



Glaucoma

Introduction

- Glaucoma is an optic neuropathy associated with characteristic damage to the optic nerve head (cupping) and the visual field (nerve fibre bundle defects).
- It is a blinding disease where first the peripheral visual field becomes constricted, followed by loss of central visual acuity

- Glaucoma, if defined with either field or nerve criteria, has a prevalence of 5.6%
- If defined with both field and nerve criteria, it has a prevalence of 2.4%
- The appearance of the optic nerve head and visual fields are the major factors for a diagnosis of glaucoma

Risk Factors

- Age
- Ethnicity
- Family History
- Intraocular pressure (IOP) is the most important risk factor
- Trauma
- Eye surgery
- Drugs
- Refractive errors

Non-IOP dependent risk factors

- Systemic Vascular Dysregulation
 - Raynaud's, Prinzmetal Angina, Migraine
- Nocturnal Hypotension
- Sleep Apnea

Intraocular Pressure IOP

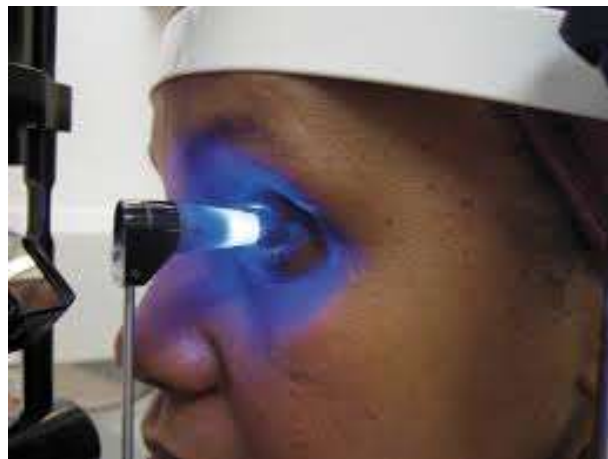
- An elevated eye pressure is neither necessary nor sufficient to make the diagnosis:
 - in “normal tension glaucoma”, the patient is never found to have a pressure over the normal limits
 - in “ocular hypertension” the patient has high eye pressures but no signs of optic nerve or visual field damage

- The level of the intraocular pressure is the main risk factor, and is important in the monitoring of treatment
- Gonioscopy is of major importance in the classification of the glaucoma type
- The extent of damage to the optic nerve and visual field determines the stage of the glaucoma

Basic Principles-IOP

- Intraocular pressure (IOP) represents the equilibrium between the rigidity of the cornea and sclera, and the outward pressure of the ocular contents
- As the vitreous is of fixed volume, the most important variable is the amount of aqueous humour, which varies with respect to production and drainage.
- The pathology of elevated intraocular pressure is due to inadequacies of aqueous outflow rather than production

- The normal mean IOP is 15.5 mm Hg
- Range is 10-21 mm Hg
- Diurnal variations exist
- Many ways to measure it but standard is Goldmann Applanation Tonometry -GAT



Basic Principles- Aqueous Humor

- The volume of the aqueous humour in the anterior segment is 0.25 cc or 250 μL
- One quarter of this is in the posterior chamber and three quarters in the anterior chamber
- The ciliary body produces 2.5 μL per minute, with complete turnover of the aqueous in about 100 minutes

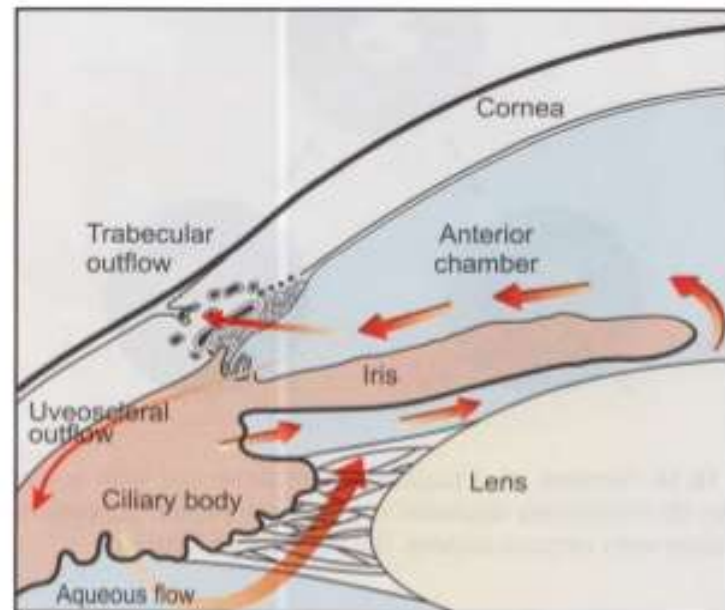
Aqueous Humor

- It is produced by the non pigmented epithelium of the ciliary processes
- Produced by :
 - Ultrafiltration
 - Active secretion

Aqueous Humor Pathway

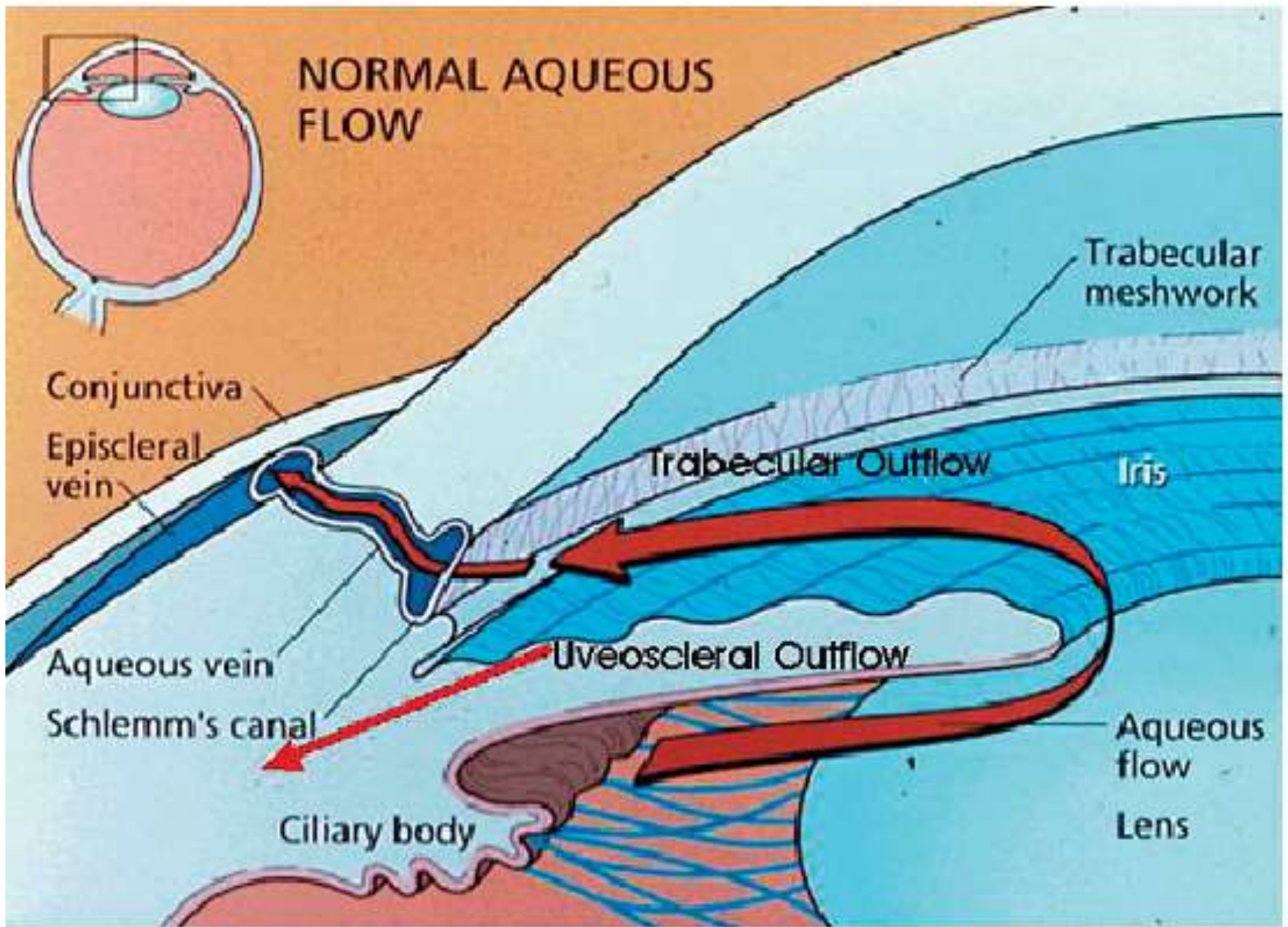
- Aqueous humor passes from the posterior chamber between the iris and the lens through the pupil into the anterior chamber

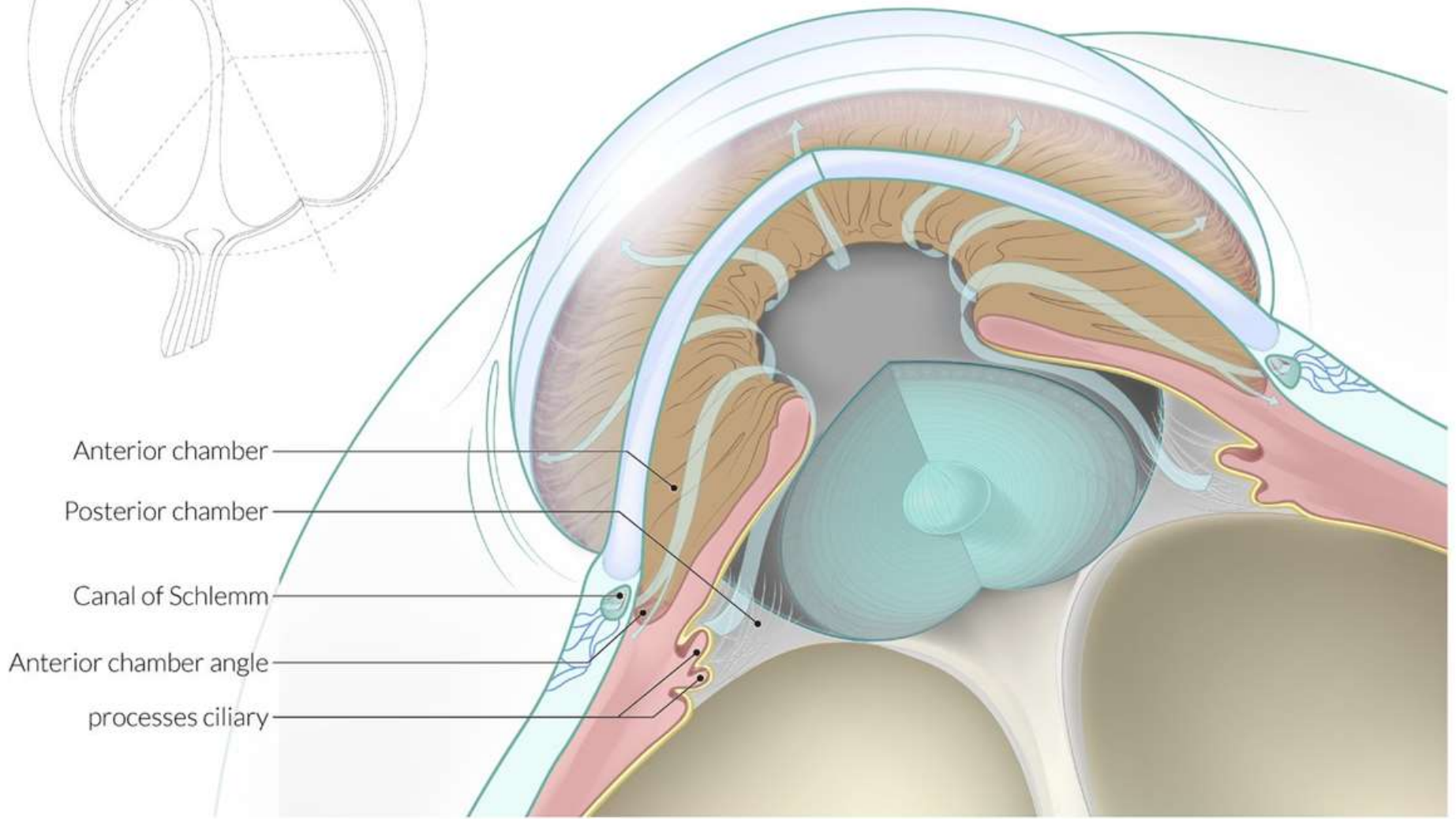
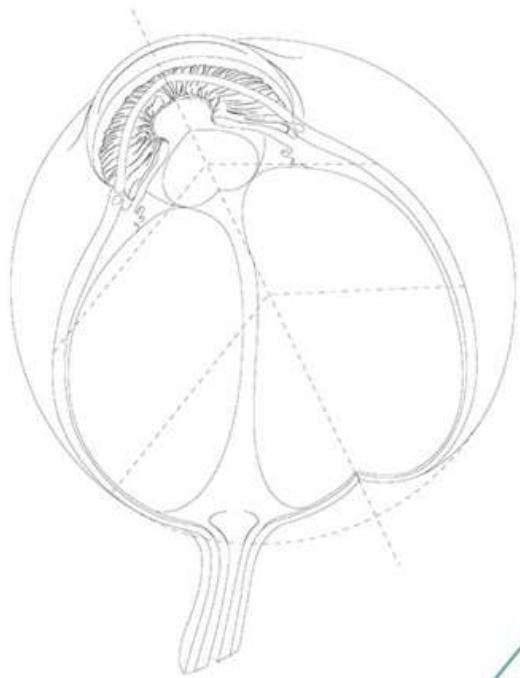
AQUEOUS HUMOUR DYNAMICS



Aqueous Humor Pathway

- It drains through 2 pathways:
 - Conventional (80-90%): trabecular meshwork, Canal of Schlemm, aqueous veins and episcleral veins
 - Uveoscleral (10-20%): Face of the ciliary body and iris to the supraciliary/suprachoroidal space.





Anterior chamber

Posterior chamber

Canal of Schlemm

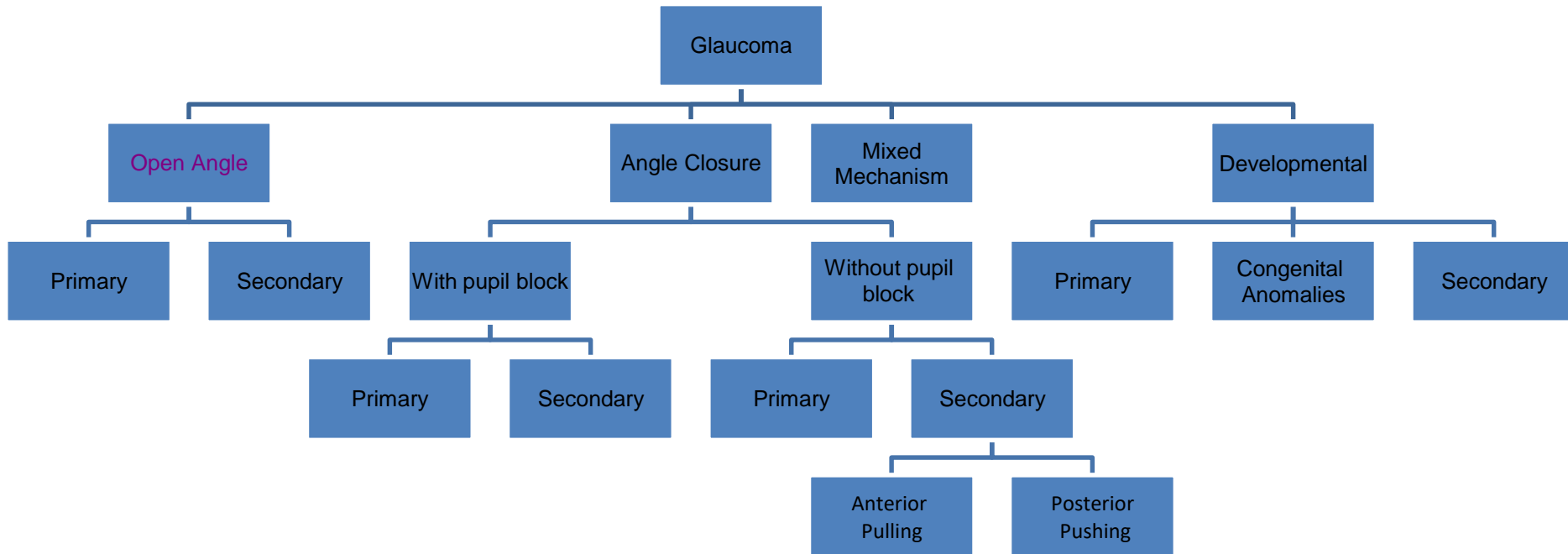
Anterior chamber angle

processes ciliary

Classification

- Glaucoma is not a single disease, but a large number of similar conditions with factors in common.
- It is usually classified on the basis of the anatomy of the anterior chamber angle as open or closed, and each type has primary and secondary sub-categories.

Glaucoma Classification



Open Angle Glaucoma

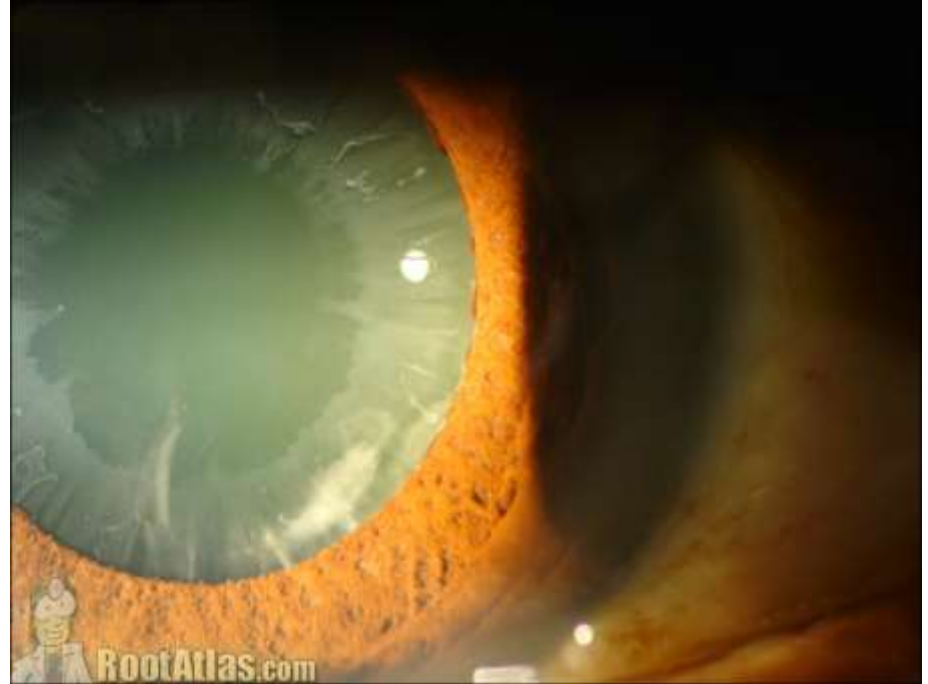
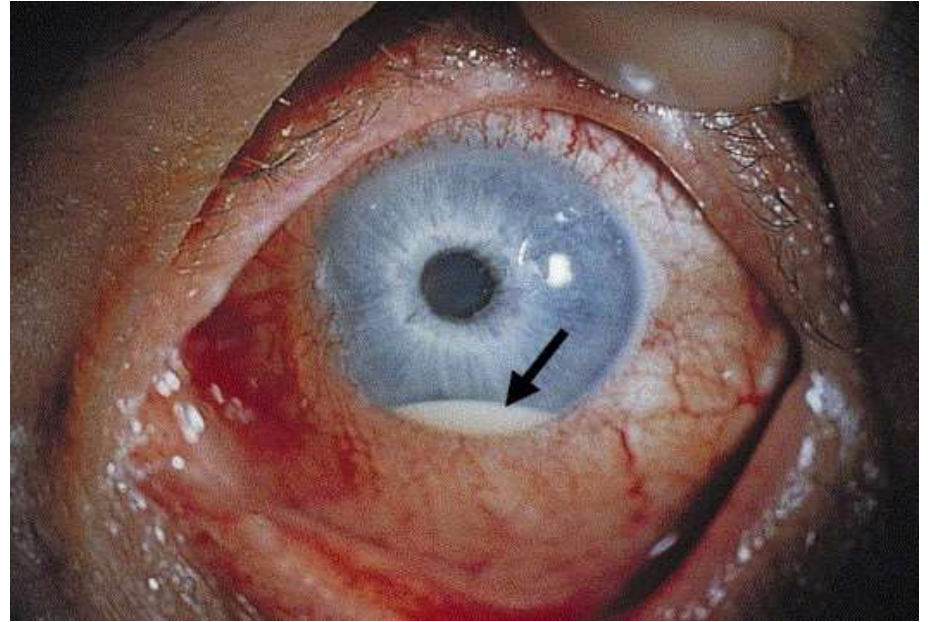
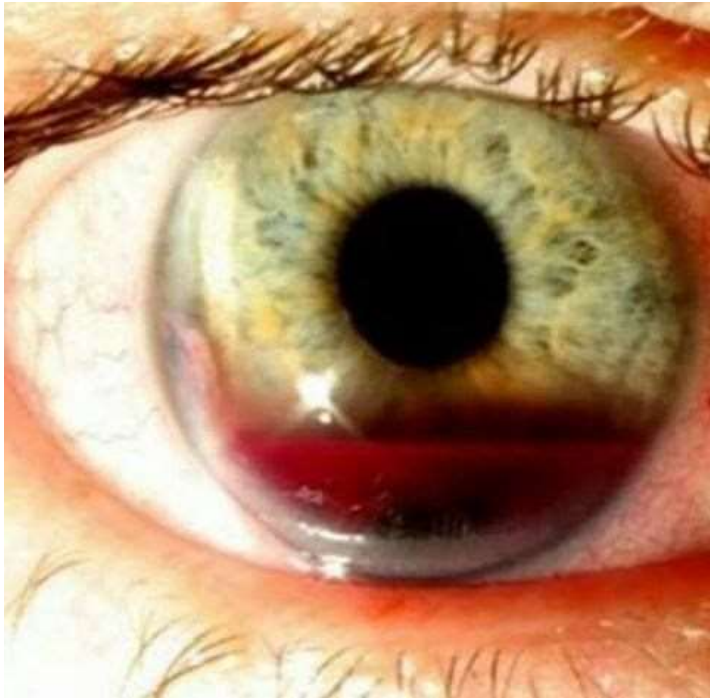
- Primary Open Angle Glaucoma POAG:
 - Idiopathic increase in outflow resistance
- Secondary Open Angle Glaucoma SOAG:
 - Clogging of trabecular meshwork TM
 - Increased episcleral venous pressure EVP
 - Scarring of TM
 - Increased TM resistance due to medications (steroids)

POAG

- Most prevalent type
- Female = male
- More common in myopes
- Asymptomatic till late in the disease
- IOP 20-40 mmHg

SOAG

- Clogging:
 - RBCs: Hyphema
 - WBCs: Uveitis
 - Pigment: pigment dispersion syndrome, melanoma
 - Proteins: Pseudoexfoliation syndrome
lens proteins



SOAG

- Increased EVP:
 - Carotid cavernous fistula
 - Sturg Weber Syndrome
 - SVC obstruction
- Scarring:
 - Angle recession (trauma)



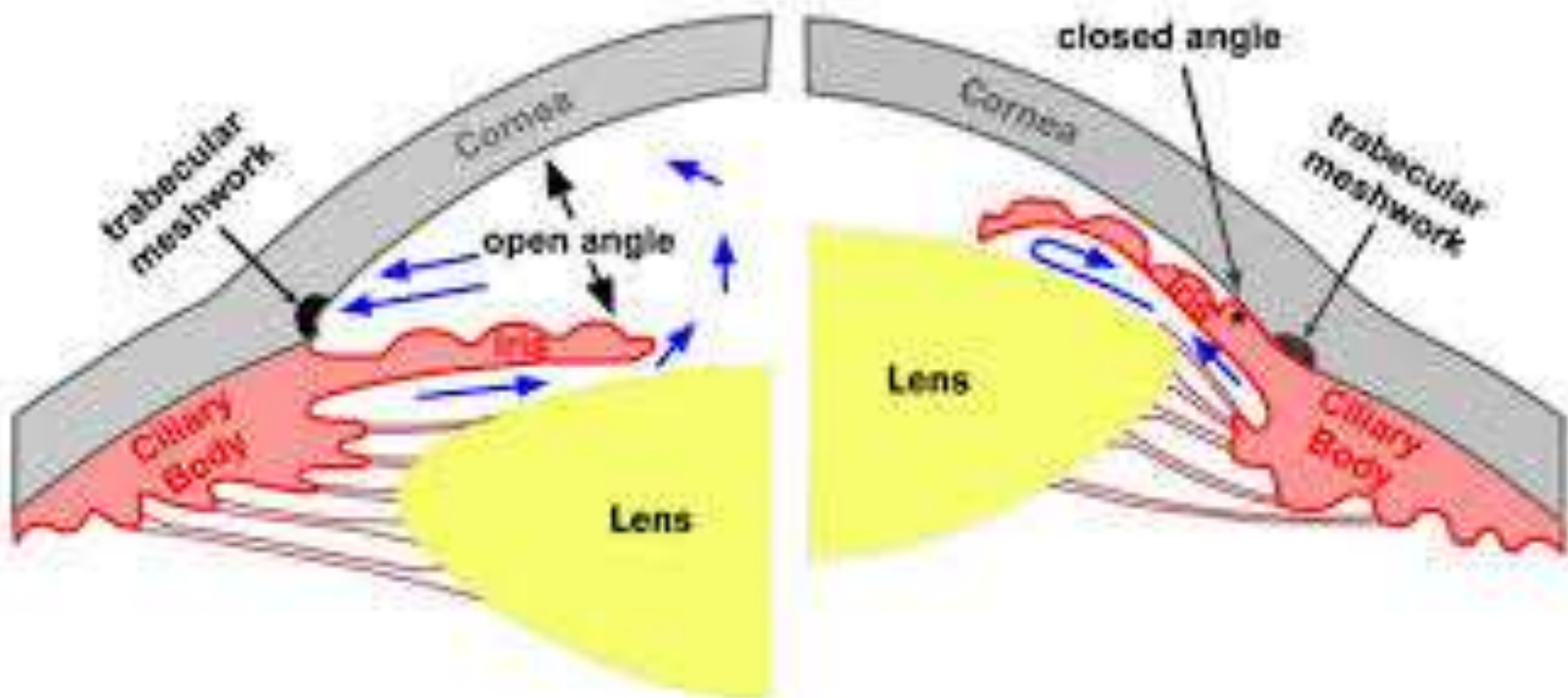
Closed Angle Glaucoma

- Anatomic features predisposing to angle closure: shallow anterior chamber (e.g., hyperopia, short eye)
- Advanced age (>60 years).
- Female gender
- Inuit and Asian ethnicity
- Eye injury with scarring and adhesions
- Rubeosis iridis
- Drugs: Sulfonamides, TCA, MAOi, antihistamines
- Mydriasis
 - I. Drug-induced: mydriatics
 - II. Darkness
 - III. Stress/fear response

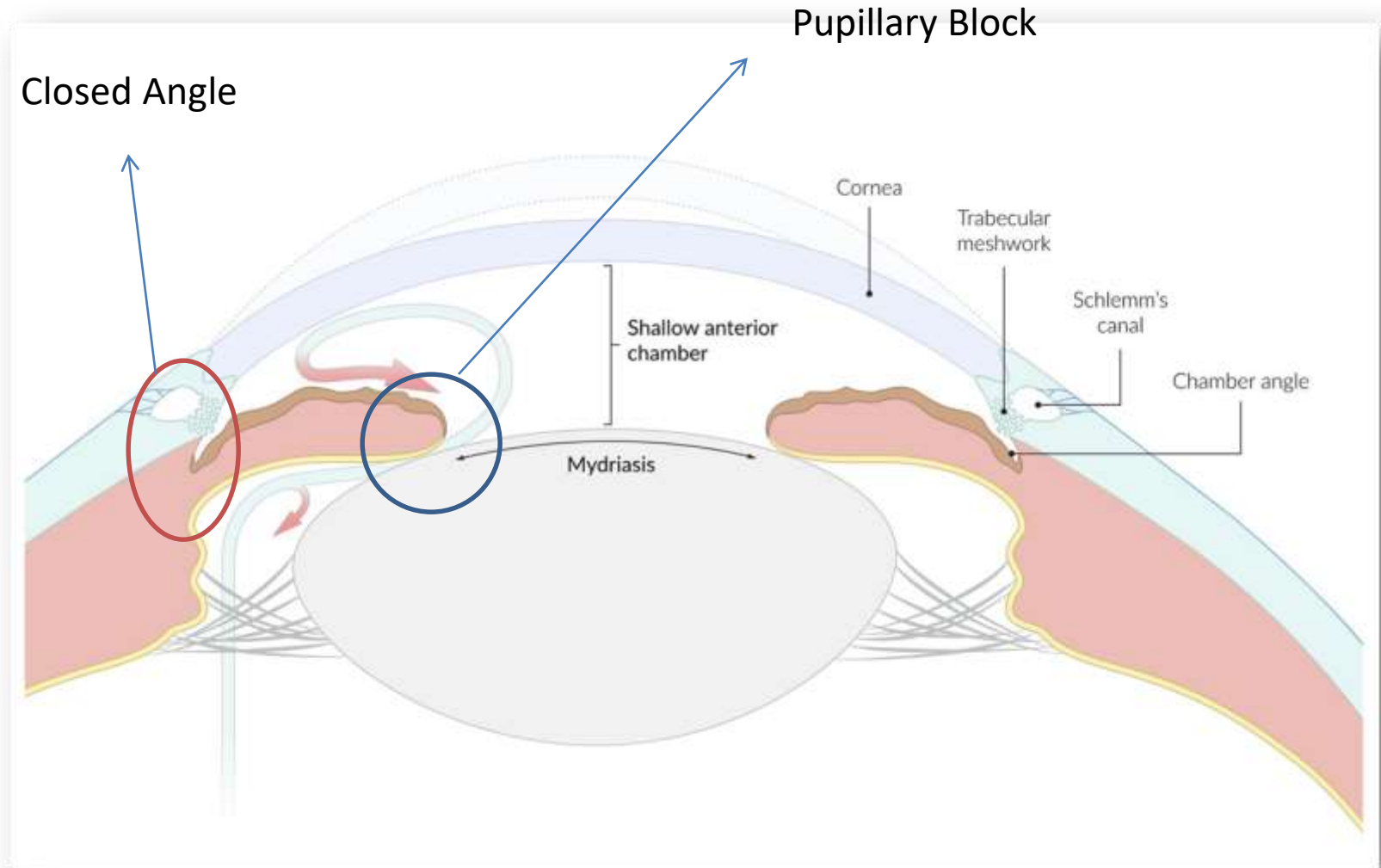
Pathophysiology

- Blocked trabecular meshwork → decreased drainage of aqueous humor from the eye → sudden ↑ in IOP
 - A. Primary : the chamber angle is narrowed due to the peripheral iris obstructing the TM.
 - B. Secondary :
 - A. Scarring: PAS or PS
 - B. Lens luxation/ large cataracts
 - C. Rubeosis iridis (neovascular glaucoma)

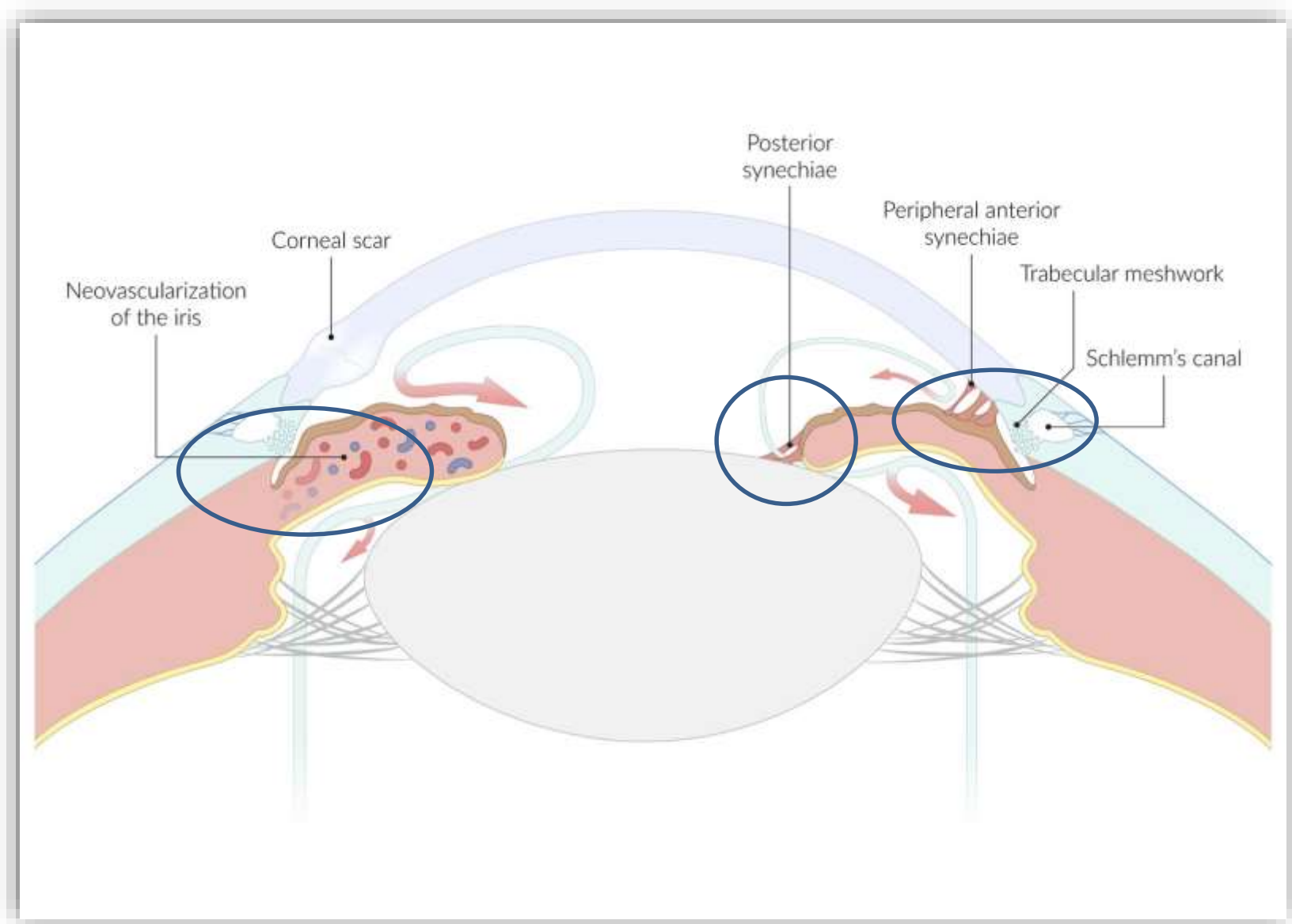
Open vs Closed Angle



Acute Angle Closure with pupillary Block



Chronic Angle Closure-NVG/ Uveitis



Clinical Presentation of Acute Angle Closure Attack

- Sudden onset of symptoms
- Severely painful eye (hard on palpation), redness
- Photophobia and excessive tearing
- Headache, nausea and vomiting
- Blurred vision and halos seen around lights
- Complications: irreversible damage of the optic nerve

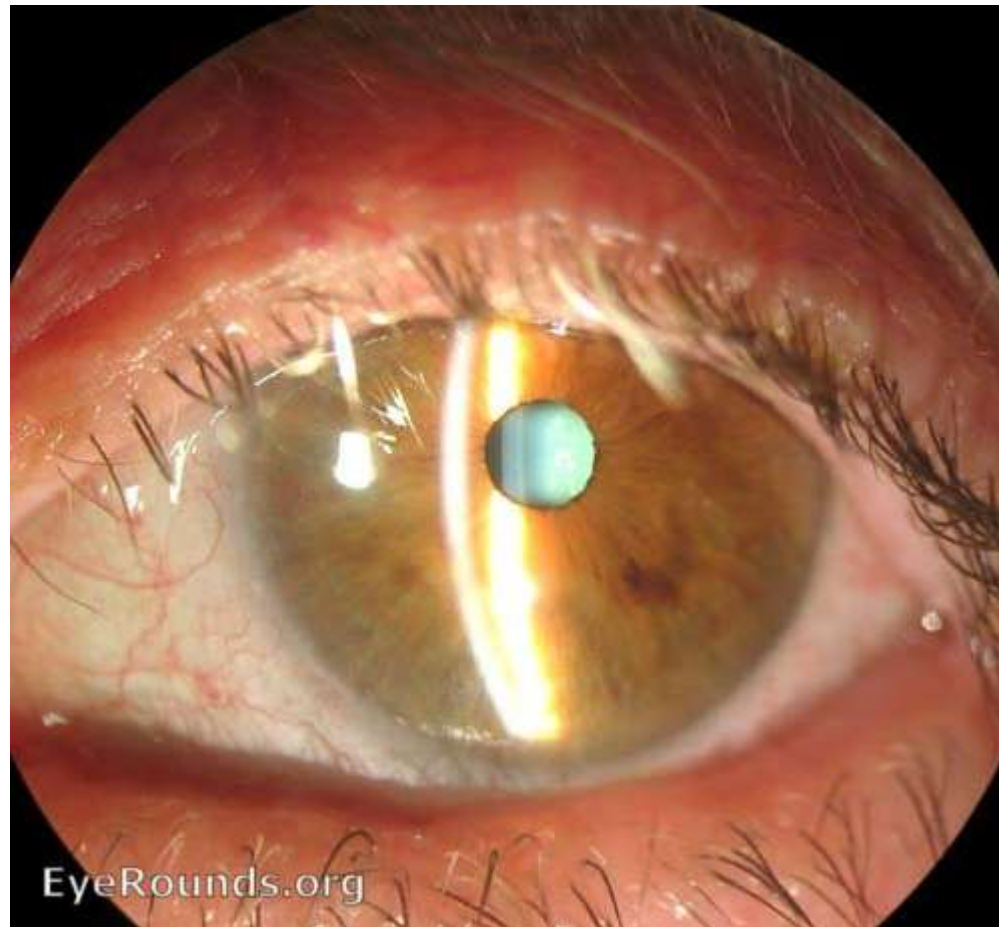
Diagnosis- Physical Examination

- Decreased visual acuity
- Non reactive, fixed oval pupil
- Cloudy edematous cornea
- Shallow anterior chamber
- Closed angle on gonioscopy
- IOP > 40 mm Hg

Injected, Cloudy cornea, oval pupil



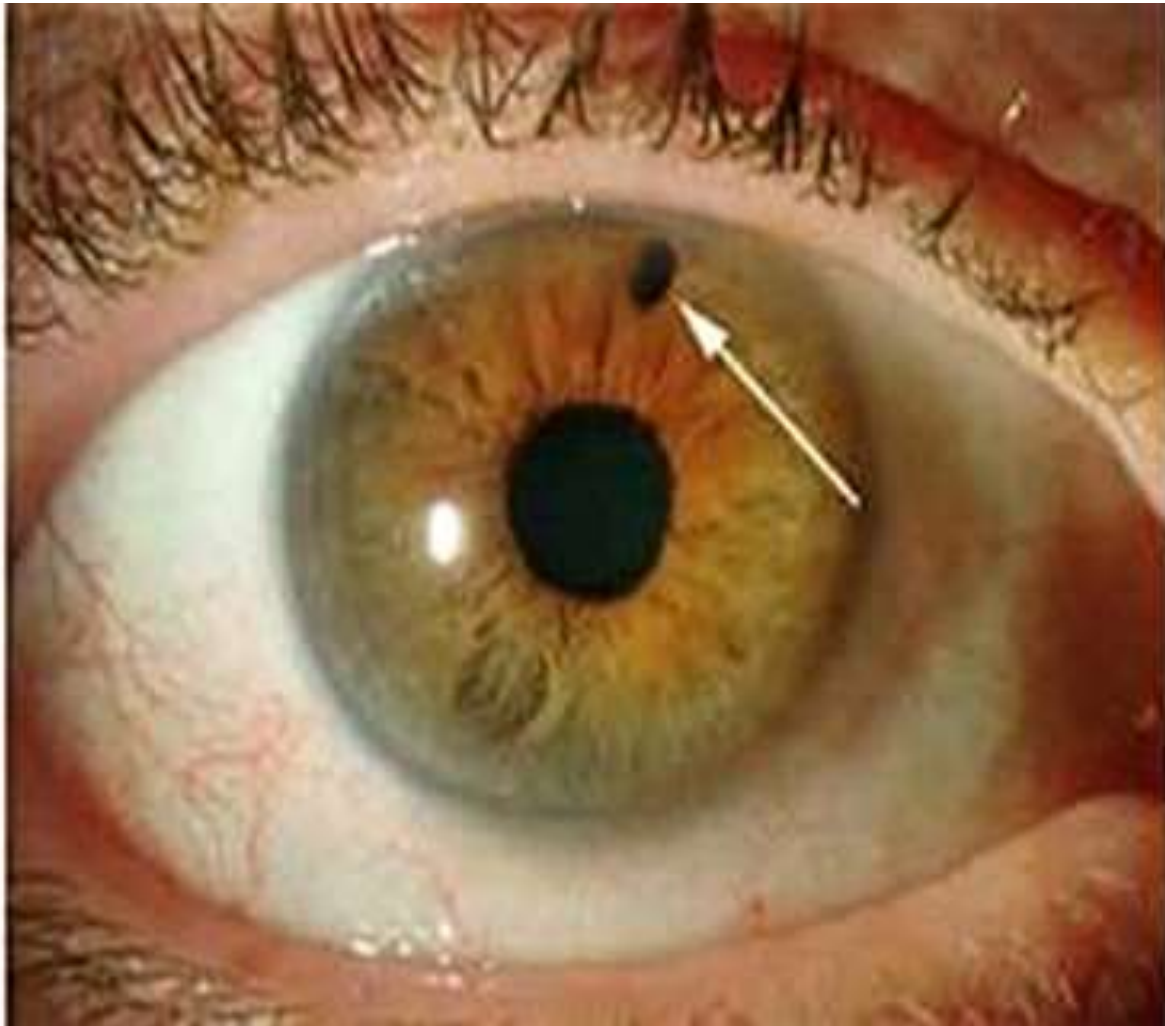
Shallow A/C



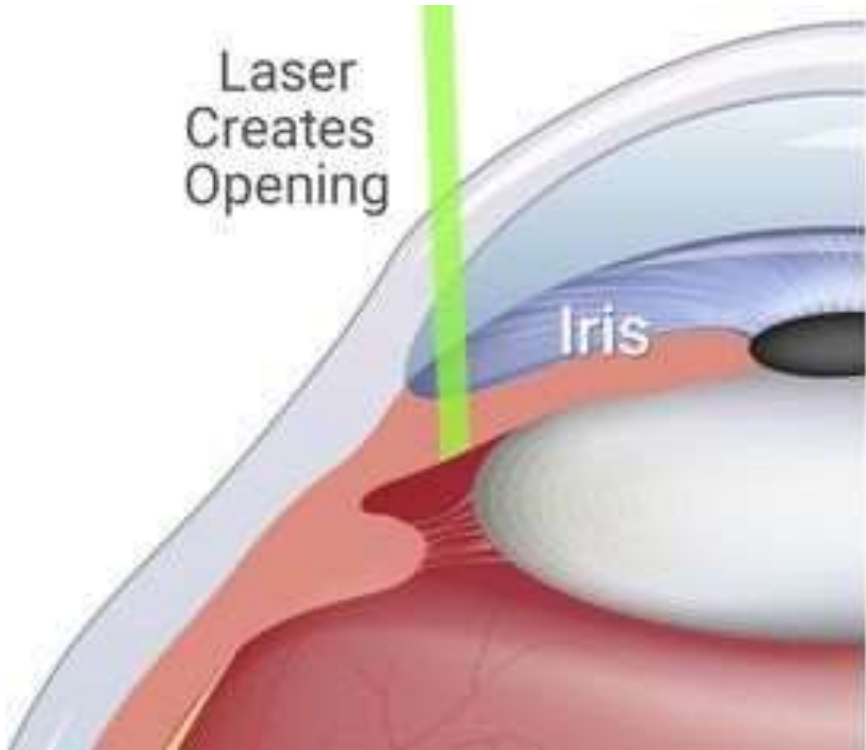
Management outline

- Lower IOP:
 - Systemically : IV Acetazolamide/ Mannitol
oral Acetazolamide
 - Topical Eye drops: B blockers, α agonists, Carbonic anhydrase inhibitors, pilocarpine
- Break the angle closure cycle:
 - YAG laser Iridotomy/ Surgical iridectomy
- Examine second eye and treat prophylactically

YAG Laser Iridotomy

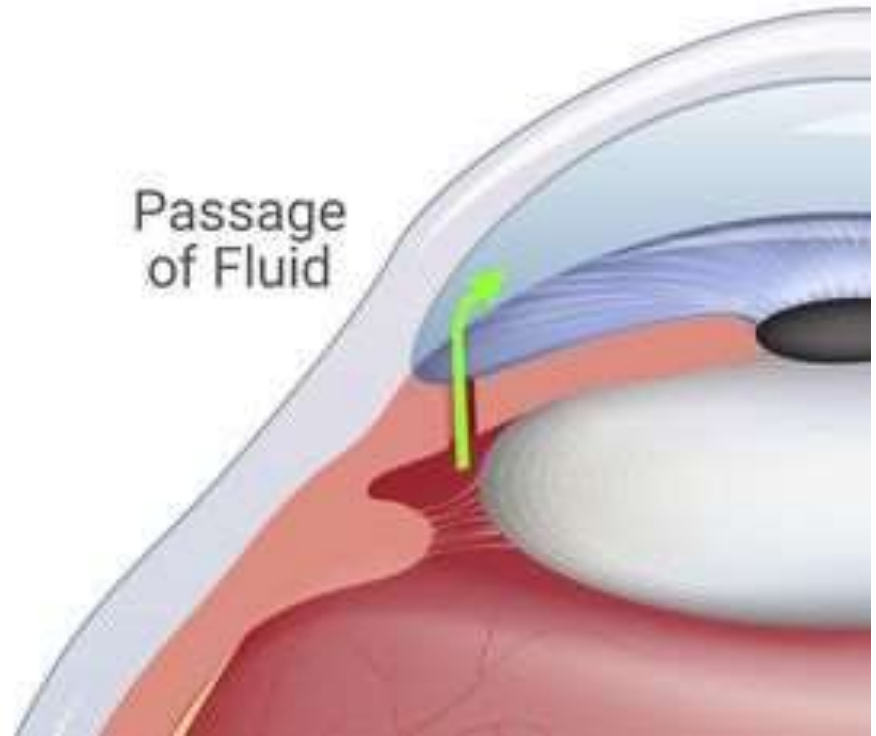


Laser
Creates
Opening



Iris

Passage
of Fluid

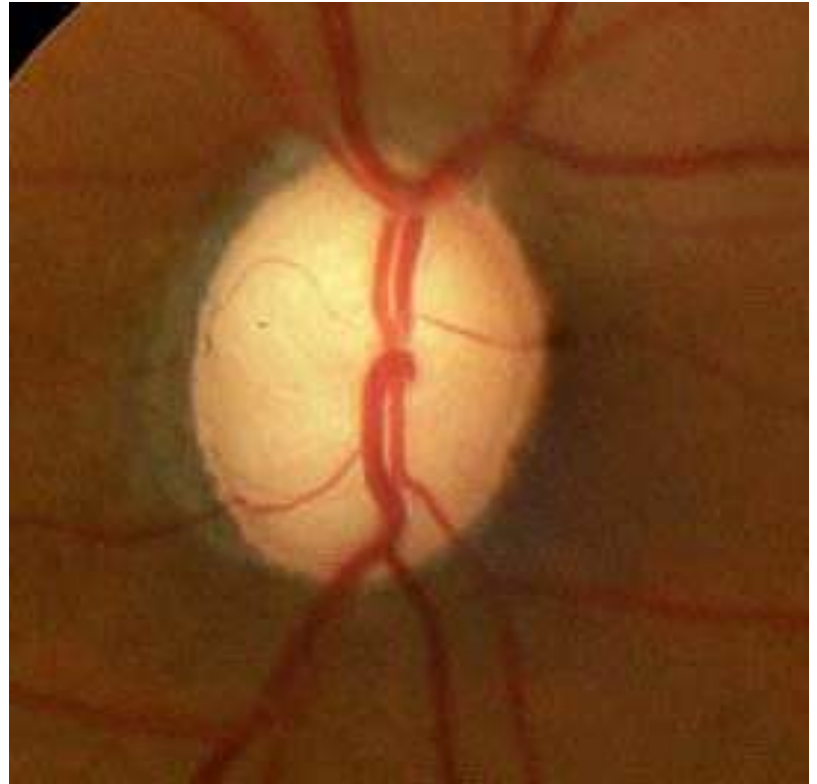


Glaucoma Diagnosis: Tips and Tools

- History
- Physical Examination
- Special Tests

Tip One

- Every patient has glaucoma until proven otherwise



What are glaucoma risk factors?

- History

- Family history

- Race

- Age

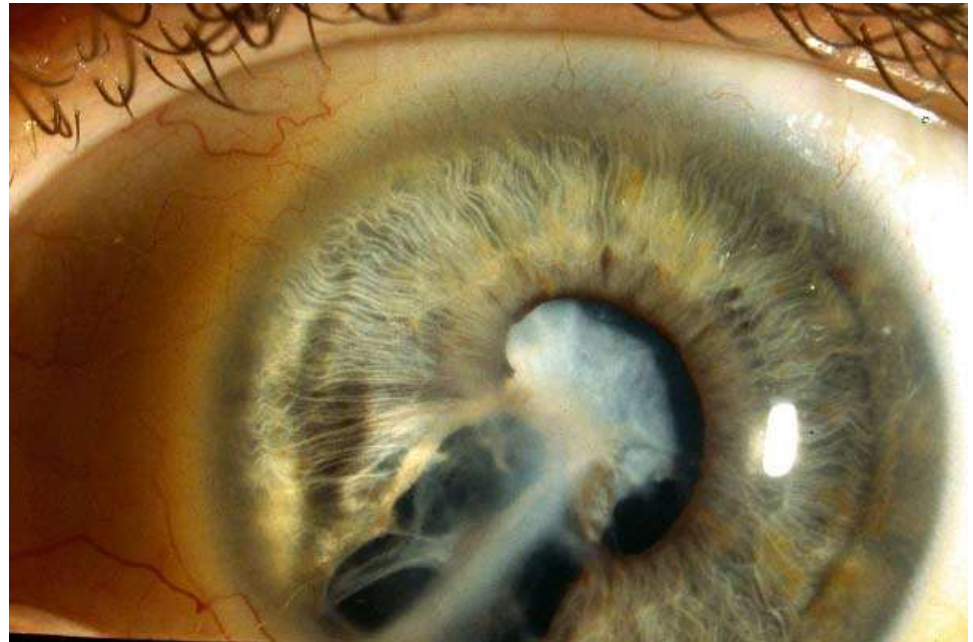
- Refractive Error

- Diabetes

- Vasospasm

Ocular History

- Trauma
- Laser
- Surgery
- Other

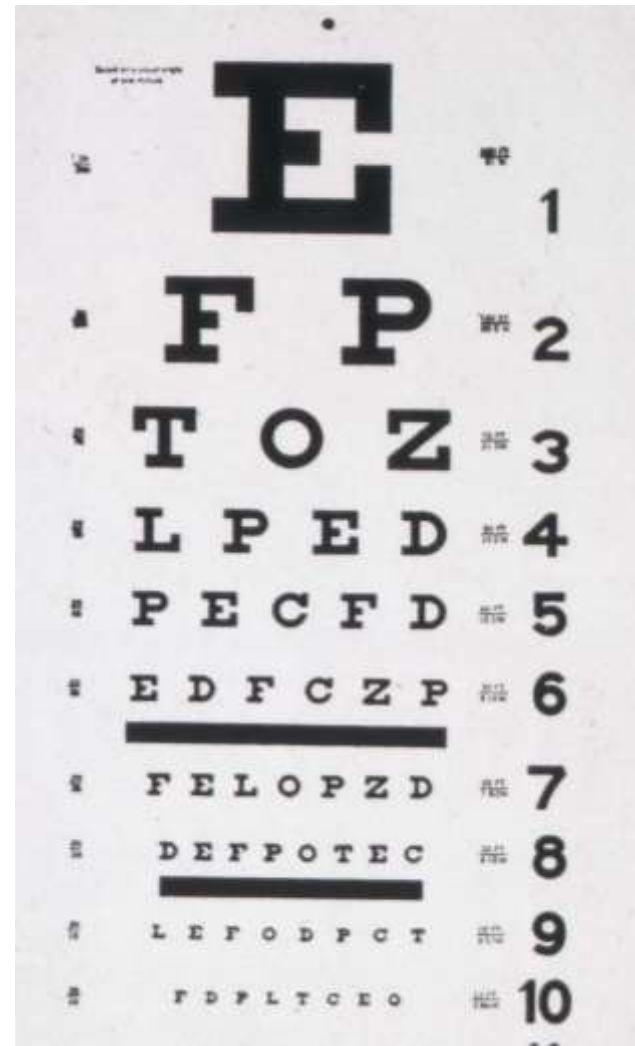


Medical History

- Diabetes
- Hypertension
- Asthma
- Other
- Known drug allergies/reaction

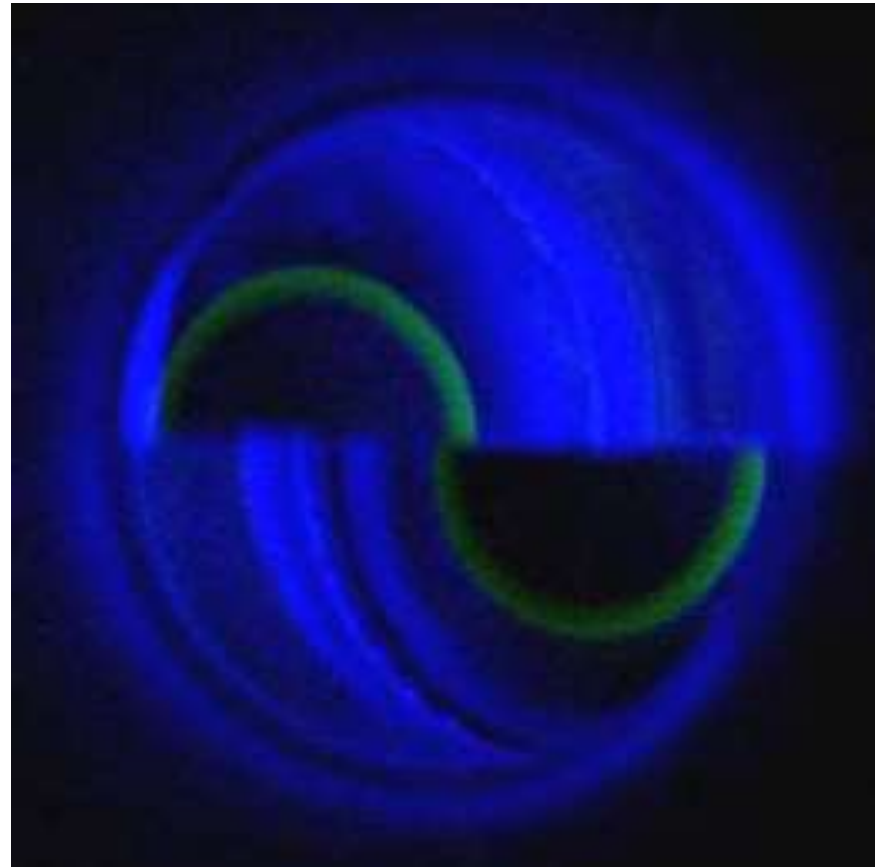
Vision

- BSCVA
- State refraction –
myope vs.
hyperope
- RAPD



Tip Two

- Intraocular pressure is neither necessary nor sufficient for the diagnosis of glaucoma
- Intraocular pressure is, however, the most important risk factor



IOP

- The higher the pressure the higher the risk
- Goldmann technique preferred
- Tonopen, etc. if necessary



What else is important?

- Slit lamp examination
 - Classify type based on angle structures
 - Look for signs of secondary glaucoma
- Optic nerve head examination
 - Stage disease based on ONH damage
- Visual field examination
 - Stage disease based on VF damage

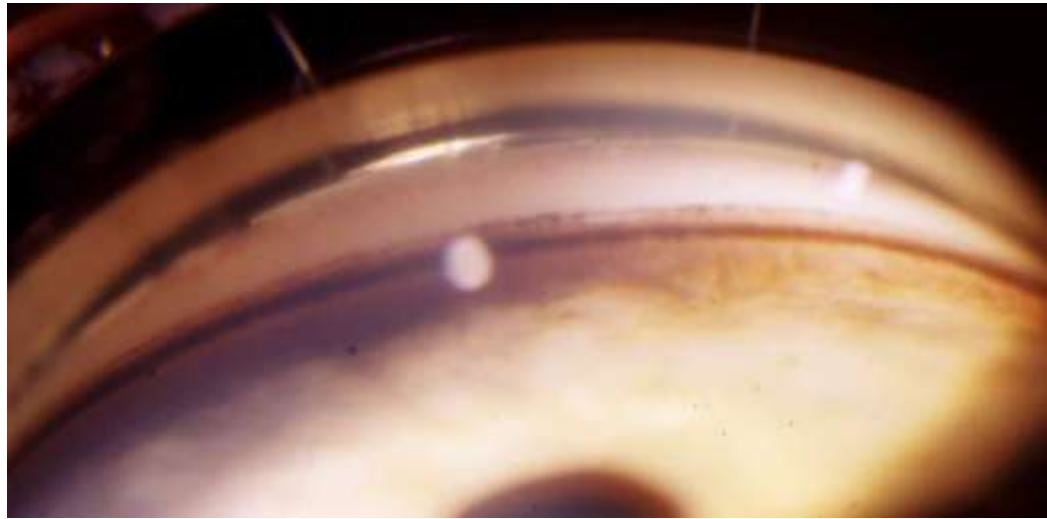
Slit lamp examination

- Lids/lacrimal
 - Conjunctiva/
 - sclera
 - Cornea
 - Lens
- Other
 - PXE
 - PDG
 - NVI
 - Iritis
 - PI

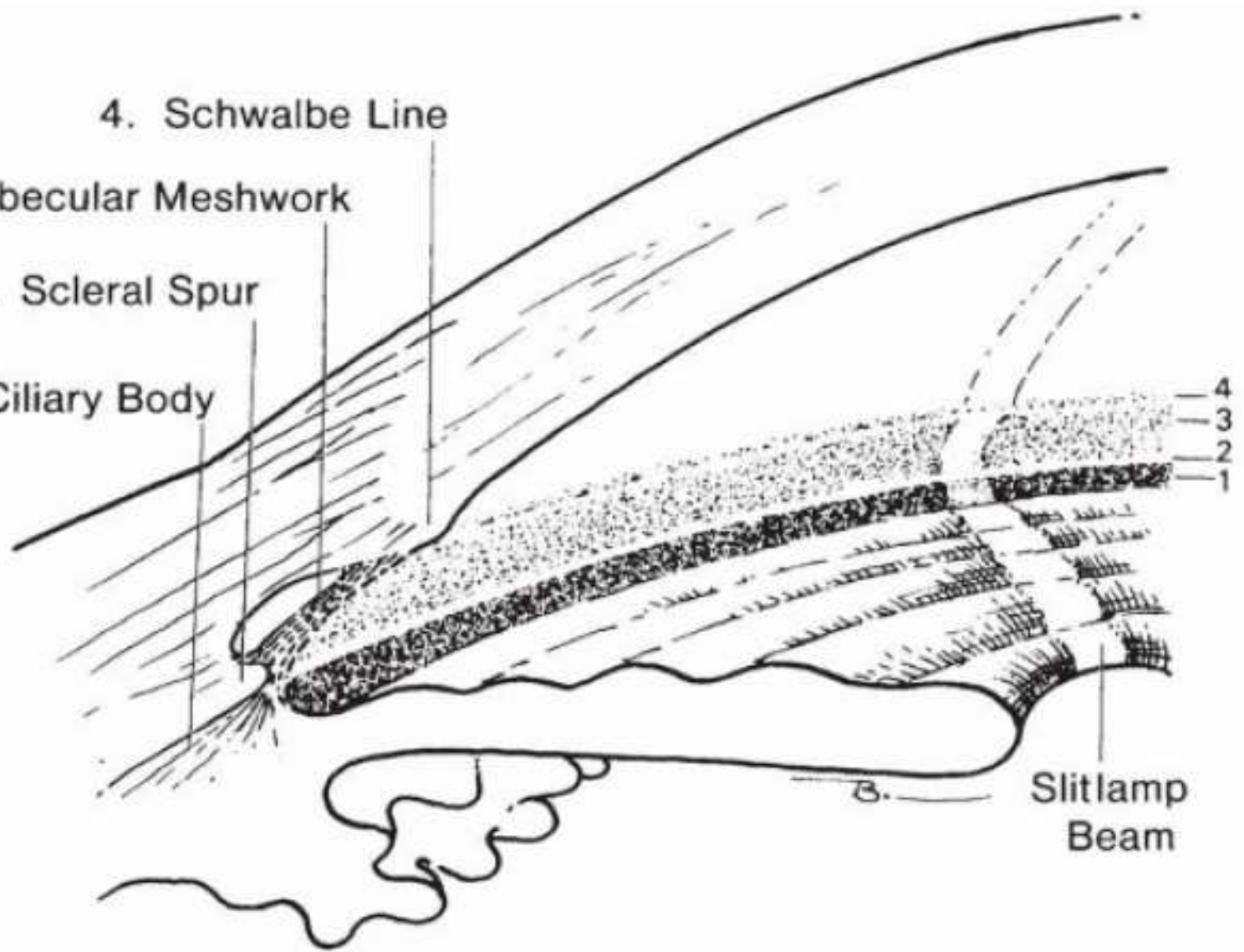


Gonioscopy

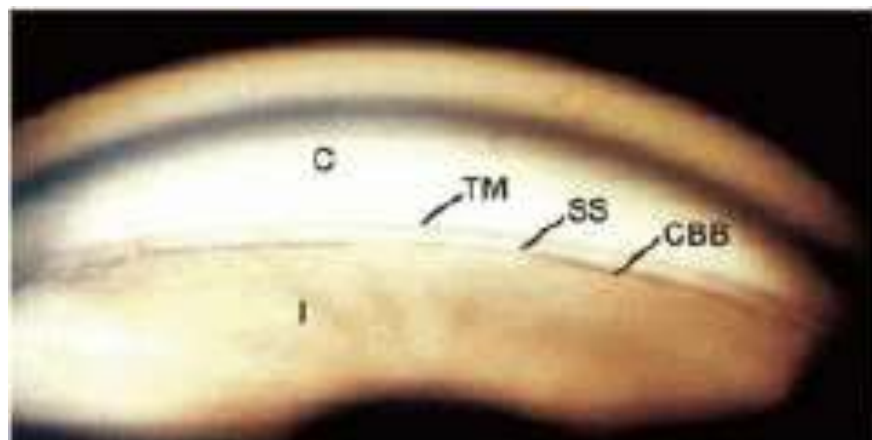
- Always performed on any patient where glaucoma is a possibility
- Classify into open vs. narrow vs. closed angle



- 4. Schwalbe Line
- 3. Trabecular Meshwork
- 2. Scleral Spur
- 1. Ciliary Body



Slitlamp
Beam



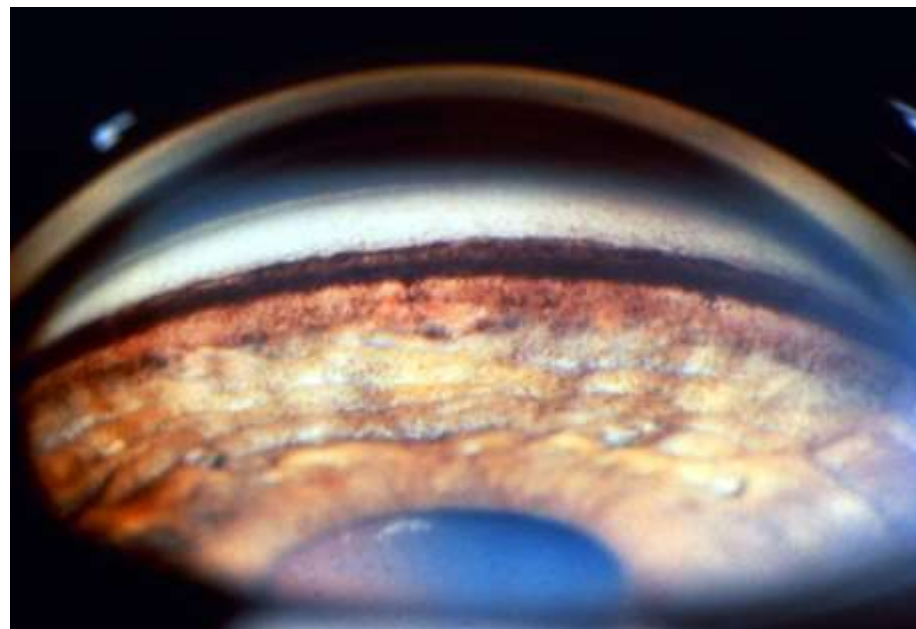
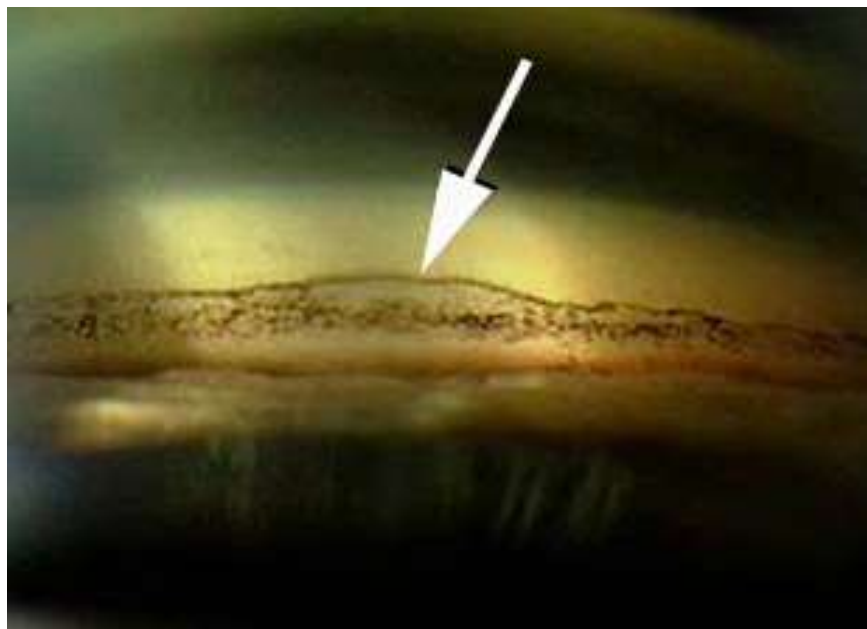
A



B



Gonioscopy- Look for secondaries



Tool One

- Four mirror lens excellent for compression gonioscopy
 - This differentiates between appositional and synechial closure
- Three mirror lens also fine



Gonioscopy lenses/mirrors



Tool Two

- Corneal thickness is becoming more and more important in glaucoma diagnosis
- Pachymetry is not, however, currently a part of the standard of care
- Prior LASIK will result in very thin central cornea

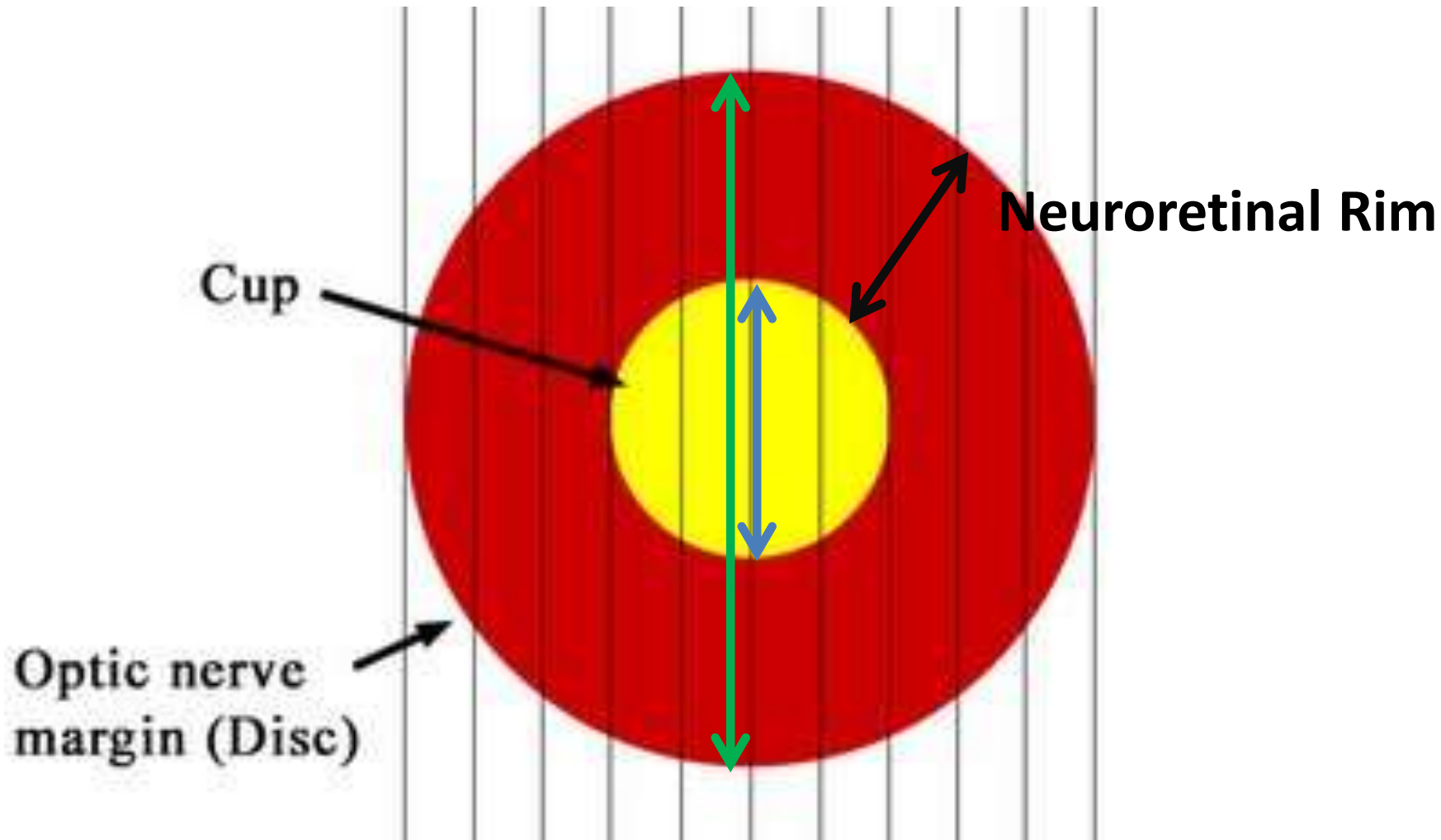


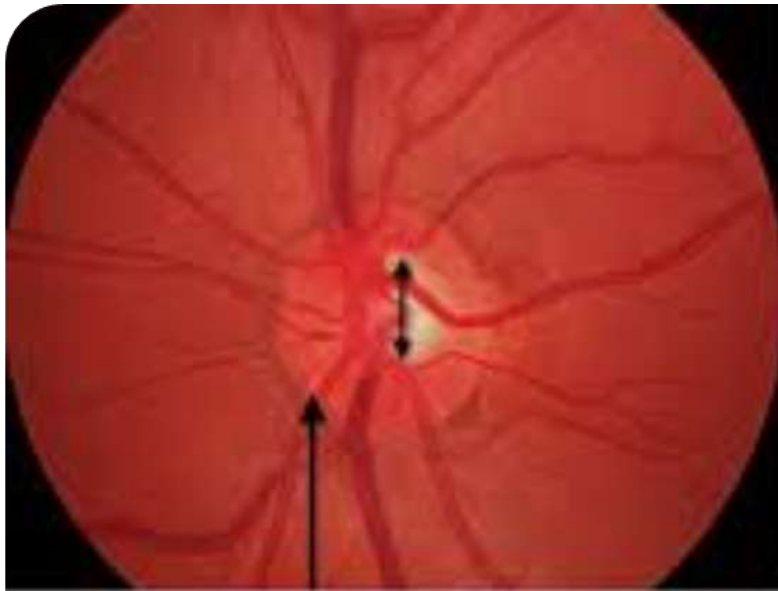
- Thin cornea can give a falsely low IOP reading
- Thick cornea can give falsely high IOP

Optic Nerve & Retina

- State C:D ratio
- Note other findings:
 - Thin rim
 - Notch
 - Drance hemorrhage
 - Peri papillary atrophy, α or β
- State relevant retinal findings:
 - AMD, etc

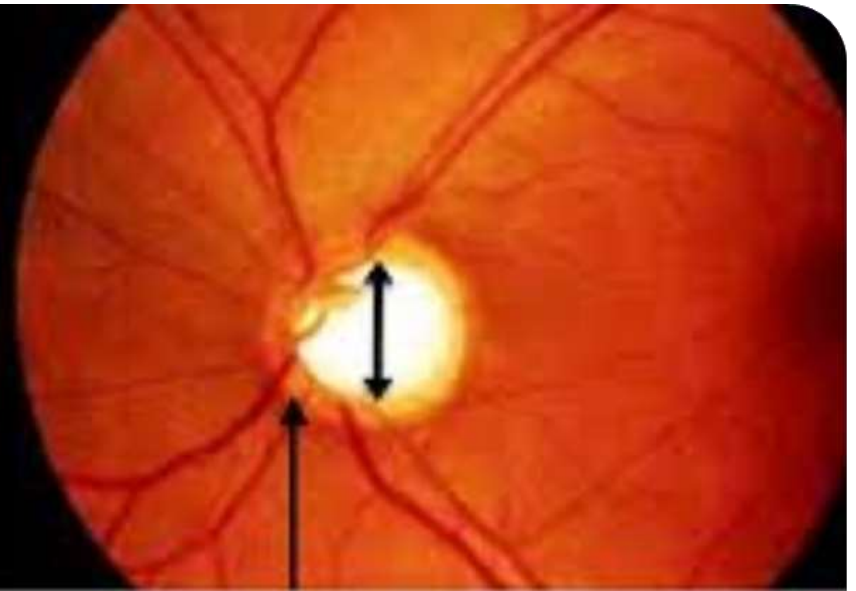
Vertical **Cup** to **Disc** Ratio





Normal optic nerve head

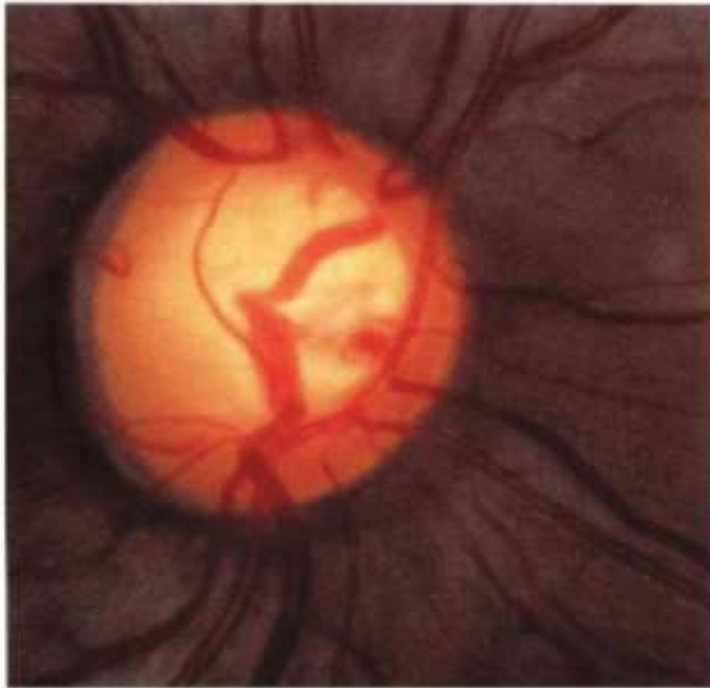
Normal optic nerve head



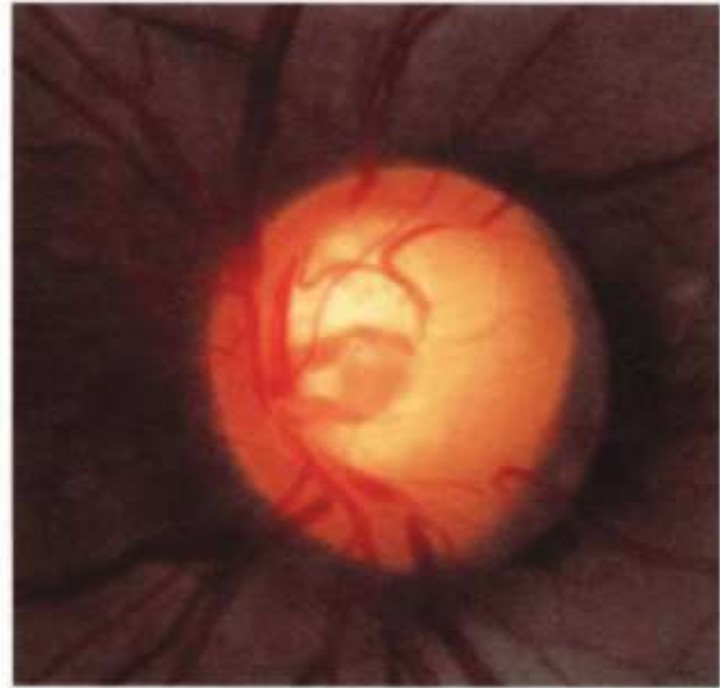
Glaucomatous cupping

Glaucomatous cupping

Variants of Normal

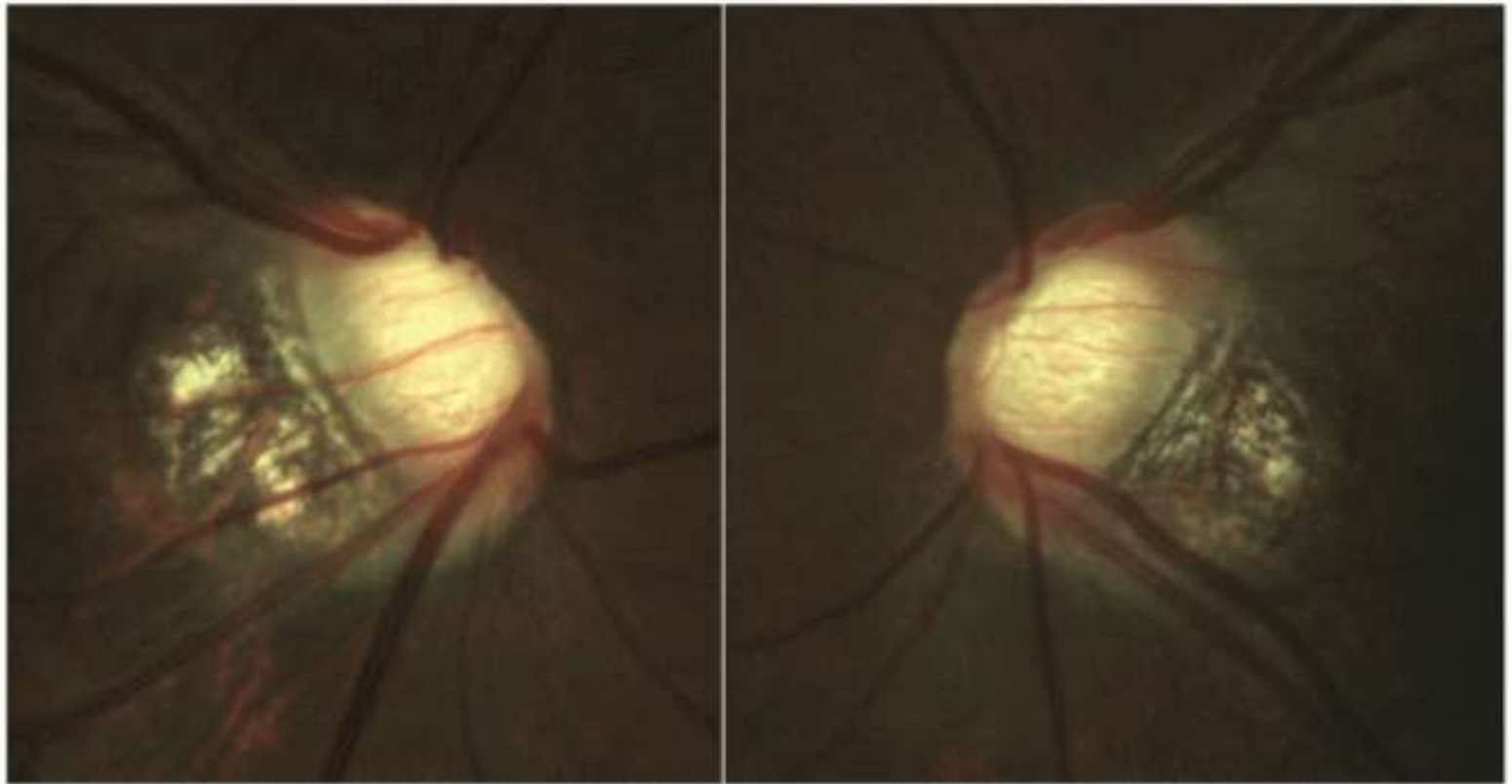


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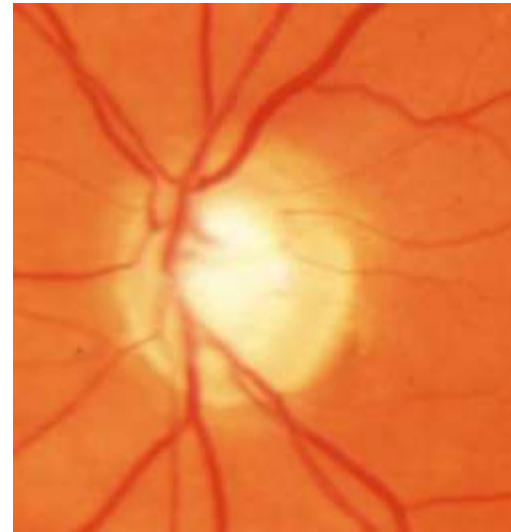
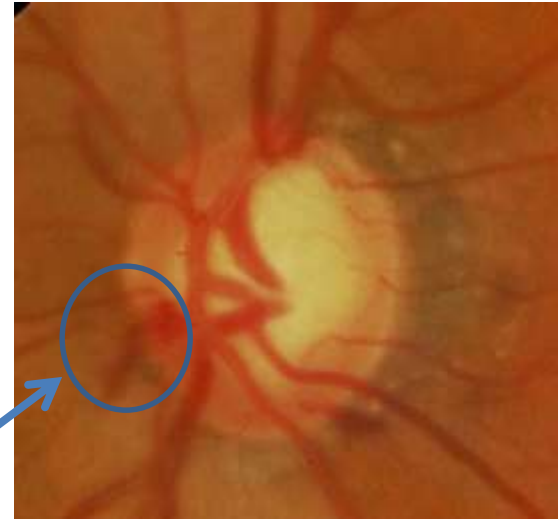
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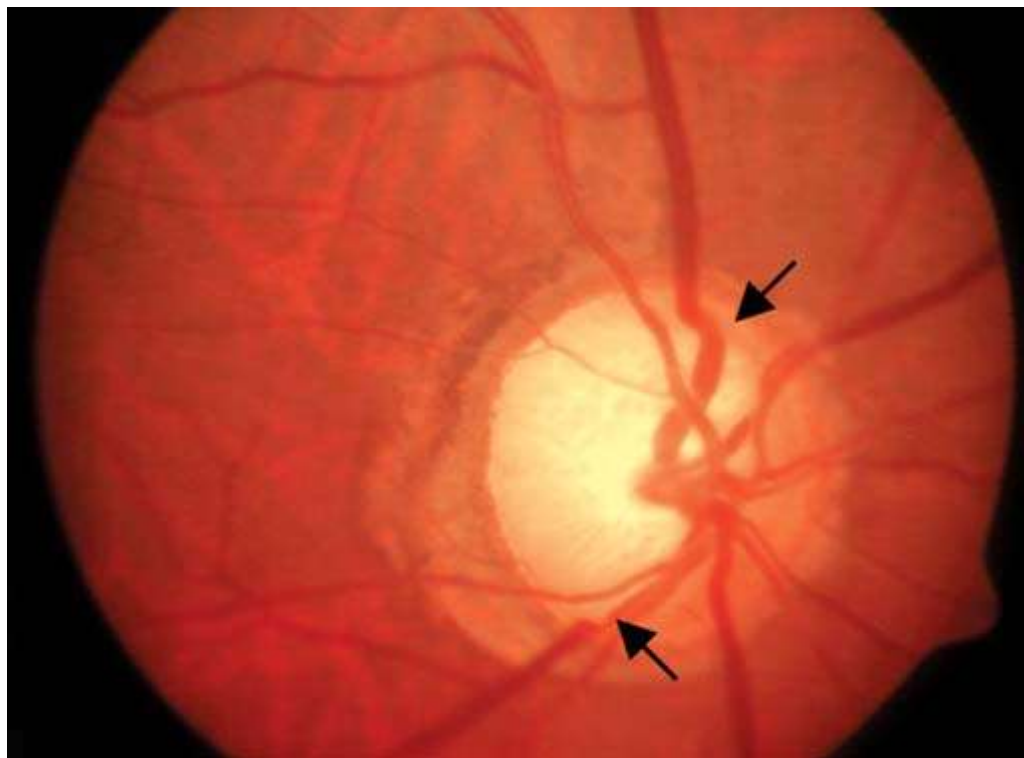
Challenging Nerves



Optic Nerve Head Examination

- Look for:
 - Cupping
 - Asymmetry
 - Notching
 - Hemorrhages
 - ISNT rule



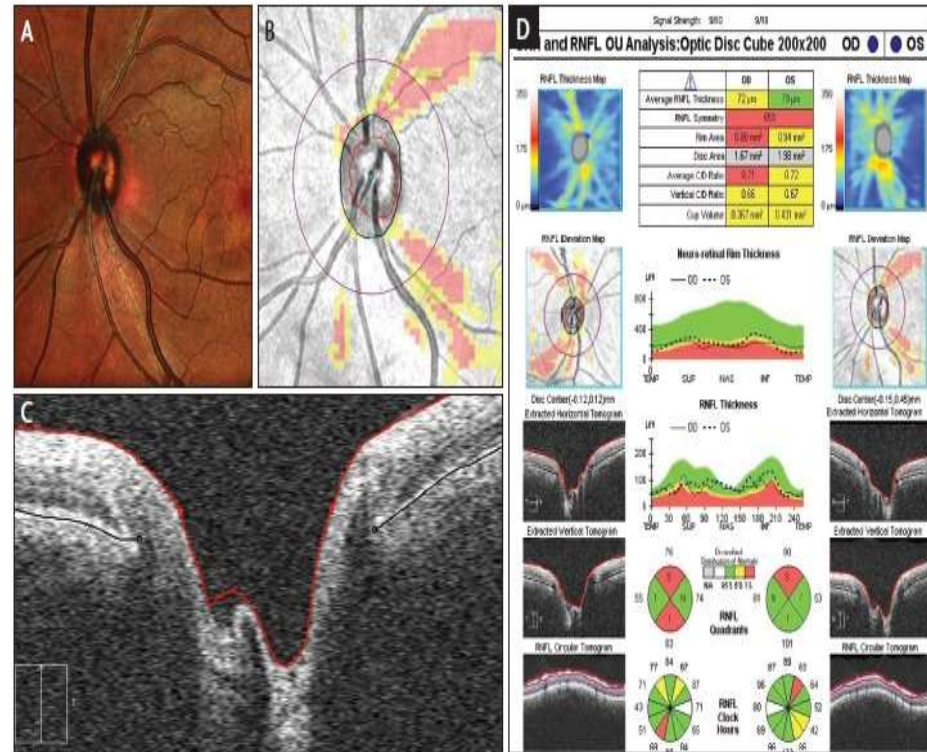


Tool Three

- Stereoscopic viewing at the slit lamp with a 66D or 78D lens and a dilated pupil

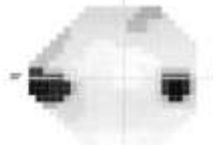


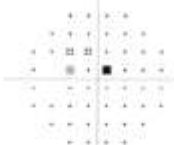

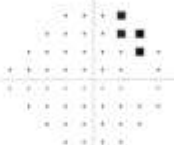
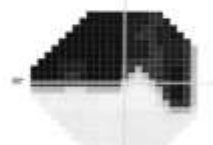
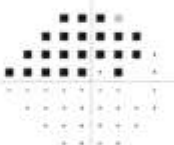

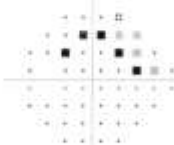

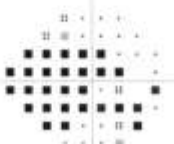
Tool Four – Optic Nerve Head Imaging

- Computer aided imaging of optic nerve and/or nerve fibre layer
 - Confocal scanning laser ophthalmoscopy / HRT
 - Optical coherence tomography/OCT
 - Polarimetry / GDx
- These are all commercially available – and costly!



Tool Five – Visual Field Examination

- Assess functional damage prior to patients perception of field loss
- Assess patient's performance in relation to age matched normal database (Statpack, SITA, Octopus)

Nasal Step		
Paracentral		
Temporal Wedge		
Altitudinal		
Arcuate		
Advanced		

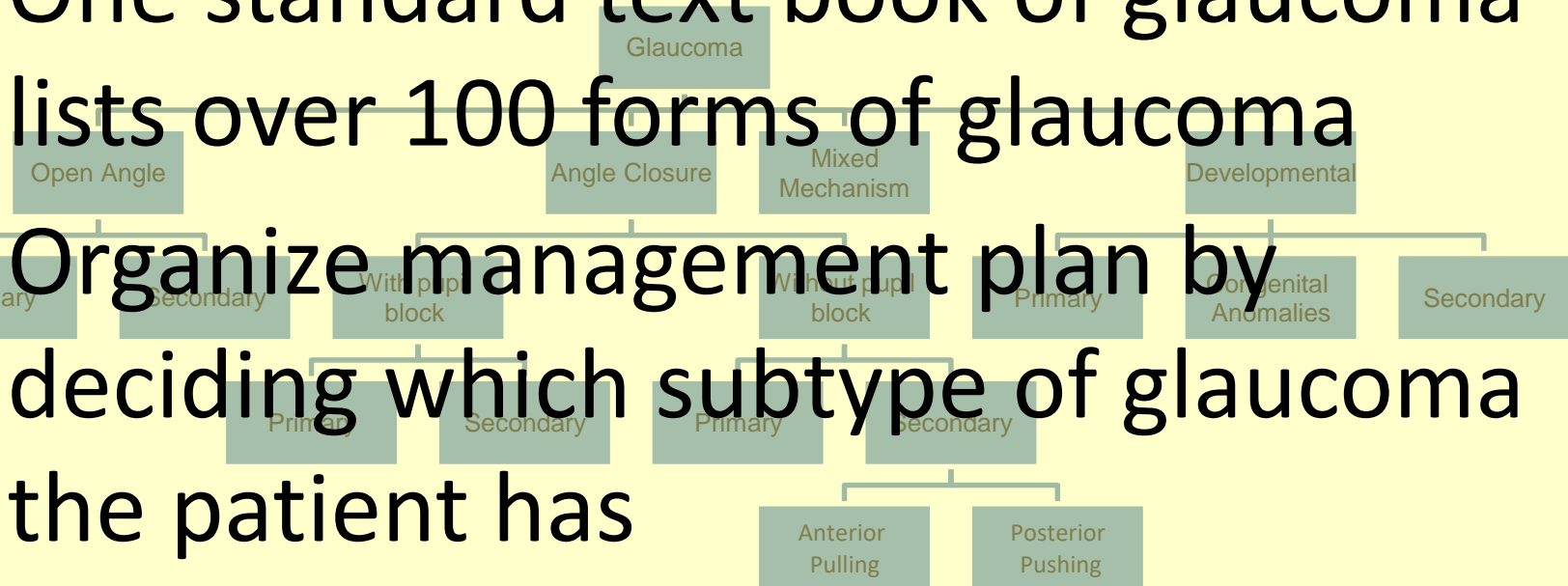
Probability Symbols

- ⦿ P < 5%
- P < 2%
- P < 1%
- P < 0.5%

Tip Three

- One standard text book of glaucoma lists over 100 forms of glaucoma

- Organize management plan by deciding which subtype of glaucoma the patient has



Treatment

- Optic nerve damage in glaucoma is irreversible
- Treatment is aimed at maintaining the residual optic nerve function
- Most modifiable risk factor is IOP
- Lowering IOP Increases the chances of slowing down or stopping nerve damage.

Treatment Modalities

- Medical
- Laser
- Surgical

Medical Treatment

- Prostaglandin analogues (PGAs):
 - Once daily
 - Increases uveoscleral outflow
 - Proinflammation- causing hyperemia

Side effects

Ocular

- Conjunctival hyperaemia
- Eyelash lengthening, thickening hyperpigmentation
- Irreversible iris hyperpigmentation
- Periorbital fat loss
- deepening of the upper lid sulcus
- Hyperpigmentation of periocular skin – Common but reversible



- B blockers:
 - Twice daily
 - Decrease aqueous production
 - Contraindicated in patients with bradycardia/
heart block/ asthma

- α_2 agonists :
 - Aqueous Suppressant
 - Neuroprotective
 - Can cause severe allergic reactions/ contact dermatitis



- Carbonic Anhydrase Inhibitors
 - Systemic (Acetazolamide)
 - Topical
 - Sulfonamide derivative/ watch out for allergy
 - Aqueous suppressant

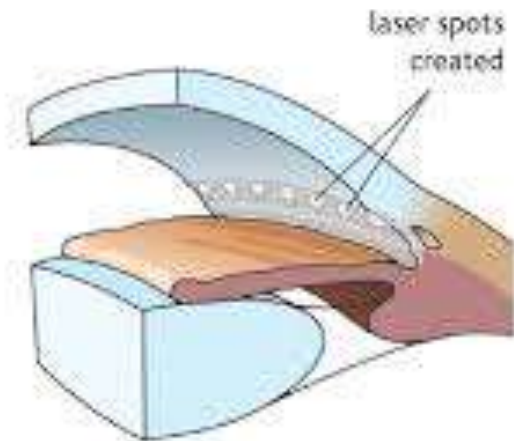
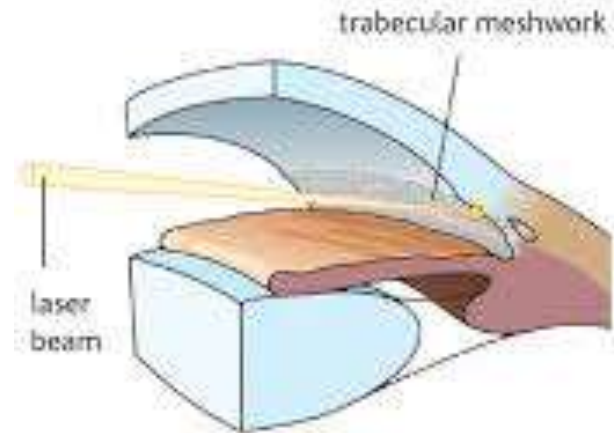
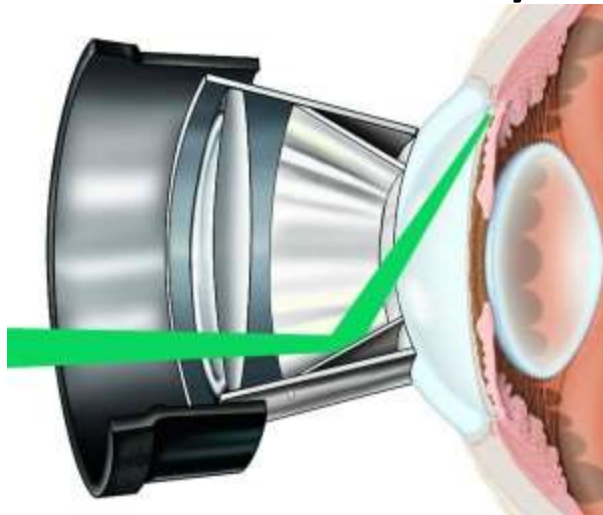
- Parasympathomimetic / Cholinergic agonists
 - Pilocarpine
 - Increase conventional pathway outflow
 - Cause miosis, myopic shift
 - May increase retinal detachment risk

Laser Treatment

- Increase Outflow Facility
 - Trabeculoplasty
 - Iridotomy
- Decrease Aqueous production
 - Cyclodiode laser

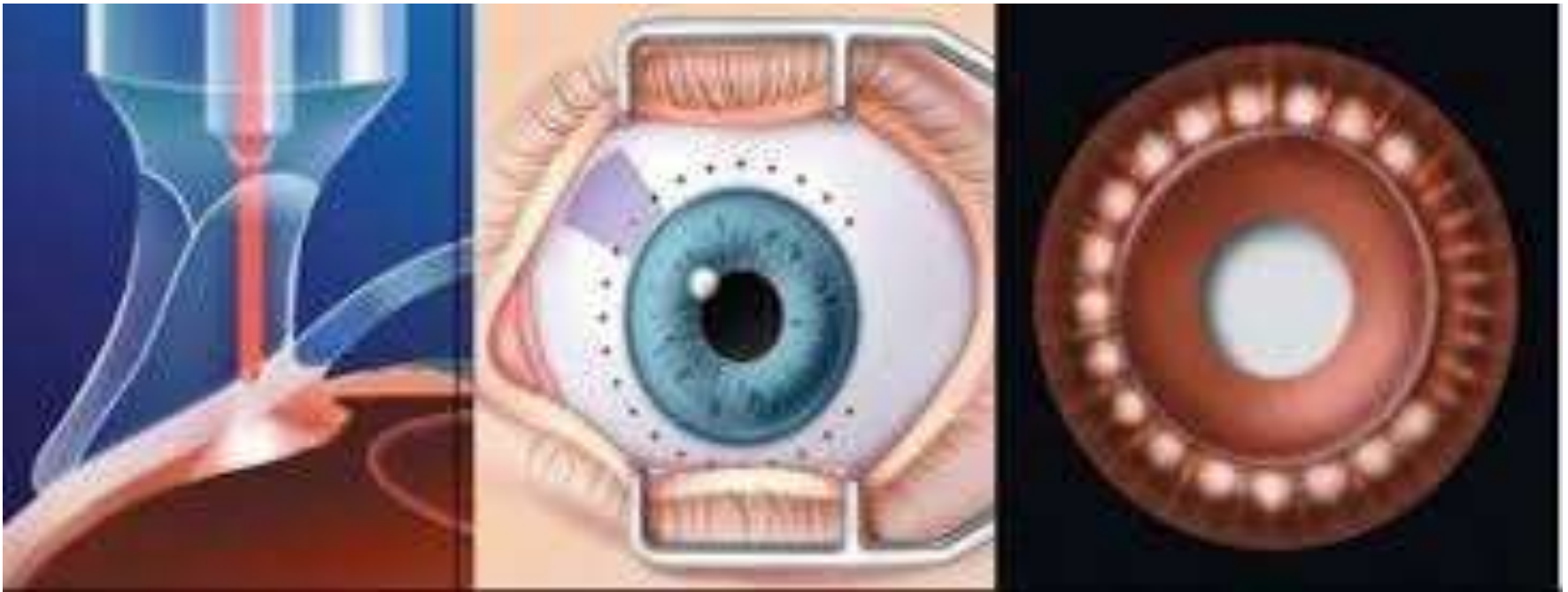
Trabeculoplasty

- Series of laser burns at the TM to increase outflow facility



Cyclodiode

- Transscleral Ciliary body Ablation to decrease aqueous production

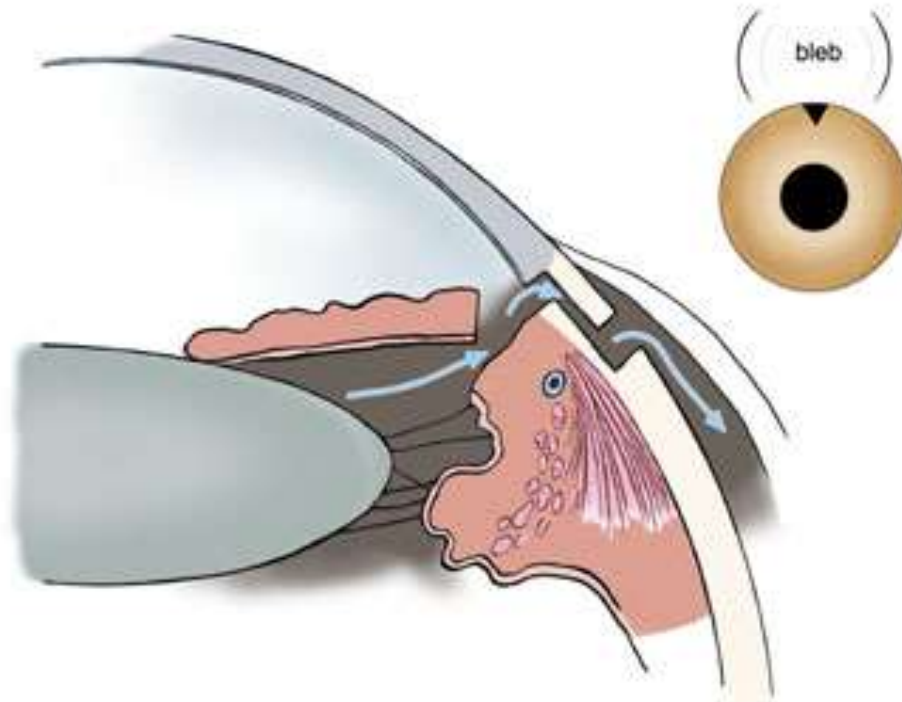


Surgery

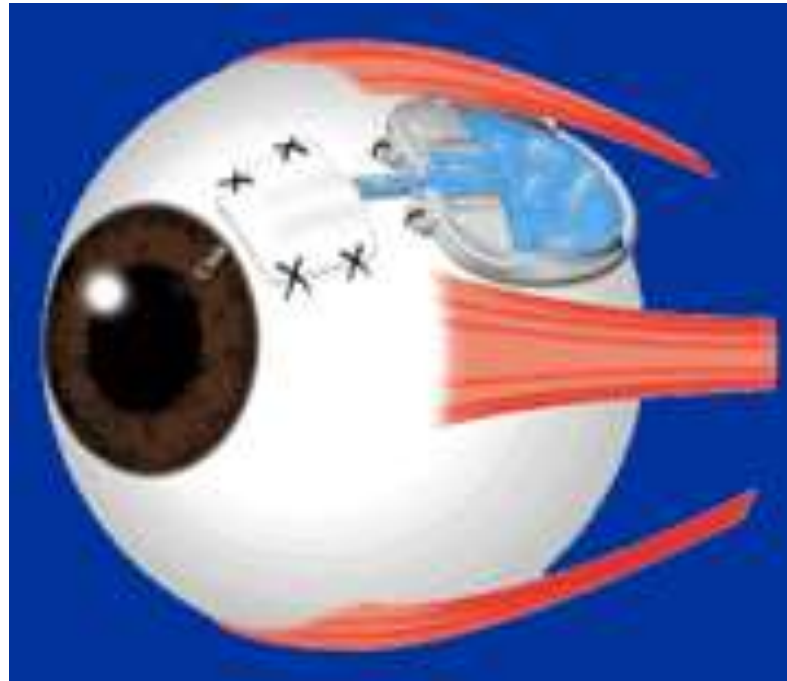
- Trabeculectomy
- Glaucoma Drainage Devices

Trabeculectomy

- A fistula between the anterior chamber and the subtenon space



Glaucoma Drainage Devices



Summary

- **All** patients have glaucoma until proven otherwise
- **Risk** assessment is based on IOP, other risk factors
- **Classify** based on gonioscopy and other anterior segment findings
- **Stage** the disease based on optic nerve and field changes

The End