13 Clinical Evaluation of Glaucoma Patient

In a general ophthalmic practice majority of patients who are diagnosed as glaucoma or glaucoma suspects do not present with complaints related to glaucoma but are picked up in the clinic during their routine eye check-up or when they come for their glass prescription including presbyopic glasses. A high index of suspicion is therefore the key to pick up patients with glaucoma before they become symptomatic. In these days of 'cataract hunting' it is not uncommon for people to miss obvious glaucoma-related eye changes and seeing only a cataract. By the time the patients with primary chronic glaucoma are symptomatic reversing the symptoms is not possible at this present time. Early diagnosis and prevention of disability is therefore the key in management of glaucoma.

Three decades ago people had little doubt that glaucoma was a disease cause by raised intra-ocular pressure (IOP). Today that has changed. While reducing intraocular pressure (IOP) is still the mainstay of treatment, the definitions of glaucoma have changed. More than one-third of patients with primary open angle glaucoma have normal IOP.

13.1 Definitions

Glaucoma: Glaucoma is a chronic optic neuropathy characterised by typical disc and field changes (described later) where 'elevated' intra-ocular pressure is the primary and only modifiable risk factor. A very humbling definition, saying in effect that we do not fully understand glaucoma. Imagine if we defined Cholera as 'a disease characterised by watery stools that can result in death and eating outside is a known risk factor'!

Pre-perimetric glaucoma: This is a condition where there are glaucomatous changes in the disc with normal visual fields on standard testing. This definition implies two things. First, that field changes occur only after some amount of nerve damage. It is said that field changes occur only after 20–30% of ganglion cell loss. The second implication is the importance of clinical evaluation of the disc in the diagnosis of glaucoma/pre-perimetric glaucoma.

Angle closure disease (ACD): This is the spectrum of conditions where the iris at the angle of the eye is in contact with the trabecular meshwork, preventing drainage of aqueous through the trabecular meshwork and ciliary body. This in turn can cause rise in IOP and lead to glaucomatous changes.

Though according to definition glaucoma is associated with disc and field changes, the term glaucoma is also used when IOP is very high and field changes are yet to be demonstrated. Acute angle closure glaucoma, malignant glaucoma and other secondary glaucomas are some of the examples.

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13.2 Applied Anatomy and Physiology

Being a gel- and water-filled sphere, the eye needs an intraocular pressure to keep the eyeball inflated. To exchange nutrients and keep the eyeball filled, the eye produces aqueous humour and also has a mechanism to exchange this fluid. Aqueous humour is produced by ciliary body in the posterior chamber. The production of this aqueous is by a combination of active and passive secretion and can be effected by many chemicals. The aqueous from the posterior chamber passes through the pupil and reaches the anterior chamber. The aqueous exits at the angle of the anterior chamber via two pathways (blue track in Fig. 13.1). About 80% of the aqueous gets out via the trabecular meshwork and Schlemm's canal. 20% of aqueous drains via the uveo-scleral pathway on the anterior surface of the ciliary body. From the Schlemm's canal aqueous drains into the systemic circulation via the aqueous veins on the sclera. The trabecular meshwork lies anterior to the scleral spur to which it is attached. This meshwork has pillars of tissue covered with cells that transport the aqueous to the Schlemm's canal which lies on the outside. Blockage of space between the mesh can raise the IOP. From the Schlemm's canal the aqueous escapes via collector channels to the episcleral veins lying outside the sclera. From here it mixes with the venous blood and leaves the orbit via ciliary veins, superior ophthalmic vein to the cavernous sinus.

The iris is thinnest at the root of the iris near the ciliary body. Any build up of aqueous in the posterior chamber due to a block in the flow of aqueous through the pupil results in the peripheral iris bowing out before the pupillary iris is lifted up. This bowing out of the iris occludes the angle, cutting off both the trabecular meshwork and uveo-scleral out flow of aqueous. If the occlusion covers a significant part of the angle then there is a sudden acute rise in the IOP.

The angle structures start anteriorly at the Schwalbe's line, which is the junction between

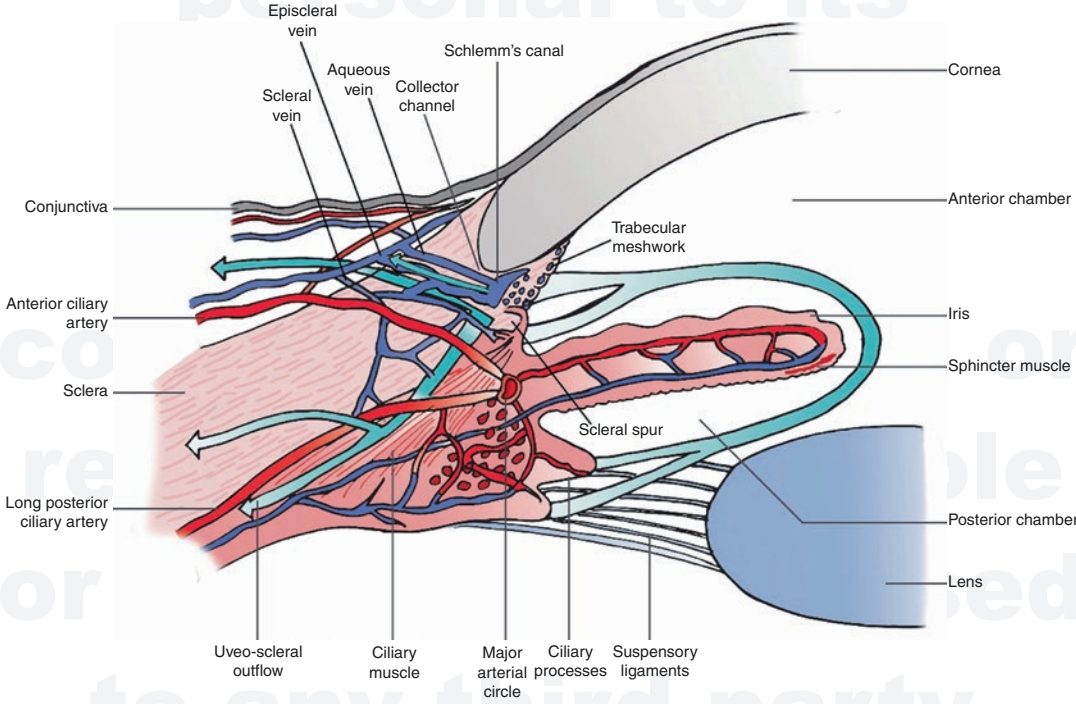

Fig. 13.1 Angle structures showing the site of aqueous production at the ciliary body and the outflow pathways

the cornea and the sclera (Fig. 13.2). The Schwalbe's line is dark in colour. Just posterior to that is the trabecular meshwork. The trabecular meshwork extends from the Schwalbe's line to the scleral spur. The anterior part of the trabecular meshwork is normally white in colour.

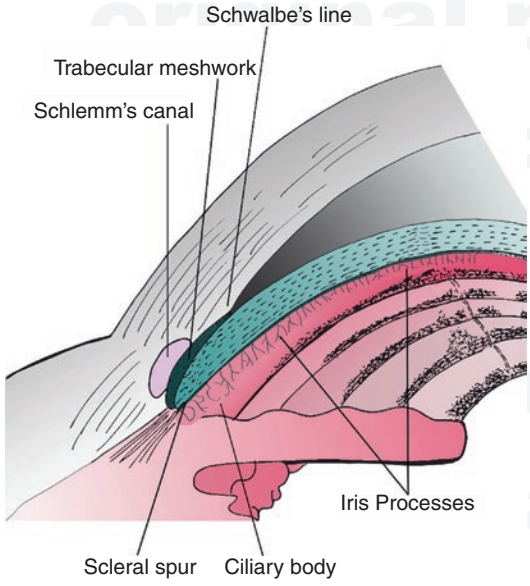

Fig. 13.2 Drawing of the normal angle structures

The middle functional part of the meshwork is more brownish and gets darker with time. The posterior part of the mesh merges with scleral spur. In conditions like pigmentary glaucoma or uveitis the entire meshwork can be coloured with the central part being the most pigmented. Posterior to the trabecular meshwork and just anterior to the ciliary body is a more whitish tissue that is the sclera spur. The trabecular meshwork inserts on the scleral spur which in turn is attached to the ciliary body. On the posterior limb of the angle is seen the ciliary body band from which arise the iris further medially. From the iris arise fine iris processes which go over the ciliary body to reach up to the scleral spur and trabecular meshwork. The angle structures thus appear as a dark, light, dark, light, dark area at the angle.

The optic nerve head receives the nerve fibres which are the non-myelinated axons arising from the ganglion cells of the retina. Since the axons from all over the retina has to pass in an orderly manner through the optic nerve head (ONH) without compromising the function of the macular region, they follow a curvilinear pattern to reach the optic disc (Fig. 13.3). The arcuate fibres from the retinal ganglion cells (RGC) which reach the superior and inferior

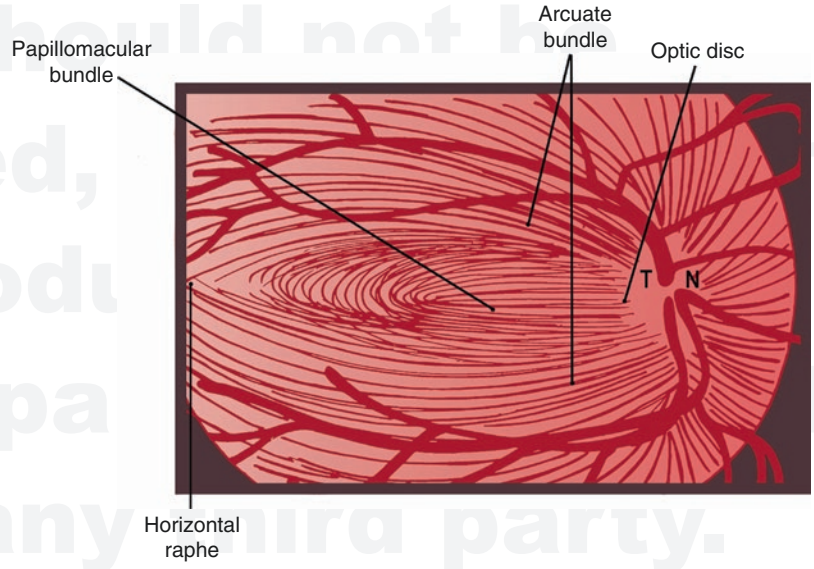

Fig. 13.3 Nerve fibre layer arrangement of the retina and optic disc

margins of the optic disc either seems to be more susceptible to glaucomatous damage or damage in them is more easily detectable by clinical methods. The vertical diameter of the average ONH or disc is about 1.5 mm. In coloured race it can be larger.

The blood supply to the optic nerve head is from the four posterior ciliary arteries arising from the ophthalmic artery before it branches. The short posterior ciliary arteries are multiple small branches of the posterior ciliary arteries given off just behind the globe. They penetrate and supply the prelaminar, laminar (part of the optic nerve at the lamina cribrosa of the sclera) and postlaminar parts of the optic nerve. Some fine branches of the central retinal artery (CRA) also supply the prelaminar branches of the ONH. The inner layers of the retina including the ganglion cell layers are supplied by the CRA.

Besides the RGC axons numbering 1–1.5 million the optic nerve has glial tissue, extracellular matrix and blood vessels. The space that is not occupied by the axons appear as a depression on the optic disc and is called the **optic cup**. The axons of the ganglion cells get out of the eye by going through a posterior sieve like defect in the sclera called lamina cribrosa. The connective tissue beams of the lamina cribrosa can be seen clinically if the cup is very deep. The scleral rim through which the nerve enters the eye is just outside the rim of the optic nerve and should not be mistaken for the optic disc rim.

13.3 Types of Glaucoma

An understanding of the types of glaucoma is necessary to understand the clinical features of the various clinical types. Though the role of IOP is not as central in glaucoma now as it used to be in the past, due to historical reasons and for treatment strategies, glaucomas are still classified using IOP and their reasons for elevation as the basis for classifications.

Traditionally glaucoma have been divided into childhood glaucoma and adult glaucoma. Adult glaucoma is further divided into primary glaucoma and secondary glaucoma. Each of the primary and secondary glaucomas can be divided into open or closed angle glaucomas. Table 13.1 gives the classification with their characteristics.

The prevalence of glaucoma or pre-test probability of glaucoma shows racial variation. In the population over 40 years of age the prevalence among Caucasians is about 1–2%. It increases in coloured population to up to four times that value. Therefore doing, only an investigation on the population to diagnose glaucoma would make no sense. A combination of history and clinical examination should be used first to take the pre-test probability above 40% at least so that any further investigation carried out will be useful and increase the diagnostic post-test probability significantly.

13.4 History

Since a good number of patients with glaucoma go to the ophthalmologist/optometrists for other reasons, history tailored for glaucoma suspects may have to be asked after the examination has started and the doctor has found something suspicious. Family history of glaucoma should be asked routinely to patients in the eye clinic. For patients who may not be aware of the diagnostic entity, questions like anybody being blind in the family, or was told to have raised eye pressures needing eye drops every day, can give us a hint. Patients should be asked for presence of systemic diseases associated with glaucoma. Thyroid eye disease, migraine, use of systemic steroids, history of taking antihypertensive medication (causing low blood pressure at night), snoring at night and ocular trauma are all risk factors for glaucoma and patients should be quizzed about them. It will only be a relative who can tell you about the snore while sleeping! Old ocular trauma is easily forgotten and so one should ask about the games played, involvement in an accident or fist fights, etc. History of severe blood loss in an accident or during surgery may explain a field loss with no other evidence of glaucoma. History of intermittent haziness of

Table 13.1 Types of glaucoma and their characteristics

Disease type	Characteristics	Prototype disease
Primary glaucoma		
Primary open angle glaucoma (POAG)	Typical disc and field changes with no ocular or systemic causes that raised IOP. Open angles on gonioscopy	Age related POAG
Normal tension glaucoma (NTG)	Like above but with no recording of raised IOP	Age related NTG
Juvenile open angle Glaucoma (JOAG)	Like POAG in patients between 5 and 35 years of age	JOAG
Ocular hypertension (OHT)	Elevated IOP but no disc and field changes as yet	Age related OHT
Primary angle closure suspect (PACS)	Iridotrabecular contact of at least 270° on gonioscopy, normal IOP, disc and visual fields	PACS
Primary angle closure (PAC)	Iridotrabecular contact of at least 270°, with either raised IOP and/or PAS but with normal disc and visual fields	PAC
Primary angle closure glaucoma (PACG)	PAC with evidence of disc and field changes	PACG
Acute angle closure glaucoma	Acute rise in IOP to levels high enough to make the patient have headache or eye pain with congestion of the eye	Acute angle closure attack
Sub-acute angle closure glaucoma	Brief episode of raised IOP with above symptoms, narrow angles	Intermittent angle closure glaucoma
Chronic angle closure glaucoma	Elevated IOP with disc and field changes. Angle closed with areas of peripheral anterior synechiae in the angle	CACG
Secondary glaucoma		
Secondary open angle glaucoma	Angles are open with raised IOP due to outflow obstruction caused by underlying ocular or systemic causes	– Pigmentary glaucoma-pigment blocking the trab-meshwork – Angle recession glaucoma – Carotid artery fistula with raised episcleral venous pressure
Secondary angle closure glaucoma due to pupillary block	Pupillary block due to causes like posterior synechiae causes peripheral iris bowing and angle closure with raised IOP	– Seclusio pupillae due to exudates in the pupillary area – Phacomorphic glaucoma
Secondary angle closure glaucoma with no pupillary block	Angles close due to pushing of the iris by the ciliary body or pulling up of the peripheral tissue by fibrous tissue	– Post scleral buckling – Neovascular glaucoma
Plateau iris syndrome	Narrowing of the angle because iris root abnormality and not pupillary block	
Childhood glaucoma		
Primary congenital glaucoma	Dysgenesis of trabecular meshwork	Buphthalmos
Glaucoma with congenital anomalies	Raised IOP associated with ocular malformations Raised IOP associated with extra-ocular malformations	Peters anomaly Sturge-Weber syndrome
Secondary glaucoma in childhood	Raised IOP with either open or closed angle, secondary to an ocular pathology	Retinoblastoma associated raised IOP

vision with headache may be suggestive of intermittent angle closure glaucoma. History of transient ischaemic attack or cardiac problems may explain some of the field defects due to embolic phenomenon in the branches of central retinal artery. Before starting medications one should take a history of obstructive pulmonary airway disease (contradiction for use of beta blockers) and allergies to sulpha group of drugs to which carbonic anhydrase inhibitors belong.

Patients already diagnosed as glaucoma can be asked for previous reports, especially disc photographs and fields. This will give a clue to the progression of the disease. These patients should also be asked about any visual disability like bumping into things, involvement in any accidents, etc. Rarely patients seek help only when they are visually handicapped, in which case an assessment of their functional capabilities with respect to their vision should be done. Many of these patients have an associated cataract.

Patients already on medications should be asked for the list of medications they are on and how they are tolerating their drops. A direct question regarding compliance with the use of drops will seldom give a correct answer. Question about how long a bottle takes to finish, how often drops are missed or under what circumstances one may miss using a drop should be asked. A person who claims to never ever miss a drop is a suspect and is best if a relative corroborates. Side effects of the drops if any should also be enquired into.

13.5 Examination

13.5.1 General Examination

Glaucoma, especially the secondary glaucomas, may have systemic associations that can give a clue to the diagnosis. A cafe au lait spots or swellings all over the body is suggestive of phacomatosis like neurofibromatosis where patients are prone to develop raised IOP. A very tall stature compared to other family members could suggest Marfan's syndrome, the subluxated lens can cause secondary angle closure glaucoma. Obesity if present should be noted and could be a pointer to sleep apnoea. A reddish discoloration on one side of the face with or without hypertrophy is suggestive of Sturge Weber syndrome. Here the raised episcleral venous pressure causes glaucoma.

Thyroid eye disease (TED) is a predisposing factor for glaucoma. Mild lid retraction, prominent eyes and ocular discomfort can all be early indictors of TED.

The upper cheek being pulled back due to maxillary hypoplasia along with microdontia is suggestive of Axenfeld Rieger syndrome which is associated with malformation in the eye and glaucoma.

Having a recording of patient's blood pressure and pulse rate is useful if the patient has never had them checked.

13.6 Ocular Examination

Examination of all parts of the eye have relevance in the diagnosis and management of patients with glaucoma. Of these, measurement of IOP, evaluation of the angle and examination of the ONH is most important.

Vision and refraction should be done not only to get an idea of the patient's disability but also to make sure that the fields are done with the appropriate correction. For patients with very high refractive errors like in aphakia, appropriate contact lens is needed for field assessment.

Hypermetropia is associated with angle closure glaucoma and nanophthalmos. High myopia may be associated with tilted disc which may mimic fields similar to glaucoma. Index of suspicion for glaucoma should be high in a myope. Myopia only in one eye, associated with trauma, may indicate a subluxated lens.

13.6.1 Face and Ocular Adnexa

Before doing the slit lamp examination, the lids, surrounding areas and the face should be examined with a torchlight or bright sunlight. It is worth standing back and looking at the face as a whole. The patient is also asked to open the mouth to look for dentition and high arch palate. On the lids look for any red patches, hyperpigmentation or areas of bogginess giving the upper lid an S-shaped contour. Red patches suggest a haemangioma, hyperpigmentation of the lids and the sclera on one side is seen in nevus of Ota. S-shaped lid with or without nodules on the face is suggestive of neurofibromatosis. All of these

conditions can raise IOP on the side of the lesion. A prominent eye or proptosis may be due to thyroid eye disease. Proptosis with redness of eye and orbital pulsation would suggest a carotid cavernous fistula. Keeping the bell of the stethoscope on the globe may give a bruit. Any scars on the face due to trauma may suggest an ocular injury that was probably ignored at the time of injury. A floppy, easily evertable lid is suggestive of floppy eyelid syndrome with possible sleep apnoea. Abnormally long lashes and sunken eyes are seen in patients who have used prostaglandin analogues over a long time.

13.6.2 Ocular Surface

A combination of slit lamp and torchlight should be used to examine the ocular surface. If the patient is not on any medications mild congestion of bulbar conjunctiva with papillary reaction of the tarsal conjunctiva suggests ocular allergy and patient should be asked about use of topical steroids. Allergy to topical medications including antiglaucoma medications also show a similar picture. Scarring of tarsal conjunctiva suggest old allergies or trachoma. Small stain positive areas on cornea can also be a sign of toxicity to topical medications.

Dilated blood vessel under the conjunctiva in one eye is suggestive of a carotid cavernous fistula or an arterio-venous malformation. A dilated subconjunctival anterior ciliary artery (sentinel vessel) could point to an underlying melanoma and associated glaucoma. A featureless limbus with hypoplastic iris is aniridia that is associated with raised IOP. The presence of a localised elevation of the conjunctiva or scarring in the upper limbus suggestive of a past filtering surgery should be looked for. The conjunctiva may be boggy and elevated in the region of a functioning filtering bleb. There may be an associated defect in the iris in the periphery suggestive of a peripheral iridectomy. If the conjunctiva over the bleb is avascular and thin, a Seidel test (see chapter on examination of cornea) is done to see if there is any bleb leak. Before trabeculectomy the conjunctiva should be examined for limbal scars so that the surgery can be sited in an area away from the scar.

13.6.3 Cornea

Corneal size and clarity should be first looked at with the torchlight before a slit lamp examination is started. A cornea measuring more than 11 mm in the horizontal axis is not normal and glaucoma should be suspected.

Storage disorders like muco-polysaccharidosis show cloudy corneas, this and congenital hereditary endothelial dystrophy (CHED) should be ruled out before diagnosing congenital glaucoma. Storage disorder will show collection of material in the stoma of the cornea. The stroma will be thick in CHED.

Using slit lamp the corneal epithelium is examined for punctate erosions to rule out toxicity to drops. In patients with very high IOP there may be epithelial oedema that can be made out with retro-illumination or sclerotic scatter (see Chap. 6).

Examination of the endothelium will give many a clues to the cause of secondary glaucoma. A copper beaten appearance of the endothelium with pigments is seen in irido-corneal endothelial (ICE) syndrome. Keratic precipitates (KPs) seen as white or brownish white dots on the endothelial surface of the cornea is seen in Posner Schlossman syndrome and other uveitic glaucomas including viral uveitis. Pigment dusting on the endothelium vertically in the form of a cigar (**Krukenberg spindle**) is seen in pigmentary glaucoma. Stellate KPs diffusely scattered on the endothelium point to a Fuchs heterochromic iridocyclitis.

The Schwalbe's line move more centrally (**posterior embryotoxon**) in developmental glaucomas. A horizontal break of the endothelium (**Haab's striae**), best seen on retro-illumination, is seen in congenital glaucoma.

Thickness of cornea should be assessed, though it can be measured with a slit lamp, it is more accurate when measured with an optical biometer or ultrasound pachymeter. Thin corneas measure IOPs artificially low and is a risk factor for glaucoma progression.

13.6.4 Sclera

The sclera can be pigmented in Nevus of Ota. In long-standing cases of high IOP the sclera stretches and the underlying uveal tissue bulges (**staphyloma**) at the limbus or at the region where the anterior ciliary artery pierces the sclera. This should be looked for by lifting the lid.

13.6.5 Anterior Chamber

Anterior chamber (AC) should be examined first with the torchlight and then with the slit lamp. An initial assessment of the depth of the anterior chamber is made using the flash light.

In **flash light test** the light is shown from the temporal side parallel to the Iris plane. This is best done by resting the rim of a 1–2 inch diameter torch on the zygomatic bone laterally (Fig. 13.4) Light from the temporal side should fall on most of the nasal iris if the entire iris is in one plane. If however the central iris comes forward as it happens in shallow anterior chamber, the temporal half of the iris will cast shadow on the nasal iris and it will not be illuminated. Based on the assumption that the amount of iris under the shadow has a relation to the depth of the chamber it has been divided into four grades. Grade 1 is no iris shadow, grade 2 less than one-third, grade 3 is shadow covering one-third to half and grade 4 is shadow covering more than half of the nasal iris. Grade 4 shadow is a poor indicator for angle closure. Its **usefulness is in its negative predictive value**. That is, if the shadow does not cover more than half the iris, then it is unlikely that there will be an angle closure on gonioscopy.

Van Herrick method is another method of assessing the AC depth using slit lamp. Here a thin slit is shown just inside the temporal limbus keeping the lighthouse about 5–10° from the microscope. The corneal thickness is compared to the distance from the endothelium to the iris. If the corneal thickness is equal to the AC depth, it is graded as 4, grade 3 is if the AC depth is half the thickness of the cornea. If AC depth is quarter the thickness of cornea, it was grade 2. Grade 1 was if the AC was less than quarter the corneal thickness. Here again, like flash light test, the usefulness is in the negative predictive value. That is, if the Van Herrick is grade 2 or more, the chance of angle closure is minimal.

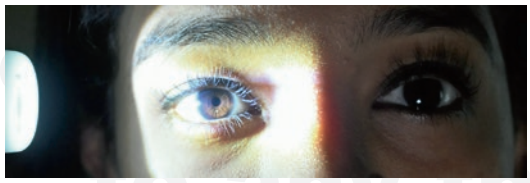

Fig. 13.4 Flash light test; the light is shown from the temporal side parallel to the eye

Irregular shallowing of the AC may be suggestive of a lens subluxation, iris cyst or a ciliary body melanoma.

IOP may be raised in viral uveitis. So the AC should be looked at for cells and flare in cases the raise in OP is unilateral. Posterior synechiae causing iris bombe can be seen uveitis.

Anterior chamber should be checked to see if it is clear and there is nothing hindering the view of the iris. Hyphaema (collection of blood in the AC), hypopyon (collection of white blood cells inferiorly with a horizontal pus level), inverse hypopyon (white emulsified silicon oil seen floating near the upper limbus) with the horizontal demarcation line blow the emulsified oil can all cause raised IOP. Using slit lamp one should look for vitreous in the AC in cases of trauma and in aphakic patients. Vitreous in AC is identified by looking at the dispersed pigments or cells in the anterior chamber. If the pigments are stationary in the AC and not moving along the aqueous currents, it means they are trapped in the vitreous in the AC. In fact the pigment may show a vibratory movement along with the vitreous in which it is entangled. If the vitreous is going to the wound, then pupillary distortion caused by the pull of the vitreous will give a hint to its presence.

13.6.6 Lens

Lens abnormalities itself can be a cause of glaucoma. Careful examination will give hints as to

the aetiology of the raised pressures. This examination is best done with the slit lamp. A mature swollen lens block the pupil and causes a phacomorphic glaucoma. The AC will be uniformly shallow here. In phacolytic glaucoma the lens will be hypermature with a cloudy AC and resembles endophthalmitis; however KPs are absent. On dilating the pupil, pseudo-exfoliative material on the lens will point to a pseudo-exfoliation glaucoma (Fig. 13.5).

A small spherical lens is suggestive of microspherophakia and can cause intermittent angle closure glaucoma if the lens comes into the AC or blocks the pupil. Phacodonesis is a fine tremulousness of the lens seen when the eye makes small saccadic movement as after a blink. This is suggestive of lens subluxation and should be looked for in all shallow ACs. The subluxated lens may cause a pupillary block.

A posterior subcapsular cataract may suggest long-term use of steroids which may be the reason for the glaucoma. Sometimes localised lens opacities especially near the posterior capsule can give field defects that mimic glaucoma.

Anterior chamber intraocular lens can cause pupillary block glaucoma or UGH syndrome. Fine white plaques under the anterior capsule near the pupillary border (glaukomflecken) is suggestive of previous acute rise in IOP as seen in angle closure attacks.

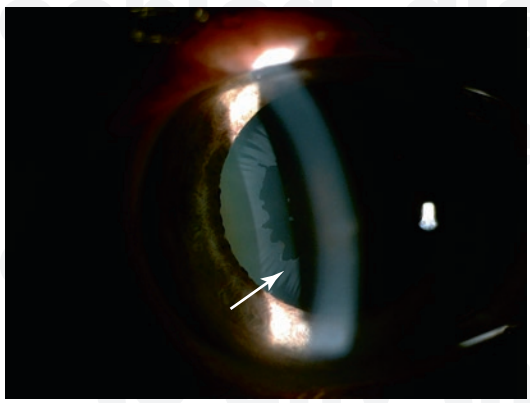

Fig. 13.5 Pseudo-exfoliation on the lens (white arrow)

13.6.7 Pupils and Iris

Relative afferent pupillary defect (see Chap. 11) should be looked for in all suspected cases of glaucoma because it is a very sensitive sign of asymmetrical optic nerve damage. Glaucomatous nerve damage is rarely symmetrical.

Pupillary sphincter rupture will be seen as small discontinuities in the pupillary margins with the torn edges going outward to the iris root. This suggests ocular trauma and may be associated with angle recession. Iridodonesis (tremulousness of iris) if present is added evidence of lens subluxation and trauma. Area of atrophy of the iris near the pupillary border and a vertically oval pupil are suggestive of past attack of angle closure glaucoma. Atrophic areas on the iris can also be suggestive of past attacks of viral uveitis which can raise IOP.

The pupillary edge of the iris should be examined for white flakes like that seen in dandruff. This suggests pseudo-exfoliation which can cause a secondary open angle glaucoma. Iris transillumination may be seen with retroillumination in pigmentary glaucoma and pseudo-exfoliation, especially in patients with light-coloured iris. In patients with heavily pigmented iris this finding is rare.

The colour of the iris in the two eyes should be noted. In nevus of Ota the iris on the affected eye will be darker. In Fuchs iridocyclitis the iris will be muddy with some loss of its features, moth-eaten appearance and lighter in colour compared to the fellow eye in pigmented iris. In light-coloured iris the affected eye may be darker. Nodules on the iris may suggest phacomatosis or uveitis, both of which can cause raised IOP.

Circular and some radial vessels may be seen at the root of the iris in 10% of lightly coloured iris. If these vessels become more prominent and more haphazard in distribution with some new vessels developing at the pupillary margin, then neovascularisation should be suspected. As neovascularisation worsens, the posterior pigmented layer of the iris migrates anteriorly and produces ectropion pupillae. The neovascularisation also causes peripheral anterior synechiae and raised IOP.

The periphery of the iris should be inspected for any laser peripheral iridotomy (PI) done in the past. Retro-illumination can help in the identification especially in pigmented eyes. In light-coloured iris, transillumination can be deceptive and one confirms the patency of PI by being able to see the ciliary body or lens capsule through the iridotomy site.

13.6.8 Gonioscopy

Assessment of the angle of the anterior chamber should be routinely done as a part of the eye examinations in most patients after the age of 40 years. Gonioscopy is one of the most difficult clinical examinations in ophthalmology; competing with indirect ophthalmoscopy for the pride of place. It is therefore all the more important that during training, gonioscopy is performed in as many normal people as possible to become good at picking out the abnormal angle. Seeing into the angle and its structures directly is difficult because the light from the angle does not come out of the eye due to the total internal reflection of the rays at the cornea air interface. The light from the angle has to travel from a denser medium to a rarer medium to be seen by us. To circumvent this a gonioscopy contact lens (gonioscope) has to be used. This contact lens made of either glass or plastic removes the cornea air interface and at the same time changes the angle of exit to allow the light rays coming from the angle to be seen.

Two broad types of gonioscope, direct and indirect, exist (Fig. 13.6). The direct gonioscope of which Koeppe lens is the prototype is a concavo-convex contact lens placed on the cornea. The anterior convexity of this lens is such that the angle of the incident light from the angle is less than the critical angle, thus allowing direct viewing. However for the direct viewing of the angle one had to view from the opposite limbus and cannot use the slit lamp for the same. This lens is used with the patient lying down and operating microscope adjusted so that one looks from the opposite side at an angle of about 80° to the cornea. This lens is useful for examination under anaesthesia and modifications of this lens like Barkan and Swan-Jacob lens can be used for surgical procedures in the angle because the image here is not inverted unlike the mirrored lenses. Fundus examination with a direct ophthalmoscope and high plus is also possible with the Koeppe lens on. To place the lens on the cornea its concave surface has to be filled with methyl cellulose or saline and quickly turned over avoiding air bubbles. The clarity of the angle is better if saline is used but it is still not as good as indirect lenses.

The indirect lenses are the more commonly used lenses in the clinic. Here the anterior surface of the lens is flatter. The diameter and the concavity of the posterior surface depends on the type of lens used. Inside the body of the lens are mirrors placed at an angle of around 62° from the line perpendicular to the iris. The mirror inside the lens redirects the reflected image to the straight ahead direction so that it can be viewed through the slit lamp microscope. Though there are many types and makes of indirect gonioscopy lenses available, only the two main prototypes of these lenses will be described here.

The Goldmann lens is a single or two mirror lens (Fig. 13.6a). The diameter of the posterior concave surface is about 12 mm with a curvature (radius of curvature 7.3 mm) steeper than the cornea. The mirrors inside (if 2, kept opposite to each other) has a tilt of about 62°. To view the angle the

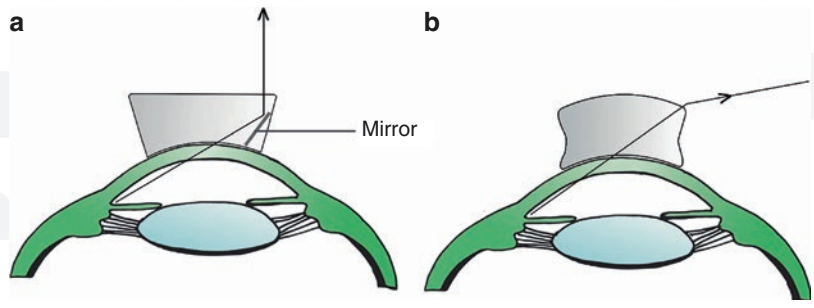

Fig. 13.6 Optics of gonioscopy, (**a**) indirect and (**b**) direct gonioscopy

cup of the concave surface is filled till the brim with coupling fluid having methyl cellulose. The patient's eye is anaesthetised and chin rested on the slit lamp after explaining the procedure and patient told not to move back during the lens insertion. The patient is now asked to look down while the upper lid is held up with one hand. By looking down the patient is not intimidated by the approaching lens and the first point of contact is the sclera. With the other hand the fluid-filled gonio-lens is touched on the lower lid and quickly turned on to the ocular surface before the coupling fluid escapes. By manipulating the lens air bubbles if any that may have been trapped is allowed to escape. A block of wood or sponge may be needed to be kept under the elbow to stabilize the hand holding the lens to facilitate examination.

The angle is best examined with the room dimly lit. A slit illumination is used and the intensity is kept as minimum as possible. The height of the slit beam should be shortened so that no part enters the pupil. This is done so that light in the pupillary area does not cause a pupillary constriction and open up a normally occludable angle. The beam angle is made such that one gets a good slit view of the angle with the light extending from the endothelial side of the cornea over the angle and on to the surface of the iris. The Schwalbe's line at the junction of the cornea and sclera is made out by looking at the intersection of the lines demarcating the anterior and posterior surface of the cornea as it reaches the corneal periphery (Fig. 13.7). Confirming this landmark is especially important in closed angles where a pigmentation on the corneal endothelium anterior to the Schwalbe's line can be mistaken for trabecular meshwork and the closed angle is missed. The corneal endothelial line and the iris line should intersect at the apex of the angle if the entire angle is being seen. If instead of an intersection one sees two separate lines it means the last roll of iris is blocking the view into the angle and one will have to look 'over the hill' to see into the angle. To do this one will have to ask the patient to look towards the viewing mirror. After confirming the position of the Schwalbe's line one will have to identify the trabecular meshwork, sclera spur, ciliary body band, peripheral iris and the iris processes.

The angle viewed through the mirror of the indirect gonioscopic lens has a left to right inversion but the antero-posterior relation is unchanged. To see parts of the angle not covered by a particular position of the mirror, the lens is rotated to bring into view the area that needs to be seen. The view of the angle is end-on. Therefore the angle estimation by looking at the angle of intersection between the corneal and iris ray is inaccurate with lot of variability. A more objective and functionally relevant way to assess the angle is to describe what part of the angle can and cannot be seen. The grading given in Table 13.2 is based on such a philosophy. Along with this one could make an effort to describe if the angle is wide open or narrow, if there is synechiae, the

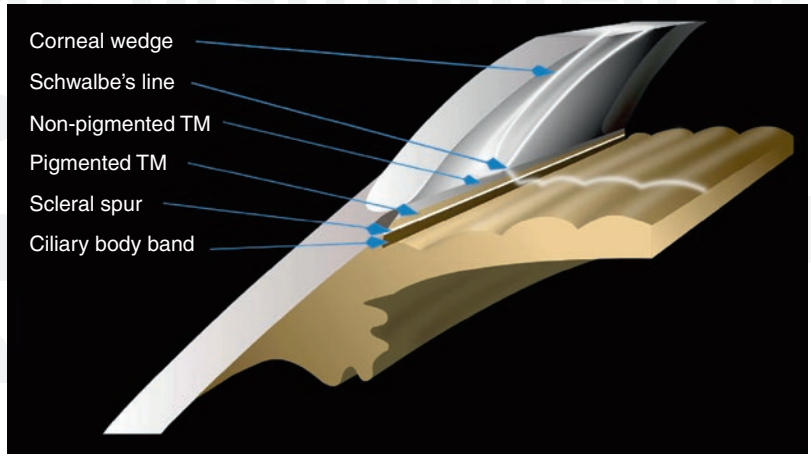

Fig. 13.7 Slit illumination at the angle of a normal eye showing the meeting of anterior and posterior corneal lines at the Schwalbe's line (courtesy of Heiko Philippin, originally published www.cehjournal.org")

Table 13.2 Grading of angle (RPC grading)

RP Centre gonioscopic grading	
Grade 0	No dipping of the beam
Grade 1	Dipping of the beam
Grade 2	Schwalbe's line and ant 1/3 of trab mesh seen
Grade 3	Up to anterior 2/3 of trab mesh seen
Grade 4	Posterior 1/3 of trab mesh seen
Grade 5	Scleral spur seen
Grade 6	Ciliary body visualised

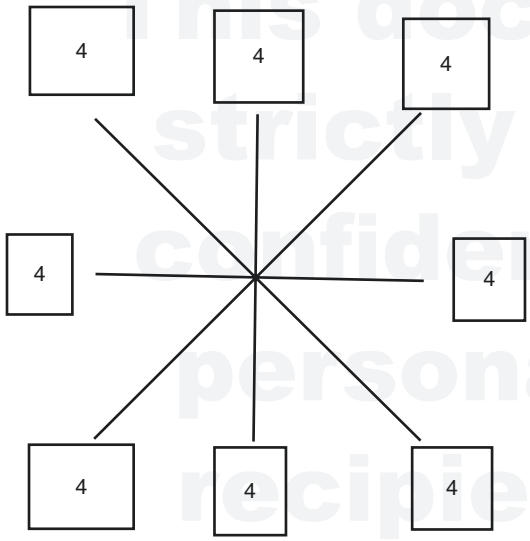

Fig. 13.8 Documentation of angle structure if the angle is open to grade 4 all around

level of insertion of the iris, the integrity of the iris process and any other abnormality seen in the angle. The extent of these findings in terms of the number of clock hours and the location can be noted as in Fig. 13.8.

With large diameter contact lens when pressure is applied to the lens it either presses over the angle and makes it appear closed or presses further into the cornea to open the angle.

The indentation gonioscope or Susman's lens was designed to circumvent some of these issues. It has four mirrors in it placed opposite to each other and at 90° to one pair (Fig. 13.9). These lenses have a posterior diameter of only 9 mm and a radius of curvature of 8.1 mm which is flatter than the corneal curvature. Due to the flatter posterior surface and smaller diameter, there is no need for a coupling fluid and the lens can be directly placed on the anaesthetised cornea to do gonioscopy. The gonioscopy lens should only just touch the cornea. If excessive pressure is put on the lens, it will not only distort the cornea but also open up an occluded angle due to the displacement of the aqueous from the centre of the AC to the periphery as a result of the central indentation by the lens. This central indentation produced when needed is also the advantage of using the lens. If the initial inspection of the angle shows that the peripheral iris is apposed to the cornea, the next question to ask is if this is permanent due to synechiae or just an appositional closure due to forward bowing of the iris. On indenting the central cornea by applying pressure on the cornea with the lens, if iris moves away and angle opens up, it is only appositional closure. If it does not open, then one assumes it is a synechial closure. Sometimes only some areas will open up and this gives us the location of the anterior synechiae. With four mirror contact lens the lens does not have to be rotated to see all around. Shifting view from one mirror to the other would suffice. The extent of the apposition and synechiae in degrees or clock hours should be recorded.

Examination of the angles can reveal early signs of neo-vascularization of the angle. Fine vessels first appear to cross-over the sclera spur and on to the trabecular meshwork. As more new vessels develop the iris gets pulled over the trabecular meshwork to form synechiae. In contrast to this, the vessels in Fuchs heterochromic cyclitis are even finer and synechiae do not develop.

The trabecular meshwork is normally only lightly pigmented in the central part overlying the Schlemm's canal. In pigment dispersion syndrome, uveitis, trauma, following intraocular surgery, melanoma, pseudo-exfoliation syndrome etc., the trabecular meshwork can be heavily pigmented. Normally, blood will not be seen in the Schlemm's canal. Blood enters the canal if the venous pressure outside the eye is high like in

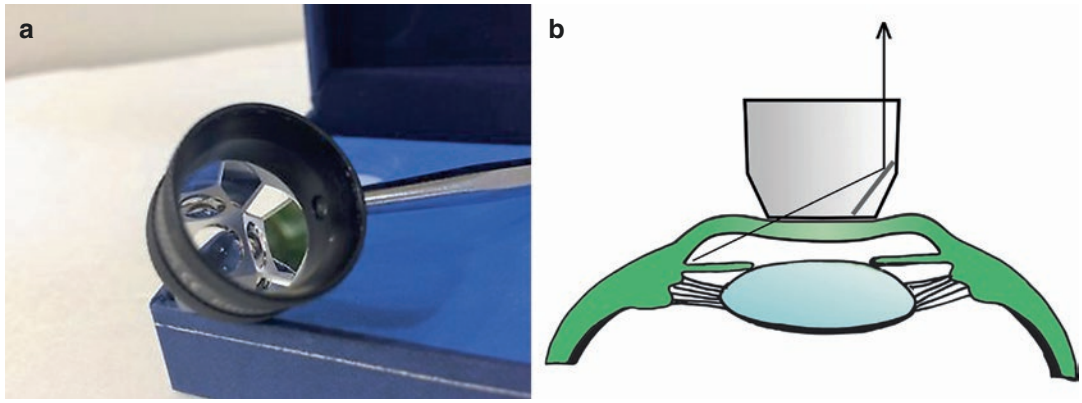

Fig. 13.9 (**a**) 4 mirror Indentation gonioscope. (**b**) Pressure on cornea opening the angles

carotid cavernous fistula or AV malformation. In hypotony when the intraocular pressure is very low, then too blood can enter the Schlemm's canal.

The thin lacy iris process should not be mistaken for peripheral anterior synechiae (PAS). The latter is more thick and broad and obscures the angle. The identification of iris process is important in making out angle recession. In **angle recession** there is disinsertion for the ciliary muscle from the scleral spur. In areas of angle recession, the iris process get disrupted and cannot be seen. On either side of the recession the iris process will be present and is a good method to identify angle recession. In addition, in a recessed angle the scleral spur will be more prominent and the ciliary body band will be wide. If in doubt the same area of the fellow eye should be compared to see the difference. In **cyclodialysis** a gap can be seen between the scleral spur and the ciliary body. The gap may be seen going inward like a cave.

Gonioscopy can also help evaluate the block excision in trabeculectomy, check for foreign body in the angle, look for micro hyphaema or hypopyon, detect early KF ring on the endothelium and pigmentation at the lens equator in pigment dispersion syndrome, etc.

To prevent transmission of infection gonioscopes should be disinfected between patients. Like applanation prisms the lens can be dipped in Daikin's solution between patients and rinsed with saline at the time of use.

13.6.9 Fundus and Optic Disc Evaluation

The techniques of fundus examination is discussed in chapter on examination of the retina. The very basis of glaucoma diagnosis is currently dependant on the clinical findings of the disc and its immediate surrounding areas. Typical field defects is the other feature to diagnose glaucoma.

In glaucoma, examination of the fundus beyond the disc helps us rule out non-glaucomatous pathology that give field defect similar to glaucoma. A branch retinal vein or artery occlusion can give rise to a glaucoma like field defect. Localised chorioretinitis patch in the arcuate area can give isolated scotomas in the Bjerrum's area of the field. A central retinal vein occlusion can be the result of raised IOP. A worsening diabetic retinopathy or laser treatment for the same can cause worsening of the field defect. Vasculitis can cause vessel occlusion and field defects. A retinoschisis or a localised retinal detachment may cause a nasal step like field defect. It is therefore obvious why examination of the entire retina is important before one comments on the field defect or its progression.

The optic disc evaluation is central to glaucoma evaluation. That glaucoma is not a single entity is clear now. It is still not clear what the primary reason for the optic nerve damage is. Is it

an autoimmune disease triggered by raised IOP? Does the primary insult occur in the retinal ganglion cell, optic nerve head, postlaminar optic nerve or even in the lateral geniculate body is not clear. Loss of nerve fibre axons, ganglion cells, blood vessels and glial cells have all been noted histopathologically in these patients. The biggest problem in distinguishing glaucomatous disc from non-glaucomatous ones is the wide variation seen in normal disc of the population. Added to this are the racial variations. One should therefore be well versed with normal variations to suspect if a disc is glaucomatous even before the field defects develop. When field defect is seen, one should try and corroborate with the disc changes. It is said that 20–30% nerve is lost before field defects appear.

As has been mentioned previously the disc rim has the axons and the cup is the area devoid of axons. The disc is best evaluated with a slit lamp so that one can get a three dimensional view of the disc at high magnification. Along with the slit lamp one should use a 60 or 78 D lens to evaluate the disc. The advantage of these lenses is that the nerve fibre layer and macula can be evaluated under higher magnification. If a 90 D lens is used, the magnification of the slit lamp can be increased. When measuring the disc however, one needs to use a multiplication factor with the slit lamp measurement to get the actual measurement of the disc parameters. With a 60 D lens this is not needed. The multiplication factor depends on the lens used. With a 90 D lens if the slit lamp measurement is 1.2 mm, then that value is multiplied by 1.5 to get the actual measurement. With 78 D lens the multiplication factor is 1.1.

The disc is best evaluated after dilating the eye. To examine the right eye, the high power positive lens is held with the right hand between the thumb and index finger in front of the eye between the slit lamp and the eye (Fig. 13.10). An elbow support like with gonioscopy facilitates the examination. The patient is asked to look at the right ear of the examiner with the left eye. In case the patient is one eyed, he is asked to look straight ahead. The lens is kept parallel to the face in front of the pupil and slit lamp moved closer to the eye. As one moves in the disc will start coming into view and

Fig. 13.10 Examination of the disc with a 78 D lens

the slit lamp is focused on the disc. The illumination should be as low as possible. The angle between the microscope and the light housing can be zero degree, 5° or greater (till binocular viewing is possible). By tilting the lens and moving the slit lamp from side to side the disc and its surrounding areas can be examined. For examination further into the periphery of the retina patient should be asked to look to the area of interest and the lens tilted a bit. To measure the disc size the slit light is made coaxial with the microscope. The slit is narrowed and shown on the disc. The height of the slit is now reduced till its edges coincide with the disc margin. The measurement on the slit lamp housing scale multiplied by the correction factor gives the size of the disc. Once the disc is seen an effort should be made to look at the disc margin, identify the scleral opening and examine the neuro-retinal rim, both the margins and the surface. The cup should be identified not only by the paler colour but also by the change in the contour of the surface that is made out on stereoscopic viewing. The dipping of the vessels on the surface of the disc at the margin of the cup will also give a clue to the edge of the cup.

Direct ophthalmoscope has many disadvantages when evaluating the disc. The high magnification restricts the amount of retina one can see

at a time. The lack of stereopsis makes the evaluation of the cup unreliable. The magnification of the indirect ophthalmoscope is not high enough to see the details of the disc clearly. However indirect ophthalmoscopy is mandatory for examination of the retina up to the retinal periphery. Three mirror examination is good but the contact lens examination makes it cumbersome and view of the rest of the eye after an examination is difficult.

Normally the vertical disc diameter is about 1.5 mm. The normal cup size can be 0–0.5. The cup size is described as a proportion of the total disc diameter. The diameter of the disc is given a size of 1. A 0.5 cupping would mean 50% of the disc diameter is occupied by the cup. A cupping of more than 0.7 in either eye should raise the suspicion of a possible glaucomatous change. If an enlargement of cup over and above what is expected with age can be shown by comparing old disc photos, then one is more certain that the cupping is pathological. The area of the pallor being less than the cup area is an indication that the aetiology may be glaucomatous. One should keep in mind that larger discs will have larger cupping because there is only a fixed number of about 1.5 million axons that need to pass through the neuro-retinal rim and the rest of the space has to be made up by the cup. Measuring the disc diameter in every eye will give the examiner an idea of the relation between the disc size and cupping in their population. Between the two eyes if there is a difference of more than 0.2 in the cup size the larger cupped disc is suspect. In these cases also it is worth while measuring the diameter of the disc. If the disc size is the same and the cupping is asymmetrical, then it is more likely that there may be a pathological change. Care should be taken when evaluating a patient with tilted disc. It is easy to miss the cupping in a patient with tilted disc because the cupping is not seen 'end on'. The vertical and horizontal cup size can be different. It is usually only the longer vertical cupping that is measured.

Besides the fallacy in estimating the disc cupping in patients with tilted disc there may be artefactual field defects too in these patients.

The nerve fibre rim or the neuro-retinal rim is not uniformly thick in all around. Though not universal, in majority of the normal people, the inferior rim is the thickest, followed by superior, then nasal and the thinnest rim is temporal. This is called **ISNT rule**. In a glaucoma suspect if you find the inferior rim thinner than the nasal rim, the suspicion of a glaucomatous change should increase. More useful than the overall rim thickness is the detection of localised thinning of the neuro-retinal rim. Usually near the superior or inferior rim one may appreciate a local outward bulge of the cup in an area. This **notch** should be about 0.1 cup in size to be significant. Associated with the notch one may see a **disc margin haemorrhage** or **wedge-shaped nerve fibre layer defect** (Fig. 13.11). There could be a field defect associated with the notch. The neuro-retinal rim is normally not pale in early glaucoma. If there is pallor other causes should be thought of.

The arteriolar branches of the central retinal artery normally follows the rim of the cup on the surface of the disc. In case the cup enlarges it may lie on the bed of the cup and is called '**baring of the circumlinear vessels**'. If the cupping occurs at a level deeper to the disc surface, the vessel that is climbing to come to the surface gets pushed in and the whole arteriole on cross-section looks like a bayonet—called the **bayoneting**

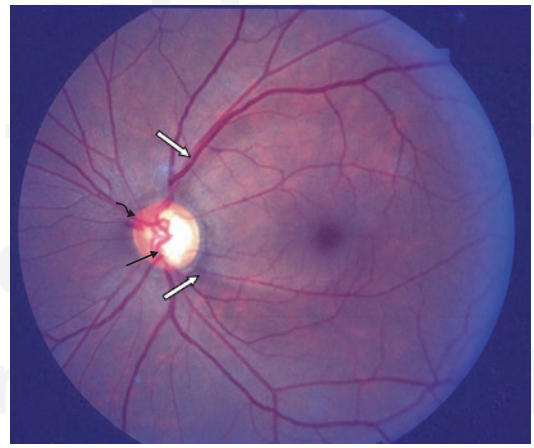

Fig. 13.11 Glaucomatous disc showing notching of the inferior cup (black arrows) along with the arcuate nerve fibre defects (white arrows) and disc margin haemorrhage (curved arrow)

sign. The number of vessel on the neuro-retinal rim is also reduced in glaucoma. Increased prominence of the lamina cribrosa was previously thought to be a pointer to glaucomatous change but does not seem to have much diagnostic value.

The presence of disc margin haemorrhage is abnormal and is thought to be a sign of glaucoma, especially the normal tension glaucoma (Fig. 13.11). Here flame-shaped haemorrhage is seen at the junction of the disc and the retina. The haemorrhage is transient and in the region of the haemorrhage one sees nerve fibre layer loss and associated field defect later. Other causes of disc margin haemorrhage like posterior vitreous detachment, diabetic retinopathy, branch vein occlusion, etc. should be ruled out.

The nerve fibre layer (NFL) of the retina is best seen with the red free light. It is seen as a white fibrillar sheen around the disc, more clearly seen near the superior and inferior pole of the disc. Wedge-shaped defects in this sheen (Fig. 13.11) starting at the disc margin is suggestive of glaucomatous nerve fibre loss. There may or may not be an associated neuro-retinal notching and a field defect corresponding to the NFL defect. The significance of the NFL defect increases if there is a corresponding visual field defect. Thin streaks of NFL defect can be normally seen in people and has no clinical significance. Sometimes with the NFL loss, before the cup enlarges fully, the glial tissue at the rim hold the vessels up. This is called the **over pass phenomenon**, i.e. the blood vessels floating over a collapsed disc below.

Peripapillary atrophy can be seen in normal people, especially the myopes. There are two morphological types seen. Alpha zone atrophy seen as an area of hypo- and hyper-pigmentation around the temporal disc (temporal crescent) is mostly in myopes and is not associated with glaucoma. Beta zone atrophy has a characteristic whitish appearance and represents the loss of choriocapillaries and retinal pigment epithelial layer in that region. This is seen more in areas where there is an associated field defect. The alpha zone lies outside the beta zone (Fig. 13.12).

With the current ease of availability of disc photography a disc photograph is the best record

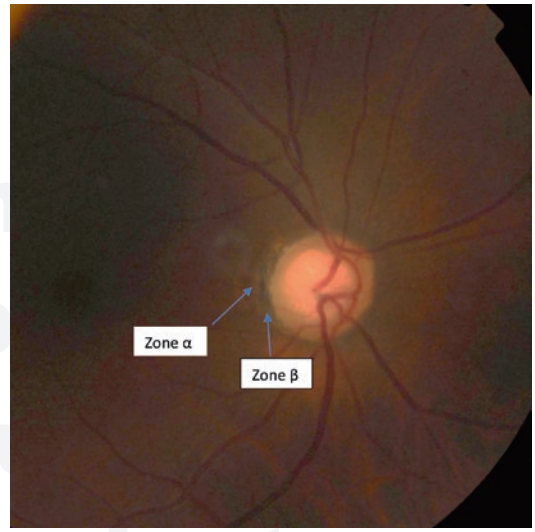

Fig. 13.12 Disc showing alpha and beta zones

for glaucoma patients. One can draw a diagram of the disc marking the cupping, notching, NFL defects, etc. on it. The size of the disc and amount of cupping should also be mentioned.

When evaluating the disc in glaucoma one isolated sign mentioned above may not have much significance. Two or more signs appearing together and matching with the rest of the finding and history should give one a better confidence in the diagnosis. One should also keep in mind other optic nerve pathologies like optic disc pit, coloboma, Drusen, myopic disc, tilted disc, etc. when evaluating a patient with suspected glaucoma.

Finally, assessing the glaucomatous disc is a pattern recognition. It is only a matter of time before artificial intelligence will classify the disc better than humans. However to pick up all other features of clinical presentation and put it together will still need doctors!

13.7 Intraocular Pressure Measurement

Measuring the IOP is central to the current management of glaucoma. However it has so many factors affecting it that the absolute number one gets when measuring IOP is not important. There

is confusion as to what the normal IOP distribution pattern is. Is it Gaussian (parametric) at all? Is it skewed to the right? Is it Gaussian in some age groups? The classical teaching of the normal range of IOP being in between 10 and 21 mmHg is based on the assumption that IOP distribution in the population is Gaussian when in fact it may be skewed to the right. Due to the variability in pressures seen in patients with glaucoma, it is difficult to define normal IOP in terms of the pressure that do not cause glaucoma! Added to this is the variation of IOP with age, race, sex, the time of day it is measured, the position in which it is measured, the instrument used to measure, etc.

IOP is thought to increase with age probably due to a reduction in outflow facility. Asians and Blacks have higher IOP than Caucasians. Females after menopause are said to have higher IOPs. Diurnal variation of IOP is a story of variation in itself! Classically it is thought that most people have the highest IOP is in the morning; but peaks at midmorning, afternoon have been reported. More important than the actual peak is probably the fluctuation. A difference of more than 10 mmHg IOP during 24 h is thought to be pathological. 6 mmHg is suspect. While a 24 h measurement is ideal, practically one could take measurements in the clinic every 2 h for the whole day to get an idea. The supine position increases the IOP. The equipment used to measure the IOP also affects the reading. In spite of all this IOP is the only parameter we can control in the management of glaucoma.

13.7.1 Applanation Tonometry

The gold standard for measuring IOP is still the Goldman applanation tonometer (GAT). In all measurements of IOP the basic assumption is that the amount of deformation of the globe by whatever method is affected by the pressure in the eye. The more the IOP, more difficult the deformation will be.

In GAT the applanating cylinder/prism is touched on the cornea in such a way that a prefixed area of the cornea is flattened. The force required to applanate is dependent on the intraocular pressure (Imbert-Fick principle). The applanation tonometer comes as an extra attachment to the slit lamp. Once attached, it can be moved into position before measuring the IOP. The eye is anaesthetised with proparacaine first. Then a drop of fluorescein is used to stain the tears. The applanating prism should not be used directly from Dakin solution in which it is kept to prevent transmission of infection from patient to patient. The prism is washed in sterile saline, dried and mounted on the prism holder in the applanation tonometer. It is kept in such a manner that the red mark on the prism coincides with the white mark on the holder (Fig. 13.13). The stem of the holder should be supported while inserting the prism so as to not affect the calibration. The measurement knob on the side of the tonometer is turned so that the 1 mark (not zero) on its circular scale coincides with measurement spot on the side of the applanation box. It is at the 1 reading that prism balances with the counter

Fig. 13.13 Applanation tonometer with the prism fixed correctly

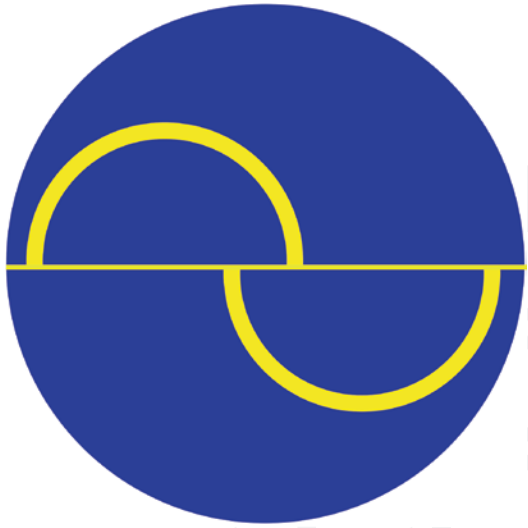

Fig. 13.14 View through the applanation prism showing the mires of the cornea

weight and is floating freely to move either side. Keeping the prism coaxial, the beam of light is made a full spot with maximum brightness. The lighthouse is kept in such a way (usually at 60° angle to the microscope) that all the light falls on the side of the prism. The cobalt blue filter is now put on and the prism viewed monocularly through the eyepiece which is aligned to the prism. One will see a blue circular disc divided by a horizontal line (Fig. 13.14). The patient is now told that his eye is anaesthetised and that he should keep the eye open while the prism is brought in contact with the cornea to measure the IOP. As the prism approaches the eye, before it touches the cornea two blue half circles will appear on either side of the central horizontal line and signifies that the prism is about to touch the cornea. These semicircles should be symmetrical. If not, make it symmetrical by moving the slight lamp up or down. Moving the slit lamp up or down after the cornea is touched can abrade the cornea. Now gently move in a bit further till the two symmetrical circles become greenish (Fig. 13.14) signifying that the prism has touched the tear film. These rings may or may not intersect with each other. Now turn the knob on the side till the two circles overlap in such a way that the inner aspect of the two half circles just touch each other. If the circles pulsate significantly, then the circle is made to overlap in such a way that the oscillation due to the pulsation is equal on both sides of the end point. A slight pull back of the slit lamp after the measurement should disengage the prism from the cornea. If that does not happen it means the prism has been pushed in too much to take the measurement and that will underestimate the IOP. Again if the fluorescein ring is too thick due to excessive tears it will over-estimate the IOP and vice versa if it is too thin. Eye should be wiped gently or more fluorescein put to make the rings optimal. Measuring without holding the lid would be ideal. However due to reflex blinking you may need to hold the lid up. If the lid is help up by the examiner, one should ensure that the patient does not squeeze the lid and the examiner does not press on the eye as both can increase the IOP. Once the end point is reached the slit lamp is drawn back and the prism taken out after stabilising the holder with the examiners fingers. The prism is put back into the Dakin solution. Alternatively hydrogen peroxide solution can be used. The reading is measured off the circular scale. 1 corresponds to 10 mmHg and 2, 20 mmHg.

In patients with thyroid eye disease IOP is also measured in the up gaze position by asking the patient to look up; before looking up, the patient's chin is tilted down so that the cornea realigns in the straight ahead position when the patient looks up. An increase in IOP of over 6 mmHg suggests that the muscles are tight and the IOP may increase even in the sleep due to Bell's phenomenon.

If the corneal astigmatism is high, the IOP measurement in the standard horizontal position of the prism is not accurate as the mires will not be circular. To counter this one can measure the IOP with the prism line horizontal and vertical and then take an average. Alternatively there is a red line on the prism that should be aligned to the flatter axis of the cornea by using the degree mark on the prism holder.

Since the IOP measured by the applanation tonometer is affected by corneal thickness and

corneal thickness by itself is a risk factor (thin corneas increase the risk of glaucoma), corneal thickness is measured using ultrasound or light pachymeters and recorded if the equipment is available. Though correction factors have been described based on the corneal thickness it is not very accurate.

13.7.2 Calibration of Tonometer

Any equipment that measures need to be calibrated regularly to ensure no additional errors due to the equipment creeps in. The applanation tonometer has a calibration rod that is fixed to a slot on the side of the applanation box to do the calibration. The manufacturer gives the instructions for calibration. Calibration should be done once a month.

In presence of corneal oedema and scars, applanation measurements are not accurate and an electronic tonometer like Tonopen is better. Since a tonopen is not available as a standard equipment in the examination cubicle it is not described here.

13.7.3 Digital Tonometry

Digital tonometry is only useful to make assessment of IOP at the extreme ends of the IOP spectrum. This is really useful only in a non-clinic situation like in a non-ophthalmology ward, emergency room or in the clinic when patient is not co-operative. Realistically only three readings are possible by this method, very low (less than 5 mmHg), very high (more than 40 mmHg) and 'in between somewhere'!

To do the digital tonometry, patients should be asked to look down (not close the eye) so that the hard tarsal plate moves down and out of the way of the upper part of the lid through which our fingers feel the globe. Now globe should be alternatively pressed with the two fingers above the tarsal plate. Essentially what one is doing here is looking at the ease of fluctuation in the globe as a surrogate for the IOP (Fig. 13.15).

Fig. 13.15 Examination of intraocular pressure using digital tonometry

With the patient looking down the outer three fingers of both the hands is rested on the forehead to take the weight of the forearm. Now the index fingers of both the hands is kept on either side of the globe and pressed alternatively. When one finger presses the other feels the transmitted pulsation. The softer the eye greater is the movement of the globe wall. This test can really only rule out a very soft eye or a very hard eye. This is ideally useful to assess a patient in the emergency room who comes with one sided headache and vomiting to rule out angle closure glaucoma. A very soft eye in a patient after a traffic accident could suggest an occult rupture of the globe.

13.7.4 Indentation Tonometry

Indentation tonometry done using Schiotz tonometer is the other method of assessment of the IOP in the clinic without the use of expensive equipment. Schiotz tonometer has a central plunger located in the middle of a footplate of the equipment. With the footplate rested on the cornea the central plunger can move up or down. The amount of downward movement on the plunger depends on the IOP. Softer the eye the more the plunger moves in. The plunger is attached to a scale which then reads off the IOP based on a conversion table that is available with the tonometer. To get more reliable conversions, the scale readings should be within two and five divisions. To ensure this weights are added to the plunger so that any IOP measurement can be brought to this

Fig. 13.16 Examination of intraocular pressure using Schiotz tonometer

reading. Since indentation is dependant on the scleral rigidity the validity of this measurement is not good.

The measurement is taken with the patient lying down (Fig. 13.16). After explaining to the patient that the tonometer will be kept on the eye to measure the IOP the eye is anaesthetised with topical proparacaine. Keeping the lid open with two fingers of one hand the tonometer is placed on the cornea using the handle of the tonometer. The reading when the footplate is fully rested on the cornea gives a value on the scale which should be then converted to the IOP reading using the conversion sheet given with the tonometer. With hand held applanation tonometer (Perkin's tonometer) and Tonopen available for patients who cannot sit at the slit lamp the use of indentation tonometer is limited to outreach work in remote areas.

In spite of all the uncertainties, because of the centrality of IOP in our management strategies it is important to measure IOP as accurately as possible. These measurements are most useful when we are measuring the IOP for the same individual with the same instrument, multiple times. It is also important to give IOP no more importance than it deserves especially considering all the variability associated with its measurements.

13.8 Field Examination

For glaucoma evaluation and management, in the present day there is no role for examining the visual fields with methods like confrontation, tangent screen, etc. Doing the same for a glaucoma patient even in exit examinations for trainees is not justified.

Investigations: Automated fields evaluation, nerve fibre layer and ganglion cell study using OCT, disc photo evaluation using artificial intelligence and electronic diurnal IOP monitors are the important investigations that will aid one in further evaluation and management of glaucoma patients. Students will need to refer to specialist books on these subject to understand the scope of these investigations better.