

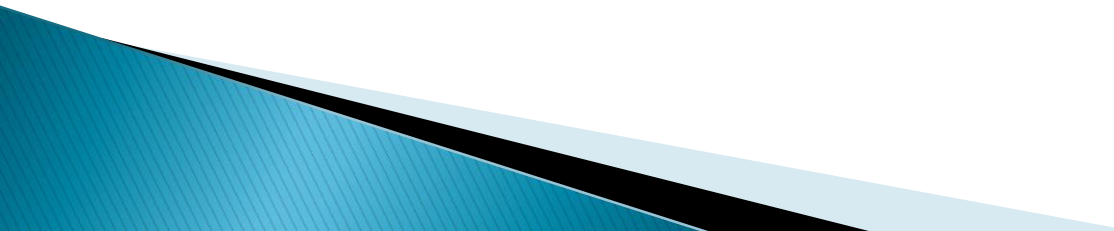


Glaucoma

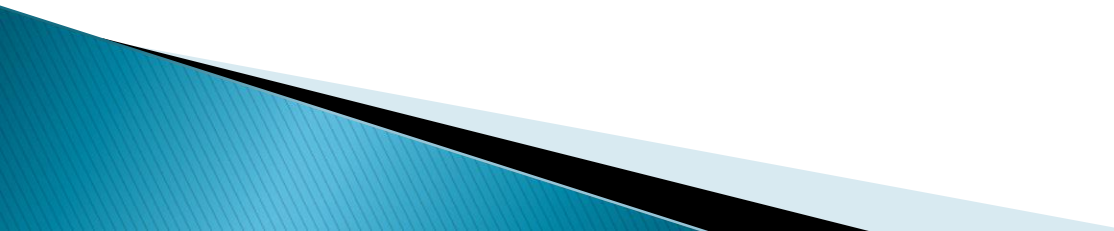
Dr Sana' Muhsen
Associate Professor of
Ophthalmology

Introduction

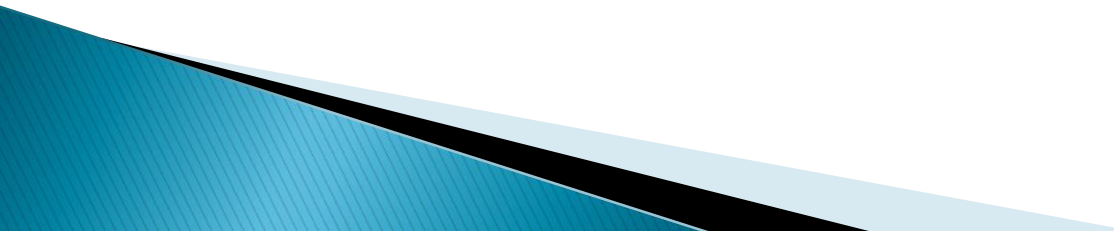
- ▶ Glaucoma is an optic neuropathy associated with characteristic damage to the optic nerve head (cupping) and the visual field (nerve fibre bundle defects).
- ▶ It is a blinding disease where first the peripheral visual field becomes constricted, followed by loss of central visual acuity

- ▶ 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
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Risk Factors

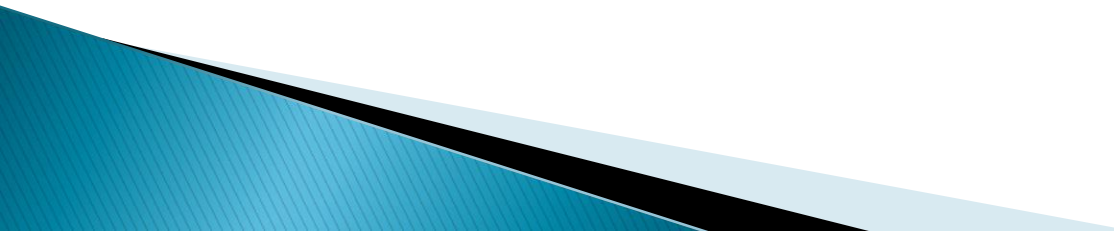
- ▶ Age
 - ▶ Ethnicity
 - ▶ Family History
 - ▶ Intraocular pressure (IOP) is the most important risk factor
 - ▶ Trauma
 - ▶ Eye surgery
 - ▶ Drugs
 - ▶ Refractive errors
- 

Non-IOP dependent risk factors

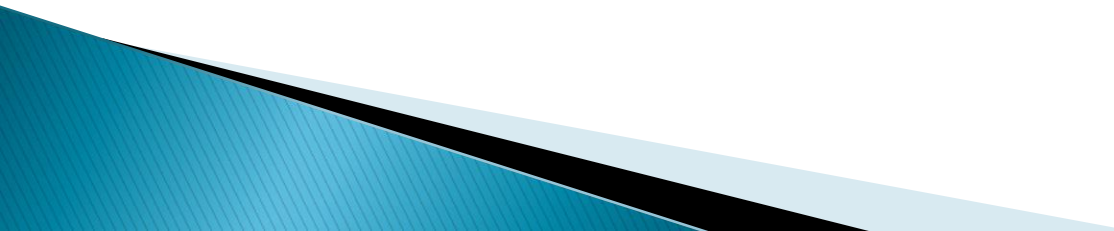
- ▶ Systemic Vascular Dysregulation
 - Raynaud's, Prinzmetal Angina, Migraine
 - ▶ Nocturnal Hypotension
 - ▶ Sleep Apnea
- 

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
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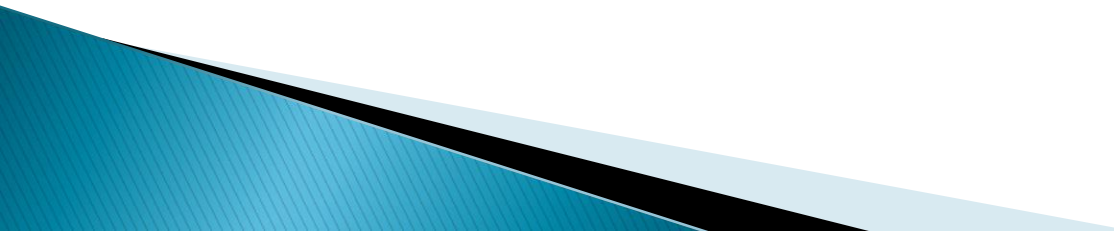
Basic Principles–IOP

- ▶ 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
- 

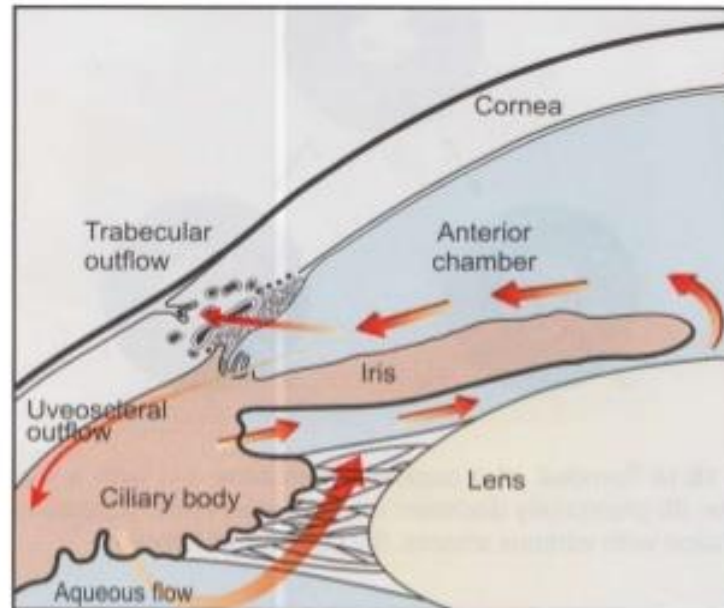
Aqueous Humor

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

Aqueous Humor Pathway

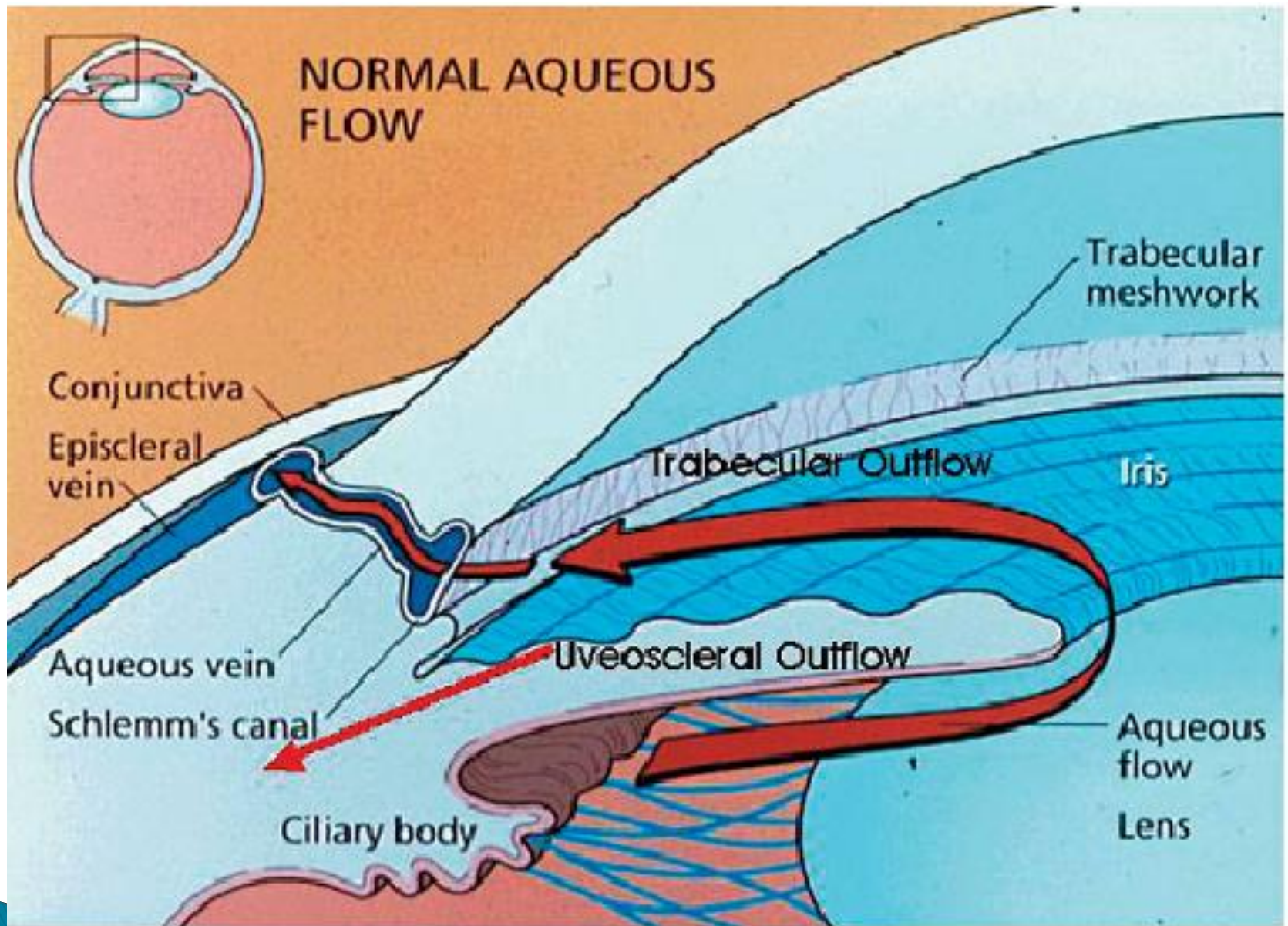
- ▶ Aqueous humor passes from the posterior chamber between the iris and the lens through the pupil into the anterior chamber

AQUEOUS HUMOUR DYNAMICS



Aqueous Humor Pathway

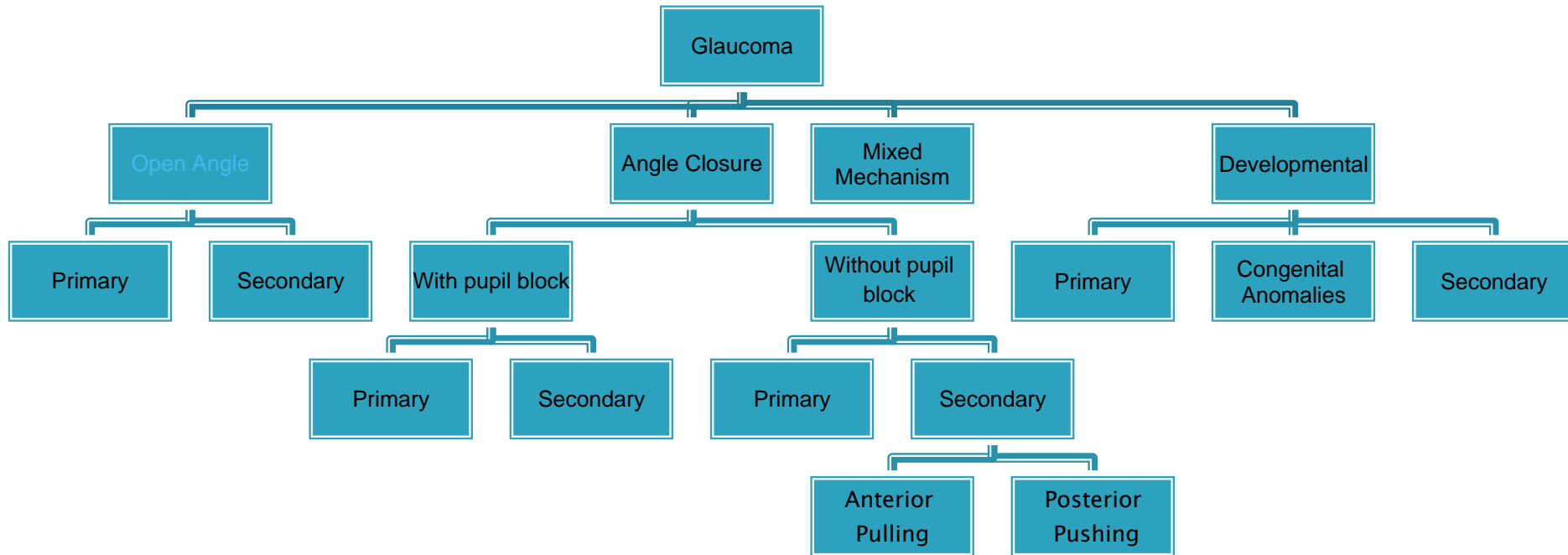
- ▶ It drains through 2 pathways:
 - Conventional (80–90%): trabecular meshwork, Canal of Schlemm, aqueous veins and episcleral veins
 - Uveoscleral (10–20%): Face of the ciliary body and iris to the supraciliary/suprachoroidal space.



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.

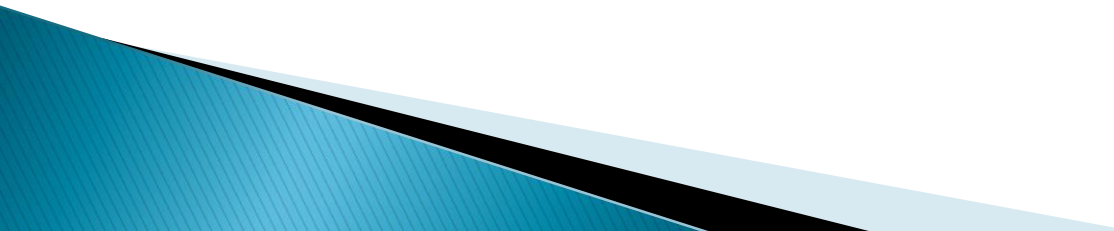
Glaucoma Classification



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)

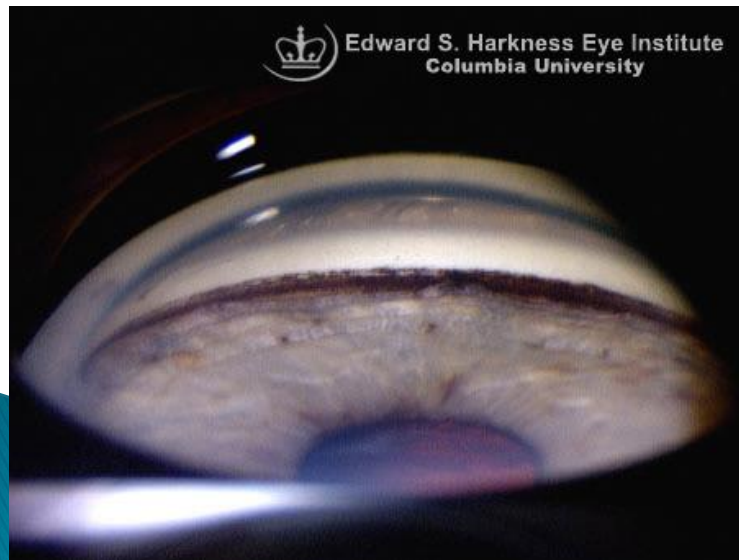
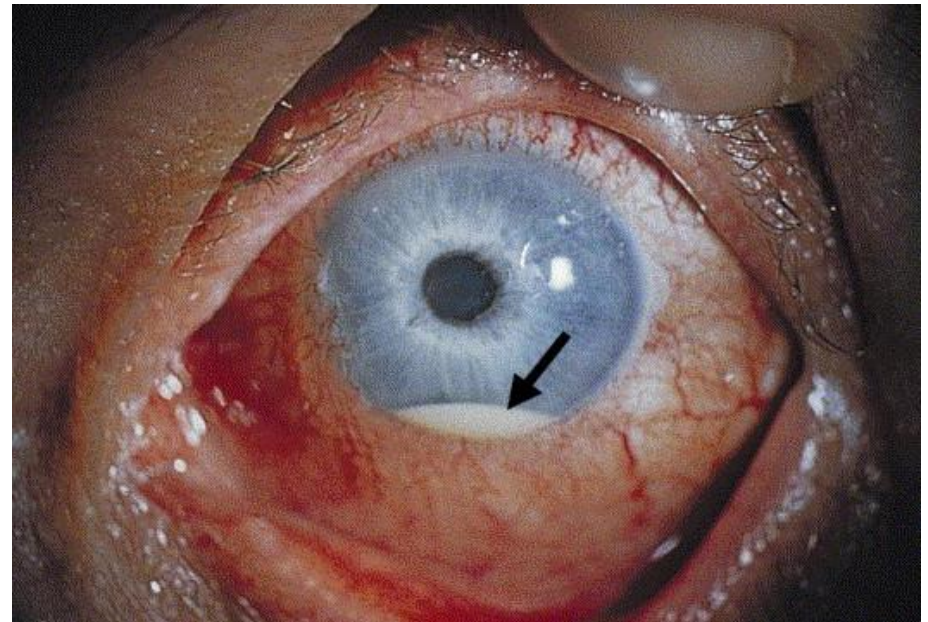
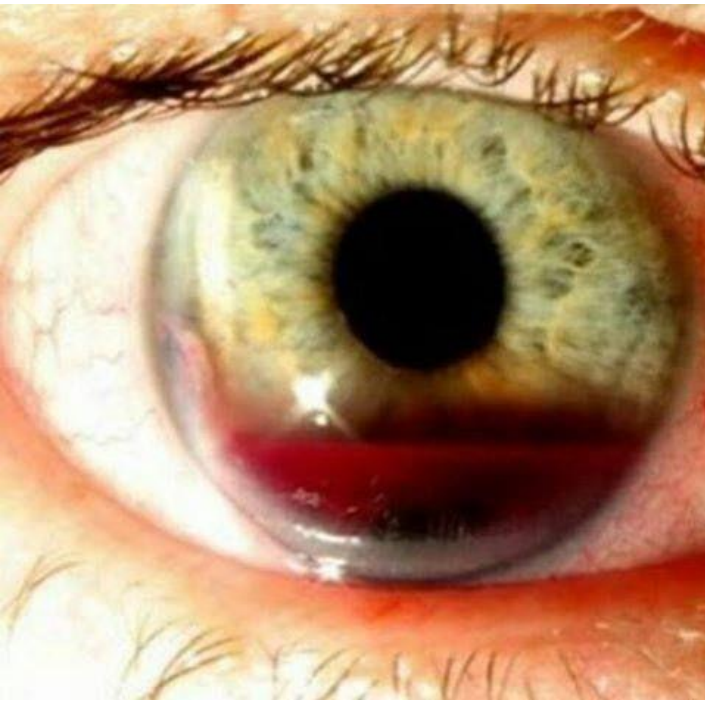
POAG

- ▶ 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
 - Sturge Weber Syndrome
 - SVC obstruction
- ▶ Scarring:
 - Angle recession (trauma)



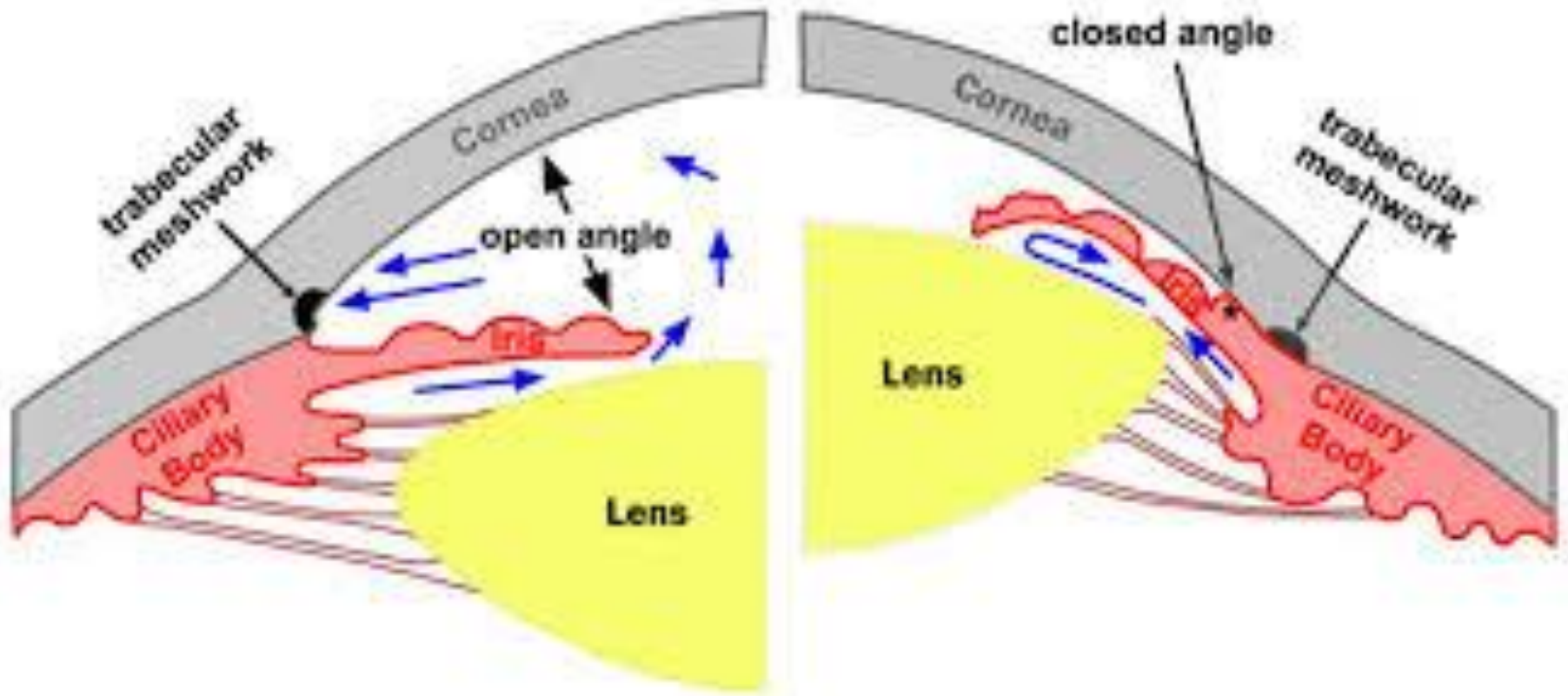
Closed Angle Glaucoma

- ▶ 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

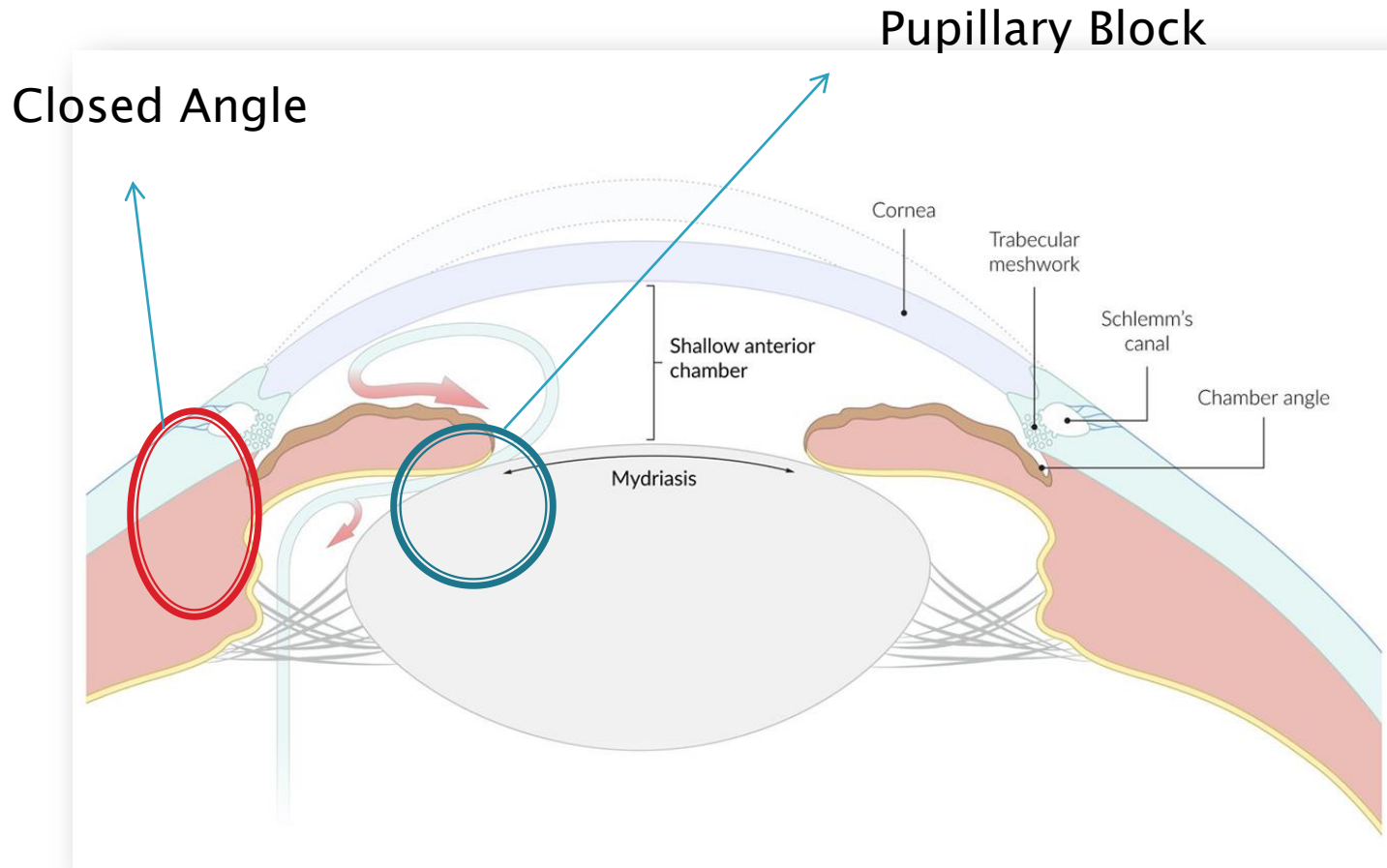
Pathophysiology

- ▶ 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)

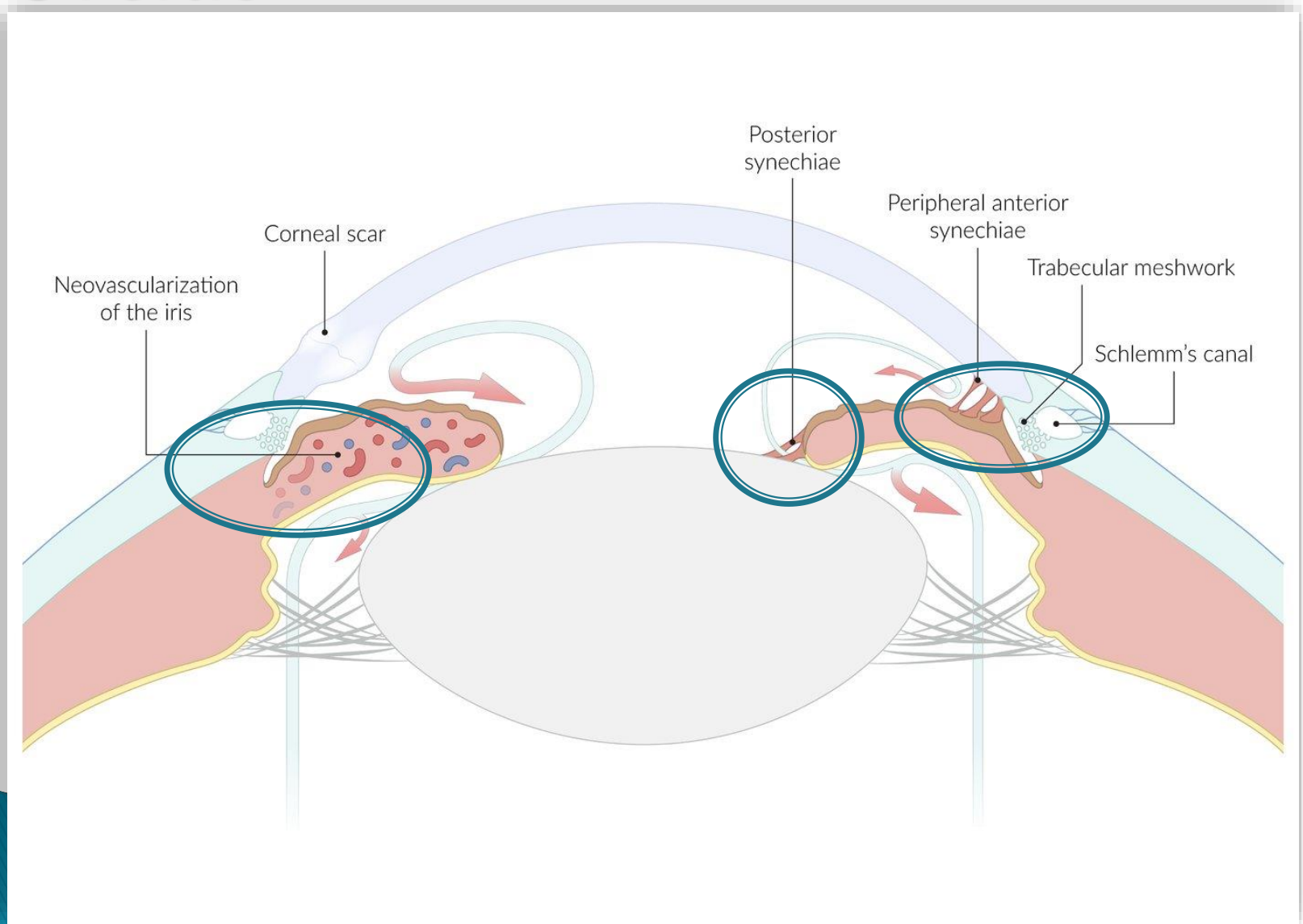
Open vs Closed Angle



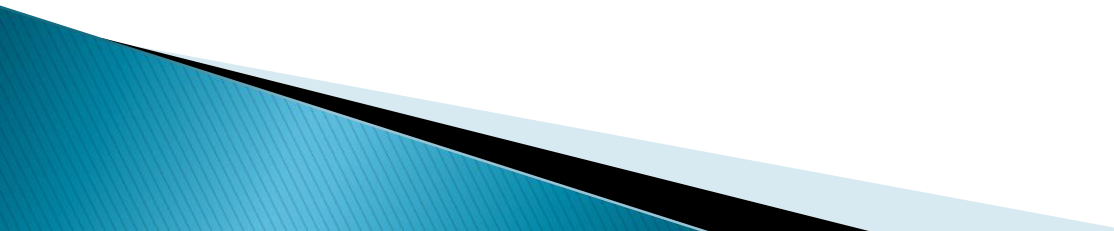
Acute Angle Closure with pupillary Block



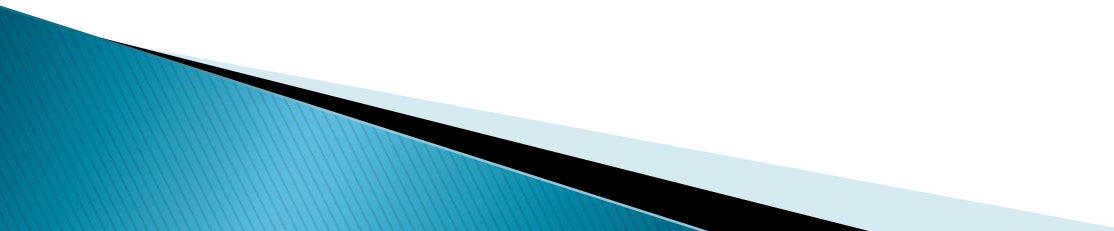
Chronic Angle Closure–NVG/ Uveitis



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
- 

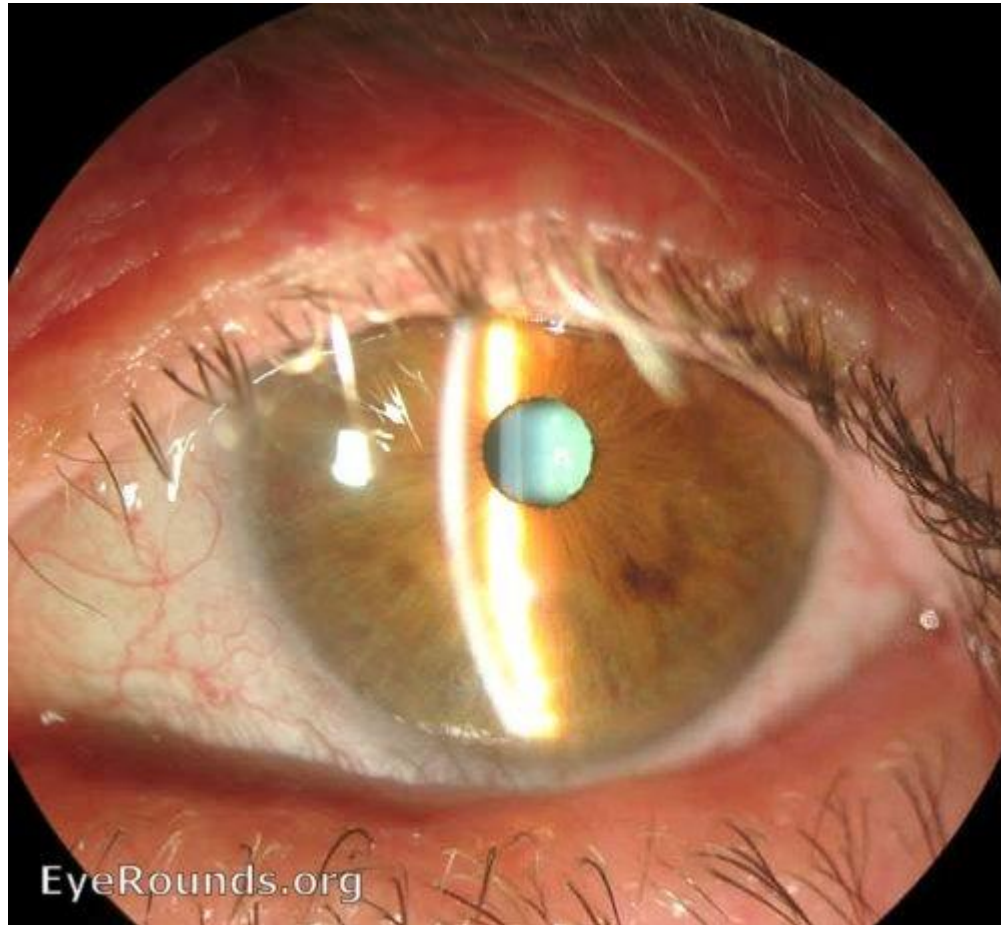
Diagnosis– Physical Examination

- ▶ 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



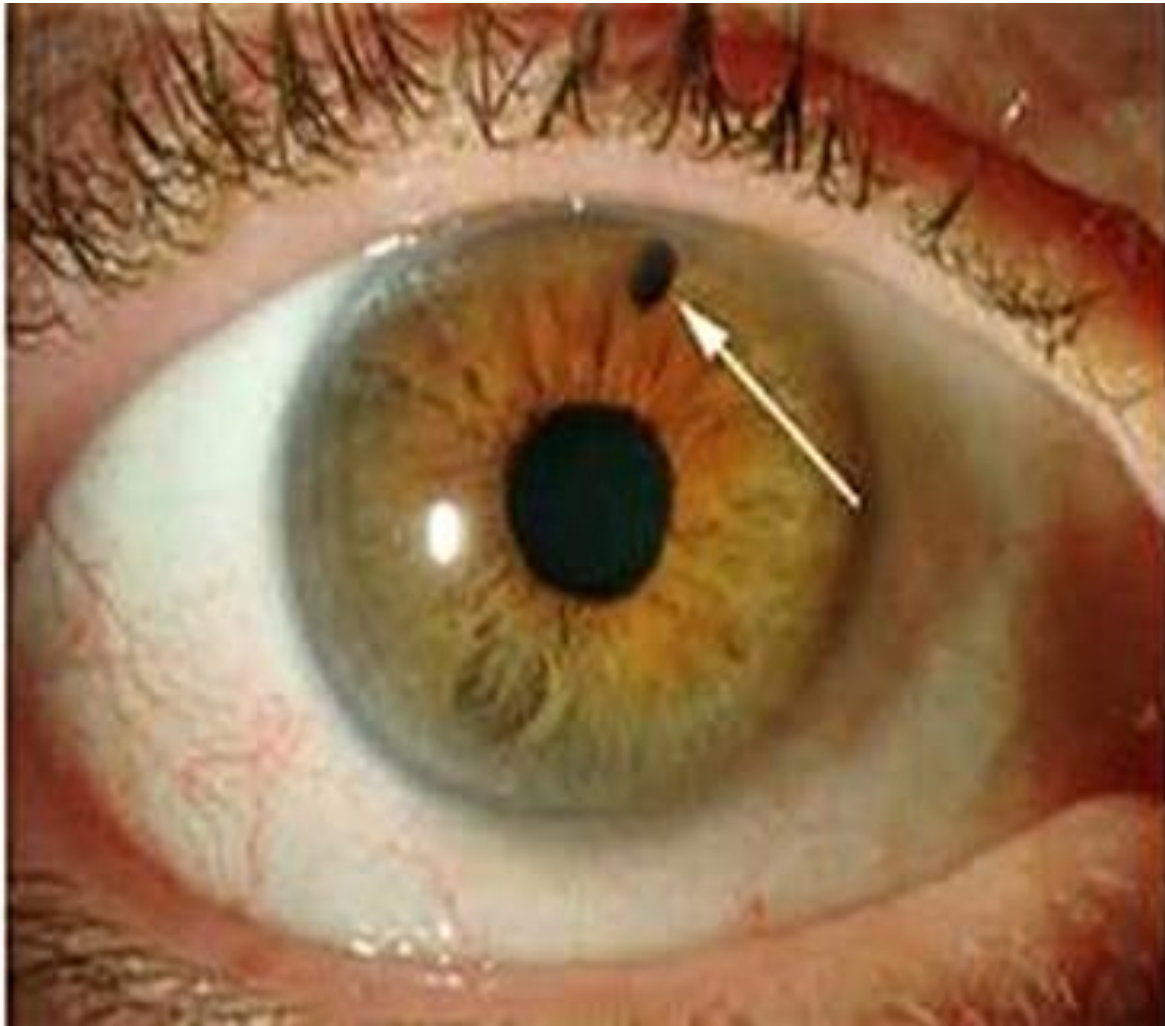
Shallow A/C



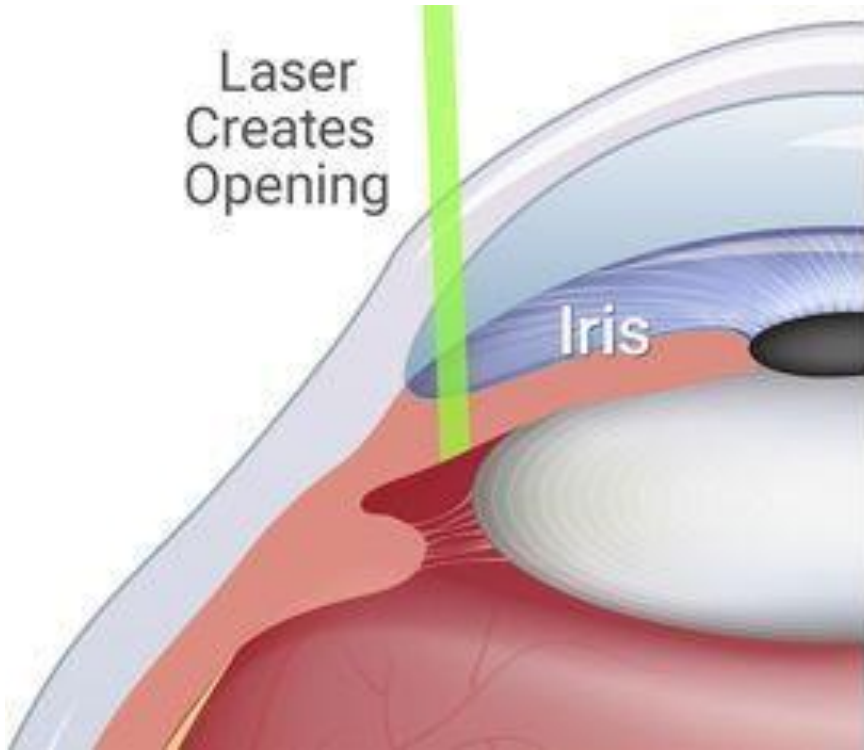
Management outline

- ▶ 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

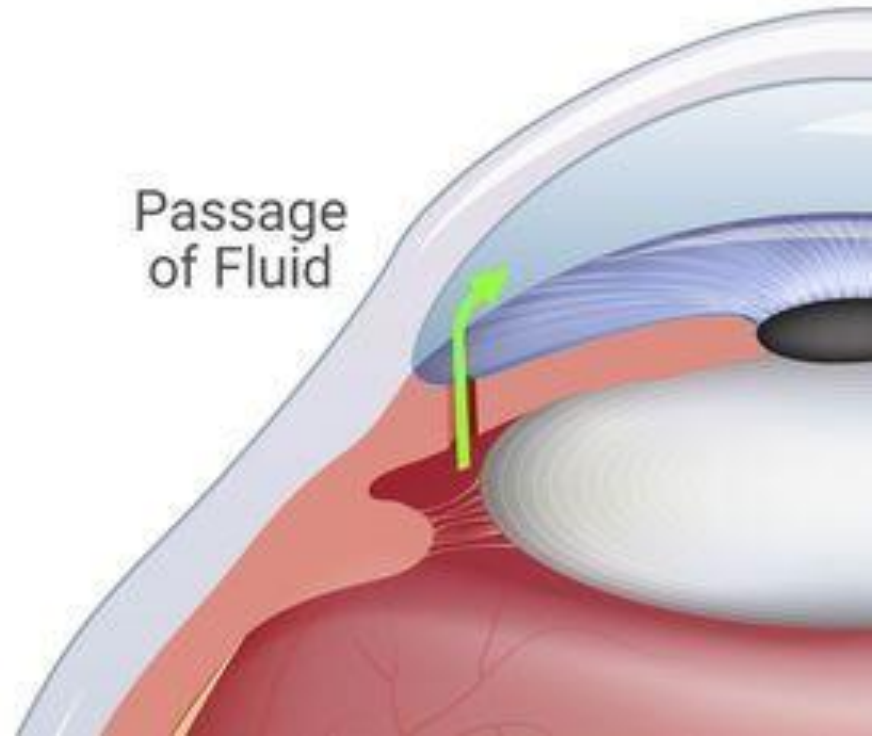
YAG Laser Iridotomy



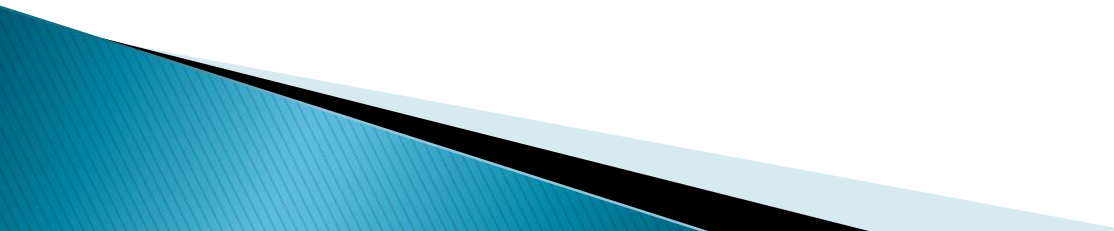
Laser
Creates
Opening



Passage
of Fluid

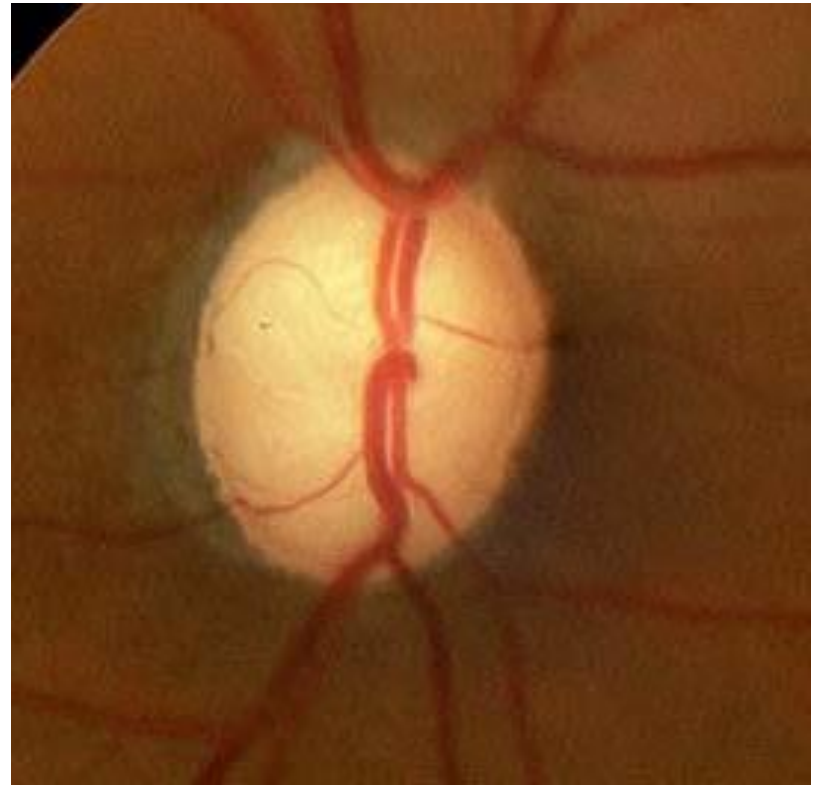


Glaucoma Diagnosis: Tips and Tools

- ▶ History
 - ▶ Physical Examination
 - ▶ Special Tests
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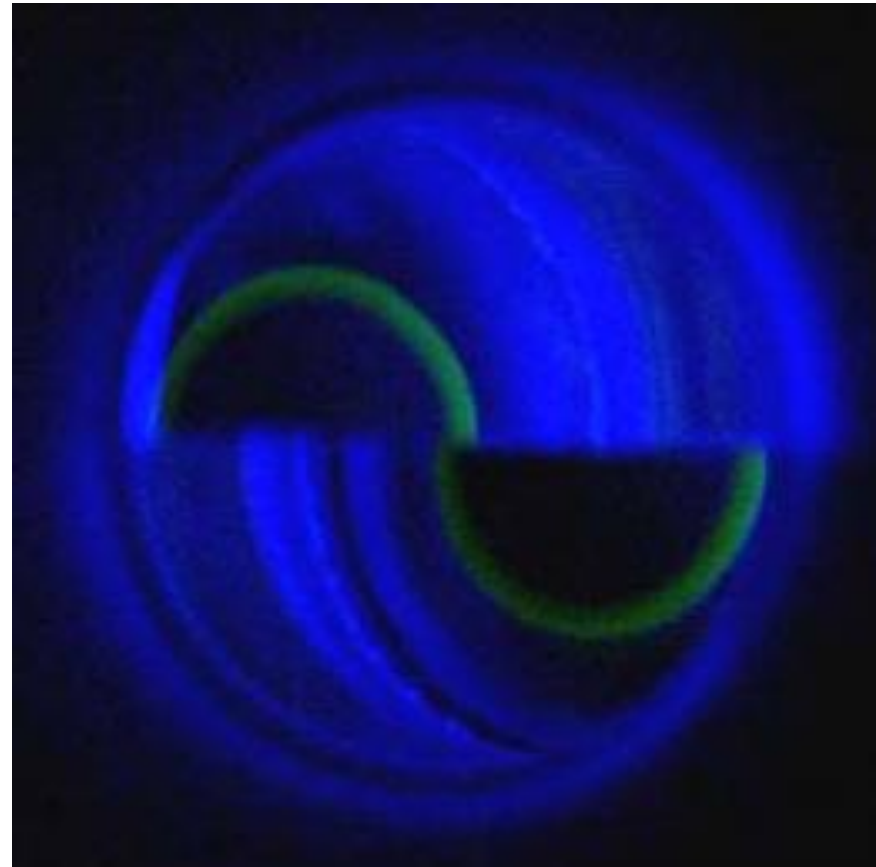
Tip One

- ▶ Every patient has glaucoma until proven otherwise



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

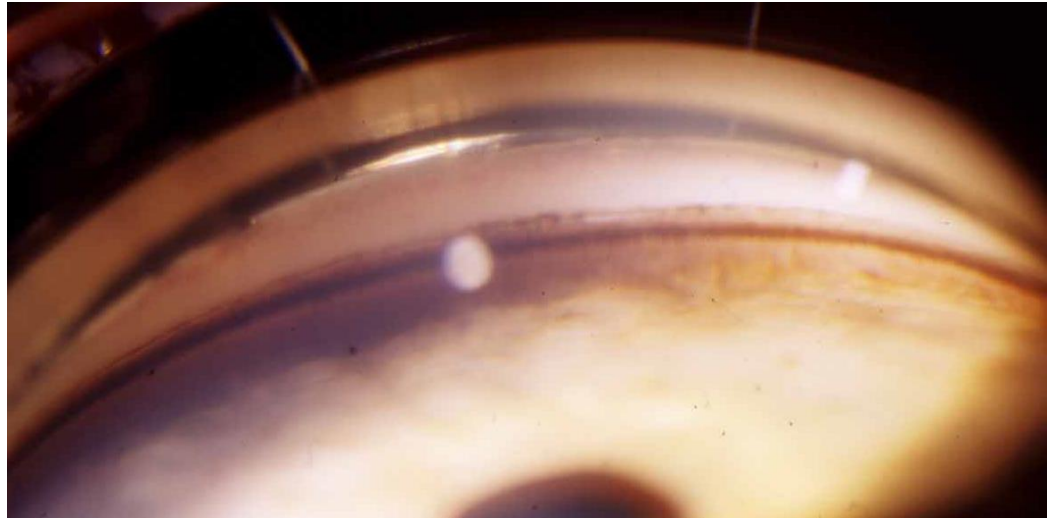
Slit lamp examination

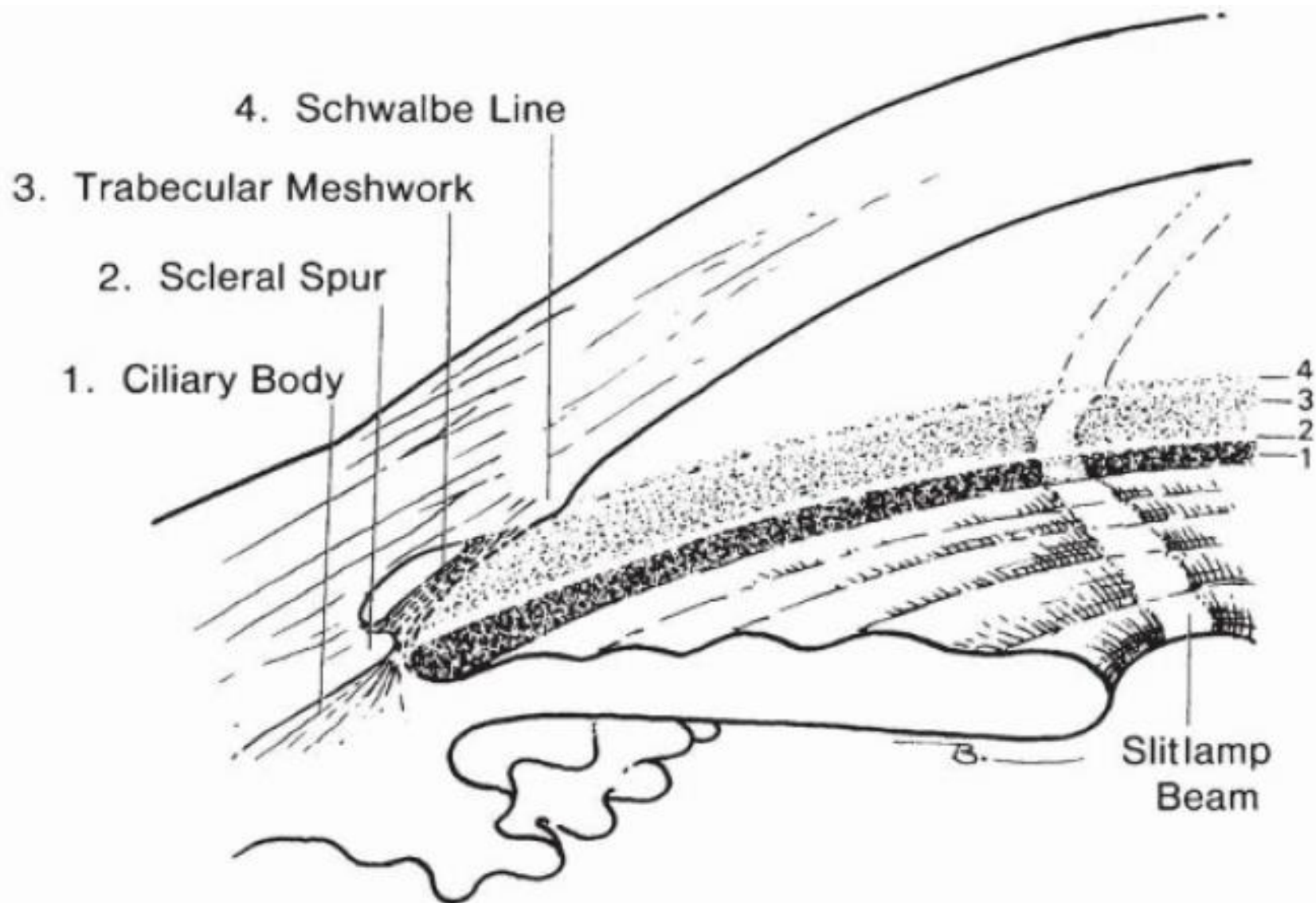
- ▶ Lids/lacrimal
- ▶ Conjunctiva/sclera
- ▶ Cornea
- ▶ Lens
- ▶ Other
 - PXE
 - PDG
 - NVI
 - Iritis
 - PI

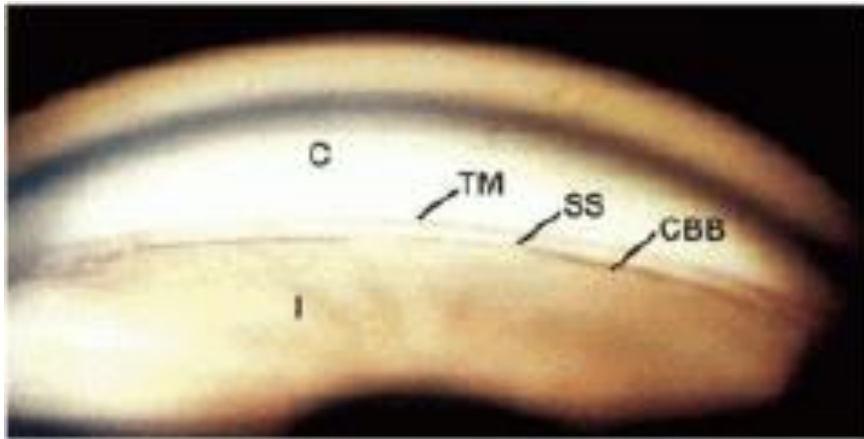


Gonioscopy

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







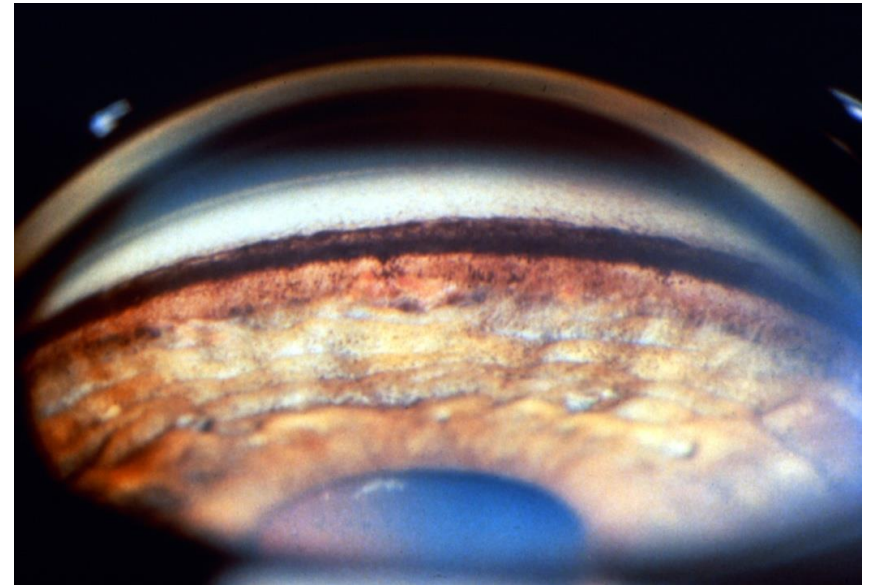
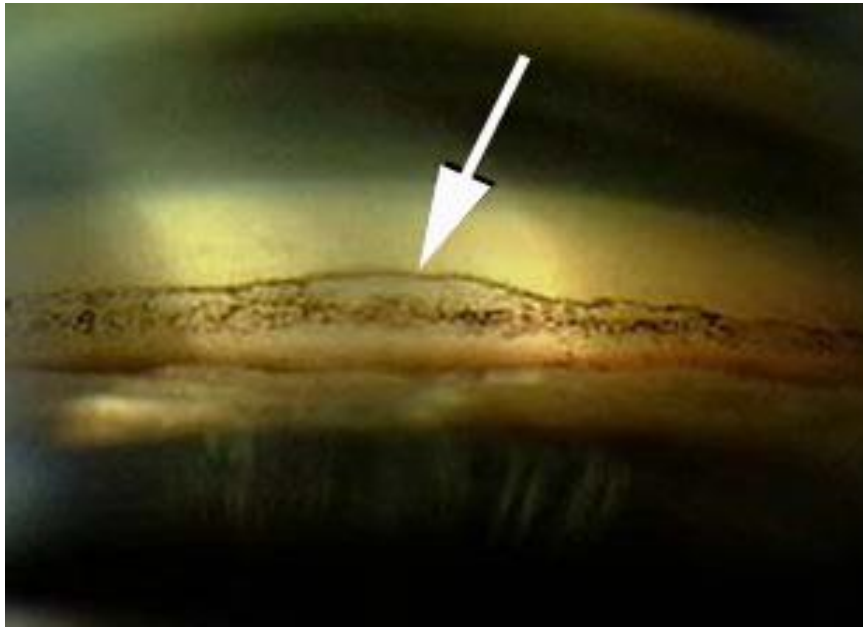
A



B



Gonioscopy- Look for secondaries

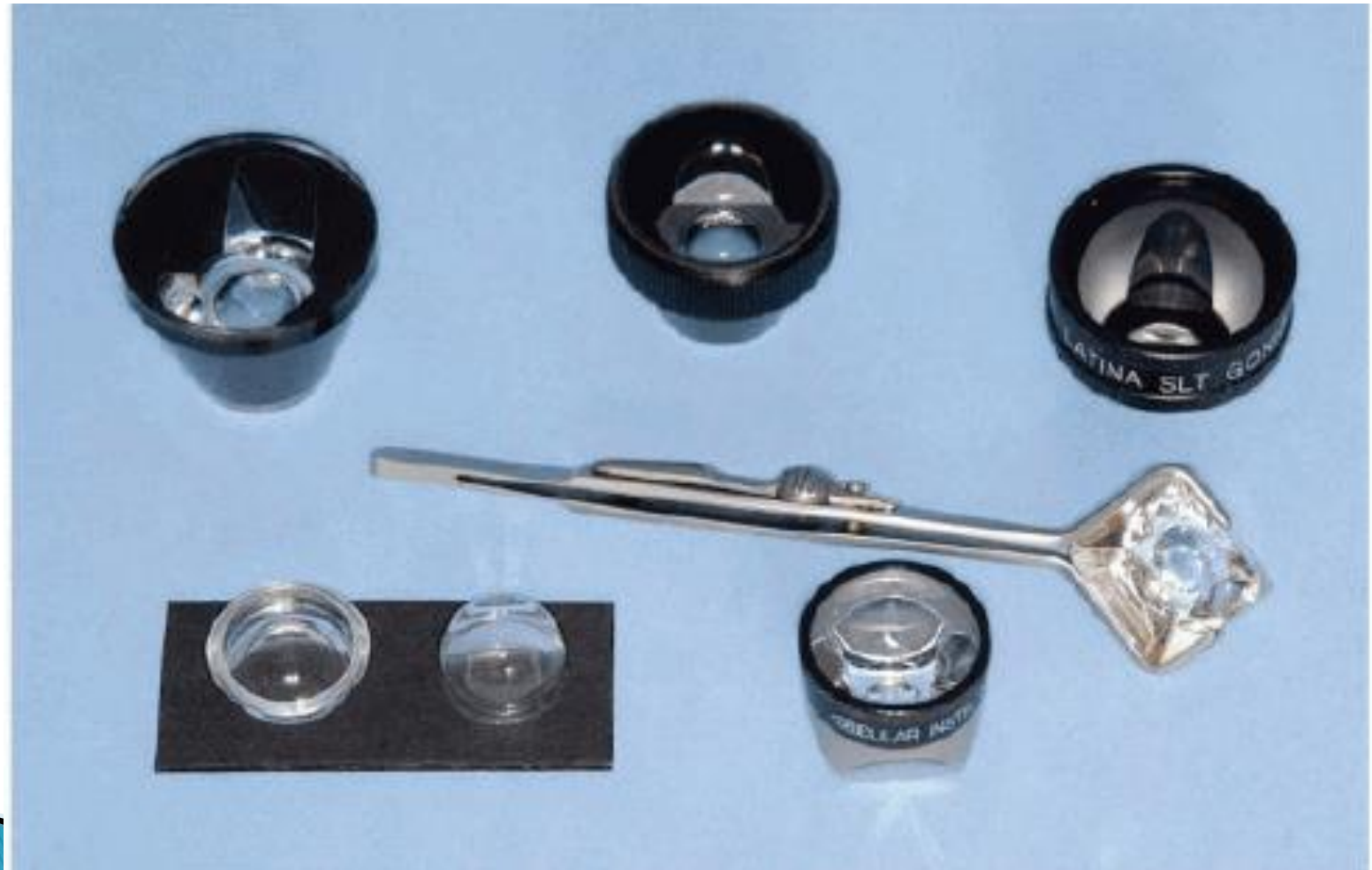


Tool One

- ▶ 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

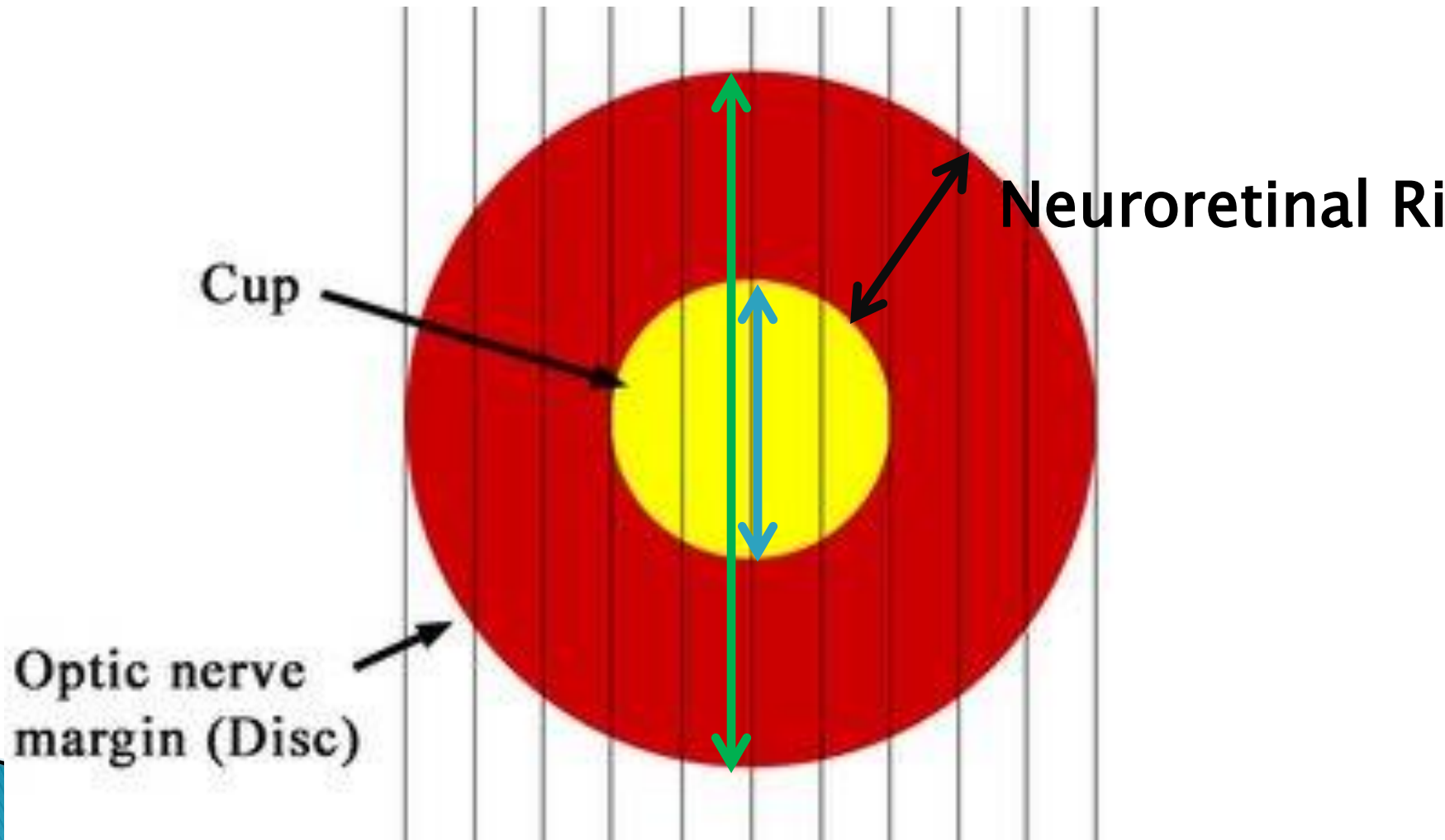


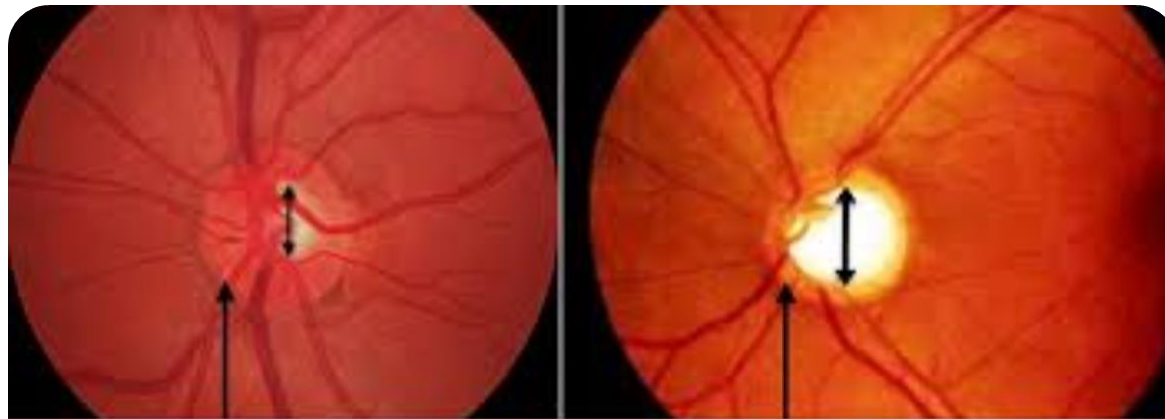
- ▶ Thin cornea can give a falsely low IOP reading
- ▶ Thick cornea can give falsely high IOP

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

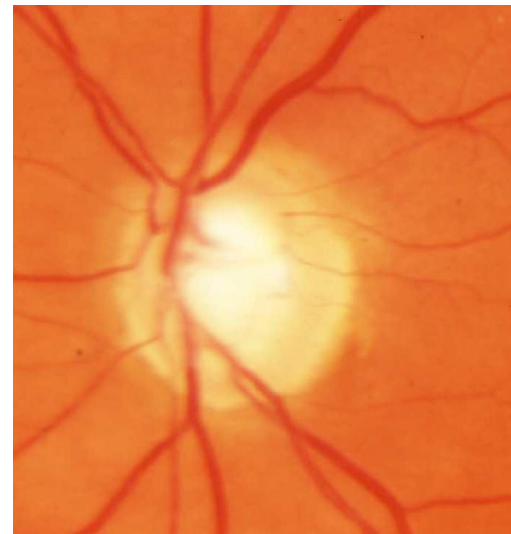
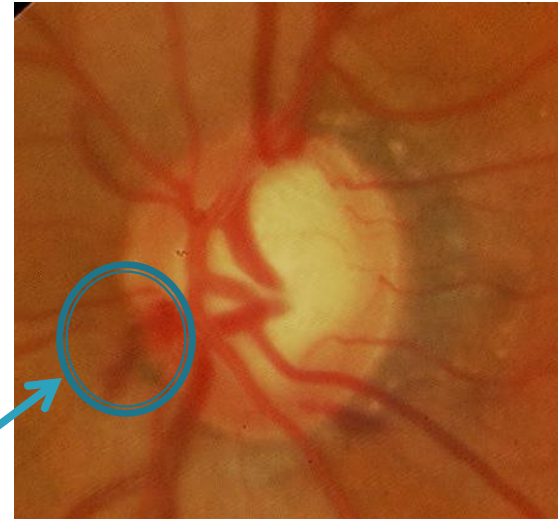
Glaucomatous cupping

Normal optic nerve head

Glaucomatous cupping

Optic Nerve Head Examination

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



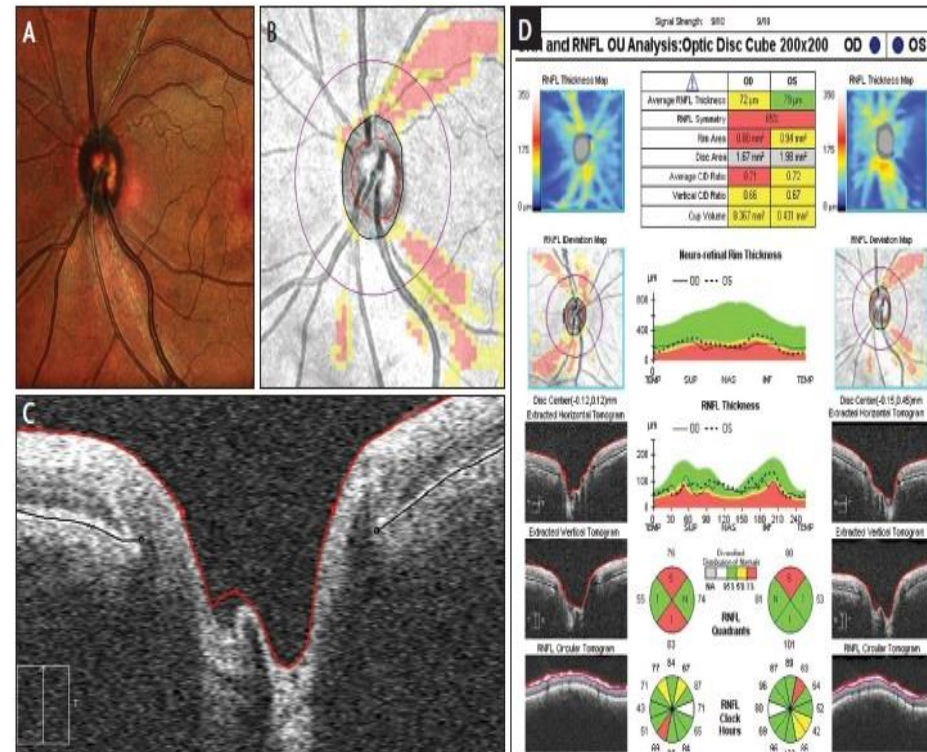


Tool Three

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

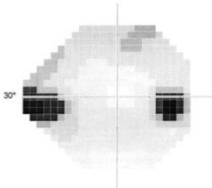
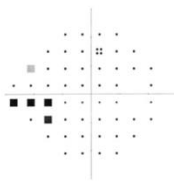
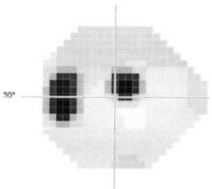
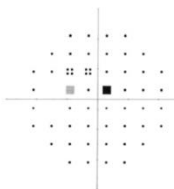
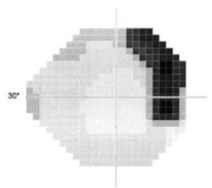
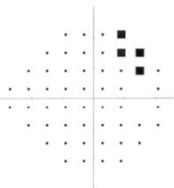
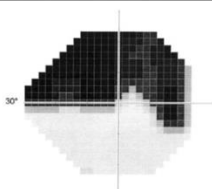
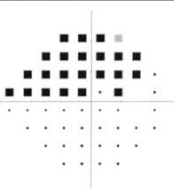
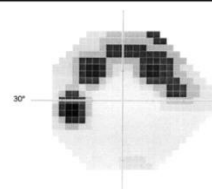
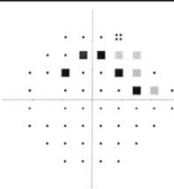
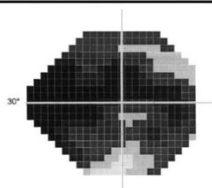
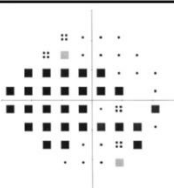




Tool Four – Optic Nerve Head Imaging

- ▶ Computer aided imaging of optic nerve and/or nerve fibre layer
 - Optical coherence tomography/OCT



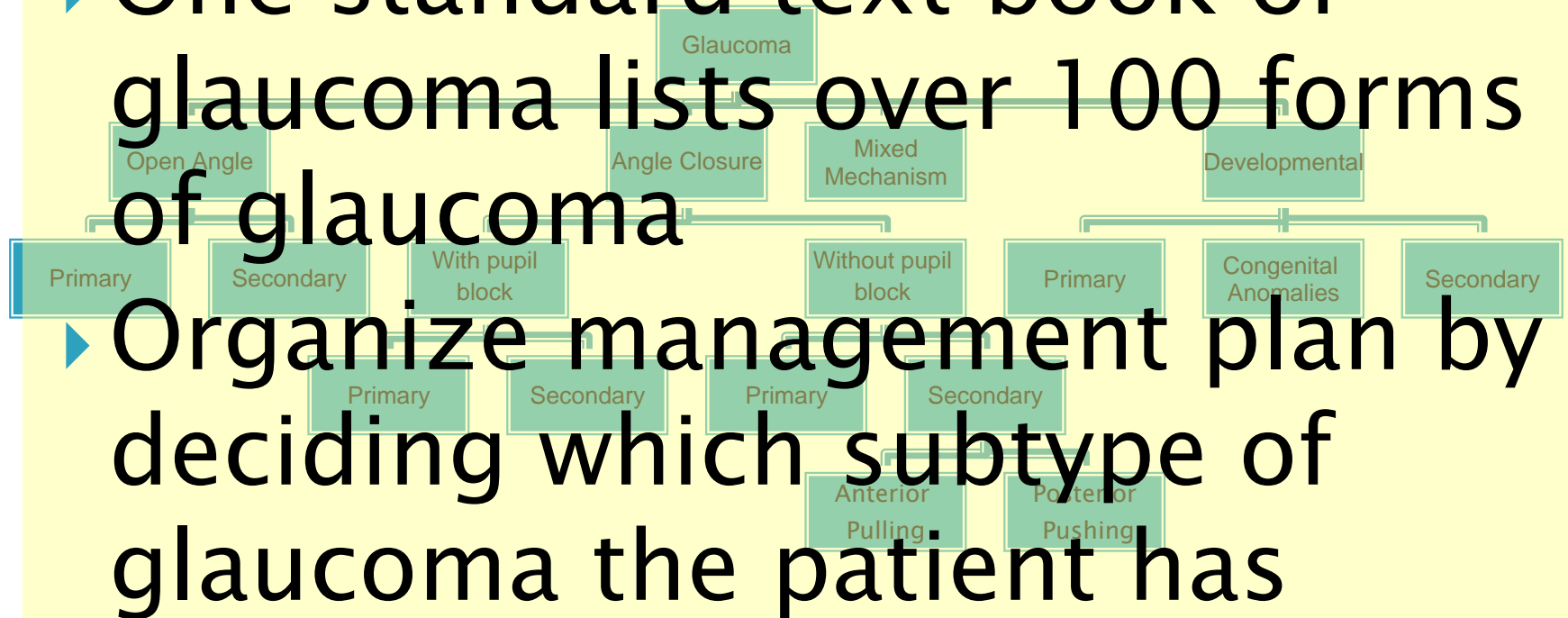
Tool Five – Visual Field Examination

- ▶ 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		
	<p>Probability Symbols</p> <p>  P < 5%  P < 2%  P < 1%  P < 0.5% </p>	

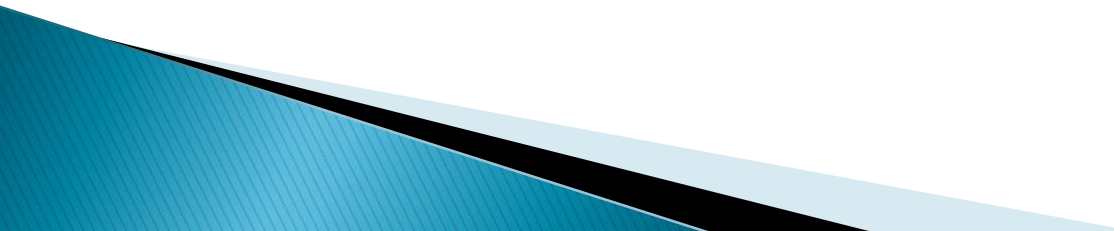
Tip Three

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

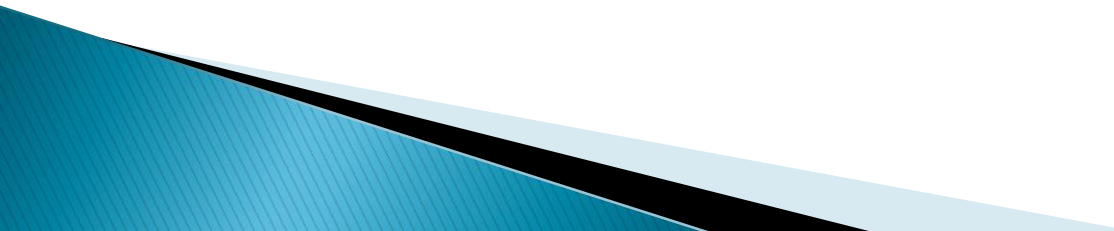


- ▶ Organize management plan by deciding which subtype of glaucoma the patient has

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
- 
- A decorative graphic element in the bottom-left corner of the slide, consisting of overlapping blue and black geometric shapes.

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

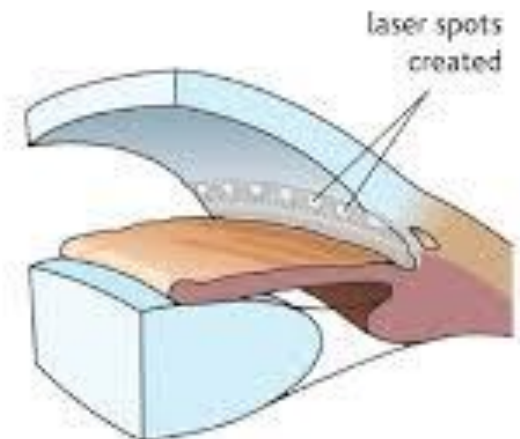
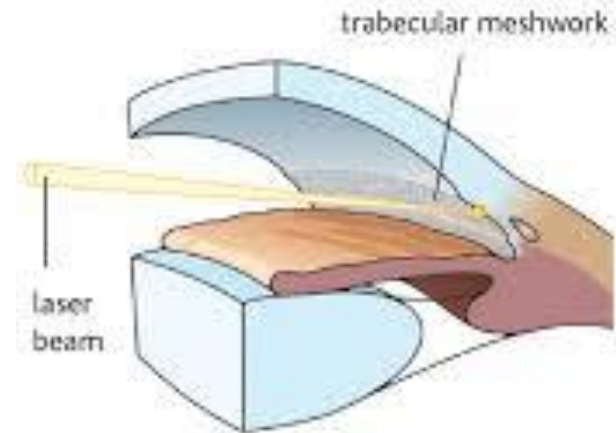
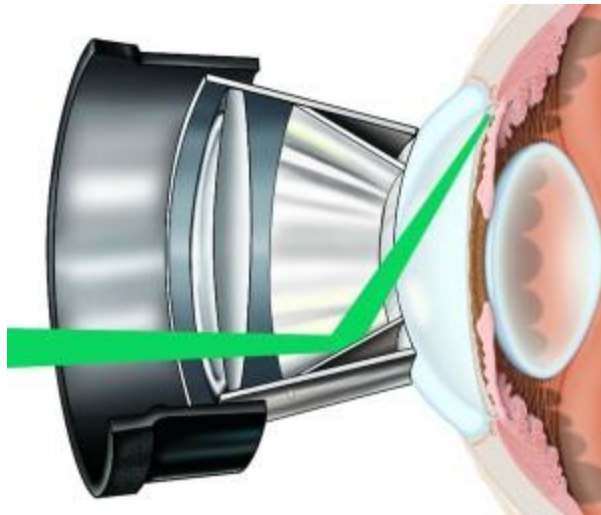
- Pilocarpine
- Increase conventional pathway outflow
- Cause miosis, myopic shift
- May increase retinal detachment risk

Laser Treatment

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

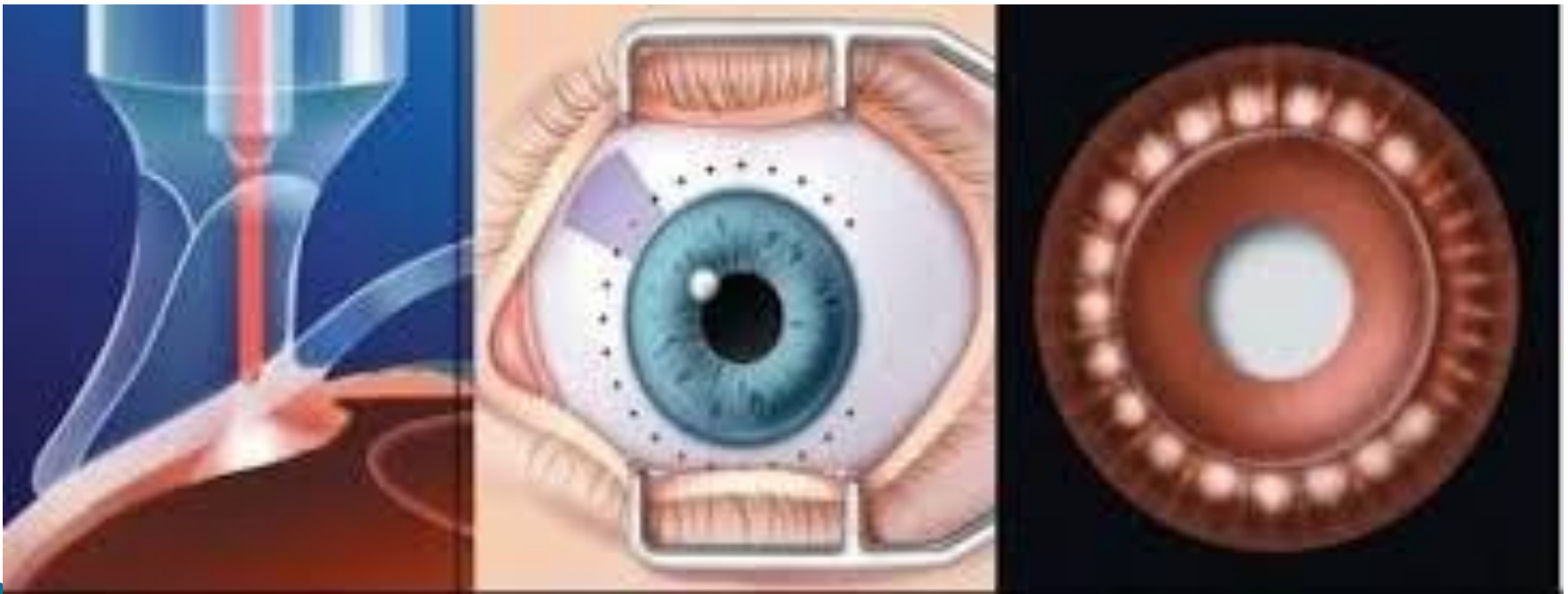
Trabeculoplasty

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



Cyclodiode

- ▶ Transscleral Ciliary body Ablation to decrease aqueous production

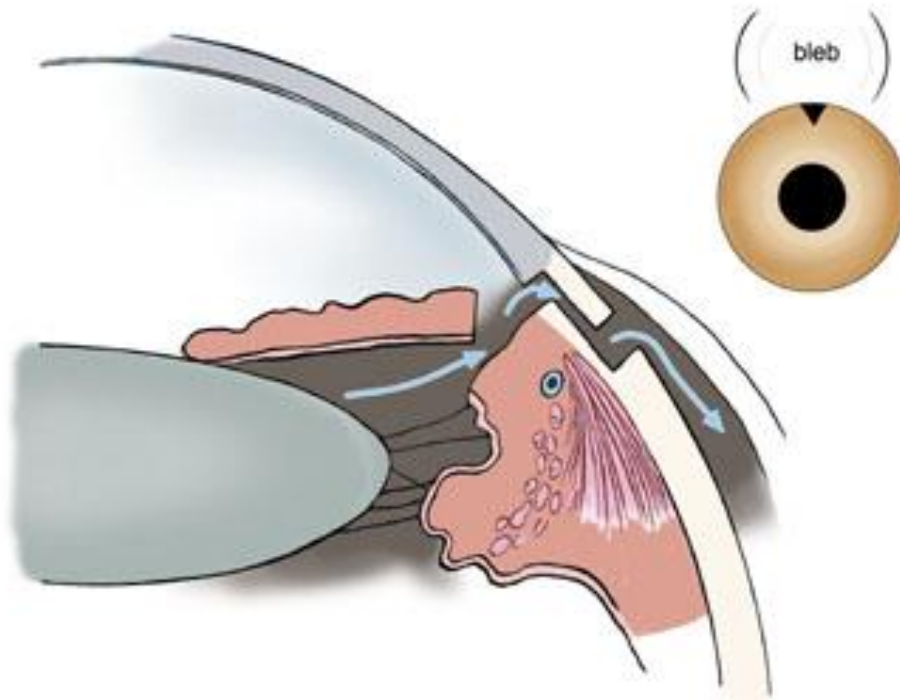


Surgery

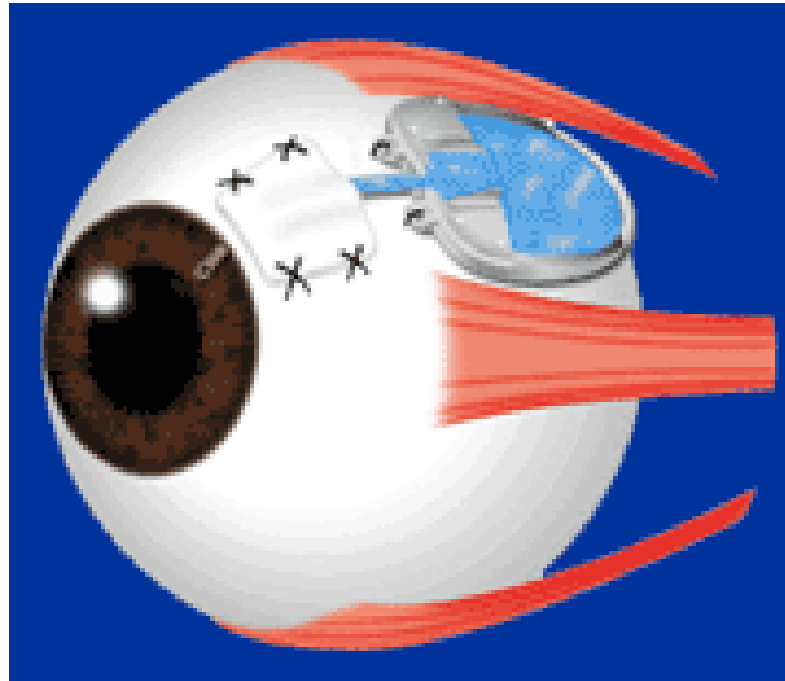
- ▶ Trabeculectomy
- ▶ Glaucoma Drainage Devices

Trabeculectomy

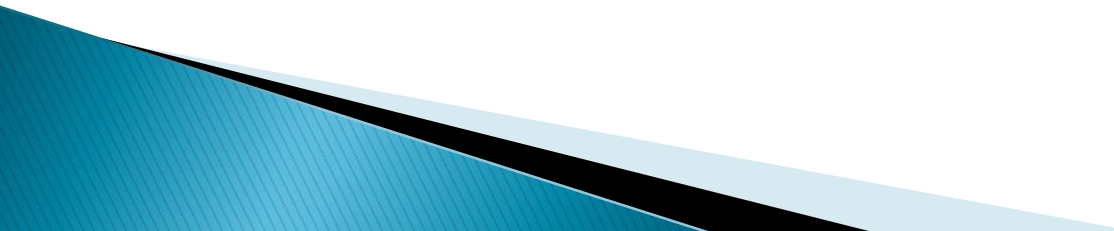
- ▶ A fistula between the anterior chamber and the subtenon space



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