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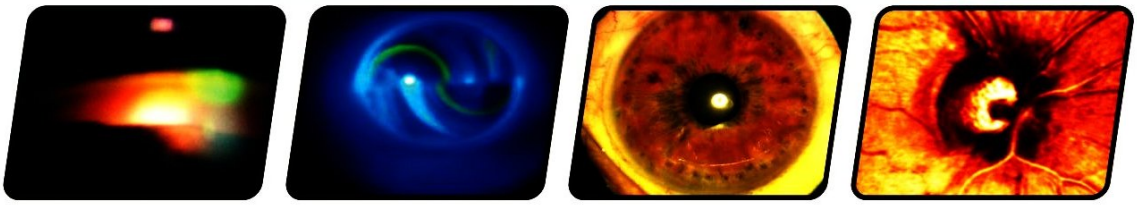
World Glaucoma Week

6th March - 12th March 2016

Glaucoma Update



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FROM EDITOR'S DESK

Dear Friends ,

Seasons Greetings

It's matter of immense pleasure for me that after launching 3rd volume Nov 2015 successfully, now we are here coming with edition on Glaucoma Update. We all know that Glaucoma being 3rd leading cause of blindness in India contributing to 12.8 % of countries blindness approximately 12 million people .

As we are aware about Glaucoma week (6th – 12th March 2016) and under the banner of BDOS we planned many activities like Glaucoma walk, Glaucoma Rath, Meet the experts and Question answer session in Glaucoma and in the same context we dedicate this complete volume exclusively to Glaucoma diagnostics and management. Now also looking forward to a CME on Glaucoma. While editing this issue, I realised that glaucoma as a super-speciality is developed in our city too. The current Text is an attempt to demystify the diagnosis and management to a level where general ophthalmologists will find themselves at ease in diagnosing glaucoma at an early stage.

I am grateful to all authors of this issue who have done a commendable job. I also observed this time that members of BDOS has shown a keen interest in terms of writing skills and let it not be a part of teaching fraternity only. My special thanks to Dr. Prateep Vyas Sir for his valuable notes.

I acknowledge constant motivation and moral support that I received from all of you. I also acknowledge the support from R.K.D.F. Medical College Hospital and Research Centre. Looking forward to your views and opinion about this issue so that improvements can be done at finer level.

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CLASSIFICATION OF GLAUCOMA

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Early diagnosis and intervention in glaucoma requires a good clinical knowledge about the changes occurring in glaucoma. IOP is still the most studied risk factor in glaucoma and the classification systems depend upon the mechanism of aqueous outflow. Mostly the current treatment strategies are based on reducing IOP; an understanding of mechanism of aqueous outflow obstruction is a rationale for early glaucoma intervention by controlling IOP in each type of glaucoma. [1]. Knowledge of a detailed classification system is required to approach these cases.

The main disadvantage of a classification based on the aqueous outflow obstruction is that it ignores the causes unrelated to IOP. Many of the glaucoma may have more than one mechanism of outflow obstruction at different times during the course of the disease.

Barkan,[2] first recognized the distinction between open-angle and angle-closure types of glaucoma, which led to the basis for the mechanistic classification of glaucoma. The most widely used classification system of glaucoma separates the angle-closure glaucoma from open-angle glaucoma. This fundamental distinction still holds.

Historically, angle-closure disease has been variably defined in terms of pupillary block mechanism of irido-trabecular obstruction that leads to functional angle closure.

In open- angle glaucoma, there is relative impairment of aqueous outflow through trabecular meshwork-Schlemm`s canal-venous system; on gonioscopy angle appears to be open with emphasis that patient retains some vision till the last but manifest as optic nerve damage and ganglion cell demise.

Becker-Shaffer proposed Simple glaucoma classification into three major divisions, (1) angle-closure glaucoma; (2) open-angle glaucoma; (3) developmental glaucoma.

A similar classification system divides glaucoma into conditions that affect internal flow (pupillary block or malignant glaucoma), and condition that affect the outflow of aqueous humor (neovascularization at trabecular meshwork or compromised schlemm`s canal, collector channels and the elevated episcleral venous pressure)[3]

Alternative classification system [4] are based on – (1) the site of outflow obstruction divided into at the level of pre-trabecular passage (e.g. posterior synechiae), at the level of trabecular flow (e.g.glaucoma after administration of alpha-chymotrypsin), and the level of post trabecular flow (e.g. elevated episcleral venous flow); (2) the tissue principally involved (e.g. glaucoma caused by diseases of lens or retina); (3) the proximal initial events (e.g. steroid induced glaucoma); (4) the age of patient (e.g. congenital, juvenile). However few cases may share the features of both open angle and angle closure glaucoma these cases are now reclassified into mixed mechanism glaucoma.

One should understand that all classifications are arbitrary and limited. As the pathogenesis of glaucoma is not completely understood so some cases may not fit completely into one category or another. Also with time as the disease progresses they may need to be reclassified. Thus there is no single classification which is complete, but mostly includes all possible pathophysiology studied so far. A thorough knowledge of this aids in making early diagnosis and intervention on the basis of different pathogenetic mechanisms that leads to glaucoma.

Classification of glaucoma based on mechanisms of aqueous outflow obstruction -

OPEN ANGLE MECHANISMS:ANGLE CLOSURE MECHANISMS:

	A. Pre- overgrowth trabecular (membrane)		**A. Anterior ("pulling" mechanism)**
1.	Fibrovascular membrane	1.	Contracture of membrane
2.	Endothelial layer, often with Descemet-like membrane- a. iridocorneal endothelial syndrome b. posterior polymorphous dystrophy c. penetrating and non-penetrating trauma		a. Neovascular glaucoma b. Iridocorneal endothelial syndrome c. posterior polymorphous dystrophy d. penetrating and non-penetrating trauma
3.	Epithelial down growth	2.	Contracture of inflammatory precipitates
4.	Fibrous ingrowth		B. Posterior ("pushing" mechanism)
5.	Inflammatory membrane – a. fuchs heterochromic iridocyclitis b. luetic interstitial keratitis	1.	With papillary block – a. Pupillary block glaucoma b. Lens-induced mechanisms – (1) Intumescent lens (2) Subluxation of lens (3) Mobile lens syndrome c. Posterior synechiae – (1) Iris intraocular lens block in
	B. Trabecular (occlusion of intertrabecular spaces)		
1.	Idiopathic - a. Chronic open-angle glaucoma b. Steroid induced glaucoma		

2.	Obstruction of trabecular meshwork – a. Red blood cells – 1) Hemorrhagic glaucoma 2) Ghost cell glaucoma b. Macrophages – (1) Hemolytic glaucoma (2) Phacolytic glaucoma (3) Melanocytic glaucoma c. Neoplastic cells – (1) Malignant tumors (2) Nevus of Ota (3) Juvenile xanthogranuloma d. Pigments particles – (1) Pigmentary glaucoma (2) Exfoliation syndrome (3) Uveitis (4) Malignant melanoma e. Protein – (1) Uveitis (2) Lens-induced glaucoma f. Viscoelastic agents – g. Alpha-chymotrypsin-induced glaucoma h. Vitreous		pseudophakia (2) Uveitis with posterior synechiae (3) Iris-vitreous block in aphakia
		2.	Without pupillary block – a. Plateau iris syndrome b. Ciliary block (malignant) glaucoma c. Lens-induced mechanisms (1) Intumescent lens (2) Subluxation of lens (3) Mobile lens syndrome d. After lensextraction(forward shift) e. Secondary to sclera buckling surgery f. Secondary to panretinal photocoagulation g. Central retinal vein occlusion h. Intraocular tumors – (1) Malignant melanoma (2) Retinoblastoma i. Cyst of the iris and ciliary body j. Retrolenticular tissue contracture– (1) Retinopathy of prematurity(retrolental fibroplasias) (2) Persistent hyperplastic primary vitreous
3.	Alterations of the trabecular meshwork a. Edema – (1) Uveitis (trabeculitis) (2) Scleritis and episcleritis (3) Alkali burns b. Trauma (angle recession) c. Intraocular foreign bodies (hemosiderosis, chalcosis)		**DEVELOPMENTAL ANOMALIES OF THE ANTERIOR CHAMBER ANGLE:**
C. Post-trabecular		A.	High insertion of anterior uvea 1. Congenital (infantile) glaucoma 2. Juvenile glaucoma 3. Glaucomas associated with other developmental anamolies
1.	Obstruction of schlemm`s canal – a. Collapse of canal b. Obstruction of schlemm`s canal (e.g. sickled red blood cells)	B.	Incomplete development of trabecular meshwork/Schelmm canal 1. Axenfeld-Rieger syndrome 2. Peters anamoly 3. Glaucomas associated with other developmental anamolies.
2.	Elevated episcleral venous pressure – a. Carotid-cavernous fistula b. Cavernous sinus thrombosis c. Retrobulbar tumors d. Thyrotropic exophthalmos e. Superior venacava obstruction f. Mediastinal tumors g. Sturge-Weber syndrome h. Elevated episcleral venous pressure	C.	Iridocorneal adhesions 1. Broad strands (Axenfeld- rieger syndrome) 2. Fine strands that contract to close angle (aniridia)

The ongoing revolution in genetics and molecular pathophysiology of glaucoma may change this classification in future. But as per the current understanding of the disease the above classification includes almost all possible causes and mechanism.

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GLAUCOMA SUSPECT

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Glaucoma suspect describes a person with one or more risk factors that may lead to glaucoma, but this individual does not have definite glaucomatous optic nerve damage or visual field defect. A great overlap can exist between findings in patients with early glaucoma and those who are glaucoma suspect without the disease. The clinical findings that define a glaucoma suspect are characterized by one or more of the following-

- Appearance of the optic disk or retinal nerve fiber layer suspicious for glaucomatous damage.
- A visual field suspicious for glaucomatous damage.
- Consistently elevated intraocular pressure (IOP) associated with normal appearance of the optic disk and retinal nerve fiber layer and with normal visual field test results.

Broadly we can divide suspects into two varieties – (A) angle closure glaucoma suspect and Open angle glaucoma suspects. Open angle suspects further could be (B) Normal Tension Glaucoma (NTG) suspects and (C) Ocular Hypertensive (OHT), all 3 will be discussed in detail below.

Worldwide more than 100 million people have elevated IOP and more than 3 million people are blind secondary to POAG; about 2.4 million people develop POAG each year. In India, glaucoma is the third leading cause of blindness with 12 million people affected accounting for 12.8% of the countries blindness. Population-based studies report prevalence between 2 and 13%.

The following ocular conditions that have been implicated as risk factors for developing glaucoma--

- **Myopia:** This is a risk factor for glaucoma; but disc evaluation (myopic fundus, tilted disc) and visual field testing (fundus abnormalities, refraction-related inaccuracies) are difficult to differentiate myopic damage and glaucoma.
- **Pseudoexfoliation:** Increases with advanced age.
- **Pigment dispersion:** 25%-50% risk of developing glaucoma.
- **Ocular trauma**
- **Glaucoma in one eye:** Associated with increased risk of future damage in the other eye. The development of visual field defects in an average of 5 years in about 29% of untreated undamaged fellow eye.
- **History of uveitis/inflammatory ocular disease**
- **Congenital anomalies**
- **Prior eye surgery**
- **Retinal vascular occlusion:** In susceptible individuals, increased IOP associated with a risk of developing central retinal vein occlusion.
- **Current or past use of steroids:** steroids in any form may elevate pressure in certain individuals, usually seen within a few weeks of starting topical steroids. Optic nerve damage may be residual from previous increased IOP associated with steroid use.

The following systemic conditions that have been associated as risk factors for developing glaucoma-

- **Low blood pressure:** also includes overmedication of systemic hypertension.
- **A previous episode of hypotensive shock, trauma, vascular surgery or hemorrhage** leads to onetime insult to optic nerve damage.
- **History of vasospastic disorders**: A higher prevalence of migraine headaches and Raynaud syndrome exists with normal-tension glaucoma.
- **Medications:** In susceptible individuals, steroids may cause a rise in IOP; anticholinergics (antihistamine and antipsychotics) may precipitate angle-closure glaucoma.
- **Cardiovascular disease** may be a factor in low-tension glaucoma.
- Hypertension
- **Diabetes mellitus:** Small association; some studies have reported a higher prevalence of increased mean IOP and POAG with diabetes mellitus.

- **Smoking** is risk for development of glaucoma while Omega-3 fatty acids have been associated with a protective effect.
- **Family history** is a definite risk factor, nearly 10-20% of patients with glaucoma have a positive family history. The Baltimore Eye Survey showed risk of having glaucoma is increased 3.7-fold for individuals who have siblings with POAG.

WORK UP IN CASE OF GLAUCOMA SUSPECT—

(1) **History:** ocular and systemic is very important to rule out all risk factors mentioned above.

(2) **Review of old records:** Note previous IOP, cup-to-disc ratios, ocular surgery and past visual fields. These old documents will help to compare today's findings with past (Caution: Poor agreement and observer variability can occur in disc examinations over time.)

(3) **Examination;** vision, detail anterior segment examination, gonoioscopy all are mandatory. other workup includes-

Tonometery: Applanation tonometery is considered gold standard for glaucoma diagnosis and management. Careful Schiotzs or noncontact tonometer multiple readings can be used for mass screening. Corneal pachymetery is must in all glaucoma suspects because abnormally thick corneas may result in artificially high IOP measurements, while thin corneas may result in artificially low IOP measurements by applanation tonometery. Elevated IOP is a definite and important risk factor for developing glaucomatous damage but is not sufficient for a diagnosis.

Evaluation of the optic nerve head: The best examination method is a slit lamp combined with a 60-D, 78-D, or 90-D Hruby lens or a posterior pole lens through a dilated pupil, which offers the benefits of high magnification, stereoscopic view with excellent illumination with special attention to be given to contour of cup then color. It is important to document optic nerve appearance by stereo disc photographs or detailed description and drawings for future comparison. Automated techniques can be used like Scanning lasers and polarimetery having advantages of accurate, reproducible method with patient comfort (nondilated) but disadvantage is the cost.

Evaluation of the retinal nerve fiber layer: The nerve fiber layer defects/dropout can be detected by ophthalmoscopic examination with red-free (green) filter and documentation by good color photography or red-free photography for future reference. The automated techniques of Scanning lasers ophthalmoscopy (HRT) or Polarimetry (GDx) Or Coherence tomogragphy (OCT) though accurate, but are expensive.

Visual field testing: Results of visual field testing should be normal in glaucoma suspect. But absence of visual field defects does not ensure absence of glaucoma because, as many as 50% of optic nerve fibers in a single optic nerve may be damaged before visual field defects are found by Goldmann Perimetery. Proper interpretation of visual field testing and its comparison with similar type of previous fields is very important. Short wavelength automated Perimetery (SWAP) uses blue target on a yellow background to isolate those visual pathways that are believed to be damaged selectively in early glaucoma, and is useful in detecting progression to glaucoma in high risk glaucoma suspect. Frequency-doubling technology Perimetery uses a coarse striped grating of rapidly alternating dark and light bands. It is effective in monitoring visual field progression and that it may detect field defects earlier than standard automated Perimetery.

TREATMENT:

Decision to treat glaucoma suspect or to follow, depends on interplay of various factors like risks and the rate at which glaucomatous damage and decreased visual function can occur, the patient's desires, expected longevity, and tolerance of treatment. Carefully weigh the risk factors and individualize the decision to treat glaucoma suspect. Other factors which also contribute in decision are ability to examine the optic disc, reliability of visual field testing and availability of follow-up visits. if patient is kept on followup then the frequency and the composition of follow-up evaluation depend on many factors like the age of the patient, the level of elevation of IOP, the appearance of optic nerve head cupping, a family history of glaucoma, the presence of additional risk factors and the stability of the patient's clinical course. On every followup visit check IOP, detail optic nerve assement, Gonioscopy and fields (every 3- 12 months) and compared with baseline documents. Treatment can be in the form of drug or laser. Once treatment is initiated principle remains the same as any glaucoma treatment to set target IOP and follow. Failure to achieve and maintain a target pressure should trigger a reassessment of the treatment regimen in light of the potential risks and benefits of additional or alternative treatment.

CONCLUSION:

Most of glaucoma suspect do not develop glaucomatous optic nerve damage and/or visual field loss. Nearly 2% of individuals with ocular hypertension develop glaucoma per year; the risk is higher for patients with additional risk factors. Glaucoma causes silent damage; follow-up is essential to exclude any progressive change over time that may warrant treatment. Left untreated, patients with optic nerve damage may progress, resulting in progressive loss of nerve fibers and eventually total optic nerve atrophy and irreversible blindness.

(A) PRIMARY ANGLE CLOSURE SUSPECT-

A person with 180 degree or more of irideotrabecular contact in primary gaze on gonioscopy with no peripheral anterior synechia, and normal IOP is considered a primary angle-closure suspect (PACS).Prevalence of PACS in India varies from 1.4% (APEDS) to 10.3% (the Vellore eye survey). Study by Thomas at all about 22% of PACS cases will progress PAC to show some evidence of angle closure within 5 years but none developed functional damage. Bilateral PACS was a clinical risk factor for progression. PACS patients may be followed for development of IOP elevation, evidence of progressive narrowing, or synechial angle closure. Treatment of PACS is by prophylactic LPI but not for all PACS it's not needed and it will account for nearly 10.2% of Indian population aging over 35 of years. The decision to proceed with iridotomy requires a variety of factors to be considered: the patient's access to care, whether the lens may soon require cataract surgery, the status of the fellow eye, the patient's age and ethnicity etc.LPI can be done foe for selected cases like established PAC or PACG in fellow eye, family history of primary angle closure, patients cannot come for regular follow up, cases found positive on provocative test or require frequent pupillary dilatation like diabetics, or patients with topical or systemic medications that pose a significant risk of precipitating angle

closure glaucoma and a patient with narrow angles with symptoms of sub acute angle closures or signs of previous episode of angle closure.

(B) NORMOTENSIVE GLAUCOMA (NTG)

NTG is define as characteristic glaucomatous optic disc cupping and associated visual field defects with normal or low intraocular pressure, with absence of ocular or systemic features contributing to other forms of optic neuropathy. NTG has no fundamental difference from ordinary chronic primary open angle glaucoma (POAG), except that the etiologic trigger or pathogenic process is accelerated at a lower level of IOP. NTG will have certain peculiar findings like

A region of absent retinal pigment epithelium (RPE) -as a crescent or halo at the disc border associated with cupping which is seen more in the region of absent RPE with field loss in corresponding region.

Features of cupping: it can be in the form of notch (due to localized region of thin or absent neuro-retinal rim) a "focal ishcemic" type of cupping with localized dense arcuate field defect or even a dense upper hemifield defect. Or it can be diffuse shallow cupping with pale disc and surrounding tissue, leading to a "senile sclerotic" disc. Or it can be a mixture of both. There may be some relationship of the focal ischemic type to vascular dysregulation and of the senile sclerotic type to systemic atherosclerosis. High myopes may be particularly susceptible to NTG, but their discs are difficult to evaluate. With a frequent temporal crescent, scotomas tend to be closer to fixation than the paracentral scotomas of non-myopes.

Splinter hemorrhages are reported more frequently in NTG, but may also be seen in uncontrolled POAG. Hemorrhages may simply indicate poor control therefore further lowering of pressure to be achieved. **POAG and NTG intermingle in the same families**, suggesting they are the same or related conditions. **Vascular dysregulation** include cold hands and feet as an over-reaction to cold or stress. Patients may report sleeping with socks on even in warm climates, and noticeably cold hands when greeted with a handshake. Arterial blood pressure tends to be low. **Migraine headaches**, especially with a visual aura, are more common and are more frequent in women. They do not have a sensation of thirst even when dehydrated. **Shock-induced neuropathy**. **Acute ischemic episode** or chronic obstructive arterial disease and sleep apnea can be considered either a **special variety of NTG** or one type of **"pseudo-NTG"**. Some patients with severe cardiovascular event in the past can have excavated optic nerves which are recognized later on routine eye examination. There can be chronic atherosclerosis, obstructive arterial disease rather than dysregulation. These cases may tend to be non-progressive, if the underlying vascular etiologic cause has been corrected, just as ordinary glaucoma is expected to become stable after the inciting elevation of IOP is lowered. Interestingly in the magnetic resonance imaging (MRI) evidence of microinfarcts was found in 22% of cases with NTG, the approximate percentage of cases that progressed despite treatment in the collaborative normal-tension glaucoma study. Hypothetically, there might be a form of NTG that represents a primary optic neuropathy unaffected by IOP and lowering the IOP would be irrelevant in such cases. Though rare, but some cases of NTG continue to be progressive even after the IOP has been lowered to the range of 8 to 10 mm Hg at all times.

What is Pseudo-glaucoma? Or glaucoma mimicking cases which are misdiagnosed -

A. Large physiologic cup with no field loss. These cases are labeled as NTG due to normal IOP with large disc with a large physiologic cup.

B. congenital anomaly of the disc- There may be a notch in the rim of the disc with a colobomatous absence of RPE at the bottom edge of the disc, absent nerve fibers in a wedge shape with corresponding field defect. The abnormality can be bilateral. Unilateral cases might have amblyopia since childhood, or even secondary strabismus.

C. Anterior ischemic optic neuropathy. The non-arteritic form typically occurs in eyes with small discs, but if the arteritic form occurs in an eye with a large physiologic cup, the loss of nerve fibers can create a localized thinning or notch in the rim, associated with a dense field defect in either the upper or lower hemifield. If the disease is unilateral, an erythrocyte sedimentation rate and evaluation for giant cell arteritis is needed so as to protect the other eye or prevent recurrence in the initially involved eye.

D. Branch retinal vessel occlusion. Some time the acute event went unnoticed by the patient, and subsequently present with loss of retinal nerve fibers in arcuate region with corresponding arcuate visual field defect. If the disc had a physiologic cup of moderate or large size, the neuro-retinal rim may be locally thinned in the region with lost nerve fibers can mimic NTG.

E. Optic nerve"giant" drusen. With careful inspection, a fleeting glisten can be seen deep in the disc tissue, due to disc drusen and it is associated with corresponding visual field defect gives confusion of glaucoma.

F. Orbital or intracranial tumor- unilateral tumor such as an orbital meningioma producing a prechiasmal type of visual field defect if it is associated with large optic nerves large cups can mimic glaucoma but careful examination and unilateral appearance will rule out glaucoma.

It is important to see if we are missing "High-Pressure" glaucoma due to-

1. **Inaccurate tonometry.** Applanation tonometers may give a false low reading if the cornea is very thin. Indentation tonometers were known long ago to give false low pressure in large, often myopic, eyes. Therefore accurate measurements and many readings are required.
2. **Variable intraocular IOP.** The IOP may be abnormally high at certain times of the day, or certain days of the week. Therefore diurnal variation is needed before to make diagnosis of NTG.
3. **Past elevation of IOP.** Possible causes include chronic use of corticosteroids for contact lens comfort or allergy, or uveitis that is no longer active. Chronically intermittent angle closure may also have caused considerable elevation of IOP. Pigmentary glaucoma produces elevated pressure during middle-age years, during which time damage to the optic nerve occurs, but the IOP may return to normal before glaucomatous cupping is found during an eye examination for new glasses or early cataract. Episodes of glaucomatocyclitic crisis may have produced high IOP in the past. Finally some cases of "burned out" glaucoma in which changes in aqueous humor dynamics has caused a previously high IOP to return to normal.
4. **Chronic POAG** in which the pressure has been lowered by systemic medication. Beta blocker used for hypertension systemically can bring the IOP into the normal range.

DIAGNOSIS OF NTG

Diagnosis of NTG is by exclusion. Diagnostic evaluation of NTG should include history to rule out nonglaucomatous causes of optic neuropathy, such as ocular trauma or central nervous system (CNS) pathology, cold extremities, migraine headaches, systemic hypotension, or vascular dysregulation. A comprehensive eye examination including gonioscopy needed to rule out potential secondary causes of glaucoma. Differentiation between high tension and normal tension glaucoma can be difficult based on a single IOP measurement; a diurnal curve can be helpful to detect episodes of elevated IOP outside of routine clinic visits. CNS imaging or neuro-ophthalmic consultation is usually unnecessary in the presence of classic findings, such as APON or optic disc hemorrhages with corresponding visual field defects. Further work-up should be considered in the presence of asymmetric disease or visual field defects suggestive of a compressive CNS lesion or other nonglaucomatous processes. A systemic evaluation for potentially contributing conditions, such as OSA or Raynaud's phenomenon, sleep studies or 24 hours ambulatory blood pressure monitoring is often valuable in cases of disease progression refractory to IOP-lowering therapy.

Differential Diagnosis of Normal Tension Glaucoma

- **Undetected POAG (diurnal IOP fluctuation)**
- **Intermittent IOP elevation (e.g., intermittent angle closure)**
- **Glaucomatocyclitic crisis/uveitic glaucoma**
- **"Burned-out" pigmentary glaucoma**
- **Corticosteroid-induced glaucoma**
- **Nonglaucomatous optic nerve**
  - **Congenital disc anomalies/cupping**
  - **Myopia with peripapillary atrophy**
  - **Optic nerve coloboma/pit**
- **Vascular etiology**
  - **Temporal arteritis (AION)**
  - **Carotid & ophthalmic artery occlusion**
  - **Central retinal artery occlusion**
- **Neurological etiology**
  - **Meningioma**
  - **Pituitary adenoma**
  - **Empty sella syndrome**
  - **Leber's optic atrophy**
  - **Syphilis**
- **Tonometric error/thin cornea**

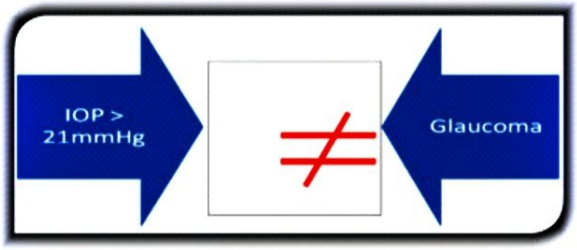
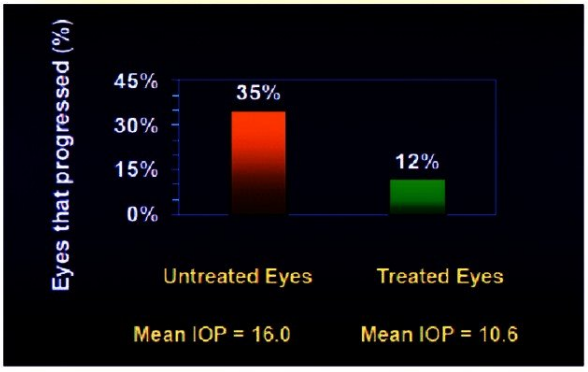


Figure 1: IOP above 21 mmHg is not always glaucoma. Glaucoma is considered as an optic neuropathy where eye pressure has an important role.



TREATMENT

The treatment for NTG is matter of debate the aim of treatment is to control IOP; as this is widely accepted risk factor in the progression of NTG. **The Collaborative Normal Tension Glaucoma Study** reported that ag-gressive IOP reduction of at least 30 percent from baseline levels re¬duced the ex¬tent of progressive visual field loss. The study results showed that aggressive IOP re¬duction with medications and/or sur¬gery de¬creased the risk of progression from 35 percent to 12 percent over a five-year period. However, a substantial percentage of the untreated patients (65 percent) did not progress, thereby demonstrating the extremely variable clinical course observed in NTG. A faster rate of progression occurs in women (risk ratio=1.85), in patients with migraine headaches (risk ratio=2.58), and in the presence of disc hemorrhages (risk ra¬tio=2.72). Before initiating treatment of NTG, the clinician must first determine whether the affected patient is at risk for progression (i.e., progressive vs. non-progressive form). If progressive, then determine a target pressure level and IOP measurements should be taken at different times of the day to rule out diurnal fluctuation. The target pressure may need to be reset (and therapy changed) if fluctuation in diurnal IOP and/or progression of optic nerves/visual fields are ob¬served. It may be helpful to initiate a unilateral trial of the medication so that the contralateral eye can be used as a control to assess the drug's therapeutic and side-effect profile. The current gold standard for medical glaucoma therapy is the prostaglandin analogues. Topical beta blockers have the potential for significant systemic side effects, such as nocturnal hypotension, that may be of particular concern in NTG, therefore alpha 2 agonist or carbonic anhydrase inhibitors can be considered 2nd line rather than beta blockers. If medical therapy is in¬sufficient and/or intolerable in reach¬ing the target IOP level, laser trabeculoplasty may be considered. In certain instances, glaucoma filtering surgery is indicated to obtain IOP control. Use of antifibrotic agents such as mito¬mycin-C or 5-fluorouracil may im¬prove the overall success rate of filtration surgery.

Future Treatment- Despite apparent adequate IOP lowering, some patients still have progression of their disease. Number of other risk factors besides IOP has been implicated in the pathogenesis of glaucoma including vascular, mechanical and genetic aspects, as well as myopia, endocrine abnormalities and autoimmune phenomena. Di¬urnal variations in the microcirculation of the optic nerve head may play a role in the progression of NTG in patients with vascular dysregulation, leading to both low perfusion pressure and impaired autoregulation and ischemia. Therefore vasodilator drugs such as calcium-channel blockers like brovincamine- a re¬latively selective cerebral vasodilator used to improve cerebral circulation and metabolism, Nimodipine, another selective calcium-channel blocker, has been reported to improve performance in visual field and color vision testing in NTG patients. Memantine, a po¬tential neuroprotective agent that blocks excessive NMDA-type glutamate receptor activity without disrupting normal activity, is under trial. Thus, further research is

required to improve our un¬derstanding of the pathogenesis, diagnosis, and treatment of this challenging disease.

(C) OCULAR HYPERTENSION

Ocular Hypertension is defined as an IOP > 21 mmHg without any evidence of glaucomatous optic nerve damage or visual field defects. There is no underlying ocular or systemic cause of elevated intraocular pressure. The prevalence of ocular hypertension varies in different ethnic groups; highest prevalence of 12.6% was reported amongst Afro-Caribbean population in one study. In southern India prevalence of 1.1% in individuals above 40 years of age has been reported. Its prevalence increases with age, in the Framingham Eye Study conducted in Whites, its prevalence was 6.2% below 65 years age group, while 8.7% in individuals above 75 years of age. Individuals with ocular hypertension are 8 times more susceptible for the development of primary open angle glaucoma (POAG) as compared to normal subjects. Therefore, early diagnosis and initiation of anti glaucoma medication in high risk group may reduce the incidence of POAG and subsequent visual disability. Population and clinical-based studies report that 0.24 to 2.2% of OHT progresses to POAG per year.

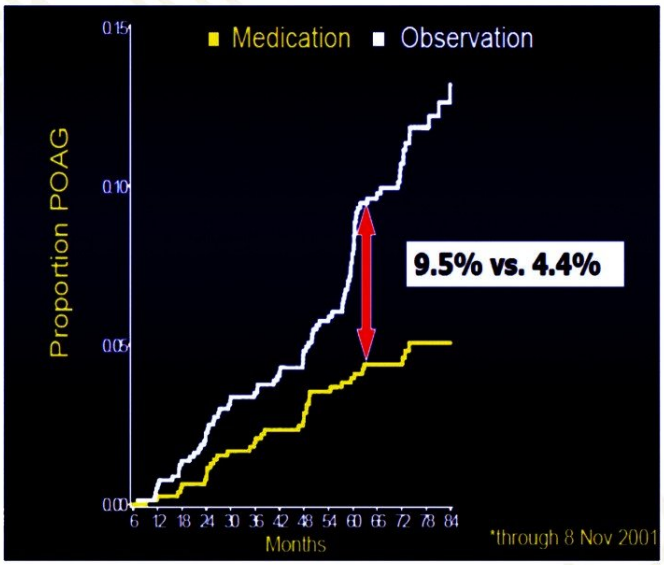
Risk Factors for Subsequent Conversion to POAG- According to Ocular Hypertension Treatment Study showed on 5 years follow up; the incidence of POAG was 9.5% in non treated group as compared to 4.4% in the treatment group (nearly 50% reduction).

The risk factors includes—

1. Central Corneal Thickness (CCT) – CCT was found to be a powerful predictor for the development of POAG and independent risk factors for glaucoma.CCT less than 555µ were found to be at greater risk than eyes with CCT more than 588µ.The relative risk of POAG increased by 81% for every 40µ decrease in CCT.

2. IOP - Normal IOP range of 10-21 mmHg. Although, IOP readings may show considerable variations among glaucoma patients, IOP reading more than 22 mmHg is a positive predictive factor for the development of POAG. Diurnal variations are known to be an independent risk factor for progression of glaucoma.

3. Age – Age is an independent risk factor for the development of POAG. Individuals with older age had a greater risk for conversion to glaucoma. OHTS found an increased risk of POAG with age (per decade), of 43% in the univariate analysis and 22% in the multivariate analysis.



4. Pattern Standard Deviation (PSD) - Although the patients with ocular hypertension may not have visual field defects on Standard Automated Perimetery (SAP), OHTS found that greater PSD on SAP correlated with increased risk of progression to POAG. With 0.2dB increase in PSD, 22% increase in relative risk in OHTS.

5. Optic Nerve – Although OHT patients have no apparent glaucomatous disc changes, increased vertical and horizontal cup-disc ratio is a risk factor for progression to POAG. Increase in cup-disc ratio by 0.1 leads to 32% and 27% increase in relative risk in vertical and horizontal cupping, respectively.

6. Family history and Black race were not found to be significant in multivariate analysis in OHTS. However, other studies have shown them to play significant role in the development of POAG. Bilateral OHT appears to be a risk factor for progression.

DIAGNOSIS

Ocular hypertension is a diagnosis of exclusion. Thorough ocular examination including tonometery, gonioscopy, optic disc evaluation and visual field testing should be done to rule out any underlying cause of IOP elevation. History of ocular trauma and steroid use should be ruled out. Frequency doubling Perimetery (FDP) or short wavelength automated Perimetery (SWAP) detects glaucomatous damage at a very early stage, 4 years before the changes appear in white-on-white Perimetery.

TREATMENT

Considering the low rate of progression to POAG (only 1-2% patients progressed to POAG in a yearly follow-up in OHTS trial), cost of ocular antiglaucoma medications, long term compliance issues and side effect of drugs, not every case of ocular hypertension needs treatment. Therefore, treatment is recommended only in high risk group by 20% IOP reduction either by topical beta blockers or prostaglandin analogues. Risk factors includes

Low risk: Follow-up every 2 years. (1). IOP 22-23 mmHg with central corneal thickness more than 588 microns.

(2). Vertical cup-disc ratio 0.4 or more with central corneal thickness more than 588 microns.

Moderate risk: needs annual follow-up and treatment initiated at the earliest documented glaucomatous damage. – (1) IOP 24-29 mmHg without retinal nerve fiber layer damage. (2). IOP 22-25 mmHg with central corneal thickness <555 microns. (3). Vertical cup-disc ratio 0.4:1 or more with central corneal thickness between 555-588 microns.(4). Family history of POAG in first degree relative (5). High Myopia.

High risk: Requires treatment by 20% IOP reduction.(1) Retinal nerve fiber layer defects. (2). Parapapillary changes. (3). IOP > 30 mmHg (4). IOP > 26 mmHg with central corneal thickness <555 microns. (5). Vertical cup-disc ratio 0.4:1 or more with central corneal thickness <555 microns.

Early recognition and treatment of high risk patients can limit the visual disability due to POAG. Patients under monitoring, FDP or SWAP may be beneficial in early initiation of treatment.

APPPLANATION TONOMETRY IN GLAUCOMA

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INTRODUCTION: It has always been a challenge to measure the intraocular pressure(IOP) accurately. As IOP measurement is not an independent entity it is affected by many factors such as corneal thickness, corneal biomechanics, scleral rigidity, refractive error and the time of the day. Corneal biomechanical properties such as viscosity, elasticity, hydration and curvature of cornea are found to have significant impact on IOP measurement.
The measurement of IOP must be considered, as important as vision in ophthalmology outpatient department for each and every patient as the IOP is the only modifiable risk factor in glaucoma so accurate assessment and monitoring of IOP is very essential.
The Goldmann Applanation tonometer (GAT) is considered as the standard method of measuring IOP although it does not take into account the corneal biomechanical properties, 1,2,3 the influence of Central corneal thickness has been explored.

GENERAL PRINCIPLES: GAT measures the force applied per unit area. It is based on " Imbert-Fick' law". P=F/A Where P is the pressure inside the sphere that is intraocular pressure, F is force required to flatten the cornea and A is the area of cornea which is being applanated 4. GAT is a variable force tonometer consisting of a double prism with a diameter of 3.06mm. It is till now considered as most accurate tonometer used in ophthalmic practice and most widely used too.

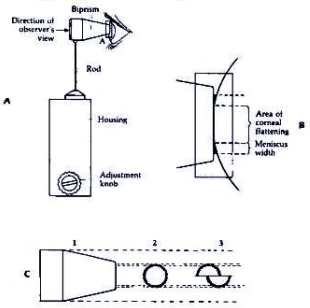
Goldmann Applanation prism

Applanation tonometer

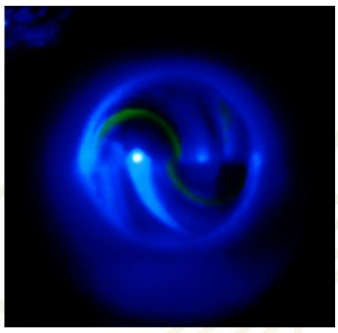
PREREQUISITES:
Tonometer, Applanation prism, Local anaesthetic drops, Fluorescein strips, gauze swabs.

METHOD:

- The prism is disinfected with isopropyl alcohol 70% (methylated spirit) or sodium hypochlorite 1%. The prism must be rinsed thoroughly in sterile water and wiped dry with a clean swab (residue of the disinfectant may cause a caustic burn on the cornea, uveitis and damage to the tip of prism if in contact with disinfectant for the longer duration).
- The graduation marked '0' on the measuring prism is aligned with the white marker point on the tonometer head.
- The calibrated dial of the tonometer is set at 10 mmHg
- The fluorescein stain (preferably strip) is used to stain the cornea of patient after anaesthetising the ocular surface..
- The patient sits comfortably at the slit lamp: at the right height, with their chin on the rest and their forehead against the headband.
- The magnification of the slit lamp is set at ×10 with maximum illumination with cobalt blue light to look clearly the stained mires.
- The tip of the Applanation tonometer is used to make the mires on the cornea by gently touching the cornea. The dial is used to align the mires that the inner edges of the two semicircles just touch, at that moment the dial reading is the IOP of patient.



APPLANATION TONOMETER



The point of recording of IOP

POTENTIAL ERRORS

1. Inappropriate fluorescein pattern – (a)Thick mires lead to overestimation of IOP and it is usually due to over-staining, excessive pressure of prism over cornea or using the wet prism. (b) Thin mires lead to underestimation of IOP. Thin mires are usually due to inadequate staining.
2. External pressure on the globe will lead to artificially high readings usually occurs while taking the lids of patient and pressing the globe simultaneously and patient squeezing the eyes.
3. Incorrect calibration-it is important to check the calibration at regular intervals.
4. Corneal pathology- e.g. Gross edema and distortion of cornea. Thin corneas produces underestimate and thick cornea d/t increased collagen gives overestimate, if cornea is thick d/t edema gives underestimate of IOP.

5. Position of globe- An upward gaze leads to high readings of IOP.
6. Excessive fluid intake or alcohol consumption- Lead to high readings.
7. >3D astigmatism- produces elliptical rather than circular area of contact with the cornea, with semicircles displaced horizontally. IOP underestimated by 1 mmHg for every 4D of WTR astigmatism, vice versa for ATR astigmatism.

RELATION WITH CCT:

Central corneal thickness values which are less than 555 micron will cause an underestimation of IOP by GAT. Thickness significantly greater than normal will cause an overestimation of IOP. Around 20 micron change in CCT will affect the IOP by 1 mmHg 5, that is why pachymetry plays an important role in IOP measurement by GAT 6.

CALIBERATION OF APPLANATION TONOMETER:

Accurate measurement of IOP depends on the accurate calibration of tonometer. At least once in a six month calibration is recommended. A calibration rod and a weight holder available along with the tonometer, weight holder fixes in the space given in the tonometer. Rod is having five circular marks on it. Central representing 0 others 2 and 6 away from the central one (as shown in the picture).

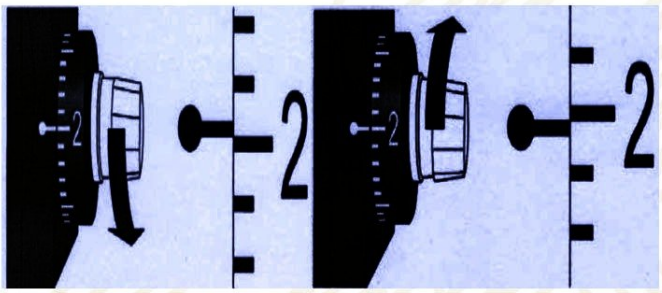
HOW TO CALIBRATE THE TONOMETER

The position of person who is calibrating is on the examiner side on the slit lamp the rod is fixed in the weight holder. All the three circular marks 0, 2 and 6 are checked one by one. Similar procedure is done for three marks, so it is done in three steps.

First step- The calibration rod is fixed on the weight holder with its centre, then the dial of the tonometer is slowly rotated in counter-clock wise direction until the head rock towards us. The tension at this moment should be 0-2 mmHg. Same way dial is put back to zero position and rotated in clock wise direction. The tension at this moment should be 0-2 mmHg..

Second step- The calibration rod is set on the position 2 is set precisely on the index mark of the weight holder. The rod is fitted in the weight holder provided that the longer portion of rod is towards examiner side. The dial of tonometer is rotated in counter clock wise direction slowly till the head of tonometer moves toward examiner at this moment the reading on the dial should be 17-20mmHg . Now the dial is rotated in clock wise direction slowly then the moment of head observed at this moment the reading on the dial should be20-23mmHg.

Third step- similar to above the control weight is at 6 and position of circle in rod is 6. Readings are there in the following table.



Checking at dial position and rod position "2"

Checking at dial position and rod position "2"
Checking at dial position and rod position "6"

Table ; Normal values of calibration

S.n.	Calibration Rod Position	Direction of Movement of Dial	Reading of In Tension mmhg
1.	0	Counter clock wise	0-2
2.	0	clock wise	0-2
3.	2	Counter clock wise	17-20
4.	2	clock wise	20-23
5.	6	Counter clock wise	56-60
6.	6	clock wise	60-64

ERRORS IN TONOMETER

If the error of 2 mmHg is found while calibrations then the IOP value ranges between ±2 mmHg. For example if IOP of a patient is 14 mmHg and there is an error of 2 mmHg, the IOP of pt will range from 12 to 16 mmHg, that is a wide range which may entirely change the management of a particular patient.

In case of errors tonometer is to be sent for its recalibrations to its manufacturer.

COMPLICATIONS OF PROCEDURE:

The most common complication is corneal abrasions .It is avoidable if the tip of tonometer is allowed to touch the surface of cornea gently and as the cornea is anaesthetized patient should be explained not to rub the eye post manoeuvre.

Discussion

Few tonometery methods are coming into practice and they are comparable with GAT they are Dynamic contour tonometry (DCT) and ocular response analyzer (ORA) and Corvis ST.

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ROLE OF CENTRAL CORNEAL THICKNESS (CCT) IN GLAUCOMA

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Introduction:

- Cornea Thickness at the centre (2-3 mm) can be taken as a value for reference.

Methods of Measurement:

- Ultrasonic Pachymeter (Gold standard), OCT, Orbscan, Pentacam and UBM.

Understanding GAT

Goldmann applanation tonometry, the current gold standard for the measurement of IOP, is based on the Imbert-Fick law. Goldmann observed that when the specific area applanated, the surface tension due to the tear film counterbalanced the resistance to indentation of the cornea, thus making it unnecessary to consider the rigidity of the globe and the surface tension of the tear film in applanation tonometry. Variations in corneal thickness change the resistance of the cornea to indentation so that this is no longer balanced exactly by the tear film surface tension.

A thinner cornea may require less force to applanate it, leading to underestimation of the true IOP.While a thicker cornea would need more force thus giving an artifactually high IOP reading. Goldmann himself discussed the influence of variations of central corneal thickness (CCT) on IOP measured by applanation. However, he felt that significant variations in CCT occurred only rarely and hence assumed a "normal" CCT of 520 µm for his instrument. The underestimation of IOP was as much as 4.9mmHg in thin corneas, while thick corneas produced an overestimation of about 6.8mmHg. Accordingly it has been suggested that measurement of corneal thickness is necessary for the accurate interpretation of applanation tonometry. It has been calculated that applanation tonometry over/ under-estimated IOP by 5mmHg for every 70 µm corneal thickness.

A correction factor for IOP, to adjust for CCT measurements that differ from "normal CCT" was proposed as follows:

Practical Guide

CCT	CORRECTION FACTOR
470	+3.5
480	+2.8
490	+2.1
500	+1.4
510	+0.7
520	0
530	-0.7
540	-1.4
550	-2.1
560	-2.8
570	-3.5
580	-4.2
590	-4.9
600	-5.6

CASE EXAMPLE:

32 year male with DM presents for routine eye check up. IOP by GAT is 26 both. Disc shows : 0.4 CDR. NRR is normal. Visual Fields are normal. Now, he will be labelled as **Ocular hypertension**. One may consider starting anti-glaucoma medication since IOP is more than "normal".

Now CCT is measured - 600 microns.

Correction Factor: - **5.6** , Corrected IOP is **26 - 5.6 = 20.4**

No treatment is required since corrected IOP is now normal

GONIOSCOPY

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INTRODUCTION:

Gonioscopy forms part of a complete ophthalmic examination and is mandatory for diagnosis and managment of glaucoma.The purpose of gonioscopy is to view the drainage system of eyes and determine the cause for elevated intraocular pressure. Gonioscopy permits to identification of eye at risk for angle closure and open also detect angle abnormalities that could have diagnostic and therapeutic implication.

PRINCIPLE

In healthy eyes, the angle cannot be visualized directly because of the optical principle known as the critical angle

(Approximately 46 degree for cornea-air interface). Normally the light coming from the anterior chamber angle is totally internally reflected making the viewing of the angle impossible without the aid of a gonioscopic lens.

The gonioscopic lens changes the interface from cornea-air to lens-air, changing the critical angle and thus permitting viewing of the anterior chamber angle.

TYPES OF GONIOSCOPY

DIRECT GONIOSCOPY

It is performed with a steep convex lens which permits light form the angle to exits eye closer to the perpendicular at the lens air interface. The lenses included are-The koppe,Hoskin- Barkans and Swan-Jacob lenses.

INDIRECT GONIOSCOPY

Indirect gonioscopy uses mirrors or prism to overcome the problem of total reflection.

Types of indirect gonioscopic lenses are thoses needed coupling agent such as Goldmann 3 mirror ,2 mirror ,1 mirror lenses.

The lenses that do not need coupling agents or lenses for indentation gonioscopy include the zies 4 mirror, the Posner lens and the Sussmann lens.

INDICATIONS

Gonioscopy is an invaluable tool in diagnosing and planning management for glaucoma cases.One of the most common indications for performing gonioscopic examination is to indentify angles at risk of closure and distinguish between primary angle closure disease and primary open angle glaucoma. Other critical uses include diagnosis of neovascularization of the angle in eyes with retinal ischemia.

Other diagnostic indications include the following:

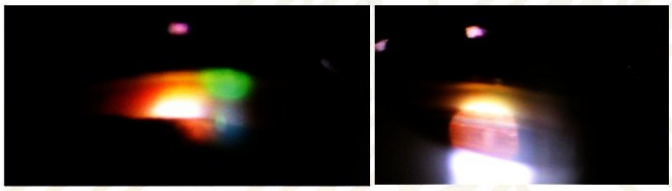


fig:1 Closed angle **fig:2 open angle**

- Classification of glaucoma –open angle or closed angle.
- To assess the anterior chamber angle recess and risk of angle closure.
- To indentify plateau iris.
- To note the presence and extent of neovascularisation of angle.
- Assessment of abnormal angle pigmentation.
- Visualization of pseudoexfoliative material in the angle.
- To look for post traumatic angle recession,cyclodialysis.
- Rule out foreign body in the angle after open globe injury.
- Neoplastic invasion into angle structures.
- Diagnosis of blood in the Schlemm's canal.
- To view copper deposition on Descemet's membrane.
- Evaluation of trabeculectomy fistula.
- Visualisation of glaucoma drainage devices.
- To diagnose anterior insertion of iris in developmental glaucoma.
- Visualization of congenital anomalies.

Therapeutic

- Laser trabeculoplasty / goniophotocoagulation.
- Reopening of a blocked trabeculectomy opening.
- Nd-YAG laser after deep sclerectomy.
- Laser of suture tied around tube of a glaucoma drainage device.
- Indentation gonioscopy to break an acute attack of angle closure.

When to perform gonioscopy?

Gonioscopy should be performed as a part of routine evaluation for all patients visiting an ophthalmologist and is mandatory for

all glaucoma patients at diagnosis and during follow up(at least once a year).

How to perform gonioscopy?

Gonioscopy is best performed in a dark room with minimal slit lamp illumination and beam height aimed at the angle, taking care that the slit beam never crosses the pupil and the patient maintains gaze in the primary position. This avoids pupillary constriction which can lead to artificial opening up of the angle in eyes with angle closure.

What to look for during gonioscopy?

While doing gonioscopy the observation should comment on the visibility of the angle structures and report the posterior most structure visible.The following key features should be reported:

1. Sclera spur visible or not
2. The width of the angle recess
3. Level of insertion of the iris
4. Degree of trabecular meshwork pigmentation
5. Shape of iris
6. Effect of manipulation on a narrow angle
7. Presence and extent of peripheral anterior synechiae
8. Symmetry of gonioscopy finding between the two eyes
9. Other pathologies like neovascularisation of the angle, angle recession, silicone oil, foreign bodies , blood reflux in schlemm's canal etc.

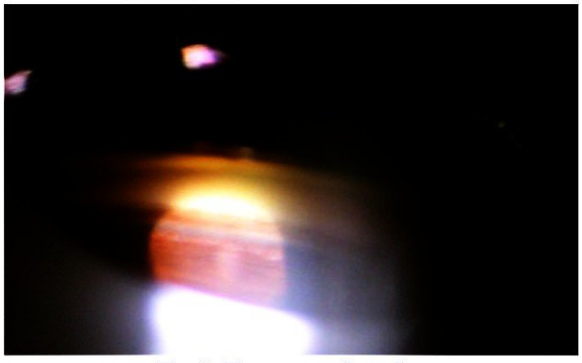


Fig:3 The corneal wedge

Identification of the corneal wedge is the key step in defining the angle structure. By using a thin slip of light inclined 15-20° from the angle of the oculars and sharp focus, projected onto the iridocorneal angle, 2 light reflections are note, one from the external surface of the cornea and the other from the internal surface of the cornea. These two reflections meet at the end of Descement's membrane which is the beginning of Schwalbe's line.

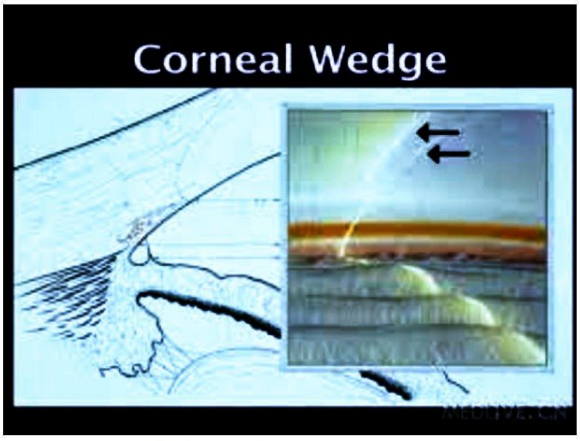


Fig:4 Grading of angles

Van Herick method: grading: illumination beam at 60° angle from viewing axis.

Grade of angle	Depth of prep.Ant. chamber
4	→PAC>1 CT
3	→PAC>1/4-1/2 CT
2	→PAC=1/4 CT
1	→PAC<1/4 CT

Modified gonioscopy grading

N No dripping of the beam

D Dipping of the beam

SL Schwable's line and anterior 1/3 of the trabrcular meshwork

TM Middle 1/3 of trabrcular meshwork visualization

SC Posterior 1/3 of trabrcular meshwork

SS Sclera spur visualization

CB Ciliary body band visualization

Based on the angular width of angle recess

Grade	Angle width	Description	Risk of closure
4	45-35°	Ciliary body band	Impossible
3	35-20°	Scleral spur	Impossible
2	20°	Trabecular meshwork	Possible
1	<10°	Schwalbe's line	Possible
Slit	Slit	No iridocorneal contact	Possible
0	0°	No corneal wedge	Closed

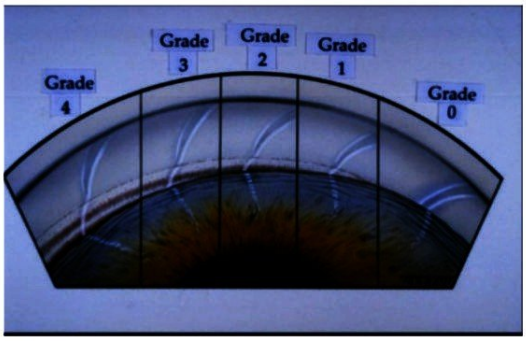


fig:5

Manipulative Gonioscopy

Manipulative is of value in studying angle anatomy in narrow iridocorneal angles. This can be achieved in Goldmann type lenses by simply asking the patient to look in the direction of the mirror or moving the mirror towards the angle being viewed.

The examiner should report the normal angle view in primary gaze and then document the opening of the angle on manipulation by asking the patient to look into the mirror of the gonioscope, opposite to the angle being examined.

INDENTATION GONIOSCOPY

This type of gonioscopy requires the use of special type of gonioscope known as corneal type goniolenses, 4 mirrors. With the corneal type goniolenses, they have a small diameter, the central cornea may be indented to force the aqueous out and artificially widen the angel. When the iridocorneal angle is optically narrow, indentation gonioscopy also facilities the identification of angle structure. Should the angle be closed, indentation helps differentiate appositional from synechial angle closure. This is important as synechial closure is irreversible, while appositional closure can be reversed.

Closed angle with no structure visible **Fig:6** **with indentation all the structure are visible**

ABNORMALITIES IN ANGLE

Narrow angle: if the angle is sufficiently narrow and iris base sufficiently distensible, the iris is forced against the surface of the trabecular meshwork, blocking aqueous flow into Schlemm's canal.

· Peripheral anterior synechiea: they should be differentiated from prominent iris process:

Iris processes	Peripheral anterior synechiae
Fine process	Broad
Extend into sclera spur	Extend beyond sclera spur
Follow concavity of recess	Bridge concavity of recess
Do not obscure the angel structures	Obscure structure
Iris moves with indentation	Resist movement
Broken with angle recession	Intact

Fig:7 Iris processes **fig:8**

Peripheral anterior synechiea

Sampaolesi's line: It is the line of irregular pigmentation deposition anterior to schwalbe's line. This pigmentation in Sampaolesi's line has a dark granular and discontinuous appearance Sampaolesi's line can be mistaken for trabecular meshwork in narrow angle.

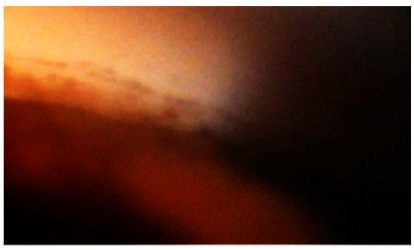


fig:9 Sampaolesi's line

Angle recession: an angle recession the ciliary body may be broadly exposed. 4-9% of patients with angle recession more than 180° eventually after many years may develop glaucoma. So it is important to follow up patient with angle recession indefinitely.

Neovascularization of the angle:

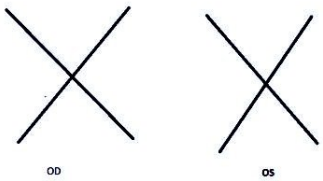
Normal vessel	Neovascularization of the angle
Radial orientation	Fine and irregular orientation across the angle
Thick and dull red	Bright red
Non branching	Arborising
Do not cross the scleral spur	Cross the scleral spur

Iris Neovascularization, in most cases, is first seen on the peri-pupillary iris although it may be seen first seen n angle in a few cases. Iris Neovascularization is most commonly seen in retinal hypoxic disease

- **Fuchs heterochromic iridocyclitis**
- **Plateau iris:** Iris exhibits a flat approach to the angle. AC is swallowed/there is angle closure attack inspite of patient PI. Peripheral iris is anteriorly displaced, so that when the pupil is dilated it bunches up and close the angle despite a patient PI.
- **The angle in pseudo exfoliation:** There is clumped brown pigment over the pigmented trabecular meshwork. Ther is also a line of pigment along schwalbe's line and another, wavy line of pigment anterior to this line.
- **Cyclodialysis:** It is the dis-insertion of ciliary body from Scleral spur. It reveals the following features-
  1. Deep angle
  2. Decreased IOP
  3. White coloured band seen on gonioscopy.
- **Miscellaneous conditions:** tumors of the anterior segment, ciliary body cysts, intraocular foreign body and early detection of a Kayser- Fleisher Ring is possible with gonioscopy.

Diagramming gonioscopy:

Diagrammatic representation of angle findings, mentioning the date of examination is very essential in practice because the condition of the angle is not static throughout life.



dia:1

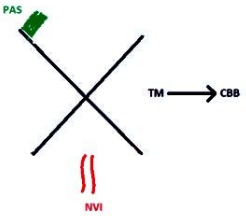
CLEANING OF CONTACT LENSES

- Adenovirus type 8 can be removed by soaking the lens for 5-10 min in dilute sodium hypochlorite(1:10 household bleach),3% hydrogen peroxide or 70% isopropyl alcohol or wiping with alcohol or hydrogen peroxide.
- HSV1 can be eliminated by swabbing with 70% isopropyl alcohol.
- 10 min of continuous rinsing under tap water was reported to remove all HBV surface anti agent.
- Wiping with 3% hydrogen peroxide or 70% isopropyl alcohol swabs can completely disinfect it from HIV1.

HOWTO DEPICT GONIOSCOPIC FINDINGS

There are different classification system used to grade the angle like Shaffer, Spaeth, and Scheie etc but are not practical for routine use. Simply the gonioscopic finding should mention the posterior most structure visible on gonioscopy in the primary position in the superior and inferior angle. Making a diagram of the gonioscopic finding can help us to compare our finding on serial gonioscopic examination and is also easy to interpret for other clinicians.

If manipulation/indentation is done, an arrow is put and the structure thus exposed is mentioned. Presence of peripheral anterior synechiae or other pathologies like new vessels can be drawn in the corresponding clock hour as visible on gonioscopy.



Dia:2

LIMITATIONS OF GONIOSCOPY

- Gonioscopy Is a contact investigation which causes discomfort to the patient.
- It can transmit a conjunctival infection to the patient.
- Gonioscopy should not be performed in suspected open globe injury or early in the course of closed globe injury with hyphaema as pressure can precipitate re-bleed.
- Gonioscopy is difficult in cases of acute angle closure with corneal oedema and eyes with corneal opacification.
- Excessive pressure while using Goldmann type of lens may artefactually close the angle and while using corneal type of gonioscopes it may give a open appearance in narrow recess angle configuration.
- Use of slit lamp illumination while doing gonioscopy leads to pupillary constriction and opens up/changes the angle configuration.
- Gonioscopy cannot objectively quantitate the angle parameters and there is a wide inter-observation variability .
- Gonioscopy is not useful to identify pathologies behind the iris.
- Indentation gonioscopy can lead to formation of corneal folds, distorting the view of angle structure and may cause corneal epithelial injury.
- Mastering gonioscopy has a long learning curve requiring regular practice on a large number of patients.

Gonioscopy remains 'Gold standard for evaluation of the anterior chamber and should be performed as a basic test like ophthalmoscopy or tonometery

HOW TO ANALYZE VISUAL FIELD CHART

Dr. Vasudha Damle (Assistant Professor, R.K.D.F., Medical College Bhopal)
Dr. Girish Lale (Consultant, Sankalp Eye Hospital Bhopal)

Introduction: Single Field analysis printout is divided into 10 zones which can be divided into 2 groups:

ZONES INDEPENDENT OF NORMATIVE DATA AND STATPAC ANALYSIS

ZONE 1 Patient data / test data

ZONE 2 Reliability indices/foveal threshold

ZONE 3 Raw Data

ZONE 4 Grey Scale

ZONES DEPENDENT ON NORMATIVE DATA AND STATPAC ANALYSIS

ZONE 5 Total deviation numerical plot

ZONE 6 Total deviation probability plot

ZONE 7 Pattern Deviation numerical plot

ZONE 8 Total deviation probability plot

ZONE 9 Global Indices – Mean deviation(MD)
Pattern standard deviation(PSD)
corrected pattern standard deviation(CPSD) &
Short term fluctuation(SF)

ZONE 10 Glaucoma hemifield test:
a. Outside normal limits
b. Border line
c. Abnormally low sensitive
d. Abnormally high sensitivity
e. Within normal sensitivity

ZONE 1 - PATIENT DATA AND TEST DATA

1 Apart from name correct age is important otherwise patient's raw data will be compared with mean threshold values of wrong age group thus wrong total deviation plot.

2 Size should be 3 -4 mm. and should be same for present and previous test, media opacities can reduce field with pupil less than 2 mm.

3 Proper near correction .

4 Fixation target could be central, small diamond or large diamond .

5 Size 3 for routine and in advanced glaucoma size 5 is used to know macular split

ZONE 2 - FOVEAL THRESHHOLD AND RELIABILITY INDICES

1 Foveal threshold is measured at the beginning , if patient is not properly focussed foveal sensitivity will be reduced along with remaining fiel. If VA is good and threshold is low correction is not proper

2 Fixation losses should not be more than 20% . if patient fixes at central fixation target he should not respond to 5 % stimuli presented on blind spot.

3 If false positive rate exceed 33% in print out XX is printed next to the rate. In established and advanced glaucoma 20% of false positive, negative and fixation losses are acceptable.

4 Unusual cause for fixation losses is that selected fixation target is central and patient fixes at diamonds .

ZONE 3 - RAW DATA

1 The raw data is the exact retinal sensitivity in db units of the selected points calculated by field analyser .Only numerical values are displayed and db units are omitted.

2 "0" indicates absolute scotoma i.e. no response to max. intensity of Light 10,000 asb units in Humprey's analyser. 40 is response to 1asb units

3 Raw data is strategy specific , hence for follow ups same strategy Should be used.

4 Whether measured sensitivity is normal or decreased can be assessed By total deviation plot not by raw data.Retinal sensitivity at 5 degree is higher than sensitivity at 10 degree so slope of vision is smooth.

ZONE 4 - GREY SCALE

1 The conversion of raw data to grey scale does not involve any statistical Calculation or normative analysis

2 The sensitivity from 50 db to 0 db is divided into 10 zones . 1st column Includes 50 db to 41 db and then each step corresponds to a change of 5db. High sensitivity areas are represented by lighter shades while low sensitivity areas are represented by darker shades.

3 The grey scale just gives rough idea of the pattern of visual field loss.

4 In high false positive, **white zones** appear while high false negatives gives **clover leaf appearance**.

5 Colour is imparted as per numerical value and that value could be normal at one location and abnormal at another so its misleading.

ZONE 5 -TOTAL DEVIATION NUMERICAL PLOT (TDNP)

1 Raw data is compared with normative data of same age group And calculates the difference between the measured sensitivity and the normative data at each point and plots the deviation as TDNP.

2. If measured sensitivity is less than normal, (-) sign is allotted and if Measured sensitivity is better than mean normal value of the same age Group , no sign is given and "0" deviation indicates no loss of sensitivity Higher (-db) indicates deep scotomas and lower (-db) values indicates Superficial

scotomas if there is no significant field loss , TDNP contains Deviation values in the range of 0 to -2 db. If there is uniform field lossTDNP contains almost similar (- ve db) values all over and difference between highest and lowest values is minimal. If irregular loss of sensitivity is there difference between highest and lowest values will be high.

ZONE – 6 TOTAL DEVIATION PROBABILITY PLOT

1 The loss of retinal sensitivity is now expressed in terms of it's p value And p value is given a symbol. Darker the symbol the greater the probability of abnormality as indicated by p value. So higher the P value Lesser the chances of field being abnormal.

2 STATPAC calculates the p value for the points where there is loss of Sensitivity. If the loss of sensitivity has P value 0 .5 % , it simply means that loss of sensitivity of that point seen is one in 200 normal population for that age but it doesn't tell whether it is abnormal or diseased.

ZONE - 7 PATTERN DEVIATION NUMERICAL PLOT

1 The pattern deviation plot is created to know the pattern and the Extent of deep scotomas masked by generalised depression in the total deviation probability plot.

2 The 7th best sensitivity point of TDNP is selected and the db value that converts it into 0 deviation is worked out .Now this db value is added to all points of total deviation numerical plot to convert it into PDN plot. The generalised depression is removed by elevating the value of Sensitivity of each point by certain db value to form the new numerical Plot and corresponding probability plot and this will expose deep scotomas.

ZONE -8 PATTERN DEVIATION PROBABILITY PLOT

1 Pattern deviation probability plot is nothing but symbolic representation of P value of PDNplot.

2. If pattern deviation probability plot does not show any scotoma , we Consider the generalised depression as uniform generalised depression.

3. In irregular generalised depression , the pattern and the extent of the Deep scotomas masked inTDPP are highlighted in PDPP If TDPP shows generalised field defect and the PDP shows shows localised scotoma , It indicates (cataract +glaucoma). If TDPP shows generalised field defect and normal pattern deviation Plot, it indicates no evidence of glaucoma in a case of cataract.

ZONE -9 (GLOBAI INDICES)

MEAN DEVIATION(MD)

- Mean deviation index signifies the average of overall severity of field loss. **It is the average of all the numbers shown on TDNP except two points in area of blindspot.** Thus the points with low variance that is closer to the fixation affect the MD value more than do the eccentric points which have a higher variance.
- The drop of the sensitivity from centre to Periphery gives characteristic The sensitivity of normal visual field is depicted by the solid diagonal lines and general depression is depicted by broken lines.
- With suspicious disc (a) A difference of mean deviation of 2 db between two eyes (b) an average 1.5 db difference must be maintained between two eyes on two consecutive tests (c) An average difference of 1db must be maintained between 2 eyes on 4 consecutive tests.
- The increase in MD >0 .08 db per year should be considered abnormal.
- The MD Index is higher generalized field defects and value of MD index in localized field defect depends on the extent and the depth of the field defects .
- It Gives shape and contour to the hill of vision.
- TheMD Index is expressed by the change in height of hill of vision and PSD is expressed by changes in smoothness of the contour of hill of vision.

PATTERN STANDARD DEVIATION

- PSD is an index to express dissimilar deviation values in the total deviation numerical plot or in other words to express contour of hill of vision whether is smooth or rough.
- If roughness is not significant PSD will be a simple number without P value
- **So PSD with significant P value indicates the numbers in the TDNP are not similar to each other.PSD helps to diagnose glaucoma at an early stage , once the glaucoma is diagnosed PSD has very minimal role in management of glaucoma.**
- Higher the irregularity in the loss of sensitivity , higher the PSD value
- In localised defects , contour is irregular without affecting the height
- When there is irregular generalised defect heightof hill is decreased as well as contour is irregular (high MD And high PSD)
- In uniform generalised depression contour of hill is smooth and height of hill is decreased (Low PSD and high MD)
- **Increase in MD Index and no change in PSD indicate there is a progression of the field defect and it is uniform generalised type.PSD will be high in localised and irregular generalised field defects.**

SHORTTERM FLUCTUATION(SF) :

The SF and and corrected pattern standard deviation (CSPD) will Be calculated by full threshold and FAST PAC strategies. SITA Strategies donot calculate SF and hence CSPD cannot be calculated. SF value is always less than 3 db. and is a indicator of reliability

CORRECTED PATTERN STANDARD DEVIATION(CPSD)

Intra testing variability is removed from PSD to produce CPSD .CPSD is not calculated if SF is not estimated.

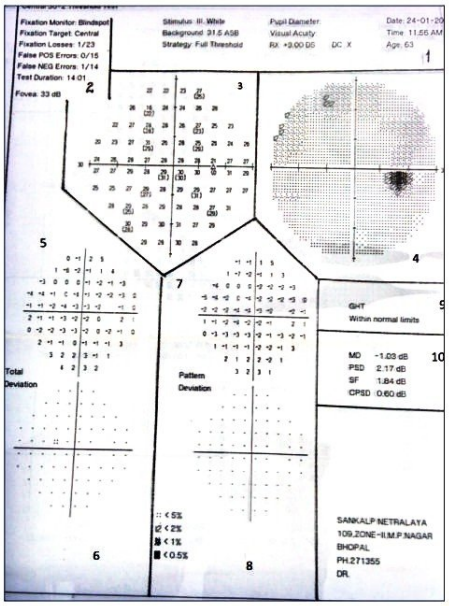
ZONE 10 - GLAUCOMA HEMIFIELDTEST

GHT is refined PSD . GHT is developed to pick up the dissimilarity among the sensitivities of the corresponding points on either side of

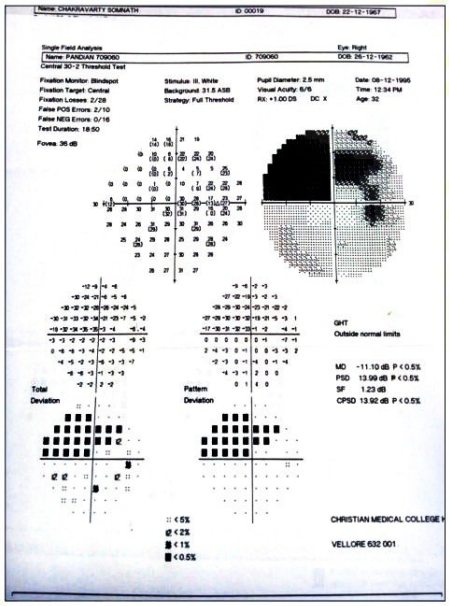
horizontal axis to diagnose glaucoma at an early stage .GHT evaluates five zones in the upper field and compares these zones to mirror image zones in the lower field . Grading is as follows:

1. **Out side normal limits**
2. **Borderline**
3. **General reduction of sensitivity**
4. **Abnormally high sensitivity**
5. **Within normal limits**

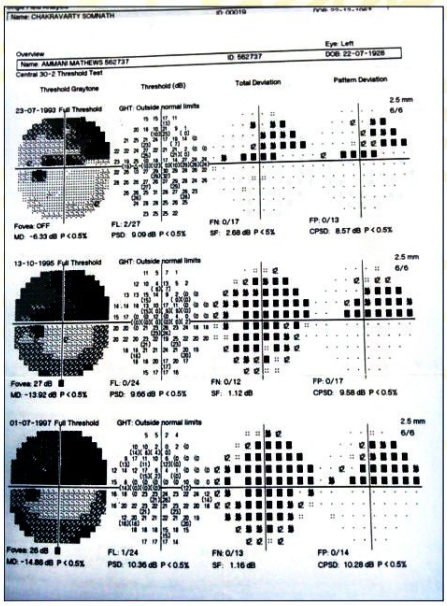
Apart from this we should know about strategies also and in follow ups we should keep in the mind about starategy followed earlier. As the standard threshold strategy takes longer time to perform now new strategy (SITA – Swedish interactive threshold Algorithm) strategies are available without really compromising the results.



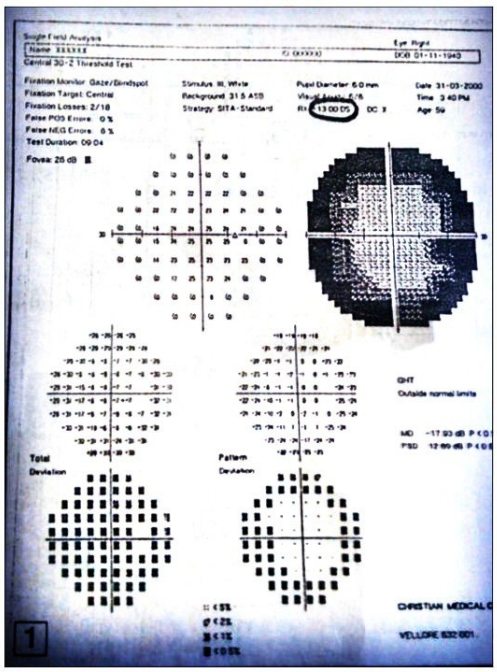
Normal Fiedl Chart Divied into 10 Zones



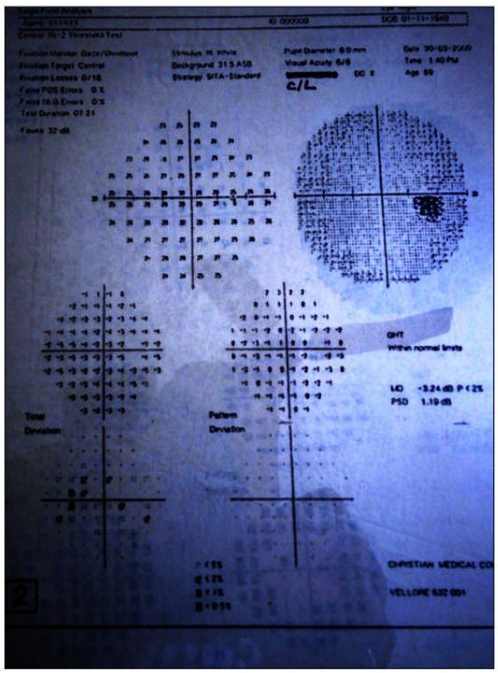
Superarcuate defect Sup.Caecocentral scotoma



Progression of glaucomatous defect in Follow up print out



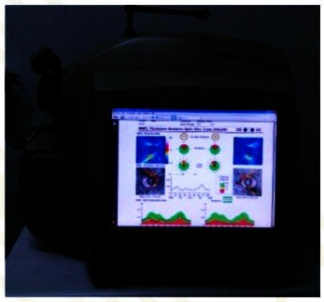
Classic Ring Scotoma Due to Ahakic glasses



scotoma vanished with contact lens

ROLE OF OCT IN DIAGNOSIS OF GLAUCOMA

Dr Hemlata Yadav (Assist. Prof.), **Dr Anjali Sharma** (Assist. Prof.),
Dr Deepti Sinha Mr K B Upadhyay, BMHRC Bhopal.



Introduction :- Glaucoma is a progressive optic neuropathy resulting in characteristic damage to the optic nerve and defects in the visual field, because it can lead to irreversible vision loss, timely identification of glaucoma is crucial yet diagnosis is often uncertain. Early changes are apt to be subtle, and structural and functional defects commonly do not appear simultaneously. Neural damage often manifests before statistically significant visual field changes.

Common characterstics of glaucomatous neuropathy include

1. An increase in the cup-to-disc ratio
2. Verticalization of the cup.
3. Notching of the neuroretinal rim.
4. Nerve fibre layer defects.
5. Vessel changes within the optic nerve head.
6. Disc haemorrhage.

There is often significant disagreement in assessing the optic nerve, even among experienced clinicians. A disc with a large cup may be normal, while one with a small cup may be glaucomatous. A number of conditions including high myopia, tilted discs, or optic pits also affect the optic nerve, making it more difficult to identify glaucomatous optic neuropathy in their presence.

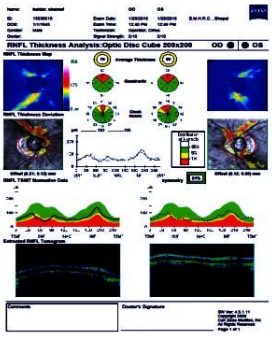
Optical coherence tomography (OCT) is a commonly used imaging technology in the evaluation of glaucomatous structural damage. OCT was introduced over 20 years ago and is a non-invasive optical technique that allows in vivo cross-sectional imaging of the ONH and retina. The recent commercially available iteration of the OCT technology, spectral domain (SD)-OCT, has theoretical advantages in glaucoma assessment over the earlier generation of time domain (TD)-OCT due to increased axial resolution and faster scanning speed that lead to lower susceptibility to eye movement artefacts. The evidence to date suggests that SD-OCT offers improved reproducibility; however, the glaucoma diagnostic accuracy of SD-OCT and TD-OCT is statistically similar.

SD OCT Parameters

There are three main parameters relevant to the detection of glaucomatous loss. 1. Retinal nerve fibre layer 2. Optic nerve head 3 Ganglion cell complex

Retinal Nerve fibre Layer

Glaucoma is a group of many conditions sharing a final common pathway characterized by accelerated death of retinal ganglion cells and their retinal nerve fibre layer (RNFL) resulting in characteristic visual field defects and corresponding optic nerve head anatomical changes. SD-OCT can directly measure and quantify RNFL thickness by calculating the area between the internal limiting membrane (ILM) and RNFL border .The Cirrus RNFL map represents a 6 x 6 mm cube of A-scan data centred over the optic nerve in which a 3.4 mm diameter circle of RNFL data is extracted to create what is referred to as the TSNIT map (temporal, superior, nasal, inferior, temporal). It is displayed as a false colour scale with the thickness values referenced to a normative database. The TSNIT map displays RNFL thickness values by quadrants and clock hours, and the RNFL peaks give a sense of the anatomic distribution of nerve fibre axons represented by the superior and inferior bundles that emanate from the optic nerve.



Assessment of Optic nerve Head

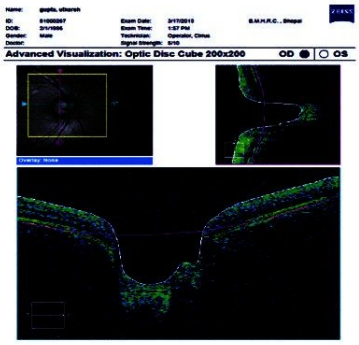
The gold standard for assessing the optic nerve head for glaucomatous changes has traditionally been stereo disc photography. Photographic interpretation, however, is both qualitative and subjective. In recent years, imaging devices such as spectral-domain optical coherence tomography (OCT) have become more common place in the diagnosis and management of the disease.

Ganglion cell complex The ganglion cell layer is thickest in the perimacular region and decreased macular thickness has been observed in glaucomatous eyes likely due to thinning of the ganglion cell layer in this region. However segmenting the ganglion cell layer alone is very difficult based on reflectivity and thus Cirrus chose to measure its Ganglion Cell Analysis (GCA) consisting of the combined ganglion cell layer (GCL) and inner plexiform layer (IPL). The Cirrus macular scan map is displayed using a similar colour scale and divided into various pie sectors around the fovea. Again the calculated sectors are compared to a normative database.

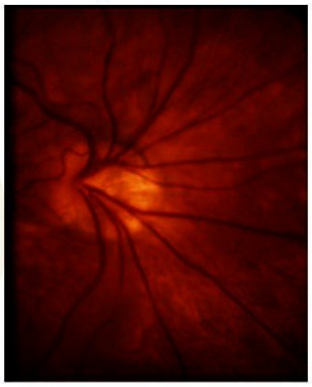
Interpretation of OCT finding

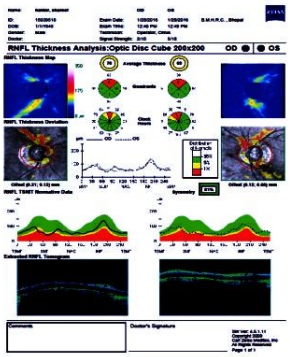
1. Figure shows the OCT of 72 year old woman who was followed as glaucoma suspect due to having maximum IOP in the high 20s mm of hg . The RNFL thickness map, thickness deviation and ISNT curves shows RNFL thinning superiorly greater than

inferiorly in both eyes. A close look at the optic nerve photograph shows tilted hypoplastic disc with peripapillary atrophy and absence of characteristics glaucomatous cupping.



The assessment was that the abnormal OCT was due the hypoplastic disc rather than the glaucoma. In this case the OCT alone cannot distinguish between abnormalities due to glaucoma and anomalous disc





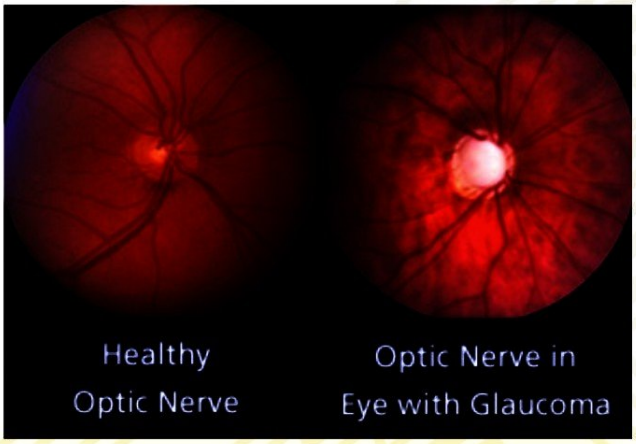
2. Figure showing normal optic disc and classical glaucomatous damage of optic disc along with OCT finding of these patients.

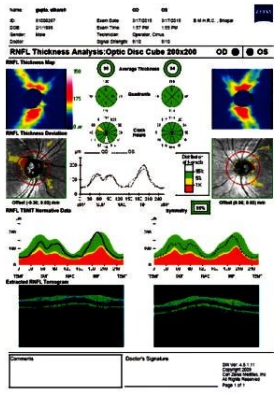
Repeatability and Reproducibility of SD-OCT

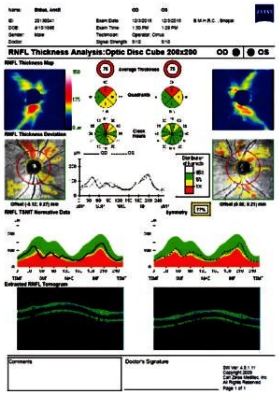
Studies have evaluated the reproducibility and repeatability of SD-OCT measures relevant to assessing change over time. One study of Cirrus found excellent intra visit and inter visit reproducibility of measurements of peripapillary RNFL thickness and ONH parameters. The inter visit tolerance limit for average RNFL thickness was 3.89 µm suggesting that a reproducible decrease of 4 µm or more may be a statistically significant change. A more cautious cut off would be a 10 µm change, roughly twice the maximum standard deviation found in that study. However, it is important to exclude any scans that are not of adequate quality. Scans with a signal strength less than 6, with eye movement or blinking artefacts within the 1.73-mm radius around the optic nerve head, or with segmentation errors should be repeated

Trend and Event Analysis

Glaucoma progression algorithms can be divided into event-based and trend-based approaches, similar to visual field progression detection methods. Event-based analysis defines progressive change when a follow up measurement exceeds a pre-established threshold for change from baseline. Alternatively, trend-based analysis defines progression by monitoring the change over time using regression analysis to provide a rate of progression and corresponding significance level.







Use of SD-OCT in Detection of Glaucomatous Progression

Guided Progression Analysis (GPA)

GPA, introduce in 2009 on the Cirrus, compares the RNFL thickness of individual clusters of A-scans, referred to as pixels, between baseline and follow up RNFL thickness maps to estimated test-retest variability. Local pixels exceeding such test-retest variability are coded in yellow at the first event and in red if the same changes are seen on three consecutive images. In order to generate an overall trend plot, two baseline scans with three follow up scans are necessary.

Progressive RNFL Thinning

Progressive RNFL thinning measured on SD-OCT can often be used to detect progressive disease. The top three RNFL progression patterns are;

1. Widening of an existing RNFL defect.
2. Deepening without widening of an existing RNFL defect,
3. Development of a new RNFL defect.

In one study, the inferotemporal quadrant was the most frequent location for RNFL progression.

Correlation of OCT with Visual Field

Due to the variability or possible artefacts with SD-OCT measurements, all changes on OCT should be correlated with visual field changes before confirming definite progression. When such correspondence is not found, caution should be exercised and sources of erroneous measurements should be sought.

Conclusion: OCT is a valuable clinical tool for glaucoma diagnosis and detection of progression. RNFL parameters have been demonstrated to provide accurate information for disease diagnosis and sensitive method for disease progression. Initial studies evaluating macular and ONH parameters show encouraging results.

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HEIDELBERG RETINA TOMOGRAPH GDX NERVE FIBER LAYER ANALYZER

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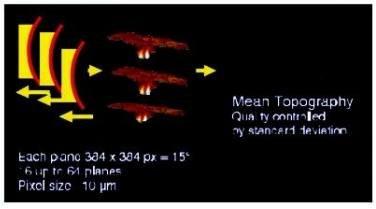
Glaucoma causes typical structural changes in the optic nerve head and retinal nerve fiber layer (RNFL). In clinical practice, the evaluation of structural change is performed by fundus biomicroscopy but it lacks objectivity and quantification. Objective and quantitative measurements (HRT GDx and OCT) of the optic nerve head and RNFL can complement the assessment of glaucomatous damage and can aid in detecting disease progression. Heidelberg Retina Tomograph is a Confocal laser scanning ophthalmoscope used to measure the Retinal Nerve Fiber Layer (RNFL) thickness at the Optic Disc in Glaucoma patients. It uses a 670nm low power laser and acquires 3 separate image series where each image comprises of 16-64 planes and each plane itself is formed of 384*384 pixels. Each pixel is 10u in size and the image quality is measured as standard deviation.

The Principle

It is observed that Papillomacular Bundle (PMB) remains intact even during very advanced stages of Glaucomatous optic neuropathy(GON) . Its thickness is 50 um and this is where the HRT defines the reference plane .The component above this considered the Neuro-retinal Rim (NRR) and the component below is the cup. Based on this premise,

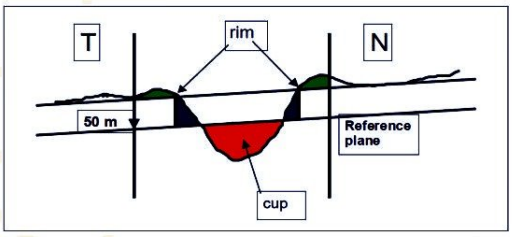
various stereo-metric parameters are calculated and the five important ones are

- Rim Area
- Rim Volume
- Height Variation Contour
- Mean RNFL Thickness
- Cup Shape Measure

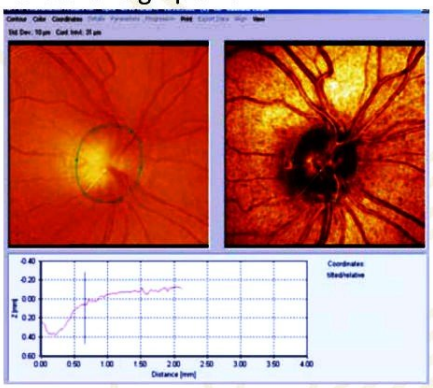


Recording

First the camera is adjusted by cleaning the chinrest, front fixation and the objective. Expected refraction is adjusted on the objective and if required, astigmatism correction lenses are applied. Camera takes two images, a topography image and a reflectance image. The quality of the image is determined by the displayed image quality display.



Using 6-8 points a contour line is marked to delineate the ONH.The contour line has to be placed at the inner edge of the scleral ring. The depressed band can be identified as a 'valley' using the interactive height profile



HRT Printout

Section 1- Patient examination & data

Section 2 -

- Red = Cup
- Blue = Sloping Rim
- Green = Stable Rim

Section 3 - Stereo-metric parameters (Top5)

- Rim Area
- Rim Volume
- Cup Shape Measure

Height Variation Contour

- Mean RNFL Thickness

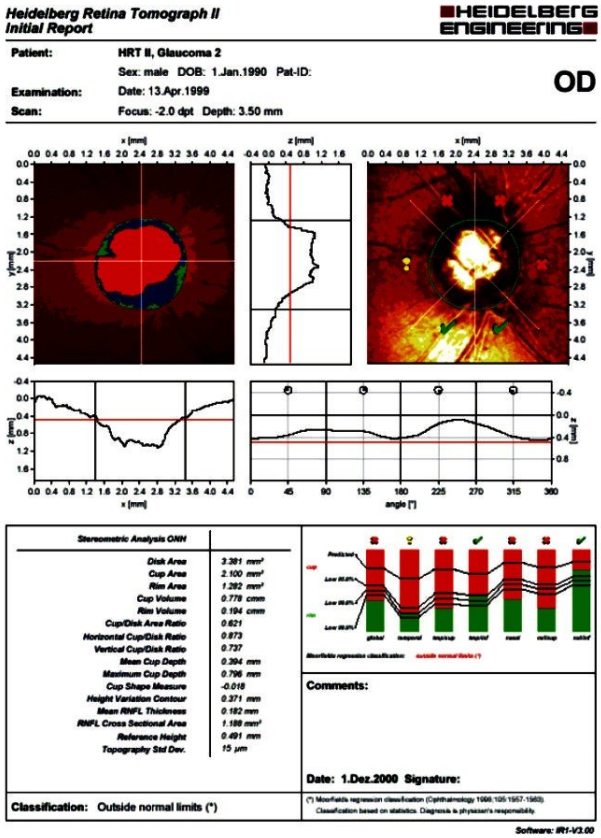
Standard Deviation

- < 10 Excellent
- 10-20 Very Good
- 20-30 Good
- 30-40 Acceptable
- 40-50 Probably Cataract

Section 4 – Reflectance image

Compared to Normal Database with Simple Classification

- Normal (Green)
- Borderline (Yellow)
- Outside Normal (Red)



Section 5- Retinal surface height profile

- Along Contour Line(Green)
- Reference Plane (Red) set 50 microns below Papillomacular bundle

Section 6- Moorfield's regression analysis

- Column = Total ONH
- Cup (Red) v Rim (Green)
- Age Dependent Confidence Intervals

a) "Within Normal Limits" (Green) % Rim 95% Limit

b) "Borderline" (Yellow) % Rim between 95% and 99.9% Limits

c) "Outside Normal Limits" (Red) % Rim lower than 99.9%

Interpreting Progression

Progression requires 3 consecutive readings (Baseline + 3 follow-up) to perform a Topographic Change Analysis (TCA).

TCA can be done by two methods:

i) Change Probability Maps

ii) Stereo-metric Parameters

(i) Change probability maps are independent of the reference plane and the contour line and are calculated automatically comparing mean topography images.
Red signifies "Significant" Depression
Green signifies "Significant" Elevation

A height change is considered significant

- If it is repeated in at least two (better: three) consecutive follow-up examinations
- If it is region of at least 20 connected super-pixels.

(ii) Parametric change is evaluated in the follow-up diagram that plots normalised streo-metric values vs. time.

If average normalised parametric values decrease by more than -0.05 significant in 2 consecutive examinations it is deemed "suspected"

If it appears in 3 consecutive examinations it is considered "confirmed" progression

GDx Nerve Fiber Layer Analyzer

The GDx nerve fiber layer analyser is a Scanning laser polarimeter

Principle: Infrared Laser light enters the eye at specific orientation. As it goes through tubules, it returns at a different orientation/ axis. This delays return and this difference is the measurement of the fiber thickness. Laser double passes the retinal nerve fiber layer (NFL) and is split into 2 parallel rays by the birefringent fibers. The two rays travel at different speeds and this difference (called retardation) is directly correlated to the nerve fiber thickness. 65,536 points are measured in a full 15 x 15 degree grid centered at the optic nerve head. The central ellipse denotes an area of 1.75 disc diameter in size.

The sensitivity and specificity are said to be 96% and93% respectively

Measurement: It is performed with an undilated pupil of at least 2mm diameter. Time taken is 0.7 seconds. Total chair time is less than 3 minutes for both eyes. The test is totally objective.

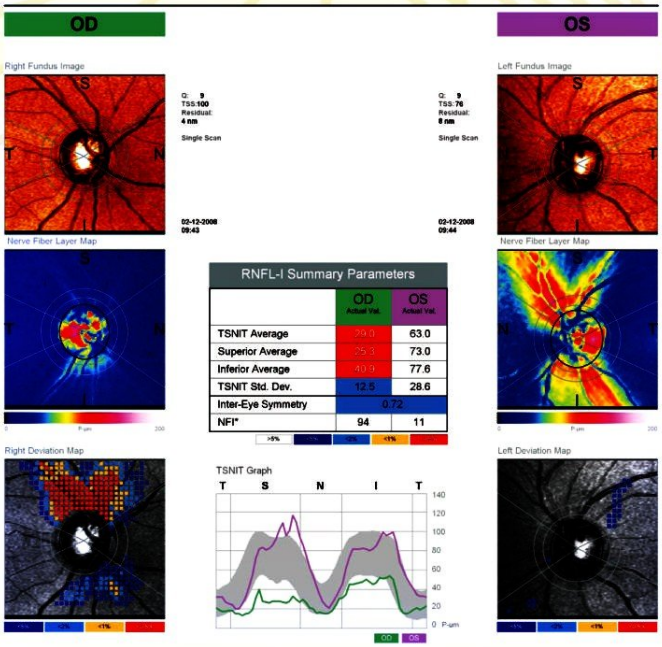
Image quality check: The quality of image is affected by cataracts and poor media clarity. Scanning ellipse should be placed around optic nerve. A black border around the scan indicates eye movement during imaging.

GDx: Normative Values

Normative data base from healthy volunteers is available for different races. Parameters thought to best differentiate normals from glaucoma patients were identified and comparison of patients is made to age- and race matched normals.

GDx Nerve Fiber Layer Analysis: Interpretation

The first image is a color fundus image.



Thickness (Polarization) Map
Bright colors (yellow and red) indicate thick NFL
Dark colors (blue) indicate thin NFL

Deviations from normal are highlighted in
Yellow if they are borderline ($p<0.10$)
Red if they are outside normal limits ($p<0.05$)
Green for all normal values
Normal pattern is a symmetrical hourglass shape of bright colors superior and inferior and dark colors nasal and temporal.

Abnormal pattern includes:

1. Diffuse loss of NFL, causing areas that should be yellow to fade to red
2. Focal defects are seen as concentrated dark areas
3. Asymmetry between superior and inferior quadrants
4. Asymmetry between the 2 eyes
5. Higher than normal nasal and temporal thickness (red and yellow where blue should be)

TSNIT (Double Hump) Graph

It is a quick, easy look at how the patient compares to normal.

In evaluating TSNIT graph, one needs to consider the symmetry between superior and inferior humps. A flat TSNIT means loss of NFL.

Symmetry Analysis Gives an overlay of OD and OS of TSNIT graphs of patient

Nerve Fiber Analysis It compares patient's thickness values to an age and race-matched population of normals and evaluates a series of ratios, averages, and other parameters. Any parameter flagged as 'borderline' or 'outside normal' indicates that the patient does not match normative values . No single parameter is more or less sensitive in detecting glaucoma.

Diagnostic parameters

Diagnostic Parameters	*OD Actual Val.*	*OS Actual Val.*	*Difference from OD*
The Number	11	14	3
Symmetry	1.18	1.28	8%
Superior Ratio	5.07	4.89	4%
Inferior Ratio	4.30	3.82	11%
Superior / Nasal	2.84	3.69	23%
Max Modulation	4.07	3.89	5%
Superior Maximum	84.43	94.80	11%
Inferior Maximum	71.49	74.00	3%
Ellipse Modulation	7.47	3.73	50%
Normalized Sup. Area	0.1534	0.1620	5%
Normalized Inf. Area	0.1292	0.1399	8%
Ellipse Std. Dev	25.14	24.95	1%
Ellipse Average	54.07	56.61	4%
	p>10%	p<10%	p<5%

Nerve Fibre Index: The Neural network analyzes more than 200 parameters from GDx image and assigns a number where '0' means normal and '100' means glaucoma.

Currently the following guideline should be used:

0-30 normal (low likelihood of glaucoma)
31-70 glaucoma suspect
71-100 high likelihood of glaucoma

One must note that the Number is highly dependent upon ellipse placement.

Rule of thumb:

Everything 50 and under is good and normal

Everything over 51 is suspect.

The closer to 100 that the number is, the higher the likelihood of glaucoma.

The next four parameters- Symmetry, Superior Ratio, Inferior Ratio, and Superior/ Nasal Ratio demonstrate sensitivity and specificity for glaucoma of 96% and 93% respectively

Symmetry: Ratio of the average of the 1500 thickest pixels in the superior quadrant over the average of the 1500 thickest pixels in the inferior quadrant. The closer to 1.0 this number is, the more symmetrical the NFL superior and inferior and the more normal the patient.

Superior Ratio: The ratio of the average of the 1500 thickest pixels in the superior over the average of the 1500 median pixels in the temporal quadrant

Inferior Ratio: The ratio of the average of the 1500 thickest pixels in the inferior over the average of the 1500 median pixels in the temporal quadrant

Superior/ Nasal: The ratio of the average of the 1500 thickest pixels in the superior over the average of the 1500 median pixels in the nasal quadrant

Max Modulation: An indication of the difference between the thickest to thinnest parts of the NFL

- The higher the number, the greater the difference
- In a normal eye where the superior and inferior region is thicker than the nasal or temporal region, the modulation number will usually be greater than 1.0. Lower numbers are associated with glaucoma

Superior Maximum: The average of 1500 thickest pixels in the superior quadrant

Inferior Maximum: The average of 1500 thickest pixels in the inferior quadrant

Ellipse Modulation: An indication of the difference between the thickest and thinnest parts of the NFL .

Normalised Sup area: The total area (in mm2) under the curve of the NFL along the superior portion of the ellipse surrounding the ON

Normalised Inf area: The total area (in mm2) under the curve of the NFL along the inferior portion of the ellipse surrounding the ON

Ellipse Average: The average thickness (in microns) of the NFL in the ellipse surrounding the ONH

TSNIT Parameters		*OD Actual Val.*		*OS Actual Val.*
TSNIT Average		54.07		56.61
Superior Average		71.17		75.86
Inferior Average		62.60		63.96
TSNIT Std. Dev.		25.14		24.95
Inter-Eye Symmetry			0.92	
NFI		11		14
p>5%	P<5%	P<2%	P<1%	P<0.5%

TSNIT parameters

TSNIT average: Average thickness of all pixels in the image

Superior Average: The average thickness (in microns) of the NFL in the superior portion of the ellipse

Inferior Average: The average thickness (in microns) of the NFL in the inferior portion of the ellipse surrounding the ONH

Despite all the advanced technology and machines, one should not forget the most valuable HRT, GDx and OCT present in our pockets

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MEDICAL MANAGEMENT OF GLAUCOMA

DR. Joginder Singh, Glaucoma Consultant

MANAGEMENT OF GLAUCOMA CAN BE DISCUSSED UNDER THE FOLLOWING HEADS :

A. SET A TARGET PRESSURE

a. BASELINE IOP
b. BASELINE DAMAGE
c. BASELINE TARGET PRESSURE
d. WEIGHTED BASELINE TARGET PRESSURE

B. LOWER THE PRESSURE

a. DRUGS
b. LASERS
c. SURGERY

C. REVIEW THE RESPONSE

a. PATIENT REPORT
b. EFFECT-EVALUATION
c. THERAPY MODIFICATIONS

A. SET A TARGET PRESSURE

A theoretical IOP which will either stop further damage or slow it down so considerably as to make it of no consequence to the patient.

May vary in the two eyes of a patient due to varying severity of glaucoma.

May vary in two patients with apparently similar severity of glaucoma.

a. BASELINE IOP

1. Ideally, diurnal readings--with IOP taken every 2-3 hours for 24 hours--are needed.
   However, 3-5 readings--covering morning, afternoon, and night--may suffice.
2. Either the highest IOP or the mean IOP may be used as the baseline IOP.
   Using just one IOP reading to arrive at the baseline IOP is strongly discouraged

b. BASELINE DAMAGE

1. Both field loss and nerve damage classified as early, moderate and severe.
2. Either field loss or nerve damage or both may be used as the baseline damage.
   Using tedious measurements to arrive at the baseline damage is not recommended

c. BASELINE TARGET PRESSURE

1. Early baseline damage -- 20-30% reduction from the baseline IOP.
2. Moderate baseline damage -- 30-40% reduction from the baseline IOP.
3. Severe baseline damage -- 40-50% reduction from the baseline IOP.
   Using complicated formulas to arrive at the baseline target pressure is unnecessary

d. BASELINE TARGET PRESSURE WEIGHTED

Adding an extra 10% reduction from the baseline IOP :

1. Patients younger than 50 years of age.
2. Patients who are monocular for whatever reason.
3. Patients who have a family history of glaucoma, especially severe glaucoma.
4. Patients who have other optic nerve or retinal pathology so follow-up is tougher.
   Multiple other factors may need consideration when deciding on target pressures

B. LOWER THE PRESSURE

a. DRUGS

1. (1) Usually preferred by the patients.
   (2) Side effects not too much of an issue.
2. Drugs are the usual first choice for lowering the pressure .
   Drugs are costly, bothersome, affect quality of life and may jeopardise later surgery

b. LASERS

1. (1) Not that effective and lose effect over time.
   (2) Question of easy accessibility may be an issue.
2. Laser trabeculoplasty as the initial choice for lowering the pressure :
   (1) Compliance issues.
   (2) Inability to use drops.
   (3) Contradiction to drops.
   (4) Patients with PXG or PG.
   Lasers can be quantified, sometimes repeatable and do not jeopardise later therapy

c. SURGERY

1. (1) Success is not always assured and lasting.
   (2) Potential complications may be a serious issue.
2. Incisional surgery as the initial choice for lowering the pressure :
   (1) Very high pressures.
   (2) Frequency of follow-up is an issue.

Surgery is cheaper in the long-term, gives the best control and lessens the follow-up

C. REVIEW THE RESPONSE

a. PATIENT REPORT

1. Tolerance.
2. Compliance.

Before effect evaluation be sure there are no tolerance and/or compliance issues

b. EFFECT-EVALUATION

1. Percentage lowering of IOP.
2. Target pressure achieved or not.

If a drug gives < 20% IOP reduction, use another drug or treatment and start again

c. THERAPY MODIFICATIONS

1. With target pressure achieved :
   Plan the follow-up schedule.
2. With target pressure not achieved :
   (1) Increase the dose or frequency of the initial drug and repeat the cycle.
   When used in full dosage, decide if the reduction is acceptable :
   i. If IOP is below the target pressure, plan the follow-up schedule.
   ii. If IOP is above the target but within 2-3 mmHg of the target pressure
   - in early disease, accept it and plan the follow-up schedule.
   - in moderate or severe disease, substitute or add another drug as below.

   (2) Substitute the initial drug and/or add another drug to it and repeat the cycle.
   On full, well-tolerated, topical therapy, decide if the reduction is acceptable :
   i. If IOP is below the target pressure, plan the follow-up schedule.
   ii. If IOP is above the target pressure but at least 20% below the baseline :
   - in early disease, accept it and plan the follow-up schedule.
   - in moderate or severe disease, use additional therapy as below.

   (3) Additional therapy with oral CAI, laser trabeculoplasty or incisional surgery.
   Using the above additional therapy, decide if the reduction is acceptable :
   i. If IOP is below the target pressure, plan the follow-up schedule.
   ii. If IOP is above the target pressure but at least 20-30% below the baseline :
   - in moderate disease, at 20% plan the follow-up schedule.
   - in severe disease, at 30% plan the follow-up schedule.
   In advanced glaucoma a 50% reduction or an IOP of 10-12 mm Hg is the target
   In end-stage glaucoma a 60% reduction or an IOP of 10 mm Hg or less is sought

LASERS IN GLAUCOMA

Dr Prateep Vyas
Medical Director - Centre For Sight, Indore
Hon. General Secretary Glaucoma Society of India

Lasers are used in Glaucoma to

1) Reduce IOP
2) Control of IOP
3) Prophylactic Treatment for Primary Angle Closure Suspect(PACS)
4) To relieve symptoms in intractable painful glaucoma
5) Performing anti glaucoma surgery

Lasers used in Glaucoma

1) YAG laser(Including frequency double)
2) Argon laser
3) Diode laser
4) Excimer laser

Laser Procedures performed in Glaucoma

1) Primary open angle glaucoma
   Selective laser trabeculoplasty(SLT)
   Argon laser trabeculoplasty(ALT)
2) Angle closure glaucoma
   Laser Iridotomy

Iridoplasty

3) Surgical treatment

Trans scleral diode cyclo photo co agulation(TSCPC ,Diode CPC)

Excimer laser assisted Trabeculectomy

CO2 laser assisted Sclerostomy(CLASS)

Argon laser suterolysis

Endo CPC

Nd YAG Goniopuncture

Nd YAG Hyelodotomy

Nd YAG Internal revision of Trabeculectomy

Argon laser to shrink overhanging bleb

SLT

SLT is commonly used to treat patients with open-angle glaucoma [i.e. primary open-angle glaucoma (POAG), pigmentary glaucoma, exfoliative glaucoma]. However, recent studies have examined the efficacy and safety of SLT to lower IOP in other glaucoma subtypes as well including ACG

Method

Use the lowest energy setting needed in order to release champagne bubbles; start at 0.6-0.8 mJ and titrate upwards or downwards, as required.In order to prevent post-procedural IOP spike, always administer miotics and a tab of Diamox 250mg one-hour prior to treatment.Use a Latina lens due to its zero magnification.Apply 50 spots over 180-degrees of the trabecular meshwork (TM); apply 100 spots over 360-degrees of the TM.Apply topical NSAID post-procedure for three days, in order to prevent IOP spike. A heavily pigmented TM requires less energy. Take care not to confuse pigmentation of Schwalbe's line with pigmentation of the TM. Focus the treatment beam over the full height of the TM in order to achieve optimal results.Check IOP post procedure at one-hour and four-hour intervals, at day 3 and 7, at week 4 and week 14, followed by quarterly check-ups.

Iridoplasty

Note:

approximately 20 percent of patients experience an IOP spike following the procedure.

When performing repeat SLT.Wait one month in order to evaluate treatment performance; if target IOP is not achieved, continue anti-glaucoma medication.

YAG Iridotomy for ACG

Laser iridotomy isused to treat nearly all types of narrow or closed-angle glaucoma. The Nd:YAG laser alone is fre-quently used to perform the iridotomy but mayfollow pretreatment with an argon laser, particularlyfor thick, heavily pigmented irises or patients onblood thinners in whom the risk of bleeding may beincreased.LPI has also been utilized in the treatment of pigmentary dispersion syndrome (PDS) based on thetheory of reverse pupillary block as a primary mechanism.

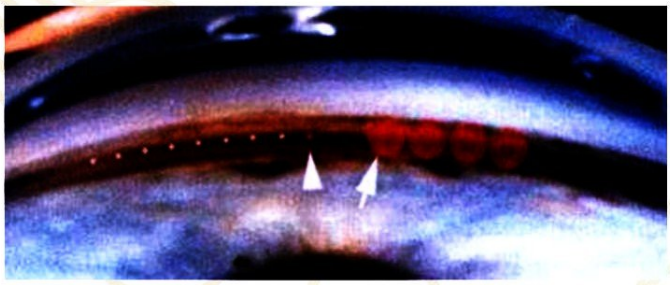
Method

Prepare the Pupil by constricting with frequent Instillation of Pilocarpine drops.Identify iris crypt at superior location. Slightly de focus aiming beam posteriorly Use 3-6 MJ single burst Usually 2-3 shots are adequateLook for aqueous gush with pigments.Treat the patient with topical steroids for 3-5 days

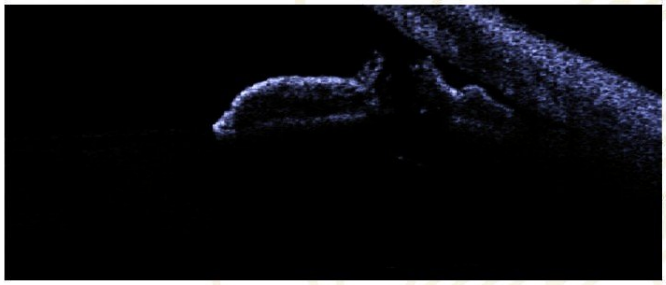
Iridoplasty

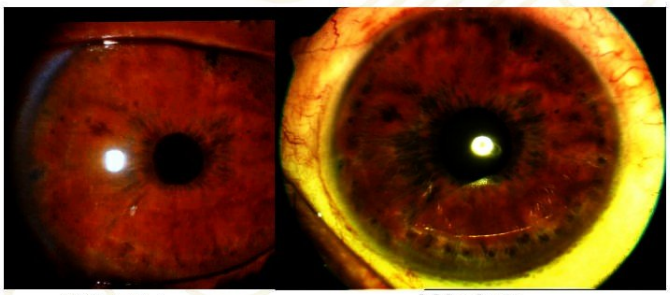
Indications :

1. Plateau iris syndrome
2. An unbreakable attack of Angle closure glaucoma where laser iridotomy is not possible
3. Angle closure glaucoma due to lens intumesence.
4. Adjunct to laser trabeculoplasty Done with Argon laser,use Gonio lens Argon laser setting at 500microns spot size, 0.2 to - 0.5 second duration and Power between 200 – 400 mJ are applied with the contact lens in place aiming at the about 1 mm away from the root of Iris. Approximately 20-40 spots are given over the 360 degree.



SLT





YAG Iridotomy

Iridoplasty

LASERS IN GLAUCOMA

Indications:

For reduction of IOP in eyes with refractory glaucoma, unresponsive to conventional outflow enhancing surgeries, like Trabeculectomy, and glaucoma valve implants.Can be a primary procedure in eyes with limited visual potential.

Procedure:

The procedure is performed under retro bulbar or peribulbar anesthesia. A810nm semiconductor diode laser with a G probe (600 µm diameter quartz fiber with a spherical protruding tip oriented by the footplate of the hand-piece).

Laser settings:

With a power of 1500 to 2000 mW for 1-1.5 sec.Hear a audible "pop" sound.

About 18 applications over 270 degrees is good

Further Reading

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SURGICAL MANAGEMENT OF GLAUCOMA

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The goal of glaucoma therapy is to maintain good vision for the patient's lifetime. To make the right recommendation to the patient, the surgeon must consider the life expectancy of the patient, the rate of progression of the disease and the possible benefit of less serious additional therapies. The surgeon must weigh the surgical benefit (likelihood of successfully preventing further visual loss) against the risks of surgical failure or complications.

INDICATIONS FOR THE FILTERING SURGERY

1. Documented progressive deterioration of visual field or optic nerve damage, despite maximum tolerated medications and laser therapy, that threatens patients vision in their lifetime.
2. Anticipated progressive damage eg. experience in the same or fellow eye that indicates the current course will lead to loss of vision, very high intraocular pressure, other parameters unreliable.
3. Medication failure due to ineffectiveness, intolerance, poor compliance or complications.
4. Dysfunctional ocular tissues, corneal oedema ,pulsating central retinal artery.
5. Combined with cataract procedure if there is borderline intraocular pressure control, advanced damage, history of postoperative intraocular pressure rise in the fellow eye.

SURGICAL PATHOPHYSIOLOGY.

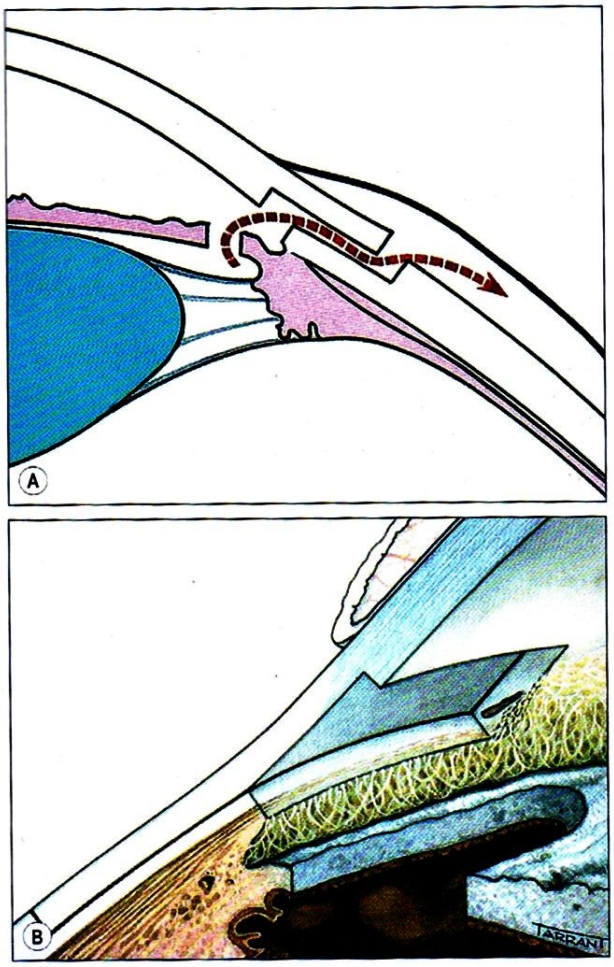
No single operative procedure is optimal for all forms of glaucoma and it is necessary to tailor the particular surgery to fit the requirements of the individual case.

Surgery in glaucoma is directed at modifying aqueous dynamics that will prevent or reduce the intraocular pressure elevation. Unfortunately lowering intraocular pressure does not always stop progression of visual field loss, thus surgery may be successful but disease continues. The immediate goal of surgery is to control the intraocular pressure with fewest possible complications and the long term goal is to prevent further loss of vision. By careful documentation of postoperative state by gonioscopy, visual field test, optic nerve head drawing, visual acuity and refractive error recording and comparisons with preoperative records Glaucoma surgery can be divided into two categories, procedures to eliminate block to flow of aqueous humor and procedures to reduce the production of aqueous humor. **Fig 1 A and B**

CLASSIFICATION OF AQUEOUS FLOW BLOCK AND SURGICAL MANAGEMENT.

1. **Internal Flow Block**- Blocks the flow of aqueous in the eye
   - **A.** Cilary block glaucoma-treatment by **removal of vitreous, rupture of anterior hyaloid and restoration of anterior chamber**
   - **B.** Pupillary block glaucoma- Treatment by **peripheral iridectomy**

TRABECULECTOMY PRINCIPLES FIG 1 A AND B



A PATHWAY OF AQUEOUS EGRESS FOLLWING TRABECULECTOMY.BAPPERANCE FROM INSIDE THE FOLLOWING COMPLETION

2. **Outflow Block**-blocks the flow of aqueous out of the eye. treatment by **trabeculectomy or treat the primary cause.**

   A. Trabecular Block Glaucoma-aqueous prevented from reaching schlemms canal from the anterior chamber.

   1. Trabecular Covering-

   a. Iris against the trabecular meshwork-

   1. With ciliary or pupillary block.

   2. Witoutciliary or pupillary block(iris apposition, peripheral anterior synechiae,plateau iris.)

   3. Posterior space occupying lesions(expulsive hemorrghe,uvealeffusion,block to vortex venous return as with encircling bands for retinal detachment.

   b. Fibrous,fibrovascular or cuticular membranes.

   c. Tumours.

   2 .Trabecular Impermeability.

   a. Primary (primary open angle glaucoma). treatment by trabeculectomy and setons.

   b. Secondary (pigment, exfoliation , zonular fragments after alpha chymotrypsin, uveltis, macrophages, blood debris ,traumatic tear of meshwork, tumour)

   c. Developmental-TREATMENT BY TRABECULECTOMY AND GONIOTOMY

   1. Trabecular dysgenesis(infantile glaucoma)

   2. Mesodermal and ectodermal goniodysgenesis (Riegers, Axenfelds, Peters anomalies, aniridiaand others)

   B Limbal Block-resistance to outflow through collector channels or aqueous vein.treatment of primary cause .

   1 Increased venous pressure

   aPrimary.

   b.Arteriovenous fistula and others.

   2 External scarring (chemical burns ,diseases like trachoma, pemphigus

   3 External tumors(such as conjunctival lymphoma)

If there is no internal flow block and intraocular pressure remains too high despite maximally tolerated medical therapy surgery to relieve outflow block is needed.

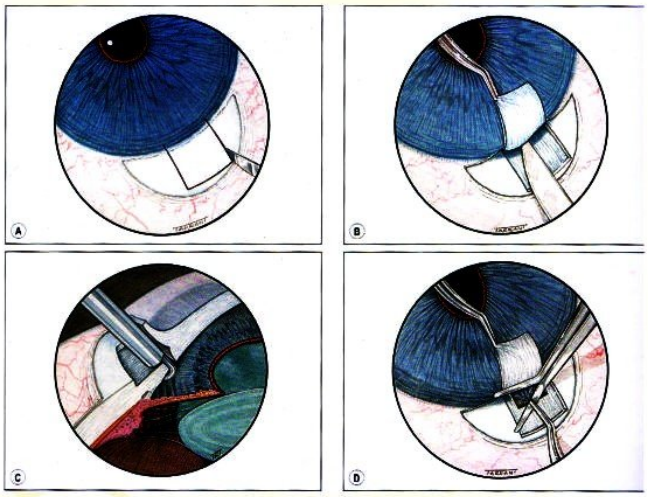
External Filteration Surgery-- are two types guarded filteration procedures and full thickness procedures.

1. **Full Thickness Procedure—**thermal sclerostomy,posterior or anterior lip sclerectomy or elliots trephination have no guard over the external surface of the sclereostomy other than conjuntiva and tenon's capsule.these procedures are referred to as the scheie procedure.these procedures were associated with high degree of postoperative complications incuding shallow anterior chambers,premature cataracts and late infections.

2. **Guarded Filteration Procedures-** cairns introduced modern day trabeculectomy in 1960s.major filteration occurred by the filteration of aqueous into the subconjuctival space.trabeculectomy can be combined with catract extraction with excellent results.indications are all types of

Trabeculectomy Procedure- Site is superonasal making a fornix based counjuctival flap. Episcleral flap is lightly cauterized to reduce bleeding by wetfield cautery. The scleral flap is usually one third to one half scleral thickness, rectangular (4mm by 6mm) or triangular(3mm by 3mm) in shape and dissected anteriorly towards the limbus. Antimetabolites are administered before making or after scleral flap is made but before anterior chamber is entered. A block of tissue is removed 1.5mm to 2.5mm in size with a descemets punch just anterior to the scleral spur. The block can be excised by vannas scissors. Peripheral iredectomy is done. Anterior chamber is formed through paracentesis site with ringer lactacte. Scleral flap is sutured with 10/0 nylon. Conjuctiva is sutured 10/0 nylon. **fig 2 a to d; photos figs 2 to 14.**

The Use Of Antimetabolites Like Mitomycin C And 5fu Provide Longer Controlled Lowering of Iop.MMC is more potent than 5fu. Concentrations of 0.2 to 0.50mg are used for 2 to 3 minutes. MMC soaked sponges are used for 2 minutes on intact scleral surface. The scleral bed is irrigated with 15 ml to 30ml of BSS solution to wash of MMC, before entering anterior chamber.



TRABECULECTOMY STEPS---FIG 2 A TO D

A. OUTLINE OF SUPERFICIAL SCLERAL FLAP

B. DISSECTION OF SUPERFICIAL SCLERAL FLAP.

C. EXCISION OF DEEP SCLERAL TISSUE WITH A PUNCH.

D. PERIPHERAL IRIDECTOMY

TRABECULECTOMY STEPS

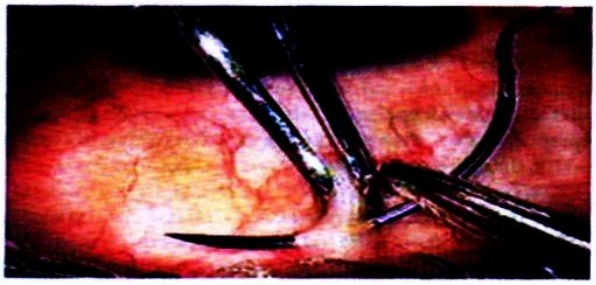


Fig-3 Superior rectus bridle suture

Fig-4 Limbus-based conjuctival flap

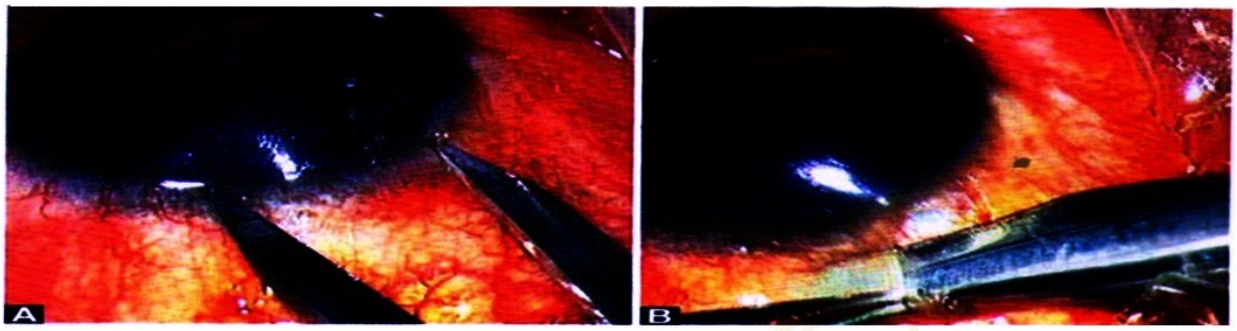


Fig-5A and B Construction of a fornix based conjunctival flap

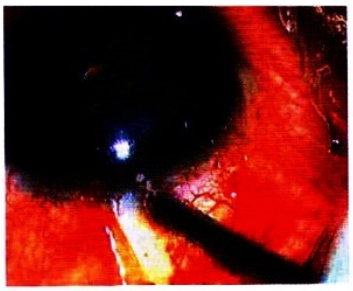


Fig-6

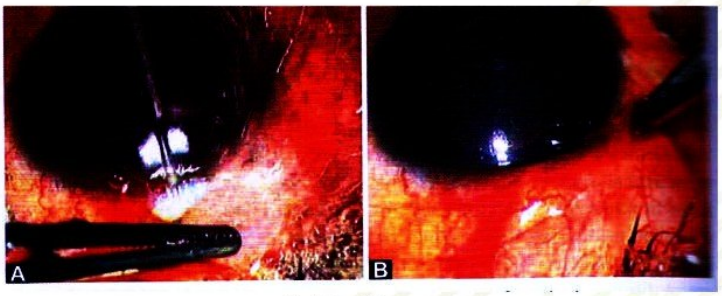


Fig-7A and B

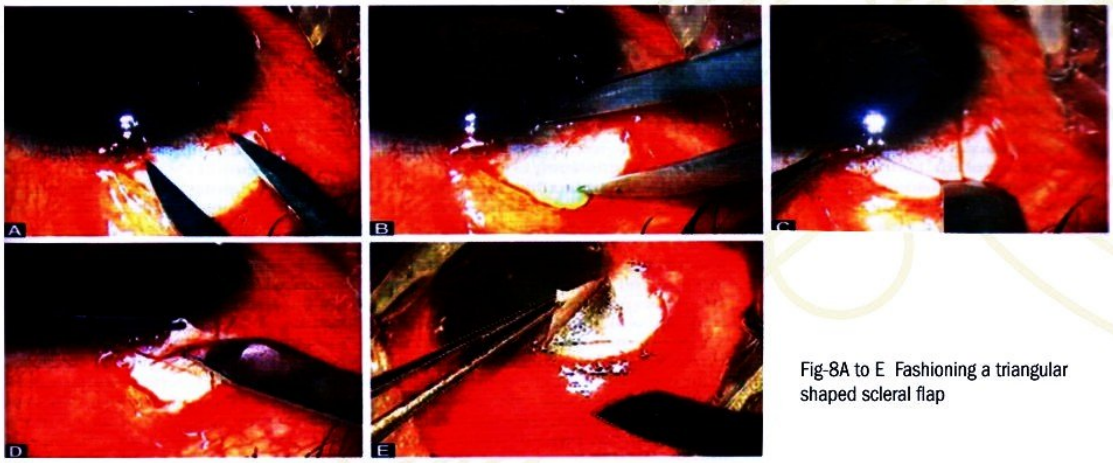
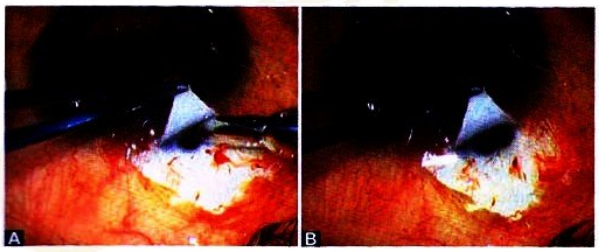


Fig-8A to E Fashioning a triangular shaped scleral flap



Figs 9A and B The trabeculectomy ostium is fashioned with a Kellys descemet punch

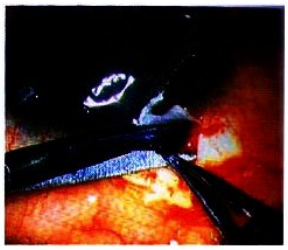


Fig. 10 Peripheral iridectomy

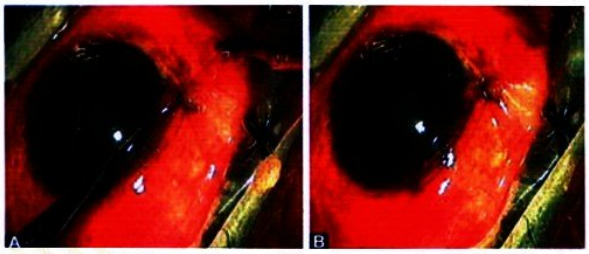


Fig. 11 A andB The conjunctval flap is sutured with 8-0 Vicryl

Fig. 12 Anterior chamber is reformed with BSS through the paracentesis

GLAUCOMA DRAINAGE DEVICES—setons are solid stents or wicks using the principle of surface tension flow procedure.the valved devices available are krupin or ahmed valves,ex-press implant.for fluid to exit the anterior chamber while structually maintaining a patent drainage device.the devices available are molteno implant,schoket

INDICATIONS - uncontrolled glaucoma despite previous trabeculectomy with adjunctive antimetabolite therapy. secondary glaucomas like rubeotic glaucomas and traumatic glaucomas.eyes with severe conjuctival scarring preculding accurate conjuctival scarring.certain congenital glaucomas where goniotomy,trabeculectomy,trabeculotomy have failed.

RECENT ADVANCES-

The XEN Gel Stent is made of a permanent, soft, collagen-derived, gelatin. Upon implantation, it creates a gentle, diffuse outflow of aqueous from the anterior chamber into the non-dissected tissue of the subconjunctival space. The AqueSysgelatin is well accepted by the human body, and is non-inflammatory. The pliability and softness allows it to conform to the ocular tissue, which may be shown to minimize many of the issues seen with synthetic materials (e.g., migration, erosion, corneal endothelial damage).

Potential benefits of the XEN Gel Stent include:

- Significant and sustained reduction of IOP through the globally accepted and well-studied subconjunctival outflow pathway
- Minimally invasive subconjunctival approach bypasses all potential aqueous outflow obstructions
- Soft gelatin material minimizes complications related to synthetic materials
- Significant ocular tissue available after the procedure, leaving all other treatment options available for the patient's future care

The AqueSys XEN Gel Stent has CE mark internationally, and is intended to reduce intraocular pressure in patients with primary open angle glaucoma where previous medical treatments have failed.

POSTOPERATIVE COMPLICATIONS AND MANAGEMENT CARE OF TRABECULECTOMY

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Peoples College of Medical Science and Research Centre

Trabeculectomy is a non physiological route of aqueous outflow from the anterior chamber to the sub tenon space by creating a fistula.

POSTOPERATIVE COMPLICATION AND MANAGEMENT

Early postoperative complications -

1. Hypotony and flat / shallow anterior chamber Occur on 2nd or 3rd postoperative day.

Cause-

- Conjunctival wound leak.
- Excessive filtration/over infiltration.
- Serous choroidal detachment.
- The prolonged shallow AC may leads to decrease corneal endothelial cell count and peripheral anterior synechiae formation.

Conjunctival wound leak

- Disintegration of conjunctival flap.
- Thin bleb.
- Necrosis of epithelium due to excessive use of antifibrotic agent during intraoperative period.
  Seidel's test positive.

Management

- Management depends upon the size and position of the leak, the appearance of the bleb, depth of the AC
- Pressure patch over the lid in the area of fistula
- Therapeutic soft contact lens
- Scleral / Simmons shell temponade
- Tissue adhesive -Cynoacrylate, collagen shields
- Autologous blood injection
- Conjunctival autografts
- Surgical m/m if leakage is large and along the suture line.

Excessive filtration

- Scleral flap leakage due to insufficient resistant to outflow from scleral flap
- Descemet folds present in cornea

- Seidel test positive

Management

- Firm patch over the eye
- Topical Atropine
- Maintained the AC by injecting Balance Salt Solution(BSS), Sodium hyluronate, large air bubble, gas Sulfur hexafluoride

SEROUS CHOROIDAL DETACHMENTS

- Collection of fluid in suprachoroidal space
- Seidal test positive
- It reduces the aqueous production and increase uveoscleral outflow so decrease the IOP

Management:

- Conservative therapy because many time it resolve spontaneously
- Frequent use of topical steroids and cycloplegics with or without systemic steroids
- Surgical intervention in case of large non-resolving effusions
- Escape of straw-colored fluid is seen on the Suprachoroidal space, draining by the sclerotomies in the inferior quadrants and deepening the AC with BSS or viscoelastic.

2. HYPOTONY AND DEEP ANTERIOR CHAMBER

- IOP lower than the normal.
- Markedly reduced visual acuity
- Fundus examination -fine macular striae radiating from fovea, tortuous retinal vessels, ± disc swelling

Risk factors -

- Young age,
- Myopic
- Preoperative use of carbonic anhydrase inhibitors
- Intraoperative excessive use of anti metabolites 5-FU,MMC

Management -:

- Pressure patch
- Bandage contact lens
- Autologous blood injection + bleb compression sutures
- Subconjunctival platelet
- Nd-YAG laser
- Complications -Corneal blood staining, delayed hyphaema, loss of vision due to autologous blood injection
- -Subconjunctival bleeding due to high energy Nd-YAG laser

3. ↑ IOP AND FLAT ANTERIOR CHAMBER

By three mechanisms -:

- Pupillary block -can occur in psuedophakia and aphakia
- ↑se IOP and shallow AC
- Seidel test negative
- Management -: YAG laser

II. Malignant glaucoma (aqueous misdirection) is caused by posterior diversion and pooling of aqueous in the vitreous cavity.

- ↑se IOP and absent bleb
- Seidel test negative

Management -:

- Mydriatics and Cycloplegic
- Posterior sclerotomy with air injection OR anterior vitrectomy followed by lens extraction
- Nd -YAG laser in Aphakic and pseudophakic
- Posterior sclerotomy and air injection
- Anterior pars plana vitrectomy
- M/M of fellow eye prophylactic laser iridectomy in angle closer glaucoma

III. Suprachoroidal hemorrhages – Occurs within 1st few days of postoperative period. Suprachoroidal hemorrhage occurs because the anterior displacement of Ciliary process and iris due to the ciliary detachment and forward pressure of the vitreous.

Clinical features-

- Sever pain, ± nausea, diminission of vision
- ↑se IOP with shallow AC
- Direct ophthalmoscopic examination Loss of red reflex on fundus
- USG in eye Large choroidal detachment and blood present on Suprachoroidal space

Management -:

- Small hemorrhages are well responds to topical and systemic steroids
- Surgical drainage of large hemorrhages after 7- 14 days when clot get liquefied

4. ↑se IOP AND DEEP ANTERIOR CHAMBER

- ↑se IOP and deep anterior chamber due to inadequate filtration –
- Obstruction of the fistula by scar tissue
- Absent or poor functioning bleb

I. Encapsulated filtering bleb

II. Failing bleb

I. Encapsulated filtering bleb k/a tenon's capsule cysts- developed after 2 months of surgery. Confirmed by Gonioscopy

Most common-

- Males
- congenital and juvenile glaucoma
- prolonged use of topical antiglaucoma and steroid

C/F - Bleb are highly elevated, smooth domed bleb with large vessels

Management -:

- Digital pressure
- Needling -25- 30 gauge needle ballooned up the conjunctiva then passed into the bleb to puncture and incise the fibrous episcleral tissue

- Subconjunctival injection 5-FU

II. Failing bleb- due to obstruction of the fistula or poorly functioning bleb C/F-Bleb is highly vascular but no microcysts

Management -:

- Frequent use of topical corticosteroid
- Digital pressure of the lower lid for 15 seconds
- Argan Laser suture lysis / removed the releasable suture
- Tissue plasminogen activator (5-FU)injected into AC

5. UVEITIS AND HYPHEMA –Iritis and fibrin present in AC

- Topical steroid and mydriatic- cycloplegic
- Propped up position and limited activity

6. LOSS OF CENTRAL VISION / SNUFF OUT SYNDROME

Risk factors are-

- Old age
- Preoperative macular spliting in the visual field and severe hypotony

LATE POSTOPERATIVE COMPLICATION

1) FAILURE OF FILTRATION- occurred within months to year after initial successful surgery.

- Closure of the fistula because scaring and cyst formation which is characterized by inflammation, fibroblasts and deposition of new collagen

Management-:

- Digital pressure
- Argon laser photocoagulation to removed pigmented tissue
  Nd YAG laser to eliminate non pigmented tissue
- To revised bleb with incisional surgery + antimetabolites MMC or 5-FU

2) LEAKING BLEBS - Due to disintegration of conjunctiva overlying the sclerotomy.

Risk factors are -

- Excessive use of antimetabolites during surgery eg.MMC
- severe coughing
- Clinical features-
- Low IOP and avascular bleb
- Seidel test initially negative, later test is positive because formation of hole
- Shallow AC and choroidal detachment

Management

- Conservative treatment
- Soft contact lens application
- Pressure patch with Simmons shell / collagen shield
- Tissue adhesive and trichloroacetic acid
- Nd YAG laser
- Autologous tenon's, partial thickness of sclera and conjunctival patch graft
- Amniotic membrane transplant

C. BLEB RELATED INFECTIONS are blebitis and endophthalmitis.

Risk factors are-

- Conjunctivitis
- Upper respiratory infection
- High dose of MMC intraoperative
- Long term use of antibiotic
- Inferior or nasally located bleb
- Combined surgery
- Diabetic
- H. influenza, Streptococcus, Staphylococcus spp. are related to poor visual prognosis

Clinical features-

- Pain with diffused conjunctival injection
- Sudden visual loss
- 'Milky white' bleb ,± epithelial defect
- In AC hypopyon
- Vitritis
- Hypopyon, Endophthalmitis, Leak ,Pain--HELP syndrome are suggested to bleb related to ocular infection

BLEBITIS-Without involvement of vitreous

Clinical features-

- Mild discomfort and redness
- White bleb contain inflammatory material
- Red reflex present

Management-

- Topical fluoroquinolone or combination with antibiotics
- Cycloplegic
- Fortified aminoglycoside, vancomycin, cephalosporin
- Oral Co- amoxiclav500/125 mg t.i.d. and ciprofloxacin 750 mg b.d. for 5 days ; alternative azithromycin 500 mg

ENDOPHTHALMITIS-

Clinical features-

- Rapid worsening of vision, pain, redness, foreign body sensation
- Inflammation of anterior segment, lens, choroid and vitreous
- Uveitis with Hypopyon
- White milky bleb contain pus
- Red reflex absent
- Vitreous Biopsy report positive

Management:

- Confirm the diagnosis by Biopsy (aqueous and vitreous)
- High dose of Topical and systemic antibiotics
- Intravitreal injection antibiotics according to culture and sensitivity
- Vitrectomy

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