Student's Companion

IN

OPHTHALMOLOGY

BY

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முக்கிய அறிவித்தல்

சாவகச்சேரி பிரதேச சபை
பொது நூலகம்

நீங்கள் எடுத்துச் செல்லும் புத்தகத்தில் கிறுக்கல், வெட்டுதல், கிழித்தல், அழித்தல், அழுக்குப்படியவிடல், எழுதுதல், கிறுக்கல், பக்கங்களை மடித்தல்மற்றும் ஊறுபாடுகளைச் செய்ய வேண்டாமெனக் கேட்டுக் கொள்கிறோம். புத்தகங்களை நீங்கள் எடுக்கும் போது இத்தகைய குறைபாடுகளைக் கண்டால் நூலகப் பொறுப்பாளரிற்கு உடன் தெரிவிக்கவும் அல்லாவிடில் நீங்கள் எடுத்துச் செல்லும் புத்தகம் நல்ல நிலையில் இருந்ததெனக் கருதப்படுவதுடன் ஊறுபாடுகளிற்கு நூலகப் பொறுப்பாளரினால் விதிக்கப்படும் தண்டத்தையும் நீங்கள் ஏற்கவேண்டிய நிர்ப்பந்தமும் ஏற்படும்.

குறிப்பிட்ட நாளுக்குப் பிந்தும் ஒவ்வொரு நாளிற்கும் 50சத வீதம் குற்றப் பணம் அறவிடப்படும். பொது நூலகத்தின் சிறப்பான சேவைக்காகவும், வாசகர்களின் நலனிற்காகவும் தங்களின் ஒத்துழைப்பு மிகவும் அவசியமாகத் தேவைப்படுகின்றது.

விசேட ஆணையாளர்,
சாவகச்சேரி பிரதேச சபை.

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TO MY FATHER

PREFACE

This little pocket book is the outcome of the compilation of notes made for the teaching of undergraduates, post-graduates, house surgeons and nurses. It is to be used at the bedside and in the clinic as a ready reference and for making notes for which blank leaves are bound.

It is the practice in the U. K. for teachers to hand out to students typed or printed notes so as to make intelligible what the teacher was going to say. This booklet was intended to achieve the same object but as eye specialists are few and blindness is rampant in this country a note book of this type with additions made by the "student" will serve as a useful guide to his practice after graduation.

Criticisms and suggestions will be most welcome.

P. S.

5th Dec. 1957.

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METHODS OF EXAMINATION

Introduction

The eye and its adnexa are in a prominent and easily accessible place on the face. Despite the complexity of the visual organ and the numerous minute investigations possible with highly specialized apparatus, several simple clinical methods are available for the examination of the eye from which valuable deductions may be made regarding the disease process, its aetiology and prognosis. By the same methods may be assessed the progress of a case and proper treatment instituted or altered according to the needs.

1. Equipment

i. Test Types
   (a) Distance—Snellen's
   (b) Near—Snellen's
   (c) Near—Jaeger
   (d) Near—Faculty of Ophthalmologists'

ii. Condensing Lens (+ 13) — convex lens of focal length 7 cm.

iii. A corneal magnifying lens (loupe) of x 8 or x 10 diameters.

iv. An electric lamp on a movable arm.

v. A plane and concave mirror with central perforations—retinoscope.

iv. Ophthalmoscope
   (a) Non-luminous (Morton's)
   (b) Self luminous (battery or mains model)

2. Examination

It should not be forgotten that the eye is part of the whole organism and so should not be treated as an organ islolated from the rest of the body. Besides, the eye has a duplex origin—a part an outgrowth of the brain and another derived from ectoderm.

Incorporated in the eye are also mesodermal structures, the sclera, the extraocular muscles and blood vessels. Hence it is common for certain eye manifestations to appear in such diverse conditions as nerve diseases, skin and joint disorders and vascular diseases. From this brief resume it would be apparent that in all patients reporting with eye complaints a casual glance at the body generally will provide corroborative evidence of a diagnosis already made or at least provide a clue to the eye affection. In typically ocular affections like conjunctivitis there may not be any general manifestations though even here a meningococcal meningitis or gonorrhoeal urethritis or an exanthem may coexist.

Examination of the eye may be conveniently divided into :-

I. External
II. Functional
III. Dark Room

3. I. External : Conducted by (a) Inspection, (b) Palpation (c) Auscultation.

(a) Inspection

If ambulant, the patient's manner of progression will give a clue to the nature of affection e. g., in the case of cataract the patient walks about fairly confidently whereas in glaucoma even with good central vision he stumbles into objects in his way. A person with optic atrophy will not move without aid for if he does he will meet with calamity. Incidentally the gait may reveal other defects e. g., ataxia.

The following is an easy scheme to adopt in examining the eye:-

Tension—digital (say N or +, +2 or—, —2)

Eyelids—Colour, position, swelling, fissure, retraction, ptosis, lashes—number of rows, dropping of lashes. Lid borders : ulceration, coloboma, entropion, ectropion.

Conjunctiva—Colour, blood vessels, type of injection, growths. (Both palbebral and bulbar conjunctivae should be seen)

Lacrimal apparatus—Gland, sac ; and swelling, regurgitation of mucus or pus from the latter. Puncta: position of these, patency. Canaliculi : swelling, discharge, concretions, foreign bodies.

Extraocular Muscles—Any obvious squint or nystagmus. Actions, diplopia test, cover test. Convergence.

Cornea—Size, shape, clarity, colour. SENSATION. Ulceration, abrasion, foreign body, blood vessels.

Anterior Chamber—Depth, contents: Pus—hypopyon Blood—hypaema ; foreign body.

Iris—Colour, pattern, hyper or hypopigmentation steadiness or tremulousness (iridodonesis).—Coloboma (defect), growths, exudates, foreign bodies.

Pupil—Size, reaction : direct and consensual to light. Regularity. Position (central ?) Effect of accommodation.

Lens—Position, steadiness, clarity. Absence (aphakia).

Vitreous—Clarity, solidity, abnormal contents.

Fundus—Nature of reflex. Is the fundus reflex uniformly red or grey in parts ?

(a) DISC—Colour, circumference, circulation, cupping, curiosities.

(b) MACULA

(c) PERIPHERY

(b) Palpation

This method of examining an eye has less value than in other parts of the body. However the same principles govern palpation of swellings of the ocular

adnexa, viz., lids, lacrimal apparatus and the orbit. Digital tonometry (§ 4) is a special application of palpation.

Surgical emphysema, the feel of a lipoma, the 'bag of worms' feel in neurofibroma, pulsations of aneurysmal swellings are parallel to these in other parts of the body. The margins of the orbit should be palpated in all cases of injury.

Under the subject of proptosis special palpatory methods are described (cf. proptosis).

Pressure on the globe with a finger causes pain in two well known conditions. In iridocyclitis and endophthalmitis pressure over the ciliary body region brings on pain—this is known as ciliary tenderness. Pain is also caused by pressing on the globe backwards in retrobulbar neuritis.

In testing for patency of the lacrimal passages pressure is exerted over the lacrimal sac with a finger. In cases of nasolacrimal duct obstruction regurgitation of tears, mucopus or pus occurs; in some such cases the pent up sac discharges its contents into the nose.

(c) Auscultation

This, the least practised method in ophthalmology, assumes first rate importance in cases of orbital and intracranial aneurysms for the presence of a bruit over the eye or the temple is convincing proof of an aneurysm in one of those sites. *It should be a golden rule to auscultate over the eyeball and temple in every case of proptosis.*

SOME CLINICAL METHODS

4. Digital Tonometry (Tension)

The patient is asked to look down and the fore-fingers of the two hands are placed over the upper lid of the eye to be examined in close proximity to each other and pressure applied over the globe with one finger while the other receives the impulse through the globe. The same process is repeated in the reverse order. Thus a series of indentations with one finger and registration with the other gives the examiner an impression of the hardness of the eye examined. The two eyes are examined for comparison. If the resistance offered to the fingers is great the tension is denoted T + or T + 2. If normal it is denoted N, if low T —.

5. Instrumental Tonometry

The most commonly used tonometer is the one advocated by Schiotz and its use will be described here. A surface anaesthetic, holocaine, is instilled into both eyes and the patient is made to lie flat on a table or bed. With the lids held apart the foot piece of the tonometer is placed on the cornea while the patient looks directly forwards (towards the ceiling). The scale reading is noted and converted into millimetres of mercury pressure, from a conversion table or graph. A reading of 18—20 mm. Hg. pressure is considered normal

Two types of the above tonometer are available. In one, readings are taken after addition of weights to the plunger. The weights used are 5.5 G., 7.5 G., 10 G., and 15 G. The other type is the weightless model and is easy to use.

6. Everting the upper lid

This is a very essential procedure for diagnostic and therapeutic purposes. The patient is instructed to look

down and the examiner grasps the lashes of the upper lid with his thumb and forefinger and draws the lid down a little, the thumb of his other hand is insinuated into the sulcus between the globe and the orbital rim. With a quick movement of the finger and thumb grasping the lashes the lid is folded over the opposite thumb. A glass rod may be substituted for the second thumb.

7. Inspection of the cornea

The cornea is a highly polished transparent structure. If there be any area where the corneal epithelium is lost the polish will be lost over it. This is easily detectable by allowing a light source to play on the cornea while the patient is asked to look in varying directions. A regular image of the light source (may be a window) is to be expected when the cornea is intact. Such will not be the case where the corneal surface is irregular—the latter being due to oedema, abrasion, erosion or to the presence of a foreign body.

A second method of detecting loss of corneal epithelium as in abrasion or ulceration is by *staining* the cornea with fluorescin. A drop of 2 percent fluorescin is instilled into the affected eye, the excess wiped out and the eye inspected. A greenish stain is shown by an abrasion or ulceration, the stained area corresponding exactly in size and shape to the area denuded of epithelium. Thus it will be noted that this is an invaluable aid in delineating the extent of an ulcer prior to cauterization. The method is also useful in assessing the progress of the ulcer with treatment for as the ulcer heals the staining diminishes.

On occasions one is confronted by a situation where the digital retraction of lids is rendered difficult or impossible either owing to the tightness of the lids (oedema, emphysema, growth) or owing to age as in the case of an

infant or child. Here one has to employ instrumental *retraction*. Desmarre's lid retractor is the most convenient one to use. It is introduced under the upper lid and the lid drawn upwards. The most unruly child's cornea could be inspected in this manner. Even when a child is cooperative certain lesions of the eyes or lids cause spastic eversion of the lids on attempted retraction. The above method is applicable here too.

CAUTION

In attempting to open the lids of an eye afflicted with mucopurulent or purulent ophthalmia great care should be exercised by the medical attendant in protecting his or her eyes from the pent up discharge squiriting into their own. Gentle separation of the lids without exerting undue pressure on the lids will achieve this or the examiner should wear protective goggles at the time of inspection of such cases.

8. Testing for Corneal Sensation

In the investigation of many ocular as well as neurological disorders the testing of corneal sensation is an important step. Of the former group may be mentioned ophthalmic herpes and of the latter cerebello-pontine angle tumour may be cited. The simplest method for assessing the corneal sensation is by means of a wisp of cotton wool. The lids are kept separated by forefinger and thumb of one hand while the cornea is gently stroked with a wisp of cotton wool held in the other.

9. Testing for Lacrimal Secretion

In certain pathological conditions of the eye and in general disorders there is diminished secretion of tears. In order to prevent catastrophic complication such as keratitis from dryness it is necessary to assess the state of lacrimal secretion. This is estimated by the method

of Schirmer. Two strips of filter paper 3 cm. long by $\frac{1}{2}$ cm. wide are allowed to hang over the lower fornices by folding 2 mm. of one end of the strips of paper. Care is taken to avoid touching the cornea while inserting the strips into the fornices. At the end of 5 minutes at least 15 mm. of the strips should be moist with tears if the secretion of tears is normal. Less than this is indicative of undersecretion. Comparing the two sides gives a good estimate of the defective gland. Such estimates are of value in kerato-conjunctivitis sicca, in threatened neuro-paralytic keratitis and facial palsy.

10. II Functional : Under this heading are included :—

i. visual acuity
ii. visual field
iii. colour sense

(i) The visual acuity is tested by means of test types recommended by Snellen. Distance vision is expressed as a fraction, the numerator being the distance in metres of the subject from the test types while the denominator is the distance in metres at which a normal person is able to read the test types in question. Snellen's types are so constructed as to subtend 5 minutes of arc at the nodal point of the eye. Each stroke of a type subtends 1 minute of arc. Near vision is recorded by means of test types based on similar calculation (Snellen's) or by means of the ordinary printer's founts (Jaeger's). Recently the Faculty of Ophthalmologists of Great Britain has recommended the use of certain grades of type the smallest of which is N_5 .

The standard distance at which the subject is placed is 6 metres away from the test types and a person possessing normal visual acuity will be denoted by the fraction 6/6.

2845 DENOMINATOR - IN METRES
NORMAL PERSON - KMPH

$\frac{6}{60}$

$\frac{6}{36}$

$\frac{6}{24}$

$\frac{6}{18}$

$\frac{6}{12}$

$\frac{6}{9}$

$\frac{6}{6}$

$\frac{6}{5}$

When vision is blunted to a great extent a person may only see hand movements which is denoted by the letters HM. Some may only perceive light, which is denoted by the letters PL.

(ii) Field of vision estimations are made in one of three ways. The *confrontation test* is simple and excludes gross defects. The patient stands or sits on a level with the examiner, the former shuts his right eye while the examiner shuts his left eye. The examiner approximates a target such as a ladies' hat pin from the periphery towards the centre of the field comparing his own field with that of the subject. This method is useful not only to spot peripheral defects but also to identify central or caeco-central defects. This process is repeated in all quadrants and for the companion (left) eye of the patient, the examiner occluding his right eye.

The second method is useful in cases of patients with severe restriction of vision such as in cataract. In this the *projection of light* is estimated by shining a spot of light on to one eye of the patient from the four principal quadrants of gaze and asking the patient to point out or indicate the direction from which the light is shining. The companion eye whether affected or not is kept covered during the test. The second eye may be similarly examined if it is deemed necessary. It is necessary to instruct the patient not to turn his eyes while the test is being conducted. This test is also applicable in cases of dense leucoma awaiting keratoplasty.

The third method is *perimetry*. Lister self-registering perimeter is satisfactory. In examining a patient on the perimeter the following practical points should be noted :—

1. The eye under examination should be directly opposite and in a level with the fixation spot.

2. The other eye must be fully occluded, the occluder or patch should not project forward more than is necessary.

3. The examiner should watch the patient's eye carefully and instruct him that the eye should not wander from the fixation spot.

4. The target is moved slowly from the periphery into the centre, the patient being instructed to tap on the table as soon as the object is discerned in the periphery.

An elaboration of perimetry is the detailed examination of the central field of vision on a flat screen—CAMPIMETRY. The subject is seated at a distance of 1 or 2 metres according to the size of screen used, 1 metre square in the former case and 2 metres square in the latter case. Targets mounted on a special holder are moved over the screen in various meridia, these as well as the isopters being marked on the black screen with black thread. The test objects range in size from 1 to 60 mm. in white, red and green. It would be seen that a flat screen of 2 metre square provides sufficient amplification of small defects hardly detectable with the perimeter. Bjerrum's name is associated with the screen for it was he who first popularised this method of examination.

(iii) Estimation of colour sense is important in certain affections of the eye and also in the selection and employment of personnel in certain transport systems and in industry.

Defects of colour vision may be congenital or acquired.

Methods of testing for colour sense

1. Edridge-Green's Lantern Test
2. Ishihara's plates
3. Holmgren's wool test

None of the above tests is infallible and to fail a prospective candidate on the basis of any one test is being unduly harsh. *It is therefore imperative that any candidate who appears to have an unstable colour sense should be subjected to at least three colour vision tests* before he is condemned. It is common knowledge that some subjects classed as red-green blind on the Ishihara's plates are able to name all spectral colours correctly. On the Ishihara plates the individual spots are recognized as different and correctly named.

The above tests are applicable to congenital colour blindness. Acquired defects of colour vision are met with in toxic amblyopia, retrobulbar neuritis and optic atrophy. As these defects preferentially occur due to the affection of certain nerve fibres in the retina and optic nerve the colour defects are patchy and have to be elicited by colour field estimation. Such patchy defects are called scotomata—meaning blind areas. Scotomata may be charted by help of the perimeter or by means of the simple confrontation method.

11. III Dark Room Examination:

Once the external and functional examinations have been conducted it may be necessary to examine a patient in the dark room to confirm certain findings, to study the refraction or to gain a view of the ocular fundi. The following is a systematic way of carrying out the dark room examination:

(a) Oblique illumination
(b) Retinoscopy
(c) Ophthalmoscopy
(d) Transillumination

(a) *Oblique Illumination* with a condensing lens will, in addition to enhancing what has already been seen in daylight, reveal gross lesions of the cornea, iris and lens. It is advantageous to employ magnification with a loupe to see details of pathological changes in the structures referred to above.

It should be remembered that the condensing lens has a focal length of 7 cm. and so the source of illumination should not be closer than this to the eye being examined. The examiner should place his eye as close as possible to the loupe. Any retraction of the patient's lid may be carried out with the middle finger of the hand that holds the loupe. The other hand (holding the lens) may help in the retraction of the second lid, usually the lower if such retraction is necessary. The condensing lens as well as the loupe should be steadily moved forwards as the plane of examination extends deeper from the cornea to the lens.

By this method the polish of the corneal surface, oedema, erosions or vascularisation of the cornea, keratic precipitates (K. P.), exudates in the anterior chamber, blood vessels or haemorrhages on the iris fine tremors of the iris, nodules and any alteration in the pattern of the iris may be made out. Lenticular changes include opacities, tremulousness and dislocation. In the last mentioned the edge of the lens is clearly visible within the pupillary aperture. Careful inspection of the space between the lens edge and the pupillary margin will show a herniation of the vitreous the summit of which is stippled with fine uveal pigment.

With pupillary dilatation achieved by the use of 2 percent homatropine and cocaine further details may be made out. Posterior synechiae may stand out more prominently and the pupillary aperture may present a crenated appearance. A subluxated lens may be more easily seen. It is worth learning to search for Purkinje's images, three of which are bright and easily placed. One is due to the reflection of the light source on the cornea, the second and third due to reflections on the anterior and posterior lens surfaces. The importance of this sign is in the differentiation of a black cataract from a total vitreous haemorrhage in which the pupil is black (on distant ophthalmoscopy). In the former only two Purkinje's images are seen whereas in the latter all three images are seen. In a recent vitreous haemorrhage, however, the pupillary area may appear red due to the blood showing up from behind the lens.

(b) *Retinoscopy* is the method commonly used in clinical practice for the estimation of the state of refraction of the eye. A plane mirror with a central perforation is used with which a beam of light is shone into the eye under examination after securing pupillary dilatation with homatropine drops. The mirror is tilted up and down and from side to side observing what happens to the illuminated fundus glow through the dilated pupil. A movement of this illuminated spot (which has a dark edge due to the shadow cast on the fundus by the pupillary edge) with the mirror indicates emmetropia, hypermetropia or myopia of less than 1.5 dioptres. A movement against the mirror is indicative of myopia. Once an idea of the probable nature of the refractive state is gained lenses are placed before the eye till the movement of the spot of light fringed by its dark edge is reversed. In the case of the 'with movement' plus lenses are slowly

added and a note of the lens that causes a reversal is kept; once one meridian is neutralised the other at right angles to it is next dealt with in the above manner. In cases of 'against movement' minus lenses are added till the point of reversal is reached. In all cases where a mydriatic has been employed it is usual to subtract algebraically 1. 5 dioptres from the figure arrived at by refraction. Thus a plus 1. 5 means emmetropia, a plus 0.75 means a myopia of −0.75 and a plus 5 will indicate a hypermetropia of +3.5.

It is hardly necessary to stress the fact that the art of refraction. is enriched by practice and that after several years of assiduous work under a chief. It cannot be mastered from any book.

It is to be noted that retinoscopy is an important step in the routine examination of an eye for what at first looks to be a puzzling case of visual loss turns out to be a case of high myopia or gross astigmatism. With a mere tilt of the mirror one may gain much information in an early part of the examination rather than proceed with more complicated examinations and still remain puzzled.

(c) *Ophthalmoscopy.* There are three methods of ophthalmoscopy viz., distant direct, indirect and direct. In *distant direct* the plane mirror of the non-luminous ophthalmoscope is used. The light source is behind and above the patient's head. The details to be noted are the nature of the red reflex and any opacities of the media. The red reflex should be uniform in all directions of gaze but in cases of retinal detachment the red glow gives way to a grey reflex. Opacities in the media appear dark owing to the interception of the light returning from the fundus by the opacities. The location of foreign bodies in the media is possible by this method. A foreign body on the


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cornea will move in the same direction as movement of the eye on the patient being instructed to look up. A foreign body or opacity in the lens surface will remain stationary on similar excursion of the eye, whereas an opacity on the posterior surface of the lens or in the vitreous will move in a direction opposite to that of the excursion of the eye. In other words all opacities in the media in a plane anterior to that of the pupil will move with, and those posterior to the pupillary plane against, the movement of the eye. Those in the pupillary area remain stationary. Vitreous opacities are further characterized by motility independent of sustained motion of the eye. A lens opacity, for instance, moves as the eye is moved to and fro but is brought to a halt at the moment the eye stops moving. In the case of a vitreous opacity stoppage of ocular movement does not halt the motion of the former; this is due to the momentum imparted to the opacity floating in a semisolid vitreous by the ocular rotation.

In *indirect ophthalmoscopy* the plane mirror is replaced by a concave mirror and a condensing lens is interposed between the patient's eye and the examiner. The image is inverted and is seen between the lens and the observer's eye. *The magnification obtained by this method is five diameters.* It is very important to proceed systematically in conducting this examination. The optic disc is an easy landmark for the beginner; to get a view of the disc, if the right eye is being examined the patient is instructed to look towards the examiner's right little finger which he keeps stretched out while the same hand is engaged in holding the ophthalmoscope against his right eye. In examining the patient's left eye the patient is requested to look towards the examiner's left ear. In this manner a view of the

disc is first obtained. Next by moving the condensing lens to and fro and up and down areas of the fundus adjoining the disc may be seen. The area of the fundus next in importance is the macula lutea. As the examination proceeds the patient is instructed to look into the examiner's light whereupon the macula is seen as a tiny red area with a speck of light in its centre. The speck of light is the reflection of the examiner's source of light reflected by the little concave mirror that the macula really is. A general survey of the fundus should be made including a study of the vascular tree, any gross abnormalities such as a detached retina, coloboma of the choroid, crescents round the disc or large sheets of haemorrhage. This method of examining the fundus especially the extreme periphery is important particularly in retinal detachment surgery for it is essential not merely to diagnose a retinal detachment but also to locate the tear or hole in the retina.

In *direct ophthalmoscopy* an electric ophthalmoscope worked by a battery or adapted to the mains is substituted for the non-luminous ophthalmoscope. *A magnification of nearly 14 diameters is available.* The image is erect and owing to the magnification only small areas of the fundus are visible at any time. The same systematic approach is required in examining the fundus as with the indirect method. As mentioned earlier the extreme periphery of the fundus requires a thorough search for pathological lesions and particularly for tears. Besides maximum pupillary dilatation, denting the sclera with a muscle hook (or special fountain pen clip-like one slipped on to the little finger) greatly facilitates such examination. The most important practical application of direct ophthalmoscopy is the detection of medial opacities from cornea to vitreous and elevations or depressions in

the fundus or optic disc. In order to include these in the routine of examination it is best to start viewing the fundus with no lens at the peep hole of the ophthalmoscope and rotate the battery of lenses so as to place increasing strengths of plus lenses at the peep hole. Thus a tumour, cyst or detachment of the retina will be made more prominent; so also with a + 6 D lens the vitreous will be under scrutiny a + 8 D will enable inspection of the lens and a + 10 or +12 D will throw the cornea into focus. If the optic disc looks hollowed out (cupped) it is necessary to turn the lenses in the opposite direction till minus lenses appear at the peep hole. The measurement of disc swelling is of importance both from the diagnostic and therapeutic points of view. Thus arbitrarily it is held that a disc swelling of 2 D and under is due to papillitis and any swelling above this amount is termed papilloedema. More important still is the fact that if the papilloedema is over 6 D a decompression operation on the skull is urgent, whatever the cause of the papilloedema. Any refractive error of the patient must be corrected by the appropriate lens at the peep hole of the ophthalmoscope.

(d) *Transillumination* of the globe is at times useful in as much as it gives information regarding the transparency of the coats of the eyeball. The presence of new growths may be revealed by the absence of a glow through the pupil when the transilluminator is applied to a certain part of the globe.

12. Biomicroscopy is the elaboration of the simple clinical method of examining an eye with oblique illumination and the loupe. Here the illuminating system is in the form of a slit beam and the observing system is a binocular microscope. There is very little information that can be added to that already gained by the loupe exami-

nation but owing to the magnification afforded by the biomicroscope greater degree of detailed examination is possible. The illuminating system also provides an optical section of the cornea and lens and with additional appliances the vitreous in its posterior third and retinal lesions may also be studied in section. In an early case of iritis the diagnosis may be clinched by the use of the biomicroscope. A narrow cone of light is used for the examination. In normal cases the cornea and the lens stand out in relief leaving an optically empty zone—the aqueous in the anterior chamber. In early iritis before posterior synechiae begin to form the increased cellular content of the aqueous picks up the beam of light and a 'flare' is visible. This phenomenon is simply compared to particles of dust in a darkened room being lighted by sunlight streaming through a narrow chink in a window. This sign is sometimes referred to as the Tyndall phenomenon.

Two highly specialised applications of biomicroscopy are slit lamp funduscopy and gonioscopy.

13. **Slit-lamp Funduscopy** is carried out by the insertion of a special fundus viewing contact lens under the lids of the eye to be examined and directing the slit beam through the centre of the pupil. The observer views the fundus through the binocular microscope. The contact lens of a power of − 55 D neutralises the corneal refraction and permits the light as well as the observer's view to reach the posterior vitreous and the fundus. The points to be noted with this method of examination are the presence of a 'flare' round the nerve head for such a sign is indicative of optic neuritis (as distinct from oedema), the differentiation of a retinal hole from a thin walled cyst—the latter will show an anterior wall by the

narrow beam and displacements of the vitreous. The last two are of great importance in retinal detachment both as to its aetiology and treatment.

14. Gonioscopy means viewing the angle of the anterior chamber. It will be recalled that normally the bottom (or the extreme periphery) of the angle of the anterior chamber is not visible to the naked eye owing to the overlapping of the scleral fibres at the corneo-scleral junction. A special contact lens with a reflecting mirror incorporated in it is placed under the lids of the eye to be examined, the angle of the anterior chamber being viewed with the naked eye or with the biomicroscope. The study of the angle of the chamber has assumed great importance during the past decade owing to the role played by certain changes in and around the angle in the aetiology, classification, treatment and prognosis of glaucoma.

15. Note

In all instances where examination in the dark room has entailed the use of mydriatics to dilate the pupils it is incumbent on the examiner either to instil eserine drops into the patient's eyes or to see that such treatment is carried out by a responsible person. *The danger of inducing an attack of closed-angle glaucoma in susceptible persons by inattention to this important precautionary measure cannot be over emphasised.*

SOME SPECIAL INVESTIGATIONS

16. Provocative Tests for Glaucoma.

To await the classical triad of raised tension, field loss and cupping of the disc for the diagnosis of glaucoma is to adopt a passive attitude. As is well known treatment of glaucoma must be early and such treatment

only aims at halting the disease and does nothing to restore lost vision. Hence the importance of early diagnosis should be appreciated. Provocative tests are designed to bring on an elevation of intra-ocular pressure by subjecting an eye to duress such as may be operating in the actual causation of glaucoma.

It must be mentioned at the outset that none of the tests to be described is infallible. Excepting in research institutions it is not possible to carry out all the tests in any given case for they are time consuming and most inconvenient to the patient.

17. Lability Test

A sphygmomanometer cuff is applied to the neck of the patient and tonometry is performed. The cuff is inflated to 45 mm. Hg while one hand of the patient is dipped in ice water. Tonometry is performed after a minute and the cuff deflated. An increase of tension of 9—11 mm. Hg may be considered pathological.

18. Caffeine Test

After tonometry the patient drinks a cup of black coffee (45 g) or pure caffeine (0.4 g). Tensions are recorded at 15, 30, 45 and 60 minutes. A rise of tension of 9 mm. Hg is pathological.

19. Water-Drinking Test

Tonometry is followed by imbibition of 1000 ml. water. Tonometry is performed at 15, 30, 45 and 60 minutes. A rise of 8—9 mm. Hg pressure may be considered pathological.

20. Homatropine Test

With the patient in a recumbent position the tension is recorded and a drop of 1 percent homatropine is ins-

tilled and tonometry is repeated at 30, 60, 90, 120 minutes. *It is important to instil strong miotics into the eye till the pupil contracts.* If the tension shoots up to a high level intensive miotic therapy should be instituted (see section on glaucoma). An increase of tension by 8—11 mm. Hg may be considerd pathological.

21. Dark Room Test

After tonometry the patient is allowed to remain in the dark for 40—60 minutes. At the end of this period in subdued light the tension is again recorded and a rise of 6 mm. Hg is considered pathological.

22. Priscol Test

After tonometry 1 ml. priscol solution is injected subconjunctivally after topical anaesthesia. Tonometry is performed at 15, 30, 60, 90 minutes. A rise of 11—14 mm. Hg is considered pathological.

23. Rationale of Provocative Tests

Present knowledge permits us to give only a hypothetical explanation and that too only in the case of the water drinking and homatropine tests. In the former there is osmotic hypotony of the blood serum. From this it is to be inferred that for a short period there is increased flow of water into the eye. In the homatropine test there is thought to be collection of aqueous within the eye owing to hampered drainage. This is in all probability due to paralysis of accommodation which results in narrowing of the trabecular spaces and Schlemm's canal, opening of the ciliary arteries and narrowing of the veins. It has been found that this test was positive in cases where the chamber-angle remains open.

One poignant criticism of these tests is that we are dealing with one aspect only of glaucoma—ocular hypertension which by the way is not the be-all and end-all of glaucoma.

SYMPTOMS AND SIGNS OF OCULAR DISEASE

Symptoms

As is to be expected symptoms of ocular disease will centre round the visual function. Apart from these there is the inevitable concomitant of disease elsewhere—pain —in many ocular disorders.

24. Pain in and around the eye may be due to ocular disease or referred from some structure lying close to the eye. *It is axiomatic that pain due to ocular disease is always accompanied by redness of the eye, the only exception being retrobulbar neuritis.* Pain due to diseases of the eye may sometimes be felt over the vertex and the neck; this distribution is due to the ophthalmic division of the trigeminal nerve supplying the scalp up to the vertex and to the close segmental association of the spinal root of the trigeminal with the cervical plexus of nerves. Pain referred from disease of the adjacent nasal sinuses or teeth is easily excluded from the history and from any obvious signs that may be present such as tenderness over the frontal sinus, rhinorrhoea or a carious tooth. Some of the common conditions that give pain in the eye are foreign bodies on the cornea, corneal ulcers, iritis, glaucoma, keratitis and episcleritis.

25. Redness of the eye may be due to accumulation of blood under the conjunctiva as in fracture of the base of the skull, in localized injuries to the eye, or whooping cough. It may also be due to inflammatory reactions of the eye be they infective or allergic. A red eye usually

excites the sufferer and there is much justification for this for often it may be an acute emergency. In the first category there is a large blotch of blood under the conjunctiva and unless there is pain from other causes these eyes are painless. In the second category come painful eyes. The differential diagnosis of diseases in the latter group is of great importance and the following table summarises the chief features.

26. Table I — Differential diagnosis — Red Eye

	DISEASE		
Features	*Conjunctivitis*	*iritis*	*Glaucoma*
Discharge	plenty	nil	nil
Vision	unaffected	affected	affected
Pain	nil or slight	severe	severe
Injection	conjuntival	ciliary	ciliary
Cornea	clear	k. p.	steamy
Pupil	normal	small	dilated
Tension	normal	normal	raised
General	nil	nil	vomiting, prostration

27. Specks before the eye sometimes stationary and at others mobile may be the chief complaint of a patient. In the condition named *muscae volitantes* patients are in the habit of producing elaborate drawings of what they see before their eyes. These may range from tiny specks to threads rather like mycelia of fungi and float about and are best seen against an evenly illuminated surface. These are due to the persistence of embryonic vitreous cells and are not pathological. The sudden appearance or increase of such opacities should, however, be treated seriously for they may forebode a retinal detachment. Ophthalmoscopically there are no opacities to be seen.

Stationary opacities are usually lental. Ophthalmoscopically visible opacities may occur in the vitreous but these are mobile.

28. **Flashes of Light** or photopsiae are occasionally complained of by patients and are due to retinal irritability. The latter may be due to trauma as in a blow to the eye (the well known description of seeing stars) or more commonly to a less dramatic form of trauma as friction of a semisolid vitreous against the retina. Such a situation exists in senile eyes, in myopia with vitreous degeneration or in vitreous displacements. As such, photopsiae are sometimes precursors to a retinal detachment. Flashes of light are an indication of retinal irritability as in inflammations of the eye especially when they cause shrinkage of the eye. They also occur as halucinations occasionally. A common malady in which special types of flashes occur is migraine. Whatever their origin *photopsiae* as a complaint *must be investigated at the first opportunity with a view to excluding retinal detachment.*

29. **A Watering eye** may be due to over-production of tears or defective drainage. The former is due to such causes as foreign bodies on the cornea, conjunctiva or under the upper lid. Ulcers and inflammations of the cornea likewise cause excessive lacrimation. Defective drainage may arise from blockage of the puncta, canaliculi, sac or nasolacrimal duct. In some instances defective drainage is determined by certain mechanical factors as malposition of the puncta as in entropion or ectropion of the lower lid. One cause of epiphora (overflow of tears) that may appear elusive is a new growth of the maxilla which causes pressure on the nasolcrimal duct.

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30. **Dry eye** is a complaint of elderly persons more commonly in females and is typically met with in the syndrome of kerato-conjuctivitis sicca (Sjogren's syndrome). In this condition there is a generalised dryness of the body including the eye, bronchi, vagina and skin together with hyposecretion of the gastric glands, a secondary anaemia and a Plummer-Vinson syndrome. Apart from this some skin affections like pemphigus cause dryness by strangling the ducts of the lacrimal gland. In a similar way trachoma causes a dry eye. Removal of the lacrimal gland does not cause drying of the conjunctiva because of the presence of accessory lacrimal and conjunctival mucus glands.

Note :

The state of lacrimal secretion may be determined by Schirmer's test (§ 9).

31. **Sticky lids** are a feature of conjunctivitis, the stickiness ranging from a very mild degree to actual gluing of the lashes according to the nature and quantity of discharge.

32. **Crusts** on the lids are a sign of blepharitis.

33. **Loss of lashes** is met with in any condition in which the lash follicle is damaged as in blepharitis, trachoma and severe scarring of the lid.

34. **Feeling of grit, sand or foreign body** is a common complaint and apart from being due to actual foreign bodies it is a symptom of acute catarrhal conjunctivitis, keratitis and episcleritis. Vesicles on the cornea due to herpes may also account for these symptoms.

35. Haloes are produced by oedema of the cornea and particulate matter settling on the cornea e. g., atebrin dust. Mucus and tears (or even rain drops on the cornea) also produce haloes. Lenticular opacities or thickening can produce haloes. Those due to tears, water or mucus disappear on wiping the eye. The differential diagnosis between lenticular and glaucoma haloes (due to corneal oedema) is very important. Glaucoma haloes come on at times especially when an attack of glaucoma is imminent as when a person has been in the dark for a time. Such haloes disappear with the use of miotics such as pilocarpine nitrate 1%. Lenticular haloes will persist throughout. There is a difference in size between the two types of haloes. Glaucoma haloes are larger than lenticular; at a distance of 10 feet from a wall a glaucoma subject will project a halo that measures 15—25 inches. *A pathological halo is not synonymous with glaucoma but is an expression of corneal oedema.*

36. Blurring of vision is a frequent complaint and may range from the slightest *qualitative* degree to a quantitatively detectable amount. Such blurring may be due to accommodative defects, muscular anomalies, iridoplegia, cycloplegia, or medial opacities or retinal disturbances such as oedema, detachment and vasospasm. Optic and retrobulbar neuritis are also responsible for blurred vision. Certain features may help in the elucidation of the causative factor. Jumbling of print for instance is a point in favour of muscular imbalance, clear vision giving place to blurred vision after a few moments is indicative of ill-sustained accommodation, dim vision in bright light improving in poor illumination is met with in central lens opacities and a central blackout denotes an affection of the nerve, commonly a retrobulbar neuritis. Sudden blurring is usually connected with a vascular crisis, retinal or cerebral, retinal detachment and

glaucoma. In retinal vascular disturbances the loss is unilateral but in cerebral events one field of vision is lost. Intermittent or transient blurring may be physiological as with change of posture or pathological as in hypertension, eclampsia or nephritis. Malingering and hysteria should be kept in mind.

37. Double vision is a not too infrequent symptom and may be noticed by a patient while reading or while at games such as tennis and may be only in certain directions of gaze. Uniocular diplopia is met with in medial opacities such as corneal and lental opacities in multiple pupils (polycoria), in iridodialysis and in high astimatic defects. Binocular diplopia is due to any cause that dislocates the normal equipoise of the ocular muscles. Thus it is a feature of heterophoria as much as it is that of heterotropia (squint). Paralytic squints of recent origin usually exhibit double vision especially in adults in whom supression is not the rule.

It may transpire in the history of a case that double vision is transient or that it increases with fatigue, examples of the former being tabes dorsalis and disseminated sclerosis and of the latter myasthenia gravis. On occasions double vision may come on quite dramatically and associated with general symptomatology. Diplopia with food poisoning is known in botulism, with delirium in atropine poisoning and with excruciating pain in the head in intracranial aneurysm. A lumbar puncture is sometimes complicated by double vision and is due to involvement of the sixth cranial nerve as part of the conus syndrome; owing to descent of the brain stem the sixth nerve (with the longest intracranial course) gets kinked against the petro-sphenoidal ligament.

38. **Day Blindness** apart from being a congenital anomaly is met with in certain well defined eye conditions. Corneal inflammations produce this symptom by virtue of the severe photophobia they induce in daylight. Central lental opacities, macular lesions and retrobulbar neuritis are characterized by day blindness owing to the constriction of the pupil in the first condition, the lental opacity then cutting off the light entering the eye; in macular lesions central vision (day vision) is naturally curtailed and because the papillo-macular bundle is often affected in most cases of retrobulbar neuritis day blindness results from a similar cause. In congenital day blindness there is total colour blindness and the affected children feel comfortable in dim illumination or by night though their visual acuity is not increased by such surroundings.

39. **Night Blindness** is synonymous with vitamin A deficiency. The deficiency may be due to defective absorption, improper assimilation, too hurried elimination of bowel contents. Despite good storage of vitamin A the retina may not be able to make use of it owing to disease processes. Thus it will be seen that night blindness occurs in diverse conditions some of which have nothing to do with the eye. As examples may be cited local ocular diseases such a retinitis pigmentosa, choroiditis, myopia; cirrhosis of the liver, coeliac disease and malnutrition account for this symptom in some general affections. In connection with vitamin A deficiency it is now necessary to modify our views for it has been found that vitamin B complex or at least one of its components —a derivative of nicotinic acid—is an important catalyst in the vitamin A resynthesis. This is an enzyme diphosphopyridineucleotide (DPN). It would appear therefore that in deranged vitamin A metabolism or deficiency both A and B vitamins should be administered.

40. Signs of ocular disease are as varied as in other parts of the body subject to disease. Owing to the white background provided by the sclera discoloration due to affections of the eye in general and of the sclera in particular is easily noticeable. The transparency of the media of the eye permits certain observations to be made; some of these may be made with simple devices while others have to be searched for with special equipment such as the biomicroscope, gonioscope and ophthalmoscope. The more important and also the more easily demonstrable signs will be dealt with below.

41. Swelling of the lids may be due to cellulitis, surgical emphysema, tumours or tarsitis. Regional swelling as in the lateral portion may be due to lacrimal adenitis. The character of the swelling may give a clue to diagnosis as for instance the bag of worms feel in neurofibroma. Indurated painful swellings along the lash border of the lids are hordeola externa (stye). Rounded sessile swellings with a yellow-looking circumscribed area on eversion of the lids are meibomian cysts. Swellings in close proximity to the puncta and canaliculi probably indicate canaliculitis and on occasions lacrimal concretions. Hordeola in the two corners of the eye produce oedema and pain out of proportion to the size of the offending lesions.

42. Discoloration of the lids is commonly seen in ecchymosis. Naevi of the capillary variety will indicate the presence of angiomatous malformations. In children ecchymoses may be seen in whooping cough and rarely in neuroblastoma of the adrenals with orbital deposits. Depigmentation (vitiligo) is a sign of herpetic affections of the lids.

43. Scales along the lashes is a sign of blepharitis and may be associated with dandruff. What may look like scales

at first sight on closer inspection may turn out to be parasites and their nits—the usual one is the body louse. The nits are small, brownish and stick out at an angle to the lashes and are tenacious.

44. Loss of lashes (madarosis) is a sign of damage to the lash follicles as in blepharitis, trachoma and cicatricial conditions. *Irregular lashes* are met with in the above named conditions as well as in some skin diseases that affect the lids. In short any affection of the lids that causes scarring will cause the lashes to stick out irregularly a common anomaly being incurving lashes, *trichiasis*. Such lashes produce much irritation of the cornea, watering and may on occasions give rise to corneal ulceration. *Double row of lashes* is a rare finding in some persons in whom the second row may cause friction on the cornea.

45. A swelling at the inner canthus may be due to a dacryocystitis or mucocoele of the sac. More rarely it may be a tumour of the sac. *Lacrimal sac swellings are situated below the level of the medial palberal ligament.* Swellings above this ligament belong to the ethmoidal sinus.

46. Drooping of the upper lid (ptosis) is a common enough sign to warrant description. It may not only indicate local disease such as the mechanical ptosis of trachoma (due to tarsitis) or that caused by a fracture of the upper orbital margin but also it may be a sign of neurological disorders. Myasthenia gravis is one disease in which ptosis is common. Lesions of the third nerve from the base of the brain down to its ramification in the muscle may cause ptosis. Some examples of such lesions are tumours of the midbrain, aneuryms of the Circle of Willis, and the internal carotid (intra-cranial part). Ptosis is also met with as a congenital anomaly

in which case it may be associated with superior rectus palsy on the affected side.

47. **Improper closure of the lids** (lagophthalmos) is best exemplified by facial palsy and all conditions where scarring of the lids prevents their closure. *Colobomata* of the lids, some congenital and others cicatricial, also cause improper closure of the lids but from a different cause.

48. The everted lids may show *follicles*, *granules* meibomian cysts and *concretions*. A scarring of a characteristic nature known as *Arlt's line* (see later under trachoma) is pathognomonic of trachoma. Granulomatous excrescences point to rhinosporidiosis—a fairly common condition in this country. *Membranes*—false and true—may be met with in various types of conjunctival infections. True membranes are seen in diphtheria and false ones in streptococcal conjunctivitis.

49. Vascular changes in the conjuntiva are a good guide to certain ocular and at times general diseases. *Varices* of the conjunctiva are suggestive of diabetes mellitus. Congestion of the eyes may be seen in alcoholics or even in normal persons. Congestion of a certain pattern has special significance in many ocular diseases. Reference has already been made to ciliary and conjunctival injections. It is only necessary here to stress the importance of the two types of injection. *Ciliary injection* is seen in corneal inflammations and injuries, iritis, iridocyclitis and glaucoma. *Conjunctival injection* is a feature of conjunctival inflammation. At times especially in prolonged states of congestion a *caput medusae conjunctivae* appears as is met with in chronic glaucoma, tumours of the ciliary body or choroid and aneurysms of the orbit and cavernous sinus.

50. Pannus, a vascular invasion of the superficial layers of the cornea is another characteristic feature of certain corneal and conjunctival diseases. Although pannus is seen in phlyctenular keratitis, leprosy and in degenerative states of the eye its greatest importance and significance is in trachoma, so much so that the word pannus is synonymous with trachoma. The pannus of trachoma invades the cornea from above. In interstitial keratitis (syphilitic) the vascularisation is deep seated.

51. Vascularisation of the cornea may be superficial or deep. One type of superficial vasularisation has already been dealt with (§ 50). In the superficial type the blood vessels may be continued on to the conjunctiva from the corneal surface. In the deep type the vessels are deeper seated and on following them to the limbus they suddenly disappear into the depths of the episclera. The deep type is characteristic of interstitial keratitis whereas superficial vessels indicate other types of keratitis. In phlyctenular keratitis sometimes a leash of vessels is seen to run onto the cornea at the head of which is an ulcer—fascicular ulcer (*fasciculus* = bundle). The significance of vascularisation is thought to be a metabolic demand for oxygen. Thus in states of inflammation where necrotic products and metabolites accumulate the ordinary oxygen carriage system in the cornea is ill-adapted for the task of supplying all the necessary oxygen. In ariboflavinosis too corneal vascularisation has been described which many authorities think has been exaggerated.

52. Size of the cornea varies in individuals but in certain well defined clinical entities it may be large—*megalocornea*, an inherited anomaly of males complicated by cataract—or it may be small. Where the cornea alone is small the term microcornea is applied but where the smallness of the cornea is in proportion to the whole

globe the term nanophthalmos is applied. Microphthalmos is often complicated by several anomalies of the whole globe, one common association being cataract.

53. Shape of the cornea is a feature worth taking note of, for altered shapes can cause visual disturbances due to the associated gross astigmatism. Cone shaped cornea or keratoconus is a dystrophic affection accompanied by an irregular myopic astigmatism. The condition is best spotted by viewing the cornea in profile and by the use of Placido's disc which has a series of concentric black and white alternating rings with a central perforation. The disc is held with the rings facing the patient's cornea and the observer peeps through the central perforation. In a normally curved cornea the rings will present a regular pattern on the patient's cornea but in keratoconus the rings are distorted. So will they appear in badly scarred corneae or in gross astigmatism.

54. Rings on the cornea may be white or coloured. The former is typically seen in arcus senilis and is caused by a deposit of fat at the margins of the cornea. There is a clear interval of about ½—1 mm. between the limbus and the arcus. It begins at the upper or lower limbus and slowly encircles the whole cornea. It never spreads to the central zones of the cornea and so does not interfere with vision. It is to be distinguished from a ring shaped opacity due to sclerosing keratitis where there is no clear interval between the limbus and the opacity.

Coloured rings are seen in hepatolenticular degeneration and in copper intra-ocular foreign body. The names of Kayser and Fleischer are associated with these rings which are best seen with the biomicroscope.

55. Anaesthesia of the cornea is met in several ocular affections such as herpes simplex and zoster of the cor-

nea, corneal dystrophies and after alcohol injection of the trigeminal ganglion. At times it may be the first sign of a tumour in the cerebello-pontine angle. One of the most important complications following corneal anaesthesia is neuro-paralytic keratitis.

56. **Striate folds** (keratitis) are seen in states in which there has been a sudden release of the intra-ocular tension as after cataract extraction. They may also be seen in eyes in which hypotony follows a period of hypertension such as is met with in uveal inflammations. The folds are produced by rucking of Descemet's membrane.

57. **Keratic Precipitates (K. P.)** are deposits of white cells on the back of the cornea in an area classica situated in a triangular segment between 5 and 7 o'clock positions, seen typically in cases of iridocyclitis. In tuberculous iridocyclitis the K. P. are larger earning the name mutton fat k. p. and are more universally scattered over the back of the cornea.

58. **Hypopyon** or pus in the anterior chamber is a concomitant of corneal ulcers, iridocyclitis and endophthalmitis. A pseudohypopyon is seen in the last stages of a new growth within the globe due to actual tumour cells collected at the bottom of the anterior chamber.

59. **Hyphaema,** a collection of blood in the anterior cham- is indicative of trauma, acute inflammations of the iris especially in certain types of infection e. g., herpes, in growths of the iris and choroid and in blood diseases. Post-operatively hyphaema occurs in the diabetic and the hypertensive. Increased intra-ocular tension complicating hyphaema may cause *blood staining* of the cornea.

60. **Vesicles on the cornea** occur in herpetic infections and in any condition causing an increased intra-ocular tension as in glaucoma and uveitis. Certain dystrophies may also show vesicles.

61. **Filaments on the cornea** composed of ropy mucus and resisting attempts to dislodge them from it are part of the dry eye syndrome named after Sjogren (§ 30).

62. **Staphyloma** is a protrusion on the surface of the cornea or sclera having a fancied resemblance to a bunch of grapes. An anterior staphyloma arising from the anterior aspect of the cornea is clear evidence of past ulceration of the cornea. One situated near the limbus or slightly behind it is evidence of increased intra-ocular tension from whatever cause. Equatorial staphylomata also occur in glaucomatous globes. On occasions such protrusions are due to weak scarring after injuries.

63. **Cystoid cicatrix,** a similar protrusion, is a cystlike swelling lined by iris tissue along the upper limbus and is due to inclusion in an operation scar of iris which slowly expands under the effect of the intra-ocular tension.

64. **Oedema of the cornea** produces a lack-lustre appearance and inspection with a loupe shows an irregular reflection of the light. Gross oedema gives rise to a *bullous keratitis*. Oedema of the cornea is common in almost all flammatory conditions of the cornea. Uveitis, keratitis and glaucoma would aptly summarise the causes of oedema. A *dry lustreless* cornea is a feature of *keratomalacia*.

65. **Discoloration of the sclera** gives a clue to some diseases. Blue sclerotics are a feature of fragilitas ossium. Patchy grey areas are seen in naevi of the episclera, such patches being seen from infancy. Following scleri-

tis slate grey scars are visible. Brownish areas at the canthi are a sign of the rare condition named alkaptonuria.

66. **Discoloration of the conjunctiva** of a dirty yellow-brown colour is usual in the pre-Bitot stage of xerosis conjunctivae. White scaly areas at the outer canthus indicate xerosis conjunctivae, the patches being the classical Bitot's spots. Yellow tinging is due to jaundice and a paleness is caused by anaemia.

67. The **iris** presents useful signs of disease. Firstly its *colour:* patches of hyperpigmentation are congenital. Unilateral discoloration—heterochromia—is seen in heterochromic cyclitis. Atrophy of the iris accompanied by depigmentation follows prolonged raised intra-ocular tension or inflammation of the iris. The *pattern* of the iris is altered in iritis, the folds, crypts and surface of the iris generally losing their normal texture, the whole presenting a *muddy* effect. The pupillary border may show cystic excrescences. Strands of iris tissue may bridge the pupillary gap representing remanants of the pupillary membrane. Such strands always extend from one margin of the colarette to another. Irregularity of the pupil due to such strands must be distinguished from posterior synechiae due to iritis. The latter extend from the pupillary border to the lens capsule and are easily demonstrable on causing dilatation of the pupil by atropin.

Defects in the iris may be congenital *colobomata* which are always situated inferiorly, or artificial (operative) situated above. Tears of the iris occur either at the pupillary border or at the ciliary border. The latter, *iridodialysis*, is peripherally situated and presents a

black gap. Such an appearance may suggest other than a tear, a naevus or tumour of the iris or ciliary body growing forward at the angle of the chamber. In iridodialysis the pupil is characteristically 'D' shaped, the flattened side facing the dialysis. Besides this ophthalmoscopy gives two fundus glows (double vision is sometimes a symptom of dialysis).

68. **Rubeosis iridis** or vascularisation of the iris is a sign of diabetes mellitus, vascular glaucoma, tumours of the iris and severe inflammation of the iris.

69. **Iridodonesis** or tremulousness of the iris is a sure sign of dislocation of the lens or aphakia (absence of lens) and is due to the lack of support normally afforded to the iris by the lens. It is met with in buphthalmos where the chamber is deep and the lens recedes owing to the expansion of the ciliary ring.

70. **Nodules** on the iris are due to granulomatous uveitis, tumours and ophthalmia nodosum due to caterpillar hairs.

THE PUPIL

71. The pupil affords reliable diagnostic clues of diseases, both of the eye and of the central nervous system. Normally the pupil is *black* due to the fact that very little light is reflected back from the fundus and what little is reflected back is difficult for the observer to pick up unless he places himself directly in line with the path of the light entering the eye of the observed. Under certain conditions the pupil becomes luminous as when light enters the eye by routes other than the pupil exemplified by albinism, or when the observer places his eye in the centre of the beam of light shone into the subject's eye as done

in ophthalmoscopy or when the reflectivity of the retina is enhanced by its displacement towards the pupil. Such displacement of the retina occurs in tumours of the retina and owing to the nearness of the retina to the surface of the eye more light is reflected, the effect being further enhanced by irregular reflection of light occasioned by an uneven retinal surface.

72. Such an appearance has been aptly described as the **Amaurotic Cat's Eye** and is diagnostic of *retinoblastoma*. This characteristic appearance is easy for even untrained eyes to notice quite early in the disease for it is the mother who declares such a history to the doctor. *Any child who exhibits this phenomenon must be thoroughly examined both under a mydriatic and an anaesthetic, if the need arises, with the express purpose of ascertaining the presence of any growth within the eye and its companion.*

73. The *size* of the pupil is dependant on light, darkness, turgescence of the iris blood vessels and above all on the interplay between the sympathetic and parasympathetic systems. The two pupils may differ in size to a small degree, such inequality or *anisocoria* being within physiological limits. The pupil is small at the extremes of age.

The size of the pupil is of diagnostic importance in certain ocular and nervous disorders. A *small* pupil is seen in iritis, morphine poisoning, lesions of the sympathetic pathways to the eye and after the use of miotics (eserine, pilocarpine). A large pupil occurs in glaucoma of the closed angle type, atropine poisoning, lesions of the parasympathetic supply to the eye, optic atrophy and after mydriatics (atropine, homatropin).

74. Pupillary Reactions. In essence these comprise the reactions to light—direct and consensual, and accom-

modation and convergence. The direct reaction may be lost due to a lesion of the afferent pathways viz., the optic nerve: it is also absent when a lesion affects the efferent pathways viz., the third nerve. A simple method of finding out which pathway is affected is to shine a light into the companion eye and watch the pupil under investigation. If the consensual reaction is present the efferent pathway i. e., the thired nerve is intact. In a destructive lesion of the nerve or retina both the direct and consensual reactions to light on ipsilateral stimulation are lost. A failure of light reflexes associated with visual loss is due to lesions of the pathways common to visual and pupillary afferents. If unassociated with visual loss the lesions lie in parts where the two pathways have separated, as in the posterior part of the optic tract. *In any case where the pupillary reactions appear to be incompatible with the clinical condition it is necessary to inquire from the patient whether any drug was used locally in the eye recently.* An answer in the affirmative does not necessarily imply that a drug acting on the pupil had been used, but will lead to further questioning.

Note. A practical point worth stressing in testing pupillary reactions is to avoid the influence of the near reflex over the light reflex. This is best done by instructing the patient to look far away while the light is suddenly shone into the eye. If the examination is close to a window and an artificial light is being used to test the reactions the patient should be looking away from the window.

75. The Argyll Robertson pupil is classically a small pupil, eccentric, irregular, bilateral and while *it does not react to light it does so to accommodation.* The classical Argyll Robertson pupil as described above is a feature of syphilis of the central nervous system but an *atypical*

form—isolated light rigidity only—is said to occur in *mesencephalic tumours, encephalitis lethargica, disseminated sclerosis, herpes zoster and trauma.* It responds to atropin poorly.

76. A small pupil is one of the features of **Horner's syndrome,** the others being a slight ptosis, enophthalmos and narrowing of the palpebral fissure. The miosis is slight with retention of the light and near reflexes. Causes are syphilis, pontine tumours, thrombosis of the posterior inferior cerebellar artery in the brain stem, lesions of the brachial plexus as by a cervical rib and apical lesions of the lung. In the neck tumours, enlarged lymph nodes, thyroid swellings and aneurysms account for it.

77. **Pin-point pupils** occur in pontine haemorrhage, due either to central action or to destruction of the descending sympathetic tracts and in morphine poisoning due to central depression (though direct action of morphine on the iris musculature is known).

78. **A small irregular pupil** as seen in iritis should not be overlooked. In acute cases there is in addition ciliary injection and in both acute and chronic cases there are posterior synechiae (§ 67).

79. **Claude Bernard's Syndrome** is the opposite of Horner's syndrome (§ 76); the pupil is large and there is slight exophthalmos and widening of the palpebral fissure. Causes are as enumerated under Horner's syndrome with the difference that these in the irritative stage produce the syndrome under discussion.

80. **Adie's Pupil** is a tonic pupil—large, with delayed reaction to light and convergence. This may be associated with loss of tendon reflexes. With the combined stimulus of light and near reflexes the pupil contracts but

slowly and on removal of the dual stimulus it slowly dilates. It is unilateral.

81. Hutchinson's Pupil is a dilated immobile pupil usually homolateral to the cranial lesion—cerebral injury or haemorrhage. It is now held to be due to pressure on the 3rd nerve by a herniation of the temporal lobe of the brain into the tentorial hiatus.

DRUGS AND THE PUPIL

82. Mydriatics

I. *Parasympathetic Blocking Agents*

(a) Acting on nerve endings: Atropine, homatropine

(b) Acting on muscle fibres: Narcotine, papaverine.

(c) Acting on preganglionic fibres: Nicotine (late)

II. *Sympathetic Stimulators*

These act on the post-ganglionic sympathetic nerves and the muscles they innervate: Cocaine. Adrenaline is quickly inactivated by amine-oxidase.

83. Miotics

I. *Parasympathetic Stimulators*

(a) By preventing cholinesterase activity: Prostigmine, physostigmine (eserine). Di-isopropyl-fluorophosphate (DFP).

(b) By stimulating effector muscle cell: Choline, acetylcholine, pilocarpine.

THE FUNDUS

84. In the interpretation of fundus lesions due to the various disease processes affecting the body generally and the eye in particular many of the ophthalmoscopic signs are common and it is the peculiar combination of some or several of these signs that enables the clinician to label

any given picture as typical of a certain disease. Thus haemorrhages, exudates, pigment disturbances, sheathing of vessels, oedema of the disc, white patches in the retina and new blood vessels are some of the many fundus signs one usually sees; these may be the manifestations of inflammation, degeneration, nutritional disturbances or hereditary affections. It is important therefore to familiarise oneself with the fundus appearances to be described below.

85. **The colour** of the fundus may be altered from the normal red to a pale red or yellow in anaemia; the fundus acquires a washed out appearance in leukaemia. In polycythaemia the colour is a duller red. A grey colour is seen in retinal detachment.

86. **Pigmentation** of the fundus may be normal as in the *tesselated* or *tigroid* fundus; here owing to preponderance of choroidal pigment polygonal areas between the choroidal blood vessels stand out prominently. In congenital melanosis the characteristic appearance of a cat's paw marks on wet paint is unmistakable. Abnormal pigmentation is met with in retinal as well as choroidal affections. Bone corpuscle shaped pigment deposits are classically seen in retinitis pigmentosa. Heavy dark pigment deposits fringing a white patch is seen in the late stages of choroidal inflammation. Irregular blotches of pigment may be seen in both inflammations and degenerations. Perivascular sheathing with pigment is a feature of retinitis pigmentosa. Pigmentary changes in inflammations is a sign of old disease.

87. **White Patches** in the fundus may range from genuine exudates to atrophic areas in the retina and choroid. Superficial exudates are woolly owing to the fact that these, lying in the nerve fibre layer, are free to spread out. Deep exudates are circumscribed and rounded owing to restrictions imposed by the supporting elements of the retina. At

times large whitish areas appear scattered over the retina replacing what were once haemorrhages. In long standing oedema of the retina and optic nerve again white patches make their appearance—these are really cytoid bodies. *Opaque (medullated) nerve fibres* present a characteristic appearance lying close to the disc and spreading out fanwise (mare's tail) fine striations of the nerve fibres being easily visible. Many exudative phenomena tend to involve the macula and its surroundings. The *macular fan and star* occur in hypertensive, arteriosclerotic and nephritic retinopathies; they occur in papilloedema too. These seem to be an expression of retinal oedema, the remarkable linear deposits of exudates in a regular geometric pattern being determined by the anatomical structure of the retina. A *circinate* arrangement of the exudates occurs in circinate retinopathy. Atrophic retina and choroid show the bare sclera over areas of choroiditis (late). A large tongue shaped white area inferiorly extending up to the disc is indicative of a *choroidal coloboma* usually associated with a similar defect in the iris. Strands of white 'tissue' extending from the disc to other parts of the fundus suggest *retinitis proliferans*. Such strands carry blood vessels on them. Folds of white-grey retina are seen in retinal detachment. Colloid bodies are white and at times are diffusely scattered so as to merit the name *guttate choroiditis*. When these bodies aggregate in the form of polygonal masses at the macula the condition is termed *honeycomb choroiditis*.

88. **Haemorrhages** in the retina are red in colour and may be streaky and *flame shaped* when superficial and rounded when deep seated for the same reasons mentioned under exudates. Large sheets of haemorrhage occur in some vascular catastrophies such as thrombosis of the

central retinal vein. Blotchy ill-defined haemorrhages are seen in anaemias, leukaemias and in trauma to the eye. Pre-retinal or *subhyaloid* haemorrhage is a feature of subarachnoid haemorrhage and takes the shape of a half moon with the convexity inferiorly lying in the macular zone. Minute rounded haemorrages occurring in the central area are met with in diabetes mellitus. These are really *micro-aneurysms* and occur in hypertension and arteriosclerosis and not exclusively in diabetes mellitus. Roth's spots are tiny white spots sur rounded by a halo of haemorrhage and occur in any septic retinitis but are typical of bacterial endocarditis.

89. **The vascular tree** bears important evidence of disease. Alteration of the arterial calibre and the ratio between the arterial and venous calibres from the normal 2 : 3 is suggestive of blood vessel disease. Excessive tortuosity in itself may only mean a congenital anomaly with hypermetropia. Straightening of arteries is evidence of sclerosis. Nipping of the arterio-venous crossings is again met with in hypertension and arteriosclersois. Beyond such constrictions the vein may be dilated sometimes to a bulbous shape : such a phenomenon is termed *banking*. On occasions dilatation of a vein beyond the arterio-venous crossing may be extensive involving a good length of the vessel. Marcus gunn first described this and is usually known as *Gunn's sign*. Aneurysmal formations of the retinal vessels are known to occur. Attenuation of arteries together with a burnished coppery and later a silvery sheen indicate marked sclerosis. New blood vessels at the disc or along the course of a vein or within the vitreous are proof of a collateral circulation following a venous occlusion. New vessels in the vitreous usually take on the form of a veil on the surface of which the most grotesque patterns of intertwining blood vessels are

visible. This inspired older ophthalmologists to append the term *rete mirabile* to this network of vessels.

Arterial spasm may be observed in certain conditions such as hypertension, eclampsia and migraine. These spasms may be generalised and involve large segments of the arteries or may be localised to small segments. In both types the patterns of vasospasm are similar. Sausage shaped constrictions, a bloodless ring of constriction, a dilated vessel forcing its way into a narrow thread like portion of the vessel ahead are some of the types of peristaltic waves encountered. When the blood flow is suddenly reduced as in central arterial occlusion the blood column in the veins is broken up in small segments giving rise to a '*cattle truck*' appearance.

90. Macular changes are at times so characteristic of certain disease processes that their detection is important. A *cherry-red* spot at the macula is seen in central arterial occlusion, Tay-Sachs' disease, quinine amblyopia and occasionally in central venous thrombosis. This effect is due to a contrast phenomenon, the normal redness of the macula being accentuated by the surrounding pale retina. In all excepting Tav-Sachs' disease the cherry-red spot disappears with time. Pin-head exudates in a circumscribed area of yellowish looking oedematous retina is typical of *angiospastic retinopathy*.

91. Disc changes include pallor, cupping, swelling, vascular abnormalities and curiosities such as coloboma. A normal disc shows a central depression—the *physiological cup*—at the bottom of which may be seen little grey dots representing foramina in the sclera through which optic nerve fibres leave the eye. Owing to the shifting of the blood vessels to the nasal side of the disc the temporal half of the disc is usually paler. The blood vessels

run flush with the disc edge excepting when the disc is cupped in which case the vessels dip into the cup and in papilloedema they climb over the swelling. The use of the battery of lenses will greatly help in eliciting these two important signs (§ 11c). The earliest sign of papilloedema is a filling in of the physiological cup. *Temporal pallor* of the disc is seen in disseminated sclerosis but takes time to develop and is due to affection of the papillomacular bundle of nerve fibres which are selectively involved in this disease. A *halo* round the disc is a physiological variation seen once in a while though it may occur in advanced glaucoma and in peripapillary choroidal sclerosis. A *temporal crescent* is a concomitant of progressive myopia and is due to stretching of the retina and choroid resulting in exposure of the sclera. A *pigment crescent* round the disc is due to the retina stopping short of the disc edge and the choroid showing through the crescentic gap.

A misshapen disc is seen in astigmatism ; a small disc is a feature of hypermetropia while a large disc is met with in myopia. These size differences are dependent on the refractive state of the eye which imposes varying magnifications of structures within the fundus.

A uniformly pale disc is seen in optic atrophy: with a clear cut disc edge this atrophy is termed primary and when the disc edge is fuzzy the atrophy is secondary to papilloedema.

Sheathing of blood vessels may be congenital especially near the disc and is usually connected with Bergmeister's papilla. In sclerosis of the retinal arteries sheathing may be parallel or pipe-stem. Sheathing also occurs after papilloedema and is due to overflow of neuroglial tissue from the disc.

PART II

SOME COMMON OCULAR DISORDERS

AFFECTIONS OF THE NEWBORN

92. Birth Injuries

Both owing to normal and abnormal forces of labour the foetus is subjected to strains and stresses as it traverses the birth canal. The eye in common with other parts of the body suffers damage due to the moulding processes. An *orbital haematoma* may arise from a tear in the periorbita; such a haemorrhage may cause a *proptosis*. Extreme proptosis may cause *exposure keratitis* (see later) and loss of the eye through corneal ulceration. Extra-ocular muscles, chiefly the lateral rectus, may get damaged and produce *paralytic squint*. Rarely the whole eye may protrude forwards between the lids due to an *avulsion* of the optic nerve—such an injury being consequent on forceps application.

Corneal damage takes the form of *rupture of Descemet's membrane*. Corneal opacities of varying degrees sometimes resembling porcelain may be encountered. These clear up with time though the condition looks alarming at the start.

Subconjunctival and Retinal haemorrhages occur in a proportion of cases. *Optic atrophy* may occur in some.

Treatment. Excepting avulsion of the optic nerve all other injuries have a good prognosis. In the case of proptosis with threatened exposure keratitis every precaution must be taken to avert disaster. The cornea should be kept moist with N. saline drops applied frequently; to prevent minor trauma from foreign particles and to lubricate the movements of the lids over the cornea sterile

liquid paraffin drops would be ideal. In the presence of infection antibiotic drops or ointment should be used freely. If ulceration sets in atropine 1 percent. drops should be instilled and the eye covered with a dressing. Reassurance of the parents, careful watching and the simple measures suggested above will in most instances tide over this emergency.

Paralytic squints require surgery for their correction and these must be corrected as early as possible in order to secure binocular single vision and avoid compensatory deformities such as ocular torticollis.

Retinal and subconjunctival haemorrhages clear up without causing impairment of vision. Lid haematoma or *facial paralysis* caused by the blade of a forceps also recover fully. Caution is necessary in the second instance for inability to close the lids may cause exposure keratitis especially as tears are not secreted during the first two weeks of life.

93. Blocked Nasolacrimal Duct. Due to improper canalisation owing to delayed necrosis of cells forming the epithelial column or owing to retention of necrosed cells in the lower part of the duct or folds and partitions in the duct there may be symptoms of blockage. As tears are not formed till the third week of postnatal life defective drainage is noticeable only after this period. The mother complains that tears constantly overflow from the corner of one eye (usually unilateral). In the early stages there is no redness; after sometime, however, there is slight redness and mucoid discharge at the inner canthus.

Treatment. The only effective treatment is probing of the nasolacrimal duct for one such dilatation early will clear up the block. Conservative measures such as digital pressure over the sac and local antibiotics may be given a

short trial in case the parents are not willing to have the child's duct probed. Under general anaesthesia the lower punctum and canliculus are dialted and a probe, size 1 is passed into the canaliculus till it hits bone. Thereupon the probe is turned at right angles down and slightly backwards into the nasolacrimal duct. Dilatation is effected by passing in succession probes of varying sizes 1—6. Care should be taken to avoid creating false passages. Usually one dilatation suffices, but a second or third dialatation may be done if necessary at intervals of 2—3 weeks.

94. Ophthalmia Neonatorum. This may be defined as hyperacute conjunctivitis of whatever cause. The conjunctivitis, often bilateral is usually purulent though it may be catarrhal and rarely membranous. The diagnosis is simple for there is discharge of mucopus or pus or crusting of lids. *Any discharge from the eyes of a newborn within 21 days of birth is suspicious* of this condition. Among causative organisms staphylococcus accounts for 25%, gonococcus 35% virus 20% and other organisms for 20% of cases. It is seen that the gonococcus figures only in about one third the number of cases.

Mode of infection is one of three ways: (a) before birth, a rare event; (b) during birth, the commonest and (c) after birth from contaminated fingers of midwives, nurses or mother, dirty clothes, dressings or from infected material from the body being washed into the eyes at the first bath.

Complications sometimes take a serious turn in this disease. Owing to the infant cornea having two instead of five layers of epithelium and owing to the absence of tear secretion for about three weeks after birth, the defence against bacterial attack is poor. Pent up pus

and multiplication of organisms behind glued lids cause damage to the cornea with ulceration and loss of the eye. If the eye is not lost the cornea is scarred to a severe degree that hardly any vision develops. In other words *one must bear in mind blindness as the ultimate result in neglected ophthalmia neonatorum.*

Treatment is both prophylactic and curative. *Prophylaxis* is achieved by attention to obstetric and ocular hygiene. *Obstetric* attention is directed to the investigation and treatment of maternal leucorrhoea, swabbing of the lids at birth and scrupulous cleanliness of the obstetric attendant and the paraphernalia used in the labour room and bath. *Ocular* prophylaxis is mainly Crede's method of instillation of silver nitrate drops 1 percent into the eyes at birth. Although this method did reduce the blindness rate in England where once ophthalmia neonatorum ranked first among blinding diseases, it is now losing popularity owing to certain disadvantages associated with its use. Silver nitrate itself causes a 'conjunctivitis' and is effective against gonococci in the conjunctiva of the new-born but not against staphylococci and other organisms. The drug has been confused with disinfectants usually provided in a midwife's bag. The drug tends to get concentrated with time and a stronger solution may produce drastic effects. As a result the consensus of opinion is against the use of silver nitrate as a prophylactic though Crede's method may still be practised using an antibiotic as the substitute for silver nitrate. It is best to use a streptomycin-penicillin compound ointment[1] or polyfax ointment[2]. Penicillin alone whether in solution or ointment

1. Penicillin 100,000 units, Streptomycin 1 G, base 2 oz.
2. Trade name for an ointment incorporating bacitracin and polymyxin B in the proportion 500 : 10,000 U/G base.

form is not of much value especially in institutions where so many resistant strains of staphylococci exist. Combination with streptomycin is therefore advisable. Where a bacteriological examination is possible proper treatment can be instituted with the appropriate antibiotic.

Curative treatment in established cases is on the lines suggested by Sorsby. A smear is taken for culture, the excess of discharge is wiped with a swab and antibiotic therapy is started. While a nurse sits with a baby on her lap another instils penicillin (or penicillin-streptomycin) drops every minute for half an hour and every five minutes for the next half hour. Thereafter the child is returned to its cot and drops instilled every half hour for six hours, every hour for 12 hours every four hours for 24 hours and gradually tailed off till apparent clinical cure has lasted two days. Oral sulphamezathine may be given, 0.5 G initially followed by 0.25 G six hourly and continued for 48 hours after apparent cure. Other antibiotics may be tried in resistant and virus infections. In case the cornea looks cloudy atropine 1% drops should be instilled to prevent complications. For home treatment it is best to use a compound ointment four to six times a day and once at night, supplemented by oral sulpha drugs.

95. Cataract in the new-born may be lamellar or total. The former unless very dense passes undetected till the child is older. It is due to some metabolic disturbance coupled with a deficiency of vitamin D ; the latter is responsible for defective incisor teeth later in infancy. Total cataract is seen in infants born of mothers who had had rubella in the early months of pregnancy. Treatment is operative.

96. Buphthalmos or ox eye is due to raised intraocular pressure caused by an embryological anomaly at the angle

of the anterior chamber. Persistence of embryonic mesoderm at the angle prevents the aqueous gaining access to the drainage paths. Consequent on the raised tension the anterior part of the eye bulges, producing a large cornea, a deep anterior chamber and usually a myopic refraction. The cornea appears steamy owing to oedema. Injection, pain, photophobia and vomiting are some of the signs and symptoms.

Treatment is mainly surgical and must be undertaken early. Corneo-scleral trephining, cyclodialysis, cyclodiathermy and goniotomy are the usual operative procedures adopted. The last mentioned aims at dividing the mesodermal remnants at the angle of the anterior chamber so that the aqueous may gain access to the canal of Schlemm.

Some Affections of Infancy and Childhood

97. Keratomalacia, a major cause of blindness in children, is caused by avitaminosis A. In protein malnutrition in which there is vitamin A deprivation keratomalacia is one of the manifestations of the syndrome. Usually preceding the full blown picture of keratomalacia, there is xerosis of the conjunctiva. At first the cornea becomes lustreless, dry and shows desquamation. Owing to loss of corneal sensation associated with this condition minor traumata quickly cause ulceration and loss of the eye through panophthalmitis. It is hardly necessary to mention that the corneal condition progresses downhill directly in proportion to the dehydration and lack of proteins. Directly the body fluids return to normal level and nutrition improves the cornea quickly regains its lusture and healing is prompt. In many cases useful vision is regained if general treatment is resorted to quickly.

Treatment aims at the cause of the marasmus firstly supported by a diet of skim milk initially which is changed to half-cream milk. Vitamins A and B complex are given parenterally. The value of vitamin B_{12} in the treatment of this condition is not fully evaluated.

Ocular treatment aims at resisting infection by the use of antibiotics. Complications such as iritis accompanying corneal ulceration are treated with atropine drops; perforation of the cornea and other sequelae are treated on the same lines as for corneal ulcers generally.

98. Glioma of the retina is the name given to any retinal growth and has come to stay in ophthalmic literature. It is a misnomer and though true gliomata (astrocytoma) occur the common retinal growths are retinoblastoma and neuro-epithelioma. Under the microscope the former shows pseudo-rosettes whereas the latter is characterized by true rosettes.

Clinically growths of the eye pass through four stages—quiesence, intra-ocular spread, glaucoma and extra-ocular extension. These stages are arbitrary and are of little moment from the point of diagnosis and therapy. One would like to spot all cases of new growth at the earliest opportunity. As a history is unobtainable from a child with a retinal growth one has to depend on the mother's story. A history of loss of sight as indicated by inability of the child to recognise its parents or toys, eccentric gaze, or roving movements of the eyes and the amaurotic cat's eye appearance are the only diagnostic points. The differential diagnosis centres round a group of disease processes collectively termed pseudoglioma (§99).

Treatment is excision of the first eye and radon seed implantation over the site of the tumour in the second eye

if and when involved. After the first eye is dealt with a strict supervision of the child is necessary with the sole purpose of spotting a growth in the second eye early. Metastasis is by direct spread.

99. Pseudoglioma is the term applied to a group of conditions which mimic glioma clinically and it embraces metastatic endophthalmitis, toxoplasmosis, syphilitic and tuberculous choroiditis, congenital detached retina and retrolental fibroplasia. Of these the first mentioned is the commonest and is due to uveal metastasis from some infective focus in the body. Furunculosis or some febrile illness in a child round about the age of 2 is often responsible. *A history of such illness is of importance in the diagnosis of this condition though by no means conclusive.* The surest proof of the diagnosis is histological.

Clinically the eye presents the amaurotic cat's eye appearance. On ophthalmoscopy the white mass behind the lens is smooth whereas in 'glioma' of the retina the surface of the mass shows new blood vessels on a detached retina. Glioma may be met with at birth but pseudoglioma occurs round about 2.

X-rays of the globe and orbit may be of help in diagnosis for in glioma calcification of the globe occurs and also if the growth has started to invade the cranium the optic foramen would be enlarged. These changes are not seen in pseudoglioma.

Treatment is of no avail once the metastatic infection has established itself. Excision of the eye may be necesssary for a histological proof of malignancy or otherwise.

100. Blepharitis or inflammation of the lid margin may be dry or ulcerative. In the former the lid margin is red and shows bran-like scales and is associated with seborr-

hoea of the scalp. In the latter the inflammation is more acute; there is ulceration round lash follicles with yellow adherent crusts. Chronic blepharitis results in loss of lashes (madarosis) atrophy of the lid margin and trichiasis (§ 44).

The causative factors are debilitating diseases, irritants, continued close work especially in the presence of refractive errors. Diseases such as trachoma, lacrymal obstruction and infestation of the lashes by parasites also cause blepharitis. Acne rosacea is another condition accompanied by it. The possibility of a diabetic background should not be overlooked.

Treatment is prophylactic including correction of refractive errors. Of local treatment the first important step is to remove the crusts as otherwise any amount of topical therapy with antibiotics will be of no avail. Crusts may be removed by soaking the lids with a solution of sodium bicarbonate gr. 4 to the ounce of water or olive oil. In the curative treatment of this as well as other lid infections consideration should be given to the causative organism. As the staphylococcus is the commonest offender any of the antibiotics may be used. Penicillin itself is sufficient but of recent years resistant forms have been encountered and also penicillin acts as a sensitizing agent. As lid infections are due to mixed flora including *Pityrosporum ovale* it is inadvisable again to use penicillin as the latter aids the production of yeast forms. To be exact therefore a bacteriological study is essential. A workable suggestion is not to use peniciilin generally and at random; a combination of antibiotics would appear to be better and in fact is popular. Polyfax ointment, a combination of polymyxin B effective against Gram negative organisms and bacitracin effective against Gram positive organisms, is useful.

Caution: The lids may be affected in several skin diseases and it is the practice amongst some dispensers and others not acquainted with skin disorders to prescribe *chyrsarobin* ointment to be used on the forehead and face. It is unavoidable that such applications easily find their way into the eyes with disastrous results. A severe kerato - conjunctivitis follows such use. Psoriasis is one of the skin diseases for which this drug is employed. *It is imperative that this drug be not prescribed for use on the face.* There is a Health circular prohibiting its use on the face.

101. Phthiriasis Palpebrarum is the term applied to describe infestation of the lashes with the crab-louse, an inhabitant of the pubic region. The parasite is probably transferred to the lashes by the hand. The louse clings to the root of the lashes. Nits are usually seen in numbers (§ 43) along the lashes. A blepharitis accompanies this parasitic infestation.

Treatment consists in delousing the initial reservoir of the parasite by DDT or benzyl benzoate; shaving the part may be necessary. Locally, applications of yellow oxide of mercury (HOF) 1% or physostigmine 1% drops or ointment suffice.

102. Stye (Hordeolum) is a minute abscess in relation to a sebaceous gland of a lash follicle. It is characterized by pain, swelling and great tenderness of the lid margin. On occasions especially if the stye is in one of the corners of the eye, the swelling and chemosis of the conjunctiva are out of all proportion to the size of the offending lesion. On the summit of the swelling appears a tiny yellow spot in a day or two after which the stye may open at this point. Causes are general debility and refractive errors.

Treatment is directed towards the correction of the aforesaid defects. Locally the lash around which the swelling has developed is pulled out. Hot fomentations and instillations of penicillin drops or the use of penicillin ointment three or four times daily should effect a cure rapidly. It may happen that penicillin may invite an allergic reaction in which case some other antibiotic has to be substituted. Polymyxin B may prove more effective owing to the above stated reason. In resistant cases incision is made parallel to the lid border to avoid cutting a blood vessel and a visible scar.

103. Meibomian (tarsal) Cyst. This is a cyst affecting the tarsal gland and produces a rounded hard swelling beneath the skin of the lid and away from the lid border. On everting the lid the cyst appears to be a yellowish circumscribed area surrounded by a halo of reddish discoloration. Left to nature such a cyst may burst open at the yellow spot and extrude a jelly-like mass of granulation tissue. This mass assumes a polypoid shape, flattening to accommodate itself under the lid and closes the opening on the lid. At times infection causes an abscess which causes severe pain and induration of the lid. The essential pathology of this condition is an obstruction of the duct of the tarsal gland with infection. Round cells, epithelioid cells and giant cells compose the granulation tissue within the cyst.

Usually painless unless infected the cyst declares itself as a swelling on the lid. It may cause astigmatism by pressure on the cornea.

Treatment aims at general attention to health and correction of refractive errors. Locally the use of antibiotic ointment with lid massage may effect a cure in early cases. In others incision and curettage of the cyst cavity

is the only rational measure. The incision is vertically placed on the everted lid. Curettage is effected with a chalazion spoon. The eye is kept bandaged for a day. Thereafter some antibiotic may be used topical y. It should not be forgotten that the cyst cavity will fill up with blood immediately after scraping out all the granulation tissue within it. The patient should be reassured that such a swelling is to be expected and that it will pass off with time.

104. Phlyctenular Disease is a major cause of morbidity in children. This terminology includes phlyctenular keratitis and conjunctivitis. Phlycten means a blister but in reality it is a solid pustule composed of polymorphs, mononuclears and giant cells. In essence there is a sub-epithelial abscess. Several factors have been incriminated as causative agents. It is now known that phlyctenulosis is an allergic manifestation of tuberculosis. The tubercle bacillus has never been seen in the lesion ; the reaction is dependant on a prior sensitization of the tissues and later intoxication by tuberculo-protein. The latter is supported by the high incidence of tuberculin sensitivity in children with this disease coupled with the production of phlyctenules on the instillation of tuberculin in the eyes of hypersensitive individuals. Further evidence is provided by the fact that these children are exposed to frankly infectious tuberculous persons and by the beneficial results of tuberculin desensitization.

Clinically a *conjunctival phlycten* appears as a pink elevation which later presents a small pit owing to break down of the surface. Epithelialization quickly follows. Crops of phlyctens may occur from time to time. These classically appear near the limbus. Secondary infection usually supervenes producing a muco-purulent conjunctivitis. Owing to discharges running over the lids itching

of the lids is a feature; rubbing the lids adds a further nidus for organisms to grow and also to invade the conjunctiva.

A *corneal phlycten* appears as a small grey nodule in the superficial layer of the cornea ; when this breaks down a shallow *ulcer* results which on healing leaves minimal or no scarring. Another type of this condition is termed a *fascicular* ulcer owing to its characteristic appearance of an ulcer trailed by a leash of blood vessels. Such an ulcer usually starts at one limbus and while its limbal edge heals its opposite edge eats away into the surrounding cornea until it reaches the opposite limbus ; the path of traverse of the ulcer is delineated by a non-branching band of blood vessels. At times a *phlyctenular pannus* develops in relation to a corneal infiltrate and this unlike the trachomatous pannus does not electively affect the upper limbus. A rare form of ulceration is a deep ulcer invading the substantia propria ; superficial and deep vascularization occur with deep scarring. Secondary infection may lead to nasty effects, perforation being a rare event.

Treatment consists in attacking the tuberculous focus with appropriate antibiotic therapy. Locally cortisone drops or ointment 1—2 percent may be used with benefit. Atropine should be used in all cases of corneal phlyctens. Protective goggles or shade will prevent annoyance from glare. Generally vitamins and good diet are indicated. The antibiotic therapy should be fully carried out. In the presence of secondary infection antibiotics may be used locally, while the topical cortisone, unless combined with an antibiotic, should be stopped.

Severe photophobia may be dealt with by frequent applications of ice water to the lids and face. Rubbing of the lids and excoriation of the corners of the eyes should be prevented.

105. Interstitial Keratitis is an inflammation of the parenchyma or substantia propria of the cornea and is caused by certain specific agents e. g., tuberculosis, leprosy and syphilis. In most cases there is associated with the the keratitis an anterior uveitis.

The *syphilitic keratitis* will be first described. It is a late manifestation of hereditary syphilis and it, as well as other stigmata of congenital syphilis constitute important criteria in diagnosis. The other stigmata are Hutchinson's teeth and deafness. It is to be noted that this disease is becoming rare.

Interstitial keratitis occasionaliy occurs in acquired syphilis (3%). It is usually uniocular and runs a milder course being limited to a sector of the cornea.

The corneal inflammation is allergic in nature, the cornea being at first sensitized by the treponema and subsequently excited by some toxins liberated by the organism elsewhere in the body. This may even be produced by treatment.

The essential pathology is a massive lymphocytic infiltration associated with vascularization; there is associated necrosis of the corneal parenchyma to be followed by a reparative process. The latter is due in part to proliferation of the corneal corpuscles and in part to fibroblastic activity of the invading cells. It is the posterior layers of the cornea that are invaded, the endothelium and Descemet's membrane being inflamed. Folds in Descemet's membrane and 'k. p.' indicate the severity of the involvement of these. New blood vessels invade the deeper layers of the cornea mainly as a defence mechanism and long after the reparative processes are over these remain as empty flattened tubes and present curious geometric patterns. Owing to their appearance and their emptiness they are referred to as *ghost vessels.*

Clinical features. The congenital type affects preferentially the end of the first and second decades of life. It is bilateral as a rule, some weeks intervening before the onset of symptoms in the second eye.

Pain, photophobia, lacrimation and spasm of the lids initiate the disease. A lilac ciliary injection with corneal haze at multiple foci coalescing to produce a ground glass appearance are to be seen. The vascularization is of an extreme degree: the conjunctival vessels distend and form an *epaulet* over the corneal edge while the deeper vessels invade the cornea in such numbers that parts of the cornea appear salmon pink. Folds of Descemet's membrane, endothelial inflammation and k. p. produce a dense central opacity. This, the florid stage of the disease lasts about 2 to 3 months.

The stage of clearing sets in and is slow from the periphery centrally. Faint opacities and lines, the latter being ghost vessels, persist in some.

Owing to an attendant cyclitis there may arise a *secondary glaucoma.* Very rarely an eye might shrink following such a complication.

Prognosis on the whole is good.

Treatment is general and local. General treatment consists in treating the syphilitic condition. Procaine penicillin is the sheet anchor at present. Locally atropine is indicated to avoid complications. Penicillin subconjunctivally in doses of a million units every 24 hours is indicated. Cortisone drops or ointment 2.5% frequently will bring much relief. This is in fact the most rational line of therapy knowing the inflammation to be mainly allergic. Photophobia and lid spasm may be relieved by

frequent cold applications to the lids. Pain may be relieved by the liberal use of aspirin and sedatives. Dark glasses will add to the comfort of the patient by day. The most important point to remember in the treatment of these cases is to see that the antisyphilitic treatment is complete. Raised tension is no indication for stopping atropine.

Raised tension may be dealt with by surgery supplemented by acetazoleamide (diamox) administered orally.

Residual corneal opacities if interfering with vision will require keratoplasty for the restoration of sight.

Interstitial keratitis due to *tuberculosis* while having similar manifestations differs from the above by affecting only one eye and involving a sector of the cornea.

106. Catarrhal Conjunctvitis is a mild inflammation of the conjunctiva caused by bacteria and excited by irritation by dust, smoke or fumes. Conjuncitval injection a slight discharge and a smarting sensation are the chief manifestations.

Mild antiseptics like boric acid lotion (10 grains to the ounce) or chemotherapy with sulphacetamide 10% drops or ointment effects rapid relief.

107. Muco-purulent Conjunctivitis is an acute conjunctivitis of bacterial origin. The causative organisms are the Koch-Weeks bacillus and the pneumococcus. The former causes "pink eye"; epidemics are caused by both. The disease is highly contagious and is easily propagated from person to person by contact and by flies. The fruit fly is the chief offender in Ceylon. The dry dusty atmosphere is also conducive to the easy transmission of the disease.

Clinical features. Incubation period is about 48 hours. The disease is ushered in by a feeling of soreness, lacrimation and photophobia. Discharge at the canthi and lid borders of a muco-purulent type and severe congestion of the conjunctiva are features that develop soon after. Petechial haemorrhages under the conjunctiva, usually in the pneumococcal type, add further to the redness of the eye. On waking in the mornings the lashes are glued together by thick discharge. After a course of about five days the disease slowly fades away. In the pneumococcal variety resolution by lysis is known.

Treatment. Owing to certain complications that follow neglected or fulminant cases of mucopurulent conjunctivitis active treatment MUST be instituted as early as possible. The major complication to be feared is corneal ulceration which may impair vision to a great extent and on some occasions destroys the eye.

PROPHYLAXIS. In Ceylon epidemics of 'pink eye' or sore eye occur during certain seasons. The hot, dry dusty climate is partly responsible for the propagation of the condition. As it is not possible to have any control over the elements it is necessary to follow certain codes to avert epidemics of the disease especially in institution like schools, prisons and camps. Common baths, basins, towels and bedding should be eschewed. All treatment utensils, dirty swabs, medicine droppers and glass rods should be properly looked after. Soiled bedding and clothes should be disposed of so as not to attract flies. Fruit flies may be destroyed on a large scale within the house by spraying DDT or by scorching them with a flame after they are allowed to settle on lengths of string attached to the eaves. It is important that a person with pink eye should avoid places of public entertainment or work.

CURATIVE. The principles of therapy are:-

1. Riddance of discharge and debris from the eyes.
2. Attack on the invading organisms.
3. Avert or deal with complications.

Crusting of the lid borders, sticky lashes and discharge should be dealt with by swabbing with wet cotton or by washing with an undine or irrigator. If the latter are used care should be taken not to direct the lotion onto the cornea. Once the excess discharge is removed thus, an antibiotic should be introduced. It is important to ascertain the causative organism before using a particular drug.

The causative organisms are the *Koch-Weeks bacillus, Pneumococcus, Influenza bacillus, the Morax-Axenfeld bacillus* and *Staphylococcus.* Penicillin is effective in the case of pneumococcal and staphylococcal infections. Streptomycin is effective in Koch-weeks, Influenza bacillus and Morax-Axenfeld infections. It is at once apparent that a combination of penicillin and streptomycin would be an ideal therapeutic combination to attack all the above organisms especially when a bacteriological examination is not possible. When such examination is possible the drugs of choice are as follows in the order mentioned:-

1. Koch-Weeks	(a) *Streptomycin* (b) Chlortetracycline (c) Oxytetracycline, Polymyxin B.
2. Pneumococcus	(a) *Chlortetracycline* (b) Oxytetracycline (c) Penicillin.
3. Influenza B.	(a) *Chlortetracycline* (b) Chloramphenicol (c) Streptomycin.
4. Moraxella	(a) *Oxytetracycline, Polymyxin* (b) Streptomycin (c) Chlortetracycline.
5. Staphylococcus	(a) *Carbomycin* (b) Oxytetracycline (c) Penicillin.

N. B.—This scheme is after Braley. The drug of first choice is in italics.

Polymyxin B and bacitracin in the form of an ointment may also be used instead of a combined preparation of penicillin and streptomycin. Reference to the use of these combinations has already been made in § 94.

Supportive treatment in the form of mydriatics is called for if there is corneal involvement. Shading the eyes is necessary in the presence of photophobia. In very fulminant cases systemic antibiotics or chemotherapy is called for. It is to be remembered that frequency of application of the various medicaments is as important as the choice and concentration of the drugs. Oedmea of the lids and conjunctiva may be massive in some and in these frequent cold applications would help. A watch for allergic reactions due to any of the above drugs should be kept. Preparotid lymph node enlargement if encountered requires reassurance of the patient.

108. Spring Catarrh or vernal conjunctivitis as the name suggests is an affection prevalent during the spring in countries where seasons exist or during the warm months of the year. It occurs in children and young adults and is allergic in nature. Photo-sensitivity and sensitivity to pollens suggest an allergic aetiology. There is evidence that this condition is associated with asthma, hay fever and E. N. T. infections—a further evidence in support of the allergic basis of this disease. Itching of lids and presence of eosinophils in the smears from the conjunctiva indicate an allergic basis. Two types are met with, one confined to the lids, the *palpebral* and *limbal or ocular*. In the former the everted upper lid shows nodules arranged like cobble stones on a pavement. In the later finer nodules of a gelatinous consistency and appearance lie all round the limbus. In both types there is the appearance of the surface being washed with milk.

Treatment is difficult in some cases. Elimination of allergens that are responsible in any particular case should be attempted. Adrenalin or the more recent antistine-privine drops afford much relief. Cortisone preparations have also proved effective in a good proportion of cases, relieving itching and cutting down the allergic reactions responsible for the tissue reactions. Syrup of calcium and dark glasses are useful. In resistant cases irradiation may be necessary. The disease is self-limiting and tends to die down after some years.

SOME AFFECTIONS OF ADULTS

There is an overlap in the incidence of certain eye diseases in the two groups—childhood and adolescence. Examples of these are blepharitis, hordeolum, meibomian cyst, interstitial keratitis, conjunctivitis and rarely phlyctenular disease. It should also be noted that some of these such as blepharitis, hordeolum, meibomian cyst and conjunctivitis may affect any age.

109. Angular Conjunctivitis is a chronic conjunctivitis caused by the Morax-Axenfeld diplobacillus—a Gram-negative organism. As the name suggests there is a classical angular distribution of the inflammation. This is due to the affinity the organism has for epithelial debris at the corners of the eye, on which it feeds. The organism secretes a dermolytic ferment by virtue of which it attaks the skin at the corners of the eye. The symptoms consist of irritation and watering and the chief sign is redness of the conjunctiva at the angles. Discharge is scanty unless secondary invaders take a foothold. Excoriation of the skin at the angles of the eye is also a characteristic sign.

Treatment. Zinc sulphate drops instilled thrice daily for a period of three to four weeks is sufficient to effect a cure. Zinc sulphate is a specific for it appears to enter into a substrate competition with the proteolytic enzyme manufactured by the organism and thus 'starves' it to death. Resistent cases should be treated with oxytetracycline. Multivitamin therapy is a suitable adjuvant.

110. Trachoma, otherwise known as Egyptian or military ophthalmia is a hypertrophic conjunctivitis caused by a filter-passing virus of the Rickettsia group. The virus,

however differs from this group sufficiently to warrant its classification in a separate family with the virus of inclusion conjunctivitis. The disease is rare in Ceylon and the few that suffer from it are immigrants from India and a few Muslims. Poverty and poor hygiene appear to be predisposing factors. All ages are attacked though in Ceylon only adults succumb to it.

Pathologically the basic lesions are an initial desquamation of the conjunctival epithelium followed by hypertrophic proliferation of the deeper cells. Concomitant with the above, lymphocytes invade the subepithelial tissue to form the *follicles* characteristic of trachoma. Later, in the fornices appear papillae which are highly vascular. Owing to the blockage of the mouths of the conjunctival glands caused by the thickened conjunctiva, blebs appear. Simultaneously with these there is a slow invasion of the cornea by blood vessels and lymphocytes, constituting the *pannus* (§ 50).

Clinically the diseases starts like a case of acute catarrhal conjunctivitis but as weeks pass the signs of trachoma slowly get established. Very often secondary invaders cloud the clinical picture for some time. The *diagnosis* depends on:-

1. Identifying inclusion bodies in scrapings of the conjunctiva.
2. The presence of follicles and blebs on the everted tarsal plate.
3. The presence of pannus

and later on on:-

4. Scarring of the conjunctiva of the upper lid,
5. Ectropion or entropion.
6. Trichiasis.

Course of the disease is prolonged but it is essentially self-limiting and all the ocular damage and visual loss are due to extensive scarring which is the ultimate end result of the hypertrophy and proliferation of the conjunctiva.

Complications include corneal ulcer, tarsitis with ptosis, entropion, ectropion, symblepharon, trichiasis and xerophthalmia.

Treatment. As in most conjunctival affections secondary infection is the rule and it is very necessary to bring such infection under control quickly. The spread of trachoma is made easier by secondary infection for the conjunctival discharge which is then profuse is easily transferred from person to person by contact or through flies and clothes. Control of secondary conjunctivitis is on the lines indicated earlier (§ 107).

Specific treatment consists of sulphonamides and the antibiotics used topically and systemically. *Sulphonamides* may be used as drops or ointment. Sulphacetamide ointment or drops may be used. Sulphonamides are effective because trachoma is a nonsuppurative condition. *Penicillin* has produced satisfactory results. *Chloramphenicol* by mouth and topically also help. *Chlortetracycline* and *oxytetracycline* have given promising results. Recently *tetracycline* has been used with benefit. A 0.5 percent ointment of chlortetracycline and oxytetracycline applied to the eyes three or four times daily effects rapid relief. In acute trachoma oxytetracycline is superior to chlortetracycline and if treatment is started early a cure may be expected in 7 days. In cases of longer duration the antibiotic applications are requirred over a longer period ranging from 4—8 weeks. In chronic trachoma treatment may be required for 2—3 months.

Complications like corneal ulceration require atropine in addition to antibiotics. Trichiasis. ptosis and entropion require surgical treatment.

Prophylaxis. As trachoma is a highly contagious disease particular care must be taken to avoid the transmission of the disease through fomites. This applies both to personal and institutional hygiene. Doctors, nurses and students should wash up immediately after seeing a case of trachoma. The fly nuisance should be controlled if that is possible. Acute infections of the conjunctiva must be brought under control rapidly and effectively.

111. Keratitis Superficialis Punctata is a major cause of ocular disability and is characterised by a conjunctival inflammation without discharge initially, followed by the appearance of small discrete corneal infiltrates. These are subepithelial and several of these coalesce to form macules—hence the name macular keratitis. *Aetiologically* it is presumed to be due to a virus closely allied to that causing epidemic keratoconjunctivitis. K. S. P. does not exhibit epidemic tendencies and pre-auricular adenopathy is only occasionally met with.

Clinically the evolution and features of the disease are as follows. A conjuctivitis free of discharge is the forerunner; within a week the conjunctival inflammation subsides giving place to a punctate keratitis confined to the superficial layers of the cornea. Vision is curtailed and there is much photophobia. It is at this time that most patients seek treatment on account of the visual disabillty and because they have noticed white spots on the cornea. In a few cases there are enlarged and tender pre-auricular lymph nodes.

Course. After variable periods ranging from about 6 weeks to 6 months the disease dies down and the vision clears up. In some instances the opacities become confluent to form macules; in others they involve the deeper layers of the cornea and produce a picture similar to disciform keratitis. Iritis is not usual in this condition.

NOTE. Trauma appears to be an exciting factor, a state of affairs not unknown in other types of corneal disease. Herpetic inflammations are sometimes initiated by injuries. Neurotrophism is also thought to be responsible, for corneal anaesthesia is met with in many of these cases.

Treatment. It used to be the fashion to treat this condition with dionin. With the advent of antibiotics trends in therapy are towards the use of these. Only the newer antibiotics have some effect and they should be employed before the corneal opacities make their appearance. Chlortetracycline and oxytetracycline may be tried. The secret of success depends on the early intstitution of therapy. Once corneal opacities appear these drugs certainly help to control the disease but the keratitis runs its own course. Cortisone may be given subconjunctivally or in the form of an ointment, 2.5 percent. In cases where there is a respiratory catarrh oral antibiotic therapy is indicated. A shade or goggles may be worn to ward off glare. Pre-auricular adenopathy requires no special treatment excepting applications of warmth and the administration of analgesics.

112. Herpetic Keratitis. The viruses of herpes simplex and of zoster produce among other manifestations keratitis. The virus of herpes simplex attacks the ectodemal layers of the cornea while that of zoster attacks

the mesodermal layers of the eye—the corneal parenchyma and the uvea. Though the two types of ocular involvement are grouped under the same heading there are several ways in which they differ from one another. These include clinical and therapeutic dissimilarities which are summarized in the following table adapted from Paton and Duke-Elder and modified by the author.

TABLE II. Differential Diagnosis—Ocular Herpes

Epidemic Herpes Zoster	Herpes Simplex
1. Ocular mesoderm affected.	Ocular ectoderm affected
2. Virus not one of normal conjunctival flora.	Virus lives in symbiosis with conjunctival cells.
3. No preceding disease.	Follows infective diseases, usually respiratory.
4. Lesions follow distribution of nerves affected.	Not necessarily so.
5. Preceding severe neuralgia.	No neuralgia preceding onset.
6. Confers immunity.	Recurrence common.
7. Attacks deeper layers producing scars.	No scarring.
8. Ocular complications common: iritis, scleritis, keratitis muscle palsies, optic neuritis.	Few ocular complications.
9. Duration 3-4 weeks; anaesthesia prolonged.	Duration shorter, often a few days.
10. Cortisone treatment beneficial.	Cortisone treatment harmful.

A. **Herpes simplex** or febrilis affects any age and is preceded by a febrile illness. Vesicles appear on the lids and cornea. The latter break down to form arborescent superficial ulcers—dendritic ulcers. Recurrence is common. Pain photophobia, lacrimation and impaired sight are the main clinical features.

Spread of the ulcer may be superficial or deep. In the latter a disciform keratitis may result. Iritis, sometimes

with a hyphaema is a known complication. A late complication is *keratitis metaherpetica* in which small round ulcers recur after the initial lesions have healed. **Treatment.** The time-honoured carbolisation of the ulcer is still the best remedy as antibiotics and chemotherapy have done little to improve the outlook in these cases. Atropine drops are necessary to forestall any iritis. Pain is relieved by analgesics.

B. **Herpes Zoster Ophthalmicus** may be epidemic or symptomatic.

The epidemic variety is caused by a virus. The symptomatic variety is due to implication of the ganglion in some other lesion, inflammatory, traumatic or neoplastic.

The onset of the eruption on the face and forehead is preceded by agonising neuralgia. The skin eruption which is vesicular, closely follows the distribution of the first division of the Vth. Nerve. The keratitis is deep and is composed of minute subepithelial dots which coalesce to form round spots. In severe cases Descemet's membrane shows folds. There is corneal anaesthesia. Iridocyclitis is a major complication due to which at times a secondary glaucoma occurs. Other complications include scleritis, ocular palsies, optic neuritis and dacryoadenitis.

Treatment. Analgesics and sedatives are needed for relief of pain. The skin lesion is best dealt with by a collodion dressing. Keratitis and iritis require atropine drops twice daily. To prevent secondary infection antibiotics should be used, notably the tetracyclines. Cortisone (ophthalmic) may be used to advantage. Pituitrin 1 ml. deep subcutaneously may be given in

cases of intractable pain. Vitamin B_1 and B_{12} may be administered.

113. Corneal Ulcer (Keratic Ulcer, Suppurative Keratitis). Ulceration of the cornea occurs at any age but the bulk of cases is during adolescence. Briefly ulcers of the cornea are divisible into (a) *Marginal* and (b) *Central.*

(a) Marginal: 1. Metastatic; 2. Infective—*Primary*: zur Nedden's bacillus; *Secondary*: Staphylococcus; 3. Mooren's ulcer.

(b) Central: 1. Pneumococcal; 2. Diplobacillus of Petit; 3. Pyocyaneus; 4. Mycosis.

The following account will deal with central corneal ulcer, a common cause of blindness in Ceylon.

Serpiginous Ulcer (Hypopyon Ulcer). As causative factors three agents figure, the Pneumococcus, injury and poor tissue resistance. The bacterial agent is of course a potent one and in a person with an infected stagnant lacrimal sac such ulcerations are highly probable owing to the presence of bacteria in abundance.

Clinical Features. Around an abrasion on the cornea a grey-white infiltrate appears. Quickly this assumes a disc shaped opacity. Denudation of the surface epithelium causes a shallow ulcer to form. Owing to the swelling of the lamellae of the cornea adjacent to the ulcer an edge begins to appear. Gradually this edge creeps (hence the term *ulcus serpens*) owing to rapid and unhindered multiplication of bacteria at the periphery of the ulcer. Meanwhile the concurrent iritis goes apace producing a bound-down pupil. Exudation of inflammatory products into the anterior chamber from the iris surface is responsible for pus in the anterior chamber—hypopyon. This is usually

inspissated and alters its level with the position adopted by the patient. In the erect posture the pus collects at the bottom of the chamber with a horizontal fluid level. The conjunctiva is congested—ciliary injection—and at times chemosed. The lids swell up while incessant lacrimation and lid spasm make for more marked oedema of the lids and even excoriation of the angles of the eye. Pain and loss of vision are the chief symptoms. Pain mounts for a time and subsides when the nerve fibres in the ulcerated area also necrose. It may however reappear owing to rising intra-ocular tension. This secondary glaucoma is attributable to a marked rise in the osmotic pressure within the eye brought on by the increased cellular contents in the anterior chamber.

Course and Complications. In healthy persons and if treatment is instituted early the ulcer may heal with minimal scarring. In old subjects in whom resistance to bacterial attack is poor the ulcer rages fast causing wholesale necrosis of the cornea—*phthisis corneae.* In others the ulcer spreads deeply and ends in perforation, whereupon the anterior chamber is lost. The iris is forced against the edge of the perforation, to which it gets plastered. Healing takes place rapidly now owing to the availability of blood vessels. Healing results in a scar to which the iris is attached —adherent leucoma. When such a scar is small and pracentral, useful vision results however small and deformed the pupil may be, When however the perforation is extensive the iris only helps to seal the gap and a false fibrous, and therefore a weak scar results. Under the normal intra-ocular pressure the scar gives in places, the iris tissue bulging between strands of this false fibrous scar producing a fancied resemblance to a bunch

of grapes—*staphyloma*. When confined to a portion of the cornea it is called a *partial anterior staphyloma* and when the whole cornea is involved the term *total anterior staphyloma* is applied.

At times an adherent scar may give rise to a secondary glaucoma which may destory sight. This rise of tension is due to a shallowing of the anterior chamber at the periphery caused by a bowing forward of the whole iris. With the onset of secondary glaucoma pain may reappear. If unattended the increased pressure causes protrusions of the uvea through the sclera in the region of the ciliary body—*ciliary staphyloma*—or at the equator—*equatorial staphyloma*. On occasions the raised pressure causes atrophy of the ciliary body with slow collapse of the globe—*phthisis bulbi*. At perforation infection of the globe may lead to *panophthalmitis*— a fulminant inflamation of the whole eye. The end result of this again is phthisis bulbi.

When an ulcer is arrested early the scar is usually confined to the superficial layers of the cornea. A faint scar is called a *nebula*, a medium density earns the name *macula* and a dense scar is termed a *leucoma*. Defective epithelialisation results in a facet.

Vision is usually curtailed by the faintest scar. Astigmatism and irregular scattering of light by the scar are responsible for a poor retinal image.

Treatment should be planned to fight bacterial invasion, to check iritis, to overcome complications and lastly to restore sight in cases where the integrity of the eye is not lost. These entail the following measures:

1. The use of antibiotics
2. The use of atropine
3. Paracentesis of the anterior chamber
4. Keratoplasty when the eye is quiet.

Purgatives, analgesics and sedatives are necessary in all cases. Antibiotics may be used in the form of drops, ointment, subconjunctival injections and on occasions in the crystalline form blown on to the ulcer. An important attack on the reservoir of the bacteria in some cases will be the eradication of the lacrimal sac where this is the cause.

The choice of antibiotics is listed below (after Braley). The drug of first choice is in italics.

1. Staphylococcal Marginal ulcer	*a*. *Oxytetracycline*, *b*. Chlortetracycline, *c*. Penicillin
2. Pyocyaneus	*a*. *Polymyxin B—Chloramphenicol*, *b*. Streptomycin, *c*. Bacitracin—Polymyxin B
3. Pneumococcus	*a*. *Penicillin*, *b*. Oxytetracycline, *c*. Chlortetracycline
4. Proteus vulgaris	*a*. *Oxytetracycline—Streptomycin*, *b*. Bacitracin—Polymyxin B
5. Moraxella	*a*. *Oxytetracycline*, *b*. Chlortetracycline, *c*. Streptomycin
6. Koch-Weeks	*a*. *Oxytetracycline*, *Streptomycin*, *b*. Chlortetracycline, *c*. Streptomycin.

It would appear from the above that a workable proposition is to use a combination of penicillin and streptomycin as a first measure and later pass on to use the others if resistance is evident or if laboratory investigations or clinical features indicate a particular specific organismal cause.

Atropine drops or ointment 1% should be used in all cases of corneal ulcer for iritis is a complication of some severity. By paralysing the iris and ciliary musculature atropine causes sedation for once the spasm of these muscles is abolished pain is abolished. Secondly owing to the relaxation of the ciliary muscle the major arterial circle of the iris opens up and the blood supply to the eye

is enhanced. In the aged and in the presence of a hypopyon a dilated pupil may add to the risks of a secondary glaucoma. This is indeed a problem; in such cases it would be wise to perform a paracentesis of the anterior chamber to evacuate any abnormal contents of the chamber and at the same time to effect a wash out of the chamber with antibiotic solution.

A paracentesis done in the last stages of a nasty ulcer will only serve to help the ulcer in healing, with a dense opaque scar and also alleviate pain.

Keratoplasty may be undertaken in the active stage of an ulcer under certain circumstances. Such a step is a therapeutic measure and usually a lamellar graft is performed. A graft is indicated in indolent ulcers resisting all the orthodox methods of therapy.

Tattooing of the cornea may be performed in some cases so as to obscure an unsightly scar.

Panophthalmitis requires energetic treatment to cut down the violence of the inflammation. On occasions evisceration may have to be undertaken to terminate a protracted and painful illness.

Secondary Glaucoma due to extensive synechiae is best dealt with by an iridectomy which may also serve as an optical iridectomy.

Prolapse of the iris requires excision. If a staphyloma appears cauterisation of the staphyloma with an actual cautery will help to keep the bulge from increasing and also give a chance to the reparative processes to consolidate a firm scar.

114. Pterygium is a wing shaped growth of conjunctiva encroaching the cornea and usually situated on the nasal side. It is a degenerative condition, probably excited

by wind, dust and warmth, its predilection for the nasal limbus furnishing further support to the theory mentioned above. The eddies of air current at the inner canthus cause minor trauma and prolonged repeated minor trauma appears to trigger the onset of fine degenerative changes at the level of Bowman's membrane of the cornea. Coincident with these corneal changes the conjunctiva in the interpalpebral zone nasal to the cornea thickens and folds over and is drawn gradually on to the cornea. Extension onto the cornea is preceded by corneal infiltrates described above.

Symptoms are hardly any in the early stages. The chief complaint is on account of an unsightly appearance. Later as it spreads corneally it may interfere with sight by partly or wholly obstructing the pupil. Secondly a large fleshy pterygium may hinder ocular movements to such an extent as to cause diplopia. From time to time a pterygium may get inflamed and cause irritation. Cystic degeration may occur at the head of the pterygium accompanied at times by melanosis.

Treatment is surgical. Prophylactic measures, such as wearing goggles in hot desert countries are reported to be fruitful in preventing the onset of pterygium. Hyaluronidase has been tried by a few with some measure of success.

Excision of the pterygium with transplantation of the head into a pocket of conjunctiva above or below the original level of the pterygium is the basis of many transplantation operations. D'Ombrian devised a method of excision radically of the head, neck and part of the body, leaving a raw area for epithelialisation before the pterygium has a chance to grow again.

Excision combined with carbolisation is popular in Egypt and is a method advocated by Kamel. The head, neck and body of the pterygium are dissected and to their under surface carbolic acid is applied. The head and neck are cut off and the body is allowed to fall back on to the globe and the conjunctiva is stroked into place. Kamel claims good results with this method. Repeated growths have been dealt with by some by irradiation.

Recurrent pterygia can also be discouraged by lamellar keratoplasty.

115. *Keratoconus* or conical cornea is a dystrophic affection of the cornea characterised by a cone shaped protrusion of the cornea due to chages at its apex, an irregular astigmatism, an axial myopia and a progressive course.

The cone is best viewed from profile or in the early stages with the aid of Placido's disc. The apex of the cone shows a few opacities due to rupture of Descemet's membrane with consequent imbibition of water. A coloured ring, Fleischer's ring, is usually visible at the base of the cone. On shining a light with a mirror into the fundus a bizarre type of shadow effect is noticed. The upper lid appears peaked (Munson's sign).

Symptoms, apart from a grossly curtailed vision, may include at times acute pain, lacrimation and photophobia—acute keratoconus—leading to near perforation of the cone.

Treatment is ideally by means of contact lens. If the cone cannot be successfully kept at bay by the splinting action of a contact lens surgery must be resorted to, which entails a penetrating keratoplasty. The

contact lens not only abolishes corneal refraction and greatly improves vision, much to the delight of the patient but it also splints the cone and causes the breaches in the Descemet's membrane to heal, thus dispersing the corneal opacities.

In keratoplasty for this condition care must be taken to select a size of graft large enough to include the base of the cone. Donor material should be from adult cornea.

116. Scleritis is an inflammation of the sclera due to a number of aetiological agents such as focal sepsis, tuberculosis, syphilis and leprosy but in all these the scleral inflammation is allergic in nature rather than due to actual invasion by the organisms concerned. It is also associated with rheumatoid arthritis and other collagen diseases. Topographically it is classified into *anterior, brawny, sclero-keratitis* and *posterior.* The inflammation is severe in all types, with an accompanying uveitis. The course is chronic and prolonged. There is much pain, redness and swelling of the sclera. In the posterior variety a retinal detachment may give the clue. Limitation of ocular movements due to extreme swelling of the sclera and overlying muscles is another feature. Loss of an eye may result from secondary glaucoma or from the cutting off of limbal nutrition to the cornea by the massive chemosis. For the various reasons given above vision will be greatly diminished. In sclerosing keratitis the cornea adjacent to the limbus assumes the appearance of the sclera due to inflammation.

True organismal invasion of the sclera does occur in metastatic pyogenic scleritis, tubercle and syphilis of the sclera.

Treatment see § 117.

117. Episcleritis. Here the inflammation is superficial and involves the epislceral tissues. Two types are recognised: (a) Nodular episcleritis and (b) Episcleritis fugax. Nodular episcleritis must be distinguished from phlyctenular conjunctivitis; in the former the conjunctiva is freely movable over the nodule, and in the latter ulceration is usual. Fleeting from site to site is characteristic of episcleritis fugax; often the attack of the disease comes and goes with remarkable suddenness.

Uveitis is again a constant accompaniment of episcleritis.

Treatment is general and local, specific and nonspecific. Radical extirpation of any septic focus should be undertaken. Specific measures include the use of antisyphilitic and antitubercular drugs. The sulphones are indicated in leprosy. In metastatic inflammation of the sclera due to pyogenic cause sulphonamides and antibiotics are necessary.

Non-specific methods include the exhibition of salicylates, cortisone and non-specific protein shock therapy. Local therapy consists of application of warmth, leeching if congestion and pain are marked, atropin drops twice daily, cortisone drops, ointment or subconjunctival injection and a protective shade or goggles.

118. Uveitis is the term applied to inflammation of the uveal tract, viz., choroid, ciliary body and iris. Topographically it is divided into anterior and posterior uveitis while the modern classification of uveitis is as follows:-

1. Systemic infections caused by actual organisms
2. Anaphylactic reactions to——
   (a) ocular organ specific proteins
   (b) systemic infections

3. Bacterial allergic reactions——

(a) Granulomatous or plastic uveitis due to living organisms

(b) Non-granulomatous or serous uveitis due to hypersensitivity to attenuated organisms or to bacterial products.

Systemic infections include tuberculosis, syphilis, sarcoidosis, brucellosis and pyaemic conditions. The last often causes panophthalmitis.

2. *Anaphylactic* reactions are typified by endophthalmitis phaco-anaphylactica and sympathetic ophthalmitis —the former due to hypersensitivity to lens protein and the latter due to hypersensitivity to uveal pigment. Very rarely an antigen introduced into the system may produce an anaphylactic uveitis as in serum sickness.

3. *Allergic Uveitis:*

(a) Granulomatous uveitis is met with in tuberculosis, syphilis, brucellosis, streptococcal and virus infections. Owing to the protracted hypersensitive reaction the inflammation is accompanied by caseation and necrosis and a compensatory overgrowth of granulomatous tissue.

(b) Non-granulomatous (serous) uveitis is present in rheumatoid arthritis or in chronic gonococcal infection.

Clinical Features of Uveitis, For this purpose it is convenient to divide uveitis into anterior and posterior. (i) *Anterior Uveitis.* Inflammation of the iris and ciliary body—iridocyclitis—are charaterised by certain symptoms and signs distinct from those of inflammation of the choroid. Some of the features of iridocyclitis have already been dealt with in §26 and §27.

Redness of the eye, pain, photophobia, lacrimation and some dimness of vision are the symptoms. Ciliary injection, a muddy iris, small irregular pupil with posterior synechiae, 'k. p.' on the back of the cornea and a normal tension usually are the signs. Hyphaema and hypopyon may be seen occasionally in severe forms of iridocyclitis.

Raised tension may be the sequel in fulminant or neglected cases and is due to abnormal contents of the anterior chamber or due to the formation of total annular synechia. Under such circumstances aqueous is pent up behind the iris which becomes bowed forwards in its mid-zone to form the classical iris bombé. Neglected, a secondary glaucoma eventually destroys sight. Finally the globe shrinks due to atrophy and cessation of function of the ciliary body.

Some special features are worthy of note. In the *granulomatous type* the inflammation is of a plastic nature and so the organic changes include actual nodule formation on the iris and aggregation of epithelioid cells at the pupillary border constituting Koeppe's nodules. Posterior synechiae are the invariable rule, plastic exudates occur and the keratic precipitates are bulky, aptly described as 'mutton-fat' type.

In the *non-granulomatous type* the onset is acute with severe ciliary injection. 'K. p.' are small, exudates may be fibrinous or gelatinous and recovery is usual and remarkable despite an early stormy course. (ii) *Posterior Uveitis.* Inflammation of the choroid too is classifiable in the above fashion. In the *granulomatous type* the choroid shows a massive reaction in which there is much subretinal oedema and vitreous opacities. In the non-granulomatous type there is similar

subretinal oedema but there are no heavy vitreous opacities. Topographically choroidal inflammation may be divided into juxta-papillary, central and peripheral or anterior. Clues as to the aetiology of the inflammation cannot be gained by this distribution of the lesion.

Clinical features. Blurring of vision together with absence of pain and redness are the main manifestations. The anterior segment of the eye appears normal, there being no ciliary injection nor 'k. p.'. The virteous shows varying degrees of turbidity, some times large floaters and at others dust-like opacities. The affected area stands out as a swollen yellow mound with most of the fundus details in the immediate vicinity obscured. When near the disc papilloedema is mimicked very closely. Slit lamp funduscopy in such a case might show a flare, thus distinguishing it from papilloedema. An old case of choroiditis is characterised by atrophic, white areas fringed by brown-black pigment.

Diagnosis. One of the most difficult aspects of uveitis is the diagnosis of its cause. Though some diseases produce rather characteristic appearances such as are seen in leprosy, tuberculosis especially the miliary type, brucellosis and gonoccoccal arthritis, there are numerous instances when the aetiological agent appears elusive. Examination of the blood in syphilis, radiological exa mination of the short long bones in sarcoidosis and of the chest in tuberculosis are a few of the methods available in hunting down the aétiological agent. Complement fixation tests are of value in gonorrheoa and brucellosis, while skin sensitivity tests are of some use in determining sensitivity to staphylococci though not to streptococci.

Treatment of uveitis may be considered under two headings: *local* and *general*. For convenience the general lines of therapy will be considered first.

General treatment. Owing to the fact that hypersensitivity plays an important role in uveitis there are three broad principles on which treatment is launched:-

1. Eradication of infective foci in the body or the killing of the organisms.

2. Desensitisation by which the allergic response of the patient is lowered.

3. Enhance immunological response of the patient. These principles are embodied in Rich's Law which states that:

$$\text{The lesion} \propto \frac{\text{No. and virulence of organisms} \times \text{Allergy}}{\text{Resistance}}$$

1. Eradication of systemic infection involves attention to foci in the tonsils, teeth, sinuses, appendix, gall bladder, prostate, cervix uteri etc. This indeed is a Herculean task both for the patient and doctor and very often needless sacrifice. It is obvious that this line of therapy is a shot in the dark.

2. Extermination of bacteria by the use of antibiotics and chemotherapy is really efficient nowadays. The acute infections respond better than chronic ones. Specific drugs are anti-tuberculous, anti-syphilitic, anti-leprotic and amoebicides. To these must be added 'Daraprim' which is of value in toxoplasmosis.

Desensitisation has been generally disappointing. When different strains of an organism exist the vaccine must be from the proper strain. The dosage should be adjusted to avoid allergic reaction and the treatment continued until desensitisation is complete. Treatment should commence with small doses.

Reduction of the hypersnsitive reactions can be effected with cortisone and ACTH. Though these agents are not curative they have a place in ocular therapeutics for by blocking hypersensitive reactions much of the destructive phases of uveal inflammation can be cut down.

3. Enhancing the immunity of the patient is a difficult task. One of the measures adopted very often is the administration of shock therapy with the aid of foreign proteins. The mode of action is not known though it is thought now that the 'protein shock' acts rather like cortisone by stimulating the adrenal cortex.

Local Treatment is of importance in anterior uveitis and hardly has a place in posterior uveitis. Atropinisation to obtain maximal pupillary dilatation is the chief aim in order to prevent or free iritic adhesions. A watch should be kept on the intra-ocular tension and diamox given parenterally if there is a raised tension. *Atropine should not be stopped* but rather other anti-glaucoma measures should be adopted. A pracentesis or a four point iridotomy may have to be performed to avert any calamity resulting from such a secondary glaucoma.

Cortisone applied topically would do much to allay the inflammatory reaction. Hot bathing may have to be resorted to in cases with much congestion and pain. A leech or two to the temple may bring relief in some cases.

In cases where despite all efforts the iris gets plastered to the lens capsule or where the pupil is occluded an iridectomy should be done to restore communication between the anterior and posterior chambers.

SOME AFFECTIONS OF THE ELDERLY

119. Cataract is the term applied to an opacification of the lens. In essence the changes in the lens involve a precipitation of its proteins, initiated by an acidity of the lens, an imbibition of water followed by an alkalosis with precipitation of calcium in the lens and disappearance of essential substances such as vitamin C and glutathione from the lens rendering it asphyxiated.

Classification :-

A. Developmental—lamellar
B. Physiological—senile
C. Pathological
   1. Diabetic
   2. Endocrine
      a. Aparathyroidea tetany
      b. Myotonia dystrophica
      c. Mongolism
      d. Cretinism
      e. Dermatogenous
   3. Toxic
      a. Dinitrophenol
      b. Ergot
      c. Cachexia—malaria
   4. Complicated Cataract
      a. Corneal ulcer
      b. Iridocyclitis
      c. Choroiditis
      d. Retinitis pigmentosa
      e. Tumours of the retina and choroid
      f. Retinal detachment
      g. Glaucoma
   5. Traumatic
      a. Concussion
      b. Penetrating
      c. Foreign bodies (copper catract)
   6. After Cataract

Symptoms. At first there is increasing difficulty in reading with the presbyopic glasses and no improvement can be effected with fresh lenses. On account of an index myopia some presbyopes are able to discard their readers and engage in close work for some length of time—second sight. Later as the cataract progresses this ability passes off. There is day blindness owing to pupillary constriction in bright day light so that even a small lental opacity if in line with the pupil will cut off much of the light entering the eye. In subdued light the vision improves as with a dilated pupil light is able to enter the eye past the lental opacity. For this reason patients usually shun the glare and complain bitterly. Drivers of vehicles are badly handicapped at night by the glare of the oncoming vehicle. Even with a fully mature cataract a patient is usually able to see vaguely figures and objects and is therefore not so blind as another with advanced glaucoma.

Signs. Reduction of visual acuity is the main symptom. The reduction is most marked in the central lental opacities situated posteriorly (cupuliform) even if small owing to the interception of light at this, the nodal point.

By oblique illumination with a dilated pupil it is ofien possible to discern an opacity of the lens. The opacity may be posteriorly placed in the axis of the lens—cupuliform cataract. It may extend towards the centre from the periphery in the form of small wedge shaped intrusions into clear lens—cuneiform cataract. By transmitted light such a cataract in the early stages gives the appearance of a cartwheel the 'spokes' appearing black while the intervening areas appear red. In

the nuclear type the opacity is confined to the nuclear zones of the lens. By transmitted light such an opacity may stand out as a disc varying in colour from a dirty pink to a grey. When the fundus is viewed with a direct ophthalmoscope the optic disc or other fundus detail exhibits marked parallactic displacement. Oblique illumination imparts an amber colour to the core of the lens.

In cases of hypermaturity the cataract presents a milky white colour with a faintly brown nucleus at the bottom of the lens. At times a cataract takes on a blackish colour—the so called black cataract. The method of distinguishing between a total vitreous haemorrhage and a black cataract is discussed in § 11 a. There are two methods of finding out whether a cataract is mature. On oblique illumination if the iris casts a shadow on the lens and if on indirect ophthalmoscopy a fundus reflex is still obtainable with the patient's eye directed in various axes, then the cataract is immature.

Treatment. In a percentage of cases in the early stages the increasing index myopia may be corrected by concave lenses. As glare is most annoying to patients with central lental opacities tinted lenses often give relief and owing to slight pupillary dilatation brought on by the tinted glasses vision improves to an appreciable degree.

The prolonged use of mydriatics to achieve pupillary dilatation to enable the patient to see past a central opacity is strongly deprecated owing to the risk of glaucoma. This risk is all the greater in the age groups usually afflicted with cataract.

There is no medical treatmeut for cataract; neither drops nor injections will help to arrest or resolve a cataract.

The only rational treatment for cataract is surgical. Following surgery special aphakic glasses will be necessary. Owing to the fact that the magnification afforded by an aphakic eye corrected with glasses is about 33% a monocular aphakic will not be able to use cataract glasses as long as the companion eye has useful vision. If a contact lens is worn on the aphakic side the size of the image is 11% larger than that produced by an emmetropic eye. This appliance therefore would permit a monocular aphakic to use the operated eye with the companion eye. The best solution is of course Ridley's operation of the introduction of a plastic lenticulus into the eye or the anterior chamber lenticulus insertion devised by Strampelli.

An unattended cataract may lead to certain complications which may end in much suffering and also rendér any surgery on such an eye a most hazardous process. These are (a) spontaneous dislocation of the lens due to zonular degeneration and (b) secondary glaucoma.

Method of Ascertaining Operability

In patients who have attended an institution from the early stages of a cataract, it is possible to study the fundus but in the majority of patients such opportunities do not arise and so reliance on other tests is essential to arrive at a decision as to the suitability of a case for operation.

These are:-

1. Light perception at 5—6 metre distance
2. Light projection

3. Intact pupillary reactions
4. Colour perception—red and green glasses are alternately placed before the eye in question, the other being occluded, while the patient is gazing at a light.
5. Ability to see the 'line' when a Maddox rod is placed before the eye being tested.
6. Electroretinogram. This is not yet a routine clinical procedure and is not infallible.

120. Glaucoma is predominantly an affection of old age though infants, children and adults may succumb to it. From a green appearance of the eye due to water logging of the cornea and enhanced by sclerosis of the lens rendered more apparent by a dilated pupil the disease received its apellation. The suspicion that it was due to a 'humour' in the eye was later at least found to have some basis in as much as the disease in many instances was found to be associated with a raised intraocular pressure. *The raised intraocular pressure is incidental and not causative. On the other hand glaucoma can exist without a raised intraocular pressure.*

To this must be added some gleanings from new knowledge on the formation and elimination of aqueous. Suffice it to say that there is a region of inflow of aqueous extending from the ciliary body with a maximal head of pressure, to the choroid in its mid-zone and the retina in the same zone. Outflow of aqueous occurs through the angle of the anterior chamber, canal of Schlemm and the episcleral plexus of veins—the aqueous veins being channels of communication between the last two.

Increase in inflow as a factor in the causation of glaucoma is not accepted by all whereas both on experimental and clinical evidence it is accepted that the outflow is hampered in some types of glaucoma. Another vital factor in the aetiology of glaucoma is an abnormality of the circulation. Sclerosis of the blood vessels supplying the optic nerve and the retina causes a slow loss of vision with cupping and atrophy of the disc and field loss. Sclerosis of the trabeculae in the angle of the anterior chamber may add an element of impediment to the outflow of aqueous and cause in addition to the above an elevated tension.

Primary glaucoma is divisible into (a) closed angle glaucoma and (b) simple glaucoma.

Closed angle glaucoma is met with in oversensitive, hypher-excitable and highly-strung individuals and is characterised by an episodic and violent attack of ocular pain, misty vision, haloes and a markedly raised tension. It is not associated with field defects or cupping of the disc in the early stages.

In its causation an impeded and hampered outflow plays an important role, usually in an eye with a shallow chamber though not invariably so, brought about by angle closure by the approximation of the root of the iris to the extreme periphery of the cornea. This is effected by a physiological pupillary block or seclusion. As stated above the chamber is shallow with the iris-lens diaphragm lying forward; in a state of semidilatation of the pupil the pupillary border impinges on the lens while the rest of the iris bows forward in the form of an iris bombé. The pressure in the posterior chamber mounts and jams the iris root against the cornea effecting thereby a hampered drainage.

Many authorities attribute this chain of events to a structural derangement but some (Duke-Elder) are of opinion that the underlying cause is vasomotor instability which is responsible for periodic rises of pressure within the small vessels of the eye, specially in the ciliary body, resulting in excessive inflow.

The diagnosis of this type of glaucoma is easy especially in the phase of raised tension. In the intervals practically every aspect of ocular physiology and function (as far as is ascertainable at present) is normal.

However for an early and accurate diagnosis of this condition the following points should be borne in mind:-

1. Careful ocular history
2. Complete clinical examination of the eye including gonioscopy
3. Provocative tests.

Simple Glaucoma is not preceded by premonitory symptoms nor is its course violent. It is characteristically insidious in onset, slowly progressive and exhibits a classical triad of signs—field defects, cupping of the disc and raised intraocular pressure. Though at first the tension has a phasic variation it becomes permanently raised later. The tension of course may be normal. It does not affect persons of any particular phychological make-up nor are the affected eyes typically of any anatomical configuration or show any particular refractive error.

In its causation vaso-sclerosis preced by vaso-instability appears to of prime importance. Vaso-sclerosis of the posterior segment would give rise to cupping of the disc and field defects with a normal intraocular pres-

sure—the so-called soft or low-tension glaucoma. From the appearance of the optic nerve under the microscope this is termed lacunar or cavernous atrophy. If the sclerosis affects principally the anterior segment of the eye there would be raised tension while if the whole eye is affected there would be cupping of the disc, field defects and raised tension.

In the diagnosis of this condition in the early stages reliance must be placed on the following:-

1. The diurnal variation curves
2. Perimetry and ophthalmoscopy
3. Outflow tests—tonography and water-drinking test.

Regarding the diurnal variation a fluctuation of more than 5 mm. Hg. is suggestive of glaucoma. The priscol test may be employed (§ 22), for it is a measure of vaso-instability.

In ophthalmoscopy special care should be exercised in looking for early signs of cupping which manifests itself as a slight dip classically in the lower temporal segment.

Perimetry must be directed towards spotting an early nasal step, while campimetry (§ 10, ii) should explore the central field for a depression of the upper field, nasal step and for scotomata in the 10° — 20° circles. The target used should be 2 mm. diameter and the distance 2000 mm. while the illumination should be dim.

A simple method of tonography is to rest a Schiotz tonometer for four minutes on the eye, readings being taken on first implanting the instrument on the cornea and at the end of four minutes. The diffe-

rence in the readings is calculated as a percentage of the first value. In a normal eye the reading should be 30 per cent or more.

The water drinking test is a good provocative test as it is an index of the efficiency of the drainage channels.

Early Diagnosis

One should pay heed to the following symptoms:-

1. Complaint of seeing haloes.
2. Rapid changing of glasses (presbyopia)
3. Defective dark adaption
4. Headaches on being in the dark

One should suspect glaucoma under the following circumstances:-

1. In those who have the affection in one eye.
2. Those with a family history.
3. In myopes who go downhill despite a healthy macula.
4. Those in whom the intraocular tension is raised above the normal level—for this one reading is useless. Admission to hospital for 24 hours at least is necessary and a tension curve should be plotted.
5. A swing of more than 6. mm. Hg. in the diurnal variation during a period of 24 hours.
6. A difference of more than 5 mm. Hg. in the tension of the two eyes at any given time.
7. In those with vascular glaucoma in one eye.

Treatment

This is a controversial subject but there are some basic principles which must be adhered to.

A. *Closed angle glaucoma.*

1. Elimination: purge or retention enema of 50% magnesium sulphate solution 6 ounces.
   Diuretics especially acetazoleamede should be given. 250 mg. twice daily may be given at first. This drug has a special virtue in that it reduces inflow of aqueous.

2. Restrict the intake of fluids, coffee, beer or any large drink.

3. Sedation. Drugs for pain and sleep should be freely administered. If morphia is being given it should be given free of atropine.

4. Local treatment consists of frequent application of miotics: eserine ¼% to 1% may be used as frequently as ½ hourly in an acute case. Pilocarpin 1% or even 2% may be employed alone or combined with eserine. Adrenaline could be used as drops in addition with benefit. As the severity of the illness begins to wane, the frequency of application may be reduced.

5. Hypertonic saline intravenously may help to reduce tension though its effects are temporary.

   If the tension does not drop and the eye quieten down in 24—48 hours, resort to surgery must be had.

6. Surgical treatment aims at reducing the inflow or facilitating the outflow. The former is achieved by *retrociliary diathermy*. As most of the cases appear to have an increased resistance to the outflow of aqueous, surgical measures should be directed to reduce the resistance. A small peripheral *iridectomy* or even an *iridotomy* is held

to be effective by some. More usually the classical broad glaucoma iridectomy is favoured by many. Limitations to these two methods are the presence of peripheral anterior synechiae and chronicity. If operative treatment is being planned on an eye that has had an attack for over 24 hours or on one that has peripheral anterior synechiae one of the filtration operations will have to be considered. Either Elliot's corneo-scleral trephining or iridencleisis should be done, the former preferably in a quiescent period.

B. Simple Glaucoma.

The greatest caution and judgment are required in the treatment of simple glaucoma. The important considerations are the tension, resistance to outflow and field changes. In view of the fact that sclerosis of the blood vessels plays an important role it would be rational to treat the patient on medical lines provided the tension is normal, the fields not badly affected and the outflow reasonable. In fact according to some authorities the appearance of scotomata is a contraindication to surgery for these scotomata are evidence of vascular defects. In such cases intensive vasodilator therapy would appear to be preferable to surgery which is useless.

If the tension is normal but the resistance to outflow great then clearly surgery is indicated. The type of operation will of course be of the filtering variety. Post-operatively it is necessary to perform outflow tests to ascertain the efficacy of the operation. If the outflow is poor miotics should be continued.

PROPHYLACTIC SURGERY

It is the opinion of some surgeons that a prophylactic operation on the fellow eye in a person with glaucoma in one eye is a sound step. For instance in a case of closed angle glaucoma a small peripheral iridectomy in the companion eye may forestall an attack. When pre-planned such an operation can safely be conducted at a time when the eye is not in a critical phase.

121. Secondary Glaucoma is similar in its symptomatology to closed angle glaucoma, with severe pain, congestion of the eye and attended by general symptoms such as vomiting and collapse. Unlike in primary glaucoma the causes of this condition are known:-

1. Trauma
2. Corneal ulcer
3. Iridocyclitis
4. Intumescent cataract
5. Intraocular tumours
6. Intraocular haemorrhage
7. Retinal detachment
8. Venous thrombosis (retinal)
9. Capsular exfoliation

Pathogenesis of secondary glaucoma is well understood in most instances. Trauma is accompanied by an unstable tension for sometime ending either in hypotension or hypertension in a proportion of cases. Often there is severe damage within the eye such as a dislocation of the lens, tears of the iris, venous thrombosis, capillary paralysis or haemorrhage. In iridocyclitis, intumescent cataract and tumours the tension goes up owing to hampered drainage of aqueous. In detached retina and central venous thrombosis osmotic pressure differences contribute a share. Capsular exfoliation causes mechanical hindrance to the outflow of aqueous at the angle but this view is not favoured by some opthalmologists. *Symptomatology* will include in addition to what has

already been detailed under closed angle glaucoma, features peculiar to the primary condition which caused the glaucoma.

Treatment will naturally be directed to the primary disease. The intensive miotic regime outlined for closed angle glaucoma must be instituted early. Acetazoleamide by mouth supported by adrenaline and eserine drops in the eye will help. Retrociliary diathermy is ideal in many of these cases. In post-operative cases cyclodialysis is called for, this being ideal in cataract and keratoplasty cases. In iridocyclitis atropine may be continued as it is essential in these to keep the pupil dilated and free of adhesions.

Sedation is essential as is elimination. It is hardly necessary to state that all operations on such eyes must be under a general anaesthetic.

122. Mydriatic Glaucoma is an iatrogenic condition given by a doctor to his patient unwittingly or negligently. The modern era is one in which physicians, neurologists and students are beginning to look into the eye on a much larger scale than ever before. Apart from eye men who are alive to the dangers of mydriatic glaucoma the other medical men do not appreciate the actual dangers attending the use of mydriatics for ophthalmoscopy.

It is specially described under a separate heading to stress the importance of the condition.

In a person who has no semblance of glaucoma at all, within a few moments of instilling a mydriatic acute symptoms suddenly appear, of pain, redness, vomiting and blurring of vision. The effect of the mydriatic in dilating the pupil is enhanced by keep-

ing the patient in the dark (for purposes of examination) Of interest to ophthalmologists are the provocative. tests that combine mydriasis and dark adaptation to detect an incipient glaucoma. This test is positively dangerous and should not be undertaken lightly (see § 16, 20, 21).

In every instance when a mydriatic is employed in the eye for examination purposes it is imperative that a miotic be instilled immediately. In any case where even the slightest symptoms suggestive of acute glaucoma appear strong miotics should be instilled frequently, keeping a check on the tension tonometrically, till the tension returns to normal. It is even expedient to admit such a patient into an institution and to treat him as a case of closed angle glaucoma not forgetting that eventually surgery may have to be resorted to if the tension is not controllable medically.

123. Retinal Detachment or separation of the retina is the term reserved for a condition in which the neural layers of the retina part company with the epithelial layer and is characterised by a dramatic obscuration of vision. Such a separation is called idiopathic detachment as opposed to secondary detachment which is due to some local or general cause. As there are sharp distinctions between these two types, aetiologically, clinically and prognostically it is essential to study them, as finally treatment depends entirely on the type of detachment.

TABLE III. Detachment of the Retina

	Primary	Secondary
Causes	Myopia Senility Trauma	Medical: Diabetes, Hypertension, Nephritis Obstetric-Toxaemias of pregnancy Tumours: Retina, Choroid
Pathology	Retino-vitreal adhesions, tears, separation of the two embryonic layers	A mass or transudate in the chorid pushes the retina forward (both layers)-*pulsion*. Fibrovascular tissue from within the vitreous can pull off the retina-*traction*.
Features	Muscae, photopsiae Hole or tear Markedly undulated Migration Lowered tension No new vessels	No muscae No hole Less undulatory * No migration Tension raised often* New vessels*
Treatment	Surgical: Principles are :- 1. Seal the tear 2. Evacuate subretinal fluid	Treat cause

* These are seen in tumours ; new vessels may also be encountered in vascular retinopathies with detachment.

Having reviewed the features of the two types it is only necessary to deal with the idiopathic detachment hereafter.

Idiopathic Detachment

Symptoms will depend on the site and extent of the detachment and on the powers of observation of the patient. Flashes of light, muscae and distorted vision may be precursors in some cases while a sudden 'curtain' or 'mist' may obscure vision in some.

Signs can be elicited by ophthalmoscopy. By the distant direct method the fundus reflex is not uniformly red but in part or whole there is a grey-white reflex. The refraction may be hypermetropic even if the patient was myopic. This is due to the forward displacement of the retina. In viewing the fundus by the direct method therefore a + lens has to be inserted into the peep hole. The detached area looks greyish with numerous folds over which black blood vessels course—the latter look black owing to the fact that they intercept light returning from the choriocapillaris. A hole or tear is visible in some part of the detached retina. The tear may be round, arrow-head shaped, irregular, single or multiple, small or giant and appears red. Near the ora a dialysis may occur.

Course. Usually a detachment tends to spread especially if it starts in the upper part. If not attended to, the fluid behind it gravitates to the bottom and the lower area detaches while the tear is above—hence a moral: *always look above in lower detachment if a tear is not seen below.* Further spread will end in a total detachment leaving the retina anchored at the ora and the disc, the only two points at which it is firmly fixed.

Diagnosis. Allusion has been made to this already. Once in a way a detachment may present itself with a vitreous haemorrhage if the tear occurred across a blood vessel. In such cases one has to await clearing of the vitreous before ophthalmoscopy can be of help. This brings us to another moral: *never permit a patient with a vitreous haemorrhage to walk about until one is satisfied he has no detachment of the retina.* *Prognosis* depends on duration of the detachment, shorter

the better prognosis; on site and extent of the tear, single small holes carrying a better prognosis than multiple or giant tears.

Treatment as already outlined consists of sealing the tear and evacuating the subretinal fluid. In order to seal the tear the localisation must be accurate and an adequate examination in the dark room under full mydriasis cannot be over-emphised. A drawing is indispensable. Indirect ophthalmoscopy with a large concave mirror similar to a frontal mirror and binocular indirect ophthalmoscopy are gaining in popularity.
If a tear is not detected postural treatment should be adopted permitting the retina to settle down a little so that a tear that was hidden by a fold comes into view.

Postural treatment is a *must* in upper detachments and instead of raising the foot of the bed and making it miserable for the patient it is advisable to double-pad the eyes and permit a flat position in bed with a pillow. The eyes roll up in a double-padded state and so the detachment would now be in a dependant position.

Weve's diathermy operation is now standard but much progress has been made in detachment surgery in recent times. In order to seal the tear more effectively resection of the sclera was started so that a chemical or diathermy sealing could be brought nearer the torn retina. As full thickness resection is a hazardous process lamellar resection was advocated by Shapland. A further improvement and at the same time a simplification of this operation is now the scleral buckling operation born of resection and the reefing operations of the sclera.

As retraction of the vitreous is a concomitant of detached retina a buckling of the sclera approximates the latter to the detached retina, the infolded sclera acting as a prop or rib to which the retina can easily get anchored.

A very novel method of sealing a tear is being tried at the movement with light which is admitted through the pupil and directed on to the tear at which the surgeon is gazing with an ophthalmoscope.

Though a high percentage of repositions is claimed by experienced surgeons the prospects in retinal detachment are gloomy; as one surgeon put it "detached retina is like a torn rag, you mend it and it tears again somewhere else". As vascular sclerosis and tissue necrosis are responsible for the initiation of a tear the scope for surgical measures appears to be limited.

124. Macular Degeneration is a cause of blindness in old age though in Ceylon it does not play an important role either in the morbidity or the blindness rates. *Senile macular degeneration* is due to sclerosis of the choriocapillaris with consequent degeneration of the overlying retina characterised by pigmentary and exudative changes, the latter assuming a discoid mound shape and giving it the name of *disciform degeneration* of the macula. The condition is usually bilateral and is resistant to treatment.

Symptoms include metamorphopsia, micropsia or macropsia in the early stages followed by a slow and steady loss of central vision. In the initial stages examination with Amsler's charts may reveal the existence of disorted vision before ophthalmoscopically visible changes occur.

Treatment is of little avail. Systemic check up may help. Telescopic spectacles will serve as a useful aid in the early stages at least. Cortisone has been tried with equivocal results. Cervical sympathectomy appears to be of some value in selected cases. It has been remarked that in the tropics owing to the greater prevalence of cataract in the old, macular degeneration has a lower incidence suggesting that the cataractous lens shields the macula from the injurious rays of the sun. If this be true there is something to be gained by wearing tinted glasses as a protective measure in all countries where the glare is excessive,

125. Dacryocystitis is an inflammation of the lacrimal sac induced by an obstruction of the nasolacrimal duct. Reference has already been made to this in the newborn (§ 93). The duct obstruction may be partial at the start and become complete later. Owing to the blocking, mucus and tears collect in the sac giving rise to a swelling—*mucocele* which can be emptied by pressure over the sac. In this enviroment organisms multiply and cause a chronic inflammation—chronic dacryocystitis. Fibrosis and sacculation go on and after repeated attacks of inflammation the canaliculi and puncta get stenosed. At this stage emptying of the sac does not occur and with increasing tension of the contents of the sac ulceration of the sac wall occurs. The inflammation now spreads to the perisaccular tissues causing a peridacryocystitis which is acute. This is the stage of acute dacryocystitis which may lead to abscess formation if neglected.

One of the worst complications that may befall a person with chronic duct obstruction is ulceration of the cornea.

Treatment depends on the extent of the obstruction. If partial (which can be diagnosed by syringing the sac) repeated weekly washing of the sac with a nasal pack of cocaine and adrenalin and the use of a nasal decongestant at home might help. Nasal pathology requires attention.

If the obstruction is complete, operative treatment is the only choice. Extirpation of the sac, anastomosis of the sac to the nose and intubation of the nasolacrimal duct may have to be given a trial according to the nature of the case and the surgeon's fancy.

In the case of acute dacryocystitis analgesics to allay pain, antibiotics to combat infection and local hot applications would relieve much suffering. Incision of the abscess would become necessary in any case which resists the usual line of therapy.

126. Ocular Tumours of importance at this age are epibulbar and intraocular. Of the first, malignant papilloma of the limbus and carcinoma of the limbus are common. These present a sessile mushroom appearance and bleed readily on manipulation. They lie astride the limbus, a part on the cornea but not invading it while the rest lies on the conjunctiva. Spread is in three directions, one towards the cornea, the other towards the canthus and the third along the limbus.

These tumours are of low malignancy owing to the fact that they cannot invade the cornea because of its compact lamellar arrangement and because of the paucity of lymphatics.

Of *intraocular tumours* choroidal melanoma and metastatic deposits are the chief. For purposes of clinical description the stages of an intraocular tumour are

divided arbitrarily into three (1) stage of intraocular spread, (2) stage of glaucoma, (3) stage of extra-ocular spread.

Symptoms of intraocular tumour. In the first stage slight visual symptoms may be complained of. The fundus may show a retinal detachment without a tear of a dark brown colour with new blood vessels on its surface. There may be haemorrhages on it and by transillumination a shadow is seen. Gradually increasing field defect with sharp borders is characteristic of a detachment due to a tumour.

Prognosis depends on the type of cell that composes the tumour and on the amount of reticulin. The pure spindle-celled tumour carries the best prognosis and the epithelioid cell the worst. The more reticulin there is the better the prognosis, the reticulin acting as a barrier.

Secondaries usually appear in the liver and the time lapsing between the appearance of the primary and the secondary may vary and the survival rate again depends much on the nature of the growth and the duration the growth has remained undetected.

Treatment is excision of the eye. Orbital involvement requires exenteration and intracranial spread via the optic nerve is usually beyond surgery. X radiation is in effecive though radium plaques have been tried by some surgeons.

OPHTHALMIC EMERGENCIES

127. Injuries of the Eye. The eye is housed in a bony socket, cushioned by fat and protected by rigid bony margins on all sides excepting temporally where the receding margin exposes the globe. The lids with their lashes act as sentinels triggered by a very efficient axon reflex system operating through the trigeminal nerve. The response to a warning signal is of a triple kind, lacrimation. closure of the lid and upward rotation of the globe. Were man today as primitive as he was in ages gone by the above mentioned protective mechanisms would have sufficed but with so many hazards threatening modern man these devices are feeble indeed.

Though the prognosis in quite a good proportion of eye injuries is poor a number of eyes can be saved by prompt, energetic and sometimes simple treatment of a first aid nature.

128. Contusion of the eyeball results from injury with blunt weapons such as a fist, club, door-knob, ball, umbrella-end or walking stick. The resulting lesion may range from the ordinary black eye to rupture of the globe. Following are some of the conditions met with :-

Laceration of the cornea	Dislocation of lens
Conjunctival ecchymosis	Retinal detachment
Tears of the iris	Rupture of choroid
Retroflexion of the iris	Glaucoma
Cataract	Commotio retinae

Rupture of the globe

The damage to the eye will depend on the force of the violence, the direction of its application and the du-

ration of impact modified by the acceleration of the head in space. The same force applied to the orbit in a head that is free to move and in another that is fixed (say against a wall) will cause more damage in the second instance. The size of the missile is of equal importance, for any object that is larger than the orbital aperture will not damage the globe. A third factor one must remember is that if the force of the violence is not spent on the surface or anterior segment of the eye, the posterior pole suffers by *contre coup*, though the globe is intact.

A force that is directed antero-posteriorly causes an axial compression at the same time that there is equatorial expansion, a most favourable situation for dislocation of the lens, tears and dialysis of the iris. Retroflexion of the iris could occur together with an anterior dislocation of the lens. If the force is directed from below and laterally the globe is usually pushed against the trochlea of the superior oblique which is ossified or against the spine to which the trochlea is attached. The point of impact is the Canal of Schlemm, a weak spot on the globe, at which rupture usually occurs.

Tears of the iris may also occur as a result of the momentary impact of the iris against the lens (on which it leaves its imprint—Vossius ring) caused by an indentation of the cornea on receipt of the injury. Opacification of the lens may arise as a result of the contusion but it is unlikely that such opacity can be initiated without a capsular tear which is not visible either because it is fine or is situated equatorirally. Capsular opacities, lental opacities and capsulolenticular opacities occur, the most characteristic of which is the

rosette-shaped cataract occurring in the posterior cortex. Commotio retinae better described as Berlin's Oedema is due to severe capillary paralysis, a choroidal haemorrhage follows resulting in pressure ischaemia of the overlying retina. The latter in a classical case presents the same appearance as a case of central retinal arterial occlusion. In other less severe cases the retina appears milky and swollen. The visual acuity is slightly off normal but latter drops as pigmentary and even cystic degeneration of the macula with whole formation ensues.

Retinal detachment as a result of contusion is rare unless there is a predisposition to it. Often the choroid ruptures, showing up as white crescnetic areas with the concavity towards the disc.

Treatment. Sedatives to lessen suffering moist compresses locally and careful watching are all that can be done. In tears of the iris atropine should not be used. If a hyphaema is noticed, the tension of the eye should be checked for if it is high, blood-staining of the cornea may occur. In such cases atropine should not be used, a paracentesis may be urgently required and the exhibition of acetazoleamide a wise step. The last remark would also apply in the case of traumatic glaucoma unaccompanied by intraocular haemorrhage. In commotio retinae dark goggles, calcium, subtenon cortisone and vasodilators may be given a trial as the condition is sometimes desperate.

Detachment of the retina and rupture of the globe require surgical attention. In the case of the latter no eye should be considered too bad for repair for it is surprising what function can return with appropriate *debridement* and suturing.

Antitetanus serum may be called for in some cases. Lental opacities do not require urgent surgery unless the whole lens is opaque and swollen so as to cause glaucoma. In such a case curette evacuation of the soft cataract is a procedure that must be adopted early. In the elderly an extraction may have to be attempted under a general anaesthetic with a relaxant.

A careful watch for early signs of sympathetic ophthalmitis must be maintained. A guarded prognosis is the wisest counsel in any eye injury, however trivial it may seem.

129. Penetrating Wounds may involve any part of the eye but most commonly the anterior segment suffers preferentially. The cornea, the sclera and the corneoscleral junction are the usual sites. Any of these may or may not be accompanied by prolapse of uvea.

Syptomatology. Severe pain, lacrimation and loss of vision follows the impact of the agent causing the wound. Lid spasm and photophobia are present according to the nature of the wound. Inspection reveals a slight flattening of the globe visible through the upper lid. On opening the lids the wound is seen; the lips are swollen and look grey-white owing to imbibition of water. A black mass caught in the lips of the wound is uveal tissue, iris if the wound is corneal or corneoscleral or ciliary body if the wound is further back. The anterior chamber is shallow and the pupil drawn towards the wound. These features may be masked on occasions by a hyphaema. The tension is low and visual acuity is diminished. In deeper injuries the lens may be cataractous; in some the soft lens matter may be seen to be running out through the wound. In corneoscleral and scleral wounds

vitreous may present in the wound. In the worst type of case the site of the wound is literally a 'mess' consisting of swollen lacerated cornea, blood, lens, uvea and vitreous which get matted in the delayed case one often sees in this country.

Complications are infection—endophthalmitis or panophthalmitis, sympathetic ophthalmitis from all of which the eye may be lost. In other cases anterior synechia results with distortion of the pupil and astgmatism; in such cases the eye remains irritable for long periods.

Treatment of penetrating wounds is of the utmost urgency. Debridement and direct suturing of the wound are imperative. Prolapsed uveal tissue is excised cleanly and the wound sutured using interrupted silk sutures through the lips. A conjunctival flap may be used to cover a wound if a sufficiency of the former can be found and if direct suturing cannot be done. Anterior synechiae should be divided if the eye does not quieten down early or later if vision is being interfered with. Cataract may be dealt with by discission or curette evacuation.

Corneal scars if central require optical iridectomy of keratoplasty for visual reasons.

Excision of the eye is required in cases of severe damage accompanied by loss of much vitreous and collapse of globe, uncontrollable bleeding, retention of a large foreign body and in sympathetic ophthalmitis (see later).

130. Foreign Bodies in the Eye. (a) *Surface*:- Under the upper lid; unilateral lacrimation and conjunctivitis with a history of some foreign particle flying into the eye are the features. Eversion of the upper lid will

reveal the foreign body which can be removed with a wisp of cotton. Wings of insects and ants cling on to the mucosa firmly and have to be removed with a spud or forceps.

Corneal foreign body causes much lacrimation and pain. The reflection of a window or light is uneven suggesting a loss of polish and an elevation at the site of the foreign body. In metallic foreign bodies there is usually a ring of corneal infiltration round them after a few days of neglect. Ulceration may commence in the absence of treatment.

Removal of a foreign body on the cornea must be done under cocaine anaesthesia. A 4% solution is instilled at frequent intervals for ten minutes. The lids are held apart with the finger and thumb of one hand, the thumb pressing on the globe so as to steady it and prevent it from moving. With a spud or needle the foreign body is picked up. Antibiotic drops and homatropine drops are instilled and the eye bandaged. *No person from whose eye a corneal foreign body has been removed should be sent away without a bandage.* This will be appreciated if it is realised that :-

1. the foreign body has damaged the cornea
2. cocaine causes drying and desquamation of the corneal epithelium
3. instrumental removal of the foreign body causes further damage to the epithelium
4. an unbandaged eye under these circumstances is painful and can get infected; epithelialisation is quicker when the cornea is splinted by the lids.

No medicament containing cocaine or other surface anaesthetics should ever be prescribed to a patient with a corneal foreign body with the idea of allaying pain. This will certainly allay pain but it procrastinates proper treatment being taken. This applies whether the foreign body has been removed or not.

(b) *Intraocular* foreign bodies may enter the eye through the cornea, corneosclera, sclera, anteriorly or posteriorly. They may reach the deeper parts of the eye through the pupil, iris, ciliary body or choroid, in the first two damaging the lens and in the latter two missing it. Depending on the force with which they are driven in (velocity) they may lodge in the iris, lens, vitreous, retina or may even reach the sclera. On occasions a foreign body is ricocheted twice or thrice within the eye.

Symptoms may be so slight as to escape notice or in the presence of intraocular haemorrhage or a fast forming lental opacity visual loss may be great and dramactic. Pain is usual with injuries of greater severity especially if there is prolapse of uveal tissue

Signs. There may be no indication of any trouble in some cases, not even the site of entry can be seen as the latter may be in conjunctiva or may be masked by a subconjunctival haemorrhage or may be so far back on the sclera that it is undetectable.

The tension is often low for in anteriorly placed penetrations the aqueous and in posterior penetrations the vitreous escape. The anterior chamber, if clear will be collapsed or may show a hyphaema. The foreign body may be within the anterior chamber. Vision will be affected in proportion to the damage

done to the transparent media immediate or delayed and in accordance with other complications such as haemorrhage, detachment of the retina and siderosis (if the foreign body is iron) or chalcosis (if the foreign body is copper).

Foreign matter such as eyelashes, bits of skin and vegetable fibre may be in the wound depending on the circumstances of the accident.

Molten metal and certain missiles may strike the lid first in which case there will be corroborative evidence in the form of a burn or a peforating wound in the lid. This brings to light a golden rule that *in all injuries of the lids it is absolutely essential that the globe be inspected, if the need be under an anaesthetic.*

Complications. Some foreign bodies such as porcelain, stone, concrete, glass, alluminium and alloys remain inert and do not cause much chemical reaction. Particles of chemicals used for mannfacturing crackers and for blasting rocks are very irritant. *Iron* produces a condition called siderosis in which there is a rusty deposit in the cornea, lens, iris, ciliary body and retina ending in a slow loss of vision—rather following the pattern of visual disturbances of retinitis pigmentosa. Copper if of some size produces a violent aseptic chemical inflammation which only stops with the complete destruction of the eye. If small it causes other characteristic changes namely a coloured ring at the limbus—Fleischer's ring—and a lental opacity in the form of a sun flower.

Diagnosis. From the history it is possible to suspect an intraocular foreign body a proof for which can be

obtained in the case of radio-opaque substances by X-ray examination. Simplest of all methods is to take two shots with half exposures of the eye looking in two different directions. A foreign body within or on the coats of the eye will be duplicated. Localisation of the foreign body for purposes of removal can be done by very elaborate methods. Some of the more simpler ones will be mentioned.

1. *Scleral rings:* a steel ring with a bead is placed over the globe so that it slips into the fornices. With the eye looking up and then down two half exposures are made. From the known size of the ring used (24, 25 or 26 mm.) and the magnification produced by the X-ray plate the size of the body as well as its distance from the surface of the globe can be calculated.
2. *Contact lens* with radio-opaque markings. The principle is the same as the above.
3. *Sweet's bone free method.* A small dental X-ray plate is steadied against the inner canthus and with the X-ray tube on the lateral canthus slightly behind the plane of the lateral orbital margin an exposure is made so that only the soft tissues are included in the picture. Only foreign bodies in the anterior segment of the eye will be visualised by this method.

It is of some importance to ascertain the size of the foreign body for if it exceeds 3 mm. it should be removed through the posterior route.

Treatment. Magnetic foreign bodies should be removed with the giant magnet. The history may suggest the nature of the foreign body. However in doubtful cases an application of the giant magnet would at least give a clue. The magnet is applied under local anaesthesia so that in the case of magnetisable foreign bodies the

pull would cause pain. This is an indication for continued or repeated applications of the magnet. The chances of removing a foreign body are greater of course the earlier the attempt is made.

Non-magnetic foreign bodies if known to be inert may have to be left alone unless accurate localisation will allow a direct approach either from the surface of the globe or with a special 'endoscope' under visual guidance. It is obvious that for such manipulations the vitreous should be clear.

Antibiotics should be exhibited to prevent or curb infection. In the event of sympathetic ophthalmitis complicting an intraocular foreign body cortisone should be used topically as well as systemically.

Excision of the eye when it harbours a very large foreign body or if it is badly injured may have to be considered.

131. Chemical Injuries. Some of these are peculiar to Ceylon such as the latex of some plants, caterpillar ophthalmia (nodosum) and 'holy-ash' keratitis. The first of these causes a corneal burn, the second a violent kerato-conjunctivitis and the third a vesicular keratitis. All three cause much pain and suffering. Latex burns present a peculiar appearance as though the cornea is tanned especially if severe or neglected.

Treatment of these consists of getting rid of the offending material either by irrigation or by meticulous picking out of particles of holy ash or of caterpillar hair. Secondly the use of cortisone locally is of immense value in cutting down the inflammatory phase of the injury. Atropine is indicated if iritis is a complication.

Acid and Alkali Burns are of common occurence in this country being very often the result of assault. Acetic

acid is the most frequently used though hydrochloric and nitric acids are also employed. In the milder cases the lids and the lower part of the conjunctiva and cornea are affected as the upper cornea gets rolled under the upper lid at the first signal of danger. In extensive spalshes the damage done is not only widespread but also beyond repair. Acid as well as alkali burns occur in certain industrial occupations such as garages, soda-water and soap manufactories.

Prognosis is poor in the case of alkali burns and is favourable in acid burns. This is not dependant on the pH of the solution but on the affinity the agent has for the corneal protein. Acids have great affinity for corneal proteins with which they form insoluble albuminates; thus they cannot penetrate the anterior chamber. Desquamation of the corneal epithelium is not marked. Alkalis on the other hand form soluble albuminates with the corneal proteins with resultant easy penetration ; desquamation of the corneal epithelium and infection are common. Alkalis also cause hydrolysis of the corneal mucoid causing marked swelling. The inflammatory reaction is more severe in the case of alkali burns. Necrosis of the cornea, a state of affairs obtaining even in purely conjunctival and scleral burns is due to the ischaemia brought on by blanching of the circumcorneal vessels. An eye that looks dead white has a very bad prognosis though at the time of first examination the eye may look innocent. This is typical of ammonia burns.

Treatment is based on the following broad principles:-

1. Dilute the offending agent,
2. Get rid of the chemical,
3. Neutralise the chemical,

4. Combat infection,
5. Prevent complications.

Dilution of the chemical is best achieved by the use of copious amounts of water, irrigations being conducted for an hour at a time. In factories this is done very simply by water spouts attached to a wash basin.

Getting rid of the chemical is partly done by the above method and in cases of lime burns it is necessary after the wash to evert the upper lids and pick out the particles of lime, for these by prolonged contact with the cornea may damage the latter slowly but steadily to an irreparable extent. Repeated paracentesis of the anterior chamber is required if the chemical, especially ammonia, is to be eliminated from within the eye. Nasty iridocyclitis is a usual complication of chemical burns.

Neutralising the chemical is theoretically attractive but in practice it never works. *It is pointless looking for an antidote; this time could be better used for irrigating the eye.*

Infection should be prevented by the free exhibition of antibiotics both locally and systemically.

Complications such as symblepharon, iritis and necrosis of the cornea should in the first place be forestalled. Excision of the ischeamic conjunctiva and grafting with cadaver conjunctiva or mucous membrane from the mouth, labia or prepuce will help re-establish the circulation in the perilimbal area and prevent necrosis of the cornea. This as well as the grafting of amniotic membrane will prevent symblepharon. If amniotic membrane is used it should not cover the entire cornea but only 2/3rds of its surface. If am-

niotic membrane is not available it may be necessary to insert a contact lens so as to keep raw inflamed surfaces apart; or the old classical method of the glass rod smeared with lubricant in the lower fornix may have to be tried. Established symblepharon requires plastic operation involving keratectomy, keratoplasty and conjunctivoplasty. The prognosis regarding keratoplasty in cases of burns is poor especially if a good portion of the cornea is involved.

132. Sympathetic Ophthalmitis is the name given to a panuveitis affecting a normal eye in perforating injuries of the companion eye—the term being derived from the fact that the second eye sympathises with the injured eye. It is fulminating in onset, relentlessly progressive and ends in destructive changes after a violent and stormy course.

It occurs commonly in perforating injuries especially in wounds in the 'dangerous area' of the eye—viz., the ciliary zone, limbus and the outer zone of the cornea. Operative trauma and foreign bodies, more especially the retained ones also can excite this condition. On occasions a subconjunctival dislocation of the lens is complicated by sympathetic ophthalmitis. Rarely choroidal tumours have provoked it.

All ages are prone to it though children, by virtue of the fact that they are exposed to this type of injury, appear to be more afflicted. The interval between injury and onset of the inflammation is about three weeks, though much longer intervals are known to occur. In cases where the companion eye has been involved about 30—40 years later it is conjectural whether the inflammation was really one of sympathetic ophthalmitis.

Pathology. A massive infiltration of the choroid with round cells and epithelioid cells with a tendency to follicle formation is characteristic. Cuffing of the emissary veins is again typical if found. Dalen-Fuchs' bodies—excrescences on Bruch's membrane are seen in many instances.

Aetiology. Two theories are favoured—bacterial and allergic. Of the former tubercle and a virus were incriminated. The latter viz., the allergic theory is the more probable especially after the work by Woods, who has helped to clarify much of the mystery round this condition. In the light of this theory an allergic sensitisation to patient's own uveal pigment occurs with the resultant inflammation. The occurrence of a blood eosinophilia and the extremely good response to cortisone therapy add further support to the allergic theory.

Symptoms and Signs. These in the injured eye are what are commonly seen in cases of acute iridocyclitis and are to be expected in most perforating injuries. The crux of the matter is the *early diagnosis* of trouble in the companion eye. *It is axiomatic that a close watch should be kept on the companion eye in a case of perforating injury of one eye.* In the second eye signs of involvement are usually lacrimation, photophobia an unwillingness to be examined, pain and loss of vision. Signs include ciliary tenderness a spastic pupil and an artificial myopia due to ciliary muscle spasm or a hypermetropia due to swelling of the macula. *If an irregular pupil with synechiae and / or 'k' p.' are seen then* the case is one of established sympathetic ophthalmitis.

Complications. In the injured eye a severe inflammation usually destroys sight and even the eye. If pyogenic infection supervenes sympathetic inflammation does not

occur in the usual course of events. All known complications of anterior uveitis can occur. In the companion eye the inflammation is even more fulminating and may cause loss of the eye. Optic neuritis, detachment of the retina, secondary glaucoma and complicated cataract are some of the complications met wtth. A tragic end result is occasionally a phthisis bulbi. Bleaching of eyelashes and brows and of the hair on the head with patchy baldness are peculiar concomitants of sympathetic ophthalmitis.

Treatment at one time was desperate and included an excision of the exciting eye inorder to save the companion eye. With the advent of cortisone excision of the eye has been playing a minor role in the treatment of sympathetic ophthalmitis. The exciting eye is dealt with surgically if there is a wound requiring suturing or repair. Antibiotics and atropine are used to prevent infection and anterior uveitis respectively. Cortisone topically is of immense value in cutting down the inflammatory reaction in a potential case. In the face of threatend sympathetic opthalmitis cortisone locally in the second eye as well as systemically administered is of paramount importance to forestall the inflammation. In the case of an adult 300 mg. daily for three days followed by 200 mg. daily for three days and tailed off with 100 mg. and 50 mg. for a week would suffice.

If excision is deemed necessary in a particular case it should be done at the earliest opportunity before established sympathetic ophthalmitis sets in.

133. Sudden Uniocular Blindness is a dramatic event requiring prompt treatment. In the conditions that cause it, the blindness may range from a blunting of sight to total blindness. The following are the causes :-

(a) Acute glaucoma (closed angle)
(b) Central retinal arterial occlusion
(c) Central retinal vein thrombosis
(d) Acute retrobulbar neuritis
(e) Detachment of the retina
(f) Intraocular haemorrhage
(g) Trauma

(a) *Acute glaucoma* has already been considered in §120, 122.

(b) *Central retinal arterial occlusion* may be due to *spasm*, *thrombosis* or *embolism*. Spasm is caused by hypertension, drugs like quinine, arteriosclerosis and is encountered in papilloedema. Occlusion by thrombosis is the cause in syphilitic endarteritis. Embolism is the rarest cause and is met with in mitral stenosis.

Clinically the obscuration of vision is sudden and complete depending on the pattern of the blood supply to the retina. In those with a cilioretinal artery supplying the macula, central vision is preserved. In those with a cilioretinal artery blockage, central vision will suffer. Ophthalmoscopically the fundus is a pale yellow with a *cherry-red spot* at the macula, an appearance due to the normal choriocapillaris showing through at the fovea (which is normally thin) and accentuated by the paleness of the swollen, bloodless retina. The arterial tree is thin and on occasions the blocked area may be seen at the disc. In cases with a cilioretinal artery a tongue of red retina is surrounded by ischaemic retina. The veins get broken up giving rise to the classical "*cattle truck*" appearance, the fragments making forward and retrograde movements. As time passes the retinal pallor disappears and so does the cherry-red spot, a point of distinction between this and Tay-Sach's disease in which the spot persists till death.

Treatment must be prompt and energetic. The aim is to cause immediate and efficient dilatation of the retinal artery and to achieve this vasodilators must be employed. An ampoule of amyl nitrite may be broken open and given the patient to inhale. Intramuscular and even intravenous vasodilators may be started as soon as possible. Priscol and nicotinic acid may be given intramuscularly. Parenterovite or Eupaverine may be given intramuscularly. Retro-ocular priscol, acetylcholine or nicotinic acid may be given to produce a quick and local action. In certain resistant cases an attempt at something more drastic may have to be tried. This consists in a stellate ganglionectomy. A watch on the response to therapy may be kept by frequent ophthalmoscopy. Oxygen inhalations under pressure is worth a trial. Local surgical measures to affect a lowering of the intraocular pressure have been advocated usually in the form of a drainage operation but no time should be wasted dilly-dallying with ineffective remedial measures.

(c) *Central retinal vein thrombosis* occurs in two distinct age groups. In the young a definite aetiology has not been ascribed though tuberculosis has been incriminated. In the older age groups arterial disease is the cause. Owing to the peculiar relationship of the artery to the vein in the retina changes in the former in arterio-sclerosis causes compression of the vein with a slow occlusion of the lumen of the vein and consequent thrombosis behind the obstruction.

Clinically a case of established thrombosis of the central retinal vein is unmistakable and the fundus has been aptly compared to the world famous Stephen's ink advertisement—a splash of haemorrhage. This picture is

modified if only a tributary is thrombosed. In the sector drained by the tributary in question there are large haemorrhages while the rest of the fundus shows little abnormality excepting nippings of arterio-venous crossings.

Diagnosis is easy as already mentioned if one encounters a case of established thrombosis be it total or tributary. From the point of view of treatment diagnosis must be made quite early so that anticoagulants may be used with benefit. The *pre-thrombotic sign* first described by Bonnet is of great importance in this connection. At the point of crossing of a vein and artery the former is pinched a little with a halo of haemorrhage and exudates and / or a halo of pallor. In such instances even after a lapse of some months thrombosis has been noted to follow.

Complications. In tributary thrombosis (commonly the superior temporal because of more arterio-venous crossings here) fair to good visual results may be expected. In main vein thrombosis a secondary glaucoma follows roughly a hundred days after the first accident. This time lag is due to the slow development of new blood vessels which invade the angle of the anterior chamber. It is held that in such cases there is also a coincident vortex vein thrombosis.

Treatment of the juvenile type should be directed towards the cause if any. This invariably is anti-tubercular. In the adult type while attention to any aetiological agent is necessary, anticoagulants should be used. Opinion differs on the choice of case as well as of the drug to be used but it is now accepted that anticoagulants have no place in established thrombosis. It

is in the early prethrombotic stage and junctional thrombosis that anticoagulants have a place. A fat free diet is advisable.

(d) *Acute retrobulbar neuritis.* In this condition there is pain on moving the eye about and tenderness can be elicited by pressing the globe back into the socket. The pain is due to the traction on the nerve and its sheaths. *This is the only condition in which with a white eye there is pain.*

Among the causes of retrobulbar neuritis disseminated sclerosis is held to be the only known one (in some countries). It is obvious that there are other causes of which nasal sinus disease should not be lost sight of.

Symptoms consist of blurring of vision with a large "spot in the centre" of the field, pain and alteration of colour vision.

Signs are a characteristic pupillary reaction—a slightly dilated pupil with an ill-sustained pupillary reaction, a caeco-central scotoma (i. e., involving blindspot and macula) for white and / or for red and green and a normal fundus except in disseminated sclerosis where a temporal pallor manifests itself sometime after the attack of retrobulbar neuritis.

Treatment. Elimination or treatment of the cause is the first step. Vasodilators and vitamin B_1 should be given in large doses. Smoking and alcohol should be prohibited or reduced.

(e) *Detachment of the retina* has been dealt with in §123.

(f) *Intraocular haemorrhage* may result from local or general causes. In tumours of the retina or choroid, as an accompaniment of detachment of the retina and in retinal vein thrombosis haemorrhage may obscure vision

by collecting in a preretinal position or by ploughing into the vitreous. Similarly in hypertension, diabetes, head injuries and blood diseases visual disturbances may be dramatic.

Symptoms and signs. In total vitreous haemorrhage the visual loss is profound and examination will reveal a black pupillary area both by oblique and ophthalmoscopic illumination. Differentiation from a black cataract has been discussed in §11a. A hyphaema may give a clue to the intraocular catastrophe. In a recent case a red clot may be seen just behind the lens. In pre-retinal or subhyaloid haemorrhage the visual loss is confined to the central field if the leak is central as often happens. Ophthalmoscopy shows a characteristic 'U' - shaped haemorrhage. Any case in which the aetiology is obscure should be treated with great care especially if the fundus is not visible as there might be an associated retinal detachment.

Attention must be drawn to a particular type of intraocular haemorrhage occurring in young adults—Eales' disease. Recurrent vitreous haemorrhages occur with proliferative retinitis and detachment followed by secondary glaucoma. It is held to be due to tuberculous periphlebitis on slender evidence. Antitubercular treatment and diathermy application to the site of periphlebitis are present methods of treatment.

(g) *Trauma.* Injuries which cause extensive damage to the eye will naturally produce sudden loss of vision. Of these gun shot wounds of the orbit with severance of the optic nerve, traumatic detachments of the retina, intraocular foreign bodies, intraocular haemorrhage and rupture of the globle comprise the more important group.

134. Sudden Binocular Blindness. Ocular causes rarely figure in this catasrophe. Certain poisons such as quinine, methyl alcohol and lead are responsible on occasions while central causes operate in other instances. In the latter group there are evidences of *cortical blindness*—blindness with normal fundi and normal pupillary reaction. In the first group the fundi show extreme attenuation of arteries, pallor of the disc, most marked in methyl alcohol poisoning and a general paleness of the retina. Among the causes of cortical blindness the following should be borne in mind :-

Trauma to the occipital region

Space-occupying lesions

Inflammatory lesions—meningitis

Vascular lesions : angiospastic causes are the commoner as met with in hypertension, nephritis, eclampsia uraemia and plumbism.

In hypertension the duration of amaurosis is short, lasting a few minutes, in nephritis it is longer lasting several hours, it is so in uraemia while in eclampsia the duration is on the average 12 hours. In lead poisoning too the visual loss is prolonged over hours or even days but recovery is usual.

Treatment is directed towards the cause, which might mean drastic steps such as termination of pregnancy in eclamptics. In quinine (and salicylate) poisoning all measures described under central arterial occlusion should be tried seriatim.

MOTOR ANOMALIES

135. Heterophoria is the term applied to imbalance of the muscles of the two eyes. It is also referred to as latent squint. Latent deviations are of the following types: *Exophoria* meaning lateral deviation, *Esophoria* meaning medial deviation, *Hyperphoria* meaning an upward deviation and *Cyclophoria* meaning rotational deviation on an antero-posterior axis.

Tests for Hetrophoria: The simplest test is the *cover test.* When properly conducted this test reveals the type of deviation and also gives information about the state of binocular vision. The test consists of covering one eye and then the other with a card and observing what happens to the eye under cover as soon as the card is either transferred to the other eye (monocular uncovering) or removed (binocular uncovering). The following information may be obtained:

A. Monocular uncovering

i. Neither eye deviates—orthophoria. Any suggestton of squint is false.

ii. Each eye squints when covered and returns to fixation when uncovered—heterophoria or hetrotropia is present.

B. Binocular uncovering helps to distinguish between a latent and manifest deviation.

i. The uncovered eye fixes while the other deviates on covering and refixes when uncovered—heterophoria.

ii. Neither eye deviates—manifest squint of covered eye. The other fixes constantly.

iii. Both move—manifest squint of the uncovered eye which fixes only when the sound eye is covered.

iv. Each eye moves alternately—alternating squint.

A seond useful and much practised method of detecting muscle imbalance is the *Maddox rod test.* The Maddox

rod consists of a row of cylinders mounted on a round disc, the glass being of a red colour. When held before a spot of light a red line is seen owing to refraction in a plane at right angles to the axes of the cylinders. The patient is seated at a distance of 6 metres from a spot-light. A trial frame with a Maddox rod in its right cell is worn by the patient. He is instructed to observe the spot-light with both eyes and to report what he sees. What is the relationship of the 'red line' to the spot-light? The rod is placed so that the cylinders are vertical: if the line cuts the light there is no imbalance in the vertical plane. If the line is below the light there is right hyperphoria and if the light is above the light there is left hyperphoria. Now the rod is turned so that the cylinders are horizontal: if the line cuts the light there is orthophoria (normal balance). If the line is to the right of the light there is esophoria and if the line is displaced to the left there is exophoria. Cyclophoria will be indicated by an oblique position taken by the line despite the correct orientation of the rod before the eye. The amount of phoria is measured by the prism placed in the left cell that will bring the line across the light. As prisms displace objects towards their apices the apex of the correcting prism will have to point towards the direction of displacement of the line.

Muscle balance at reading distance is measured by the Maddox wing.

Symptoms of Heterophoria will depend on whether the muscle imbalance is compensated or not. This in turn will depend on one's reserve muscle power which helps to maintain the two eyes in harness and the desire for binocular single vision. Decompensation occurs under certain conditions of debility, fatique or if one or other of the factors mentioned above is deficient. Symp-

toms of decompensation are headaches, difficulty in changing focus, blurring of print fleeting diplopia, squint of an intermittent type, difficulty in judging distances, depth and defective stereoscopic vision.

Treatment consists of correcting refractive errors and general debility. In addition orthoptic treatment is indicated in selected cases especially in cases of exophoria with convergence deficiency. It is contraindicated in the over-worked or fatigued person and the neurotic.

136. Heterotropia or Manifest Squint *is said to exist when the visual axes of the two eyes do not meet at the point of regard.* It is divisible into paralytic (non-comitant) and concomitant (non-paralytic) types. The following Table shows the points of distinction between these two types:-

TABLE IV. Concomitant and Non-comitant Squints

	Concomitant	Non-comitant
1.	Primary and secondary deviations equal	Secondary deviation greater than primary.
2.	Axes of the two eyes maintain the same angle in all directions of gaze.	Angle between axes varies as gaze varies.
3.	No diplopia	Diplopia present
4.	No vertigo	Ocular vertigo
5.	Refractive error in some	Nervous or muscular cause

It is to be remembered that time is an important factor in the production or alteration of some of the clinical features of the types of squint mentioned above. For instance diplopia is present only in a recently acquired

paralytic squint, while in an old standing paralytic squint certain secondary changes in the muscles tend to mask the exact nature of the squint and moreover mimic a concomitant squint. These changes are:

1. Contracture of the ipsilateral antagonist.
2. Overaction of the contralateral synergist.
3. Overaction of the ipsilateral synergist.
4. Inhibitional palsy of the contralateral antagonist.

Diagnosis of true from false squint is important, the cover test providing a simple means of detecting one from the other. In the case of infants this test is inapplicable and so reliance must be placed on a very simple test described by Hirschberg. A light is shone on to both eyes from a distance of a foot or more and the reflections of this source on the two corneae are noted. If the reflections are central there is no squint despite any apparent deviation that may be present (false squint). If the reflection is against the edge of the pupil the degree of squint is 15°, if the half-way between the limbus and centre of cornea 20° and at the edge of the cornea 45°. This is a very practical test though not accurate for the basis of the test is that for every displacement of the light reflection by 1 mm. there is a deviation of 7°, quantities too small to discern with the naked eye.

Causes of False Squint

1. Abnormal angle gamma—the angle between visual and optic axes. In the normal this angle averages about 5°; in myopia it is less and in hypermetropia it is more. Clinically it is not possible to measure *angle gamma* owing to difficulties in estimating the centre of the cornea. The corneal reflex is made use

of: while the eye is fixing a light if the visual axis and the central pupillary line coincide the reflection of the light will appear to be central. Usually the reflex is nasal to the centre in emmetropia more so in hypermetropia and may be on the temporal side (negative) in myopia. This angle is termed the *angle kappa.* An apparent divergence is noticed in hypermetropia and an apparent convergence in myopia. it will be apparent from the above that in any evaluation of the angle of squint, angle kappa must be allowed for. In a case of say divergent squint with a positive angle the angle of squint is made larger by the amount of the angle kappa.

2. *Epicanthic folds* give the appearance of a convergent squint.
3. *Wide separation of orbits* gives a divergent look.
4. *Improperly developed ethmoids* gives rise to a false convergent squint.

Aetiology of concomitant squint. Several factors enter into the causation of this type of squint of which the following appear to be important. *Anatomical* peculiarities predisposing to faulty positioning of the eyes, deranged *accommodation-convergence* link and a faulty *fusion*, play important roles. Hypermetropia and myopia are responsible for anomalies of the accommodation-convergence link, a hypremetrope accommodating in excess of his convergence and precipitating a convergent squint. A congenital myope uses his convergence only and goes in for a convergent squint. In acquired myopia any accommodation that may have developed is rapidly neglected and with a further lack of desire (or necessity) for accommodation, a divergent squint is

usual. A divergent squint also results in a case in which one eye is myopic and the other hypermetropic (anisometropia). A blind eye tends to deviate for the same reasons, converging in childhood and adolescence and diverging in adult and old age.

Sequelae of Squint. Amblyopia or blunting of sight usually develops in the squinting eye (which is the more defective eye from the point of view of refraction). In some a psychologically accepted diplopia is present. In others an inacceptable diplopia drives them to a doctor or they develop suppression to avoid the second image of the eye that is not fixing. Alternation is another solution and is seen in persons who have equal vision in each eye or when one eye is emmetropic or hypermetropic and the other myopic. An adult confronted by diplopia would adopt the easiest dodge and cover the deviating eye, suppression being very slow and unusual at this age.

Treatment of Concomitant Squint. Refractive errors should be corrected fully if hypermetropia is detected. Amblyopia should be overcome by occlusion of the sound eye with adhesive plaster over dressing. Occlusion is maintained for a month at least during which time the child is allowed to look after himself in all respects such as conducting himself at school and at home at lessons, meals and play. No parent or teacher should pity the child and offer to help at every little step the child and takes the first few days. It is surprising how the child picks up quickly and within a week vision improves to a remarkable extent provided of course the amblyopia is one of extinct and not of arrest.

The next step is orthoptics firstly to study the degree of binocular vision the child has. Suppression is dealt with by special exercises designed to stimulate the suppressed macula. These exercises are best carried out with the major amblyoscope. Orthoptics does nothing to the angle of squint, it only trains the mind and not the muscles. It is a diagnostic aid and in a few instances it helps the surgeon and prepares the patient for an operation by eliminating perverted visual habits. When the preliminaries are over the question of operation has to be considered. In some, glasses alone may render the eyes straight. In others glasses and orthoptics re-establish binocular single vision. There is however a good percentage of cases that requires operative treatment. This usualy entails the adjustment of one or more muscles so as to render the eyes straight. The principles governing operative measures are that an over-acting muscle should be relaxed and that mucles of both eyes should be operated on if the angle of squint is too large. A weak muscle may be re-adjusted but this usually involves the mere tightening up of a lax muscle so as to provide a better mechanical advantage for it to contract. Operations on the extra-ocular muscles may be divided into lengthening and shortening procedures: lengthening is achieved by recession, tenotomy and marginal myotomy while shortening is done by advancement, cinching tucking and resection.

SOME AFFECTIONS OF NEUROLOGICAL INTEREST

137. Ocular Palsy. While ocular palsy is of importance in diagnosing the site of an intracranial lesion it should not be forgotten that some palsies are *false localising signs* due to the fact that certain nerves have a long intracranial course during which displacements of the brain may impinge on the nerve though the offending lesion is nowhere near the site of pressure and what is worse may be on the opposite side. The sixth nerve is classically described as one most prone to this type of behaviour. The third nerve too may be affected similarly by herniation of the hippocampus into the tentorium jamming the third nerve tight against the tentorial margin.

Classification

1. *Supranuclear*, characterised by *disturbances of conjugate eye movements* such as vertical movements (Parinaud's syndrome), loss of lateral conjugate deviation and of convergence. Lesions of the frontal lobes cause a dissociation of voluntary ocular movements from the fixation reflex. This results in a *spasm of fixation* from which the patient cannot unhinge his eyes. If his sight is fogged by a strong plus lens the spasm disappears. If the patient's head is turned while he fixes an external object his eyes will deviate in directions in which they failed to move voluntarily. There is *no diplopia.*
2. *Nuclear* are characterised by involvement of individual muscles or groups of muscles. True nuclear palsies are difficult to spot owing to the absence of satellite symptoms but their association with brain stem lesions gives the clue. Diplopia is present.

3. *Infranuclear* characterised by individual muscle palsies with diplopia.

Aetiology.

1. Neoplasms — Optic N. tumours

A NERVE
- a. Orbital — Osteoma, Pseudotumour
- b. Sphenoidal ridge — meningiomas
- c. Pituitary tumours
- d. Endothelioma of nasopharynx
- e. Brain stem

B. NUCLEAR

C. SUPRANUCLEAR

2. Vascular

a. Hypertension and atheroma
b. Aneurysm
c. Ophthalmoplegic migraine (aneurysms not the only cause.
d. Brain stem syndromes, Weber's, Benedikt's, Nothnagel, Millard Gubler and Foville's.

3. Trauma

Fractures of skull

4. *Inflammatory*

a. Syphilis—Tabes dorsalis, G. P. I.
b. Meningitis—tubercular
c. Cavernous sinus thrombosis
d. Sphenoidal fissure fibrositis
e. Otogenic—Gradenigo's syndrome (6th N.)
f. Poliomyelitis
g. Encephalitis lethargica
h. Rabies
i. Herpes zoster

5. *Primary Muscular Affections*

a. Myasthenia gravis
b. Exophthalmic ophthalmoplegia
c. Myopathies including ocular
d. Myositis

6. Demyelinating Diseases

7. Toxic and Metabolic

a. Alcoholic polyneuritis
b. Diabetes mellitus
c. Diphtheria
d. Botulism
e. Tetanus
f. Lead, veronal.

8. Degenerative

Progressive Nucular ophthalmoplegia

9. Heredofamilial

a. Friedreich's ataxia
b. Sanger-Brown ataxia

10. Developmental

Aplasia of muscle

Treatment will depend on the cause of the palsy. Generally speaking if a palsy does not recover in three to six months surgery should be considered. In aplasia or absence of muscles transplantation of muscles may be necessary. In degenerative conditions no treatment is of avail.

138. Ptosis or drooping of the lid may be classified on the above lines and may be caused by most of the agents mentioned under ocular palsy but it is specially described under a separate head to include certain local causes viz., mechanical causes such as a heavy upper lid due to trachoma or neurofibromatosis or complete severance of the levator tendon in injuries of the upper lid. In those who do not wear a prosthesis after enucleation ptosis develops as a form of disuse atrophy. Owing to the lack of support to the upper lid from the globe or a substitute the levator has no position of vantage to contract and so atrophies by disuse. It is also an important feature of Horner's syndrome.

Treatment is medical in medical conditions. In the congenital variety operative treatment should be instituted at the time of commencing to walk as otherwise faulty postures are adopted by the child. In myasthenia gravis crutch glasses are advisable. In ocular myopathy an operation utilising the frontalis muscle may be performed to lift the lid off the pupil. Wherever surgery is indicated the following principles govern the plan of action.

1. Skin excision
2. Skin excision and burial of flaps linking the lid tissues to the frontalis.
3. Use of tantalum wire, fascia lata and catgut to hitch the lid to the frontalis.
4. Utilisation of the levator muscle by advancement coupled witb resection.
5. Transplantation of a slip from the superior rectus to the upper border of the tarsal plate.

Of all operations enumerated the most satisfactory procedure is the 4th all else being *a*-physiological. As a ptosis operation is a beauty operation certain points must be borne in mind when undertaking surgery. The two upper lids should be level with each other after surgery. The upper palpebral groove should not be obliterated, nor should the lid border get peaked in the mid point. Beauty apart, no danger should befall the eye due to exposure keratitis by undue lift of the lid.

139. Papilloedema means oedema of the optic nerve head and is caused by a number of aetiological factors which may be local or orbital, intracranial or general. The mode of causation is open to much speculation but there are some factors which are present in most of the cases so as to throw some light on the mechanism of produc-

tion. Raised intracranial pressure or a lowered intraocular pressure, obstruction to the outflow of blood along the central retinal vein and the water binding property of the optic nerve play a part in the pathogenesis of papilloedema.

Signs. The earliest sign is a filling in of the physiological cup at which stage perimetric examination reveals enlargement of the blind spot. Later the margins of the disc appear hazy while its surface shows increased redness and the venous pulse disappears even on pressure. Greyish-white striations make their appearance round the disc. With the ophthalmoscope the edges of the disc are brought into a sharp focus by successively turning plus lenses into the peep hole. To see the centre of the disc stronger plus lenses may have to be added. Away from the disc there may be haemorrhages and 'exudates'. If the macula is involved a central scotoma may be noted. Peripheral contraction of the visual field occurs when secondary atrophy sets in.

Visual disturbances may be nil and the pupils are normal.

Papilloedema has to be differentiated from papillitis, an inflammation of the nerve head. Any swelling more than 2 dioptres in extent is arbitrarily considered to be papilloedema. More important than this is that in papillitis there is profound loss of vision.

Causes of Papilloedema

1. *Local*
   a. Lowering of intraocular tension.
   b. Tumours of the orbit and optic nerve.
2. *Intracranial*
   a. Tumours of the brain
   b. Brain abscess

c. Hydrocephalus
d. Thrombosis of sinuses
e. Meningitis

3. General

a. Hypertension
b. Anaemia
c. Leukaemia
d. Emphysema

Differential Diagnosis of papilloedema from papillitis is important and the points of distinction are shown in Table IV.

TABLE IV. Papilloedema *vs* Papillitis

	Papilloedema	Papillitis
1.	Visual impairment not consistent with degree of Oedema.	Visual loss profound and more than warranted by amount of swelling.
2.	Progressive slow deterioration of vision.	Rapid fall of sight but recovery usual.
3.	Enlargement of blind spot.	Caeco-central scotoma.
4.	Disc swelling more than 2D.	Disc swelling up to 2D.
5.	Venous engorgement not marked.	Marked venous engorgement.
6.	Evidence of raised intracranial tension in many cases.	No raised intracranial tension.

Both the above conditions should be carefully distinguished from "*pseudo-neuritis*" which mimics them very closely. The latter is due to a heaping of neural and glial tissue at the disc and is congenital usually associated with marked tortuosity of the blood vessels and a hypermetropic refraction. Repeated and prolonged observation together with perimetric examination and fundus photography are necessary in difficult cases.

Indications for Decompression

1. Large degree of swelling
2. Great engorgement of veins

3. Presence of extensive haemorrhage
4. Early appearance of exudative spots
5. Progressive deterioration of sight.

140. Optic Atrophy is the term applied to a degeneration of the optic nerve. It is characterized by progressive loss of vision both central and peripheral with pallor of the nerve head. Disturbances of colour vision may be met with. Pallor is extremely variable and while it is a guide in many cases too much importance should not be attached to it. Firstly if one were to wait for pallor to be established it may be too late for proper treatment to be instituted. Secondly in diseases like disseminated sclerosis there is marked pallor but good visual function. In the infant, in whom auxiliary investigations are not possible it is extremely difficult to pronounce a verdict on optic nerve function by looking at the disc especially when one encounters a duck-egg blue colour in many normal infantile discs and when it is now known that a delayed myelination of the optic nerve may retard visual function.

Classification. Optic atrophy is classified into (i) primary, (ii) secondary and (iii) consecutive.

Primary clinically no disease of the optic nerve is detectable. The edges of the disc are cler-cut.

Secondary — following papilloedema (q. v.). The disc edges are fuzzy due to spilling over of neuroglial tissue.

Consecutive—following retinal disease. Evidence of retinal disease is present.

Evolution of Optic Atrophy. The rate of onset and progress of optic atrophy may give a clue to the probable casue. For instance a rapid loss of vision is likely to be caused by some vascular factor, one in which a central

scotoma exists is probably due to a tumour of the optic nerve while one in which recovery occurs is most likely due to a demyelinating disease.

Investigation of a case of optic atrophy should include X-ray examination of the nasal sinuses, optic foramina and fissures, pituitary fossa and cerebral arteriography. Perimetric and tonometric studies are also of great importance.

141. Proptosis is the term applied to a protrusion of the eyeball whatever its cause though by usage protrusion due to endocrine disturbances is called *exophthalmos*. *Investigation.* The following points should be specially looked for:-

1. Direction of proptosis
   a. Axial in optic nerve tumours
   b. Laterally in ethmoidal affections
   c. Upwards in maxillary affections
   d. Down and laterally in frontal sinus affections
2. Reducibility
3. Pulsation
4. Bruit
5. Effect of carotid compression
6. Effect of jugular compression
7. Endocrine disturbances
8. Enlargement of optic foramina (X-rays)
9. Refraction, ocular movements and fundi.

Signs. In early cases the presence of proptosis may be difficult to detect. A proptometer or an exophthalmometer may be used but a simple clinical method is Naffziger's sign. With the patient seated on a stool the examiner stands behind and observes the two corneae from above, tilting the head backwards till the observer's

line of vision coincides with the plane passing vertically past the apices of the corneae of the subject. Any slight protrusion is easily spotted by this method.

Orbitonometry is the method of estimating orbital tension. A special instrument is used to study the resistance offered by the retro-ocular tissues to weights placed over the globe. The nature of the pathological lesion can be ascertained by this method.

Diagnosis. There are some pitfalls in the diagnosis of proptosis. An enophthalmos of one eye may give an impression of proptosis of the companion eye. So also a ptosis on one side may indicate a false proptosis of the opposite side. A widening of the palpebral fissure on one side may point to a proptosis on the other side. A lid lag also would point to the same conclusion. A swelling of the lids may erroneously imply a proptosis. A large globe or a large cornea may again mimic a proptosis.

Clinico-pathology. The orbit resembles the cranium in many respects, firstly in that it is a bony and therefore rigid cavity, secondly in that a vital organ, the eye and its nerve are housed in it. Increase in orbital contents can be accommodated upto a certain limit, the only outlet for expansion being in front. But the orbital septum and the globe offer resistance anteriorly to an expanding lesion. Thus the orbital contents must of necessity be strangled especially in acute episodes. Hampered venous drainage, pressure on the central vein of the retina through the nerve sheath would add further to the threatened damage to the visual function. From sheer exposure of the cornea over which tight lids may not be able to close the former may ulcerate, a situation aided and abetted by massive chemosis met with in some pathological conditions affecting the orbit.

Treatment of proptosis will depend on the cause. Emergency care is often necessary to avert loss of an eye through corneal ulceration. In birth injuries (§92), in malignant exophthalmos and other types of proptosis where the cornea is threatened some urgent step is necessary. Protection of the cornea with dressing, contact lens or a tarsorrhaphy may be called for. More elaborate operations such as the decompression operation of Naffziger should be adopted for progressive endocrine exophthalmos.

PART III

OPERATIVE, PRE- AND POST-OPERATIVE TREATMENT

142. Pre-operative Measures. As the margin of safety in eye operations is small every precaution should be adopted pre-operatively for the safe conduct of the operation and of uneventful convalescence. As many ophthalmic operations are performed under local anaesthesia the patient has to be free of apprehension and extend as much co-operation as he can during the operation. The following is a scheme of action designed for the welfare of the patient.

History of cough, asthma, retention of urine, constipation and of alcoholism should be inquired for and noted. All the above militate against a safe operation unless counter measures are taken to eliminate or at least minimise the above mentioned conditions. Restlessness and straining may cause opening of wounds such as in keratoplasty or cataract operation.

Examination should include a general clinical check-up with special attention to hypertension, diabetes mellitus and infective foci in teeth, sinuses and skin. Owing to the risk of haemorrhage after operation hypertension requires treatment. To avoid infection diabetes as well as the foci referred to above must be adequately controlled

Personal Hygiene is important in eye operations. Daily baths, shortly trimmed nails and good bowel action should be seen to.

Conjunctival Culture. A bacteriological study is useful as certain harmful pathogens contraindicate operation.

Of these B. pyocyaneus, Staphylococcus aureus, B. coli are to be dreaded. Coagulase test is an invaluable aid in diagnosing pathogenicity of streptococci and staphylococci. Given favourable conditions even the B. xerosis can incite severe inflammation. Opinion regarding the feasibility and validity of bacteriological examinations has tended to fluctuate in recent times so much so that some surgeons do not routinely subject their patients to this examination. From experience some have found that in cases considered clean on such examination the most severe inflammation has occurred post-operatively. In others in whom the culture indicated pathogenic organisms the eye has done well. Between collection of a conjunctival smear and again between reporting of a 'clean' smear and operation there is ample time for infection or contamination of the conjunctiva, on many occasions self-inoculated. It is therefore imperative that ordinary hygienic measures be insisted upon together with toilet of the face and eyes.

An obviously infected conjunctival sac is a contraindication. This may be due to dacryocystitis, some infective lesion adjacent to the lids or a primary infection. A simple clinical method of determining whether an eye is harbouring pathogens is to apply a pad and dressing to the eye in question overnight and inspect the eye and the pad for excess of mucus or discharge. If free of discharge the eye may be operated on.

Eye Exercises. Although the modern eye surgeon is expected to perform eye operations without having to rely on the patient for cooperation on the table it is still advisable for our patients to have instructions on what and how to do these before during and after the operation. Looking in various directions on command, raising and placing his head on the table before the operation

and lying releaxed are points the patient should fully understand and anticipate. He should not lift his head off the table at the end of the operation for the application of the bandage ; this will be done by the staff. He should be warned about the injections he may receive on the table. If he is deaf or liable to be confused on the table regarding the directions in which he is to look, signalling to look down by tapping on his feet will have to be resorted to. Sneezing may be prevented by squeezing the area between the philtrum and septum of the nose.

The patient should be instructed that for some time after operation he will be strictly confined to his bed and he should be acquainted with the use of a urinal and bed-pan. His position in bed should be explained to him.

Preparation for Operation. A day prior to the operation the patient has an aperient or an enema. He has a bath and his eyes are washed and some antiseptic or antibiotic drops instilled. The lashes are trimmed by cutting them with a pair of scissors whose blades have been smeared with vasleine. Premedication may be commenced if ordered. In cases of cataract, atropine and in cases of glaucoma, eserine may be instilled the day previous to the operation.

On the day of operation the eye is washed with N. saline, massaging the lids and the lacrimal sac. A few drops of antiseptic drops are instilled. The mydriatic or miotic drops may be used as directed. The sedative may be administered one hour before the operation and the patient allowed to rest in bed till he is called up for the operation. From the ward the patient is taken to the theatre where at frequent intervals local anaesthetic is

applied. If premedicated he is allowed to lie on a trolley. From the trolley he is transferred to the operating table with the minimum of disturbance.

The Theatre should be suitably darkened for it is only then that a focal lamp as is used in ophthalmic operations gives a good illumination. There should be perfect silence for the patient should not be distracted by clatter of instruments and the needless chatter of the staff. It is then that the patient will quickly respond to commands given him.

The Sister should be one who understands firstly the steps in a surgical operation, secondly the needs of a particular surgeon. It is difficult to carry in one's head all that the different surgeons require. It is best to enter in a book the routine of the different surgeons.

The Assistant has very little to do in an eye operation but the little that is to be done must be well executed. Careful watching is necessary in order to anticipate the steps of the operation and render just the help expected of him. It is wrong for an assistant to take hold of a suture or a structure on the eye and turn away from the field of operation to pick up something from the side table. The result of this can be imagined if the patient were to roll his eye about. This well illustrates the need for attention throughout the operation.

143. Anaesthesia and Akinesia. Cocaine drops, 2 or 4% are instilled every quarter hour starting an hour before the operation. Cocaine causes in addition to its anaesthetic effects a pupillary dilatation through its action on the sympathetic system. It also causes desiccation of the corneal epithelium.

Akinesia is effected by injection of 2 or 4% procaine (1) retro-ocularly and (2) into the upper fibres of the

facial nerve; the latter may be done by blocking the nerve in front of the ear or at the lateral angle of the eye in a triradiate manner.

Dangers of Retro-ocular Injections.

1. Orbital haematoma
2. Paresis of extra-ocular muscles,
3. Very rarely damage to the optic nerve.

These apply to all injections excepting air injection in which the latter two do not occur. In the presence of an orbital haematoma it is folly to proceed with the operation. The patient should be sent back to the ward and taken up after a few days. Owing to the increased orbital tension a grave risk of vitreous escape exists in such circumstances.

Akinesia may be enhanced by the exhibition of curare. As the ocular muscles are the first to be affected a small dose is sufficient for eye operations. A dose of 3—10 mg. is adequate for most cases. It is best started when the surgeon is ready, 3—4 mg. being given intravenously at first followed by a further 2—3 mg. as required, the needle being left inside the vein.

144. Steps in Operative Procedure. Retraction of the lids is usually effected by some kind of speculum. Some prefer to use lid sutures while some use lid clamps of Castroviejo while still others prefer muscle hooks to retract the two lids apart.

A last wash on the table is uncalled for; it is in fact more harmful for one thing it prolongs the operation and the wash helps to press out lurking organisms into the conjunctival fornices.

All instruments must be handed over *dry* a point of great importance in eye surgery for infective material can ride into the eye when an instrument dribbling water is in-

troduced into the eye. For the same reason the surgeon's hands should be dry.

Gloves are not necessary especially in cataract and corneal graft surgery for it has been reported that the dusting powder and fine shreds of cotton from swabs used in slipping the fingers of the glove on to the hand have delayed wound healing. If operating without assistance the surgeon should wear gloves for injecting and up to the stage of inserting the speculum. Thereafter he should wash his hands after removing the gloves before proceeding to operate. In long operations the cornea should be kept moist with saline which is dropped on the cornea with a dropper.

Fixation of the Globe is achieved by one or more muscle stitches through one of the recti. In the case of the superior rectus stitch in cataract surgery particular care should be taken to pass the suture through the muscle a few mm. behind its insertion otherwise the wound would gape.

In addition to the above method the globe is steadied by grasping it with forceps. Conjunctival fixation is achieved by using special toothless forceps or muscle fixation is effected by means of toothed forceps.

Bleeding points are touched with hot probes or with Wright's cautery if topical adrenalin is not effective. Small swabs cut out from ribbon gauze are used for swabbing the eye. If ribbon gauze is not available ordinary gauze may be used provided the borders are neatly folded in. Where *diathermy* is used the indifferent electrode is strapped to the forearm, the latter having been smeared with Cetavlon or K. Y. jelly. The active electrode is passed through a sterile linen sleeve which includes the handle and a length of the flex. Most of these handles are boilable.

Ophthalmoscopy during operation is done with an opthalmoscope wrapped in a sterile towel or linen sleeve. Care should be taken to keep the cornea clear and moist by instilling saline on to it during the operation. The cornea dries due to the local anaesthetic used, the inability of tears to flow over it as the lids are kept separated and due to the operating lamp desiccating it.

The introduction of drops or ointment into the operated eye must be done after a word of caution to the patient. The lids are gently closed ; in cases where lid sutures had been used the upper lid sutures may be fixed to the lower lid by leukoplast. Over the closed lids may be placed a square of tulle gras. The latter may be made by placing cut squares of book muslin in a tin box into which white vasline is also introduced in just adequate quantities and autoclaved. The vaseline spreads evenly over the squares of muslin. Over the squares of muslin are placed one or two pads and the eyes bandaged with a binocular bandage such as Moorfields'. In cataract operations a shield is strapped over the operated eye. This may be of aluminium, compressed cardboard or stiff vanista paper. There are of course small variations in all these steps from surgeon to surgeon. Figure of eight bandaging is practised by some while others use a plaster of Paris bandage which is removed by cutting for inspection and replaced over the eye if required.

145. Post-Operative Care. From the operating table the patient is carefully carried off on a canvas sheet to a stretcher. In the ward the canvas sheet with the patient on it is left to remain on his bed, the stretcher poles being removed from the side tunnels.

Nursing is very important thereafter. The patient is blindfolded and is unable to help himself. It is all the more important that a close watch should be kept on him. The following points are of practical importance,

PAIN. After ocular operations pain is not a feature unless some complication has occurred. If pain is only slight reassurance of the patient is all that is necessary. A mild sedative may be given. If severe an inspection of the eye is called for. A clue as to what is happening may be given by a blood stained bandage. In a cataract operation this would mean a vitreous haemorrhage—a delayed expulsive haemorrhage. In others probably a prolapse of the iris has occurred or the wound is gaping. Unless very necessary the eye should not be disturbed. Pain may be due to the injection punctures in sensitive patients or to a very tight bandage.

ABDOMINAL DISTENSION may be due to gas or urinary retention. Meteorism may be dealt with by the administration of carminatives or calcium pantothenate. A rectal tube answers in stubborn cases. *An enema should not be ordered unless the surgeon who operated on a case gives instructions.* The danger to the eye by rough handling of the patient during the administration of an enema with a syringe hardly requires any description. If an enema is really required it should be given by the gravity method.

Urinary retention is to be expected in elderly males who may be having hypertrophied prostates. In the recumbent position the difficulty in passing water is certainly greater. Hot water bottle applied to the hypogastrium or alternate heating and cooling (with an ethyl chloride spray) often helps in the voidance of urine. In cases that have had a suture a semi-sitting position or even sitting over the edge of the bed with assistance of course will enable the patient to pass water. The administration of parasympathetic drugs acts dramatically in most instances. If all measures fail as a last resort catheteri-

sation may be performed under strict aseptic precautions for if bladder infection supervenes the eye is certain to be ruined.

If bowel action does not normally occur on the third or fourth day an enema may be given.

DELIRIUM is met with in old people and is due to the total bandaging of both eyes, boredom and a fear of some impending calamity. This is of course aided by the use of atropine pre-operatively. In some the delirium is due to the sudden withdrawal of alcohol. It is expedient in all cases to inquire into alcoholic habits and allow little alcohol if a history is forthcoming. Stoppage of atropine and the administration of sedatives would help just as much as uncovering the un-operated eye.

POSITION IN BED.

Cataract. If no suturing of the wound is done the patient lies flat on a low pillow for 24 hours being permitted to turn on the unoperated side thereafter. If a suture is employed the patient is propped up in bed on high pillows or on a bed rest.

Retinal Detachment. The position in bed depends on the site of the detachments. In lower detachments a semi-Fowler position is advisable. In upper detachments a Trendelenberg position with its attendant discomforts is nowadays dispensed by double-padding the eyes and allowing the patient to lie on a low pillow. The rolling up of the eyes on closure brings the detached part to a dependent position. In lateral or medial detachments the position will depend on the site of the tear, always making sure that this is the most dependant part.

Keratoplasty. In lamellar grafts a certain amount of freedom is permissible but to be on the safe side and

also to ensure uniformity of post-operative care in all graft cases it is best to be rigid in enforcing certain rules. In penetrating grafts immobility is always advisable, because movements of the head and body may end in dislocation of the graft.

Glaucoma. The patient is nursed in the sitting up position so as to allow good drainage of venous blood from the head and eye. In cyclodialysis for instance this position also helps gravity to act on the ciliary body and the vitreous to fall away from the area of dialysis, thus ensuring free drainage of aqueous into the supra-choroidal space through the newly created passage.

Squint. Apart from preventing movements of the two eyes by binocular bandaging no restriction need be imposed on the patient.

Enucleation, Evisceration and Sac Operations may be given freedom of movement as long as there is no haemorrhage.

SITTING UP. The sitting-up time varies from surgeon to surgeon and from country to country and obviously depends on such factors as the use of sutures, the type of operation and nursing facilities. If sutures are employed patients may be allowed to sit up on the second day, otherwise on the third day. With the illiterate and stupid types it is wise to prolong the recumbent position by a day. This regime would apply to cataract cases mainly. In kearatoplasty of the penetrating type more care should be exercised, not permitting sitting up for a week. In detachments again prolonged rest in bed is necessary. However with the more modern operations of scleral resection and scleral buckling confinement to bed is cut down.

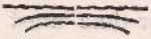
DIET. A liquid diet for the first two days and a low soft diet for the next two days are insisted upon for two reasons. Firstly chewing movements are not without effects on the eye indirectly through the orbicularis muscle; secondly bowel action is to be discouraged in the first few days. Besides this an over full stomach would cause discomfort to a person in bed.

INSPECTION OF THE EYE. The bandage is removed and the lids swabbed gently. The patient is instructed to open his eyes gently, aided if necessary by one's thumb placed over the patient's brow. Traction on the lid is effected through the skin of the brow. If a light is needed for inspection it should be shone from the side in stages so as to give the patient a chance to get used to it. In a cataract case a quick look at the wound, the level and size of the pupil is sufficient for the first inspection. In a graft case the position of the graft is all important to exclude dislocation of the graft. In squint cases one should not be disappointed if the eyes do not appear straight, for time must lapse during which the oedema over the muscle or muscles subsides. It is only after this initial period one can judge whether a squint has been under- or overcorrected.

In dacryocystectomy the nose should be inspected with a view to seeing if the cavity is blocked with blood clots or crusts. These should be gently removed.

REMOVAL OF SUTURE. This is very easy in most cases of ophthalmic operations as it is only a running stitch. In cataract cases however great care must be exercised for an upward movement of the eye during attempted removal of a suture can lead to rupture of the wound and prolapse of the iris. Proper cocainisation

is essential. A retractor or speculum may be used but in many cases retraction of the lid may be accomplished by one's thumb. The scissor points should engage the suture at an angle of 60° or 120° and not at 180°. In this position even if the patient rolls his eye up there is no risk of the uncut suture getting entangled in one blade of the scissors. Once the suture is cut it could be picked up with forceps. The same care is needed in the removal of sutures after keratoplasty. The sixth to the eighth day is usually a convenient time for removal of sutures in cataract and keratoplasty cases.



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பகுப்பெண்: சேர்க்கை எண்:

இத்தப் புத்தகம் திருப்பி ஒப்படைக்க வேண்டிய காலத்திற்கு முன் ஒப்படைக்க வேண்டும்.

மேற்கொள்ளும் காலதாமதமாகும் ஒவ்வொரு நாளுக்கும் -/10 சதம் தீர்வையாக அறவிடப்படும்.