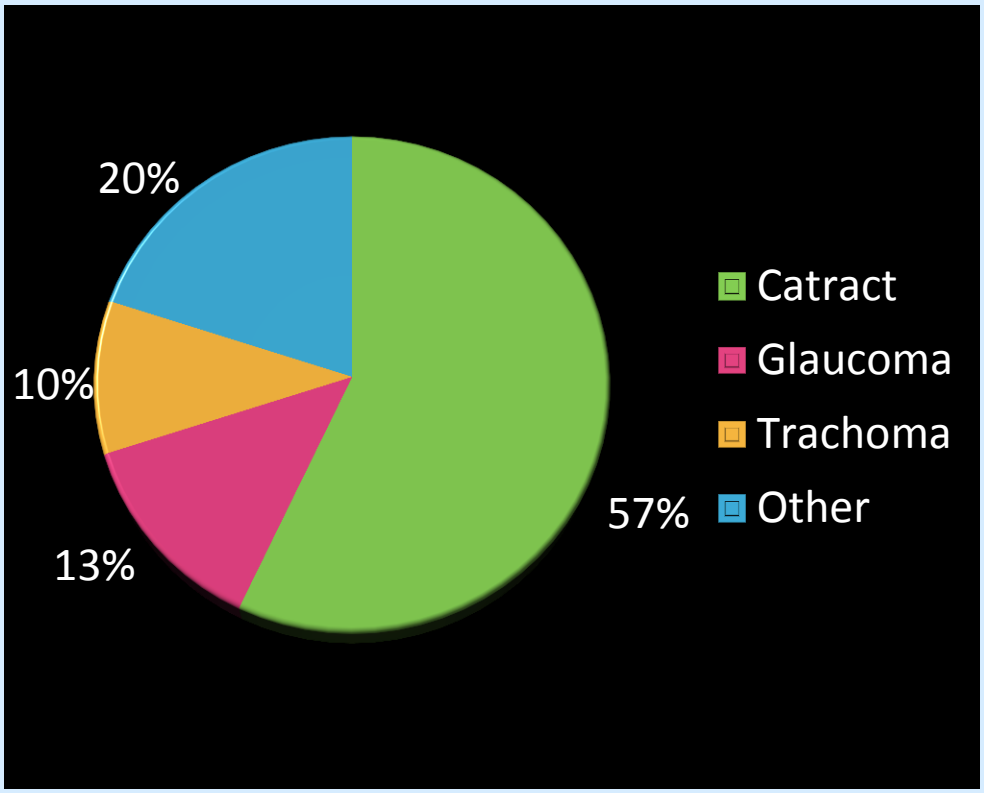
Glaucoma

SILENT THIEF OF SIGHT

Introduction to session

- Definition of glaucoma
- Prevalence
- Classification of glaucoma
- Pathophysiology of Glaucoma
- Risk factors
- Symptoms
- Diagnosis
- Management of glaucoma
- Role of GP in glaucoma diagnosis & treatment

India : Causes of Blindness



- Second leading cause of preventable blindness in INDIA
- Primary open angle glaucoma is estimated to affect 6.48 million persons. The estimated number with primary angle-closure glaucoma is 2.54 million.
- >90% were not aware of the disease.
- 16 million Indians will be affected by 2020.

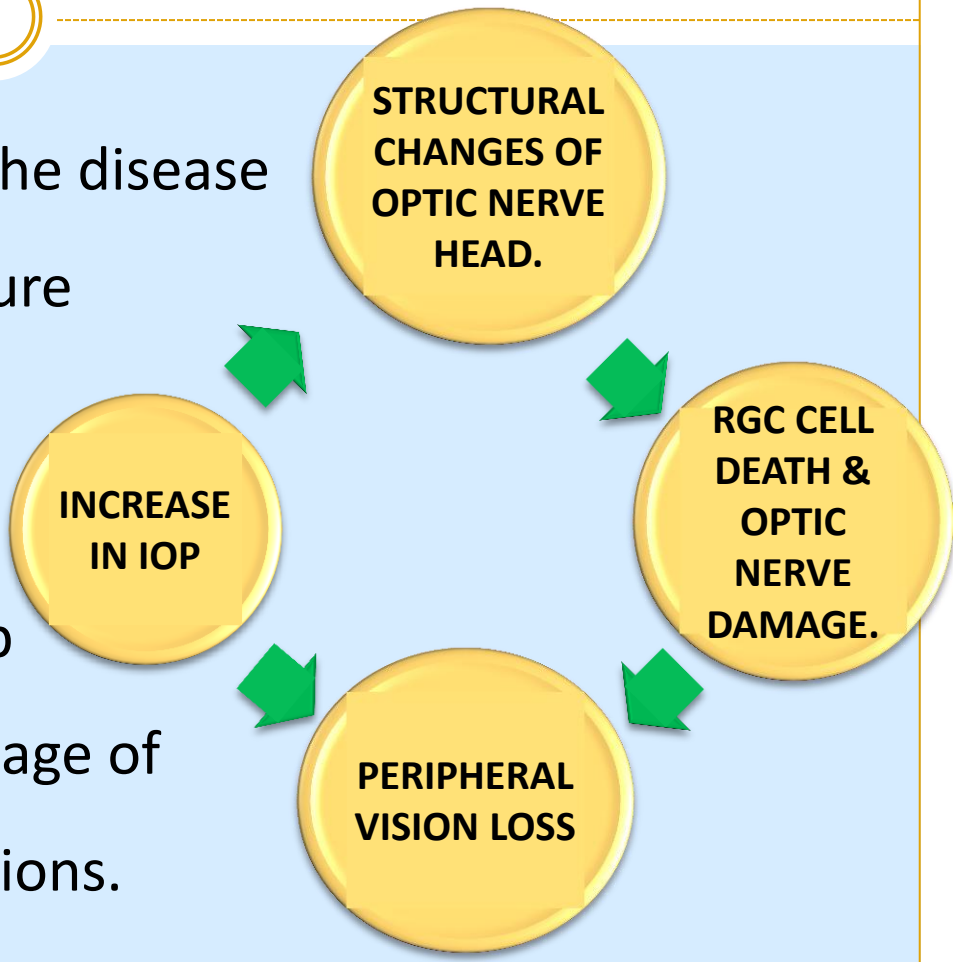
Ref: Asian J. of Ophthalmology vol.3. No.3. 4, 2001 suppl.
J. of Glaucoma, vol 19 no (6),2010:391-397

Why Glaucoma?

- Second leading cause of preventable blindness in INDIA
- Silent thief of sight (stealing vision without warning & often without symptoms).
- Leads to progressive & irreversible vision loss.
- No cure.
- However if diagnosed early, can be treated by life long therapy which aims to prevent/delay further vision loss.
- Awareness regarding glaucoma is very low compared to cataract.

GLAUCOMA: HOW DEFINITON HAS EVOLVED OVER THE YEARS

- Glaucoma was widely known as the disease related to rise in intraocular pressure (IOP) > 21 mm Hg
- Glaucoma is chronic progressive optic neuropathy caused by a group of ocular conditions leading to damage of optic nerve with loss of visual functions.



Ref: AAO preferred Practice Patterns

Glaucoma

PATHOPHYSIOLOGY

GLAUCOMA : AQUEOUS HUMOR

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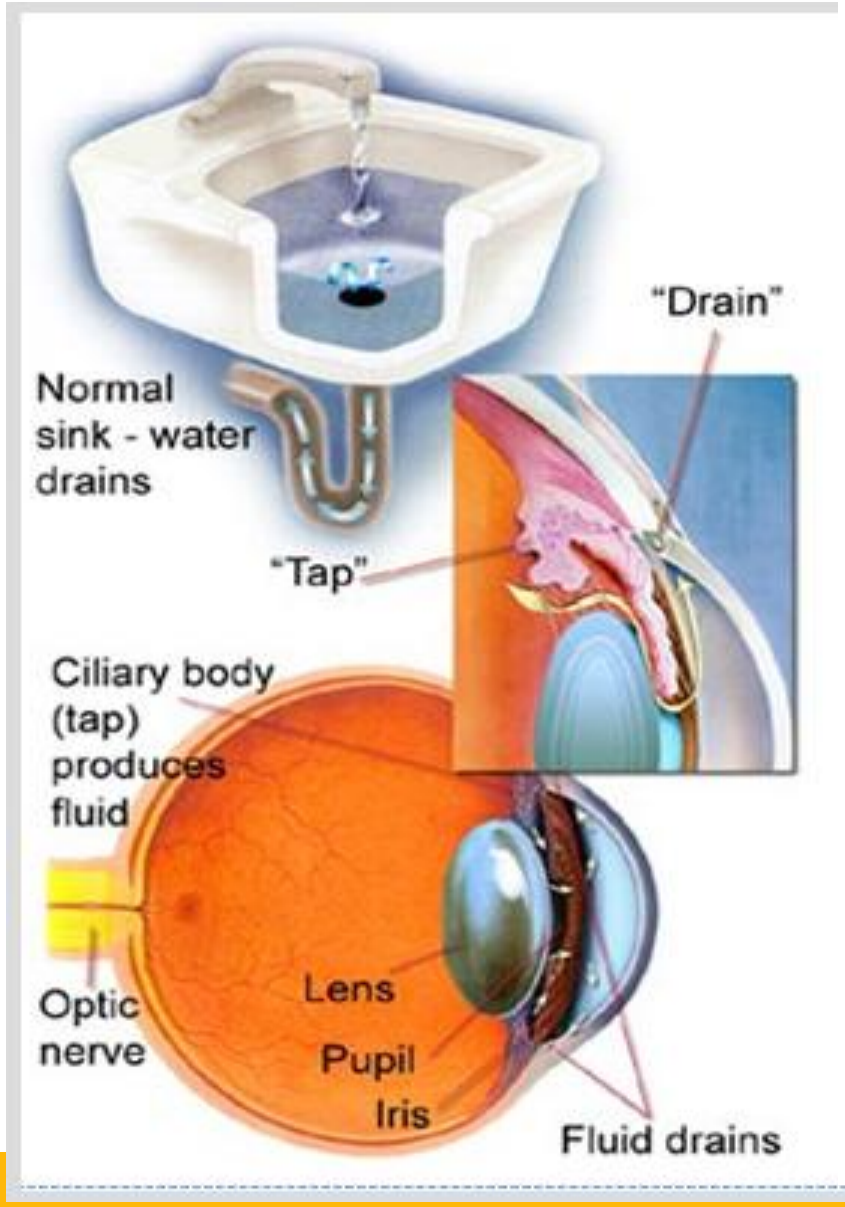
Trabecular meshwork & aging



Optic nerve damage-Optic Disc Cupping

IOP rises

↓

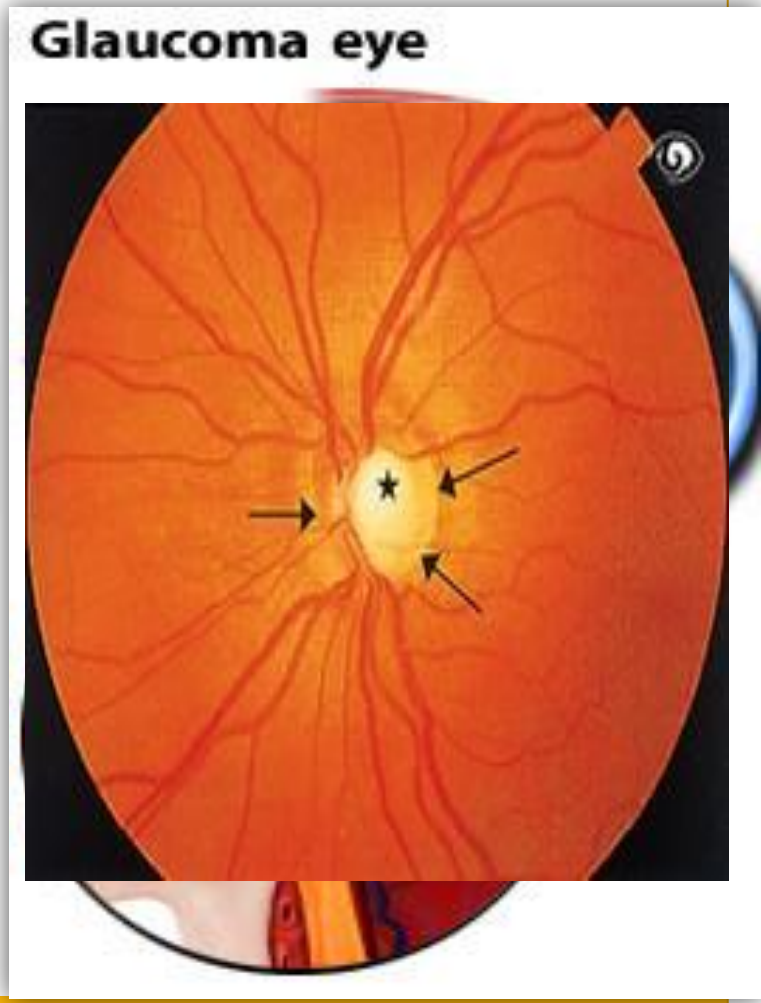
Back pressure on retina

↓

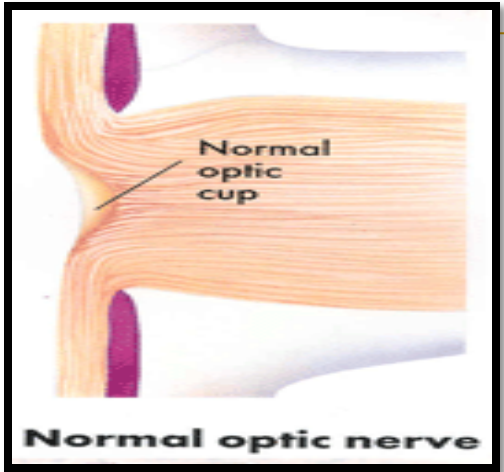
Nerves get damaged.

↓

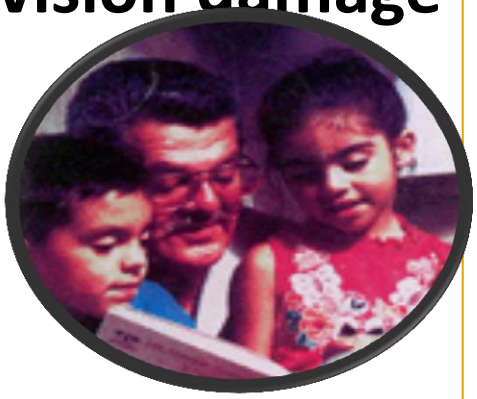
Optic cup becomes more hollow, 1 can't distinguish between optic cup & disk .



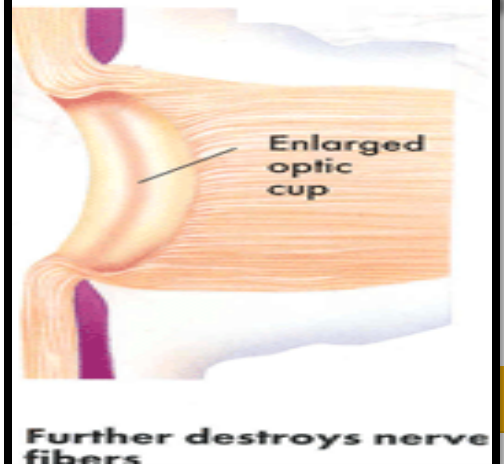
Progression of Glaucoma & vision damage



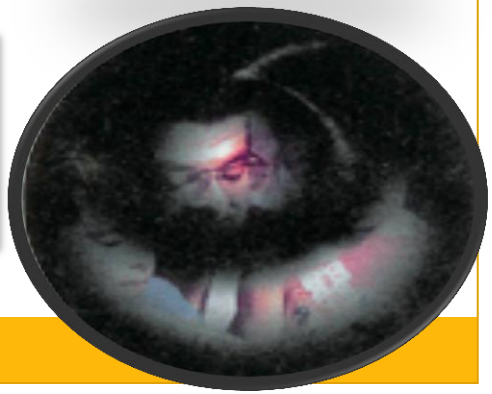
When nerve fibers are healthy, optic cup is small and vision is normal



If nerve fibers are damaged, optic cup enlarges."Side-vision" is reduced but central vision is normal



As more nerve fibres are damaged, optic cup enlarges more."Tunnel vision" & then blindness can occur



Classification of Glaucoma

Congenital (Bupthalmos)- Defect right from birth.

Acquired- develops over time

Primary- has no underlying cause

Secondary- is due to underlying causes like injury, inflammation, use of steroids etc

Primary angle closure glaucoma- Narrow irido corneal angle obstructs outflow of fluid. This increases IOP & hampers optic nerve functioning.

Open angle- Occurs in people above 40 years.

Primary open angle glaucoma (POAG)- Draining channels are blocked due to age related changes

Normal tension/pressure glaucoma (NTG/NPG)

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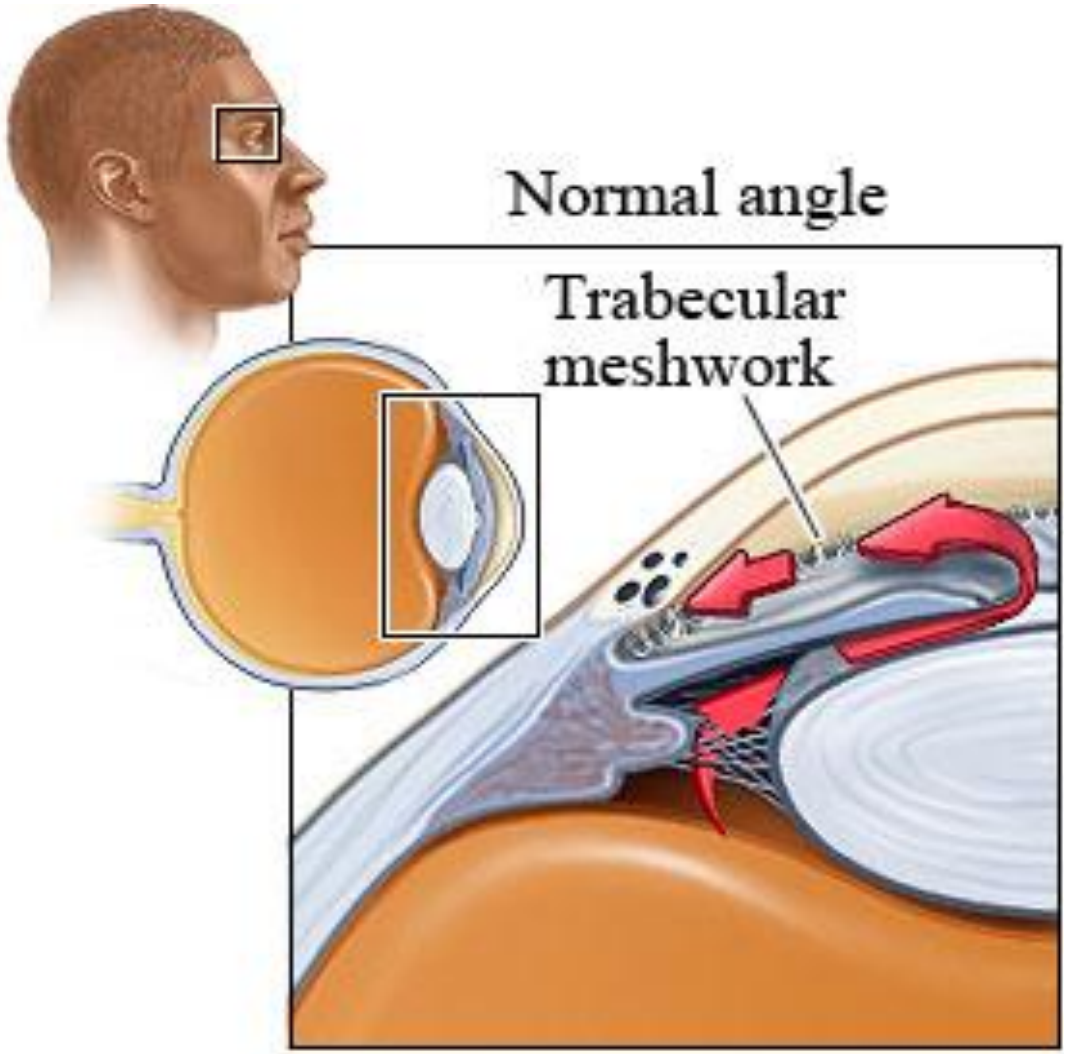

- **Ocular hypertension** (OHT) is intraocular pressure higher than normal in the absence of optic nerve damage or visual field loss.
- Such individuals are called glaucoma suspects.

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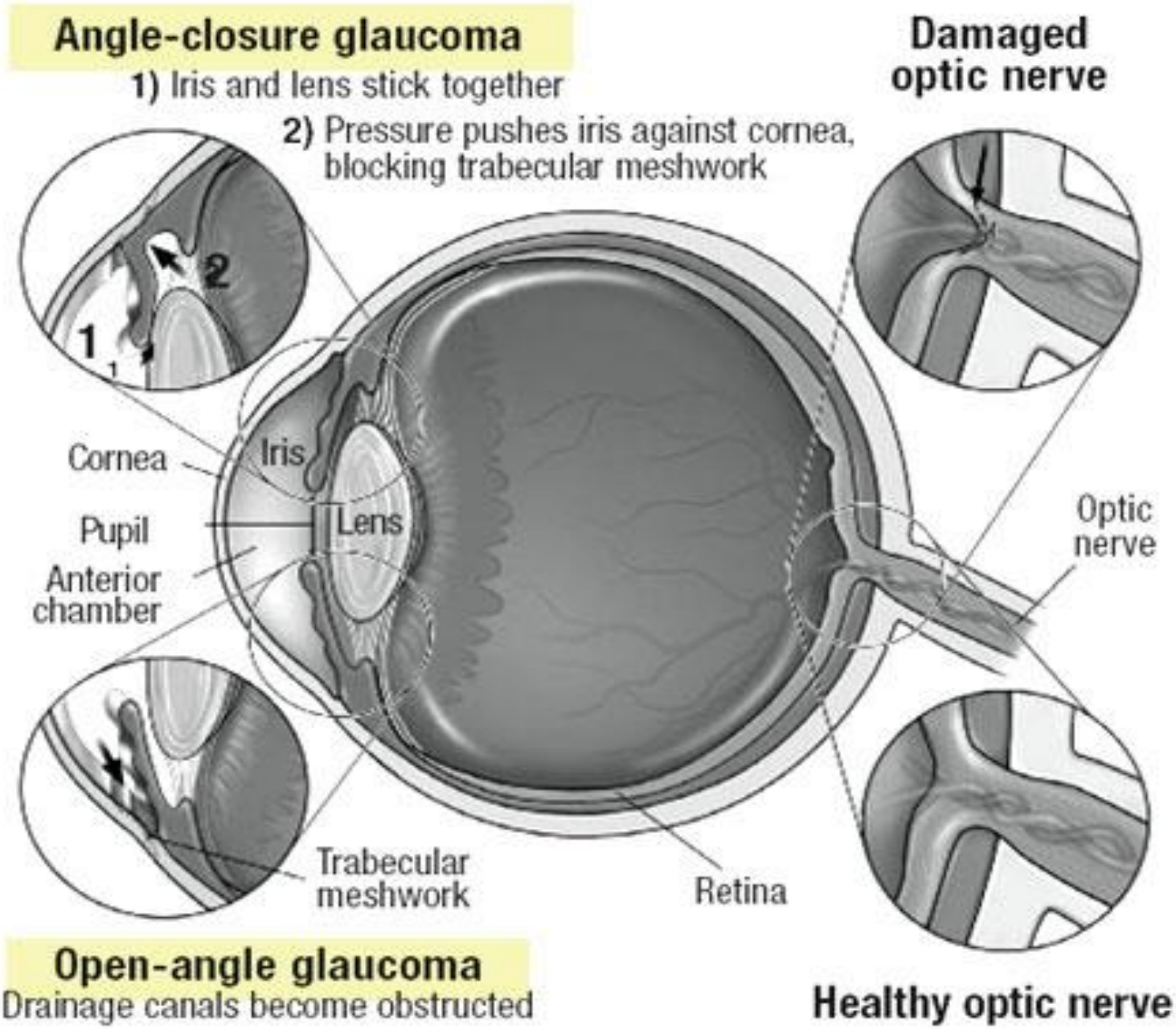

Angle-closure glaucoma
1) Iris and lens stick together
2) Pressure pushes iris against cornea, blocking trabecular meshwork
Damaged optic nerve
2
1
Cornea
Iris
Pupil
Lens
Anterior chamber
Optic nerve
Trabecular meshwork
Retina
Open-angle glaucoma
Drainage canals become obstructed
Healthy optic nerve

Secondary glaucoma

- The most common causes of secondary open angle glaucoma include the following:

✓Treatment with steroids (topical & systemic, asthma inhalers, nasal sprays, and even topical ointments)

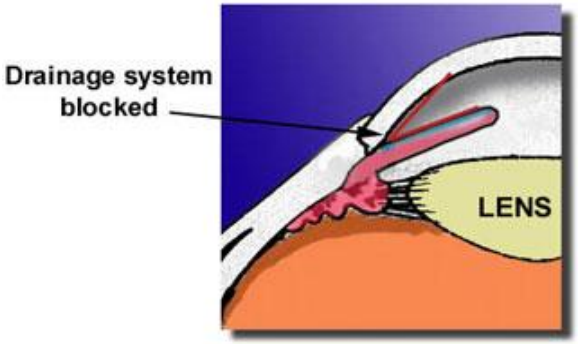
✓Particles blocking trabecular meshwork (malignant cells, red blood cells, inflammatory cells or pigment)

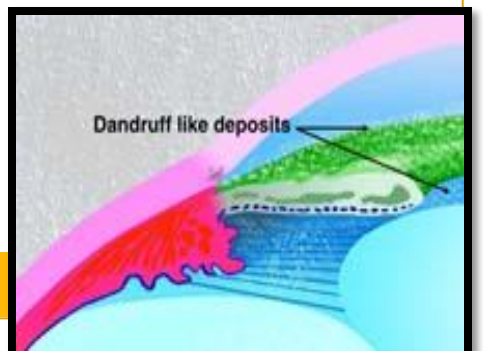
✓Membranes in the anterior chamber angle

✓Trauma to the trabecular meshwork

✓Neovascularisation in the angle, e.g. in diabetics, or after central retinal vein occlusion

✓Pseudoexfoliation syndrome





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Symptoms

Halos-circular rings around light

Pain in eyes

Normal vision

Blurred peripheral vision –mild damage

Tunnel shaped vision-advanced stage

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Signs & symptoms

Open angle glaucoma

- Usually painless, patient is unaware of the condition.
- Visual field loss is only noticed at a late stage, because visual loss is gradual
- Glaucoma patients with bilateral visual field loss have associated decline in motility & driving ability

Angle closure glaucoma

- Painful red eye, headache & frequently, nausea and vomiting.
- Vision is blurred
- Impaired visual acuity
- Pupil is semi-dilated and fixed, with no reaction to light

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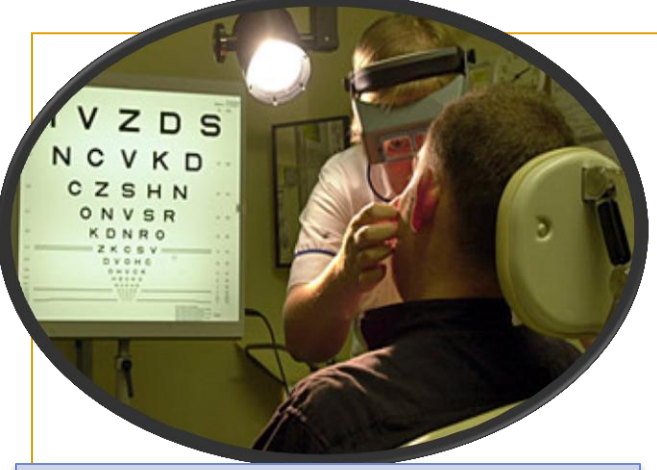


Refraction- test short/long sighted vision .

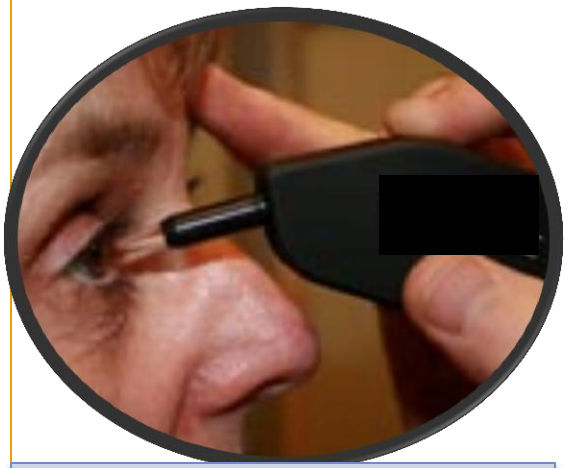
Tonometry- measure IOP.

ophthalmoscope- examine interior of eye. (lens, retina, optic nerve)

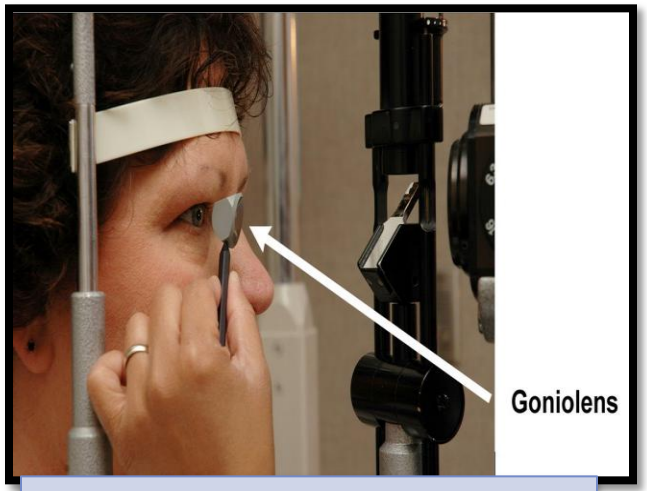
History

Diagnosis

Slit-lamp examination.



Pachymetry-measure corneal thickness.



gonioscopy-measure irido-corneal angle.

Perimetry-measure extent of vision loss.

http://vision.about.com/od/glaucoma/tp/testsforglaucoma.htm0Last accessed 23rd november 2012

Risk factors

Open angle glaucoma

- Higher IOP > 21 mmHg
- Increasing age (< 40 years = rare; 40–60 years = 1%; 60–80 years = 2%; > 80 years = 4%)
- Genetic make-up
- Diabetes mellitus
- Myopia (near-sightedness)
- Vascular factors (migraine; vasospastic disease; hypotension &hypertension)
- Thin corneas
- Retinal diseases

Angle closure glaucoma

- Hypermetropic (far-sighted) patients
- Women (3–4 times higher)
- EHighest incidence: 55–65 years of age
- Genetic make-up

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Management of glaucoma

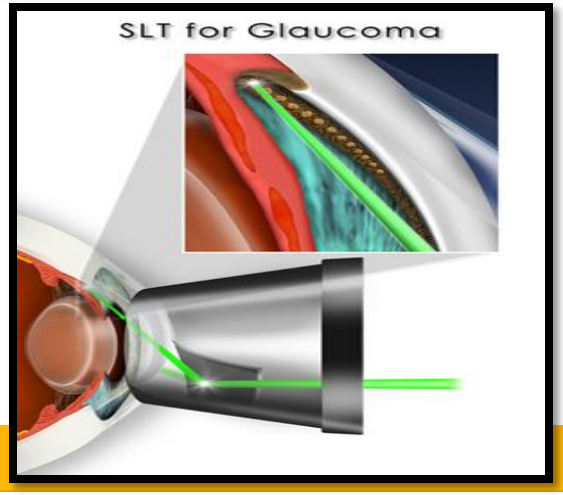
- ✓ **Prevention or modification of risk factors.**
- ✓ **Raised IOP is only modifiable risk factor & primary goal in management.**

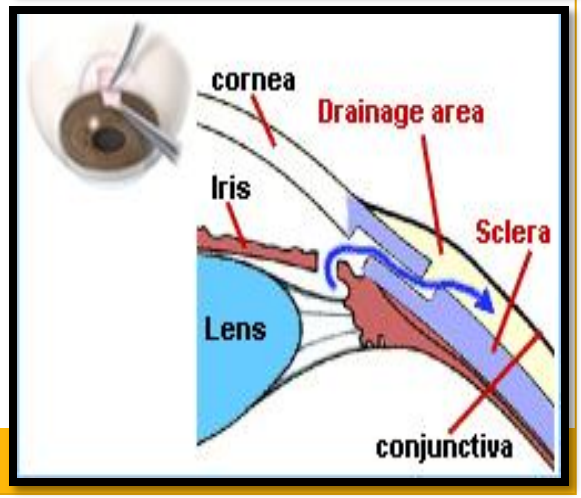
Glaucoma management

Pharmacotherapy – involves administration of various topical anti-glaucoma eyedrops or pills taken orally to lower & control IOP & thereby prevent/delay further damage to eyes.

Laser treatment-Treatment of choice for angle closure & may be preformed for open angle glaucoma. Laser trabeculoplasty, laser peripheral iridotomy & cycloablation are commonly performed laser treatments for glaucoma.

Surgical treatment- Failure of medications & laser treatment to control IOP is an indication for surgery. Trabeculectomy is most common surgery performed for glaucoma.





Treatment algorithm for managing POAG.

Step 4-Surgery

Insufficient fall of IOP
Progression of visual field defects

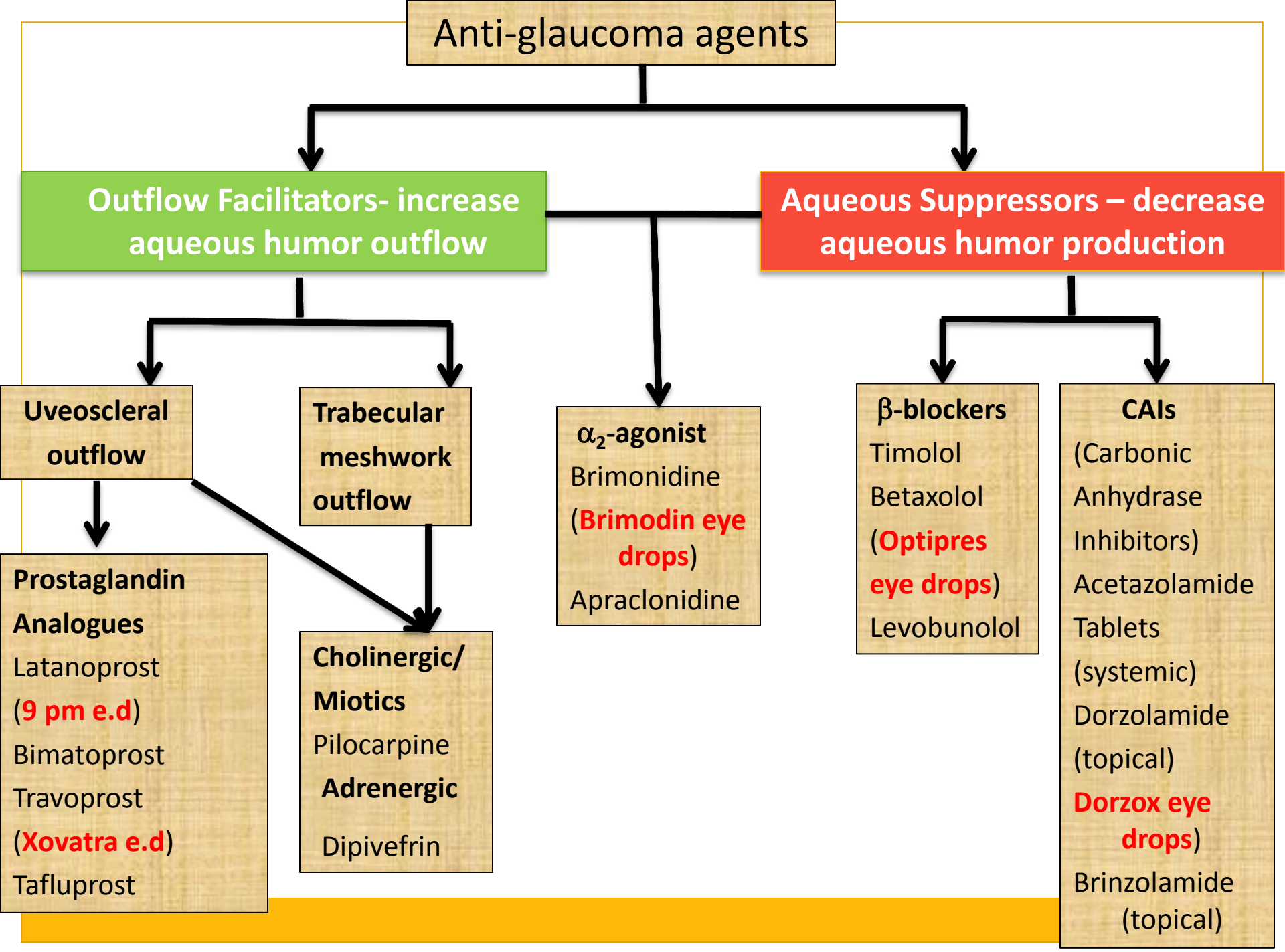
Step 3-Adjunctive/ Add-On Therapy:
Combining anti-glaucoma drugs.
β-blockers + CAIs / PGA/ α_2-agonist

Insufficient fall of IOP
Progression of visual field defects

Step 2-Replacement Monotherapy :
Topical carbonic anhydrase inhibitors (CAIs), prostaglandin analogues (PGA), α_2-agonist

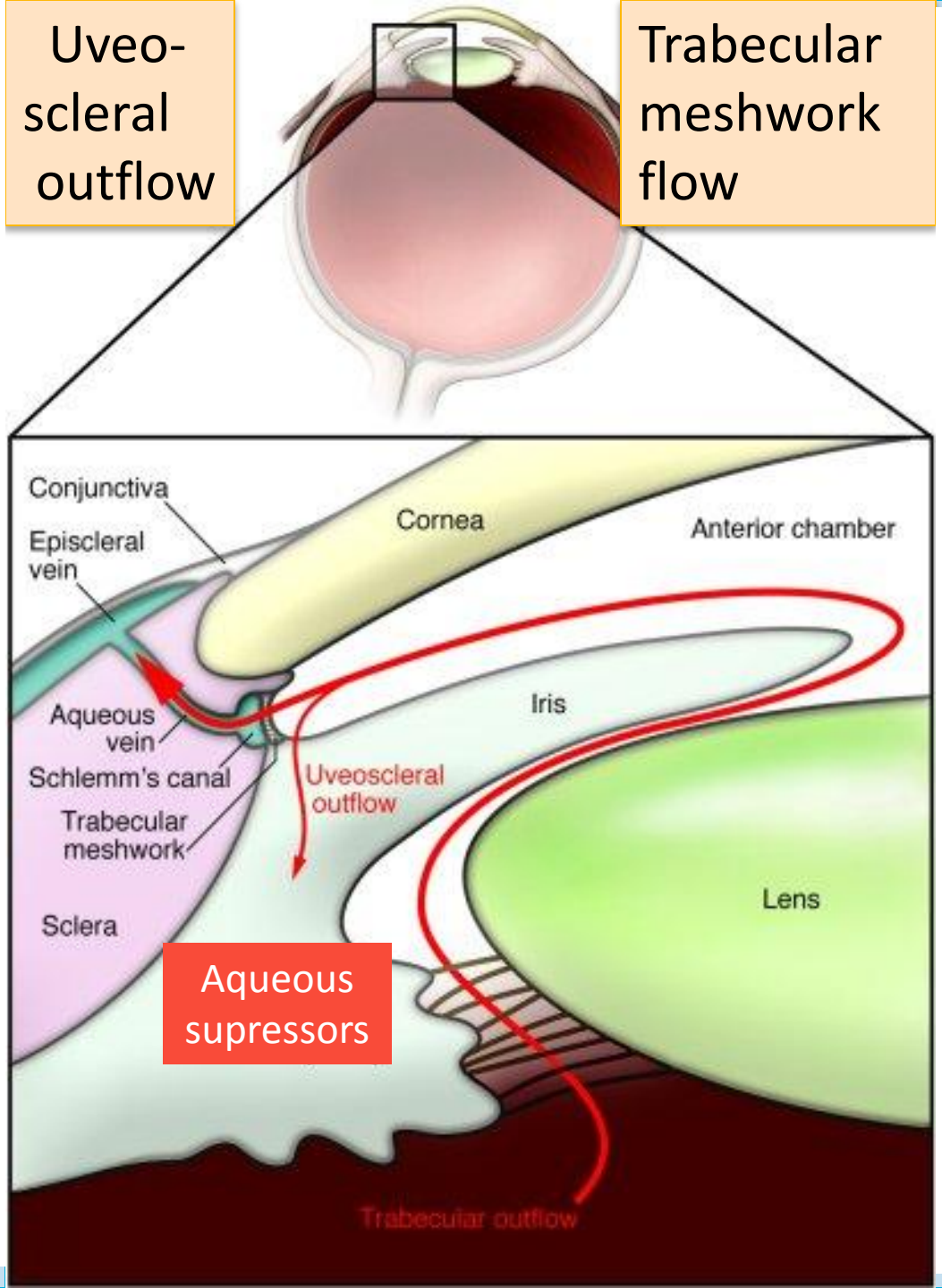
Insufficient fall of IOP
Adverse effects

Step 1-Monotherapy : β-blockers/ prostaglandin analogues (PGA)


Anti-glaucoma agents
Outflow Facilitators- increase aqueous humor outflow
Aqueous Suppressors – decrease aqueous humor production
Uveoscleral outflow
Trabecular meshwork outflow
α2-agonist
Brimonidine
(Brimodin eye drops)
Apraclonidine
β-blockers
Timolol
Betaxolol
(Optipres eye drops)
Levobunolol
CAIs
(Carbonic Anhydrase Inhibitors)
Acetazolamide Tablets (systemic)
Dorzolamide (topical)
Dorzox eye drops)
Brinzolamide (topical)
Prostaglandin Analogues
Latanoprost
(9 pm e.d)
Bimatoprost
Travoprost
(Xovatra e.d)
Tafluprost
Cholinergic/ Miotics
Pilocarpine
Adrenergic
Dipivefrin

Uveo-scleral outflow

Trabecular meshwork flow



Aqueous supressors

Currently available drugs

Drug class	Drug and daily frequency	Route	Mechanism of action	Side effects in class
Prostaglandin analogs	Latanoprost 1x Travoprost 1x Unoprostone 2x Bimatoprost 1x	Topical	Increased trabecular drainage	Eye lash thickening, eye lid darkening, eye staining
Beta blockers	Betaxolol 2x (selective) Carteolol 2x Timolol 1–2x Levobunolol 1–2x	Topical	Decreased aqueous fluid production	Eye irritation, hyperemia, blurred vision, impaired lung function
Diuretics (carbonic anhydrase inhibitors)	Brinzolamide 3x Dorzolamide 3x Acetazolamide 2–4x Methazolamide 2–3x	Topical Oral	Decreased aqueous fluid production via HCO_3^- unavailability	Blurred vision, bitter taste, acidosis, hepatic necrosis
Cholinomimetics	Carbachol 3x Pilocarpine 3–4x Physostigmine 1–4x	Topical DOG Plastic film	Open the TM by contraction of ciliary muscle forces	Night blindness, blurred vision, burning eye sensation
Alpha agonists (selective)	Epinephrine 1–2x Dipivefrin 2x	Topical	Increased trabecular flow	Tremor Palpitation
Alpha agonists (non-selective)	Brimonidine[a] 3x Apraclonidine[b] 3x	Topical	Reduced aqueous production and increased uveoscleral flow	Hyperemia, allergic conjunctivitis, itching, lacrimation

Notes: [a]Brimonidine has a dual mechanism of action; [b]apraclonidine only reduces aqueous production.
Abbreviations: POAG, primary open-angle glaucoma; DOG, drops, ointment, gel.

Combining Anti-Glaucoma drugs

- IOP is not adequately regulated with monotherapy.
- Causes-

1. Initial insufficient effect of drug.
2. Development of tolerance during long term therapy.
3. Or by progress of the disease.

The idea of combining two or more drugs is to produce the additivity of desired therapeutic effect but not of the side effects

2. Fewer daily drops than concomitant therapy
3. Convenient dosing regimen
4. Risk of elimination of first drop from conjunctival sac by instillation of second drops is completely eliminated.
5. Ensures increased patient convenience & compliance
6. Improves therapeutic efficacy.

Am J Ophthalmol 2000, 130:832-833

Approved Combinations : Currently Available

- Dorzolamide + Timolol (DORZOX-T)
- Latanoprost + Timolol (LATIM)
- Brimonidine + Timolol (BRIMOCOM)
- Travoprost + Timolol
- Bimatoprost + Timolol

Adjuncts

First line agents

Cipla's complete glaucoma basket

Fixed dose combination

Latest technologies in glaucoma

- Diagnosis-Fourier domain (FD) OCT, continuous IOP monitoring system (contact lens), scanning laser polarimetry, short wavelength automated perimetry
- Genetics- genetic screening, genetic counselling, gene therapy
- Medication-preservative free unims of anti –glaucoma drugs (tafluprost, dorzolamide/ timolol FDC)
- Future drugs- Bis(7)- tacrine, vitamin E, N-actylcystiene & other anti-oxidants,Mirtogenol,Erythropoietin, nimodipine, new topical ophthalmic drug delivery device
- Surgeries- Micropulse Laser Trabeculoplasty (MLT), Canaloplasty, Trabectome surgery, Ex-Press Mini Shunt, Glaucoma drainage devices (GDD).

http://www.revophth.com/content/d/glaucoma_management/c/27665/
Cinical ophthalmology.2012;6:1699-1707.
http://www.glaucoma.org/treatment/the-future-of-glaucoma-surgery.php

Role of GP in glaucoma diagnosis & treatment-

FAMILY PRACTITIONER IS OFTEN

THE FIRST LINE OF MEDICAL CONTACT WITH THE PATIENT.

Role of GP in glaucoma diagnosis & treatment-

- Recommend all patients above 40 yrs of age to undergo routine eye examination once in a year.
- Possess a basic knowledge of glaucoma medications and their systemic and local side effects
- Recognise & refer to an ophthalmologists in case of-

➢ Clinical manifestations of glaucoma- acute attack of angle closure glaucoma

➢ Symptoms progression or treatment side effects for reassessment .

➢ Previous glaucoma surgery (even years before), & who presents with a red eye or signs of infection, to exclude blebitis or endophthalmitis.

➢ Risk factors - hypertension, diabetes, migraine, steroid use, previous eye trauma, family history & age.

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http://www.glaucoma.org.nz/files/challenge-of-glaucoma.pdf

Role of GP in glaucoma diagnosis & treatment

- When referring to ophthalmologist, provide patients complete medical history & systemic medications if any.
- Monitor therapy: Avoid steroid use in glaucoma patients
- Before prescribing any systemic medication it is important to know his topical medical therapy to prevent drug-drug interaction.
- Review repeat prescriptions for medical schemes, preferably 6 monthly.
- Provide support, encouragement & counselling to the patient, because of the chronic course of the disease. Discuss the impact of visual loss with patients & their families especially for advanced glaucoma cases .
- Educate patients about screening of family members, follow-up visits, and use of medication.

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Any questions?

THANK YOU