ANATOMY OF EYE : LAYERS AND CORNEA ANATOMY

Introduction :

Shape of the eyeball is aspherical (oblate spheroid).

Volume of the eyeball : 6 ml.

Length/Axial length/AP diameter of eyeball : 24 mm/2.4 cm.

Eyeball is situated within a bony cage called orbit.

Has 4 walls : Roof, floor, lateral & medial wall.

Volume of the orbit : 30 ml.

Structure of the eyeball

00:02:50

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The eyeball is made up of 3 layers : Outer, middle and the inner layer.

Outermost layer :

Known as fibrous layer.

- Sclera (white) :
  Protective opaque covering that covers most of eyeball.
  Extends from cornea in front to optic nerve in back.
  Extra ocular muscles are attached to sclera.
- Cornea :
  Anterior convex transparent part of eye that covers front portion of eye.
- Limbus :
  Junction between cornea & sclera.

Middle layer :

Known as Vascular layer.

- It is also known as uvea.
- Made of :
  Iris.
  Ciliary body.
  Choroid.

Lens is a structure (Does not belong to any layer) suspended in the middle by Cilliary zonules (Present 360 degree around lens)

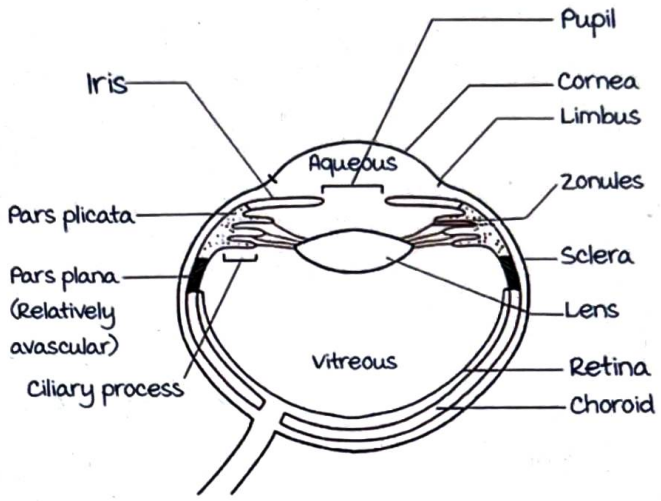
Active space

Innermost layer :

Known as neural layer.

- It is also known as retina.
- Central retinal artery & vein enters eye through optic nerve/disc.

Anatomy of eyeball



Anterior/ Convex : Cornea

Posterior/ Opaque : Sclera

Ciliary body : Pars plicata (Ant) & pars plana (Posterior, Avascular). Ciliary process secrete aqueous.

Choroid is posterior part of uvea lined inside by retina.

Space in front of lens : Contains Aqueous.

Space behind lens : Contains Vitreous

Retina : Covers/lines only the posterior part of the eye.

Cornea 00:26:03

Anterior and is transparent(As it is Avascular).

Nerve supply :

Mnemonic : TON.

60e6b3eeaa8ded0e4e7e5ea7 Trigeminal nerve (5th cranial nerve).

↓

Ophthalmic nerve (has 3 branches) :

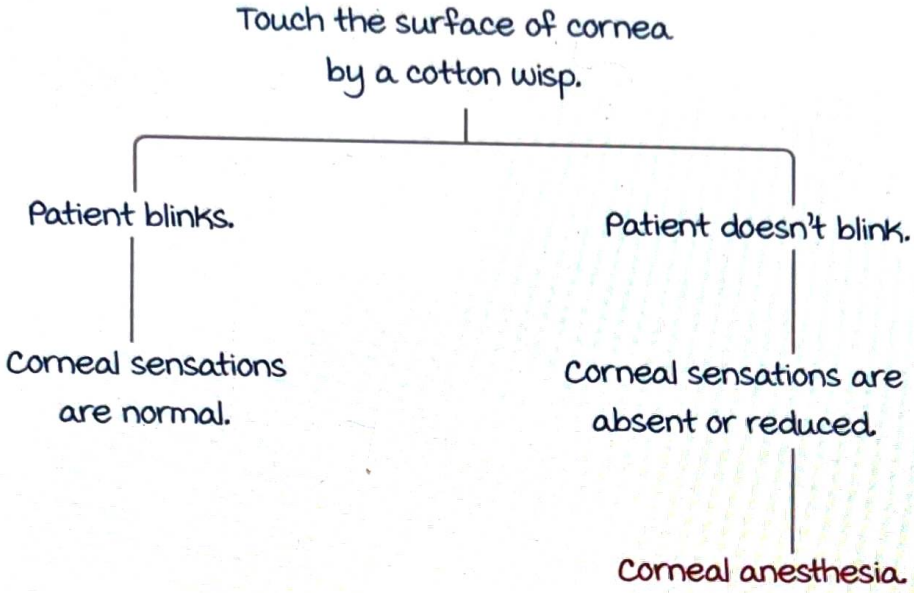
Lacrimal nerve.

Frontal nerve.

Nasociliary nerve : Supplies cornea.

Testing corneal sensation :

Illicit corneal blink reflex using cotton wisp.



The causes of corneal anesthesia :

Mnemonic : HDL ACTS.

Herpes.

Diabetes.

Leprosy.

Acoustic neuroma.

Contact lens.

Topical Timolol (Beta blocker) long term use.

Surgical trauma.

Corneal blink reflex

00:37:08

- Afferent pathway : Sensory → 5th cranial nerve.
- Efferent pathway : Motor → 7th cranial nerve to orbicularis oculi.

- Clinical correlation :

  Bell's palsy is characterised by orbicularis ocular palsy. It leads to inability to close eyes/blink called Lagophthalmos.

Layers of cornea

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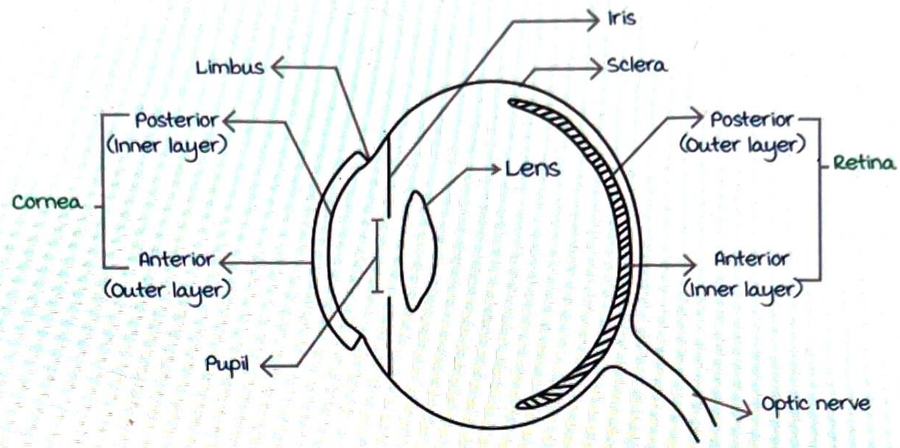
There are 6 layers :



1. Outermost or anterior layer :

   Epithelium :

   - Non-Keratinised.
   - Stratified : i) Superficial : Squamous.
     (8 strata) ii) middle.
     iii) Basal : Columnar.
     Basal cells can undergo mitosis.
     Therefore corneal epithelium can regenerate.

2. Bowman's layer :

Not a true basement membrane.
PAS negative :
Does not stain with Periodic Acid Schiff reagent as in true basement membranes.

Acellular :

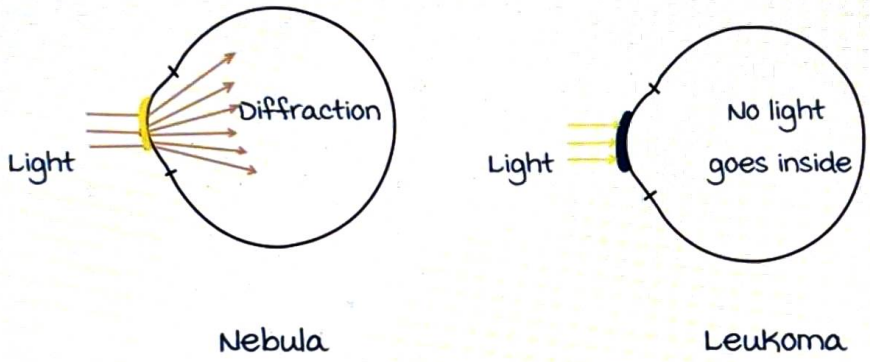
- Cannot regenerate.
- It heals by scar(corneal opacity) formation.
- The patient can suffer from loss of vision.

Active space

Types of corneal opacity :

Types	Pupillary margin	Iris structural details
Nebular (faint)	Visible	Visible
Macular	Visible	Not visible
Leukomatous (dense/ completely white)	Not visible	Not visible

Corneal opacity causing max visual loss : Leucomatous
Causing maximum visual disturbance or distortion : Nebular.
Patient complains that he can see but not clearly.



Nebula Leukoma

Nebula makes the corneal surface irregular causing diffraction of light rays, this will be perceived as visual disturbance/distortion.

Layers of cornea: Stroma, Dua's layer and Descemet's membrane 01:07:44

3. Stroma :
   - It contributes to 90 % of total corneal thickness.
   - most common type of collagen : Type I.
   - most common type of Glycosaminoglycans : Keratin sulphate.

4. Dua's layer :
   - Discovered by Dr Havinder Dua.
   - Strongest corneal layer.
   - Thickness ranges from 7 to 14 microns.

5. Descemet's membrane :

- It is secreted continuously by the underlying endothelial cells.
- It can regenerate.
- It's thickness increases throughout life.
- Peripheral termination of Descemet's membrane is known as Schwalbe's line.

Layers of cornea: Innermost layer 01:14:24

6. Innermost/ posterior layer :

The innermost layer is called as endothelium.
Single cell layer.
metabolically active layer.

Helps to maintain corneal transparency by :

- Active Sodium Potassium ATPase pump to maintain corneal dehydration.
- Tight junctions between adjacent endothelial cells.

Endothelial cells :

- Prevent aqueous from going into cornea (Zona Adherence / tight junctions forms a barrier).
- Breach in endothelial cell layer can cause aqeous leakage into cornea seen as corneal edema.
- Normal endothelial cell count :

  2400 to 3000 cells/ mm^2
- If endothelial cells are < 2400 but > 500 cells/ mm^2, cornea adapts by Polymegathism and Polymorphism. i.e by increasing size and changing shape and plug the gap.
- If endothelial cells are < 500/ mm^2, it leads to Corneal decompensation (hazy, edematous cornea).
- Investigation done for endothelial cell count :

  Specular microscopy.

Corneal transparency 01:26:52

Factors that help to maintain corneal transparency :

1. Tear film.
2. Avascularity of cornea.
3. Collagen fibres in stroma : Uniform in diameter.
   - Uniform in diameter.
   - Equal distance between all collagen fibrils, that is less than half the wavelength of light.
     Destructive interference between light rays will cancel out scattering of light.
4. Corneal dehydration maintained by endothelium.
5. Normal intraocular pressure.
6. Crystalline proteins in corneal stroma reduce the backscatter of light.

ANATOMY OF EYE : SCLERA, LIMBUS AND OCULAR ROUTES OF DRUG ADMINISTRATION

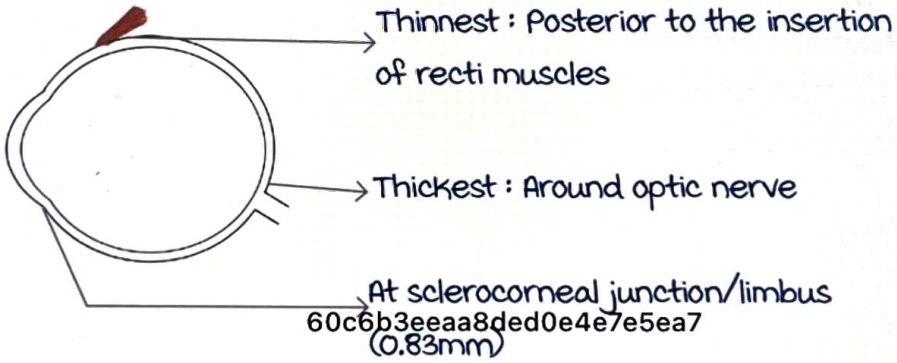
Sclera

00:00:06

Forms the posterior part of outer most layer.
It covers the posterior 5/6th of the eyeball
White in colour/opaque because collagen fibres have an irregular thickening/bundelling which prevents the light to pass through them.

Thickness varies :

- Sclera thickest (1mm) → Around the optic nerve.
- Sclera thinnest (0.3mm) → Posterior to the insertion of recti muscle.



Layers of Sclera

00:03:30

1. Episclera (outermost part)
   Densly vascular layer.
   It has two groups of blood vessels :
   - Superficial blood vessels.
   - Deeper blood vessels (closer to sclera proper)
2. Sclera proper (avascular layer)
3. Lamina fusca (Inner most layer)

Episcleritis	Scleritis
Inflammation of superficial episcleral plexus.	Inflammation of deeper vascular plexus.

Active space

Reddish hue	Violaceous hue
Vasoconstrictor test : (2.5% Phenylephrine e/d) Redness disappears → Blanching.	Vasoconstrictor test : Redness/violaceous hue does not disappear → No blanching.
No tenderness.	Pain/tenderness is present.
Idiopathic in most cases.	Commonly associated with systemic diseases like Rheumatoid arthritis.
	

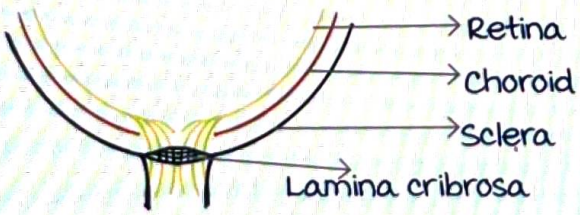
Types of scleritis :

- Anterior non-necrotising.
- Anterior necrotizing :
  - With inflammation → Associated with Rheumatoid arthritis, Wegeners granulomatosis, Polyarteritis nodosa.
  - Without inflammation/Scleromalacia perforans (white eye, diagnosed late, complication → Perforation of sclera)

Openings in Sclera :

- Lamina cribrosa (posteriorly) → Sieve like opening to transmit the optic nerve fibres to pass.
- middle (4–7 mm posterior to the equator) → For vortex veins.
- Anteriorly → For anterior ciliary artery and nerves.

Posterior segment of the eye



Active space

Limbus

00:17:48

Limbus is the corneoscleral junction.

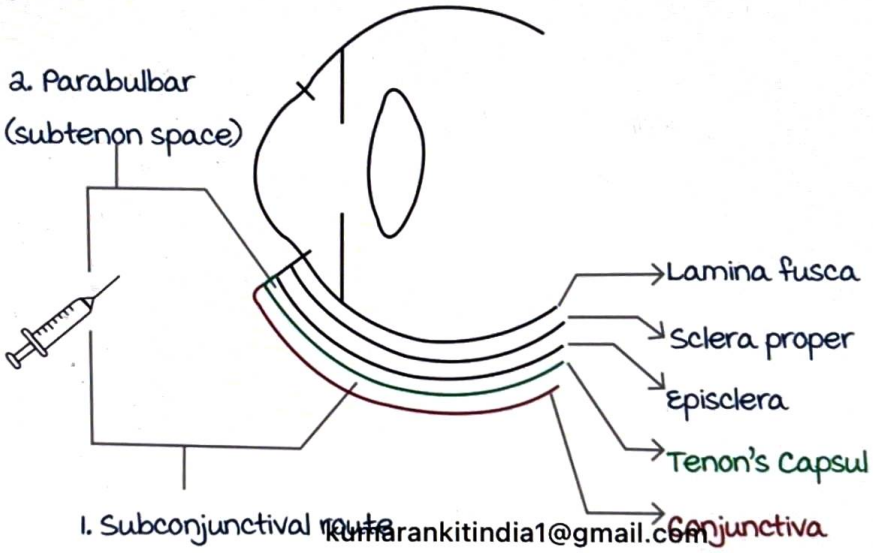
Contains stem cells (pleuripotent cells) :

- Have the ability to differentiate.
- Location → Epithelium in specialized areas called Palisades of Vogt.
- Specific marker for limbal stem cells → ABCG2
- Universal marker → CD34

Limbal stem cell deficiency disorder → Pterygium (overgrowth of conjunctiva on the cornea)

Routes of ocular drug administration

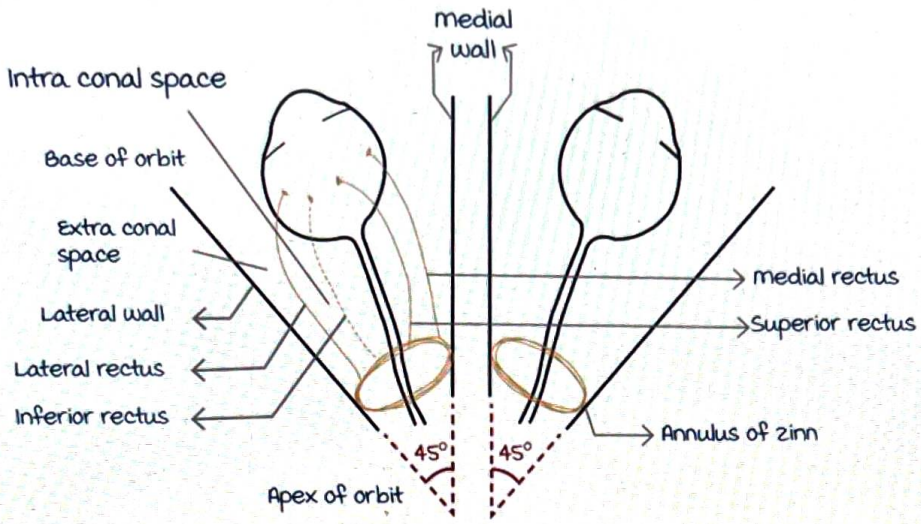
00:24:41



1. Subconjunctival route.
2. Parabulbar route (Subtenon space) → Nozik technique.
3. Topical (On the ocular surface).
   - Eye drop → Administered at least three minutes before an eye ointment.
   - Eye ointment → Provides more time of contact. (preferred in children)
4. Intravitreal route.
5. Intracameral injection → In anterior chamber.
6. Peribulbar route of ocular local anesthesia.
7. Retrobulbar route of ocular local anaesthsia

Active space

Peribulbar and Retrobulbar routes 00:34:02



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Both medial walls are parallel to each other (0°)
Angle between the two lateral walls of the orbits → 90°.

Annulus of Zinn → A common tendinous ring at the apex of the orbit.
All the ocular muscles except inferior oblique take its origin from Annulus of Zinn.

Peribulbar → Around the eyeball, injection in Extraconal space
Retrobulbar → Behind the eyeball, injection in Intraconal space.

Technique of peribulbar block :

- Choose the site of injection → Through the lower lid at the junction of middle and lateral 1/3rd of the inferior orbital rim.
- Open the lower lid with left thumb, and a 24/25 G needle is inserted through the lower lid along the floor of the orbit taking care to not pierce the eyeball
- Confirmation is done by aspirating the syringe to rule out puncturing any vessel.
- Slowly inject 5 mL of the local anesthetic mixture in the peribulbar space.
- Post-injection, compression of the eye for 5 mintues is done to lower the intraocular pressure.

Active space

ANATOMY OF EYE - IRIS, UVEA, ACCOMMODATION AND ITS ANOMALIES

Middle layer of the eye 00:00:08

Anatomical classification of uvea/uveal tract.

(From anterior to posterior) :

- Iris.
- Ciliary body.
    1. Pars plicata.
    2. Pars plana.
- Choroid.

Functional classification :

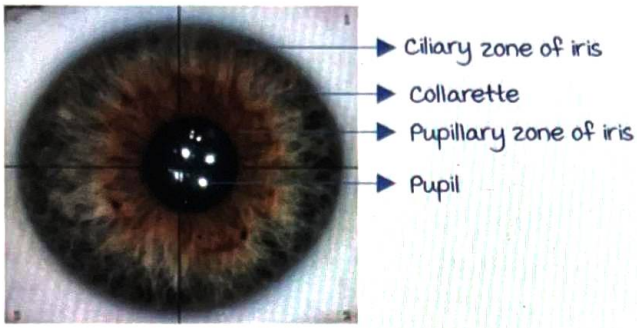
- Anterior uvea : Iris and pars plicata (anterior uveitis/ iridocyclitis).
- Intermediate uvea : Pars plana (intermediate uveitis/ pars planitis).
- Posterior uvea : Choroid (choroiditis).

Iris 00:02:50

It is a muscular tissue which has an aperture in the center known as the pupil.

The thickness of iris varies :

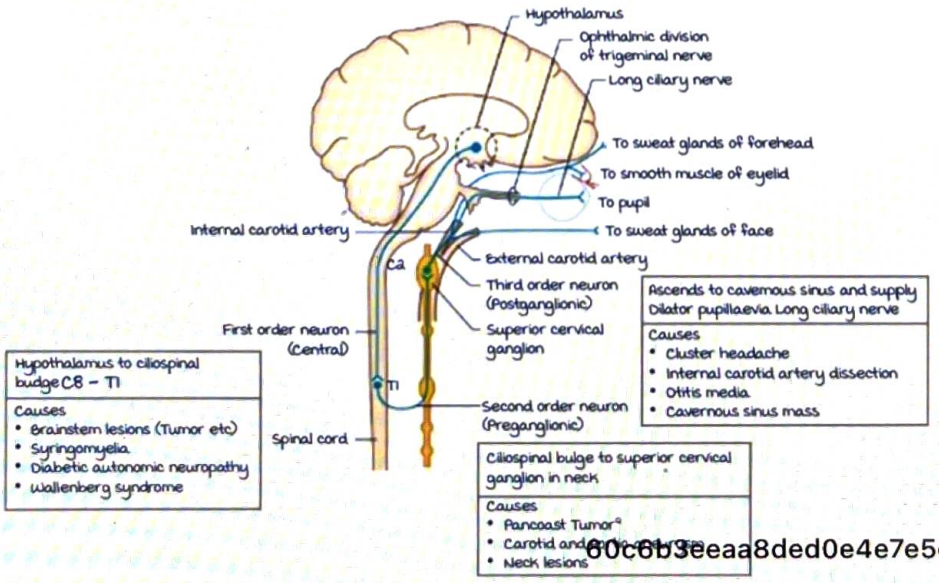
- Thickest : Collarette (the zone between pupillary and ciliary zones of iris).
- Thinnest : Root of iris, due to which it is a site of iris detachment (iridodialysis).



Muscles of iris :

	Sphincter pupillae	Dilator pupillae
Function	Constriction of pupil (miosis).	Dilatation of pupil (mydriasis)
Nerve supply	Parasympathetic (CN 3,7,9,10) Oculomotor nerve ↓ Inferior branch (also supplies inferior oblique muscle) ↓ Short ciliary nerve	Sympathetic nervous system Long ciliary nerve

Sympathetic pathway to the eye 00:13:12



Active space

Horner's syndrome/HS

00:15:59

Oculo - Sympathetic palsy.

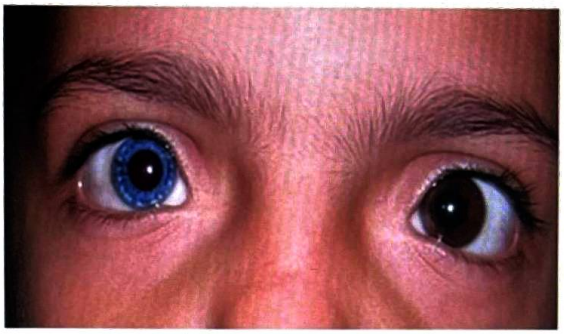
Clinical features : (Mnemonic - HI MAPLE).

Heterochromia iridis : Different colors of both iris.

- Hypochromic heterochromia iridis : Loss of color in the affected eye.

  This type is seen in Horner's syndrome.

  It is seen only in congenital cases.

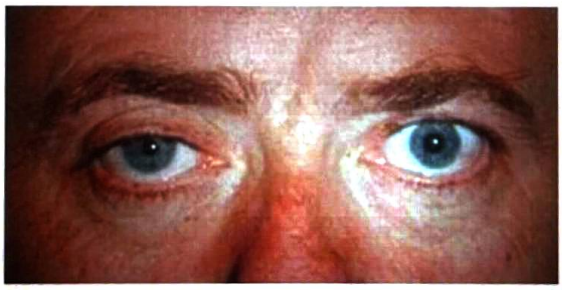


Heterochromia iridis

- Hyperchromic heterochromia iridis : Darker color of the affected eye.

  Causes : Latanoprost (antiglaucoma drug, PG-$F_{a}\alpha$ analogue).

  Siderosis bulbi (Iron foreign body injury).



Horner's syndrome

Inferior eyelid elevation.

Miosis (due to paralysis of dilator pupillae).

Anhydrosis : Ipsilateral loss of sweating.

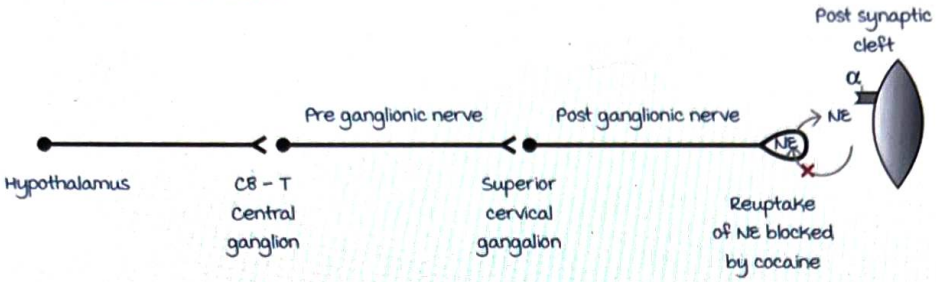
Ptosis : Drooping of the ipsilateral upper eyelid (due to paralysis of muller's muscle).

Loss of ciliospinal reflex.

Enophthalmos (sunken eyeballs) : Relative enophthalmos.

Test for diagnosis of Horner's syndrome :

1. 4% cocaine test :



Cocaine blocks the reuptake of norepinephrine (NE) and increases its level at the postsynaptic cleft and thus causing mydriasis of the iris.

- In normal patients, on cocaine test, the pupils will dilate.
- In Horner's syndrome (no epinephrine due to lesions in the sympathetic pathway of the eye), there is no dilatation.

2. 1% Apraclonidine test :

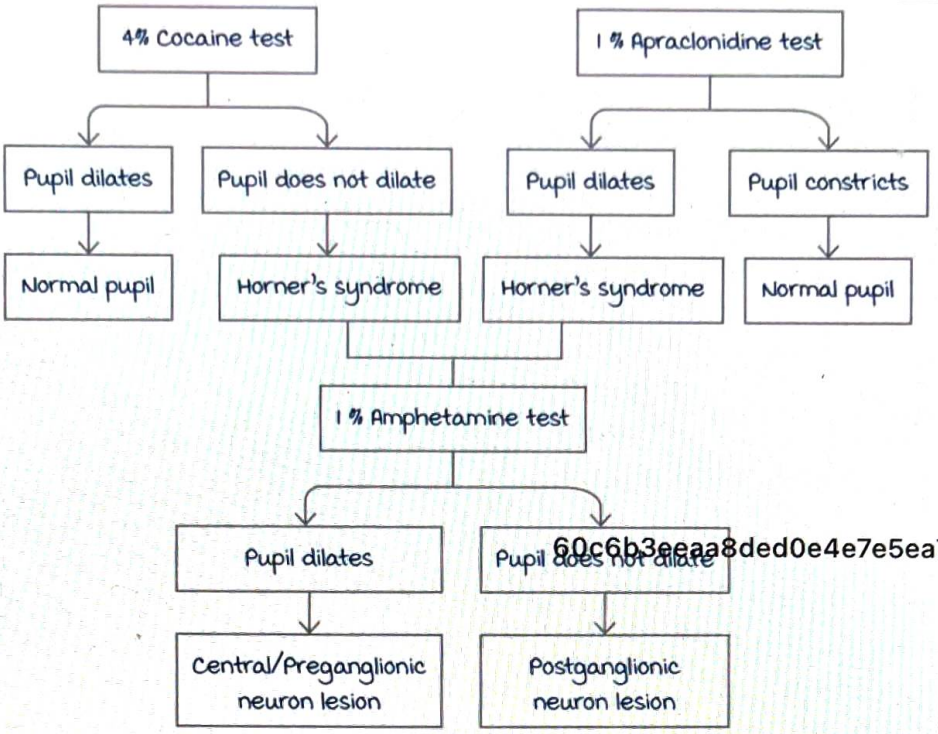
Apraclonidine : Nonselective α agonist, acts on both α 1 and α 2 receptors.

- In normal patients, it acts more on α 2 (decreases NE) : Miosis.
- In HS (no NE) : There is upregulation of α 1 receptor, on which apraclonidine acts and leads to mydriasis.

3. Localization of the lesion is by 1% Amphetamine test :

Amphetamine acts by displacement and increases the release of NE into the postsynaptic cleft.

- A lesion in central (1st order)/pre-ganglionic (2nd order) neurons : Amphetamine can act at postganglionic (3rd order) and releases the NE l eading to mydriasis.
- A lesion at post-ganglionic neuron : Amphetamine cannot act on these damaged nerve endings and thus there is no mydriasis.



Causes of miosis and mydriasis

00:38:59

Miosis (mnemonic : HI COPS)	Mydriasis (mnemonic : BIG OATS)
Horners syndrome, Hemorrhage (Pontine) Iridocyclitis Clonidine, Carbolic acid Organophosphates, Opioids Parasympathomimetic, Phenothiazines, Pilocarpine Sleep, Sedatives	Belladona poisoning Internal ophthalmoplegia Glaucoma (ACG) Occulomotor nerve palsy Atropine Tonic pupil Sympathomimetics

Ciliary body

00:40:45

Parts :

- Pars plicata.
- Pars plana (relatively avascular).

Active space

Functions :

- Secretion of aqueous from the non-pigmented epithelium of ciliary process.
- Accommodation : A part of near reflex (is the changes taking place in the eye when looking at a near object) which comprises of :
    1. Accommodation.
    2. Convergence.
    3. Miosis.

Accommodation 00:44:22

Ciliary zonules are the thread-like structures attached on the lateral end to the ciliary muscle.

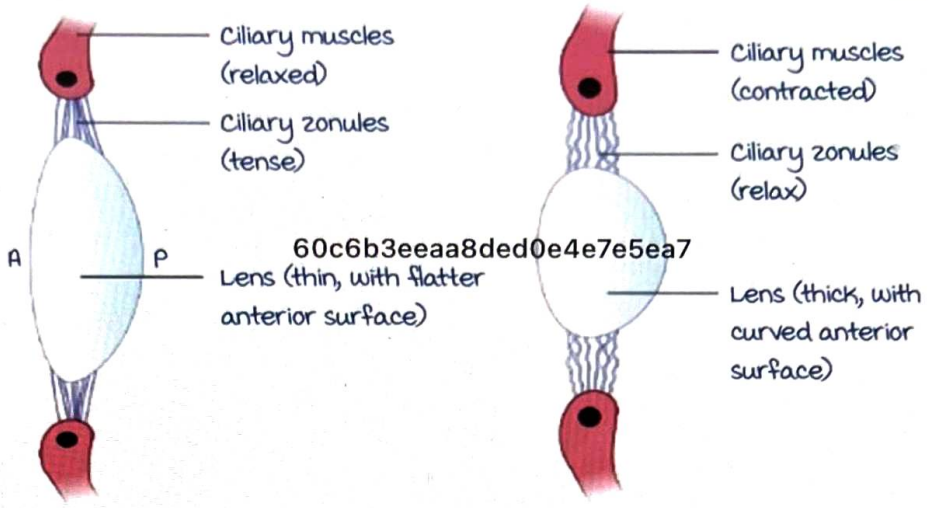
When looking at a far object :

- The zonules are stretched/tense.
- The ciliary muscle is relaxed.
- The anterior surface of the lens becomes flat.
- Parallel rays of light (far vision) are converged by the lens on to the retina.

When looking at a near object :

- more power is required to converge the diverging rays coming from a near object.
- Ciliary muscle contracts.
- The zonules are relaxed.
- The lens's anterior curvature increases and the power of the lens is increased.
- The anteroposterior diameter of the lens is increased.
- No change in the posterior curvature of the lens.

Active space



Anomalies of accommodation

00:53:32

Presbyopia :

It is the physiological insufficiency of accommodation.

Age : Around 40 years of age.

The patient complains of loss / difficulty in the near vision (inability to use mobile).

Treatment : Spectacles : (+)/convex lens.

The power of the spectacles depends on the age :

- 40 years : + 1 D (Every 5 years, power increases by +0.5 D.
- 45 years : + 1.5 D.
- 50 years : + 2 D.
- 55 years : + 2.5 D.
- 60 years : + 3 D.

Types of spectacles :

Case 1 : Myopic patient i.e., already uses spectacles for far vision :

- Bifocal spectacles (for far and near vision).
  Called as D shaped bifocals.
- Progressive spectacles (far, intermediate and near visions)

Bifocal lens Progressive lens

Bifocal and progressive spectacles

Executive bifocals

Executive bifocals :

Upper area : Far vision (concave lens).

Lower area : Near vision (convex lens).

There is a central line present between the two lenses.

Used in cases of paediatric aphakia.

Case 2 : Emmetropic patient : Monofocal spectacles for near vision.

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Spasm of accommodation

01:04:21

Spasm / permanent contraction of ciliary muscles.

There is a continuous state of accommodation.

Near vision is clear.

Far vision is compromised.

Looks like a case of myopia (near-sightedness), thus it causes pseudomyopia.

Treatment : mydriatics-cycloplegics (pharmacological relaxation/paralysis of ciliary muscle).

- Atropine.
- Homatropine.
- Cyclopentolate.

Tropicamide is only a mydriatic.

Active space

ANATOMY OF EYE - INNERMOST LAYER AND BLOOD SUPPLY OF EYE

Retina

00:00:19

Retina is the innermost, incomplete layer of the eyeball that covers the eyes only posteriorly.

It is the neural layer of the eye, consisting of all the nerve fibres that collects to form the optic nerve.

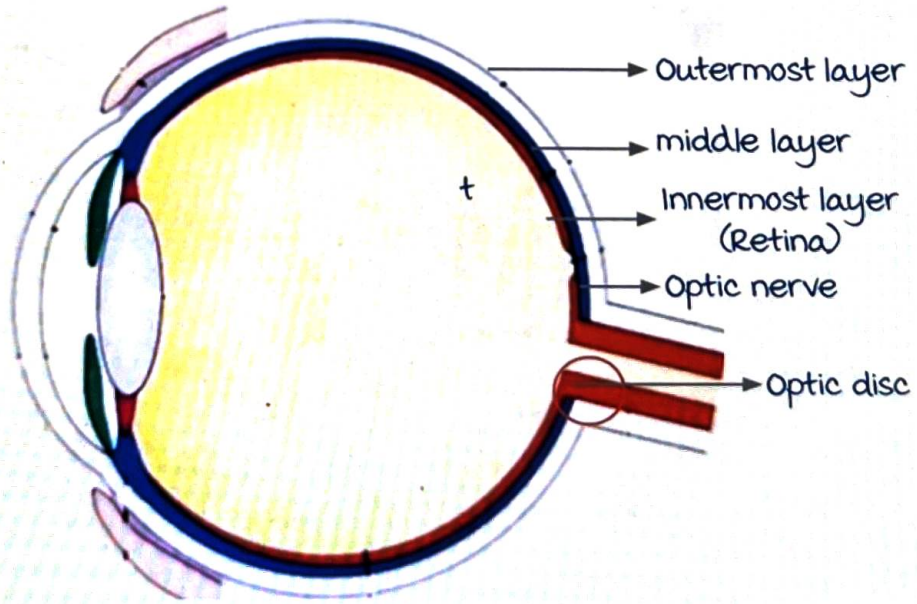
major function is to record the image acting as a film and send it to the cortex via optic nerve.

Gross anatomy of retina

00:02:18

It has 3 parts :

1. Optic disc/blind spot : First part of optic nerve.
   - It's the point from where the optic nerve leaves the eyeball or, the 1st part of the optic nerve.
   - It is also known as the blind spot because there are no rods and cones present here, thus no vision.

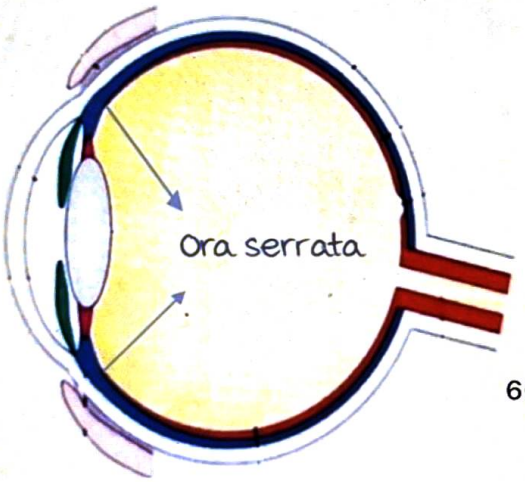


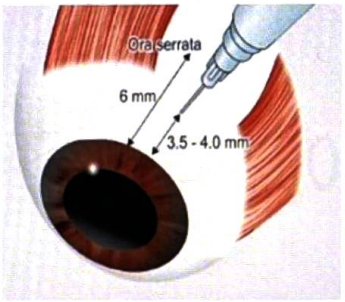
2. Macula :

- It is responsible for central vision.
- In all macular diseases, there will be loss of central vision, example : CME (cystoid macular edema).
- The centre of macula is called fovea.
  Fovea contains maximum concentration of cones, resulting in best image, as it is most sensitive to light.

3. Ora Serrata :

- It's the point of anterior termination of retina, or junction between ciliary body (anterior to ora serrata) and choroid (posterior to ora serrata).
  Hence, retina is an incomplete layer, covers eye only posteriorly.





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- It is the site for intravitreal injection.
  Site of intravitreal injection :
  - Anterior to ora serrata or,
  - Posterior to limbus or,
  - Between ora serrata and limbus or,
  - By piercing sclera and pars plana.

  Distance between limbus and ora serrata is about 6mm.
  Appropriate site for intravitreal injection, with limbus as the landmark :
  1. 3 mm posterior to limbus : If aphakia (absent lens)
  2. 3.5 mm posterior to limbus : If pseudophakia (IOL+).
  3. 4 mm posterior to limbus : If phakia (normal crystalline lens +)

Active space

Steps of intravitreal injection :

1) To have a clear field of view, the eyelid is kept open by eye speculum (held in surgeons left hand) and the infratemporal region of the eye is sterilised by cotton bud dipped in betadine (held in right hand).

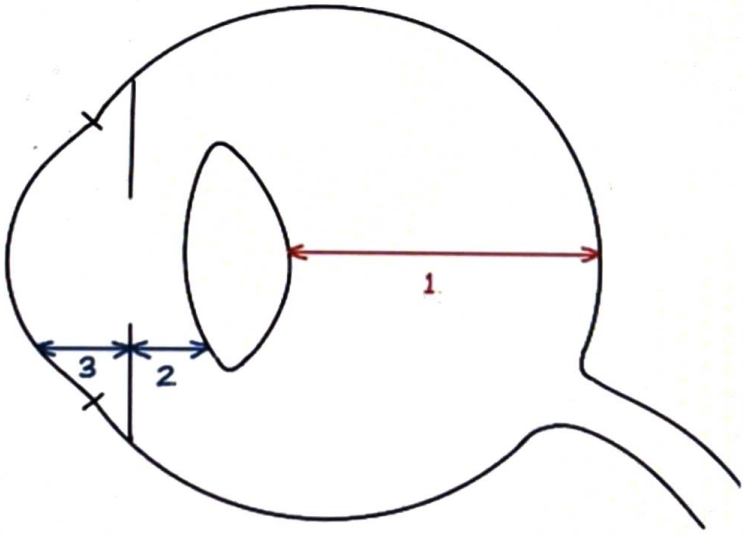
2) The patient is asked to look up for better visualisation of infratemporal region.

3) With a caliper opened to the desired distance, mark a point on the sclera measured from the limbus.

4) With the help of 1 ml syringe and 30 G needle, intravitreal injection is given into the vitreous through the marked point. Injection is done by making slow twisting in order to pierce the sclera.

Spaces inside the eyeball

00:29:46



Space 1 : Posterior segment → Contains vitreous.

Space 2 : Posterior chamber → Space b/w iris and lens.

Space 3 : Anterior chamber → Space b/w cornea and iris.

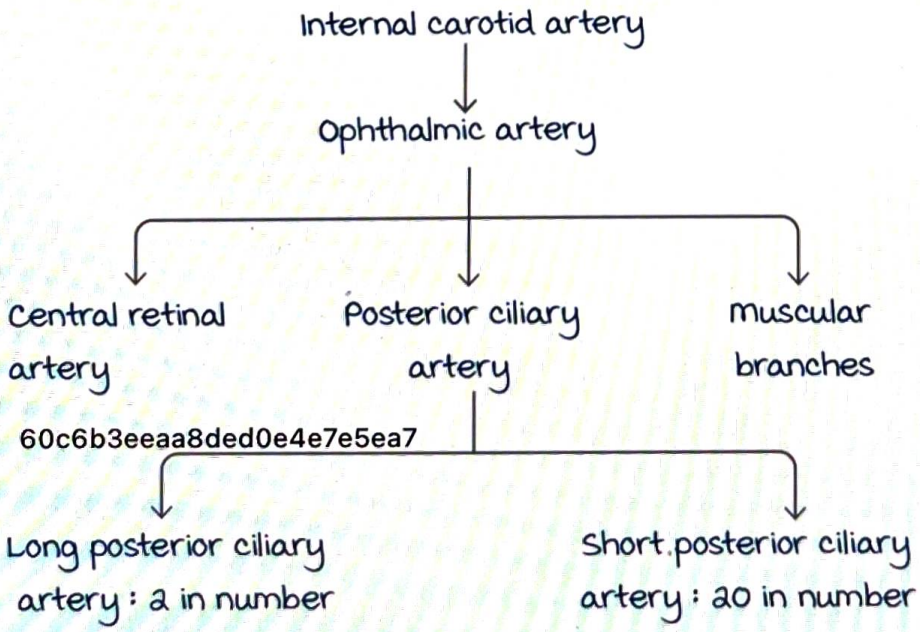
Posterior chamber + anterior chamber → Anterior segment → Contains aqueous humor.

Aqueous	Vitreous
Liquid state	Gel form (it contains Hyaluronic acid)
Volume is 0.31 ml (0.25 ml in anterior chamber and 0.06 ml in posterior chamber	Volume is approximately 4 ml
Rate of secretion is 2.3 microlitre/min; by ciliary processes and outflow is by trabecular meshwork	Not continuously secreted, as it is formed only during the development of eye

- Facility of aqueous outflow (C-value) : 0.18 to 0.25 microlitre/minute.
- Investigation to measure C-value : Tonography.
- Investigation to measure IOP (exerted by aqueous) : Tonometry.
- Anterior chamber depth : Normal – 2.5 to 4.4 mm. Increased in young myopic males.
- Aqueous contains higher concentrations of : Lactate, ascorbate and pyruvate >plasma.

Blood supply of the eye

00:42:00



Central retinal artery travels within optic nerve and supplies the inner 6 layers of retina.

Long posterior ciliary artery : Enters eye by piercing sclera away from optic nerve, travels b/w sclera and choroid and reaches up to ciliary body, divides into 2 branches ⟶ Supplies ciliary muscle.

These 2 branches anastomose near the root of iris, to form major arterial circle of iris and supply the periphery of iris.

Branches of major arterial circle move centrally and form minor arterial circle of iris, that supplies the pupillary margin and some central part of iris.

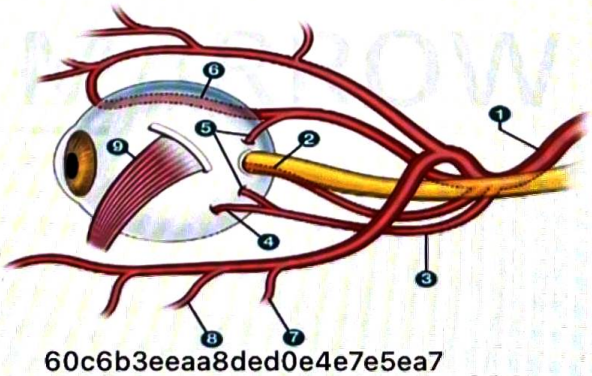
Short posterior ciliary artery : 20 in number, enters eye by piercing sclera around the optic nerve.
Supply sclera, choroid and outer 4 layers of retina.

Muscular branches are total 7 in number :

- 2 each from superior rectus, medial rectus and, inferior rectus.
- 1 from lateral rectus.

All these together form anterior ciliary arteries, pierce sclera 5 mm posterior to limbus and supplies conjunctiva, anterior sclera, part of iris and ciliary body.

Retinal blood supply



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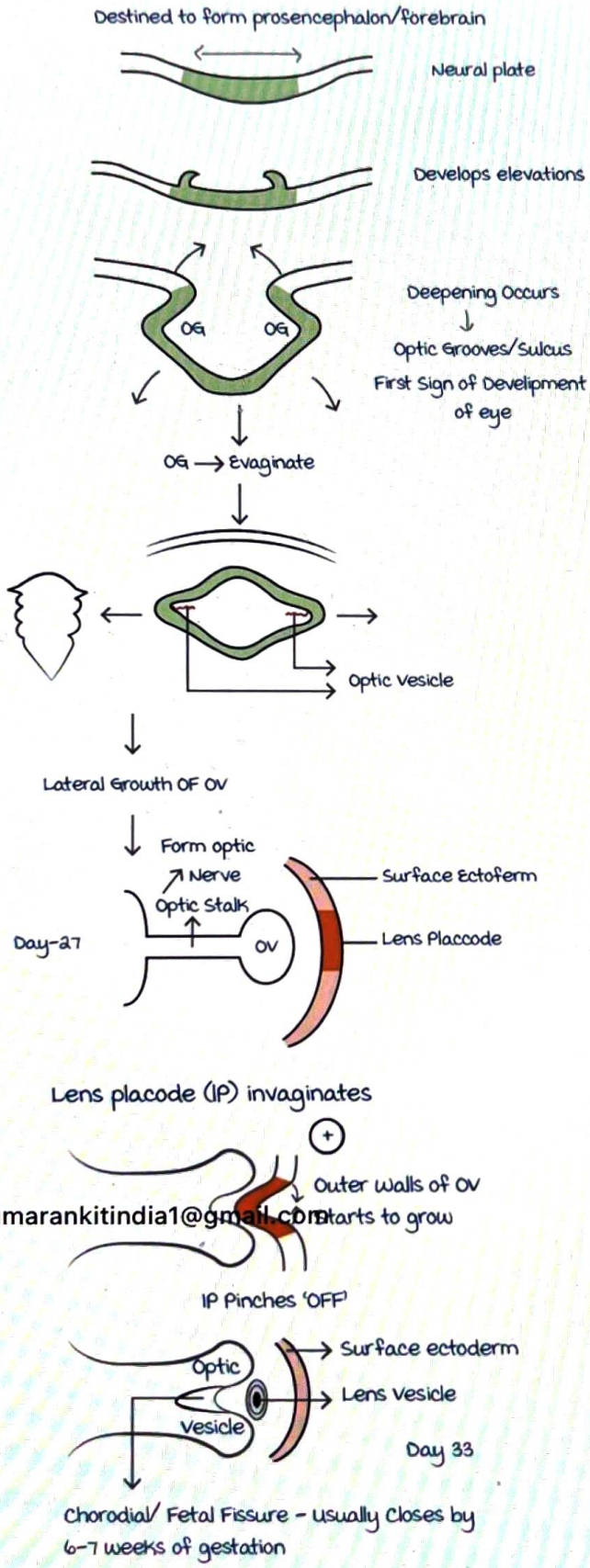
1. Ophthalmic artery.
2. Central retinal artery.
3. Posterior ciliary artery.
4. Long posterior ciliary artery.
5. Short posterior ciliary artery.
6. Anterior ciliary artery.

Active space

Development of the eye

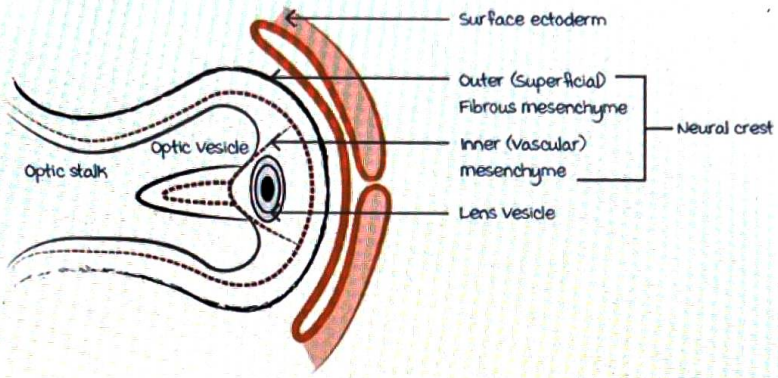
00:59:00

Commences at the 3rd week of gestation (Day 21).



Mesenchyme : Derived from mesoderm or neural crests.
Neural crests : Covering the developing neural tube which forms meninges.
Neural crest mesenchyme - Divides into 2 layers :

- Superficial fibrous layer.
- Deep vascular layer.



Derivatives :

1) Surface ectoderm :

- Epithelium of cornea and conjunctiva.
- Skin of eyelids.
- Lens (pinched inside from surface ectoderm).
- Tarsal glands/meibomian glands.
- Lacrimal glands.

2) Neuro ectoderm :

- Optic nerve.
- Retina : Inner 9 layers (Neurosensory retina from inner wall of optic vesicle) and Outermost layer (Pigment epithelium from outer wall of optic vesicle).
- Vitreous.
- Epithelium of iris and ciliary body.
- Sphincter pupillae and dilator pupillae (smooth muscles of iris).

3) Mesoderm : Extra-ocular muscles (7).
4) Neural crest cells : Everything else.
Ciliary muscle.
Stroma of cornea.
Choroid etc.

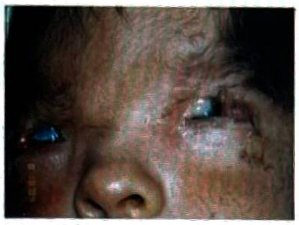
Active space

Coloboma

01:21:03

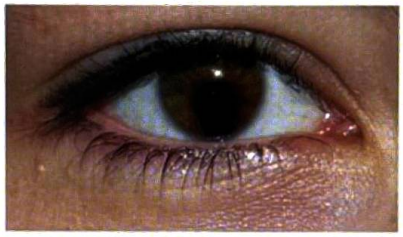
Greek word "Koloboma" i.e., missing part.
Congenital malformation due to non closure of choroidal/fetal fissure by 6-7 weeks of gestation.

Eyelid coloboma :
MC seen in Upper eyelid at a junction of middle and medial 1/3rd.



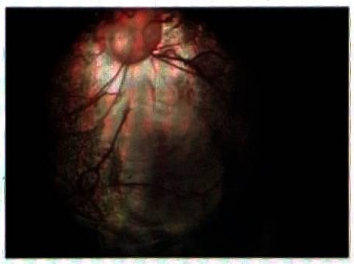
Main complication : Exposure Keratopathy.
Rx : Lubricants/patching/surgical closure of defect, based on the size of the defect.

Iris coloboma : 2 types.
Typical coloboma : Infero nasal coloboma.
Atypical coloboma : Anywhere.



Can be associated with colobomas of ciliary boy/choroid/retina/optic nerve.
Iris coloboma usually : Bilateral.
May lead to nystagmus/squint/glaucoma.
Clinical features :
1) Diplopia : Lens is not present inferiorly. Light gets refracted in 2 places, at +16 D where there is lens, and at 0 D where there is no lens. Thus, 2 images are formed in the retina which manifests as diplopia.
2) Photophobia.
3) Cosmesis.
Rx : Cosmetic contact lens or Surgical closure with sutures.

Retino-choroidal coloboma



Lens coloboma : Not a true coloboma as it is not due to non closure of choroidal fisssure.

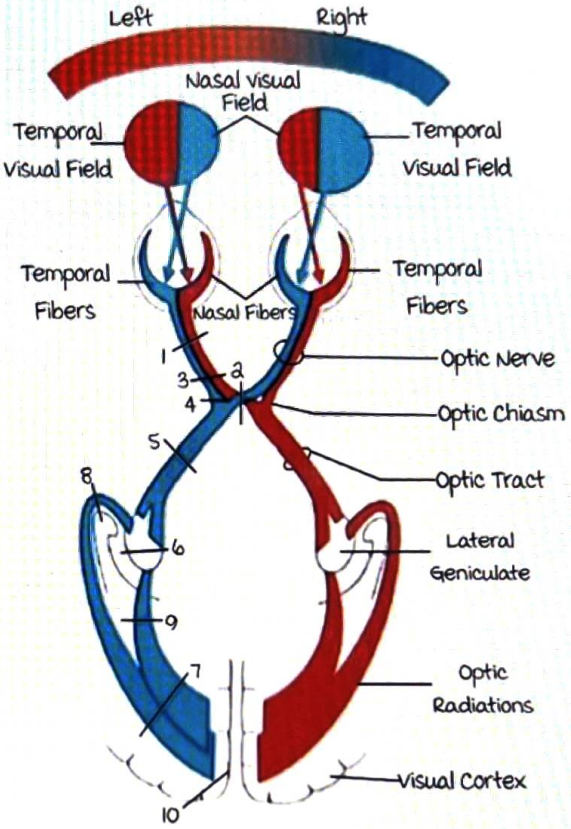
Active space

NEURO-OPHTHALMOLOGY - VISUAL PATHWAY AND VISUAL FIELD DEFECTS

Visual pathway

00:01:35

Unilateral condition → One side is affected.
Bilateral condition → Both sides are affected.
In a unilateral condition :
If same side is affected → Ipsilateral.
If opposite side is affected → Contralateral.



In the image given :
The semilunar structure present in the eyeball is the retina.
The temporal and nasal fibers are shown in different colors.
The nasal and temporal fibers combine to form the optic nerve → Forms the first part of the visual pathway.

The right optic nerve comprises of right nasal and right temporal fibers → Ipsilateral visual fibers.
The nasal fibers from both sides cross over to the opposite side at the optic chiasm.
Optic chiasm contains bilateral nasal fibers.
The temporal fibers continue along the same side.
Optic tract contains ipsilateral temporal and contralateral nasal fibers.

Active space

The fibers from the optic tract relay into the lateral geniculate bodies → Ipsilateral temporal and contralateral nasal fibers.
Optic radiations contain ipsilateral temporal and contralateral nasal fibers.

These eventually relay into the visual cortex, which is primarily in the occipital lobe, with some parts in the parietal lobe as well.
Visual cortex receives ipsilateral temporal and contralateral nasal fibers.

Below the optic chiasm, the fibers in all parts of the visual pathway are ipsilateral temporal and contralateral nasal fibers.

Since light travels in a straight line, the temporal fibers carry the impulse from the nasal visual field → Visual field is opposite to the visual fibers.
For e.g., If there is an injury to the optic chiasm due to the presence of a tumor, the bilateral nasal fibers are affected, leading to visual impairment in the bilateral temporal fields.

Visual field defects

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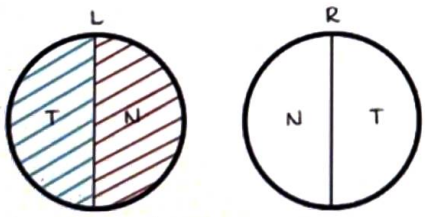
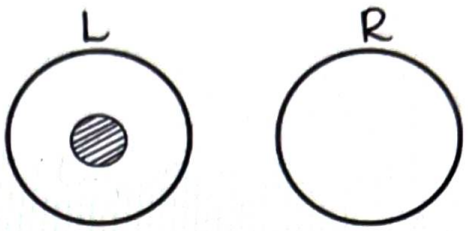


Lesion I → At the left optic nerve.
Left optic nerve contains left (ipsilateral) temporal and left (Ipsilateral) nasal fibers.
Loss of left temporal fibers result in loss of left nasal field.
Loss of left nasal fibers result in loss of left temporal field.
Causes left (ipsilateral) anopia i.e., optic nerve lesions cause ipsilateral anopia.

Active space



Lesion I (a) :

Earliest visual fibers affected in any optic nerve disease are macular fibers.

In an early optic nerve lesion of the left eye, there is central visual loss in the same eye.

Causes left (ipsilateral) central scotoma.

Scotoma → used to describe a black area in the visual field.

Causes of central scotoma :

- Optic neuritis → most common cause is multiple sclerosis.
- Hereditary optic neuropathy.
- Toxic/nutritional optic neuropathy.
- Cone dystrophy (stargardt's disease).

Optic nerve

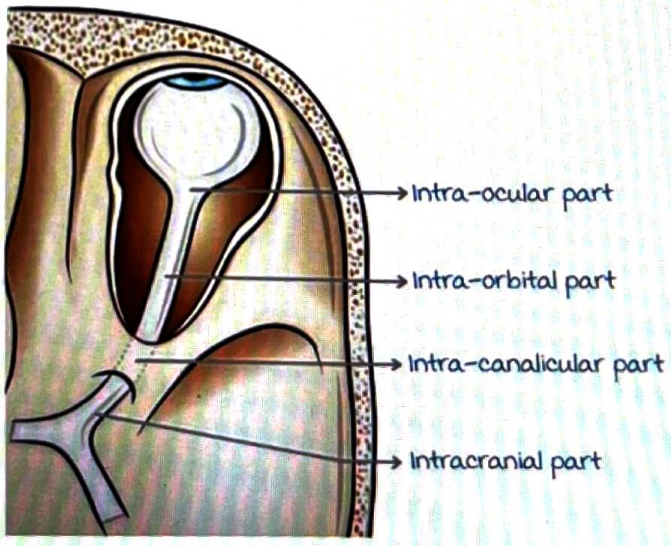
00:26:51

Anatomy of optic nerve :

Cranial nerve that is a collection of axons of retinal ganglion cells.

Total length → 47–50 mm.

Can be divided into four parts :

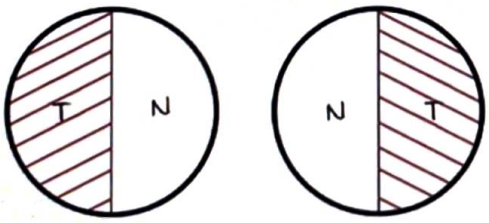


Intra-ocular part/optic disc → 1st part of the ocular nerve.
Smallest part → 1 mm in length.
Diameter is 1.5 mm.
Intra-orbital part :
Longest part → 25-30 mm.
Optic nerve acquires myelin sheath → Diameter increases to 3-4 mm.
Central retinal artery and central retinal vein, enter the optic nerve by piercing inferomedially.
Intra-canalicular part.
Intra-cranial part → Lies above the cavernous sinus.

Optic nerve function tests :
Light brightness comparison test :
Light is shone in each eye, using an indirect ophthalmoscope.
The patient is then asked if the light appears :
Equally bright in both eyes → Both optic nerves are normal.
Dissimilar in brightness → Optic nerve disease seen in the eye with least brightness.

Visual field defects

00:35:59



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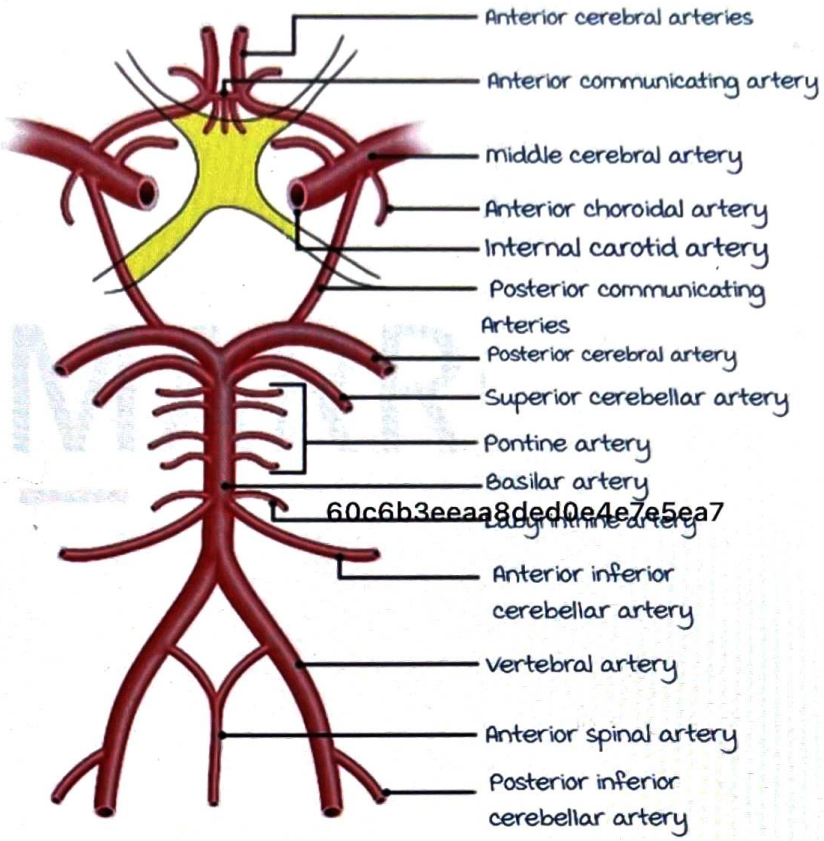
Lesion 2 → At the optic chiasm.
Fibres affected → Bilateral nasal fibres are affected.
Causes bitemporal hemianopia.
AKA heteronymous hemianopia.
This is because the sides of the visual field loss are opposite.
The left side loses left side of visual field and right side loses right side of the visual field, such that both eyes lose the vision in opposite sides.

Active space



Causes of bitemporal hemianopia : Buy 3PC and AC.

Bitemporal hemianopia.

3rd ventricular glioma.

Pituitary adenoma.

Microadenoma : < 1 cm/ 10 mm → Does not cause bitemporal hemianopia.

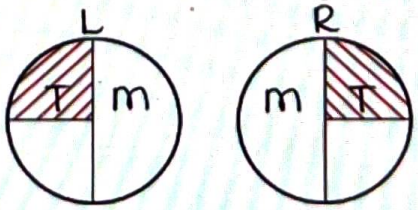
Macroadenoma : > 1 cm/ 10 mm → Causes bitemporal hemianopia.

Pituitary adenoma is the inferior relation of optic chiasm.

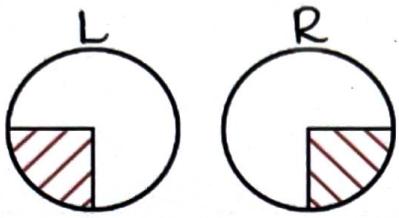
Craniopharyngioma occurs in Rathke's pouch (superior relation to optic chiasm).

Anterior cerebral/communicating artery aneurysm.

Cavernous sinus thrombosis.



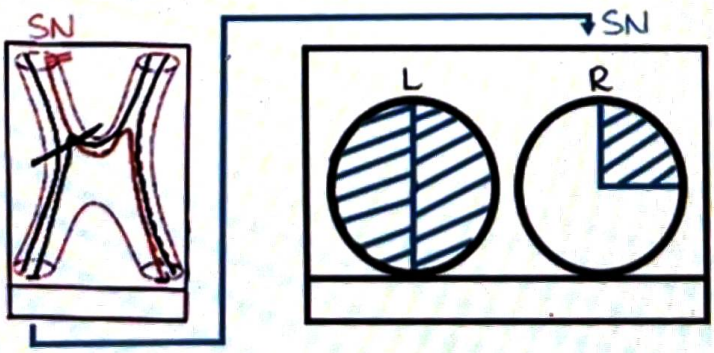
Lesion 2 (a) → Earliest lesion by pituitary adenoma.
As the pituitary gland is an inferior relation, the earliest fibres affected are inferonasal fibres.
Causes superotemporal visual field defect.
Bitemporal superior quadrantanopia.



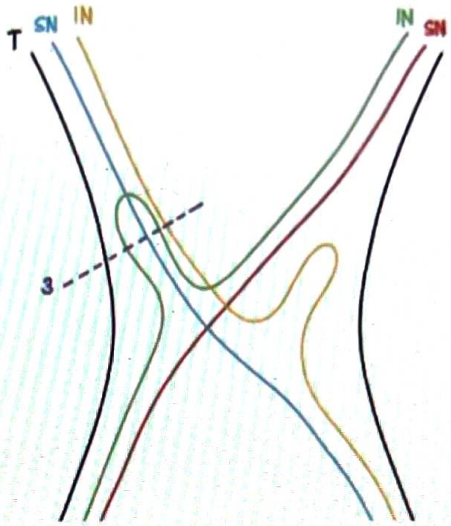
Lesion 2 (b) → Earliest lesion of craniopharyngioma.
Earliest fibres lost : Superior part of bilateral nasal/bilateral superonasal fibres.

Visual field defect → Inferotemporal.
Bitemporal inferior quadrantanopia.

Lesion 3 → At the junction of optic nerve and optic chiasm.
Also called lesion of proximal optic nerve/lesion of anterior Knee of von Willebrand.
Fibres affected → Left temporal and nasal fibres, along with right inferonasal fibres i.e., Ipsilateral temporal and nasal fibres with contralateral inferonasal fibres.
Visual field defect → Ipsilateral anopia and contralateral superotemporal quadrantanopia.
AKA Junctional scotoma.
Most common cause of junctional scotoma → Meningioma.



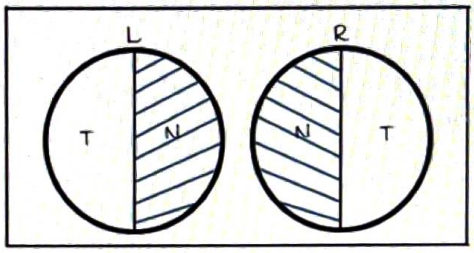
Active space



Junctional scotoma

Visual field defects

01:01:09



Lesion 4 → Lateral part of optic chiasm.

Always bilateral.

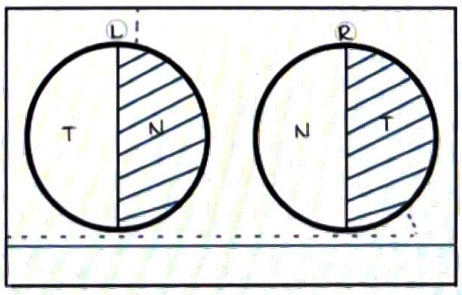
Fibers affected → Bilateral temporal fibers.

Visual field defect → Binasal hemianopia.

This is the rarest visual field defect.

Causes :

- Hydrocephalus.
- Primary empty sella syndrome.



Lesion 5 → At the optic tract.

Lesion 6 → At the lateral geniculate body.

Lesion 7 → At the optic radiations.

In all the three, since the fibers are same, the fibers lost are ipsilateral temporal (left) and contralateral nasal (right).

Visual field defect → Right (contralateral) homonymous hemianopia → Loss of right visual field in left eye and right eye.
Contralateral as the lesion is on the left side and defect on right.

To localise between the lesions :

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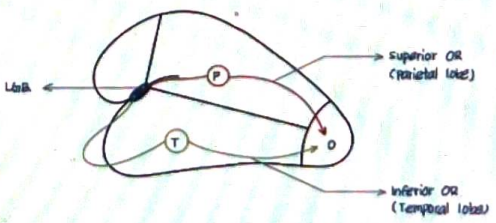
Features	Optic tract lesion	Lateral geniculate body lesion	Lesion at the optic radiations
Pupil	Abnormal (Wernicke's pupil)	Normal	Normal
Congruity	Incongruous	Incongruous	Congruous
Optic atrophy	Bilateral optic atrophy as optic nerve is a 3rd order neuron, and so is the optic tract.	Bilateral optic nerve atrophy, as it is also a 3rd order neuron.	No optic atrophy, as it is 4th order neurons.

Congruity signifies the extend and pattern of visual loss between both the eyes, in temporal and nasal fields.

Optic tract lesion :
Associated with 3rd nerve involvement and contralateral hemiplegia (internal capsule involvement).
Causes :

- Syphilitic meningitis.
- Tubercular meningitis.
- Posterior cerebral artery aneurysm.

Lesion 8 and 9 :
Lesion at 8 + Lesion at 9 = Lesion at 7.
The optic radiations carry the impulse from the lateral geniculate body to



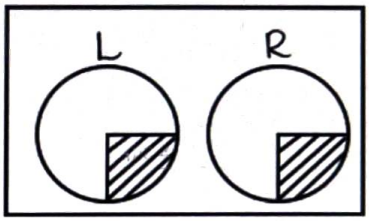
Active space

the visual cortex at the occipital lobe.

Superior optic radiations are fibres passing superiorly, via the parietal lobe.
Inferior optic radiations are fibres passing inferiorly, via the temporal lobe.

Superior optic radiations are composed of superior part of ipsilateral temporal and contralateral nasal fibres.
Inferior optic radiations are composed of inferior part of ipsilateral temporal and contralateral nasal fibers.

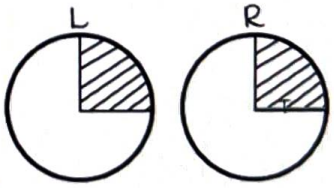
Lesion 8 → At superior optic radiation.
AKA Lesion of optic radiation passing through parietal lobe/ Baum's loop.
Fibres affected → Ipsilateral superotemporal and contralateral superonasal.



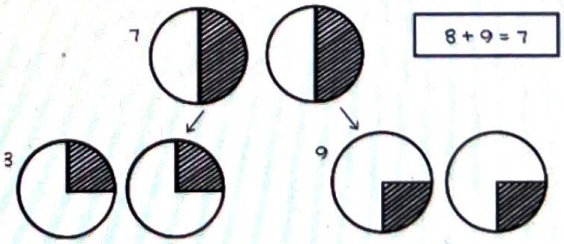
Visual field loss → Right (contralateral) inferior homonymous quadrantanopia.
AKA Pie on the floor visual defect.
Parietal lobe lesions cause :

- Pie on the floor visual defect.
- Agraphia, acalculia, agnosia, apraxia.

Lesion 9 → At inferior optic radiation.
AKA Lesions of optical radiation passing through temporal lobe/ meyer's loop.
Fibres lost → Ipsilateral inferotemporal and contralateral inferonasal fibres.



Visual field loss → Right (contralateral) superior homonymous quadrantanopia.
AKA Pie in the sky.



Active space

Mnemonic → TIPS.

Temporal lobe.

Inferior optic radiation.

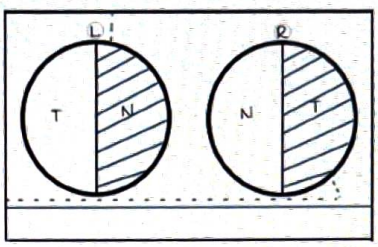
Pie in the Sky.

Temporal lobe lesion :

- Pie in the sky visual defect.
- Uncinate fits, seizures and hallucinations.

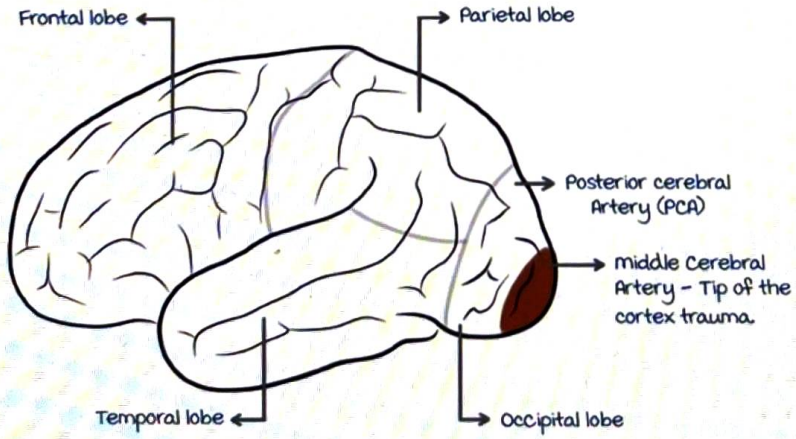
Lesion at visual cortex

01:31:02



Lesion IO → At the visual cortex.
Fibres affected → Ipsilateral temporal and contralateral nasal fibres.
Visual field defect → Right (contralateral) homonymous hemianopia.
This rarely occurs, as such extensive loss of visual cortex is rare.

Occipital lobe is supplied by the posterior cerebral artery, except the tip of the occipital cortex.
The tip is supplied by middle cerebral artery.
Trauma to the tip is common in cases of trauma.



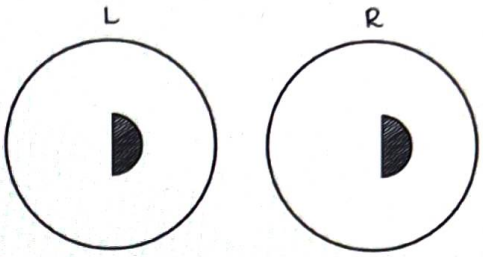
Tip of cortex = Macula.

Active space

Lesion IO (a) → At the tip of the cortex.

Fibres affected → macular representation of ipsilateral temporal and contralateral nasal fibres.

Visual field defect → Right (contralateral) homonymous macular defect.
AKA Cookie cutter defect.



Lesion IO (b) → At the visual cortex except the tip.
Area supplied by posterior cerebral artery.
Fibres affected → Ipsilateral temporal and contralateral nasal fibres except macular representation.
Visual field defect → Right (contralateral) homonymous hemianopia with macular sparing.

Miscellaneous lesions

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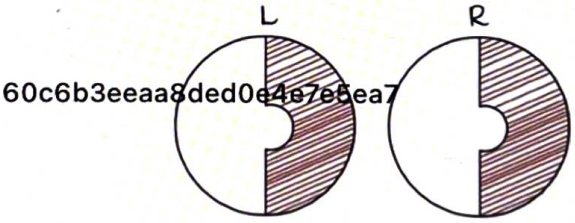
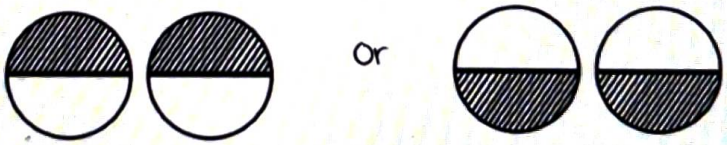


Altitudinal visual field defects :



Superior Hemianopia Inferior

Respect the horizontal midline.
Can be superior or inferior hemianopia.
Diseases causing it : (mnemonic → GOA).

- Glaucoma.
- Optic disc drusen.
- Anterior ischemic optic neuropathy.

Active space

Cortical blindness :

- Bilateral occipital lobe lesion.
- Has no vision.
- Patient is in denial of blindness → Anton's syndrome.
- Suffers from Riddoch phenomenon → Patient is able to appreciate kinetic movement but not static.
- VEP (visual evoked potential) → used to diagnose and prove the patient has cortical blindness.
- The electrodes placed in the occipital lobe would generate impulse on visual stimulation.
- Cortical blindness → VEP absent.
- Can be used to differentiate between organic and functional blindness.

  Organic blindness → Cortical blindness.

  Functional blindness → malingering → Person with normal vision pretends to be blind.

Enlargement of blind spot :

Blind spot is optic disc.

Causes :

- Papilledema.
- Papillitis.
- Congenital anomalies of optic disc.
- Optic disc drusen.

Cogan's dictum :

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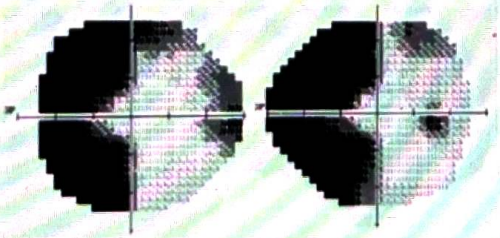
Optokinetic nystagmus test (OKN) is done.

Nystagmus is induced in this test, and the movements are observed.

Symmetric movement → Lesion is in occipital lobe.

Erratic pursuit → Lesion in parietal lobe.

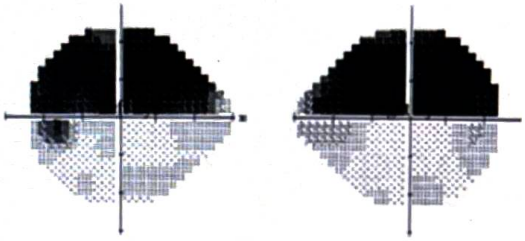
Diagnose the site of the lesion.



Active space

Left homonymous hemianopia with macular sparing.
Right sides visual cortex involvement without the tip.

Identify the lesion given below.



Superior altitudinal hemianopia.

NEUROOPHTHALMOLOGY PUPILLARY REFLEXES, LIGHT REFLEX PATHWAY AND ITS LESIONS

Pupillary reflexes

00:00:52

1. Near Reflex : Changes taking place in eye while looking at a near object.
   - Accomodation + convergence + miosis (constriction).

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2. Cilio-spinal reflex : Ipsilateral dilatation of pupil.
   - When there is pain in neck/trunk because of any action, I/L pupil will dilate.
   - Lost in Horner's syndrome.
3. Psycho-sensory reflex : Dilatation of pupil in response to stress.
4. Light reflex : Pupils constrict (to control the excess amount of light getting into the eye), when exposed to sunlight.

Note : Pupils dilate in dark/dim light in order to let more light get into the eye.

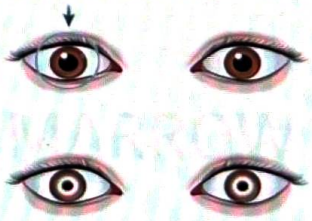
Function of pupil : Test the light reflex (dark room procedure) :

- Ask the patient to sit in a dark room for 5-10 mins → Pupil fully dilated → Ask patient to hold his/her hand in between eyes → Take a torch light and throw some light in both the eyes seperately → Both pupils constrict.

Light Reflex : Types

00:06:10

1. Direct light reflex : Constriction of Ipsilateral pupil when illuminated.



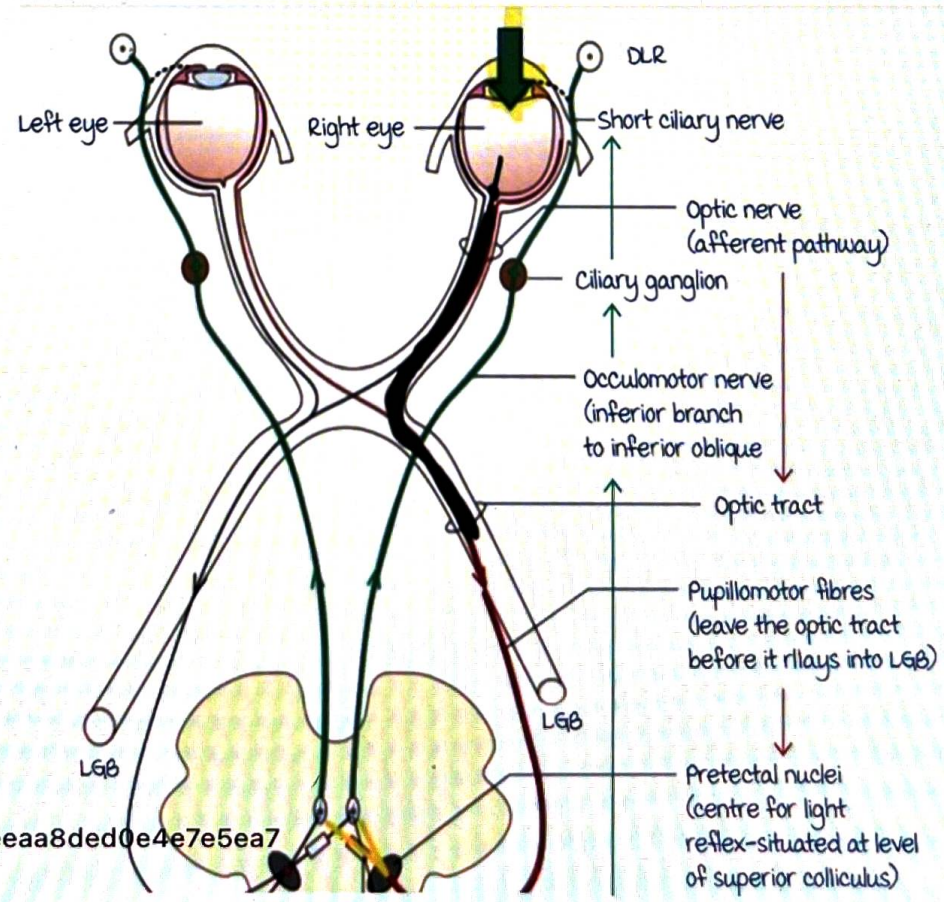
2. Consensual light reflex : "Self will" : Constriction of contra-lateral pupil, which was not illuminated.

When light is thrown on one eye → Pupil of that eye constricts (direct light reflex) + Pupil of the other eye also constricts (consensual light reflex).

Pathway for light reflex :

Light thrown on the right eye → Lights falls onto the retina → Optic nerve → Temporal part of optic chiasma → Right optic tract → Visual fibers into the lateral geniculate body + Pupillomotor fibers go into the pretectal nuclei (Centre for light Reflex).

Pretectal nuclei → Impulse travels to the right and left side (ipsilateral and contralateral) into the Edinger Westphal nucleus → Direct + consensual light reflexes through internuncial neurons → Respective 3rd cranial nerve up to the ciliary ganglion → Short ciliary nerve → Innervates both pupils → Pupils constrict.



Neurons connecting Pretectal and Edinger Westphal nucleus/EWN : Internuncial neurons → Half of them go to the Ipsilateral EWN and half go to the contralateral EWN.
EWN = Accessory nucleus of 3rd cranial nerve.

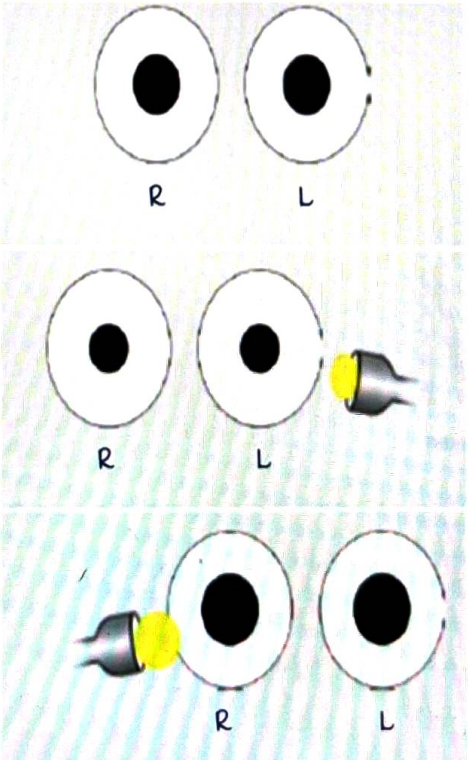
- Afferent nerve = 2nd cranial nerve.
- Efferent nerve = 3rd cranial nerve.
- Structure responsible for Consensual light reflex = Internuncial neurons > Edinger Westphal nucleus.

Lesions of the optic pathway

00:16:10

Afferent pathway defect :

1. AAPD/TAPD/Amaurotic Pupil : Absolute/Total Afferent Pathway Defect –
   - Due to complete optic nerve lesion (End stage optic atrophy/severe trauma).
   - On presentation, both pupils would be equal size (isocoria). As one optic nerve would be intact → Both eyes would be constricted.
   - On light reflex test:
     When light thrown on unaffected eye → Both pupils constricted.
     When light thrown on affected side → No light can go down the nerve → Both pupils not responding.



   - Clinical features:
     1. Isocoria.
     2. Ipsilateral anopia.

Active space

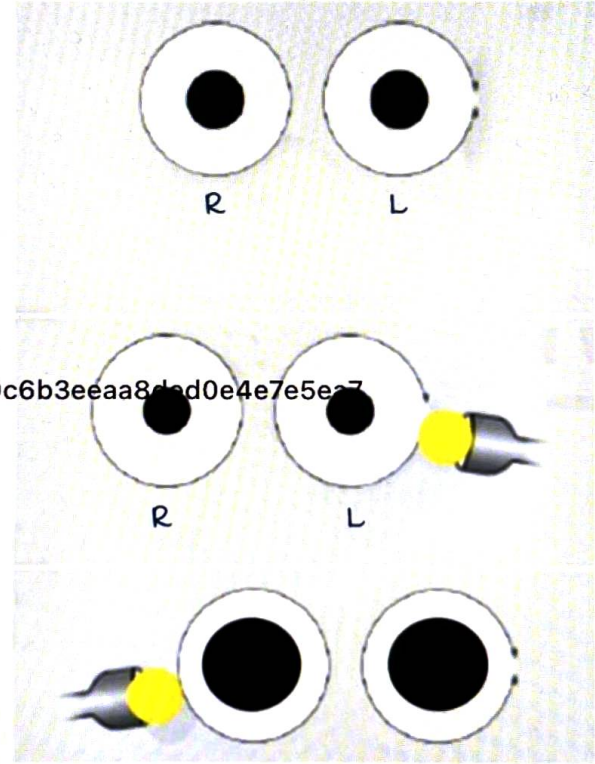
3. Light reflex absent : I/L direct light reflex absent + C/L consensual light reflex absent
4. Near reflex still present.

RAPD

00:26:10

RAPD/Marcus Gunn pupil : Relative afferent pathway defect :

- Incomplete lesion.
- Caused by : Incomplete optic nerve lesion (Optic Neuritis, Retinal Detachment, CRAO, CRVO).



Swinging torch light/flash light test

00:31:25

- Test to diagnose RAPD = Swinging torch light/flash light test.
- On presentation, both pupils would be equal size (Isocoria).
- On light reflex test :
  When light thrown on unaffected eye → Both pupils

constrict.

When light thrown on affected side → Both pupils dilate. As the optic nerve is partially functional, On repeated swinging, affected optic nerve fatigues earlier than normal nerve.

Thus,

1. Light on affected eye → Both pupils constrict.
2. On withdrawing light → Both pupils start to dilate.
3. Light again on affected eye → Impulse for constriction gets weaker than impulse which is already happening for dilatation → Pupils dilate.

- This is known as pupillary escape phenomenon/ paradoxical dilatation.

Wernicke's hemianopic pupil 00:41:02

- Site of lesion : Optic tract → C/L hemianopsia.

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a. Light thrown in hemianopia visual field which is lost → Pupils do not constrict.

b. Light thrown in hemi visual field which is present → Both pupils constrict.

Efferent pathway defects 00:46:16

1. Argyll Robertson's pupil :

- Caused by :
  1. Neurosyphilis (tertiary stage of syphilis).
  2. Tabes dorsalis.
  3. Aortic regurgitation.
- Site of lesion :
  1. Rostral midbrain (Peri-aqueductal grey matter : They accommodate the internuncial neurons.

2. Posterior Commissure.

- Clinical features :
  1. No light reflex : B/L.
  2. Near reflex : present.
  3. Pupil does not respond to miotics/mydriatics (pharmacological).
  4. Pupil on presentation, are B/L constricted.
     a. Because lesion is in the rostral mid brain → Also lesions the supranuclear inhibitory fibers to EWN → EWN is not receiving any inhibitory impulse → It keeps giving impulse to 3rd nerve → Ciliary ganglion → Short ciliary nerve → Both pupils remain constricted.

Absent light reflex + present near reflex : Light-near dissociation (accommodation reflex present, pupillary light reflex absent)

Inverse ARP :

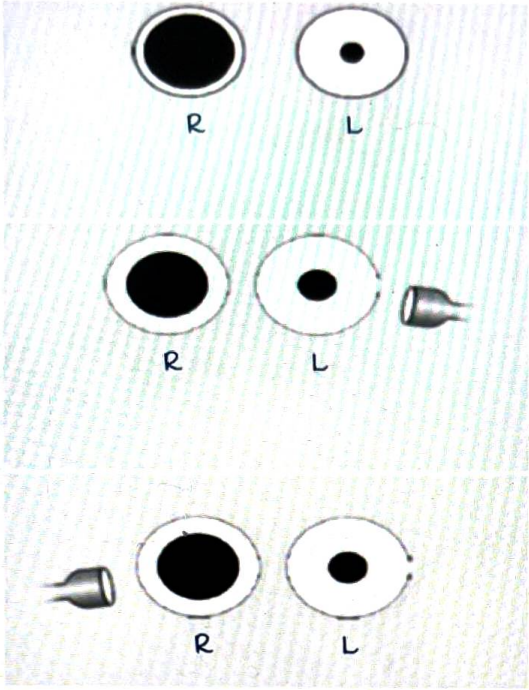
- Acomodation reflex : Absent.
- Light reflex : Present.
- Lesion : Occipito-tectal tract.

2. Tonic pupil/Adie's pupil

00:54:22

- Site of lesion : Either ciliary ganglion or short ciliary nerve.
- Causes:
  1) Herpes Zoster virus (HZV) ganglionitis.
  2) Trauma to the orbits.
  3) Neuropathy (peripheral/autonomic) due to diabetes or alcoholism.
- On presentation/light reflex, in whichever eye the light falls/is thrown, affected pupil will never constrict and the unaffected side will constrict, as normal.

- Both pupil will be of different size (anisocoria).



Clinical features:

1. Light reflex : Absent (affected eye : Both direct and consensual reflexes of the affected eye are absent).
2. Near reflex : Present, but shows slow, vermiform, redilating movements.
3. Denervation hypersensitivity : Constricted to diluted Pilocarpine.

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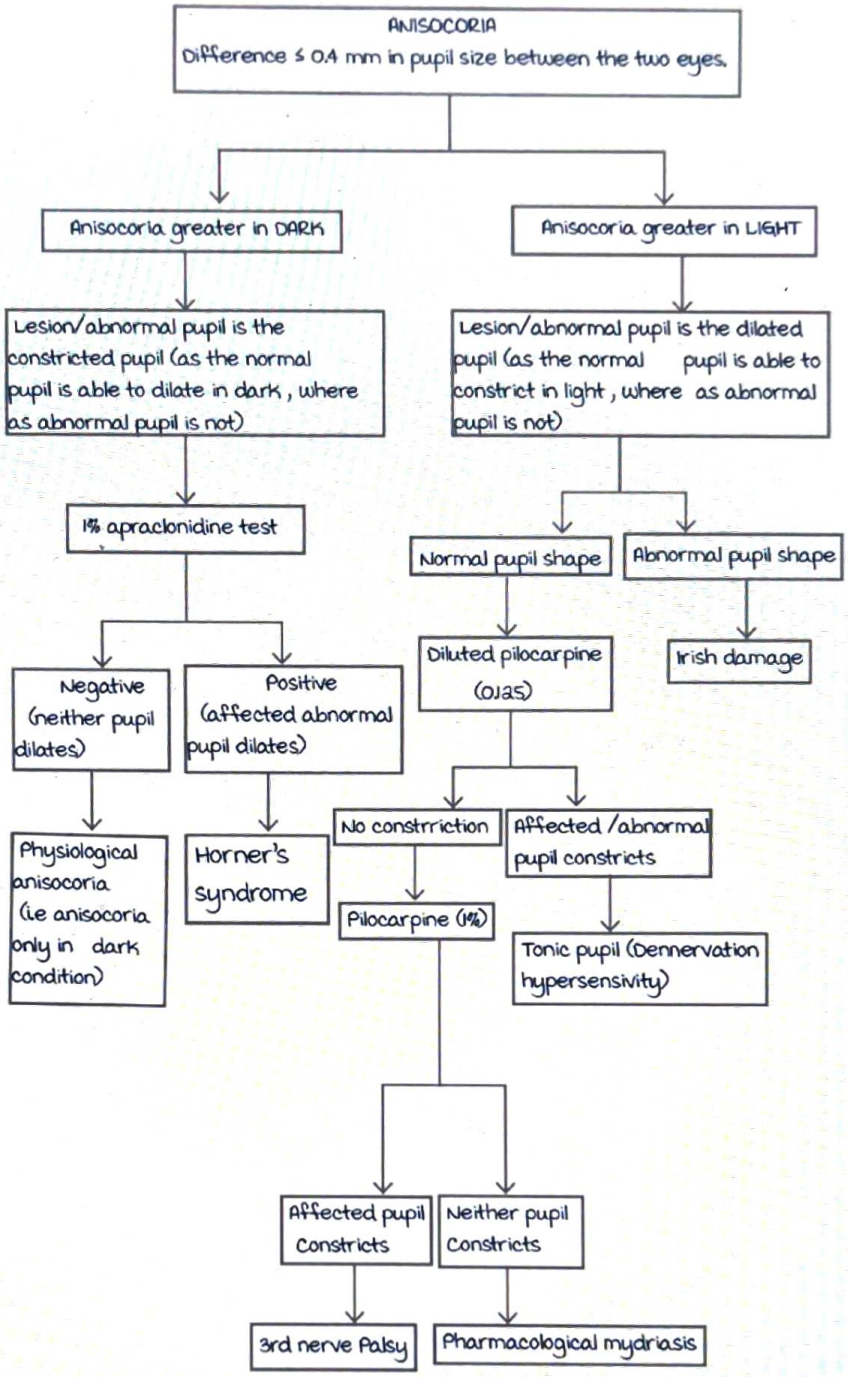
3. Pharmacological mydriasis 01:02:16

- Due to use of mydriatic agents : Atropine
- Light reflex : Affected eye never constricts, unaffected eye constricts normally (similar to tonic pupil).
- Near reflex : Absent (Atropine : Cycloplegic : No accommodation : No near reflex).
- Does not constrict even with 1% pilocarpine.



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NEURO OPHTHALMOLOGY - OPTIC ATROPHY

Optic atrophy

00:00:34

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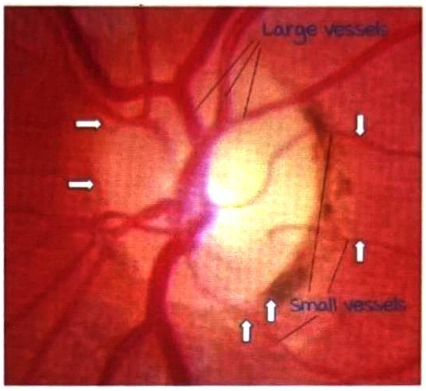
Atrophy of optic nerve.

Signs of optic atrophy :

- Pallor of optic disc.
- Kestenbaum index :
  1. Normally small blood vessels : 7 – 10.
     In optic atrophy : < 7.
  2. Visual loss in optic atrophy does not correlate.



Types of optic atrophy :

	Primary optic atrophy	Secondary optic atrophy	Consecutive optic atrophy
	Optic atrophy without any antecedent optic disc swelling.	Optic atrophy due to existing Optic disc swelling.	Optic atrophy due to diseases of inner retina and its blood supply.
Color of Optic disc	Whitish / pale Yellow, margins are clear	Dirty grey and blurring of disc margins.	Waxy pale optic disc.
Causes	• Optic neuritis (mc/c multiple sclerosis). • Trauma. • Tumor. • Toxic agents. • Heriditary.	• Papilledema • Papillitis. • Anterior Iscemic Optic neuropathy (AION).	• Retinitis pigmentosa • CRAO / CRVO.

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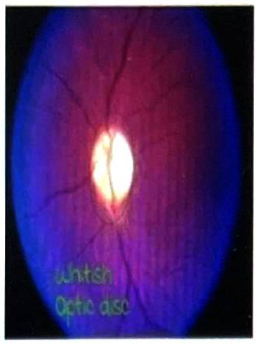
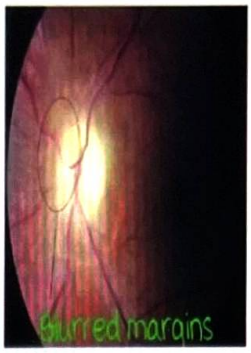
Images			
			

Optic neuritis

00:14:30

Inflammation of optic nerve.

3 types :

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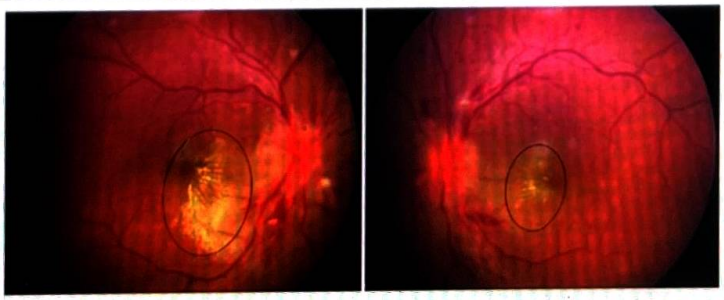
Types	Description	Inflammation of	
		Optic disc	2nd, 3rd, 4th part of optic nerve
Papillitis :	MC in children. Hyperemia (congestion) present	Present	Absent
Retro-bulbar neuritis :	Loss of vision. Nothing can be visualized. MC in adults.	Absent	Present
Neuro retinitis :	macular star appearance.	Present : Inflammation of optic disc along with macula.	Absent



macular star appearance

Causes of macular star appearance :

- Hypertensive rentinopathy.
- Papilledema.
- Retinal angioma.
- Neuro retinitis.

Clinical features OD optic neuritis 00:21:06

Mnemonic : a (CUP) mar.

C : Central scotoma.

C : Color blindness (Red > Green).

U : Unilateral visual loss → Sudden, painless.

U : Uhthoff sign → Exacerbation on increased body temperature.

P : Pulfrich sign → Kinetic objects perception is affected.

P : Pain on movement of eye.

Mar : Marcus Gunn pupil (RAPD) → Earliest sign.

Treatment :

1) Optic neuritis treatment trial (ONTT) :

- IV methylprednisolone 1g daily for 3 days.

followed by

- Oral prednisolone 1 week/ Kg/ day for 11 days.

Faster recovery.
Less chance of recurrence.
Final visual outcome : Not affected.

Only oral steroids should not be given as they increase chance of recurrence.

ONTT trial should be started if :

1. Visual loss < 6 / 12.
2. Previous episode of ON / MS.
3. At least 1 lesion : Demyelinating lesion on MRI.

2) CHAMPS :

β interferon 1a → Reduces the chances of multiple

sclerosis after optic neuritis.

3) Glatiramer acetate (20 mg S.C).

Hereditary optic neuropathy 00:31:13

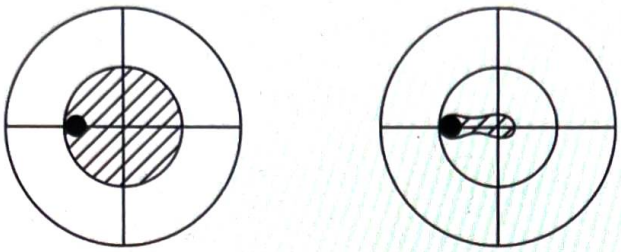
Name	Inheritance	Systemic association
KJER	AD	
BEHR	AR	ANM : Ataxia. Nystagmus. mental retardation.
WOLFRAM	AR	DI DM OA D Diabetes Insipidus, Diabetes Mellitus, Optic Atrophy. Deafness.
LEBER (LHON)	mitochondrial : maternal transmission → MC transmitted to son.	C/F : 6P's Visual loss : Bilateral, painless. Occurs due to point mutation at 11778 codon. Peripapillary Telangiectasia. Pseudo papillitis. Pathognomic sign : Papilo-macular nerve fibre bundle dropout. Primary optic atrophy. No treatment.

Toxic or nutritional optic neuropathy 00:40:04

Causes :

- Deficiency of vitamin B_1/ B_2/ B_6/ B_{12}.
- methanol poisoning.
- Drugs : DICE.
  Digoxin.
  Isoniazid.
  Chloroquine and Ethambutol.

Centro-cecal scotoma > Central scotoma.



Central Scotoma

Centrocecal Scotoma

Treatment : 1000 units of hydroxocobalamin weekly for 10 weeks.

Papilledema

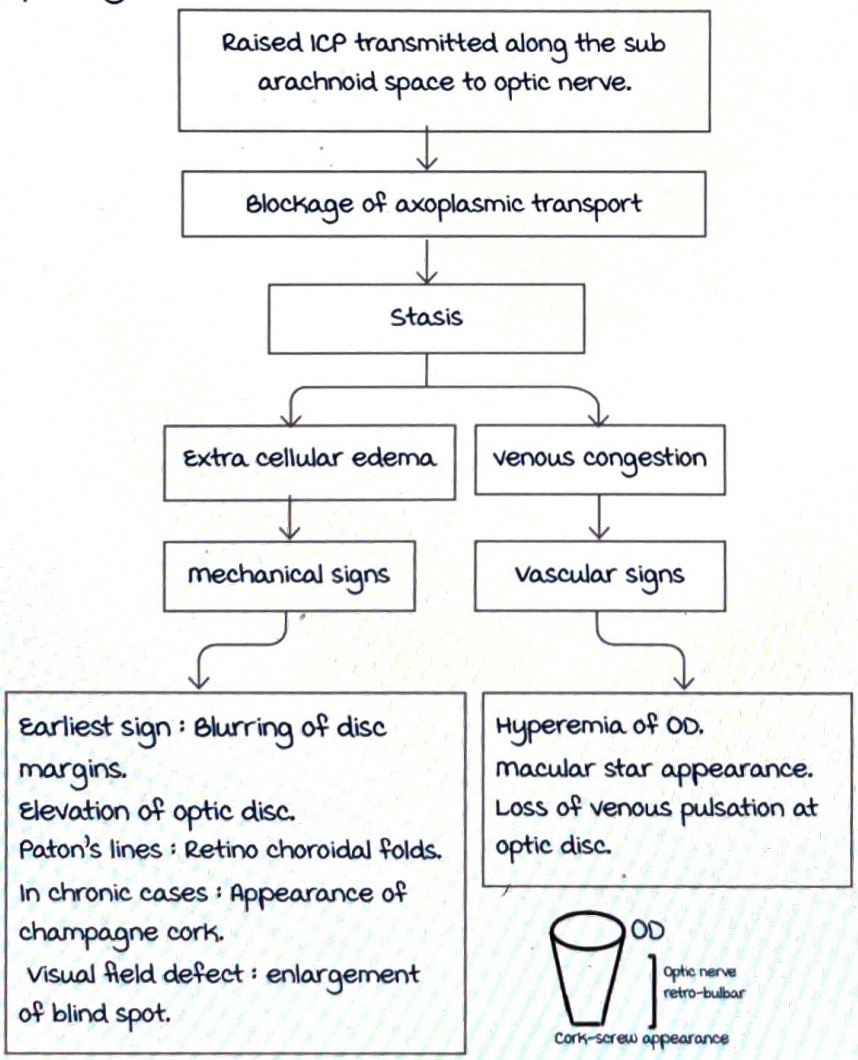
00:44:15

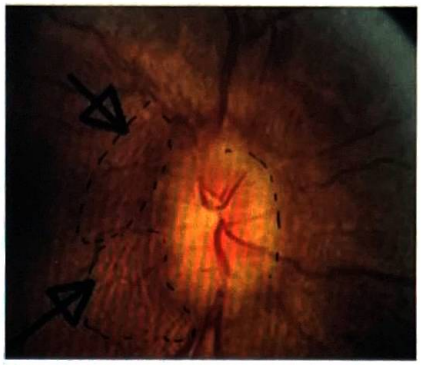
Swelling of optic disc due to raised intra cranial pressure.

Non inflammatory swelling → Painless.

Always bilateral except Foster-Kennedy syndrome.

Etiopathogenesis :

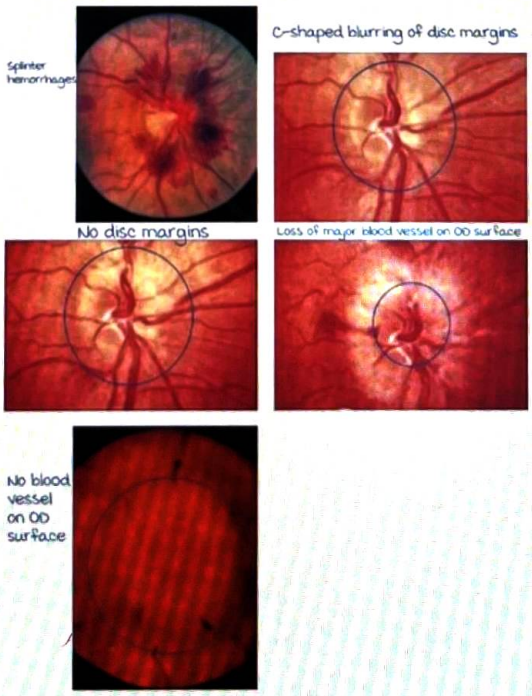




Paton's lines

Symptoms of papilledema 00:56:50

- Earliest : Severe headache (Worse in morning).
- Projectile vomiting.
- Vision : Normal (blind spot is affected).
  Papilledema → Secondary optic atrophy → Gradual painless loss of vision.
- Normal pupillary reaction.
- Horizontal diplopia due to associated 6th CN palsy.
- Amaurosis fugax : Transient visual loss.



Frisen's grading

Anterior ischemic optic neuropathy 01:05:19

Arteritic AION	Nonarteritic AION
Due to giant cell arteritis.	Due to occlusion of short posterior ciliary artery.
Sudden, severe visual loss (<6/60)	Sudden, moderate visual loss (>6/60, non progressive)
m/c in females, 7th – 8th decade	m/c in males, 5th – 6th decade
Premonitory symptoms : Pain, amaurosis fugax present	Absent
Usually bilateral : Other eye affected within days to weeks.	Usually unilateral (>70%).
Pale optic disc	Hyperemic optic disc with small cup
Associated with headache, palpable and tender, non pulsatile temporal artery, CN palsies and jaw claudication	Associated with hypertension, diabetes, hyperlipidemia, collagen vascular disease, hypotensive shock, etc.
On FFA : Disc and choroidal filling delay	On FFA : Disc filling delay only
Treatment : Corticosteroids : 1-2g/day IV methylprednisolone, Aspirin (150mg/day). Immunosuppressants.	Treatment : Levodopa-Carbidopa combination for 3 weeks.
Poor prognosis	Good prognosis
Visual field defects ; Inferior altitudinal defect Central scotoma.	Visual field defects ; Inferior nasal altitudinal defect. Central scotoma.

Congenital anomalies of OD 01:09:33

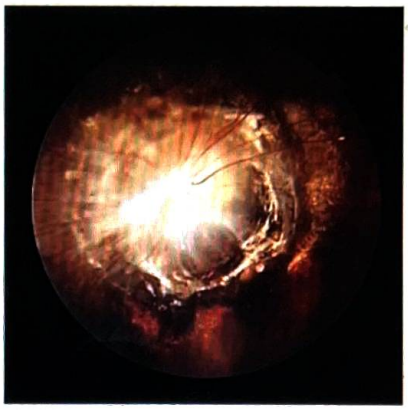
1. Morning glory syndrome :

   Congenital unilateral malformation of optic disc.

   Due to :

   - Peripapillary scleral defect.

- Absence of lamina cribrosa.
- Axial retro displacement of optic nerve.



2. myelinated nerve fibres :

Retinal nerve fibres carries myelination intra ocularly gives appearance of whitish radiation.

No visual loss.

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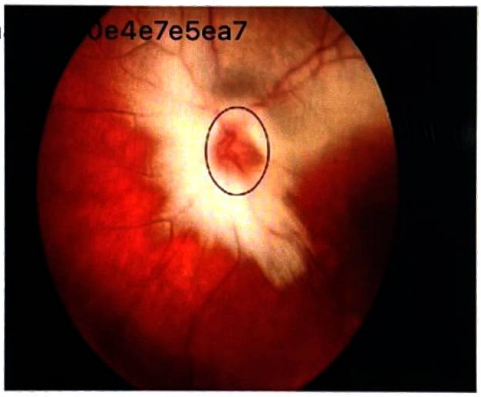


Active space

NEURO OPHTHALMOLOGY - COLOUR BLINDNESS AND CONGENITAL ANOMALIES

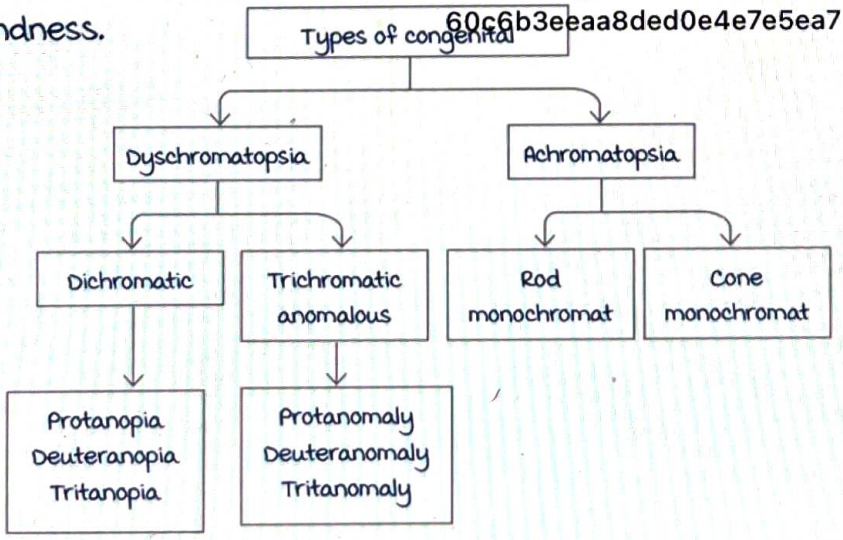
Primary colours :

- Red : Prota.
- Green : Deutra.
- Blue : Trita.

Classification of colour blindness 00:03:40

1. Acquired :
   - All optic nerve diseases : Red-green colour blindness, except in glaucoma.
   - Macular diseases : Blue-yellow colour blindness, except Stargardt's disease.
   - Old age and nuclear cataract : Blue colour blindness.
2. Congenital :
   - Most common mode of inheritance : X-linked recessive.
     Commonly seen in males (females are carriers).
   - Genes for :
     - Red and green : Chromosome X.
     - Blue : Chromosome 7.

Most common colour blindness : Red and green colour blindness.



Active space

Dyschromatopsia : Problem in colour vision.

Dichromatic : Perception of 1 colour is absent.

Patient is blind for 1 colour (blind : Anopia).

Patient can see 2 colours.

- Protanopia : Red colour blind.
- Deuteranopia : Green colour blind.
- Tritanopia : Blue colour blind.

Trichromatic anomaly : Perception of all 3 colours present but defective (anomaly) for 1/2 colours.

- Protanomaly : Red colour defect.
- Deuteranomaly : Green colour defect.
- Tritanomaly : Blue colour defect.

Most common colour blindness : Deuteranomaly.

Achromatopsia : No colour vision.

Rod monochromat :

- Autosomal recessive inheritance.
- Total colour blindness/day blindness.

Cone monochromat :

- Only 1 colour perception present (single colour cone present).

Because there is no differentiation of colour (colour makes sense due to colour opponent theory) it gets classified as achromatopsia.

Colour blindness : Tests

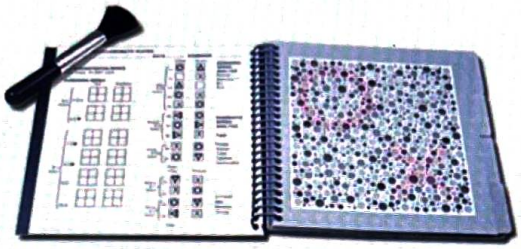
00:15:50

- Ishihara chart : Most common test.

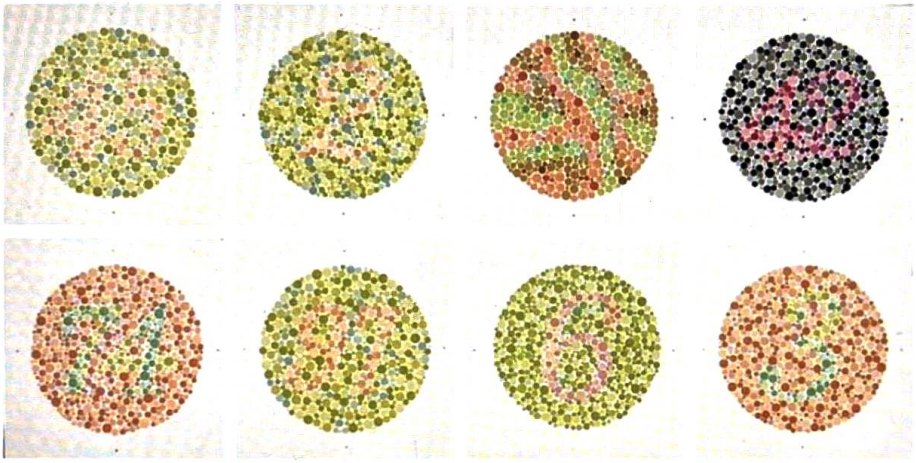
  It cannot detect tritan defects.
- Hardy Rand Riltler : Paediatric use.
- Holmgren's wool.
- Nagel's anomaloscope : Used to ascertain the severity of colour vision loss.
- Farnsworth Munsell 100 hue : Most sensitive test.
- Farnsworth Munsell 15 hue : Used to differentiate between congenital and acquired colour blindness.
- Lantern test : Used for vocational testing.

Pseudoisochromatic charts :

1. Hardy Rand Riltler test :



2. Ishihara chart



Farnsworth munsell 100 hue test :

kumarankitindia1@gmail.com

Farnsworth munsell 15 hue test :

SQUINT - EXTRA-OCULAR MUSCLES, BINOCULAR SINGLE VISION AND CLASSIFICATION/TYPES OF SQUINT

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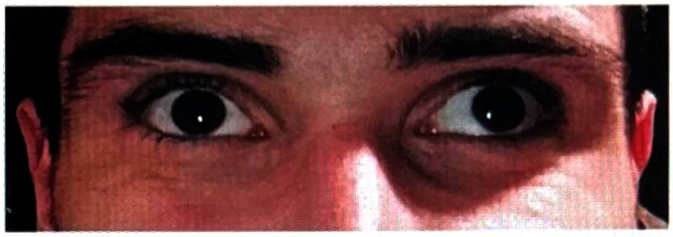
A.K.A strabismus.

It is the deviation of one or both eyeballs due to problem in the extraocular muscles and ocular movement.



Actions of extraocular muscles

00:03:53

The number of extraocular muscles : 7.

1. LPS/Levator Palpebri Superioris : Elevation of the upper eyelid.
2. MR/Medial Rectus : Inward movement/adduction.
3. LR/Lateral Rectus outward movement/abduction.

Mnemonic : SO4 LR6 (lateral rectus is supplied by abducents nerve).

Muscles	Primary action	Secondary action	Tertiary action
4. SR/Superior rectus	Elevation	Intorsion (mnemonic : SIN)	Adduction
5. IR/Inferior rectus	Depression	Extorsion	
6. SO/Superior oblique	Intorsion	Depression	Abduction (mnemonic : ABO)
7. IO/Inferior oblique	Extorsion	Elevation	

Intorsion and extorsion eye movements are clinically not visible.

Active space

The rules for clinical actions of extraocular muscles

00:18:07

All the primary actions of the muscles predominate in the abducted eye.

All the secondary actions of the muscles predominate in the adducted eye.

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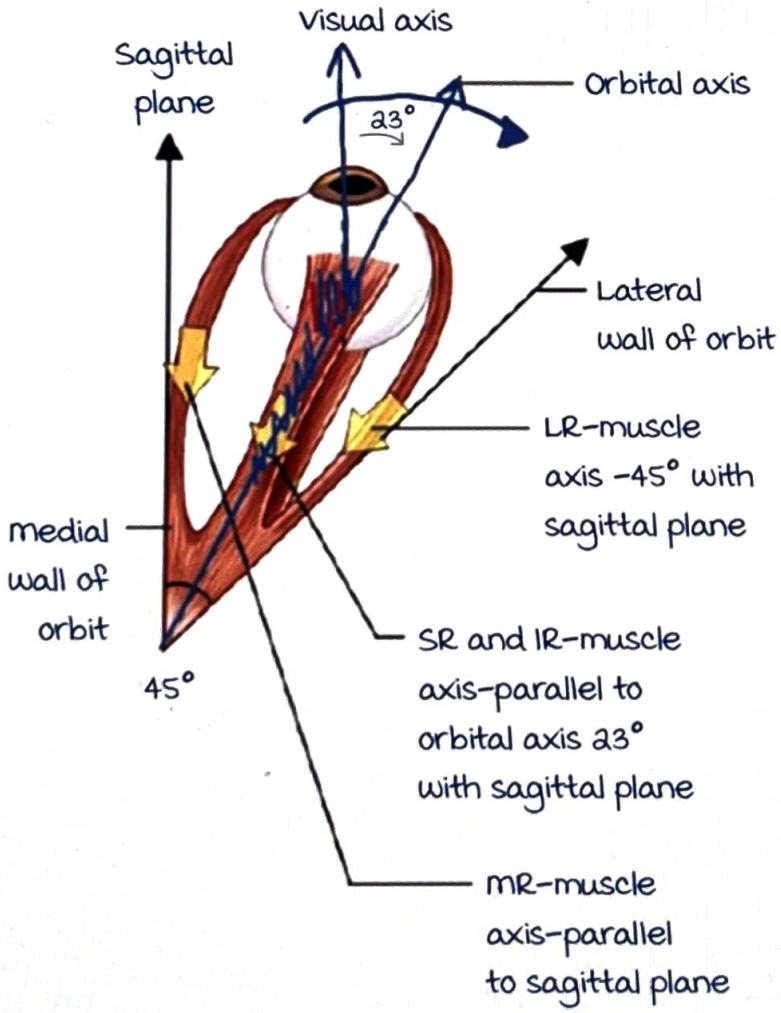
Clinical actions/clinical testing :

SR : Elevation in abduction.

IR : Depression in abduction.

SO : Depression in adduction (ask the patient to look at the tip of the nose).

IO : Elevation in adduction.

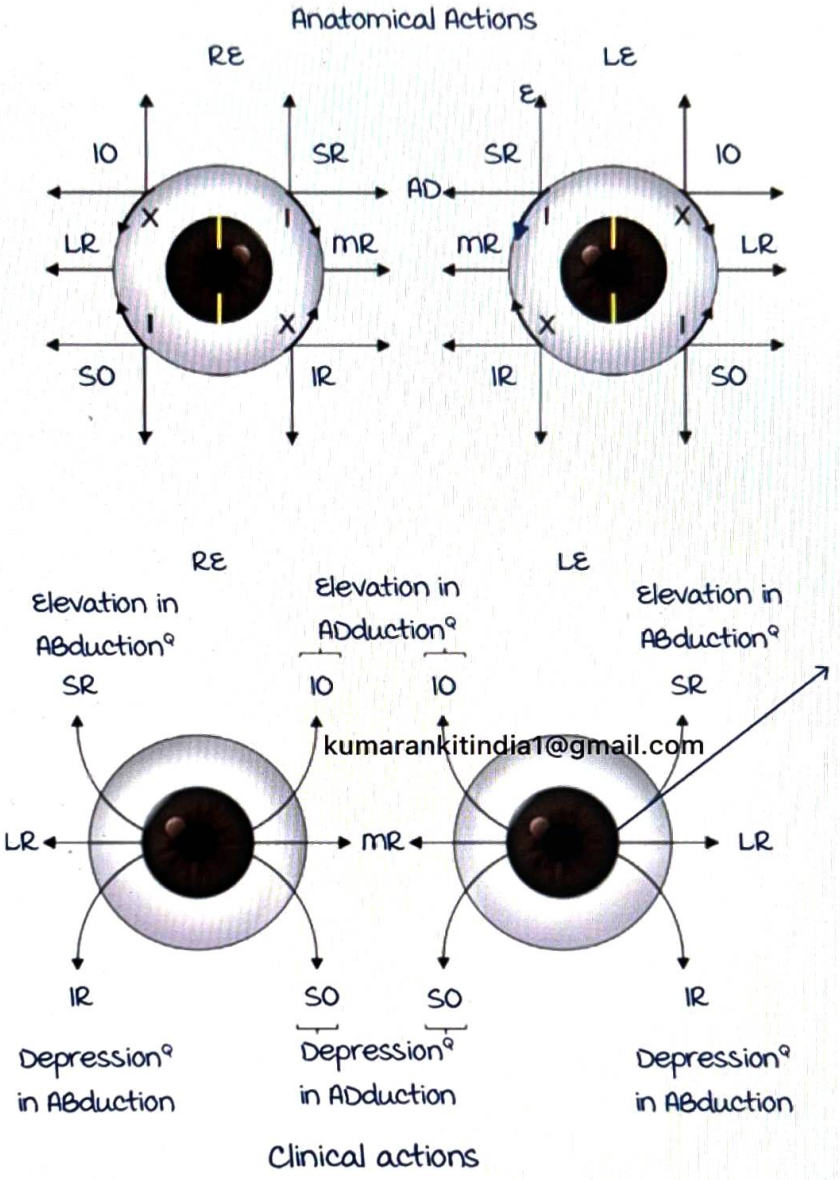


The angle between the visual axis (VA) and the SR muscle axis (MA) is 23°.

The muscle becomes a pure elevator when the VA = MA, i.e.,

when the eye is abducted by 23°, VA = MA of SR.

Anatomical actions and clinical actions of extraocular muscles :



Binocular single vision

00:27:43

Two eyes forming a single image is called binocular single vision.

It is possible because both the eyeballs are looking at the same object as both eyes are looking in the same direction.

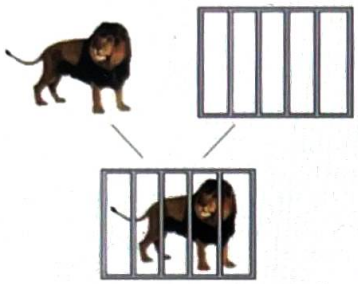
In squint, due to the presence of deviated eyes which have different directions, results in loss of binocular vision.

Grades of binocular single vision :

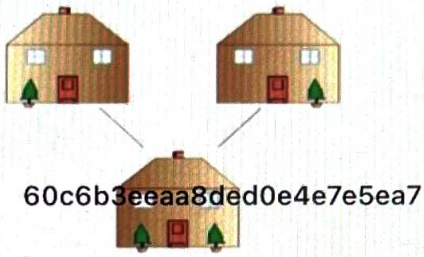
Grade 1 : SMP (Simultaneous Macular Perception).

Grade 2 : Fusion (images formed in the right and left eyes superimpose on each other in the brain).

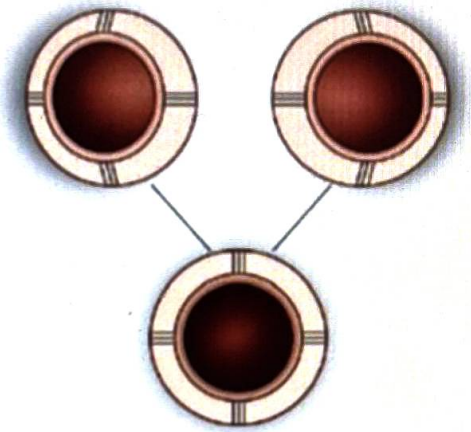
Grade 3 : Stereopsis (binocular depth perception).



Grade 1



Grade 2



Grade 3

Mechanism of formation binocular single vision :

1. Antagonists : A pair of muscles having opposite actions in the same eye.
   Example : RLR and RMR.
2. Synergists : A pair of muscles having the same action in the same eye.
   Example : RSR and RIO.
3. Yoke muscles/contralateral synergists : A pair of muscles (one in each eye) that help both eyeballs move together in the same direction, known as version/conjugate movement.

Active space

Example :

- LLR and RMR : Levoversion.
- RLR and LMR : Dextroversion.
- RSR (elevation in abduction) and LIO (elevation in adduction) : Dextroelevation.
- RIR (depression in abduction) and LSO (depression in adduction) : Dextrodepression.
- RSO (depression in adduction) and LIR (depression in abduction) : Levodepression.
- RIO (elevation in adduction) and LSR (elevation in
- abduction) : Levoelevation.
- RSR + RIO (elevation of right eye) and LSR + LIO (elevation of left eye) : Bilateral elevation (sursumversion).
- RIR + RSO (depression of right eye) and LIR + LSO (depression of left eye) : Bilateral depression (deorsumversion).

4. Hering's law of equal innervation to a pair of yoke muscles :

   It is applied in :

   - Secondary deviation.
   - Inhibitional palsy.

   Not followed in DVD (Dissociated Vertical Deviation).

5. Sherrington's law of reciprocal inhibition : For two yoke muscles to contract, each of the antagonist muscles of the yoke muscles should relax.

   For example, to look to the right side, RLR and LMR (yoke muscles) should contract (according to Hering's law) and RMR and LLR (antagonist muscles) should relax (according to Sherrington's law).

For each binocular vision, there are at least 4 muscles which act together, wherein two of them contract and two of them relax.

Contraction of the muscles is affected in nerve palsies.

Relaxation of the muscles is affected in spasmodic

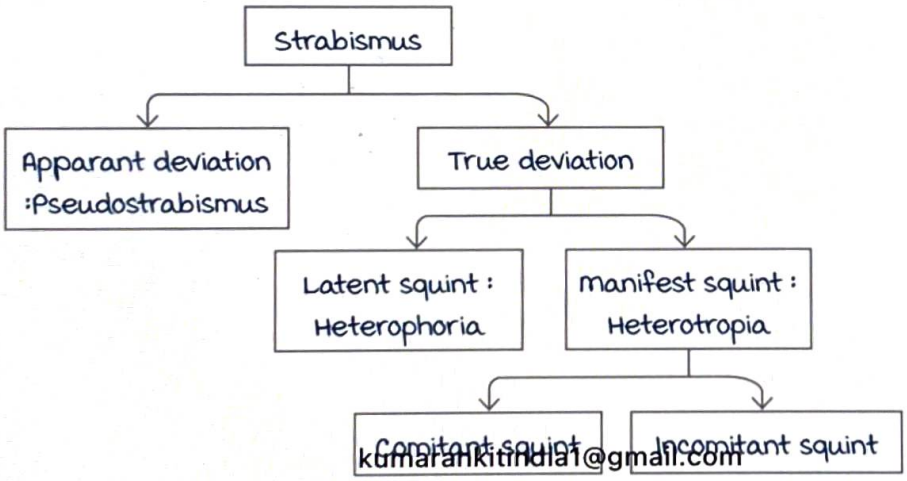
conditions.

Classification of strabismus

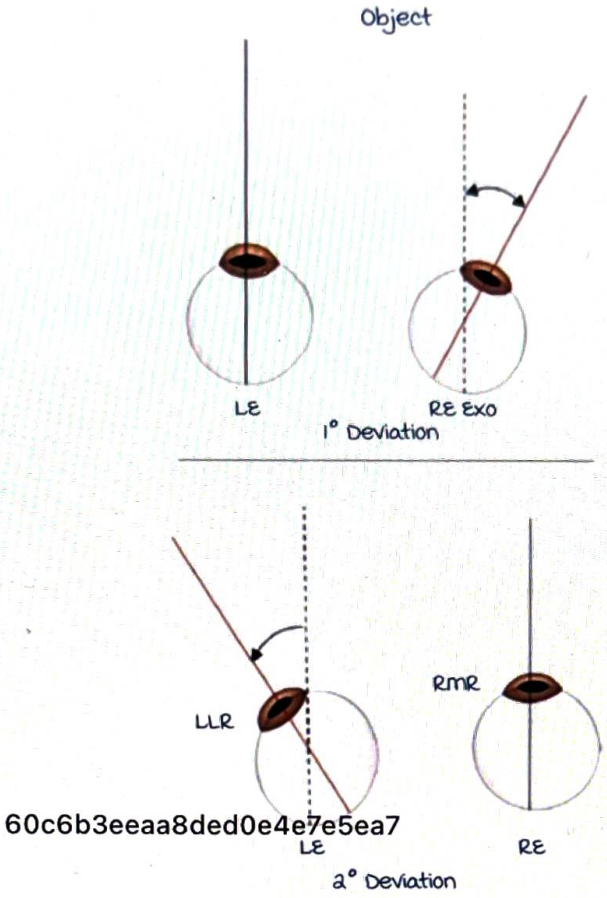
00:54:14

1. Apparent squint : AKA pseudostrabismus.
2. True squint : Types :

- Latent squints : Are hidden deviations which manifest under ocular stress like excessive near work.
- manifest squint is the squint which is always visible.
  1. Comitant squint : Is when the deviation remains constant in all directions of gaze. (1° deviation = 2° deviation).
  2. Incomitant squint : Is when deviation varies in different directions of gaze. (1° deviation < 2° deviation).



Case : A patient presenting with 45° exo in the right eye (1° deviation), was asked to look at an object from the right eye so, the right eye adducts due to the action of RMR muscle and LLR (according to Hering's law of equal innervation to a pair of yoke muscles). As a result of which, the right eye moves in and the left eye moves out leading to the formation of squint (2° deviation).



1° deviation : It is the deviation with which the patient presents where, the normal eye is looking at the object.

2° deviation : Is when the deviated eye is looking at the object and the normal eye appears deviated.

Differences between comitant and in-comitant squint

01:10:49

Parameter	Comitant squint	Incomitant squint
Deviation	1° deviation = 2° deviation	1° deviation < 2° deviation
Diplopia	Absent	Present
Head posture	Absent	Present
Sensory anomalies	Present	Absent
Ocular movements	Full/normal	Restricted

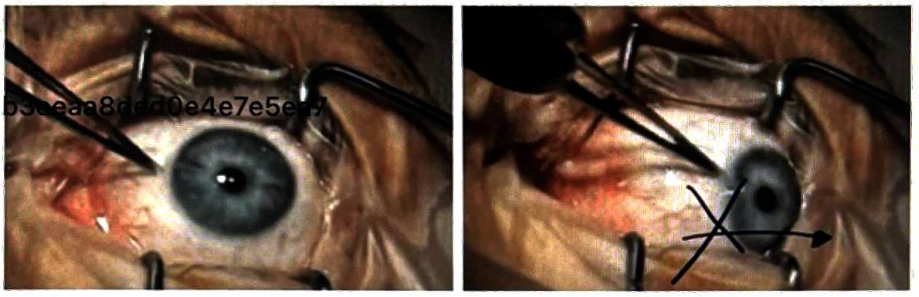
Types of incomitant squint :

- Paralytic squint : Due to paralysis of cranial nerves.
- Restrictive squint : Due to an external factor causing restriction of eye movement.

Forced duction test (FDT) :

It is a test for restriction by applying external force.

- If eye moves with external force : Paralytic squint.
- If eye does not move with external force : Restrictive squint (FDT positive).



It is used to diagnose restrictive squint.

Directions of deviation/nomenclature of deviation 01:18:31

Esotropia : Inward deviation (moves in).

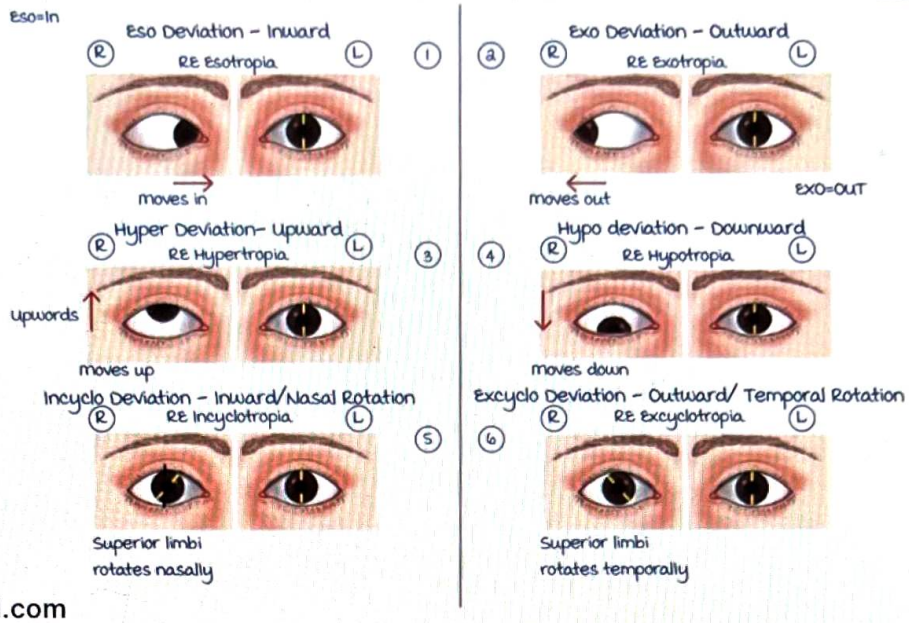
Exotropia : Outward deviation (moves out).

Hypertropia : Upward deviation (moves up).

Hypotropia : Downward deviation (moves down).

Incyclotropia : Inward/nasal rotation of the vertical axis of eye (superior limbi rotates nasally).

Excyclotropia : Outward/temporal rotation of the vertical axis of eye (superior limbi rotates temporally).



Consequences of squint 01:24:42

Diplopia/double vision :

- Occurs in small angle (deviation <5°) squint.
- Two images of one object are formed due to images in both retinas being formed on non-corresponding points.

Confusion :

- Occurs in large angle squint.
- Two images of two objects due to both eyes looking at different objects in different directions.

Adaptations to squint 01:31:09

Adaptations to diplopia :

1. Motor adaptation/fusion : It occurs due to the extra power of extraocular muscles.
2. Sensory adaptation : Anomalous retinal correspondence (ARC).

Adaptations to confusion :

1. Changes in head posture.
2. Sensory adaptation/suppression : Suppression of the image from the deviated eye.
   This leads to amblyopia (loss of vision without organic cause).

SQUINT : INVESTIGATIONS/TESTS

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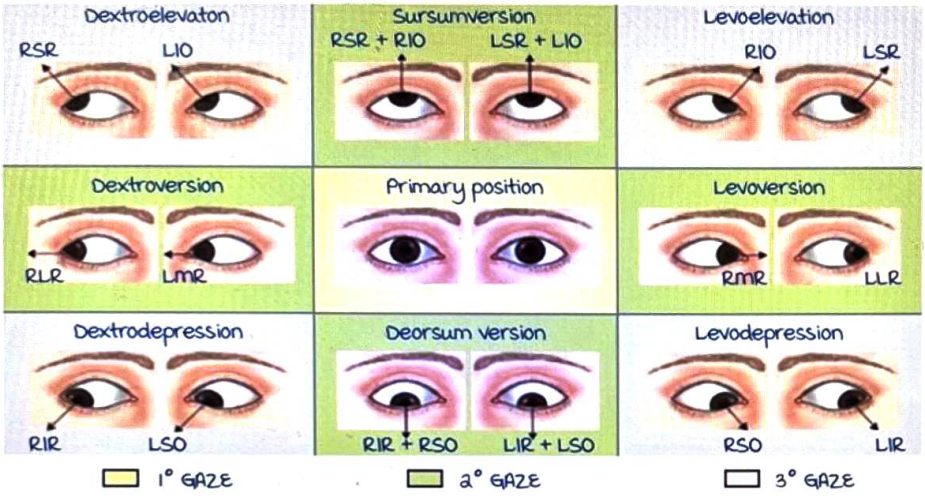
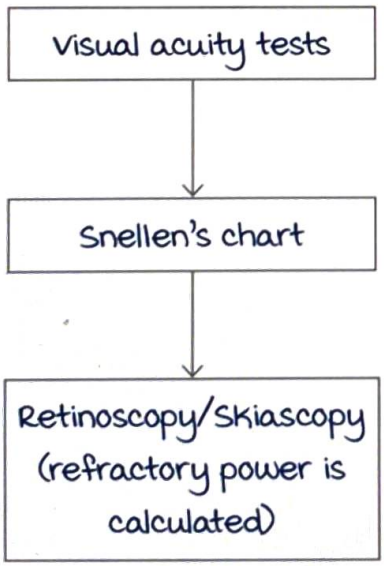
Examination of squint 00:00:57

Tests for squint :

- Examination of ocular movements in all direction of gaze → 9 number of gazes.
  Usually done to narrow down the group of muscles affected.



- Assessment of visual acuity (vision) →



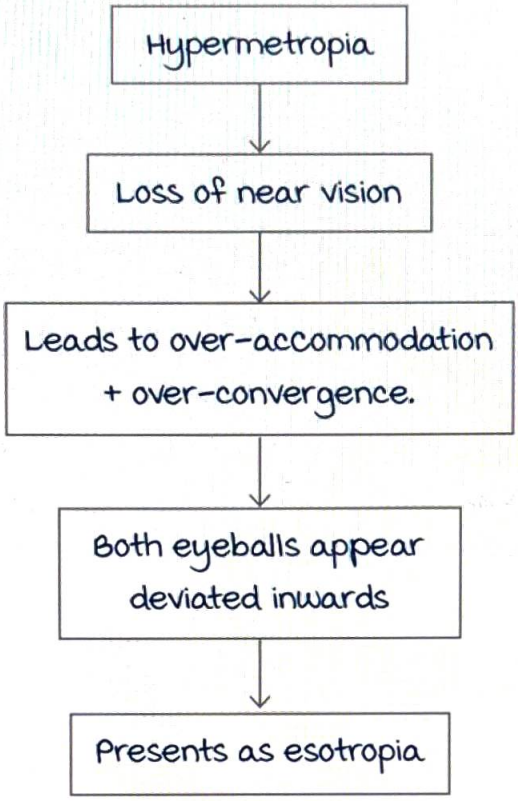
Reasoning behind these tests →
Uncorrected hypermetropia leads to Esotropia.

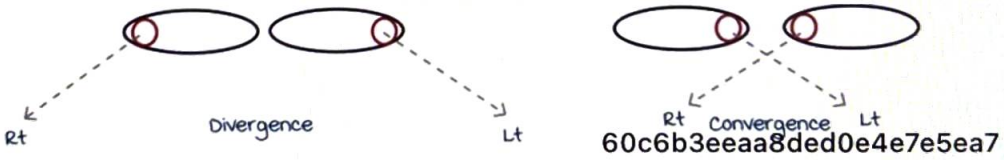
This is known as accommodative squint.

Active space

Treatment : Spectacles.

- measurement of vergence power →
  Each extra ocular muscle have a reserve fusional power/ vergence power which each muscle may use to keep fusion.

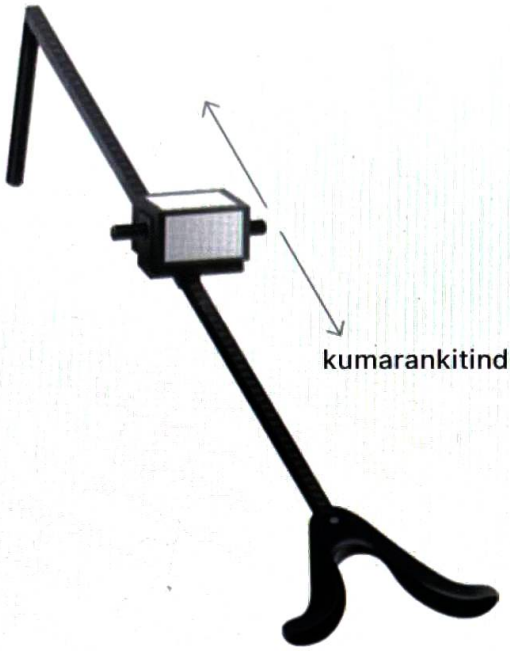


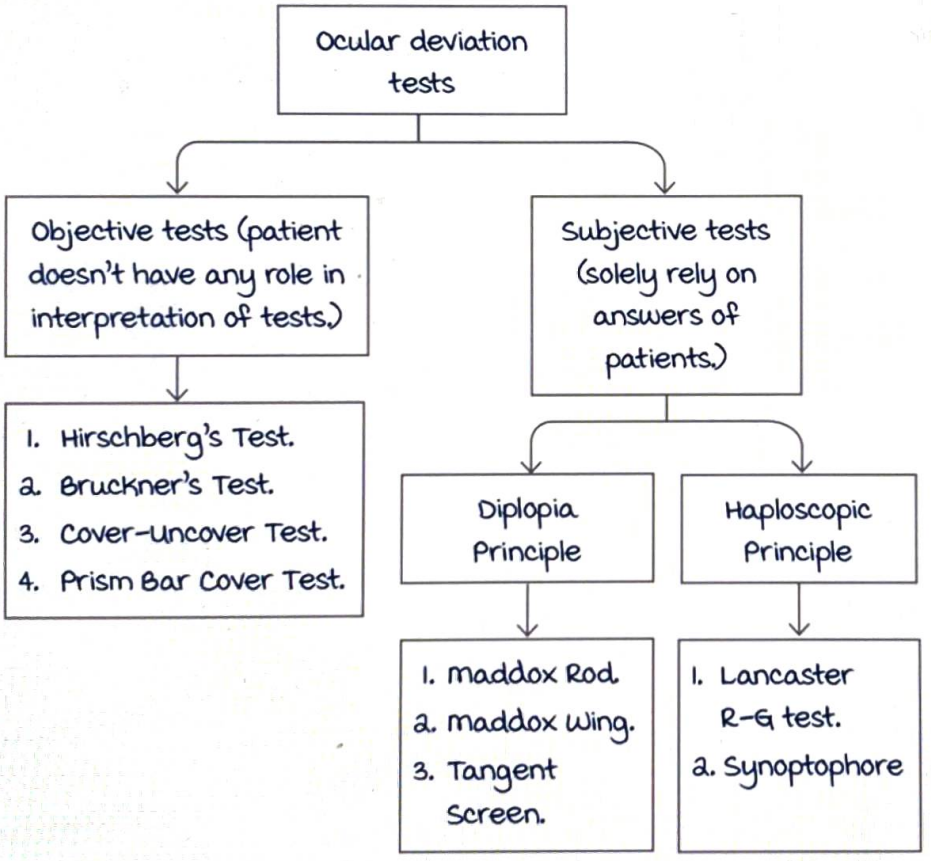


Tests →

1. Synoptophore test.
2. Pencil test.
3. Livingstone Gauge test.
4. RAF ruler test (Royal Air Force) → The patient moves the target up and down up to a point where diplopia occurs → Near point of convergence/ NPC.
   If the vergence power is normal → NPC < 10 cm.

- measurement of ocular deviation :





Active space

Hirschberg's (corneal reflection) test

00:17:37

Uses :

1. To identify the direction of deviation :

   Deviation.

   Opposite.

   Of.

   Reflection.

   Pupillary margin.

   Corneal reflection → Temporal to Centre.

   Limbus

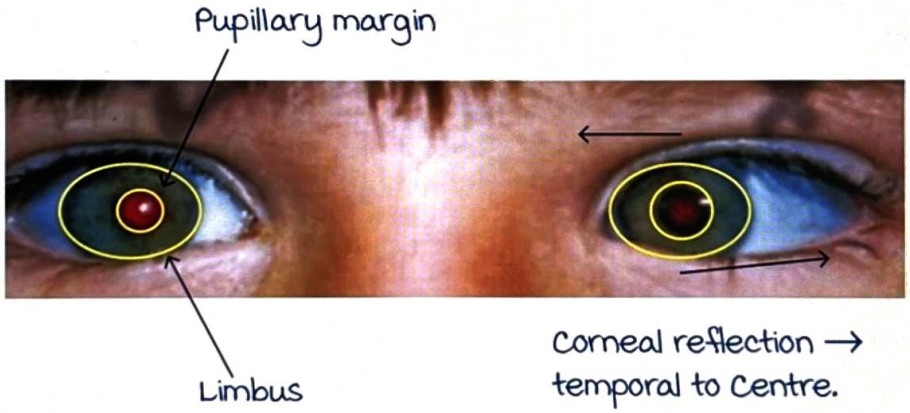
   Normal corneal reflection (CR) = Centre of pupil.

   CR temporal to centre → Esotropia.

   CR Nasal to centre → Exotropia.

   CR above the centre → Hypodeviation.

   CR below the centre → Hyperdeviation/ hypertropia.



2. To calculate degree of deviation →

   1 mm deviation of CR from center of pupil = 7° Squint

   Or

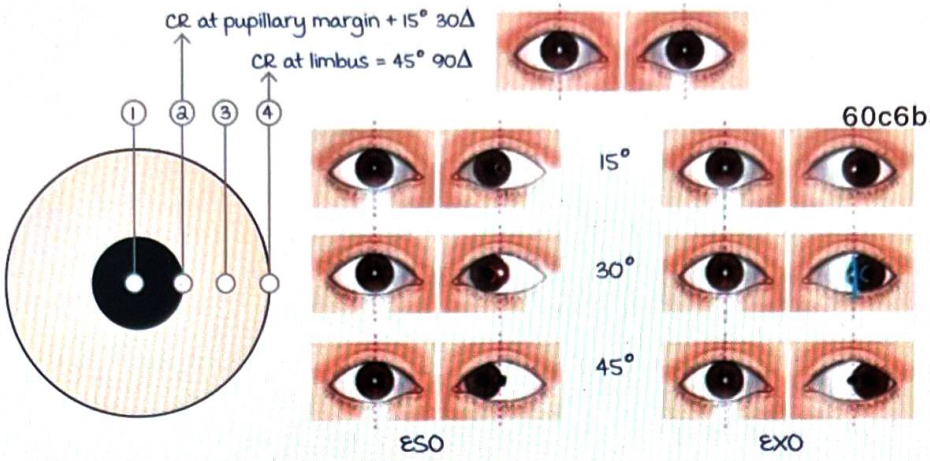
   1 mm = 14 prism dioptre squint.

   1 prism dioptre = 0.5° degrees.

   In an image, degree of deviation :

   If CR at limbus → 45°.

   If CR at pupillary margin → 15°.



Q. A patient on examination has CR visible below the centre of people at limbus. Diagnosis?

Answer : 45° hypertropia.

Bruckner's test

00:31:08

Used for screening of squint in infants.

Method → By direct ophthalmoscope to illicit fundal red reflex → Compare brightness and intensity in both eyes.

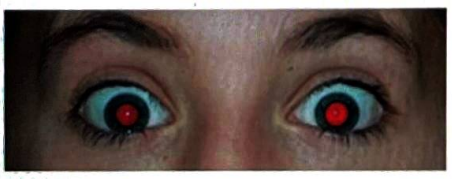
- Equal brightness & colour → Normal eyes.
- Crescent of different colour intensities → Refractory error.
- Difference in brightness → Darker eye : Normal brighter eye → Squint.

Bruckner's Test.

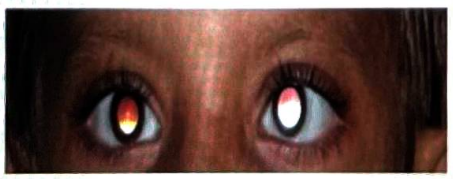
Equal brightness and colour.

Crescent of different colour intensities.

Difference in brightness.



Equal brightness and colour



Crescent of different colour intensities

Difference in brightness

Active space

Cover uncover test

00:34:44

Cover test :

used to confirm tropia.

(CT : Cover test Tropia).

Uncover test :

used to diagnose phoria.

(Uncover test Phoria).

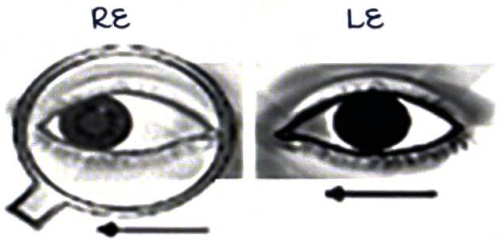
Deviation.

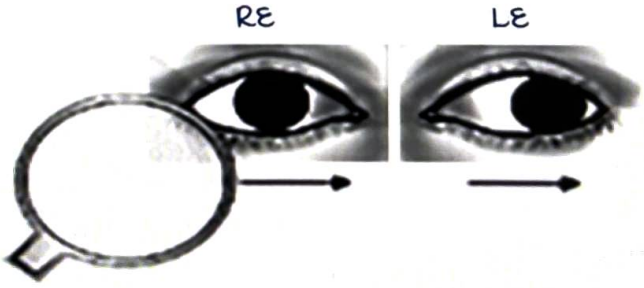
Opposite.

Of.

Movement seen.

(LE) Exotropia



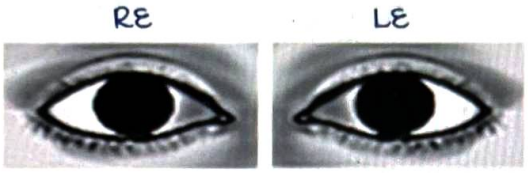


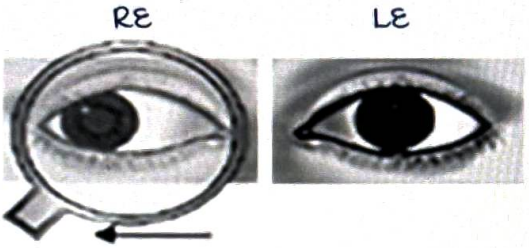
On presentation of left eye exotropia → Confirmatory cover test needs to be done → Cover normal eye (right) → Left eye moves inwards → Left eye exotropia is confirmed → Left eye moves out again as soon as the right eye is uncovered.

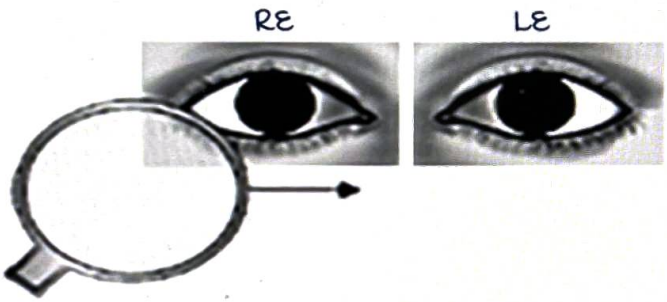
On presentation both eyes look normal (no deviation : Can be normal eye or phoria) → While covering the right eye :

Active space

No movement is seen → While uncovering the right eye (uncover test) it is seen that it moves inwards → Right eye exophoria.







Q. On right eye cover test, left eye is seen to move inwards. Diagnosis?

Ans. Left eye exotropia.

Q. On right eye uncover test, right eye is seen to move outwards. Diagnosis?

Ans. Right eye esophoria.

Left eye exotropia.

Right eye exophoria.

Prism bar cover test / PBCT

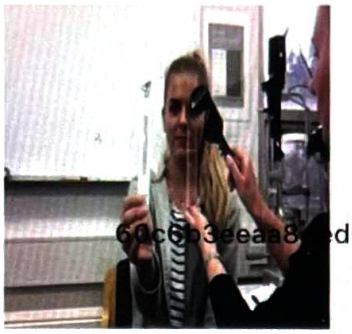
00:48:27

60c6b3eeaa8ded0e4e7e5ea7

Active space

Best test to measure the degree of tropia → PBCT.

Principle : Neutralization (whenever light passes through a prism, it always deviates towards the apex.)



PBCT

Orientation of prism :

Deviation

Opposite.

Of.

Base of prism.

Eso → Base out prism.

Exo → Base in prism.

Hyper → Based down prism.

Hypo → Base up prism.

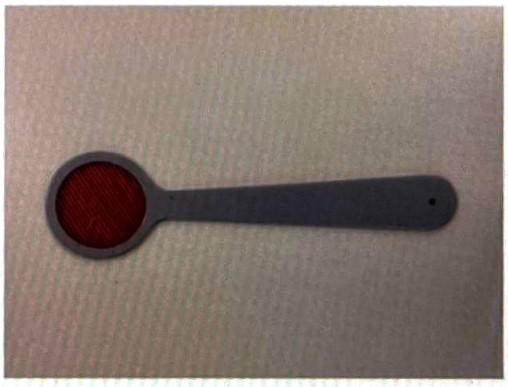
Maddox rod test

00:53:28

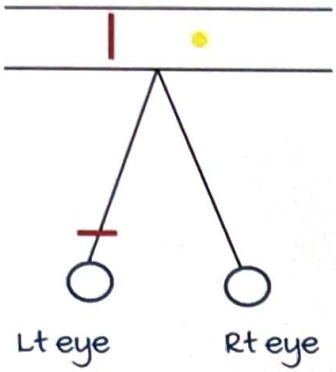
Red coloured striated (plano-convex) lens.

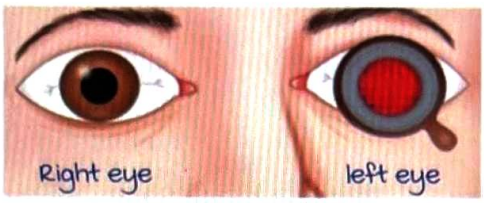
Used :

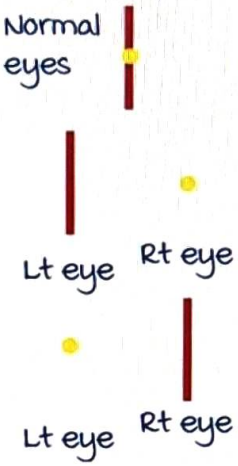
- To measure degree of phoria at far fixation.
- Macular function test.
- Double Maddox rod test :
  Maddox rod is kept over both the eyes.
  used to test for cyclotropia.



Active space







When patient covers the right eye and look through the striated lens in front of the left eye → Striated red line.

When patient covers the left eye, sees the object properly.

Results :

1. Patient sees a red streak with a yellow point source of light in the middle → (the images fuse) Normal eyes/ortho.
2. Patient sees a red streak on the left side and a yellow point source on the right side (the images are not fusing) → Uncrossed diplopia.
   Seen in patients with esodeviation → Esophoria.
3. Patient sees a red streak on the right side and a yellow point source on the left side (the images are not fusing and are on opposite sides) → Crossed diplopia.
   Seen in patients with exodeviation → Exophoria.
   Exo → Cross → Crossed diplopia.

Active space

Maddox wing test

01:01:46

Obsolete now.

Used to measure degree of phoria at near fixation.

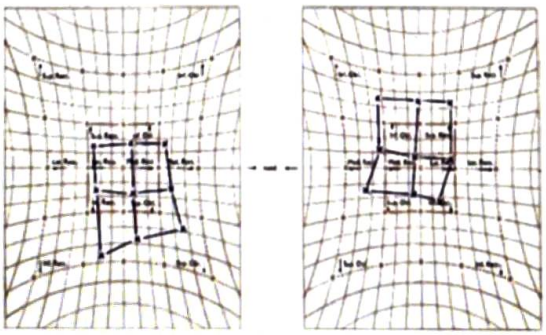
Maddox wing test

Tests for diplopia

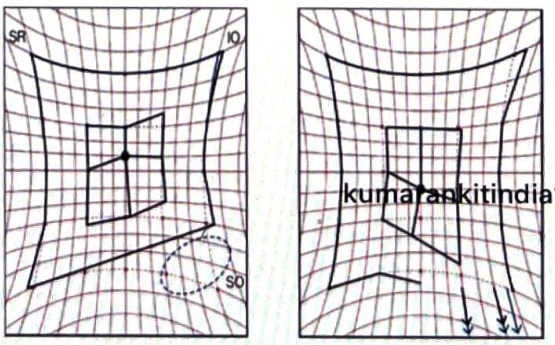
01:03:15

Based on disassociation of images.

- Red green goggles : Different coloured lens in front of each eye to dissociate the images → Assess the fusion power.
- Hess chart : A printed chart with concave lines.
  It helps with diplopia charting and diagnosing the paralysed muscle.



Hess Chart



Hess Chart

Active space

E.g.

The red dots on the chart denote the extent of the visual field.

We ask the patient to look in each direction and plot till they can easily see.

The superior oblique muscle is deficient in the visual field and therefore the antagonist muscle inferior oblique starts to over act slightly.

Due to the Herring's law of equal innervation of yoke muscles, the yoke muscles of the opposing eye will start to overact.

Right inferior rectus start getting more stimulus and the visual field and activity increases tremendously.

- Lee's screen.

Tests for sensory anomalies 01:07:39

Tests which differentiate between :

- BSV → Binocular single vision.
- ARC → Anomalous retinal correspondence → Occurs as sensory adaptation in case of small angle squint
  Diplopia → Corresponding points change.
  Over of the normal eye corresponds with the extra-foveal point of slightly deviated eye to adapt for diplopia.
- Suppression → Sensory adaptation in confusion where 2 images are formed. The brain will suppress the image from the deviated eye.

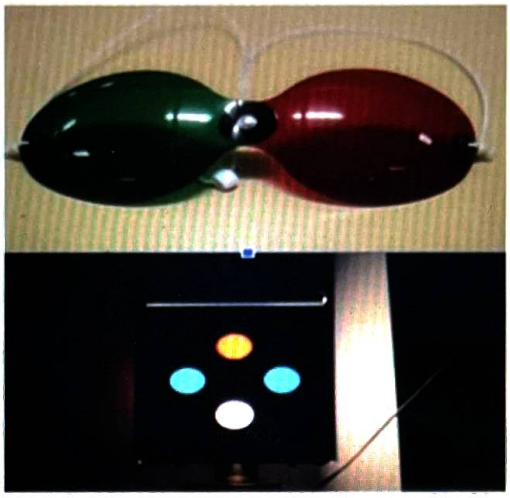
These tests help in differentiation between a normal eye and a squint eye.

The tests done are :

1. Worth's 4 dot test : We show the patient a box of light/screen with 4 dots on it.
   The colour of the 4 dots are 1 Red, 2 green /blue & 1 white/yellowish in colour.
   Method : We ask the patient to wear red green

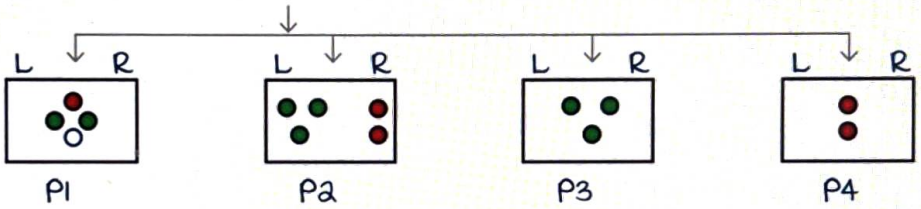
Active space

goggles.



Worth's 4 Dot Test

To standardise the test red lens over the right eye and green over the left side to filter the images.
The patient looks at the 4 dots →



Patient 1 is wearing red green goggles and is able to see all the 4 dots → Patient has BSV/harmonious ARC.

Patient 2 is able to see 3 green dots and 2 red dots → (both the eyes are functional but the images are not fusing) patient has Diplopia/ Non Harmonious ARC.

Patient 3 is able to see 3 green dots → Right eye suppression (the white dot also becomes green)

Patient 4 is only able to see 2 red dots → Left eye suppression (the white dot also becomes red).

2. Bagolini striated glasses : most physiological or least dissociative test.

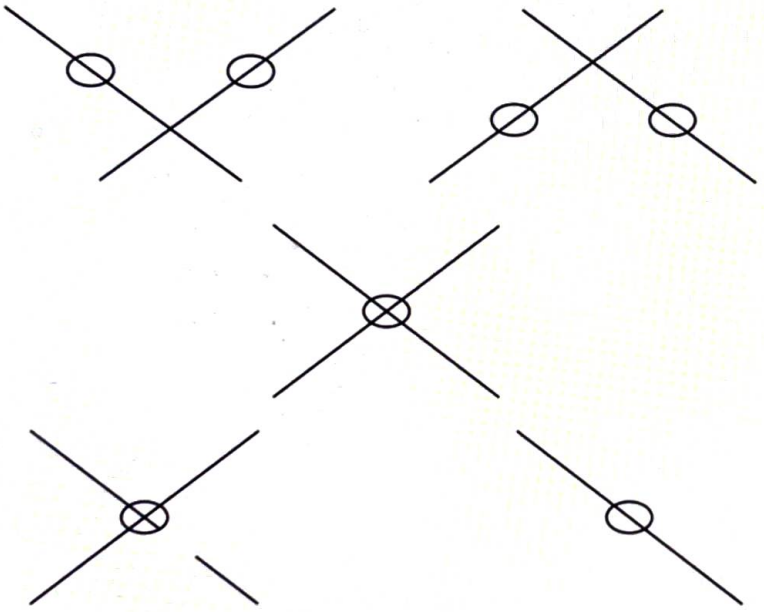
Active space

Bagolini striated glasses

The glasses are striated with 45° striation in one eye and 135° striation in the other eye.

Results : if both the lines are intersecting with each other → BSV/harmonious ARC.

If only one line is seen → Indicates suppression.



Bagolini Striated Glasses Result Test

Border lines will be visible but the central part will be missing → Diagnosed as central suppression scotoma.

If both the lines are visible but are not intersecting → Diplopia.

3. Synoptophore.
4. 4 dioptre prism test : used in Microtropia (squint < 5 prism dioptre).
5. After image test.

Active space

Tests for stereopsis

01:18:30

- a pencil test for gross stereopsis.
- Titmus Fly test → The patient has to wear polarised glasses and the fly will be seen in different angles.

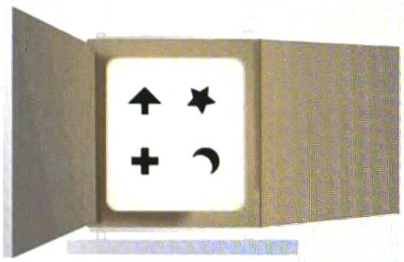
Titmus Fly Test

- Random Dot Stereogram Test (Better than Titmus Fly Test).

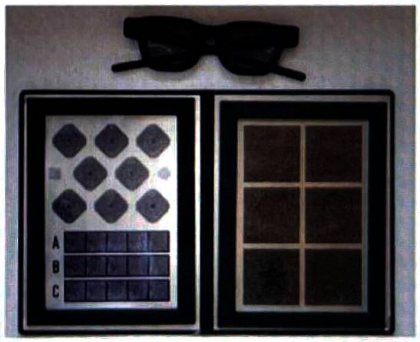
  They eliminate mono-ocular clues to depth perception (they only rely on binocularity).

1. Frisby-Davis Distance Stereo test
2. TNO Test
3. Frisby Stereo test
4. Lang test → A car, a cat and a star can be seen.

Frisby-Davis Distance Stereo test



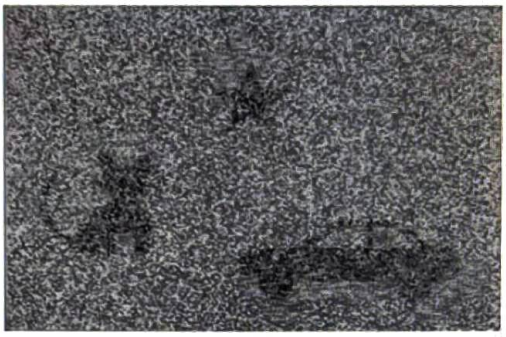
TNO Test



Active space

Frisby Stereo test

Lang Test



Depth perception

During binocular vision	During mono-ocular vision

• Stereopsis
• Retinal disparity
  • Binasal disparity → Remoteness (for) perceived.
  • Bitemporal disparity → Nearness perceived.
	• Motion Parallax (objects moving fast are perceived clear).
	• Overlay of contours.
	• Linear perspective.
	• Change in apparent size (Larger objects appear nearer).
	• Aerial perspective.
	• Shadows and highlights.
	• Looming.

Active space

SQUINT: PARALYTIC SQUINT

Paralytic Squint

00:00:57

Paralytic squint occurs when there is an acquired defect of movement of an eye. Occurs due to disease of the III, IV and VI cranial nerves.

III[rd] cranial nerve or, oculomotor nerve palsy

- Course :

Nucleus situated in mid-brain at level of superior colliculus.

↓

III[rd] CN enters lateral wall of cavernous sinus

↓

Passes through superior orbital fissure within annulus of Zinn

- Nucleus has 2 parts :

Main motor nucleus → Supplies extraocular muscles → Superior rectus, inferior rectus, internal oblique, medial rectus → Supplied contralaterally

Edinger-Westphal nucleus → Supplies sphincter pupillae muscle & ciliary muscle

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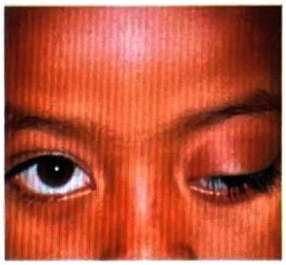
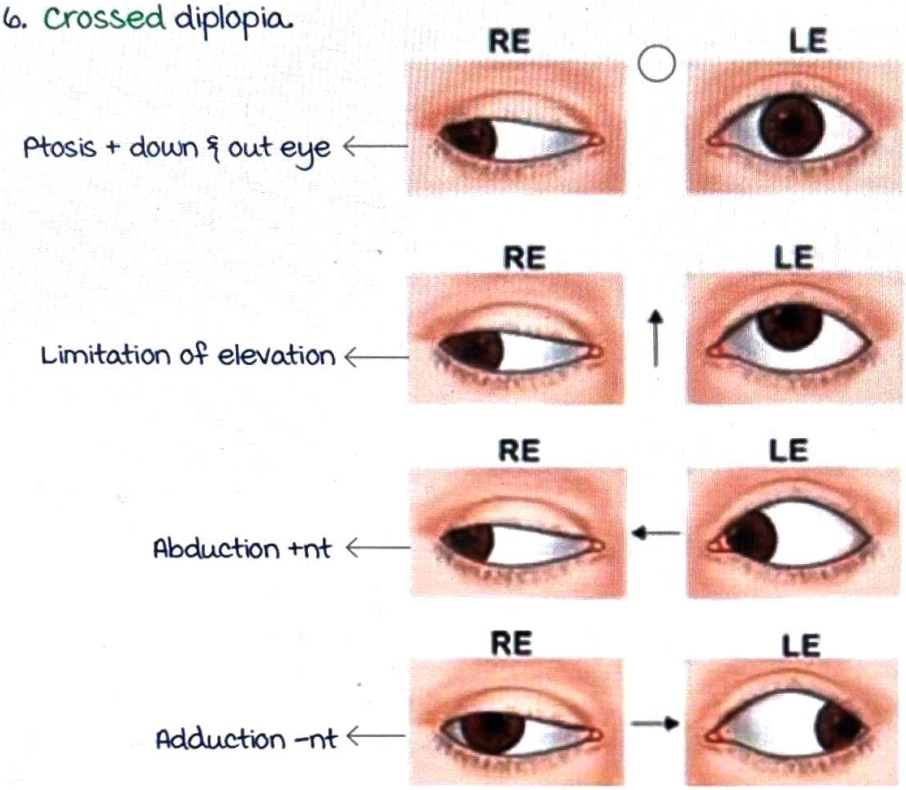
Levator palpebre superioris → Supplied by an unpaired nuclei

Active space

- Clinical features of III[rd] cranial nerve palsy :

1. Ptosis.
2. Hypotropia.
3. Exotropia.
4. Mydriasis (no light reflex).
5. Loss of accommodation (no near reflex).
6. Crossed diplopia.





Special clinical features :

00:15:49

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1. Diabetes or HTN
   - III[rd] CN palsy with pupillary sparing.
   - Light reflex present.
   - Near reflex absent.

2. Uncal herniation : Only mydriasis seen.

3. Benedikt's syndrome
   - III[rd] CN palsy with flapping tremors.
   - Lesion is through red nucleus.

4. Weber's syndrome
   - III[rd] CN palsy with contralateral hemiparesis.
   - Lesion is through cerebral peduncle.

Active space

5. Nuclear III[rd] CN palsy :
   - Bilateral ptosis (due to unpaired nuclei for LPS).
   - Contralateral superior rectus weakness.

   — Daroff's rule

Treatment for III Cranial nerve palsy 00:24:32

1. Observation for at least 6 months.
   (as III[rd] CN can regenerate)

2. Surgery.

Aberrant regeneration

Pseudo Von- Graffe sign

Fibres of IR misdirected to LPS

On downgaze, both IR & LPS contract

Due to IR Depression of eye

Due to LPS Elevation of upper lid

Pseudo ARP

Fibres of ciliary muscle misdirected to sphincter pupillae

On near vision : Accommodation occurs & pupils constricts.

Fibres of sphincter pupillae completely damaged.

No light reflex

Thus, looks like lid- lag

IV Cranial nerve palsy 00:33:50

- Trochlear nerve palsy.
- Course :

Nucleus (in mid-brain at level of inferior colliculus)

↓

Leaves mid-brain on the dorsal surface after decussation

↓

Enters cavernous sinus on lateral wall but, passes through

superior orbital fissure outside annulus of Zinn

↓

Supplies contralateral superior oblique muscle

60c6b3eeaa8ded0e4e7e5ea7

- Clinical features of IVth cranial nerve palsy :

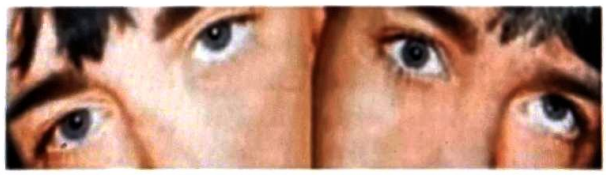
1. Excyclotorsion
2. Hypertropia.
3. Increased hypertropia on adduction/ increased hypertropia on opposite gaze.
4. Vertical diplopia (Experienced on climbing downstairs).
5. Head posture :
   a. For depression : Chin depression.
   b. For intorsion : Head tilt towards opposite side.
   c. For depression in adduction : Face turn towards opposite side.

Treatment for IV Cranial nerve palsy 00:49:30

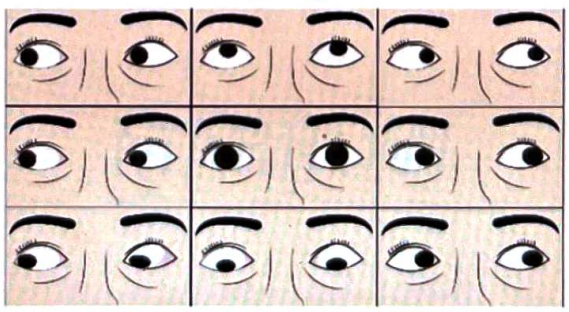
Treatment is Surgical (according to Knapp's classification).
Most common procedure is inferior oblique weakening.
For diagnosis of any superior or inferior muscle palsy :
Park's 3 step test

Step 1 :
Identify the hypertropic eye.

Step 2 :
Hypertropia increases in direction of clinical action of paralysed muscle, either in right gaze or left gaze.



Step 3 :
Hypertropia decreas on tilting of head in same direction of cyclical action



Active space

of paralysed muscle.

VI Cranial Nerve palsy 00:59:49

- Course :

Nucleus (situated in pons)

↓

On exit from brain stem, it passes through Dorello canal

↓

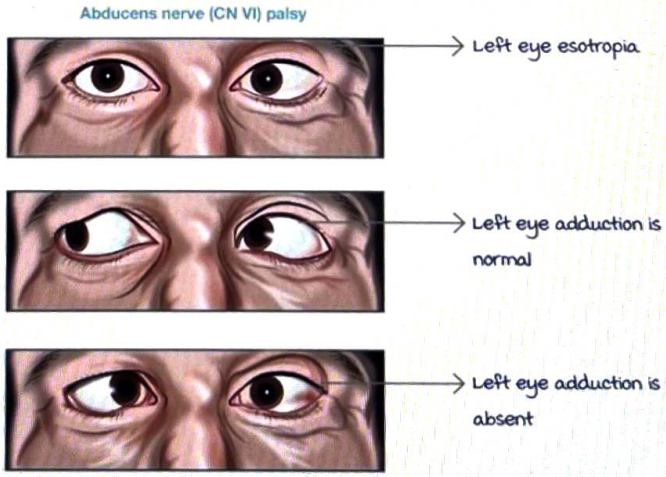
Enters cavernous sinus, runs in lateral wall then passes through superior orbital fissure inside annulus of Zinn

↓

Supplies ipsilateral lateral rectus muscle

- Clinical features of VIth cranial nerve palsy :

1. Esotropia.
2. Uncrossed diplopia.
3. Head posture : Face turn towards same side.
4. Ipsilateral horizontal gaze palsy.



- Causes of VIth cranial nerve palsy :

1. Foville syndrome : Ipsilateral V+VI+VII+VIII cranial nerve palsy with central Horner's syndrome.
2. Millard Gubler syndrome : Ipsilateral VI + VII cranial nerve palsy with contralateral hemiplegia.
3. Moebius syndrome : Bilateral VI+VII cranial nerve palsy.

SQUINT - RESTRICTIVE SQUINT, PALSY, COMITANT SQUINT, PSEUDOSTRABISMUS AND OCCULAR MYOPATHIES

Restrictive squint

00:00:10

Diagnosed by forced duction test (FDT).

Rare presentation.

most common presentations (syndromes) include :

1. Duane's retraction syndrome (DRS) :

Types/ Clinical features	a) Limitation of movement.	b) Retraction of the globe. c) Closure of palpebral fissure.
1	Abduction.	Adduction.
2	Adduction.	Adduction.
3	Abduction & Adduction.	Abduction & Adduction.

Type 1 :

Lateral rectus (LR) innervated by fibers of 3rd nerve, instead of the 6th nerve.

On impulse for abduction via 6th nerve.

↓

Lateral Rectus does not act.

↓

No abduction.

On impulse for adduction via 3rd nerve.

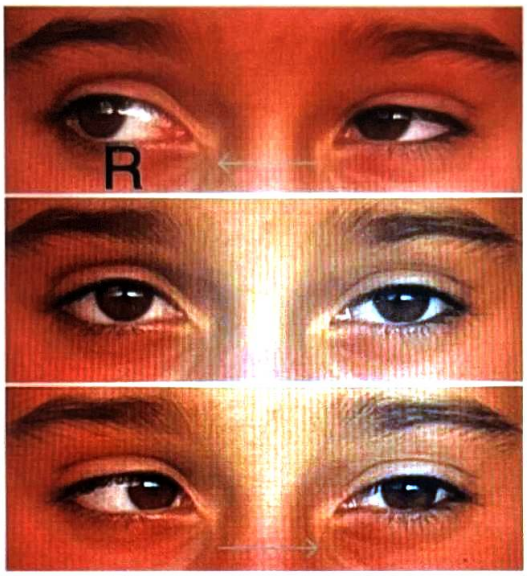
↓

Both Lateral Rectus & medial rectus (MR) contract.

↓

Retraction of the globe.

Active space



Right gaze :
Left eye adduction is present.
Left eye closure of palpebral fissure.

Primary gaze :
Normal.
No deviation.

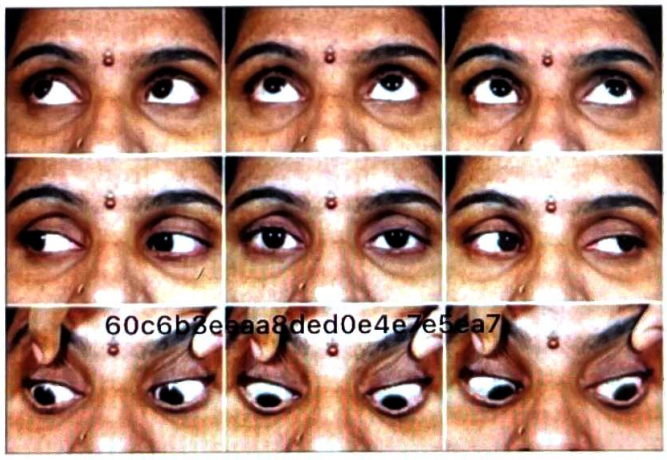
Left gaze :
No Left eye abduction.

This is a case of Type I DRS of the left eye.

Brown's syndrome

00:12:44

2. Brown's syndrome :
Occurs due to congenitally tight superior oblique tendon.
↓
Superior oblique remains contracted.
↓
Superior oblique cannot relax.
↓
Its antagonist, Inferior oblique (IO) cannot contract.
(Sherrington's law of reciprocal innervation)
↓
The patient is unable to perform elevation in adduction
(clinical manifestation of IO).



Right Eye (RE) Brown's syndrome (RE unable to perform elevation in adduction

Supranuclear Gaze Control 00:17:05

Supranuclear Gaze Control.

↓

Cerebral Cortex.

Frontal cortex

↓

Initiates Saccades.

↓

Rapid, Involutary fixating movement.

Horizontal Saccade/Gaze

↓

Contralateral (C/L) control.

Vertical Saccade/Gaze

↓

Bilateral (B/L) control.

Occipital cortex..

↓

Initiates Pursuits.

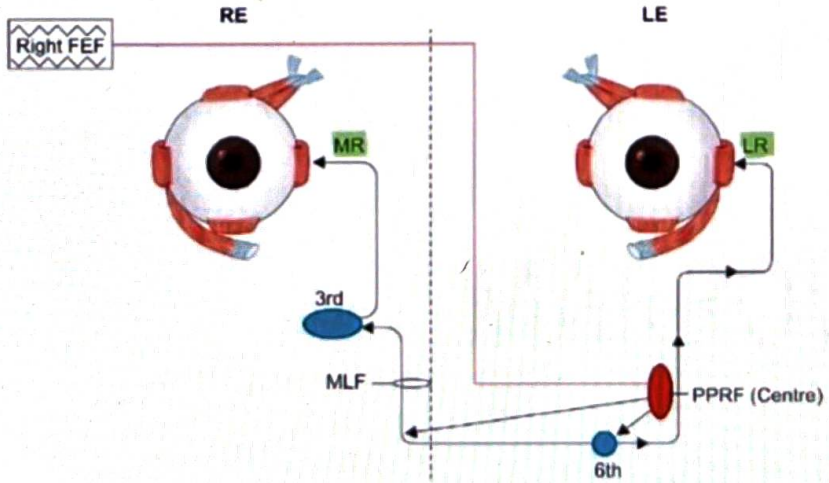
Slow, voluntary, following movement.

↓

Ipsilateral (I/L) control.

Horizontal Gaze Pathway 00:21:21

This can be understood by taking the example of the left gaze.



In the left gaze :

- Left eye lateral rectus will cause abduction.
- Right eye medial rectus will cause adduction.
- Net result of eyes moving towards the left side.

The left gaze signal starts from the right frontal eye field. (FEF)

↓

The signal goes to the left side. → Para Pontine Reticular Formation (PPRF) (Centre for horizontal gaze)

↓

Left PPRF sends a signal to the left lateral rectus muscle → Abduction of the left eye.

↓

Left PPRF sends a signal to the right side via medial longitudinal fasciculus (MLF).

↓

Via the right MLF, the signal goes to the right 3rd cranial nerve nucleus → Right medial rectus muscle. → Right eye adduction.

↓

Both eyes left gaze.

Left horizontal gaze is controlled by :

1. Right (C/L) FEF.
2. Left (I/L) PPRF.
3. Right (C/L) MLF.

There can be lesions at 3 sites in the left horizontal gaze pathway :

1. Right FEF lesion.
2. Left PPRF lesion.
3. Right MLF lesion.

Right FEF lesion :

Left LR → Unable to contract.
Right MR → Unable to contract.
→ Both eyes do not move towards left.
↓
Left gaze palsy.

Therefore, FEF lesion leads to C/L gaze palsy.

Left PPRF lesion :

Left LR → Unable to contract.
Right MR → Unable to contract.
→ Left gaze

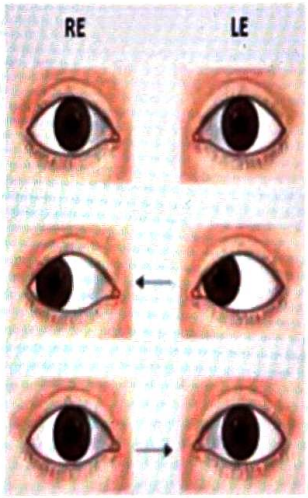
Therefore, PPRF lesion leads to I/L gaze palsy.

Right MLF lesion :

Left LR → Able to contract → Left (C/L) abduction present.
Right MR → Unable to contract → Right (I/L) adduction is absent.
↓
Left gaze Defect.

Therefore, MLF lesion leads to C/L gaze defect.

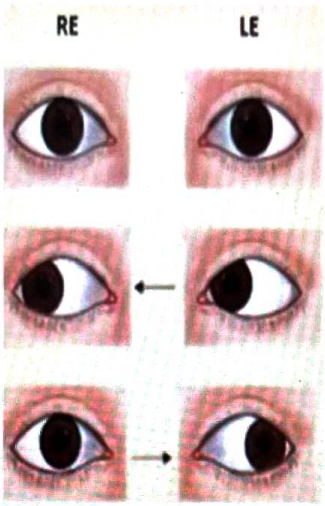
- This condition is known as Internuclear Ophthalmoplegia.
- Most common cause → Multiple Sclerosis.



Primary and right gaze are normal.
Left gaze palsy.
Lesion at :

Right FEF. (or) Left PPRF.

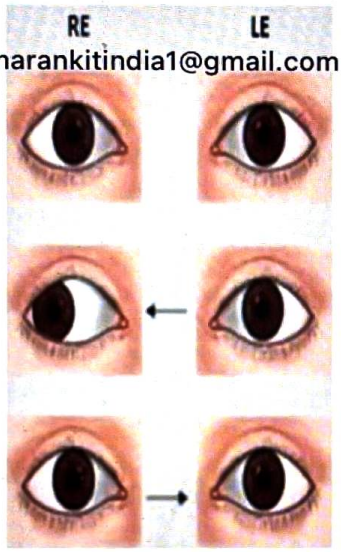
Active space



Primary and right gaze normal.

In left gaze :
Left eye abduction is present.
Right eye adduction is absent.

Therefore, the lesion is at right MLF.



Primary gaze is normal.

In right gaze :
Right eye abduction is present.
Left eye adduction is absent.

Therefore, the lesion is at left MLF.

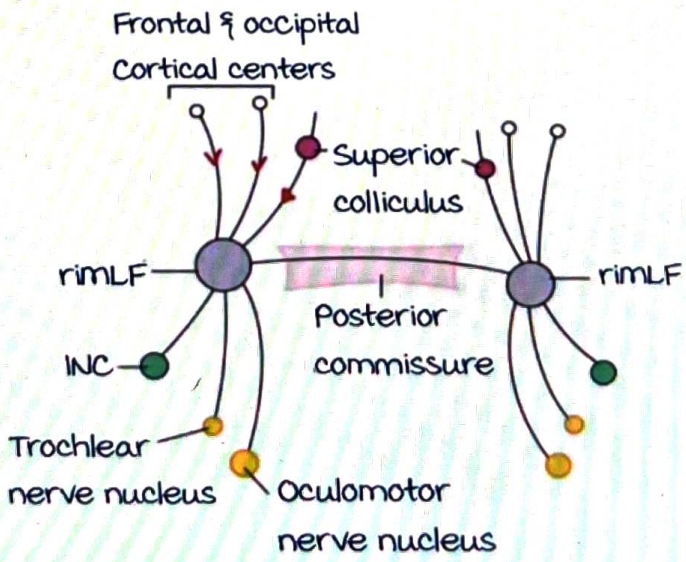
On attempting left gaze, there is left gaze palsy. Therefore, the lesion is at left PPRF.

Hence, there is left MLF + Left PPRF lesion.

This is known as One and a half Syndrome.

Vertical gaze pathway

00:37:54



- riMLF (rostral interstitial MLF) → Primary center for vertical gaze.
- The riMLF of both sides communicate via the posterior commissure.
- The riMLF receives inputs for vertical gaze from the frontal & occipital lobes & from the superior colliculus.

Outputs from the riMLF go to 3 structures :

- INC Interstitial nucleus of Cajal.
- Trochlear nerve nucleus (SO).
- Oculomotor nerve nucleus (IR, IO & SR).

↓

Secondary center of vertical gaze.

Causes of vertical gaze palsy

00:40:49

1. Distal basilar artery ischemia → most common cause.
2. Progressive supranuclear palsy → 2nd common cause.
   - Initial gaze affected is down gaze.

↓

Upgaze.

↓

Global gaze palsy.

↓

Convergence palsy.

   - Occurs in old age.
3. Parinaud's dorsal midbrain syndrome :
   - Site of lesion → Posterior commissure.
   - Most common cause → Pinealoma.
   - Clinical features :

     Upgaze palsy.

     Collier's sign → Upper eyelid retraction.

Light near dissociation → Light reflex is absent & near reflex is present.

Comitant squint

00:45:17

Usually presents as Esotropia (convergent squint).

Comitant esotropia.

- Accommodative.
  - Refractive.
    - Hypermetropia
      - Esotropia
  - Non refractive.
    - Due to either :
      - Increased convergence / Decreased accommodation.
      - Esotropia at near point > far point.
      - Treatment :
        1. Spectacles (bifocal).
        2. Miotics like ecothiophate.
        3. Surgery :
           - Bimedial recession.
           - FADEN surgery. (retro equatorial myopexy).

Non-accommodative esotropia :

It is known as Essential Infantile esotropia.

Associated with :

- Latent horizontal nystagmus.
- IO overaction.
- Dissociated vertical deviation.

Always a large angle squint > 30 prism dioptre → Leads to amblyopia in the deviated eye.

Treatment :

1. Preventive amblyopia
   - Occlusion therapy → Patch normal eye.

     method :

     Occlude the normal eye for days = Age of the

child in years.
Followed by occlusion of the deviated eye = 1 day.

- Penalization → use atropine eye drops in the normal eye.

2. Surgery :

Recession ↓ Lengthening ↓ Weakening a muscle.

&

Resection ↓ Shortening ↓ Strengthening a muscle.

So, for esotropia medial rectus recession & Lateral rectus resection is done.

Pseudostrabismus (apparent deviation) 00:59:34

Pseudostrabismus

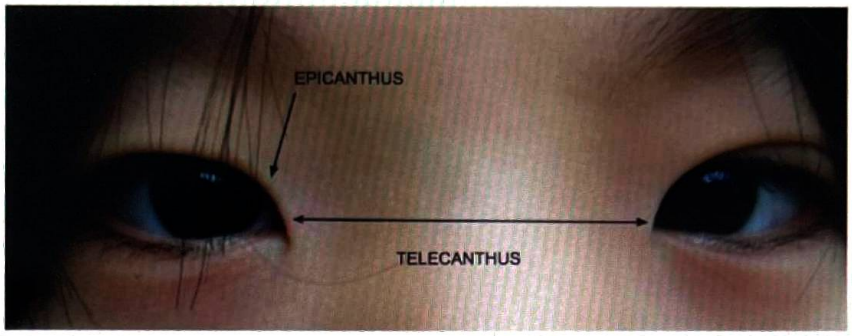
- Pseudoesotropia. (Apparent convergent squint).
- Pseudoexotropia. (Apparent divergent squint).

1. Telecanthus : Increased Inter canthal distance (ICD). Normal Inter-pupillary distance (IPD).	1. Hypertelorism : Increased ICD. Increased IPD.
2. Epicanthus : A congenital nasal fold of skin connecting the medial part of the upper lid and lower lid.	2. Retinopathy of Prematurity (ROP).
3. Myopia.	3. Hypermetropia.

Epicanthus :

Medial canthi not seen.

Example of Pseudoesotropia.



Ocular myopathies

01:06:34

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1. Myasthenia gravis :

Ocular features include :

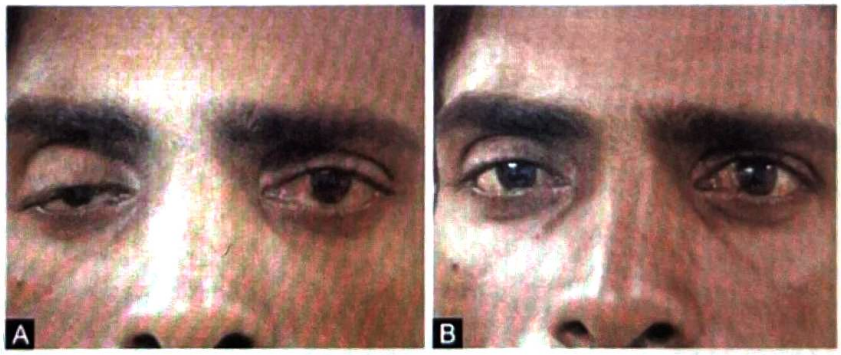
- Ptosis → Usually bilateral & asymmetrical.
- Ophthalmoplegia (restriction of ocular movements) → Partial/ Total.
- Diplopia.
- Cogan lid twitch sign → upshoot of upper lid when changing gaze from down to primary position.

Diagnosis :

Ice pack test.

Tensilon test / Edrophonium test :

- Short acting anticholinesterases.
- 0.2 mg EDR ——→ Increased Ach at cleft. ——→ Improved myasthenia ptotic eyelid.



1. Chronic Progressive Extenal Ophtalmoplegia (CPEO) : mitochondrial disorder.

Causes :

- Idiopathic.
- Kearus sayre syndrome.
- Associated with Oculopharyngeal dystrophy.

Ocular features :

- Ptosis → First sign.
- Ophthalmoplegia → Upward ——→ Lateral——→ Downward (Order of loss of Gaze).
- No Diplopia.

LENS: ANATOMY AND METABOLISM

Introduction to Lens

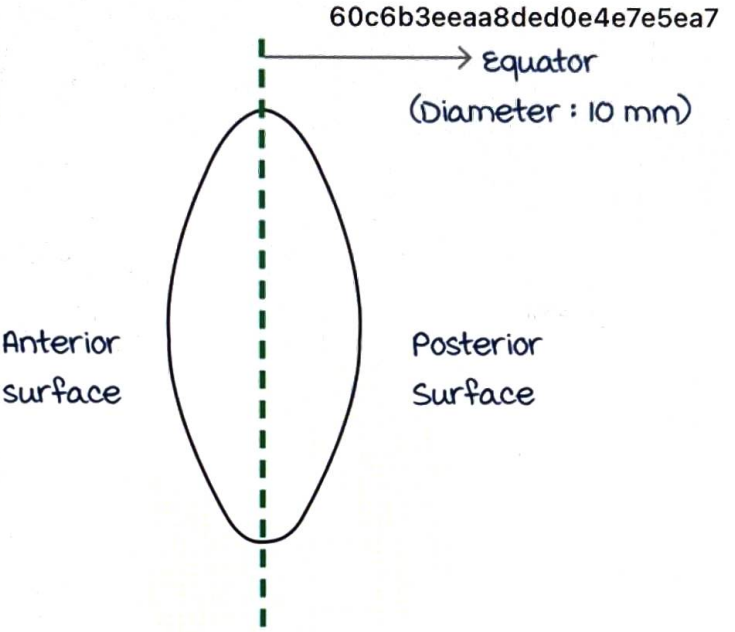
00:01:43

Colour : Transparent.

Blood supply : Avascular.

Nerve supply : No innervation.

Shape : Biconvex



- Anterior surface : Less convex, slightly flatter.
  Radius of curvature : 10 mm.
- Posterior surface : More convex, curved.
  Radius of curvature : 6 mm.
- Equator diameter : 10 mm.
- Anteroposterior diameter.

Position / site of lens : Patellar fossa

Refractive power of lens : +16D to +19D.

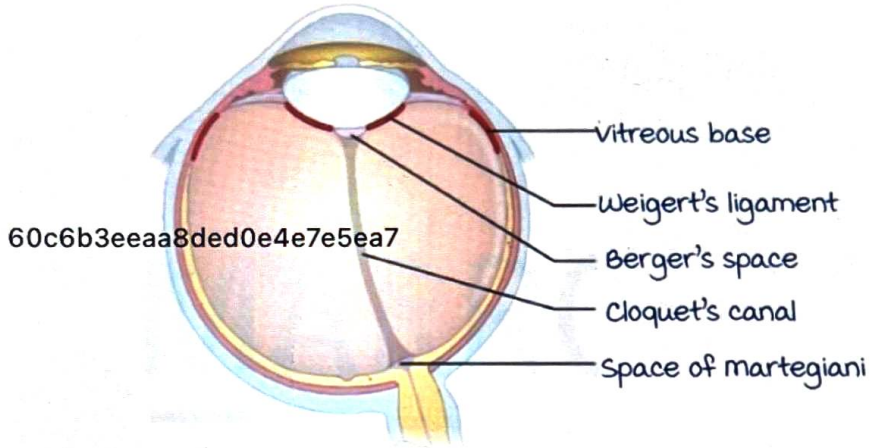
Refractive index of lens :

- Central part : 1.406 (Part of the eye with maximum refractive index).
- Peripheral part : 1.386

Lens develops from : Surface ectoderm.

Active space

Lens and associated structures 00:11:50



Weigert's ligament : Attachment between posterior surface of lens and anterior face of vitreous.

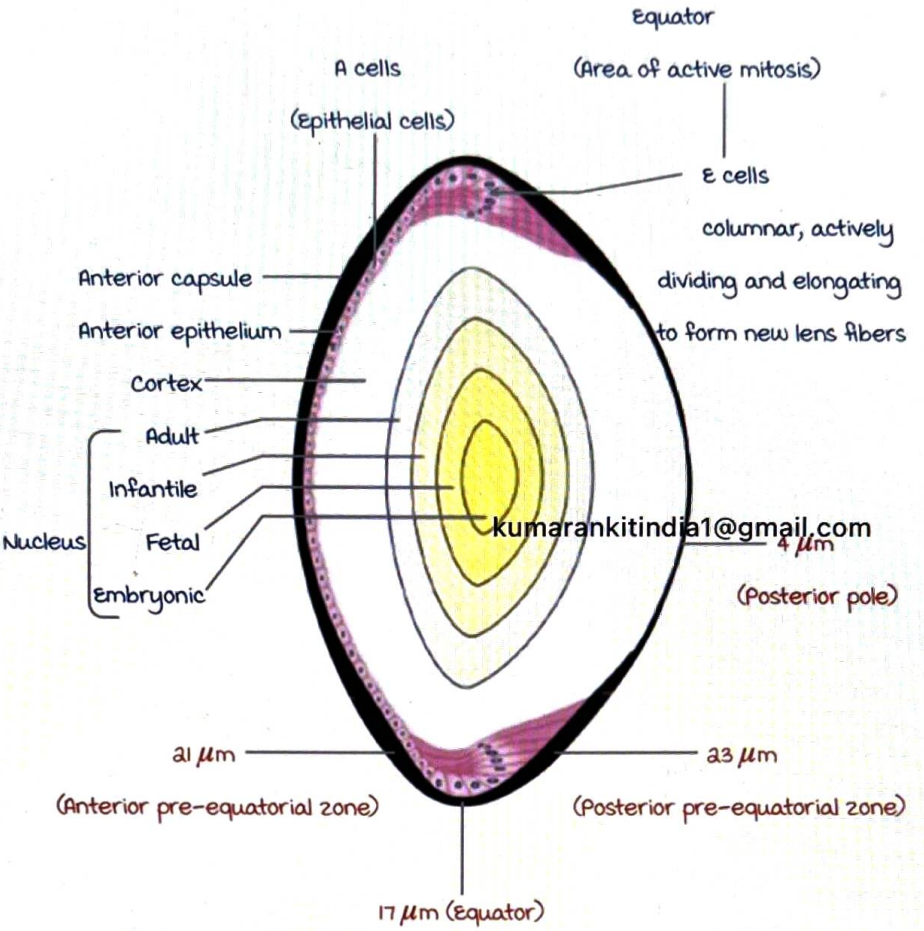
Berger's space / Retrolental space :
Space between the lens and vitreous.

Cloquet's canal :

- Potential space for Hyaloid artery to supply lens during embryogenesis.
- Cloquet's canal and Hyaloid artery disappear by 8 months of gestation.
  If they persist, can lead to :
  1. Mittendorf dots :
  Remnant of Hyaloid artery on the posterior surface of the lens.
  2. Bergmeister's papillae :
  Remnant at the optic disc.

Layers of lens

00:18:25



1. Outer most layer : Capsule
   - Capsule is the thickest basement membrane in the body : Type IV collagen.
   - Thinnest at : Posterior pole.
   - Thickest at : Posterior pre-Equatorial zone .

2. Anterior epithelium :
   - Have 2 cells :
   1. A cells : metabolism
   2. E cells : Form new lens fibres as they are capable of mitosis.
   - Posterior epithelium is absent as during embryogenesis, posterior epithelial cells elongate to form Primary lens fibres.

3. Cortex
   - Young lens fibres found in Outer cortex.

4. Nucleus :
   - 4 zones :

i. E : Embryonic center : Primary lens fibres.
Oldest lens fibres.

ii. F : Fetal
iii. I : Infantile
iv. A : Adult
} Secondary lens fibres.

Both Cortex and Nucleus contain lens fibres.

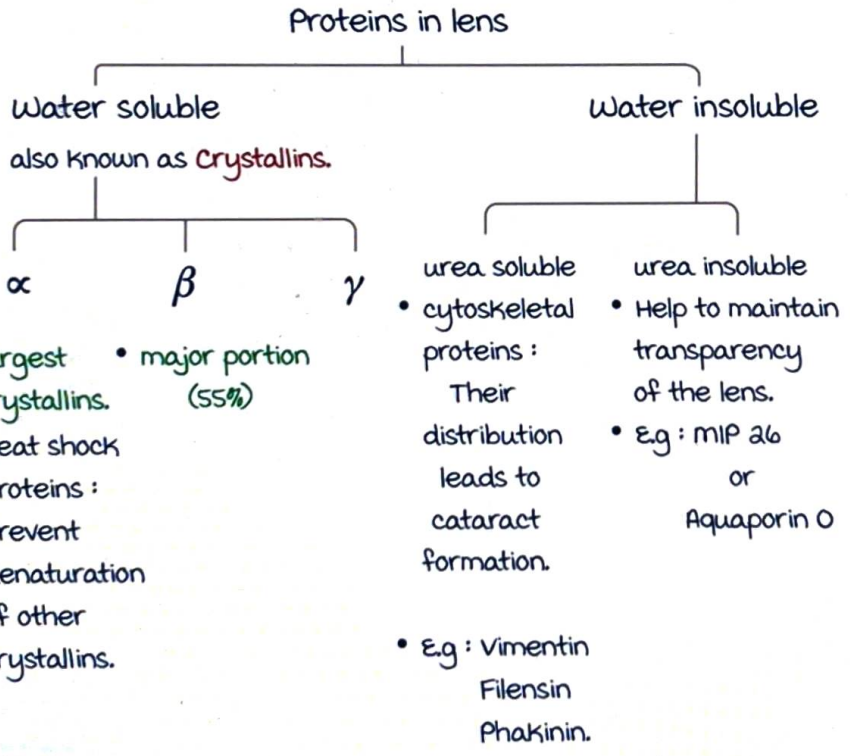
Sutures in lens 00:31:49

Formed during fetal period. 60c6b3eeaa8ded0e4e7e5ea7

Anterior suture : Y shaped.

Posterior suture : Inverted Y shaped.

Proteins in lens 00:34:50



HMW (High Molecular Weight) proteins :

- HM 1, HM2 } Water soluble
- HM3, HM4 } Increase in concentration in cataract.

HM4 alone shows increased concentration in Nuclear cataract.

Active space

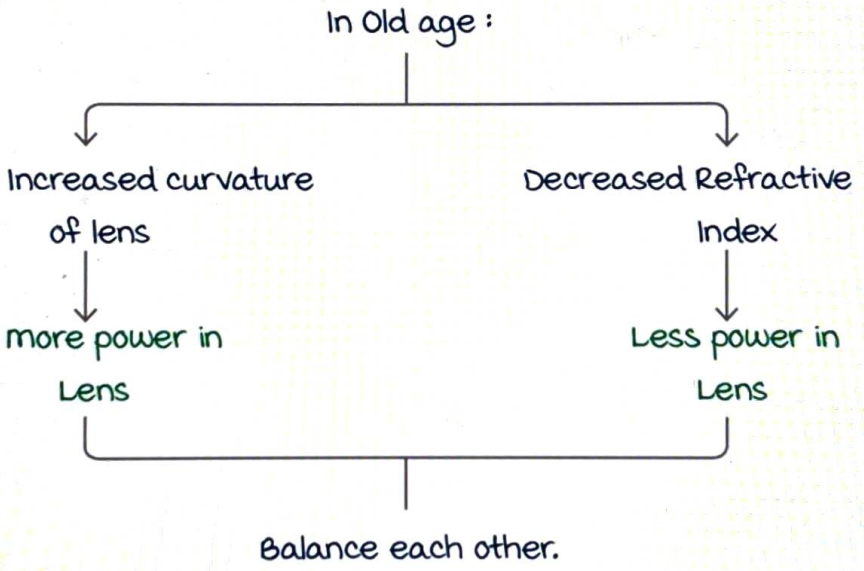
Antioxidant mechanisms in lens and Lens paradox

00:42:50

1. Superoxide dismutase
2. Catalase
3. Glutathione reductase
4. Vitamin C
5. Vitamin E
6. Low oxygen levels around lens :
   In vitrectomy, low oxygen level is lost. This leads to increased incidence of cataract formation.

Vitamin A is not an antioxidant in lens.

Lens paradox :



Metabolism in lens

00:48:55

Glucose diffuse inside lens from Aqueous (mainly) and also from Vitreous.

This glucose is metabolised by :

- Anaerobic glycolysis : > 80%
- Krebs cycle or HMP shunt : Around 15%
- Sorbitol pathway : <5%

Active space

Sorbitol pathway is active in :

- Diabetes
- Galactosemia

] Sugar cataract

Glucose —Aldose Reductase→ Sorbitol
(cannot diffuse out of the lens)

↓

Accumulates in lens.

↓

Increased osmotic pressure in lens.

↓

Fluid is drawn inside lens

↓

Opacification, known as Cataract.

Transport of ascorbic acid in lens occurs via Sodium dependant SVCT2 (Active process).

Active space

LENS : ACQUIRED CATARACT

Introduction 00:00:30

Any opacity in lens is called cataract.
MC cause of gradual painless loss of vision → Cataract.
MC cause of blindness in India → Cataract.

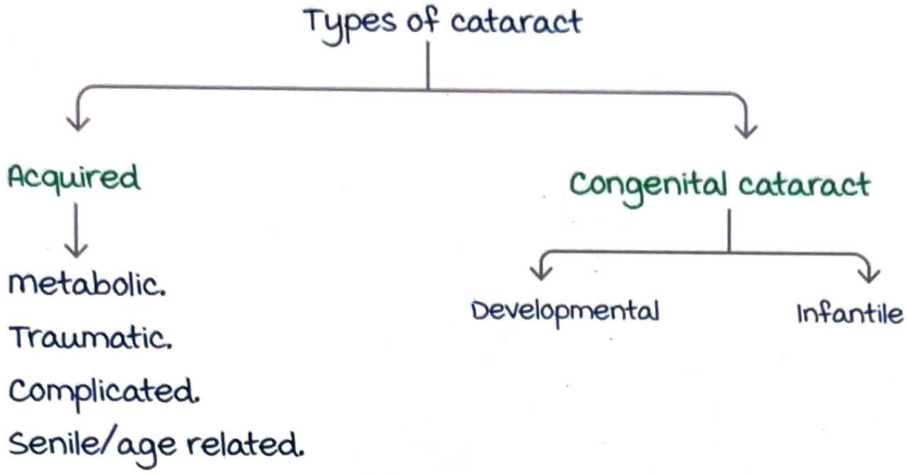
Blindness :

Program → NPCBVI (National Programme for Control of Blindness and Visual Impairment).
Definition → Visual acuity < 3/60 in the better eye with best possible correction or visual field < 10°.

Risk factors of cataract 00:06:06

Risk factors for cataract	Pathomechanism of loss of transparency
• Increasing age.	• Oxidative damage to membranes and proteins.
• Sunlight (UV-A, UV-B).	• Hydration.
• Severe diarrheal dehydration.	• Denaturation of proteins.
• Vitamins A, C, E deficiency.	• Opacification of lens fibres with fibrous metaplasia.
• Diabetes.	• Opacification of lens epithelium.
• Smoking.	• Accumulation of pigmented molecules (3-hydroxyky-nurenine and chromophores which lead to yellowing).
• Corticosteroids.	• Disturbance in balance.
• Genetics.	• Failure of ion pumps (increased intralenticular calcium & sodium with decrease in potassium)

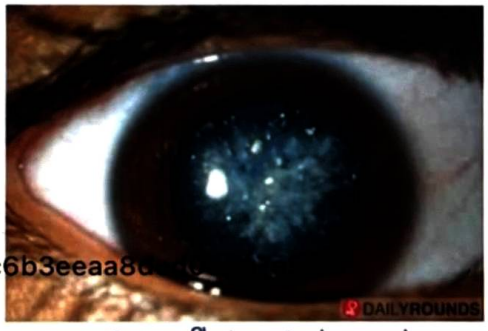
Active space



Metabolic cataract : Diabetes mellitus 00:08:08

Diabetes mellitus causes :

1. Snow flake cataract (cortical cataract).
   MC in type 1 DM (Young adults).
2. Presenile cataract: MC in type 2 DM (senile cataract before 50 years of age) it occurs due to increased carbamylation of crystallines.
3. Fluctuating refractive errors.
4. Diabetic retinopathy.



Snowflake Cataract

Metabolic cataract : Galactosemia 00:13:47

Galactosemia causes :

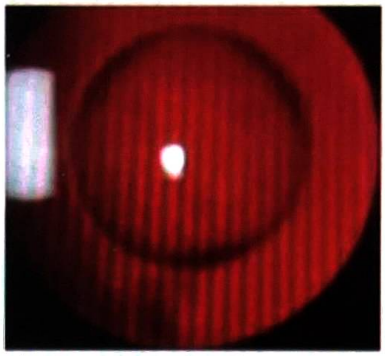
Oil droplet cataract (posterior subcapsular cataract).

This is the only reversible cataract.

Galactosemia is an autosomal recessive disorder.

Galactose ——×——→ Glucose

Active space



Galactosemia → Oil Droplet Cataract

Galactose & its metabolites galactitol (dulcitol) accumulate in the lens to cause cataract.

The enzymes deficient are :

- Gal-1-PUT.
- UDP galactose 4 epimerase.
- Galactokinase.

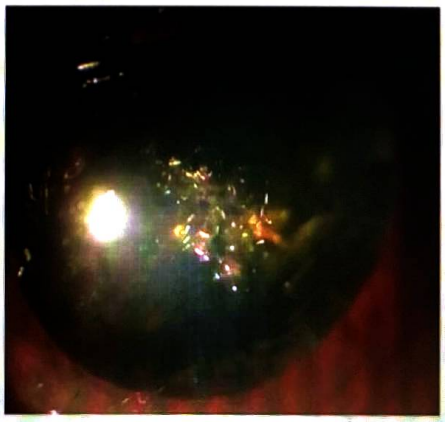
Galactosemia due to galactokinase deficiency causes only cataract.

Galactosemia due to Gal-1-PUT and UDP galactose-4-epimerase causes mental retardation, hepatomegaly, failure to thrive.

Metabolic cataract : Myotonic dystrophy 00:17:50

It causes :

1. Christmas cataract (posterior subcapsular cataract)
2. Presenile cataract



Myotonic Dystrophy → Christmas tree cataract

Metabolic cataract : Wilson's disease

00:19:37

It causes :

Sunflower cataract → Due to the deposition of cuprous oxide and it can affect multiple sites of the lens.

It is stellate shaped.

Kayser-fleischer ring → Golden brown ring due to the deposition of copper in descemet's membrane in the periphery of cornea.

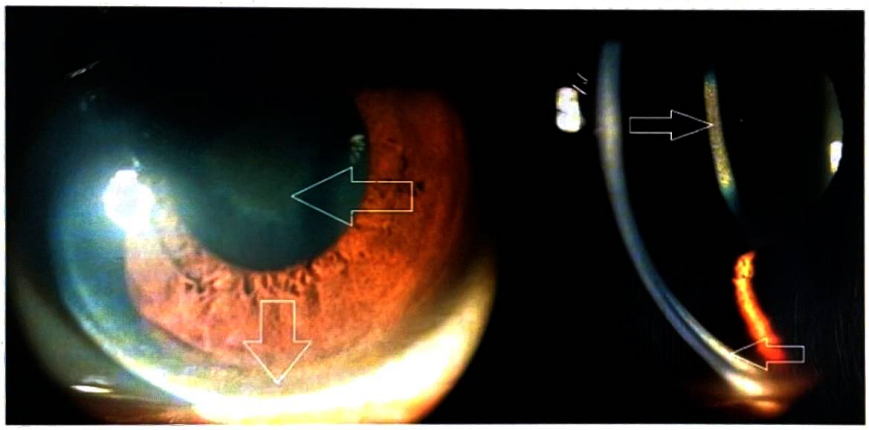
Also extends into the angle of anterior chamber-trabecular meshwork.

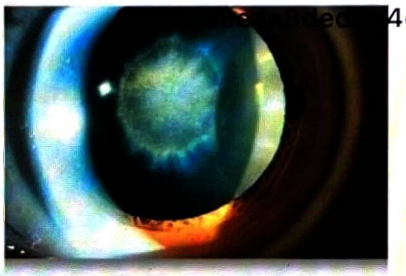
There is no loss of vision due to KF ring.

KF ring is seen in all patients with neurological manifestations (Deposition of Cu in basal ganglia).

Seen in around 50% patients with hepatic involvement.

Starts superiorly ⟶ Inferiorly ⟶ Laterally.





Treatment :

1. Chelating agents → Penicillamine and trientine (Lifelong).

KF ring disappears after 3-5 years.

2. Maintainence therapy with zinc acetate, it stops the absorption of copper.

KF ring can also be seen in :

1. Primary biliary cholangitis.
2. Neonatal hepatitis.

Parathyroid tetany :

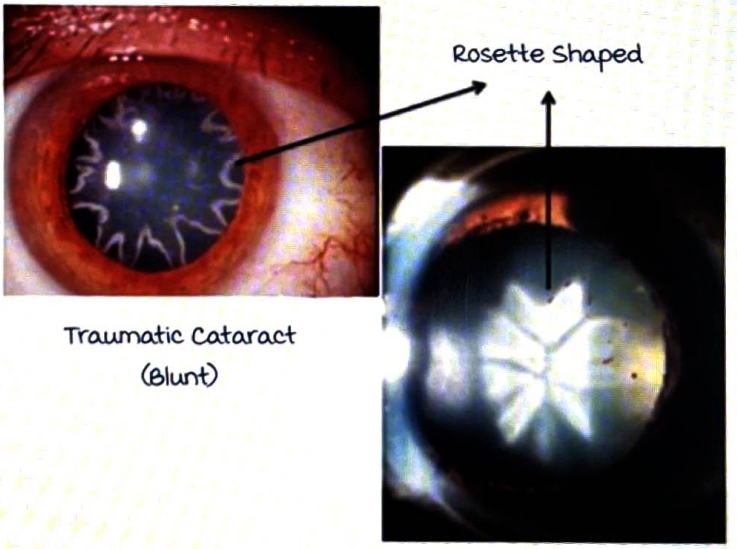
It leads to hypocalcaemia, which can lead to cataract in both children and adults.

Adults → Anterior or posterior subcapsular cataract

Congenital → Lamellar cataract.

Traumatic cataract 00:29:56

1. Blunt trauma causes rosette shaped cataract which is a posterior subcapsular cataract.
2. Infrared rays causes Glass blower's cataract, occurs due to true exfoliation of lens capsule.
3. Lightning/electric shock → Anterior capsular opacities.



Traumatic Cataract (Blunt)

Complicated cataract 00:33:29

The causes are :

1. Uveitis → most common cause.
2. Myopia.
3. Angle closure glaucoma.
4. Retinitis pigmentosa.

Appearance → Bread crumb appearance with polychromatic lusture.

Site → Posterior subcapsular cataract.

Senile cataract : Nuclear cataract pathology 00:36:55

It is also known as age related cataract.
It occurs around 50 to 70 years of age.
There are four types :
1. Nuclear cataract :
AKA Central cataract.
Clinical features → Loss of vision in daytime.
Improvement of vision in dim light/night.

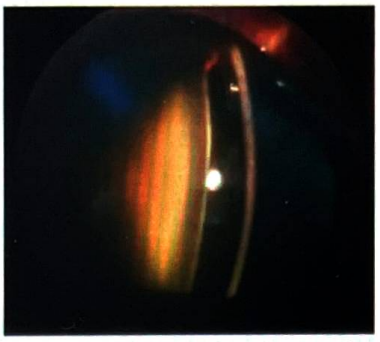
Pathology → It occurs due to sclerosis, it causes hard cataract.
↓
Increase in refractive index of lens.
↓
Increase in power of lens (converging of lens).
↓
Index myopia : Loss of far vision or near sightedness leads to second sight phenomenon.

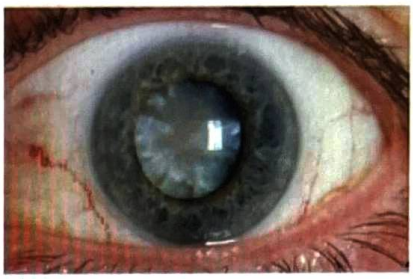
Second sight → Improvement of near vision due to nuclear cataract in a patient who is already presbyopic/ hypermetropic

Senile Cataract : Nuclear cataract colour 00:48:30

Colour
Grade 1 : Yellow.
Grade 2 : Amber.
Grade 3 : Brown called as cataracta brunescens.
Grade 4 : Back called as cataracta nigra.

Color is due to the deposition of urochrome pigment.
Clinically, patient complains of yellow colored vision known as xanthopsia.





On slit lamp examination with oblique illumination → Grading.

Senile cataract : Cortical cataract

00:54:45

AKA Peripheral cataract.

Loss of vision in dim light/night/nyctalopia.

Pathology → Hydration of lens causes soft cataract.

The earliest change : Formation of water clefts & vacuoles.

↓

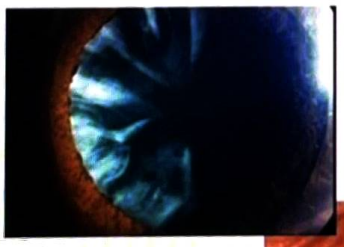
Lamellar separation.

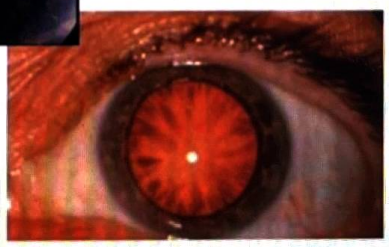
↓

Wedge shaped opacities are seen : Cuneiform cataract (MC seen inferonasally).

↓

Immature cataract





Immature cataract

00:59:45

It is seen as greyish white colour.

Iris shadow is seen over lens surface.

As the fluid accumulates it causes swelling of the lens and immature cataract progresses to intumescent cataract.
It is also called a mature cataract.
It is chalky white in colour ; No iris shadow.

Mature cataract progresses to hypermature cataract.

It can present as

- Morgagnian
  - Liquefaction of cortex.
  - ↓
  - Leads to sinking of nucleus.
- Sclerotic
  - Anterior capsule becomes wrinkled, leathery.
  - Pearly white as calcification.

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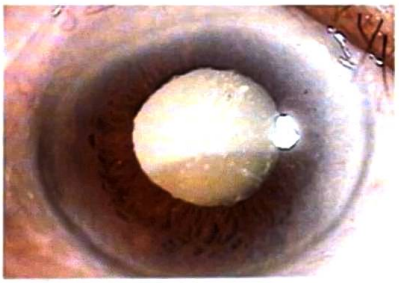
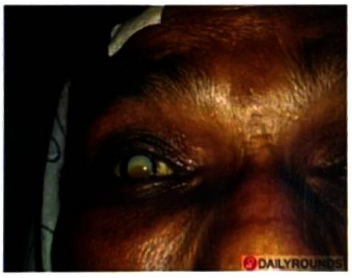
MC complication of hypermature cataract is subluxation of lens.
MC complication of morgagnian cataract is phacolytic glaucoma.





Clinical features :
1. Diplopia.
2. Glare.

Posterior subcapsular cataract 1:08:28

AKA Cupuliform cataract.
It causes maximum visual loss because opacity is present axially at the nodal point of the eye.
C/F → more loss of near vision & daytime (central).
Vision improves in dim light.
Glare (inability to drive at night).

Active space

Pathology → It is due to dysplasia of 'E cells'

↓

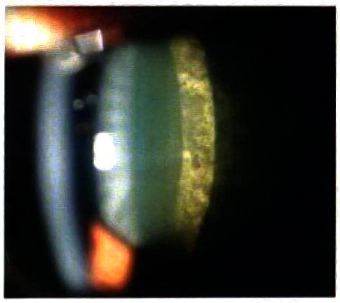
Leads to posterior migration & enlargement.

↓

Formation of plaque at the posterior surface called bladder cells or Wedl cells.

Causes of posterior subcapsular cataract :

- Senile.
- Galactosemia.
- Myotonic dystrophy.
- Blunt trauma.
- Complicated cataract.
- Corticosteroids (systemic).
- X rays.
- Chloroquine.
- Busulfan.
- Neurofibromatosis 2.



Anterior subcapsular cataract

1:17:15

Other causes of anterior subcapsular cataract :

1. Atopic dermatitis : Shield cataract.
2. Amiodarone.
3. Chlorpromazine.
4. Gold deposits.
5. Acute angle closure glaucoma (AACG).

LENS : CATARACT SURGERY AND COMPLICATIONS

Cataract surgeries 00:01:11

1. ICCE : Intra capsular cataract extraction.
   The whole lens with capsule is removed.
   Not performed anymore.
   Only indication : Subluxation of lens due to > 180° of zonular dehiscence.

2. ECCE technique based surgeries :
   Extra capsular cataract extraction.
   Whole of the lens removed except the capsule.
   a) Conventional ECCE.
   b) SICS : Small incision cataract surgery.
   c) Phacoemulsification.

	Site of incision	Size of incision
Conventional ECCE	At the limbus	8 – 10 mm (since equitorial length of lens is 10–10.5 mm)
SICS	Sclera	5 – 7 mm
Phacoemulsification	Cornea	2.2 – 3.2 mm
microincision (MICS)	Cornea	< 1.8 mm

Smaller the size of incision, greater the speed of healing and lesser chance of post op infection.

d) FLACS : Femtosecond laser assisted cataract surgery.
   Latest surgery.
   Laser used : Nd:Glass.
   Wavelength : 1053 nm.
   Mechanism of action :
   1. Burning : Photoablation.

2. Breaking : Photodisruption.

3. Coagulation.

In cataract surgery : Photodisruption.

Laser is fired for femtosecond = 10^{-15} second.

This reduced the collateral damage to the surrounding tissues.

Phacoemulsifaction 00:13:19

Most commonly done surgery nowadays.

Steps :

1. Preoperative :

- Dilate the pupil : mydriatic and cycloplegic, usually : Tropicamide + phenylephrine or cyclopentolate.
- Local anaesthesia :

    1. Topical.
    2. Peribulbar.
    3. Retrobulbar.

  Peribulbar > Retrobulbar :

  Bupivacaine + Lignocaine + 1 : 2,00,00 Adrenaline + Hyalurodinase (for better penetration).

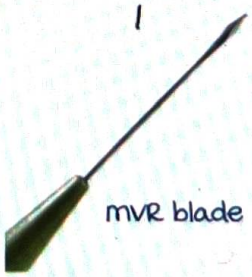
  Nowadays, topical anesthesia is used :

  Proparacaine eye drops.
- Universal prophylaxis : 5% povidone iodine or betadine solution instilled into the conjuntival sac.

Intraoperative steps of phacoemulsifaction 00:27:20

1. Side port incision : Can be made with either of these instruments :

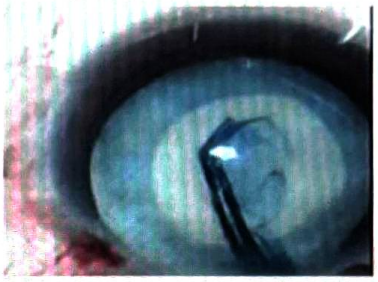
15 degree blade



MVR blade

2. Staining of the anterior capsule of lens : Trypan blue dye.

3. CCC : Continous curvilinear capsulorhexis :
Created with the help of a 26 gauge bent needle or created using a utrata forceps.



4. Main incision is created : Preferred to create the incision after capsulorhexis by a 2.2 keratome.

5. Hydrodissection :
Injection of jet of fluid to separate the cortex of the lens from the capsule.
A plane of separation is created so that while evacuating the lens the posterior capsule is untouched.

6. Hydrodelineation :
Injection of jet of fluid to separate the endonucleus from epinucleus.
Optional step.
Done if in nuclear cataract, the hard endonucleus requires more energy to emulsify, hence creating planes is easier to remove the parts individually.

7. Nuclear fragmentation and emulsification with phacoemulsification probe :
The probe, at the tip has a peizoelectric crystal.
This crystal converts ultrasonic waves in mechanical to and fro motion.

Active space

Hence, fragments the nucleus.

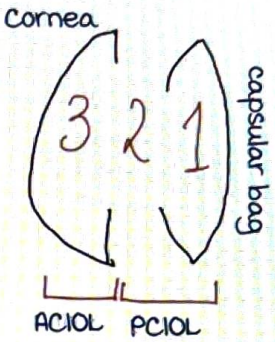
8. Irrigation and aspiration :
For cortical clean up.
Either done with a two-way cannula or bimanually.

9. Implantation of IOL.

Intraocular lens (IOL)

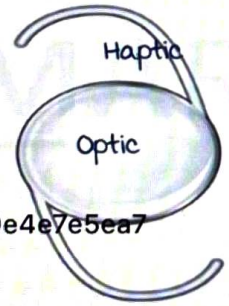
00:38:10

Implantation can be done :
1. Capsular bag. (PCIOL) : Best site.
2. In the sulcus : If the posterior capsule is ruptured during surgery.
3. Anterior chamber (ACIOL).



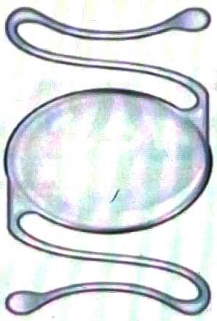
ACIOL :
Made up of PMMA : Polymethyl methacrylate.

PCIOL :
1. Rigid IOL : PMMA.
2. Foldable IOL : Acrylic (rare : silicone).

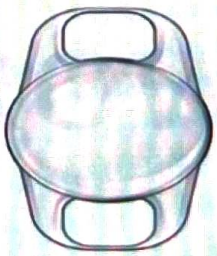


PCIOL
(modified C loop type)

PCIOL
(four loop type)



ACIOL
(Kelman multiflex)



Singh and Worst's
iris claw lens

If C shaped haptic : PCIOL.

If S shaped haptic : ACIOL.

Multifocal IOL :

Has power for far, intermediate and near vision.

Has concentric rings on the IOL.

Side effect : Leads to glare in bright lights.

Loss of brightness in scotopic conditions.

Toric IOL :

Used when there is preexisting astigmatism.

Square edged IOL :

Decreased the chances of posterior capsular opacification.

Biometry

00:48:10

Calculation of IOL power.

Step 1 : measure the corneal curvature : Keratometry.

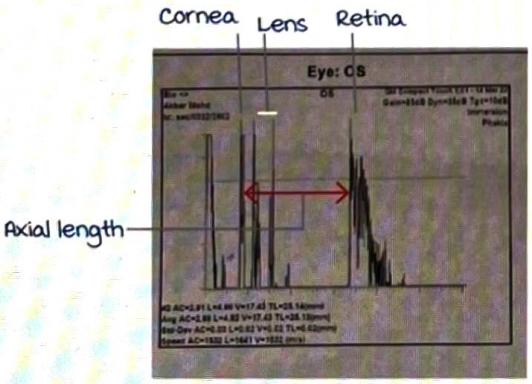
Step 2 : Axial length of eyeball :

Anteroposterior diameter.

Normal : 2-4 cm/ 24 mm.

measured by A (amplitude) scan USG.

Uses 8 MHz sound waves.



Step 3 : Formulae

a) SRK -T formula.

b) Holladay's formula (if AL > 24.5 mm).

c) Hoffer Q formula (if AL < 22 mm).

d) Haigis L formula : Forth generation formula used post LASIK cases.

Active space

Complications of cataract surgery 00:55:54

Operative complications :

1. Posterior capsular rupture.

2. Uveitis- glaucoma-hyphema syndrome (UGH syndrome) :
Due to iris chaffing by ACIOL.
Iris is a movable structure hence will move against the IOL and lead to chaffing which leads to uveitis.
Due to pigimented nature of the iris, the molecules block the trabecular meshwork and lead to glaucoma.
During rubbing the vascular iris might rupture a vessel and lead to hyphema.
Hyphema : Collection of blood in anterior chamber.

3. Vitreous loss :
Most serious complication.

4. Expulsive choroidal hemorrhage : Bleeding from choroidal artery, due to sudden IOP changes.
Source : Short posterior ciliary artery.

5. Damage to superior rectus while passing bridle suture :
Only in SICS and ECCE to stabalise the eyeball.

6. Descemet membrane detachment :
During instrumentation if the descemet membrane is detached, there can be formation of an opacity.
Therefore, reattached by injecting air bubble or gas in the AC : Descmetopexy.

Acute postoperative complications 01:05:06

Complications occuring within 3 weeks.

1. Iris prolapse.

2. Acute postoperative endophthalmitis :
Suppurative inflammation of all layers of the eyeball except

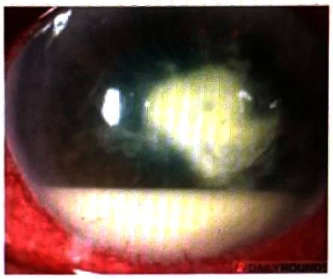
sclera.

Infection

Acute post operative endophthalmitis :	Late onset post operative endophthalmitis :
Within 7 days of surgery	6 weeks to 6 months after surgery
Staphylococcus epidermidis (CONS)	Propionibacterium acnes

Clinical features :

1. Severe pain.
2. Redness.
3. LOV.



On examination :

1. Congestion/ Hyperemia.
2. Cornea haze.
3. Hypopyon : collection of pus in AC.
4. Vitritis : Yellow exudates in vitreous.

Treatment :

Based on vision

Blind : No perception of light	Vision still present
Evisceration	Antibiotics : Intravitreal : Vancomycin Img on 0.1 mL + Ceftazidime 2.25 mg in 0.1mL Topically : Broad spectrum. + Pars plana vitrectomy

No requirement of systemic antibiotics.

3. Uveitis.
4. Retained lens matter.
5. Hyphema.
6. Astigmatism.
7. Retinal detachment.
8. Toxic anterior segment syndrome (TASS) :

Active space

Sterile inflammatory reaction to toxic products on IOL or viscoelastic.

Treatment : Steroid.

9. Flat/shallow AC :

a) Wound leak.

b) Ciliary choroidal detachment.

c) Pupillary block glaucoma.

d) Malignant glaucoma.

Late postoperative complications 01:21:22

Complications occuring after 1 month to years.

1. PCO : Posterior capsular opacification :

Most common late complication.

Also known as secondary cataract/after cataract (misnomer).

6 to 10 weeks post surgery.

Gradual painless LOV.

Treatment : Nd:YAG laser posterior capsulotomy.

Wavelength : 1064 nm.

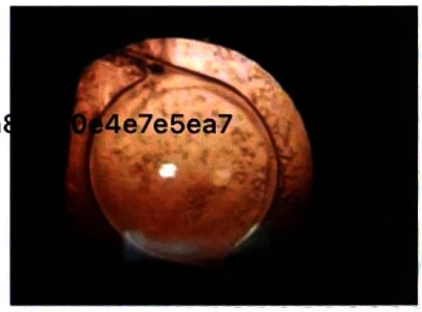
Mechanism of action : Photodisruption.

Types of PCO :

1. Elshnig pearls.

(most common, 90%)

2. Sommering's Ring.



2. Irvin Gass syndrome :

Cytoid macular edema + bullous Keratopathu + vitreous touch

3. Diplacement of IOL :

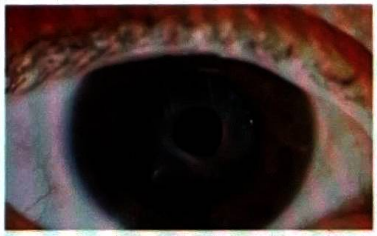
1. Sunset syndrome : inferior subluxation.

2. Sunrise syndrome : Superior subluxation.

3. Lost lens syndrome : Complete dislocation.

4. Anterior capsular phimosis :

Comtration of anterior capsule due to fibrosis causing IOL decentration



5. Late onset endophthalmitis.

Active space

LENS - CONGENITAL CATARACT, AND ECTOPIA LENTIS

Pathogenesis of cataract (C) starts in utero and presents at birth or at later life.

Classification :

- Infantile cataract :

  Opacities develop before 1 year of age.

  Causes severe vision loss as it interferes with foveal fixation.

  Foveal fixation develops around 3-4 months of age.

  Foveal fixation :

  Brain ascertains that fovea is the best point of vision by 3-4 months of age. Then on visual axis oriented. Such that the light coming from object falls on fovea.

  Fovea : most sensitive part of retina.

  High concentration of cones.

  Central vision is through fovea.

- Developmental cataract :

  Opacities presents after 1 year of age.

  Minimal vision loss.

  Will not interfere with foveal fixation.

  Blue dot/punctate cataract :

  Scattered small blue coloured opacities.

  Puncta : Dot.

  Most common developmental cataract/ MC congenital cataract/MC congenital cataract that is visually insignificant.

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  Associated with Downs syndrome.

Lamellar/zonular catarct

00:06:38

MC infantile/visually significant congenital cataract.

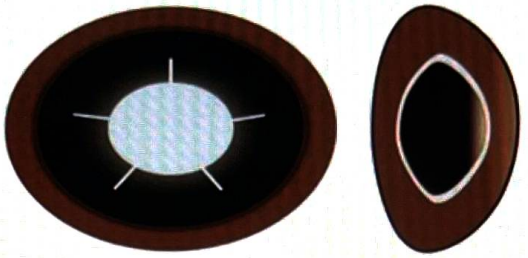
Associated with :

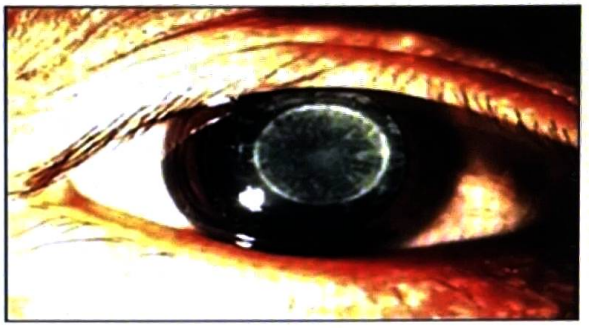
1. Congenital rubella syndrome.

2. Vitamin D deficiency.
3. Inherited as autosomal dominant.

Lamellar cataract is most commonly associated with congenital rubella syndrome .

Most common type of cataract in congenital rubella syndrome is nuclear pearly





Clinical features :

Opacity in fetal nucleus.

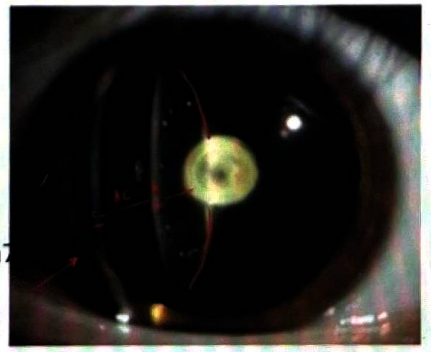
Spoke like opacities are seen known as riders.

Posterior Polar Cataract (PPC) 00:10:58

Onion ring/whorl appearance.

Cataract is identified by the whorled appearance or the location at the posterior capsule seen in the oblique slit examination

Associated with mittendorf dots (remnants of tunica vasculosalentis).



Persistent Hyperplastic Primary Vitreous (PHPV).

PITX3 gene mutation.

Treatment :

Surgery : ECCE techniques.

Lens aspiration + Posterior Capsulorhexis (PC) +anterior vitrectomy (AV) + IOL implantation.

Remove lens but not capsule.

Congenital cataract is soft, hence lens has to be aspirated and nuclear fragmentation is not done.

PC : A window in the posterior capsule is created to reduce posterior capsular opacification (PCO).

AV : To prevent vitreous leakage to anterior segment and vitreous mixing with aqueous.

IOL implantation : Usually after 2 years of age.

But if < 2years of age : Undercorrect IOL power by 20%.

If 2- 8 years of age : Under correct IOL power by 10 %.

This is done as size of eyes increase& AL increases.

Timing of surgery (Sx) :

- U/L cataract & dense opacity :
  One vision is normal in on eye and light cannot go into the other eye, leading to sensory deprivation amblyopia.
  Sx done in 4-6 weeks of age (as early as possible).
  Before foveal fixation & amblyopia.
- B/L cataract & dense : Sx by 12 weeks of age.
- U/L & partial : Occlusion therapy to prevent amblyopia.

Ectopia Lentis (EL)

00:21:19

Ectopia : Displaced, Lentis : Lens.

Displacement of lens.

Causes :

- Blunt trauma (most common).

- Hypermature cataract :
  Most common (MC) complication of hypermature senile cataract is subluxation of lens/EL.
  MC complication of morgagian cataract is phacolytic glaucoma.
- Aniridia.
- Familial :
  Autosomal dominant inheritance.
- Marfan syndrome :
  Direction of EL : Superotemporal.

Other ocular features of Marfan's syndrome.

Megalocornea : Larger corneal diameter.

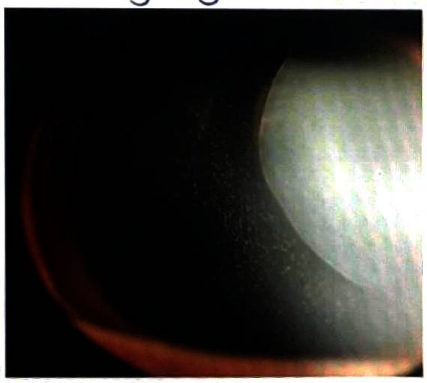
Cornea plana : Flat curvature.

Hypoplasia of dilator pupillae muscle : Incomplete pupil dilation.

AL of eye increases leading to myopia.

- Homocystinuria :
  Direction of EL : Infero nasal.
  Loss of accommodation due to zonular dehiscence.
- Weill-Marchesani syndrome : Forward displacement /anterior EL.
  Causes microspherophakia.
  Patient have short stature, short stubby fingers.
- Hyperlysinemia.
- Sulfite oxidase deficiency.
- Ehlers-Danlos syndrome.

O/E A golden crescent seen in pupillary area.



Len's margin visible.
Zonules are in periphery usually but in EL d/t lens displacement zonules seen.

Clinical feature :

Diplopia :

2 areas of refraction & 2 set of light passing through the pupil.

Hence 2 sets of images formed :

One image is forming in retina but other not forming in retina.

Active space

Lenticonus (LC) 00:32:55

Conus : Cone shaped & lentis : lens.

Cone shaped lens.

On oblique Illumination,

Anterior lenticonus

Anterior LC :

Causes :

S : Spina Bifida.

A : Alport syndrome.

W : Waardenburg syndrome.

Posterior LC :

Cause : Lowe syndrome.

Posterior lenticonus

Microspherophakia :

Micro : Small; Sphero : Spherical.

Small & spherical lens.

Normal lens : Biconvex lens and large.

Resulting in :

Spherical :

Increased curvature of lens.

↓

Power of lens increases.

↓

Curvatural myopia.

Small :

Leads to glaucoma due to luxation of lens to anterior chamber leading to pupillary block.

Lens equator is visible 360° (within pupillary area on dilating).

GLAUCOMA - BASICS

Glaucoma 00:02:02

Definition :

1. Progressive, characteristic optic disc changes.
2. Irreversible visual field changes.
3. IOP may or may not be increased.

Raised IOP is a risk factor for glaucoma.
Optic disc examination done by ophthalmoscopy.
Visual field testing is done by perimetry.
IOP is measured by tonometry.

Theories of glaucoma 00:04:50

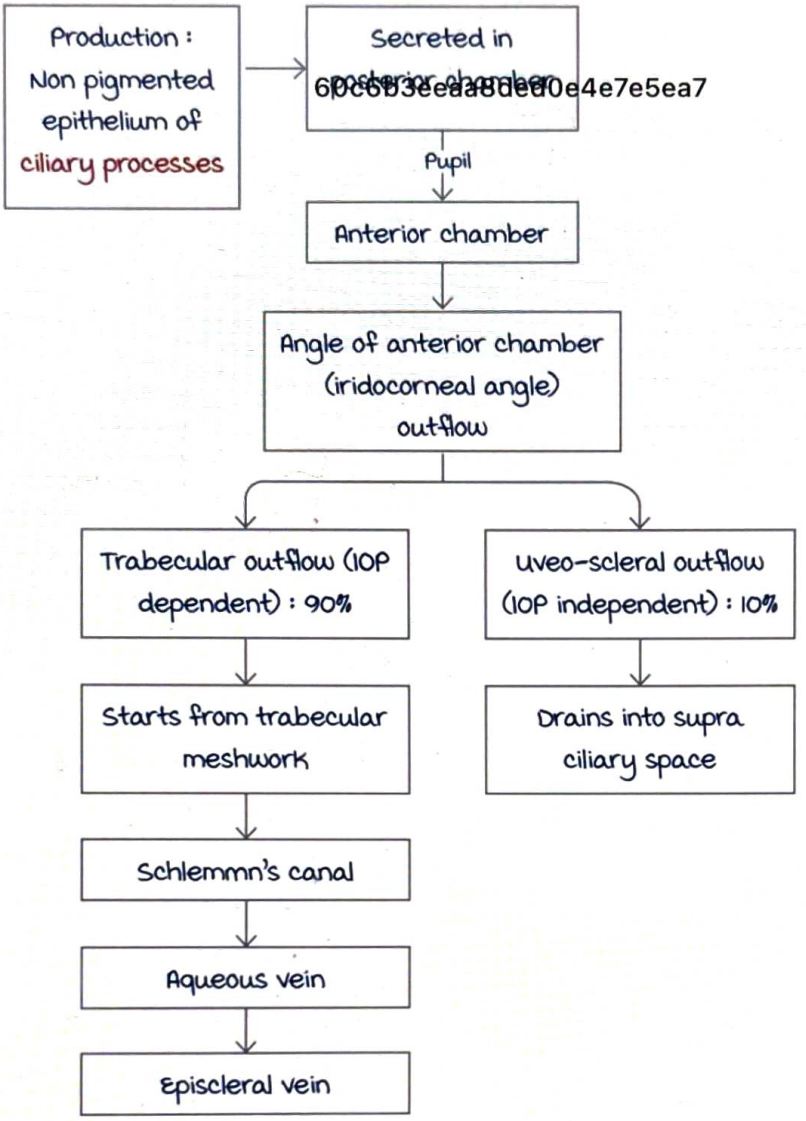
- Mechanical theory (IOP dependent).
- Vascular or ischemic theory (IOP independent).

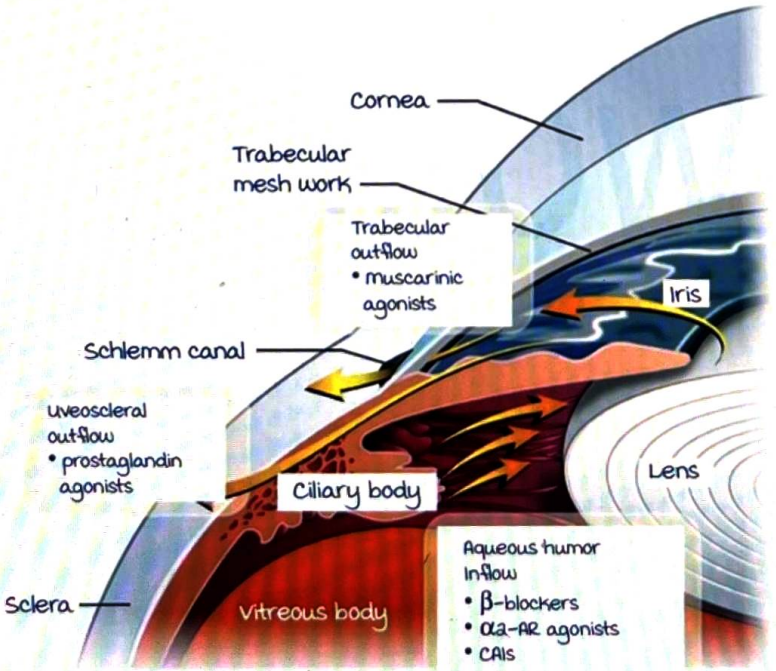
Mechanical theory :
Increased IOP → Blockage of axoplasmic flow → Optic atrophy.

Ischemic theory :
Vascular changes in blood supply to optic nerve → decreased optic disc perfusion → Optic atrophy.

Flow of aqueous

00:09:25





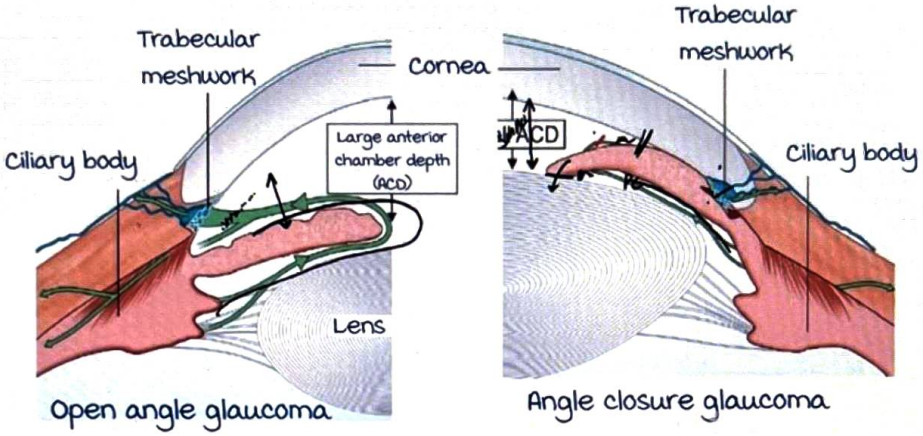
Active space

Angle closure glaucoma :

Structural abnormality → iris moves forward → touches cornea in the periphery → angle closed → Angle closure glaucoma.

Pathology in iris.

Rx : Iridectomy.



Open angle glaucoma :

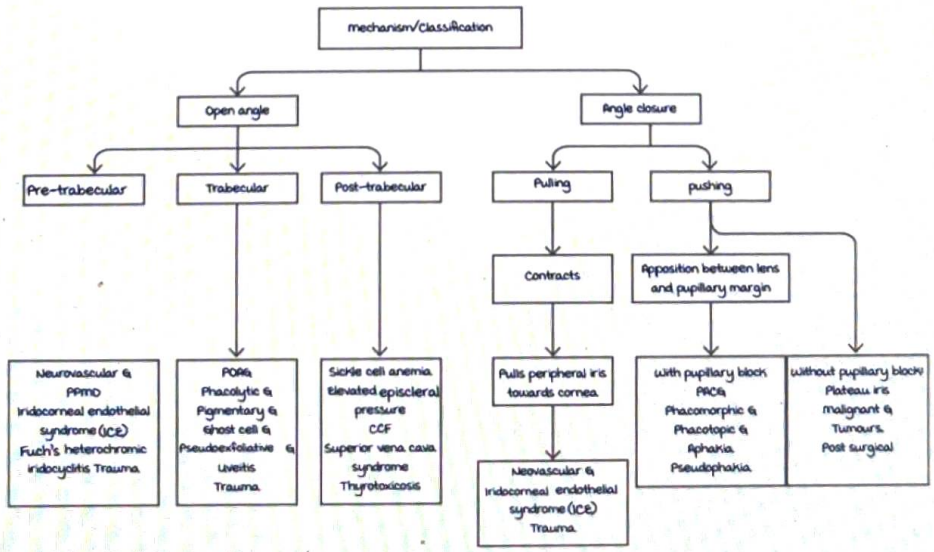
Blockage of trabecular meshwork.

Pathology in trabecular meshwork.

Rx : Trabeculectomy.

Mechanism/classification

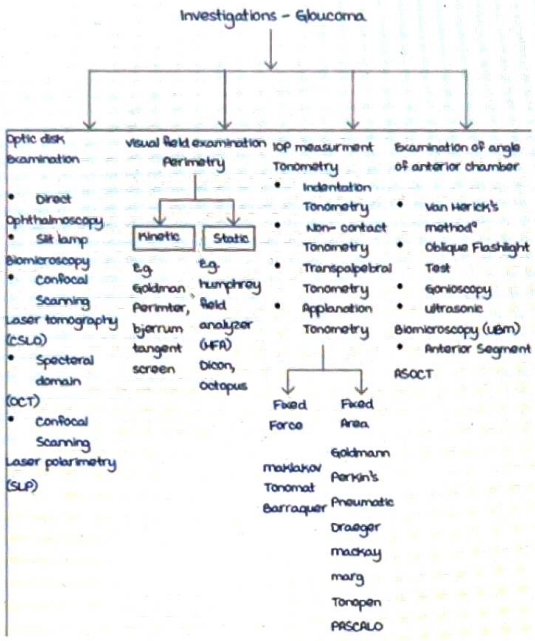
00:23:35



GLAUCOMA - INVESTIGATIONS

Investigations of glaucoma

00:00:53



IOP measurement : Tonometry.

Normal IOP : 11 to 21 mmHg.

IOP has a diurnal variation.

IOP highest in morning (8 am - 12 pm).

IOP measurement is affected by corneal thickness.

Types of tonometry :

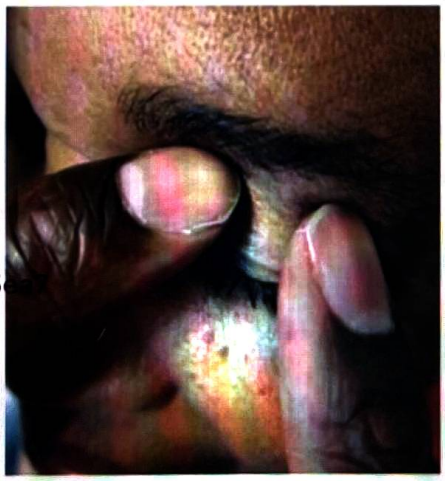
1) Digital tonometry : Done by palpation of eyes.

   Hard touch/rock hard eye : IOP high.

   Soft touch : IOP low.

   Firm touch : Normal IOP.

   Tonometry of choice in patient with Kerato-prosthesis



Active space

2) Indentation tonometry : 2 instruments.

Schiotz tonometer : Intra-observer and inter observer variation.

Steps :

Patient in supine.

Cornea anaesthetized (to eliminate corneal reflex).

Foot plate placed on cornea and plunger is pressed.

Recording of IOP (dependent on ocular ridgidity).

Converted into actual IOP by Friendenwald's nomogram.

Rebound tonometer : Automated tonometer of choice for self monitoring.

Sciotz tonometer

Footplate Plunger

↓

Rests over cornea

3) Applanation tonometry :

Principle : Imbert Fick law → P=F/A.

Types : Fixed force and fixed area.

Fixed force : Maklakov and Barraquer (not done).

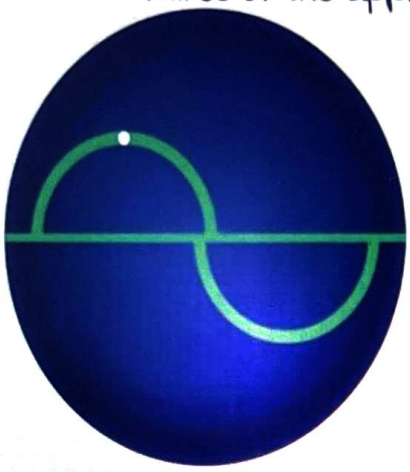
Fixed area : Goldmann's, Perkin's, Mackaymarg, Pneumatic and Pascal.

Goldmann's AT : Gold standard.

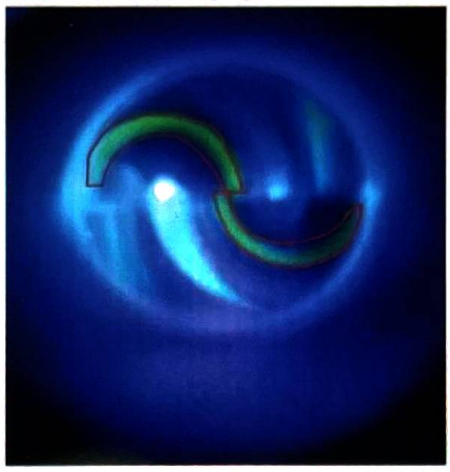
Fixed area that is applanated = 3.06 mm^2.

Mounted on slit lamp.

mires of the applanation tonometer



Perfect alignment of mires



Inner rings of AT mires touch each other → correct end point

Procedure :

Anaesthetize cornea (topical)

↓

Stain cornea with fluorescein dye

↓

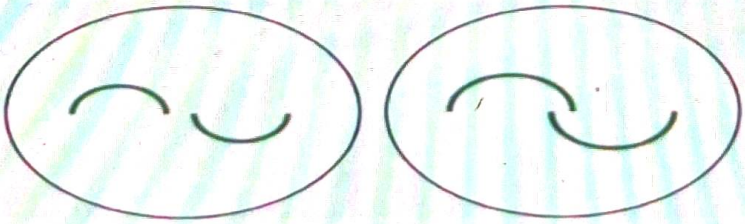
Tip of goldmann's AT illuminated under cobalt blue light touches corneal surface

↓

Biprism in the tip, splits light into 2 semicircles

↓

Adjust force till they properly align



Improper alignment of mires

Perkins AT :

Tonometry of choice in

- Children.
- Bed-ridden.
- Anesthetized (OT).

Hand held instruments.

Digital tonometry

Mackay Marg AT : Tonometry of choice in irregular edematous cornea.

Pneumatic AT : Tonometry of choice in

Continuous IOP measurement.

Through soft contact lenses.

Pascal AT/dynamic contour testing : Does not depend on changes in corneal thickness : Tonometry of choice.

Thick cornea → False high IOP (more force needed)

Thin cornea → False low IOP.

10 μ m increase in CT ∝ IOP changes by 1.

4) Non-contact tonometry :

a) Pulsair/Grolman's tonometry : Used for screening of glaucoma/IOP.
Fires a pulse/jet of air which compresses cornea & measures IOP electronicallly.
Not very accurate.

b) ORA (ocular reichert analyzer) : measures corneal hysteresis (physiological changes that occur in cornea in response to stimulations).
Used only in studies.

60c6b3eeaa8ded0e4e7e5ea7

5) Trans palpebral tonometry :
measure IOP through upper eyelid

a) Diaton tonometry.

b) Proview tonometry.

Active space

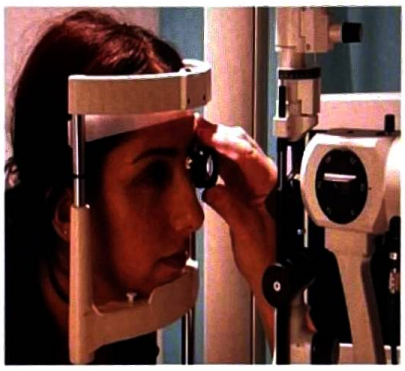
Definition of glaucoma:
Progressive, charecteristic optic changes seen (through ophthalmoscopy).
Irreversible visual field (through perimetry) changes.
IOP may/may not be raised.

Optic disc examination

00:34:17

- Via direct ophthalmoscopy.
- Slit lamp microscopy.
- Confocal scanning laser tomography (CSLO).
- Spectral domain OCT.
- Confocal scanning Laser Polarimetry (SLP).

Slit lamp uses 90 D convex lens for examination of optic disc.

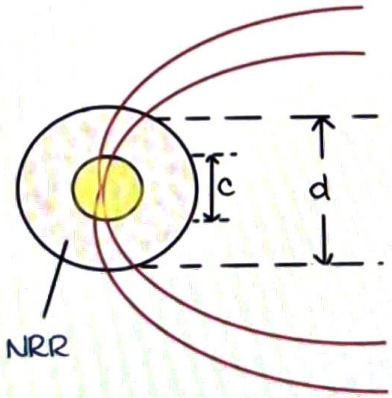


SLP is used to determine retinal nerve fibre layer thickness.

Optic disc anatomy

0:38:13

c → Optic cup (central depression which allows the vessels to travel through).
d → Optic disc.
NRR → Neuro-retinal rim (containing optic nerve fibres around the periphery of OD).
d = c + NRR.
Shape of OD = Vertically oval.
Diameter of OD = 1.5 mm / 1500 microns.
C/D ratio → Normally = 0.3

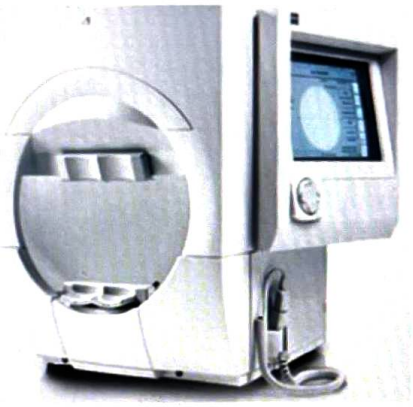


Visual field examination/ Perimetry

00:44:28

- Kinetic → moving stimuli used to measure visual field.
  Goldmann perimeter, Bjerrum tangent screen.
- Static → (Commonly used) Humphrey field analyzer (HFA → MC used), Dicon, octopus.

HFA → Bowl type screen.
Uniocular visual field is examined.

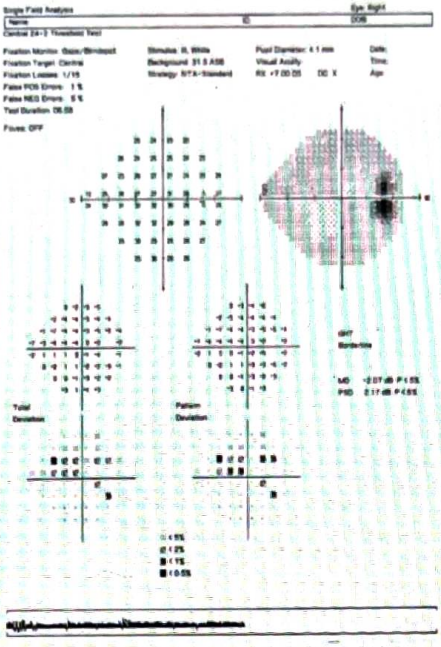


Testing patterns :

- 24 - 2 → Central 24° Visual field is being examined.
  Between 2 test points, there is a gap of 6°.
- 30 - 2 → Central 30° Visual field is being examined.
  Between 2 test points, there is a gap of 6°.
- 10 - 2.
- Full Field (FF) 120 point screening → Tests center along with periphery.

Testing strategies :

- Suprathreshold strategy → used for screening of visual field.
- Threshold strategy → used for glaucoma monitoring.
- SITA (Swedish Interactive Testing Algorithm) → MC used in HFA.
  All these strategies use white on white stimuli.
  Background of bowl and the lights (stimuli) which patient sees is white in colour.
- SWAP → uses blue on yellow stimuli ; Tests Koniocellular pathway.
- FDT → uses flickering black & white stimuli.
  useful in children, tests the magnocellular pathway (responsible for rapid motions / flickers).
- Ring → used for peripheral visual field, tests parvocellular pathway.

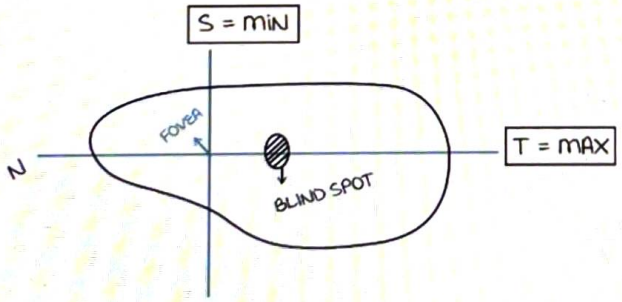


Result of HFA :

The sparsely dotted area (where the patient took more time to see the stimuli) is the abnormal part.

Normal visual field 00:58:56

Shape of normal visual field → Horizontally oval with an infero-nasal notch.



2D plot of visual field - Isoptre

Visual field is maximum at temporal region.

minimum at superior region due to cutting off by frontal bone.

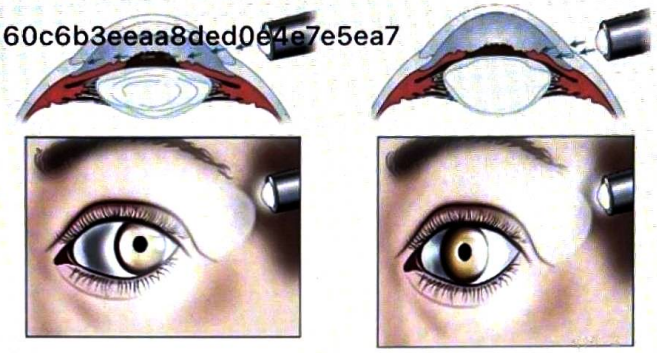
Examination of angle of anterior chamber 01:02:48

- Oblique flashlight test.
- Van Herick's method.
- Gonioscopy.
- Ultrasound biomicroscopy/UBM.

Active space

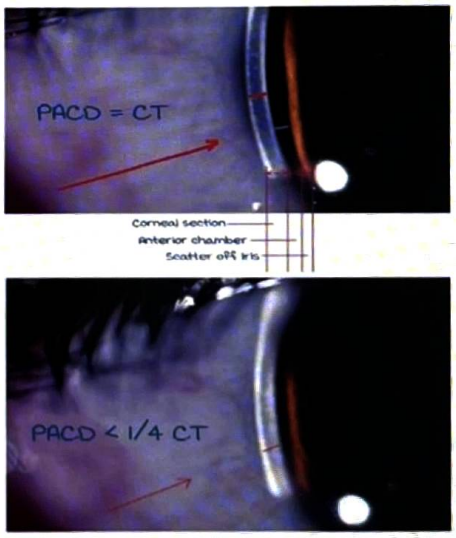
- Anterior segment → AS OCT.

Oblique flashlight test :
Light is thrown from temporal side by a pen-torch → Temporal iris is illuminated.
A. Nasal iris is also illuminated :
Deep anterior chamber → Open angle.
B. Shadow over nasal iris → Eclipse sign → Shallow anterior chamber → Angle closed.



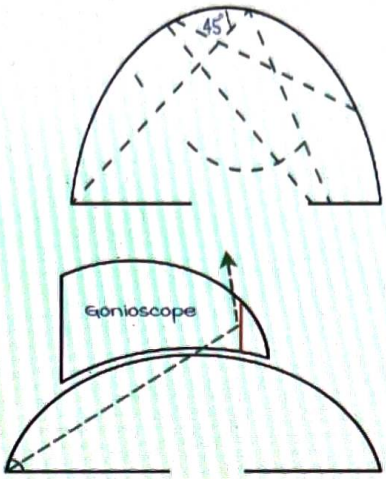
Open angle Closed angle

Van Herick's method :
On slit lamp, compare CT (Corneal Thickness) to PACD (peripheral anterior chamber depth).
PACD = CT : Angle open.
PACD <1/4 CT : Closed angle.



PACD/CT	Grade	
≥ 1	4	Wide open angle.
<1/2 to >1/4	3	Open angle → Incapable of closure
1/4	2	moderately narrow → Should be gonioscoped.
<1/4	1	very narrow angle (potentially occludable)

Gonioscopy :
Main method used to measure angle of anterior chamber.



Principle → It overcomes Total internal reflection (TIR).
Critical angle = 46°.
Gonioscope with same refractive index as cornea and mirror is placed over the cornea.
Light from anterior chamber strikes the cornea and travels through and gets reflected by the mirror in gonioscope, thus reaching our eye.

Types of gonioscopy :

- Direct → E.g Koeppe Goniolens, Barkan Goniolens.
  Done in supine position.
  Used in bed-ridden patients, anaesthetized patients, children.
- Indirect → Done through Gonioprisms.
  E.g. Zeiss four mirror, Posner four mirror, Goldmann's three mirror.
  Done in sitting position on a slit lamp.
  Used in adults.

Structures in angle of anterior chamber that can be visualised by gonioscope.
Mnemonic → Can't See This Stuff.

- Ciliary body band
- Scleral spur.
- Trabecular meshwork
- Schwalbe's line (peripheral termination of Descemets membrane)

Posterior
Anterior

As the iris moves forward, the structures that can be seen from posterior to anterior decreases. Finally when only Schwalbe's line is visible or nothing can be seen, the angle is said to be closed.

Grading of gonioscopy findings :

1. Scheie : Based on number of structures visible.
   All strictures visible → G1 → Angle open.
   3 strictures visible → G2.
   2 strictures visible → G3.
   Only Schwalbe's lines visible → G4 → Angle closed.

2. Shaffer : Based on degree of angle.
   < 10° → G1 → Angle closed.
   35°-45° → G4 → Angle open.

3. Spaeth → Based on 12 parameters.

Q. Which investigation cannot be done in a dilated pupil?
A. Examine OD.
B. Perimetry.
C. Gonioscopy.
In dilated pupil, iris muscles bunch up in periphery and obscures the view of structures visible by gonioscopy.

IOP measurement → Tonometry

- Indentation tonometry.
- Non-contact tonometry.
- Transpalpebral tonometry.
- Applanation tonometry :

1. Fixed force → Maklakov, Tonomat, Barraquer.
2. Fixed area → Goldmann's, Perkin's, Pneumatic, Draeger, Mackay marg, Tonopen , Pascal.

GLAUCOMA - OPEN ANGLE AND ANGLE CLOSURE GLAUCOMA

Primary open angle glaucoma (POAG) 00:00:12

Primary : Not due to any other disease.
It was also known as chronic simple glaucoma (not used any longer).
more common in old age > 50 years.
It is bilateral and symmetrical.
Family history is present, genes associated are :

- Optineurin.
- MYOC.
- WDR 36.

Symptoms of POAG :

- Headache (non characteristic)
- Delayed dark adaptation (Normal : 30-45 secs).
- Frequent changes of presbyopic glasses.

Signs of PAOG 00:06:34

IOP (intraocular pressure) :

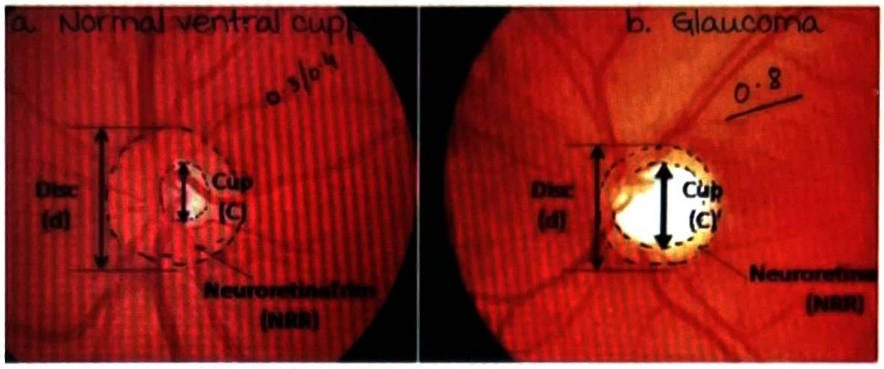
> 21 mmHg.
> 5 mmHg difference between both eyes (even if individual IOP is within normal limits).
> 8 mmHg difference in diurnal variation.

Optic disc signs :

- Loss of neuroretinal rim (NRR) : Initially, it affects inferotemporal NRR called polar notching and lastly, nasal NRR is affected.
- Vertical cupping.

The nerve fibres are damaged and are lost in the neuroretinal rim which causes the enlargement of the cup

called cupping, c/d ratio > 0.5.

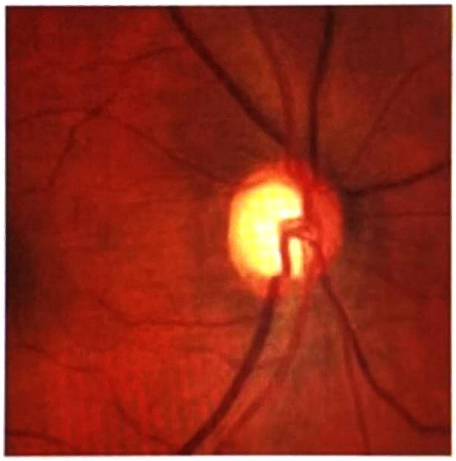


Other Signs of PAOG

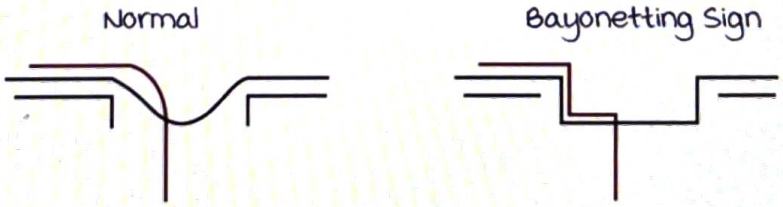
00:11:41

- Laminar dot sign : Fenestration of sclera are visible as grey white dots.

- Splinter haemorrhages on optic disc surface.
- Bayonetting sign : Double angulation of blood vessel.



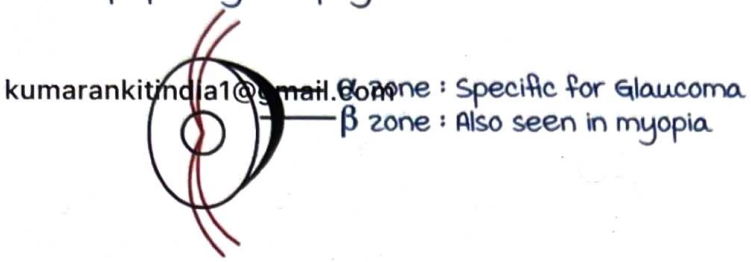
Bayonetting sign



Blood vessel bends twice to go along the walls and the floor of cup.

- Retinal nerve fibre layer wedge shaped defects : Earliest sign of glaucoma. Used for early detection of glaucoma.

- Peripapillary atrophy :



Visual field changes

00:18:04

Isoptre contraction : Earliest VF defect

↓

Baring of blind spot → Occur in Bjerrum's area

↓

Paracentral scotoma.

↓

Seidel's scotoma (sickle shaped)

↓

Arcuate/Bjerrum scotoma.

↓

Double arcuate/ring scotoma.

↓

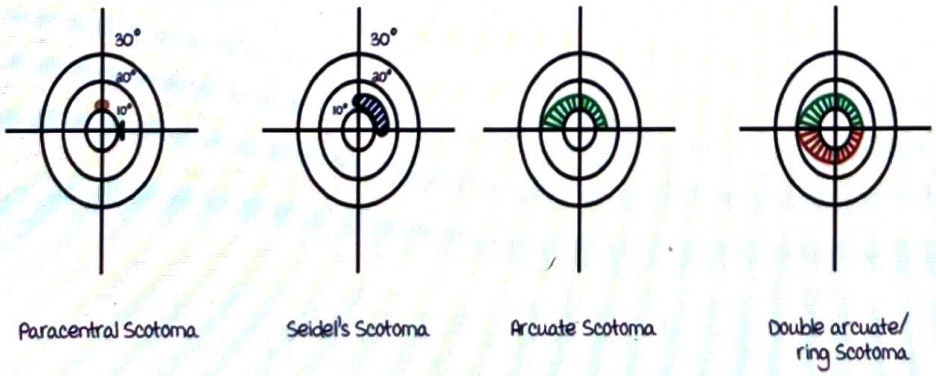
Step defects : Due to unequal contraction /loss of nerve fibres in upper & lower parts ; It can be central or Roenne's peripheral step defects.

↓

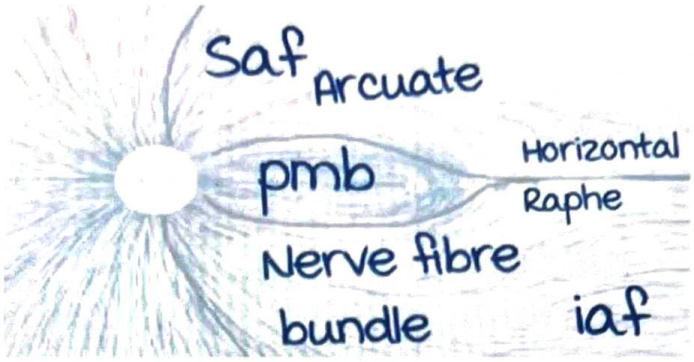
Loss of central visual field (patient presents with LOV).

↓

Loss of temporal vision (last affected).



Active space



Orientation of nerve fibre bundles :

From optic disc, fibres known as pmb (papulo-maculo bundle) arch and go through the macula.

Arcuate fibres arise from the optic disc, arch above the macula and meet at the horizontal raphe. Arcuate scotoma affects arcuate fibres.

Medical treatment of POAG : PGF2α analogues 00:29:17

Route of administration : Topical.

PGF2α analogues : DOC.

E.g. Latanoprost, Bimatoprost.

Mechanism of action : Increased uveoscleral outflow

Dosing : OD at 9 PM.

Ocular S/E : Hyperpigmentation of iris.
Hypertrichosis (growth of eyelashes).
Uveitis.
Cystoid macular edema.
Reactivation of herpetic keratitis.

Bimatoprost is FDA approved for treatment of hypotrichosis.

Medical treatment of POAG : β blockers, α agonists 00:35:20

β blockers :

E.g., Timolol (taken BD), Betaxolol.

Mechanism of action : Decreased aqueous secretion.

Ocular S/E : Blepharoconjunctivitis (eyelid + conjunctiva)
Nasolacrimal duct blockage.
Corneal anaesthesia.

Aphakic cystoid macular edema.
(only Betaxolol, hence CI in aphakia)

Contraindications : Arrhythmia.
Bronchospasm.
CHF (congestive heart failure).
Diabetes mellitus.

α agonists :

E.g. Apraclonidine, Brimonidine.

Mechanism of action : Decreased aqueous secretion and increased trabecular outflow

Ocular S/E of Apraclonidine : Follicular conjunctivitis.
Mydriasis
60c6b3eeaa8ded0e4e7e5ea7
(used in Horners syndrome)
Eyelid retraction.

Ocular S/E of brimonidine : Drowsiness.
Depression.
Apnea.
Bradycardia.

Brimonidine is a CNS depressant and is contraindicated in children.

Apraclonidine is the DOC for IOP elevation post laser treatment.

Medical treatment of POAG : Carbonic anhydrase inhibitors & new drugs

00:43:18

Carbonic anhydrase inhibitors :

E.g. Acetazolamide (not given topically), Brinzolamide, Dorzolamide (used in children).

Mechanism of action : Decreased aqueous solution.

Contraindicated in Sulfa allergy.

New drugs :

1. Nitric oxide donating prostaglandin analogues
   E.g. Latanoprostene Bunod : When administered, it is

Active space

broken into

Latanoprost → Increases uveoscleral outflow.

Butanediol → Donates NO → Relaxes trabecular meshwork → Increase trabecular outflow.

2. Rho Kinase inhibitors : E.g. Netarsudil.
   Mechanism of action : Decreases aqueous production by inhibiting norepinephrine transport and increases trabecular outflow by promoting actin-myosin contraction.
   Ocular S/E : Cornea verticillata (spindle shaped deposits in the cornea).

Surgical treatment of POAG

00:49:12

Laser trabeculoplasty (LT) : Repair trabecular meshwork

ALT
Argon LT

SLT
Selective LT
Laser applied selectively to pigmented trabecular meshwork.
Frequency doubled Nd:YAG laser (532 nm)

Mechanism of action of argon and ND:YAG laser is photocoagulation.
Increase fibrosis in one part ⟶ Contraction of that area ⟶ Pulls surrounding mesh ⟶ mesh on the other side opens up.
SLT is the treatment of choice for prophylaxis in the other eye.

Trabeculectomy :
A fistula is created between anterior chamber and subtenon's space.

mcc of failure : Fibrosis of fistula - To prevent fibrosis, use antimetabolites (mitomycin, 5-FU)

Non penetrating surgeries :

- Deep sclerectomy.
- Viscocanalostomy.

Trabecular meshwork is bypassed.

They have less chances of postoperative complications.

Surgical treatment of POAG : MIGS & Glaucoma drainage devices 00:56:30

Micro incision glaucoma surgeries (MIGS)

Canal based procedures : Increased Trabecular outflow	
Istent micro Bypass	Heparin-coated, non-ferromagnetic Titanium stent implanted ab interno insertion into Schlemm' canal) through 1.5mm corneal incision.
Suprachoroidal procedures - Increase Uvoscleral outflow	
Cypass microstent	6.35 mm long tube polymide material. Viable Alternative for patients with elevated episcleral venous pressure.
Subconjunctival procedures : Increase outflow into subtenon space	
XEN gel stent	6 mm, translimbal tube implant composed of gelatin indicated for refractory glaucoma patients with failed surgical treatment.

Advantage of MIGS :

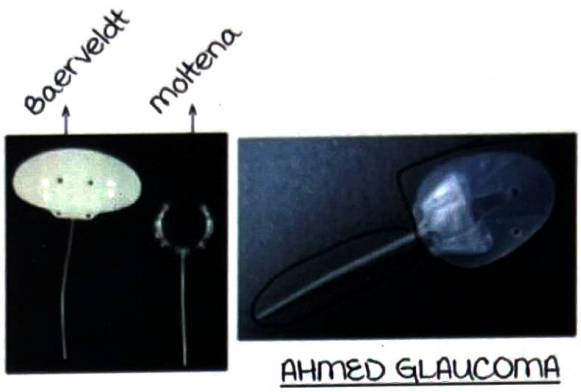
Small incision ⟶ Lesser healing time.

Less risk of infection.

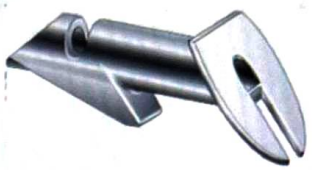
Glaucoma drainage devices/seton surgery :

Not microincision surgery,

It is done in refractory, controlled glaucoma and glaucoma with aniridia.



AHMED GLAUCOMA VALVE



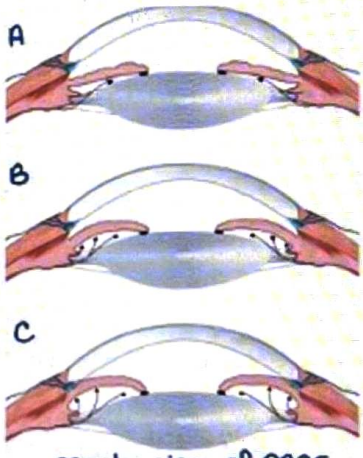
Stainless steel Glaucoma shunt

Primary angle closure glaucoma – PACG 01:01:10

It is also known as acute congestive glaucoma.
MC in young adults 20- 40 years.

Mechanism of PACG :

Relative pupillary block (A)
Apposition of pupillary margin against lens surface in a middle dilated (3.5-6 mm) pupil.
↓
Aqueous cannot flow from PC to AC.
↓
Iris bombe (BO - convex iris)
Anterior iris bowing due to increased aqueous pressure in PC.
↓
Iridotrabeculocorneal (ITC) contact
↓
Peripheral anterior synechiae (PAS)
Due to continued iris pushing anteriorly, iris gets adhered to cornea and trabecular meshwork.
↓
PACG
Central deep AC, peripheral shallow AC.



Mechanism of PACG

Risk factors & classification of PACG 01:06:03

Risk factors :

- Hypermetropia (small axial length).
- Small cornea (diameter decreased).
- Shallow anterior chamber.
- Females.
- Plateau iris (anteriorly located iris).

All these factors can be precipitated by dim light.
Hence, patient is asked not to visit cinema halls.
mid-dilatation of pupil in dim light is a precipitating factor.
Atropine is contraindicated in PACG.

Classification of PACG :
PACS (Primary angle closure suspect) :
Normal IOP.
Normal optic disc.
Apposition with ≥ 3 quadrants – ITC contact.

PAC (Primary angle closure) :
Increased IOP.
Normal optic disc.
Peripheral anterior synechiae present.

PACG (Primary angle closure glaucoma) :
Optic disc and visual field changes are present.

Clinical features of PACG 01:11:42

- Headache.
- Loss/ blurring of vision.
- Pain in the eye.
- Nausea/vomiting.
- Redness.
- Halos around light.

The halos can also be seen in :

1. Mucopurulent conjunctivitis (due to discharge).
   On washing the eye, discharge is washed away and halos disappear.
2. Cataract
3. ACG.

To differentiate between cataract and PACG : Do Fincham's test using stenopaeic slit. On rotation

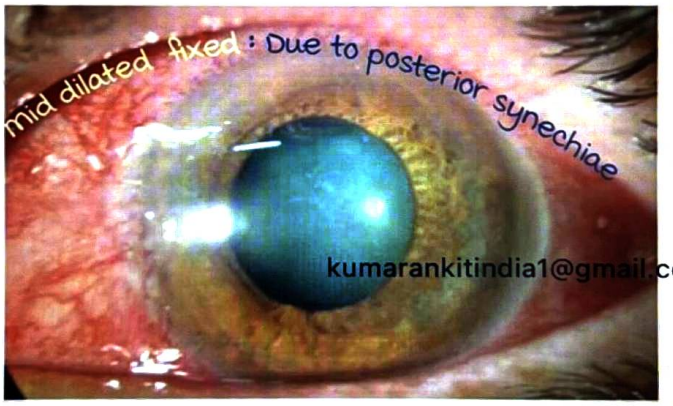
If halos appear broken → Cataract

If halos appear intact → PACG

PACG is a diagnosis of exclusion.

Signs of PACG

01:16:09



1. Increased IOP >40 mmHg : Causes severe pain in the eye.
2. On digital tonometry : Rock hard eye.
3. On torchlight test : Eclipse sign (due to shallow anterior chamber).
4. Pupil : Vertically oval.
   mid dilated
   Fixed : Due to posterior synechiae.

Slit lamp examination of PACG 01:20:08

1. Edematous cornea (coloured halos around light due to prismatic effect)/Hazy cornea (blurring of vision).
2. Iris atrophy due to pressure on iris muscles.
3. Posterior synechiae : Adhesion between pupillary margin and lens.
   Anterior synechiae are also seen.
4. Cataract known as Glaukomflecken.
   Anterior capsular opacities due to lens epithelial ischemia and necrosis.

Treatment of PACG 01:25:40

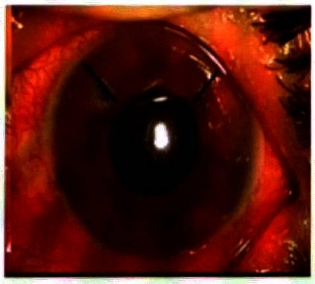
Ocular emergency.
Acute exacerbation ⟶ Reduce the IOP immediately.
Step 1 : I.V mannitol 500ml of 20% solution.
Dosage : 1-2g/Kg infused over 30 minutes.

Or

Tab. Acetazolamide 500mg stat.

Step 2 : Pilocarpine eye drops (2-4%).
(miotic : It pulls the iris and thus opens the angle of anterior chamber.)
DOC : Pilocarpine.
DOC in acute exacerbation : I.V mannitol.

Step 3 : Treatment of choice : Laser iridotomy (creation of hole in the iris).
It is done between 11 o'clock and 1 o'clock position (which is normally covered by eyelid, to avoid diplopia).
Nd:YAG laser is used.



GLAUCOMA: SECONDARY GLAUCOMA(S), CONGENITAL GLAUCOMA

Secondary glaucoma

00:00:10

Lens induced Glaucoma :

a. Phacolytic glaucoma :

- Caused by Morgagnian cataract.
- Secondary open angle glaucoma.

b. Phacomorphic glaucoma :

- Caused by Intumescent cataract (The swollen lens touches the pupil).
- Secondary angle closure glaucoma.

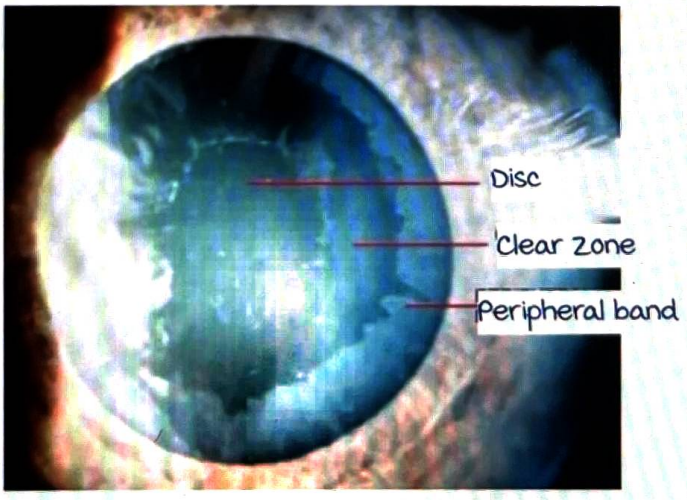
c. Phacotopic glaucoma :

- Occurs in case of microspherophakia.
- Secondary angle closure glaucoma.

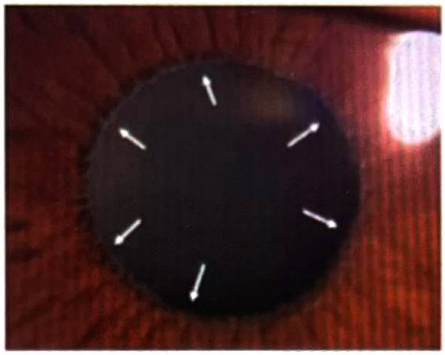
Pseudo – exfoliation glaucoma :

- Secondary open angle glaucoma.
- Occurs due to LOXL I gene mutation.
- Clinical features :

  a. Target sign : On lens.



  b. Fnocks : Whitish deposits on pupillary margin.



c. Poor mydriasis.

d. Sampaolesi line : Deposition anterior to Schwalbe's line.

Pigmentary glaucoma :

- Secondary open angle glaucoma.
- Most common in Myopes who are young males.

Clinical features	Deposition of pigment on :
a. Krukenberg spindle	Corneal endothelium.
b. Zentamyer line	Posterior surface of lens near insertion of zonules. Characteristic, diagnostic feature of pigmentary glaucoma.
c. Sampaolesi line	Deposition anterior to Schwalbe's line.

Secondary glaucoma: Steroid induced, Malignant 00:09:11

Steroid induced glaucoma :

- Due to increased production and decreased destruction of extracellular matrix of Trabecular meshwork.
- Leads to Secondary open angle glaucoma.
- Steroid responders :

High	moderate	Non
>31 mm Hg IOP	25-31 mm Hg IOP	<20 mm Hg IOP
or	or	or

Active space

>15 mm Hg increase in IOP	6–15 mm Hg increase in IOP	<6 mm Hg increase in IOP

- Treatment : Beta blockers or Laser trabeculoplasty.

Malignant glaucoma :

- Also known as Ciliary block glaucoma /Aqueous misdirection syndrome /Inverse glaucoma.
- The cause is incisional or laser surgery.
- mechanism : Anterior rotation of ciliary processes leading to Cilio-lenticular block.

↓

Posterior flow of the aqueous.

↓

Iris-lens diaphragm gets pushed forward.

↓

Secondary angle closure glaucoma (without pupillary block).

- Treatment : Atropine (Cycloplegic).

Relaxation of ciliary muscles.

↓

Stretching of zonules.

↓

Pulls the lens back

Childhood glaucoma

00:19:20

Types :	Onset	% of total cases
Newborn	At birth	40
Infantile	< 3 years	55
Juvenile	3 – 16 years	5

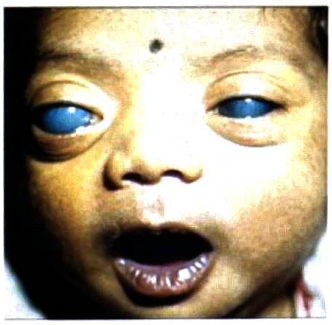
Newborn and infantile glaucoma are together known as Primary congenital glaucoma.

Primary congenital glaucoma :

- Onset < 3 years of age.
- mechanism : Trabeculo-dysgenesis (Open angle glaucoma).

- Symptoms – Triad of : Blepharospasm.
  Photophobia (earliest).
  Lacrimation.
- Signs :
  1. Earliest : Hazy cornea.
  2. Haab's striae : Horizontal or concentric breaks in Descemet's membrane.

  3. Buphthalmos :
  Corneal diameter > 13 mm
  (normal ~ 10.5 mm).
  Blue sclera : Scleral thinning due to stretching of Sclera.
  Deeper anterior chamber.
  Myopia.
  Blue eyes – Congenital glaucoma



  4. Gonioscopic features :
     a. Anterior iris insertion.
     b. Barkan membrane.
     C. Loch-Ness monster phenomenon.

- Treatment :
  1. Treatment of choice : Trabeculectomy + Trabeculotomy
  2. If in Congenital glaucoma, patient presents with clear cornea → Goniotomy.

Secondary congenital glaucoma 00:31:29

It is also known as Developmental glaucoma.
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Mechanism : Irido-corneo-trabeculo dysgenesis.

Causes :
a. Sturge Weber syndrome.
b. Aniridia (75% cases causes glaucoma).
c. Axenfeld Reiger syndrome.
d. Peters anomaly.

Active space

OPTICS : TESTS FOR VISION AND OPTICS OF EYE

Introduction to optics 00:00:13

If a patient comes to the OPD :
The vision of the patient is recorded.
If the vision is normal, the optical system is assessed.
If the recorded vision is abnormal, the eye is checked for refractory errors.
The refractory error is corrected using dark room procedures.

Visual acuity 00:01:35

Components :

1. Minimum visible.
2. Minimum resolvable :
   Separation between two points such that they are visible separately.
   Minimum angle of resolution : 30-60 seconds of an arc.
3. Minimum discriminable :
   Offset 3-5 seconds of an arc known as hyperacuity.
   E.g : Vernier acuity.
   Stereo acuity.

Tests for vision : Snellen chart 00:04:50

Tests for vision :
Acuity at far point : Far vision.
Acuity at near point : Near vision.

Snellen's chart

Far point :
Snellen's chart :
7 rows of letters are seen.
The topmost letter is the largest & going down the letters become smaller.
A standard snellen chart has a fixed number written along

the left side of each row of letters.
The numerator denotes that the testing needs to be done at the distance of 6 metres.
The denominator denotes the distance from which each specific letter can be read clearly by a normal person.

The right side of the chart shows the converted values from metres to feet.

method :
Vision cannot be tested binocularly & hence the patient needs to cover one eye & distance needs to be 6 m.

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Patient stands/sits at 6 m and reads letters from top to bottom

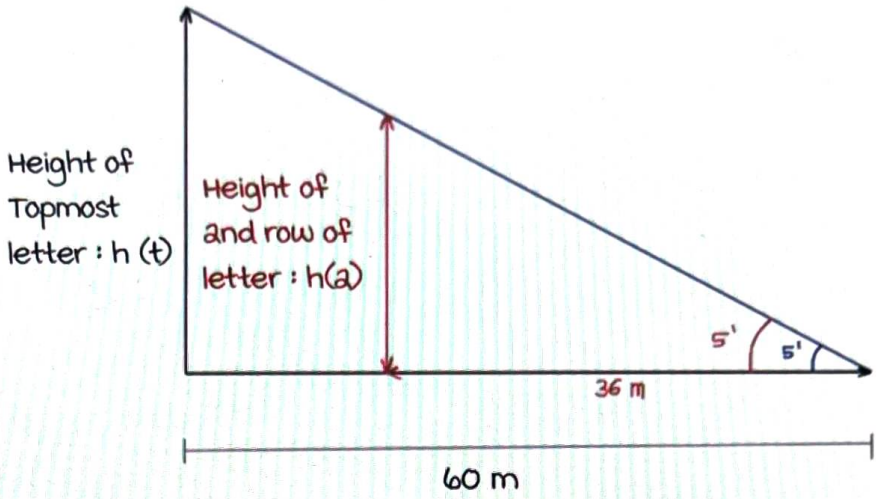
- If the patient reads all the rows uptll last (7th) row (6m) → 6/6 : Normal vision
- If the patient reads only till the 4th row (18m) → 6/18 : Decreased vision

Order of decreasing vision from the normal :
6/6 > 6/9 > 6/12 > 6/18 > 6/24 > 6/36 > 6/60 > 5/60 >4/60 > 3/60 >2/60 > 1/60 > Finger counting close to face (FCCF) > hand movement perception/HM > perception of light/ PL.

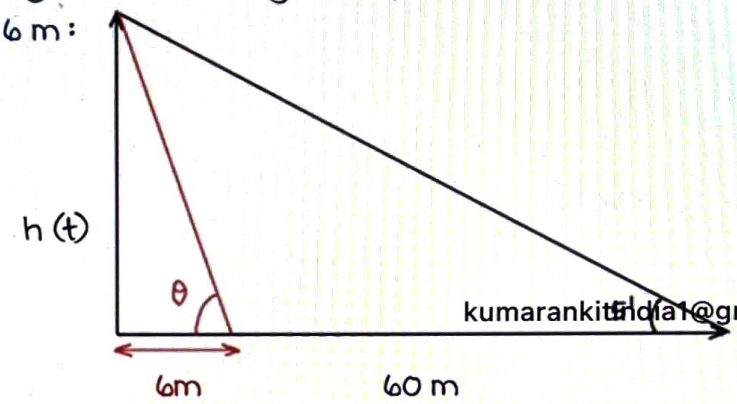
Normal visual acuity : 6/5.
Best visual acuity : 6/3.
Least vision recordable on snellen chart : 1/60.
Distance of examination : 6 metres.

Principle of snellen chart 00:17:18

Each letter (optotype) subtends an angle of 5 minutes of arc at the nodal point of eye, when viewed from its respective distance.



The angle subtended by the topmost letter when viewed from 6 m :



From the diagram, it is evident that the angle increases.

$$\text{Angle subtended} = \frac{\text{Distance of respective letter} \times 5}{\text{Distance of testing}}$$

$$= \frac{60}{6} \times 5 = 50 \text{ minutes of arc.}$$

Other tests for vision in adults

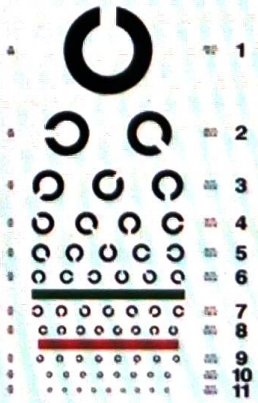
00:24:45

1. Landolt's C-ring test :

Also known as detection visual acuity test.

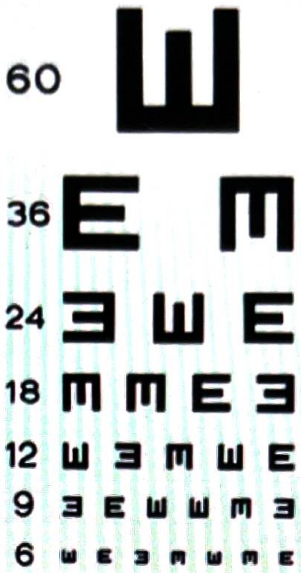
Direction of the cut needs identification in the test.

Used in people who cannot read alphabets/illiterates.

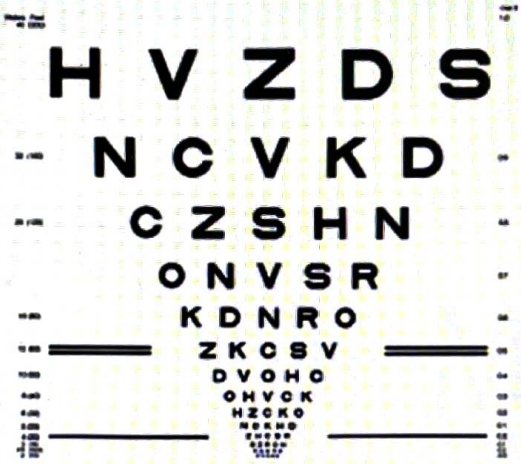


Active space

2. Tumbling E chart :
Direction of E needs identification.
Used in illiterates.



3. Bailey – Love logmar chart :
Each row contains 5 letters.
Gives more accurate visual acuity.
Designed on logarithm basis.
Not done in India.



Tests for vision in children

00:28:30

Pre-school children : Tests based on matching letters.
1. Sheridan-Gardiner HOTV test.
2. STYCAR (Screening Test For Young Children & Retards).

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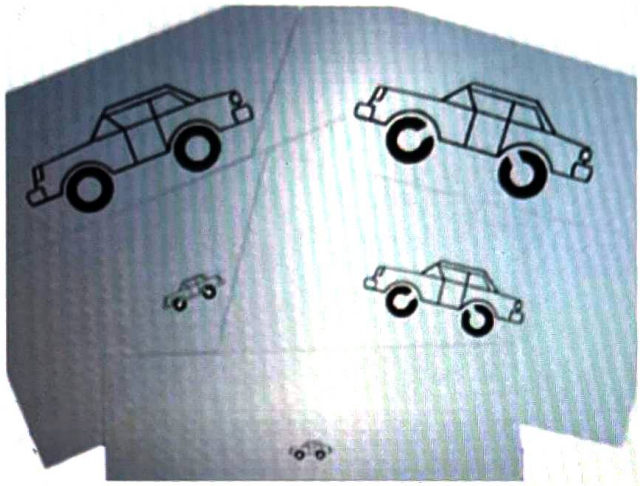
Active space

Tests based on matching pictures.

1. Broken wheel test : Similar to Landolt's C-ring test.



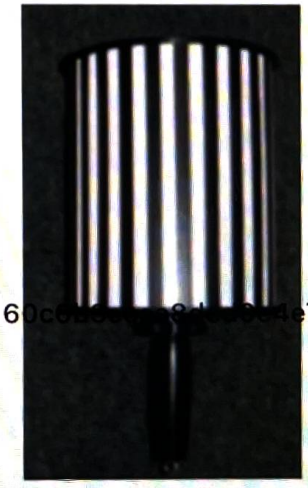
2. Kay picture test.

3. Allen pre-school test.

Infants :

1. Opto Kinetic Nystagmus test :

OKN drum & Catford drum can be used.
OKN drum has vertical lines.
used for resolution acuity.
Catford drum has different size dots.
used for detection acuity.



OKN drum

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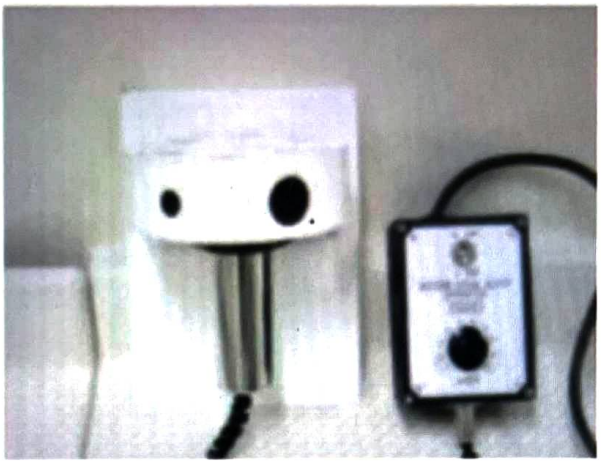
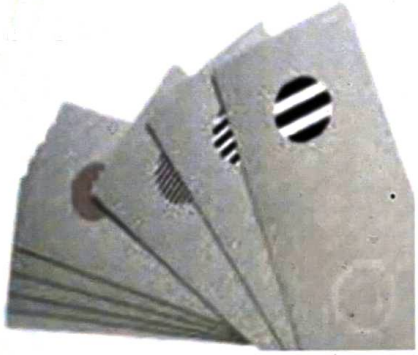
We check what size of the line/dot the infant is able to fixate on & follow.



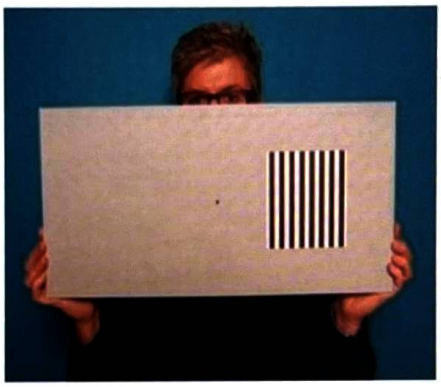
Catford drum

2. Preferential looking tests / PLT :

The infant will prefer to look at a striped pattern rather than a plain pattern.



Keeler gratings



Teller acuity cards

3. Indirect Assessment :

Blink reflex (part of Cbme curriculum) :

Presence since birth.

Closure of eyes in response to illumination.

Menace reflex :

Develops around 5 months of age.

Infant closes eyes/moves the face on approach (direct) of an object towards his/her eyes.

Tests for near vision and contrast sensitivity 00:38:37

Tests for near vision :

1. Jaeger's chart : Visual acuity is numbered from J 1 to J 7.
2. Snellen's near vision chart : Visual acuity is numbered

Active space

from N 6 (best) to N 36 (worst).

Normally a patient should be able to read the last line N 6 at a distance of 25 cm.

In presbyopia , patient would read lesser.

Tests for contrast sensitivity :

Pelli-Robson chart : MC used clinically.
Topmost letters on the chart are the darkest whereas the bottom line has letters of the lightest colour.

Pelli Robson chart

In cataract, the contrast is lost before vision.
Tests used :
C : Cambridge low contrast grating.
R : Regan chart.
A : Arden gratings.
P : Pelli-Robson chart

Normal optical system of the eye 00:42:23

Normal optical system converges the light rays and the refraction from 2 optical surfaces (lens and cornea).

Gullstrand's schematic eye :

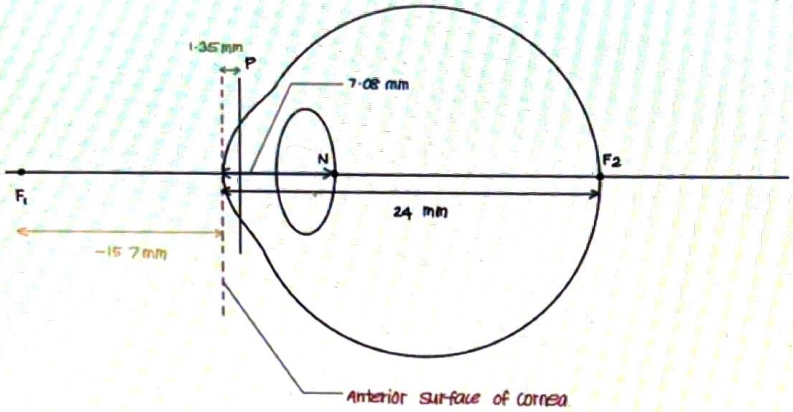
Power of Cornea : + 43D to + 44D.
The cornea has 2 surfaces : Anterior surface : + 48.3 D.
Posterior surface : − 5.88 D.
The refrative index/ RI of cornea : 1.376.
The refrative index of aqueous : 1.336.

Light is going from posterior surface (denser) into the aqueous (rarer medium) causing minimal divergence.

Power of lens : + 16 to +19 D.

Maximum power in eye : Anterior surface of cornea : +48.83 D.

Listing's reduced eye :



P : Principal point.

N : Nodal point.

Fl, F2 : Focal points.

The principle point is located 1.35 mm posterior to the anterior surface of cornea.

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The nodal point is located 7.08 mm posterior to the anterior surface of Cornea.

F2 is located 24 mm posterior to the anterior surface of cornea.

Fl is located 15.7 mm anterior to the anterior surface of cornea.

Refractory power depends on :

1. Radii of curvature.
2. Difference in refractive index between various structures.
3. Axial length of eyeball.

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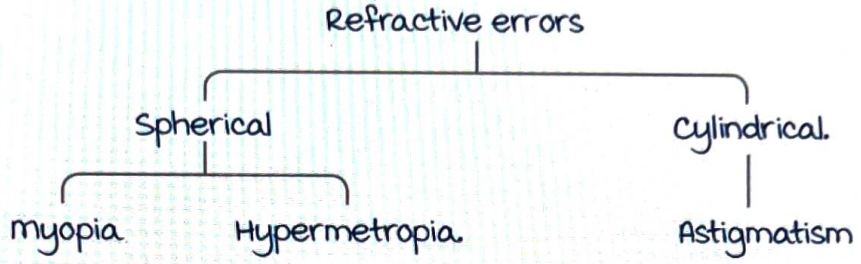
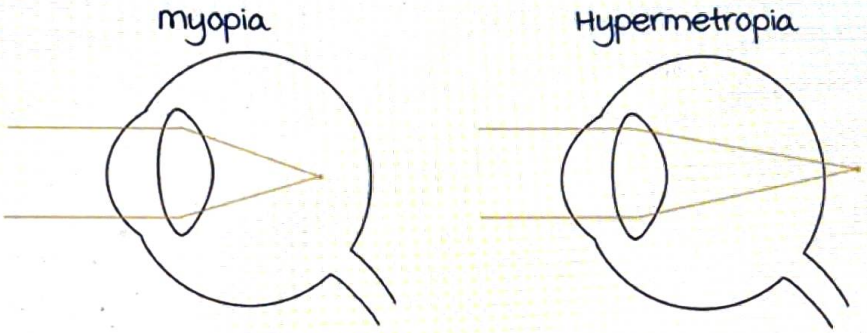
OPTICS - MYOPIA AND HYPERMETROPIA

Introduction to Refractory errors 00:00:26



Myopia vs Hypermetropia: Mechanisms 00:03:32



	Myopia	Hypermetropia
Power of the eye	More	Less
Image formed	Infront of the Retina	Behind the Retina
Mechanism /Causes	1. Axial myopia : • Axial length of eye : Increased • Imm increase in axial length = 3D Myopia. • Buphthalmos 2. Curvatural myopia : • Curvature of cornea : Increased • Radius of curvature : Decreased • Keratoconus 3. Index myopia : • Refractive index of lens : Increased • Nuclear cataract	• Axial length of eye : Decreased • Curvature of cornea : Decreased • Radius of curvature : Increased • Refractive index of lens : Decreased

	4. Positional myopia : • Position of lens : Anterior • P = 1/f • Weil-marchesani syndrome	• Position of lens : Posterior

Myopia vs Hypermetropia: Symptoms and Signs

00:16:06

	Myopia	Hypermetropia
Symptoms	1. Loss of far vision or Short/Near sightedness 2. Floaters : Due to vitreous liquefaction	1. Loss of near vision or Long/Far sightedness 2. Asthenopia (Tired eyes)
Signs	1. Eyeball : Large Leads to decrease in scleral rigidity 2. Anterior chamber : Deeper 3. Leads to Exophoria : • Far point comes closer. • Patient does not need to accommodate for near vision. • No accommodation = No convergence • The patient develops convergence insufficiency and presents as Exophoria.	1. Eyeball : Small 2. Anterior chamber : Shallower (Hypermetropia is a risk factor for Angle closure glaucoma) 3. Leads to Esotropia

Myopia vs Hypermetropia: Fundus signs

00:24:48

Fundus is a view of all the following structures visible in the posterior segment :

- Vitreous
- Retina
- Choroid

} Transparent

Active space

Fundus signs in Hypermetropia :

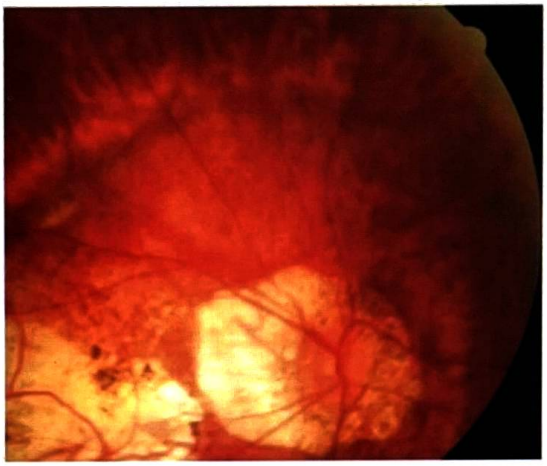
a. Pseudo-papillitis

b. Shot silk fundus

Fundus signs in Myopia :

- They are seen only in cases of Pathological / Degenerative myopia : If myopia > 6D or axial length > 26 mm.

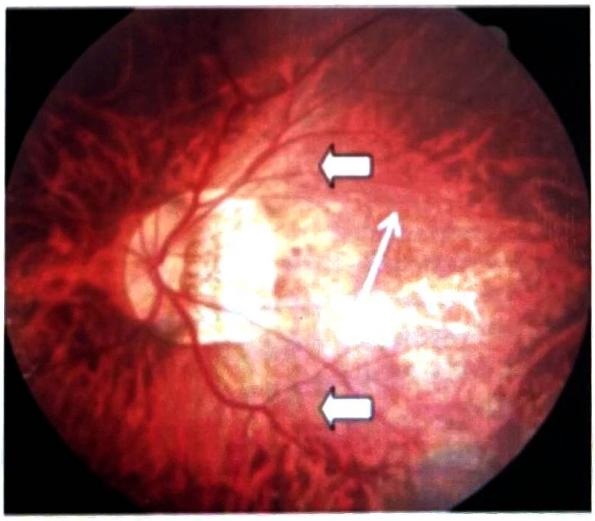
a. Temporal myopic crescent : Zone of Chorioretinal atrophy around the optic disc on temporal side.



b. Tesselated /Tigroid appearance of fundus : Occurs due to diffuse attenuation of retinal pigment epithelium.

c. Supertraction : Leads to tilted optic disc.

d. Lacquer cracks : Fine breaks in the Bruch's membrane.



The above image shows :

1. Lacquer cracks
2. Temporal myopic crescent
3. Tilted optic disc

e. Foster fuch spots : Areas of subretinal haemorrhage at the macula.

f. Posterior staphyloma : Bulging of sclera.

Different types of Staphyloma :

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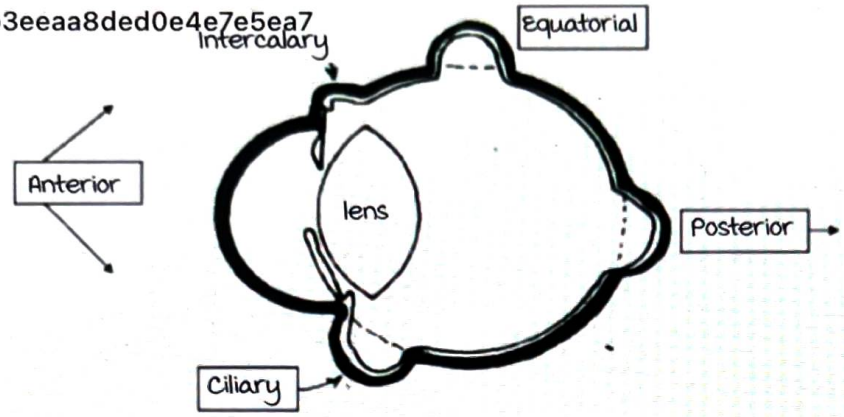


- Intercalary : Bulging of limbus lined by root of iris.
- Ciliary : Bulging of sclera lined by ciliary body.
- Equatorial : Bulging of sclera lined by choroid.
- Anterior : Corneal bulging.

g. Vitreous hemorrhage

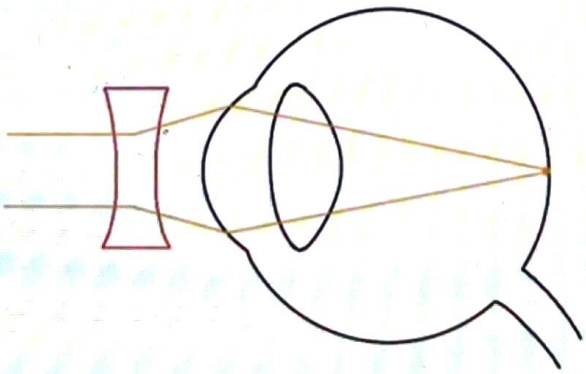
h. Rhegmatogenous retinal detachment

Treatment of Myopia and Hypermetropia: Spectacles and Surgeries

00:45:15

Treatment of myopia : Spectacles

1. Spherical /Concave [-] or Diverging lens



Treatment of Hypermetropia : Spectacles

- Spherical /Convex [+] or Converging lens

Active space

2. Surgeries :

For Hypermetropia :

a. Conductive Keratoplasty 60c6b3edaa8dd0e4e7e5ea7

b. Thermal laser Keratoplasty

c. Hyperopic LASIK

For Myopia :

a. Radial Keratotomy :

To flatten the curvature of the cornea.

b. Laser surgeries :

1. PRK : Photorefractive Keratectomy (-2D TO -6D)
2. LASIK : Laser assisted in situ Keratomileusis

- Excimer laser : Argon Fluoride (193 nm)
- mechanism : Photoablate – flattening of cornea.
- Steps :

Use a microkeratone to create a nasally hinged epithelial flap

↓

Ablate the corneal stroma

(Roughly 1D = 10μm ablation)

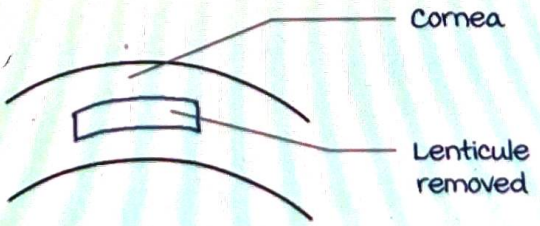
↓

Residual stromal thickness ≥ 250m

- Contraindications to LASIK :
  - Age less than 18 years.
  - Spectacle power is not constant for past 1 year.
  - Corneal thickness < 450μm

3. Latest surgeries :

- Small incision Lenticule extraction (SMILE) or Refractive Lenticule extraction (ReLEx)



- On the removal of Lenticule, the thickness and curvature is decreased.
- Laser used : Nd : Glass laser
- Flapless and bladeless surgery

c. Fucala's operation :
Extraction of clear lens and implantation of IOL.

d. Phakic refractive lenses (PRL) :
Implant an IOL – ICL (Implantable Collamer Lens), over the normal crystalline lens.

3. Contact lens :
Preferred in High myopia.

OPTICS : ASTIGMATISM, TESTS FOR REFRACTION - RETINOSCOPY, SPECTACLE PRESCRIPTION

Astigmatism

00:00:12

Different refractory power in both principal meridian/axis of eye.

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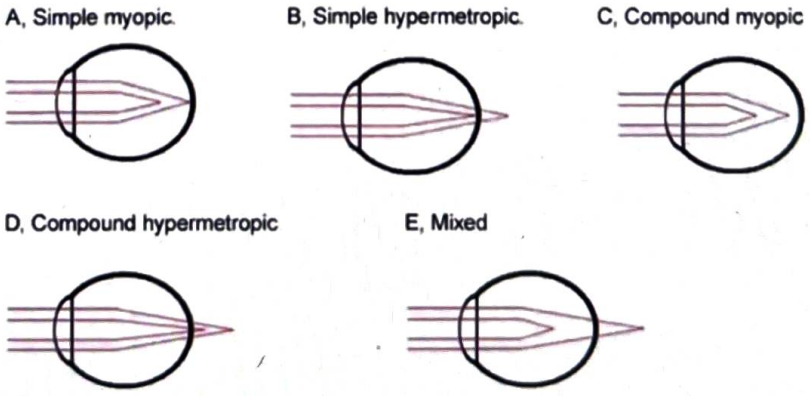
Classification

Regular astigmatism
- When both principal meridian are at 90° to each other

Irregular astigmatism
- When both principal meridian are not at 90° to each other

With the rule.	Against the rule.	Oblique
Vertical meridian is more curved.	Horizontal meridian is more curved.	Both principal meridian are at 90° to each other but not vertical and horizontal.

Refractive types of regular Astigmatism:



The most common type of astigmatism → Compound myopic.

Active space

Astigmatism: Symptoms and Treatment 00:08:17

Symptoms of Astigmatism :

- Asthenopia.
- The patient complains of letters running together at the edges and appearing as if they are merged.
- High astigmatism → Patient can show tilting of the head.

Treatment of astigmatism :

- Spectacles → Cylindrical (concave or convex based on the refractive type of astigmatism).
- Surgery :
  1. Astigmatic LASIK.
  2. Limbal relaxing incisions (LRI).
  3. Ruiz procedure for Post Keratoplasty astigmatism.
- Contact lenses : TORIC

Miscellaneous refractory conditions 00:12:18

Aniseikonia → Distortion of vision.
Different size and /or shape of image between both eyes.

Anisometropia :
Different refractive power between both eyes.
Significant only if the difference between
both the eyes > 2.5D.
E. g : If Right eye = +2D and Left eye = +6D.
1 D = 2% magnification.
Hence, Right eye → 4%, Left eye → 12% magnification.
If the difference in image size is > 5%, brain cannot fuse the images and diplopia occurs.

Emmetropia → Refractory power is normal.

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Aphakia → Absence of lens.
Causes Hypermetropia.
Pupil → Jet black
Purkinje images → Normally 4

Active space

In Aphakia a

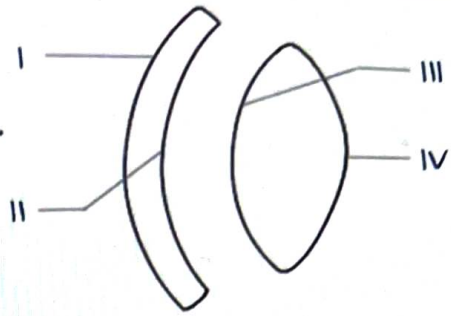
I → Anterior surface of cornea.

II → Posterior surface of cornea.

III → Anterior surface of lens.

IV → Posterior surface of lens.

(concave → Inverted image)



Treatment :

- Treatment of choice → IOL Implantation.
- Contact lens.
- Spectacles → Convex (+10 D to +13 D)

  Side effects of a high powered spectacle :

  a. Magnification is increased to ~ 25%
  b. Pin-cushion defect (Spherical aberration).
  c. Jack in box phenomenon (Prismatic aberration) or Roving ring scotoma.

Refraction and retinoscopy

00:24:20

Procedure of determining and correcting refractive errors known as refraction

- Objective refraction
  - Retinoscopy
    - Manual
      - Reflecting mirror retinoscope
      - Priestley-Smith's retinoscope
      - Self illuminated streak retinoscope
    - Automated refractometers (uses Scheiner disc principle)
- Subjective refraction
  - Adjustment of refraction
    - Trial and error method
    - Fogging technique
  - Refinement of refraction
    - Cylinder
      - Jackson's cross-cylinder test
      - Astigmatic fan test
    - Sphere
      - Duochrome test
      - Pinhole testing
  - Binocular balancing
    - Duochrome test with fogging
    - Prism dissociation method

Reflecting mirror retinoscope | Priestley-Smith's retinoscope | Self illuminated Streak retinoscope

Retinoscopy/skiascopy/shadow test :

Distance of examination → 1 metre.

Retinoscope used → Self illuminating streak retinoscope.

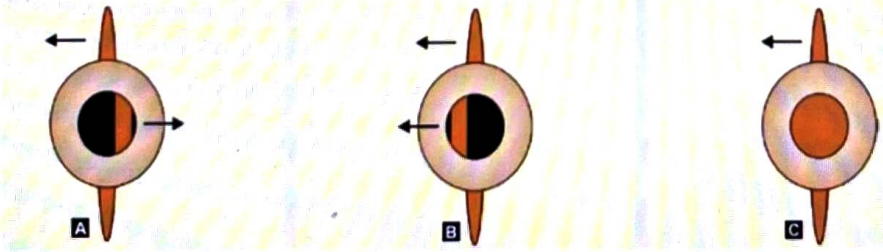
Method :

- The retinoscope is held in front of the examiner's eye.
- The patient is asked to focus at a distant object, then the light is shown in the patients eye.
- A streak of light enters the patient's eye and falls onto the patients fundus (red reflex/shadow).
- Movement of fundal red streak is noted in comparison to the movement of retinoscope to note the refractory status of the eye :

a. With the movement → < 1 D myopia or
Emmetropia or
Hypermetropia

b. Against the movement → > 1 D myopia

c. No movement → 1 D of myopia.



Against the motion With the motion Neutral

Correction factors in retinoscopy findings :

- C1 → Correction for distance of examination (d)
  $C_1 = 1/d$
  Example : 1m, $C_1 = 1/1 = 1$

- C2 → Tonus allowance for cycloplegia.

Active space

Accomodation is abolished by the use of cycloplegics

Atropine : 1

Homatropine : 0.50

Cyclopentolate : 0.75

Cycloplegic DOC in children → 1% Atropine eye ointment.

Cycloplegic DOC in adults → 0.5% or 1% Cyclopentolate eye ointment.

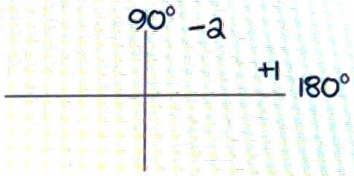
Prescription of spectacles

00:36:42

Given retinoscopy findings (RF) :

Distance of examination (d) = 1 m

Cycloplegic used → Atropine



Steps to prescribe spectacles :

1. Calculate the corrected RF

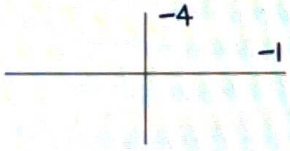
Corrected RF = RF − Cl − Ca

In this example,

- Cl = 1/d = 1/1 = 1
- Ca for Atropine = 1
- Given RF : −2 (vertical axis) and +1 (horizontal axis)
- Corrected RF = − 2 − 1 − 1 = − 4
- Corrected RF = + 1 − 1 − 1 = −1

2. Prescribe spectacles from the corrected RF :

- Choose any axis as spherical and mark other axis as X



Suppose − 1 is chosen as spherical,

S = − 1 D sphere,

X = − 4

- Calculate cylinder (c) as (X−S) = − 4 − (−1) = −3

So, Cylinder = − 3 D

- Choose axis of cylinder 90° to X or same as spherical

So, axis of cylinder = 180°

Refractory error = −1 D s / − 3 D c at 180°

If − 4 is chosen as spherical, S= − 4 D sphere, X = − 1 D

Active space

Cylinder = X-S = -1 - (-4) = + 3 D
Axis of cylinder 90°
Refractory error = - 4D S / + 3 D C at 90°

Reading a spectacle prescription 00:45:30

If the patient has been prescribed :

- -2D Spherical → Myopia
- -1D cylindrical → Simple myopic astigmatism
- -1D S / -2D C → Compound myopic astigmatism
- +0.75D S → Hypermetropia.
- +1.5D C → Simple hypermetropic astigmatism
- +1D S / +0.75D C → Compound hypermetropic astigmatism
- -4D S /+3D C or +4D S /-3D C → Not mixed astigmatism because the power of sphere is more than the power of the cylinder.
- -1D S /+3D C or +1D S /-3D C → mixed astigmatism (Power of the cylinder is more than the power of the sphere)

Rules of mixed astigmatism :

- Cylinder and sphere of opposite power .
- C power > S power.

- -1D C at 180° → Simple myopic astigmatism with the rule.

	180°	90°
With	Concave [-]	Convex [+]
Against	Convex [+]	Concave [-]

- -2.5D S/ -1D C at 180° → Compound myopic astigmatism against the rule

Simple transposition :
New sphere = Old S + Old C
New Cylinder = Opposite sign of old C
New axis = Perpendicular to old axis (180° becomes 90° and 90° becomes 180°)

Active space

- -4D S / +3D C at 90°:

New S = -4 + 3 = - 1

New C = -3

New axis = 180°

Transposed number → -1D S / -3D C at 180° is Compound myopic astigmatism with the rule

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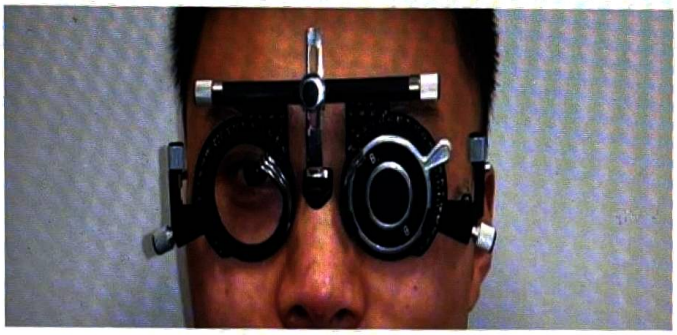
Subjective refraction

01:02:04

Adjustment of refraction :

By Trial and error method



Trial frame

Left eye is occluded

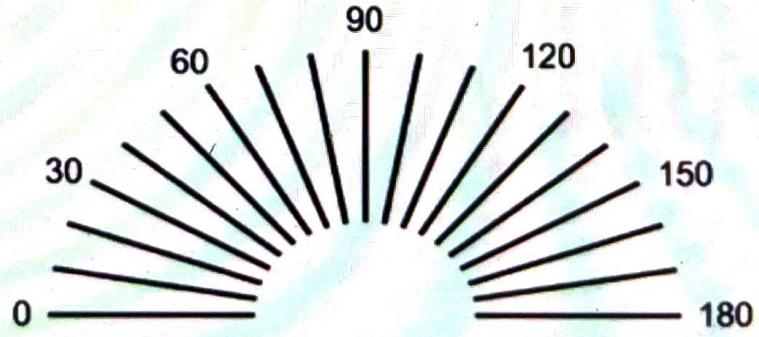
Refinement of Refraction :

1. Cylinder :

a. Jackson's cross-cylinder test :

- - 0.5D sphere with + 1.0D cylinder → Spherocylindrical lens.

b. Astigmatic fan test



- If all the lines are equal in intensity : No problem of axis.
- If a particular is more clearly visible : Indicates the requirement of refinement of axis.

2. Sphere :
   a. Duochrome test :

- If letters on green background are more clear → Residual hypermetropia.
- If letters on red background are more clear → Residual myopia.

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   b. Pinhole test :

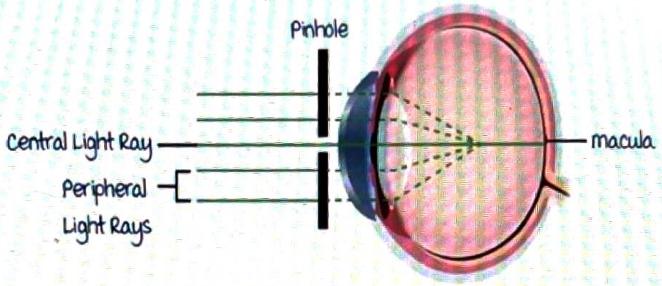
- Size of the pinhole : 1 mm

Pinhole in front of the patient.
- vision improves. → Residual refractory error (upto 4D)
- vision decreases. → Macular pathology.

Why does the vision decrease on pinhole test if there is macular pathology?
Normally, the central light ray doesn't need any convergence. Convergence actually takes place for peripheral light rays. if there is myopia, the peripheral light rays converge in front of the retina.

Active space

If a pinhole is placed in front of this patient, then pinhole covers all the peripheral light rays and allows only the central light ray (doesn't require any refraction)
So the vision improves in cases of residual refractory errors.
If there is any pathology at the macula, the vision of the patient decreases on pinhole as the peripheral vision will not be able to support the problem of central vision.



- Use :
  (1) Can refine any spherical error.
  (2) Diagnosis of refractory error up to 4D.
  (3) Diagnosis of macular disease.
  (4) Used as Low vision aids (LVA) → multiple pinholes.
  (5) 2 point discrimination test → 2 pinholes.

Binocular balancing :

1. Duochrome test with fogging.
2. Prism dissociation method.

Emmetropization, Accommodation and Amaurosis

01:14:45

Emmetropization :

- New born has +2D to + 3D Hypermetropia (as eye is small).
- Achieves Emmetropia by 7 years of age.

Accommodation :

- Maximum in children.
- Least in old age.

Amaurosis :

- Complete loss of vision (unilateral / bilateral) without any findings/signs.
- Causes of bilateral Amaurosis :

  1. Acute Nephritis
  2. Uremia → Bilateral Amaurosis

  ] Toxins act on higher visual centres.

  Pupil → Light reaction normal.

Convex lens vs. Concave lens 01:19:14

Convex lens	Concave lens
Magnification	Minimification
If you look through the covex lens and move the lens, the image moves in opposite direction.	Image moves in the same direction.
• Hypermetropia. • Aphakia. • Prebyopia. • Accomodative esotropia. • Ophthalmoscopes.	• Myopia. • Used as Hruby lens (-58.6 D) to visualise the fundus.

RETINA : ANATOMY AND INVESTIGATIONS

Introduction to retina

00:01:27

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Colour of the Retina → Transparent.

Layers of retina → 10

Gross anatomy :

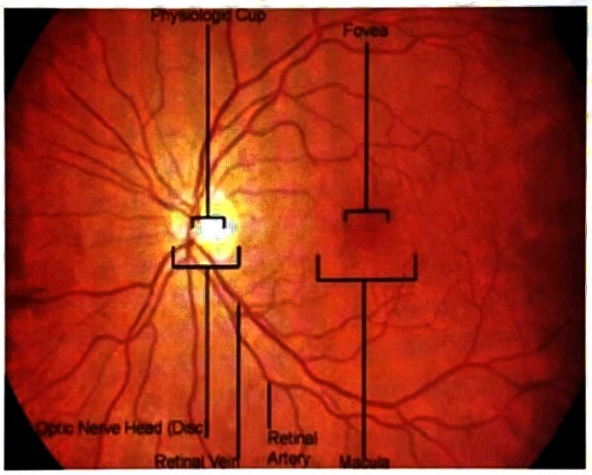
Optic disc

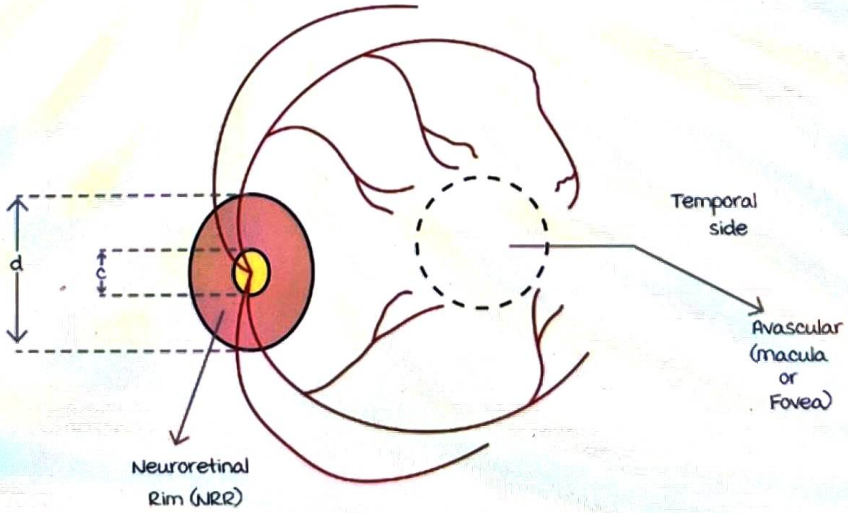
Macula → Central vision.

Ora serrata → Anterior termination of retina.

Fundus → A view of vitreous transparent retina

Choroid → Red





c/d ratio = 0.3

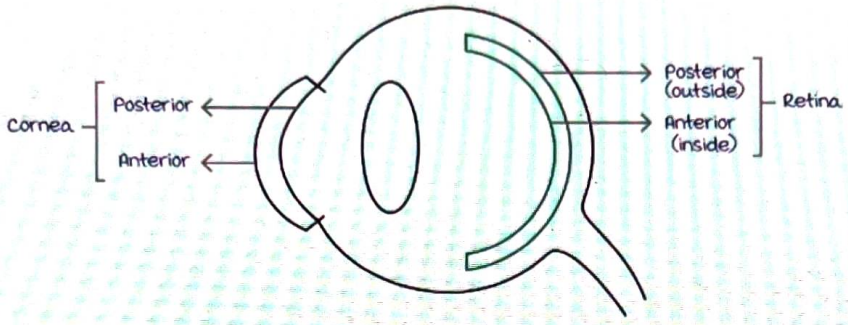
Optic disc diameter = 1.5 mm

Macula (relatively avascular central part) → Temporal to optic disc by 2 disc diameters.

Active space

Layers of retina

00:07:39



There are 10 layers in retina.

Mnemonic → ING I_a O_a eP_a or ING (IOP)a

Internal limiting membrane (ILM)

- Inner most layer.
- True basement membrane.
- Formed by footplates of muller cells.

Nerve fibre layer (NFL)

- Forms Optic nerve (Collection of axons of ganglion cells).
- 3rd order neurons.

Ganglion cell layer (GCL)

- Ganglion cells.
- 3rd order neurons.

Inner plexiform layer (IPL)

- Synapse between Ganglion cells and Bipolar cells.

Inner nuclear layer (INL)

- Bipolar cells.
- 2nd order neurons.

Outer plexiform layer (OPL)

- Synapse between Bipolar cells and Photoreceptors.

Outer nuclear layer (ONL)

External limiting membrane (ELM) → Not a true basement membrane.

Photoreceptors :

- Rods and cones.
- Light travels through the layers of retina and reaches the rods and cones which generate the impulse → 1st order neurons.

Retinal Pigment epithelium (RPE)

- Forms the outer blood retinal barrier.
- Phagocytic function.

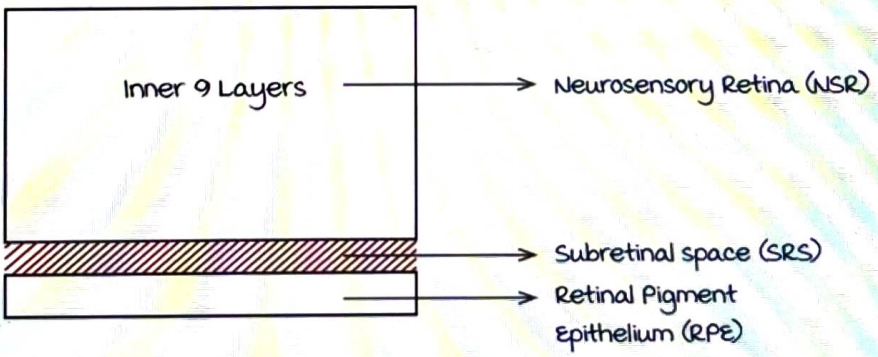
Anatomy of retina

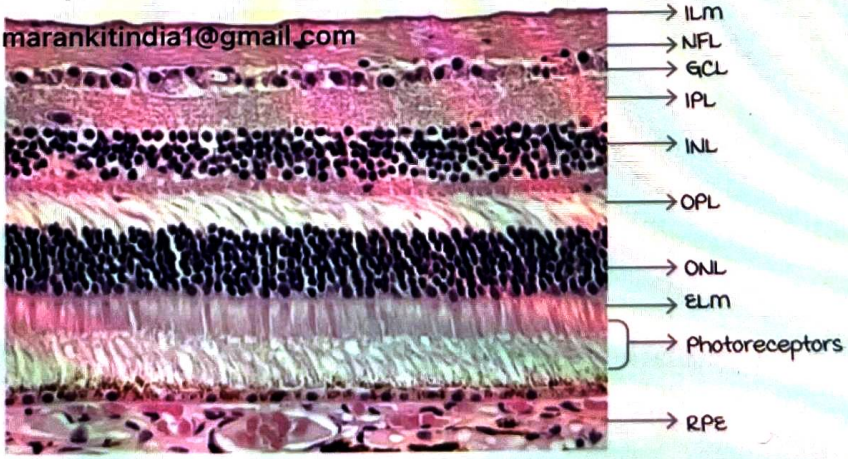
00:21:57

Ganglion cells → First site of generation of action potential.
Photoreceptors generate local graded potential which is transmitted via electronic conduction.
Neuroglial cells in retina :

- Astrocytes.
- Muller's cells.
- Microglia.

Subretinal space is in between the neurosensory retina & RPE.





Phagocytic function.

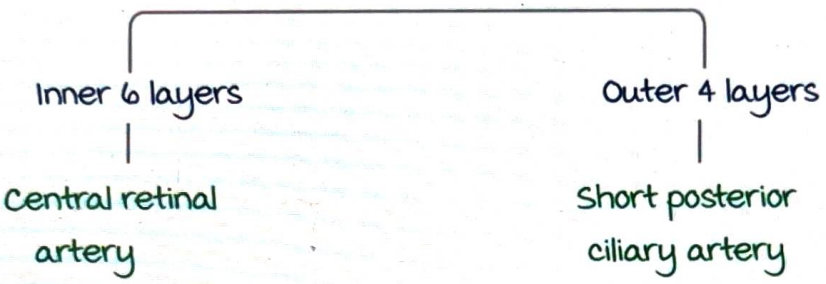
Blood retinal barrier

Outer blood retinal barrier → Formed by RPE.

Inner blood retinal barrier → Formed by endothelial cells of retinal capillary in OPL.

Blood supply of the retina

00:30:25



Macula :

- Supplied by Central retinal artery.
- In some cases also by Cilioretinal artery. (Branch of short posterior ciliary artery).

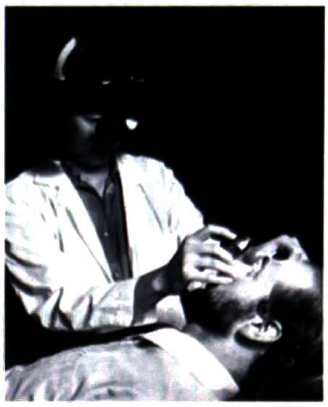
Investigations : Ophthalmoscopy

00:36:02

1. Ophthalmoscopy : Fundus examination.

Ophthalmoscopy	Direct	Indirect
Features	 Looks like a retinoscope.	
magnification	15 times	3 – 5 times
Lens	Convex (inside the instrument)	Convex

Active space

		• most common power +20 D  • Condensing lens in hand → +20 D
Image formed	Virtual and erect.	Real and inverted.
Area visualised	Smaller a. Central fundus or b. Optic disc and macula or c. 2 Disc diameters d. 10° view. Cannot be done if hazy media.	Larger a. Central + Peripheral fundus or b. Optic disc + macula + Ora serrata or c. 8 Disc diameters d. 45° view. Can be done if hazy media.
Stereopsis Depth perception (function of binocular vision)	Absent	Present
Distance of examination	As close to the face as possible	An arm's length
Patient's position	Sitting or Standing	Supine

Direct ophthalmoscopy → Glaucoma, Papillary edema.
Indirect ophthalmoscopy → Retinal detachment.

Investigations : FFA, ICGA and OCT 00:55:18

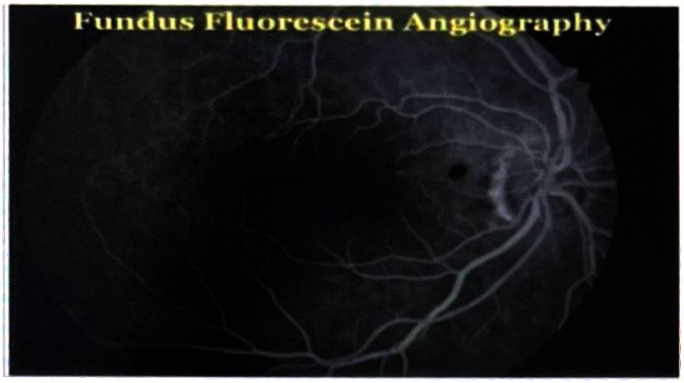
2. Fundus Fluorescein Angiography (FFA)

- Fluorescein → Dye used for measuring fluorescence

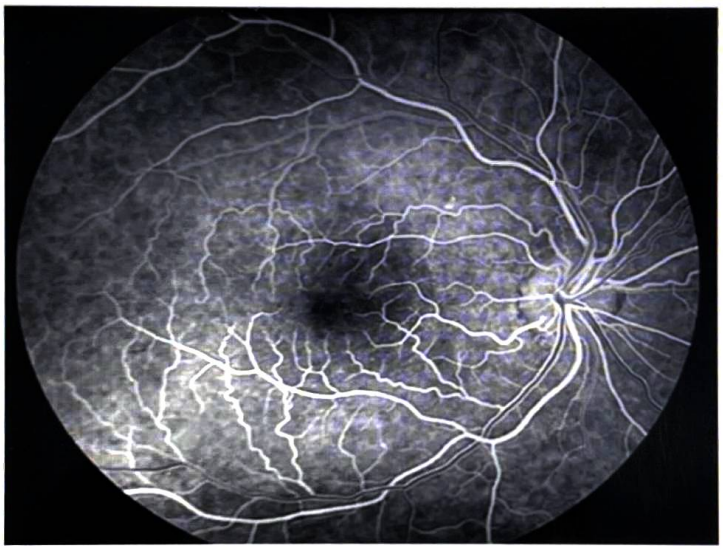
Water soluble orange coloured dye.

- Injected as 5mL of 10 % solution in antecubital vein.
- Blue light → Excitation of the fluorescein molecules at (520 to 530) nm.

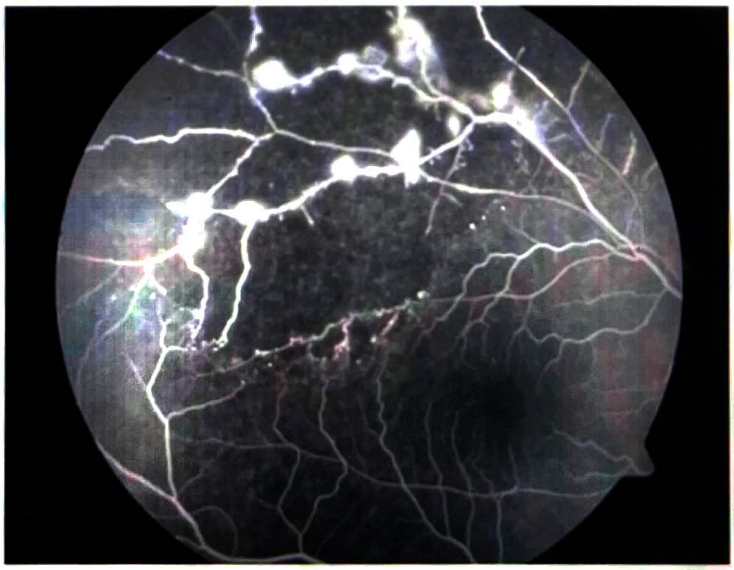
Normal FFA



- Used to diagnose :
  a. Vascular pathologies



Leakage of dye → Hyperfluorescent areas.

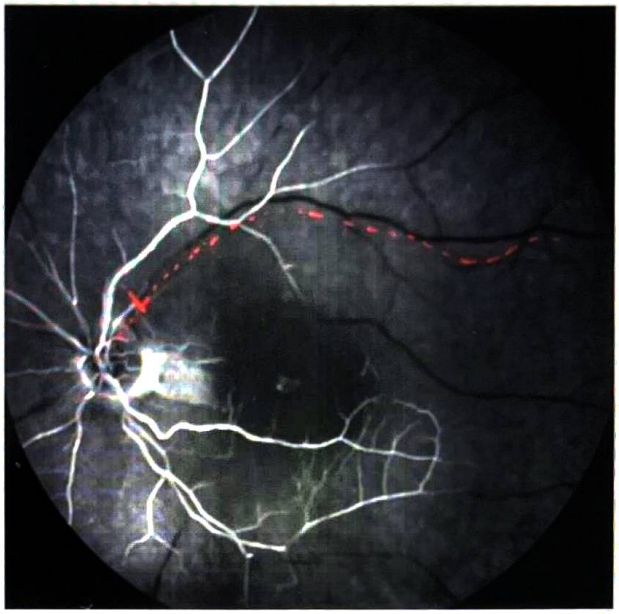


Active space

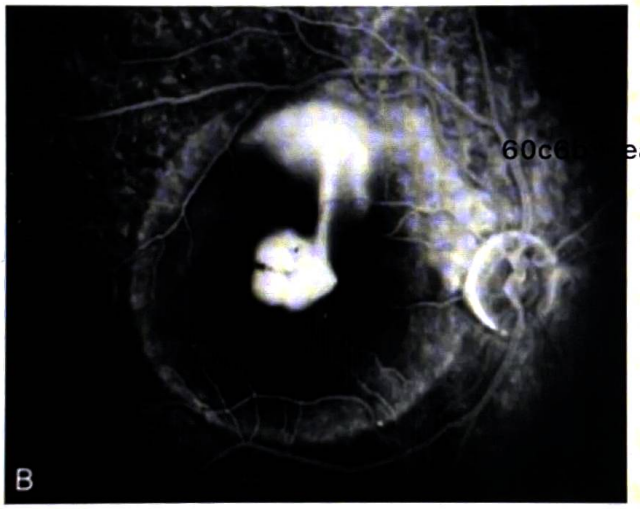
Hypofluorescent due to blockage.

b. Macular pathologies

(Normal macula appears dark on FFA)



Leakage/Pooling of dye at Macula → Macular disease



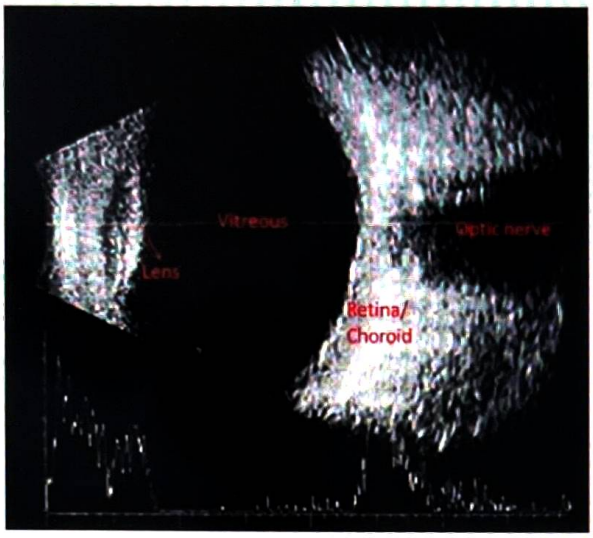
3. Indo-Cyanine Green Angiography (ICGA)

- Dye → Indo-Cyanine Green.
- Better for visualisation of choroidal circulation as :
  1. It is 98% protein bound → Better retention.
  2. Fluoresces at 805nm → Better penetration.
- Contraindication → Iodine hypersensitivity.

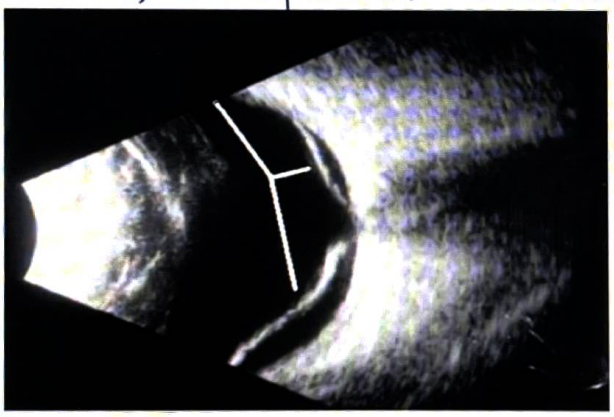
4. B-Scan Ultrasonography

- Brightness scan.
- Uses (7.5 – 10) MHz ultrasonic waves.

- Generate echoes which get recorded as hyperechoic (appear bright)/ hypoechoic (appear dark).
- Ultrasonography is done in patients with ocular media opacity → Corneal opacity or Cataract.
- Normal B – Scan



Vitreous → Hypoechoic, Black colour.
Retina (anterior), choroid (posterior) → White colour.



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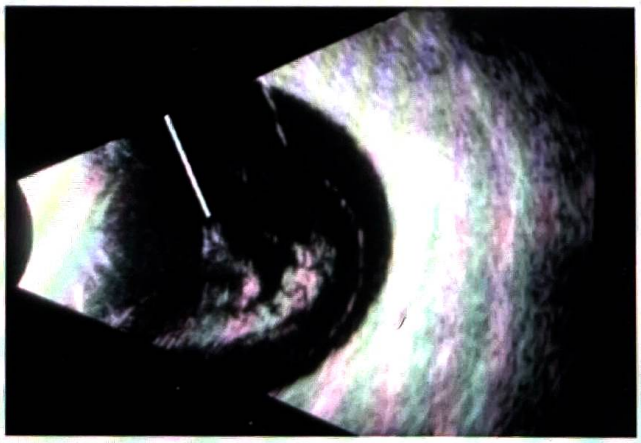
Retinal detachment.



Vitreous hemorrhage

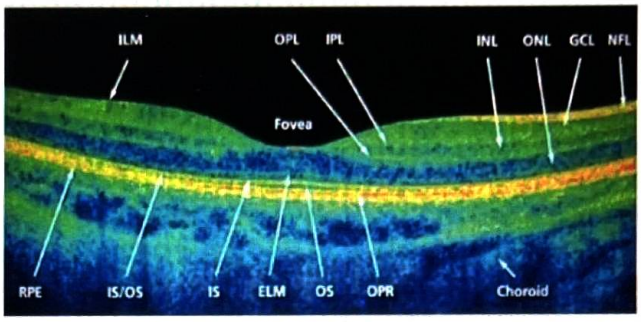
Active space

5. OCT (Optical Coherence Tomography)

Principle → Low coherence interferometry.

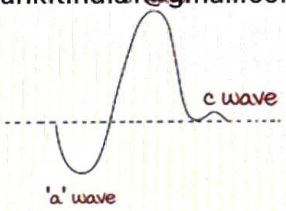
Optical biopsy to see the microstructure.

Normal :



6. Electroretinography (ERG)

- A record of action potential.
- Source → Panretina



- 'a' wave → Generated by photoreceptors.
- 'b' wave → Generated by muller cells.
  Conducted by Bipolar cells.
- 'c' wave → metabolism in retinal pigment epithelium.

Special ERGs :

a. Early Receptor Potential (ERP)
b. Multifocal ERG.

} Generated by cones alone.

c. Pattern ERG → used to diagnose Glaucoma, as it records responses from Ganglion cells, correlating with optic nerve function.

Electrooculography 01:25:05

A record of standing potential.

Source :

a. Photoreceptors.
b. Retinal pigment epithelium.

} Outer retina.

Therefore helps in the diagnosis of diseases of outer retina.

Responses are measured as Arden's ratio :

- Arden's ratio = $\frac{\text{Light peak (highest value in lit condition)}}{\text{Dark trough (lowest value in dark)}}$
- Normally Arden's ratio > 1.85 or 185%.

RETINA: FUNDUS DYSTROPHIES

Introduction 00:00:16

Dystrophies which affect photoreceptor retinal pigment epithelium choriocapillaris complex and lead to rod & cone cell degeneration.

Rods	Cones
Night vision (scotopic vision)	Colour/day vision (photopic)
Peripheral vision, as rods are maximum in perifoveal area	Central vision, as cones are maximum at fovea
Lesion : Nyctalopia (night blindness) Peripheral loss of vision	Lesion : Hemeralopia (colour/dayblindness) Central scotoma

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Causes of Nyctalopia – OMG! (CRAP)$_2$
Oguchi disease
Myopia,
Gyrate atrophy
Congenital stationary night blindness
Cataract cortical
Retinal detachment
Retinitis pigmentosa
Albipunctatus—Fundus
A vitamin A deficiency
Phenothiazine toxicity
Post refractive surgery

Causes of Hemeralopia – C$_3$, A$_3$, T$_1$
- **C** →
  - Cone dystrophy
  - Central cataract
  - (Nuclear and posterior subcapsular)
  - Cohen syndrome
- **A** →
  - Achromatopsia
  - Aniridia
  - Albinism
- **T** → Trimethadione

Retinitis pigmentosa 00:07:59

- MC hereditary fundus dystrophy.
- MC mode of inheritance : Autosomal recessive.
- Autosomal dominant : Best prognosis.
- X linked recessive : Worst prognosis.

Symptoms :
It is a rod – cone dystrophy.
Initially, rods are affected and later cones.

Active space

mid peripheral superior scotoma

↓

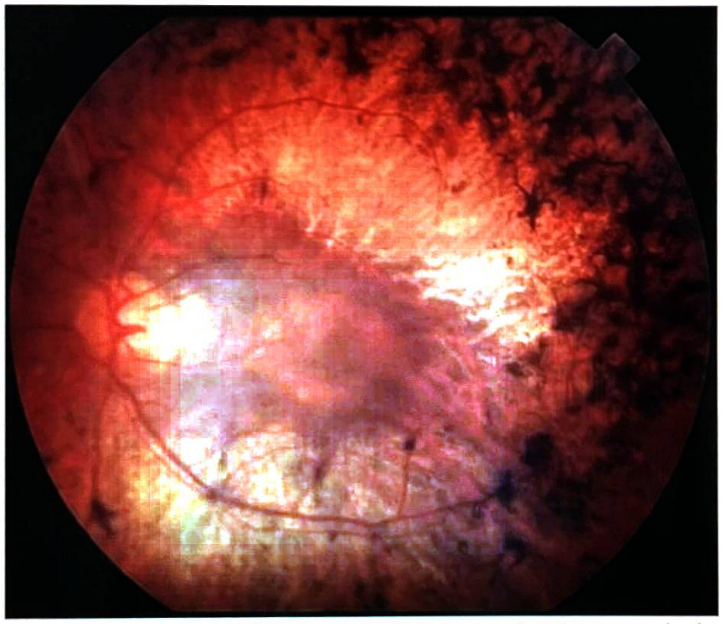
Ring scotoma or doughnut scotoma (central vision preserved and peripheral vision loss)

↓

Tubular or tunnel vision.

Signs of retinitis pigmentosa 00:13:35

1. Arteriolar attenuation.
2. Optic disc : Waxy, pale (consecutive optic atrophy).
3. Bony spicule pigmentation (perivascular).
4. Shining reflex at macula known as tapetal reflex : Only in X linked recessive female carriers. 60c6b3eeaa8ded0e4e7e5ea7



Retinitis Pigmnetosa - Bone Spicule Pigmentation

Diagnosis :

IOC : ERG (Electroretinography)

Treatment :

In India : No treatment

The possible treatment :

- ARGUS II retinal prosthesis : Restores only central 20° vision.

Active space

- Voretigene neparvovec :
  Used if there is biallelic RPE65 gene mutation.
  It is adeno associated virus vector based gene therapy .

Systemic associations of retinitis pigmentosa 00:21:17

Laurence Moon Biedl syndrome – most common

- Retinitis pigmentosa.
- Mental retardation.
- Obese.
- Polydactyly.

Usher's syndrome :

- Retinitis pigmentosa.
- Deafness.

Refsum disease :

- Retinitis pigmentosa.
- Cerebellar ataxia.
- Polyneuropathy.

Bassenkornzweig syndrome :

- Retinitis pigmentosa.
- Abetalipoproteinemia.
- Acanthocytosis (spur cells).

Best's macular dystrophy 00:24:38

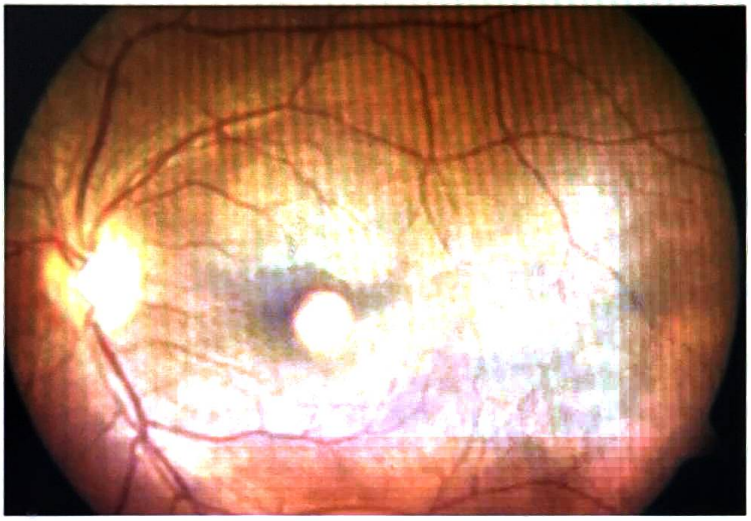
It is autosomal dominant.

Symptoms :

It is primarily a cone dystrophy.
C/F : Hemeralopia (colour/day blindness).
Central scotoma.

Signs :

1. Lipofuscin accumulation
2. Fundus appearance : Vitelliform lesion (egg yolk like)
3. Pseudohypopyon : Vitelli eruptive stage



Diagnosis

IOC : EOG : measured by arden's ratio < 1.50 or 150 %.

ERG is normal.

Stargardt's disease

00:29:59

It is autosomal recessive.

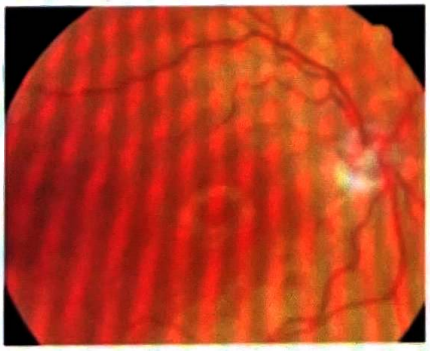
mc juvenile macular dystrophy : Cone dystrophy.

C/F : Hemeralopia (colour/day blindness).

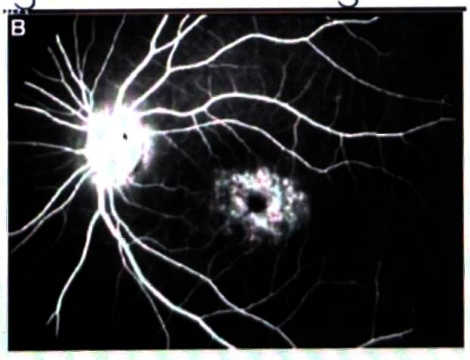
Central scotoma.

Signs : 69c8b3eeaa8ded0e4e7e5ea7

1. Bull's eye maculopathy.
2. On FFA : Dark choroid sign or silent choroid sign.



1.



2.

Active space

Normal FFA : Background fluorescence due to dye in choroidal vessels

Bull's eye maculopathy 00:34:34

It is due to chloroquine toxicity.

Stages :

1. Pre maculopathy : It is reversible, there is normal vision with central scotoma.
2. Maculopathy : Bull's eye lesion seen, it is irreversible and decreased visual acuity.

   It occurs if : Daily dose >3.5 mg/kg or 250 mg.

   Cumulative dose >460 g.

   Duration of treatment > 5 years.

   Renal or hepatic function compromise.

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Chloroquine can also lead to vortex keratopathy.

Congenital night blindness disorders 00:38:12

It presents with infantile onset nyctalopia.

Disease	ERG	Fundus
Congenital stationary night blindness	Negative	Normal
Oguchi blindness	Normal	Abnormal
Enhanced S - cone syndrome	Negative	Abnormal
Fundus albipunctatus	Absent rod responses	Abnormal

Oguchi blindness

- There is no damage to rod cells, instead there is damage in conduction.
- Therefore, it is diagnosed by the mizuonakamura phenomenon : In light conditions fundus has golden yellow colour.

Miscellaneous conditions:

- X linked retinoschisis (schisis - split).
- There is split in retina between nerve fibre layer and rest of retina.
  It presents with Bicycle wheel maculopathy.
- Stickler's syndrome : MC inherited cause of RD in children.

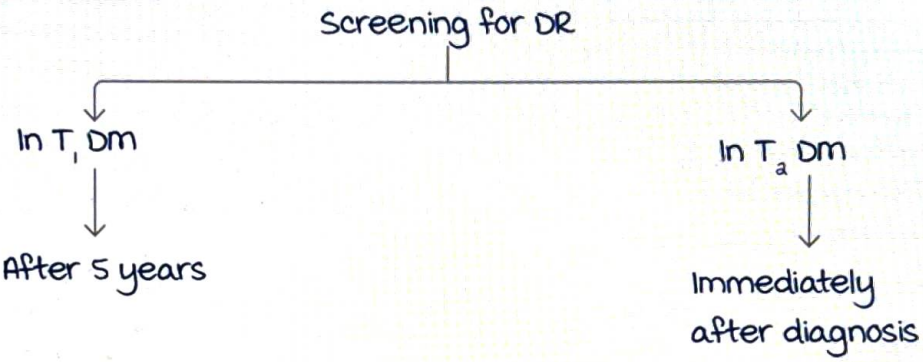
RETINA - VASCULAR DISORDERS PART I

Vascular retinal disorders are group of disorders that affect the blood vessels of retina.

Diabetic retinopathy (DR) 00:01:47

Risk factors :

- Duration of diabetes is directly proportional to risk.



- Increased HbA_1C.
- Pregnancy.
- Hypertension and hyperlipidemia.
- Diabetic nephropathy.

Pathogenesis :

Biochemical mechanisms of cellular damage –

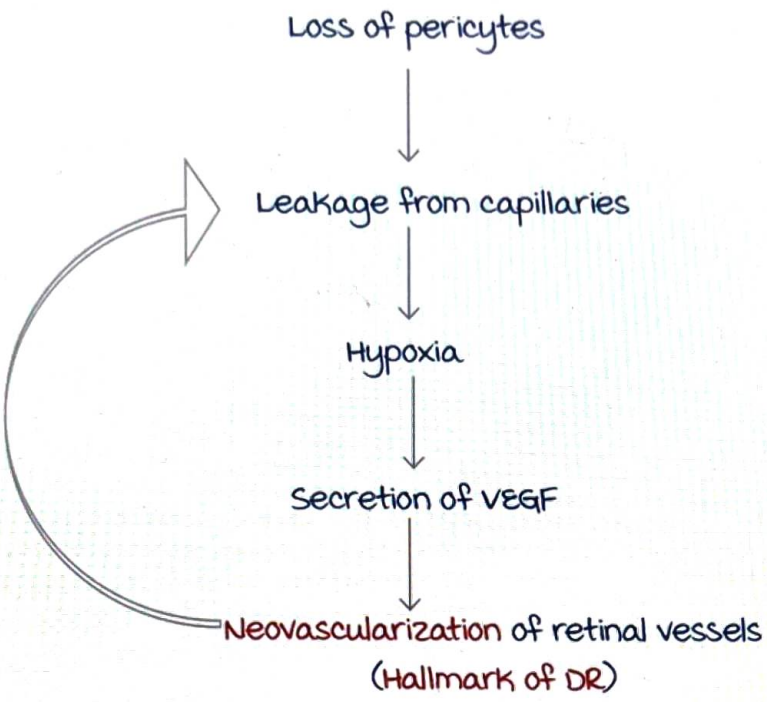
1. Increased sorbitol accumilation.
2. Protein Kinase C activation.
3. Accumulation of Reactive oxygen intermediates (ROI).
4. Accumulation of advanced glycation end products.

All these lead to Anatomical lesions.

Earliest anatomic pathological change : Loss of pericytes.

Pericytes : Layer of protection over capillaries (strengthening of endothelium).

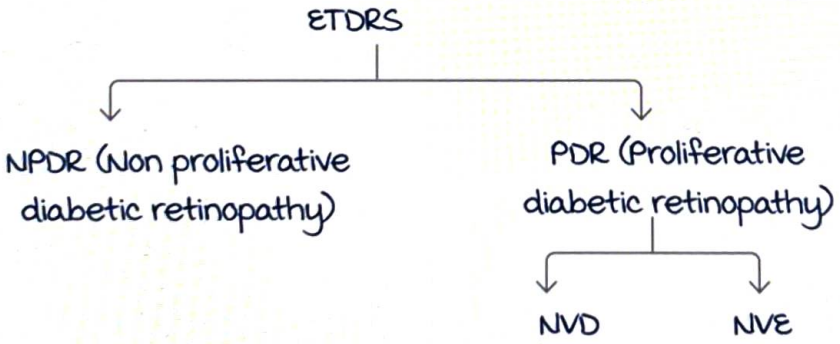
Has 2 functions : Protective and contractile function (pumping of blood).



Neovascularization : These are leaky vessels.

Clinical features of DR

Classification : ETDRS – Early treatment DR study.



Proliferative DR :

Signs : Neovascularization +.

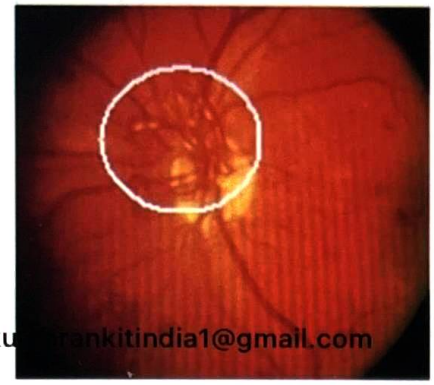
2 types : NVD (disc) and NVE (elsewhere).

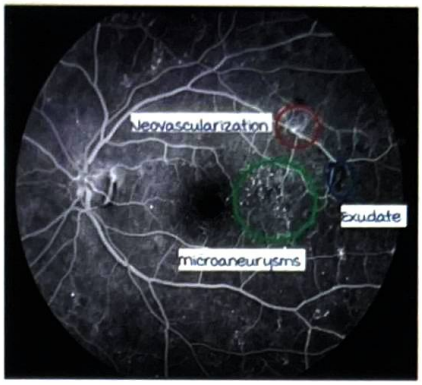
NVD : Neovascularization at the disc or within 1 disc diameter.

O/E :

New vessels appears as tortuous tuft of vessels.

Investigations : FFA – Hyperfluoroscent.





Non Proliferative Diabetic Retinopathy :

Earliest sign : Micoraneurysms.

O/E : Tiny small red dots.

FFA : Tiny hyperfluorescent dots.

Layer of abnormalities is seen in inner nuclear layer.

Hemorrhage :

Flame shaped hemorrhage : Long and thin found in superficial nerve fibre layer.

Dot and blot hemorrhage : Deep and found in deep plexiform layer.

On FFA : Hyperfluorescent areas.

Exudates : Lipid deposits in outer plexiform layer.

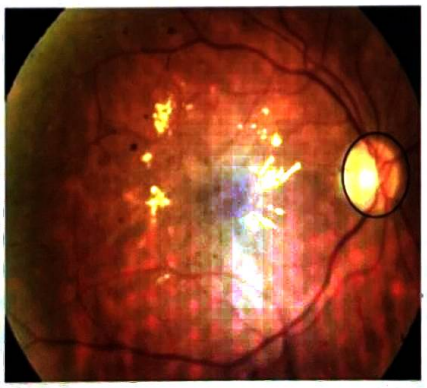
O/E : Yellowish irregular deposits.

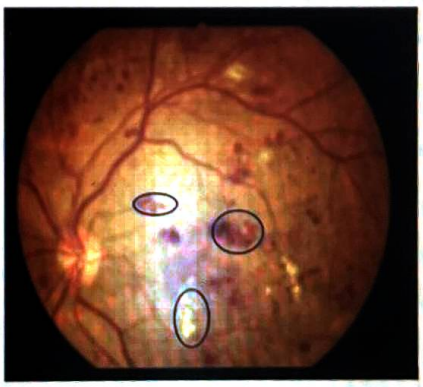
FFA : Hypofluorescent area.

Cotton wool spots : Aka soft exudates.

Collections of neuronal debris in nerve fibre layer.

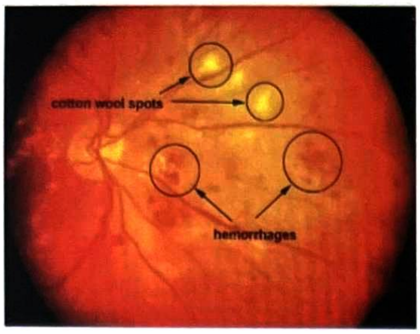
O/E : Yellowish large deposits, regular smooth edges.

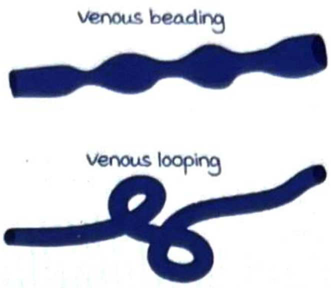




Exudates along with hemorrhages are seen.

Active space





Venous beeding/looping (Aka venous tortousity) due to hypoxia.

IRMA : Intra retinal microvascular abnormalities.

- Leakage of blood vessels due to which the areas of retina in front of these vessels become hypoxic.
- Rest of the vessels forms a shunt at that area.
- These are precursors to new vessels.
- These bypass the capillary bed.

Treatment

00:33:35

Divided into 2 stages based on treatment.

NPDR	PDR
Glycemic control and follow up.	RxOC : Pan retinal photocoagulation (PRP). Pan retinal as the new vessels can be anywhere. Laser used : Argon green. Done in multiple sittings. m/c side effect : visual field defects
4 stages : mild, moderate, severe and very severe. Stage — Follow up mild — 9-12 months moderate — 6 months Severe — 4 months very severe — 2 months	Anti VEGF drugs : Ranibizumab, Bevacizumab, etc.

Severe and very severe : 4/2/1 rule : 4 quadrants of hemorrhage, 2 quadrants of venous bleeding and 1 quadrant of IRMA. Out of 3, any 1 seen : Severe NPDR, Out of 3, > 1 seen : very severe NPDR.	Not all cases of PDR are treated. Done only in high risk PDR. a) NVD ≥ 1/3 disc area. b) NVD with vitreous hemorrhage. c) NVE > 1/2 disc area with vitreous hemorrhage.

Complications

00:43:05

1. Diabetic macular edema : Due to leakage.

m/c cause of loss of vision in diabetic retinopathy.

m/c cause of loss of vision in non proliferative DR : Cystoid macular edema.

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Rx :

a) RxOC : Focal laser photocoagulation.

b) Intravitreal triamcinolone : Preferred if

- Ischaemic maculopathy,
- Pseudophakic patients.
- Patient with co-existing cardiovascular disease.

c) Anti VEGF drugs : Preferred if centre involving edema (within 300 microns of centre of macula with foveal thickness > 250 microns). Photocoagulation not done as fovea may get targeted accidentally.

Treatment is done only if :

Clinically significant macular Edema (CSME) present

i.e., retinal thickening within 500 microns of centre of macula

or

Exudates within 500 microns of centre of macula (but if retinal thickening present anywhere).

or

Retinal thickening or exudates > 1 Disc Diameter (1 DD) in size within 1 Disc Diameter of centre of macula.

(1 DD = 1500 microns).

2. Vitreous hemorrhage :

Vitreous is avascular.

Source of hemorrhage : Neovascularization of retina.

m/c cause of loss of vision in PDR.

As vitreous is transparent, collection of blood will obstruct light from striking the retina.

Rx :

- Small vitreous hemorrhage (not in visual axis) – Observe. Keep head elevated, wait for it to get absorbed.
- Large or recurrent – Endophotocoagulation (done internally) or pars plana vitrectomy.

m/c cause of vitreous hemorrhage :

- In adults : Diabetic retinopathy.
- In young adults (20–40) : Blunt trauma (bleeding from normal retinal vessels).

m/c cause of recurrent vitreous hemorrhage : Eale's disease.

3. Retinal detachment : m/c cause of blindness.
4. Rubiosis iridis : Neovascularization of iris.

It occurs due to oxygen demand by the posterior segment on anterior segment.

Iris lends blood supply to posterior segment. In turn, it becomes hypoxic. This leads to neovascularisation of iris.

RETINAL VASCULAR DISORDERS PART 2

Hypertensive Retinopathy (HTNR) 00:00:09

HTN
↓
Vasoconstrictive phase
↓
Sclerotic phase (atherosclerosis)
↓
Exudative phase

Clinical features of HTNR :

Keith-Wagner classification :

Grade 1 :

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Arteriolar attenuation.

Normal AV (arteriovenous) ratio (diameter) = 2 : 3.

HTNR AV ratio = 1 : 3.

Grade 2 :

Salus sign : Deflection of blood vessel.

Grade 3 :

Bonnet sign : Banking of blood vessel.

Gunn sign : Tapering of blood vessel.

Flame shaped haemorrhages with cotton wool spots.

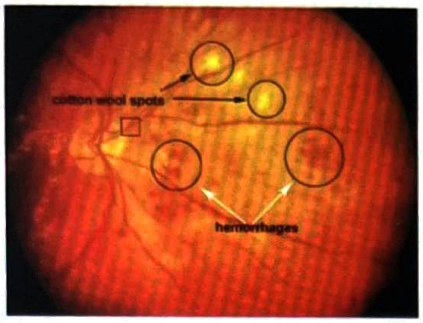
Copper wiring of arterioles.

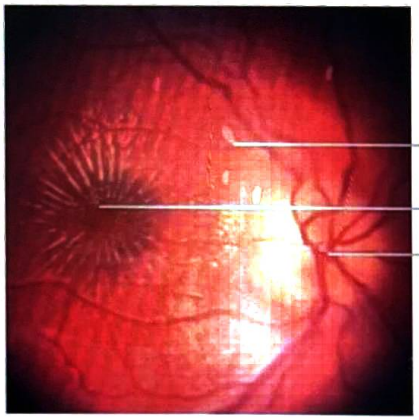
Deflection, banking and tapering are seen at AV crossings in veins.

Grade 4 :

Silver wiring of arterioles.

Papilledema.





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Cotton wool spot

Papilledema

Macular star appearance, seen in malignant HTN

Central Retinal Artery Occlusion (CRAO)

00:10:18

Causes of CRAO :

1. Most common : Atherosclerosis.
2. Emboli : Hollenhorst plaque
   ↓
   Cholesterol emboli
   Originates from bifurcation of common carotid artery.
3. Giant cell arteritis.

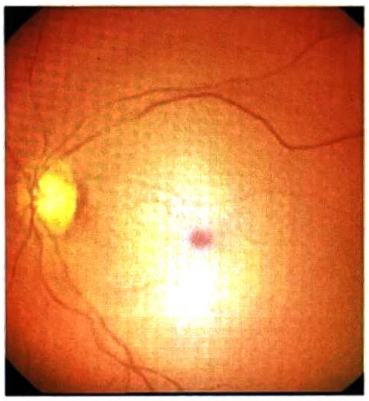
Clinical features of CRAO :

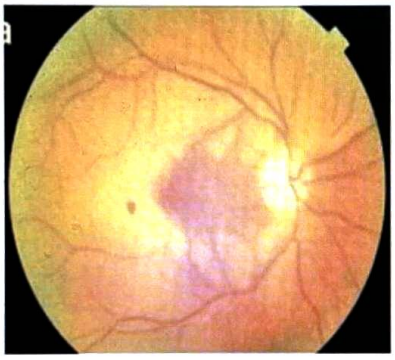
1. Sudden and painless loss of vision.
2. Optic atrophy : Consecutive.
3. Pupil : RAPD or Marcus Gunn pupil.
4. Fundus appearance : Pale fundus (due to retinal edema) : Cattle tracking fundus (due to segmentation of blood column).
5. Cherry red spot at macula : Due to collection of fluid in ganglion cell layer of retina, which is absent at

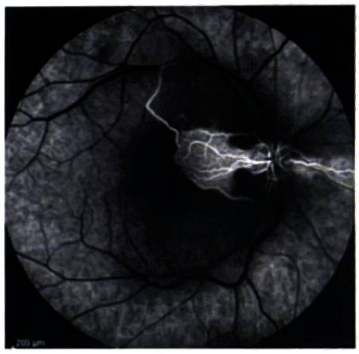
Active space

foveola making it transparent.

Whole retina is edematous or hazy : Pale fundus as choroid red in colour is not visible due to retinal hemorrhage.
But foveola remains transparent (choroidal red colour is visible at foveola).







CRAO with macular cilioretinal artery spared.
On FFA : No dye in any blood vessels, dye filling cilioretinal artery.

Differential diagnosis of cherry red spot at macula :
Mnemonics : Cherry Trees Never Grow Tall in Sand, Mud and Grime.

a) CRAO.
b) Trauma.
c) Neimann Pick's disease
d) Gangliosidosis–type I.
e) Tay Sach's disease.
f) Sandhoff's disease.
g) Metachromatic leukodystrophy.
h) Gaucher's disease.

Gaucher's disease : 3 types.
Type 1 : No cherry red spot.
Type 2 : Cherry red spot +.
Type 3 : may be +.

Gaucher's is the least common cause of cherry red spot.

Treatment of CRAO 00:26:38

Treatment should be done within 4 hours (within 24–48 hours occlusions become complete).

1. Ocular massage : Dislodging emboli.
2. Vasodilatation :
   Sublingual isosorbide nitrate.
   Carbogen inhalation (95 % O_2 + 5% CO_2).

5% CO_2 : Hypercarbia (increased PCO_2) ⟶ Decreased pH ⟶ vasodilation ⟶ respiratory acidosis.

3. Lower the intraocular pressure :
   IV mannitol.
   Paracentesis (aspiration of aqueous).

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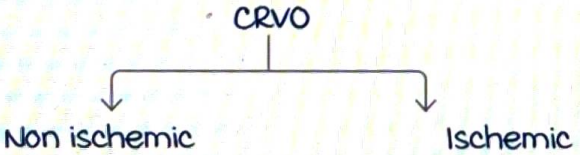
Central Retinal Vein Occlusion (CRVO) 00:32:02

Causes of CRVO :
Most common cause : Hypertension.



Non-ischemic :
Occurs due to stasis of blood.
Hypoxia.
Increased vascular permeablity leading to macular edema and resulting in loss of vision.

Treatment :

Intravitreal triamcinolone.

0.7 mg dexamethasone intravitreal implants (based on geneva studies).

Anti VEGF drugs.

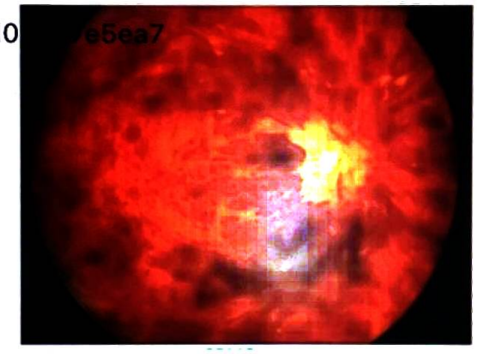
Ischemic CRVO :

Severe hypoxia leads to damage to capillary endothelium.

Clinical features :

Severe flame shaped hemorrhages :

Fundus : Tomato Ketchup/splash appearance.



Rubeosis iridis (neovascularization of iris)

↓

Occurs due to O_a demand by posterior segment

↓

Leaking of blood in anterior chamber

↓

This blocks trabecular meshwork

↓

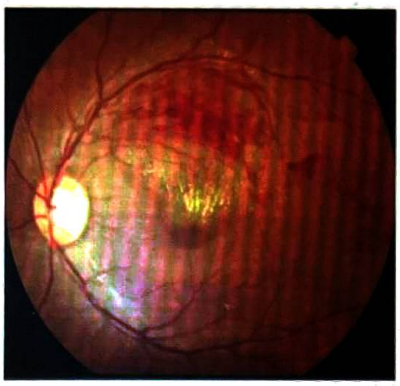
Causes neovascular glaucoma

Neovascular glaucoma in CRVO occurs after around 3 months, known as 100-day glaucoma.

m/c cause of neovascular glaucoma : Diabetic retinopathy.

Rx of ischemic CRVO : PRP (Pan Retinal Photocoagulation).

Branched retinal vein occlusion (BRVO) 00:43:10



Most common site of occlusion : AV crossings.
Most common quadrant affected : Superotemporal.

Retinopathy Of Prematurity (ROP) 00:44:32

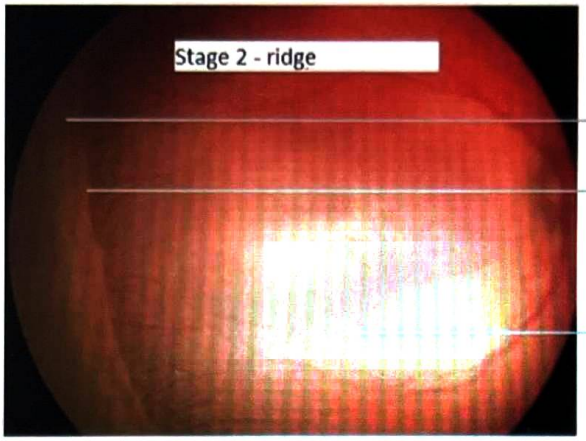
Occur in premature child, when :
Age at birth ≤ 32 weeks.
Birth weight ≤ 1750 gram.

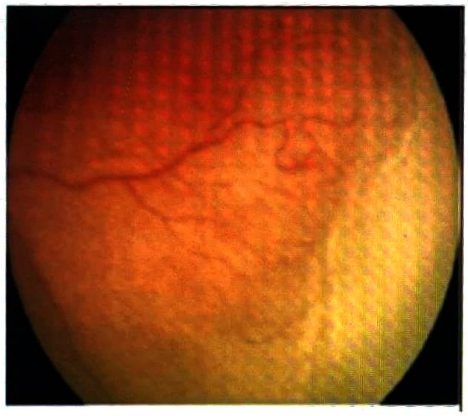
Screening for ROP at 4 weeks after birth :
Age at birth ≥ 28 weeks.
and
Birth weight ≥ 1200 grams.

Screening for ROP at 2-3 weeks after birth :
Age at birth < 28 weeks.
or
Birth weight < 1200 grams.

Signs/stages of ROP :
Stage 1 : Demarcating line between vascularized retina and the peripheral avascular retina.
Stage 2 : Ridge.
Stage 3 : Extra retinal neovascularization on the ridge moving into vitreous.
Stage 4 : Partial retinal detachment.
Stage 5 : Total retinal detachment.

Active space





Stage 3 ROP

Treatment of ROP :

Laser photocoagulation : Done if pre threshold ROP type 1 as told by ETROP study).

or

Pars plana vitrectomy (if retinal detachment).

Pre threshold ROP type 1 :

Zone 1 affected + any stage + plus disease.

Zone 1 + stage 3.

Zone 2 + stage 2/3 + plus disease.

Plus disease is when venous tortousity and dilatation in ≥ 2 quadrants.

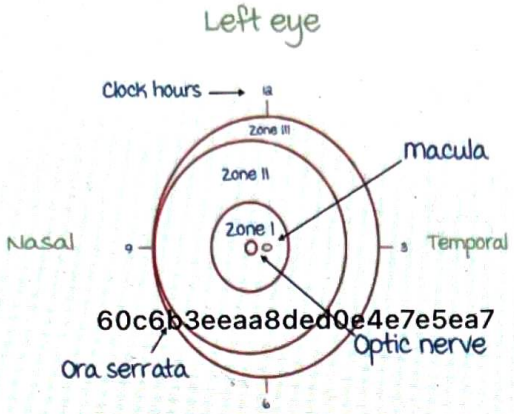
Zones :

Zone 1 : From optic disc and 6 mm (4DD) around it.

Zone 2 : From zone 1 boundary extending upto nasal ora serrata.

Zone 3 : Seen only temporally (remaining part).

Active space



Miscellaneous diseases

00:57:22

Eale's disease

Triad of :

- Occlusion.
- Periphlebitis (inflammation around veins).
- Neovascularization of retina (MCC of recurrent vitreal Hemorrhage).

Rx : PRP.

Coat's disease

Idiopathic retinal telangiectasia.

M/C in males.

Unilateral.

C/F :

- Painless and non inflammatory loss of vision.
- Strabismus.
- Leukocoria.
- Exudative RD.

RETINA : RETINAL DETACHMENT

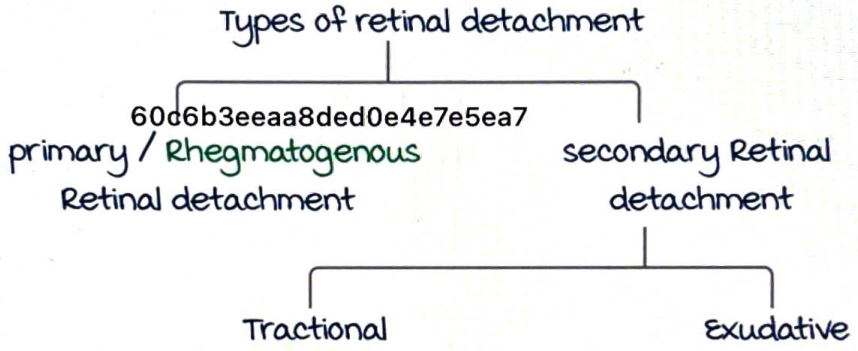
Introduction 00:00:58

It is the separation of the neurosensory retina from the retinal pigment epithelium and collection of subretinal fluid.

Investigations :

- Investigation of choice : Indirect ophthalmoscopy.
- Optical Coherence Tomography (OCT).
- B-scan ultrasonography.

Types of retinal detachment and primary retinal detachment 00:03:10



Primary retinal detachment is also known as Rhegmatogenous retinal detachment.

'Rhegma' means a break (tear or hole) in the retina.

Pathogenesis :

- Syneresis (liquefaction of vitreous).

↓

Leads to posterior vitreous detachment (PVD).

↓

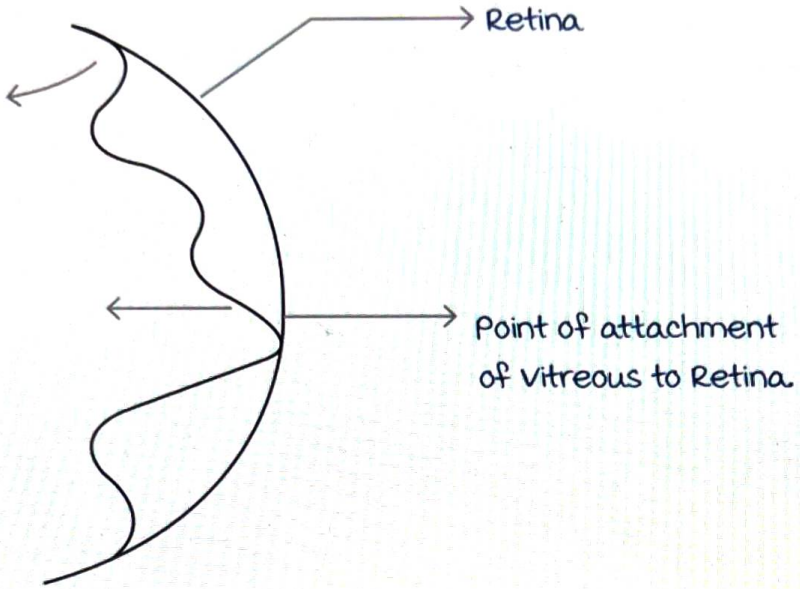
Some part of vitreous still attached exerts dynamic vitreoretinal traction.

↓

Retinal break develops.

↓

The fluid enters through the break and seeps into the subretinal space.



Causes :

1. MC : Pathological myopia.
2. Blunt trauma.
3. Cataract surgery (aphakia).
4. Lattice degeneration of retina.

Symptoms :

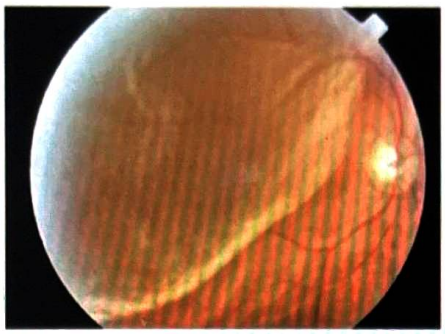
1. Photopsia : Flashes of light.
2. Floaters : Small black flying dot like opacities.
   Due to vitreal opacities.
3. The patient complaints of curtain falling in front of the eye.
4. Loss of vision : Sudden and painless.

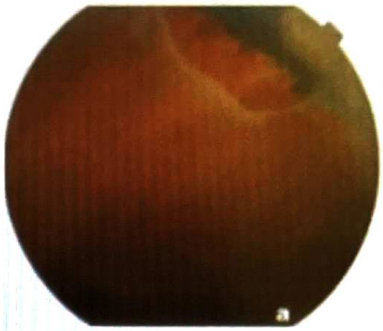
60 Primary retinal detachment : Signs and prophylaxis 00:13:09

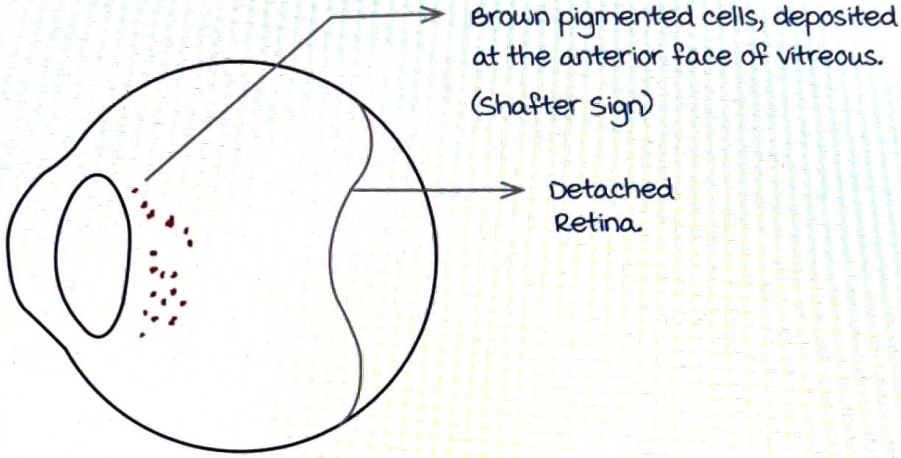
Signs :

1. Detached retina : Gray/opaque, convex shaped.
2. Retinal detachment : Extends till Ora serrata.
3. Shaffer sign : Tobacco dust appearance.

Active space







4. Relative afferent pupillary defect.
5. In long standing cases :
   a. Retinal atrophy.
   b. Subretinal demarcation lines known as High water marks if (>3 months).
   c. Intra retinal cysts : If >1 year.

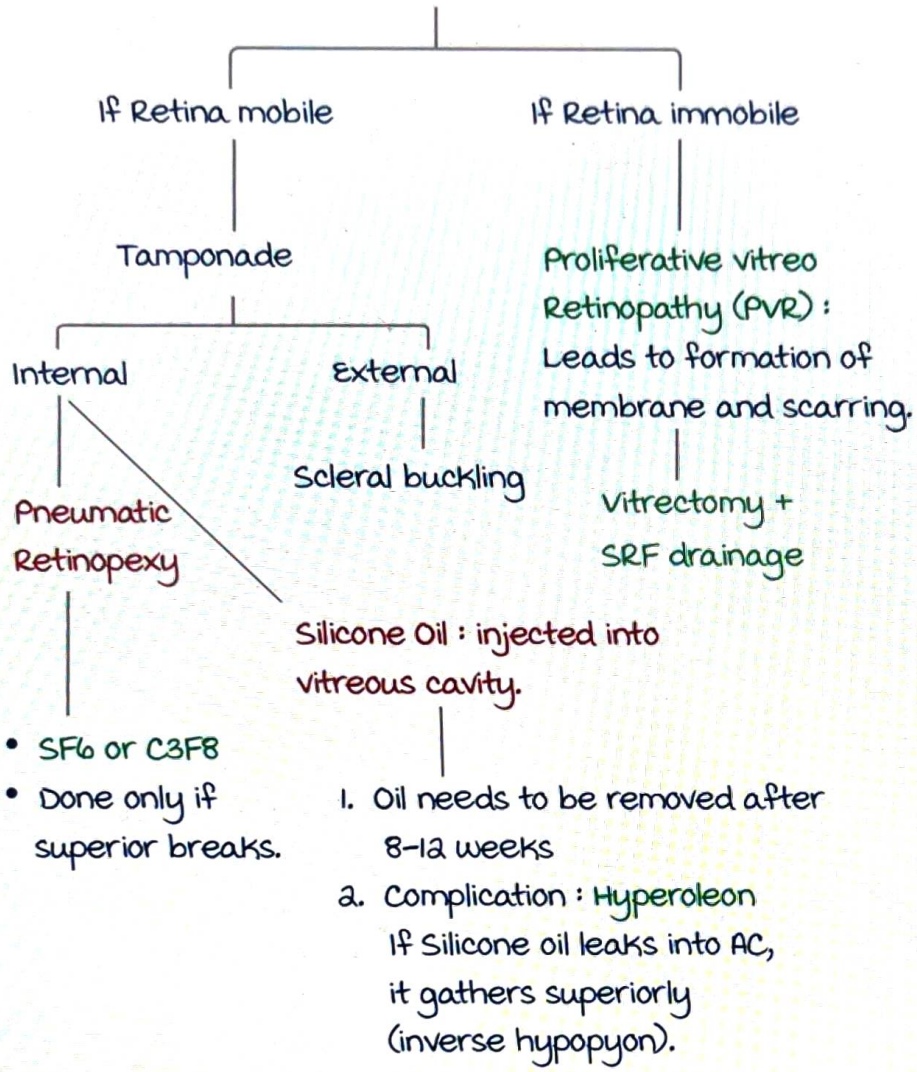
Prophylaxis :

OCT image in long standing Rhegmatogenous retinal detachment.

Breaks that are most commonly seen in Superotemporal quadrant can be closed by laser photocoagulation.

Active space

Primary retinal detachment: Treatment 00:19:42



Lincoff rules : Rules that help to locate the break in retina.

Tractional retinal detachment 00:26:54

No break.
No photopsia, no floaters.
Occurs due to static vitreoretinal traction.

Causes : 60c6b3eeaa8ded0e4e7e5ea7

1. MC : Diabetic retinopathy.
2. Retinopathy of prematurity.
3. Penetrating trauma.
4. Sickle cell retinopathy : Presents with Roth spots (hemorrhage with a clear centre).

Active space

Clinical features :

1. Loss of vision: Gradual and painless.
2. No photopsia, no floaters.
3. Does not extend till Ora serrata.
4. Detached retina is concave in shape.

Exudative retinal detachment 00:31:02

Causes :

1. MC : Choroidal Melanoma.
2. Toxemia of pregnancy.
3. Coats disease.
4. VKH syndrome.
5. Central serous retinopathy.

Clinical features :

1. Loss of vision : Sudden, painless.
2. Convex detached retina.
3. Hallmark : Shifting fluid.

CORNEA-SPECIAL INVESTIGATIONS

Keratometry, Pachymetry and Corneal topography

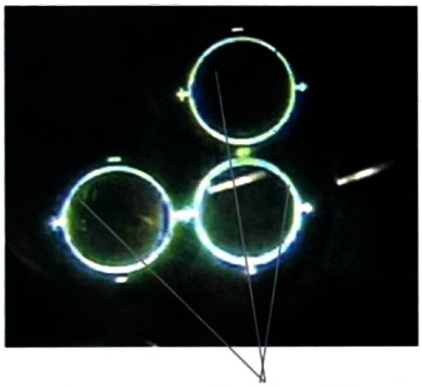
00:02:22

Keratometry :

- measures the corneal curvature.
- Principle :

  Anterior surface of cornea acts like a convex mirror. Size of the image formed is inversely proportional to the curvature.

- Image is seen in form of Keratometric mires.

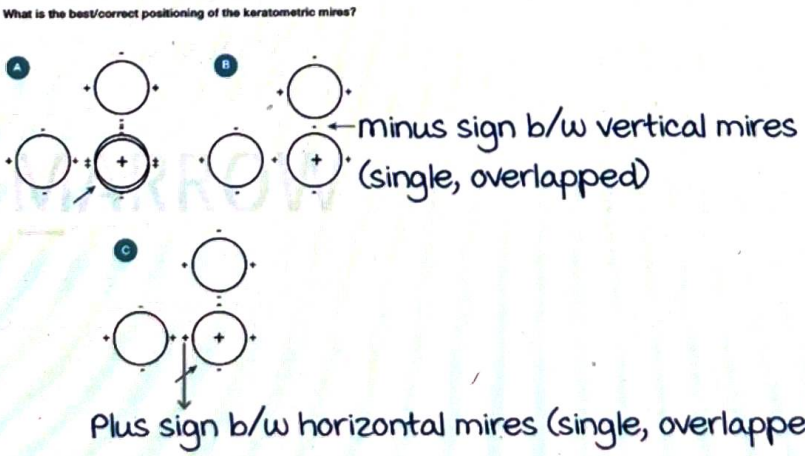


Keratometric mires

Bausch & Lomb Keratometer

Keratometer image

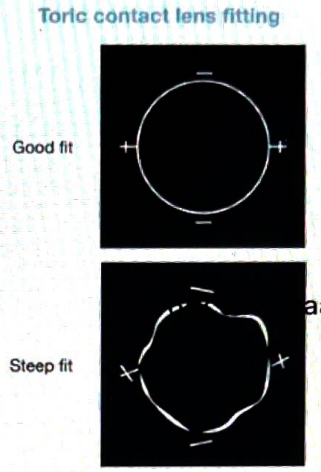
- Correct positioning of mires :



Active space

Keratometry uses :

1. Measures corneal curvature.
2. To diagnose astigmatism.
   - mires horizontally oval : With the rule astigmatism.
   - mires vertically oval : Against the rule astigmatism.
3. To ascertain proper fitting of a contact lens.

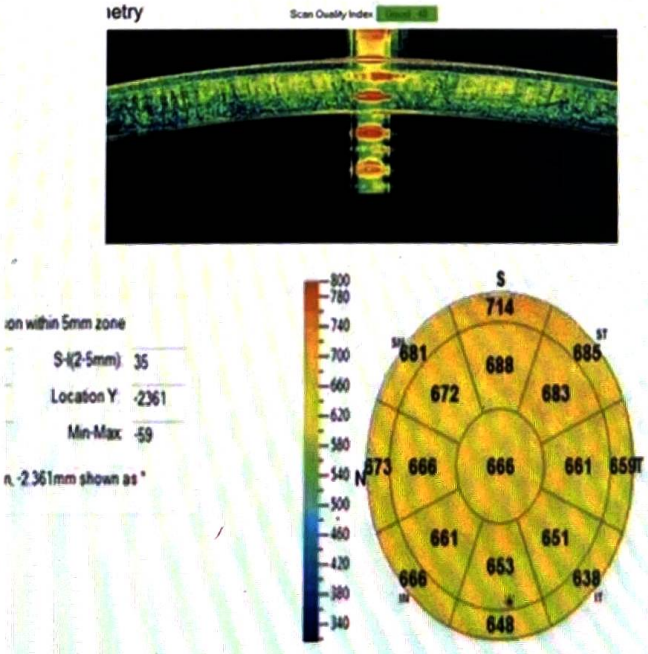


4. To diagnose Keratoconus : Pulsating mires are seen.

Pachymetry

00:11:14

- measures (central) corneal thickness.
- Normal corneal thickness (CT) : 0.54 mm, or 540 μm.
- Corneal thickness increses from centre to periphery.

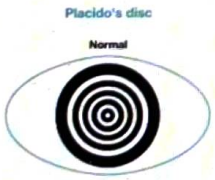


Uses :

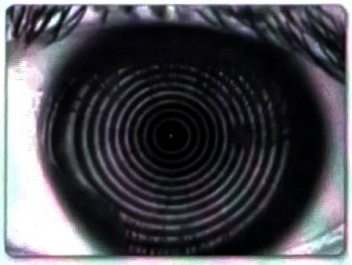
- In cases of LASIK (to ablate cornea).
  LASIK is contraindicated if corneal thickness (CT) is < 450 μm.
- In cases of IOP measurement.
  1 mmHg IOP α 10 μm corneal thickness.
  i.e., IOP false high if CT is more.
  IOP false low if CT is less.

Corneal Topography 00:15:15

- To examine the corneal surface.
- Done using
  a. Placido disc.
  b. Orb-scan (measures corneal thickness + topograph). uses the principle of split scan imaging.
  c. Penta-cam (measures corneal thickness + curvature + topographic elevation of maps of anterior surface + topographic elevation of maps of posterior surface + anterior segment imaging).



Placido disc

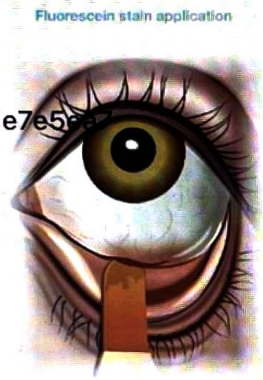


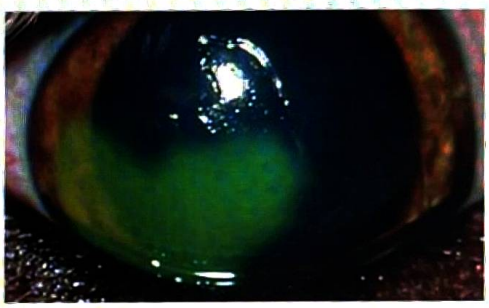
Active space

Corneal vital staining- Fluorescein dye 00:20:12

Stains area of denuded (broken) epithelium.
Stains floor/base of ulcer.
Visualized under cobalt blue light (seen as green fluorescence)
Other uses :

- Goldman's AT. (Applantation tonometry).
- For diagnosing dry eye.
- Jones's dye disappearance test (lacrimation assessment).
- Seidel's test (done in penetrating trauma)

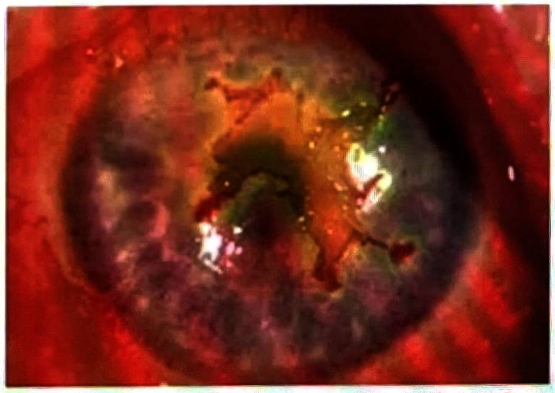




Fluorescein Staining

Corneal vital staining- Rose Bengal dye 00:25:26

Stains devitalized/necrotic tissue.
Stains margin of ulcer.



Rose Bengal Staining

Alcian blue dye :
Stains mucin deposits and filaments.

Lissamine green :

It stains devitalised/ damaged tissue

Advantage : It is not toxic to healthy tissue, as it does not stain healthy tissue. It is preferred over rose bengal stain.

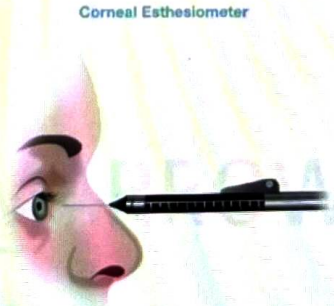
uses : To diagnose dry eye by conjunctival staining.

Confocal microscopy 00:30:42

Non-invasive technique for in-vivo imaging of all layers of living cornea.

Corneal aesthesiometer :

- Examines corneal sensations (also by cotton wisp).
- It is done using a pen like instrument with filament (6 cm long).
- Touch the patient's cornea with the filament → If sensation not felt → Retract the filament by 0.5 cm & check → Repeat till the patient feels the sensation.
- Smaller the length of filament which illicits the corneal sensations, lower the corneal sensations than normal.



Active space

CORNEAL ULCER / KERATITIS

Corneal ulcer :

- Is Abrasion + Keratitis.
- Discontinuity in the corneal epithelium with cellular infiltration and necrosis of the underlying layers.

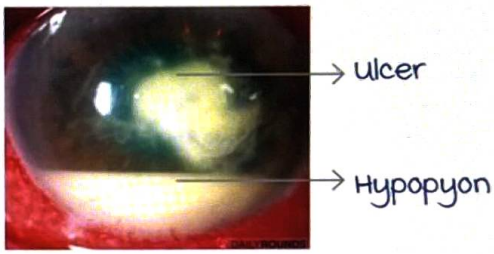
Bacterial Keratitis 00:02:41

most common cause in the world : Staphylococcus aureus.

most common cause in India : Pneumococcus

Ulcer serpens :
Centre to periphery

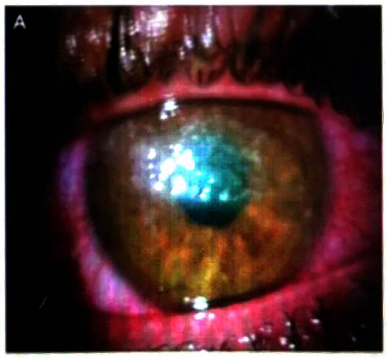
Hypopyon corneal ulcer



Most common cause of bacterial corneal ulcer in contact lens users : Pseudomonas (Always with a green coloured discharge).

Most common cause of bacterial corneal ulcer in patients with prior history of LASIK surgery : Atypical mycobacteria.

Nocardia : Causes Wreath shaped ulcer/ Pin-head ulcers.



Bacteria which invades intact epithelium :

- Neisseria meningitidis, Neisseria gonorrhea.

- Corynebacterium diphtheriae.
- Listeria.
- Shigella.
- Hemophilus aegypticus.

Pathogenesis of Bacterial keratosis 00:11:25

Stage 1 : Initial stage.

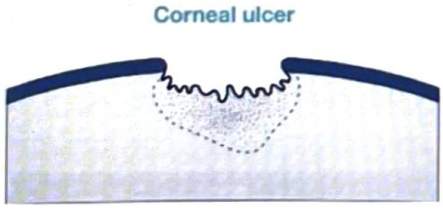
- Saucer shaped ulcer with overhanging margins.

Stage 2 : Progressive stage.

- Infiltration of Polymorpho neutrophils.

Hypopyon — Keratouveitis

- Mobile : liquid like pus.
- Sterile.



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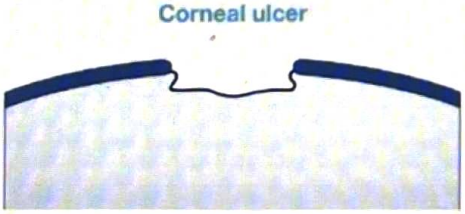
Stage 3 : Regressive stage.

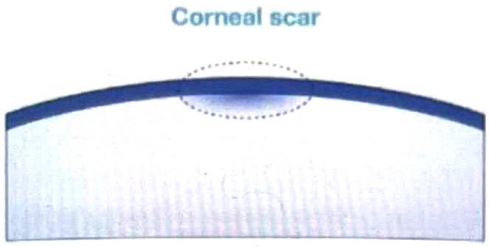
- The floor, edges of ulcer becomes smooth and vascularization is also seen.



Stage 4 : Scar/ Cicatrization.

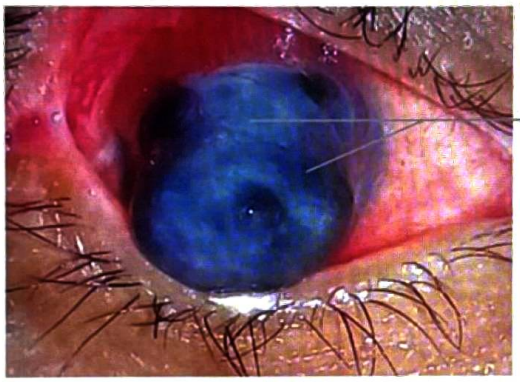
- Permanent visual loss.

Active space

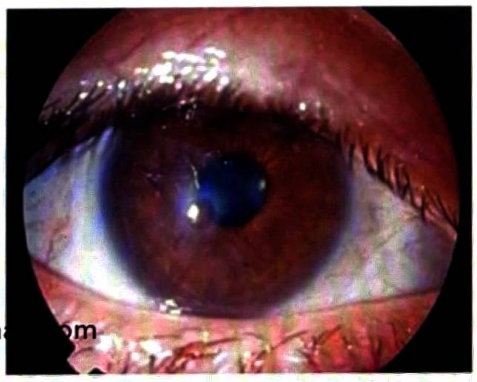


Complications of Bacterial keratosis 00:16:59

1. Ectatic cicatrix :
   Due to thinning, bulging of cornea (maybe temporary/ permanent).
2. Anterior staphyloma :
   Bulging of cornea due to thinning along with incarceration of iris tissue.

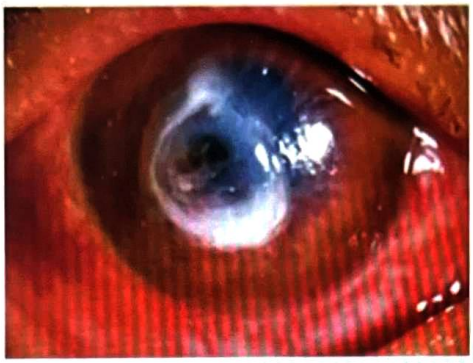


3. Descemetocoele :
   Descemet's membrane herniates through the corneal ulcer.



4. Perforation of cornea :
   - Aqueous leak out : Decreased IOP.
   - Iris prolapse.
   - Pseudocornea : Corneal surface formed due to plugging of perforation by iris tissue.

- Endophthalmitis/ Panophthalmitis



Management of Corneal ulcer 00:28:57

1. Scrape the base of ulcer with 15 No. blade
   ↓
   Send scrapings for Gram's stain and culture.
   ↓
   Antibiotics (Fortified) only after report.
   Usual : Fortified Cephazolin 5%
   +
   Fortified Tobramycin 1.3%

2. Atropine eye drops : Supportive therapy to relieve ciliary spasm.
3. Steroids are contraindicated : They promote epithelial thinning.
4. In cases of impending perforation :
   - Oral Doxycycline.
   - Soft bandage contact lens (BCL).
   - Cyanoacrylate glue.

Fungal corneal ulcer 60c6b3eeaa8ded0e4e7e5ea7 00:32:58

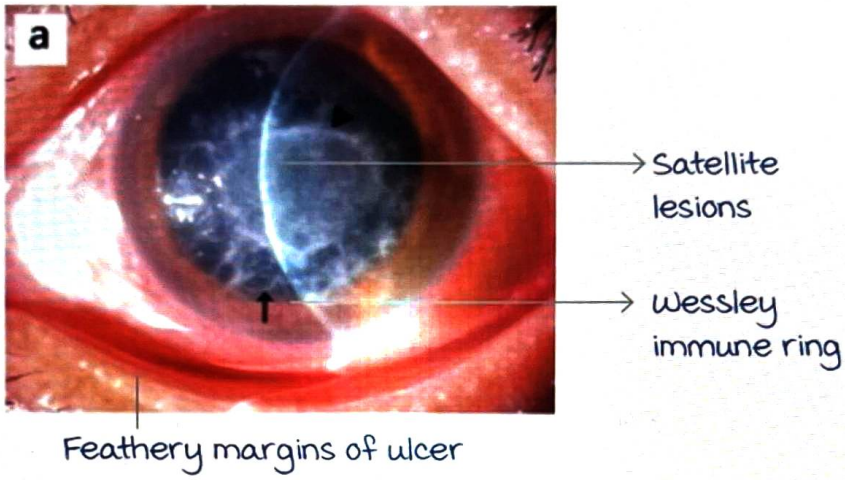
most common fungi : Aspergillus (Filamentous fungi with septate hyphae).

Patient gives history of trauma with vegetative material.
Eg : A 52 year old farmer from Rajasthan gives history of injury with sugar cane 5 days back.
Signs > > > Symtoms.

Active space

Signs :

a. Dry looking ulcer.
b. Feathery margins of ulcer. 60c6b3eeaa8ded0e4e7e5ea7
c. Satellite lesions.
d. Wessely immune ring.
e. Hypopyon : Immobile/ thick pus
Asterile.



Treatment :

1. Antifungals :
   a. Natamycin 5% eye drops (DOC in Aspergillus, Fusarium)
   b. Amphotericin B 0.15% drops
2. Atropine : Supportive therapy
3. Steroids are contraindicated.

Acanthamoeba keratitis 00:41:34

Risk factors :

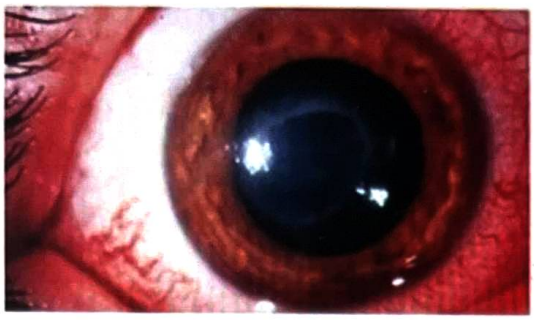
a. Contact lens users : With contaminated solution /if contact lens is washed with tap water.
b. Swimming pool.

Symptom : Severe pain

Clinical features : Symptoms > > > Signs

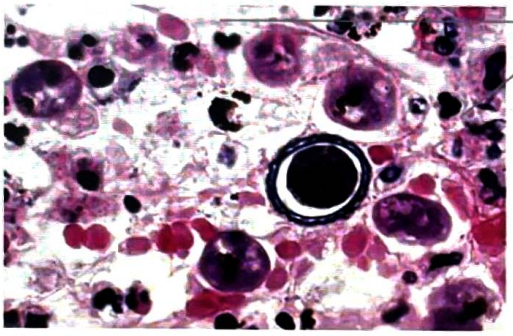
Signs :

1. Pseudodendritic Keratitis.
2. Radial Keratoneuritis.
3. Ring abscess.



Diagnosis :

a. Gram staining



Double walled acanthamoeba cysts

b. Culture media : Non-nutrient agar enriched with E.coli.

Treatment :

1. DOC : Polyhexa methylene biguanide (PHMB) 0.02%.
2. Chlorhexidine 0.02%

Inhibits membrane synthesis

3. Propamidine : Inhibits DNA synthesis

Viral keratitis

00:48:43

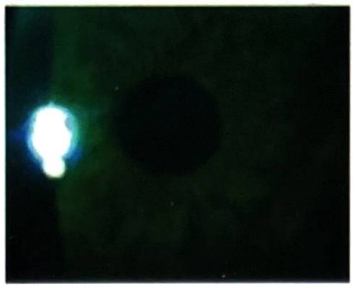
Herpes Simplex Virus (HSV) :

- Primary infection : Conjunctivitis
- Recurrent : Keratitis

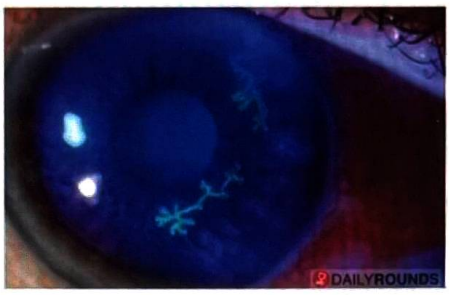
Clinical features :

1. Epithelial lesions :
   a. Superficial punctate (dot like) keratitis :

Active space



b. Dendritic ulcer with terminal buds :



- Differential diagnosisc :
  a. Acanthamoeba Keratitis
  b. HZV Keratitis.
  c. Tyrosinemia.
  d. Contact lens overuse.

c. Geographic ulcer :

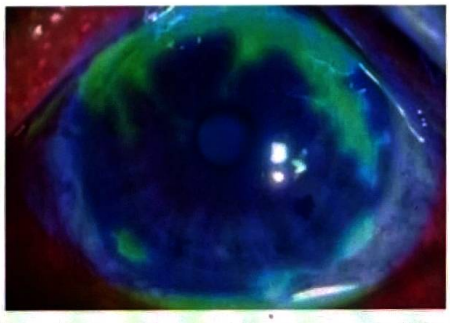
Treatment of epithelial lesions :

a. 3% Acyclovir eye ointment 5 times/day x 10 – 14 days.
b. Steroids are contraindicated.

All epithelial lesions are due to active viral replication.

2. Stromal Keratitis :

a. Immune stromal Keratitis :



Due to Type 3 hypersensitivity.
Almost always occurs after epithelial Keratitis.

Treatment : 3% Acyclovir eye ointment

+

Topical steroids.

b. Necrotising stromal Keratitis.

3. Endothelial lesion :

d. Disciform Keratitis :

Disc shaped areas of deep stromal/ endothelial edema.

Type 4 hypersensitivity reaction.

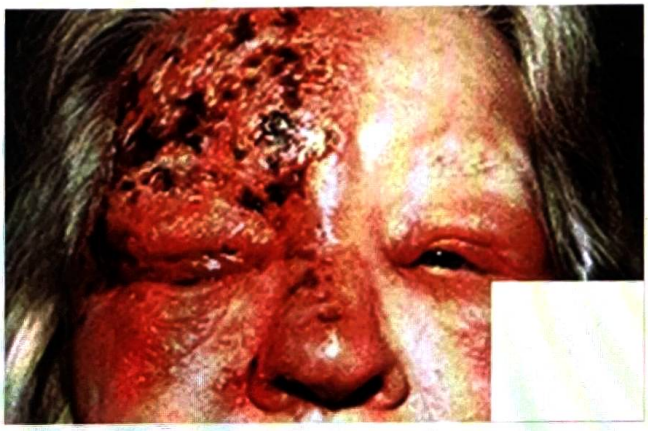
Hallmark of HSV Keratitis : Corneal anesthesia

↓

Metaherpetic Keratitis/ Neurotrophic ulcer due to 5th cranial nerve palsy.

Herpes zoster ophthalmicus

01:01:28



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Also Known as Shingles.

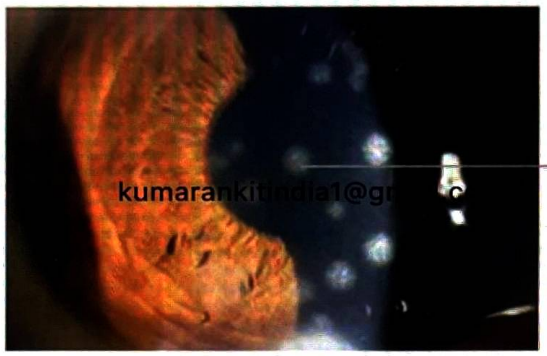
Usually affects dermatome supplied by Ophthalmic branch of 5th cranial nerve.

Active space

Hutchinson's sign : If the rash affects the tip of nose, the chances of HZO are increased.

Clinical features :

1. Pseudo-dendritic ulcer : No terminal buds.
2. Nummular Keratitis : Coin shaped subepithelial lesions.



3. Mucous plaque Keratitis : Chronic cases.
4. Stromal Keratitis
5. Cranial nerve palsies (most common affected : 3rd CN)
6. Least common : Scleroketatits.
7. Post herpetic neuralgia :
   Even after one month of resolution of HZO, pain persists (worse at night).

   Treatment : Tab. Acyclovir 800 mg (5 times a day) x 2 weeks

Active space

CORNEA - CORNEAL DYSTROPHIES, KERATOCONUS & MISCELLANEOUS

Corneal dystrophies

00:00:24

They are primary corneal diseases : Present in 1st & 2nd decade.

They are non-inflammatory.

They are progressive.

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They are bilateral.

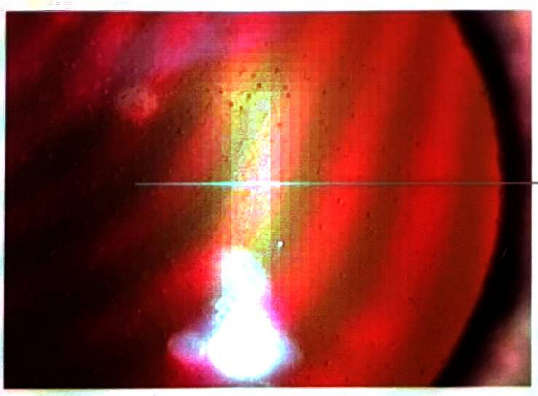
Inheritance : Autosomal dominant.

Types of corneal dystrophies :

Epithelial	
C – Cogan's epithelial basement membrane dystrophy/Map Dot	***Most Common corneal dystrophy***
M – Meesmann epithelial dystrophy	*(Nonprogressive, due to Keratin gene mutation*
L – Lisch epithelial dystrophy	AD/***XLD***
Bowman layer	
R – Reis Bucklers dystrophy/CDB I / GCD III	Fish Net
S – Schnyder central crystalline dystrophy	Corneal Lipid metabolism disorder
T – Thiel Behnke dystrophy/CDB II	Honeycomb pattern of opacities

Stromal	
L – Lattice corneal dystrophy I/Biber Haab Dimmer	neuropathy seen
Lattice corneal dystrophy II/Finnish	70 to 90 years
Lattice corneal dystrophy IIIA	***AR***, Mulberry like appearance
G – Gelatinous Drop like dystrophy/Japenese	Amyloid deposit – stained by Congo Red
G – Granular corneal dystrophy I/Groenouw	**Hyaline deposit – stained by Masson Trichrome stain^**
Granular corneal dystrophy II	Hyaline + Amyloid deposit
M – Macular dystrophy/Groenouw II	***AR*, Least common corneal dystrophy^**
F – Francois central cloudy dystrophy	Inborn error of keratin sulphate metabolism
Endothelial	
F – Fuch's endothelial dystrophy	**Cornea Guttata – wart like excrescences**
P – Posterior Polymorphous corneal dystrophy	
C – Congenital hereditary endothelial dystrophy I	**Both have AR inheritance and have perinatal onset**
Congenital hereditary endothelial dystrophy II	

meesmann epithelial dystrophy :

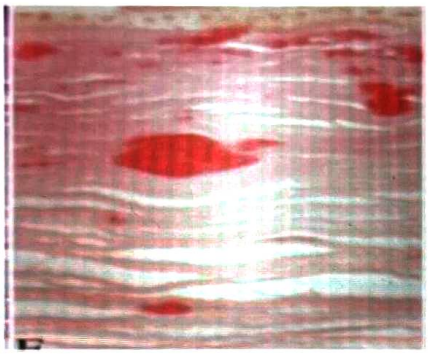


→ microcysts in the corneal epithelial

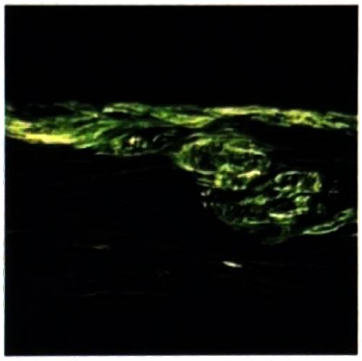
Active space

In lattice corneal dystrophy – type I :

PAS stain : Amorphous deposit

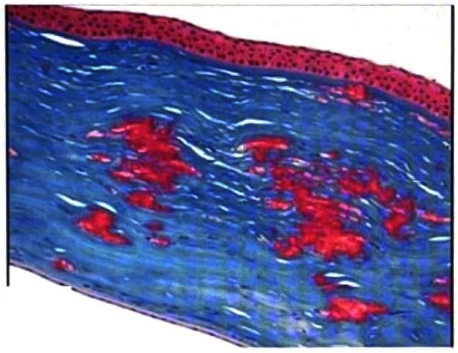


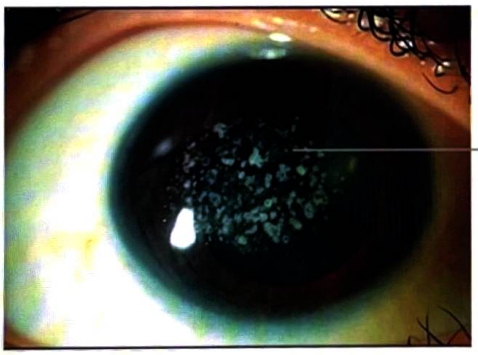
Congo red stain : Apple green birefringence



Granular dystrophy :

Masson trichrome stain : Hyaline deposits.

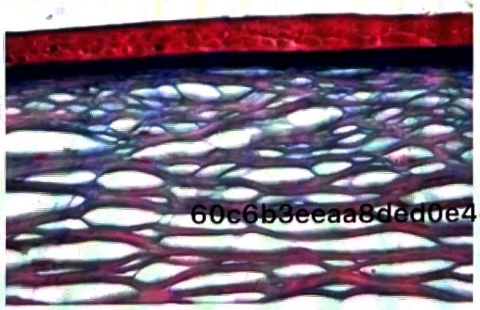




Bread crumb like granules → separated by clearly demarcated spaces in cornea

Macular dystrophy :

Acid mucopolysaccharidosis : Alcian blue stain.



Corneal dystrophies – Fuch's endothelia dystrophy

00:13:00

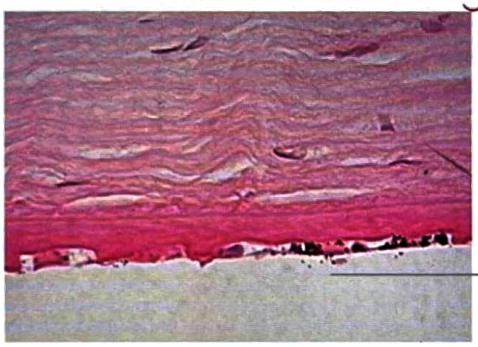
It is due to the mutation of COAX a gene.
Due to the mutation the descemet membrane and endothelium is damaged.

↓

Leads to wart like excrescences on cornea.

↓

Known as corneal guttae.



→ Corneal guttae

Causes of recurrent corneal erosions :

1. Cogan.
2. Thiel Behnke.
3. Reis buckler.
4. Lattice type I.

Disorders of metabolism :

1. Meesmann epithelial dystrophy.
2. Macular dystrophy.
3. Schnyder central crystalline dystrophy.

Treatment of corneal dystrophies :

If visual loss is present → Keratoplasty/ corneal transplantation.

Keratoconus

00:17:09

Keratoconus is a form of corneal ectasia.
It is non inflammatory, progressive, ectasia & usually B/L.
Etiology :

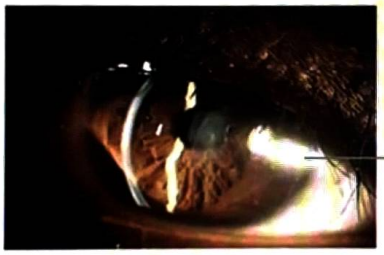
1. Congenital weakness.

2. Associated with : Trauma
VKC (vernal keratoconjuctivitis)
Down syndrome.

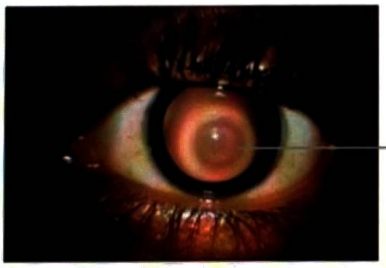
Clinical features of Keratoconus 60c6b3eeaa8ded0e4e7e5ea7

1. Myopia astigmatism.
2. Hallmark – stromal thinning (central/paracentral)
↓
Inferior to the centre.
3. Munson's sign : Bulging of lower eyelid on downgaze.
4. Rizzuti's sign : Arrowhead pattern of light, formed over nasal limbus when light is thrown from temporal side.

Munson's sign :
Bulged lower eyelid on down gaze



Rizzuti's sign :
Arrowhead pattern of light over nasal limbus



Oil droplet reflex

5. On retinoscopy : Scissoring reflex.
6. On ophthalmoscopy : Oil droplet reflex.
7. VOGT's striae : Stromal stress lines.

Keratoconus – Investigation and treatment 00:27:59

IOC : Corneal topography.

Treatment for Keratoconus :

A. Rigid contact lens (may lead to opacity).

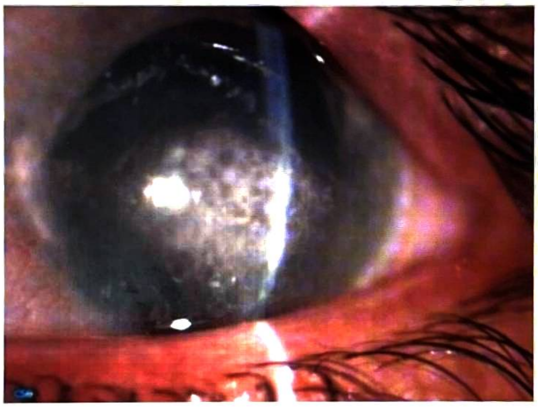
B. Keratoplasty.
C. C3R (corneal collagen cross linking using riboflavin & UVA radiation).

Band shaped keratopathy and Arcus senilis

Band shaped Keratopathy :

It is due to the deposits of calcium in Bowman's layer and stroma (superficial).

- Cause in adults : Uveitis.
- Cause in children : Still's disease.
- Treatment : Chelation with EDTA.



Arcus senilis

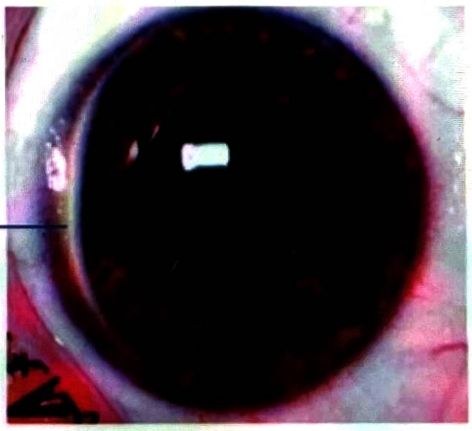
It occurs in old age.

It is due to the lipid deposition on Descemet's membrane near periphery.

It Involves stroma.

Lucid interval of VOGT (clear zone of cornea between arcus and limbus).

Lucid interval of VOGT



Interstitial keratitis

00:34:07

Inflammation only of corneal stroma.

Causes :

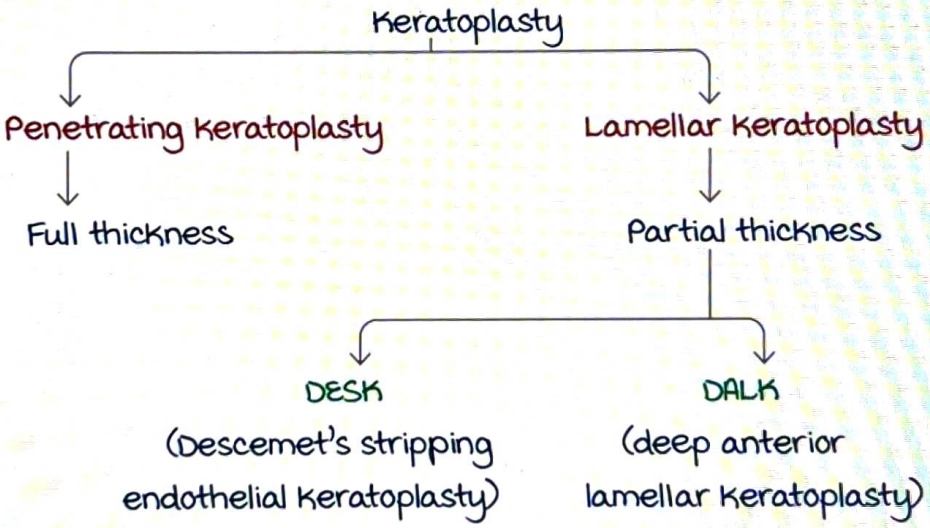
- Congenital syphilis.
- Cogan syndrome (interstitial keratitis + deafness)
- Tuberculosis.
- Herpes simplex virus [HSV].

Treatment : Steroids.

Keratoplasty

00:35:28

Commonly known as corneal transplantation.



DALK :

Removal of corneal tissue from epithelium upto deep stroma (i.e., anterior 3 layers).

Indicated in keratoconus with acute hydrops.

DESK :

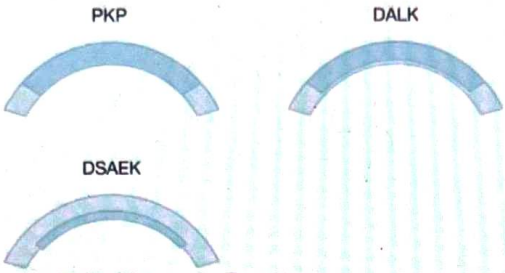
Removal of only endothelium and Descemet's membrane (i.e., posterior 2 layers).

Penetrating keratoplasty :

Indicated in pseudophakic bullous keratopathy.

Removal from epithelium to endothelium (i.e., all 5 layers).

Keratoplasty Techniques



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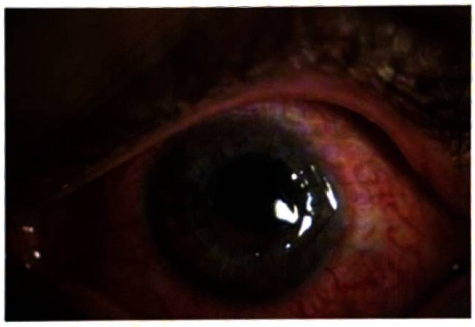
Procedure of Keratoplasty :

The host cornea is cut using a trephine (diameter : 7.5 mm).

Implant the graft cornea, 0.25mm larger than host opening.

Trephine

Post Keratoplasty



Graft rejection :

MC rejection : Endothelial rejection.

Endothelial rejection diagnosed by : Khodadoust line.
(Linear Keratic precipitates)

Storage media for corneal transplantation :

1. Short term (<48 hours) : 4°C, moist chamber.
   Short term (48–72 hours) : MC Carey Kauffman medium.
2. Long term (upto 35 days) : Cryopreservation (–70°C).

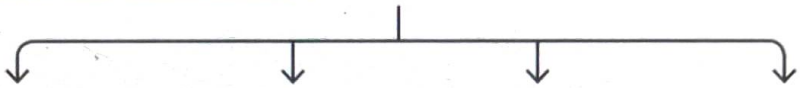
Active space

UVEA - ANTERIOR UVEITIS

Classification of uveitis

00:01:49

Anatomical classification :



Anterior Uveitis :

- Also known as Iridocyclitis.

Iritis (iris) + cyclitis (Pars plicata)

Intermediate Uveitis :

- Also known as Pars planitis/ Posterior cyclitis.

Involvement of

- Peripheral retina : Basal retinochoroiditis.
- Vitreous : Vitritis/ hyalitis.

Posterior Uveitis :

- Also known as Chorioretitnitis/ Retinochoroiditis.

Panuveitis

Clinical classification :

- Acute : < 3 months.
- Chronic : > 3 months.

  +

  Relapse < 3 months.
- Recurrent : ≥ 3 months between 2 episodes.

Classification based on causes :

- Granulomatous.
- Non-granulomatous.

Anterior uveitis: Symptoms

00:08:36

Involves inflammation of iris and pars plicata.

Symptoms :

- Pain : Referred along branches of 5th nerve.
- Redness.
- Blurring of vision : Due to
  a. Corneal haze.
  b. Aqueous turbidity.
- Photophobia : Severe.
- Lacrimation.

Anterior uveitis: Signs 00:11:25

1. Circumcorneal congestion.
2. Cornea :
   - Raised IOP/ Toxins : Corneal haze.
   - Keratic precipitates (KPs) :
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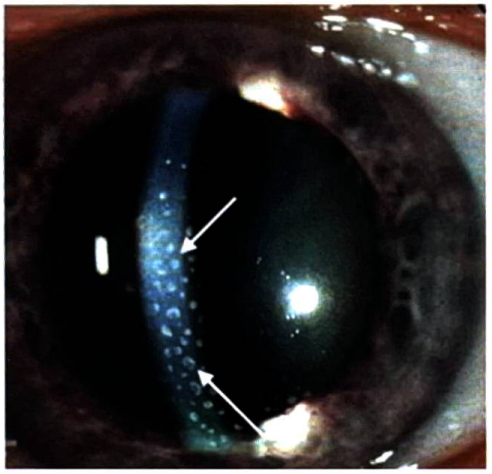
     Cellular deposits on the corneal endothelium.
     Most common : Inferiorly in an imaginary area known as Arlt's triangle.

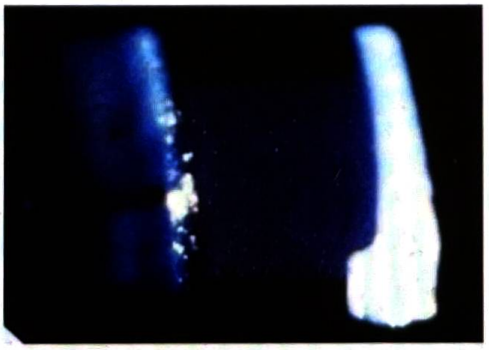


Types of KPs :

1. Mutton fat KPs : Macrophages.
   Granulomatous uveitis.
2. Fine granular KPs : Lymphocytes.
   Non-granulomatous uveitis.
3. Pigmented KPs : Chronic non-granulomatous uveitis.
4. Stellate KPs : Star shaped, diffused distribution.
   Seen in herpetic uveitis, toxoplasmosis, Fuch's Hetero chromic Iridocyclitis (FHI).

Anterior uveitis : Anterior chamber signs 00:21:27

3. Anterior chamber signs :



1. Aqueous cells :
   - They are markers of disease activity.
2. Aqueous flare :
   - Turbidity /translucency of aqueous due to leakage of proteins.
   - Visible due to Tyndall effect /Brownian movement.
3. Hypopyon :
   - Cause of Hypopyon associated with anterior uveitis : Behcet's disease

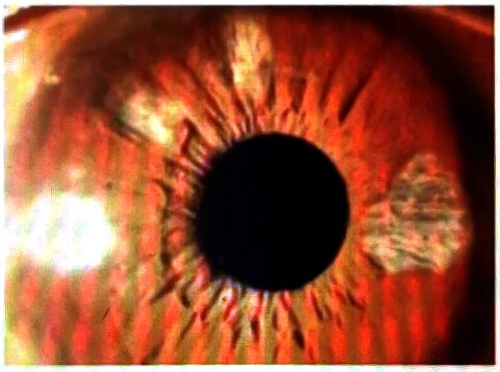
4. Hyphema :
   - Causes of Hyphema associated with anterior uveitis :
     Herpetic uveitis.
     Toxoplasmosis.
     Syphilis.

Anterior uveitis: Iris signs

00:28:04

Iris signs :

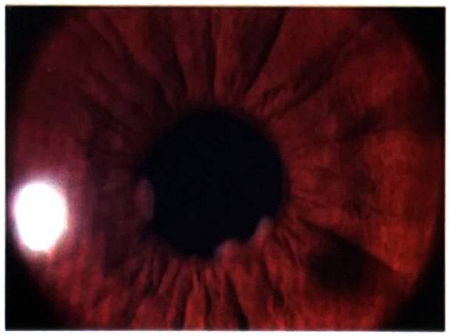
1. Pseudo-rubeosis : Neovascularisation of iris.
   Dilated iris vessels
   ↓
   Iris vessels become visible
   ↓
   They look like Rubeosis iridis.
2. Acute cases : Water logged iris (muddy iris)
3. Chronic cases : Iris atrophy :
   - Sectoral : Herpetic uveitis.
   - Diffuse : FHI.



Sectoral iris atrophy

4. Iris nodules :

- Koeppe nodules : at Pupillary margin

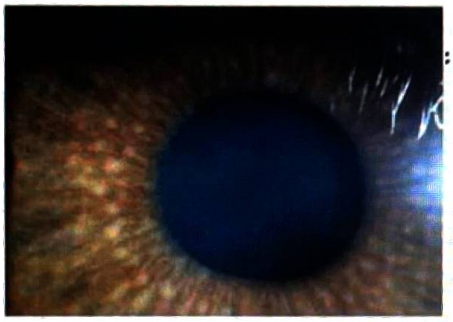


Found in granulomatous and non-granulomatous uveitis.

- Busacca nodules

at Iris Stroma

Found in granulomatous uveitis.



- Berlin nodules : Angle of Anterior chamber

Anterior uveitis: Pupil signs and IOP 00:34:05

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Pupil signs :

1. Acute cases : Miosis.
2. Chronic cases : Festooned pupil :

Exudation leads to the formation of incomplete posterior synechiae (adhesion between pupillary margin and lens).

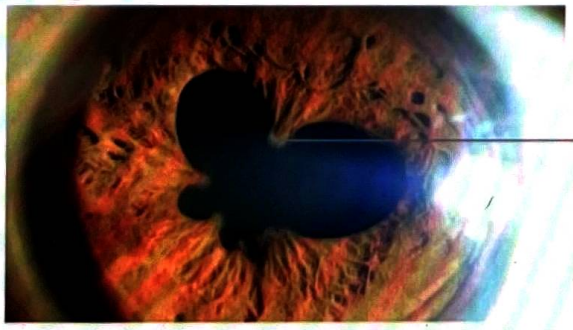
↓

Dilate the pupil.

↓

Pupil dilates irregularly (does not dilate at sites of posterior synechiae formation)

3. Recurrent cases : Fixed pupil.



Active space

- Annular/ Ring synechiae (Synechiae over 360^0)

↓

Pupil completely in adhesion with lens : Fixed

↓

Aqueous not able to flow from posterior chamber to anterior chamber.

↓

Secondary angle closure glaucoma.

4. Total posterior synechiae :

- Due to exudates pouring in the Posterior chamber.

↓

Adhesion of complete posterior surface of iris with lens.

- Cyclitic membrane : Due to exudates pouring behind the lens which cover the ciliary processes and posterior surface of the lens.

↓

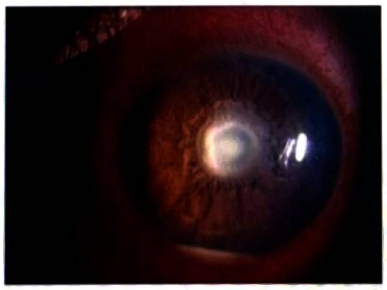
No aqueous secretion.

↓

Decrease in IOP known as hypotony.

5. Occlusio pupillae :

- Exudates get organised across the pupil, blocking the pupil.60c6b3eeaa8ded0e4e7e5ea7



IOP :

1. Acute cases :
   - Raised IOP due to Trabeculitis and clogging of Trabecular meshwork by inflammatory cells : Secondary open angle glaucoma.
2. Chronic cases :
   - Cyclitic membrane + shut down of ciliary body leads to reduced IOP.

Active space

Complications

00:48:45

1. Most common : Complicated cataract.
2. Most common in recurrent cases : Secondary angle closure glaucoma.
3. Band shaped Keratopathy.
4. Most common cause of visual loss due to posterior involvement in anterior uveitis : Cystoid macular edema.

Treatment :

1. Steroids (Topical)
   - Prednisolone.] Taper them
   - Difluprednate]

   +
2. Cycloplegics (Atropine)
   - Relax ciliary muscle to relieve pain.
   - Break any posterior synechiae formed.
   - Prevent formation of posterior synechiae by keeping pupil mobile.
3. Antimetabolites
   - If inflammation; recurrent episodes requiring long term steroids.

     Side effects of steroids:
     - Galucoma – Topical
     - Cataract – Systemic

Anterior uveitis: Causes

00:56:15

Causes:

1. Granulomatous Uveitis
   a. TB.
   b. Leprosy :
      - Most common : LL (Lepromatous).
      - Pathognomic feature : Iris pearls.
   c. Sarcoidosis :
      - It causes anterior uveitis as : Lofgren syndrome (Erythema nodosum + Bilateral hilar

lymphadenopathy + Anterior uveitis).
Heerfordt syndrome/ Uveoparotid fever.

- Most common ocular manifestation of sarcoidosis : Granulomatous anterior uveitis.

d. Syphilis :

- Anterior uveitis is most common in late congenital syphilis.
- Iris roseolas.

e. Herpes :

- Anterior uveitis + raised IOP + Hyphema + stellate KPs + Sectoral iris atrophy

2. Non-granulomatous Uveitis :

a. Acute

(i) Most common cause : Idiopathic

(ii) HLA B27 associated diseases :

- Ankylosing spondylitis : Lower back pain + stiffness + unilateral anterior uveitis.
- Reiter's disease : History of travel and infection with shigella. Urethritis + Conjunctivitis + Arthritis + U/L anterior uveitis + Keratoderma blennorrhagicum + Circinate balanitis.
- Psoriatic arthritis : Sausage digits + B/L anterior uveitis.

(iii) Behcet's disease :

- Associated with HLA B5, B51

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- B/L recurrent anterior uveitis + Cold mobile hypopyon + Aphthous mouth ulcers.

(iv) Inflammatory bowel disease :

- Ulcerative colitis (12%) > Crohn's disease (2.4%)

(v) Drug induced uveitis :

Mnemonic : CML Please Relapse Slowly
Cidofovir.
metopranolol.
Latanoprost,
Pilocarpine.
Rifabutin.
Sulfonamides.

Chronic non-granulomatous uveitis : Causes 01:10:20

1. Juvenile Idiopathic Arthritis (JIA) :
   - most common cause of anterior uveitis in paediatric age group.
   - 3 types :

Pauciarticular
- ≤ 4 joints involved.

Polyarticular
- > 4 joints involved.

Still's disease.

Type 1
- Girls
- RF negative (seronegative)
- ANA positive
- most commonly associated with Anterior uveitis.

Type 2
- Boys.

2. Fuch's Heterochromic Iridocyclitis (FHI) :
   - Clinical features :

     Mnemonic : NO F U C H S

     No posterior synechiae.

     Floaters : Only symptom

     Unilateral.

     Cataract (>80% cases).

     Heterochromia iridis : Hypochromia in the affected eye (due to loss of iris crypts).

     Stellate Keratic precipitates.
   - Amsler's sign : Bleeding on paracentesis.

UVEA - INTERMEDIATE, POSTERIOR, PAN-UVEITIS & MISCELLANEOUS

Intermediate uveitis

00:00:07

Also known as pars planitis.

Causes :

1. Most common : Idiopathic.
2. Associated with :
    - Lyme's disease .
    - Sarcoidosis.
    - Multiple sclerosis.
    - TB.

Symptoms : B/L Floaters and blurring of vision.

Signs :

1. Spillover anterior uveitis.
2. Snowballs : Aggregation of cells in vitreous.
3. Snow banking : Fibrovascular plaque.
4. Cystoid macular edema : Most common cause of vision loss.

Treatment :

1. Steroids : Posterior subtenon injection

↓

Fails

↓

2. Steroids : Systemic

↓

Fails

↓

3. Immunomodulatory drugs (Azathioprine)

↓

Fails

↓

4. Pars plana Vitrectomy + Photocoagulation of the snow bank.

Posterior uveitis

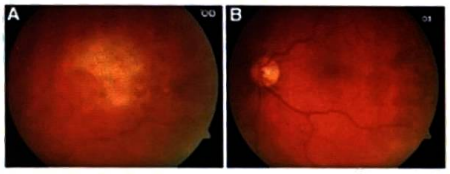
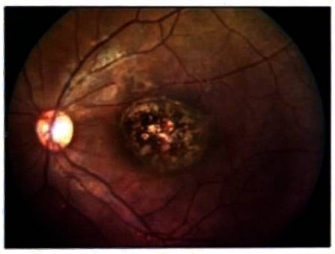
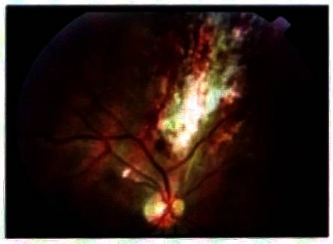
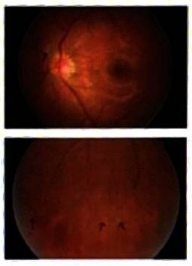
00:10:00

Inflammation of Choroid and Retina, also known as Chorioretinitis.

Clinical features :

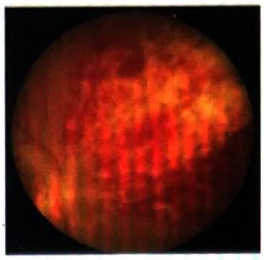
1. Patient complains of Floaters
2. Painless loss of vision.
3. Scotoma.
4. Metamorphopsia .
5. Photopsia.

Causes :

Infectious	Non-infectious
1. Most common : Toxoplasmosis a. Active : Headlight in fog appearance (due to associated active vitritis).  b. Chronic : Punched out scar.  • Cats are definitive hosts. • Toxoplasmosis causes focal chorioretinitis. 2. Toxocariasis : • Unilateral LOV. • Leukocoria (white pupillary reflex). 3. CMV retinitis : • Most common cause of LOV in ocular HIV. a. Pizza pie appearance. b. Scrambled egg & Ketchup appearance : 	1. Sarcoidosis : • Candle wax dripping appearance (due to perivascular inflammation and sheathing).  • Lander's sign : Pre-retinal nodules 2. Behcet's disease. 3. Birdshot chorioretinopathy : • HLA A29 association. 4. Serpiginous choroiditis : • HLA B7 association.

Active space

c. Brushfire extension :



4. TB

Focal chorioretinits : Toxplasma, toxocariasis, CMV.

multifocal chorioretinitis : HSV, TB, Syphilis.

Treatment :

1. Infectious cases: Treat the cause

+

Systemic steroids.

2. Non-infectious cases : Systemic steroids

or

- Adalimumab : Visual studies I-II.
- Sirolimus : Sakura program.
- Voclosporin : Luminate trials.

Panuveitis

00:24:03

1. Vogt-Koyanagi-Harada (VKH) Syndrome :
   - It affects :

Ocular	Ears	Skin	Meninges.
• Bilateral, Granulomatous panuveitis.	• Tinnitus.	• Alopecia • Vitiligo	• meningismus • CSF pleocytosis

   - Sunset glow fundus
   - Suiguira sign : Perilimbal vitiligo.
   - Exudative Retinal detachment.

2. Sympathetic ophthalmitis :

- Ocular clinical feature : Granulomatous panuveitis.
- Cause : Accidental penetrating trauma affecting the ciliary body.
- Pathogenesis : Trauma to one eye (Exciting eye)

↓ >2 weeks

Sympathetic ophthalmitis in other non-traumatic eye (Sympathising eye)

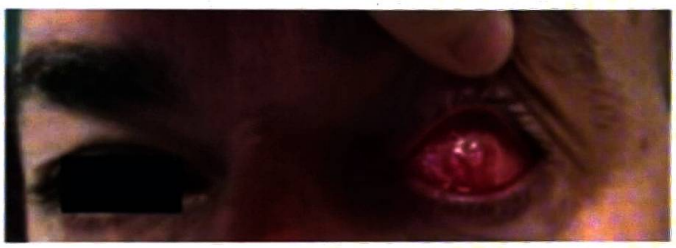
- Prevention of Sympathetic ophthalmitis in non-traumatic eye : Enucleation of traumatic eye within 14 days.
- Treatment : Steroids.

Miscellaneous

00:35:18

Surgical removal of eye :

- Enucleation :



Removal of the whole eyeball along with optic nerve.

Indications : Retinoblastoma.
Trauma.

Contraindication : Panophthalmitis.

- Evisceration :

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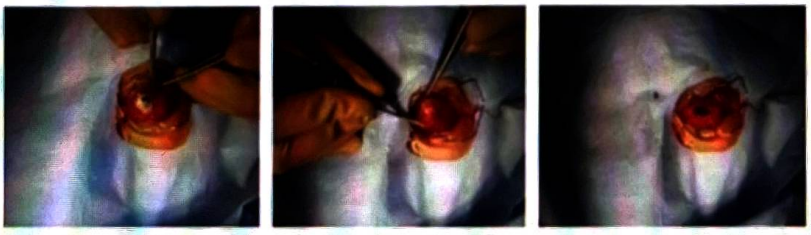
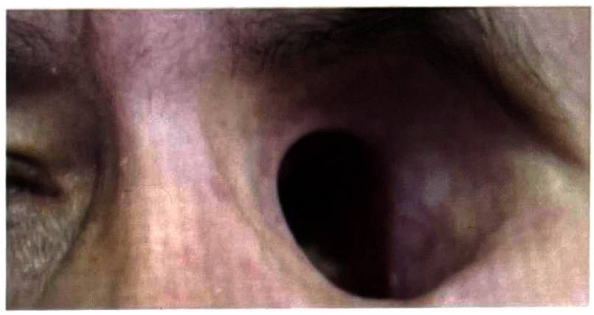


Removal of all layers of the eyeball except sclera.

Indications : Endophthalmitis.

Contraindication : Retinoblastoma.

- Exentration :



Removal of the eye and orbital contents.
Done only in orbital mucormycosis/ metastatic tumors to orbit.

Gyrate atrophy

00:39:50

Mnemonic : GO AIR.

Gyrate atrophy.

Ornithine aminotransferase (gene mutation) : Increase levels of Ornithine.

Treatment : Arginine restriction In diet (Ornithine is synthesised from arginine).

Autosomal Recessive.

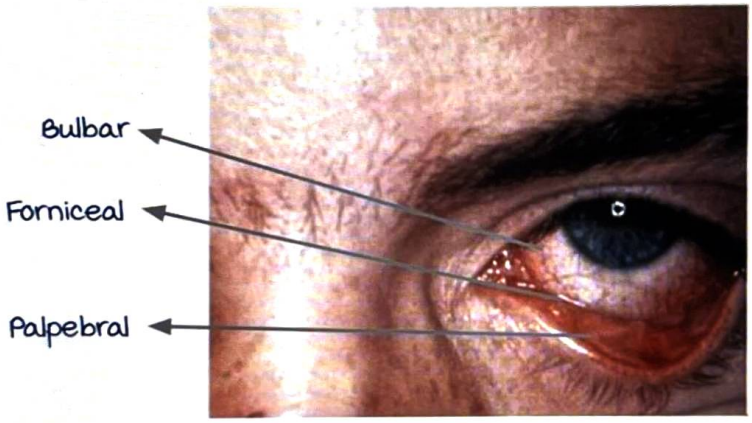
CONJUNCTIVA

Parts of conjunctiva 00:00:20

Conjunctiva is transparent/translucent mucous membrane.
There is no conjunctiva over the cornea.

Three parts :

1. Bulbar conjunctiva : Covers the sclera.
2. Palpebral conjunctiva : Lines the inner surface of eyelids.
3. Forniceal conjunctiva : Shallow depression between palpebral and bulbar conjunctiva.



There are two specialized folds :

- Plica semilunaris : Represents the nictitating membranes.
- Caruncle : Contains hair follicles.

10. Plica semilunaris.
11. Caruncle.

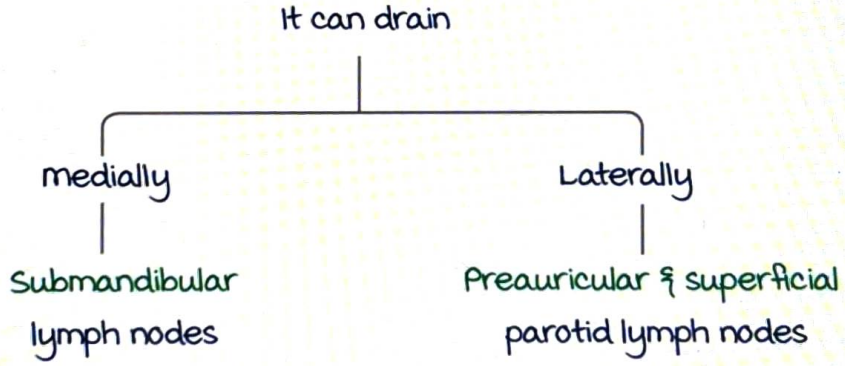
Histology of conjunctiva

1. Outermost layer :
   Epithelium (non-keratinised stratified squamous)
   It contains : 1. Goblet cells, which secrete mucin on parasympathetic stimulation (mucin forms the innermost layer of tear film).
   2. Melanocytes.
   3. Langerhans cells.

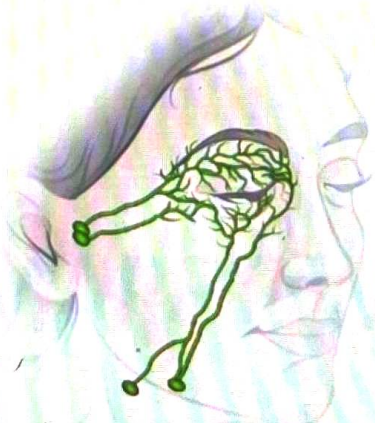
2. Adenoid/lymphatic layer : Develops after 3 months of age.
3. Fibrous layer.

Lymphatic drainage of conjunctiva 00:06:09

The lymphatic drainage in the eye occurs only from the conjunctiva.



Lymphatic drainage of conjunctiva



Conjunctivitis

00:09:07

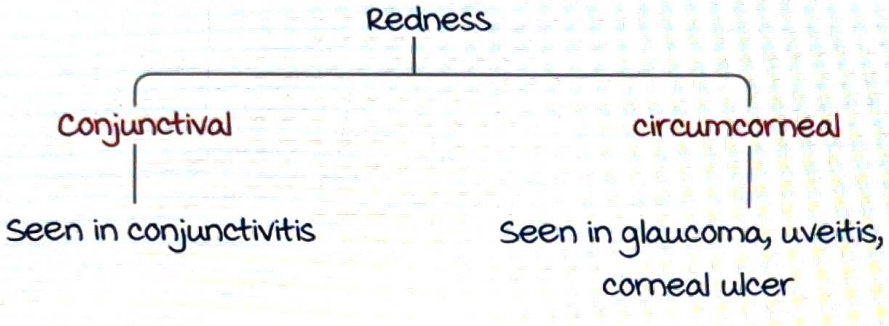
Eye flu symptoms :

Redness, stickiness, foreign body sensation, lacrimation, itching (allergic).

If pain and loss of vision present, there is corneal involvement.

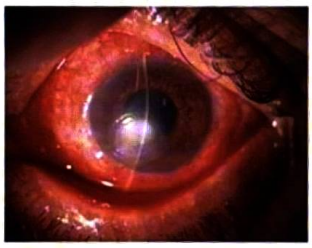
Signs :

1. Congestion/hyperaemia :



Conjunctival

Circumcorneal



2. Subconjunctival haemorrhage :

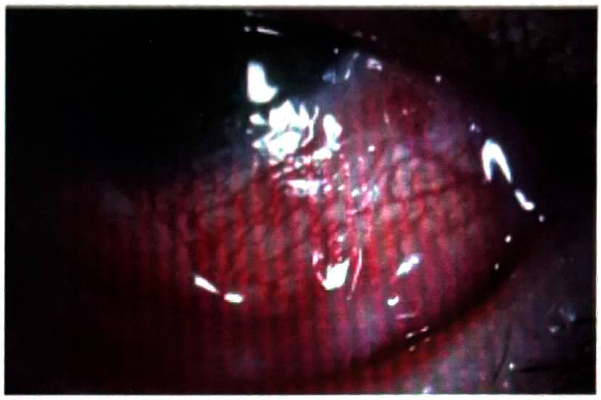
No treatment required.

Resolves spontaneously in 2 weeks.

Hemorrhage

3. Chemosis

It is swelling of the bulbar conjunctiva.

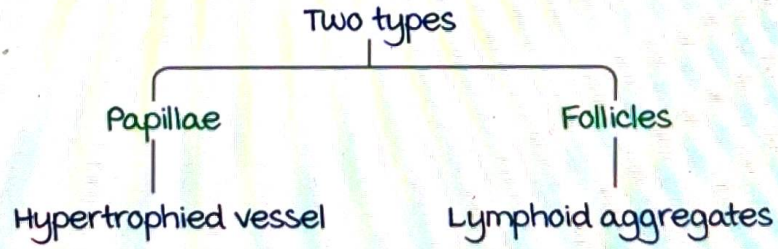


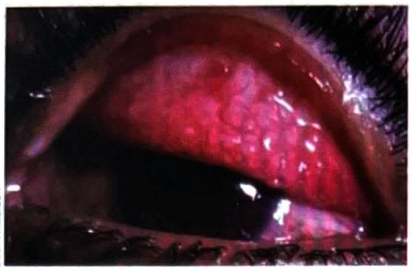
4. Discharge

It can be mucopurulent or watery. It leads to stickiness and coloured halos.

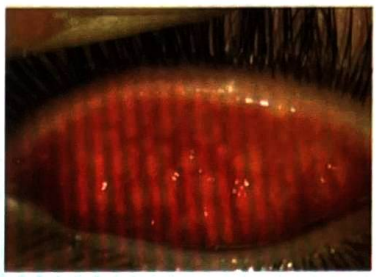
Inflammatory reactions of conjunctiva

00:14:31





Papillae



Follicles

Discharge	Papillary reaction	Follicular reaction
mucopurulent	Bacterial conjunctivitis	Chlamydia conjunctivitis
watery	Allergic conjunctivitis	Viral conjunctivitis

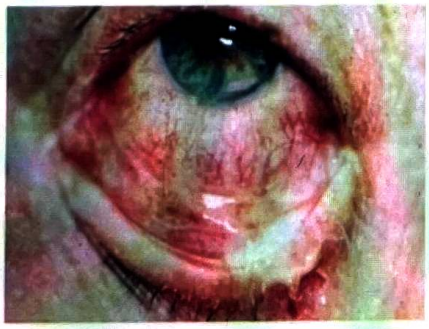
Bacterial conjunctivitis

1. Acute mucopurulent conjunctivitis :

Most common cause : Staphylococcus aureus.
Treatment : Topical antibiotics.

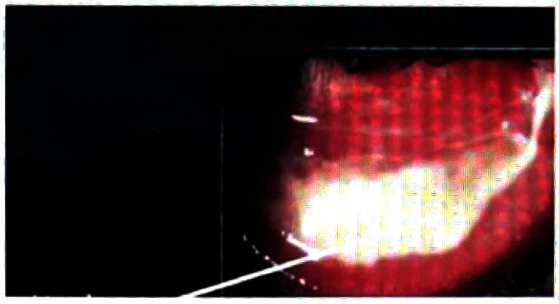
2. Acute purulent conjunctivitis/ hyperacute/ blennorrhea :

Most common cause : Neisseria gonorrhoeae (oculogenital transmission)
C/f : Copious discharge.
Swelling of eyelids (overhanging).
Intense pain.
Preauricular lymphadenopathy.
Treatment : Inj. ceftriaxone 1g single dose, followed by
Oral erythromycin for 2 weeks.



3. Acute membranous conjunctivitis :

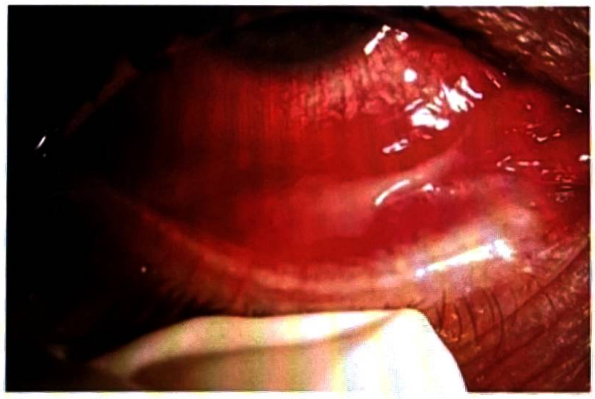
most common cause in non immunized children :
C. diphtheria
most common cause in adults/overall : Pneumococcus.
The membrane is fused with the underlying epithelium and it bleeds on removal.



Bleeds on removal
Firmly adherent

4. Acute pseudomembranous conjunctivitis :

The membrane does not fuse with underlying epithelium and does not bleed on removal.



Peeling no bleeding

5. Angular conjunctivitis/ diplobacillary conjunctivitis:

most common cause Moraxella lacunata/axenfeld
C/F : Excoriation of skin at medial canthi.
Reddening of intermarginal strip.
Blepharitis.

Treatment : Tetracycline 1% eye ointment for 2 weeks.
Zinc boric eye drops.

Viral conjunctivitis

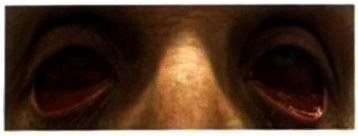
00:28:38

1. Adenoviral conjunctivitis :
   - Non-specific follicular conjunctivits : Type 1-11 and 19.
   - PCF (phayngoconjunctival fever) : serovar 3, 4,7 : Presents with preauricular lymphadenopathy.
   - EKC (epidemic keratoconjunctivitis) : serovar 8, 19, 37, associated with preauricular lymphadenopathy

2. Apollo conjunctivitis/ acute haemorrhagic conjunctivitis :

Causes are :

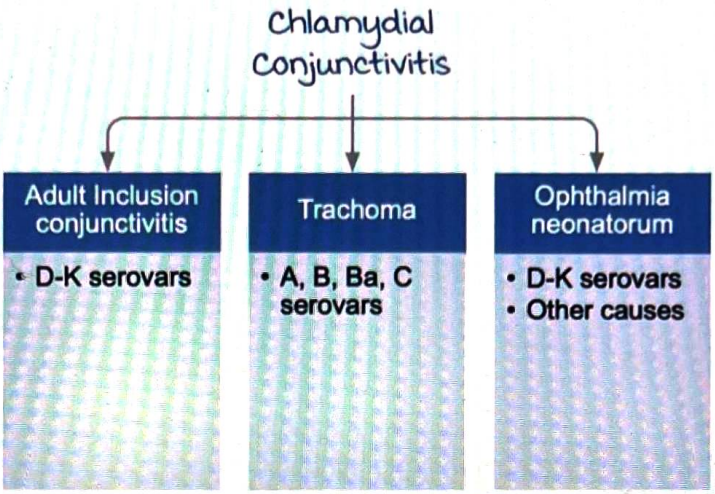
1. MCC : Picornavirus :
   Enterovirus type 70.
   Coxsackie A24.
2. Adenovirus type 11



3. Molluscum contagiosum :

C/F : Unilateral,painless node with umbilicated appearance.

Chlamydia conjunctivitis 00:33:55



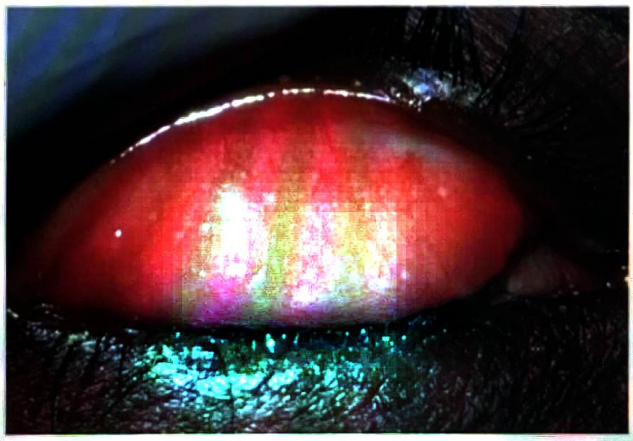
Trachoma :

It is also known as Egyptian ophthalmia.
It is caused by Chlamydia trachomatis A, B, Ba, C serovars.
Transmission : Fingers, flies, fomites.
Most common in children < 10 years of age.

Clinical signs of trachoma 00:38:22

It is type IV hypersensitivity reaction and active inflammation with conjunctival scarring present together.

1. Sago grain follicles :
   Most commonly seen in upper palpebral conjunctiva
   These follicles show necrosis and Leber cells.



Active space

2. Cicatricial sign

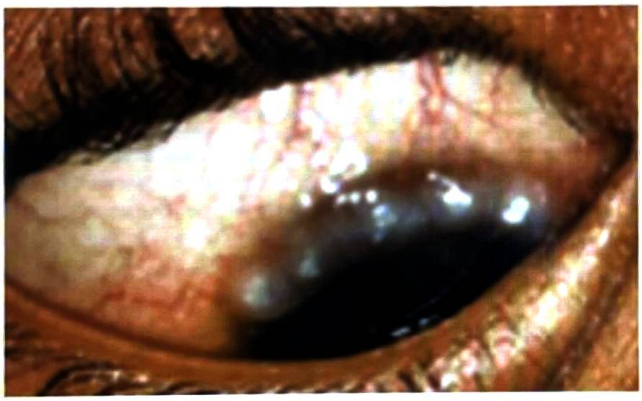
Arlt's line : Line of cicatrization present between the lower 2/3rd and upper 1/3rd.

Arlt's Line

3. Herbert pits :

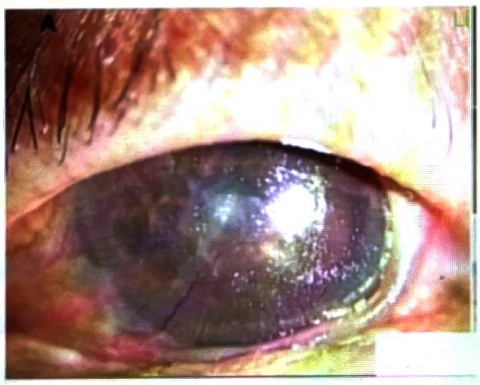
It is pathognomonic for trachoma.

It is due to healing of limbal follicles.



4. Pannus :

It is vascularisation of cornea superiorly.



Pannus

Only complication : Corneal ulcer (due to trichiasis)

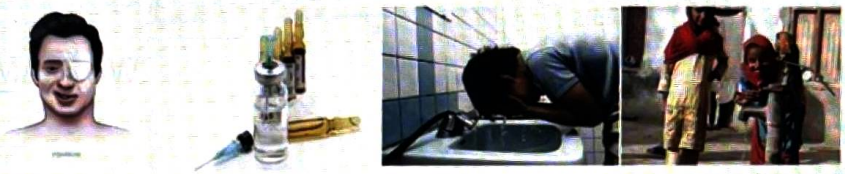
↓

Heals to form scar.

↓

Leads to loss of vision.

Treatment and classification of trachoma 00:45:25



Surgery
for inturned eyelids

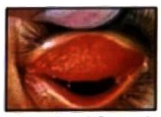
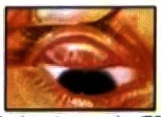
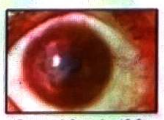
Antibiotics
Pfizer donated Zithromax to treat and prevent infection

Facial cleanliness
to prevent disease transmission

Environmental change
to increase access to water and sanitation

Indications for treatment

- If prevalence is >10% in children, mass prophylaxis is given to all children from 1 to 9 years of age
- If 5 – 10% , treatment is given to affected children and families.
- If < 5% , only facial cleanliness and environmental change is advised.

 Trachomatous inflammation-follicular (TF)	**Trachomatous Inflammation – Follicular (TF):** The presence of five or more follicles in the upper tarsal conjunctiva. Follicles are round swellings that are paler than the surrounding conjunctiva, appearing white, grey or yellow. Follicles must be at least 0.5mm in diameter.
 Trachomatous inflammation-follicular and intense (TI)	**Trachomatous Inflammation – Intense (TI):** Pronounced inflammatory thickening of the tarsal conjunctiva that obscures more than half of the normal deep tarsal vessels. The tarsal conjunctiva appears red, rough and thickened. There are usually numerous follicles, which may be partially or totally covered by the thickened conjunctiva.
 Trachomatous scarring (TS)	**Trachomatous Scarring (TS):** The presence of scarring in the tarsal conjunctiva. Scars are easily visible as white lines, bands, or sheets in the tarsal conjunctiva. They are glistening and fibrous in appearance. Scarring, especially diffuse fibrosis, may obscure the tarsal blood vessels.
 Trachomatous trichiasis (TT)	**Trachomatous Trichiasis (TT):** At least one eyelash rubs on the eyeball. Evidence of recent removal of inturned eyelashes should also be graded as trichiasis.
 Corneal Opacity (CO)	**Corneal Opacity (CO):** Easily visible corneal opacity over the pupil. The pupil margin is blurred viewed through the opacity. Such corneal opacities cause significant visual impairment (less than 6/18 or 0.3 vision), and therefore visual acuity should be measured if possible.

Classification of trachoma :

TF (Trachomatous Inflammation – Follicular) :

- Active disease, predominantly follicles.
- At least 5 or more follicles in upper palpebral conjunctiva.

TI (Trachomatous Inflammation – Intense) :

- Pronounced inflammatory thickening of upper palpebral conjunctiva obscures more than half of normal deep tarsal vessels.

TS (Trachomatous Scarring) :

- Presence of scarring in tarsal conjunctiva.
- Seen as white bands or sheets of fibrosis.

TT (Trachomatous Trichiasis) :

- When at least 1 eyelash rubs the ocular surface.
- Evidence of recently removed trichiatic eyelashes.

Corneal Opacity :

- Early visible corneal opacity present in pupillary area.
- Causes significant visual impairment.

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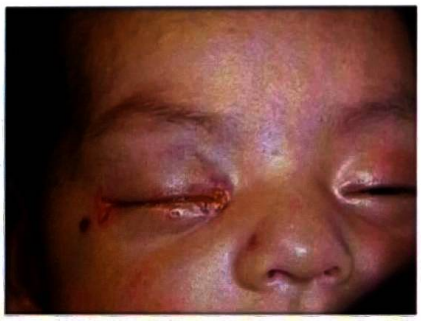
Ophthalmia neonatorum

00:48:05

It is conjunctivitis in neonates (< 28 days of onset).

Onset (hours after birth)	Cause
Within first 6 hours	CHEMICAL (Silver nitrate) conjunctivitis
24–48 hours	Neisseria gonorrhoeae
2–5 days	Other Bacteria (Gram positive more common than Gram-negative)
5–7 days	HSV-II
> 1 week	Chlamydia Trachomatis (D-K)



Prevention :

1. Crede's method : Topical 1% silver nitrate (not recommended anymore).
2. 0.5% erythromycin or 1% tetracycline : Given within 1 hour of birth.

Active space

Allergic conjunctivitis

00:52:20

It presents with a papillae reaction with watery discharge and itching.

Vernal Keratoconjunctivitis/spring catarrh :

It is type I hypersensitivity reaction (e.g., pollen).

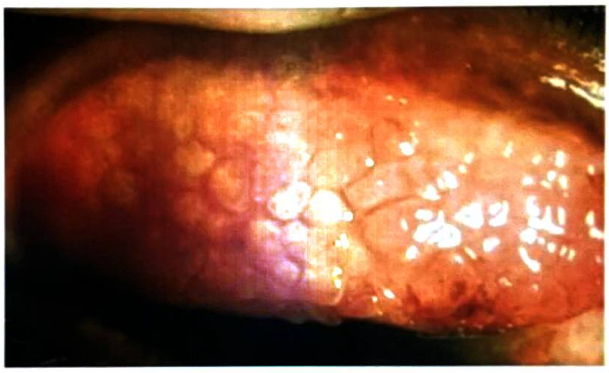
most common in boys (5–15 years).

 Present season in spring and summers.

History of atopy present.

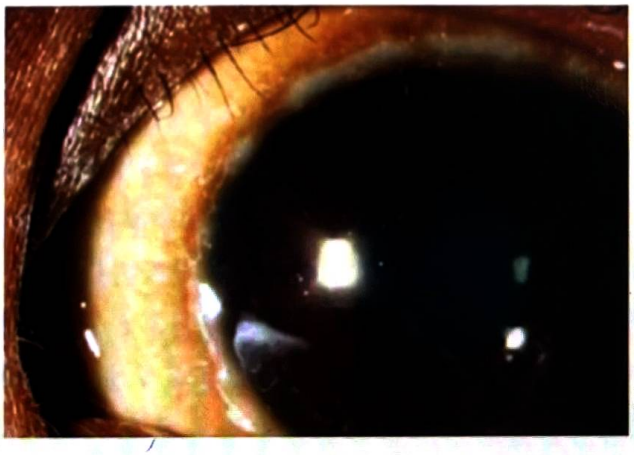
Clinical signs

1. Papillary hypertrophy : Cobble stone appearance.



2. Horner trantas dots :

It is a collection of eosinophils & epithelial debris on the limbus.



3. Pseudogerontoxon :

It is a paralimbal grey white band of lipid deposition seen in children.

4. Shield ulcer
5. Dennie morgan line : An extra lower lid crease
6. Maxwell Lyon sign : Ropy discharge.

The excessive itching : Leads to greater incidence of Keratoconus

Treatment of Vernal keratoconjunctivitis 01:02:20

1. Cold compression.
2. Topical anti-histamines.
3. Topical mast cell stabilizers.
4. DOC : Olopatadine or Alcaftadine.
5. Topical steroids in acute exacerbations.

Atopic Keratoconjunctivitis :

It occurs in old age 20-50 years.

Eyelids : Hertoghe's sign the lateral eyelashes are lost.

Giant papillary conjunctivitis :

It is > 1mm in diameter.

Type IV hypersensitivity reaction.

It is mechanically induced by : Contact lenses
Ocular prosthesis
Protruding sutures.

Treatment : Anti-histamines.

Phlyctenular keratoconjunctivitis 01:06:27

It is type IV hypersensitivity reaction.

most common in India : Tuberculosis.

most common in western countries : Staphylococcus aureus.

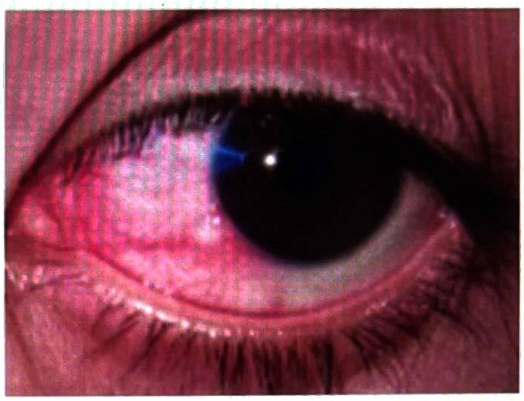
Clinical sign :

Phlycten : It is a nodule near the limbus, grows towards the cornea and ulcerates, giving rise to three types of

ulcer :

- Sacrofulous ulcer.
- Fascicular ulcer.
- Miliary ulcer.

Treatment : Steroids.



Pterygium

01:12:25

A triangular, fibrovascular growth of degenerative subconjunctival tissue over the cornea, destroying Bowman's and superficial stroma is called pterygium.
It is associated with UV ray exposure.
It is associated with dust, humidity.
It occurs also due to limbal stem cell deficiency.

Clinical signs :

1. Triangular growth with apex towards cornea.
2. Stocker's line : Deposition of iron in front of apex of pterygium.
3. Loss of vision : Due to corneal astigmatism.
   Growth encroaching on the visual axis (covers the pupillary area).

Pterygium

Treatment

Surgical excision : It has a high rate of recurrence.

To prevent recurrence :

- Topical mitomycin C.
- Autografting : Graft harvested from the same eye (usually superiorly).
  Sutures or fibrin glue or autologous serum are used to fix it.

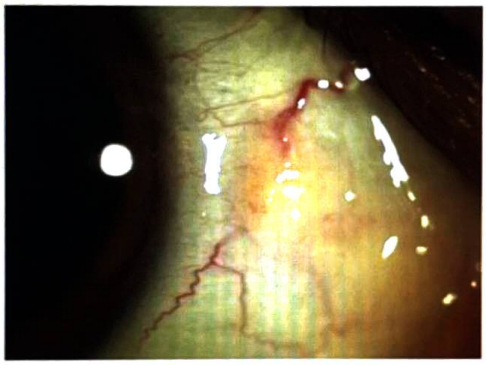
Pinguecula and concretions

01:19:45

It is an elastotic degeneration of collagen fibers in stroma of conjunctiva.

Appearance : Fat-like nodules.

MC on nasal part of conjunctiva.



Concretions :

On blinking : Eyelids rub against the bulbar conjunctiva & cornea

Leading to irritation.

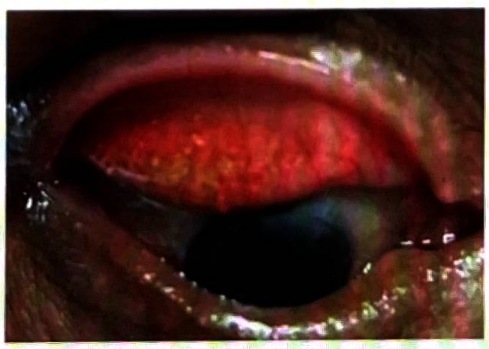
It is a collection of epithelial debris and mucus.

There is no calcium deposition.

Appearance : Minute, yellowish, white elevations.

Treatment : Removal with 26 G needle.

Spontaneously resolve with good lubrication.



EYELIDS : ANATOMY AND PATHOLOGIES

Eyelids or cover of the eye, are a movable fold of skin and muscle that can be closed over the eyeball or opened at will.

Anatomy of eyelids

00:01:01

3 muscles :

Muscle	Nerve supply	Action	On inability to perform its action/ lesion
Levator Palpebra Superioris muscle.	3rd cranial nerve.	Elevation of upper eyelid.	Ptosis.
Muller's muscle.	Sympathetic supply.	Elevation of upper eyelid.	Ptosis.
Orbicularis oculi.	7th cranial nerve.	Eyelid closure.	Lagophthal-mos.

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3 glands :

Meibomian gland (tarsal gland).	Modified sebaceous gland.	Opens posterior to anterior surface of eyelid margin.
Gland of Zeis.	Modified sebaceous gland.	Opens at base of eyelash follicles.
Gland of Moll.	Modified sweat gland.	Joins the ducts of gland of Zeis.

Active space

Cut surface of eyelid.

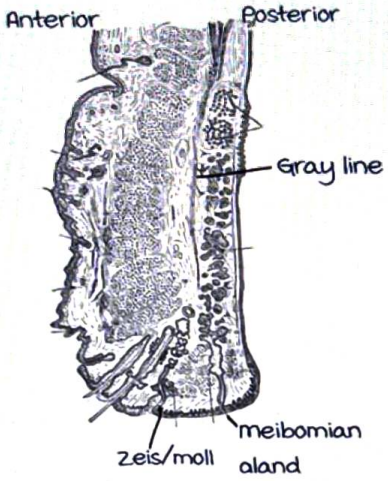
Eyelid has 2 surfaces :

- Anterior surface → Eyelashes seen at this surface.
- Posterior surface.

Gray line divides eyelid into 2 parts :

Anterior lamina.
Consists of :
Skin.
Subcutaneous tissue.
muscles.

Posterior lamina.
Consists of :
Tarsal plate (Fibrous tissue with no cartilage).
Palpebral conjunctiva.



Pathologies of eyelids

00:10:02

1. Chalazion :
   - It is lipogranulomatous inflammation of meibomian gland.
   - Non infective.
   - Clinical features :
     Painless swelling away from lid margin.
   - Treatment :
     a) Incision and drainage using chalazion scoop.
     Incision on posterior part (conjunctiva).
     Incision should be vertical (to preserve eye glands).
     Anterior surface is not incised as it results in scar formation.
     b) Intralesional triamcinilone (Shrinks the chalazion).
   - If recurrent → Suspect sebaceous cell carcinoma.

Active space

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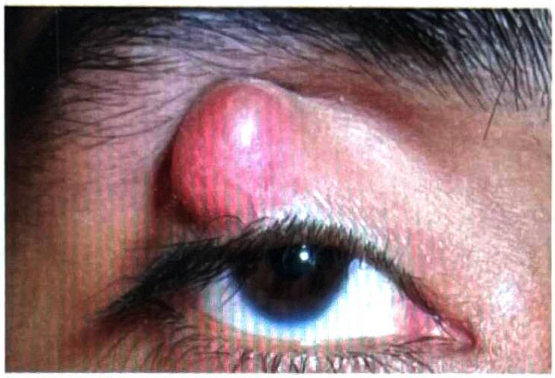


Chalazion → Swelling away from lid margin.

2. External hordeolum :
   - AKA Stye.
   - It is suppurative inflammation of gland of Zeis.
   - Infective agent → Staph. aureus.
   - Clinical feature → Painful swelling at the lid margin.
   - Treatment :
     a) Hot compresses.
     b) Oral antibiotics (if recurrent).

External hordeolum → Diffuse swelling at lid margin

3. Internal hordeolum :
   - It is suppurative inflammation of meibomian gland.

4. Trichiasis :
   - It's the inward misdirection of eyelashes.
   - Treatment :
     a) Epilation (Removal of lashes).
     b) Cryotherapy of lash base.

     Can cause corneal opacity if left untreated.

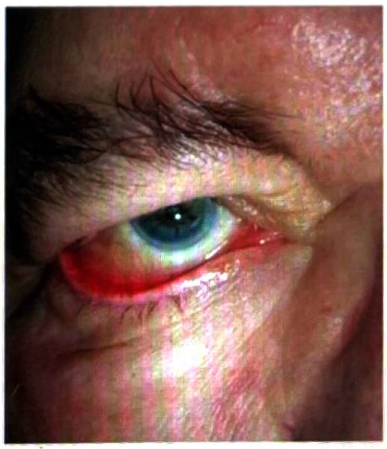
Eye lashes turned inwards.

5. Entropion :
   - It's the inward turning of eyelid margin.
   - Treatment :
     a) Spastic entropion → Botulinum toxin injection.
     b) Senile entropion → Modified Wheeler operation or, Weiss operation.
     c) Cicatrical entropion → Modified Burrow's operation or wedge resection.

Eyelids turned inwards.

6. Ectropion :
   - It's the outward turning of eyelid margin.
   - Most common cause is senility.
   - Treatment :
     a) Medial conjunctivoplasty.
     b) Byron Smith operation.

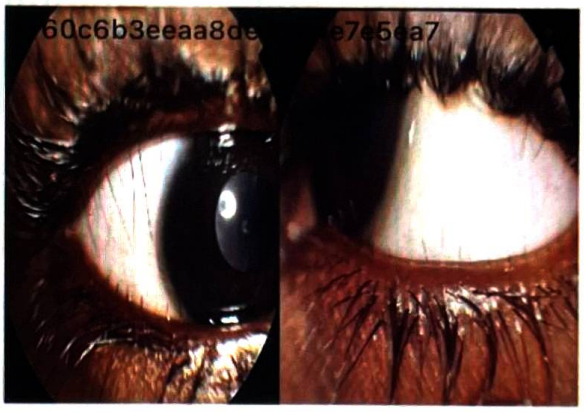
Lower lid turned outward.



Active space

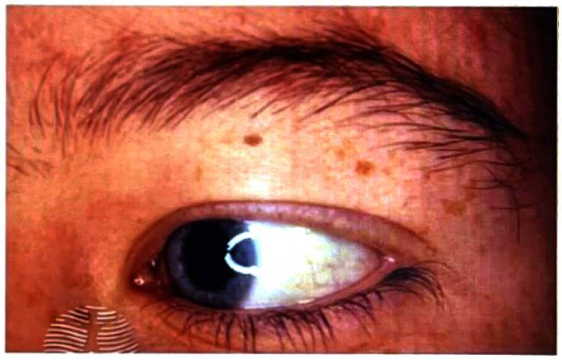
7. Distichiasis :
   - Double rows of eyelashes or, an extra posterior row of

eyelashes.



Anterior & posterior rows of eyelashes.

8. Madarosis :
   Loss of lateral 1/3rd of eyelashes.



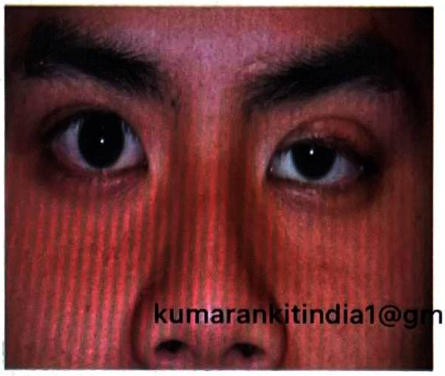
Lateral 1/3rd of eyelashes absent.

9. Tylosis :
   - It's the thickening of the eyelid margin.

10. Ankyloblepharon :
    - It's the fusion of eyelid margin of upper lid and lower lid.

11. Congenital ptosis :
    - Occurs due to defective development of LPS muscle.
    - Diagnostically → Upper lid crease is absent.
    - Lid lag → On down gaze, eye goes down, but eyelid doesnot go down.

Absence of upper lid crease in the left eye.



- Treatment :

a) LPS resection → For moderate ptosis + Fair LPS function.

b) Frontalis sling surgery → For severe ptosis + Poor LPS function.

- Synkinetic phenomena (related to congenital ptosis) : Marcus Gunn jaw winking syndrome → Occurs due to trigemino-occulomotor nerve synkinesis.

Jaw closed → Ptosis present.

Jaw opens → Upper lid moves up.

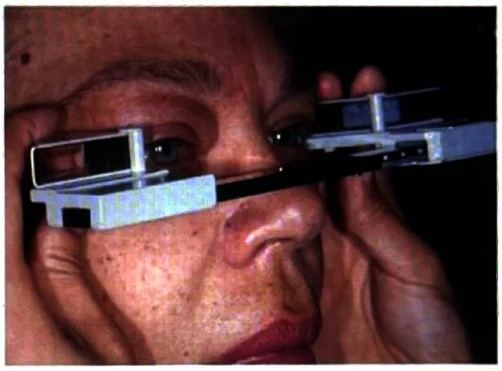
ORBIT

Proptosis

00:00:51

Protrusion of the eyeballs.
measured as the distance from lateral orbital rim & anterior most part of eye.

Hertel's exophthalmometer :



If protrusion >21 mm :
Proptosis.

Intermittent proptosis :

1. If proptosis increases with upper respiratory tract infection : Cause is orbital lymphangioma.
2. If proptosis increases on crying :
   Capillary hemangioma (child) or
   Encephalocele (infants).
3. If proptosis increase on bending forwrad/on valsalva manuovre : Orbital varices.

Orbital varices :

- Always unilateral.
- Seen among children.
- Bag of worms consistency.
- MRI/CT sacn : Phleobiths.

Pulsatile proptosis

00:06:05

Causes :

1. Orbital roof fracture as in NF I.
2. Caratico Cavernous Fistula (CCF) :
   Cause :
   Due to trauma in 75 % of cases.
   Spontaneous in 25% of cases (adults).

- Bilateral, starts unilateral.
- Severe ipsilateral headache + 3rd/4th/6th cranial nerve palsy (complete ophthalmoplegia : Restriction of ocular movements).

Inflammatory causes of proptosis :

Cavernous sinus thrombosis	Orbital apex syndrome
Nerves passing through :	
• 3rd cranial nerve. • 4th cranial nerve. • 6th cranial nerve. • First branch of 5th nerve (V1) : Ophthalmic nerve. • Second branch of 5th nerve (V2) : maxillary nerve.	Optic canal : • 2nd cranial nerve. Superior orbital fissure : • 3rd cranial nerve. • 4th cranial nerve. • 6th cranial nerve. • First branch of 5th nerve (V1) : Ophthalmic nerve. • Second branch of 5th nerve (V2) : maxillary nerve.
Sequential ophthalmoplegia. 6th nerve : Earliest. 3rd nerve ⟶ 4th nerve.	Concurrent ophthalmoplegia.
Corneal anesthesia (V 1).	Corneal anesthesia (V 1). Vision lost (2nd nerve).
Bilateral ,starts unilaterally.	Always unilateral.
Chemosis & proptosis : marked.	Chemosis & proptosis : mild to moderate.
Edema in mastoid region present.	Edema absent.
Systemic signs : Abrupt onset & severe.	Systemic signs : Slow onset & mild.

Orbital cellulitis

00:15:08

Usually occurs as an extension from paranasal sinuses.

Predisposing cause : Ethmoidal sinusitis.

Unilateral.

3rd/4th/6th cranial nerve involved : Complete ophthalmoplegia.

2nd cranial nerve : Vision lost.

First branch of 5th nerve (VI) not affected : No loss of corneal sensation.

Orbit related MC :

MC cystic orbital lesions : Ductal cysts of lacrimal gland.

MC cystic orbital tumor : Epidermoid and dermoid.

MC orbital neoplasm (pediatric age) : Epidermoid & dermoid.

MC orbital malignant tumor (pediatric age) :
Rhabdomyosarcoma.

MC orbital and periorbital tumor in children :
Capillary hemangioma.

MC benign orbital tumor (adults) : Cavernous hemangioma.

MC malignant orbital tumor (adults) : B cell NHL (lymphoma).

MC intrinsic lacrimal gland lesion/lacrimal gland epithelial neoplasm : Pleomorphic adenoma.

MC Orbital metastasis (pediatric age) : Neuroblastoma.

MC peripheral neural tumor of the orbit :
Plexiform neurofibroma.

MC primary source of orbital metastases :

- Breast : 42%.
- Lung : 11% (most deaths).
- Neuroblastoma (pediatric age).

Thyroid associated ophthalmopathy :

Also known as grave's ophthalmopathy.

Most common cause of proptosis (unilateral & bilateral) in adults.

Can occur in hyperthyroid, euthyroid or hypothyroid patients.

Signs :

Earliest sign : Goldzehier's sign (conjunctival congestion).

most common sign : Dalrymple sign (eyelid retraction).

2nd most common sign : Proptosis.

Von graefe sign : Lid lag.

Eyeballs go down, eyelids do not go down.

Kocher's sign : Staring appearance.

Stellwag's sign : Reduced frequency of blinking.

Due to edema & fibrosis of EOM : Restrictive myopathy.

Earliest muscle affected : Inferior rectus.

Earliest action affected : Elevation (Inferior rectus cannot relax → Superior rectus cannot contract → Elevation is affected).

OCULAR TRAUMA

Mechanical ocular trauma :

1. Blunt trauma (closed globe injury).
2. Penetrating trauma (sharp object).

Blunt ocular trauma

00:01:30

Closed globe injuries/contusion.

Examples : Injury by fist, tennis ball injury, champagne cork injury.

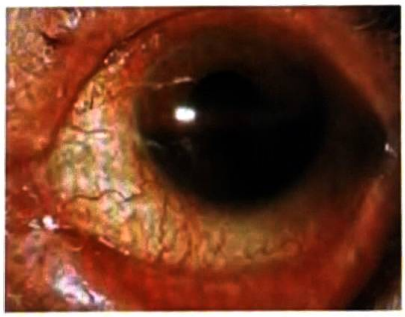
Signs :

Black eye : Periocular hematoma.

Conjunctiva : Sub conjunctival hemorrhage. (It is very common in trival trauma like injury with nails or pencil. It usually occurs in children).

Hyphema : It is collection of blood in anterior chamber.

It is most common sign of blunt ocular trauma.



Source of bleeding : Circulus arteriosus major (blood supply of iris branch of long posterior ciliary artery).

Treatment :

- Topical steroids are given to prevent rebleeding and to treat associated inflammation.
- Atropine : Controversial.
- If bleeding does not resorption, then paracentesis is performed or wash the anterior chamber.

Iris

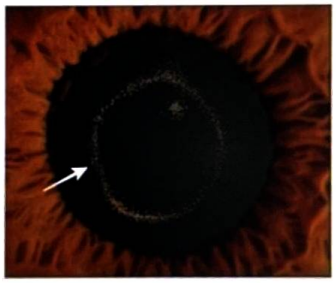
00:08:18

- Iridodonesis : "Tremulous iris". When root of iris detatches from ciliary body, it trembles/flutters because of aqueous flow.
- Iridodialysis : Complete detachment of iris from ciliary body in some parts. Iridodialysis leads to "D shaped pupil".

Iridodialysis

Lens :

- Rosette shaped cataract (Posterior subcapsular cataract).
- Ectopia lentis/Subluxation of lens.
- Vossius ring : Imprint of miotic pupil on the anterior surface of lens as structures of eye are pushed back due to force. Imprint is caused by pigmented iris.

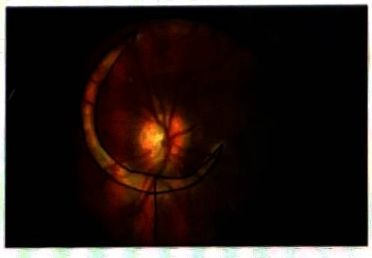


- Phacodonesis : Tremulous lens.

Fundus :

- Cherry red spot at macula due to collection of fluid which in blunt trauma is known as Berlin's edema.
- Fundus appearance is known as Commotio retinae (cloudy grey) appearance.
- Traumatic optic neuropathy : RAPD. Self limiting.
- Choroidal rupture : Bucket handle configuration.

Scleral show due to choroidal rupture



Globe rupture :

Usually occurs due to fall on an object like a knob.

Site : Supro-nasal limbus near Schlemn's canal.

Penetrating trauma

00:21:02

1. Sympathetic opthalmitis (uveitis).
2. Intraocular foreign body.

Intraocular foreign body :

- most common intraocular foreign body : Iron or steel chips.
- Iron foreign body : MRI is contraindicated.

↓

CT scan is investigation of choice

↓

Leads to siderosis bulbi

↓

a. Deposition of a rust ring on anterior capsule of lens.
b. Hyperpigmentation of iris.
c. Secondary open angle glaucoma.

↓

Diagnosis : Prussian blue reaction.

- Ophthalmia nodosa : Caterpillar hair causing uveitis.

Most common fracture in orbital trauma is floor of orbit known as blow out fracture of orbit.

Orbital trauma

00:25:02

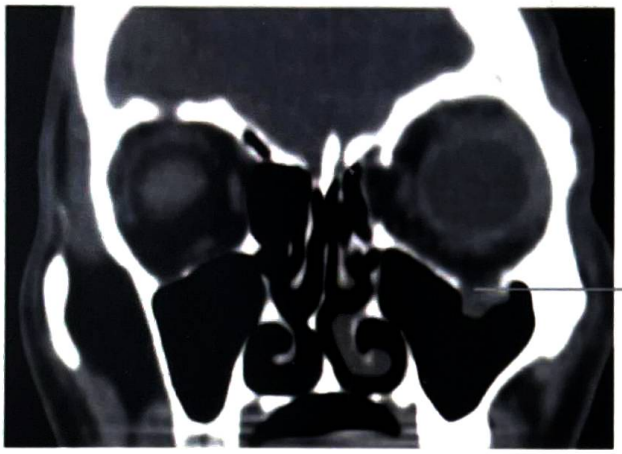
Trauma to bones forming the orbit.

MC fracture : Floor of orbit (blow out fracture).

Clinical features :

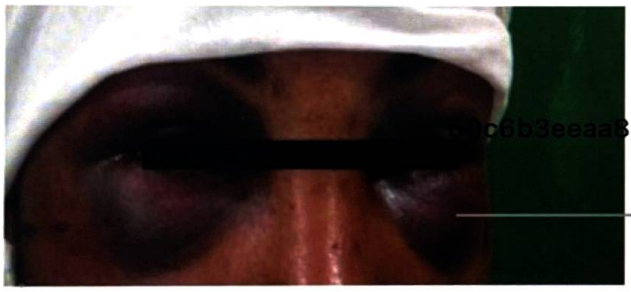
a. X-ray : Tear drop sign.
b. Infraorbital nerve anaesthesia.

c. Double diplopia (diplopia in up-gaze and down gaze, due to muscle entrapment of inferior oblique and inferior rectus by fractured bone pieces). Known as restrictive squint.



Tear drop sign : Orbital contents going down due to # floor of orbit

Fracture of roof of orbit : Presents with racoon eyes or, panda eyes.



Racoon/ Panda eyes

Chemical injuries 00:30:54

Causes

Alkalis	Acids
a. Cause necrosis of the tissue.	a. Cause coagulation of the tissue.
b. Penetrate deeper (due to necrosis).	b. Do not penetrate.

Active space

Roper Hall classification for Ocular surface burns

Grade	Prognosis	Limbal schemia	Corneal involvement
I	Good	None	Epithelial damage
II	Good	< 1/3	Haze, but iris details available
III	Guarded	1/3 to 1/12	Total epithelial loss with haze that obscures iris details
IV	Poor	> 1/2	Cornea opaque with iris & pupil obscured

Dua's classification for ocular surface burns

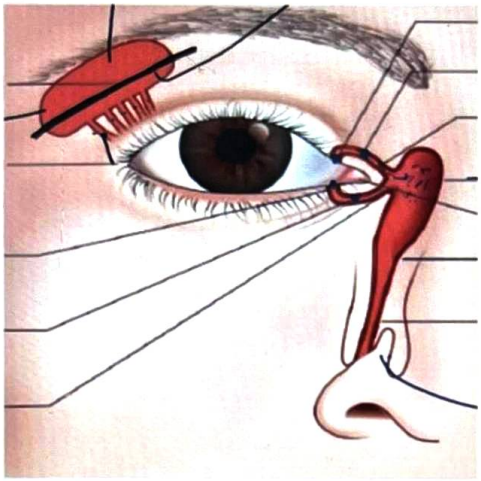
Grade	Prognosis	Clinical findings	Conjnctival involvement	Analogue scale
I	Very good	0 clock hours of limbal involvement	0%	0/0%
II	Good	≤ 3 clock hours of limbal involvement	≤ 30%	0.1–3/1–29.9%
III	Good	3–6 clock hours of limbal involvement	30–50%	3.1–6/31–50%
IV	Good – guarded	6–9 clock hours of limbal involvement	50–75%	6.1–9/51–75%
V	Guarded – poor	9–<12 clock hours of limbal involvement	75– <100%	9.1–11.9/75.1–99.9%
VI	Very poor	12 clock hours of limbal involvement	100%	12/100%

Management :

1. Copious irrigation of eye using balanced salt solution or, ringer lactate by double eversion of eyelid to reach fornices of eye.
2. Topical antibiotics to prevent secondary infection.
3. Atropine for ciliary spasm.
4. Lubricating eyedrops helps in epithelial regrowth.
5. Topical sodium citrate drops prevents proteolytic activity.

LACRIMAL APPARATUS : ANATOMY, WATERING EYE AND DRY EYE

Anatomy of lacrimal apparatus 00:00:59



Components :

- Secretion of tears.
- Drainage of tears.

Secretion of tears :

Main lacrimal gland →

Divided into two parts by levator palpebrae superioris (LPS) passing through the center of the lacrimal gland.

- Superior part → Orbital part
- Inferior part → Palpebral part (main excretory ducts are attached)

Responsible for reflex tear secretion.

Accessory lacrimal glands of Krause and Wolfring :

- Situated in fornix.
- Responsible for basal secretions.

Parasympathetic (secretomotor) supply of lacrimal gland.

↓

Superior salivatory nucleus (Pons).

↓

Greater superficial petrosal nerve (branch of facial nerve).

↓

Relays into Pterygopalatine ganglion.

↓

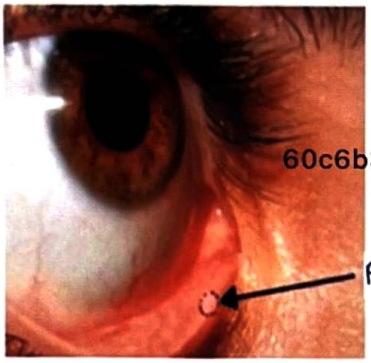
Zygomatic and lacrimal
(branch of maxillary division of trigeminal nerve)

↓

Main lacrimal gland stimulated (secretions).

Drainage of tears :

- Puncta in the upper and lower eyelid.



- Canaliculi in the upper and lower canaliculi.
- Lacrimal sac (surrounded by orbicularis oculi muscle due to which there is drainage of tears during blinking).
- Nasolacrimal duct (NLD) → Opens in inferior meatus of nose which is gaurded by Valve of Hasner.

MC site of blockage in congenital dacryocystitis → Valve of Hasner

Watery eye

00:09:44

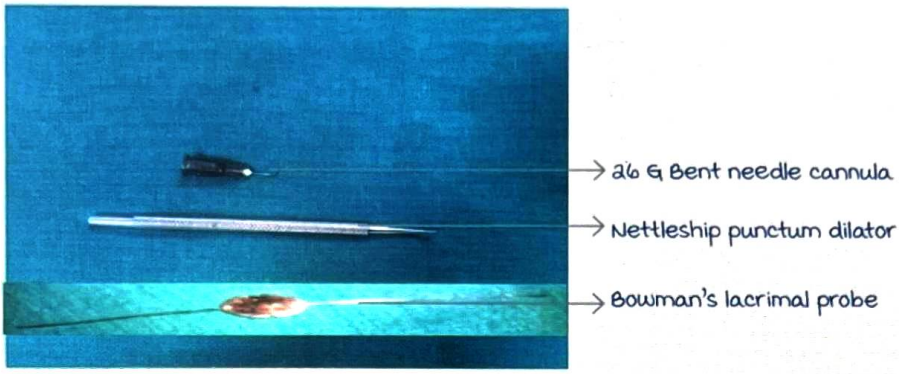
Watery Eye

Hyperlacrimation	Epiphora
Due to emotional stress	Due to blockage
Or ocular inflammation	

Testing of watering eye →
Done by a syringing, probing and Jones dye test.

Active space

a. Nettleship punctum dilator.
b. Bowman's lacrimal probe.
c. 26 G Bent needle cannula.



Procedure :

Punctum is dilated.
↓
26G needle is inserted into the puncta and canliculi.
↓
Saline is injected.

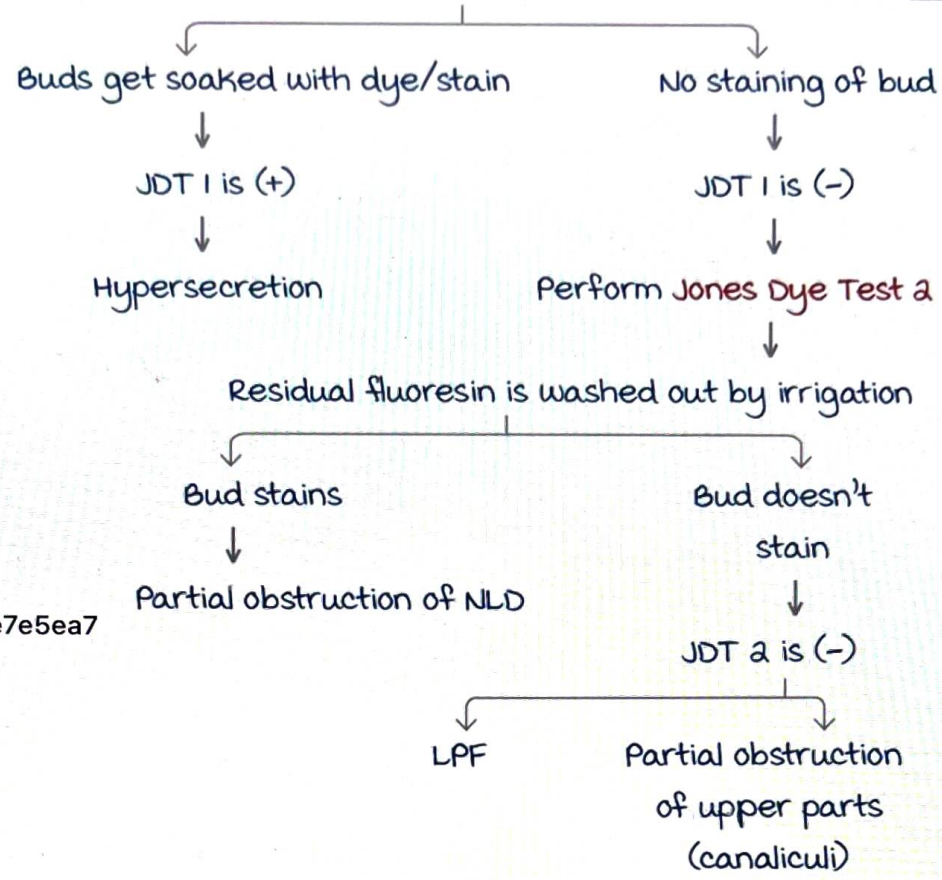
- Fluid is flowing into nose and throat → No Blockage → (Patent Drainage System)
  - Hyperscretion (H)
  - Lacrimal Pump Failure(LPF)
  - Partial Obstruction
- Regurgitation of Fluid → Obstruction/Blockage → Probing
  - Hard Stop: NLD blockage
  - Soft Stop: Lacrimal sac or canaliculi block

Differentiation between Hypersecretion, Lacrimal pump failure and Partial obstruction 00:17:28

To differentiate between (H), (LPF), and (PO).

Jones Dye Test I (JDT I) :

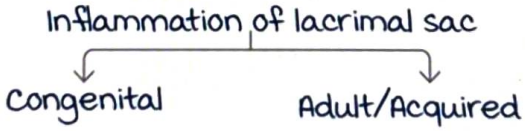
2% fluorescein dye instilled into conjuctival sac
↓
Place cotton at opening of NLD in the nose



Dacryocystography is used to differentiate LPF and partial obstruction of upper parts.

Dacryocystitis

00:23:51



Congenital cause :

C/o watering since birth due to Non canalization of NLD

Occulsion is MC at valve of Hasner

Treatment :

- Lacrimal sac massage + Topical antibiotics.
- 9–12 months → Syringing.
- > 1 year → Probing.
- > 4 years → Dacryocystorhinostomy (DCR).

Adult/acquired cause :

Caused due to inflammation by Streptococcus hemolyticus.

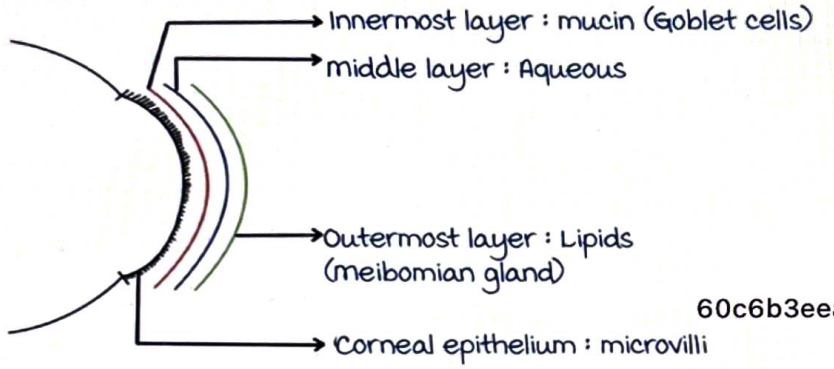
Treatment :

- Antibiotics (oral)
- DCR → If fistula is formed

Tumors of Lacrimal Gland 00:27:56

1. Two types :
   - Pleomorphic Adenoma → Benign tumour.
   - Adenoid Cystic carcinoma → Malignant tumour.
2. The eye is pushed down and medially (As lacrimal gland is situated superotemporally)

Tear film :

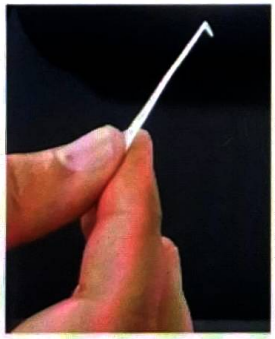


Dry eye

Aqueous Deficiency	Lipid Deficiency	Mucin Deficiency
Keratoconjunctivitis Sicca (KCS)	Evaporative dry eye	Examples : Vitamin A deficiency.
Example : Sjogrens Syndrome.	Examples : • meibomian gland • Contact lens wear	

Tests :

1. Schirmer Test
   - Quantitative test → measuring tear production.
   - Uses Whatman's filter paper no 41.



Method → Folded at blue line and Inserted into lower fornix, then wait for 5 mins.

↓

After 5 minutes

- > 15 mm strip wet : Normal
- < 10 mm : Dry eye

2. Tear film breakup time(TBUT).

2% fluorescein dye instilled into conjuctival sac

↓

Illuminated with blue light

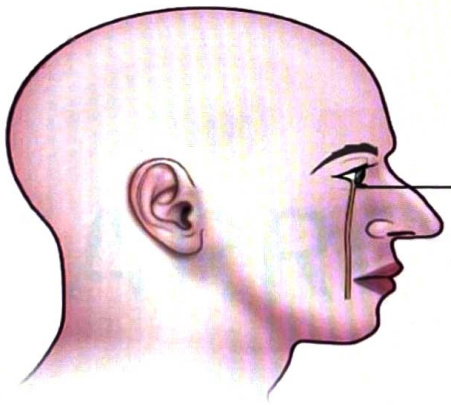
↓

Interval between a blink and appearance of 1st spot of dryness on cornea

If < 10 sec : Mucin Deficiency

Dry spot at the same location : Corneal disease

3. Phenol red thread test.



use a thread impregnated with phenol red dye (yellow in color) which on contact with tears becomes red.

After 15 seconds

If < 6 mm : Dry eye

If > 15 mm : Normal

Treatment of dry eye :

- Aqueous deficiency → Lubricants.
- Lipid deficiency → Omega-3-fatty acids.
- Sjogren's syndrome → Cyclosporine.

COMMUNITY OPHTHALMOLOGY

NPCBVI

00:00:39

National Programme for Control of Blindness and Visual Impairment.

- Previously called as NPCB.

Definition of Blindness

- Presenting visual acuity <3/60 in better eye

  Or

- Visual Field limitation to <10 degree

  Same as definition by WHO.

Prevalence : 1.05%

Main Causes :

1. Cataract (62.6% cases) : most common
2. Refractive errors (19.7% cases).
3. Glaucoma (5.8% cases).

Vision 2020

00:04:38

Aim :

1. To reduce blind people to 25 million by 2020.
2. To eliminate avoidable blindness by 2020.

Launched : In 1999 in Geneva but India adopted in 2001 at Goa.

Disease Covered in India :

1. Cataract.
2. Refractive errors.
3. Glaucoma : Last disease covered under Vision 2020.
4. Corneal Blindness.
5. Childhood Blindness.
6. Trachoma.
7. Diabetic Retinopathy.

Disease Covered Globally :

1. Cataract.
2. Refractive errors.
3. Childhood Blindness.
4. Trachoma.
5. Onchocerciasis.

2	1/50 crore
20	1/5 crore
200	1/50 lac
2000	1/5,00,000
20,000	1/50,000

School Eye Screening Programme

00:11:52

- Done by Teachers.
- Aim : 10 – 14 years (Standard 5 to 8).
- 1 Teacher / 150 Students.
- Refer to PHC if vision is < 6/9.

Active space