A handbook of diseases of the eye and their treatment / by Henry R. Swanzy.

Contributors

Swanzy, Henry R. 1843-1913.
Werner, Louis
University College, London. Library Services

Publication/Creation

London : H. K. Lewis, 1895.

Persistent URL

https://wellcomecollection.org/works/kq4zaaxj

Provider

University College London

License and attribution

This material has been provided by This material has been provided by UCL Library Services. The original may be consulted at UCL (University College London) where the originals may be consulted.
This work has been identified as being free of known restrictions under copyright law, including all related and neighbouring rights and is being made available under the Creative Commons, Public Domain Mark.

You can copy, modify, distribute and perform the work, even for commercial purposes, without asking permission.

wellcome collection

Wellcome Collection
183 Euston Road
London NW1 2BE UK
T +44 (0)20 7611 8722
E library@wellcomecollection.org
https://wellcomecollection.org

28116345 47

OPHTHALMOLOGY HC731 SWANZY [2]

DISEASES OF THE EYE.

A HANDBOOK

OF THE

DISEASES OF THE EYE

AND THEIR

TREATMENT

BY

HENRY R. SWANZY, A.M., M.B., F.R.C.S.I.

SURGEON TO THE NATIONAL EYE AND EAR INFIRMARY, AND OPHTHALMIC SURGEON TO THE ADELAIDE HOSPITAL, DUBLIN

FIFTH EDITION

WITH ILLUSTRATIONS

EDITED, UNDER SUPERVISION OF THE AUTHOR, BY

LOUIS WERNER, M.B., B.Ch.

ASSISTANT SURGEON NATIONAL EYE AND EAR INFIRMARY, AND OPHTHALMIC SURGEON TO THE MATER MISERICORDIÆ HOSPITAL, DUBLIN; EXAMINER IN OPHTHALMIC SURGERY IN THE UNIVERSITY OF DUBLIN, AND IN THE ROYAL UNIVERSITY OF IRELAND.

LONDON

H. K. LEWIS, 136 GOWER STREET

1895

London
H. K. Lewis, 136, Gower Street, W.C.

I DEDICATE THIS BOOK

TO

THEODORE LEBER,

PROFESSOR AT THE UNIVERSITY OF HEIDELBERG,

AS A MARK OF

MY ADMIRATION FOR HIS EMINENT SERVICES

TO OPHTHALMOLOGY,

AND OF

MY SINCERE REGARD.

Digitized by the Internet Archive
in 2014

https://archive.org/details/b21641250

PREFACE TO THE FIFTH EDITION.

The Fourth Edition of this book was published in October 1892.

In this Edition I have kept the same end in view as before, namely, the providing of a succinct account of the present state of our knowledge of Diseases of the Eye, and of the most approved methods for their treatment.

Owing to pressure of other literary work I have been obliged to enlist the aid of Dr. Louis Werner in the preparation of this Edition, and he has very kindly undertaken for me, under my supervision, the main portion of the labour connected therewith. I do not think the book will be found to have suffered in consequence, and my best thanks are due to him for the infinite pains and care he has taken in the task.

The book is larger by 44 pages. Amongst the additions to it may be mentioned: descriptions of the astigmometer and its use, of the effects of electric light on the eyes, of scintillating scotoma, of ophthalmia nodosa, and of enophthalmos. The articles on tubercle of the iris and on tumours of the optic nerve have been re-written. The most important changes are in Chapters XV. and XVII.,

the Diseases of the Retina having been grouped and arranged in more systematic order, while the account of the affections of the visual centres and path has been revised, and brought up to date in so far as the size of the book permits. Five new figures have been introduced, and two of the former figures omitted.

23, MERRION SQUARE,
December 1894.

PREFACE TO THE FOURTH EDITION.

The Third Edition of this book was published in October 1890, and I am gratified that the work continues to find favour, not only with students, for whom it is mainly intended, but also with practitioners.

The book has now again been revised throughout, and brought up to date.

In an Appendix, Holmgren's Method for Testing the Colour Sense has been described in greater detail than before.

Some new illustrations have been added.

The great difficulty of an author in the preparation of a book like this consists in "Saying not all he might, but all he ought." It is his duty to give a succinct and practical account of his subject in its most modern aspect, without weighting his pages with excessive detail and prolonged discussion. This has been my aim. For deeper and wider information, larger handbooks, and original monographs, must be consulted.

23, Merrion Square,
October 1892.

CONTENTS.

CHAPTER I.

PAGE

Some Elementary Optics—Numbering of Trial-Lenses and Spectacle Glasses—Normal Refraction and Accommodation—The Metre Angle—The Angle Gamma—The Sense of Sight (Light-Sense, Colour-Sense, Form-Sense)—The Field of Vision . . 1

CHAPTER II.

ABNORMAL REFRACTION AND ACCOMMODATION.

Hypermetropia—Correction of H.—Amplitude of Accommodation in H.—Angle Gamma in H.—Cramp of Ciliary Muscle in H.—Accommodative Asthenopia in H.—Internal Strabismus in H.—The Prescribing of Spectacles in H. 27

Myopia—Determination of Degree of M.—Amplitude of Accommodation in M.—Angle Gamma in M.—Complications of Progressive M.—Management of M.—The Prescribing of Spectacles in M. 33

Astigmatism—Symptoms of As.—Estimation of Degree of, and Correction of As.—The Astigmometer—Lental As.—Irregular As. 42

Anisometropia 55

Anomalies of Accommodation—Presbyopia—Paralysis of Accommodation—Cramp of Accommodation 56

CHAPTER III.

THE OPHTHALMOSCOPE.

Why Necessary—Helmholtz's Ophthalmoscope—Modern Ophthalmoscope—Direct Method—Indirect Method 62

Estimation of the Refraction by aid of the Ophthalmoscope—Direct Method—Retinoscopy 69

Focal Illumination 83

PAGE

The Normal Fundus Oculi as seen with the Ophthalmoscope—The Optic Papilla—The Retina—The Macula Lutea—The General Fundus Oculi—The Retinal Vessels 83

CHAPTER IV.

DISEASES OF THE CONJUNCTIVA.

Hyperæmia—Conjunctivitis—Catarrhal, or Simple Acute Conjunctivitis—Follicular Conjunctivitis—Spring Catarrh—Granular Conjunctivitis, Granular Ophthalmia, or Trachoma—Acute Granular Ophthalmia—Chronic Granular Ophthalmia—Lymphoma of the Conjunctiva—Acute Blennorrhœa of the Conjunctiva, or Purulent Ophthalmia—Croupous Conjunctivitis—Diphtheritic Conjunctivitis—Conjunctival Complication of Small-pox—Amyloid Degeneration—Tubercular Disease of the Conjunctiva — Lupus — Pemphigus — Xerophthalmos — Pterygium — Pinguecula — Subconjunctival Ecchymosis — Nævus — Polypus — Dermoid Tumours — Lipoma—Syphilitic Disease of the Conjunctiva—Papilloma, or Papillary Fibroma — Epithelioma — Sarcoma — Simple Cysts — Subconjunctival Cysticercus—Lithiasis—Uric Acid Deposits—Injuries of the Conjunctiva 89

CHAPTER V.

PHLYCTENULAR, OR STRUMOUS, CONJUNCTIVITIS AND KERATITIS

Solitary, or Simple, Phlyctenula of the Conjunctiva—Multiple, or Miliary, Phlyctenula of the Conjunctiva—Modes of Secondary Corneal Affection—Primary Phlyctenular Keratitis—Different Forms of Same—Symptoms of Phlyctenular Keratitis—Causes of Phlyctenular Ophthalmia—Treatment 136

CHAPTER VI.

DISEASES OF THE CORNEA.

Inflammations of the Cornea—(*a*) Ulcerative Inflammations of the Cornea—Simple Ulcer—Deep Ulcer—Ulcus Serpens—Rodent Ulcer—Marginal Ring Ulcer—Absorption Ulcer—Neuro-Paralytic Keratitis—Infantile Ulceration of the Cornea with Xerosis of the Conjunctiva—Herpes—Filamentous Keratitis—Bullous Keratitis—Dendriform Keratitis 145

PAGE

(*b*) Non-Ulcerative Inflammations of the Cornea—Abscess—Diffuse Interstitial Keratitis—Keratitis Punctata—Sclerotising Opacity—Ribandlike Keratitis 169
Ectasies of the Cornea—Staphyloma Corneæ—Conical Cornea . 176
Tumours of the Cornea 184
Injuries of the Cornea—Foreign Bodies—Losses of Substance . 184
Opacities of the Cornea—Nebula, Macula, Leucoma—Sclerotising Opacity—Arcus Senilis 186
Pigmentation of the Cornea 190

CHAPTER VII.

DISEASES OF THE EYELIDS.

Eczema—Herpes Zoster Ophthalmicus—Primary Syphilitic Sores—Secondary Syphilitic Sores—Vaccine Vesicles—Rodent Ulcer—Marginal Blepharitis (Ophthalmia Tarsi)—Phtheiriasis Ciliorum—Hordeolum (Stye)—Chalazion (Meibomian Cyst, Tarsal Tumour)—Millium—Molluscum—Nævus—Xanthelasma—Chromidrosis—Epithelioma, Sarcoma, Adenoma and Lupus—Tonic Cramp of the Orbicularis Muscle—Blepharospasm—Ptosis—Operations for its Cure—Lagophthalmos—Symblepharon—Blepharophimosis—Canthoplastic Operation—Distichiasis and Trichiasis—Operations for their Cure—Entropium—Operations for its Cure—Spastic Entropium—Senile Entropium—Operations for its Cure—Ectropium—Operations for its Cure—Ankyloblepharon—The Restoration of an Eyelid—Injuries—Ecchymosis—Epicanthus—Congenital Coloboma 191

CHAPTER VIII.

DISEASES OF THE LACRIMAL APPARATUS.

Malposition of the Punctum Lacrimale—Stenosis and Occlusion of the Punctum Lacrimale—Obstruction of the Canaliculus—Stricture of the Nasal Duct—Blennorrhœa of the Lacrimal Sac—Acute Dacryocystitis—Dacryoadenitis—Hypertrophy of the Lacrimal Gland—Adenoma, Adeno-Sarcoma, Lymphoma, and Tuberculosis 237

CHAPTER IX.

DISEASES OF THE SCLEROTIC.

PAGE

Inflammations of the Sclerotic—Episcleritis—Deep Scleritis—Injuries of the Sclerotic—Tumours of the Sclerotic—Pigment Spots 248

CHAPTER X.

DISEASES OF THE UVEAL TRACT.

Iritis—Simple Plastic Iritis—Serous Iritis—Parenchymatous (including Purulent) Iritis 254

Injuries of the Iris—Punctured Wounds—Foreign Bodies—Iridodialysis—Retroflection—Rupture of the Sphincter Iridis—Traumatic Aniridia—Anteversion—Traumatic Mydriasis . 265

New Growths of the Iris—Cysts—Granuloma—Tubercle (Miliary, Solitary)—Sarcoma—Ophthalmia Nodosa 267

Congenital Malformations of the Iris—Heterophthalmos—Corectopia—Polycoria—Persistent Pupillary Membrane—Coloboma Irideremia 270

Operations on the Iris—Iridectomy—Iridotomy 271

Cyclitis—Plastic Cyclitis—Serous Cyclitis—Purulent Cyclitis—Injuries of the Ciliary Body—New Growths of the Ciliary Body 275

Chorioiditis — Disseminated Chorioiditis — Syphilitic Chorioido-Retinitis—Central Senile Guttate Chorioiditis—Central Chorioiditis—Central Senile Atrophy of the Chorioid—Purulent Chorioiditis—Posterior Sclero-Chorioiditis—Detachment of the Chorioid 278

Injuries of the Chorioid—Foreign Bodies—Incised Wounds—Rupture—New Growths of the Chorioid—Sarcoma—Carcinoma—Tubercle—Sarcoma Carcinomatosum — Myosarcoma — Osteo-Sarcoma 284

Congenital Defects of the Chorioid—Coloboma—Albinismus . . 288

Sympathetic Ophthalmitis 289

CHAPTER XI.

MOTIONS OF THE PUPIL IN HEALTH AND DISEASE.

The Size of the Pupil in Health—Contraction of the Pupil—Dilatation of the Pupil 302

The Action of the Mydriatics and Myotics on the Pupil . . . 308

The Size of the Pupil in Disease—Myosis—Mydriasis . . . 309

CHAPTER XII.

GLAUCOMA.

PAGE

Primary Glaucoma—Chronic, or Non-Inflammatory, Glaucoma—Acute, or Inflammatory, Glaucoma—Glaucoma Fulminans—Sub-Acute Glaucoma—Etiology—Pathology—Treatment . 314
Secondary Glaucoma—Hæmorrhagic Glaucoma 336
Congenital Hydrophthalmos 338

CHAPTER XIII.

DISEASES OF THE CRYSTALLINE LENS.

Complete Cataracts—Senile Cataract—Progress, Pathogenesis, and Etiology—Treatment 339
Complete Cataract of Young People—Diabetic Cataract—Complete Congenital Cataract—Black Cataract 347
Partial Cataracts—Central Lental Cataract—Zonular, or Lamellar, Cataract—Anterior Polar, or Pyramidal, Cataract—Fusiform, or Spindle-Shaped, Cataract 349
Secondary Cataract—Posterior Polar Cataract—Total Secondary Cataract 351
Capsular Cataract 352
Traumatic Cataract 352
Operations for Cataract—Extraction of Cataract—Linear Extraction—The Modified Peripheral Linear Extraction—The Three Millimetre Flap Operation—Cataract Extraction without Iridectomy 355
Discission or Dilaceration—Suction Operation—Secondary Cataract and its Operation—Capsulotomy—Iridotomy 380
Dislocation of the Crystalline Lens—Lenticonus—Aphakia . . 385

CHAPTER XIV.

DISEASES OF THE VITREOUS HUMOUR.

Purulent Inflammation—Other Inflammatory Affections—Opacities—Muscæ Volitantes—Fluidity (Synchysis)—Synchysis Scintillans—Foreign Bodies—Cysticercus—Blood Vessels—Persistent Hyaloid Artery—Detachment 389

CHAPTER XV.

DISEASES OF THE RETINA.

PAGE

Alterations in Vascularity—Hyperæmia—Anæmia 400
Functional Affections—Hyperæsthesia—Neurasthenic Asthenopia, or Retinal Anæsthesia 400
Inflammation of the Retina or Retinitis—Syphilitic—Hæmorrhagic—Albuminuric—Diabetic—Leucæmic—Retinitis Punctata Albescens—Development of Connective Tissue or Retinitis Proliferans—Retinitis Circinata—Purulent Retinitis . . . 403
Atrophy of the Retina—Retinitis Pigmentosa 412
Diseases of the Retinal Vessels—Apoplexy of the Retina—Embolism of the Central Artery—Thrombosis of the Central Artery—Thrombosis of the Central Vein—Aneurism of the Central Artery—Sclerosis of the Retinal Vessels (Perivasculitis) . . 413
Injury to the Retina by Strong Light—Sunlight—Snow-Blindness—Effect of Electric Light on the Eyes 419
Tumour of the Retina—Glioma 422
Parasitic Disease of the Retina—Cysticercus 423
Detachment of the Retina 424
Traumatic Affections of the Retina—Traumatic Anæsthesia—Commotio Retinæ or Traumatic Œdema of the Retina . . 429

CHAPTER XVI.

DISEASES OF THE OPTIC NERVE.

Optic Neuritis (Papillitis), due to:—Cerebral Tumours—Tubercular Meningitis—Acute Myelitis—Hydrocephalus—Tumours of the Orbit—Inflammatory Processes in the Orbit—Exposure to Cold—Suppression of Menstruation—Chlorosis—Syphilis—Rheumatism—Lead Poisoning—Multiple Sclerosis—and to Hereditary, and Congenital, Predisposition 431
Chronic Retrobulbar Neuritis, or Central Amblyopia (Toxic Amblyopia)—Optic Neuritis Associated with Persistent Dropping of Watery Fluid from the Nostril 438
Atrophy of the Optic Nerve, due to:—Optic Neuritis—Pressure—Embolism of the Central Artery of the Retina—Syphilitic Retinitis, Retinitis Pigmentosa, Chorioido-Retinitis, and to Disease of the Spinal Cord (Spinal Amaurosis)—Optic Atrophy as a purely Local Disease 443

PAGE

Tumours of the Optic Nerve—Hyaline, or Colloid, Outgrowths—Injuries of the Optic Nerve—Amblyopia due to Hæmorrhages from the Stomach, Bowels, or Uterus 446

CHAPTER XVII.

AMBLYOPIA AND AMAUROSIS DUE TO CENTRAL AND OTHER CAUSES.

Hemianopsia—Arrangement of the Cortical Visual Centres, their Relations to the Retina, and the Course of the Optic Fibres between these two points—Localisation of the Lesion in Hemianopsia — Transitory Hemianopsia or Scintillating Scotoma—Alexia, or Word-Blindness — Visual Aphasia — Dyslexia—Amnestic Colour-Blindness—Visual Hallucinations —Mind-Blindness or Optic Amnesia—Congenital Amblyopia—Reflex Amblyopia — Hysterical Amblyopia — Nyctalopia — Uræmic Amblyopia—Glycosuric Amblyopia—Quinine Amaurosis—Pretended Amaurosis—Erythropsia 450

CHAPTER XVIII.

THE MOTIONS OF THE EYEBALLS, AND THEIR DERANGEMENTS.

Actions of the Orbital Muscles—Inclination of the Vertical Meridian in the Several Principal Positions—Muscles called into Action in the Several Principal Positions—The Field of Fixation—Strabismus 475

Paralyses of the Orbital Muscles—General Symptoms—Paralysis of the External Rectus—Paralysis of the Superior Oblique—Paralysis of the Internal Rectus, Superior Rectus, Inferior Oblique, and Levator Palpebræ—Ophthalmoplegia Externa, or Nuclear Paralysis—Cerebral Paralysis of Orbital Muscles—The Localising Value of Paralyses of Orbital Muscles in Cerebral Disease 481

Convergent Concomitant Strabismus—Causes—Single Vision in—Amblyopia of Squinting Eye—Clinical Varieties of—Measurement of—Mobility of Eye in—Treatment—Orthoptic Treatment—Operative Treatment—Tenotomy—Advancement of External Rectus—Dangers of the Strabismus Operation—Treatment Subsequent to Operation 510

Insufficiency of the Internal Recti, and Divergent Concomitant Strabismus—Muscular Asthenopia—Treatment — Operative Treatment 535

Nystagmus 541

CHAPTER XIX.

DISEASES OF THE ORBIT.

PAGE

Orbital Cellulitis—Periostitis of the Orbit—Caries of the Orbit—Injuries of the Orbit—Tumours of the Orbit—Diseases of Neighbouring Cavities—Hernia Cerebri—Exophthalmic Goitre—Enophthalmos 544

APPENDIX I.—Holmgren's Method for Testing the Colour-Sense . 561

APPENDIX II.—Regulations as to Defects of Vision which Disqualify Candidates for Admission into the Civil, Naval, and Military Services, the Royal Irish Constabulary, and the Mercantile Marine 564

INDEX 571

TO THE STUDENT.

You should read carefully Chapters i., ii., and iii., omitting at first the small print, either before or immediately on joining the Ophthalmic Hospital, or Department.

H. R. S.

DISEASES OF THE EYE.

CHAPTER I.

Some Elementary Optics—Numbering of Trial-Lenses and Spectacle Glasses—Normal Refraction and Accommodation—The Metre Angle—The Angle Gamma—The Sense of Sight (Light-Sense, Colour-Sense, Form-Sense)—The Field of Vision.

SOME ELEMENTARY OPTICS.

Refraction.—The light emitted by a luminous point is propagated in all directions in straight lines, which are called "rays." A ray of light passing from one medium into another of different density does not alter its course if it be perpendicular to the surface separating them; but, if it enter the second medium at an angle to the surface, it becomes deviated in its path, and is said to be "refracted." The phenomenon itself is called "refraction," and obeys a definite law.

A ray of light (*R A B*, Fig. 1) passing from air into glass (which of course possesses a greater density), and meeting the surface at an angle of incidence (*i*), becomes deviated *towards* the perpendicular *P*, forming the angle of refraction *r*. Now when the ray *A B* passes out into the less refracting medium air it deviates *away from* the perpendicular; and, if the surfaces of the glass be parallel, as in this case, the ray *B C* will be parallel to its original direction *R A*; but if, as in

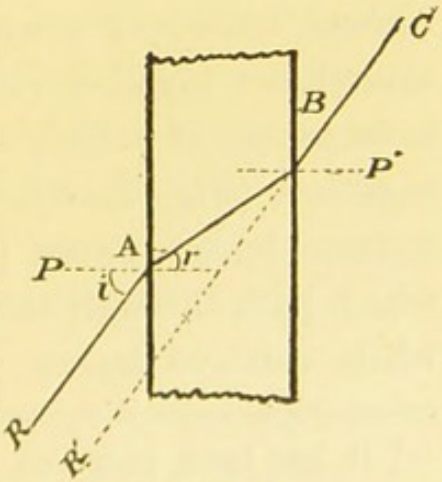


FIG. 1.

Fig. 2, the surfaces be inclined to each other, to form what is called a prism, then the ray of light will undergo refraction towards the base of the prism, at each of the surfaces, and an observer who is placed at *C*, so as to receive into his eye the emerging rays, *projects*, or thinks he sees, at *R'*, in a prolongation of *C B*, the object from which the rays come—that is, displaced towards the apex of the prism. The object does not appear to be altered in size, because a prism does not change the relation of rays to one another; if they were parallel, convergent, or divergent, before falling on it, they remain the same after their passage through it.

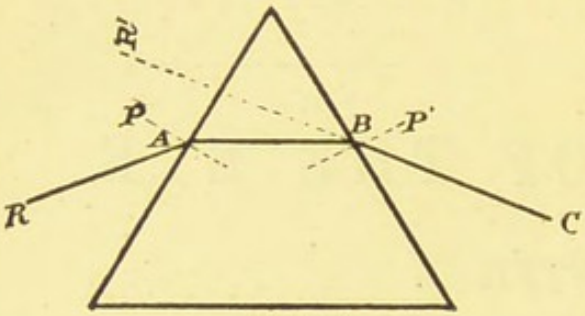


FIG. 2.

The deflection which a ray undergoes by passing through a prism increases with the size of the angle at the apex of the latter. In crown glass the deviation is about equal to half this angle. Prisms are described as being of 1°, 2°, 3°, etc., according to the size of this apex, or refracting angle.[1]

Convex and concave lenses may be regarded as being composed of prisms: convex lenses of prisms placed with their bases together (Fig. 3, *A*); concave lenses of prisms with their edges together (Fig. 3, *B*). Consequently, convex lenses cause pencils of rays which pass through them to converge, while concave lenses produce divergence of the rays.

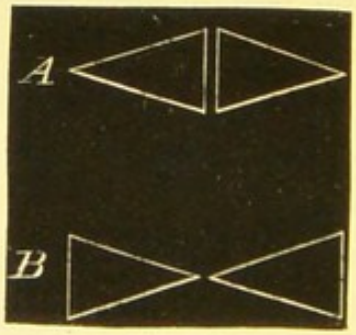


FIG. 3.

[1] It has been proposed to measure prisms for ophthalmic practice either according to their angle of minimum deviation, by prism-dioptries, or by metre-angles, but none of these methods seem to be free from objection, or to offer any marked advantage over the current method.

The Principal Axis of a lens is a line (*P F*, Fig. 4) passing through the centres of curvature of both its surfaces.

The Optical Centre of a lens is a point through which the rays must pass in order that they may not undergo deviation.[1] In a lens with equally curved surfaces it lies on the principal axis, midway between them.

Any rays passing through the optical centre, except the principal ray or axis, are called Secondary Rays.

All the other rays of the pencil undergo refraction.

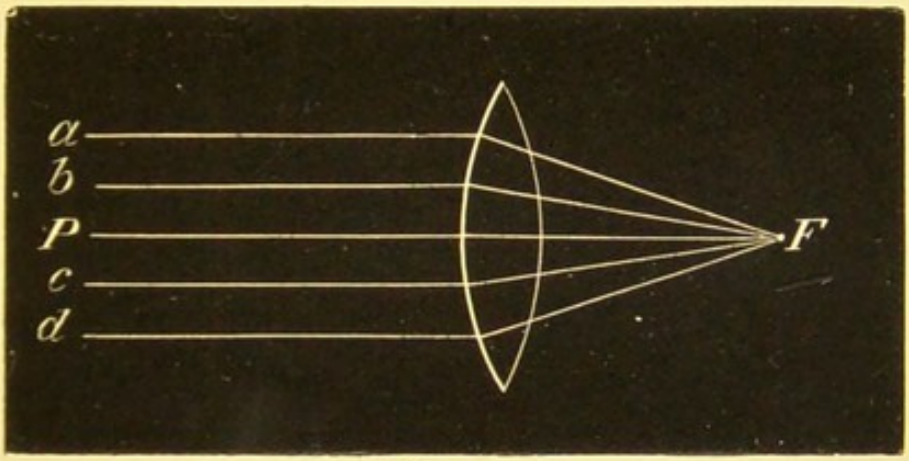


FIG. 4.

Convex lenses (Fig. 4) bring parallel rays of light (*a b P c d*, Fig. 4) passing through them to a focus at a point (*F*) a certain distance on the other side. This point is called the Principal Focus of the lens, and the distance from it to the lens is termed the Focal Length of the lens. The more curved the surface of the lens, the shorter will be its focal length, and the more "powerful" the lens. Rays diverging from a light placed at *F*, and falling on the lens, are made parallel when they reach its other side.

Divergent rays, *i.e.*, those coming from a near object (such

[1] Although sufficient for practical purposes, this is not theoretically correct, as all secondary rays passing through the optical centre are slightly deviated, but remain parallel to their original direction. Strictly speaking, the principal axis is the only one which undergoes no deviation.

as *O*, Fig. 5), do not meet at the principal focus of the lens, but at a point (*F'*) beyond it. This latter point is farther from the lens the nearer *O* is to the principal focus *F*, until,

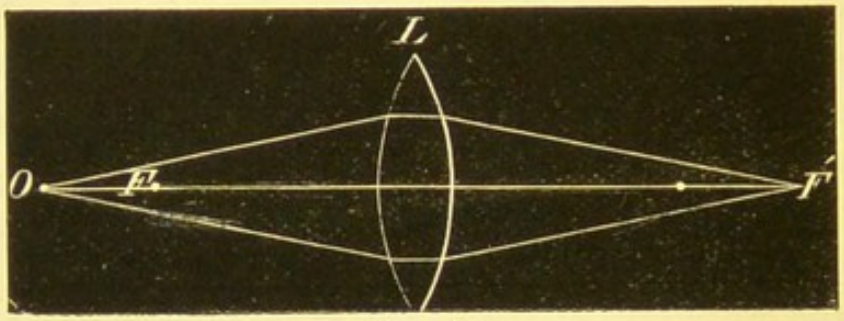


FIG. 5.

when *O* reaches *F*, *F'* becomes infinitely distant, and then the rays, after passing through the lens, are parallel. In like manner, rays from *F'* would focus at *O*, and hence these two points are termed Conjugate Foci.

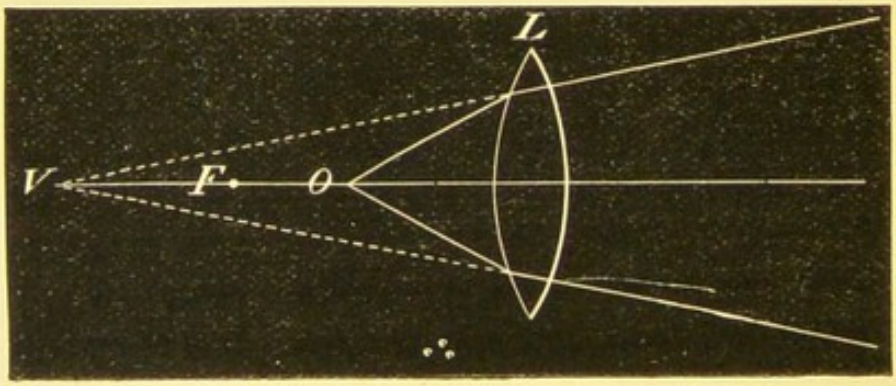


FIG. 6.

If the point (*O*, Fig, 6) from which the rays come be nearer the lens than its principal focus (*F*), they will not be made convergent, or even parallel, by the lens, but will remain divergent, although not so much so as before their entrance into the lens. If we imagine those still divergent rays to be prolonged backwards, they would meet at *V*, which would be called the Virtual Focus.[1] *O* and *V* are also conjugate foci,

[1] It is called "virtual" because there is no real convergence of the rays at that point; but to an observer placed on the other side of the lens (*L*), into whose eyes the rays fall, they would *seem* o come from that point.

and hence rays converging towards V from the other side of the lens will be united by the lens at O.

A Concave Lens (L, Fig. 7) makes parallel rays of light ($a\ b\ c\ d\ e$) divergent on passing through it; and if the direction of the divergent rays be prolonged backwards, they meet

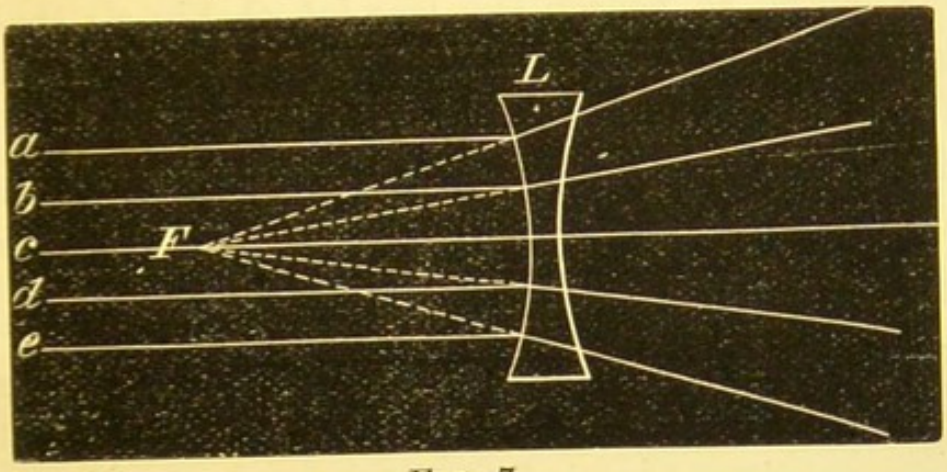


FIG. 7.

at a focus (F), which is therefore virtual, although it is the principal focus of the lens. In the case of concave lenses, then, there are only virtual foci.

When we speak of the *image* formed by a lens, we mean the collection of foci produced by it of pencils of rays coming from the various points of an object. For example: if $O\ B$ (Fig. 8) be the object, and a pencil of rays pass from its upper

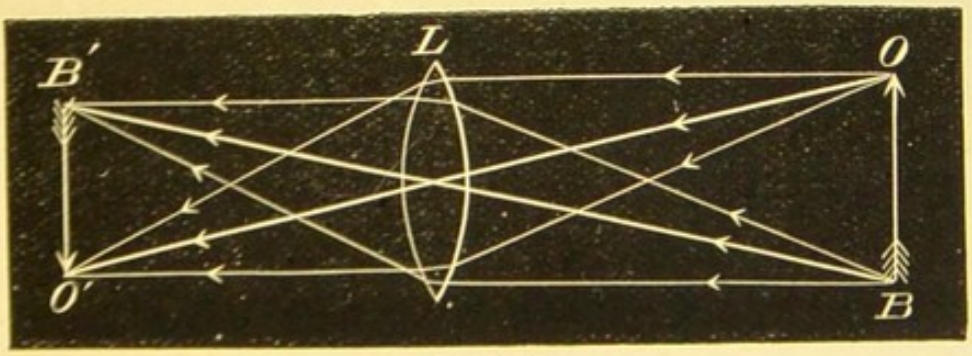


FIG. 8.

end O through the convex lens L, they will be united again at O' on the secondary axis $O\ O'$, and will form there an image of the point from which they come; while the rays from B will form an image of that point at B' on the secondary axis $B\ B'$. Similarly, images of all the points between O and B

are formed between O' and B'. Hence, the real images[1] formed by convex lenses are inverted.

But if the object be at the principal focus of the lens (at F, Fig. 4), the rays on emerging at the opposite side are made parallel, and the image is formed at an infinite distance.

If the object be nearer the lens (at O, Fig. 6) than the principal focus (F), the image will be an erect enlarged virtual one (at V) on the same side as the object.

With concave lenses the images are virtual and smaller than the object. In Fig. 9 the large arrow is the object, and F the principal focus of the lens. Rays passing through the

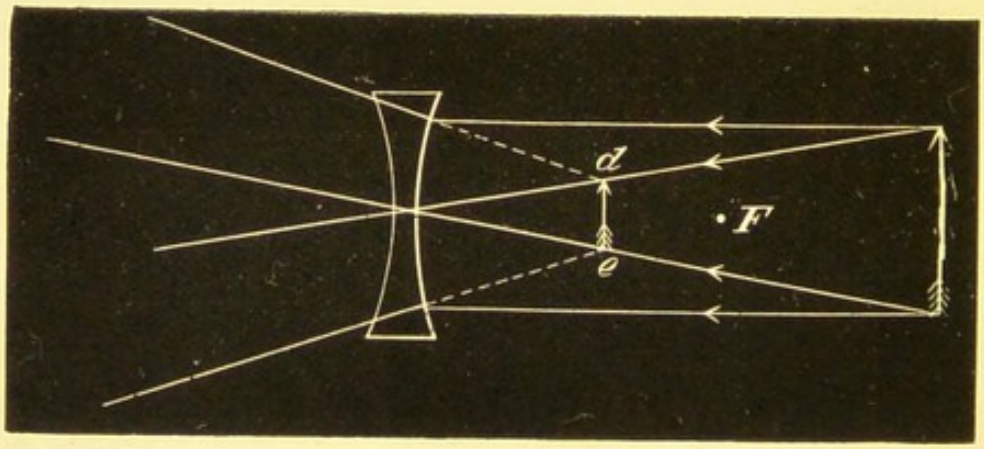


FIG. 9.

lens from the large arrow are made more divergent, and the image seems to be at a point (d e) found by prolongation backwards of the direction of those rays, after refraction.

It will be convenient for the reader that I should here describe the method in use for—

The Numbering of the Trial-Lenses.—The lenses in trial-cases, and in spectacles, are numbered according to the metrical system.

The lens of one metre (39½ inches) focal length is called the Dioptric Unit, or the Dioptry (1 D), of the metrical system. 2 D, 3 D, 4 D, etc., indicate the number of metre

[1] They are called "real" images because they have a real existence, and can, in fact, be caught upon a screen.

lenses, or dioptries, contained in each of these lenses. 2 D is therefore twice as powerful a lens (its focal length only half as long) as 1 D.

Convex lenses are indicated by the + sign placed before their number, thus, + 5 D; and concave lenses by the − sign, thus, − 5 D.

If it be required to ascertain the focal length of a given lens, divide 100 (1 metre = 100 centimetres) by the number of the lens, and the answer will give the focal length in centimetres. For example, the focal length of 10 D is $\frac{100}{10} = 10$ cm.

If the focal length of the lens be known, and it be desired to ascertain its dioptric number, we find it by dividing 100 cm. by the focal length. For example, if the focal length be 33 cm., then $\frac{100}{33} = 3$ D.

Reflection.—When a ray of light meets a polished surface it rebounds from it, or is "reflected" by it, changing its direction, and the phenomenon is termed Reflection.

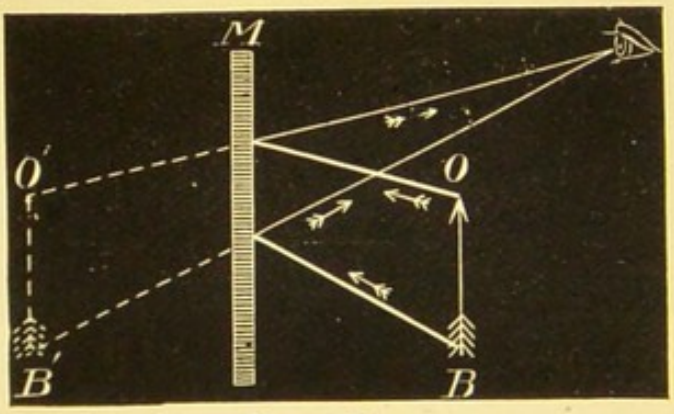


FIG. 10.

The images formed in plane mirrors are upright and virtual. If *O B* (Fig. 10) be the object, rays pass from it to the mirror *M*, and are reflected. Some of the reflected rays reach the eye of the observer, and there seems to him to be an upright image of *O B* formed at *O' B'* behind the mirror in a prolongation of the reflected rays, and at the same distance from the mirror as the object.

The images formed by concave spherical mirrors are inverted and real, provided the object be beyond the principal focus of the mirror.[1] For example, if *O B* (Fig. 11) be the

[1] As this is always the case in practical ophthalmoscopy—the source of light being always beyond the focus of the ophthalmoscopic mirror—it is the only condition considered here.

object, and *M* the mirror, the rays *O A* and *O C*, coming from *O*, will be reflected so as to meet at *O'*, and the rays *B E* and *B D*, coming from *B*, will be reflected to *B'*, and thus form a real inverted image *B' O'* in front of the mirror.

NORMAL REFRACTION AND ACCOMMODATION.

The eye is a dark chamber, containing a series of convex refracting surfaces—namely, the cornea, and the anterior and

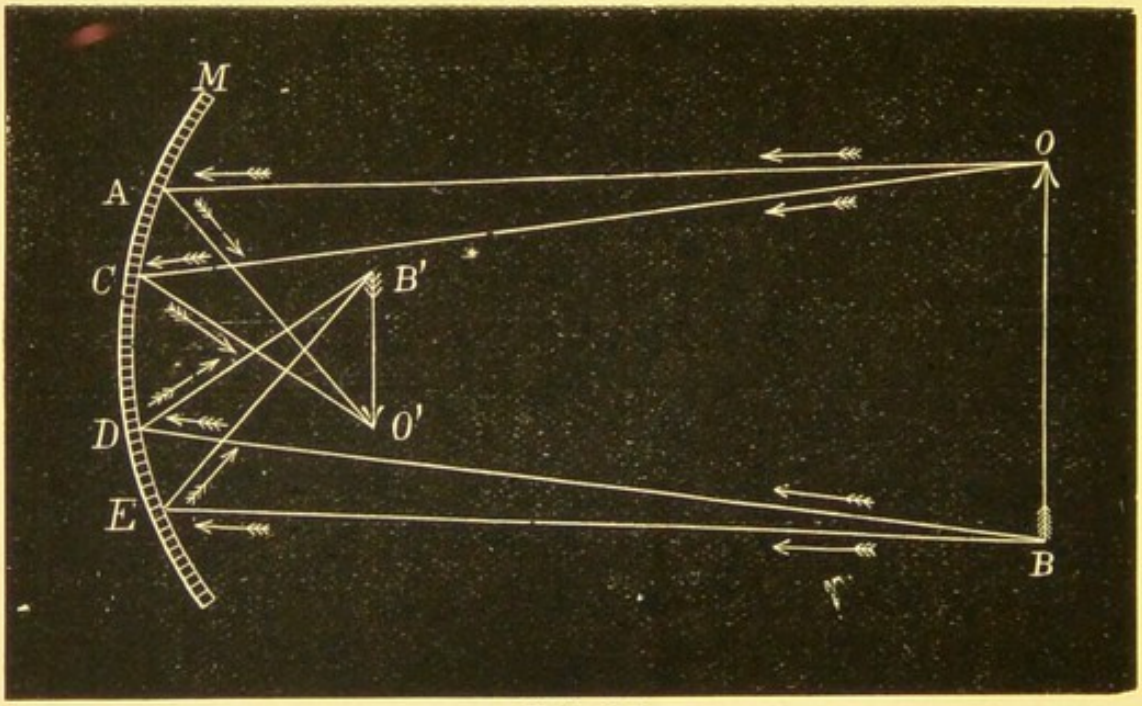


FIG. 11.

posterior surfaces of the crystalline lens; and certain "intraocular media"—namely, the aqueous humour, the substance of the crystalline lens, and the vitreous humour. By aid of this apparatus, which is called "the dioptric system of the eye," distinct inverted images of external objects are formed on the retina.

The refracting media are centred on the optical axis (*O A*, Fig. 12), a line which, passing through the optical centre (*N*) of the eye, meets the retina at a point (*A*) slightly to the inner side of the macula lutea (*M*).

In treating of the eye we have to consider two sets of

visual objects—viz., distant objects and near objects. Distant objects are those at 6 metres and more from the eye; near objects are those closer to the eye than 6 metres. For practical purposes, the rays which pass through the pupil, coming from any given point of a distant object, are as good as

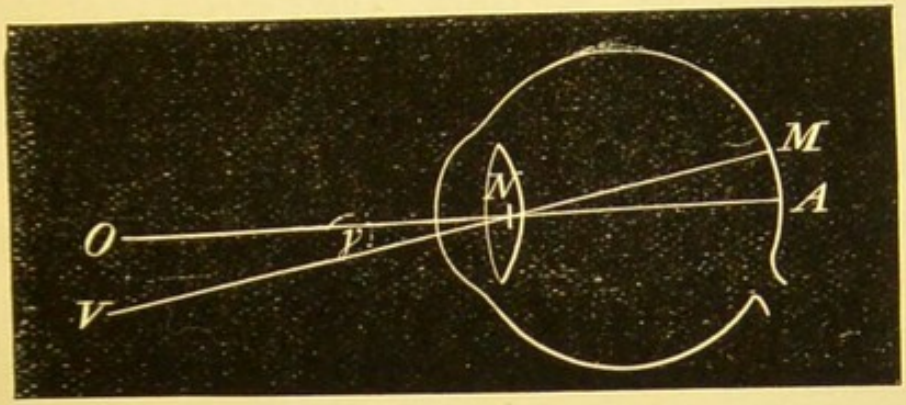


FIG. 12.

parallel, their divergence being so very slight when they reach the eye, and we regard them as being parallel.

REFRACTION.—By the Refraction of the Eye, is meant the faculty it has *when at rest* (*i.e.*, without an effort of accommodation) of altering the direction of rays of light which pass

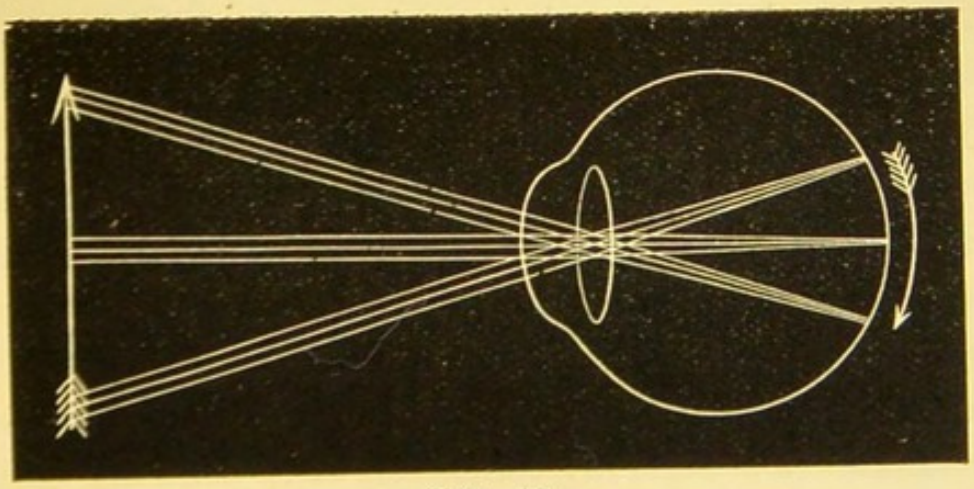


FIG. 13.

into it, making parallel rays convergent, and divergent rays less divergent.

In Normal Refraction, or Emmetropia (ἔμμετρος ὤψ), as it is termed, parallel rays (see Fig. 13, in which the object from which the rays come is supposed to be 6 metres or more from the eye) in passing through the dioptric media are given

such a convergence that they are brought to a focus on the layer of rods and cones of the retina, and form there a distinct inverted image of the point or object from which they come. In other words, the retina is placed at the principal focus of the dioptric system of the eye, which is thus adapted for parallel rays, and its "far point" (*vide infra*) is at infinity.

Accommodation.—But the eye can see near objects distinctly, as well as distant objects, although the rays from any given point (*a*, Fig. 14) of a near object reach the eye with a divergence so considerable that they could not be brought to a focus on the retina by the unaided refraction, but would

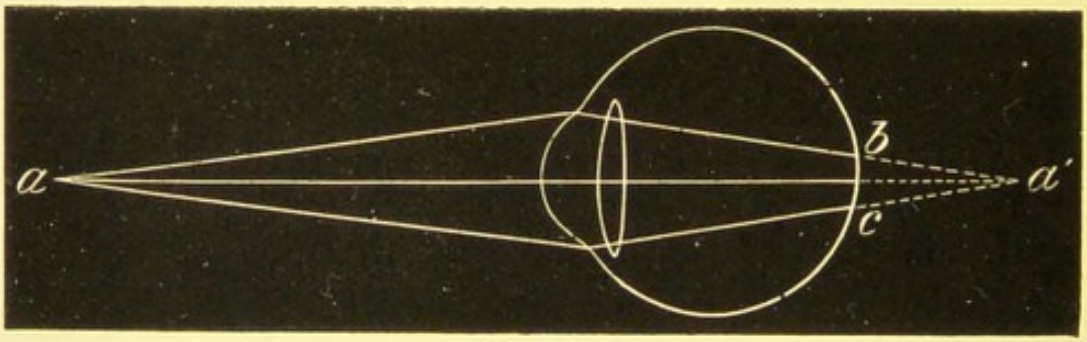


Fig. 14.

converge towards a point (their conjugate focus *a'*) behind the retina, and would not form a distinct image on the latter, but merely a blurred image or circle of diffusion (at *b c*). It is obvious, therefore, that an increase of refracting power in the eye is necessary, in order that near objects may be distinctly seen. It is this increase in the refracting power for the purpose of near vision which is called Accommodation.

The Mechanism of Accommodation is as follows:—The ciliary muscle (*m*, Fig. 15) contracts, thus drawing forward the chorioid and ciliary processes, and relaxing the zonula of Zinn (*z*), which is attached to the latter. The lens (*l*), which was flattened by the tension of the zonula, is now free to assume a more spherical shape, in response to its own elasticity. The posterior surface of the lens scarcely alters in shape, being fixed in the patellary fossa; but the anterior surface becomes more convex, thus increasing its refracting

power. Associated with the act of accommodation is a contraction of the pupil. The accompanying figure (Fig. 15) represents the changes which take place in accommodation, the dotted lines indicating the latter state.

The Far Point, and the Near Point.—It is possible for the eye to see objects accurately at every distance, from its Far Point—*i.e.*, its most distant point of distinct vision (Punctum Remotum,—R.), up to a point only a few centimetres from the eye, called the Near Point (Punctum Proximum,—P.).

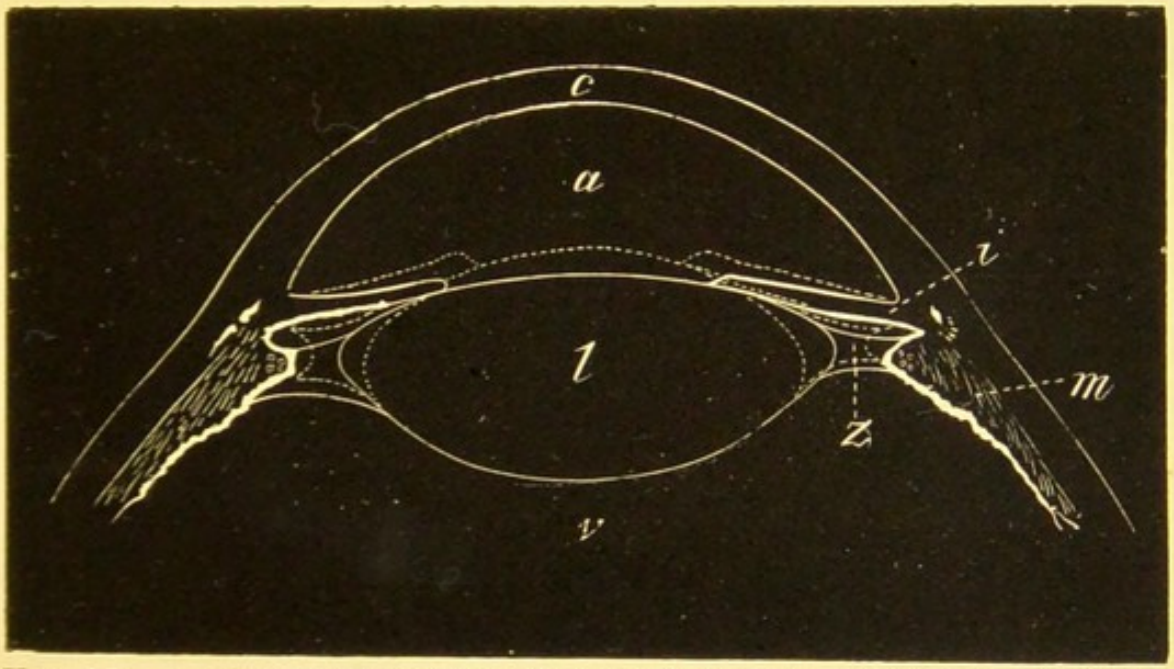


FIG. 15.—*c*, cornea; *a*, anterior chamber; *l*, lens; *v*, vitreous humour; *i*, iris; *z*, zonula of Zinn; *m*, ciliary muscle.

We can find the latter by directing the patient to look at a page printed in small type, and by bringing it slowly closer and closer to his eye, until a point is reached where he cannot distinguish the words and letters, which become blurred. A point very slightly more removed from the eye than this, where he can read distinctly, is the near point. Between the near point and the eye, vision is indistinct; because no effort of the ciliary muscle can produce the amount of convexity of the lens required for so short a distance.

The Amplitude of Accommodation.—This is the amount of accommodative effort of which the eye is capable—*i.e.*, the effort it makes in order to adapt itself from its far point R

up to its near point P. The amplitude of accommodation (a), therefore, is equal to the difference in the refracting power of the eye at rest (r), and when its accommodation is exerted to the utmost (p), as expressed by the formula $a = p - r$. It may be represented by that convex lens placed close in front of the eye, which would take the place of the increased convexity of the lens, or, in other words, which would give to rays coming from the nearest point of distinct vision a direction as if they came from the far point. The number of this lens expresses the amplitude of accommodation in a given eye.

For example: if, in an emmetropic eye (E, Fig. 16), the

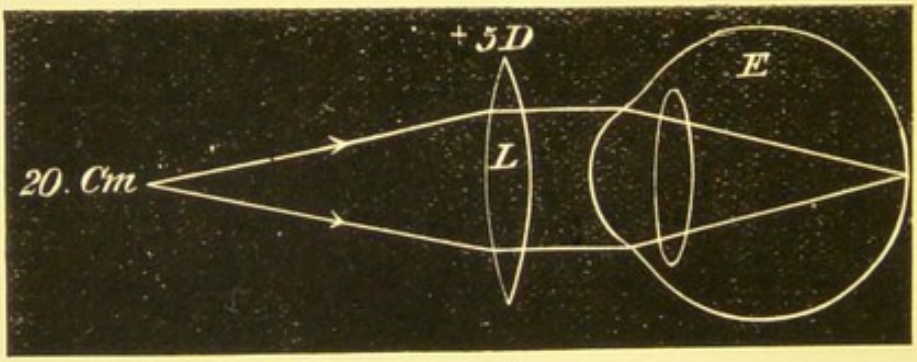


FIG. 16.

near point be situated at 20 cm., then a convex lens (L) of 20 cm. focal length placed close to the eye (between that point and the eye) would give to rays coming from the near point a direction (*i.e.*, would make them parallel) as though they came from a distant object, and this normally refracting eye would then be enabled, by aid of its refraction alone, to bring these rays to a focus on the retina. Making use of the above equation, we find in this case—since a focal length of 20 cm. represents a lens of 5 D—that $a = 5 - r$, but R being situated at infinity, we designate it by the sign ∞ ; hence, $r = \frac{1}{R} = \frac{1}{\infty} = 0$; therefore $a = 5 - 0 = 5$ D.[1]

[1] It must be observed that R represents the *distance* of the Far Point from the eye, while r represents the *refractive power* which is added to the eye by accommodation or by a lens, in order to adapt it for the distance R. Hence, it is evident that $r = \frac{1}{R}$, because the strength, or refractive power, of a lens is inversely as its focal length—*e.g.*, a lens of the strength of 4 D will

The amount of amplitude of accommodation (*i.e.*, the number of the lens which would represent it) is the same in every kind of refraction, according to the age of the individual, but in emmetropia alone is $a=p$ as above, because in it alone is $r=0$.

Under the head of "Anomalies of Accommodation," chap. ii., will be found Professor Donder's diagram representing the amplitude of accommodation at different ages.

Connection between Accommodation and Convergence (Relative Accommodation).—With every degree of convergence of the visual lines, a certain effort of accommodation is associated.[1] Thus, if the object be situated 2 metres from the eye, the visual lines converge to that point, and a certain effort of accommodation is made. But this connection between accommodation and convergence is somewhat elastic, for the accommodative effort may be increased or decreased, while the object is kept distinctly in view, and the same convergence maintained. That it may be increased is shown by the experiment of placing a weak concave glass before the eye, when it will be found that the object is still distinctly seen. And if a weak convex glass be then held before the eye, the object will also be clearly seen, showing that the accommodative effort may be lessened, without affecting vision or convergence. This amplitude of accommodation for a given point of convergence of the visual lines, found by the strongest concave and strongest convex glasses with which the object can still be distinctly seen, is called the Relative Amplitude of Accommodation. That part of it which is already in use, and is represented by the convex lens, is

---

have a focal length of $\frac{1}{4}$ that of a lens of 1 D—*i.e.*, $\frac{1\text{ m.}}{4}=\frac{100\text{ cm.}}{4}=0{\cdot}25$ cm. (see above, numbering of Trial-Lenses). Similarly, $p=\frac{1}{\text{P}}$ and $a=\frac{1}{\text{A}}$; P representing the *distance* of the Near Point, and A the focal length of the lens which represents the Accommodation.

[1] A common centre in the brain governs these motions, and contraction of the pupil.

termed the negative part; while the positive part is represented by the concave lens, and has not been brought into play. For sustained accommodation at any distance, it is necessary that the positive part of the relative amplitude of accommodation be considerable in amount.

Moreover, the convergence may be altered, while the same effort of accommodation is maintained, as is shown by the experiment of placing a weak prism with its base inwards before one eye. In order that the object may then be seen singly, it will be necessary for the eye before which the prism is placed to rotate somewhat outwards; and it will be found that the individual can do this while at the same time he sees the object with the same distinctness, showing that the same effort of accommodation has been maintained, although the angle of convergence of the visual axis is less than before.

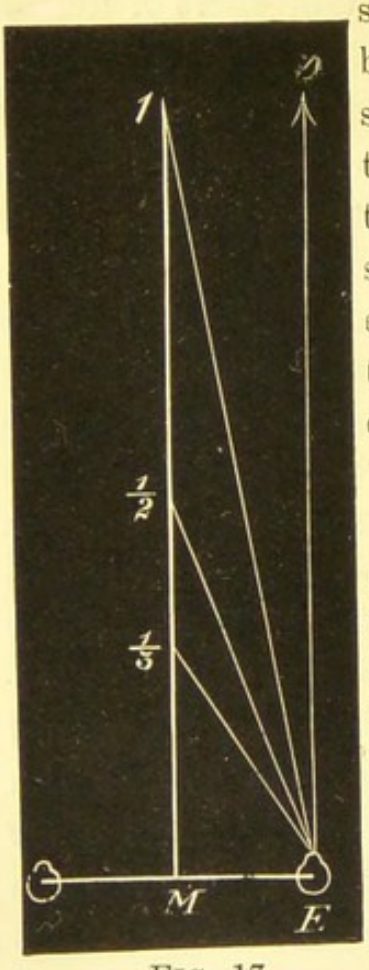


FIG. 17

THE METRE ANGLE.

If the visual line (*E* 1, Fig. 17) of an eye *E* have to be brought to bear on a point (1, Fig. 17) 1 metre distant from it in the median line (*M* 1), the angle of convergence (*E* 1 *M*) which the visual line thus makes with the median line is called the Metre Angle. It expresses the degree of convergence necessary for binocular vision at that distance, and is employed as the unit for expressing other degrees of convergence. If, for example, an object be situated ½ a metre (½, Fig. 17) from the eye, the angle of convergence (*E* ½ *M*) must be practically twice as large as at 1 metre: C. (Convergence) = 2 metre angles. If the object be only ⅓ of a metre distant, 3 metre angles are required: C. = 3 metre angles. If the object be situated 2 metres from the eye, the angle of convergence will be only one-half as great as at 1 metre, and here C. = ½ metre angle; while if the eye be directed towards a distant object (*D*), there will be no angle of convergence, and if the visual lines be divergent the metre angle will be negative.

Now, the average normal emmetropic eye requires, for each distance of binocular vision, as many metre angles of convergence as it requires dioptries of accommodation. For a distance of 1 metre an effort of accommodation of 1 dioptry is required, and also 1 metre angle of convergence; at $\frac{1}{3}$ metre from the eye 3 D of accommodation and 3 metre angles, and so on; while for distant objects neither angle of convergence nor effort of accommodation is required.

The Angle Gamma.

The Optic Axis is an imaginary line (P' P, Fig. 18) which passes through the centre (C) of the cornea and the posterior pole (P) of the globe, *i.e.*, a point situated between the macula lutea (M) and the optic papilla (D). *The Visual Line* (M O) unites the point of fixation (O)—the object looked at—with the macula lutea; it does not coincide with the optic axis, but crosses it at the principal optic centre (K) of the eye. *The Line of Fixation* (R O) joins the centre of rotation (R) of the eye with the point of fixation. *The Angle* γ is the angle $O R P'$ formed at the centre of rotation by the optic axis and the line of fixation.

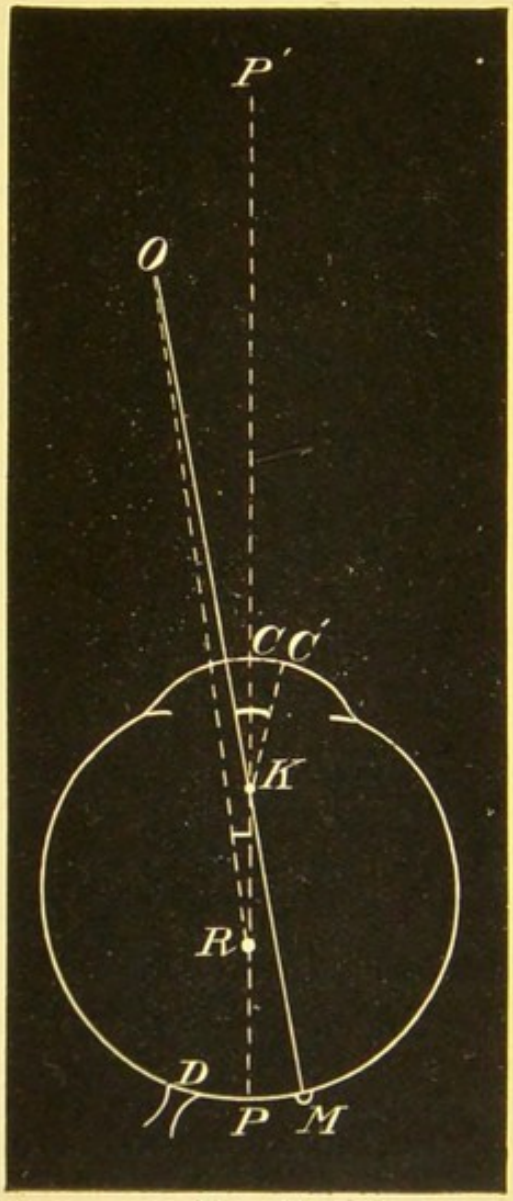


FIG. 18.

The line of fixation and the visual line so nearly coincide that in practice we regard them as identical; and hence, *in practice, the angle* γ *is the angle* $O K P'$. It should not be confounded, as is often the case, with *The Angle Alpha*, which is the angle $O K C'$ formed at the nodal point by the visual line and the major axis (C' K) of the corneal ellipse. This axis rarely passes through the centre of the cornea; but, as it never lies far from the latter, the difference in dimension between the two angles is very slight.

In order to measure the angle γ, the eye is placed at the perimeter as for an examination of its field of vision. By means of the corneal reflection of a candle-flame, which latter is moved along the arc of the perimeter, the centre of the cornea is found. The position of the flame at the perimeter then gives the angle γ. The average size of the angle γ is 5°.

THE SENSE OF SIGHT.

The Sense of Sight consists of three Visual Perceptions or Sub-Senses—namely, the Light-Sense, the Colour-Sense, and the Form-Sense. (See chap. xvii.)

The Light-Sense is the power the retina, or the visual centre, has of perceiving gradations in the intensity of illumination. The most convenient clinical method of testing the light-sense seems to be the photometer[1] invented by Messrs. Izard and Chibret. On looking through this instrument towards the sky two equally bright discs are seen. By a simple mechanism one of the discs can be made darker. If the eye does not perceive the difference in illumination between the two discs within 5°, its light-sense is abnormal; or we may say its L.D. (Light Difference) is too high. Again, if one disc be made quite dark, and be then gradually lighted, the patient is required to indicate the smallest degree of light, or L.M. (Light Minimum), by which he can observe the disc issuing from the darkness. This should not be more than 1° or 2°.

Another good method is that of Bjerrum, in which the light-sense is tested by grey letters on a white ground, the letters being constructed on the same principle as Snellen's (see Form-Sense).

In practical ophthalmology the light-sense is not yet of much interest; but it is stated that diseases primarily involving the nervous elements in the optic nerve show a tendency to defective L.D., while diseases primarily involving the chorioid and retina cause defective L.M.[2]

[1] To be had of Roulot, Paris.

[2] The Light-Sense and the Adaptation of the Retina, although related functions, must not be confounded one with the other. By the latter is meant the power the retina has of gradually adapting itself to see when the individual passes from a bright into a dim light. When it cannot do this with normal rapidity, or to a normal degree, the symptom called night-blindness results. It is quite possible for the light-sense to be normal, and yet for the retinal adaptation to be very defective, and *vice versâ*.

The Colour-Sense is the power the eye has of distinguishing light of different wave-lengths. According to the Young-Helmholtz theory the retina possesses three sets of colour-perceiving elements, those for Red, Green, and Blue or Violet. These are termed primary colours, all other colours being compounds of them.

According to Hering's theory, the colour-sense and the light-sense depend upon chemical changes in the retina, or in the "visual substances" situated in the retina. He suggests the existence of three different visual substances, the white-black, the red-green, and the blue-yellow, by the using up or "Dissimilation," and restoration or "Assimilation," of which substances the sensations of light and colour are produced. In the case of the white-black substance the sensation of white, or of light, corresponds to the process of dissimilation; while the sensation of black, or of darkness, corresponds to the process of assimilation. For the red-green and blue-yellow substances it cannot be said which colour-sensation implies assimilation, and which dissimilation. The members of the black-white pair can mingle with each other and with those of the other two pairs; but the respective members of the two colour pairs (being "contrast colours"), *e.g.*, blue and yellow, cannot unite with each other.

In testing the colour-sense the spectral colours are the best for exact experiments, but the difficulty of producing them at every moment, and of combining them, renders them of little clinical use.

The clinical method commonly employed for testing the colour-sense is that of Professor Holmgren, of Upsala, which is based upon the Young-Helmholtz theory. The test-objects used are coloured wools, of which a large number of skeins of every hue are thrown together.

Test I. (*vide* inside of end cover) consists in presenting to the individual, in good diffused daylight, a pale but pure green sample, and requiring him to select out of the bundle

of wools of all colours before him all of those samples which seem to him to correspond to the test sample. If he do this correctly, it is unnecessary to proceed further ; the individual has normal colour-sense. Amongst the skeins, however, there are some which are termed colours of confusion (greys, buffs, straw-colour, etc.) ; and if he select one, or several, of these, he is colour-blind.

If, now, we want to ascertain the kind and degree of his defect, we proceed to Test II.*a*. A pink (mixture of blue and red) skein is given to be matched. If this be correctly done, we term the person incompletely colour-blind. But if blue and violet, or one of them, be selected, he is red-blind (sees only the blue in the mixture of blue and red). If he select green or grey, or one of them, he is green-blind.

In order to corroborate the investigation we may employ Test II.*b*. A vivid red skein is given. The red-blind chooses, besides red, green and brown shades darker than the red ; while the green-blind chooses green and brown shades lighter than the red. But I believe myself, and I think it is now very generally recognised, that red-blindness and green-blindness invariably go together. In violet (or blue) blindness, purple, red, and orange will be confused in Test II.*a*, but this is an extremely rare variety of colour-blindness. Total colour-blindness will be recognised by a confusion of all shades having the same intensity of light, and is also rare. It is impossible by this test for any colour-blind person to escape detection, except in the case of a small central colour scotoma.[1]

The individual tested should not be allowed to name the colours, but merely to match them, as above described. The reason for this is twofold. First, because, although the congenitally colour-blind person is usually unaware of his defect, yet experience has taught him which of his sensations are

[1] See case reported by MacGillivray, *Brit. Med. Journ.*, July 1892.

called blue, red, etc., by other people; and hence he can often apply the right names to colours which he really does not see as such. He is assisted in this by whatever of colour-sight is left to him, and by the brightness and saturation of the different colours, but is liable to frequent mistakes. Again, when the colour-blind person does happen to know of his defect, he is often desirous of concealing it, either because he is ashamed of it, or from interested motives.[1]

A certain proportion of people (3·5 per cent. of men and less than 1 per cent. of women) are congenitally colour-blind, in greater or less degree, without any diminution in the other visual functions.

Acquired colour-blindness is found in toxic amblyopia, and in atrophy of the optic nerve.

The Form-Sense (Acuteness of Vision) is the faculty the eye possesses of perceiving the shape or form of objects, and, in clinical ophthalmology, the testing of this function is an important and ever-recurring duty.

In order that an eye may have good sight it is necessary, not only that its optic nerve, retina, chorioid, and refracting media be healthy, but also that its refraction and accommodation be normal. When applied to by a patient on account of imperfect sight, it is our first duty, as a rule, to ascertain accurately the condition of refraction and accommodation of his eyes. Should these be abnormal, and it be found that by aid of the correcting glasses perfect vision is obtained, we may in general conclude that the eye is organically sound, and that the patient's complaints are due to the defect in accommodation or refraction. If the glasses do not restore perfect vision, we must then, by the ophthalmoscope and other methods, decide the nature of the defect.

By Acuteness of Vision (V.) is meant the power which an eye, or rather its macula lutea, has of distinguishing form,

[1] More detailed information on colour-blindness and Holmgren's test will be found in Appendix I.

any anomaly of its refraction, if such exist, having been first corrected—*i.e.*, while the patient wears the correcting glasses.

Now, in order to measure the acuteness of vision, we must have a normal standard for comparison—*i.e.*, we must find what is the size of the smallest retinal image whose form can be distinguished. We cannot measure this image directly; but, as its size is proportional to the visual angle—the angle which the object subtends at the eye—it is sufficient to determine the smallest visual angle under which the form of an object can be distinguished. It has been found, experimentally, that the average size of this angle is 5′.

In order practically to ascertain the acuteness of vision, we place our patient with his back to the light, while facing

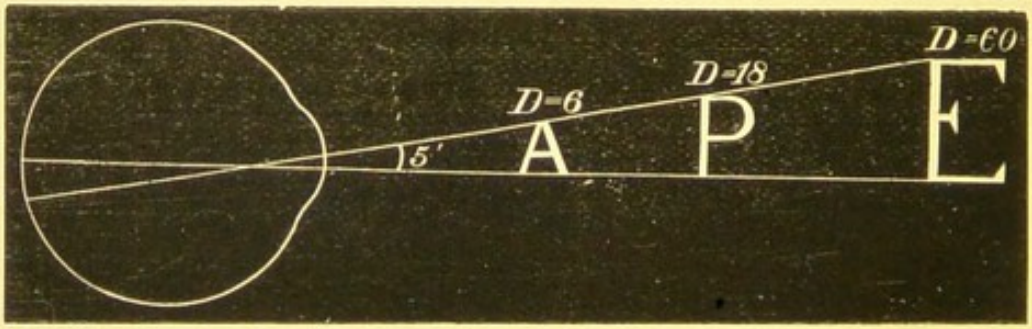


FIG. 19.

him, at a distance of 6 metres, and in good light, are placed Snellen's Test-Types for distance. These types are so designed that, at the distance at which they should be seen, they each subtend an angle of 5′ at the eye. The largest type should be seen at 60 metres (Fig. 19) by the normal eye, and the types range from this down to a size visible not farther off than 6 metres. If V = Acuteness of Vision, d = the distance from the eye to be tested to the test-types, and D = the distance at which the type should be distinguishable, then $V = \frac{d}{D}$. For example:—if d = 6 metres, a distance which most rooms can command, and if the eye see type D = 6, then $V = \frac{6}{6} = 1$, or normal V; but if at 6 metres the eye see only D = 60, which should be seen at 60 metres, then $V = \frac{6}{60}$, or very imperfect vision.

Should the patient's sight be so bad that he is unable to read any of the letters, it may be tested by trying at what distance he can count the surgeon's fingers ; and if he cannot even do that, then his power of perception of light, his "P.L.," should be tested. This is done by means of a lamp in a dark room, the eye being alternately covered and uncovered, and the patient being required to say when it is "light," and when "dark." If the flame be gradually lowered, the smallest degree of illumination perceptible will be ascertained.

The eyes must be examined separately, that one not under examination being excluded from vision by being shaded with the patient's own hand, or other suitable screen ; but it must not be at all pressed on, as any pressure would dim its vision when its turn for examination may come.

With the advance of age, the acuteness of vision undergoes a slight but steady reduction, owing to certain senile changes in the eye.[1]

THE FIELD OF VISION.

By the field of Vision (F.V.) is meant the space within which, when one eye is closed, objects can be seen by its fellow, the gaze of the latter being fixed the while on some one object or point. Thus if, standing on a hill, we fix the gaze of one eye on some object on the plain below, the field of vision includes not only that object, but many others also for miles around it. If the fixation object be nearer to us, the area taken in by our field of vision will be proportionately diminished in extent.

The fixation object is seen by central or direct vision, its image being formed on the macula lutea ; the other objects in the field of vision correspond with as many different points in the more peripheral parts of the retina, and are seen by

[1] *Von. Græfe's Archiv.*, xxxix., II., p. 71.

eccentric, or indirect, vision. Eccentric vision is of great importance for the guiding of ourselves and avoiding obstacles in our way. Its use may be realised by the experiment of looking through a long small-bore cylinder (*e.g.*, a roll of music) with one eye, thus cutting off its eccentric field, while the other eye is closed.

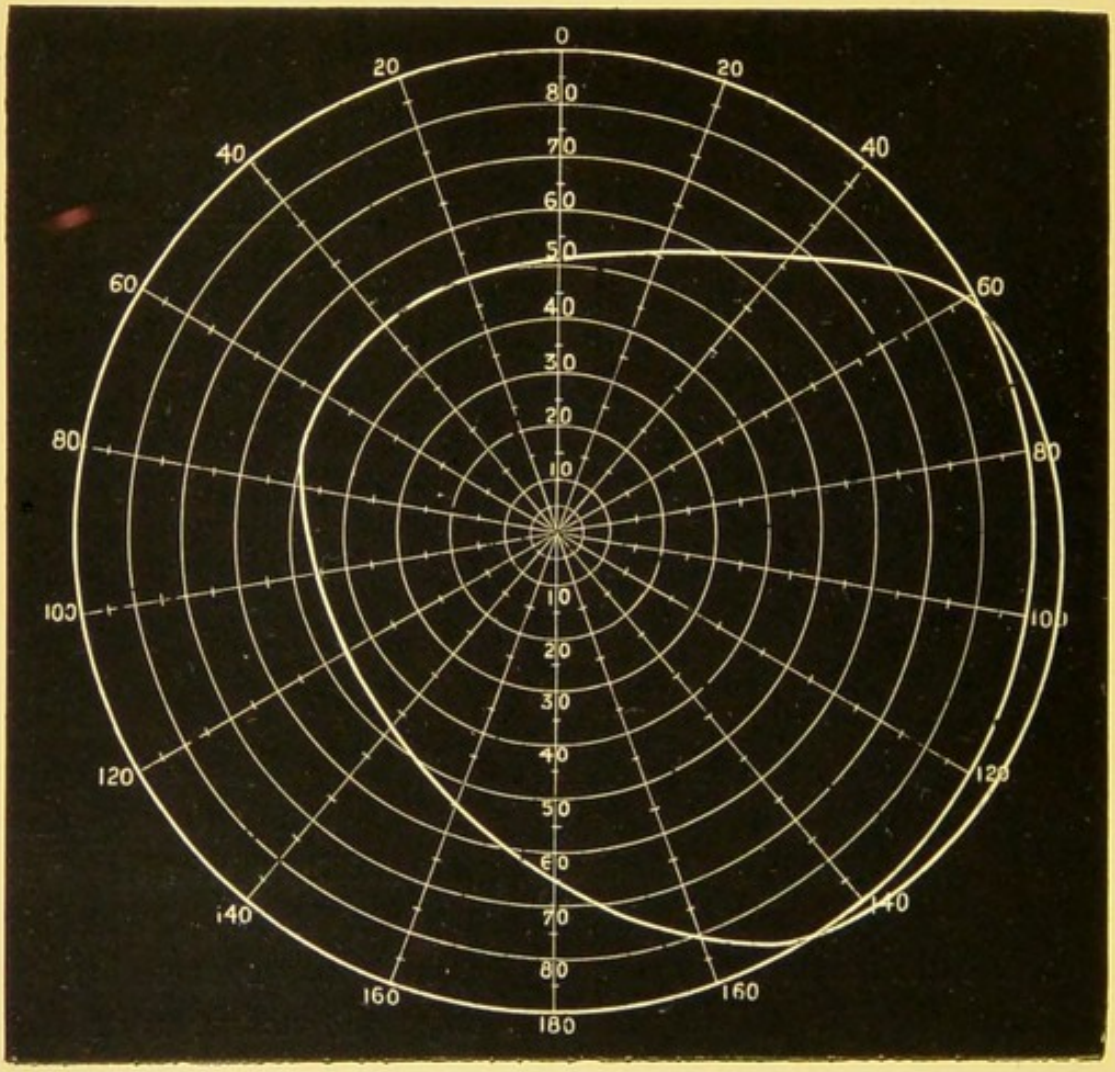


FIG. 20.—Chart of F.V. of Right Eye.

The Dimensions of the Field of Vision may be measured by means of an instrument called the perimeter. This is a semicircular metal band, which revolves upon its middle point, being in this way capable of describing a hemisphere in space. The arc is divided into degrees marked on it, from 0° placed at its middle point, to 90° at either extremity. At the centre of

the hemisphere is situated the eye under examination, while the fixation point is placed exactly opposite, in the centre of the semicircle. A small square bit of white paper, the test object, is slowly moved along the inner surface of the arc from the periphery towards the centre, until it comes into view. The horizontal, vertical, and two intermediate meridians, at the least, should be examined by placing the arc of the perimeter in the corresponding planes. The boundary of the field may

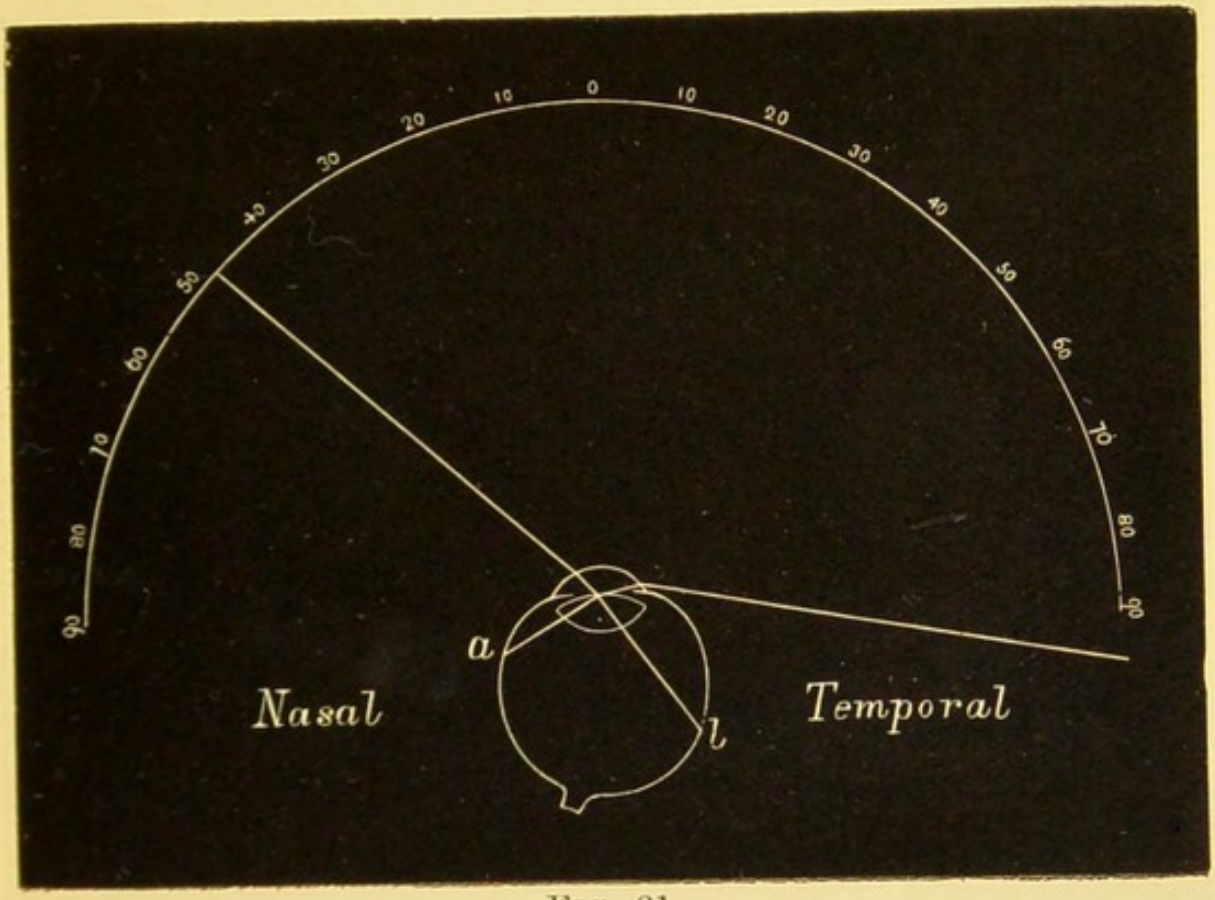


FIG. 21.

be noted on a diagram or chart (Fig. 20), which represents the projection of a sphere on a plane surface.

The radii represent different meridians, which may be determined by a dial with pointer on the back of the perimeter, while the concentric circles correspond with the degrees marked on the arc. A pencil mark is placed on the chart at the spot corresponding to that on the perimeter at which the test object comes into view, and, when the different meridians have been examined, these marks are united by a continuous line, which then represents the outer boundary of the F.V.

The normal F.V. is not circular, but extends outwards about 95°, upwards about 53°, inwards about 47°, and downwards about 65°, as represented by the strong curve in Fig. 20. The limitation upwards and inwards is partly due to the projection of the supra-orbital margin and the bridge of the nose,

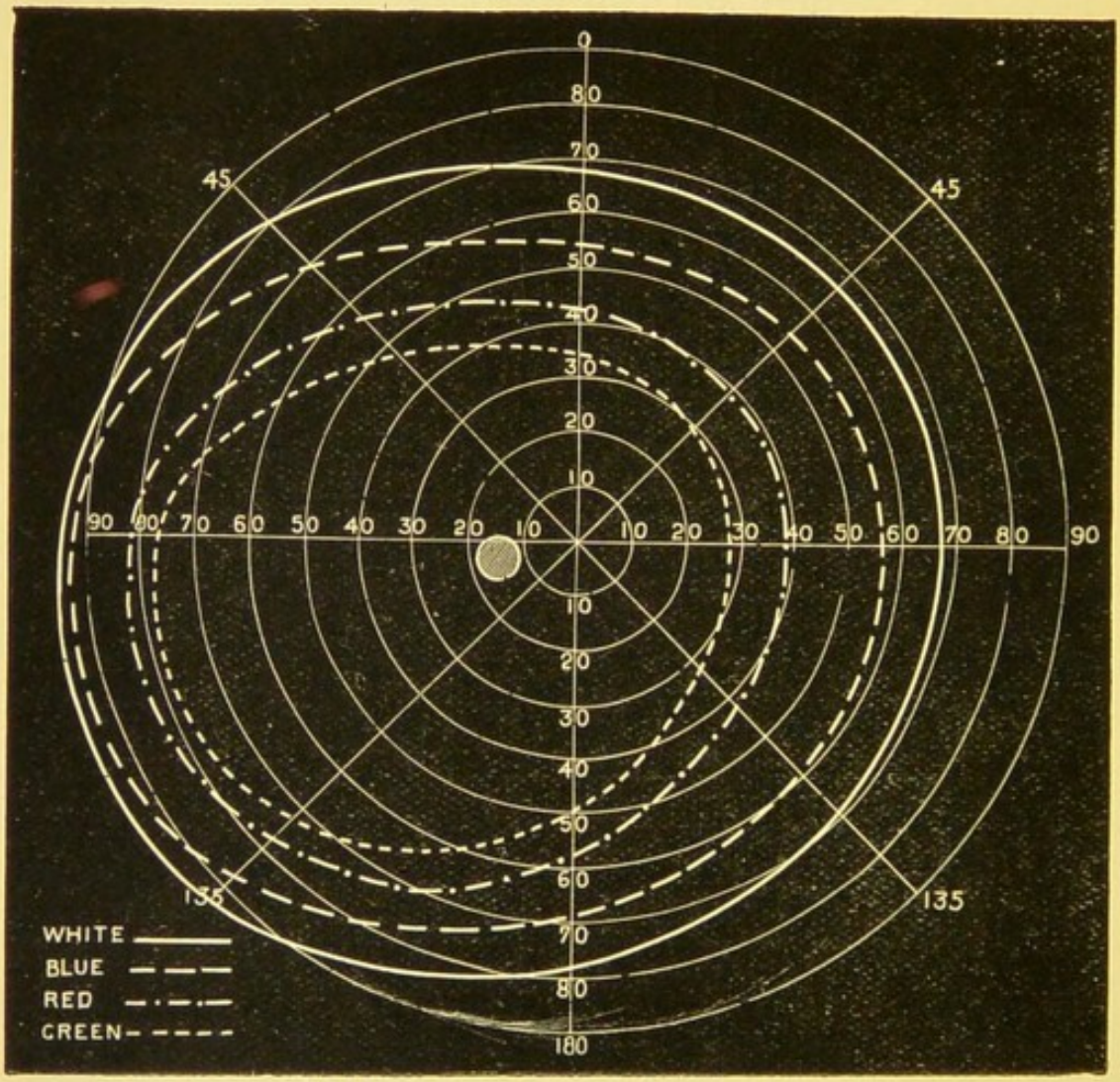


FIG. 22 (*Landolt*).—Chart of F.V. of Left Eye.

but also to the fact that the outer and lower parts of the retina are less practised in seeing than are the upper and inner parts, and their functions consequently less developed. The acuteness of vision diminishes progressively towards the periphery of the field, two points of a certain size close together being distinguishable from each other only a short

distance from the fixation point, while the farther towards the periphery the larger must be the test objects.

Fig. 21 serves to illustrate the projection of the field of vision on the semicircle of the perimeter to its extreme temporal (95°) and its extreme nasal (47°) boundaries, as well as the portion of the retina (*a* to *b*) which corresponds to this extent of field, and it shows that the sensitive portion of the retina, or rather perhaps the portion of the retina which is most used, extends farther forward on the nasal than on the temporal side. The diagram also explains the

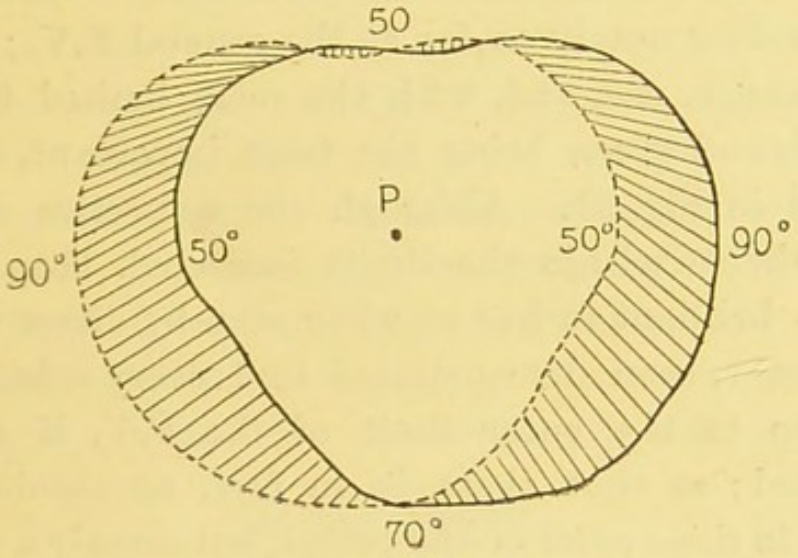


FIG. 23.—Binocular Field of Vision.

remarkable fact that the field extends in the temporal direction more than 90°. It must be remembered that the fields of vision overlap, as the two visual axes meet at the fixation point. Fig. 23 represents the binocular portion white, P. being the fixation point.

The Blind Spot of Mariotte is a small blind island, or scotoma, situated about 15° to the outer side of the point of fixation, and just below the horizontal meridian. It is shown as a white spot in Fig. 22. It is due to the optic papilla, for at that place the outer layers of the retina are wanting, and hence there is there no power of perception. There are also, occasionally, minute blind spots in the field due to large

retinal vessels, which interfere with the formation of the image upon the layer of rods and cones.

The Perception of Colours in the Periphery of the Field can be examined with the perimeter, by means of bits of coloured paper 4 mm. square. It has been in this way ascertained that the boundaries of the power of eccentric perception for the different colours do not seem to correspond with the boundary for white light, nor do the boundaries of the different colours coincide. Examining from the periphery towards the centre by ordinary daylight, blue is the colour which can be distinguished as such most eccentrically, its field extending nearly as far as the general F.V.; then come yellow, orange, red, and, with the most limited field, green. Blue, red, and green being the most important, their fields are noted in Fig. 21. Although the respective colours are distinguishable within the limits indicated, they are by no means so brilliant in hue as when seen by direct vision. It has, however, been demonstrated that every colour is recognisable up to the outer limit of the F.V. if sufficiently illuminated; so that there is, in fact, no absolute colour-blindness in these parts of the retina, but merely a diminished sensitiveness to coloured light.

The Perception of Form in the Periphery of the Field is very defective, and its examination is not of much practical importance, but this portion of the field is very sensitive to movements.

CHAPTER II.

ABNORMAL REFRACTION AND ACCOMMODATION.

I HAVE explained what is meant by Normal Refraction, or Emmetropia (ἔμ-μέτρον, *the standard*; ὤψ, *eye*). We recognise three different forms of Abnormal Refraction, or Ametropia (*a*, *priv*; μέτρον, *standard*; ὤψ). 1. Hypermetropia (ὑπερ, *over*; μέτρον, *standard*; ὤψ), in which the principal focus of parallel rays of light lies behind the retina. 2. Myopia (μύειν, *to close*; ὤψ), or Short-sight, in which the principal focus of such rays lies in front of the retina. 3. Astigmatism (*a*, *priv.*; στίγμα, *a point*), in which the refraction of the eye in its different meridians is different.

HYPERMETROPIA.

In a large proportion of cases, this form of Ametropia is due to the eyeball being too short in its antero-posterior axis (Axial H). It may also depend upon deficient refracting power in the dioptric media (Curvature H).

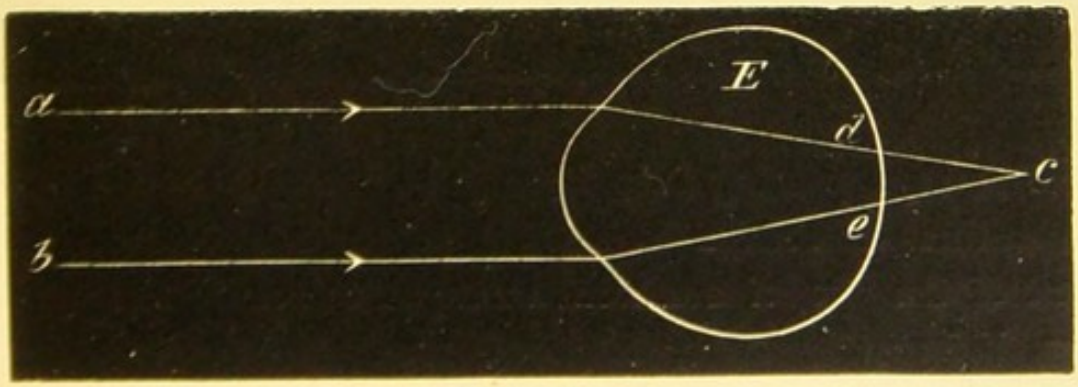


FIG. 24.

Parallel rays of light falling into the hypermetropic eye (*E*, Fig. 24) do not meet on the retina, but converge towards

a point (*c*) situated behind it. Consequently, these rays do not form on the retina a distinct image of the object looked at, but produce there a "circle of diffusion" (*d e*), or blurred representation of the object.

Since, therefore, in hypermetropia the retina is in front of the principal focus of the dioptric system, rays passing out of

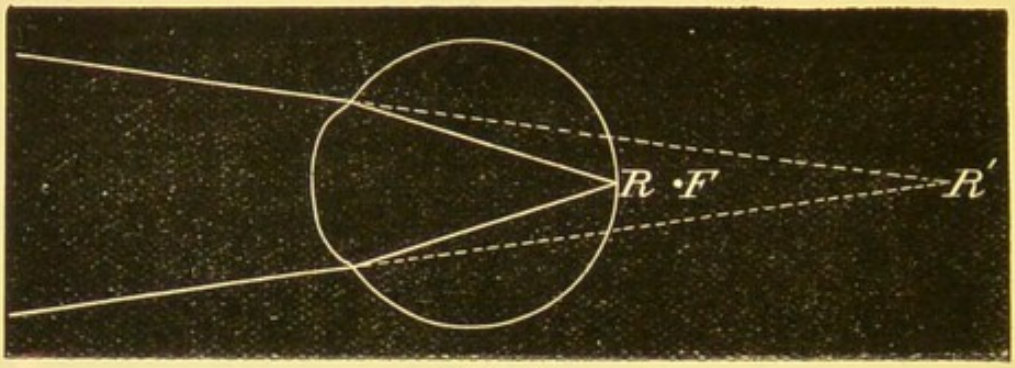


FIG. 25.

the eye from any point (*R*, Fig. 25) on this retina will pass out as divergent rays, and will appear to come from a point (*R'*) situated behind the eye, which point is the virtual conjugate focus of the point *R* (compare Fig. 6).

Now, in order to correct the hypermetropia—so that

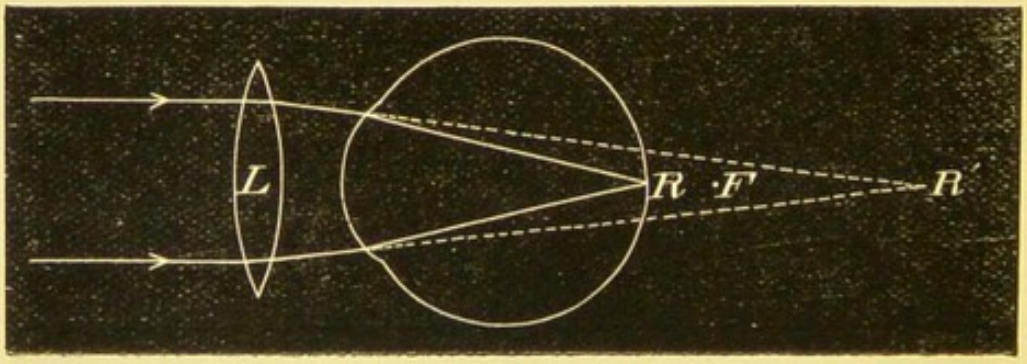


FIG. 26.

parallel rays passing into it may be brought to a focus on the retina—a convex lens (*L*, Fig. 26) must be placed in front of the eye, of sufficient strength to render the parallel rays, before they enter the eye, convergent towards *R'*, so that, when they meet the eye, they may be brought to a focus on the retina *R*, which is the conjugate focus of *R'*. The higher the hypermetropia, *i.e.*, the shorter the antero-

posterior axis of the eyeball, the stronger must the correcting glass be. It may be found that, with a lens of some dioptries less power, the eye will see equally well; but this it does by means of an effort of accommodation, which supplements the inadequate refracting power of the lens placed before it. As we proceed to higher lenses, the effort of accommodation is relaxed, until, finally, the strongest lens with which vision is still at its best is reached, when, it may for the present be assumed, no further effort of accommodation is made, and *L* represents the whole error of refraction. In low degrees of hypermetropia the eye can frequently see distant objects distinctly by an effort of accommodation, which completely takes the place of *L*. When such an eye is found to have full vision without a glass, a beginner might fall into the error of regarding it as emmetropic; but if he take the precaution of placing a low convex lens in front of it, and then find that the acuteness of vision—the effort of accommodation being now relaxed—remains as good as without the glass, he will avoid the mistake.

If a glass a single number higher than the exact measure of the defect be placed before the eye, vision again becomes indistinct, because the rays are then brought to a focus in front of the retina, and a circle of diffusion is formed on the latter. The eye, in fact, is put by such a glass in a condition of myopia. Therefore, *the strongest convex glass with which a hypermetropic eye can see distant objects* (*the test-types*) *most distinctly is the glass which corrects its hypermetropia, and is the measure of the latter.* Very commonly it is only the manifest hypermetropia (*vide infra*) which is ascertained by this method, unless the accommodation has been previously paralysed by atropine.

This method of determining the refraction by means of the trial-lenses and test-types is not relied on nowadays by ophthalmic surgeons to the same extent as formerly, the examination of the upright ophthalmoscopic image, or else retinoscopy,

having largely taken its place. In conjunction with these it is a valuable method.

The degree of the hypermetropia is indicated, as has been said, by the number of the lens which corrects it.[1] Thus, if the number of the glass L (Fig. 26) required to correct the hypermetropia of the eye E be 2·0 D, we say this eye is hypermetropic two dioptries, or has a hypermetropia of two dioptries or we would write it down, (H = 2·0 D).

Amplitude of Accommodation in Hypermetropia.—When at rest the refraction of the hypermetropic eye is deficient; consequently r must be negative ($-r$), and the amplitude of accommodation must include the power required to adapt the eye to infinity; therefore—

$$a = p - (-r) = p + r.$$

For example: if the punctum proximum of a hypermetropic eye of 5 D be at 30 cm., what is the amplitude of accommodation? 5 D ($= r$) is necessary in order to make the eye emmetropic, and to accommodate the emmetropic eye to 30 cm. 3·25 D ($\frac{100}{30} = 3{\cdot}25$) is required. Hence $a = 3{\cdot}25 + 5 = 8{\cdot}25$ D.

The Angle γ in Hypermetropia.—In hypermetropia, as in emmetropia the cornea is cut to the inside of its axis by the visual line; but in hypermetropia the angle which the visual line forms with the axis of the cornea is very much greater, owing to the shortness of the eyeball, the effect of which is to increase the distance between the macula lutea (M) and the optic axis (A) (Fig. 27). Consequently, in extreme cases, when the visual lines of a hypermetropic individual are directed to an object, the axes of the corneæ may seem to diverge, and thus the appearance of a divergent strabismus will be given (apparent strabismus, see chap. xviii.).

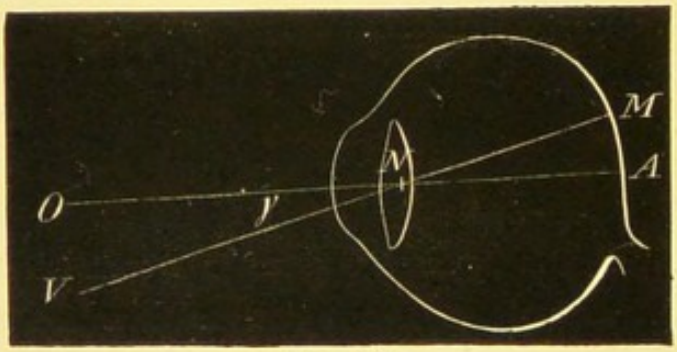


FIG. 27.

[1] Theoretically the glass which measures the error of refraction should be in contact with the eye, but for practical purposes the distance between the glass and the eye may be neglected, especially if the glasses are worn at the same distance from the eye as they occupied during the testing

The eyes of animals and of uncivilised nations are hypermetropic; children, too, are hypermetropic at birth; but, as they grow older, the refraction increases, and they become less hypermetropic, or emmetropic, or even myopic.

The evil effects of the constant and excessive demand upon the accommodation in hypermetropia are chiefly these:—

1. **Cramp of the Ciliary Muscle.**—Its persistently maintained contraction frequently gives rise to a tonic cramp of the muscle. This spasm is not, or may be only partially, relaxed when the correcting convex glass is held before the eye; and, consequently, the whole or part of the hypermetropia may be masked by the cramp. That part of the hypermetropia which is thus masked is called latent (Hl), while the part which is revealed by the convex glass is called manifest (Hm). The entire hypermetropia is made up of the latent and manifest H (H = Hm + Hl).

If the cramp be excessive, parallel rays may be kept convergent on the retina by it alone, and vision then would be made worse, rather than better, by even a weak convex glass held before the eye, a circumstance which might lead the surgeon to think he had to do with an emmetropic eye. In this case we say that the whole hypermetropia is latent.

Or, in extreme cases of accommodative spasm, parallel rays may be united in front of the retina, and the eye made apparently myopic, the vision being capable of improvement by concave glasses. Serious errors might therefore arise if this cramp were overlooked, as it is very apt to be in the examination with the trial-lenses. When it is present in a high degree, the patient cannot maintain a sustained view of an object at any distance, without suffering pain in and about the eyes. It is frequently the reason why perfect acuteness of vision is not obtained by aid of the trial-lenses, and the surgeon must be careful not to be led into an error of diagnosis by it. Examination with the ophthalmoscope, or paralysis of accommodation with atropine, will enable him to avoid mistakes.

In order to relieve this cramp, the ciliary muscle must be paralysed by a solution of atropine freely instilled; and it will often be necessary to keep the accommodation paralysed for some days, and to commence the use of the correcting spectacles before the effect of the atropine begins to wear off. In this way a recurrence of the spasm may be often prevented.

As life advances, and the power of accommodation diminishes, the manifest part of the hypermetropia increases, while the latent part decreases, until finally Hm = H.

2. **Accommodative Asthenopia.**—In looking at distant objects the accommodation of the normal eye is at perfect rest, and does not come into play until the object is approached close (within 6 m.) to the eye. But even for distant objects the hypermetropic eye must accommodate; and, having for those distances used up part of its accommodative energy, it has for near objects actually less at disposition than the normal eye. Hence we find that hypermetropic people often complain of inability to sustain accommodative efforts for near objects for any length of time. After reading, sewing, etc., for a short time, sensations of pressure in the eyes and of weight above and around them come on, and the words or stitches become indistinct, and cannot be distinguished. The work must then be interrupted, and after a few minutes' rest it can be resumed, but must soon again be given up. After a Sunday's rest the patient is often able to get on better than on the previous Saturday. These symptoms depend simply upon inability of the ciliary muscle to perform the excessive demands made upon it.

Accommodative Asthenopia (*a, priv.; σθένος, strength; ὤψ*), as this group of symptoms is called, often appears suddenly during or after illness. The explanation of this is that although hypermetropia had always existed, yet in health the ciliary muscle was equal to the great efforts required of it, but in sickness it shared the debility of the system in general. To relieve accommodative asthenopia, we have

merely to prescribe those lenses for near work which correct the hypermetropia, and by this means to place the eyes in the position of emmetropic eyes.

3. **Internal,** or **Convergent, Concomitant Strabismus.**—This condition has a certain relation to hypermetropia. It will be treated of in the chapter on the Motions of the Eyeballs and their Derangements (chap. xxi.).

The Prescribing of Spectacles in Hypermetropia.—If a person be found to be hypermetropic, but his acuteness of vision without glasses be good, or as good as he desires, and he complain of no asthenopic symptoms, glasses need not, indeed should not, be prescribed for him. No disease in his eye will result from his going without glasses. At the most he may get cramp of accommodation.

If the patient complain of imperfect distant vision due to hypermetropia, then those lenses which correct the Hm may be prescribed for distant vision, to be worn either constantly or occasionally, as he may desire. Such a patient is almost certain to complain also of accommodative asthenopia; while many patients will be met with who complain of the latter, yet express themselves as perfectly satisfied with their distant vision. For relief of the asthenopia it is usually enough to prescribe spectacles for near work which will correct the Hm, along with 1 D or 2 D of the Hl, if the latter exist.

If there be excessive cramp of accommodation, glasses to correct the whole hypermetropia should be worn while the eye is under atropine; and afterwards as much of the Hl as possible, along with the Hm, should be corrected by glasses to be worn constantly.

Myopia, or Short-sight.

This form of ametropia is due, in a vast majority of cases, to the antero-posterior axis of the eyeball being too long (Axial M.), and hence, its refracting media not being proportionately diminished in power, parallel rays of light (*a b*, Fig. 28) are

not brought to a focus on the retina, but in front of it (at *f*), and form on the retina circles of diffusion (*c d*).

Myopia may also be caused by abnormally high refracting power in the crystalline lens, as in spasm of the ciliary

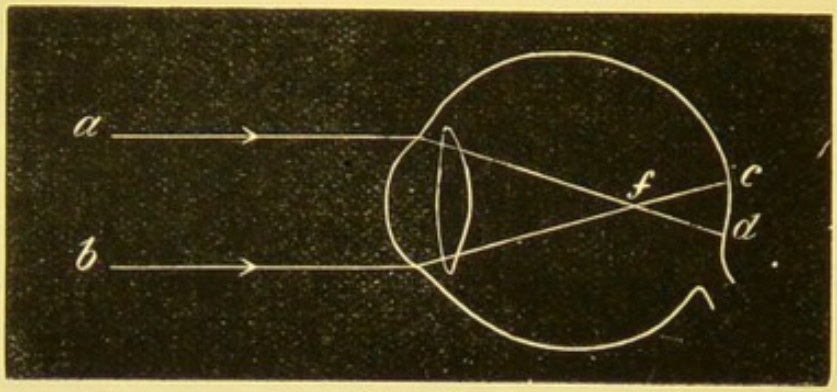


FIG. 28.

muscle, and in some cases of commencing cataract, and also by conical cornea (Curvature M.).

Since, in the myopic eye, the retina is beyond the principal focus of the dioptric system, rays emerging from any point

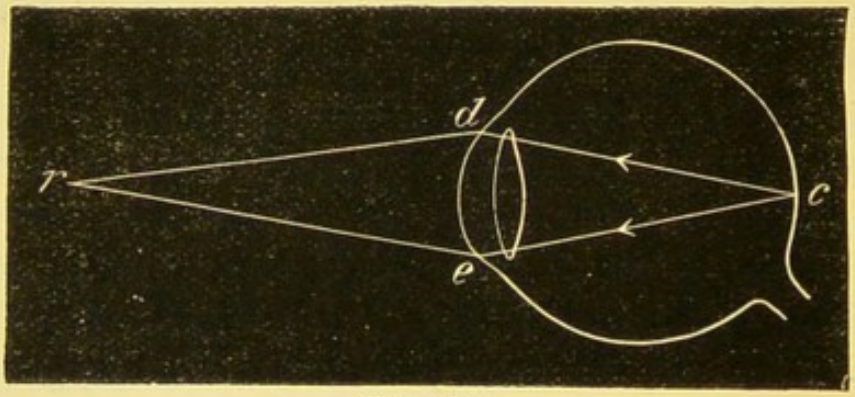


FIG. 29.

(*c*, Fig. 29) of the fundus will pass out convergently, and will unite in front of the eye at the conjugate focus of the retina (*r*). (Compare Fig. 5.)

Conversely, rays diverging from a certain point (*r*) in front of the eye will be focussed on the retina (*c*).

If an object be brought towards the eye, the divergence of those rays which pass from it into the eye increases, until, when it has reached the point *r*, their divergence is just sufficient to allow them to be united at the conjugate focus *c*, which

is on the retina. This point r is the punctum remotum [1] of the myopic eye. In order, therefore, that the short-sighted eye may be able to see distant objects, it is necessary that the parallel rays coming from those objects should be given such a degree of divergence before they pass into the eye as though they came from this punctum remotum. This can readily be effected by placing the suitable concave lens in front of the eye, and the number of this glass will indicate the degree of the myopia—*i.e.*, by how many dioptries the refracting power of the eye is in excess of that of an emmetropic eye. The focal length of the correcting glass corresponds, of course,

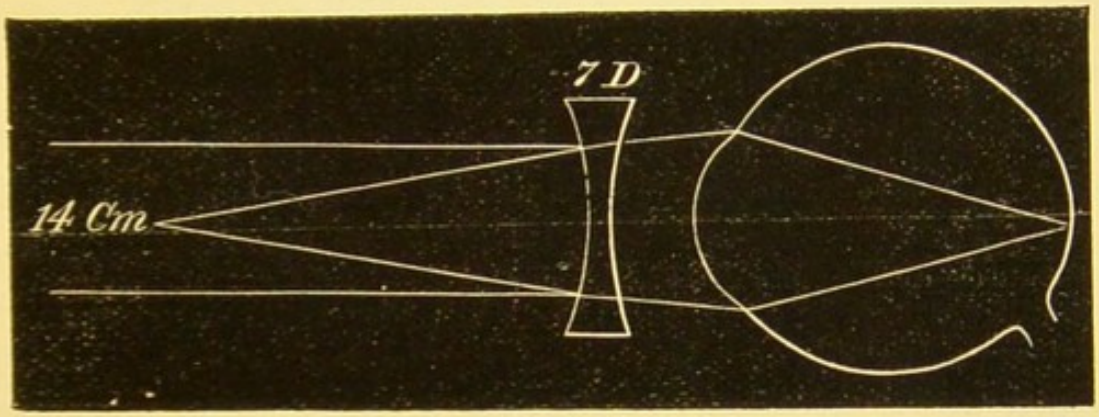


FIG. 30.

with the distance of the punctum remotum from the eye, provided the glass be held close to the cornea. The focus of the glass and the punctum remotum of the eye are then identical, and, therefore, parallel rays, after passing through the glass, will have a divergence as though they came from this point, and will form an exact image of the object from which they come on the retina.

[1] The punctum remotum is always the conjugate focus of the retina. In an emmetropic eye it is at infinity, since the retina is at the principal focus of the eye, and the rays pass out parallel. In hypermetropia it is behind the eye, and is virtual or negative, because the retina is in front of the principal focus, and the rays pass out divergently, as if coming from a point behind the retina. Lastly, in myopia it is situated at a finite distance in front of the eye, and is real and positive, because the retina is beyond the principal focus, and the rays emerge convergently.

For example: if the punctum remotum (Fig. 30) be situated at 14 cm. from the eye, then the number of the correcting lens will be 7 D, because the focal distance of this lens is 14 cm. ($\frac{100}{14} = 7$). In practice, however, we cannot hold the glass so close to the cornea, and, therefore, we must subtract the distance between it and the cornea from the focal distance of the required lens. In the above case, suppose the distance from cornea to glass be 4 cm., the required lens will be 10 D ($\frac{100}{10} = 10$).

Determination of the Degree of Myopia.—The degree, or amount, of myopia, as of hypermetropia, may be determined either by the ophthalmoscope, or experimentally, by means of the trial-lenses and test-types.

By the latter method, examining each eye separately, we find the correcting glass by placing our patient as directed in the section on Acuteness of Vision. A weak concave trial-glass is then held before the eye under examination, and higher numbers are gradually proceeded to, until the glass is reached which gives the eye the best distinguishing power for the types. We often find that there are several glasses, with each of which the patient can see equally well. *The weakest of these is the measure of his myopia.* When a higher class is used the eye may still see well, but it does so only by an effort of accommodation, and the glass employed represents not merely the myopia present, but also this accommodative effort. No more serious mistake can be made than the prescribing of too strong concave glasses for a myopic individual, as will be seen farther on.

The Amplitude of Accommodation in Myopia.—The myopic eye has an excess of refractive power as compared with the emmetropic eye; therefore, in calculating its amplitude of accommodation, this excess must be subtracted from the positive refractive power (p) which would be required to adapt the emmetropic eye to the same punctum proximum; or, in other words, the myopic eye has need of less accommodative power than the emmetropic eye, because, even at rest, it is adapted for a distance (R., its punctum remotum) for which the emmetropic eye has to

accommodate; hence, in myopia—

$$a = p - r.$$

For example, a myopic person of 10 D who can accommodate up to 8 cm. ($p = \frac{100}{8} = 12$ D) has an amplitude of accommodation of $12 - 10 = 2$ D.

The Angle γ in Myopia.—In myopia, owing to the length of the eye-ball, the cornea is cut much closer to its centre by the visual line than in emmetropia; or, these two lines may coincide; or, the cornea may

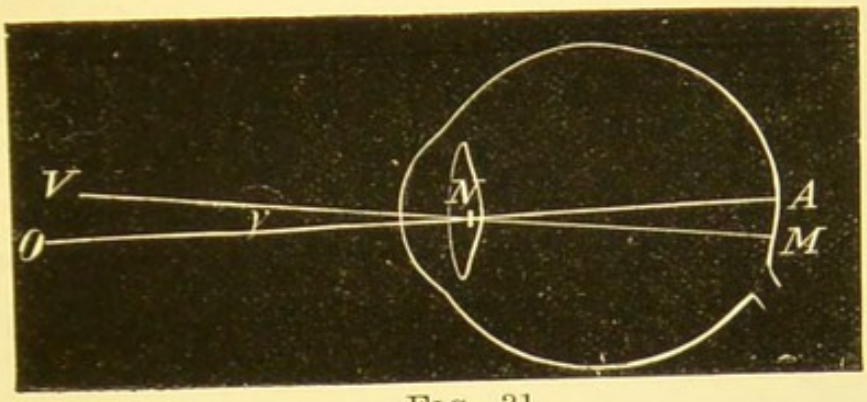


FIG. 31.

even be cut to the inside of its centre by the visual line (*vide* Fig. 31). In any of these cases, but especially in the latter, the effect will be that of an apparent convergent strabismus.

Myopia is rarely, or never, congenital, but is, for the most part, the result of the demands made upon the eyes by modern civilisation. It generally first shows itself from the eighth to the tenth year, and is apt to increase, especially during the early years of puberty. Its progressive increase is encouraged by use of the eye for near work, such as reading, sewing, drawing, etc., and is due to a further elongation of the antero-posterior optic axis. But it is certain that, in addition to this exciting cause, there must be some predisposing condition, or conditions, as only a few children become short-sighted, although they are all educated in a very similar manner, so far as the use of their eyes is concerned. Moreover, high degrees of myopia are occasionally met with in young children, before they have begun to use the eyes much for close work. Stilling[1] and Seggel[2] have found that a low orbit is usually associated with a myopic formation of

[1] *Trans. Internat. Ophth. Congress*, 1888, p. 97.

[2] *Von Græfe's Archiv.*, xxxvi., II., p. 1.

eyeball, and they are inclined to regard these largely in the light of cause and effect. For with a low orbit, and when, as often happens, the tendon of the superior oblique has an almost transverse direction, the combined pressure of the two obliques upon the plane of the equator during the period of growth would tend to cause elongation of the antero-posterior diameter of the eyeball. Opposed to this view, is the fact that in scholars of Esthic nationality, who have broad faces and low orbits, the proportion of myopes is less than in Europeans.[1] Certain it is that myopia is often hereditary, and seen in several members of a family. The whole question of the predisposing causes of myopia must still be regarded as *sub judice*.

In cases of commencing cataract a slight degree of myopia may sometimes be noticed to come on. This is due to a higher refracting power in the lens as the result of the changes beginning in it.

Hirschberg[2] states that late myopia coming on without cataract from the fortieth to the sixtieth year is a very certain sign of diabetes. He offers no explanation of its occurrence in this way. I have not myself seen such a case.

Many short-sighted people half close their eyes when endeavouring to distinguish distant objects, in order that the rays may be prevented, so far as possible, from passing through peripheral parts of the crystalline lens, which would increase the circles of diffusion. This habit it is which has given the name of myopia to the condition.

Progressive Myopia frequently becomes complicated with Organic Disease—viz., 1. *Posterior Staphyloma.*—This condition is recognised by the ophthalmoscope as a white crescent at the outer side of the optic papilla. Owing to bulging of the eyeball, the chorioid becomes atrophied at this place, and admits of the white sclerotic being seen. The

[1] Pymsza, *Inaug. Dissert.* Dorpat 1892.

[2] *Deutsche Med. Wochenschr.*, 1891, No. 13.

staphyloma sometimes extends all round the optic papilla; and, by stretching of the retina in these extreme cases, its functions may become deranged, and, in consequence, the blind spot increased in size.

2. *Chorioidal Degeneration in the Neighbourhood of the Macula Lutea.*—This should always be carefully looked for, as the region of the yellow spot is very liable to disease in bad cases of progressive myopia. The disease seems to begin in the chorioid, giving the appearance of small cracks or fissures, which later on develop into a patch of chorioidal atrophy. The retina at the spot becomes disorganised, and very serious disturbance of vision is the result, the patient being disabled from reading.

3. *Hæmorrhage in the Retina at the Yellow Spot* may occur, causing similar visual defects; and, when the hæmorrhage becomes absorbed, the macula lutea may not recover its function, owing to the delicate retinal tissue having been seriously damaged. Yet we often meet with cases of this kind, which do regain their former vision.

4. *Detachment of the Retina.*—This is a frequent and most serious complication of progressive myopia. It will be fully considered in the chapter on Diseases of the Retina (chap. xv.).

5. *Opacities in the Vitreous Humour.*—These often accompany the chorioidal alterations.

Insufficiency of the Internal Recti Muscles is another anomaly which we find very commonly associated with progressive myopia; but it can hardly be regarded as an organic disease, or as a result of progressive myopia. It may more properly be looked upon as a concomitant congenital irregularity, and, perhaps, as one of the causes of the progressive nature of myopia. It will be fully discussed in chapter xviii.

Cramp of Accommodation is often present in myopic eyes, and will cause the myopia, examined with trial-lenses and test-types, to seem higher than it is. The surgeon, being aware of this source of error, will guard against it.

In some cases of myopia a peculiar bright crescentic reflex,

first described by Weiss, can be seen close to the nasal side of the disc. Its significance has not yet been positively ascertained.

The Management of Myopia.—The great danger of myopia being its progressive increase, with consequent or attendant organic disease, its management is one of our most important and difficult tasks, especially in these days of high-pressure education. Many cases of myopia are not progressive, and cause no anxiety; others are periodically progressive; and again, others are continuously or absolutely progressive. In the periodically progressive form the age of puberty is usually the time of greatest increase and greatest danger, the myopia often becoming stationary later on. In the absolutely progressive cases the increase goes on rapidly until after puberty, and then more slowly, but it usually leads to considerable loss of vision unless the greatest care be taken.

In the progressive forms, close approximation of the eyes to the work, meaning convergence of the visual lines and accommodative effort, as, also, everything which tends to cause congestion of the eyes and head, are what we have to try to prevent. In order that these patients may not be obliged to approach close to their work, they should occupy themselves with large, and not with minute objects, and only by good light. When possible (*vide infra*) such spectacles should be prescribed for them as will enable them to read at a distance of 25 to 30 cm. In reading and writing, the books and papers should be on a slope, to facilitate an upright position of the head, and the table should not be too low. They should pause to rest for some minutes occasionally during the spell of work, while the number of working hours in the day should be restricted. The action of the bowels should be regulated, the feet kept warm, and all excessive bodily exertion avoided, so that congestion of the head and eyes may be prevented. Where posterior staphyloma, hæmorrhages at the macula lutea, or opacities in the vitreous humour are present, Heurteloup's

artificial leech applied to the temple, mild purgatives, and complete rest of the eyes, with the use of atropine for some weeks to immobilise the ciliary muscle, are to be ordered. If the chorioidal changes be very marked, small doses of the perchloride of mercury are indicated. The eyes should be protected from light by blue or smoke protection-spectacles, this latter precaution being especially necessary during the use of atropine. Insufficiency of the internal recti should be corrected by prisms, or by operation.

The correction of the myopia by suitable glasses is an important and difficult matter. In some cases of slight myopia (2·5 D and less), in young patients with good amplitude of accommodation, the correcting glasses may be prescribed to be worn constantly for near as well as for distant objects, and thus the patient is placed in the position of an emmetrope. In other cases, where the error of refraction is not excessive, and the eye is organically healthy, the whole defect may be corrected for distant vision, if the individual be warned not to use his glasses for near work, lest he should strain his accommodation. In high degrees of myopia strong glasses may be given for distant vision, but it is wise to give them 1 D or 1·5 D less than the full correction, so that all danger of accommodative effort may be avoided. In these same cases, provided there be no ophthalmoscopic changes, or only some of minor significance, and if the vision be good, such a glass may be given as will enable the patient to read at 25 to 30 cm. This glass may be found by subtracting from the number of the glass representing the degree of the myopia (say 7 D) the lens whose focal length corresponds to the distance (say 30 cm.) required (this here would be 3·25 D, because $\frac{100}{30} = 3{\cdot}25$, and then $7{\cdot}0 - 3{\cdot}25 = 3{\cdot}75$ D the glass required). By aid of such glasses this myope can read at a distance much more favourable for the convergence of his optic axes, and for the erect position of his head; but there is a danger associated with their use—namely, that if the patient approach his book

closer than the prescribed distance, he does away with the advantage he should gain from them, and, by necessitating an effort of accommodation, turns them to a serious source of danger for the eye. Patients in whom the acuteness of vision is much lowered are liable to approach their work in this way, in order to obtain larger retinal images, the more so as the concave glasses diminish the size of the images, and in such cases it is better not to give glasses for near work. It is often necessary to provide patients with spectacles which will enable them to use their eyes for some special purpose at a given distance—*e.g.*, the pianoforte, painting, etc., and these can be found as above explained.

In high degrees of myopia of 15 D and more removal of the lens has been adopted with a certain amount of success, the acuteness of vision in some cases having been improved after the operation. The method consists in repeated discissions with subsequent evacuation of the softened lens substance. Removal of the lens in myopic eyes seems to diminish the refraction by about 15 D.[1] I have employed this treatment with success.

Astigmatism.

This is a compound form of ametropia, due to the cornea being more curved in one meridian than in another, similarly as the back of the bowl of a spoon is more convex from side to side than from heel to point.

In Regular Astigmatism the directions of the greatest and least curvations of the cornea are always at right angles to each other, and usually fall precisely in the vertical and horizontal meridians, the meridian of greatest curvature being most frequently the vertical. Consequently, we say the astigmatism is "with the rule" in those cases in which the meridian of greatest curvature is the vertical; and, where that

[1] *Bericht. d. Ophth. Gesellschaft zu Heidelberg*, 1893.

meridian is the one of least curvature, we say the astigmatism is "against the rule." The result of this is, that a pencil of rays passing into the eye, instead of meeting at a common focus, is irregularly refracted, those rays passing through the vertical meridian of the cornea being brought to a focus much earlier than those which fall through its horizontal meridian; and, therefore, at the focus of the former the latter rays form a horizontal streak of light. The intermediate, or oblique, meridians will probably be of regularly intermediate refracting power.

The interval between the foci of the two principal meridians is called the Focal Interval, and is a measure of the astigmatism.

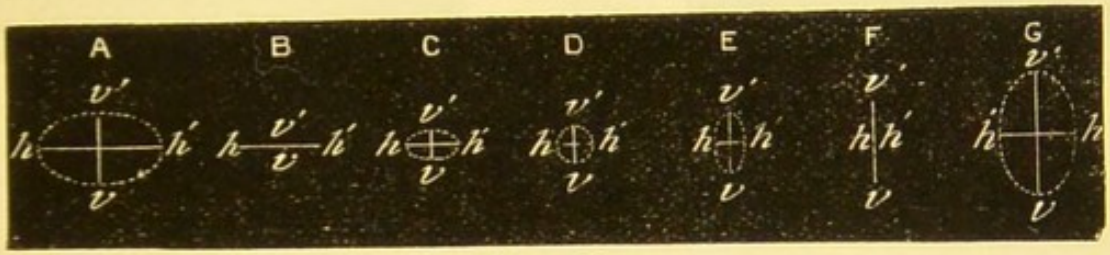


FIG. 32.

The accompanying diagram (Fig. 32), after Donders, will assist in the understanding of the course of a pencil of rays after they have passed through an astigmatic cornea, those rays belonging to the horizontal and vertical meridians being chiefly considered.

At A neither vertical (v, v') nor horizontal (h, h') rays have yet been united at their foci, but the vertical rays are the nearest to their focus; and, therefore, the appearance which the pencil of rays would give, if caught here on an intercepting screen, is an oval with its long axis horizontal. At B the vertical rays have met at their focus, but the horizontal rays not as yet at theirs, and the result is therefore a horizontal straight line. At C the vertical rays are diverging again from their focus, and the horizontal rays have still not come to theirs. At D the same conditions exist, only a little farther on, where the one set of rays is diverging, the other still

converging, but each at the same angle ; hence the shape of the figure is round. At F the horizontal rays have met, and the result is a vertical straight line. At G both sets of rays are divergent, and the figure is an oval with the long axis perpendicular.

There are various kinds of regular astigmatism, according to the position of the two principal foci with reference to the retina, as follows : —

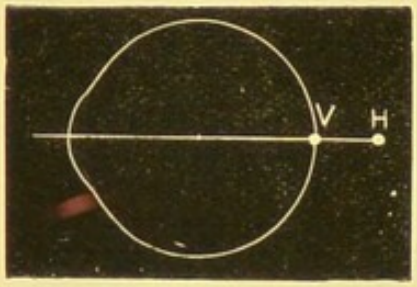


FIG. 33.

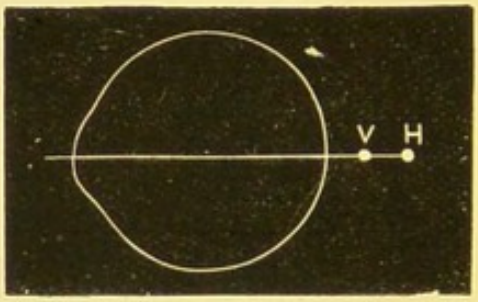


FIG. 34.

1. *Simple Hypermetropic Astigmatism.*—When the focus (V, Fig. 33) of the vertical rays is situated on the retina (emmetropia in that meridian), while that (H) of the horizontal rays lies behind the retina (hypermetropia in that meridian).

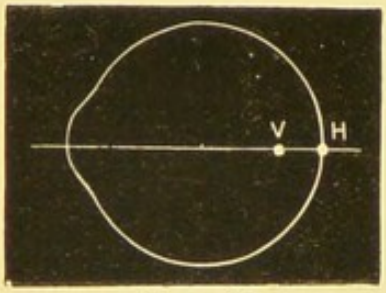


FIG. 35.

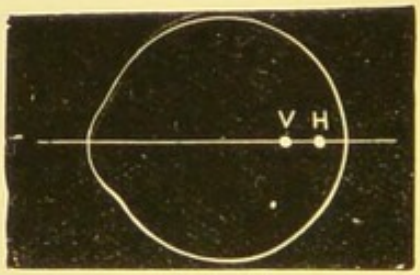


FIG. 36.

2. *Compound Hypermetropic Astigmatism.*—When the foci of both sets of rays is behind the retina, that (H, Fig. 34) of the horizontal rays farther back than that (V) of the vertical rays.

3. *Simple Myopic Astigmatism.*—When the focus (H, Fig. 35) of the horizontal rays is situated on the retina (emmetropia in that meridian), while the focus (V) of the vertical rays is situated in front of the retina.

4. *Compound Myopic Astigmatism.*—When the foci of both

sets of rays are situated in front of the retina, but farther forward in the case (V, Fig. 36) of the vertical rays.

5. *Mixed Astigmatism.*—When the focus (H, Fig. 37) of the horizontal rays falls behind the retina (hypermetropia in that meridian), and the focus (V) of the vertical rays in front of the retina (myopia in that meridian).

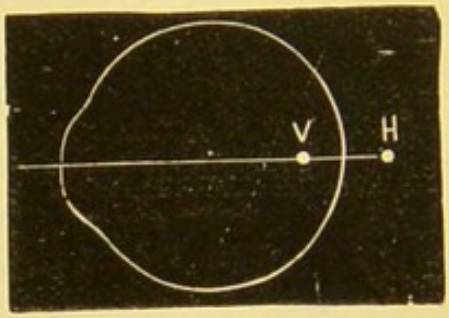


FIG. 37.

Symptoms of Astigmatism.—We may conclude that an individual is astigmatic if he sees horizontal (or vertical) lines, such as the horizontal portions of Roman capital letters, or the horizontal lines in music, distinctly, while the vertical (or horizontal) lines seem indistinct. We have such a complaint, for example, when the retina is situated at the focus of the parallel rays passing through the vertical meridian of the cornea.

Suppose an eye to be emmetropic in the vertical meridian, and ametropic in the horizontal meridian. We must first consider how a point will be seen by such an eye. The rays of light emitted from the point and passing through the horizontal meridian will not be brought to a focus on the retina, but will produce a blurring of the retinal image of the point at each side; while the vertical rays will unite on the retina, and, consequently, the point will appear distinctly defined above and below.

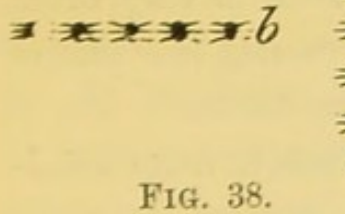


FIG. 38.

A line may be regarded as a number of points, and, in order to understand how lines will be seen by an astigmatic eye such as the above, it is only necessary to arrange a number of points in vertical and horizontal lines—as at *a* and *b* in Fig. 38. It is evident at once from mere inspection that the horizontal line will appear distinct, because the rays which diverge from each point of the latter in a vertical plane—*i.e.*, at right angles to the direction of the line, are brought to a

focus on the retina; while those rays diverging in a horizontal plane, although not meeting on the retina, do not render the picture of the line indistinct, because the diffusion images resulting from them exist in the horizontal direction, and, consequently, cover or overlap each other on the horizontal line, and therefore are not seen. At the ends of the line only

FIG. 39.

(*b*, Fig. 39) do the diffusion images cause a "fuzziness," or make the line seem longer than it is. In this case a vertical line (*a*, Figs. 38 and 39) seems indistinct; because, the horizontal meridian being out of focus, the diffusion images existing in that direction are very apparent, as they do not overlap. On the other hand, in order to see a vertical stripe accurately, it is necessary only that the rays diverging in a horizontal plane should have their focus on the retina; and, therefore, if an individual can only see vertical lines distinctly at 6 metres, we know that his eye is emmetropic in the horizontal meridian (and probably myopic in the vertical meridian). We do not, however, hear this complaint as often as might be expected, because simple astigmatism is not so common as one or other of the compound forms.

Astigmatic people do not generally see very distinctly, either at long or at short distances.

Even in hypermetropic astigmatism the book is very often brought close to the eyes, in order, by increasing the size of the retinal image, to make up for its indistinctness.

Astigmatic individuals frequently suffer much from headache, due to constant effort to see distinctly, and we cure the headache when we correct the astigmatism.

It has been stated that epilepsy, if not capable of being produced by refractive errors, especially astigmatism, in persons with stable brains, may sometimes have such errors as its exciting cause where there is already a predisposition to the disease.

All these signs and symptoms appertain more to the rather high degrees of astigmatism. Slighter degrees may cause no annoyance beyond some indistinctness of vision ; and, indeed, slight degrees of hypermetropic astigmatism often pass unnoticed until late in life, when the accommodation begins to fail.

We are often led to suspect and to seek for astigmatism when, in examining the refraction with spherical glasses, we are able to bring about some improvement of vision, but cannot obtain normal V. with any glass, while there is no organic disease to account for the defect. Also if, in examining with spherical glasses, we find V. benefited equally by several glasses of considerable difference in power, even perhaps by convex as well as by concave glasses.

The ophthalmoscope affords us an admirable means of diagnosing astigmatism, and of determining its amount. Just as the astigmatic eye cannot see horizontal and vertical lines equally well at the same moment, so is an observer unable to see both the vertical and horizontal vessels in the retina of the eye simultaneously, but must alter his accommodation to be able to see first the one set, and then the other.

A comparison of the shape of the optic papilla, as seen in the upright and in the inverted images, also gives a clue to the presence of astigmatism. Inasmuch as the fundus oculi is very much magnified in the upright image by the dioptric media through which it is seen, and as this enlargement is greater in the direction of the meridian of shortest focus (meridian of highest refraction), which is most commonly the vertical meridian, a circular object such as the papilla will seem to be of an oval shape with its long axis vertical. But in the inverted image, in the meridian of highest refraction, the image lies nearer the convex lens than in the meridian of lowest refraction, and, hence, is much less magnified in the former than in the latter meridian ; and here, consequently, the round optic papilla is seen as an oval with its long axis horizontal.

Sometimes the papilla is really of an oval shape, and not round, and then the diagnosis is readily made by observing that in one image it is seen as an oval, while in the other image it is circular. Care must be taken in the indirect method not to hold the lens obliquely, as this would be sufficient to make a circular disc appear oval, the long axis of the oval being in the direction of the axis round which the lens is rotated. The determination of the degree of astigmatism can also be accomplished with the ophthalmoscope, and will be treated of in the next chapter.

The Estimation of the Degree of Astigmatism, and its Correction.—It is evident that, to correct astigmatism, the ordinary spherical lenses would be of little use, for they affect the refraction of the light passing through them equally in every direction. We employ, therefore, what are termed cylindrical lenses, being sections of cylinders parallel to their axes, which refract light in one direction only—viz., that corresponding to their curvatures and at right angles to their axes. The rays which pass through these lenses in a direction corresponding to their axes are not refracted, but pass on without deviation as they would do through a piece of plane glass.

Although astigmatism is nowadays almost universally estimated by means of the ophthalmoscope or by the astigmometer (see p. 52), yet, in order to give the student a clear idea of the matter in the simplest way, I shall here describe a subjective method for its estimation, while its objective estimation by aid of the ophthalmoscope will be treated of in the next chapter.

Simple Astigmatism.—If, now, a case come before us in which we suspect astigmatism, we place Snellen's Sunrise (*vide* diagram at end of book), or some such diagram, at 6 metres from the eye—the other eye being excluded—and inquire of the patient whether there be any line which he sees much more distinctly than the others, and can trace farther towards the

central point. If that be so, we know that he is emmetropic in the meridian at right angles to that line, provided his accommodation be at rest, and ametropic in the meridian corresponding to that line.

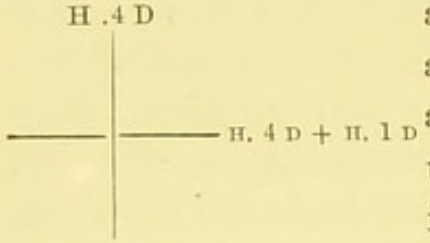
In case the horizontal line below at each side be the distinct one, the eye is emmetropic in the vertical meridian; and probably hypermetropic in the horizontal meridian, because the latter is generally that of least curvature. Consequently, a convex cylindrical lens, held with its curvature horizontally (axis vertical) before the eye, will correct the defect. The highest convex cylindrical glass which gives the patient the best possible distant vision will be the correcting glass. This is a case of Simple Hypermetropic Astigmatism (As. H.). If the lens required be + 2 D Cyl. it would be As. H. 2 D; and in prescribing for the optician we should write "+ 2 D Cyl. Ax. Vert."

If the central vertical line be the distinct one, then emmetropia exists in the horizontal meridian, and probably, therefore, myopia in the vertical meridian; and a concave cylindrical lens held before the eye with its curvature vertical (axis horizontal) will correct the defect. The lowest concave cylindrical lens which gives the patient the best possible distant vision, will be the correcting lens. This is a case of Simple Myopic Astigmatism (As. M.). If the lens be — 2·5 Cyl. it would be As. M, 2·5 D; and for the optician we should write, "— 2·5 D Cyl. Ax. Horiz."

I advise the reader to make now a few experiments for himself with cylindrical lenses, by means of which he can produce artificial astigmatism in his own eye. If he hold a + 1·0 Cyl. before his eye, with its axis horizontal, it gives a myopia of 1·0 D to the vertical meridian of the eye, while the horizontal meridian remains emmetropic; and, consequently, he will see the central vertical line of the diagram distinctly, while the horizontal lines will be indistinct. By placing a — 1·0 Cyl. with its axis vertical before the eye, in

addition to the + 1·0 Cyl., the artificial astigmatism produced by the latter is corrected, and the whole diagram becomes distinct. Every other kind and degree of astigmatism can be similarly represented by lenses, and similarly corrected.

Compound Astigmatism.—If no line be very distinctly seen, then we may commence our examination with Snellen's Distance Test-Types, and test in the ordinary way with spherical lenses, until we find that one which gives the best distant vision. This we place in a spectacle frame before the eye, and proceed, as already explained, to ascertain the meridians of greatest and least curvature of the cornea. If the spherical lens be + 4 D, and with it the horizontal lines in the sunrise diagram be the most distinct, then the vertical meridian is shown to be corrected, and the eye is probably still hypermetropic in the horizontal meridian, and requires a + cylindrical lens with its axis vertical, in addition to the spherical lens, to correct the entire defect. Suppose this cylindrical lens be found to be + 1 D. Cyl., then the H. in the horizontal meridian will be shown to be 5 D, and the astigmatism to be 1 D.



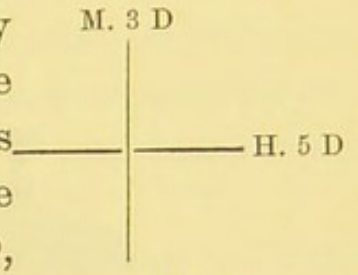
The latter noted down would be of little practical value, and therefore we prefer to write in our note-books the factors of the Astigmatism, thus:—"H. 4 D + As. H. 1 D Horiz."; or, as for the optician, "+ 4 D Sph.⌒ + 1 D Cyl. Ax. Vert."[1] This is Compound Hypermetropic Astigmatism.

In an analogous way we examine for Compound Myopic Astigmatism, in which every meridian is myopic, but the vertical more so than the others.

Mixed Astigmatism.—Lastly, we come across cases in which both concave and convex spherical lenses produce a certain amount of improvement, but neither give full vision. Placing then one or other before the eye in the spectacle frame, the

[1] The sign ⌒ indicates "combined with."

examination is proceeded with by aid of Snellen's Sunrise. We ascertain, for example, what is the lowest concave spherical lens which will bring out one horizontal ray distinctly; let this be — 3 D; we have then myopia of 3 D in the vertical meridian. Now, having removed the — lens, we find what is the highest convex lens which will bring out one vertical line distinctly; let it be + 5 D; this indicates hypermetropia of that amount in the horizontal meridian. We may correct such a case in either of two ways—(*a*) By a Sph. — 3 D, which will correct the vertical meridian, but will increase the hypermetropia in the horizontal meridian by 3 D, making it 8 D, which can then be corrected by combining a cylindrical lens of + 8 D, axis vertical, with the above spherical lens. (*b*) By a spherical + 5 D, which will correct the horizontal meridian, but will increase the myopia in the vertical meridian to 8 D, necessitating the combination of a — Cyl. lens of that number, with the + 5 D Sph. For reading, writing, etc., an over-correction of the horizontal meridian with + 8 D Cyl., thus rendering the eye myopic 3 D in every meridian, and enabling the patient to read at, or near, his far point, might be the most suitable arrangement.



As it is necessary, in order to test the degree, etc., of astigmatism accurately, that the accommodation be at rest, it is desirable, before the examination for any of the hypermetropic forms, to instil atropine into the eye.

Measurement of the Degree of Astigmatism by the Astigmometer.—This is one of the most rapid and satisfactory methods of determining both the degree of astigmatism and the position of the meridians of greatest and least refraction. It is based on the principle of the ophthalmometer, an instrument by which Helmholtz demonstrated the changes in the curvature of the lens during accommodation.

The cornea reflects images of objects in the same manner

as a convex mirror, and the smaller the radius of curvature the smaller will the image of any given object be. It is easy to calculate the radius of curvature of the cornea knowing the size of the object, the distance of the object from the

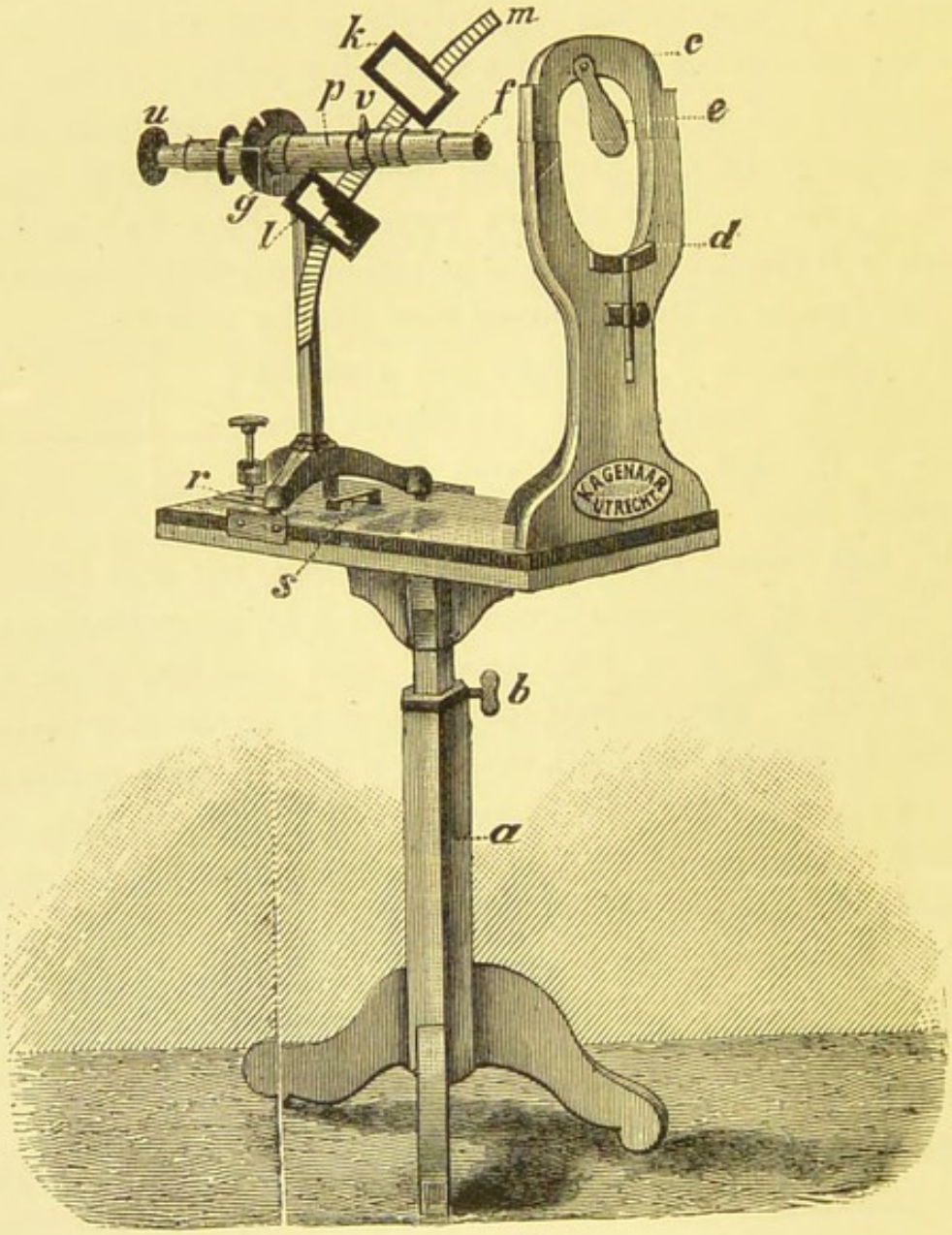


FIG. 40.

cornea, and the size of the corneal image. The only difficulty lies in the measurement of this image; and it has been found that the best method of effecting this is to double the image, by looking at it through a double refracting prism, and then

to alter the strength of the prism until the two images just come into contact. When this has taken place, a displacement equal to the size of the image has been produced. The amount of displacement, and hence the size of the image, can easily be calculated.

The astigmometer was first brought into practical use by Javal and Schiotz. The instrument which is in use at the National Eye and Ear Infirmary, and which has proved of great service, is a modification of Javal's, made by Kagenaar of Utrecht. In order to measure the degree of astigmatism by it we do not require to know the radius of curvature of the cornea, but merely to find out the difference in refractive power between the meridians of greatest and least curvature, and this the astigmometer enables us to do in a few seconds without any calculation.

It consists (Fig. 40) of a telescope *p* containing a double refracting prism between the object glasses, and two reflectors *k* and *l*, which are movable on an arc *m*, which is fixed to the telescope tube. The latter turns on its own axis, and enables the arc to be placed in any meridian, its position being indicated on a graduated circle *g*. The patient places

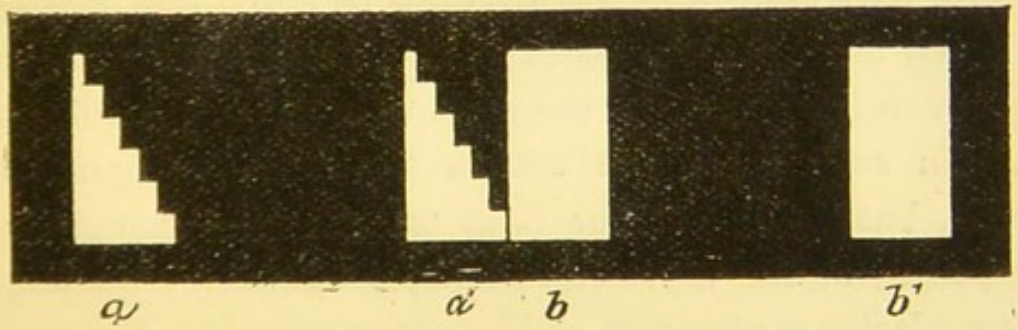


FIG. 41.

his chin on the rest *d* and looks into the tube at *f*, the eye which is not under observation being covered by the disc *e*. The surgeon then looks through the telescope at *u*, turns the arc *m* into a horizontal position, and observes the corneal images of the reflectors, which he gets into focus. He then moves the reflectors until the central images just come into contact; the four images will then occupy the relative positions

shown in Fig. 41. The arc is then rotated into the vertical meridian, and if the curvature of the cornea be the same as before, the central images will still appear to be in contact. But, if the radius of curvature be smaller, the intervals a to b and a' to b' will diminish, and consequently the central images will overlap as in Fig. 42, each step of a' representing a difference of 1 Dioptry. So that in this case there would be an astigmatism of 2 D. And the greatest refraction would be in the vertical meridian.

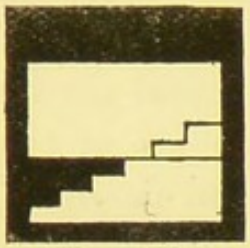


FIG. 42.

It is generally best to begin with the arc in the horizontal meridian. If the axes of the meridians of greatest and least curvature are oblique, then the images will not lie in one line, and the arc must be turned until they do so. An index which moves on the circle g gives the position of the axes. It will be seen from the above description that the astigmometer merely registers the amount of astigmatism, but does not enable us to estimate the refraction of the eye. Moreover, it is the corneal astigmatism alone which is determined, but it will be found that in the vast majority of cases this is the only astigmatism present.

Lental Astigmatism.—Disturbances of vision due to astigmatism often make their appearance for the first time at middle age or even later, and are then apt to be mistaken for amblyopia. In such cases the cornea has been astigmatic all through, but the defect has been masked by a compensating astigmatism of the crystalline lens, produced by an unequal accommodative contraction of the ciliary muscle. When, now, as life advances, the amplitude of accommodation diminishes, the power of the ciliary muscle to produce this active compensatory lental astigmatism also diminishes, and finally disappears; and, consequently, the corneal astigmatism comes to the front. Or, in astigmatic individuals the astigmatism may alter in degree at this time of life. Under atropine, too, astigmatism may appear, the existence of which was not

previously known. This is termed active, or dynamic, lental astigmatism.

Passive, or static, lental astigmatism is due to irregularity in the shape of the unaccommodated lens, and, as the case may be, gives rise to disturbances of vision similar to those caused by corneal astigmatism; or, it increases existing corneal astigmatism; or, it more or less completely compensates the corneal astigmatism. It has no clinical importance which does not attach to corneal astigmatism.

Irregular Astigmatism.

In irregular astigmatism the refraction of the eye differs, not only in different meridians of the eye, but even in different parts of one and the same meridian. It is frequently due to irregularities on the surface of the cornea, the result of former ulcers, and also sometimes to irregular refracting power in different parts of the crystalline lens. It cannot be corrected. Its presence can be detected by the distortion and irregular movement of the disc, when the lens is moved during the indirect method of examining with the ophthalmoscope, and also by the irregular shadow in retinoscopy. In some cases there is a certain amount of regular astigmatism combined with it, correction of which may improve the vision.

Anisometropia [1]

means a difference in the refraction of the two eyes, one being myopic, hypermetropic, or astigmatic, while the other is emmetropic, or ametropic in a way different from its fellow. So long as the difference in refraction is but slight, say 1 D or 1·5 D, it is generally possible to give the correcting glass to each eye. When the difference is considerable, it is often impossible to fully correct each eye, because binocular vision having never really existed, the patients are unable to tolerate the presence of a clear image on each retina. We must then

[1] *a, priv.; ισος, like; μέτρον, a measure.*

be content with correction of the least ametropic eye, or of that one which has the best vision; or, we may partially correct the most ametropic, and fully correct the least ametropic eye. Each such case must be dealt with as it permits.

ANOMALIES OF ACCOMMODATION.

Presbyopia.

This is a diminution in the amplitude of accommodation, which commences at an early age, and is due solely to natural changes taking place slowly in the crystalline lens. It might not, therefore, strictly speaking, be considered as an anomaly. The power of accommodation commences to diminish in early childhood, the near point beginning then to recede from the eye. Donders it was who first discovered this fact, and ascertained the laws which govern the progressive decrease of accommodative power. He designed the accompanying diagram (Fig. 43), which illustrates the decrease from the tenth year of age, and indicates the amplitude of accommodation at different ages.

The numbers along the upper horizontal line refer to the ages, those along the left-hand perpendicular line to the dioptries. The curve *r r* shows the refraction of the eye when in a state of rest. This is unchanged until the 55th year, when it begins to diminish; the emmetropic eye then becoming hypermetropic, the hypermetropic eye more hypermetropic, and the myopic eye less myopic. The curve *p p* shows the positive refracting power of the eye, corresponding to the punctum proximum, and its gradual diminution as life advances; and how, at the age of 65, it becomes even less than the minimum refraction in former years. The two curves meet at the age of 73, and then all power of accommodation ceases. The number of dioptries included between the two curves on the vertical line corresponding to any given age represent the amplitude of accommodation at that age—*e.g.*,

at 30 years of age the amplitude is 7 D ; at 50 years it is only 2·5 D. The amplitude of accommodation is the same at the same age in all forms of ametropia, as well as in emmetropia.

The cause of presbyopia lies chiefly in a progressive change in the crystalline lens, which becomes less elastic, and more homogeneous in its different layers, and refracts light less strongly than before. In more advanced life diminished

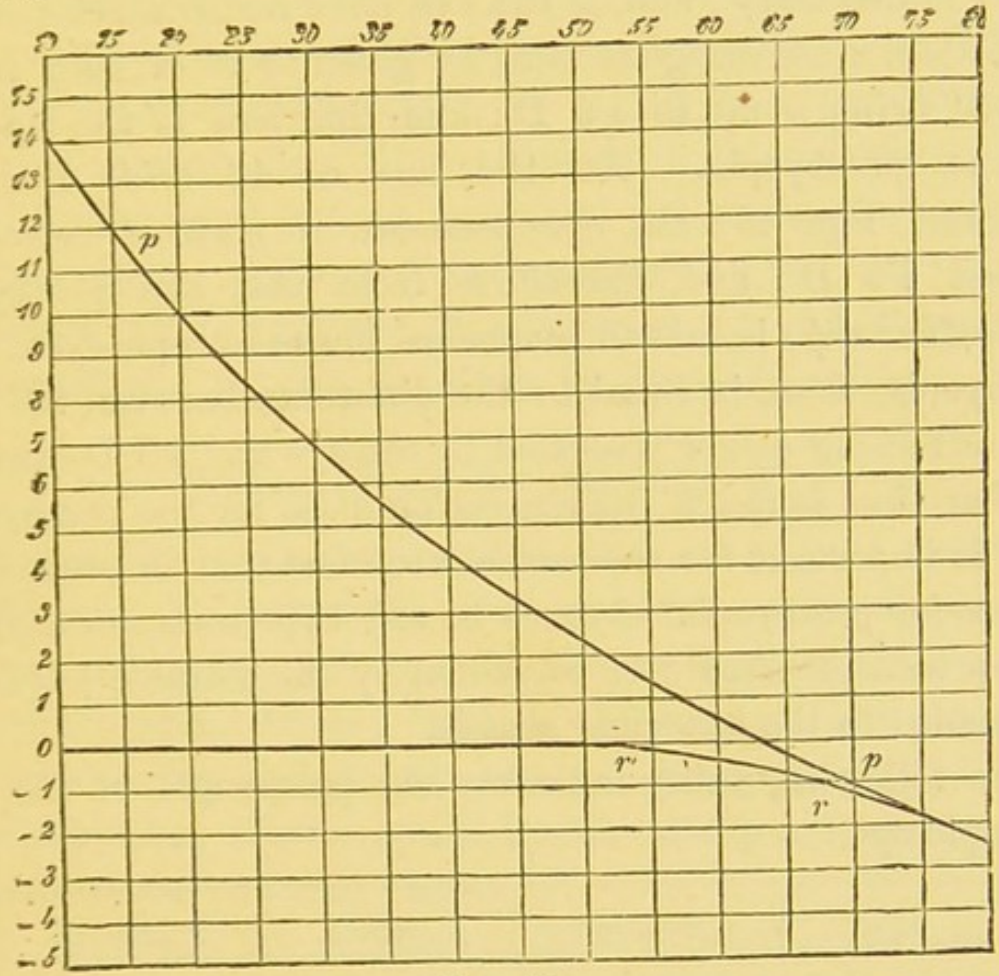


FIG. 43.

energy of the ciliary muscle probably becomes a second factor in the production of presbyopia.

The near point gradually recedes from the eye, until it reaches a distance beyond that at which the person usually reads, writes, sews, etc. Employments of this kind then become difficult, because the retinal images are too small to be clearly discerned, owing to the increased distance at which the work must be held from the eye ; and, in order to make up for this smallness of the images, the individual

is often seen to improve their brilliancy by procuring stronger light.

Presbyopia is usually said to be present when the near point lies at more than 22 cm. from the eye, and we correct it by giving such a convex glass for reading, etc., as will bring the near point back to 22 cm. Now, in order to see at that distance, a positive refracting power (p) of ($\frac{100}{22} =$) 4·5 D is necessary; and, if the eye have not so much positive refraction, a convex glass must be given to it of such power as will bring p up to 4·5 D; and this lens is the measure of the presbyopia. At the age of 40 (*vide* Donder's diagram, Fig. 43) the eye possesses a positive refraction of just 4·5 D; and, therefore, from this age presbyopia (πρέσβυς[1] ὤψ) is said to commence in emmetropic eyes. The presbyopia, then, is equal to the difference between the positive refracting power possessed by the eye and 4·5 D, and the number thus found is the correcting glass for the presbyopia.

It is important for the patient's comfort that in prescribing glasses for presbyopia, if there be any hypermetropic astigmatism present, it should be corrected by the suitable + cylinder lens added to the spherical glasses.

The following table indicates the presbyopia of the emmetropic eye:—

Age.	p. required.	p. existing.	Presbyopia.
40	4·5	4·5	0
45	4·5	3·5	1·0
50	4·5	2·5	2·0
55	4·5	1·5	3·0
60	4·5	0·5	4·0
65	4·5	0·25	4·25
70	4·5	−1·0	5·5
75	4·5	−1·75	6·25
80	4·5	−2·5	7·0

It is hardly necessary to point out that presbyopia comes

[1] An old man.

on at a much earlier age in hypermetropes than in emmetropes; while in myopes its advent is postponed; or, in the higher degrees of myopia, it may not come on at all. The hypermetrope of 3 D would be presbyopic at the age of 27; because, in order to arrive at the 4·5 D of positive refraction required, he must have an amplitude of accommodation of (3 D + 4·5 D) 7·5 D, and this he has only up to that age (*vide* Fig. 43).

The myope of 4·5 D can get along until something over 60 years of age without any glass for reading (*vide* above Table). At 65, if he were emmetropic, he would have presbyopia of 4·25; consequently, he will now require a + glass of only 0.25 D.

Presbyopia must not be mistaken for slight paralysis of accommodation. They are distinguished by the fact that, in the former, the amplitude of accommodation corresponds to the age of the patient as given in Donder's table.

Paralysis of Accommodation.

This may be partial or complete, and one or both eyes may be affected. It is usually combined with paralysis of the sphincter iridis (mydriasis), and the condition is then called ophthalmoplegia interna; but it is also seen without paralysis of the sphincter, and either alone or with paralysis of some of the orbital muscles supplied by the third pair—rarely with paralysis of the external rectus.

The Symptoms are similar to those of presbyopia, and give inconvenience to the patient according to the state of his refraction. If he be emmetropic, his distant vision continues good, while his vision for near work is much impeded. If he be hypermetropic, as he requires his accommodation for distant objects, vision for distance is interfered with, and, still more so, vision for near objects. If he be myopic, vision is less affected than in either of the other forms of refraction; indeed, if he be very near-sighted, being able to see near objects at his far point, he may suffer little or no inconvenience.

Micropsia is a common symptom in cases of partial paralysis

of accommodation, and is due to the fact that, while the retinal image is unaltered in size, the great effort of the defective accommodation gives the sensation of the object being much nearer to the eye than it really is.

Causes.—The most common cause of paralysis of accommodation is the action of atropine; but it is also the result of, or is attendant upon, various diseases. It is one of the symptoms of paralysis of the third nerve; it may be due to exposure to cold; or it may depend upon syphilis, syphilitic periostitis at the sphenoidal fissure, syphilitic gumma, or syphilitic inflammation of the nerve itself.

In cases of double paralysis of accommodation a central cause must often be looked for. Paralysis of accommodation and mydriasis are sometimes forerunners, by many years, of serious mental derangement.

Diphtheria is a frequent cause of paralysis of accommodation, usually without, but sometimes with, mydriasis. The onset occurs most commonly some weeks after the throat affection, which need not have been of a severe character. Indeed, the faucial attack may have had no apparent diphtheritic character, and may have been so slight as almost to have escaped the notice of the patient. The lesion in these cases is probably a nuclear one, and the evidence points to miliary extravasations of blood in the floor of the fourth ventricle; but there are those who hold that the paralysis is due to a poison, that it is a toxic paralysis.

During the recent epidemics of influenza (*la grippe*) cases of paralysis of accommodation were recorded, occurring some of them during the acute stage and others during convalescence. They all recovered except one, which seems to have gone on to bulbar paralysis.[1]

Paralysis of accommodation in middle life may be due to diabetes, and should make us suspicious of the presence of this disease.

[1] Uhthoff in *Deutsche Med. Wochenschr.*, No. 10, 1890.

Blows on the eye are apt to cause paralysis of accommodation, usually with mydriasis.

The Treatment depends, of course, upon the cause of the paralysis. The instillation of a 1% solution of sulphate of eserine, or of muriate of pilocarpine, may be employed in all cases, and will at least produce temporary improvement of sight; but it can hardly be said to assist in the cure, except perhaps in slight diphtherial cases. Iodide of potassium, and mercury, are indicated in syphilitic cases; and iodide of potassium, and salicylate of sodium, in rheumatic cases. The prognosis in these cases must be very guarded, as it often happens that recovery does not take place. Where cure does not result, the patient may be enabled to make better use of his eye, or eyes, by means of a convex glass, or spectacles; but, in this matter, each case must be dealt with for itself—no general rule can be laid down.

In diphtheritic cases a general tonic treatment, especially iron, is indicated; and here the prognosis is invariably favourable.

Accommodative Asthenopia

has been already treated of under the head of Hypermetropia (p. 32).

Spasm of Accommodation.

Spasm, or cramp, of accommodation in connection with hypermetropia and myopia has already been referred to. A few cases of acute spasm of accommodation have been reported.[1] Occurring in an emmetropic or slightly hypermetropic eye, such a spasm produces apparent myopia. In some of the cases there was no assignable cause for the spasm, in some it was due to overwork, and in one to trauma of the cornea. The treatment is a lengthened course of atropine locally.

[1] A. v. Graefe, *Archiv f. Ophthal.*, vol. ii., pt. 2, p. 308; Liebreich, *Archiv f. Ophthal.*, vol. iii., pt. 1, p. 259; C. E. Fitzgerard, *Trans. Ophthal. Soc.*, vol. v., p. 311.

CHAPTER III.

THE OPHTHALMOSCOPE.

Although the dioptric media of an eye be perfectly clear and normal, yet no detail of its fundus can be discerned by the unaided eye of an observer who looks through the pupil, the latter being for him merely a dark opening. The reason of this is, that these dioptric media are composed of a system of convex lenses. To explain :—Suppose the inside of a small

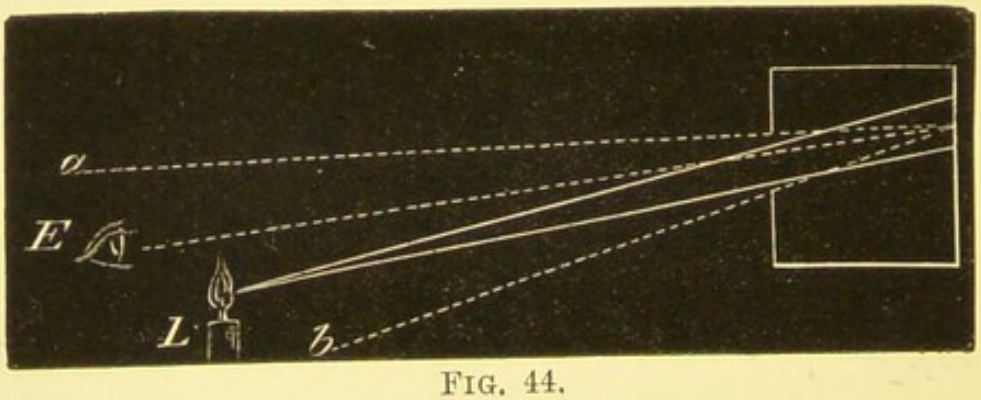


Fig. 44.

box (*vide* Fig. 44) to be blackened, and on its floor some printed letters fastened, and a hole cut in the lid, which is then replaced : it will be found that, by aid of a lighted candle and with a little experimentation, the letters may be read through the aperture. The rays passing from the light *L* into the box through the aperture illuminate the opposite surface, and from this surface the rays *a*, *b*, and others, pass out again through the opening, and some of them fall into the observer's eye at *E*.

But if, in order to make this box represent an eye more accurately, we place a convex lens immediately within the aperture, the course of the rays is altered. All the rays

passing into the box (Fig. 45) from L are brought to a focus on its opposite side at m by the convex lens n; and, according to the optical law of conjugate foci, all the rays passing out from the box meet again at the source of light L, and hence none of them can be received by the eye (a) of the observer, nor can this eye be placed in any position where it could

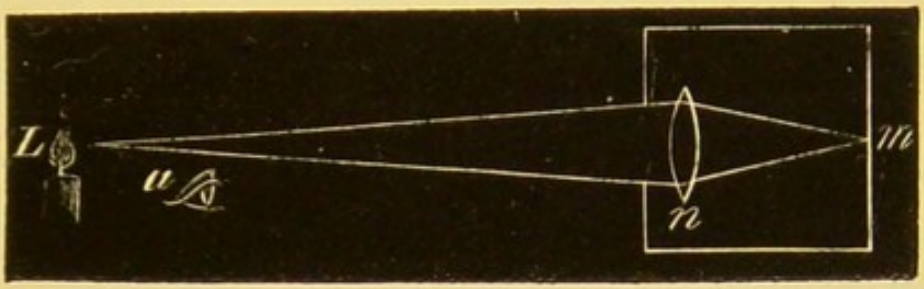


FIG. 45.

catch any of these rays; for, if it be placed anywhere between the aperture and L, it would cut off the light passing from L into the box.

Helmholtz's Ophthalmoscope.—If the eye of the observer could itself be made the source of light, the difficulty would be solved; and, practically, this is what Helmholtz accomplished

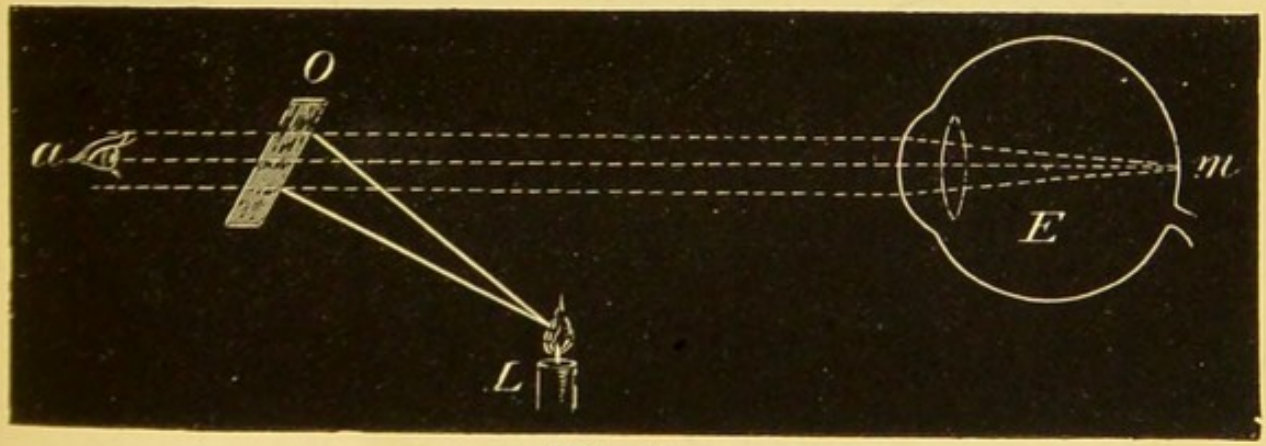


FIG. 46.

with his ophthalmoscope in the year 1851. The instrument he invented was composed of a number of small plates of glass, O (Fig. 46), from which light from L was reflected into the eye E, and thus the fundus of the latter illuminated. From m rays pass back again by the same course to the ophthalmoscope, some being reflected back to L; but some, passing through the ophthalmoscope, and falling into the observer's

eye placed close behind the instrument at *a*, form in it an image of *m*.

Modern Ophthalmoscope.—For the original ophthalmoscope of Helmholtz a concave mirror of 20 cm. focal length with a central opening has been substituted. This mirror *O* (Fig. 47) throws convergent rays into the eye *E*; and these, being made more convergent by the refracting media, cross in the vitreous humour, and light up part (*a b*) of the fundus. From every point of this illuminated surface rays are reflected back again out of the eye. If the latter be emmetropic, the rays from any one point become parallel on leaving it; and some of these parallel rays, passing through the

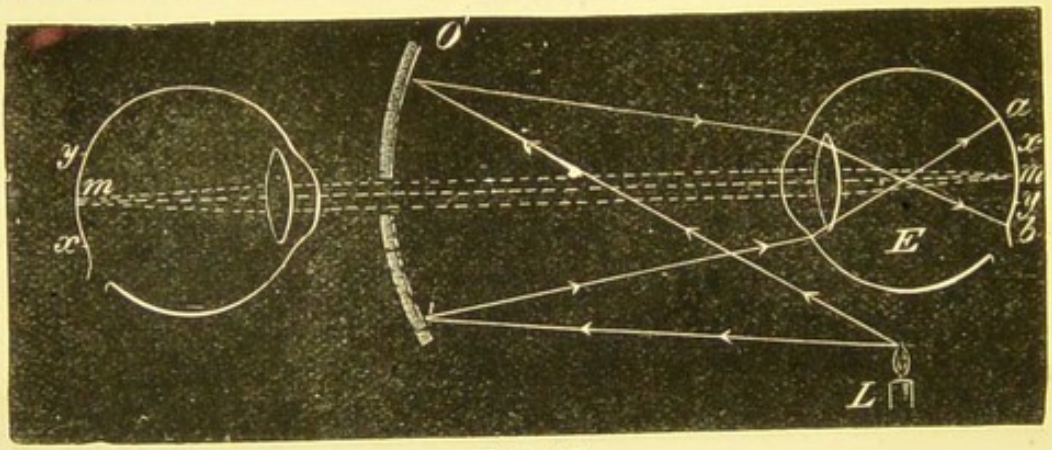


FIG. 47.

aperture (*c*) of the ophthalmoscope, fall into the observer's eye, and, if it be emmetropic, are brought to a focus on its retina; the rays from *m* at *m'*, those from *x* at *x'*, and those from *y* at *y'*; and thus an image of the part *x m y* is formed on the observer's retina.

The foregoing method of examining with the ophthalmoscope is called **The Direct Method,** or **the Examination of the Upright Image.** By it the various parts of the fundus are seen in their natural positions, but much enlarged (about 15 diameters in the emmetropic eye); and it is, consequently, very valuable for examining minute details.

It is necessary for this method that the surgeon should approach his eye as close as possible to the eye under

examination, in order to receive as much of the light coming out of it as possible.

It is also necessary for this method that the accommodation both of the surgeon's and of the patient's eye be at rest, as otherwise the rays coming from the latter cannot form an image on the retina of the former, at least if both be emmetropic.

If the patient exert his accommodation, the rays will, on leaving his eye, become convergent, instead of parallel, and, falling into the surgeon's eye, will be brought to a focus in front of his retina. If the surgeon exert his accommodation, the parallel rays from the patient's eye will likewise, on falling into his, the surgeon's eye, be brought to a focus in front of his retina. And if both patient and surgeon accommodate, the focus of the rays from the patient's fundus oculi will, of course, lie still farther in front of the surgeon's retina. The patient's accommodation can be relaxed by making him gaze at the black wall behind the surgeon's head, or his accommodation may be paralysed with atropine. But atropine should never be used unless absolutely necessary, owing to the inconvenience it causes the patient.

Voluntary relaxation of the accommodation on the part of the surgeon is often a matter of much difficulty to beginners. The ciliary muscle, not being a voluntary muscle, is not under our direct control, and can be influenced only in a secondary way through the convergence of the optic axes; for this convergence is regulated by voluntary muscles (the internal and external recti), and is intimately associated with the effort of accommodation. With parallel optic axes our accommodation is relaxed; therefore, when we want to relax our accommodation, we produce parallelism of our optic axes. This sounds easy enough; yet, when the beginner approaches his eye close up to that of his patient, the knowledge that he is so close to the object he wishes to see renders the accomplishment of this parallelism and relaxation of accommodation very difficult to many.

It is not easy to teach another person how to relax his accommodation, but the following hint may be of use. Take a printed page, and hold it at the ordinary reading distance, so that the type may be clearly seen; then gaze vacantly at it, so that the type may become indistinct. The accommodation is now relaxed, and the act is accompanied by a peculiar sensation in the eyes. When examining in the erect image, cause this same sensation to take place; and it may be assisted if, with the eye which is not in use, the black wall behind the patient's head be gazed at.

The Indirect Method, or **The Examination of the Inverted Image,** is employed in order to obtain a more general view of the fundus than the direct method admits of.

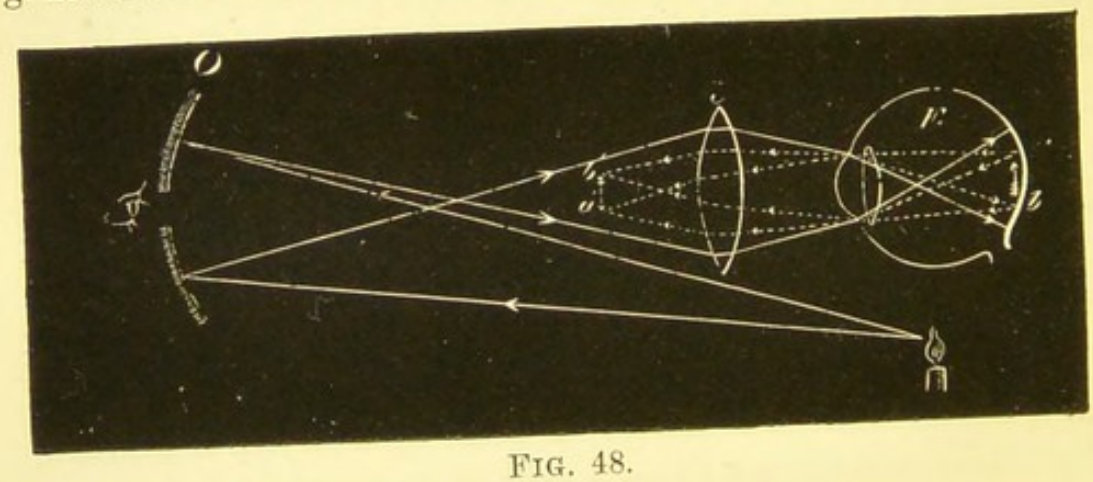


FIG. 48.

In addition to the ophthalmoscope, a convex glass (l, Fig. 48) of about 14 D is here used. The latter is held about 10 cm. from the eye (E) under examination, while the observer throws the light through it into the eye. In passing through the lens the rays are made convergent, and this convergence is increased by the refracting media, so that the rays cross in the vitreous humour, and light up a portion of the fundus oculi. From any points a and b of this illuminated place pencils of rays pass out again from the eye, and, becoming parallel, pass through the lens, and are united by it at a' b'; and thus a real inverted image is formed of the part $a\,b$, which image may be seen by the observer whose eye is placed behind O. The stronger the lens l, the more

convergent must rays from the examined eye be made; and, consequently, the closer must $a'\ b'$ be to each other, and the smaller and brighter must be the image formed. The weaker the lens l, the larger and less brilliant is the image, and the less annoying to the surgeon are the reflexes from the surfaces of the lens.

In examining by the indirect method, the observer first places the upper edge of the ophthalmoscope to his right supra-orbital margin, and, taking care that he is looking through the central opening of the mirror, he reflects the light of the lamp into the patient's eye at a distance of about 50 cm. A red glare from the fundus will then be seen in the pupil. Keeping the pupil illuminated, the convex 14 D, held between the forefinger and thumb of the surgeon's left hand, is brought up in front of the patient's eye, and kept there in the perpendicular position; the surgeon steadying this hand with the tip of the little finger on the patient's forehead. The convex glass is now removed just far enough from the patient's eye to cause the margin of the pupil to disappear out of the surgeon's field of vision. The observer then ceases to look into the eye, and fixes his gaze on the convex glass, when the inverted image of the fundus should at once become visible—and will seem to be situated in the convex lens, although it really is in the air somewhat this side of the lens.

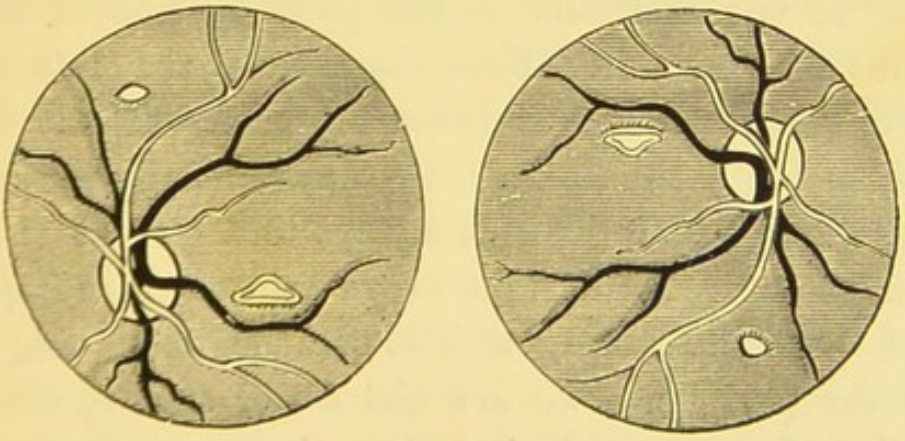


FIG. 49.

The diagram (Fig. 49) serves to illustrate the effect of inversion of the image.

The left eye is seen in the upright image in the left-hand

picture, while the same eye is seen in the inverted image in the right-hand picture. In the diagram the two images are of the same size, for the sake of convenience; although, of course, in reality the upright image is much larger than the inverted image. Moreover, it should not be supposed that nearly the whole fundus oculi, as here represented, can be taken in at one view with the ophthalmoscope. The portion visible with the ophthalmoscope at one moment, even in the inverted image, is small; so that it is necessary to examine the different regions in detail, in order to become acquainted with their condition.

The reflex from the surface of the cornea gives a good deal of annoyance to every beginner. It cannot be done away with; but, as it moves in the opposite direction to a motion of the object lens, it is possible to see past it. The reflections from the convex object-lens are also extremely annoying, but may be removed to a great extent from the line of sight by a slight rotation of the lens on its axis. If a very high convex lens, say + 20 D, be used, the reflections from it are more disturbing than from a lower number, say + 14 D.

To examine *The Optic Nerve*, the surgeon sits in front of the patient, and directs him to turn his eye somewhat to the nasal side, and slightly upwards; because the papilla is situated about 15° to the inner side of the posterior pole of the eye, and about 3° above it. For instance, if the left eye be examined, the patient is to direct his gaze, without turning his head, to the right and a little upwards, say towards the surgeon's left ear. It is well always to seek out the optic papilla in the first instance, not only because it is so important a part of the fundus oculi, but also because, examining from it towards the periphery, we are the better able to determine the locality of any pathological alteration.

Should the patient not direct his gaze in such a way as to enable the surgeon to see the optic papilla, or other desired region, it may be brought into view either by a motion of the

surgeon's head in the opposite direction, or by a motion of the convex lens in the same direction, or by a combination of both these manœuvres.

The Macula Lutea should then be examined. It may be seen by directing the patient to look straight at the hole of the ophthalmoscopic mirror, for it will then correspond with the macula lutea of the observer's eye. It is more readily seen in the inverted than in the upright image; but its examination is often very difficult, owing to contraction of the pupil produced by the strong light falling on so sensitive a portion of the retina, and by the reflections from the surfaces of the cornea and crystalline lens, which fill the area of this contracted pupil. It is, therefore, a better plan to direct the patient to look somewhat to the side of the eye under examination—*e.g.*, to the right side of the observer's forehead, if the right eye be under examination, and then by motions of the convex lens to bring the macula lutea into view.

After this *The Periphery of the Fundus* in every direction is to be examined by making the patient look upwards, downwards, to the right, to the left, etc.

Estimation of the Refraction by Aid of the Ophthalmoscope.

From what has been said with reference to the Direct Method of ophthalmoscopic examination, it will have become evident that this method affords a means for determining the refraction of the eye.

At a little distance from the observed eye into which light from the ophthalmoscopic mirror is thrown, the surgeon will be able to see some of the details of the fundus, if it be either myopic or hypermetropic; but if it be emmetropic he will be unable to do so. The reason for this is that in myopia the rays coming out of the eye form an inverted image at the far point of the eye in the air, and this image can be seen by the observer who accommodates his eye for that point. In hyper-

metropia the rays coming out divergently from the eye pass into the observer's eye, and, by an effort of accommodation on his part, he will see an upright image of the portion of the patient's fundus oculi from which they come. But in emmetropia, inasmuch as the rays come out parallel, those from any two points (*m*, *n*, Fig. 50) at a short distance from each other in the fundus, on emerging from the eye, diverge quickly from each other, and the observer a little way off (at *A*) receives none of them into his eyes, or obtains only an indistinct image, or red glare. If he go very close to the eye he can see details.

If, on the observer moving his head from side to side, the

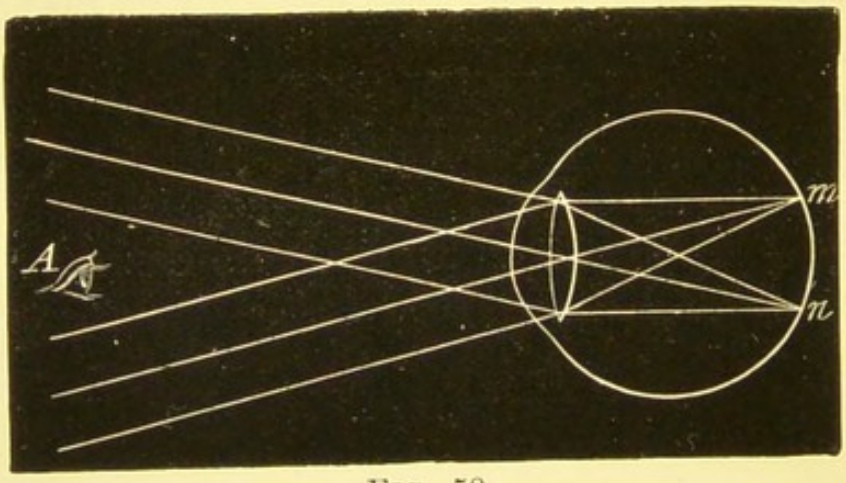


FIG. 50.

vessels, etc., of the observed fundus move with him, the case is one of hypermetropia, because the image is an erect one, which is situated behind the plane of the pupil to which it is referred. If the vessels, etc., move in the opposite direction to that of the observer's head, the observed eye is myopic, because there the image is inverted, and in front of the pupil.

For the quantitative determination of ametropia a refraction ophthalmoscope is required. This instrument provides a number of convex and concave lenses, capable of being brought into position behind the sight-hole in rapid succession by a simple mechanism.

It is necessary, in the first instance, that the surgeon be aware of the nature of his own refraction.

If the Surgeon be Emmetropic, he can see the fundus oculi of an emmetrope in the upright image without any lens, provided he go close enough; as the parallel rays coming from the examined eye will be focussed on his retina, because his eye is adapted for parallel rays.

In order to see the fundus oculi of a hypermetrope, without any effort of accommodation, he must place such a convex lens behind his ophthalmoscope as will render the divergent rays coming from the patient's eye parallel, before they pass into his eye. This lens is the measure of the patient's hypermetropia, because it shows how many dioptries the eye wants of being emmetropic; or, in other words, so that the rays coming from it may be made parallel. The lens which makes the divergent rays coming from the patient's retina parallel would also give to parallel rays passing into the eye such convergence that they would meet on the retina—*i.e.*, it would correct the hypermetropia.

The emmetropic surgeon can, of course, see the fundus oculi of a hypermetrope by the direct method without the correcting glass, if he use his accommodation to overcome the divergence of the rays, and this is usually the case in the lower degrees of hypermetropia. The surgeon generally relaxes his accommodation according as he substitutes convex lenses for it, until he reaches the *strongest* lens with which he can distinctly see the fundus. This is the correcting lens.

To see the fundus oculi of a myope, the emmetropic surgeon must place a concave glass behind his ophthalmoscope, in order that the convergent rays coming from the observed eye may be made parallel, before they pass into his eye; and the *lowest* concave lens which enables him to see the fundus oculi is the measure of the myopia, as showing by how many dioptries it is in excess of emmetropia.

The emmetropic surgeon cannot possibly see the fundus oculi of a myope without the correcting glass, as the rays are brought to a focus in front of his retina, and if he use his

accommodation he merely makes them still more convergent. But, by means of an effort of his accommodation, he can see the myopic fundus with a lens which over-corrects the myopia, and hence the importance of selecting the *weakest* concave glass with which the fundus is distinctly seen.

If the surgeon be ametropic, he may either correct his ametropia by wearing the suitable lens, and then proceed as though he were emmetropic; or else, and which is perhaps the better plan, he may add or subtract the amount of his ametropia from that of his patient's. For example :—

The Hypermetropic Surgeon of, say 3 D, requires a + lens of 3 D in order to see an emmetropic fundus oculi, this lens going altogether to correct his own defect. If, in order to examine the fundus of another eye, he require a + lens of 6 D, the examined eye must be hypermetropic 3 D, the other 3 D going to correct the surgeon's H. If he be able to see the fundus oculi under observation without any lens, it shows that the eye has an excess of refraction corresponding to the want of refraction in his own eye—that is to say, it is myopic 3 D. If he require a concave 2 D, his want of refraction—his hypermetropia—is not enough by that number of dioptries, and he has to do with an eye which is myopic 5 D (3 D + 2 D). Again, if he can see the fundus distinctly with a + lens, say + 1·0, which is less than his own correcting glass, this shows that the eye he is examining is myopic, but myopic to a lesser degree—in this instance by 1 D—than he himself is hypermetropic, and the examined eye here would be M. 2·0 D (*i.e.*, 3·0 − 1·0).

If the Surgeon be Myopic, say 2 D, he requires a − 2 D to see the fundus of an emmetropic eye, this lens going wholly to correct his own ametropia. If he see the fundus with a − 7 D, the examined eye has M. 5 D, because 2 D has been used in correcting the surgeon's M. If he be able to see a fundus without any lens, the patient has H. 2 D, the want of refraction in the latter's eye compensating exactly for the excess of

refraction in the surgeon's eye. If he find it necessary to use a + lens of 7 D, it will indicate that his excess of refraction is not able to make up for the defect of refraction in his patient's eye, and that the latter has H. = 9 D (2 D + 7 D). If he have to use a − lens, say − 1·0, which is less than his own correcting glass, this shows that the eye he is examining is hypermetropic to a lesser degree—in this instance by 1·0 D—than he himself is myopic, and the hypermetropia here would be 1·0 D (*i.e.*, 2·0 − 1·0).

The Existence and Degree of Astigmatism may be Determined with the Ophthalmoscope.—We know that astigmatism is present, if, in the upright image, we see the upper and lower margins of the disc and the horizontal vessels well defined, while the lateral margins and the vertical vessels are blurred, or *vice versâ*. Again, we know that astigmatism is present, if, in comparing the shape of the optic disc in the upright and inverted images, we find it to be an oval with its long axis perpendicular in the former, and with its long axis horizontal in the latter, showing that the refracting media are more powerful in the vertical than in the horizontal meridian.

We may ascertain the kind and degree of astigmatism as follows:—If, in the upright image with relaxed accommodation, we can see the retinal vessels in one meridian distinctly, while in order to see those in the opposite meridian a concave or convex lens behind the ophthalmoscope is required, we know that the case is one of simple myopic or hypermetropic astigmatism; the emmetropic meridian being that at right angles to the vessels [1] seen without any lens, and the number of the lens indicating the amount of ametropia in the other meridian.

If, in the two principal meridians, two concave lenses or two convex lenses of different strength be required, we have to deal with a case of compound astigmatism, myopic or hypermetropic; the greatest error of refraction being in the

[1] The vessels may be regarded as lines, and the explanation given on pp. 45 and 46 applies to them also.

meridian at right angles to that one, the vessels of which are made distinct by the strongest lens.

If a concave lens be required to bring into distinct view the vessels in one meridian, while a convex lens is required for the opposite meridian, the case is one of mixed astigmatism. Myopia exists in the meridian at right angles to that in which the vessels are brought into view by the concave lens, and hypermetropia exists in the opposite meridian.

I would again impress upon the reader the absolute necessity of thoroughly relaxing his accommodation in all examinations in the upright image. Paralysis of the patient's accommodation with atropine is necessary in most cases, where accuracy in the determination of the refraction with the ophthalmoscope is required, and can hardly be done without in cases of hypermetropia and of hypermetropic astigmatism, owing to the cramp of accommodation which is almost always present.

Retinoscopy.

Another and very useful method for determining the refraction by the ophthalmoscope is termed The Shadow Test, or Retinoscopy. The appearances upon which this method depends are due to the play of light, reflected from the mirror, on the fundus oculi. Either a concave or a plane ophthalmoscopic mirror may be employed. I invariably use a plane mirror; but, as I believe the majority of ophthalmologists still use the concave mirror in retinoscopy, I shall describe the theory and use of the method by its aid, and then that by aid of the plane mirror will be readily understood.

If the rays from a light (*L*, Fig. 51) be reflected from *The Concave Mirror* (*m*) of an ophthalmoscope, they cross at a certain point (*A*), and form there an inverted image of the flame, and then diverge again. If these diverging rays be made to pass through a convex lens (*B*) placed at such a distance in front of a screen (*E*) that the rays meet at a focus on the latter, a very small and brilliant upright image (*O*)

of the flame is there formed, surrounded by a deep shadow. If the screen be moved slightly towards the lens (to *H*), so that the focus of the rays would lie behind it, or if it be removed slightly away from the lens (to *M*), so that the focus come to lie in front of it, the brilliancy of the image on the screen and the intensity of the surrounding shadow are reduced. Because, in each instance, a circle of diffusion, and not an accurate image, is formed on the screen, and the farther the focus of the pencil of rays is situated from the screen in either direction, the weaker does the image become, and the more ill-defined the shadow.

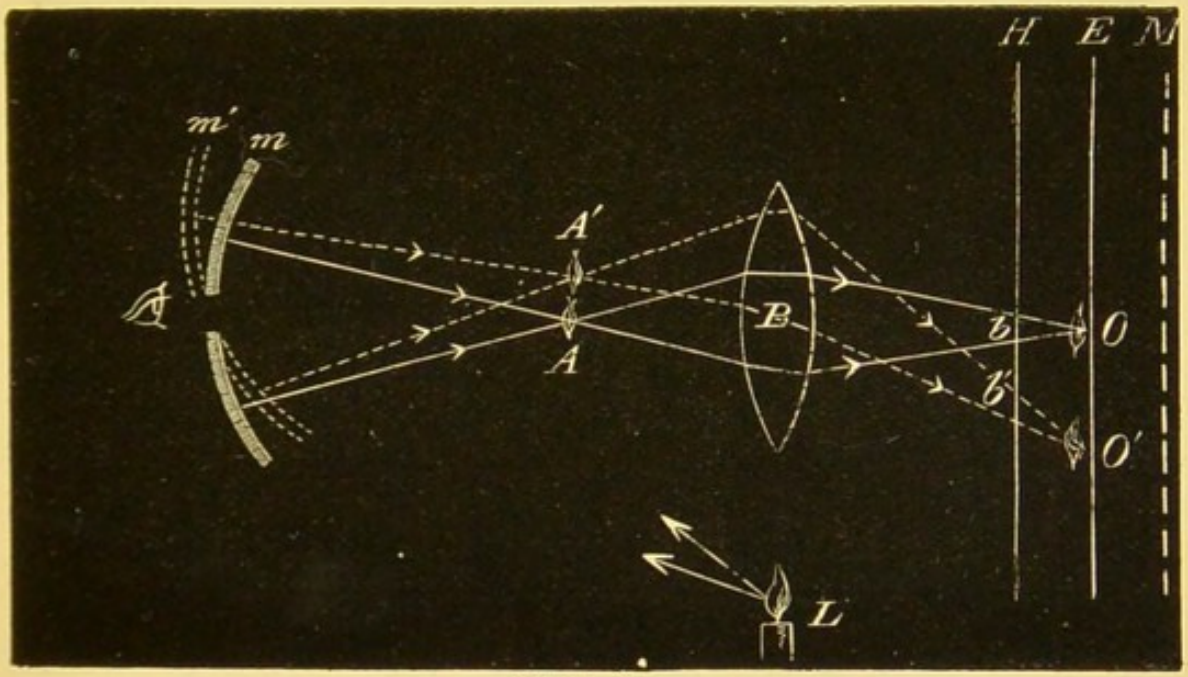


FIG. 51.

If the mirror be rotated in various directions, the illuminated part[1] and the shadow are seen—care being taken to look at the screen directly, and not through the lens—to move on the screen in the opposite direction to the motion of the mirror. For example, if the position *m'* (Fig. 51) be given to

[1] "The area of light," "the image," "the illuminated area, or part" of the fundus, and "the illumination," are different terms for one and the same thing. "The shadow" or "shade" refers merely to the margin of the illuminated area—*i.e.*, where the illumination ceases, and darkness begins; it does not mean that the shadow of any object is thrown on he fundus oculi. When we speak of the motion of the shadow, we

the mirror, the path of the rays reflected from it is shown by the dotted lines, and the image of *O* is moved to *O'*. This will also be the case if the screen be at *H* or at *M*. These three positions of the screen may be supposed to represent emmetropia (*E*), hypermetropia (*H*), and myopia (*M*). Fig. 51 more particularly illustrates the motion of the light and shade in *E* and *H* only, while Fig. 52 demonstrates that in *M*.

In the eye, in like manner, the area of light and shade in the pupil moves against the motion of the mirror. Now, we cannot, of course, see the real motion on the retina directly,

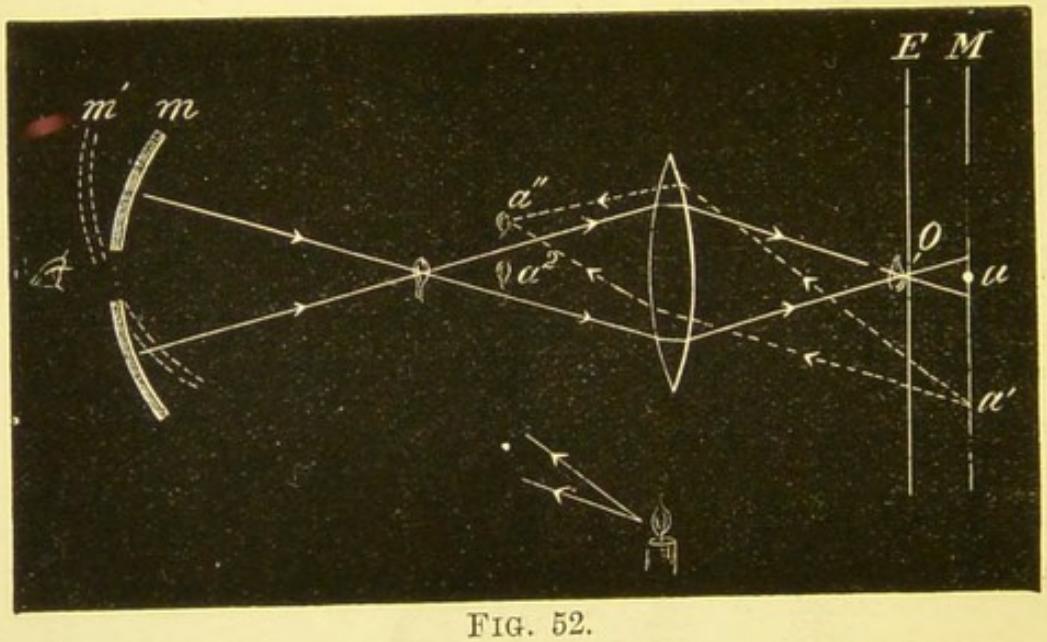


FIG. 52.

but only through the dioptric media, and they will influence the apparent motion according to the condition of the refraction.

In emmetropia and in hypermetropia the rays coming out of the observed eye are parallel and divergent, respectively; and, consequently, an upright image being formed by them

mean that the margin of the illuminated area, or boundary-line between illuminated and non-illuminated area, moves along with the illuminated area in response to the motion of the mirror. It is easier to learn how the illuminated area moves by watching the margin of the shadow (which comes across the pupil from behind the iris like a revolving shutter across a shop window), and hence we have come to talk always of the motion of the shadow, and not of the motion of the illuminated part.

in the observer's eye, the true motion given by the mirror is perceived.

In myopia, at least in all cases of more than 1 D, the observer does not see an upright image of the flame on the fundus of the observed eye, but a real inverted aerial image formed between his mirror and the unobserved eye. The reason of this is, that the rays coming out of the patient's eye are convergent, and meet at a focus, which is the far point of the eye, and form there an inverted image of the object from which they come, and which, in this instance, is an upright image of the flame (the illuminated area). When, therefore, the upright image on the fundus moves against the mirror, the inverted image (which the observer sees) moves in the opposite direction—*i.e.*, with the mirror. For example, if in Fig. 52 we suppose a to be the position of the image on the fundus of a myopic eye, and a^2 the position of its real inverted aerial image, a motion of the mirror to m' (the rays reflected from m' are omitted, in order to avoid confusion in the diagram) throws the image of a to a', as already explained, but the inverted aerial image of a' is formed at a''—*i.e.*, it seems to have moved with the mirror.

In myopia alone, then, does the image move with the mirror; while in emmetropia and hypermetropia it moves against the mirror. In low myopia (1 D and less), as will just now be seen, the image also moves against the mirror.

From what has been said, it is evident that the higher the ametropia (the farther from the screen, in Fig. 51, the focus of the rays) the larger and feebler the illumination becomes (*i.e.*, the greater the circles of diffusion), and the more crescentic the margin of the shadow, because it is the margin of a circle of diffusion.

Again, the extent of the motion of the image, and its rate, are in inverse proportion to the degree of the ametropia. Thus, if Fig. 53 represents a myopic eye, whose far point is situated at a^2, a motion of the mirror to m' may be supposed to throw

the illuminated part to a', and then a^2 will move to $a^{2'}$. But, if the myopia be of less degree, so that the far point is at a^3, the same motion of the mirror will throw a^3 to $a^{3'}$, and the distance between these two latter points is evidently much greater than that between a^2 and $a^{2'}$. In a hypermetropic

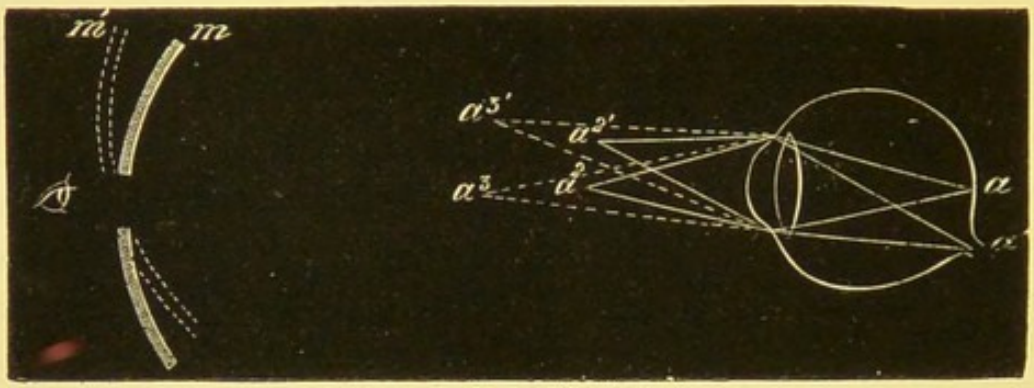


FIG. 53.

eye (Fig. 54) the image may be supposed to be formed at a, and a motion of the mirror to m' will throw it to a'; while in a higher degree of hypermetropia it would be formed at b, and the same motion of the mirror would throw it to b'. The distance between b and b' is much greater than that between a and a'.

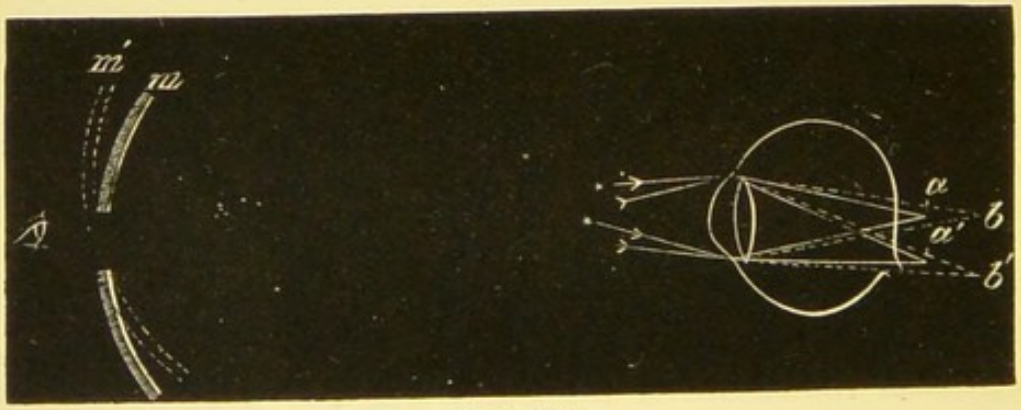


FIG. 54.

In practising retinoscopy with the concave mirror the surgeon sits 1·20 m. in front of the patient. The eye to be examined is shaded from the direct rays of the lamp, if the latter be placed beside the patient; but a better plan is to have the light above his head. The focus of the mirror

should be 22 cm., and any error of refraction of the surgeon is to be corrected. The light is then thrown into the eye at an angle of about 15° with its axis of vision, so that, if the pupil be not under the influence of atropine, the macula lutea may be avoided. In children, and when the pupil is very small, it is advisable to dilate it with atropine, and then the region of the macula lutea may be utilised. When now the ophthalmoscope is rotated in different directions, motions of the light and shade on the fundus oculi are seen in the pupillary area. The surgeon directs his attention to the *edge of the shadow* rather than to the illuminated part, for its motion is more easily appreciated. If the edge of the shadow move *with* the motion of the mirror, *myopia* is present; if it move *against* the mirror, emmetropia, hypermetropia, or myopia of 1 D or less, is present.

The reason why the motion is against the mirror in cases of M. 1 D and less is, that the surgeon being seated only 1·20 m. from the eye he is examining, if that eye have a myopia of 1 D, its far point is so close to his eye that he cannot clearly observe the image there formed; but if the myopia be of even slighter degree, the image will be formed behind the surgeon's head, and he gets a shadow moving against the motion of his mirror, because the image he then sees is the upright one of the patient's fundus oculi and not the inverted aerial image.

We proceed as follows:—A trial spectacle-frame is put on the patient's face. If the shadow move with the mirror, we know at once the eye is myopic. To find the degree of myopia, the surgeon puts a low concave-glass, say − 1 D, into the frame; and, if the shadow still move with the mirror, he puts in a higher number, say − 1·5 D, and so on, until he comes to a glass which makes the image move against the mirror. If this be − 3 D, the myopia is 3 D. It might be supposed, as the shadow now moves against the mirror, that this glass over-corrects the myopia, but this is not so; because, as already explained, when the myopia is very low, the image

is formed close to the surgeon's eye, or behind his head, and he consequently gets a shadow moving against the mirror, although low myopia, and not emmetropia, is present. Consequently, — 0·5 D or — 1 D has to be added on to the lens, which gives the effect of no distinct shadow; or rather, by the above plan, it is not deducted from the lowest lens, which makes the shadow move against the mirror.

If the shadow move against the mirror, we have to determine whether the eye be emmetropic, hypermetropic, or slightly myopic. Should the illumination be bright, and the shadow well defined, the eye is emmetropic, or not far removed from it; and, if the shadow be ill defined and crescentic, we may feel sure the eye is highly hypermetropic. We first put on + 1 D, and, if the motion be still against the mirror, the case is one of hypermetropia, and higher numbers are at once proceeded with, until that one is reached which causes the shadow to move with the mirror. The measure of the hypermetropia is 1 D less than the glass so found, for it has evidently over-corrected the defect.

If, however, on putting on + 1 D we find the shadow to move with the mirror, we change it for + 0·5 D; and, if still the motion be with the mirror, the eye is, beyond doubt, slightly myopic, — 0·5 D or so. But, if with + 1 D the shadow move with the mirror, while with + 0·5 it continue to move against it, the eye is emmetropic.

It may be found that in two opposite meridians there is a difference in the motion of the shadow, and this leads us to diagnose the presence of astigmatism. When the difference is one merely of rapidity of motion, or of intensity of illumination and shadow, we know that we have to deal with either simple or compound astigmatism. But if in the two meridians there be a difference in the direction of the motion, then it is a case of mixed astigmatism. The best method for ascertaining the degree of astigmatism and its correcting glass is to correct each of the principal meridians separately with spherical

lenses. In compound astigmatism the difference between the two lenses found indicates the degree of astigmatism, and also the cylindrical lens, which, combined with the correcting spherical lens for the least ametropic meridian, is required to neutralise the defect. In mixed astigmatism the addition of the two numbers gives the cylindrical lens, while one or other of them, usually the + D, is used as the spherical lens.

With *The Plane Mirror* the source of illumination of the observed eye is not a real inverted image of the light, as in the case of the concave mirror, but a virtual upright image behind the mirror; and, as this image moves in the opposite direction to the motion of the mirror, the motion of its illumination on the fundus of the patient's eye must be *with* the mirror in all cases, and not against it, as in using the concave mirror.

With the plane mirror, therefore, the shadow is seen to move *with* the motion of the mirror in H. and E. But in M. it seems to move *against* the motion of the mirror, for what we here see is an inverted image of the fundus situated at the far point of the eye. If the myopia be high, this inverted image will be close to the eye; if low, it will be far away from it. In using the plane mirror it is important to remember this point, because, if the observer go nearer to a myopic eye than its far point, he will not obtain a myopic motion, but one which is the same as that in E. or H. Consequently, in using the plane mirror, the rule is to go as far from the eye under examination as possible. If at the beginning the surgeon retire a little more than 2 metres from the eye, and there obtain a with-motion, he at once knows that the eye is not myopic 0·5 D; or, if he stand a little more than 4 metres away, and obtain the same motion, he knows there is not a myopia of even 0·25 D present. If the myopia be high, he will be able to begin close to the patient, but must gradually retire from the eye as he increases the number of the concave glass put up—for the far point is

thereby moved farther off—in order that he may not think he has corrected the myopia before he really has done so. Again, if at every distance the motion be with the mirror, the surgeon has to decide whether this indicate E. or H. He does this by putting a low lens, say + 0·25, before the patient's eye, and if then, standing at a distance of 4 metres, the motion be altered by this glass to one against the mirror, he knows that the eye has not a hypermetropia of 0·25 D, consequently that it is emmetropic; but if this lens do not at that distance cause a change in the motion of the shadow as originally obtained, the eye must be hypermetropic to at least the extent of 0·25 D; and, in order to ascertain how much more of H. than this may be present, it is now only necessary to go on increasing the strength of the lens in front of the patient's eye, until one is reached which, at 4 metres from the eye, produces the myopic motion. The observer knows that he has now slightly over-corrected the hypermetropia of the eye, and that the next lens lower is its measure.

With some practice it is possible, unless the pupil be small, to obtain sufficient light from the fundus with the plane mirror at a distance of 4 metres.

I find this method much more easily worked than that with the concave mirror. It has the advantage, too, of not requiring any wearisome addition to, or subtraction from, the data obtained.

The pleasantest plane mirror is one of 4 cm. diameter, and of which the sight-hole is 4 mm. in diameter.

Methods for estimating the refraction of the eye by finding the position of the inverted image have also been devised; the best one being that of Schmidt-Rimpler.

Opacities in the Refracting Media can be best observed by examination with strong convex lenses, in the upright image. The further forward the opacity lies the more hypermetropic (so to speak) it is, and the stronger the lens required. Very

minute opacities of the cornea can be seen in this way with a + 18 or + 20 D lens in the ophthalmoscope.

FOCAL, OR OBLIQUE, ILLUMINATION

is employed for the examination of the cornea, iris, and lens. With a high + lens (16 to 18 D) the light of the gas flame is concentrated on the part to be examined with an oblique, not a perpendicular, incidence of the concentrated rays. Small foreign bodies in the iris, cornea, or lens, or opacities in either of the latter, can be thus detected. Extremely delicate opacities in the cornea are not seen best with the strongest illumination which can in this way be produced; but, rather, by the half-light which is obtainable at the edge of the cone of light passing from the lens. In examining the centre of the crystalline lens, the incidence of the light must necessarily be more perpendicular.

THE NORMAL FUNDUS OCULI AS SEEN WITH THE OPHTHALMOSCOPE.

Reference has been made to the enlargement of the image of the fundus oculi seen with the ophthalmoscope. The cause of this enlargement is, that the fundus is observed through a dioptric system, at, or close to, the principal focus of which it is situated, and which, consequently, magnifies it to our view. The enlargement of the inverted image is not so great as that of the upright image, and it is smaller the shorter the focal length of the convex lens employed. The inverted image of a hypermetropic eye is larger than that of an emmetropic eye, and the latter larger than that of a myopic eye. It is possible to determine mathematically the degree of enlargement of the image, but into this it is not necessary to enter.

The Optic Papilla.—This is the first object to be sought for by the observer. It presents the appearance of a pale pink disc, somewhat oval in shape, its long axis being vertical. Occasionally the long axis lies horizontally, and sometimes

the papilla is circular. The papilla is generally surrounded by a white ring, more or less complete, called the sclerotic ring, and often, outside this again, by a more or less complete black line, the chorioidal ring. The sclerotic ring is due to the chorioidal margin not coming quite up to the margin of the papilla, the foramen in the chorioid for the passage of the optic nerve fibres being somewhat larger than that in the sclerotic, and, consequently, a narrow edging of the white sclerotic is exposed. The chorioidal ring is the result of a hyper-development of pigment at the margin of the chorioidal foramen. The complexion of the optic papilla results from the pink hue derived from its fine capillary vessels, combined with the whiteness of the lamina cribrosa, and the bluish shade of the nerve fibres. It is frequently not equal all over, but is paler on the outer side, where the margin is more defined, and where the nerve fibres are often fewer than on the inner side. The apparent colour of the papilla depends, also, upon the complexion of the rest of the fundus. If the latter be highly pigmented, the papilla appears pale in contrast; while, if there be but little pigment in the chorioid, the papilla may appear very pink. The complexion of every normal papilla is not identical, and care must be taken not to make the diagnosis "Hyperæmia of the papilla," where merely a high physiological complexion is present. The upper and lower margins of the papilla are often, especially in young people, a little indistinct, and show a delicate striation by the direct method of examination. This may be greatly exaggerated in hypermetropes, and has in them been sometimes taken for optic neuritis.

A physiological excavation of the optic papilla is often met with. It is always on the temporal side of the papilla, and can be recognised from the parallax[1] which may be produced, and from the paleness of this portion of the papilla. When

[1] For explanation of the parallax see chap. xii.

the excavation is very deep, one may sometimes observe the lamina cribrosa in the form of grey spots (the nerve fibres), surrounded by white lines (the fibrous tissue of the lamina).

A physiological differs from a pathological excavation, by the fact that it does not reach the margin of the papilla all round. It is caused by the crowding over of the nerve fibres to the inner side of the papilla. Yet sometimes a healthy optic papilla will be met with, in which the excavation apparently reaches the margin all round. Doubtless, in such cases, the thickness of the translucent nerve-fibre layer alone it is, which is interposed between the sclerotic margin and the margin of the cup all round.

The Normal Retina is so translucent that it cannot be seen; or, at most, a shimmering reflection, or shot-silk appearance, is obtained from it, particularly about the region of the yellow spot and along the vessels, but also towards the equator of the eye, and especially in dark eyes, and in young people.

A peculiar, but physiological, appearance, known as "opaque nerve fibres," is occasionally seen. It is produced by some of the nerve fibres forming the internal layer of the retina regaining the medullary sheath on the distal aspect of the lamina cribrosa, or near the margin of the papilla, which they had lost in the optic nerve just before entering the lamina cribrosa; the rule being that the nerve fibres lose their medullary sheath at the latter place definitely, and enter the retina as axis cylinders only, and hence are quite translucent. Instead of that, in these cases their fibres reflect the light strongly, giving the effect of an intensely white spot, commencing at the papilla, extending more or less into the surrounding retina, and terminating in a brushlike extremity. This appearance is constant in the rabbit's eye.

The Macula Lutea is generally seen as a bright oval ring with its long axis horizontal, this ring being probably a reflex

from the surface of the retina. It is remarkable that this halo is not visible with the direct method of examination—a fact due probably to the illumination being much weaker than with the indirect method. The area inside the ring is of a deeper red than the rest of the fundus, and at its very centre there is an intensely red point, the fovea centralis. This ring is not seen in old people.

The General Fundus Oculi surrounding the optic papilla and macula lutea varies a good deal in appearance, according to the amount of pigment contained in the chorioid, and in the pigment-epithelium layer of the retina. 1. If there be an abundant supply of pigment in each of these positions, the chorioidal vessels are greatly hidden from view, and the effect is that of a very dark red fundus. 2. If there be but little pigment in the pigment-epithelium layer, the larger chorioidal vessels may be visible, and the fundus may appear to be divided up into dark islands surrounded by red lines. 3. If the individual be a blonde, there is little pigment either in the pigment-epithelium layer or in the chorioid, and the fundus is seen of a very bright red colour, the chorioidal vessels down to their fine ramifications being discernible. In albinos even the chorioidal capillaries may be seen.

The Retinal Vessels.—The arteries are recognised as thin bright red lines running a rather straight course, in the centre of each of which is a light-streak. As to the cause of this light-streak there is considerable divergence of opinion. Some attribute it to reflection from the coats of the vessel, or from the surface of the blood column; while others believe, that the light is reflected from the fundus through the vessel, which then acts as a very strong cylindrical lens. This light-streak divides the vessel into two red lines. The veins are darker, wider, and more tortuous in their course than the arteries, and, their coats not being so tense, the light-streak is very much fainter.

On reaching the level of the nerve-fibre layer of the retina,

the central artery and vein divide into a principal upper and lower branch. This first branching often takes place earlier in the vein than in the artery, and the former may even branch before appearing on the papilla, as in Fig. 55. The second branching may take place in the nerve itself; and, when this occurs, it will appear as though four arteries and four veins

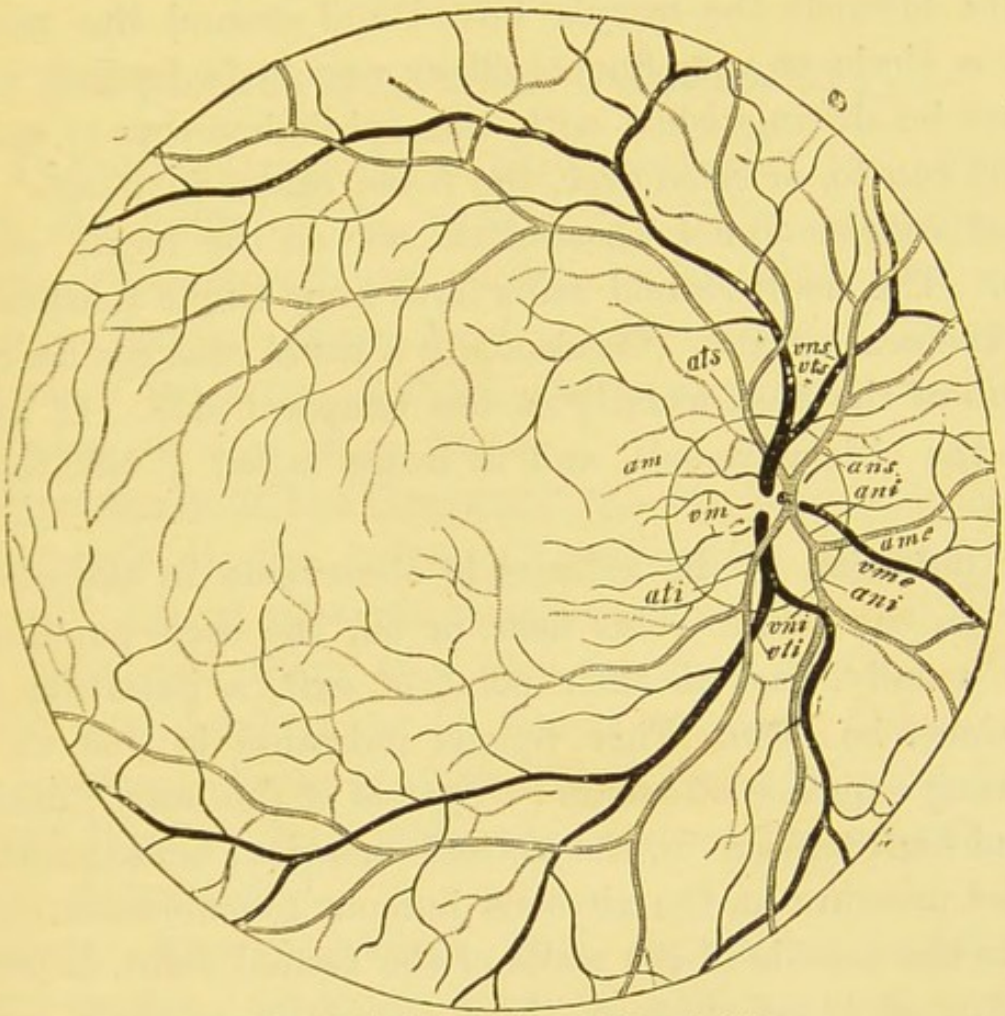


FIG. 55 (*Graefe and Sæmisch*).

a.n.s. Art. nas. sup.; *a.n.i.* Art. nas. inf.; *a.t.s.*, *a.t.i.* A. temp. sup. and inf.; *v.n.s.*, *v.n.i.*, Ven. nas. sup. and inf.; *v.t.s.*, *v.t.i.* Ven. temp. sup. and inf.; *a.m.e.*, *v.m.e.* Art. and ven. median; *a.m.*, *v.m.* Art. and ven. macularis.

sprang from the optic papilla; but, more usually, this branching occurs on the papilla, as in Fig. 55. The vessels produced by this second branching pass respectively towards the median and temporal side of the retina, and are termed by Magnus the Art. and Ven. nasalis and temporalis sup. and inf. (*vide* Fig. 55). The temporal branches run in a radial direction towards the anterior part of the retina. A small horizontal

branch, the Art. and Ven. mediana of Magnus, from the first principal branches, is found passing towards the nasal side of the retina. The temporal branches do not run in a horizontal direction, but make a *détour* round the macula lutea, sending fine branches towards the latter. Two or three minute vessels from principal branches run directly from the papilla towards the macula lutea; and around the macula lutea a circle of very fine capillary vessels is formed, which cannot be distinguished with the ophthalmoscope; but no vessels run to, or cross over, the fovea centralis itself. The retinal arteries do not anastomose, nor do the larger retinal veins. The small retinal veins have some slight anastomoses near the ora serrata. Occasionally a vessel emerges near the margin of the disc, usually at the temporal side. It arises from the ciliary vessels, and is hence called a cilio-retinal vessel.

No pulsation of the arteries is observable in the normal eye. In the larger veins near, or on, the optic papilla, or, more usually, just at their point of exit, a pulsation may sometimes be seen. This venous pulsation is due to the following sequence of events:—Systole of the heart; diastole of, and high tension in, the retinal arteries; consequent increased pressure in the vitreous humour; communication of this to the outside of the walls of the retinal veins, impeding the flow of blood through them, especially in their larger trunks, which offer little resistance, or at their exit from the eye, where they offer the least resistance; and in this way the veins are emptied: the blood gradually coming on from the capillaries overcomes the resistance, and the veins are for a moment refilled. The phenomenon can be most readily observed, if the normal tension of the globe be slightly increased by pressure of a finger.

CHAPTER IV.

DISEASES OF THE CONJUNCTIVA.

The Conjunctiva consists of three portions: the *palpebral*, lining the inside of the eyelids, the *bulbar*, covering the sclerotic, and a loose folded portion, uniting these two, which forms the *fornix*. When it reaches the margin of the cornea it overlaps the latter a little, and this region is known as the *limbus* conjunctivæ, or corneæ.

Hyperæmia of the Conjunctiva.—In this condition the blood-vessels of the palpebral conjunctiva especially are engaged. Slight chemosis sometimes appears, small vesicles may form, and there may also be some swelling of the papillæ, and development of lymph follicles. There is not any abnormal discharge from the conjunctiva, and herein lies the chief point of difference between this affection and simple conjunctivitis.

Causes.—Foreign bodies. Foul air, or air loaded with tobacco-smoke. Alcoholic excesses. Accommodative asthenopia. Stenosis lacrimalis, and other forms of lacrimal obstruction. The use of unsuitable spectacles, or the use of the eyes for near work without spectacles when the condition of the accommodation (—*e.g.*, hypermetropia, presbyopia) requires them.

Symptoms.—The eyes are irritable. There is lacrimation and photophobia, with hot, burning sensations, and sensations as of a foreign body in the eye, and the eyelids feel heavy. All these symptoms are aggravated in artificial light.

Treatment.—In addition to the removal of the cause, iced

compresses are to be applied to the closed eyelids for twenty minutes several times a day, and the instillation of a drop of tincture of opium and distilled water in equal parts morning and evening will be found beneficial. It is also desirable to wash out the lacrimal passages with an Anel's syringe, even where no decided lacrimal obstruction is present.

The eyes should be protected from the glare of light by dark glasses, and out-of-door exercise is to be recommended.

Conjunctivitis in general.—In addition to hyperæmia, there is here abnormal secretion. There are several forms of conjunctivitis, the discharge from each being more or less contagious. The secretion from any given form will not, however, always reproduce that form, but may give rise to another of greater or less severity. Infection takes place by the direct application of the secretion, or also—it is very generally thought—through the air, in which float particles of the infecting substance. This latter mode is especially liable to exist, it is said, in an ill-ventilated room, where a number of people affected with conjunctival diseases are lodged with others who possess healthy eyes—*e.g.*, in crowded charity-schools. The palpebral conjunctiva is often affected when the bulbar portion remains normal, and the conjunctiva of the lower lid is more frequently attacked than that of the upper lid.

Catarrhal, or **Simple Acute, Conjunctivitis.**—In mild cases the affection is confined to the palpebral conjunctiva, often even to the conjunctiva of the lower lid; but in the severer cases it extends to the bulbar conjunctiva. Lymph follicles and enlarged papillæ are frequently present, but not necessarily so. There is a sticky, serous secretion, which causes the eyelids to be fastened together on awaking in the morning, and sometimes produces ulceration of the inter-marginal portion of the eyelids (intermarginal blepharitis). In some of the very mildest cases this "stickiness" or "gumming" on awaking in the morning is a valuable diagnostic

sign, for it is, in such cases, difficult or impossible to recognise the very slight variation from the healthy appearance of the conjunctiva.

In the severer cases the papillæ are markedly swollen, and may even conceal the Meibomian glands from view. Also one often sees small ecchymoses in the bulbar conjunctiva, especially in certain epidemics. But these have no serious import.

Minute grey infiltrations sometimes form at the margin of the cornea. When there are many of them, they may become confluent and form a small grey crescent, which ulcerates, and thus a crescentic marginal ulcer is formed, and very occasionally such an ulcer is followed by iritis.

The catarrh may become chronic, and then the papillæ are more developed, while the blepharitis is liable to extend over to the cutis, causing eversion of the lower punctum lacrimale with resulting stillicidium, and this, in its turn, aggravates the conjunctival affection.

The Symptoms are those of a severe case of hyperæmia (sensations of sand in the eye, hot, burning sensations, weight of the eyelid), with the addition of the annoyance consequent on the secretion, which, by coming across the cornea, may cause momentary clouding of sight. Photophobia is not generally severe, unless there be some corneal complication.

Causes.—Draughts of cold air. Contagion. Foul atmosphere. As an epidemic. Foreign bodies. As a sequel of, or attendant on, scarlatina, measles, and smallpox.

Diagnosis.—The presence of the gummy secretion distinguishes this affection from mere hyperæmia of the conjunctiva. A common mistake amongst those not familiar with eye diseases is to regard a case of iritis as one of simple acute conjunctivitis, the redness of the white of the eye in the former affection being taken for conjunctival hyperæmia, etc.; and, moreover, a slight secondary conjunctivitis does, undoubtedly, attend many cases of iritis.

The circum-corneal sub-conjunctival vessels, which are the episcleral branches of the anterior ciliary vessels, are those which become engorged in iritis, and their engorgement gives rise to a pink or pale violet zone around the cornea, of which the separate vessels cannot be distinctly seen. The conjunctival vessels may be distinguished from the subconjunctival or ciliary vessels by the possibility of moving the former along with the membrane in which they are, by manipulations which can be made with the lower lid of the patient, while these manipulations do not affect the ciliary vessels. The separate conjunctival vessels, too, can be easily distinguished, and they are of a bright red colour. The condition of the iris itself, however, is that upon which the diagnosis finally depends. (See *Iritis*, chap. x.)

The Prognosis is good, if there be no reason to suspect that the mild form is but the commencement of a more severe inflammation. The infiltrations, and even the ulcers, which sometimes form at the margin of the cornea, are not often of serious import, and heal according as the treatment restores the conjunctiva to health.

Treatment.—Cold, or iced compresses, with the use of a 4 per cent. solution of boracic acid as a lotion, should be used frequently at the first onset, and, in mild cases, will alone bring about a cure. But the habit, which some patients so readily acquire, of bathing the eyes frequently with cold water, should not be permitted, for it is deleterious to the conjunctival affection. When, in a day or two, the irritation and swelling have somewhat subsided,—or from the very commencement, if there be not much irritation,—a solution of nitrate of silver, of from 5 to 10 grains to ℥j, should be applied by the surgeon to the palpebral conjunctiva with a camel's-hair pencil, the lid being well everted, and this then should be thoroughly neutralised with salt water, the whole being finally washed off with plain water. The application is to be repeated in twenty-four hours, by which time the

slight loss of epithelium, the result of the superficial slough, will have been repaired. Immediately after such an application, cold sponging or iced compresses are useful, and grateful to the patient. The greatest care is required in the use of nitrate of silver in conjunctival affections for any prolonged period, lest it cause that brownish staining of the membrane, called Argyrosis (*ἄργυρος, silver*); thorough neutralisation and washing, as above recommended, being the best safeguards. I am opposed to the use even of weak solutions of nitrate of silver as eye-drops to be used at home by the patient, for staining is very apt to be caused in this way.

Should the surgeon be unable to see the patient daily, the following simple eye-drops are capable of effecting a rapid cure in most cases:— ℞ Acid Boracici, gr. v; Zinci Sulph. gr. ii; Tinct. Opii, ʒi; Aq. destill. ad ℥j; one drop in the eye morning and evening, or only once a day in mild cases. Solutions of alum (gr. iv to f. ℥j of water) and of tannic acid (gr. v to viij to f. ℥j of water) are often prescribed, but are not so effectual as the foregoing.

A weak boracic acid ointment, to be applied along the margins of the lids at bedtime, is to be ordered. It prevents the "gumminess" in the morning, which is not only unpleasant to the patient, but is also injurious, by fastening the eyelids together, and thus preventing free drainage of the secretion.

Follicular Conjunctivitis.—This is catarrhal conjunctivitis, to which is added the presence in the conjunctiva of small round pinkish bodies the size of a pin's head, which disappear completely as the process passes off, leaving the mucous membrane as healthy as they found it.

These little bodies are situated chiefly in the lower fornix of the conjunctiva, and may be discovered by eversion of the lower lid, when they will be seen arranged in rows parallel to the margin of the lid. Whether they are easily discovered or not depends on their size and number, and on the amount

of co-existing hyperæmia or chemosis of the conjunctiva. The structure of these bodies shows them to be lymph follicles.

Follicular conjunctivitis is a very tedious affection, lasting often for months. According to Sæmisch, it is more apt to give rise to marginal ulceration of the cornea than the simple catarrhal form; but I have not myself observed this. I agree with those who hold that the disease has nothing to do with granular ophthalmia, although some authors regard it as an early stage of the latter.

The Symptoms are much the same as those of catarrhal conjunctivitis. Frequently, there is a little or no injection of the bulbar conjunctiva, and the chief symptom is asthenopia —an inability to continue near work for any length of time — and much distress in artificial light. Boys and girls from five to fifteen years of age are those most liable to this affection.

Causes.—These are also much the same as in simple catarrhal conjunctivitis. The long-continued use either of atropine or of eserine is liable to bring on the disease.

Treatment.—The remedy I have found most useful in this troublesome affection is an ointment of sulphate of copper of from gr. ss. to gr. ij in ʒj of vaseline. The weaker ointments should be used at first, and later on the stronger ones, if it be found that the eye can bear them. The size of half a pea of the ointment is inserted into the conjunctival sac with a camel's-hair pencil once a day. Eye-drops of equal parts of tincture of opium and distilled water are of use in some cases; and the eye-douche should be recommended. Abundance of fresh air, with change from a damp climate or neighbourhood to a dry one, is of importance. If the use of a solution of atropine have induced the disease, it should be discontinued; and, if a mydriatic be still required, a solution of extract of belladonna (gr. viij ad ʒj) may be employed in its stead.

Spring Catarrh is the eye complication which accompanies

that troublesome affection known as "Hay Fever." It is not, strictly speaking, a catarrhal affection, for it is usually unattended by secretion, and the prefix "Spring" is misleading, as it is seen also in summer and autumn. The hay harvest is the most common period for it, owing probably to certain minute particles which then float in the air.

The bulbar conjunctiva is chiefly affected. It becomes injected, slightly œdematous, and, close around the cornea, somewhat elevated, with greyish swellings. The margin of the cornea itself is apt to become invaded with minute infiltrations.

Some individuals are liable to be attacked at each hay harvest. The chief symptoms are photophobia and lacrimation. The affection is unattended with danger to the eye.

The microscope shows (Uhthoff) that the conjunctival swelling is due to hypertrophy of the epithelial layer of the conjunctiva in this situation, combined with sub-epithelial infiltration with a substance which is, or is similar to, coagulated albumen. The deeper layers of the conjunctiva remain tolerably normal.

Treatment.—This is usually an excessively troublesome affection to cure. Dark glasses for protection from the light, weak astringent collyria (sulphate of zinc, acetate of lead), with cold sponging, or the douche, are useful; or, iodoform ointment (1 in 15), a little put into the eye once a day. Pagenstecher highly recommends massage twice daily in conjunction with strong precipitate ointment.

Granular Conjunctivitis, Granular Ophthalmia, or **Trachoma** (also called Egyptian Ophthalmia, and Military Ophthalmia).—In this disease, in addition to the usual appearances of simple conjunctivitis, there are developed greyish, or pinkish-grey, bodies, about the size of the head of a pin, situated in and close to the fornix conjunctivæ, chiefly of the upper lid, but also disseminated over other parts of the membrane. They do not form on the bulbar

conjunctiva. These bodies are the "granules" or "granulations," and, in the acute form of the disease, they somewhat resemble the follicles of follicular conjunctivitis, but are paler, not so apt to occur in rows, and are more isolated. Microscopically the granulations have no capsule, as have the follicles, but seem to grow from, or in, the stroma of the conjunctiva. In the acute form the granulations consist of lymph cells alone, but in the chronic form this is true of them only towards their surface, while at their bases they are formed chiefly of connective tissue. They are to be regarded as new growths in the conjunctiva.

According to some observers (Reid, Muttermilch [1]) who have examined various forms of conjunctivitis microscopically, the differences between them are only in the degree of the hypertrophy of the sub-epithelial adenoid tissue, which occurs in all. All stages of transition exist, they say, from small sub-epithelial collections of round cells, to characteristic trachoma, which, according to them, is not a distinct disease, but can be developed from other forms of conjunctivitis, under certain conditions of health and surroundings.

The disease comes under our notice in two forms: the acute, and the chronic. The latter may result from the former, but, more commonly, we find it as the primary condition, without any appreciable acute stage having gone before.

Causes.—Both forms are contagious, and, probably, the infection occurs only by transference of the secretion from one eye to the other by means of fingers, towels, handkerchiefs, etc. Hence, the more slovenly in their personal habits, and the more crowded in their dwellings, families, schools, or regiments, nations may be, the more likely is this disease to spread from one individual to another, when it once gains a foothold. A great deal, however, remains to be learned as

[1] *Annales d'Oculistique*, 1893, p. 41.

to the manner in which contagion takes place ; for instance, inoculation with discharge from an acute case may give rise only to catarrhal or purulent conjunctivitis, which may recover completely. Again, the infectiousness of chronic cases cannot be very great, for nurses and doctors rarely, if ever, get infected by their patients. Neither do we see granular patients infecting others in the hospitals in this country, where the disease is so prevalent. Were the infectiousness of the disease very great, even the precautions taken in a well-ordered hospital against contagion would hardly be sufficient to prevent such an occurrence occasionally.

It has been stated that the acute form is often epidemic in places where the hygienic conditions are bad ; but in this country I have never seen it as an epidemic, and sporadically not often, although the chronic form is extremely common in Ireland.

Amongst the better classes, here and elsewhere, the disease is very uncommon. High, dry, mountainous countries are almost free from this disease. So that, probably, the atmospheric conditions play some part in the etiology.

Some hold that the affection is dependent on constitutional disease, such as scrofula, tuberculosis, syphilis, etc. ; but I cannot endorse this view. No doubt many of these patients are anæmic and out of health, but this is due to the moping habits they contract, and the little open-air exercise they take, in consequence of their semi-blindness.

Acute Granular Ophthalmia.—As already stated, this is an affection rarely seen in this country. An attack commences with swelling of the upper lid, great injection of the whole of the bulbar and palpebral conjunctiva, and swelling of the papillæ, with development of the characteristic "granulations." There may be but little discharge ; but there is generally much lacrimation, with photophobia, and great pain in the brow and eye. Superficial marginal ulcers of the cornea may form.

The inflammation and papillary swelling increase for a week or so to such a degree, that the granulations are hidden from view; and then, taking on a blennorrhœic form, the process gradually subsides, until, in the course of two or three weeks longer, the blennorrhœa disappears, having brought about absorption of the granulations, and ultimately the mucous membrane is left in a healthy state.

If, however, in the blennorrhœic stage, the inflammation be excessive, the eye may run all the dangers of an attack of acute purulent conjunctivitis. Or if, on the other hand, the inflammation be very slight, it may not be sufficient to effect absorption of the granulations, and the process may run into the chronic form.

Egyptian ophthalmia, which is an acute form of granular ophthalmia, seems to be a combination of granular disease with purulent ophthalmia, as the gonococcus can always be found in the discharge.[1]

Treatment.—It is desirable to abstain from active measures in the commencement of the affection, owing to the tendency to natural cure which is often present, and, in particular, astringents and caustics should be avoided. At the utmost, an antiseptic lotion of boracic or salicylic acid, and cold applications for relief of the pain and heat are admissible. Dark protection-glasses are agreeable, and, wearing them, the patient should be encouraged to take open-air exercise. But, if it be evident that the inflammatory reaction is not active enough, poultices or warm fomentations should be employed to promote it. Once the blennorrhœic stage has been reached, great care is required to control it; and, if it threaten to exceed safe bounds, it must be restrained by means of suitable applications, such as acetate of lead, nitrate of silver, or sulphate of copper in solutions of medium strength; or, it may be necessary to use them in strong solutions; or to employ the solid mitigated nitrate of silver.

[1] Demetriades, *Annal d'Ocul.*, 1894, p. 19.

Chronic Granular Ophthalmia.—The first onset of this disease is often without inflammation, and is then unattended by any distressing symptoms, except that the eye may be more easily irritated by exposure to cold winds, foreign bodies, etc., or more easily wearied by reading and other near work. If such a case come under our notice, the conjunctiva will be found free from injection or swelling; but greyish-white semi-transparent granulations, of the size of a rape seed and less, will be seen disseminated over the conjunctival surface, and protruding from it. Gradually these granulations give rise to a more or less active vascular reaction, attended with swelling of the papillæ and purulent discharge—in short, blennorrhœa. The patients then begin to be more inconvenienced, owing to the discharge which obscures their vision, and to sensations of weight in the lids, and of foreign bodies in the eye; and this, consequently, is generally the earliest stage at which we see the disease. The enlarged papillæ sometimes grow to a great size, completely hiding the granulations. In this stage the granulations may become absorbed, and the disease undergo cure; but, more commonly, it makes further progress. Fresh granulations appear, while the old ones increase in size, until they often become confluent, leaving only here and there an island of vascular mucous membrane.

These chronic granulations consist of lymph cells towards their surface, but towards their bases are formed chiefly of connective tissue. Gradually the cellular elements are transformed into connective tissue, and, in this way, cicatricial degeneration of the conjunctiva is brought about at each spot where a granulation was seated.

As the disease advances, the submucous tissue becomes implicated in the connective tissue alterations, while the tarsus undergoes fatty degeneration, and becomes hypertrophied. The granulations disappear, having reduced the conjunctiva to a cicatrix. Contraction of the diseased con-

junctiva on the inner surface of the lid causes entropion, and distortion of the bulbs of the eyelashes, giving rise to irregular growth of the latter, with resulting trichiasis and distichiasis. These changes are represented in Fig. 56.

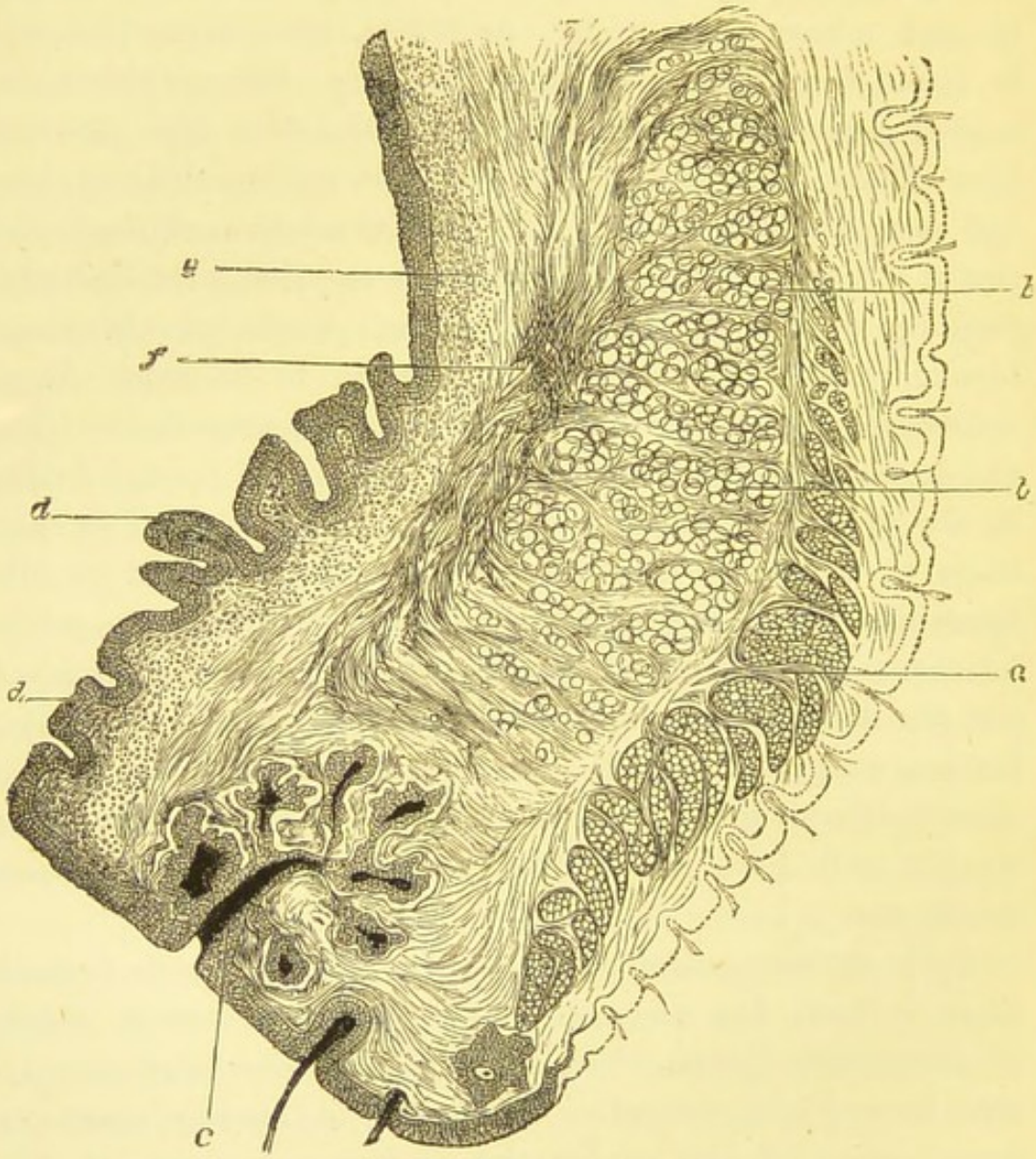


FIG. 56 (*Sæmisch*).

a, Muscle; *b b*, Tarsus having undergone fatty degeneration; *c*, Atrophied Meibomian Gland; *d d*, Hypertrophied Papilla; *e*, Cicatricial Tissue in the conjunctiva; *f*, Tarsus.

The great danger of granular ophthalmia lies in the complications which may attend it, or which follow in its wake. The former consist in pannus and ulcers of the

cornea, and severe purulent conjunctivitis; while the latter are the distortions of the lids and eyelashes just referred to.

Pannus (*Lat.*, a cloth rag) presents the appearance (Fig. 57) of a superficial vascularisation of the cornea, with more or less diffuse opacity, and often small infiltrations. It invariably commences in the upper portion of the cornea, extending generally over the upper half, and frequently remains confined to this region. But, in many cases, at a later stage, it extends to the whole surface of the cornea; and this latter occurrence often takes place almost suddenly; and the vascularisation and opacity sometimes become so intense, as to present quite a fleshy appearance completely hiding the corresponding part of the iris from view. Histologically, pannus consists of a new growth, which is extremely rich in cells, and which closely resembles the conjunctiva when occupied with confluent granulations. It is situated between the corneal epithelium and Bowman's layer, and is permeated by vessels derived from the conjunctival vessels. After a length of time Bowman's layer becomes destroyed in places, and then the cellular infiltration gains access to the true cornea, and gives rise to permanent changes in its transparency and curvature. In some bad cases of old-standing pannus the latter undergoes

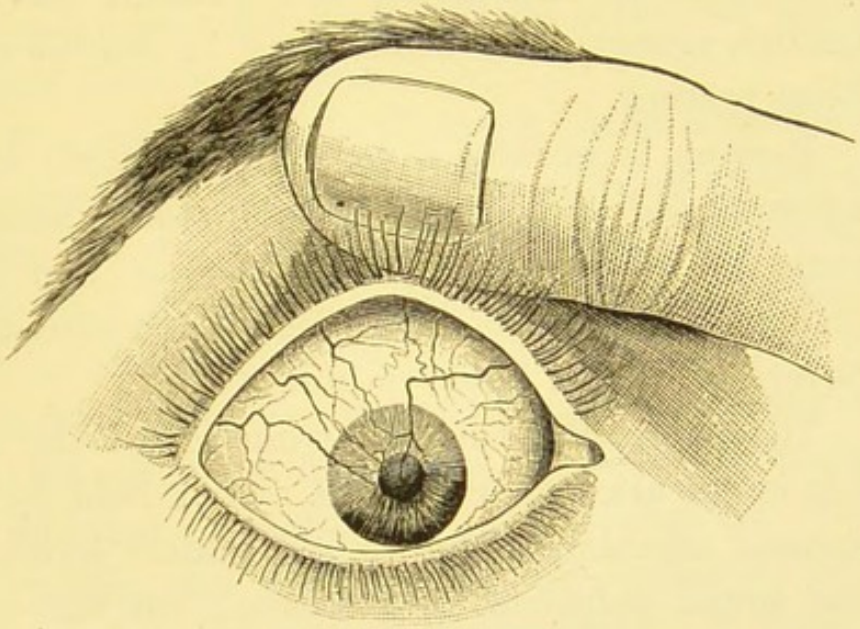


FIG. 57.

a connective-tissue change. It then becomes smooth on the surface, and the vessels almost disappear, so that the cornea is covered with a thin layer of connective tissue, which obstructs the passage of light, and is not capable of cure.

Another result of pannus, sometimes, is a bulging or staphylomatous condition of the cornea, the tissues of which have become so altered that they give way before the normal intraocular tension.

A pannus in which as yet there is no connective tissue alteration, and where there is no staphylomatous bulging, is capable of undergoing cure without leaving any opacity behind, except that which may be due to ulcers that have been present.

Pannus is usually a painless affection, but is sometimes accompanied by photophobia and ciliary neuralgia. It may come on at any stage of the disease, and causes defective vision, in proportion to the degree and extent of the opacity. Severe pannus is liable to induce iritis.

The connection between pannus and the condition of the lids is not altogether evident. It is held by many that this corneal affection is due to mechanical irritation, caused by the rough palpebral conjunctiva; but this view is obviously incorrect, for severe pannus is often seen with a comparatively smooth conjunctiva, while with a truly rough conjunctiva the cornea is frequently perfectly clear. But there can be little doubt that pannus is analogous to the granular disease in the conjunctiva. It is, in fact, the same disease modified by reason of the different tissue in which it is situated, this different tissue being itself a modification of the conjunctiva; and it would seem probable that the cornea becomes diseased by direct inoculation from the conjunctiva of the upper lid. Yet it is remarkable, that the bulbar conjunctiva, lying between the upper margin of the cornea and the fornix of the upper lid, never becomes diseased.

Prognosis.—At any period prior to cicatrisation of the

conjunctiva, an attack of purulent blennorrhœa is liable to come on. If not too severe, this may result in a cure by absorption of the granulations, and should not be checked. If, however, the attack be very severe, the eye runs dangers similar to those of an ordinary attack of purulent conjunctivitis. These dangers are less, the more complete and the more intense the pannus.

On the whole, if the disease come under treatment at an early period, it may be hoped that vision will be retained in a majority of cases, although a radical cure may be difficult, or impossible. These cases require to be under constant, or intermitting, treatment for long periods, often for years, and are extremely liable to relapses.

Treatment.—The aim of this is to bring about absorption of the granulations with the greatest possible despatch, in order to prevent the destruction of the mucous membrane to which they tend. No caustic application should be made with the object of directly destroying the granulations, for this can only be done at the expense of the mucous membrane around them. As already said, in cases of chronic granular ophthalmia in which a blennorrhœic attack comes on, when this passes off again, the granulations are found to have become much fewer, or to have quite disappeared. Following the hint nature thus gives us, we should endeavour by our treatment to produce a certain papillary reaction. For chronic cases, with little swelling of the papillæ (blennorrhœa), and with little or no cicatrisation, the best application is the solid sulphate of copper lightly applied to the conjunctiva, especially at its fornix; but, when there is considerable papillary swelling, I prefer a 10-grain solution of nitrate of silver, properly neutralised after its application with a solution of salt, or a light application of mitigated lapis, similarly neutralised. An interval of twenty-four hours at least should be allowed to elapse between each application, whether of sulphate of copper or nitrate of silver, and cold sponging

for fifteen minutes should be employed immediately after the application. A change of treatment will be occasionally required, even if the remedy first used answer well in the beginning, and one or other of the following can be adopted: —Pure carbolic acid liquefied has been used[1] with good result, but I have no experience of it. It is applied with a camel's-hair pencil, and the excess washed off with plain water. Liq. plumb. acetatis dil., never to be used except with everted lids, and washed off with plain water by the surgeon; and not even in this way if there be ulcers of the cornea, as the corneal tissue forming the floor of the ulcer is liable to become impregnated with a white deposit, probably the albuminate of lead, which is by no means easy to remove by operation subsequently. Tannin ointment:—Tannin gr. j, to vaseline ʒj, the size of half a pea, to be put into the eye once a day. Sulphate of copper ointment:—Same strength as the last, and to be used in the same way. Solution of alum:—Gr. x to ℥j of distilled water; one drop in the eye once a day. Where an active pannus is present, a drop of solution of atropine should be instilled into the eye once a day, as a precaution against iritis.

Some surgeons employ scarifications of the conjunctiva when it is much swollen and the papillæ too exuberant, but I have never adopted them, fearing the resulting cicatrices.

"Grattage," or scraping of the conjunctiva with a sharp spoon, with subsequent rubbing in of 1-500 corrosive sublimate solution, has recently been recommended.

Again, it has been proposed to excise, or abscise, the granulations, and this may, perhaps, be allowable if they are isolated, and protrude much over the surface of the conjunctiva.

Squeezing out the granulations between the thumb-nails used to be practised by the late Sir William Wilde, of Dublin,

[1] Recently again by E. Treacher Collins, *Roy. Lond. Ophthal. Hosp. Rep.*, vol. xi., p. 340.

and has recently again come into use. But the proceeding of "expression" is nowadays performed by means of an instrument instead of by the finger-nails. The best instrument for the purpose is Knapp's roller forceps[1] (Fig. 58). Two small grooved cylinders are inserted in the forked ends of a strong forceps, so that they roll over the surfaces of a body which may be grasped between them when the instrument is drawn upon. The retro-tarsal fold of the lower or upper lid is grasped as far back as possible between the cylinders, compressed and drawn upon, and in this way the granulation tissue is squeezed out without laceration of the conjunctiva. The instrument has to be reinserted, and a neighbouring part of the conjunctiva treated in the same way, and so on until the whole con-

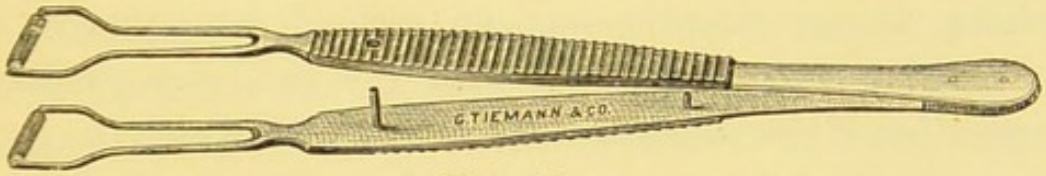


FIG. 58.

junctiva of each affected eyelid has been operated on. The four eyelids may be manipulated at one sitting, and the evacuation should be so complete that a repetition of the proceeding will not be required. Particular care should be taken to reach the part of the conjunctiva which is hidden under the commissures. If the tarsal portions are affected, one cylinder may be applied to the outer surface of the lid, and the instrument so drawn across the lid that the other cylinder presses the granulations out of the tarsal conjunctiva. As the operation is painful, and cocaine not of much avail in it, it is, as a rule, desirable that the patient should be under the influence of an anæsthetic. Some cases are immediately and permanently cured by this operation, while others, although greatly benefited, will still require a further

[1] *Trans. Amer. Ophthal. Soc.* for 1891, and *Archives of Ophthalm.* (English edition). 1893, p. 111.

routine treatment with local remedies. Expression is indicated only where trachomatous substance can be pressed out. My experience with this method leads me to regard it as a useful one for the acceleration of the cure of some cases of granular ophthalmia, before the cicatricial stage has come on.

Excision of the fornix conjunctivæ has been proposed by Schneller,[1] and largely practised by him and other surgeons. It is claimed for this method:—That it shortens the treatment of all forms of the disease; that, after it, existing corneal processes undergo rapid cure; that the granular disease in the palpebral conjunctiva, although not directly included in the operation, disappears quickly; that recurrences of the disease are rarer than by other plans of treatment; and that the resulting linear cicatrix has no serious consequence, and is as nothing when compared with the extensive cicatricial degeneration of the whole mucous membrance, which the operation is calculated to prevent. Supplemental treatment with the customary local applications is employed until the cure is obtained. I have myself but little experience of this method.

Infusion of Jequirity (*Abrus precatorius*, Paternoster Bean), long used in the Brazils, has been introduced to the notice of European surgeons by de Wecker. The infusion is made by macerating 154 grains of the decorticised jequirity seeds in 16 oz. of cold water (a 3 per cent. infusion) for twenty-four hours. Twice a day, for three days, the lids are everted, and the infusion thoroughly rubbed into the conjunctiva with a sponge or bit of lint. The result is a severe conjunctivitis of a somewhat croupous tendency (even the cornea being often hidden by the false membrane), accompanied by great swelling of the eyelids, much pain, and considerable constitutional disturbance, rapid pulse, and temperature of 100°, or more. In the course

[1] *Von Graefe's Archiv*, vol. xxx., No. 4, p. 131; and vol. xxxiii., No. 3, p. 113.

of eight or ten days the inflammation subsides, and the cornea in many cases will then be found to be free from pannus, or almost so, while complete cure of the granular ophthalmia itself is rarer. Iced compresses to the eyelids should be used during the inflammation. A fresh infusion (not more than seven days old) must be employed, in order to secure the best reaction. The majority of surgeons, amongst them myself, find the remedy harmless, if not always successful; but a good many cases are on record where violent diphtheritic conjunctivitis, followed by blennorrhœa of the conjunctiva, and by more or less extensive ulceration of the cornea, and even complete loss of the eye, were produced. I have, two or three times, seen a small superficial ulcer form on the lower third of the cornea without further injury. De Wecker regards the presence of a purulent discharge from the conjunctiva as a contra-indication for the remedy, which he finds is then liable to increase the intensity of the blennorrhœa in a dangerous degree. Cases where there is little or no papillary swelling, but nearly dry granulations with pannus, are the most suitable for its use, and I cannot recommend it too highly in these cases. It is marvellous to see the rapid and beautiful cures of the severest pannus by this remedy, in properly selected cases. But the presence of well-marked pannus of the cornea without ulceration is, I think, the only thing that can render the employment of jequirity justifiable, and, in addition to this, the conjunctiva should be free from blennorrhœa.

The occurrence of acute dacryocystitis sometimes forms an unpleasant complication of the jequirity treatment, even in cases in which the sac was previously quite normal, but I have never myself seen it to occur.

After the subsidence of the jequirity inflammation, some of the local remedies above referred to should be regularly applied, for the purpose of completing the cure of the conjunctival condition.

Besides local remedies, it is of great importance that the

hygienic surroundings of patients suffering from granular ophthalmia be seen to, and that they be obliged to spend a considerable time daily in the open air.

If the upper lid be tightly pressed on the globe, as it sometimes is, the pressure varying in different individuals, an impediment is offered to the cure by any method, and pannus is promoted. It is then necessary to relieve the pressure by a canthoplastic operation. (See chap. vi.)

Peritomy.—This procedure is adopted for the cure of pannus, by destruction of the vessels which supply it, and is as follows:—About 5 mm. from the margin of the cornea an incision is made in the conjunctiva with scissors, and carried at this distance all the way round the cornea. This ring of conjunctival tissue is then separated up from the sclerotic, and cut off at the corneal margin; and the underlying connective tissue is dissected off the corresponding portion of the sclerotic, which is thus laid quite bare. The proceeding is not always satisfactory, and of late years I have practised it but little.

Lymphoma of the Conjunctiva.—Under this heading cases have been recently described[1] which present the appearances, at first sight, of acute granular ophthalmia; but the "granulations," which are enormous in size, attack both lids, and are associated with enlarged lymphomatous masses in the neck, which do not lead to ulceration or scarring. The conjunctival affection runs a rapid and favourable course, without any cicatricial contraction.

Acute Blennorrhœa of the Conjunctiva, or **Purulent Ophthalmia.**—We most commonly find this very dangerous affection either as gonorrhœal ophthalmia, or as blennorrhœa neonatorum.

Etiology.—In the former, the etiological moment is the introduction of some of the specific discharge from the urethra,

[1] Goldzieher, *Centralblatt. f. Augenheilk.*, 1893, p. 112.

or vagina, into the conjunctival sac; while, in the latter, the infection is believed to take place either during, or just after, the passage of the head through the vagina, by an abnormal secretion from the latter finding its way into the infant's eyes. A few instances have been observed of infants born with the disease. Inoculation may also occur a few days after birth by pus conveyed by the fingers of the mother or nurse, or by towels, etc., used for washing the child's face. It is never due to exposure to strong light or to cold, as is popularly supposed.

The more severe cases of blennorrhœa neonatorum are caused by a vaginal discharge, which is always gonorrhœal. Neisser, who first observed the presence of a peculiar micrococcus in the gonorrhœal discharge, also found the gonococcus in the pus from the conjunctiva in cases of gonorrhœal ophthalmia, and the same micrococcus has been found in the conjunctival discharge in cases of blennorrhœa neonatorum. But the slight cases of the latter affection, which amount to little more than a catarrh of the conjunctiva, may be caused by a vaginal discharge which is not of the specific gonorrhœal nature.

If the infection take place during, or immediately after, birth, the disease appears from the second to the fifth day, according to the virulence of the secretion. If the inflammation come on later than the fifth day, it may be concluded that the infection was produced by the vaginal discharge being introduced into the eye by the fingers of the mother or nurse, etc. Acute conjunctival blennorrhœa also comes about without any assignable cause; but, in all such cases, it may be regarded as certain, that the introduction of some infective pus into the eye has taken place, although without the knowledge of the patient.

Symptoms and Progress.—In mild cases the bulbar conjunctiva may be but little, or not at all, affected; the palpebral conjunctiva alone becoming velvety and discharging a small

amount of pus, while there may be no swelling or œdema of the eyelids. Such mild cases are not uncommon in ophthalmia neonatorum. In severe cases of blennorrhœa of the conjunctiva, there is, soon after the onset, serous infiltration of the palpebral mucous membrane—which, consequently, becomes tense and shiny—serous chemosis (*χαίνω*, to gape open[1]) of the bulbar conjunctiva, serous discharge, dusky redness and swelling of the eyelids—which makes it difficult to evert them—pain in the eyelids, often of a shooting kind, burning sensations in the eye, and photophobia. This first stage lasts from forty-eight hours to four or five days.

Then begins the second stage, in which, owing to swelling of the papillæ, the palpebral conjunctiva becomes less shiny and more velvety, while the discharge alters from serous to the characteristic purulent form, the chemosis, however, remaining unaltered, or becoming more firm and fleshy. The swelling of the lids continues, the upper lid often becoming pendulous and hanging down over the under lid; while, at the same time, it becomes less tense, and more easily everted. Gradually the chemosis and swelling of the conjunctiva and eyelids subside, and the discharge lessens, the mucous membrane finally being left in a normal state, unless in a small percentage of cases in which chronic blennorrhœa remains. A moderately severe attack of conjunctival blennorrhœa lasts from four to six weeks.

Complications with corneal affections form the great source of danger from this affection. They occur chiefly in four different forms: 1. Small epithelial losses of substance on any part of the cornea. If these occur at the height of the inflammation, they are apt to go on to form deep perforating ulcers. 2. The whole cornea becomes opaque (diffusely infiltrated), and towards its centre some greyish spots form, which are interstitial abscesses or purulent infiltrations. 3. The infiltration may

[1] Probably from the appearance produced when the conjunctiva in this condition is much elevated round the margin of the cornea.

form at the margin of the cornea, and extend a considerable distance around its circumference, giving rise to a marginal ring ulcer, and, later on, to sloughing of the whole cornea. 4. A clean-cut ulcer may form at the margin of the cornea without any purulent infiltration of the corneal tissue, and may also extend a long way round the cornea. Such ulcers are particularly apt to occur where there is much chemosis which overlaps the margin of the cornea; and, being hidden in this way, these ulcers are easily overlooked. The chemosis should be pushed aside with a probe, and these peculiar ulcers looked for. They are very liable to perforate.

All the foregoing forms of corneal complication occur both in ophthalmia neonatorum and in gonorrhœal ophthalmia. They may appear at any period of the affection, but the earlier they occur, the more likely are they to result seriously.

The danger of these ulcers consists in the perforation of the cornea they are apt to produce, of which more later on.

The severer the case, especially the more the bulbar conjunctiva is involved in the process, the more likely is it that corneal complications will arise. For the corneal process is to be regarded as the result of infection by the conjunctival secretion; and this infection is all the more apt to occur, where the nutrition of the cornea is impeded by a dense chemotic swelling of the bulbar conjunctiva. Severe chemosis is less common in the blennorrhœa of the new-born than in gonorrhœal ophthalmia, and this is the chief reason for the fact that the latter is the more dangerous affection of the two.

Treatment.—The prophylaxis of purulent ophthalmia must here first engage our attention.

The prophylaxis of blennorrhœa neonatorum is a most important matter, and should form part of the routine of lying-in practice. Careful disinfection of the vagina before and during birth, and the most minute care in cleansing the face and eyes of the infant immediately after birth with a non-irritating

disinfectant (*e.g.* a solution of corrosive sublimate 1 in 5,000), are to be recommended. The method of the late Dr. Credé has found very general acceptance, and is a good one. It is as follows:—When, after division of the umbilical cord, the child is in the bath, the eyes are carefully washed with water from a separate vessel, the lids being scrupulously freed, by means of absorbent wool, of all blood, slime, or smeary substance; and then, before the child is dressed, a few drops of a 2 per cent. solution of nitrate of silver are instilled into the eye. Many obstetricians employ this method now, in a routine manner, in their lying-in hospitals, for all the infants, whether or not it be suspected that there is danger of infection; and by its aid Credé reduced the percentage of his cases of ophthalmia neonatorum from 8 or 9 per cent. to 0·5 per cent.

The action of the nitrate of silver solution depends, probably, upon the destruction of the superficial layers of the conjunctival epithelium, and of the gonococci contained in them. Other antiseptic applications which have been tried do not act as well, for they do not destroy the superficial epithelium.

In all cases of gonorrhœa it is the duty of the surgeon to explain to his patients what is the danger of their carrying any of the urethral discharge to their eyes; and to charge them to exercise punctilious cleanliness as regards their hands and finger-nails, and care in the use of towels, handkerchiefs, etc.

In respect of Local Treatment when the disease has once broken out. In the commencement of the affection, the only local applications admissible are antiseptic lotions (Boric Acid; Corrosive Sublimate) and iced compresses, or Leiter's tubes. With the former the conjunctival sac should be freely washed or irrigated, not syringed, out. In syringing out the conjunctival sac, a morsel of the corneal epithelium may be removed, and through this the cornea

become infected, and therefore this method is objectionable. The iced compresses, or Leiter's tubes, should be kept to the eye for an hour at a time, with a pause of an hour, and so on; or even continuously. In this and in the next stage the chemosis should be freely, and daily, incised with scissors. If the swelling of the lids be great, the external canthus should be divided with a scalpel from without, leaving the conjunctiva uninjured, in order to reduce the tension of the eyelids on the globe, and, by bleeding from the small vessels, to deplete the conjunctiva. Depletion alone can be obtained by leeching at the external canthus, and, in many cases, is of great benefit at the very commencement. If, in adults, the chemosis, palpebral swelling, and rapidity of the onset indicate that the inflammation is severe, it is well, in my opinion, to place the patient quickly under the influence of mercury by means of inunctions, or small doses of calomel, as, by so doing, the chemosis is often rapidly brought down, and one source of danger to the cornea removed.

In the second stage, *i.e.* when the conjunctiva has become velvety, and the discharge purulent, caustic applications are the most trustworthy, and in this respect iodoform and other lauded means cannot compete with them. The application employed may be a solution of nitrate of silver of 15 to 20 grains in ℥j of water, which should be applied by the surgeon to the conjunctiva of the everted lids, and then neutralised with a solution of common salt, as described when discussing the treatment of simple catarrhal conjunctivitis. Or, the solid mitigated nitrate of silver (one part nitrate of silver, two parts nitrate of potash) may be used, the first application being mild, in order to test its effect, while careful neutralisation with salt water and subsequent washing with fresh water are most important.

The immediate effect of a caustic application to the conjunctiva is the production of a more or less deep slough, under

which a serous infiltration takes place. This latter increases, and finally throws off the slough, and then the epithelium begins to be re-formed. From the time the slough separates, until the epithelium has been regenerated, a diminution in the secretion may be noted; but the discharge again increases, as soon as the regenerative period is ended, and this now is the moment for a new application of the caustic. From one caustic application of ordinary severity until the end of the regenerative period about twenty-four hours usually elapse. Immediately after a caustic application iced compresses should be used for thirty minutes or longer. Between the caustic applications the pus should be frequently washed away from the eyelids, and from between the eyelids, with a 4 per cent. solution of boric acid, or with a 1 in 5,000 solution of corrosive sublimate, and boric acid ointment should be smeared along the palpebral margins to prevent them from adhering, and thus retaining the pus.

No corneal complication contra-indicates the active treatment of the conjunctiva by the method just described. Iodoform, finely pulverised, has been much praised as a local application in the second stage of acute blennorrhœa of the conjunctiva. It is to be dusted freely on the conjunctiva once or twice a day. For my part I should trust to it in mild cases only.

When but one eye is affected, it is important to protect its fellow from infection by means of a hermetic bandage. This may be made by applying to the eye a piece of lint covered with boracic acid ointment, and over this a pad of borated cotton wool. Across this, from forehead to cheek and from nose to temporal region, are laid strips of lint soaked in collodion in layers over each other; or, a piece of tissue gutta-percha may take the place of the lint and collodion, its margins being fastened to the skin by collodion. The shields invented by Maurel and by Buller are very serviceable for this purpose.

Treatment of Corneal Complications.—Many surgeons, I understand, use solution of the sulphate of eserine (gr. ij ad aq. f. ℥j) dropped into the eye, as soon as any corneal complication arises, and as long as it continues; on the ground that this drug is believed to have the effect of reducing the intra-ocular tension (a circumstance to be desired in these instances), and also to act as an antiseptic. Its power to reduce the normal intra-ocular tension is not great, and its antiseptic action, if it exist, must be very insignificant, while, in my opinion, it has a decided tendency to promote iritis in these cases, where the iris is so liable to become inflamed secondarily to the corneal process. I therefore do not recommend its use in these cases. I employ atropine here with the object of diminishing the tendency to iritis. Only if a marginal ulcer should perforate, with prolapse, or danger of prolapse, into the opening, is eserine indicated, and then simply for the purpose of drawing the iris out of, or away from, the perforation, by the contraction of its sphincter.

On the first appearance of an ulcer or infiltration of the cornea, besides the use of atropine, nothing can be done further than the steady continuance of the conjunctival treatment, no remission or relaxation of which is indicated, or, indeed, admissible. Greater care is now required in everting the lids, lest pressure on the globe might cause rupture of the ulcer; and it should be remembered that, when a case of acute blennorrhœa first presents itself, the surgeon, not knowing the condition of the cornea, must use the utmost caution in making his examination, and yet must never fail to get a view of the cornea for the purposes both of prognosis and of treatment. At each visit the cornea must be examined, and it may be found that, as the conjunctival process subsides, any existing corneal affection also progresses towards cure, infiltrations becoming absorbed and ulcers filled up. But, even though the conjunctiva be improving, and still more so if it be not, the corneal process may progress, the infiltration becoming an

ulcer, and the ulcer becoming gradually deeper, until, finally, it perforates.

Should a corneal ulcer become deep, and seem to threaten to perforate, paracentesis of the floor of the ulcer must be resorted to without delay. By thus forestalling nature, a short linear opening is substituted for the circular loss of substance, which would have resulted in the ordinary course of events. Through this small linear opening no prolapse of the iris, or else a relatively small one, takes place; and, consequently, the ultimate state of the eye is usually a better one than it would otherwise be. The reduction of the intra-ocular tension after the paracentesis promotes healing of the ulcer. It is often desirable to evacuate the aqueous humour by opening the little incision in the floor of the ulcer with a blunt probe, on each of the two days after the operation.

If an ulcer perforate spontaneously, the aqueous humour is evacuated, and, unless the ulcer be opposite the pupil and at the same time small in size, the iris must come to be applied to the loss of substance. Should the latter be very small, the iris will simply be stretched over it and pass but little into its lumen, and, when healing takes place, will be caught in the cicatrix, which is but slightly, or not at all, raised over the surface of the cornea, and the resulting condition is called Anterior Synechia.

If the perforation be larger, a true prolapse of a portion of the iris into the lumen of the ulcer takes place. This prolapse may either act as a plug, filling up the loss of substance and keeping back the contents of the globe, but not protruding over the level of the cornea; or, it may bulge out over the corneal surface as a black globular swelling, and may then play the part of a distensor of the opening, causing fresh infiltration of its margins. In either case cicatrisation will eventually occur; and if the scar be fairly flat it is called an Adherent Leucoma, but if it be bulged out the term Partial Staphyloma of the Cornea is used.

If the perforation be very large, involving the greater part of the cornea with prolapse of the whole iris and closure of the pupil by exudation, the result is a Total Staphyloma of the Cornea. The lens may lie in this staphyloma, or it may retain its normal position, but become shrunken.

The question of the treatment of a recent prolapse of the iris in cases of blennorrhœic conjunctivitis is an important one. It has been, and is still largely, the practice to abscise small iris-protrusions down to a level with the cornea; or, if large, to cut a small bit off their summits with the object of obtaining flat cicatrices. Horner[1] pointed out that, in cases of blennorrhœa, this proceeding opens a way for purulent infection of the deep parts of the eye, and that serious consequences are not rare. He confined interference with the iris in these eyes to incision of the prolapse, when it seems to be acting as a distensor of the opening, causing fresh infiltration of the cornea. Under other circumstances he restricted his treatment of the prolapse to the instillation of eserine, which has a marked effect in diminishing the size of the protrusion.

It may occur that, on the surgeon's visit to a case of blennorrhœa of the conjunctiva, he will find the margins of the eyelids gummed together by sero-purulent secretion, while the eyelids are bulged out by the pent-up fluid behind them. The attempt to open the eye should then be very cautiously made, lest some of the retained pus spurt into the surgeon's eye. The surgeon should also be most careful to thoroughly wash and disinfect his hands and nails at the conclusion of his visit.

In cases of blennorrhœa neonatorum, when the ulcer has been small, on perforation taking place, the lens, or rather its anterior capsule, comes to be applied to the posterior aspect of the cornea. The pupillary area is soon filled with fibrinous secretion. The opening in the cornea ultimately becoming

[1] Gerhardt's *Handbuch der Kinderkrankheiten*, Bd. V., Abth. 2, p. 268.

closed, the iris and lens are pushed back into their places by the aqueous humour, which has again collected. Adherent to the anterior capsule on the spot which lay against the cornea is a morsel of fibrine, which gradually becomes absorbed by the aqueous humour. In the meantime, changes have been produced by this exudation on the corresponding intra-capsular cells, which result in a small, permanent, central opacity at that place, where there is also a slight elevation of pyramidal shape over the level of the capsular surface. This condition is called central capsular cataract, or pyramidal cataract, and rarely results from corneal perforation in adults.

In cases of blennorrhœa neonatorum an inflammatory swelling of the joints, so-called gonorrhœal arthritis, is very occasionally seen. Deutschmann[1] found the gonococcus in the fluid removed from the joints in two such cases, while other observers found in their cases only the usual pyogenic cocci.

Croupous Conjunctivitis.—This is a disease of early childhood, and is not common. The palpebral conjunctiva is a good deal swollen, and is covered with a false membrane, that may be peeled off, leaving a mucous surface underneath, which bleeds little or not at all. The disease is not a severe one, and does not cause secondary corneal affections, unless when the bulbar conjunctiva, as it very rarely does, participates in the attack. It must not be mistaken for diphtheritic conjunctivitis, from which it is readily distinguished by the ease with which in it the false membrane can be removed, and by the vascular condition of the underlying mucous membrane.

This is usually regarded as nothing more than a severe form of catarrhal conjunctivitis, in which the secretion happens to be rich in fibrine, and hence possessed of a marked tendency to coagulate on the surface of the conjunctiva. But the presence of virulent diphtheria bacilli has been

[1] *Arch. für Ophthal.*, xxxvi. 1, p. 109.

demonstrated in a case of apparent croupous conjunctivitis, which ran a favourable course.[1]

Some cases of membranous conjunctivitis become chronic, lasting for months.[2]

Causes.—Contagion, Epidemic.

Treatment.—Iced compresses, or Leiter's tubes, to the eyelids during the croupous stage, with antiseptic cleansing of the conjunctival sac (Sol. Hydrarg. Perchlor. 1 in 5,000, or Sol. Acid Borac. 4 per cent.). No caustic should be used in this stage, as it is apt to produce corneal changes. Sulphate of quinine sprinkled on the conjunctiva is praised by some surgeons as a useful application at this period. When the false membrane ceases to be formed, a slight blennorrhœa comes on; and this is to be treated with nitrate of silver applications in the usual way.

Diphtheritic Conjunctivitis.—There is no more serious ocular disease than this, for it may destroy the eye in twenty-four hours; while in severe cases treatment is almost powerless. Fortunately, it is almost unknown in these countries, while in Berlin it used to be so frequent that von Graefe set apart two wards for it in his hospital, which were under my care as his assistant. It is now a much less common disease there, owing probably to the improved hygiene of the city.

The subjective symptoms of its initial stage are similar, although severer, especially in the matter of pain, to those of blennorrhœic conjunctivitis. The objective symptoms differ from those of blennorrhœa, in that the lids are excessively stiff, owing to plastic infiltration of the subepithelial and deeper layers of the conjunctiva, while the surface of the mucous membrane is smooth, and of a greyish or pale buff colour. If an attempt be made to peel off some of the superficial exudation, the surface underneath will be found of the same grey

[1] Uhthoff, *Berlin. Klin. Wochenschr.* 1893, No. 11.

[2] *Trans. Ophthal. Soc. Un. Kingd.*, vol. xiii., p. 26.

colour, not red and vascular as in croupous conjunctivitis. This stage of infiltration lasts from six to ten days, and constitutes the period of greatest peril to the eye; for, while it lasts, the nutrition of the cornea must suffer, and sloughing of that organ is extremely apt to take place. Towards the close of the first stage the fibrinous infiltration is eliminated from the eyelids, and the conjunctiva gradually assumes a red and succulent appearance, and at the same time a purulent discharge is established. This constitutes the second or blennorrhœic stage. A third stage is formed by cicatricial alterations in the mucous membrane, which often lead to symblepharon, or to xerophthalmos; so that, even if the eye escape corneal dangers in the first and second stages, others almost as serious may await it in the final stage.

Corneal complications are most likely to occur in the first stage, and are then also most likely to prove destructive to the eye. The earlier they appear, the more dangerous are they. If the blennorrhœic stage come on before corneal complications appear, or even before an ulcer contracted in the first stage has advanced far, they are more easily managed.

Causes.—It is difficult to assign a cause for this disease, which chiefly attacks children. It is frequently epidemic, is extremely infectious, and, although similar in its nature, is rarely, if ever, found in connection with an attack of diphtheritis of the fauces.

Treatment.—In the first stage, frequent warm fomentations, with antiseptic cleansing, are the only local measures admissible. No caustic or astringent application should be used. Internally, the patient should be treated with iron and quinine, and generous diet. In the second, or blennorrhœic stage, careful caustic applications are to be used. Corneal ulcers must be dealt with whenever they arise, in the same way as though the case were one of blennorrhæic conjunctivitis. When the purulent discharge ceases, solutions of

soda, milk, or glycerine may be prescribed as lotions for the conjunctiva, to arrest, if possible, the xerophthalmos.

Conjunctival Complication of Smallpox.—Of this I have, fortunately, too little experience to enable me to speak authoritatively. The following embodies the views of the late Professor Horner,[1] who studied the subject during an epidemic in 1871. A good deal of uncertainty prevailed previously, for the initial stages of the eye affection were not carefully observed by physicians, owing to the swelling of the eyelids, while the ophthalmologist saw only the results of the process in the period of convalescence.

Smallpox pustules on the cornea are, Horner believed, extremely rare; indeed, he saw but one such case. The most frequent, and most serious, mode of attack consists in a greyish-yellow infiltration in the conjunctiva close to the lower margin of the cornea, not extending to the fornix conjunctivæ, nor far along the inner or outer margin of the cornea. It occurs in the eruptive stage, and is to be regarded clinically as a variola pustule. This infiltration or pustule gives rise to a corneal affection, as does a solitary marginal phlyctenula, either in the form of a marginal ulcer, or as a deep purulent infiltration, ulcerating, perforating, leading to staphyloma, purulent irido-chorioiditis and panophthalmitis; results which are often first observed long after the primary conjunctival affection has disappeared.

Horner believed that the germ of the conjunctival infiltration makes its way between the eyelids, and that the constancy of the position of the infiltration, below the cornea, is accounted for by this theory; that part of the conjunctiva, with closed eyelids and eyeball consequently rotated upwards, being the most exposed to particles entering.

Treatment.—On this ground he recommended the prophylactic use of boracic acid ointment on lint applied over the eye-

[1] *Loc. cit.*, p. 297.

lids. If a conjunctival pustule have already formed, without any, or only commencing, corneal affection, he would destroy the pustule with fresh chlorine water, or with mitigated lapis carefully neutralised. Corneal complications are treated as in blennorrhœa of the conjunctiva or diphtheritis.

The frequency with which the eyes become affected varies in different epidemics.

As true post-variolous eye-affections, Horner recognised diffuse keratitis, iritis, and irido-cyclitis, with opacities in the vitreous humour, and glaucoma. In the hæmorrhagic form of the disease, hæmorrhages in the conjunctiva and retina; and, where pyæmic poisoning comes on, septic affections of the chorioid and of the retina takes place.

Amyloid Degeneration.—This rare disease attacks chiefly the palpebral conjunctiva, but is also seen in the bulbar portion. It causes great tumefaction of the affected lid, without any inflammatory symptoms. The eyelid can be but partially elevated, and is often so stiff and hard that it can be everted only with difficulty. The conjunctiva has the appearance of white wax. The disease ultimately extends to the tarsus, but is a strictly localised process, and not associated with amyloid disease in any other part of the system. It sometimes seems to be developed from granular ophthalmia, but occurs also as a primary disease. The positive diagnosis can be made by submitting a small portion of the diseased conjunctiva to the iodine test.

Hyaline Degeneration of the conjunctiva has also been observed. It cannot clinically be distinguished from Amyloid Degeneration, and is really an early state of the latter condition.

Treatment consists in the removal of the diseased parts, by the knife and scraping, so far as may be possible.

Tubercular Disease of the Conjunctiva.—This is an extremely rare disease. It commences in the palpebral conjunctiva of the upper lid usually, very rarely in the bulbar

conjunctiva, as small round yellowish-grey nodules, which soon ulcerate. The margins of these ulcers are well defined, and their floors of a yellowish lardaceous appearance, or covered with greyish-red granulations. The surrounding conjunctiva is swollen, and if the palpebral conjunctiva be much involved, the lid becomes enlarged in every dimension, and the ulcerative process may soon destroy part of the lid. It may also extend to the bulbar conjunctiva, and the cornea may become covered with pannus. The pre-auricular and submaxillary glands usually become enlarged. The positive diagnosis of the nature of the disease should be made by an examination of portions of the floor of the ulcer for the characteristic tubercle bacillus, which will distinguish this from secondary syphilitic ulceration of the conjunctiva, between which and the tubercular ulceration there is sometimes a resemblance. Tubercular conjunctival disease is usually unattended by pain, or there is only a slight burning sensation ; but, again, when the ulceration is extensive, severe pain may set in.

This is a very chronic disease, its progress sometimes extending over many years, and it is rarely met with except in youth. Some of those whose eyes are attacked are already the subjects of tuberculosis in other organs, but very many of them are perfectly healthy in that respect. In fact, we have reason to believe (Valude, Leber) that tuberculosis of the conjunctiva is much more often a primary disease, the result of an ectogenic infection, even in cases where already tuberculosis exists elsewhere, than of infection occurring through the blood. Tubercle bacilli introduced into the normal conjunctival sac have, it is true, been found to be harmless, for the intact epithelium offers an insuperable obstacle to their entrance into the tissue. But a superficial loss of substance of the conjunctiva is sufficient to allow of its inoculation with the bacilli, and then the disease becomes established. The frequent lodgment of foreign bodies under

the upper lid explains why this is the most common place for the disease to begin in. But, although conjunctival tubercular disease is not often secondary to tubercular disease in other parts of the system, yet it is itself liable to be the starting-point of general tuberculosis.

Treatment.—The fact last mentioned makes it most important, in cases of primary tubercular disease of the conjunctiva, to thoroughly eradicate the diseased focus, so as to avert an infection of other organs, and this can often be effected. If the ulcers be not already too extensive, they must be scraped, and the actual cautery freely applied to them; and, where the disease has already spread to the cornea, sclerotic, iris, or chorioid, enucleation of the eyeball is instantly called for.

Lupus of the conjunctiva usually occurs as an extension of the disease from the surrounding skin, or rarely from the lacrimal sac, as in a case of Dr. Werner's where the disease extended from the mucous membrane of the nose through both lacrimal sacs, to the inferior palpebral conjunctiva. It is seen as a patch or patches of ulceration, covered with small dark-red protuberances or granulations, chiefly on the palpebral conjunctiva, which bleed easily on being touched.

Like lupus of the skin, these ulcerations undergo spontaneous healing and cicatrisation in one place (unlike tubercular ulceration in that respect), while they are still creeping over the surface in another direction. But we now know that lupus, wherever it occurs, is really a tubercular disease, and that the two forms differ only in their clinical aspect.

The *Treatment* is scraping with a sharp spoon, and the application of the actual cautery.

Pemphigus of the Conjunctiva.—This is another rare disease. It has been seen in connection with pemphigus vulgaris of other parts of the body, but it also occurs as an independent disease. It is attended by attacks of much pain, photophobia, and lacrimation; and the conjunctiva, at each

place where subconjunctival exudation of serum has been situated, undergoes degeneration and cicatricial contraction. Such attacks succeed each other at shorter or longer intervals, for weeks, months, or years; until, finally, the entire conjunctiva of each eye may have become destroyed, and the eyelids are adherent to the eyeball. The cornea gradually becomes completely opaque, or, having ulcerated, becomes staphylomatous. In the course of the disease the eyelashes are apt to become turned in on the eyeball, or even entropion may form; and these conditions aggravate the suffering of the patient.

The foregoing is a description of a severe case. In less severe cases the conjunctiva may not be completely destroyed, and the cornea may not be affected.

The formation of a true bulla hardly ever occurs, for the conjunctival epithelium is so delicate, that it cannot be disturbed in this way by the serous exudation beneath it, but rather breaks down at once. Consequently, the conjunctival surface is found, in these cases, to be covered by what looks like a membranous deposit, upon removal of which a raw surface is exposed; and these appearances have led to the mistaken diagnoses of croupous, and of diphtheritic, conjunctivitis.

Treatment is helpless in respect of arresting the progress of the disease, or of restoring sight when lost in consequence of it. The most one can do is to relieve the distressing symptoms by emollients to the conjunctiva, and by the use of closely fitting goggles, to protect from wind, dust, and sun. Internally, arsenic is indicated.

Xerosis (*ξηρὸς, dry*), or **Xerophthalmos**, is a dry lustreless condition of the conjunctiva, associated, in the severer forms, with shrinking of the membrane. There are two forms of the affection—the parenchymatous, and the epithelial.

In Parenchymatous Xerophthalmos there is a more or less extensive cicatricial degeneration of the conjunctiva, depen-

dent upon changes in its deeper layers, while its surface and that of the cornea become dry, and the latter becomes opaque, and the eye consequently sightless. The conjunctiva shrinks so completely, in many of these cases, that both lids are found adherent in their whole extent to the eyeball, which is exposed merely at the palpebral fissure, where the opaque and lustreless cornea is to be seen. From what remains of the conjunctiva, scales, composed of dry epithelium, fat, etc., peel away. The motions of the eyeball are restricted, in proportion to the extent of the conjunctival degeneration. There is no cure for this condition.

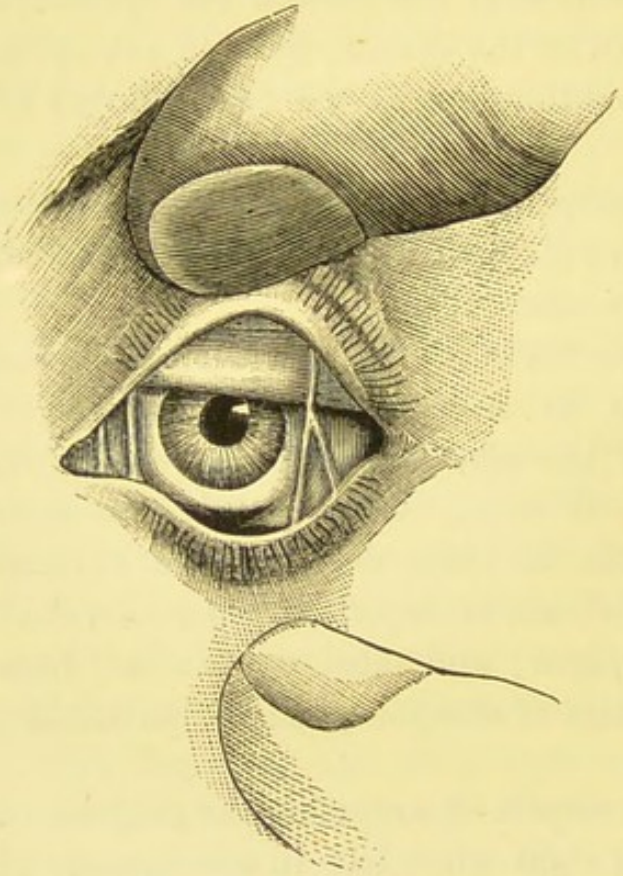


FIG. 59.

Fig. 59 represents a case of xerophthalmos, the result of pemphigus, which was under my care in the National Eye and Ear Infirmary. Here the eyelids were not wholly adherent to the eyeball, and the cornea remained clear.

The Causes of parenchymatous xerosis of the conjunctiva are :—Granular ophthalmia, diphtheritic ophthalmia, pemphigus, and the condition is said to be very occasionally seen as a primary disease, described as essential shrinking of the conjunctiva. Many observers altogether deny the existence of the primary affection, and maintain that the cases described as of that nature are merely the result of pemphigus, and I am inclined to agree with this view.

Treatment.—As cure is impossible in this form of xeroph-

thalmos, the only indication is to afford relief, so far as it can be done, from the distressing sensations of dryness of the eyes, which are complained of. The best applications are milk, glycerine, olive oil, and weak alkaline solutions, and the eyes should be protected from all irritating influences by protection goggles.

Epithelial Xerosis of the conjunctiva is confined to the epithelium of that part of the conjunctiva which covers the exposed portion of the sclerotic in the palpebral opening. It there becomes dry and dull and covered with small white spots ; while the whole bulbar conjunctiva is loose, and easily thrown into folds by motions of the eyeball, and there may be a good deal of secretion. This form of xerophthalmos often occurs in epidemics, but also sporadically, accompanied, oddly enough, by night-blindness (the light-sense unimpaired) and contraction of the field of vision. The combined condition has been noticed, chiefly in persons of debilitated constitution, who have been exposed to strong glares of light, and is said to have appeared in epidemics, under these conditions, in foreign prisons and barracks.

Treatment by rest, protection from glare of light, nutritious diet, and tonics, invariably restores the eyes to their normal functions.

Again, epithelial xerosis occurs in very young children in connection with a destructive ulceration of the cornea (see Infantile Ulceration of the Cornea with Xerosis of the Conjunctiva, chap. viii.).

Pterygium (*πτέρυξ, a wing*).—This is a vascularised thickening of the conjunctiva, triangular in shape, situated most usually to the inside of the cornea, sometimes to its outer side, and rarely either above or below it. The apex of the triangle, the "head" of the pterygium, is on the cornea ; and its base, the "body," at the semi-lunar fold. The "neck" of the pterygium is that part of it at the margin of the cornea. There is frequently, but not always, a tendency of

the growth to advance into the cornea, of which it seldom reaches the centre, and still more rarely extends quite across it.

In its early growth the pterygium is rather thick and succulent-looking, and very vascular. But finally it ceases to grow, and then becomes thin and pale, and this is its retrogressive stage; yet it never entirely disappears. Sight is not affected, unless the pterygium extend over the pupillary region of the cornea. A limitation of the motion of the eye to the other side, and consequent diplopia, is sometimes caused by a pterygium; but, for the most part, the disfigurement alone it is which brings these cases to the surgeon.

Cause.—The starting-point of a pterygium is often an ulcer at the margin of the cornea, which in healing catches a morsel of the limbus conjunctivæ and draws it towards the cicatrix, throwing the mucous membrane into a triangular fold. The ulcer then forms anew in the cornea immediately inside the cicatrix, and, in healing, the point of conjunctiva is drawn into it again, and is carried a little farther into the cornea, and so on. The hollow lying between a pinguecula (see below) and the margin of the cornea is apt to lodge small foreign bodies, which cause shallow marginal ulcers, and these, in healing, draw the pinguecula over on the cornea. A marginal ulcer in phlyctenular keratitis, or in acute blennorrhœa, may serve the same end. The only objection to this theory of the causation of pterygium is that an ulcer is not always to be found at the head of the growth.

Fuchs [1] believes that pterygium develops from the pinguecula, and that the latter causes nutritive changes in the cornea, loosening the superficial lamellæ, and allowing the connective tissue of the limbus to grow in.

Pterygium is a rare affection in this country, but is more common in countries, or localities, where the air is filled with fine sand, or other minute particles.

[1] *V. Graefe's Archiv*, xxxviii., Part 2, p. 1.

Treatment.—Unless the pterygium be very thick, and have invaded the cornea to some extent, or be progressing over the cornea, it is well to let it alone ; the more so as, by removing it, a quite normal appearance is not given to the eye, for a mark is necessarily left both on cornea and conjunctiva. If it be progressive, or very disfiguring, it should be removed, other proposed modes of dealing with it being futile. This may be effected either by ligature, or excision.

In the method by ligature, a strong silk suture is passed through two needles. The pterygium being raised with a forceps close to the cornea, one needle is passed under it here, and the other needle in the same way close to its base, the ligature being drawn half-way through. The thread is cut close behind each needle, thus forming three ligatures, which are respectively tied tight. In four or five days the pterygium comes away.

For excision, the apex is seized with a forceps and dissected off, either with a scissors or fine scalpel, care being taken not to injure the true cornea. A good plan is to pass a strabismus hook under the pterygium, when raised up from the sclerotic, and to forcibly separate the corneal portion by drawing the hook under it. The dissection is continued towards the base of the pterygium, where it is finished with two convergent incisions meeting at the base. The mucous membrane in the neighbourhood of the base is separated up somewhat from the sclerotic, and the margins of the conjunctival wound are then carefully brought together with sutures. Skin grafts, according to Thiersch's method, have been used with success to cover the defect.

Pinguecula (pinguis, *fat*) is the name given to a small yellowish elevation in the conjunctiva near the margin of the cornea, usually at its inner side, more rarely at its temporal margin, but sometimes in each place. It contains, notwithstanding its name, no fat, but is composed of connective tissue and elastic fibres. It is supposed to be due to the irritation

caused by small foreign bodies. It rarely grows to a large size, and requires no treatment, unless it become very disfiguring, when it may be removed with forceps and scissors.

Subconjunctival Ecchymosis.—The rupture of a small subconjunctival vessel in the bulbar conjunctiva, without conjunctivitis, is of frequent occurrence. It suddenly gives a more or less extensive purple hue to the "white of the eye," causing the patient much concern. It is common enough in old people, but may occur in the young, and even in children, from severe straining, as in hooping-cough, vomiting, or raising heavy weights. It is occasionally significant of diabetes. It also occurs sometimes during epileptic fits, and profuse subconjunctival hæmorrhage is occasionally found in cases of fracture of the base of the skull, having made its way along the floor of the orbit. It is of no importance, so far as the integrity of the eye is concerned.

Treatment.—None is required, the extravasated blood gradually becoming absorbed.

Nævus of the conjunctiva may occur along with the same condition of the lids, but it also occurs separately, especially on the plica or caruncle.

Treatment.—Electrolysis or ligature. Good results have been obtained with ethylate of sodium carefully painted on.[1]

Polypus of the conjunctiva, for which it is difficult to assign a cause, is sometimes seen. It is generally small, in connection with the semi-lunar fold or caruncle, and can readily be removed with the scissors. Granulations occurring after tenotomy for strabismus are sometimes, and incorrectly, called polypi.

Dermoid Tumours.—These are pale yellow in colour, and in size from that of a split pea to that of a cherry. They are smooth on the surface, and sometimes have fine hairs, and sit

[1] Snell, *Trans. Oph. Soc. Un. K.*, vol. xiii., p. 39.

usually at the outer and lower margin of the cornea; but Fig. 60 was drawn from a case on which I operated, where the dermoid was situated on the inner side of the cornea, extending over somewhat on the latter, and not at the most usual seat. In structure they resemble that of the skin. They are congenital tumours, supposed to be due to an arrest in development, but they often have a tendency to extend over the cornea. If this tendency be present, the tumour must be removed by dissecting it off the cornea, care being taken not to go into the deep layers of the latter.

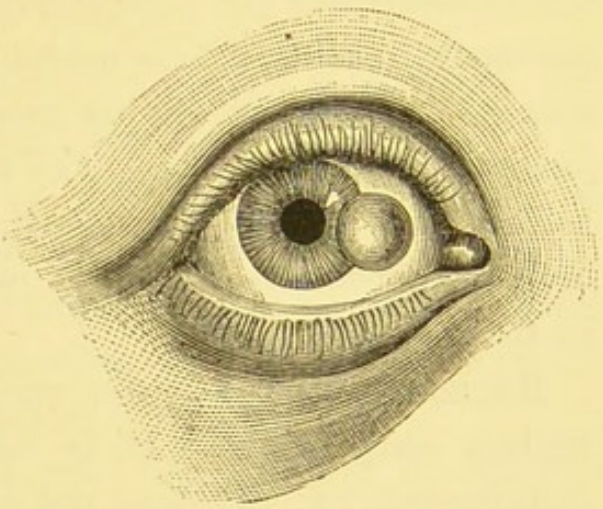


FIG. 60.

Lipoma occurs as a congenital subconjunctival formation of fat, usually situated between the superior and external recti muscles.

Syphilitic Disease of the Conjunctiva occurs both as primary and as secondary disease. It will be treated of in chap. vi., on Diseases of the Eyelids.

Papilloma, or **Papillary Fibroma.**—This is a non-malignant growth, which may spring from any part of the conjunctival sac. It appears in the beginning as a small round red knob. The papillomata growing from the tarsal conjunctiva, and from the semi-lunar fold, frequently take on a cauliflower appearance; while on the bulbar conjunctiva, and in the fornix, the growths are liable to be pedunculated, with a papillary surface. The limbus of the conjunctiva is a favourite seat for a papilloma, and in the early stage it may be impossible to distinguish it from an epithelioma. But if the case come under observation at a later stage, when the growth has overlapped the cornea, this difficulty does not arise; for the papilloma merely lies on the cornea, and can be

lifted freely off it with a probe, while the epithelioma infiltrates the corneal tissue.

Treatment.—Thorough removal with knife or scissors, and actual cautery, as otherwise the growth is liable to recur.

Epithelioma is not common as a primary disease of the conjunctiva. When it is so found, it is seen as a little non-pigmented tumour growing from the limbus of the conjunctiva, surrounded by vascularisation, and may in this stage be mistaken for a phlyctenula—of which, however, the margins are not so steep—or for a papilloma (*vide supra*). As the tumour increases in size, it becomes lobulated, and ulcerates, and soon attacks the cornea, giving rise on the latter to an appearance very like pannus. The neighbouring lymphatic glands become enlarged. The so-called coccidia which have lately been discovered in epithelial cancers have also been observed in these tumours.[1]

Sarcoma, too, is rare, and also takes its origin in the limbus conjunctivæ. It is usually a pigmented tumour, a melanosarcoma. It does not attack the cornea so readily as the epitheliomatous growths, although it often overlaps the surface of the cornea. In its later stages this tumour grows to an enormous size. That these sarcomata are pigmented, is explained by the fact that the limbus contains pigment; although usually so slight in amount as not to be visible to the naked eye.

Treatment.—Both epithelioma and sarcoma of the conjunctiva demand prompt operative removal, in order to prevent an extension of the growth to the rest of the eye, if the case be seen early, as well as to avert metastases to other organs. The knife and actual cautery may save the eye and the life in the early stages; but, later on, removal of the whole eye is often called for.

Simple Cysts of the conjunctiva are very rare. They

[1] Lagrange, Etudes sur les tumeurs de l'œil, de l'orbite, etc., Paris, 1893.

appear as clear spherical protuberances of about the size of a pea, seated usually on the bulbar conjunctiva. The walls of the cysts contain but few vessels, are thin, and almost transparent; while for contents they have a clear limpid fluid. These cysts cannot, as a rule, be moved from their position, because they are adherent to the conjunctiva, which indeed takes part in the formation of their walls. They are, very probably, dilated lymphatic vessels. Small bead-like strings of dilated lymphatics are very frequently seen on the bulbar conjunctiva.

These simple cysts are most commonly congenital, but they may begin to be developed during life.

Treatment.—The cyst may be dissected out, or it may be sufficient to abscise its anterior wall.

Subconjunctival Cysticercus is a little more common than simple cyst of the conjunctiva, and yet only forty-six examples of it have been placed on record.[1]

Cysticercus is distinguished from simple cyst by its free mobility under the conjunctiva, to which it is not attached; by its thicker and more vascular walls; and, above all, by the presence of a round, white, opaque spot on the anterior surface, first pointed out by Sichel, and looked on by him as pathognomonic of a cysticercus. This spot indicates the position of the receptaculum; and occasionally, when this comes to be placed on the posterior surface of the cyst, it may be difficult, or impossible, to make the diagnosis with certainty.

Treatment.—The cyst may be pushed to one side under the conjunctiva, an incision made in the latter, the cyst then pushed back again, and out through the opening.

Lithiasis consists in the calcification of the secretion of the Meibomian glands, which are seen as little brilliantly white spots not larger than a pin's head in the conjunctiva. There

[1] L. Werner, "Subconjunctival Cysticercus," *Trans. Ophthal. Soc.*, ix., p. 74. The literature of the subject is there fully given.

may be one only, or very many. These concretions often give rise to much conjunctival irritation; and, if they protrude over the surface of the conjunctiva, may injure the cornea. Each one—the eye having been cocainised—must be separately removed by a needle, with which first an incision has been made into the conjunctiva over the concretion.

Uric Acid Deposits have been seen in the palpebral conjunctiva in gouty cases. They occur more frequently than is supposed, and give the murexide reaction.[1]

Injuries of the Conjunctiva.—Foreign bodies frequently make their way into the conjunctival sac, and cause much pain, especially if they get under the upper lid, by reason, chiefly, of their coming in contact with the corneal surface during motions of the lid and of the eye. If the foreign body be under the lower lid, it will be easily found on drawing down the latter, and, provided it be not actually embedded in the mucous membrane, it is easily removed with a camel's-hair pencil, or with the corner of a soft pocket-handkerchief. But, if the foreign body be under the upper lid, it is necessary to evert the latter before it is reached. Should the foreign body be embedded in the conjunctiva, it must be pricked out of its position with the point of a needle or other suitable instrument, and the little proceeding will be made easier, both for patient and surgeon, by the instillation of a few drops of solution of cocaine (2 per cent.) into the eye.[2]

The conjunctiva is frequently injured in severe wounds of the eyelids or eyeball. The interest, and treatment, centre here chiefly on the other more important parts, which have been injured. A tear, or wound, of the conjunctiva (usually of the bulbar portion), when it occasionally occurs without injury to other parts, is in general of very slight moment.

[1] *Trans. Clin. Soc.* London, Jan. 1893.

[2] The continuous, or frequently recurring, sensation of a foreign body in the conjunctival sac, while nothing of the kind, nor any hyperæmia, is present, is sometimes a premonitory sign of mental disease.

If the wound be extensive, its edges should be drawn together with a few points of suture; but, otherwise, healing will take place with the aid simply of a bandage to keep the eye closed for a few days.

The common form of injury, which may involve the conjunctiva alone, is a burn by acid or lime. In the case of a strong acid getting into the eye, if the patient be seen immediately after the occurrence, the whole conjunctival sac should be well washed out with an alkaline solution; while, in the case of lime, a weak solution of a mineral acid is indicated for the purpose. Cocaine may be employed to relieve the pain. Subsequently, protection of the eye, with the use of olive or castor oil dropped into it, will best promote the healing process.

In the case of a severe burn of the conjunctiva, the resulting cicatrix is liable to produce a more or less extensive union of the eyelid to the eyeball (Symblepharon), which often interferes with the motion of the latter, or even with vision, if the cornea be obscured. No measures taken during the healing process can prevent symblepharon, if the degree of the burn be such as to bring it about. The relief of symblepharon by operation will be dealt with in chap. vi., on Diseases of the Eyelids.

CHAPTER V.

PHLYCTENULAR, OR STRUMOUS, CONJUNCTIVITIS AND KERATITIS.[1]

Both from a clinical and nosological point of view, it would be incorrect to divide this affection into two, under the heads of Diseases of the Conjunctiva, and Diseases of the Cornea; and, therefore, I treat of it here as one disease; and, being a very important disease, I devote a special chapter to it. It is important, because it is excessively common, and because it is capable of causing considerable damage to sight. Moreover, even when it occurs on the cornea, it should probably be regarded as a conjunctival disease, for the corneal layer, which it primarily attacks, is the epithelium, and this —if not also, as some authors state, Bowman's membrane and the anterior layers of the true cornea—as we know from the fœtal development of the membrane, is a continuation of the conjunctiva, in a modified form, over the cornea.[2]

Horner[3] termed it Eczema of the Conjunctiva and Cornea. It is characterised by the eruption of phlyctenulæ (*φλύκταινα*, a *vesicle*, or *pustule*) on the conjunctiva bulbi (but never on the palpebral conjunctiva), on the conjunctival limbus, or on the cornea, and is chiefly a disease of children up to the eighth or tenth year of age.

---

[1] *κέρας*, a horn.

[2] The posterior epithelium—or even, according to some, this along with the membrane of Descemet and the posterior layers of the true cornea—is to be reckoned to the uveal tract; while the true cornea—or, according to some, only its central layers—is a modification of the sclerotic.

[3] *Loc. cit.*, Bd. V., Abth. 2, p. 279.

Notwithstanding the derivation of the word, a phlyctenula, or phlyctene, is originally neither a vesicle nor a pustule; but, when on the conjunctiva, is a solid elevation consisting of a collection of lymph cells, and is of a greyish colour. In a late stage, or under unsuitable treatment, the phlyctenula may, it is true, become a pustule. On the conjunctiva two types of the disease can be recognised :—

1. **The Solitary,** or **Simple, Phlyctenula.**—Of this there may be one or several, varying in size from 1 mm. to 4 mm. in diameter. The vascular injection is immediately around the phlyctenula, and is not diffused over the conjunctiva. At first there may be shooting pains and lacrimation, but these soon pass away. If the phlyctenulæ be not seated close to the cornea, the affection is not serious; and the length of time required for its cure depends on the size of the phlyctenulæ, varying from seven to fourteen days, as a rule.

2. **Multiple,** or **Miliary, Phlyctenulæ.**—These are very minute, like grains of fine sand, and are always situated on the limbus of the conjunctiva, which is swelled. The general injection and swelling of the conjunctiva are considerable; and occurring, as it does, almost exclusively, in young children, the affection may be called Eczematous Conjunctival Catarrh of Children (Horner). The irritation, and so-called photophobia, and lacrimation are often considerable, and there is a good deal of conjunctival discharge. This form is very apt to appear after measles and scarlatina.

Both forms are liable to extend to the cornea, and then, only, does the disease become serious. This event may come about in the following different ways :—

The Solitary Phlyctenula may be seated partly on the limbus conjunctivæ and partly on the margin of the cornea, and may undergo resolution.

Or, it may give rise to a deep ulcer, which either heals, leaving a scar; or perforates, causing prolapse of the iris, etc.

Or, it may form the starting-point of a progressive riband-like keratitis (Fascicular Keratitis), the pustule becoming an ulcer, at the margin of which the corneal epithelium is raised and infiltrated in crescentic shape. This now steadily advances for many weeks towards the centre of the cornea, followed by a leash of vessels which has its termination in the concavity of the crescent. The process is accompanied by much irritation of the terminal branches of the fifth nerve in the cornea, and the consequent reflex blepharospasm. A permanent mark indicates the track of the ulcer.

The Multiple Miliary Phlyctenulæ on the limbus conjunctivæ may cause some slight superficial infiltration, and vascularisation, of the cornea in their immediate neighbourhood, which pass off when the phlyctenulæ disappear.

Or, they may be accompanied by deeper marginal infiltrations of the cornea, which become confluent and result in an ulcer, that extends along the margin of the cornea for some distance, and is termed a Ring Ulcer. It is a serious form of ulcer; for, if it extend far round, it may destroy the cornea in a few days by cutting off its nutrition.

Primary Phlyctenular Keratitis occurs principally in three different forms:—1. Very small grey subepithelial infiltrations, which are apt to turn into small ulcers, and then heal, leaving a slight mark. This mark may ultimately quite disappear, especially in the case of children, and when situated peripherically. 2. Somewhat larger and deeper infiltrations, resulting in ulcers of corresponding size, which heal by aid of vascularisation from the margin of the cornea. The opacity left after these ulcers is rather intense, and clears up but little, especially if the situation be central. 3. Large and deep-seated pustules, often at the centre of the cornea, giving rise to large and deep ulcers, which may be accompanied by hypopyon and even by iritis, and which frequently go on to perforation.

Photophobia is usually a prominent symptom in phlyctenular keratitis. The term photophobia, however, is not alto-

gether correct, for it is the fifth nerve (from the cornea), which is mainly the afferent nerve here, rather than the optic nerve. This is evident from the fact, that in the dark the patient does not get complete relief. The explanation of this reflex blepharospasm has been given by Iwanoff,[1] who showed that the round cells, in making their way from the margin of the cornea to their position under the epithelium, follow the course of the nerve filaments, which they irritate

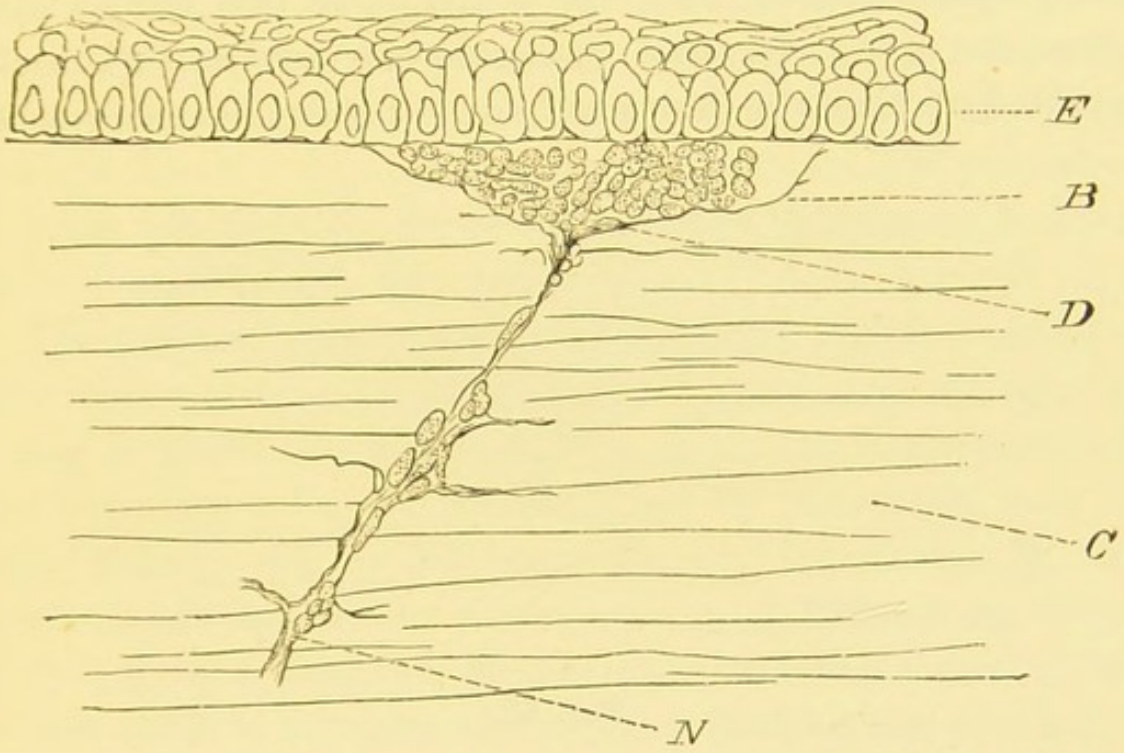


FIG. 61.—*E*, Epithelium; *B*, Ant. elastic Lamina; *C*, True Cornea; *N*, Nerve Filament, with Lymph Cells on its course; *D*, Phlyctenula.

in their progress. The accompanying Figs. 61 and 62 are from his original paper.

Eczema of the eyelids, face, and external ear, and catarrh of the Schneiderian mucous membrane, frequently accompany phlyctenular conjunctivitis and keratitis.

In these cases, in children of three or four years of age, temporary amaurosis has sometimes been observed after a severe and long-continued blepharospasm has passed away. The patient is found to be unable to see even large objects,

[1] *Klin. Monatsblätter f. Augenheilkunde*, 1869, p. 465.

or to find his way; although the pupil reflex is active, and a strong light may still be distressing. There are no ophthalmoscopic appearances. This blindness passes away completely in from two to four weeks. It has been regarded as a reflex phenomenon, and again it has been held to be due to disturbance of the intraocular circulation from pressure of the eyelids on the eyeball. But the view (Leber, Uhthoff) which represents it as having a central cause is probably the correct one. It is likely, at this tender age, when the psycho-physical processes are not as yet firmly established, that the desire not to see, and the active with-

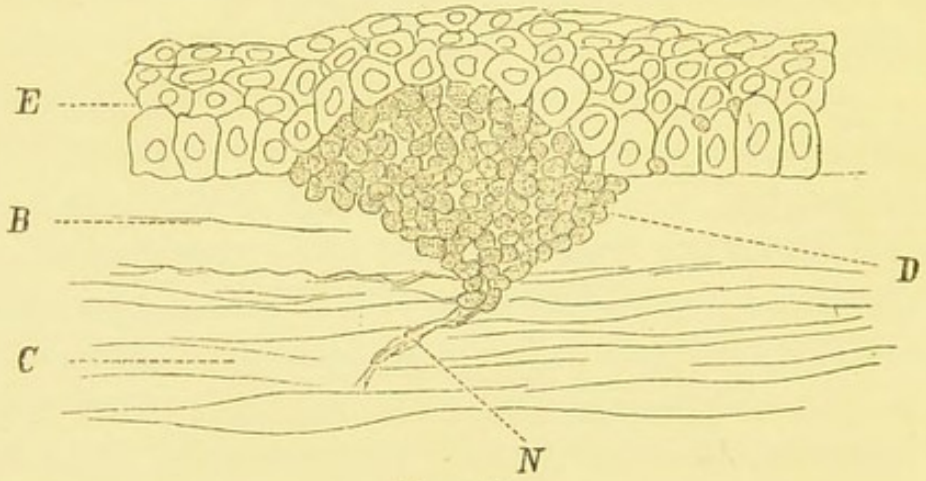


FIG. 62.

drawal from the act of vision, leads in a short time to a functional paralysis of the visual centres in the brain; and that these take some time to recover, or to re-learn their functions, when the ground for the suspension of the latter has ceased.

Cause.—This is a disease of childhood, although it is rare in the very first year of life. In adults it is uncommon.

The strumous constitution, as indicated by the swollen nose and upper lip, and sometimes by the enlarged lymphatics in the neck, as well as by the eczema just mentioned, is that most liable to this affection. Often, however, it will be found in strong children with apparently perfect general health; but, even in them, there is probably some allied irregularity

of nutrition, of which the great tendency to recurrence of the eye affection is evidence.

Colonies of straw-coloured micrococci may be found in the contents of the phlyctenulæ; but what etiological relationship to the production of the phlyctenulæ they possess is not yet known.

Treatment.—The solitary phlyctenula is best treated with the yellow oxide of mercury ointment[1] (commonly known as Pagenstecher's ointment), of which the size of a hemp-seed should be put into the eye once a day. Or, a small quantity of pure calomel dusted into the eye once a day will also cure; but this remedy should not be employed if iodide of potassium is being taken internally, for then iodide of mercury is liable to be formed in the conjunctiva.

The miliary phlyctenular conjunctivitis is best treated at first with cold or iced applications. Freshly prepared chlorine water (1 part Liq. Chlori., 9 parts water) to be dropped into the eye once a day is recommended by some, and later on Liq. plumbi dil. or Sol. argent. nitr. (grs. v ad ℥j, and neutralised) applied to the everted conjunctiva; or, if the phlyctenular appearance predominate over the catarrhal, the yellow oxide of mercury ointment, or insufflations of calomel, may be preferred. I myself rarely employ any remedy other than the two latter, which I find applicable to all these cases.

When the cornea is slightly affected near the margin, in cases of miliary phlyctenulæ, calomel, or Pagenstecher's ointment, and warm fomentations, should be used.

When a large pustule on the margin of the cornea has resulted in a deep ulcer, with tendency to perforate, and

[1] ℞.—Hydrarg. Perox. Præcip. gr. xxx. Vaselin, ʒj.—M.

NOTE.—Hyd. Perox. Præcip. is prepared by precipitating the Bichloride of Mercury with Liq. Sodæ, and washing the resulting oxide. I learn from Mr. Jabez Hogg that this ointment was in use by the late Mr. Guthrie, in the year 1849.

accompanied by much pain, I cannot too highly recommend paracentesis of the anterior chamber through the floor of the ulcer, the pupil having first been brought well under the influence of eserine, to prevent prolapse of the iris. The good effect of this will be very soon apparent: the pain disappears, the patient sleeps, the ulcer becomes vascularised, and healing sets in. Cauterisation of the ulcer in an early stage with the galvano-cautery is also good practice; but in these cases I prefer the paracentesis. Many surgeons trust very much to eserine, warm fomentations, and a pressure bandage.

For the fascicular keratitis the yellow oxide of mercury ointment is in its place. When the crescentic infiltration is very intense, it is well to touch it with the galvano-cautery. Division of the leash of vessels at the margin of the cornea has a beneficial effect.

For the ring ulcer, a pressure bandage, under which an antiseptic dressing (boracic or salicylic acid, or perchloride of mercury) has been placed, is, perhaps, the best method of treatment. Warm fomentations promote vascular reaction, and may be used with benefit at each change of bandage.

For primary phlyctenulæ of the cornea in the form of the minute grey superficial infiltration or ulcer, nothing beyond atropine, with warm fomentations, and a protective bandage to keep the eyelids quiet, should be used. When reparation of the ulcer has commenced, calomel, or weak yellow oxide of mercury ointment, may be employed.

For the large purulent phlyctenula, resulting in a large and deep ulcer, often situated at the centre of the cornea, with hypopyon and iritis, warm fomentations (camomile, or poppy-head, at 90° F., for twenty minutes three times a day), atropine, iodoform as ointment or powder, and a protection bandage form the treatment in the early stages. Here, also, I often puncture the ulcer with the very best results in respect of hastening the cure, and the galvano-cautery may be used

with advantage. In the stage of reparation, Pagenstecher's ointment or insufflations of calomel are very useful.

In all forms of phlyctenular ophthalmia, those favourite remedies blisters, setons, and leeching should be avoided. The first two worry the patient, give rise to eczema of the skin, and are not to be compared in their power of cure with the measures above recommended; while leeching gives, at best, but temporary relief, and deprives the patient of blood which he much requires.

For relief of the blepharospasm, in addition to the use of atropine, plunging the child's face into a basin of cold water, and keeping it under until he struggles for breath, and this immersion repeated two or three times in rapid succession, and used every day if necessary, is a most efficacious means. It should always be used where the blepharospasm is severe, as this is not only distressing to the patient, but also an obstacle to the cure.

The general treatment, notwithstanding the so-called photophobia, should consist in open-air exercise before everything else; unless, indeed, there be an ulcer which threatens to perforate. It is not well to keep the patient's face or eyes covered with bandages and shades, nor to confine him to a dark room. A pair of dark-blue glasses are the best protection from strong glare of light; and shady places can be selected when the patient is out of doors. Cold or sea baths, followed by brisk dry rubbing. Easily assimilated food at regular meal hours, but no feeding between meals. Regulation of the bowels. Internally: cod liver oil, maltine, iron, arsenic, syr. phosph. of lime, and such-like remedies are indicated.

The great tendency to recurrence is one of the most troublesome peculiarities of all kinds of phlyctenular ophthalmia; and in order to prevent this, so far as possible, it is important to continue local treatment, until the eye is perfectly white on the child's awaking in the morning, and even for fourteen

days longer. This prolongation of the treatment will also assist in clearing up opacities, as best they may be. For this after-course of treatment calomel insufflations should be used.

Nothing can be done for the opaque scars left on the cornea by ulcers, when all inflammatory symptoms have subsided. If the ulcer have been very superficial, the resulting scar in young children may disappear in the course of time. Deep ulcers cause more opaque and permanent scars, and ulcers which have perforated produce the greatest opacity. Some of the very disfiguring scars may be tattooed (see chap. viii.).

The degree of the defect of vision to which an opacity of the cornea may give rise depends, in the first instance, on the position of the opacity. If it be peripheral, the vision may be perfect; but if it be in the centre of the cornea, sight may be seriously damaged. Even a slight nebula, barely visible to the observer, will cause serious disturbance of vision, if situated in the centre of the cornea; while, in the same situation, the very opaque scar of a deep ulcer will produce a proportionately greater defect. If a central, but not deep, ulcer should not become completely filled up in healing, and a facet remain, vision will also suffer much in consequence of irregular refraction, although there may be but little opacity.

CHAPTER VI.

DISEASES OF THE CORNEA.

The importance of a knowledge of the diseases and injuries of the cornea depends on their great frequency, coupled with the fact that nearly every one of them is liable to leave behind it some opacity, with resulting defect of sight and disfigurement of the eye; while several of them are very apt to lead to complete loss of sight.

Inflammations of the Cornea.

From a clinical point of view these will be most conveniently considered under the headings—(*a*) Ulcerative Inflammations, and (*b*) Non-ulcerative Inflammations.

(*a*) Ulcerative Inflammations of the Cornea.—Before an ulcer can form in the cornea, there must be a cellular infiltration of its tissue near its anterior surface; and this cellular infiltration is brought about, we nowadays believe, by the entrance into the cornea of certain micro-organisms, the gonococcus, or the staphylococcus pyogeneus, or other, as yet undescribed, forms. One recognises the existence of an infiltration by seeing an opaque spot in the cornea, with a dulness of the layers over it, and often of the corresponding part of the epithelium. Before long the epithelium covering the infiltration comes away, and soon the intervening layers of the true cornea break down, and then we have an ulcer established.

But, although all ulcers of the cornea originate in an

infiltration; yet, when once established, they take on a great variety of type, in consequence, it may be, of a variety in the nature of the originating micrococcus. Some ulcers are purulent, others non-purulent; some tend to spread over the surface of the cornea, others tend to go deep into it; some attack by preference the central region of the cornea, while others are confined to its margin; some readily give way to treatment, and others are very obstinate, or almost incurable. Again, some ulcerative corneal processes are attended by much circumcorneal injection, severe pain in and about the eye, great reflex blepharospasm, and lacrimation; whilst others, which may really be more severe processes in so far as the integrity of the eye is concerned, can run their course with scarcely any injection of the eyeball, and with little or no distress to the patient.

Etiologically, corneal ulcers are primary or secondary. The primary ulcers are those in which the diseased process originates in the cornea, most commonly as the result of traumata, but also in phlyctenular keratitis, or as the result of corneal abscess, or where the nutrition of the cornea is interfered with, etc. Secondary ulcers are those which are the result of disease elsewhere, usually in the conjunctiva, as in acute blennorrhœa, and in conjunctival diphtheritis.

Corneal ulcers are more common in advanced than in early life. Indeed, in early life, unless in cases of blennorrhœa neonatorum, and of phlyctenular disease, corneal ulcers are, I may say, unknown. The greater liability to these affections in advanced life is due, no doubt, to a less active nutrition at that period in this already lowly organised part. Hence, slight traumata, or the presence of a slight conjunctival catarrh, which would have no ill effect in a young person, may form the starting-point of a corneal ulcer in an old person, or even in one of middle age. For the same reasons, corneal ulcers are much more common in the lower orders than amongst the well-to-do; for the general nutrition of

the poor is often defective, while they are more exposed to traumata than are the better classes.

The Diagnosis of the presence of a large corneal ulcer is very simple. Inspection of the cornea in ordinary daylight at once reveals the loss of substance, more or less extensive, deep, and infiltrated. If the ulcer be very small and shallow, the difficulty is greater, especially if there be much blepharospasm. In such cases the surgeon must endeavour to inspect the cornea from different points of view, either by directing the patient to move his eye, or by moving his own head, until he succeed in obtaining such an incidence of the light, as will display the minute loss of substance, with its margin, and more or less grey infiltrated floor. Or, he may employ the oblique illumination with artificial light. An instillation of cocaine may be necessary to facilitate the examination, by diminishing the blepharospasm.

It is obviously important to decide at the outset whether a grey spot in the cornea be an infiltration (= a collection of cells which may shortly become an ulcer), an ulcer, or a scar (= a healed ulcer, or other loss of substance). The surface covering an infiltration, although flush with the general surface of the cornea, has usually a steamy appearance, due to some disorganisation of the corneal epithelium, and is not polished. With an ulcer the appearances above described will be found. The surface of a scar is usually, although not always, flush with the general surface of the cornea, and it is a polished surface—*i.e.*, covered with normal epithelium, not rough, irregular, or even steamy.

A very beautiful method for ascertaining the presence and true extent of a corneal ulcer, or traumatic loss of substance, is the instillation of a 2 per cent. solution of fluorescin. Almost immediately afterwards, the tissue forming the floor of the loss of substance assumes a greenish tint, which clearly differentiates it from the surrounding normal cornea.

The presence of *Hypopyon* (*ὑπό*, *under*; *πῦον*, *pus*) is the

rule with some types of corneal ulcer. Hypopyon is a deposit of pus in the anterior chamber, and, as the patient sits or stands, it lies in the lowest part of the chamber, to which place it has gravitated. If the patient lie in bed, say, on the side of the affected eye, the hypopyon will of course change its position, and gravitate towards the outer side of the chamber. Sometimes the hypopyon is so small as to be detected with difficulty; and again it may fill the whole anterior chamber, completely obscuring the iris. It will be asked:—From whence does the pus come, which forms hypopyon in cases of corneal ulcers? It might be supposed that it is derived directly from the purulent floor of the ulcer, by passage of the pus-cells through the posterior layers of the cornea. But this is not so. No pus cells do, or indeed can, pass through the membrane of Descemet. Moreover, copious hypopyon is often present, when the corneal ulcer is quite small, and non-purulent. The pus-cells which form hypopyon in cases of corneal ulcer come from the iris, in compliance with the law which causes leucocytes to wander out of blood-vessels in the neighbourhood of an inflammatory focus, and to make their way towards that focus. When these leucocytes from the iris reach the anterior chamber, they can go no farther, owing to the barrier imposed to their progress by the membrane of Descemet.

The pus forming a hypopyon contains, in its early stages at least, no microbes. These interesting facts concerning the genesis and nature of hypopyon have been discovered by Professor Leber.[1]

The Dangers attendant upon Corneal Ulcers are, first of all, the opacities, the scars, which even the slightest of them are apt to leave behind.

Fig. 63 represents a section made through a deep ulcer in its progressive stage. At the margin of the ulcer the epithelium (*e*) and Bowman's membrane (*b*) cease. The floor

[1] *Die Entstehung der Entzündung*, Leipzig, 1891.

of the ulcer is seen covered with pus, which also infiltrates the corneal tissue in the neighbourhood. As soon as cure commences, the floor of the ulcer begins to get clear, *i.e.* it becomes gradually less covered with pus, until it is finally

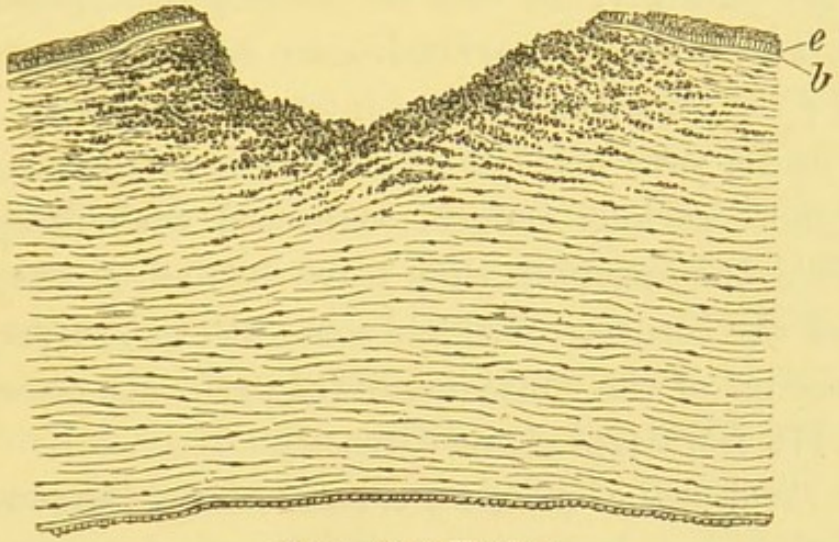


FIG. 63 (*Fuchs*).

quite free from it, and *pari passu* the surrounding infiltration is absorbed. Then, the epithelium, growing in from the margin (*m m*, Fig. 64) all round, gradually carpets over the floor of the ulcer, and, underneath this newly formed epithe-

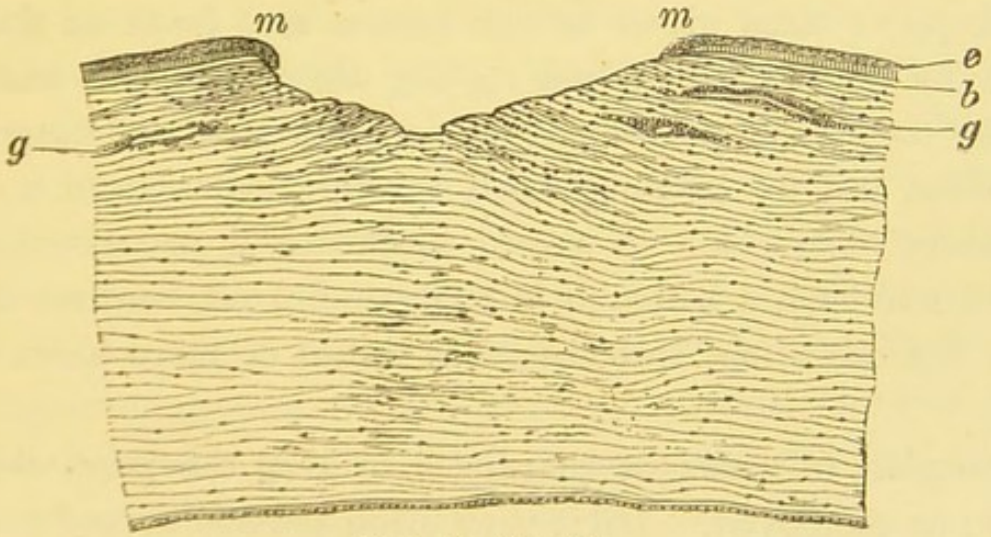


FIG. 64 (*Fuchs*).

lium, the new tissue, which is to close the loss of substance, is laid down. This new tissue, however, is not corneal tissue, but is ordinary connective tissue, and is therefore opaque. Hence, the deeper the ulcer has been, the more intense will

be the resulting opacity. Bowman's membrane never becomes restored over the cicatrix.

The ulcers which are situated at the centre of the cornea, in the pupillary area, are more serious for sight, than those situated peripherally, as can be readily understood. The opacity left by a very superficial ulcer is slight, and is called a nebula; a somewhat more intense opacity is called a macula; and a very marked white star is called a leucoma.

But a more serious danger, connected with ulcers of the cornea, than the opacities they leave behind, is that of perforation of the cornea, to which some ulcers are very prone. For an account of the consequences of perforation see pp. 116, 154, and 176 (on Staphyloma Corneæ).

In the *Treatment* of primary corneal ulcers the student will soon perceive that a bandage, atropine, and warm fomentations play prominent parts.

The bandage should be put on with firm pressure,—but should not be made uncomfortably tight,—the eye having been previously padded out, especially at the inner canthus, so that equal pressure may be exercised on the globe all over. The support thus given to the cornea and front of the eye promotes the healing process in the ulcer, and the bandage is also useful by preventing the eyelids from rubbing over the ulcer, and by keeping small foreign bodies from it. In secondary ulcers, due to severe conjunctival processes, such as blennorrhœa, a bandage is contraindicated, because it retains the secretion, and therefore would do more harm than good.

Atropine, in sufficient quantities to keep the pupil dilated, should be employed. Iritis very often attends severe corneal ulcers, and here the indication for atropine is obvious. But rest of the affected part is, we know, an important element in preventing, or in curing, any inflammation; and, in the affections we are now treating of, even where there is no iritis, atropine acts by procuring rest of the iris, and of the ciliary

muscle, the constant motion of which would otherwise tend to augment the inflammatory process in the cornea.

Some surgeons use myotics (eserine, or pilocarpine) in preference to atropine in the treatment of corneal ulcers. They hold that their power of reducing the intraocular tension encourages healing of the ulcers; while they also think the more extended surface of iris presented facilitates absorption of the hypopyon. But it is doubtful whether myotics do reduce the normal tension, although they often have that effect upon abnormal tension; and my objection to them in these cases is that they increase, I believe, the tendency to iritis.[1] Absorption of the hypopyon will only come about when the cornea begins to recover, whatever the treatment may be. I am not singular in this view of the use of eserine in corneal ulcers. An indication for myotics, however, is given by the presence of an ulcer near the corneal margin with a tendency to perforate, for here the myosis would assist in preventing prolapse of the iris, should perforation take place.

Warm formentations promote the healing process by stimulating tissue-changes in the cornea. One usually orders them to be made with poppy-head water or chamomile tea, although no doubt warm water would be equally efficacious. Hot solutions of 4 per cent. boracic acid, or 1-5000 corrosive sublimate, may be used with advantage. The bandage having been removed, a compress of lint dipped in the stupe at about 120° Fahrenheit is laid upon the eye, and frequently replaced by fresh compresses out of the stupe, so that the one on the eye may be always hot. This is continued for half an hour at a time, and repeated every two or three hours.

In an ulcer of a purulent or sloughing nature, the insuffla-

[1] I would feel disposed to qualify this statement in the case of purulent phlyctenular ulcers, with hypopyon, in children. They are much less prone to iritis than adults, and more rapid absorption of the hypopyon in these cases may be obtained with eserine than with atropine.—L. W.

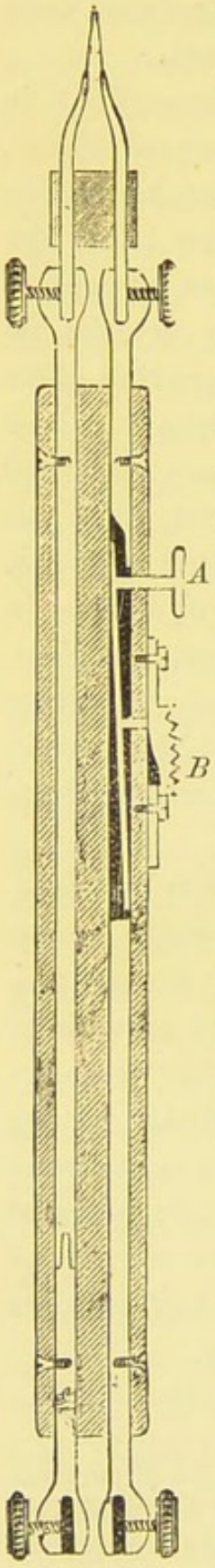


FIG. 65.

tion on its floor of very finely divided iodoform powder is useful.

Thorough scraping of the floor of the ulcer with a small sharp spoon is a very important and valuable method.

The actual cautery has, of late years, come much into use in the treatment of purulent and serpiginous corneal ulcers. It acts by destroying the micro-organisms, which keep the process going. Either a thermo-cautère, in the form of a very fine point, or the galvano-cautery (Fig. 65), may be employed. To the latter a medium-sized bichromate of potash bottle-battery is attached, and the platinum wire brought to a red-heat. The eye having been cocainised, the red-hot cautery is brought into contact with the whole surface of the ulcer, so as to thoroughly destroy its superficial layer, and special attention is paid to any part of the margin of the ulcer, where it seems inclined to spread to as yet healthy tissue. Fluorescine may be used to show the extent of the ulcerated surface. The cauterisation can be repeated as often as the progress of the ulcer makes it desirable. It is well to perforate the cornea with the cautery, and to evacuate the aqueous humour and hypopyon; or, this may be done with an ordinary paracentesis needle, after the cauterisation is completed. My own experience of the cautery in

Fig. 65.—The bolt A being pushed forwards, the current is completed, and passes through the platinum wire which forms the cautery. By pressure on the button B the current can be momentarily intercepted during use of the instrument.

these cases is extremely satisfactory. It seems to give the best percentage of cures with the least amount of opacity.

Snellen and others prefer scraping to the cautery, on the grounds that the former does not injure the healthy tissue as the latter may do. After scraping he touches the surface of the ulcer with tincture of iodine.

FIG. 66

Paracentesis of the anterior chamber through the floor of the ulcer is another most valuable therapeutic measure for some corneal ulcers, and deserves a more routine application in these cases, than is at present accorded to it; the more so, as the valuable little operation is simple and dangerless. But there are, I think, two imperative indications, two golden rules, for its use, namely:—(1) If there be great pain. Very shortly after the operation, which for the moment increases the neuralgia, the patient experiences the greatest relief, and passes the first good night after many wakeful ones. (2) If perforation seem to be imminent. This may often be recognised by a bulging forwards of the thin floor of the ulcer; but sometimes it is not easily foreseen, and if there be any doubt on the point, paracentesis should be performed. It is important to forestall spontaneous perforation of the ulcer by this proceeding; because, the opening made by the latter being linear, it heals easily, and leaves but a slight scar without anterior synechia; while the natural opening would be a complete loss of substance, and, therefore, the more readily involve adhesion of the iris in the resulting comparatively extensive cicatrix. Other indications for the operation are, increased tension and the presence of a large hypopyon.

Paracentesis of the anterior chamber is best performed by means of a paracentesis needle (Fig. 66), which is a somewhat shovel-shaped instrument, with a shoulder or stop. If this be not at hand, a small iridectomy knife, or a broad

needle, will answer the purpose. The eye having been cocainised, a spring lid-speculum is inserted, the eye fixed with a fixation forceps, and the point of the paracentesis needle applied to the floor of the ulcer, in such a way that the plane of the little blade may be at an angle of about 45° with that of the floor of the ulcer. The point is pushed gently through the floor, and the plane of the blade is then immediately changed, so that, as the instrument is being advanced up to the shoulder, it may be almost in contact with the posterior surface of the cornea. The withdrawal of the instrument should be effected with extreme slowness, in order that the aqueous humour may flow off gradually, and not with a rush. If these precautions be taken there need be no danger of injuring the crystalline lens, of causing intraocular hæmorrhage, or of having prolapse of the iris in the incision. If the latter should occur, it can usually be reposed with the spatula. It may happen that, when the needle has been quite withdrawn, a considerable portion of the aqueous humour may still remain in the anterior chamber, unable to escape owing to the valve-like closure of the wound, It should be evacuated, by making the wound gape by gentle pressure with a spatula on its posterior lip. If it be desirable to tap the anterior chamber on the next day, it can be done by simply opening up the wound with a spatula, or with the probe-like instrument at the other end of the handle (Fig. 66), without the aid of any cutting instrument.

If the case do not come under the care of the surgeon until perforation of the ulcer with prolapse of the iris has taken place, the very important question as to the best method of dealing with the condition is presented. The same question arises in other forms of perforating ulcer. If the loss of substance occupy one-third or more of the cornea, with correspondingly large prolapse of iris, little can be done beyond the use of eserine—and here I would use eserine—to reduce the intraocular pressure, along with the application of

a firm bandage; for, in such cases, the formation of a corneal staphyloma is almost inevitable. But, if the ulcer and prolapse be small, an attempt may be made to free the iris; so that no anterior synechia may form, and in order that the cicatrix may be flat, and not raised over the surface of the cornea, and, therefore, exposed to injury. The importance of such an attempt lies in the fact, that a corneal cicatrix with iris entangled in it—not merely adherent to its posterior surface—affords a constant source of danger, especially if situated near the margin of the cornea; for, in such eyes, sudden and uncontrollable purulent inflammation of the iris and chorioid may come on, after an apparently slight trauma, and end in total destruction of the eye. This event is due to septic infection reaching the interior of the eye through a superficial loss of substance, the direct result of the trauma. The surgeon's attention should therefore be directed to obtain at least as flat a cicatrix as possible, or, still better, a non-adherent cicatrix. The practice which I, as well as many other surgeons, have commonly followed, is to draw the prolapsed portion of iris slightly forwards with a forceps, and to snip it off level with the surface of the cornea; and then, with a spatula, to endeavour to free the iris from any adhesions it may have formed with the margin of the ulcer. Atropine or eserine, according to the position of the ulcer, is then instilled, and a bandage carefully applied. This proceeding is only of use when a fresh prolapse can be dealt with, before cicatrisation sets in; and the result is often satisfactory, so far as the securing of a flat cicatrix is concerned, but an anterior synechia can rarely be avoided.

Dr. da Gama Pinto has successfully employed the following method for obtaining a non-adherent cicatrix:—Having abscised the prolapsed portion of iris as above, and freed all adhesions to the margin of the ulcer with a spatula, he covers the opening in the cornea with a flap cut from the bulbar conjunctiva,—and this flap should be twice as large

as the opening, in order to admit of its shrinkage,—and then pushes the flap into the opening with a blunt probe. A firm binocular bandage is applied—but no iodoform. The eye is not dressed until the third day, when the anterior chamber is often found restored, the iris all in its proper plane, and the conjunctival flap healed into the ulcer. Ultimately, all trace of the flap disappears, and an ordinary non-adherent corneal scar is presented. I have employed this method twice, and in each case with a good result.

Aniline dyes, more particularly pyoctanin, have been recommended in the treatment of purulent ulcers, especially by Stilling.[1] They are only useful as long as staining takes place.

From time to time, different types of corneal ulcers have been recognised and described, and the following are the chief of them :—

Simple Ulcer.—This may result from a slight trauma, or from the bursting of a phlyctenula. It presents the appearance of a minute and shallow depression, with a grey floor, on the surface of the cornea. There is circumcorneal vascularity, especially at that part of the corneal margin nearest to which the ulcer is situated ; the pupil is apt to be contracted, although iritis is not present ; and there is often a good deal of pain, lacrimation, and photophobia.

Treatment and Prognosis.—The eye is to be bandaged, warm fomentations applied several times a day, and a drop of solution of atropine instilled night and morning. When of phlyctenular origin, stimulation with the yellow oxide ointment is indicated. Cure, with slight opacity remaining, comes about in a week or ten days. But, occasionally, this form of ulcer may pass over to the deep ulcer.

Deep Ulcer.—This is a purulent ulcer, and commences in a purulent infiltration of the cornea. It forms a tolerably deep pit in the cornea, towards its centre, the floor of the

[1] *Deutsche. Med. Wochenschrift*, No. 10, 1892.

ulcer being covered with purulent deposit and detritus, and the corneal tissue immediately surrounding it being somewhat infiltrated with pus. The ulcer is generally round, but it may assume any shape. Hypopyon is often present, and a marked tendency to iritis exists. The pain is usually very severe, violent frontal neuralgia being a common symptom.

This ulcer has no great tendency to spread over the surface of the cornea, but has a very decided tendency to perforate through it. As it does not generally attain wide dimensions, the perforation it may produce is small, and gives rise to a small adherent leucoma, rather than to a staphyloma.

Causes.—This form of ulcer is a frequent one in purulent conjunctivitis, and it may be caused by the lodgment of foreign bodies, and other injuries of the cornea.

Treatment.—If the ulcer be due to a conjunctival process, the latter should be actively treated.

If the cause be other than conjunctival, a pressure bandage, to give support to the ulcer, is important, and periodical warm fomentations are most beneficial. Atropine should be instilled several times daily. Antiseptic applications, too, especially iodoform in finely divided powder, are useful.

Paracentesis of the anterior chamber through the floor of the ulcer is a proceeding always followed by improvement in the condition of the eye, and is very important as a preventive of natural perforation. The actual cautery, too, is in its place here.

Ulcus Serpens (Sæmisch's Ulcer, Infecting Ulcer). This, also, is a purulent ulcer, the characteristic of which is its tendency to extend over the surface of the cornea, especially in some one direction, rather than to strike deep into its tissue. Its position is chiefly central, and it presents a greyish floor, which is more intensely opaque at some places. One part of the margin takes the form of a curve, or of several closely placed curves, and at this place becomes yellowish-

white in colour and somewhat raised, and the floor of the ulcer seems deeper in its neighbourhood. Immediately around the ulcer the cornea is slightly opaque, but farther out it is quite normal.

The degree of pain and irritation varies much, being almost absent in some cases, while in others it is extremely intense. Iritis is apt to come on at an early period, and may pass into irido-cyclitis. Hypopyon is almost always present. The ulcer creeps over the surface of the cornea in the direction of the curved and intensely infiltrated margin. At a still later stage the whole cornea is apt to become infiltrated, and the entire margin of the ulcer to extend, and the anterior chamber becomes quite full of pus. Perforation now takes place, or may do so somewhat earlier. If the perforation be small, an adherent leucoma results ; but, if large, a staphyloma is produced.

Causes.—Ulcus Serpens always has its origin in a superficial corneal abscess (*vide* p. 170), caused in its turn by a trauma, which has produced, it may be, only a slight abrasion of the epithelium. In a large percentage of the cases chronic dacryocystitis is present, and a considerable proportion of them occur in the agricultural population, especially in harvest-time. The investigations of Leber,[1] and others, make it probable that a fungus (aspergillus) obtaining entrance through the loss of epithelium sets up the abscess, which results in this peculiar ulcerative process. This fungus is probably present in the abnormal secretion of the lacrimal sac, or floats in the air during the oats, barley, and wheat harvest.

Prognosis.—From the above description it will be seen, that the process is a very severe one in many instances, and the prognosis bad ; yet, some cases do recover useful, although damaged, sight, under careful treatment, if it has been resorted to in time.

[1] *v. Graefe's Archiv*, xxv., pt. 2, p. 285.

Treatment.—If the case be not severe, atropine, with protection of the eye, may cure in a few days. Here, too, some surgeons prescribe eserine, and I am opposed to its use (p. 115). Warm fomentations are useful; and a pressure bandage, provided there be no dacryocystitis. Antiseptic measures should always be employed, iodoform being the application most likely to prove of use. It may be employed either in the form of a strong ointment (gr. xxx ad ℥j) put into the eye, or it may be dusted on the floor of the ulcer with a camel's-hair pencil. Scraping the floor of the ulcer with a sharp spoon has also been suggested. But it is in all respects wiser to deal with these cases, even the apparently mild ones, actively in the very commencement, by means of one or other, preferably the second, of the two following methods.

Sæmisch's Method consists in division of the ulcer with a Graefe's cataract knife. Cocaine having been applied, the point of the instrument is entered about 2mm. from the margin of the ulcer in the healthy corneal tissue, and, having been passed through the anterior chamber behind the ulcer, the counter-puncture is made in the healthy cornea some 2mm. from the opposite margin of the ulcer. The edge of the knife being then turned forwards, the section is slowly completed. The incision should divide the intensely infiltrated part of the margin in halves. The aqueous humour and hypopyon are evacuated, atropine is instilled, a bandage is applied, and the patient soon gets relief from pain. Every day, until healing of the ulcer is well established, the wound must be opened up from end to end with the point of a fine probe or spatula, the contents of the anterior chamber being thoroughly evacuated on each occasion, and atropine instilled. The result is that, in a vast majority of cases, the progress of the ulcer is arrested, and healing soon sets in. The little operation should not be delayed long, but it may be employed with advantage even in late stages of the process.

But the actual cautery is the most valuable method of treatment for this ulcer. The infiltrated and undermined margin of the ulcer is the part which should be most thoroughly cauterised; but its floor, if much infiltrated, is also to be dealt with. The application of fluorescin just before the use of the cautery is of much value, as it enables the operator to clearly discern the whole of the diseased part requiring cauterisation.

It will not be out of place to describe here a method of treatment which has been introduced by Darier[1] for many affections of the eye, but chiefly for infective ulcers of the cornea, and which has given very satisfactory results not only in his hands, but also in the hands of others, although it has its opponents. This method consists in subconjunctival injections of corrosive sublimate. One-twentieth of a milligramme (0.00005 gramme) is injected under the conjunctiva at a distance of about 1·0 cm. from the corneal margin. As it is a rather painful procedure, cocaine must be first instilled. Some œdema of the conjunctiva and swelling of the lids may be present on the following day, but it soon subsides. The only complication which ever occurs is a slight scar in the conjunctiva. It is well to use an "iridised platinum" needle on the hypodermic syringe, as it can be sterilized each time by passing it through a flame. The injections may be repeated every third or fourth day, according to the amount of reaction.[2]

Rodent Ulcer.—This is a rare and extremely dangerous form of ulcer. It appears as a small—sometimes even pin-head—grey infiltration near the corneal margin, not differing

[1] *Annal d'Oculist*, 1893, t. cix., p. 241; *ibid.*, t. cx., p. 145 (resumé of various opinions); also Gepner, *Centralblatt, f. prak. Augenheilk.*, Jan. 1894.

[2] The other affections in which this treatment has been of service are: injuries of the eyeball (to prevent infection), keratitis diffusa, iritis, chorioido-retinitis, and scleritis.

in appearance from many a harmless infiltration. This rapidly ulcerates. Other similar infiltrations appear in the neighbourhood and at other parts of the margin, and ulcerate. The ulcers do not go deeper than about one-third of the thickness of the cornea. They never penetrate. Before long they begin to heal, but leave an intense cicatrix behind. After a time more such ulcers form inside the position occupied by the first irruption, and these also heal, leaving further opacity. This process goes on until, finally, the whole surface of the cornea has been eaten away, its centre being the last place affected, and then loss of sight is complete. The disease usually comes on in both eyes, although there may be an interval between the onset in each. It attacks decrepit people of over middle life. The progress of the disease is very slow, as many weeks, or even some months, may elapse, before the surface of the whole cornea has been destroyed.

Treatment.—Some of these cases are amenable to the actual cautery, and then its use will arrest the disease and save the eye. But I have seen cases in which this and every other conceivable treatment was tried in vain, and where both eyes were irretrievably lost.

Marginal Ring Ulcer is a rare form, which commences as a clean-cut, or but slightly infiltrated, yet rather deep, ulcer at the corneal margin. Its tendency is to extend along the margin of the cornea; and, in some instances, healing takes place in the older parts of the ulcer, while it is still progressive at the newer parts. It may extend all round the cornea, and finally give rise to complete sloughing of the latter by cutting off its nutrition. This ulcer may result in children from a marginal phlyctenular infiltration (p. 138), but is more common in adults, or in aged people, whose nutrition has fallen very low.

Treatment.—The actual cautery. Paracentesis through the ulcer, eserine having been first instilled. Insufflation of

iodoform. Warm fomentations. A bandage. Quinine, iron, and strychnine internally, with nutritious diet.

Absorption Ulcer (Facetted Ulcer, Superficial Transparent Ulcer) is the term applied to a certain definite superficial ulceration, which is accompanied by but little opacity, and by no vascularisation, and which is usually seated at or near the centre of the cornea, where it presents the appearance of a shallow pit about 2 mm. broad, with rounded margin. If the eye be exposed to cold wind or other irritation, some circumcorneal injection makes its appearance, and the eye waters; but these symptoms soon pass off again. The portions destroyed by the ulcerative process come away in the course of a few weeks, the surface begins to be covered with new epithelium, and reparation of the corneal tissue commences. It takes months for this healing process to be completed; and, often, the defect is never quite filled up, but a small facet is left, which is liable to interfere with vision.

The absorption ulcer does not tend to perforate, nor to spread over the surface of the cornea.

It occurs chiefly in childhood, and probably indicates malnutrition of the general system; some observers, indeed, think there is a close relationship between it and phlyctenular ophthalmia. It is also seen in granular ophthalmia, with and without pannus.

Treatment consists in atropine and protection in the early stages; and the yellow precipitate ointment, when the epithelium has become restored.

Neuro-Paralytic Keratitis.—In paralysis of the Ophthalmic Division of the Fifth Nerve, purulent infiltration and ulceration of the cornea is often observed. It was formerly believed that the fifth nerve had an influence over the nutrition of the cornea, and, hence, that this was a trophic process; but experiment has shown that this is not the case, and that the affection is merely due to the loss of sensation, which renders it possible for foreign substances to remain on

the cornea, unremoved by a reflex motion of the lid. This disease, therefore, cannot be regarded as of neuropathic origin, in the strict sense of the term.

Treatment consists, chiefly, in protection of the cornea by a bandage on the eye; or, by keeping the lids fastened together with a dermic suture.

Infantile Ulceration of the Cornea, with Xerosis of the Conjunctiva, first described by von Graefe,[1] is a very rare affection, of which a few cases came under my care at von Graefe's clinique. It attacks some wretchedly delicate marasmatic children early in the first year of life, making its appearance at, or near, the centre of the cornea. Iritis always supervenes in severe cases. That portion of the bulbar conjunctiva, which is exposed in the palpebral aperture at either side of the cornea, undergoes slight epithelial xerosis, as in functional night blindness due to retinal exhaustion (see chap. xvii.). Sometimes the xerosis of the conjunctiva is absent.[2] Ulceration of the cornea soon comes on, through necrosis of the layers lying over an interstitial infiltration; and this ulceration spreads, until it involves the whole of the cornea, except a very narrow margin. Finally, perforation, with prolapse of the iris, and panophthalmitis, may supervene.

Both eyes become affected, as a rule, although the disease usually attacks one some time before its fellow. The patients almost always die of diarrhœa, pneumonia, etc.

Cause.—Streptococci have been found[3] in the corneal ulcer, and in the conjunctiva; while a general invasion of the vascular system of the whole body is also present. To the latter circumstance are referred the symptoms, which lead to a fatal termination.

---

[1] *A. v. Graefe's Archiv*, xii., pt. 2, p. 250.

[2] Holmes Spicer, *Trans. Ophth. Soc. Un. K.*, vol. xiii., p. 45.

[3] Leber and Wagenmann, *A. v. Graefe's Archiv*, xxxiv., 4, p. 250.

Treatment is, unfortunately, of very little avail; but warm fomentations, and the use of non-irritating antiseptic lotions, etc., are indicated, along with an antiseptic bandage. Such means as may promote improvement of the general system will, of course, be employed.

Herpes Corneæ.—Not only in herpes zoster ophthalmicus, but also in herpes febrilis (or catarrhalis) is a vesicular eruption liable to occur on the cornea. According to Horner, herpes corneæ febrilis is a rather common affection, and, he believed, is often not recognised by ophthalmologists, because it usually first comes under their notice when the secondary ulcers have formed. The following is Professor Horner's description of the disease :—

On the surface of the cornea of one eye is formed a group of clear vesicles, each from 0·5 to 1·0 mm. in diameter, their appearance being accompanied by much lacrimation, but without any swelling of the eyelid. They usually form in a line, which runs obliquely across the cornea, or sometimes in a vertical direction. Now and then they are arranged in trefoil shape, or in a circle. The covering of the vesicles is short-lived, and, as already remarked, the resulting ulcer is that which the surgeon usually first sees. Even it, however, is thoroughly characteristic. On the surface of the clear cornea is an irregular loss of epithelium, along the margins of which may still sometimes be seen the shreds of the late covering of the vesicle. The margin of the region which is bared of its epithelium is dentated, and can only be mistaken for a traumatic loss of epithelium. The latter, however, would never present the peculiar "string-of-beads" appearance. The floor of the loss of substance is formed by the superficial layers of the cornea, and the anæsthesia of the cornea is confined to this place, and does not, as in herpes zoster, extend to the rest of the cornea. The tension of the eye is generally reduced. Under favourable circumstances this loss of epithelium may be rapidly repaired;

although, even then, more slowly than one of equal dimensions, but of traumatic origin. Usually the healing process is slow; and, sometimes, more or less intense opacities form in the area and at the margin of the ulcer, with hypopyon iritis, etc., and the loss of substance becomes deep, with a dentated margin. This more unfavourable course is the result of secondary infection of the ulcer.

The subjective sensations are those of a foreign body in the eye, with lacrimation and photophobia, and are relieved immediately after the bursting of the vesicles.

The vesicular eruption is often regarded as irritation from a foreign body merely; or, occurring in the course of a serious disease (pneumonia, typhoid fever, intermittent fever, etc.), it passes wholly unnoticed, and its relationship to the latter remains unrecognised.

The only affection for which herpes corneæ is likely to be mistaken is phlyctenular keratitis; but the clear elevated vesicles will readily be distinguished from the flatter greyish mass of cells, which form the phlyctene. In herpes there is never—although often in phlyctenular keratitis—a vascularisation of the cornea. The shape of the loss of epithelium after bursting of a herpes vesicle is characteristic. Phlyctenular keratitis is a disease of childhood, while herpes corneæ is rare under puberty.

The derangements of the system in which herpes corneæ febrilis occurs, are naturally those in which herpes febrilis labii, nasi, etc., are found. These are, more especially, the inflammatory affections of the respiratory tract, from an acute catarrh of the Schneiderian mucous membrane, to a severe pneumonia. On two occasions, with an interval of three years, Professor Horner saw herpes corneæ occur in the course of an attack of pneumonia in a boy. In just such cases, herpes on the lips, ala nasi, external ear, and eyelid of the same side are found; and, in a case of double pneumonia in an adult, occurred the only binocular herpes

corneæ which Professor Horner had seen. He explicitly states, that he had seen herpes corneæ in connection with whooping cough; and, often, with intermittent and typhoid fevers.

But primary herpes corneæ—*i.e.* unconnected with any other disease—is occasionally met with; and some patients are liable to recurrent attacks of it. It is accompanied by severe neuralgia in the frontal and temporal regions, and pain on pressure of the supraorbital notch may be present. There is much lacrimation. The upper lid is red and swollen. The bulbar conjunctiva, especially around the cornea, is much infected, and there may be a few vesicles on it. Over the surface of the cornea, but sometimes confined to some one district of it, there are a number of minute vesicles, some shreds of epidermis—the remains of ruptured vesicles—and round greyish-white superficial infiltrations, not larger than a pin's head. The mucous membrane of the nostrils is also apt to be attacked, causing swelling of it, with much secretion, and the formation of scabs.

Treatment, at an early stage, before the vesicles have burst, or the loss of substance has become infiltrated, consists in protection of the eye; and, when infiltration has set in, in disinfection with protection. If the vesicles give great pain, they may be ruptured by dusting a little calomel into the eye, or by brushing it with a camel's-hair pencil wet with solution of boracic acid, after which a well-fitting antiseptic bandage is applied. Cocaine is valuable in these cases for relief of the pain. Atropine and warm fomentations should also be employed, and a weak Pagenstecher's ointment is of use in some cases. Where the nostrils are affected, weak sublimate, or other antiseptic, washes should be applied to the Schneiderian mucous membrane.

Filamentous Keratitis (Fädchen-Keratitis).—Of this form of keratitis I have as yet seen but one case. It may occur with or without superficial injury to the cornea. Its name

is due to the fine threads, like twisted spun-glass, several of which hang from the surface of the cornea, and give the condition its characteristic appearance. These threads never reach a length of more than 3 or 4 mm.

Different views are held as to the mode of origin of the threads. Fischer and Uhthoff[1] have observed, that small vesicles, with clear or turbid contents, appear in groups upon part of the cornea, then burst, and from the centre of each resulting depression a thread hangs out. The onset of the vesicles is accompanied by much pain and photophobia, and probably has its cause in some affection of the fifth nerve. The duration of an attack is usually short, but there may be several relapses at brief intervals, and finally the process ceases without permanent damage to the cornea. These same authors hold, that the threads are composed of the peculiar fibrinous contents of the vesicles. But it has been proved now beyond doubt by the investigations of Hess[2] and Nuel[3] that the threads are composed of twisted proliferating epithelial cells, each thread ending in a bulbous enlargement, caused by degeneration of the epithelium. A peculiar diseased condition of the corneal epithelium precedes the formation of the vesicles and threads. Leber now admits the epithelial origin of the filaments, although he originally believed them to be fibrinous products.

Treatment.—Protection of the eye with a bandage. Atropine. Yellow oxide of mercury ointment put into the eye. Warm fomentations.

Bullous Keratitis.—Bullæ very rarely form on the cornea. They are never the primary condition, but depend on an interstitial diseased process in the cornea. This latter may itself be a primary disease; but more commonly it is secondary to

[1] *Bericht d. Ophthal. Gesellsch.*, 1889.

[2] *A. von Graefe's Archiv*, xxxviii., part 1, p. 160; *ibid.*, xxxix., part 2, p. 199.

[3] *Archiv d'Ophthalmologie*, xiii., 4, p. 193.

deep changes in the eye, such as absolute glaucoma, irido-cyclitis, etc. The formation of a bulla is attended by much pain and photophobia, which disappear, as soon as the bulla ruptures. One, or more than one, bulla may form at a time. After a day or two they rupture, and their walls then hang in shreds from the surface of the cornea, and the seats of the bullæ present shallow depressions. These losses of substance heal without leaving any permanent opacity. After an interval of days or weeks, another crop of bullæ appears, and runs the same course.

Treatment.—The bullæ should be opened, and their walls snipped away with a scissors, and a bandage applied. The recurrent attacks may cease after a length of time; but, so far as treatment can influence them, it can only be done by relieving the process in the cornea which gives rise to them. If it be a primary process, warm fomentations, atropine, and a bandage, with remedies directed to correction of any fault in the general state of the health which may exist, are suitable; or if, as is more common, a deep ocular process (glaucoma, etc.) be the cause, the recognised treatment for this latter must be adopted.

Dendriform (*δένδρον, a tree*) **Keratitis.** This is a rare affection, to which attention was first drawn by Hansen Grut, of Copenhagen. It is a very superficial and chronic ulceration, with but little infiltration of its margins or floor, and presenting the appearance of a fine groove on the cornea. It spreads, chiefly, over the central region of the cornea, by throwing out branches on either side. The pain and irritation are sometimes severe, and again but slight, or quite wanting. Some permanent opacity often remains, when cure has been effected.

The Cause has not been definitely ascertained, but the peculiar progress of the affection renders it almost certain that some special fungus is engaged.

Treatment.—Scraping with a sharp spoon, with the subse-

quent application of 1 in 1,000 solution of corrosive sublimate to the cornea, is recommended by some, and the actual cautery is of great use. But I can strongly recommend the application of absolute alcohol, which I find affords a certain and rapid cure.[1] I soak a bit of lint in the alcohol, and scrub the surface of the cornea with it. This may require to be repeated once or twice, at intervals of a day or two.

(*b*) NON-ULCERATIVE INFLAMMATIONS OF THE CORNEA.—**Abscess.**—This affection is on the borderland between the ulcerative and non-ulcerative inflammations of the cornea; for in one case it will result in an ulcer—usually the ulcus serpens—while again it will run its course without ulceration. The abscesses which are seated in the more superficial layers are those which go on to ulceration; those in the deeper layers are less likely to do so.

Abscess differs from infiltration, in that the pus which forms it destroys the true corneal tissue—the fibrillæ and fixed corpuscles—and does not merely lie between them.

Signs and Symptoms.—The appearance presented is that of a yellowish circumscribed opacity, more intense at its margin than at its centre, seated at or near the middle of the cornea, and surrounded by a light grey zone. It is usually round in shape, but, when situated near the edge of the cornea, it is apt to be crescentic. The surface of the cornea just over the abscess is at first a little elevated over the general surface, but later on becomes flattened, owing to a falling-in of the normal layers anterior to the abscess; and the epithelium of the flattened part has a dull, breathed-on

[1] I can corroborate Mr. Swanzy's statement as to the efficacy of absolute alcohol. I have had a case of dendritic keratitis which lasted three months, and in which various remedies were tried, including freshly prepared chlorine water, without success, until a single application of alcohol quite stopped it. The rubbing was done very thoroughly, and the only complication was some œdema of the upper lid, which disappeared in a few days.—L. W.

look. The rest of the cornea may also lose its brilliancy, although in a much less degree. Hypopyon, and iritis, are constant attendants upon corneal abscess. There is much injection of the conjunctival and ciliary blood-vessels. Severe pain in and about the eye, and blepharospasm, are common. Occasionally, a corneal abscess will be attended by but little pain, or other irritation.

Progress.—The abscess spreads through the cornea, usually in some one direction; and this direction is indicated by the yellowish opacity being more intense at the advancing side of the abscess. Before long, if the abscess be superficial, the layers of cornea covering it come away, and the condition is changed into that of the ulcus serpens, already described. The deeper abscesses spread through the cornea more or less widely, and ultimately become absorbed, without having caused ulceration. But even these abscesses leave considerable opacity behind. Of the two, the process which ends in ulceration is the more common.

Etiology.—Abscess is the result of infection of the cornea with pyogenic organisms, which reach it either from without, through some traumatic loss of substance of the corneal epithelium, or from within, by the agency of the blood. The micro-organisms, which are introduced through a superficial loss of substance, may either have been present on the foreign body which produced the injury, or they may have been present in the conjunctival sac. Infection through the blood is occasionally seen in some acute exanthematous diseases, such as scarlatina, measles, and smallpox; more especially in the latter, in its convalescent stage.

Treatment.—Atropine, warm fomentations, and a bandage. But, if these mild measures do not in a day or so arrest the progress of the abscess, resort must be had to the actual cautery.

Diffuse Interstitial, or Parenchymatous, Keratitis.—This affection occurs, most commonly, between the ages of five and fifteen.

It commences at some one part of the margin as a light greyish opacity, accompanied by slight injection of the ciliary vessels. The rest of the corneal margin soon becomes similarly affected; and then, gradually, the opacity extends concentrically into the cornea, or does so by sending in processes which afterwards become confluent. In this way the whole cornea becomes affected by degrees; and its epithelium acquires the breathed-on, or ground-glass, appearance, which is seen, also, in acute glaucoma. The opacity lies in the deep layers of the true cornea, and is slightly more intense in spots here and there. It is sometimes only a very light cloud, while again the cornea may be so opaque as to render the iris quite invisible. When the whole cornea has become opaque, it begins to clear up at the margin, and the central portion becomes even more opaque than the margin had ever been: a fact which shows that the very cells which entered the cornea at its margin have advanced to its centre. The clear margin gradually increases in width, until only a rather intense central opacity is left. This central opacity slowly breaks up, and becomes absorbed, but not always completely; and then considerable and permanent impairment of vision may remain. Occasionally, the opacity commences in the centre of the cornea, and the margin remains clear all through. Again, a very intense vascularisation (the so-called "salmon patch") may gradually occupy the whole cornea, following the progress of the opacity. There is no tendency to ulceration in this affection of the deep layers.

The affection is often accompanied by a good deal of pain and blepharospasm, especially in the vascular forms. It is very liable to be complicated with iritis, or even with iridocyclitis, and herein lies its greatest danger. The iritis is usually of the serous form, but may be plastic, and opacities in the vitreous humour often result from it. Exudative chorioiditis, and optic neuritis, also, very occasionally com-

plicate it. The tension of the eyeball in these cases may be much diminished for a time.

The acute stage of the disease lasts from six to eight weeks, or longer. But the entire process may not be completed for many months, and, in one case which I saw, the opacity did not begin to clear away for eleven months after the cornea was first attacked, the whole process extending over a period of two years.

Both eyes invariably become affected, although not always at the same time, the second eye being often not attacked until the inflammation in the first has made some progress, or, perhaps, not until it has undergone cure. It is important to acquaint the patient, or his parents, with the likelihood of this course of events, in the very commencement of his treatment.

Diffuse interstitial keratitis occurs also in adults, but I have never seen it in persons of over thirty or thirty-five years of age. These adult cases present a greater variety of type than those in children, and, on the whole, they are less severe in character. Most commonly, one eye alone becomes diseased, the degree of opacity is often slight, the extent of diseased cornea limited, the duration of the process comparatively short, and the complete clearing-up relatively frequent.

Causes.—The affection is more common in girls than in boys; and, most frequently, appears during second dentition, when the upper incisors are being cut; or, at puberty. It depends upon some serious derangement of the general nutrition; and this, in over 50 per cent. of the cases, is inherited syphilis, a fact which was first pointed out by Mr. Jonathan Hutchinson. The children are generally thin, anæmic, and of stunted growth; with flat nose, cicatrices at the angles of the mouth, often more or less deaf, and the peculiarities of the incisor teeth, so well known from Mr. Hutchinson's description, are present in about one half of the cases.

Occurring in adults, the affection is rarely due to inherited

syphilis, although acquired lues may sometimes be taken as its cause; while, again, it will often be impossible to assign any origin for it, other than the universal one of exposure to cold, etc. Von Hippel[1] is of opinion that some cases are due to tubercular disease. He found microscopic appearances, very suggestive of tubercle, in the iris and deeper parts, in an eye with interstitial keratitis.

Prognosis.—In children, in view of the possibility of an incomplete clearing of the cornea, and the irregularity of its surface which the process may cause; as well as of the serious complications liable to supervene, and which may completely annihilate vision; the prognosis must be guarded—although by no means hopeless—in those cases where the opacity is very intense, or where there is much vascularity. Yet, in the milder cases, a very favourable prognosis may be given. I have never seen the affection recur, but it is said to do so very rarely.

In adults, as stated, the prognosis is much more favourable.

Treatment.—In the early stages no irritants should be locally applied. Atropine is important for the prevention of iritis, or of posterior synechiæ; and the use of warm moisture, in the form of poultices or fomentations, promotes vascularisation, and hastens absorption of the cellular elements, which form the opacity. When the acute stage is ended, the yellow precipitate ointment may be employed with benefit, for stimulating the absorbents to carry off the remains of the opacity. Massage may be used with advantage in both stages, to disperse the infiltration. In the severe cases I would advise a course of mercurial inunctions continued for several weeks, care being taken not to allow stomatitis to exceed very moderate bounds. In mild cases a tonic plan of treatment, with iodide of iron and cod-liver oil, is the most suitable.

In adults, where it is desirable to use mercurial treatment,

[1] *Centralbl. f. Augenheilk.*, June 1893, p. 174.

a good method is the hypodermic injection of perchloride of mercury $\frac{1}{40}$ to $\frac{1}{20}$ gr. once a day. From this I have had satisfactory results, but mercurial inunctions also answer well, and are less painful.

Counter-irritation, in the form of blisters to the temple, or a seton in the scalp, is extensively employed by some surgeons. I have never adopted this treatment, as I doubt its value, and am loath to add a worry to the troubles inseparable from so wearisome a disease.

Keratitis Punctata is the name commonly given to a condition which occurs in cyclitis, in irido-cyclitis, and in sympathetic ophthalmitis (chap. x.). It is never a primary disease of the cornea.

It is due to the deposit of minute beads of lymph on the membrane of Descemet, which gives to the affected part of the cornea a finely-dotted appearance. The lymph is usually found only on the lower quadrant of the cornea—because it gravitates to the lowest part of the anterior chamber—in a triangular space, of which the base is at the corneal margin, while its apex is directed towards the centre of the cornea. This triangular shape is the result of the motions of the eyeball, which throw the lymph beads against the cornea. In some cases the spots are scattered irregularly over the whole surface of the posterior elastic lamina.

When the process, which gives rise to this condition, passes off rapidly, the cornea is restored to its normal state. But, when the primary disease is chronic, the nutrition of the true cornea, in the triangular space corresponding to the deposit of lymph, is apt to be interfered with—by reason of degeneration of the endothelium of Descemet's membrane, which protects the cornea from the aqueous humour—so that it becomes intensely, and permanently, opaque.

Some authors do not use the term keratitis punctata for the foregoing condition, but reserve it for some cases of interstitial keratitis which present a spotted or dotted appearance.

Sclerotising Opacity of the cornea sometimes complicates scleritis, affecting the cornea in the neighbourhood of the scleral affection, but not extending more than 2 to 3 mm. into the cornea, except in very severe cases. It is an intense white opacity, situated in the true cornea, and is apt to remain as a permanent opacity, even when the scleritis undergoes cure. In such cases of sclero-keratitis, iritis is often present.

Treatment.—Warm fomentations, massage, and the treatment of whatever diathesis (rheumatism, syphilis) may be taken as giving rise to the scleritis.

Ribandlike Keratitis (Transverse Calcareous Film of the Cornea; Calcareous Film of the Cornea).—This is an alteration which occurs chiefly in the corneæ of eyes destroyed by severe intraocular processes, such as iridocyclitis, sympathetic ophthalmitis, glaucoma, etc. It occupies that transverse strip of the cornea, which is uncovered in the commissure of the eyelids during waking. It usually commences on the inner margin of the cornea, but soon appears at the outer margin, and advances from each direction towards the centre, where the two sections join. It presents the appearance of a greyish-brown opacity, with, in many, but not in all, cases, white calcareous deposits in and under the epithelium. Magnus[1] points out that in blind eyes which are constantly rolled upwards, the opacity is found, not in the central transverse section of the cornea, but in its lower third; and from this circumstance he argues, that the chief factor for its production is exposure of the part affected. He believes, moreover, that so large a proportion of the affected eyes having suffered severely in their general nutrition, indicates that the opacity is a further development of this malnutrition. He proposes for the affection the name Keratitis trophica.

[1] *Klin. Monatsbl. f. Augenheilkunde*, February 1883, p. 45.

Ectasies of the Cornea.

Staphyloma Corneæ is the result of a perforating ulcer of the cornea. This, having healed, may present a weak cicatrix, which becomes bulged forwards by even the normal intraocular tension (Figs. 67 and 68). If the iris be not involved in this cicatrix, the anterior chamber will be made deeper (Fig. 68).

Staphyloma corneæ, in which the iris is involved, is probably a more common condition than the above.

When the ulcer is large, a correspondingly large portion of iris is liable to become prolapsed into it, and to form a

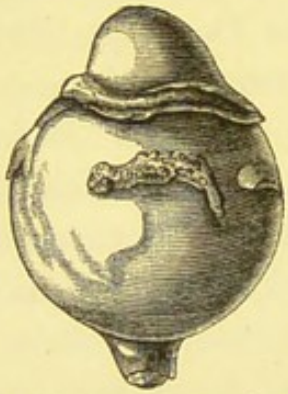


Fig. 67. (*Pagenstecher.*)

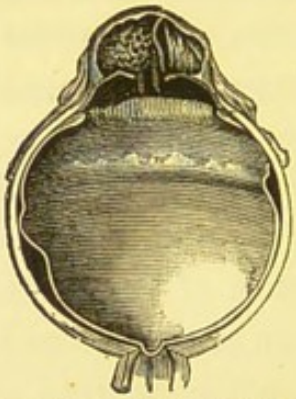


Fig. 68. (*Pagenstecher.*)

bulging mass outside the eye. This may burst and collapse, and a flat cicatrix may be formed. Or, if it do not rupture, it may form what is termed a partial staphyloma of the cornea and iris, the latter becoming consolidated by the formation of a layer of connective tissue over it.

If the whole, or a very large part, of the cornea be destroyed by an ulcer, the iris is completely exposed. It soon begins to be covered with a layer of lymph, which develops into an opaque cicatricial membrane. Should this not be strong, the normal intraocular tension is sufficient after a time to make it bulge; or, increased intraocular tension may arise, in consequence of further changes within the eye, and then bulging of the pseudo-cornea more surely comes on, and the condition is termed total staphyloma of the cornea. Sometimes a total

staphyloma has a lobulated appearance, owing to the pseudo-cornea having some fibres stronger than others, and hence the name given to the condition, from σταφυλή, a bunch of grapes. Such staphylomata are apt to gradually increase to a very large size.

Treatment.—In cases of partial staphyloma, where a clear portion of the cornea remains, an iridectomy is frequently indicated for the reduction of the tension—so that further bulging may be arrested—as well as for the sake of the artificial pupil, which may improve sight, in cases where the normal pupil is obliterated by corneal opacity. When, sight having been lost, the staphyloma is very bulging, or when total staphyloma is present, enucleation of the eyeball, or one of the following operative measures, must be adopted.

Abscision.—A Beer's cataract knife being passed through the base of the staphyloma, with its edge directed upwards, the upper two-thirds of the staphyloma are separated off, while the remaining third is detached by means of a scissors. If the lens be present, it must now be removed. The wide opening becomes filled up with granulations, and cicatrises over.

In de Wecker's [1] method the opening is closed with conjunctival sutures. He begins the operation by separating the conjunctiva all round the margin of the cornea, and by then loosening it from the eyeball nearly as far back as its equator. Four sutures (*a*, *b*, *c*, *d*), of different colours, are then passed through the conjunctiva about 2 to 3 mm. from the margin of the wound, as represented in Fig. 69. In order to keep the field of operation clear, the ends of two of these sutures are laid over on the nose, while the others are laid over on the temple. The staphyloma is now abscised, and the sutures drawn together and tied. The conjunctival scar, de Wecker states, can be tattooed in the centre at a later period, and by this means the wearing of an artificial

[1] *Chirurgie Oculaire*, p. 188.

eye made unnecessary. De Wecker has also recommended repeated puncturing of the sclerotic behind the ciliary region for the purpose of diminishing the size of the globe.

The foregoing, and other, methods of abscision are only applicable, where the tension is either low or normal. If it be high, the liability to intraocular hæmorrhage during the operation makes enucleation, evisceration, or Mules' operation, more suitable proceedings. Indeed, I, and probably

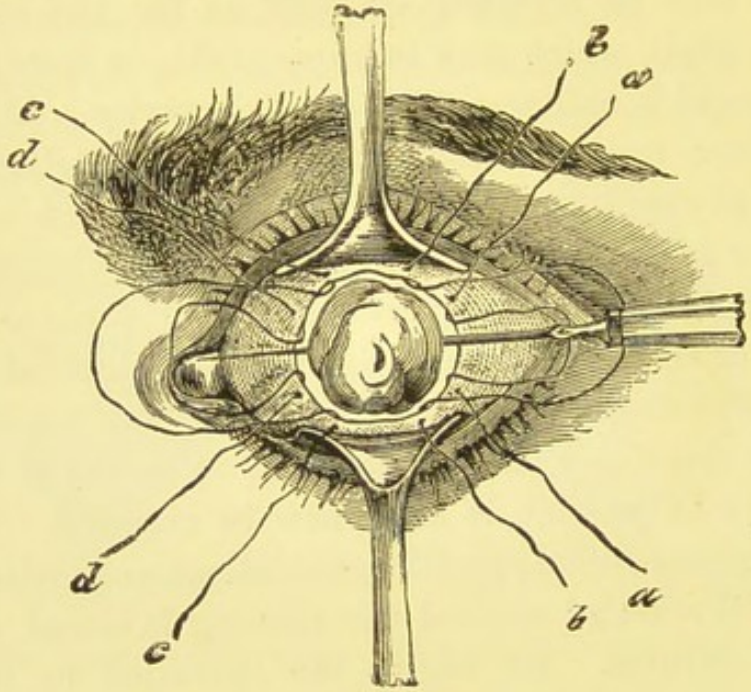


FIG. 69.

most surgeons, would now employ one of the two latter operations in all these cases.

Evisceration (Exenteration) was proposed about the same time by Professor Graefe, of Halle,[1] to prevent death from meningitis after the removal of suppurating globes, and by Mr. Mules,[2] of Manchester, chiefly to take the place of enucleation in cases of sympathetic ophthalmitis. There are some who are opposed to its employment in those cases; but, for staphyloma of the cornea, it cannot meet with any such opposition.

[1] *Centralbl. f. Augenheilk.*, 1884, p. 378. [2] *Ibid.*, 1885, p. 32.

The cornea is removed by making an incision with a Graefe's knife, so as to include one half of the corneo-scleral margin, and then completing the circumcision with scissors. All the contents of the globe are then evacuated by means of Mr. Mules' scoop, care being taken to remove the chorioid unbroken, by carefully peeling it from the sclerotic margin backwards, until it is only held at the lamina cribrosa. The scoop is then used to lift the separated unbroken chorioid, and its other contents, out of the globe.

Finally, the margins of the sclerotico-conjunctival wound are drawn together with a few points of suture. The whole proceeding should be done with strict antiseptic precautions, chief among which is the free use of irrigation with a 1 in 5,000 solution of corrosive sublimate, before, during, and after the operation, the interior of the globe being most carefully washed out with the solution in a full stream. The result is a good and freely movable stump for the application of an artificial eye.

Mules' Operation.—This proceeding, a modification of the foregoing, was also proposed by Mr. Mules[1] for cases of threatened sympathetic ophthalmitis, and, like simple evisceration, has not yet met with universal acceptance in those cases. Its object is to provide a still better stump for the artificial eye, by the insertion into the scleral cavity of a hollow glass ball, called an "Artificial Vitreous Humour." It is performed as follows:—

The cornea is removed—the conjunctiva having first been freed from the scleral edge towards the equator of the eyeball—and the contents of the eyeball evacuated, as in simple evisceration. The opening is now enlarged vertically, to admit of the introduction of one of the glass spheres. This introduction is best effected by means of a special instrument designed for the purpose by Mr. Mules. The spheres are

[1] *Trans. Ophthal. Soc.*, vol. v., p. 200.

made[1] in several sizes to suit different cases, and it is well not to use the largest which will fit into any given eye. The margins of the sclerotic opening are now united vertically by some points of interrupted suture; for which purpose I prefer silk to catgut, as the latter is apt to undergo absorption before complete union has taken place. The conjunctival opening is then closed, by another set of sutures placed at right angles to the sclerotic line of closure. Similar antiseptic precautions are required as in simple evisceration, and care must be taken that all bleeding in the cavity has ceased before the glass sphere is inserted. Before the lids are closed, the anterior surface of the globe is well covered with powdered boric acid. A firm antiseptic bandage is applied. I do not dress the eye for forty-eight hours, and subsequently once every twenty-four hours, using the corrosive sublimate solution freely, and boric acid powder. There is generally some reaction, consisting of chemosis, swelling of the eyelids, and pain, and sometimes these symptoms are very marked, especially if rather too large a sphere have been employed. In the course of a week or so this all passes off, and a very perfect stump is obtained.

To prevent excessive reaction, Mr. Mules burrows into the orbit at the outer side, so that the points of the scissors may penetrate well beyond the back of the globe, and then introduces deeply a drain of gold wire, such as is used by dentists, bringing it out between the lids at the outer canthus. An icebag is applied. The drain is left in about three days.

The danger that the glass sphere may get broken by a blow upon the eye has been put forward as an objection to this method. No doubt it is an accident which may occur, and would then necessitate the enucleation of the eye; but no case of the kind has as yet been recorded, although the operation has been in use for ten years. Silver spheres, instead

[1] By Messrs. Armstrong, of Deansgate, Manchester.

of those of glass, have been sometimes employed to obviate the danger referred to. I practise this method a great deal, and I can heartily recommend it. The only trouble I have had with it is, that sometimes the sclerotic opening does not close well, and the glass ball has to be removed. The case then becomes one of simple evisceration.

Conical Cornea, or **Keratoconus.**—In this the cornea is altered in shape to that of a cone. The change is due to a gradual and slowly advancing atrophic process in the cornea, especially at its centre, in consequence of which the normal intraocular tension acts on it, so as to distort it into the form represented in Fig. 70. Tweedy [1] has shown that there may be some congenital weakness in the centre of the cornea as the result of its mode of development. The cornea remains clear, except sometimes just at the apex of the cone, where a slight nebula may be present. The condition is easy of diagnosis in its advanced stages by mere inspection of the cornea, especially in profile, but in its commencement it may not be so.

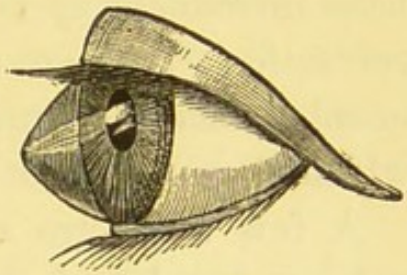


FIG. 70.

In the early stages, when the light is thrown on the cornea from the ophthalmoscope mirror, as for retinoscopy, the corneal reflex will be noticed to be smaller at the centre, owing to the greater curvature there. Moreover, a dark shadow, circular or crescentic in shape according to the incidence of the light, appears between the corneal margin and centre; and, finally, when the fundus is examined its details will be seen distorted.

The process begins in early adult life, progresses slowly, never leads to rupture or ulceration of the cornea; and, finally, after many years, ceases to progress, but does not undergo cure. Both eyes are apt to become attacked, one

[1] *Trans. Ophthal. Soc. Un. K.*, vol. xii., p. 67.

after the other. The disturbance of vision is very great, owing to the extreme irregular astigmatism produced.

Treatment.—In the early stages, or in slight cases, an improvement in vision may be obtained by means of concave spherical, or sphero-cylindrical, glasses; for, as is evident, the change in shape of the cornea must cause the eye to become myopic. The refraction of the central portion of the cornea may be ascertained by retinoscopy, with the aid of a stenopæic disc in the trial frame, as recommended by Mackay.[1] At a later period these glasses are of little use. Hyperbolic lenses have been employed, but, although they may raise the acuteness of vision, there are obvious difficulties in the way of the practical every-day use of them. A stenopæic slit renders assistance in some cases.

Glass shells, which are known as "contact glasses," have been introduced by Fick for the temporary relief of irregular refraction; they are worn in contact with the eye, and may enable some patients to work for hours at employments which they could not otherwise carry on.

A few cases are reported in which the keratoconus was much reduced, and vision greatly bettered, by instillations of eserine and the application of a pressure bandage continued for several months.

But it is upon operative measures we must chiefly rely in this affection, for any improvement in sight.

Von Graefe's Method consists in flattening the cornea by the production of an ulcer on the apex of the cone, and the resulting cicatricial contraction. From the surface of the cornea, a little to one side of the apex of the cone, a morsel of corneal substance is removed with a cataract knife, care being taken not to open the anterior chamber. On the second day after this proceeding, the wound is touched with mitigated lapis (solid), and this is repeated every third day

[1] *Ophthal. Review*, Dec. 1893, p. 317.

for a fortnight or three weeks. Paracentesis of the anterior chamber is then performed through the floor of the ulcer, and the aqueous humour is evacuated every second day for a week, after which the healing process is allowed to take its course. A bandage must be worn during the whole course of the treatment. Finally, when the contraction and consequent flattening are completed, a narrow iridectomy may be necessary, in consequence of the central, or almost central, and rather intense corneal opacity.

In Bader's Method a small elliptical flap of the cornea at its apex is removed, and the margins are brought together by one or two fine sutures. The sutures are omitted by many surgeons as useless, and as liable to cause irritation. Opinion is divided as to whether the ellipse should lie vertically or horizontally in the cornea. Anterior synechia takes place in a large number of the cases, and a subsequent optical iridectomy is always required. I have myself no experience of this operation, but it is said to be attended with unusual risk of suppuration of the cornea, going on to destruction of sight.

Sir William Bowman's Method consisted in cutting a disc on the apex of the cornea with a small trephine, and then severing this disc with forceps and cataract knife. Cicatrisation of the wound produces the desired flattening of the cone. Septic infection is here also a danger, although it has not come under my own observation.

I have myself, in one case, employed the electro-cautery to produce the desired loss of substance on the apex of the cone, but I am not as yet in a position to speak of the ultimate result. I believe that others have used the electro-cautery with good result for sight. The proceeding is free from all risk of septic infection.

With the same object some surgeons have had recourse to Multiple Puncturings of the apex of the cone with a fine cataract needle. The summit of the cone is transfixed from

three to six times at each sitting, and this may be repeated at intervals of two weeks or more. The first effect of the punctures is to allow some of the aqueous humour to escape, and then the eye is firmly supported with a bandage. The pupil is kept under the influence of eserine. Eventually, a net-work of cicatricial tissue forms, which flattens the cone without giving rise to much corneal opacity.

Tumours of the Cornea.

Primary tumours of the cornea are extremely rare. Epithelioma and sarcoma have their origin not in the cornea, but in the limbus conjunctivæ (p. 132). Dermoid tumours are usually seated partly on the conjunctiva and partly on the cornea (p. 131). Yet a very few cases of papilloma, epithelioma, and fibroma are recorded as taking their origin in the cornea. Corneal cysts also occur.

Injuries of the Cornea.

Foreign Bodies in the Cornea, such as morsels of iron, stone, coal, etc., are amongst the most common accidents of the entire body. The pain caused by these foreign bodies is very considerable, as may be imagined, when the rich nervous supply of the cornea is remembered.

The dangers which may follow on the presence of a foreign body in the cornea depend, partly upon the infection or non-infection of the foreign body, and partly upon the depth at which it is buried in the cornea. The deeper a foreign body lies, the more difficult will be its removal, and the greater must be the laceration of the cornea caused by its removal. A foreign body which carries infection upon it, will be more likely to set up serious inflammatory reaction, than one which is aseptic or nearly so. For this reason, it is important to ascertain, if possible, the origin of the foreign body, although an apparently aseptic origin must not set all suspicion on this point at rest.

Many foreign bodies are so small as to defy detection, until the cornea is searched with the oblique light; an aid which should always be made use of, whenever the symptoms, or history, in the remotest way suggest the presence of a foreign body.

A foreign body which lies only in the epithelium, or in the superficial layers of the cornea, is easily removed. The eye having been thoroughly cocainised, the patient is seated, and leans his head against the chest of the surgeon, who stands behind him. With the index-finger of the left hand the surgeon then lifts the upper lid of the injured eye, pressing the margin of the lid upwards and backwards, while with the second finger he depresses the lower lid in a similar manner; and between these two fingers he can, to a great extent, restrain the motions of the eyeball. The foreign body is now to be pricked out of the cornea with a special needle, with as little injury of the general surface as possible, the patient all the while directing his gaze steadily at some given point. If the foreign body be deep in the layers of the cornea, it must be dug out, as it were; and a minute gouge is made for this purpose.

Care must be taken not to infect the cornea in the removal of a foreign body, and consequently thorough antiseptic precautions must be taken. After the foreign body is removed the place where it was seated should be washed with a 1 in 5,000 solution of corrosive sublimate. A bandage is worn until the epithelium is regenerated—*i.e.* for several days.

Every surgeon and general practitioner should possess the two small instruments required for the removal of superficial corneal foreign bodies, and should understand the use of them.

The magnet is of no use whatever for the removal, even of superficially seated, foreign bodies of steel or iron in the cornea.

Sometimes a foreign body in the cornea will be so long as to protrude somewhat into the anterior chamber, and there

is danger that in the attempts at removal it may be pushed farther on, and fall into the anterior chamber. Here it is necessary to pass a keratome through the cornea, and behind the foreign body, so as to provide a firm base against which to work; or, the keratome may be made to push the foreign body forwards.

The wing-cases of small beetles, and scales of seeds, may get into the eye, and adhere to the cornea by their concave surface for several days.

Simple Traumatic Losses of Substance of the surface of the cornea, involving the most anterior layers of the true cornea, or perhaps merely the epithelium, are very common, from rubs or scratches with branches of trees, finger-nails, etc., etc. These injuries heal readily by protecting the eye with a bandage; but, when neglected, or if septic matter have been introduced when the injury occurred, or if it be present in the conjunctiva or lacrimal sac, these losses of substance are capable of forming the starting-point of corneal abscess (p. 169), ulcus serpens (p. 157), etc.

Opacities of the Cornea.

Nebula. Macula. Leucoma.—These terms are applied to opacities in the cornea, of varying degrees, the result of some diseased process, or consequent upon an injury. The first term is used for very slight opacities, often discoverable only with oblique illumination. Macula indicates a more intense opacity, recognisable by daylight. Leucoma is a completely non-translucent and intensely white opacity, the result always of an ulcer, which has destroyed most of the true corneal tissue at the affected place; indeed, it is often the result of an ulcer which has eaten its way through the cornea. In these latter cases the iris may have become adherent in the corneal cicatrix, and then the term leucoma adhærens is employed.

Very often, eyes with a nebulous condition of the cornea of

old standing are myopic. It is probable that this myopia is produced by the habitual close approximation of objects to the eye, owing to the diminished acuteness of vision from the opacity of the cornea.

Treatment.—Little or nothing can be done to reduce these opacities. In slight and fresh cases, massage may render them less intense.

In nebulous cornea a stenopæic apparatus often improves the sight. This consists of a metal plate with a small central hole or slit, which is placed before the patient's eye in a spectacle frame. By this arrangement a large portion of the rays which pass through irregular parts of the cornea, and which merely confuse the sight, is cut off. Where myopia is present, the suitable concave glasses for distant vision should be prescribed.

The Operation of Tattooing was first proposed by de Wecker, and is a valuable proceeding for improvement of the appearance of the eye in cases of leucoma.

But it is also an extremely useful method for the improvement of the sight in certain cases of nebula of the cornea, where the nebula occupies only part of the pupillary area of the cornea. In these cases much disturbance of sight is caused by the dispersion of the light which makes its way through the nebula; and when, by tattooing the scar, all light is prevented from getting through, brighter and distincter vision is enjoyed with the part of the cornea, opposite the pupil, which is absolutely clear.

In the case of a leucoma, either the whole surface of the leucoma may be tattooed, or only part of it; *e.g.* its centre, in order to represent a pupil.

The material used is fine Indian ink, rubbed into a very thin paste. The eye having been cocainised, the leucoma is spread over with this paste, and then covered with innumerable punctures by means of de Wecker's multiple tattooing-needle, each stab of which carries into the corneal tissue

some of the black pigment. The coloration continues sufficiently intense for some months, but then often begins to get pale; owing, probably, to the pigment falling out of the punctures. A better method of tattooing, by which the pigmentation lasts longer, is performed with de Wecker's single grooved needle. The pigment is placed in the groove of the instrument, which is then passed into the true cornea, a long canal being made in a plane parallel to its surface. On withdrawal of the needle the pigment remains behind. A large number of such canals must be made in close proximity to each other, until the desired intensity of colour is obtained.

In cases where the whole cornea is leucomatous, and, consequently, where no restoration of sight can be obtained by means of an artificial pupil, *Transplantation of a Portion of Clear Cornea* from a rabbit's eye, or from a freshly enucleated human eye, has been repeatedly performed by ophthalmologists in various parts of the world. Very many of these operations have been perfectly successful in a surgical sense, *i.e.* in so far as the healing-in of the transplanted flap was concerned; but, with a few exceptions, they all ended in disappointment, in consequence of the flap not retaining its transparency. In the course of a week or two, the transplanted portion invariably becomes as opaque as the leucoma had been before. The mode of proceeding consisted in removing a portion of the leucoma with a trephine, and then, with the same instrument, cutting a disc out of the clear cornea to be utilised, and inserting it into the opening in the leucoma.

Various theories were formed to account for the occurrence of the opacity in the transplanted flap, but into all of these it is unnecessary to enter. Von Hippel[1] came to the conclusion that the onset of the opacity was due to the entrance

[1] *Bericht der Ophthal. Gesellschaft zu Heidelberg*, 1886, p. 54.

of the aqueous humour into the substance of the cornea, owing to the solution of continuity in its posterior epithelium; Leber's experiments[1] having shown that, unless this epithelial layer be intact, the transparency of the cornea cannot be maintained. Von Hippel, acting on this theory, applied a trephine to the leucoma as deep as the posterior elastic lamina, and then dissected off the superficial layers contained within the ring, leaving only the posterior elastic lamina and posterior epithelium. With the same trephine he then excised a disc of its entire thickness from a rabbit's cornea, and applied it to the wound. Iodoform was dusted over this, and a bandage applied. Healing took place readily, and, twenty months afterwards, the flap continued transparent, and vision = $\frac{20}{200}$. Von Hippel has had some other successful cases.

Sclerotising Opacity of the cornea sometimes complicates scleritis, affecting the cornea in the neighbourhood of the scleral affection, but not extending more than 2 to 3 mm. into the cornea, except in very severe cases. It is an intense white opacity, situated in the true cornea, and is apt to remain as a permanent opacity, even when the scleritis undergoes cure. In such cases of sclero-keratitis, iritis is often present.

Treatment.—Warm fomentations, massage, and the treatment of whatever diathesis (rheumatism, syphilis) may be taken as giving rise to the condition.

Arcus Senilis.—This is a change which is developed in the cornea without previous inflammation. It presents the appearance of a greyish line a little inside the margin of the cornea and all round it, most marked above and below, and never advancing farther towards its centre. It is most common in elderly people, but is sometimes seen in youth, and even in childhood. No functional changes are caused by

[1] *A. von Graefe's Archiv*, vol. xix., p. 87.

it, nor does it interfere with the healing of a wound which may be made in that part of the cornea. Arcus senilis is caused by a hyaline degeneration of the corneal cells and fibrillæ, and is not a sclerosis, as is stated by some authors.

Pigmentation of the Cornea.—A rusty-brown discoloration of the cornea, due to hæmatin granules, has been occasionally observed, associated with hæmorrhage in the anterior chamber. A somewhat similar discoloration occurs in cases where particles of iron have been imbedded in the eye. Siderosis (*σίδηρος, iron*) is the name given to this latter condition.

CHAPTER VII.

DISEASES OF THE EYELIDS.

Erythema, erysipelas, phlegmonous inflammation, and abscess are all liable to attack the eyelids, but require no special observations in this work.

It should merely be stated, that erysipelas of the eyelids may extend to the connective tissue of the orbit, and ultimately give rise to atrophy of the optic nerve.

Eczema.—This is very often seen on the eyelids, most frequently in connection either with eczema of the face in general, or with phlyctenular ophthalmia, which latter is to be regarded as eczema of the conjunctiva and cornea. The lacrimation in phlyctenular ophthalmia increases the eczema, which then, by causing contraction of the skin of the lower lid, produces eversion of the inferior punctum lacrimale, and this, in its turn, causes increased lacrimation, and thus a vicious circle is set up.

Atropine infiltration of the eyelid, from long use of solution of atropine in some persons, is often accompanied by a moist form of eczema of the lids and face.

Treatment should consist in the daily removal of the scabs, in such a way as to cause no bleeding of the surface underneath ; and, for this purpose, a warm solution of bicarbonate of potash is useful. The place should afterwards be well dried, and painted with a strong solution of nitrate of silver (gr. xx ad ℥j), and a boracic acid ointment (gr. xxx ad ℥j), or the following, applied over this :—Ol. Cadin, m. xv ; Flor. Zinci, gr. xx ; Lanolin, ℥ij.—M. If the inferior lacrimal punctum be everted, the canaliculus should be slit up.

Herpes Zoster Ophthalmicus is an herpetic eruption, which affects the region supplied by the supraorbital division of the fifth nerve of one side, and sometimes its nasal branch, and, in rare instances, the infraorbital division of the same nerve. The occurrence of the eruption is preceded, for some days, by severe neuralgic pain and swelling, with redness of the part. The number of vesicles varies much, and may be but three or four, or so numerous as to become confluent. As soon as the eruption appears, the pain usually becomes much diminished, and, indeed, often disappears. Vesicles are liable to form on the cornea, and these may result in ulcers, which, on healing, leave opacities. The keratitis and ulcers of the cornea are nearly always accompanied by more or less anæsthesia of the affected portion, which may persist for a very long time.[1] Iritis has also been observed as a complication, and even cyclitis, resulting in loss of the eye. The vesicles on the skin soon become purulent, and gradually turn into scabs, which fall off, and leave deeply pitted scars, recognisable during the remainder of life. The affection never crosses the middle line of the forehead. Some neuralgia, with anæsthesia of the skin, may remain for a long time afterwards.

Inflammation of the Gasserian ganglion, with extension of the inflammatory process down the nerve, was found (O. Wyss) in the only case in which a *post-mortem* examination has been made during the acute stage of the disease.

The affection is most common in elderly people, but I have seen it, also, in young and healthy individuals.

The Treatment can only be expectant; or, at most, directed to relief of the patient's suffering, by means of hypodermic injections of morphia and other sedatives, and by emollients applied locally. Complications in the cornea and iris are to

[1] I have seen the anæsthesia to last for two years, although the ulceration had completely healed.—L. W.

be dealt with on the principles laid down in the chapters on the diseases of those organs.

Primary Syphilitic Sores occur on the eyelids, usually near the margin of the upper or lower lid, or at the inner or outer canthus. The first appearance is generally a "pimple," which ulcerates and becomes characteristically indurated about its base. The margin of the ulcer is clean-cut, and its floor somewhat excavated, and covered with a scanty greyish secretion. Occasionally there is no ulcer present, but the entire lid is swollen, greatly indurated, purple, and shiny; and then the diagnosis may be rendered difficult. The præauricular and submaxillary glands are almost always swollen; and this is a valuable, although not altogether positive, diagnostic sign, as it is seen also in tubercular diseases of the conjunctiva. The occurrence of the sore is followed by the usual constitutional symptoms of syphilis. Very rarely is there any permanent damage done to the eyelid.

The most common modes of infection are by a kiss from a syphilitic mouth, or by a dirty finger.

Treatment.—Locally, sublimed calomel by Kane's method, dusting with finely powdered iodide of mercury, or the black wash, may be used; while the usual general mercurial treatment is employed.

Secondary Syphilis gives rise to ulcers on the margins of the lids, to loss of the eyelashes (madarosis), and to the secondary skin affections which attend it in other parts of the body.

In **Tertiary Syphilis** ulcerating gummata of the lids sometimes are seen, accompanied by remains of previous iritis or keratitis.

Vaccine Vesicles on the eyelids are produced by accidental inoculation at the intermarginal part of the lid; or on the outer surface of the lid, if the skin be abraded by the finger-nail, or otherwise. Sometimes the vesicle develops into a

large ulcer with yellowish floor, and hard and elevated margin. There is much pain, much swelling of the eyelid, and chemosis.

Although distressing for a week or so while it lasts, the affection is not a dangerous one, further than that a cicatrix in the skin is left behind, and the eyelashes at the affected part are lost.

Treatment.—A warm chlorate of potash lotion (gr. v ad ℥j) is the best application.

Rodent Ulcer (Jacob's Ulcer).—This disease commences as a small pimple or wart on the skin near the inner canthus, or over the lacrimal bone, as a rule; but it may also originate in any other part of the face. The scab or covering of the wart is easily removed, and underneath is found a shallow ulcer with a well-defined indurated margin, the skin surrounding the diseased place being healthy, and continuing so to the end of the chapter. The progress of the disease is extremely slow, extending over a great number of years, and, in the early stages, the ulcer may even seem to heal for a time, but always breaks out again. In mild cases the ulceration may remain superficial; but, more usually, it strikes deep, in the course of time eating away every tissue, even the bones of the face, and the eyeball. The latter is often spared until after the orbital bones have gone.

The disease is an epithelial cancer of a non-malignant, or purely local, kind. There is no tendency to infiltration of the lymphatics. It is rarely seen in persons under forty years of age.

Treatment.—Extirpation of the diseased part affords the best chance of relief for the patient. Recurrence of the growth is the rule, but this should not deter from operative measures, nor even from the renewal of them, as they afford much comfort to the patient and prolong his life. Even in advanced stages operation is frequently called for. The application of chloride of zinc, or of the actual cautery, should

be employed, after the disease has been as thoroughly removed with the knife as is possible.

Bergeon's Treatment.—This consists in the internal administration of 5 grains of chlorate of potash three times a day, with the local application of a saturated solution of chlorate of potash to the ulcer, and by aid of it remarkably good cures can be effected.[1]

Marginal Blepharitis (*βλέφαρον, eyelid*), or **Ophthalmia Tarsi,**[2] is nothing else than eczema of the margin of the eyelid. It is found either as **Blepharitis Ulcerosa** (Eczema Pustulosa), or as **Blepharitis Squamosa** (Eczema Squamosa). In the former, small pustules form at the roots of the eyelashes, and these, having lost their covering, become ulcers, which scab over. The whole margin of the lid may then be covered with one large scab, in which the

[1] I have treated a considerable number of cases of rodent ulcer by scraping and subsequent application of chlorate of potash in powder, with uniformly satisfactory results, including two cases where enucleation had been advised. The process must be repeated daily, or at least every second or third day. It is certainly painful, but not unbearably so. Sometimes a green slough is produced, and when this is the case there is generally some surrounding inflammation, which should be allowed to subside a little before going on with the treatment. As the healing process does not begin until the diseased tissue has been removed, the progress may seem slow for the first week or fortnight, but no case resists the treatment if it be persevered with. While the chlorate of potash destroys the disease, it does not act injuriously on the delicate epithelium which begins to grow in from the margin as healing sets in, and it should therefore be continued until the whole surface has healed. Another fortunate peculiarity is that is has no effect on the normal conjunctiva, and may be used without fear, if the latter be involved in the disease. As to the permanency of the cure, I cannot make any positive statement, as none of my patients returned; but this fact renders it very probable that the disease did not recur, for had it done so, some of them would scarcely have failed to renew the treatment, as they were all greatly pleased at the result.—L. W.

[2] The term *tinea tarsi* is not employed in modern ophthalmology.

eyelashes are matted, and under which the lid will be found swollen, red, and moist, with many minute ulcers and pustules. Many eyelashes come away with the scab, and others are found loose and ready to fall out.

The disease is chronic, and is most commonly seen in strumous children. It is frequently accompanied by phlyctenular ophthalmia, or by simple conjunctivitis, which may have been its cause, or which promotes it, by keeping the margin of the lid constantly wet.

If neglected, ulcerous blepharitis is liable to produce trichiasis, by giving a false direction to the bulbs of the cilia.

Many ophthalmologists hold, that blepharitis is often caused by ametropia, especially by hypermetropia or hypermetropic astigmatism, in consequence of the incessant efforts of accommodation. I cannot go thus far; but perhaps, if blepharitis be once set up, such anomalies of refraction may help to keep it going.

The Treatment of Ulcerous Blepharitis consists in careful removal of the scabs, without causing any bleeding of the delicate surface underneath. Such bleeding indicates that the newly formed epithelium has been torn away, and it is important, therefore, to soften the scabs by soaking the eyelid with olive oil, or with a solution of bicarbonate of potash, before removing them. Any pustules found under the scab should be punctured, and all loose eyelashes taken away, and the ulcers touched with a fine point of solid mitigated lapis. The surface should then be well dried by pressure, not by rubbing, with a soft cloth, and the following ointment (Hebra) applied:—℞. Ol. Rusci (or, Ol. Juniperi) ʒss, Hydrarg. Ammon. Chlor. gr. iv, Cer. Galeni, Lanolin a.a. ʒij. This ointment is to be continued until healing is thoroughly established. In many mild cases a boracic acid ointment (gr. v ad ʒj of vaselin, or of lanolin) will be found efficacious instead of the above, and a white precipitate

ointment of from 1 to 2 per cent. acts well. A creolin ointment suits many cases, viz., Creolin, 1 to 5 min.; Aq., ʒij; Lanolin, ʒvj.

Or, again, after the scabs and loose eyelashes have been removed as above, the margins of the eyelids may be freely bathed with a wash of ten to twenty minims of creolin to eight ounces of water, as recommended by Dr. Glasgow Patteson for chronic eczema,[1] and after this the creolin ointment may be applied. I have found this method very successful. But in all cases, whatever the lotion or ointment ordered may be, the ulcers should be touched with mitigated lapis, as above recommended, and all loose eyelashes removed.

All complications with conjunctival affections, or lacrimal obstruction, must be dealt with, and the patient's general system carefully attended to. Any error in refraction should be suitably corrected.

Squamous Blepharitis comes on after the ulcerous form has passed away; or, it is found as a primary affection, especially in chlorotic women. The margin of the lid is somewhat swollen and red, and covered with loose epidermic scales. It is an extremely chronic affection.

The Treatment of Squamous Blepharitis is also an ointment of Hebra's:—

℞. Emplast. Diachylon Co.,[2] ʒij; Ol. Olivar, q. s.;

or, the Boracic Acid ointment may be used.

Chlorosis, if present, is to have suitable remedies.

Phtheiriasis (*φθείρ, a louse*) **Ciliorum.**—The pediculus pubis occurs on the eyelashes. It gives rise to excessive itching and burning sensations, and the consequent rubbing produces excoriations of the margin of the lid. The lice

[1] *Dub. Journ. Med. Sciences*, July 1891.

[2] Emplast. Diachylon Co. is made as follows:—Emplast. Litharg. B. P., 12 parts; Corn flour, 1½ part; Ammoniac, Galbanum, Turpentine, of each 1 part.

occupy chiefly the roots of the eyelashes, while the shafts of the cilia are covered with their brown egg-capsules, and this gives to the cilia the peculiar appearance of being covered with dark brown powder, which enables the diagnosis to be easily made. The fully developed parasites, as well as the eggs, may be more readily seen by aid of a strong convex glass.

Treatment.—With a cilium forceps the pediculi may be, to a great extent, if not completely, removed, as well as some of the eggs from the cilia. This proceeding repeated daily, along with the application of mercurial ointment, or of a weak red precipitate ointment, to the margin of the eyelids morning and evening, will soon effect a cure.

Hordeolum (*hordeum*, a grain of barley), or **Stye,** is a circumscribed purulent inflammation, situated at the follicle of an eyelash. It commences as a hard swelling, with more or less tumefaction and œdema of the general surface of the lid, and often with some chemosis, especially if it be situated at the outer canthus. In its early stages there is much pain associated with it. It gradually suppurates, and may then be punctured or allowed to open of itself.

Styes frequently come in rapid succession one after the other, and then, probably, a constitutional disturbance exists as the cause. In the earliest stage cold applications may be successful in putting back a stye, but, later on, warm stupes will hasten the suppuration and relieve the pain. Habitual constipation is a common source of hordeolum, and should be met by the occasional use of cascara sagrada, some aperient mineral water, or other mild laxative. Sulphide of calcium, $\frac{1}{10}$ gr. every hour, or $\frac{1}{2}$ gr. twice a day, for an adult, has been recommended (D. Webster) as a specific in these cases.

Chalazion (*χάλαζα, hail*), **Meibomian Cyst,** or **Tarsal Tumour,** is probably a granuloma in connection with a Meibomian gland, and not a mere retention cyst. Micro-

organisms[1] have been found by some observers in these tumours, but what relation exists between them and the tumours is a matter upon which opinions differ. It has its origin in a chronic inflammatory process in the connective tissue surrounding the gland, which usually passes off without having attracted the attention of the patient. These tumours vary in size, from that of a hemp-seed to that of a hazel-nut, causing a marked and very hard swelling in the lid. They occasionally open spontaneously on the conjunctival surface, giving exit to contents which are usually viscid or grumous, but sometimes purulent.

Treatment.—No application can bring about absorption of these tumours. The lid should be everted, the tumour opened by a single incision from the conjunctival surface, and its contents thoroughly evacuated by aid of a scoop or small sharp spoon. Difficulty is sometimes experienced in finding the point in the conjunctiva corresponding to the tumour, but it is usually indicated by a dusky or greyish discoloration. Immediately after the evacuation, bleeding into the sac often takes place, and causes the tumour to remain for a day, or more, as large as before; a fact of which the patient should be warned. The operation may occasionally require to be repeated two or three times. The interior of the sac should not be touched with nitrate of silver; and the incision and evacuation should never be made through the skin, because more or less disfigurement from the scar would result.

More than one chalazion is often present at a time, and some people become liable to them periodically during a number of years.

Milium (*milium*, a millet seed) presents the appearance of a perfectly white tumour, not much larger than the head of a pin, in the skin of the eyelid. It is a retention tumour

[1] Lagrange, *Tumeurs de l'œil*, etc., Paris, 1893; Fukala, *Centralbl. f. prakt. Augenh.*, Oct. 1893; Parisotti, *Annales d'Oculist*, June 1893, p. 417.

of a sebaceous gland, and can readily be removed by puncture and evacuation.

Molluscum, or **Molluscum Contagiosum.**—This is a white tumour in the skin of the eyelid, which may attain the size of a pea. At its summit is a depression, which leads to an opening into the tumour, through which the contents can be pressed out. It is probably a diseased condition of a sebaceous gland, and contains altered epithelial cells, and peculiar bodies termed molluscum corpuscles, which are of a fatty nature. Many such tumours may form in the lids at the same time.

It is held by some observers that this affection is contagious, although in what way is not clear, inasmuch as experimental rubbing of the contents of a molluscum into the skin has not given rise to the tumours.

Treatment.—Each separate tumour must be evacuated by simple pressure, or after it has been opened up with a knife or scissors.

Teleangiectic Tumours, or **Nævi**, of the eyelids, occur congenitally.

Treatment.—Small tumours of this kind may be destroyed by touching with nitrate of silver or hydrochloric acid, or by performing vaccination on them. Larger tumours may be ligatured, or treated with the galvano-cautery, and electrolysis is a very effectual method in many cases.

Xanthelasma (*ξανθός, yellow; ἔλασμα, a layer*) is the term applied to yellowish plaques raised slightly over the surface of the skin, with very defined margins. The patches are generally bilateral and symmetrical, and are most frequently situated in the neighbourhood of the inner canthus. The shape of these plaques is extremely irregular, and they may attain the size of a shilling or larger. The appearance is caused by hypertrophy of the sebaceous glands, with retention of their contents, and fatty degeneration of the subcutaneous connective tissue.

Treatment can only consist in removal by careful dissection, and this is hardly to be recommended, except in extreme cases.

Palpebral Chromidrosis (*χρῶμα, colour; ἵδρωσις, sweating*).—The phenomenon of an exudation of pigment upon the eyelids, of which about fifty cases have been recorded, has given rise to much discussion. The opinion held by many is that these cases are always the result, either of deception in hysterical individuals, or of accidental circumstances, such as the exposure of a patient with seborrhœa palpebrarum to an atmosphere loaded with coal-dust or pigmentary matter, in some manufacturing district. Of the fact that the appearance has occurred under both of these conditions, there can be no doubt. There would seem also to be evidence, that some genuine cases of colour-sweating on the eyelids have been observed; but they must be extremely rare. The discoloration is blue or black, and occurs in the form of fine powder upon the skin of one or both eyelids, of both eyes. It can be wiped off, and is said to begin to reappear after a short interval. The subjects of it have been chiefly young girls, but it has also been seen in women of advanced years, and even in middle-aged men.

The *Treatment* in a genuine case may consist in the application of a lotion of Liq. plumbi and glycerine; and, internally, iron, quinine, and arsenic, along with the regulation of the general system, particularly in respect of any uterine derangement.

Epithelioma, Sarcoma, Adenoma, and **Lupus,** are all seen in the eyelids, but require no special description here.

Clonic Cramp of the Orbicularis Muscle, or of a portion of it, is often seen, and is popularly known by the name of "life" in the eyelid. It is frequently due to overuse of the eyes for near work, especially by artificial light, or if there be defective amplitude of accommodation.

Treatment should consist in the regulation of the use of

the eyes for near work, and the correction by glasses of any defect in the accommodation.

Blepharospasm, or **Tonic Cramp of the Orbicularis Muscle,** is commonly the result of irritation of the ophthalmic division of the fifth nerve by reflex action, as in phlyctenular ophthalmia and some other corneal and conjunctival affections; or, from foreign bodies on the conjunctiva or cornea, etc.; or, it may continue for some time after the relief of any such irritation. It occurs, also, independently of such causes, and is then difficult to account for, unless as a hysterical symptom. Yet even in these obscure cases the spasm is probably often a reflex from the third nerve, and it will be found that pressure upon the supraorbital nerve at the supraorbital notch may arrest the spasm; or, if not there, then pressure on the infra-orbital, temporal, malar, or inferior alveolar branch may have the desired effect: or, at even still more remote regions, and in the course of other nerves, the "pressure point" may be discovered.

Treatment.—If the cause of the reflex cannot be ascertained, or have passed away, and the cramp be very distressing, stretching or resection of the branches of the fifth nerve, from which the reflex proceeds, may be tried. Morphium hypodermically has been of use in some cases, but it would be undesirable to continue this treatment for long.

Ptosis (*πτῶσις, a fall*), or **Blepharoptosis,** is an inability to raise the upper lid, which then hangs down over the eyeball. It is either congenital or acquired; and, in the latter case, is most usually the result of paralysis of the branch of the third nerve supplying the levator.

Persons affected with ptosis involuntarily endeavour to raise the eyelid by an over-action of the frontalis muscle. The drooping lid and elevated eyebrow give a peculiar and characteristic appearance.

The Causes of Paralytic Ptosis are similar to those of paralysis of other branches of the third pair, more especially

exposure to cold draughts of air while the body is heated, and syphilis or rheumatism affecting the branch to the levator palpebræ in its course. It may also be due to focal cerebral disease.[1] The branch to the levator may be paralysed alone, or in conjunction with other third nerve branches, and the loss of power may be partial or complete.

The Treatment of a recent case of ordinary paralytic ptosis depends upon its cause. If this be syphilis, then a course of mercurial inunctions or of iodide of potassium ; if rheumatism, then salicylate of soda or iodide of potassium ; with, in either case, protection of the eye and side of the head with a warm bandage. Cases in which these remedies have failed, and which have become chronic, often demand operative treatment. Attempts have been made, with success in some cases, to obviate the inconvenience of ptosis, by giving support to the lid by wire splints worn like an eyeglass, or attached to the upper edge of spectacle frames.

Ptosis due to a cerebral lesion rarely comes within the scope of treatment.[1]

Operative Treatment is indicated in cases of paralytic ptosis —where other measures have produced no result—in ptosis adiposa, and in congenital cases. A very common proceeding consists in the excision of a sufficiently large oval piece of integument, its long axis lying in the length of the lid, with the subcutaneous connective tissue and fat, and, in paralytic cases, a small portion of the orbicular muscle. The fold of integument to be abscised is seized by two pairs of forceps—one of them held by an assistant—at the inner and outer ends of the lid, and by this means the necessary size of the fold is estimated. The abscision is performed with a pair of scissors, the margin of the wound lying close to the points of the forceps. The subcutaneous tissue, etc., is then removed, and

[1] The value of ptosis as a localising symptom in cerebral disease will be treated of in chap. xviii.

the edges of the wound drawn together by a few points of suture.

Pagenstecher's Method is as follows:—Its object is to enable the patient to derive more benefit from the effort of his frontalis muscle, which he is constantly making with so little result, by transferring its action more directly to the eyelid. A needle carrying a thick ligature is entered under the skin of the forehead, about half an inch above the centre of the eyebrow, and passed subcutaneously as far as the margin of the eyelid at its middle point. The suture is closed, not very tightly at first, but each day somewhat more tightly, until it has cut its way through the skin. As the result of this, a cicatrix is formed in the course of the ligature, which gives the frontalis much more power over the eyelid. I have tried this method, but I have not been satisfied with it.

Birnbacher's Operation is an improvement on former attempts to connect the tarsus with the frontalis by cicatrices. An incision, with its convexity upwards, is made in the skin corresponding to the upper edge of the tarsus. Three sutures with a needle at each end are passed through the upper border of the tarsus, so as to form three loops, one central and two lateral; the two needles of the central loops are passed vertically upwards under the skin, and are brought out quite close to one another in the eyebrow. The lateral loops are treated in the same way, but are made to diverge on each side from the central one, instead of being parallel. The ends of the threads are tied over a small roll of lint, and tightened until the edges of the lids just touch when the patient closes the eye. They may be left in from twenty to twenty-five days.[1]

Panas' Method.[2]—The object of this operation is to bring

[1] *Centralblatt. f. prakt. Augenheilk.*, 1892, p. 129.

[2] *Archives d' Ophthalmologie*, Janvier-Février, 1886.

about a union between the lid and the frontalis muscle, by forming a flap in the former, which is fastened to the skin of the forehead, and to the surface of the muscle.

Before the operation commences, and while it is in progress, an assistant applies his hand firmly to the patient's forehead, in such a way as to prevent shifting of the skin of the eyelid over the underlying tissues, which would interfere with the exactitude of the proceeding.

A horn lid-spatula is inserted under the lid. Fig. 71 explains how the eyelid flap is formed. The horizontal incision along the top of the flap has a slight convexity upwards, is not quite an inch long, lies over the orbital margin, and goes through all the tissues down to the periosteum. Another incision, parallel to this one, rather more than an inch long, is made along the upper border of the eyebrow, and as deep as the periosteum. The flap of skin and muscle is now dissected from the tarsus down to its ciliary border, but the suspensory ligament of the lid must not be interfered with. The bridge of tissue between the two horizontal incisions is now to be undermined, without injury to the periosteum or suspensory ligament. The flap is then drawn up under the bridge by means of the sutures (*a a'*), and secured to the upper edge of the upper incision. Inasmuch

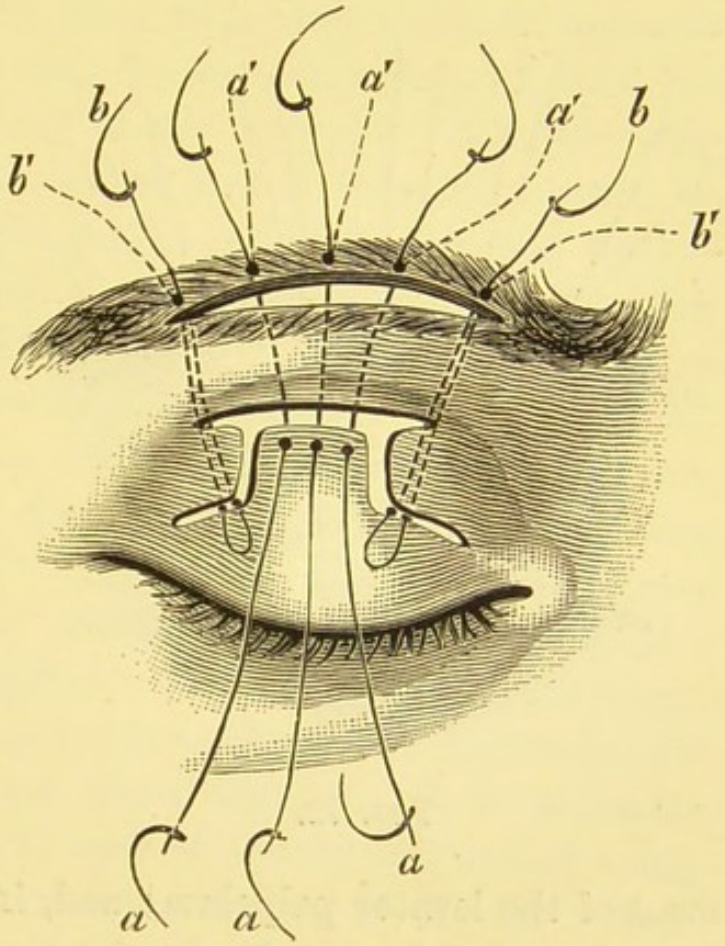


FIG. 71.

as the traction exercised by the flap when so fixed tends to produce ectropion of the lid, two lateral sutures (*b b'*) are applied deeply through the suspensory ligament and conjunctiva to the exclusion of the skin, and are attached, like the other sutures, to the upper lip of the upper incision, thus counteracting the tendency to ectropion. Fig. 72 shows the effect of the operation.

Fuchs has published[1] some cases of bilateral ptosis in elderly people, which were due, in his opinion, to primary atrophy of the levator palpebræ muscles. The eyelids were elongated and thinned, so that the eyeball showed plainly through them. The loss of power had in each case been very slowly increasing for many years.

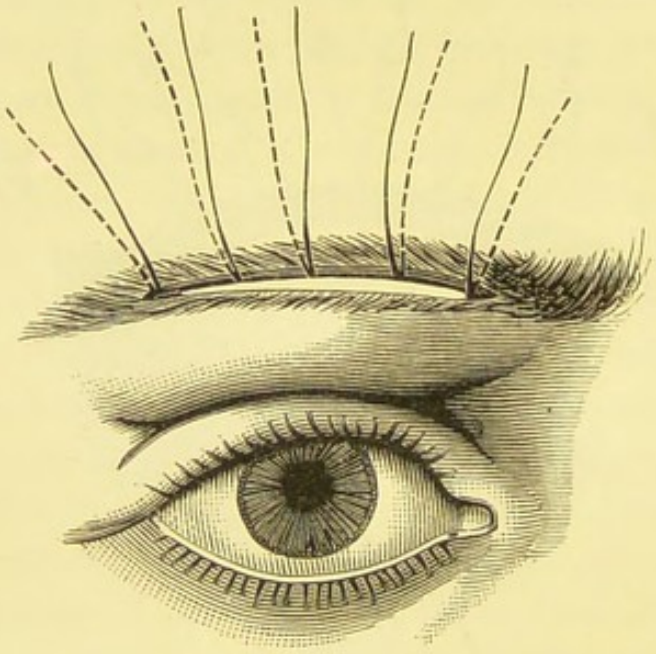


FIG. 72.

Congenital ptosis is generally present in both eyes. It is due, in some cases, to an imperfect development of the levator palpebræ; and, in others, to an abnormal insertion of this muscle, its tendon being attached to the tarsus too far back. Either Birnbacher's or Panas' operation may be employed here. Eversbusch has proposed[2] the following proceeding more particularly for congenital ptosis:—

Eversbusch's Operation for Congenital Ptosis.—The object of the operation is to increase the power of the levator by

[1] *Von Graefe's Archiv*, xxxvi. 1, p. 234.

[2] *Monatsbl. f. Augenhk.*, 1883, p. 100.

advancing its insertion, or rather by doubling it down over the tarsus to which it forms fresh adhesions Snellen's lid-clamp is applied, so that the plate is passed well up into the fornix; and, before the ring is screwed down, the skin of the lid is drawn down, so that its prolongation just under the eyebrow may be forced into the instrument. The skin and the underlying orbicularis are now divided in the entire width of the lid, parallel to its free margin, and at a distance halfway between this margin and the eyebrow. The skin and the subjacent muscle are then separated up, both upwards and downwards, for 4 mm. in each direction, so that the insertion of the levator may be well exposed. A suture with a small curved needle at either end is then introduced, by means of one of these needles, horizontally into the tendon at its insertion, and near the centre of the latter, in such a way that about $2\frac{1}{2}$ mm. of the tendon may be included in the suture. Each needle is now passed vertically downwards between the tarsus and orbicularis, and brought out at the free margin of the lid at a distance from each other of about $2\frac{1}{2}$ mm. Two more such double sutures, one in the temporal, the other in the nasal, third of the tendon are similarly applied. The margins of the horizontal skin and muscle wound are now drawn together, and then the three sutures are closed tightly. It is desirable to slip glass beads over the ends of the sutures before tying them, to prevent cutting into the margin of the lid. Both eyes are bandaged, and the sutures left in for a week or more. Figs. 73 and 74 serve to render the foregoing explanation more lucid. Congenital ptosis is sometimes associated with epicanthus. (See end of this chapter.)

A remarkable condition is *Congenital Ptosis with Associated Movements of the Affected Eyelid during the action of certain muscles.* There are only about thirty cases of this on record. It is most commonly the left lid which is affected, and the paralysis may be congenital or acquired. Three con-

ditions have been observed, viz., elevation of the drooping lid when the eye is adducted, when the eye is abducted, or when the mouth is open. A synchronous contraction of the pupil has been noticed in some cases, while in some the elevation of the lid occurs also with a lateral motion of the jaw, and with deglutition. Gower's explanation is that in these cases the levator is not wholly supplied by the third nerve, but partly also by nerve fibres which take their

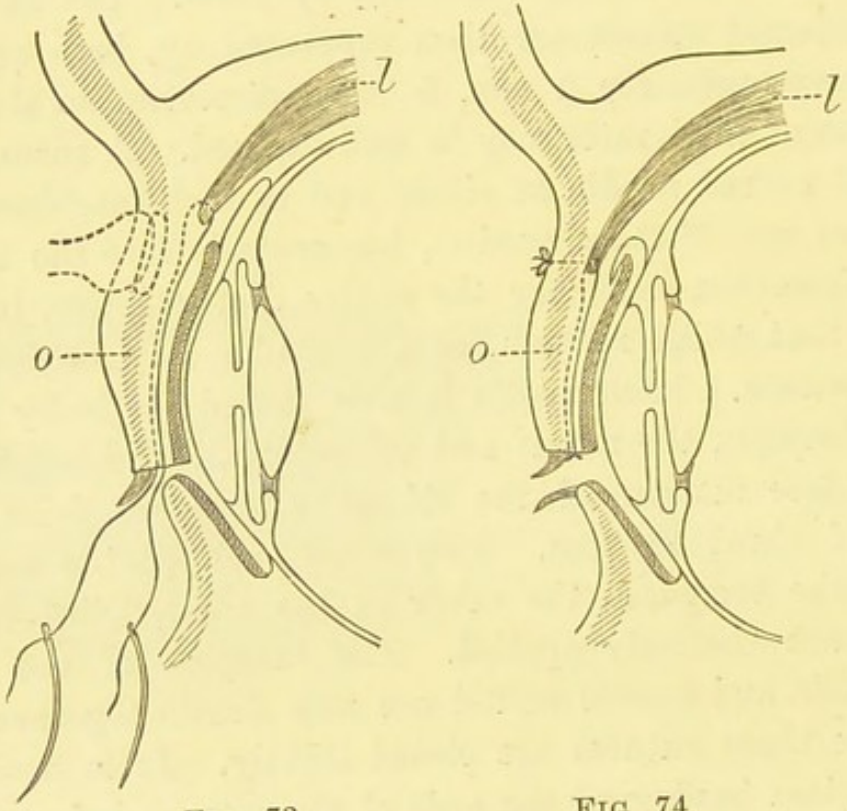


FIG. 73. FIG. 74.

l, levator palpebræ; *o*, orbicularis.

origin, in the third variety, in the nucleus of the fifth pair, and which also supply the external pterygoid and digastric muscles. But this theory does not hold good in all cases, for Bull[1] describes a case in which the lid was also raised when the head was bent back, thus stretching the digastric. He regards these as associated or reflex movements. In some instances the lid can be raised voluntarily on closing

[1] *Archiv. of Ophthalm.*, xxi., p. 354.

the other eye. Needless to say, no remedy can be applied for relief of this condition.

The term "ptosis" is also given, although not very correctly, to cases in which increased weight of the lid causes it to droop, as in conjunctival affections, or where a tumour has formed in the eyelid, or where there is a hyper-development of the subcutaneous fat.

Lagophthalmos (λαγώς, *a hare*, as it was supposed that this animal sleeps with its eyes open ; ὀφθαλμός), or inability to close the eyelids, is most commonly due to paralysis of the portio dura, and is then associated with the other symptoms of the latter affection. On an effort to close the lids being made, the eyeball is rotated upwards under the upper lid, owing to the associated action of the superior rectus; and in sleep this upward rotation also occurs—a fact which explains, to a great extent, the immunity of the cornea from ulceration in many of these cases. Lagophthalmos may also be due to orbital tumours pushing the eyeball forwards, to exophthalmic goitre, to staphyloma, or to intraocular growths distending the walls of the eyeball; in all of which conditions the eyelids are often mechanically prevented from closing over the eyeball, or can be closed only by a strong effort of the will. The danger to the eye depends upon the tendency to ulceration of the cornea from its dryness, caused by exposure to the air, and from foreign substances not being removed from it by nictitation.

When lagophthalmos occurs as a symptom in focal cerebral disease, it is useful in localising the disease, by assisting in differentiating a lesion in the internal capsule, or in the facial motor centre of the cortex, from one implicating the portio dura in the pons, as it is absent, or very slight, in the former cases, but very often markedly present in the latter. With a lesion in the lower part of the pons we are apt to have lagophthalmos with crossed hemiplegia ; but, if the lesion be in the upper part of the pons—the fibres from the opposite

side having here joined the motor tract—the hemiplegia and lagophthalmos will be homonymous.

Treatment.—In cases of non-paralytic lagophthalmos, protection of the cornea, by keeping the eyelids closed with a bandage, or a few epidermic sutures in the margins of the eyelids, should be our first care. Tarsoraphy may be employed in those cases where circumstances indicate that it would be useful, *e.g.* in some cases of exophthalmic goitre, or of staphylomatous eyeball.

In paralytic cases, the primary cause of the paralysis (syphilis, rheumatism, etc.) must be treated, so long as there is a prospect of restoring power to the muscle. Locally, galvanism and hypodermic injections of strychnia may be employed. During cure, the cornea should be protected as above. In incurable cases, the opening of the eyelids must be reduced considerably in size by an extensive tarsoraphy.

The Operation of Tarsoraphy consists in uniting the margins of the upper and lower lids in the neighbourhood of the external commissure, so as to reduce the size of the opening of the eyelids. The commissure should be caught between the finger and thumb, and the edges of the lids approximated, so as to enable the operator to form an estimate of the required extent of the operation. A horn spatula is then passed behind the commissure, and the necessary length of the margin of each lid, including the bulbs of the cilia, abscised with a sharp knife. The raw margins are then brought together with sutures.

Symblepharon (*σὺν, together*; *βλέφαρον, the eyelid*) is an adherence, partial or complete, of the eyelid to the eyeball. It is usually the result of burns of the conjunctiva by fire, acids, or lime. The shortening of the conjunctival sac, which is seen as the result of pemphigus or of granular ophthalmia, and which I have above described under the heading of Xerophthalmos, is sometimes, but I think wrongly, called

symblepharon. If the symblepharon interfere seriously with the motions of the eyeball, or, if it cause defect of vision by obscuring the cornea, it becomes desirable to relieve it by operation. Should it consist of a simple band stretching from lid to eyeball, it may be severed by ligature, and, if the band be broad, two ligatures may be employed, one for either half. A symblepharon which occupies a considerable surface cannot be got rid of in this way, and, for such cases, a transplantation procedure like that of Teale [1] or of Knapp [2] may be employed, the great difficulty in dealing with these cases being the tendency there is to reunion of the surfaces, unless one or both of them be carpeted with epithelium.

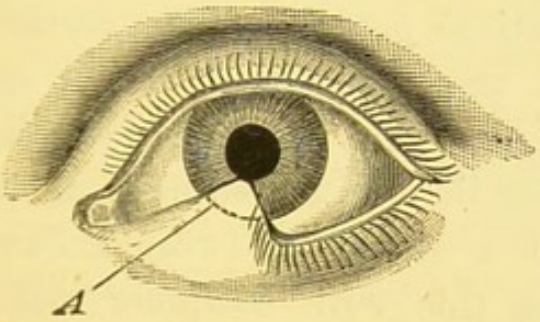


FIG. 75.

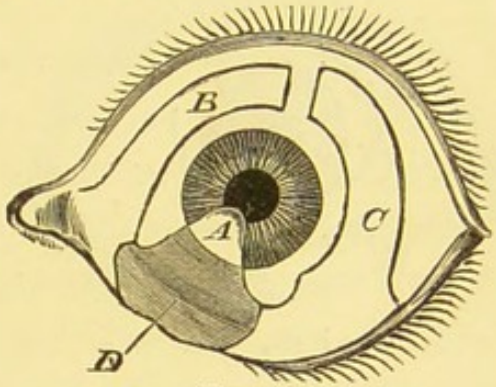


FIG. 76.[3]

In *Teale's Operation*, if we suppose the case to be similar to that represented in Fig. 75, an incision is carried along the line of the margin of the cornea at *A*, through the whole thickness of the symblepharon, and the lid is dissected off from the eyeball as far as the fornix. Two conjunctival flaps are now formed, as at *B* and *C* in Fig. 76, and one of them (*B*) is turned to form a covering for the wounded surface of the inside of the eyelid, while the other (*C*) is used to cover the bulbar surface (Fig. 77), the flaps being held in their places by fine sutures. That part of the symblepharon

[1] *Ophthal. Hosp. Rep.*, vol. iii.

[2] *Archiv f. Ophthal.*, xiv., pt. 1, p. 270.

[3] Mr. Teale now makes his flaps, as in Fig. 76, wider than he originally did. I have to thank him for altering this drawing with his own hand for this work.

which is left adherent to the cornea soon atrophies and disappears. No great tension of the flaps should exist as they lie in their new positions.

Teale, again, has suggested the formation of a bridge-like conjunctival flap above the cornea, and the removing of it across the latter to cover the loss of substance situated below. After the sutures to keep the flap in its place have been introduced, the latter is separated at its bases.

A simple plan, which would be applicable to such a case as that depicted in Fig. 75, where the adhesion is not very extensive, and perhaps even to some more extensive ones, consists in dissecting the conjunctival process off the cornea, and then turning it down on the raw inner surface of the under lid, and fastening it there with a suture or two. I have done this with complete satisfaction.

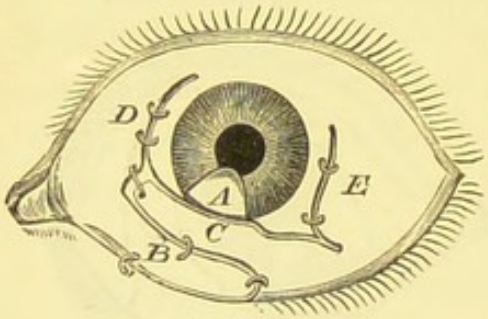


FIG. 77.

The transplantation of a portion of rabbit's conjunctiva, as suggested by Wolfe, or of a portion of mucous membrane from the lips, or from the vagina, as employed by Stellwag, is undoubtedly the best method for many cases of extensive symblepharon. The chief precautions necessary for success in this proceeding are :—That the flap to be transplanted be not applied in its new position, until all bleeding at the latter place has ceased. That the flap be nothing more than mucous membrane, all sub-mucous tissue being carefully removed. That it be sufficiently large to cover the defect without any stretching ; and it should be remembered, that the flap shrinks to two-thirds of its size after being detached from its own bed. That the flap be kept moist and warm during the period, as short as possible, which may elapse between its detachment and its adjustment. And, finally, that it be kept firmly in its new position by a sufficient number of points of interrupted suture.

Harlan's Operation.[1]—This is specially applicable to extensive symblepharon of the lower lid, and differs from the foregoing operations in that it provides a covering of skin, and not of mucous membrane, for the raw surface of the under-lid. Operations on the same principle have been proposed by Snellen, and by Kuhnt. An incision A B (Fig. 78) through the whole thickness of the eyelid, and corresponding in length to the latter, is made along the lower margin of the orbit. Below this a skin flap C D is then formed. The flap is dissected up, and the incisions are carried a little more deeply as A B is approached, to enable the flap to turn the more readily. The flap is then turned up as on a hinge, slipped through the button-hole, and sutured securely to the inner surface of the under-lid. After a time the skin surface turned towards the eyeball becomes considerably modified, so as to be somewhat like mucous membrane. The bare space left by the removal of the strip of skin is covered without strain, by making a small horizontal incision, D E, at it outer extremity, and forming a sliding flap.

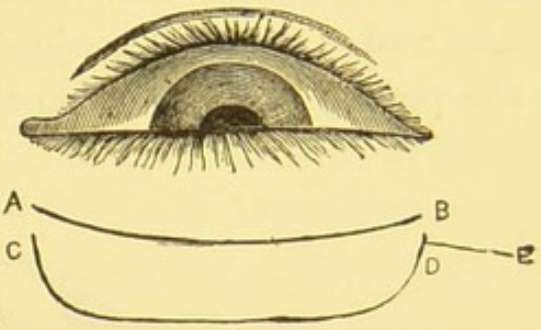


FIG. 78.

Blepharophimosis (*βλέφαρον, eyelid; φίμωσις, narrowing*) is a contraction of the outer commissure of the lids, with consequent diminution in size of the opening between the latter; and is commonly due to shortening of the skin, from long-continued irritation of it, caused by the discharge in a case of conjunctivitis.

It is remedied by a *Canthoplastic Operation.* The outer commissure is divided in its entire thickness, in a line which is a prolongation of the line of junction of the lids when closed, by a single stroke of a strong straight scissors, one blade of which has been passed behind the commissure. The integumental incision should be made a little longer than that in the con-

[1] *Ophth. Rev.*, vol. ix., p. 351.

junctiva. An assistant then draws the upper lid up and the lower lid down, so as to make the wound gape. The conjunctival margin and the dermic margin are now united in the centre by a point of suture (C, Fig. 79), while two more sutures (A and B) are applied, one above and the other below the first. This operation is also employed in cases of granular ophthalmia, and of purulent conjunctivitis, when it is desired to relieve the pressure of the lid on the globe.

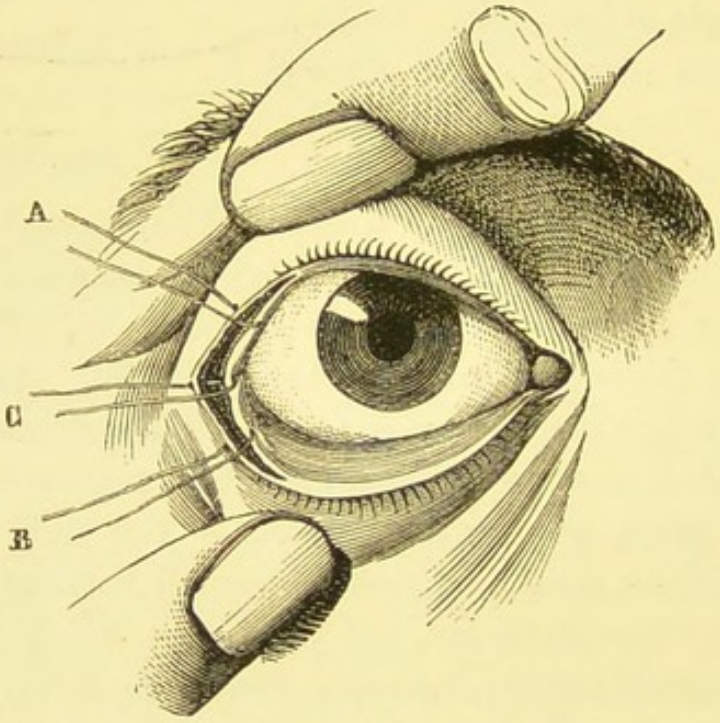


FIG. 79 (*de Wecker*).

Distichiasis (δὶς, *twice* ; στιχος, *a row*), and **Trichiasis** (τρίχος, *a hair*).—The first of these terms indicates the growth of a row of eyelashes along the intermarginal portion of the lid, in addition to the normal row ; while trichiasis indicates a false direction given to the true cilia. Both conditions are often found coexisting, and they are also often present along with entropium. They may both be produced by chronic blepharitis, or by chronic granular ophthalmia. It has been commonly held that cicatricial contraction, giving a false direction to the hair follicles, is the immediate cause

of these conditions; but Raehlmann has recently [1] shown that the false cilia are developed as buds or offshoots from the follicles of the cilia, and primarily from the cuticle of the free margin of the lid. The latter mode of development is a novel discovery by Raehlmann, which he seems to have definitely proved by his pathological investigations. His view is, that hyperæmia of the margins of the lids, and inflammation of a proliferating type, is what gives rise to this primary development of hairs. The symptoms to which they give rise, and the dangers to the eye attendant on them, are due to the rubbing of the irregular eyelashes on the cornea, which produces pain, blepharospasm, and opacity of the cornea, or even ulceration of it.

Operations for Distichiasis and Trichiasis :—

Epilation.—The false cilia may be pulled out with a forceps; but this cannot be regarded as a cure, for the hairs grow again.

Electrolysis has been proposed by Dr. Charles Mitchell, of Missouri,[2] and by Dr. A. Benson, of Dublin.[3] A needle is attached to the negative pole, and its point passed into the bulb of the eyelash to be removed, the positive pole being placed on the temple. On closure of the circle, if the battery be working properly, bubbles of gas should rise up round the needle, and a slough forms at the root of the hair, which becomes loose, and is removed. It does not grow again, for the bulb is destroyed. Each hair must be separately operated on. The proceeding is very valuable where only a few cilia are to be dealt with.

Illaqueatio.—Snellen has revived this ancient operation, for cases where only a few isolated hairs are out of order. Both ends of a bit of very fine silk thread are passed through the

[1] *Von Graefe's Archiv*, xxxvii. 2, p. 66.

[2] *Trichiasis and Distichiasis, their Nature and Pathology, with a Radical Method of Treatment;* and *Klin. Monatsbl.*, April 1882.

[3] *Brit. Med. Journal*, December 16th, 1882.

eye of a fine needle, so as to form a loop. The needle is now entered as close to the point of exit of the hair as possible, and the counter-puncture is made in the position which the hair should normally occupy in the row of its fellows. The needle is drawn completely through, as also the ends of the thread, but the loop not as yet. Into the loop the eyelash is now inserted by aid of a fine forceps, and by traction on the ends of the thread, loop and eyelash are drawn through the tunnel. Unfortunately, the eyelashes frequently regain their abnormal position by reason of their own elasticity.

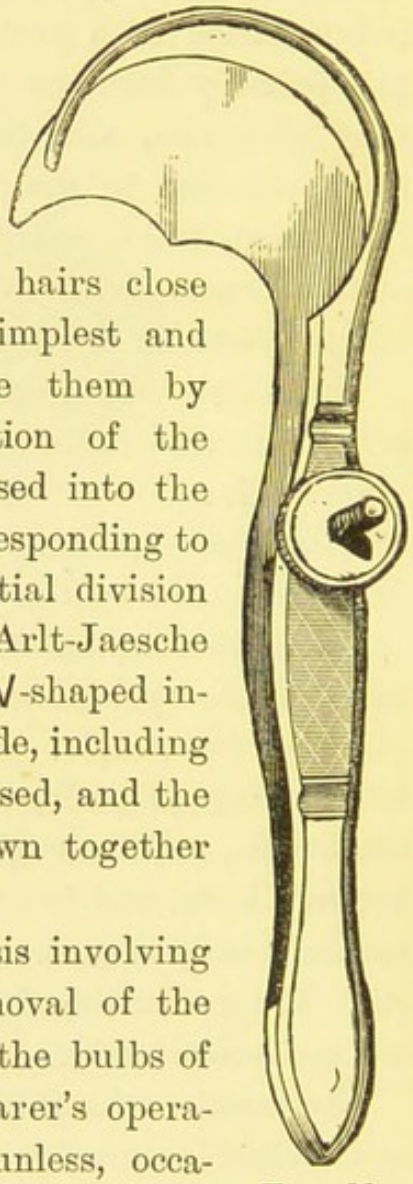


FIG. 80.

Excision.—When some half-dozen hairs close together are growing wrong, the simplest and best plan is to completely remove them by excision of the corresponding portion of the ciliary margin. A fine knife is passed into the intermarginal region, at the place corresponding to the hairs to be dealt with, and a partial division of the lid into two layers, as in the Arlt-Jaesche operation (*vide infra*), is effected. A V-shaped incision in the skin of the lid is then made, including the erring hairs, the whole flap is excised, and the margin of the loss of substance drawn together with sutures.

In cases of distichiasis or trichiasis involving the whole length of the eyelid, removal of the marginal portion of skin containing the bulbs of all the eyelashes, true and false (Flarer's operation), is not to be recommended—unless, occasionally, in the underlid—because it unnecessarily deprives the eye of an ornament, and of a protection against glare of sun and foreign bodies.

Transplantation, or Shifting, of the marginal portion of the integument containing the hair bulbs, true and false, is

a preferable proceeding in these complete cases. One of the oldest, and most valuable, operations of this kind is that of Jaesche, modified by Arlt. It is performed as follows:—Knapp's, or Snellen's, clamp (Fig. 80) having been applied to prevent bleeding, the lid in its whole length is divided in the intermarginal part into two layers (Fig. 81), the anterior containing the orbicular muscle and integument with all the hair bulbs, the posterior containing the tarsus and conjunctiva. The incision in the intermarginal portion is about 5 mm. deep. A second incision is now made through the integument of the lid; parallel to its margin, and from 5 to 7 mm. removed from it. This incision also extends the whole length of the lid. A third incision is carried in a curve from one end to the other of the second incision. The height of the curve is proportional to the effect required, varying from 4 mm. to 7 mm. The piece of integument included between the second and third incisions is dissected off with forceps and scissors, without any of the underlying muscle being touched, and the margins of the loss of substance are brought together by sutures. By this procedure the lower portion of integument containing the hairs and their bulbs is drawn up, and away from contact with the cornea.

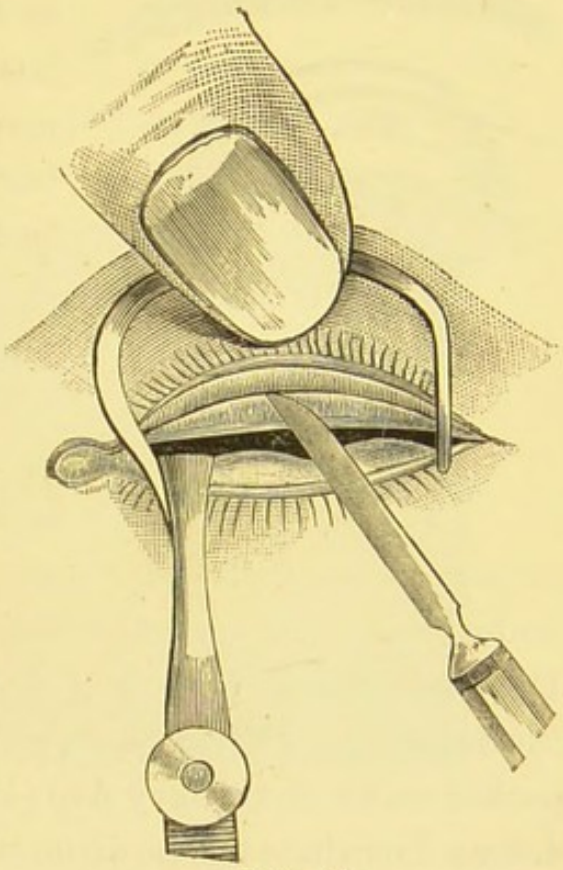


FIG. 81.

Spencer Watson,[1] Nicati,[2] Schoeler,[3] Burchard,[4] Dianoux,[5]

[1] *Ophthal. Hosp. Rep.*, vol. vii., 1873, p. 440. [2] *Marseille Médicale*, 1879.
[3] *Klinischer Bericht*, 1880. [4] *Charité Annalen*, p. 633.
[5] *Annales d'Oculistique*, 1882, p. 132.

and Gayet[1] have all proposed double transplantation operations.

Dianoux's Operation is as follows:—Snellen's (or de Wecker's) clamp is applied (omitted in figures for simplicity), and an incision (Fig. 82) is made parallel to the free margin of the lid, about 4 mm. from it, extending the whole length of the lid, and penetrating to the tarsus, but not through the latter. The ciliary portion of the lid, marked off by this means, is now detached from the tarsus by an incision in the intermarginal portion of the lid, as in the Arlt-Jaesche operation. An incision through the skin alone is then made about 3 mm. above the first incision and parallel to it, but extending some 2 mm. beyond it at either extremity. The skin flap is separated off from the underlying muscle, except at either end, where it is left attached. The underlying portion of the muscle is then separated from the tarsus, and allowed to retract upwards. A forceps being passed under the ciliary flap (Fig. 82), the skin flap is seized and drawn down into the position of the former (Fig. 83), where it is made fast by three sutures to the margin of the tarsus. The ciliary flap is moved up, and carefully stretched upon the tarsus bared of the orbicularis, the latter being drawn back with a strabismus hook, and the flap is secured in its place by sutures to the tarsus. An antiseptic dressing is applied, and the sutures may be removed on the third day. Although the wounded surface of the ciliary flap does not become vitally

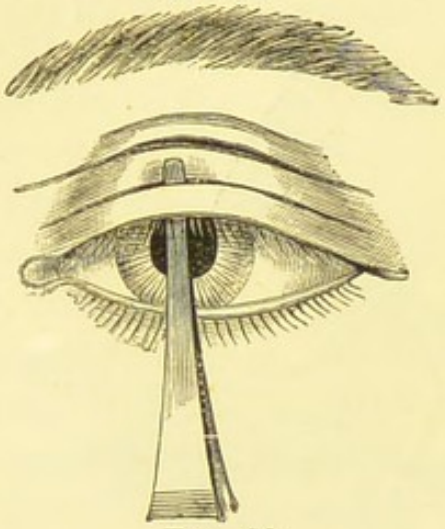


FIG. 82.

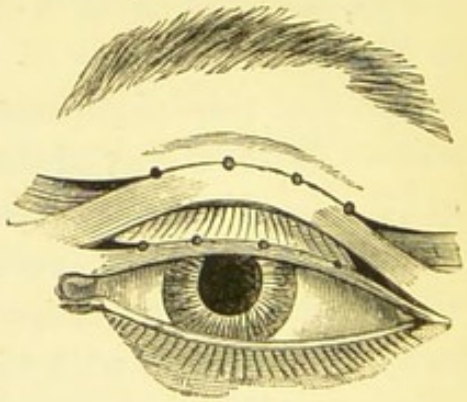


FIG. 83.

[1] *Ann. d'Ocul.*, 1882, p. 27.

united with the epidermic surface of the skin flap, yet no practical ill result follows.

A real objection lies in the circumstance that, occasionally, the cutaneous hairs on the transplanted flap irritate the cornea, and these hairs, being much finer than cilia, are more difficult to deal with.

Vossius' Operation.[1]—If, for example (Fig. 84), the whole extent of the right upper lid be affected with trichiasis, a horn lid spatula (the clamp will not answer) is passed under the lid, and held by an assistant. An intermarginal incision is made, as in the Arlt-Jaesche operation, about 3 mm. to 4 mm.

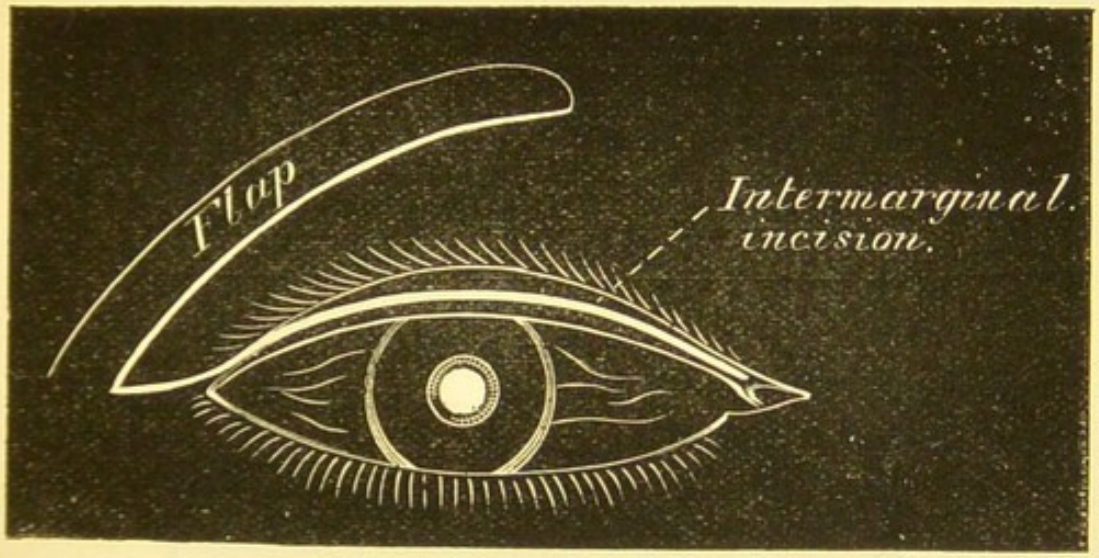


FIG. 84.

deep. This incision is then prolonged through the skin merely, over the external commissure for 5 mm. to 6 mm. It is then turned upwards at an angle with the free margin of the lid about 35°, and a flap about 5 mm. wide is marked out with the knife in the usual crease, or fold, of the upper lid. A narrow, sharp, and pointed scalpel is then thrust under the flap at its base, and carried towards its inner end, so as to separate it off without the aid of forceps, scissors, or any other instrument. The margins of the wound thus made are brought together with four or five sutures, and the flap turned down, and secured in the gaping inter-

[1] *Bericht d. Ophthal. Gesselsch.* (Heidelberg, 1887), p. 42.

marginal incision, by means of four or five sutures between each of its edges and the corresponding palpebral margin. One suture fastens the free end of the flap in the median corner of the wound. The position of the cicatrix, just in the fold of the upper eyelid, prevents its causing any disfigurement. Were the case one of partial trichiasis, the intermarginal incision should extend a little beyond the point where the abnormal condition ceases. If it be the inner half only of the margin of the lid which is affected, the intermarginal incision is prolonged towards the nose, and the flap so formed

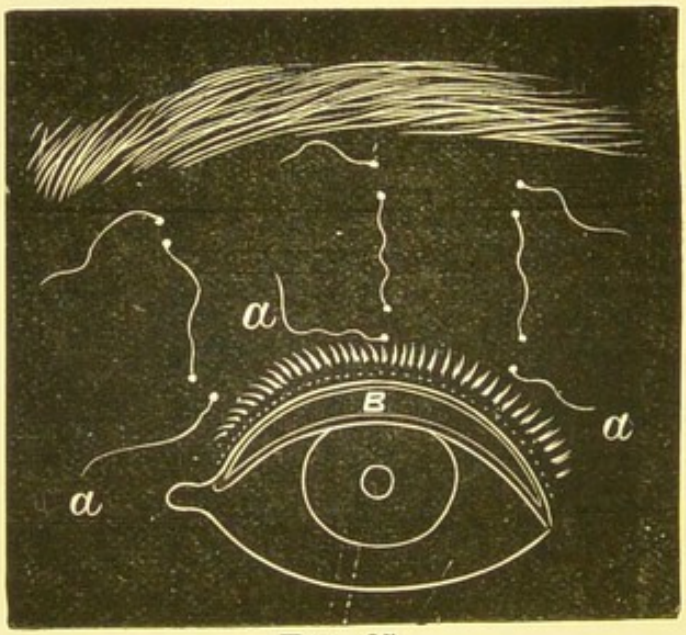


FIG. 85

that its base lies over the inner canthus. The flap heals in readily, and, although it shrinks somewhat, secures a wide intermarginal portion. The same drawback in connection with the cutaneous hairs on the transplanted flap holds good here, as in Dianoux's operation.

Van Millingen's Operation[1] consists in splitting the eyelid, as in the Arlt-Jaesche operation, from end to end, sufficiently to produce a gap (*B*, Fig. 85) 3 mm. in width at the central part of the lid, and gradually becoming narrower towards the canthi. The gap is kept open by sutures passed through

[1] *Ophthalmic Review*, 1887, p. 309.

folds of skin on the upper lid (*a*, *a*, *a*), by means of which also the lid is prevented from closing for twenty-four hours at the least. As soon as the bleeding has ceased, a strip of mucous membrane of the same length as that of the lid, and 2 to 2½ mm. in breadth, is cut out with two or three snips of a curved scissors, from the inner surface of the patient's under lip, and is placed at once into the gap in the intermarginal space. It should then be pressed into position with a pledget of cotton wool steeped in sublimate solution (1 in 5,000). Sutures are superfluous, according to Van Millingen, but I like them, and do not find that they do harm. The eyelid is then covered over with a piece of lint, on which is spread a thick layer of iodoform vaseline, and on this is placed a wad of cotton wool. Both eyes should be bandaged. The sublimate lotion is used for disinfecting the eye and lip during, before, and after the operation. The bandage should be renewed once in twenty-four hours, and the sutures in the upper lid ought not to be removed before the second day.

Van Millingen does not think it advisable to transplant small strips of mucous membrane if the trichiasis be partial. He regards this condition as only the commencement of complete trichiasis, and therefore recommends, even in these cases, the filling up of the entire length of the intermarginal space with a flap of mucous membrane. In cases of shortening of the conjunctival surface, in which it has been reduced to ½ cm., a strip of mucous membrane measuring 4 mm. in width at the centre may be transplanted.

The strip to be transplanted is generally taken from the angle of the lip, and from the line of demarcation between the dry and moist surfaces of the lip. A couple of fine sutures, which serve to unite the margins of the wound in the lip, arrest the bleeding at once, and accelerate union of the part, which is generally completed in twenty-four hours.

The transplanted tissue in this instance being free from

hairs, the method is not open to the objection referred to in Dianoux's and in Vossius's operation, while it is equally effectual in permanently providing a good intermarginal space, and in thus relieving the condition.

Entropium (*ἐν, in; τρέπω, to turn*),or **Inversion of the Eyelid,** is due to some organic change in the conjunctiva or tarsus, or to spasm of the palpebral portion of the orbicular muscle.

A large proportion of the former class of cases is the result of chronic granular ophthalmia, and is most common in the upper lid.

Spastic entropium usually occurs in the under lid. It is frequent in old people (senile entropium) from relaxation of the skin of the eyelid, and is also produced by the wearing of a bandage after operations, etc., and by œdema of the conjunctiva in inflammation of that membrane.

Treatment.—Organic entropium, in which the tarsus is not distorted, can often be corrected by one of the methods described for trichiasis and distichiasis. But many of these cases are accompanied by, or rather are due to, abnormal curvature with hypertrophy of the tarsus.

In all such cases the operation must include an attack on the tarsus itself, or the result will be abortive. Indeed, I have little doubt that much of the disappointment experienced in the treatment of entropium has been due to imperfect appreciation of this fact.

Streatfield's Operation is as follows:—The clamp having been applied, an incision is made through the integument of the eyelid parallel to its margin, 2 mm. distant from the latter, and extending its whole length. The muscle is dissected up so as to lay bare the tarsus, and then a wedge-shaped piece, 2 mm. wide and the length of the lid, its edge pointing towards the inner surface of the lid, is excised from the tarsus. A corresponding portion of muscle and skin is also removed, and the wound left to heal by granulation.

The shrinking of the resulting cicatrix causes the marginal portion of the tarsus to return to its correct position.

Snellen's Operation.—Snellen's clamp (very similar to Knapp's, which can equally well be used) is applied. About 3 mm. from the margin of the lid, and parallel to it, an incision is made through the skin alone, extending the whole length of the lid. The orbicular muscle is exposed by dissection of the skin upwards, in order to promote retraction of the latter, and, along the edge of the lower margin of the wound, a strip of about 2 mm. broad of the orbicular muscle

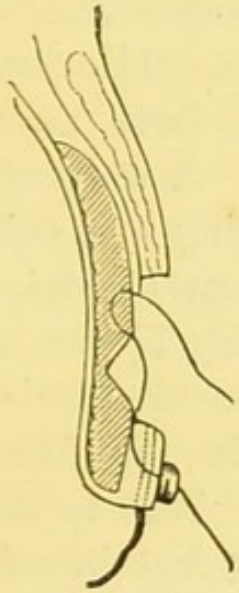


FIG. 86.

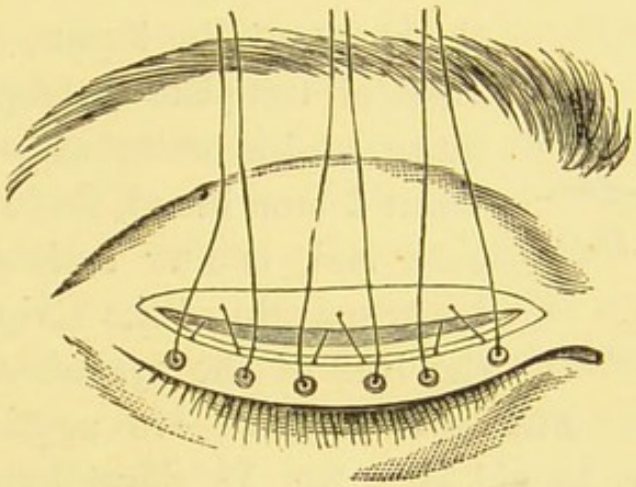


FIG. 87.

is removed, and the tarsus to the same extent exposed to view. A wedge-shaped piece, corresponding to the exposed part of the tarsus, is now excised from it with a very sharp scalpel or Beer's cataract knife, the edge of the wedge pointing towards the conjunctiva, which latter, however, is left intact. The hypertrophy of the tarsus, which is always present, facilitates this procedure. A silk suture carrying a needle on each end having been prepared, one needle is passed from within outwards through the band of muscle and integument left at the margin of the lid. The second needle is also passed from within outwards through the upper lip of the tarsal loss of substance, and then from

within outwards through this same marginal band, at a distance of about 4 mm. from the point of exit of the first needle. The ends of the suture are now tied together, a small bead having first been strung on each to prevent it from cutting through the skin. Three such sutures are employed. The accompanying woodcuts (Figs. 86 and 87) make the foregoing description more intelligible.

Green's Operation.[1]—An incision is made on the inner surface of the lid, in a line parallel to, and about 2 mm. distant from, the row of openings of the Meibomian ducts. It is carried through the conjunctiva and whole thickness of the tarsus, and should extend, in cases of complete entropium, from near the inner to the outer canthus. A strip of skin about 2 mm. broad, and tapering to a point at each end, is now excised from the lid,—the lower margin of the strip being $1\frac{1}{2}$ mm. above the line of the eyelashes. The muscle is left intact. Fine silk sutures are applied in the following manner, by aid of a No. 12 glover's needle bent to an arc of about a third of a circle. The needle is first introduced a little to the conjunctival side of the row of eyelashes, and is brought out just within the wound made by the excision of the strip of skin (Fig. 88, *A*); it is then drawn through, inserted again in the wound near its upper margin, and passed deeply backwards and upwards, so as to graze the front of the tarsus, and emerge through the skin a centimetre or more above its point of entrance (Fig. 88, *B*). On tying the two ends of the thread together the skin-wound is closed, and the loosened lid-margin is at the same time everted, and brought into a correct position. Three sutures generally suffice for the accurate adjustment of the lid-margin. In the spaces between and beyond the sutures it is often

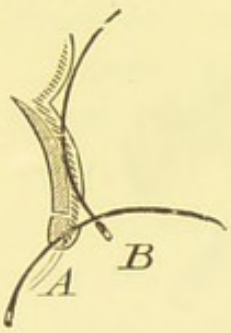


FIG. 88.

[1] *Trans. American Ophthal. Soc.*, vol. iii., p. 167.

practicable, and advantageous, to turn the eyelashes upwards against the front of the eyelid, and fix them there by means of collodion. The stitches should be removed, at latest, on the day after the operation; the line of suture being then strengthened by collodion, or, in case the cilia are very short, a few short fibres of cotton are used with the collodion.

Berlin's Operation.—Knapp's clamp is applied. The first incision lies 3 mm. above the margin of the lid, extends its whole length, and divides it in its entire thickness, including the conjunctiva. The skin and muscle at the upper edge of the wound are pushed or dissected up, so as to expose the tarsus. The upper edge of the tarsal incision is now seized at its centre with a finely toothed forceps, and an oval piece with the adherent conjunctiva, about 2 to 3 mm. wide in its widest part, and in length corresponding with that of the eyelid, is excised from it with a fine scalpel. The wound is closed with three sutures through the skin. If it be thought desirable to increase the effect, a skin-flap may be excised from the lid. The objection to this operation, that a portion of the mucous membrane is removed, is not of importance. Except for an occasional granulation forming on the bulbar aspect of the wound, I have found the operation free from inconvenience, and its result satisfactory, and, in most instances, permanent.

Spastic Entropium, as the result of bandaging, usually disappears when the use of the bandage is given up, or, if the bandage must be continued and the inverted lid cause irritation, an epidermic suture at the palpebral margin and fastened to the cheek below will give relief.

Senile Entropium is, of spastic kinds, the one which most commonly demands operative interference. The methods in general use for it are:—

The Excision of a Horizontal Piece of Skin, with a portion of the underlying orbital part of the orbicular muscle, so as

to give rise to sufficient cicatricial contraction to draw the margin of the lid outwards.

The application of *Subcutaneous Sutures* (*Gaillard's Sutures*). —The point of a curved needle carrying a silk suture is entered in the centre of the lid near its margin, passed deeply into the orbicular muscle, brought out at a point some 10 mm. below, and the suture tied tightly. Two more similar sutures, one on either side of the first and about 5 mm. distant from it, are placed, and the resulting suppuration, with consequent cicatrisation, brings the lid into its position.

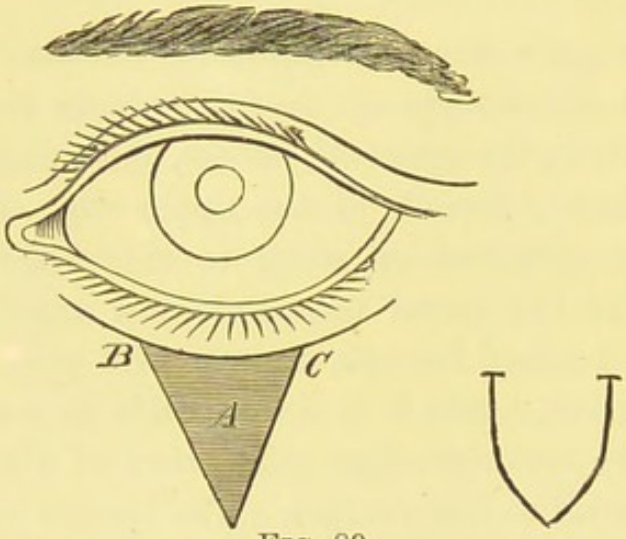


FIG. 89.

Von Graefe's Operation. —3 mm. from the margin of the lid an incision is made, as in Fig 89, through the skin, and a triangular skin flap, *A*, excised. The edges, *B* and *C*, of the triangle are dissected up a little, and brought together by three points of suture, while the horizontal incision is not sutured. The size, especially the width, of the triangular flap to be excised is proportional to the looseness of the skin. When a very marked effect is desired, the flap to be removed is given the shape as represented at the right of the figure. I have found this proceeding extremely satisfactory, and its result, as a rule, permanent.

All the foregoing, and other such measures, produce a good result at the time, but are sometimes followed by recurrence of the entropium. Hotz[1] believes the cause of this to be, that the cicatrix, be it dermic or dermo-muscular, upon which the result depends, has no *point d'appui ;* and, consequently, while

[1] *Klin. Monatsbl. f. Augenhk.*, 1880, p. 149.

it may draw the eyelid out, it is just as liable to draw the skin of the cheek up, and thus neutralise its desired effect. He proposes the following ingenious operation :—

Hotz's Operation.—A horn spatula is inserted under the lid, and then, at 4 to 6 mm. below the margin of the latter, a horizontal incision is made through the skin from the inner to the outer end of the lid. This incision is at the boundary between the palpebral and orbital portions of the orbicular muscle, and just over the lower margin of the tarsus. An assistant then draws the upper edge (Fig. 90, *a*) of the wound upwards with a forceps, while the surgeon draws the lower edge (*b*) downwards, in this way exposing and stretching the orbicular muscle. A few strokes of the knife in the direction of the incision are now sufficient to separate the palpebral portion (*l*) of the muscle from the orbital portion (*p*), and to lay bare the lower edge of the tarsus (*t*), which has a yellowish tendinous appearance. That part of the palpebral portion of the muscle which covered the lower edge of the tarsus, and which was drawn up with the palpebral edge of the first incision, is now removed with forceps and scissors, to the extent of about 2 mm. in width, through the whole length of the lid. All such muscular fibres, also, which may still adhere to the lower third of the tarsus must be carefully cleaned off, and now the palpebral skin may be brought into union with the tarsus. Four sutures are generally applied about 5 mm. apart. The needle is passed through the palpebral skin, close to the margin of the wound (at *a*). The bare tarsal edge is then seized in the forceps, the needle placed perpendicularly on it (at *d*), and carried through it by a short

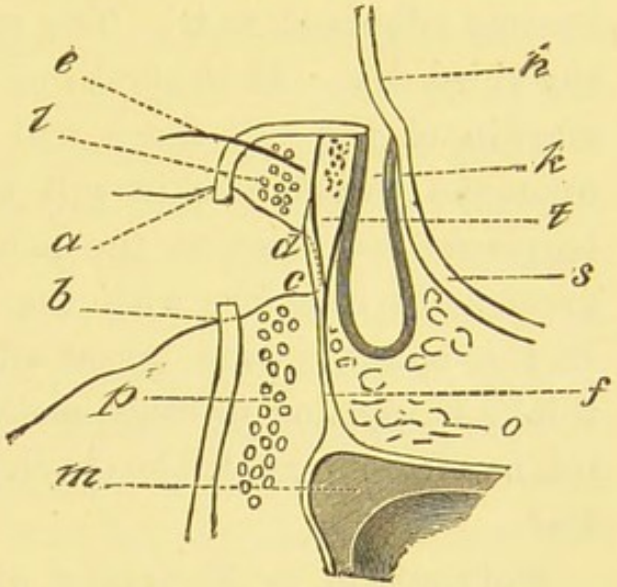


FIG. 90.

downward curve, until its point appears (at *c*) below the tarsus in the tarso-orbital fascia (*f*). The needle is now passed out through the lower edge of the incision (at *b*), care being taken that none of the fibres of the orbital portion of the muscle are included in the suture. Upon the suture being tightly closed, the edges of the skin wound are drawn into the tarsus, and become adherent to it. The sutures may be removed about the third day. If the first incision be placed too far from the margin of the lid, there will be no result, as the traction upon the palpebral skin will be too slight. If the incision be placed too close to the margin, the traction may be so great as to interfere with the union of the skin and tarsus. In this operation the tarsus affords the fulcrum, which Hotz thinks is wanting in other methods. The tarsus of the lower lid is often very little developed, and may be difficult to find.

Ectropium or **Eversion of the Eyelid.**—Of this there are two chief kinds: 1. Muscular, or Spastic; 2. Cicatricial.

Muscular Ectropium may be caused by œdema of the conjunctiva, which everts the edge of the eyelid, and this eversion is increased, and encouraged, by spasm of the palpebral portion of the orbicular muscle, so that the name palpebral paraphimosis has been given to the condition. In the recent stage it may generally be remedied by a properly applied bandage, combined with the suitable conjunctival measures. In chronic cases Snellen's sutures (*vide infra*) may be required.

Muscular ectropium is often seen in old people, and is then given the name of Senile Ectropium. Here it is due to atrophy of the palpebral portion of the orbicularis, and relaxation of the skin of the face. When these have resulted in slight eversion of the inferior punctum, a flowing of tears is produced, causing excoriations of the skin and edge of the lid, which then, in their turn, increase the tendency to ectropium. If the condition be not extreme, with secondary changes in the conjunctiva, slitting up of the canaliculus,

with the use of a boracic ointment for the lids and mild astringents for the conjunctiva, will give much relief. In pronounced cases a more active treatment of the conjunctiva, and the performance of tarsoraphy, the latter preceded by the application of Snellen's sutures, are demanded. Muscular ectropium is also caused by paralysis of the orbicular muscle.

Snellen's Sutures.—A silk ligature is threaded at either end with a needle of moderate size and curve. The point of one of these needles is passed into the most prominent point of the exposed and everted conjunctiva, and brought out through the skin 2 cm. below the edge of the lower lid. The other needle is entered in the same way, 5 mm. from the first, and made to take a nearly parallel course, the points of exit on the cheek being 1 cm. apart. Equal traction is applied to each end of the suture, while the lid is assisted into its place by the finger. The suture is tied on the cheek, a small roll of sticking-plaster having been inserted under it, to protect the skin from being cut. Two, or even three, such sutures may be required.

Argyll Robertson's Operation[1] has been designed for those cases of ectropium which result from long-continued chronic inflammation of the conjunctiva of the lower lid. He thinks the difficulty in severe cases of this kind depends upon the abnormal curvature, which is gradually acquired by the tarsus. The following is his description of the operation, from which he has obtained satisfactory results :—

The materials required are—

1. A piece of thin sheet-lead about 1 inch long and $\frac{1}{4}$ inch broad, rounded at its extremities, and with its cut margins smoothed. This piece of lead must be bent with the fingers to a curvature corresponding to that of the eyeball.

2. A waxed silk ligature about 15 inches long, to either extremity of which a long moderately curved needle is attached.

[1] *Edinburgh Clinical and Pathological Journal*, December 1883; and *Ophthal. Rev.*, February 1884.

3. A piece of fine india-rubber tubing, of the thickness of a fine drainage-tube.

The operation is performed by perforating the whole thickness of the lid with one of the needles at a point (*b*, Fig. 91) one line from its ciliary margin, and a quarter of an inch to the outer side of the centre of the lid. The needle having been drawn through (at *a*), is passed directly downwards over the conjunctival surface of the lid, till it meets the fold of

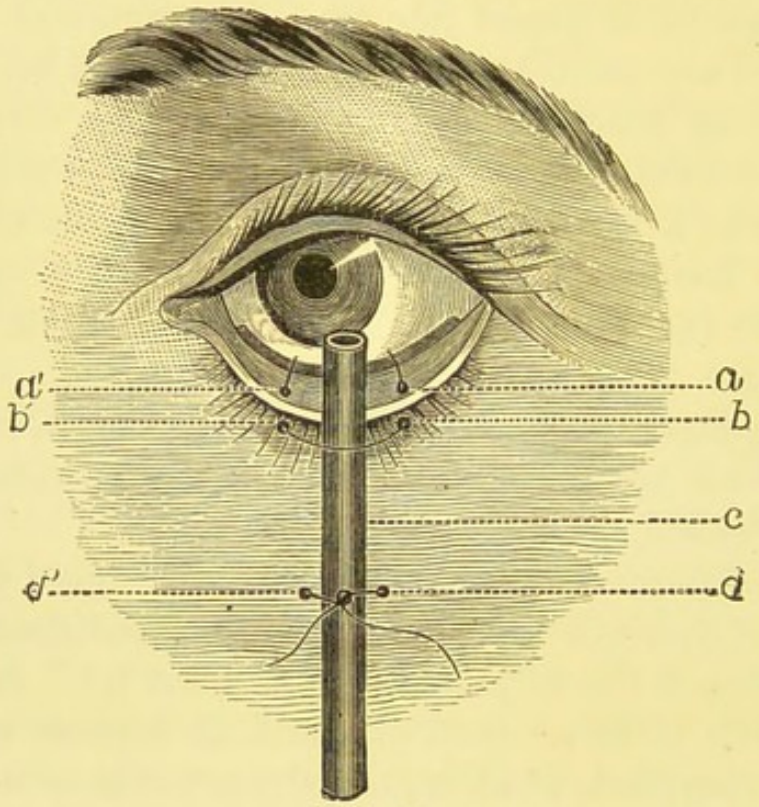


FIG. 91.

conjunctiva reflected from the lid on to the globe, through which the needle is thrust—the point being directed slightly forwards—and pushed steadily downwards under the skin of the cheek, until a point (*d*) is reached about 1 inch or $1\frac{1}{4}$ inch below the edge of the lid, when the needle is caused to emerge, and the ligature is drawn through. The other needle is, in like manner, thrust through the edge of the lid at a corresponding point (*b'*) a quarter of an inch to the inner side of the middle of the lid, then passed over the conjunctival surface of the lid, through the oculo-palpebral fold of con-

junctiva, and downwards under the skin, till the point emerges at a spot (*d'*) a quarter of an inch outwards from the point of emergence of the first needle (*d*). The ligature is kept slack, or is slackened so as to permit of the piece of lead being introduced under the loops of the ligature that pass over the conjunctival surface of the lid, and of the piece of india-rubber tubing (*c*) being slipped under the loop at the edge of the lid (between *b* and *b'*). The free ends of the ligature are now drawn tight, and tied moderately tightly over a lower part of the india-rubber tube. The excess of india-rubber tube is cut off—about three-quarters of an inch beyond the ligature—and the operation is complete.

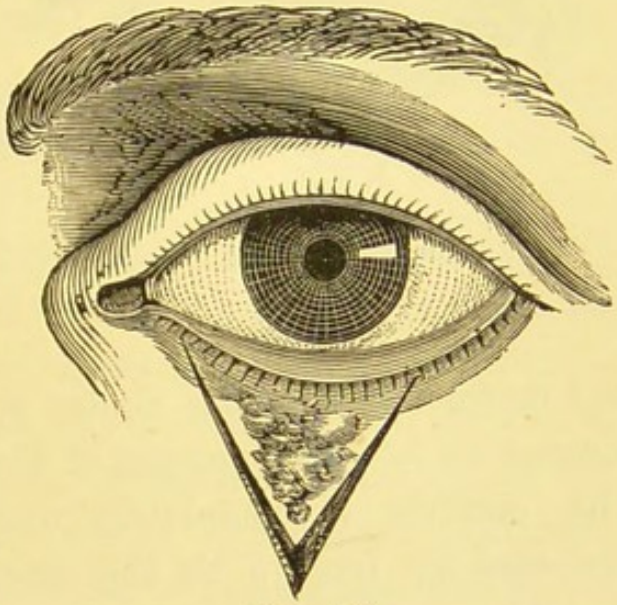


FIG. 92.

The result of the procedure is, that the edge of the lid is made to revolve inwards over the upper edge of the piece of lead, while the tarsus is caused to mould itself to the curve of the lead, and the eyelid at once occupies its normal position. A certain amount of redness and œdema of the lid follows the operation, and suppuration occurs in the track of the ligature; but, as the india-rubber tube yields somewhat to the tension on the ligature, the resulting irritation is moderate, so that the apparatus need not be removed for five, six, or seven days, by which time the tarsus has become pretty well fixed in its new curvature. A slight relapse may occur when the apparatus is removed, but this is readily amenable to treatment by astringent applications.

The suppuration occurring in the tracks of the ligature leads to cicatricial formation, which appears to impart a degree of rigidity to the lid, that helps to keep the latter in its new position.

Cicatricial Ectropium is caused by scars from wounds or burns, or from caries of the orbit, and can only be cured by operation.

Wharton Jones's Operation is as follows:—The cicatrix is circumscribed by a V-shaped incision (Fig. 92), and the skin made thoroughly movable in its neighbourhood. The edges of the wound are now brought together so as to form a Y (Fig. 93).

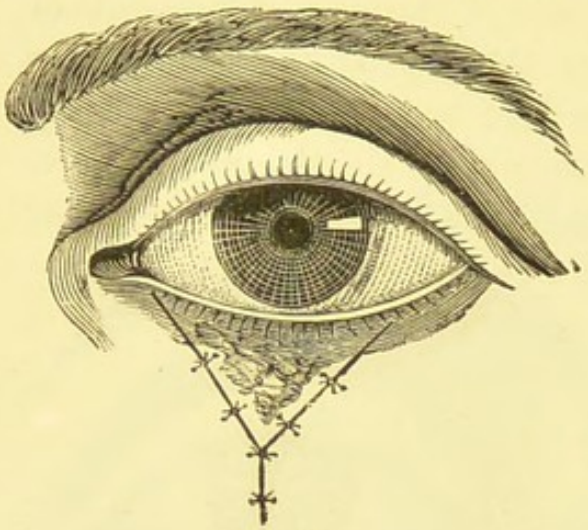


FIG. 93.

Arlt's Operation, for cases due to caries of the margin of the orbit.—If the cicatrix be situated at *e* (Fig. 94), the incisions at *a b* and *b c* are made through the skin and muscle, so that an acute, or at most a right, angle is formed at *b*. The margin of the lid from *c* to *d* is excised. The cicatrix is completely undermined, and the triangle dissected up from *b* to the margin of the tarsus, so that the lid can be readily put into its position, and the edge *c b* of the flap united to *d c*. The size of the exposed surface on the cheek can, according to Arlt, be diminished by drawing its edges together after the manner of a hare-lip, but possibly the transplantation of a piece of skin from the arm to fill the gap might be a better plan.

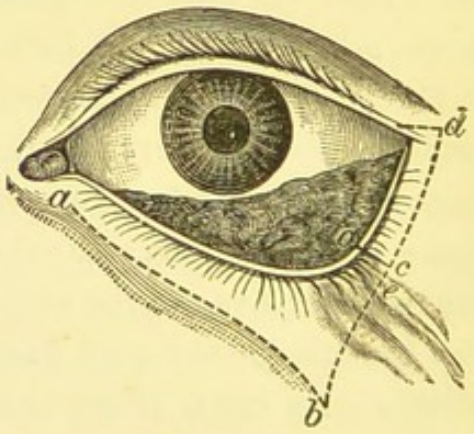


FIG. 94.

The foregoing and similar operations are difficult, or impossible, in many cases, where there has been great destruction of the skin of the eyelids and surrounding parts by burns,

ulcers, etc., and, at best, the deformity is liable to recur. Transplantation of skin from different parts is in these cases a more promising proceeding. A description of the method is given in the next paragraph but one.

Ankyloblepharon (*αγκύλη, a string; βλέφαρον, an eyelid*) is a uniting of the upper and lower eyelids along their margins. It may be partial or complete, and often goes with symblepharon. Like the latter, it is usually caused by burns and ulcers.

The condition can only be relieved by operation, of which the result is often unsatisfactory, owing to the difficulty of preventing reunion taking place. To avert this, it is always necessary to cover the wounded surface with conjunctiva or skin.

The Restoration of an Eyelid.—It is an extremely rare event for the whole substance of one or both eyelids to be destroyed by lupus, or other ulceration, or by accidents, which do not at the same time injure the eyeball seriously. In this rare event, the eyeball, especially if the upper lid be destroyed, is exposed; the patient is subject to extreme discomfort; and, owing to ulceration of the cornea, the eye is ultimately lost.

The formation of an eyelid from the skin of the forehead or cheek in these cases is a most disappointing proceeding, and one the description of which does not, I consider, come within the scope of this book. Indeed, my own feeling, in such a case, would be, to recommend enucleation of the eyeball, provided the fellow eye were good, rather than propose a plastic operation which, at the best, would give but an imperfect result.

But, fortunately, the class of cases with which we commonly meet are essentially different in their nature; for in them the whole thickness of the eyelid is not destroyed. They are usually the result of burns (epileptics and children falling in the fire) and scalds, which only destroy the integument of

one or both eyelids. A granulating surface replaces the skin, and, when healing commences, the shrinking process draws the free margin of the upper eyelid up towards the eyebrow, and that of the lower lid down towards the cheek, while the conjunctival surface of the eyelids becomes everted, and the cornea exposed, as the eyelids cannot now be closed. We have a satisfactory method for dealing with these cases.

In the first place, the eyelid—let us suppose it to be the upper eyelid—is dissected down into its place to the utmost limit, so that the most extensive raw surface possible may be obtained. The margin of the lid is now fastened to the cheek with three points of suture. A portion of skin, one-third larger (to allow for shrinkage) than the raw surface of the eyelid, is then taken from the inside of the arm, and, after being freed of its subcutaneous fat, is laid upon the raw surface and fastened to it by a large[1] number of fine sutures around the margin. A non-irritating antiseptic dressing is applied, and the graft usually heals on in the course of a few days. This method of grafting was introduced by Wolfe and Lefort.

The flap sometimes becomes separated from the wounded surface by oozing of blood or serum from the wound, and then sloughs. To prevent this, Wickerkiewicz has employed secondary transplantation with satisfactory results. The flap is applied to the wounded surface, from two to five days after the latter has been prepared, while, during the interval, the wounded surface has been protected with moist antiseptic dressings. He states that union by first intention occurs readily by this method.

In this operation it is most important to preserve and utilise any part of the eyelid which remains, especially its ciliary border with the eyelashes.

[1] Yet sutures have two drawbacks: in the first place, they tend to injure the edges of the flap, and in the next, they cause a certain amount of bleeding under it.—L. W.

Injuries of the Eyelids.—All kinds of injuries of the eyelids—contusions, incisions, burns, etc.—are common.

In consequence of the looseness of the integument, œdema and ecchymosis, one or both, are often seen in a marked degree as the result even of slight injuries.

Owing to the direction of the fibres of the orbicularis, an incised wound of the eyelid, if in the vertical direction, will gape, while a similar wound in the horizontal direction will not do so. Hence, the scar left after the former wound is apt to be very visible, but that after the latter may be almost imperceptible. If the eyelid be divided vertically in its entire thickness, unless union by first intention can be obtained, a deep furrow is left in the eyelid, and, perhaps, at its margin an unsightly coloboma.

Emphysema of the eyelids is sometimes seen after a blow on the eye, and is a sign of fracture of the orbit, with a communication between the subcutaneous connective tissue of the eyelids and the nose, the ethmoid sinus, the frontal sinus, or the antrum of Highmore. An emphysematous lid is swollen, and soft and crepitating to the touch.

Ecchymosis of the lower lid, usually with ecchymosis of the lower conjunctiva, after falls or blows on the head, is a sign of fracture of the base of the skull, the blood making its way along the floor of the orbit.

Simple ecchymosis of the eyelids from blows, commonly known as "Black Eye," never gives rise to further complication. It requires some fourteen days or more, according to the quantity of blood extravasated, before the eye recovers its normal appearance.

Treatment.—Injuries of the eyelids, of whatever kind, are, of course, treated upon general surgical principles. Incised wounds should be carefully and neatly drawn together with sutures, as soon after the injury as possible, and with antiseptic precautions. Emphysema may be assisted in its absorption by the application of a rather tight bandage, and

directions should be given to the patient to blow his nose as gently as possible, so as to avoid recurrence of the emphysema.

Epicanthus is a congenital deformity, usually binocular, which, in the most pronounced cases, consists in partial paralysis of the levator palpebræ (ptosis) and of the rectus superior, with a narrow palpebral fissure, and a fold of integument at the inner canthus concealing the caruncle from view, and giving the appearance of great breadth to the bridge of the nose. The term is also used for cases in which the integumental fold at the inner canthus is the only abnormal condition, and this deformity can be somewhat diminished by the removal of an oval piece of skin from the bridge of the nose, its long axis vertical, and its width varying according to the effect required. The margins of the wound being brought together, the abnormal folds are diminished in width.

Congenital Coloboma of the upper lid, sometimes associated with a dermoid cyst of the limbus of the cornea corresponding to the cleft in the lid, and even congenital absence of the eyelids, have been occasionally observed.

CHAPTER VIII.

DISEASES OF THE LACRIMAL[1] APPARATUS.

Malposition of the Punctum Lacrimale.[2]—Inversion of the punctum accompanies entropium of the lower eyelid, while eversion of it is present with ectropium of the lid. A slight eversion, quite sufficient to cause epiphora, may exist without any marked ectropium of the lid, and it is these cases which more properly belong to this chapter. They are the result generally of some chronic, although it may be slight, skin affection of the lower lid, which draws the inner end of the latter a little away from the eyeball.

The prominent symptom of this, and of all the following lacrimal affections, is Epiphora (*ἐπιφορά δακρύων, a sudden burst of tears*), a flowing of tears over the cheek.

Stenosis, and **Complete Occlusion of the Punctum Lacrimale.**—Either of these conditions may result from conjunctivitis or from marginal blepharitis, although they may not appear for a length of time after those affections have passed away, and the original affection may have been so slight as to have escaped the observation of the patient. In stenosis, the size of the punctum may become so extremely minute, that even the normal flow of tears is too great to make its way through it. Complete occlusion is, probably, only a more advanced stage of stenosis.

The Treatment, in cases of eversion of the punctum, of stenosis and of complete occlusion, is similar, namely, the

---

[1] *Lacrima,* a tear.

[2] In this chapter, and elsewhere in the book, the terms "punctum lacrimale" and "canaliculus" refer to the inferior passage, unless it be otherwise expressly stated.

opening up of the punctum, and its conversion into a slit. This is done with a Weber's knife (Fig. 95), the probe-point of which is passed into the punctum in cases of eversion, forced into the small opening in cases of stenosis, or forced through the usually thin covering of the punctum in cases of occlusion. In doing this, the lower lid should be stretched tightly, by a finger of the surgeon's left hand placed near the external canthus. The edge of the knife being now directed towards the eyeball, the instrument is pushed on a little into the canaliculus, until 2 mm. of the latter has been opened up, and it is then withdrawn. If the edge of the knife be directed outwards in this proceeding, the incision comes to lie on the outer edge of the intermarginal portion of the lid, and not in contact with the eyeball; consequently, the tears are not carried away, and the disfigurement produced is considerable. A slitting up of the whole, or the greater part, of the canaliculus in these cases is unnecessary, and interferes with the physiological action of the tear passage. For two or three days after the little operation, it is necessary to pass a probe along the portion of the canaliculus which has been slit up, to prevent union taking place.

FIG. 95.

Obstruction of the Canaliculus.—The canaliculus may be diminished in its calibre, or entirely closed, by contraction, the result of inflammation which had extended to it from the conjunctival sac. It is not possible to diagnose the presence of either of these conditions, which may be associated with stenosis or occlusion of the punctum lacrimale, except by the introduction of a very fine probe into the canaliculus. The passage may also be obstructed by an eyelash, a chalky deposit, or a mass of leptothrix.

Treatment.—Where there is merely diminution in the calibre

of the passage, the introduction of probes, increasing in size, is frequently sufficient to effect a cure. Dilators, on the same principle as Holt's instrument for the dilatation of urethral strictures, have been employed. If dilatation fail, recourse must be had to slitting up the canaliculus; but, if it can possibly be avoided—that is, if a less extended opening will answer—the passage should not be slit up in its entire length. At least 3 mm. of its median end ought to be left intact, as otherwise regurgitation of tears from the lacrimal sac is liable to trouble the patient ever afterwards. If the canaliculus be completely closed by adhesions, so that a fine probe cannot be pushed through it, it becomes necessary to rip it up with the point of any small knife, following the known course of the passage from the outside. If the canaliculus be closed as far as the opening into the sac, or if only at that point, the obstruction must be pierced with the point of a fine knife. A great difficulty in all these cases is to keep the passage patent, when once formed. A plan which affords tolerable certainty of this is the frequent passage of probes into the sac, until the tendency to closure seems to have ceased; but, even under favourable conditions, recurrences of the closure are apt to occur. In order to cure this condition, and in the hopes of doing so permanently, Dr. W. E. Steavenson and Mr. Walter Jessop[1] have employed electrolysis, which they apply to the canaliculus by means of a platinum probe fitted in a handle, and connected with the negative pole of a Stöhrer's battery. A flat electrode connected with the positive pole is placed on the back of the neck. A current of two to four milliampères is sufficient, and the operation lasts thirty seconds. By this procedure the canaliculus is rendered wide enough, but time has yet to show whether recurrence of the stricture is less frequent than after treatment by other methods.

[1] *Brit. Med. Journal*, December 24th, 1887.

Stricture of the Nasal Duct is usually the result of swelling of its mucous membrane in catarrhal attacks, or of membraneous or cicatricial contraction resulting from long-continued catarrh. It also occurs in consequence of disease of the bones of the nose, *e.g.* in syphilis, acquired or congenital, and from blows which fracture the bridge of the nose.

Treatment.—Bony stricture may be regarded as incurable. Stricture due to inflammatory swelling of the mucous membrane, also membraneous or cicatricial strictures, are best treated by means of probes, in the manner proposed by Sir William Bowman. The inferior canaliculus is slit up to a slight extent, so as to admit the point of one of Bowman's smallest probes, which is given a curve to suit that of the nasal duct. With the fingers of the left hand the surgeon stretches the lower lid, and, entering the probe into the canaliculus, pushes it gently along its floor, until the point reaches the lacrimal bone forming the posterior wall of the sac. The point being kept pressed against this bone, the direction of the probe is now altered by carrying its free end upwards towards the bridge of the nose, until the point at the other end, in the lacrimal sac, is directed towards, or aimed at, the sulcus between the ala of the nose and the cheek. The probe then is in a position corresponding to the prolonged axis of the nasal duct, down which it is pushed with a slow and gentle motion. Any obstacles met with on the way are overcome, if possible, by an increase of the pressure; but if, at any part of the proceeding, much difficulty be encountered, rather than that any violence be used, all further manipulation should be postponed to another day; and it will often be found that, at the second or third visit, the probe is passed with comparative ease. Thicker probes are gradually introduced at successive sittings, until the largest size has been reached.

The most common seats for stricture of the nasal duct are at its entrance into the sac, where it is narrowest, and at its

lower end, where it is most exposed to catarrhal processes in the nostril.

Where there is reason to think that the stricture is due to chronic catarrhal swelling of the lining mucous membrane of the duct, astrigent injections into the canal, in addition to the probing, are of use.

Otto Becker used very fine probes, which he passed by the upper canaliculus. Weber's probes are conical, and of very large calibre at their thickest part. Their inventor passes them by the superior canaliculus, but many other surgeons pass them by the lower. I do not employ these probes, because, when passed into the nasal duct, their thickest part, which is 3 to 4 mm. in diameter, corresponds with the upper end of the duct, which is its narrowest part, being only 3 mm. in diameter: consequently, the probe becomes more or less impacted at this place at each operation, and is apt ultimately to give rise there to hypertrophy of the periosteum, and finally to stricture; so that, while the immediate effect of their use is good, the ultimate result is often the reverse. When used by the inferior canaliculus their size makes it necessary to slit that passage in its entire length, and the entrance of the passage into the sac must be enormously dilated by so large an instrument, both of which circumstances are most undesirable.

To prevent closure of the duct when once made free, Dr. Arthur Benson (Dublin) advocates the use of leaden styles, removable by the patient. He first divides the canaliculus, by preference the upper one, and dilates the stricture with probes in the ordinary way, and then introduces into the duct a piece of leaden wire 1·5 mm. to 2 mm. in diameter, cut to length, and smoothed off at the ends. The upper end is curved so as to lie out on the cheek. This style is at first removed daily, and the duct syringed, until any existing inflammation and discharge have almost ceased. The intervals are then increased; and, as soon as practicable, the patient

is taught to remove the style and to replace it himself. When he is able to do this easily, he is directed to leave the style out for some hours each day, and finally to wear it only at night.

Stilling has proposed an operation, which he calls stricturotomy, for the cure of membraneous obstructions in the duct. Having slit up the canaliculus, and ascertained with a probe the position of the stricture, Stilling passes his knife, with the cutting edge directed forwards, down the duct and through the stricture; he then withdraws it a little, turns the edge in another direction, and pushes it again through the stricture, and performs this manœuvre a third time before removing the knife. On subsequent days large probes are passed. This method has never gained much popularity.

Very obstinate strictures can sometimes be freed by electrolysis.

The most favourable cases of stricture for cure are those due to inflammatory swelling of the mucous membrane, and next in order come those caused by membraneous or cicatricial contraction, while those due to bony obstructions must, as already stated, be regarded as incurable.

Now and then, cases of persistent lacrimation will be met with, in which the nasal duct, and the rest of the lacrimal apparatus, are in perfect order. These are often due to a catarrhal affection of the nasal mucous membrane, slightly involving the very lowest extremity of the nasal duct. Here applications directed towards relief of the nasal affection are indicated.

Blennorrhœa of the Lacrimal Sac is commonly caused, in the first instance, by stricture of the nasal duct. In consequence of this stricture, the tears, and the normal mucous secretion of the lining membrane of the sac, are retained there, and offer favourable conditions for the development of the micro-organisms, which are constantly present on the surface of the eye, and are carried into the lacrimal sac by the tears. These decomposing contents of

the sac, then, set up inflammation of its mucous membrane, with discharge of a muco-purulent nature.

But one, not seldom, comes across cases of lacrimal blennorrhœa, where, upon examination, no stricture of the nasal duct is found. Yet, in many of these cases there has been a stricture due merely to catarrhal swelling of the lining membrane of the duct, which swelling has subsided in the course of time without treatment, and the duct has then again become free, while still the lacrimal blennorrhœa, to which the stricture gave rise, continues. It is very probable, however, that lacrimal blennorrhœa may occasionally come on, where there has never been a stricture of the nasal duct, and merely as an extension of catarrh from the nostrils, especially in cases of ozæna, or as an extension of catarrh from the conjunctiva.

Symptoms.—The patients usually complain of nothing more than epiphora. Those who are more observant of themselves may have noticed a swelling, which we call a "lacrimal tumour," or "mucocele," in the region of the lacrimal sac; and, also, that the conjunctival sac, especially when the swelling is pressed upon, becomes, now and then, more or less filled with a somewhat thick and opaque discharge, which obscures the sight until wiped away. Occasionally there is no lacrimal tumour, for the contents of the sac may not be copious enough to bulge it out.

In order to ascertain in each case of epiphora whether or not lacrimal blennorrhœa be present, the surgeon presses with his finger over the lacrimal sac, when, if there be blennorrhœa, the discharge will be evacuated through the puncta into the conjunctival sac. In those cases in which there is no longer a stricture of the nasal duct, the discharge may pass downwards into the nose, and the patient will feel it in his nostril, and can blow it out of the latter.

Conjunctivitis must sometimes be regarded, not as the cause, but rather as the effect, of a lacrimal blennorrhœa,

by reason of the decomposing discharge from the sac making its way into the conjunctival sac. Blepharitis, too, is seen as a further result of irritation from the discharge, in old-standing cases.

Treatment.—It is important, in the first place, to ascertain whether there be a stricture of the nasal duct, and for this purpose water should be injected by means of an Anel's syringe through the canaliculus into the duct. If the fluid make its way freely into the nose or pharynx, it may be taken for granted that the nasal duct is not obstructed; but if, instead of passing through—or only under high pressure—it distend the lacrimal tumour to a greater size, a stricture may be assumed. If stricture of the nasal duct be present, it must be relieved, or all other measures will prove futile. Should there be no stricture, and also before and after any existing stricture has been freed, the treatment consists in the very frequent pressing out of the contents of the sac by the patient, so that no distension of it may occur; and, in this manœuvre, he should endeavour to cause the discharge to pass down the nose, rather than into the eye; while the surgeon, having, if necessary, dilated the canaliculus, injects astringent solutions (sulphate of zinc, nitrate of silver, alum, sulphate of copper) into the sac daily, to relieve the catarrh.

The caustic treatment, recommended farther on for acute dacryocystitis, is often of the greatest benefit in these chronic cases. Any existing conjunctivitis, or nasal catarrh, should be treated.

Acute Dacryocystitis (*δακρύω, to weep; κυστις, a bladder*).—Acute inflammation of the lacrimal sac most usually comes on, when chronic lacrimal blennorrhœa is already present. Caries of the nasal bones may cause it, and it occurs idiopathically, probably as the result of exposure to cold.

The region of the lacrimal sac, and the surrounding integument, become swollen, tense, and red, and these conditions often spread to the lids, giving an appearance which may be

readily mistaken for erysipelas; but the history of the case, showing the previous existence of lacrimal obstruction, etc., will assist the diagnosis. Great pain accompanies the inflammatory process. Gradually, the region corresponding to the lacrimal sac becomes the most prominent one of the swelling, and the abscess, pointing there, opens. When the pus has been discharged, the inflammation subsides, and the opening through the skin may either close, the parts resuming their normal functions, or the opening may remain as a permanent fistula.

The difference between chronic blennorrhœa of the lacrimal sac and acute dacryocystitis, besides the fact that one is a chronic and the other an acute inflammatory process, is that the former process is confined to the mucous membrane of the sac, while in the latter the submucous tissue is involved, with phlegmonous inflammation as the result.

Treatment.—In the early stages poultices and purgatives should be employed. As soon as palpation of the sac indicates the presence of pus, it must be evacuated. This can be effected, either through the canaliculus, by opening it up to its entrance into the sac, or by an incision through the integument over the sac. The latter is the method I prefer, as it admits of free access to the interior of the sac. The day afterwards, the walls of the sac are to be freely touched with solid mitigated nitrate of silver; or, a plug of cotton wool soaked in a strong solution of nitrate of silver may be inserted into its cavity, and left there for some hours; or, various astringent solutions may be injected into the sac. The aim of the treatment, whatever it may be, is to secure a rapid return of the mucous membrane to its normal condition. If stricture of the nasal duct be present, it must be treated *pari passu*. By these means, the discharge from the sac is arrested, and the external opening closes up.

If a fistula should form, it may be induced to close, in many cases, by simply freeing an existing stricture of the

nasal duct. Or, it may be necessary to pare its edges, and bring them together by sutures. Or, especially if there be a long fistulous passage, the galvano-cautery, in the form of a platinum wire, can be applied with advantage.

Obliteration of the sac may have to be brought about in some very chronic cases, where repeated attacks of acute inflammation, and fistula, occur; or, where there is constant discharge, and disease of bone; and, when all other methods have failed to relieve the patient. This can be done by the application of a galvano-cautery to the lining membrane of the sac, or by dissecting it out. But I must say that, in my experience, obliteration of the lacrimal sac is one of the most difficult undertakings in ophthalmic surgery.

Removal of the lacrimal gland, or excision of the palpebral portion (de Wecker), is sometimes performed for the relief of incurable epiphora. The palpebral portion can be removed from the conjunctival surface. It can be seen in the upper cul-de-sac by separating the eyelids widely at the outer canthus, while the patient looks well down and to the nasal side.

Dacryoadenitis (*δακρύω, to weep; ἀδήν, a gland*), or **Inflammation of the Lacrimal Gland,** occurs both in an acute and in a chronic form, but is extremely rare in either. I have seen one case of acute purulent dacryoadenitis, but no instance of the chronic affection. Swelling and hyperæmia over the gland, and of the whole lid, with chemosis of the conjunctiva, and much local pain, increased on pressure, are the most marked symptoms of acute dacryoadenitis. When suppuration has taken place, the abscess may open into the conjunctiva, as it did in my patient, or through the skin. In the latter case it is liable to leave a fistula behind it, and indeed the chronic form may also, it is said, lead to fistula.

Numerous cases of chronic enlargement of both lacrimal glands have been recorded. Good results have been obtained

by administration of potassium iodide or mercury in some cases.

Treatment in the early stages consists in poultices and purgatives. When pus has formed, the abscess may be opened through the skin, or from the conjunctiva.

Hypertrophy of the Lacrimal Gland is also of rare occurrence. It may attain such dimensions as to push the eyeball out of its position. It can only be dealt with by—

Extirpation of the Lacrimal Gland.—This is performed by making an incision through the integument under the outer third of the orbital margin ; the fascia under this is dissected up, the gland drawn out with a hook, and dissected out with a scalpel.

Adenoma, Adeno-Sarcoma, Lymphoma, and **Tuberculosis** of the lacrimal gland have been observed occasionally in the same order of frequency as here mentioned.

CHAPTER IX.

DISEASES OF THE SCLEROTIC.

Inflammation of the sclerotic is not a common disease, although the diagnosis "scleritis" is often made by inexperienced persons, every "redness of the white of the eye" being taken for inflammation of the sclerotic. Beginners are warned against this error. Iritis, cyclitis, and conjunctivitis, as well as scleritis, cause redness of the white of the eye.

The diagnosis from conjunctivitis is easily made by observing whether the conjunctival vessels can be moved over the affected part or not; while in iritis and cyclitis the ciliary injection is confined to the part immediately surrounding the cornea. Moreover, in iritis the appearances of the iris itself are conclusive; and in scleritis, as will just now be seen, the appearances are characteristic.

Scleritis attacks only that part of the sclerotic which is anterior to the equator of the eyeball, and is either superficial or deep. The superficial form is known as episcleritis. Yet it is not always possible to distinguish between these two forms in a given case, as the appearances in the early stages are very similar. They are probably only different degrees of the same disease. But the necessity of admitting the existence of two forms depends upon the different course they each take; the superficial form being a relatively harmless disease, while the deep form entails serious consequences.

Episcleritis appears as a circumscribed purplish, rather than red, spot, close to, or 2 to 3 mm. removed from, the corneal margin. It is often unattended by pain, unless

when the eye is exposed to irritating causes, and need not be elevated above the level of the sclerotic; but, in severe cases, there is a decided node at the affected place, with pain, more or less pronounced, and increased on pressure. All the symptoms disappear in the course of a few weeks, and reappear at an adjoining place; and, in this way, in time, the whole circumference of the sclerotic will have been attacked. The duration of the affection is usually long; and, in those instances where the entire sclerotic becomes affected by degrees, the process may last for years, on and off. Both eyes are often affected. The disease is liable to leave behind it a dusky discoloration of the sclerotic where each node was seated, but otherwise no harm to the eye ensues. But the patient should be made acquainted with the tedious nature of the affection. Very mild attacks of episcleritis will be met with, which pass away in a few days, and do not recur.

Causes.—The affection is often of rheumatic origin; it occurs sometimes in persons of scrofulous or syphilitic constitution; and it is more frequent in senior adults than in children or young people, and more commonly attacks women than men.

Treatment.—No irritant should be applied to the eye. Local treatment should be confined to warm fomentations and protection. In addition to these, massage should be used, if there be not too great tenderness on pressure. Leeching at the external canthus is of use when the pain is severe. As regards internal remedies, where a syphilitic taint is present, mercury should be employed; if struma, cod-liver oil, maltine, etc.; or if, as is most frequently the case, the rheumatic taint be the source of the evil, large doses of salicylate of sodium, say 20 grains four times a day, will often be found to act well. Salicylate of lithium is recommended in preference to the sodium salt by some. Iodide of potassium, and hypodermic injections of pilocarpine, are useful

remedies in some cases of this obstinate disease. Subconjunctival injections of corrosive sublimate, or of salicylate of soda, have also been recently tried.

Deep Scleritis.—Here the whole sclerotic is more likely to be affected at once than in the milder form; although cases often enough occur where only an isolated node is present at a time.

It is the progress of the case alone which can render the diagnosis certain, and hence the importance of a guarded prognosis in the early stages of every case of scleritis. In the deep form, changes—thinning and softening—of the scleral tissue take place, which render the latter less resistant, and consequently, expose it to distension by even the normal intraocular tension. The result of this is a bulging (staphyloma) of the anterior part of the eyeball. This bulging in itself produces myopia, and has a deleterious effect upon the sight; but, at a later period, vision is often wholly destroyed by secondary glaucoma. It may happen that the thinning, etc., of the sclerotic affects only a portion, and not the whole, of its anterior surface; and, in such a case, the resulting staphyloma will be confined to that part of the sclerotic. A staphyloma, whether total or partial, presents a bluish-grey appearance, due to the shining through the thinned sclerotic of the uveal tract.

Either with or without such staphylomatous changes, sclerotising opacity of the cornea may come on, and iritis, chorioiditis, and opacity of the vitreous humour are not un common complications, especially in strumous subjects. Both eyes are usually affected.

Causes.—Young adults are the most common subjects of deep scleritis, and females more often than males. Congenital syphilis, rheumatism, struma, and disturbances of menstruation are the most common assignable causes.

Treatment.—There are few diseases less amenable to treatment. When any of the above causes can be assumed

to be present, the suitable remedies are, of course, indicated. Besides this, warm fomentations, dry cupping on the temple, or the artificial leech, complete rest of the eyes, and protection with dark glasses are to be recommended.

When all acute inflammation has passed away, an iridectomy is sometimes indicated—either for optical purposes, when the pupil is obstructed by corneal opacity, or for the purpose of reducing glaucomatous tension, or of diminishing a staphyloma.

Injuries of the Sclerotic.—Ruptures and perforating wounds are those which have to be considered. Mere losses of substance may be said not to occur.

The primary danger of a rupture or perforating wound of the sclerotic—apart from the loss of contents of the eyeball, which is often associated with it—consists in the possibility of infecting organisms being introduced into the interior of the eye, and there setting up serious inflammatory reaction.

A large and gaping wound is easily recognised. A portion of the chorioid, ciliary body, or iris, according to the position of the wound, probably lies in it, or part of the vitreous humour may be found in it; while the vitreous humour, as seen through the pupil, will be full of blood (hæmophthalmos), and blood may be present in the anterior chamber (hyphæma, ὑπὸ, *under*; αἷμα, *blood*), especially if the wound be far forwards. Small wounds may be concealed by subconjunctival hæmorrhage, and here reduced tension of the eyeball is sometimes a valuable diagnostic sign.

Clean-cut perforating wounds of the sclerotic often heal without inflammatory reaction, even when portions of the uveal tract, or vitreous humour, are prolapsed into it, these prolapsed parts becoming incarcerated in the cicatrix. Even irregular ruptures of the sclerotic from blows, with prolapse of uvea, and vitreous humour, and, as sometimes occurs, evacuation of the lens, may heal without inflammatory reaction. It may here be mentioned, that these ruptures

from blows almost always occur close to the corneal margin, and concentrically with it, and lie usually near its upper, or upper and inner, margin. And one often sees the conjunctiva remain intact over the rupture, with perhaps the lens dislocated under it.

When inflammatory reaction follows upon one of these injuries, it may either be of the purulent or plastic form. In the former case all the contents of the eyeball take part in the suppuration, and we term it panophthalmitis ; phthisis bulbi being its ultimate result. In the plastic form the iris and ciliary body alone are implicated, and sight is slowly lost ; the eye here, too, becoming phthisical. Of the two, the latter process is the more serious, as it may give rise to sympathetic ophthalmitis, a danger which is not associated with the eye lost through panophthalmitis.

Where the wound has been produced by a small foreign body, which has remained in the interior of the eye, the seriousness of the position is much aggravated ; but the discussion of this matter will be treated of in chap. xiv., on Diseases of the Vitreous Humour.

Treatment.—In cases where the wound is small, no suture need be applied ; a bandage will be sufficient to promote the natural tendency to healing. But, where the wound is large and gaping, any prolapsed chorioid, etc., should be first freely irrigated with sublimate lotion, 1 in 5000, and reduced as well as possible, and then the margins of the wound drawn together by a few points of suture in the sclerotic ; or, better still, by passing the sutures through the conjunctiva, at some distance from the edges of the wound. The traction on the conjunctiva is there sufficient to close the scleral wound. A bandage is applied, and the patient kept quiet in bed. But if the injury be such—very wide wound, much loss of contents of the eyeball, or extensive intraocular hæmorrhage—as to render restoration of useful sight beyond reasonable hope, it will be wiser to remove the eyeball at once, rather than run

the risk of sympathetic ophthalmitis without compensating advantage.

Tumours of the Sclerotic, as primary growths, are almost unknown; but fibroma, sarcoma, and osteoma have been so observed.

Pigment Spots of a yellowish-brown colour are often seen in the sclerotic, close to the corneal margin. They are congenital, and of no importance. Occasionally a black pigmented patch may be associated with pigmented sarcoma of the ciliary region.

CHAPTER X.

DISEASES OF THE UVEAL TRACT.

(Iris, Ciliary Body, and Chorioid.[1])

If it be remembered that the iris, ciliary body, and chorioid closely resemble each other histologically, that their blood supply is identical, and that they form with each other a continuous membrane; it is a matter of surprise to learn, that any one of these three divisions of the uveal tract can undergo inflammation, while the other two remain perfectly healthy. Yet this is, by no means uncommonly, the case. But it is, perhaps, more common for at least two of them, and especially the iris and ciliary body (*irido-cyclitis*), to be simultaneously inflamed; and the entire tract may, of course, be affected at once. Clinically we cannot always know whether only one, or more than one, division of the uveal tract is in a state of inflammation, and this uncertainty of diagnosis is particularly liable to arise, when there is severe acute iritis; for then the symptoms present might all be derived from the iritis alone. It may be taken for granted, that in every rather severe case of iritis, particularly in those of syphilitic origin, more or less cyclitis is also present; while a deep anterior chamber, or tenderness on pressure, increases the suspicion. In slight cases of iritis there will, probably, be no cyclitis.

It is only after the acute inflammatory symptoms have subsided, and the pupil has become clear, that disseminated changes in the chorioid, opacities in the vitreous humour, and even retinitis and optic neuritis, which may lead to optic

[1] χόριον, *a skin, the chorion;* hence chorioid, *like the chorion.*

atrophy, can be discovered, with their corresponding depreciation of vision.

It is desirable, in a systematic consideration of inflammation of the uveal tract, to discuss it under the separate headings of the iris, ciliary body, and chorioid; and the same remark applies to the other diseases, and to the injuries, of this tunic.

IRITIS.

The most rational division of the different kinds of iritis is that founded on their pathology, namely: (1) *Simple Plastic Iritis;* (2) *Serous Iritis;* and (3) *Parenchymatous* (including *Purulent*) *Iritis.*

Their *Common Characteristics*, more or less marked, are:—

Discoloration, loss of lustre and of distinctness of pattern, and functional disturbances (impaired mobility) of the iris, with contraction of the pupil. The loss of lustre and of distinctness of pattern is due to an alteration in the endothelium, which covers the surface of the iris, to the presence of lymph, and to cloudiness of the aqueous humour. The change in colour is due to hyperæmia of the iris, as well as to the presence of the inflammatory products; a blue iris becomes greenish, a brown iris yellowish. The impaired mobility and the contracted pupil are due to the hyperæmia, to spasm of the sphincter iridis, and to posterior synechiæ.

Exudation of inflammatory products is present, in greater or less degree, in all of these forms, and is found:—(1) on either surface of the iris and in the pupil, in plastic iritis; (2) in the aqueous humour and posterior surface of the cornea, in serous iritis; (3) in the tissue of the iris, in parenchymatous iritis.

Posterior synechiæ (*συνέχειν, to bind together*)—*i.e.*, adhesions between the iris and the anterior capsule of the lens—occur as the result of inflammatory exudation on the posterior surface, or on the pupillary margin, of the iris. The presence of posterior synechiæ is ascertained by observing

the motions of the pupil, when the eye is placed alternately in strong light and in deep shadow; or, by observing the effect of a drop of atropine solution on the pupil, the latter dilating only at those places where there are no synechiæ. If the entire pupillary margin have become adherent, the condition is termed complete posterior synechia, circular posterior synechia, ring synechia, or "exclusion" of the pupil; and in such cases, if of some standing, atropine has no effect on the pupil. If the area of the pupil be filled with exudation, circular synechiæ being usually also present, the condition is known as "occlusion" of the pupil. Total posterior synechia is that condition in which the whole posterior surface of the iris is adherent to the capsule of the lens.

In addition to the foregoing, circumcorneal injection of the ciliary vessels is a common symptom in most cases of iritis.

The subjective symptoms in iritis consist, in the first place, of pain, due to irritation of the ciliary nerves in the inflamed part. Yet this pain is not always referred to the eye itself, but often appears in the form of supra-orbital neuralgia, or affecting the infra-orbital division of the fifth nerve. Dimness of vision is the second subjective symptom of iritis. It may be due to cloudiness of the aqueous humour or cornea, or to exudation of lymph in the pupillary area on the anterior capsule of the lens, or, where the ciliary body is implicated, to opacities in the vitreous humour.

Cases of iritis in which there has been no pain, and no circumcorneal injection, and in which the failure of sight alone it is which brings the patient to the surgeon, are not uncommon. Examination then discovers the presence of extensive posterior synechiæ, which have probably been gradually forming for a long time back. These cases of "quiet iritis" belong to the plastic form, and are, in my experience, usually due to rheumatism (*vide infra*).

A mistake, into which beginners very often fall, is to take a case of iritis to be conjunctivitis or scleritis (see p. 91 and 248);

the "redness of the white of the eye" being that which misleads. The condition of the iris itself will assist chiefly in the diagnosis. Moreover, the pain in iritis is of neuralgic character, but in conjunctivitis it is similar to that caused by a foreign body in the conjunctival sac. In iritis there is no discharge, while in conjunctivitis the eyelids are gummed in the morning by muco-purulent secretion. Of course iritis and conjunctivitis may occur together.

Simple Plastic Iritis is the most common form. In it the circumcorneal injection is generally well marked, sometimes causing elevation of the limbus of the conjunctiva, and even general, although slight, chemosis. In very mild cases, however, as also in chronic cases, the injection may be slight. The loss of lustre, and of distinctness of pattern, of the iris is well marked, and there is considerable change in the colour of the iris.

Posterior synechiæ are very apt to form. In some rare cases of plastic iritis, an enormous quantity of gelatinous exudation is present in the anterior chamber.

In *Secondary Syphilis* one often sees iritis, which, although doubtless due to the syphilitic taint, presents no clinical characteristic different from ordinary simple plastic iritis, yet it is probable that many of these cases are parenchymatous, or condylomatous.

Rheumatic Iritis is of the simple plastic form, but accompanied by circumcorneal injection, which is great in proportion to the other signs of iritis present. The pain in rheumatic iritis is often peculiarly severe. Yet, as I have already stated, "quiet iritis" is most often due to rheumatism.

Gonorrhœal Iritis is a mixture of the plastic and serous forms. It does not attend on, nor immediately follow, a gonorrhœa; but an attack of rheumatic arthritis, usually of the knees, always intervenes. Gonorrhœal iritis is extremely rare.

Serous Iritis. (Keratitis punctata. Aquocapsulitis. Descemetitis.)—Here the exudation is mainly a serous fluid. From

this fluid fibrinous elements, in the form of very fine yellowish spots, are precipitated on the posterior surface of the cornea, chiefly in its lower quadrant, and often in a triangular shape, the base of the triangle corresponding with the lower margin of the cornea, the apex being directed towards the centre of the cornea, and the finer dots near the apex. The triangular shape is a mechanical result of the motions of the eyeball.

In cases where the corneal deposit continues for a length of time, owing to resulting degeneration of the posterior epithelium, permanent secondary changes in the true cornea are produced, and a consequent peculiar triangular opacity at the lower part of the cornea will ever afterwards indicate the nature of the process which has gone before.

In serous iritis the pupil is usually not contracted, and the circumcorneal injection is slight.

The anterior chamber is often deep, owing to the quantity of fluid secreted, and the aqueous humour is cloudy. The increase in the contents of the anterior chamber frequently causes increase in the intraocular tension. Pure serous iritis is perhaps not so common as a mixed form of sero-plastic iritis.

But there is good reason for regarding all these cases of keratitis punctata as due, not to inflammation of the iris, but to cyclitis.

Parenchymatous (including **Purulent**) **Iritis.**—Here the inflammatory product is situated in the tissue of the iris. The consequent swelling of the iris may be present over its whole extent, or may be confined to a circumscribed part of it. In the latter case the swelling is sometimes called a condyloma. The colour of the iris changes remarkably, at the affected part, to a yellowish or reddish-yellow hue, and new vessels are formed in it.

In Syphilis, late in the secondary stage, a form of iritis occurs, which may be always recognised as syphilitic. It is characterised by the formation of circumscribed tumours, or small condylomata of a yellowish colour, the rest of the

iris being, apparently, intact. These tumours vary in size from that of a hemp-seed to that of a small pea, and are situated usually at the pupillary margin, occasionally at the periphery of the iris, and very rarely in the body of the iris. There may be but one tumour present, and there are seldom more than three or four. This form belongs to the parenchymatous class, and is by no means common. Yet many authors hold that in most, if not all, cases of syphilitic iritis, condylomatous tumours are present, but of such small size as to escape detection with our ordinary clinical methods.

Hæmorrhagic Iritis is not a special form of iritis, but is merely a severe inflammation of the iris with hyphæma. It is chiefly seen in iritis, due to operations and injuries, in some cases of sympathetic iritis, and in old people.

Symptoms of Iritis in General.—1. Pain. This is situated not so much in the eye as in the brow over it, in the corresponding side of the nose, and in the malar bone, and may even extend to the whole side of the head. It varies in its intensity; it is usually more severe at night, and is often called neuralgia by the patients. The simple plastic form is the one attended by the most severe pain; the serous form is generally unattended by pain, while the parenchymatous form is often excessively painful, and again completely painless. 2. Lacrimation and photophobia are occasionally present, but never to such a degree as is often observed in certain corneal affections. 3. Dimness of vision. This is usually complained of as soon as the inflammation is pronounced. Cloudiness of the aqueous humour, and keratitis punctata, affect sight in proportion to their degree; and exudation in the pupil may reduce vision to a quantitative amount.

The tension of the eye in iritis is usually normal; but in some cases of violent plastic, serous, and parenchymatous iritis, the tension will be found high.

Prognosis.—The length of duration of an attack of iritis

cannot be foretold at the outset. Cases which are, in other respects, mild, *i.e.*, where the pupil dilates well and rapidly to atropine, where the aqueous humour is clear, and where but little lymph is thrown out, often continue for weeks irritable and painful, with a marked tendency to relapse if treatment be at all relaxed. An attack of iritis may last from two to eight weeks; the plastic form being the most rapid, and the serous form the slowest. Recurrences of the inflammation are common; owing to continuance of the constitutional taint, which gave rise to the iritis in the first instance.

It is possible that an attack of any form of iritis, if carefully treated from the beginning, may leave the eye in as healthy a condition as before; but it is quite as common, in spite of every effort, to find posterior synechiæ, isolated or as a circular synechia, left behind. The presence of a few isolated synechiæ, if the pupil be clear, is in itself harmless to sight; but, if relapses take place, and fresh adhesions be formed, a complete posterior synechia may ultimately be established. When this occurs, the aqueous humour being still secreted behind the iris, the latter becomes bulged forward, like the sail of a ship, until it touches the peripheral part of the cornea; while the centre of the anterior chamber retains its normal depth. This condition is very liable to induce glaucomatous tension (secondary glaucoma), and consequent loss of vision; or, if the eye escape this danger, the traction on the ciliary body produced by the tensely stretched iris may develop chronic inflammation of the ciliary body and chorioid—so-called chronic irido-cyclitis, or irido-chorioiditis; and this may lead to diminished tension and phthisis bulbi, with detachment of the retina, and calcification of the lens. Or, the eye having been first blinded by high tension, may at a later period undergo phthisis bulbi.

Complete posterior synechia may of course result from the

first and only attack of iritis, and not by means of repeated relapses.

In some cases of plastic or sero-plastic iritis, the vitreous humour becomes more or less opaque, and this condition does not always disappear as the iritis gets well. Or, it may not be possible to ascertain its presence until after the inflammatory process in the iris has subsided. Very great and permanent deterioration of vision may result in such instances; and they emphasise the importance of a cautious prognosis at the commencement. There can be no doubt but that in these cases the ciliary body is inflamed along with the iris, although the fact cannot be directly ascertained.

Causes.—Iritis is not common in children, except as complicating a corneal process, or as a result of congenital syphilis or tuberculosis (see New Growths of the Iris, p. 268). Towards puberty slight plastic iritis is sometimes found in girls. Youth and middle age are the times of life in which iritis is most often seen, while in old age it again becomes rare.

More than 50 per cent. of the cases depend on syphilis, and a large proportion of the remainder are due to rheumatism. During desquamation after smallpox, plastic iritis is sometimes observed. In metria and septicæmia purulent iritis occurs, as also with typhoid fever,[1] pneumonia, and recurrent fever. Diabetes sometimes causes iritis of a plastic or purulent form.

Treatment.—Atropine is, above every other, the most important means. It is most commonly used in solution (Atrop. sulph. gr. iv, Aq. dest. ℥j) as drops; but an atom of sulphate of atropine in substance, placed in the conjunctival sac, gives a very active reaction, and I prefer its use in one or other of these forms. It is also used in the form of an ointment (Atrop. sulph. gr. iv, vaselin ℥j), and gelatine discs containing atropine are manufactured. By paralysing the sphincter iridis, atropine provides rest for

[1] Typhoid bacilli have been found in the anterior chamber in this form of iritis (Gillet de Grandmont, *Archiv d'Ophthal.*, xii., 10, p. 623).

the inflamed iris; and, if adhesions have already formed, the dilatation of the pupil may break them down; while, if none are as yet present, the dilatation will greatly aid in preventing their formation. To produce a maximum effect, where it is desired to break down adhesions, six drops should be instilled into the eye, with an interval of from five to ten minutes between each; and, in this way, every drop has time to make its way into the anterior chamber, and finally the accumulated effect of all six is obtained. More than one drop can hardly be retained in the conjunctival sac at a time. The usual run of cases of iritis require a drop in the eye from twice to four times a day.

Some individuals are peculiarly susceptible to Atropine Poisoning, of which the symptoms are:—Dryness of the throat, fever, fulness in the head, headache, delirium, coma. The antidote is morphia, of which $\frac{1}{4}$ grain used hypodermically neutralises $\frac{1}{30}$ grain of atropine in the system. Atropine poisoning occurs by reason of introduction of the solution into the stomach through the lacrimal canaliculi and the nose and fauces; and, in order to prevent this, the finger (of the patient) may be placed in the inner canthus, so as to occlude both canaliculi during, and for some moments after, the introduction of the drop into the eye. After long use of atropine, the skin of the lower eyelid, or of both eyelids, from infiltration with the drug, often becomes eczematous, red, swollen, and painful; and, in other cases, follicular conjunctivitis is induced. If these occur, sol. extr. belladonnæ (gr. viij ad ℥j) should be substituted for atropine, and suitable remedies (see pp. 88 and 136) used for skin or conjunctiva. In old people, tenesmus and retention of urine sometimes result from use of atropine.[1]

[1] *Scopolamine*, a drug which has the same effects on the eye as atropine, has recently been used by Raehlmann and others. It is much stronger than atropine, a solution of 1 in 500 being equal to a 1 per cent. solution of the latter. It also acts more rapidly, and its effect

Atropine, while it is so useful a means in the treatment of inflammations of the iris, ciliary body, and cornea, is of no benefit in many other diseases of the eye, and is positively harmful in some of them. It is necessary to make this statement very explicitly, for some, perhaps I should say many, medical men, who have not devoted attention to the subject of eye-disease, habitually include atropine in every eye-lotion they prescribe. If the disease prescribed for be conjunctivitis, as it very often is, the atropine is calculated rather to increase than to relieve the conjunctival affection; while, if the patient be advanced in life, there is always the danger that a tendency to glaucoma may be present, and, in such a case, the dilatation of the pupil caused by the atropine will be sufficient to bring on an attack of acute glaucoma. In these days, it falls to the lot of most ophthalmic surgeons to be called, at one time or another, to a case of acute glaucoma, brought on by the gratuitous use of atropine in this manner. It is to be feared that the reason for this random prescribing of atropine is to be found in an ignorance of diagnosis, which leads practitioners to throw atropine with a number of other drugs into their eye-lotions, in the hope that some of the ammunition will hit the mark, wherever the latter may be.

Dark protection spectacles should be worn by patients suffering from iritis; and, in severe cases, they should be confined to a dark room, and even to bed.

In Simple Plastic Iritis, iodide of potassium, or perchloride of mercury, may be given internally. If there be much irritation, pericorneal injection, or chemosis, leeching at the

passes off more quickly. When the pupil dilates under its influence, it sometimes assumes an oval or egg-shaped form, due to the action beginning below. (See *Klin. Monatsbl. f. Augenheilk.*, Feb. 1893, and *Belljarminow, Centralblatt f. prakt. Augenheilk.*, Aug. 1893, p. 187.) Some use of the drug at the National Eye and Ear Infirmary has verified the reports given of its action.

external canthus is of use. Intermittent warm fomentations (every two hours) promote healthy vascular reaction. Pain is to be relieved by hypodermic injections of morphia, and by chloral internally.

In rheumatic iritis, and in iritis due to diabetes, salicylate of sodium in large doses (20 to 30 grains every three hours) has often a remarkably favourable effect.

In Serous Iritis a small quantity of atropine will suffice, as there is little tendency to the formation of synechiæ; and, the irritation being slight, leeching is unnecessary. The skin (pilocarpine hypodermically, Turkish baths, and dry rubbing), kidneys, and bowels should be acted on; and to the diuretics prescribed some iodide of potassium may be added. Turpentine in ʒj doses, as recommended by Carmichael, of Dublin, is often a useful remedy here. Mr. John Tweedy prefers Chian turpentine in 5-grain doses every three, four, or six hours.

Blistering on the temples, or behind the ears, is with many surgeons a favourite remedy. It adds to the annoyance of the patient, but I have no belief in it as a remedy in this, or, indeed, in any other eye disease.

Great care is required in watching the tension of the eye in this form of iritis, and, if it be found to increase and to remain high for three or four days, paracentesis of the anterior chamber must be performed to reduce it temporarily, while the iritis is still progressing towards cure. This little operation will also be called for if there be much deposit on the posterior surface of the cornea; as, by means of it, the deposit, to a great extent, may be floated away. (For mode of performing paracentesis see p. 153.)

In Parenchymatous Iritis it is important to obtain rapid absorption of the inflammatory products, which are so abundantly thrown out, and which, in an organ like the eye, would soon cause extensive destruction. Consequently, unless it be the purulent form, the system should be put under

the influence of mercury as quickly as possible, by the use of inunctions of mercurial ointment, or by small doses of calomel internally; and this treatment is indicated, even when the inflammation is not of syphilitic origin. Warm fomentations are useful. An after-treatment with iodide of potassium is to be employed.

In syphilitic iritis of the plastic form von Graefe was fond of the following formula:—

℞ Hydrag. biniodid. gr. vj; Potass. iodidi, ʒiss.; Aq. destill. ℥ss.; Syr. Aurant. ℥iiss. ℿ. A teaspoonful to be taken once a day. The dose to be gradually increased.

In Purulent Iritis, quinine and salicylate of sodium are the most suitable internal remedies.

Injuries of the Iris.

Punctured Wounds of the eye frequently implicate the iris, but rarely do so without also injuring the crystalline lens, or ciliary body, on which then the chief interest centres, as being the organs from which serious reaction is apt to emanate. If a simple incised wound of the iris be observed, it may be regarded as of little importance, for inflammatory reaction need not be feared, and any extravasation of blood into the anterior chamber (hyphæma) becomes absorbed, while, as a whole, the functions of the iris will probably not be affected.

Foreign bodies of small size, such as bits of steel or iron, may perforate the cornea and fasten in the iris, the puncture in the cornea closing rapidly, and possibly no aqueous humour being lost. It is necessary always to remove such a foreign body without delay, although for some time it may cause no reaction. An incision should be made with a Graefe's knife at the margin of the cornea corresponding to the position of the foreign body, and the portion of iris containing the foreign body is then removed with forceps and scissors.

Blows on the Eye are apt to cause one of several remarkable lesions of the iris, namely :—

1. *Iridodialysis* (ἴρις, διάλυσις, *a separating*), *i.e.*, separation of the iris from its attachment to the ciliary body, which is usually accompanied by considerable hyphæma. As much as one half of the circumference of the iris may be involved in the lesion (Fig. 96), or the latter may be so small as to be detected only by aid of light transmitted to the eye by the ophthalmoscope; and then not only the physiological pupil, but also the minute marginal traumatic pupil will be illuminated. The functions of the eye after such injury, even when extensive, may be but little disturbed, or there may be monocular diplopia.

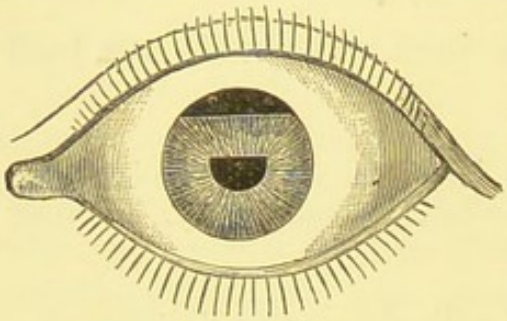


FIG. 96.

Restoration to the normal state in these cases rarely takes place. I have observed one case in which the iridodialysis, a very minute one, was healed, and there is one other such case recorded. The lengthened use of atropine is the most likely way to promote such a result, which can only be hoped for if the iridodialysis be not extensive, and the case be seen early.

2. *Retroflexion of the Iris.*—A portion of the iris in its entire width becomes folded back on the ciliary processes, giving the appearance of a coloboma produced by a wide and peripheral iridectomy. In a true coloboma the ciliary processes would be easily seen, but not so in retroflexion, for the processes, being covered by the retroflected iris, present a smooth surface. A slight dislocation of the lens in the direction away from the iris lesion is often observed. Retroflexion of the iris cannot be cured.

3. *Rupture of the Sphincter Iridis.*—There are not many cases of this lesion recorded; although, according to Hirsch-

berg,[1] in all cases of permanent traumatic mydriasis the margin of the pupil is torn. My observations do not agree with this view of Hirschberg's, nor do I agree with him in thinking, as he seems to do, that rupture of the sphincter would be sufficient to account for traumatic mydriasis. This condition is also incurable.

4. *Traumatic Aniridia.*—The whole iris may be torn from its ciliary insertion and found lying in the anterior chamber, or under the conjunctiva, having in the latter case passed through a rent at the corneo-scleral margin.

5. *Anteversion.*—This must always be accompanied with iridodialysis. The detached portion of iris is then twisted on itself, so that the uveal surface is turned to the front.[2]

6. *Traumatic Mydriasis.*—Permanent dilatation of the pupil after a blow is not very uncommon, and is commonly referred to paralysis of the sphincter, the result of concussion of the delicate nerve endings in the sphincter itself. (See above, under Rupture of the Sphincter Iridis.)

New Growths of the Iris.

Cysts.—These vary from a very small size to that which would fill the anterior chamber. They may have either serous or solid contents. The serous kind was said to result always from a trauma causing an anterior synechia, or otherwise shutting off a fold of the iris, which became distended into a cyst by accumulation of aqueous humour. A case, however, which was not preceded by a trauma has come under my notice. The cysts with solid contents (epidermoid elements) are believed to have their origin in an eyelash or morsel of epidermis, which may have made its way into the anterior chamber by occasion of a perforating corneal wound. All these cysts are sources of serious danger to the eye (irido-chorioiditis,

[1] *Centralbl. f. Augenheilk.*, 1886, p. 368.

[2] L. Werner, in *Ophth. Rev.*, 1887, p. 104.

glaucoma, etc.), and, it is stated, may even be the cause of sympathetic ophthalmitis, and hence their removal is called for. This can be effected without much difficulty if the tumour be small, but if it have attained a large size the attempt may be unsuccessful. A long incision should be made in the corneo-scleral margin, and the cyst, along with the portion of iris to which it is attached, drawn out and cut off.

Granuloma is the name given to a benign neoplasm of the iris, of which the structure resembles granulation tissue. Clinically it is a small pale tumour, or there may be several such tumours, which gradually grow to fill the anterior chamber, rupture the cornea, and finally induce phthisis bulbi. It is held by some that these growths depend on a syphilitic taint, and by others that they are tubercular.

Tubercle (Tubercular Iritis).—This disease occurs generally in children or young adults, who may or may not present evidence of general tuberculosis, such as enlarged or caseating glands, or diseases of joints, etc. It is met with in two forms, viz., disseminated or miliary tubercle, and conglomerate or solitary tubercle.

Miliary Tuberculosis of the iris is a relatively mild form, which presents the clinical appearances of a chronic iritis, sometimes with keratitis punctata; but it is chiefly characterised by the formation of a number of greyish, or cinnamon coloured, semi-translucent nodules on the surface of the iris, and at the angle of the anterior chamber. Occasionally they are not very numerous. The disease may either run its course, and finally cause shrinking of the eye from plastic iridocyclitis; or it may subside, even spontaneously. It is to this form of iritis that Leber[1] has given the name "attenuated tuberculosis" of the iris; but it is not due to any attenuation of the virus, for inoculation in the anterior chamber of a rabbit's eye of an excised portion of such an iris produces severe local and general tuberculosis. This form of tuber-

[1] *Bericht. der Ophth. Gesellschaft zu Heidelberg*, 1891, p. 44.

culosis of the iris is probably the result rather of some constitutional peculiarity.[1]

Solitary tubercle may be accompanied by a few smaller growths, but it generally begins as a single yellowish-white tumour, often without iritis, which gradually increases in size, until it may fill the anterior chamber. It finally involves the cornea, which it perforates, forming a fungating mass, which subsequently breaks down, leaving only a small stump in the socket. Microscopically both varieties present the usual structure of tubercle, but bacilli are very difficult to detect in either of them.

Treatment, in the miliary forms, consists of the usual local and constitutional means. Internal administration of creosote has recently been recommended. If the disease continue to progress, enucleation may be necessary.

Should a solitary tubercle be seen in an early stage, it may be removed by an iridectomy;[2] but, if the disease have advanced too far, or the iridectomy have failed, the eye must be removed. Operative treatment will depend very much on the view which the surgeon takes of the origin of the disease. It has until recently been generally believed that, while tubercle of the chorioid was a disease secondary to tuberculosis elsewhere, tubercle of the iris was a primary affection, and, as such, necessitated immediate enucleation of the eye, in order to prevent it from becoming a source of general infection. The impression, however, seems to be growing, that tubercle of the iris is also a secondary affection, and

[1] Samelsohn, *ibid.*, 1893, p. 75.

[2] Iridectomy, even when it fails to remove all the diseased tissue, may exert a favourable influence, as shown by Leber (*loc. cit.*), in cases of miliary tuberculosis. A case which I have had recently under my care recovered completely after an attempt at iridectomy, although apparently quite hopeless in the beginning. It suggested to me that the breaking up of the nodules, combined with the evacuation of the aqueous humour, may act in the same way as does flushing out of the abdominal cavity in tubercular peritonitis.—L. W.

that, consequently, enucleation is not always called for. Of course in those cases, which are not uncommon, where both eyes are affected, enucleation cannot be entertained.

Primary Sarcoma (or Melano-Sarcoma) is a rare disease of the iris. When the tumour is very small it may be removed by an iridectomy, and in this way an attempt made to preserve the eye; but when it has attained any size, the whole eyeball must be removed.

Ophthalmia Nodosa is a very rare affection, of which about seven cases have been recorded.[1] It is caused by the irritating secretion contained in the hollow hairs of certain caterpillars. In nearly all cases there was a history of caterpillars having accidentally come into forcible contact with the eye. The disease, which is very chronic, is characterised by the presence of small hard nodules in the conjunctiva and iris, and may lead to severe irido-cyclitis. The diagnosis is confirmed by the presence of brownish hairs, or by the examination of an excised nodule, which shows the hair in sections as a yellow oval body with a central cavity.

Congenital Malformations of the Iris.

Heterophthalmos (ἕτερος, *different*, ὀφθαλμός).—This term indicates that the colour of the iris in one eye is different from that in the other.

Corectopia (κόρη, *the pupil*; ἔκτοπος, *out of position*), or malposition of the pupil. The pupil sometimes occupies a position farther from the centre of the iris than normally.

Polycoria (πολύς, *many*; κόρη, *the pupil*).—Where there is more than one pupil. The supernumerary pupil may be separated by only a small bridge from the normal pupil, or it may be situated very near the periphery of the iris. In neither case has it a special sphincter.

[1] Kruger, *Archiv f. Ophthalm.* (German ed.), vol. xxiv., p. 147. This paper contains abstracts of previously observed cases.

Persistent Pupillary Membrane appears in the form of very fine threads stretched across the pupil; and these differ from posterior synechiæ in being attached to the anterior surface of the iris, some distance from the margin of the pupil. They do not interfere with the motions of the pupil, nor with vision.

Coloboma (*κολοβός, maimed*).—This is a cleft in the iris caused by an arrest of development, or inflammation, causing incomplete closure of the chorioidal fissure. It is situated almost always in the lower inner quadrant, at a position corresponding to the chorioidal fissure in the fœtus, and it varies much in size in different cases. It is sometimes continued into the ciliary body and chorioid, and may be present in both eyes; and a notch at a corresponding situation in the crystalline lens is not uncommon. The sphincter lines the edges of the coloboma, thus distinguishing it from the defect which results from iridectomy. When uncomplicated, it causes little or no defect of vision.

Irideremia (*ἶρις, ἐρημία, want of*).—This may be complete or partial. In the latter case it may be the inner circle which is wanting, giving the pupil the appearance of dilatation with atropine. Where the entire iris is absent, the ciliary processes can be seen all round. The condition may be double-sided. The patients suffer chiefly from dazzling by light; for which either protection, or stenopæic, spectacles are to be prescribed.

Operations on the Iris.

Iridectomy.—This is performed for optical purposes, as in zonular cataract, corneal opacities, or closed pupil; for anti-phlogistic purposes, as in recurrent iritis, etc.; to reduce abnormally high intraocular tension, in primary and secondary glaucoma; and for the removal of tumours, or foreign bodies, in the iris.

The instruments required are:—A spring speculum, a

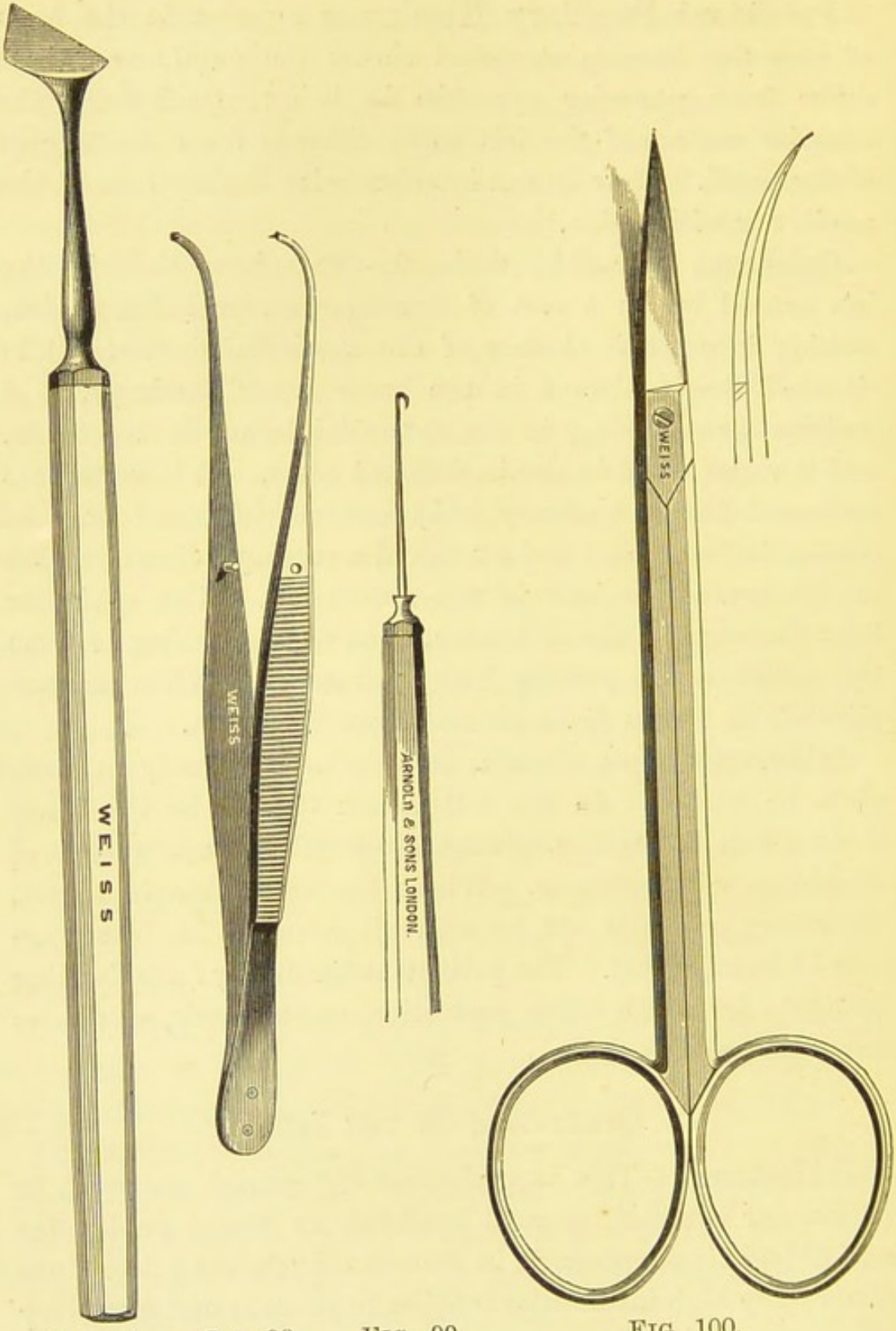


FIG. 97. FIG. 98. FIG. 99. FIG. 100.

fixation forceps with spring catch (Fig. 101), a lance-shaped iridectomy knife (Keratome) (Fig. 97), or a Graefe's cataract

knife, a bent iris forceps (Fig. 98), or a Tyrrell's hook (Fig. 99), an iris scissors curved on the flat (Fig. 100), or a de Wecker's scissors, and a small spatula.

The width of the coloboma depends a good deal on the length of the corneal incision, for it cannot be wider than the incision is long. *Its depth* depends on the proximity of this

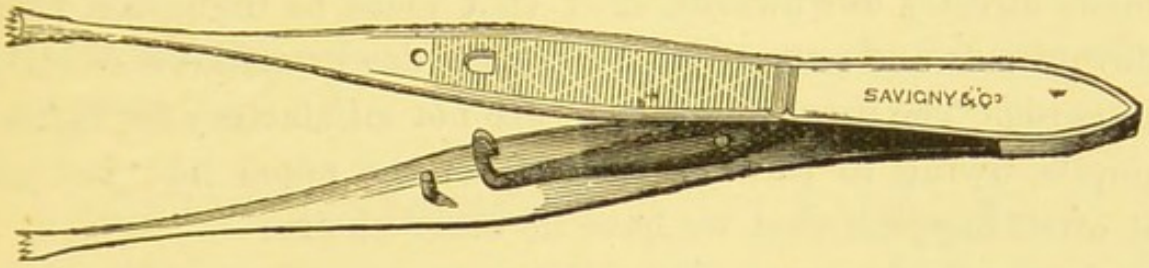


FIG. 101.

incision to the corneo-scleral margin. If a wide and very peripheral coloboma be desired, the incision must be long, and must lie actually in the corneo-scleral margin; the iris forceps being then introduced, a portion of the iris corresponding to the length of the incision may be seized and cut off, and a coloboma, as at Fig. 102, produced. Somewhat inside the corneal margin will give a pupil, as in Fig. 103.

FIG. 102.

FIG. 103.

FIG. 104.

A narrow coloboma (Fig. 104) is obtained by a short corneal incision, which may be more or less peripheral as circumstances require, and by using a Tyrrell's hook, instead of an iris forceps, for catching and drawing out the iris.

In glaucoma, a wide and very peripheral coloboma is required. For antiphlogistic purposes, a wide iridectomy is also necessary. But for optical purposes, a narrow iridectomy is required; because, with a wide coloboma, the diffusion of light may be very troublesome to the patient.

The best position for an iridectomy for glaucoma, or for

antiphlogistic purposes, is in the upper quadrant of the iris; as, there, the subsequent dazzling by light and the disfigurement are least. But the position, by preference, for an optical pupil is below and to the inside, being that most nearly in the direction of the axis of vision. If, however, this position be occupied by a corneal opacity, the coloboma should be made directly downwards, or if that place be ineligible, then downwards and outwards, or directly downwards, or directly inwards. The upward positions are not satisfactory for optical pupils, owing to the overhanging of the upper lid; but yet it often happens that we have no other choice.

In the Performance of an Iridectomy the eye should be fixed with a forceps at a position on the same meridian as that in which the coloboma is to lie, but at the opposite side of the cornea, and close to the latter. The point of the lance-shaped knife is then to be entered almost perpendicularly to the surface of the cornea, and made to penetrate the latter. The handle of the knife is then at once lowered, and the blade passed on into the anterior chamber in a plane parallel to the surface of the iris, until the incision has attained the required length. The handle of the knife is now lowered still more, so as to bring the point of the blade almost in contact with the posterior surface of the cornea, in order to prevent any injury to the lens in the next motion. The knife is then very slowly withdrawn from the anterior chamber. At the same time the aqueous humour flows off, and the crystalline lens and iris come forwards. The fixation forceps is now given over to the assistant, and the bent iris-forceps, held in the left hand, is passed closed into the anterior chamber, its points directed towards the posterior surface of the cornea, so as to avoid entangling them in the iris. When the pupillary margin has been reached, the forceps is opened as widely as the corneal incision will permit, and the corresponding portion of the iris its seized and drawn out to its full extent through the corneal incision. With the scissors, held in the

other hand, the exposed bit of iris is snipped off quite close to the corneal incision. Care should now be taken that the angles of the coloboma do not remain in the wound; and, if they are seen to do so, they may be reposed by stroking the region of the incision with a hard-rubber spoon, or by actually pushing them into their places gently with the spatula.

Iridotomy.—For description and uses of this operation see chap. xiii.

Cyclitis (Inflammation of the Ciliary Body).

Cyclitis is often present to a slight degree as an extension of inflammatory affections of the iris or chorioid, although its presence in many of these cases cannot be clinically determined.

The Symptoms of Cyclitis in general are:—marked circumcorneal injection, ciliary neuralgia, pain on pressure of the ciliary region, very deep anterior chamber, opacity in the anterior part of the vitreous humour, and, sometimes, hypopyon in the anterior chamber. Œdema of the margin of the upper lid frequently occurs, for instance in cyclitis after cataract extraction.

There are three forms of cyclitis:—

1. **Plastic Cyclitis.**—Here the circumcorneal injection is very decided, and there is venous congestion of the iris. The anterior chamber is deep, owing to retraction of the periphery of the iris by inflammatory exudation in the ciliary body, and, for the same reason, the pupil is dilated. The inflammation may extend to the iris, or to the chorioid, in which latter case the vitreous may become very opaque. Violent ciliary pains attend the affection, and the eyeball is very tender on pressure of the ciliary region. The intraocular tension is reduced.

2. **Serous Cyclitis.**—The circumcorneal injection is but slight. The anterior chamber is often at first deeper than

normal, owing to hypersecretion of aqueous humour, or to effusion of fluid inflammatory products from the ciliary body; there is keratitis punctata, and the anterior part of the vitreous humour is filled with a fine dust-like opacity. Serous iritis may come on, and the danger of glaucomatous increase of tension is very great. Unless increase of tension give rise to it, pain does not often attend this form.

3. **Purulent Cyclitis.**—In this form the circumcorneal injection is very well marked. The vitreous humour is filled with membraneous opacities. There is hypopyon in the anterior chamber, which has the characteristic of appearing and disappearing at intervals of a few days. There is severe ciliary neuralgia. Purulent iritis, or chorioiditis, or both, are apt to supervene.

Prognosis.—In an early stage all these forms are capable of undergoing cure, and of leaving the eye in a fairly useful condition. On the other hand, serous cyclitis, as already stated, is liable to produce secondary glaucoma; while the purulent form leads to atrophy of the iris and chorioid, disorganisation of the vitreous humour, detachment of the retina, cataract, and phthisis bulbi; and the plastic form, in addition to serious damage to the affected eye, similar to that produced by purulent cyclitis, has, more than either of the other forms, the tendency to cause sympathetic uveitis of the other eye. The shrunken eyes resulting from this affection are often very liable to attacks of inflammation, and frequently remain painful to the touch, circumstances which indicate that chronic cyclitis is still present, and consequently such stumps are a constant source of danger to the sound eye.

Causes.—Primary idiopathic cyclitis is a rare affection. Traumata are the most common causes of the affection. Both the plastic and the purulent form are liable to occur after cataract operations.

The Treatment for cyclitis is similar to that for iritis. Leeching at the outer canthus is often of great benefit.

Injuries of the Ciliary Body.

Punctured Wounds, and Foreign Bodies perforating the sclerotic at a distance of about 5 mm. around the cornea, are almost certain to implicate the ciliary body. If there be no prolapse of the ciliary body, nor any foreign body in the interior of the eye, the sclerotic wound may heal by aid of a bandage without further ill results. If a prolapse of the ciliary body or iris be present, it is to be abscised; and, if the sclerotic wound be large, it may be thought desirable to unite its margins with sutures. Wounds of the ciliary body are apt to cause cyclitis, especially if the former be caught in the sclerotic wound in healing, or if a foreign body be present in it, or indeed anywhere within the eye; and this traumatic cyclitis is more likely to produce sympathetic ophthalmitis than the idiopathic form. Hence, a region around the cornea about 5 mm. wide is aptly termed by Nettleship the "Dangerous Zone."

New Growths of the Ciliary Body.

Sarcoma of the ciliary body is generally pigmented, and often passes unobserved, until it attains considerable size as a brown mass, which was at first concealed from view by the iris. Occasionally it makes its first appearance at the angle of the anterior chamber. It runs the same course as sarcoma of the chorioid. Removal of the eyeball is indicated, and will often for a time be declined by the patient, as sight may be but slightly affected in the early stages.

Myosarcoma originating in the ciliary muscle has been observed a few times.

Carcinoma.—Primary carcinoma of the ciliary body is an extremely rare disease. Two cases have been recorded recently by Lagrange[1] and Collins.[2] Its occurrence in this situation

[1] *Etudes sur les tumeurs de l'œil, etc.*, Paris, 1893, p. 93.

[2] *Trans. Ophthal. Soc. Un. K.*, vol. xi., p. 55.

is easily explained, if the ciliary body, which secretes the intra-ocular fluid, has a glandular structure; and from the researches of Collins and Nicati[1] there seems to be every reason to believe that it does contain tubular glands.

Secondary carcinoma may occur in the ciliary body, as in the chorioid (p. 287), but is very rare.

Chorioiditis.

There are two great forms of inflammation of the chorioid: the exudative, and the purulent. Of the exudative form, again, there are several kinds, namely, disseminated chorioiditis, central chorioiditis, central senile chorioiditis, guttate chorioiditis, and syphilitic chorio-retinitis.

Disseminated Chorioiditis.—The usual *Ophthalmoscopic Appearances* of this disease consist either in round white spots of different sizes with irregular black margins, or in small spots of pigment; these changes being surrounded by healthy chorioidal tissue. Or there may be few or no white patches, but rather spots of pigment surrounded by a pale margin. The retinal vessels pass over, not under, the patches. The number of these patches or spots varies according to the intensity of the disease. Their position is at first at the periphery of the fundus only, but later on they appear also about the posterior pole of the eye.

These appearances represent a rather late stage of the disease, the early stage not usually coming under observation. It consists in small circumscribed plastic exudations into the tissue of the chorioid, which, seen with the ophthalmoscope, give the appearance of pale pinkish-yellow spots. The exudations may undergo absorption, leaving the chorioid in a fairly healthy state; but, more usually, they give rise to atrophic cicatrices, in which the retina becomes adherent, with proliferation of the pigment-epithelium layer in their

[1] *Archives d'Ophthalmologie*, 1890, p. 490.

neighbourhood, and hence the white patches with black margins above described.

Sometimes, in addition to the above changes, the pigment-epithelium layer all over the fundus becomes atrophied, exposing to view the vascular network of the chorioid, while here and there small islands of pigment are present.

Opacities in the vitreous humour are sometimes found.

Symptoms.—Diminution in the visual acuity, especially if the macula be involved; there may also be subjective sensations of light or colours; positive scotoma (a dark area visible to the patient), and distortion of objects (metamorphopsia), or alteration in their size (megalopsia and micropsia). Night-blindness is not uncommon.

Causes.—Disseminated chorioiditis is due to acquired syphilis in a considerable number of the cases. But in a very large proportion of cases no ascertainable cause exists; and these, there is reason to suspect, are congenital; and, probably, many of them are dependent on an inherited syphilitic taint. In eyes with congenital cataract, patches of chorioiditis are often found.

Prognosis.—Disseminated chorioiditis is always a serious disease, and complete recovery cannot be looked for. The degree of defect of sight it causes, depends much on the extent to which the region of the macula lutea has become involved.

Treatment.—In fresh cases due to acquired syphilis, a prolonged but mild course of mercurial inunctions is the most suitable measure; to be followed by a lengthened course of treatment with iodide of potassium. Where an inherited syphilitic taint is suspected, iodide of iron or iodide of potassium internally may be of use; while in the cases due to other causes, small doses of perchloride of mercury may be given; and in all cases, from whatever cause, dry cupping on the temple, or even the artificial leech, should be employed. Dark protection spectacles should be worn, and absolute rest

of the eyes from all near work insisted upon, so long as the disease is active. Subconjunctival injections of corrosive sublimate are also used in these cases (see p. 160).

Syphilitic Chorioido-Retinitis.—See Syphilitic Retinitis, chap. xv.

Central Senile Guttate Chorioiditis.—Under this name an appearance has been described by Mr. Waren Tay and others, which consists of fine white, pale yellow, or glistening dots, best seen in the upright image, and situated chiefly about the macula lutea, or between this and the optic papilla. These dots are due to colloid degeneration with chalky formations in the vitreous layer of the chorioid,[1] which give rise to secondary retinal changes. The functions of the retina usually suffer in a marked manner, so that a partial central scotoma may be produced; but some cases have been observed, in which vision was but little, or not at all, affected.

This disease attacks both eyes, either simultaneously or with an interval, and is most often seen in persons of advanced life, although also found in middle age, and even in youth. It should always be looked for in cases of incipient cataract; for when the lental opacity is more advanced it cannot be seen, while functional examination does not then detect it.

Treatment is of no avail.

Central Chorioiditis is an exudation at the macula lutea, without any similar disease elsewhere in the fundus. Absolute central scotoma is its prominent symptom, and syphilis its usual cause.

Treatment.—Active mercurialisation; and, where this can be adopted early, the prognosis for recovery of sight is fair.

Central Senile Areolar Atrophy of the Chorioid.—Although this is not an inflammatory process, yet it is most convenient to refer to it here. It is not a very rare disease, and presents the appearance of a white patch, often of consi-

[1] Hirschberg and others, *Centrabl. f. prakt. Augenheilkunde*, 1884, p. 46.

derable extent, at and around the macular region. I think I have observed that, in some cases, a hæmorrhage in the chorioid, and posterior layers of the retina, formed the starting-point of the disease. The retinal functions always suffer much; for an absolute central scotoma is produced, which renders all near work impossible; although locomotion is not much impeded, as the periphery of the field remains intact. The discovery of the presence of this disease, after a cataract has been successfully removed, is sometimes a source of intense disappointment both to patient and surgeon, which cannot be guarded against, unless it has been noted while the cataract was still incipient.

Treatment is of no avail.

Purulent Chorioiditis.—This consists at first in a purulent extravasation between the chorioid and retina, and into the vitreous humour, recognisable by the yellowish reflection obtained from the interior of the eye on illuminating it. The eyeball may become hard, the pupil dilated, and the anterior chamber shallow. Purulent iritis with hypopyon soon comes on, and the cornea may also become infiltrated and slough away. There is usually considerable chemosis. The eyeball is pushed forwards by inflammatory infiltration of the orbital connective tissue. The eyelids are swollen and congested. There is intense pulsating pain in the eye, and radiating pains through the head; and in this stage all the tissues of the eyeball are engaged in the purulent inflammation, and the condition is termed Panophthalmitis.

Purulent chorioiditis does not always reach this latter stage, but may remain confined chiefly to the chorioid, vitreous humour, and iris. The pain in such cases is not severe; and, when the affection occurs in children, it may be mistaken for glioma; indeed, the name pseudo-glioma has, unfortunately, been given to it. It is distinguished from the malignant disease by the muddy vitreous usually present in it, by the posterior synechiæ, and by the retraction of

the iris periphery, with bulging forwards of its pupillary part.

Prognosis.—The ultimate result, in the vast majority of cases, is loss of sight, with phthisis bulbi. The severe cases go on to bursting of the eyeball through the cornea or sclerotic, after which the pain subsides. It would seem from the description of authors who have seen much of epidemic cerebro-spinal meningitis (Niemeyer), that a certain number of cases of irido-chorioiditis occurring in the course of that disease, do recover with retention of good sight.

The shrunken eyeballs produced by panophthalmitis are not generally painful on pressure, nor are they very liable to give rise to sympathetic ophthalmitis; which latter observation is also true of the acute purulent process itself.

Causes.—The most common causes of purulent chorioiditis are wounds of the eyeball, whether accidental or operative; foreign bodies piercing and lodging in the eyeball; and purulent keratitis. It may also come on suddenly, in eyes which are the subjects of incarceration of the iris in a corneal cicatrix.

It is seen as embolic or metastatic chorioiditis, in connection both with epidemic and sporadic cerebro-spinal meningitis; in some cases of metria, similarly as purulent retinitis (chap. xv.); in pyæmia of the ordinary type; and in endocarditis.

In infancy and childhood, besides its occurrence with cerebro-spinal meningitis, it has been known to be caused by, or associated with, inherited syphilis, measles, bronchitis, diarrhœa, whooping-cough, and omphalo-phlebitis; and it is more than probable that, in every instance, some infective blood-disease is the fundamental cause of the process; although it may not always be possible to detect the existence of that blood-disease.

Treatment may be said to be powerless in this disease. The most one can do is to try to diminish the pain by warm

fomentations, poultices containing powdered conium leaves, hypodermic injections of morphium, or, finally, by giving exit to the pus by a free incision in the eyeball, followed by a copious irrigation with weak sublimate lotion, so as to wash out the whole contents of the scleral cavity. Quinine and chlorate of potash are suitable internal remedies.

I agree with those who think that enucleation of the eyeball should not be undertaken during purulent chorioiditis in the acute stage, as it is liable to lead to purulent meningitis and death; but there are surgeons who do not recognise any such danger, and who practise enucleation in this condition.

Posterior Sclero-Chorioiditis, or **Posterior Staphyloma.**—This condition is described in connection with myopia (p. 38), which is its almost constant cause.

Detachment of the Chorioid.—As the result of copious loss of vitreous, during operations or from injury, detachment of the chorioid is not uncommon, but it does not require to be specially diagnosed in these instances, and therefore it is not important to consider it further here.

But idiopathic detachment of the chorioid, although extremely rare,[1] is of importance as forming a well-defined diseased condition in itself.

The Ophthalmoscopic Appearances here are apt to be taken at first glance for a simple detachment of the retina, or for leuco-sarcoma. But, on closer inspection, the chorioidal stroma is observed to lie immediately behind the detached retina, and its vessels, etc., are seen in the upright image by aid of the same lens as are the retinal vessels. The chorioid is not completely detached, but is separated from the sclerotic in several different places, and these detachments are seen in the form of apparently solid hemispherical protuberances rising abruptly from the fundus into the vitreous humour. In other places the chorioid is in contact

[1] The most recent cases of the kind are reported by Story in *Trans. Ophth. Soc.*, 1891, p. 12, and by Mules, *ibid.*, 1893, p. 65.

with the sclerotic, although in some of these positions there may be detachment of the retina alone. The vitreous humour is more or less opaque. Needless to say, vision is greatly lowered or quite destroyed.

It is probable that a chronic chorioido-retinitis has been an antecedent condition in all of these cases. Indeed, there often are signs of old retinitis present, such as perivasculitis, and connective tissue striation; and, in one case, a retinitis was actually observed long before the detachment of the chorioid came on. Adhesions between the chorioid and sclerotic are formed in consequence of this inflammation; and then inflammatory exudation takes place behind the chorioid, and separates it from the sclerotic, where it happens not to be adherent to the latter.

The process ends either in phthisis bulbi, in consequence of vascular changes and disturbances of nutrition; or in cure of a certain kind, in so far as by absorption of some of the exudation, and by alteration of the remainder of it into connective tissue, a return of the chorioid and retina to their normal position is rendered possible; but, even then, restoration of sight, with tunics so disorganised, cannot be looked for.

Treatment hitherto seems to have been of no avail. Probably active mercurialisation might afford the best chance of doing good, should a case come under notice.

Injuries of the Chorioid.

Small Foreign Bodies may pierce the sclerotic, or the cornea and lens, and lodge in the chorioid, and can often there be detected with the ophthalmoscope. They require operative removal by the magnet, if metallic; or if this cannot be carried out, or that the foreign body is non-metallic, the eyeball must be removed, to avert sympathetic ophthalmitis.

Incised Wounds of the sclerotic very frequently involve the chorioid (see p. 251).

Rupture of the Chorioid is often produced by blows on the eye, and is seen with the ophthalmoscope as a whitish-yellow (the colour of the sclerotic) crescent some two or three papilla-diameters in length, and one or so distant from the optic entrance, the concavity of the crescent being directed towards the papilla. Immediately after the accident, extravasated blood sometimes prevents a view of the rupture. Some chorioiditis may result, but, when this passes away, good vision is frequently restored and maintained, provided detachment of the retina does not ultimately supervene from cicatricial contraction at the seat of the rupture.

Treatment.—Careful protection of the eye, and abstinence from use of it, with dry cupping at the temple.

New Growths of the Chorioid.

Sarcoma.—This is by far the most common neoplasm of the chorioid, and is seen at all times of life, but most frequently between the ages of 40 and 60. When highly pigmented, it is termed melano-sarcoma. It may originate in any part of the chorioid.

If seen in a very early stage, it is easily recognised from its projecting over the general surface of the fundus; but, unless it be in the region of the macula lutea, it may not cause any serious disturbance of vision, and hence may not at that period be brought under the notice of the surgeon.

The new growth soon gives rise to detachment of the retina, by reason of serous exudation from the chorioid; and this is accompanied by opacity in the vitreous humour, which renders the diagnosis with the ophthalmoscope difficult or impossible. If the detachment be shallow, and the retina translucent, the tumour may still sometimes be seen through the subretinal fluid by aid of strong illumination; and even direct sunlight may be employed in some such cases. Owing to the great

defect of vision which comes on in this stage, we very commonly see these cases then for the first time. The history of the case may aid us; and the absence of the more usual causes of detachment of the retina should make us suspicious of an intraocular tumour.

Soon the intraocular tension increases; and this makes the diagnosis again more easy in many cases, for the combination of detached retina and increased tension exists only with intraocular tumours. The increased tension may come on very slowly, and without ciliary neuralgia; or more rapidly, and with all the signs and symptoms of acute glaucoma. Still, if the case come now under observation for the first time, the diagnosis may be by no means easy, should the refracting media be opaque (as always in acute glaucoma), and, consequently, the detachment of the retina be concealed from view. Here, again, the history of the case is all we have to depend on; especially the fact of the patient having noticed a defect at one side of his field of vision, previous to the onset of glaucoma.

In the next stage of the growth it perforates the cornea or sclerotic, and, increasing rapidly in size, although still covered with conjunctiva, it pushes the eyeball to one side, the upper lid being stretched tightly over the whole. On raising the lid the tumour is seen as a bluish-grey mass of irregular surface. The conjunctiva is now soon perforated, and the surface of the tumour becomes ulcerated, with a foul-smelling discharge and occasional hæmorrhages. The tumour gradually invades the surrounding skin and the bones of the orbit, and, by extending through the sphenoidal fissure and optic foramen, reaches the base of the brain.

It is, usually, upon the neighbouring tissues of the eyeball becoming involved, that secondary growths begin to form in other organs, the one most prone to be affected being the liver. The lungs, stomach, peritoneum, spleen, and kidneys may all be attacked.

Chorioidal sarcoma is almost always primary, but it has been seen a few times as a metastatic disease.

The entire progress of such a growth varies considerably. It may occupy but a few months, or it may extend over many years.

Carcinoma.—This is extremely rare, and the cases of it on record were all of metastatic origin, the primary disease being in the breast. It is not possible to distinguish chorioidal sarcoma from chorioidal carcinoma by the ophthalmoscope.

Tubercle is sometimes seen in cases of acute miliary tuberculosis as round, slightly prominent, pale yellowish spots, of sizes varying from 0·5 to 2·5 mm. in diameter, situated always in the neighbourhood of the optic papilla and macula lutea, and unaccompanied by pigmentary or other chorioidal changes. There may be but one of these tubercles, or there may be many of them. They occur, as a rule, in a late stage of the general disease, but have occasionally been noted long before its appearance. In obscure cases of the general disease, the ophthalmoscope has sometimes rendered valuable diagnostic aid, by discovering these minute tubercles in the chorioid.

Very rarely, a tubercular tumour grows in the chorioid in cases of general chronic tuberculosis, and attains a large size, the growth destroying the eye similarly as sarcoma, or carcinoma. In young children it may be impossible to diagnose between a tubercular tumour of the chorioid, and a glioma of the retina (chap. xv.). Yet, as in either case enucleation is indicated, the diagnosis is not of much practical importance.

Other, but rare, forms of tumour of the chorioid are:—

Sarcoma Carcinomatosum,[1] and, in a case of my own, *Osteo-Sarcoma*.[2]

[1] *Von Graefe's Archiv*, x., pt. 1, p. 179; Landsberg, *Archiv f. Ophthal.*, xi., pt. 1, p. 58; *Trans. Acad. Med. in Ireland*, i., p. 47.

[2] *Bericht der Heidelberger Ophthal. Gesellsch.*, 1883.

Treatment.—So long as, in cases of sarcoma and carcinoma, the tumour is wholly intraocular, enucleation of the eyeball should be performed, and may be done with fair hopes of saving the patient's life, if the disease be primary. When the orbital tissues have become involved, extirpation of all the contents of the orbit, and even, if necessary, removal of portions of its bony walls, ought to be undertaken, should the general health permit, in order to rid the patient of his loathsome disease; although the probable presence of secondary growths elsewhere renders but small the prospect of saving the patient's life.

Cases of miliary chorioidal tubercle do not call for direct treatment. In cases of tubercular tumour, the question of removal of the eyeball must depend upon the general state of the patient; but, if it seem probable that life will be prolonged until after the ocular growth has become extra-ocular, removal of the eye should be recommended.

Congenital Defects of the Chorioid.

Coloboma.—This is a solution of continuity occurring always in the lower part of the chorioid, and usually associated with a similar defect in the iris. It may commence at the optic papilla, and involve the ciliary body also, and even the crystalline lens may have a corresponding notch; or, it may not extend so far in either direction. The condition is recognised ophthalmoscopically by the white patch, due to exposure of the sclerotic where the chorioid is deficient. Sometimes the retina is absent over the defect in the chorioid, a circumstance which may be ascertained by the arrangement of the retinal vessels; but, even if it be present, its functions at that place are wanting, and a defect in the field of vision exists. Central vision is often normal.

Albinismus, or the want of pigment in the chorioid and iris. This is usually accompanied by defective pigmentation of the hair of the body. The iris has a pink appearance, due

to reflection of light from its blood-vessels, and from those of the chorioid, and, with the ophthalmoscope, the latter vessels can be seen down to their finest branchings. The light, not being partially absorbed by pigment, causes the patient much dazzling, and high degrees of the condition are usually accompanied by nystagmus. In childhood the albinismus and attendant symptoms are more marked than later on, when some degree of pigmentation usually takes place.

Much advantage may be derived in many of these cases by the use of stenopæic spectacles, at least for near work. Any defect of refraction should be carefully corrected.

Sympathetic Ophthalmitis.

By this term we understand a uveitis (irido-cyclitis, irido-chorioiditis) caused by an irido-cyclitis of the other eye, the latter being usually of traumatic origin.

The affection owes its name to the theory, held until a few years ago, that it was due to reflex action of the ciliary nerves. Although this view, which is no longer in accord with modern pathology, has given place to another, yet the original name of the disease is still retained, and we often speak of the injured eye as "the exciting eye," while the secondarily affected eye is called "the sympathising eye."

The cyclitis most likely to cause sympathetic ophthalmitis, is that set up by a punctured wound of the eyeball, especially a wound involving the ciliary body. The cyclitis set up by a foreign body, which pierces the tunics of the eye and lodges in its interior, is also of serious import, even though the ciliary body may not have been injured. Perforating corneal ulcers, and even simple incisions of the cornea, may form the starting-point of sympathetic ophthalmitis. It is an important, and interesting, fact, that eyes which are, or have been, the subject of purulent panophthalmitis, do not give rise to sympathetic ophthalmitis.

There is considerable doubt as to whether sympathetic

ophthalmitis can occur without a perforating lesion of the exciting eye. The only instances, in which it appears really to have thus occurred, are cases of chorioidal sarcoma, in which there was irido-cyclitis. It has been held that a dislocated crystalline lens, or cyclitis caused by a blow on the eye, could serve as excitants of sympathetic ophthalmitis; but, if such cases do occur, they are very rare. I have myself never seen an instance of the kind.

In cases of sympathetic ophthalmitis, the cyclitis of the exciting eye may be but slight, so slight indeed that vision is not seriously affected; or it may be severe. The degree of severity of the attack in the sympathising eye does not depend on that of the inflammation in the exciting eye; for, in many cases, the process in the sympathising eye is a more severe one, and more destructive to sight, than that in the exciting eye.

Sympathetic ophthalmitis is met with in persons of every time of life, but children under the age of puberty are more prone to it than in later years.

Sympathetic Irritation, or Neurosis, is a condition of the second eye sometimes seen, and which must not be confounded with sympathetic ophthalmitis; nor is it to be regarded as a premonitory sign of the latter, for it may pass away without leaving any organic changes behind it. It consists in photophobia, lacrimation, pericorneal injection, and accommodative asthenopia, and is, very probably, a reflex neurosis.

Premonitory Sign of Sympathetic Ophthalmitis.—Shrinking pain (the patient draws back his head in a most characteristic way) on pressure of the ciliary region of the exciting eye is almost always present, where sympathetic ophthalmitis supervenes; although it does not necessarily indicate that the latter is imminent, nor even that its ultimate appearance is certain. But there are no premonitory signs in the sympathising eye prior to the attack of inflammation in it.

Progress of Sympathetic Ophthalmitis.—Slight optic neuritis has been noticed in the sympathising eye in some cases prior to, or simultaneously with, the outbreak of irido-cyclitis, and is, probably, of tolerably constant occurrence. But it is not the sign or symptom which commonly first attracts the attention of the patient, or of the surgeon. The process is usually first observed in the sympathising eye as a serous irido-cyclitis, with increased depth of the anterior chamber and keratitis punctata, and may maintain this character to the end. As a rule, it soon passes over to a plastic form, with development of new vessels in the iris, and shallowness of the anterior chamber. The tissue of the iris and ciliary body becomes infiltrated with lymph cells, and on their posterior surfaces and in the pupil a deposit of lymph cells takes place, the chorioid also becoming similarly infiltrated, and connective tissue is developed in its exudation. The vessels of the uveal tract are destroyed by pressure of the newly-developed connective tissue; the vitreous humour consequently shrinks, causing detachment of the retina, cataract, and phthisis bulbi.

Or, the process may be confined chiefly to the anterior segment of the eyeball, the iris, ciliary body, and lens, and may merely cause disorganisation of those parts with shallow anterior chamber—a condition known as phthisis anterior—while the vitreous humour, retina, and chorioid remain healthy. In such cases, of course, vision is much damaged. Or, again, very occasionally, in some mild cases, the exudation may become absorbed, and leave a tolerably clear pupil and media, with more or less useful sight.

The shortest period at which, after irido-cyclitis has been set up in the injured eye, sympathetic ophthalmitis is liable to appear, seems to be about twelve or fourteen days, and the longest about twenty years. The most usual interval is from six to eight weeks.

Nature of the Disease.—Investigations made in recent

years,[1] especially those of Deutschmann, have placed it beyond doubt, that sympathetic ophthalmitis is an inflammation due to micro-organisms, and propagated by them to the sympathising eye by direct continuity through the optic nerves and chiasma from the exciting eye, as erysipelas extends over the skin. Hence it is sometimes called Migratory Ophthalmitis. A great many objections have of late been raised against the migratory theory of the disease, but they have been satisfactorily disposed of by Deutschmann[2] in a new work on the subject. The staphylococcus pyogenes is the microbe which has been experimented with, and it has also been found in eyes which have given rise to sympathetic ophthalmia; but it is true, that the real micro-organism has not yet been discovered.

Prognosis.—This disease is one of the most serious to which the eye is liable, leading as it does, in the vast majority of cases, to absolute and incurable blindness. It is but rarely that the sympathising eye escapes with some useful vision.

Treatment.—The most important point is the prevention of the extension of the inflammation to the other eye. Sir W. Bowman[3] found it possible, in private practice, by careful nursing for a year or more, to save some eyes with severe

[1] Knies, *Sitzungsber d. Ophth. Gesellsch.*, 1879, p. 52; Leber, *A. v. Graefe's Archiv*, xxvii., pt. 1, p. 325: Brailey, *Trans. Internat. Med. Congress*, 1881, vol. iii.; Snellen, *Trans. Internat. Med. Congress*, 1881, vol. iii.; Macgillivray, *Amsterdam Internat. Med. Congress*, 1879; Berlin, *Volkmann's Samml. Klin. Vorträge*, No. 185, 1880; Deutschmann, *A. v. Graefe's Archiv*, xxx., pt. 3, p. 77, xxxi., pt. 2, p. 277, and *Ueber die Ophthalmia Migratoria*, 1889; Gifford, *Archives of Ophthalmology*, 1886, p. 281. Randolph, in *Arch. of Ophthal.*, vol. xvii., p. 188, does not support the theory of extension of the process through the optic nerves and chiasma, but he does not offer an alternative explanation.

[2] *Beiträge f. Augenheilk.*, March 1893. Schirmer, *von Graefe's Archiv*, xxxviii., pt. 4, p. 93. This is an exhaustive article, and contains a bibliography of nearly 300 papers.

[3] *Ophthal. Rev.*, 1882, p. 288.

wounds, and to prevent the occurrence of sympathetic ophthalmitis.

Abadie recommends[1] that, when the case comes under treatment early, antiseptic measures be taken to prevent infection, the best being subconjunctival injections of corrosive sublimate (see chap. vi.); and that, if these fail, the actual cautery be applied to the wound; and that, if this be not enough, one or two drops of a 1 in 1000, or 1 in 500, solution of sublimate be injected into the wounded eye; and, where the second eye has become affected, one or two drops of the same solution be injected into the vitreous humour of that eye.[2] He has found these injections of use in checking, or ameliorating, sympathetic ophthalmitis.

But the only measures generally admitted to be certain prophylactics, when employed in time, and the only ones applicable to the great mass of those with whom we have to deal, are removal of the injured eye, evisceration, and Mules's operation; and a most difficult question sometimes presents itself, when, in a given case, we have to decide as to the necessity for one of these measures. The following rules guide me in my own practice at present:—

1. Although danger to the second eye practically does not arise, until inflammation has been set up in the exciting eye;[3] yet, I would perform primary enucleation, evisceration, or Mules's operation, on the latter, if it had been so injured as to make recovery of sight almost hopeless, and the onset of irido-cyclitis in it almost certain.

[1] *Annales d'Oculistique*, Mars-Avril, 1890.

[2] Berry (*Trans. Ophthal. Soc. Un. King.*, 1893, p. 220) finds that injections of chlorine water are better tolerated by the retina and vitreous, and that they prevent purulent hyalitis after inoculation of the vitreous with pus. De Schweinitz (*Journ. of American Med. Assoc.*, Oct. 1893) made experiments on rabbits with unsatisfactory results.

[3] A few cases are recorded in which, although the exciter was removed almost immediately after the injury, yet sympathetic ophthalmitis supervened.

2. I would enucleate[1] in the same case, were irido-cyclitis already set up in the injured eye.

3. I would enucleate in a case of irido-cyclitis, where a foreign body, which could not be safely extracted, was present in the eye, even though the vision were fairly good; because, we know that here the danger of sympathetic ophthalmitis amounts almost to a certainty.

4. I would enucleate in a case of acute irido-cyclitis, traumatic or idiopathic, where vision was lost, especially if the eye were tender on pressure; for here the eyeball is useless and disfiguring, and apt to be a source of danger to its fellow.

5. I would enucleate in a case of phthisis bulbi, even of old standing, where there was shrinking pain on pressure, for the same reasons as in No. 4.

6. I would enucleate in a case where the sympathising eye is already affected, provided vision in the exciting eye be lost, and hopes of its recovery but slight, if any; for improvement in the sympathising eye, or a greater amenability of it to treatment, has been frequently observed after this has been done. Brailey, however, holds that enucleation is not in this instance to be recommended, as he believes it tends to aggravate the condition of the sympathising eye—to change a serous into a plastic uveitis.

7. I would enucleate in a case of sympathetic irritation, if the sight of the exciting eye were very defective, and the neurosis very persistent.

1A. I would not remove any injured eye, unless it contained a foreign body which I could not extract, if its sight were fairly good, and as yet no sign of inflammation present. For inflammation may not come on, and the eye may possibly be saved.

2A. I would not enucleate the exciting eye, if sympathetic ophthalmitis had already appeared, should the vision of the

[1] For the sake of brevity the word enucleation only is used in what follows, but evisceration, or Mules's operation, is equally implied.

exciting eye be fairly good. (Contrast this with Rule 6.) For it often occurs, that the process in the sympathising eye is not arrested by the proceeding, and that, where the latter is not undertaken, the exciting eye turns out in the end to be the organ with the better vision.

Cases have been observed in which sympathetic ophthalmitis broke out some days after removal of the exciting eye. In these instances the inflammation, no doubt, had already started on its journey from the exciting eye, the removal of which did not arrest its progress. Inasmuch, then, as the inflammation takes some twelve to fourteen days (*vide supra*) to travel from one eye to the other, one cannot feel certain of having averted sympathetic ophthalmitis before that period at least has elapsed after enucleation of the exciter; and it is well to impose abstinence from use of the eye, or exposure of it to much light for that time, or longer. This fact is not to deter the surgeon from recommending enucleation when indicated; for, in the vast majority of cases, it has the desired effect; and, even in the cases where sympathetic ophthalmitis was not averted, the inflammation in the sympathiser was usually of a mild type, and yielded to treatment.

As substitutes for enucleation of the eyeball in these cases, division of the optic nerve in the orbit (optic neurotomy), resection of a piece of the optic nerve in the orbit (optic neurectomy), and evisceration or exenteration of the eyeball, have all been proposed and practised.

Optic Neurotomy is still employed by some surgeons; but by most it has been abandoned, under the impression that it does not afford good protection against sympathetic ophthalmitis; for the cut ends of the nerves reunite, and at least one case[1] has been observed, in which, several months after the optic neurotomy, sympathetic ophthalmitis appeared.

[1] Leber, *A. v. Graefe's Archiv*, xxvii., pt. 1, p. 339.

Optic Neurectomy was first advocated by Schweigger,[1] and is, in his opinion, a better protective than enucleation. The views of other surgeons have not yet been published, and I have myself too little experience of the method to form an opinion on it, but it would seem to recommend itself as rational.

Evisceration is still on its trial as a prophylactic measure for sympathetic ophthalmitis. A few cases[2] are on record in which the good eye became affected not long after evisceration of the exciting eye, but this has taken place, too, as above stated, after enucleation; and, so far as we can yet form an opinion, the prophylactic value of evisceration is at least as great as that of enucleation. The mode of performing the operation, and Mr. Mules's modification of it, are given at pp. 178 and 179. The indications for these various procedures are the same as for enucleation.

Sympathetic ophthalmitis having broken out, and the question of enucleation, or other prophylactic measure, having been decided in one sense or the other, the means to be directed against the process in the sympathising eye have to be considered. The patient should be confined for a lengthened period to a dark room, and atropine used for the eye; while the general system is maintained by a tonic but non-stimulating treatment. It is doubtful whether other means are of much value. Mercurialisation is employed by some surgeons in these cases, but its value is problematical.

No operation should be undertaken for the formation of an artificial pupil in the sympathising eye until the inflammatory process has completely subsided, the tension of the eye improved, and the vascularity of the iris diminished. This period is, at the least, from twelve to eighteen months after the onset of the disease. If operative interference be resorted to during that period, the result is an aggravation,

[1] *Archives of Ophthalmology*, xiv., p. 223.

[2] By F. R. Cross, *Proceed. Ophthal. Soc.*, July 1887.

or rekindling, of the inflammation, with closure of the artificial pupil which may have been made, in consequence of proliferation of the layer of retro-iritic connective tissue. Not even if the eyeball become of glaucomatous hardness, as sometimes happens, should the surgeon be tempted to operate. This is a golden rule.

Of the operations employed for the establishment of an artificial pupil, in an eye which has suffered from sympathetic ophthalmitis resulting in anterior phthisis, iridectomy most naturally suggests itself, and is the least satisfactory. The reason of this is that, owing to its very disorganised state, the iris tears when drawn on by the forceps; and, hence, the formation of a satisfactory coloboma is almost impossible; and, even if this be obtained, it is extremely liable to close again, from proliferation of the retro-iritic connective tissue set going anew by the irritation of the operation. Yet, sometimes, after repeated iridectomies, a permanently clear pupil may be obtained.

Von Graefe operated by making a peripheral linear incision as for cataract, but passed the knife behind the iris, and in doing so he opened the capsule of the lens. An iridectomy is then made by seizing a wide portion of the iris and corresponding retro-iritic connective tissue with a special forceps, one blade of which is passed behind these structures, whilst the other enters the anterior chamber, and then the iris, etc., having been drawn out, the exposed portion is cut off. The partially, or completely, opaque lens, or a considerable portion of it, becomes evacuated during this proceeding; or, if not, the usual measures are taken to extract it. With this method, also, the pupil frequently closes again, and even more than one supplementary iridectomy, or iridotomy (see chap. xiii.), may be required, but must not be undertaken until all irritation subsides. The iridectomy, as above described, is now with advantage often replaced by a V-shaped one, made with de Wecker's forceps-scissors.

The late Mr. George Critchett's Method for the formation of a pupil in certain of these cases consists in passing a discission needle, by a boring motion, through the lenticular capsule; another needle is passed in close to the first, and then, by separating one point from the other, a rent is made in the centre. This is followed, generally, by the escape into the anterior chamber of a small quantity of cheesy lens matter. The latter is allowed to become gradually absorbed, and, in the course of some weeks, the capsule closes again. The operation has to be repeated several times before a clear pupil is obtained; care being taken that all irritation from the previous operation has subsided, before another is undertaken.

Mode of performing Enucleation of the Eyeball.—There are two chief methods:—

1. Bonnet's Method. The speculum having been inserted, an incision is made in the conjunctiva all round the cornea, and about 6 mm. removed from the latter. The bulbar conjunctiva is separated from the globe freely in all directions with a scissors. With a strabismus hook each orbital muscle is caught up, and its tendon divided close to the sclerotic. The globe can now often be dislocated forwards by pressure of the wire speculum, or, of the margins of the lids, backwards, and is then held in the fingers of the left hand, while the optic nerve is divided with a strong scissors passed into the orbit from the median side. If the globe cannot be dislocated, it may be drawn forwards with a strong toothed forceps, while the nerve is being divided.

2. The Vienna Method. The only instruments used in this operation, in addition to the speculum, are a strong straight scissors and a strong toothed forceps. The tendon of the internal rectus at its insertion, with the overlying conjunctiva, is seized in one grasp with the forceps, and so held until the conclusion of the operation. Immediately behind the forceps the tendon is divided with the scissors; and

now the forceps is holding merely the stump of the tendon adherent to the globe. Through the opening necessarily made at the same time in the conjunctiva one blade of the scissors is passed, and pushed on under the tendon of the inferior rectus muscle, which is then divided along with the over-lying conjunctiva. In the same way the superior rectus is divided. The globe is now drawn well forwards and rotated outwards, the scissors passed into the orbit, the optic nerve felt for, and divided. With one or two strokes of the scissors the external rectus and the two obliques are divided close to the globe, and the operation is completed. This method is very rapid. It is not suited to any globe of which the walls are weak (fresh perforating injury, extreme staphyloma, etc.), for a good deal of pressure is exercised on the eyeball during its performance.

Careful antiseptic precautions are to be employed in connection with enucleation of the globe. Of these, I think, the most important is the use of a full stream of corrosive sublimate solution (1 in 5000) into the cavity of the orbit, as soon as the eyeball is removed; the irrigation being maintained for several minutes. The interior of the orbit is to be then well covered with finely powdered boric acid, a piece of drainage tube placed in the outer canthus, so as to ensure exit of any discharge which may form, and a wood-wool, or other antiseptic pad applied with a bandage. The orbit should be similarly dressed every twenty-four hours.

I have never seen the slightest trouble after enucleation of the eyeball, but some cases of meningitis following upon the operation, and which have proved fatal, are reported. There can be no reasonable doubt but that, in these instances, septic matter made its way along the lymphatics of the optic nerve to the meninges; and that this septic matter was introduced upon the instruments, or escaped, in purulent cases, from the interior of the eyeball. Hence, the very

great importance of the careful antiseptic precautions above indicated.

Occasionally, in ten days or longer after the operation, a granulation forms in the apex of the orbit, and requires to be snipped off. To prevent this it is desirable to unite the conjunctival opening with a suture, after the eyeball has been removed.

An artificial eye can usually be inserted after a fortnight, but should not be constantly worn for a month at least; as, until that period elapses, it is liable to cause irritation and conjunctivitis.

Mode of performing Resection of the Optic Nerve.—An opening is made into the conjunctiva about 3 mm. behind the insertion of the internal rectus muscle; this muscle is laid bare, and two curved blunt strabismus hooks are inserted beneath it. The hooks are drawn in opposite directions, so that one is caught in the angle of insertion of the tendon with a tendency to roll the eye outwards, while the other will draw the muscle forwards out of the orbit. Near the latter hook a catgut thread is passed through muscle and conjunctiva, first from within outwards, and then the opposite way. The muscle is now divided at a distance of at least 5 mm. from its insertion into the sclerotic, and the ends of the catgut thread are tied in a knot. A second thread is passed through the terminal stump of the muscle, and similarly tied in a knot. The wound is now extended both towards the superior and inferior recti muscles; and a small pointed double hook is inserted into the sclerotic far back, in order to draw the globe forwards and outwards. A pair of scissors curved on the flat are inserted alongside the globe, and the optic nerve cut through as near the optic foramen as possible. The posterior aspect of the globe can now be exposed to view by means of the double hook. The stump of the optic nerve remaining on the eyeball is then cut off near its insertion into the sclerotic, the insertion of

the oblique muscles divided, and the whole of the posterior circumference of the sclerotic bared by dissection. The eyeball is replaced, the wound closed by means of the catgut threads previously introduced, and, as a precaution against sanguineous exophthalmos, the eyelids are united by three sutures.

CHAPTER XI.

THE MOTIONS OF THE PUPIL IN HEALTH AND DISEASE.

The Size of the Pupil in Health depends, chiefly, on the intensity of the light to which the eye is exposed, contracting when light falls into the eye, and dilating in the shade. However defective vision may be, if quantitative perception of light remains, the reaction of the pupil, as a rule, takes place.

There is no absolute *Standard for the Physiological Size of the Pupil.* The latter varies in different healthy individuals, being in general smaller in elderly people than in youthful subjects; for, with increasing age, the energy of the sympathetic—the dilating nerve of the iris—is reduced, while there is sclerosis of the walls of the vessels of the iris, and rigidity of its stroma. Persons with blue irides have, in general, smaller pupils than those with dark eyes, for in them more light reaches the retina, and hence the pupil-reflex is stronger. It has also been stated that hypermetropic eyes are apt to have small pupils, owing to the constant effort of accommodation; while in myopia, for the converse reason, the pupils are said to be wide; but the observation is not generally accepted. The diameter of the pupil, when the accommodation is at rest, has been found[1] to vary between 2·44 and 5·82 mm., giving an average diameter of 4·14 mm.

Contraction of the Pupil.—Contraction to light is a reflex motion, the optic nerve being the afferent nerve, and the third nerve the efferent nerve innervating the sphincter pupillæ. It has been shown, by a high authority,[2] that there are special afferent fibres in the optic nerve for the pupil-reflex, distinct from those for vision, and that it is possible to distinguish, with the microscope, these two kinds of nerve fibres from each other.

The anatomical investigations of Meynert[3] have shown, that between the corpora quadrigemina and the centre for the third nerve run communicating fibres (2, Fig. 105), which probably enable this reflex to take

---

[1] Woinow, *Ophthalmometrie*, Vienna, 1871.

[2] B. von Gudden, *Sitzungsber. d. Münch. Ges. f. Morphol. u. Physiol.*, 1886, i., p. 1.

[3] Vom Gehirn der Säugethiere, *Stricker's Handbuch*, Leipzig, 1870.

place. Owing to the semi-decussation of the fibres in the optic chiasma, the stimulus of light, when applied to one eye alone, passes up each tract with equal power to the corpora quadrigemina, and thence, by Meynert's fibres, to the centre for the third nerve (or rather to that portion of it which acts as a special centre for the sphincter pupillæ), and from that point down the myotic, or short ciliary, branches of this nerve to each ciliary ganglion, the ciliary nerves, and each sphincter iridis, causing as active a contraction of the pupil in the non-illuminated eye (consensual contraction) as in its fellow. It is probable, however,[1] that, in addition to this method of bringing about consensual contraction of the pupil, there is a communication, direct or indirect, between the centres for the third nerve of each side capable of effecting it. In no other way can the fact be explained, that consensual contraction of the pupil is maintained in cases of homonymous hemianopsia. If, for instance (Fig. 105), there be a lesion of the right tractus opticus giving rise to left hemianopsia, the centre of the left third nerve alone can be primarily stimulated; but, as both pupils act, a communication between the centres of the third nerves must exist. Merkel[2] believes that there is a direct anastomosis between these centres.

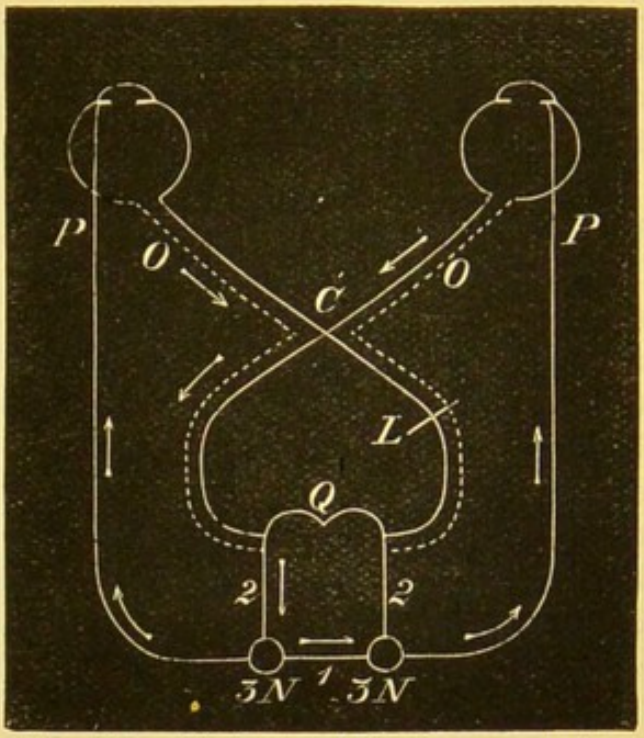


FIG. 105.—3*N*. Centre of third nerve. 1. Connection between nuclei of third nerves. 2. Meynert's fibres. *Q*. Corpora quadrigemina. *C*. Chiasma. *O*. Optic nerve. *P*. Myotic fibres of third nerve. *L*. Seat of Lesion. Arrows show path of impulse in lesion of right tract at *L*.

But it must be stated that there is a good deal of divergence of opinion as to the path by which the pupil-reflex is brought about. Bechterew is of opinion that the centripetal pupillary fibres pass uncrossed from the chiasma directly to the grey matter surrounding the third ventricle, and thence backward to the pupillary nucleus of the oculo-motor nerve of their own sides respectively. Gudden made experiments which seemed to him to prove that the corpora quadrigemina had nothing to do with this path, and ascribed to the external geniculate body the part usually assigned to the corpora quadrigemina. Mendel's experiments[3]

[1] Lesser, *Die Pupillarbewegung in Physiologischer und Pathologischer Beziehung*, Wiesbaden, 1881, p. 14.

[2] *Graefe-Sæmisch Handbuch*, vol. i.

[3] *Neurolog. Centralbl.*, 1890, p. 184.

would lead to the view that it is the ganglion habenulæ which is the centre for the pupillary reflex in animals, and in this he is largely supported by Darkschewitz, who holds that the pupillary fibres from the optic tract pass both into the pineal gland and the ganglion habenulæ. According to Mendel the reflex path would be:—Optic nerve, optic tract, to the ganglion habenulæ of the same side, thence by the posterior commissure to the nucleus of the third nerve, and thence to the ciliary nerves.

The reflex mobility of the pupil to light is tested, most commonly, for the purpose of deciding the existence, or otherwise, of posterior synechiæ. The next most common object of the test, and the one with which we are here concerned, is to determine the sensitiveness to light of the retina, or of the visual centre. It affords, generally, a sufficient test of the presence or absence of quantitative perception of light; but it must be remembered that the latter function may be wanting in certain diseased states, and yet the pupil-reflex take place; or, the pupil-reflex may be wanting, and still perception of light be present. The test is best performed in diffuse daylight, with the patient's face directed towards the window, a distant object being looked at, and the eye which is not under examination being carefully excluded from the light. The surgeon then, having observed the size of the pupil to be examined, excludes the eye from light with his hand for some moments. On removing the excluding hand, a normally reacting pupil will be found to have become dilated; and this dilatation, after an interval of about half a second, will be observed to give way to an extreme contraction, which is maintained only for a moment, and is then succeeded by a moderate dilatation, and the pupil then again contracts somewhat, and so on, until, after some further minute oscillations, it comes to a standstill. The explanation for this phenomenon—which is termed Hippus—is, that each contraction of the pupil, by diminishing the supply of light to the retina, contains in itself the cause of the succeeding dilatation; and, for the converse reason, each dilatation sets agoing the succeeding contraction, until at last equilibrium is attained. A comparison between the maximum of dilatation and maximum of contraction, along with the promptness and rapidity with which the contraction takes place, enables the observer to form an estimate of the activity of the pupil-reflex. In performing this test it is important that the patient's gaze should be fixed all through on a distant object—hence, unless where a mere trace of perception of light remains, the test used with the artificial light is not so reliable as that with daylight—so that the pupil-contraction which is associated with convergence or accommodation (*vide infra*), may not vitiate the experiment. The danger of a vitiation of the experiment by the reflex dilatation from the skin (*vide infra*), caused by the excluding hand, is insignificant in practice. The consensual reflex of the pupil, as well as the direct, should always be

tested; one eye being alternately excluded and exposed, the motions of the pupil of the other eye are observed and compared with those of its fellow. In examining the pupils we have also to decide whether they be of equal size; and, in order to avoid error through posterior synechiæ, the comparison should be made, with both eyes open, successively in two very different brightnesses of light. Under normal conditions equality in size of the pupils will exist, not only with both eyes open, but also if one eye be shaded; for the normal consensual pupil-reflex is equal to the direct reflex. If the pupils be found of different sizes, the least movable one is usually the pathological pupil, but this question is often difficult to decide. Finally, it should be noted, whether the direct pupil-reflex be similar in all respects in each eye.

In addition to the stimulus of light, the pupil-contracting centre is excited by, or simultaneously with, the effort of accommodation for near vision. The object of this contraction is to cut off rays falling on the peripheral portions of the lens, which latter are not curved in the change for accommodation to the same degree as is the centre of the lens. This contraction, however, is much more intimately connected with convergence of the visual lines than with the effort of accommodation. It has been shown,[1] that the contraction increases with the effort of accommodation, but not proportionately to the distance of the fixation point from the eye; and,[2] that the pupils do not contract, if accommodation be effected without convergence; but that, in convergence without accommodation, contraction is observed. It has also been found, that the contraction was proportional to the degree of convergence, and that in myopes of high degree contraction of the pupil takes place at the other side of the far point, where, of course, the accommodation does not come into play. Aubert[3] thinks there is probably a common centre for the three actions, convergence, accommodation, and pupil-contraction, a view supported by Priestley Smith[4]; and Henson and Völckers[5] have found that in dogs, in the posterior part of the floor of the third ventricle, the centres for the branches to the ciliary muscle, the sphincter pupillæ, and the rectus internus, occur in close succession, and they think that this region may be regarded as the centre assumed by Aubert. The existence of such a centre has been placed beyond controversy by Eales's case[6] of paralysis of convergence and accommodation, and of the associated pupillary contraction. These three motions, then, are not dependent on each other, but are co-effects of one and the same cause, *i.e.*, a stimulus applied to the centre for convergence, accommodation, and pupil-contraction.

---

[1] Adamük and Woinow, *Archiv für Ophthalmologie*, xvii., pt. 1.
[2] E. H. Weber, *De Motu iridis*, Lipsiae, 1851.
[3] *Graefe und Sæmisch Handbuch*, ii., p. 660.
[4] *Ophthal. Hosp. Rep.*, vol. ix., p. 32.
[5] *Arch. f. Ophthal.*, xxiv., pt. 1, p. 23.
[6] *Trans. Ophthal. Soc.*, Jan. 10, 1884.

In examining the mobility of the pupils in a given case, the contraction on convergence should not be omitted. If the patient be blind of both eyes, the observation can be made by calling on him to direct his eyes towards his own hand at about 12 inches' distance. If both accommodative contraction and light reflex are wanting, a lesion in the course of the centrifugal pupil fibres is indicated; while, if the light reaction alone is wanting, the lesion is in the course of the centripetal fibres.

Dilatation of the Pupil.—The most reliable investigations [1] have distinctly proved that there is no such muscle as the dilator pupillæ. The dilatation of the pupil is, in all probability, largely the result of an inhibitory action of the sympathetic, a view maintained also by Gaskell [2] and Jessop.[3] The posterior limiting membrane of the iris is its only structure which is not thrown into folds when the pupil dilates (Fuchs); and, therefore, there can be little doubt but that it takes an active part in dilating the pupil, probably by reason of its elasticity. Yet, inasmuch as when the pupil is dilated from paralysis of the third nerve, a further dilatation can be produced by atropine, it is probable that some other, as yet unascertained, dilating power resides in the iris. The mydriatic, or long ciliary, nerves, originating (Henson and Völckers) in the front part of the floor of the aqueduct of Sylvius, pass to a region in the lower cervical and upper dorsal portion of the cord, called by Budge [4] the ciliospinal centre, and from thence pass out with the two first dorsal nerves, and, by way of the rami communicates, to the sympathetic in the neck, and thence to the cavernous plexus, Gasserian ganglion, ophthalmic division of the fifth nerve, nasal branch of this division, ganglionic branch of this nerve, ciliary ganglion, there joined by more branches from the cavernous plexus, and from thence by the short ciliary nerves reach the eye.

The dilating nerve fibres are probably of twofold nature, muscular and vasomotor. The experiments of Grünhagen,[5] Salkowski,[6] Donders, and Hamer,[7] Stellwag,[8] and J. Arlt, jun.,[9] indicate this; and, that the centre for each kind of fibre is different, though both are situated in the medulla oblongata, and their fibres probably run the same course to the eye. The centre for the muscular fibres is called the oculo-pupillary centre. That the vasomotor fibres have a decided, and

[1] Schwalbe, *Handbuch der Sinnesorgane*; Eversbusch., *Bericht d. Ophthal. Gesellsch.*, 1884; Fuchs, *Graefe's Archiv*, xxxi., pt. 3, p. 39; Jessop, *Proceed. Roy. Soc.*, 1886, p. 478.

[2] *Journ. of Phys.*, vii., 1, p. 38.

[3] *Proceed. Roy. Soc.*, 1886, p. 484.

[4] *Ueber die Bewegungen der Iris*, 1855.

[5] *Zeitschrift f. rat. Med.*, xxviii., and *Archiv. f. d. Gesam. Physiol.*, Bd. liii.

[6] *Ibid.*, xxix., p. 167.

[7] *Nederl. Tijdschr. v. Geneesk*, 1864.

[8] Ueber Atropin., *All. Wiener Med. Zeitung*, 1872, p. 146.

[9] *Archiv für Ophthal.*, XV., i.

independent, influence in dilating the pupil, has been shown by Rouget,[1] Schoeler,[2] and others. It is not certain what the mechanism of this influence may be, but it probably consists in a diminution in volume of the iris, from anæmia caused by contraction of the muscular coat of the vessels.

Langley and Anderson[3] find that stimulation of the cervical sympathetic causes dilatation of the pupil, before the vessels of the iris contract, and that stimulation of a portion of the iris can produce a displacement of the pupil towards the side stimulated, without relaxation of the sphincter. They assume, therefore, that there must be some radial contractile substance in the iris, but in what form they do not say.

While light is the only stimulus capable of bringing about a reflex contraction of the pupil, the pupil-dilating centre reacts to every sensitive stimulus, *e.g.*, the prick of a pin or a pinch on the neck, galvanism applied to the leg,[4] the tickling of a sensitive place in the region of the fifth nerve on the face,[5] etc., and Westphal[6] observed dilatation on shouting loudly into the ear of a person under chloroform. Schiff and Foa[7] found that, in curarised dogs and cats, a dilatation took place on the application of every stimulus, not necessarily painful, applied to the nerves of common sensation in any part of the body. The centre for this reflex is probably in the medulla oblongata,[8] but, inasmuch as it takes place if the cervical sympathetic be divided,[9] it is evident that all the dilating fibres do not run to the eye by way of the cervical sympathetic. Schiff,[10] indeed, thinks it probable, that the Gasserian ganglion receives pupil-dilating fibres from the sympathetic traversing the cavum tympani.

Some psychical emotions produce dilatation of the pupil. The pupils of a cat in anger dilate, and those of a frightened child. In sleep, or when under the complete influence of an anæsthetic, the pupils are contracted, for then all psychical and sensitive stimuli are reduced to a minimum. Facts authorise the conclusion, that the medium dilatation of the pupil in the healthy state depends chiefly on the intensity of these stimuli, habitually transmitted through the sympathetic. If, in any individual, they be slight, his pupil is contracted; if intense, it is dilated. Arndt[11] asserts that in delicate, nervous, excitable people the pupils are often much, and habitually, dilated.

---

[1] *Comptes rendus et Mém. de la Soc. de Biologie*, 1856.
[2] *Experimentelle Beiträge zur Irisbewegung:* Inaug. Diis. Dorpat, 1869.
[3] *Journal of Physiology*, 1892, vol. xiii., No. 6.
[4] Arndt, *Griesenger's Archiv f. Psych.*, ii.
[5] Hecker, *Tageblatt der 45 Versam. deutscher Naturforscher in Leipzig*, 1872.
[6] *Virchow's Archiv*, xxvii., p. 409.
[7] La pupilla come estesiometro. *L'Imparziale*, 1874.
[8] Salkowski, *loc. cit.*
[9] Vulpian, *Archiv de physiol., etc., de Brown-Sequard.* Janvier, 1874.
Untersuchungen zur Naturlehre, x., 1867, p. 423
[11] *Archiv f. Psychiatrie*, ii., p. 589.

In addition to those already mentioned, there are causes for the dilatation of the pupil which can hardly be referred to simple reflex action, but which seem to be, like the contraction of the pupil on convergence of the visual lines, associated with those of other centres in the medulla oblongata, especially with those for respiration and uterine action. With every *deep* inspiration or expiration a considerable pupillary dilatation takes place, not identical with that slight dilatation occurring on each ordinary inspiration, and depending on variation of blood pressure, but due[1] to simultaneous stimulation of the respiratory and pupil-dilating centres, by retention of carbonic acid gas in the blood. Raehlmann and Witowski[2] have observed marked dilatation at the beginning of each labour pain, to be explained as an associated action of the neighbouring centres for uterine movements and pupil-dilatation.

Besides the normal pupillary motions described in the foregoing, and visible for the most part to the naked eye of the observer, there is a phenomenon of pupillary motion which is discoverable only by aid of a corneal microscope or loup, consisting in perpetual, but very minute and irregular, fluctuations in size of the pupil. This hippus has been aptly termed by Laqueur[3] the Unrest of the Pupil, and is due to the ever-varying sensitive and psychical reflexes, which are thus constantly manifesting their influences on the pupil.

The Fifth Nerve has been held by some to have an influence over the motions of the iris similar to that of the sympathetic. This is, according to Leeser, a mistaken view[4]; the effect on the pupil following section of the fifth within the cranium being due to paralysis of the sympathetic fibres contained in it, and not to the lesion of the proper fibres of the fifth nerve. But Spallita and Consiglio[5] found, after removal of the superior cervical ganglion of the sympathetic, and when sufficient time for degeneration had been allowed to elapse, that stimulation of the fifth nerve caused myosis. Others[6] again have ascribed to the fifth nerve a direct influence over the contraction of the pupil; but this is to be regarded as a reflex action merely, Merkel indeed having demonstrated[7] the existence of a direct fibrillar connection between the centres of the fifth and third nerves.

Action of the Mydriatics on the Pupil. *Atropine.*—Inasmuch as a maximum mydriasis can only result from paralysis of the pupillary branches of the third nerve, combined with excitation of the pupillary branches of the sympathetic; and as atropine effects such a mydriasis,

[1] Schiff, *loc. cit.*
[2] *Archiv f. Physiologie*, 1878, p. 110.
[3] *Klin. Monatsbl. f. Augenheilk.*, Dec. 1887.
[4] Leeser, *loc. cit.*, pp. 46-48.
[5] *Archivio de Ottalmologia*, 1893, vol. i.
[6] Grünhagen, *Berl. Klin. Wochenschr.*, 1866, No. 24; Rogow, *Zeitschr. f. rat. Med.*, vol. xxix., p. 289.
[7] *Graefe und Sæmisch's Handbuch*, i., p. 140.

it is evident that it acts in the way indicated on these nerves.[1] A. von Graefe proved[2] that the aqueous humour of an eye into which atropine has been instilled, acts as a mydriatic when applied to another eye. *Duboisine*, *Hyoscyamine*, *Scopolamine*, and *Daturine* act similarly to atropine. *Cocaine* mydriasis seems[3] to be induced merely by a local irritation of the endings of the sympathetic in the iris, both of the vaso-constrictor fibres and of the pupil-inhibitory fibres. Strychnine and curare are not, strictly speaking, mydriatics, as they only indirectly affect the pupil; the mydriasis observed in poisoning by these drugs being, according to Schiff[4] and others, the result of the retention in the blood of carbonic acid gas.

Action of the Myotics on the Pupil. *Eserine* (or *Physostigmine*).—This drug is in all respects a complete antagonist of atropine,[5] paralysing the peripheral endings of the sympathetic in the iris, and stimulating the endings of the branch of the third nerve in the sphincter pupillæ. *Pilocarpine* and *Muscarine* act similarly, but not with the same energy. *Nicotine*, applied to the eye, is found to act like eserine.[6] Morphium has an antagonistic effect to atropine, both as regards the pupil and the general nervous system, and is employed in cases of poisoning by atropine (*vide* p. 262).

Chloroform in the first or excitation stage of anæsthesia, according to the investigations of Westphal,[7] Budin,[8] and Hirschberg,[9] stimulates the pupil-dilating centre, and in the second stage gradually reduces the excitability of this centre, until, finally, it is completely paralysed, so that no form of stimulation causes any dilatation. Following on this is a still further contraction to a pin-hole pupil, due to stimulation of the pupil-contracting centre. Should the inhalation of the anæsthetic be continued longer, a dilatation of the pupil, often sudden, takes place, and this indicates paralysis of the pupil-contracting centre, and the most serious consequences for the life of the patient.

The Size of the Pupil in Disease.—*Myosis* may be caused by a diseased process irritating the pupil-contracting centre or nerve-fibres (the Irritation Myosis of Leeser), or by one causing paralysis of the pupil-dilating centre or nerve-fibres (the Paralytic Myosis of Leeser), or by a combination of both. Either cause alone would produce a medium myosis; a combination of the two would give a maximum myosis.

---

[1] Hermann, *Lehrb. der exp. Toxicologie*, 1874.
[2] *Archiv f. Ophthal.*, i., pt. 1, p. 462, foot-note.
[3] Jessop, *Proceed. Roy. Soc.*, p. 441, 1885.
[4] *Pflüger's Archiv*, 1871, p. 229.
[5] Harnack, *Arch. f. exp. Pathol.*, ii., p. 307; A. Weber, *Archiv f. Ophthal.*, xxii., pt. 2, p. 231.
[6] Rogow, *Zeitschrift f. rat. Med.*, xxix., p. 1; Schur, *Zeitschrift f. rat. Med.*, xxxi., p. 402.
[7] *Virchow's Archiv*, xxvii., p. 409.
[8] *Gazette des Hôpitaux*, 1874, p. 910.
[9] *Berl. Klin. Wochenschr.*, 1876, p. 652.

Irritation Myosis, according to Leeser, is not usually increased by the stimulus of light, nor on convergence of the visual axes, nor does it diminish in the shade. Mydriatics dilate such a pupil widely; myotics contract it ad maximum. In paralytic myosis the pupil reacts well to light and on convergence, but does not dilate on application of sensitive or psychical stimuli, or with co-ordinated motions. Mydriatics dilate such a pupil only partially, while myotics contract it ad maximum. In maximum myosis every reaction is wanting, strong mydriatics alone producing a medium dilatation.

Irritation myosis is found in:—*a.* The early stages, at least, of all inflammatory affections of the brain and its meninges, in simple, tubercular, and cerebro-spinal meningitis. When, in these diseases, the medium myosis gives place to mydriasis, the change is a serious prognostic sign,[1] indicating the stage of depression with paralysis of the third nerve. *b.* In cerebral apoplexy the pupil is at first contracted, according to Berthold,[2] who points out that this contraction is a diagnostic sign between apoplexy and embolism, in which latter the pupil is unaltered. *c.* In the early stages of intra-cranial tumours situated at the origin of the third nerve or in its course. *d.* At the beginning of an hysterical or of an epileptic attack.[3] *e.* In tobacco amblyopia,[4] probably from stimulation of the pupil-contracting centre by the nicotine. *f.* In persons following certain trades, as the result of long-maintained effort of accommodation[5] (watchmakers, jewellers, etc.), the pupil-contracting centre being subject to an almost constant stimulus. *g.* As a reflex action in ciliary neurosis; consequently, in many diseased conditions of those parts of the eye supplied by the fifth nerve.

Paralytic myosis occurs:—In spinal lesions above the dorsal vertebræ, *e.g.*, injuries, and inflammations, especially of the chronic form. The contracted pupil occurring in grey degeneration of the posterior columns of the spinal cord has been long known as Spinal Myosis. In the simple form of this myosis the pupil has but a medium contraction, and reacts both to light and on convergence. This condition is found in the early stages alone, when the disease has attacked merely the cilio-spinal centre, or higher up, as far as the medulla oblongata; later on, when Meynert's fibres become engaged, we have the Argyll-Robertson pupil. The very minute pupil, often seen in tabes dorsalis, is probably due to secondary contraction of the sphincter pupillæ.[6]

Argyll-Robertson was the first to point out,[7] that in tabes dorsalis the pupil, although contracted, and responding to light by further con-

[1] Leeser, *loc. cit.*, p. 82.
[2] *Berl. Klin. Wochenschr.*, 1869, No. 39.
[3] Wecker, *Graefe und Sæmisch's Handbuch*, iv.
[4] Hirschler, *Arch. f. Ophthal.*, xvii. pt. 1.
[5] Seiffert, *Allgem. Zeitschrift für Psychiatrie*, x., 1853, p. 544.
[6] Hempel, *Archiv f. Ophthal.*, xxii., pt. 1.
[7] *Edin. Med. Journal*, xiv., 1869, p. 669, and xv., 1870, p. 487.

traction but slightly, or not at all, does become more contracted on convergence of the visual axes (or accommodation). He explained this phenomenon as being due to paralysis of the cilio-spinal nerves, which he therefore regarded as the nerves supplying the sphincter iridis. But Raehlmann points out[1] that the myosis and the motor phenomenon are not directly connected; for it sometimes happens that pupils which do not react to light, and do contract on convergence, are not habitually contracted, and may even be somewhat dilated. The two symptoms are, no doubt, often present together in tabes. The myosis is a sign, and an important one, of disease of the posterior columns, while the defective reaction to light with retained contraction on convergence indicates disease at some distance from the spinal cord, namely, in Meynert's fibres; and this is probably the correct explanation of the Argyll-Robertson symptom. Disease in Meynert's fibres, however (as also disease of the optic nerve), may be in direct connection with disease of the cord, Stilling having found[2] fibres passing directly from the optic tract into the crus cerebri.

Some authorities regard myosis as one of the earliest symptoms of tabes, while others do not. Raehlmann also thinks that, perception of light being present, if the pupils do not react to light, while they do contract on convergence, the symptom is usually one of serious central disease.

Paralytic myosis is also found in general paralysis of the insane. In acute mania the pupil is usually much dilated, and when this mydriasis is changed for myosis, approaching general paralysis may be prognosticated.[3] Myosis, following on irritation mydriasis, is also found in myelitis of the cervical portion of the cord. In bulbar paralysis, if paralytic myosis occurs, the disease is probably complicated with progressive muscular atrophy, or with sclerosis of the brain and spinal cord.[4]

Hirschler states[5] that he has frequently noticed a contracted pupil in alcoholic amblyopia, due, probably, to an affection of the medulla oblongata, possibly fatty degeneration. Myosis may also be due to paralysis of the cervical sympathetic, resulting from injury, from pressure of an aneurism of the carotid, innominate, or aorta, or from pressure of enlarged lymphatic glands. In apoplexy of the pons Varolii, myosis is present, but it is not yet certain whether it is an irritation myosis,[6] or a paralytic myosis.[7]

Mydriasis may be caused by a diseased process giving rise to irritation

---

[1] *Loc. cit.*, p. 7.
[2] *Beilageheft zu Zehender's Monatsblätter*, xvii., pp. 203-207.
[3] Seiffert, *loc. cit.*
[4] Leeser, *loc. cit.*, p. 94.
[5] *Archiv f. Ophthal.*, xvii., pt. 1, p. 229.
[6] Larcher, *Pathol. de la protub. Annulaire, deux. tirage*, p. 54.
[7] Jüdell, *Berl. Klin. Wochensch.*, 1872, No. 24.

of the pupil-dilating centre or fibres, or by paralysis of the pupil-contracting centre or fibres.

The former is termed Irritation (or Spasmodic) Mydriasis, and, according to Leeser, is characterised by a moderately dilated pupil, contracting somewhat to light and on convergence, but not dilating on sensitive or psychical stimuli; easily dilated ad maximum by mydriatics, but with difficulty contracted ad maximum by myotics. The latter is called Paralytic Mydriasis, and in it there is a moderately dilated pupil, reacting to sensitive and psychical stimuli. The reaction to light and on convergence varies according to the seat of the lesion. If the lesion lie between the iris and the pupil-contracting centre, the direct and consensual reaction to light is wanting, as also the associated motion on convergence of the visual lines. But, if the lesion lie between the retina and the pupil-contracting centre, the direct contraction to light is wanting, while the consensual contraction, and that on convergence, are retained.[1] In either case the pupil can be dilated ad maximum by mydriatics, but not contracted more than to medium size by myotics.

Irritation of the pupil-dilating centre and paralysis of the pupil-contracting centre existing simultaneously, give rise to maximum mydriasis. In it there is absolute immobility to stimuli of all kinds, except to strong myotics, which may bring the pupil back to the normal size.

Irritation Mydriasis occurs:—*a*. In hyperæmia of the cervical portion of the spinal cord, and in spinal meningitis. *b*. In the early stages of new growths in the cervical portion of the cord. *c*. In cases of intracranial tumour and other diseases causing high intracranial pressure, according to Raehlmann, although Leeser points out that these may also give rise to paralytic mydriasis. *d*. In the spinal irritation of chlorotic or anæmic people, after severe illness, etc. *e*. As a premonitory sign of tabes dorsalis. *f*. In cases of intestinal worms, owing to the stimulation of the sensitive nerves of the bowel, and sometimes in other forms of intestinal irritation. *g*. In psychical excitement, *e.g.*, acute mania, melancholia, progressive paralysis of the insane (often then unilateral, with myosis in the other eye).

Unilateral mydriasis occurring at short intervals, now in one eye and now in the other, is, according to von Graefe,[2] a premonitory sign of mental derangement. Von Graefe observed madness, in the form of manie des grandeurs, to come on some months after the occurrence of this symptom.

[1] Heddæus (Knapp, *Archiv f. Ophthal.*, xxvii., 1893, p. 38) and Turner (*Royal London Ophthal. Hosp. Rep.*, Dec. 1892) assume that the sphincter derives its nerve fibres from two centres, *viz.*, from the special sphincter centre, and also from the centre for convergence (or accommodation).

[2] *Archiv f. Ophthal.*, iii., pt. 3, p. 350.

Paralytic Mydriasis (Iridoplegia) may be due, either to a paralysis of the pupil-contracting centre, or as the result of the stimulus not being conducted from the retina to that centre. It may be found under the former circumstances:—*a*. Sometimes in progressive paralysis, where at first there was myosis. *b*. In various diseased processes at the base of the brain affecting the centre of the third nerve. *c*. In a late stage of thrombosis of the cavernous sinus.[1] *d*. In orbital processes which cause pressure on the ciliary nerves. *e*. In glaucoma. *f*. In cases of intraocular tumours which have attained a certain size.

In paralytic mydriasis, due to non-transmission of the stimulus of light to a healthy pupil-contracting centre and nerves, contraction of the pupil will take place only on convergence of the visual lines. The same condition of pupil will be found, if the lesion lie in the course of Meynert's fibres, although vision may be normal. If the lesion lie in the centre of vision, or in the course of the fibres connecting this centre with the corpora quadrigemina, although absolute amaurosis exist, the reaction of the pupil to light will be perfect. Paralytic mydriasis, due to non-conduction of light stimulus, is found in most cases of optic atrophy.

Damsch has noticed[2] a marked increase of the hippus of the pupil in certain diseased states, namely, multiple sclerosis, acute meningitis, apoplectic attacks followed by secondary tremor and spasms of the paralysed muscles, and in neurasthenia. He is inclined to liken the hippus in these cases to the increase of the tendon reflexes, while immobility of the pupil would be the homologue of loss of tendon-reflex. Yet he does not think an exclusively reflex origin for the exaggerated hippus can be adopted in these cases, as it continues to an abnormal degree even when all reflex irritation is avoided; and, consequently, he concludes that an increase of the physiological hippus must be included as a cause.

Forster[3] finds that in tabes dorsalis the oscillations of the pupil diminish in intensity, while the rhythm remains unaltered; but that in progressive paralysis the rhythm is lost. When the pupil has lost its power of reaction to light, the hippus still continues for a while.

---

[1] Knapp, *Archiv f. Ophthal.*, xiv., pt. 1, p. 220.

[2] *Neurolog. Centralbl.*, 1890, p. 258; also Zeminski, abstract in *Annales d'Oculist*, March 1894, p. 239.

[3] "Versammlung deutscher Naturf. u. Aerzte Nürnberg, 1893" (*Deutsch. Med. Wochenschr.*).

CHAPTER XII.

GLAUCOMA.[1]

The chief, and essential, symptom of this disease is Increased Intraocular Tension—increased hardness of the eyeball—due to over-fulness of the globe.

There is Primary Glaucoma, and Secondary Glaucoma.

In primary glaucoma the increased tension comes on without any previous recognisable disease of the eye; and it is with it we have mainly to do in this chapter.

In secondary glaucoma, the increased tension comes on in consequence of obvious antecedent disease in the eye.

Primary Glaucoma.

Of primary glaucoma, commonly called "glaucoma," there are two great kinds: the Non-inflammatory, Non-congestive, or Chronic Glaucoma; and the Inflammatory, Congestive, or, more or less, Acute Glaucoma. In using the term "inflammatory" here, it is not to be supposed that acute glaucoma is an inflammation in the strict pathological sense of the term, or, if so, to but a slight extent. The term is employed rather on account of some symptoms which are present (pain, redness of the eyeball, lacrimation), and which we are wont to see with inflammations of the eye—symptoms which are wanting in chronic glaucoma.

Increased intraocular tension, then, is the chief, and

[1] From γλαῦκος, *sea-green*. The name was given to the disease by the old writers, on account of the greenish reflection obtained from the pupil in some cases. But this greenish reflection is seen in other diseased conditions, and therefore is not characteristic of glaucoma.

essential, symptom of glaucoma, whatever form of it may come before us; although this increased tension may not be present in the same degree, or indeed at all, at every time.

If the surgeon place the tips of his index fingers close together on a normal eyeball, and make gentle pressure with them alternately, he will observe that the eyeball pits slightly on this pressure, and that a sensation of fluctuation is given to the fingers. The amount of this pitting or fluctuation varies according to the degree to which the eyeball is filled with its humours, and also, to some extent, according to the thickness of the sclerotic coat, and is not precisely the same in every normal eye. The glaucomatous eyeball is felt to be more resistent, to be harder, than the normal globe.

But there are eyes which have normally a low tension, *i.e.*, below the average normal tension; and others which have a tension somewhat above the average normal tension; and, in eyes of the latter class, it is occasionally difficult to decide whether or not the tension is abnormally high; especially if there happen to be symptoms which might be due to high tension. If it be a question of one eye only, then a comparison of its tension with that of its fellow decides the matter, for the physiological tension is always the same in each eye.

Some clinical experience is necessary before the surgeon can appreciate, by palpation, those degrees of tension which are just above, or just below, the normal; and no other method is equally satisfactory. Tonometers have, indeed, been invented for the purpose, but for ordinary use the educated fingers are to be preferred.

For the purposes of clinical notation, Sir W. Bowman suggested some signs, which have been very generally adopted. Normal tension he indicated by the letter T, slight increase of tension = T + 1, still higher tension = T + 2, while T + 3 indicates stony hardness of the eyeball. In the same way, diminished tension is T — 1, T — 2, and T — 3. T + ?

and T —? indicate that it is doubtful whether the tension be slightly above or below the normal. But the application of these symbols to the varying degrees of tension depends very much upon the observer. "T + 2," for instance, will not always convey precisely the same idea to every surgeon.

The other symptoms of glaucoma are largely due to the increased tension, but in chronic glaucoma there are by no means so many symptoms as in acute glaucoma. Let us now discuss these two great forms of primary glaucoma separately. And first as to **Chronic**, or **Non-Inflammatory, Glaucoma** (also

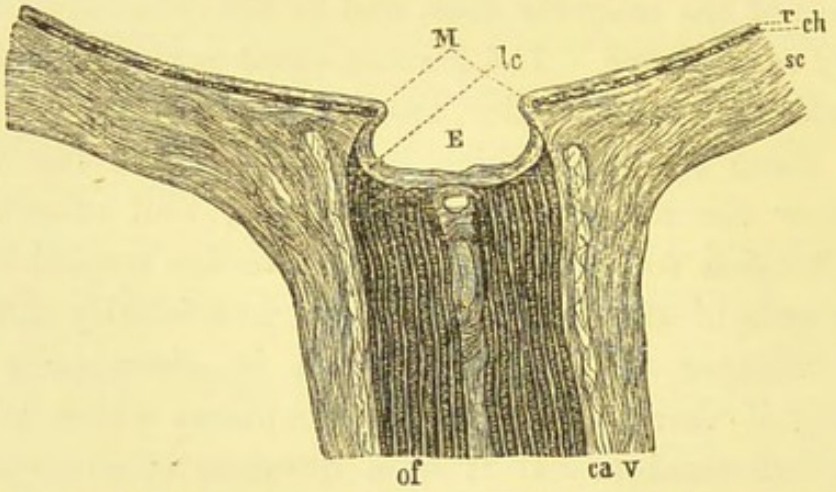


FIG. 106.—(*Ed. Jaeger*), sc, Sclerotic; ch, Chorioid; r, Retina; of, Optic nerve; ca, Intervaginal space; v, External sheath of the optic nerve; E, Excavation of the papilla; M, Margin of the excavation; lc, Lamina cribrosa.

known as Simple Glaucoma, as Simple Chronic Glaucoma, and as Chronic Non-Congestive Glaucoma).—*Symptoms.* The tension is raised. Sometimes the eye will be very hard (T + 2, or more), and again it may be but slightly raised (T + 1). Even in one and the same eye the tension usually varies, and may be at one time too high, and at another almost, or quite, normal.

The external appearance of the eye is usually quite normal, and the pupil reacts well to light. The anterior chamber is sometimes a little shallow.

On examination with the ophthalmoscope the optic papilla

is found to be "cupped." The optic papilla, being the weakest part of the ocular wall, is the first place to give way to the high tension; and, after a time, it becomes depressed or cupped, the excavation being often deeper than the outer surface of the sclerotic, and the lamina cribrosa being pushed back (Fig. 106). This cupping of the papilla is a most important sign of glaucoma, and differs essentially in appearance from the physiological cupping (*vide* p. 85),

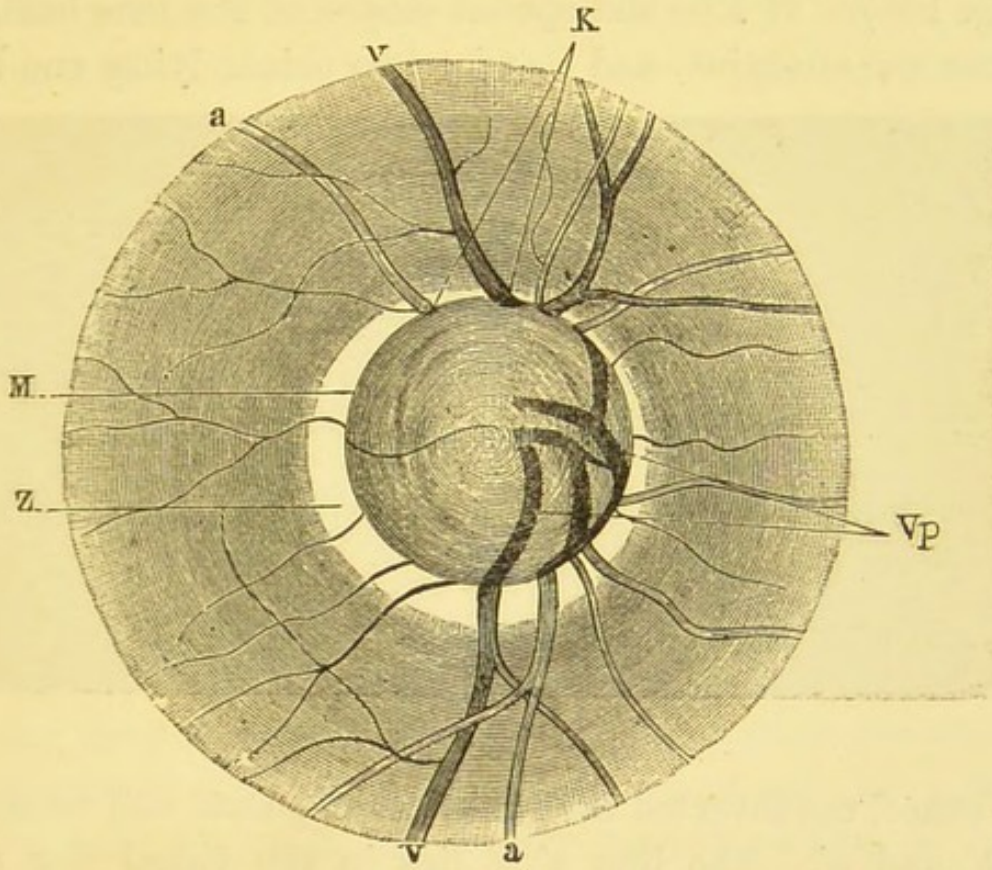


FIG. 107.—(*Ed. Jaeger*), a, Arteries; v, Veins; K, Bending of vessels at margin of the papilla; Vp, Vessels on the floor of the excavation; z, Glaucomatous ring.

inasmuch as it occupies the entire area of the papilla, and has steep, not shelving sides. As shown in Fig. 106, the walls of the excavation are often hollowed out, and the ophthalmoscopic effect of this is to give to the retinal vessels the appearance of being broken off at the margin of the papilla (Fig. 107), where they pass round the overhanging edge of the excavation, and become hidden by it, while on the floor of the excavation they reappear.

The presence of an excavation may be recognised ophthalmoscopically in the examination by the indirect method, by means of lateral motions of the convex lens. It will be then seen that, while the whole fundus seems to move along with the motion of the lens, the floor of the excavation apparently moves in the same direction, but at a slower rate. This parallax is the more marked the deeper the excavation. The phenomenon is explained by the accompanying figure (Fig. 108). If *o* be the optical centre of the lens being used in the examination, and *b* and *a* two points lying one behind

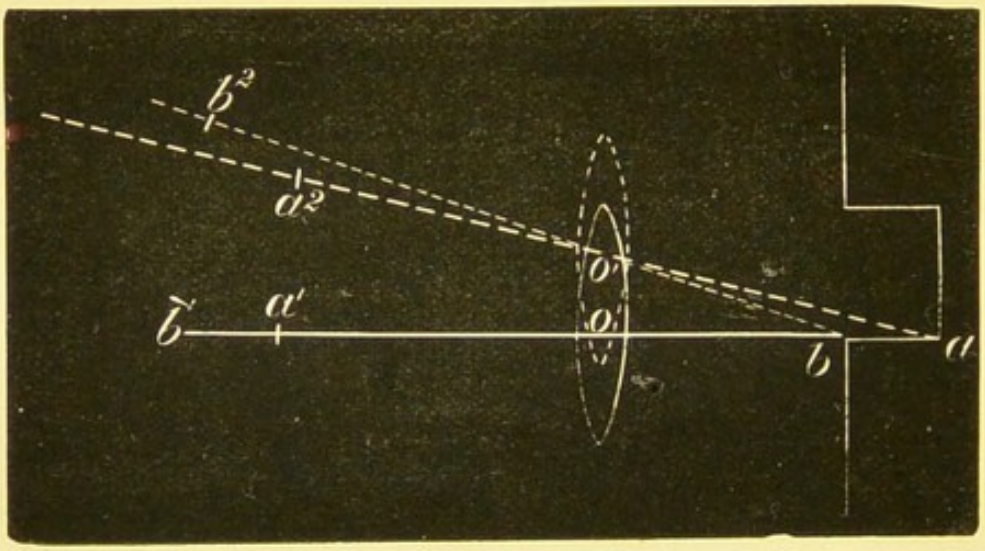


FIG. 108.

the other, the inverted images of these points will be situated at *b'* and *a'*. The line *a' b'* lies in the visual line of the observer; and, if the lens be moved upward a very little, so that the optical centre comes to *o'*, the inverted images of *b* and *a* will be removed to b^2 and a^2. If the observer has not altered his point of view, it will seem to him that the point *b* has made a more extensive motion than the point *a*; or, that it has moved more rapidly than *a*, and has glided between *a* and the observer. Short and rapid motions of the lens from side to side, or from above downwards, will best show the parallax.

In the upright image, the existence of an excavation may be ascertained, by observing that a lens of a different power is

required in order to obtain a clear image of the margin of the papilla, and of its floor. The depth of the excavation may be estimated by noting the difference between these two lenses; *e.g.*, if the general fundus of the patient be emmetropic, and the emmetropic observer require 3 D to see the floor of the excavation, the depth of the latter is about 1 mm., and in the same proportion up to 10 D.

Besides being cupped, the optic papilla becomes atrophied from the pressure, and its consequent pallor serves to aid the diagnosis between this and a physiological excavation. But we meet with cases in which the optic disc is cupped and pale, and in which the existence of increased tension is doubtful; and where there is no history of glaucomatous attacks. And here, sometimes, the diagnosis between glaucoma and primary atrophy of the optic nerve, with cupping of the disc, is one of the most difficult to be met with—indeed, it must sometimes be regarded as impossible. The examination of the field of vision may not always assist, for in each of these diseases it is liable to be contracted.[1] Possibly the effect of a myotic on the intraocular pressure may aid the diagnosis, for it would not materially influence normal tension, while it would reduce abnormally high tension. Also the fact that in glaucoma the L.M. is affected, and the L.D. is almost normal, while in optic atrophy the reverse is apt to be found.

Around the margin of the glaucomatous excavations, especially in chronic simple glaucoma, one usually sees the whitish appearance termed the glaucomatous ring (Fig. 107), which is said to be due to atrophy of the chorioid from pressure.

A pulsation of the arteries on the optic papilla may be

[1] *Bjerrum* regards cases of "atrophy with excavation" as truly glaucomatous, because he finds the fields resemble those of undoubted glaucoma, in their shape, and in the tendency, which the contraction often has, to approach the blind spot. (*Nordisk Ophthalm. Tidsskrift.* v. 1.)

often noted; or, if not present, may be easily produced by very slight pressure with the tip of a finger on the eyeball; because blood can only be forced into these vessels by a pressure greater than that opposed to it. In the normal eye there is no arterial pulsation,—and slight pressure with the tip of the finger would not bring it on—for the tension of the coats of the vessels is greater than the intraocular tension; and, therefore, the blood passes on in a continuous stream; but, in the glaucomatous eye, the intraocular tension opposes so great an obstacle to the arterial flow, that at the systole alone can it make its way through.

Arterial pulsation also occurs, although rarely, in exophthalmic goitre (see chap. xix.); and it occurs where the pressure in the arteries themselves is low (weak heart's action, aortic regurgitation, etc.), although that in the vitreous chamber be normal.

The acuteness of vision is diminished, and increasing dimness of sight is the only symptom in chronic simple glaucoma of which the patient complains. Besides this, the field of vision becomes contracted, in consequence of interruption to the conduction in the retinal nerve-fibres, from pressure on them at the margin of the depressed optic papilla. This contraction of the field must always be examined for by the recognised methods. It commences at the nasal side, as a rule; while, at the same time, central vision is lowered, and later on, the temporal portion of the field becomes contracted, and gradually absolute blindness is brought about.

The Light-Sense in glaucoma is defective, both as regards L.M. and L.D.; or else only as regards L.M., which is much greater than normal.

The progress of the disease is extremely slow, extending often over several years, and ends in total blindness if untreated. It usually attacks both eyes, but generally one of them long before its fellow. Sometimes chronic simple

glaucoma, after a time, takes on the acute, or the sub-acute, form.

Acute, or Inflammatory, Glaucoma. (Also called Acute Congestive Glaucoma.)—In this form the increase of tension is always very marked. In addition to this, there are the following symptoms:—

Diminished Depth of the Anterior Chamber, from pushing forwards of the lens and iris.

Diminution of the Refracting Power of the Eye, by reason of the nearer approach of the latter to a globular shape.

Diminution of the Amplitude of Accommodation, and Anæsthesia of the Cornea, owing to pressure on the ciliary nerves as they pass along the inner surface of the sclerotic.

Opacity of the Cornea, giving its surface a peculiar "steamy" or "breathed-on" appearance, due to œdema of the corneal tissue and epithelium, by infiltration into them of the intraocular fluids from high tension. A similar opacity of the cornea is sometimes seen in iritis and irido-chorioiditis, and in interstitial keratitis.

Indistinctness of the Pattern of the Iris, similarly due to œdema.

Opacity of the Aqueous and Vitreous Humours.

Dilatation and Immobility of the Pupil, the result, according to some, of paralysis of the ciliary nerves, but, according to others, of anæmia of the iris from pressure on its vessels. The pupil is oval, with its long axis vertical.

The Episcleral Veins are large and tortuous, owing to the pressure on the vasæ vorticosæ preventing the discharge by those channels of the chorioidal venous blood, which must then pass off by the anterior ciliary veins.

Subjective Appearances of Light and Colour, and coloured halos, or rainbows, around lamps and candles, are complained of. Similar appearances are sometimes experienced by persons suffering from chronic conjunctivitis.

Pain is a very marked symptom of acute glaucoma, both

in the eye, and radiating over the corresponding side of the head. This pain is often very violent.

Vision is greatly affected, and the field of vision will be found contracted, in cases of some standing.

The Optic Papilla, when the media are sufficiently clear to admit of its being examined, is seen to be cupped, if the disease have continued sufficiently long to bring about this change.

In acute glaucoma we recognise certain *Premonitory Symptoms*, viz. :—Sudden diminution of the amplitude of accommodation, evidenced by the rapid onset or increase of presbyopia, and the consequent necessity for higher + glasses for near work ; and the occasional appearance of coloured halos around the flames of lamps or candles, with attacks of fogginess of the general vision. The duration of one of these foggy attacks may be from a few minutes to several hours. Such attacks are apt to occur after a sleepless night, or after a meal, and are sometimes accompanied by peri-orbital pains. Slight opacity of the aqueous humour, and sluggishness of the pupil, with some dilatation, are present during an attack ; but afterwards, the eye returns to its normal condition, and remains so for weeks or months, until another similar attack comes on. Such a premonitory stage may last a year or longer, but cases also occur in which there is no premonitory stage.

The onset of *The True Glaucomatous Attack* is usually at night. It is accompanied by violent pain radiating through the head from the eye, by pericorneal injection, chemosis, and lacrimation. The aqueous humour is cloudy, the anterior chamber shallow, the iris discoloured, and the pupil dilated to medium size and of oval shape, the cornea "steamy" and anæsthetic. The patient frequently complains of subjective sensations of light, and vision is very defective, or may be quite wanting. Vomiting very frequently accompanies acute glaucoma, and has often led to errors of diagnosis, the

patient's ailment having been taken to be a gastric disease, while the ocular symptoms were regarded as accidental coincidences, as "a cold in the eye," "neuralgia," etc.

An attack such as that just described may, to a great extent, pass away in the course of a few days, but a complete remission of all the symptoms does not come about. Some defect of central vision is left, or, it may be, some slight peripheral defect in the field of vision; the tension does not become quite normal again, and the pupillary motions remain slightly sluggish. Another acute attack of glaucoma comes on in the course of some weeks or months, and it, too, may pass away, leaving the eye in a still worse condition than it found it. The attacks gradually become more frequent; and if, in the intervals, the eye be examined, the cornea and vitreous humour will be found opaque, the optic papilla cupped, and an arterial pulsation may be discovered. Finally, there is no remission from the attack, the violent glaucomatous symptoms become permanent, and all vision is for ever destroyed.

Even when vision has been destroyed, the high tension continues, and gradually produces disorganisation of the tissues of the eyeball (glaucomatous degeneration). The iris becomes atrophied, the lens becomes opaque, and the cornea frequently ulcerates, while hæmorrhages are apt to occur in the anterior chamber. In time, the excessive intraocular tension causes staphylomatous bulging of the sclerotic in the ciliary region, or further back; and, finally, such eyes may become the subjects of acute purulent chorioiditis, and end in phthisis bulbi.

Acute glaucoma almost always comes in both eyes, either at the same time, or with an interval, it may be of weeks, or of months.

The reason why there is so marked a difference between the symptoms and course of chronic and of acute glaucoma, probably is, that, in the former, the increase of tension is very gradual, and, therefore, the eye gradually becomes

accustomed to it; while, in acute glaucoma, the increase is rapid, or sudden, and the circulation of the eye has not time to accommodate itself to the new state of things.

Glaucoma Fulminans is the name given by von Graefe to a form of the disease, which is more acute than the ordinary acute glaucoma just described. It has no premonitory stage, and, coming on with all the symptoms of acute glaucoma greatly exaggerated, does not remit; and causes complete permanent destruction of vision in the course of a few hours. It is a rare form.

Subacute Glaucoma.—This form differs from acute glaucoma, in that its premonitory stage merges gradually into the actual disease, without the occurrence of an acute attack. The eye gradually becomes hard, the pupil dilated, the anterior chamber shallow, the aqueous humour opaque, while the cornea is "steamy" and anæsthetic, and the episcleral veins are distended. Ophthalmoscopically the cupped disc and pulsating arteries may be seen, where the opacities of the media permit. Vision sinks, and the field is contracted towards its nasal side. The progress of the disease is very slow; and, in its course, attacks of ciliary neuralgia, with greater increase of the tension, greater opacity of the aqueous humour, increase of the corneal opacity and anæsthesia, and further dimness of vision, are experienced.

These attacks pass off again in the course of a few days or hours, leaving the eye harder and blinder than before. The subacute glaucoma sometimes takes on the acute form. It is liable to bring about the same glaucomatous degeneration of the eye, as does the latter.

Etiology of Glaucoma.—Glaucoma is a disease of advanced life, occurring most usually after fifty years of age, and rarely under the thirtieth year. It is not peculiar, or more common to any one constitution or temperament. Anxiety, sorrow, and influences in general which depress the spirits, have often been noticed to precede the onset of acute glaucoma.

As regards the *Pathology of Glaucoma*, the theory which, of late years, has obtained most acceptation, owes its origin to Max Knies [1] and Adolf Weber,[2] and is known as the Retention

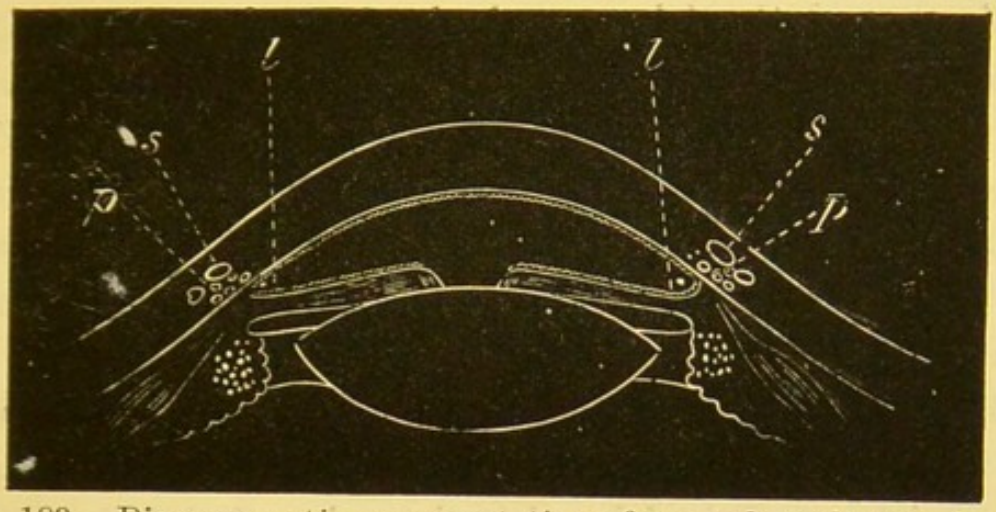


FIG. 109.—Diagrammatic representation of normal condition. *l*. Angle of anterior chamber, and ligamentum pectinatum. *s*. Canal of Schlemm. *p*. Venous plexus of Leber.

Theory. These observers ascertained that, in glaucomatous eyes, the periphery of the iris lies in contact with the periphery of the cornea (Figs. 109 and 110) in the region of the canal

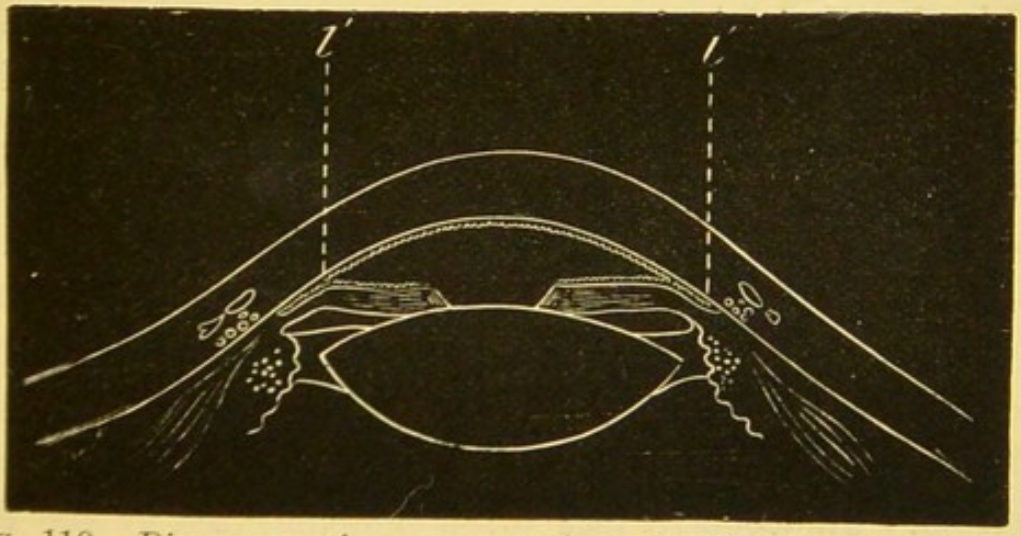


FIG. 110.—Diagrammatic representation of glaucomatous condition. *l'*. Obliterated angle of anterior chamber.

of Schlemm, venous plexus, and ligamentum pectinatum. But this region, and these tissues, having previously been proved by Leber [3] to be the ways of exit of the effete intraocular

[1] *Von Graefe's Archiv*, xxii., pt. 3, p. 163, and xxiii., pt. 2, p. 62.
[2] *Ibid.*, xxiii., pt. 1, p. 1.
[3] *Ibid.*, xix., pt. 2, pp. 87-185.

fluids, which flow to that point from the posterior part of the aqueous chamber through the pupil, Weber and Knies concluded, that the blocking of these passages from the close application of the iris caused glaucoma, by preventing the effete fluids from escaping; and thus the disease was rendered one of retention, rather than of hypersecretion, as it had previously been considered to be. Weber believes that swelling of the ciliary processes, from one cause or another, pushes the periphery of the iris forwards, and gives the starting-point for glaucoma.

Brailey,[1] to a certain extent, adopts this view of Weber, but regards[2] a chronic inflammation of the ciliary processes and periphery of the iris, with distension of the blood-vessels of these parts, to be the chief factor in the earliest history of the disease.

Max Knies[3] also now regards glaucoma as an irido-cyclitis, which, owing to varying intensity, produces the different forms of the disease.

Priestley Smith[4] adopts the retention theory; and holds, that the main predisposing cause of primary glaucoma is an insufficient space between the margin of the lens and the structures which surround it; and he attributes the greater liability of elderly people to the progressive increase in the size of the lens, which he has proved[5] to occur as life advances. In eyes in which the circumlental space is insufficient, by reason either of the original structure of the eye—and small eyeballs, as Priestley Smith has shown, are specially liable to primary glaucoma, a fact often demonstrated by the small

[1] *Ophth. Hosp. Rep.*, x., p. 282.

[2] *Ibid.*, ix., p. 199, and x., pp. 14, 89, 93.

[3] *Archiv. f. Ophthalm.*, Knapp and Schweigger. German Ed., xxxviii., 1894, p. 193.

[4] On Glaucoma, 1879, *Ophth. Hosp. Rep.*, x.; *Trans. Internat. Med. Congress*, 1881; *Ophthalmic Review*, July 1887. *Pathology and Treatment of Glaucoma*, London, 1891.

[5] *Trans. Ophth. Soc. United Kingdom*, iii., p. 79.

size of the cornea in the eyes attacked—or of the enlargement of the lens, any condition, which tends to overfill the veins of the head and uveal tract, may initiate an attack of acute glaucoma, as follows :—An increase in the amount of blood in the uveal tract must be compensated by the expulsion of some other fluid from the eye—the aqueous humour filters out more rapidly at the angle of the anterior chamber. As the contents of the chamber diminish, the lens and iris move forwards towards the cornea. Now, in the normal eye, and especially in the youthful eye, this compensation is effected without danger to the angle of the anterior chamber, because the lens is comparatively small, the circumlental space large, and the anterior chamber deep. But, when the lens and ciliary processes are already in close relation to each other, and the anterior chamber already shallow, then any increased fulness of the uveal tract involves danger to the angle of the chamber. The turgid ciliary processes find insufficient space for their expansion ; they are carried forwards together with the lens, and, pressing upon the base of the iris, lock up the angle of the anterior chamber. Thereupon, the further escape of fluid being impossible, high tension of the eyeball is established. According to this explanation, then, the high tension is due to impeded escape of the intraocular fluid, not to hypersecretion, and depends primarily rather upon an increase in the amount of blood in the eye, than on an excess of the intraocular fluid. Mr. Priestley Smith considers, that, in chronic simple glaucoma, the predisposing causes are the same as in acute glaucoma, but that in the former, the vascular disturbance being gradual and slight, the vessels adapt themselves to the slowly increasing pressure, and the angle of the anterior chamber is more or less compressed, but not tightly closed.

Von Graefe[1] believed that a serous chorioiditis lay at the root of the disease, which he thought was caused by exudation

[1] *Archiv f. Ophthal.*, xv., pt. 3, p. 108, and elsewhere.

of serous fluid into the vitreous humour; while Donders,[1] von Hippel and Grünhagen,[2] and others, held that irritation of the fifth pair of nerves, governing the secretion of the intraocular fluids, gave rise to hypersecretion of those fluids.

Others again held that changes in the sclerotic, rendering it rigid, and leading to some shrinking of it, caused the increased intraocular tension.

Laqueur[3] believes, that some such sclerotic changes produce obstruction of the posterior ways of exit of the intraocular lymphatics, namely, those which pass out with the four vasæ vorticosæ, and that glaucoma depends largely upon this obstruction.

Treatment.—The performance of an iridectomy is the means discovered by von Graefe,[4] in the year 1857, for the cure of glaucoma, a disease which had hitherto been incurable. This measure held an undisputed position as the sovereign remedy for the disease until a few years ago, and even yet has not suffered much from the competition of the operation of sclerotomy.

To ensure the success of an iridectomy for glaucoma, so far as possible, it is necessary:—1. That the incision should be peripheral, *i.e.*, as far back in the corneo-sclerotic margin as is compatible with the introduction of the knife into the anterior chamber, and with the avoidance of injury to the ciliary body. 2. That the portion of iris removed should be wide, *i.e.*, involving about one-fifth of the entire circumference of the iris (see p. 273 and Fig. 102).

It is, moreover, important to withdraw the knife very slowly from the anterior chamber, when the corneo-sclerotic section is complete; in order that the aqueous humour may flow off gradually, and the occurrence of an intraocular

[1] *Archiv f. Ophthal.*, ix., pt. 2, p. 215.
[2] *Ibid.*, xiv., pt. 3, xv., pt. 1, and xvi., pt. 1.
[3] *Von Graefe's Archiv*, xxvi., pt. 2.
[4] *Archiv f. Ophthal.*, iii., pt. 2, p. 456.

hæmorrhage from the sudden reduction of tension be avoided. The portion of iris should be most carefully abscised, so that no tag of it may remain in the wound, and become caught in the cicatrix in the course of healing. Such an occurrence is apt to produce a cystoid cicatrix, which may at a later period become the starting-point of irritation, and even of serious inflammation. Some operators prefer von Graefe's cataract knife for the performance of the operation, but the ordinary lance-shaped iridectomy knife is the instrument usually employed. For the purpose of reducing the intraocular tension, it matters nothing what region of the iris be abscised; but, as a rule, the upper quadrant is to be preferred, for there the resulting coloboma, being covered to a great extent by the upper lid, will give rise to less diffusion of light than in any other position.

Immediately after the operation, palpation of the eyeball should show a marked diminution of tension. When this is not so, the prognosis is unfavourable. Should an increase of tension occur on the day after the operation it is of no consequence, as it passes off again in the course of the next few succeeding days. Until then the anterior chamber will not be restored, and we see cases where the anterior chamber does not appear for a week or more. The bandage should be worn until the anterior chamber is completely restored. I do not care for the use of eserine after a glaucoma operation, as I think it sometimes produces iritis. Von Graefe recommended that if, immediately after the iridectomy, the intraocular tension continue high, no bandage should be applied, as he believed it to do harm, but advised that the eyelids should simply be kept closed with a strip of court plaster. The pain for some time after the operation is considerable, but may be relieved by a hypodermic injection of morphia in the corresponding temple.

Very occasionally, immediately after iridectomy, although the operation may have been faultlessly performed, the case

takes what we call a "malignant" course. In these cases the lens seems to be violently pushed forwards, blocking the wound, obliterating the angle of the anterior chamber, and preventing any fluid from escaping from the eye, so that very soon it is as hard, or harder, than before. This complication seems to be caused by the retention of fluid behind the lens, and is more likely to occur in cases of chronic simple glaucoma than in the acute forms of the disease.

Unless the method recommended by Adolf Weber for these cases be employed with success, all such eyes are inevitably lost, are apt to become very painful, and must often be excised. Weber[1] introduces a broad needle, or a Graefe's knife, through the sclerotic, 8 or 10 mm. behind the outer margin of the cornea, and gives the blade a quarter turn on its axis, so as to make the wound to gape. At the same time he applies a gradually increasing pressure, by means of the upper lid, on the centre of the cornea. This causes fluid to escape through the scleral wound by the side of the knife, and it also causes the lens to go back into its place, with restoration of the anterior chamber. The pressure on the cornea may be maintained, with advantage, for a minute or somewhat longer. Mr. Priestley Smith speaks highly of this procedure, but, I am happy to say, I have not had the necessity to resort to it.

As a rule, the more acute the form of glaucoma, and the earlier in the disease the iridectomy be performed, the more favourable is the prognosis in respect of the result which may be expected. The saving of normal vision can only be looked for in those cases, chiefly of the acute form, where it has as yet fallen but little, or not at all, below the normal, and where the contraction of the field has barely commenced. When the disease has interfered seriously with vision (of course I do not refer here to the enormous loss of sight immediately attendant upon an attack of acute glaucoma, for this is

[1] *Von Graefe's Archiv*, xxiii., 1, p. 86.

usually restored), we should not expect more than the retention of the *status in quo*. But our prognosis, even in this respect, should be most guarded, especially in chronic simple glaucoma, when the contraction of the field is found to have approached close to the fixation point, although central vision may be fairly good. Because, in such cases, while the iridectomy may prove successful so far as reduction of tension is concerned, yet the contraction of the field, *i.e.*, the progress of the atrophy of the optic nerve, is often not arrested, and shortly afterwards may be found to engulf the centre of vision. I go so far as to think that in such cases any operation is liable rather to hasten, than to retard, the blindness, and I therefore never operate on them. It may, indeed, be stated, that while the result obtained from iridectomy in acute and subacute glaucoma, on the bases above laid down, can be regarded as amongst the most satisfactory in the whole range of ophthalmology ; in chronic simple glaucoma iridectomy does not act with the same degree of success, and the prognosis should therefore be guarded in these cases.

In cases of acute or subacute glaucoma, it has frequently been observed that shortly, even within a few hours, after the performance of the iridectomy, the other eye, previously healthy, or, at most, affected with but slight premonitory symptoms, is attacked with glaucoma. It is probable that this is due to dilatation of the pupil, with crowding of the iris into the angle of the anterior chamber, in consequence of confinement in the dark room. Hence some operators put eserine into the sound eye as a precaution.

It may here again (*vide* p. 263) be stated that the use of atropine, or of any other mydriatic,[1] in an eye with a tendency to glaucoma, is liable to bring on an acute attack of the disease, and must be carefully avoided in such cases.

If the tension be not relieved by the iridectomy, a supple-

[1] See *Ophthal. Review*, 1893, p. 69, where Story records a case of glaucoma produced by cocaine in a child of thirteen years of age.

mental iridectomy may be performed after a time, and von Graefe recommended that it should be placed at the opposite side of the pupil from the first coloboma.

The Mode of Action of the Operation is not clearly known. Von Graefe at one time believed it to act by diminution of the secreting surface of the intraocular fluids. De Wecker[1] and Stellwag[2]—even previously to the formulation by Knies and Weber of the retention theory of glaucoma already referred to—held that the cure depended, not on the removal of the portion of iris, but on the incision in the corneo-sclerotic margin, or rather, on the nature of the cicatrix resulting from that incision. They maintained that this cicatrix was formed of tissue, which admitted of a certain amount of filtration through it of the intraocular fluids, and that in this way the intraocular tension was kept down to the normal standard. This theory has gained support from that of Knies and Weber.

Priestley Smith has satisfied himself that, in a large number of successful iridectomies, the success is due to a permanent corneo-scleral fistula—not merely a filtration cicatrix—having been formed. The same view is held by Treacher Collins,[3] who finds that this permanent gap is maintained by a prolapse of a fold of iris into the wound. The latter author, indeed, entirely and definitely discards the filtration-cicatrix theory, for which he considers there is no evidence. In those cases where a fistula, as described, is not formed by the operation, Treacher Collins considers that the obstruction becomes freed, either by the iris being torn away at its thinnest part, that is, its extreme root, thus leaving a large portion of the filtration angle open for drainage; or, by the escape of the aqueous, and the drag on the iris, incident on the iridectomy, being sufficient to dislodge the periphery of an iris, which has only recently come into apposition with the cornea.

[1] *Bericht der Ophthal. Gesellsch. zu Heidelberg*, 1869.

[2] *Der Intraoculare Druck*, etc. Vienna, 1868.

[3] *Roy. Lond. Ophthal. Hosp. Rep.*, Dec. 1891.

De Wecker, Stellwag,[1] and Quaglino[2] sought to produce the corneo-scleral filtration-cicatrix without the removal of a portion of iris. The peripheral position of the wound, however, rendered the proceeding difficult or impossible, owing to the tendency to prolapse of the iris which necessarily existed. The introduction of eserine into ophthalmic practice at last enabled de Wecker to place the operation on a surer footing, as the myosis, produced by instillation of a solution of this drug into the eye, ensured the operator, to a great extent, against the danger of prolapse of the iris, and hence

Sclerotomy, as the operation is called, has come to be cultivated as a method for the relief of glaucoma, and has proved useful as such. It has hitherto been employed more in chronic simple glaucoma; a form in which, as I have stated, iridectomy is less satisfactory than in acute or subacute glaucoma. Care must be taken that the pupil is contracted to pinhole size or nearly so, when the operation is about to be performed, as otherwise the danger of prolapse of the iris is very great. In those cases where eserine will not produce a sufficient myosis, sclerotomy should certainly not be performed.

Priestley Smith and Treacher Collins explain the cure by sclerotomy in the same way as they do that by iridectomy.

The instrument used for performing the operation is von Graefe's cataract knife. A speculum having been applied, and the eyeball fixed, the point of the knife is entered into the anterior chamber, through the corneo-sclerotic margin, at a point of its circumference corresponding to that selected for the puncture in cataract extraction, but 1 mm. removed from the corneal margin, as represented at *a* in Fig. 111. The counter-puncture is made at a point

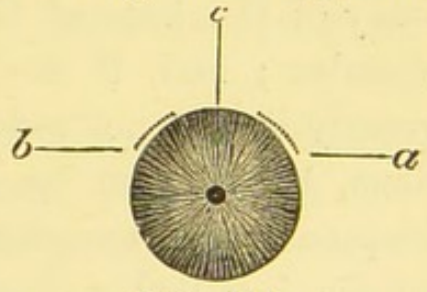


FIG. 111.

[1] *Bericht der Ophthal. Gesellsch. zu Heidelberg*, 1871; *Chirurgio. Oculaire*, p. 212. Paris, 1879.

[2] *Annali di Ophthalmologia*, i., pt. 2, p. 200, 1871.

(*b*) corresponding to this, at the other side of the anterior chamber. With a sawing motion of the knife the section is enlarged upwards, until only a bridge of tissue, about 3 mm. broad, remains at *c*, and this is left undivided, the better to guard against prolapse of the iris. The knife is now slowly withdrawn from the eye, care having been first taken that the aqueous humour is thoroughly evacuated, which can be effected by tilting the edge of the knife slightly forwards, so as to make the lips of the wound gape somewhat. If the pupil be quite round at the conclusion of the operation, the bandage may be applied, a drop of solution of eserine having been first instilled. But if the pupil be oval, or of other irregular shape, a tendency to prolapse of the iris is indicated, and the hard rubber or silver spatula should be introduced into the anterior chamber, to restore the pupil to its normal shape by gentle pushing of the iris. If there be an actual prolapse of the iris, an attempt may be made to repose it with the spatula; but, should this not prove satisfactory, the prolapse is to be abscised with scissors, thus turning the sclerotomy into an iridectomy.

The Treatment of Glaucoma by Myotics.—Eserine and pilocarpine as eye-drops in 2 per cent. solutions often have the power of reducing glaucomatous tension. This power depends on the contraction of the pupil, and consequent drawing away of the base of the iris from the angle of the anterior chamber; and, if the myotic does not contract the pupil greatly it will not reduce the tension. Cases of acute glaucoma, brought on by the injudicious use of atropine, may frequently be completely and permanently relieved by a myotic instilled a few times. In acute glaucoma of the ordinary type, the use of a myotic in the premonitory stage will often postpone the true glaucomatous attack, and even sometimes relieve the latter for the time; but the myotic treatment cannot produce a radical cure, and it should only be used to preserve the health of the eye until the operation

is performed. In chronic simple glaucoma, also, myotics bring down the tension if they contract the pupil, and may be used in those cases where the patient will not submit to an operation, or where an operation in the fellow-eye has not resulted satisfactorily, or where an operation is contra-indicated by a very contracted field. The anti-glaucomatous action of the myotic only lasts so long as the pupil is contracted; and, if the pupil cannot be contracted, no such action is to be looked for.

In the myotic treatment of glaucoma, Priestley Smith recommends the combination of cocaine with the myotic, in such proportions (say, about $\frac{1}{5}$% of cocaine to 1% of the myotic) that the myotic will have the mastery over the pupil. For although, like every dilator of the pupil, when used alone, cocaine may promote high tension, yet it has the powers, invaluable in glaucoma, of contracting the ciliary blood-vessels, and of diminishing the sensibility of the ciliary nerves; and, when used in the foregoing manner, the advantage of each drug may be obtained, without any of the disadvantages of either.

It may be here, once more, stated, that while myotics possess the power of reducing glaucomatous tension, atropine, and all mydriatics used alone, bring on glaucoma, where there is already a tendency to it. In all old people, therefore, before atropine is used, it is well to ascertain that the tension is not too high,

Treatment of Painful Blind Glaucomatous Eyes.—Eyes blind of acute glaucoma may, as I have stated, continue to be painful; and may in this way render the patient's life very miserable. Iridectomy is very commonly performed to relieve the pain, although all hope of restoration of sight is lost; but the operation sometimes fails in its object. Neurectomy (chap. x., p. 296) seems to offer a more certain result, and, of course, enucleation or evisceration would have the same effect.

Secondary Glaucoma.

In addition to the different forms of primary glaucoma above described, we find, as already stated, that high tension occurs as a sequel of diseased conditions previously existing in the eye. There are several diseased states which are liable to become complicated with glaucomatous tension; but it should be clearly understood that, in almost every instance, the immediate cause of the high tension is the same as in primary glaucoma, namely, a closure of the angle of the anterior chamber. The following are the chief conditions which are liable to lead to secondary glaucoma:—

a Complete Posterior, or Ring Synechia (*vide* p. 256). The iris being pushed forwards by the aqueous humour, pent up behind it in the posterior part of the aqueous chamber, is pressed tightly against the cornea, and obliterates the angle of the anterior chamber and the ways of exit. An iridectomy relieves the high tension here.

b Perforating Wounds or Ulcers of the Cornea, followed by incarceration of the iris in the resulting cicatrix. The iris being drawn tautly towards the cornea, a large portion, or the whole, of the filtration angle may be closed by it. An iridectomy is indicated. Lang divides anterior synechiæ by means of his twin knives.

c Dislocation of the Crystalline Lens into the Anterior Chamber. Here the normal flow of the intraocular fluids through the pupil (*vide* p. 325), on its way to the filtration angle, is arrested by reason of the presence of the lens in the anterior chamber. The onward current then presses the iris against the posterior surface of the lens, and its periphery, which is unsupported by the lens, against the periphery of the cornea, and in this way the angle of the anterior chamber is closed. Here the lens must be removed from the eye.

d Lateral (traumatic) Displacement of the Crystalline Lens. The lens being pushed in between the ciliary processes

and the vitreous humour, drives the root of the iris forwards against the cornea at that place, while in other parts of the circumference the displaced vitreous acts in the same way. In these cases, too, the lens must be removed.

e Injury of the Crystalline Lens (*vide* chap. xiii.). The swelling lens pushes the iris forwards against the angle of the anterior chamber. Evacuation of the lens should be performed.

f After Cataract Extraction. For explanation of this see chap. xiii.

g Intraocular Tumours (*vide* p. 286). The growth of the tumour gives rise to a transudation of serum from the chorioid which detaches the retina, and after a time pushes the lens, the ciliary processes, and the iris forwards, and thus closes the filtration angle.

h Serous-Cyclitis, or Iritis. Here the filtration angle is not closed. Mr. Priestley Smith thinks that the increased tension is due to diminished filtration-power of the eye, and perhaps by tissue changes around the filtration angle.

Another, and very peculiar, form of secondary glaucoma is :—

Hæmorrhagic Glaucoma.—Retinal hæmorrhages of the ordinary type are sometimes followed, a few weeks later, by increased intraocular tension, which generally assumes the symptoms of acute or subacute glaucoma, and, more rarely, those of chronic simple glaucoma. A satisfactory explanation for these cases has not, so far as I am aware, been offered. When such a glaucoma has become pronounced, it is not usually possible to distinguish it from a primary form of the disease.

Treatment.—Iridectomy in hæmorrhagic glaucoma is more likely to do harm than good, the operation being almost invariably followed by fresh intraocular hæmorrhages, and by a further increase of tension. Sclerotomy is said by some to act with fairly good results in hæmorrhagic glaucoma. The myotic treatment is powerless.

Congenital Hydrophthalmos,

also known as Buphthalmos, and Cornea Globosa, is a disease of early childhood, of which the incipient stages are believed to be intra-uterine. The cornea becomes enormously enlarged in diameter, the anterior chamber deep, the iris trembling, and the sclerotic thinned. Increase of tension, often attended by severe pain, and cupping of the optic papilla, are usually present. The disease is regarded as a secondary glaucoma, although it is by no means certain that it should not rather be considered as a form of primary glaucoma, occurring in young children.

Treatment.—Iridectomy and sclerotomy are alike followed by disastrous results in this disease. The myotic treatment is the only one applicable, and in a few cases it arrests the disease.

CHAPTER XIII.

DISEASES OF THE CRYSTALLINE LENS.

Cataract, by which is meant an opacity of the lens, may be said to be the only disease of this part of the eye. Cataract may be complete, *i.e.*, occupying, in its final stage, the whole, or nearly the whole, of the lens; or partial, *i.e.*, occupying only part of the lens, and with little or no tendency to extend to other parts of it.

Complete Cataracts.

Of these, the most common is **Senile Cataract.** It occurs in persons of over fifty years of age, rarely in those under forty-five years of age.

Progress, Pathogenesis, and Etiology of Senile Cataract.—In commencing or incipient senile cataract, the opacity is found in the cortical layers of the lens, especially at its equator, and in the latter position can often only be detected with transmitted light from the ophthalmoscope mirror, or with oblique light, even when the pupil is dilated with atropine. This opacity takes the form of lines, or of triangular sectors, of which the bases are towards the equator of the lens, while the apices are towards its centre. These lines and sectors look black with transmitted light, but grey with oblique light, and between them clear lens substance is present. Or, incipient cataract may first appear as a diffuse opacity in the layers surrounding the nucleus of the lens. Or, the opacity may commence both near the equator and around the nucleus at about the same time. Or, again, the opacity may in the beginning be disseminated through the

cortex, in the form of flocculi, dots, and lines. In some cataracts, in a very incipient stage, there are no absolute opacities; but, with weak transmitted light, *i.e.*, from a plane mirror, numbers of fine dark lines will be seen in the lens, which vanish and reappear, according as the incidence of the light is altered; while a little later on true opacities make their appearance. Gradually the cataract extends to other parts of the lens, until the whole cortical portion is opaque.

In senile cataract the very nucleus itself does not become cataractous, although it is usually sclerosed (harder and drier). Sclerosis of the nucleus of the lens is a physiological condition of advanced life, and will be found in many an eye where there is no cataract. It gives to the non-cataractous lens, as seen with a dilated pupil, or with focal illumination, a peculiar smoky appearance, which is often mistaken by inexperienced persons for cataract; but examination with transmitted light will show that there is no opacity. When a senile cataract has become complete, the sclerosed nucleus imparts to its centre a brownish or yellowish hue, while the other parts of the lens are of a greyish white. As a rule, the most peripheral layers of the cortex are the last to become opaque. According as the lens becomes opaque, it swells somewhat; and the anterior chamber, consequently, becomes a little shallower.

Until the whole cortex is opaque, a clear interval will be present between the iris and the cataractous part, and, on examination with the oblique light, a shadow of the iris will be thrown on the cataractous part at the side from which the light comes; and the cataract, in this way, is proved to be immature. If the whole cortical substance be opaque, the thickness of the capsule alone will intervene between the pupillary margin and the opacity. In addition to this examination with the focal light, the pupil should be dilated, and the lens examined by transmitted light from the ophthalmoscope mirror, when a completely opaque cataract should

permit of no red reflection being obtained in any direction from the fundus oculi.

As soon as the whole of the cortical substance has become opaque, the swelling of the lens begins to subside, and the anterior chamber finally regains its normal depth. If there be no glittering sectors in the cortex, the cataract is now "mature," or "ripe" for operation, *i.e.*, if an extraction operation be now undertaken, it is possible to deliver the lens in its entirety; whereas, prior to this stage, some cortical substance would have been liable to adhere to the capsule, and be left behind.

But a cataract is immature, despite the absence of shadow from the iris, of the illuminable pupil, and even though the anterior chamber be of normal depth, if the cortex present well-marked, glittering sectors. The glitter of the different sectors varies with the angle of illumination, so that the surface appears faceted. In such a lens there are thin transparent flakes, as well as opaque flakes, close beneath the capsule; and, if extraction be undertaken, the former are very apt to remain within the eye in spite of every effort to remove them. A few months later the sectors lose their sharp contour, break down, and finally disappear. We can then depend upon the exit of the whole cataract.

Yet, I will not deny that in persons over sixty years of age, in whom the nucleus is usually large, many a cataract can be completely removed which does not quite come up to the standard of maturity just laid down; and, at that time of life, I would not hesitate to operate, without waiting for absolute maturity, if the patient were materially incommoded for want of sight.

The foregoing is the most common course of events in the progress of a senile cataract, but there is a rather rare form of it, in which total opacity of the cortical layers never does come about. In this form the lens is occupied by radiating linear opacities up to the very capsule; but between these

opaque lines there are clear intervals, which may even admit of the fundus oculi being examined, although dimly, and which allow of a certain amount of sight.

After the stage of maturity, a cataract gradually goes on to be hypermature. Here one of two changes takes place. Either the cortical substance breaks down, and becomes fluid, the nucleus retaining its consistency, and gravitating to the lowest part of the capsule (Morgagnian cataract); or, more commonly, the cortical substance dries up, as it were, and finally comes to form, with the nucleus, a hard flat disc. Accompanying these changes in the lens substance are changes in the epithelium lining the inner surface of the anterior capsule, which result in a thickening of the capsule. In a Morgagnian cataract the fluid cortex finally undergoes absorption, and the anterior and posterior capsules come in contact (cataracta membranacea). In some cases the capsule remains more or less transparent, and the sight may greatly improve. About twenty cases are on record of spontaneous cure of cataract, due to intracapsular absorption.[1]

The investigations of Priestley Smith[2] have shown, that a diminished rate of growth of the lens precedes the formation of cataract; and it is held that the cataractous process in the senile lens is the result, in the first instance, of a rapid sclerosis and shrinking of the nucleus. If the process of sclerosis and shrinking be very gradual, cataract does not appear, because the cortical layers of the lens have time to accommodate themselves to the altered state of things; but, if the shrinkage be rapid, the cortical layers cannot so rapidly accommodate themselves, and then the fibrillæ of these layers become separated somewhat from each other, and fluid collects in the interspaces. This fluid it is, which causes the disintegration of the lens substance, gradually leading to opacity of the whole lens. As the opacity in-

[1] Mitralsky, *Centralbl. f. prakt. Augenheilkunde*, Oct. 1892.

[2] *Trans. Ophthal. Soc.*, 1883, p. 79.

creases, more fluid is present in the lens, and it is this which causes the swelling of the lens already referred to. When the whole cortex has become opaque, the fluid contents begin to diminish, and the lens returns to its normal size. Senile cataract, then, is entirely a local process, and is not dependent on any disordered state of the general health.

The dimensions of the nucleus vary a good deal. In some cataracts it is very small, and these are called soft cataracts, as they consist chiefly of the soft cortical substance. In others—and, as a rule, in patients over sixty years of age—the nucleus is large, and these are called hard cataracts, although they are not hard throughout. The size of the nucleus can be estimated pretty accurately, by the extent and intensity of the yellowish or brownish reflection, which is obtainable by focal illumination out of the centre of the cataract.

In some senile cataracts the sclerosis is not confined to the nucleus, but extends to the cortical layers as well. This causes much disturbance of sight, and the term cataracta nigra is given to these lenses, from their very dark hue, although they are not cataracts in the true sense of the term. They require operation, and, as they are always of large size, wide openings have to be made to deliver them.

In the lenses of young people there is no nucleus; consequently, in the complete cataracts of children, and of young adults, there is no nucleus; the whole lens becomes opaque, and the cataract is always soft. Although the starting-point of cataract in children and young adults cannot be a shrinking of the nucleus, as there is none, yet the opacity is, no doubt, due to the taking up of fluid by the lens.

The Symptoms to which senile cataract gives rise consist, in the earliest stages, in the appearance of motes before the eyes, and of monocular polyopia. Motes are complained of also in disease of the vitreous humour, but in those cases they float over a large portion of the field of vision, while in

commencing cataract they occupy always the same relative position in the field. The polyopia is the result of irregular refraction in the media, which causes many images of the objects looked at to be formed on the retina. This symptom seems to annoy the patients, more especially in the evening, when they look at gas or candle flames, the moon, etc. It is often complained of before there is any actual opacity in the lens, at a time when only the clefts filled with fluid between the fibrillæ can first be detected with weak transmitted light, as dark lines vanishing and reappearing according as the incidence of the light is altered.

In some cases of incipient cataract, there is an increase in the refracting power of the lens; with the result, that the patient becomes slightly myopic, if, previously, he have been emmetropic.

Gradually, as the opacity of the lens extends to other parts of it, the acuteness of vision become affected; and this is the more marked, the more the cortex at the anterior and posterior poles of the lens is involved. In those cases where the equatorial parts of the lens are but little affected, while the polar regions are a good deal so, the patients see better in the dusk, or with their backs to the light, than when their eyes are exposed to a strong light. The reason for this is, that in the dusk the pupil is dilated, and light can pass through the clearer periphery of the lens, while in a strong light the pupil is contracted. On the other hand, when the opacity is confined rather to the equator of the lens, a strong light is not disturbing to sight; or, if the centre of the lens be quite clear, a strong light may even be pleasant to the patient.

But, according as the lens becomes more and more opaque, the acuteness of vision is reduced; until, finally, even large objects cannot be discerned, and only quantitative perception of light is left. Some cataracts, when quite ripe, still admit of finger-counting at a few feet.

In advanced stages of the disease, as the opacities occupy a great portion of, or the entire cortex, they are easily recognised even by ordinary daylight, often giving a greyish appearance to the pupil. Inflammatory exudation in the area of the pupil would afford a somewhat similar appearance, but would be attended by other signs of the previous inflammatory process, such as synechiæ, disorganisation of the iris, etc., and it would be seen to lie more in the plane of the iris, than does any lental opacity.

The length of time occupied by the ripening of a cataract varies, in different cases, from a few months to many years. In the very old the progress is, in general, more rapid than at an earlier time of life. That form which commences at the equator as fine lines, is slower than that with flocculent opacities, or than that in which the cortex around the nucleus is likewise implicated at an early period.

All examinations as to the condition of the lens are rendered easier, and more conclusive, if the pupil be previously dilated with atropine; but the tension of the eye should be ascertained before atropine is instilled, lest glaucoma, or a tendency to it, be present.

Treatment.—No external local applications, nor internal medicines, are of any avail in the treatment of cataract at any stage. Removal of the cataract from the eye by operation is the only cure for blindness caused by it.

In cases of incipient cataract, or in those, rather, which have advanced somewhat beyond this stage, we often find that vision is improved, or made more pleasant, by the wearing of tinted glasses to moderate the light. With commencing cataract where slight myopia has come on, low concave glasses for distant vision will be found of service; while for reading, stenopæic glasses often give good results.

Dilatation of the pupil with atropine is, in many cases, of the greatest benefit, especially where the nucleus is much more opaque than the cortical portion; but, sometimes, the

diffusion of light resulting is most distressing to the patient, and greater impairment and confusion of vision are produced, and for this reason care in the prescription of atropine is demanded.

Patients with incipient or advancing cataract may, with immunity, be allowed to make every use they can of the sight they possess; and the surgeon should give them hints as to the arrangement of light in their rooms, and for their work, etc., so as to enable them to use their eyes to the best advantage.

The truly distressing period in the progress of cataract, when both eyes are affected, lies between the advent of that degree of blindness which incapacitates the patient for reading or writing, or for making his way about alone, and the occurrence of maturity, or of that degree of maturity which is deemed requisite for successful removal. This is often a lengthened time; it may be months or years. Fortunately, in many instances, one cataract is much more advanced than that in the other eye; and, then, no such trial need be gone through.

Artificial Ripening.—In order to hasten the maturity of a cataract, puncture of its anterior capsule has been proposed and practised with success, but has not been generally adopted, from the fear that it might set up iritis, and produce increased tension from excessive swelling of the cataract. Förster[1] effects artificial ripening by performing an iridectomy, which can afterwards be utilised for the extraction. This in itself often expedites the ripening, probably by disturbing the arrangement of the lens-fibres when the aqueous humour flows off, and Förster promotes the disturbance by gently rubbing or stroking the lens through the cornea immediately after the iridectomy, with the angle of a strabismus hook. This same massage of the crystalline lens may be employed with good

[1] *Archives of Ophthalmology*, xi., p^{t}. 3, p. 349.

result after simple tapping of the aqueous humour without iridectomy. Soon after this, a rapid increase in the opacity is often noticed, so that in from four to eight weeks extraction can be undertaken. The difficulty of this rubbing or massage of the lens lies in the estimation of the pressure to be applied; for, if it be excessive, the zonula may easily be ruptured, with the result of loss of vitreous when the extraction comes to be performed. The best results are obtained in cases of cataract where there is a firm and somewhat opaque nucleus, and where a certain amount of opacity already exists in the anterior cortical substance. I occasionally employ the method, and with satisfactory results, but some operators have seen iritis follow the proceeding.

The question, whether the cataract in one eye should be extracted until both are blind, is often asked by patients. The answer is:—A patient with one mature cataract, and the other progressing towards maturity, should have the ripe cataract removed. Hypermaturity is thus avoided, and also the stage of blindness above referred to. Again, if there be a ripe cataract in one eye, and not even incipient cataract in the other, it is often advisable to operate for the purpose of increasing the binocular field of vision.

Complete Cataract of Young People.—The spontaneous occurrence of total cataract in the youthful lens is of rare occurrence, and its pathogenesis is still unknown.

Treatment.—Discission.

Diabetic Cataract.—This is a complete opacity of the crystalline lens occurring in diabetes, and due to disturbed nutrition.[1] It has been proved by experiment that cataract can be produced by injecting solutions of sugar into the blood; but analysis of the aqueous humour in diabetic patients shows, that the amount of sugar contained in it is

[1] Cataract due probably to altered nutrition has been observed in ergotism, and has been produced in animals by administration of naphthaline.—L. W.

not sufficient to account for the cataract. The cataract does not differ in appearance or consistency from other cataracts, according to the time of life of the patient.

Treatment and Prognosis.—Contrary to a very general opinion, these cases are favourable for extraction operations. I have operated on several cases of this kind, and always with success, save once, when the eye was lost by intraocular hæmorrhage, and I have also seen such cases operated on successfully by others. There is no other method of restoring sight to these patients, who often live a long time. But some ophthalmic surgeons of distinction have informed me that, occasionally, patients operated on for diabetic cataract die of coma within about a fortnight or so after the operation; and they seemed to think that this was not diabetic coma of the ordinary kind, but coma caused in some way by the nervous system being upset by the operation.

The operation of discission in these cases is apt to be followed by severe iritis.

Complete Congenital Cataract.—Children are sometimes born with crystalline lenses opaque in all their layers, while the other tissues of the eye are healthy. With congenital cataract, defects of the chorioid or retina, or congenital amblyopia without ophthalmoscopic appearances, are also sometimes present, and these are usually indicated by nystagmus.

Treatment.—Discission.

Black Cataract.—This name is sometimes given to cases of extreme sclerosis of the lens, in which it assumes a dark brown colour; but in other cases the lens is really black, the pigment being derived from the blood (hæmin, or hæmatin). An instance has recently been observed in which the lens was jet-black from this cause. The prognosis in these cases is not good, as they are often complicated with disease of the chorioid, or with hæmorrhages in the vitreous humour.

Partial Cataracts.

These are nearly all congenital.

Central Lental Cataract.—This is a congenital, and usually non-progressive, form. It is an opacity of the central, or oldest, lens-fibres, while the peripheral layers remain clear.

Treatment.—Discission, or iridectomy.

Zonular, or Lamellar, Cataract.—This is congenital, or forms in early infancy, and is the most common kind of cataract in children. It usually is present in both eyes, but it has been seen in one eye only. In it the very centre of the lens is clear (Fig. 112), while around this is a cataractous layer or zone, and outside that again the peripheral layers are transparent. Most of these cases are non-progressive, but, occasionally, the whole lens does become opaque, and usually then there have been previously some slight opacities in the otherwise clear cortical layers.

FIG. 112.

With oblique illumination the cortical layers of the lens are seen to be clear, while towards the centre of the lens a uniform grey circular opacity will be observed. The diameter of this opacity may be small, perhaps not more than 3 mm. or 4 mm., or it may extend very nearly to the equator of the lens. If the pupil be dilated, and the lens examined with transmitted light, the cataractous portion will be seen as a more or less dark disc in the centre of the lens, while all around it is seen the red light reflected from the fundus oculi. The margin of the disc is either of the same degree of darkness as its centre, or but little darker; and this point serves to distinguish this form of cataract, from one in which the whole centre of the lens is opaque. In the latter case, it is evident that the centre of the opacity must be darker than its margin. In many cases, small radial opacities are seen round the equator of the lens, passing from the anterior to the posterior surface, and their concavity embracing the circumference of the central opacity.

It is probable that lamellar cataract is due to some transient disturbance of nutrition, occurring at the time the affected layers of the lens are being laid down. But against this view is the fact, that one half of the lens only may present the appearance of zonular cataract.[1] The subjects of it are usually rickety, as shown by the irregular, and imperfect, development of the teeth, and in rachitic alterations in the bones of the skull. Convulsions during infancy, in these patients, are common.

The Treatment of central lental cataract and of zonular cataract is similar, and consists in either discission or iridectomy. The latter is very decidedly to be preferred in those cases in which the central opacity is so small that, on dilatation of the pupil, the acuteness of vision, with the aid of a stenopæic slit, is increased in a satisfactory degree. When the improvement is but slight, the breaking up of the lens with a needle is indicated. The advantage of iridectomy over discission, when the former can be adopted, is, that no spectacles are afterwards required, and that the power of accommodation is retained.

Congenital cataracts may be needled any time after dentition is completed.

Anterior Polar, or **Pyramidal, Cataract** may be either congenital or acquired. In the former case it must be referred to some inflammatory disturbance occurring about the third period of development of the lens. In both cases the mode of origin of the opacity is the same, whether it be punctiform, flakelike, or pyramidal; namely, by contact of the lens with an inflamed cornea. In fœtal life, this may occur without any perforation of the cornea, as there is then no anterior chamber. After birth, a perforating ulcer of the cornea is a necessary precursor of it, but the ulcer need not be central (p. 118). This contact with an inflamed and ulcerating cornea may lead to subcapsular cell-proliferation, at that portion of the capsule

[1] *Centralblat f. prakt. Augenheilkunde*, 1894, p. 33.

which is exposed in the pupillary area. No *Treatment* is required, as vision is not affected.

Fusiform, or **Spindle-Shaped, Cataract** is also congenital, and is rare. It consists in an axial opacity extending from pole to pole, and may be combined with central or lamellar opacity.

The foregoing forms of cataract, with the exception, perhaps, of the pyramidal and genuine black cataract, are primary; that is to say, they are not dependent on, or the result of, disease in other parts of the eye.

But, the fact has to be recognised, that some diseased states of the eye give rise to

Secondary Cataract.

Of this a partial kind is

Posterior Polar Cataract.—This form is seen, with transmitted light, as a star-shaped or rose-shaped opacity in the most posterior layers of the posterior cortical substance, its centre corresponding with the posterior pole of the eye.

Posterior polar cataract is usually found in eyes which are the subjects of disseminated chorioiditis, retinitis pigmentosa, or diseased vitreous humour. It sometimes progresses, and becomes a complete cataract; and then the prognosis for sight after extraction is not very good, owing to the disease which is present in the deep parts of the eye.

The additional disturbance of sight caused by the presence of posterior polar cataract depends, a good deal, upon its density.

Total Secondary Cataract often ensues upon contact of the lens with inflammatory products in the eye, *e.g.*, where false membranes have been produced by inflammation in the uveal tract. It is sometimes then called *Cataracta Accreta*, when the iris or ciliary processes are adherent to it. Cataract is also caused by detachment of the retina, intraocular tumour, absolute glaucoma, dislocation of the lens, etc. The

reason of this is, that the lens, in these cases, imbibes abnormal nutrient fluid from the diseased tissues with which it is in contact.

Such cataracts often undergo a further degeneration, and become calcareous. Calcareous cataracts are easily recognised by their densely white, or yellowish white, appearance; and almost always indicate deep-seated disease in the eye, even when the functions, so far as they can be tested, are fairly good.

These secondary cataracts rarely come within the range of *Treatment*, as the diseases which give rise to them are usually destructive of sight. When, occasionally, they can be dealt with, they should be extracted.

The term "secondary cataract" is also used in cases in which, after a cataract extraction, the capsule of the crystalline lens, which is left behind, presents an obstacle to good sight. This will be referred to again further on, and is not to be classed with the conditions dealt with in this paragraph.

Capsular Cataract

means an opacity of the anterior capsule, or of the capsular epithelium. It is usually confined to the centre, or anterior pole, and is most frequently seen in over-ripe senile cataracts, and in secondary cataracts.

Traumatic Cataract.

Every injury which opens the capsule of the lens is liable to cause cataract, by reason of the admission of some of the surrounding fluids to the lenticular substance.

Perforating injuries with sharp instruments, or the entrance of small foreign bodies—in both cases, as a rule, through the cornea—are the most common injuries that produce traumatic cataract. But blows upon the eye, without any perforating wound, also, although rarely, produce cataract. In these latter cases there is a rupture of the capsule, either

at the equator of the lens, or on its posterior or anterior surface.

Within a few hours after a perforating injury of the anterior capsule, the lens substance in the immediate neighbourhood of the opening becomes opaque, swells, and protrudes, as a grey, fluffy-looking mass, through the opening and into the anterior chamber, where it breaks up, dissolves, and becomes absorbed. It is immediately followed by other portions of the lens which have become cataractous, until, gradually, the whole lens may have disappeared, and the pupil again become black. Marcus Gunn suggests[1] that the explanation of the solution of the cataract in the anterior chamber consists in the fact, that globulin is normally soluble in a weak solution of chloride of sodium, such as we have in the fluid of the anterior chamber. The absorption of a traumatic cataract takes many weeks, and ultimately the eye sees well, if a suitable convex lens be put before it.

But the course of events just sketched is the most favourable one, and is hardly likely to take place in a case which is wholly untreated. In the first place, the swelling of the lens—especially if it be rapid, in consequence of a wide opening in the capsule—is liable to irritate the iris, and to cause iritis; or, to push the periphery of the iris forwards against the periphery of the cornea, block the angle of the anterior chamber, and cause secondary glaucoma (p. 337).

Moreover, violent plastic or purulent uveitis may come on, as the consequence of the introduction of infective matter on the perforating object, or foreign body, which causes the cataract. Where this occurs, the case enters into the category of diseases of the uveal tract; and the cataract, as such, becomes a minor consideration.

Again, we sometimes meet with traumatic cataracts which do not undergo any absorption process, but simply remain

[1] *Ophthalmic Review*, 1889, p, 235.

stationary; or, in the course of years, undergo secondary changes similar to those which occur in senile cataract. In these instances the trauma is usually a blow on the eye, not a perforating injury; and it is believed that the rupture of the capsule closes soon after the blow, and, hence, no lens matter can escape into the anterior chamber; and, in addition to this, the rupture in many of these cases is probably at the equator of the lens, where the aqueous would not readily get access to the lenticular substance.

Where the cataract is produced by a small foreign body flying through the cornea and into the lens, it is a matter of importance, for the prognosis, to decide whether the foreign body be in the lens, or have passed through it into the deeper parts of the eye. In the former case, we may hope to extract it with the cataractous lens; while, in the latter case, we must fear that it will set up dangerous inflammatory reaction. In such cases the lens should be well searched with focal illumination, and the transmitted light may also be of use; but it must be remembered that in these traumatic cataracts there are often glittering sectors in their deep parts, which may readily be mistaken for a metallic foreign body.

Treatment.—The pupil should be kept dilated with atropine, in order to draw the iris out of the way of the swelling lens matter; and nothing more is necessary if complications do not arise. But should iritis, or high tension, come on—and the surgeon must constantly test the tension—it is important, without further delay, to extract as much as possible of the cataract. This may be done, either without an iridectomy, through a linear incision some 10 mm. long in the upper third of the cornea, or with an iridectomy, through an incision in the upper margin of the cornea.

If a foreign body be present in the lens, extraction of the latter with the foreign body should invariably be undertaken.

Where violent purulent or plastic uveitis is set up by the trauma, the treatment resolves itself into that for these inflammations.

Operations for Cataract.

With regard to the *State of Health of the Patient* about to be operated on, it is desirable, as in every operation, that it should be good. Still, we have so often in these cases to deal with very old people, that we cannot in every instance require sound organs, and a robust constitution; and, as a matter of experience, I have not found serious disease of the heart, lungs, and liver, even when they all existed in the same individual, any impediment to a successful operation. Diabetes is no absolute contra-indication, and even in the presence of Bright's disease I have operated successfully. Very advanced years form no obstacle. I have frequently operated for cataract on persons over eighty years of age, and always with success.

The State of the Eye itself should be carefully investigated prior to proposing or undertaking an operation for cataract, and is a much more important matter than the general health. Above all things, it is to be determined whether there be intraocular complications, which would neutralise the result of a successful operation, such as detachment of the retina, disseminated chorioiditis, atrophy of the optic nerve, etc. The examination of the eye in question before the lens has become opaque, if the surgeon have had that opportunity, will be the most reliable basis upon which to go; and, for this reason, a careful note should be taken of the condition of the fundus in each case of incipient cataract. The examination of the fundus of the other eye, if its lens be clear, may help in determining the point; in so far as those intraocular diseases are concerned which are apt to be binocular. Again, the condition of the anterior capsule of the lens should be observed, for a defined glistening white square patch, about

2 mm. broad, situated in the centre of the capsule, tells the tale of intraocular mischief. It cannot be confounded with the more diffused striated and punctated capsular alterations due to over-ripeness.

Finally, the functions of the eye should be examined. With an uncomplicated cataract of the most opaque kind good perception of light should be present, so that the light, say, of a candle some two metres distant may be distinguished. In less dense cataracts fingers may be counted at 1 m. or 1·5 m. when full maturity has been attained. The field of vision must be examined by means of the "projection of light," *i.e.*, a lighted candle held in different parts of the field should be recognised by the patient, who is required to point his finger in the direction of the light, as it is moved rapidly from one part of the field to another. This examination can also be made by means of the light reflected from the ophthalmoscope mirror. If the patient fail to project the light in any direction, a diseased condition in the corresponding part of the retina may be suspected. In cases of very old uncomplicated cataract, the patients often project the light in one direction, no matter where it may come from. A certain degree of intelligence on the part of the patient is required for this test.

By the foregoing means most intraocular complications of a serious nature can be detected, but there is at least one against which I know of no safeguard, namely, a small circumscribed spot of chorioido-retinal degeneration at the macula lutea (central senile chorioiditis). After removal of a cataract from an eye affected in this way, the patient's vision is so much improved as to enable him to go about alone, but reading will still remain an impossibility for him.

The Cornea should be Examined.—Such corneal opacities as would seriously compromise vision may contraindicate the operation; but slighter opacities, discernible only with oblique illumination, would merely diminish the future acuteness of

vision, and would require a corresponding prognosis to be given before operation.

The Condition of the Appendages of the Eye, too, must be examined. Should there be any conjunctivitis, blepharitis, or dacryocystitis, it ought to be cured, or alleviated, before the operation is undertaken. Very successful operations may be performed in the presence of chronic dacryocystitis, or granular ophthalmia; but it is in all respects wiser to reduce their activity to a minimum. Some surgeons, in cases of dacryocystitis, temporarily obliterate the lacrimal puncta by introducing a red-hot needle.

Extraction of Cataract.

Linear Extraction.—The extraction through a linear incision in the cornea is applicable only to soft, or fluid, cataracts, in persons up to the age of twenty-five. The instruments

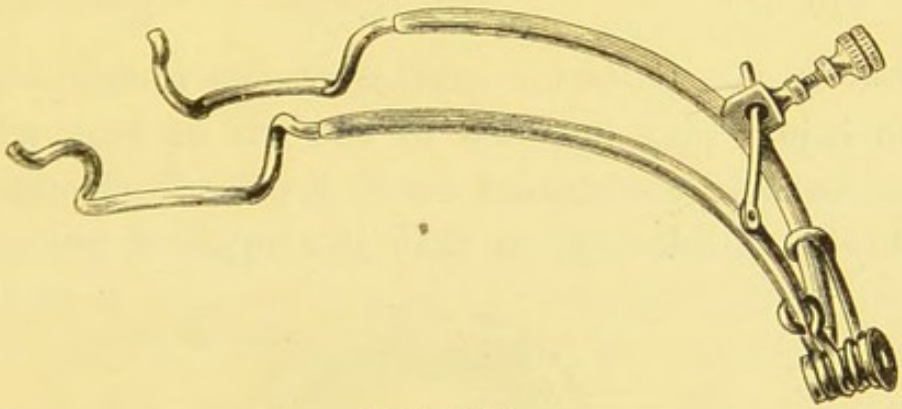


Fig. 113.

required are:—A spring lid elevator (Fig. 113), a fixation forceps, a wide lance-shaped iridectomy knife (Fig. 114), a cystotome (Fig. 115), and a Critchett's vectis (Fig. 116).

The speculum having been applied, a fold of conjunctiva close to the margin of the cornea, and at the inner end of the horizontal meridian of the latter, is seized (Fig. 117) with the fixation forceps, and the eye fixed by it throughout the operation. The point of the knife is now entered into the cornea in its horizonal meridian, about 4 mm. from

its outer margin, and is passed into the anterior chamber. The blade of the knife is then laid in a plane parallel to that of the iris, and pushed on until the corneal incision has attained a length of 6 or 7 mm. The point of the knife being now

FIG. 114.

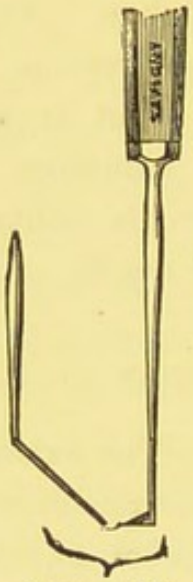


FIG. 115.

FIG. 116.

laid close to the posterior surface of the cornea—in order that no injury may be done to the iris or lens, when the aqueous humour commences to flow off—the instrument is very slowly withdrawn, so that the aqueous humour may

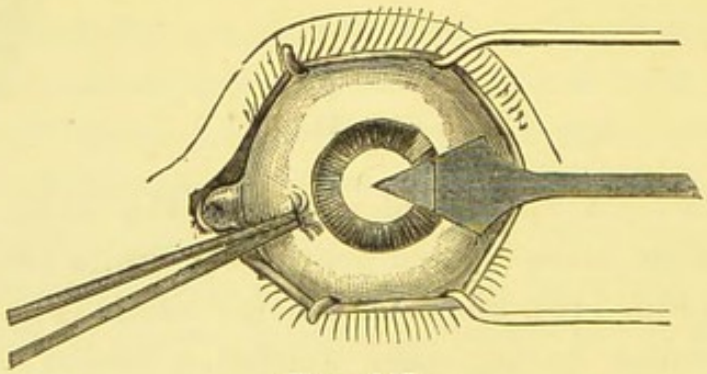


FIG. 117.

come away gradually, without causing prolapse of the iris. In withdrawing the knife it is well to enlarge the inner aspect of one or other end of the wound, by a suitable motion of the instrument in that direction.

The knife being now put aside, the cystotome is passed into the anterior chamber (Fig. 118) as far as the opposite pupillary margin, care being taken, by keeping the sharp point of the instrument directed either up or down, not to entangle it in the wound, or in the iris. The point is now turned directly on the anterior capsule, and, by withdrawing the cystotome towards the corneal incision, an opening in the capsule of the width of the pupil is produced. The cystotome is then removed from the anterior chamber, with the same precautions as on its entrance.

The edge of the vectis is then placed on the outer lip of

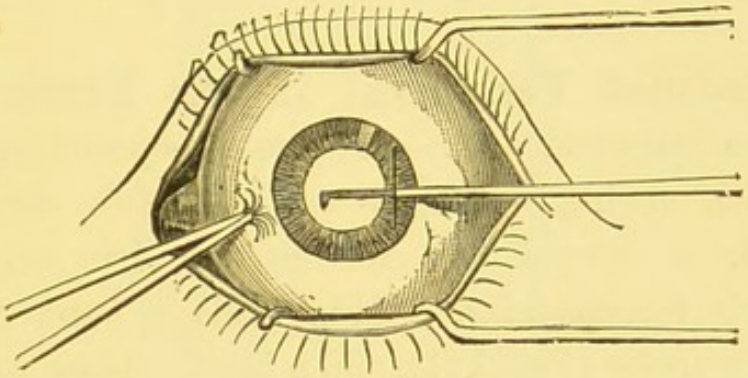


FIG. 118.

the corneal incision, and the latter is made to gape somewhat, gentle pressure being at the same time applied to the inner aspect of the eye by the fixation forceps, and in this way the lens is evacuated. When the pupil has become quite black the operation is concluded. If pressure does not at first clear the pupil completely, the speculum should be removed, the eyelids closed, a compress applied, and a few minutes allowed to elapse, in order that some aqueous humour may be secreted. A renewal of the efforts to clear the pupil will probably now be successful, or, if not, another pause may be made, and then fresh attempts employed until the pupil is quite clear. It is unwise to insert the vectis into the eye to withdraw the fragments; and, if some of these should be left behind, no ill results need necessarily follow, although iritis is more apt to supervene than if the lens be thoroughly

evacuated. Fragments left behind become absorbed. If there be a prolapse of the iris which cannot be reposed, it must be abscised.

Von Graefe, Waldau (Schuft), and Critchett endeavoured, by increasing the size of the incision, placing it in the corneo-sclerotic margin, performing an iridectomy, and introducing a vectis for delivery of the cataract, to make the linear extraction applicable to senile cataracts. The successes derived from these modifications were not, however, more satisfactory than those obtained from the old Flap Operation. But these experiments led von Graefe to the operation, a modification of which is now very generally employed. He called his operation

The Modified Peripheral Linear Extraction.—The instruments required are:—A wire lid-speculum, a fixation forceps with spring catch, a von Graefe's cataract knife (Fig. 119), a curved iris forceps, an iris scissors, or a de Wecker's forceps-scissors (Fig. 120), a bent cystotome, a hard rubber spoon (Fig. 121), and a hard rubber, tortoise-shell, or silver spatula (Fig. 122).

Before proceeding to operate, the eye is now-a-days thoroughly cocainised by the instillation of about three drops of a 2 per cent. solution of hydrochlorate of cocaine, at intervals of two or three minutes. Previously to the introduction of cocaine, general anæsthesia with ether or chloroform was commonly employed in England. I never used it.

Antiseptic Measures, similar to those used for the Three Millimetre Flap Operation (*vide infra*), are to be carefully attended to.

The Operation.—The speculum having been applied, the eye is steadied by seizing a fold of conjunctiva with its sub-conjunctival tissue, close to the lower margin of the cornea, and in a prolongation of the vertical meridian of the latter. The eye is now drawn gently downwards, the patient assisting in the motion. The point of the Graefe's knife, its cutting

FIG. 119.

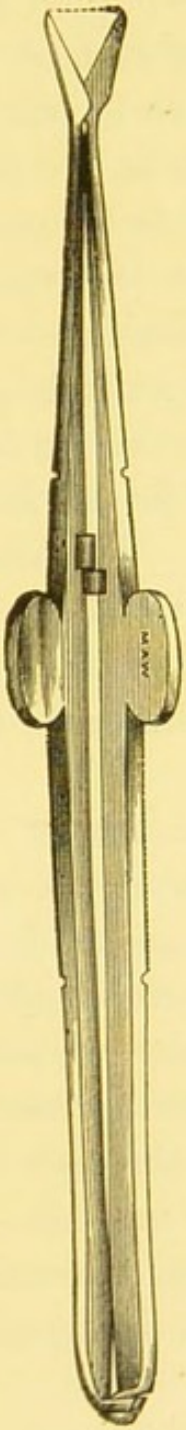


FIG. 120.

FIG. 121.

FIG. 122.

edge being directed upwards, is then entered into the corneo-

sclerotic margin at a point (*A* in Fig. 123) about 1·5 m. from the outer and upper corneal margin, and 2 mm. below the level of the tangent which would pass through the highest point of the corneal margin. The blade is held in a plane parallel to that of the iris, and is pushed on into the anterior chamber until its point reaches the point *C*, some 7 or 8 mm. of the blade being now in the anterior chamber. The handle of the knife is then lowered, so that the point of the blade is brought up to *B*, where it is made to pass out through the corneo-sclerotic margin, this counterpuncture corresponding in position, with reference to the corneal margin, to the point of entrance *A*. The edge of the knife is now turned slightly forwards, and, by one or two sawing motions, the incision *A B* is completed in the corneo-sclerotic margin. The blade still lies under the conjunctiva, which is divided, the edge of the instrument being turned more forwards, or even somewhat downwards, as it is not desirable to have too large a conjunctival flap.

FIG. 123.

The advantage of this incision lies in its peripheral position, which is almost in the plane of the crystalline lens, and consequently enables the cataract to be delivered without revolution on its axis. At a later period von Graefe altered the incision, so that, puncture and counterpuncture lying as described, the centre of the incision passed through the apex of the clear cornea, instead of through the corneo-sclerotic margin. This, by making the incision more nearly a segment of a greater circle of a sphere, made it as linear as possible, and consequently, in his opinion, its margins adapted themselves more readily.

The next step in the operation is an iridectomy, a portion of iris corresponding to the whole length of the wound, or nearly as much, being excised. This iridectomy is necessary, or advisable, chiefly because of the peripheral position of the wound, which would render prolapse of the iris very liable to

occur, but it also facilitates the delivery of the lens and cortical masses. The subsequent stages—capsulotomy and delivery of the lens—are similar in their details to those in the Three Millimetre Flap Operation, to be presently described.

It was found that the advantages of the position and form of the incision in this procedure were largely counterbalanced by the danger of prolapse of the vitreous, the difficulty of proper reposition of the angles of the coloboma, and the liability to cyclitis, all entailed by the peripheral incision, and, consequently, this incision has been abandoned by nearly all operators.

Out of this method grew that one which is known as **The Three Millimetre Flap Operation,** first proposed by de Wecker. I shall describe the operation as I am in the habit of performing it; and I may here say that, for success in the cataract operation, it is necessary not only to select the method which seems the most rational, but also to devote the utmost attention to a series of minute details in its performance.

Preparation of the Patient.—A gentle purgative is given the day before the operation, so that the bowels need not be disturbed for two days after the operation. In the case of hospital patients, the face is washed with hot water and soap shortly before the operation.

Preparation of the Eye.—Half an hour before the operation, a drop of a 2 per cent. solution of sulphate of eserine (made with a 1 in 5000 solution of corrosive sublimate) is dropped into the eye, and this is repeated a quarter of an hour later. Just before the operation, at intervals of two minutes, three drops of a 2 per cent. solution (made with corrosive sublimate solution) of muriate of cocaine are dropped into the eye. Finally, the lids having been everted, the conjunctival sac is washed out with solution of corrosive sublimate, 1 in 10,000, particular attention being paid to the fornix of each lid, and

to the inner and outer canthus. Then the skin of the eyelids, and immediate surroundings of the eye, are freely washed with the same solution.

Preparation of the Instruments.—The instruments required are the same as those for the Modified Linear Extraction. Immediately before the operation they are sterilised by boiling; out of the boiling water they are plunged for a moment into absolute alcohol, and then laid in a bath of a 1 in 2300 solution of hydro-naphthol, until required for use.

During the Progress of the Operation small bits of lint, wet with the 1 in 10,000 sublimate lotion, are employed to wipe away coagula, cortical masses, etc., and are not employed a second time. An assistant should place the instruments in the surgeon's hand in their turn, and take out of his hand those he has used, in such a manner as to render it unnecessary for him to look away, even for a moment, from the field of operation.

The Operation.—A spring wire lid-speculum is applied. The eye is fixed with a catch fixation-forceps by a fold of conjunctiva and subconjunctival tissue, below the vertical meridian of the cornea, or a little to one side of this line (Fig. 124).

The point of the knife is entered just in the margin of the clear cornea, at the outer extremity of a horizontal line which would pass 3 mm. below the summit of the cornea. This line is easily found by placing the knife, which is about 2 mm. broad, horizontally across the cornea, so that a margin of clear corneal tissue 1 m. broad may remain exposed between the knife and the summit of the cornea. The knife is then passed cautiously through the anterior chamber, and the counterpuncture made in the corneal margin at the inner extremity of the horizontal line described, and the incision finished in the corneal margin by a few slow to-and-fro motions of the knife.

Owing to the action of the eserine the iris does not pro-

lapse. The incision, between puncture and counterpuncture, lies in the clear cornea at its very margin, as represented by the dotted line in Fig. 124. This incision is no longer linear, but slightly curved. It is found, however, to adapt itself readily, and, being less peripheral than the true von Graefe incision, the objections to the latter are obviated.

The Second Stage of the Operation consists in an Iridectomy. The fixation of the eye having been given over to the assistant, the iridectomy is performed by passing a curved iris forceps into the anterior chamber, seizing the smallest possible portion of the sphincter of the iris at a point corresponding to the centre of the incision, drawing it out, and with the forceps-scissors excising a very small central bit of iris. This is done, either by making two snips in the iris, one at either side of and close to the forceps, each of them reaching to the periphery of the iris, and then a third cut which joins these two at the base; or, the forceps-scissors being approached from over the cornea, the coloboma may be formed with one snip of the instrument, and, if care be taken to keep the blades close to the forceps, a narrow, neat coloboma may thus be obtained. It is unnecessary to excise a large portion of iris, although in von Graefe's original operation a portion corresponding to the entire length of the wound used to be taken away. A small coloboma, say of 2 mm. to 3 mm. in width, as in Fig. 104, is sufficient to allow of an easy delivery of the lens, by doing away with the resistance of the sphincter iridis, and its advantages over a wide iridectomy, from an æsthetic point of view, are obvious. It is always, therefore, my object to obtain the smallest possible coloboma. The procuring of a neat coloboma is much facilitated if, prior to the operation, the pupil has been contracted (see Fig. 124) by the instillation of one or two drops of solution of sulphate of eserine, as above recommended.

The Third Stage of the Operation is the Capsulotomy. The operator takes the fixation forceps from his assistant,

who then raises the speculum and eyelids slightly off the globe, in order that no pressure may be exerted on the latter during the remainder of the operation. The surgeon, passing the cystotome into the anterior chamber, divides the anterior capsule of the lens by two incisions, each from the lower pupillary margin upwards, one directed outwards, the other inwards, as far as the anterior surface of the lens can be seen, while finally a third incision is made along the upper periphery of the lens. An extensive opening in the capsule is of great importance, as otherwise difficulty in delivery of the lens may be experienced, and because a small opening renders the occurrence of secondary cataract more likely. In dividing the capsule it is important not to dig into the lens, as this, in the case of a hard cataract, is apt to dislocate it. A rather oblique application of the cystotome to the capsule is, for this reason, the best.

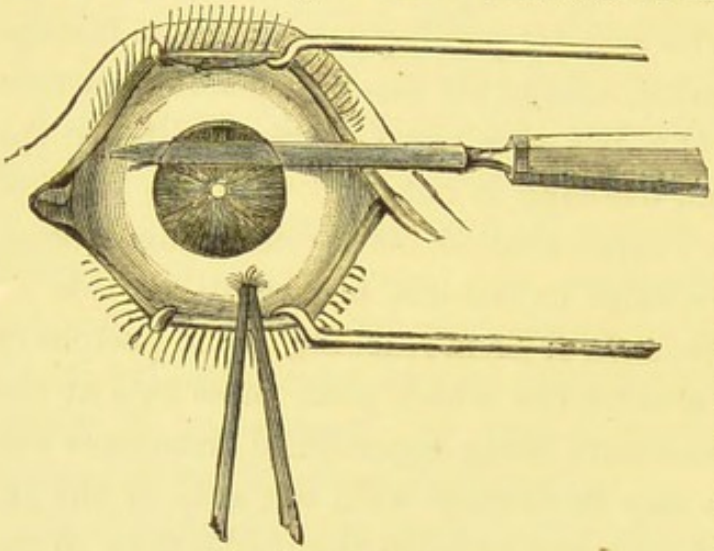


FIG. 124.

The cystotome often drags a tag of the capsule into the corneal wound, where it lies until the end of the operation, and where, owing to its transparency, it may easily pass unnoticed. Such a tag acts as a foreign body, and may subsequently form the starting-point of troublesome complications.

Capsule forceps have been invented for the purpose of taking away a large portion of the anterior capsule, but this does not altogether obviate the danger of capsule in the wound, nor does it do away with the likelihood of secondary cataract. I have no objection to the method, but it does

not seem to have any advantages over that just described, in cases where the capsule is not thickened. But, when the anterior capsule is thickened, it is always desirable to tear away a central portion of it with forceps.

Gayet of Lyons,[1] and Knapp of New York,[2] have proposed a method of opening the capsule termed peripheral division—*i.e.*, they make only one opening in the capsule at the upper periphery of the lens with a very sharp "needle cystotome," which is passed along the whole length of the corneal section, a wide iridectomy having been made for this purpose. The chief advantages claimed for this method are :—Safety from a tag of capsule in the wound, and safety from iritis caused by irritation from particles of lenticular substance left behind after delivery of the lens. On the other hand, it has the disadvantages of the wide iridectomy, and of the secondary operation on the capsule, which is necessary in a large proportion of the cases.

The Fourth Stage is the Delivery of the Cataract. The eye is drawn gently downwards—the patient being called on to assist in this motion by looking towards his feet—the convex edge of the hard rubber spoon is placed just below the lower edge of the cornea, and gentle pressure is exercised on this place, the pressure to be gradually increased until the upper margin of the lens presents itself in the wound, when, the same pressure being maintained, the spoon is advanced over the cornea in an upward direction, pushing the lens before it and out through the wound. As soon as the greatest diameter of the lens has passed the wound, the pressure of the spoon should at once be diminished, lest rupture of the zonula be caused. The fixation-forceps and speculum are now removed from the eye, and a cold compress with sublimate lotion is laid on the closed lids.

The Fifth Stage consists in Freeing the Pupil of any

[1] *Gazette Hebdomadaire*, 1875, No. 35.

[2] *Archives of Ophthalmology and Otology*, vol. vi., p. 545.

Cortical Masses which may have been rubbed off in the passage of the lens through the wound, and in what is called the "Toilette" of the Wound.

The presence of cortical remains is recognised by the pupil not having become quite black; or, by the vision not being such as it ought to be (fingers counted at several feet); or, by inspection of the cataract just removed showing that some portions of it are left behind. The use also of the oblique illumination for the detection of cortical fragments is very advantageous. If any fragments be present, the cold antiseptic compress having lain on the eye for a few minutes to enable some aqueous humour to collect, the operator, facing the patient, raises the upper lid with the thumb of one hand, while, with the first and second fingers of the other laid on the lower lid, light rotatory motions are made with this lid over the cornea so as to collect the masses towards the pupil, and then a few rapid light motions upwards, with the margin of the lid, drive these masses towards, and out of, the wound.

Care and delicacy of touch are required in order to perform this lid-manœuvre successfully, without rupturing the hyaloid by undue pressure.

With an iris-forceps the blood-clots which may adhere to the wound are now removed.

I then invariably employ the following means, to prevent the possibility of any portion of capsule being incarcerated in the wound during healing. A bent iris-forceps is passed open between the lips of the wound, closed, and drawn gently out again. Frequently a tag of capsule will have been captured by the forceps, and is snipped off with the scissors; or, it may be, that no capsule is caught. The forceps is then similarly inserted at an adjacent part of the wound; and, in this manner, the wound is searched from end to end for capsule. In about 25 per cent. of the cases a tag of capsule is found present. I regard this measure, which I am not aware that any other surgeon has previously recom-

mended, as an important one, for I believe that it effectually removes the one serious drawback to the valuable operation under consideration.

Finally, the coloboma has to be seen to. The peripheral portions of the iris, corresponding to the ends of the wound, are apt to have become prolapsed in the course of the operation, and to have displaced the angles of the coloboma upwards. If this be not corrected, the prolapsed portions of the iris heal in the wound, and cause bulgings there later on, the pupil in the course of some months becoming drawn up towards the cicatrix. Hence, in every case, even where everything seems to be in order, it is important to pass the narrow spatula into the anterior chamber, and to gently stroke down each pillar of the coloboma as far as it can be brought. The instillation of eserine before the commencement of the operation will cause the sphincter iridis to assist in producing the desired result. All this is aptly termed the toilette of the wound.

The sight of the eye should then be tested by finger-counting, as this affords the patient satisfaction, and lends him courage for the next few days of strict quiet.

Having secured the required advantage from the effect of the eserine, a drop of atropine is put into the eye before applying the bandage, in order to do away with the myosis, which might give a tendency to iritis.

The dressing is now applied. A piece of lint, sufficiently large to extend $\frac{1}{4}$ inch beyond the orbital margin in every direction, is soaked in a solution of corrosive sublimate (1 in 5000) and laid on the closed eyelids. Pledgets of absorbent cotton wool, soaked in the same solution, are laid on this, the hollows at the inner canthus, etc., being carefully filled up; so that, when the bandage is put on, it may exert equal pressure on every part of the eye. Over all comes a layer of oiled silk protective. I apply three turns of a narrow flannel roller over the dressing and round the head, in the manner which was customary in von Graefe's clinique; but various other, and

doubtless equally good, forms of bandage are in use. The pressure of the bandage need only be sufficient to maintain the dressing firmly in its place. It is usual to keep the other eye closed by a light bandage.

I am opposed to the after-treatment of cataract operations without bandage, as advocated by some surgeons. It is by no means a new method, and I do not doubt that many cases recover under it. I do not believe, however, that, in a long series of cases, the same percentage of recoveries can be obtained by it as with the bandage.

Accidents liable to occur during the Operation.—The Wound may be Made Too Small. The delivery of the lens, consequently, may be so difficult that the margins of the wound are contused, and then suppuration may be promoted. The zonula, too, may be ruptured by the excessive pressure from efforts to force the lens out, and prolapse of the vitreous may ensue. If the directions above given be carefully attended to, the vast majority of both hard and soft cataracts may be extracted without difficulty; but should the wound be made too small, it can best be enlarged by the forceps-scissors, or a blunt-pointed knife made for the purpose. Where the presence of an unusually large hard cataract is diagnosed, it is important to make the incision larger *ab initio* by placing puncture and counterpuncture nearer to the horizontal meridian of the cornea than above directed.

Hæmorrhage into the Anterior Chamber may take place. It may be from the iris, from the corneo-sclerotic margin, or from the conjunctiva. Pressure with the spatula on the cornea, which causes the wound to gape, is often successful in clearing the chamber of blood, which might interfere with accurate division of the capsule. Yet, when this cannot be completely got rid of, the capsulotomy may be performed with the exercise of greater care. Cocaine, by its power to contract the blood-vessels, has rendered this hæmorrhage a less common complication than it used to be.

Prolapse of the Vitreous Humour. This may be due to a too peripheral position of the wound, support being thus taken away from the zonula, and the danger of its occurrence was a disadvantage of the completely corneo-sclerotic wound practised at one time by von Graefe. The Three Millimetre Flap Operation is less liable to be attended with loss of vitreous. This accident may also be caused by undue pressure made on the eyeball by the speculum, fixation forceps, or spoon, or by the under-lid during the lid-manœuvre. It may be due to defective zonula with fluid vitreous humour. If the vitreous prolaps prior to delivery of the lens, the latter falls back into the eye, and can only be delivered by at once drawing it out with a Critchett's, Taylor's, or other suitable vectis; and this may be regarded as one of the most serious accidents which can occur in the course of the operation. Loss of vitreous after delivery of the lens is less serious; indeed, a considerable portion of the vitreous may then be lost without ill result to the eye. Yet, it increases the traumatism, and renders inflammatory reaction more liable to occur. Opacities in the posterior chamber of the eye are frequently an ultimate result of loss of vitreous; but a much more serious consequence is sometimes seen in detachment of the retina.

Normal After-Progress.—Soon after the completion of a normal operation, the effect of the cocaine having passed off, some smarting commences, and continues for four or five hours. After that time the patient has no unpleasant sensation in the eye, unless it be some itching, or a slight momentary pain or sensation of a foreign body, especially when the eye is moved under the bandage. The first dressing is made in forty-eight hours, in a manner similar to that immediately after the operation, a drop of atropine being instilled, as also at each successive dressing; and the corrosive sublimate solution is used for freely washing the margins of the eyelids, some of it being allowed to trickle into the conjunctival sac. At this first dressing it is well to abstain from a minute, or

lengthened, examination of the eye ; but, if the lid be gently raised, the wound will be found closed, the cornea clear, the anterior chamber completely restored, and the pupil semi-dilated and black. The subsequent dressings are made night and morning, for the purpose of instilling atropine. On the third day after the operation the patient may be allowed to sit up, the room being kept moderately dark ; and, on the fifth or sixth day, the bandage may be left aside permanently, and dark glasses worn in its stead. In the course of a few days more, the patient, having been gradually used to more light, may be allowed out of doors. It is desirable to continue the use of atropine for about a fortnight longer, or until all abnormal vascular injection of the white of the eye has disappeared, as, until then, there is danger of iritis. (For selection of glasses in aphakia see end of this chapter.)

Irregularities in the Process of Healing.—The pain may continue longer than four or five hours, and it is then well to quiet it by a hypodermic injection of morphia in the corresponding temple. Should severe pain come on some hours later, it is apt to be due to an accumulation of tears under the eyelids, and it immediately subsides on the bandage being removed and exit given to the tears by slightly opening the eye. Antiseptic precautions are to be observed while this is being done.

Late Appearance of the Anterior Chamber. At the first dressing it will sometimes be found that there is no anterior chamber, although the appearance of the wound is quite satisfactory ; but this need occasion no alarm, as the anterior chamber is sometimes not restored for a week or more.

Striped Keratitis. At this dressing also it may be observed that there is a more or less well-marked striated cloudiness of the cornea, extending over nearly the whole of it, or occupying only a part in the immediate neighbourhood of the wound.

This opacity is, according to some, the result of injury to the endothelium of the posterior surface of the cornea during

the operation by instruments, or by the chemical action of the antiseptic lotion. Leber has shown that the entrance of even the aqueous humour, through a loss of substance in the endothelium, is sufficient to cause the fibres of the true cornea to swell and become opaque, just as the crystalline lens is acted on if its capsule be opened. The endothelium of the posterior surface of the cornea in fact it is, which protects the latter from being infiltrated by the aqueous humour.

The explanation given by Hess,[1] however, seems a very reasonable one, namely, that it is due to folding of the posterior layers of the cornea, on account of the difference in tension in the vertical and horizontal direction. His conclusions are based on microscopic examination and experiment.

This so-called striped keratitis is, for the most part, of no serious import, as it usually passes away in a few days, and leaves the cornea perfectly clear; and folding of the posterior layers would account for these cases. But now and then cases do occur, in which the process is very intense, and where a permanent white opacity remains in the cornea over the pupillary area, with consequent serious deterioration of vision. These severe cases are most apt to be caused by the introduction of the antiseptic solution into the anterior chamber; for the chemical action of the antiseptic on the corneal tissues is more damaging, and therefore the opacity it produces more permanent, than is the action of the aqueous humour. Sublimate lotion is the antiseptic which has been most often to blame, probably because it is the antiseptic in most general use. With the 1 in 5000 solution which I have until recently employed, I never had the severe form, and rarely the mild form; but then I never deliberately introduced the solution into the anterior chamber. I have had only one case of the severe form, and in it, by mistake, a sublimate lotion of 1 in 2500 was used for irrigation of the surface of the eye. As stated above, I now employ a solution

[1] *Von Graefe's Archiv*, xxxviii., 4.

of only 1 in 10,000. No doubt, in irrigation of the surface of the eye, some of the lotion used is liable to make its way into the anterior chamber. Boric acid solutions (3 per cent.) do not injure the endothelium, but I have little faith in their antiseptic properties.

Suppuration of the Wound. This is a danger which is very much rarer than it was prior to the introduction of antiseptics into surgery; indeed, it is almost banished from the cataract operation. When it occurs, it usually does so between the twelfth and thirty-sixth hour after the operation, rarely earlier or later, and is a very serious event; for, in the vast majority of cases, do the surgeon what he may, it leads to loss of the eye. Its onset is made known by severe pain of a continuous aching kind in and about the eye; and is thus easily distinguished from the slight, short, stabbing pain, with long intermissions, which some patients complain of, and which has no evil import. On removing the bandage the eye will be found full of tears, and the wound covered with a layer of muco-pus, which can be removed with the forceps in one mass, while the aqueous humour and cornea may already present some opacity. In some hours more the corneal opacity increases considerably, the iris becomes distinctly inflamed, and the pupil filled with a mass of inflammatory exudation. The inflammatory process may remain confined to the wound and iris, and when, in the course of some weeks, it entirely subsides, it leaves the pupil drawn up towards the wound, so that an appearance as in Fig. 125 is presented. Or, the inflammation may strike into the ciliary body and chorioid, and produce purulent panophthalmitis, with total destruction of the eye.

FIG. 125.

To combat Suppuration, the best method is the immediate cauterisation of the corneal wound in its whole extent with the galvano-cautery. Also, the wound may be opened up from end to end with a spatula, the aqueous humour evacuated, and

the anterior chamber washed out with injections of corrosive sublimate solution, while the conjunctival sac is irrigated with the same solution. If necessary, these measures are to be repeated at intervals of eight or ten hours. Subconjunctival or intra-ocular injections of corrosive sublimate may also be tried (see p. 160).

Iritis. Apart from the iritis which occurs in connection with suppuration of the wound, this complication is most usually due to irritation from masses of cortical lens-substance left behind. It may also be due to dragging from a tag of the iris being left in the wound; or, to too early exposure to the daylight. Cortical masses do not usually give rise to it for some days after the operation. It is ushered in with the usual symptoms of pain, and is generally of the plastic variety. If it extend to the ciliary body, sympathetic ophthalmitis may result. Its *treatment* consists in strict confinement to a dark room, atropine, warm fomentations, leeching, and, internally, salycilate of soda is most useful. In these cases vision is liable to be damaged by pupillary exudation, which remains as a permanent obstruction to vision.

Cystoid Cicatrix. After convalescence, all the foregoing dangers having been escaped, the cicatrix in the corneal margin sometimes bulges and becomes semi-transparent, presenting the appearance of a vesicle, and may attain a large size. The extremities of the late incision are the most common positions for this condition, but it may occupy the entire length of the cicatrix. It does not generally come on for some weeks, or more, after the operation. In some cases it is caused by a tag of iris which is incarcerated in the wound, but in other cases probably by a bit of capsule, which has similarly healed in the wound. Irregularity in curvature of the cornea, and consequent irregular astigmatism, are the least of its evil consequences. If the condition be caused by incarceration of iris, the pupil will be gradually drawn close to the upper corneo-sclerotic margin; while, if it be caused by a portion of

capsule, irido-cyclitis may be produced. Whether the iris or the capsule be the cause, these eyes are always exposed to the danger of a sudden onset of purulent irido-chorioiditis (see p. 281). All this demonstrates the immense importance of attention to those details of the operation, which are calculated to obviate incarceration of iris, or of capsule, in the cicatrix.

Cataract Extraction without Iridectomy.[1]—This method is older than the Linear, von Graefe's, or the Three Millimetre Flap Operation, and used to be known as The Flap Operation. It has been revived within recent years by some distinguished ophthalmic surgeons, chiefly in Paris, but also in Germany, America, and in England. It differs from the Three Millimetre Flap Operation, in that the incision occupies a greater extent (about one-third) of the circumference of the cornea, and that no iridectomy is made. Formerly the knife used was triangular in shape (Beer's knife), but von Graefe's cataract knife is the instrument now employed. The round pupil, and consequent somewhat prettier appearance of the eye, is the one advantage which can be claimed for this procedure over the Three Millimetre Flap Operation, as it has been above described; for, the vision with a circular pupil is not better, than where a small iridectomy has been done. As a set-off against the circular pupil, the extraction without iridectomy exposes the eye to the serious danger of prolapse of the iris into the wound. These operators make it a rule to perform an iridectomy in all cases where they cannot satisfactorily repose the iris after delivery of the lens; but, even where they can repose it well, they are not, they state, secure against the occurrence of a prolapse within the first two or three days after the operation; nor do they find that eserine, or any other means, provides the desired safeguard. It is admitted, that prolapse of the

[1] Known very generally now as The Simple Method, while the operation combined with an iridectomy is commonly termed The Combined Method.

iris takes place after a number of these operations,[1] and that there is no means of foretelling in what eyes it will occur. The prolapsed portion of iris heals in the wound, which then, in a few weeks, becomes more or less cystoid and bulging, causing displacement of the pupil, and irregular curvature of the cornea, with resulting deterioration of vision. Nor is this all, for such eyes are liable, weeks, months, or even years after the operation, to take on severe irido-cyclitis, ending in total loss of sight. Another disadvantage of this operation is, that removal of cortical remains cannot be so effectually performed as where a coloboma has been made.

Therefore, while admitting the charm of a circular pupil, I am of opinion, that the question is not whether the appearance of some of the eyes operated on is pleasing to us, and to others who inspect them; but, rather, what advantage the greatest number of persons operated on derive from the operation. With sentimental talk about "mutilation" of the iris I cannot pretend to sympathise. If the advocates of the method under discussion should find a means of ensuring the eye against prolapse of the iris, the operation will be placed upon a different footing; but, until then, the procedure cannot, I think, be recommended.

It is easy to understand why, in the simple extraction, prolapse of the iris with subsequent incarceration is so liable to occur, even some days after the operation, and why it is so difficult to devise a sure means for preventing the accident; as, also, how it is that even a very narrow coloboma is sufficient to protect the eye from this disaster. And yet I am inclined to think, that among those oculists who have reverted to the simple method, there are some who do not realise the *modus operandi* in either case.

[1] Even Knapp, who has had very good results, admits from 6 to 12 per cent. of prolapses. He now lets the patient go to bed without a bandage, and if no prolapse occur in from 5 to 30 minutes he applies one. Should a prolapse appear it is abscised.

Within a few hours after the operation the wound in the corneal margin most commonly closes, the aqueous humour collects, and the anterior chamber is restored. But it takes many hours more for the delicate union of the lips of the wound to become quite consolidated, and during this time it requires but little—a cough, a sneeze, a motion of the head, the necessary efforts in the use of a urinal or bed-pan, no matter how careful the nursing—to rupture the newly-formed union; and, as a matter of fact, this often does take place. The aqueous humour then flows away through the wound with a sudden gush, and, where the simple extraction has been employed, carries with it the iris. Doubtless, in this event, it is that portion of the aqueous humour which is situated behind the iris, which is chiefly concerned in the iris-prolapse; the aqueous humour in the anterior part of the anterior chamber probably flows off without influencing the position of the iris. The advocates of the simple operation endeavour to prevent secondary iris-prolapse by a spastic contraction of the pupil, produced by eserine, which is instilled at the conclusion of the operation, and, again, by some operators, a few hours afterwards. In most instances the desired end is by this means effected. But there is a considerable percentage of the cases in which the contraction of the sphincter iridis is overcome by the pressure of the aqueous humour from behind, and iris-prolapse takes place.

How, then, does the formation of a coloboma prevent prolapse of the iris when the wound bursts, as I have described? Not because the portion of iris which is liable to prolapse has been taken away. That would mean nothing less than the whole of that part of the iris which corresponds to the length of the opening in the corneal margin. But the coloboma averts secondary iris-prolapse, because it provides a gateway, a sluice, for the aqueous humour, contained in the posterior part of the anterior chamber, to escape directly

through the wound, without carrying with it the iris in its rush; and it is evident, that the narrowest coloboma which can be formed will be amply sufficient for the purpose. To my mind a narrow iridectomy here is no "mutilation of the iris," but rather a measure which rests upon a sound scientific basis, and which is calculated to ensure the safety of the eye in an important particular.

In 200 consecutive extractions [1] I have had two (1 per cent.) iris-prolapses with incarceration, as compared with 6 to 12 per cent. of iris-prolapses which the best statistics of operators by the simple method now show.

As to disfigurement of the eye, there is practically none, when the coloboma is so narrow, and is situated in the upper part of the iris. The pupil, too, is movable, almost, if not quite, as much so, I venture to say, as in most cases of simple extraction. For, it is entirely a mistake to suppose that a narrow coloboma renders the pupil immovable. Where there are no adhesions between the pupillary margin and the capsule, as frequently happens, the reaction to light is active, a drop of atropine will dilate the pupil widely, and a drop of eserine will contract it.

Mental Derangements after Cataract Extractions.—After cataract extractions, during the period of confinement to bed, passing mental disturbances are sometimes seen in old people. This usually takes the form of confusion of ideas, hallucinations, and terror. It is hard to assign a cause for it, but probably it is mainly due to the quiet and the exclusion of light, following on a period of some anxiety and excitement. A few doses of sulphonal, and permission to sit up—at least in bed—with the admission of more daylight, will be the best measures to adopt in such a case; and speedy restoration of mental equilibrium may be looked for with confidence. Care should be taken not to mistake the symptoms of atropine poisoning for this form of mental disturbance.

[1] *Trans. Ophthal. Soc. Un. King.*, 1893, p. 174.

Secondary Glaucoma after Cataract Extraction occurs now and then, by whatever method the extraction may have been performed. This is contrary to what one would have expected, in view of the diminished contents of the globe, by reason of absence of the lens, and especially in those cases where an iridectomy has been made. High tension in these instances may come on soon after recovery from the cataract operation, or after a good result has existed for many years. Treacher Collins'[1] and Natanson's[2] microscopic investigations show that in these cases either the iris, the capsule, or the hyaloid has become entangled in the wound, and it seems that this leads in some cases to closure of the filtration angle in its entire circumference; but, obviously, further information is required on this rather obscure question.

FIG. 126.

A wide iridectomy, or a sclerotomy, should be made as soon as possible after the high tension shows itself, and by this means many of these eyes may be saved. Simple division of the capsule has produced a good effect in some cases.

Discission or **Dilaceration** means the tearing of the anterior capsule of the lens with a needle, so as to give the aqueous humour access to the lenticular fibres, which causes them to swell, and gradually to become soft, and then to be absorbed. The larger the capsular opening, the more freely is the aqueous brought in contact with the lens, and the more rapid is its swelling. The rapidity of the swelling and absorption depend, also, on the consistence of the lens. The softer it is, the more rapid is the process, the completion of which may require from a few weeks to many months. It is wise to make the first discission

[1] *Trans. Ophth. Soc.*, vol. x., p. 108.

[2] *Ueber Glaucom in Aphakischen Augen*, Dorpat, 1889.

of moderate dimensions, in order to test the irritability of the eye, especially in adults.

The instruments required are a spring speculum, a fixation forceps, and a Bowman's stop-needle (Fig. 126). The shoulder on the latter instrument prevents its advance too far into the eye. The pupil is to be dilated with atropine.

The eye having been cocainised, the speculum applied, and the eye fixed close to the inner margin of the cornea, the needle is passed perpendicularly through the cornea in its lower and outer quadrant, at a point corresponding to the margin of the dilated pupil. It is then advanced upwards to the upper margin of the pupil (Fig. 127), where it is passed into the capsule, but not deeply into the lens, and a vertical incision is effected by withdrawing the instrument slightly. If an extensive opening in the capsule be wished for, a horizontal incision can be added to the vertical, by a corresponding motion of the needle. During these manœuvres, the cornea, at the point of puncture, must form the fulcrum for the motions of the instrument. The instrument is then withdrawn, and some aqueous humour escapes through the opening. Atropine is instilled, and the bandage applied. The patient is kept in bed, in a darkened room, for a day, and then the bandage may be dispensed with, and dark spectacles worn. The iris is to be kept well under the influence of atropine, until the absorption of the lens is completed. Repetition of the operation is called for, if the opening be so small as to admit of but a very slow absorption of the lens, or if, as sometimes happens, the opening should become closed up.

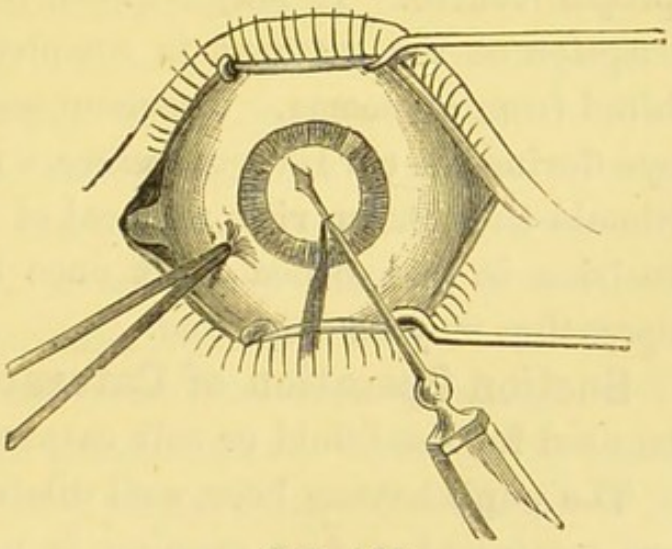


FIG. 127.

This method is applicable to all complete cataracts up to the twenty-fifth year of age, and to those lamellar cataracts in which the opacity approaches so close to the periphery of the lens, that nothing can be gained by an iridectomy. After the above age, the increasing hardness of the nucleus, and the increasing irritability of the iris, render the method unsuitable.

Discission is a safe procedure, when used with the above indications and precautions. The danger chiefly to be feared is iritis, from pressure on the iris of the swelling lens masses. When this occurs, or is threatened, removal of the cataract by a linear incision in the cornea should be at once performed. A safeguard against iritis may be had in a preliminary iridectomy (von Graefe), and it is perhaps well to do this in all cases over fifteen years of age, the discission following some weeks afterwards.

Another danger consists in glaucomatous increase of tension (secondary glaucoma), which may come on without any subjective symptoms, while the absorption of the lens runs its proper course. It may happen, in this way, that when absorption of the cataract is completed, the eye will be found blind from glaucoma. Frequent testings of the tension of the eye during the cure are, therefore, a most important precaution. Should the tension rise, removal of the lens through a linear incision in the cornea is at once indicated; or, the suction operation may be employed.

Suction Operation of Cataract.—This method can only be used for semi-fluid or soft cataracts.

The pupil having been well dilated with atropine, and the eye cocainised, a free opening is made in the capsule of the lens with a discission needle. A linear incision is then made in the cornea about half-way between its centre and its margin, and the point of a Bowman's or a Teale's syringe introduced through it, and through the opening in the capsule, into the substance of the lens. Gentle suction is then applied, and the lens substance drawn into the syringe.

The syringe should not be passed behind the iris. If it be thought that the cataract is not sufficiently soft, it is desirable to allow some time (a fortnight or so) to elapse between the discission and the suction, in order that the lens substance may undergo disintegration by the action of the aqueous humour.

Secondary Cataract and its Operation—Capsulotomy.—The term "secondary cataract," as here used (compare p. 351). usually means a closure of the opening in the anterior capsule left after the removal of a cataractous lens, with sometimes a thickening of the capsule, by which an impediment is offered to the rays of light in passing through the pupil. The thickening may have pre-existed in the capsule, or it may be due to subsequent proliferation of the epithelial cells on the inner surface of the capsule. The term is also used with reference to those cases in which no central opening has been made in the capsule (peripheral capsulotomy), and where the latter causes imperfect vision. It is also used in those cases where, after cataract extraction, an exudation in the pupil, consequent upon iritis, has occurred. Finally, and most incorrectly, it is applied to the cases which Fig. 125 represents, in which, after suppuration of the wound with irido-cyclitis, the iris is dragged upwards, and the pupil consequently obliterated.

The most simple form of secondary cataract occurs as a very fine cobweb-like membrane, extending over the whole area of the pupil, which can often only be discovered by careful examination with oblique illumination. It may not appear until some months after the extraction, and then causes the patient to complain of diminished acuteness of vision. It is a simple matter to make a rent in this delicate membrane with a discission needle.

Where there are thick opacities in the capsule, or inflammatory exudation into the pupil, with, probably, adhesions of the iris to the pupillary membrane, extraction of the latter has been proposed and practised, but is associated with so much

danger, from the unavoidable dragging on the ciliary body and iris, that the proceeding is not often employed.

Sir W. Bowman's Method with two needles is here much preferable. In it the point of a discission needle is passed through the inner quadrant of the cornea, and into the centre of the opacity (Fig. 128), and then, with the other hand, a second needle is passed through the outer quadrant of the cornea, and into the membrane, close beside the first needle.

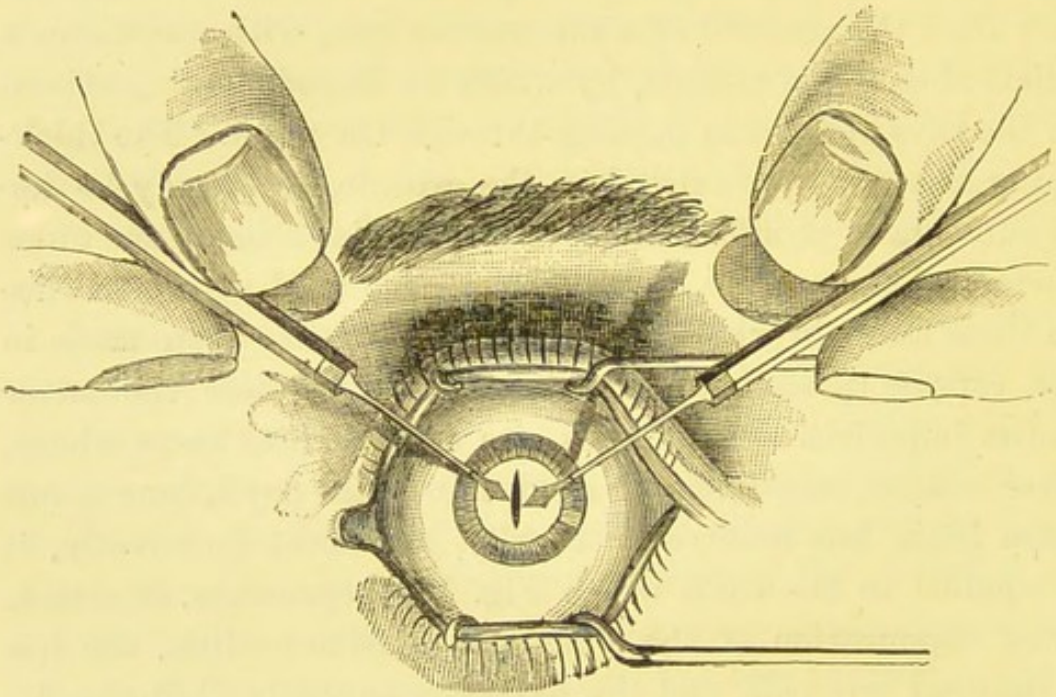


FIG. 128.

The points of the needles are now separated from each other by approximation of their handles, and in this way a hole is made in the membrane. A very small opening in the capsule, if quite clear, is sufficient to establish good vision.

Dr. Noyes's Method.[1]—A Graefe's cataract-knife is entered in the horizontal meridian of the cornea at its temporal margin, and a counter-puncture made in the same meridian at the inner corneal margin. The point of the knife is now withdrawn into the anterior chamber, and made to puncture the secondary cataract, and is then removed from the eye. Two blunt-

[1] *Diseases of the Eye* (London, 1882), p. 251.

pointed hooks are then entered into the anterior chamber, one through each corneal puncture, and the point of each passed through the opening in the membrane made with the knife. By traction on the hooks this opening is enlarged, without any dragging on the iris or ciliary body.

Iridotomy.—For the cases as in Fig. 125, where the iris forms a complete and tightly stretched curtain across the pupil, iridectomy is the operation which readily suggests itself. In very few cases, however, does it give a satisfactory result, owing to the inflammatory products which lie behind the iris, and which close up any artificial pupil by their proliferation, which is set going by the dragging of the iris with the forceps. Repeated iridectomies may finally produce a clear pupil, but iridotomy, in which there is no dragging of the iris, is a better operation in these cases.

There are several modes of performing iridotomy, that of de Wecker being the best. A vertical incision having been made in the cornea, about 3 mm. long, and the same distance removed from its inner margin, the closed blades—one of which has a sharp point—of de Wecker's forceps-scissors are passed into the anterior chamber. The blades are then opened, and the sharp point of one of them is forced through the stretched iris, and some 3 or 4 mm. behind it. By now closing the blades, the tightened iris fibres are cut across, and on their retraction a central clear pupil is formed in the iris and retro-iridic tissue.

Dislocation of the Crystalline Lens.—This may be congenital, and due to arrested development of the zonula of Zinn; or, it may be the result of disease, such, for example, as anterior sclero-chorioiditis; or, it may be caused by a blow or other trauma.

The dislocation may be partial, or complete. In the former case it is often so slight as to be discoverable only when the pupil is widely dilated, the margin of the lens becoming then visible as a black line, in some one direction, by aid of the

ophthalmoscope mirror. Or, the displacement may be so great as to bring the margin of the lens across the centre of the undilated pupil, in which case one part of the eye will be highly hypermetropic, while in another part it will be myopic. Complete dislocation may take place into the anterior chamber, into the vitreous humour, and even under the conjunctiva, if the sclerotic have been ruptured.

The symptoms in partial dislocation are those of loss of power of accommodation, and monocular double vision. Iridodonesis (*i.e.*, trembling of the iris when the eye moves) is present, as a rule, in consequence of the loss of support provided by the lens. In complete dislocation, the symptoms are those of aphakia, *i.e.*, extreme hypermetropia, and want of power of accommodation.

Treatment.—In partial dislocation it is rarely that any treatment can be of service. The prescribing of spectacles suited, so far as it is practicable, to the faulty refraction is indicated. In complete dislocation of the lens into the anterior chamber its extraction is usually required, especially if it cause symptoms of irritation. Dislocation into the vitreous humour is generally unattended by irritation; but when the latter does arise, removal of the lens by aid of a spoon, through a peripheral corneal incision, has to be attempted.

Lenticonus is a very rare congenital anomaly of the lens, in which its anterior surface, or, still more rarely, its posterior surface, is cone-shaped.

Aphakia (*a, priv.*; *φακός, a lentil, lens*), or **Absence of the Crystalline Lens.**—The condition of the emmetropic eye after the removal of a cataract is one of high hypermetropia, and the power of accommodation is wanting. Consequently, in order that the eye may have the best possible sight for distant objects, a high convex glass has to be experimentally found to suit it, and stronger lenses must be prescribed for shorter distances.

The degree of vision obtained varies considerably in different

cases; frequently V = $\frac{6}{6}$ is obtained, but V = $\frac{6}{18}$ may be regarded as a satisfactory result; and even lower degrees, which enable the patients to find their way about with comfort, are classed as successful operations. The vision often improves for some months after the operation, patients who at first had only $\frac{6}{18}$ or so advancing up to $\frac{6}{9}$ or $\frac{6}{6}$. For reading, writing, etc., at about 25 cm., a still higher convex glass must be provided. If the correcting lens for distant vision be + 10 D, its power, for vision at 25 cm., must be increased by the lens which would represent the amplitude of accommodation from infinite distance up to 25 cm. This lens is 4 D (because $\frac{100}{25} = 4$), therefore + 14 D is the lens required. With these two lenses most patients are satisfied. For distinct vision at middle distances, they learn to vary the power of the lenses by moving them a little closer to, or farther from, the eye; but, if necessary, a lens can be prescribed for distinct vision at any desired distance.

In the case of hospital patients, one is often obliged to select the + glasses in a fortnight or three weeks after the operation, but the result is more satisfactory when the selection can be postponed for six weeks or two months. Permanent wearing of the + glasses should not be permitted until all redness of the eye has passed off, and this varies in different cases. Until then, also, dark protection spectacles should be worn.

In the majority of cases, after cataract operations, the best vision is not obtained until a certain degree of astigmatism is corrected. It is caused by a flattening of the vertical meridian of the cornea, due to the incision at its upper margin, and hence is "against the rule" (see p. 43). An obliquity in the incision often produces an obliquity in the principal meridians of the astigmatism. The degree of astigmatism varies, and may be very high. Out of forty-eight cases studied by Jackson,[1] only eleven had less than 2 D of

[1] *Ophthalmic Review*, Dec. 1893, p. 349.

astigmatism. It rapidly reaches its maximum after the operation, and then gradually diminishes for weeks or months, and in some cases completely disappears; hence glasses for permanent use should not be prescribed for at least a month or two subsequent to the operation.

For an account of Erythropsia after cataract extraction see chap. xvii.

CHAPTER XIV.

DISEASES OF THE VITREOUS HUMOUR.

Purulent Inflammation of the Vitreous Humour (to which, unfortunately, the name pseudo-glioma is sometimes applied) occurs only as the result of perforating injuries, or of the lodgment of a foreign body, or as an extension of a purulent process from the chorioid (p. 281).

Ophthalmoscopically, a purulent deposit in the vitreous humour gives a yellowish reflection. It is to be distinguished from a somewhat similar appearance in glioma of the retina by the history, by its early complication with more or less severe iritis, by the very frequent retraction of the periphery of the iris, with bulging forwards of its pupillary part, and by the diminished tension of the eye, while a lobulated appearance is not so usual in it as in glioma. Again, in glioma the vitreous humour remains clear, while in this disease it is hazy.

The condition, if at first confined to the vitreous humour, soon extends to the surrounding tissues, and usually leads to panophthalmitis and complete destruction of the eye.

Inflammatory Affections of the Vitreous Humour, other than the purulent form, are, for the most part, the consequence of diseases of the chorioid, ciliary body, or retina, and display themselves as opacities of various kinds. These either are cells derived from the primarily diseased tissue, or they are secondary changes (connective tissue development), the result of this cellular invasion.

The chief *Varieties of Vitreous Humour Opacities* are—

1. The Dust-like Opacity so characteristic of syphilitic disease of the retina and chorioid. It may occupy the entire vitreous humour, but is frequently confined to the region of the ciliary body, or to that of the posterior layers of the vitreous humour. 2. Flakes and Threads. These occur with chronic affections of the chorioid or ciliary body, and may be the result also of hæmorrhages into the vitreous humour. They invade every portion of the humour. 3. Membraneous Opacities, which are rare, and are probably the result either of extensive hæmorrhagic extravasations or of chorioidal exudations.

Hæmorrhages into the vitreous humour are not uncommon, and are the result of certain diseases of the retina and chorioid, which are accompanied by hæmorrhages in those membranes. They are common in old people, but very large hæmorrhages also occur in young adults (see Apoplexy of the Retina). They are also caused by blows on the eye, which rupture the chorioidal or retinal vessels. Most of the alterations occurring in the vitreous humour are attended with, or give rise to, fluidity of it, and may lead to detachment of the retina.

The Diagnosis of opacities in the vitreous humour is made with the ophthalmoscope mirror and a not very bright light, or with the plane mirror. If a very bright light and a concave mirror be employed, the finer opacities will not be readily seen. The pupil being illuminated, the patient is directed to look rapidly in different directions, when the opacities will be seen to float across the area of the pupil, as they are thrown from one side of the eye to the other.

Opacities in the vitreous can be distinguished from those in the lens, by the fact that the latter are fixed, and are arranged mostly in a radiating manner. Opacities which lie behind the centre of curvature of the cornea, as examined with the ophthalmoscope mirror, seem to move in the opposite direction, when the patient moves his eye ; while those which lie in front of that point move in the same direction as

the eye. Therefore, opacities in the lens and anterior part of the vitreous humour, about 0·6 mm. behind the lens,[1] will move in the same direction.

Another, and very fine, method for the detection of delicate opacities in the vitreous, is by placing a high + lens, say + 10 D, behind the ophthalmoscope mirror, and then going close to the eye, as in the examination of the upright image. Minute opacities will then be seen as black dots floating in the vitreous humour.

When the vitreous humour is full of blood, no red reflex can be obtained with the ophthalmoscope, and the pupil looks quite black. By focal illumination we can observe, in this case, that the lens is perfectly clear, and sometimes the red colour of the extravasated blood can be seen behind it.

The ophthalmoscope does not always detect changes in the chorioid or retina when there are opacities in the vitreous; and, in many such cases, we are led to the belief that the diseased changes in the chorioid or retina are too fine to be seen with the ophthalmoscope, or that they are situated in the region of the ciliary body which is out of view.

Vision is affected by opacities in the vitreous humour in proportion to their density, and to the extent to which the vitreous humour is occupied by them. The patients often observe them as floating positive scotomata in their field of vision. These "entoptic appearances" are caused by the shadows of the opacities thrown on the retina.

The Prognosis depends on the cause of the opacities. Small hæmorrhagic extravasations in young people are readily absorbed. The dust-like opacity of specific retinitis is also favourable for absorption, while extensive hæmorrhages in older people, and the "flake and thread" opacities, frequently

[1] Radius of curvature of cornea, 7·829 mm. Distance from anterior surface of cornea to posterior surface of lens, 7·2 mm. (Landolt and Wecker, *Traité* T., iii., p. 113.)

remain as permanent obstructions. Moreover, by shrinking, many of the more organised opacities give rise to detachment of the retina from the chorioid, and consequent blindness.

Treatment consists, above all, in that for the exciting cause. Besides this, Heurteloup's artificial leech, or dry cupping on the temple, is most useful; and in many cases, soon after the application, a marked clearing up of the vitreous is apparent. Pilocarpine hypodermically is worthy of trial. In one case von Graefe operated on membraneous opacities by tearing them with a needle, and with a successful result.

Mouches Volantes, Muscæ Volitantes, and **Myodesopsia** (*μυῖα, a fly*; *ὄψις, seeing*) are terms applied to the motes which people frequently see floating before their eyes, but which do not interfere with the acuteness of vision, nor can the ophthalmoscope detect opacities in the vitreous humour, or any other intraocular disease. These motes are most apparent when a bright surface, such as a white wall or the field of a microscope, is looked at. Mouches volantes have no clinical importance. Those annoyed with them should be strongly recommended not to look for them, as in that case others are very apt to become visible. They depend, probably, upon minute remains of the embryonic tissue in the vitreous humour.

Fluidity of the Vitreous Humour, or **Synchysis** (*σύν, together*; *χέω, to pour*), is not rare. It can only be diagnosed with certainty when the humour contains floating opacities. Low tension of the eyeball does not always indicate fluidity of the vitreous, although soft eyeballs nearly always contain fluid vitreous humour. Trembling of the iris is also no sign of fluid vitreous, but merely indicates that the iris is not supported in the normal way by the crystalline lens. Defective zonula of Zinn, however, is often caused by, or is a concomitant of, fluid vitreous, and by causing displacement of the lens, would allow of trembling of the iris. *The Causes* of synchysis are,

chorioiditis, and staphyloma of the chorioid and sclerotic, and it also occurs as a senile change.

Synchysis Scintillans is a fluid condition of the vitreous humour, with cholesterine and tyrosine crystals held in suspension in it. The ophthalmoscopic appearances are very beautiful, resembling a shower of golden rain. A satisfactory explanation for the occurrence of these crystals in this position has not yet been given. They usually occur in old people, and seldom cause any marked deterioration of vision.

Fluidity of the vitreous humour is not, *per se*, a condition of serious import, unless the eye come to be the subject of an operation involving an incision in the corneo-sclerotic coat, when it renders prolapse of the vitreous more liable to take place.

Foreign Bodies in the Vitreous Humour.—One of the most common accidents to the eye is perforation of the sclerotic, or of the cornea and crystalline lens, by a small foreign body (shot, morsel of iron, copper, stone, or glass), which then lodges in the vitreous humour.

In cases where the ophthalmoscope fails us, owing to extravasation of blood, etc., it is sometimes not easy to say whether the foreign body be in the eye, or whether it may merely have punctured the sclerotic and fallen to the ground. If it be known to have been a small foreign body, which has flown against the eye with force, the probabilities are that it is lodged in the eye.

But if the case be brought immediately, or soon after the accident, and there be no intraocular hæmorrhage to obscure our view, the foreign body may frequently be detected with the ophthalmoscope in the vitreous humour as a dark or glittering body, according to its nature. And focal illumination with dilated pupil will often help us to discover a foreign body situated in the anterior part of the vitreous humour. Or, if it cannot be seen, an opaque streak through

the vitreous humour, one end of which corresponds with the sclerotic wound, may indicate the track taken by a foreign body.

In case the foreign body have perforated the cornea, and reached the vitreous humour through the circumlental space, a counter-opening will be found in the iris; while, if it be supposed to have passed through the cornea and lens, the openings both in the anterior and posterior capsule of the lens should be sought for.

It is rarely that a foreign body, whether it remain free, or, as sometimes happens, become encapsuled, is tolerated permanently in any part of the interior of the eye, and the event should never be calculated on in the treatment of such a case.

As a rule, foreign bodies in the vitreous, as elsewhere within the eye, soon produce violent inflammatory reaction. This occurs either by reason of infective micro-organisms being introduced into the eye with the foreign body, or it may be caused by the oxidisation of the foreign body, when, as is most common, it is of iron or copper. The form of inflammation may be either a plastic or purulent uveitis, in the latter case with purulent infiltration of the vitreous humour and hypopyon.

An eye which contains a foreign body that is not, or cannot be, at once removed, may be regarded as lost; and such an eye becomes, moreover, one of the surest sources of sympathetic ophthalmitis.

It is, consequently, of the utmost importance to remove every foreign body from the interior of the eye if possible, and with the least delay; or, if not, carefully to watch the eye, and at any sign of inflammatory reaction to remove the eyeball. Indeed, in view of the fact that this inflammatory reaction almost invariably comes on sooner or later, I should be inclined to remove most of these eyes at once, when the foreign body cannot be extracted.

Removal of the Foreign Body is very often an extremely difficult and disappointing undertaking, but it should always be attempted when, being neither steel nor iron, it is visible within the eye, so that its position can be determined with the ophthalmoscope or by focal illumination. The introduction of the magnet for the removal of fragments of the two metals named has made it unnecessary that they should in every case be visible, although here too the chances of success are much enhanced if the foreign body can be accurately localised. In all these operations it is necessary that the patient should be deeply under the influence of an anæsthetic, in order that as little vitreous humour as possible may be lost. And again, strict antiseptic measures must be observed, lest by our operation the very form of mischief be produced which it is our desire to avert.

There are several methods of proceeding. Atoms of glass, copper, stone, etc., may sometimes be removed through an incision in the sclerotic which is either an enlargement of the opening made by the foreign body, or is a special one, at a point more clearly corresponding to the actual position of the latter in the eye. This incision should lie between two recti muscles, should have an antero-posterior direction, and, in order that it may gape but little, should be a puncture with a broad keratome. Prolapse of the vitreous is then produced by pressure on the eyeball, and the foreign body is evacuated.

This method should only be tried when the foreign body is situated in the periphery of the vitreous, and towards the equator of the eye, where the opening for its exit can be made in its immediate neighbourhood; but the proceeding is often attended with disappointment, much vitreous being lost, while the foreign body remains in the eye.

Or, a forceps is passed in through the opening, and while the foreign body is kept in view with the ophthalmoscope, it is seized and drawn out. This plan is also

unsatisfactory, as, loss of vitreous occurring, the cornea becomes flaccid, and the view of the foreign body is soon obscured.

Again, some surgeons prefer to make their opening not close to the foreign body, but exactly at the opposite side of the eyeball, by which means they can often reach the foreign body with greater ease, and with less injury to the tissues.

The magnet, thanks to M'Keown, of Belfast,[1] has of late years come into use for the removal of fragments of steel and iron from the interior of the eye, and especially from the vitreous humour. Electro-magnets are those now employed for this purpose, the instruments of Hirschberg[2] and of

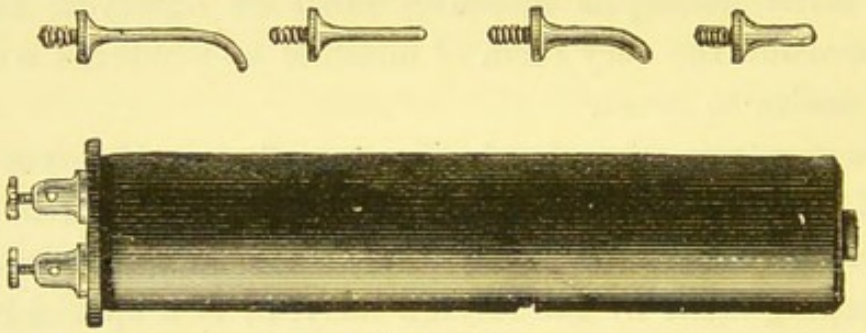


FIG. 129.

Simeon Snell[3] being the most suitable. Fig. 129 represents Snell's instrument in two-thirds its actual size. It is a core of soft iron, around which is placed a coil of insulated copper wire, the whole enclosed in an ebonite case. To one end of the instrument are attached the screws to receive the battery connections. At the other extremity the core projects just beyond the ebonite jacket, and is tapped, and into it screws the needle. Needles of various kinds or shapes can be adjusted to the magnet according to the case to be dealt with. The battery used is a quart bichromate element. A needle being passed through the sclerotic opening, is

[1] *Brit. Med. Journal*, 1874, vol. i., p. 800, and elsewhere.
[2] *Centralblatt für prak. Augenheilkunde*, 1879, p. 380.
[3] *The Electro-magnet*, etc. (London, 1883).

advanced towards the foreign body, when the latter adheres to it, and is drawn towards the wound. Much care is required in drawing it through the opening, lest it be rubbed off the needle in its passage. A forceps is generally used at this part of the proceeding, either to dilate the wound, or to seize the foreign body and extract it. Haab[1] recommends the use of a very powerful magnet applied to the surface of the eyeball; he has succeeded with it in removing bits of steel through the wounds inflicted by them.

The magnet may also be used for determining the presence of a fragment of steel or iron in the vitreous, if, on bringing it close to the eye, motions are imparted to the fragment. T. R. Pooley,[2] made some very elegant experiments to ascertain the presence of a piece of steel in the eye, upon the principle that if a fixed magnet attracts a movable piece of steel, a fixed piece of steel will attract a movable magnet. He magnetised a sewing-needle, and suspended it by a fibre of silk attached to its centre, and on bringing it near an eye which contained an atom of steel the needle dipped towards the foreign body. Or, if he magnetised the foreign body by passing a galvanic current through the eye, the motion of the suspended magnet was even more decided.

Cysticercus in the Vitreous Humour is not of rare occurrence in some parts of Germany, but there have been only one or two such cases observed in the British Isles.

The original seat of the entozoon is usually beneath the retina (see chap. xv.), through which it breaks to reach the vitreous humour; but it also sometimes makes its first appearance in the vitreous. It is recognised by its peculiar, somewhat dumb-bell shape, its iridescence, and its peristaltic motions. The vitreous humour often becomes full of peculiar membraneous opacities.

Treatment.—Removal by operation. The prospects for the

[1] *Bericht. d. Ophthal. Gesellschaft zu Heidelberg*, 1892.

[2] *Archives of Ophthalmology*, 1880, p. 219.

eye are very much worse than in the case of a subretinal cysticercus.

Blood Vessels are sometimes formed in the vitreous humour; they spring from the retinal vessels, often in connection with connective tissue formations which accompany hæmorrhages; but sometimes small loops arise in the neighbourhood of the disc, without any hæmorrhagic disease.

Persistent Hyaloid Artery.—In intra-uterine life the hyaloid artery is a prolongation of the central artery of the retina, and runs from the papilla to the posterior surface of the crystalline lens. It completely disappears prior to birth, except in those rare cases where it remains as an opaque string, which may stretch the whole way from papilla to lens, or may extend only part of the way. It is then thrown into wave-like motions by the motions of the eyeball, and is easily recognised with the ophthalmoscope. It does not usually cause any disturbance of vision.

Detachment of the Vitreous Humour from the Retina, although probably a common diseased condition, cannot as yet be recognised with certainty during life, and rarely becomes the immediate cause of blindness. Its danger lies in its liability to bring about detachment of the retina.

Detachment of the vitreous may be either idiopathic or due to trauma. In the idiopathic cases, chronic chorioiditis is the primary disease, which gives rise to a change in the fine connective tissue elements of the vitreous, with consequent shrinking of this body. Yet, with the ophthalmoscope, the chorioid may seem normal; and moreover, although floating opacities may be present in the vitreous chamber, yet it is quite possible for a perfectly clear vitreous to be detached.[1]

The condition occurs in connection with high degrees of myopia, where chorioiditis is also common, and is probably the most important factor in the production of the detachment

[1] Nordenson, *Die Netzhautablosüng* (Wiesbaden, 1887).

of the retina, so frequent in these eyes. Anterior staphyloma, hæmorrhages into the vitreous humour, and neoplastic growths between the vitreous and retina, also give rise to detachment of the vitreous.

Detachment of the anterior portion of the vitreous occurs in many cases of irido-cyclitis.

With regard to traumatic cases, all perforating injuries attended with loss of vitreous, including cataract operations—and sometimes, when the wound is in the sclerotic, without loss of vitreous—are liable to be followed by detachment of the vitreous.

I have[1] recorded a case in which detachment of the vitreous was the chief lesion in the eye, and was the cause of blindness, the vision being reduced to perception of light. The detachment had probably been brought about by an idiopathic hæmorrhage from the ciliary body into the anterior part of the vitreous. It lay (Fig. 130) immediately behind the lens, and in contact with it, and presented the appearance of a greyish opacity, much like a detached retina, but for the absence of retinal vessels. Suspicion of an intraocular tumour existing, the eye was removed. The vitreous lay against the ciliary body and lens, while the vitreous chamber was filled with serous fluid, and the retina was in its normal position. In the retina, towards the ora serrata, there were a few minute hæmorrhages.

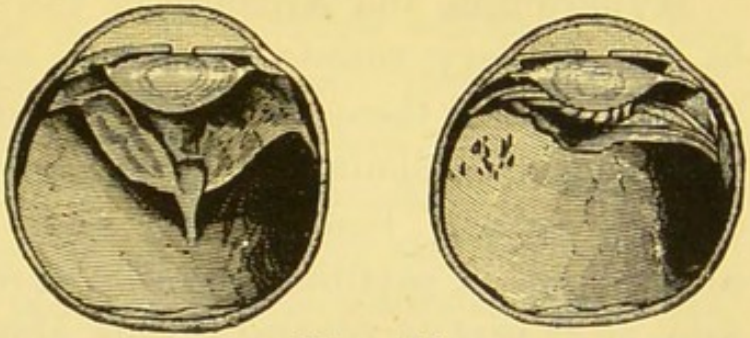


FIG. 130.

[1] *Trans. Ophthal. Soc.*, 1882, p. 41.

CHAPTER XV.

DISEASES OF THE RETINA.

Diseases of the retina may, for the purpose of description, be conveniently grouped as follows:—Alterations in Vascularity, Functional Affections, Inflammation, Atrophy, Diseases of the Bloodvessels, Injury by Strong Light, Tumours, Parasitic Disease, Detachment, and lastly, Traumatic Affections.

Alterations in the Vascularity of the Retina.

Hyperæmia and **Anæmia** of the retina, due to changes in the capillary vessels, cannot be seen with the ophthalmoscope, hence these terms are used to denote apparent enlargement or diminution of the principal branches of the central vessels. *Venous Engorgement* may occur as a local condition, as in papillitis, retinitis, thrombosis of the central vein, or as part of general venous obstruction in cardiac and pulmonary diseases. *Contraction of the Arteries* may also be due to local disease of the vessels, as embolism, albuminuric retinitis, etc., and spasm (malaria, quinine), or, more rarely, to diminished blood supply from general causes (cholera). The opposite conditions, namely, diminution in the size of the veins, and enlargement of the arteries, are rarely noticeable.

Functional Affections.

Hyperæsthesia of the Retina.—The symptoms of this affection are photophobia, lacrimation, and blepharospasm

when the patient is exposed to ordinary daylight. There are no *Ophthalmoscopic Signs*. The chief causes are hysteria, long-continued use of the eyes with very bright objects, and neuralgia of the fifth pair.

Treatment consists in removal of the cause, rest of the eyes, and protection from light, with suitable measures for the general health.

Neurasthenic Asthenopia, or **Retinal Anæsthesia.**—This peculiar and rather rare affection is one about which we have still much to learn. It is a complex of eye-symptoms in connection with a debilitated state of the general nervous system, the eye itself being organically healthy. *These Symptoms* are—1. Diminished, but fluctuating, acuteness of vision. The effort or desire to see well is often the signal for the acuteness of vision to fail. 2. The rapid disappearance of objects from view, if looked at too long. 3. Attacks of defective sight, with positive scotomata coming on suddenly and without provocation, and lasting for a few minutes. 4. Apparent contraction of the field of vision. If a perimetrical examination be made, the field will be found contracted; but, as Wilbrand[1] has pointed out, this contraction is merely a sign of retinal exhaustion, as indicated by the fact that the longer the examination is continued, the more contracted does the field become. Forster has also shown, that, in these cases, the test object is seen further out, when brought from the periphery up to the centre, than when it is moved from the centre outwards. If the test object be carried, in each meridian, from one side of the perimeter to the other, and then back again, two fields will be obtained which overlap each other. 5. Optical impressions are retained but a short time. The appearance of persons, places, etc., is not remembered when seen soon again. 6. Muscular asthenopia. Insufficiency of the internal recti is often present, as well as

[1] *Archives of Ophthalmology*, xii. 428.

defective accommodation. 7. Hyperæsthesia of the retina. Dazzling is caused by even moderate light, and strong contrasts of light and shade are distressing, while the acuteness of vision is often improved when blue or smoked glasses are worn.

The Ophthalmoscopic Appearances are normal, or consist merely of some hyperæmia of the optic papilla.

The *Concomitant General Symptoms* of the condition consist in insomnia; tinnitus; subjective sensations of hearing; exalted, or, again, defective sensations of taste and smell; sensations referred to the skin, such as formication, itching, burning, numbness, heat and cold; great restlessness of body; depression of spirits; want of mental energy; absent-mindedness; lassitude.

The persons in whom the affection is most common are children before and at the time of puberty, and women labouring under hysteria, anæmia, chlorosis, ovarian irritation, or displacement of the uterus: but it is also occasionally found in men.

Treatment.—Any uterine or other local disorder must be relieved, so far as possible. Rest of body and mind is to be enjoined, with fresh air and moderate exercise. Strychnine hypodermically is a valuable remedy in the affection, and with it iron and quinine internally may be associated, and bromide of potassium with hyoscyamus to promote sleep. In some, especially in hysterical, cases, valerianate of zinc is beneficial. Sea-bathing, and cold shower-baths, with change of air, are valuable adjuncts of the treatment. Blue, or smoked, protection glasses are most grateful to the patient, and promote the cure, but the spectacle-frames often cause annoyance by their pressure on the nose or face. Errors in refraction should be corrected.

The Prognosis is favourable, inasmuch as ultimate recovery is assured, but the course of the affection is excessively chronic, extending over months or years, with frequent relapses.

Inflammation of the Retina—Retinitis.

Retinitis, in general, is characterised by the following ophthalmoscopic appearances: *diffuse cloudiness*, especially of the central portion of the fundus, due to loss of transparency in the retina; the *optic papilla* becomes more or less congested, with indistinctness of its outline, which in the erect image resolves itself into a delicate striation; *vascular engorgement*, the retinal veins especially becoming enlarged and tortuous. The inflammation in some cases may subside at this stage, but, as a rule, *hæmorrhages*, and whitish *exudations* soon make their appearance.

The various forms of retinitis are distinguished by the predominance of some of the above signs, and also by the peculiar appearance and grouping of the exudations.

If the optic papilla be not merely congested, but also swollen, the condition is called Neuro-Retinitis. In some cases of retinitis, the chorioid is also involved, and to these the name chorio-retinitis is given. Inflammation of the retina is rarely a local affection, being, in most cases, due to general diseases, and hence it commonly occurs in both eyes.

Syphilitic Retinitis (or Syphilitic Chorioido-Retinitis).—Inherited, or acquired, constitutional syphilis is liable to induce a form of chronic diffuse retinitis. In the acquired disease it is a later secondary symptom, coming on between the sixth and eighteenth month, often only in one eye.

With the Ophthalmoscope a slight opacity of the retina is seen extending from the papilla some distance into the retina, and very gradually disappearing towards the equator of the eye. The papilla is but slightly hyperæmic, while its margins are indistinct, like those of the moon seen through a light cloud. The artery is not generally altered, and the vein but slightly distended. Opacities in the vitreous humour are not uncommon. They may be membraneous or thread-like, but

a diffuse dust-like opacity, filling the whole vitreous humour, is almost pathognomonic of a syphilitic taint, and may create much difficulty in the ophthalmoscopic diagnosis of the retinal affection.

Disseminated chorioidal changes, in the form of small yellowish spots with pigmentary deposit, are very frequent, especially towards the equator of the eye. Many observers, indeed, hold, that the whole process is primarily in the chorioid, and that the retina is only secondarily affected. Fine whitish dots, and pigmentary changes, often occur about the macula lutea.

Vision may be but slightly affected, but in the advanced stages it is usually much lowered. Central, or peripheral, or ring scotomata, or concentric defects of the field, are found. The scotomata are often positive, *i.e.*, they can be seen by the patient as dark spots in the field. Night-blindness is a constant symptom, and the light-sense is enormously diminished. The patients sometimes complain of sparks or lights, which seem to dance before their eyes, and occasionally also of a diminution in the size (micropsia) of objects, or of a distortion (metamorphopsia) of their outlines. The micropsia is believed to be due to a separation from each other of the elements of the layer of rods and cones, by subretinal exudation. The image of an object then comes into relation with fewer of these elements, and thence the mental impression is that of a smaller object than is conveyed by the image formed in the sound eye, or on a sound part of the same retina.

The Progress of the Disease is very slow, and is liable to relapses. In the late stages extensive pigmentary degeneration of the retina may come on, or disseminated chorioiditis. But if the cases come under suitable treatment in an early stage, a cure may often be effected.

Treatment.—The only remedy which is of real value is mercury, and that in an early stage. It should be used in a protracted course of some weeks by inunction, combined at

discretion with small doses of calomel internally. Perchloride of mercury hypodermically, in $\frac{1}{20}$ gr. doses once a day, is also a suitable measure. If mercurialisation be effected, it should not go farther than a very slight stomatitis. Pilocarpine hypodermically, Turkish baths, and the artificial leech at the temple may be employed as adjuncts to the treatment. When the mercurial course has been completed, iodide of potassium should be prescribed as an after-treatment. Complete rest of the eyes, and protection from strong light by dark glasses, are also necessary, in this, as in many forms of retinitis.

Hæmorrhagic Retinitis.—In this affection the retina contains a number of small hæmorrhages. They occur chiefly between the fibres of the inner layer, and consequently present a flame-like appearance as seen with the *Ophthalmoscope*. Any which lie in the outer layers are most apt to be round or irregular in shape. In addition to the hæmorrhages, there is diffuse opacity of the retina, and sometimes white spots of degeneration. The papilla is often much swollen, and the retinal veins distended and tortuous, while the arteries are small; but these appearances, as well as the number of the hæmorrhages, vary much in different cases. When there are but few hæmorrhages, they are situated in the neighbourhood of the papilla and macula lutea. The appearances occasionally resemble those of albuminuric retinitis, but in the latter, as a rule, the proportion of white spots to hæmorrhages is greater than in this affection. Probably many cases described as hæmorrhagic retinitis are due to thrombosis of the central vein. See p. 418.

Causes.—The affection is found most commonly in connection with cardiac disease, *e.g.*, valvular insufficiency, and hypertrophy of the left ventricle; or, with diseases of the vascular system, *e.g.*, atheroma, and aneurisms of the large vessels. Where it is due to disease of the coats of the arteries, the ophthalmoscope will occasionally reveal an

arterial branch altered to the appearance of a white thread; but, usually, the degenerative change does not interfere with the transparency of the vascular coats. In the majority of cases dependent on cardiac or vascular disease the retinal affection is monocular. This, and the frequently sudden onset of the retinitis, led Leber[1] to think that some second factor for its occurrence exists, probably multiple embolisms of the small branches of the central artery. Suppression of menstruation, or other wonted discharge—such as that from piles—has been observed as an immediate cause of hæmorrhagic retinitis.

A peculiar form of hæmorrhagic retinitis is sometimes associated with secondary syphilis. In addition to the usual opacity of the retina in syphilitic retinitis (*vide infra*), a portion of the retina is covered with numbers of small round hæmorrhages lying in the different layers of the retina, while a connective tissue development is occasionally found in the nerve-fibre layer, in the form of white striæ along the course of the blood-vessels.

The disturbance of vision is considerable, especially if the neighbourhood of the macula lutea be much involved.

The Prognosis is bad in severe cases of hæmorrhagic retinitis. Relapses are common, while the ultimate tendency is towards atrophy of the retina and papilla. In very mild cases recovery may come about.

The Treatment must be chiefly expectant, or directed, at most, towards procuring rest for the general system, or for the organ primarily at fault. Dry cupping on the temple, hot foot-baths, and iodide of potassium internally may be employed.

Retinitis Albuminurica occurs as a complication in many cases both of acute and chronic nephritis, and in the albuminuria of pregnancy. It is most common with the small

[1] *Graefe und Sæmisch's Handbuch*, Bd. V., p. 570.

granular kidney, but may attend any chronic form of Bright's disease. It occurs in 6 or 7 per cent. of the cases.[1] Simon[2] has found typical violet-blindness in retinitis albuminurica, and considers this not rare, and a symptom characteristic of the affection.

The Defect of Vision in the chronic form, although often an early, or even the first, symptom, is never associated with an early stage of the kidney disease, but rather with a late stage of it, and with dilated left ventricle. Both eyes, as a rule,[3] are affected, although often not equally so; vision is much lowered, and even perception of light may be wanting; but the blindness is not always all due to organic changes in the retina, being often largely the result of uræmia.

Ophthalmoscopic Appearances.—These are venous hyperæmia and swelling of the papilla, and of the retina in its neighbourhood; hæmorrhages on the papilla, and in the nerve-fibre layer of the retina; and round or irregularly shaped white spots in the retina, arranged in a zone around the papilla, some three papilla diameters from it. These changes take place in the order in which I have enumerated them. The hyperæmia and engorgement of the veins, often very great, become less, according as the white spots become more developed. Near the macula lutea no very coarse changes usually occur, but fine white dots are found, with a star-like arrangement converging towards the macula. In some cases the spots spread out only on the inner side of the macula, towards the papilla. The degree in which all these different changes are present varies in different cases, no one of them being pathognomonic of the kidney affection, but rather the grouping of the whole picture being suggestive. Sometimes the papillitis is so intense as to simulate that formerly

[1] Berger, *Maladie des Yeux et pathologie générale*, Paris, 1893, p. 246.

[2] *Centralbl. f. Augenhk.*, May 1894, p. 132.

[3] A few cases are recorded in which only one eye was attacked, and in several of these it was found that but one kidney was present.

known as "congestion papilla" in cases of intracranial tumour; while the white spots are sometimes developed to such a degree as to become confluent, and to form one large white plaque. Again, the papillitis, or white spots, or both, may be but slightly marked. The number and size of the hæmorrhages are also liable to great variation. Detachment of the retina has been observed in a few cases, and in some the hæmorrhages burst into the vitreous humour.

Some of the white spots are caused by fatty degeneration of the outer layers of the retina (the retinal vessels passing over them), others by hypertrophy of the nerve-fibre layer (the retinal vessels hidden by them). The fine dots about the macula lutea are the result of fatty degeneration of the inner ends of Müller's fibres. Small aneurismal dilatations of the arteries occur very occasionally.

The connection between the renal and retinal affections is not known with certainty, but the theory that the latter is due to chronic uræmia is probably correct.

Prognosis.—In these cases the prognosis as regards the patient's life is bad. The majority die within eighteen months or two years; but, if the general disease remain stationary, or improve, or recover, the retinal changes may improve or disappear, and leave the retina with normal appearances and functions; or, the swelling, hyperæmia, white spots, and hæmorrhages may give place to optic atrophy, with diminution in size of the arteries, pigmentary alterations in the retina, and blindness.

Treatment.—Dry cupping at the temple is about the only remedy which can be employed directly for the retinal affection, and I will not say that it is of much use. Taking into consideration the serious import of this eye-symptom for the life of the patient, it is a question whether, in many cases of pregnancy with albuminuric retinitis, abortion should not be resorted to, especially if the pregnancy have still some months to run. But, on the whole, the prognosis is more favourable

in the albuminuria of pregnancy, than in interstitial nephritis. Complete recovery has been observed.

Retinal Affections in Diabetes.—There is no one condition of the retina characteristic of diabetes, although undoubtedly retinal affections occasionally do complicate it in an advanced stage. Small retinal hæmorrhages, with fine changes, in the form of glistening dots, about the macula lutea, somewhat similar in appearance to those which occur in Bright's disease, except that they rarely form the well-marked star, are perhaps the most common and suggestive appearances. In other cases retinal hæmorrhages alone are found, and in others hæmorrhagic retinitis; while, again, the so-called typical appearances of Bright's disease may be presented. There are often opacities of hæmorrhagic origin in the vitreous humour, and iritis may come on. Leber lays down the important rule, that in all cases of retinal hæmorrhages, and of retinitis hæmorrhagica, the urine should be examined for sugar.

Retinitis Leucæmica.—In not more than one-third or one-fourth (Leber) of the cases of leucocythemia does a retinal affection occur, and it is not always of the same type. It may consist in a slight diffuse retinitis, accompanied by some extravasations of pale blood, while the blood-vessels are also pale, the veins being much enlarged, and rather flattened than over-distended, the arteries small, and the chorioid of an orange-yellow colour. Or, it may resemble a case of ordinary hæmorrhagic retinitis.

The Appearances most characteristic of the affection are:—a pale papilla, with indistinct margins; slight opacity of the retina, especially along the vessels; small hæmorrhages; round, white, elevated spots, up to 2 mm. in diameter, with a hæmorrhagic halo, situated by preference towards the periphery of the fundus and at the macula lutea, but not at all, or only in very severe cases, in the zone between the macula and the equator of the eye. These white spots consist of

extravasations of leucæmic blood, the result, Leber thinks, of diapedesis.

Vision may be but little affected if the macula lutea be fairly free. Hæmorrhage into the vitreous humour may cause complete blindness.

Retinitis Punctata Albescens (Mooren); **Retinitis Centralis Punctata et Striata** (Hirschberg).—A few cases of this peculiar affection have been described.[1] These have occurred in middle-aged or elderly people, whose general health was good; or, if disordered, was not similarly so in any two cases. The defect of vision may come on rapidly, or may be gradually developed in the course of many years. It consists in a lowering of the central vision, with positive or relative scotoma, or both may be present; but the eccentric field remains intact.

The Ophthalmoscope discovers great numbers of minute white glistening dots and fine white striæ in the retina, chiefly between the papilla and macula. A retinal hæmorrhage was noted in one case, and in only one was slight papillitis present. The affection is probably of inflammatory origin.

Treatment consisted in Heurteloup's leech, iodide of potassium, protection of the eyes, and care of the general health. Cure took place in one case, while in no instance did serious blindness come on.

Development of Connective Tissue in the Retina, or **Retinitis Proliferans.**—Extensive white striæ, formed of connective tissue, are sometimes seen in the retina, and may even conceal the vessels and papilla. They are the result of hæmorrhages, traumatic or otherwise, according to Leber, and of an inflammatory process according to Manz, and are formed by proliferation of Müller's fibres and new growth of connective tissue.[2] Hæmorrhages in the retina or in the

[1] Mooren, *Fünf Lustren Ophthalmologischer Wirksamkeit*, p. 216. Hirschberg, *Centralblatt f. prak. Augenheilkunde*, 1882, p. 330.

[2] Banholzer, *Archiv. of Ophthal.*, xxii. 2, p. 212.

vitreous humour, or in both, are generally present at some period (p. 390). Vision is often but slightly affected, but the danger of recurrent intraocular hæmorrhages renders the ultimate prognosis bad, as a rule.

Treatment.—Heurteloup's leech. Iodide of potassium, or perchloride of mercury. Protection spectacles.

Retinitis Circinata is a rare disease, first described by Fuchs.[1] It occurs in old people, chiefly women, and is characterised by very remarkable appearances. At the macula is a grey or yellowish cloudy patch, which may attain the size of the papilla, and sometimes presents hæmorrhages on its surface; surrounding this, but separated from it by a healthy zone, is a ring composed of numerous, closely set, small white spots, which are confluent in places. According to de Wecker [2] this disease is only the result of a variety of hæmorrhagic retinitis.

Purulent Retinitis is observed as the result of septic embolism of the retinal arteries in septicæmia after surgical operations, etc., and very frequently in cases of metria, and it is usually, in the latter condition, a fatal sign.

In an early stage, the *Ophthalmoscope* shows a number of small hæmorrhages in the retina, with general cloudiness of the retinal tissues, while the actual embolisms, which are usually multiple, may not be visible.

The inflammation makes rapid progress, soon destroying sight, and extending to the chorioid, iris, and vitreous humour, until finally panophthalmitis is reached. The retina is sometimes alone the primary seat of the embolic attack, and sometimes the chorioid is also involved. The embolisms are often little more than masses of micrococci.

---

[1] *Von Graefe's Archiv*, vol. xxxix., part 3, p. 227.

[2] *Archiv d'Ophthalmologie*, Jan. 1894. I have seen a most typical example of this disease, in one eye, in an old woman of sixty. De Wecker and Fuchs have in all seen twenty-seven cases.—L.W.

The retina becomes secondarily implicated in many purulent processes, which commence in other parts of the eye.

Atrophy of the Retina.

Atrophy, or degeneration, of the retina is characterised by diminution, or even complete obliteration, of the retinal vessels, accompanied by more or less atrophy of the papilla. It may be caused by severe forms of retinitis, and also by embolism or thrombosis.

Retinitis Pigmentosa is a degenerative, rather than an inflammatory, affection of the retina. It is extremely chronic in its progress, coming on, most commonly, in childhood, and often resulting in complete, or almost complete, blindness in advanced life.

Vision is much affected, but the symptom most complained of is night-blindness (nyctalopia = *νὺξ, night; ὤψ, the eye*), due rather to defective power of retinal adaptation than to defective light-sense. The field of vision, moreover, becomes gradually contracted, until only a very small central portion remains; so that, although the patient may still be able to read, he cannot find his way alone—a function for which the eccentric parts of the field are the most important. An annular defect in the field is seen in some cases. Finally, the last remaining central region becomes blind.

The Ophthalmoscopic Appearances consist in a pigmentation of the nerve-fibre layer of the retina, which commences in the periphery, but not at its extreme limits, and in the course of years advances towards the macula lutea. The pigment is arranged in stellate spots, of which the processes intercommunicate, so that the appearance reminds one of a drawing of the Haversian system of bone. Pigment is also deposited along the course of many of the vessels, hiding them from view. The degree of pigmentation varies much, and in some cases is quite absent, and the diagnosis then has to depend upon the other appearances, and on the symptoms.

The papilla is of a greyish-yellow colour, never white, and the vessels are very small.

The chorioid is sometimes slightly affected, irregularity in its pigmentation being observable.

Pathology.—The pigment in the retina is believed to wander into it from the pigment-epithelium layer. The other pathological changes in the retina consist in hyperplasy of its connective tissue elements, and thickening of the walls of the vessels at the expense of their lumen.

The chorioidal vessels, too, are altered,[1] owing to an endarteritis, which causes hypertrophy of their coats, with more or less obliteration of their lumen. In fact, it seems probable, that the primary seat of the diseased process is in the chorioid; and that it is the changes in it, which cause the pigment from the pigment-epithelium layer to wander into the retina.

Causes.—Retinitis pigmentosa often affects more than one member of a family; and the patients, too, are frequently defective in intelligence, or deaf and dumb. Many of them are the offspring of marriages of consanguinity, and in others an inherited syphilitic taint is present, while in others no cause can be assigned. Other congenital defects, supernumerary digits, etc., are sometimes present. A few cases of retinitis pigmentosa with glaucoma have been observed.

Treatment is of little use. At best one may stimulate the torpid retina by hypodermic injections of strychnia, or with the continuous current. The latter means has found an advocate in Dr. Marcus Gunn,[2] and I have seen several cases in which temporary benefit was obtained from it.

Diseases of the Retinal Vessels.

Apoplexy of the Retina.—This differs from hæmorrhagic retinitis, in that the hæmorrhages are found in a retina free from other diseased appearances, especially retinitis.

[1] Wagenmann, *A. v. Graefe's Archiv*, xxxvii. 1, p. 230.

[2] *Ophthal. Hosp. Rep.*, vol. x., p. 161.

With the Ophthalmoscope, the extravasations of blood appear as red, or almost black, spots of various sizes and shapes. Their number and position in the fundus are also variable. They may be in any layer of the retina, and may sometimes burst into the vitreous humour, and sometimes become extravasated between the retina and chorioid.

Vision is interfered with according to the position and extent of the hæmorrhages. Wherever an apoplexy be situated, the function of the retina at that place is suspended. If it be at the macula lutea, central vision will be seriously impaired; while the scotoma produced by an apoplexy at the periphery of the fundus may pass unnoticed by the patient.

Causes.—Retinal apoplexies are most common in advanced life, with atheroma of the blood-vessels, and are then valuable as a warning of possibly impending cerebral mischief. Other causes are:—hypertrophy of the left ventricle; suppression or irregularity of menstruation, or at the climacteric period; the sudden reduction of tension of the eyeball after iridectomy for glaucoma; the gouty diathesis (Hutchinson); progressive pernicious anæmia, or anæmia from loss of blood (hæmatemesis, etc.), or from exhausting diseases. In connection with this latter cause of retinal apoplexy, Stephen Mackenzie has pointed out[1] that when the corpuscular richness of the blood falls below 50 per cent., whatever the cause of the anæmia, the tendency to retinal hæmorrhage is present.

In young people of both sexes, from the fourteenth to the twentieth year of age, large retinal apoplexies, which may extravasate into the vitreous humour, are sometimes seen, and it is difficult to assign a cause for them. Some of the subjects are weak or anæmic, while many of them are in perfect health. Neither Eales[2] nor Nieden[3] has found these apoplexies in young women, but this does not conform with my experience,

[1] *Trans. Ophthal. Soc.*, December 13th, 1883.
[2] *Ophthal. Review*, 1882, p. 41.
[3] *Bericht d. Ophthal. Gesellsch.*, 1882.

nor with that of many others. Eales has noted irregular circulation, epistaxis, and constipation in these cases.

Prognosis.—The apoplexies are observed, in the course of weeks or months, to become paler and smaller, often leaving after them chorioidal changes, or greyish spots dependent on degeneration of the retina, and in some extreme cases atrophy of the whole retina may result.

Occasionally, absorption of the hæmorrhages is accompanied by complete restoration of vision, but usually the scotomata remain. Recurrences of the hæmorrhages are very common. Glaucoma comes on as consecutive to retinal apoplexies in some instances, and is then known as hæmorrhagic glaucoma, an incurable form of the disease (p. 337).

In other cases the hæmorrhage, having invaded the vitreous humour, gives rise to dense permanent opacity in it, followed, perhaps, by detachment of the retina.

Treatment.—Active measures are of little use. Cold compresses at first, with a pressure bandage, and dry cupping to the temple, may be employed. The general state of the patient must be attended to, and no violent muscular efforts permitted.

Embolism of the Central Artery of the Retina.—Sudden or very rapid blindness, beginning at the periphery of the field, and advancing towards the centre, is the only symptom experienced by the patient.

Immediately after the attack, the *Ophthalmoscope* shows a marked pallor of the papilla, while the artery and its branches are empty of blood, resembling fine white threads, and the veins are diminished in size at the papilla, but increase somewhat towards the periphery. Pressure on the eyeball produces no pulsation, nor change in calibre, of the vessels, as it does in a sound eye. Usually, on the following day, the central region of the retina begins to assume a greyish-white, opaque appearance, consequent on disturbance of nutrition, in the midst of which the macula lutea is seen as a purple-red spot. De Schweinitz has seen this cherry-red spot at the

macula twenty minutes after the embolism took place. The little blood contained in the vessels may soon be seen divided into short columns with colourless interspaces, and these move along the vessels with a slow jerky motion. Minute hæmorrhages often occur, most commonly between the macula and the papilla.

The peculiar appearance of the macula lutea is certainly not due to hæmorrhage. According to Liebreich it is merely a contrast effect, the red colour of the chorioid shining through, where no nerve-fibre layer is present. Leber suggests that the colour may be due to the retinal purple.

The infiltration of the retina passes away in a few weeks, and also the peculiar appearance of the macula lutea, while atrophy of the retina and papilla usually supervene.

Embolism of a branch only of the central artery has been observed. In these cases the infiltration, and the defect of vision, are confined to the part of the retina supplied by the embolised branch.

Prognosis.—Vision may improve for a time, but when atrophy commences, it falls back again, and, finally, power of perception of light is lost. Cases of embolism of a branch are more likely to recover.

Causes.—Endocarditis; mitral disease; atheroma of the large arteries of the body; aneurism of the aorta; pregnancy; Bright's disease. Two cases of chorea with embolism of the central artery are recorded.[1] But it is said also to occur in healthy persons without any discoverable cause.

Treatment.—Repeated paracentesis of the anterior chamber has been tried, and also iridectomy, with the object of reducing the tension, and in this way promoting a collateral flow of blood, by means of the only ascertained (Leber) communications between the retinal and chorioidal vascular systems; namely,

[1] H. R. Swanzy, *R. L. O. H. Reports*, September 1875; and A. Benson, *Ophthal. Review*, January 1886.

at the entrance of the optic nerve.[1] These attempts have been unsuccessful.

Several cases have been published, in which the circulation, which probably was not completely impeded by the embolus, was restored, and good vision regained; the recovery being probably due to the manipulations of the eyeball made in each case for the purpose of observing the effect of pressure on the vessels. So long as the pressure was maintained, a column of blood was being stored up behind the embolus, and, on removal of the pressure, rushed forward against the impediment, carrying it into some more remote vessel, or into the general vascular system. In fresh cases, massage of the eyeball suitably applied would, therefore, always be worth the trial.

Thrombosis of the Retinal Artery.—Blocking of the artery may occur spontaneously, from thrombosis due to failure of the heart's action and slowing of the arterial flow, the result, in its turn, of cardiac disease, spasm of the blood-vessels, disease of the walls of the vessels, or alterations in the quantity and amount of blood.

The *Ophthalmoscopic Signs* are in all respects similar to those of embolism.

The *Diagnosis* between thrombosis and embolism of the central artery can only be made by certain symptoms, which precede or accompany the attack in thrombosis, but are wanting in embolism. These are:—previous attacks of transient blindness in the blind eye, a simultaneous attack of blindness

[1] Gowers (*Manual of Medical Ophthalmoscopy*, p. 31) is of opinion that there are other anastomoses between these systems, probably by connection with the long ciliary arteries. A cilio-retinal vessel passing from the chorioid or sclerotic at the papilla to the region of the macula lutea is not an uncommon vascular anomaly; and Benson has published a case of embolism (*Ophthal. Hosp. Rep.*, vol. x., p. 336) in which the presence of such an artery seemed to have a favourable influence for the progress of the case, good central vision being recovered, although the field remained concentrically contracted.

in the fellow eye, and faintness, giddiness, and headache at the onset of the blindness.

Treatment.—When transient attacks of blindness are complained of, it is important to overhaul the patient's general state, and to correct, so far as possible, any condition which might be the cause of feeble circulation. When the true attack comes on, manipulation of the eyeball applied immediately, or paracentesis of the anterior chamber, might prove of use.

Thrombosis of the Retinal Vein, the occurrence of which was first proved anatomically by Michel, is seen chiefly in old people with atheromatous arteries, or cardiac troubles. Orbital cellulitis, from erysipelas or other causes, may also produce it.

The Ophthalmoscopic Appearances are: extreme engorgement of the retinal veins, with great narrowing of the arteries; the whole fundus is thickly studded with dark hæmorrhages; the optic papilla after a time becomes pale and undergoes atrophy, and the hæmorrhages, having become absorbed, leave an atrophied retina with thready arteries.

The Prognosis is very bad, sight becomes permanently damaged or lost, and *The Treatment* can only be directed to the general condition.

Aneurism of the Central Artery of the Retina occurs either as a relatively large dilatation, on the main branches of the artery, a very rare condition,[1] or as small miliary aneurisms, which may indicate the presence of others in the small arteries of the brain. Two interesting cases of the latter kind have been recorded by Story and Benson[2] and by Perinow[3] in men, aged respectively twenty and forty. In one of these cases there were also extensive connective tissue changes in the retina, the veins were dilated in places, and

[1] Sous, *Annales d'Ocul.*, 1865, liii. p. 241.
[2] *Trans. Ophthal. Soc.*, 1883, p. 108.
[3] *Centralbl. f. Augenheilkunde*, 1883, p. 392.

only one eye was affected. The minute aneurismal dilatations were globular, and situated laterally on the vessels; or they were fusiform, and involved the whole of its lumen. The number of aneurisms in an eye varied from three to nine. Neither case was followed to its end; but, it is to be presumed, that such eyes would 'run great risk of being ultimately lost through intraocular hæmorrhage.

A rational *Treatment* for the condition can hardly be devised.

Sclerosis of the Retinal Vessels (Perivasculitis, or, more rarely, Endarteritis) reveals its presence by narrowing of the blood column, and by the appearance of white lines along the vessels. It usually begins in the large trunks on the papilla, and may not extend much beyond the latter, as in some cases of optic atrophy; while in other cases (Bright's disease, hereditary syphilis[1]) it involves the small branches as well, and may even lead to obliteration of the lumen of the vessels, so that they look like white branching streaks. The arteries are more liable to this condition than the veins.

Injury of the Retina by Strong Light.

Blinding of the Retina by Direct Sunlight.—This is especially likely to occur on the occasion of solar eclipses, by observation with unprotected eye.

Immediately after the exposure, the patients complain of a dark, or semi-blind, spot in the centre of the field of vision; a positive scotoma, in short, which may even be absolute, and which interferes with vision in proportion to the length of the exposure. There may also be a central defect for colours, which may extend over a larger area.[2] A peculiar oscillation, or rotatory movement, is frequently observed in the

[1] Holmes-Spicer *Trans. Ophthal. Soc.*, vol. xii., p. 116.

[2] Mackay, *Ophthal. Review*, 1894, January, February, and March. A very interesting paper which contains abstracts of all reported cases.

scotoma, and is very persistent. Objects may also seem twisted or otherwise distorted (metamorphopsia).

The Ophthalmoscopic Appearances may be normal, but as a rule some changes exist, such as an alteration or loss of the light reflex at the macula, or a minute pale orange spot near the fovea, with, especially in the later stages, some darkening or pigmentation. When the cases are not severe, improvement in vision takes place, but complete recovery is not common. Hitherto no case with vision less than $\frac{1}{3}$ has regained $\frac{6}{6}$.

Czerny, and also Deutschmann,[1] demonstrated that concentration of the direct rays of the sun on the rabbit's retina gives rise to coagulation of the retinal albumen, with vascular reaction, diapedesis of blood corpuscles, and pigmentary disturbances. A bright white spot, with a dark red ring surrounding it, was seen with the ophthalmoscope. But the changes in the human retina produced by exposure to direct sunlight are not of similar nature, for, as has been shown by Widmark,[2] the intensity of light employed by those experimenters was much greater than in the clinical cases, the heat being sufficient to blister the skin.

Treatment.—Hypodermic injections of strychnia, the constant galvanic current, and dry cupping on the temple afford the best chances for promoting the cure. Rest and dark protection glasses are very important.

Snow-Blindness.—Exposure of the unprotected eyes for a length of time to the glare from an extensive surface of snow produces, in some persons, a peculiar form of ophthalmia, which may be followed by a temporary, or even permanent, amount of amblyopia. Although this condition is chiefly an affection of the conjunctiva, it is described here in order to compare it with the effects of sunlight and electric light.

Snow-blindness begins with sensations of a foreign body in the eye, photophobia, blepharospasm, and lacrimation; later

[1] *A. v. Graefe's Archiv*, Bd. xxviii., Abt. 3, p. 241.

[2] *Nordisk. Ophthal. Tidsskrift.*, v. 2.

on chemosis, with small opacities, or ulcers, of the cornea, comes on. The condition passes off in three or four days without leaving any permanent bad results, except in rare cases, when there may be some secondary hyperæmia of the retina.

Treatment.—The preventative treatment consists in the wearing of dark smoked glasses when travelling on the snow; while for the ophthalmia cold applications and cocaine are recommended, to relieve the distressing symptoms.

Effects of Electric Light on the Eyes.—The degree of intensity of light required to produce injurious effects on the eye is not known, but this much is certain, that no bad results have been observed from the use of the incandescent light.[1] Two groups of symptoms are observed from the action of a strong electric light on the eyes.

(*a*) *Electric Ophthalmia.* This has been chiefly seen in those employed in electric welding operations, and less frequently in electricians who use strong arc-light.[2] The symptoms begin shortly after exposure to the light, always within twenty-four hours, and are the same as those present in snow-blindness; the lids also are swollen, and even erythematous at times. The pupils are contracted. A slight muco-purulent secretion from the conjunctiva appears after the subsidence of the above symptoms. Recovery takes place in a few days, with complete restoration of vision, except in rare cases.

(*b*) *Blinding of the Retina.* This is the same affection as in blinding of the retina by sunlight. The central scotoma may persist after an attack of electric ophthalmia, or may occur without it. The injurious action of the electric light on the eye has been attributed to the chemical action of the ultra-violet rays, to the accompanying heat rays, and to dazzling of the retina. Widmark's experiments show that

[1] Hartridge, *Brit. Med. Journ.*, Feb. 1892, p. 382.

[2] Hewetson, *Brit. Med. Journ.*, June 1893, p. 1315. Berger (*loc. cit.*), p. 435.

changes can be produced in the retina by the electric light, without any heat coagulation. These changes consist in œdema, with more or less destruction of the nervous elements of the retina, namely, the outer layers, including the rods and cones, and the inner layer of nerve fibres.

Treatment.—The preventative treatment consists in the use of coloured glasses. Yellow glass has been recommended by Maklakoff. In the electric welding works in Germany a combination of deep blue and red is used, while the Sheffield workers prefer several layers of ruby glass. For the rest of the treatment see snow-blindness, and blinding by sunlight.

It may be as well to mention here that, for illuminating purposes, electric light possesses many advantages over gas, so far as the use of the eye is concerned. It has a greater illuminating power, produces less heat and no products of combustion, and hence it does not vitiate the atmosphere, or tend to cause conjunctival irritation. The electric light is much steadier than gas; and, on account of the smaller quantity of red rays which it emits, it forms a nearer approach to sunlight than does gas.

Tumour of the Retina.

Glioma of the Retina.—This is a malignant growth, which is found almost exclusively in young children,[1] and may even be congenital. It is the only growth which occurs in the retina. Owing to the age of the patients, the incipient stages of the disease are seldom observed, for they are unattended by pain or inflammation.

The growth commences as small, white, disseminated swellings in the retina, usually in one or other of the granular layers, more rarely in the nerve-fibre layer. The retina is apt to become detached at an early period, but there are

[1] A case of Glioma Retinæ in a man aged twenty-one is reported by Mervill, in the *Trans. American Ophthal. Soc.*, vol. iii., p. 364.

exceptions to this, especially when the disease starts from the nerve-fibre layer. In the early stages there is no iritis, cyclitis, or opacity of the vitreous humour, and the iris periphery is not retracted—points which especially enable us to distinguish it from pseudo-glioma (*vide* Purulent Inflammation of the Vitreous Humour, chap. xiv., p. 389). Secondary glaucoma finally comes on. The optic nerve may become involved at an early period; but, sooner or later, it invariably does so, leading then to glioma of the brain. When the tumour has filled the eyeball, it bursts outwards, usually at the corneo-sclerotic margin, and then grows more rapidly, and often to an immense size, as a fungus hæmatodes. The orbital tissues become involved, and even the bony walls of the orbit, while secondary growths in other organs, more especially in the liver, are not rare.

The diagnosis between glioma of the retina and tubercle of the chorioid (p. 287), when the latter occurs in young children, is sometimes difficult or impossible; but, in view of treatment, not of great importance, as in either case the eye must be enucleated.

Treatment.—The only hope of saving the patient's life lies in enucleation at an early stage, or before the optic nerve becomes diseased. It is important, in removing the eyeball, to divide the nerve as far back as possible; and, if the orbital tissues be already diseased, to remove all suspicious portions of them. Several cases in which there was no return of the growth have been observed.[1]

Parasitic Disease.

Cysticercus under the Retina.—The cysticercus of the tænia solium in the eye is extremely rare in these countries, but not so in Germany. Its most frequent seat is between the retina and chorioid, where it is recognised with the

[1] Lagrange, *Études sur les Tumeurs de l'Œil, etc.*, 1893, p. 160.

ophthalmoscope as a sharply defined bluish-white body, with bright orange margin. At one point of the cyst there is a very bright spot, which corresponds with the head of the entozoon. Wave-like motions along the contour of the cyst should be looked for, to confirm the diagnosis. The cysticercus may move from its original position, and, in so doing, cause considerable detachment of the retina. Delicate veil-like opacities are apt to form in the vitreous humour, and are almost characteristic of the presence of cysticercus.

The entozoon may become encapsuled behind the retina; or it may burst into the vitreous humour (p. 397); and finally, chronic irido-cyclitis, with total loss of sight and phthisis bulbi, is apt to come on.

Treatment.—We are not acquainted with any anthelmintic which will act upon the entozoon in the eye. Removal of the cyst by operation is the only means by which the eye can be saved; and this measure can only be resorted to, when the position of the cysticercus admits of it. By a well-placed puncture through the sclerotic and chorioid the entozoon may then be evacuated.

Detachment of the Retina.

This condition consists in a separation of the retina from the chorioid, the intervening space being occupied by a clear serous fluid. It is not usual to employ the term, when it is a solid neoplasm only that lies between retina and chorioid.

If the media be clear, and the detached portion extensive, the diagnosis is not difficult.

The Ophthalmoscope shows a greyish reflex from a position in front of the fundus oculi, and to the surface from which the reflex is obtained a wave-like motion is imparted, when the eyeball is moved. Over this greyish surface the retinal vessels run, and they serve to distinguish a detached retina from any other diseased condition with a somewhat similar

appearance. They seem black, not red, in consequence of absorption of the transmitted light, and are hidden from view here and there, in the folds of the detached retina. In many cases a rent in the detached retina, through which the chorioid can be discerned, will be discovered.

The detachment may commence in any portion of the fundus, but most commonly above; yet, owing to gravitation of the fluid, it ultimately settles in the lower half of the fundus, and hence this is the most common place to find it, the part first detached having become replaced. The diagnosis is more difficult if there be but little fluid behind the retina, or if there be opacities in the vitreous humour.

Vision is affected according to the position and extent of the detachment. Central vision may be quite normal, if the macula lutea and its immediate neighbourhood are intact. The patients complain of seeing objects distorted, of a black veil which seems to hang over the sight, and sometimes of black floating spots before the eye, due to opacities in the vitreous humour. These symptoms often come on suddenly in an eye which has hitherto had good sight.

The field of vision, on examination, will show a defect corresponding to the position of the detachment. If, for example, it be below, the defect will be in the upper part of the field. If the detachment be fresh, the retina not having yet undergone secondary changes, and if the quantity of subretinal fluid be not great, the defect may only amount to an indistinctness of vision; but, later on, when infiltration and connective tissue degeneration of the detached part come about, fingers may not be counted at the same place. The phosphenes[1] of the detached portion are wanting.

[1] Phosphene is the subjective sensation of light experienced when the eyeball is pressed upon. For clinical purposes it is best tested by gentle pressure with a blunt point (head of a bodkin, or large-sized probe) applied to the eyeball through the eyelid. The phosphene of any region is tested by applying pressure to that part of the globe; thus, if in a

Should the detachment become complete, little more than power of perception of light may be present. Total detachment is followed by cataract, and often by iritis and phthisis bulbi. The detachment may remain stationary, and not extend to the whole fundus, or the retina may return to its normal position. Such a happy result, however, is most rare.

Causes.—Myopic eyes—which we know are so frequently affected with chorioiditis and disease of the vitreous humour—are those most subject to detachment of the retina, but idiopathic detachment occurs also in eyes which are apparently healthy. Blows upon the eye may produce detachment, the retro-retinal fluid being serous or bloody. Some punctured wounds of the sclerotic, also, in the course of healing, by dragging on the retina, give rise to it. Chorioidal tumours, especially those situated in the posterior segment of the fundus, usually cause detachment in an early stage of their growth, and the complication renders their diagnosis more difficult (p. 285).

Leber[1] observed that, in non-traumatic detachment, a perforation, or rent, in the detached portion is very frequently to be seen with the ophthalmoscope, and holds that it is probably always present, although sometimes, from being hidden behind a fold of the retina, it cannot be found. He was led from this, and from his pathological investigations and experiments upon animals, to think that the detachment was due to shrinking of a diseased vitreous, which first became slightly separated from the retina, and that then—at some place where the retina and hyaloid had become adherent from the inflammatory process—a rent was produced in the retina by the shrinking process in the vitreous. And he

healthy eye the individual look down, and pressure be applied to the upper part of the globe through the eyelid, the phosphene will be seen appearing below, but if there be a detachment of the retina at the place pressed on no phosphene is seen.

[1] *Bericht d. Ophthal. Gesellsch.*, 1882, p. 18.

concluded that through this rent the fluid, which is always present behind the vitreous in cases of detachment of that body, makes its way behind the retina, and separates the latter from the chorioid. All this has been fully borne out by Nordenson's pathological researches,[1] who has ascertained, moreover, that disease of the ciliary body and chorioid is the primary cause, although we may not be always able to detect it with the ophthalmoscope, and that the pathological change in the vitreous humour consists in an alteration in its connective tissue elements, resulting in the deleterious shrinking.

Raehlmann,[2] however, from the results of recent experiments, and also from clinical observation, concludes that detachment of the retina is due to exudation from the chorioidal vessels of a fluid, which is more albuminous than the fluid in the vitreous humour. Hence, he thinks, diffusion takes place through the retina, and a greater quantity of the less albuminous vitreous fluid passes through the retina, thus producing and increasing the detachment. Rupture of the retina is not, in his view, a necessary factor in the causation, but it may occur if the tension behind the retina be higher than that in front of it.

Treatment.—Evacuation of the subretinal fluid by puncture of the sclerotic was first proposed by Sichel, and has been cultivated by de Wecker. He uses an instrument like a broad needle, with a sharp point and two blunt edges, which is entered through the sclerotic and chorioid at a place corresponding to the position of the detachment, but not so deeply as to reach the retina, lest thereby it be further displaced. The instrument is then given a quarter of a rotation, to make the wound gape, so as to admit of the flowing off of the fluid. If possible, a position near the equator of the globe, and between two recti muscles, should be selected for the operation. Moreover, the incision should lie parallel to the

[1] *Die Netzhautablösung* (Wiesbaden, 1887).

[2] *Archiv für Ophthal.* xxvii., Part I., p. 1.

direction of the orbital muscles, so that the chorioidal vessels may be injured as little as possible. A firm bandage is applied, and the patient kept in bed for eight or ten days.

The dorsal position in bed, with a pressure bandage on the eye, maintained for from four to six weeks, has produced reposition of the detachment in some cases. The method, if properly carried out, is most trying to the patient.

The few cures which have been reported as accomplished by these means probably depended upon the retina again coming in contact with the chorioid, and, owing to some slight inflammatory process, adhering to it. For the most part, the cure is only temporary, and in such cases we may suppose that no adhesion sprang up, but that the temporary cure was due to a return of the subretinal fluid, through the hole in the retina, to its original position between the retina and vitreous. Soon, however, it makes its way back again through the hole, and the detachment recurs.

Schoeler[1] injects tincture of iodine into the vitreous humour in front of the detached retina, in order to press it back to the chorioid, and to produce a plastic chorioido-retinitis, which may unite the two coats. He has reported several good results by this method, but some who have tried it have experienced violent inflammatory reaction in the eyes operated on, with disastrous consequences.

Grossmann[2] tried aspiration of the subretinal fluid, with simultaneous increase of the pressure in the vitreous humour, by injections into the latter of four or five drops of an indifferent fluid, namely, a 0·75 per cent. lukewarm solution of common salt. The results obtained were encouraging in the three cases treated, but I am not aware of any further reports.

Electrolysis has recently been tried.

---

[1] *Zur operativen Behandlung und Heilung der Netzhautablösung* (Berlin, 1889).

[2] *Ophthalmic Review*, 1883, p. 89.

Galezowski[1] simply aspirates the subretinal fluid.

Pilocarpine used hypodermically has been praised by some as a mode of treatment, as, also, salicylate of sodium internally.

Formerly an active mercurial treatment used to be employed, with the object of obtaining absorption of the fluid.

The Prognosis of every case of detached retina is bad, spontaneous cure being extremely rare, and the treatment of the disease remaining one of the weakest points of ophthalmic therapeutics. Moreover, both eyes are often affected, one after the other. The cures by any one, or by any combination of the above methods of treatment, are few and far between; and when, sometimes, the retina does return to its place, there is still the danger of a recurrence of the detachment. The most favourable cases are those due to chorioiditis, the most unfavourable those due to posterior staphyloma.

Traumatic Affections of the Retina.

In addition to detachment and rupture of the retina, the undermentioned conditions occur as the results of injuries.

Traumatic Anæsthesia of the Retina.—A blow on the eye from a fist, cork from a bottle, etc., is liable to produce considerable amblyopia, with concentric contraction of the field, which may continue for a long time, while *the Ophthalmoscopic Appearances* are normal. Ultimately these cases usually recover, an event which may be decidedly promoted by the use of strychnine hypodermically; but very defective sight sometimes remains permanently.

Commotio Retinæ, or **Traumatic Œdema of the Retina,** is the result of a blow upon the eye. Within a few hours after the accident, *The Ophthalmoscope* reveals a white cloudiness of a portion of the retina, usually in the neighbourhood of the optic papilla and macula, but sometimes more

[1] *Recueil d'Ophthalmologie*, Mars 1888.

eccentrically; and sometimes there are two opaque patches. The opacity increases in intensity, and spreads somewhat. The retinal vessels remain normal; there may be some small hæmorrhages, and sometimes the papilla is redder than normal. These appearances completely disappear in the course of a few days. Vision is only slightly affected, and recovers according as the retinal changes pass off.

CHAPTER XVI.

DISEASES OF THE OPTIC NERVE.

Optic Neuritis.—*The Ophthalmoscopic Appearances* of inflammation of the optic nerve vary a good deal with the intensity of the process. Common to every case is hyperæmia and swelling of the papilla, with haziness ("woolly" appearance) of its margins, and increase in the size of the central vein, while the central artery remains of normal dimensions, or is contracted. The swelling and haziness extend but a short distance into the surrounding retina, and the distension of the vein is also not continued to the periphery of the fundus. In slight cases these appearances may barely exceed the normal.

In extreme instances the papilla is swollen to a great size, and may even assume quite a mushroom shape, while the veins are enormously distended and tortuous, and the arteries are contracted so as to be barely visible. Greyish striæ, also, extend from the papilla into the surrounding retina, some flame-shaped hæmorrhages are present on or near the papilla, and, occasionally, white spots in the retina, and a stellate arrangement of small white dots about the macula lutea produce an appearance which cannot be distinguished from albuminuric retinitis. This extreme form is still sometimes termed Congestion Papilla, or Choked Disc (*Stauungspapille*), although the theory which originally suggested the term has been abandoned. Papillitis (Inflammation of the Optic Papilla) is a better term, expressing, as it does, more truly the pathological condition.

The Vision, even in cases where the ophthalmoscopic signs are highly developed, is frequently but little below the normal;

while again in other, and possibly less well-marked cases, it may be reduced to perception of light, or even that may be wanting. When due to cerebral tumour the neuritis appears as a rule before the vision becomes affected. These remarkable differences in the degree of blindness depend, probably, on the extent to which the nervous elements of the inflamed part are pressed on or altered, and this cannot be gauged by the ophthalmoscopic appearances.

Sometimes the field of vision is normal, while again it is concentrically or irregularly contracted, or it may be hemianopic.

Attacks of temporary loss of sight, sometimes called epileptiform amaurosis, is a very common symptom in cerebral tumours; it may occur several times a day, and may last from a few minutes to half an hour.

Pathologically, the changes in the papilla consist in venous hyperæmia, œdema, hypertrophy of the nerve fibres, infiltration of lymph cells, and development of connective tissue. Inflammatory changes, although less pronounced, are also present in the trunk of the nerve and its sheaths.

Causes.—Inflammation of the optic nerve is most commonly found in connection with coarse encephalic disease. A Cerebral Tumour (syphiloma, tubercle, glioma, and abscess), in particular, is the most common cause, and is, moreover, usually present when the papillitis is of an intense kind (choked disc). The neuritis, except in very rare instances, is bilateral, and is one of the general symptoms. Hemianopsia may co-exist as a localising symptom if the visual centre or fibres on one side be involved. A very small tumour situated anywhere[1] in the brain is capable of pro-

[1] Hughlings Jackson (*Trans. Ophthal. Soc.*, vol. i., p. 79), states that optic neuritis has not been noted with tumours of the medulla oblongata. Edmunds and Lawford (*Trans. Ophthal. Soc.*, vol. iv., p. 185) find that tumours of the cortical motor area do not commonly produce neuritis, while it is very frequent and severe in tumours of the cerebellum.

ducing optic neuritis, although unattended by meningitis. Cerebral cysts do not often cause it.

Tubercular Meningitis is the next most common cause. Non-tubercular meningitis occasionally gives rise to optic neuritis, and sometimes, also, cerebro-spinal meningitis does so.

Optic neuritis is occasionally associated with acute myelitis, so that inflammation of the optic nerve, with paralytic phenomena, does not exclusively indicate cerebral disease.

The Connection between Optic Neuritis and Intracranial Diseases has given rise to much discussion. In cases of tumour, as well as of tubercular meningitis, a considerable exudation of fluid usually takes place into the cavity of the third ventricle. This, along with the pressure of the new growth, or, alone in cases of meningitis, increases the intracranial pressure. By reason of this increased pressure, the subarachnoid fluid is believed to be driven into the subvaginal lymph space of the optic nerve, and to produce there that dropsy of the sheath, which is found in nearly all these cases on careful *post-mortem* examination.

Leber holds[1] that this fluid is probably an irritant, and, as such, sets up the inflammation, a view which has been corroborated by Deutschmann.[2]

The inflammation, although most intense at the papilla, near which the fluid is collected in greatest quantity in the *cul-de-sac* formed by the termination of the intervaginal spaces, is not confined to that place, as was believed, but extends up the trunk of the nerve, as microscopic examination reveals.

Many observers[3] state that, in a large number of cases, cerebritis, recognisable only with the microscope, is present,

[1] *Trans. Internat. Med. Congress*, 1881, vol. iii., p. 52.

[2] *Ueber Neuritis Optica* (Jena, 1887).

[3] S. Mackenzie, *Brain*, vol. ii., p. 257. W. Edmunds, *Trans. Ophthal Soc.*, vol. i., p. 112. Brailey, *Trans. Internat. Med. Congress*, 1881, vol. ii., p. 111.

and that an extension of this process down the optic nerve takes place. They have ascertained that the whole trunk of the nerve is involved in the inflammation, and they seem to regard the dropsy of the sheath as of little or no importance in the causation of the optic neuritis.

Again, others [1] maintain that œdema, but not inflammation, of the optic trunk is conducted from the brain.

The view originated by von Graefe, that the extreme form of papillitis, called by him *Stauungspapille* (Choked Disc), is due to obstructed outflow of blood through the retinal vein, is now abandoned.

Other causes for Optic Neuritis are—

Hydrocephalus.—Here the pathogenesis is probably the same as in the foregoing, but the occurrence of optic neuritis is, on the whole, not very common in this connection.

Tumours of the Orbit.—The path by which these growths bring about papillitis is still unknown.

Inflammatory Processes in the Orbit, such as caries, inflammation of the retro-orbital areolar tissue, erysipelas of the head and face extending to the orbital tissues, and periostitis. The presence of the latter may often be recognised by pain on motion of the eyeball, pain in the eye and forehead, and especially by pain on pressure of the globe backwards, and is frequently of rheumatic origin. Often in these cases one eye only is affected; and, although *The Ophthalmoscopic Appearances* are sometimes very slight, yet vision may be quite lost in a few hours or days, atrophy of the nerve then rapidly setting in.

Very many of the cases, however, do not go on to atrophy, but end in recovery of useful vision.

Exposure to Cold, especially if the skin be heated and perspiring.

Suppression of the Menstruation.—If, during the menstrual period, the flow be arrested by exposure to cold, wet

[1] Ulrich, *Archives of Ophthal.*, xviii., p. 65.

feet, etc., acute optic neuritis with rapid blindness may come on. Spontaneous amenorrhœa, or even irregularity of menstruation, and the climacteric period are liable to have a similar, but more chronic, result. Nothing is definitely known with regard to the connection between the uterine and ocular disorder; but it is believed that the latter is due to "determination of blood" taking place to the base of the brain. instead of to the uterus. In these cases *the Ophthalmoscopic Appearances*, as well as the blindness, are apt to be extreme. *Treatment* here should be directed chiefly to restoring the normal uterine functions. Hot foot-baths and Heurteloup's leech to the temples are of use.

Chlorosis.—Here optic neuritis often is present, due to the disordered state of the blood, and usually yields under the influence of iron.

Syphilis.—The trunk of one or both optic nerves may be the seat of specific inflammation in connection either with congenital or with acquired syphilis, but this primary specific optic neuritis is a relatively rare disease. In cases of acquired syphilis it makes its appearance in from six months to two years after the inoculation.

The Ophthalmoscopic Appearances may be normal (retrobulbar neuritis), or may present any grade of neuritis, even to the most pronounced papillitis. In the latter case it would not be possible to say whether the papillitis be a primary one, or be due to a syphilitic gumma within the cranium. The inflammation often extends as far up as the chiasma.

The Treatment in these cases of specific papillitis must be active mercurialisation. By this treatment, even if perception of light be lost for a period of not more than eight to fourteen days, hopes may be entertained of its complete or partial recovery. Later on, iodide of potassium is indicated.

Cases of double optic neuritis of syphilitic origin have been observed, in which complete recovery took place, the papilla returning to its normal condition.

But, as a rule, some optic atrophy, at the least, with slight concentric contraction of the field, results. The prognosis is all the better the sooner the optic neuritis follows upon the primary syphilitic affection.

Rheumatism.—There is no doubt whatever but that the rheumatic diathesis is occasionally the cause of optic neuritis, although the fact is not unreservedly accepted by every author. Other manifestations of rheumatism are sometimes well marked, but may be slight, *e.g.*, in a case which I saw, neuralgia of the face and head in damp weather, and even with a shower of rain, was the only other sign of rheumatism. One or both optic nerves may be attacked.

The Ophthalmoscopic Appearances often amount to extreme papillitis, but in many cases fall short of this.

If the case come early under suitable treatment, *The Prognosis* is fairly favourable; but when the inflammation is of some standing, consecutive optic atrophy must be feared.

The Treatment consists of full doses of salicin, salicylate of sodium, iodide of potassium or of sodium, Turkish baths, and other recognised anti-rheumatic measures.

Lead-Poisoning.—In some cases of lead-poisoning, optic neuritis, not to be distinguished from that of primary cerebral affections, is found. Sometimes *The Ophthalmoscopic Appearances* are very slight, and again quite pronounced, the changes extending into the retina, and simulating the retinitis of Bright's disease, and, in such cases, renal disease is likely to have much to do with the causation of the retinitis. Indeed, there are those who, with good opportunities for forming a correct opinion, deny the existence of a specific lead neuritis, and hold that the neuritic affection in all such cases is to be referred to albuminuria, to effusion into the ventricles of the brain and subarachnoid space, or to suppression of menstruation. Occasionally, optic atrophy is the first ophthalmoscopic appearance seen but it is probably consecutive to retro-bulbar

neuritis, as shown by white striæ (perivascularitis) along the vessels.

The Vision is often much affected, and it is stated that sudden complete blindness in connection with an intercurrent attack of lead colic may appear, and pass off again. Consecutive atrophy is liable to come on, and then vision may be seriously and permanently damaged.

The Diagnosis depends entirely on the presence of the other well-known symptoms of lead-poisoning, the ophthalmoscopic appearances presenting nothing pathognomonic.

The Treatment is that for general lead-poisoning, or for the immediate cause of the neuritis.

In Peripheral Neuritis, optic neuritis is occasionally found.

Multiple Sclerosis.—In these cases the inflammation is very ephemeral, and rapidly gives place to atrophy. Uhthoff states that it occurs in about 13 per cent. of the cases of this disease.

Hereditary and Congenital Predisposition.—It has been observed that optic neuritis, without immediate cause, may attack several members of a family, and that the tendency to it may extend over several generations. It makes its appearance in these instances about the eighteenth or twentieth year of age, and confines itself almost exclusively to the males. The patients may be perfectly healthy in all other respects, but many of them suffer from other affections of the nervous system. Both eyes are affected, the defect of vision being a central amblyopia, from which recovery is rare; but yet, although the ophthalmoscopic appearances gradually become those of atrophy, the peripheral portions of the field retain their functions.

As to *The Treatment* of these cases, due to hereditary and congenital predisposition,[1] Mooren employs a seton in the back

[1] Mooren, *Ophthal. Bericht.*, 1867, p. 305, and 1874, p. 87; and *Fünf Lustren Ophthal. Wirksamkeit*, 1882, p. 248. Norris, *Trans. Amer. Ophthal. Soc.*, 1884, p. 662.

of the neck in the early periods, and, later on, nitrate of silver internally. Leber has found benefit from a mild course of mercurial inunction.

Optic Neuritis also occurs occasionally in fevers; it has been observed in Measles, Scarlatina, Typhoid, and Malaria. It may follow Influenza, causing contraction of the field of vision or central scotoma which usually disappear, but, on the other hand, it may lead to optic atrophy.

The two following diseases, Chronic Retro-bulbar Neuritis or Central Amblyopia, and Optic Neuritis with Persistent Dropping from the Nostril, must be treated of separately, owing to the well-defined etiology of the one, and the peculiar symptoms of the other.

Chronic Retro-bulbar Neuritis, or **Central Amblyopia** (Toxic Amblyopia).—Until within recent years, it was not clearly known whether these two terms should be applied to one and the same disease, or whether we had to deal here with two distinct processes. There is a class of cases which were admittedly due to an inflammatory process in the trunk of the nerve, the causes and symptoms of which were very similar to those of central amblyopia; while there was strong presumptive evidence that the latter affection, often known as Toxic Amblyopia, was due to a retro-bulbar inflammation; yet direct proof of the fact was wanting. But there is now no doubt, that we have here to deal with only one disease.[1]

Symptoms.—The affection of vision often comes on rather rapidly. The patient may complain of a glimmering mist which covers all objects, especially in a bright light, and the acuteness of vision is reduced. The patient generally states he can see better in the dusk than in bright light. At the commencement there is no defect in the field of vision, but

[1] Samelsohn, *A. v. Graefe's Archiv*, xxviii., pt. 1, p. 1. Nettleship and Walter Edmunds, *Trans. Ophthal. Soc.*, vol. i., p. 124. Uhthoff, *von Graefe's Archiv*, xxxii., p. 4. Sachs, *Archiv of Ophthal.*, xviii., p. 133.

simply a general dimness of vision. At a somewhat later stage, examination of the field discovers no defect for a white object; yet, if a small pale green object be employed, it will generally be ascertained that, at a region close to the point of fixation, the colour is not recognised, but seems grey or white; pink may seem blue, and red may appear brown or black; while in other parts of the field the colours are recognised up to their normal boundaries. This is a central colour-scotoma. As the disease advances, a white object will be but indistinctly seen in the scotoma; and, in some rare cases, all power of perception within its area may be lost, even the flame of a candle not being recognised. Hence the name Central Amblyopia. The scotoma is usually oval in shape, its long axis horizontal, and extends from the fixation-point towards the blind-spot of Mariotte (paracentric scotoma); but occasionally it is of much larger dimensions, and sometimes surrounds the fixation point (pericentric scotoma).

Even when the scotoma is very pronounced, it remains "negative," *i.e.*, it is not observed by the patient as a dark spot in the field, as is a scotoma due to disease in the outer retinal layers. The affection is almost always binocular, and, as a rule, there is but little difference between the vision of the two eyes.

The Progress of the disease is slow, occupying weeks or months. Restoration of normal vision usually takes place, if the defect of vision, although of extreme degree, be not of old standing. In the latter case, while recovery of central vision cannot be expected, the functions in the periphery of the field are usually maintained; and, consequently, these people, although incapacitated from reading, writing, and other fine work, do not lose their power of guiding themselves.

Causes.—With but few exceptions, the subjects of this disease are men, probably because their habits and modes of life expose them, more than women, to the influences which produce it. These are exposure to cold and wet; cold

blasts on the body, especially the heated face (Samelsohn); but the most common cause is excess in the use of alcohol, or of tobacco (toxic amblyopia), or of both. I think the kind of alcoholic indulgence most likely to develop the disease is the frequent drinking of small doses of the stimulant. The individual who often gets thoroughly drunk, and between times drinks but little, is less liable to central amblyopia, than he who, although never incapable of transacting his business, takes many half-glasses of whisky or brandy during the day. Dyspepsia, and loss of appetite, are constantly present in these cases. Other signs of chronic alcoholism need not be present, but one often sees trembling of the hand and head, sleeplessness, and even delirium tremens. The kind of tobacco most likely, when used in excess, to give rise to central amblyopia, is shag or twist. Other kinds of pipe-tobacco and cigars may cause it, but I have not known of a case due to cigarette-smoking.

Excess in alcohol is usually combined with excessive smoking, but cases of pure alcohol-amblyopia certainly do occur—although some English authors deny it—as well as pure tobacco-amblyopia. The most common age for tobacco-amblyopia is from thirty-five to fifty, a time of life when men do well to give up, or to very much reduce, their use of tobacco, as well as of alcohol.

Central amblyopia has also been observed in diabetes, in poisoning from bisulphide of carbon,[1] so largely used in the manufacture of indiarubber, from dinitro-benzol,[2] used for explosives, and in iodoform poisoning.[3]

Retro-bulbar neuritis very occasionally attends disseminated sclerosis, with atrophy of the temporal side of the papilla; but yet central scotoma is not always present here.

[1] *Trans. Ophth. Soc.*, vol. v., p. 149.

[2] S. Snell, *Brit. Med. Journ.*, March 3rd, 1894.

[3] P. Smith, *Ophthal. Rev.*, 1893, p. 101; and Valude, *Revue d'Ophthal.* 1893, p. 231.

The Ophthalmoscopic Appearances in the beginning are either quite normal, or there is slight hyperæmia of the papilla and retinal vessels; or, in addition, there may be slight indistinctness of the margins of the papilla, and sometimes white striæ along the vessels, especially before they leave the papilla. All the primary appearances, if any be present, soon pass away, and give place to a greyish whiteness of the temporal side of the papilla, while the nasal portion remains of normal appearance, as do also the vessels. At a very advanced stage, in some cases, the whole papilla presents the appearance of white atrophy.

The Pathological Changes observed by Samelsohn, Nettleship and Walter Edmunds, and Uhthoff, in the optic nerve, consist of an interstitial neuritis at its axis, commencing so high up as the optic foramen, and leading to proliferation of connective tissue, and to secondary descending atrophy of a certain bundle of nerve fibres. These are, doubtless, the fibres which supply the region of the macula lutea. The changes are analogous to those, which take place in the liver and brain as the result of chronic alcoholism.

Treatment consists, above all, in total abstinence from the poison in question. The patients are generally ready to promise this, but they often do not act up to their intentions. When they do so, improvement rapidly takes place in most cases which are not too far gone, without any other treatment; but the cure may be promoted by the use of iodide of potassium in large doses, Heurteloup's artificial leech, or dry cupping, to the temples, hot foot-baths, and Turkish baths. Strychnine hypodermically ($\frac{1}{30}$ grain daily) in the temple is often of use, and phosphorus and strychnine may be given internally. Whatever remedy be used internally, care should be taken that it does not produce or increase dyspepsia; and it may be necessary to restrict the internal medicine for a time, or altogether, to a stomachic tonic. Sleeplessness should be combated with chloral and bromide of potassium. Treatment

may have to be continued for some weeks before a cure can be noted.

Optic Neuritis associated with Persistent Dropping of Watery Fluid from the Nostril.

Nettleship [1] (one case), Priestley Smith [2] (two cases), Leber [3] (one case), and Emrys Jones [4] (one case), have placed on record five well-observed cases of this remarkable disorder, and three others have been observed by Elliotson, Baxter, and Paget. These patients suffered from a persistent watery discharge from the nose (usually the left nostril), with more or less severe cerebral symptoms—violent headache, epileptic attacks, vomiting, stupidity, sleepiness, unconsciousness, delirium, weakness of the lower extremities, and a high degree of amblyopia, or even blindness, of both eyes, due to papillitis followed by atrophy. In Leber's case, moreover, there was loss of smell, and in Nettleship's case palpitation of the heart and prominence of the eyes. The fluid which ran from the nostril was, according to Leber, identical in its analysis with that of the cerebro-spinal fluid, while Nettleship and Priestley Smith found it to differ somewhat from that fluid. When, in P. Smith's case, it occasionally ceased to flow, the cerebral symptoms were brought on, or increased in violence. Leber's case was one of internal hydrocephalus, and he regards the fluid as coming from the third ventricle through a small opening in the ethmoid bone; or, the fluid possibly passed from the subdural space along the lymph spaces, which, according to Axel Key and Retzius, surround the olfactory nerves. But Priestley Smith and Nettleship considered the fluid as simply nasal, and due to the presence of small polypi, and did not try to account for its occurrence in connection with the cerebral and ocular symptoms. The first of these two views is probably the correct one.

[1] *Ophthal. Rev.*, 1883, p. 1.
[2] *Ibid.*, 1883, p. 4.
[3] *A. v. Graefe's Archiv*, xxix., pt. 1, p. 271.
[4] Meeting British Med. Assoc., Dublin, 1887.

The Prognosis for vision is bad, while the cerebral affection threatens even the life of the patient.

Treatment, which should be in conformity with the head symptoms, has not proved of use.

Atrophy of the Optic Nerve.—This disease may be secondary to some other optic nerve or retinal affection, or it may be a primary disease. *The Vision* is seriously affected, and complete blindness is the usual result. With *The Ophthalmoscope* the optic papilla is seen to have lost its delicate pink colour, and to have become white or greyish, while it is often cupped, and the vessels are apt to be diminished in calibre.

Secondary Atrophy of the Optic Nerve may result :—

1. *From Optic Neuritis.*—The ophthalmoscopic appearances consist in a white or greyish-white colour of the papilla, with very diminished retinal vessels ; and, along both sides of the vessels, far into the retina, are seen white lines, which sometimes even obscure the vessels, and which are due to hypertrophy of their coats. The diminution in calibre of the vessel is a sign of neuritic atrophy, but is not always present, and is moreover found with other forms of atrophy. Other signs of this form, also not constant, are :—a certain opacity of the papilla, and that the lamina cribrosa is not generally visible, owing to development of connective tissue at the papilla. It is, evidently, not always possible to recognise any given case as of neuritic origin. This form of atrophy is commonly called "consecutive atrophy."

Symptoms.—Central vision is lowered, and, as a rule, the field of vision becomes contracted, usually more at the nasal side. Subsequently, the temporal side of the field becomes contracted, and, finally, a small eccentric portion of the field to the temporal side may be all that remains, or even this may disappear and absolute amaurosis result. The colour-vision is always much affected. The light-sense is affected, so that there is diminished sensibility for differences of illumination ; while, in chorioido-retinal diseases, there is defect in the quantitative

perception of light, the minimum quantity being larger than normal.[1]

2. *From Pressure.*—This may be brought about by a tumour anywhere in the course of the nerve, by inflammatory exudations, by a splinter of bone in cases of fracture of the skull, and, also, by pressure upon the chiasma by the floor of the distended third ventricle, in cases of internal hydrocephalus.

3. *From Embolism of the Central Artery of the Retina.*—In these cases the contraction of the vessels is usually extreme.

4. *From Syphilitic Retinitis, Retinitis Pigmentosa, and Chorioido-retinitis.* The vessels here are much attenuated, and the altered colour of the optic disc is a dull yellow, rather than white or grey.

Primary Optic Atrophy is often found associated with Disease of the Spinal Cord (*Spinal Amaurosis*), especially locomotor ataxy. Optic atrophy is often an early symptom in the latter disease; but, again, it may not come on until the affection of the gait is well pronounced; while, in other cases, it is never present at all. It is a remarkable and important fact, first pointed out by Benedikt of Vienna, that there is an antagonism between atrophy of the optic disc and the other symptoms of tabes dorsalis. It is rare for a tabetic patient, in whom optic atrophy comes on in an early stage of his disease, to become ataxic; and frequently, in these cases, when the blindness has advanced, the pains, too, become less severe. But, if amaurosis do not come on until the ataxy be well developed, no improvement in the latter is likely to be noted.

Atrophy is found more rarely with insular sclerosis and lateral sclerosis of the spinal cord; and in general paralysis of the insane, although spinal disease is not always present, atrophy of the papilla frequently occurs.

It is probable that the disease commences at the papilla in

[1] Bjerrum, *v. Graefe's Archiv*, xxx., pt. 2, p. 201.

spinal cases. The ophthalmoscope displays a papery-white or bluish-white papilla, which, in advanced stages, often becomes cupped. The retinal arteries are usually extremely reduced in calibre, and the veins, too, may be small; but, again, the retinal vessels may differ but little, or not at all, from the normal.

Symptoms.—Central vision is affected at an early stage in the disease, and eccentric contraction of the field usually appears at the same time. The contraction may be concentric, or it may be more marked in one direction than another, and opinion is divided as to the direction commonly first involved. This concentric contraction advances gradually towards the centre of the field from every side, until it finally engulfs the fixation point.

Occasionally the affection begins as a central scotoma, accompanied by eccentric defects of the field. Colour-blindness is an almost constant symptom. As a rule, absolute blindness is brought about in the course of a year or two.

Primary Optic Atrophy, of the progressive form just described, may occur, *As a Purely Local Disease*, without any other defect in the system. The prognosis for the sight in such cases is as bad as in spinal cases.

Treatment.—In neuritic atrophy, so long as there are still signs of active inflammation, antiphlogistic measures—Heurteloup's leech to the temple, hot foot-baths, rest of body and mind, dark room, iodide of potassium, and, especially, mercury internally, when otherwise admissible—are to be adopted. At a later period, hypodermic injections of strychnia ($\frac{1}{30}$ gr., increased gradually to $\frac{1}{20}$ or $\frac{1}{18}$ gr. once a day), and galvanism, may be tried. Hypodermic injections of antipyrin (about $7\frac{1}{2}$ grains every second day) have been recently given with some benefit in these cases.[1]

In spinal amaurosis, and in optic atrophy occurring as a local disease, strychnia hypodermically and the galvanic

[1] Valude, *Annales d'Oculist*, 1893, p. 161; and 1894, p. 68.

current sometimes improve vision for a time. Phosphorus internally may be given.

The treatment for optic atrophy, due to causes 2, 3, and 4, is to be directed to the primary disease.

The Prognosis is very serious; for, although every therapeutic measure may have been employed, amaurosis is the ultimate result, as a rule.

Tumours of the Optic Nerve.—Ninety-four cases of this rare affection have been recorded.[1] It occurs at all ages, but 75 per cent. of the patients are under twenty years of age. The tumours are generally situated about the centre of the nerve, and do not reach to the ocular end. The symptoms are:—Slow and gradually increasing protrusion of the eyeball forwards and outwards, with retention of its motion, and without displacement of its centre of rotation. The tumour is sometimes soft, so that the eyeball can, as it were, be pushed back into it; but, in any case, pressure does not cause pain. The sight is usually very defective, or quite lost, through optic neuritis or atrophy. The pupil reacts consensually. The tumour may be felt by palpation.

These tumours are either myxosarcomata, or, less frequently, endotheliomata, and are usually encapsuled by the sheath of the nerve. They are benign, in the sense that they do not lead to glandular enlargements, or to metastases; but, in rare cases, they extend into the cranial cavity.

Treatment.—It is sometimes possible to remove such a tumour, and yet to preserve the eyeball, by dislocating the latter during the operation. As a rule, it is necessary to enucleate the eyeball in order to reach the tumour; and, if the growth have involved the surrounding orbital tissues, these, too, must be taken away. Braunschweig, in some cases, made a flap containing a wedge-shaped piece of the outer wall of the orbit, in order to facilitate access to the back of the orbital cavity.

[1] Braunschweig, *von Graefe's Archiv*, xxxix., pt. 4, p. 1.

Hyaline, or Colloid, Outgrowths from the optic papilla, are occasionally met with. They present the appearance of bluish-grey, mulberry-like nodules. According to Iwanoff[1] they originate in the lamina vitrea of the chorioid at the margin of the papilla, or within the area of the papilla; for the lamina vitrea is often prolonged into the papilla, and takes part in the formation of the lamina cribrosa. But Gurwitsch[2] disputes this view, and states that these growths spring from the coats of the vessels in the optic papilla. These outgrowths do not always cause a defect of sight, and rarely cause serious blindness. It is often found that a blow upon the eye has been received some time previously, and it is probable that such a trauma may have to do with the growth, by rupturing the very brittle lamina vitrea.

Injuries of the Optic Nerve.—In addition to those injuries which result from direct violence with sharp instruments, etc., entering the orbit, the optic nerve may be injured in falls on the head. Fractures of the base of the skull frequently involve injury to the optic nerve. But, even where no fracture occurs, blindness with atrophy of the optic nerve may come on, usually only in one eye, and in these cases concussion of the nerve at its passage through the optic foramen, or an extravasation of blood in the sheath of the nerve, is probably the direct cause of the atrophy.

Hæmorrhages from the Stomach, Bowels, or Uterus are capable of giving rise to serious and incurable blindness.

Blindness during, or immediately after, a severe hæmorrhage is probably due to insufficient blood-supply to the nerve-centres and retina, accompanying general exhaustion of the system. For such cases the prognosis is favourable.

But there is another class of cases of very much more serious import. In these the defect of vision does not come on for from two to fourteen days after the hæmorrhage, when

[1] *Klin. Monatsbl. f. Augenhlk.*, vi., p. 425.
[2] *Centralbl. f. Augenhlk.*, Aug. 1891.

the general system is recovering. Even comparatively slight hæmorrhages, which caused no marked anæmia, are said to have been followed by blindness. The connection between the loss of blood and of sight in these cases is not yet clearly made out. Leber inclines to the belief that the blindness here is due to an extravasation of blood at the base of the skull, and into the sheath of the optic nerve; but, even then, the relationship between this and the stomachic or uterine hæmorrhage is not made clearer. Papillitis has been several times noted with the ophthalmoscope in these cases; and this circumstance makes it probable that neuritis is the immediate cause of blindness—even in those cases which show no ophthalmoscopic sign of it—and hydræmia may be presumed to be the poisonous influence which calls forth the neuritis.

The Defect of Vision may be but slight, or it may amount to absolute amaurosis. Both eyes are usually affected in equal degree; but cases have been observed in which one eye was completely amaurotic, while the vision of the other eye was quite normal, and one such case is sufficient to prove that the lesion is peripheral; in fact, that it lies in each instance on the distal side of the optic chiasma. The field of vision is frequently contracted, either concentrically or segmentally; and, even when central vision recovers, the field may remain contracted.

The Ophthalmoscopic Appearances which are present immediately on the occurrence of the blindness have not as yet been observed. A few weeks later they have been found to be different in different cases. They have been found, at this period, normal; or, presenting slight paleness of the papilla and contraction of the arteries; or, there was marked paleness of the papilla, and the arteries were extremely contracted, with slight distension of the veins; or, paleness of the papilla was present, but its margins were indistinct, and the surrounding retina somewhat swollen, while the retinal vessels were normal. Small hæmorrhages have repeatedly been seen in the neigh-

bourhood of the papilla. At later periods, well-marked optic atrophy is frequently observed.

Prognosis.—If, in the beginning, the defect of vision be merely amblyopia and not complete blindness, hopes may be entertained of marked improvement, or of complete recovery. But Mooren has seen slight amblyopia pass into permanent amaurosis.

Hæmorrhages from the stomach are those which are followed by the most complete and permanent blindness, while uterine hæmorrhages are more commonly followed by less serious degrees of blindness.

The Treatment must consist of internal remedies calculated to correct the general anæmia, such as iron, beef tea and meat extracts, wine, etc. Strychnine hypodermically, to stimulate the nerve, may be employed.

CHAPTER XVII.

AMBLYOPIA AND AMAUROSIS[1] DUE TO CENTRAL AND OTHER CAUSES.

Hemianopsia (ἥμισυς, *half*; *a*, *priv.*; ὄψ, *the eye*).—This term implies a loss of sight in one half of the field of vision, usually of each eye, consequent upon a lesion at the centre of vision, at the chiasma, or at some point in the course of the visual fibres between these two placcs. It is not used for cases in

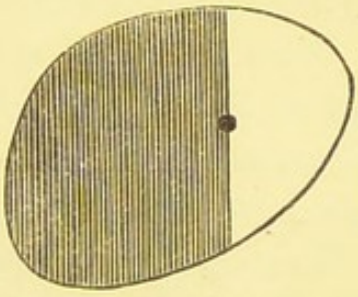


FIG. 131.

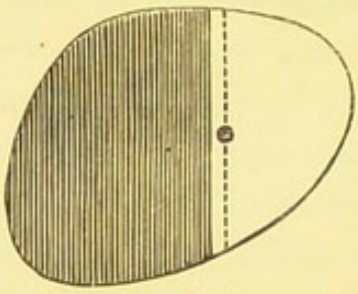


FIG. 132.

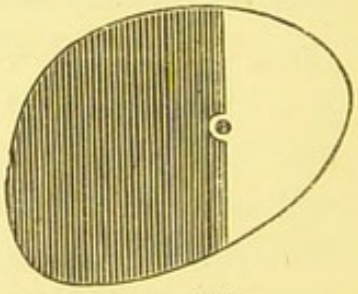


FIG. 133.

which one-half of the field is lost, owing to disease within the eye itself.

The line dividing the seeing from the blind half passes vertically down the centre of the field, as in Fig. 131. Sometimes this line lies a little to one side of the centre of the field, so as to admit of the latter being included in the

[1] These terms have been handed down to us from the old writers. Amblyopia (ἀμβλύς, *blunt*; ὄψ, *the eye*) is nowadays usually employed to signify defective vision due to disease or functional disturbance of the retina, optic nerve, or visual centre, but with healthy ophthal moscopic appearances, or with signs only of optic atrophy. Amaurosis (ἀμαυρός, *dark*) means total loss of sight, with similar ophthalmoscopic appearances. Yet, the use of these terms will be found sometimes to exceed the definitions here stated.

seeing part, as in Fig. 132; and sometimes—although in other respects the dividing line lies in the centre of the field—the fixation point is circumvented by it, so as to leave that point free, as in Fig. 133; and, probably, this is the most common arrangement. This subject will be further discussed on p. 456. Again, although rarely, the dividing line may have an oblique direction, as in Fig. 134. It is probable that such a field as Fig. 134 is due to some peculiar arrangement in the decussation of the nerve fibres in the individual case. Furthermore, cases occur which are properly regarded as hemianopsia, and yet in which only a sector of one side of the field is wanting, as in Fig. 135. Figs. 131, 132, 133, and 134

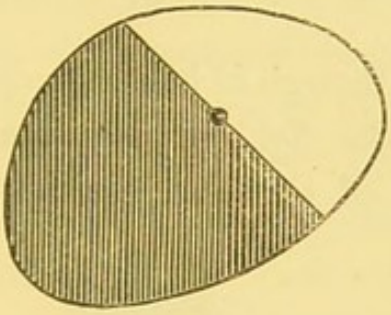


FIG. 134.

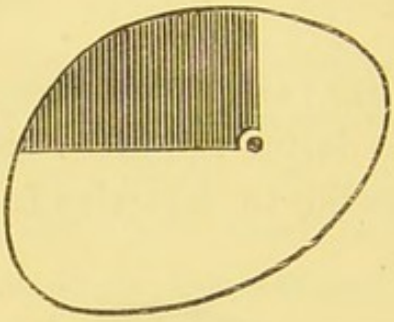


FIG. 135.

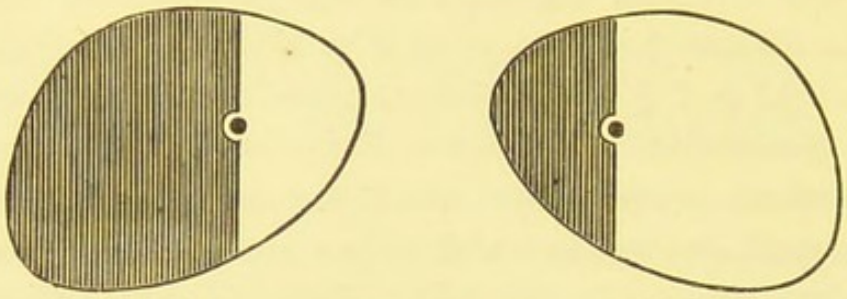
would be called complete hemianopsia, while Fig. 135 would be termed incomplete, or partial, hemianopsia. Finally, if all three visual perceptions be lost, the hemianopsia is called absolute; but, if only one (colour) or two (colour, and form) be wanting in the defective part of the field, it is termed relative hemianopsia. The vast majority of cases of hemianopsia are absolute.

Homonymous Hemianopsia is the most frequent form. In it the corresponding half—the right half or the left half—of the field of each eye is wanting, as in Fig. 136, in which the left side of the fields, from the patient's point of view, is blind, implying a loss of function in the right half of each retina.

Temporal Hemianopsia is loss of vision in the outer side of each field, in consequence of loss of power in the median half

of each retina (Fig. 137). It is by no means so common as the homonymous form.

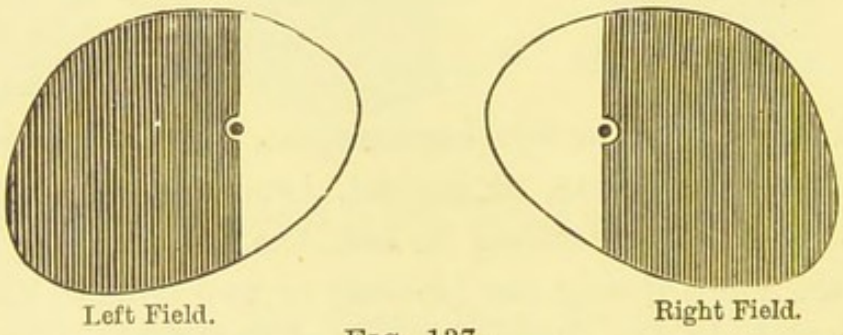
Superior or Inferior Hemianopsia, also called Altitudinal Hemianopsia, in which the upper or lower half of the field is blind, is very rare; and it is doubtful whether Nasal Hemian-



Left Field. Right Field.

FIG. 136.

opsia has really been observed, although it has been described. In the latter the inner side of the field of one eye only is lost, owing to defective function of the temporal side of the retina.



Left Field. Right Field.

FIG. 137.

It will be convenient here to set forth the prevailing views as to

The Arrangement of the Cortical Visual Centres, their relations to the Retina, and the Course of the Optic Fibres between these two points.

Pathological anatomy leaves little doubt but that, in man, the visual centre is situated on the mesial surface of the occipital lobe, rather than in the angular gyrus or elsewhere; and the evidence goes to show, that the absolute optical

centre chiefly occupies the cortex of the cuneus and of the superior occipito-temporal convolution.

Henschen,[1] as the result of clinico-pathological investigations, believes it to be situated in the middle part of the calcarine fissure, which lies between these structures; and that the upper, or cuneic, lip represents the homonymous dorsal retinal quadrants; while the lower, or lingual, lip represents the homonymous ventral quadrants of the retina. Vialet,[2] on the other hand, thinks that the visual centre embraces all the mesial surface of the occipital lobe included between the occipito-parietal fissure and the lower border of the third occipital convolution, and that it extends above and behind as far as the free border of the hemisphere. The calcarine fissure he, however, also regards as of great importance[3]; and I think, indeed, he rather proves than disproves Henschen's view of its middle third being the actual cortical centre for vision.

It is universally recognised that the nerve fibres from the homonymous half of each retina, *e.g.*, from the temporal half of the right and from the median half of the left retina, pass wholly through the corresponding optic tract,—in this case the right tract,—to the corresponding cortical centre for vision (Fig. 138, p. 454).

A case published by Hun,[4] in which the left lower quadrant in each field was blind, and where the autopsy showed a lesion (atrophy) strictly limited to the lower half of the right

[1] *Klinische und Anat. Beiträge zur Pathol. des Gehirns*, Upsala, 1890—92.

[2] Vialet, *Les Centres Cerebraux de la Vision*, Paris, 1893.

[3] One of the most important cases which has been published in connection with this question is that of Dejerine and Vialet (Societé de Biologie, Paris, Dec. 1893), in which both eyes became suddenly blind, without loss of consciousness or other symptoms. The patient lived for a long time, and after death from pneumonia the post-mortem showed lesions of the structures bordering the calcarine fissure on both sides.

[4] *American Journal of the Med. Sciences*, Jan. 1887.

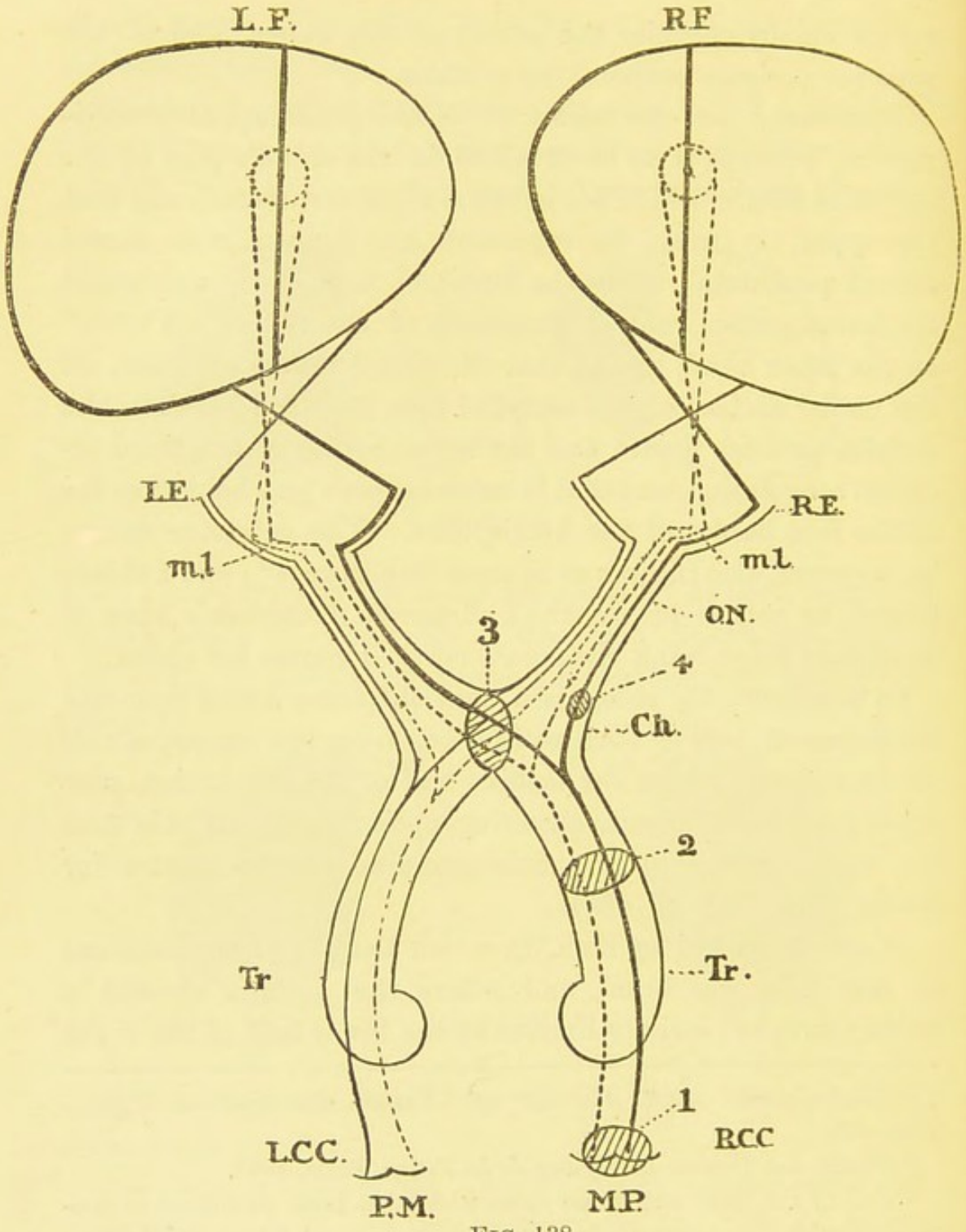


FIG. 138.

EXPLANATION OF FIG. 138.

FIG. 138.—Diagram of Course of Optic Fibres, with the Cortical Centres and Relations to Fields of Vision, to illustrate one theory of the Macular Supply; according to which the macula is supplied on the same plan as the rest of the retina, *i.e.*, each side of it from the corresponding side of the brain.

R.F., Right field of vision; *L.F.*, Left field of vision; *R.E.*, Right eye (retina); *L.E.*, Left eye (retina); *ml.* and *ml.*, Macula lutea; *O.N.* and *O.N.*, Optic nerves; *Ch.*, Chiasma; *Tr.* and *Tr.*, Optic tracts; *R.C.C.* and *L.C.C.*, Right and left cortical centres; *M.* and *M.*, Macular fibres; *P.* and *P.*, Peripheral fibres.

1. Lesion of right cortical centre = left homonymous hemianopsia, the line of demarcation passing round the left side of the fixation point in cases of embolism and thrombosis, but through the fixation point in cases of hæmorrhage (see p. 456).

2. Lesion of the right optic tract = left hemianopsia, the line of demarcation passing through the fixation point.

3. Lesion of the chiasma = bitemporal hemianopsia, the line of demarcation passing through the fixation point.

4. Lesion involving fasciculus lateralis only to right eye, causing nasal hemianopsia in the right field.

Diagram 138 also illustrates the fact that, as regards its relation to the optic tracts, the field of each eye is divided unequally, and not in halves, *e.g.*, the right tract governs about one-third of the field of the right eye, while the other two-thirds is governed by the left optic tract.

cuneus, renders it probable that there is in man a correlation between parts of the retina and of the occipital lobe, as Munk had already proved to be the case in dogs, and that the optic fibres from the right lower quadrant of each retina terminate in the adjacent part of the right superior occipito-temporal convolution, the left halves of the retina and left optic centres being of course similarly correlated. If this view be correct, as seems probable from Henschen's investigations, it is evident that altitudinal hemianopsia can hardly occur as the result of a central lesion, as nothing short of disease confined to the lower half of each cuneus would produce it.

It is now generally believed that relative hemianopsia (*e.g.*, colour hemianopsia alone) is the result of a lesion of less intensity than that which causes absolute hemianopsia. Mackay[1] points out that cases of apparently pure hemiachromatopsia may, with careful tests, show some diminution

[1] *British Med. Journ.*, Nov. 10th, 1888.

of the form-sense in the half fields which are defective for colour-sensations. Non-cortical lesions, even at the chiasma,[1] may also give rise to hemi-achromatopsia. Thus it would seem that the colour-sense is more easily affected by lesions than the form or light senses, and that too irrespective of the position of the lesion in the visual path.

It is now generally conceded that the macula lutea is specially represented in the cortical centre. But there are, at least, two very distinct views as to the arrangement of these macular centres, and as to the course of the macular fibres. These different views have been called into existence by the desire to explain the fact, that, in hemianopsia, the line of demarcation sometimes passes through the fixation point in the field, and sometimes leaves it in the seeing half. It seems to me that neither of these theories is satisfactory, and I regret that I cannot offer one that is so.

According to one theory, illustrated by Fig. 138, the macular region of the retina is invariably supplied on the same plan as the rest of the retina, *i.e.*, each side of it from the corresponding side of the brain. In order, then, to explain why it is that in some cortical lesions the line of demarcation passes through the fixation point in the field, while in others it deviates towards the blind side, the supporters of this view state, that the cortical centre for the macular region is more richly supplied with blood-vessels than the rest of the visual centre; as is the macula lutea itself in relation to the rest of the retina. Hence, when the lesion is an embolism, or thrombosis, of the vessels supplying that part of the brain, this special region, by reason of abundant anastomoses, preserves its functions, and then fields as in Fig. 136 are produced. But, if the lesion be a hæmorrhage, the macular region of the cortex would be apt to be involved in the lesion with the rest of the visual

[1] Hill Griffith, *Medical Chronicle*, Jan. 1887.

centre, and loss of function in the corresponding half of the macula lutea, with the line of demarcation passing through the fixation point, results.

According to the other theory, the whole of the macular region—and in some instances even more than this—of each retina being innervated from each hemisphere, there is an overlapping, as it is called, of nervous supply to these retinal regions. Consequently, if there be a lesion at the centre for vision in one occipital lobe, the centre for vision in the other occipital lobe being sound, the functions of the whole of each macula—or even of more than this—of the defective side of each retina, will be preserved. Cases where, occasionally, in cortical lesions, the line of demarcation in the field does go through the fixation point, would be accounted for, under this theory, by an individual variation in the supply of the maculæ, which, in these instances, would be similar to that of the remainder of the retinæ.

But any such theory, to be satisfactory, must be capable of explaining the phenomenon in question, not only when the lesion is in the cortex, but also where the hemianopsia is caused by a lesion in the tract or chiasma. Yet an examination of Fig. 138 will show that, according to the theory it represents, in lesions of the tract (2), or of the chiasma (3), the line of demarcation would pass through the fixation point. And, according to the other theory, a lesion either at the tract or at the chiasma would always cause the dividing line to circumvent the fixation point. It happens, however, that with lesions at either of these situations, the dividing line sometimes passes through the fixation point, and sometimes to one side of it. Consequently, I do not think we have yet solved the problem of the nervous supply of the macula lutea.

Some ophthalmologists hold that the line of demarcation always passes through the fixation point, and that it is merely imperfect fixation on the part of the patient which makes it seem to pass round it. This is an erroneous view,

but there are no doubt cases in which it is difficult to determine this, and where the line of demarcation approaches so close to the fixation point that it comes to be a question of degree.

The Localisation of the Lesion in Cases of Hemianopsia is a subject of interest, and, in view of the advances made within recent years in cerebral surgery, it is of great practical importance.

Lesions of the centre of the *chiasma* injuring the crossed fibres produce as their characteristic symptom bitemporal hemianopsia, which may be relative at first, beginning for instance as a hemi-achromatopsia, but later on becoming absolute. In some cases (basal meningitis, periostitis, hyperostosis), the diseased process comes to a standstill, and the bitemporal hemianopsia remains. But the disease generally extends to the uncrossed fibres, ultimately causing complete blindness. Even when the disease is non-progressive, central vision is impaired; whereas, in homonymous hemianopsia, it is not always affected. Optic atrophy, often commencing on the inner side of the papilla, is nearly always present at some period of the disease. Syphilitic gummata may cause transient recurrent attacks of bitemporal hemianopsia.

In Altitudinal Hemianopsia the lesion must also, as a rule, be at the chiasma, encroaching on it from above or below. Symmetrical cortical lesions might, and optic neuritis sometimes does, produce it.

In Nasal Hemianopsia, too, the lesion must be at the chiasma, and must be so situated in its outer angle as to involve only the fasciculus lateralis of the affected eye. The occurrence of binocular nasal hemianopsia is evidently almost impossible, implying, as it does, symmetrical lesion of the fasciculus lateralis of each tract. According to Henschen, a tumour in the external chiasma angle is apt to affect the crossed fibres as well as the uncrossed, and to produce a form of bilateral homonymous hemianopsia. Other symptoms

which may be present in lesions of the chiasma are anosmia, paralysis of orbital nerves, and anæsthesia of the conjunctiva and cornea. The causes are: fractures of the body of the sphenoid, cysts, tubercle, tumours, exostoses, distension of the infundibulum of the third ventricle, and, most frequently, tumours of the pituitary body. In the latter case proptosis, discharge of fluid from the nostril, and diabetes may be present.

Bitemporal Hemianopsia is a very common and early symptom in *Acromegaly*, a disease characterised by great hypertrophy of the face and extremities, associated with enlargement of the pituitary body, and other conditions which are not so constant.

In Homonymous Hemianopsia—the commonest form of the symptom—localisation of the lesion is a more difficult matter than in any of the other forms; for here the disease cannot be situated at the chiasma, but may be in the optic tract, or in the visual centre, or anywhere in the lengthened course of the fibres which connect these two parts.

Can we distinguish a complete and absolute hemianopsia, due to a lesion confined to the occipital lobe, from a similar defect in the field, due to a lesion in the optic radiations, internal capsule, pulvinar, or optic tract? We may conclude that the hemianopsia depends upon an occipital lesion, if it be unaccompanied by hemiplegia, motor aphasia, or paralysis of cerebral nerves, as direct symptoms; but be it remembered that one and all of these are liable to accompany lesions of the occipital lobe as distant[1] symptoms.

[1] I suggest the term "distant symptom" in preference to those in common use, namely, "indirect symptom," and "pressure symptom." We cannot assume that these symptoms are less the direct result of the lesion than any of the others which are present; and, in many instances at least, it is certain that they cannot be due to pressure. In short, we do not yet know what produces these symptoms,—they may be caused by inhibition,—we only know that they are the result of interference with

Aphasia, too, occasionally accompanies right cortical hemianopsia (*i.e.*, due to a lesion in the left occipital lobe), although it is not easy to offer a satisfactory explanation of the fact.

But the chief diagnostic symptom is what is known as negative vision, "vision nulle,"—that is to say, the patient, though he may be aware of the loss of half of his visual field, has no sensation of darkness in it, and is just as unconscious of the defect as a healthy person is of his blind spot.

Cortical hemianopsia may be a distant symptom. Gowers has observed that, at the onset of many attacks of cerebral hæmorrhage, hemianopsia is present as a distant symptom of very fleeting character, so transitory, indeed, that it does not complicate attempts at localisation; but I have seen it to last as long as three weeks. Except under this condition, distant hemianopsia seems to be rare, a fact which enhances the localising value of the symptom.

Cortical hemianopsia may be incomplete, inasmuch as the homonymous quadrant only of each field may be wanting. The explanation of this has been given, when speaking (p. 455) of the correlation of the visual cortical centres to parts of the retina.

So much for absolute hemianopsia. But the lesion may be such as to destroy only the colour centre, without reaching those for form and light. Eight cases of hemiachromatopsia are on record.

Again, the form-sense may be lost in the half field along with the colour-sense, while only the light sense is retained. Furthermore, cases of hemianopsia are on record in which, in part of defect, both the colour and form senses were absent,

functions of parts of the brain not involved in the lesion, and the term "distant symptom" conveys this idea—although perhaps not quite grammatically—without committing us to any theory. The corresponding German term is "Fernwirkung."

but the light-sense present, while in the remainder of the defect all three visual perceptions were lost.

It is generally held that lesions of the *Optic Radiations* cause homonymous hemianopsia, but it has not yet been proved that they are really visual fibres. Henschen believes that only a small portion of them can be regarded as such, while Vialet's investigations [1] seem to show that the visual path includes the whole of the optic radiations. A lesion here would be distinguished from one in the cortical centre by the possibility of hallucinations of vision occurring in the former and not in the latter; and further, there would not be "vision nulle" in the hemianopic defects, from lesion in the optic radiations. Lesions of the posterior third of the posterior limb of the *Internal Capsule* (Charcot's "Sensory Crossway") are still believed to cause hemianopsia and hemianæsthesia of the opposite side of the body; but analysis of clinical cases affords no support to this view, for there are no recorded cases which furnish any definite evidence in this respect. Yet anatomically fibres have been traced from the occipital cortex through the optic radiations and internal capsule to the basal ganglia, and from thence into the optic tract. The fibres passing through the internal capsule from the external geniculate body may perhaps be simply reflex fibres.

The symptoms due to lesion of *The Primary Optic Ganglia* (Pulvinar, Anterior Corpus Quadrigeminum, and External Geniculate Body) have not been as yet ascertained; the clinical evidence is indefinite. In lesions of the pulvinar alone, two typical symptoms occur, viz., hemianopsia and athetosis, and sometimes hemianæsthesia may be present. Hemianopsia from lesions of the *Optic Tract* is characterised by the absence of such symptoms as mind-blindness, word-blindness, etc., which are apt to occur in cortical affections,

[1] *Annales d'Oculist*, March 1894.

and by the presence, probably, of other symptoms pointing to a basal lesion. The defects in the fields may be relative (hemi-achromatopsia) or incomplete (only homonymous quadrants being lost).

Lesions of the optic tract are, of course, apt to implicate the crus cerebri, but do not necessarily do so; and then we would have hemiplegia of the opposite side of the body associated with the hemianopsia. Symptoms may also be caused by implication of cranial nerves, especially of those which supply the orbital muscles.

Atrophy of the optic nerve, and sometimes neuritis, according to the nature of the lesion, are frequently present.

The characteristic sign which enables us to localise a lesion in the optic tract from one elsewhere causing hemianopsia, is the hemiopic pupil (Wernicke's pupil-symptom). Illumination of the amaurotic half of the retina produces a more sluggish reaction, than when the light is thrown on the sound side; or, there may even be no contraction at all, because the lesion is on the distal side of the corpora quadrigemina, and, consequently, the impulse cannot reach Meynert's fibres to be conducted to the centre for the third nerve (see pp. 303 and 312).

It must be stated that some observers deny the occurrence of the hemiopic pupil. But, on the other hand, many observers have obtained the symptom. A great obstacle in observing it lies in the difficulty of concentrating the light on the blind side of the retina, without allowing it to fall on the good side. It is true that if the pupil-fibres in the optic nerve run as Bechterew thinks they do (p. 303), the hemiopic pupil could not occur.

If present, this is a valuable sign; but its absence is not decisive, on account of the difficulty of obtaining it.

The Forms of Diseased Process causing a lesion of the optic tract are: syphilitic gummata, and syphilitic meningitis; new growths, including tubercle; softening, and hæmorrhage,

are rare. Tumours of the optic thalamus, lenticular nucleus, or temporo-sphenoidal lobe, may also injure the tract by extension, or pressure.

The Prognosis for recovery of vision in the defective half of the fields depends, of course, upon the nature of the lesion; but recovery does not usually take place, especially in the most common class of cases—those, namely, which are due to cerebral apoplexy.

In Right Homonymous Hemianopsia, wherever the position of the lesion may be, a greater difficulty in reading is experienced than in left hemianopsia. This is partly due to the fact that we read from left to right; and that, owing to the defect being on the right side, the word immediately following that at which the patient is looking cannot be seen at the same moment. Knies offers another explanation, namely, that, owing to the right-sided defect, there is loss of the fine co-ordinated movements of the eyes to the right.

Transitory Hemianopsia or **Scintillating Scotoma.**—This affection is characterised by (1) symmetrical defects in the fields of vision, usually of the hemianopic type, and (2) vibrating or scintillating luminous sensations, which, after a short time, disappear, and are followed by an attack of (3), migraine. In fact, the visual troubles belong to the symptoms of this latter affection.

The scintillations and defects in the fields, either of which may occur first, commence over a small area, generally near the macula lutea, and gradually widen out; the flashing increases in intensity, and often assumes a zigzag shape, like fortifications, at the periphery of the defect in the field. And this defect may exist as symmetrical scotomata, complete or partial homonymous hemianopsia, or even altitudinal hemianopsia. In some cases the scintillation may be absent, while in others the attack of migraine does not follow. The ocular symptoms, which last for a period varying from a few

minutes to half an hour, are not accompanied by any changes in the fundus oculi, and always end in complete recovery. Vertigo, nausea, or sickness, and even slight aphasia sometimes accompany the headache.

This affection occurs most frequently in intellectually active individuals; fatigue, long reading, and hunger have been known to bring on attacks. The symptoms are most probably due to disturbances in the cerebral circulation.

Treatment should be directed to the cause of the migraine. Lying with the head low, stimulation of the circulation by wine or nitro-glycerine, sometimes cut short an attack.

Alexia (*a, priv*; λέξις, *speech*) or **Word-Blindness** is the term given by Kussmaul to an inability to understand written or printed characters, although they, and other small objects, can be distinctly seen. Other visual objects are named with ease (no visual aphasia). The patient can express his ideas in writing, or write from dictation, yet cannot understand what he has just written, nor can he copy written or printed words. He does understand the meaning of spoken words, and the use of all objects around him (no mind-blindness). He can generally recognise individual letters with some difficulty. This is "pure word-blindness," or "subcortical alexia." When combined with inability to write spontaneously or from dictation it is known as "cortical alexia" (Wernicke). The condition has been occasionally complicated with hemianopsia. In those cases where an autopsy was obtained, the lesion was found in the left occipital lobe. Word-blindness with agraphia, or cortical alexia, is due, according to Dejerine and Wernicke, to a lesion in the centre for visual memory for words; which, in right-handed people, is in the left-angular gyrus, and inferior parietal lobule.

Visual Aphasia consists in inability to name objects seen, the use of which is known. The objects can be named, if the patient be allowed to feel them, even with his eyes closed. A few cases of this affection have been recorded,

and in all there was right homonymous hemianopsia. Alexia and agraphia sometimes coexisted.

Dyslexia.—This symptom was first described by Berlin.[1] In a wide sense, it belongs to the aphasic group. It consists in a want of power on the patient's part to read more than a very few—four or five—words consecutively, either aloud or to himself. The difficulty is not caused by dimness of sight, nor by pain in the eye or head, but simply by an unconquerable feeling of dislike or disgust, due to the mental effort. After a few words, which can be well understood, have been read, the book is pushed away, and the head drawn backwards and turned aside; and then in a moment or two the patient may be tempted to repeat the effort, but with the same result after a very few words have been read. The symptom comes on suddenly, and has been usually the first sign of the presence of cerebral disease. Although, in most of the cases, the dyslexia disappeared in the course of a few weeks, either permanently, or to recur later on, yet other symptoms soon followed its first onset, such as headache, giddiness, aphasia, hemianopsia, paralysis of the tongue, hemianæsthesia, hemiplegia, twitching of the facial muscles, etc. Seven or eight cases are on record, and all have ended fatally. The lesion was situated, in all but one of those cases where an autopsy was obtained, in the neighbourhood of Broca's lobe. In one case the left hemisphere was normal, while the right hemisphere was extensively diseased.

Amnestic Colour-Blindness is a symptom which is most probably due to a lesion in the occipital lobe, interrupting the paths between the centre for vision and the speech centre. It has always been accompanied by right homonymous hemianopsia. In this condition the patient sees colours and can recognise them, and he can perform Holmgren's tests, but he is unable to name each colour.

Visual Hallucinations may occur in cases of homony-

[1] *Archiv. f. Psych.*, vol. xv., p. 276, and in his monograph, *Eine besondere Art der Wortblindheit* (Dyslexie), Wiesbaden, 1887.

mous hemianopsia in the blind side of the field only, being due to irritation of the visual-memory centre. Homonymous hemiopic hallucinations, persisting for years without hemianopsia, have also been observed. Visual hallucinations also occur very occasionally in connection with glaucoma, and of this I have seen an example. Hallucinations differ from illusions in that the former are completely subjective, while the latter are perverted sensory impressions.

Mind-Blindness, also called **Optic Amnesia,** is a symptom first observed by Munk[1] in his experiments upon animals. It consists in the loss of power of recognising objects, while the power of seeing them continues. A whip is seen by the animal, but inspires no terror; a tempting morsel is seen, but excites no desire. The symptom was caused by destruction of a region situated chiefly in the posterior division of the second external convolution of the dog's brain. Ferrier[2] seems disinclined to accept Munk's experiments. The symptom, however, has also been observed in man. The patient fails to recognise the most familiar objects by sight. In a case of my own the patient could not recognise his own wife until she spoke. There are two forms of mind-blindness—the cortical, and the transcortical. In the former the lesion is in the centre for memory, and in it the patient has lost the power of visual imagination, and cannot describe visual objects from memory. In the latter, the connecting path between the centre for vision and the visual memory centre is interrupted, and the patient, though he can describe an object from memory, is unable to recognise it when looking at it. Hemianopsia is present in the majority of cases of mind-blindness, and colour-blindness, complete or hemiopic, is not unusual. The lesion has been found in the occipital lobe, sometimes involving the parietal

[1] "Zur Physiologie der Grosshirnrinde," *Archiv f. Anat. und Physiol.*, v. and vi., pp. 162 and 547.

[2] Ferrier, *Functions of the Brain* 2nd ed., p. 298.

convolutions. It usually consists in hæmorrhage or softening, and is consequently sudden in outset. But it also occurs from tumours. Exhausting illnesses reducing the mental energy may produce a condition of mind-blindness.

Some authors localise the centre for visual memory in the angular gyrus, whilst others take for it the whole of the occipital lobe, except the cuneus and its neighbourhood.

Congenital Amblyopia.—This condition is not very uncommon. Ophthalmologists, in the course of their practice, come across people in whom the vision of both eyes is below the normal standard, even with perfect correction of any error in refraction, and who declare that they never have seen better, and that their sight is not getting worse. Still more common is congenital amblyopia in one eye. As a rule, the field of vision and the colour-vision are normal, but cases are seen in which there is contraction of the field, with defective colour-sight.

The *Ophthalmoscopic Appearances* are normal.

Reflex Amblyopia is said to have been observed, and chiefly in connection with irritation of the fifth pair, especially its dental branches; but I have not seen these cases, and I am rather sceptical as to their occurrence. Carious molar teeth are reputed to be its frequent cause; usually with severe toothache, but sometimes without it. The defect of vision may be confined to the side of the carious tooth, and is nearly always most marked on that side. It is said that it may be of extreme degree, vision being reduced even to the merest perception of light.

More generally recognised than amblyopia, as the result of toothache, are:—Hyperæsthesia of the retina, photophobia, subjective sensations of light, and diminution in the amplitude of accommodation.

All these symptoms, even amblyopia of the severest type, disappear, when the dental affection is relieved.

Many cases are on record, in which wounds of the supra-

orbital nerve were looked on as the cause of amblyopia, or of amaurosis; but it is by no means certain, that an ophthalmoscopic examination would not have afforded another explanation in many of these cases. Yet, even nowadays, many hold that wounds of the supra-orbital region can produce amblyopia, as cases are said to have been cured by division of the nerve involved in a cicatrix, that was tender on pressure.

Sympathetic Irritation (p. 290) is to be included under this heading. It is seen in the sound eye in some cases of cyclitis, and must not be confounded with sympathetic ophthalmitis, which comes about in quite a different way. Its symptoms are:—Diminution of the amplitude of accommodation, asthenopia, hyperæsthesia of the retina, lacrimation, and subjective appearances of light.

Removal of the exciting eye, if otherwise indicated, always relieves sympathetic irritation; but, where this is not admissible, the dark room, atropine, dry cupping at the temple, with bromide of potassium internally, may be employed.

The *Ophthalmoscopic Appearances* in reflex amblyopia are normal.

Hysterical Amblyopia.—In hysterical individuals amblyopia is sometimes seen, either as the only symptom, or in combination with others. It takes various forms, *e.g.*, complete blindness, even to loss of perception of light; defective central vision, with concentric contraction of the field, or, with segmental peripheral defects in the latter; or, as central scotoma. The colour vision is often affected.

The *Ophthalmoscopic Appearances* are normal, and the *Prognosis* good.

Treatment must be directed to the general system.

Nyctalopia (Night-Blindness).—This is a well-recognised symptom of the disease known as Retinitis Pigmentosa (p. 412). I have seen an instance of congenital night-blindness in five members of a family of ten children, without ophthalmoscopic signs; and Richter, quoted by Lawrence, observed a

similar instance. But the condition of which I have here to speak is Acute, or Idiopathic, Night-Blindness.

The patients can see well in good daylight; but, of a very dull day, or in the dusk of evening, or by indifferent artificial light, their vision sinks very much more than that of persons with normal eyes. They are then unable to see small objects, which are quite plain to other people, and, in a still worse light, they fail even to recognise large objects visible to every one else. This peculiar visual defect is due to imperfect adaptation power of the retina, and not to defective light-sense, as is sometimes stated.

Conjunctivitis and xerosis of the conjunctiva are often present in acute nyctalopia (p. 127). Some observers have found micrococci and bacilli in the conjunctiva in these cases, and have regarded these organisms as the cause of the conjunctival affection. It seems now more probable that they are merely secondary to the xerosis.

The connection between nyctalopia and xerosis conjunctivæ remains to be explained; but it is likely that they are both results of the one cause.

Acute nyctalopia is often the result of long-continued dazzling by very bright sunlight, or of lengthened exposure to bright firelight (*e.g.*, in foundries), and it is probable that in many, if not in most, instances of this affection, defective nutrition of the system plays the chief *rôle* in rendering the patients liable to it. Thus, in scorbutus, acute nyctalopia has been frequently seen, when the patients have been exposed to strong glares of sunlight.

Treatment consists in protection from light,—in short, in complete darkness for a time,—and then gradual return to ordinary daylight; while the system is to be strengthened by careful dietary and suitable tonic medicines.

Uræmic Amblyopia.—This is most commonly seen in connection with the nephritis of pregnancy and scarlatina, but may occur in any case of uræmic poisoning. It is met

with in acute forms of nephritis, in which albuminuric retinitis is not so liable to occur. The blindness is usually absolute, and may come on suddenly, or with a short previous stage of dimness of vision. It lasts from twelve hours to two or three days, and may recover completely, but in some cases a central scotoma remains.[1]

The *Ophthalmoscopic Appearances* are negative.

Treatment can only be directed to the general condition.

The Prognosis for vision is good, as it always recovers if the patient's life be spared.

Glycosuric Amblyopia.—In addition to the retinal affections dependent upon diabetes, we recognise the occasional occurrence in that disease of defects of vision, which are referred to disorder of the optic nerve, and which are not always accompanied by ophthalmoscopic changes. These defects of vision are found in the form of :—1. Central Amblyopia (see p. 440), or, in slighter cases, as amblyopia without central scotoma. Occasionally, higher degrees of amblyopia with concentric contraction of the field of vision, and yet negative ophthalmoscopic appearances, are present. 2. Atrophy of the optic nerve. This may appear in the usual form as progressive blindness, with concentric contraction of the field of vision ; or it may come on after the slighter form of amblyopia has existed for some time. 3. Hemianopsia and colour blindness (Samelsohn). It is probable (Leber) that these apparently different kinds of blindness depend upon similar pathological processes, and merely indicate degrees of the latter. In what these processes consist is still unknown ; but the tendency to hæmorrhages in the retina in diabetes makes it likely, that hæmorrhages in the optic nerve are sometimes the source of the amblyopia in question ; while, in the cases with central scotoma, it is no doubt[2] due to retro-bulbar neuritis similar to that produced by tobacco, etc.

[1] Berger, *Maladie des Yeux et Pathologie Générale*, p. 389.

[2] Nettleship and Edmunds, *Trans. Ophthal. Soc.*, vol. i., p. 124.

Amblyopia is sometimes the only symptom of diabetes; and, consequently, as Leber points out, it is of the utmost importance to examine the urine for sugar in every case of amblyopia, where the ophthalmoscopic appearances are negative, or where the only abnormality is atrophy of the optic papilla.

The Treatment indicated is solely that for the general disease, and the prognosis for vision depends upon the amenability of the latter to treatment, and upon the extent to which organic changes in the optic nerve have gone. Hirschberg [1] inclines to the view that diabetic amblyopia constitutes a serious symptom for the life of the patient.

Quinine Amaurosis.—Quinine in large doses, and very occasionally in small doses, is liable, in some individuals, to cause amblyopia, which may amount to absolute blindness, accompanied for some hours or days by great deafness. This absolute blindness is never more than temporary, although it may last for some weeks; but in severe cases concentric contraction of the field is apt to remain permanently, with or without some defect of central vision. In the only instance of this more serious result which I have seen, the colour and light-senses, notwithstanding the contracted field and marked seeming optic atrophy, were normal; but the adaptation of the retina, as shown by considerable night-blindness, was defective.

In what may be called the acute stage the *Ophthalmoscopic Appearances* are sometimes normal, but pallor of the optic papilla, with scarcity and smallness of the retinal vessels, is the usual condition. Where the case is chronic—the fields remaining contracted, although central vision has improved—the ophthalmoscope may discover a very pale optic papilla with minimal vessels.

The retinal ischæmia is doubtless the immediate *Cause* of the amblyopia, and, in its turn, is the result of diminished

[1] *Centralbl. f. Augenheilk.*, 1886, p. 199.

heart's action and lowered arterial tension, both of which have been shown to be produced by large doses of quinine.

Treatment.—Cessation of the use of the quinine. Digitalis internally to raise the arterial tension, nitro-glycerine or inhalations of nitrite of amyl, hypodermic injections of strychnia, and general tonic treatment are the means most likely to promote a cure.

Pretended Amaurosis.—Malingerers rarely pretend total blindness of both eyes, and such cases can often only be detected by constant observation of their actions.

Presence of pupillary reflex is no proof that the patient sees, for this would be quite compatible with a cortical lesion causing total loss of sight (p. 313).

The crossed diplopia test (*vide infra*) may be employed in these cases, as, if both eyes see, the one with the prism will rotate inwards for the sake of single vision, while, if both eyes be blind, of course no such motion will take place. Again, if the malingerer's own hand be placed in various positions, and he be asked to look at it, he will, in all probability, look in some other direction; whereas, a truly blind man usually makes a fair attempt at directing his eyes towards his own hand.

Pretended monocular amaurosis can generally be detected by The Diplopia Test. If the malingerer be made to look, with both eyes open, at a lighted candle placed some feet off, while a prism with its base downwards is held before the admittedly good eye, he will say he sees two images of the light, one over the other. Were he blind of one eye, he would not see two images.

Another method—The Crossed Diplopia Test—consists in holding a prism of some 10° or 12° with its base outwards before the pretended blind eye; when, if it sees, it will make a rotation inwards for the sake of single vision, an effort which a blind eye would not make.

Alfred Graefe's Method.—In this test the pretended blind

eye is covered with the surgeon's hand from behind the patient, while, with the other hand, a prism (about 10°) is held base down before the good eye, so that its edge may pass horizontally across the centre of the pupil. Monocular double vision results, as the rays pass through the upper part of the pupil normally, while through the lower part of it they are refracted downwards by the prism. The double images stand over each other. If now the hand, which excludes the pretended blind eye, be rapidly removed, while, at the same moment, the prism is moved upwards, so that the entire pupil is covered by it, a malingerer will still see double images standing one over the other; for now the diplopia must be binocular.

Harlan's Test[1] consists in placing a trial frame on the patient's nose with a very high + lens—say + 14 D—opposite the good eye, by which means it is excluded from distant vision, and a plane glass—or a 0·25 D convex or concave lens, which of course would not materially interfere with its distant vision—opposite the pretended blind eye. The patient then, believing there is much the same kind of glass before each eye, will read the test-types; and, if it be now desired to expose the deception, the pretended blind eye is excluded from sight, and the malingerer will then be unable to read the test-types.

Snellen's Coloured Types may also be used for this purpose. These types are printed in green and red. If a person be really blind of one eye, he will, of course, see both the green and the red letters with the good eye. But, if a green glass be held before the good eye, the rays from the red letters will be excluded, and he will now only see the green letters; or, with a red glass the red letters alone will be seen. A malingerer may be detected by holding before his admittedly good eye a green glass; and, if he now still

[1] *Trans. Amer. Ophthal. Soc.*, vol. iii., p. 400.

see the red letters, it must be that he does so with the so-called blind eye.

It is well to have this variety of tests, in order that they may be used to corroborate each other.

Erythropsia (*ἐρυθρός, red*)—**Red Vision.** A large number of cases of this remarkable affection are on record; indeed, it will have come under the notice of nearly every ophthalmic surgeon of any experience. Two-thirds of the cases have been subjects of successful cataract operations, whilst the remainder have possessed normal eyes. In some cases the red vision remains only a few minutes, and does not again return; whilst in others it appears every day for a short time, for weeks or months; and, again, in others it continues for several days, and then disappears for good, or recurs at intervals. In the aphakic cases, it does not usually appear for weeks or months after the removal of the cataract, and, in one case, the interval was two years. During the attacks the patients see all objects of a deep red colour, and occasionally of a purple or violet hue. In no instance is the acuteness of vision affected, either during or after the attacks.

A satisfactory explanation for the affection has not yet been offered. It seems probable that it is due to over-excitation of the visual nervous apparatus—some believe of the visual centre, others of the retina—set agoing by exposure of the retina to strong light, along with other favouring circumstances, especially general over-excitement of the body, or mind. More than this cannot at present be said. Why aphakic eyes should be so much more liable to erythropsia, than eyes which possess their crystalline lenses, is an enigma.

Treatment seems to have but little effect. Protection of the eyes from light has not been of use. Bromide of potassium internally seems to have done some good in those cases, where it was tried.

CHAPTER XVIII.

THE MOTIONS OF THE EYEBALLS, AND THEIR DERANGEMENTS.

The eyeball moves round a point on its antero-posterior axis, situated (in the emmetropic eye) 14 millimetres behind the cornea, and 10 millimetres in front of the posterior surface of the sclerotic. Its motions are effected by means of the six orbital muscles, arranged in three pairs, each pair consisting of two antagonistic muscles; thus, the rectus internus and rectus externus are antagonistic, the former rotating the eye inwards, and the latter rotating it outwards. The two remaining pairs are the recti superior and inferior, and the obliqui superior and inferior.

The Primary Position of the Eyeball is that one in which, the head being held erect, the gaze is directed straightforwards in the horizontal plane. This is the starting-point from which the actions of the muscles are considered.

The Rectus Externus and Rectus Internus, lying from their origin to their insertion in a plane which corresponds with that of the horizontal plane of the eyeball, move the latter on its perpendicular axis directly inwards and outwards, and have no other action.

The plane of *The Rectus Superior and Rectus Inferior* does not quite correspond with the vertical plane of the eyeball, and, consequently, the axis on which they rotate the globe is not its horizontal axis, but one which, passing from within and before, backwards and outwards, forms with the antero-posterior axis an angle of 70° (Fig. 139). While, then, their

action is mainly to rotate the eyeball upwards and downwards, these muscles rotate it also somewhat inwards. Moreover, the superior rectus giving to the vertical meridian of the cornea an inward inclination, or inward wheel-motion[1] of the eye (*vide infra*), while the inferior rectus gives this meridian an outward inclination, or outward wheel-motion of the eye, the power of these muscles over the upward and downward motions is greatest, when the eye is turned out; for then their axis of rotation coincides most closely with the horizontal axis of the globe; and their influence over the wheel-motion is greatest, when the eye is turned in; for then their axis coincides most closely with the antero-posterior axis of the globe.

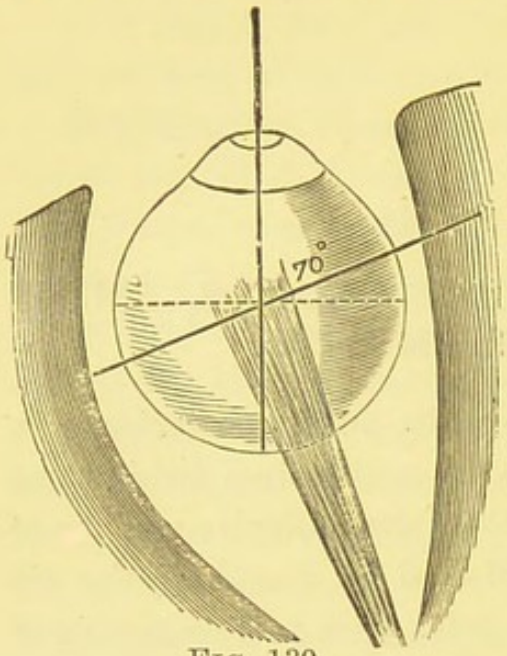


FIG. 139.

The plane of *The Oblique Muscles* of the eyeball also approaches the vertical plane of the eyeball, the axis upon which they rotate the latter passing from within and behind, forwards and outwards, and making with the antero-posterior axis an angle of 35° (Fig. 140). The principal action, accordingly, of the oblique muscles is to incline the vertical meridian of the

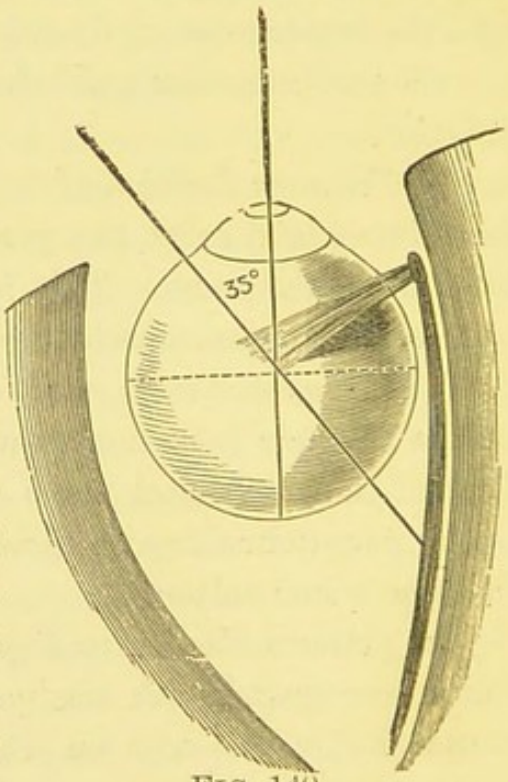


FIG. 140.

[1] In speaking of the inclination of the vertical meridian of the cornea, it is the upper extremity of this meridian which is meant.

cornea; the sup. oblique inclines it inwards (wheel-motion inwards), the inf. oblique inclines it outwards (wheel-motion outwards). In addition to this action, the oblique muscles, respectively, rotate the eyeball downwards and outwards (sup. oblique), and upwards and outwards (inf. oblique). It is evident that the power of these muscles over the upward and downward motions of the eyeball is greatest, if the eye be turned in; and that their power over the wheel-motion is greatest, when the eye is turned out.

To sum up, then, the superior oblique and rectus produce wheel-motion inwards, while the inferior oblique and rectus produce wheel-motion outwards. The action of the obliques on the wheel-motion is greatest, when the eye is rotated outwards; and of the recti, when the eye is rotated inwards.

In considering the motions of the eyeballs, we have to think of the motions of one eyeball as associated with those of its fellow; *e.g.*, the action of the internal rectus of the left eye is associated with the action of the external rectus of the right eye, in rotation of both eyeballs to the right.

The vertical meridian of the eyes becomes inclined to the right or left in different positions of the globe, as was experimentally proved by Donders.

1. In the primary position, as also when the eyes are turned directly inwards, outwards, upwards, or downwards, the vertical meridians (*a*, *b*, Figs. 141—145) maintain their vertical direction (Fig. 141).

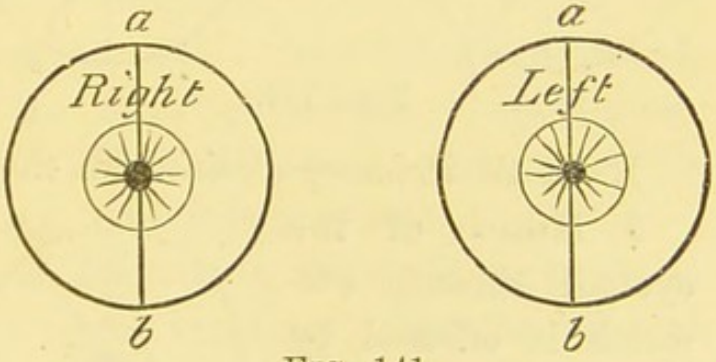


FIG. 141.

2. When the eyes are turned to the *left, and upwards*, the vertical meridian of each eye is inclined at the same angle to the left (Fig. 142). Wheel motion to the left.

3. When the eyes are turned to the *left, and downwards*,

the vertical meridian of each eye is inclined to the right at the same angle (Fig. 143). Wheel-motion to the right.

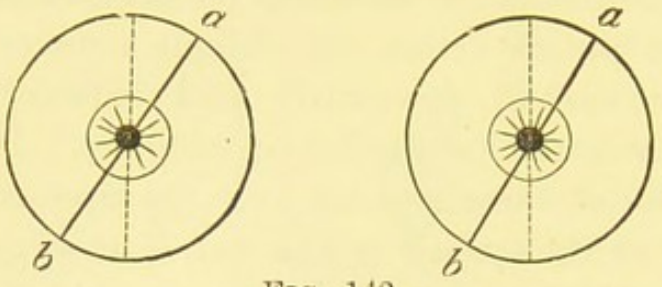


FIG. 142.

4. When the eyes are turned to the *right, and upwards*, the vertical meridian of each eye is inclined at the same angle to the right. Wheel-motion to the right (Fig. 144).

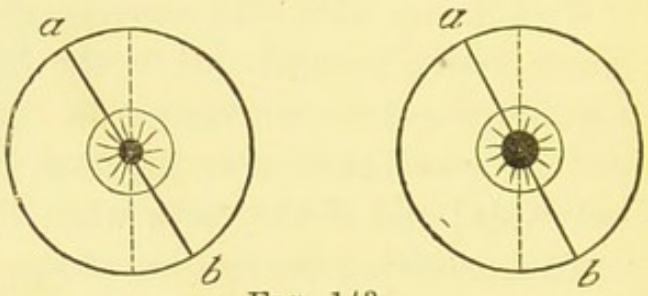


FIG. 143.

5. When the eyes are turned to the *right and downwards*, the vertical meridian of each eye is inclined at the same angle to the left. Wheel-motion to the left (Fig. 145).

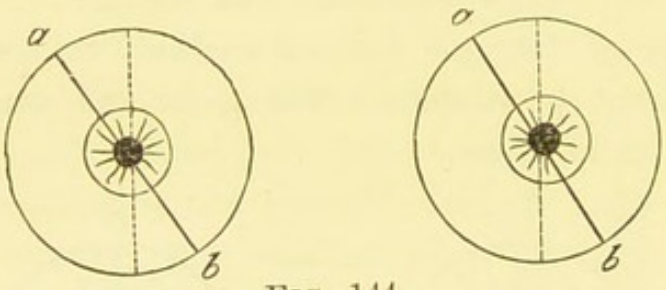


FIG. 144.

We shall now consider what muscles are called into action, when an individual requires to place his eye in the several principal positions.

1. *In the Primary Position* all the muscles are at rest.

2. Motion of the eyeball *directly outwards* is effected by the external rectus alone, and motion *directly inwards* by the internal rectus alone.

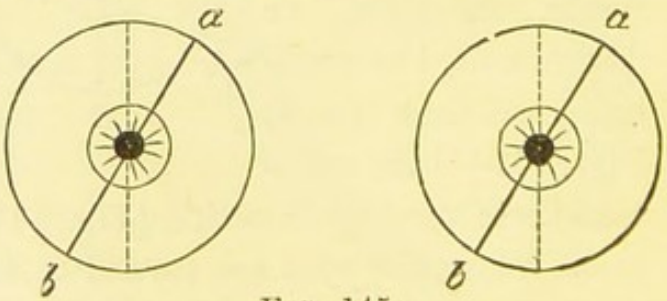


FIG. 145.

3. Motion of the eyeball *directly upwards* and *directly downwards* is effected chiefly by aid of the sup. and inf. recti

But these muscles, acting alone, rotate the eyeball slightly inwards, and give a certain inclination to the vertical meridian, which, in this position, should be upright. Consequently, in rotation of the globe directly upwards, the inf. oblique, which rotates the eye slightly outwards (as well as upwards), and inclines the vertical meridian outwards, must be associated with the sup. rectus, in order to counteract in these particulars the tendency of its action. In rotation of the eyeball directly downwards, the inf. rectus must be associated with the sup. oblique, which acts antagonistically to this rectus in respect of rotation inwards, and of outward wheel-motion.

4. Rotation *upwards and outwards* is chiefly effected by aid of the rectus superior and rectus externus. But the latter muscle has no influence over the wheel-motion, while the former produces wheel-motion inwards. Yet, the inclination of the vertical meridian is outwards in this position; and, therefore, a third muscle, which will supply this inclination in a high degree, is required, namely, the inferior oblique, whose power over the wheel-motion of the eyeball is greatest when the latter is in this position.

5. Rotation *downwards and outwards* is chiefly effected by the rectus inf. and rectus ext. Inasmuch, however, as the former inclines the vertical meridian outwards, while the latter has no influence over it at all, a third force is required, which will bring about the required inward wheel-motion, namely the sup. oblique, whose influence in this respect is most powerful when the eye is in this position.

6. Rotation *upwards and inwards* is chiefly brought about by the rectus superior and rectus internus. But the effect of the former upon the inward wheel-motion of the eye would be so great as to interfere with parallelism of the vertical meridians of the two eyes, that of the other eye not being inclined outwards in a corresponding degree. A third force, therefore, is required, which will to a certain extent counteract the influence of the sup. rectus in this respect, and this is the

inf. oblique, which, in this position of the eyeball, has but slight power over its wheel-motion.

7. Rotation *downwards and inwards* is chiefly the result of contraction of the rectus inf. and rectus int. The power of the former over the outward inclination of the vertical meridian would, in a similar way, be too great, and must be similarly corrected by the action of the superior oblique.

The Field of Fixation.

The field of fixation contains all the points which the eye can successively see or "fix" without movement of the head. It can be measured with the perimeter, as in testing the field of vision, except that here the patient is made to move the eye as far as possible in each meridian, and the limit of each movement is measured by observing the corneal reflex of a candle flame, or ophthalmoscope mirror, which is moved along the arc of the perimeter. The *binocular* field of fixation contains all those points which can be seen as single, with the two eyes, and without movement of the head. According to Landolt[1] the averages give, for movement of one eye, inwards 44°, outwards 46°, upwards 44°, and downwards 50°.

Strabismus.

When looking at any object with the two eyes, it is necessary, in order to avoid seeing double, that the visual axis of the eyes should meet at the point fixed. When this does not take place, one of the eyes must be in a faulty position, or, as it is commonly termed, it squints. This condition is called *Strabismus*, and may arise either from over-action, or from paralysis of one of the muscles. Strabismus may occur in any direction, but vertical and oblique deviations are less common than the convergent or divergent forms.

In order to find out, in slight cases, which is the deviating eye, the patient is made to fix a certain object, and one of

[1] Landolt and Wecker, *Traité d'Ophthal.*, vol. iii., p. 782.

the eyes, say the left, is suddenly covered with the surgeon's hand; then, if the right eye make no movement, it must have been looking at the object; but if, on covering the right eye, the left make a movement in order to fix the object, then this eye must be the squinting one. The movement is always in the opposite direction to the deviation. For instance, if the eye be turned inwards too much, it must, naturally, turn outwards to fix the object, when its fellow is covered. Another good method consists in observing the position of the corneal reflex when the patient looks at the ophthalmoscope (see Measurement of Strabismus). But the most delicate test is the character of the diplopia, when the latter exists.

Apparent Strabismus is due to a large angle γ (p. 15). In this case, as the visual axes are both directed to the point fixed, there will be no movement of either eye, on covering the other, as in real strabismus.

Paralyses of the Orbital Muscles.

Loss of power of one or more of the muscles of the eyeball is, of course, always to be regarded as a symptom, not as itself a disease.

It may be due to lesions in several different situations, namely:—1. Lesions situated in the orbit. 2. Basic lesions—lesions situated at the sphenoidal fissure, and those at the base of the skull, between that point and the pons. 3. Pontine lesions, which may be Fascicular—involving the ocular nerve fibres in the substance of the pons, or nuclear—only attacking the nuclei of the nerves in the aqueduct of Sylvius and floor of the fourth ventricle. 4. Cerebral lesions—lesions above the nuclei, in the internal capsule, corona radiata, or cortex. These four classes differ considerably in their clinical aspect, in their pathological causes, and in their significance for the well-being of the patient.

The first class—loss of power due to orbital lesions—will be referred to in the chapter on Diseases of the Orbit.

The second class—those due to basic lesions—provides by far the largest number of cases of paralyses of the orbital muscles. Let us now consider the

General Symptoms of this class. They include symptoms to be found in each of the other classes. 1. Diplopia. The affected eye being deviated from its correct position, and being more or less incapable of associated motions with the other eye, the image of the object looked at is not formed on identical spots of the retina in each eye, and hence the object seems doubled. 2. Indistinct vision. If the paralysis be but slight, actual diplopia may not be present, but the double images overlapping each other will cause dimness or confusion of sight. 3. Giddiness, due partly to the diplopia, and partly to faulty projection of the object. By faulty projection is meant the false idea of the position of the image in the field of vision [1] 4. Some patients turn the head towards the side of the paralysed muscle, in order to diminish or eliminate the diplopia; *e.g.*, if the left ext. rectus were paralysed, the head would be turned towards the left; if it were the left int. rectus, the head would be turned towards the right. By this manœuvre the loss of the action of the affected muscle is less felt, for those objects which lie straight in the patient's path, while he walks about; because it involves a rotation of the eye towards the side of the healthy antagonist, in which region of the binocular field the diplopia is reduced to a minimum. Some patients close one eye to procure single vision. 5. In peripheral paralysis it is most common to find only the muscle, or muscles, supplied by some one nerve—the third, fourth, or sixth—affected; although of course

[1] Landolt has recently shown (*Archives d'Ophthalmologie*, No. 5, 1893) that the angular value of the false projection is equal to the difference between the primary and secondary deviation. L. W.

exceptions to this are not rare, especially where a neoplasm forms at the base of the skull.

In studying a case of paralysis of an orbital muscle, the following *General Principles* should be borne in mind. 1. The defective mobility, and the diplopia, increase towards the side of the affected muscle, *e.g.*, towards the left, if the left external rectus be paralysed; towards the right, if the left internal rectus be paralysed. 2. The secondary deviation (*i.e.*, the deviation of the sound eye while the affected eye fixes) is greater than the primary deviation (*i.e.*, the deviation of the affected eye while the sound eye fixes). Because, the muscle in the sound eye, which is associated in its action with the paralysed muscle in the affected eye (*e.g.*, the rect. int. with the rect. ext.), must receive a nervous impulse of equal intensity to that sent to the weak muscle, and, as the latter requires a considerable impulse to excite its action, its associate will be over-excited. Let us suppose the left external rectus to be paralysed, and that, shading the right eye with a hand, we direct the patient to fix with his left eye an object held somewhat to his left-hand side; we may notice, on removing the shading hand, that the right eye has been rotated inwards to an extent far exceeding that of the primary deviation of the left eye, and has now to make an outward motion, in order again to fix the object. 3. The image formed on the retina of the affected eye is projected (seems to lie) in the direction of the paralysed muscle; *e.g.*, if the left ext. rect. be paralysed, the image corresponding to that eye will be projected to the left of the image belonging to the right eye. Where the image of the affected eye lies to the corresponding side, as in this instance, the diplopia is termed homonymous, and such double vision always indicates convergence of the visual lines. Fig. 146 explains the occurrence of homonymous diplopia in convergent strabismus.[1] The right eye "fixes" the

[1] For the sake of simplicity in the diagram, the effect which rotation of the eye has on the nodal point is omitted.

object *o*, and its image falls on the macula lutea *m*; but the left eye, on account of the paralysis of the external rectus, is turned in, and its visual axis lies in the direction *m v*, and the image of *o* falls to the inner side of the macula lutea at *a*. Now why should this image not be referred to its correct position along the line *a o*? The reason is, that the patient is not conscious of the deviation of this eye; and, having always been in the habit of superposing his fields of

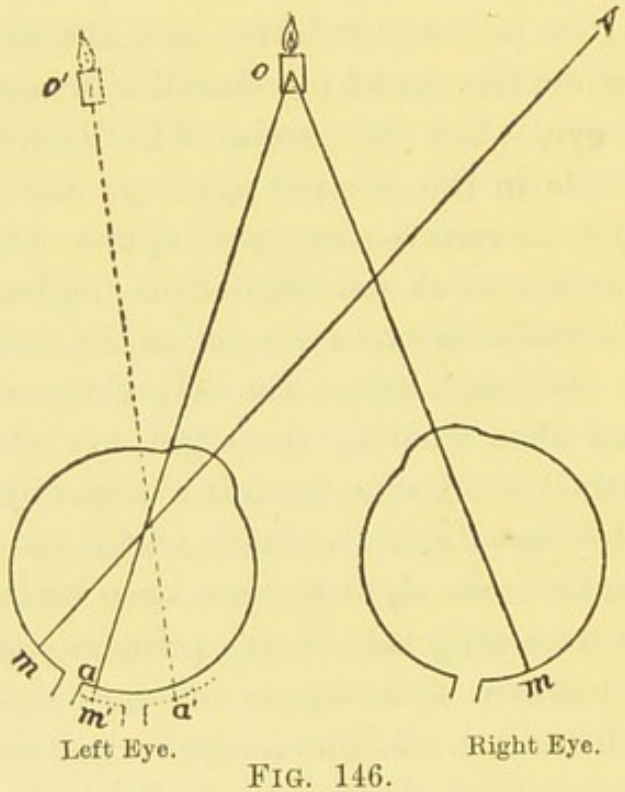


FIG. 146.

vision, so that the visual axes of the eyes meet at the object fixed, he imagines that this is still the case, and that *v m* lies in the position of *o a*, and that the macula lutea *m* is at *m'*. Now if this were the case *a* would be at *a'*, and in this position of the eye, indicated by the dotted line, images formed at *a'* to the inner side of the macula lutea are projected to the outer side of the field, along the line *a'o'*, and the patient imagines that *o* occupies the position *o'*, as seen with the left eye.

But, if we suppose the internal rectus of the left eye to be

paralysed, the image on the retina of that eye falls then to the outside of its macula lutea, and must, therefore, be projected to the right of the true position of the object; this is crossed diplopia, and attends divergence of the visual lines.

Paralysis of the External Rectus of the Left Eye.—If this be complete, or considerable, it is easy of diagnosis, as marked loss of power and motion of the eyeball outwards is present, and the patient complains of double vision. He keeps his head turned to the left, in order to diminish the influence of the paralysed muscle as much as possible.

If, however, the paralysis be but slight, the patient may not complain decidedly of diplopia, but only of indistinctness or confusion of sight, especially when he looks towards the left. To decide the diagnosis in such a case the double images must be examined. A long lighted candle is used as the object to be looked at; and one eye—let us say here the left eye—is covered with a bit of red stained glass, in order to differentiate the images.[1] The candle is now held on a level with the patient's eyes, and straight opposite him, at about three metres' distance (eyes in primary position). *a.* In this position the images are seen very close together, or overlapping each other, both of them upright and on the same level, the red candle to the left, the white to the right, *i.e.*, homonymous diplopia = convergence. This convergence must be due to paralysis of one or other external rectus muscle, but we cannot say at this stage of the experiment which of them is affected. *b.* In order to determine this point, the candle must be carried from side to side, and the increasing, or decreasing, distance of the images from each other noted. If the candle be carried slowly to the right, the patient following it with his eyes while his head remains fixed, the images come still closer together, or only one candle is seen. But, if the candle be

[1] Maddox' Rod Test, described further on, is very suitable here, and in the investigation of other forms of ocular palsy.

carried to the patient's left-hand side, the images go farther apart, their relative positions being maintained. We now know that it is the left external rectus which is affected; because, towards the left—the direction in which the action of this muscle is most wanted, and consequently its loss most felt—the distance between the double images increases. The images are erect, as no wheel-motion is caused by action of the external rectus. *c.* If, however, the candle be held to the left and raised aloft, the image belonging to the left eye will seem to lean away from that of the right eye (Fig. 147). The reason of this is, that, owing to the paralysis of the external rectus, the left eye cannot look upwards and outwards as it should, but merely looks upwards. The vertical meridian therefore remains vertical. But the right eye, which is free to follow the candle, looks up and to the left. Its vertical meridian is therefore inclined to the left. That is, the vertical meridians of the two eyes converge at the top, which necessitates a divergence of the upper extremities of the images. The rotation of the right eye in this position is physiological, and its image is therefore judged to be vertical; while the image of the left eye diverging from that of the right, though really vertical, is judged to be oblique. An analogous derangement of the vertical meridian takes place in the position below and to the outside. *d.* If the patient be told to direct his gaze specially towards the red candle, the distance between the two candles will be much greater than if he direct his gaze towards the white candle. This is explained by General Principle No. 2 (p. 483).

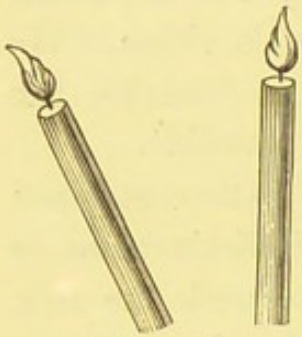


FIG. 147.

If the patient's good eye be closed, and an object (surgeon's finger) be held up within his reach, but towards his left-hand side, and he be requested to aim rapidly at it with his fore-finger, he will aim to the left of it. The nervous impulse sent

to his left external rectus, to enable him to turn the eye towards the object, is of such intensity as to lead him to fancy that the object lies much farther to the left than it does (incorrect projection of the field of view); for we, to a great extent, estimate the distance of objects from each other by the amount of nervous impulse supplied to our orbital muscles in motions of the eyeball.

A prism held horizontally before the affected eye, with its base outwards, brings the double images closer together; or, if the correct prism be selected, the images will be blended into one.

Paralysis of the Superior Oblique of the Left Eye.—This paralysis will be most apparent when a demand is made for motion of the eyeball downwards and inwards, motion in this direction being that over which the superior oblique has most influence. Still, absolute defect of motion is sometimes difficult to detect even in complete paralysis of this muscle, owing to vicarious action of the inferior rectus and internal rectus. Careful examination of the secondary deviation will often be successful as to this point, but it is the examination of the double images upon which we must chiefly depend for the diagnosis.

a. In the whole of the field of vision above the horizontal plane there is single vision. Below the horizontal plane in the median line diplopia appears, the image belonging to the left eye standing lower than that belonging to the right, because the superior oblique being a muscle which assists in rotating the eye downwards, the latter, for want of the action of this muscle, now stands higher than its fellow (right eye), and, consequently, the image will not fall on its macula lutea (as it does in the right eye), but above it, and will therefore be projected below the image of the right eye. The position, downwards and inwards, of the eyeballs is that in which the greatest demand is made upon the superior oblique for rotation of the eye downwards: therefore, it is in this position

its want for this purpose is most felt; and when the candle is held in this position, the vertical distance between the double images is greatest. *b.* The superior oblique assists also in rotation of the eye outwards: therefore, loss of its power must commit the eyeball to a certain extent to the power of the muscles which move it inwards, and a rotation in this latter direction (convergence) takes place, with the result of making the image belonging to the left eye stand to the left of the image belonging to the right eye (homonymous diplopia). *c.* The superior oblique inclines the vertical meridian inwards: therefore, in rotation directly downwards, loss of its power commits the eye to the outward wheel-motion imparted to it by the inferior rectus. This gives to the image belonging to the left eye an inclination to the patient's right hand. *d.* The power of the superior oblique to incline the vertical meridian inwards is greatest, when the eye is turned downwards and outwards: consequently, in this respect, its paralysis will be most felt in this position, and therefore here the inclination of its image to that of the sound eye will be most marked. *e.* A remarkable phenomenon usually noticed in this paralysis (and sometimes in paralysis of the inferior rectus), and for which a good explanation does not exist, is, that the image belonging to the affected eye seems to stand nearer the patient than that of the sound eye.

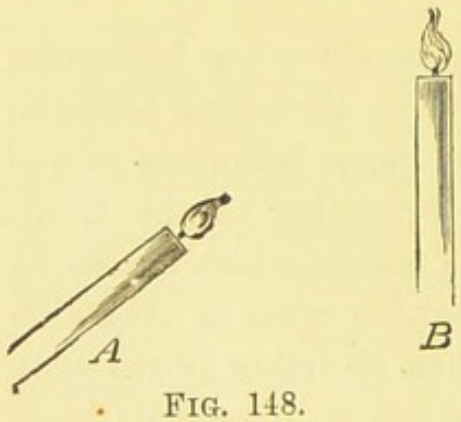


FIG. 148.

To sum up, then (*vide* Fig. 148): below the horizontal plane there is homonymous diplopia, while the image (*A*) of the affected eye stands on a lower level, is inclined towards the other image, and seems to be nearer the patient. Furthermore:—

f. In an extreme lower and outer position, the image of the affected eye may sometimes seem to stand higher than that

of the sound eye, owing to an excessive outward inclination of the vertical meridian, which throws the image on the lower and outer quadrant of the retina.

In order to do away with or to diminish the diplopia, the patient inclines his head forwards, and turns it to the side of the good eye.

For the prismatic correction of the diplopia two prisms will be required, one with its base downwards in front of the left eye to correct the vertical difference, and a second with

FIG. 149.

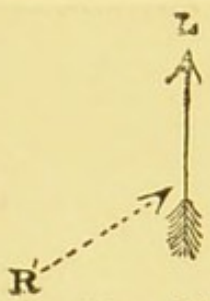


FIG. 150.

Fig. 149. Paralysis of left sup. oblique. Homonymous diplopia. R, image of right eye. L, image of left eye.

Fig. 150. Paralysis of right inferior rectus. Crossed diplopia. R′, image of right eye. L′, image of left eye.

its base outwards in front of the right eye to correct the lateral difference.

To make the diagnosis between the foregoing paralysis, and paralysis of the right inf. rectus,—in which the diplopia is also below the horizontal plane only, and the false image lower than the true one and inclined towards it—it has merely to be remembered that there is here crossed—instead of homonymous—diplopia, because the superior oblique, which now chiefly effects the downward motion of the eyeball, turns it at the same time somewhat outwards. The figures 149 and 150 will assist in this explanation.

Paralysis of the Internal Rectus, Superior Rectus, Inferior Rectus, Inferior Oblique, and Levator Palpebræ.—Complete paralysis of all the branches of the third nerve produces a remarkable appearance. The upper lid

droops (ptosis), the pupil is semi-dilated and immovable, the power of accommodation is destroyed, and the eyeball is often slightly protruded, owing to the backward traction of the recti being lost to it. Motion inwards exists but to a slight degree, and motion downwards is effected only by aid of the superior oblique, and is accompanied by marked inward wheel-motion. If the paralysis be of some little standing, the external rectus obtains rule over the eyeball, and rotates it permanently outwards.

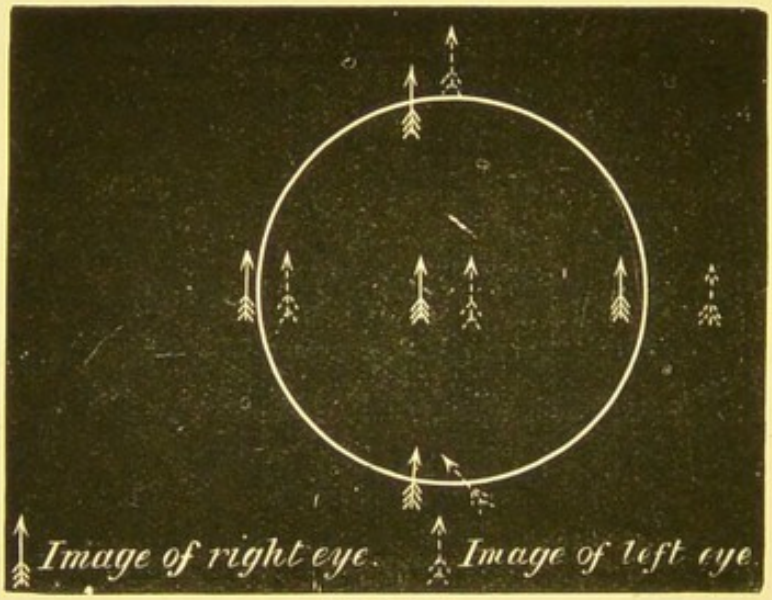


FIG. 151.

The diagnosis, then, in cases of complete paralysis of all branches of the nerve, is easily made; but not so, sometimes, if the paralysis be only partial. The examination of the double images then is of value. If (see Fig. 151) the left third nerve be partially paralysed, in all or most of its branches, there will be crossed diplopia, either in the whole of the field of vision—for want of power in the internal rectus—or towards the patient's right at the least, and the lateral distance between the images will increase as the visual object is carried farther towards the right. When the visual object is held aloft, the left eye will remain behind—for want of the action of both of the muscles which turn the eye upwards—and, consequently,

in this position, its image will stand, not only to the right of, but also above that of the right eye; while, when the visual object is held below the horizontal plane, the eye will—owing to paralysis of the inferior rectus—remain higher than the right eye, and, consequently, its image will appear to be lower than that of the right eye. It will, moreover, be inclined towards the latter image, in consequence of the inward wheel-motion imparted to the eye by the superior oblique.

When in each eye some branches of the third are paralysed, the diagnosis is often extremely complicated. The ptosis, however, which is nearly always present, and is readily recognised, and the paralysis of the sphincter iridis (mydriasis) and of accommodation, which often exist, and are also easily observed, give valuable aid. Moreover, any loss of motion upwards must be due to paralysis of the third nerve; but, if there be loss of motion downwards, the differential diagnosis between paralysis of the inferior rectus and of the superior oblique has to be made. For this see the paragraph on paralysis of the latter muscle (p. 489).

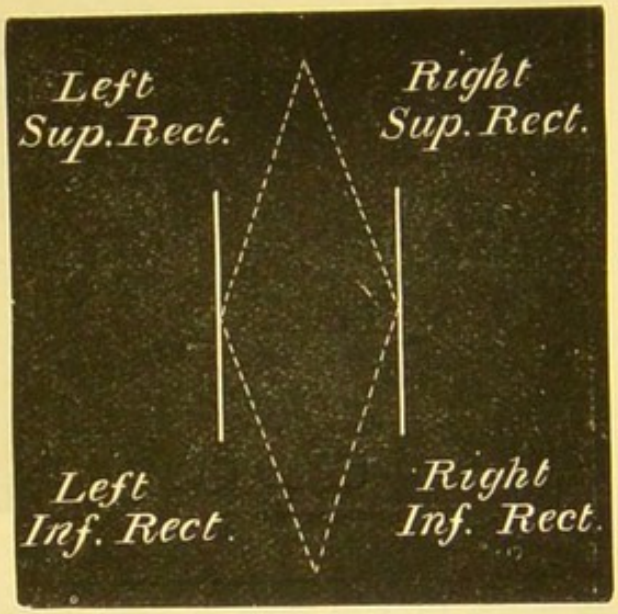


FIG. 152.

As may be imagined from the foregoing, it is often difficult in practice to keep clearly before one's mind the different actions of the orbital muscles, and, from the character of the diplopia, to deduce the paralysis which may be present. An aid in this respect has been provided by Dr. Louis Werner[1] by means of two diagrams (Figs. 152 and 153).

[1] *Ophthalmic Review*, March 1886.

The form of diplopia which characterises paralysis of each muscle, is expressed by the position of the dotted line bearing the name of the muscle. The dotted lines represent the "false images," the continuous lines the "true images."[1]

In the case of the recti (Fig. 152) the false images enclose a lozenge-shaped space situated between the true ones, whereas in the case of the oblique muscles (Fig. 153), the true images, which, for the sake of simplicity, are combined in one line, lie between the four "false images," which diverge from one another so as to form an X. It will also be noted that the dotted lines extend upwards and downwards beyond the others, indicating respectively that the "false images" are higher or lower than the true ones. Another fact which the diagrams indicate is that, in the case of the muscles represented in the upper halves of the figures, the diplopia occurs in the *upper* part of the field of fixation, or, in other words, in upward movements of the eyes. A similar rule holds good with regard to the lower halves.

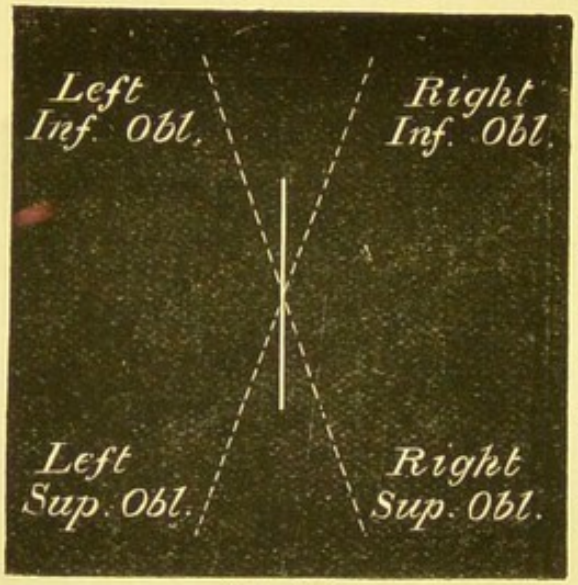


FIG. 153.

The method of using the diagrams will be better understood by taking a particular muscle as an example. Suppose, for instance, that we wish to know what kind of diplopia results from paralysis of the *left inferior* rectus; it is simply necessary to look at the *left inferior* portion of Fig. 152 (recti), which gives the diplopia. If we analyse this we find: (1) That the diplopia is "*crossed*," for the false image corresponding to the

[1] The "false image" corresponds to the affected eye, and the "true image" to the sound eye.

left eye is on the *right* of the true image, *i.e.*, the right image corresponds to the left eye; (2) That the false image has its *upper end inclined towards the true one*; (3) That the false image is *lower* than the true one, for the dotted line extends *lower* than the other one; (4) That the diplopia occurs in *downward movements* of the eyes, for it is in the *lower* half of the diagram that the false image lies.

The same method applies to the other recti: the diplopia for the *right upper* rectus is found in the *right upper* quadrant, and so on for the rest.

The same rules also apply to the obliques (Fig. 153), with one difference. The recti move the eye in the direction indicated by their names, the superior moving it upwards, and the inferior downwards; but in the case of the obliques the reverse takes place, the superior oblique moving the eye downwards, and the inferior upwards. Therefore, for the *superior* obliques we must look at the *lower* half of Fig. 153, and for the *inferior* obliques at the *upper* part.

This is an extremely simple method. By bearing the figures in mind, it is possible to tell immediately what kind of diplopia would result from paralysis of any one of these muscles, and conversely, given the diplopia, to determine to which muscle it is due.

The Causes of Peripheral Paralyses of Orbital Muscles are chiefly of rheumatic or syphilitic nature.

Rheumatic paralysis, to which the external rectus is specially prone, will be noted, if there are symptoms of general rheumatism, or if there is a history of exposure to cold or wet immediately preceding the attack.

Syphilis will be suggested as a cause, if there be a specific history, and that other causes can be excluded. Peripheral paralyses of the orbital muscles due to syphilis are amongst the later symptoms of the disease, and may depend on exostoses or gummata at the base of the skull, or to syphilitic neoplasms, or meningitis, in the course of the nerve. The third nerve

seems to be particularly liable to be attacked by a solitary gumma at the base of the skull, especially at the sphenoidal fissure, ptosis being commonly the first symptom.

Other neoplastic growths can, of course, cause these paralyses in the same way.

Prognosis.—In peripheral paralyses recovery is very frequent; much, however, depending on the nature of the lesion. In cases where a cure is not effected, the antagonist muscle often becomes contracted, and the eye is then rotated permanently and excessively in the corresponding direction. In cases of old standing, a permanent contraction of the muscles of the neck may be brought about, from the inclination of the head which the diplopia has obliged the patient to adopt.

Treatment.—In these cases the medical treatment consists in drugs suitable to the fundamental disease (rheumatism, syphilis, etc.). Local depletion at the temple by the artificial leech in the early stages, and galvanism later on, may be employed with advantage. The most common method of applying galvanism is through the closed lid; but, it is probable that the episcleral method, *i.e.*, with the electrode placed directly over the muscle, is more effectual; and, by aid of cocaine, this can now be done painlessly. Dr. Buzzard's method[1] seems to be a very admirable one. He applies a moistened plate rheophore to the nape of the patient's neck, and connects it with one pole of a Leclanche battery. He then takes the other rheophore, well wetted, in his left hand, and, securing good contact with the skin of his palm, applies the index finger of his right hand to the patient's globe, in the situation of the various external muscles of the eye. The finger is covered with a single thickness of well-moistened muslin; the conjunctiva should be previously rendered insensitive by cocaine. The strength of the current advised is from 1·5 to 2 milliampères, and the alternate application and lifting of the finger, by closing and opening the circuit, gives

[1] *Trans. Ophth. Soc.*, vol. ix., p. 191.

rise to a feeling of a slight electric shock in the terminal point of the finger. The operator should first test the strength of the current upon the patient's cheek. The point of the finger thus employed acts as a sentient rheophore, and can be applied with nicety and delicacy to various parts of the eye, the operator being constantly aware, by the feeling in his finger, of the strength of the current employed.

Passive orthopædic treatment[1] occasionally gives a rapid and brilliant result, while again it is useless. It is performed as follows:—The conjunctiva at the corneo-scleral margin, near the insertion of the paralysed muscle, is seized with a forceps, and the eyeball is drawn in the direction of the muscle, and as far as possible beyond its ordinary limit of contraction, and back again. These movements are continued for about a minute, once a day, and cocaine is used.

Prismatic glasses may be used, either to eliminate the diplopia, or to excite the weak muscle to exert itself. In the former case, the glass selected must completely neutralise the diplopia; but, as it can do so only for one position of the eyes, prisms are rarely employed in this way. In the latter case, a prism slightly weaker than that sufficient to completely neutralise the diplopia is selected; in order that, with a little effort, the weak muscle may be enabled to bring about single vision, and, this effort having been successfully maintained for some days, a still weaker prism is then prescribed, and so on.

It is very important for the patient's comfort, while awaiting his cure, unless a cure by prisms as above described is being attempted, that the affected eye should be covered, so that the distressing double vision may be obviated.

Surgical treatment is justifiable only when other means have failed to restore muscular equilibrium. If the deviation amount to 3 or 4 mm., tenotomy of the antagonistic muscle,

[1] First proposed by Prof. J. Michel, *Klin. Monatsbl. f. Augenheilk.*, 1887, p. 373.

with subsequent tenotomy of the associate muscle in the other eye, will be sufficient; but, if the deviation amount to 5 or 6 mm., advancement of the paralysed muscle, in addition to the tenotomy, may be required. This surgical treatment, applied to the internal and external rectus, gives satisfactory results; but, in case of the superior and inferior recti, it is not so satisfactory, while the oblique muscles should not be operated on.

A peculiar and rare form of peripheral or basal paralysis is Intermitting Paralysis of one Third Nerve, for which Charcot suggested the name of Ophthalmoplegic Migraine. The patients are generally children or young adults, who usually suffer from headache on the side corresponding to the paralysed eye, and sometimes from vomiting. The paralysis may be complete or partial, and the attack varies in its duration from a few days to a few months. Some cases are purely periodical, *i.e.*, in the intervals between the attacks of paralysis all the muscles supplied by the third nerve act in a completely normal manner; while, in other cases, those muscles, or some of them, do not completely recover their functions in the intervals. We are as yet quite in the dark as to the cause of these periodical paralyses of the third nerve. Some hold that the purely periodical cases are of a functional nature, possibly hysterical or reflex, and that the periodically exacerbating cases alone are due to a lesion of the root of the nerve of an undefined kind at the base of the skull, while others are of opinion that both forms depend upon a diseased process at the base. In three cases in which an autopsy was made there was disease of the trunk of the nerve at the base of the skull.

In intermitting paralysis *The Prognosis* of the purely periodical form is favourable, inasmuch as the attacks in the course of time become fewer and less severe, until, finally, they cease entirely. In the exacerbating form the prognosis for complete recovery is less favourable. Out of twenty-six

cases collected by Darquier[1] only one patient died from a cerebral cause.

In view of the obscurity which still surrounds the causation of these intermitting paralyses, their *Treatment* must consist, in each case, in the relief of any general dyscrasia or concomitant symptoms which may be present.

The third class of paralyses of orbital muscles above enumerated—those due to lesions of the nuclei of the orbital muscles, in the aqueduct of Sylvius, and floor of the fourth ventricle—are known by the term

Ophthalmoplegia Externa and also as **Nuclear Paralysis.**—The first of these terms was originally employed to denote those remarkable cases in which all, or nearly all, the orbital muscles of both eyes are paralysed, while the intraocular muscles often remain intact. There can be no doubt, however, that these cases do not differ in their nature from many of those in which, in one eye, several orbital muscles supplied by different nerves, *e.g.*, the third and fourth, are wholly or partially paralysed; or, where all the orbital muscles in one eye are wholly or partially paralysed; or, where in each eye muscles supplied by the same nerve, *e.g.*, both sixth nerves, are wholly or partially paralysed; for such cases are often mild forms of the disease, or else stages in its development. At one time it was considered essential for the diagnosis that the intraocular muscles should retain their functions, but cases occur in which the sphincter iridis and ciliary muscle are paralysed.

When these two latter muscles alone are paralysed, the condition is called Ophthalmoplegia Interna. When both they and groups of orbital muscles are paralysed, the terms Ophthalmoplegia Interna et Externa, or Ophthalmoplegia Universa, are employed.

[1] *Annales d'Oculist*, Oct. 1893, p. 257. This paper contains a complete bibliography.

The term Nuclear Paralysis indicates any orbital paralysis due to a lesion of the nuclei of the orbital nerves in the pons, and ophthalmoplegia externa comes within this category.

Ptosis, even in cases of complete binocular ophthalmoplegia externa, is often incomplete, and it is remarkable that in some chronic cases, without any improvement in the condition itself, the diplopia, which was at first present, quite disappears.

Occurrence and Progress.—The condition may be congenital, or may make its appearance soon after birth, and may remain permanently without becoming complicated with any further disturbance. Congenital ptosis, which is frequently combined with loss of power in the superior rectus, and is usually binocular, is of this nature. But Nuclear Paralysis is more commonly seen as an acquired condition in childhood or in adult life, either in an acute or chronic form. Marked cerebral lethargy is often seen with both forms, and the tendon reflexes may be defective.

Acute Nuclear Paralysis is due either to an acute inflammatory process in the nuclei—comparable to the process which produces polio-myelitis anterior acuta; and hence it is called by Byrom Bramwell polio-myelitis acuta—or to hæmorrhagic lesions.

The acute inflammatory cases are apt to have a sudden onset, attended with fever, headache, vomiting, and convulsions, which may subside after a few days, leaving only the ophthalmoplegia behind; and this, too, after a lengthened period, may undergo cure, partial or complete. Sometimes these attacks are complicated with paralysis of the facial nerve, or the diseased process may extend to the spinal cord, and the symptoms of acute polio-myelitis become developed; or, again, acute bulbar paralysis may come on.

Acute peripheral neuritis of the ocular nerves, which is sometimes seen in cases of alcoholic poisoning, may be confounded with acute nuclear palsy. The symptoms of the two states are the same, except that in the cases of

peripheral neuritis there are no head symptoms at the commencement.

The onset of acute hæmorrhagic ophthalmoplegia is sudden, but unattended by headache, vomiting, or convulsions. It takes different courses. Sometimes it is rapidly fatal, again it goes on to softening of the nuclei and becomes chronic, while, again, it undergoes a slow cure.

It is extremely probable that to this hæmorrhagic class the paralyses of orbital muscles belong, which sometimes follow on an attack of diphtheritic sore throat. These paralyses appear in from one to six weeks after the outbreak of the primary affection. The latter need not have been of a severe kind; indeed, sometimes patients are unaware that they have had a sore throat. These diphtheritic paralyses always recover in the course of some weeks.

In diabetes, paralyses of orbital muscles are not very uncommon, and are probably to be classed as nuclear. The same may be said of orbital paralyses in lead-poisoning and in epidemic influenza ("la grippe"). Other causes are cold, poisoning by nicotine, sulphuric acid, carbonic oxide, and tainted meat.

The Prognosis in all these instances is favourable.

Chronic Nuclear Paralysis (Chronic Poliencephalitis Superior of Wernicke) is much more common than the acute form. It depends on a degenerative atrophy of the nerve nuclei, analogous to that which occurs in progressive muscular atrophy and in chronic bulbar paralysis. The onset is gradual, the loss of power in the muscles being at first very slight, but ultimately complete paralysis of the affected muscles results. There is no fever nor any cerebral symptom. The condition may become associated with chronic bulbar paralysis, with progressive muscular atrophy, or with locomotor ataxy. But this is not so liable to occur in infants as in adults.

In some cases there may be partial paralysis of the

orbicularis palpebrarum,[1] which according to Mendel is innervated from the third nerve nucleus through the facial nerve, along with other muscles of the oculo-facial group (frontalis and corrugator supercilii).

Coarse lesions, especially tumours of the pons and of its neighbourhood which press on it, may produce orbital paralyses closely simulating those due to nuclear lesions. But here the paralysis is only one of the symptoms in the case, which are likely to include headache, vomiting, optic neuritis, hemianopsia, hemiplegia, etc. Softenings, patches of disseminated sclerosis, and internal hydrocephalus with over-distension of the aqueduct of Sylvius, are other lesions which may give rise to similar orbital paralyses, but which cannot be regarded as true nuclear ophthalmoplegia. The mode of onset, and the concomitant symptoms of each case, must serve as our guides in arriving at a diagnosis, which will sometimes be difficult enough.

Conjugate Lateral Paralysis of the eyes is a symptom which may be caused by a lesion in the pons. We believe that the voluntary motor impulses, coming down from the cortex to produce associated lateral motions of the eyeballs —*i.e.*, action of the external rectus of one eye, along with action of the internal rectus of the other eye—first reach the nucleus of the sixth nerve, and then pass on, through fibres called the posterior longitudinal bands, under the corpora quadrigemina, and join with the fibres of the opposite third pair for the supply of the internal rectus of that side. The sixth pair of one side supplies in this way the external rectus of its own side, and, to a slight extent, the internal rectus of the opposite side; and it is quite probable that similar decussations may exist in the nerve supply of other orbital muscles. Hence a lesion, at, let us say, the left sixth nerve nucleus, would paralyse the conjugate lateral motions of the eyes towards the left side; and there would,

[1] Hughlings Jackson, *Lancet*, July 15th, 1893.

in consequence, be conjugate lateral deviation of the eyes towards the right—the eyes look away from the lesion. In conjugate paralysis, or deviation, whether due to a pontine lesion, or, as in a later paragraph, to a cerebral lesion, the combined action of the internal recti for the purpose of convergence of the eyes is retained.

Paralysis of the orbital muscles from nuclear disease may occur in Locomotor Ataxy, Disseminated Sclerosis, General Paralysis, and more rarely in Exophthalmic Goître and Severe Multiple Neuritis.

Fascicular Paralyses are mainly distinguished by the presence of other symptoms due to involvement of neighbouring structures, and are rarely symmetrical. Vertigo is common with third nerve paralysis, owing to implication of the red nucleus in the tegmentum which is connected with the superior peduncle of the cerebellum.

Cerebral Paralysis of Orbital Muscles form the fourth and last of the classes enumerated. They include all the orbital paralyses due to lesions above the nuclei, *i.e.*, in the cortex, corona radiata, or internal capsule. They are usually associated with other symptoms which aid us in localising, more or less accurately, the lesions which cause them. These paralyses are always physiological, associated, or conjugate, as they are variously and with equal correctness termed—they are, in short, paralyses of motion rather than of muscles. Conjugate lateral paralysis—loss of power of motion of the eyes to one side or to the other, while the power of convergence of the optic axes is retained—is by far the most common form of this symptom. We do not as yet know where the cortical centre for the associated lateral motions of the eyes is situated.[1] But, even if we did

[1] The centre has been placed by various authors in the inferior parietal lobule (Wernicke, Henschen, Munk, etc.), and in the second frontal convolution (Ferrier, Horsley, and Beevor). But stimulation of the centres of vision (occipital lobe) has also been found to produce

know its position, it is not likely that much would be gained, so far as clinical localisation of the cerebral lesion is concerned; for this centre, wherever it may be, is extremely sensitive, and is apt to be thrown out of gear by lesions of many different parts of the cortex. Conjugate deviation is, in short, very apt to be a distant symptom, especially in cerebral hæmorrhage, when it is often accompanied by a rotation of the head in the same direction, and lasts only a short time. Moreover, it is thought that, when this centre may happen to be actually involved in the lesion, its function, being largely bilateral, is rapidly taken up by the opposite hemisphere; and, hence, even when conjugate lateral deviation plays the part of a direct cortical symptom, it can never be recognised as such, owing to its evanescent character. In paralysing lesions the deviation of the eyes is of course towards the side of the lesion—the eyes look at the cerebral lesion, as Prevost has expressed it—while, in irritating lesions, the spasm of the affected muscles causes the deviation to be from the side of the lesion. These conditions are the reverse of what happens in conjugate lateral deviation due to lesions in the pons (p. 500), and we are thus enabled to differentiate between lesions in the two positions.

There are four possible cases:—

Cerebral Lesions	Destructive.	Eyes turned	away	from paralysed side.
	Irritative.	„	„	towards convulsed side.
Pontine Lesions	Destructive.	„	„	towards paralytic side.
	Irritative.	„	„	away from convulsed side.

The cerebral cases show that the centre for associated movements is on the opposite side of the brain, *e.g.*, in movements of eyes to the left, the left external rectus and right internal

conjugate movements (Schaefer, Munk), and these have been regarded as reflex by some; but Knies holds that the visual centre contains the motor centre as well. Moreover, it is stated that the visual cortex contains motor pyramidal cells.

rectus are innervated by the right hemisphere of the brain, consequently a destructive lesion here would produce paralysis of the left side of the body, and of the associated movements of the above orbital muscles, and therefore the eyes would be drawn to the right by their opponents, *i.e.*, away from the paralysed side. A destructive lesion of the right side of the pons would also, of course, produce paralysis of the left side of the body; but, involving the right sixth nucleus, it would cause paralysis of the associated movements of the right external rectus and left internal rectus, and, consequently, the eyes would be drawn to the left by the opponents, *i.e.*, towards the paralysed side.

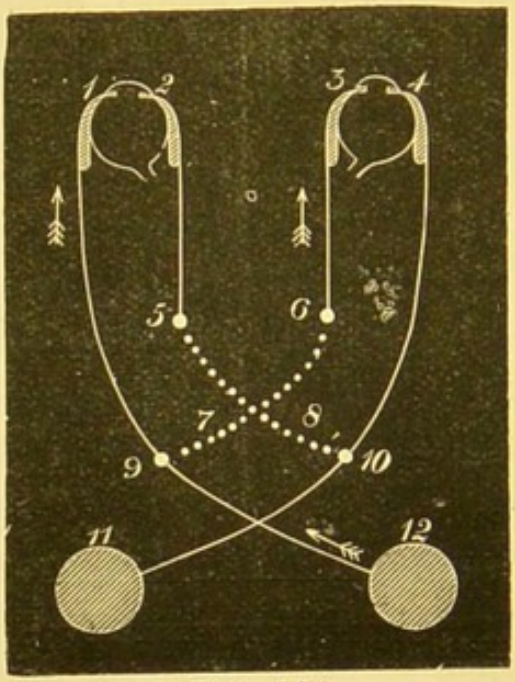


FIG. 154.

1. Left Ext. Rectus; 2. Left Int. Rectus; 3. Right Int. Rectus; 4. Right Ext. Rectus; 5. Nucleus left third nerve; 6. Nucleus right third nerve; 7 and 8. Post. longitudinal bands from sixth nerve to opposite third nerve; 9. Nucleus left sixth nerve; 10. Nucleus right sixth nerve; 11 and 12. Left and right cortical centres. An impulse starting from 12 would travel down to 9, and produce an associated movement of the eyes to the left.

The reverse of the foregoing would occur in irritative lesions. Fig. 154 will serve to illustrate the points referred to.

A destructive lesion at 12, the right cortical centre, involving also motor centres of the body, would cause left hemiplegia; and, since the external rectus of the left eye and internal rectus of the right eye would be paralysed, the antagonists would turn the eyes to the right, *i.e.*, away from the paralysed side. A destructive lesion of the right side of the pons, also producing left hemiplegia, if it involve the sixth nucleus, will produce paralysis of the external rectus of the right eye and of the internal

rectus of the left eye, and then the antagonists would turn the eyes to the left, *i.e.*, towards the paralysed side. It is easy to see how irritative lesions would produce exactly the opposite effects.

Hemianopsia interferes, to a certain extent, with the conjugate movement towards the affected side, in so far as this is guided by visual impressions. According to Knies the difficulty in reading in right hemianopsia is mainly due to this cause.

It seems important here, even at the risk of some repetition, to direct special attention to

The Localising Value of Paralyses of Orbital Muscles in Cerebral Disease.—*Paralysis of the Third Nerve.* As regards this nerve, we are struck with the fact that ptosis, partial or complete, may be present as a focal symptom in cortical lesions—cerebral ptosis, as it is called—without any other third-nerve branch being paralysed. That a separate cortical centre for this branch of the third nerve exists, and that it innervates the muscle of the opposite side, is very probable. The existence of such a centre would not be inconsistent with the view that, as regards the motions of the eyeballs, associated centres alone are present; for although, as a rule, the elevators of the lids are associated in their motions, yet by an effort of the will most people can throw one of them into motion separately, or more than the other. No doubt the power to voluntarily innervate one levator and orbicularis alone varies in different individuals, and in many persons the levator centres are practically associated centres, and probably this is the reason why cerebral ptosis is rather rare. The position of this centre is still an open question, but it is believed to be situated in front of the upper extremity of the ascending frontal convolution close to the arm centre.

Ptosis, then, has no value as indicating the locality of a lesion in the cortex; but it may be of use in distinguishing

a cortical lesion from one situated elsewhere in the brain, for monolateral ptosis, as the only focal symptom, occurs with cortical lesions alone.

It is probable that ptosis, as the result of a cortical lesion, is a distant symptom in not a few of the cases where it is present.

Ptosis on the side of the lesion has occasionally formed a symptom in disease of the pons, without paralysis of the other branches of the third nerve—except, sometimes, in so far as conjugate deviation (*vide supra*) is concerned—and without the third nerve being involved in the lesion.

Again, ptosis, by forming a factor of a crossed paralysis, may serve to localise a lesion in the crus cerebri. When the third nerve is paralysed by a lesion in this situation, it is the rule to find it paralysed as a whole, but paralysis of only some of the third-nerve branches may be produced by a lesion of the cerebral peduncle, and the branch to the levator palpebræ seems to be the one most frequently implicated alone.

In order now to complete this subject of ptosis as a focal symptom, I must refer to a rare form of it which has been described by Nothnagel, and which does not depend on a lesion of the third nerve. It may be called sympathetic, or pseudo-ptosis, and is accompanied by other eye-symptoms, as well as by symptoms of vasomotor paralysis of one side of the body, such as elevation of temperature, and redness and œdema of the skin. In these cases, this author says, there is:—1. Apparent ptosis on the paralysed side, owing to the contraction of the palpebral aperture, but the lid can be raised. 2. Contraction of the pupil on the same side. 3. A shrinking back of the eyeball into the orbit, so that it seems to have become smaller. 4. An abnormal secretion of thin mucus from the corresponding nostril, of tears from the affected eye, and of saliva from the corresponding side of the mouth. Nothnagel states he has found this train of symptoms in lesions of the corpus striatum.

A common sign of disease of the crus cerebri is what is known as crossed hemiplegia. Paralysis of the third nerve on the side of the lesion, with hemiplegia, hemianæsthesia, often facial, and sometimes hypoglossal, paralysis of the opposite side of the body is a frequent form of it. The lesion may implicate all the branches of the third nerve, or only some of them. But the localising value of crossed hemiplegia, as Hughlings Jackson long ago pointed out, depends chiefly on the hemiplegia and paralysis of the cranial nerve coming on simultaneously. If they occur at different times, they may be due to two distinct lesions, neither of which may be in the crus ; for the hemiplegia might be due to a lesion in the hemisphere, and the third-nerve paralysis to a basal lesion of earlier or later date. Yet a few cases have been observed where, with a lesion in the cerebral peduncle, the third-nerve paralysis preceded the hemiplegia by a considerable interval.

That basal lesions are by far the most frequent cause of paralysis of the third nerve is beyond a doubt : and here it is usual, but not constant, to find it paralysed in all its branches. The diagnosis to be made, when direct symptoms are being considered, is, for the most part, between a lesion in the crus and a lesion at the base. We cannot pretend to be able to make this diagnosis with certainty in all cases. Complete paralysis of every branch of the third nerve without any other paralysis is almost always basal ; so also are those cases in which, where there is hemiplegia, it is slight as compared with the degree of the third-nerve paralysis ; and those cases, too, to which I have already referred, where there is an interval between the onset of the paralysis of the extremities and of the third nerve, are apt to be basal. Of course, there may be such a combination of paralysis of the other cerebral nerves with that of the third nerve as to leave no doubt with reference to the basal position of the lesion.

The third nerve may be paralysed by lesions in the inter-

peduncular space, in which case the paralysis may be partial (ptosis alone, or abolition of upward and downward motion only [1]) or complete, single or double. When both nerves are affected there is generally also paralysis of the other orbital nerves, or of the facial; and hemiplegia or hemianopsia may also be present.

Thrombosis of the cavernous sinus invariably produces paralysis of the third nerve, but all the orbital nerves as well as the fifth and the optic nerve may be involved, giving rise to complete immobility of the eye, with loss of conjunctival and corneal sensation. The pupil is usually contracted at first, but later on dilates. The venous obstruction causes exophthalmos, œdema of the lids, and chemosis. Congestion papilla is sometimes found. The general symptoms are rigors, high temperature, and vomiting. Its principal causes are, infective inflammation of the orbital cavity; erysipelas of the face; infective inflammation in the buccal, nasal, and pharyngeal cavities, and of the body of the sphenoid; extension of thrombosis of the sinuses from purulent otitis. The thrombosis in more than half the cases spreads to the other side through the circular sinus. When the invasion occurs from the intracranial direction, pain in some or all of the branches of the first division of the fifth nerve is usually an early symptom.

Third-nerve symptoms—in addition to those included under the headings conjugate deviation, or paralysis, and ptosis—are sometimes distant symptoms. Tumours of the cerebral hemispheres, more particularly if accompanied by violent general head symptoms, indicating probably high intracranial pressure, are the lesions most apt to produce these distant third-nerve symptoms. As a rule, the slighter the general cerebral symptoms are, the more likely are the third-nerve paralyses to be direct symptoms. This rule, indeed, applies to other, as well as to third-nerve, focal symptoms.

[1] Uhthoff, v. *Graefe's Archiv*, xl. 1.

Paralysis of the Fourth Nerve, when combined with paralysis of other motor eye-nerves, is difficult to recognise; and consequently, in such cases, it supplies but little aid for localisation. Solitary paralysis of this nerve, as a symptom of cerebral focal lesion, is extremely rare. Nieden has placed a case on record, in which paralysis of one fourth nerve was the only focal symptom to which a tumour of the pineal gland, of the size of a walnut, gave rise. But the isolated fourth-nerve paralysis is more apt to be produced by a basal lesion. Pfungen [1] has pointed out that, in meningitis, exudation in the space between the corpora quadrigemina and the splenium of the corpus callosum may implicate the fourth nerves in the valve of Vieussens, and believes it is prone to do so in tubercular meningitis. In combination with paralysis of the third nerve it speaks for a lesion in the cerebral peduncle, extending back to the valve of Vieussens, and has, I believe, been utilised by Meynert in this sense.

When *Paralysis of the Sixth Nerve* occurs as the only focal sign, it is probably due to disease at the base, or it is a distant symptom. There is no cranial nerve so liable to provide a distant symptom as the sixth. Gowers refers this liability to the lengthened course these nerves take over the most prominent part of the pons, which renders them readily affected by distant pressure. One or both nerves may in this way be paralysed. Wernicke states that sixth-nerve paralysis is most apt to be present as a distant symptom when the lesion, especially a tumour, is situated in the cerebellum differing in this way from the third nerve, which, as I have said, is more likely to give distant symptoms with a lesion in the cerebral hemisphere.

Paralysis of the sixth nerve, simultaneous in its onset with hemiplegia of the opposite side of the body, indicates a lesion in the pons, usually a hæmorrhage, on the side corresponding to the paralysed nerve. We know that the fifth and facial,

[1] *Wien. Med. Blätt.*, Nos. 8-11, 1883.

and sometimes the auditory, spinal accessory, and hypoglossal nerves, may all, in varying combinations, form one of the elements in a crossed paralysis from a lesion in this position; but, if special localising value is to be given here to the participation of any one cranial nerve, that nerve is the sixth. The paralysis of this nerve, simultaneously with palsy of the opposite side of the body, while other conditions point to an intracranial lesion, speaks then almost certainly for pontine disease.

Basal paralysis of the sixth nerve is frequently double, especially in syphilis. Fracture of the apex of the petrous portion of the temporal may also cause it.

Paralysis of the facial with the sixth is not an uncommon combination caused by a lesion in the pons, which at the same time produces hemiplegia of the opposite side of the body. This combination is a natural one, in view of the close relations of the nuclei of the sixth and seventh nerves. Indeed, Lockhart Clarke, Meynert, and others are of opinion that there is one nucleus which is common to both nerves, a view not shared in by Gowers and others. The manner in which the root of the facial nerve winds round the sixth-nerve nucleus, must also have an important bearing on the occurrence of associated paralyses of these nerves. (See also Lagophthalmos, chap. vii.)

Hemiplegia due to a lesion of the cortical motor region, which might happen to be combined with paralysis of the sixth nerve as a distant symptom, offers no difficulty in its diagnosis from hemiplegia with sixth-nerve paralysis in pontine disease; for, while the latter is a crossed paralysis, the former is homonymous.

Paralysis of the Fifth Nerve with hemiplegia of the opposite side points to disease in the pons. Neuroparalytic ophthalmia is said to be the rule in basal lesions of the fifth nerve, and to occur very rarely in nuclear or fascicular lesions.

The *orbicular sign* may be noticed in some attacks of

apoplexy with hemiplegia after consciousness has returned. It consists in this, that the hemiplegic person, who during health has been able to close each eye separately, and who even now can close both eyes together, or the eye on the sound side alone, is unable to close the eye on the paralysed side by itself. This sign usually passes away after a short time. Sometimes when both eyes are closed it requires a greater effort to bring the eyelids together on the paralysed side. I saw the orbicular sign very well marked and persistent in an obscure case of Dr. Wallace Beatty's, where a gross cerebral lesion was suspected.

Extensive basal lesions, especially the syphilitic, may produce symptoms due to involvement of widely separate structures, without interfering with those which intervene. Hence they tend to implicate several nerves without reference to system or function.

Convergent Concomitant Strabismus.—This is the condition which is popularly known as inward "cast" or "squint." It makes its appearance in children, when they begin to take an interest in small objects, such as toys and pictures; or, a little later, when the first lessons are learned; in short, when they begin to make frequent and prolonged demands on their internal recti and accommodation, most commonly from the age of three to six years.

The term "concomitant" (*concomitatus*, accompanied) is given to it in contradistinction to "paralytic" strabismus; because in it the squinting eye, by virtue of the normal innervation of the associated muscles, accompanies the straight one in all its movements to an equal extent. At the primary position of the eyeballs, in a case of concomitant squint, the parallelism of the visual axes is defective, and, as the eyes are moved from side to side, the defective parallelism continues in the same degree, neither increasing nor decreasing. Moreover, if the straight eye be shaded by the surgeon's left hand, and the squinting eye by this means be obliged to fix

the object of vision—*e.g.*, the tip of the index finger of the surgeon's right hand held up two or more feet distant in the median line—it will be found that the straight eye is now squinting inwards. This deviation of the straight eye is called the secondary deviation, and, in these cases of concomitant strabismus, it is equal in degree to the primary deviation of the squinting eye. Because, the internal rectus of the good eye, being associated in its action with the external rectus of the squinting eye, when the latter muscle is forced, in the foregoing experiment, to roll its eye outwards in order to bring it to fixation, the internal rectus of the good eye, receiving a similar nervous impulse, rolls that eye inwards to the same extent as the squinting eye has been rolled outwards; and the good eye will therefore present, under the covering hand, an internal strabismus of the same amount as that which had previously been present in the squinting eye. This is an important point, for it is an aid in the differential diagnosis of this form of strabismus from the paralytic form, in which the secondary deviation is greater than the primary one (see General Principle No. 2, p. 483.

In order to decide which is the squinting eye, it is merely necessary to direct the patient to look at an object held up in the median line on a level with his eyes, and a few feet in front of him.

In concomitant strabismus, of course, both eyes never squint simultaneously, as one hears it sometimes stated by parents.

Causes.—Squint is never due, as is popularly supposed, to fright, imitation, or naughtiness; nor is it ever brought on by the patient looking at a lock of hair, or other object, which may happen to hang very much to one side.

Donders[1] pointed out, that, in a large proportion of cases of convergent strabismus, the refraction is hypermetropic, and he drew the conclusion, that hypermetropia is to be regarded as the cause of the strabismus, in the follow-

[1] *Accommodation and Refraction of the Eye*, p. 292.

ing way :—It has been shown (chap. i., p. 13), that, with each degree of normal convergence of the optic axes, a certain effort of accommodation is associated. The greater the angle of normal convergence the greater the possible effort of accommodation.

Of this physiological fact, Donders said, the hypermetrope often unconsciously takes advantage, and, in order to brace up his accommodation in an excessive degree for the sake of distinct vision with one eye, he increases the angle of con-

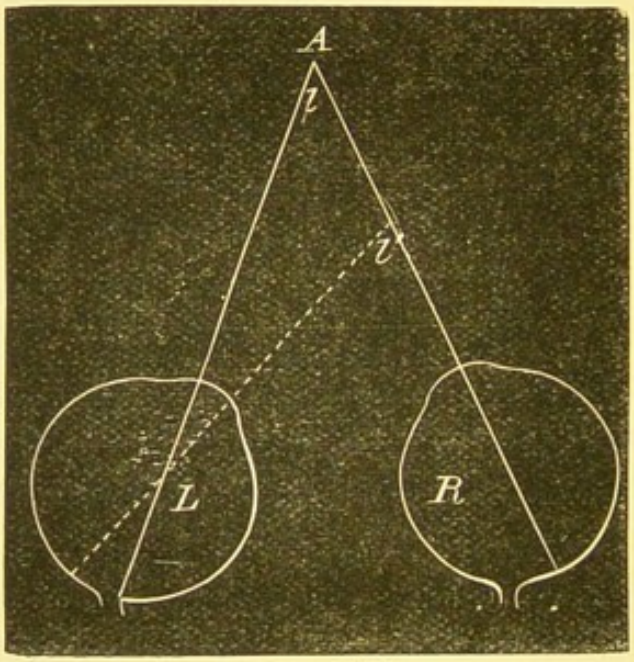


FIG. 155.

vergence of the optic axes by rotating the other eye (*L*, Fig. 155) somewhat inwards. The angle *l'* is thus made larger than the angle *l*, and the effort of accommodation normally belonging to the angle *l'* is obtained for the eye *R*, which, consequently, receives a clearer image of the visual object *A* on its retina. But, inasmuch as all hypermetropes do not squint, Donders considered that there were contributing circumstances which caused each hypermetrope to unconsciously decide between distinct monocular vision with strabismus, and indistinct binocular vision. The latter, he said, is likely to be preferred if the condition of the refraction and the acuteness of vision is

the same in each eye; while, if the retinal images differ much, by reason of one eye being more ametropic than its fellow, from nebulæ of cornea, or from other causes, the desire for binocular vision would be less strong, and the imperfect eye would deviate inwards for the sake of the resulting increase of accommodation in the perfect eye.

It is admitted on all hands that hypermetropia is one of the causes of internal strabismus, but, as Schweigger [1] has pointed out, it is not the only cause, and, probably, not even the principal cause; for the following reasons:—1. If Donders' theory be complete, convergent strabismus must always appear, whenever there is binocular hypermetropia, along with the conditions which reduce the value of binocular vision. But strabismus is often absent while the degree of ametropia is markedly different in the two eyes; or while the acuteness of vision is very defective in one eye. 2. According to Donders' theory, the higher the degree of the hypermetropia, the greater should be the tendency to strabismus; and, yet, clinical observation shows that this is not the case. 3. In periodical strabismus the influence of hypermetropia, and of the accommodative effort, is very evident; and yet these cases only go to show, that, while hypermetropia is very frequently one of the causes of strabismus, it is not the only or most important one; for here, clearly, some factor necessary for the production of a permanent squint is wanting. 4. Donders' theory fails to explain the occurrence of convergent strabismus in emmetropic and in myopic individuals, where, of course, no excessive effort of accommodation is required.

Schweigger considers that a want of equilibrium between the muscles is the chief cause of strabismus (divergent as well as convergent), and that convergent strabismus is mainly due to a preponderance in the power of the internal over the

[1] *Ueber das Schielen* (Berlin, 1881), and *Handbuch der Augenheilkunde*, 5th ed., p. 146.

external recti; or, with equal accuracy one might say, to an insufficiency of the external recti. It would seem that, in hypermetropia, the external recti are apt to be congenitally less powerful than the internal recti; while, in myopia, congenital insufficiency of the internal recti is the more common condition. The internal recti do, however, sometimes preponderate in emmetropia, and even in myopia; and, therefore, convergent strabismus does sometimes occur in these forms of refraction. Whatever be the condition of refraction, strabismus is more apt to be developed, if the value of binocular vision be diminished by imperfect sight in one eye. Schweigger does not, however, give any proofs of this preponderance of certain muscles.

Spontaneous cure of strabismus does sometimes take place, most commonly between the tenth and sixteenth year of age. That it may happen with hypermetropia, and with defective vision in one eye, is strongly against Donders' theory.

According to Hansen Grut's view,[1] convergent squint originates in, and is maintained as the result of, an innervation which induces in the interni a shortening greater in amount than that which is desirable.

Single Vision in Concomitant Convergent Strabismus.—For the most part, these patients do not complain of double vision; although diplopia is the rule in cases of convergent strabismus

[1] *Bowman Lecture*, 1889. Hansen Grut's theory may be summed up as follows:—If the muscles of the eye be completely relaxed, as in sleep, natural or artificial, or in the dead body, the eyes assume a divergent position which is the "anatomical position of rest." When we are awake the eyes are kept parallel by an effort of the internal recti, but from habit it becomes an unconscious effort. In most instances, if one eye be occluded when looking at a distant object, they remain parallel or become slightly divergent, but rarely convergent; this position is the "functional position of rest." Convergent Strabismus is an unusual functional position of rest of the eyes, due to excessive innervation of the internal recti, whereas Divergent Strabismus is a passive movement of the eyes towards the anatomical position of rest, as a result of a withdrawal of the innervation of the internal recti.—L. W.

due to paralysis of the external rectus. Why is this? The image of the object looked at, it will correctly be said, must be formed in the squinting eye in each of these kinds of strabismus, on a part of the retina not identical with that in the fixing eye, but lying to the inside of it; and, hence, the image of the object should be projected by the squinting eye to its own side of the true position of the object (homonymous diplopia), and the latter should therefore be seen doubled. It is seen doubled in the paralytic form; why not also in the concomitant form? The only explanation of this circumstance which had been, until within the last few years, put forward, was, that convergent concomitant strabismus being a quasi-physiological condition, the patient's mind involuntarily suppresses the annoying image belonging to the squinting eye, in a manner analogous to that by which, when we are deeply interested in conversation, all extraneous sounds are unperceived, although they, too, must reach the nerve of hearing. This suppression of the image belonging to the squinting eye was believed to be the more easy owing to the indistinctness of the image itself, formed as it is on a peripheral part of the retina, while, in the good eye, it falls on the macula lutea. We often find, moreover, that the squinting eye is *ab initio* more defective (macula cornea, higher degree of hypermetropia, astigmatism, etc.) than its fellow, and it was held that this, too, rendered suppression of its image more easy. Such a suppression of the image is possible, and it no doubt does occur in many cases of strabismus; but it is certain, as pointed out by Schweigger, that it does not occur in all of them, perhaps not even in most of them. It would be beyond the scope of this handbook were I to go into the arguments on this point. Suffice it to say, that, in those cases where suppression of the image of the squinting eye does not take place, a certain participation in the act of vision on the part of this eye, when not too blind, is implied. One of two events takes place in those cases:—Either the region of the retina, on

which, in the squinting eye, the image of the visual object is formed, becomes functionally developed into a spot to a great extent physiologically "identical" with the macula lutea of the straight eye, and then something approaching normal binocular fusion of the images comes about, and hence single vision; or else, diplopia is actually present, although, as a rule, it passes unnoticed by the patient, owing to its having become habitual to him. In some cases the first of these conditions is the actual state, in others it is the second which exists. I shall mention one fact in support of each, but must refrain from entering more deeply into the subject. In support of the first is the occurrence, not rarely observed, of crossed diplopia after operation for concomitant convergent strabismus, even when there is no divergence produced; and in support of the second, the diplopia which intelligent patients often admit, when they are carefully examined with the aid of a red glass before the good eye.

Amblyopia of the Squinting Eye.—In a large proportion of the cases of internal concomitant strabismus, the squinting eye—even where there is no marked astigmatism, and where the media are clear—is amblyopic. Schweigger states the proportion of these amblyopic cases to be 30 per cent., but I believe the percentage to be much larger. It has been a very generally accepted opinion, that this amblyopia is due to want of use on the part of the squinting eye, in consequence of the suppression of the image on its retina, and, hence, it is termed amblyopia ex anopsiâ. If this view were the correct one, we ought always to find only slight amblyopia of the squinting eye in children soon after strabismus comes on; while, it should be of high degree—in fact, the eye should be almost useless—in adults who have not been operated on, and in whom monolateral strabismus had been present since childhood. And yet marked amblyopia may often be found in children in the squinting eye, while in adults the squinting eye often has very good vision—in short, the amblyopia of the squinting

eye is not progressive, as it would be were it ex anopsiâ. Again, many squinting eyes, when the straight eye is covered, instead of fixing the visual object with the macula lutea, remain unchanged in position, or even turn inwards more than before—(amblyopia with excentric fixation)—and, in less well-marked cases of the same sort, although there is no excentric fixation, yet the preference for fixation with the macula lutea is lost, and uncertainty of fixation results, no one part of the retina being more useful for that purpose than another. It is held by many, that this form is characteristic of amblyopia ex anopsiâ, and is the result of the strabismus; but, it is identical with a form of congenital amblyopia often present in only one eye without strabismus (p. 467). A strong argument in favour of amblyopia ex anopsiâ is the improvement which often seems to take place in the vision of the squinting eye by systematic separate use, or after the strabotomy. But it is tolerably certain that, where the improvement takes place, the defective vision has been due rather to retinal asthenopia than to amblyopia; and if, at the outset, patients be pressed to discern the test-types, they often succeed in producing a better acuteness of vision than they at first seemed to possess. In many cases, separate use fails altogether in improving the vision of the squinting eye, even when it is not very defective; a fact which is unfavourable to the amblyopia ex anopsiâ theory. The circumstance that, in alternating strabismus, the sight of each eye is good, cannot be regarded as proof in favour of amblyopia ex anopsiâ rather than against it.

The explanation which Schweigger gives of the very frequent presence of amblyopia in the squinting eye, is, that it is congenital; and, far from being the result of the strabismus, is a factor in its production, just as opacities of the cornea, or high degrees of ametropia, have always been admitted to be.

There are *Three Clinical Varieties of Convergent Conco-*

mitant Strabismus.—1. Periodic. 2. Permanent alternating. 3. Permanent monolateral. Periodic strabismus occurs only when some great effort of accommodation is required. It sometimes is the first stage of permanent monolateral or of alternating strabismus ; but these two latter forms do not always have their beginning in the periodic form, which often continues as periodic to the end of the chapter. In alternating strabismus, the patient squints sometimes with one eye and sometimes with the other. In permanent monolateral strabismus the squint is confined to one eye.

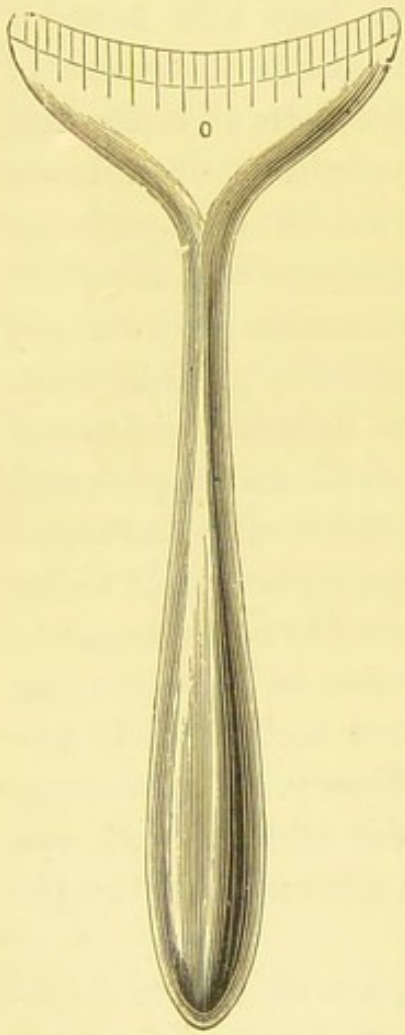


FIG. 156.

Measurement of Convergent Strabismus.—The amount, or degree, of the deviation of the squinting eye from its normal position is not the same in every case, and the size of the squint is measured by one of the following methods. Whichever of them be used, it is important that the patient be directed, during the test, to regard a distant object placed in the median line and on a level with his eyes. If he look at a near object, the squint may be over-estimated, by reason of its increase with accommodation.

1. By the Linear Method, we measure the number of millimetres by which the eye deviates from its normal position. The good eye is shaded, and the squinting eye is caused to fix an object in the median line—by preference a distant object. Close under the margin of the lid a strabometer (Fig. 156) is then placed, so that the 0 point may coincide with a perpendicular let fall from the centre of the

cornea. The shade being removed from the good eye, the squinting eye is allowed to resume its abnormal position, and the degrees recorded on the instrument, under a perpendicular let fall from the centre of the cornea in this position, are read off. They give the amount of the deviation.

2. Hirschberg's Method[1] consists in estimating the degree of deviation by the position of the corneal reflex of a candle flame held straight in front of, and about a foot from, the eye. Where there is no squint, this reflex is situated at, or (with large angle γ) slightly to the inner side of, the centre of the pupil in each eye. In a convergent squinting eye it

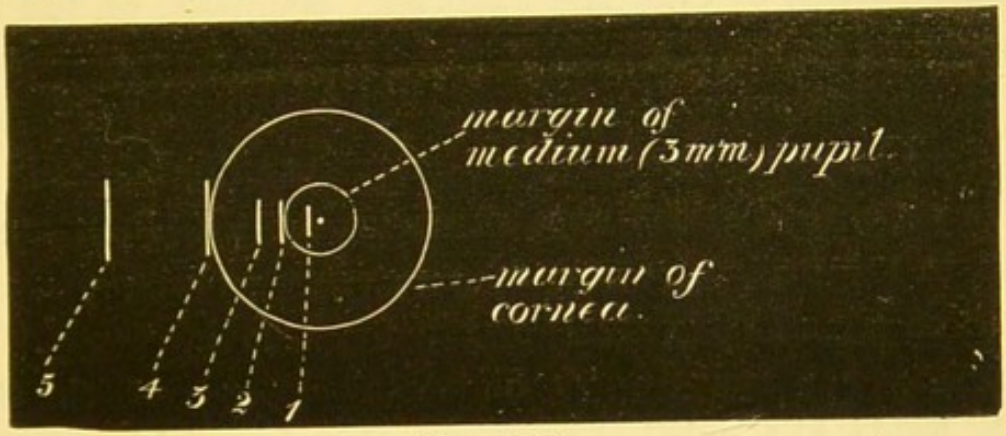


FIG. 157.

is displaced outwards, and Hirschberg recognises five groups of strabismus. Group 1 (Fig. 157 representing the right eye), in which the reflex is nearer to the centre than to the margin of the pupil. This represents a strabismus of less than 10°, and no operation is indicated. Group 2, in which the reflex is at or about the margin of the pupil, representing a strabismus of 12° to 15°, and indicating a simple tenotomy, with occasionally a tenotomy of the other int. rectus. Group 3, in which the reflex is outside the pupillary margin, about halfway between the centre of the pupil and the corneal margin. This represents a strabismus of about 25°, and indicates a tenotomy of the internal rectus, combined with a moderate advancement of the external rectus.

[1] *Centralblatt f. p. Augenheilkunde*, 1886, p. 5.

Occasionally, later on, a tenotomy of the other internal rectus will be required. Group 4, in which the reflex is on or near the corneal margin; representing a strabismus of 45° to 50°, and indicating a tenotomy of the internal rectus, along with energetic advancement of the external rectus, and sometimes a later tenotomy of the other internal rectus. Group 5, in which the reflex is on the sclerotic, between the margin of the cornea and the equator bulbi. This represents a strabismus of 60° to 80°, and requires the combined operation, with strongest possible advancement of the externus. Even this is sometimes insufficient, and a tenotomy of the internal rectus, or even the combined operation on the other eye, may be subsequently required. This is a modification of the linear method, and is a convenient one in routine practice.

3. Priestley Smith measures strabismus by means of a double tape, used in conjunction with the ophthalmoscope, as shown in the accompanying figures. The patient places the ring *P* on one of his fingers, and holds it to his cheek. The observer places the ring *O* on the forefinger of the hand which holds the ophthalmoscope; this keeps his eye at a distance of one metre from the patient's face. He uses his disengaged hand as a fixation object for the patient, holding it edgewise towards the patient, and letting the graduated tape slide between his fingers. A small weight at the end of the tape keeps it stretched as the hand moves in either direction.

Fig. 159 illustrates the measurement of a convergent strabismus of the right eye. The patient, seated below the lamp and holding the tape as above described, is told to look at the mirror. The observer, holding the ring *O* and the mirror in the right hand, throws the light on the patient's left eye (*L*), *i.e.*, the fixing eye. He sees the corneal reflex in the centre of the pupil, and knows thereby that this eye is fixing properly. He then throws the light on the right eye (*R*), and sees the reflex situated excentrically outwards, and knows that this eye deviates inwards. Taking the graduated

tape between the fingers of his left hand, and telling the patient to watch this hand, he moves it outwards along the tape (see Fig. 158), and meanwhile watches the corneal reflex in the deviating eye. When this latter reaches the middle of

FIG. 158.

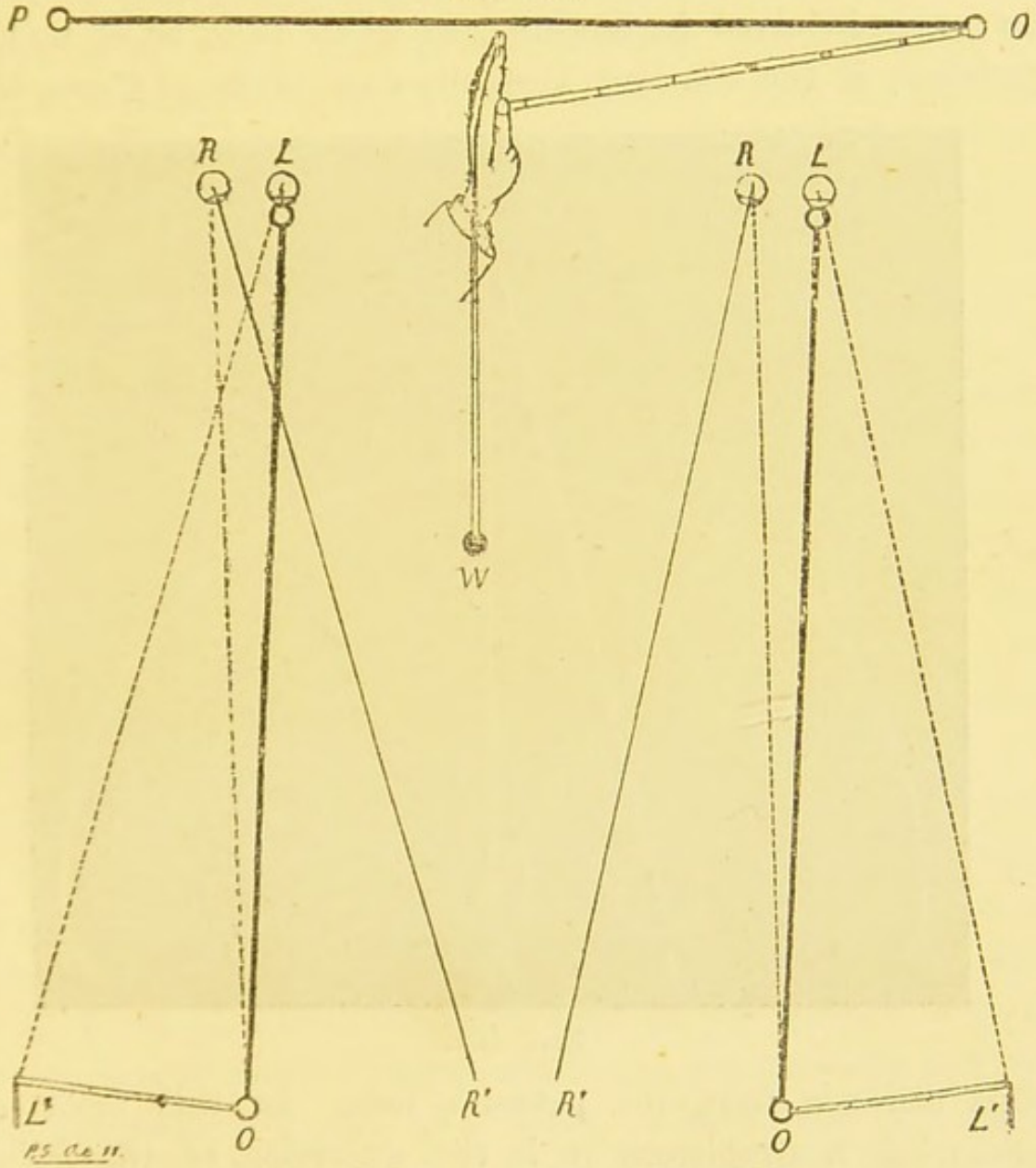


FIG. 159. FIG. 160.

the pupil, he reads the position of the hand upon the tape. The axis of the deviating eye (R) has moved from R' to O, through the angle $R'\ R\ O$. The axis of the non-deviating eye (L) has moved through an equal angle $O\ L\ L'$. The angular movement of L, as measured by the tape, equals the angular deviation of R.

Fig. 160 illustrates the measurement of a divergent strabismus of the right eye. The hands must be reversed, but the principle is the same as before.

The graduated tape is a substitute for a graduated arc of a circle, but does not exactly correspond with such an arc: the error involved is, however, so small as to be of no importance, if the observer keep his two hands at about the

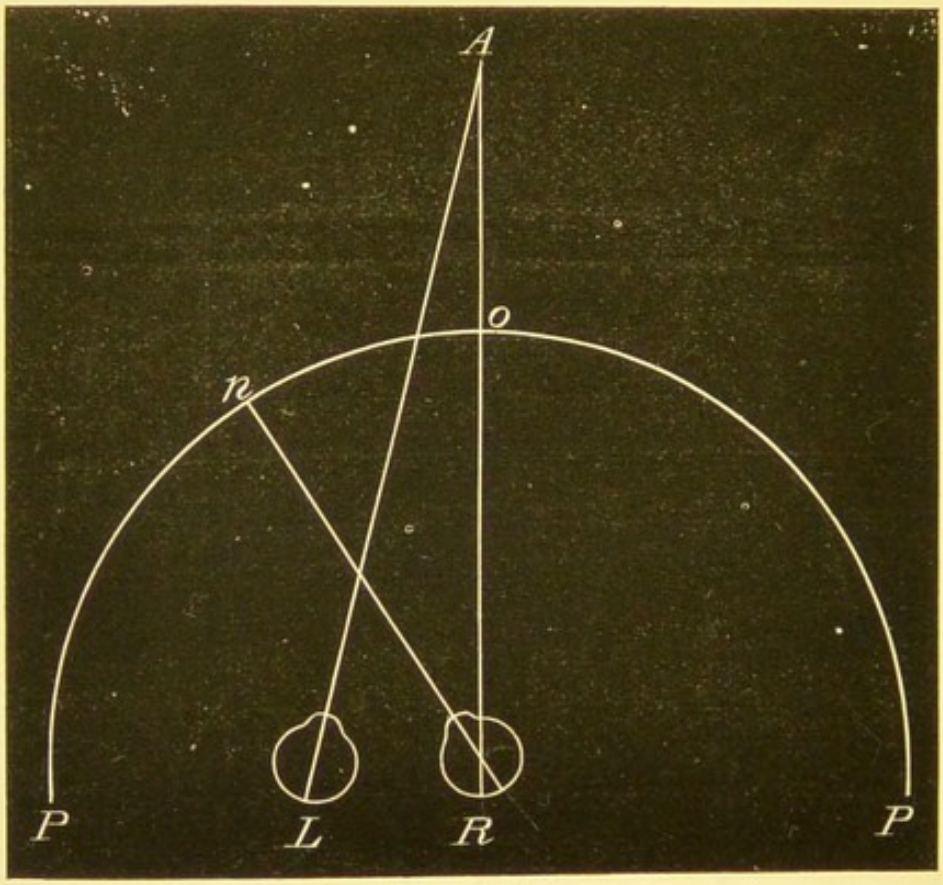


FIG. 161.

same distance from the patient's face. In this mode of measuring a strabismus it is the excursion of the fixing eye which is actually measured, and the excursion of the deviating eye is supposed to be equal to it. If the excursions of the two eyes are unequal, the result would be at fault. The method, though difficult to explain in words, is very quick and satisfactory in practice.

4. The Angular Method.—The object aimed at here is to determine the size of the angle, which the visual axis of the

squinting eye makes, with the direction it should normally have. For this purpose a perimeter is employed. Let us suppose that the right eye (*R*, Fig. 161) be the squinting eye, and that *P o P* be the arc of the perimeter. The patient is placed at the instrument, as though the field of vision of his squinting eye were about to be examined. He is directed to look at a distant object (*A*) with his good eye (*L*). The visual line from *R* should now pass through the point *o*, but it passes through the point *n*, and therefore *o R n* is the angle of the strabismus. The surgeon finds the position of *n* by carrying the flame of a candle along the perimeter, until, with his eye placed behind the flame, he finds that the corneal image of the flame occupies the centre of the pupil. The flame itself will then be at *n*, and the size of the squint-angle may be read off there. This gives us the optical axis of the eye; but, to be strictly accurate, we must remember that the position of the visual axis is what we require, and that it lies a few degrees farther inwards, according to the size of the angle γ. The angular method is now in general use instead of the linear method, than which it is more accurate.

5. A good subjective method for determining the dimension of a strabismus, but which can only be used where diplopia is present, is what may be called the Method by Tangents. Upon a wall of the consulting-room in a horizontal line, and so as to be on a level with the eyes of the patient, who is placed about 3 metres from the wall, are, permanently marked out, tangents of angles of 5° each, as seen from the place where the squinting eye is. Exactly opposite to the squinting eye is 0°, while towards the right and left the points are marked up to 45° or more. The flame of a candle being held at 0°, and one eye of the patient being covered with a red glass, he is called on to indicate the position of the image belonging to the squinting eye, and the number on the wall which corresponds to this gives the angle of the strabismus. For the purpose of estimating paralyses of the orbital muscles,

a similar row of tangents, or several such, may be marked out in the vertical direction. No well-ordered ophthalmic institution should be without this simple arrangement.

Mobility of the Eye Outwards in Convergent Concomitant Strabismus.—This is often defective in the squinting eye, and sometimes also in the fixing eye. The excursiveness of the lateral motions of the eyeball may be measured by the perimeter. Placing the patient as though the field of vision, say of his right eye, were about to be examined, the patient is directed to follow with his eye the flame of a candle carried along the perimeter from 0° towards 90° in the temporal direction, and when it is found that the eye cannot be turned any farther in this direction, the extreme position is noted by the position of the candle at the perimeter. The corneal image of the flame must, of course, be central, when the position of the flame is read off. In a similar way, the mobility of the eye inwards may be measured. In the normal eye the mobility in each direction is about 45°. In strabismus we simply compare the outward mobility of the squinting eye with that of the good eye, to ascertain how much, if anything, the former lacks of its normal amount.

Treatment.—The bearing of hypermetropia on the production of many cases of strabismus, long since suggested the idea of curing the deviation by spectacles, which would correct any existing hypermetropia. The accommodation having been paralysed by atropine, is kept under its influence for some weeks or months; spectacles, which completely correct the hypermetropia and astigmatism, being meantime constantly worn. Should the patient require to use his eyes for near work while under treatment, it is necessary that he should have suitably higher + glasses for his near work. Occasionally, good cures are effected by this means; and, when a periodic strabismus in a child comes under my care, I always think it worth while to attempt its correction in this way; but, in general, it is, by itself, of no use whatever.

Orthoptic Treatment.—To Javal[1] is due the credit of devising this method; but, although he did so some years ago, it is only recently that the treatment has been introduced into practical ophthalmology.

In order that the treatment may be carried out, it is necessary, in the first instance, that the strabismic person should have diplopia. If the latter be not present spontaneously, it has to be developed; and it is sometimes possible, when the sight in the squinting eye is not too defective, to give the patient diplopia, *i.e.*, to make him continuously conscious of the presence of the image belonging to the squinting eye. This may be done by means of exercises with a prism, base downwards, before the deviated eye, and a candle flame as visual object. The exercises are to be repeated daily until diplopia without a prism is established. Javal recommends the following exercise to develop diplopia:—A screen—*e.g.*, a large sheet of cardboard—is held vertically between the two eyes, while the patient is directed to look at a candle flame about 2 metres in front of him. Double vision may immediately appear; but, if it does not, it may be brought out by, now and then, covering the good eye for a moment, or, by placing before it a red glass, which can soon be done without. Less brilliant visual objects are gradually substituted, until, finally, the double vision will continue even when, at first cautiously, the screen is removed.

Double vision having been established, we proceed to enable the patient to fuse the double images, *i.e.*, to obtain binocular vision, and, when we have succeeded in doing this, we have cured the squint. The end in view is best effected by means of a stereoscope, into which, in place of the usual prisms, + 6 D lenses have been introduced. The focal distance of these lenses being about the length of an ordinary stereoscope, rays coming from the slides, and passing through them,

[1] *Annales d'Oculistique*, Juillet et Août, 1871. See also Mars et Avril, Mai et Juin, and Nov. et Déc., for the same year.

fall into the observer's eye as parallel rays; the accommodation consequently is suspended, and, under normal conditions, the visual lines are parallel, as though looking at a distant object. In the normal state the double picture, or diagram, will seem to be single, but to the strabismic patient in whom diplopia is present it will be double. Our duty, then, is to diminish the distance between the pictures, until the patient finds himself just able to fuse the images into a single impression. After a day or two the distance is increased slightly, and so on, until, finally, the normal position is reached. It is needless to say that in these exercises all errors of refraction must be eliminated by the proper glasses.[1]

The pictures used in the stereoscope should be geome-

[1] The existence, or otherwise, of true binocular vision may be ascertained by the simple experiment of giving the patient a book to read, and holding a cedar pencil halfway between his eyes and the page, perpendicularly to the lines of type. If binocular vision be present, the pencil will not offer any impediment to the reading; but, if it be not present, parts of the page will be hidden behind the pencil. The surgeon may prove this by performing the experiment on himself, first with both eyes open (binocular vision), and then with one eye shut.

Another method is that known as Hering's Drop Experiment. A cylinder about 25 cm. long, and wide enough to take in both eyes of a person, is provided, at the opposite end from that placed around the eyes, with two strong wires 18 inches long, which jut out in continuation, as it were, of the cylinder, but which are bent outwards sufficiently to keep them out of view of the patient. Between the ends of these wires a fine thread is stretched, with a small bead fastened at its middle point, so that the bead may occupy the centre of the field when the patient looks through the cylinder. During the experiment the thread is in the horizontal position, and the bead is used as the patient's fixation point. Small balls of different sizes (peas, beans, etc.) are then let fall from a height, one after another, a couple of dozen times or more, some of them in front of the thread, some of them behind it. If the patient have normal binocular vision, he will be able to say each time with certainty whether the ball falls in front of or behind the thread; but, if he have not true binocular vision, if only one eye be used, he will merely guess at the position of the falling ball, and will make frequent mistakes.

trical figures, or specially designed pictures, in order that both surgeon and patient may the more readily recognise their fusion.

Only the very slight degrees of strabismus are adapted for the attempt at cure by orthoptic treatment. A marked deviation will not be amenable to it. Moreover, it makes demands both upon the patience and intelligence of the patient, which are rarely fulfilled, especially in hospital practice. A field more fertile in good results for this treatment is found in the completion of cures, which have been commenced by operative measures.

Operative Treatment.—Division of the tendon of the internal rectus muscle, combined, sometimes, with advancement of the insertion of the external rectus, is the measure which has to be applied in most of the cases which come under our notice. I am strongly opposed to operative interference in patients under five years of age, and very much prefer that they should be seven or eight years old, or even older. Early childhood offers a decided obstacle to the careful adjustment of the operation, and to orthoptic treatment.

In order that the operative proceeding may be adapted to each case, the following points must have been previously noted with care :—*a.* The dimension of the strabismus angle. *b.* The lateral mobility of the eyes, especially the mobility outwards of the squinting eye. *c.* The refraction of the eyes, and the acuteness of vision of the squinting eye, as well as the presence or otherwise of diplopia. The first, in order that glasses for the correction of any hypermetropia may be worn if desirable after the operation ; the second, because, *ceteris paribus*, an operation for convergent strabismus will produce a more marked effect, if the vision in the squinting eye be good, than if it be very defective ; and the third, because the presence of diplopia encourages the hope that binocular vision may be restored.

Rules which will ensure in every case, with absolute

certainty, the desired degree of operative effect, cannot be laid down. The following will be found to answer in the majority of cases, and, if the effect be now and then too great, it can easily be adjusted by bringing forward the internal rectus, or by setting back the external rectus, within a few days after the operation. In every instance it should be the desire of the surgeon to leave 2° or 3° of strabismus behind; for the effect of the operation is apt to increase within a year, and, if absolute parallelism be present at first, divergence may ultimately supervene. The establishment of binocular vision, when possible, would do away with this remnant of strabismus; but, under any circumstances, the latter does not detract from the cosmetic result.

If the vision of the squinting eye be fairly good, and the deviation amount to not more than 15° or 20°, and the power of the external rectus be sufficient, the correction can be effected by the tenotomy of the internal rectus of the squinting eye. A strabismus of 20° will require the free separation of the delicate connections between the anterior surface of the tendon, or capsule of Tenon, and the conjunctiva as far back as the caruncle, in order that the tendon may be free to contract. For a deviation of 15° or less this separation should not be so free, or should be quite omitted; or, if a very slight effect be desired, it can be produced by drawing the conjunctival wound together, after an operation which has been confined strictly to the insertion of the tendon.

If the vision of the squinting eye be fairly good, and the power of the external rectus sufficient, and if the squint be more than 20°, it is advisable to divide the proceeding between the eyes, *e.g.*, if it be 30°, about 20° are corrected by tenotomy of the internal rectus of the squinting eye, and the remainder by tenotomy of the internal rectus of the fixing eye. If desired, the effect of the tenotomy in one or both eyes may be increased by a suture passed through a fold of conjunctiva at the outer side of the globe, and tied tightly.

If, although the vision of the squinting eye be good, and the deviation not more than 20° or 25°, there be marked loss of power of the external rectus muscle, tenotomy of the internal rectus alone will often lead to disappointment, and a good result will require this tenotomy to be combined with advancement of the external rectus; the operative measures being confined to the squinting eye. But advancements in such cases as this must be very cautiously carried out, as an excessive effect may easily be produced. The external rectus should be but slightly brought forward.

If the deviation exceeds 35°, even when there is good vision in the squinting eye, and no loss of power in the external rectus, tenotomy of the internal rectus of each eye is rarely sufficient, and, as a rule, advancement of the external rectus of the squinting eye must be combined with these measures.

With a deviation of 30° to 35° and loss of power in the external rectus, the demand for advancement of the external rectus becomes more imperative. The correction of squints of 40° and more are, in every instance, to be effected by tenotomy with vigorous advancement in the squinting eye, and subsequent tenotomy of the internal rectus in the good eye.

In cases where the vision of the squinting eye is much reduced, the deviation great, and the insufficiency of the external rectus marked, the combined operation in one or both eyes is the proper proceeding.

Mode of Operating for Strabismus. *Tenotomy.*—The instruments required for this operation are, a spring-stop speculum, a small-toothed forceps, a blunt scissors somewhat curved on the flat, and two strabismus hooks (Fig. 162).

FIG. 162.

The eye having been thoroughly cocainised, the patient is placed on his back, the surgeon standing in front of him and on his left-hand side, if the left eye is to be

operated on; or behind him, if it be the right eye. The speculum is then applied, and the conjunctiva over the insertion of the tendon of the internal rectus is seized with the forceps, and incised with the scissors between the forceps and the eye. Into the opening thus made the points of the closed scissors are inserted, and, with a snipping action, a passage is made through the subconjunctival tissue; from the conjunctival aperture to the upper border of the tendon in case of the left eye, or to its lower border in the right eye. The scissors are now laid aside, but the conjunctiva is still held in the forceps; and, with the right hand, the point of the hook is passed through the opening and along the passage, until the edge of the tendon is reached. The point of the hook being kept in contact with the sclerotic, the instrument is then turned rapidly round and under the tendon, and is brought close up to the insertion of the latter into the sclerotic, care being taken that the whole breadth of the tendon lies on the hook. The forceps are now laid aside, and the hook carrying the tendon is transferred to the left hand. One blade of the scissors (held in the right hand) is now inserted between the globe and the tendon, and the latter is completely divided at its insertion. The second hook is then employed for searching, above and below, for any strands of the tendon which may be left undivided; the test for complete division being, that the hook can be brought up without obstruction to the margin of the cornea. If the smallest segment of the tendon be left undivided, the result of the operation is apt to be unsatisfactory. Immediately after the operation a marked diminution in the mobility of the eye inwards should be looked for; as this motion can now only take place by aid of any remaining connective tissue attachments of the muscle to the eyeball and capsule of Tenon. If this defect in motion be not present, or in only a slight degree in comparison with the supposed extent of operation, it may

be concluded that the tendon is imperfectly divided, and a new search for undivided filaments must be made. To estimate this loss of motion, it is necessary before the operation to note the degree of mobility of the eyeball inwards, and to compare it with the inward motion of the other eye.

The effect of the operation may be diminished, if found necessary, by drawing the edges of the conjunctival wound together with a suture, the tendon being thus prevented from uniting with the globe so far back. The more conjunctiva we include in the suture at each side of the wound, the more will the effect of the tenotomy be reduced. This restricting suture should be applied when the immediate result of the tenotomy is greater than expected or desired.

As the edges of the conjunctival wound cannot be accurately adjusted with sutures, none are applied for that purpose. They are only used, as above, to diminish the operative effect; or, when an extensive loosening of the subconjunctival tissue has been performed, to prevent sinking of the caruncle.

The Subconjunctival Operation for Strabismus, proposed by the late Mr. Critchett, is performed as follows:—A fold of conjunctiva is seized, close to the lower margin of the insertion of the muscle, and incised with a blunt-pointed scissors, so as to expose the tendon. A strabismus hook is passed through the opening, and under the tendon. The scissors is now inserted and opened slightly, one point being kept close to the hook while the other is passed between the tendon and the conjunctiva, and the tendon is divided close to its insertion. This method is very generally adopted by English surgeons. For myself, I prefer the operation (von Graefe's) previously described, as it much more readily admits of modifications of the effect.

In von Arlt's Method, instead of a hook being passed under the tendon in the first instance, it is seized with the forceps with which, just before, the conjunctiva had been raised. In

other respects the proceeding is the same as von Graefe's, than which it is said to be less painful.

The immediate and ultimate effects of a tenotomy are by no means identical. Immediately after the operation the effect is very marked, owing to the loosening of the tendon from its insertion. In a few days, when it becomes re-attached, the effect diminishes, and in the course of some weeks there is again an increase in the effect, and this increase continues for about a year, as above stated.

The ultimate result may, with tolerable certainty, be estimated immediately after the operation, by testing the power of convergence. If the patient be directed to look with both eyes at the surgeon's finger held in the middle line, and it be approached to within 12 or 15 cm. of his nose, and if the convergence of the eyes can be maintained at that distance, the effect will not be too great. But if, at a distance of from 18 to 20 cm., the operated eye ceases to converge, or begins to diverge, or if even at 12 cm. the convergence, although accomplished, cannot be maintained for more than a few moments, and that then the operated eye deviates outwards, ultimate divergence may be expected, even though the actual position of the visual axes be correct. A restricting suture must be applied in such cases.

Sometimes, although the patient converges up to 12 cm. satisfactorily, and maintains the convergence at that distance for some moments, the eye will then rotate inwards. In such cases there is apt to be a recurrence of the strabismus.

Advancement.—In cases of convergent squint, in which it is desirable to combine advancement of the external rectus with tenotomy of the internal rectus, the latter is done first, as above described, at the same sitting.

An opening is then made in the conjunctiva immediately over the insertion of the external rectus, and as long as the breadth of the tendon. The band of conjunctiva between the opening and the cornea is separated up with the scissors from

the sclerotic, for to it the tendon has to be fastened later on. A strabismus hook is now passed under the tendon, and brought well up to its insertion; care being taken that the whole width of the tendon is held on the hook. A needle carrying a fine silk suture is introduced from its upper margin between the tendon and sclerotic, and passed through the tendon at its middle line. In the same way, another suture is passed behind the tendon from its lower margin, and through it, close to the first suture. Each of these sutures is knotted firmly on the tendon, a long end being left to each (Fig. 163). The

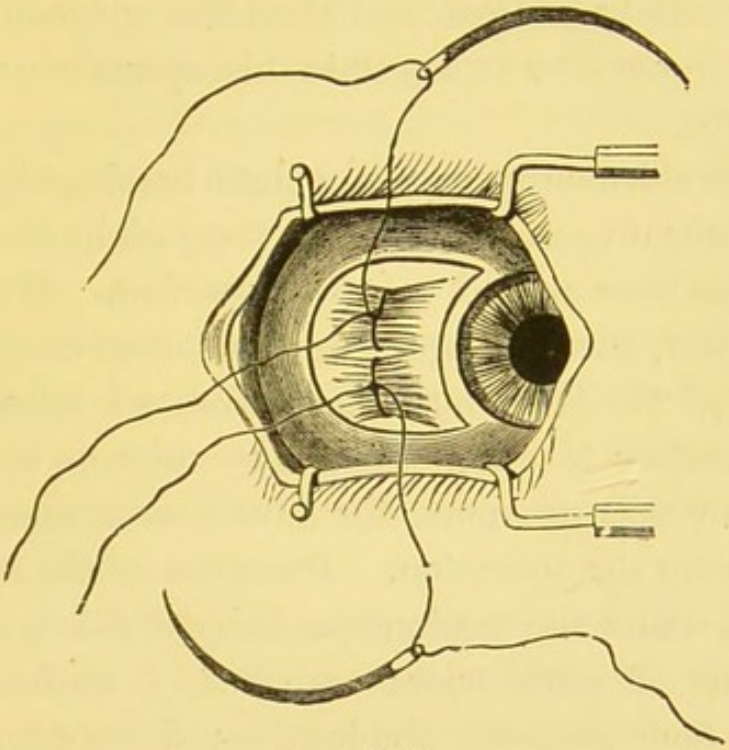


FIG. 163.

tendon is separated off with the scissors from the sclerotic, close to its insertion. The sutures are passed through the conjunctival flap in the direction of the muscle, and are respectively tied with their own ends. A greater or less effect is produced, according as the sutures are placed farther or nearer to the insertion of the tendon, and according as they are drawn more or less tightly. I have found this method perfectly satisfactory.

Immediately after the combined operation is finished, there should be no divergence, nor should there be marked loss of

motion of the eyeball inwards. In either case the effect is too great, and must at once be diminished by an adjustment of the advancing sutures, or a bringing forward of the internal rectus.

In my opinion, even if it lie in the plan of the treatment to supplement the tenotomy, or combined operation, on the squinting eye by a tenotomy, or combined operation, on the fixing eye, both eyes should not be operated on at one and the same sitting. An interval of a fortnight or more should elapse, in order that the true effect of the first proceeding may be accurately gauged, and then the surgeon will be in a position to know how to regulate his operative measures for the other eye.

After a strabismus operation, a light bandage is applied, and is changed morning and evening for forty-eight hours, when, if no suture has been used, it may be discarded. If sutures have been employed, the bandage is retained until they come away.

Dangers of the Strabismus Operation.—I have never seen any inflammatory reaction after a strabismus operation, not even after an advancement, nor have I ever seen any serious accident during the operation. Puncture of the sclerotic with the scissors, while the tendon was being divided, has occurred in the hands of some operators; but, I confess, I cannot understand how such an accident could happen, unless the operator had his own eyes shut. It is also stated that eyes have been lost after squint operations through orbital cellulitis, which, I fancy, must have been brought on by the introduction of septic matter upon the instruments.

Occasionally, a small arterial branch may be divided during the operation, and this, bleeding into the capsule of Tenon, may cause rather alarming exophthalmos. The protrusion goes back in a few days with use of a pressure bandage. I have only seen the occurrence twice.

Sinking of the caruncle, some months after the tenotomy, when it does rarely occur, can be remedied in the following

way :—The conjunctiva is divided vertically about 6 mm. from the caruncle. The inner lip of the wound is raised, a scissors curved on the flat passed in, and the subconjunctival tissue as far as under the sunken caruncle separated. The subjunctival tissue under the outer lip of the wound, and as far as the corneal margin, is loosened in the same way, and the two flaps are brought together with a suture, which includes a sufficiency of conjunctiva to draw the caruncle well forwards.

Treatment subsequent to Operation.—It is generally necessary for the patient to wear the correcting spectacles for his hypermetropia, either constantly, or for near vision only, according as the result of the operative measures makes it more or less desirable to suspend the accommodation. After some months, it is usually possible to leave off the spectacles, except for near vision.

A cure of the strabismus, in the sense of removal of the deformity, can be attained by operation in every case ; and, by itself, affords ample reason for undertaking the operation. But a cure, in the true sense of the term, involves restoration of binocular vision,[1] and this is very rarely obtained by operative measures alone.

Orthoptic exercises with the stereoscope (p. 525) are of great value in completing a cure, which has been almost effected by operation. The deviation, which has been reduced to a minimum by the operation, may sometimes be quite eliminated, and, still more important, binocular vision may sometimes be developed. Where the attending circumstances of the case, both clinical (acuteness of vision, diplopia) and personal (patience and intelligence of the patient), admit of it, an effort should always be made to effect such a cure.

Insufficiency of Convergence, or Insufficiency of the

[1] The importance of binocular vision consists in the fact, that it is chiefly by its aid we estimate distances finely, and observe the shape of objects. Even plane surfaces are seen much more accurately with binocular than with monocular vision.

Internal Recti Muscles, and Divergent Concomitant Strabismus.—In the normal condition, the orbital muscles are in a state of equilibrium, no one muscle, or pair of muscles, having more power over the eyeballs than its fellow.

Insufficiency of the Internal Recti Muscles, or Insufficiency of Convergence, as it is more correctly called, implies a disturbance of this equilibrium. The converging power of the internal recti, in these cases, is so much weakened that they are obliged to make a constant effort to prevent the eyes, or one of them, from becoming divergent, and it is only the demand for binocular vision which stimulates the muscles to this effort.

Muscular Asthenopia is the symptom caused by this insufficiency. The patients complain that, after reading, writing, sewing, or employment at other near work for a time, they begin to find the objects spreading, becoming indistinct, and perhaps doubled. Pain in and about the eyes comes on. These symptoms gradually increase, until the work has to be discontinued.

A great deal has been written within recent years upon the relationship of some nervous diseases, especially epilepsy, to want of power in one or more of the orbital muscles. It has been thought that "eye strain," from want of co-ordination in these muscles, sometimes aggravated, if it did not actually cause, epilepsy; but the outcome of the whole discussion seems to be that there is no such connection.

The diagnosis of insufficiency of convergence can be made by the following methods. The patient is directed to look at the tip of the surgeon's finger held up in the middle line. The finger is brought slowly closer to the eyes until a certain point is reached where the internal rectus of one eye ceases to act, the other eye still remaining in fixation. The first eye, upon the finger being advanced a little more, usually becomes divergent.

Or, if the tip of the finger be held some 20 cm. from the

patient's eyes, and if, with his other hand, the surgeon cover one of the eyes, say the right, while the left is caused to fix the finger-tip, it will be found that the eye under the hand is diverging, and, when the hand is removed from it, it makes an inward motion, in order again to fix the finger-tip. The explanation of this is, that, when one eye is covered, there is nothing to be gained in the way of single vision by an excessive exertion of the weak internal recti; and, consequently, the eye which is excluded from the act of vision, is abandoned to the control of the external rectus, and only returns to its normal position, when, being restored to participation in the act of vision, diplopia would otherwise be present.

FIG. 164.

The following is von Graefe's Test for Insufficiency of the Internal Recti:—A dot with a line drawn vertically through it (Fig. 164) on a sheet of white paper is given to the patient to look at, at his usual reading distance. Before one eye, say the right, a prism of about 10° with its base downwards is held vertically. This, in the normal condition, would produce a double image of the dot, so that the figure would seem to be a line with two dots, the upper dot being the image belonging to the right eye. In insufficiency of the interni, the image of the right eye would not only be higher than that of the left, but it would also stand to the left (crossed double images) more or less, so that here the picture is that of two lines, each with a dot, the upper line and dot standing to the left-hand side (Fig. 165). This crossed diplopia indicates divergence. The explanation of the experiment is as follows:—When a prism is held before the right eye, the possibility of binocular

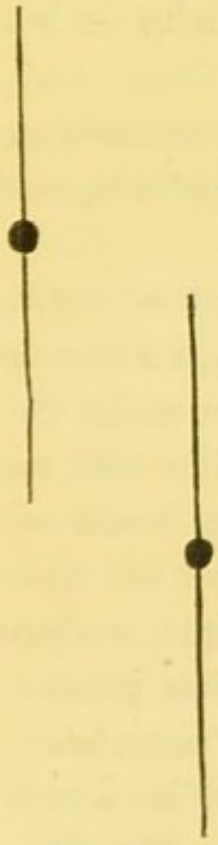


FIG. 165.

vision is removed, and, insufficiency existing, the weak internal rectus of the right eye has no object in greatly exerting itself, and consequently abandons the eye to the traction of the external rectus. Hence the divergence, and the projection of the image of this eye to the opposite side.

The degree of insufficiency existing may be determined by this same experiment. If a weak prism be held with its base inwards before the left eye, in the above case, the images of the lines will appear to be brought closer. By gradually increasing the power of the prism, one will be found which brings the lines together, so that the picture will now be that of two dots over each other on one line. This prism is the measure of the insufficiency.

Landolt estimates the amount of insufficiency of convergence by means of the metre angle and amplitude of convergence. For an account of the method I must refer the reader to his valuable work.[1]

Maddox' Rod Test is an admirable method for ascertaining the condition of the muscular equilibrium of the eyeballs, and for estimating any existing derangement of it.

The apparent lengthening of a flame into a line of light, when looked at through a strong cylinder, is utilised to make the two images so dissimilar that no desire to unite them remains. The chief advantage of this principle is that slight malpositions do not, as with prisms, vitiate the result materially. A glass rod mounted in a circular metal disc, as in Fig. 166, may be used; or a plano-cylinder with a radius of about 20 mm.: or a piece of corrugated glass; or a flat series of thin glass rods side

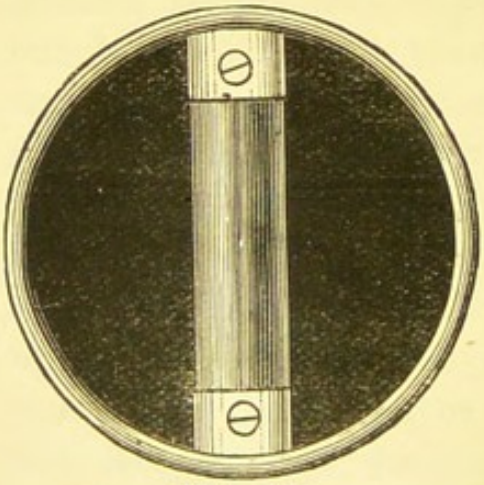


FIG. 166.

[1] *The Refraction and Accommodation of the Eye*, 1886, p. 501.

by side. The best flame is that of a gas-jet turned low, at a distance of 5 mm. or 6 mm., and the appearance is improved by a piece of blue glass before the other eye, to equalise the illumination of the two images. The line of light is at right-angles to the axis of the cylinder. If it pass through the flame the balance is perfect; if not, the defect is measured by the deviating angle of the prism which is found to bring them together, or, preferably, by a lithographed scale, placed with its zero just behind the flame, so that the figure crossed by the line of light gives the deviation in degrees. For vertical diplopia the scale should be vertical, and for horizontal diplopia horizontal. In either case the axis of the cylinder should be parallel to the scale. When the cylinder is vertical, it should be shaded from the light of the window. By placing the patient's head in different positions, the diplopia can be measured in all parts of the motor field. Vertical and horizontal scales should, for this purpose, be fixed on the wall, with their zeroes coinciding at the position of the flame. For near-vision tests, a flame is too large. A scale should be used on a black background, with a small silvered hemisphere or bead fixed to its zero, to be a source of reflected light from the window or from a flame.

This test is also very serviceable in overcoming the suppression of the false image in old squints, and for discovering the latent paresis of an ocular muscle.

Insufficiency of the internal recti is a common attendant upon myopia. It is also found with emmetropia, and even with hypermetropia.

Concomitant divergent strabismus is a further development of the same condition.

Treatment.—In moderate degrees of myopia, the use of such concave glasses as will permit the patient to read at 35 cm. distance may relieve the asthenopic symptoms.

Decentration of these glasses may give further aid. If the glasses be so set in the spectacle frame that their centres

are on the outer side of the visual lines, the inner half of the glasses act as prisms with their bases inwards (chap. i., p. 2), and by them the rays are broken inwards, *i.e.*, towards the macula lutea in each eye, so that a slight divergence may take place without diplopia, etc. In this way the internal recti are relieved. Should the case be one demanding the use of convex glasses (hypermetropia, presbyopia), the decentration must be inwards.

A more perfect and accurate method is that of prescribing prisms, bases inwards, to be worn for reading and other near work. These may be combined with concave or convex glasses, where such are indicated. The prism which is the measure of the insufficiency (see above) is divided between the two eyes. If it be 4°, a prism of 2° is placed, base inwards, before each eye for near work. Very high prisms cannot be ordered, owing to the colour effects they produce; and, in cases where they would be required, the insufficiency can be only partially corrected.

Operative Treatment.—This consists in weakening the too strong external rectus by tenotomy. The danger of the method is, that convergent strabismus with homonymous diplopia for distant objects may result, unless the case be suitable for operation. Only those cases are suitable in which absolute divergent strabismus is present; or those in which, with a prism of not less than 10°, base inwards, before one eye, the flame of a candle at 3 m. distance is seen single, or if it be perhaps doubled for a moment, then becoming again single. When, with such a prism, single vision is present, the external rectus by an effort must have overcome the effect of the prism, and it is admissible to deprive the muscle of the power represented by that effort or prism. If diplopia be produced by a prism of 10°, the tenotomy is contraindicated, for the effect of the latter could not be modified to the slight power of abduction indicated by a weaker prism. A source of error in the ascertaining of this abduction prism which

must be guarded against is, that the patient may suppress the image of one eye, and that his single vision may be merely due to the fact that he is seeing with the other alone. The higher the abduction prism, the more extensive may be the division of the subconjunctival tissue, etc., while with weak abduction the effect must be diminished by a conjunctival suture.

Immediately after the operation there should be a certain amount of convergence, as shown by homonymous diplopia in the middle line for the flame of a candle at 3 m. distance. This convergence, or diplopia, should not be greater than can be corrected by a prism of 10°. Moreover, if the candle be moved from the middle line 15° to the opposite side from the operated muscle (to the right if the left external rectus has been tenotomised), there should be no convergence (no diplopia), and a vertical prism before one eye should only cause double images placed directly over each other. If, by these experiments, it be shown that the operation has produced an excessive effect, the latter must be diminished by a suture drawing the lips of the conjunctival wound together, and including more or less conjunctiva, according to the excess to be corrected. Or, if a suture have already been applied, and the result be still in excess, it must be withdrawn, and a still more restricting suture inserted. In all these cases, convergence must necessarily be present when the candle is carried over to the side of the operated muscle; but this disappears—except perhaps at the very most extreme position on that side—as also the convergence in the middle line, by reason of cicatricial contraction at the new insertion of the tendon; always provided, that the indications for the operation and its performance, as above set forth, have been accurately attended to.

NYSTAGMUS.

This term indicates an involuntary oscillation of the eye-

balls from side to side (the most common form), in the vertical direction, or rotary (caused by the oblique muscles).

It is most commonly found with congenitally defective vision—microphthalmos, coloboma of the chorioid, in albinos, etc.—but it may be acquired, and is often seen in those employed in coal mines. It occurs in about one-half the cases of disseminated sclerosis.[1]

In the congenital cases it is probable that the absence of the stimulus which accurate retinal impressions afford, interferes with the functional development of the co-ordinating centres for the orbital muscles. In coal mines, the very defective light, and the blackness of the surroundings, deprive the miners of any defined retinal impression, and hence their co-ordinating centres are apt to become deranged. But, as it is chiefly those who work in one constrained position on their sides, with eyes directed obliquely upwards, who become affected, it seems likely,[2] that this unnatural and long-continued direction of the eyeballs is an important factor in the production of the affection; indeed, it is probably to a great extent a professional cramp, like writer's cramp. In fact a case of acquired nystagmus in a compositor, due to working in a strained position, has been recorded.[3]

Those patients in whom nystagmus is due to a congenital defect of vision, do not complain of oscillation of the objects they look at; but individuals who become affected with it in later life are much troubled with that symptom, especially at the onset.

Treatment.—In congenital cases which admit of improvement of vision, a cure, partial or complete, is sometimes

[1] According to Gowers (*Diseases of the Nervous System*, vol. i., 2nd Ed.), Nystagmus occurs often in ataxic paraplegia, primary spastic paraplegia and hereditary ataxia, sometimes in severe multiple neuritis and syringomyelia, but rarely in progressive muscular atrophy.

[2] *Vide* S. Snell, *Brit. Med. Journ.*, July 11th, 1891.

[3] Snell, *Trans. Ophthal Soc*, vol. xi., p. 102.

brought about, when the vision improves. If strabismus be present, it should be cured, after which a diminution in the oscillations may result. In miner's nystagmus, the all-important measure is a permanent relinquishment of mine work; and this is frequently followed by satisfactory results.

CHAPTER XIX.

DISEASES OF THE ORBIT.

Orbital Cellulitis, or **Inflammation of the Connective Tissue of the Orbit.**—*The Symptoms* of this affection are :—Erysipelatous swelling of the lids, especially of the upper lid ; serous chemosis ; pain in the orbit, increased on pressure of the eyeball backwards ; violent facial neuralgia ; exophthalmos, with impairment of the motions of the eye in every direction ; and high fever.

Vision is not generally affected, but sometimes it is so from optic neuritis, and then, too, mydriasis is seen. The cornea is often completely or partially anæsthetic.

The surgeon, by pressing the tip of his fourth finger between the eyeball and the margin of the orbit, may feel a more or less resistant tumour. This gradually increases in some one direction, the integument in that position becomes redder, fluctuation becomes pronounced, and the abscess finally opens through the skin, or into the conjunctival sac, the pointing being usually at the upper and inner angle of the orbit. Restoration to the normal state, as a rule, comes about ; but, in some cases, complete atrophy of the optic nerve supervenes.

Causes.—1. Idiopathic (*e.g.*, cold) ; 2. Traumatic (perforating injuries, foreign bodies) ; 3. Extension of inflammation from surrounding parts (erysipelas, diseased tooth, ethmoidal cells) ; 4. Metastasis (pyæmia, metria) ; 5. Sequelæ of fevers (scarlatina, typhoid, purulent meningitis, influenza).

Treatment.—Locally, poultices or warm fomentations ; and, when pus has formed, its earliest possible evacuation,—by pre-

ference from the conjunctival sac. The general constitutional treatment suitable to each case need not be discussed here.

Periostitis of the Orbit.—Acute periostitis has many symptoms in common with phlegmonous inflammation of the orbital connective tissue which generally accompanies it; but may usually be distinguished from the latter inflammation occurring independently, by the fact, as first pointed out by the late Mr. John Hamilton, of Dublin,[1] that, in it, pressure on the orbital margin is painful. The absence of this tenderness, however, is not always conclusive of the absence of periostitis, especially when the latter is restricted to the deep parts of the orbit. In periostitis the eyelids are not usually so swollen as in inflammation of the orbital tissues. Suppuration may take place, necrosis in consequence of detachment of the periosteum may come on, and communications with the neighbouring cavities be formed.

In secondary syphilis, or in later stages of the disease, a syphilitic gumma of the orbital wall may form. This is accompanied by violent frontal neuralgia or headache, increasing at night. Proptosis[2] (*πρό, forwards; πτῶσις, falling*) occurs, with marked loss of motion in the eyeball in one or more directions. This loss of motion is a very characteristic symptom, and serves to assist in the diagnosis between this affection and other orbital tumours. It is probably due to an extension of the inflammation to the connective tissue of the orbit, and to the muscles themselves.

Again, periostitis of a chronic form, and without tendency to suppuration, occurs most commonly in persons with a constitutional rheumatic tendency. It is accompanied by pain in and about the orbit, and there is increased tenderness on pressure of the eyeball backwards. Exophthalmos, and all other outward signs, are usually wanting.

The Prognosis depends much on the seat of the inflamma-

[1] *Dublin Journal of Medical Sciences*, 1836.

[2] Protrusion of the eyeball.

tion. If this be in the deep parts of the orbit, thickening of the periosteum may cause permanent protrusion of the eye-ball; extension of the inflammation of the optic nerve may result in optic atrophy; the orbital muscles, or the nerves which supply them, may be implicated, with consequent paralysis; or, finally, the periostitis may strike into the meninges of the brain. When the inflammation is near the margin of the orbit, early evacuation of pus, if it has formed, reduces the process within safe bounds; and this position is one of less danger in respect of its surroundings.

Causes.—Periostitis of the orbit may be caused by blows or other traumata, by extension from neighbouring cavities, by syphilis, or rheumatism.

Treatment.—Warm fomentations. Exit given to pus, if possible. Constitutional measures.

Caries of the Orbit is very frequently the result of periostitis, but often commences in the bone, and, in either case, is usually due to tubercular disease of the bone. It is also seen in very late syphilis. A trauma is sometimes the immediate cause of its onset.

It may attack any part of the orbital walls, its favourite seats being the margin above and to the outside, or below and to the outside. When it is seated deeply in the orbit, it often causes exophthalmos and pain. At the margin of the orbit it produces œdema and swelling of the eyelids with conjunctivitis, suppuration comes on, and the abscess finally opens through the integument, or conjunctiva. A fistula is apt to remain for a length of time, and, the skin being drawn into this, ectropion of the lid is produced. If a portion of dead bone come away, the resulting cicatrix is liable to maintain the ectropion (p. 232).

Treatment.—The evacuation of purulent collections at the earliest possible moment—if deep in the orbit, by the careful introduction of a long bistouri—the insertion of a drainage-tube, and the regular washing out of the cavity with anti-

septic solutions, until no more rough or bare bone can be felt with the probe.

Injuries of the Orbit.—Wounds of the soft parts in the supra-orbital region, involving the supra-orbital nerve, are believed by some to be capable of producing a reflex amaurosis (p. 467), and many such cases have been recorded under the name of supra-orbital amaurosis. By the light of modern physiology and ophthalmology it is not probable, I might say not possible, that any such reflex could take place, and it seems likely that the blindness in those recorded cases was brought about in some other way, *e.g.*, orbital periostitis, concomitant injury to the eyeball itself, facial erysipelas, intracranial lesions, and so on.

Perforating injuries of the orbit through the eyelids by prods of walking-canes, etc., and the lodgment of foreign bodies in the orbit, are serious accidents. They are liable to be followed by phlegmonous inflammation, or, if a pointed weapon (stick, sword-cane, etc.) has been pushed into the orbit with some force, it may pass through the bony wall and perforate the brain, with fatal result.

It is remarkable what large foreign bodies may be concealed in the orbit. I saw a case in which a bit of wood, $\frac{3}{4}$-inch long by $\frac{1}{2}$-inch wide, lay unsuspected in the orbit for many weeks, without causing any marked displacement of the eyeball.

Treatment.—Foreign bodies should be removed by dilatation of their wounds of entrance, or by the formation of a new passage through the conjunctival fornix—and great care should be taken to prevent the onset of inflammation, or to keep it within safe bounds.

Tumours of the Orbit necessarily give rise to proptosis, and the motions of the eyeball are generally impaired. Vision often remains good until a very late period, unless, as rarely occurs, the optic nerve should early become involved in the growth; but, ultimately, optic neuritis, or optic atrophy, induces blindness. The upper lid, becoming wonderfully enlarged,

protects the cornea from exposure and consequent ulceration, until, at last, the excessive protrusion of the eyeball no longer admits of this, and the cornea sloughs.

When the tumour has attained a certain size, it may be felt by the tip of the finger passed into the orbit, and some idea of its consistency and mobility can be formed.

In every case, the history, the rapidity of growth, the age and general condition of the patient, are important items for consideration.

Cysts.—Dermoid cysts are amongst those most frequently found. They grow slowly, and finally reach very considerable size, and then bulge out between the eyeball and margin of the orbit. Pressure upon this protruding part causes it to diminish, while the exophthalmos is at the same time increased, and distinct fluctuation in the protruding part can be felt. The growth of the cyst is unaccompanied by pain or other inconvenience. The contents are generally either serous or honey-like, and, occasionally, hairs and other epidermic formations have been found in them.

Hydatid cysts also occur in the orbit, and several such cases have been observed in England.

A mucocele originating in the frontal or in the ethmoid sinus may absorb the orbital wall, protrude into the orbit, and give rise to all the signs and symptoms of a true orbital tumour.

Treatment.—The cyst should be freely opened at the most prominent point, evacuated by gentle pressure backwards of the eyeball, and the sac syringed out two or three times daily with an antiseptic solution, until all discharge has ceased. The opening will then close, while the eyeball will already have returned to its place. If the contents of the cyst are solid, or nearly so, it becomes necessary to extirpate it *in toto*. To do this, as in other tumours also, a horizontal incision must be made along the orbital margin through the eyelid, in order that the cavity of the orbit may be reached ; or, two

perpendicular incisions at either canthus through the upper lid may be made, and the latter turned upwards. With hooks or forceps, and scalpel or scissors, the cyst wall must then be carefully separated from all adhesions.

Exostoses occur as the result of inflammation of the bone and of periostitis, and are usually of the kind known as ivory exostoses. They spring most commonly from the ethmoid, or from the frontal bone. Their surface is tuberous. Their growth is extremely slow; in many instances commencing in infancy, and lasting into advanced life.

Operative interference in cases of exostosis of the orbit is only justifiable when the tumour does not grow from the roof of the orbit, as it often involves the cranial cavity, and where there is reason to think it is attached to the orbital wall by a narrow base or pedicle. Several instances are on record in which the growth has become spontaneously separated by necrosis of its pedicle. Beyond destruction of the eyeball, there is no danger associated with these tumours, even if their growth take an intracranial direction; but they cause serious disfigurement, and much pain.

Carcinoma and Sarcoma.—The first of these tumours takes its origin in some neighbouring cavity, and grows into the orbit; or, it may start from the orbital walls; or, from the retro-bulbar connective tissue. Sarcoma may originate in many different positions, most frequently, perhaps, in the periosteum, and in the connective tissue about the lacrimal gland. These malignant tumours, after destruction of the eyeball by pressure, or by phthisis following ulceration of the cornea, attack the bony walls of the orbit and its surroundings. Many forms of sarcoma, however, are non-malignant, especially those which lie free in the orbit and arise from the connective tissue.

The early extirpation of the tumour with complete evisceration of the orbital contents affords, in general, the only prospect of saving the patient's life.

Pulsating Exophthalmos.[1]—This title includes a great variety of vascular tumours, the majority of them having their origin within the cranium, while the remainder are truly orbital. Symptoms common to all these are :—Proptosis ; the presence of peculiar bruits which can be heard over the orbit, and usually also over a more or less extensive portion of the skull ; and pulsation, apparent in the eyeball, or at some point of the orbital aperture. The last symptom may occasionally be absent during the whole, or part, of the progress of the case. The intracranial vascular tumours with which we are most likely to have to deal are :—Aneurism of the ophthalmic artery at its point of origin from the internal carotid ; aneurism of the latter vessel ; and, most commonly, arterio-venous aneurism from communication of the internal carotid with the cavernous sinus—this latter of traumatic origin. In the orbit the following occur :—True aneurism of any of the arterial branches ; diffused or circumscribed traumatic aneurism ; arterio-venous aneurism, of traumatic origin ; aneurism per anastomosis ; and teliangiectic tumours.

Hæmorrhage is liable to prove fatal in these cases.

Treatment.—Ligature of the common carotid affords the best prospect of cure. Digital compression of the same vessel has produced cure in some cases. Spontaneous cure has been observed occasionally, in cases of arterio-venous aneurism.

Diseases of Neighbouring Cavities are sometimes liable to encroach upon the orbital space and to displace the eyeball. The frontal sinus and the antrum of Highmore are the cavities which chiefly come under notice here, but disease of the ethmoidal and sphenoidal sinus does so more rarely.

The condition in these cases is, as a rule, retention of

[1] Exophthalmos without pulsation, and with complete paralysis of the ocular muscles, may be due to true aneurism of the internal carotid in the cavernous sinus, as in a case which I have recently had under my care, and which was completely cured by ligature of the common carotid.—L. W.

normal secretion from obstruction of the outlet of the cavity, or else empyema. The most common initial cause is catarrh of the nasal mucous membrane extending into these cavities, or a blow which fractures the bone in such a way as to occlude the ways of exit. Polypi sometimes grow in these cavities, and distend them.

When a collection of fluid occurs in the frontal sinus, or in the antrum, it is usually present for a long time before the orbital cavity is invaded by bulging of its corresponding wall, and this is accompanied, or preceded, by a swelling in the forehead or cheek, respectively. Finally, the orbital wall is apt to become perforated. Then, in the case of a frontal empyema, an elastic swelling appears at the upper and inner angle of the orbit, near the pulley for the tendon of the superior oblique; while, in the case of the antrum, the tumefaction appears between the eyeball and the inferior orbital margin. The eyeball in each instance is more or less displaced in the opposite direction, and forwards.

As there is no frontal sinus in children under eleven years of age, frontal empyema cannot occur in young children.

Prior to perforation of the orbital wall the only *Treatment* that can be adopted is the formation, by means of a gouge, of an opening into the cavity, either through the orbit or nostril in the case of frontal disease, or through the orbit or cheek in the case of disease of the antrum. After perforation, evacuation of the fluid must of course be made where it points in the orbit. When the fluid has been evacuated, the cavity must be washed out daily with sublimate or astringent lotions, to bring about a healthy state of the lining mucous membrane, and to arrest the discharge. But the complete cure of these cases is often an extremely tedious matter, and may extend over many months.

Empyema of the Ethmoidal Cells presents itself as an orbital abscess, pointing at the upper and inner angle of the orbit, causing displacement of the eyeball forwards, outwards,

and downwards. It is not possible to diagnose with certainty the place of origin of the abscess, unless some of the purulent discharge come through the nostril. After the abscess has been opened lotions syringed into the opening may flow out by the nose.

Empyema of the Sphenoidal Sinus is extremely rare, and is in most instances difficult or impossible of diagnosis; for, if it give rise to exophthalmos, there is nothing to distinguish the latter from protrusion of the eyeball due to many other causes.

Ethmoidal and Sphenoidal Empyema may produce blindness by pressure on the optic nerve, or by setting up orbital cellulitis.

Hernia Cerebri, either in the form of meningocele, or of encephalocele, may invade the orbit. Its most common situation is the upper and inner angle of the orbit, to which it gains access through the suture between the frontal and ethmoid bones. It appears as a fluctuating, often transparent, pulsating congenital tumour. Pressure causes it to disappear, but gives rise, at the same time, to symptoms of cerebral irritation, or pressure.

A congenital tumour in the upper inner angle of the orbit must always be regarded with suspicion, lest it be a cerebral hernia, even though it do not pulsate, or on pressure cause cerebral symptoms. In the large cerebral hernia death in the first few days of life is, we know, the rule.

Exophthalmic Goitre—(Graves' Disease, Basedow's Disease).

Symptoms.—The three cardinal symptoms of this disease are:—Increased rapidity of the heart's action, which may reach two hundred beats per minute; tumefaction of the thyroid gland; and exophthalmos. Of these the cardiac symptom is the most constant, and usually the first to appear; either, or both, of the others may be wanting. There is often also great emaciation, with outbursts of sweating and diarrhœa.

A venous murmur may be heard in the neck. And, in females, there is very commonly irregularity, or suppression of menstruation.

The disease has been observed at all ages, but is most common in early adult life.

Von Graefe's Sign is a very early, tolerably constant, and almost pathognomonic one: it consists in an impairment of the consensual movement of the upper lid in association with the eyeball. When, in the normal condition, the globe is rolled downwards, the upper eyelid falls, and thus its margin is kept throughout in a constant relation to the upper margin of the cornea. In Graves' Disease the descent of the upper lid does not take place, or only in an imperfect manner; and, consequently, when the patient looks down, a zone of sclerotic becomes visible between the margin of the lid and the cornea. This symptom is often present prior to any exophthalmos, and hence its great diagnostic value. It may also continue after the latter disappears,—although it is perhaps more common for it to disappear before the proptosis—and it is not seen, or but very rarely so, in protrusion of the globe from other causes. But the sign is not so absolutely pathognomonic as it was held by von Graefe to be. It may be absent in Graves' Disease, although very rarely so, in the early stages, and it is sometimes present in other diseased states, and even in health.

Stellwag's Sign is also very constant. It is incompleteness, and diminished frequency, of the act of involuntary nictitation.

This act occurs sometimes only once in a minute: or, several rapid nictitations take place, and then a lengthened pause. The nictitation each time is incomplete, the margins of the lid. not being brought together. The result of this may be, that the lower third of the cornea becomes covered with pannus vessels, owing to the constant exposure; for even during sleep the eyelids remain partially open.

Dalrymple's Sign consists in an abnormal widening of the

palpebral aperture, due to retraction of the upper eyelid. It is this gaping of the eyelids which gives the characteristic staring aspect to the patient. This sign is often erroneously attributed to Stellwag, or is included in his sign. The error is due to the fact, that in the same paper[1] in which Stellwag first drew attention to what is above described as his sign, he discussed this other previously observed sign. According to White Cooper[2] it was Dalrymple who first pointed out the latter.[3]

Probably each of these "signs" is due to the one cause suggested by Sharkey,[4] namely, loss of power in the orbicularis, rather than over-action of the levator.

Otto Becker states, that, in a majority of the cases, spontaneous pulsation may be seen in the retinal arteries, but I have only found it sometimes. The vision—unless when corneal complications supervene—and condition of the pupil are unaffected by the disease. In some cases there is an increased flow of tears, but most of the patients complain of a dryness of the eyeballs. The sensibility of the cornea is diminished. Ulcers of the cornea are not common, but are said (von Graefe) to be more frequent in men than in women, although Graves' Disease is more common in women. The exposure of the eye and dryness of the cornea are the chief causes of ulceration, when it occurs; but Sattler inclines to the belief, that it is also largely due to paralysis of the nervous supply of the cornea.

The patients are often hysterical; and even marked psychical disturbances have been noted, such as a peculiar and

[1] *Wiener Med. Jahrbücher*, xvii., p. 25, 1869. See also *Klin. Monatsbl. für Augenheilkunde*, 1869, p. 216, and *Graefe und Sæmisch's Handbuch*, vi., pp. 955 and 956.

[2] *The Lancet*, May 26th, 1849, p. 553.

[3] Other conditions which produce widening of the palpebral aperture, or "Staring Eye," are:—1. Orbital Tumour (mechanically). 2. Stimulation of the Cervical Sympathetic. 3. Cocaine (in slight degree, probably by reason of 2.—Jessop). 4. Women after childbirth (hysteria). 5. In tetanus (spasm of occipito-frontalis). 6. In complete amaurosis.

[4] *Trans. Ophth. Soc.*, vol. xi., p. 204.

unnatural gaiety, rapidity of speech, and great irritability; or, on the other hand, extreme depression, and even attempts at suicide have been observed. Also, loss of memory, and inability to make a mental effort. The motions of the eye-ball have in some cases been defective, a fact for which the exophthalmos does not account. Trousseau's Cerebral Macula is often well marked.

The Progress of the Disease is, as a rule, very chronic, extending over months or years, but liable to fluctuations in the intensity of its symptoms. A few cases have been recorded in which it became fully developed in the course of some hours, or days. After a lengthened period and many fluctuations, the symptoms usually slowly disappear. Occasionally, a slight permanent swelling of the thyroid may remain, and, very often more or less exophthalmos. About 12 per cent. of the cases go from bad to worse, and end fatally from general exhaustion, organic disease of the heart, which may have come on, cerebral apoplexy, hæmorrhage from the bowels, or gangrene of the extremities.

Causes.—Anæmia and chlorosis are general conditions very often present; as are, also, irregularities of menstruation, but it is probable that the latter should be regarded, rather as a concomitant symptom, than as a cause. Severe illnesses are recorded as having gone before the onset in many cases, and also excessive bodily or mental efforts. Great sexual excitement has been known to be followed by Graves' Disease, and depressing psychical causes are not unfrequent forerunners of it. In many instances, however, the patients have been perfectly healthy, and no cause could be assigned.

The Enlargement of the Thyroid is due, in the first instance, to dilatation of its vessels; but, in a late stage, hypertrophy of the gland tissue may be produced, and increase of its connective tissue, and even cystic degeneration. *The Exophthalmos is due* to hyperæmia of the retro-bulbar orbital tissues, as is demonstrated by a vascular bruit often present, and the fact

that steady pressure on the globe diminishes the protrusion. Hypertrophy of the orbital fat may be found *post mortem*, but is, doubtless, secondary to the hyperæmia.

The Theory until of late widely held as to the *Nature of the Disease*, represents it as a lesion of the cervical sympathetic, which causes paralysis of the vaso-motor nerves, and consequent goitre, exophthalmos, and pulsation and dilatation of the carotids and retinal arteries ; while it causes excited cardiac action, by reason of a permanent irritation of the excito-motor nerves of the heart, which also run in the cervical sympathetic. Here the difficulty arises, that two of the chief symptoms are supposed to be explained as the result of paralysis, while the third is said to be due to excitation. The absence, as a rule, of a pupillary symptom is a strong argument against a lesion of the sympathetic. That a state of continuous irritation of the sympathetic should exist is improbable, and is without proved physiological analogy. With regard to paralysis of the sympathetic causing the goitre and exophthalmos, it is doubtful whether it could do so ; for experimental division of the sympathetic has not produced these symptoms in animals, nor have they resulted in clinical cases of paralysis of that nerve in man, although the pupillary symptoms have been marked. *Post-mortem* examination has, no doubt, in a very few instances, revealed alterations in the cervical sympathetic, but they were of an inconstant nature, and were wholly wanting in the vast majority of cases which have been microscopically examined.

These considerations tend to discredit the sympathetic theory.

Professor Sattler, of Leipzig,[1] has advanced a theory which is worthy of consideration. He assumes a lesion of those circumscribed portions of the vaso-motor centre in the brain, which preside over the vaso-motor nerves of the thyroid gland and of the intraorbital tissue ; and believes, that the

[1] *Graefe und Sæmisch's Handbuch*, vol. vi., p. 984, *et seq.*

great constancy with which enlargement of the thyroid and exophthalmos are present indicates an intimate local relation of these two portions. He attributes the cardiac symptoms to a lesion of the cardio-inhibitory centre for the pneumogastric. He also regards Graefe's symptom as due to a central lesion; one, namely, of the co-ordinating centre for the associated motions of the lids and eyeball; while Stellwag's symptom, he believes, as does Stellwag himself, to be due to a lesion of the reflex centres, which are excited by stimuli from the retina and from the sensitive nerves of the cornea and conjunctiva. Sattler's theory derives important support from the experiments of Filehne.[1] When this observer divided the restiform bodies in their upper quarter, although the incision was not carried so deep as to wound the roots of the vagus, yet the functions of the latter nerve become impaired, exophthalmos was produced, and, although the thyroid did not swell, there was vaso-motor paralysis in the ears, thyroid, and anterior part of the neck. Hence, Filehne concludes:—that Graves' Disease may be produced by paralysis of certain nerve-regions controlled by the medulla oblongata, and that the points traversed in common by the nerve-paths concerned are the restiform bodies; that the exophthalmos and goitre depend on dilatation of the blood-vessels; and that the increased heart's action is due to diminution or abolition of tone in the pneumogastric. *Post-mortem* examinations in the human subject are necessary to establish Filehne's theory, but he points out that negative results from some of these would not be fatal to his theory, as the occurrence of functional affections of the central nervous symptom is admitted. Dr. William A. Fitzgerald[2] has pointed out, that exophthalmic goitre is frequently

[1] Zur Pathogenese der Basedow'schen Krankheit, *Sitzungsber. d. Phys. Med. Soc. zu Erlangen*, July 14th, 1879, p. 177. See also *Graefe und Sæmisch's Handbuch*, vol. vi., p. 1001.

[2] Theory of a Central Lesion in Exophthalmic Goitre, *Dublin Journ. Med. Soc.*, March and April 1883.

complicated by symptoms which are clearly due to a central lesion, such as symmetrical paralysis of the external recti, paralysis of the associated motions of the eyes, and glycosuria.

Hale White has recorded [1] a case of Graves' Disease in which, after death, the only lesions were small hæmorrhages in the floor of the fourth ventricle.

A very able explanation of the marked preference shown by the symptoms for the right side of the body is given by Dr. W. A. Fitzgerald (*loc. cit.*). Bilateral symmetry (double exophthalmos, and swelling of each half of the thyroid), although not uncommon, is not always present, especially in the early stages; and, when want of symmetry exists, the preponderance of the symptoms is on the right side—the right eye protruded, and the right lobe of the thyroid enlarged. It has occurred to him, that the extreme constancy of the cardiac symptoms affords a clue to the problem of this preference, for he believes that it, too, is a right-sided symptom; as it is more than probable that it is the right vagus which is chiefly concerned in the inhibition of the heart, and that the left has but little power of the kind. Arloing and Tripier's experiments,[2] and those of Masoin [3] and of Meyer,[4] show this; and several cases are on record, in which irritation of the right pneumogastric in man caused marked cardiac inhibition. Fitzgerald thinks, also, that the mode of development of the heart affords an explanation of the supply of that organ by the right rather than by the left vagus; for, soon after its appearance in the embryo, it projects to the right side, where it comes in relationship with the corresponding vagus.

The theory has recently been advanced, that Graves' disease is due to a toxæmic secretion of the thyroid gland, on

[1] *Brit. Med. Journ.*, March 30th, 1889.

[2] *Archives de Physiologie*, tome v., p. 166, 1873.

[3] *Bull. de l'Acad. Roy. de Med. de Belg.*, tome vi., 3me serie, p. 4.

[4] *Das Hemmungsnervensystem des Herzens*, p. 61, 1869.

the grounds that cures have been obtained by excision of the gland, and that belladonna, which checks its secretion, also exerts a beneficial effect on the course of the disease.

Treatment.—A principal part of this consists in the careful regulation of the patient's general health and functions. Freedom from mental anxiety and excitement, regular hours, moderate exercise, and change of air are the most important items.

The fluctuations which occur in the intensity of the symptoms render it difficult to arrive at definite conclusions with regard to the efficacy of remedies, a vast number of which have been tried and lauded from time to time. In mild forms of the affection, and especially if the anæmia be well marked, iron internally is beneficial, but in severe cases it has the opposite effect. Quinine in moderate doses has been employed with benefit in some cases. Trousseau recommended digitalis in large doses, but its effect must be watched. The beneficial action of iodide of potassium in ordinary goitre has suggested its use in this disease; but, under its influence, the symptoms are sometimes aggravated, and it is doubtful whether they are ever relieved by it. Mr. Hulke[1] speaks highly of aconite, and Dr. Samuel Wilks[2] has no doubt as to the value of belladonna. Ergotin internally has been tried, and with advantage in some instances. Sattler warmly recommends a well-regulated hydropathic treatment, when the patient is not too excitable. Paroxysms of cardiac palpitations, etc., are best combated with ice applied to the head, heart, and goitre. The sympathetic theory has induced the trial of a galvanic treatment of the cervical sympathetic.

Dr. Gauthier[3] recommends antipyrin before everything else. Lemke of Hamburg,[4] in two cases, removed one half of the

[1] *Trans. Ophthal. Soc.*, vol. vi., p. 34.
[2] *Ibid.*, vol. vi., p. 56.
[3] *Rev. de Méd.* 1890, p. 409
[4] *Detusche Med. Wochenschr.*, Jan. 8th, 1891.

enlarged thyroid, with the result of curing the exophthalmos and the excited heart's action, while the other half of the thyroid became spontaneously reduced to normal size.

The great number of remedies which have been proposed for it demonstrate the incurable nature of most cases of this disease.

In cases where the exophthalmos is so great that the cornea is exposed, even during sleep, it is desirable to perform tarsoraphy (p. 210) ; and the same operation is indicated when, the disease having subsided, the exophthalmos still remains to a degree which gives the patient a disagreeable expression.

Enophthalmos or sinking of the eye back into the orbit occurs, to a certain extent, in extreme emaciation, in Asiatic cholera, in paralysis of the sympathetic and in facial hemiatrophy, but it has been observed to an extreme degree as a result of injury. Thirteen cases of traumatic enophthalmus are on record. Beer[1] attributes it to atrophy of the retrobulbar cellular tissue, while Lang[2] explains it by fracture or depression of a portion of the orbital wall.

[1] *Archives of Ophthal.* (Knapp and Schweigger), vol. xxii., No. 1, p. 98. This paper gives abstracts of all the recorded cases.

[2] *Trans. Ophthal. Soc.*, vol. ix., p. 41.

APPENDIX I.

HOLMGREN'S METHOD FOR TESTING THE COLOUR-SENSE.

For the purposes of this method, a selection of Berlin worsteds is made, including red, orange, yellow, yellow-green, pure green, blue-green, blue, violet, purple, pink, brown, grey; several shades of each colour being present, and at least five gradations of each tint, from the deepest to the lightest. Green and grey, several kinds each of pink, blue, and violet, and the pale grey shades of brown, yellow, red, and pink, must be well represented. But no two samples are to be of precisely the same shade of the same colour. This large number of colours and shades is used because the colour-blind person escapes detection with more difficulty, and the diagnosis therefore is all the more certainly made, the greater the variety of colours. The normal-eyed individual readily selects the right ones from the mass; whilst the colour-blind person, although the right ones are directly before him, picks out wrong ones, thereby disclosing the character of his defect.

The test-colour with which Holmgren invariably begins his examination is a pale pure green, because green is the whitest of the spectral colours, and, consequently, the one in which the presence of colour is most difficult to recognise—the one, in short, most easily mistaken for grey (= no colour). Furthermore, as we all experience the most difficulty in deciding whether there be any "colour" at all present in the very deepest shades (nearly black), and in the very palest shades (nearly white), it was plainly either a very dark or a very pale shade of green that should be employed, and Holmgren's experience made him decide for the pale shade, as providing the most delicate test.

As a test for the diagnosis of the particular kind of colour-blindness, Holmgren recommends a purple (deep pink) sample—that is, the whole group of colours in which red (orange) and blue (violet) are combined in nearly equal proportions, or at least in such proportions that no one of them sufficiently preponderates over the others, to the normal sense, so as to give its name to the combination. Purple is of especial importance in the examination of the colour-blind, for the reason that it forms a combination of two fundamental colours (red and blue)—

the two extreme colours—which are never confounded with each other.

The Method of Examination and of Diagnosis is as follows:—The worsteds are placed, in a pile, on a table in broad daylight. The test skein is taken from the pile, and laid at a short distance from it, so as not to be confounded with the other skeins during the trial, and the person examined is then requested to select other skeins most resembling this in colour, and to place them by the side of the sample. It is necessary he should have clearly understood what is required of him; namely, that he should search the pile for the skeins making an impression on his chromatic sense similar to that made by the sample, and independently of any name he may give the colour. Indeed it is not desirable that he should be asked to name the colours, and he should be discouraged from doing so. The examiner should explain that resemblance in every respect is not necessary; that no two specimens are just alike; that the only question is the resemblance of colour; and that, consequently, he must endeavour to find something lighter and darker of the same colour. If the person examined cannot understand this verbal explanation the examiner must resort to action. He must himself make the trial by searching with his two hands for the skeins, thereby showing what is meant by a shade, and afterwards restoring the whole to the pile, except the sample-skein. Or, when a large number of persons have to be examined together, it will be more rapid to begin at once with such a demonstration before the assemblage. There is no loss of security in this, for no one with defective chromatic sense finds the correct skeins in the pile any the more easily from the fact of having a moment before seen some one else looking for and arranging them.

On the card which is attached to the inside of the back cover of this book there are two classes of wool-samples. 1. The Test-Samples, which are placed horizontally. 2. The Colours of Confusion,—that is, those which the colour-blind person selects from the heap of wools, because he confuses them with the colour of the sample,—and these are arranged vertically under their respective test-samples.

The test is conducted as follows:—*Test I.* The green sample is presented. This sample, as already explained, should be of the palest shade of very pure green, which is neither yellow-green nor blue-green to the normal eye, but fairly intermediate between the two. The examination must be continued until the person examined has selected all the other skeins of the same colour, or else, with these or separately, one or several skeins of the class corresponding to the "colours of confusion" (1 to 5), until he has sufficiently proved, by his manner of doing it, that he can easily and unerringly distinguish the confusion colours, or until he has given proof of unmistakable difficulty in accomplishing his task. He who places beside the sample one of the

colours of confusion (1 to 5), that is to say, finds that it resembles the test-sample, is colour-blind. He who, without being quite guilty of this confusion, evinces a manifest disposition to do so, has a feeble chromatic sense.

If we want to know the kind and degree of the colour-blindness which the failure to perform *Test I.* shows to be present, we must proceed to—

Test IIa. A purple skein is shown to the person being examined. The trial must be continued until he has selected all, or the greater part of, the skeins of the same colour, or else, simultaneously or separately, one or several skeins of "confusion" (6 to 9). He who confuses, selects either the light or deep shades of blue and violet, especially the deep shades (6 and 7), or the light or deep shades of one kind of green or grey, inclining to blue (8 and 9). 1. He who is colour-blind by *Test I.*, and who, upon *Test IIa*, selects only purple skeins, is termed "incompletely colour-blind." 2. He who, in *Test IIa*, selects with purple only blue and violet, or one of them, is "completely red-blind." 3. He who, in *Test IIa*, selects with purple only green and grey, or one of them, is "completely green-blind." The red-blind never selects the colours taken by the green-blind, or *vice versâ*. Often the green-blind places a violet or blue skein beside the green, but only the brightest shades of these colours. This does not influence the diagnosis.

The examination may end here, and the diagnosis be regarded as settled. But to convince railway employers, and shipowners, and their employés, a still further trial may be made. It only serves to corroborate the diagnosis.

Test IIb.—The red skein is presented. It is necessary to have a vivid red colour, like the red flag used as signals on railways. This test, which is applied only to those either "completely red-blind" or "completely green-blind," should be continued until the person examined has placed beside the specimen all the skeins belonging to this shade, or the greater part of them, or else, separately, one or several "colours of confusion" (10 to 13). The red-blind then chooses, besides the red, green, and brown shades, which (10 and 11) to the normal sense seem darker than red. On the other hand, the green-blind selects opposite shades, which appear lighter than red (12 and 13). Every case of complete colour-blindness discovered does not always make the precise mistakes just mentioned with *Test IIb*. These exceptions are either persons with comparatively inferior degrees of complete colour-blindness, or of colour-blind persons who have been exercised in the colours of signals, and who try not to be discovered. They usually, but not always, confound at least green and brown. Total colour-blindness is extremely rare, but such a case would be recognised by a confusion of every colour having the same intensity of light.

Violet-blindness will be recognised by a genuine confusion of purple, red, and orange in *Test IIb.*

I have described Holmgren's Method at some length, as it is probable that in the near future (see footnote 1, p. 568) more importance will be attached by the Board of Trade to colour-blindness in sailors and in railways employés, than has hitherto been the case. This method has been specially arranged for the examination of persons in these employments, and has had, for many years, an extensive trial in various countries. The test is equally suited to the examination of acquired and of congenital colour-blindness.

If further information on the subject be desired, the reader should consult Professor Holmgren's original monograph, "De la Cécité des Couleurs," Stockholm, 1877, or Dr. Joy Jeffries' "Colour-Blindness," Boston, 1879.

APPENDIX II.

REGULATIONS AS TO DEFECTS OF VISION WHICH DISQUALIFY CANDIDATES FOR ADMISSION INTO THE CIVIL, NAVAL, AND MILITARY GOVERNMENT SERVICES, THE ROYAL IRISH CONSTABULARY, AND THE MERCANTILE MARINE.

By an army circular issued by the War Office on September 1st, 1887, and which remains in force:—

Candidates for Commissions in the Army are required to possess the following visual powers. These regulations apply to all branches of service, including the Medical Department.

Letters and numbers corresponding to Snellen's Metrical Test-Types (Edition 1885) will be used for testing the standard of vision. If a candidate's vision, measured by Snellen's test-types, be such that he can read the types numbered D = 6 at 6 metres, or 20 English feet, and the types numbered D = 0·6 at any distance selected by himself, with each eye separately, and without glasses, he will be considered fit.

If a candidate cannot read with each eye separately, without glasses Snellen's types marked D = 36 at a distance of 6 metres, or 20 English feet, *i.e.*, if he do not possess one-sixth of Snellen's standard of normal acuteness of vision, although he may be able to read the types D = 0·6 at some distance with each eye, he will be considered unfit.

If a candidate can read with each eye separately, Snellen's types, numbered D = 36, at a distance of 6 metres, or 20 English feet, without glasses, but cannot read them beyond that distance, *i.e.*, if he just possesses one-

sixth of normal acuteness of vision, and his visual deficiency is due to faulty refraction, he may be passed as fit, provided that, with the aid of correcting glasses, he can read Snellen's type D = 6 at 6 metres, or 20 English feet, with one eye, and at least Snellen's types D = 12 at 6 metres, or 20 English feet, with the other eye; and, at the same time, can read Snellen's type marked D = 0·8 with one or both eyes, without the aid of glasses, at any distance the candidate may select.

[*I.e.* As a minimum, a candidate must have with each eye separately $V = \frac{6}{36}$ without glasses; as well as $V = \frac{6}{6}$ with one eye, and $V = \frac{6}{12}$ with the other eye, with glasses.]

Squint, inability to distinguish the principal colours, or any morbid condition, subject to the risk of aggravation or recurrence in either eye, will cause the rejection of a candidate.

The following are taken from Sir Joseph Fayrer's "Regulations as to Defects of Vision, etc."[1]

The Royal Navy.—1. A candidate is disqualified unless both eyes are emmetropic. The candidate's acuteness of vision and range of accommodation must be perfect.

2. A candidate is disqualified by any imperfection of his colour-sense.

The author has it on good authority, that no absolute rule as to vision is laid down with regard to candidates for entry into the Naval Medical Service. Each case is determined at the physical examination at the Naval Medical Department, which takes place shortly before the competition.

Full normal vision is not necessarily essential in all cases for Naval Medical officers.

3. Strabismus, or any defective action of the exterior muscles of the eyeball, disqualifies a candidate for the Royal Navy.

The Home Civil Service.—With reference to the Home Civil Service, the Commissioners refer each case to "a competent medical adviser, leaving him to apply whatever tests he may deem suitable, and whatever standard the particular situation may require."

The Indian Civil Service (*Covenanted and Uncovenanted*).—1. A candidate may be admitted into the Civil Service of the Government of India, if ametropic in one or both eyes, provided that, with correcting lenses, the acuteness of vision be not less than $\frac{6}{9}$ in one eye, and $\frac{6}{6}$ in the other; there being no morbid changes in the fundus of either eye.

2. Cases of myopia, however, with a posterior staphyloma, may be admitted into the service, provided the ametropia in either eye do not exceed 2·5 D., and no active morbid changes of chorioid or retina be present.

[1] Second Edition, J. A. Churchill, 1887

3. A candidate who has a defect of vision arising from nebula of the cornea is disqualified, if the sight of either eye be less than $\frac{6}{12}$; and, in such a case, the acuteness of vision in the better eye must equal $\frac{6}{6}$, with or without glasses.

4. Paralysis of one or more of the exterior muscles of the eyeball disqualifies a candidate for the Indian Civil Service. In the case of a candidate said to have been cured of strabismus by operation, but without restoration of binocular vision, if with correcting glasses the vision reach the above standard (1), and if the movement of each eye be good, the candidate may be passed. The same rule applies to the case of unequal ametropia without binocular vision, both eyes having full acuteness of vision with glasses and good movement.

The Indian Medical Service.—1. A candidate may be admitted into the Indian Medical Service if myopic to the extent of 5 D., provided that with correcting lenses his acuteness of vision in one eye equal $\frac{6}{12}$, and in the other $\frac{6}{6}$, there being no morbid changes in the fundus of the eyes. Cases of myopia, however, with a posterior staphyloma, may be admitted into the service, provided the ametropia in either eye do not exceed 2·5 D., the acuteness of vision with correcting glasses being equal to the above standard, and no active morbid changes of chorioid or retina being present.

2. Myopic astigmatism does not disqualify a candidate for the service, provided the combined spherical or cylindrical glasses required to correct the ametropia do not exceed -5 D.; the acuteness of vision in one eye when so corrected being equal to $\frac{6}{12}$, and in the other eye $\frac{6}{6}$; the accommodation being normal with the correcting glasses, and no progressive morbid changes of the chorioid or retina being present.

3. A candidate having total hypermetropia not exceeding 5 D. is not disqualified for the service, provided the sight in one eye (when under the effect of atropine) equal $\frac{6}{12}$, and in the other $\frac{6}{6}$, with $+5$ D. or any lower power.

4. Hypermetropic astigmatism does not disqualify a candidate for the service, provided the combined lens required to correct the total hypermetropia do not exceed 5 D. The acuteness of vision in one eye must equal $\frac{6}{12}$, and in the other $\frac{6}{6}$, with or without the correcting glass.

5. A candidate may be accepted with a faint nebula of one cornea, reducing the vision to $\frac{6}{12}$, provided the eye in other respects be healthy. In such a case the better eye must be emmetropic, and possess normal vision. Defects of vision arising from pathological or other changes in the eye which are not referred to in the above rules, may exclude a candidate for admission into the Indian Medical Service.

6. A candidate is disqualified if he cannot distinguish the principal colours—red, green, violet or blue, yellow, and their various shades (Dischromatopsia).

7. Paralysis of one or more of the exterior muscles of the eyeball disqualifies a candidate for the Indian Medical Service.

The Indian Marine Service (*Including Engineers and Firemen*).—1. A candidate is disqualified if he have an error of refraction in one or both eyes which is not neutralised by a concave, or by a convex 1 D. lens, or some lower power.

2. A candidate is disqualified if he cannot distinguish the primary colours and their various shades—red, green, violet or blue, and yellow.

3. Strabismus, or any defective action of the exterior muscles of the eyeball, disqualifies a candidate for the Marine Service.

Royal Irish Constabulary.—Candidates for cadetships in the Royal Irish Constabulary, and recruits, must be able to read with each eye separately, and without glasses, Snellen's metrical test types (Edition 1882) number D = 10, at 20 English feet, and those numbered D = 0·8 at any distance selected by the candidate himself.

Squint, inability to distinguish the principal colours, or any morbid condition, liable to the risk of aggravation or recurrence in either eye, will involve the rejection of the candidate.

The British Mercantile Marine.—An Appendix (Exn. 1, Appendix T) to the regulations relating to the Examinations of Masters and Mates in the Mercantile Marine has been issued by the Board of Trade, and came into force on September 1st, 1894.

It is entitled "Form Vision, Colour Vision, and Colour Ignorance Tests," and enacts the following rules :—

1. Examinations for Form Vision, Colour Vision, and Colour Ignorance are open to all persons serving or intending to serve in the Mercantile Marine, and all such persons are recommended to take the earliest opportunity of ascertaining by means of these examinations whether their vision is such as to qualify them for service in that profession.

2. The examination consists of three parts :—

(*a*) Form Vision Test; (*b*) Colour Vision Test; (*c*) Colour Ignorance Test.

No candidate will be examined in the Colour Vision Test until he has passed the Form Vision Test, or in the Colour Ignorance Test until he has passed the Colour Vision Test.

3. Any person serving or intending to serve in the Mercantile Marine, if desirous of undergoing the Form Vision, Colour Vision, and Colour Ignorance Tests *only*, must make application to the Superintendent of a Mercantile Marine Office on the Form EXN. 2A, and must pay a fee of One Shilling.

4. Every candidate for a Certificate of Competency who is not already in possession of such a Certificate will be required to pass the three tests mentioned in Rule 2 before he can proceed to the examination in Navigation and Seamanship for the Certificate which he desires to obtain, even though he may have passed the tests on some previous occasion.

5. Every candidate who is already in possession of a Certificate of Competency, and who desires to obtain a Certificate of a higher grade, will be required to pass the three tests mentioned in Rule (2) before he can proceed to the examination in Navigation and Seamanship for the Certificate of a higher grade.

That is to say, no Candidate will be permitted to proceed with the examination in Navigation and Seamanship for a higher Certificate if he fail to pass the three tests.

6. If a Candidate fail to pass any of the three tests, a note of the fact of his having done so will be written across the face of the Certificate which he already possesses before the Certificate is returned to him.

7. If a Candidate who undergoes the Form Vision, Colour Vision, and Colour Ignorance Tests *only* (See Rule [3]) be in possession of a Certificate of Competency, he must hand in his Certificate before the examination commences, and if he fail to pass any of the three tests, a statement of his failure will be written on the Certificate before it is returned to him.

8. Candidates who fail to pass the Form Vision Test or Colour Ignorance Test can be re-examined at intervals of three months, but candidates who fail to pass the Colour Vision Test cannot be re-examined. It is open, however, to any candidate who has failed to pass that test to appeal to the Board of Trade, who may, if they think fit, remit the case to a special examiner or body of examiners for final decision.

9. The expenses of candidates who are examined by the special examiners, *and are reported by them to have passed the three tests*, will, under certain circumstances, be paid by the Board of Trade, at a rate which will be notified to the candidate, but no payment whatever will be made towards the expenses of candidates who upon their own application are examined by the special examiners and are reported by them to have failed. The special examinations will be held in London only.

10. When a Candidate fails to pass the Colour Test the Examiner will point out to him the conditions under which he can appeal. Appeals are to be made through the Examiner, and forwarded to the Board of Trade with the Examiner's remarks.

11. The holder of a Certificate which bears on it a statement of failure in the first test (Form Vision) or in the third test (Colour

Ignorance) can have the statement removed by passing after the prescribed interval the test to which it refers, but the instruction in the last paragraph of Rule (2) must be followed.

12. The fee paid for examination for a Certificate of Competency includes the fee of One Shilling for examination in Form Vision, Colour Vision, and Colour Ignorance, and if the candidate fails to pass those tests, will, with the exception of One Shilling, be returned to him.

13. Only Examiners who have themselves passed the Colour Test are to undertake these examinations.

Form Vision Test—The tests to be used are Snellen's Letter Test for candidates who can read, and the dot tests for those who cannot read. The sets of tests which have been supplied to the examiners consist respectively of eight sheets of Snellen's letters, and two sheets of dots.

Candidates may use both eyes or either eye when being tested, but they must not be allowed to use spectacles or glasses of any kind. If the candidate can read correctly, at a distance of sixteen feet, three of the five letters in the fifth line from the top, or four of the letters in either of the two lines below, he may be considered to have passed the test. If he cannot do so he should be treated as having failed.

If the candidate cannot read, he must be tested with the sheet of dots. For this test he is to be placed at a distance of precisely eight feet from the test sheets, and exactly opposite them. One of the sheets of dots is then to be exposed, and the candidate should be asked to name the number of dots in one or two of the lines or groups. Lines and groups of dots can be formed by holding a piece of white paper over part of the sheet, but care must be taken that when this is being done the candidate's view is not obstructed, or the light on the test sheet in any way obscured.

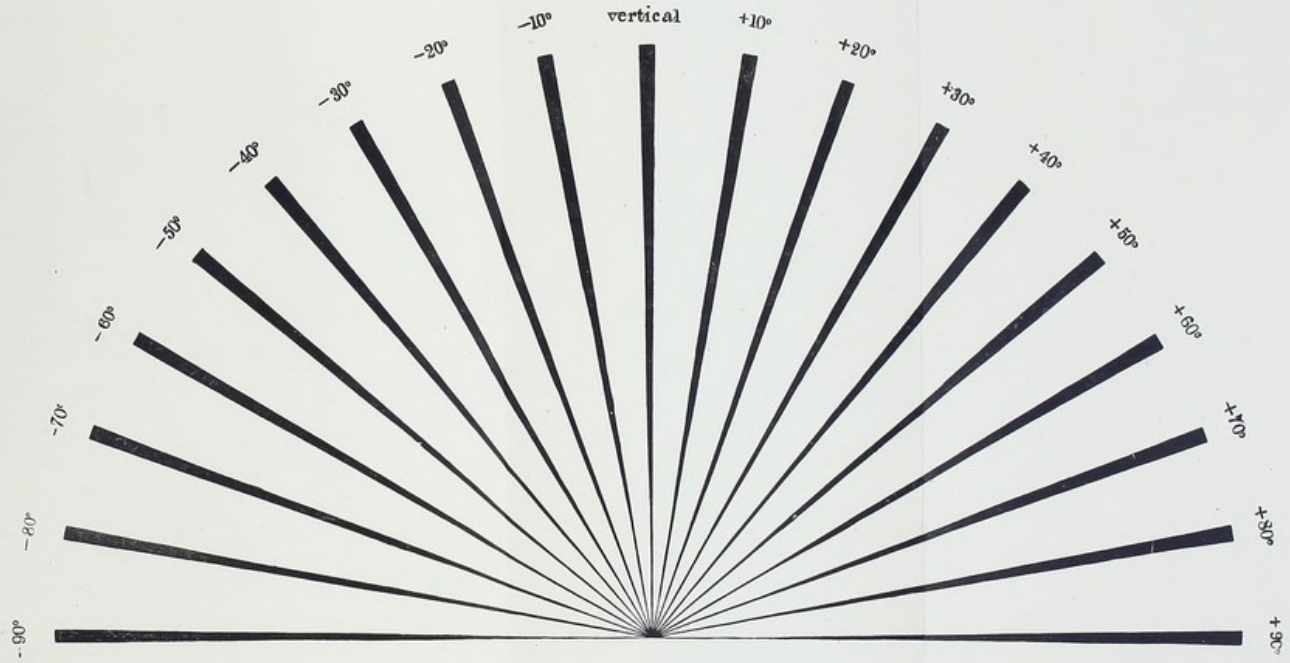
If the candidate answer the questions put to him by the examiner with complete or very nearly complete accuracy, he should be treated as having passed. If he does not answer with very nearly complete accuracy, he should be treated as having failed.

Colour Vision Test.—The Colour Vision of candidates is to be tested by means of Holmgren's wools.

Colour Ignorance Test.—The object of this test is simply to ascertain whether the candidate knows the names of the three colours—red, green, and white—which it is important for every seaman to be acquainted with, and the test is to be confined to naming those colours.

One or two of the purest red and green skeins should be selected from the set of wools, and the candidate should be required to name their colours. He should also be required to name the colour of any white object, such as a piece of white paper.

If he answer correctly he should be considered to have passed the test. If he make any mistake he should be tried with the lantern which was formerly used for colour tests, the plain glass, and the standard red and green glasses being employed for the purpose. If he does not name these glasses correctly he should be reported as having failed to pass the test.


vertical
−10°
+10°
−20°
+20°
−30°
+30°
−40°
+40°
−50°
+50°
−60°
+60°
-70°
+70°
−80°
+80°
-90°
+90°

INDEX.

Abscess of the cornea, 158, 169.
Accommodation, amplitude of, 11; anomalies of, 56; cramp of, 31, 34, 39, 61; and convergence, connection between, 13; mechanism of, 10; normal, 10; paralysis of, 59, 61; relative, 13; relative amplitude of, 13; voluntary relaxation of the, 65.
Accommodative asthenopia, 32, 33, 61, 89, 290.
Acromegaly, 459.
Acuteness of vision, The, 19.
Adenoma of the eyelid, 201.
Adaptation of the retina, 16 footnote, 469.
Advancement, operation of, for strabismus, 532.
Albinismus, 288.
Albuminuria, 406, 436.
Albuminuric retinitis, 405, **406**, 431, 436.
Alcoholism, 311, 440, 498.
Alexia, 464.
Amaurosis, 450; epileptiform, 432; pretended, 472; quinine, 471; spinal, 444; supra-orbital, 467, 547; temporary, after blepharospasm, 139.
Amblyopia, 450; alcoholic, 311, 440; central, 438; congenital, 348, 467, 518; due to central lesions, 450.
Amblyopia ex anopsia, 516; glycosuric, 470; from hæmorrhage (hæmatemesis, etc.), 447; hysterical, 468; from injury to supra-orbital nerve, 467, 547; reflex, 467, 547; in strabismus, 516; tobacco, 310, 438; toxic, 438; uræmic, 469.
Ametropia, 27.
Amnesia, optic, 466.
Amnestic colour blindness, 465.
Amplitude of accommodation, 11; in hypermetropia, 30; in myopia, 36; in presbyopia, 56.
Amplitude of convergence, 14, 538.
Amyloid degeneration of the conjunctiva, 122.
Anæmia, 402, 414; progressive pernicious, 414.
Aneurism of carotid, 311, 550; of central retinal artery, 418; orbital, 550.
Angle, alpha, 15, 30, 37; gamma, 15; gamma in hypermetropia, 30; gamma in myopia, 37; the metre, 14, 538; of strabismus, 522; the visual, 20.
Aniridia, 267, 271.
Anisometropia, 55.
Ankyloblepharon, 233.
Aphakia, 386.
Aphasia, 460, 464.
Apoplexy, cerebral, 310; of pons Varolii, 311; of retina, 413.
Arcus senilis, 189.
Argyll-Robertson's operation for ectropium, 229; pupil, 310.
Argyrosis, 93.
Arlt-Jaesche operation for trichiasis, 217.
Arlt's operation for ectropium, 232.
Arthritis, gonorrhœal, 118, 257.
Asthenopia, 94, 98; accommodative, 32, 33, 61, 89; muscular, 536; neurasthenic, 401; retinal, 401, 517.
Astigmatism, 27, 42; after cataract operations, 387; estimation of degree of, 48; irregular, 55, 182,

375; lental, 54; mixed, 45, 50; ophthalmoscopic diagnosis of, 47, 73, 80; regular, 42; retinoscopy in, 80; spectacles in, 48; symptoms of, 45.
Astigmometer, the, 51.
Ataxia locomotor, 310, 312, 313, 444, 501; hereditary, 542.
Ataxic paraplegia, 542 footnote.
Atheroma, general, 405, 414, 416, 418.
Athetosis, 461.
Atrophy, progressive muscular, 311.
Atropine, 32, 60, 74, 94, 142, 150, 159, 162, 173, 256, **261**, **308**, 335, 336, 345; poisoning, 262, 309; glaucoma from, 263.

BADER'S operation for conical cornea, 183.
Berlin's operation for entropium, 225.
Binocular vision, 526 footnote.
Bisulphide of carbon, amblyopia from, 440.
Black eye, 235.
Blennorrhœa of the conjunctiva, 108, 157; of the lacrimal sac, 242; neonatorum, 108; prophylaxis of, 111.
Blepharitis, Intermarginal, 90; marginal, 90, 195, 237, 244; squamosa, 195; ulcerosa, 195, 214.
Blepharophimosis, 213.
Blepharoptosis, 202.
Blepharospasm, 139, 143, 202.
Blind spot, the, 25.
Blow on the eye, 61, 251, 266, 352, 354, 385, 390, 426, 429, 447.
Bowman's operation for conical cornea, 183.
Brain, localisation of disease in the, 209, 458, 464, 465, 467, 501, 504.
Breast, cancer of the, 287.
Broca's lobe, lesion of, 465.
Bronchitis, 282.
Bulbar paralysis, 60.
Buphthalmos, 338.

CANALICULUS, obstruction of the, 238.
Canthoplasty, 108, 213.
Capsule, lesion of the internal, 461.
Capsulotomy, 365, 383.
Carcinoma of the breast, 287; of the chorioid, 287; of the ciliary body, 277; of the orbit, 549.
Cardiac disease, 400, 405, 414, 416, 417, 418.
Caries of nasal bones, 244.
Cataract, 38, 276, 281, 291, **339**; adherent (or accreta), 351; anterior polar, or pyramidal, 118, 350; artificial ripening of, 346; black, 348; calcareous, 352; capsular, 352; central capsular, 118; central lental, 349; complete, 339; complete of young people, 347; congenital, 279, 348; diabetic, 347; discission for, 380; extraction of, without iridectomy, 376; flap operation for, 376; fusiform, 351; linear operation for, 357; morgagnian, 342; operations for, 355; partial, 349; posterior polar, 351; ripeness of, 341; secondary, 351, 383; senile, 339; spectacles after extraction of, 386; in incipient, 345; suction operation for, 382; symptoms of, 343; three millimetre flap operation for, 363; traumatic, 352; treatment of, 345; von Graefe's operation for, 360; zonular, or lamellar, 349.
Catarrh, conjunctival, 90; spring, 94.
Cautery, the actual, 142, 152, 160, 161, 169, 183, 374.
Cavernous sinus, thrombosis of the, 313, 507.
Cerebellum, tumour of the, 432 footnote, 508.
Cerebral abscess, 432; cysts, 433; disease, 203, 209, 432, 481, 501, 504; embolism, 310; hæmorrhage, 460, 462; localisation, 209, 458, 504; softening, 462; tumour, 432, 463, 507.
Chalazion, 198.
Chemosis, 89, 94, **110**, 198.
Chiasma, lesion of the, 456, 457, **458**.

Chloroform narcosis, the pupil in, 309.
Chlorosis, 197, 312, 402, 435.
Choked disc, 431.
Cholera, 400.
Chorea, 416.
Chorioid, central senile atrophy of the, 280, 356; colloid degeneration of the, 280; coloboma of the, 288; congenital defects of the, 288; detachment of the, 283; diseases of the, 254; hæmorrhage in the, 285; injuries of the, 284; tubercle of the, 269, 287, 423; tumours of the, 285, 426.
Chorioidal ring, 84.
Chorioiditis, 278; central, 280; central senile guttate, 280; disseminated, 254, 278, 351, 404; purulent, 281.
Chorioido-retinitis, 280, 403.
Chromidrosis palpebrarum, 201.
Ciliary body, coloboma of the, 271, 288; diseases of the, 254; inflammation of the, 275; injuries of the, 277; new growths of the, 277.
Ciliary muscle, action of the, 10; cramp of the, 31, 34, 39, 61.
Climacteric, the, 414.
Cocaine, 135, 147, 159, 166, 185, **309**, 331 footnote, 360.
Coloboma, congenital, of the chorioid, 288; of the ciliary body, 271, 288; of the crystalline lens, 271; of the eyelids, 236; of the iris, 271, 273.
Colour-blindness, 17, 439, 445, 468, 561; amnestic, 465.
Colour-sense, the, 16, 17, 561; in periphery of field, 26; method of testing the, 17, 561; theories of the, 17.
Commotio retinæ, 429.
Congestion papilla, 408, 431.
Conjugate lateral paralysis, 500, 501.
Conjunctiva, amyloid degeneration of the, 122; cicatrices of the, 99; cysticercus under the, 133; cyst of the, 132; dermoid tumour of the, 130; diseases of the, 89; epithelioma of the, 132; essential shrinking of the, 126.
Conjunctiva, hæmorrhages in the, 90, 122, **130**; hyaline degeneration of the, 122; hyperæmia of the, 89; injuries of the, 134; lipoma of the, 131; lithiasis of the, 133; lupus of the, 124; lymphoma of the, 108; nævus of the, 130; papilloma of the, 131; pemphigus of the, 124, 125; pinguecula of the, 129, 130; polypus of the, 130; sarcoma of the, 132; syphilitic disease of the, 131; transplantation of the, 212; tubercular disease of the, 122, 193; uric acid in the, 134; xerosis of the, 125, 469.
Conjunctival complication of small-pox, 121.
Conjunctivitis, 90; catarrhal, 90, 243, 248; croupous, 118, 126; diphtheritic, 119, 126; follicular, 93; gonorrhœal, 108; granular, 95, 210, 214; phlyctenular, 136, 137; purulent, 98, 103, **108**, 214, 146, 157.
Contact glasses, 182.
Corectopia, 270.
Cornea, abscess of the, 146, 158, **169**, 186; absorption ulcer of the, 162; arcus senilis in the, 189; bulla of the, 167; calcareous film of the, 175; cauterisation of the, 142, 152; cicatrices in the, 144, 148; conical, 181; deep ulcer of the, 116, 148, **156**; dermoid tumour of the, 130, 184; diagnosis of ulcer of the, 147; diseases of the, 145; ectasies of the, 176; epithelioma of the, 184; facetted ulcer of the, 162; fibroma of the, 184; foreign bodies in the, 158, 162, 184; globosa, 338; herpes of the, 164; infantile ulceration of the, with xerophthalmia, 163; inflammations of the, 145; injuries of the, 158, 170, **184**, 336; leucoma of the, 150, 186, 187; macula of the, 150, 186; nebula of the, 150, 186; non-ulcerative inflammations of the 169; opacities of

the, 83, 150, 186; papilloma of the, 184; paracentesis of the, 142, **153**, 157, 161, 183; pigmentation of the, 190; ring ulcer of the, 111, 138, 142, 161; rodent ulcer of the, 160; sarcoma of the, 184; sclerotising opacity of the, 175, 189, 250; simple ulcer of the, 156; staphyloma of the, 102, 116, 121, 209, 158, **176**; tattooing of the, 187; transplantation of the, 188; tumours of the, 184; ulcerative inflammations of the, 145, 176; ulcus serpens of the, 157, 169.
Corneal complications in catarrhal conjunctivitis, 91; in purulent conjunctivitis, 110, 115, 157; in smallpox, 121.
Corrosive sublimate, subjunctival injections of, **160**, 250, 292.
Cramp of accommodation, 31, 34, 39, 61; of orbicularis, 139, **201**.
Critchett's operation in sympathetic ophthalmitis, 298.
Croupous conjunctivitis, 118, 125.
Crus cerebri, lesion of the, 505, 506.
Cupping, pathological, of the optic disc, 85, **317**; physiological, of the optic disc, 84.
Cyclitis, 171, 192, 218, **275**; plastic, 275; purulent, 276; serous, 171, 275.
Cyst, meibomian, 198; of the conjunctiva, 132; of the iris, 269.
Cysticercus in the vitreous humour, 397; under the conjunctiva, 133; under the retina, 423.
Cystoid cicatrix, 329, 375.

DACRYOADENITIS, 246.
Dacryocystitis, acute, 107, 244; chronic, 158, 242.
Daturine, 309.
Decentration of spectacle glasses, 539.
Defects of vision disqualifying candidates, Appendix II., p. 564.
Dermoid tumours of conjunctiva and cornea, 130, 184.
De Wecker's operation for staphyloma cornea, 177.
Diabetes, 38, 60, 130, 261, 264, 347, 355, 409, 440, 470, 499.
Dianoux operation for trichiasis, 218.
Dilator pupillæ, 306.
Dioptric unit, the, 6; system of the eye, 8.
Dioptry, the, 6.
Diphtherial paralysis of accommodation, 60; of orbital muscles, 499.
Diphtheritic conjunctivitis, 118, 125, 126.
Diplopia in convergent concomitant strabismus, 514, 525; crossed, 485; homonymous, 483; in insufficiency of the internal recti, 537; in paralysis of orbital muscles, 483; monocular, 266, 386.
Discission, 348, 349, 380.
Distichiasis, 214; operations for, 215.
Duboisine, 309.
Dyslexia, 465.

ECCHYMOSIS of the conjunctiva, 91, 130.
Eclipses, blinding of the retina in, 419.
Ectropium, 228; cicatricial, 228, 232, 546; muscular, 228; senile, 228; spastic, 228.
Egyptian ophthalmia, 95.
Electric light, effects of, on the eyes, 421.
Electrolysis for detached retina, 428; for nævi, 200; for stricture of canaliculus, 239; for trichiasis, 215.
Embolism, cerebral, 310; of retinal vessels, 400, 415, 444.
Emmetropia, 9; amplitude of accommodation in, 12.
Endarteritis, 419.
Endo-carditis, 282.
Enophthalmos, 560.
Entropium, 125, 222; organic, 222; operations for, 222; senile, 222, 225; spastic, 222, 225.
Enucleation of the eyeball, 293, **298**.
Epicanthus, 236.
Epilation of eyelashes, 215.

Epilepsy, 46, 233, 310, 536.
Epiphora, 237, 243.
Episcleritis, 248.
Epithelioma of the conjunctiva, 132; of the cornea, 184; of the eyelid, 201.
Ergotism, 347 footnote.
Erysipelas of the eyelids, 191, 245, 418, 434.
Erythropsia, 388, 474.
Eserine, 94, 115, 151, 154, 159, 161, 182, 184, 309, 363, 365.
Evisceration of the eyeball, 178, **296**.
Exophthalmic goitre, 209, **552**,
Exophthalmos, pulsating, 550.
Expression of granulations, 105.
Eyeballs, the motions of the, and their derangements, 475.
Eyelids, adenoma of the, 201; chromidrosis of the, 201; coloboma of the, 236; cramp of the, 139, 201; diseases of the, 191; ecchymosis of the, 235; eczema of the, 191, 195; emphysema of the, 235; epithelioma of the, 201; erysipelas of the, 191, 245, 418, 434; eversion of the, 228; herpes zoster of the, 192; injuries of the, 235; inversion of the, 222; lupus of the, 201; millium of the, 199; molluscum of the, 200; nævus of the, 200; restoration of an, 233; rodent ulcer of the, 194; sarcoma of the, 201; syphilitic sores on the, 193; vaccine vesicles on the, 193.

FACET on the cornea, 144, 162.
Facial centre, lesion of the, 209.
Far point and near point, 11.
Field of vision, 22; binocular, 25.
Fixation, field of, 480; line of, 15.
Fluorescin, **147**, 160.
Focal illumination, 83; internal, 43.
Focal length of a lens, 3.
Focus, conjugate, 4; principal, 3; virtual, 4.
Follicular conjunctivitis, 93.
Fomentations, warm, 151.
Form-sense, the, 16, **19**, 451, 460.
Fornix conjunctivæ, 89.
Fourth ventricle, diseases of floor of, 497.
Fovea centralis, 86, 88.
Fracture of base of the skull, 130, 235.
Fundus oculi, the normal, 83, 85, 86.

GAILLARD'S sutures for entropium, 226.
Galvano-cautery, **153**, 160, 161, 169, 183, 374.
Glaucoma, 122, 168, 171, 263, 271, 313, **314**, 466; acute, 321; chronic, 316; etiology of, 324; fulminans, 324; hæmorrhagic, 337, 415; pathology of, 325.
Glaucoma, primary, 314; secondary, 250, 260, 268, 276, 286, 297, 336, 353, 380, 382, 413; sub-acute, 324; treatment of, 328.
Glaucomatous cup, 317.
Glaucomatous degeneration, 324; ring, 319.
Glioma of the brain, 423, 432; of the optic nerve, 446; of the retina, 287, 389, **422**.
Goitre exophthalmic, 209, 501, **552**.
Gonorrhœal arthritis, 118, 257; conjunctivitis, 108; iritis, 257.
Gout, 414.
Granular conjunctivitis, 95, 122, 126, 162.
Granuloma of the iris, 270.
Green's operation for entropium, 224.

HÆMATEMESIS, 414, 447.
Hæmophthalmos, 251.
Hæmorrhage into the anterior chamber, 251, 265.
Hæmorrhoids, 406.
Hallucinations, visual, 465.
Hay fever, 95.
Headache, 46.
Hemiachromatopsia, 455, 458, 460.
Hemianæsthesia, 461, 462.
Hemianopsia, altitudinal, 452, 455, 458; complete and partial, 451; homonymous, 303, 451, 459; localisation of lesion in, 458; nasal, 452, 458; relative and absolute, 451, 455; superior and

inferior, 452, 455, 458; temporal, 451, 458; transitory, 463.
Hemiopic pupil, the, 462.
Hemiplegia, 459, 506, 509; crossed, 209, 505, 506.
Hering's drop experiment, 526 footnote; theory of the colour-sense, 17.
Hernia cerebri, 552.
Herpes corneæ, 164; zoster ophthalmicus, 164, 192.
Heterophthalmos, 270.
Hippus, 304, 313.
Holmgren's tests for colour-blindness, 17, and Appendix I.
Hooping cough, 130, 166.
Hordeolum, 198.
Hotz's operation for entropium, 227.
Hydrocephalus, 434, 442.
Hydrophthalmos, 338.
Hyoscyamine, 309.
Hypermetropia, 27; amplitude of accommodation in, 30; angle γ in, 30; asthenopia in, 32; axial, 27; curvature, 27; cramp of ciliary muscle in, 31; determination of degree of, 28, 71, 78; direct ophthalmoscopic method in, 71; internal strabismus in, 33, 511; latent, 31, 33; manifest, 31, 33; prescribing of spectacles in, 33; retinoscopy in, 76, 80.
Hypermetropic astigmatism, 44.
Hyphæma, 251, 265.
Hypopyon, 138, **147**, 157, 158, 163, 275, 276, 394.
Hysteria, 310, 401, 402, 468.

Illaquæatio, 215.
Illumination, focal, 83.
Image, erect ophthalmoscopic, 64; inverted ophthalmoscopic, 66.
Image formed by a lens, 5; by mirrors, 7; real, 6; virtual, 6.
Influenza, epidemic, 60, 438, 499, 544.
Intermittent fever, 165, 166.
Internal capsule, lesion of the, 209, 461; recti, insufficiency of the, 39, 536.
Intestinal worms, 312.
Intraocular growths, 209; injections, 293; tension, 115, 258, 259, 264, 271, 275, 281, 286, **315**, 354, 382.
Iridectomy, 271, 328; for glaucoma, 271, 273, **328**, 337, 414; in cataract operations, 365, 376; optical, 274.
Irideremia, 271.
Irido-chorioiditis, 260, 267, 289.
Irido-cyclitis, 171, 174, 254, 260, 289.
Iridodialysis, 266.
Iridodonesis, 386.
Iridoplegia, 313.
Iridotomy, 275, 385.
Iris, absence of the, 267, **271**; anteversion of the, 267; coloboma of the, 271, 288; cysts of the, 267; diseases of the, 254; foreign bodies in the, 265; granuloma of the, 268; injuries of the, 265; malformations of the, 270; new growths in the, 267; operations on the, 271; persistent pupillary membrane of the, 271; posterior limiting membrane of the, 306; prolapse of the, 116, 154, 155, 163.
Iris, retroflexion of the, 266; rupture of the sphincter of the, 266; sarcoma of the, 270; trembling of the, 386; tubercle of the, 268
Iritis, 91, 102, 104, 122, 163, **255**, 375; diabetic, 261; gonorrhœal, 257; hæmorrhagic, 259; parenchymatous, 255, 258; plastic, 255, 257; purulent, 255, 264; quiet, 256; rheumatic, 257, 264; serous, 255, 257, 337; syphilitic, 257, 258, 265; tubercular, 268; treatment of, 261.

Jacob's ulcer, 194.
Jequirity, 106.

Keratitis, 122, 136, 138, 146: bullosa, 167; dendriform, 168; diffuse interstitial, 170; fascicular, 138, 142; filamentous, 166; neuroparalytic, 162; phlyctenular, 136, 138, 162; punctata, 174, 257, 276, 290; riband-like, 175: striped, 372.
Keratoconus, 181.

LACRIMAL apparatus, diseases of the, 237; canaliculus, obstruction of the, 238; duct, stricture of the, 240; fistula, 245; gland, extirpation of the, 247; hypertrophy of the, 247; inflammation of the, 246; obstruction, 89, 238, 240.
Lacrimal punctum, eversion of the, 91, 191, 228, 337; inversion of the, 237; malposition of the 237; occlusion of the, 237; stenosis, of the, 237.
Lacrimal sac, acute inflammation of the, 244; blennorrhœa of the, 242; mucocele of the, 243; obliteration of the, 246.
Lagophthalmos, 209.
Lamellar cataract, 349.
Lamina cribrosa, 84.
Lead poisoning, 436, 499.
Lens, action of a concave, on rays, 2, 5; of a convex on rays, 2, 3; focal length of a, 3; optical centre of a, 3; principal axis of a, 3; principal focus of a, 3; the image formed by a, 5.
Lens, crystalline, absence of the, 386; calcification of the, 260; change of in accommodation, 10; change of in presbyopia, 56; coloboma of the, 271; diseases of the, 339; dislocation of the, 252, 290, 336, **385**; injuries to the, 337, **352**; removal of, in myopia, 42.
Lenses, cylindrical, 48; numbering of the trial, 6; spherical, 2; hyperbolic, 182.
Lental astigmatism, 54.
Lenticonus, 386.
Leucocythemia, 409.
Leucoma, 150, **186**; adherent, 116, 157, 158, **186**.
Lice on the eyelashes, 197.
Light difference (L.D.), 16; minimum, 16; projection of, 356.
Light-sense, the, **16**, 320, 404, 412, 443, 451, 460, 471.
Limbus conjunctivæ, 89.
Linear cataract extraction, 357.
Lipoma of the conjunctiva, 131.
Lithiasis of the conjunctiva, 133.
Localisation, cerebral, 209, 458, 501.
Locomotor ataxy, 310, 312, 313, 444, 501.
Lupus of the conjunctiva, 124; of the eyelid, 201.

MACULA CORNEÆ, 150, 186.
Macula lutea, disease of the, 39; nervous supply of, 456; normal appearances of the, 85; ophthalmoscopic examination of the, 69.
Madarosis, 193.
Maddox rod test, 538.
Malaria, 400, 438.
Mania, acute, 311, 312.
Massage, 95, 187, 189, 249, 417.
Measles, 91, 170, 282, 438.
Media, opacities in the, 82; the intraocular, 8; the refracting, 8.
Meibomian cyst, 198.
Melancholia, 312.
Meningitis, 299, 310, 433, 458, 462, 493, 544; cerebro-spinal, 282; following enucleation, 299; tubercular, 310, 433.
Menstruation, 250, 406, 434.
Mental derangement, sign of, 60, 312; after cataract operation, 379.
Metamorphopsia, 404, 420.
Metre angle, 14, 538.
Metria, 261, 282, 411, 544.
Metrical system of numbering lenses, 6.
Meynert's fibres, 303, 462.
Micropsia, 59, 404.
Migraine, 463; ophthalmoplegic, 496.
Military ophthalmia, 95.
Milium, 199.
Mind-blindness, 466.
Mirror, concave, 7; in retinoscopy, 74.
Mirror, plane, 7; in retinoscopy, 81.
Molluscum, 200.
Morphia, action of, on the pupil, 309.
Motions of the eyeballs, 475.
Mucocele, 243.
Mules' operation, **179**, 293, 296.
Muscarine, 309.

Muscæ volitantes, 392.
Mydriasis, 59, 311, 544; traumatic, 267.
Mydriatics, action of the, 308.
Myelitis, 311, 433.
Myopia, 37, **33**, 426; amplitude of accommodation in, 36; angle γ in, 37; apparent, 61; axial, 33; cause of, 37; complications of, 38, 426; cramp of accommodation in, 39; curvature, 34; detachment of retina in, 39, 426; determination of the degree of, 36, 71, 77; direct ophthalmoscopic method in, 71; insufficiency of the internal recti in, 39, 514, 535; macular disease in, 39; management of, 40; opacities in vitreous humour in, 39; posterior staphyloma in, 38; prescribing of spectacles in, 40, 41; progressive, 38, 39; removal of lens in, 42; retinal hæmorrhage in, 39; retinoscopy in, 77.
Myopic astigmatism, 44.
Myosarcoma of ciliary body, 277.
Myosis, 309; spinal, 310.
Myotics, action of the, 309; use of, in glaucoma, 334.

Nævus of the conjunctiva, 130; of the eyelids, 200.
Nasal catarrh, 165, 240, 243.
Nasal duct, stricture of the, 240, 243.
Near point, 11.
Nebula of cornea, 150.
Nephritis, 409, 469.
Nerve fibres, opaque, 85.
Neurasthenia, 313.
Neurasthenic asthenopia, 401.
Neurectomy, optic, 296, 300.
Neuro-retinitis, 403.
Neurotomy, optic, 295.
Nicotine, 309.
Night-blindness, 127, 163, 404, 412, **468**.
Nuclear paralysis, 497.
Nyctalopia (*see* Night-blindness).
Nystagmus, 288, 348, **541**.

Objects, distant and near, 9.
Occipital lobe, lesion of the, 452, 459.
Omphalo-phlebitis, 282.
Opaque nerve fibres, 85.
Ophthalmia, Egyptian, 95; gonorrhœal, 108; granular, **95**, 210, 214; military, 95; phlyctenular, 136; purulent, **108**, 214; tarsi, 195.
Ophthalmia, nodosa, 270.
Ophthalmoplegia externa, 497; interna, 59, 497.
Ophthalmoscope, the, 62; direct method of examination by, 64; estimation of refraction with the, 69; indirect method of examination by the, 66.
Optic axis, the, 15.
Optic ganglia, lesion of the Primary, 461.
Optic nerve, the, 68, **83**; atrophy of the, 191, 254, 319, 331, 434, **443**, 446, 447, 462, 544, 546, 547; colloid outgrowths of the, 447; diseases of the, 431; injuries of the, 447; resection of the, 300; tumours of the, 446.
Optic papilla, 68, 83; cupping of the, 84, 317; radiations, lesion of the, 461; neuritis, 171, 254, 291, **431**, 443, 458, 462, 544, 547; retrobulbar, 435, 438; tract, lesion of an, 461.
Optic neuritis, with dropping of watery fluid from nostril, 442.
Optical axis, the, 8; centre, the, 8.
Orbicular sign, the, 509.
Orbit, aneurism of the, 550; carcinoma and sarcoma of the, 549; caries of the, 546; cysts of the, 548; diseases of neighbouring cavities, 550; diseases of the, 544; exostosis of the, 549; foreign bodies in the, 547; fracture of the 235, 547; inflammation in the, 434; injuries of the, 546, 547; penetrating wounds of the, 547; periostitis of the, 434, 545; syphilitic gumma of the, 545; tumours of the, 209, 434, **547**.
Orbital cellulitis, 544; muscles, paralysis of the, 481.

Osteo-sarcoma of the chorioid, 287.
Ozæna, 243.

PAGENSTECHER'S ointment, 141.
Pannus, 101, 162.
Panophthalmitis, 121, 163, 252, **281**, 289, 389, 411.
Papillitis, 431.
Papilloma of conjunctiva, 131.
Paracentesis of the cornea, 116, 142, **153**, 157, 161, 183, 416.
Parallax, 84, 318.
Parallel rays, 9.
Paralysis of accommodation, 59, 497; bulbar, 60, 311, 499; of the cervical sympathetic, 311; conjugate lateral, 500, 591; diphtherial, 60, 499; of the facial nerve, 209, 509; fascicular, 481, 501; of the fifth nerve, 162, 509; of the fourth nerve, 487, 508; general, of the insane, 311, 312, 444, 501; intermittent, of the third nerve, 496; of the levator palpebrae (*see* Ptosis); nuclear, 497; of the orbital muscles, 481; of the sixth nerve, 59, 485, 508; of the sphincter iridis (*see* Mydriasis); of the third nerve, 59, 489, 504, 507.
Pemphigus of the conjunctiva, 124, 125, 209.
Perimeter, the, 22.
Peripheral neuritis, 437, 498, 501, 542 footnote.
Peritomy, 108.
Perivasculitis, 419.
Phlyctenular conjunctivitis, 136; keratitis, 138, 163, 165.
Phosphene, 425.
Photometer, 16,
Photophobia, 95, 102, 110, **138**.
Phtheiriasis ciliorum, 197.
Phthisis anterior, 291; bulbi, 252, 260, 276.
Physostigmine, 309.
Pilocarpine, 151, 309.
Pineal gland, tumour of the, 508.
Pinguecula, 129.
Pituitary body, 459.
Pneumonia, 163, 165, 261.
Polycoria, 270.
Polyopia, monocular, 343.
Polypus of the conjunctiva, 130.
Pons, lesions of the, 209, 311, 481, 500, 502, 509.
Posterior staphyloma, 38, 283.
Pregnancy, 408, 416, 469.
Presbyopia, 56.
Primary optic ganglia, lesion of the, 461.
Prism, action of a, 2; in diplopia, 495.
Progressive muscular atrophy, 542 footnote.
Proptosis, 545, 547.
Pseudo-glioma, 281, 389, 423.
Pterygium, 127.
Ptosis, **202**, 236, 489, 498, 504; with associated movements, 207.
Pulmonary obstruction, 400.
Pulsation in retinal vessels, 88, 319, 324, 554.
Pulvinar, lesion in the, 459, 461.
Punctum lacrimale, eversion of the, 91, 191, 228, 237; inversion of the, 237; occlusion of the, 237.
Punctum proximum, 11, 56; remotum, 11, 35, 56.
Pupil, action of mydriatics on the, 308; action of myotics on the, 309; the Argyll-Robertson, 310; artificial, 271; change of, in accommodation, 11, 305; contraction of the, 302; in chloroform narcosis, 309; dilatation of the, 306; exclusion of the, 256; hemiopic, 462; hippus of the, 304; influence of the fifth nerve on the, 308; of the optic nerve on the, 302; of the sympathetic nerve on the, 306, 307; of the third nerve on the, 302, 303; malposition of the, 270; occlusion of the, 256; reflex contraction of the, 302; sign of the, in disease, 309; in health, 302; supernumerary, 270; unrest of the, 308.
Pupillary, membrane persistent, 271.
Purulent chorioiditis, 281; conjunctivitis, 98, 103, **108**, 146, 157, 214; inflammation of vitreous humour, 389; iritis, 255, **264**; retinitis, 411.

Pyæmia, 122, 282, 545.

Quinine amaurosis, 400, 471.

Recurrent fever, 261.
Red vision (*see* Erythropsia).
Reflection, phenomenon of, 7.
Refraction and accommodation, abnormal 27; normal, 8.
Refraction, estimation of, by retinoscopy, 74; by the upright image, 69; the phenomenon of, 1.
Resection of the optic nerve, 296, 300.
Retina, adaptation of the, 16 footnote, 469; alterations in vascularity of the, 400; aneurism of the central artery of the, 418; apoplexy of the, 413; atrophy of the, 412; blinding of the, by direct sunlight, 419; cysticercus under the, 423; detachment of the, 39, 260, 276, 285, 351, 392, 415, 408, **424**; development of connective tissue in the, 410; diseases of the, 400; electric light, action of, on the, 421; embolism of the central artery of the, 415; functional affections of the, 400; glioma of the, 422; hæmorrhage in the, 39, 122, 337, 405, 413, 409, 410; hyperæsthesia of the, 400, 402, 467, 468; inflammation of the, 403; normal, 85; septic affections of the, 122, 411; thrombosis of the central artery of the, 417; thrombosis of the central vein of the, 400, 405, **418**; traumatic œdema of the, 429; traumatic anæsthesia of the, 429; tumours of the, 422; parasitic disease of the, 423.
Retinal affections in diabetes, 409; anæsthesia, 401; asthenopia, 401, 517; ischæmia, 471.
Retinal vessels, the, 86, 400; diseases of the, 413; pulsation of the, 88, 319, 320, 324, 554; sclerosis of the, 419.
Retinitis, 403; albuminuric, 405, **406**, 431, 436; circinata, 411; hæmorrhagic, 405, 411; leucæmic, 409; pigmentosa, 412, 444, 468; proliferans, 410; punctata albescens, 410; purulent, 122, 411; syphilitic, 403.
Retinoscopy, 74.
Retrobulbar neuritis, 435, **438**.
Rheumatism, 61, 203, 210, 175, 249, 250, 252, 256, 257, 261, 264, 434, 436, 493, 545, 546.
Rodent ulcer of the cornea, 160; of the eyelid, 194, 195 footnote.

Sæmisch's Ulcer, 157.
Sarcoma of the chorioid, 285; of the ciliary body, 253, 277, 290; of the conjunctiva, 132; of the cornea, 184; of the eyelid, 201; of the iris, 270; of the sclerotic, 253.
Scarlatina, 91, 170, 438, 469, 544.
Scintillating scotoma, 463.
Scleritis, 175, 189, 248, 250.
Sclero-chorioiditis posterior, 283.
Sclerosis, multiple, 313, 437, 440, 444, 501, 542.
Sclerotic, diseases of the, 248; pigment spots on, 253; injuries of the, 251, 426; ring, 84; tumours of the, 253.
Sclerotising opacity of the cornea, 175, 250.
Sclerotomy, 332.
Scopolamine, 262, 263 footnote.
Scorbutus, 469.
Scotoma, central, 18, 279, 439, 445; positive, 390, 404, 401; relative, 410, 439; scintillating, 463.
Septicæmia, 261, 411.
Shadow-test, the, 74.
Short sight, 33.
Shot silk retina, 85.
Siderosis, 190.
Sight, the sense of, 16.
Silver nitrate, action of, 112.
Skull, fracture of the, 130, 235, 444, 447, 459.
Small-pox, conjunctival and corneal complications of, 121, 170.
Small-pox, ocular sequelæ of, 91, 121, 170, 261
Snellen's operation for entropium, 222; sutures, 229.
Snow blindness, 421.
Spectacles in accommodative as-

thenopia, 33; in albinismus, 289; in anisometropia, 55; in aphakia, 386; in astigmatism, 49; in conical cornea, 182; in convergent strabismus, 524, 535; in cramp of accommodation, 32; in hypermetropia, 33; in incipient cataract, 345; in insufficiency of the internal recti, 539; in irideremia, 271; in myopia, 41; in nebulous cornea, 187; in paralysis of accommodation, 61; in paralysis of orbital muscles, 495; in presbyopia, 58.
Sphenoidal fissure, lesion at the, 481; periostitis at the, 60.
Sphenoid, fracture of the, 459.
Spinal amaurosis, 444; cord, diseases of the, 310, 311, 312, 313, 437, 440, 444; myosis, 310.
Spring catarrh, 94.
Squint (*see* Strabismus).
Staphyloma, anterior, 209, 250, 323; treatment of, 177; of the cornea, 102, 115, 125, 158, **176**; posterior, 38, 283.
Stenopæic spectacles, 182 187, 289, 350.
Stomach, hæmorrhage from the, 447.
Strabismus, apparent, 15, 481; apparent convergent, 37; apparent divergent, 30.
Strabismus, 480; convergent concomitant, 33, 510; advancement of internal rectus in, 532; amblyopia in, 516; angle of, 522; clinical varieties of, 517; dangers of operation for, 534; hypermetropia in, 33, 511, 524, 535; measurement of, 518; mobility of eye in, 524; operation for, 527, 529; orthoptic treatment of, 525; single vision in, 514; tenotomy in, 529.
Strabismus, divergent concomitant, 536, 540; tenotomy in, 540; treatment of, 539.
Streatfield's operation for entropium, 222.
Strumous ophthalmia, 136.
Stye, 198.
Symblepharon, 120, 135, **210**.
Sympathetic irritation, **290**, 468; ophthalmitis, 174, 175, 178, 252, 267, 268, 277, 282, **289**, 394.
Synchesis, 390, 392; scintillans, 393.
Synechia, anterior, **116**, 336; posterior, **255**, 260, 336.
Syphilis, 60, 61, 131, 172, 173, 189, 193, 203, 210, 240, 249, 250, 256, 257, 258, 261, 264, 279, 280, 390, 403, 406, 419, 435, 444, 458, 462, 493, 510, 545, 546; of the conjunctiva, 131; of the eyelids, 193; inherited, 172, 261, 279, 282, 403, 413, 435.
Syphilitic chorioido-retinitis, 403; iritis, 257, 258, 265, 403, 444.
Syringomyelia, 542 footnote.

Tabes Dorsalis, 310, 312, 313, 444, 501.
Tarsus, fatty degeneration of the, 100; tumour, 198.
Tarsoraphy, **210**, 229.
Tattooing the cornea, 187.
Teeth, diseases of the, 544; Hutchinson's, 172.
Tension of the eyeball, 314, 315.
Test-types, 20.
Trachoma, 95.
Transplantation of conjunctiva, 212; of cornea, 188; of skin, 216.
Trial lenses, numbering of the, 6.
Trichiasis, 214; operations for, 215.
Tubercle of the brain, 462; of the chorioid, 269, 287, 423; of the conjunctiva, 122, 193; of the iris, 269.
Tubercular meningitis, 310, 433.
Typhoid fever, 165, 166, 261, 438, 544.

Upright ophthalmoscopic image, 64.
Uræmia, 408, 409, 469.
Uræmic amblyopia, 469.
Uric acid in the conjunctiva, 134.
Uterine derangements, 402; hæmorrhage, 447.
Uveal tract, diseases of the, 254.

VACCINE vesicles on the eyelids, 193.
Van Millingen's operation for trichiasis, 220.
Vessel, cilio-retinal, 88.
Virtual focus, 4; image, 6.
Vision, acuteness of, 19; binocular, 526, 526 footnote, 535, 535 footnote; central, 21; eccentric, 22; field of, 22; field of, in glaucoma, 320.
Vision, defects of which disqualify for the army, 564; for the Army Medical Department, 564; for the British Mercantile Service, 567; for the Home Civil Service, 565; for the Indian Civil Service, 565; for the Indian Marine Service, 567; for the Indian Medical Service, 566; for the Navy, 565; for the Royal Irish Constabulary, 567.
Vision nulle, 460, 461.
Visual angle, the, 20; aphasia, 464; centre, 452; centre, lesion at the, 459; line, 15; memory, 464, 466; subsenses, 16.
Vitreous humour, cysticercus in the, 397; detachment of the, 389; diseases of the, **389**, 426; fluidity of the, 390, **392**; foreign bodies in the, 393; hæmorrhage in the, 251, 390, 408, 414; inflammatory processes in the, 389, 394; muscæ volitantes of the, 392; opacities in the, 39, 122, 250, 261, 275, 276, 279, **389**, 391, 403; persistent hyaloid artery in the, 398; purulent inflammation of the, 281, 389, 394; vessels in the, 398.
Von Græfe's operation for cataract, 360; for conical cornea, 183; for entropium, 226; in sympathetic ophthalmitis, 297.
Vossius's operation for trichiasis, 319.

WERNICKE'S pupil-symptom, 462.
Wharton Jones's operation for ectropium, 232.
Whooping cough, 166, 282.
Word-blindness, 464.

XANTHELASMA of the eyelids, 200.
Xerophthalmia, 120, 125, 126, 469; with ulceration of cornea, 163.

YOUNG-HELMHOLTZ theory of the colour-sense, 17.

ZONULA of Zinn, change of, in accommodation, 10.
Zonular cataract, 349.

SELECTED LIST
OF
NEW AND RECENT WORKS
PUBLISHED BY
H. K. LEWIS,
136 GOWER STREET, LONDON, W.C.
(ESTABLISHED 1844).

*** *For full list of works in Medicine and Surgery published by H. K. Lewis see complete Catalogue sent post free on application.*

D'ARCY POWER, M.A., M.B. OXON., F.R.C.S. ENG.
Demonstrator of Operative Surgery at St. Bartholomew's Hospital; Surgeon to the Victoria Hospital for Children, Chelsea; Examiner in the University of Durham; Member of the Conjoint Examining Board of the Royal College of Physicians (Lond.) and of Surgeons (Eng.)

THE SURGICAL DISEASES OF CHILDREN AND THEIR TREATMENT BY MODERN METHODS. With Illustrations, crown 8vo, 10s. 6d. [*Now ready.*

DR. A. DUHRSSEN.
Privat Docent in Midwifery and Gynæcology in the University of Berlin.

A MANUAL OF GYNÆCOLOGICAL PRACTICE FOR STUDENTS AND PRACTITIONERS. Translated and Edited from the fourth German edition, by JOHN W. TAYLOR, F.R.C.S., Surgeon to the Birmingham and Midland Hospital for Women, &c.; and FREDERICK EDGE, M.D. LOND., M.R.C.P., F.R.C.S., Surgeon to the Wolverhampton and District Hospital for Women. With 120 Illustrations, cr. 8vo, 6s. [*Now ready.*

DR. A. ONODI.
Lecturer on Rhino-Laryngology in the University of Budapest.

THE ANATOMY OF THE NASAL CAVITY, AND ITS ACCESSORY SINUSES. An Atlas for Practitioners and Students, translated by ST CLAIR THOMSON, M.D. LOND., F.R.C.S. ENG., M.R.C.P. LOND. Plates, small 4to, 6s. *nett.*
[*Now ready.*

JAMES F. GOODHART, M.D. ABERD., F.R.C.P.
Physician to Guy's Hospital, and Consulting Physician to the Evelina Hospital for Sick Children.

ON COMMON NEUROSES; OR THE NEUROTIC ELEMENT IN DISEASE AND ITS RATIONAL TREATMENT. Second edition, crown 8vo, 3s. 6d.

JAMES ANDERSON, M.D., F.R.C.P.
Late Assistant Physician to the London Hospital, &c.

NOTES ON MEDICAL NURSING; from the Lectures given to the Probationers at the London Hospital. Edited by E. F. LAMPORT, Associate of the Sanitary Institute. With an Introductory Biographical Notice by the late SIR ANDREW CLARK, BART. Second edition, crown 8vo, 2s. 6d. [*Now ready.*

HARRISON ALLEN, M.D.
Consulting Physician to Rush Hospital for Consumption.

A HANDBOOK OF LOCAL THERAPEUTICS. General Surgery by R. H. HARTE, M.D., Surgeon to the Episcopal and St. Mary's Hospitals; Diseases of the Skin by A. VAN HARLINGEN, M.D., Professor of Diseases of the Skin in the Philadelphia Polyclinic; Diseases of the Ear and Air Passages by H. ALLEN, M.D.; Diseases of the Eye by G. C. HARLAN, M.D., Surgeon to Wills Eye Hospital. Edited by H. ALLEN, M.D. Large 8vo, 14s. *nett.*

E. CRESSWELL BABER, M.B. LOND.
Surgeon to the Brighton and Sussex Throat and Ear Hospital.

A GUIDE TO THE EXAMINATION OF THE NOSE WITH REMARKS ON THE DIAGNOSIS OF DISEASES OF THE NASAL CAVITIES. With Illustrations, small 8vo, 5s. 6d.

FANCOURT BARNES, M.D., M.R.C.P.
Physician to the Chelsea Hospital; Obstetric Physician to the Great Northern Hospital, &c.

A GERMAN-ENGLISH DICTIONARY OF WORDS AND TERMS USED IN MEDICINE AND ITS COGNATE SCIENCES. Square 12mo, Roxburgh binding, 9s.

H. CHARLTON BASTIAN, M.A., M.D., F.R.S.
Examiner in Medicine at the Royal College of Physicians; Physician to University College Hospital, etc.

I.

PARALYSES: CEREBRAL, BULBAR, AND SPINAL. A Manual of Diagnosis for Students and Practitioners. With numerous Illustrations, 8vo, 12s. 6d.

II.

VARIOUS FORMS OF HYSTERICAL OR FUNCTIONAL PARALYSIS. Demy 8vo, 7s. 6d.

DRS. BOURNEVILLE AND BRICON.

MANUAL OF HYPODERMIC MEDICATION. Translated from the Second Edition, and Edited, with Therapeutic Index of Diseases, by ANDREW S. CURRIE, M.D. Edin., etc. Crown 8vo, 3s. 6d.

RUBERT BOYCE, M.B., M.R.C.S.
Professor of Pathology in University College, Liverpool.

A TEXTBOOK OF MORBID HISTOLOGY FOR STUDENTS AND PRACTITIONERS. Royal 8vo, with 130 coloured figures, 31s. 6d.

CHARLES H. BURNETT, A.M., M.D.
Clinical Professor of Otology in the Woman's Medical College of Pennsylvania; Aural Surgeon to the Presbyterian Hospital, Philadelphia.

SYSTEM OF DISEASES OF THE EAR, NOSE AND THROAT. By 45 Eminent American, British, Canadian and Spanish Authors. Edited by CHARLES H. BURNETT. With Illustrations, in two imperial 8vo vols., 48s. *nett.* [*Now ready.*

HARRY CAMPBELL, M.D., B.S. LOND., M.R.C.P.
Physician to the North-West London Hospital.

I.

THE CAUSATION OF DISEASE. An exposition of the ultimate factors which induce it. Demy 8vo, 12s. 6d.

II.

FLUSHING AND MORBID BLUSHING, THEIR PATHOLOGY AND TREATMENT. With plates and wood engravings, royal 8vo, 10s. 6d.

III.

DIFFERENCES IN THE NERVOUS ORGANISATION OF MAN AND WOMAN, PHYSIOLOGICAL AND PATHOLOGICAL. Royal 8vo, 15s.

IV.

HEADACHE AND OTHER MORBID CEPHALIC SENSATIONS. Royal 8vo, 12s. 6d. [*Just Published.*

R. E. CARRINGTON, M.D., F.R.C.P.

NOTES ON PATHOLOGY. With an Introductory Chapter by J. F. GOODHART, M.D., F.R.C.P., Physician to Guy's Hospital. Edited by H. E. CROOK, M.D. Lond., F.R.C.S. Eng., and G. MACKESON, L.R.C.P., M.R.C.S. Cr. 8vo, 3s.

ALFRED H. CARTER, M.D. LOND., F.R.C.P.
Senior Physician to the Queen's Hospital; Professor of Therapeutics, Mason College, Birmingham, &c.

ELEMENTS OF PRACTICAL MEDICINE. Seventh Edition, crown 8vo, 10s. [*Now ready.*

FRANCIS HENRY CHAMPNEYS, M.A., M.B. OXON., F.R.C.P.
Physician Accoucheur and Lecturer on Obstetric Medicine at St. Bartholomew's Hospital; Examiner in Obstetric Medicine in the University of London, &c.

LECTURES ON PAINFUL MENSTRUATION. Royal 8vo, 7s. 6d.

W. BRUCE CLARKE, M.A., M.B. OXON., F.R.C.S.
Assistant Surgeon to, and Senior Demonstrator of Anatomy and Operative Surgery at St. Bartholomew's Hospital, &c.

THE DIAGNOSIS AND TREATMENT OF DISEASES OF THE KIDNEY AMENABLE TO DIRECT SURGICAL INTERFERENCE. Demy 8vo, with Illustrations, 7s. 6d.

WALTER S. COLMAN, M.B. LOND.
Pathologist and Registrar to the National Hospital for the Paralysed and Epileptic.

SECTION CUTTING AND STAINING: A Practical Guide to the Preparation of Normal and Morbid Histological Specimens. Illustrations, crown 8vo, 3s.

W. H. CORFIELD, M.A., M.D. OXON., F.R.C.P. LOND.
Professor of Hygiene and Public Health in University College, London.

DWELLING HOUSES: their Sanitary Construction and Arrangements. Third Edition, with Illustrations, crown 8vo., 3s. 6d. [*Now ready.*

EDWARD COTTERELL, M.R.C.S. ENG., L.R.C.P. LOND.

ON SOME COMMON INJURIES TO LIMBS: their Treatment and After-Treatment including Bone-Setting (so-called). Imp. 16mo, with Illustrations, 3s. 6d.

SIDNEY COUPLAND, M.D., F.R.C.P.
Physician to the Middlesex Hospital, and Lecturer on Practical Medicine in the Medical School, etc.

NOTES ON THE CLINICAL EXAMINATION OF THE BLOOD AND EXCRETA. Third Edition, 12mo, 1s. 6d.

CHARLES CREIGHTON, M.D.

ILLUSTRATIONS OF UNCONSCIOUS MEMORY IN DISEASE, including a Theory of Alteratives. Post 8vo, 6s.

H. RADCLIFFE CROCKER, M.D. LOND., B.S., F.R.C.P.
Physician for Diseases of the Skin in University College Hospital.

DISEASES OF THE SKIN: THEIR DESCRIPTION, PATHOLOGY, DIAGNOSIS, AND TREATMENT. Second edition, with Illustrations, 8vo, 24s.

EDGAR M. CROOKSHANK, M.B. LOND.
Professor of Bacteriology, King's College, London.

I.

MANUAL OF BACTERIOLOGY. Third Edition, coloured plates and wood engravings, 8vo, 21s.

II.

HISTORY AND PATHOLOGY OF VACCINATION. 2 vols., royal 8vo, coloured plates, 20s. *nett.*

HERBERT DAVIES, M.D., F.R.C.P.
Late Consulting Physician to the London Hospital, and formerly Fellow of Queen's College, Cambridge.

THE MECHANISM OF THE CIRCULATION OF THE BLOOD THROUGH ORGANICALLY DISEASED HEARTS. Edited by ARTHUR TEMPLER DAVIES, B.A., M.D. Cantab., M.R.C.P. Crown 8vo 3s. 6d.

HENRY DAVIS, M.R.C.S. ENG.
Teacher and Administrator of Anæsthetics at St. Mary's Hospital, and Assistant Anæsthetist at the Dental Hospital of London.

GUIDE TO THE ADMINISTRATION OF ANÆSTHETICS. Second edition, fcap. 8vo, 2s. 6d.

ALEXANDER S. FAULKNER.
Surgeon-Major, Indian Medical Service.

A GUIDE TO THE PUBLIC MEDICAL SERVICES. Compiled from Official Sources. Demy 8vo, 2s.
[Just Published.

W. SOLTAU FENWICK, M.D., B.S. LOND., M.R.C.P.
Assistant Physician to the Evelina Hospital for Sick Children, &c.

THE DYSPEPSIA OF PHTHISIS: Its Varieties and Treatment, including a Description of Certain Forms of Dyspepsia associated with the Tubercular Diathesis. Demy 8vo, 6s.
[*Now ready.*

J. MILNER FOTHERGILL, M.D.

I.

INDIGESTION AND BILIOUSNESS. Second Edition, post 8vo, 7s. 6d.

II.

GOUT IN ITS PROTEAN ASPECTS. Post 8vo, 7s. 6d.

III.

THE TOWN DWELLER: HIS NEEDS AND HIS WANTS. Post 8vo, 3s. 6d.

ERNEST FUCHS.
Professor of Ophthalmology in the University of Vienna.

TEXTBOOK OF OPHTHALMOLOGY. Translated from the Second German Edition by A. DUANE, M.D., Assistant Surgeon, Ophthalmic and Aural Institute, New York. Large 8vo, with 190 Illustrations, 21s.

PROFESSOR DR. PAUL FURBRINGER.
Director of the Friedrichshain Hospital, Berlin, &c.

TEXTBOOK OF DISEASES OF THE KIDNEYS AND GENITO-URINARY ORGANS. Translated by W. H. GILBERT, M.D., Physician in Baden-Baden, &c. Vol. I., demy 8vo, 7s. 6d.

SIR DOUGLAS GALTON, K.C.B., HON. D.C.L., LL.D., F.R.S.,
Formerly Secretary Railway Department Board of Trade; Assistant Inspector-General of Fortifications, &c.

HEALTHY HOSPITALS. OBSERVATIONS ON SOME POINTS CONNECTED WITH HOSPITAL CONSTRUCTION. With Illustrations, 8vo, 10s. 6d.
[*Now ready.*

JOHN HENRY GARRETT, M.D.
Licentiate in Sanitary Science and Diplomate in Public Health, Universities of Durham and Cambridge, &c.

THE ACTION OF WATER ON LEAD: being an inquiry into the cause and mode of the action and its prevention. Crown 8vo, 4s. 6d.

ALFRED W. GERRARD, F.C.S.

Examiner to the Pharmaceutical Society; Teacher of Pharmacy and Demonstrator of Materia Medica to University College Hospital, etc.

ELEMENTS OF MATERIA MEDICA AND PHARMACY. With the New Official Remedies, 1890. Crown 8vo, 8s. 6d.

JOHN GORHAM, M.R.C.S.

Fellow of the Physical Society of Guy's Hospital, etc.

TOOTH EXTRACTION: A manual on the proper mode of extracting teeth. Fourth edition, fcap. 8vo, 1s. 6d.

GEORGE M. GOULD, A.M., M.D.

Ophthalmic Surgeon to the Philadelphia Hospital, etc.

I.

A NEW MEDICAL DICTIONARY. A compact concise Vocabulary, convenient for reference, based on recent medical literature. Small 8vo, 12s. 6d.

II.

A POCKET MEDICAL DICTIONARY, giving the Pronunciation and Definition of about 12,000 of the Principal Words used in Medicine and the Collateral Sciences. 32mo, 4s. *nett.*

LANDON C. GRAY, M.D.

Professor of Nervous and Mental Diseases in the New York Polyclinic; Visiting Physician to St. Mary's Hospital, &c.

A TREATISE ON NERVOUS AND MENTAL DISEASES FOR STUDENTS AND PRACTITIONERS OF MEDICINE. With 168 Illustrations, 8vo, 21s.

DR. JOSEF GRUBER.

Professor of Otology in the University of Vienna, &c.

A TEXT-BOOK OF THE DISEASES OF THE EAR. Translated from the second German edition, and Edited with additions by EDWARD LAW, M.D., C.M. EDIN., M.R.C.S. ENG., Surgeon to the London Throat Hospital for Diseases of the Throat, Nose and Ear; and COLEMAN JEWELL, M.B. LOND., M.R.C.S. ENG. Second edition, with 165 Illustrations, and 70 coloured figures on 2 lithographic plates, royal 8vo, 28s.

BERKELEY HILL, M.B. LOND., F.R.C.S.
Professor of Clinical Surgery in University College
AND
ARTHUR COOPER, L.R.C.P., M.R.C.S.
Surgeon to the Westminster General Dispensary, &c.

SYPHILIS AND LOCAL CONTAGIOUS DISORDERS. Second Edition, entirely re-written, royal 8vo, 18s.

NORMAN KERR, M.D., F.L.S.
President of the Society for the Study of Inebriety; Consulting Physician, Dalrymple Home for Inebriates, etc.

INEBRIETY: ITS ETIOLOGY, PATHOLOGY, TREATMENT, AND JURISPRUDENCE. Third Edition, 8vo, 21s. [*Now ready.*

F. CHARLES LARKIN, F.R.C.S. ENG.
Surgeon to the Stanley Hospital,
AND
RANDLE LEIGH, M.B., B.SC. LOND.
Senior Demonstrator of Physiology in University College, Liverpool.

OUTLINES OF PRACTICAL PHYSIOLOGICAL CHEMISTRY. Second Edition, with Illustrations, crown 8vo, 2s. 6d. *nett.*

J. WICKHAM LEGG, F.R.C.P.
Formerly Assistant Physician to Saint Bartholomew's Hospital, and Lecturer on Pathological Anatomy in the Medical School.

A GUIDE TO THE EXAMINATION OF THE URINE. Seventh Edition, edited and revised by H. LEWIS JONES, M.D., Medical Officer in charge of the Electrical Department in St. Bartholomew's Hospital. With Illustrations, fcap. 8vo, 3s. 6d. [*Now ready.*

LEWIS'S POCKET MEDICAL VOCABULARY.
Second Edition, 32mo, limp roan, 3s. 6d.

WILLIAM THOMPSON LUSK, A.M., M.D.
Professor of Obstetrics and Diseases of Women in the Bellevue Hospital Medical College, &c.

THE SCIENCE AND ART OF MIDWIFERY. Fourth Edition, rewritten, with numerous Illustrations, 8vo, 18s.
[*Just published.*

LEWIS'S PRACTICAL SERIES.

In Crown 8vo Volumes, with Illustrations.

THE SURGICAL DISEASES OF CHILDREN AND THEIR TREATMENT BY MODERN METHODS. By D'ARCY POWER, M.A., M.B. Oxon., F.R.C.S. Eng., Demonstrator of Operative Surgery at St. Bartholomew's Hospital, &c. With Illustrations, 10s. 6d.
[*Now ready*

DISEASES OF THE NOSE AND THROAT.
By F. de HAVILLAND HALL, M.D., F.R.C.P. Lond., Physician in Charge of the Throat Department at the Westminster Hospital, &c. 10s. 6d

PUBLIC HEALTH LABORATORY WORK.
By H. R. KENWOOD, M.B., D.P.H., F.C.S., Instructor in the Hygienic Laboratory, University College, London, &c. 10s. 6d.

MEDICAL MICROSCOPY.
By FRANK J. WETHERED, M.D., M.R.C.P., Medical Registrar to the Middlesex Hospital. 9s.

MEDICAL ELECTRICITY.
By H. LEWIS JONES, M.A., M.D., M.R.C.P., Medical Officer in charge of the Electrical Department in St. Bartholomew's Hospital. Second Edition, 10s. 6d.

HYGIENE AND PUBLIC HEALTH.
By LOUIS C. PARKES, M.D., D.P.H. Lond. Univ., Lecturer on Public Health at St. George's Hospital, etc. Fourth Edition, 10s. 6d.

MANUAL OF OPHTHALMIC PRACTICE.
By C. HIGGENS, F.R.C.S., Lecturer on Ophthalmology at Guy's Hospital Medical School, &c. Illustrations, crown 8vo, 6s.

A PRACTICAL TEXTBOOK OF THE DISEASES OF WOMEN. By ARTHUR H. N. LEWERS, M.D. Lond., M.R.C.P., Obstetric Physician to the London Hospital. Fourth Edition, 10s. 6d.

ANÆSTHETICS, THEIR USES AND ADMINISTRATION.
By DUDLEY W. BUXTON, M.D., B.S., M.R.C.P., Administrator of Anæsthetics at University College Hospital, etc. Second Edition, 5s.

TREATMENT OF DISEASE IN CHILDREN.
By ANGEL MONEY, M.D., F.R.C.P. Second Edition, 10s. 6d.

ON FEVERS: THEIR HISTORY, ETIOLOGY, DIAGNOSIS, PROGNOSIS, AND TREATMENT. By ALEXANDER COLLIE, M.D. (Aberdeen). 8s. 6d.

HANDBOOK OF DISEASES OF THE EAR.
By URBAN PRITCHARD, M.D. (Edin.), F.R.C.S., Professor of Aural Surgery at King's College, London, etc. Third Edition.

A PRACTICAL TREATISE ON DISEASES OF THE KIDNEYS AND URINARY DERANGEMENTS. By C. H. RALFE, M.A., M.D. Cantab., F.R.C.P., Physician to the London Hospital. 10s. 6d.

DENTAL SURGERY FOR GENERAL PRACTITIONERS AND STUDENTS OF MEDICINE. By ASHLEY W. BARRETT, M.B. Lond., M.R.C.S., L.D.S., Dental Surgeon to the London Hospital. Second Edition, 3s. 6d

BODILY DEFORMITIES AND THEIR TREATMENT.
By H. A. REEVES, F.R.C.S. Ed., Senior Assistant Surgeon and Teacher of Practical Surgery at the London Hospital. 8s. 6d.

JEFFERY A. MARSTON, M.D., C.B., F.R.C.S., M.R.C.P. LOND.
Surgeon General Medical Staff (Retired).

NOTES ON TYPHOID FEVER: Tropical Life and its Sequelæ. Crown 8vo, 3s. 6d.

WILLIAM MARTINDALE, F.C.S.
AND
W. WYNN WESTCOTT, M.B. LOND.

THE EXTRA PHARMACOPŒIA with the additions introduced into the British Pharmacopœia 1885 and 1890; and Medical References, and a Therapeutic Index of Diseases and Symptoms. Eighth Edition, limp roan, med. 24mo, 9s.

A. STANFORD MORTON, M.B., F.R.C.S. ENG.
Assistant Surgeon to the Moorfields Ophthalmic Hospital, &c.

REFRACTION OF THE EYE: Its Diagnosis, and the Correction of its Errors, with Chapter on Keratoscopy. Fifth Edition, small 8vo, 3s. 6d.

C. W. MANSELL MOULLIN, M.A., M.D. OXON., F.R.C.S. ENG.
Surgeon and Lecturer on Physiology at the London Hospital, &c.

I.

SPRAINS; THEIR CONSEQUENCES AND TREATMENT. Second Edition, crown 8vo, 4s. 6d.

II.

ENLARGEMENT OF THE PROSTATE: its Treatment and Radical Cure. With plates, 8vo, 6s.
[*Now ready.*

WILLIAM MURRAY, M.D., F.R.C.P. LOND.
Consulting Physician to the Children's Hospital, Newcastle-on-Tyne, &c.

ILLUSTRATIONS OF THE INDUCTIVE METHOD IN MEDICINE. Crown 8vo, 3s. 6d.

WILLIAM MURRELL, M.D., F.R.C.P.
Lecturer on Materia Medica and Therapeutics at Westminster Hospital.

I.

MASSOTHERAPEUTICS; OR MASSAGE AS A MODE OF TREATMENT. Fifth Edition, crown 8vo, 4s. 6d.

II.

WHAT TO DO IN CASES OF POISONING. Seventh Edition, royal 32mo, 3s. 6d. [*Now ready.*

G. OLIVER, M.D., F.R.C.P.

I.

PULSE-GAUGING: A Clinical Study of Radial Measurement and Pulse Pressure. With Illustrations, fcap. 8vo, 3s. 6d. [*Now ready.*

II.

ON BEDSIDE URINE TESTING: a Clinical Guide to the Observation of Urine in the course of Work. Fourth Edition, fcap. 8vo, 3s. 6d.

III.

THE HARROGATE WATERS: Data Chemical and Therapeutical, with notes on the Climate of Harrogate. Crown 8vo, with Map of the Wells, 3s. 6d.

WILLIAM OSLER, M.D., F.R.C.P. LOND.

Professor of Medicine, Johns Hopkins University, &c.

ON CHOREA AND CHOREIFORM AFFECTIONS. Large 8vo, 5s. [*Now ready.*

LOUIS C. PARKES, M.D. LOND., D.P.H.

Lecturer on Public Health at St. George's Hospital, &c.

INFECTIOUS DISEASES, NOTIFICATION AND PREVENTION. Fcap. 8vo, roan, 4s. 6d.

R. DOUGLAS POWELL, M.D., F.R.C.P.,

Physician Extra-ordinary to H.M. the Queen; Physician to the Middlesex Hospital; Consulting Physician to the Hospital for Consumption and Diseases of the Chest at Brompton.

DISEASES OF THE LUNGS AND PLEURÆ INCLUDING CONSUMPTION. Fourth Edition, with coloured plates and wood-engravings, 8vo, 18s. [*Now ready.*

DR. THEODOR PUSCHMANN.

Public Professor in Ordinary at the University of Vienna.

HISTORY OF MEDICAL EDUCATION FROM THE MOST REMOTE TO THE MOST RECENT TIMES. Translated by EVAN H. HARE, M.A. (OXON.), F.R.C.S. (ENG.), F.S.A. Demy 8vo, 21s.

FRANCIS H. RANKIN, M.D.

President of the Newport Medical Society.

HYGIENE OF CHILDHOOD: Suggestions for the care of children after the period of infancy to the completion of puberty. Crown 8vo, 3s.

SAMUEL RIDEAL, D.SC. (LOND.), F.I.C., F.C.S., F.G.S.
Fellow of University College, London.

I.

PRACTICAL ORGANIC CHEMISTRY. The detection and properties of some of the more important organic compounds. 12mo, 2s. 6d.

II.

PRACTICAL CHEMISTRY FOR MEDICAL STUDENTS, Required at the First Examination of the Conjoint Examining Board in England. Fcap. 8vo, 2s.

J. JAMES RIDGE, M.D., B.S., B.A., B.SC. LOND.
Medical Officer of Health, Enfield.

ALCOHOL AND PUBLIC HEALTH. Second Edition, Crown 8vo, 2s. [*Now ready.*

SYDNEY RINGER, M.D., F.R.S.
Holme Professor of Clinical Medicine in University College; Physician to University College Hospital.

A HANDBOOK OF THERAPEUTICS. Twelfth Edition, revised, 8vo, 15s.

FREDERICK T. ROBERTS, M.D., B.SC., F.R.C.P.
Professor of the Principles and Practice of Medicine in University College Physician to University College Hospital, &c.

I.

THE THEORY AND PRACTICE OF MEDICINE. Ninth Edition, with Illustrations, large 8vo, 21s. [*Now ready.*

II.

THE OFFICINAL MATERIA MEDICA. Second Edit., entirely rewritten in accordance with the latest British Pharmacopœia, with the Additions made in 1890, fcap. 8vo, 7s. 6d

---

ROBSON ROOSE, M.D., F.R.C.P. EDIN.

I.

GOUT, AND ITS RELATIONS TO DISEASES OF THE LIVER AND KIDNEYS. Seventh Edition, crown 8vo, 4s. 6d.

II.

NERVE PROSTRATION AND OTHER FUNCTIONAL DISORDERS OF DAILY LIFE. Second Edition, demy 8vo, 18s.

WILLIAM ROSE, B.S., M.B. LOND., F.R.C.S.
Professor of Surgery in King's College, London, and Surgeon to King's College Hospital, &c.

ON HARELIP AND CLEFT PALATE. Demy 8vo, with Illustrations, 6s.

BERNARD ROTH, F.R.C.S.
Fellow of the Medical Society of London.

THE TREATMENT OF LATERAL CURVATURE OF THE SPINE. Demy 8vo, with Photographic and other Illustrations, 5s.

DR. C. SCHIMMELBUSCH.
Privat-docent and Assistant Surgeon in Prof. v. Bergmann's University Clinic at Berlin.

THE ASEPTIC TREATMENT OF WOUNDS.
With a Preface by Professor Dr. E. von Bergmann. Translated from the Second German Edition by A. T. Rake, M.B., B.S. Lond., F.R.C.S. Eng., Registrar and Pathologist to the East London Hospital for Children. With Illustrations, crown 8vo, 5s.
[*Now ready.*

G. E. SHUTTLEWORTH, B.A., M.D.
Late Medical Superintendent, Royal Albert Asylum for Idiots and Imbeciles of the Northern Counties, Lancaster; formerly Assistant Medical Officer, Earlswood Asylum.

MENTALLY-DEFICIENT CHILDREN: THEIR TREATMENT AND TRAINING. With Illustrations, crown 8vo, 4s. [*Just published.*

ALEXANDER J. C. SKENE, M.D.
Professor of Gynæcology in the Long Island College Hospital, Brooklyn.

TREATISE ON THE DISEASES OF WOMEN.
Second Edition, with 251 engravings and 9 chromo-lithographs, medium 8vo, 28s.

JOHN KENT SPENDER, M.D. LOND.
Physician to the Royal Mineral Water Hospital, Bath.

THE EARLY SYMPTOMS AND THE EARLY TREATMENT OF OSTEO-ARTHRITIS, commonly called Rheumatoid Arthritis. With special reference to the Bath Thermal Waters. Small 8vo, 2s. 6d.

LEWIS A. STIMSON, B.A., M.D.
Professor of Clinical Surgery in the Medical Faculty of the University of the City of New York, etc.

A MANUAL OF OPERATIVE SURGERY. With three hundred and forty-two Illustrations. Second Edition, post 8vo, 10s. 6d.

ADOLF STRÜMPELL.
Professor and Director of the Medical Clinique at Erlangen.

A TEXT-BOOK OF MEDICINE FOR STUDENTS AND PRACTITIONERS. Second edition translated from the German by Dr. H. F. VICKERY and Dr. P. C. KNAPP, with Editorial Notes by Dr. F. C. SHATTUCK, Visiting Physician to the Massachusetts General Hospital, &c. Complete in one large vol., with 119 Illustrations, imp. 8vo, 28s.

JUKES DE STYRAP, M.K.Q.C.P.
Physician-Extraordinary, late Physician in Ordinary to the Salop Infirmary; Consulting Physician to the South Salop and Montgomeryshire Infirmaries, etc.

I.

THE YOUNG PRACTITIONER: With practical hints and instructive suggestions, as subsidiary aids, for his guidance on entering into private practice. Demy 8vo, 7s. 6d. *nett.*

II.

A CODE OF MEDICAL ETHICS: With general and special rules for the guidance of the faculty and the public in the complex relations of professional life. Fourth edition, demy 8vo, 3s. 6d. *nett.*

III.

MEDICO-CHIRURGICAL TARIFFS. Fifth edition, fcap. 4to, 2s. *nett.*

IV.

THE YOUNG PRACTITIONER: HIS CODE AND TARIFF. Being the above three works in one volume. Demy 8vo, 10s. 6d. *nett.*

C. W. SUCKLING, M.D. LOND., M.R.C.P.
Professor of Materia Medica and Therapeutics at the Queen's College, Physician to the Queen's Hospital, Birmingham, etc.

I.

ON THE DIAGNOSIS OF DISEASES OF THE BRAIN, SPINAL CORD, AND NERVES. With Illustrations, crown 8vo, 8s. 6d.

II.

ON THE TREATMENT OF DISEASES OF THE NERVOUS SYSTEM. Crown 8vo, 7s. 6d.

JOHN BLAND SUTTON, F.R.C.S.
Lecturer on Comparative Anatomy, and Assistant Surgeon to the Middlesex Hospital.

LIGAMENTS: THEIR NATURE AND MORPHOLOGY. Wood engravings, post 8vo, 4s. 6d.

HENRY R. SWANZY, A.M., M.B., F.R.C.S.I.
Examiner in Ophthalmic Surgery in the Royal University of Ireland; Surgeon to the National Eye and Ear Infirmary, Dublin, etc.

A HANDBOOK OF DISEASES OF THE EYE AND THEIR TREATMENT. Fifth Edition, Illustrated with Wood Engravings, Colour Tests, etc., large post 8vo, 10s. 6d.

ALBERT TAYLOR.
Associate Sanitary Institute; Chief Sanitary Inspector to the Vestry of St George, Hanover Square, etc.

THE SANITARY INSPECTOR'S HANDBOOK. Cr. 8vo, with Illustrations, 5s.

A. J. WALL, M.D. LOND., F.R.C.S. ENG.
Medical Staff of H.M. Indian Army (Retired List).

ASIATIC CHOLERA: ITS HISTORY, PATHOLOGY, AND MODERN TREATMENT. Demy 8vo, 6s.

E. G. WHITTLE, M.D. LOND., F.R.C.S. ENG.
Senior Surgeon to the Royal Alexandra Hospital, for Sick Children, Brighton.

CONGESTIVE NEURASTHENIA, OR INSOMNIA AND NERVE DEPRESSION. Crown 8vo, 3s. 6d.

SIR JOHN WILLIAMS, M.D., F.R.C.P.
Professor of Midwifery in University College, London, &c.

CANCER OF THE UTERUS: BEING THE HARVEIAN LECTURES FOR 1886. Illustrated with Lithographic Plates, royal 8vo, 10s. 6d.

E. T. WILSON, M.B. OXON., F.R.C.P. LOND.
Physician to the Cheltenham General Hospital, &c.

DISINFECTANTS AND ANTISEPTICS: HOW TO USE THEM. In Packets of one doz. price 1s., by post 1s. 1d. [*Just thoroughly revised.*

BERTRAM C. A. WINDLE, M.A., M.D. DUBL.
Professor of Anatomy in the Queen's College, Birmingham; Examiner in Anatomy in the Universities of Cambridge and Durham.

A HANDBOOK OF SURFACE ANATOMY AND LANDMARKS. Illustrations, post 8vo, 3s. 6d.

OSWALD ZIEMSSEN, M.D.
Knight of the Iron Cross, and of the Prussian Order of the Crown.

THE TREATMENT OF CONSTITUTIONAL SYPHILIS. Post 8vo, 3s. 6d.

LEWIS'S DIET CHARTS. A Suggestive set of Diet Tables for the use of Physicians, for handing to Patients after Consultation, modified to suit Individual Requirements; for Albuminuria, Alcoholism, Anæmia and Debility, Constipation, Diabetes, Diarrhœa, Dyspepsia, Fevers, Gout, Nervous Diseases, Obesity, Phthisis, Rheumatism (chronic); with Blank Chart for other diseases. 6s. 6d. per packet of 100 charts, by post, 6s. 10½d.

LEWIS'S FOUR-HOUR TEMPERATURE CHART. This form has been drawn up to meet the requirements of a chart on which the temperature and other observations can be recorded at intervals of four hours. They will be found most convenient in hospital and private practice. Each chart will last a week. Prices, 20, 1s.; 50, 2s.; 100, 3s. 6d.; 500, 14s.; 1000, 25s.

CHART FOR RECORDING THE EXAMINATION OF URINE. These Charts are designed for the use of medical men, analysts and others making examinations of the urine of patients, and afford a very ready and convenient method of recording the results of the examination. Prices, 10, 1s.; 100, 7s. 6d.; 250, 15s.; 500, 25s.; 1000, 40s.

CLINICAL CHARTS FOR TEMPERATURE OBSERVATIONS, ETC. Arranged by W. RIGDEN, M.R.C.S. Price 1s. per doz., 7s. per 100, 15s. per 250, 28s. per 500, 50s. per 1000.

Each Chart is arranged for four weeks, and is ruled at the back for making notes of cases; they are convenient in size, and are suitable both for hospital and private cases.

LEWIS'S CLINICAL CHART, SPECIALLY DESIGNED FOR USE WITH THE VISITING LIST. This Temperature Chart is arranged for four weeks, and measures 6 × 3 inches. 30s. per 1000, 16s. 6d. per 500, 3s. 6d. per 100, 1s. per 25, 6d per 12.

LEWIS'S NURSING CHART. 25s. per 1000, 14s. per 500, 3s. 6d. per 100, 2s. per 50, or 1s. per 20.

*** MR. LEWIS is in constant communication with the leading publishing firms in America and has transactions with them for the sale of his publications in that country. Advantageous arrangements are made in the interests of Authors for the publishing of their works in the United States.

MR. LEWIS'S publications can be procured of any Bookseller in any part of the world.

Complete Catalogue of Publications post free on application.

Printed by H K. Lewis, Gower Street, London, W.C.

CARD EXPLANATORY OF HOLMGREN'S TESTS FOR COLOUR BLINDNESS.

Test I.

Confusion Colours.

1 2 3 4 5

Test II. *a.*

Red Blindness. 6 7

Green Blindness. 8 9

Test II. *b.*

Red Blindness. 10 11

Green Blindness. 12 13

N.B.—*This Card is merely intended to illustrate the text (The Colour Sense, Chap. I. and Appendix I.), and not itself for use as a test.*