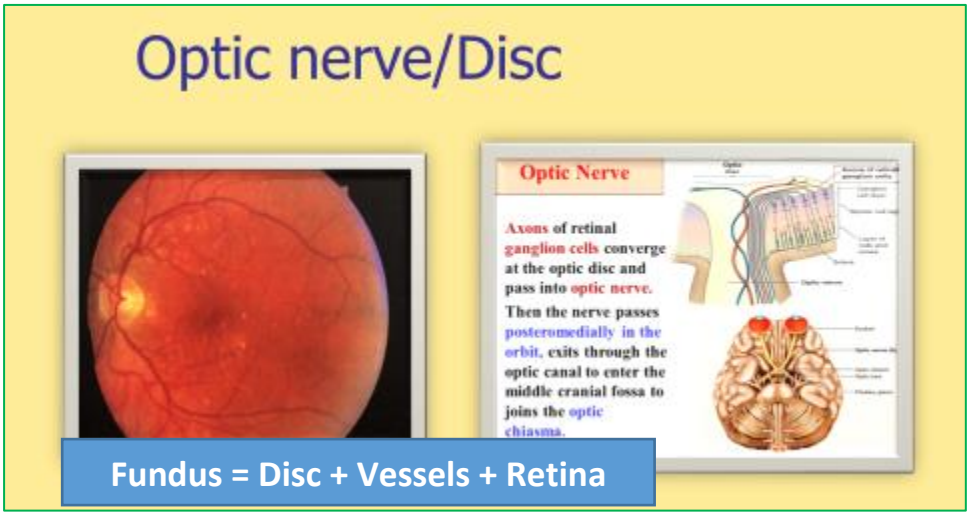
2.4 Glaucoma

Plan

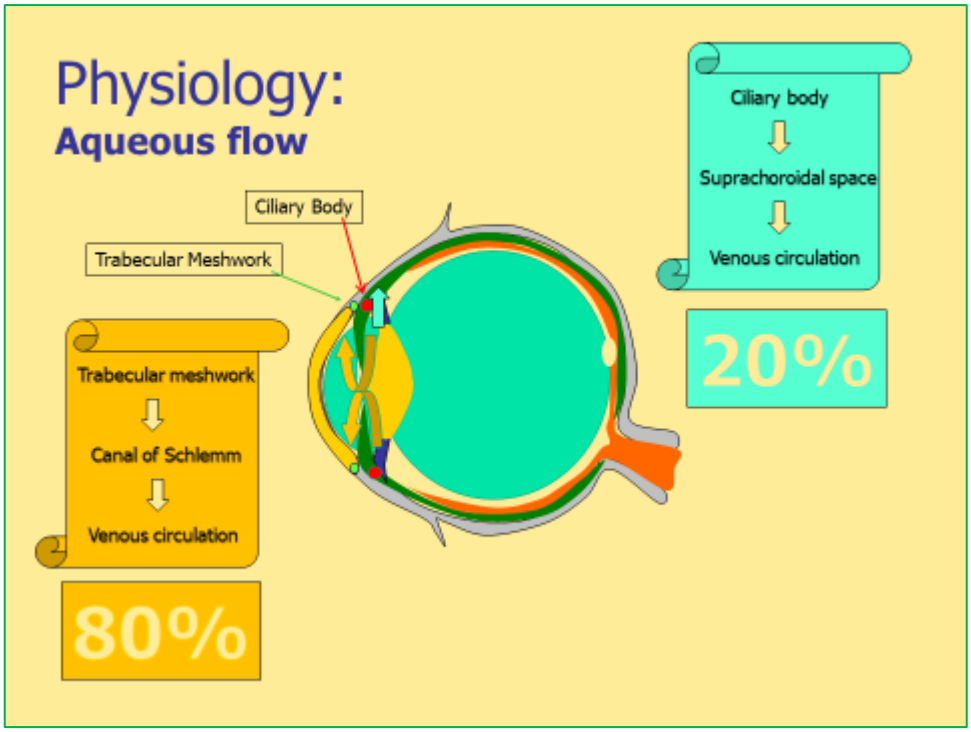
Anatomy & physiology
Definition
Examination/Investigation
Pathogenesis
3 steps: How to diagnose glaucoma
    Is this glaucoma
    Is this open angle or close angle
    Is this primary or secondary
Glaucoma/No glaucoma/Glaucoma suspect/Normal tension glaucoma/Ocular hypertension
Open angle glaucoma: Three step plan
    Mechanism, Treatment, Classification
    Primary open angle glaucoma
    Secondary open angle glaucomas
Angle closure glaucoma: Three step plan
    Mechanism, Treatment, Classification
    Primary angle closure glaucoma
    Secondary angle closure glaucomas
Miscellaneous glaucomas

Anatomy & physiology

Optic disc is visible part of optic nerve.



Physiology: Aqueous formed in ciliary body, comes through pupil in to anterior chamber. Drainage is 80% trough trabecular meshwork and 20% through suprachoroidal space.



Definition

Glaucoma is an optic neuropathy which is characterized by
1 high intraocular pressure, 2 increased cup disc ratio and 3 nerve fiber damage.

Examination & Investigations

1. High intra ocular pressure (IOP).

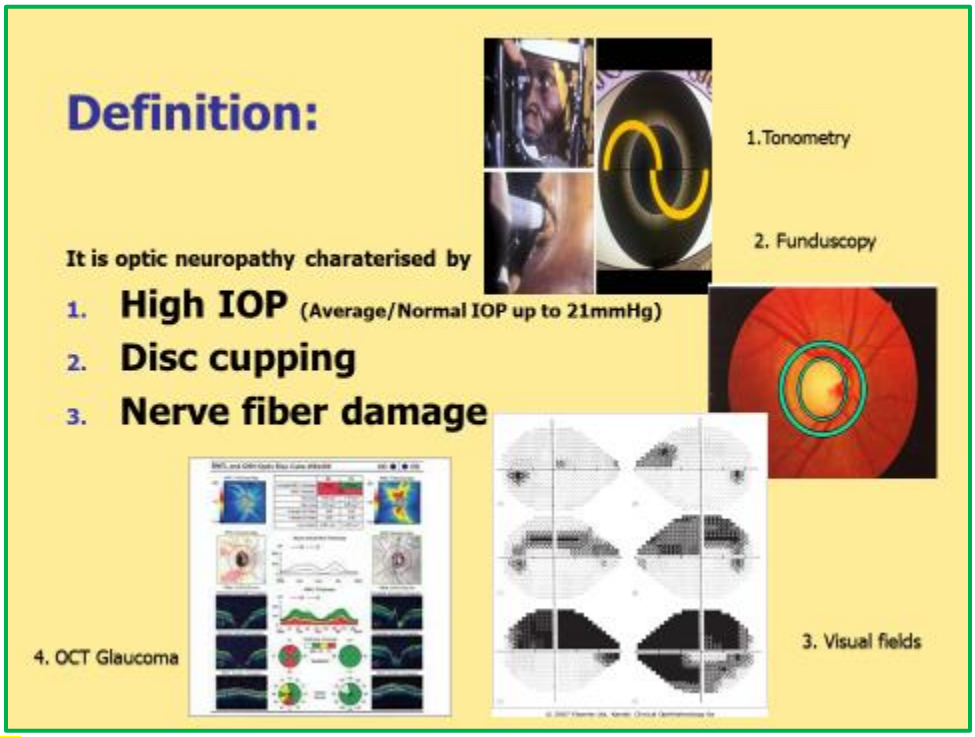
Normal IOP is 21mmHg. Normal IOP is actually average IOP so people with >21IOP may be normal while people with <21 IOP may have glaucoma. . IOP is measured with the help of tonometers. We have different tonometers and all of them measure IOP. Different tonometers are Goldmann tonometer which is attached to slit lamp and is gold standard. Other tonometers are Schiotz tonometer, perkins tonometer and air puff tonometer and tonopen.

2. Disc cupping

Optic disc is part of optic nerve which is visible on fundoscopy. There is a central depression in optic disc which we name optic disc cup. Size of cup is usually 30% of size of optic and because of 30% we describe it as cup disc ration as 0.3% (CD 0.3). when cup size increases to 40% size of optic disc then CD is 0.5, when cup size increases to 50% of disc size then CD is 0.5 and so on.

3. Nerve fiber damage

Nerve fibers are damaged in glaucoma and nerve damage can be assessed with visual fields and optic coherence tomography (OCT).



Remember:

Glaucoma is not like systemic hypertension in which high blood pressure means systemic hypertension. Not every high IOP is glaucoma and not every normal IOP is non-glaucoma.

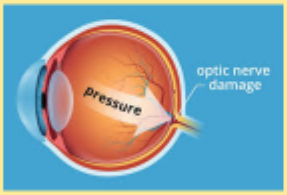
Pathogenesis

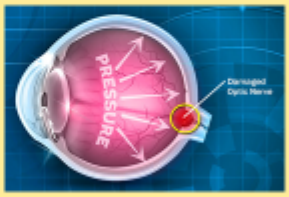
Exact mechanism is not known but
According to ischemic theory increased intraocular pressure puts pressure on optic nerve which reduces blood supply and hence causes ischemia
AND
According to mechanical theory increase intraocular pressure directly damages ganglion cells and eventually killing them.

Pathogenesis of Glaucomatous damage

- **Ischemic theory:**

It postulates that with elevated IOP,reduced blood flow to the optic nerve which starves the cells of oxygen and nutrients





- **Direct mechanical theory:**

It suggest that chronically raised IOP distorts the lamina cribrosa,crimping the axons of retinal ganglion cells as they pass through the lamina cribrosa and eventually killing the cells

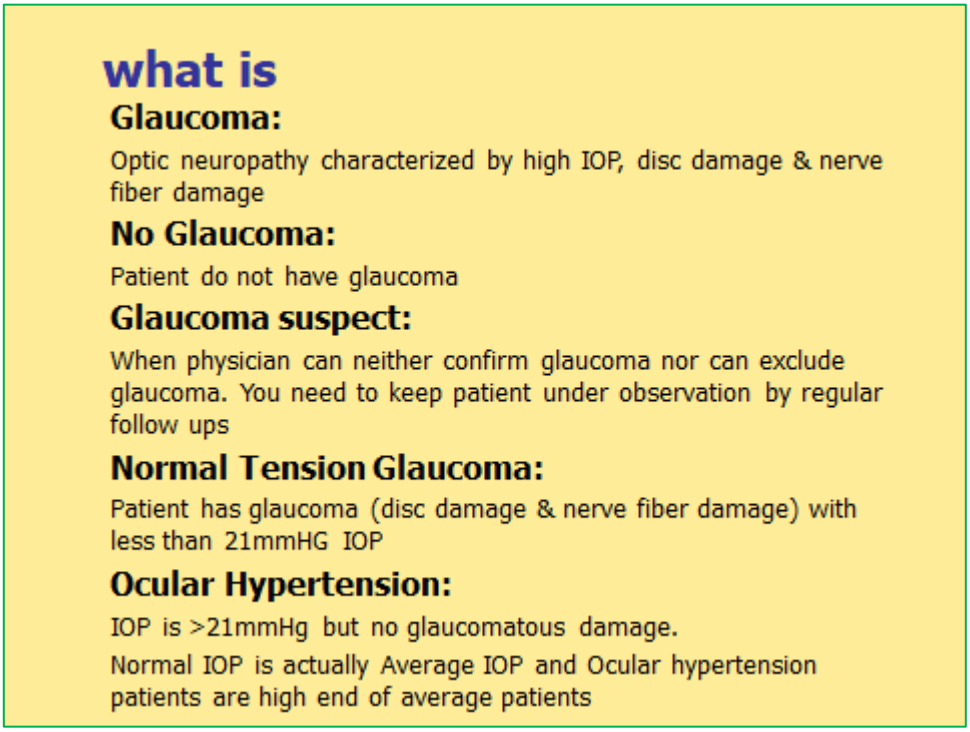
Three step approach to diagnose type of glaucoma

Next is three step plan to diagnose to confirm glaucoma and type of glaucoma.
Step 1. First decide is this glaucoma, glaucoma suspect, ocular hypertension or normal tension glaucoma
Step 2. If glaucoma confirmed in step 1 then decide it is open angle or close angle glaucoma
Step 3. Is this primary or secondary glaucoma

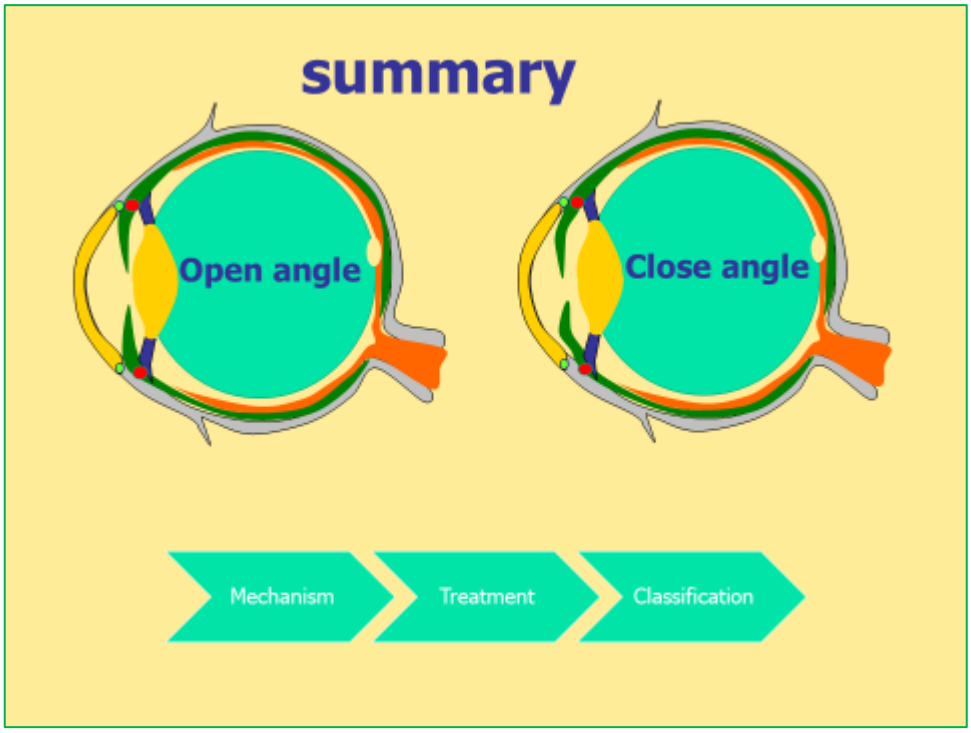
Step 1

First of all decide whether patient

- Has glaucoma
- Do not have glaucoma
- Suspect: may or may not have glaucoma
- Has normal tension glaucoma
- Has ocular hypertension



Step 2 **Is glaucoma open angle or close angle?**



Step 3 **Is glaucoma primary or secondary?**

If glaucoma is without predisposing cause then we call it primary glaucoma but if another condition is responsible then it is called secondary glaucoma.

Primary OR secondary open angle glaucoma: if patient has glaucoma with open angle and unknown etiology then it is called primary open angle glaucoma (POAG) or chronic simple glaucoma (CSG). If patient has glaucoma with open angle and there is underlying pathological process which is blocking trabecular meshwork then we call it secondary open angle glaucoma.
Primary OR secondary angle closure glaucoma: If patient has glaucoma with close angle and unknown etiology then it is called primary angle closure glaucoma or angle closure glaucoma (ACG). If patient has glaucoma with close angle and there is condition causing angle closure then we call it secondary angle closure.

Now first we will discuss

1. Open angle glaucoma in general then
2. Primary open glaucoma then
3. Secondary open angle glaucomas then
4. Close angle glaucoma in general then
5. Primary angle closure glaucoma then
6. Secondary angle closure glaucomas then
7. Miscellaneous glaucomas

1 Open angle glaucoma

Open angle glaucomas is a group of glaucomas in which patient has glaucoma (High IOP, increased CD ratio and nerve fiber damage on Visual fields & OCT).

Mechanism:

In POAG exact mechanism is not known but it is postulated that increased resistance in trabecular meshwork reduces drainage of aqueous humour and hence high IOP.

In secondary open angle glaucoma pseudoexfoliation material, pigment, inflammatory cells or red blood cell block trabecular meshwork which produces resistance to putflow and hence high IOP.

Types:

If patient has glaucoma (step1) and angle is open (step2) then step 3 is to determine whether open angle glaucoma is primary open angle glaucoma (POAG) or secondary open angle glaucoma.

If cause of open angle glaucoma is not known then we call it primary open angle glaucoma.

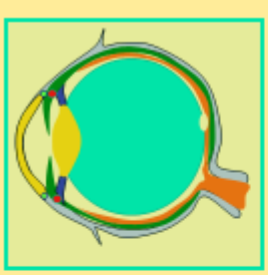
If trabecular meshwork is blocked because of pigment, pseudoexfoliation, red blood cells or inflammatory cells then we call it secondary open angle glaucoma.

If trabecular meshwork blocked because of pigment it is called pigmentary glaucoma.

If trabecular meshwork blocked because of exfoliation it is called pseudoexfoliation glaucoma.

If trabecular meshwork blocked because of blood it is called ???? glaucoma.

If trabecular meshwork blocked because of inflammation it is called inflammatry glaucoma.



	POAG or CSG	Secondary OAG
cause	unknown	-Pigment dispersion glaucoma -Pseudoexfoliation glaucoma -Inflammatory exudate -Blood cells
Mechanism	Increase resistance in trabecular meshwork	Blockage of trabecular meshwork causing resistance
Medical treatment	First medical treatment	First medical treatment
Laser treatment	Augment by laser	Augment by laser
Surgical treatment	Last option	Last option

Treatment open angle glaucoma

Main reason for increased IOP is increase resistance in trabecular meshwork but main treatment is reducing production of aqueous and increasing uveoscleral outflow.

Reducing aqueous production:

Beta blockers: Timolol, Betaxolol, Levobunolol

Carbonic anhydrase inhibitors: Dorzolamide, Brinzolaamide

Adrenergic agonists: Bromidine

Increasing outflow:

Parasympethomimetics: Pilocarpine; opens spaces in trabecular meshwork

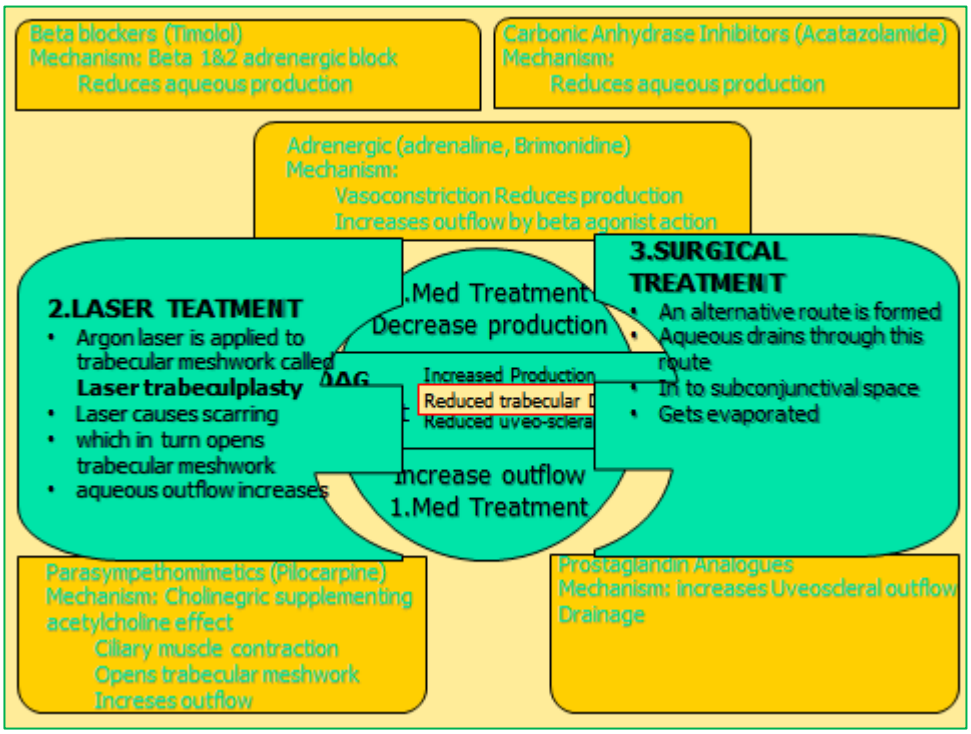
Prostaglandin analogues: Latanopost, Travoprost

Laser treatment:

Argon laser trabeculoplasty:

Surgery:

Trabeculectomy



2 Primary open angle glaucoma

Glaucoma with open angle and unknown etiology

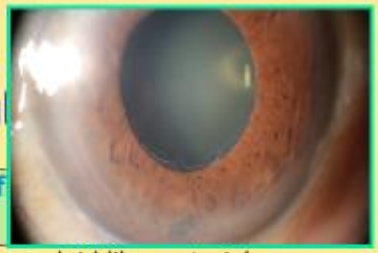
Primary Open angle Glaucoma (POAG)
Chronic Simple Glaucoma (CSG)

- **High IOP, Disc cupping, Nerve fiber damage**
- **Open angle & No known cause**
- **Incidence:** 6th decade onwards, Both genders, usually bilateral
- **Risk factors:** Age, Family history, Myopia, Black race, Diabetes
- **Etiology:** Genetic bases; Family members at more risk
- **Mechanism:** Increased resistance in trabecular meshwork
- **Treatment:** Medical ➡ Laser trabeculoplasty ➡ Trabeculectomy
- **Associated ocular pathologies:** Myopia, Retinitis Pigmentosa, Retinal vesesles occlusion, Retinal detachment, Fuchs' endothelial dystrophy

3 Secondary open angle glaucoma Pseudoexfoliation glaucoma

Glaucoma with open angle and known cause

Secondary open angle:
Pseudo exfoliation g



- **High IOP, Disc cupping, Nerve f**
- **Open angle & cause is PXF**
- Pseudo exfoliative (PXF) material is amyloid like material
- PXF gets deposited on all ocular structures
- PXF material deposited in angle blocks trabecular meshwork
- **Diagnosis:**
  - Dandruff like PXF material on lens, pupil and angle
  - Open angle
  - Increase IOP, Disc cupping and nerve fiber damage
- **Treatment:** Medical ➡ Laser trabeculoplasty ➡ Trabeculectomy

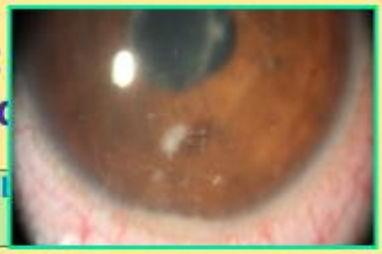
Secondary open angle glaucoma
Pigmentary glaucoma

Secondary open angle:
Pigmentary glauoma

- **High IOP, Disc cupping, Nerve fil**
- **Open angle & cause is pigment d**
- Rubbing of iris posterior pigment layer against zonules
- Release of iris pigment cells from iris pigment epithelium
- Pigment cells block trabecular meshwork
- IOP increases
- **Diagnosis:**
  - Pigment cells on lens, endothelium and angle
  - Iris transillumination defects because of loss of pigment
  - Open angle
  - Increase IOP, Disc cupping and nerve fiber damage
- **Treatment:** Medical ⇨ Laser trabeculoplasty ⇨ Trabeculectomy

Secondary open angle glaucoma
Inflammatory glaucoma

Secondary open angle:
Inflammatory glauc



- **High IOP, Disc cupping, Nerve fil**
- **Open angle & cause is uveitis**
- Patient diagnosed as uveitis
- Inflammatory cells block trabecular meshwork
- Glaucoma resolves when uveitis treated
- **Diagnosis:**
  - Signs of uveitis
  - Open angle
  - Increase IOP, Disc cupping and nerve fiber damage
- **Treatment:** Medical ⇨ Laser trabeculoplasty ⇨ Trabeculectomy

4 Close angle glaucoma

Mechanism: Primary angle closure glaucoma

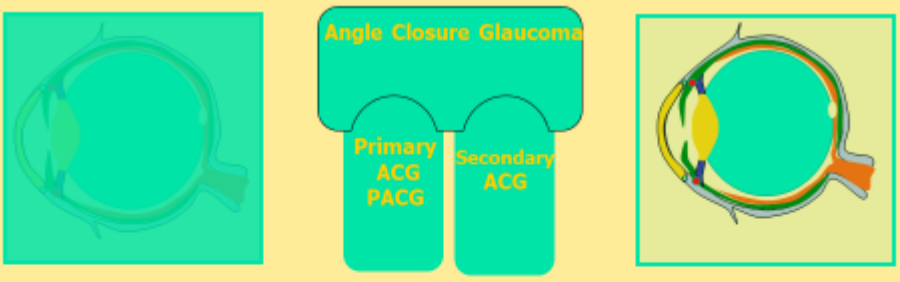
In primary angle closure glaucoma peripheral iris bows forward thus blocking anterior chamber angle

Mechanism: Secondary angle closure glaucoma

In secondary angle closure glaucoma is either with pupil block or without pupil block. In pupil block lens or posterior synechiae blocks pupil and causes iris to bow forward thus causing secondary angle glaucoma. In without pupil block fibrovascular membrane pulls peripheral iris forward and blocks anterior chamber angle thus causing secondary angle closure glaucoma.

Types of angle closure glaucoma: Primary or Secondary

If patient has glaucoma (step1) and angle is closed (step2) then step 3 is to determine whether angle closure is primary angle closure glaucoma or secondary angle closure glaucoma.
If cause of angle closure glaucoma is without any predisposing cause then we call it primary angle closure glaucoma.
If cause of angle closure is pupil block (swollen lens, dislocated lens, posterior synechiae or anterior chamber IOL) or without pupil block by pulling iris forward (fibrovascular membrane or anterior synechiae) then we call it secondary angle closure glaucoma.



	PACG	Secondary ACG
Cause	-Small eye -Forward movement of lens with age	- With pupil block (swollen/ dislocated lens, synechiae) - Without pupil block (fibrovascular membrane)
Mechanism	-Pupil block -Angle closure	Less drainage because of closed angle
Medical Treatment	Miotics to relieve pupil block Decrease aqueous production	Treat the cause
Laser treatment	Peripheral iridotomy to relieve pupil block	Laser may work
Surgical treatment	Trabeculectomy	Trabeculectomy

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Primary angle closure glaucoma

Primary Angle Closure Glaucoma

- **High IOP, Disc cupping, Nerve fiber damage**
- **Close angle & No known cause**
- **Incidence:** 5th decade onwards, Female 4 times more, usually bilateral
- **Risk factors:** Hypermetropia
- **Mechanism:** small eye with lens blocking pupil
- **Treatment:** Medical ⇨ Laser iridotomy ⇨ Trabeculectomy

Stages of Primary Angle Closure Glaucoma (PACG)

- PACG suspect
  - Predisposed to ACG
  - No symptoms
  - Treatment: YAG laser iridotomy
- Intermittent PACG
  - Symptoms: Intermittent blurry vision with halos
  - Treatment: Prophylactic peripheral iridotomy
- Acute PACG (see next slide)
  - Symptoms: Severe pain, Halos, Red eye, Decrease vision
  - Treatment Of acute attack: Reduce pressure
  - Permanent treatment: Laser iridotomy or trabeculectomy
- Chronic PACG (angle closes gradually and permanently)
  - Symptoms: Loss of vision and intermittent pain
  - Treatment: Medical treatment & trabeculectomy

Acute PACG

- Physiological pupil dilation causes pupil block which results in bowing of iris peripherally and hence angle closure
- Symptoms: acute severe pain, vomiting, redness, loss of vision
- Signs: High IOP, corneal edema, redness, shallow anterior chamber, fixed mid-dilated pupil

Treatment:

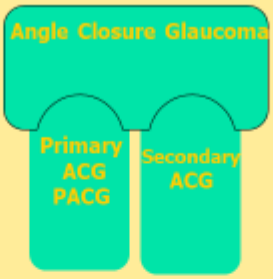
- Treatment of acute attack:
  - Systemic acetazolamide or mannitol intravenously
  - Pilocarpine to constrict pupil (to relieve pupil block) & drop
  - Other topical treatment to reduce pressure
  - Peripheral iridotmy
- Treatment for future attacks:
  - Peripheral iridotomy or trabeculectomy
- Treatment of fellow eye:
  - Piolcarpine drop to prevent attack and then Peripheral iridotomy

Mechanism: Secondary angle closure glaucoma With pupil block

In secondary close angle glaucoma **with pupil block** lens or posterior synechiae block pupil. This in turn blocks circulation of aqueous and aqueous accumulates behind iris causing peripheral iris to bow forward and cause secondary angle closure glaucoma.

Mechanism: Secondary angle closure glaucoma Without pupil block

In secondary close angle glaucoma **without pupil block** peripheral iris is pulled forward because of abnormal fibrovascular tissue. This blocks anterior chamber angle and causes secondary angle closure glaucoma.



With pupil block	Without pupil block
Swollen lens	Fibrovascular membrane
Dislocated lens	Anterior synechiae
Posterior synechiae	
Anterior chamber IOL	

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Secondary angle closure glaucoma With pupil block

Secondary close angle
With pupil block

Following conditions block pupil. Increased pressure in posterior chamber pushes peripheral iris to block angle

- 360 degree posterior synechiae can block pupil
- Subluxated lens blocks pupil
- Mature cataract can swell up to block pupil
- Anterior chamber implant can block pupil

Secondary angle closure glaucoma Without pupil block

Secondary close angle
Without pupil block

Following conditions cause adherence of peripheral iris to back of cornea to block angle

- Contraction of fibro vascular membrane
- Peripheral anterior synechiae

Treatment: angle closure glaucoma

Main reason for increased IOP is closed anterior chamber angle making it difficult to pass aqueous from posterior to anterior chamber. Medical treatment has role but main treatment is making alternative route by YAG laser iridotomy and trabeculectmy.

Reducing aqueous production:

Beta blockers: Timolol, Betaxolol, Levobunolol

Carbonic anhydrase inhibitors: Dorzolamide, Brinzolaamide

Adrenergic agonists: Bromidine

Increasing outflow:

Parasympethomimetics: Pilocarpine; opens spaces in trabecular meshwork

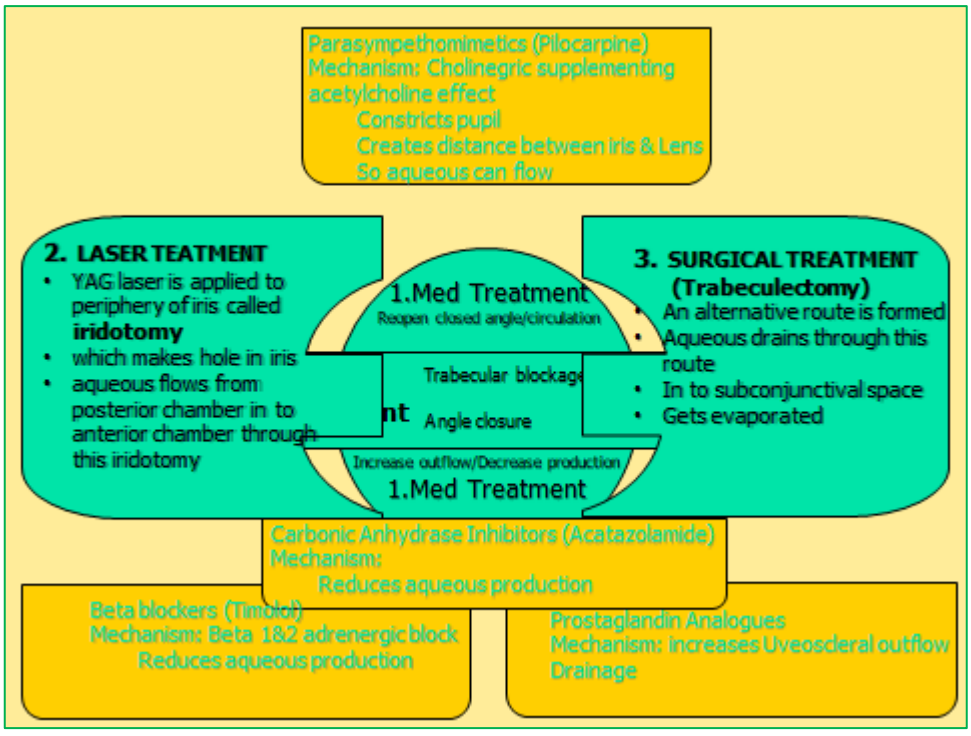
Prostaglandin analogues: Latanopost, Travoprost

Laser treatment:

YAG laser iridotomy: Hole is made in peripheral iris with YAG laser

Surgery:

Trabeculectomy: Alternative route made for aqueous drainage



7 Miscellaneous glaucomas

Miscellaneous glaucomas:
Congenital glaucoma

- Increased IOP + no associated eye anomaly
- Maldevelopment of anterior chamber angle causes increase IOP
- Mostly bilateral and males

1- True congenital Glaucoma: IOP increased before or at birth

2- Infantile glaucoma: IOP increase before age 3 years

3- Juvenile glaucoma: IOP increase between age 3 to 16years

- Symptoms:
  - Buphthalmos (enlarged eye ball)
  - Large & Cloudy cornea
  - Lacrimation, photophobia
- Signs:
  - High IOP
  - Hazy & edematous cornea due to endothelial damage
  - Large corneal diameter: >12mm
- Treatment:
  - Medical therapy not usually enough
  - Surgery
    - Goniotomy (Incision in anterior chamber to open it)
    - Trabeculotomy (Trabecular meshwork excised) OR Trabeculectomy

Miscellaneous glaucomas:
Lens induced glaucoma

- **Phacolytic glaucoma:**
  - Hypermature cataract release protiens
  - Protiens block trabecular meshwork to produce secondary open angle glaucoma
- **Phacomorphic glaucoma:**
  - Mture cataract swells up and blocks pupil
  - Result is secondary angle closure glaucoma
- **Phaco pupil block:**
  - Dislocated lens blocks pupil
  - Produces secondary angle closure glaucoma

Miscellaneous glaucomas:
Neovascular glaucoma

- Causes: CRVO, BRVO, Eales disease, CRAO, Diabetic retinopathy
- Pathogenesis: Hypoxic retina produces angiogenic factors mainly VEGF. This VEGF induces retinal and anterior segment new vessels
- Mechanism:
  - Secondary open angle: Initially neovascular blocks an open angle
  - Secondary close angle: Later membrane contracts to close angle
- Treatment:
  - Anti VEGF intravitreal injections to arrest pathogenesis
  - Argon laser photocoagulation (PRP) to arrest pathogenesis
  - Topical medical treatment
  - Trabeculectomy
  - Cyclodestruction (cryo or laser to destroy part of ciliary body)

Miscellaneous glucomas:
Absolute glaucoma

- End stage of all open and close angle glaucomas
- More commonly after neovascular and inflammatory glaucomas
- Because of persistent elevated pressure eye becomes hard, blind and painful
- Symptoms & signs:
  - Hard & painful eye
  - No vision
  - Fixed dilated pupil
  - Secondary glaucomatous optic atrophy
- Treatment
  - Treat cause like Anti-VEGF for new vessels
  - Medical treatment
  - Surgery
  - Cyclodestruction (cryo or laser to destroy part of ciliary body)