# Chronic visual loss

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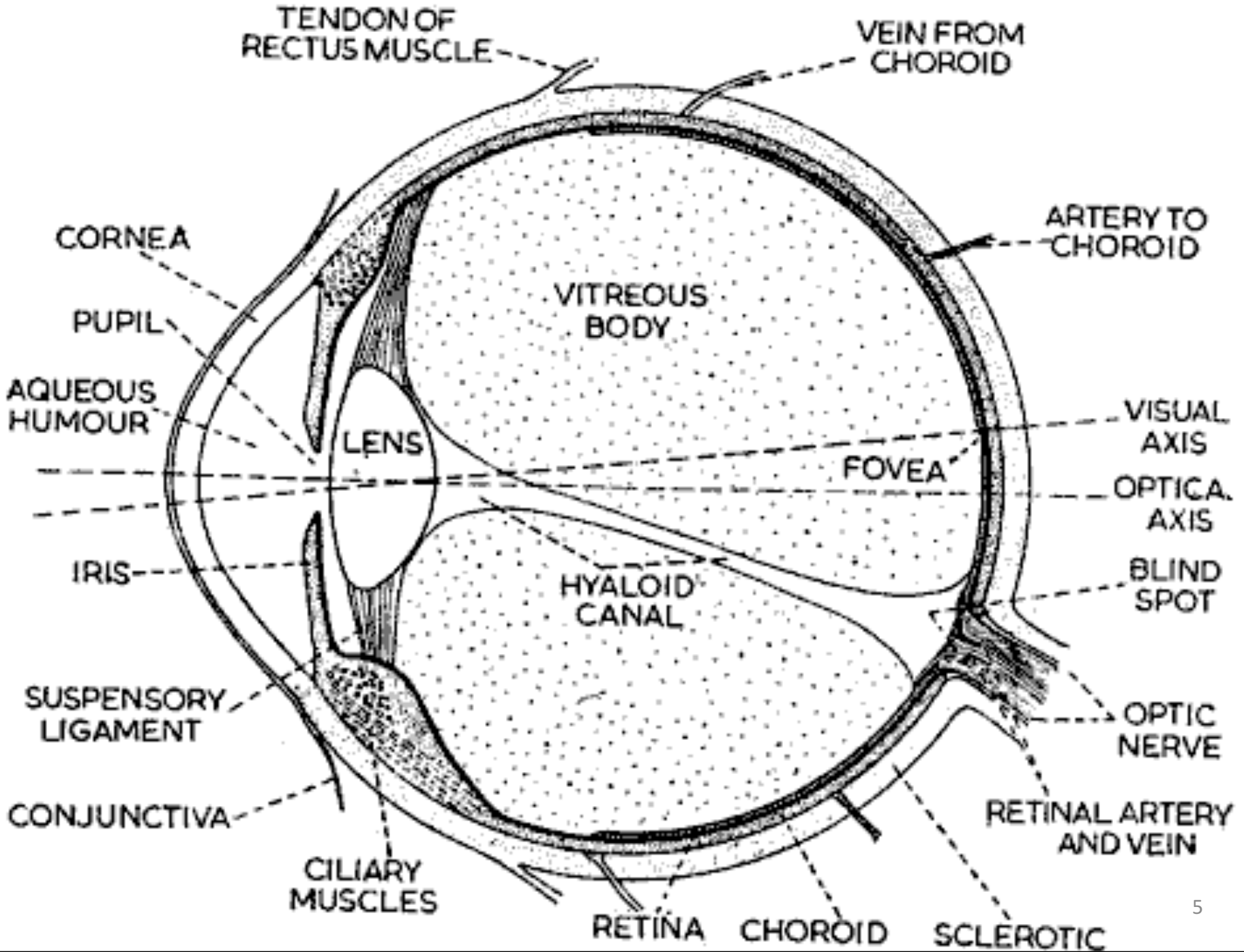
# Gradual decrease in vision

- **Cataract**
- **Open Angle Glaucoma**
- **Age related macular degeneration**
- Diabetic Retinopathy, hereditary retinopathies.
- Corneal  Opacity

# CATARACT

- Lens : Is biconvex and normally transparent .
- It is held in position by the suspensory ligament (Zonule).
- Diseases that affect the lens may affect:
  1) Structure
  2) Shape
  3) Position


TENDON OF RECTUS MUSCLE
VEIN FROM CHOROID
CORNEA
ARTERY TO CHOROID
PUPIL
VITREOUS BODY
AQUEOUS HUMOUR
LENS
VISUAL AXIS
FOVEA
OPTICAL AXIS
IRIS
HYALOID CANAL
BLIND SPOT
SUSPENSORY LIGAMENT
OPTIC NERVE
CONJUNCTIVA
RETINAL ARTERY AND VEIN
CILIARY MUSCLES
RETINA
CHOROID
SCLEROTIC

# Cataract (Definition)

- Opacification of the lens of the eye
- It is the most common cause of treatable blindness in the world

# Causes

## Age related cataract (Senile)

- Occur as a result of cumulative exposure to environmental and other influences, such as smoking, UV radiation and elevated blood sugar levels.

## *Pre-senile*

- Which may be associated with specific ocular or systemic diseases

# Ocular conditions

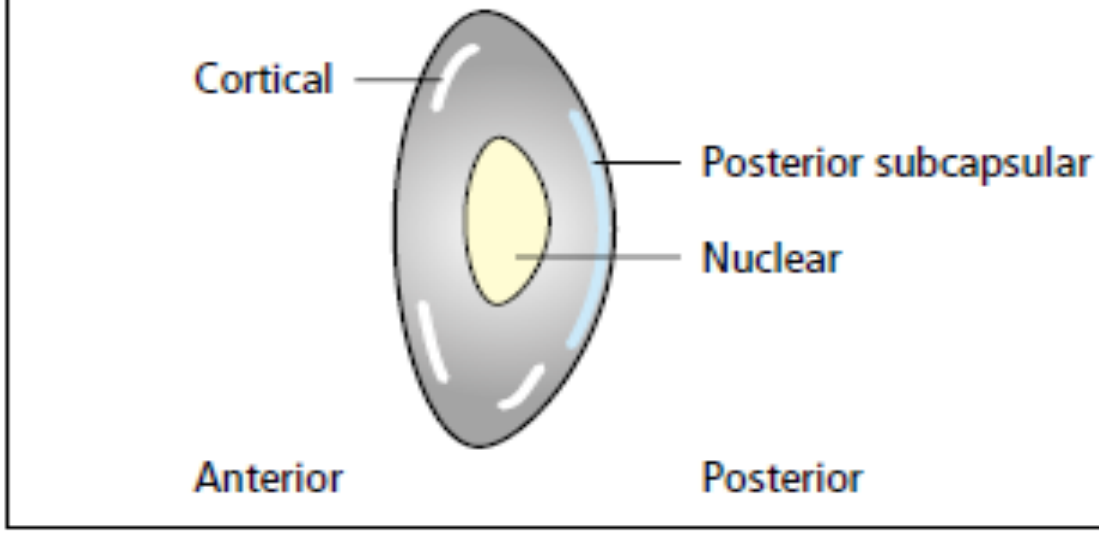
- Trauma
- Uveitis
- High myopia
- Topical medication (Steroid )
- Intraocular tumors

# Systemic causes

- Diabetes
- Other metabolic disorders (galactosaemia, hypocalcaemia)
- Systemic drugs ( steroids , chlorpromazine )
- Infection (congenital rubella)
- Myotonic dystrophy
- Atopic dermatitis
- Systemic syndromes (Down ' s, Lowe ' s)
- Congenital Cataract ( inherited )
- External radiation

# Age Related Cataract

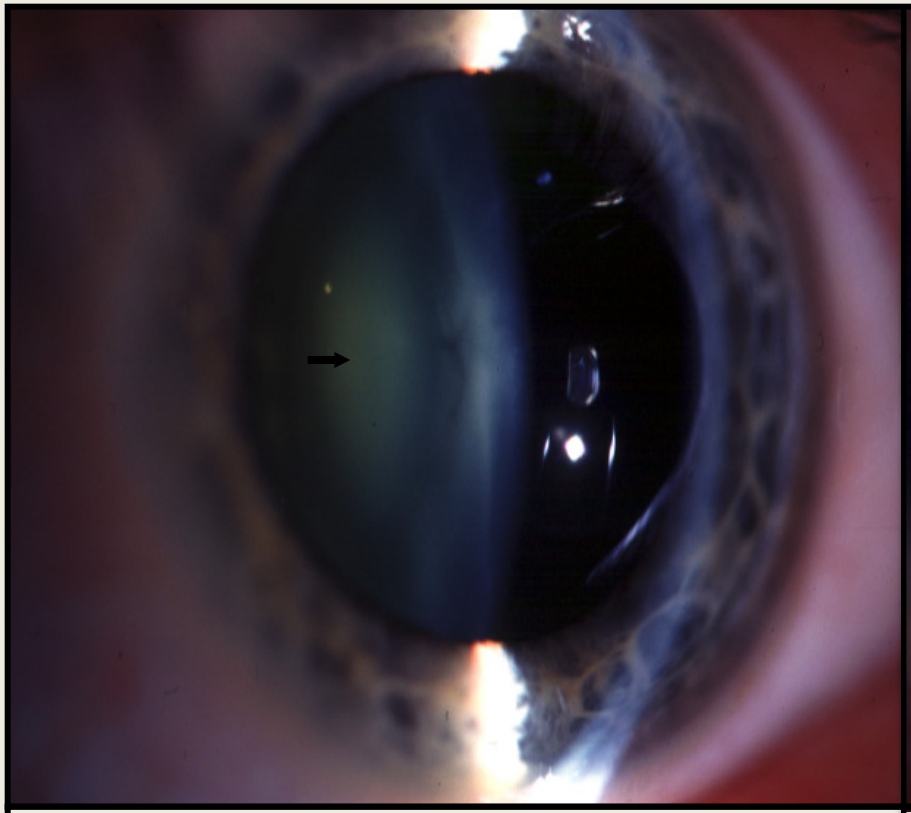
- **The main types** of age-related cataracts are :
  1. Nuclear sclerosis
  2. Cortical
  3. Posterior subcapsular.



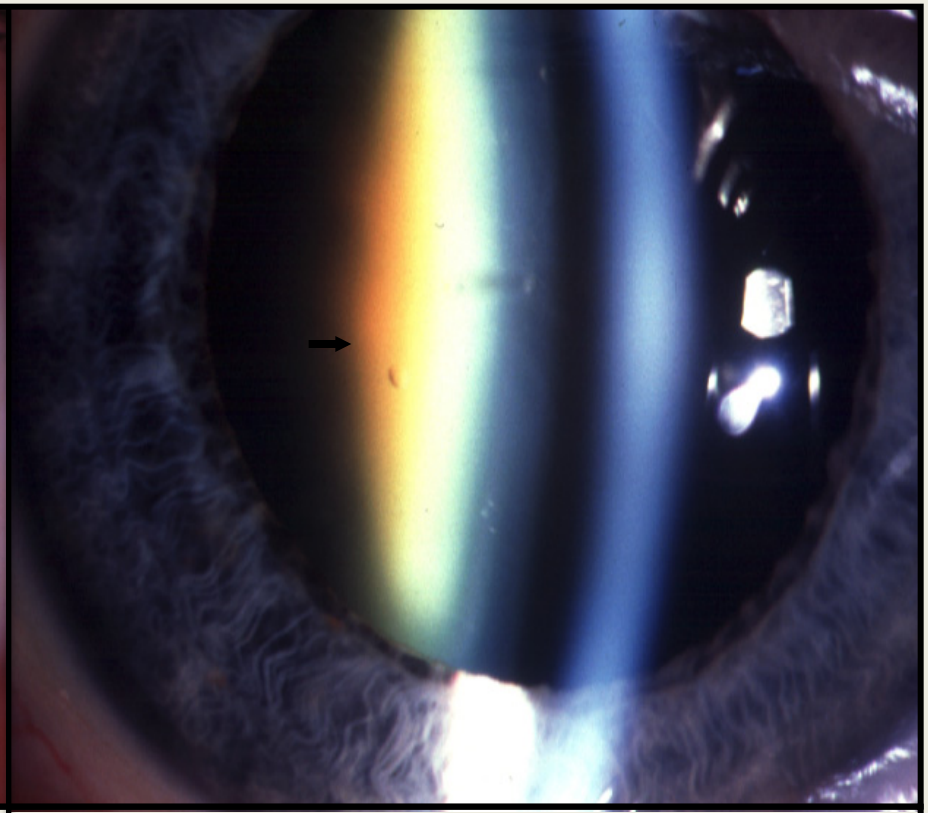
# 1)Nuclear sclerosis cataract

- The most common type of cataract and many consider them to be a normal maturation of the lens.
- Over time, the lens becomes larger and brunescent (yellow or brown) especially in the denser central nucleus.
- The lens can become so big that it pushes the iris forward, placing the patient at increased risk for *angle closure glaucoma.*

- Some patients with nuclear sclerotic cataracts will develop so called **“second sight”** where it seems like the vision improves. This is because the round cataract lens is more powerful and offsets the coexisting presbyopia allowing older patients to read better.



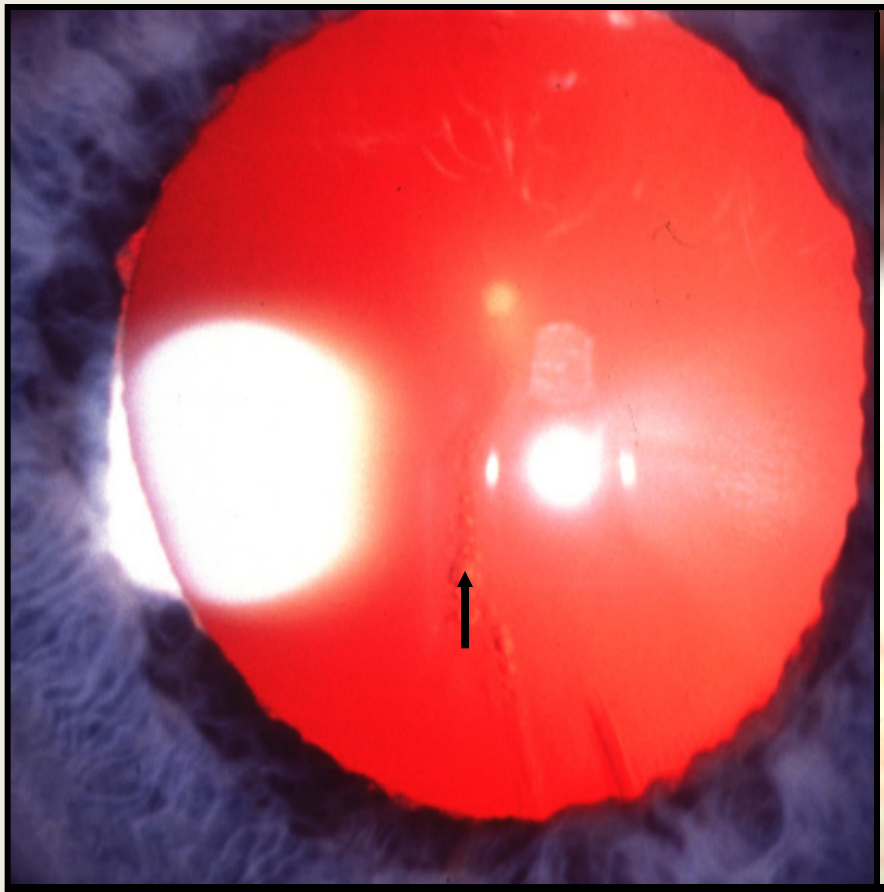
- Exaggeration of normal nuclear ageing change
- Causes increasing myopia



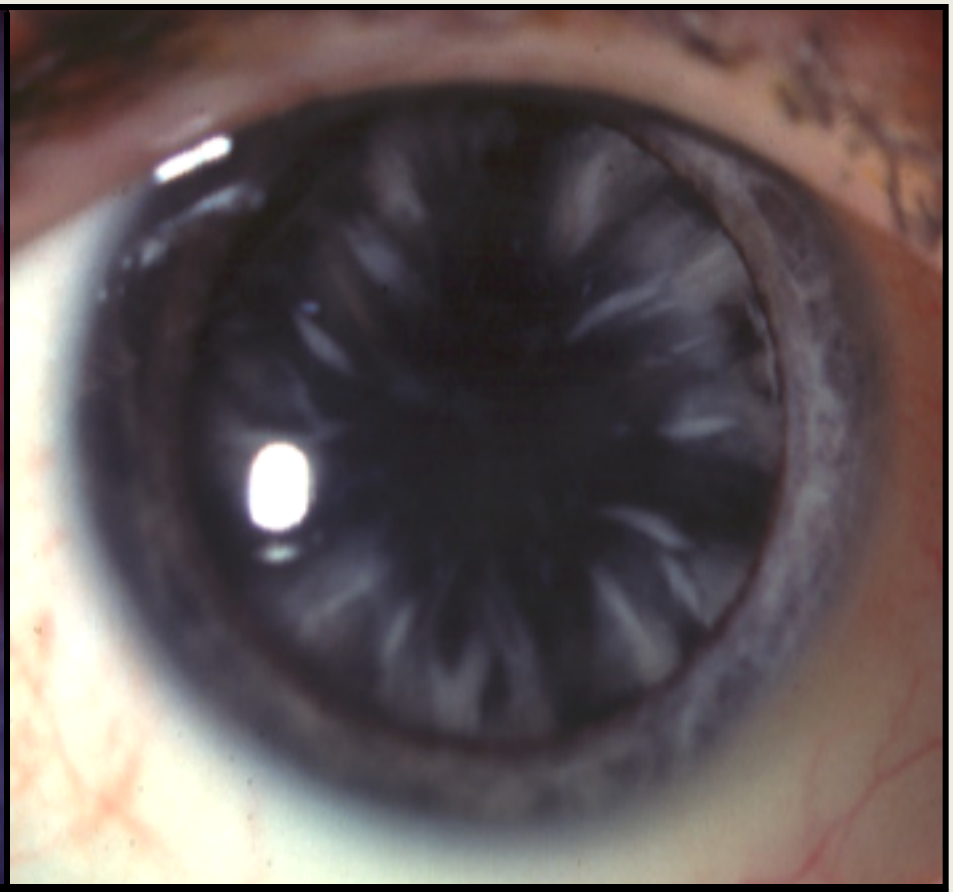
- Increasing nuclear opacification
- Initially yellow then brown

# 2) Cortical Cataract

- Are due to opacification of the lens cortex (outer layer). They occur when changes in the water content of the periphery of the lens causes fissuring.
- When these cataracts are viewed through an ophthalmoscope or other magnification system, the appearance is similar to white spokes of a wheel pointing inwards.
- *Symptoms* often include: problems with glare and light scatter at night.



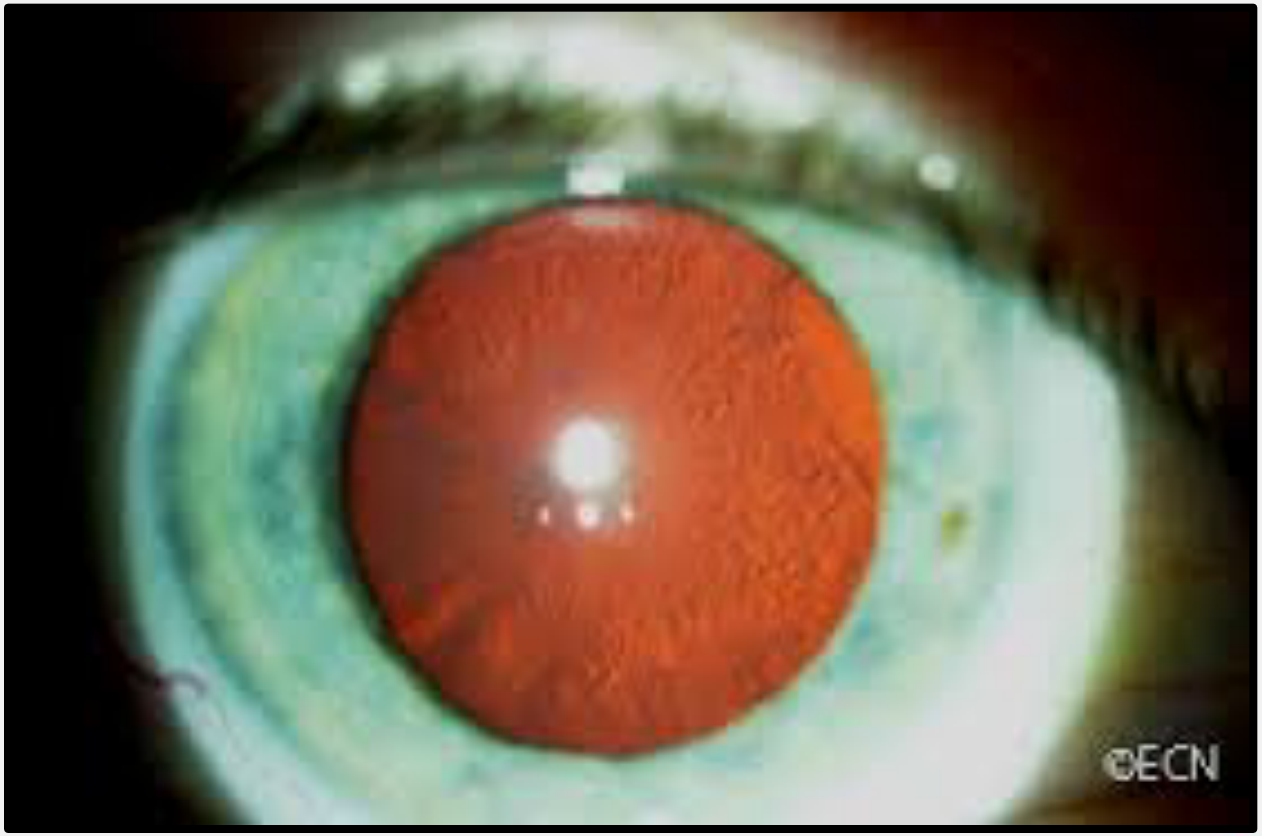
Initially vacuoles and clefts



Progressive radial spoke-like opacities

# 3) Posterior subcapsular

- Forms on the back of the lens, on the inner surface of the posterior capsule bag.
- These cataracts tend to occur in patients on steroids, with diabetes, and those with history of ocular inflammation.
- The opacity looks like breadcrumbs or sand sprinkled onto the back of the lens. This posterior location creates significant vision difficulty despite appearing innocuous on slit-lamp exam.


©ECN

# Congenital cataract

- It is found in about 1/250 live births
- Roughly one third is hereditary with autosomal dominant being the most common,
- One third is associated with a syndrome or metabolic disease, and the cause in the remainder is unknown.
- Congenital cataract is still one of the most common causes of blindness in children worldwide.

# Symptoms

- A cataract of sufficient degree causes:
  1. Painless loss of vision (Progressive)
  2. Glare
  3. Change in refractive error
  4. Amblyopia (in children)

# Signs

1. Decreased visual acuity  especially when measured in light
2. Lens opacity
3. Black spot against the red reflex
4. Location of the cataract

# Investigations

- Visual acuity
- fundoscopy
- Further investigations is required if systemic disease is suspected or if the cataract is congenital or appears at an early age.

# Treatment

Although much effort has been directed towards slowing progression or preventing cataract, **management remains surgical.**

## Techniques:

1. Phacoemulsification (new)
2. Extra Capsular Cataract Extraction (ECCE )
3. Intra Capsular Cataract Extraction ( ICCE ) (old)

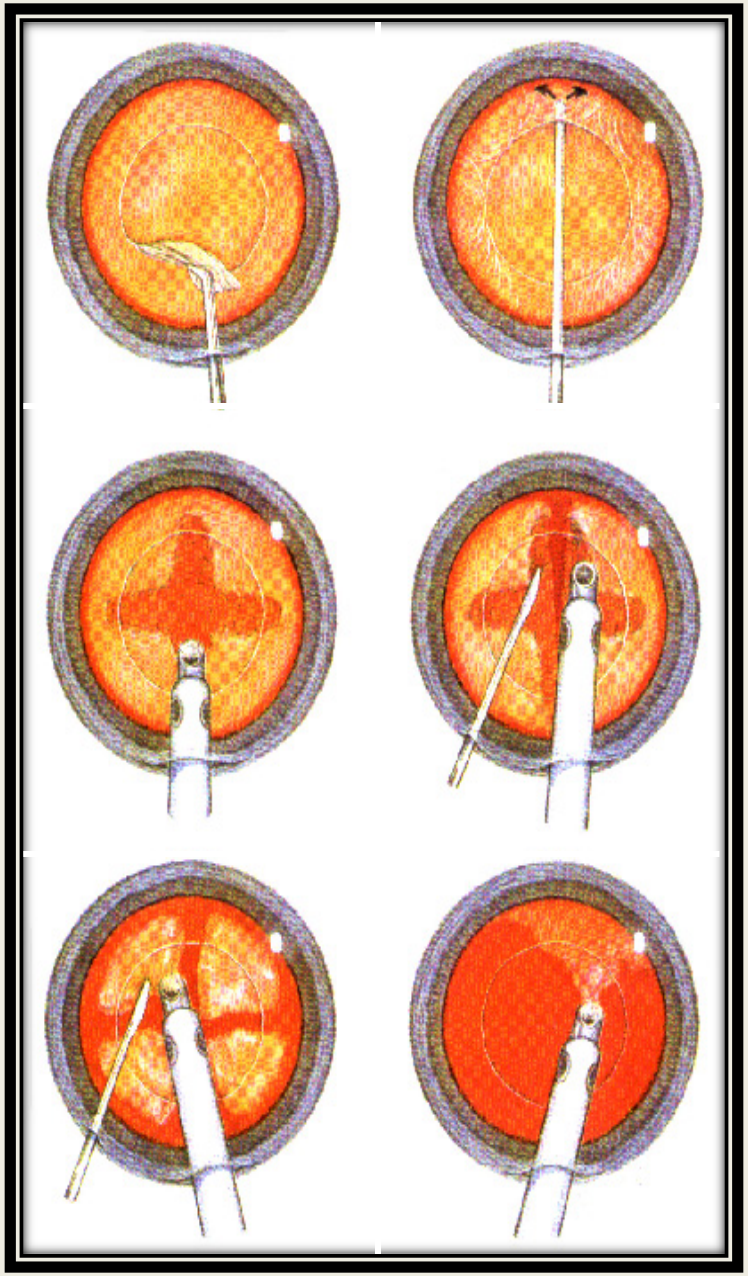
- Phaco and ECCE involves removal of the lenticular material leaving part of the lens capsule mostly the posterior part to give supprot for the  artificial lens that will be implanted .

- ICCE  removal of the whole lens

# Phacoemulsification

1. Capsulorhexis

2. Hydrodissection



3. Sculpting of nucleus

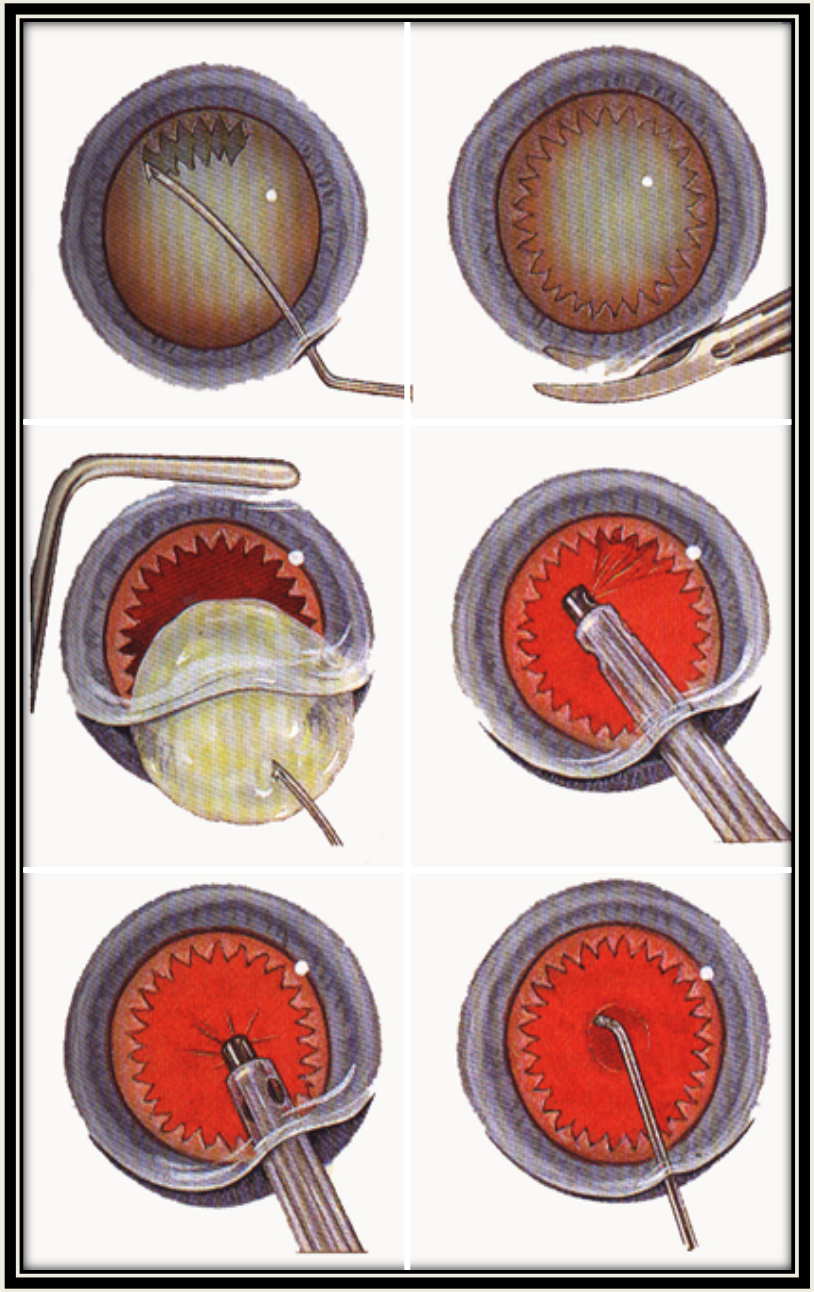
4. Cracking of nucleus

5. Emulsification of each quadrant

6. Cortical cleanup and insertion of IOL

# Extracapsular cataract extraction

1. Anterior capsulotomy



2. Completion of incision

3. Expression of nucleus

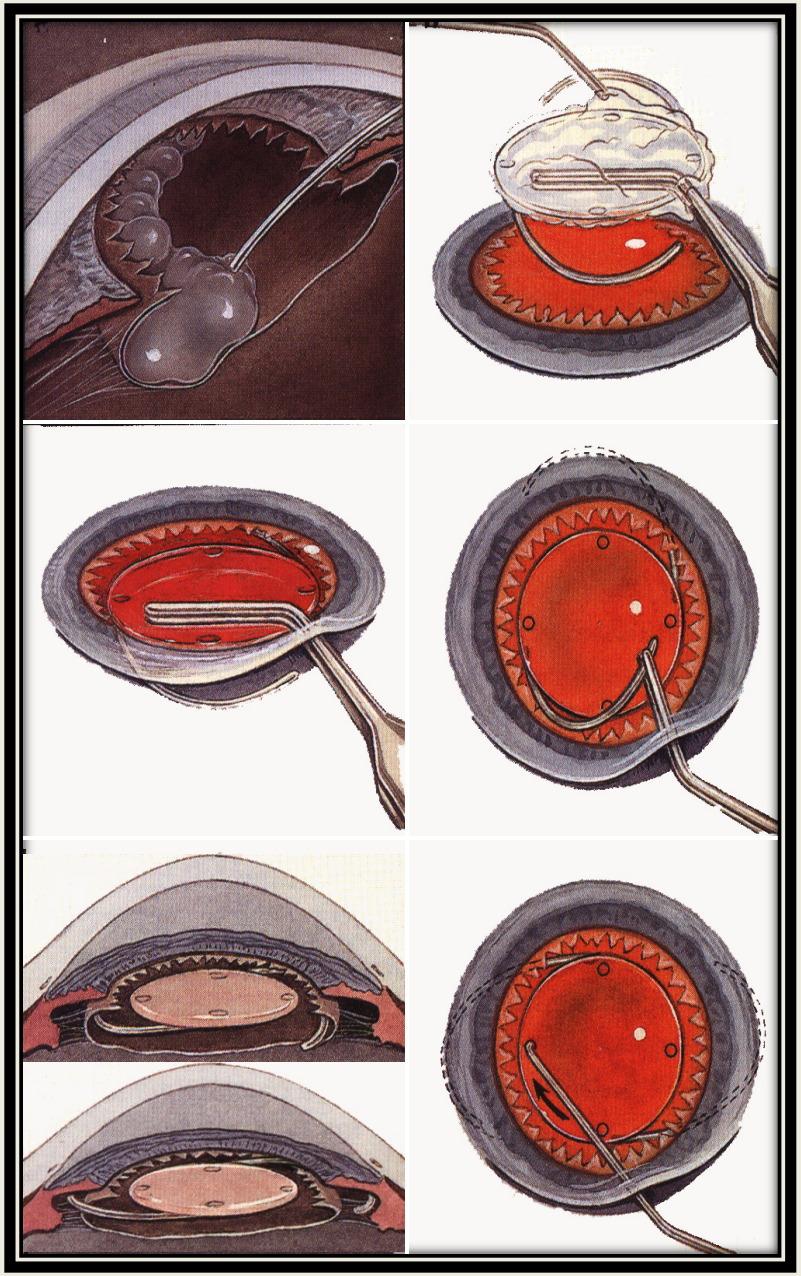
4. Cortical cleanup

5. Care not to aspirate posterior capsule accidentally

6. Polishing of posterior capsule, if appropriate

# Extracapsular cataract extraction ( cont. )

7. Injection of viscoelastic substance



8. Grasping of IOL and coating with viscoelastic substance

9. Insertion of inferior haptic and optic

10. Insertion of superior haptic

11. Placement of haptics into capsular bag and not into ciliary sulcus

12. Dialing of IOL into horizontal position

# Post-Operative care

- Steroids ( topical ,systemic )
- Antibiotics ( topical , systemic )
- Near vision adds

# Complications of Surgery

- Can be divided into :
- **Preopertaive**: which can be further divided into those related to ocular diseases and those related to systemic diseases.
- **Intraoperative**: divided into those related to anesthesia and those related to surgery itself
- **Postoperative**: divided into early and late

# Treatment of cong. cataract

- Visual outcome depends on :
  - ✓ The density of the cataract at and before the time of surgery
  - ✓ Age of onset
  - ✓ Age at time of surgery and
  - ✓ Very important a commitment parents.

- The timing of cataract surgery is the major factor in visual rehabilitation
- Before the age of 2 months, nystagmus is generally not present and the visual prognosis is excellent, provided that aggressive treatment of aphakia and amblyopia take place
- Bilateral cataracts have a better outcome than monocular ones. This is because the amblyopia of the cataract eye puts it at an irreversible disadvantage in comparison with the fellow eye as the child learns how to see.

- Babies with dense cataract who are not operated before the age of 2 months, showing nystagmus, have a guarded prognosis

- Acuity may never be better than 20/200. Because poor vision may be reversed, surgery should be performed as soon as possible

- The recommended surgery is lensectomy and vitrectomy which will result in aphakia, which is as amblyopiogenic as the cataract itself

- Therefore aggressive treatment of the aphakia is essential

- After the age of 2 years there is a general agreement to use intraocular lenses (IOLs)
- Before the age of 2 years, the use of IOLs is still controversial and usage of contact lenses or glasses is the rule
- Placement of secondary IOL at a later age is accepted

# GLAUCOMA

# Glaucoma

- A heterogeneous group of diseases in which the end result is damage to the optic nerve fibers .
- Mostly caused by the effect of raised intraocular pressure ( IOP )
- Normal values of IOP is 16 (±5).

# IOP

Depends on the balance between production and removal of aqueous humour.

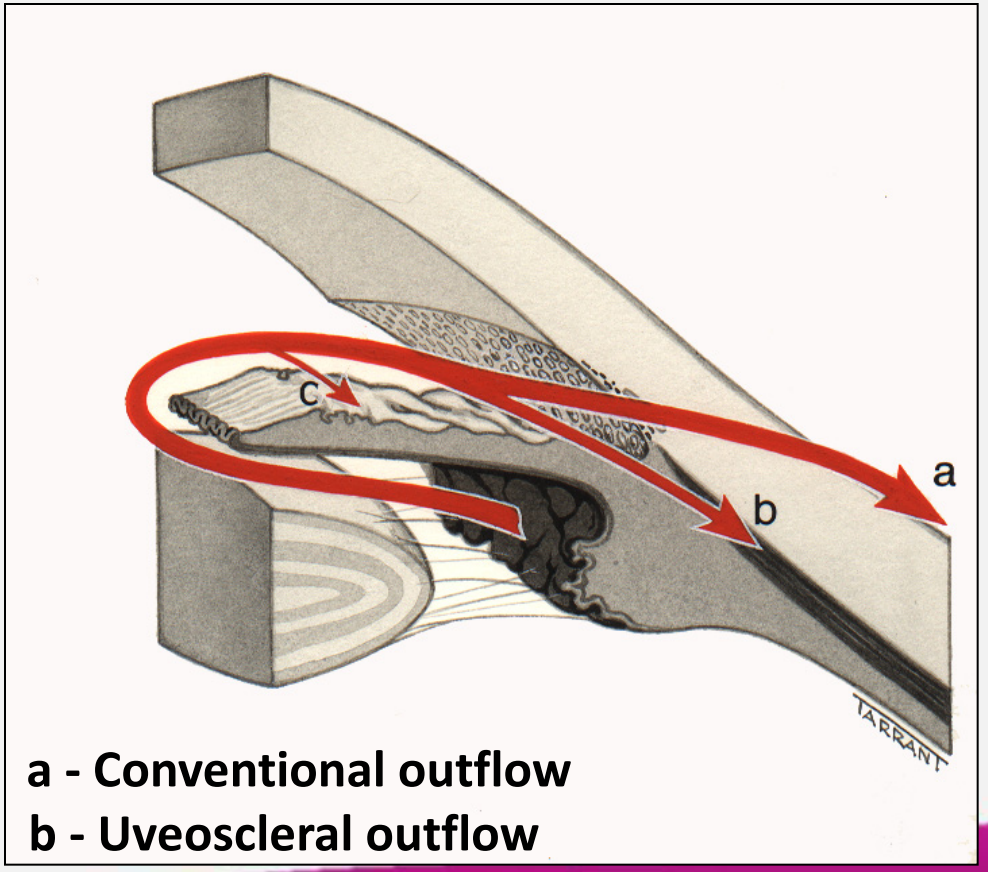
# Aqueous humour production

- Active secretion by the ciliary processes
- Ultra filtration

# Aqueous humour excretion

- Conventional pathway :(95%) via trabecular meshwork ,Schlemm's canal and the episcleral veins
- Uveoscleral pathway : (5% ) through the iris and ciliary body into the supra-choroidal space and the venous system through the sclera

# Aqueous outflow




**a - Conventional outflow**
**b - Uveoscleral outflow**

# Mechanism of Optic nerve fiber damage

- Mechanical damage by the raised IOP
- Ischemia of nerve fibers caused by impaired perfusion pressure

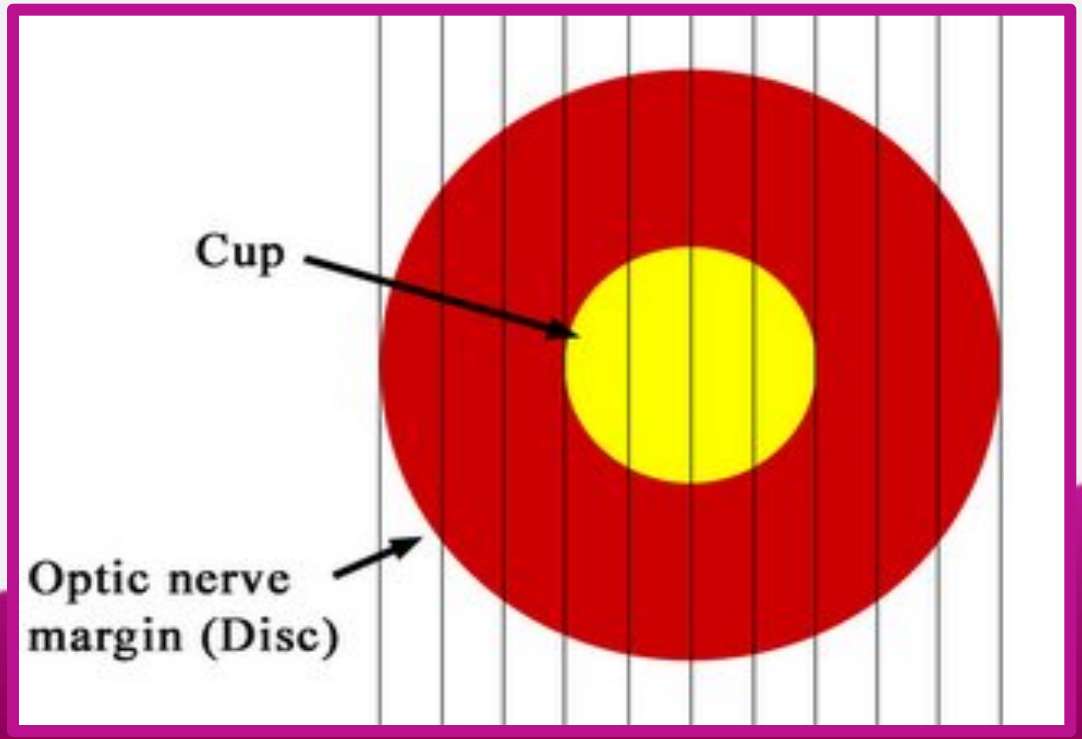
# Cup-to-Disc Ratio ( CDR)

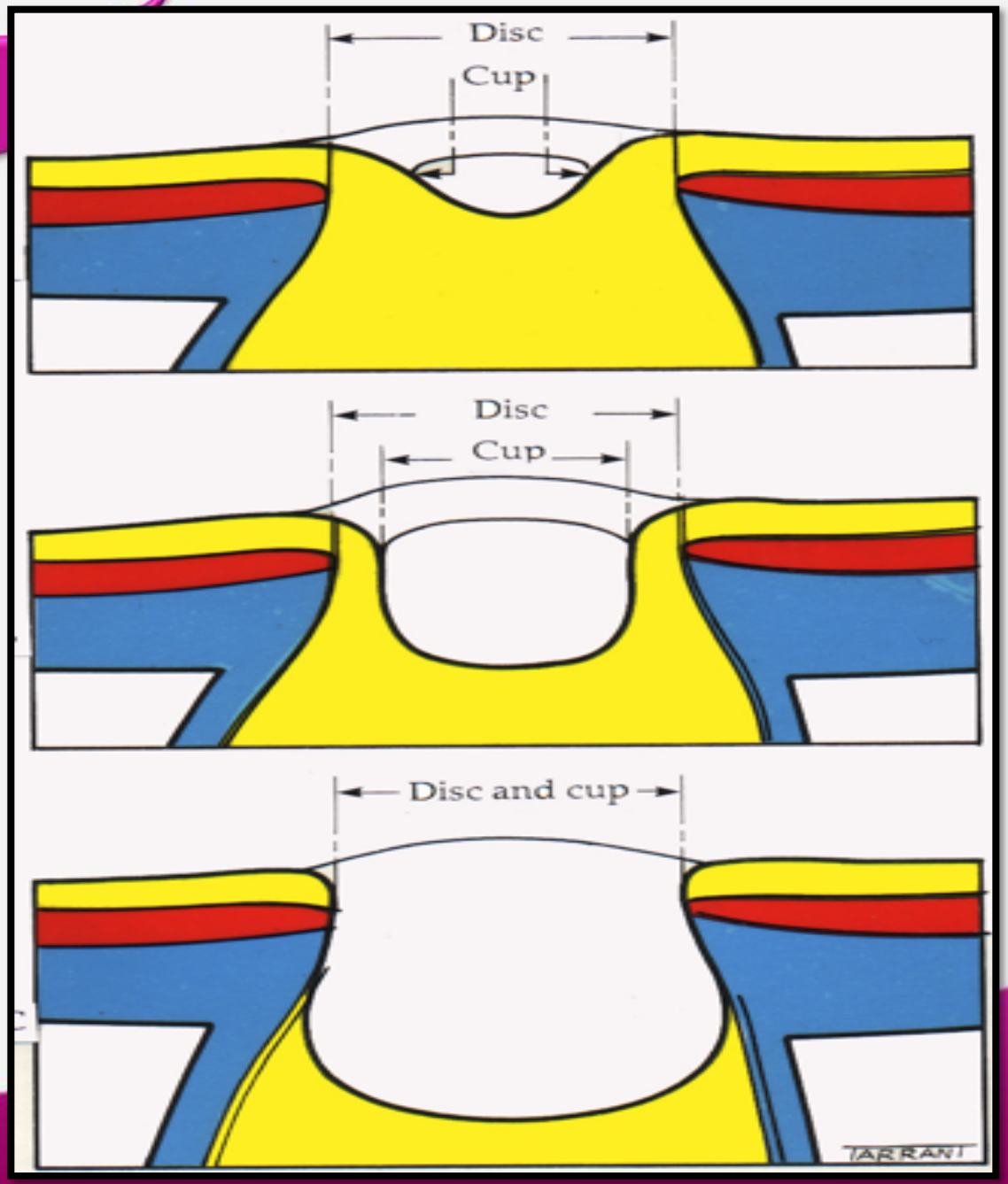
- The cup-to-disc ratio is a measurement used in ophthalmology **to assess the progression of glaucoma**.
- The **optic disc** is the anatomical location of the eye's "blind spot", the area where the optic nerve and blood vessels enter the retina.
- The *optic disc can be flat or it can have a certain amount of normal cupping*. But glaucoma, which is due to an increase in intra-ocular pressure, produces additional **pathological cupping of the optic disc**.

With the loss of nerve fibers from glaucoma, the cup becomes progressively larger because there is less space occupied by the remaining nerve fibers

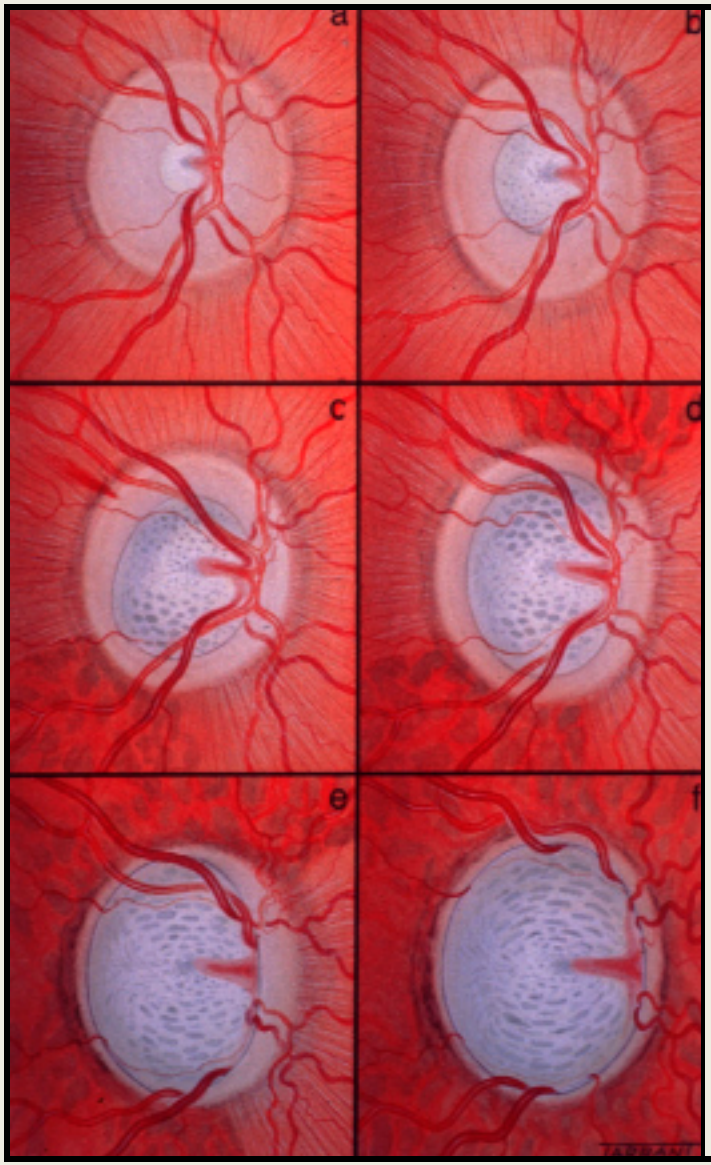
The *optic nerve is divided into tenths* and the cup is compared to the entire optic nerve (optic disc) to obtain the cup-to-disc ratio.

The normal c/d ratio is 0.3(The C/D ratio here is 0.4)




Disc
Cup
Disc
Cup
Disc and cup
TARRANT

# Progression of glaucomatous cupping




a. Normal (c:d ratio 0.2)

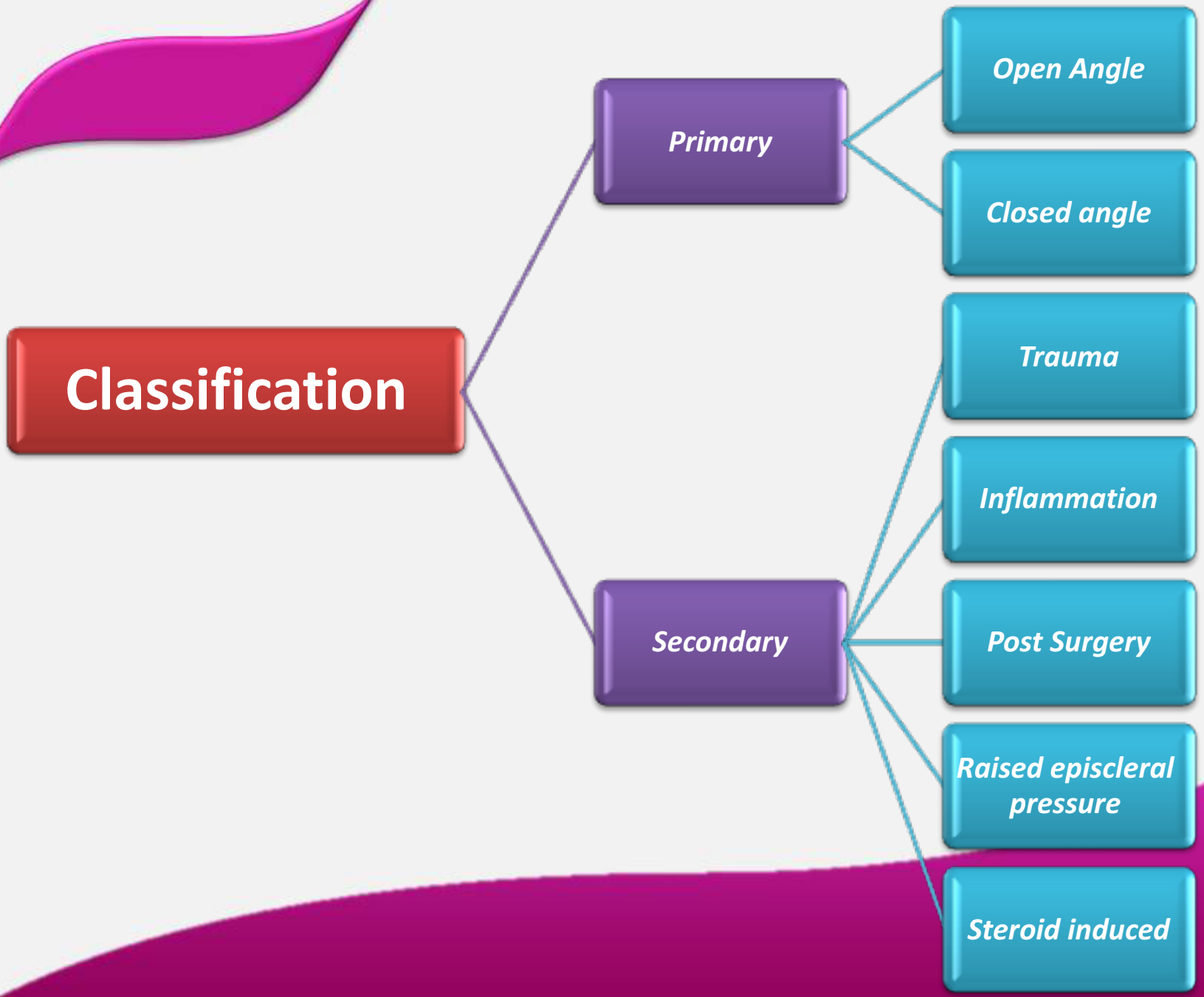
b. Concentric enlargement (c:d ratio 0.5)

c. Inferior expansion with retinal nerve fibre loss

d. Superior expansion with retinal nerve fibre loss

e. Advanced cupping with nasal displacement of vessels

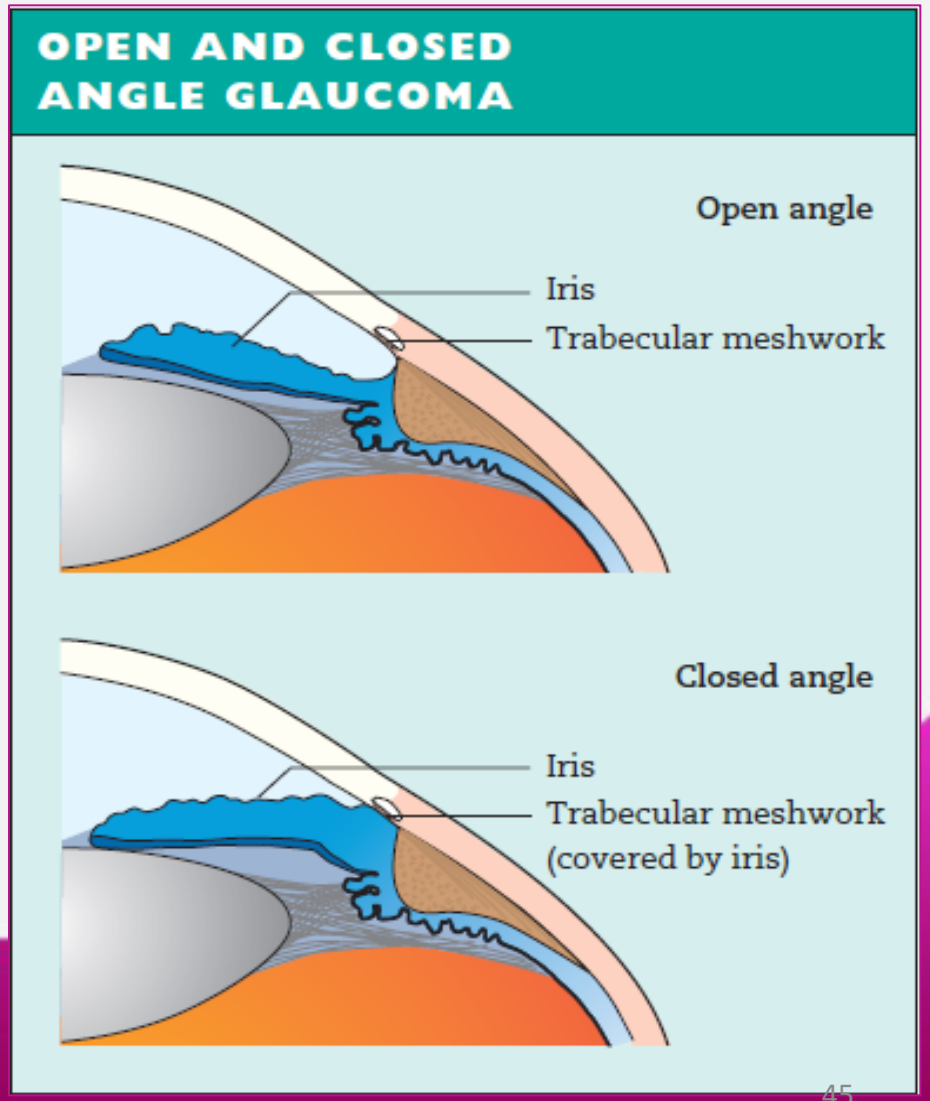
f. Total cupping with loss of all retinal nerve fibres


Classification
Primary
Open Angle
Closed angle
Secondary
Trauma
Inflammation
Post Surgery
Raised episcleral pressure
Steroid induced

# Primary Glaucoma

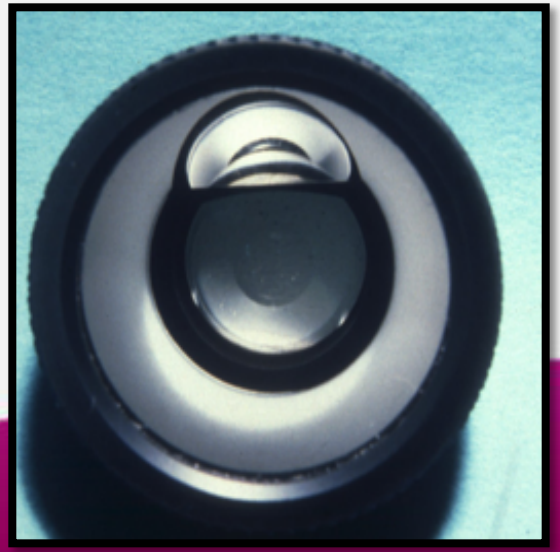
- Classification of the primary glaucomas is based on whether or not the iris is:
  - Clear of the trabecular meshwork (open angle)→ ( **POAG** )
  - Covering the meshwork (closed angle)→ (**PACG** )

***ANGLE: the iridocorneal angle***



# How to examine the ANGLE

- Using special lenses which allows visualization of the angle structure to determine whether the angle configuration is closed or open [***Goniolenses***] .



# Pathogenesis open Angle

- In OAG the trabecular meshwork appears normal but offers an increased resistance to the outflow due to :

1* Thickening of the trabecular lamellae

2* Reduction in the number of lining trabecular cells

3* Increased extracellular materials in the trabecular meshwork

# Chronic Open Angle Glaucoma

- Affects 1 in 200 of the population > 40y
- Males and Females are equal
- Incidence increase with age 10% in > 80y
- Exact mode of inheritance not clear though incidence increase in first degree relatives .

- **History :**

Usually *asymptomatic* because the rise in IOP is slow and chronic → Patients will present with progressive loss of vision or Visual field defects .

- **Examination :**

- Vision - Visual field - Slit Lamb Exam
- IOP (normal range) - Gonioscopy
- Ophthalmoscopy to examine Cup to Disc ratio

# Treatment

1- Medical Treatment
2- Laser Treatment
3- Surgical Treatment

Aim of treatment is to decrease the IOP.

Amount of decrease depends on the level at which no more damage to the optic nerve happens

# Medical treatment

| TREATMENT OF GLAUCOMA | | |
|---|---|---|
| **Topical agents** | **Action** | **Side effects** |
| Beta-blockers (timolol, carteolol, levobunolol, metipranolol, betaxolol-selective) | Decrease secretion | Exacerbate asthma and chronic airway disease<br>Hypotension, bradycardia |
| Parasympathomimetic (pilocarpine) | Increase outflow | Visual blurring in young patients and those with cataracts<br>Initially, headache due to ciliary spasm |
| Sympathomimetic (adrenaline, dipivefrine) | Increase outflow<br>Decrease secretion | Redness of the eye<br>Headache |
| Alpha2-agonists (apraclonidine, brimonidine) | Increase outflow through the uveoscleral pathway<br>Decrease secretion | Redness of eye<br>Fatigue, drowsiness |

| | | |
|---|---|---|
| Carbonic anhydrase inhibitors (dorzolamide, brinzolamide) | Decrease secretion | Stinging<br>Unpleasant taste<br>Headache |
| Prostaglandin analogues (latanoprost, travaprost, bimatoprost, unoprostone) | Increase outflow through the uveoscleral pathway | Increased pigmentation of the iris and periocular skin<br>Lengthening and darkening of the lashes, conjunctival hyperaemia<br>Rarely, macular oedema, uveitis |
| **Systemic agents** | | |
| Carbonic anhydrase inhibitors (acetazolamide) | Decrease secretion | Tingling in limbs<br>Depression, sleepiness<br>Renal stones<br>Stevens–Johnson syndrome |

If intraocular pressure remains elevated the choice lies between:

- adding additional medical treatment;
- laser treatment;
- surgical drainage procedures.

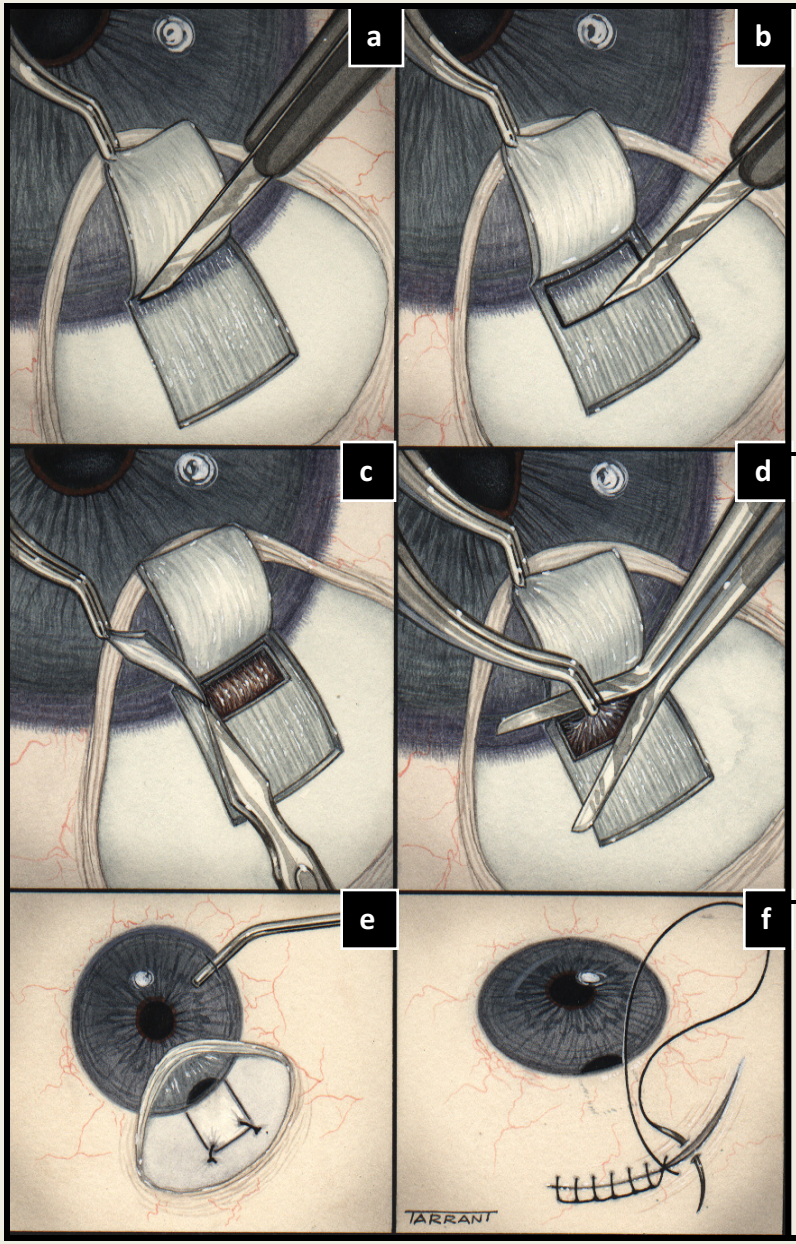
# LASER TRABECULOPLASTY

Involves placing series of laser burns in the trabecular meshwork to improve aqueous flow

# Surgical Treatment

- Drainage surgery ( Trabeculectomy) by creating a fistula between the anterior chamber and the subconjuctival space

# Technique




a. Cutting of deep block - anterior incision

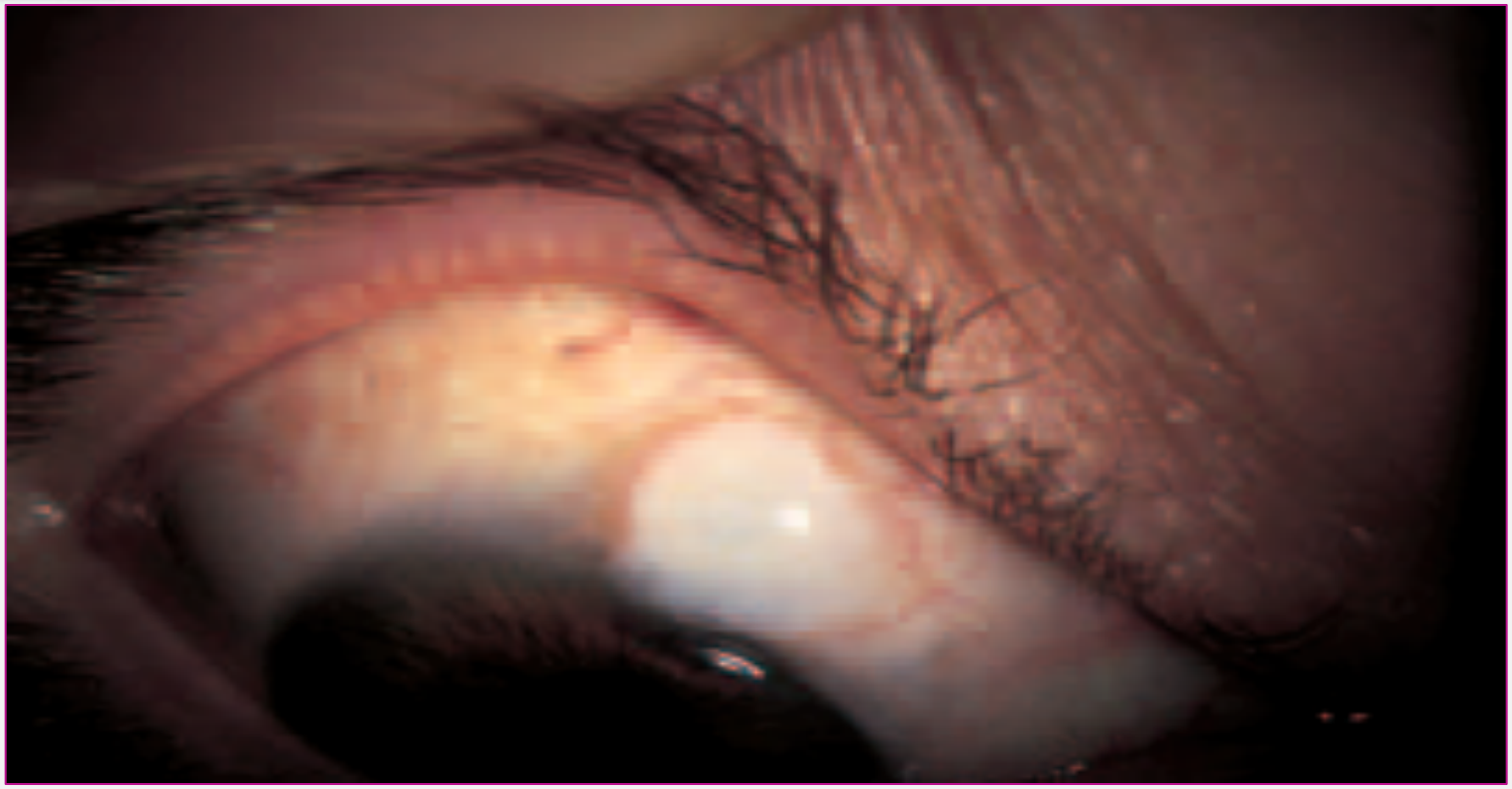
b. Posterior incision

c. Excision of deep block

d. Peripheral iridectomy

e. Suturing of flap and reconstitution of anterior chamber

f. Suturing of conjunctiva



The appearance of a trabeculectomy bleb.

# AGE-RELATED MACULAR DEGENERATION

# Age-Related Macular Degeneration

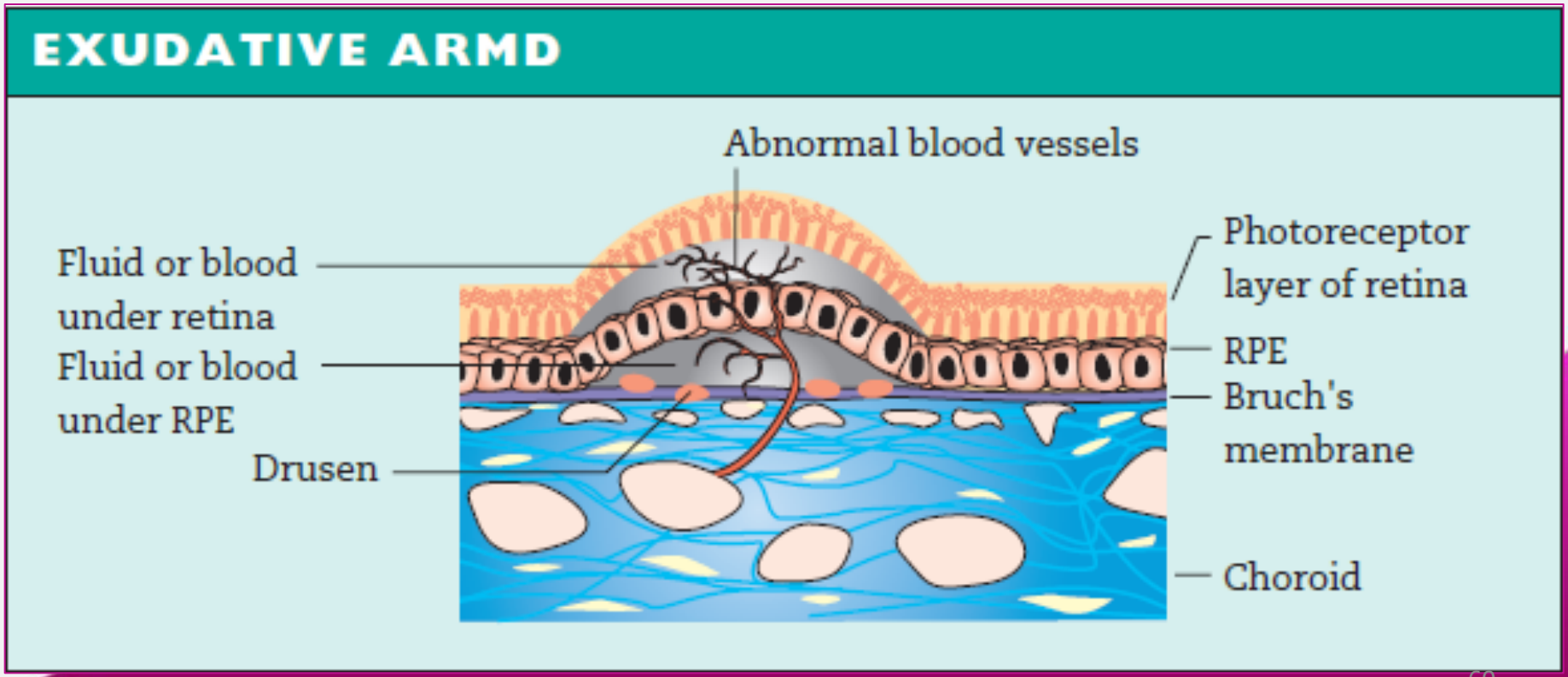
- Age related macular degeneration (AMD) is the commonest cause of irreversible visual loss in the developed world.

# Dry \ Non-exudative ARMD

***Pathogenesis*** :

- Normally the RPE metabolizes the outer segment of the photoreceptors and get rid of the lipofucin and other metabolites resulting from its damage.
- With age this function decreases and these metabolites start to accumulate in the Bruch's membrane .

- Deposits form which can be seen with the ophthalmoscope as discrete sub-retinal yellow lesions called ***drusen***.



# Wet\Exudative ARMD

- There is a new blood vessels growing from the choroid invading the Bruch"s membrane and growing under the retina (*sub-retinal neovascular membrane).*
- The hallmark: choroidal neovascularization (CNV)
- This will lead to subsequent hemorrhages , scaring and foveal detachment.

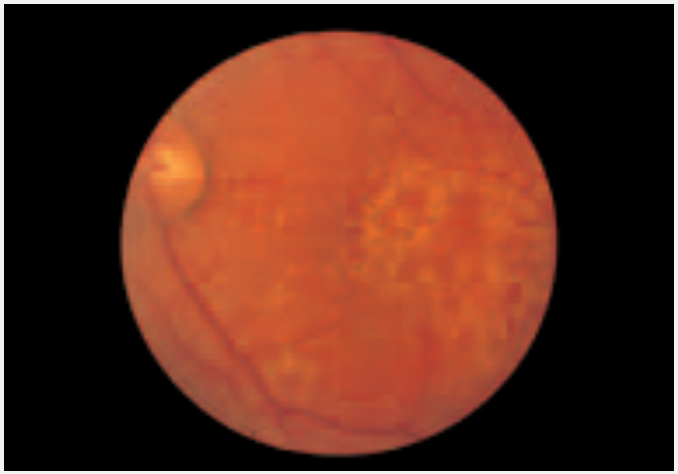
## Dry macular degeneration

- ✓ The tissues of the macula are thin as cells disappear.
- ✓ No evidence of scaring or of bleeding or other fluid leakage in the retina.
- ✓ Both eyes may be affected simultaneously.
- ✓ Central vision slowly and painlessly worsens.

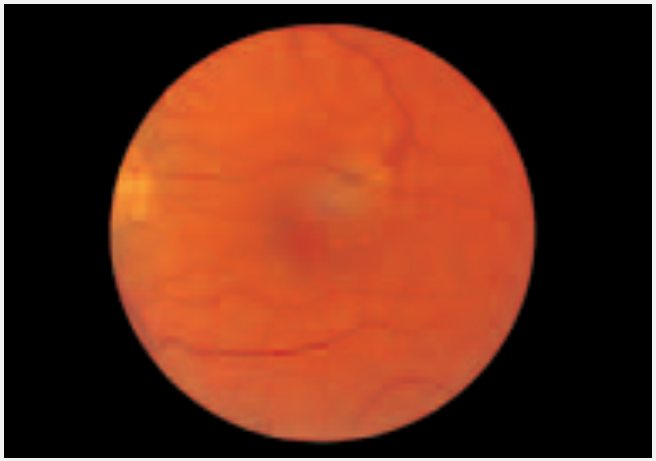
## Wet macular degeneration

- ✓ Abnormal blood vessels develop in the layer of tissue under the macula
- ✓ These vessels may leak fluid and blood under the retina in that area. Eventually a mound of scar tissue develops under the retina.
- ✓ It develops in one eye first but eventually affects both eyes.
- ✓ Loss of vision tends to progress quickly and may be particularly sudden if one of the abnormal blood vessels bleed.

- A doctor can usually diagnose macular degeneration by examining the eyes with an ophthalmoscope or slit lamp. Sometimes fluorescein angiography is used to determine the diagnosis



**dry AMD, note the discrete scattered yellowish sub-retinal drusen**



**wet AMD, note the small haemorrhage associated with the sub-retinal membrane**

- **Management:**

- For dry AMD.. No medical or surgical treatment is available; however, the AREDS trial found benefits with some vitamin along with high doses of antioxidants.
- For wet AMD.. Antiangiogenics can cause regression of the abnormal blood vessels and improve vision as:
  I. Bevacizumab (Avastin)
  II. Ranibizumab (Lucentis)


thanx