight of his right eye failed "suddenly"; "a curtain appeared before it coming upwards over the sight." On the 8th inst. he had spent some hours digging in his garden.

R. does not see $\frac{6}{6}\overline{6}$ with correction of the myopia (11 D.); sees hand movement fairly well all round except downwards and towards nasal side.

Oph. In upper part a very large detachment of retina, loose and steep. It is greyish, but not wholly opaque; the vitreous is fairly clear and free from large opacities.

L. $\frac{-10\text{ D. sph.}}{-1\text{ D. cyl.}}$ V. $= \frac{6}{9}$ partly.

His medical attendant (Dr. McNair) reports that patient has high arterial tension, but is free from albuminuria.

Complete rest in bed was advised, the right eye to be closed by a pad and bandage, and kept atropised.

Patient stayed in bed continuously for three weeks, and for the five succeeding weeks he was in bed nearly all the time. He describes two recurrences of the "curtain" over the right eye during this time, which disappeared on returning to and remaining in bed.

July 13th, 1913 (fifteen months after his first visit to me).

R., with correction $= \frac{6}{36}$.

L., „ „ $= \frac{6}{9}$.

Ophth. R.: No detachment can be made out with certainty in any part of the fundus, but there is some suspicion of a shallow detachment below. Several large floaters in vitreous; lens clear.

April 20th, 1915 (three years after his first visit).

R., with correction, $\frac{6}{36}$ and Sn. 0·5 slowly; lines of print appear curved. T.n., pupil active to light.

L., with correction, $\frac{6}{9}$, and Sn. 0·5 readily.

Ophth. R.: No detachment in any part of the fundus; no atrophy of choroid, or lines of scar-tissue at the site of former detachment; some disturbance of pigment between O.D. and macula; lens clear. A few floating opacities in vitreous.

Patient has had slight attacks of an anginal character, and is living very quietly and carefully under medical advice.

Capt. R. R. Cruise:

I desire to submit for your consideration and criticism a theory as to the possible causation of a certain number of cases of spontaneous detachment of the retina. I mean the type of detachment of the retina that occurs in a healthy eye without assignable cause, such as tumour, injury, or inflammatory exudate.

That theory in brief is that detachment of the retina is frequently a sequela of disease of the retina itself.

The case which first directed my thoughts to this hypothesis is unusual and instructive.

Mrs. O. M— first consulted me on October 27th, 1911. Her brother I had previously seen with complete detachment of the retina in both eyes, which had taken place some years before without any obvious cause; the second eye failed some months after the first, and he was totally blind. He had enjoyed good sight up to the time of his sudden affliction.

His sister, the patient in question, a couple of years before, had lost the sight of her right eye snddenly. On examination I found complete detachment of the retina, and the vision was reduced to hand movements in the temporal field; the vision had previously been good.

She consulted me for a glass for the left eye and desired to know whether it was healthy. Her vision with $-1{\cdot}5$ sph. with $-1{\cdot}5$ cyl. axis vertical $-\frac{6}{6}$. There were a few small specks of retinitis in the very extreme periphery of the lower and outer quadrant of the eye, and one red spot in this area which was just observable, and concerning which I was in doubt whether to regard it as a hæmorrhage or a tear. There was a history of offensive nasal discharge, and I had her examined by Dr. William Hill, who reported "left ethmoiditis." I prescribed Liquor Donovani, and she returned to Ireland.

She returned in March, 1912, complaining of a black floating mist in the upper nasal field, and on examination I found a fairly extensive detachment of the retina in the region of the inflammatory patch, the red spot now being definitely observable as a tear in the retina between areas of pigmentary disturbance with the choroid showing through. The parallax between the edges of the tear and the choroid was very evident. The course of the case was disastrous and immaterial to the subject of this paper.

My interpretation of the above facts is as follows: There was a singular family liability to a low form of chronic anterior retinitis — this patient had some ethmoiditis. That it was a pure retinitis is proved, I think, by the ophthalmoscopic appearance and by the absence of ad-

hesions between retina and choroid when detachment of the retina took place.

A very sluggish chronic retinitis would lead to absorption, thinning, weak cicatrisation and contraction of the area affected, much as in certain forms of chronic bone disease a rarefying osteitis is produced.

The area affected, *i. e.* the extreme anterior portions of the retina, does seem peculiarly susceptible to inflammation or infection. How frequently one finds in an otherwise healthy eye a few specks at the periphery; again cystic degeneration of the retina is practically confined to this region.

The retina lies loosely on the choroid in the concavity of an arc attached firmly only at the ora serrata and the optic disc.

The word tear suggests traction. The points of traction here would be at the ora serrata and optic disc; the fibrosis in the retina would tend to straighten out the curve between those two points, whereas the intraocular pressure of the vitreous would tend to keep the retina bowed out against the choroid; under these opposing forces the only solution possible is for the retina to tear.

Once torn, the edges turn inwards, and the vitreous passes like a hernia through the opening, and detachment progresses.

The subretinal fluid consisting at first of vitreous, possibly disorganised in the neighbourhood of the retinitis, is subsequently added to by exudation from the choroid, and in all the detachments that I have operated on the evacuated fluid certainly resembled such a mixture.

It may well be thought presumptuous to build up a theory on a single case, but since 1911 I have seen several cases which have contributed to substantiate this theory in my own mind.

The tear in the retina is the important point, and my first suggestion is that these tears always take place in the extreme periphery and therefore *can* only be seen in a few fortunate cases when they come within ophthalmo-

scopic range. Incidentally, Nordenson states that the retina is ruptured in all cases of detachment of the retina.

Secondly, the surgeon is generally consulted after detachment of the retina has taken place, not, as in my case, when detachment of the retina was feared.

The combination of anticipated detachment of the retina and its occurrence under observation must necessarily be rare and would account for the overlooking hitherto—as far as I am aware—of this theory of the mechanism of detachment of the retina.

Adopting this theory, spontaneous cure is perfectly explicable, and I note that Steffan explains spontaneous cure by presupposing a rent in a hitherto intact detached retina.

The theory again satisfactorily accounts for tears in the retina, which seem to be the main stumbling-block to certain authorities in accepting the diffusion theory.

Myopic eyes are more prone to retinitis, and naturally the elongation of the globe renders the retina more likely to tear.

Frequently in cases of detachment there is a history of a blow, with late onset of the detachment of the retina, and I submit that the explanation is that, when the retina was not actually ruptured at the time of injury, there was traumatic peripheral retinitis, and detachment of the retina only took place when the retina yielded.

There are points I should have liked to add, and others I should have liked to amplify, but I should be trespassing too much on your time, so in conclusion I summarise and submit that in a proportion of cases of detachment of the retina taking place in an apparently healthy eye the sequence of events is :

(1) A peripheral rarefying retinitis, leading to absorption, thinning, and contraction.

(2) Consequent on the contraction there is a tendency to straighten the arc from ora serrata to optic disc.

(3) This tendency is resisted by the pressure of the vitreous subjected to intra-ocular tension.

(4) And so the retina yields and tears and fluid passes behind it, producing detachment of the retina.

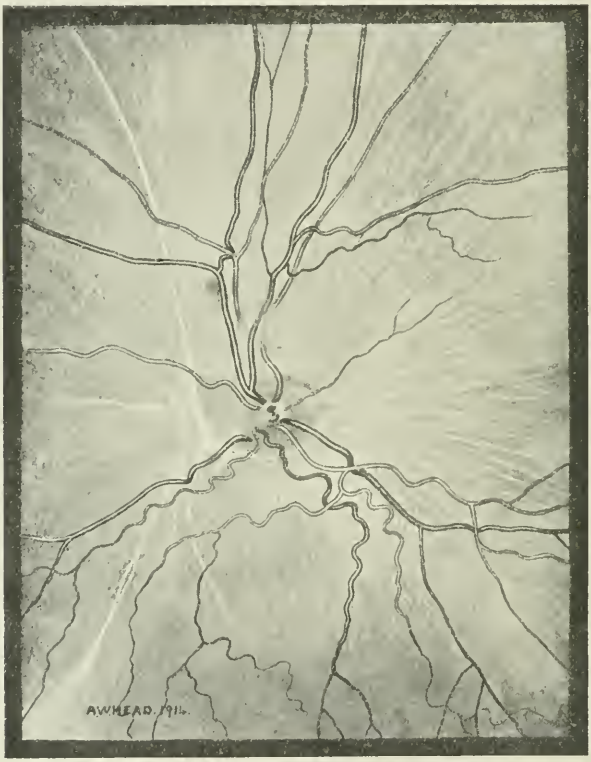
Capt. Cruise added that he desired to say a few words in endorsement of Mr. Paton's optimism with regard to operation. He thought the majority of these cases of detachment of retina would go on to disaster if something of the kind were not attempted. He had been fortunate in one or two instances of operation. He was associated with Mr. Paton at St. Mary's Hospital when that gentleman operated upon the celebrated gamekeeper. Capt. Cruise had himself operated upon several cases, and he did not want to speak of success without being able to bring forward definite statements. A case in his private practice had less than $\frac{6}{60}$ vision, was operated upon four years ago, and now retained vision which varied from $\frac{6}{12}$ to $\frac{6}{18}$. Adhesions between the retina and the choroid were manifest in the places where the cautery was applied to the sclerotic. He operated by double puncture, as he always did, on each side of the rectus, and that patient had maintained vision of $\frac{6}{18}$ to $\frac{6}{12}$ for four years. Another was a case in hospital of a woman who had vision noted as $\frac{1}{60}$, with extensive detachment. He operated upon her six years ago, and she had maintained $\frac{6}{12}$ vision in that eye. Those cases he thought might legitimately be regarded as cured. He urged that vagueness of statement as to improvement after any kind of treatment was to be deprecated. What was required was the brief definite statement as to what the vision was before operation—or treatment—and what it was subsequently, and the interval that had elapsed. Loose generalisations such as that the "patient was benefited" or that the "sight was clearer afterwards" were useless and misleading.

Mr. Sydney Stephenson

said that he, like other surgeons, had seen a certain number of cases of re-attachment of the retina. Some of these had been cured spontaneously and some had

followed quite trivial operations. He would like to say a word or two about two of these cases, one because of the very unusual cause, and the other because of the equally unusual result.

Fig. 15.



Miss G. N—. Left eye. May 18th, 1914.

The first case occurred in a woman, æt. 23 years, who was admitted to Queen Charlotte's Hospital in the last stage of pregnancy. During the day or two that she remained in hospital her urine was found to contain much albumen, and she developed symptoms of eclampsia. She then complained of some defect of sight, and it was at the

FIG. 16.

Miss G. N—, May 18th, 1914. 10 mm. square (white).

time taken to be a case of hemianopsia. After her baby had been delivered the sight failed still more, and Mr. Holmes Spicer was good enough to see the patient, as he, Mr. Stephenson, was away at the moment from London. Mr. Spicer found she had a detachment of each retina. The speaker saw the case a day or two later, and found she had a large globular detachment at the lower half of each retina. The detachment was not transparent; on the contrary, it was of a very dark colour, and strongly suggested that the underlying cause might have been bleeding from the choroidal vessels. That young woman, a week later, presented very few signs of detachment. There was only some rucking of the parts and some slight changes; and she eventually recovered $\frac{6}{5}$ vision with a full field, including even the field for blue. He knew the cure had persisted for many years.

The second case was that of a young woman, æt. 32 years, who had attended the Ealing Hospital in October, 1910, on account of a detachment of the retina in one eye, the cause of which was unknown. Mr. Inglis Taylor was then in charge of the eye department of that institution, and he admitted the patient to the hospital and performed two operations on the affected eye (the nature of which was unknown to the speaker), at an interval of a fortnight between. The patient remained in hospital two months, and was discharged as unrelieved. A year later a note was made on the out-patient paper to the effect that the detachment had increased in size. When he saw the young woman in May last year, *i. e.* $3\frac{1}{2}$ years after she had been operated upon, he could find practically no trace of the detachment which Mr. Taylor had treated. There were—as he supposed there usually were—slight disturbances, such as lines in the fundus of the eye which had been affected, but the retina appeared to be in place everywhere. The curious point was that though the field was fairly full, and the retina in place, the vision in that eye was only $\frac{1}{18}$ of the normal. He exhibited a drawing of the condition of the fundus when he saw the patient.

Mr. D. Leighton Davies :

In the opening papers in this discussion a large number of facts and theories have been placed before us. This indicates the very widely differing conditions which may be found associated with detachment of the retina. We only know of one fact which we can affirm to be common to all forms—*i. e.* the anatomical structure of the retina and its attachments. The apparently exciting causes of, or the conditions associated with, the detachment may be of very varied characters, a point which has been already emphasised.

In view of this discussion, I have looked through my cases for the last five years, and I find that I have seen forty cases of detached retina, including both hospital and private cases; this gives a percentage of 0·5 on the total number of all cases seen.

I will not weary you with details of all the cases, except to point out the low proportion of cases occurring in myopes. Out of the forty cases only thirteen of them occurred in myopic eyes, equal to a percentage of thirty-three. This is, of course, much lower than the usual figures (50–90 per cent.)

In the series of forty cases there are seventeen cases of detachment of the retina occurring in emmetropes or hypermetropes, who show no evidence of any other intra-ocular affection or constitutional disease, such as diabetes or Bright's disease, etc., to explain their onset—*i. e.* they are the so-called simple or idiopathic cases. Of these seventeen cases three happen to be cases of combined extraction of the lens. In these cases the shortest time between the operation and the onset of the detachment was eighteen months, and the longest about five and a half years. One of the cases had had a double extraction, with iridectomy, and a sudden detachment occurred in the right eye. It is possible that in these cases the operation may have been the disturbing factor which led to the detachment.

I therefore exclude these three cases. There remain, therefore, fourteen cases (= 36 per cent.) which cannot be explained. I have looked through my notes of these cases to see if there is any factor common to all. There is apparently none. But there are, however, some points which are very suggestive. Of the fourteen cases nine are in people considerably over forty years of age. In four of these nine I have noted definite arterio-sclerosis of the retinal vessels; another has definite generalised arterio-sclerosis, and yet another, an old lady, æt. 70 years, has frequent attacks of epistaxis and subconjunctival hæmorrhages, fairly good presumptive evidence of arterio-sclerosis.

Granting for the moment that arterio-sclerosis is present in all these older cases, in itself it does not take us much further. In what way does it predispose to detachment of the retina? Arterio-sclerosis is a very common condition in advancing years; detachment of the retina is relatively uncommon. Its influence may be exerted in two ways: (1) It may lead to thrombosis in the choroidal vessels, or (2) it may lead to some change in the secretory function of the ciliary body. In this respect we must remember that arterio-sclerosis is generally associated with increased blood-pressure. This would lead us to expect an increased formation of aqueous and not a diminution. We have an analogy in the kidney, for arterio-sclerosis with heightened blood-pressure is generally associated with increased secretion of urine.

Here I would remark that in several of these cases where the detachment was sudden I have found a tension — 1 or — 2 within two or three days of detachment. Did the hypotonus precede or follow the detachment? I am unable to say, but it does suggest that there was no excessive formation of aqueous.

I am therefore inclined to believe that the arterio-sclerosis leads to thrombosis in the choroidal vessels. An awkward point in this thesis, however, is the fact that five of the fourteen cases occurred below the age of forty, one

of them was only twenty, when, of course, apart from syphilis, arterio-sclerosis is uncommon.

I am very much interested to note that Mr. Mayou reports cases in children where hæmorrhages were shown to have occurred in the choroid.

None of my cases were examined with a view to finding arterio-sclerosis; the association only struck me as I was examining the notes. But in view of these facts, I think there is a useful field for investigation in the relation to the cardio-vascular system in cases of detachment of the retina. I would, therefore, in conclusion venture to suggest that in cases of simple or idiopathic detachment of the retina, the examination is not complete without a careful examination of the cardio-vascular system by a competent physician.

Dr. Maxwell

said it had not been his intention to make any remarks on this subject, and he had not brought notes of his cases.

With regard to the sympathetic detachment, which Mr. Paton spoke of, he thought predisposition would explain the facts quite well.

In reference to treatment, from his experience he would emphasise two points. Non-operative treatment, by rest in bed and bandaging of the eyes, was beneficial in some cases, but the improvement was often not permanent. In the operative treatment he had found some benefit from the use of the cautery, but from other methods, without the cautery, he had had disastrous results.

He wished to describe one case of spontaneous cure. In the month of August, a gentleman, æt. 36 years, consulted him. About a month previously he had seen a London ophthalmic surgeon, who had told him that both eyes were normal and equal. But he had 6 D. of myopia. In the right eye vision was $\frac{6}{6}$ with correction, in the left he could only see hand movements, and there was a detachment of retina in the lower and temporal side. It was a

hemispherical detachment, of whitish semi-translucent appearance, and about 4 or 5 mm. high. The history he gave—and this was the important point—was that he had been in low health for two or three months, had been getting thinner, and had lost all energy. He was told that he was run down, and needed a change. While starting a motor car two days before, he felt as if something had gone wrong in his eye, and he noticed a black object before it. The patient was told what had happened, that the condition was grave, and that it was advisable to seek another opinion. He accordingly went to see Mr. Storey, who confirmed the diagnosis. He was put to bed, both eyes were bandaged, and salicylate of soda was given. On the second day when he came to see him, the patient was out of his mind, and was hunting about the room, partly dressed, looking for the leprechaun, a creature found only in Ireland, of human shape, with active motion and malignant habits. The patient's bandages were off. When he got near enough to examine, he found that the tongue was dirty, the breath very disagreeable, and the only motion he had recently passed exceedingly offensive. A nerve specialist, Dr. Leeper, was called in, and it was decided that the bowels must be attended to; it took six days of assiduous treatment with enemata to get rid of the foul motions. He considered that toxæmia was the cause of the condition in this patient. On the sixth day he was again in his right mind, and nothing wrong was evident in his eye. When he came to see the speaker a few days later, he had $\frac{6}{\frac{1}{2}}$ vision in that eye. A month later, he had $\frac{6}{9}$. He then went home. Six months later a letter was received from him in which he said that his sight was then even better, and that there was no difference between the two eyes. It was a very good instance of spontaneous cure, because apparently everything which ought not to have been done was done. The toxic element in patients with detachment was one to be kept in mind. Though it was not present in every case, it was right to include in this class patients with albuminuria.

Mr. Charles Higgens

said he hoped that the remarks he had to make on detachment of the retina would not hurt anybody's feelings. They might prove amusing, if not edifying. With regard to treatment of detachment of the retina, we were about in the position of the ancient Britons; we had not got much further. If the ancient Britons had detachment, no doubt some of them got well, and at the present day some patients also recovered without treatment. He could remember three cases in particular; he had not the notes, but he read a paper on the subject before the Society seven or eight years ago, and related a case which recovered completely. That patient still saw as well as she did at the time he read the paper. Another case was that of a lady nearly 80 years old. In both these instances treatment was by rest in bed. He did not remember what the vision was.

A third had a detached retina, and the sight was very bad indeed. He had seen her since, and the condition had disappeared entirely; yet, as far as he knew, she had had no treatment whatever. Another patient, whom he had attended for twenty-five years, was on one occasion seen in consultation with Mr. Nettleship, who after he had examined her, said: "Where is the detachment of the retina? She had one when I saw her some years ago." Mr. Higgens had never seen it. She had choroiditis and the vision was deteriorating, but the detachment had disappeared, and he could not see any trace of it. Mr. Nettleship was not the sort of man to say a patient had detachment if he did not see it. The first case during her treatment had the uterus and one ovary removed.

He believed he had carried out for the condition every operation which had been invented; in fact he believed he had invented one or two himself, but he had not seen any operation do good. He had punctured in all sorts of places. He had treated the retina as an obstruction, and

put in silver wire drains. There was one ray of hope left to him, and that was provided by what he had heard concerning the use of the cautery in these cases. He had not used it yet, but he certainly would do so when a suitable case presented itself. Previously, his attitude had been that some got well of themselves, and others, whatever was done or not done, did not improve.

Dr. George Mackay

said he had come to the debate with a deep sense of his ignorance, but with a hope that he might learn something from it. He would like to emphasise the optimism of Mr. Treacher Collins. Not merely Mr. Collins' experience in the two cases he had recorded, but the experience of many others who had spoken to-day, encouraged the hope that detachment of the retina in some of its types was a condition which would be overcome and defeated by medical art. He had seen at least two cases of spontaneous recovery, both in young men, with the details of which he would not detain the meeting. But it was evident that the retina might become attached again and vision restored.

Nothing had been said about one feature of detachment which might be of considerable importance in relation to the degree of restoration. So far as he could gather, it had been held that when the retina became detached it left the retinal pigment behind it. He raised the question whether there were some cases in which the latter came away and adhered to the retina. It had been asserted that the less transparent the detachment, the greater the presumption that the hexagonal pigment had come away with the retina. He doubted the value of this point as a diagnostic sign, because the character of the sub-retinal fluid must largely determine the transparency or otherwise of the detachment.

He would like to know from pathologists whether there were cases in which, on sectioning the retina, it was found

that the hexagonal pigment had come freely away, and others in which it had remained behind. Because, if the hexagonal pigment, which is so intimately associated with the sensory functions of the retina, can come away in due contact with the percipient cells and is not parted from them, it must make an enormous difference to the possible restoration of function when the retina returned to its bed.

He had always avoided operating for detachment of the retina, because he had not hitherto felt any confidence in advising operation. He was brought up under Argyll-Robertson, who warned him against touching these cases. He had, therefore, used rest, atropine, and the application of absorbents. From what Mr. Higgens had just said he felt glad that he had no case of damage to the eye to lay on his conscience. He would like to know whether any member had had operative experience in detachment associated with aphakia, *e. g.*, after extraction for high myopia. Detachment was also one of the dangers following extractions of cataract. He had in mind the case of a young girl on whom he operated for double congenital cataract with great satisfaction many years ago. Recently she returned with a large detachment of one retina, and he asked whether ophthalmic surgeons might deal with these cases as freely as with those in which the lens was present.

Mr. F. Richardson Cross

said that when the subject of this debate was suggested, there were some who felt doubtful whether much good would come of it. It was thought that the best thing was to submit the question of cases cured after retinal detachment to a sub-committee. But it was now obvious that this debate had been of the greatest possible value; and from the opening papers, as well as from the experiences of subsequent speakers, it would now be felt that cases of detached retina were not hopeless at any rate. Some of them got well more or less spontaneously with

rest and simple measures; if these failed, then operation was of value.

Before calling on the openers to reply, he wished to second Mr. Treacher Collins's proposition: "That a Committee be appointed to collect, critically examine, and collate cases of reported cure of detachment of the retina."

(The following gentlemen were accordingly nominated: Mr. J. B. Story, Dr. Maitland Ramsay, Mr. Leslie Paton, Mr. Stephen Mayou, Mr. R. Affleck Greeves, and Mr. E. Treacher Collins.)

Replies by Openers of Discussion.

Dr. Maitland Ramsay, in reply, said he had nothing further to say, except to thank the meeting for the kind way in which his paper had been received. It was a gratification to him to learn that better results had been achieved by operation than he himself had been able to obtain.

Mr. Leslie Paton, in reply, said he had little to add to what he had already expressed. Mr. Jessop had been quoting to him a remark made by Helmholtz, that when he considered cases of detachment he concluded that Nature had made a bad job of the eye, and that he could have made a better job of it himself. Perhaps others also felt that it was not a good mechanical arrangement by which the retina was normally kept in position. His physiological days were now distant, but he could not agree with Mr. Mayou as to the equal tonicity of the subretinal fluid and of the aqueous. He always thought the isotonic co-efficient of blood-serum was higher than the isotonic co-efficient of aqueous. Though undoubtedly colloids had a lower isotonic co efficient than crystalloids, the fact that they did not diffuse through animal membranes gave them a much greater osmotic power. A

subretinal exudate was a fluid with a higher co-efficient than aqueous; there was a different tonicity of fluid on two sides of the membrane.

A remark which was often made in connection with this subject was, if one claimed a cure in a case of detachment, what evidence had one that a detachment ever existed? He was in a position to produce notes about one of the cases which Mr. Collins had cured. In the left eye the upper part of the retina was focussed with — 12 D. The detachment ran horizontally across from the disc on the nasal side, sloping a little downwards on the temporal side. Straight down below the disc the retina was seen with + 12; and to the nasal side + 4. He advised the patient to have an operation done in the lower outer quadrant, and that was the point which Mr. Collins attacked first. He could fully confirm the fact and extent of the detachment in this case.

Captain Cruise's paper had interested him very much, because he thought detachment, owing to direct retinal causes, must be taken into account.

With reference to Mr. Leighton Davies' remarks, he thought contraction of retinal vessels might occasionally play the part of drawing the bowstring away from the arc, and in that way arterio-sclerosis might have much to do with the condition. In reference to Dr. Maxwell's remarks, often an accident seemed to be the cause of a detachment, and apparently it might also be an agent in cure, but on this side of St. George's Channel, though one could still get leeches, leprechauns were not a market commodity.

As Mr. Higgens had once more said, cases seemed to do well when no operation was done, and some did not do well, whatever was or was not done for them. Hence one could not decide what share Providence had in a good result.

Mr. Mayou, in reply, said Mr. Cruise had suggested that the retina was detached by its contraction, like the bowstring of an arc, and that the retina was pushed

outwards by the vitreous. If that were so, the retina should bulge outwards, whereas in fact it bulged inwards. Another point was that the consistency of the fluid on the two sides of the membrane would, in that case, be the same, or, if there were any difference, it would be more fluid on the outer side of the membrane. He did not believe that was the case, but the reverse; the fluid on the outer side contained more albumen. Further, the retina would not be mobile, but drawn tight like a bow-string.

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1. *Cases of healed detachment of the retina.*

By Leslie Paton.

The first of the series of three cases which he was showing was that in which the detachment went back to its proper position after three months' rest, without operation. There were one or two points of interest about it. He had shown the case because of the suggestion that the lines which were evident in it might represent a rupture of the membrane of Bruch. There was one point in the case which he would like physiologists or psychologists to explain, namely, the not uncommon phenomenon of greenish vision frequently occurring in the early stages of detachment. This patient had a very marked greenish cloud in the early stages. Her full correction was 5·5 D., and blurring had been present some time before she became conscious of this greenish light. Her corrected vision in the right eye, when first seen, was $\frac{6}{5}$ in the left, $\frac{6}{9}$ in the right. The detachment was very easily seen. She had six weeks in bed, with hot-air baths, mercury, and potassium iodide, and six weeks on the couch, and with that the detachment completely subsided. That was in 1908, and she had since remained quite well and had done her work as a secretary and librarian. With regard to the idea that it might be a case of the rupture of the

membrane of Bruch, there was the other explanation which Mr. Greeves had suggested, and which was his own first feeling about it, namely, that the lines showed where the detachment began. But there was the fact that in high myopia one met with ruptures of the membrane of Bruch. In this case the line was so clearly defined, with peppering with pigment, that it might be a rupture of the membrane of Bruch. At no time was there obvious vitreous disturbance. This was an easily seen detachment in its whole extent; it was not a bulbous detachment, which overshadowed other parts of the retina; there was no tearing, and the retinal vessels could be traced.

The next case was that of Miss P—, æt. 41 years. When he first saw her, in 1907, she had 15 D. myopia. There were very copious notes about her case before detachment occurred, because she had an old detachment in the left eye of many years' standing, and had been seen at six- to nine-monthly intervals for seven or eight years by various surgeons. Detachment occurred in her right eye early in June; he operated upon her in August. Before the detachment her vision had never been better than $\frac{6}{12}$. It was a large detachment on the nasal side, and the scar of operation was now easily visible. The detachment went back into position on the nasal side, and an almost equally large detachment appeared on the temporal side after the operation; in other words, the retina pulled right across. He operated on the opposite side on September 9th, and she was kept in bed until the middle of October. She then began to get up and left the nursing-home in November. In November, 1907, her vision had not much improved, and there was still a small detachment at the original side. By 1908 the vision had improved to $\frac{6}{18}$. Now, eight years after operation, her vision was four letters of $\frac{6}{9}$ with — 16 D., and J. 1 easily with — 13 D., and there was no obvious detachment, as members would see for themselves.

The third case he now showed was that of W. P—. His first detachment occurred in the right eye in 1874,

owing to the explosion of a firework. In July, 1906, vision in the left eye began to fail, and by April, 1907, he could not read. When admitted, he could see fingers at one foot with the right eye; with the left his vision was $\frac{3}{60}$, though occasionally he could get a glimpse of $\frac{3}{36}$ (*vide* opening paper for further details). He showed the man in the autumn of 1908 at a meeting of the Ophthalmological Society, when some critical opinions were expressed concerning the existence of a little detachment in the lower part of that eye. Nowadays, he would pay no attention to that, but it was one of the first cases in which he had performed this operation, and fearing it might be a fresh detachment commencing, he did a third operation. The scar of the third operation was the lowest visible. Vision at that time was $\frac{6}{12}$; in August, 1910, the vision was $\frac{6}{9}$; in December, 1910, he was shooting partridges. Recently he got thirteen rabbits with thirteen shots. Since 1910 his vision has varied a little between $\frac{6}{6}$ full and $\frac{6}{9}$ full, but his reading vision has always been J, with correction.

Mr. F. Richardson Cross thought the Society would congratulate Mr. Paton on these successful cases. There appeared to be merit in the galvano-cautery method; Mr. Paton had operated three times on one of the eyes. The results were excellent. He examined the gamekeeper and Miss P—, and there was no evidence now of any detachment, nor any abnormality associated with the retina, except the pigmented spots, which were due to the operations.

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2. *A case of subsidence of the albuminuric retinitis of pregnancy with the coincident disappearance of bilateral globular retinal detachments.*

By R. Foster Moore.

A. B—, female, æt. 26 years. The patient was confined in May, 1914. Two weeks before the child was

born the sight was lost suddenly. She had had scarlet fever at 10 years of age and dropsy with two previous pregnancies.

June 22nd, 1914.—There were all the signs of chronic nephritis, the urine contained 0·428 per cent. of albumen and the blood-pressure was 240 mm. Uræmia appeared to be imminent, venesection was therefore performed.

Ophthalmoscopically.—The changes of albuminuric retinitis were very extensive, the veins were distended and tortuous, and much fluffy-looking exudate was present. There was a very large bilocular globular detachment of the lower part of each retina.

August 16th, 1914.—The detachments are present, but are much flatter, and flaccid in appearance.

March 3rd, 1915.—She is very well and can do a hard day's work. There are well-marked signs of secondary optic atrophy, the arteries are very fine with white lines along them. The retina shows a moderate amount of fine, powdery-looking exudate, and a mass of fibrous tissue at the macula. There is extensive disturbance of the pigment epithelial layer. There is no sort of detachment in either eye, and indeed the site of the previous detachments is now the most normal looking part of the fundus. The fields are irregularly contracted but not greatly so.

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3. *A case of subsidence of albuminuric retinitis in which bilateral retinal detachments have disappeared.*

By R. Foster Moore.

W. C—, male, æt. 45 years.

May 2nd, 1913.—At this time the patient attended Moorfields under Mr. Fisher's care, who afterwards transferred him to me. His sight had failed for six

weeks. He had all the essential signs of chronic nephritis; his blood-pressure was 215 mm.

Ophthalmoscopically he had severe retinitis; the disc was swollen and hazy, the veins were engorged, and there was much massive exudate. A small flat retinal detachment was present in each eye.

July 1st.—Retinal changes are more extensive.

September 2nd.—Subsidence of retinitis began, the engorgement of the veins having disappeared, and the detachments were no longer seen. Since this time the patient has been under regular observation every six weeks and has maintained moderate health. Through September last he was working eighty hours a week at a small arms factory.

Vision.—The worst vision recorded was $\frac{6}{36}$ and $\frac{1}{60}$ on September 30th, 1913. On December 1st, 1914, it was $\frac{6}{18}$ and $\frac{6}{60}$.

Urine.—This at one time contained much albumen; on the last two occasions on which it was examined there was not the faintest trace to be found.

Blood-pressure.—The highest pressure recorded was 235 mm. on September 2nd, 1913; from this it has gradually fallen to 190 mm. on December 1st, 1914.

Intraocular pressure.—This, as taken by Schiötz's tonometer, has varied between 13 and 18 mm.

At the present time the fundi show the changes due to a retinitis which has subsided. The discs are pale and hazy. The vessels are small. A small amount of powdery-white exudate is seen. Pigment spots due to pigment epithelial proliferation are present, and there is no sign of retinal detachment. He is still doing an average of sixty-five hours a week.

4. *A case of spontaneous simple detachment of the retina treated by multiple puncture; permanent recovery.*

By Cyril H. Walker.

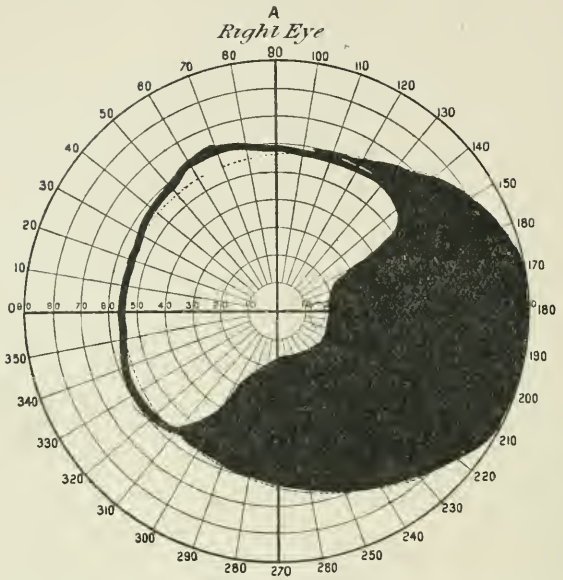
On September 28th, 1901, L. H. R—, æt. 39 years, a typist and teacher of shorthand, came to me complaining that a "film" had come over his right eye two days previously. The night before I saw him he had some pain in the eye, and he was obliged to give up his work.

He had worn glasses for twenty years, and was then using R. − 4·5 D. sph., L. − 3 D. sph., which he had had for some two or three years. There was no history of any kind of injury. Vision: R. $\bar{c}$ − 4·5 D. sph. ⌒ − ·75 D. cyl. axis 70° D. down in = $\frac{6}{12}$ and J. 1 with difficulty. L. $\bar{c}$ − 3 D. sph. ⌒ − ·75 D. cyl. axis 45° down in = $\frac{6}{6}$ and J. 1. T.n. in both eyes. Examination showed a large detachment of the retina above, the summit of which appeared wrinkled, and was seen with a + 12 D. lens. The extreme periphery of the detached area appeared red; the bulging lower edge was white. No rent in the retina or bands in the vitreous could be seen. The disc and lower part of the retina were normal. The lower and outer portion of the field of vision were much contracted (*vide* perimeter chart). [The first nine of the accompanying charts were taken with a 5 mm. square of of white; the tenth chart was taken with 3 mm. squares of red and white.] Complete rest in bed was ordered, the eye was tied up, and atropine instilled daily. At the end of a week he was allowed up for an hour a day, increasing to three hours a day at the end of a fortnight.

By October 12th the detachment had sunk to the lower part of the fundus. The field of vision was altered correspondingly. V. with above correction = $\frac{6}{9}$.

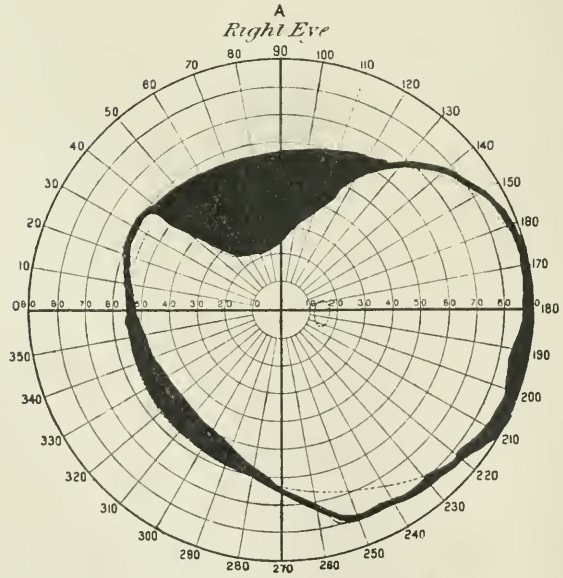
On October 26th the chart showed that some vision was returning in the detached area. He was allowed out, but in consequence of the improvement not continuing and

Fig. 17.



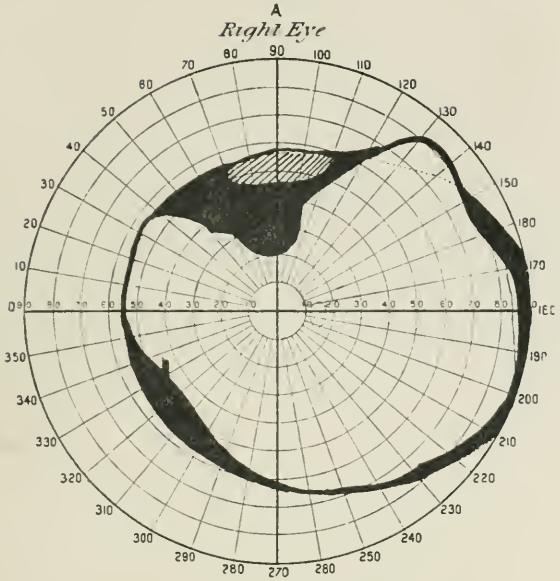
L. H. R—, September 28th, 1901. V. = $\frac{6}{12}$.

Fig. 18.



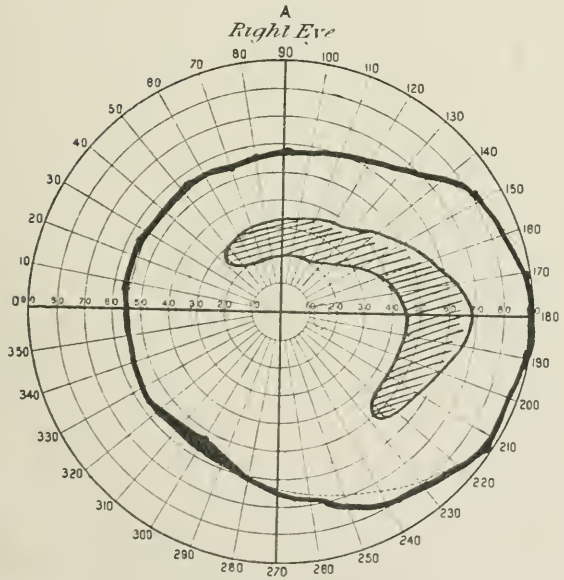
L. H. R—, October 12th, 1901. V. = $\frac{6}{9}$.

Fig. 19.



L. H. R—, October 26th, 1901. V. = $\frac{6}{9}$.

Fig. 20.



L. H. R—, November 6th, 1901. V. = $\frac{6}{9}$.

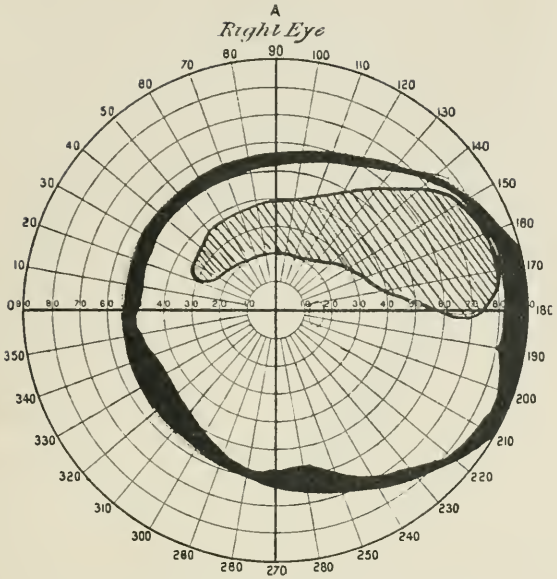
the "film" or defect of field becoming more pronounced, he took another week's rest in bed. The eye was slightly painful at this time, but the vision continued $\frac{6}{9}$ and J. 1.

When examined on November 6th the perimeter showed that the field was normal peripherally, but that there was a roughly crescentic area, in which defects looked as if seen through water. It was possible to map out this area with considerable exactness, because the man was extremely alert and quick in detecting the test object, and took a most intelligent interest in his condition. A doubtful detachment was visible with the ophthalmoscope.

On November 21st a "greenish film" had begun to appear. There had been occasional pain for fourteen days. He had been ordered two blisters on the temple, but no benefit resulted. The vision had gone back to $\frac{6}{12}$ partly and J. 10. On the whole, in spite of resting a great part of the day, and moving about "as if in an egg and spoon race" as he called it, the eye was decidedly worse, and I was not surprised on December 10th to find that the attachment had reappeared below, and the vision was reduced to $\frac{6}{60}$. The perimeter chart of that date showed that the greater part of the upper portion of his field had gone, and that an area of misty vision was forming in the lower and outer quadrant (*vide* Fig. 22).

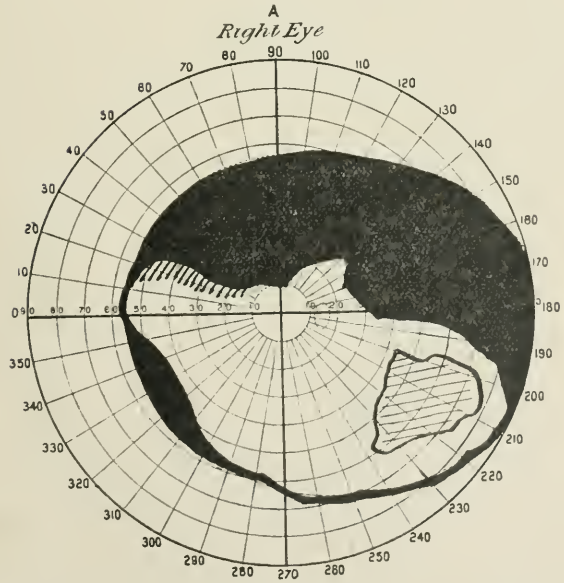
On December 14th, under cocaine, five punctures were made with a rather large cutting edged discission needle, at a distance about 10 mm. from the limbus of the cornea. The needle was plunged in to a depth of 10 or 12 mm. so as to make sure of pricking the retina, but no attempt was made to cut the retina or divide any imaginary vitreous bands. A little subretinal fluid followed the fourth puncture, and as the needle was withdrawn the fifth time it was rotated 90°, and a considerable amount of subretinal fluid escaped subconjunctivally. The patient was kept flat in bed for a week with the eye bandaged. Atropine was used daily, but the eyelids were not opened until the third day after the operation. The moment the upper lid was raised he was convinced that there was an

Fig. 21.



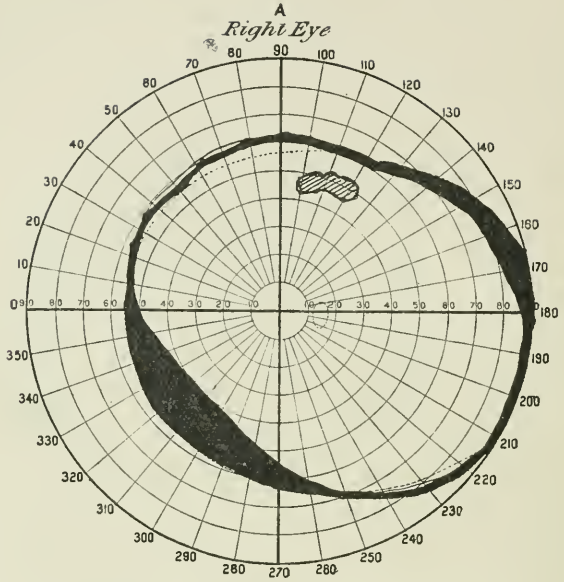
L. H. R—, November 21st, 1901. V. = $\frac{6}{12}$.

Fig. 22.



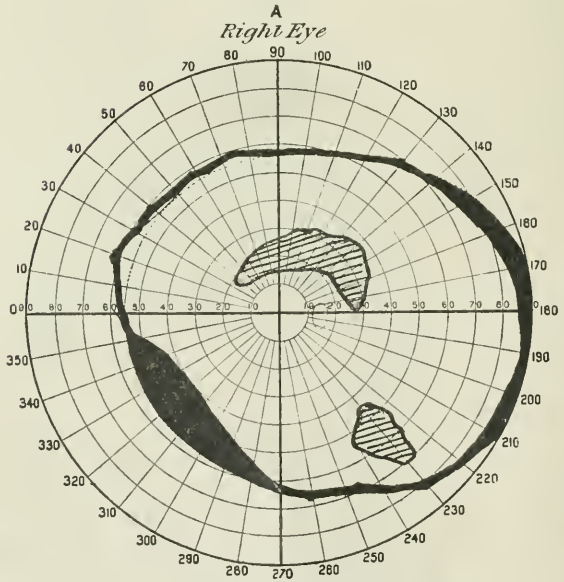
L. H. R—, December 10th, 1901. V. = $\frac{6}{60}$.

Fig. 23.



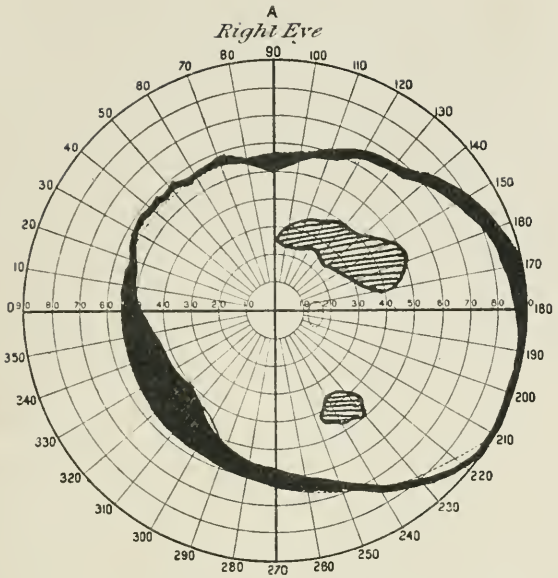
L. H. R—, January 3rd, 1902. V. = $\frac{6}{12}$.

Fig. 24.



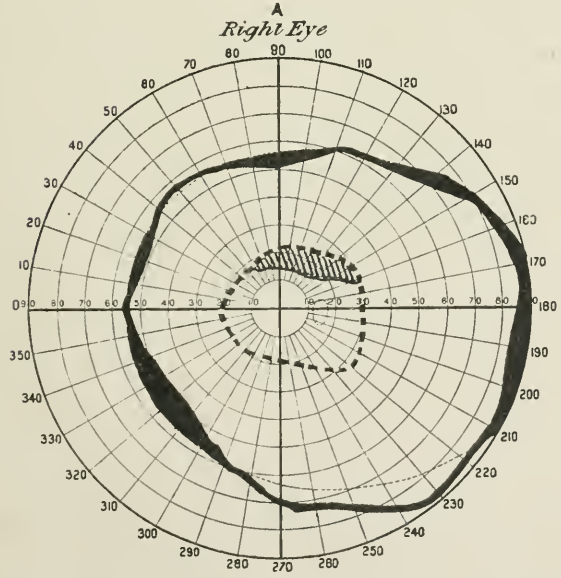
L. H. R—, January 17th, 1902. V. = $\frac{6}{9}$.

Fig. 25.



L. H. R—, February 10th, 1902. V. = $\frac{6}{6}$, two letters.

Fig. 26.



L. H. R—, June 29th, 1910. V. = $\frac{6}{6}$. Continuous line = 3 mm. white.
Broken line = 3 mm. red.

immense improvement in the field. At the end of about a week he was allowed to sit up in bed for meals.

No detachment could be seen on January 3rd, 1902, and the field, rapidly taken, appeared normal with the exception of a small doubtful area above. The vision had improved to $\frac{6}{12}$. A fortnight later (January 17th), after he had been out for short walks, he had occasional pain in the eye, but the vision was $\frac{6}{9}$. The field was normal peripherally, but there were two distinct areas in which objects appeared misty—a white handkerchief appeared greenish. This defect persisted for some months, and even now is slightly noticeable in some lights.

He resumed full work on March 4th, 1902, with no evidence of detachment, a normal field of vision, and vision $= \frac{6}{6}$ 4 letters. Ever since then he has been engaged in teaching shorthand and typewriting. Vitreous opacities have worried him a little, but he regards the right eye as slightly better than his left.

At my request he came for examination on March 26th, 1915. The field is normal. Ophthalmoscopically there are no changes of any importance except streaks of pigment, arranged as in retinitis pigmentosa, in the extreme lower portion of the fundus. With 4 D. sph. $\bigcirc$ − 1·5 cyl. axis 75° down in he sees $\frac{6}{6}$ 5 letters.

Multiple punctures have always appeared to me to be more likely to produce a firm re-attachment of the retina than a single puncture with a Graefe knife. I have done the operation about twenty times on detachments of moderate degree. In one case there was slight chronic iritis, in several there was distinct temporary improvement. In one other case (a man of about 23) there was complete recovery, but I am unable to say if it was permanent.

Mr. F. Richardson Cross asked whether there was any rupture of retina, or any suggestion of fluid or blood in the case; also what was the degree of myopia.

III. DISEASES OF THE ORBIT.

1. *Spontaneous pulsating exophthalmos in a boy.*

By Thos. Snowball.

Charles M—, æt. 13 years, was first seen by me on December 6th, 1914.

The history then obtained from his uncle was to the effect that up to the age of two years he very frequently had "convulsions" (*i. e.*, he "fainted over, there was no struggling during such attacks"), but had had no other illness up to the present one; he had always been very nervous. He was an illegitimate child, his mother dying when he was born, æt. 21 years; it is not known if instruments were used at his birth. His mother's brother, now 48 years of age, was formerly a colliery fireman and when 21 years old he had "inflammation of the brain with brain fever" and has since lost his sight completely.

History of present illness.—On August 30th, 1914, he complained of being sick and vomited pretty freely from time to time for three days. On the 31st his right eye began to bulge forwards; this prominence increased for the next two or three days, so that he was unable to raise the upper lid, and then gradually subsided until it was again possible to move the lid more freely. Dr. Gandy (Accrington), who attended him at that time, has recently informed me that the main symptoms were intense headache, vomiting, and proptosis of the right eye; temperature 101° F. The vomiting was irregular—it would stop for a day or two and then recur. Great malaise; the boy was constantly sighing. He found no evidence of any

cardiac or vascular disease. He thought the case was one of cerebral growth or meningitis.

No history of injury, fall, coughing, or other unusual exertion prior to the onset of the disease, or of any sudden noise in his head at the time of onset can be elicited.

The boy has never complained of diplopia or throbbing in his head.

Present condition.—Patient is a pale, very nervous boy, small for his age; he is very prone to cry, but is said to be smart at school. He has a somewhat prominent forehead; there is no suggestion of syphilis in the upper central incisor teeth or elsewhere. He does not complain of headache.

The hard palate shows no abnormality; the nasal passages are very narrow, but there is no indication of sinus trouble; a little mucopus is seen coming down from behind the soft palate (slight acute rhinitis is present). Illumination of the accessory sinuses reveals nothing abnormal.

Eyes.—Marked intolerance of bright light, accompanied by free lacrymation.

The *right eye* is proptosed straight forwards in a very marked degree (Fig. 27). The movements of the globe are extremely limited—upwards, outwards, and inwards they are practically totally absent. There is very slight movement on following an object downwards, and when the boy is asked to follow it upwards again the globe moves back to its previous position in a slightly oblique direction upwards and inwards. The eyelids are markedly puffy, the upper being of a slightly purplish tint. No circumscribed fulness or new growth can be made out on deep palpation of the orbit at any part of its circumference.

There is slight swelling on the right temple immediately around the outer side of the orbital margin, and in this situation the veins show distinct engorgement. The veins on the right side of the bridge of the nose are even more markedly engorged. There is no tenderness on pressing the globe backwards.

There is some injection of the ocular conjunctiva, but no chemosis.

FIG. 27.

December, 1914.

Left eye.—External appearances and movements normal. R. V. $\frac{6}{12}$; L. V. $\frac{6}{6}$; under homatropine the retinoscopy is

R.— | — +1, +2   L.— | +1, — +0·5

Ophthalmoscopic examination.—Media clear.

Right : Retinal veins distinctly fuller than in left eye ;

O.D. shows slight fuzziness of its margin on the inner half; optic cup distinct and clear.

Left: Surface of O.D. not increased, but while its outline is distinct there is some, though slight, swelling of the inner and upper half of the papilla, judging from the way in which the retinal vessels bend over its surface out of the optic cup, which is distinct.

Treatment.—Syrup. ferri iodidi, with small doses of hydrarg. c̄ cret. intermittently.

Shortly after this date an X-ray examination of the patient's head was made, but the plates failed to reveal any pathological condition in the outlines of the skull.

February 27th, 1915.—The patient has been seen from time to time; little change was noted as regards his eye, although his general condition has distinctly improved. To-day the exophthalmos and œdematous condition of the lids on the right side are more pronounced than in December last, the lower lid being particularly puffy in its outer half. The upper lid shows a general congested appearance with slightly purplish tint; fine capillaries are seen all over its surface, while the larger veins stand out distinctly—more particularly towards the outer canthus, where they are now more noticeable than those in the neighbourhood of the inner canthus. The external lateral ligament of the lids is very easily made out on palpation.

Right eye.—The proptosis is straight forwards, but the globe is on a slightly lower level than the left when the patient looks straight forwards. The external ocular movements are nearly the same as described above—outwards, entirely absent; inwards, only very slight; slightly greater downwards, and on resuming the horizontal position the eye then moves upwards and inwards in the same manner as noted before. On palpating the orbit through the lower lid no swelling or thrill is detected, but on gentle pressure on the upper lid a steady thrill, with regular beats, is felt. This is more distinctly made out on pressing more firmly backwards into the orbit,

when a distinct pulsatile movement synchronous with the pulse is felt, and is shown in the upper lid by very fine, regular movements while the pressure is kept up by the fingers. No tumour mass is felt through the upper lid, except what are evidently engorged vessels in its outer half, which transmit the thrill.

On auscultation a most pronounced pulsatile bruit is heard over the eyelids, but it is also very distinctly audible over the left eye as well as through the facial bones and over the entire cranium; over the right temple a continuous harsh, grating noise accompanies the systolic accentuations, but elsewhere the sound is more like the throbbing of a steam engine.

There is some difficulty in making adequate pressure on the carotid arteries in the neck, but the intensity of the bruit is not altered on pressing on either right or left carotid separately. On making pressure on both simultaneously the bruit is slightly diminished.

There is the same fulness over the zygoma in the neighbourhood of the outer orbital margin; in the upper temporal region a few superficial veins stand out clearly, although a similar vein is noticed in the corresponding position on the left temple.

The caruncle is much congested; the ocular conjunctiva is also congested, the subconjunctival vessels below, and to the upper and outer side of, the cornea being very markedly engorged.

There is still intolerance to light with a tendency to lacrymation.

The pupils are equal, circular, and small; the left pupil reacts promptly to light, but the right only very slightly. The ophthalmoscopic appearances are practically unchanged.

March 18th.—The proptosis and œdema of the eyelids (right) are still more marked (Figs. 28 and 29), and the congestion of the superficial vessels of the lids is greater than before. A fine thrill can be felt all round the upper orbital margin from the root of the nose to the external

lateral ligament of the lids, but there is no visible pulsation of the globe. Change of posture or pressure on the globe backwards with the fingers makes no alteration in the

Fig. 28.

March, 1915.

amount of proptosis; when pressure is made one is conscious of a firm resistance in the depth of the orbit. The photophobia and lacrymation are more intense and are increased on testing the vision or making an ophthalmoscopic examination. The exaggerated action of the frontalis muscle is very noticeable.

Right eye.—V. = $\frac{6}{24}$, Jaeger 4; no hyperæmia of iris. Oph.: Retinal arteries slightly contracted, retinal veins engorged. No pulsation of the vessels on the optic disc

Fig. 29.

March, 1915.

or pulsating movement of the papilla can be observed; there are no hæmorrhages in the retina. The optic cup is quite definite, but elsewhere the papilla is somewhat congested, its upper, lower, and nasal margin being slightly blurred.

Left eye.—V. = $\frac{6}{6}$, Jaeger 1. Oph.: Appearances as

before. The sensibility of the skin of the lids and forehead on the right side is not affected; there is no paralysis of the facial nerves or any vertigo or abnormality of hearing. Heart and lungs normal.

On March 21st an operation was performed by Mr. J. H. Watson, F.R.C.S. Just before it was commenced the effect of pressure on the right common carotid artery was again tested, when it was found that this produced complete disappearance of the bruit and of the thrill felt over the orbital veins. The right common carotid was tied close to its bifurcation, the external jugular and common facial veins being also ligatured. The veins of the neck encountered on exposing the carotid artery were much engorged. Incisions were made through the skin over the inner and outer ends of the upper orbital margin, and mass ligatures applied to the veins in these situations.

For the first two or three days after the operation the œdema of the lids showed little or no change; there was moderate chemosis of the ocular conjunctiva in the region of the palpebral fissure, and on the third day a small opacity was noted in the centre of the lower half of the cornea (due either to accidental abrasion of its surface at the time of the operation, or to the not quite complete closure of the lids after it). On the sixth day the œdema was unchanged, but the proptosis was distinctly less than before operation; the pupil was widely dilated (under atropine), and the corneal ulcer at the site of the above-mentioned opacity showed no sign of spreading. The patient complained of headache and "pricking and jumping" over his left eye and left half of his head, and he "couldn't feel his head," but examination failed to elicit any sign of disturbance of sensation. During the whole of the week he showed extreme intolerance of light. He was very fretful and peevish, but there was never any evidence of paralysis or other cerebral disturbance. On the seventh day (March 28th) the diminution in the amount of proptosis and œdema of the lids was most marked, and no bruit could be heard on auscultation;

indeed, his condition seemed so satisfactory that the pressure bandage over his eye was removed.

On the following day, however, the eyelids were slightly more swollen, although he had passed a very good night and was now free from pain. On the morning of the ninth day he died suddenly. He was in the act of using the bed-pan, when he was heard to give a loud scream and was found in a state of unconsciousness, from which he never rallied.

After the operation the temperature was never higher than 99·2° in the evening (except on the sixth day, after his relatives had visited him, when it rose to 100°); respirations 24; the pulse varied from 84 to 120 per minute (usually between 100 and 108).

Post-mortem examination (March 31st).—The degree of proptosis and œdema of the lids was not quite so marked as was noted on the 28th.

On removal of the cranial vault and the dura mater a considerable clot of blood was found lying over the *left* parietal region of the brain and still more in the *left* middle fossa of the base of the skull. Fluid blood was observed in the other fossæ at the base on both sides but in much smaller quantity. In the apex of the left temporo-sphenoidal lobe a clot of blood was found protruding from a cavity about the size of a walnut, and the brain substance covering it showed distinct signs of softening.

The left cavernous and circular sinuses were markedly engorged and contained recent blood-clot.

The right cavernous sinus was distended and distorted and contained a dark red blood-clot with some white fibrinous streaks in it; the internal carotid of the same side showed a very distinct aneurysmal dilatation which communicated at its upper part by a small aperture with the cavernous sinus.

When the roof of the orbit was removed the contents protruded readily into the opening: there was intense general dilatation of the ophthalmic veins.

The number of cases of spontaneous pulsating exophthalmos is so comparatively small that it seems worth while to put this one case on record, especially as it presents one or two unusual features. The majority of the cases of the idiopathic type, according to the analysis given in the monograph of de Schweinitz and Holloway,* have occurred in women, and in this class the left eye has been somewhat more often affected than the right. As regards age, this case is very much below the average when compared with the thirteen spontaneous cases of a series of sixty-nine analysed by these authors—indeed, amongst these thirteen, apart from an infant of sixteen months, this condition did not show itself until the third decade of life.

The disease was not immediately preceded by any ascertainable predisposing factor, its onset being ushered in by vomiting, which was succeeded by obvious exophthalmos on the following day. With regard to symptoms it is somewhat remarkable that during the whole period that the boy was under observation prior to the operation there was never any history of headache or complaint of a throbbing in the head. At the time of the first examination it was unfortunately omitted to test for the presence of a bruit with the stethoscope, but certainly no palpable thrill in the region of the orbit was detected for two months after, and visible pulsation of the eye itself, the orbital tissues, or the retinal vessels was never present.

With regard to the vision, it was not so acute in the affected eye on the occasion of the last examination as it had been, but the photophobia was then so intense that it was impossible to get the patient to concentrate his attention on the types, and for this reason, too, any attempt to measure the amount of accommodation in the right eye (or to test the vision or examine the fundi subsequent to the operation) was rendered futile.

The intolerance to light is a symptom which does not appear to have been noted in any of the cases reviewed

* *Pulsating Exophthalmos*, 1908.

by the writers above-mentioned. Here it was a most prominent and distressing feature throughout the whole period of observation, and can hardly be explained by the congestion of the conjunctiva (the ulceration of the cornea was a very late complication) or the appearances in the fundi.

The autopsy revealed a condition posterior to the right orbit similar to that which was observed by Karplus,* but while he reported in his case a softening of the brain on the same side as the aneurysm of the internal carotid, hæmorrhage and softening were found here on the opposite side of the brain. This seems to indicate that disease of the blood-vessels at the base of the brain was not confined to the right internal carotid, and the immediate cause of death was apparently due to the softened brain substance giving way before an increase of the hæmorrhage caused by the momentary rise in the intra-cranial blood-pressure.

The immediate results of the operation seemed very satisfactory as regards the obliteration of the bruit, the exophthalmos and œdema of the lids (although the pressure bandage was obviously removed too early), but the time was too short to allow of any real estimate as to the value of the particular line of treatment that was adopted.

I had hoped to be able to append a report on the histological appearances of the wall of the aneurysm, branches of the left middle cerebral artery, and the tissues of the right orbit behind the eye, but this has not been found possible.

In conclusion, I desire to acknowledge my indebtedness to Dr. Crump, for making an X-ray examination of the boy, to Dr. Graham Stevenson (late house-surgeon at the Victoria Hospital, Burnley), for making the post-mortem examination on my behalf, and particularly to Mr. J. H. Watson for performing the operation as well as for his ready and invaluable assistance at the autopsy.

* *Ibid.*

2. *A note on lymphangioma of the orbit treated with X rays.*

By George Mackay.

On February 26th, 1906, I was asked by Dr. Black, of Dunbar, to see in consultation a child, æt. 15 months (Catharine G—), with some unusual condition affecting her right upper lid and orbit. The case was difficult to examine, for the child cried as soon as an observer approached, but it was obvious that the right eyeball was displaced downwards, outwards, and forwards by a swelling which occupied the upper, inner, and posterior part of the right orbital cavity. To make a satisfactory examination it was necessary to put the child lightly to sleep under chloroform. It was then found that the pupils were equal and responsive to light, media clear, fundi normal. While the left eye was rotated upwards in the manner usual under an anæsthetic, the right remained displaced as already mentioned. On raising and everting the upper lid, a bluish swelling, like a large vein or thin-walled, blood-containing cyst, could be seen through the conjunctiva above the tarsal border and extending backwards over the upper fornix. It lay over the upper inner quadrant of the right eyeball, but it was impossible to determine how far it extended backwards upon the surface of the globe. So far as its outline was visible in the thin-walled, bluish area, its longer axis was transverse to that of the orbit, its vertical dimension being narrower, presumably due to compression between the globe and the orbital roof. No pulsation could be felt nor bruit heard. There was no increase of swelling under crying, no proper subsidence under pressure. It could be to a certain extent displaced by compression, but did not actually subside and refill like a meningocele. On palpation it felt, as indeed it was, a soft, thin-walled, cystic swelling. There was no sensation of any cluster

of worms such as a varicocele would present. The rest of the conjunctiva and general textures of the eye were free from inflammation or dilated veins, and when the child was awake there did not appear to be any tenderness or discomfort in connection with the proptosis. No solid mass could be felt. There was a suspicion of some thickening of the orbital periosteum at the upper and inner angle of the orbit, but nothing definite. The whole appearance seemed consistent with the diagnosis of a simple cyst containing blood, or a hæmatoma. There was no history of instrumental delivery or other damage at or after birth, and the mother had not noticed the condition until the child was about a year old.

I decided to puncture the dusky bluish cyst wall at its most prominent part and evacuated about a dessertspoonful of dark bloody fluid. The proptosis therewith disappeared, the eyeball returning to its natural position. A pressure bandage was applied but, owing to the fretful condition of the child, was not well retained, and the maintenance of treatment by pressure was found impracticable at that time.

In a couple of days fluid re-formed and the eye again became prominent. Though subsidence was at once obtained by another puncture, the advantage was only temporary, for the swelling soon recurred.

Frankly confessing that the condition was one with which I had had no previous experience, I arranged for a consultation with Mr. Harold J. Stiles. His wider experience of surgical affections in children enabled him to diagnose the condition as one of lymphangioma with hæmorrhages, such as are met with in other parts of the body where there is loose cellular tissue. In view of the inefficiency and uncertainty of relief by repeated puncturing, he advised excision of the cyst wall so far as practicable, and I decided to leave the operation in his hands, for one did not know what further complication might be found, the source of the hæmorrhage being quite uncertain.

Having enlarged the palpebral aperture by dividing the outer canthus, Mr. Stiles made an incision through the conjunctiva, laid hold of the thin wall of the sac, and proceeded to dissect it out. It was difficult to determine the limits of the extremely delicate wall, which simply consisted of displaced subconjunctival connective tissue, and it was impossible to prevent the escape of the contents at an early stage. In its collapsed state it was not easy to determine whether the cavity was single or multiple, but it appeared to me to consist of more than one distended space, separated or partly separated by thin dissepiments. It was situated apparently between Tenon's capsule and the conjunctiva. The dissection was carried back as far as seemed prudent without interfering with the muscles or the nutrition of the globe.

For a few weeks the condition was decidedly improved, but in the end of March there was some recurrence of proptosis, presumably from fluid gathering once more behind the eyeball in a situation where surgical intervention would have been very hazardous. We accordingly decided to try the effect of X-ray treatment. Seven applications at intervals of a week were given by Dr. Harry Rainy, and during most of this time a bandage was also employed exerting moderate pressure.

By May 19th there was only slight proptosis and slight divergence, with good movements of the eyeball in all directions, no thickening to be felt, nor discoloration to be seen in the orbit. The child seemed bright and happy. The pupil was active. After one or two more applications of X rays, at intervals of a fortnight, the treatment was discontinued.

A year later, in April, 1907, the child was brought to me again, as the parents thought that the protrusion of the globe which, I believe, had never been entirely absent, was increasing. I found slight proptosis and divergence with good movements in all directions, the pupils equal and reacting, the fundi normal. The application of X rays was again advised, and carried out for a short

time, but rather irregularly, owing to the child living in the country at some distance and one of the parents being in bad health.

I did not see the case again until four years later, when, on May 20th, 1911, Dr. Black again referred the child to me. I was informed that the eye had only recently again become prominent. There was now an almost pure proptosis, the globe being pushed directly forward, with only a very slight trace of deviation downwards or outwards. The lids could completely cover the globe, the eye and lid movements were not appreciably restricted in any direction, the pupils were equal in size and reaction, the fundi normal, and the child being now able to recognise letters had vision equal to some letters of $\frac{6}{9}$ with each eye and J. 1. There was a conjunctival hæmorrhage of considerable extent occupying the lower nasal quadrant of the conjunctiva on the right globe, but none of the old dark cystic swelling was visible. There still seemed to be a suspicion of thickening of the orbital wall at the upper nasal angle appreciable to the finger, but no definite growth was otherwise to be felt.

In the absence of Mr. Stiles, Mr. Miles saw the case with me, and as he agreed that X-ray treatment should be again tried, Dr. Hope Fowler (at Dr. Rainy's request) was asked to undertake the further investigation and treatment. I am indebted to him for his careful notes of his subsequent treatment of the case.

Radiographs of the orbit taken at this time showed nothing abnormal in the orbital shadow. Neither neoplasm nor bony thickening was revealed. The X-ray treatment consisted of four applications during the month of *June*, each consisting of one quarter of an erythema dose given at one week's interval. In *July* two applications were made during the last fortnight, each application being one half an erythema dose; and in *September* one full erythema dose was given.

The result was again satisfactory, and no further treatment was employed until *April*, 1912, when, at intervals of

a week, one fourth of an erythema dose was given, followed at lengthening intervals by applications of one third of an erythema dose until the end of the year.

After *January*, 1913, no application was made. The child had an attack of scarlet fever, and the eye is said

Fig. 30.

Photo. showing proptosis of right eye when X ray commenced. Miss Catharine G—. May, 1914.

to have remained well until about *March*, 1914. At that time her father died, and it was not until *April* 25*th*, 1914, that the girl was brought again to Dr. Fowler and X-ray applications resumed. Her appearance a week or two later is shown in the photograph taken in *May*, 1914, which I now exhibit. The relative prominence of the right eye is well marked (Fig. 30).

X-ray treatment was continued now with greater regularity, so that in all seventeen applications were made from *April 25th*, 1914, to *February 25th*, 1915. The dosage varied from one eighth to one third of an erythema dose at each application.

Fig. 31.

Photo. taken April, 1915, after X-ray treatment, showing recession of right eye. Miss Catharine G—.

The method employed in estimating the dosage, and details as to the X-ray values employed to prevent injuries by the rays, will be found in an appended note.

The second photograph exhibited shows well the reduction of proptosis, amounting almost to an enophthalmos, now present, the photograph having been taken on *April 15th*, 1915 (Fig. 31).

I shall conclude with a few general remarks on the subject of lymphangiomata, for it receives but scant attention in the text-books of ophthalmology.

Lymphangiomata are congenital tumours, consisting essentially of distended lymphatic spaces with a thin connective-tissue wall and simple endothelial lining. They are found in many situations, such as the root of the neck, where they commonly extend into the deep lymphatics about the carotid vessels, and may even communicate with the axillary space, or may occur independently in the axilla, in the lip, in the tongue, producing macroglossia in the form of a general enlargement of the tongue or multiple cysts in the mucous membrane of the tongue; they occur sometimes in the arm or leg; they are often associated with other congenital deformities, *e.g.* giant development of great toe or foot; and may be met whereever there is loose cellular tissue, so thus in the orbit. In the mesentery they may cause obstruction of the bowel by stretching or pressure on the bowel, and may be a possible cause of chylous ascites, if the cyst is in connection with the big lymphatics or thoracic duct and bursts. They may crop up in unexpected places, and diagnosis is not always easy. These tumours may disappear spontaneously in the course of a few months in childhood, but, if persisting, an effort is usually made to remove them by dissection. In any situation where this is hazardous or likely to interfere with other important structures treatment by X rays or by radium may be employed with advantage.

A good description of their general pathology may be found in a case of lymphangioma of the arm, reported by Mr. Harold J. Stiles in vol. i of the *Edinburgh Hospital Reports*, published in 1893; and I now exhibit in the epidiascope three plates illustrating his description.

Mr. Stiles points out the existence of muscular fibres as part of the components of these tumours, and it seems not improbable that the benefit of X-ray treatment is due to the stimulation of these fibres promoting contraction of the mass.

I have found one reference to a case of lymphangioma circumscriptum which is said to have recovered under X-ray treatment, reported by Engman and Mook in the *Journal of Cutaneous Diseases,* published at New York in 1913, vol. xxxi, p. 266.

But from a somewhat cursory study of ophthalmic literature I have not come across any orbital case recording the treatment of this condition by X rays. From the case which I now report it is obvious that where the mass is situated in the posterior part of the orbit it would appear that there is a considerable liability to recurrence unless the X-ray treatment is continued from time to time.

Dr. Fowler's opinion is that with the experience which he has gained he could considerably shorten the period of treatment and the number of applications of X rays. My object is merely to draw attention to the fact that X-ray treatment may be distinctly helpful in dealing with these cases, and to invite a further trial of the method.

Note on X-ray Technique supplied by Dr. Hope Fowler.

Methods used in estimating dosage :

Sabouraud Teinte A and Teinte B .	Pastilles.
Holzkneckt quantimeter . . .	Pastilles.
Kienböck quantimeter . . .	A photographic process.

10x (Kienböck) = 5H (Holzknecht) = 1B (Sabouraud).

1B is the dose of X-rays which if exceeded will bring about a more or less severe reaction—a skin reaction.

Tubes used were Müller water-cooled of the largest size.

The X-ray filters used during these applications consisted of 1 mm. thickness aluminium; 3 ply chamois leather; several layers of black photo-paper.

Tube-hardness measured by a Bauer qualimeter registering 7·5 or 8, *i. e.* on the Wehnelt scale 10·5 to 12.

During 1915 2 mm. aluminium was used instead of 1 mm. At no time was there a suspicion of a reaction produced. From various unpreventable causes the treatment was interrupted, and this chiefly accounts for its prolongation over a period of four years.

Mr. J. Herbert Fisher remarked that he had nothing to say about this condition in the orbit, which he regarded as unique. But he recollected having seen, before he devoted himself to ophthalmic work, one or two cases of lymphangioma in general surgery. Those patients were liable to recurring attacks, which were spoken of as inflammatory; they had that appearance. The lymphatic changes were close to the skin surface, and from time to time the skin became reddened and hot, with an increase in the degree of swelling. The attacks subsided, and the condition then reverted to its quiescent stage. During these exacerbations there was some general febrile disturbance. In Dr. Mackay's case there were obviously variations in the volume of the tumour mass in the orbit, and possibly these might have taken place in conjunction with some inflammatory changes in the lymphatic formation. He asked whether a temperature record was kept in this case, or whether there was evidence of local febrile disturbance at any period. If so, the fact might help in the diagnosis of an obscure condition.

Dr. G. W. Thompson said he had had the opportunity of removing three lymphangiomata from the orbit in young children, and in each case, as they were practically encysted, their removal was easy. He made an incision over the skin of the lid, and thus got to the swelling, and a dissector enabled him to separate it readily from a connective-tissue sheath which, in its turn, protected the delicate structures of the orbit from his operative manipulations. In the first of the cases he got the dissector almost as far back as the optic foramen, about 1½ in., and the growth came away perfectly well. At the first attempt he feared to go into the orbit, as a subcutaneous

bluish structure over the swelling made him anxious lest he might be dealing with an orbital nævus from which troublesome hæmorrhage might arise, but at the second attempt he disregarded this bluish structure, and was surprised at the readiness with which this lymphangiomatous tissue could be separated from a surrounding connective sheathing. The second case was a very large lymphangioma, which caused the lower lid to project considerably. In that case an incision was made across the tissues of the lid to get on to the growth, a dissector was put in, and dissection was made all round until a growth somewhat larger than a big bean was brought away. The third patient was a baby, and there was redness of the plica semilunaris. A vertical incision was made over the plica, and the growth was then carefully dissected from the globe. It was a rather complicated case, because there were extensions upwards, downwards, and backwards into the orbit. All the cases healed, and he had no subsequent trouble.

Mr. Charles Wray said that some years ago he had a case of lymphangioma in an infant a few weeks old. The tumour was situated at the upper and inner part of the orbit and was increasing rapidly in size. The eyeball was displaced downwards and outwards and caused great disfigurement and alarm. It was impossible to ascertain the limits of the growth, but, in view of the smallness of the operation field in so young an infant and the fact that the growth encircled the attachment of the superior oblique, recourse was had to the injection of 75 per cent. alcohol. The injections were made in three parts of the growth through the same skin puncture under a general anæsthetic. Three operations were necessary, after which the tumour entirely disappeared and the eyeball went back into its normal position, and at the present time nothing remained but a little fibroid induration, which could be felt beneath the skin. During the injection a bone spatula in the upper fornix was pressed firmly against the roof of the orbit and held there for ten minutes afterwards. The

amount injected was about 10 minims. The resulting reaction was inconsiderable.

Dr. George Mackay, in reply, said he was sorry he could not answer Mr. Fisher's question as to variations in temperature in his patient. The child was brought from the country occasionally and under considerable difficulties, for her father was ill and was of peculiar temperament. He was indebted to Dr. Fowler for having carried out the treatment, and seldom saw the child himself. The father had since died, the child could now be seen more frequently, and the temperature would be noted. He congratulated Mr. G. W. Thompson on the success he had had in operating on his cases, which must have been situated further forward in the orbit. At the operation all the cyst wall that could be found was taken away, but it was too hazardous to dissect in the neighbourhood of the optic nerve. The condition was relieved by puncture of the cyst, and the wall was so delicate that there was little to guide one in making a further attempt at removal. It was recurrence of the proptosis which raised the question as to whether anything further could be done in preference to using the knife. He had had no experience of injections of alcohol for the condition.

3. *The diagnosis and treatment of osteoma of the orbit.*

By C. Wray.

The diagnosis of orbital osteomata may be a very simple matter or one of no small difficulty and uncertainty; indeed, Weil had a case of osteochondroma under his care for five years before he was able to make a certain diagnosis. Bony growths of the orbit as they occur in ophthalmic practice are generally obviously orbital, but to be more exact they are nearly all sinus

growths, though a few arise from the periosteum and subperiosteal tissue of the orbital margin.

The diagnosis in the earliest stages is sometimes impossible, as shown by the case of orbital osteoma in the Museum of the Royal College of Surgeons. It was successfully removed by Sir Victor Horsley from the neighbourhood of the optic foramen, where it was attached

Fig. 32.

Exostosis of left frontal sinus. (Museum of Royal College of Surgeons.)

by a narrow pedicle. Although the dura was exposed the patient made a perfect recovery, but its successful removal is of less interest than the fact that the subjective would precede the objective signs, *i. e.* proptosis would be a later symptom and not, as is so common in other situations, the earliest. As the first step in the investigation of orbital tumours is by palpation, the following remarks may be of interest. The orbit may be examined by means of the finger, the probe, or the spatula, but the probe is a dangerous instrument and can only be used with safety if the extremity is blunt and bent to the shape of a

strabismus hook. Nothing need be said as to examination by means of the fingers. As regards the spatula, it can be used more efficiently than the straight probe and should be of flexible metal, 10 mm. wide, with the extremities 2 mm. thick. Here it may be stated that the average orbit is about 50 mm. deep. The spatula pressed on the skin into the orbit under the supra-orbital notch can be passed to a depth of 15 mm., but by way of the upper fornix 33 mm., which is within 12 mm. of the sphenoidal fissure, and a still deeper examination is possible by means of a curved needle—hollow or otherwise—which can be passed between the bent spatula and the orbital wall. At the lower border of the orbit, skin and fornix measurements are respectively 12 and 16 mm. At the inner canthus the inner wall can be examined for 15 mm. by pressure on the skin and 21 mm. *viâ* the fornix, whilst at the outer canthus the distances are 11 and 22 mm., and as the distance from the orbital margin to the rectus spine is 45 mm. it is thus possible to examine half the entire outer wall of the orbit. The figures given were obtained on the cadaver and verified by a friend ignorant of my own measurements, and not improbably better results can be obtained on the living subject in view of co-operation and the absence of contraction due to rigor mortis.

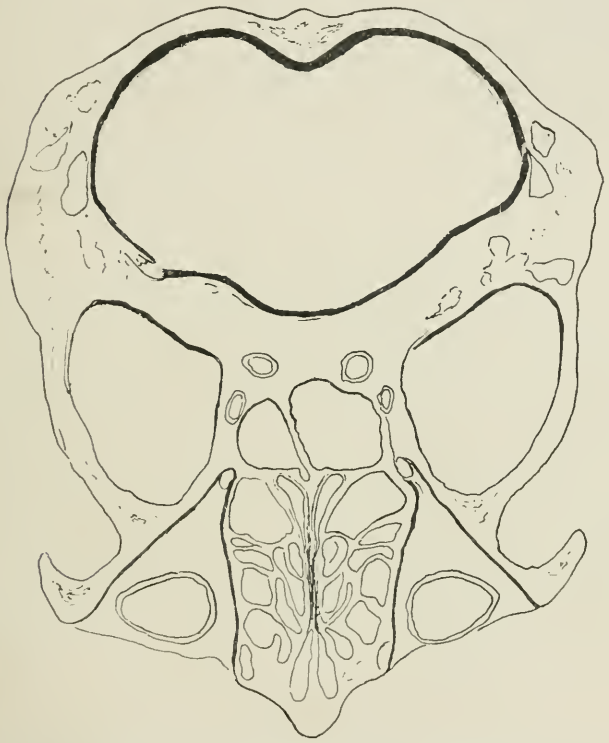
Needless to say, special preparation is necessary for examination on these lines, *e. g.* morphia an hour, and deep injections of novocaine fifteen minutes before the proposed examination. Some years ago I performed a Krönlein on a case of stony hard fibro-sarcoma. Had the examination been made as above it would have been possible to remove a piece for microscopic examination and satisfy oneself that one was dealing with a large growth attached to the anterior surface of the sphenoid up to the spheno-malar suture, as that line is not more than 15 mm. from the margin of the orbit, in other words 7 mm. within the range of the spatula.

The Röntgen rays are essential in elaborating the diagnosis of all orbital growths, but especially the osteo-

matous. Visibility, of course, depends on the density, size, and position of the new formation, and the most easy of detection are the ethmoidal.

In every case sagittal and profile negatives should be

Fig. 33.



Horizontal section of the head, showing the position of the bones likely to interfere with the radiography of the orbit. (From Pirogoff's *Atlas*.)

obtained. The time at my disposal does not admit of a discussion of "posing" in orbital work. It may, however, be stated the technique should vary with the position of the growth as suggested or indicated by the nature of the exophthalmos, and is mainly directed to efforts to avoid shadows from the petrous portion of the temporal

bone, the clinoid process of the sphenoid and the superposition of the sinus shadows. Also the fewer unnecessary lines the better the photograph. It was hoped from stereograms of the orbit and sinus to obtain better definition, but a careful examination of the results obtained is disappointing.

The main points to be noted in a radiogram are:

(*a*) Is the new formation single or multiple?

(*b*) The density of the shadow.

(*c*) The symmetry or asymmetry of the shadow.

(*d*) The presence or absence of shadow lines between the growth and a sinus or the cranial cavity.

(*e*) The appearance of the roof of the orbit.

(*f*) The limitations of the growth.

The difficulty of this method of diagnosis is due partly to the variation in quality of the photographs and also to the possibility of misinterpretation. Whilst as a matter of routine photographs should be taken, it is still necessary to rely mainly upon old-established clinical lines of examination.

Treatment.—Many people believe slow-growing osteomata should be left alone if they are producing no symptoms, but Birch Hirschfeld presents the subject in its true perspective by means of the following calculations from the literature since the adoption of asepsis to the date of his paper in 1907.

His figures are:

Mortality in Cases of Osteoma of the Sinuses.

	Unoperated.	Operated.
Frontal	48·2 per cent.	13·6 per cent.
Ethmoid	80·0 „	12·7 „
Sphenoid	100·0 „	33·0 „

These statistics emphasise the wisdom of operating, and especially whilst the growths are small, but they are unsatisfactory in that they apply to the whole of the published cases regardless of the size of the growth and the

age and condition of the patient. Undoubtedly the mortality would be lower amongst the operated cases had the growth been small and the cavities but slightly distended. The division, too, into ethmoidal and frontal sinus growths is arbitrary, for it is often extremely difficult to be sure as to which sinus was originally at fault. It will be noted that the mortality after ethmoidal and frontal operations is pretty much the same, but as some diagnosed as ethmoidal would be frontal the post-operative mortality of the latter would be less than 13·6 per cent.

The size of frontal sinus osteomata varies within wide limits, and the same remark applies to those of the ethmoid, the anterior cells of which may extend as far as the supra-orbital notch forming the inner part of the roof of the orbit for a distance of 10 mm. from the margo orbitalis. The difficulties of removal depend on the size and hardness of the growth and the nature and extent of its attachment.

One rule that applies to all operations for osteomata of the orbit is that when a membrane exists it and the periosteum should be most carefully preserved unbutton-holed, so as to shut off as completely as possible the field of operation from the tissues of the orbit. It has then to be ascertained by means of a drill or gouge whether we have to deal with a cancellous or ivory growth. If the former we first of all use slight leverage, in the hope it may be attached by means of a narrow pedicle. If much force, however, seems to be needed, we must remove the growth by means of a small saw, or cut it away piecemeal by means of bone forceps, until the seat of its origin is exposed, or its involvement of a subjacent sinus made clear. The next step will be to ascertain the size and shape of the sinus portion, as it is important to know whether it is possible to extract it through the aperture in the orbital wall. For this purpose a strong needle has been used. If we are satisfied that we cannot remove the growth through the already existing aperture, it will be necessary to remove part of the orbital wall, after which

the growth will probably come away more or less easily. In dealing with the ivory variety the principles of treatment are essentially the same. Here, however, we cannot cut away the bone to the level of the orbital wall, so that if leverage fails it will be necessary to perforate the thin bone to ascertain, as far as practicable, the size of the concealed part of the growth. In dealing with ethmoidal cases it is well to bear in mind that the inner canthus is almost exactly over the anterior extremity of the middle turbinated bone, and the anterior ethmoidal foramen is in a line with the cribriform plate. If slight leverage has failed to remove an ivory growth of the ethmoid, and it does not extend more than 40 mm. from the inner canthus after preliminary chiselling around the periphery that is get-at-able, the growth may be wrenched out by means of bone forceps. After removal all blood-clot should be removed, the edge of the periosteum stitched together, leaving a small aperture below for a drainage-tube, the skin incision brought together, and the parts dressed in the ordinary way.

At first it might appear advisable to perform a Krönlein so as to give more room, but this vastly increases the extent of the operation, and does not seem to have been found necessary.

Most osteomata of the orbit begin in the frontal sinus and attain considerable dimensions before they come under treatment. The result is the anterior and inferior wall become very thin, and this conduces materially to successful treatment. Few operators would care to attempt removal in the face of pronounced abnormality of the orbital roof. The growth naturally increases most rapidly where the wall is thinnest, viz. at the floor and posterior wall, but as its passage backward is opposed by the septum separating the sinus from the anterior ethmoidal cells, and in addition by the floor of the cranial cavity, it is exceptional for the growth to come in contact with the dura. Judging from published cases suppuration of the

sinus rarely complicates osteoma, and this simplifies after treatment.

The operation consists in making an incision from the beginning of the outer third of the orbit to the inner canthus. All the soft tissues are then dissected from the bones, unbutton-holed if possible. An attempt should then be made, by means of the knife or needle, to obtain some idea of the limits of the growth, after which bony flaps, as in Golvine's osteo-plastic operation, should be made and the growth removed without undue violence. After removal it does not seem necessary to deal specially with the infundibulum, and it is customary merely to bring the incision together by means of superficial and deep sutures and then insert a drain. If there is an unhealthy mucous membrane, the whole of it should be removed and a piece of gauze inserted, which should be rung out in 20 per cent. argyrol or the compound tincture of benzoin. The dura mater may be exposed, but if the technique of the operation is properly carried out the danger does not seem at all great.

Mr. C. Higgens said he had had one or two cases of ivory exostosis, and had managed to get them away fairly satisfactorily; he had not seen bad results in any case. In one case he did not succeed in getting out the whole of the tumour.

4. *A case of sarcoma of the socket.*

By J. Gray Clegg.

The patient, A. M—, first came under my own personal care on August 3rd, 1910. He was then æt. *circa* 40 years. I had previously assisted the late Dr. David Little in removing the globe. He had been wearing an artificial eye for some seven years, and had recently found difficulty in

keeping it in position. On removing the Snellen eye I found a large granular red mass in the outer portion of the socket. It extended more than one third of the transverse diameter of the palpebral fissure from the outer canthus. It was pedunculated, the pedicle being about one fourth of the area of the mass itself. The patient had only noted its presence for a few days, but almost certainly it had been growing for some time. On the next day I excised the mass, which was friable, and within a week healing had taken place.

Microscopical specimens were prepared and submitted to Prof. Lorraine Smith and Dr. Powell White, who at first thought the tumour to be an endothelioma, but later decided that it was not malignant, but rather a kind of granuloma with included epithelial cells similar to those often seen in the healing of abdominal wounds.

Relying on this opinion, I did not send for the patient, and he refrained from coming to see me until November 8th, when I discovered a large fleshy mass in the same situation. It was hard to the touch and broadly pedunculated. The patient's health was excellent, and there were no glands involved. I then submitted a section of the original growth to Mr. Treacher Collins, who described the growth as undoubtedly a sarcoma mainly composed of oval spindle-shaped cells with numerous vascular channels coursing through it, some of them with thin walls and others which appeared to be bounded by the cells of the tumour itself.

Mr. Collins also saw the patient and strongly advised removal of the whole orbital contents without loss of time.

Mr. Herbert Parsons also examined the growth and advised the same procedure. He did not commit himself definitely to an opinion as to its nature beyond that it was malignant.

I also submitted the specimen to Prof. Walker Hall, of Bristol, and he wrote me saying: "I consider it malignant in character, and belonging to the group of peritheliomata. It does not seem to be a rapid grower, so that with the

entire contents removed the patient should be free for some time."

Sir John Tweedy was also consulted by the patient, and he advised exenteration of the orbit.

I believe that it was Mr. Parsons who pointed out that the cause of the tumour was the presence of a crack in the artificial eye which had been worn by the patient continuously. I present the specimen. The crack is easily discoverable, but I must confess that I, myself, had overlooked this possible source of irritation.

Acting on the advice just given, which was in consonance with my own view, I made an incision through the skin of the lid 3 mm. from the palpebral margin all round, and then dissected up the skin to the orbital margin. The incision was then deepened right down through the periosteum, and the whole of the orbital contents removed in one mass. (Specimen shown.)

Excellent healing took place, the greater part of the cavity being covered by the skin of the lid, which was drawn inward by the contraction of the new tissue lining the orbit. With this, and with the aid of a few grafts taken by the Thiersch method, a perfectly dry socket resulted.

Later, I advised the wearing of artificial lids with an artificial eye together with framed glasses. A very excellent cosmetic result was produced, but, although the only trouble to the patient was the inserting of the artificial lids with a little spirit-gum daily into the socket, he preferred to wear a black patch, and continues to do so.

No recurrence has taken place, and the patient is still in perfect health.

Prof. Walker Hall has kindly furnished me with the following description of the microscopical appearances of the recurrent growth :

"The sections present the appearances frequently met with in orbital sarcomata. The cells are large in type and show numerous mitoses. Here and there a few smaller cells occur, and occasional polynuclears are seen, but on

the whole the cells are typical of orbital growths. The arrangement is atypical, although there is a tendency to form layers around a central blood channel, which is suggestive of perithelioma. The blood is supplied partially *via* the central channels, and partially *via* simple passages between the cells. Scattered hæmorrhages are present. Foci, exhibiting degenerative changes, are infrequent, and the cells generally show average vitality. The appearances are those of a slow-growing neoplasm, and suggest the prognosis of delayed recurrence."

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5. *Osteoma of orbit ?*

By C. Wray.

The patient first came under my care towards the end of 1914, suffering from a slight proptosis and induration at the upper and outer angle of the right orbit, the vision in each eye being $\frac{6}{6}$ and the fundus normal. The globe was displaced slightly downwards and its movements were free in all directions excepting upwards and outwards, where there was a very slight diminution. Both pupils were equal and reacted well to light and accommodation. She had never suffered from diplopia, nor had she had pain or neuralgia. The history was that the prominence of the eye was first noticed twelve months previously. Radiography gave a negative result.

She was next seen on February 8th, when the disc appeared congested and the vision had become reduced to $\frac{6}{18}$.

Recently the sight had become much worse, and there was considerably more proptosis, and the movements of the globe were distinctly limited.

There exists at the situation of the lacrymal gland a hard nodular growth, and this apparently implicates the outer wall of the orbit above the external palpebral

ligament. The stereogram shown is one by Sir Mackenzie Davidson. It shows a very suspicious appearance in the roof of the orbit best seen under somewhat feeble light. Beyond doubt there is considerable displacement upwards of the roof of the orbit and a considerable departure from its usual outline.

IV. DISASES OF THE EYELIDS AND CONJUNCTIVA.

1. *Eye symptoms in Kaposi's disease, xeroderma pigmentosum.*

By F. Richardson Cross.

Freckles (lentigo), an affection of frequent occurrence and great variation, is due to localised increases of accumulations of the pigment cells in the rete mucosum.

The spots usually occur almost entirely on the face and arms, and on parts of the body surface which have been exposed to the sun (Ephelis). They first appear on the approach of summer, and, as the cold weather supervenes, they tend to pass away.

Though the spots may begin to show at any time of life, they are seldom seen *before the fourth year*; they are much more common in childhood, though they may be present in later life. The spots are unsightly, but they rarely cause other trouble or lead to any further complications.

Spots like freckles which appear in the first year or two of life are very often the precursors of the grave and rare form of skin disease which was described by Kaposi in 1870, and which goes by his name.

The pigment spots are then accompanied by dryness and atrophy of the skin, and by other more serious changes in it. The condition has been well called "lentigo maligna."

The freckles usually appear very early in life, and occasionally only a few months after birth. Two or

more brothers, or sisters (usually the same sex), are frequently affected. The disease probably depends on a congenital predisposition to a faulty innervation, which tends to irregular nutrition of the skin, or to abnormal changes and formations in the vascular and pigmentary portions of its papillary layer.

Well-defined dark or lighter pigmented spots appear on the uncovered exposed portions of the head, shoulders, and limbs—in addition the skin begins to shrink, showing white atrophied areas of contractions and thinning, hence the name preferred by Radcliffe Crocker ("*Atropho-derma pigmentosum*"). The skin is very dry and rough in the affected parts; it feels thin and tense, and stretched, resembling parchment. Kaposi prefers the name "*Xeroderma pigmentosum*" which he originally gave it, and by which the disease is usually known.

With time other forms of deterioration show themselves in the epidermis and papillary layers. Fine capillary and vascular telangiectases develop.

Some of the pigment spots coalesce and form moles. Warts and papules grow; some on fine pedicles, so that they easily fall off, others with broad bases, which may ulcerate and discharge with scabbed sores, or may increase in size and form tumours, which take on malignant changes.

The new growths show various types of tumour tissue —granuloma, epithelioma, sarcoma, myxoma, angioma; and in a growth from a case of Crocker's, examined by Politzer, all these types were seen combined together.

The disease is confined to the integuments, the tumours do not penetrate below the corium, and there seems to be no tendency to implications of the lymphatics or to metastases of the internal organs.

The prognosis is very hopeless. No definite improvement has been seen in any of the hundred or so cases which have been reported, but constant attention to treatment of the local manifestations as they arise seems to keep the disease in abeyance. Some patients live for

many years, but on the other hand early death may occur, from deep ulceration toward the brain, or other vital parts, or from exhaustion or hæmmorhage.

The most marked changes are seen in the face. The muco-cutaneous surfaces at the mouth and eyelids become thinned and shrinking, or fissured with superficial ulcers, and the lips become white and mottled with red vascular spots; any of the pathological changes earlier mentioned may occur. The ears and nose edges seem particularly liable to growths and ulceration—and as the lids and surface of the eyeball are continuous with the skin of the face, they may also participate in any phase of the disease.

The pigmentation and thinning of the eyelids is associated with a dry and rough conjunctival surface and with shrunken eye-lashes.

On the lid edges may occur warts, or inflamed papules like styes, which tend to ulcerate. Nodular swellings may grow on the skin surface of the eyelids, and towards the caruncle.

The ocular conjunctiva is usually hyperæmic from a general congestive fulness of the vessels, or with acutely inflamed patches which often run to the edge of the cornea.

Some cases are said to have shown pterygium, but the nodules are probably more frequently allied to phlycten or pinguecula. The edge of the cornea seems particularly prone to implication; a patch of hyperæmia simply, or complicated with swelling and deposition of cells which may absorb again, or may form corneal ulcers, or else lead on to the formation of a neoplasm.

The cornea itself becomes hazy from infiltration of its substance, or from swelling of the epithelium. In these cases there is photophobia and blepharospasm.

Ectropion is frequently present, and leads to opacity or ulcer of the cornea; but in many of the cases that have been depicted—Crocker's and others—the eyes are open and the light seems to be well borne.

The optic nerve and the intraocular structures are probably not affected excepting as a late complication of more superficial lesions—and even iritis is probably seldom present unless the cornea has first become very deeply affected.

Although the cases cannot be cured, persistent treatment gives much relief. The discharges from the irritable conjunctiva, or from ulcers, must be constantly washed away by soothing solutions so as to avoid irritation and eczema of the neighbouring skin.

The growths need to be repeatedly removed and eradicated and the ulcers scraped. The application of massage to the eyeball empties the superficial blood-vessels and diminishes the swelling of the cornea. I have applied a calomel ointment with good results and used it as a medium for massage. Cocain is comforting, but damages the corneal epithelium ; atropin appears to aggravate photophobia, and as iritis is probably not an early symptom, and rarely present until the eye is very seriously damaged, is not usually needed.

Coloured veils and ointments may protect the skin.

Dr. Whitfield has much improved one of the patients mentioned by the application of X rays.

A case of xeroderma pigmentosum, with post-mortem examination, was reported by Dr. T. M'Call Anderson, *Brit. Med. Journ.*, June 8th, 1889. A boy, æt. 9 years, affected with the disease showed freckles, abnormal vascular areas, and a few rounded nodules scattered over the face ; the largest of these was situated on the inner side of the left eyeball and was removed by Dr. Reid.* It soon reappeared, extended rapidly in the orbit and destroyed the sight ; finally the eye had to be enucleated. There was no return of the disease or further material change in his condition for four years, when the disease again became active on the other side of the face ; rapid malignant ulcerations destroyed the skin on the right side of the face, with the bones and cavities underlying, so that

* Dr. Maitland Ramsay made microscopic sections of this eye.

at the end of a year the boy died when only thirteen years of age.

The post-mortem examination showed that the right side of the face was eaten away into the nose and pharynx, and into the anterior middle fossa of the skull, but there was no affection of the glands or of the internal organs.

Dr. Brayton records two cases, a brother and a sister, each of whom was attacked between the fifth and sixth months of life. The boy died of a malignant disease of the face, with ulceration at nine years old. The girl, although showing the typical signs of the disease, continued to be well developed, and otherwise in good health when last seen at the age of fifteen years.

I have had under my observation during the past year two cases of Kaposi's disease. A brother, H. C—, æt. 11 years, and a sister, M. C—, eighteen months younger. They were the children of healthy parents—cousins.

The boy was born in Ceylon, and when æt. 9 months, coming to England by the Red Sea, he became very sunburnt (erythema solare). His skin became scarlet like a lobster, and a little later the face, neck, and shoulders, and arms became dotted over with pigmented spots of a brown yellowish colour; the skin also became thin and shrunken, and other symptoms of the disease followed.

The girl was born in England, and went back to Ceylon as a baby. Her journey was made when there was no special sun glare. She reached Ceylon at four months old, and until nine months old she was quite well. Then freckles began to appear on the exposed parts of her body, face, neck, hands, and a little on the legs. The skin looked dry and parched, and her general health became bad. At a year and a half old, a small nodular growth appeared on the cheek, but fell off, leaving no scar nor ulcer.

The boy had the first growth between two and three years old, both children continued to be affected and similar pendulous growths fell off.

The girl in this respect appears to have been the worse of the two. She had many flat papular swellings in various parts, the nose, cheek, and forehead. The edge of the eyelids was also implicated, producing eversion.

The boy also had many papular swellings, but the tendency to them was less well-marked, and they usually fell away. He is now practically free from them, but is much pigmented; while in the sister growths have been very troublesome, remaining fleshy and not tending to dry up. Some of these Dr. Whitfield has treated with X rays, which have also healed up some ulcers on the nose.

When the boy was four years old, he had a troublesome papule on the eye which was treated by Sir John Tweedy, who has very kindly written me the following letter on the case.

"I first saw H. C—, on April 27th, 1907. He was then under the care of the late Dr. Radcliffe Crocker for xeroderma pigmentosum. The note I made of the eye was (L) sub-conjunctival ecchymosis at the outer side near the margin of the cornea; and at the inner side of the globe, between the cornea and caruncle, a truncated conical swelling with some ecchymosis around, and conjunctivitis. There was so much photophobia that it was impossible to get a satisfactory view, so that on the following day under a general anæsthetic I examined the eye and found an indolent swelling in the conjunctiva and episclera at the inner side of the globe. I scraped this with a sharp spoon and applied powdered salicylic acid, and ordered the eye to be washed two or three times a day with a solution of salicylate of soda. The eye gradually improved, and on May 4th, the seat of the old swelling presented the ordinary appearance of a small phlycten. I did not make any examination of the scrapings.

"I did not see the child again for eighteen months. The eye had been comfortable until the hot, bright weather set in, and there was merely a small degree of apparently simple conjunctivitis. The next visit was about eighteen months later, namely, March, 1911. The eyes were much

stronger and more comfortable, and rarely inflamed. There had been no return of the growth, and the conjunctiva was fairly healthy. At the next visit, two and a quarter years' later, although there was some intolerance to light, so that it was difficult to get a satisfactory view, there was no definite disease of the conjunctiva, and with correcting glasses he saw 2 Jaeger.

"These cases must be very rare in England. Crocker had seen only two or three of these, and this is the only case I remember to have seen.

"I may say that the growth on the eye was said in

Fig. 34.

R. L.

November 6th.

Ceylon to be malignant, and might involve the removal of the globe."

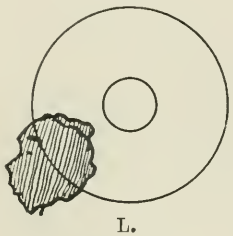
I (F. R. C.) first saw the boy on April 17th, 1914. He was æt. 11 years; his skin was tight and very pigmented. His hair was very dry; his eyelids were closely shut by the most intense photophobia without lacrymation. His nervous condition was excessively irritable, and his movements badly controlled and jerky. There was no special irritation of the nose, excepting in the light, when he sneezed continuously. I was told that he had much photophobia winter and summer, especially in the early morning, lessening as the day advances, that he scarcely ever looked at a distance, but that he could read with some difficulty; indeed, I was very surprised to find that he had been to school, and that he had a small laboratory for working at practical chemistry. I could get no look

at his eyes, but under chloroform I found a dull hyperæmia of the eyeballs, two quite small phlyctenular swellings at the right and lower margin of each cornea. The corneæ were somewhat glassy, the pupils were small but acted to light, and dilated freely to homatropin.

A few days later he saw $\frac{6}{18}$ for a moment and J. 6. He was advised massage of the eye and the use of calomel ointment. He was reported better—reading and without pain.

On November 6th he returned, the right eye acutely inflamed and red. Examination showed on it a fine

Fig. 35.



L.

January 21st.

infiltration running across the cornea obliquely like a broad superficial fascicular keratitis, but with no blood-vessels. It started from a vascular spot at the right lower edge of the cornea. The left cornea was also slightly hazy, with a marginal spot down to the right, like pterygium. Calomel ointment with massage again relieved him (Fig. 34, right and left).

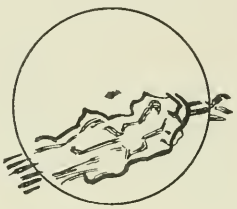
On January 19th, 1915, he again returned, complaining of the left eye, which had on it a growth, as large as a pea, protruding between the lids. Under chloroform a large white papilloma was seen growing on the inner lower edge of the corneal margin, from the corneal surface, and from the adjacent episclera; it was lightly ligatured and dissected away from a rather wide base, which was scraped and cauterised. The right cornea had cleared very much

since November 6th. A swollen infiltration on its margin was scraped and cauterised (Fig. 35, left).

While he was under chloroform it was easy to see the distribution of the pigment spots and the dryness of the skin; it covered the head, face, and neck down to the line of the mammæ, and the same level on the back well over the scapulæ, the whole length of the arms and hands, but the flexor surface of the forearms and the inner surface of the arms was less marked. The palms of the hand and plantar surface of the feet were free of pigment. The legs were pigmented up to half of the thigh.

The upper part of the thighs, buttocks, abdomen, and

Fig. 36.



R.

scrotum were covered with pale, healthy skin and free of pigment except for an occasional single definite dark spot of pigment here and there. The skin of the head and face and hair were very dry.

I saw the girl once, on January 19th, 1915. Her left eye had been red and weak for some months, and had lately got worse. The right eye had but little the matter with it and saw $\frac{6}{9}$. I found on the right cornea a wide, non-vascular infiltration running across it from side to side, supplied at each end by a leash of vessels from the conjunctiva, but which did not pass inwards beyond the corneal margin. There was a ragged erosion of the epithelium over the infiltration (Fig. 36).

Additional Note.

The condition of the eye and sight a few days after the removal of the growth were much improved. The boy

was soon able to read small print easily, though he complained of much photophobia in the morning. He dresses and breakfasts almost in the dark. On April 10th, 1915, I received a letter saying that the dread of light is much relieved and gives but little trouble, even in the early morning. He is working at his chemistry two or three hours a day, and it has been arranged that he shall go next term to a private school with his sister. The site of the growth is quite flattened and quiet.

The girl is also much more comfortable, the corneal haze has cleared up a good deal, though it is not well, and the lower lids are somewhat ulcerated.

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Journal of Cutaneous and Genito-Urinary Diseases, 1892, vol. x, p. 129; Brayton's case.

Dr. George Mackay said that when he saw the title of the paper he was not quite sure what the disease was, though he had many years ago attended Prof. Kaposi's clinique in Vienna. But when the President showed his beautiful plates he recognised that he had seen a case in Scotland in recent years, which had been referred to him by Dr. Norman Walker from his Skin Clinique. He saw the case on two occasions. There was some conjunctival irritation, and his recollection was that there was something like a commencing phlyctenule on one or both eyes. There was no definite growth on the conjunctiva such as had just been described, nor anything calling for operative interference then. Beyond that he did not remember particulars. Dr. Walker was treating the skin with some form of radiant energy. He asked whether the President or Mr. Greeves had anything to say as to the bacteriology of the condition.

Mr. Russ Wood said he had seen a case of Kaposi's disease with ocular symptoms about a year ago. The patient was æt. 35 years, and had suffered from the condition since girlhood. There was an involvement of the lower eyelid on the left side producing ectropion, and some marginal ulceration of the cornea of a phlyctenular type. There were no growths as described by the President in his cases. A very marked feature in this patient was the injurious effect of sunlight. In the summer the symptoms were always aggravated and in winter ameliorated. The family history was good.

Mr. F. Richardson Cross said, in reply, that the case mentioned by Mr. Russ Wood might be the one of which he showed pictures. He knew of no researches connected with the bacteriology of the condition. The case of Dr. Norman Walker was published in that gentleman's book on skin diseases. The name trophodœma pigmentosum was also used for the condition.

PLATE II.

Illustrates Mr. R. Affleck Greeves's paper on The Microscopical Anatomy of a Conjunctival Tumour from a Case of Kaposi's Disease (p. 213).

Fig. 1.—Low-power view of a section across the broadest part of the tumour.

Fig. 2.—High-power view of that part of the growth which shows invasion of the supporting tissue by epithelial cells.

Fig. 1.

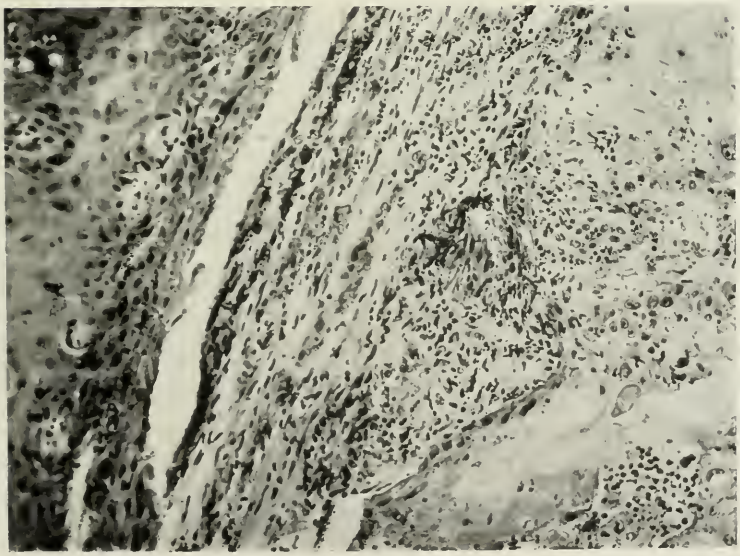

Fig. 2.

Adlard & Son, Impr.

but the blood supply to the growth is very poor. For this reason probably there is much evidence of degenerative change in the growth, and some totally necrotic areas are present, surrounding which are to be found some very large bloated cells with enormous nuclei, of which more than one are sometimes present. In some of the cells a hyaline form of degeneration is taking place.

With regard to the question of the malignancy of the growth, I think there is definite evidence in favour of its being malignant in one particular part, where isolated strands of epithelial cells can be seen invading the supporting tissue (Pl. II, fig. 2).

Karyokinesis, often of an atypical type, is taking place actively in several places.

Epithelial growths such as that here described rarely develop on mucous surfaces in this disease; they are usually confined to the skin of the more exposed parts of the body. Stelwagon, speaking of the way in which mucous membranes are affected, says: "In rare cases insignificant pigmented or telangiectatic lesions are seen on the palpebral conjunctiva, lips, and buccal cavity; sometimes on the mucocutaneous junctions." He does not mention that epithelial growths have ever been found in these situations, nor does he speak of the bulbar conjunctiva as being affected at all. Crocker mentions the presence of "pterygia" in two of his cases. The occurrence of multiple papillomata on the skin which tend to become malignant is, of course, one of the features of the late stage of the disease. According to Pollitzer, some of the malignant growths have shown parts resembling a spindle-celled sarcoma, but in all of them the epitheliomatous tissue forms the greater part of the tumour.

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3. *An epibulbar epithelioma which completely disappeared after one application of radium bromide.*

By E. Treacher Collins.

(With Plates III, IV, and V, fig. 5.)

Harry V—, æt. 78 years, in October, 1908, was seen by Mr. Priestley Smith, who has kindly allowed me to make use of his notes. The patient complained of a small patch which he had noticed in his right eye a few days previously, after a cold motor ride. At the outer margin of the cornea there was found a small superficial infiltration, or necrotic patch. There was but little injection, and he did not complain of any pain. The patch was slightly scraped and gut. dionin 2 per cent. ordered.

He had some striæ in each lens, more in the right than the left, and his vision was :

R. $\bar{c} + 1{\cdot}25 = \frac{5}{5}$. L. $\bar{c} + 1 = \frac{5}{6}$.

His next visit to Mr. Priestley Smith was on July 28th, 1914. The patch on the cornea was no larger, but there was now a subconjunctival swelling adjacent to it, suggestive of either a pterygium or episcleritis.

His vision had slightly deteriorated, being :

R. $\bar{c} + 1{\cdot}25 = \frac{5}{6}$. L. $\bar{c} + 1{\cdot}5 = \frac{5}{8}$.

The conjunctival lump was touched with the point of the actual cautery in three places.

On September 2nd, 1914, the lump was recorded as being flatter, but wider. Its margins were injected. The cautery-point was applied to several of the marginal vessels.

Three days later, under cocaine, a small piece of the lump, where untouched by the cautery, was removed with a keratome and sent to Dr. Laurence Ball for examination. At this date the diseased area in the conjunctiva measured 10 mm. vertically and 7 mm. horizontally.

Dr. Ball reported : "Fragment too small for good investigation, shows only a few epithelial cells ; am

inclined to say not malignant, but cannot give definite opinion."

On October 13th, the growth was found to be rather larger horizontally, and the eye was beginning to ache a little at times.

On October 27th, at Mr. Priestley Smith's request, the patient came to see me. I found him a very hale and active man for 80. On the surface of his right eyeball was a raised, nodulated mass which appeared firmly united to the underlying sclerotic. It measured 10 mm. in diameter horizontally and vertically, and had several large and tortuous blood-vessels running up to it. In the part where it extended into the cornea there was a small patch of a dense white colour.

I grasped a portion of the growth with fine-toothed forceps and cut it off with scissors. The piece so removed was only a small nodule, but microscopical examination of sections of it showed definitely that the tumour was an epithelial growth extending into and infiltrating the episcleral areolar tissue (Pl. III, fig. 1).

On November 12th, the patient was taken for treatment by Mr. Hayward Pinch at the Radium Institute. Ten mgrm. of radium bromide (ra. $br._2$) on a circular disc 1 cm. in diameter, unscreened, was held continuously, by a relay of assistants, over the growth for fifty minutes, the eyelids being held apart by a speculum.

I did not see the patient again for a fortnight, he then told me that the eye had been bloodshot, but that no very violent reaction had followed the application of the radium. On examination I found that the nodulated growth had disappeared except for small portions at the inner and outer margins. On the inner side at the limbus there was a thickened edge, and on the cornea itself there was still a white patch.

He was shortly afterwards seen by Mr. Priestley Smith, who can confirm the remarkable way in which the growth had disappeared.

I advised another application of the radium at the

PLATE III.

Illustrates Mr. E. Treacher Collins's paper on An Epibulbar Epithelioma which completely disappeared after One Application of Radium Bromide (p. 215).

(The figures illustrating this paper are from photo-micrographs taken by Mr. E. Collier Green. Fig. 2 by Mr. G. Coats.)

Fig. 1.—Section of growth which was cured with radium.

Fig. 2.—Epithelioma of conjunctiva which was removed by operation and did not recur.

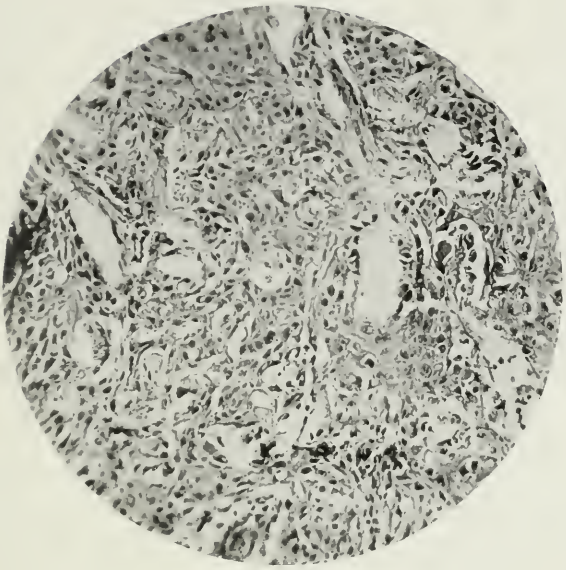

Fig. 1.

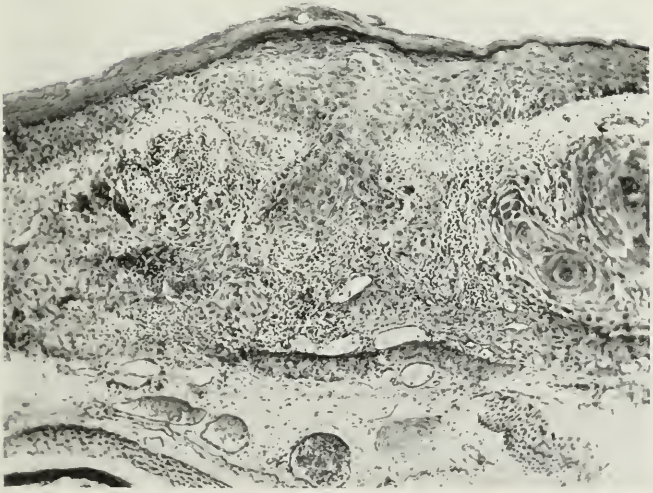

Fig. 2.

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margins, but circumstances arose which prevented this being carried out. I did not see the patient again until January 14th this year. The growth had then quite gone, leaving at the site it formerly occupied a depressed, flat, greyish area. The white patch on the cornea and all thickening at the inner margin had disappeared. The outer margin was a little higher than the former site of the tumour, but this appeared to be due to depression of the latter from loss of tissue, rather than elevation of the former by new growth.

I last saw him on April 13th, that is, five months after the application of the radium ; there was then absolutely no sign of any growth and the enlarged vessels had disappeared, so that the eye was no more injected than its fellow.

Radium is credited with a remarkable selective power on the cells of cancerous growths. It is said to lessen their vitality, causing them to necrose, and at the same time act as a stimulant to the surrounding healthy cells, increasing their vitality. In the case above recorded, it has most certainly caused the neoplasm to disappear, without in any way damaging the surrounding parts.

There are certain peculiarities in the way in which epibulbar epitheliomatous tumours grow, which led me to anticipate that they would in certain stages prove very amenable to treatment with radium. When I suggested its employment in the above case I was not aware that it had been previously employed for growths so situated. I have since found that it had been used with success in Prof. Fuchs' clinic, in what was diagnosed clinically as an epithelioma growing from the limbus ; no microscopical examination of the tumour was made. The case was described by Dr. A. F. Mattice, of New York, at the Ophthalmological Section of the 1913 International Congress of Medicine, in the discussion on a paper by Dr. W. Koster* entitled "Direct Treatment of Eye Diseases with Radium and Mesothorium." The mica-box method was employed

* *Trans.*, p. 254.

for the application of the radium. Three applications were made, in the course of six weeks, of six, twelve and fifteen minutes respectively. A reaction followed each application with the formation of a pseudo-membrane. Five months after the commencement of the treatment, when the patient was last seen, it is stated that there were only a few maculæ in the cornea left.

A case of epithelioma of the conjunctiva was shown at the Society in 1903, by Mr. A. Ogilvy,* in which X-rays had been applied locally to the tumour without producing any effect whatever.

In considering how far it is likely that the use of radium may replace operative procedures in the treatment of epibulbar epithelioma, it is necessary to study the mode of extension of this form of neoplasm. Particularly interesting is this study now, in connection with Mr. Handley's investigations as to the way cancerous growths, starting elsewhere, especially those originating in the breast, become dispersed throughout the body.

Handley has shown that the spread of cancer from the breast and the production of what are termed metastases, is mainly due to "centrifugal lymphatic permeation," and to a very small extent, if at all, to entrance of the emboli composed of cancer cells into the blood-stream.

There is considerable evidence to show that cancerous epithelium which reaches the blood-stream is either destroyed or rendered incapable of growth. The cancer cells excite thrombosis, and the thrombus as it organises contracts and destroys them.

It will be interesting, then, to consider how the process of centrifugal lymphatic permeation takes place in the structures in and around the eyeball.

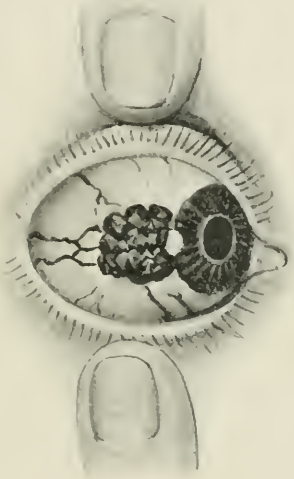
Epibulbar epithelioma, unless it originates at the seat of a scar, almost always starts at the limbus. Fuchs has pointed out that one factor which probably predisposes this part of the front of the eye to become the seat of a cancerous growth is the anatomical arrangement of its

* *Trans. Ophth. Soc. of the U. K.*, vol. xxiii, p. 5, and vol. xxiv, p. 214.

epithelium "The limbus is the only place in the ocular conjunctiva, in which small papillæ are normally present. Between these papillæ we sometimes find, in the healthy eye, a proliferation of epithelium, the latter growing in the form of conical processes in the depth of the tissue."

A growth starting in the ocular conjunctiva very soon attracts attention, and epithelioma is here likely to come

Fig. 37.



Shows the appearances of the epibulbar epithelioma in H. V—'s eye previous to the application of radium bromide.

under earlier observation than anywhere else in the body.

At first the neoplasm is confined to the conjunctiva and episcleral tissue, and is movable with the conjunctiva where it overlies the sclerotic. Its complete removal by operation from the surface of the eye at such a stage, so that no recurrence ensued, has several times been effected.

In 1905, a man, æt. 51 years, came to me at Moorfields Hospital, who three months previously had pricked his right eye with a sharp blade of grass, so that it bled a

little ; two and a half months later, a fortnight before I saw him, he had noticed a small swelling at the seat of the injury. I found a small tumour on the ocular conjunctiva, opposite the palpebral fissure, to the outer side of the cornea, and about 5 mm. from its margin ; I took it to be an "epithelial plaque," *i. e.*, an innocent form of epithelial thickening, but thought it was best to remove it. Microscopical sections of the growth were cut by Mr. Coats, who was Curator of the Museum at that time, and they showed that it was undoubtedly an epithelioma. There were extensions down of the epithelium into the episcleral tissue, and a formation of the characteristic nest-like arrangement of cells in these extensions (Pl. III, fig. 2). So far as could be made out the whole of the new growth had been removed.

I did not see the man again for ten years, during which time the eye had caused him no trouble. He came to me in January last because he had noticed a fresh little spot in his right eye, in much the same situation as the one which I had previously removed. Thinking it might be a recurrence I cut it out. Sections of it made by Mr. Greeves show, however, simply a thickening of the surface epithelium and no invasion of the deeper tissues ; nothing to suggest that it was of a malignant nature.

An epithelioma which starts to grow at the limbus, and extends into the episcleral tissue, comes into contact with the hard unyielding sclerotic, a structure with very few lymphatics in it. The result is that the growth tends to spread at first very slowly, and forms a considerable mass on the surface of the globe before it invades the deeper parts. It will often ulcerate, and fungate out, so as to overlap a larger area of the surface of the globe than that to which it is attached.

In a specimen of an eye with an epibulbar epithelioma which I examined for Mr. Simeon Snell,* and which he brought before this Society in 1900, the tumour was

* *Trans. Ophth. Soc. of the U. K.*, vol. xxi, p. 24.

PLATE IV.

Illustrates Mr. E. Treacher Collins's paper on An Epibulbar Epithelioma which completely disappeared after One Application of Radium Bromide (p. 215).

Fig. 3.—Epithelioma extending down to sclerotic at limbus and not infiltrating it.

Fig. 4.—Epithelioma at limbus extending into canal of Schlemm.

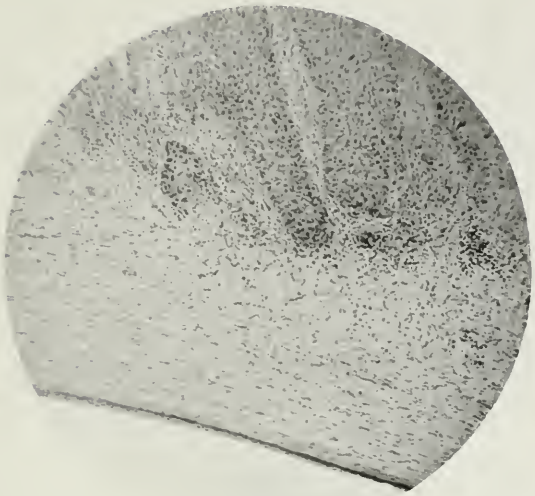


Fig. 3.

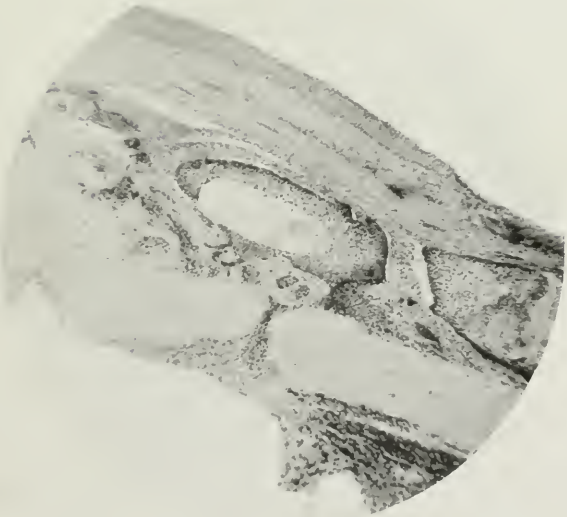


Fig. 4.

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noted as being of four years' formation. Its base, where it was attached to the globe, measured 13 mm. across, from there it fungated out and measured 17 mm. in its widest part; it was raised 8 mm. above the surface of the globe. Notwithstanding its long duration and size, it had only penetrated down through half the thickness of the sclerotic and had not permeated any of its lymphatic channels into the interior of the eyeball (Pl. IV, fig. 3). In the light of the case which forms the subject of this paper, it seems probable that this case of Mr. Snell's would have been one in which radium might have been very successfully employed, and the necessity of removing the eye have been avoided.

An epithelioma starting at the limbus, if it extends inwards, meets with Bowman's membrane on the surface of the cornea, which offers considerable resistance to its progress. The new growth is often seen to extend some distance anterior to this membrane without invading the substantia propria of the cornea. When the substantia propria is reached columns of the cancer cells extend along the lymphatic spaces between its layers.

The greater resistance offered by the sclerotic, than the cornea, to the extension of epitheliomatous growth is commented on in a recent article by Casolino,* and is explained by the presence of more numerous lymphatic spaces in the latter than the former.

A growth which has penetrated to the substantia propria of the cornea at the limbus soon reaches the lymphatics connected with the canal of Schlemm, and will extend down along them to the canal itself.

This is well shown in a specimen of an eye with epibulbar epithelioma which was kindly sent to me some years ago, for pathological examination, by Dr. Maddox. In it the growth has extended into the superficial layers of the cornea, and at the limbus are seen prolongations downwards of narrow columns of epithelial cells to the canal of Schlemm, which itself is filled with similar cells

* *Archivio di Ottalmologia*, x, 1914.

(Pl. IV, fig. 4). In this specimen the growth has ended at the canal and not extended into the eyeball.

Cases have been recorded of a similar character by Parisotti,* Remak,† Caspar,‡ Lagrange,§ and Delord and Revel,‖ where from the canal of Schlemm the neoplasm had permeated the spaces of Fontana and reached the interior of the eyeball. It had then passed into the anterior chamber at the root of the iris, and also formed a considerable mass in the lymph spaces between the uveal tract and sclerotic.

An epibulbar epithelioma starting at the limbus and extending outwards will in time reach the large lymph space of Tenon's capsule. In it the neoplasm spreads readily and grows round the external surface of the sclerotic. In the specimen sent to me by Dr. Maddox such invasion of Tenon's capsule had taken place. There was a considerable thickness of cancerous growth on one side of the surface of the eyeball, almost as far backwards as the position where the long ciliary arteries perforate the globe.

From this mass in Tenon's capsule columns of cells extended off along lymphatic channels in connection with the blood-vessels, in the superficial layers of the sclerotic (Pl. V, fig. 5). Columns of epithelial cells were also seen, cut in different directions, which were permeating lymphatics leading away from the external surface of Tenon's capsule into the orbit.

A photograph of a specimen, showing the spread of an epithelioma of the limbus to Tenon's capsule, the fornix, and the eyelid, is reproduced in Parsons' *Pathology of the Eye* (p. 143) from a specimen sent by Prof. Fuchs.

By extension of the growth in the anterior part of Tenon's capsule the whole cornea may become encircled by

* *Rec. d'Ophtalm.*, 1885, p. 272.
† *Arch. f. Augenheilk.*, 1886, Bd. xvi, p. 276.
‡ *Ibid.*, 1892, Bd. xxiv, p. 177.
§ *Traité Tumeurs de l'œil*, t. i, p. 135.
‖ *Ann. d'oculist*, 1909, t. cxlii, p. 432.

PLATE V.

Fig. 5 illustrates Mr. E. Treacher Collins's paper on An Epibulbar Epithelioma which completely disappeared after One Application of Radium Bromide (p. 215).

Fig. 5.—Epithelium on surface of sclerotic in Tenon's capsule and extending into lymphatic sheaths of the vessels in sclera.

Fig. 6 illustrates Mr. E. Treacher Collins's paper on Apparent Accommodation with Aphakia (p. 314).

Fig. 6.—The two upper drawings show the appearances of the boy's eyes, whose case is described in the paper, before dilatation of the pupils with atropine; and the lower drawings the appearances after the use of atropine. The black areas are the openings in the capsules; after dilatation of the pupil in the left eye a dark area is shown at the periphery of the capsule, which is not present in the right eye.

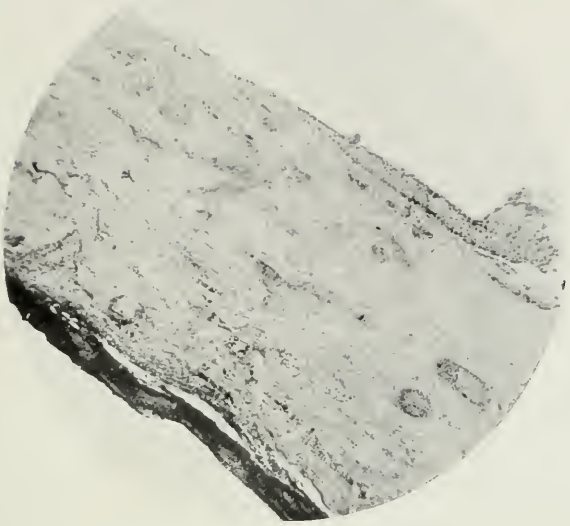

Fig. 5.

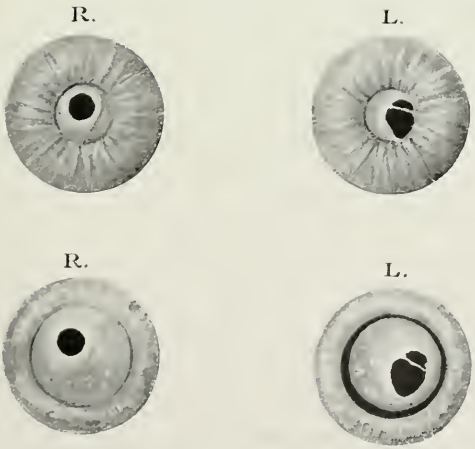

Fig. 6.

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it, *pericorneal epithelioma*. Cases where this occurred are recorded by Meyer* and Remak.† In still more extensive involvement of Tenon's capsule the whole globe becomes almost surrounded by growth, *peribulbar epithelioma*, of which descriptions have been published by Heyder,‡ Lagrange,§ and Reiss.‖

An extension to the eyelid secondary to involvement of Tenon's capsule is shown in the illustration above referred to in Parsons' *Pathology* and has also been recorded by Lagrange.¶

When an epibulbar epithelioma has extended into the interior of the eye or into the deeper parts of Tenon's capsule, it would seem very improbable that the selective action of radium would penetrate sufficiently to cause destruction of the tumour cells in all their ramifications in lymphatic channels. In such cases the operations of enucleation or exenteration will still probably have to be resorted to. The cases, however, in which this involvement of the deeper parts has been met with, are usually those in which an attempt at removal of the growth has previously been made, and where a recurrence had taken place. If, when first seen, such cases can be treated successfully with radium, the complications necessitating more radical procedures will not arise.

Involvement of the lymphatic glands in cases of epibulbar epithelioma is, as stated by Lagrange, apparently of rare occurrence. Cases in which the pre-auricular or submaxillary glands were found enlarged have been recorded by several observers. Enlargement of these lymphatic glands does not necessarily imply that they have become the seat of cancerous growth. In the case already referred to as described to this Society in 1900 by Mr. Simeon Snell, the pre-auricular gland on the affected

* *Rev. Gen. d'Ophtalmol.*, 1882, t. i, p. 34.
† *Loc. cit.*
‡ *Arch. f. Augenheilk.*, xvii, 1887, p. 294.
§ *Loc. cit.*, p. 165.
‖ *Klin. Mon. f. Augenheilk.*, xli, 1903, p. 401.
¶ *Loc. cit.*, p. 152.

side was enlarged to the size of a marble, movable, and not adherent to the skin. A few glands could also be felt under the jaw on both sides. Six days after the removal of the eye, the pre-auricular gland was excised and found suppurating and encapsuled. It was not thought to be malignant and so the removal of the glands in the neck was not performed.

Where an epithelioma is ulcerating and fungating, as in this case, collections of inflammatory cells are usually found around the true cells of the tumour, due no doubt to infection by pyogenic organisms of the surrounding tissue. A septic inflammatory condition thus started might readily give rise to an inflammatory infection and enlargement of lymphatic glands.

There is, so far, no anatomical evidence to show whether, when these glands do become the seat of carcinoma, there is a direct extension to them of the growth along lymphatic channels, or that the cancer cells are carried to them in the lymph stream.

Of cases in which still wider dissemination of a conjunctival epithelioma than to the facial glands has taken place there are exceedingly few records. I have only been able to discover the description of three possible cases which ended fatally.

Bousquet* recorded a case in which when first seen the submaxillary glands were affected. The growth was removed from the surface of the eye, but not the eyeball itself; the submaxillary tumour was also enucleated. After the operation generalisation of the growth was rapid and the patient soon died. The tumour is described as being an "encephaloid carcinoma."

Lagrange, in remarking on this case, says he thinks that if a more competent pathological examination had been made it would have proved to be an encephaloid sarcoma.

Besevi† mentions a tumour composed of pavement

* *Soc. Anatom. de Paris*, 1876.

† *Annali di Ottalm.*, 1888.

epithelium which he removed from the bulbar conjunctiva and which rapidly recurred, necessitating the removal of the eye. The patient died some time afterwards of carcinoma of the liver.

In commenting on a case shown before this Society in 1891 by Messrs. Anderson Critchett and Henry Juler, Mr. Henry Power,* who was then President, said he remembered a case which had commenced in the form of a small granulation at the sclero-corneal junction. It was on two occasions snipped off or cut away under the impression that it was benign, but its recurrence showed that it was malignant; and the eye in the first instance and the whole contents of the orbit subsequently were removed, with the result that the patient lived for a considerable period free from further trouble in the orbit, and ultimately died from the development of cancer in other organs.

In this case the evidence that the primary growth was an epithelioma and not a sarcoma is insufficient.

Mr. W. H. H. Jessop said that great interest attached to the successful use of radium in this case, because one was still very doubtful about the use of that substance on mucous membranes. He had two cases of epithelioma at the limbus, years ago, before radium was known, and one of them was of considerable interest. He was a gentleman, æt. 65 years, who died last year æt. 87 years. He had exhibited the patient. He had an epithelioma like that in Mr. Collins' case. It was removed and the base cauterised, and it never recurred. The chief point of interest was that he had an epithelioma also on his lower lip soon afterwards. It was removed by Mr. Willett by a simple procedure, not the more radical operation of the present day, and here also there was no recurrence. It was peculiar that the epithelioma should have selected two mucous surfaces like the lip and conjunctiva.

* *Trans. Ophth. Soc. of the U. K.*, vol xi, p. 47.

4. *A conjunctival pigmented mole of rapid growth.*

By R. Foster Moore.

F. R—, male, æt. 35 years. The patient states that the "clot of blood" has been present in the eye for two years, and that it is now rapidly increasing in size. He is not aware that there was any spot in the eye previous to two years ago. The greater part of the growth is pigmented, but there was a more fleshy unpigmented part (which has been removed for microscopic examination), which had slightly overgrown the surface of the cornea.

(*Card Specimen.*)

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5. *Pigmented hairy mole involving eyelids and conjunctiva.*

By G. H. Pooley.

C. B—, æt. 17 years. V.: R. $\frac{6}{60}$, highly myopic and astigmatic? L. $\frac{6}{9}$, slight myopic astigmatism.

History.—Has had large birth marks on his face all his life—some were treated years ago. The moles on his face have not changed much in size; the right eye has become worse and the growths larger since Christmas—they are growing rapidly—the moles on the margins of the lids have progressed.

Present condition.—There are extensive, hairy, pigmented moles on both sides of his face and neck, and a pigmented patch is present on the roof of his mouth. These involve the margins of the upper eyelids—not quite half of the right and fully two thirds of the left. The ocular conjunctiva of the right eye is swollen and bulges over the cornea; the upper margin of the cornea is affected from the outer

side in its whole extent to the mid-point on the inner side; the lower margin of the cornea from the outer side to the

Fig. 38.

mid-point below; the lower and inner quadrant being free. There is pigment at the advancing margin of the conjunctival growth.

Is this mole becoming malignant?

A small nodule is present also on the left eyeball.

Fig. 39.

6. *Congenital ectropion of the lower lids.*

By E. Treacher Collins.

Lilly F—, was brought to me first at Moorfields Hospital on July 18th, 1910, when she was four months old, on account of a defect in her eyelids which dated from birth.

On examination she was found to have partial ptosis on each side, well-marked epicanthus, and ectropion of the lower eyelids. It is this latter defect which forms the exceptional feature in the case.

The lower lids appeared to be too large to fit closely up to the eyeballs. When the eyes were shut, the lower lids at the outer part came outside the upper lids. Nowhere did the margins of the lower lids come into contact with the globes. The widest separation was at the outer part, the notch at the outer canthus was more pronounced than usual. The lower lacrymal puncta in each lid were present, but they stood away from the eyeballs. The conjunctiva on the inner surface of the lower lids was injected, and there was some mucous discharge from it.

The patient wrinkled the skin of her forehead and arched her eyebrows in her efforts to raise her upper lids. There was no paralysis of her facial muscles, and she was able to use her orbicularis muscles to close her eyes and to screw up her lids.

The eyes themselves appeared in every way quite healthy, and showed no congenital defects. Her body was in other ways well-developed. There was no history of any congenital defect in the eyes or eyelids of any member of the family.

Nothing was done for the condition beyond the use of sulphate of zinc drops until January this year, when the child was 4 years and 10 months old. Since she had been first seen the bridge of her nose had developed to some extent, and the epicanthus had become less marked, in other respects the condition of her eyelids remained just the same.

On January 14th, 1915, the child being under the influence of a general anæsthetic, a wedge-shaped piece of the whole thickness of the right lower lid was cut out a little way internal to the outer canthus. The base of the wedge was at the free margin of the lid, and measured about 6 mm. in length. A stitch was inserted through the two raw surfaces left, just inside the line of the lashes,

and by means of it they were drawn together and united. In this way the length of the lower lid was shortened and it was drawn up into a better position. No operation has yet been performed on the left side.

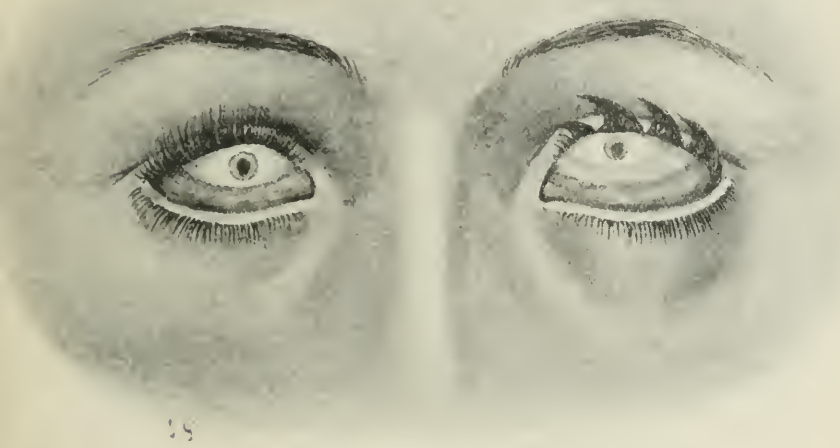
Fig. 40.

Photo by J. Bender & Co.

Microscopical examination of sections of the piece of the lower lid which was removed show the orbicularis muscle to be well-developed The group of its fibres near the free border of the lid, which are termed the muscle of Riolani, are exceptionally numerous, showing

that the defect cannot be attributed to any defect in their development. As the function of Riolani's muscle is to keep the border of the lids in contact with the eyeball, it had occurred to me that its absence might account for the ectropion, but this was evidently not the case. The epithelium of the palpebral conjunctiva was several

Fig. 41.



Dr. Geo. Mackay's case of congenital microphthalmos and ectropion with cyst in left lower lid. Symmetrical coloboma iridum.

layers thick, and there was some formation of papillæ on it, evidently the outcome of exposure.

Dr. George Mackay said he had seen one case corresponding to the first of Mr. Collins', but with the distinction that while both lower lids were congenitally everted, there was microphthalmos in both eyes and symmetrical coloboma iridum. There was also a cyst, deep-seated below the left lower lid. He had an illustration of the case (Fig. 41), and had shown it in the museum of the International Medical Congress, London (1913).

V. DISEASES OF THE LACRYMAL APPARATUS.

1. *The intra-nasal treatment of dacryocystitis.*

By W. Douglas Harmer.

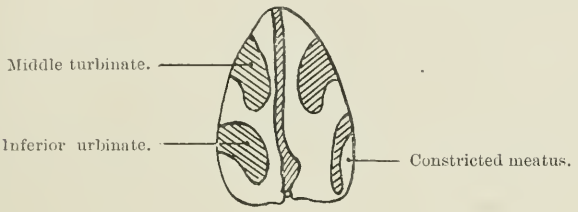
Mr. President, I must thank you for your invitation to read a paper on the intra-nasal treatment of dacryocystitis. The subject is an interesting one, but has not received till recently, in this country, as much attention as it deserves. The rhinologist is often asked to examine the nasal passages and report whether there is any cause for the inflammation of the tear-ducts, and in many instances nothing can be discovered, nor is there any definite history of rhinitis; on the other hand there may be marked deflection of the septum, enlargement of the turbinates, polypi, ethmoiditis, tumour, or an old-standing rhinitis, conditions which by themselves rarely lead to epiphora. It is important to emphasise the fact that although we examine and treat enormous numbers of these cases, we seldom find it necessary to call in the ophthalmologist because inflammation has spread to the lacrymal apparatus. Doubtless the majority of cases of dacryocystitis are secondary to nasal disease, but there must be some other factor which is not yet understood. The only abnormality which seems to occur in all the cases which I have had the opportunity of examining is a curious flattening of the inferior turbinate on the affected side, which almost obliterates the inferior meatus in its anterior part, and may cause a local rhinitis implicating the opening of the duct.

The above remarks are sufficient to bring out the first point of this paper, namely, the question whether the

treatment of such abnormalities is likely to cure the dacryocystitis, and the answer is "in the negative." As a general rule the result is very unsatisfactory.

I now pass on to the operations that have been devised for draining the lacrymal channels into the nose. According to Paterson the idea "is a very old one, having been known to the ancient Greeks and Egyptians, yet practically nothing had ever been done to carry it out." In 1904, however, attention was drawn to the subject by Toti, an Italian rhinologist, who devised an operation whereby the lacrymal sac was drained into the nasal cavity through an external curved incision near to the inner canthus.

FIG. 42.



The results obtained by this method were so promising that fresh efforts were made by surgeons on the Continent and in America to perfect the operation by an intra-nasal route, but it was only in 1912, after the results of West (Berlin) and Polyák (Budapest) had been published, that it became generally employed in the different countries. Personally, I began to perform these operations in 1913 after reading a paper by Yankauer. Shortly, Yankauer's method consists in temporary elevation of a flap from the outer nasal wall, slitting up the entire nasal duct after removal of its bony nasal canal, opening freely the lacrymal sac, and finally replacing the original flap of mucous membrane, the result being an attempt to re-establish drainage along the original channels by capillary action and syphonage. Of four cases treated thus two received no benefit at all, one was completely relieved, and in the fourth the epiphora ceased, but the patient dis-

appeared after three weeks and had not been seen again. These results were not so good as Yankauer's, who completely cured the suppuration in all, and the epiphora in eight out of nine cases. It is probable that my technique was not so good as his, and in any case the impression which I formed of the operation was that it was difficult to carry out and unnecessarily complicated, so I turned to the methods of West and Polyák, namely, of excision of the lacrymal sac above the nasal duct.

In West's operation a small quadrilateral flap of muco-periosteum is turned down in front of the anterior end of the middle turbinate bone, *i. e.* anterior to the position of the sac, the muco-periosteum and bone lying internal to the lacrymal sac are then removed by a chisel or forceps, the sac is pushed through the window by external pressure over the inner canthus and is seized with fine tooth forceps and excised. A lacrymal probe is then passed through the canaliculus, or a fine syringe inserted through the punctum, so that the contents of the sac may be thoroughly removed.

Polyák's operation is very similar, with the exception that no preliminary flap is made, the nasal duct is divided transversely immediately below its junction with the sac, the sac is entered from below and excised, the canaliculus is never slit, and no style is passed through into the nose.

During the last three years I have performed ten operations on these lines, most of them for cases with profuse muco-purulent discharge which had failed to yield to the ordinary treatments of my colleagues, including excision of the sac in a few instances, and I have added a short *resumé* of each case at the end of this paper for those who are interested to read it.

Although my experience is at present limited I should venture to say that this operation is comparatively easy for those who are accustomed to intra-nasal work and, if necessary, can be carried out under cocaine anæsthesia alone ; the exposure of the sac has presented no difficulties, but in the actual excision there has sometimes been bleeding and oozing to a troublesome degree which

has prolonged the operation from fifteen to thirty or forty minutes and made me doubtful whether enough of the sac had been excised; in three cases a submucous resection of the septum was necessary as a preliminary operation, to afford more room, but this was usually performed at the same sitting.

In one case (not included with the ten) a resection of the septum was carried out and a West's operation was then attempted, but on breaking into the bone a large ethmoidal cell containing thick pus was opened and under the circumstances it seemed advisable to postpone the removal of the sac.

In the majority of cases no packing has been inserted in the nose afterwards; there has been no return of the hæmorrhage, very little shock, and the patient has been out of bed in two or three days; occasionally slight bruising near the inner canthus is seen for a few days, but no swelling or inflammation. For a week or longer there is a definite crusting at the site of the wound, which is of no importance; no after-treatment has been carried out excepting occasional cleansing of the nose. In the favourable cases the suppuration and epiphora completely ceased and the external fistula closed in forty-eight hours after treatment; thus, Case 14 was a boy, æt. 4 years, sent to me by Mr. Jessop with a history of persistent suppuration in both sacs since he was a few days' old. The condition had not improved although he had been in the ward for four months and had received careful treatment, including slitting of the canaliculi and the wearing of styles. After a West operation on both sides the discharge and epiphora completely ceased in twenty-four hours, much to the delight of the parents. The same result has been observed in cases with old-standing fistulæ, the skin healing very soon after the operation.

Of the ten cases, six have been cured, one improved so that he now has epiphora only in a wind, one has not been traced, and two were not benefited. The cures have been maintained for periods of from three weeks to one year;

up to the present there has been no relapse in a patient who had once been relieved of the symptoms. It would seem unlikely that the fistula would close when there is a definite stenosis of the duct; on the other hand, with epiphora caused by catarrhal inflammation of the nasal duct without stenosis the fistula would be liable to heal as soon as the inflammation disappeared. This perhaps explains why dacryocystitis with suppuration is easier to cure than a simple epiphora.

It would appear that intra-nasal dacryocystostomy is a good operation. West has reported two hundred cases, Polyák forty-two, and many other surgeons smaller numbers with 90 per cent. cures. Yankauer was opposed to these methods because of the fear that the contents of the nose might be blown into the eye which, as he says, "is not only a dangerous substitute for the previous epiphora, but is dangerous to the conjunctiva."

So far I have seen nothing which confirms this fear. On the contrary in one of West's cases Silex was able to perform a cataract operation successfully. Mr. Jessop and Mr. Spicer, who have sent me most of the patients, have seen them afterwards and can determine better that I can the relative value of the treatment. Personally, I regard the results as promising, especially as these are a first series and there has not been time to grasp the difficulties of the operation or the after-treatment appropriate to each case. In the future when a cure is not obtained I shall advise a second excision of the sac which will be easy to perform under cocaine.

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No.	Sex.	Age.	Sent by	Disease.	Duration of symptoms.	Operation.	Date.	Result.
1	♀	28	Mr. Spicer	Dacryocystitis	7 months	Submucous resection of septum and Yankauer	Jan., 1913	No improvement. Seen March, 1915.
2	♂	21	„ Wm. Lang	„	10 years	Ditto	June „	Improved. Epiphora ceased during next three weeks, but not seen since 1912.
3	♀	40	„ Spicer	Epiphora	6 months	Yankauer	July „	Improved. She has slight epiphora in cold wind, but can do needlework now. Seen March, 1915.
4	♂	8	„ „	Dacryocystitis	3 years	Submucous resection of septum and Yankauer	Oct. „	No improvement. Seen March, 1915.
5	♂	22	„ Jessop	Epiphora following removal of jaw for sarcoma	6 „	West	April and May, 1913	Cured. Not seen after three weeks.
6	♀	32	„ „	Dacryocystitis	2 „	„	April, 1914	Cured. Seen March, 1915.
7	♀	34	„ Spicer	„	2 „	„	May „	Not traced.
8	♀	43	„ Jessop	„	7 „	„	July „	No improvement. Seen March, 1915.
9	♂	54	„ Spicer	„	5 „	„	Aug. „	Improved. Still slight epiphora which does not interfere with his work. Seen March, 1915.
10	♀	37	„ Jessop	„	6 „	Submucous resection of septum and West	Jan., 1915	Cured. Seen March, 1915.
11	♂	26	„ Spicer	„	6 months	West	Feb. „	Cured. Seen for 3 weeks.
12	♀	22	„ Jessop	„	4 years	„	„ „	No improvement.
13	♂	4	„ „	Dacryocystitis, bilateral	Since birth	„	Mar. „	Cured.
14	♂	13	Fr. Moorfields	Dacryocystitis	6 weeks	„	„ „	Cured.

Operation.	Cases.	Cured.	Improved.	Not improved.	Not traced.
Yankauer	4	0	2	2	0
West	10	6	1	2	1

Mr. F. Richardson Cross said the meeting would feel much obliged to Mr. Harmer for his paper. It was very important that all should recognise that these cases of epiphora and dacrocystitis with nasal trouble were often very difficult to deal with, and caused extreme discomfort to the patient. He could not say anything from experience as to these intra-nasal operations for dacrocystitis, but they appeared to be very successful. He had seen the two patients who had been operated upon by Mr. Harmer for the condition within two or three months, and, so far as he could judge from an inspection of the patients, the results were admirable, and the patients assured him they were quite comfortable, and were experiencing no unfavourable symptoms. Last year, Mr. Graham and Mr. Paton showed cases which had been treated by this method. He would like to know whether Mr. Paton had any further experience to relate in the matter.

Mr. Leslie Paton, in response to the President's question, said that Mr. Graham and he had had considerable experience of these cases. As he had not seen Mr. Graham for a few weeks, he could not state the number of cases which that gentleman had operated upon. As he, Mr. Paton, remarked last year, the operation was always supposed to belong to the nose and throat specialist, not to the ophthalmologist; hence, Mr. Graham usually dealt with them himself. In the first seven or eight cases seen by them he always assisted Mr. Graham with the operation. His own experience with the operation was not uniformly good. But the operation was usually attended with such good results that when the ophthalmic surgeon could not get a satisfactory result by simpler measures, West's seemed to be the operation of choice. But there were one or two kinds of case which did not do well with the operation. His own feeling was that it should not be done when acute suppuration was present; he remembered well one particular case, that of a girl, who had a very acute dacryocystitis. She was treated at the hospital for about a month, and the acute inflammatory

process had subsided before operation was attempted. At the operation there was very free hæmorrhage. He waited over an hour for the bleeding to cease, but it had not done so at the end of that time. Ultimately the operation was completed, and she did excellently. About six weeks afterwards, however, the opening was not draining well, perhaps owing to granulations having closed over the opening. There was no recurrence of the acute inflammatory condition, but she again had epiphora, and there was need for a further intra-nasal operation, in order to cure the condition. He understood that Mr. Coats had also had one or two cases which had not been satisfactory. He believed that Mr. Graham and he had had, in the last eighteen months, about sixteen cases of the condition, in which they had operated, and a very large percentage of those operations had been entirely successful. Mr. Graham had introduced a slight modification in the technique, which, in his opinion, rendered the operation more easy than formerly. When the flap of mucous membrane had been reflected, instead of cutting out, as West and Halle did, a portion of mucous membrane, it was turned back, and the bony part of the wall was removed. A fairly large hole was made in the bone, and then the sac was pushed through with the point of a probe. A large opening in the sac was made with a cautery. The mucous membrane was replaced over the hole, and the probe pushed a little further, and a corresponding opening made with the cautery in the nasal mucous membrane. No sewing was required. At the early operations which he did with Mr. Graham, they passed a piece of lead wire through and brought it down the nose for a couple of days, and he thought that was a distinct improvement.

Mr. W. H. H. Jessop desired to say on his own and Mr. Holmes Spicer's behalf, that they only sent to Mr. Harmer difficult cases of the condition, and they had both been much gratified by Mr. Harmer's success with those cases. One case which especially appealed to him was that of a child of two or three years of age, who

had much epiphora on both sides. He inserted a style on one side, but on the other no probe would pass down the nasal duct on account of some disease at its entrance. In that case Mr. Harmer did the operation on both sides, and next day, and ever since, the child had been perfectly well.

Mr. Charles Wray said that recently the tendency had been to resort to West's intra-nasal operation or some modification of it as the one and only treatment at our disposal for cases of dacryocystitis. He fully endorsed Mr. Paton's opinion that the patients should be referred to the rhinologist only when the resources of the ophthalmic surgeon had failed, and this would very seldom be the case. When the upper part of the duct was closed by bone there were two alternatives, viz., to remove the sac or make a permanent fistula between it and the middle fossa of the nose or the antrum of Highmore. Of the two, removal of the sac was the safer operation so far as the eye was concerned. When the bony stenosis was limited to the lower part of the duct he preferred to use a style about 2 mm. thick, and a gold-plated one could be obtained for 2*s*. 6*d*. It could be introduced in the ordinary way *viâ* the slit canaliculus, but a better plan was to introduce it into the sac by means of an incision immediately below the palpebral ligament, using a bent Bowman's probe passed along the canaliculus as a guide. Afterwards a Couper's probe was passed down the duct so as to admit the 2 mm. probe, and there was no objection to forcing the instrument into the nasal fossa. Fracture of the posterior wall was no more harmful than the trauma of the intra-nasal operations, and if the style were left *in sitû* for some weeks callus would form around it and prevent re-closure. It was impossible for ophthalmic surgeons to approve of an operation that converted the sac into an accessory sinus of the nose, and the ideal procedure was one that left the anatomy of the parts as nearly normal as possible. It was interesting to hear that a cataract

had been successfully removed after a West's operation, but a material point was, had it been done without or with a conjunctival flap. Not long ago a case was shown in the Laryngological Section of the Royal Society of Medicine in which the sac was distended when the patient sneezed or blew his nose. It was reassuring to hear that the artificial opening into the sac contracted into quite a tiny orifice, but if it might contract so much there was some ground for believing it might close entirely.

Mr. G. Coats desired to record the fact that he had seen two cases in which the results were not good. In both the sac was much dilated and its contents of very gelatinous consistence. Not improbably this was the cause of failure, the thick muco-pus being unable to drain away unless the cavity of the sac were made absolutely a part of the cavity of the nose.

Mr. Leighton Davies said he thought a great advance had been made in the treatment of these cases by the introduction of anastomotic methods. He considered that that method was infinitely superior to the method of using probes and styles. He had been doing Toti's operation for four or five years, and had performed it on about twenty cases. All but three of them had been successful. The suppuration was cured in every case, but in two of his recent cases there remained a slight epiphora after the operation. He was glad to hear Mr. Harmer's reference to the disadvantages which were supposed to attach to anastomotic methods. He had not himself seen regurgitation into the conjunctival sac on blowing the nose, nor infection spreading upwards. With regard to probes and styles, he did not think many cases were cured by them. One might make a comparison between this condition and stricture of the urethra. Patients with the latter were seldom cured without having bougies passed occasionally. It was the same with lacrymal stenosis; and the surgeon who did the operation might not know that probes had already been passed, because when relapse took place the patient might consult somebody else. He did not think

acute cases should be treated by the anastomotic method; he always treated them by syringing, generally using protargol, to cleanse the sac before operating in order to try to eradicate all the pus. He did not consider that the anastomotic method was suitable to all cases, because it would be found, on going carefully into the pathology, that some of them were tuberculous. That was so, he thought, in those cases in which the contained material was very thick and purulent when drawn out with the syringe. In one of his patients one could scrape out almost calcareous material, and in such cases he always removed the sac.

Mr. Priestley Smith wished, without criticising the newer operations, to say a word in favour of treatment by means of a silver style, because he believed that cases which were quite amenable to this treatment were at present sometimes dealt with by graver methods. He would not describe the details, as he had done this fully in the *Ophthalmic Review* in 1911. He would only mention that the style was not a tube but a piece of pure unannealed silver wire moulded by the surgeon to suit the individual case. He had cured a large number of chronic cases in this way, including many with lacrymal fistula. When the patient was cured the style was preserved in an envelope bearing a reference to the case, so that in case of relapse it might be replaced for a while. Occasionally patients returned for that purpose. More frequently they sent others to be treated in the same way.

Mr. Harmer agreed that certain cases must be reserved for this operation at present, *i. e.* until more was known concerning it. Purulent cases seemed to be particularly suitable, but it was not always possible to cure them by one operation. At the first sitting it was important to make a window and incise the sac. If there were much bleeding, he thought it was a mistake to prolong the operation by waiting for it to stop. Drainage of the sac into the nose produced, in forty-eight hours, very material

improvement in the amount of suppuration; and if the epiphora persisted, and the opening which had been made was not a satisfactory one, it was easy to cocainise the patient again, and remove more of the sac when it was less inflamed and less liable to bleed. With regard to the complications which were likely to follow this operation, he did not think they would be serious, because the tendency was for the fistula to become stenosed; and in the rare cases in which an opening was visible some time after the operation, the opening was found to be contracted to a pin-hole, and so it was unlikely that a patient, when blowing the nose, would project any material into the eye.

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2. *A papilloma in the upper canaliculus.*

By F. A. Juler.

The condition of papilloma in the lacrymal canaliculus appears to be of sufficient rarity to justify a short report of the following case. In a somewhat limited search into the literature I have been unable to find a similar case.

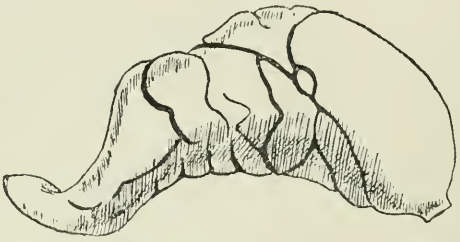
The growth was removed by Mr. Mayou at the Central London Ophthalmic Hospital from the patient's left upper canaliculus. Clinically there was a swelling of the lid in the neighbourhood of the canaliculus, and through the dilated punctum a smooth flattened knob of tissue was presenting. The canaliculus was slit for a few millimetres, and with a small spoon the tumour was lifted out complete without traction or further cutting.

The tumour was of a sinuous pear-shape, the long axis measuring 10 mm., the short axis 4·5 mm. When in position the pointed end was directed towards the lacrymal sac, and the ovoid end showed a flat-topped knob which had protruded through the punctum. The surface

of the tumour showed numerous furrows. There was no apparent pedicle.

Microscopically the structure is that of a papilloma. The epithelium is stratified, and consists of fifteen to thirty layers of cells. The stroma is made up of a sparse network of connective tissue containing capillaries. Here and there in the stroma leucocytes are numerous, but

Fig. 43.



Papilloma from canaliculus. Length, 10 mm. × 4.

sections were stained by Gram's method for micro-organisms with a negative result.

VI. DISEASES OF THE CORNEA.

1. *Remarks on dystrophies of the cornea and glaucoma, with especial reference to a familial variety of the former.*

By J. Gray Clegg.

The paper I am now about to read was suggested by the two cases I showed yesterday.

When a patient of middle age complains of seeing coloured rings round artificial lights, the first idea that occurs is whether glaucoma is present, but the spectral colours are produced whenever the anterior refractive surface of the cornea is fogged by conjunctival secretion lying on it, or by changes in the anterior epithelium or, including sometimes, the superficial substantia propria itself. No particular difficulty arises in differentiating between conjunctival secretion and corneal haze, but in my experience complaint is often made of halos without there being any corneal haze sufficiently marked to be visible objectively to the examiner by any means open to him. On the other hand, even if there is definite haze in the substantia propria not involving the surface no spectral rings are produced. It would therefore follow that spectra only occur when the anterior surface of the cornea is irregular, and not perfectly smooth, presenting to the rays of light extremely numerous fine refractive surfaces. Clinical experience agrees with the deductions of physical science. Such changes in the anterior surface, of course, occur when there is a rise of tension in the globe. The changes are well known and

are due to interference with nutrition by partial blockage of the lymphatics, etc., and withdrawal of the fluids that are necessary for the maintenance of perfect clarity. A return to the normal can, however, even take place within half an hour if tension is lowered by, say, the instillation of eserin.

It is also well known that somewhat similar changes may occur from other causes, but this is, I consider, not commonly the case. Such are described as dystrophia epithelialis of the cornea by Fuchs and are characterised by non-inflammatory degenerative changes.

Females are more commonly affected than males.

The appearances exactly resemble those seen in glaucoma. Fuchs stated that it occurs once in every 2000 patients. In my experience it is decidedly rare, at any rate in a marked degree. The degeneration is not confined to the epithelial layer, but extends into the superficial strata of the substantia propria. This is borne out by the fact that in the first case about to be described the haze is constantly present, but halos are not always observed, and further, that on some occasions the surface of the cornea is quite smooth, whereas on others it is somewhat dull and vesicles appear. The whole trouble is undoubtedly due to lowered vitality in the cornea of dystrophic origin.

When a non-glaucomatous case presents itself the difficulty is to make sure that no true rise of tension ever occurs.

Yesterday I showed two sisters in whom this comparatively rare condition exists. A third younger sister has an affection of the eyes of the same nature.

In addition the paternal grandmother had a film on one eye and was told nothing could be done, and one paternal uncle, æt. 80 years, had lost the sight of one eye. I have, however, only actually seen the two sisters myself. It would appear, therefore, that there is a familial variety of corneal dystrophy. I now give particulars of the middle sister, L. F—, æt. 44 years.

L. F—, female, æt. 44 years, first seen on February 10th, 1915. Coloured circles round lamplights had been noted for three or four months now and again. They disappear after a few hours and are replaced by radiating streaks of ordinary light. Health had been fairly good, but her medical man, Dr. Sturrocks, of Eccles, informed me that she had irregular action of the heart, although there was no valvular trouble. The cardiac muscle was weak, and the arterial blood-pressure was very low. Some general anæmia present. On examination I noted at once the corneal haze. The A.C. in each eye was of good depth, the pupils were of medium size, equal, and reacted to light and accommodation.

R.V. $= \frac{5}{24}$ $\bar{c}$ + 1 D. sph. $\bigcirc$ — 6·5 D. cyl. ax. vert. $= \frac{5}{12}$; + 2 D. sph. $\bigcirc$ — cyl. = J. 2.

L.V. $= \frac{5}{60}$ $\bar{c}$ — 4·5 D. cyl. $= \frac{5}{9}$; + 1 D. sph. $\bigcirc$ — cyl. = J. 1.

T.N. by finger test, and with Schiotz's tonometer R. with 5·5 grm. = 4 mm., and L. 5·5 grm. = 3 mm. of displacement of the needle indicator. The haze appeared to be below the surface, and was fairly evenly distributed all over each cornea.

The lenses and vitreous bodies were clear. The right disc presented a medium sized physiological cup, but the left disc was flat. The fields were full normal.

I prescribed gutt. pilocarpin nitrat. gr. iv ad ℥j.

On February 12th I could by the loupe distinguish that the haze was partially resolvable into very fine grey foci and some irregular lines. I advised gutt. dionin gr. iij ad ℥j to be used three times daily for two days, alternating with three days without, in addition to the pilocarpin.

February 26th.—Some stripping of the surface noted and a number of small vesicles. The pilocarpin was changed to eserin gr. $\frac{1}{4}$ ad ℥j.

March 1st.—Still sees rings; no definite vesicles on right. The visual acuity had improved to R. $\frac{5}{6}$ partly, and L. $\frac{5}{5}$ a few. Fields again taken. No change.

March 17th.—No eserin used to-day except at the

examination. It made no difference in the tension or acuity of vision. There is some diminution of the corneal sensibility, more marked in the left eye.

April 23rd.—No staining of the cornea is produced by fluorescin to-day. In the lower periphery of each cornea, especially in the left, there is a crescent greyer in appearance than the rest of the cornea.

She had been seen previously by an oculist, who, no doubt being struck by the haze and the history of rings, gave pilocarpin and said that operation would probably have to be resorted to. The same problem had to be attacked by me, but after going carefully into the refraction, the visual acuity, the tension, and fields, I came to the conclusion that dystrophia was the proper designation.

I have not yet adopted the bolder course of dilating the pupils with homatropin and later with atropin, awaiting the census of opinion of this Congress.

The elder sister, M. A. W—, æt. 47 years, but looking decidedly older, had fortunately been under the care of Dr. Doyne, at the Oxford Eye Hospital, where detailed notes of all cases are kept, and from them I give you the following, extracted by Dr. V. Offen-Cussen, the house-surgeon there.

"Patient M. A. W—, 1896.—Large bulla on L. cornea, central ulcer, pupil sluggish.

"R. $\frac{6}{12}$, L. $\frac{6}{60}$; T. subnormal.

"Bulla cauterised. Gutt. eserin.

"Discharged March, 1897. Bulla no longer present. Diagnosed bullous keratitis.

"October, 1900.—Epithelium of L. cornea greatly disturbed.

"Epithelium of R. cornea slightly disturbed on outside.

"Both pupils react to light.

"T. N., R. and L. R.V. = $\frac{6}{18}$ partly, $\bar{c} - 1$, sph. = $\frac{6}{12}$ partly.

"L. Fingers at three feet.

"*Treatment.*—Gutt. eserin to both eyes.

"March, 1901.—L. faint staining c fluorescin.
R. quiet.

"May, 1901.—R. E. some bullæ down and in on cornea, produced by pressing down lid.

R.V. = $\frac{6}{9}$. L.V. = $\frac{6}{60}$.

"July, 1901.—R.V. = $\frac{6}{24}$. L.V. = $\frac{6}{60}$ (?)

"Sight varies. Diag. bullous keratitis.

"These are the essential points of the history of the case. I may add that the patient attended monthly for both attacks, and that no rise of tension is recorded.

"The fields were taken in 1901 and only show a very small contraction on the temporal side."

Yesterday I seized the opportunity of examining this patient. The conjunctivæ were normal. Both corneæ were markedly hazy with irregular surface, but the upper vertical quadrant was almost smooth. There was no staining of the right corneal surface by fluorescin, but the left showed numerous fine irregular patches of green. In the left were several light brown calcareous deposits, arranged in an irregular manner in the vertical meridian. Towards the lower periphery in each eye was a greyer band, with bullæ in the right cornea only. The right cornea had low sensibility, but the left was almost anæsthetic. With the loupe an irregular network of grey lines was discernible in each. The A.C.s were of full normal depth. The irides were grey-blue in colour. The pupils were medium in size and reacted to light. The lenses and vitreous bodies appeared to be clear. The discs were of good colour and flat. Although the fundi could not be perfectly seen, there were certainly no gross lesions present. The tension of the R. was normal and that of the L. − 1. The fields, as taken by the hand, were of full extent.

R.V. with − 3 D. sph. = $\frac{3}{36}$, but with + 3 D. sph. = J. 14.

L.V. = J. 20.

Some years ago I had under my care a case of vesicular keratitis which illustrates the difficulties of deciding

whether, in a patient with steamy cornea and definite vesicles, the condition is really of glaucomatous origin. I am not yet perfectly satisfied as to the true nature of it, but I regard it as a dystrophy somewhat similar to that found in the familial cases.

R. B. W—, male, æt. 50 years, was first seen in July, 1900. At that time the R.V. $\bar{c}$ + 4·5 D. sph. $\supset$ + 1 D. cyl. ax. vert. = $\frac{5}{4}$ + 7 D. sph. $\supset$ cyl. = J. 1, and L.V. $\bar{c}$ + 5 D. sph. = $\frac{5}{9}$ + 7 D. sph. = J. 2.

He was suffering from a slight attack of iritis in the left eye. He soon improved with atropine and general treatment. Each anterior chamber was noted to be rather shallow, and there was some irregular branching pigmentation in the retina of the right eye. He recovered and did not reappear till May 14th, 1902, when the left eye had been affected one month. T. + 1, V. = J. 19, only. Eserin was instilled, and one hour later L.V. = J. 4, the corneal condition having improved. Next day I noted an infiltrated area on the cornea down and out.

On the 17th a bulla appeared at that spot.

On the 19th steamy cornea. May 20th: T.N. Whitish bleb on cornea and a clear bleb on sclera at inner side. May 28th: I scraped the corneal bullæ. June 13th: T. full. Field, as taken by perimeter, showed slight general peripheral contraction. In August iridectomy was done above and the conjunctival bleb cut off, leaving a small pit. In September, 1902, the L.V. = bare P.L.

Leucoma down and out on cornea with two small vesicles. The cornea was all hazy. In 1904 the right eye first became affected, and the patient several times saw halos round lights. The vision in it remained fairly good, viz. $\frac{5}{5}$ partly. Gutt. eserin gr. $\frac{1}{2}$ ad $\overline{3}$ j. was prescribed.

In 1905 he often complained of halos in spite of the eserin; T. full. Uses the drops when believes an attack of dimness pending.

In November, 1906, he stated he had to use eserin nightly if coloured rings were not to be in evidence about

10.30 p.m. The field remained good and the fundal appearances exactly as before, the disc being of good colour and flat.

October, 1908.—Vision still good in R., but no P.L. in left, which was grey in appearance, the coloboma being only just visible. By 1911 he was still using eserin at times. A.C. shallow; no change in fundus. In August, 1911, I thought slight cupping of disc present. In May, 1912, the refraction had altered to + 5 D. cyl. 60° = $\frac{5}{5}$ some.

May 30th, 1914.—I was sent for and found sight slowly fading. Schiotz's tonometer gave with 5·5 grm., 4 mm. of displacement of needle, but on June 1st it was only 1 to 2 mm. The field was not much contracted, but I decided to trephine above. A small peripheral iridectomy was done. Anterior two thirds of a 2 mm. disc was cut away. Healing took place without any incident of note; filtration was good.

June 19th, 1914.—R.V.: $\bar{c}$ + 0·5 D. sph. ⌒ − 4 D. cyl. 60° − 2 $\frac{5}{18}$ partly, and + 5 D. sph. ⌒ − 4 D. cyl. = J. 1.

The trephine hole was filtering well, but there was a grey haze of the cornea down and out.

On July 14th a definite bulla appeared on the right cornea.

In August, 1914, he still experienced occasional rings of colour. T.N.

He was last seen in November, 1914, when the R.V. = $\frac{5}{18}$ partly, $\bar{c}$ − 0·5 D. sph. ⌒ + 1·5 D. cyl. 50° − = $\frac{5}{9}$ partly, and + 2 D. sph. = J. 2.

The superficial layers of the cornea down and out were grey and hazy, and presented raised epithelium. The trephine hole was filtering well, the A.C. was shallow, and the fundus showed no change.

As a contrast I have had recently a case at the Royal Eye Hospital, Manchester, where there was marked rise of tension, and the eye appeared to be affected with simple acute glaucoma. Before the operation of trephining the

cornea was clear, but some ten days afterwards a vesicular keratitis appeared with absolutely normal or subnormal tension, and within a few days more definite signs of cyclitis, with keratitis punctata, appeared, the whole due to syphilis, as the Wassermann was strongly positive.

Here we have a case resembling dystrophia cornealis with vesicular formation appearing after a successful trephining operation in an eye in which rise of tension could not occur.

Lastly, I have, of course, met with cases, but comparatively few, of true vesicular and bullous keratitis in which no doubt existed but that there was no tension element. They are merely advanced cases of the same disease—dystrophy.

My view regarding the sisters is that no operation will be of service. So far I have used pilocarpin, and later eserin, because I find that the halos are rather less in evidence than when the eyes are not under the influence of these drugs, probably for two reasons: (1) the pupils are kept smaller and definition of objects is therefore sharper, and (2) the extended iris no doubt, with its widely open crypts, allows of freer circulation in the intraocular fluids, and therefore, the nutrition of all parts of the eye is better than with the iris in its normal position, or if dilated.

On the other hand, trephining the sclera is an operation that is so free from risks that it can be done without great fear of ill consequences, even if undertaken where the indications of tension are not absolutely definite. The only other measure proposed is that by Fuchs of scraping the anterior epithelium if that alone were affected.

I have brought forward this subject to obtain the views of fellow members as to frequency of occurrence of dystrophia epithelialis, and as to the best mode of treatment. In addition to the use of weak eserin, for the reasons given, I have prescribed a solution of dionin, one drop in the eye four times daily for two days, alternating with three days without for the purpose of improving the lymphatic circulation.

PLATE VI.

Fig. 1 illustrates Mr. Sydney Stephenson's paper on Striate Clearing of Corneal Cicatrices (Coarse Type) (p. 253).

Fig. 2 illustrates Mr. Sydney Stephenson's paper on "Blue Sclerotics" (p. 274).

Fig. 1.

Fig. 2.

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Attention to the general health will, of course, be of service.

Massage of the cornea might be tried.

Undoubtedly the general condition of the two sisters shown has some relation to the corneal affection, for the middle sister had markedly low arterial tension, and the elder is prematurely old in appearance.

Mr. F. Richardson Cross, referring to the last paper, said it raised the question of the treatment of bullous keratitis. His experience coincided with Mr. Clegg's, that it was a rare condition and difficult to remedy.

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2. *Striate clearing of corneal cicatrices (coarse type).*

By Sydney Stephenson.

(With Plate VI, fig. 1.)

The patient, a man, æt. 65 years, has been under my observation since February 2nd last. He was troubled with inflamed eyes from the time he was two or three years old until he reached manhood. At twelve years of age he suffered from abscesses on the left side of the neck, the scars of which are still in evidence. He has never enjoyed good health. Three years ago his hair all fell out, so that he is now compelled to wear a wig.

Present state.—Both corneæ are the seat of leucomata, the right being more affected than the left. The opacity in the right eye (as shown by the illustration) is roughly of mushroom-like shape, its stalk lying at the lower edge of the cornea. It covers the pupil. The peculiarity is that it is traversed by a number of clearer lines of varying width, so as almost to be broken up, as it were, into a number of smaller or larger leucomata. The changes are best seen on using a magnifying glass. Towards the upper margin of the cornea is a curvilinear and denser opacity, which fits cap-like over the main leucoma. The

curious striate changes can scarcely be said to be present in the leucoma of the left cornea.

R.V. $\frac{6}{36}$. L.V. $\frac{6}{24}$.

Remarks.—The condition resembles the case shown by me at the 1913 meeting of the Society (see vol. xxxiii, p. 79, with illustration), although on a coarser scale. In both the corneal condition was of long-standing, and in neither did the striate condition show any change while the patient was under my personal observation. The cause of the corneal opacity in my first case was interstitial keratitis (syphilis), and in the present one repeated attacks of phlyctenulosis in early life. The clear lines remind one of blood-vessels, and it is probable that such were present when the opacity was recent. The present condition has likely followed resorption of the vessels, and, inasmuch as many of the striæ are wider than any corneal vessel, there has almost certainly been some clearing of the opacity in the neighbourhood of the vascular streams.

It is possible that the striate metamorphosis is commoner than at present suspected, for it may be readily overlooked in the absence of a careful examination.

Mr. W. T. Holmes Spicer said he had been interested in this condition since he saw his first case in 1894. He was surprised at the small amount of attention which had been paid to the subject. The only contribution he knew of was by Fuchs ten years ago. In Mr. Nettleship's collection there was a drawing of the condition dating from 1878. Therefore these lines had been recognised for a long time. If every case of nebula were watched, he thought the condition would be found to be very common. As to their nature, he was sure they were due to the presence of blood-vessels, but in some cases the markings were much wider than blood-vessel tracks. The reason for that he did not know, but probably some contraction took place superficially. Apparently they took two forms. Sometimes they were perfectly straight lines, meeting each

other at angles, and thus forming geometrical figures; in other cases the lines appeared in interstitial keratitis, as in a case of which he showed a drawing. Another kind occurred where there was a fascicular ulcer with a track of blood-vessels running to it, ending in an opaque round blob, the scar of the ulcer. A long time afterwards an opaque cap formed on the top of the blob, and it was fringed on the inner side by the terminations of the vessels, so that it looked like the head of a mushroom. He thought it very likely that the fringe was due to a hyaline deposit in the cornea, because it was yellow in colour, and in one or two of the cases he had seen there had been circular holes such as were found in association with hyaline deposits.

Mr. J. B. Story said he had seen such lines in cases of interstitial keratitis, and had observed blood-vessels grouped in the same manner in cases in which he had not afterwards detected any such lines. Where these lines are found he thought they were due to degenerate blood-vessels, or a clearing round the course of shrivelled vessels. He believed the explanation given by Mr. Stephenson and Mr. Holmes Spicer to be correct.

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3. *Iridescent corneal scar.*

By A. C. Hudson.

Louisa T—, æt. 50 years, attended the Royal London Ophthalmic Hospital on March 11th, 1915, complaining of pain in left eye of three days' duration. The patient had attended the hospital for dendritic ulceration of the left cornea two years previously (when the ulcer was scraped and carbolised), and there was a history of several attendances previous to this, extending over a period of more than five years.

On examination there was found to be a central denuded area in the cornea with ragged surface and incomplete opaque grey fringe, apparently the site of an old scar.

The eye was treated with application of 1 per cent. nitrate of silver solution and pad and bandage, and atropine and boracic acid solution were prescribed.

April 22nd, 1915: Corneal lesion healed. The smooth scar is remarkable for the presence at its inner border of an iridescent blue and indigo halo, situated at the level of the deepest layers of the cornea.

Mr. J. B. Story said he would like to hear an explanation of the iridescent hue in the cornea of this case.

Mr. Coats suggested that it might be a diffraction effect.

VII. DISEASES OF THE SCLERA.

1. *Hyperplasia, with colloid and amyloid degeneration, of the episcleral and circumdural fibrous tissue.*

By George Coats.

(With Pls. VII, VIII, and IX.)

Summary.

Growth commencing at the age of 25, in the left conjunctival or sub-conjunctival tissues and subsequently affecting also the orbit. History of an injury ten years, and of an infection, possibly syphilitic, eight years previously. Very gradual progress during a total period of more than seventeen years. General health unimpaired throughout; blood examination negative; stationary enlargement of the thyroid without symptoms of Basedow's disease, cretinism or myxœdema.

Palpebral conjunctiva not affected by the growth; in the ocular conjunctiva it had a "fresh semi-translucent" appearance, suggesting "solid œdema"; colour darker below than above; a ring of growth surrounded and partially overlapped or encroached upon the cornea. Subsequently the whole eyeball became surrounded by enormous indurated, nodular masses, and its movements were much impeded. Vision gradually sank from $\frac{6}{12}$ to $\frac{2\frac{1}{2}}{60}$. Cupping noted even at the earliest examination, but presence of raised tension considered doubtful, the field of vision probably full, at least at first. Diagnosis = gummatous infiltration of capsule of Tenon. Treatment by repeated

anti-syphilitic courses carried the length of salivation, and occasional scrapings and partial excisions; both methods inefficacious.

Pathologically the condition is an enormous overgrowth of the episcleral and circumdural fibrous trabeculæ, with amyloid degeneration of the affected tissues. Arteries also show amyloid degeneration affecting both the muscular and adventitious coats. Both the hypertrophic and the degenerative changes much more advanced anteriorly than posteriorly. Anteriorly also calcification of the fibrous bundles, and plaques of ossification on their surface, are present, and there is considerable lymphocytal infiltration in the interspaces between the trabeculæ; posteriorly lymphocytal infiltration is present but is less in amount, and calcification and ossification are absent. Giant cells of "foreign body" type are everywhere common. The lymphocytal infiltration has penetrated along the perforating vessels and invaded the whole of the uveal tract, the iris, however, being comparatively slightly affected. Ciliary muscle shows amyloid degeneration. Optic nerve shows partial atrophy but no hypertrophy or amyloid degeneration of its trabeculæ. No true glaucomatous cupping.

The patient first came under the care of Mr. A. Quarry Silcock, at the Royal London Ophthalmic Hospital on May 20th, 1889. At that time his age was 27, and the condition of the left eye had been noticed for fifteen months. It began with a certain amount of pain, photophobia and lacrymation, and with the appearance of a blood-shot area "spreading from the nose across the sight." Some diminution of vision was first noticed about ten months before the date of his visit.

Ten years previously he had fallen against the spout of a watering pot, and scars were present at the rim of the orbit on the outer side; there was no other history of injury. Eight years previously he had had an infection believed to be syphilitic, but not followed by roseola or sore throat. He had suffered from rheumatism, and there

was a doubtful family history of rheumatism. He had never had any severe illness. There was considerable symmetrical enlargement of the thyroid without pulsation. No palpitation.

The ocular condition was as follows: Right eye externally normal, pupil active, fundus normal, vision $\frac{6}{6}$. In the left there was considerable "solid œdema" of the ocular conjunctiva, and a ring of similar appearance an eighth of an inch thick surrounded the cornea. The palpebral conjunctiva was free from change. Proptosis was present, but the ocular movements were unimpeded. The pupil reacted sluggishly, the media were clear, the disc showed cupping estimated at 6 D. in depth; pulsation was observed in the arteries. Vision with correction of a small amount of myopic astigmatism was $\frac{6}{12}$. Tension possibly raised. Field of vision good as tested by the hand.

A provisional diagnosis of gumma infiltrating Tenon's capsule was made, and on June 18th a portion of the growth was removed for microscopical examination. At this time it was thought that the conjunctival swelling and proptosis had diminished. The pathological report was, "Round-celled growth (?) sarcoma." No fungation occurred through the wound, however. Treatment consisted in mercurial inunctions till the gums were affected, and pot. iod. gr. xx thrice daily.

From this time the patient was under Mr. Silcock's observation till February, 1891. Nearly sixteen years later, in October, 1905, he came under the care of Mr. Holmes Spicer, to whom I am indebted for permission to make use of the case.

At first the presence of true proptosis was considered questionable by some observers, and during an exploratory operation in November, 1889, it is noted that nothing could be felt behind the globe. Early in 1890, however, exophthalmos appears to have become quite definite, and there was thought to be slight limitation of movement. The lids could be closed over the swollen conjunctiva.

Subsequent progress was extremely slow. In March, 1890, it is noted that the surface of the growth was much darker below than above, where it had a fresh semi-translucent appearance. The application of compresses resulted in flattening of the growth, so that the cornea was uncovered except for a small portion above. In December, 1889, and several times during 1890, the growth was incised and scraped, but in spite of this in November, 1890, it is noted that the ocular condition was little altered except that the conjunctival and subconjunctival vessels were more enlarged.

Early in 1891 there was some progress. The width of the palpebral fissures from canthus to canthus was R. 2·6 cm., L. 3·5 cm. The growth overlapped the cornea, especially above, and there was some encroachment also elsewhere; below, it had a florid purple colour with some very large tortuous veins. The pupil reacted normally to light; the cup was estimated to have a depth of 4 D.; the retinal veins were full and tortuous, but there was no arterial pulsation. Tension was not easily estimated, but was considered to be no higher than in the right eye. Mercurial treatment was several times carried the length of salivation.

After this the patient disappeared till October 26th, 1905. In the interval there had been a very gradual increase in the proptosis and diminution in the vision. Enormous indurated masses now surrounded the left eye-ball, especially above, where they encroached on the cornea to nearly half its diameter; elsewhere also the cornea appeared to be invaded, and on its inner side there was a small ulcer. The growth had a nodular, irregular surface, and was covered with stretched and hypertrophied conjunctiva. He could still close his eyelids, but all ocular movements were much impeded. True proptosis was present, but was less than a cursory glance might have led one to anticipate, the appearance being exaggerated by the masses of growth. The anterior chamber was of normal depth; the pupil slightly active; the disc atrophied and deeply cupped; the fundus otherwise normal; the vitreous

or lens a little hazy. Vision $\frac{2\frac{1}{2}}{60}$ slightly improved with − 1 D. sph.

Four days later a partial exenteration of the orbit was performed, the lids not being removed. Healing was uneventful, and after several operations a socket fit to hold a small eye was obtained. When last seen in March, 1906, there was no sign of recurrence. A blood-count in January, 1906, gave the following result:

Hæmoglobin, 95 per cent.
Red corpuscles, 5,175,000 per c.mm.
White corpuscles, 7000.

Differential leucocyte count:

Polynuclears, 68 per cent.
Lymphocytes, 23 per cent.
Large mononuclears, 7 per cent.
Eosinophiles, 1·5 per cent.
Mast cells, 0·5 per cent.

Throughout the whole course of the disease the goitre underwent little, if any, change.

Pathological Examination.

Macroscopical Examination.

The specimen consists of the globe and a portion of the optic nerve. Considerable tumour masses are attached anteriorly and posteriorly, but at the equator most of the growth has been stripped off, leaving the sclera clean and apparently uninvaded. The cut surface of the tumour has a granular or nodular aspect. It is of hard consistence and closely adherent to the globe, especially anteriorly. Above, a fold of conjunctiva covers, but does not adhere to the cornea, in about half of its vertical extent; elsewhere the growth overhangs or encroaches upon the cornea over a limited area. A small ulcer is present on the inner side. The dural sheath is greatly thickened, and is surrounded by dense masses of the growth. The nerve itself appears

to be uninvaded and normal. The recti muscles are much hypertrophied, but not infiltrated.

The globe was fixed in formalin and cut in serial section. Transverse sections of the nerve were also prepared, and pieces of the tumour were subjected to various methods of special staining.

Microscopical Examination.

The histology of the growth is best studied in the vicinity of the optic nerve, where the changes are less advanced and less complicated by lymphocytal infiltration.

The dural sheath has undergone enormous thickening, due to a process of hypertrophy or simple proliferation of its component fibrous tissue (Pl. VII, figs. 1 and 2). No precise measurements can be given, since the outer boundaries, though discernible in places in a rough kind of way, are not exactly delimitable, but pass into coarse masses of hypertrophied fibrous tissue which extend as far as the edge of the tissue removed; the thickening probably varies from three- to four-fold. The normal orbital adipose tissue is represented only by a few groups of isolated cells.

Structurally the innermost layers of the dura differ little from the comparatively loose, wavy, fibrous bundles, with scanty nuclei, which are normal in this situation. More externally, however, the bundles are coarser, not so wavy, showing less of a fibrillated structure; in places quite homogeneous and structureless. Such degenerated areas are freely intermixed with, and occur as islands among, more normal wavy fibrous tissue. The nuclei are probably for the most part preserved, but are scattered by the swelling of the intervening fibrous bundles; they lie chiefly in small slits and clefts of the tissues.

The nature of this pathological alteration in the fibrous bundles is most apparent in areas where the tissues are somewhat loose owing to the persistence of some remains of orbital fat. In such situations it can be seen that many of the smaller fibrous bundles are converted into

PLATE VII.

Illustrates Mr. George Coats's paper on Hyperplasia, with Colloid and Amyloid Degeneration, of the Episcleral and Circumdural Fibrous Tissue (p. 257).

Fig. 1.—Transverse section of the optic nerve and dura. The nerve is atrophic; its trabeculæ, the pia mater, and the arachnoid are unaffected; the inter-vaginal space is uninvaded. The dura mater is enormously thickened; its innermost layers show normal wavy fibrous-tissue bundles; its more external have a less definite structure and show homogeneous areas (not well seen with this low power); its outer limits are ill-defined, and pass into greatly thickened and indurated trabeculæ traversing the orbit.

Fig. 2.—Longitudinal section of the nerve entrance. Lymphocytal infiltration is here more pronounced in the circumdural tissues. Atrophic cupping of the disc, but with little or no bowing backwards of the lamina cribrosa. Infiltration of the choroid with lymphocytes.

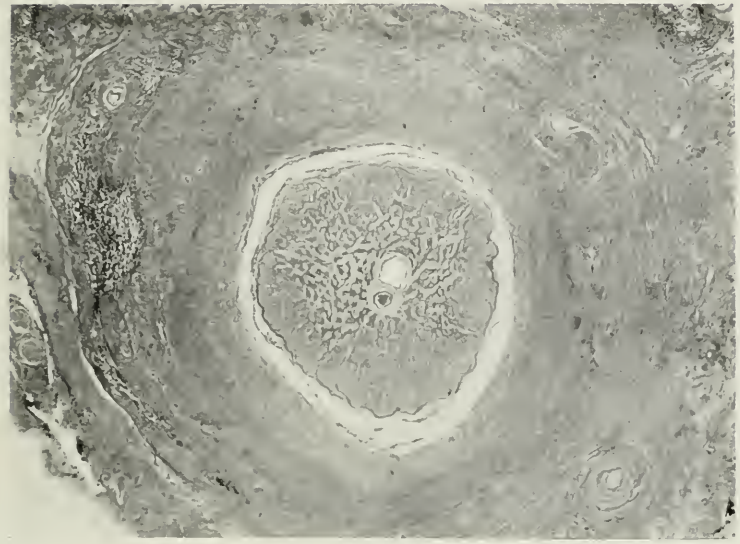

Fig. 1.

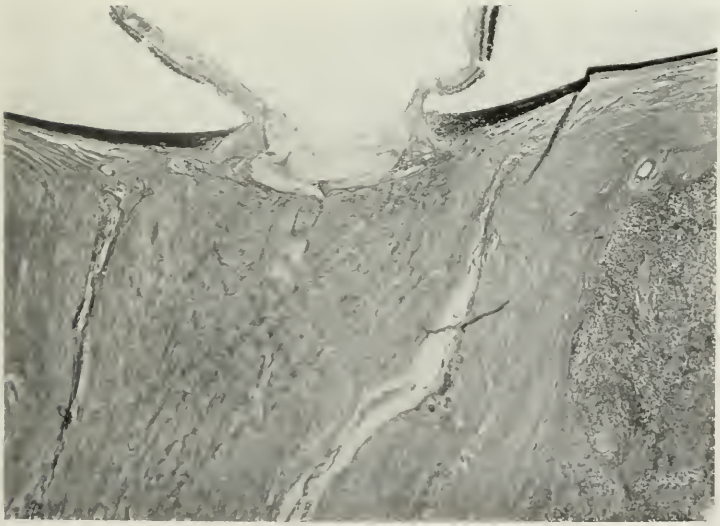

Fig. 2.

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cylinders of a swollen homogeneous substance with flattened endothelial nuclei applied to their outer surface, but without nuclei in their interior. Transverse sections of such bundles have often a sinuous contour, with bays into which the ensheathing nuclei sink. These nuclei belong to a fine fibrous sheath, which is sometimes brought into prominence by becoming a little separated from the contained bundle, owing to slight shrinkage of the latter. The degenerated trabeculæ are not always quite homogeneous; in places slight differences of staining are discernible, indicating a composition from several rounded masses (in transverse section); a structure which corresponds with the sinuous, tuberculated outline of the general contour.

The degree in which the tissues are converted into this homogeneous substance varies in different situations; in places the change is absent; in places only a few individual bundles are affected; elsewhere all the small trabeculæ show alteration while yet remaining discrete; elsewhere, again, larger masses of the same substance are present owing to the common involvement and fusion of contiguous bundles. This latter type of change is especially seen in the dense fibrous tissue of the outer layers of the dura and in the coarse trabeculæ which lead off from it. The degeneration is of somewhat irregular distribution, and islands of it are frequently surrounded by tissues of a more normal aspect.

Some of the smaller arteries show an exquisite degree of the change (Pl. VIII, fig. 3). Both the muscularis and the inner layers of the adventitia appear to be affected; the endothelium is proliferated in places but shows no other change; the outermost layers of the adventitia usually escape and form a kind of sheath round the diseased vessel. Here again the distribution is irregular, not all the arteries being affected; the veins appear to be free.

The results of the employment of special methods of staining for amyloid degeneration are not uniform. Anteriorly practically all the degenerate bundles and

masses give the characteristic coloration in a typical manner. Posteriorly, on the other hand, many bundles show pronounced homogeneous changes without the distinctive staining reaction, others show it in an incomplete or not wholly typical form, and the reaction is quite unequivocal only in the most degenerate areas and in the larger masses of homogeneous substance.

It seems evident, therefore, that the swelling and loss of structure in the least affected bundles represents a preparatory or preliminary stage towards the formation of true amyloid substance. It should be said, however, that as the nature of the case was not at first suspected no special staining tests were carried out on the unfixed tissues, and that subsequently the material at disposal for this purpose was somewhat scanty.

Giant cells are present in considerable numbers, more especially among the coarse trabeculæ in the vicinity of the dural sheath, and anteriorly in the subconjunctival and episcleral tissues. They are associated practically always with bundles which show a pronounced degree of degeneration; occasionally, however, an isolated cell is found among tissues which are but little altered. The giant cells lie always on the surface of the homogeneous masses, sending down processes into the recesses in their contour; although often deeply embayed they do not really penetrate the tissues. The surface of the masses beneath them has often a ragged, frayed aspect. These giant cells obviously belong to the same order as the endothelial cells already described as forming a sheath round the amyloid bundles; indeed, it is evident that they are direct derivatives (by a process of overgrowth) from these cells. They are usually of moderate dimensions, flattened against the surface of the bundles and with their nuclei grouped in the centre; they conform in fact to the accepted type of "foreign body" giant cells.

The dense tissues of the hypertrophied dura are free from all inflammatory infiltration. The looser tissues outside it are in places free, or show only a few plasma,

PLATE VIII.

Illustrates Mr. George Coats's paper on Hyperplasia, with Colloid and Amyloid Degeneration, of the Episcleral and Circumdural Fibrous Tissue (p. 257).

Fig. 3 × 120.—Amyloid degeneration of a posterior ciliary vessel. The muscularis is especially involved, some layers of the adventitia remaining unchanged. The endothelium is proliferated. Much surrounding lymphocytal infiltration.

Fig. 4.—From the anterior part of the globe. The episcleral and sub-conjunctival fibrous trabeculæ are converted into enormously swollen homogeneous masses with rounded, tuberculated contours. Some of these spring directly from the sclera, which, however, is itself almost free from change. Lymphocytal infiltration is universal and uniform among the trabeculæ, and is also present at the corneo-iridic angle and on the inner aspect of the ciliary muscle.

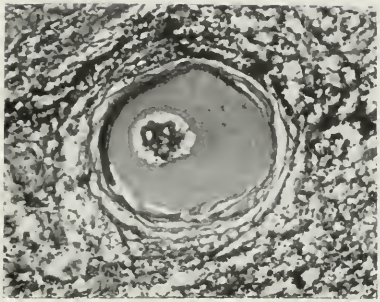

Fig. 3.

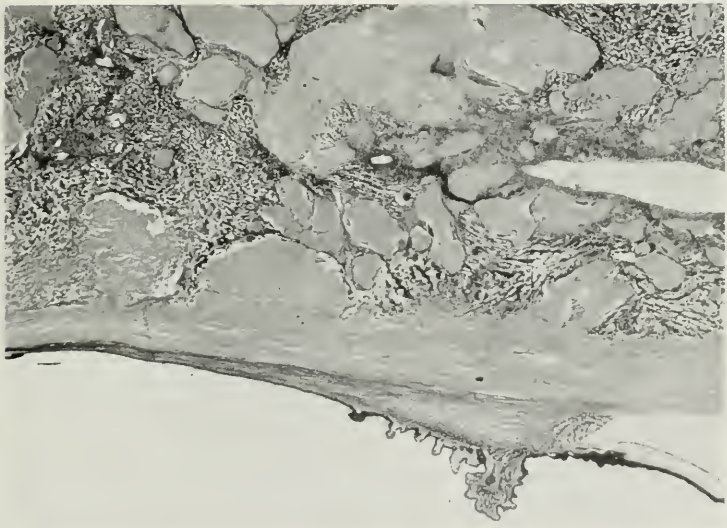

Fig. 4.

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and other chronic inflammatory cells. More commonly, however, the interspaces between the fibrous trabeculæ are occupied by a moderately dense infiltration of lymphocytes which show no special tendency to grouping in patches, or to the formation of follicles (Pls. VII, VIII, figs. 2, 3, 4).

The foregoing description, which refers more particularly to the changes in the vicinity of the nerve, may be applied with certain modifications to the growth in other situations. On the posterior aspect of the globe lymphocytal infiltration is dense, especially close to the surface of the sclera (Pl. VII, fig. 2). Coarse trabeculæ of fibrous tissue and large masses of amyloid substance are rare; rather all the small trabeculæ are swollen and more or less homogeneous, yet remaining distinct. There is some invasion of lymphocytes along the perforating vessels and nerves, but the sclera itself is intact. In the equatorial region, as already stated, the growth has been shaved off, leaving the sclera clean.

Anteriorly the changes, both degenerative and inflammatory, are much more gross (Pl. VIII, fig. 4). In the subconjunctival and episcleral tissues scarcely any fibrous trabeculæ are even approximately normal. Almost all are transformed into enormous swollen shapeless lumps, of homogeneous aspect, and with only a few nuclei, often degenerate, lying here and there in clefts throughout their substance. Patches of calcification are not infrequent, and many of the larger masses have flattened scales of true bone applied to their external surfaces. The deeper episcleral masses form a number of rough excrescences, arising by a broad base from the superficial layers of the sclera. The sclera itself, however, shows scarcely any change, and that only in its most external layers, where, in a few situations, some of the superficial fibres are somewhat swollen and homogeneous. Just as posteriorly the greatest hypertrophy of trabeculæ and amyloid change occurs in the tissues close to the dura, so here also it is the episcleral tissues which

are mainly involved; the orbital tissues some distance from the surface of the globe, in many places, show little hypertrophy and only isolated degenerate bundles.

Lymphocytal infiltration is here universal, dense, and uniform. The upper part of the cornea is overlapped by a long tongue of deeply infiltrated redundant conjunctiva, which possesses only a slight connective-tissue framework and does not show a very profound degree of amyloid change. Below, the lymphocytal infiltration is rather less; and the fibrous proliferation and amyloid degeneration do not extend beyond the normal limbus, where they end in a steep, but not overhanging edge.

The lymphocytal infiltration not only fills the intertrabecular spaces of the episcleral tissues, but has also spread along the perforating vessels into the interior of the globe (Pl. IX, figs. 5, 6). Anteriorly, tracks are visible along the line of the vessels leading to the canal of Schlemm; the tissues in the vicinity of the canal of Schlemm, the spaces of Fontana, and the iris root are considerably infiltrated, and numbers of lymphocytes lie free in the corneo-iridic angle. The stroma of the iris generally shows only a few scattered cells, but in the sphincter region there is again a concentration of lymphocytes as though the wandering of the cells had been stopped in this situation by their arrival at the pupillary margin.

In the ciliary body the loose tissue on the inner aspect of the muscle, between it and the epithelium, is fairly deeply infiltrated but not thickened, and the same description applies to the ciliary processes; this infiltration is in direct continuity with that of the iris root. On the outer aspect of the ciliary muscle, and not connected with the infiltration just mentioned, there is a dense infiltration forming a thick layer of lymphocytes which separates the ciliary muscle and pars plana from the sclera, and is much more marked below than above (Pl. IX, fig. 5). The muscle and the pars plana are themselves comparatively little invaded. In places the fibres of the ciliary muscles are

PLATE IX.

Illustrates Mr. George Coats's paper on Hyperplasia, with Colloid and Amyloid Degeneration, of the Episcleral and Circumdural Fibrous Tissue (p. 257).

Fig. 5.—Lymphocytal infiltration in the sub-ciliary and sub-choroidal space. A separate infiltration is present about the corneo-iridic angle and in the ciliary processes.

Fig. 6.—Amyloid degeneration of the ciliary muscle.

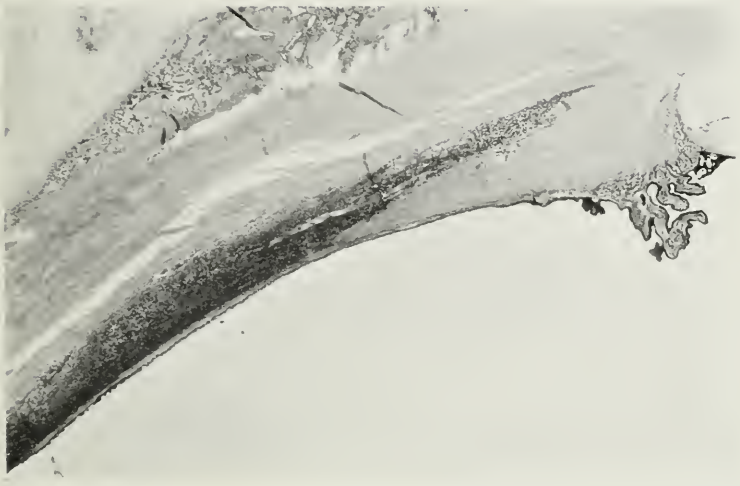

Fig. 5.

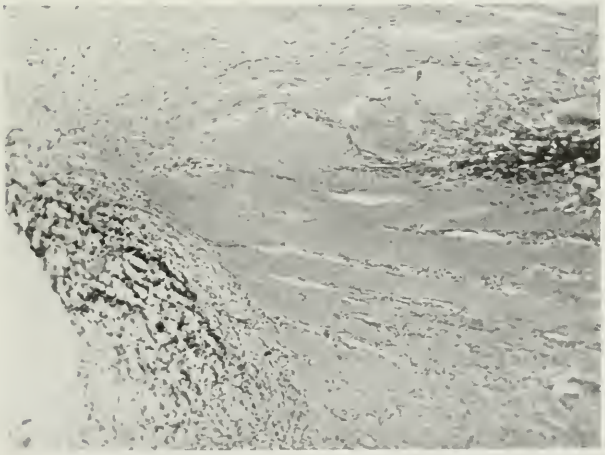

Fig. 6.

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approximately normal; elsewhere, however, especially near their anterior attachment on the lower side they have a swollen, homogeneous appearance, with an apparent diminution in the number of nuclei (Pl. IX, fig. 6). It is evident that the muscle has undergone a process of degeneration similar to that which affects the episcleral trabeculæ. Some of the fibrous strands crossing between the sclera and the ciliary body show the same change.

Continuous with the infiltration beneath the ciliary body there is a similar dense infiltration which occupies all the layers of the choroid except the chorio-capillaris. The infiltration is deeper below than above, but extends without interruption as far back as the nerve entrance. It receives accessions where the vortex veins and ciliary vessels and nerves perforate, but is not at all patchy. This, indeed, is the character of the intraocular infiltration generally—a uniform layer, causing a minimum of displacement of normal structures, with no formation anywhere of nodules or tumour masses, and with no tendency to transgress the inner limits of the choroid or ciliary body.

Other changes may be summarily dismissed. There is a small, partially healed corneal ulcer near the periphery on the nasal side. The lens is normal. The retina shows only some atrophy of its nerve fibre layer. There is no true glaucomatous cupping—the lamina cribrosa is not bowed backwards. The appearances observed during life were, no doubt, due in part to atrophy combined with a large physiological cup, partly to thickening of the choroid at the edge of the nerves entrance, which would increase the depth of the excavation. The nerve shows a moderate degree of atrophy. There is no trace of the characteristic homogeneous change in its trabeculæ or in the pia or arachnoid. The thickened dura does not encroach on the intervaginal space. The central vessels are free from amyloid degeneration or other pathological alteration. No amyloid bodies or corpora arenacea are noted in the nerve or its sheath.

Remarks.

The fundamental pathological lesion in this case was evidently a very slowly progressive overgrowth of the fibrous tissue immediately surrounding the globe and dural sheath of the nerve. The degenerative changes were manifestly secondary; the significance of the lymphocytal infiltration will be discussed presently. According to the clinical history the growth commenced in the conjunctiva on the nasal side, but inasmuch as proptosis was an early symptom it is probable that the involvement of the orbit was due not merely to a spread of the process backwards, but rather to an independent and nearly contemporaneous participation of the dural and circumdural trabeculæ. External signs of inflammation may been present in the earliest stages, but were never observed while the patient was under medical supervision. The loss of vision and optic atrophy were probably due to interference with the blood-supply of the nerve. There was no evidence of direct pressure, and in spite of the clinical appearance of cupping it is probable that the intraocular tension was never raised; the cup was not a true glaucomatous excavation, and although the corneo-iridic angle was in places stuffed with lymphocytes there was probably little interference with the drainage of fluids; at an early period, at least, the apparent cupping co-existed with a full field.

At first sight the association of amyloid degeneration of the ciliary *muscle* with a similar condition of the extra-ocular fibrous tissues may appear peculiar. It should be remembered, however, that the muscular coats of the ciliary arteries also showed the same change.

The ætiological indications were very uncertain. The history of a syphilitic infection was vague, the microscopical characters of the growth presented little analogy with those of syphilis, and anti-syphilitic remedies were totally inefficacious. The injury was of very old standing, and was followed by a long period without symptoms.

Exophthalmic goitre with unilateral proptosis and with an unusual degree of induration of the orbital tissues seems to be excluded by the absence of other symptoms, by the stationary character of the goitre, and by the fact that indurative changes of anything like this degree, especially in the anterior episcleral tissues, have never been described in that affection.

The condition which is most nearly related to the present is undoubtedly "amyloid degeneration of the conjunctiva." This disease was first described by v. Oettingen and Kyber in 1871, but the authoritative source of our information is the pathological researches of Leber (*Arch. f. Ophth.*, xix, i, p. 163, 1873, and xxv, i, p. 257, 1879) and the clinical analysis of thirty cases by Kubli (*Arch. f. Augenh.*, x, p. 578, 1881). In English literature the work of Herbert (*Trans. Ophth. Soc.*, xxii, p. 261, 1902; *Roy. Lond. Ophth. Hosp. Rep.*, xvi, p. 135, 1905), should be mentioned.

In the conjunctiva amyloid degeneration is a purely local affection, not dependent upon chronic tuberculosis, syphilis, etc., and not associated with similar changes elsewhere. Its supposed relationship with trachoma has been disproved by the statistics of Kubli. Inflammatory symptoms have occasionally been described in early stages, but usually the onset is perfectly insidious, and subsequent progress very slow. Individual cases, however, vary considerably in this respect. It most commonly begins in the fornix and spreads thence to the neighbouring ocular and tarsal conjunctiva; a primary affection of the bulbar conjunctiva is not unknown. Among thirty cases collected by Kubli the disease was unilateral in twenty-one, bilateral in nine.

The earliest signs are a little thickening of the lid, with padding out of the hollows beneath the orbital margin, and slight ptosis. As regards the conjunctival lesion, Kubli distinguishes four clinical stages, corresponding with a four-fold sub-division of the pathological changes instituted by Raehlmann.

(1) Stage of simple proliferation of the sub-conjunctival lymphoid tissue. The conjunctiva is the seat of a smooth, diffuse thickening, which is of dense but elastic consistence, and, according to the degree of its vascularity, of light yellow, yellow-red, or red-brown colour. If the bulbar conjunctiva is invaded a thickened ring may surround and overlap the cornea, rendering inspection difficult and even impeding closure of the lids. Very rarely the overlying skin is unduly vascular, discoloured or œdematous.

(2) Stage of "hyalin" degeneration. The growth takes on an exceedingly characteristic diaphanous or waxy appearance. It is of cartilaginous consistence—harder than in the first stage—and yellow-brown or brown-red.

(3) Stage of "amyloid" degeneration. The diaphanous appearance is still present, and the growth is yellow, yellow-red or dirty brown-red according to its vascularity. It may be hard, in which case it is inelastic and brittle, and is liable to crack and split when handled; or it may be of gelatinous, yielding consistence. Spontaneous hæmorrhages are not infrequent.

(4) Stage of calcification and ossification. The appearances remain the same, but white or grey-white bodies are formed at various depths in the growth.

From the pathological standpoint Leber insists that the condition is not merely a degeneration of pre-existing structures, but that there is also a true proliferation, with subsequent degeneration, of the collagenous fibrous tissue. He describes isolated, round or tuberculated "amyloid bodies," as well as degenerate fibrous tissue bundles, but believes that there is no essential distinction between the two. It has been suggested indeed by v. Hippel that the amyloid "bodies" are merely pieces of amyloid fibrous tissue bundles broken off in the process of teasing the specimens, and in view of the brittle nature of amyloid, and of the fact that isolated amyloid bodies are not prominent in sections, the suggestion seems not

improbable. Whether this be so or not, both the amyloid bodies and the degenerate bundles are found to be clothed by a cellular sheath, in association with which giant cells frequently lie on the surface or within bays of the diseased tissues. The deeper subconjunctival tissues are usually more profoundly affected than the superficial, and the conjunctival vessels show the characteristic changes, but in a degree which varies in individual instances. Calcification and ossification were first described by v. Hippel (*Arch. f. Ophth.*, xxv, ii, p. 1, 1879). A considerable lymphocytal infiltration is always present among the amyloid masses.

The above *resumé* sufficiently establishes the close similarity, indeed the essential identity, of "amyloid degeneration of the conjunctiva" and the condition now under consideration. As in that affection, so here, the disease was of a purely local nature, occurring on one side, in a man otherwise perfectly healthy. Progress was very insidious, signs of inflammation were nearly or quite in abeyance, the growth had a "fresh semitranslucent appearance," and a hard, cartilaginous consistence. Microscopically, the evidence of fibrous tissue proliferation; the homogeneous alteration of the proliferated bundles; the amyloid staining reactions where the change was most advanced; the histological details, such as the tuberculated outline of the bundles in transverse section; the cellular sheaths; the giant-cells; the calcification and ossification; and the inter-trabecular lymphocytal infiltration—all these points leave no room for doubt that the two conditions were one and the same.

It is with respect to the distribution of the changes that the present case is unique. So far as I know there is no other record of involvement of the orbital tissues, with consequent proptosis and limitation of movement of the globe. The freedom of the tarsal conjunctiva from all invasion after so long a period of years is also a somewhat rare feature.

The histological appearances fully justify the contention of Raehlmann that a stage of "hyalin" degeneration precedes the development of true amyloid substance. The amyloid reaction was indeed almost universal in the anterior episcleral tissues, but posteriorly many of the fibrous bundles were simply swollen and homogeneous without any very characteristic staining reactions, while the coloration typical of amyloid substance was unmistakable only where the homogeneous change was most pronounced. While confirming Raehlmann's observations, however, it is permissible, in agreement with Herbert (*Trans. Ophth. Soc.*, xxii, p. 261, 1902), to question the propriety of his use of the term "hyalin." In ophthalmic as well as in dermatological literature the name "hyalin" is already appropriated to the refractile globular bodies, staining brightly with eosin, which are characteristically present in some forms of superficial corneal degeneration (band opacity, etc.). From this type of degeneration the present condition differs widely, at least in its histological appearances—one has the aspect of an extraneous deposit in the tissues; the other of a transformation of the tissues themselves—and without entering into the vexed question of the precise inter-relationships of these homogeneous degenerations, it seems preferable in the present instance to follow the example of Herbert, and to speak simply of a "colloid" change, using that designation in a purely descriptive, and not in a chemically specific, sense. For this use of the word "colloid" Herbert adduces the general usage of dermatologists and the high authority of Unna.

In conclusion a few words may be devoted to an explanation of the present case alternative to that which has been adopted in this paper. Most authors have taken it for granted that amyloid degeneration is primarily a disease of the collagenous fibrous tissue. Raehlmann, on the other hand, following Recklinghausen's description of similar lesions in lymphatic glands, believes that the earliest change is a proliferation of the lymphoid tissue, and that the lymphocytes are themselves subject to the amyloid

change. He points out that in the conjunctiva amyloid degeneration usually begins in the fornix, where lymphoid tissue is most abundant. He compares the condition, therefore, with a tumour of a lymphatic gland—a lymphoma. In a case of "prolifération lymphomateuse" of the conjunctiva, v. Duyse (*Arch. d'Opht.*, vol. xxv, p. 402, 1905) has described "hyalin" degeneration of the stroma—not, however, on the massive scale which is characteristic of amyloid degeneration of that membrane. Amyloid degeneration has also been described by Prout and Bull (*Arch. f. Augenh.*, vol. viii, p. 221, 1879) in a round-celled sarcoma * of the conjunctiva.

Is it possible, then, that the condition in the present case was essentially a lymphatic new growth, whether inflammatory hyperplasia or true lymphoma, the colloid and amyloid change being merely secondary? On clinical grounds there might be urged against this view the protracted duration of the case and the unimpaired health of the patient, lymphoma of the conjunctiva, when not an insignificant overgrowth of a follicle,† being a condition which is usually associated with leucæmia or pseudo-leucæmia. But the pathological evidence is still more conclusive. The lymphocytal infiltration was greatest anteriorly precisely where an inflammatory reaction might be expected owing to exposure of the growth in the palpebral fissure. Posteriorly it was much less dense, and areas occurred in which the fibrous proliferation and degeneration were present with little or no lymphocytal infiltration. It is manifest, therefore, that the proliferation and degeneration must have been primary and the infiltration secondary.

* Their diagnosis appears to be open to much question. The tumour was smooth and flat, and did not grow during a period of observation of six weeks. Most probably the case was one of ordinary amyloid degeneration, the "sarcoma" cells being simply the lymphocytal infiltration which always accompanies that condition.

† For examples of this condition, see Coats (*Arch. of Ophth.*, May, 1915).

Summary.

To summarise, therefore, the case was one of slowly progressive hypertrophy of the episcleral and circumdural fibrous tissues, with a homogeneous type of degeneration in the proliferated bundles which, in its more advanced manifestations, gave the staining reactions characteristic of amyloid disease. The underlying cause of this hypertrophy was obscure; the ordinary sources of chronic inflammation appeared to be absent, and the histological changes did not accord well with the usual types of that condition; the distribution of the lymphocytal infiltration, and more especially its absence in some areas posteriorly where the fibrous tissue changes were characteristic, would seem to indicate that the infiltration was a phenomenon of secondary significance, due probably in part to exposure of the growth in the palpebral fissure, in part perhaps to a tissue reaction brought about under the stimulus of certain products of degeneration.

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2. "*Blue sclerotics.*"

By Sydney Stephenson.

(With Plate VI, fig. 2.)

The three cases of so-called "blue sclerotics" now shown belong to the family described by me in the *Ophthalmoscope* of May, 1910. Twenty-one members of the family in question, belonging to four generations, were affected by the anomaly.

A mother and two of her daughters are now exhibited. It may be said that this woman, herself the subject of "blue sclerotics," has had six children—four girls and two boys. Of the girls, the second and third are now shown. The first girl, æt. 27 years, has "blue sclerotics," and fractured her leg when three years of age. Her height is 4 ft. 10½ in. The fourth girl, æt. 16 years, has normal sclerotics, a height of 5 ft., and has never broken any bones. As regards the boys, the elder, æt. 18 years, has "blue sclerotics," a height of 5 ft. ½ in., and

has never fractured any bone. The eyes of the younger, æt. 13 years, are not affected, but, curiously enough, he broke his left leg when three years of age. His height is 4 ft. 6 in.

The mother, æt. 50 years, had dark-blue sclerotics, almost of a leaden hue. Her height is 4 ft. 10 in. She has broken no bones. The elder of the two daughters, now shown, æt. 22 years, has azure sclerotics. She has sprained both ankles on several occasions. Her height is 4 ft. 11 in. The sclerotics of the younger daughter, æt. 20 years, resemble those of her sister. Her height is 4 ft. 11¼ in. At two years of age she fractured one thigh by tumbling out of bed, and at four years of age she broke one leg below the knee by falling whilst running across the kitchen floor. She has sprained her ankle several times. As the mother, when recounting her history, graphically remarked, she "got quite used to picking her up broken."

It is now more or less widely recognised that "blue sclerotics" form merely part of a symptom-complex, the other components of which include small stature and a peculiar tendency to almost spontaneous fractures, dislocations, and sprains. There appears to be a deficiency or weakness of the fibrous tissues throughout the body, not excluding the sclera. The condition is known to be markedly hereditary, and to descend through affected members of such a family alone. Transmission to males through unaffected females does not appear to take place. The connection between "blue sclerotics" and brittle bones was first pointed out in 1900 by A. Eddowes (*Brit. Med. Journ.*, July 28th, 1900). He also drew attention to the hereditary nature of the scleral condition.

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VIII. DISEASES OF THE UVEAL TRACT.

1. *A contribution to the study of the microscopical anatomy of ocular syphilis, illustrated by three cases.*

By R. Affleck Greeves.

(With Pls. X, XI, XII, and XIII, fig. 8.)

The material necessary for the exhaustive study of syphilitic disease of the eye from the clinical standpoint, in all stages of the disease, is practically unlimited, and good use has been made of it. But owing to the fact that purely syphilitic eye affections are usually arrested by the specific remedies, and that enucleation of the diseased eye is infrequently called for, opportunities of investigating the histological changes which occur in the tissues of an eye thus affected are but rarely met with.

I have been able recently to examine microscopically three eyes, each of which was removed from a patient who had been infected with syphilis; in one of these the disease was congenital, and in the other two the diagnosis was confirmed by a Wassermann reaction. My observations on these three eyes form the basis of this communication.

Case 1.—The first specimen I wish to show is a section through an eye removed from a man, æt. 35 years, who had suffered from interstitial keratitis in both eyes when a boy; there was every reason to regard the keratitis as of syphilitic origin, but I could, unfortunately, not ascertain the exact age at which the active inflammation had occurred.

The patient gave a rather indefinite history of the eye

having been injured subsequently, and when seen at the Royal Westminster Ophthalmic Hospital by Mr. Brewerton, to whom I am indebted for permission to use the case, the eye was blind and unsightly, the lens opaque, the tension low, and the patient complained of intermittent attacks of pain.

The eye was enucleated, fixed in Zenker's fluid, divided horizontally, and mounted in celloidin.

Description of Microscopical Sections.

The sections showed an atrophic iris bound down to a degenerate and calcareous lens, and a completely detached retina. There were no signs of active inflammation.

The point of interest is the condition of the cornea (Pl. X, fig. 1). At the periphery, in the immediate neighbourhood of the limbus, there are numerous new vessels, which contain blood, throughout the thickness of the substantia propria. But in the more central part of the cornea the anterior half of the substantia propria is devoid of vessels, while in the posterior half, and more especially in the posterior third, there are rings of endothelial cells, flattened in the plane of the corneal lamellæ, so that their anterior and posterior walls are in contact, and consequently with no lumina or blood contents. The rings consist solely of endothelium and have no fibrous tissue element in their composition. I think it justifiable to assume that these are the old vascular channels which carried blood during the active stage of the inflammatory process in the cornea, and from which the blood contents have since disappeared. The fact that these blood-channels remain as empty endothelial tubes with potential lumina, instead of becoming sclerosed into solid cords, is especially interesting in view of the well-known clinical observation that a new vascularised patch, containing blood, may appear in a surprisingly short time in a cornea which has once been affected by interstitial keratitis, should a fresh, even slight, local irritation or inflammation

PLATE X.

Illustrates Mr. R. Affleck Greeves's paper on A Contribution to the Study of the Microscopical Anatomy of Ocular Syphilis, illustrated by Three Cases (p. 277).

Fig. 1.—Section through cornea from Case 1 (old interstitial keratitis) showing remains of blood-vessels. Anterior surface of cornea uppermost. The cystic condition of the epithelium is an artefact.

Fig. 2.—Section through cornea and iris of Case 2 showing a general view of the granulomatous tumour in the iris root.

Fig. 1.

Fig. 2.

Adlard & Son, Imp.

occur. It would seem that the old, remaining, empty, blood-channels may readily refill with blood under such conditions.

The posterior corneal endothelium shows evidence of having suffered from the inflammatory process as well as the substantia propria; the cells have lost their normal regularity, and their nuclei have become spindle-shaped.

Case 2.—My second specimen is a section through an eye in which a localised granuloma was present in the iris. The case, for the use of which I am indebted to Mr. Bishop Harman, presented some very unusual clinical features.

A married woman, æt. 32 years, came up to the West London Hospital, complaining of headaches. She had been suffering from neurasthenia. The eyes were examined, and the condition of the iris then noted. The patient was unaware of any change in the eye. In the left eye, at the root of the iris, was a small dark brown lump, the size of a millet seed, situated at the position of eight on the clock face. Its base conformed to the course of the root of the iris, while the other edge projected towards the pupil in a crescent of 2 mm. radius. Its surface was raised, so that it partly filled the receding angle, which, however, it did not appear to block. The iris was brownish, green at its root, and a clearer, richer brown round the pupil, so that the lump showed up as a dark brown spot. In the ordinary state of the pupil the opening was a perfect circle, but there was a deep crease in the iris on the pupillary side of the lump, as though the mass pushed the iris inwards. When the pupil was dilated with a mydriatic the dilatation was incomplete and left a flattened edge in the immediate region of the mass. Examined with the corneal microscope the mass was seen to be richly pigmented and finely roughened all over; no vessels were seen.

The eye was again examined a week later, and little or no change had occurred in the appearance of the mass, but deposits had appeared on the back of the cornea,

some of which were of moderate size, and a good many of minute size. There was a suggestion of fine dust in the vitreous in the region of the mass, but no sign of a bulge on the inner surface of the iris could be made out.

Transillumination gave no help. The fundus was normal.

At the limbus, immediately over against the mass, there was a cluster of dilated minute episcleral vessels; elsewhere the limbus was normal. The right eye was normal.

R.V. $\frac{6}{6}$. L.V. $\frac{6}{9}$ some.

The above history and clinical description is taken from the *Proceedings of the Royal Society of Medicine*, Ophthalmological Section, before which Society the case was shown by Mr. Bishop Harman. A suggestion was then made that the case might be one of simple pigmented cyst of the ciliary body, but the general consensus of opinion was in favour of the diagnosis of a melanotic malignant growth of the iris, and of immediate enucleation of the eye.

Excision was performed, and the eye was placed in Zenker's fluid, divided, and mounted in celloidin for microscopical examination. The sections revealed the fact that the mass at the root of the iris was an inflammatory swelling, and following on this discovery a general examination of the patient was made, as well as various diagnostic tests. No evidence of tubercle was found, but the Wassermann test gave a positive result. This test was then applied to the patient's four children, three of whom reacted positively.

On these grounds, together with the evidence of the microscopical appearances of the granuloma, it is reasonable to regard the case as one of a late syphilitic manifestation in the iris.

Description of Microscopical Sections through the Pupillary Region, including the Granuloma.

The peripheral half of the iris on the inner side is much thickened, and consists of a mass of granulation tissue;

PLATE XI.

Illustrates Mr. R. Affleck Greeves's paper on A Contribution to the Study of the Microscopical Anatomy of Ocular Syphilis, illustrated by Three Cases (p. 277).

Fig. 3.—High power view of section through the granuloma in Case 2 showing the radiating pigment cells. The areas of lymphocytes and plasma cells and epithelioid cells are also seen. Some of the pigment epithelium of the iris appears in the lower right hand corner.

Fig. 4.—Section through one of the smaller granulomata in the posterior part of the iris of Case 2. The anterior pigment layer is beginning to proliferate into the tumour.

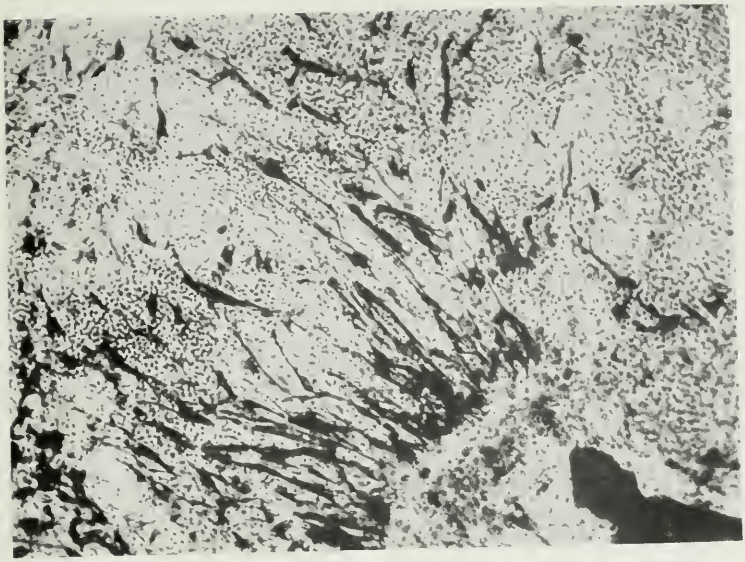

Fig. 3.

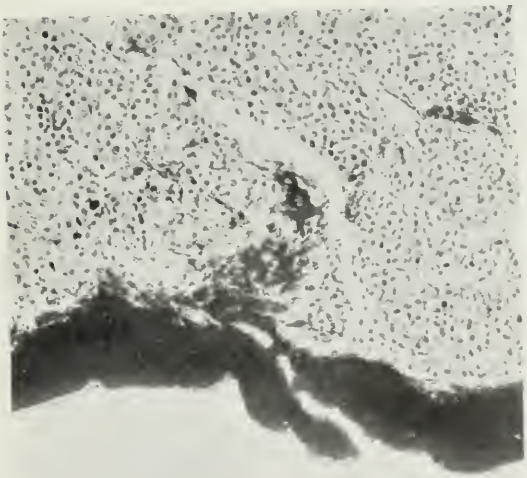

Fig. 4.

Adlard & Son, Impr.

this constitutes the tumour seen clinically (Pl. X, fig. 2). The granulomatous mass has evidently arisen in the iris itself, apparently a little to the pupillary side of the iris root, and has therefore no primary connection with the ciliary body. The granulation tissue is arranged in nodules, which are easily distinguishable in sections stained with hæmatoxylin and eosin; in these sections the tumour is subdivided into dark blue areas consisting of plasma cells and lymphocytes, the nuclei of which have taken the stain deeply, and into light reddish areas which consist of large epithelioid cells, the cell bodies of which have stained with eosin, and the large irregular nuclei of which have taken the blue stain but faintly. This appearance is strikingly reminiscent of that seen in the choroid in cases of sympathetic ophthalmitis. These epithelioid cells are arranged in a circular manner in the centres of some of the nodules, so as collectively to resemble a multinuclear giant cell, but no true giant cells are present (Pl. XI, fig. 3).

On the anterior surface of the granuloma is a layer of black, heavily pigmented cells, and the tumour is intersected by large, elongated, heavily pigmented cells, which radiate forwards, like the spokes of a fan, from a small area in the centre of the posterior surface of the tumour. The black abundant pigment contained in these intersecting cells as well as in the cells of the anterior layer, contrasts markedly with the delicate light brown, rather scanty pigment of the chromatophores of the iris stroma, and strongly resembles the pigment of the posterior epithelial layers. The normal structure of the iris stroma has been completely replaced by granulation tissue in the situation of the tumour. The posterior surface of the granuloma is lined by the original two layers of pigment epithelium of the iris, except at a spot opposite the centre of the tumour where only the posterior layer is present, not in apposition with the tumour, but separated from it by an albuminous coagulum and thus forming the posterior wall of a cyst. Besides the coagulum, this cyst contains a few swollen "ghost

cells." The anterior layer of pigment epithelium appears to have proliferated in this situation and by this means to have given rise to the radiating elongated pigment-bearing cells which intersect the tumour, as well as to the heavily pigmented cells which clothe its anterior surface.

The evidence in favour of the view that these pigmented cells are epithelial in origin, derived from the anterior pigment epithelial layer and not from the chromatophores of the stroma, is as follows :

(1) The manner in which the intersecting cells radiate from the position of the anterior pigment layer on the posterior surface. If derived from the chromatophores of the stroma it is not easy to see how this could occur, as there is normally practically no stroma pigment in the posterior part of the iris, the chromatophores being aggregated in the anterior layers.

(2) When examined by very high powers, the pigment granules in these pigmented cells in the granuloma resemble exactly the pigment granules of the epithelium, *i. e.* they are very dark in colour, large, and coarse, and regular, sometimes even rod-shaped, whereas stroma pigment consists normally of very fine, dust-like, irregular, granules.

(3) The anterior layer of the pigment epithelium of the iris consists normally of elongated spindle-shaped cells, and would presumably give rise to the same type of cells on proliferation.

The cells on the anterior surface of the granuloma are of the same nature as the large intersecting cells. This is obvious when both are examined by high powers.

The seat of origin of the granuloma appears to have been in the posterior zone of the iris stroma, close against the anterior layer of pigment epithelium, and this layer apparently proliferated into the granulation tissue as the granuloma increased in size, invading and destroying the iris stroma as it advanced forwards and laterally. The proliferated epithelial pigment contained in the tumour

ultimately became exposed on the anterior surface of the tumour, giving rise to the dark pigmented appearance which was the cause of the diagnosis of melanotic sarcoma of the iris having been made. This view as to the seat of origin of the granuloma is supported by the presence of smaller inflammatory nodules in other parts of the iris, these nodules being invariably situated in the posterior zone of the stroma, against the pigment epithelium. Into some of these the anterior layer of pigment epithelium is beginning to proliferate (Pl. XI, fig. 4).

Another point worthy of notice with regard to these smaller nodules, is the fact that they are to be found only in the pupillary and peripheral regions of the iris and never in the intermediate part. It has been stated by Benoit that syphilitic granulomata of the iris occur only in these situations, and also that those in the early stage of the disease are more commonly found in the pupillary region, while those in the later stages are usually in the iris root.

No trace of degeneration can be found in the tumour. It is poor in blood-vessels, but such as are present are wide capillary channels, obviously of new formation. The smaller nodules in the iris already referred to consist mainly of plasma cells and lymphocytes, and in some of them a few epithelioid cells are to be found, some also show an indication of commencing proliferation of the anterior layer of epithelial pigment cells. Isolated plasma cells are thinly scattered throughout the iris stroma. Only in the vessels in the immediate neighbourhood of the granuloma can any trace of endothelial proliferation be seen, and this is never so great as to occlude the affected vessels. That part of the ciliary body immediately adjacent to the tumour shows some lymphocytic and plasma cell infiltration, but the remainder is free of any but the slightest signs of inflammation.

The tumour fills the corresponding small section of the angle of the anterior chamber, and the spaces of Fontana are filled with leucocytes. The rest of the angle is open,

and, except for the presence of a few leucocytes, appears normal. None of the sections examined showed any leucocytic deposits on the cornea, but probably sections taken through the lower part of the cornea would have done so. No posterior synechiæ are present, and the rest of the eye is normal. Sections were stained for tubercle bacilli and spirochætes, but neither were found.

If the term "gumma" be used to express a granulomatous tumour arising in the tertiary stage of syphilis—whether necrotic changes be present or not—then the tumour under discussion may be termed a "gumma of the iris." But on looking into the literature it will be found that these solitary iris tumours are not confined to the later stages of the disease, but may occur at any time, from the early secondary stage onwards. They undoubtedly constitute a rare manifestation of syphilitic disease, while *multiple* inflammatory nodules in the iris very commonly occur during the course of a syphilitic iritis, which is essentially associated with an early stage of syphilis. These multiple nodules appear in the pupillary zone (Benoit), are often accompanied by blood-vessels of new formation, and are always of a transient nature, nodules and blood-vessels disappearing and leaving no trace.

The question of the nature of these different syphilitic nodules in the iris was first discussed by Widder in 1881, who came to the conclusion that two essentially different kinds may occur:

(1) The multiple variety, which are of common occurrence in the early stage, are small in size, and richly vascular (iritis papulosa).

(2) The solitary variety, associated only with a late stage of syphilis, poor in blood-vessels, and liable to degeneration (iritis gummosa).

The first group Widder compared to the condylomata, or papules, of secondary syphilis, and the second he regarded as true gummata.

But Widder's conclusions are not supported by sub-

sequently recorded cases, for it has been shown that the "solitary" nodules may occur at any stage of the disease and may be richly vascularised; and besides this, there is good evidence that each of these so-called "solitary" tumonrs is really only an exceptionally large member of a group of multiple nodules. There are on record three histological descriptions of "solitary" syphilitic tumours of the iris (Colberg, Benoit, Rumschewitz). Of these, two occurred in the secondary stage, one two and a half and the other three months after infection, and one, as in the case at present under discussion, in a late stage, *i. e.* eleven years after infection. Again, one of the early tumours was richly vascular, and showed no tendency to necrosis, while in the other there was a poor blood supply, marked endarteritis, and extensive necrosis. The recorded tertiary tumour showed no new vessels, there was marked endarteritis in those pre-existing, and necrotic changes were beginning in the tumour, while in the present case, on the other hand, some new blood-vessels were present, endarteritis was not a feature, and no necrosis was seen. In two of these cases multiple small inflammatory nodules were present in the iris, besides the large granulomatous tumour (Benoit, Rumschewitz); in the third the tumour was removed by iridectomy, and consequently the condition of the rest of the iris was not seen (Colberg).

According to Fuchs' researches it would seem that all types of syphilitic iritis tend to have a nodular form. He had the opportunity of examining a case of secondary syphilitic iritis in which, although no nodules had been seen clinically, microscopically the inflammation was of a distinctly nodular type, giant cells and epithelioid cells being present in the nodules. It is obvious, then, that it is impossible to classify separately these "solitary" iris tumours, either according to the period of the disease in which they occur or according to their vascularity or tendency to necrose. Again, in the present case the iris nodule, although solitary clinically, was not so histologi-

cally, for several small discrete granulomatous tumours were found in the iris on microscopical examination.

To conclude, it appears that inflammatory changes in the iris may occur in all stages of syphilitic disease after the primary stage, that the inflammation always tends to a nodular type histologically, that multiple nodules of a transient nature, richly vascular, are often visible clinically in the secondary stage, while larger nodules may occur in all stages; the latter may or may not tend to necrose according to the presence or absence of endarteritis, which is a variable quantity and independent of the stage of the disease. All these nodules, early and late, have a similar microscopical structure, and the cases described supply evidence that the nodules are always multiple, even in the later stages of the disease, and even though only one may be of sufficient size to appear clinically. The terms condyloma and papule seem particularly unhappy when applied to swellings on the iris, because they imply swellings or patches on epithelial surfaces, the epithelium of which takes a definite part in their formation.

The most remarkable clinical feature of the present case was the dark pigmentation of the anterior surface of the tumour. I have not been able to find any other case, either clinically or pathologically reported, in which this peculiarity was present.

Large syphilitic granulomata occur much more commonly in the ciliary body than in the iris, and many of these so-called "gummata of the ciliary body" have been subjected to microscopical examination. A glance into the literature of these tumours shows that they too may occur in the secondary and tertiary stages of syphilis, and that they may or may not show a tendency to necrosis, this depending, not on the stage of the disease, but on the poorness or richness of the blood-supply of the tumour. Tooke, in 1903, collected these cases and tabulated them. Among them are several cases of gummata arising primarily in the ciliary body and extending secondarily into the iris. These I have not

PLATE XII.

Illustrates Mr. R. Affleck Greeves's paper on A Contribution to the Study of the Microscopical Anatomy of Ocular Syphilis, illustrated by Three Cases (p. 277).

Fig. 5.—The patch of scleritis in Case 3 showing thinning of the sclera with vascularisation. A large granulomatous nodule, with a giant cell in the centre, in seen on the right.

Fig. 6.—A small isolated granulomatous nodule, containing a giant cell, on the surface of the pectinate ligament (Case 3). Note the great thickening of the fibres of the ligament.

Fig. 7.—Section to show two nodules in the iris in Case 3. One is close to the root, and the other near the pupil at the peripheral end of the sphincter muscle.

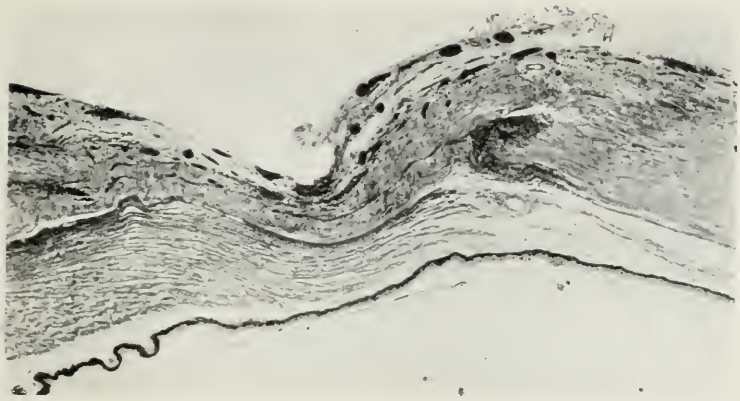

Fig. 5.

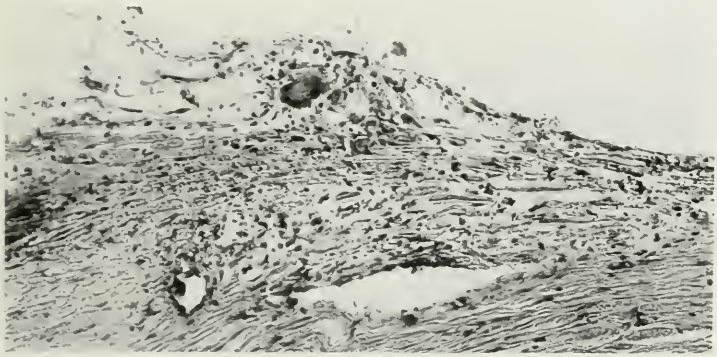

Fig. 6.

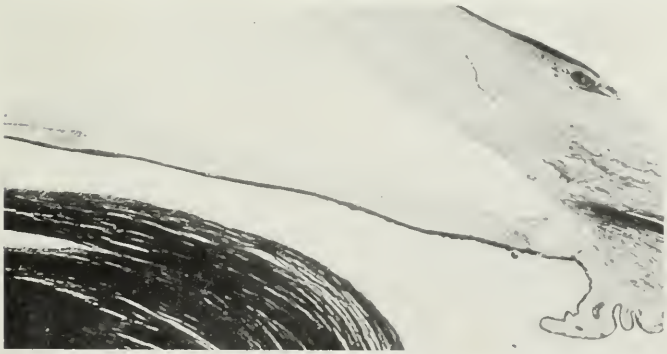

Fig. 7.

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included in the cases I have quoted of primary granuloma of the iris.

Case 3.—The next case I wish to describe is one of syphilitic disease of the sclera and choroid. I am indebted to Mr. Treacher Collins for this case; the patient was under his care at the Royal London Ophthalmic Hospital. The history is as follows:

A man, æt. 60 years, has had defective sight in the eye for fifteen years. There is a history of venereal disease twenty-five years ago, and the Wassermann reaction is positive. The right eye has been painful six months.

The left eye is slightly myopic, otherwise it is normal.

The right eye shows much ciliary injection.

A raised swelling is present on the sclera just outside the limbus; it has a dark spot inthe centre.

Cornea bright, no k. p. A.C. moderate. Iris bright, vascularised posterior synechiæ below and to the inner side. No pupil reaction.

Lens opaque, no fundus reflex.

V. = hand shadows. Projection faulty. T.n.

A breaking down gumma of the ciliary body was diagnosed, and as the right eye was painful and useless it was excised, fixed in Zenker's fluid, and prepared for microscopical examination.

When the eyeball was divided the retina was found to be *in situ* (except anteriorly on the outer side), and both retina and choroid were extremely atrophic.

Description of Microscopical Sections (Horizontal).

Corneal epithelium normal. Discrete deposits of hyaline substance present in Bowman's membrane and in the anterior layers of substantia propria. These deposits are much denser at the periphery than in the centre.

On the outer side, in the mid-horizontal plane, outside the area of attachment of the ciliary muscle, there is an area over which the sclera is much thinned (Pl. XII, fig. 5).

This area is traversed by many wide vessels of new formation, which run between the planes of the sclerotic bundles. Between these bundles there are also some scattered nodules of infiltration, consisting of lymphocytes and epithelioid cells, with an occasional giant cell. There is no sign of necrosis in the nodules. The underlying ciliary muscle is unaffected, apart from the presence of a few new vessels between its bundles. The anterior chamber is of good depth, and the angle of the anterior chamber is well open. The fibres of the ligamentum pectinatum is markedly thickened, each fibre showing a fibrillar core, often slightly pigmented, with a thick homogeneous outer lining. Here and there, on the surface of the pectinate ligament and extending between its superficial fibres, are nodules of inflammatory cells containing lymphocytes, epithelioid cells, and sometimes a giant cell (Pl. XII, fig. 6). Some of these do not seem to contain any blood-vessels, while opposite some of these nodules the iris is adherent and supplies a blood-vessel to the nodule.

The iris stroma is atrophic, and devoid of inflammatory cells, except for the presence of some isolated nodules of epithelioid cells and lymphocytes, which are present in its posterior part, lying against the pigment epithelium in the ciliary and pupillary regions (Pl. XII, fig. 7). A wide posterior synechia is present below and continued on to the surface of the lens capsule as a pupillary membrane. No synechiæ are present above. The ciliary body shows no marked changes.

The choroid and retina, on the other hand, are much affected. To take the choroid first :

Signs of active inflammation are confined to an area just in front of the equator, where there is a series of nodules of inflammatory cells, consisting mainly of plasma cells and lymphocytes, but with a few epithelioid cells in the centre of the nodules. The densest part of each nodule is in the inner layers of the choroid. The outer layer of the choroid, including the large vessels, is well

PLATE XIII.

Fig. 8 illustrates Mr. R. Affleck Greeves's paper on A Contribution to the Study of the Microscopical Anatomy of Ocular Syphilis, illustrated by Three Cases (p. 277).

Fig. 8.—Shows a mass of fibrillar tissue between choroid and retina, on the surface of the intact membrane of Bruch. The dark areas in the mass are tubes of pigment epithelial cells.

Fig. 1 illustrates Dr. Gunjiro Komoto's paper on The Pathological Anatomy of the Retina and Choroid after Complete and Partial Removal of their respective Blood Supplies (p. 295).

Fig. 1.—Retina and choroid near the optic disc. The retina is reduced to a swollen necrotic mass. The choroid shows great pigment degeneration, the chromatophores being changed into round masses of pigment.

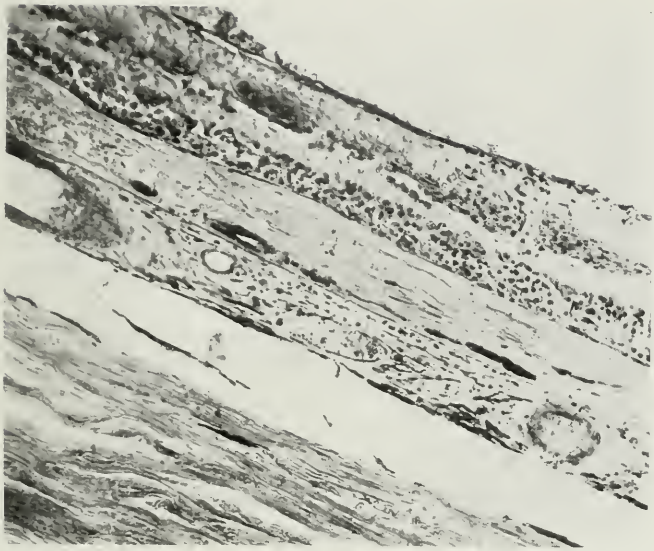

Fig. 8.

Fig. 1.

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preserved, except in that part of the choroid immediately surrounding the optic disc, where little of the original choroidal structure can be made out. Except in the area just mentioned, the choroidal pigment is fairly well preserved.

The inner layers of the choroid, on the other hand, are much altered. The chorio capillaris is only recognisable in isolated patches, and then only as a few scattered capillaries; in places there is a little new fibrous tissue present in the choroid, but not to any marked degree. The membrane of Bruch is for the most part intact, it is destroyed in the part immediately surrounding the optic disc, and is broken through just behind the ora serrata.

The retina is for the most part adherent to the choroid, it is extremely atrophic, and in many places no trace of the original arrangement of its layers can be made out. The rods and cones have entirely disappeared. There is detachment to the outer side anteriorly over this area; here the chorio capillaris is best preserved, and the pigment epithelium, though much changed, is still present on the membrane of Bruch. Large retinal cysts are present in the macular region; these cysts are filled with "ghost cells" containing pigment granules, and with granular *debris*.

Between the choroid and retina, binding the two coats together, are large plaques of dense fibrillated tissue, covering wide areas of the surface of Bruch's membrane (Pl. XIII, fig. 8). In the situations of these plaques, the pigment epithelium cannot be recognised as such, but embedded in the tissue are numerous tubules lined by pigmented cells; there are also some isolated spindle-shaped cells, many of which are pigmented. This tissue is quite free of blood-vessels except anteriorly, where Bruch's membrane is broken through, and posteriorly, round the optic disc, where this membrane is destroyed. In the latter situation the tissue is ossified in places, and the formation of bone seems to have taken place both in and on the surface of the choroid, but owing to the destruction of Bruch's

membrane in this part it is difficult to say which tissue belongs to the original choroid. In the isolated situations where the chorio capillaris is still preserved, there are a few pigment epithelial cells in their original position, and the retina and choroid are here not adherent. The retinal vessels are much thickened, both as regards intima and adventitia; this applies both to arteries and veins, which are impossible to distinguish from each other. The smaller vessels have disappeared. There is no pigmentation of the retina in any part.

The choroidal vessels are not thickened, nor indeed are any of the vessels in the whole uveal tract. The optic nerve is exceedingly atrophic.

In connection with the above description the following points are chiefly of interest:

In the first place a good example of tertiary syphilitic scleritis is seen in this eye. The area affected appeared dark in the centre clinically, and the seat of the inflammation was therefore thought to be in the ciliary body, and the dark colour to be due to the presence of ciliary epithelial pigment in a breaking down gumma which was eroding the sclera. This colour was probably really due partly to the thinning of the sclera, so that the pigment of the ciliary body was seen through it, and partly to the extreme vascularity of the inflamed part, the excess of venous blood being in part responsible for the dark hue. Scleritis in any form is prone to produce thinning of the sclerotic, and syphilitic scleritis is no exception to the rule.

A second point of interest is the presence of small discrete granulomata on the surface of the ligamentum pectinatum. These nodules are peculiar in that they have arisen in an avascular structure, and although some have a blood-supply from the adherent iris, this is an adventitious one, and has most probably developed secondarily to the formation of the nodules. Blood-vessels are therefore not essential for the transport of the syphilitic virus in the eye.

The nodules present in the iris are also interesting in

view of their situation close to one or other border, and, as in Case 2, in the extreme posterior layer of the iris stroma.

Again, in the choroid, the pathological process has affected the inner layers only, except in the area round the optic nerve, where the inflammatory process was sufficiently severe to involve all the layers. This observation agrees with those of other observers, of whom Nettleship and Baas may be quoted, both of whom had the opportunity of examining syphilitic eyes in a recent stage of inflammation. As regards the more marked severity of the inflammation in the posterior part of the choroid, Baas suggests that this is due to this region being the place of entry of the chief arteries supplying the choroid.

What is the nature of the fibrillar material which is present in large plaques between the retina and choroid? (Pl. XIII, fig. 8).

There are two possibilities with regard to its origin:

(1) It may be true fibrous tissue derived from the fibroblasts of the choroid or retinal vessels.

(2) It may be derived from the pigment epithelium, by a process of proliferation and metaplasia.

Both these views with regard to the origin of the tissue found between retina and choroid in cases of choroiditis have found supporters, but, while the first is much the more commonly accepted one, it is difficult to regard it as true in the present case for the following reasons:

(*a*) Bruch's membrane is well preserved over the greater part of the choroid, and is broken through only in the extreme anterior and posterior parts. Now this membrane is impermeable to cellular elements.

(*b*) The tissue on the surface of Bruch's membrane is avascular, except at points just opposite to those spots where the membrane is broken through; in these situations a few blood-vessels can be seen. It is most unlikely that fibrous tissue should have spread over most of the inner surface of Bruch's membrane from these isolated spots without carrying some of its blood-vessels with it, in which case

it is probable that evidences of vascularisation would be present elsewhere.

(c) There is comparatively little new-formed fibrous tissue in the choroid itself. If the tissue on the surface of Bruch's membrane were derived from the choroid, and had, so to speak, overflowed from there through the comparatively small openings in Bruch's membrane, one would expect a much greater amount of new-formed fibrous tissue to be present in the choroid itself, *i.e.* in the primary situation.

It is more likely that this subretinal tissue is derived from some structure already present on the surface of Bruch's membrane, and as regards the retina there is no evidence that any part of this membrane could give rise to such a tissue. But there is support for the view that the retinal pigment epithelium can do so. That cubical epithelium can produce a tissue almost indistinguishable from fibrous tissue is shown by the behaviour of the capsular epithelium of the lens in the production of the laminated tissue which constitutes a capsular cataract.

Again, tubes of pigment epithelium, as well as pigmented spindle-shaped cells, are embedded in the substance of the tissue.

Krückmann has shown that on cutting off the choroidal circulation in rabbits' eyes the pigment cells take on a spindle form, and he believed that a fibrillar substance may be produced by the further activity of these cells. Again, Hess found in a microphthalmic eye with a cyst attached a layer of laminated fibrillated tissue which could only have been derived from the pigment layer of the retina, and very similar tissue is often seen on the surface of choroidal sarcomata, where the pigment epithelium has been apparently stimulated to proliferate by the agency of the growth.

It would seem, then, that in certain cases of choroiditis, when the inflammation affects especially the inner layers, Bruch's membrane remaining intact, the pigment epithelium may be stimulated to proliferate and to produce

a dense layer of fibrillated tissue between the retina and choroid, binding these membranes together.

In one case of syphilitic choroido retinitis examined by Nettleship in the early stage of the disease, nodules of inflammatory cells were present in the chorio capillaris, and notches of granular effusion, containing large, round, pigment-bearing cells, looking like derivatives from the pigment epithelium, were present between retina and choroid, while Bruch's membrane was intact. Probably in a later stage of a case of this kind, conditions similar to those found in the present case would have resulted.

Clinically these plaques of fibrillated tissue between retina and choroid are of interest, in that they probably give rise to the ophthalmoscopic appearances in those cases of choroido-retinitis in which white bands of new fibrous tissue are seen in the fundus. This proliferative variety of the disease is, of course, less common than the atrophic variety; it is well pictured in Frost's Atlas, Pl. XXV, fig. 59. I do not mean to suggest that this appearance is the only one seen in syphilitic choroido retinitis; it probably results especially in those cases where the inflammatory process is confined to the inner layers of the choroid.

The absence of thickening in the vessels of the uveal tract of the eye under discussion is a point worthy of notice. That the presence of endarteritis is not necessarily an accompaniment of syphilitic inflammation of the ocular tissues is borne out by the records of other cases which have been subjected to microscopical examination.

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Mr. F. Richardson Cross said the Society was very much indebted to Mr. Greeves for the enormous amount of work he had put into the subject. He understood from the author that in these syphilitic cases the lesion usually appeared to come from the pigment layer in the iris, and

in the last case the hexagonal pigment seemed to be the centre of the disturbance.

Mr. Greeves, in reply, said he thought the layers next to the pigment epithelium were those chiefly affected, both in the iris and in the choroid. The lesion must begin in the stroma ; but it was in that part of the uveal tract which was adjacent to the pigment layer, *i. e.*, the extreme inner layer of the choroid and the extreme posterior layer of the iris, and the pigment epithelium was stimulated to proliferate in both cases.

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2. *The pathological anatomy of the retina and choroid after complete and partial removal of their respective blood supplies.*

By Dr. Gunjiro Komoto

(Kagoshima, Japan).

(With Plate XIII, fig. 1, and Plates XIV, XV.)

The changes due to circulatory disturbances in the retina and choroid have been studied exhaustively in the eyes of animals by many authors in different ways (Berlin (2), Wagenmann (9), Hertel (3), etc.), but it has not yet been shown sufficiently how far these animal experiments correspond to pathological conditions in the human eye. Wagenmann, indeed, drew attention to the fact that the results of his experiments do not altogether apply to the human eye, because the rabbit's eye differs from the human eye both in its ciliary and retinal vascular systems.

Notwithstanding the frequency of circulatory disturbances in the retinal and choroidal blood-vessels of the human eye, up to the present little has been known of the attendant pathological changes because such eyes can very seldom be obtained for anatomical examination. Although, for instance, the circulatory disturbances due

to a central artery embolus have often been observed clinically, anatomical reports of such cases are still not numerous. And reports on the examination of eyeballs after the cutting through at the same time of the central vessels and of the ciliary vessels are even fewer in number. To my knowledge there are only three such cases on record: a case each of Studer (7), of Birch-Hirschfeld (1), and of Koyanagi (6). Although I know of one other case reported by Hirsch (4), this is somewhat different to the above-mentioned cases, in that the cutting through of the optic nerve was not completely carried out.

By the kindness of Mr. Treacher Collins, I had an opportunity of examining such a case anatomically at the Royal London Ophthalmic Hospital. In this case a hæmangeioma was removed from the orbit, and during the operation the optic nerve, which was firmly adherent to the tumour, and the posterior ciliary vessels were cut through. After twelve days the eye was excised, and the following is the report of this case. It is especially interesting because the interval between division of the vessels and removal of the eye is shorter than in any of the other cases on record. In the case of Birch-Hirschfeld there was an interval of two months, and in the case of Koyanagi of one month, and in the case of Studer of twenty-two days, whereas there was an interval of only twelve days in this case.

Patient's history.—A lady, æt. 44 years, had complained two years before of frontal neuralgia, which lasted several months. Under treatment this became better, but never completely disappeared. It was associated with loss of vision in the right eye. This was accidentally discovered. It was also associated with some proptosis of the right eye, which varied somewhat. The left eye seemed all right.

Status præsens.—Right proptosis—no swelling of lids or conjunctiva. Movement of eyeball good in all directions. On pressing over the eye there is a sense of resistance. No tumour palpable in the orbit. No devia-

tion of the eyeball. No diplopia, pupil active. R.V. $= \frac{6}{36}$. Fundus healthy.

November 6th, 1913.—Diagnosed as tumour of the orbit. The growth was removed from behind the eye. The optic nerve was divided and a part removed, together with a tumour adherent to the nerve-sheath.

November 7th, 1913.—Fundus shows a grey pearly area posteriorly, with vessels coming up to it and ending abruptly at its edge.

November 14th, 1913.—Corneal ulcer to outer side (neuroparalytic).

November 17th, 1913.—Right eyeball excision.

Microscopic sections of tumour.—The tumour consists of large blood chambers, the walls of which are lined by endothelium; there is a dense fibrous stroma of which the part bordering the spaces resembles thickened endothelium. No actual involvement of nerve-sheath. No evidence of malignancy. Diagnosis: Hæmangioma orbitæ.

Right eyeball.—The right eyeball was fixed in Zenker's solution. It was divided into two parts by a horizontal section and then the upper half was mounted in cellodin. The sections were stained by the following methods: Hæmatoxylin-eosin, Van Gieson; also special stains for neuroglia. Some sections were bleached before staining.

Microscopical Sections.

The substantia propria of the ocular conjunctiva is undergoing hyaline degeneration. Hyalin masses are present, some of which are of an elongated corkscrew shape, and obviously derived from degenerated connective-tissue fibres. Cornea: An extensive ulcer is present to the outer side over which Bowman's membrane is destroyed, and the superficial layers of the substantia propria sloughy; infiltration of cornea marked. Much fibrin in anterior chamber. Angles of anterior chamber widely open, some blood in canal of Schlemm to inner side. Pupil widely dilated, vessels of iris almost empty. Circulus iridis

major almost empty. Vessels of ciliary processes contain strikingly little blood. Fibrin present in anterior part of vitreous.

Changes in the retina.—There is a shallow detachment of the retina in some parts; I think perhaps artificial, because of the absence of exudate between choroid and detached retina. Elsewhere the retina lies *in situ*. All signs of retinal inflammation are absent. All the blood-vessels of the retina are quite empty. At the posterior part, especially near the optic disc, the retinal tissue is destroyed, and reduced to a swollen, necrotic mass (Pl. XIII, fig. 1). At the most anterior part of this tissue, many broad band-shaped cells are accumulated; these are derived from a confluence of the inner and outer nuclear layers of the retina (Pl. XIV, fig. 2). The layers of nerve-fibres and of ganglion cells are destroyed, neuroglia alone remaining. The rod and cone layer is degenerated. Passing further forwards, one can easily distinguish the inner and outer nuclear layer, but the other layers are still atrophic or degenerated (Pl. XIV, fig. 3). The layer of nerve-fibres gradually becomes normal, passing forwards from this part, and while the layer of ganglion cells is entirely lost posteriorly. Anteriorly I found a few scattered ganglion cells, which were, however, to some extent, degenerate. The rod and cone layer is less degenerated in the equatorial part anteriorly than posteriorly. This is nearly the same at both sides. Therefore the changes in the retina become less marked the more one passes forward. In the degenerated retina one can find sometimes large, sometimes small clumps of pigment; also in some parts an outgrowth of the pigment epithelial layer has entered into the degenerate retina.

Changes in the optic disc and the optic nerve.—The optic disc is swollen, and here the neuroglia has already proliferated. This new tissue passes over on both sides into the necrotic layer of nerve-fibres. The nerve-fibres of the papilla are reduced to a homogeneous necrotic mass. Also one finds in the papilla numerous immigrated

PLATE XIV.

Illustrates Dr. Gunjiro Komoto's paper on The Pathological Anatomy of the Retina and Choroid after Complete and Partial Removal of their respective Blood Supplies (p. 295).

Fig. 2.—A more anterior part than Fig. 1. A confluence of the inner and outer nuclear layer of the retina is shown.

Fig. 3.—Further forward than Fig. 2. In this part one can distinguish the inner and outer nuclear layers. Note the round pigment-bearing cells in the choroid and sclera.

Fig. 2.

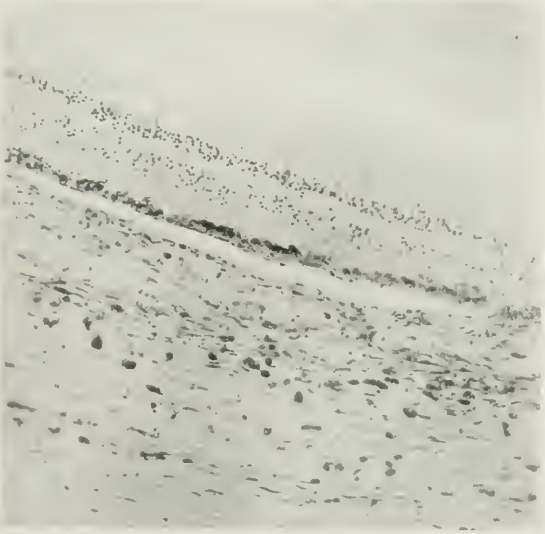

Fig. 3.

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pigment epithelial cells with large swollen cell bodies, containing a few grains of pigment. The lamina cribrosa is well preserved. The optic nerve contains numerous nuclei. Its nerve-fibres have disappeared. Its fibrous trabeculæ stand out clearly, and the rest of the optic nerve consists of neuroglial cells which are becoming altered into round swollen cells with large cell bodies, and in some places appear to have proliferated. The cut end of the optic nerve is already covered with new fibrous tissue. The nerve sheath is also thickened and contains numerous nuclei. In some places the fibrous sheath is undergoing hyaline degeneration.

Change of the pigment layer.—Nowhere can the normal pigment layer be found in the entire eyeball, except in the most anterior part. On looking carefully, one can recognise that the pigment epithelium is in a more or less depigmented state. It shows everywhere a very loose arrangement, and in some parts is quite absent, while in other parts it is arranged irregularly. Its form, too, is not normal. Free pigment from this layer has in some parts entered into the retina and can be seen sometimes as fine granula, and sometimes as large clumps of pigment. The cells also of the pigment layer have immigrated in many parts into the retina. In some parts one can find pigment granules from this layer between the cells of the rod and cone layer. In other parts one can see considerable cell enlargement and also proliferation of the pigment epithelium.

The fact that very remarkable changes have taken place in this layer is shown by the following description: If one examines this layer very carefully, one can distinguish both round or oval cell nuclei and elongated and spindle-shaped nuclei. In many parts of the various sections one can find quite definitely spindle-shaped cells, sometimes pigmented, sometimes unpigmented. These spindle-shaped cells, which are derived from the epithelium of thepigment layer, have entered in places into the other retinal layers like an outgrowth. This change is very

evident in the bleached sections, and one can find various transitional forms between the normal pigment epithelial cell and the spindle-shaped cell. I believe, undoubtedly, that these are examples of the many various states between the normal pigment epithelium cell and a laminated connective-tissue-like structure which may arise from the pigment epithelium under certain circumstances.

Changes in the choroid.—The choroidal tissue is in general very greatly atrophied and more or less depigmented. The choroidal veins are full of blood, the arteries in the posterior part are empty, but anteriorly the latter contain some blood. In a few sections one finds in some parts round cell infiltration, sometimes in groups of cells, sometimes diffused. The changes in the choroid are most marked near the optic disc, and gradually pass off anteriorly until the choroid becomes apparently normal. The changes in the choroidal pigment near the optic disc are very remarkable. Here one cannot recognise the normal elongated and branched chromatophores, but these are replaced by great round or oval clumps of pigment, which are seen in bleached sections to be single, large, round cell bodies, each with a single nucleus, such as are found frequently in choroidal sarcomata (Pl. XIII, fig. 1). The pigment of the choroid has not immigrated into retina, because the lamina elastica is well preserved. The chorio capillaris is absent at the most degenerate posterior part, but is otherwise apparently normal, though almost empty. The walls of the large vessels are apparently normal.

Changes in the ciliary vessels and the ciliary nerves.—On the outer side at the posterior part of the eyeball, especially near the optic nerve, one finds numerous ciliary vessels and ciliary nerves cut across. The lumina of the ciliary vessels are sometimes quite obliterated, sometimes half obliterated, by new-formed fibrous tissue. Around the walls there is some cell infiltration. The new fibrous tissue in some parts is undergoing hyaline degeneration. I also found that the lumen of one of the long ciliary

PLATE XV.

Illustrates Dr. Gunjiro Komoto's paper on The Pathological Anatomy of the Retina and Choroid after Complete and Partial Removal of their respective Blood Supplies (p. 295).

Fig. 4.—Anterior part. The retina is relatively well preserved.

Fig. 5.—High-power view of retina and choroid to show the metamorphosis of the pigment epithelium into spindle-shaped cells.

Fig. 4.

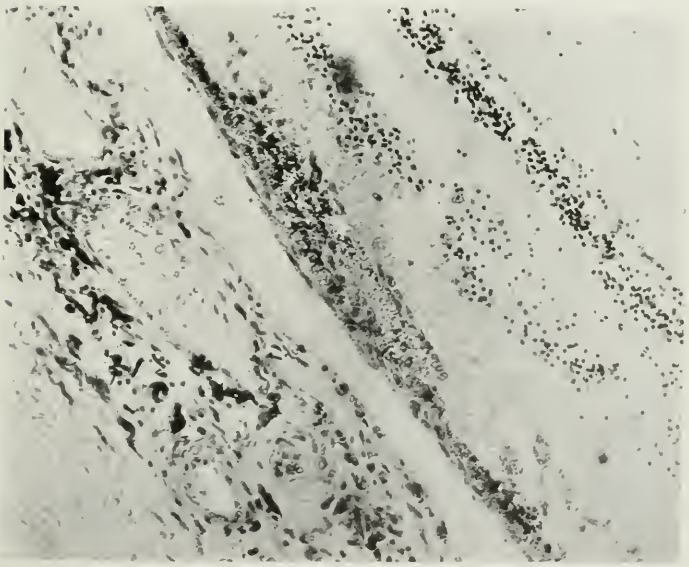

Fig. 5.

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vessels, which passes obliquely through the scleral tissue, was quite obstructed by the new-formed tissue. The ciliary nerves are undergoing degeneration, and around them, too, there is round cell infiltration.

At the posterior pole on the outer side, between the equator and the optic nerve, one finds in many sections giant cell groups, arranged in circles without any signs of inflammation or hæmorrhage. I found that in some sections a narrow, band-shaped, hyaline mass was enclosed amid the circularly arranged giant cell groups. These are probably thrombosed vessels, which are undergoing hyaline degeneration and absorption.

Summary of important points.—In the pathological anatomy of my case I wish to bring the following points into prominence as they are of especial importance. The retina is quite destroyed near the optic disc, and from this forwards to a point some distance in front of the equator it shows degeneration or loss of its nerve elements. Passing forward from here the retina becomes gradually almost normal (Pl. XV, fig. 4). Therefore not only do the changes in the retina become less marked, the more forward one passes, but even the *inner* layers are preserved anteriorly. As to the changes in the pigment layer I wish especially to accentuate the fact that it is changed in some parts into spindle-shaped cells, which in a few places have entered into the other retinal layers like an outgrowth. The choroid is in general much atrophied. Degeneration of its pigment cells is very evident near the optic disc, where they have assumed a rounded form.

Discussion.

It is often very difficult to distinguish whether pathological changes in the retina and choroid are the consequence of local inflammation, or whether they are simply degenerative and dependent on circulatory disturbances. There can be no doubt on this score in the present case, however, both because of the history, and because of the

obstructed appearance of the posterior ciliary vessels. And any round celled infiltration present must be due to the same primary cause. Now it is supposed by many authors that the nutrition of the inner retinal layers are entirely dependent on the circulation through the central artery, while the outer retinal layers are alone dependent on the choroidal vessels. We know that in the present case the degeneration of the posterior part of the retina is not only to be attributed to the circulatory disturbances in the central artery, but also to the complete interruption of the blood-supply through the posterior ciliary vessels. On the other hand any changes in the anterior part in the retina must have been caused chiefly by the effects of interference with the central system of blood-vessels, because the blood-supply to the anterior part of the uveal tract through the anterior ciliary vessels was still intact. But *all* the layers of the retina are at any rate well preserved, if not entirely normal, in front of the equator (at least so I judge from my examination). How can one explain these appearances? We can only suppose that the nutritive sphere of the central artery is much more limited in the front part than in the back part of the retina. If my supposition is not wrong, then even the inner layers of the front part of the retina must be largely provided for by other blood-vessels than the retinal system, viz. the choroidal system.

The changes in the pigment layer are most likely to be attributed to the interruption of the posterior ciliary vessels; this is supported by the animal experiments of Wagenmann and Hertel in which they found that the pigment layer remained normal after cutting through the central artery of the retina.

Concerning the violent pigment degeneration in the posterior part of the choroid near the optic disc, the natural assumption is that the circulatory disturbance in the posterior ciliary vessels was the cause of this.

But, as I have already written in the patient's history, an ulcus corneæ was present at outer side of the cornea.

This was almost certainly neuroparalytic in origin and due to the section of the ciliary nerves. How far this factor may have helped to determine the pigment changes in the choroid it is difficult to say. The normal appearance of the anterior part and the choroid, however, tend to show that the vascular factor is the important one.

I will now quote very shortly some other previously published cases, so as to draw a comparison with my case.

In the first case, that of Studer, in which at first iridocyclitis had been present, an operation for cataracta complicata was undertaken, and finally glaucoma absolutum supervened and resectio optico ciliaris with tenotomy of the M. rect. ext. was carried out. The operation, however, was not successful, therefore enucleation of the eye was performed twenty-two days later. Anatomical examination showed the following changes: the inner half of the retina was apparently normal in thickness, but the nerve fibre layer and the layer of ganglion cells were lost, while proliferation of the neuroglia and degeneration of the rod and cone layer were present. On the other hand the outer half of the retina was very thin, and its normal structure was quite lost, and only supporting tissue was to be found. A most remarkable appearance, however, was produced by deep pigment deposits throughout the whole retina. These deposits were large and round or ellipsoid in shape; in some parts the clumps of pigment were rather angular in shape in the inner layers of retina, more especially over nearly the whole temporal half. The clumps of pigment consisted of cells four to eight times larger than normal epithelium. The nasal half of the choroid was more or less atrophied. The chorio-capillaris had partly disappeared, but the large vessels were moderately hyperæmic. The choroid itself was nearly normal in thickness. The temporal half of the choroid showed marked hyperæmia, and in many parts one could find extravasated blood corpuscles.

In the second case, that described by Birch-Hirschfeld, the eye was removed two months after resectio optici with

tenotomy of the M. rect. ext.—the enucleation was performed because of tuberculosis orbitæ. In the anatomical sections he found total atrophy of the temporal half of both retina and choroid, while the nasal half showed extreme retinal atrophy and alteration of neuroglia. The nerve fibre layer and ganglion cells were quite lost. The choroid was well preserved in this part, and the chorio-capillaris, the lamina elastica, and the pigment layer were found to be normal. The change in the temporal half of the pigment layer was especially peculiar, on the inner side of the new fibrous tissue which represented retina and choroid numerous smaller or larger cystic formations were to be found. Inside these cysts granular detritus was contained, and also well-formed red blood corpuscles.

In the third case, that of Koyanagi, excision of a growth of the optic nerve with tenotomy of the M. rect. inf. was first carried out, and then one month later enucleation of the globe was undertaken. Pathologically the lower and inner and upper and outer equatorial parts of the retina showed intense atrophy of all the nerve elements, with numerous immigrations of pigment into it. The other parts of the retina showed slight atrophy of the inner layer. Between both the internal and external nuclear layers of the retina numerous round or ellipsoid cystic formations were present. The pigment epithelial cells had grown not only into the retina, but also into the optic nerve trunk. In the corresponding parts of the choroid slight sclerosis and loss of some vessels, especially of the chorio-capillaris, were to be found.

The three cases just quoted show us that on the side corresponding to the tenotomy, the retina was quite lost or consisted of supporting tissue only, while the other parts of the retina were atrophied or slightly degenerated. Studer supposed that the cutting through of both the posterior and anterior ciliary vessels would account for the extreme alteration of the retina in his case. The other two authors also agree with Studer's opinion. Hirsch, too, held the same opinion. In his case the M. rect. ex. and inf. were

divided at the operation. Four weeks later enucleation of the eyeball was performed because of the continued pain. Pathologically the temporal half of the retina showed changes which strongly resembled those found in retinitis pigmentosa.

The changes in the present case were quite different from those in the previous cases, as the front part of the retina was well preserved, while the posterior part was quite destroyed or showed very extreme changes. There was no indication that the anterior ciliary vessels had been interfered with. We must suppose that the central artery and posterior ciliary vessels do not supply the front part with nutriment, or at least only to a slight degree, and consequently we must draw the conclusion that the channels of supply for the front part of the retina are different from those for the posterior part. Possibly in the other three cases some or all of the posterior ciliary vessels may have remained intact on one side.

I will now proceed to draw a comparison between the present case and Wagenmann's animal experiments as an indication how far the human eye corresponds to the animal eye, more especially to the rabbit's eye.

Wagenmann obtained the following results by cutting through the optic nerve trunk together with all the posterior ciliary vessels. The retina was evidently thickened and partly destroyed. Necrosis with faulty staining of the cell nuclei was seen to some extent; the pigment layer had disappeared to a very large extent, and its existing cells were loose and necrotic. The medullated nerve fibres were changed into a crumbly mass. The optic disc was necrotic. Here numbers of lymphocytes had infiltrated the tissue. It is noteworthy besides that many of the walls of the capillary vessels were enormously swollen and were undergoing hyaline degeneration.

The retinal changes in the present case are more or less similar in the posterior part to those found in Wagenmann's experiments. But in Wagenmann's animals the eyes were removed only one day after the operation, while

in the present case twelve days elapsed after the operation. Yet in the present case there were fewer changes than in the rabbit's eyes. From what does this difference arise? We must consider two possibilities: The first is whether all the posterior ciliary vessels were not all cut through; the second, whether a freer anastomosis with the anterior vessels took place in the human eye than in the animal's eyes, although all the posterior ciliary vessels were cut through. Since there is little doubt that the posterior ciliary vessels were all cut through, the latter supposition seems probable.

Concerning the change in the pigment epithelial layer: I found various stages between normal pigment epithelial cells and spindle-shaped cells, and in some parts the spindle cells had entered into the other retinal layers like an outgrowth. Krückmann (5) found exactly the same changes in experiments on animals' eyes. In both the present case and in Krückmann's animal eyes, it is evident that this tissue, similar to fibrous tissue, was derived from the pigment epithelial layer.

On the outer side of the globe between the equator and optic nerve I found giant cell groups arranged in a circular fashion. Inside, a narrow band-shaped hyalin mass was enclosed. Concerning these giant cells, Studer found the same cells in his case. Besides this, he found a foreign body, a piece of silk thread. Therefore he thought these giant cells were caused by the foreign body. In the present case I found in the same position some hyalin masses, and I think it possible that these hyalin masses, probably derived from thrombosed vessels, acted as a foreign body, thus giving rise to the giant cells.

Concerning clinical observations, Studer's article refers to three cases of Schlodtmann (10), one case each of Scalinici, Gruening, and Adamück. In all these cases the changes seem less than in the present case. A few years ago E. T. Collins (8) reported a very interesting case clinically. It was an intradural tumour of the optic nerve, in which extirpation of the tumour had been undertaken

5½ years before. The eyeball itself was preserved. The appearance of the fundus was as follows :

A large white area, looking like dense connective tissue, occupies the whole of the central region of the fundus. Around its margins, and for some distance beyond, are densely black, branching patches. The optic disc is situated at the upper and outer corner of the white area ; its margin cannot definitely be differentiated, but its presence is recognised by the retinal vessels which, as branching white lines, are seen to radiate from it. Some of these show a thin central red streak. A red reflex is obtained from the periphery of the fundus in its entire circumference, but thinning of the choroid and disturbance of retinal pigment is present everywhere except to the outer side.

According to this description great degeneration of pigment took place around the optic disc for some distance. The present case, too, showed great pigment degeneration around the optic disc ; in this respect, the cases are similar. The white fibrous tissue seen in Collins's case may have been scleral tissue exposed by the degeneration of the nerve, or possibly tissue of new formation which grew in from behind and took the place of the degenerated nerve.

In conclusion, my thanks are due to Mr. R. Affleck Greeves for his friendly help and advice in the course of this work.

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3. *A case of primary pneumococcal acute choroiditis.*

By J. Gray Clegg.

The patient, B. G. H—, a healthy looking, but rather undergrown young man, æt. 27 years, came to see me on December 2nd, 1908, complaining about the vision of the right eye. The history was that eight days previously he had noted muscæ, which had been more pronounced during the latter two days, and on the 1st inst. the sight had failed.

Testing gave R. V. = J. 20.

L. V. = $\frac{6}{36}$, $\bar{c}$ — 2 D. sph. $\bigcirc$ - 0·5 D. cyl. = $\frac{6}{6}$, J. 1.

That the defect of the right eye was recent I knew from the fact that on October 19th, 1905, I had tested him and found the R. V. $\frac{6}{36}$, $\bar{c}$ — 1·75 D. sph. $\bigcirc$ — 0·5

D. cyl. $= \frac{6}{6}$ J. 1, and then the media were clear and the fundus normal.

On examination, there were no external signs and the media were clear, but the disc was slightly blurred and the retinal vessels full. Two whitish areas with fairly defined margins were visible, one above and one below the fovea. The upper one was roughly oval, and the vertical long diameter was about one and a half times that of the disc; the lower was crescentic with the concavity upwards, also one and a half disc diameter in length.

A thorough investigation of the family history and of the patient's general condition by Dr. Boyle, of Disley, gave no tangible clue as to any cause.

On December 3rd, slight injection of the conjunctiva appeared.

By the 5th, the iris was slightly discoloured and the anterior chamber was rather deeper than normal. The cornea showed a little haze resolvable by the loupe into fine lines. No deposits on Descemet's membrane. There was distinct haze in the vitreous, the exudates were much enlarged, and the disc was only just visible.

The eye was seen by Dr. Hill-Griffith, who concurred with me that the most probable cause was tubercle.

On the 7th, Mr. Treacher Collins expressed the same opinion independently.

On the 8th, the eye was more irritable, the cornea was hazy, A.C. deep, pupil wide with large dilated vessels in the iris, vitreous full of opacities, areas of exudation very indistinct and disc not visible.

December 9th.—Opsonic index taken by Dr. Loveday, reported to be 0·9.

Treatment.—Hot bathing, gutt. atropin gr. 2, ad. ℥j. Open-air, overfeeding, ol. morrhuæ.

December 11th.—The injection of the eye was less, cornea clearer, A.C. still deep, pupil medium, dull red reflection. Subcutaneous injection of $\frac{1}{30000}$ mgrm. of bovine tuberculin.

December 14th.—Cornea clearer, fundus just visible

above but directly down, a dirty white reflection. Opsonic index 1, injection of $\frac{1}{30000}$ mgrm. repeated.

On the 17th, the cornea was clear, iris intensely congested, pupil smaller. Injection changed to human tuberculin.

December 24th.—T +. General inflammation more marked, some hypopyon.

Dr. Loveday reported that index to bovine tubercle was now 2, showing the disease not due to that variety. So far the index for human had been 0·75, but to-day it had gone up to 1·33 after the one injection of human tuberculin.

December 28th.—Fully developed panophthalmitis. A.C. almost full of exudate. T. full, slight tenderness. V. = bare P.L.

December 31st.—Chloroform was administered, the cornea was incised, and specimens of the fluids taken. The cornea was then removed, and the whole of the interior of the scleral cavity scooped out and preserved. Nothing of interest occurred in the stump afterwards.

The pneumococcus of Friedlander alone was found in the specimens. The vitreous and choroid, after being kept in a closed bottle in the cold for six days, during which the cocci would die, were then introduced under the skin of a guinea-pig which was afterwards killed and examined, but no sign of tubercle was found.

The other eye remained perfectly healthy ; it was seen last week.

The case is one of especial interest in that : (1) It was seen in a very early stage, in an eye known to be previously perfectly healthy. (2) It was mistaken by three ophthalmologists for a tuberculous lesion, and the inflammatory signs were supposed to be due to the toxins arising from that lesion. (3) No primary seat of infection was discovered in any part of the body after repeated and most careful investigations by several medical men. The heart was normal. The existence of two foci at the first examination excluded neoplasms and parasites.

In the whole of my experience of ophthalmology I do not remember a similar case. Suppuration in a globe is commonly the result of pneumococcal activities, but in other cases there has been some definite lesion elsewhere, or there has been a penetrating wound allowing direct infection from without.

I shall be glad of any suggestions which will enable me to diagnose such a case in the early stages and differentiate it from acute progressive tubercle.

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4. *Acute symmetrical central choroido retinitis.*

By Walter H. Jessop.

William S—, æt. 29 years, printer's machine man, was seen first by me on March 3rd. Last September had influenza, followed by jaundice, which lasted two months. He was in bed for one month. After recovering had another attack of influenza, accompanied by bad headaches. On February 2nd he found he could not read with the right eye, but had no pain. He says that the right sight was impaired one month ago, but is now improving. The left vision is worse for fourteen days. V. = Right $< \frac{6}{60}$ — ·5–·75 cyl. | $\frac{6}{36}$. Left $< \frac{6}{60}$ —, — ·5–·75 cyl. | $\frac{6}{36}$ ptly. Nothing abnormal to be seen by naked eye; pupils act normally. Ophthalmoscop. Left eye: The optic disc has a ring of œdematous retina round it. About $\frac{2}{3}$ O.D.'s breadth from outer side of optic disc, and in the neighbouthood of the Y.S. the retina is œdematous, and there are numerous small disseminate spots of black pigment—some hexagonal in shape. The spots are not all the same size. The blackness of the spots is intense, and the retina between them has a yellowish white colour. There are no other changes in the fundus. Right: The same appearance, but the retina does not look so œdematous. Fields of

vision for white and red are normal peripherally. Centrally in the right eye the white square looks white, but there is a small scotoma for red. Left eye: The white square looks greyish and shapeless; scotoma for red. Wassermann reaction negative. No sign of tubercle. Family history: Parents, one brother, and one sister have good sight.

March 28th.—Oph. R.E. in neighbourhood of Y.S. are several small discrete black spots. The choroid between them disturbed, but no œdema now. L.E. the same.

April 20th.—Oph. R.E. and L.E. The black pigmented retinal changes are more diffused but limited by superior and inferior temporal vessels. The spots are more numerous, not so regular, and a few are much larger. Between the spots the retina is atrophied. V. = R. $\frac{6}{36}$ — 1–·75 cyl. | $\frac{6}{12}$. L. $\frac{6}{60}$ — 1–·75 cyl. | $\frac{6}{24}$. In right eye central red scotoma found with difficulty. Left eye red scotoma still present. Treatment throughout mercurial inunctions.

(*Card specimen.*)

Dr. George Mackay said it seemed to be a case of symmetrical poisoning in the macular region, associated with the toxin of influenza or the cause of jaundice. He had seen a similar appearance in a few cases; the most recent was not symmetrical. The patient was a man æt. 42 years. About a month prior to coming for advice he had noticed some redness of his eyes, and after that passed away some dimness in the vision of the R.E., with a heavy but not painful sensation towards the temporal side. The faces of his friends were blurred, and white objects had sometimes a greenish tinge. With the R.E. he deciphered letters of $\frac{6}{12}$ with difficulty, and read J. 4, and could not be improved with glasses; with the L.E he read $\frac{6}{9}$, and with a weak convex cylinder $\frac{6}{6}$ and J. 1. Looking at an astigmatic clock face, the lines in the right upper section from twelve to three

o'clock seemed less well defined, and there was a relative scotoma for colours above the point of fixation. Tested with parallel lines there was blurring but no actual metamorphopsia in the same part of the field. A small patch of superficial choroiditis could be made out in the macular region. His eye had not been under special strain from use or exposure to bright light. He had been a heavy smoker, but there was no symmetrical papillo-macular scotoma. His upper teeth had all been extracted, but he had one decayed præmolar in the left lower jaw. Its removal was advised, and it was found to be in a very foul state. Two months later the vision of the affected eye had improved to $\frac{6}{6}$ fairly. Colour and form sense had returned, and he was now only conscious of a slight dulness of perception in the area where there was previously a definite relative scotoma. With the ophthalmoscope only a fine pigmentation, very similar to that in Mr. Jessop's case, could be seen in an irregular patch on the side of the macula nearest to the disc.

IX. DISEASES OF THE LENS.

1. *Apparent accommodation with aphakia.*

By E. Treacher Collins.

(With Plate V, fig. 6.)

My attention was first directed to this subject by finding that a lad, from whom I had removed a lamellar cataract in each eye, was able to read $\frac{6}{6}$ and J. 1 with the same pair of glasses, without in any way altering their position in relation to his eyes.

Being unable to explain how this apparent accommodation of the eye was brought about in the absence of the crystalline lens, I showed the case to several of my most critical colleagues, who confirmed my observations as to the lad's remarkable capabilities, but who were likewise at a loss to offer an adequate explanation of the condition. They, however, made some suggestions to me for the further examination of the case. These, and others which occurred to me, I have carried out, and have also looked up what has been previously written on the subject. It is the outcome of these observations which I now propose to bring before you.

In the last years of the eighteenth and beginning of the nineteenth centuries several papers were read before the Philosophical Society, discussing the question as to whether the eye deprived of the crystalline lens retained any power of adjusting itself for different distances. Of particular interest was the subject at that time, when the nature of the change which occurred in the normal eye during accommodation was still undecided.

Ramsden and Home (1) described the case of a man from whom the crystalline lens had been removed, who, with a lens in front of the eye, was able to read letters of the same size at different distances. They were of the opinion that this apparent accommodative power was due to an increased curvature and moving forwards of the cornea.

Thomas Young (2) tested with an optometer, founded on the principle of Scheiner's experiment, patients from whom cataracts had been removed by Mr. Ware. Though with characteristic caution he describes the results arrived at as only "tolerably satisfactory," the outcome of his experiments went to show that in an eye deprived of the crystalline lens the actual focal distance is totally unchangeable.

Sir Everard Home (3) with the assistance of Sir Henry Englefield experimented with Young's optometer, and also with an improved pattern, on a patient with aphakia, and considered that they obtained evidence of accommodative power.

Arlt (4) reported the case of a young man with aphakia who with $+\frac{1}{3\frac{1}{2}}$ could read both at six and at twenty-four inches, and with the same glass could recognise the hands of a steeple clock at a distance of more than 500 paces.

Graefe (5), having investigated the subject, arrived at the conclusion that in aphakic patients some accommodative power remained. He remarked, however, that those who made the most accurate and, on repeated investigation, the most uniform statements had the least range.

Donders (6) stated, with all the emphasis of italics, that his investigations had led him to the conclusion "that in aphakia not the slightest trace of accommodation power remained." The test on which he relied in making this positive statement was as follows. He made a patient with aphakia look at a distant point of light, and accurately adjusted his vision for it with a lens, so that it was seen as a circle. He then directed him to

make an accommodative effort. In doing this the patient saw no alteration in the shape of the light, only a diminution in its size, attributable to a narrowing of the pupil. Whereas, if he added the weakest + or — lens, or made the slightest alteration in the position of the lens already in front of the eye, the circle of light became elongated.

In 1872 Förster (7) reported a series of twenty-two cases of apparent accommodation in aphakic eyes, laying great stress on the amount being greater in young persons than in old.

Donders (8), in reply to Förster's paper, pointed out that acuteness of vision in aphakia does not remain the same for different distances, which, he says, ought to be the case if accommodation were present. He commented on the importance of taking into consideration the size of the pupil, which, by altering the size of the diffusion circles, alters the acuteness of vision, and so enables the aphakic eye to discriminate. Such power of discrimination, he says, does not oblige us to assume that true accommodation is present.

Woinow (9) reported eleven cases with aphakic eyes, in which, on testing their near vision, he found some apparent range of accommodation present. He was unable to explain the mechanism by which these appearances were brought about. He discusses three factors to which it might possibly be due:

(1) An increase in the convexity of the anterior surface of the vitreous so that it acted as a + lens.

(2) An alteration in the depth of the fundus by the action of the ciliary muscle.

(3) Compression of the globe by the external muscles.

In Flint's *Physiology of Man* there is a description of a case of Dr. Edward G. Loring's, which is very similar to the one which forms the basis of this paper. It is that of a girl, who, when between 12 and 13 years old, had her eyes operated on for cataracts by discission. Five years later, it was found that with + $\frac{1}{24}$ she could read

fluently with either eye Snellen 30, and was able with both eyes to pick out most of the letters of 20 at 20 ft. She could read No. 10 at 10 ft., and No. 5 at 5 ft. With the same glass, and no change of position on the nose, she read No. $1\frac{1}{2}$ Snellen fluently, holding the book naturally at 12 in. The size of the pupils and movements of the iris were normal in every respect. With the ophthalmoscope the pupillary space was found in the right eye to be entirely free from any remains of capsule, while, in the left, a narrow rim of whitened membrane just encroached on the upper pupillary margin.

In 1885, T. H. Bickerton (11) described the case of a man, æt. 56 years, in whose right eye the lens and its capsule had absolutely disappeared, as the result of an injury with a piece of steel twenty-nine years previously, and who had perfect acuity of vision with apparently some power of accommodation.

With + 10 D. he read $\frac{2}{2}\frac{0}{0}$ perfectly. He could also read it with + 10·25, + 10·5 and + 10·75, but not quite so well with the latter.

With + 14 he read J. 1 with ease at 9 in. and words at $12\frac{1}{2}$ in. and $6\frac{1}{2}$ in.

The pupil in ordinary light measured $2\frac{3}{4}$ mm., in dark $4\frac{1}{2}$ mm., in bright light 2 mm. On looking from distance to near it changed from 3 mm. to 2 mm.

Silex (12), in 1889, described the case of a boy, æt. 14 years, who had a cataract removed by repeated discissions and ultimately obtained a clear, round, freely movable pupil. This patient, he says, met Donders' condition that, "only when it is demonstrated that the visual acuteness remains the same within a certain interval, there can be question of accommodation."

His distant and near vision for type with the same lens were equal to $\frac{1}{2}$. If his vision for a distant point of light was focussed with a lens so that he saw it as a clear disc, and he was then told to direct his attention to fine threads held in front of the light near to his eye, he said the luminous little disc became converted into a vertical oval.

On examination of his cornea with the ophthalmometer, no evidence of any alteration in its curvature was obtained, and there was no evidence to support Schneller's view of an elongation of the antero-posterior axis of the globe taking place by compression of it by the external ocular muscles.

In 1895, A. Edward Davis (13), in an article entitled "Accommodation in the Lensless Eye—To what is it due?" records the details of two cases which he had submitted to a most searching and thorough examination.

The first case was that of a man, æt. 42 years, who had had a sclerosed black cataract extracted from his right eye, with an upward iridectomy. Nine months later with + 11·5 D. he saw $\frac{20}{10}$ Sn., and with the same glass, held at the same distance on nose, he read J. No. 1 from 14 to 18 in. He did the same with the upper lid held up, also after the instillation of a few drops of cocaine and the introduction of a speculum to hold the lids open.

A + 0·5 D. spherical glass added to his distant glass made him see worse for distance; with a − 0·5 D. spherical added he saw the same, while a − 0·75 D. spherical made him see worse. With Donders' test and the distant point of light, an effort of accommodation on the patient's part enlarged the circle of light in the vertical meridian, just as when a weak spherical was added.

Examination with the ophthalmometer showed a slight advancement, and change in the curvature of the cornea, on an attempt at accommodation. The same changes were observed when the lids were held open with a speculum, and when the ciliary muscle was paralysed with scopolamine. From which Davis assumes that the changes in the cornea must have been produced by the action of the external muscles of the eye.

He was unable to detect any change in the depth of the fundus of the eye with the ophthalmoscope when the eye changed from a state of rest, from looking in the distance with the opposite eye, to a state of accommodation.

The shape of the pupil was an irregular oval, from the iridectomy, and free from membrane, except a very narrow margin at the edge.

His second case, a private patient of Dr. Webster's, was a school-boy who had had congenital cataracts removed by a series of discissions. His left eye with + 16 D. saw $\frac{20}{20}$, and with the same glass, J. No 1, from 10 to 18 in., not moving it and looking directly through the centre. His right eye, which had also been operated on, had no apparent accommodative power. Holding the lid up with the finger, or with a speculum, or even paralysing the eye with scopolamine did not change his vision for distance, or his near point. With Donders' test of the distant point of light an effort of accommodation elongated the circle slightly in the vertical meridian.

On examination with the ophthalmometer an effort at accommodation made absolutely no change in the relative position or size of the corneal images. No change could be detected in the depth of the fundus on accommodation by ophthalmoscopic examination. The pupil was circular, active, $3\frac{1}{2}$ mm. in diameter, but considerably encroached upon at the inner side by a crescentic band of membrane, giving it somewhat the character of a stenopaic slit. When the pupil was widely dilated it was filled up entirely by a dense membrane, except for a small central opening.

Davis sums up his conclusion from the study of the subject as follows: "That the accommodation present in the lensless eye is due chiefly if not solely to the ability of a patient in such cases, to interpret dispersion circles." He considers the slight advance and change in curvature of the cornea, in his first case, insufficient to account for the amount of accommodation power the patient possessed. He calls attention: First, to the great increase in size of the retinal images produced, by the removal of the crystalline lens and replacing it with a lens in front of the eye. Second, to the effects of narrowing of the pupil as the result of convergence and an effort at accommodation, and to that of a small central opening in a mem-

brane. Third, to the extraordinary acuteness of vision present sometimes after cataract extraction. Fourth, to the effect of slight tilting of a strong plus glass in neutralising monochromatic aberration.

Rogman (14), in 1899, published notes of six highly myopic eyes, which, by the removal of the crystalline lens, had been made nearly emmetropic, and which possessed an apparent range of accommodation varying from 2 D. to 5 D. None of them required to wear spectacles. He attributes this false accommodation to contraction of the pupil in cutting off circles of diffusion, and lays stress upon the importance of maintaining a small and active pupil in operations for removal of the lens.

Salzmann (15), in an article upon the influence of diffusion circles on vision, has demonstrated how they depend not upon the actual size of the pupil, but upon that of the virtual image of the pupil seen through the cornea, or through the cornea and whatever lens may be worn in front of it. The size of the virtual image varies with the strength of the lens, and is much larger with convex than concave lenses. The range of distinct vision in aphakia will depend on the size of this virtual image of the pupil, being greater when the glasses worn are weakly convex or concave than when they are strongly convex. Hence we should expect a greater range of vision where the crystalline lens has been removed in highly myopic eyes than in eyes with hypermetropia or emmetropia.

From the foregoing epitome of what has been written on this subject it will be seen that a number of cases have been recorded by well-qualified observers, where patients with aphakia, without any alteration in strength or position of the lens placed in front of the eye, were able to see clearly at different distances. The extent of the range of this apparent accommodation varied considerably; in some of the cases it was only slight, in others it was sufficient to enable the patient to dispense with the necessity of using two pairs of glasses, and in a few cases it has allowed the patient to have full acuity of vision at a

distance of 6 metres and at ordinary reading distance. To this latter category the case I am now going to relate belongs.

Reginald D—, when æt. $7\frac{3}{4}$ years, was admitted to Moorfields Hospital with a zonular cataract in each eye. That in the right eye was completely dissolved by two discission operations—the first in January, 1906, the second in September, 1907. That in the left eye required three discission operations before it was completely dispersed—in September, 1907, and in February and July, 1908.

In September, 1909, his vision in the right eye is noted with + 13 D. = $\frac{6}{6}$; in the left eye with + 13 D. = $\frac{6}{9}$. It was when he came to see me on February 23rd, 1914, that I discovered he was only using one pair of glasses both for distance and reading. On trying his vision with the test types I find with + 13 D. in front of his right eye he reads $\frac{6}{9}$, and without any alteration of it in relation to his eye, Sn. 0·5 at 50 cm. and at 30 cm. With + 13 D. in front of his left in a similar way he reads $\frac{6}{6}$ and Sn. 0·5 at 50 cm. and at 30 cm. He can do the same when the eyelids are held away from the eyeballs. Both pupils act briskly to light and on convergence; they are regularly circular, there being no adhesions between iris and lens capsule. On exposure to bright light they each contract up to 3 mm. in diameter, and on dull illumination dilate to 6 mm. On focal illumination in the dark room, in the right eye a regularly circular opening in the capsule 3·5 mm. in diameter is seen in the pupillary area. The centre of it is a little higher than the centre of the pupil, and it is bounded by a very well-defined white edge. When the pupil is dilated with atropine the whole of it, with the exception of this central circular patch, is occupied by grey capsule (Pl. V, fig. 6). Focal illumination of the left eye, with the undilated pupil, shows the outer $\frac{2}{3}$rds black, and free from membrane, whilst the inner third is occupied by a crescent of grey membrane, with a sharply-defined edge. A very delicate

strand stretches across the outer black area at the upper part. When the pupil is dilated in the left eye with atropine an oval sharply-defined opening, measuring about 5 mm. vertically and 4 mm. horizontally, is seen in the membrane, with a sharply-defined margin, and the delicate strand stretching across the upper part. The remainder of the pupillary area is not, however, as in the right eye, entirely filled by the grey membrane, there being a complete black ring showing between the dilated pupillary border and the outer margin of the disc of opaque capsule (Pl. V, fig. 6).

The employment of Donders's test for accommodation did not prove very satisfactory. The boy's right eye was focussed for a distant point of light with a + 13 D. lens, so that he saw it as a well-defined circle. Crossed threads were then held between him and the point of light, at which he was told to look. He said, on doing this, that the circle of light became smaller, but did not change its shape. This was in accordance with what Donders found, but the lad also denied seeing any alteration in the shape of the spot of light with this eye if, whilst he was looking at it, a + or — glass was held in front of his + 13 lens. When the same test was applied to his left eye, on looking at the crossed threads he said the spot of light became prolonged out laterally to his left side. He described a similar alteration in its shape occurring when a — 0·5 D. lens was held in front of his + 13 lens, but saw no alteration in the shape of the circle of light when a weak + glass was added.

With the assistance of Mr. Fison, the senior house-surgeon at Moorfields Hospital, I tested the boy with Sutcliffe's ophthalmometer and with Batten's continuous eye-bath, to try and ascertain if the apparent accommodation could be attributed to any alteration in either the position or curvature of the cornea.

In examining with the ophthalmometer I directed the boy to look through a correcting lens at letters in the far

distance to one side of the instrument with one eye, while the other eye was under observation. I next held up an object a foot distant from him, but in the same line as the letters, and made him concentrate his attention on it. No alteration in the relative position or definition of the images reflected in the cornea could be detected, whilst this alteration in the object looked at by the fellow eye took place.

Batten's continuous eye-bath or "Hydro-ophthalmoscope" was applied, and filled with a mixture of water and a little glycerine. This fluid, which covered the cornea, has nearly the same index of refraction as the aqueous humour; the front of the bath consists of a piece of plain glass, so that a patient, when it is applied, will have the refractive power of his cornea abolished. If his crystalline lens is also absent, then parallel rays will pass into his eye practically unbent. When this instrument was fixed in position in front of the boy's left eye he required + 31 D. to enable him to read $\frac{6}{9}$; with the same lens he was able to read words of J. 4 at 30 cm. As a + 20 D. lens was the highest I had at hand I had to combine it with a + 11 D. to obtain the + 31 D. In looking through these two lenses there must have been considerable spherical aberration and also loss of illumination. Whilst the eye-bath was applied to the boy's right eye he became faint, and the examination of it had to be discontinued before it was completed.

On examination of the fundus of the boy's eyes with the ophthalmoscope, no alteration in the depth could be detected, when he was made to change from looking in the distance to an object close at hand.

After the application of atropine, which caused wide dilatation of both pupils, his left with + 13 D. in ordinary daylight could only read J. 10, and his right only J. 4. When, however, he was taken into the dark room and a strong light was concentrated on to the test type, he was still able, with the + 13 D., to read J. 1 well with his right eye, and words slowly with his left.

After the application of cocaine, which produced in each eye a moderate dilation of the pupil, he could still read J. 1 with + 13 D. with his right eye, but only J. 2 with his left.

Various explanations have been suggested as to the way in which this apparent accommodation takes place in the absence of the crystalline lens. Most of them assume some muscular action. We may, therefore, classify the various theories by considering the different sets of muscles to which the condition could possibly be due.

(1) An alteration in the position of the lens worn by sliding it forward on the nose away from the eye. As Landolt has put it, "to make the hand play the part of the ciliary muscle." He points out that a hypermetrope who is corrected for infinity by the convex 13 placed 1 cm. from the cornea would need only to carry this glass 15 mm. further away, and it would act like a convex 16 and adapt him for 35 cm.

(2) Action of the orbicularis muscle by pressing the eyelids against the eye might possibly alter the curvature of the cornea.

(3) The extra-ocular muscles might conceivably act in four different ways:

(*a*) By compression of the sides of the globe causing elongation of the antero-posterior diameter. Such an action has been termed "external accommodation."

(*b*) By compression of the sides of the globe causing an increase curvature of the cornea.

(*c*) By retraction of the eyeball in the orbit, so drawing it away from the lens in front.

(*d*) By altering the position of the eye so that it looks through the side and not the centre of the lens.

(4) Two actions have been suggested in connection with the ciliary muscle:

(*a*) By dragging on the cornea causing an increase in its curvature.

(*b*) By compression of the vitreous humour causing its anterior surface to become convex.

(5) Action of the sphincter muscle of the pupil cutting off circles of diffusion.

I will now consider each of these possible forms of muscular action in connection with my own case :

(1) It can be definitely stated that the apparent accommodation was not brought about by any alteration of the position of the spectacles on the nose.

(2) That it was not due to any pressure of the eyelids against the globe is shown by the lad being able to see just as well at different distances with the same lens, when the eyelids were held away from the globe, as when they were in contact with it.

(3) (*a*) Any elongation of the globe by pressure of the external ocular muscles was excluded by examination with the ophthalmometer and by direct examination with the ophthalmoscope.

(*b*) Any alteration in the curvature of the cornea, by compression of the sides of the globe was negatived, by the examination with the ophthalmometer and with Batten's eye-bath. When the latter was applied, and all the refractive power of the cornea thereby removed, the lad could still see words of J. 4 with the same lens as he required to read $\frac{6}{9}$. That he could only see J. 4 and not J. 1 may, I think, be accounted for by the amount of spherical aberration produced by the + 31 D. lens and the reduced illumination. It is not, I think, necessary to assume that it was due to abolition of a changing curvature of the cornea.

(*c*) No alteration could be detected in the distance between the lens and cornea, when the boy changed from looking at letters 6 metres away to reading at a distance of 30 cm. ; such as would have occurred if there had been any retraction of the eyeball.

(*d*) It was also noted that he looked through the centre of the lenses in front of his eyes, both to see in the distance and near at hand.

(4) That his apparent power of accommodation was not due to any action of his ciliary muscle was shown, by his

being able to read J. 1 with the lens he used for the distance after atropine had been employed, provided sufficient light was concentrated on the test type.

(*a*) That no alteration in the curvature of the cornea was produced by the action of the ciliary muscle was further proved by the experiments already referred to with the ophthalmometer and Batten's eye-bath.

(*b*) That any alteration in the shape of the anterior surface of the vitreous humour would produce any accommodative effect is unlikely; seeing that the refractive index of the aqueous and vitreous humours are nearly the same.

(5) The contraction of the boy's freely acting pupils certainly cuts off some circles of diffusion, and by so doing must play some part in the production of his apparent power of accommodation. That it is not, however, the sole cause is shown by his being able to still read J. 1 with his pupils dilated with atropine when sufficient light is concentrated on the test type. Also J. 1 with the pupil dilated with cocaine in ordinary daylight.

The conclusion which I have arrived at from my investigation of this case is in keeping with the views expressed by Donders, Davis, and Rogman, on this subject. That the boy does not possess any real accommodative power and that his ability to see clearly at different distances is due to the cutting off of diffusion circles, partly by the action of his freely movable circular pupil, but mainly by the sharply defined central openings which he has in his lens capsules.

The opening in the capsule of the right eye is smaller, more circular, and has the edge more sharply defined than that in the left eye. It is the right eye which has the best vision and can see J. 1 best with a dilated pupil. When the pupil is dilated in the left eye it is probably at a disadvantage, as compared with the right, in that light can enter round the periphery of the capsule, which does not fill the entire pupillary area. The greater ease with

which the reading is accomplished under bright illumination is in keeping with the central holes in the capsules acting as stenopaic apertures.

Up to the present I have been well satisfied when I have succeeded, after a cataract operation, in getting a patient to see $\frac{6}{6}$ and J. 1 with two pairs of glasses. In the light of this case we must recognise that there is still a higher degree of perfection attainable, viz., for the patient to be able to see $\frac{6}{6}$ and J. 1 with one pair of glasses. To be able to do this is, it is needless to say, of great advantage to a patient. We may well seek to find out in what class of cases its attainment may be possible, and what form of technique is best calculated to bring it about. I would suggest that the complete removal of the nucleus of the lens and the retention of a complete ring of its cortex, can be more easily accomplished by the discission of soft cataracts than by the extraction of hard cataracts. Further, that the repeated discissions, as performed in this case, and allowing the lens matter to dissolve gradually is more likely to result in a sharply defined central opening in the membrane, and a freely acting pupil, than the more rapid procedure of curette evacuation.

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(13) A. Edward Davis.—*Manhattan Eye and Ear Hospital Reports*, January, 1895.
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Mr. Ernest Clarke said this was a very interesting case, and there could be no question that Mr. Collins's explanation of it was the true one. It was the principle of pin-hole photography. Ophthalmic surgeons had never disproved Donders's statement that there was "no accommodation in aphakia." Those who were presbyopes knew that if they had to look at small print they could read it without a plus lens through a small hole in a card. With 3 D. of presbyopia Snellen 0·25 could be read. The practice, sometimes adopted by old people, of reading with the candle placed between their eyes and the book was an example of the advantageous aid to accommodation by miosis. Donders referred to the fact that senile miosis was a very important factor in accommodation in myopes, for the latter often obtained increased acuity of vision as they became older, and did not want the same reduction of their minus glasses as would be expected. The lesson was that the surgeon should make his openings in the capsule, certainly in children with lamellar cataract, as small as possible. For many years he had done that, and with most excellent results. But he had never had such a good result as obtained in Mr. Collins's case. Still, he got them to do with one pair of glasses for general use, only putting on stronger ones when they wished to do fine work. This case taught that even in senile cataract the opening should be made as small as possible. Another lesson which this case inculcated was the importance and the value of a small pupillary opening in refraction work, which he did not think was sufficiently recognised. For

this reason it was always wise to make a post-cycloplegic examination before prescribing glasses.

Mr. Bishop Harman said he had had a case somewhat similar to that described by Mr. Collins—a boy whose cataract had been removed in the right eye so as to leave a very small central opening in a dense capsule. He could read the distance and near types with equal ease with the same glasses without moving them on his face. But there was one disability attached to this gain, for the boy had all the symptoms of "night blindness" in poor lights. The capsule being undilatable would not admit sufficient light into the eye. The drawings in Mr. Collins's case showed that the capsular pupil was larger in one eye, and there was a secondary opening, so possibly his patient had no trouble of this sort. In his own case the boy only had one effective eye. The occurrence of this symptom of night blindness with a small fixed capsular pupil, and without discoverable defect of the fundus, would appear to confirm the opinion that it was the pin-hole pupil that enabled these patients to see both far and near with the same glasses.

Mr. Gray Clegg said he believed he had had a case similar to that described by Treacher Collins. He also had a man who had 1·5 D. of hypermetropia. He was 55 years of age, and he could read $\frac{5}{4}$ and J. 1 with great ease and without any glasses. He had miosis from locomotor ataxy, and that seemed to tally with the pin-hole theory. Two pairs of glasses after cataract extraction could be dispensed with, even in cases in which there was the usual lack of accommodation, by the use of a bi-focal lens, which was of great service to people who could adapt themselves to it.

Mr. Rayner Batten said that some years ago he had three or four cases which had full distant vision and could read J. 1 with the same lens. Since then he had been in the habit of prescribing glasses in the first case only for distance in order to educate the patient. He had never been able to get any explanation of the phenomenon. In

his cases he did not think the pin-hole pupil was the cause; they got $\frac{6}{6}$ and J. 1 with the same glasses. He had never met with a case of senile cataract in which this happened.

Mr. J. B. Story said he thought members should be very much obliged to Mr. Treacher Collins for bringing forward this interesting case, and still more for the instructive paper which he had read on it. He thought it would be obvious to all that what Mr. Collins said was correct; that there was not, as Donders had declared, any accommodation in aphakia. And the lesson they might take to heart from this case, and from what had been said about it, was not that it was necessary or advisable to make very small holes in the capsule, but that the human eye had a much higher power of vision than the conventional standard. For a really sharp-sighted person, $\frac{6}{6}$ as a maximum acuity was absurd; as a young man he could himself reach $\frac{6}{4}$ quite easily, and more in a good light. Accurate accommodation is quite unnecessary to read J. 1, and much stricter tests are requisite to measure accommodation scientifically.

Dr. George Mackay said he could recall that Mr. Priestley Smith, at a meeting of this Society, drew attention to the equal difficulty of explaining some cases in which along with excellent distant vision an extraordinary capacity for reading print close at hand was retained by presbyopes, and had suggested that this might be due to some part of the lens possessing a refractive value different from the centre or some other part of the lens. The speaker wished to throw out the suggestion that if Mr. Collins's report led others to investigate their cases with more care, there was another method of testing accommodation with simple apparatus which Mr. Collins said he had not tried in his case, namely, the application of retinoscopy, which was most useful in determining whether accommodative power was present or not.

Mr. J. A. Wilson said he had seen a case very similar to Mr. Collins's, in which, with small pupils, $\frac{6}{6}$ and good near vision were obtained.

Mr. Leighton Davies remarked, in support of Mr. Collins's contention, that it was not very infrequent to find after cataract extraction that the patient could read for simple types, $\frac{6}{18}$ easily. He remembered two patients who could read print on the placards, although not small Jaeger type. One of his patients told him he much preferred to go about without a high cataract lens; he wanted glasses for reading only.

Mr. E. Treacher Collins replied that he had seen a patient of presbyopic age who could read J. 1 without glasses which would have been appropriate for his age, and in that case there was extreme miosis. He had not asked the boy, the subject of his paper, whether he had difficulty in seeing at night, but the lad had not made any such complaint. He could understand that if there were the same small openings in the capsule in both eyes, it might lead to limitation of vision in dim lights. He agreed with Mr. Story that acuity of vision was often greater than $\frac{6}{6}$. He considered that under normal conditions there was great variation in acuity of vision in different individuals ranging from $\frac{6}{4}$ to what would be called amblyopia He expected that this boy had remarkably good acuity of vision.

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2. *On dissolving senile cataract in the early stages.*

By W. B. Inglis Pollock.

Introduction.

A considerable amount of work has been done during the last fifteen years upon the medical or abortive treatment of senile cataract. Badal (1) in 1901 advised the employment of potassium iodide in lotion and drops for the eyes. His results, published in 1906, were very satisfactory. Verdereau (2), in 1904, reported a series of

successful results with the subconjunctival administration of the same drug. De Wecker (3) and others have also obtained good results with the same methods; while Marques (4) and several authors stated that they were unsuccessful. An important paper by v. Pflugk (5), in 1908, summarises the work to that date. His own results with subconjunctival injections of potassium iodide were very successful.

Bernard le Roy (6), in 1909, appears to have been the first to use sodium thiosinamate in the absorption of senile cataract. Römer (7), in 1910, reported the result of his organo-therapeutic experiments with the oral administration of lens albumen. A slight improvement was seen in visual acuity in some cases.

Dor (8), in 1911, laid great stress upon the use of nascent calcium iodide in solution, the bath to be kept upon the eye for thirty minutes. Later in the same year Dransart (9) reported good results with Badal's method, supplemented by general treatment, high frequency currents, and baths.

Colonel Smith (10), working in India, in 1912 advocated the single subconjunctival injection of cyanide of mercury. The procedure is painful, and may require chloroform. Harry (11) confirmed Smith's results, but considered that they were not permanent. Chevallereau (12) and Dalencourt (13), 1913, confirmed the work already done.

Method of Treatment.

During the last ten years I have been attempting to dissolve cataract in the early stages. The idea occurred to me while clearing corneal nebulæ by means of drugs. The first attempts were purely experimental, but the results were so hopeful that I was encouraged to persevere. Eye-baths of potassium iodide and sodium chloride in strong solution were first employed. Dionine, fibrolysine, and iodolysine were added later as eye drops. The patient is directed to keep the eye-bath applied for five

minutes, thrice daily, followed by the instillation of the drops.

Each drug is exhibited first in weak solution, and when the eyes become accustomed to the treatment the strength of the drug is gradually increased, until a stage is reached when a stronger solution would produce too great a reaction. The drug is then changed, and another of the series substituted. If one drug is found to be more effective in any given case it is continued for a longer period in weaker and stronger solutions alternatively, in order to obtain an intensive effect.

Attention must be paid to the general health. Bright's disease, diabetes, or other trouble should receive appropriate treatment. In nearly all other cases I recommend the internal administration of potassium or sodium acetate or citrate.

I have treated nearly two hundred patients by these methods, but a number of these were under observation for too short a period for statistical purposes. One hundred consecutive cases, none of which had been under my care for less than three months, have been collated from the records of my private practice for the purpose of this paper. Many of the patients have been under observation for a period of some years, reaching in one instance to seven years.

In the table the visual acuity is given for each case on the dates of the first and last consultation, and also for many cases at certain intervening periods. Correcting lenses were worn if necessary during the testing of the vision. A column has been added for the refraction, but the measurements have not been given. Each case occupies two lines—the upper for the right eye and the lower for the left. Complications, the period of observation, and relevant information have been given in the last three columns.

No.	Case.	Sex.	Age.	First visit.	Vision.	Date.	Vision.	Date.	Vision.	Date.	Vision.	Refraction.	Complications.	Period under observation. Yrs.	Mths.	Remarks.
1	Mrs. F—	F.	48	9 : 9 : '06	6/6	30 : 5 : '11	6/9	20 : 7 : '12	6/36	15 : 10 : '12	6/9	H.	—	6	3	
					6/9		6/12		6/18			,,				
2	Mrs. W—	,,	75	16 : 1 : '07	6/60	18 : 5 : '07	6/60	26 : 10 : '09	3/60	—	—	M.	—	2	9	Died, 1910, hemiplegia.
					6/24		6/12		6/18			,,				
3	H. B—	M.	51	16 : 12 : '07	6/6	27 : 8 : '10	6/6	6 : 9 : '11	6/6	—	—	,,	—	3	10	Died, 1913, appendicitis.
					,,		6/9		,,			,,				
4	Mrs. B—	F.	56	22 : 1 : '08	,,	4 : 7 : '12	6/12	25 : 10 : '12	6/9	17 : 3 : '13	6/6	,,	—	5	2	Sister of Case 6.
					,,		6/18		6/12		6/9	,,				
5	Miss F—	,,	63	12 : 2 : '08	,,	30 : 1 : '11	,,	11 : 10 : '11	6/9	23 : 2 : '15	6/5	H.	—	7	—	
					6/12		6/24		,,		,,	,,				
6	Miss J—	,,	59	20 : 3 : '08	6/6	15 : 5 : '08	6/9	24 : 6 : '12	,,	12 : 9 : '12	6/12	M.	—	4	6	Sister of Case 4.
					6/12		,,		6/36		6/9	,,				
7	T. H. B—	M.	54	19 : 5 : '08	6/6	19 : 6 : '13	6/18	30 : 6 : '13	6/24	—	—	,,	—	5	1	
					,,		6/12		6/12			,,				
8	Miss R—	F.	64	6 : 8 : '08	4/60	14 : 7 : '09	6/12	2 : 7 : '12	6/9	23 ; 6 : '14	—	H.	Mature cataract	5	10	Extr. cataract, R., successful.
					6/24		6/60					,,				
9	Dr. S—	M.	58	3 : 4 : '09	6/6	19 : 4 : '12	6/12	15 : 11 : '13	6/9	25 : 4 : '14	6/9	,,	Thrombosis, L., retinal vein	5	—	
					1/60		6/60		6/18			,,				
10	J. C—	,,	78	8 : 9 : '09	2/60	16 : 10 : '09	2/60	15 : 12 : '09	4/60	—	4/60	,,	Cataract almost mature	—	3	
					6/18		6/12		6/9			E.	—	1	—	Died 1911.
11	J. D—	,,	70	29 : 9 : '09	6/9	24 : 11 : '09	6/6	16 : 8 : '10	6/6	—	—	,,				
					P.L.		P.L.		P.L.			,,	Glaucoma absolutum			
12	J. S—		55	20 : 10 : '09	6/9	14 : 10 : '13	6/12	13 : 12 : '13	6/6	—	—	H.	—	4	2	
					,,		6/18		6/9			,,				
13	Mr. M—	,,	80	24 : 11 : '09	6/6	9 : 3 : '10	6/6	12 : 4 : '10	6/6	—	—	,,	—	—	5	Extr. cataract, L., successful.
					P.L.								Mature cataract			
14	Mrs. G—	F.	39	1 : 12 : '09	6/6	8 : 9 : '14	6/9	24 : 11 : '14	6/6	—	—	M.	—	5	—	
					,,		,,		,,			,,				

15	Mrs. McD—	F.	52	15 : 12 : '09	$\frac{6}{18}$	9 : 2 : '10	$\frac{6}{9}$	13 : 10 : '10	$\frac{6}{6}$	—	—	M.	—	—	10			
16	Miss H—	„	76	22 : 1 : '10	$\frac{6}{12}$ / $\frac{6}{9}$	26 : 10 : '11	$\frac{6}{6}$ / $\frac{6}{36}$	2 : 4 : '14	„ / $\frac{6}{18}$	9 : 1 : '15	$\frac{6}{18}$	„ / „	Chronic glaucoma	5	—			
17	Mrs. O—	„	53	26 : 1 : '10	„	16 : 3 : '10	$\frac{6}{18}$ / $\frac{6}{6}$	28 : 6 : '10	$\frac{6}{36}$ / $\frac{6}{6}$	—	—	„ / H.	—	—	5			
18	Mrs. Y—	„	47	9 : 2 : '10	„ / $\frac{6}{18}$	10 : 5 : '10	„ / $\frac{6}{9}$	13 : 2 : '14	„ / $\frac{6}{12}$	25 : 8 : '14	$\frac{6}{9}$	„ / „	—	4	6			
19	Mrs. M—	„	54	30 : 3 : '10	„ / $\frac{6}{24}$	7 : 7 : '10	„ / $\frac{6}{12}$	15 : 11 : '10	$\frac{6}{18}$ / $\frac{6}{12}$	—	—	„ / M.	—	—	8			
20	Mrs. B—	„	55	7 : 4 : '10	„ / $\frac{6}{12}$	15 : 6 : '10	„ / $\frac{6}{6}$	21 : 12 : '10	$\frac{6}{18}$ / $\frac{6}{6}$	—	—	„ / „	—	—	8			
21	Mrs. C—	„	68	24 : 6 : '10	„ / $\frac{6}{18}$	8 : 9 : '11	$\frac{6}{9}$ / „	29 : 1 : '15	$\frac{6}{9}$ / $\frac{6}{24}$	13 : 4 : '15	$\frac{6}{12}$	„ / E.	—	4	10			
22	J. W. L—	M.	48	17 : 8 : '10	$\frac{6}{9}$ / $\frac{6}{60}$	6 : 8 : '14	$\frac{6}{6}$ / $\frac{6}{60}$	12 : 8 : '14	$\frac{6}{12}$ / $\frac{6}{36}$	—	—	„ / M.	—	2	—			
23	Mrs. C—	F.	78	17 : 10 : '10	$\frac{6}{12}$ P.L.	23 : 1 : '14	$\frac{6}{24}$ / —	15 : 5 : '14	$\frac{6}{18}$ / —	16 : 4 : '15	—	„ / E.	Retinal separation	4	6			
24	Mrs. Y—	„	70	30 : 11 : '10	$\frac{6}{36}$ P.L.	13 : 3 : '11	$\frac{6}{60}$ / —	22 : 12 : '12	$\frac{3}{24}$ / —	24 : 4 : '14	$\frac{3}{18}$ / —	„ / M.	Iris bombé	3	5			
25	J. M—	M.	72	30 : 1 : '11	$\frac{6}{60}$ / $\frac{6}{60}$	25 : 4 : '11	$\frac{6}{24}$ / „	—	—	—	$\frac{6}{24}$	H.	—	—	4			
26	Mrs. M—	F.	68	4 : 4 : '11	$\frac{6}{24}$ / $\frac{1}{60}$	10 : 5 : '12	$\frac{6}{12}$ / —	3 : 6 : '13	—	21 : 4 : '14	—	M.	Mature cataract	3	—	Extr. followed by choroidal [hæmorrhage.		
27	Miss McK—	„	55	11 : 4 : '11	$\frac{6}{24}$ / $\frac{6}{18}$	27 : 9 : '11	$\frac{6}{18}$ / $\frac{6}{9}$	—	$\frac{3}{60}$ / —	—	$\frac{3}{60}$	„ / H.	—	—	5½			
28	J. S. I—	M.	73	12 : 5 : '11	$\frac{6}{9}$ / $\frac{1}{60}$	30 : 6 : '11	$\frac{6}{6}$ / $\frac{6}{60}$	17 : 11 : '11	P.L.	—	—	„ / „	Mature cataract	—	6			
29	Miss C—	F.	55	16 : 5 : '11	$\frac{6}{24}$ / $\frac{6}{9}$	26 : 2 : '15	$\frac{6}{18}$ / $\frac{6}{60}$	9 : 4 : '15	$\frac{6}{12}$ / $\frac{6}{24}$	—	—	„ / „	—	3	11			
30	Mrs. L. M—	„	71	26 : 5 : '11	$\frac{6}{9}$ / $\frac{6}{9}$	19 : 10 : '11	„ / $\frac{6}{9}$	18 : 12 : '14	$\frac{6}{24}$ / $\frac{6}{24}$	—	—	„ / „	—	3	7	Died, 1915, before treatment [recommenced.		
31	Mrs. A—	„	61	17 : 6 : '11	„ / $\frac{6}{18}$	21 : 7 : '11	„ / $\frac{6}{18}$	13 : 11 : '11	$\frac{6}{36}$ / $\frac{6}{9}$	—	—	„ / „	—	—	5			
32	Miss D—	„	50	29 : 8 : '11	„ / $\frac{6}{12}$	19 : 10 : '11	„ / —	14 : 11 : '11	$\frac{6}{12}$ / —	23 : 12 : '13	—	M. / —	Atrophy macula	2	4			
33	G. M. R—	M.	34	31 : 8 : '11	$\frac{6}{24}$ / $\frac{6}{9}$	17 : 11 : '11	$\frac{6}{18}$ / $\frac{6}{6}$	—	$\frac{6}{9}$ / —	—	$\frac{6}{9}$	M. / H.	—	—	3			

No.	Case.	Sex.	Age.	First visit	Vision.	Date.	Vision.	Date.	Vision.	Date.	Vision.	Refraction.	Complications.	Period under observation. Yrs.	Mths.	Remarks.
34	Mrs. E—	F.	54	16:10:'11	$\frac{6}{12}$	1: 2:'12	$\frac{6}{9}$	28: 1:'13	$\frac{6}{9}$	4: 9:'13	$\frac{6}{9}$	M.	—	2	—	
35	W. C—	M.	49	17:10:'11	$\frac{6}{9}$ $\frac{6}{12}$	19:12:'11	$\frac{6}{6}$ $\frac{6}{9}$	11: 2:'13	$\frac{6}{12}$ $\frac{6}{6}$	9: 9:'13	$\frac{6}{6}$	H.	—	1	11	
36	J. T. D—	,,	76	27:10:'11	$\frac{6}{9}$,,	15:12:'11	$\frac{6}{6}$,,	20: 8:'12	,,	20:12:'14	,,	,,	—	3	2	
37	Mrs. P—	F.	63	27:10:'11	$\frac{6}{18}$ $\frac{6}{9}$	18:12:'11	$\frac{6}{9}$,,	25 4:'12	$\frac{6}{12}$ $\frac{6}{9}$	10:10:'12	$\frac{6}{9}$,,	M.	—	1	—	
38	J. S. T—	M	29	21:12:'11	,, $\frac{6}{18}$	11: 4:'12	$\frac{6}{12}$,,	8: 1:'13	,, $\frac{6}{18}$	16: 4:'13	,, $\frac{6}{12}$	,,	—	1	4	
39	W. F. S—	,,	45	6: 1:'12	$\frac{6}{12}$,,	13: 4:'12	$\frac{6}{9}$,,	4: 7:'12	$\frac{6}{9}$ $\frac{6}{6}$	—	—	,, H.	—	—	6	
40	A. H—	F.	62	23: 3:'12	$\frac{6}{9}$ P.L.	9: 7:'12	$\frac{6}{6}$ —	8: 6:'14	,, —	—	—	—	Mature cataract	2	3	
41	J. K. H—	M.	44	1: 4:'12	$\frac{6}{18}$ $\frac{6}{9}$	9:12:'12	$\frac{6}{18}$ $\frac{6}{6}$	—	$\frac{6}{12}$ —	--	—	H. M	—	—	8	
42	Mrs. W—	F.	71	22: 4:'12	,, $\frac{6}{18}$	25:12:'13	,, $\frac{6}{18}$	4:11:'14	$\frac{6}{18}$	—	—	,, ,,	—	2	7	
43	Mrs. C—	,,	58	26: 6:'12	$\frac{6}{24}$ $\frac{6}{9}$	4:10:'12	,, $\frac{6}{6}$	—	,, —	—	—	,, ,,	—	—	$3\frac{1}{2}$	
44	Capt. R—	M.	74	1: 7:'12	$\frac{6}{12}$ P.L.	24:10:'12	$\frac{6}{9}$ —	15: 8:'13	—	4: 1:'15	—	,, H.	Thrombosis, retinal vein	2	6	
45	Mrs. G—	F.	52	2:10:'12	$\frac{6}{36}$ $\frac{6}{9}$	18: 9:'13	$\frac{6}{18}$ $\frac{6}{6}$	9:11:'14	$\frac{6}{24}$ $\frac{6}{6}$	15: 4:'15	$\frac{6}{18}$ $\frac{6}{6}$	,, M.	—	2	5	
46	J. A—	M.	58	3:12:'12	$\frac{6}{12}$ $\frac{6}{24}$	11: 2:'13	$\frac{6}{9}$ $\frac{6}{18}$	24: 6:'13	,, $\frac{6}{12}$	—	—	,, H.	Retinitis pigmentosa	—	$6\frac{1}{2}$	
47	T. B—	,,	74	25: 1:'13	$\frac{6}{12}$ $\frac{6}{24}$	31: 3:'13	$\frac{6}{60}$ $\frac{6}{18}$	8: 9:'13	$\frac{6}{36}$ $\frac{6}{9}$	5:12:'14	$\frac{6}{9}$	,, ,,	—	1	10	
48	Mrs. G—	F.	58	17: 2:'13	$\frac{6}{36}$ P.L. $\frac{6}{24}$	2: 6:'14	$\frac{6}{24}$ — $\frac{6}{18}$	17: 9:'14	$\frac{6}{18}$ — $\frac{6}{18}$	25: 3:'15	$\frac{6}{18}$ — $\frac{6}{36}$	,, — M.	Mature cataract, R.	2	1	

49	Mrs. M. B—	F.	53	4: 3:'13	6/36	4: 4:'13	6/24	13: 5:'13	6/18	—	—	H.	—	—	2½		
50	Mrs. R—	„	44	25: 3:'13	6/60	1: 7:'13	6/36	3:10:'13	6/60	—	—	M.	Iris bombé	—	7		
51	Mrs. L—	„	63	19: 4:'13	6/24	13: 9:'13	6/18	14: 2:'14	6/18	—	—	„	—	—	10		
52	F. G—	M.	58	6: 5:'13	6/12	9: 6:'13	6/9	5: 8:'13	6/12	—	—	„					
53	W. N—	„	72	3: 6:'13	6/60	27:10:'14	6/36	—	6/60	—	—	E.	Retino-choroiditis	—	3		
54	Miss A—	F.	58	5: 6:'13	6/12	16: 9:'13	6/36	6: 4:'14	6/24	—	—	H.	—	1	4		
55	Mrs. M—	„	74	17: 6:'13	6/9	19: 8:'13	6/6	28:10:'13	6/6	—	—	„	—	—	10		
56	Mrs. B—	„	54	24: 6:'13	6/18	9:12:'13	6/12	16:10:'14	6/9	—	—	„	—	—	4		
57	Miss E—	„	55	8: 8:'13	6/24	20:11:'14	6/18	25: 4:'14	6/36	—	—	M.	—	1	4		
58	Mrs. L—	„	78	12: 8:'13	6/9	3:12:'13	6/6	22: 6:'14	6/6	—	—	H.	—	—	8		
59	Mrs. B—	„	69	30: 9:'13	6/18	5:12:'13	6/12	13: 3:'14	6/18	19: 4:'15	6/36	M.	—	1	8	Sister of Case 60	
60	Mrs. L—	„	73	11:10:'13	6/24	11:12:'13	6/9	13: 6:'14	6/12	16: 6:'14	6/9	H. M. H.	—	—	9		
61	Mrs. B—	„	62	14:10:'13	6/9	17: 2:'14	6/18	18: 8:'14	6/6	—	—	„	—	—	8	Died 22:11:'14, abdominal tumour. Sister of Case 58.	
62	Miss W—	„	72	22:10:'13	4/60	4: 5:'14	4/24	14:10:'14	3/24	—	—	„	—	—	10		
63	Miss S—	„	76	4:11:'13	3/60	24: 3:'14	3/18	10:12:'14	4/18	—	—	M.	—	1	—		
64	Mrs. M—	„	81	4:11:'13	6/24	9: 1:'14	6/18	1: 5:'14	6/12	12: 4:'13	6/12	„	—	1	5		
65	Mrs. S—	„	72	15:11:'13	6/12	16: 3:'14	6/18	28:11:'14	6/18	23:10:'14	6/6	H.	—	1	—		
66	Mrs. G—	„	46	26:11:'13	6/24	6: 4:'14	6/60	26:12:'14	6/24	—	—	M.	Glaucoma	1	1		
67	Miss C—	„	64	9:12:'13	P.L. 6/18	3: 6:'14	P.L. 6/18	11:11:'14	P.L. 6/18	—	—	H.	Mature cataract	—	11		

No.	Case.	Sex.	Age.	First visit.	Vision.	Date.	Vision.	Date.	Vision.	Date.	Vision.	Refraction.	Complications.	Period under observation. Yrs.	Mths.	Remarks.
68	Mrs. C—	F.	64	6: 1:'14	$\frac{6}{24}$	10: 3:'14	$\frac{6}{12}$	23:10:'14	$\frac{6}{12}$	—	—	H.	—	—	$9\frac{1}{2}$	
69	Miss P—	,,	51	17: 1:'14	,, $\frac{6}{18}$	26: 4:'14	,, $\frac{6}{18}$	21: 8:'14	,, $\frac{6}{18}$	—	—	,,	—	—	7	
70	Mrs. A—	,,	50	19: 1:'14	$\frac{6}{36}$ $\frac{6}{9}$	3:11:'14	$\frac{6}{60}$ $\frac{6}{6}$	27: 2:'15	$\frac{6}{36}$ $\frac{6}{6}$	—	—	,, ,,	—	1	1	
71	Mrs. R—	,,	70	20: 1:'14	,, $\frac{6}{60}$	17: 2:'14	,, $\frac{6}{36}$	8: 5:'14	,, $\frac{6}{36}$	—	—	M.	—	—	4	
72	Mrs. M—	,,	69	2: 2:'14	,, $\frac{6}{6}$	9:10:'14	$\frac{6}{18}$ $\frac{6}{5}$	—	$\frac{6}{12}$ —	—	—	,, H.	—	—	8	
73	Mrs. C—	,,	60	3: 2:'14	,, $\frac{6}{18}$	3: 2:'15	$\frac{6}{18}$ $\frac{6}{12}$	—	—	—	—	,, M.	—	1	—	
74	Miss R—	,,	71	13: 2:'14	,, $\frac{6}{24}$ $\frac{4}{60}$	27: 3:'14	,, $\frac{5}{60}$	7: 8:'13	$\frac{6}{9}$ $\frac{5}{24}$	—	—	,, H.	—	—	6	
75	J. D. P—	M.	60	27: 2:'14	$\frac{6}{12}$ $\frac{6}{18}$	22: 5:'14	$\frac{6}{6}$ $\frac{6}{18}$	24:11:'14	$\frac{6}{12}$ $\frac{6}{18}$	30: 3:'15	$\frac{6}{6}$ $\frac{6}{18}$	,, M.	Nearly mature —	1	1	
76	Miss G—	F.	71	3: 3:'14	$\frac{6}{24}$ $\frac{6}{36}$	7: 4:'14	$\frac{6}{12}$	13;10:'14	$\frac{6}{12}$	—	—	,, H.	—	—	7	
77	Mrs. G—	,,	52	20: 3:'14	,, $\frac{6}{24}$	16: 6:'14	,, $\frac{6}{18}$ $\frac{6}{9}$	13:10:'14	$\frac{6}{12}$	—	—	,, ,,	—	—	7	
78	Mrs. McF—	,,	50	3: 4:'14	$\frac{6}{18}$	10: 6:'14	$\frac{6}{12}$	1:11:'14	$\frac{6}{6}$	19: 4:'15	$\frac{6}{6}$	,,	—	1	—	
79	J. E. B—	M.	45	20: 4:'14	$\frac{6}{24}$ $\frac{6}{60}$	18: 8:'14	,, $\frac{6}{24}$ $\frac{6}{60}$	8: 2:'15	$\frac{6}{9}$ $\frac{6}{24}$	—	—	,, ,, ,,	—	—	8	
80	Mrs. H—	F.	42	28: 4:'14	,, $\frac{6}{9}$	4:10:'14	$\frac{6}{6}$	15: 1:'15	,, $\frac{6}{6}$	—	—	,,	—	—	9	
81	Miss S—	,,	62	2: 5:'14	,, P.L.	12: 8:'14	,, —	10: 2:'15	,, —	—	—	M.	Mature cataract	—	9	
82	Miss L—	,,	65	5: 5:'14	$\frac{6}{24}$ $\frac{6}{36}$ $\frac{6}{24}$	3: 8:'14	$\frac{6}{18}$ $\frac{6}{24}$ $\frac{6}{18}$	8: 3:'15	,, $\frac{4}{60}$ $\frac{6}{18}$ $\frac{6}{12}$	—	—	H. ,, ,,	—	—	10	

83	Miss H—	F.	52	5 : 5 :'14	9	9 : 6 :'14	6	25 : 9 :'14	6	—	—	H.	—	—	$4\frac{1}{2}$	
84	J. S. McC—	M.	59	26 : 5 :'14	6	23 : 10 :'14	6	26 : 3 :'15	6	—	—	,,	—	—	10	
85	Mrs. H—	F.	57	2 : 6 :'14	0	11 : 8 :'14	—	3 : 11 :'14	—	—	—	—	—	—	5	Anophthalmos.
86	Mrs. L—	,,	64	23 : 6 :'14	6	27 : 10 :'14	6	2 : 2 :'15	6	—	—	M.	—	—	8	
87	Mr. H—	M.	56	25 : 6 :'14	6	13 : 10 :'14	6	1 : 12 :'14	6	—	—	H.	—	—	5	
88	Mrs. H—	F.	56	2 : 8 :'14	6	13 : 11 :'14	1	—	—	—	—	M	—	—	3	
89	Miss S—	,,	60	8 : 8 :'14	6	13 : 10 :'14	6	27 : 1 :'15	6	14 : 4 :'15	6	,,	—	—	8	Sister of Case 92.
90	Mrs. H—	,,	68	10 : 9 :'14	6	9 : 11 :'14	—	8 : 3 :'15	—	—	—	H.	Amblyopia, ext. anopsia	—	6	
91	Miss McK—	,,	50	18 : 9 :'14	6	19 : 1 :'15	6	26 : 3 :'15	6	—	—	M.	—	—	6	
92	W. S—	M.	73	30 : 9 :'14	6	9 : 11 :'14	6	25 : 12 :'14	6	—	—	E.		—	3	Brother of Case 89.
93	Mrs. B—	F.	70	6 : 10 :'14	P.L.	21 : 11 :'14	6	15 : 4 :'14	6	—	—	H.	Mature cataract	—	6	
94	A. McL—	M.	77	8 : 10 :'14	6	26 : 10 :'14	6	20 : 1 :'15	6	—	—	,,	—	—	3	
95	Mrs. S—	F.	83	15 : 10 :'14	6	16 : 1 :'15	6	19 : 4 :'15	6	—	—	M.	—	—	6	Treatm't stop'd after 3 months.
96	C. L—	M.	70	16 : 10 :'14	6	23 : 3 :'15	6	—	—	—	—	,,	—	—	5	
97	Mrs. S—	F.	64	17 : 11 :'14	1	30 : 3 :'15	3	—	—	—	—	,,	Atrophy macula	—	4	
98	J. B. S—	,,	43	20 : 11 :'14	6	19 : 2 :'15	6	12 : 4 :'15	6	—	—	H.	—	—	5	
99	Mrs. E—	,,	70	30 : 11 :'14	6	11 : 3 :'15	6	12 : 4 :'15	6	—	—	M.	—	—	$4\frac{1}{2}$	
100	Mrs. McC—	,,	70	15 : 12 :'14	6	9 : 2 :'15	6	30 : 3 :'15	6	—	—	H.	—	—	$3\frac{1}{2}$	

Discussion of Results.

In the case of ten patients one eye was already practically blind from some other cause; one patient had had an eye enucleated; and eleven patients had a mature or almost mature cataract in one eye. There were thus only 178 eyes available for review; and some were in the stage of immature cataract.

The opacities were examined with the + 20 D. lens behind the mirror of the ophthalmoscope, and careful drawings made at different stages. Different forms of senile cataract are included in the series.

The cortical form with peripheral striæ was most common, and yielded most easily to treatment. Sometimes the opacities disappeared within the first two or three weeks of commencing treatment, especially in patients of early middle age, but in most instances progress was much slower, and at times it required six or nine months or even longer for the cataract to entirely dissolve. In a considerable number of cases the striæ never entirely disappeared, but nevertheless normal vision was obtained, and suitable glasses ordered if necessary. In other instances an astonishing improvement in vision occurred, without any change being observed in the cataract itself.

A number of patients had the form of cataract with sand-like opacities in the lens, and improvement was obtained with more difficulty in these cases. A previous writer has also made a note of this point. The vision was not as a rule much diminished in this form of cataract; notwithstanding that, treatment was persevered with, and a partial absorption was reached.

Nuclear opacities always presented the greatest difficulties. There were only a few cases of this form of senile cataract. Case 80, J. E. B—, had 12 D. of myopia with 3 D. of astigmatism, and vision improved slightly only after a course of treatment extending to eight months.

Among the 178 eyes 79, 44 per cent., showed a great improvement; while in 72, 41 per cent., an improvement

was seen. A good result was therefore obtained in 85 per cent. In 12 cases, 7 per cent., the cataract did not become worse and the visual acuity remained stationary. In 15 eyes, 8 per cent., the treatment failed to prevent the cataract progressing, with a consequent deterioration of vision. In most of these patients the cataract was already fairly far advanced before treatment was instituted; while Case 30, Mrs. L. M—, died before the second course of treatment had been used for more than ten days, and she is only included in the series to show the relapse after three years from the first course of treatment. Some of the patients were seen before cataract developed.

The treatment lasts as a rule for from six to nine months; but sometimes a year or longer is required. When all opacities in the lenses have disappeared the patient is requested to return every six months for a year or two for observation, and a short course of treatment is often given. In other cases a return visit is advised every three months for treatment to prevent relapses. When a relapse occurs treatment is persevered with until the vision is brought up as far as possible. If glasses are necessary, they should not be prescribed until three months after the cataract has dissolved, unless for temporary purposes, because the state of the refraction usually continues to change for some time, even after treatment has been stopped.

This treatment does not cause pain or discomfort. There is, therefore, little difficulty in persuading the patients to persevere with it. The first use of dionine sometimes gives rise to alarm owing to the congestion of the eyelids; but a minimal dose (1 per cent.) should be prescribed and the patient warned of the action of the drug. The alkaline lotions may bring out a tendency to eczema. The eye bath must be discontinued, and the patient directed to instil the lotion as well as the drops into the eye. Zinc ointment or a similar application can be employed.

As regards the permanency of the results, some of the

cases have been under observation for a period of several years since all signs of cataract were present.

Case 5.—Miss F—, æt. 63 years, began treatment in February 1908, and the cataract disappeared by the end of the year. When last seen in February of the present year, for a rheumatic affection of the eyes, there was no trace of the cataract. She has, therefore, remained clear of recurrence for six years, during which I have examined her upon a number of occasions.

Case 8.—Miss R—, æt. 64 years, was under treatment in 1908–09 for the left eye, and the cataract was entirely dissolved. She returned in 1912, and in 1914, and there was no appearance of recurrence. This covers a period of almost five years.

Case 16.—Miss H—, æt. 76 years, came first for treatment in 1910 with incipient cataract and chronic glaucoma. When she returned at the end of 1911, the vision of the right eye was at its worst. All signs of lenticular opacity disappeared early in 1912, and there has been no recurrence. She was seen last in January of the present year, or a period of three years.

The question has been raised as to whether the results may have been due to spontaneous absorption of the cataract. Connor (14) collected the opinions of a large number of surgeons on this point, and found that fifty-one had observed it in 147 cases. Although the results may seem to be related, yet the percentage of improvement given by the writers already quoted and in my own series is far too high to be viewed in this light. There is no doubt from the rapidity with which the opacities disappeared in many cases that the absorption was due to the treatment.

Conclusions.

In a consecutive series of 100 patients with senile cataract, who were under treatment for not less than three months with alkaline lotions and dionine, fibrolysine, or iodolysine drops, 45 per cent. of 178 eyes showed a great

improvement, 41 per cent. an improvement, and 7 per cent. remained stationary. In 8 per cent. the treatment failed to arrest the progress of the cataract.

This method of treatment does not cause pain; and patients are easily encouraged to persevere with it for prolonged periods. Attention should be given to the general health. The internal administration of an alkaline mixture is also of advantage.

Several patients have remained clear of recurrence for a period of from three to six years.

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Mr. F. Richardson Cross said this paper gave some extraordinary statistics in regard to cure or improvement of a condition which is usually looked upon as irremediable. The author claimed most definite results by his treatment; not in a few of the cases here and there, but apparently they were nearly all improved, to some extent in 85 per cent., and greatly in 44 per cent. In only 8 per cent. of

the cases did "the treatment fail to prevent the cataract from progressing." No doubt we were in a transition stage in regard to all forms of scientific knowledge and in surgical enterprise, but to him the statements made appear to be quite incredible. The possibility of obtaining clearing in a case of cataract must be excessively rare. If it can be dissolved by such remedies as are suggested, or in any other way, why did we not occasionally see improvement take place under natural conditions. He did not know whether any members present had realised a cure or improvement in a few cases. Out of the enormous number of cases he must have seen, he could scarcely remember any where there was a distinct lessening of an ordinary senile cataract. He remembered one case which got a little better after an attack of typhoid fever. There appeared to have been some absorption and consequent improvement in vision, but he could not remember another case in which anything of the kind occurred. The author, however, seemed to have no doubt as to the efficacy of what he recommended. He understood him to say that some opacities disappear rapidly under a few weeks' treatment, but that others require a considerably longer time—the treatment is to be continued from six to nine months or a year. When all the opacities have disappeared the patient is to return every six months for observation. The author quoted cases in which there had been no recurrence for from three to six years. Nuclear opacities presented the greatest difficulty, but cortical forms with peripheral striæ were the more common and were quite amenable to treatment. In cases where the striæ did not disappear there was said to be an astonishing improvement in vision, normal vision being obtained in many cases. We all knew that there were plenty of cases of peripheral lenticular opacities that remained stationary, where vision was and remained normal for many years; but the striæ or spots remained quite evident throughout. Moreover, the speaker had been not infrequently consulted by patients from London and else-

where who were anxious to know if a cure had really taken place or if any cataract still existed after they had been under treatment by unqualified persons who were believed by the public to possess cures for cataract. In these patients cataractous changes of greater or less degree were present, and in no case had he seen any reason to suppose that the treatment had really improved the sight or altered the condition in any way. It would be a serious thing if the public were to imagine that cataract could be cured by absorption unless at any rate a very definite improvement could be brought about by this means. If it could be done Dr. Pollock deserved great credit for drawing attention to the value of such form of treatment, which is simple, and can be easily applied by those who may hope to verify the results he claims. Dr. Pollock had presented his paper in all modesty, and he (Mr. Cross) desired to make his remarks in the same spirit. He only hoped that the statistics brought forward might be supported by the experience of others; but he felt it his duty to make these comments lest the statements made in the paper should go out to the public unchallenged, and as if they were accepted by the ophthalmic surgeons present at the meeting.

Mr. Rayner Batten said he remembered a case of opacities in the lens which got well. The patient was a child, æt. 9 years, with serous iritis, K.P. and vitreous opacities. There were definite lens changes. Under treatment not only did the iritis and vitreous opacities disappear, but the lens changes also cleared up.

Mr. J. B. Story said he confessed himself a sceptic regarding this method of bringing about absorption of cataracts by medicinal or chemical treatment.

Mr. W. H. H. Jessop said it might be remembered by members that last year he read a paper before the Society on arcus senilis lentis, in which he mentioned that cases had been known to improve in regard to vision and described many in which the cortical striæ remained for years without any perceptible change. He did not under-

stand what Mr. Pollock meant in his paper by "solution." What did he dissolve?

Mr. Gray Clegg said that if, as was stated by Louis Dor, in 80 per cent. of cases whose vision was reduced to half the normal, the cataract was prevented from reaching maturity, it was a method worth trying. He had tried the method on private cases only. He could not give a very definite statement as to whether it was useful; but certainly some of the cases had not progressed since he used the treatment with a solution of calcium chloride and sodium iodide applied to the eye for half an hour daily. The method of Dr. Inglis Pollock was simpler, and if it was more efficacious he would renew his experiments. His own cases embraced every form. Louis Dor's observation referred to all types of cataract. With regard to spontaneous absorption of cataract, he remembered a child who was brought to him with a general diffuse haze of one lens, which improved very much for a time without treatment, but later the condition returned, and he had to remove the cataract by discission.

Mr. J. A. Wilson thought members should not criticise results of a method until they had tried it consistently. He had not himself much experience of this method. He would like Dr. Pollock to tell the meeting what change in refraction occurred in these cases after the treatment.

Dr. Inglis Pollock, in reply, said he did not measure the refraction if possible until treatment was finished; he tested vision with glasses if the patient wore a distance correction; but otherwise without glasses. In the case of an old lady, æt. 75 years, she had $\frac{6}{18}$ in one eye, and $\frac{6}{24}$ in the other. A fortnight later it was $\frac{6}{18}$ in both. In another fortnight it was $\frac{6}{9}$ and $\frac{6}{18}$. This month (April) she had $\frac{6}{9}$ and $\frac{6}{12}$, without glasses, with the electric light over the test-types, at the same distance in each testing. In her case there was no change apparent in the lens. Dor had suggested that in the lens, besides the opacities we knew of, there were fluids and fibres which were beginning to degenerate. Referring to the question of

"dissolving," some preferred to call it absorption; he believed it was the opacity being dissolved, because the opacity could be seen. He brought the paper forward so that every member could try the method. It was not fair to bring patients for a treatment when their condition was too advanced to give any hope of success without operation. But the treatment must be persevered in; if there was no improvement in six months, it must be persisted in as long as two years; some had been coming many months for a repetition of the treatment. Those who got better within two months were not included in the statistics he had presented. A considerable number of the cases were lost sight of.

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3. *A method of artificial maturation of cataract allowing of early extraction.*

By J. Gray Clegg.

When one is consulted by a patient found to be suffering from cataract, many problems present themselves for solution.

(1) Are one or both lenses affected?

(2) Will the cataract progress slowly or rapidly?

(3) Does the diminution in vision detract from the capacity for earning a livelihood?

(4) Is the temperament of the patient such as would enable him patiently to endure the waiting for natural maturation?

If the diminution in sight is fairly equal in the two eyes, or if the vision of the better one is not sufficient to enable the patient to pursue the tenor of his way with ease and comfort, and if the cataract in the more affected eye is not fully mature, then further questions have to be solved.

(*a*) Is it safe and wise to remove an immature cataract by the ordinary method, or

(*b*) Can one bring oneself to proceed to removal of the lens in its capsule with such confidence as to enable one to give an excellent prognosis ?

(*c*) Can any other course be pursued ?

Many years ago Förster introduced the operation of the preliminary iridectomy, accompanied by massage of the lens. Other surgeons such as Sæmisch, of Bonn, invariably performed an iridectomy preliminary to every cataract extraction.

During my time as house surgeon and assistant surgeon I often saw the operation of preliminary iridectomy with indirect massage performed, but I first witnessed massage applied directly to the lens capsule when in Berlin, fourteen years ago, by von Michel. I formerly was devoted to extraction without iridectomy, and still in many selected cases prefer the operation.

Now I consider it a waste of time for the patient to have a large section of the cornea done above in order to perform iridectomy and then massage the lens. For it does not appear to me to be reasonable to make another section of the cornea for extraction of the lens until after the lapse of at least three months, and I have therefore adopted the following procedure—for which I do not claim any originality—which has the three advantages of allowing an extraction to be made in the ordinary way, even after only the lapse of one week, of allowing the question as to whether an iridectomy shall be done at the extraction to remain over until the actual extraction itself takes place, and of leaving as little cortex for subsequent absorption as possible.

The procedure is as follows : The pupil is dilated with atropin for one or two days before the operation. A paracentesis of the anterior chamber is made by an iris knife at the other side. The aqueous is allowed to escape, an iris repositor is passed into the anterior chamber, and massage is applied directly to the lens capsule by it, taking care, however, that the capsule itself is not ruptured. Some twenty strokings of the lens capsule are made.

After this, further massage is made with the smooth lens scoop applied to the outer surface of the cornea. The movements are made in a rapid way radially from the centre and backwards. One drop of atropin is instilled and a bandage applied. During the same day atropin solution is instilled again once, twice, or thrice, according to the size of the pupil, and if the next morning the eye is perfectly quiet, no further atropin need be used.

Very commonly within two or three days definite opacification of the cortical layers of the lens is observable and in several cases extraction has been proceeded with on the seventh day after the first operation.

Extraction should not be resorted to, however, until the pupil has recovered its mobility, and is again of the ordinary size, and unless there is absolutely no sign of irritability the result of the massage operation. This method allows a business man who is becoming incapacitated for his work, to have maturation and extraction done and recovery to take place within the shortest possible space of time. Under the most favourable circumstances he need only be confined to the nursing home or hospital for three or three and a half weeks.

The advantage of having the cortical layers of the cataract opaque at the time of the extraction is obvious, in that the cortex is visible and soft, and thus can be more completely expressed.

The less lens substance left behind the better, for it may set up iritis, and certainly a good visual result is more rapidly obtained if it is away.

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4. *The capsular opacities of Morgagnian cataract.*

By Lieut.-Col. H. Herbert.

(With Plates XVI and XVII.)

The following pathological changes in lens capsule have not, to my knowledge, been shown hitherto. The

photo-micrographs are of the capsule of a very long-standing cataractous lens, which had advanced beyond the Morgagnian stage. It was one of the rare cases in which, at over 50 years of age, the nucleus entirely disappears, leaving milky fluid only enclosed in the capsule. The latter was dotted with the small white opacities frequently found in Morgagnian cataracts, and the whole was tremulous.

Pl. XVI, fig. 1 shows a section through two of the opaque capsular spots. Rounded spaces enclosed between layers of thickened capsule are filled with fluid and granular *débris*, quite like the neighbouring lens *débris*, except that many of the particles are stained (with hæmatoxylin and orcein) rather deeply. There are no cells of any kind in these or other similar cavities. Flattened elongated lens-cells are seen in one or more layers behind the posterior layer of membrane. At each end of a space the two layers of capsule join together, and the impression is given that the cavity results from separation of layers previously in contact, the space filling up mainly with the milky fluid of the cataractous lens. This impression is confirmed by examination of some small localized thickenings showing cavities in process of formation, apparently through degenerative changes in the capsule. From clear or relatively clear centres there may be a gradual transition to the firm (dark) periphery (see Pl. XVI, fig. 2). Other small spaces are filled with loose particles staining exactly like the capsular tissue. Or there may be diffuse staining of lighter tint than the enclosing capsule. It is noticeable that the bulging of the capsule is always in the one direction. The original capsule, however thin, remains quite even, while one or more of the deep, sometimes thicker, new non-resistant layers bulge backwards. Pl. XVII, fig. 3 shows large numbers of minute spaces. The contrast between the greatly thickened anterior capsule and the thin posterior capsule is well shown. (This thickening may account for the faint diffuse general loss of transparency of many Morgagnian cataracts, first notice-

PLATE XVI.

Illustrates Lieut.-Col. H. Herbert's paper on The Capsular Opacities of Morgagnian Cataract (p. 349).

Fig. 1.— × 72.

Fig. 2.— × 72. Irregular thickening of capsule. Some of the greater thickenings show spaces forming. The larger swelling near the middle is multi-locular. The dark swelling immediately above this was found, under higher magnification, filled with particles of ? broken-down capsular substance. There are two small spaces below where the capsule is but little thickened.

Fig. 1.

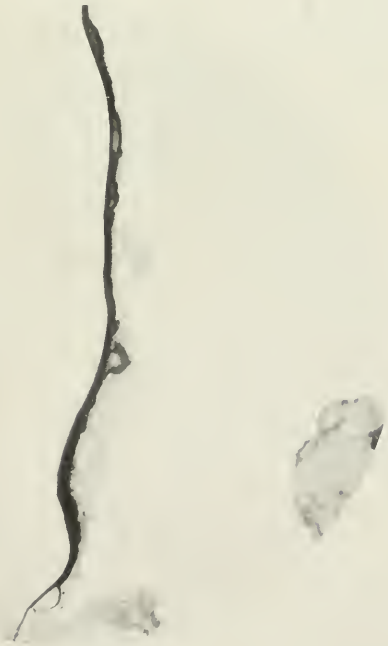

Fig. 2.

Adlard & Son, Impr.

able during operation after evacuation of the contents.) The formation of spaces is not limited entirely to obviously thickened portions.

To my mind the chief interest in this isolated case lies in the possibility that it may illustrate more or less exactly the changes accountable for the well-known clear rings or "globules" of one of the commonest forms of after-cataract. As I have never ventured to extract one of these thin after-cataracts I have not been able to test this suggestion. The clinical appearance of the globules seems much more easily explicable on this supposition than by any possible results of the proliferation of lens-cells. For the very distinct outlines of the circles, dark and defined by direct ophthalmoscopic examination, and their rounded shape, point to enclosures of some kind. And they are frequently seen in the posterior capsule, where it is uncovered by anterior capsule, so that there is no question of enclosure between the two portions of capsule, such as is frequently responsible for peripheral accumulation of grey swollen lens-matter.

One must assume that the capsular changes to-day shown are rarely, or never, met with in ordinary cataract before operation, as seen in this country. Otherwise, they would have been noticed before now under the microscope. In this country opportunities of obtaining cataractous lenses in the ultra-Morgagnian stage are very rare. Possibly it is only in the loose, tremulous capsules of some of these over-ripe cataracts that the conditions approximate to those of the capsule after cataract extraction—conditions responsible for capsular degeneration producing cavities, filled in the one case with lens *débris*, in the other case with clear aqueous. The after-cataract globules are rarely, if ever, found immediately after operation. They develop slowly in the course of months or years.

The invariable clearness of the after-cataract circles, most evident in the larger ones, exactly like that of the intervening capsule, is against the supposition of their being

filled with lens-cells. If lens-cells or their products were present it would be strange if opacity did not sometimes result from degeneration or abnormal development of the cells.

The usual cause of the white dots of capsular opacity of Morgagnian cataracts is recognised to be localised proliferations of lens-cells, with early calcareous degeneration. Calcareous granules may be present only in small patches, and the pseudo-fibroid tissue may recall that of anterior polar cataract, or of the larger anterior capsular plaque of the over-ripe, dry, senile cataract. But the proliferative effort is less sustained, degeneration soon sets in. In other parts of a capsule presenting small pseudo-fibroid patches, one may see great swelling and breaking down of cells, with excessive calcareous deposit in them. Some of these swollen calcareous cells become enclosed by the formation of a hyalin layer beneath them, generally thin, but sometimes, as in Pl. XVII, fig. 4, nearly as thick as the original capsule itself. The result here is not unlike the intra-capsular spaces shown in Pl. XVI, fig. 1, but the quite different origin is evident from examination of less advanced conditions in other portions of the same capsule.

On the whole, in Morgagnian cataract, it may be said that proliferative efforts soon come to an end, and the tendency to breaking down and disappearance of cells is great. Sometimes apparently there is complete disappearance of lens-cells. Any undegenerate cells which remain tend to spread out, and to become correspondingly thinned.

In my books on *Cataract Extraction* I have endeavoured to distinguish clinically between two processes, acting singly or together, in ordinary senile cataract. And I have recently examined histologically, the capsules of a collection of over-ripe cataracts, in attempting to carry the distinction a step further. In perhaps half the cases seen in Bombay the lens slowly

PLATE XVII.

Illustrates Lieut.-Col. H. Herbert's paper on The Capsular Opacities of Morgagnian Cataract (p. 349).

Fig. 3.— × 72.

Fig. 4.—Hæmatoxylin × 310. Showing a dense mass of calcareous granules enclosed between original capsule above and a newly formed layer beneath.

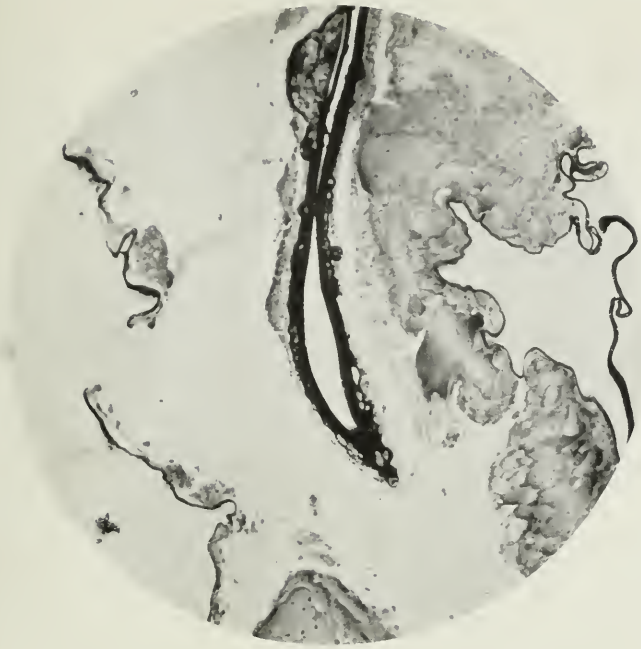

Fig. 3.

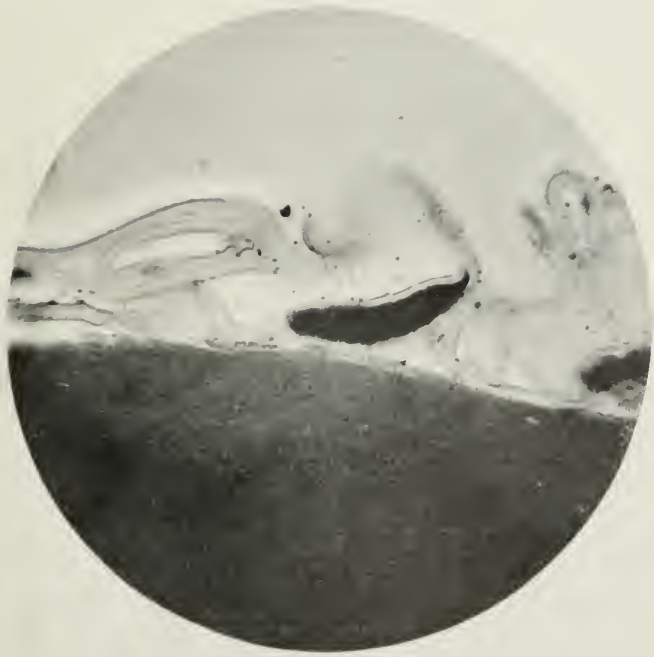

Fig. 4.

Adlard & Son, Impr.

becomes firmer and drier than normal, and progressively smaller, especially antero-posteriorly. And this change persists throughout. The cells lining the capsule remain of normal appearance, thick and closely packed, and when in the over-ripe stage a large, anterior capsular plaque may form, the tissue composing it remains firm and does not break down. Simple deficient nutrition, an early senile change, may account mainly for this dry type of cataract.

In other cases, either from the beginning or engrafted at any stage upon the above, changes are seen in greater or less degree comparable with those of cataract, resulting from a traumatic opening in the lens capsule, and doubtless attributable mainly to the same cause—abnormally free admission of aqueous to the lens substance. There is the same grey, moist swelling and breaking down of cortex, the *débris* and fluid forming in a late stage the milk of a Morgagnian cataract, eventually to be absorbed in the ultra-Morgagnian stage. With this is associated the degeneration and destruction of the lens-cells lining the capsule above referred to. And it seems right to assume an ætiological relationship between the two processes. That is, to attribute the undue passage of aqueous through the capsule and its results to deficient resistance presented by the few and feeble lens-cells. And thus to trace the whole of the changes ultimately, in part at least, to the action probably of toxins upon these cells.

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5. *Spontaneous dislocation of the lens into the anterior chamber in three members of the same family. Ectopia of the pupils. Extraction of the dislocated lenses.*

By W. T. Holmes Spicer.

Reginald S—, æt. 5 years, came to Moorfields on April 23rd, 1915. The peculiarity of the pupil had been

noticed from birth. The white spot in the right eye was first seen twelve months ago, since when it has not changed, and has not given rise to any trouble, except loss of sight.

Right eye.—Ectopia of the pupil downwards, the sphincter edge is complete, showing that it is not a coloboma. The anterior chamber is deep; the iris is tremulous; a small opaque lens is lying in the lower part of the anterior chamber; the lens appears to be in its capsule, but the rim of it appears slightly frayed out as if macerated. Tension normal.

Left eye.—Ectopia of the pupil downwards, symmetrical with the right eye; iris tremulous; lens in position and not completely opaque. Dull fundus reflex only; no details visible. Tension normal.

Both eyes rather small.

Phœbe S—, æt. 11 years, sister of the above, came under my care at Moorfields in January, 1902, thirteen years ago. The only history was that she had been born with the pupils drawn up.

In the right eye the cornea was clear; the anterior chamber was deep; the pupil was displaced upwards nearly to the corneal margin, but the sphincter was present; the iris was tremulous; the lens was not completely opaque, but no view of the fundus was obtained.

The right eye had the same characters, but the lens was lying in the anterior chamber, opaque, and with some calcareous deposit on its surface.

V. R. $\frac{6}{36}$
L. Hand movements. Tension normal in both.

The left lens lying in the anterior chamber was needled —that is, a hole was drilled in it; nothing more could be done owing to its elusiveness. No apparent change in the lens was effected, no swelling and no escape of lens matter, but the next day the tension rose and was estimated at + 3. Under anæsthetic it was removed with a scoop and some loss of vitreous. Eight months later, in September, 1902, as the result of a blow by an

indiarubber ball, the right lens became dislocated forwards, and was extracted with a scoop; there was some loss of vitreous.

The eventual vision and refraction of these eyes were as follows:

$$\text{R. } \frac{\text{Sph. } +\ 9}{\text{Cyl. } +\ 5 \text{ ax. } \diagdown 45^\circ} = \tfrac{6}{36}.$$

$$\text{L. } \frac{\text{Sph. } +\ 10}{\text{Cyl. } +\ 2\ 45^\circ \diagup} = \tfrac{6}{36}.$$

Ada S—, æt. 3 years, sister of the two foregoing, came to Moorfields in May, 1903, with the right lens dislocated into the anterior chamber.

Both irides were tremulous; pupils rather small and central; the left lens was opaque, lying in the anterior chamber.

Following the practice in the previous case I removed the lens with an incision upwards and a scoop; some vitreous was lost. The patient left the hospital after ten days had elapsed. There is no note about the vision, and it was never investigated later, nor did the other lens become dislocated, as the patient died about two years later.

There were eight others in family, none of whom were affected in a like way. No family history of others was obtained, but there was no family knowledge on either side.

The points of interest are: The variation in position of the pupils, in one case both being displaced upwards, in another both downwards, and in the third central; the tolerance of the lens in the anterior chamber, in no case did it cause disturbance or rise of tension, although an ineffective needling rapidly produced extreme tension. This reduced the choice of operation to removal. The loss of vitreous did not hinder the cure nor interfere with the result. I propose to treat the recent case in the same way, by extraction.

Mr. Charles Wray asked what was the condition of the disc, and whether the exhibitor attempted to remove the lens without making a conjunctival flap.

Mr. Holmes Spicer replied that he could not say what was the condition of the disc; it had not been visible. The lens still remained opaque. He did not make a conjunctival flap. It was not stated whether the incision was made with a needle or with a von Graefe knife.

X. DISEASES OF THE RETINA AND OPTIC NERVE.

1. *A case of retinitis pigmentosa treated by trephining.*

By M. S. Mayou.

It is with some hesitation that I bring the notes of this case before the Society, but I am tempted to do so since the improvement in the condition has been maintained for a period of four months and a half, and also because it may possibly in future throw some light on the ætiology of the disease, which is so little known. It is a well-known fact in retinitis pigmentosa that anything which improves the retinal circulation will temporarily improve the vision. As examples, administration of amyl nitrite, vomiting affecting the general circulation, local subconjunctival injections, paracentesis of the anterior chamber, will do so. Doyne has further shown that the extraction of the lens, even in cases where the opacity is not sufficient to cause bad vision, is followed by an immense improvement in the patient's vision. This he considers to be due to the fact that the patient was always in a condition of semi-daylight owing to the cataract, and therefore the retina did not receive sufficient stimulation. Although this may be so, the author thinks that the letting down of the intra-ocular tension with the flushing of the retinal vessels, by which it is followed, and the subsequent filtration of fluid through the scar, may be an additional, if not the most important, factor in the excellent results which he thus obtained. With this idea it occurred to the author that trephining

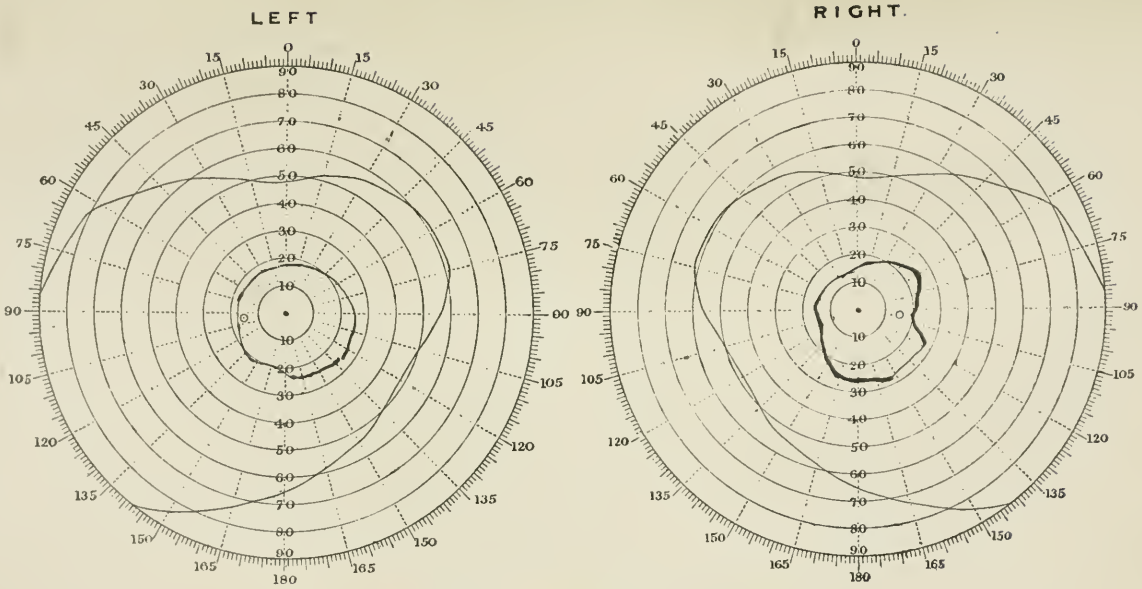
might be a means by which the retinal vessels might be flushed with blood, and that the subsequent leaking which took place underneath the conjunctiva might lead to a more rapid excretion of fluid from the eye, and at the same time drain away any possible toxic products that might be present, causing the local vascular sclerosis present in the eye.

The most striking feature about operating upon these patients is the tendency for the eye to form fibrous tissue. Doyne found that after removal of a cataract in many cases several needlings were required. In the case described below there was a distinct tendency for the trephine hole to become filled with fibrous tissue, although good leaking of the scar was obtained at the time of the operation.

The following are the notes of the case :

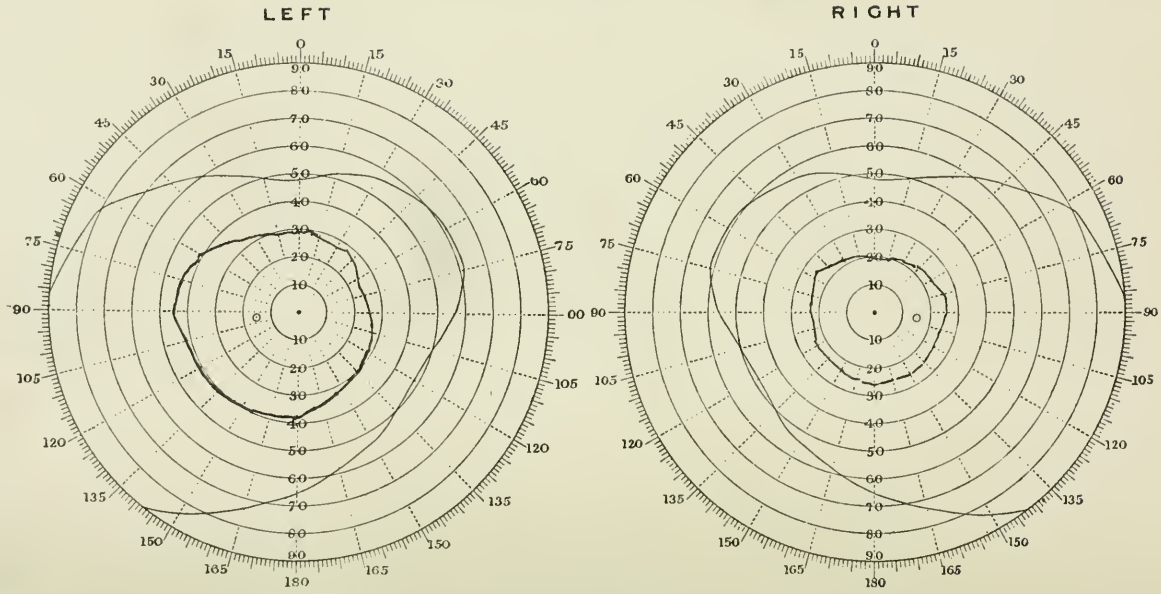
M. B—, female, æt. 17 years, domestic servant. Complains of night-blindness the last two years. Grandfather, father, one paternal aunt, and one brother are all afflicted with the same disease. The patient has four sisters, who are not night-blind. Wassermann reaction negative. Vision $\frac{6}{12} + 1\ \frac{6}{9}$ right and left. Patient is a strong, healthy girl, and she is unable to continue her work as a domestic servant as she cannot do her work in the evening. The fundus shows a typical but not very marked peripheral pigmentation of retinitis pigmentosa type. The discs are slightly grey in colour, but not at all markedly so ; the vessels are almost of normal size ; the lenses are clear and show no posterior polar opacity. The fields of vision when first examined were contracted to the ten degrees circle on both sides. These were examined on a number of different occasions, and varied between the ten and twenty degrees circles. On November 13th, 1914, paracentesis of the left eye was performed, and again on December 1st. After each paracentesis there was a distinct improvement in the fields. It was then determined to trephine. This was performed on December 8th, 1914. It was done under a general

FIG. 44.



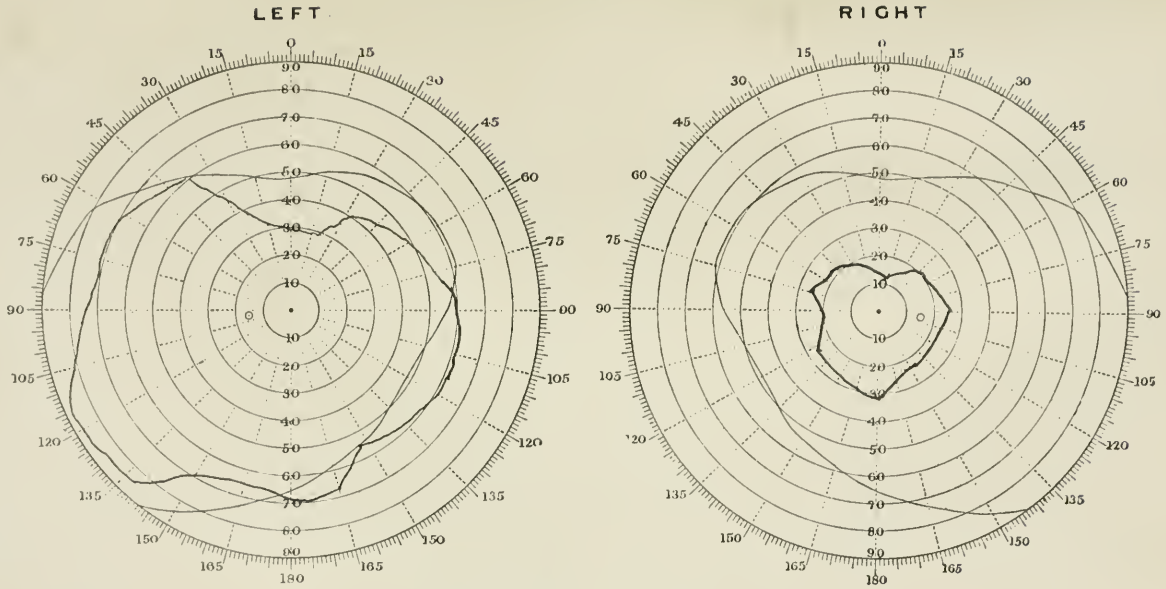
Maud B—, February 25th, 1914. White field.

FIG. 45.



Maud B—, December 3rd, 1914. After paracentesis.

Fig. 46.



Maud B—, December 18th, 1914. After trephining.

anæsthetic, as the patient was very restless. Directly the disc of sclera was removed the iris prolapsed very freely, and, although it was buttonholed after the operation, it remained a little caught in the wound; otherwise the recovery was uneventful. A week after the operation the field on the side which had been operated on had practically returned to normal, and the patient said that she had completely lost her night-blindness; this proved to be the case on examination, as she could walk about freely in a darkened room. From the time of her operation up to the present the improvement in the field has been practically maintained; if anything it may have decreased in a very slight degree. An alteration which the patient has noticed is that three weeks after her operation she can see at night time perfectly well if she covers up the unoperated eye, but if both eyes are open she is still somewhat night-blind. The explanation of this the author cannot give, but there is no doubt that it is a fact because she can move about in a dark room freely with her unoperated eye covered up, but cannot do so with both open. For this reason she is extremely anxious to have the other eye operated on.

Since operating on this patient she has brought me her aunt and father. The former I have trephined. Before the operation the aunt showed the same changes in the fundus as the patient, only far more extensive. There was very marked vascular sclerosis, and the disc had become atropic. The fields were contracted down to a pin-point, and, although the vision was $\frac{6}{36}$, the patient had to be led, and could not move about, even in her own house, alone. After the trephining the fields increased to the ten degrees circle, and when last I heard from her she was able to go about without somebody accompanying her.

The author would repeat once more that the improvement in these cases may be only for a certain length of time, but the length of time may be sufficiently long to render the operation justifiable in selected cases.

Mr. F. Richardson Cross said it was evident that in this case there was very marked improvement in the field, and the case gave hope that it might be worth while to do the operation described for these cases, though the definite histological changes in this disease made one suppose the benefit brought about might be only temporary.

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2. *Glioma of the retina with hypopyon-like deposit in the anterior chamber.*

By Cyril H. Walker.

James S—, æt. 4 years, was admitted to the Bristol Eye Hospital, on November 11th, 1914, with the history of a "film" having come over the right eye about eight months previously. This "film," whatever it was, only lasted one or two months, and then disappeared. He received no treatment and attended school regularly. The eye became a little red in June, and again in September, but until about three days before admission there had been no pain at all.

On examination there was no redness of the eye, no evidence of enlargement of episcleral vessels, and no increase of tension. The cornea was quite clear. The anterior chamber was of normal depth and about a quarter full of a yellowish-brown or snuff-coloured "hypopyon," which slowly gravitated to the lowest part of the anterior chamber in all positions. The pupil dilated well with atropin. The lens was clear, but only a very dull brown ophthalmoscopic reflex was visible. It was doubtful if there was any perception of light. The left eye was normal and the boy's general condition was good. There was no evidence of tubercle or syphilis.

A few days later Mr. Cross saw the case and suggested the possibility of its being a sarcoma. By November 20th, the brownish deposit in the anterior chamber had increased,

tension was + 1, there was slight ciliary injection, and the eye was a little painful. Paracentesis of the anterior chamber was performed, the evacuated contents were collected for pathological examination, and the anterior chamber was very gently washed out with Bishop Harman's irrigator. A great deal more pigmented matter escaped during this procedure, so that for some time the effluent was turbid and brownish.

On November 22nd, Prof. Walker Hall, pathologist, to the Bristol Royal Infirmary, to whom I am indebted for the microscopical specimens, gave the following report of his examination: "*Bacteria:* None found either in film or after culture. *Cells:* No polynuclears. No endothelials. The cells are all mononuclear and suggest a sarcomatous origin. Some of them contain large granules of pigment. The condition is not of pyogenic origin. I think that it will prove to be sarcomatous."

The eyeball was enucleated on November 26th. Examined with a lens at the time the cut end of the nerve looked normal, and the boy went home to Ilfracombe on the 29th.

On December 3rd, Prof. Walker Hall reported on the excised eye as follows: "The posterior chamber is filled with a red mass of material. Upon section this proves to be an angiosarcoma. It has extended into the optic nerve area and reached backward as far as the portion of nerve attached to the globe."

In consequence of this report the boy's doctor and parents were written to. He was re-admitted, and on December 16th some three-quarters of an inch of optic nerve, together with a mass of the tissues immediately surrounding it were removed. The report on this specimen was: "The sections show the extension of the growth along the optic nerve and also into the surrounding tissue, there is, however, a small margin of tissue histologically free from the growth."

The boy was discharged from the hospital on December 21st, and I received no news of him until April 2nd, when

Dr. Osborne, of Ilfracombe, kindly sent me the following note :

"The growth began to recur locally within a fortnight of the boy's return home, starting in the region of the optic nerve, with severe neuralgic pain in the socket. It is now the size of a large cocoanut, protruding about six to seven inches from the right socket. It has obliterated the right nasal bones and encroached on the right angle of the mouth. The growth is black with congealed blood, very offensive, and constantly bleeding. He sees perfectly well with his left eye, has all his mental faculties, and there is no evidence of metastasis in any organ."

Macroscopically, a section of the eyeball shows that the entire vitreous chamber is filled with a lobulated growth, presenting irregular patches of brown, red, and white. There is no evidence of a pathological detachment of the retina from the choroid. Microscopic sections show that the whole of the retina right up to the ora serrata is involved. The choroid appears to be invaded near the disc, and to a very slight extent in one place near the equator of the globe. Degenerative changes have taken place in a great many portions. The ciliary region and iris are not affected by the growth.

Whether the growth should be described as a glioma, a neuroblastoma, or an angiosarcoma, is a problem which has comparatively little clinical importance. The term angiosarcoma is, in the opinion of Mallory (*Pathologic Histology*, p. 272), objectionable since it indicates lack of exact knowledge. He appears to regard glioma as equally incorrect and misleading, and prefers to group this class of tumour under the heading of neuroblastoma. I have accepted the term angiosarcoma provisionally since it was the one given me by the pathologist, and because it seems to describe the histological features of the growth.

Mr. F. Richardson Cross said he had never seen any appearance exactly like that in Mr. Walker's case. So far as he remembered it, it seemed as though there was fluid in

the anterior chamber, a mixture of blood and pus: it was impossible to see beyond it into the vitreous humour. There was nothing about the eye, such as thickening or swelling or inflammation, suggestive of the presence of intra-ocular growth. The diagnosis he made was arrived at on the common-sense method "What else can it be?" It was of unusual type, and he was not surprised to hear how rapid had been its progress since. The description showed how important it was to deal with these suspicious cases at as early a stage as possible.

Mr. J. Gray Clegg said that he was recently present at an operation on a child in whom there was apparently hypopyon. He did know the exact details of the case. The idea was to open the eye and do evisceration, but on making the incision the operator found a hard mass which he concluded was some sort of growth. The eye was excised. The child was seen again later, and there was found to be a growth of the other eye. The clinical diagnosis at that time was that it was a glioma in the second eye. The hypopyon was therefore a tumour hypopyon similar to the one just mentioned.

Mr. George Coats said he thought it was of great importance to settle whether the case was one of glioma or sarcoma. This was not merely a matter of nomenclature; a sarcoma was a mesoblastic, a glioma an epiblastic, tumour. A true sarcoma at this age, and in the retina, was a thing which had never been described.

(A sub-committee consisting of Mr. E. Treacher Collins and Mr. G. Coats was appointed to examine the specimen, and reported it to be a typical example of glioma of the usual type.)

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3. *A case of hyaline bodies at the optic disc.*

By Cyril H. Walker.

William I—, æt. 13 years this month, is an only son. His mother says that she has never had another pregnancy

and that she had a very bad time indeed at his birth. His parents are both quite healthy. There is no history of consanguinity nor of any defect of sight in their families. The patient himself has never had any serious illness, and now appears to be a well-grown, healthy boy.

I first saw him at the Bristol General Hospital in 1908. He was then attending in the medical out-patient department, and there were some symptoms suggesting tuberculosis. Ophthalmoscopic examination at the time revealed nothing unusual, but as he was only six years old, and I was searching especially for tubercle of the choroid, it is possible that the very earliest stages of the present condition were overlooked. Retinoscopy showed 2·5 D. astigmatism. With + 2 D. sph. $\bigcirc$ + 2·5 D. cyl. axis vertical in each eye, he saw about $\frac{6}{24}$. This was the best result that I could get, and it did not occur to me at the time that he was suffering from night-blindness. The same indifferent result was obtained in 1909 and 1910.

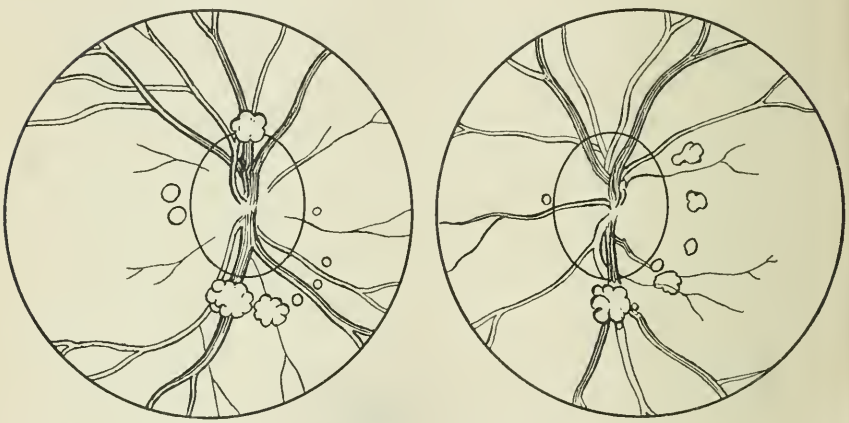
I saw him again at the Bristol Eye Hospital in November, 1911, when he was nine years old. He then had distinct night-blindness. There was decided blurring of both discs. In the right eye there was a white nodular mass consisting of a cluster of some four or five white, shining, translucent bodies. This mass was situated close to the lower edge of the optic disc. It obscured some of the inferior retinal vessels, and evidently, from parallactic movement, projected well into the vitreous. The summit of the mass could be seen with a + 6 D. lens. In the left eye there was a similarly situated group of three discrete nodules. These resembled boiled sago grains, whilst the mass in the right eye might be compared to tapioca.

In June, 1913, the night-blindness was a little more marked, but his vision in ordinary good light had not altered. The haze round the discs had increased, and there was some evidence of choroiditis both generally and at the maculæ, but the hyaline bodies had not altered appreciably. By December, 1914, there was marked retinitis pigmentosa in both eyes, but it was not of a

typical character, the pigment being more blotchy and less stellate or lace-like in its arrangement. Several new hyaline bodies had made their appearance.

April 15th, 1915 : Patient's health has been uniformly good for the last few years. His teeth are not suggestive of congenital specific disease, but the distal one third of the upper central incisors, all the lower incisors, and the four canines are extremely deficient in enamel. The lateral

Fig. 47.

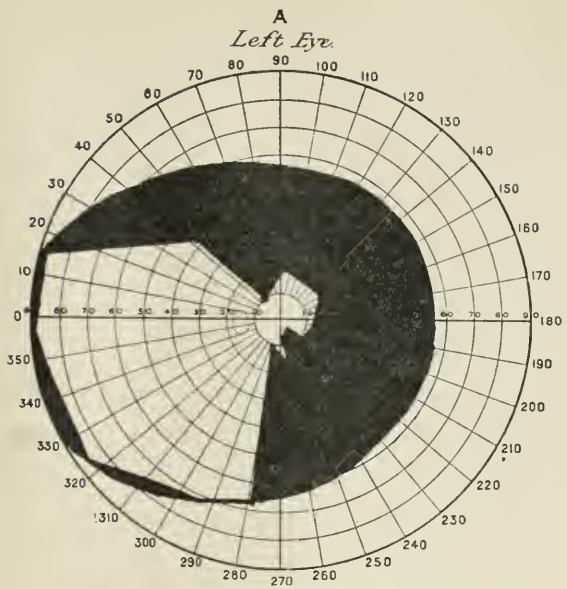


W. I—. Diagrams to show position of hyaline bodies on April 15th, 1915.

incisors in the upper jaw are well formed. His lenses show no evidence of lamellar cataract.

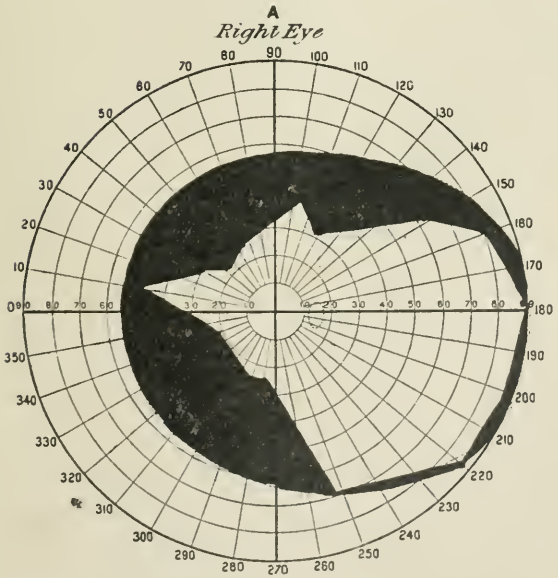
Ophthalmoscopic examination.—In the right eye there are now three large white masses of hyaline growth. One is above the disc and just overlaps its margin. Two are below and just clear of the disc margin. The long axis of the largest measures nearly half the diameter of the disc. There are also three isolated nodules on the lower and inner side of the disc, and two rather larger ones on the macular side. When these latter were first observed they were so translucent as to be visible only with difficulty. In common with the other nodules they are assuming a more pearly look as they get older. The larger

Fig. 48.



W. I—, April 15th, 1915. 6 mm. white.

Fig. 49.



W. I—, April 15th, 1915. 6 mm. white.

masses are distinctly lobulated or nodular, and can be seen with + 6 D. sph. In the left eye there is one large mass and eight smaller ones, one of which lies on the temporal side of the disc. A small branch of one of the macular vessels courses over the edge of one of the nodules, but in every other instance the nodules are in a plane considerably anterior to the vessels. Both discs are a little pale, and the retinal arteries are small, but not conspicuously so as in typical retinitis pigmentosa. Towards the periphery of the fundus there is a considerable amount of choroido-retinal pigmentation, but there is none in the immediate neighbourhood of the disc and macula.

R.V. $\bar{c}$. + 3 D. sph. $\frown$ + 2·5 D. cyl. ax. vert. = $\frac{6}{36}$ and J. 10.

L.V. $\bar{c}$. + 2 D. sph. $\frown$ + 2·75 D. cyl. ax. vert. = $\frac{6}{18}$ and J. 6.

Colour-vision is very defective, more especially for green, and night-blindness is very marked now. The fields of vision are contracted, particularly on the nasal side and above. It seems probable that the sight in the right eye has failed slightly during the last two years, but that in the left eye does not appear to have altered.

Gifford (*Diseases of the Eye, Ear, Nose, and Throat,* De Schweinitz and Randal, vol. i, p. 452) mentions the occurrence of hyaline bodies in a patient, æt. 9 years, but I have not met with a detailed description of their occurrence in a patient younger than the present instance.

Mr. G. Coats said that the pathology of the condition had been fully discussed by Mr. Parsons (*Trans. Ophth. Soc.*, xxiii, p. 135, 1903). He had himself reported the microscopical examination of a case (*Trans. Ophth. Soc.*, xxxii, p. 119, 1912).

Mr. Cyril H. Walker said he wished to show the case because he believed it was nearly the youngest, if not actually the youngest, patient in which the condition had been seen. He would like to hear opinions as to the prospects of ultimate vision.

XI. DISEASES OF THE NERVOUS SYSTEM.

1. *Case of double facial paralysis.*

By E. Treacher Collins.

A girl, æt. 7 years, who comes from a poor-law school and of whom no previous history can be obtained.

She is a well-nourished and fairly intelligent child, with paralysis of all the muscles of the face on each side. She is unable to close the eyes from paralysis of the orbicularis muscles. When told to do so, she rolls the eyes up under the upper lids. The lower lids fall a little away from the eyeballs and there is epiphora, the lower lacrymal puncta not coming into contact with the globes. She has an absolutely expressionless face, the so-called myopathic face. She never laughs and her tears are always overflowing. Her hearing on each side is quite good. She has some paralysis of the extensor muscles of her legs, which causes her to walk with a peculiar gait, her toes turning inwards.

The case is probably one of primary infantile muscular atrophy of the type described by Duchenne. So far there is no affection of the muscles of the shoulder-girdle, but it is stated that there may be an interval between the onset of the facial and the scapulo-humeral muscular atrophy of ten to fifteen or more years.

XII. COMPARATIVE ANATOMY AND PATHOLOGY.

1. *The choroid and retina of the fruit-bat.*

By George Coats.

(With Plate XVIII.)

This communication lays no claim to the credit of an original discovery. But I have thought that it might interest members of the Society to have their attention drawn to a very extraordinary peculiarity of structure, which appears at first sight to traverse principles essential to the perfection of the eye as an optical instrument, and which is confined, so far as is known, to a single family of the bats. The condition was first observed and briefly described by Lindsay Johnson * (1900), and has recently been the subject of independent and more elaborate papers by Kolmer † and Fritsch.‡ My own observations refer in part to the same animal as was studied by Kolmer (*Pteropus medius*), but I have been able also to examine material derived from an allied species—*Cynonycteris collaris*.

As seen in *Pteropus medius*, the peculiarity consists in the fact that the inner surface of the choroid is not smooth, but is strewn with innumerable conical peg-like projections which inter-digitate with corresponding depres-

* Lindsay Johnson, "Compar. Anat. of Mammalian Eye," *Proc. Roy. Soc.*, May 17th, 1900.

† Kolmer, W., *Zeit. f. wiss. Zool.*, 1911, p. 91; *Anat. Anz.*, vol. xl, 1911, p. 626.

‡ Fritsch, G., *Zeit. f. wiss. Zool.*, 1911, p. 288.

PLATE XVIII.

Illustrates Mr. George Coats's paper on The Choroid and Retina of the Fruit-Bat (p. 372).

Fig. 1.— × 120. The choroid and retina of *Pteropus medius*. Note the thread-like capillary loop emerging from the apex of the two central cones. The cones are clothed with a layer of flat, unpigmented cells.

Fig. 2.— × 120. The same depigmented. A fine straight vessel is seen running up the centre of the cone in the middle of the figure.

Fig. 3.— × 150. The condition in *Cynonycteris collaris*. The cones are relatively more elongated and narrower, but the spaces between them are partially filled in by means of a thickening of the cells of the retinal epithelium. Hence the general contour of the serrations is much the same as in Pteropus.

Fig. 1.

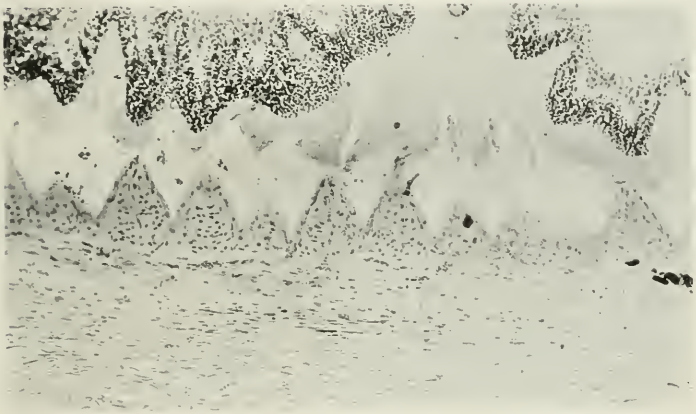


Fig. 2.

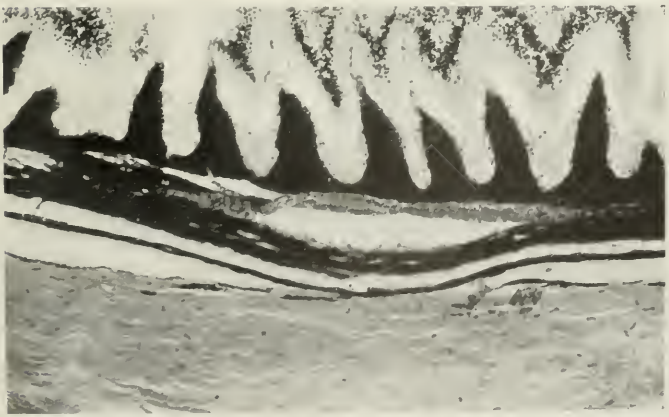


Fig. 3.

Adlard & Son, Impr.

sions in the outer aspect of the retina (Pl. XVIII, fig. 1). These cones measure about 100 μ in height by from 60 μ to 80 μ across the base. They are of very regular size over any limited area, but gradually diminish in height from behind forwards, and disappear at the ora serrata. Posteriorly they stand side by side with no flat choroid between them, anteriorly they are spaced somewhat more apart, with small intervening plane areas. Posteriorly the direction of their axes is strictly radial to the circumference of the globe; anteriorly it is tilted a little, so that their apices point slightly forward.

The projections are very heavily loaded with pigment, but in depigmented specimens it can be seen that they consist of outgrowths of the inner layers of the choroidal stroma (Pl. XVIII, fig. 2). The tissue composing them is more compact and more richly cellular than that of the stroma generally, and does not show the wavy outline or fibrillation of ordinary connective tissue. Through the centre of each there runs vertically a small vessel, somewhat above the dimensions of a capillary. It takes its origin in the medium sized vessels of the inner layers of the choroid, and on arriving at the apex of the cone does not stop there, but passes inwards in the form of a long filamentary loop which stretches into the subretinal space for a distance approximately equal to the vertical height of the cone itself (see the two central cones in Pl. XVIII, fig. 1). This capillary loop is unpigmented. On the surface of the cones, and following all their serrations, there is a well-developed chorio-capillaris. Apart from these central and superficial systems the vascular supply of the cones does not appear to be very rich.

The cells of the pigment epithelium are flat and show no obvious inter-digitation with the retinal rods. They are thicker in the pits between the choroidal cones, become thinner in ascending their flanks, and over the capillary loops are reduced to a membrane of almost invisible tenuity. Pigment granules occur in moderate abundance towards the ora serrata; posteriorly they are practically

absent ; the pigmentation is nowhere nearly so deep as in the human eye.

The other layers of the choroid present no very special features. They are well developed, contain many large vessels, and are deeply pigmented but not more so than in most animals.

There is no tapetum.

As regards the retina, my own observations, so far as they go, entirely corroborate those of Kolmer, but as my material seems to have suffered more than his from post-mortem change, I shall supplement my description with some details from his paper.

The zig-zag sectional outline imposed by the penetration of the choroidal cones affects only the outer layers of the retina; the inner surface is smooth. The visual elements, according to Kolmer, measure 51 μ in length (outer limb 30 μ, inner 21 μ) and 1 μ in breadth. As in other bats, therefore, they are all of the "rod" type. They are of approximately equal length on the summits of the retinal serrations and in the depressions between them. They do not stand perpendicularly to the varying contour of the limitans externa, but are everywhere directed radially with respect to the surface of the globe. The limitans externa is well developed. The outer nuclear is the most prominent of the retinal layers ; apart from oblique cutting it does not appear to vary greatly in breadth, being usually from eight to ten cells thick. The outline of the inner nuclear, on the contrary, is a series of triangles corresponding with the triangular section of the main retinal serrations ; at its greatest breadth it is about eight or nine cells thick ; the nuclei are much fewer in number than in the outer nuclear layer. In agreement with this arrangement of the inner nuclear layer, the outer reticular is also broadest in the base apex line of the retinal triangles.

The capillary loop which has been described as springing from the apex of the choroidal cones invades, or rather invaginates, the retina as far as the inner nuclear layer,

and in the region of its penetration the rods, outer nuclear, outer reticular and inner nuclear layers all suffer interruption. The limitans externa, on the other hand, remains intact, and in the bottom of the retinal pits, owing to the absence of rods, comes into direct contact with the capillary loop.

The broad inner reticular layer is not invaded by the loop and has the same appearance as in other mammalian retinæ. Ganglion cells are not abundant, forming a single layer of loosely scattered cells. Kolmer reckons about 400 rods to one ganglion cell, and in the periphery this proportion is probably considerably larger. The nerve fibre layer is very thin and the optic nerve is small. There is no well-defined area centralis or fovea.

As regards the other parts of the eye, the cornea is much thicker and better developed than in the insectivorous bats, the epithelium 5–6 layers thick, a membrane of Bowman absent, the membrane of Descemet prominent. The iris is thin, very heavily pigmented, and possesses a well-marked sphincter and (in opposition to the statement of Kolmer) quite a good dilatator. The ligamentum pectinatum is represented by a spongy network filling the corneo-iridic angle. The ciliary processes are prominent and complicated, and spring from the root of the iris rather than from the ciliary body proper.

I must emphatically dissent from Kolmer's statement that a ciliary muscle is absent. In my specimens it is not only unmistakably present, but reaches quite a high degree of development. It is composed entirely of meridional bundles, interspersed with streaks of chromatophores. No cones corresponding with the choroidal cones are present on the ciliary body. The ora serrata comes far forward. The lens is relatively large, measuring about 7·5 mm. by 6 mm.

The other animal whose eyes I have been able to examine is the Hottentot or collared fruit-bat (*Cynonycteris collaris*), which belongs to a genus closely allied to Pteropus, but

inhabiting Equatorial and Southern Africa. Here the conditions are essentially the same as in the Indian fruit-bat (Pl. XVIII, fig. 3). As the eye had been divided equatorially I am unable to give exact measurements. The height of the choroidal cones is about the same as in *Pteropus medius* (100 μ), but they are considerably narrower at their bases (40 μ), so that in section the saw-teeth appear more isolated and more steep. In reality, however, this difference is compensated by a remarkable arrangement of the pigment epithelium. In *Pteropus medius*, as has been seen, the cells, though somewhat thicker in the depressions between the choroidal cones, form everywhere a flat layer. In Cynonycteris, on the other hand, this increase of thickness in the cells at the bottom of the depressions is exaggerated to a very great degree, so that the depression is partly filled in with swollen epithelial elements (Pl. XVIII, fig. 3). In section these swollen cells have a homogeneous aspect and a peculiar diffuse yellow colour. A small nucleus is present near the outer border and a few pigment granules are visible, chiefly where the cells are in contact with one another. The cells rapidly flatten in mounting the flanks of the cone, and over the capillary loop which emerges from its apex they have the same endothelium-like appearance as in Pteropus. Towards the anterior part of the choroid the epithelial cells are less regularly disposed, appear to be proliferating in places, and contain much more pigment. In consequence of this padding up of the bottom of the depression the contour of the retinal serrations is practically the same as in Pteropus, in spite of the difference in the form of the choroidal cones.

The choroidal stroma and the cones in Cynonycteris are very deeply pigmented. The retina has suffered too much from post-mortem change to be worth describing. The optic nerve measures 1 mm. in diameter and has a good system of trabeculæ converging to a central core, in which only very small vessels are present. The structure of the cornea and iris differs little from that of Pteropus. The

presence of a well-developed ciliary muscle is quite unmistakable, the more so that in this animal the outer layers of the ciliary body are relatively free from pigment. The ciliary processes are long and much plicated, and are set well back, on the ciliary body itself rather than on the iris.

The above observations may be compared with those of Fritsch, who describes the condition in an animal obtained in Sumatra, and believed to be *Pteropus condorensis*. The identification, however, as the author himself admits, is open to question. According to Dobson, whose *Catalogue of the Chiroptera* is regarded as authoritative, *Pteropus medius* and *Pteropus condorensis* (or *nicobaricus*) are very closely allied, and approximately of the same size, yet the eye in Fritsch's specimen, which was believed to be adult, measured only 5 mm. antero-posteriorly, as against 12 to 13 mm. in Kolmer's.

In this animal the same peculiar structure was found, but instead of having the form of cones the choroidal prominences were finger- or spindle-shaped, measuring 80 μ long by 15 μ to 20 μ broad. They contained small, almost capillary vessels, with a tortuous course; in some a straight arterial twig was present. Fritsch could not satisfy himself of the presence of a regular capillary loop. As in my specimen of Cynonycteris the spaces between the choroidal processes were partially filled in with swollen cells belonging to the pigment epithelium, with the result that the bases of the finger-shaped processes were padded out, and a contour approximately the same as that which obtains in *Pteropus medius* was impressed upon the outer surface of the retina. In comparing his specimens with those of Fritsch, Kolmer considers it very remarkable that the same conical form of the processes should depend in one case chiefly on the mesoblastic choroidal stroma, in the other on the epiblastic pigment epithelium; in reality, however, even in *Pteropus medius* the cells are slightly thicker in the depression between the cones. The differ-

ence, therefore, is merely one of degree, not of kind, and Cynonycteris furnishes an intermediate connecting link between the conditions in the other two species.

Lindsay Johnson, to whom priority in the discovery of this interesting peculiarity is undoubtedly due, unfortunately gives few microscopical details. In his well-known monograph on the fundus in animals (p. 14) he says: "The Indian fruit-bat (*Pteropus Indicus*)* and the Australian fruit-bat (*P. poliocephalus*) have ochre yellow fundi, which appear pitted at perfectly regular intervals by dark depressions. Microscopic sections show that these depressions are due to nipple-shaped protuberances of the choroid covered with dense pigment."

Zoological Status of the Fruit-Bat.

Zoologists divide the order Chiroptera into the sub-orders *Megachiroptera* and *Microchiroptera*, corresponding nearly with a division into the frugivorous and the insectivorous bats; nearly, but not quite, for certain of the Megachiroptera are supposed sometimes to supplement their diet with animal food, while some of the Microchiroptera are frugivorous, and some blood-suckers.

The condition now under consideration has so far been described in four species, belonging to two genera of the Pteropus group of the Megachiroptera (*Pteropus medius, condorensis, poliocephalus;* and *Cynonycteris collaris*). Of these the first three are very closely allied, and belong to a genus, the "flying foxes" (not to be confused with the parachuting "flying squirrels"), which is widely distributed in the tropical and sub-tropical regions of the Eastern Hemisphere, ranging from Samoa, through Australia, the Malay Peninsula, and India, to Madagascar and the Comoro Islands in the Mozambique Channel, within two hundred miles of the African coast. The genus Cynonycteris has a similar distribution, but occurs also in Africa, Syria, and Asia Minor. The species examined by me is an inhabitant of Central and Southern Africa. The

* I presume that this is the same animal as *P. medius*.

two genera, however, are nearly related, their separate classification depending on minor differentia, such as the shape of the muzzle, the relative lengths of the metacarpal bones, the presence or absence of a tail, etc. ; the skull is much alike in both. It would be of great interest to know whether the same ocular condition is present in other allied genera (Epomorphus, Cynopterus, Harpyia, and Cephalotes) or in the other group of the Megachiroptera (the Macroglossi).

Comparison of the Eye of the Fruit-Bat with that of other Bats.

Comparing the eye of the fruit-bat with that of the Microchiroptera, the most striking difference is its large size, a difference out of all proportion to the larger size of the animal itself, as will appear from the following table :—

Megachiroptera.

	Length of animal.	Greatest diameter of eye.	Antero-posterior diameter of eye.	Proportion of body length to diameter of eye.
*Pteropus medius**	230 mm.	12·5 mm.	—	18·5 : 1
,, ,,	230 mm.	—	11 mm.	21 : 1
,, ,,	230 mm.†	11 mm.‡	11 mm.‡	21 : 1

Microchiroptera.

Plecotus auritus	45 mm.*	1·5 mm.‡	1·5 mm.‡	30 : 1
Vespertilio noctula	75 mm.*	2 mm.‡	2 mm.‡	37 : 1
*Vesperugo noctula**.	75 mm.	1·7 mm.	—	44 : 1

The significance of these figures is enhanced when it is remembered that within the same zoological order, and under similar conditions of life and environment, the smaller species usually possesses the relatively larger eye.

In other respects the development of the eye of the Microchiroptera is somewhat inferior to that of the fruit-bats. The cornea is very thin, consisting of only three

* Kolmer's observations.

† Figure given by Dobson.

‡ Personal observation. I am unable to give the measurements for Cynonycteris as the eye had been divided coronally before I received it. Roughly speaking it is similar in size to that of Pteropus.

or four lamellæ. The iris is slender and heavily loaded with pigment. The ciliary processes are fairly prominent and complex, but a ciliary muscle appears to be entirely absent. The lens is large and round (proportion to length of eye in *Plecotus auritus* 0·75 : 1 ; in *Vespertilio noctula* and in a British bat of which I do not know the species, about 0·5 : 1 ; in Pteropus 0·5 : 1). No trace of the special structure now under consideration is present in any species so far examined. In the choroid of Plecotus—whether above or below, I cannot say—there is an area in which pigment is defective in the pigment epithelium and absent in the superficial layers of the choroid, which are composed of coarse fibrous bundles ; this appears undoubtedly to represent a tapetum fibrosum. In the other British bat above referred to, there were areas in which the pigmentation of the epithelium was defective, as is the case normally over the tapetum, but since the animal was albinotic (with pigmented eyes, however), too much stress should not be laid upon this point ; a proper tapetum fibrosum appeared to be absent.

In both the Mega- and Microchiroptera the retinal structure is very similar : the visual elements are exceedingly numerous and are all of the rod type ; the external nuclear layer is correspondingly prominent ; the nuclei of the bipolar layer are large and much fewer in number ; while the ganglion cells are relatively very scanty, forming only a single, much scattered layer ; the nerve fibre layer is poorly developed, and the nerve is thin. A fovea or "area centralis" appears to be entirely absent.

Habits of the Fruit-Bats.

With regard to the habits of the fruit-bats, I gather the following particulars from an article by G. W. S. Dallas in Cassell's *Natural History*, vol. i, p. 259. They are gregarious, and hang by day in enormous crowds from the highest branches of the trees. At sunset they fly to their various feeding grounds, often covering great distances—as much as thirty miles each way, it is said—since no small

area will support their voracity. Their flight is strong, but not very rapid. They feed at night, with much squabbling and fighting, and at dawn return to their trees; during the forenoon they often come out for a couple of hours, perhaps to sun themselves and dry their wings and fur. They are almost helpless on the ground, but climb well.

Two specimens of *Pteropus medius* examined by me, one after dilatation of the pupils with atropin, showed no photophobia in the ordinary diffuse daylight of a winter day, and the atropinised animal did not resent examination even with the electric ophthalmoscope. This is contrary to the statement of Kolmer, who says that under atropin the animals are photophobic. The anterior chambers were shallow owing to the iris being pushed forward by the large lens. In confirmation of Kolmer's description and against that of Lindsay Johnson, I found the pupils extremely small—almost pin-point; it was impossible to be sure whether or not they reacted to light. Kolmer says that there is no reaction to accommodation, but does not inform us how he applied the test. The pupils dilate fairly well with atropin. The fundus is yellow,* and is uniformly sewn with innumerable small, round, greyish-black areas; it would be impossible, I think, to say with certainty whether these were elevations or depressions, but the microscopical examination, of course, proves that they are elevations. The disc is staring white, and there are no retinal vessels. The picture in *Pteropus medius* is practically the same as that which is so excellently portrayed by Lindsay Johnson in the case of *Pteropus poliocephalus* (Pl. VI); perhaps the grey areas are somewhat more closely set in the former. The animal examined by me was approximately emmetropic under atropin.

Physiology of Vision in the Fruit-Bat.

Such being the anatomical and physiological data, what function is to be ascribed to a structure at once so singular

* *Vespertilio natteri*, one of the Microchiroptera, has also a yellow fundus according to Lindsay Johnson.

and of such restricted distribution within the animal kingdom? Fritsch considers that it has analogies with the pecten of the Sauropsidæ. The comparison appears to be entirely inadmissible. There is no point of real resemblance between these small uniformly distributed prominences which nowhere pierce the retina proper, and a vascular plexus which passes through all the retinal layers and projects into the vitreous in one situation only —at the nerve entrance. When Fritsch, proceeding from this supposed analogy, goes on to discuss whether the function also may be similar—either the regulation of the intraocular tension or the shading of parts of the retina in certain positions of the head—his speculations become entirely fanciful. A glance at the figures suffices to show that no such shadow could be thrown on the visual elements, more especially, as Fritsch himself admits, in cases where the choroidal processes are not conical but finger-shaped; moreover, the tilting forward of the cones near the ora serrata is evidently a device to bring them into line with the incident rays, and so avoid any casting of shadows. It might well be asked also why shading of the retina should be required in a nocturnal animal.

According to another view the structure is a special device for the nourishment of the retina, the vascular loops which project from the apices of the cones taking the place of the absent central retinal vessels. It goes without saying, of course, that the supply of the retina must be an important part of the function of the capillary loops, and no doubt their presence accounts for the lack of central vessels,* but it is difficult to believe that the whole mechanism has been developed to this end alone. Many animals whose retinæ are infinitely thicker than that of Pteropus have no, or rudimentary, central vessels (*e.g.*, the rhinoceros and elephant); yet in these cases

* Absence of retinal vessels is considered by Lindsay Johnson to indicate a low position among the mammalian orders, and on this ground he is inclined to degrade the Chiroptera in the scale. But evidently this is scarcely to be justified in the case of the fruit-bats if they have another and equally specialised vascular apparatus.

sufficient nourishment is obtained from a flat chorio-capillaris without the sacrifice of form vision which the special arrangement in the fruit-bat necessarily entails.

Another suggested explanation is that of Kolmer—that the device represents a rudimentary species of accommodation, the image of distant objects falling on the more anterior visual elements, of near on the more posterior. In weighing this suggestion it is necessary to inquire (1) what range of accommodation would be obtainable by this device and (2) what effect the serrated disposition of the visual elements would have on form vision.

(1) The space between the most anterior and the most posterior visual elements is about 0·1 mm. In the human eye this would correspond, roughly speaking, with 0·25 D. Without entering into elaborate calculations it may be said that since the eye of Pteropus is about half the size of the human eye, and is emmetropic, parallel rays must be refracted twice as strongly as in the human eye. The error of refraction caused by a difference of level in the fundus will be approximately four times as great as in the human eye, and 0·1 mm. will correspond with about 1 D.* This implies a range of accommodation from infinity to

* This is only a very rough calculation made as follows: In the reduced human eye the posterior focal distance is 20 mm. This corresponds with a convex lens of $\frac{1000}{20} = 50$ D.; if now the screen be displaced forwards 0·1 mm. the posterior focal distance will be 20 mm. − 0·1 mm. = 19·9 mm., and the corresponding lens will be $\frac{1000}{19{\cdot}9} = 50{\cdot}25$ D. Supposing the eye of Pteropus to be half the size of the human eye (in fact its antero-posterior diameter is 11 mm. instead of 12 mm.), and granted that the measurements of the reduced eye of Pteropus are one half those of the human reduced eye, which is approximately true since it is emmetropic, then the posterior focal distance will be 10 mm. corresponding with a convex lens $\frac{1000}{10} = 100$ D.; and if the screen be shifted forward 0·1 mm. the posterior focal distance will be 9·9 mm. and the corresponding lens $\frac{1000}{9{\cdot}9} = 101$ D. The correction necessary to reproduce emmetropia after the displacement of the screen would be in the human eye 0·25 D., in the eye of Pteropus 1 D., *i.e.* as stated in the text the error of refraction caused by a difference of level would be about four times as great in the second case as in the first. This calculation supposes the most posterior visual elements to be at the plane of emmetropia, the anterior being relatively myopic; the opposite case need scarcely be worked out in detail.

1 metre—an amount which would not seem to be very useful to a nocturnal animal whose main activites are fruit-eating and fighting with its neighbours.

(2) With regard to the effect on form vision a difference of level of 0·1 mm. seems at first sight to be no great matter; a person with 1 D. of myopia or hypermetropia does not see so very much worse than an emmetrope. But how would it be if a difference of 1 D. were repeated all over the fundus at intervals of about $\frac{1}{27}$th of a millimetre ? *

Again, it must be remembered that a flat image falling upon the most posterior visual elements will be interrupted by large relatively myopic areas which are out of focus, while an image falling on the most anterior will be interrupted by equally large hypermetropic areas. In both cases a line would be imaged as a series of sharply focussed points separated by areas of blurred vision, and would have more or less of a moniliform outline. Between these two planes there will be an intermediate plane in which the areas of imperfect vision will be equal, in which the visual elements under simultaneous concentrated stimulation will be as numerous and as evenly distributed as is possible under the circumstances, and in which presumably the form vision will be the best which the anatomical conditions admit.

From these considerations it appears to follow that the formation of a clear image of any small object is quite impossible, and that no advantage would be gained by displacement of the image, such as it is, either backwards or forwards from the intermediate plane of most acute vision.

An even stronger objection to Kolmer's explanation, however, is the undoubted presence of a well-developed accommodative apparatus of the usual type in the anterior

* The distance between the apex of one cone and that of the next is approximately ·0075 mm.; the lateral distance between the most anterior and posterior visual elements in a single serration is therefore half that amount — ·0037 mm. or nearly $\frac{1}{27}$ mm.

part of the eye. Clearly this will be of advantage to the animal by keeping the image focussed on the intermediate plane above mentioned, and so making the best of the form vision, bad though it be ; and also by concentrating the light reflected from the dimly illuminated objects which are met with during the animal's nocturnal excursions. But it is extremely improbable that in the same eye two different accommodative mechanisms should be provided, one very imperfect, the other much more highly developed.

In spite, however, of the fact that the accommodation hypothesis must be rejected, it is not impossible that the changing nature of the image as it falls on successive planes of visual elements from before backwards, may impart to the animal some sense or impression, the nature of which we can scarcely conceive, concerning an approaching object.

A study of the structure of the retina itself leads independently to the conclusion that form vision must be bad. The retina belongs distinctly not to the form-perceiving, but to the light-perceiving type. In the first place cones are absent ; but of even greater importance is the disproportion between the great number of visual elements and the small number of ganglion cells. If it be true, as is probable, that a single nerve fibre transmits only a single nerve impulse to the brain, then evidently the differentiation of detail (*i.e.* good form vision) must be more perfect when a few visual elements, contained within a small space, are connected with a single ganglion cell and with its corresponding nerve fibre, than when a large number are so connected. The former arrangement increases the perfection of differentiation ; the latter increases the power of the stimulus conveyed by each individual nerve fibre, a function which is especially useful when the stimulus itself is of low intensity, *i. e.*, under low illumination.

From this point of view the retina of the bats—both the Mega- and Microchiroptera—is characteristically a

retina adapted to make the utmost use of low illuminations. It is like the human peripheral retina. In another respect also—its large size—the eye of Pteropus is a typical nocturnal eye. A large eye implies, of course, the admission of more light and a greater expanse of sensitive film; hence, relatively, the largest eyes are found in some of the deep sea fishes.

In my opinion the present peculiarity of structure exemplifies another adaptation to the same end. It is evident that the corrugated contour of the outer aspect of the retina must greatly expand the total superfices of the visual layer, and must greatly augment the number of visual elements which can be crowded into a given space, and which will therefore be stimulated by a given amount of light. Hence the formation of an accurate image is sacrificed to something of more importance to a nocturnal animal which does not pursue an active prey—namely, vision, and especially the perception of obstacles, under a low illumination. In spite of the totally different anatomical structure and physiological mechanism, therefore, it would appear that the function of the structure here described is the same as that of the tapetum, and it is noteworthy that in none of these fruit-bats is there a true tapetum (unless, indeed, as is conjectured by Kolmer, the swollen pigment epithelial cells in *Pt. condorensis* and Cynonycteris act as such), whereas in one of the two Microchiropterous bats examined by me a tapetum was present.

It may be asked why the eye of the insectivorous bats should be inferior in point of development to that of the frugivorous, why it should be so small, and why it should possess no accommodation. Might it not reasonably be anticipated that the insect-hunter would require more perfect eyes than the fruit-eater? The answer appears to be that in the avoidance of obstacles, and probably even in the capture of his prey, the insect-hunter is not guided solely, or perhaps chiefly, by vision. The Abbé Spallanzani found that blinded bats set free in a room across which cords and other obstructions had been

stretched, never touched any of these obstacles, but flew as freely as if vision were perfect. When they had settled down they rose at once if a move were made to capture them. In a tortuous cave not only did they never touch the walls, but from a distance of 18 in. they discovered a hole in the roof and changed their course so as to hide in it. This faculty is believed to depend on a tactile sense which is exquisitely acute to slight currents of air, etc., and which is believed to reside partly in the wing membranes, which are very vascular and richly supplied with end organs, partly in the expanded ears, traguses and nose membranes which form so extraordinary a feature of many bats, especially such as fly late and frequent dark retreats. A similar "sense of obstacles" is not infrequently developed in persons who become blind early in life. It is especially to be noted that such membranous appendages are wholly absent in all the fruit-bats, which would seem to show that they depend more on vision and less on the tactile sense than the Microchiroptera.

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2. *Demonstration of a filaria from the eye of the horse.*

By Lieut.-Colonel W. G. Pridmore and George Coats.

(With Plate XIX, figs. 2 and 3.)

The occurrence of free-swimming filarious parasites in the anterior chamber of the horse is well known to those who have practised in the East, and has been the subject of a comprehensive paper by Lingard (*Journ. Trop. Vet. Science*, 1906, i, p. 175). From this paper the details which follow are taken. Our communication is merely a "demonstration," and does not pretend to bring forward any new facts.

The condition, apparently, was first observed in the eye of an ox by Grisoni, in 1429. According to Manson a picture showing the removal of a filaria from the sub-

conjunctival tissues of a woman occurs in a work by Pigafetta, dating from 1598. In the anterior chamber of the horse the parasite was first described by Spigel in 1622. Modern observations of filaria in the subconjunctival tissues, anterior chamber, lens and vitreous of man are sufficiently summarised in the standard works on ocular pathology, and need not receive further consideration in this place. The parasite has been observed also in the eye of the camel and of the turkey. It is sometimes found in the lacrymal duct and conjunctival sac of animals.

In the horse the condition is common in India, Burma, and Ceylon ; comparatively infrequent in China ; rare in Europe and America. Epizootics among bovines have been recorded in France and Belgium.

Lingard's paper is founded on a study of ninety cases, which came under his notice in Government depôts, during a period of six years. Of these fifty-one were observed at Karnal, in the Punjaub ; twenty-one at Saharanpur, in the United Provinces ; and eighteen at Hosur, in the Madras Presidency. The age of the affected animals varied between nine months and seven years. The majority had been bred in India. The disease was never observed in animals entering the country from other lands, but some Australian horses acquired it after their importation. The incidence of the malady varied considerably in different years, and fell chiefly in the cold and rainy seasons, 97 per cent. of the cases occurring between October and February ; yet no exact correspondence could be traced between rainfall and incidence.

The symptoms produced are epiphora, photophobia, blepharospasm, iritis, turbidity of the aqueous, and a diffuse keratitis. If the parasite be removed at an early period the corneal infiltration may clear, and some vision may be preserved. Otherwise the inflammation subsides, but vision is lost. Usually one eye only is affected, and contains only a single filaria, but as many as three have been observed.

PLATE XIX.

Fig. 1 illustrates Mr. George Coats's paper on A Parasite in the Eyelid of the Mouse (p. 390).

Fig. 1.— × 150. The encysted parasite with surrounding inflammatory infiltration. A portion of the Meibomian gland and some fibres of the orbicularis muscle are seen in the lower part of the figure.

Figs. 2 and 3 illustrate Lieut.-Col. W. G. Pridmore and Mr. George Coats's Demonstration of a Filaria from the Eye of the Horse (p. 387).

Fig. 2.— × 120. The head.

Fig. 3.— × 120. The tail.

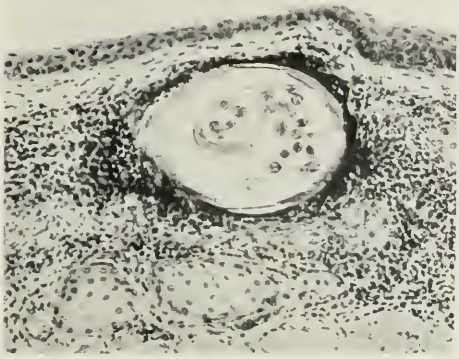

Fig. 1.

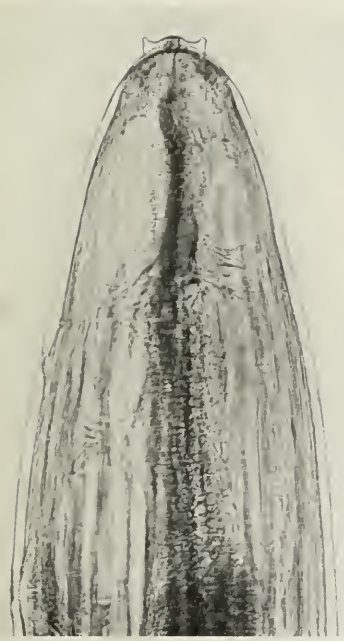

Fig. 2.

Fig. 3.

Adlard & Son, Impr.

The present specimen was obtained in Rangoon. It is 45 mm. in length, and somewhat less than 0·5 mm. at its greatest thickness. The body becomes slightly narrower towards the head, and thins off very considerably in the last quarter of its length. The integument is smooth, but with fine longitudinal markings, and has a very definite homogeneous cuticle. The mouth is furnished with projecting lips in the form shown in Pl. XIX, fig. 2. The first part of the alimentary canal is thin. It then expands considerably, and is filled with brownish contents. Seven millimetres backwards it suddenly becomes thin once more, and continues so in the rest of its extent. The extreme posterior end bends round in a hook-like curve, and is provided, just before its termination, with two small alar expansions, covered with cuticle (Pl. XIX, fig. 3). Ovaries or eggs are not distinguishable. Pre- or post-anal papillæ are not made out with certainty.

The above details correspond in the main with the characters of the ocular filaria of horses, as described by Neumann and quoted by Lingard, but differ in some particulars from the delineation of Devaine. According to Neumann the integument shows faint longitudinal markings; mouth small, round, guarded by two lateral lips semi-lunar in outline; œsophagus short, narrow, not bulbous; large intestine straight, one-eighth to one-fifth of the body-length, showing brownish contents; anus 130 μ to 160 μ from tip of tail in male, in female 300 μ to 350 μ. Three pre-anal and four post-anal papillæ. Measurement of male varies from 16 to 42 mm., average 33 mm.; of female from 28 to 43 mm. Ovaries contain no eggs. In the eye males preponderate, elsewhere in the body females.

Even yet there appears to be some doubt as to the exact classification of the filaria which infects the horse's eye, identification being difficult because the worm, in its unfavourable environment, does not attain maturity. Neumann and most other helminthologists believe it to be an immature form of *F. equina*, but others support the

claims of *F. papillosa*. However this may be, it is certain that the species in practically all cases is the same. Lingard's observation of two specimens which showed transverse striation and other aberrant features justifying a classification apart is quite exceptional. Curving of the tail and the presence of alar appendages are said by Braun (*Animal Paras. of Man*, p. 261, 1906) to be characteristic of the male.

It would seem, therefore, that the present specimen is probably an immature male of *Filaria equina*.

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3. *A parasite in the eyelid of the mouse.*

By George Coats.

(With Plate XIX, fig. 1.)

As a supplement to the foregoing paper it may be of interest to append a brief note on another nematode worm which was found by chance in the eyelid of a mouse.

Two encysted parasites are present in the subconjunctival tissues of the lower lid, one between the border of the tarsus and the fornix, one in the tarsal region, which, in the lower lid of the mouse, does not possess any very definite tarsal plate. The cyst is slightly oval, measuring about 130 μ by 160 μ. Its wall consists of a thin homogeneous envelope; beneath this there is a layer of granular material showing slight transverse striation, and interrupted at opposite points by four large cells, which project into the cyst cavity and contain each a large nucleus, a well-defined, deeply stained nucleolus, and a pale, loosely reticulated protoplasm. The much convoluted worm lies free within the cavity. The only distinguishable structures are a thin chitinous integument, beneath which some stained spots resembling nuclei are visible, and a granular

material which fills the rest of the body cavity ; no organs can be made out.

The cysts are situated in connective tissue, not among the muscle-fibres. They are surrounded by a good deal of round-cell infiltration, which spreads also among the tissues of the lid as far as the free border.

No parasites are present in the orbital tissues or in the orbital gland. The animal suffered from a severe fungating osteo-arthritis of the left ankle, but no parasites were found in the granulation tissue, nor elsewhere in the surrounding structures.

Prof. A. E. Shipley, F.R.S., of Cambridge University, who was good enough to examine the specimens, considers that the parasite is probably *Ollulanus tricuspis*. From an article in the *Encyclopædia Britannica* by that authority and Mr. F. E. Beddard, I learn that Ollulanus is one of those parasitic nematode worms which complete their life cycle in two hosts. The adult form occurs in the intestine of the cat, where the eggs are developed and the young hatched. In some cases the young penetrate the intestine of their host and become encysted in the lungs, liver, etc., but this is, as it were, a mere chance occurrence, and leads to no further phase of development. The propagation of the species depends on the young worms which are voided with the fæces. These are ingested by the mouse, find their way through the walls of the alimentary canal, become encysted in the muscles, and so remain quiescent till their new host is eaten by the cat, when they resume their development into the adult form. In its zoological position and life-history, therefore, Ollulanus is very nearly related to the better known *Trichina* or *Trichinella spiralis*, the cause of trichinosis.

APPENDIX.

The following cases and communications have also been brought before the Society:

1. Mr. John Attlee. Peripapillary Ectasia.

2. Mr. Rayner D. Batten. Collapsible Periscope.

REPORT OF COUNCIL.

The Council is happy to report the continued prosperity of the Society.

During the session 1914–15, 12 new members have been elected, 5 have died, and 5 have resigned. At present the Society has 494 Ordinary Members and 7 Honorary Members.

Among the deaths the Council records with regret that of Mr. W. A. Brailey, an original member and a trustee of the Society, who served twice on the Council, was secretary from 1883 to 1886, and became Vice-President in 1898. Dr. S. H. Habershon was also a member of the Council (1897 to 1900), and Secretary (1894 to 1897).

The Second Annual Congress of the Society, held on April 23rd, 24th, and 25th, 1914, was largely attended and highly successful. Prof. W. Uhthoff delivered the Bowman Lecture on "Ophthalmic Experiences and Considerations on the Surgery of Cerebral Tumours and Tower Skull." The great scientific value of Prof. Uhthoff's communication was recognised and appreciated by a numerous audience.

Thanks are due to the Committee of Management of the Central London Ophthalmic Hospital for the facilities which they extended to the Society in connection with the holding of its Clinical Meeting at their Institution, and to the Hon. Staff of the Hospital for the excellent dispositions which they made for the comfort and convenience of members; and to the Royal Society of Medicine for the very efficient manner in which the arrangements for the Congress were organised.

The Society has also to acknowledge its indebtedness

to Mr. Sydney Stephenson and to Dr. Emil v. Grosz who have made presentations to the Library.

The Nettleship Prize and Medal have been awarded to Mr. E. Treacher Collins in recognition of the value of his contributions to ocular pathology.

The Hon. Treasurer's report and statement of accounts are submitted, and show the financial condition of the Society, which is satisfactory in spite of the considerable increase in the cost of the last volume of *Transactions* as compared with that of the previous year. This increase was chiefly determined by the material of the Bowman Lecture, and by the numerous illustrations. For the first time no item for rent appears in the accounts. Although the balance is not quite so good as at the end of last year, a satisfactory financial position at the end of the current year may be anticipated with confidence if the membership is maintained or increased, and if the cost of the volume of *Transactions* as compared with that of last year is materially reduced.

ACCOUNT OF THE RECEIPTS AND PAYMENTS

OF THE

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM.

Receipts.

1914–15.	£	s.	d.	£	s.	d.
By Balance from last year				123	12	5
,, Subscriptions:						
Through Bank	91	14	6			
Through Collector	120	7	6			
			212	2	0	
,, Less refunded			1	1	0	
				211	1	0
,, Dividends:						
East India Railway Stock			17	12	1	
Ramsgate Corporation			14	0	7	
				31	12	8
,, Nettleship Prize Fund Dividend				8	5	0
,, Sale of 'Transactions'				34	10	9
				£409	1	10

Nettleship Prize Fund, £38 8s. 7*d*.

Payments.

1914–15.	£	s.	d.	£	s.	d.
To Printing 'Transactions'	214	17	2			
,, ,, Illustrations	76	18	0			
				291	15	2
,, Reporting				12	6	0
,, Expenses of Meeting				7	19	0
,, Secretaries Petty Cash				1	5	0
,, Collector				12	4	3
,, Bankers' Commission				0	5	10
,, Balance, including Nettleship Prize Fund				83	6	7
				£409	1	10

Examined and found correct.

E. ERSKINE HENDERSON,
R. AFFLECK GREEVES, } *Auditors.*

April 3rd, 1915.

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.

Acc.	Accommodation.
Aq.	Aqueous humour.
As.	Astigmatism.
A.C.	Anterior chamber.
C.	Cornea.
Ch.	Choroid.
cm.	Centimetre.
Cyl.	Cylindrical lens.
D.	Dioptre or dioptric.
E.	Emmetropia.
F.	Field of vision.
H.	Hypermetropia.
H.l.	Latent hypermetropia.
H.m.	Manifest hypermetropia.
I.	Iris.
L.	Left eye; and R., right eye.
m.	Metre.
mm.	Millimetre.
My.	Myopia.
M.L.	Macula lutea; and Y.S., yellow spot.
Oph.	Ophthalmoscope, ophthalmoscopic examination, ophthalmoscopic appearances.
O.D.	Optic disc.
O.P.	Optic papilla.
P.	Pupil.
Pr.	Presbyopia.
P.L.	Perception of light.
p.p.	Punctum proximum.
p.r.	Punctum remotissimum.
R.	Right eye; and L., left eye.
Ret.	Retina.
Scl.	Sclerotic.
Sph.	Spherical lens.
T.	Tension of the eyeball. T.n., tension normal. T. + 1, T. + 2, T. + 3, } degrees T. − 1, T. − 2, T. − 3, } of increase and decrease of tension.
Vit.	Vitreous humour.
Y.S.	Yellow spot; and M.L., macula lutea.
V.	Visus, acuteness of sight power of distinguishing form.

SYMBOLS.

+	Symbol for a convex lens.
−	Symbol for a concave lens.
′	Foot.
″	Inch.
‴	Line.

* See *Transactions*, vol. iv (1884), p. 365.

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