OSPE/ TOACS
OPHTHALMOLOGY

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Chairman
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Ophthalmology

Subject Marks = 200

Total Marks 100

A. Theory

Theory Marks Distribution:

i. Internal Evaluation — 10 Marks

ii. Paper — 90 Marks

MCQs — 45 Marks

(45 MCQs of One Best Type)

SEQs — 45 Marks

09 SEQs to be attempted out of 12 (Each SEQ of 05 Marks)

Total Marks 100

B. Practical

Practical Marks Distribution:

i. Internal Evaluation — 10 Marks

ii. Practical — 90 Marks

a) OSCE Stations — 60 Marks (05 marks for each OSCE Station)

12 working stations
- 07 Interactive / Observed
- 05 Non-interactive / Static

03 Rest Stations — Rest Stations carry no marks

b) 02 Short Cases — 10 Marks (05 marks each)

c) 01 Long Case — 20 Marks

OSPE/ TOACS

1. Clinical Skills
2. Investigations
3. Equipments
4. Instruments
5. Clinical Scenarios
6. Counseling

1. Clinical Skills

- Ocular Movements
- Cover Un Cover Test
  Hirshburg Test

- Visual Fields
  Humphry
  Goldman
  Confrontation (Bed side)

- Pupil Examination

2. Investigations

- B- Scan
- O.C.T
- Visual Fields
- Biometry

3. Equipments

- Slit Lamp
- Direct Ophthalmoscope
- Indirect Ophthalmoscope
- Tonometer
- Lens Box

4. Instruments

- Operating Instruments

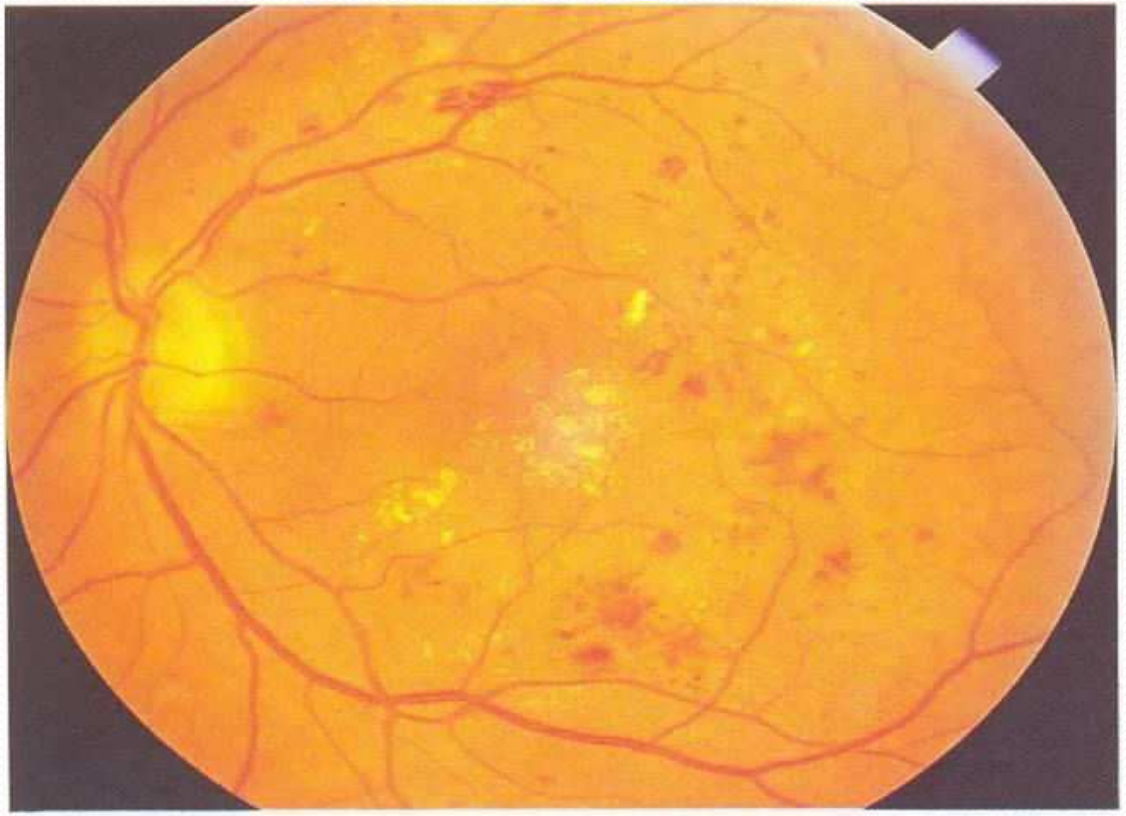
5. Clinical Scenarios

- Cataract
- Glaucoma
- Cornea
- Fundus
- Myopia / Hypermetropia
- Squint

6. Counseling

- Retinoblastoma
- Retinitis Pegmentosa
- Any Other

- Interactive /Observed
- Static

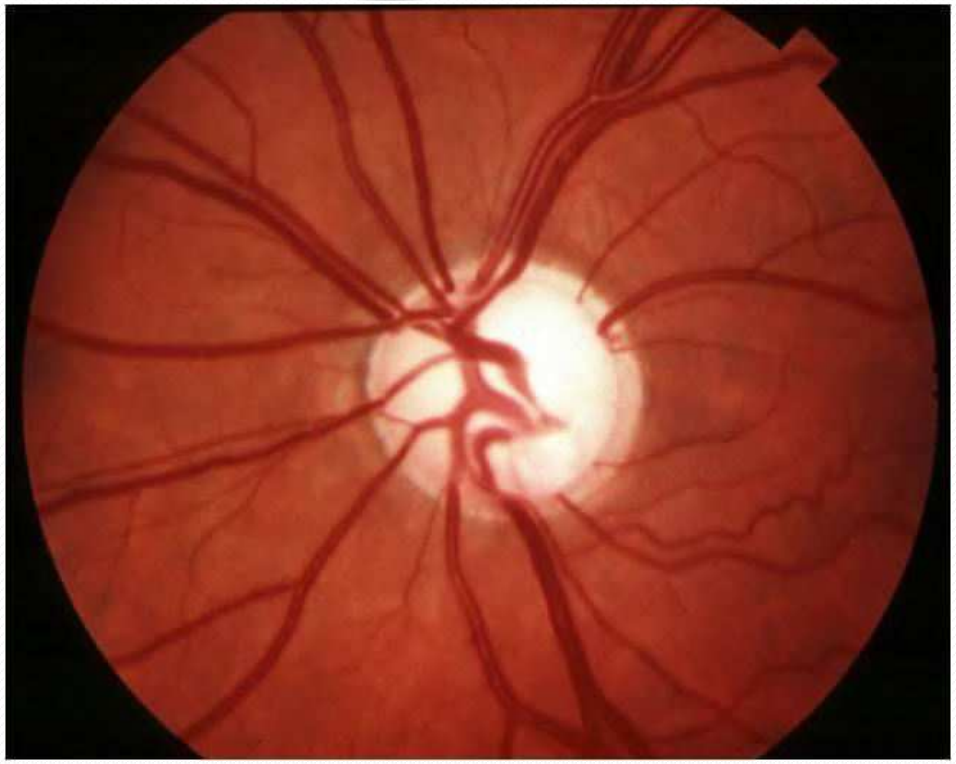


1. What findings are there in this fundus photograph?
2. What is your most probable diagnosis?
3. What is your differential diagnosis?
4. What options do we have to treat this patient?

STATION: DIABETIC RETINOPATHY

KEY:

1. Retinal hemorrhages, Retinal exudates (Hard exudates) involving macula---------- (1)
2. Diabetic Retinopathy(Non proliferative) with maculopathy-- (1)
3. Hypertensive retinopathy, CRVO, radiation retinopathy------(1.5)
4. ---------- --(1.5)
   a) Control diabetes and systemic risk factors
   b) Anti VEGF injections
   c) Focal macular laser

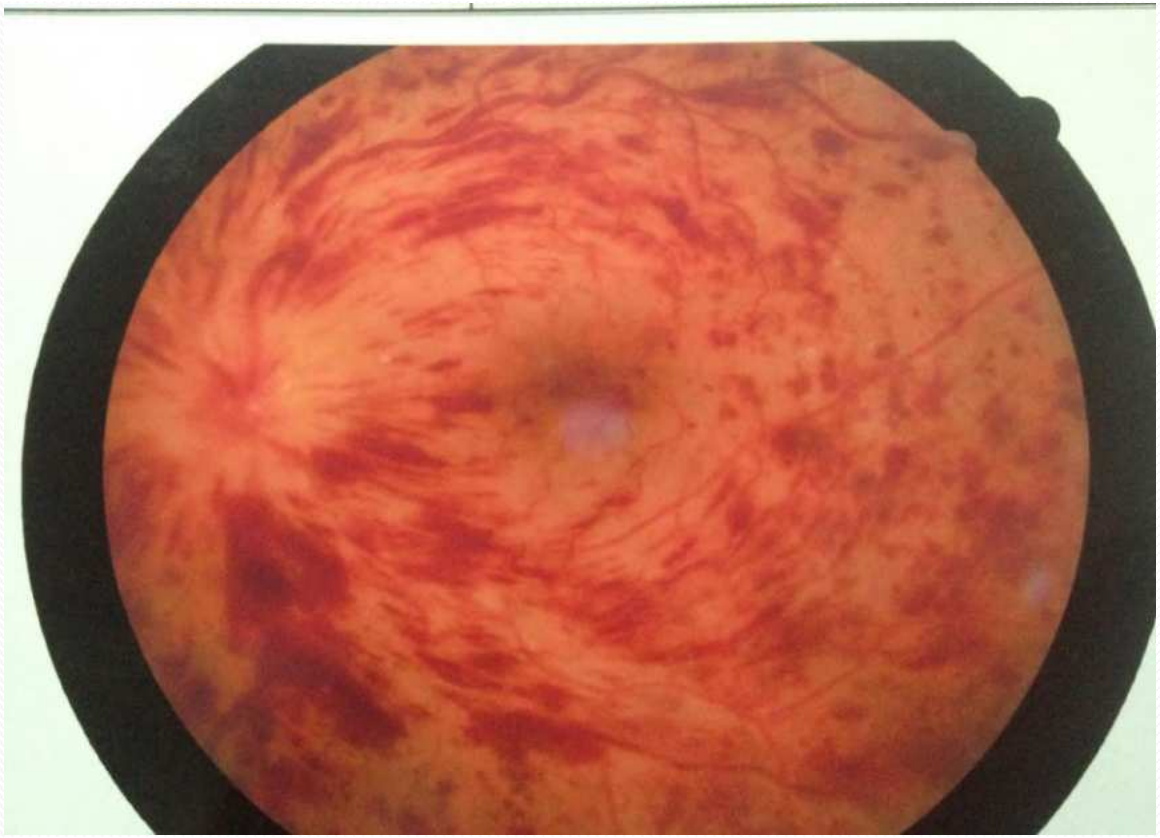


1. What findings do you see in this photograph?
2. What is your most probable diagnosis?
3. Name any three types of medications (topical) are used to treat this condition?
4. What surgical procedure is the gold standard for treating this condition?

STATION: OPTIC DISC CUPPING (OPEN ANGLE GLAUCOMA)

KEY:

1. Optic Disc cupping----------1
   (Increased cup-disc ratio)
2. Glaucoma (Open angle) ----------1.0
3.----------2 (Maximum)
   i. Prostaglandin analogues ----------0.5/each
   ii. Alpha-agonists (sympathetic) 0.5/each
   iii. B-Blockers
   iv. Para-sympathetic Pilocarpine)
   v. Carbonic anhydrase inhibitors
4. Trabeculectomy ----------1



1. What findings can be seen in this photograph?
2.What is your most probable diagnosis?
3. What is your differential diagnosis?
4. What are its possible complications?

STATION: CRVO

KEY:

1. Retinal bleeds(diffusely scattered), vascular dilatation/ tortuosity, Hyperemic disc & blurred margins, retinal/macular edema----------0.5/each(Max 2)

2. CRVO----------1
3. Diabetic retinopathy, Hypertensive retinopathy, Radiation retinopathy----------0.5/each (Max 1)
4. Anterior segment neovascularization (NVG),----------0.5/each (Max 1)

   Retinal neovascularization with its sequelae (Vit bleed & TRD)

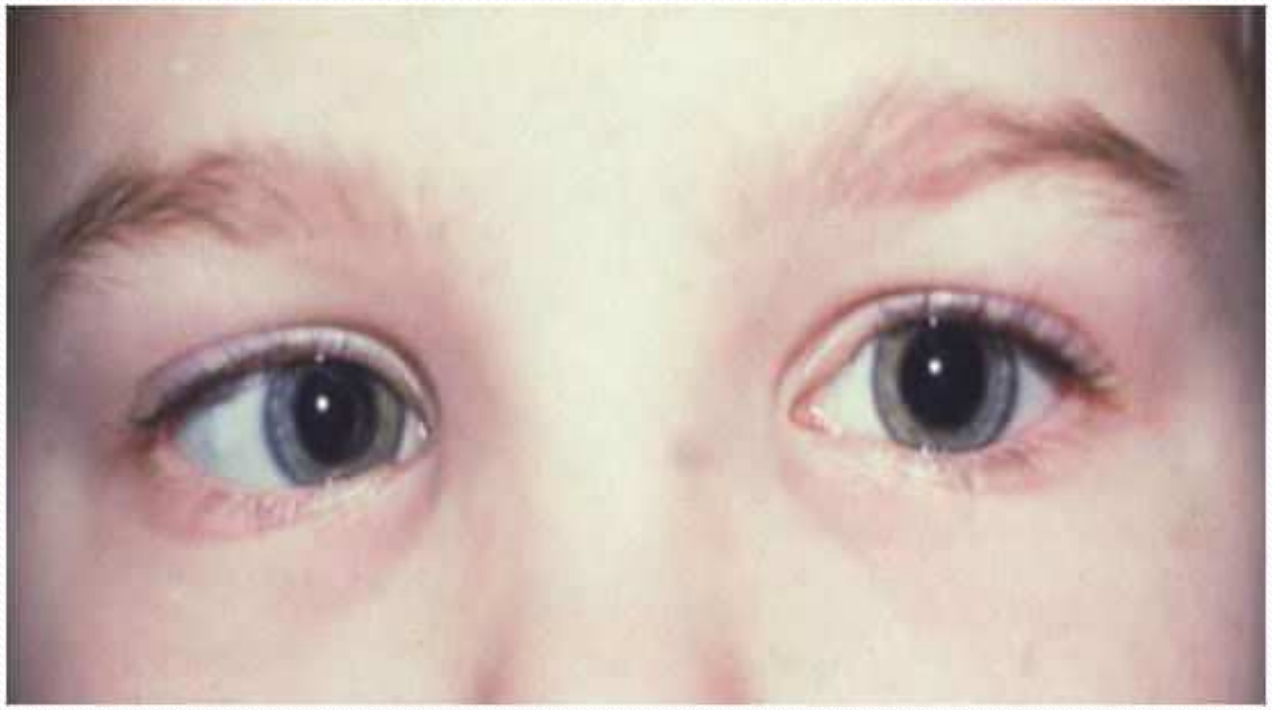
1. What findings do you see in this photograph?
2. What are your primary concerns in this eye?
3. Is there any risk to the fellow eye?
4. How are you going to treat this eye?

STATION: OGI WITH UVEAL PROLAPSE

Key

1. OGI (Scleral laceration with uveal tissue prolapse and distorted pupil)----------1.5
2.
    a) Reduce pain----------0.5/each (Max 1.5)
    b) Reduce inflammation
    c) Prevent infection
    d) Exclude IOFBs and so its related complications.
    e) Restore anatomical integrity (globe repair)

3. Sympathetic ophthalmia---------- 1.0
4. Prepare for GA, Antibiotics, Anti inflammatory ----- drugs, Globe repair after excluding IOFBs -------- 1.0



1. What findings do you see in this photograph?
2. What clinical tests/procedure you would like to perform for this patient?
3. What is the most common underlying cause in children for this condition?

STATION: RIGHT ESOTROPIA (CHILD)

KEY:

1. Right convergent squint (Esotropia) ----------1

2. ----------1/each (Max 3.0)

   a. VA check including amblyopia
   b. Squint assessment tests (Hirshberg's, Krimsky etc)
   c. Cycloplegic refraction
   d. Fundoscopy

3. Hypermetropia----------1.0

STATION: DIRECT OPHTHALMOSCOPE

Command Please examine this patient's fundus with direct ophthalmoscope

1. Consent & introduction----------0.5
2. Can hold and turn on correctly----------0.5
3. Use his / her Rt eye, for examining Rt eye of the patient ----------
----------0.5
4. Perform distant direct ophthalmoscopy----------1
5. Perform fundus examination & able to identify disc, vessels and macula----------2.0
6. Thanks to patient ----------`0.5

STATION PUPIL

Please perform pupil examination of this patient?

STATION: PUPIL

Key

1. Consent & introduction----------0.5
2. Light reflex
    a. Direct reflex----------1
    b. Indirect light reflex----------1
    c. Swinging light reflex----------1
3. Near response (reflex) ----------1
4. Thanks to patient----------0.5

Thank you

ANATOMY OF THE EYE

Dr samina karim

AP Diagnostic Ophthalmology

- Anatomy of the eye consist of
  - Orbit
  - Adipose tissues
  - Eyelids
  - Eyelashes
  - Eye ball
  - Muscles
  - Nerves and vessels

ORBIT

- Bony cavity which houses the eyeball
- 4 WALLS –
  - ✓ ROOF,
  - ✓ FLOOR,
  - ✓ MEDIAL AND
  - ✓ LATERAL WALL
- 30 mL in volume
- Has an apex where nerves and vessels emerge

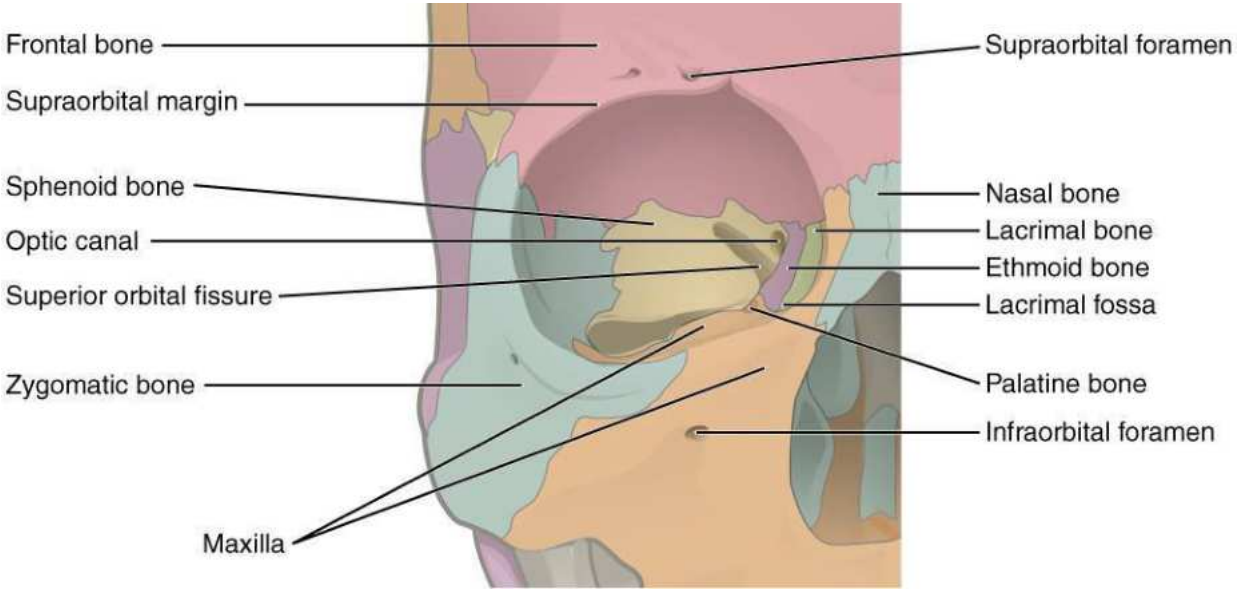
ORBITAL WALLS

- Roof
  - Composed of the lesser wing of the sphenoid, and orbital plate of the frontal bone
  - It is related to the frontal sinus

- Lateral wall
  - Separated from the roof by the superior orbital fissure
  - Composed of the greater wing of the sphenoid, zygomatic bone
- Strongest part of the bony orbit

- Floor
  - Separated from the lateral wall by the inferior orbital fissure
  - Intimately related to the maxillary sinus
  - Composed of maxillary bone, zygomatic bone and palatine bone
  - Orbital contents can herniate into the maxillary sinus in trauma cases

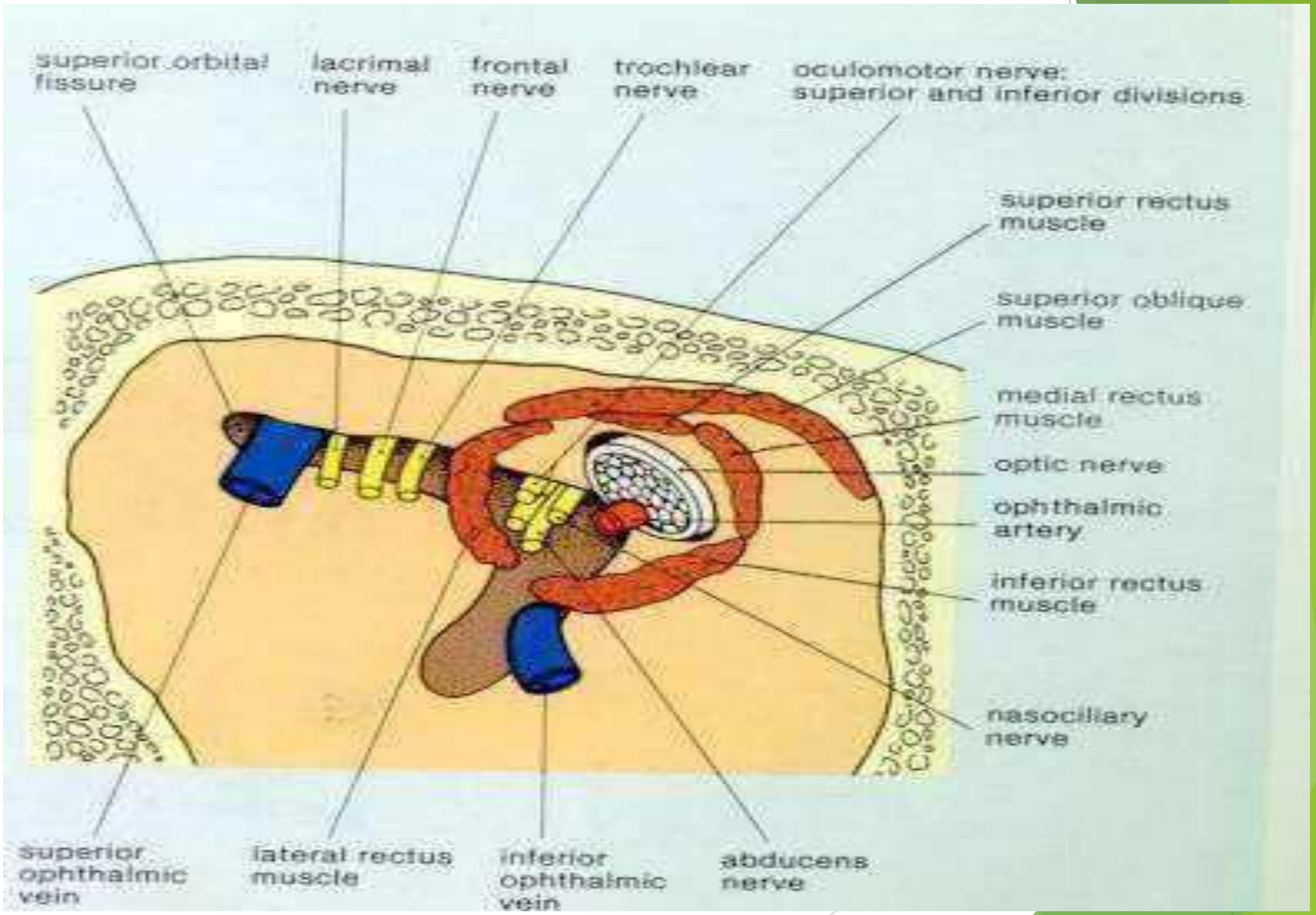
- Medial wall
  - Intimately related to the ethmoid and sphenoid sinuses
  - Composed of the ethmoid bone, sphenoid bone, lacrimal bone and maxilla


Frontal bone
Supraorbital margin
Sphenoid bone
Optic canal
Superior orbital fissure
Zygomatic bone
Maxilla
Supraorbital foramen
Nasal bone
Lacrimal bone
Ethmoid bone
Lacrimal fossa
Palatine bone
Infraorbital foramen

ORBITAL APEX

- Serves as a portal for nerves and vessels

- Site of origin of all extra ocular muscles except inferior oblique


superior orbital fissure
lacrimal nerve
frontal nerve
trochlear nerve
oculomotor nerve: superior and inferior divisions
superior rectus muscle
superior oblique muscle
medial rectus muscle
optic nerve
ophthalmic artery
inferior rectus muscle
nasociliary nerve
superior ophthalmic vein
lateral rectus muscle
inferior ophthalmic vein
abducens nerve

SUPERIOR ORBITAL FISSURE

- Lateral portion
  - Superior ophthalmic vein
  - Lacrimal nerve
  - Frontal nerve
  - Trochlear nerve
- Medial portion
  - Superior and inferior div. Of oculomotor nerve

ORBITAL APEX

- Optic canal
  - Transmits optic nerve and ophthalmic artery
- Superior orbital fissure
  - Also transmits the inferior ophthalmic vein

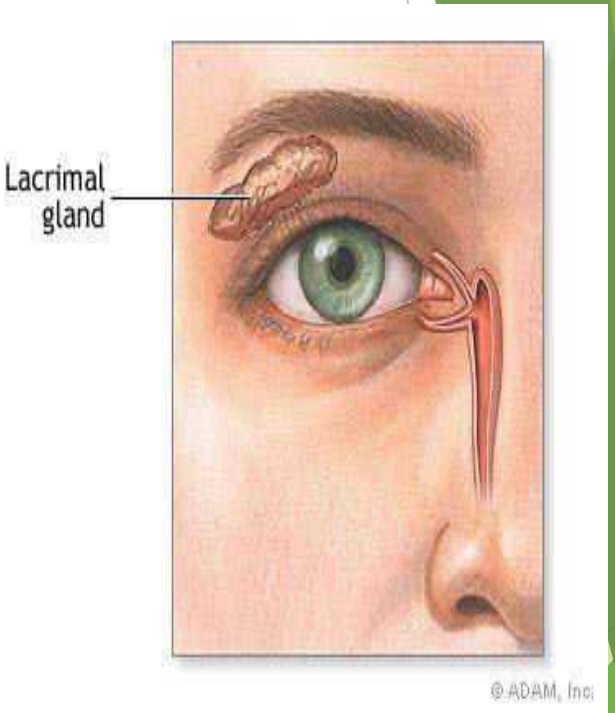
BLOOD SUPPLY OF THE ORBIT

- Ophthalmic artery
  - Central retinal artery
  - Lacrimal artery
  - Muscular branches
  - Long and short posterior ciliary artery
  - Medial palpebral arteries

Venous Drainage of the Orbit

- Superior and Inferior Ophthalmic Veins
- Vortex veins
- Anterior Ciliary Veins
- Central retinal Veins

- Lacrimal glands – produce tears that Lubricate & have a germicidal effect



- Eyebrows – protect against foreign particles, perspiration, & direct rays of light

- Eyelids – folds of skin that cover the
  - Surface of the eye; close by reflex action when an object approaches
- Eyelashes – secrete oils that prevent
  - Lids from sticking together

Eyeball

- Roughly spherical
- approximately 24.5 mm (less than an inch) in length
- About 5mL in total volume

Layers of the eye

- Three layers

1. Outer layer of the eye ball___Consist of
    1. Conjunctiva
    2. Tenon's Capsule
    3. Sclera
    4. Cornea
2. Middle layer of the eye ball--- consist of
    1. Iris
    2. Cillary body
    3. Choroid
3. Inner most layer of the eye ball---consist of
    1. retina

Outer layer of the eye ball

Conjunctiva

- Thin transparent mucous membrane which covers the posterior surface of the eyelid (palpebral conjunctiva) and the anterior surface of the sclera (bulbar conjunctiva)
- composed of two to five layers of stratified columnar epithelial cells
- contains glands which help in ocular lubrication

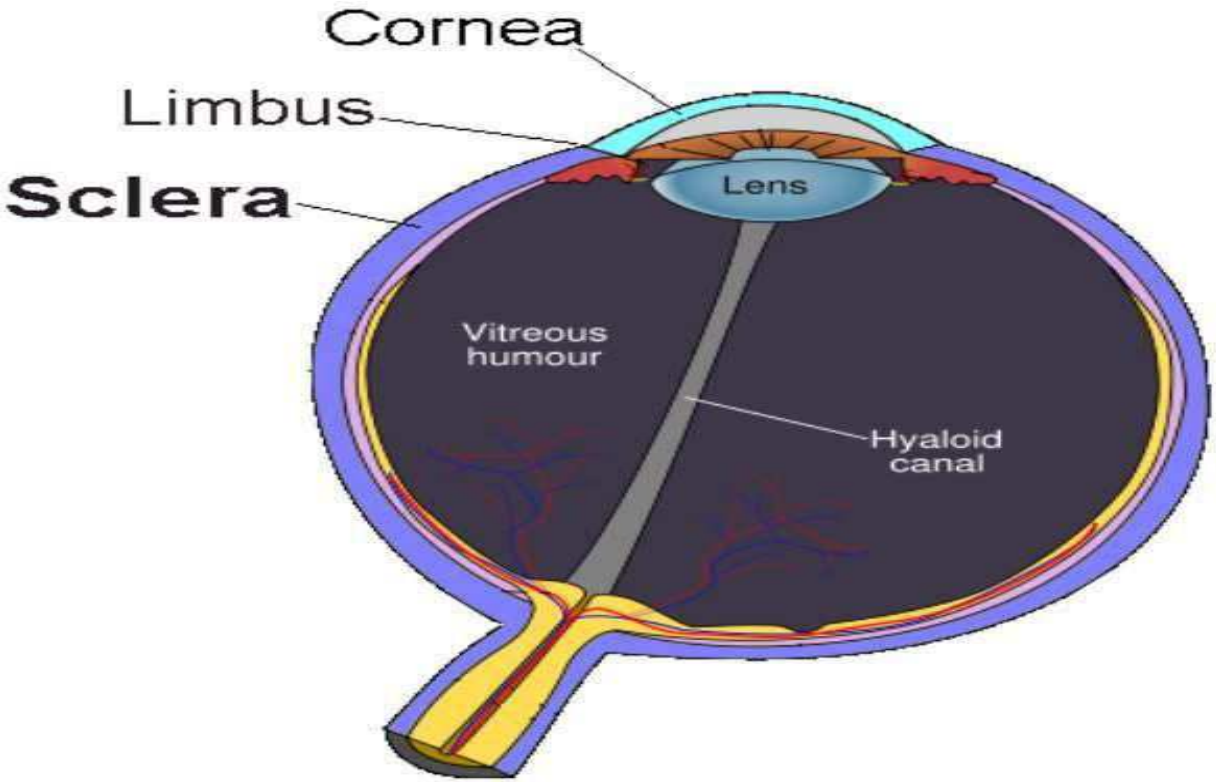
- Blood Supply
  - anterior cilliary artery
  - palpebral arteries
- Nerve Supply
  - first division of the trigeminal nerve

Tenon's Capsule

- Afibrous membrane that envelopes the globe from the limbus to the optic nerve
- continuous with the EOM's
- Thickens to form check ligaments

Sclera

- The sclera forms the posterior opaque 5/6 part of the external fibrous tunic of the eyeball.
- Its whole outer surface is covered by tenon's capsule and also by the bulbar conjunctiva in the anterior part
- Its inner surface lies in contact with the choroid with a potential suprachoroidal space in between.
- Sclera is thickest posteriorly(1mm) and gradually becomes thin when traced anteriorly.


Cornea
Limbus
Sclera
Lens
Vitreous humour
Hyaloid canal

Microscopic structure

Histologically, sclera consist of following three layers

- Epicleral tissue
- Sclera proper
- Lamina fusca

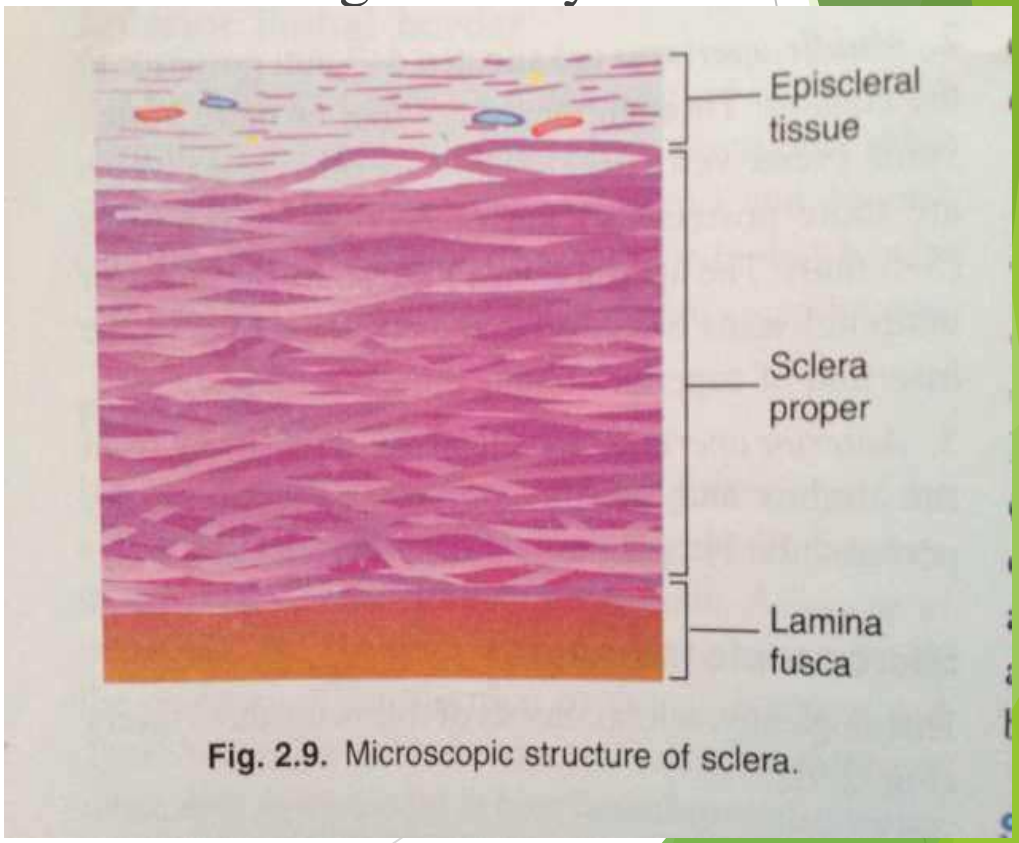


Fig. 2.9. Microscopic structure of sclera.

Blood supply of the sclera

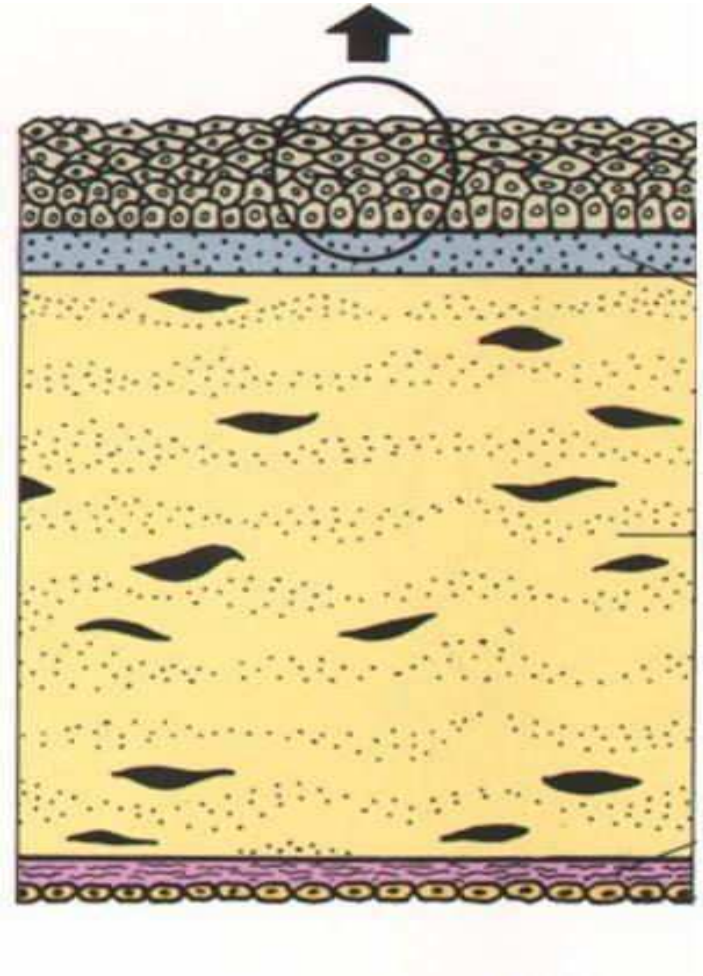
- The episclera receives its blood supply from the anterior ciliary arteries, anterior to the insertions of the rectus muscles and the long and short posterior ciliary arteries.

Nerve supply of the sclera

- The sclera is supplied by the branches from the long ciliary nerves anteriorly and short ciliary nerves behind the equator.

Cornea

- It act as a clear refractive surface and a protective barrier to infection and trauma.
- Its anterior surface is elliptical and posterior surface is circular
- It thinnest centrally and thickest in the periphery



Layers of the cornea

- Epithelium
- Bowman membrane
- Stroma
- Descemet membrane (posterior limiting layer of cornea)
- Endothelium

- The cornea is transparent because of the specialized arrangement of the collagen fibrils within the stroma, which must be kept in a state of relative dehydration.
- Function of the cornea are the
- protection against invasion of microorganisms into the eye,
- and the transmission and focusing (refraction) of light

- Nutrition and nerve supply of the cornea

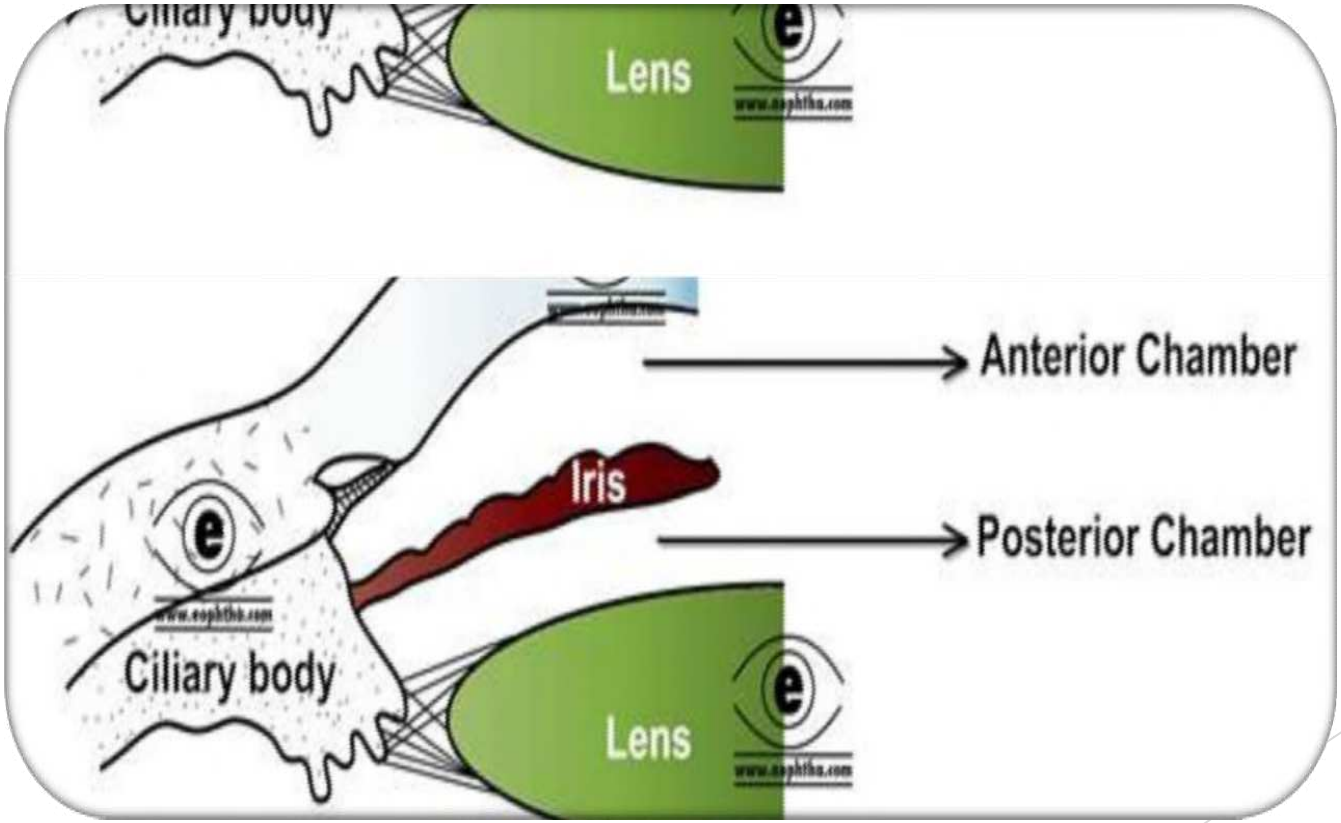
- Cornea is avascular structure

- Relies upon diffusion from limbs and aqueous for nutrion.

- First division of trigeminal nerve forms stromal and sub epithelial plexus responsible for corneal sensation.

Middle layer of the eye ball

- Iris
- It is the anterior part of the uveal tract.
- Forms diaphragm like structure in front of lens.
- Center of iris has an 3-4 mm aperture called Pupil.
- At periphery it is attached to the anterior surface of ciliary body.
- Divides the space between the cornea and lens into anterior and posterior chamber


Ciliary body
Lens
Anterior Chamber
Iris
Posterior Chamber
Ciliary body
Lens

MACROSCOPIC APPEARANCE

- **Anterior surface—**

- Divided into cilary and pupillary zone by zigzag line called
- *collarette.*

- It is the thickest region of the iris which lies about 2 mm from the pupil margin.

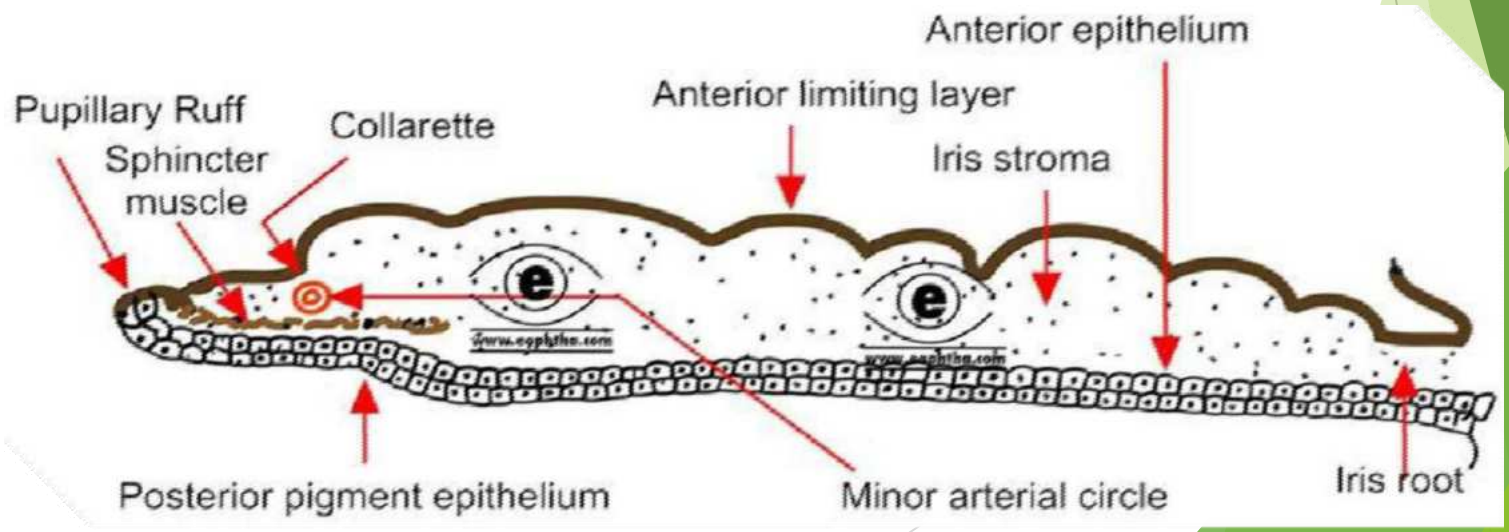
- **POSTERIOR SURFACE**

- Posterior surface of the iris is much more uniform.

- Posterior surface of the iris is darker than the anterior surface and shows numerous radial contraction folds.

MICROSCOPIC STRUCTURE

- Microscopically iris consist of four layers
- Anterior limiting membrane.
- Iris stroma
- Anterior epithelium layer
- Posterior pigmented epithelium layer



CILIARY BODY

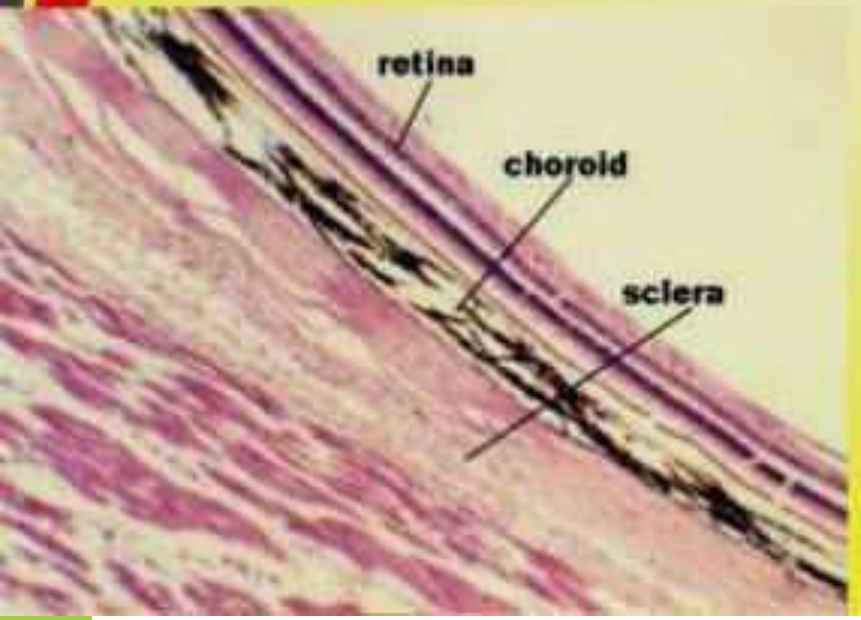
- It is the anterior portion of the uveal tract , which is located b/w the iris and the choroid.
- middle part of vascular coat of eyeball.
- Triangular in shape.
- Inner side of triangle is divided into two parts.
- The anterior portion - ***pars plicata***
- The posterior portion-***pars plana***
- The outer side of triangle lies against the sclera.

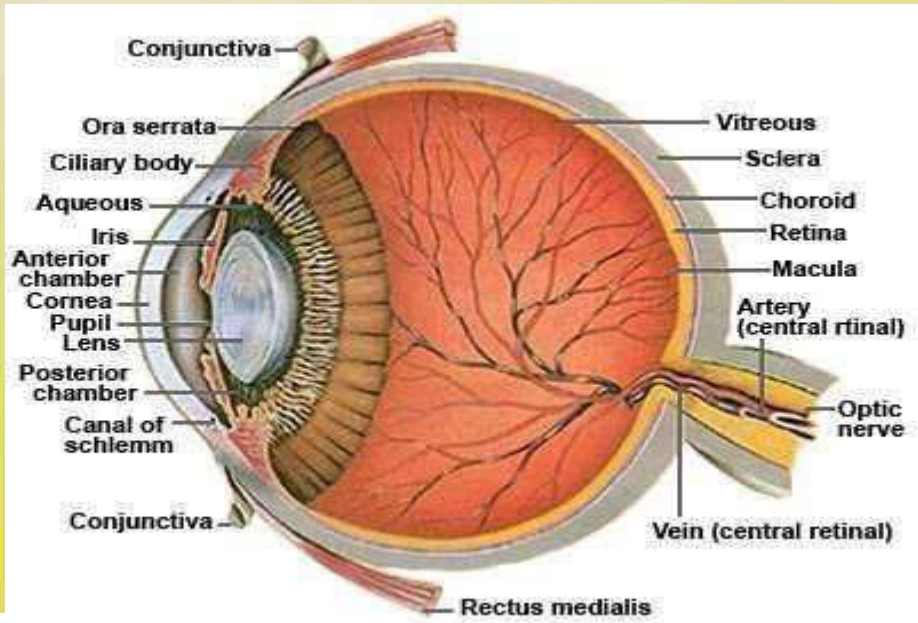
- Microscopically ciliary body consist of five layers.

  - Supraciliary lamina.
  - Stroma.
  - Layer of pigmented epithelium.
  - Layer of non pigmented epithelium.
  - Internal limiting membrane.

3. CHOROID

- Thin, highly pigmented, vascular loose connective tissue
- Rich in melanocytes gives characteristic dark color
- Situated between sclera & retina
- Extends from optic nerve to ciliary body (at ora serrata)
- Thickness decreases from post (0.22mm) to ant (0.1mm)





- **Choroid consist of four layers.**

- Suprachoroidal lamina.
- Stroma.
- Choriocapillaris.
- Bruch's membrane.

Blood supply of the uveal tract

- The **uveal tract** is supplied by three sets of artery.

- Short posterior ciliary artery-Arises as two trunks from the ophthalmic artery .
- ✓ It pierce the sclera around the optic nerve and supply the choroid in a segmental manner.
- Long posterior ciliary artery-- two in number nasal and temporal
- ✓ Pierce the sclera obliquely on medial and lateral side of the optic nerve and supply the ciliary body.

- Anterior ciliary artery– Derived from muscular branches of ophthalmic artery.
- ✓ 7 in numbers– 2 each of superior, medial, inferior rectus muscle and one from lateral rectus muscle
- ✓ these artery gives branches to sclera , limbus and conjunctiva.

NERVE SUPPLY

- The iris receives its sensory and autonomic nerve supply from the long and short ciliary nerves.

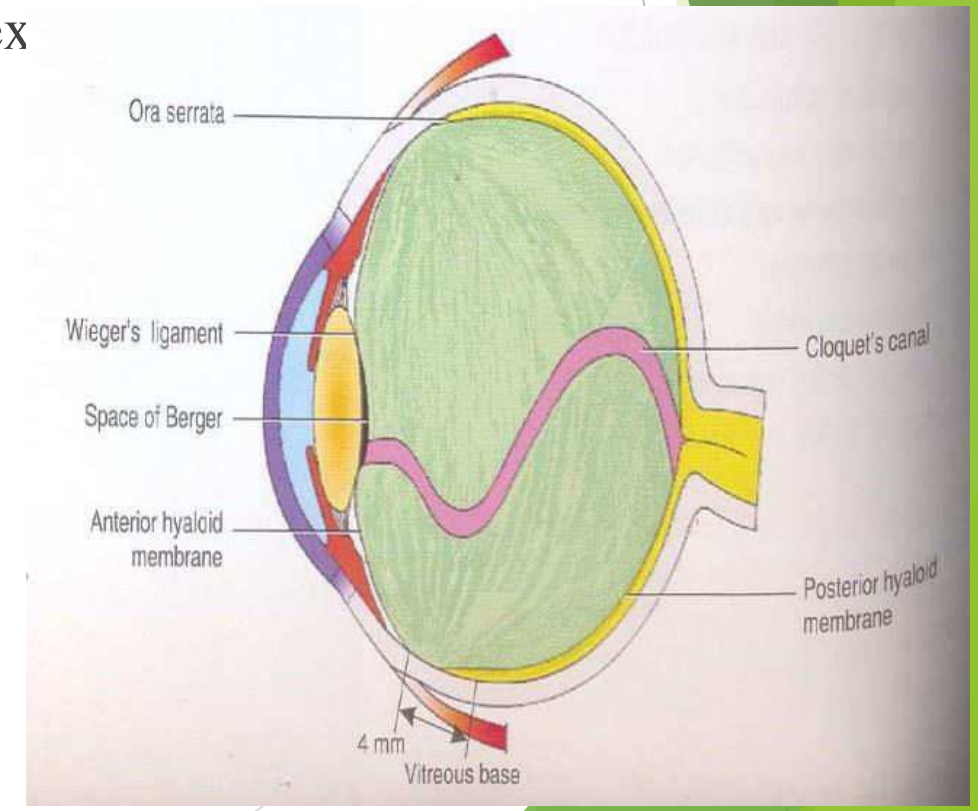
- The choroid is innervated by the long and short ciliary nerves

ANATOMY OF LENS

- **DEFINITION** :

  Lens is a transparent, biconvex structure

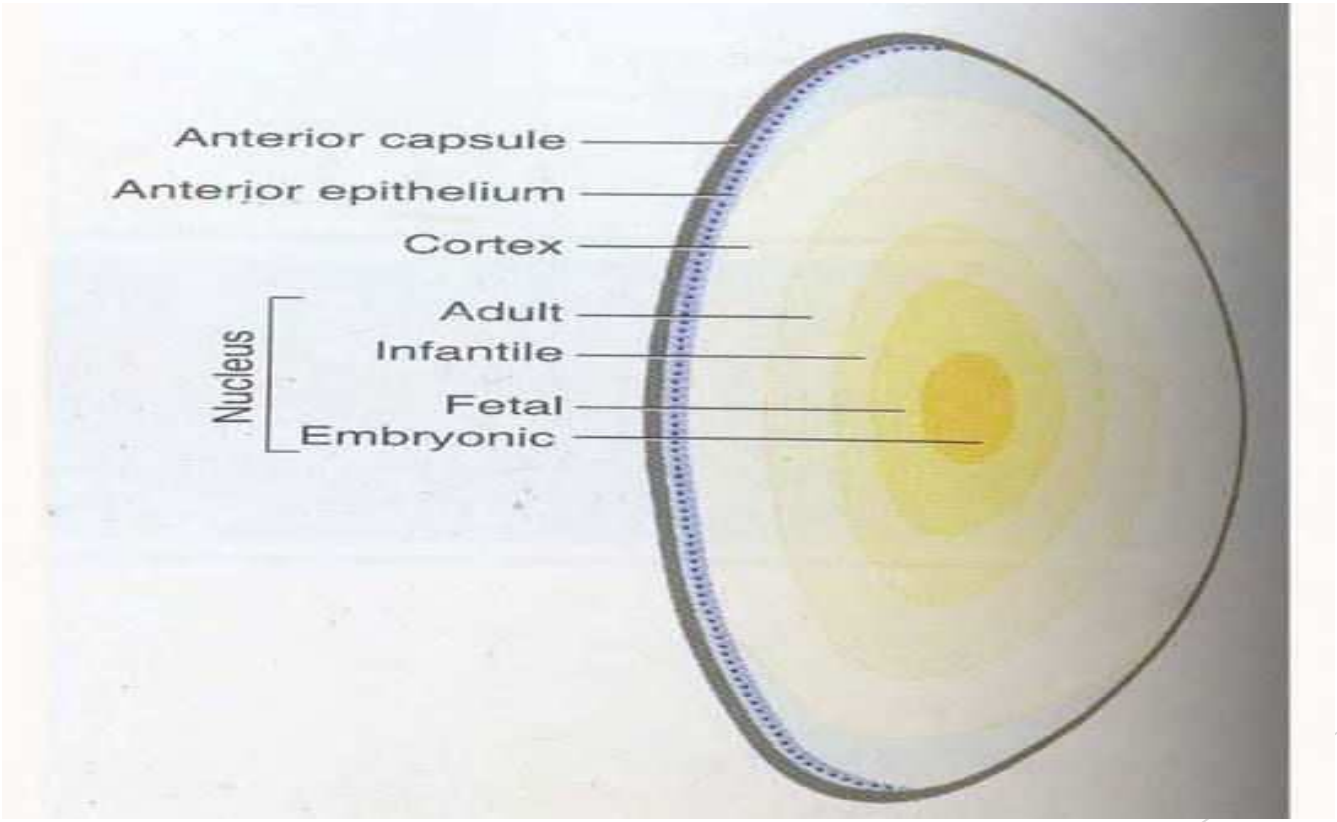
- **Position of Lens in Eye Ball :**
  - Lens lies between post surface of iris & the vitreous



- **Dimension of Lens**
  - Equatorial diameter of lens in adult is 9-10mm.
  - During birth approximately 6.5 mm.
  - Axial diameter (Thickness)
    - A birth about 3.5mm
    - At extreme of Age 5mm

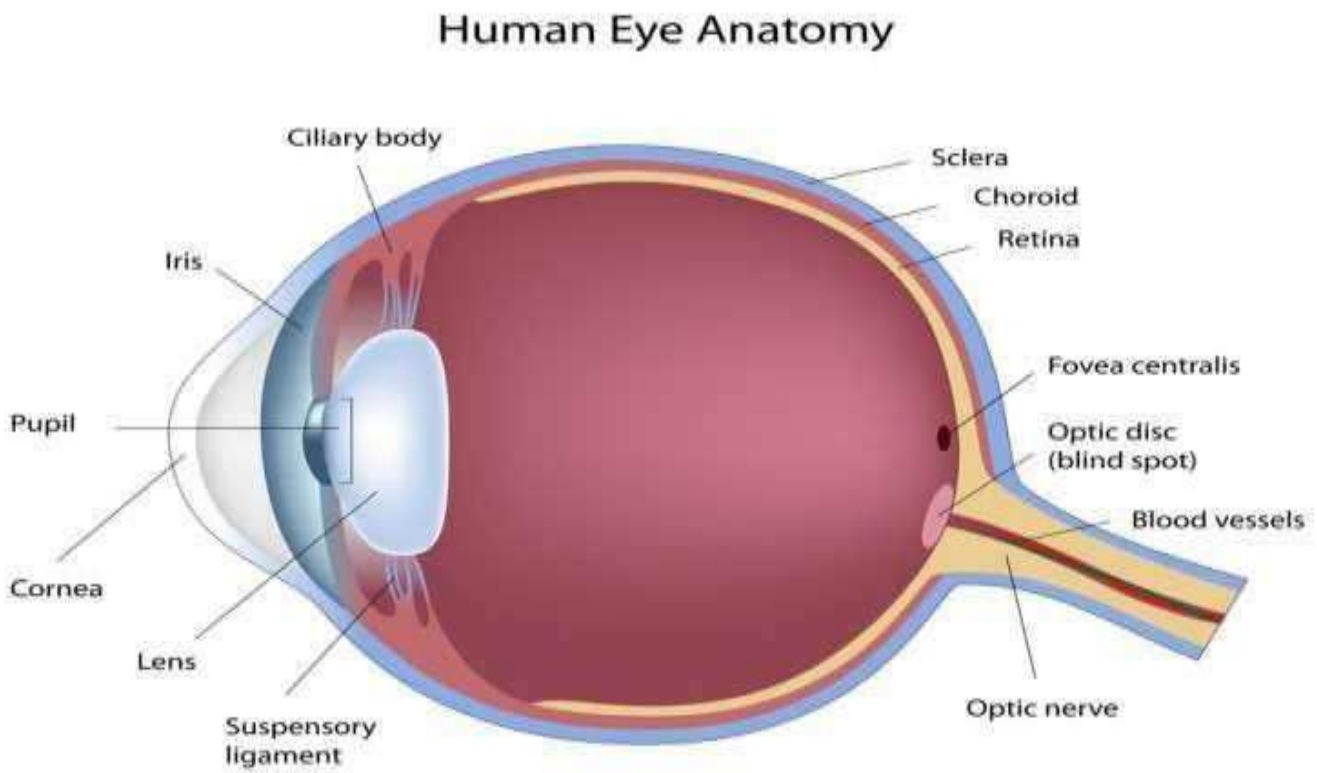
- **Surfaces of Lens**
- Two Surfaces – (i) Anterior (ii) Posterior
- The ant. Surface is less convex & is a part of sphere having radius of 8 to 14mm
- The post surface is more convex & is a part of sphere having radius of 4.5 to 7.5 mm.

- **PARTS OF LENS :**
- The Lens Capsule
- Ant Lens Epithelium and lens fibers
- Nucleus and cortex
- zonules


Anterior capsule
Anterior epithelium
Cortex
Nucleus
Adult
Infantile
Fetal
Embryonic

Inner most layers of eye ball

- Retina
- Retina is the innermost tunic of the eyeball
- Thin, delicate, transparent membrane
- Highly developed tissue of the eye
- Appears purplish red


Human Eye Anatomy
Ciliary body
Sclera
Choroid
Retina
Iris
Fovea centralis
Pupil
Optic disc
(blind spot)
Blood vessels
Cornea
Lens
Suspensory
ligament
Optic nerve

- Three distinct regions of retina:

1.optic disc

2.macula lutea

3.peripheral retina

Optic disc

- Pale pink in colour; well defined circular area
- Diameter: 1.5mm
- All the retina layers terminate here, except the nerve fibre which pass through the lamina cribrosa

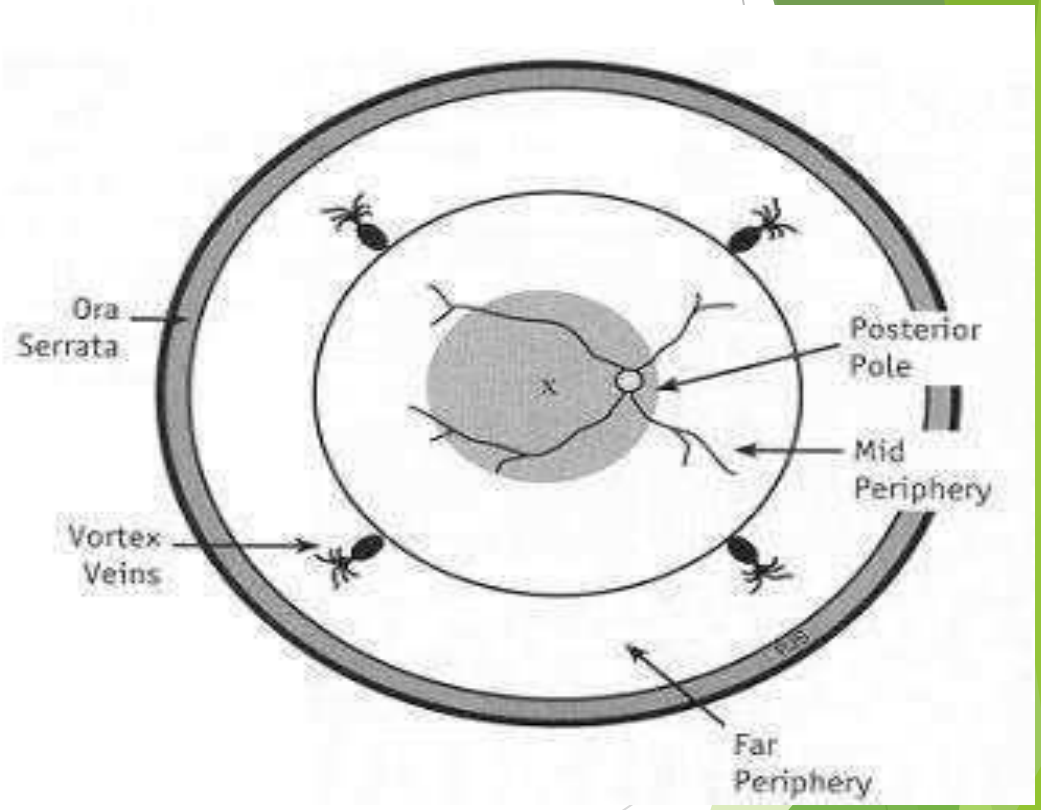
Macula lutea

- The macula lutea is comparatively dark area, 5.5 mm in diameter , situated at the posterior pole of the eyeball , temporal to optic disc , also called as yellow spot or area centralis.
- Primary function :- photoptic vision

- Fovea centralis is central depression in macula; measuring 1.85 mm in diameter & 0.25 thickness
- It is most sensitive part of retina

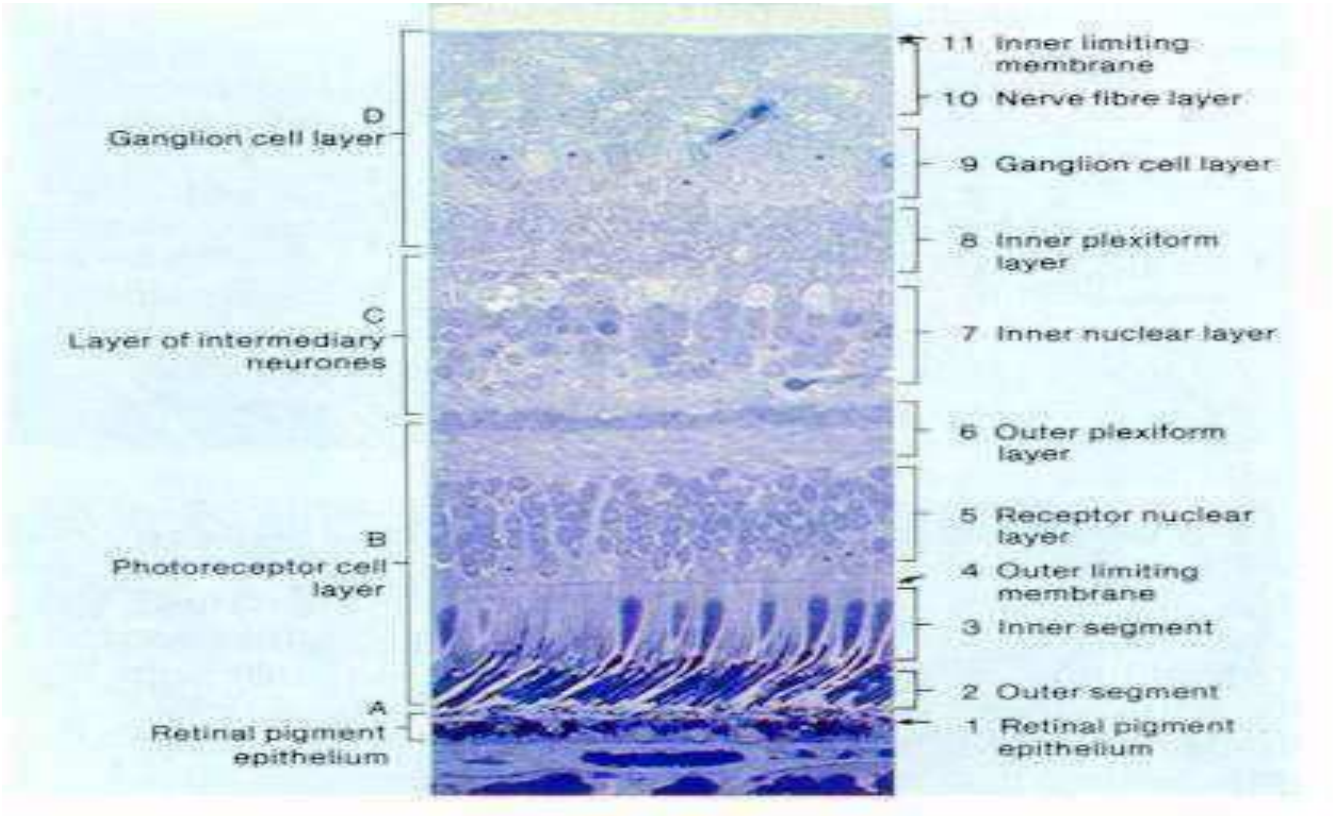
- **Peripheral retina**

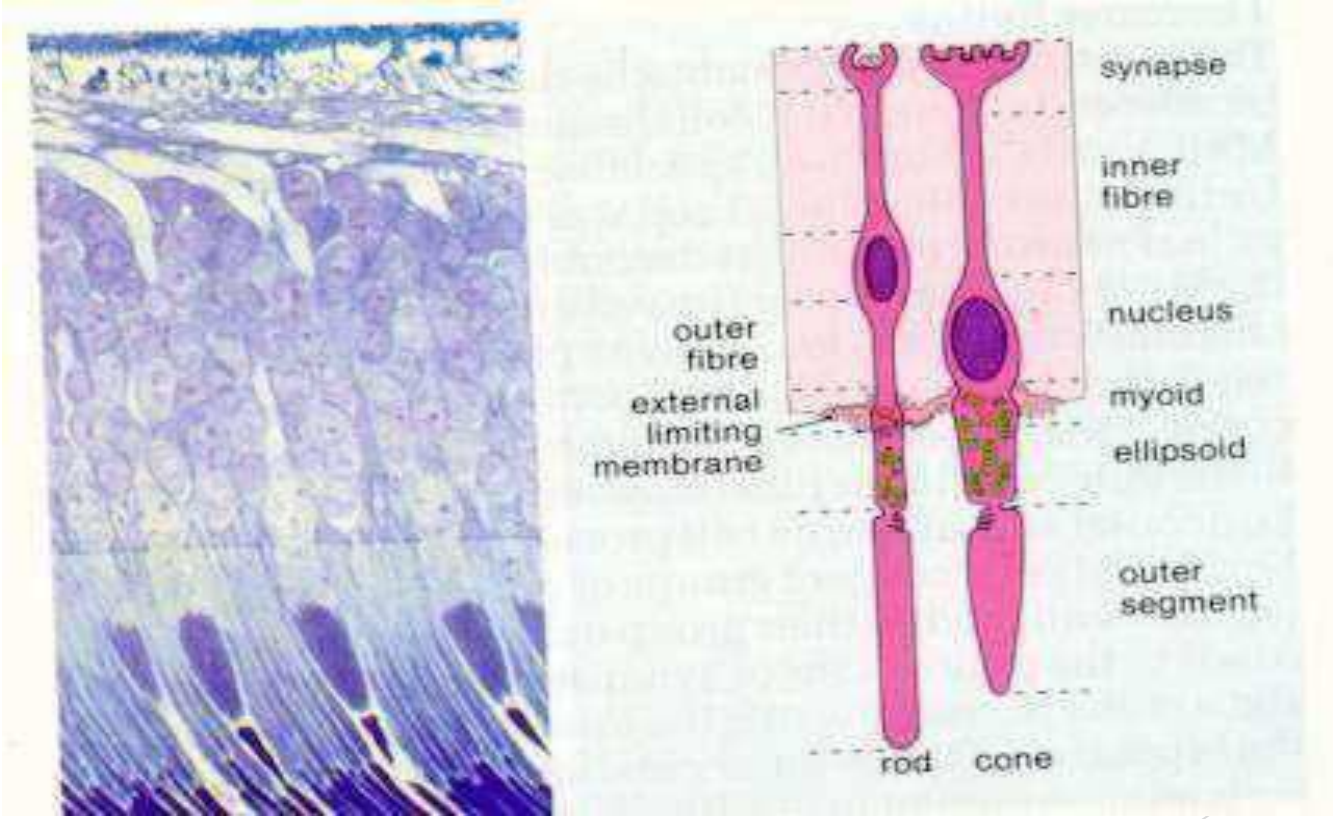
1. Near periphery
2. Mid periphery
3. Far periphery
4. Ora serrata



Microscopic structure of the retina

1. Internal limiting membrane
2. Nerve fiber layer
3. Ganglion cell layer
4. Inner plexiform layer
5. Inner nuclear layer
6. Outer plexiform layer
7. Outer nuclear layer
8. External limiting membrane
9. Photoreceptor layer (rods and cones)
10. Retinal pigment epithelium


D
Ganglion cell layer
C
Layer of intermediary neurones
B
Photoreceptor cell layer
A
Retinal pigment epithelium
11 Inner limiting membrane
10 Nerve fibre layer
9 Ganglion cell layer
8 Inner plexiform layer
7 Inner nuclear layer
6 Outer plexiform layer
5 Receptor nuclear layer
4 Outer limiting membrane
3 Inner segment
2 Outer segment
1 Retinal pigment epithelium


synapse
inner fibre
nucleus
outer fibre
external limiting membrane
myoid
ellipsoid
outer segment
rod
cone

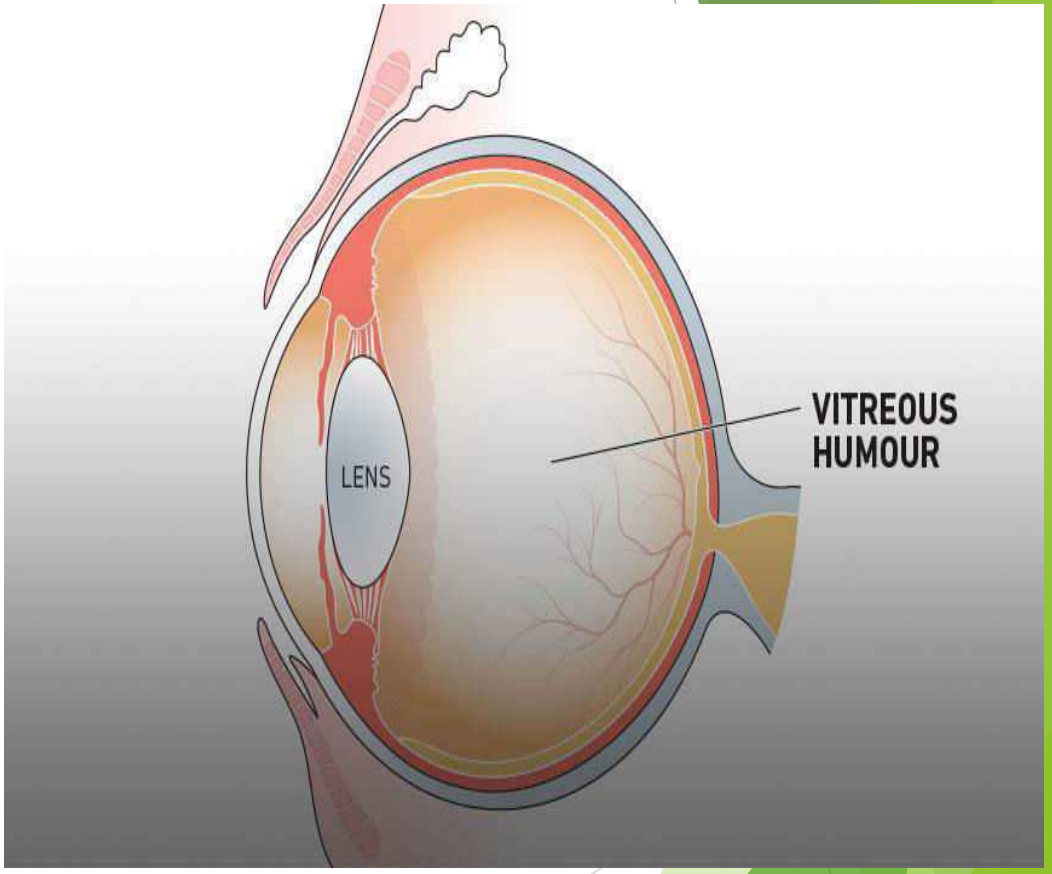
Blood supply of the retina

- The neural retina has a dual blood supply derived from branches of the ophthalmic artery, including the central retinal artery(which provides the retinal circulation) and the posterior ciliary arteries(which provides the choroidal circulation).

- Anatomically, the retinal circulation support the inner two-thirds of the retina, whereas the choroidal circulation supports the outer third of the retina.

Chamber of the eye

- Anterior chamber
- Posterior chamber
- Vitreous chamber



Anterior and Posterior chamber

- Aqueous is a thin, watery fluid located in the anterior and posterior chambers of the eye.
- The anterior chamber lies between the iris (colored part of the eye) and the inner surface of the cornea (the front of the eye).
- The posterior chamber is located behind the iris and in front of the lens.
- In addition to supporting the shape of this area, aqueous supplies nutrients and nourishment to parts of the eye that lack blood supply.

- It also removes waste.
- Improper drainage of the aqueous humor can cause an increase in intraocular pressure (pressure inside the eye).
- This increase can result in loss of vision or contribute to the development of glaucoma

Vitreous chamber

- vitreous humour (also known simply as the vitreous) is a clear, colourless fluid that fills the space between the lens and the retina of eye.
- 99% of it consists of water and the rest is a mixture of collagen, proteins, salts and sugars.
- Despite the water-to-collagen ratio, the vitreous has a firm jelly-like consistency.

Function of the vitreous

- The vitreous performs a vital role in protecting the eye.
- Most importantly, it helps it to hold its ‘spherical’ shape.
- The vitreous also comes in contact with the retina (the light-sensitive tissue at the back of the eye that acts like the film of a camera).
- The pressure of the vitreous humour helps to keep the retina in place.

Thank You

VISUAL STANDARDS AND BLINDNESS

DR SAMINA
AP OPHTHALMOLOGY

VISUAL STANDARDS

- Many occupations and activities require people to have particular levels of vision.
- For which we require certain visual standards.
- Visual standards are needed for personal and public safety.
- What professions require visual standards?
    - Motor vehicle drivers
    - Train operators
    - People in the maritime industry
    - Metropolitan Ambulance
    - People in the aviation industry
    - Defence Force and Federal Police
    - Metropolitan Fire and Emergency Services

- Visual standards are also required for
    - Operators of Cranes and forklifts
    - Laser users
    - People involved in motor sports
- Visual standards are used by
    - General Practitioners
    - Medical Specialists
    - Optometrists
    - Psychologists
    - Physiotherapists
    - Occupational therapists

- So Vision standard is define as

the minimum expected level of vision that is required for the efficient and safe performance of tasks of an individual at the workplace.

TESTS FOR VISUAL STANDARD

- Visual acuity
- Colour vision
- Visual field
- Binocular function

VISUAL ACUITY

- A measure of how clearly you can see
- Measured with a letter chart at a distance (usually 6m)
- Decreased by refractive error, cataract, etc

COLOUR VISION DEFECTS

- Most commonly red/green
- 8% of males, 0.5% of females
- Affects
  - colour discrimination,
  - colour matching and career choices

VISUAL FIELD

- A measure of how well you can see with your side vision
- Decreased with eye disease
  - Glaucoma
  - Retinitis Pigmentosa
- Computerised test

BINOCULAR VISION

- Two eyes work together for full depth perception or “stereopsis”.

- Glasses can help eyes focus equally

BLINDNESS

DEFINITIONS

- There are 4 levels of visual function, according to the International Classification of Diseases.

- NORMAL VISION
- MODERATE VISUAL IMPAIRMENT
- SEVERE VISUAL IMPAIRMENT
- BLINDNESS.

NORMAL VISION

- Visual acuity is usually measured with a Snellen chart.
- The Snellen chart displays letters of progressively smaller size.
- "Normal" vision is 20/20.
- This means that the test subject sees the same line of letters at 20 feet that a normal person sees at 20 feet

GENERAL CONCEPT OF LOW VISION AND BLINDNESS

- **Low vision**
- Best corrected visual acuity in the better eye less than 6/18 and/or visual field less than 20 degree from the point of fixation.
- '**Blindness**'
- **defined** as the best corrected visual acuity in the better eye less than 3/60, and/or visual field less than 10 degree from the point of fixation.

WHO CLASSIFICATION/CRITERIA FOR BLINDNESS

SN	Visual acuity (Snellen notation)	Classification	Grading
1	≥ 6/18	Normal/Near Normal	NORMAL
2	<6/18- 6/60	Moderate VI	LOW VISION
3	<6/60-3/60	Sever VI	
4	<3/60-PL	Legally Blind	BLINDNESS
5	NPL	Blind	

MAGNITUDE OF PROBLEM

- Estimated 180 million people are visually disabled, nearly 45 million blind, 4 out of 5 living in developing countries.

TYPES OF BLINDNESS

- Economic Blindness
- Social Blindness
- Manifest Blindness
- Absolute Blindness
- Curable Blindness
- Preventable Blindness
- Avoidable Blindness

- Visual Acuity:- Sharpness of vision, measured as maximum distance a person can see a certain object, divided by the maximum distance at which a person with normal sight can see the same object.

- Economic blindness:- – Inability of a person to count fingers from a distance of 6 meters or 20 feet.

- Social blindness:-
    - Vision 3/60 or diminution of field of vision to 10 degrees
- Manifest blindness:-
    - Vision 1/60 to just perception of light.
- Absolute blindness:-
    - No perception of light
- Curable blindness:-
    - That stage of blindness where the damage is reversible by prompt management e.g. cataract
- Preventable blindness:-
    - The loss of vision that could have been completely prevented by institution of effective preventive or prophylactic measures.

LEGAL BLINDNESS

- Is a level of vision loss that has been legally defined to determine eligibility for benefits.
- The clinical diagnosis refers to a central visual acuity of 20/200 (3/60) or less in the better eye with the best possible correction, and/or a visual field of 20 degrees or less.

GLOBAL CAUSES OF BLINDNESS

- Cataract ,
- Glaucoma
- DM
- Vascular disease
- Accidents & degeneration of ocular tissue
- Leading causes of childhood blindness
    - Xerophthalmia,
    - congenital cataract,
    - congenital glaucoma &
    - optic atrophy.

EPIDEMIOLOGICAL DETERMENTS

- **Age:** ◦
- In children & young:
    - Refractive error,
    - trachoma,
    - conjunctivitis,
    - malnutrition.
- In adults:
    - cataract,
    - refractive error,
    - glaucoma,
    - DM
- **Sex:** ◦
    - Higher prevalence of trachoma, conjunctivitis and cataract in women leading to higher prevalence of blindness in women

- **Malnutrition:** ◦
  - Infectious diseases of childhood especially measles & diarrhoea
  - Severe blinding corneal destruction due to vit. A deficiency in first 4 to 6 years of life.
-
- **Occupation:** ◦
  - People working in factories, workshop, industries are prone to eye injuries because of exposure to dust, airborne particles, flying objects, gases, fumes, radiation.

- **Social class:** ◦
  - Surveys indicate that blindness twice more prevalent in poorer classes than in the well to do.

- **Social factors:** ◦
  - Basic social factors are ignorance, poverty, low standards of personal and community hygiene and inadequate health care services.

PREVENTION OF BLINDNESS

- The components for action in national programmes for the prevention of blindness comprise the following
- Initial assessment
- Methods of intervention
    - primary eye care
    - secondary care
    - tertiary care
- Long term measures

METHODS OF INTERVENTION

- **Primary care**
- Wide range of eye conditions can be treated or prevented at grass root level by locally trained health workers who are first to make contact with the community.
- They are also trained to refer the difficult cases to the nearest PHC or district hospital.
- Their activities also involve promotion of personal hygiene, sanitation, good dietary habits and safety in general.

- **Secondary care:**

- Involves definitive management of common blinding conditions as cataract, trichiasis, entropion, ocular trauma, glaucoma.

- It is provided in PHCs and district hospitals where eye depts are established.

- May involve the use of mobile eye clinics

- **Tertiary care**

- Established in the national or regional capitals and are often associated with medical colleges and institutes of medicine.

- Provide sophisticated eye care such as retinal detachment surgery, corneal grafting which are not available in the secondary centres.

- Other measures of rehabilitation comprise education of blind in the special schools & utilisation of their services in the gainful employment

SPECIFIC PROGRAMMES FOR CONTROL OF BLINDNESS

- Trachoma control
- School eye health services: Screening and treatment , Health education
- Vit. A prophylaxis
- Occupational eye health services

LONG TERM MEASURES

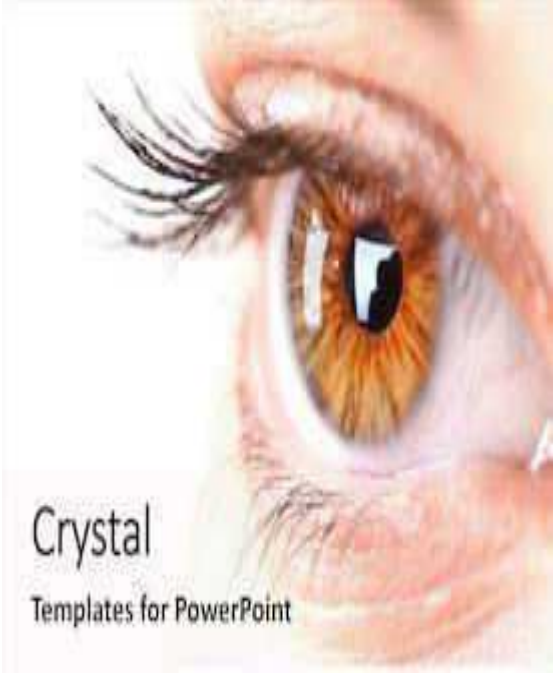
- Aimed at improving quality of life and modifying the factors responsible for eye problems.
- Poor sanitation
- Lack of adequate safe water supply
- Poor nutrition
- Lack of personal hygiene

VISION 2020

- The Right to Sight is the global initiative for the elimination of avoidable blindness, a joint programme of the World Health Organization (WHO) and the International Agency for the Prevention of Blindness (IAPB).

- It was launched in 1999 to promote: "A world in which nobody is needlessly visually impaired, where those with unavoidable vision loss can achieve their full potential."

Thanks



PUPILLARY REFLEXES

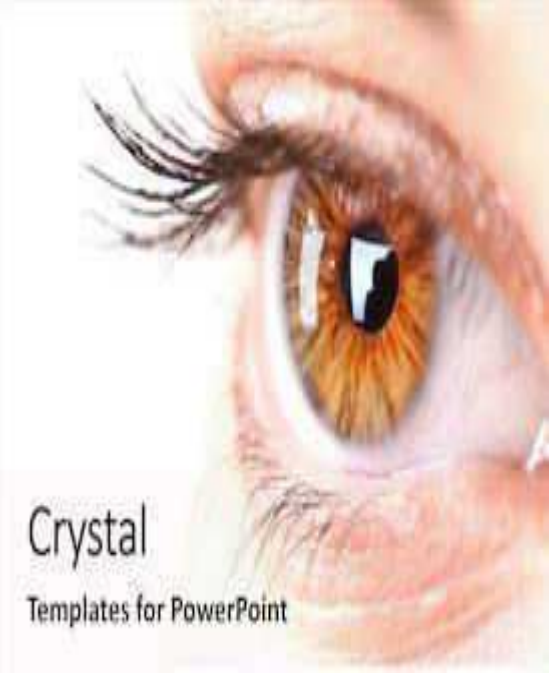
DR SAMINA

AP OHTHALMOLOGY

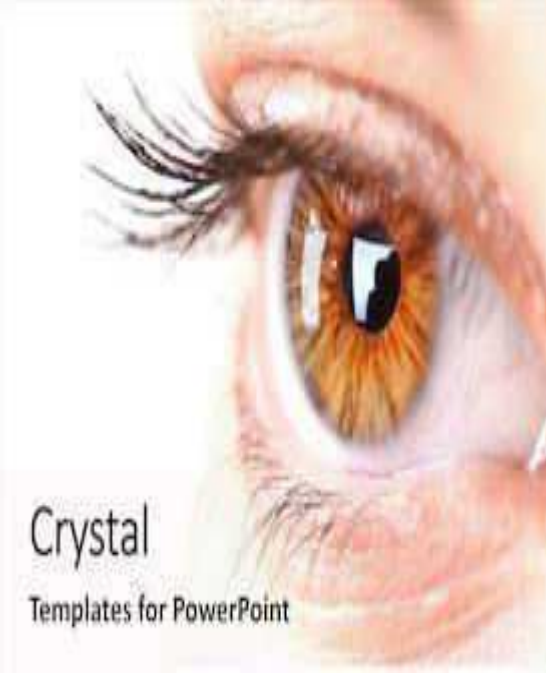
Objectives

1. what are normal pupillary reflexes

2. what are the abnormal pupillary reflexes



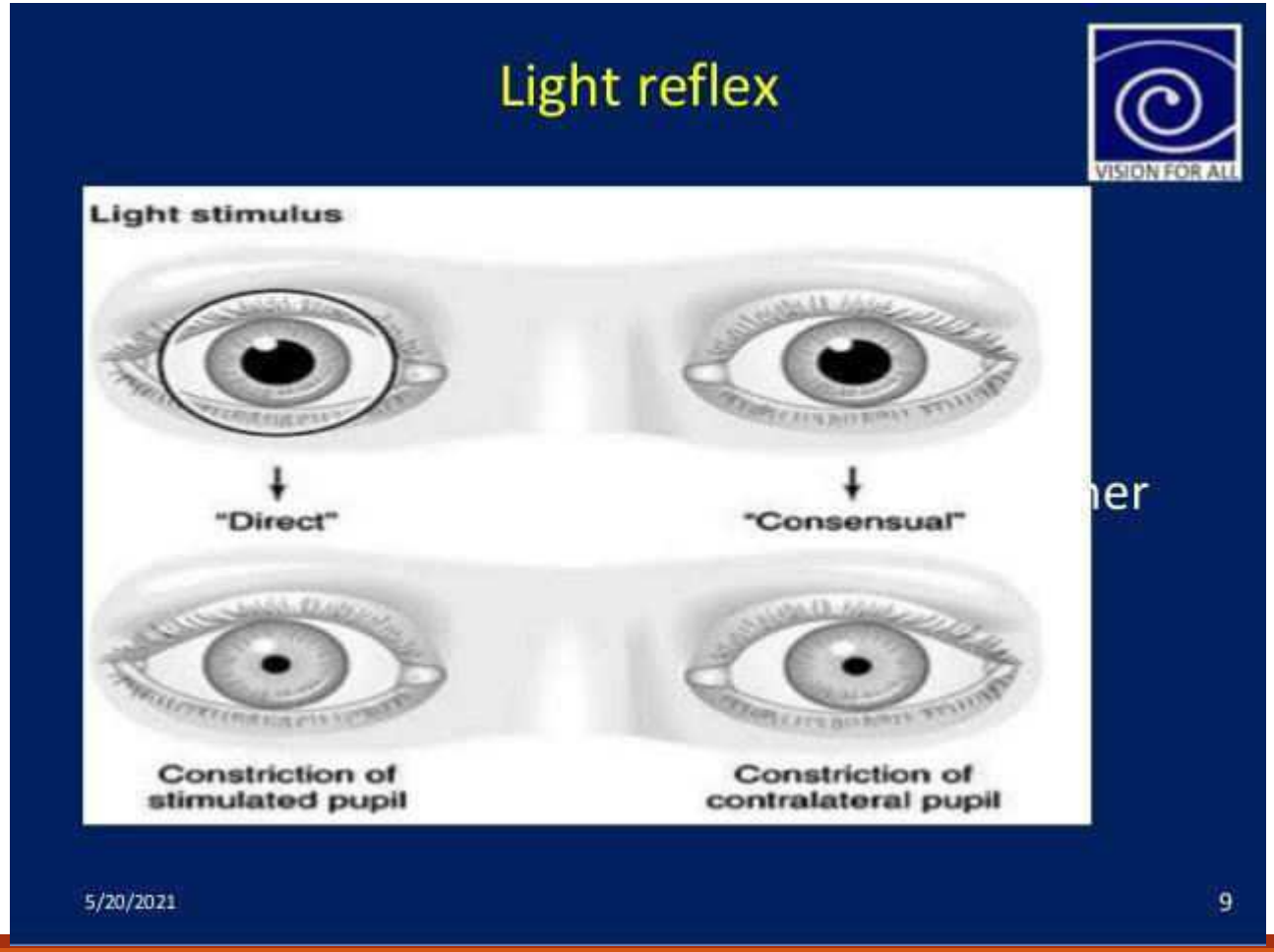
NORMAL PUPILLARY REFLEXES



- LIGHT REFLEX
- NEAR REFLEX
- OTHER REFLEXES
  - DARKNESS REFLEX
  - PSYCHOSENSORY REFLEX
  - LID CLOSURE REFLEX

LIGHT REFLEX

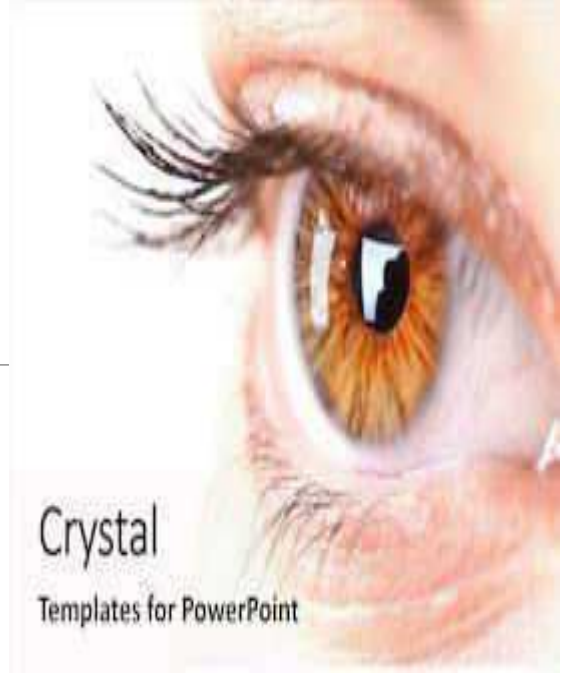
- DIRECT
- CONSENSUAL



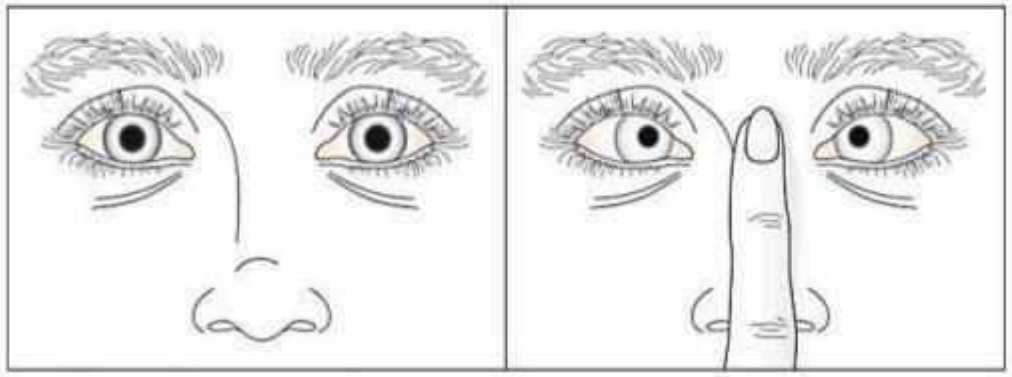
NEAR REFLEX

Near reflex is a Triad of:

- Inc. accommodation
- Convergence of visual axes
- Constriction of the pupils



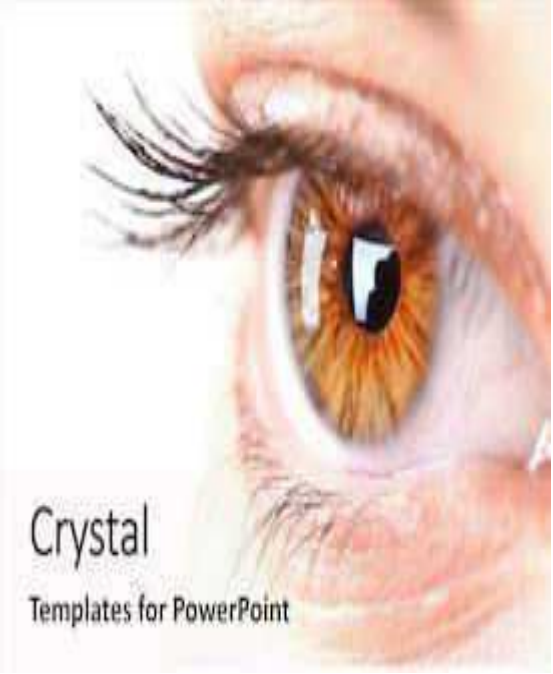
- **Accommodation reflex:**
  - **The patient is asked to look at a distant object and then at an object close to his face.**
  - **Both pupils should constrict and dilate again when distant gaze is resumed**

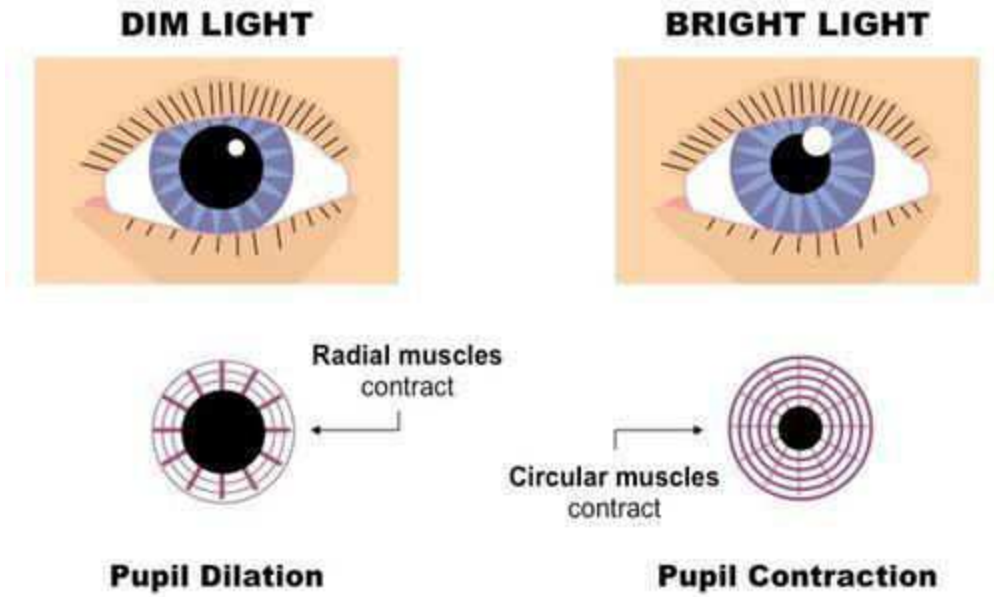


DARKNESS REFLEX

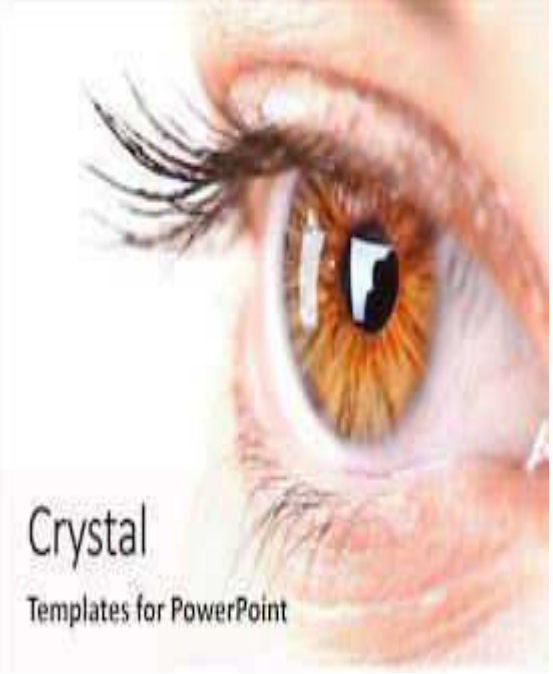
Abolition of light reflex – relaxation of sphincter pupillae and

Contraction of dilator pupillae – dilatation of the pupil -----supplied by sympathetic nervous system





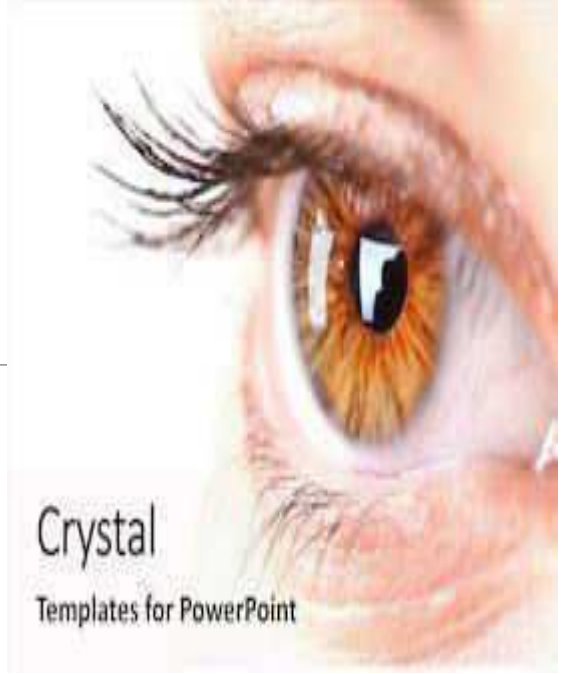
PSYCHOSENSORY REFLEX



- Dilatation of pupil in response to sensory & psychic stimuli
- Fully developed by 6 months of age
- Pathways – unknown
- Two components –
  - Sympathetic discharge to dilator pupillae muscle
  - Inhibition of parasympathetic discharge to sphincter pupillae muscle PSYCHOSENSORY REFLEX

LID CLOSURE REFLEX

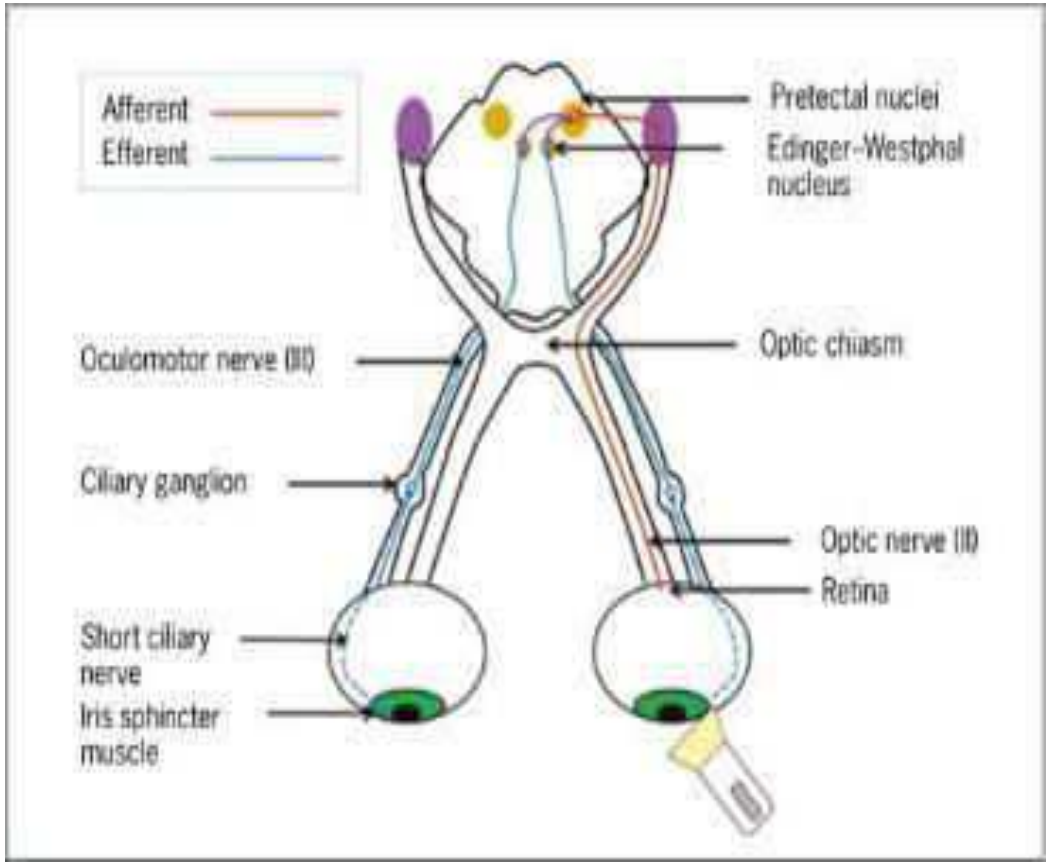
Constriction of pupil associated with blinking –



Normal visual pathway to the iris muscle

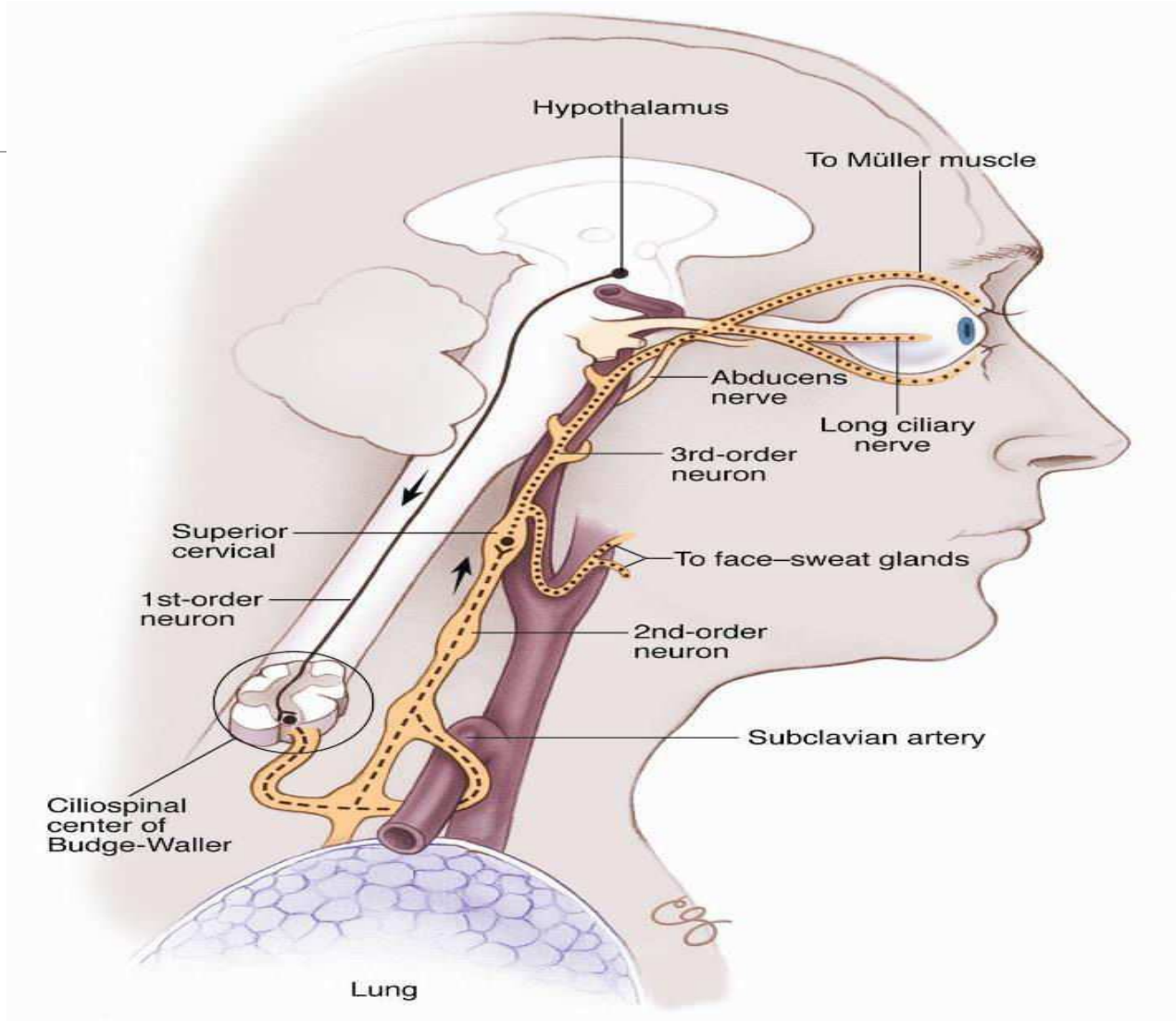
Parasympathetic pathway

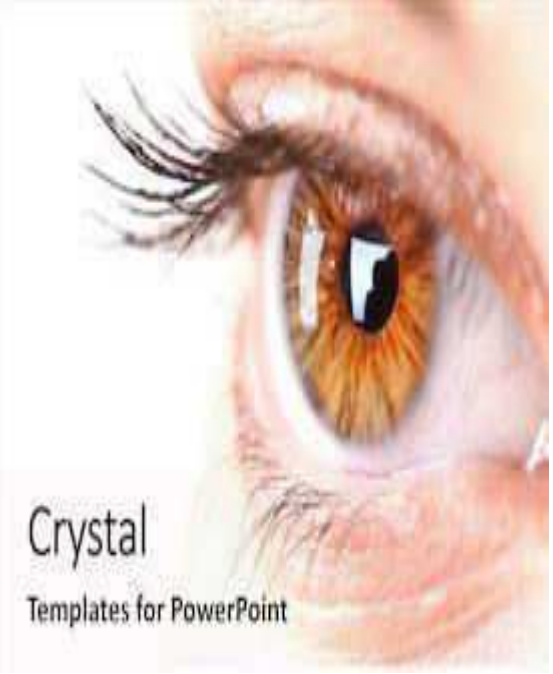
- Supply to sphincter muscle of the iris



Sympathetic pathway

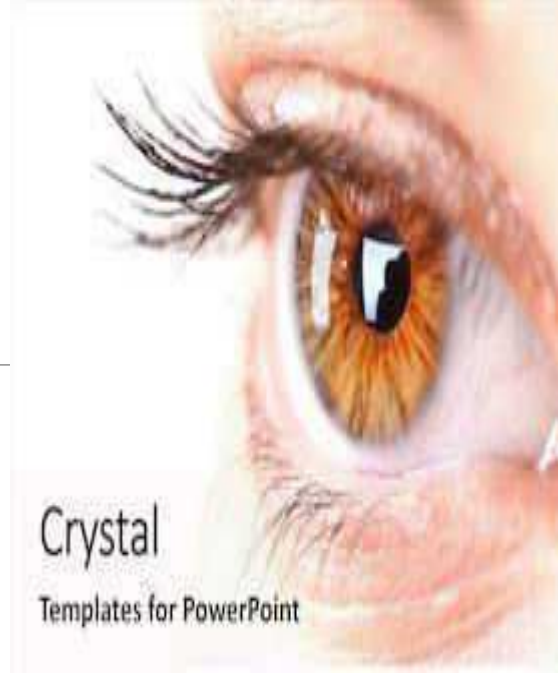
- Supply to Dilator muscle of the iris





ABNORMALITIES OF PUPILLARY REFLEXES

Abnormalities of Pupillary Reflexes



PARASYMPATHETIC PARESIS

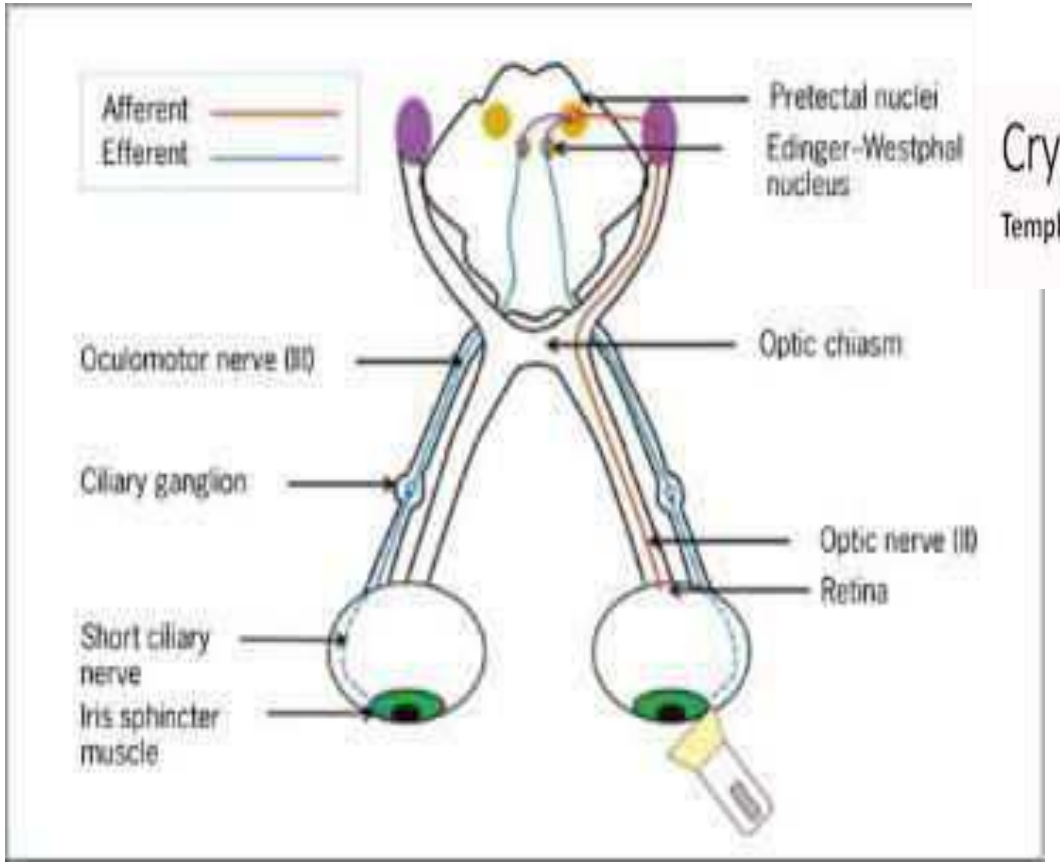
- AFFERENT PATHWAY DEFECTS
  - Total afferent pathway defect
  - Relative afferent pathway defect
  - Wernicke's hemianopic pupil
- EFFERENT PATHWAY DEFECTS
  - Tonic pupil
  - Oculomotor nerve palsy
  - Pharmacologic mydriasis
- PUPILLARY LIGHT-NEAR DISSOCIATION
  - Argyll Robertson pupil

SYMPATHETIC PARESIS

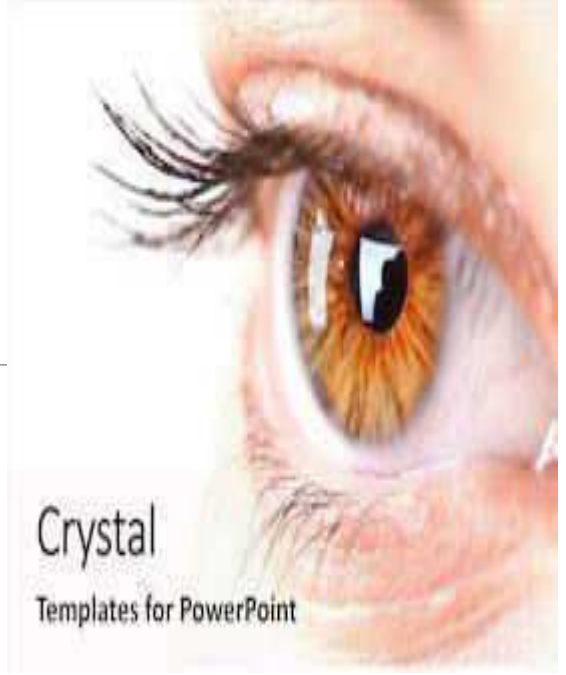
- Horner's syndrome

AFFERENT PATHWAY DEFECTS

- Total afferent pathway defect
- Relative afferent pathway defect
- Wernicke's hemianopic pupil



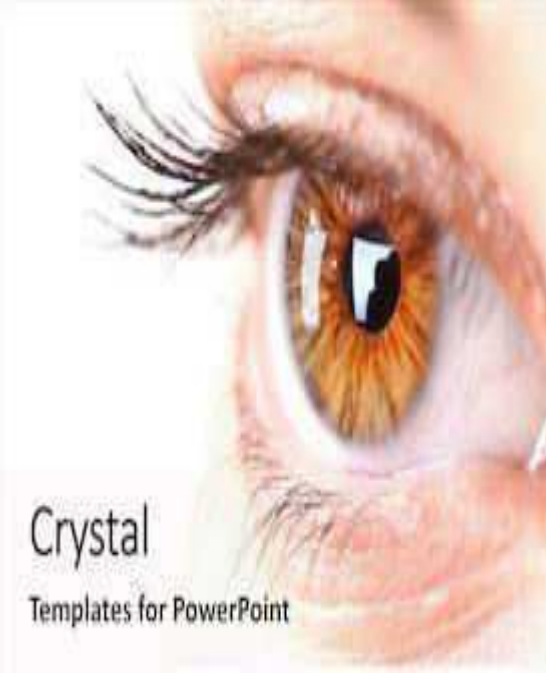
TOTAL/ABSOLUTE AFFERENT PATHWAY DEFECT



An absolute afferent pupillary defect (amaurotic pupil) is caused by a complete optic nerve lesion and is characterized by the following:

- The involved eye is completely blind (i.e. no light perception).
- Both pupils are equal in size.
- When the affected eye is stimulated by light neither pupil reacts.
- When the normal eye is stimulated both pupils react normally.
- The near reflex is normal in both eyes

Relative afferent pupillary defect



- A relative pupillary defect (Marcus Gunn pupil) is caused by an incomplete optic nerve lesion or severe retinal disease, but never by a dense cataract.
- The clinical features are those of an amaurotic pupil but more subtle.
- Thus the pupils respond weakly to stimulation of the diseased eye and briskly to that of the normal eye.
- The difference between the pupillary reactions of the two eyes is highlighted by the ‘swinging flashlight test’
- in which a light source is alternatively switched from one eye to the other and back, thus stimulating each eye in rapid succession.

A left relative defect is characterized by the following

Relative pupillary defect
(Marcus Gunn pupil)

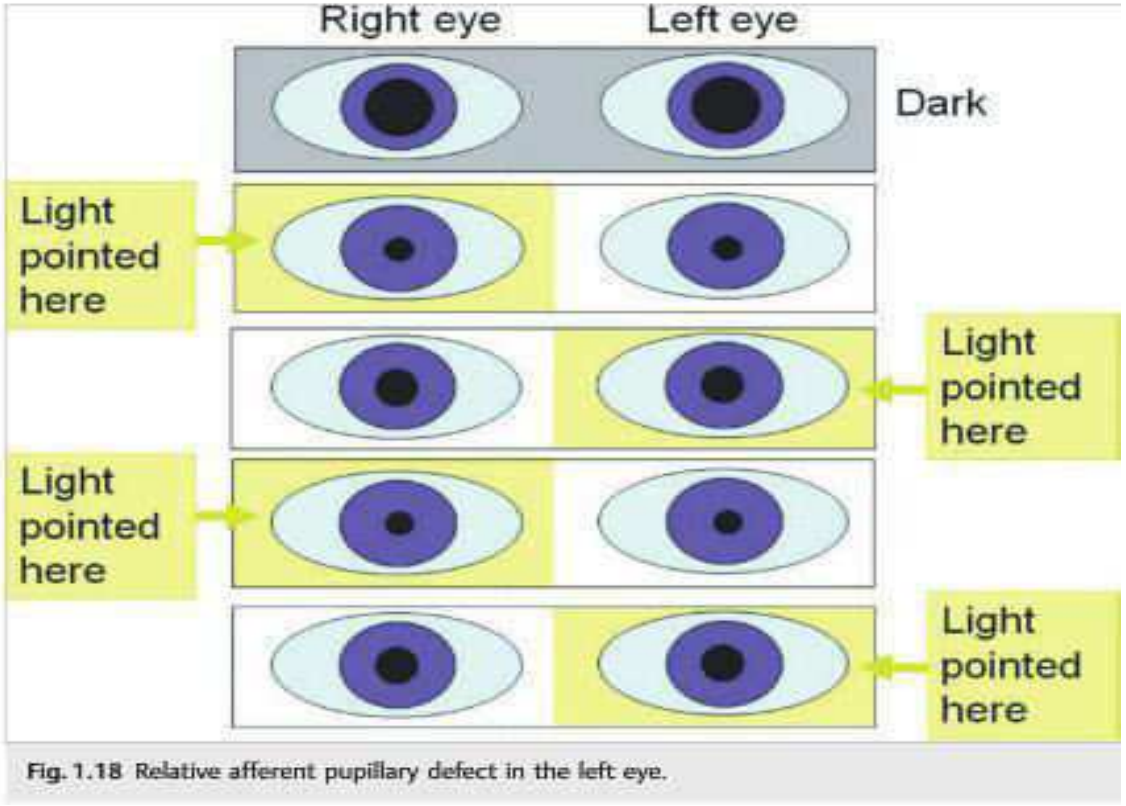
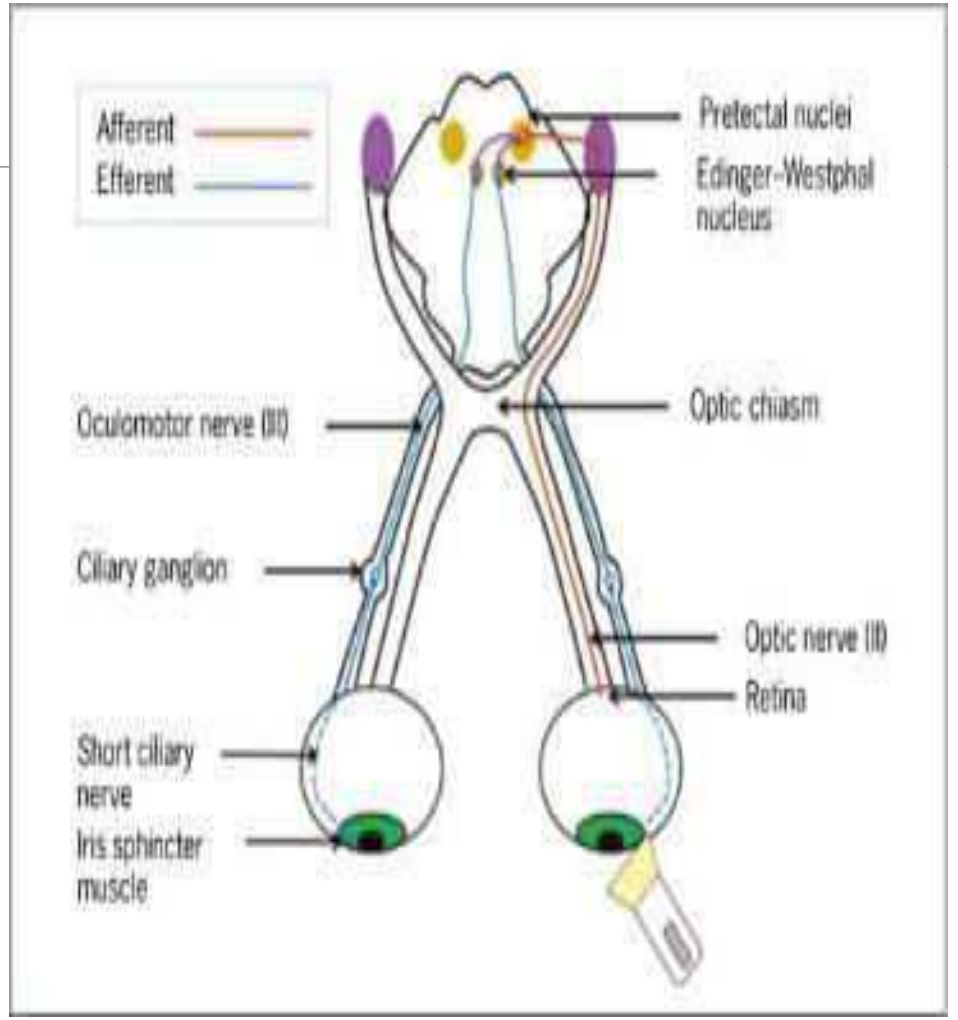


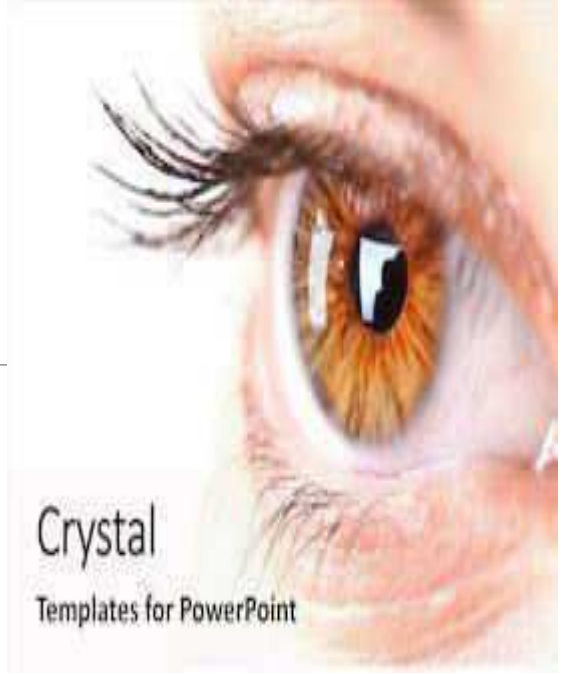
Fig. 1.18 Relative afferent pupillary defect in the left eye.

EFFERENT PATHWAY DEFECTS

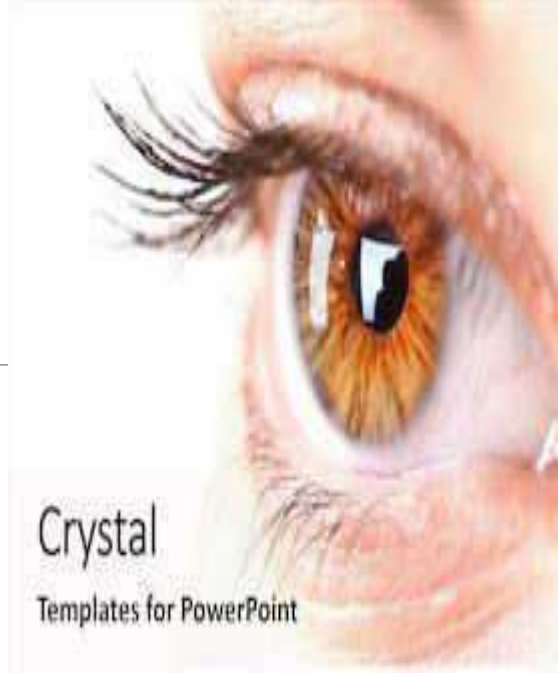
- Tonic pupil
- Oculomotor nerve palsy
- Pharmacologic mydriasis



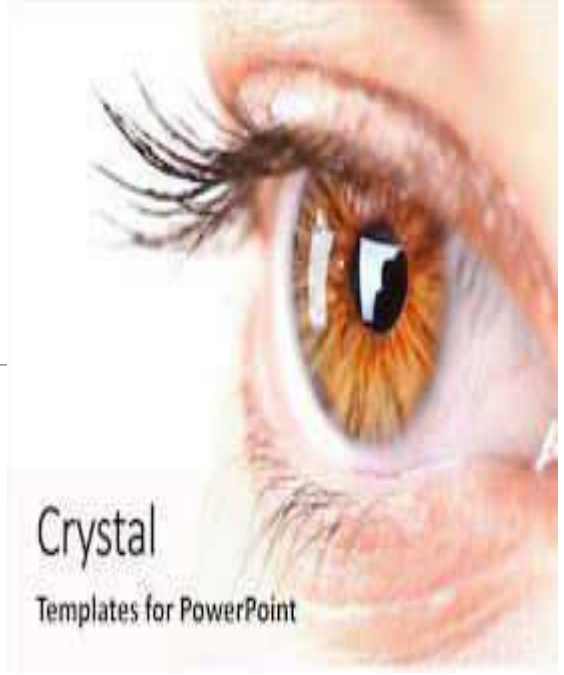
Adie /Tonic pupil



- An Adie pupil (tonic pupil, Adie syndrome) is caused by denervation of the postganglionic parasympathetic supply to the sphincter pupillae and the ciliary muscle,
- **Causes**
  - viral illness.
  - inherited in an AD pattern.
- Sites of dysfunction are presumed to be the ciliary ganglion.
- Affect young women



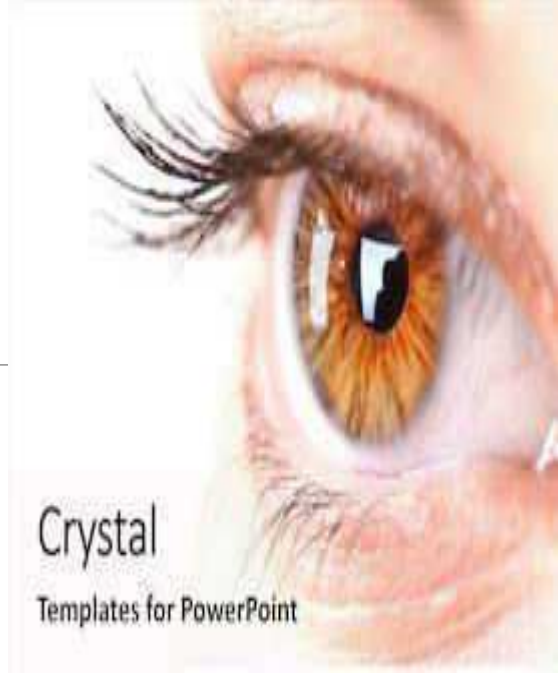
- **Symptoms**.
- Patients may notice anisocoria, or may have blurring for near due to impaired accommodation.
- **Signs**
- Large, regular pupil
- The direct light reflex is absent or sluggish
- On slit lamp examination, vermiform movements of the pupillary border are typically seen.
- Constriction is also absent or sluggish in response to light stimulation of the fellow eye (consensual light reflex)
- The pupil responds slowly to near, following which re-dilatation is also slow.



Pharmacological testing.

Instillation of 0.1–0.125% pilocarpine into both eyes leads to constriction of the abnormal pupil due to denervation hypersensitivity, with the normal pupil unaffected.

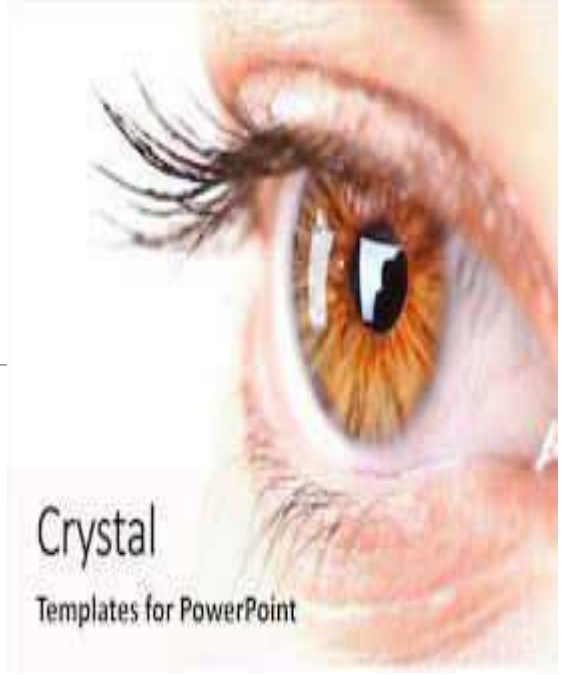
Pharmacological mydriasis



- Dilatation of one or both pupils due to instillation of a mydriatic agent can be inadvertent

- The pupil does not constrict in bright light or on accommodation and there is no response to any concentration of pilocarpine.

- There are no other neurological features.



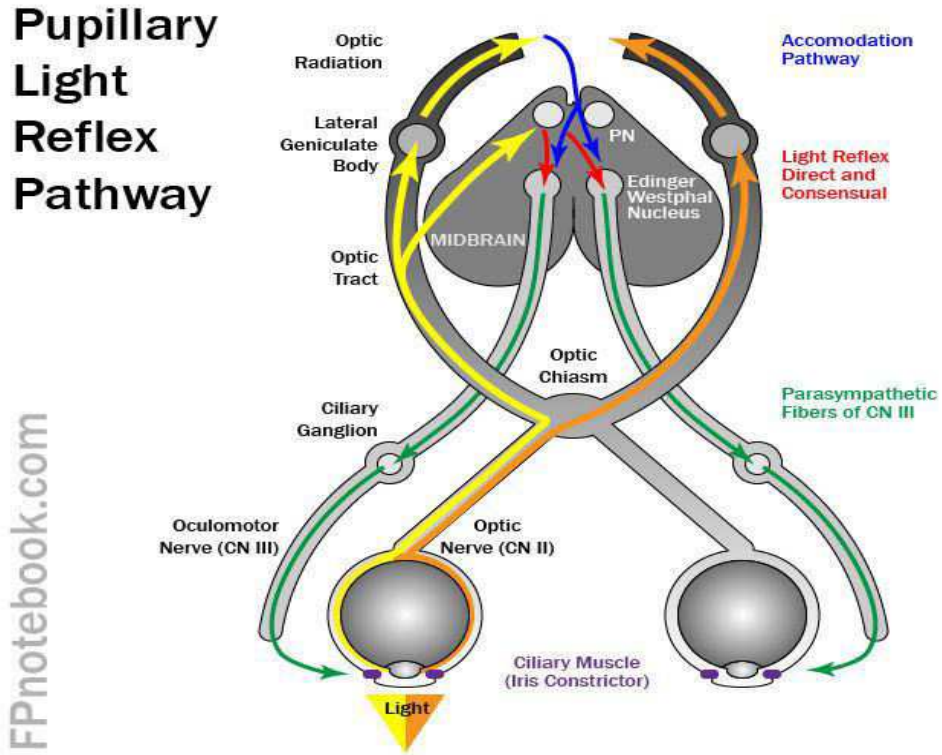
PUPILLARY LIGHT-NEAR DISSOCIATION

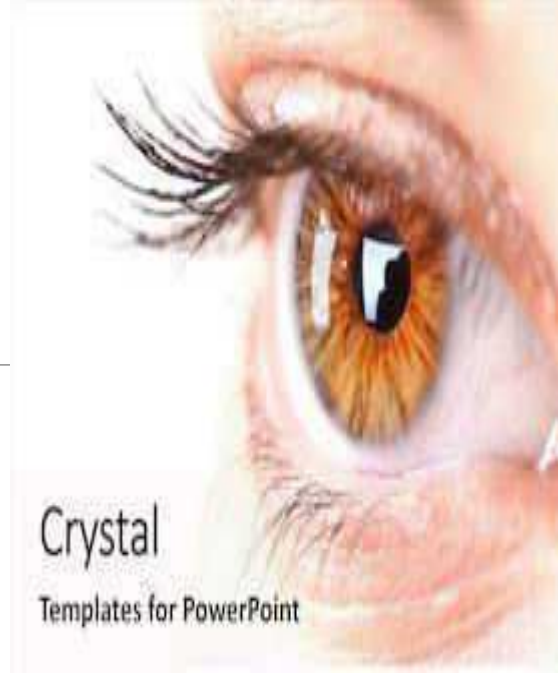
ARGYLL ROBERTSON PUPIL

Argyll Robertson pupils

- caused by neurosyphilis,

and have been attributed to a dorsal midbrain lesion that interrupts the pupillary light reflex pathway but spares the more ventral pupillary near reflex pathway – light–near dissociation intact.

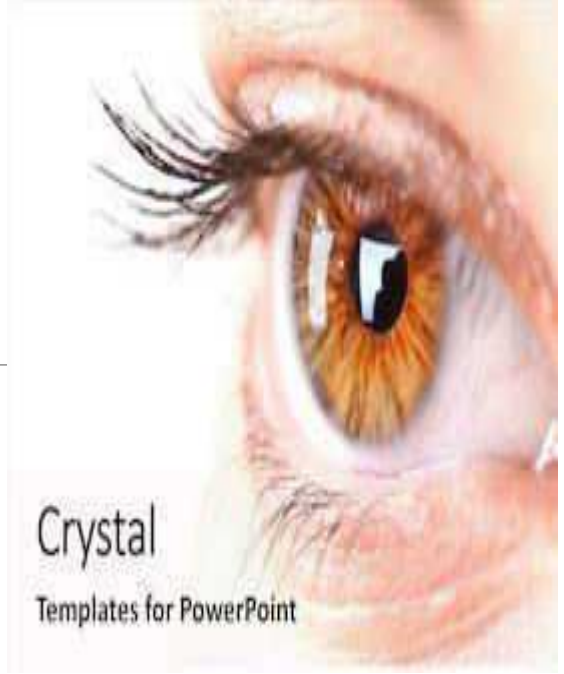




Light-near dissociation (LND) is **a pupillary sign that occurs**

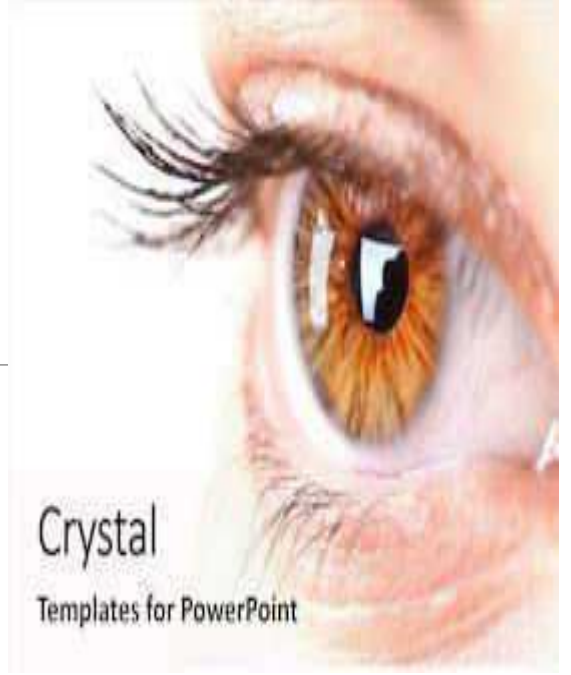
when the pupillary light reaction is impaired

while the near reaction (accommodative response) remains normal.



- **Sign ;**
- In dim light both pupils are small and may be irregular.
- In bright light neither pupil constricts,
- but on accommodation (near target) both constrict.
- The pupils do not dilate well in the dark,
- but cocaine induces mydriasis unless marked iris atrophy is present.

Causes of light–near dissociation

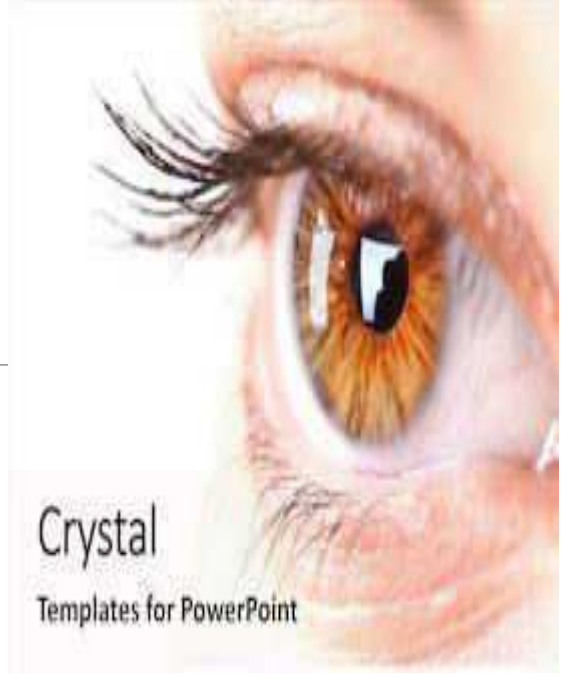


CAUSES

- Unilateral
  - • Afferent conduction defect
  - • Adie pupil
  - • Herpes zoster ophthalmicus
  - • Aberrant regeneration of the third cranial nerve

CAUSES

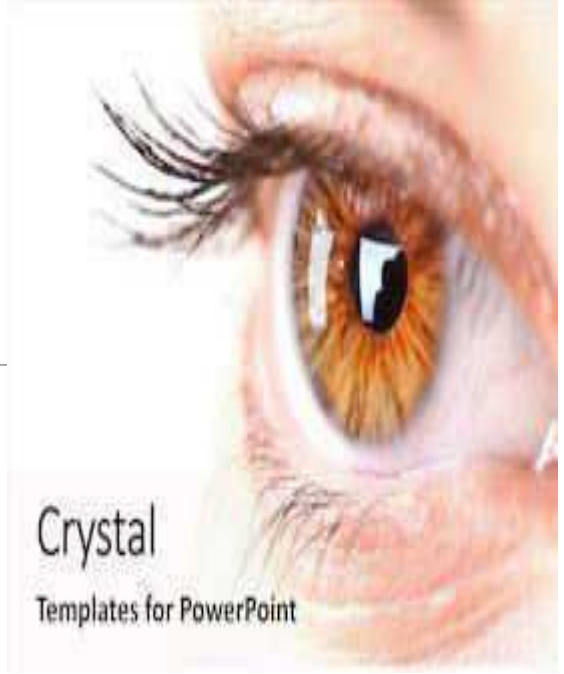
- Bilateral
  - • Neurosyphilis
  - • Type 1 diabetes mellitus
  - • Myotonic dystrophy
  - • Parinaud (dorsal midbrain) syndrome
  - • Familial amyloidosis
  - • Encephalitis
  - • Chronic alcoholis



SYMPATHETIC PARESIS

HORNER'S SYNDROME

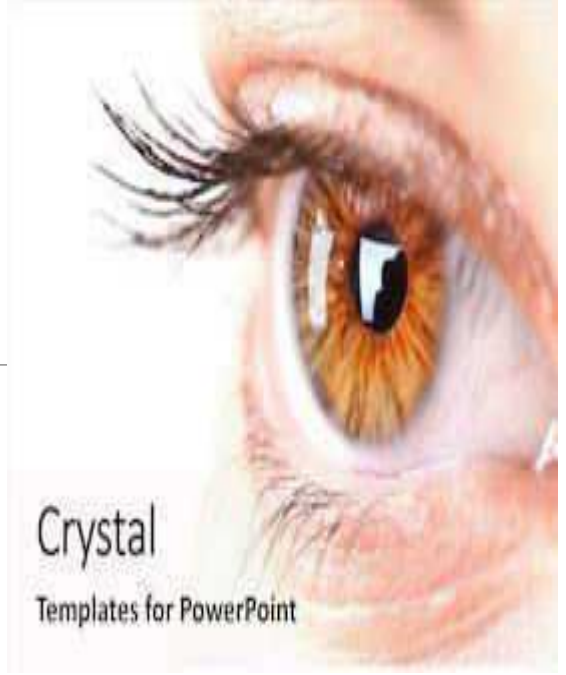
Horner syndrome (oculosympathetic palsy)



- Sympathetic supply involves three neurones

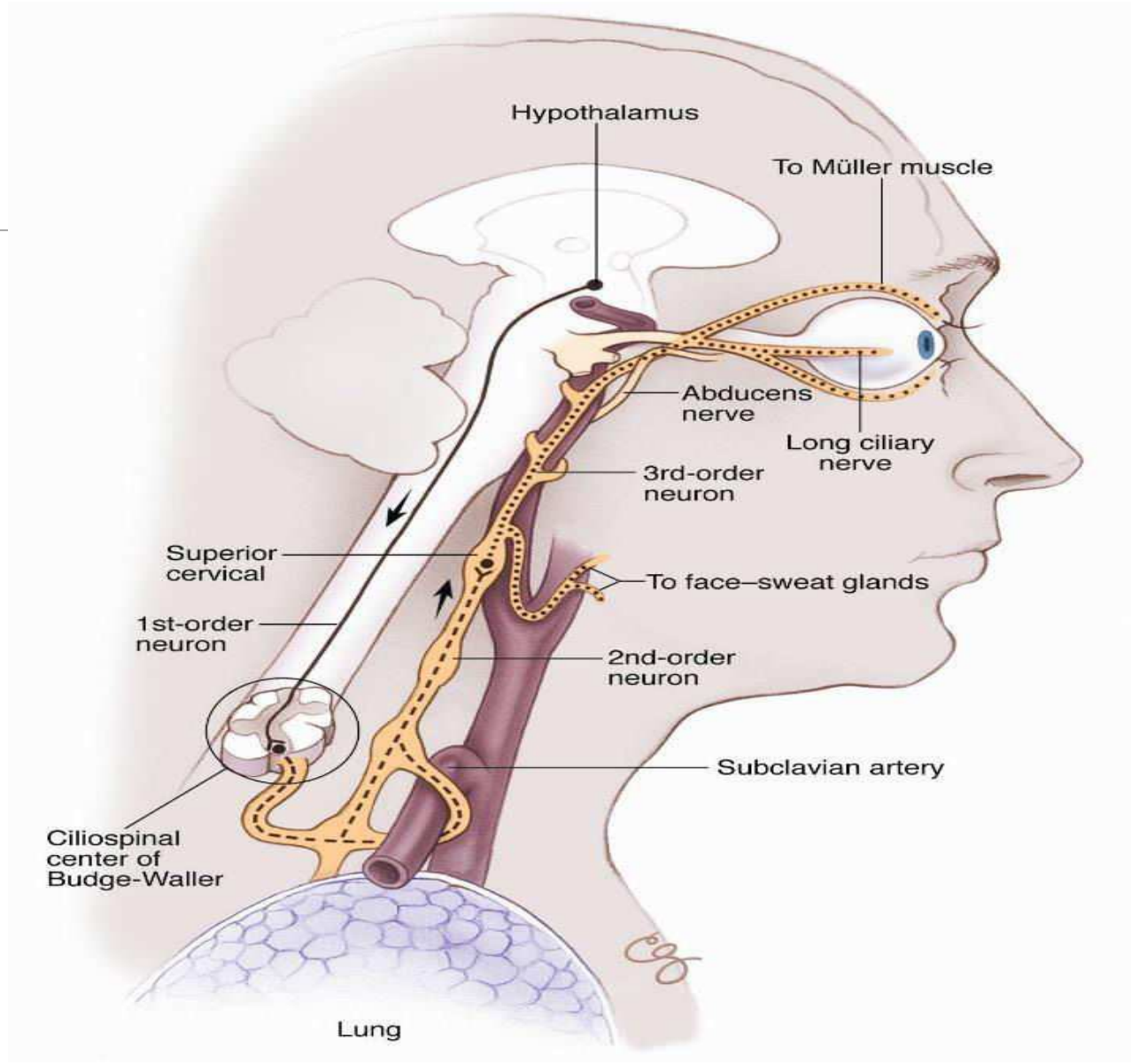
- First (central) starts
  - in the posterior hypothalamus and descends, uncrossed, down the brainstem to terminate in the ciliospinal centre of Budge, in the intermediolateral horn of the spinal cord, located between C8 and T2.

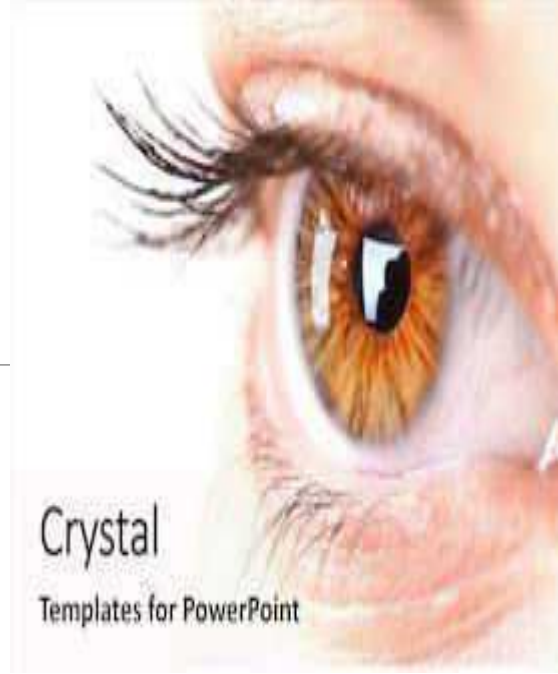
- Second (preganglionic)
  - passes from the ciliospinal centre to the superior cervical ganglion in the neck. During its long course, it is closely related to the apical pleura where it may be damaged by bronchogenic carcinoma (Pancoast tumour) or during surgery on the neck.



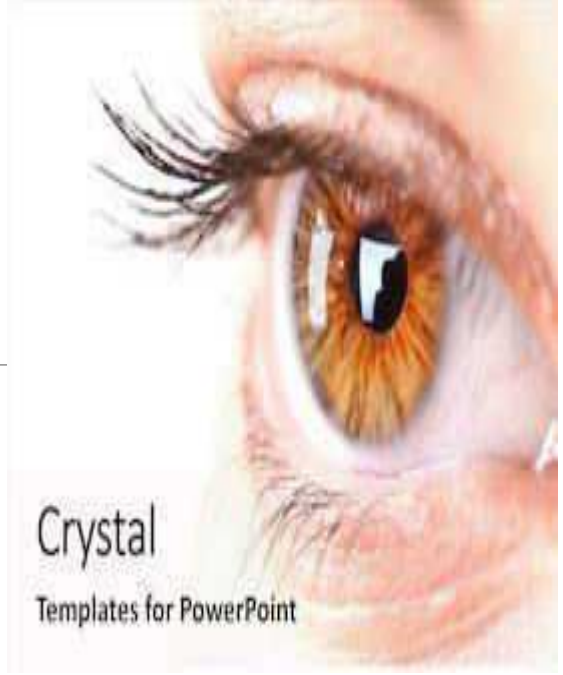
- Third (postganglionic)
  - ascends along the internal carotid artery to enter the cavernous sinus where it joins the ophthalmic division of the trigeminal nerve.
  - The sympathetic fibres reach the ciliary body and the dilator pupillae muscle via the nasociliary nerve and the long ciliary nerves.

Sympathetic supply



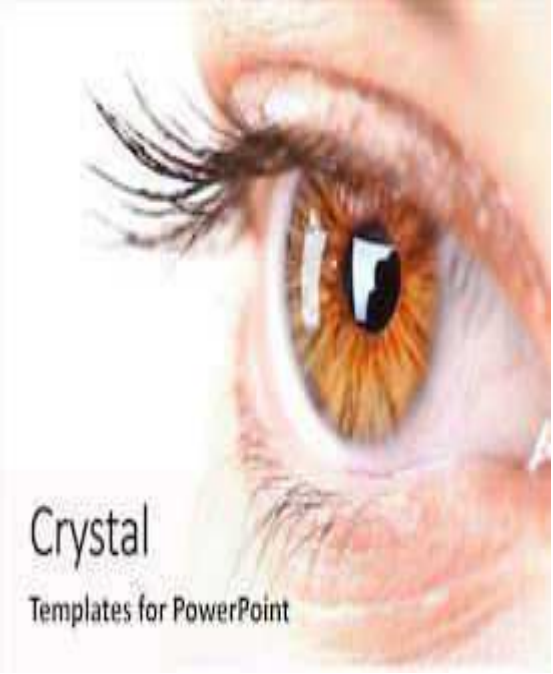


Causes of Horner syndrome

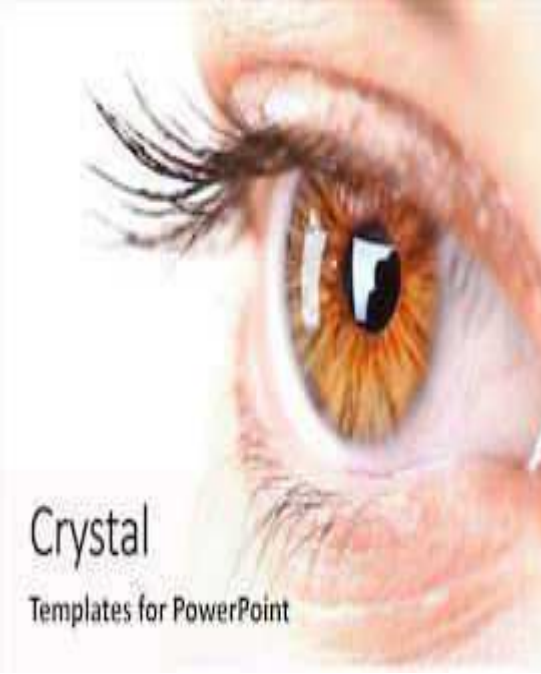


- Central (first-order neuron)
  - Brainstem disease – commonly stroke (e.g. lateral medullary infarction), but also tumor, demyelination
  - Syringomyelia
  - Lateral medullary (Wallenberg) syndrome
  - Cervical spinal cord lesion
  - Diabetic autonomic neuropathy

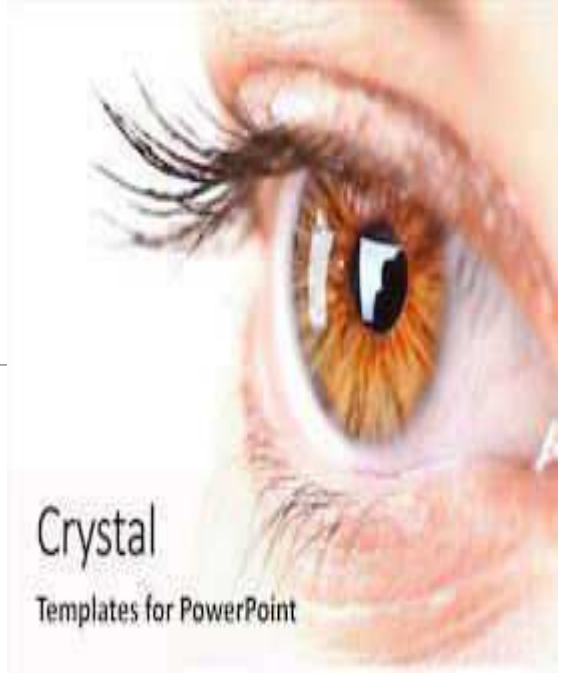
- Preganglionic (second-order neuron)
  - Pancoast tumor
  - Carotid and aortic aneurysm and dissection
  - Thoracic spinal cord lesion
  - Miscellaneous neck lesions (thyroid tumor, enlarged lymph nodes, trauma, postsurgical)



- Postganglionic (third-order neuron)
  - Internal carotid artery dissection
  - Nasopharyngeal tumor
  - Cavernous sinus mass
  - Otitis media
  - Cluster headache (migrainous neuralgia)



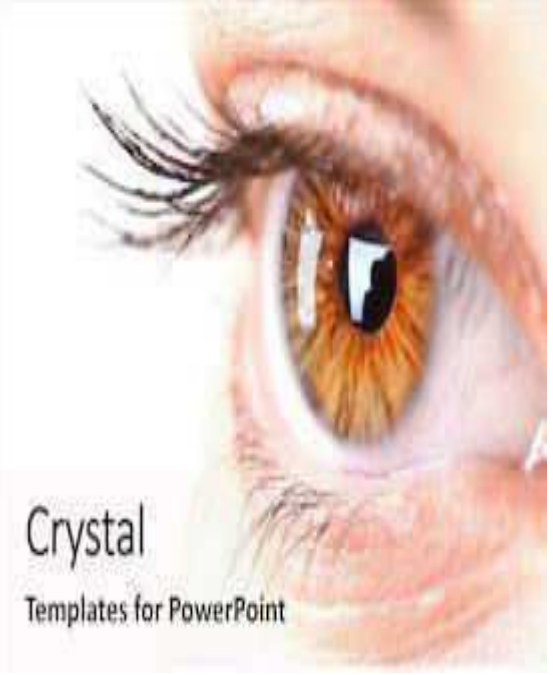
- **Presentation**
- The majority of cases are unilateral.
- Bilateral involvement occur in cervical spine injuries and autonomic diabetic neuropathy.
- Mild ptosis (usually 1–2 mm) as a result of weakness of Müller muscle,
- Miosis due to the unopposed action of the sphincter pupillae with resultant anisocoria.
- A key examination finding is that anisocoria is accentuated in dim light, since in contrast to a normal fellow pupil the Horner pupil will dilate only very slowly; the dark-induced anisocoria diminishes with time spent in the dark environment



- Pupillary constriction to light and near stimuli is normal.
- Hypochromic heterochromia (irides of different colour,

  the Horner being lighter) may be seen if congenital or longstanding .
- Slight elevation of the inferior eyelid (inferior ptosis)

  as a result of weakness of the inferior tarsal muscle.

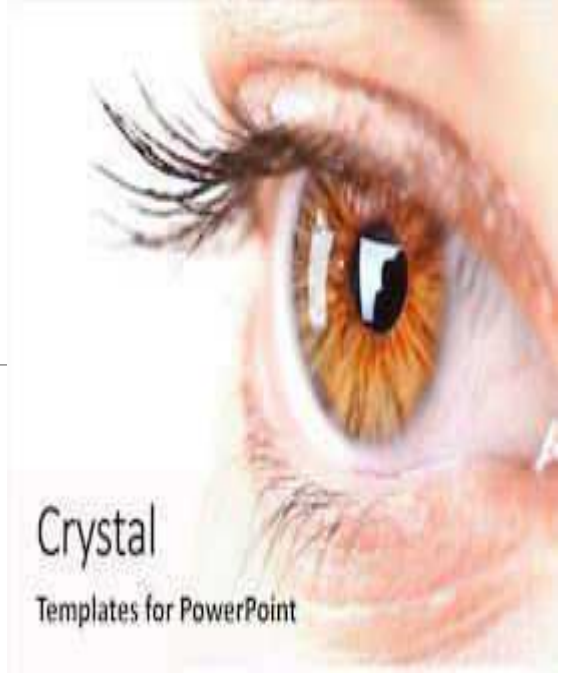


- inferior Reduced ipsilateral sweating,

- Because the psudomotor fibres supplying the skin of the face run along the external carotid artery this occurs only if the lesion is below the superior cervical ganglion;

- patients may mistakenly interpret the normal side to be sweating excessively.

Pharmacological tests



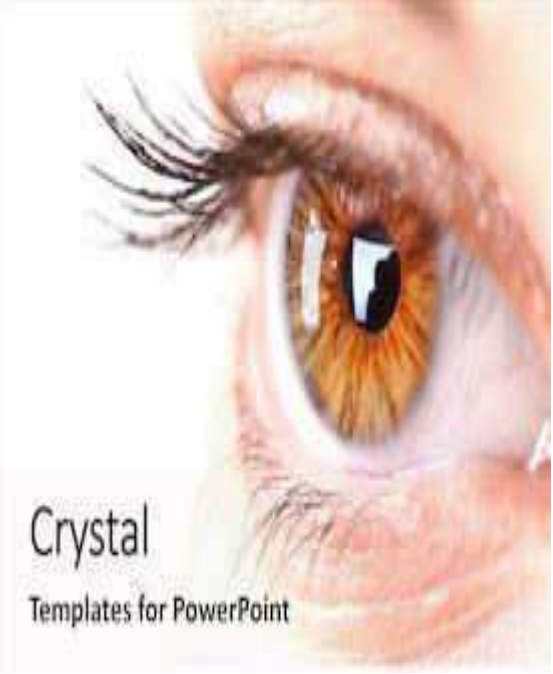
- **Apraclonidine or cocaine test**:

  used to confirm the diagnosis

- **Hydroxyamphetamine and adrenaline**

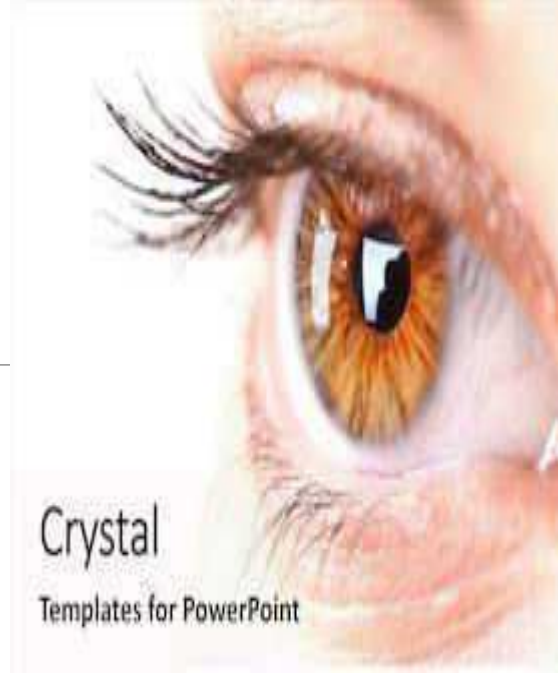
  may be used to differentiate a preganglionic (abnormal first- or second-order neurone) from a postganglionic lesion (abnormal third-order neurone)

Pharmacological tests

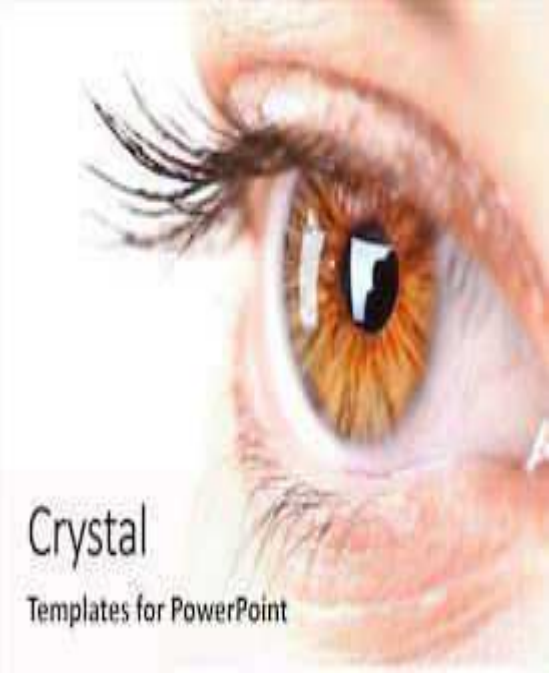


Apraclonidine or cocaine test

- Apraclonidine 0.5% or 1.0%.
- One drop is instilled into both eyes to confirm or refute the presence of Horner syndrome.
- The pupils should be checked at 30 minutes and, if negative, rechecked at 45 minutes.
- Apraclonidine penetrates the blood–brain barrier, so should be used only with great caution in infants under one year of age.



- **Result:**
- A Horner pupil will dilate but a normal pupil is essentially unaffected.
- The ptosis commonly also improves.
- Sensitivity is around 90% and
- specificity close to 100%
- **Explanation:** Alpha-1 receptors are upregulated in the denervated dilator pupillae.



➢Cocaine test

➢4% is instilled into both eyes;

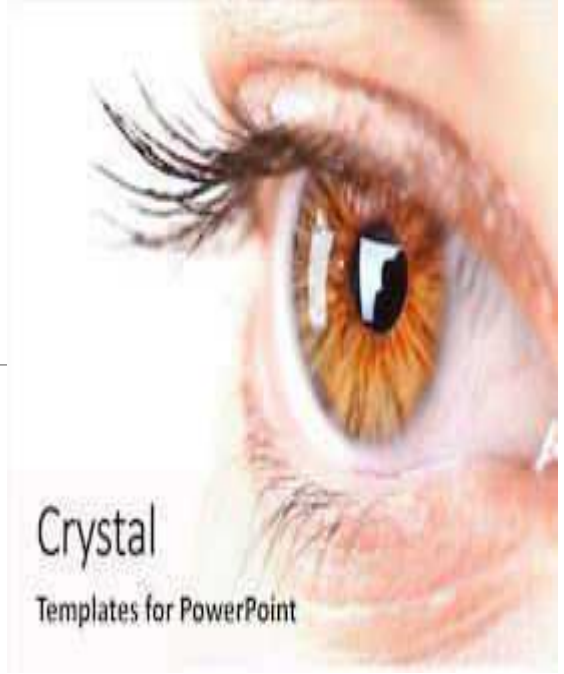
➢**Result:**

- ➢ The normal pupil will dilate but the Horner pupil will not;
- ➢Anisocoria of as little as 0.8 mm in a dimly lit room is significant.

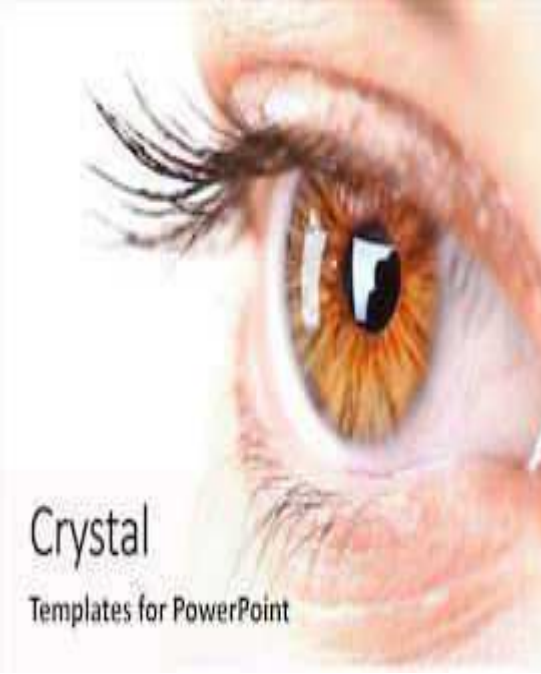
➢**Explanation:**

- ➢ Cocaine blocks the re-uptake of noradrenaline secreted at the postganglionic nerve ending, which accumulates and causes dilatation of a normal pupil.
- ➢In Horner syndrome, there is no noradrenaline being secreted, so cocaine has no effect.

Phenylephrine 1%

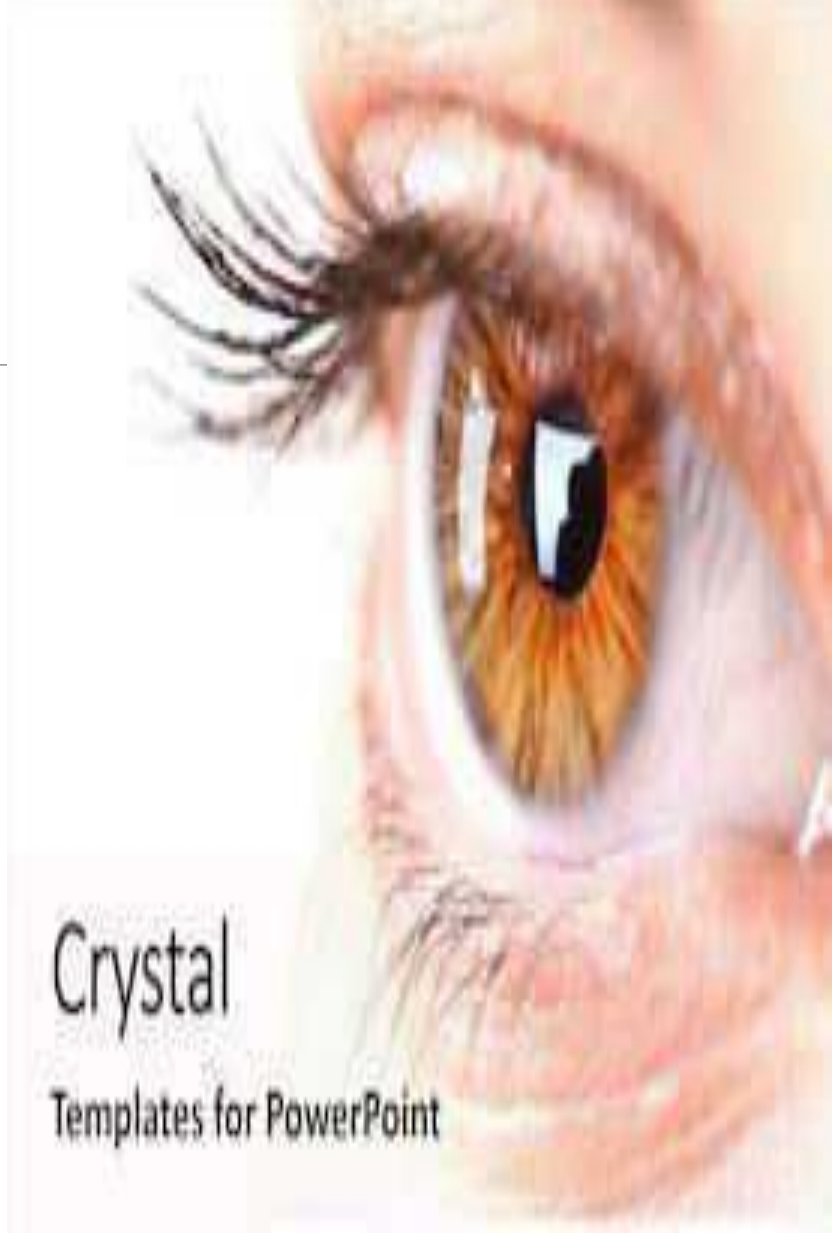


- More readily available than hydroxyamphetamine and adrenaline
- It distinguish pre- and postganglionic lesions.
- Prepared by dilution of commonly available 2.5% or 10% solution.
- Result:
  - In an established (10 days) postganglionic lesion, the **Horner pupil will dilate** and ptosis may be temporarily relieved.
  - A central or preganglionic Horner pupil and a normal pupil will not dilate or will dilate minimally.
- Explanation:
  - In postganglionic Horner syndrome the dilator pupillae muscle develops denervation hypersensitivity to adrenergic neurotransmitters due to its dysfunctional local motor nerve.



- **Hydroxyamphetamine 1%.**
- Two drops are instilled into each eye.
- It may be slightly more sensitive than phenylephrine testing.
- **Result:**
  - A normal or preganglionic Horner pupil will dilate but a postganglionic Horner will not.
- **Explanation:**
  - Hydroxyamphetamine potentiates the release of noradrenaline from functioning postganglionic nerve endings.
  - In a lesion of the third-order neurone (postganglionic) there is no release of noradrenaline from the dysfunctional nerve
- Adrenaline 0.1% has an action similar to that of phenylephrine

Thanks



Visual pathway and visual field defect

Dr samina

AP Ophthalmology

Visual pathway

- **objectives**
- Describe the different components of visual pathway
- Lesions/ defects of visual pathway

Anatomy of different components of visual pathway

OPTIC NERVE

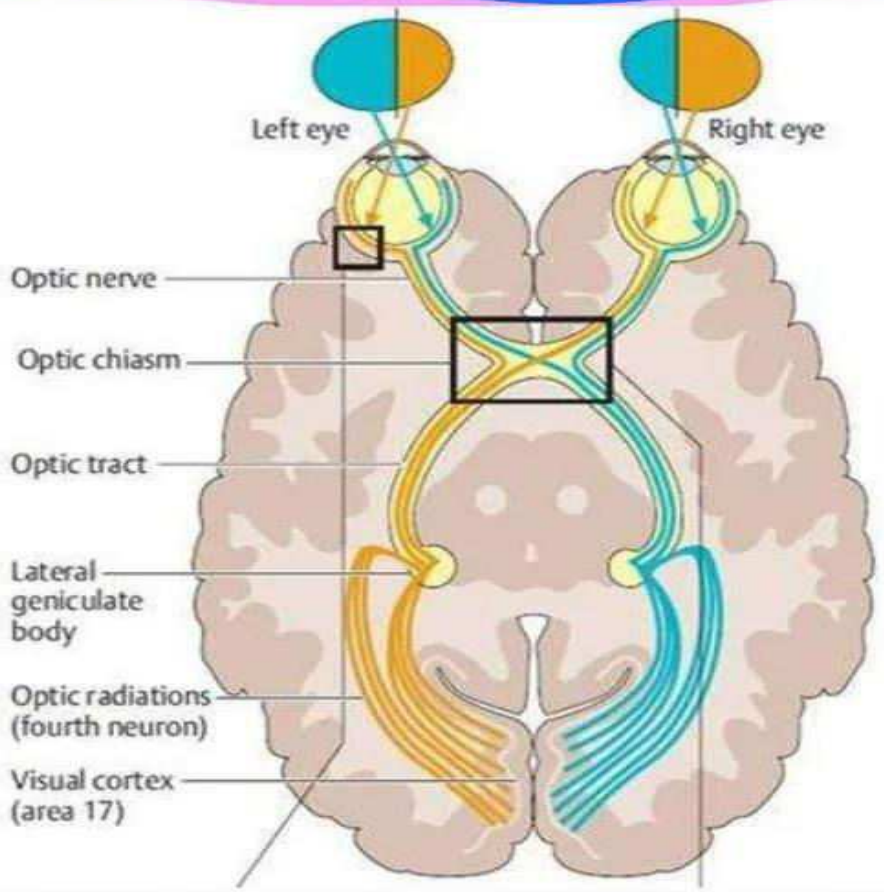
OPTIC CHIASMA

OPTIC TRACT

LATERAL GENICULATE BODY

OPTIC RADIATIONS

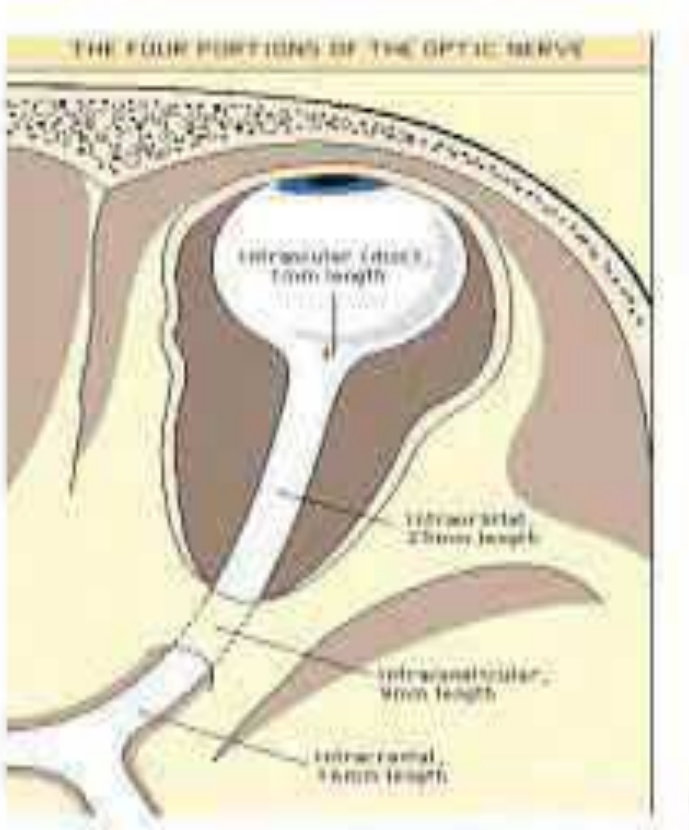
VISUAL CORTEX



Laxmi eye institute

Optic Nerve

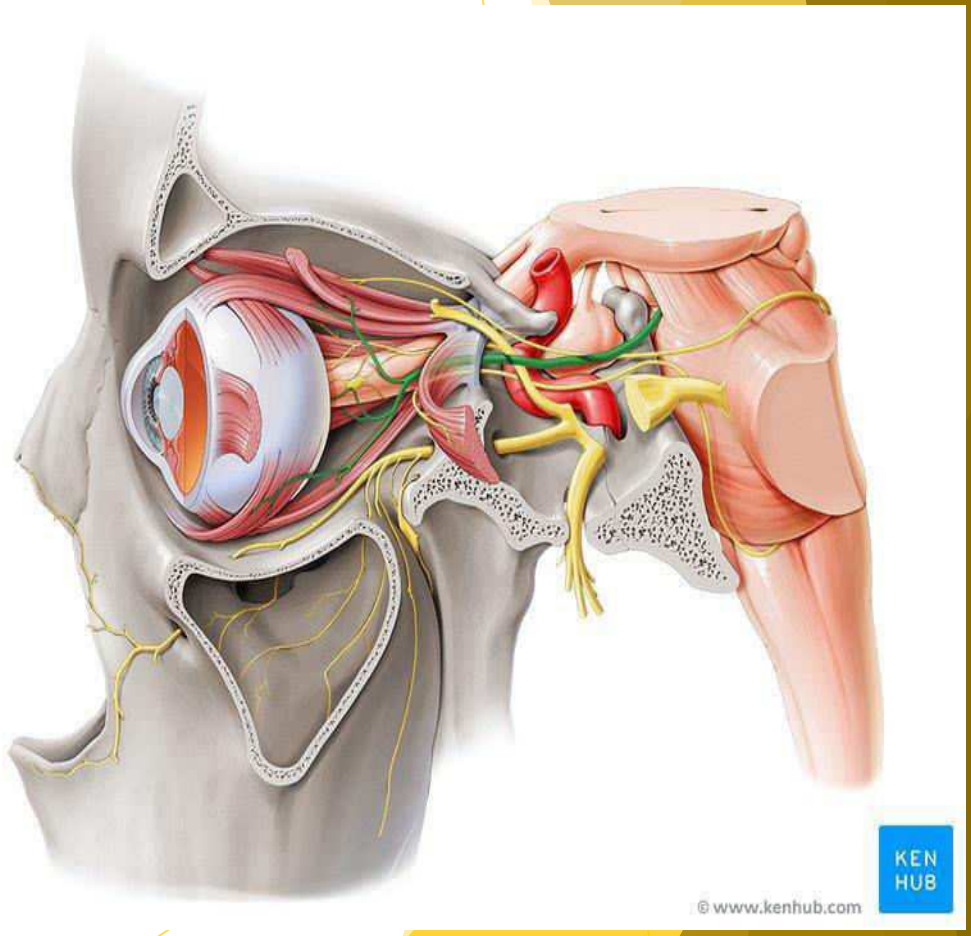
- **Parts of optic nerve**
  - Intraocular (1 mm)
  - Intraorbital (30 mm)
  - Intracanalicular (6- 9mm)
  - Intracranial (10 mm)



- Intraocular part
  - Passes through sclera , choroid and appears in eye as optic disc
  - Intraocular portion is of 1.5mm which expands to 3mm just behind eye because of myelin sheath
  - ONH has 4 portions

- Intraorbital part

  - Extends back from eyeball to optic foramina
  - Covered by dura, arachnoid and pia.
  - Central retinal artery enters nerve on its inferomedial aspect about 10mm behind eyeball
  - Some fibers of superior rectus and medial rectus are adherant to its sheath so painful ocular movements in retrobulbar neuritis.



- Intracanalicular part
  - 6-9mm length
  - closely related to ophthalmic artery
- Intracranial part
  - 10mm length
  - above cavernous sinus and converges with its fellow to form chiasma
  - covered by pia matter only

Optic Chiasma

- Flattened structure measuring 12mm Horizontally and 8mm Anterio-posteriorly
- It lies over diaphragma sella
- Continues posteriorly as optic tracts and forms anterior wall of third ventricle
- Nerve fibers arising from nasal halves of the two retinae decussate at the chiasma

Optic Tracts

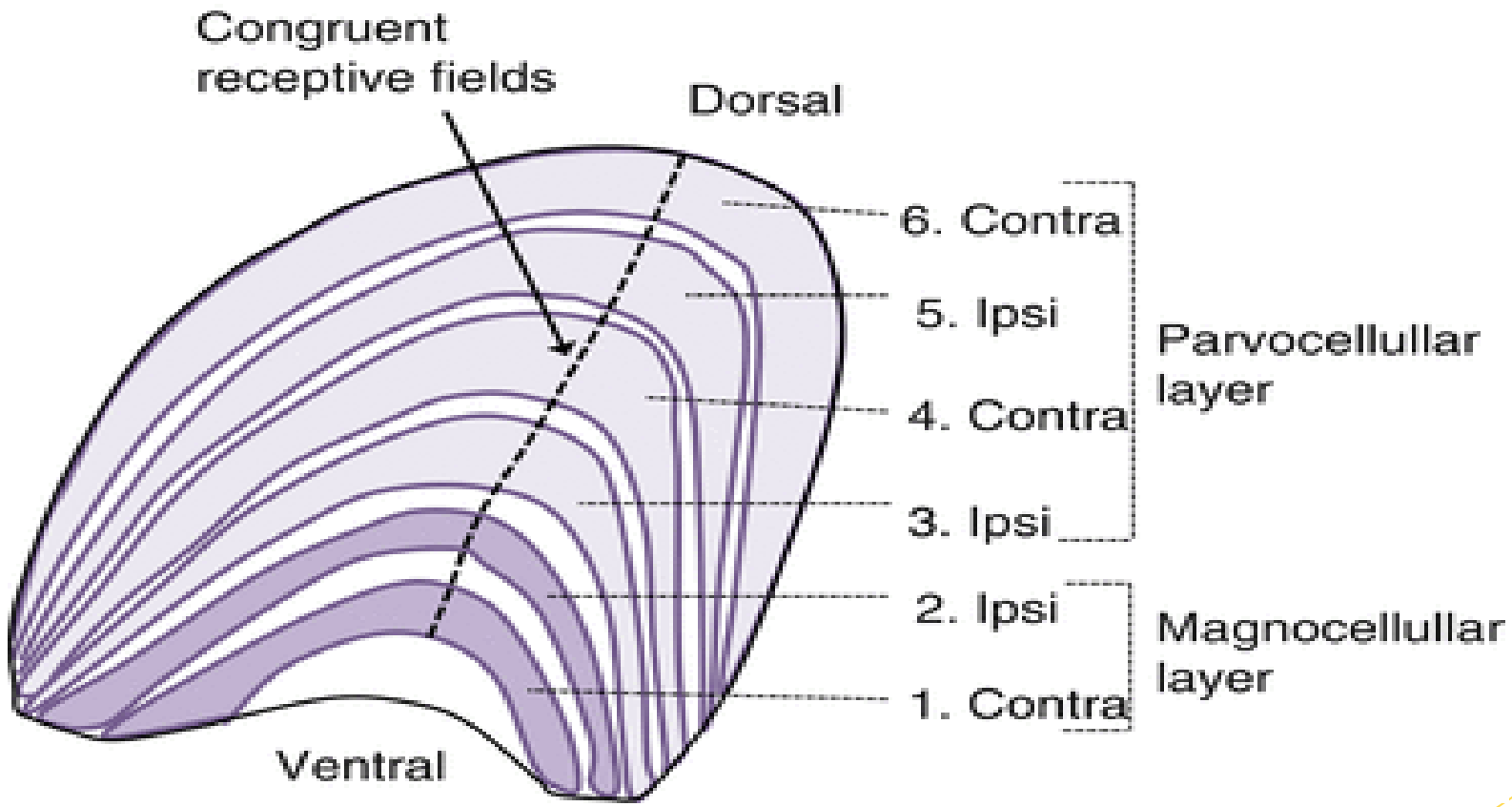
- Bundles of nerve fibers running outwards and backwards from postero-lateral aspect of optic chiasma
- Consist of temporal fibers of retina of same eye and nasal half of opposite eye
- Each optic tract end in LGB

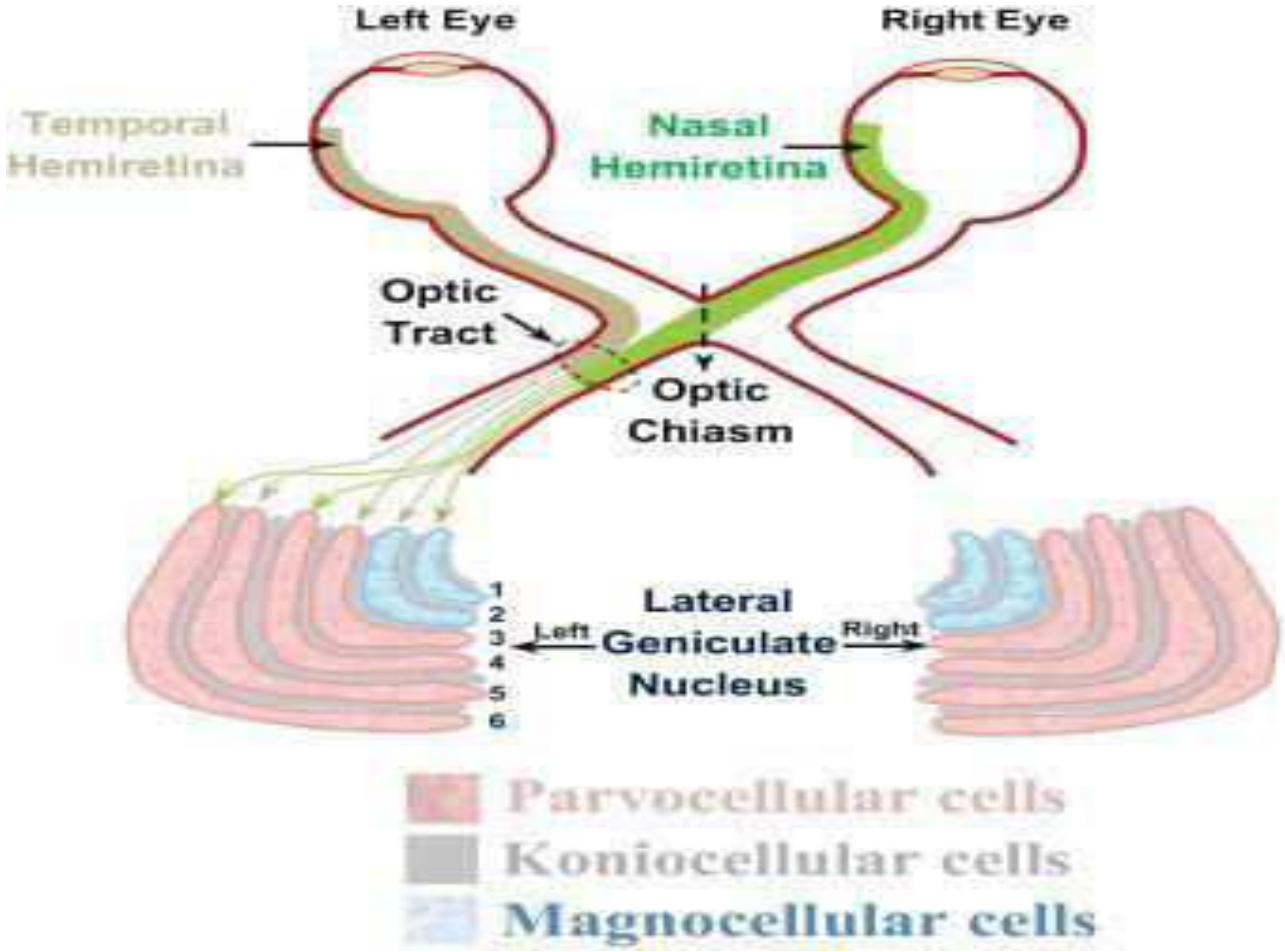
Lateral Geniculate Body

- Oval structures situated at termination of optic tract
- Each LGB consist six layers of neurons alternating with white matter
- Each body is split into 6 laminae
- Fibers from Ipsilateral Temporal Retina End in Lamina 2,3,5 and contralateral nasal retina end in Lamina 1,4,6.

  This 6 laminae divide LGB into 2 portions

Lateral Geniculate Body




Left Eye
Right Eye
Temporal Hemiretina
Nasal Hemiretina
Optic Tract
Optic Chiasm
1
2
3
4
5
6
Left
Lateral Geniculate Nucleus
Right
Parvocellular cells
Koniocellular cells
Magnocellular cells

Functions Lateral geniculate body

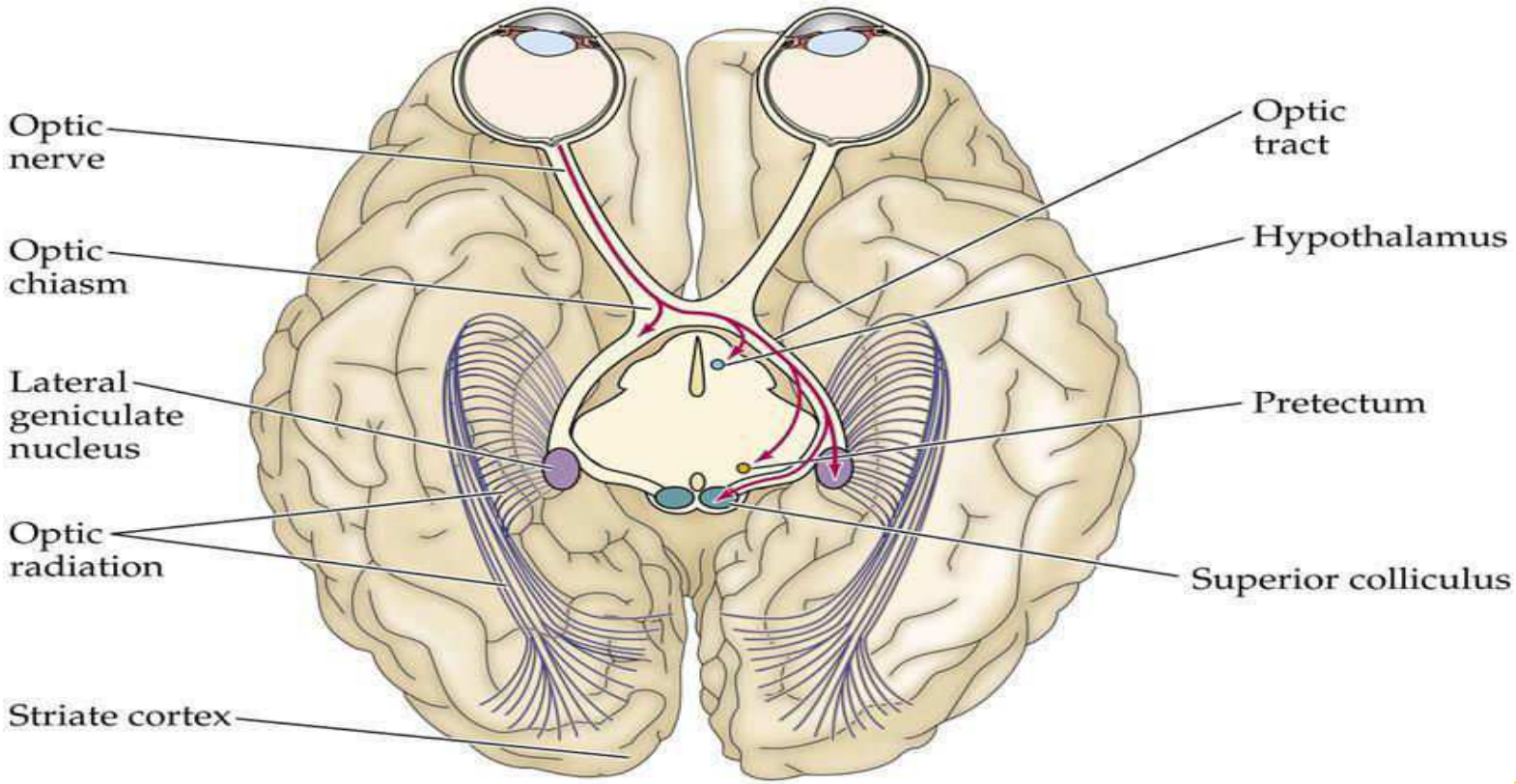
- Relay station to relay visual information from optic tract to visual cortex

- To gate transmission of signals to visual cortex

Optic Radiations

- Extend from LGB to visual cortex
- Fibers of optic radiation spreads out fanwise to form medullary optic lamina
- Superior fibers of radiation (which subserve inferior field) proceed directly posteriorly through parietal lobe
- Inferior fibers of radiation (which subserve Superior field) first sweep anteriorly in meyers loop around anterior tip of temporal horn of lateral ventricle and then into temporal lobe

Optic Radiations



Visual cortex

- Located on medial aspect of occipital lobe
- Subdivided into
  - Visuosensory area (striate area 17)
  - Visuopsychic area (striate area 18 and 19)

Arrangement of nerve fibers

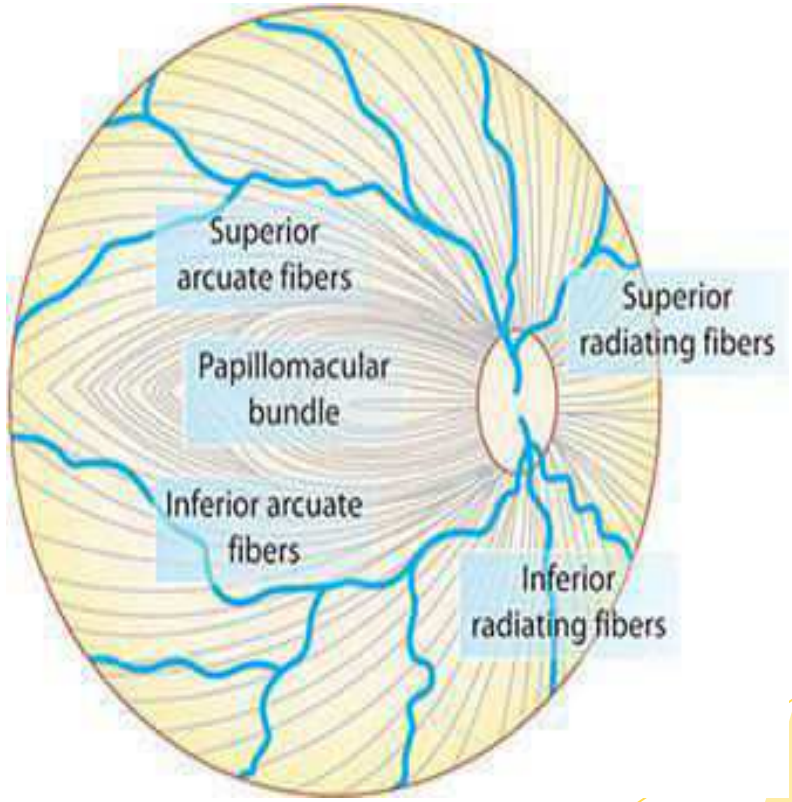
Arrangement of nerve fibers

- **Retina**

- Fibers from nasal half come directly to optic disc as superior radiating fibre and Inferior radiating fibre.

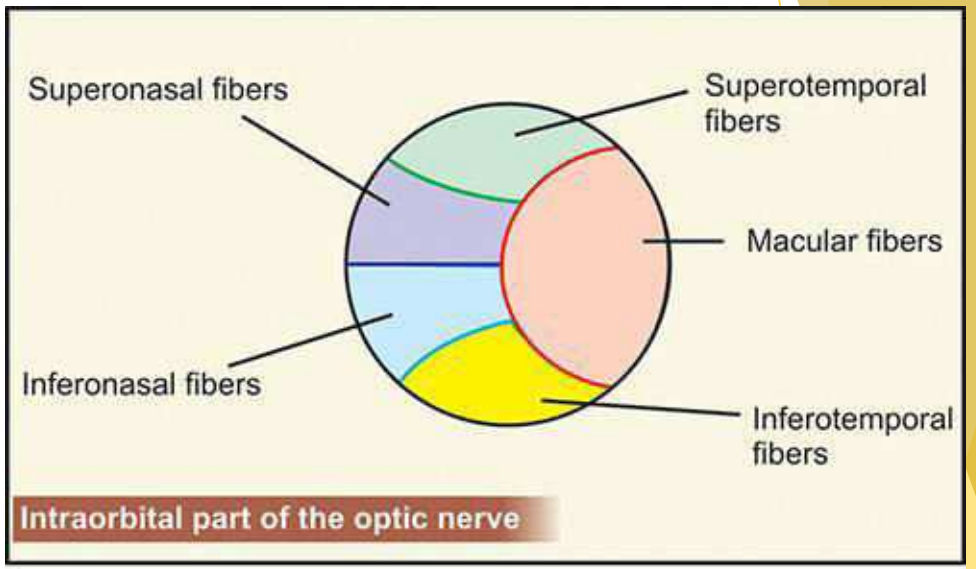
- Fibers from temporal half as superior Arcuate fibre and inferior arcuate fibre.

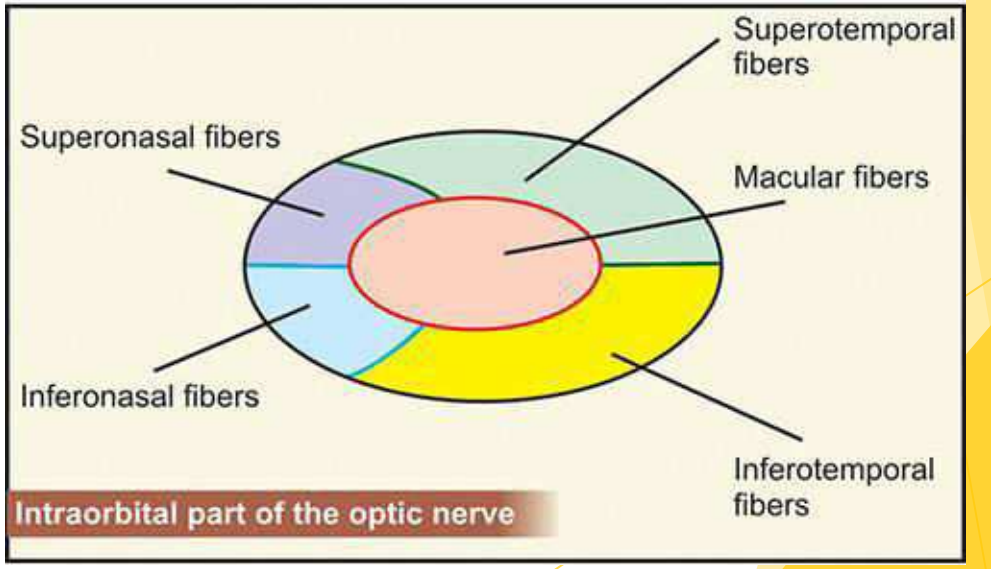
- Fibers from macular region pass straight in temporal part of optic disc as pmb



Arrangement of nerve fibers

- **Optic nerve**
- 1. **Optic nerve head** arrangement of fiber exactly same as retina
- **2. Proximal region of optic nerve**
- -macular fibers present centraly
- -Temporal fibers present temporaly and
- nasal fibers present nasally

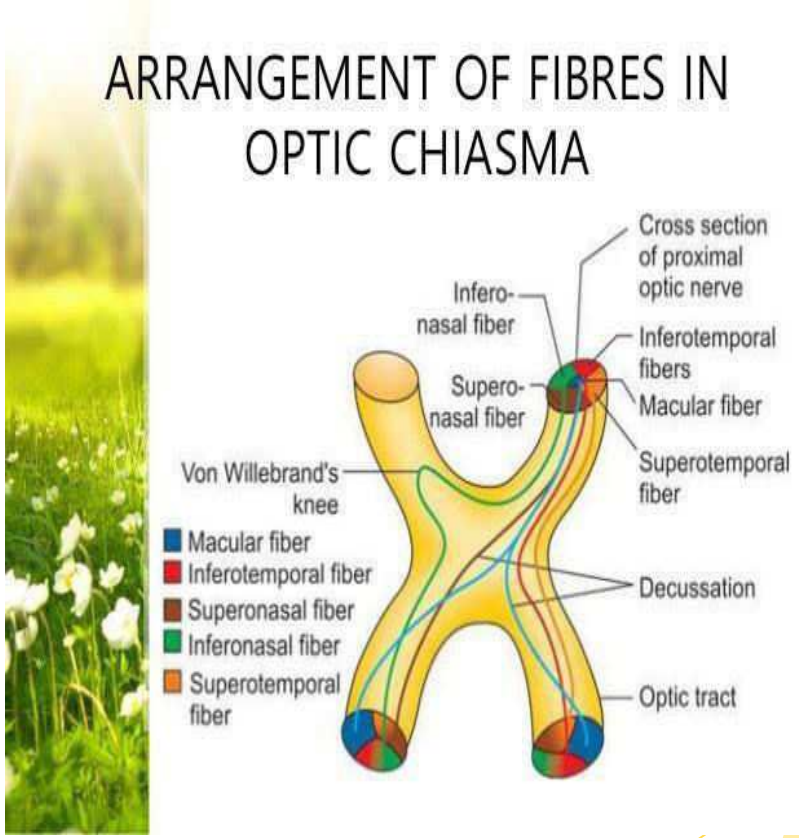




Arrangement of nerve fibers

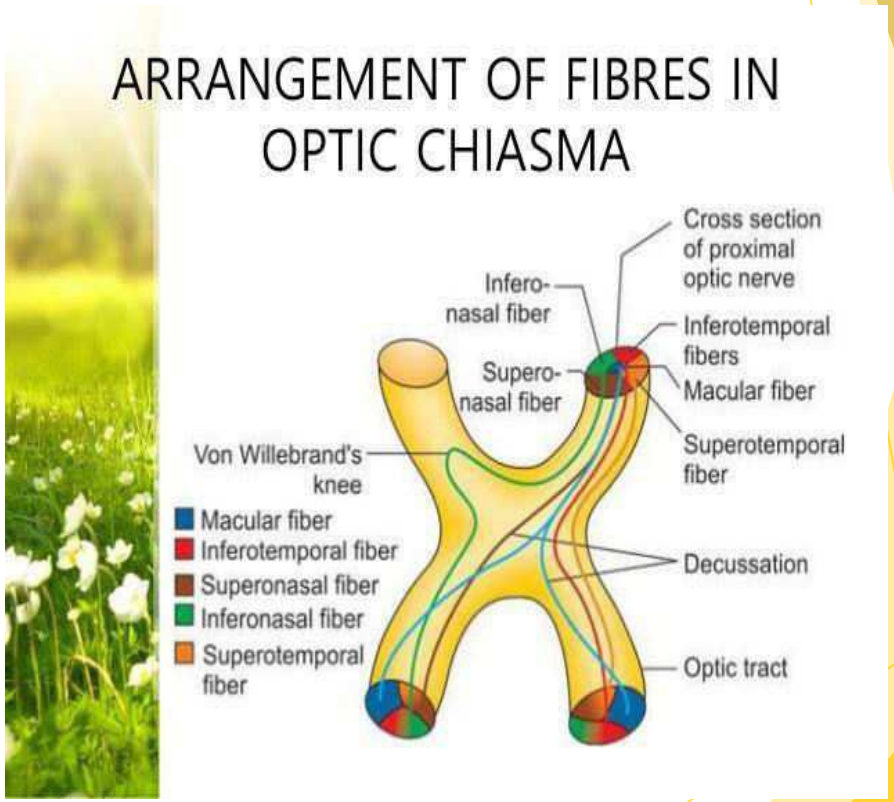
- **Optic Chiasma**

- **Temporal fibers** from retina remains uncrossed and runs backward in lateral part of optic chaisma



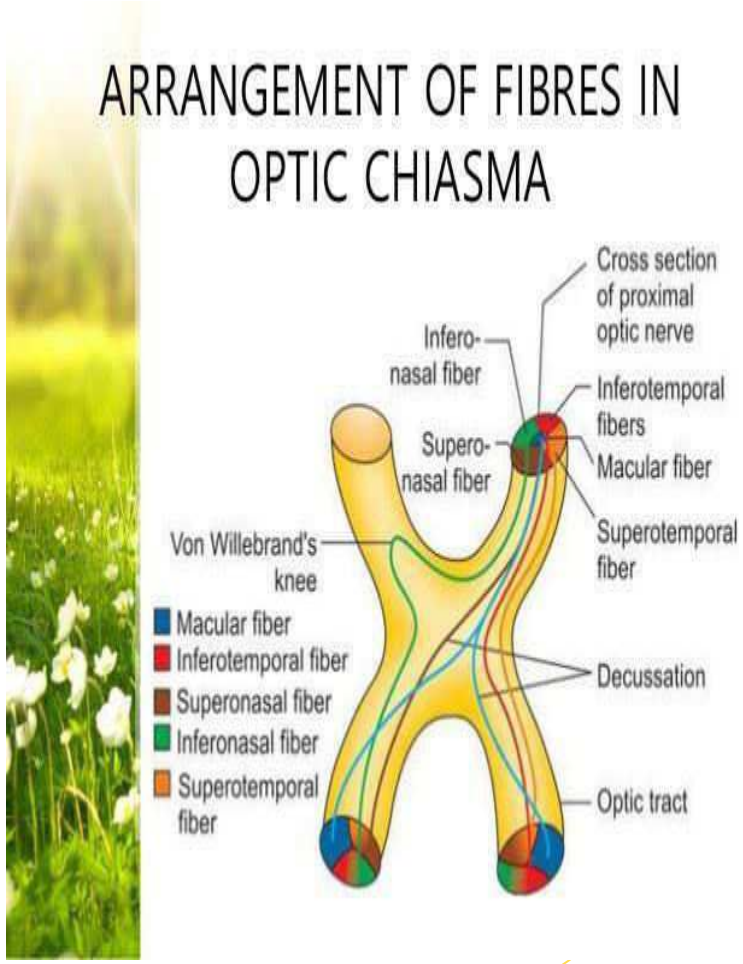
Arrangement of nerve fibers

- **Optic Chiasma Nasal peripheral fibers-**
- ¾ of fibers
- Cross over to enter medial part of opposite optic tract in following manner
- lower nasal fibers in optic tract traverse chiasma low and anteriorly
- Upper nasal fibers in optic tract trasverse chiasma high and posteriorly



Arrangement of nerve fibers

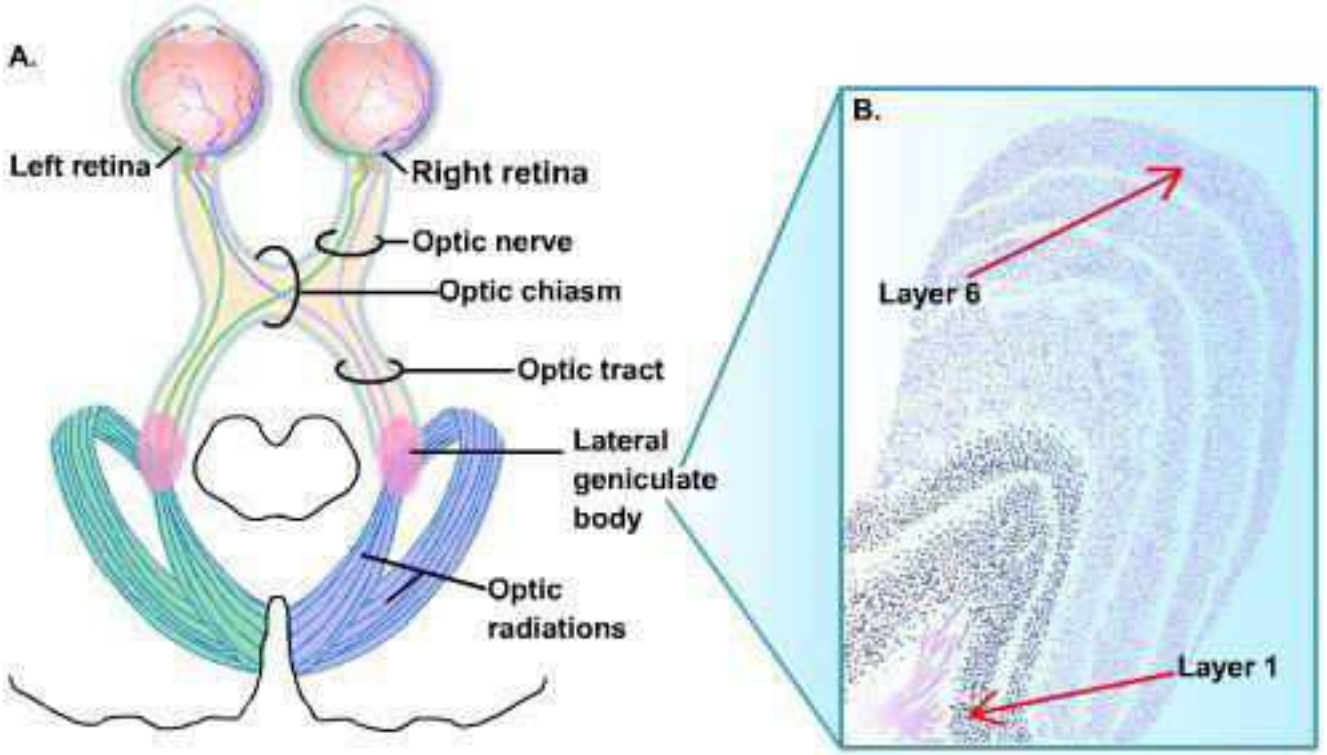
- **Optic Chiasma Macular fibers-**
- Some fibres crossed and runs backward in opposite optic tract
- Some fibers uncrossed and runs on same side in optic tract



Arrangement of nerve fibers

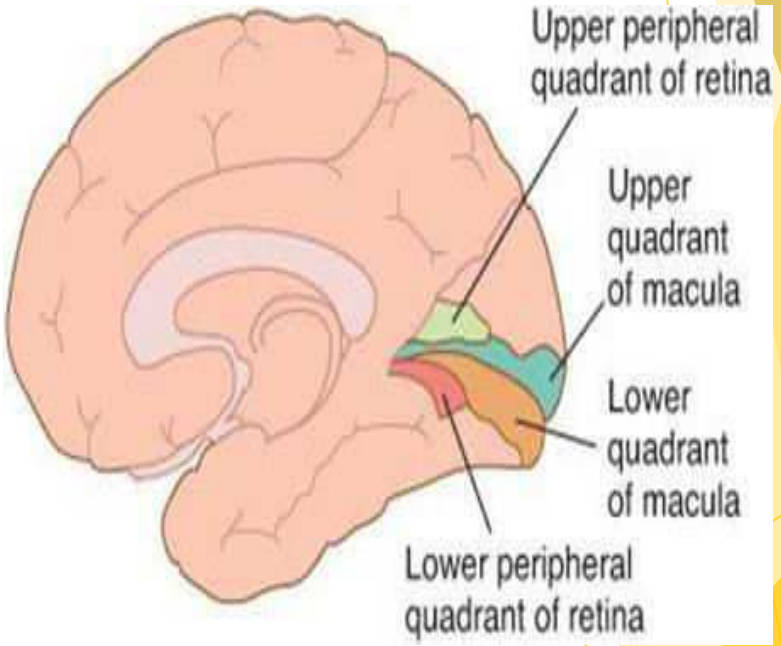
- **Optic Tract**
- Macular fibers – occupy dorso-lateral aspect of the optic tract
- Upper peripheral fibers – situated medially in the optic tract

- **LGB**
- The macula fibres coming in the optic tract occupy the posterior 2/3 of the LGB

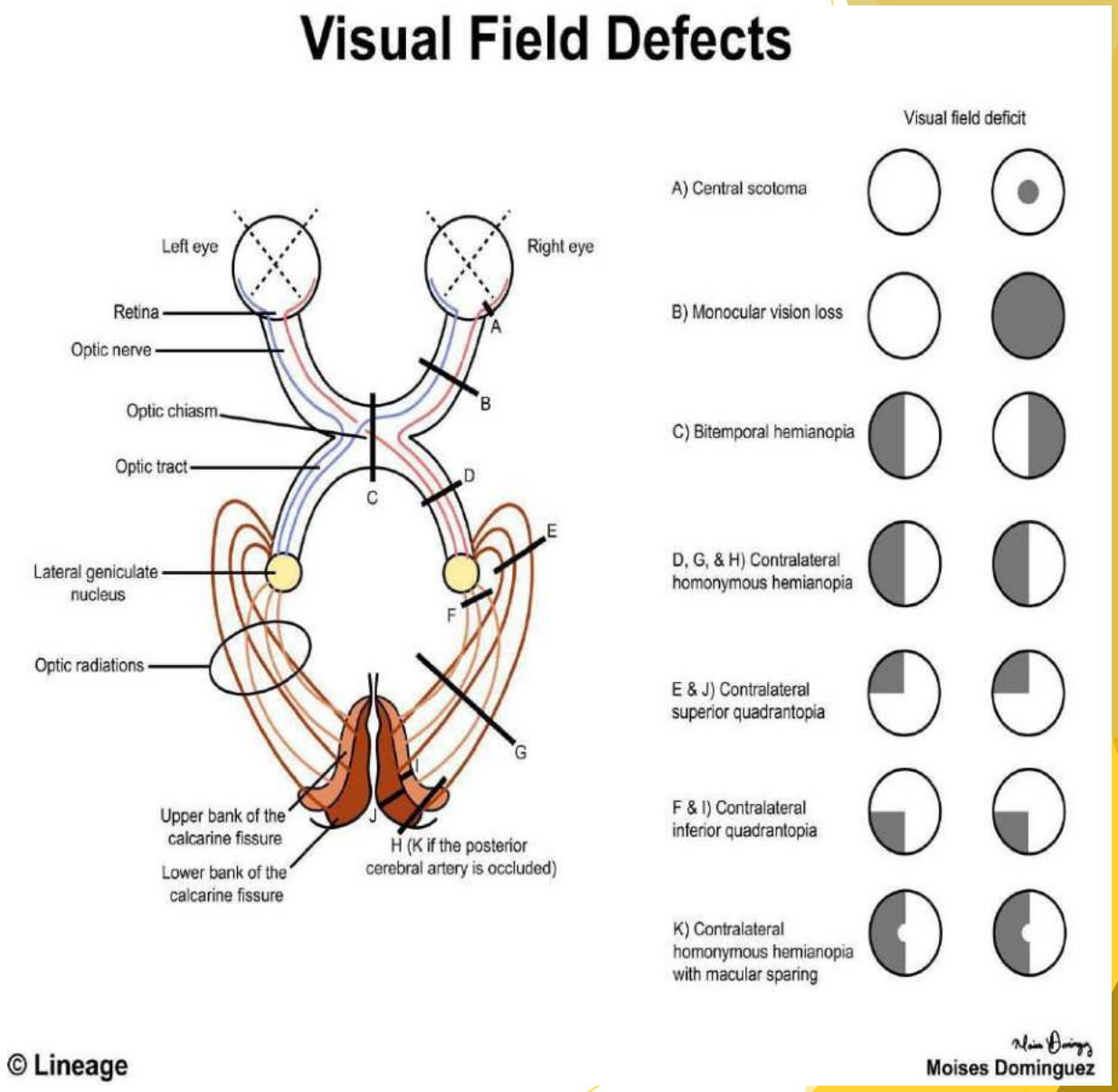

A.
Left retina
Right retina
Optic nerve
Optic chiasm
Optic tract
Lateral geniculate body
Optic radiations
B.
Layer 6
Layer 1

Arrangement of nerve fibers

- **Optic Radiations**
- Upper retinal fibers – upper part of optic radiations
- Lower retinal fibers – lower part of optic radiations
- Macular fibers – central part of optic radiations



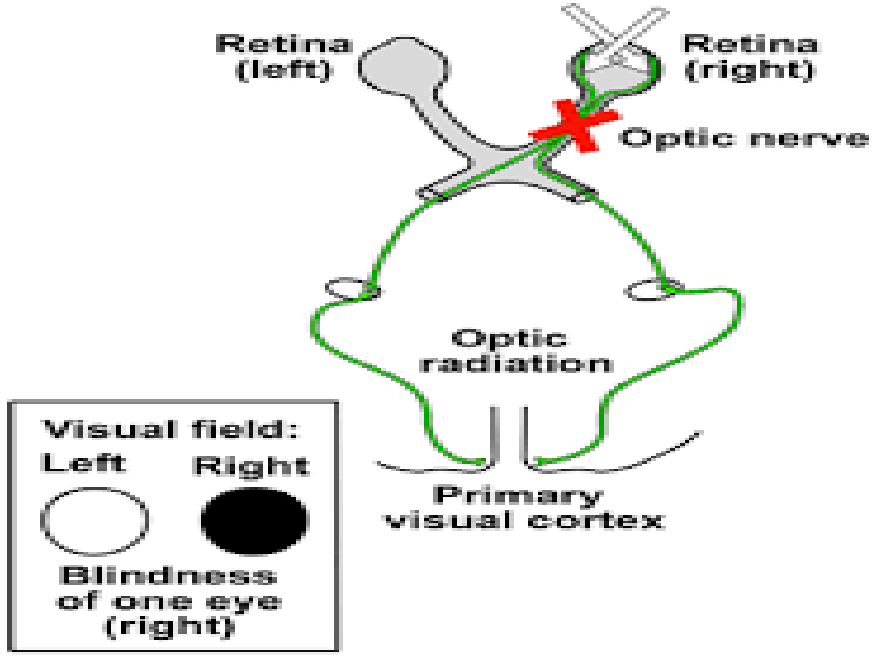
Visual field defect



OPTIC NERVE

- **Lesion of optic nerve-**
- characterised by complete blindness on affected side
- Absence of light reflex on ipsilateral side and consensual on contralateral side
- Near reflex present

- **Cause:**
  - Traumatic optic avulsion,
  - Acute optic neuritis,
  - Optic atrophy.

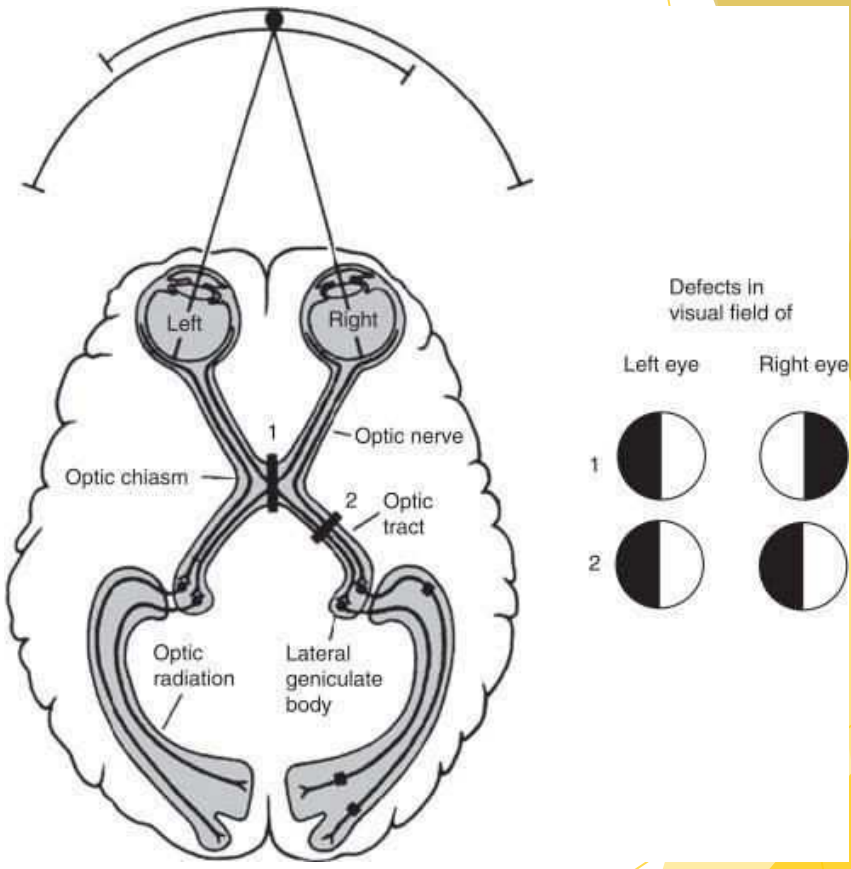


Lesion of optic chaisma-

- **1.Central chiasmal lesion-**

- Bitemporal hemianopia
- Bitemporal hemianopic paralysis of pupillary reflex
- Also leads to partial descending optic atrophy

- **Cause:**
  - Pituitary tumors Craniopharyngioma,
  - Suprasellar aneurysm.



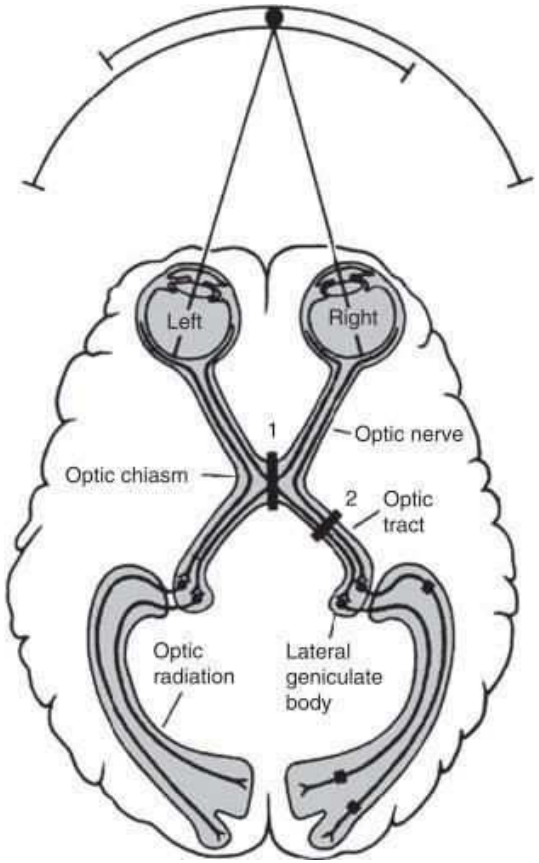
OPTIC CHIASMA

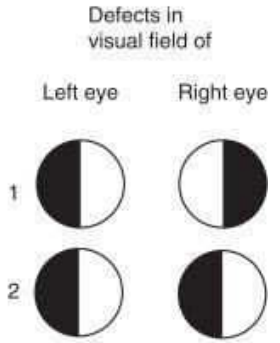
- **2.Lateral chiasma lesion-**
- Binasal hemianopia
- Binasal hemianopic paralysis of pupillary reflex
- Also leads to partial descending optic atrophy
- **Cause:**
  - Internal carotid aneurysm,
  - Lesions causing distension of third ventricle

Lesions of optic tract-

- Incongruous homonymous hemianopia
- contralateral hemianopia pupillary reaction(wernicke's reaction)
- partial descending optic atrophy

- **Causes-**
  - syphilitic meningitis,
  - tuberculosis, and
  - tumors of optic thalamus.





Lesions of Lateral geniculate body

- incongruous homonymous hemianopia
- sparing of light reflex or pupillary reflex
- Partial descending optic atrophy

OPTIC RADIATION

- Features varies depending on site of lesion
- **1.Involvment of total optic radiation-**
  - complete homonymous hemianopia (more congruous)

OPTIC RADIATION

- 2. **Involvement of part of optic radiation in temporal lobe**
  - Superior quandrantic hemianopia (pie in the sky)

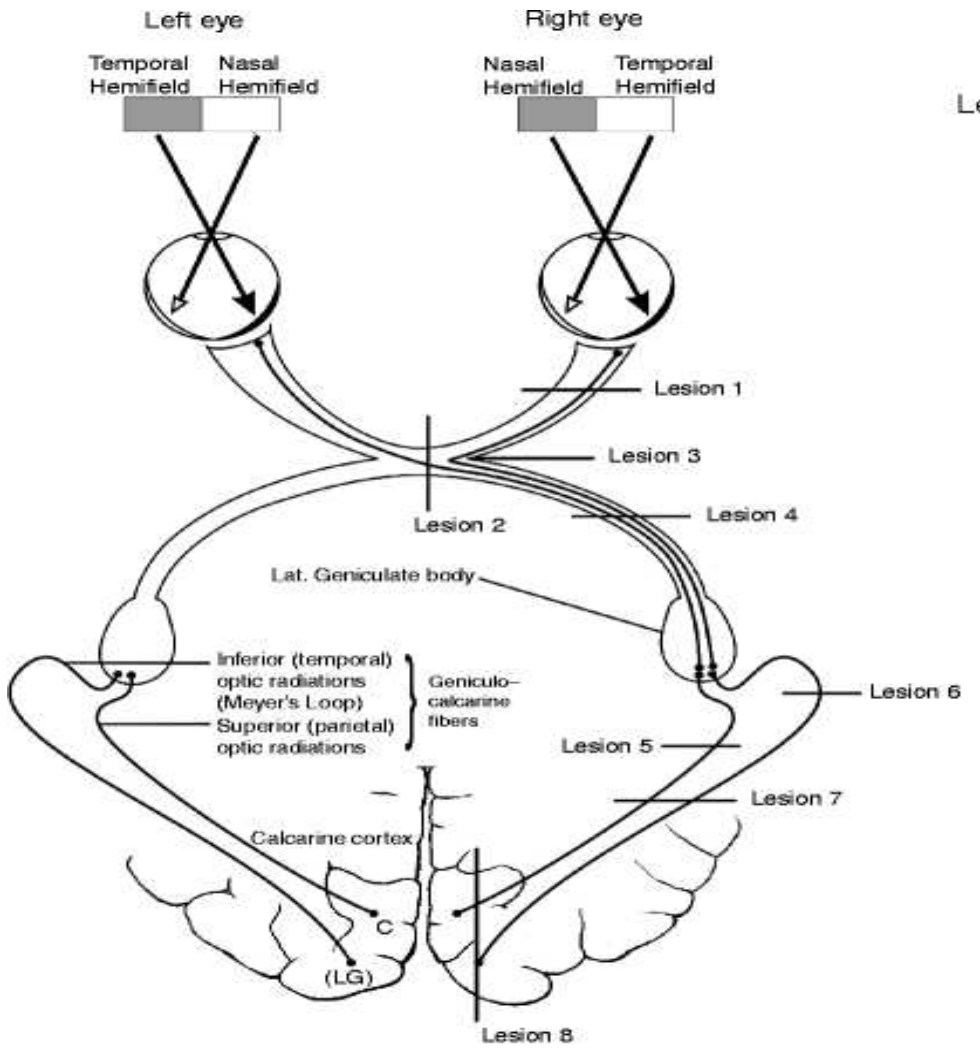
OPTIC RADIATION

- 3. Involvement of part of optic radiation in parietal lobe
  - Inferior quandrantic hemianopia (pie on the floor)

Lesions of visual cortex-

- **Anterior occipital cortex -**
- Homonymous hemianopia(sparing macula)
- **Cause -**
  - occlusion of posterior cerebral artery

- **Tip of occipital cortex**
- Homonymous hemianopia(macula defect)
- **Causes** –
  - head injury,
  - gunshot injury involving tip of cortex



Lesion	Visual fields: Lt. eye	Visual fields: Rt. eye	
1			monocular blindness
2			Bitemporal (heteronymous) heminanopia[1]
3			Ipsilateral Right nasal hemianopia
4			Left homonymous hemianopia
5			Left inferior quadrantanopia[2]
6			Left superior quadrantanopia[3]
7			Left homonymous hemianopia[4]
8			Left homonymous hemianopia with macular sparing

1 May rusult from tumor pressing on Optic Chiasm. Visual field deficit usually not this cleanly divided or symmetrical.
2 May also result from lesions restricted to Cuneus
3 May also result from lesions restricted to Lingual Gyrus.
4 Total hemianopia less likely as compared to lesions of Optic Tract.

thanks

B-SCAN ULTRASONOGRAPHY

Dr samina
AP, Ophthalmology

Learning objectives

- Define B scan?
- Which types of frequencies are used in ophthalmic ultrasound?
- What are the indications of B scan?

INTRODUCTION

- B-scan ultrasonography is an important adjuvant for the clinical assessment of various ocular and orbital diseases.

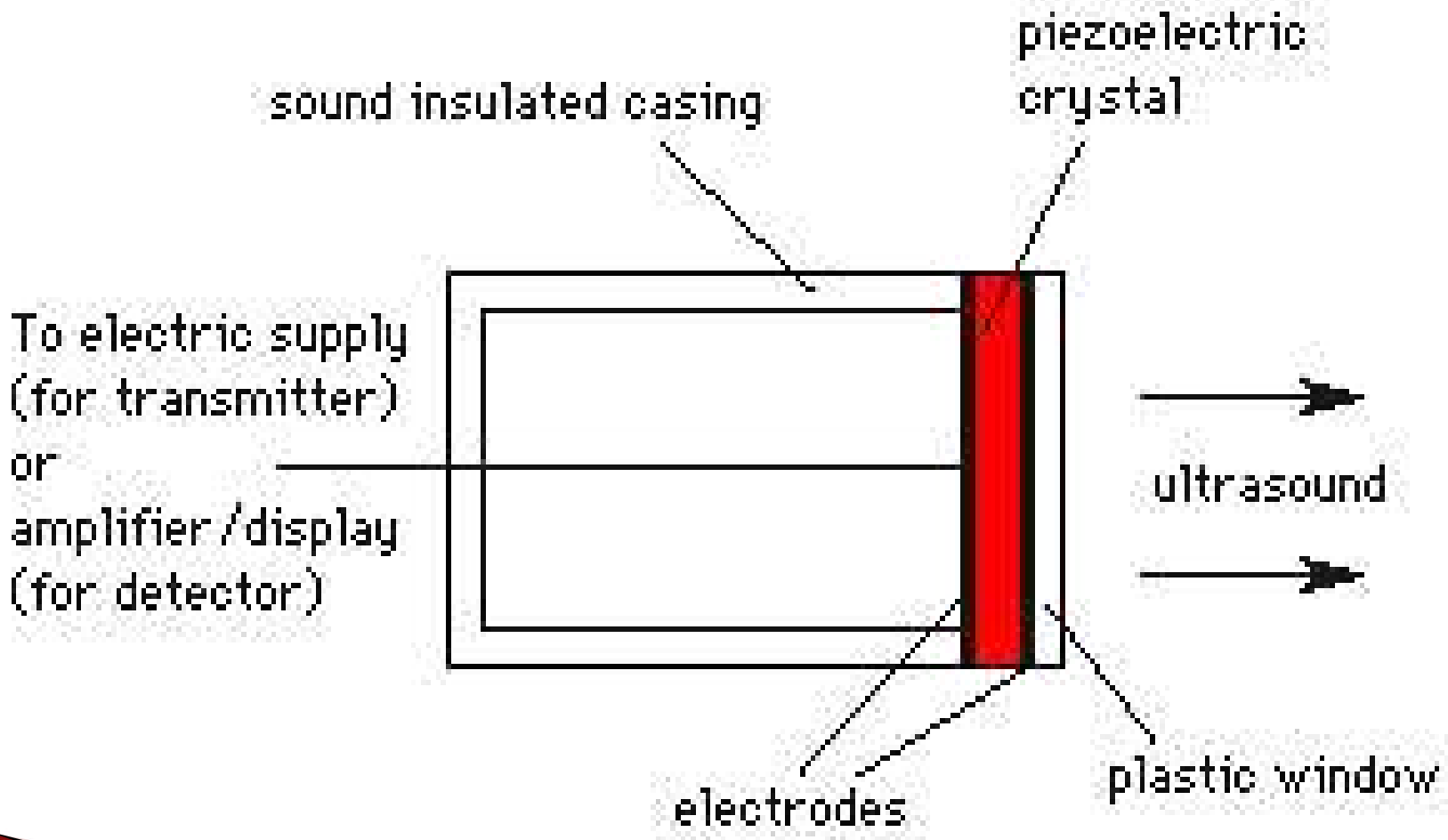
- B- SCAN is a two dimensional imaging system which uses high frequency sound waves ranging from 8-10 MHz.

Physics

- It is an acoustic wave that consists of particles within the medium
- Frequencies used in diagnostic ophthalmic ultrasound are in the range of 8-10 MHz
- These high frequencies produce shorter wave lengths which allow good resolution of minute ocular and orbital structures

- Multiple short pulses are produced with a brief interval that allows the returning echos to be detected, processed and displayed.

- The basis of the echo system is piezoelectric element which is a quartz or ceramic crystal located near the face of the probe


piezoelectric
crystal
sound insulated casing
To electric supply
(for transmitter)
or
amplifier/display
(for detector)
ultrasound
electrodes
plastic window

Types of frequency

- **Low frequency**: orbital tissue

- **Medium frequency** : (7 – 10 mhz)
  Retinal , vitreous , optic nerve

- **High frequency** : (30 – 50 mhz) : anterior chamber up to 5mm

- **DISPLAY MODES**: A SCAN/ B SCAN / BOTH

Examination technique:

The patient is either reclining on a chair or lying on a couch.
The probe can be placed directly over the conjunctiva or lid

Probe positions

- **Transverse** : most common
  - Lateral extent, 6 clock hours
- **Longitudinal** : radial ,1 clock hrs.
  AP diameter in Retinal tumors and tears
- **Axial** : lesion in relation to lens and optic nerve .

Image documentation modes

- They are of 2 types
  - stationary/static
  - moving/dynamic

- The images may be saved in different methods
  - Polaroid photographs
  - 35 mm photo
  - Ink prints
  - Thermal prints
  - Videotapes

Indications

- Anterior segment:

1. Opaque ocular media (i.e. corneal opacities)
   - Pupillary membrane
   - Dislocation / Subluxation lens
   - Cataract / after cataract Posterior capsular tear
   - Pupillary size / reaction

2. Clear ocular media
   - Diagnosis of iris and ciliary body tumors

- Posterior segment:
  1. Opaque ocular media
     - Vitreous haemorrhage
     - Vitreous exudation
     - Retinal detachment (type / extent)
     - Posterior vitreous detachment (extent)
     - Intraocular foreign body (size/ site/ type)

  2. Clear ocular media
     - Tumour (size/ site/ post treatment follow up)
     - Retinal detachment (solid / exudative)
     - Optic disc anomalies
  3. ocular trauma

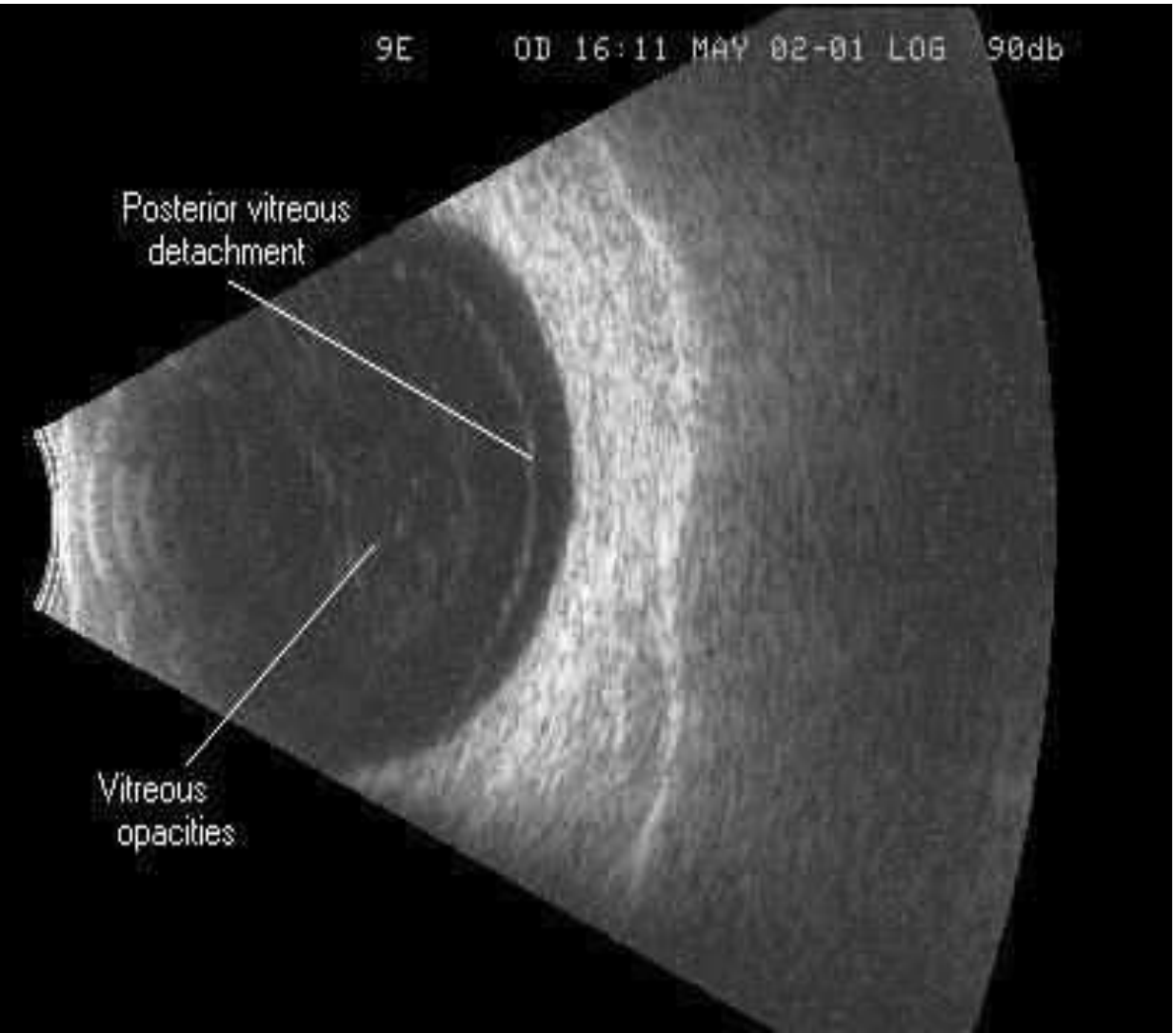
indications of b-scan in pediatric patients

Useful in the following conditions:

- Abnormal size of eye
- Abnormal shape of eye
- Congenital abnormalities
- Vitreous alterations
- Retinal detachments (type/ location)
- Ocular and orbital tumours
- Trauma

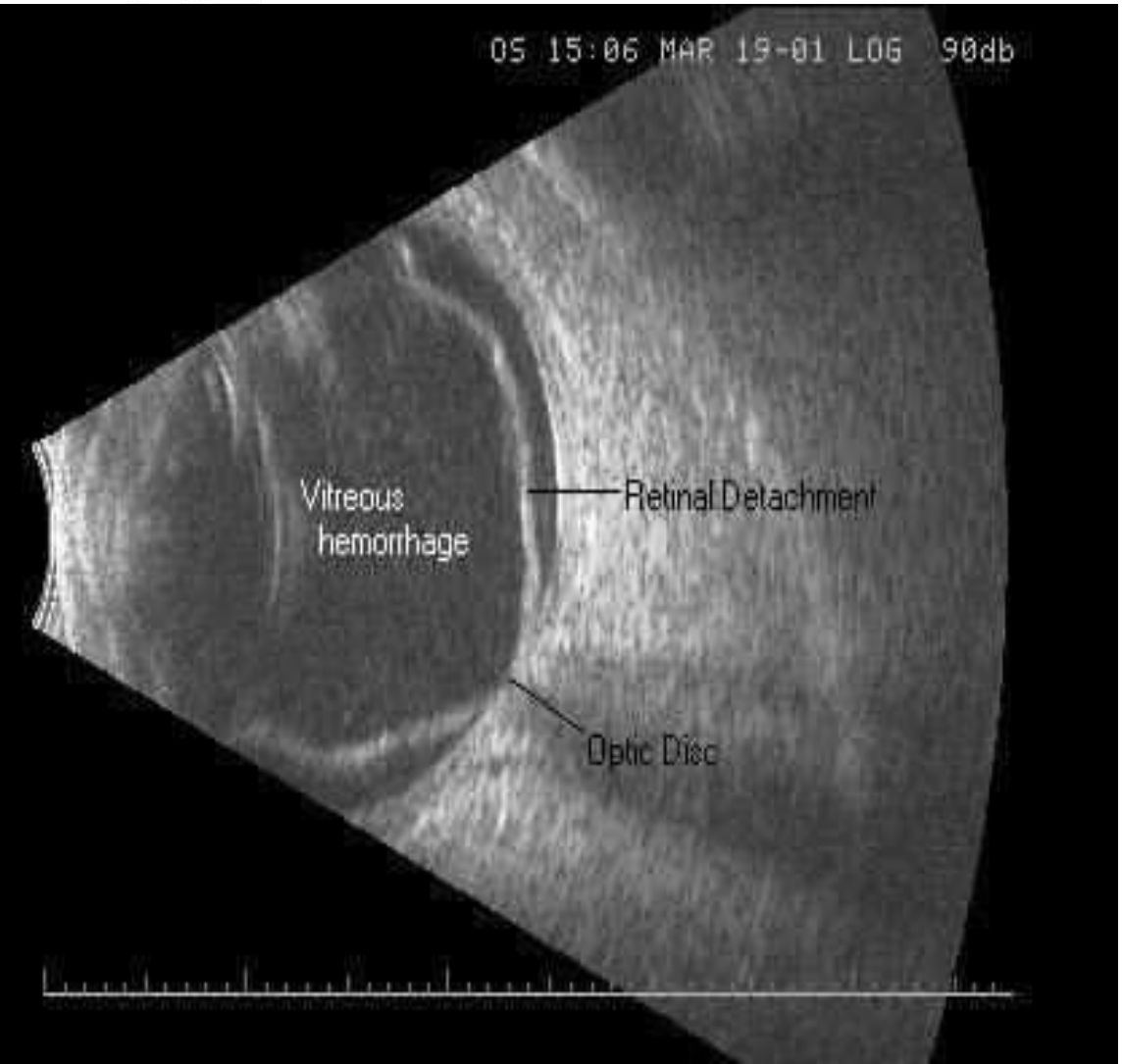
- ULTRASONOGRAPHIC CHARACTERISTICS

POSTERIOR VITREOUS DETACHMENT



Posterior vitreous detachment: The detached posterior vitreous is seen as membranous lesion with no/some attachments to the optic disc

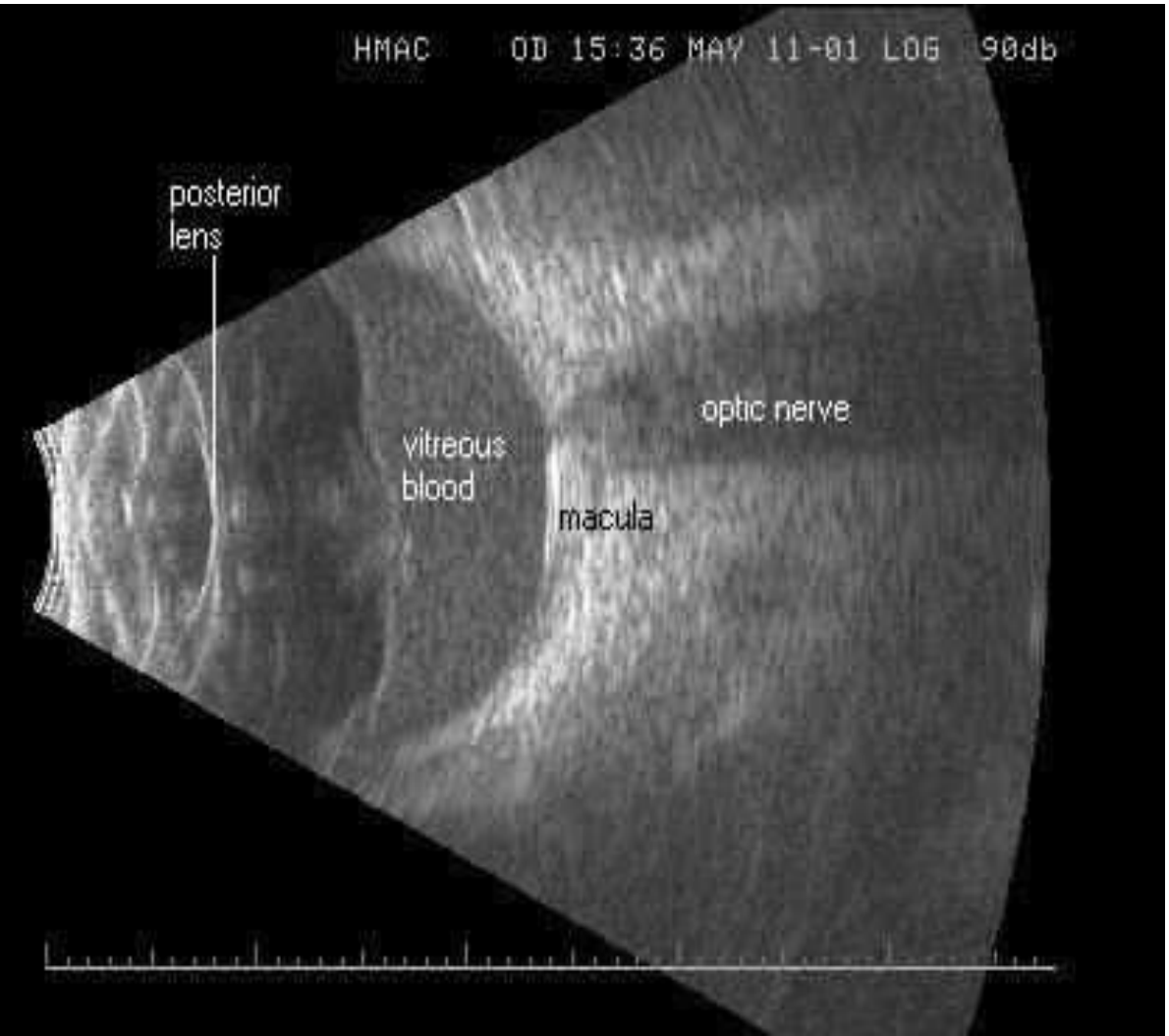
RETINAL DETACHME



The detachment produces a bright continuous, folded appearance with insertion into the disc and ora serrata.

It is to determine the configuration of the detachment as shallow, flat or bullous

VITREOUS HAEMORRHAGE

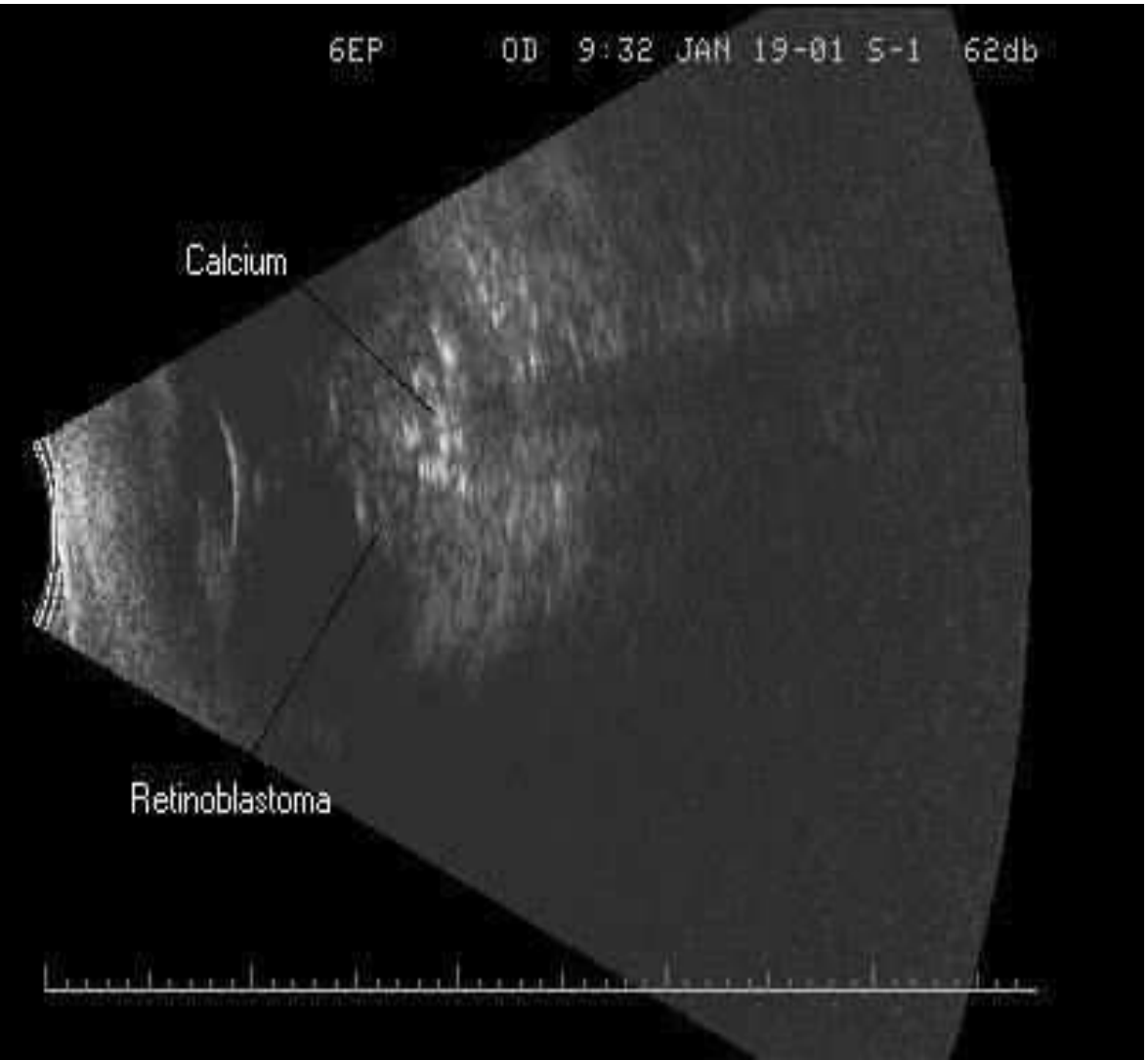


To detect extent, density, location and cause

Fresh haemorrhage shows dots or lines

Old haemorrhage the dots gets brighter

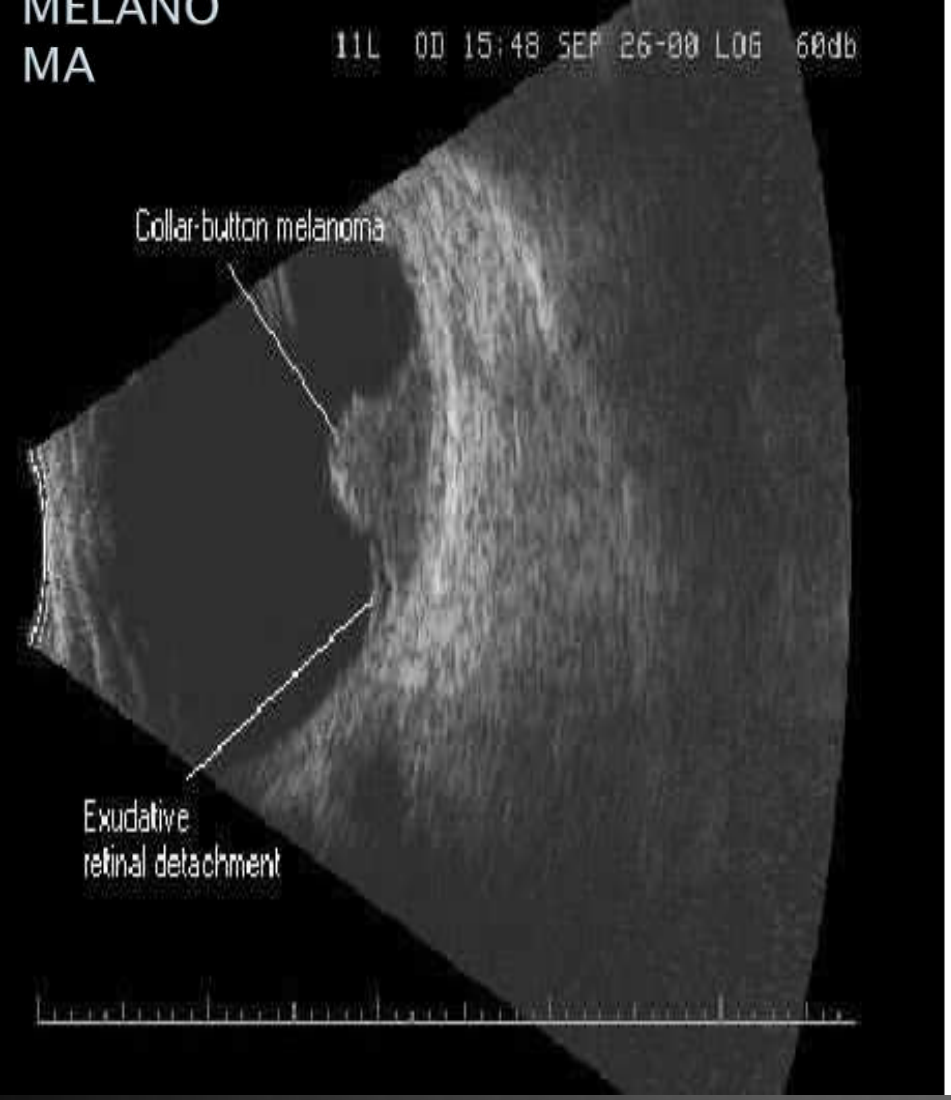
RETINOBLASTOMA

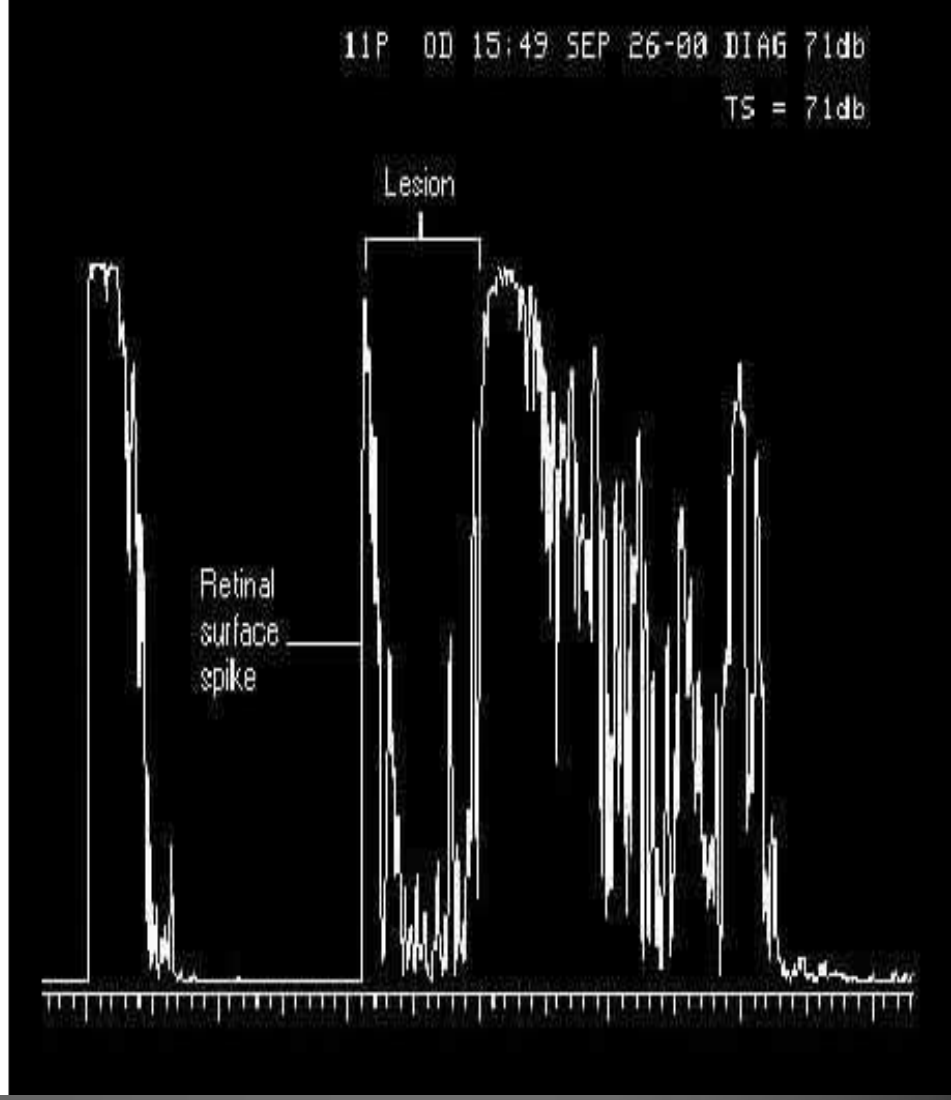


Size of the tumour

Shows irregular configuration

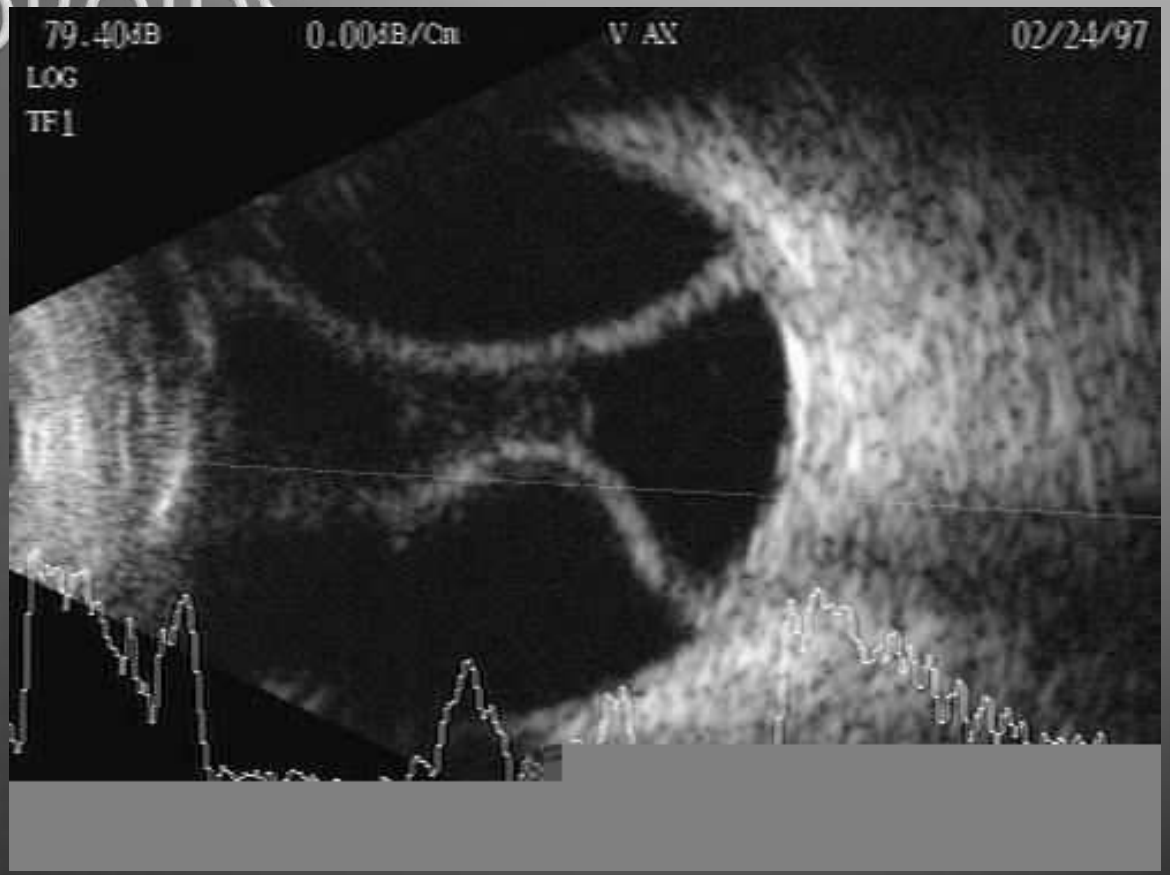
Calcification shows high internal reflectivity



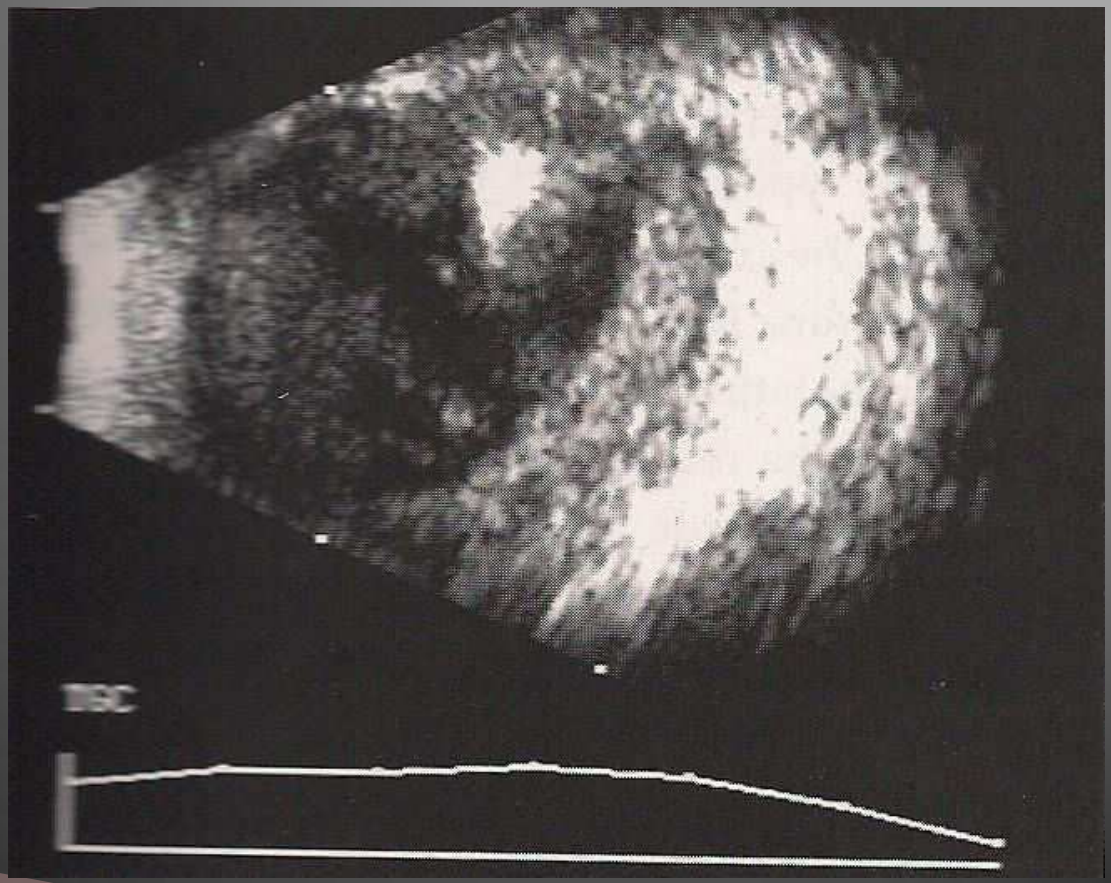


Collar button or mushroom shape.Large tumours shows acoustic hallowing

CHOROIDAL DETACHMENT KISSING CHOROIDS



INTRA OCULAR FOREIGN BODY




THANK YOU

Optic Coherence Tomography

Dr samina

AP Ophthalmology

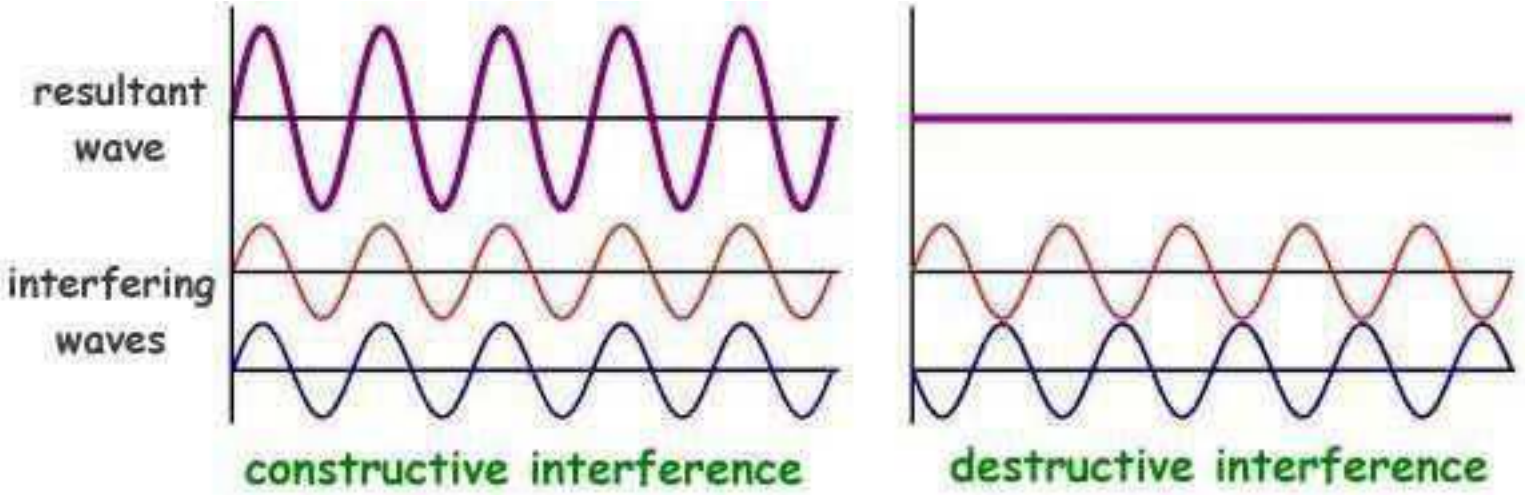
- non contact non invasive
- micron resolution
- cross-sectional study of retina
- correlates very well with the retinal histology

Principle –

Low coherence interferometry

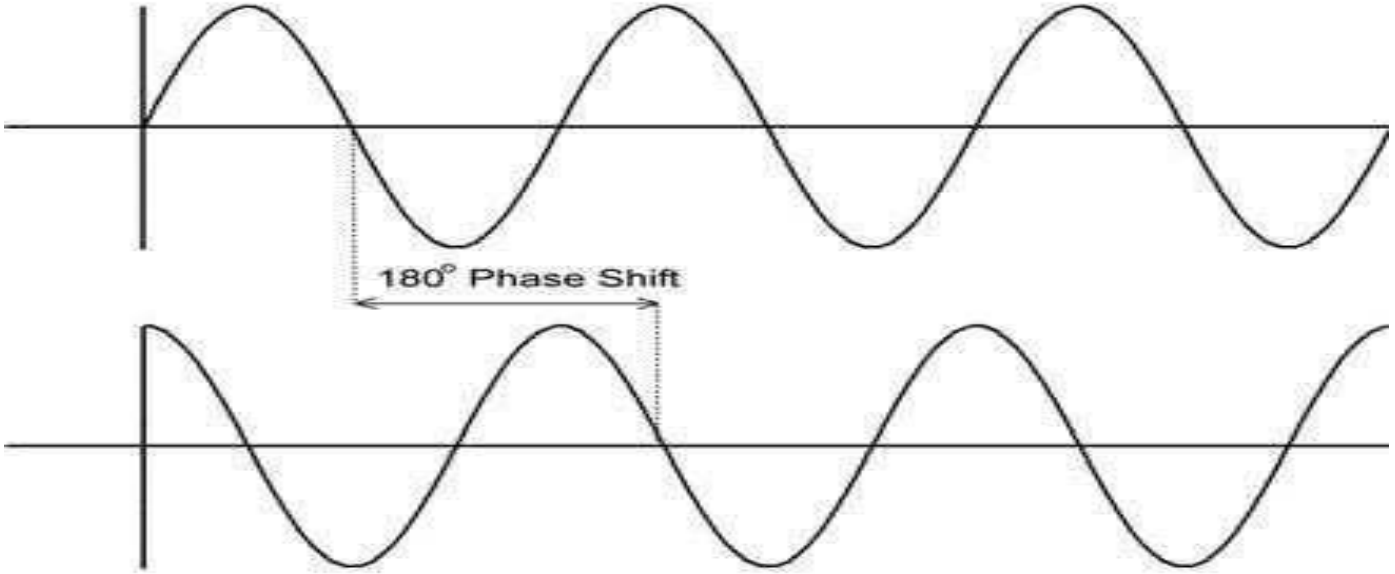
INTERFERENCE

- In physics , interference is a phenomenon in which two waves superimpose to form a resultant wave of greater or lower amplitude

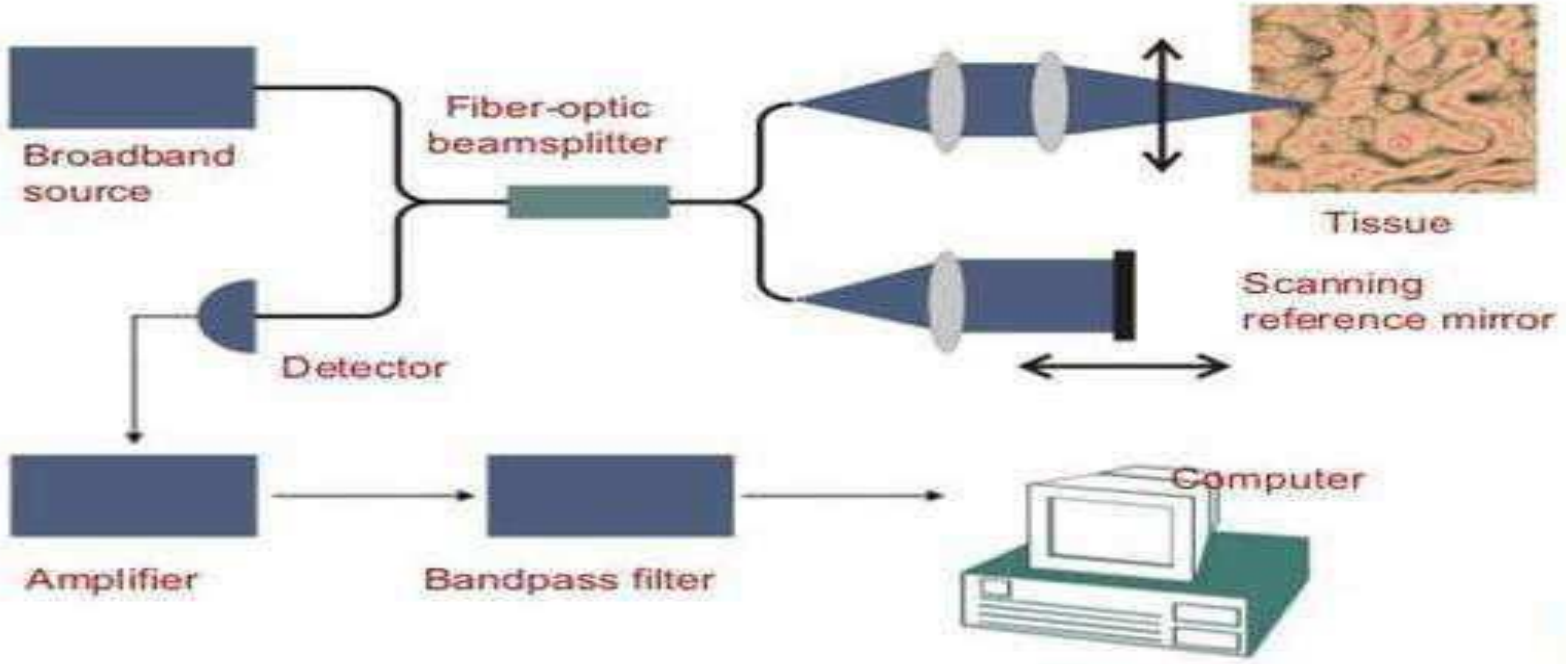


COHERENCE

- In physics two waves are coherent if they have a constant phase difference and same frequency and are non coherent if there is a constant changing phase difference



THE OCT SETUP

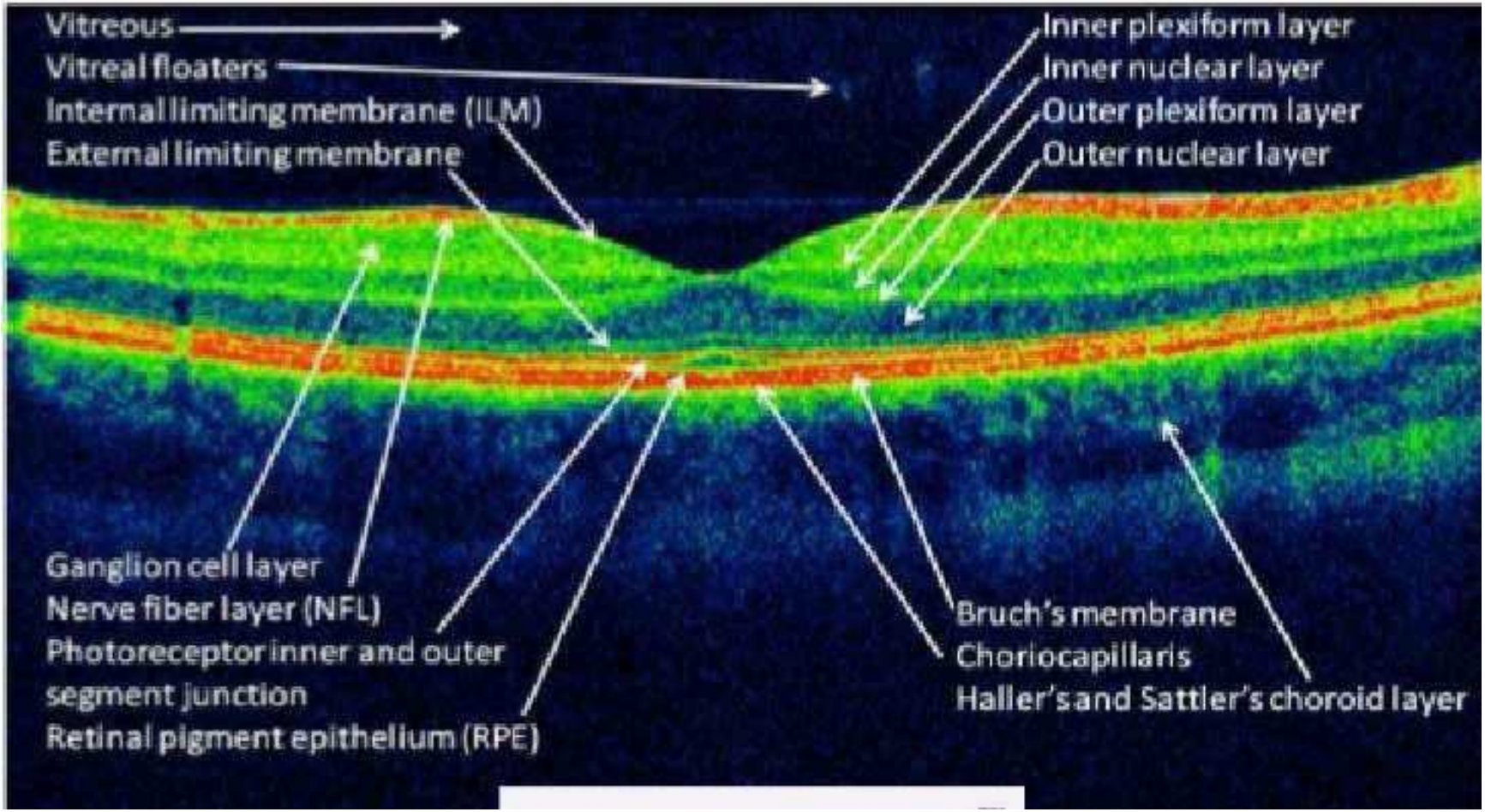


Types of oct

- **Time domain**
  - Reference mirror moves
  - 1 pixel at a time
  - Slow
  - Motion artifacts present
  - Less sharp images

- **Spectral domain**
  - Reference mirror stationary
  - 2048 pixel at a time
  - Rapid
  - No motion artifacts
  - Sharper and clear images

Anatomy of Retina on OCT

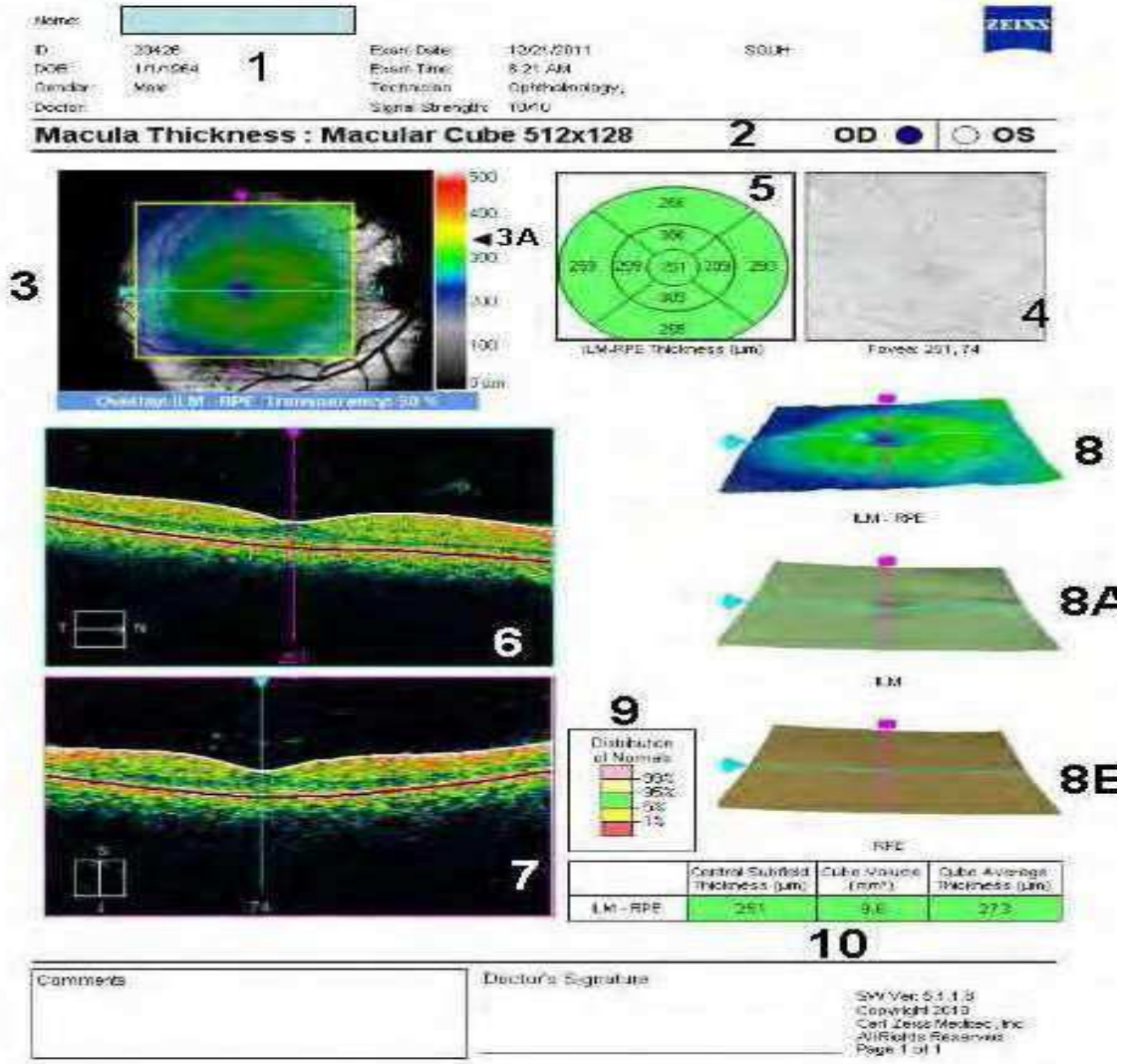


Type of Scan

- **POSTERIOR SEGMENT SCAN**
  - MACULAR SCAN
  - OPTIC DISC SCAN
  - RNFL THICKNESS ANALYSIS SCAN

- **ANTERIOR SEGMENT SCAN**

PRINT OUT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DOB: 1/1/1964
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Doctor:
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Exam Time: 8:21 AM
Technician: Ophthalmology,
Signal Strength: 10/10
SOLIH
ZEISS
Macula Thickness : Macular Cube 512x128
2
OD
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500
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3A
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Fovea: 251, 74
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8E
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Distribution of Normals
99%
95%
5%
1%
Central Subfield Thickness (µm)
Cube Volume (mm³)
Cube Average Thickness (µm)
LM - RPE
251
9.6
273
10
Comments
Doctor's Signature
SW Ver: 5.1.1.8
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Page 1 of 1

- **ADVANTAGES OF OCT**
- Its noncontact unlike USG, and noninvasive, unlike FFA,ICG.
- Children easily tolerate it.
- Very helpful for quantitative information about macular thickness.
- Valuable teaching tool for the ophthalmologist as well as patient.

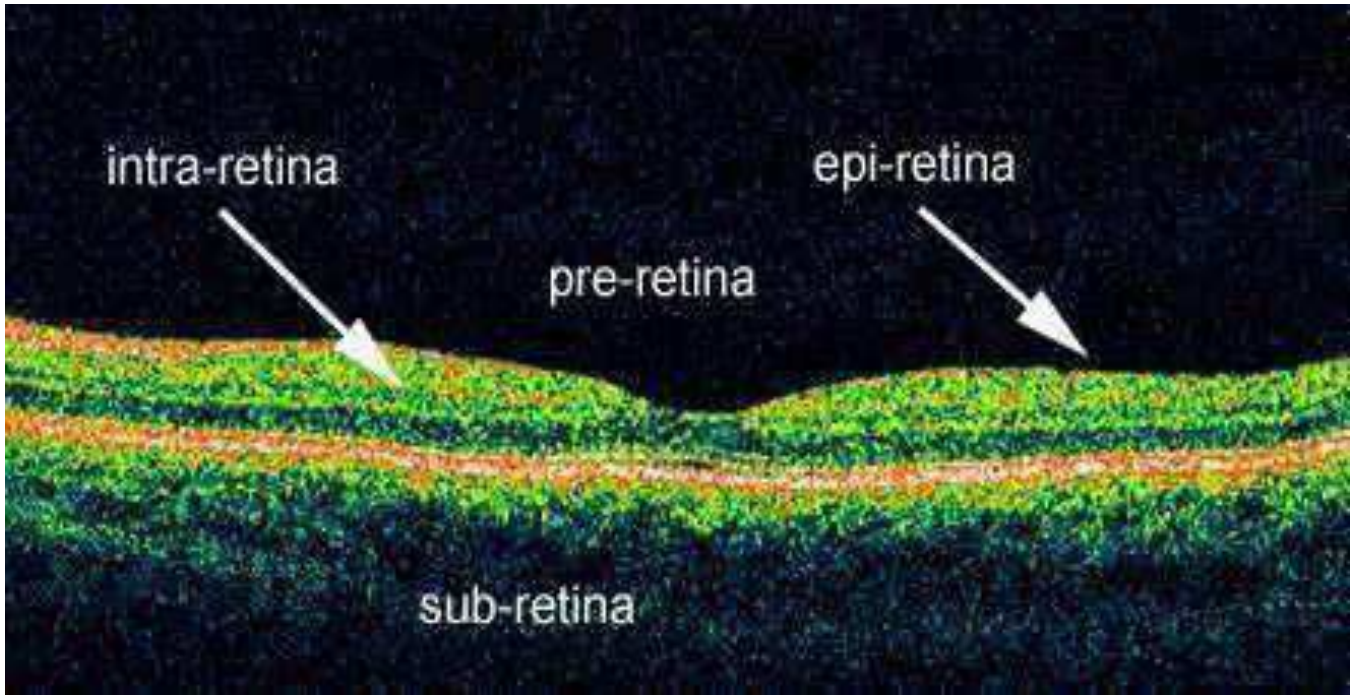
- **DISADVANTAGES**
- Media opacity.
- Scan quality depends on the skill of OCT operator.
- Not possible with uncooperative patients.
- Measurement of Fovea Thickness not accurate if scan not through the center of fovea.

Uses/ indications

- Neurological
- Ophthalmological
- Other uses

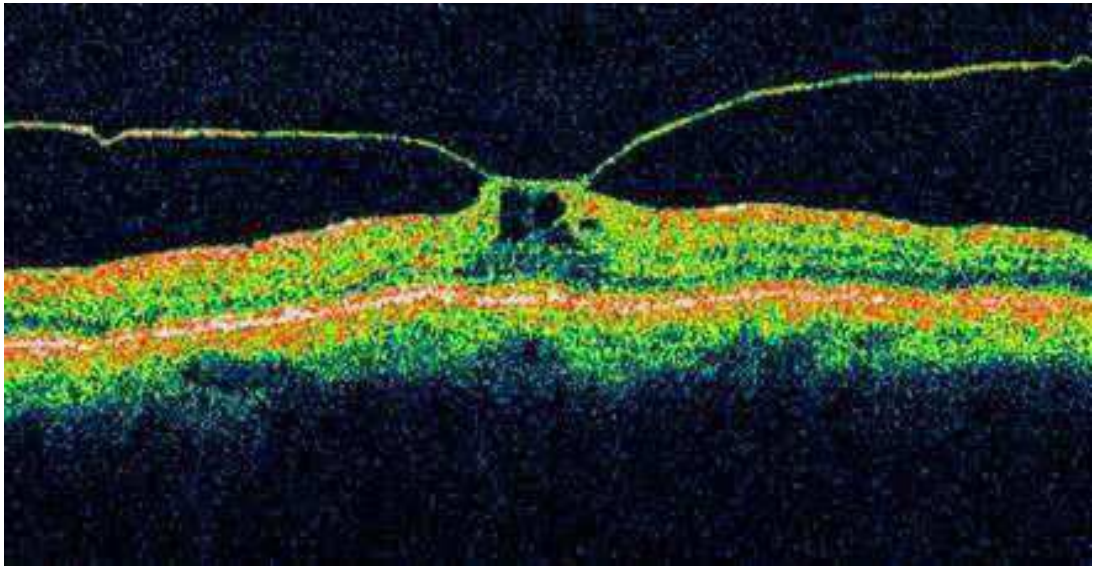
Opthalmological uses

- For purposes of analysis, the OCT image of the retina can be subdivided vertically into four regions
  - Pre-retina
  - Epi-retina
  - Intra-retina
  - Sub-retina



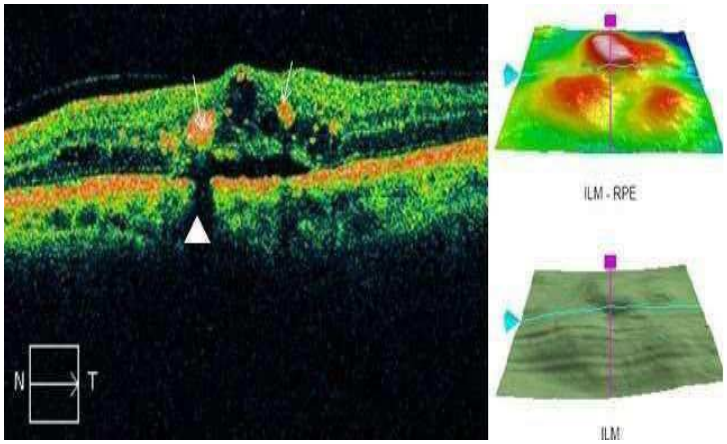
Pre retinal and epiretinal pathology

- **Anomalous structures**
  - pre-retinal membrane
  - epi-retinal membrane
  - vitreo-retinal strands
  - vitreo-retinal traction
  - pre-retinal neovascular membrane
  - pre-papillary neovascular membrane



Intra retinal pathology

- Choroidal neovascular membrane
- Diffuse intra-retinal edema
- Cystoid macular edema
- Drusen
- Hard exudates
- Scar tissue
- RPE tear



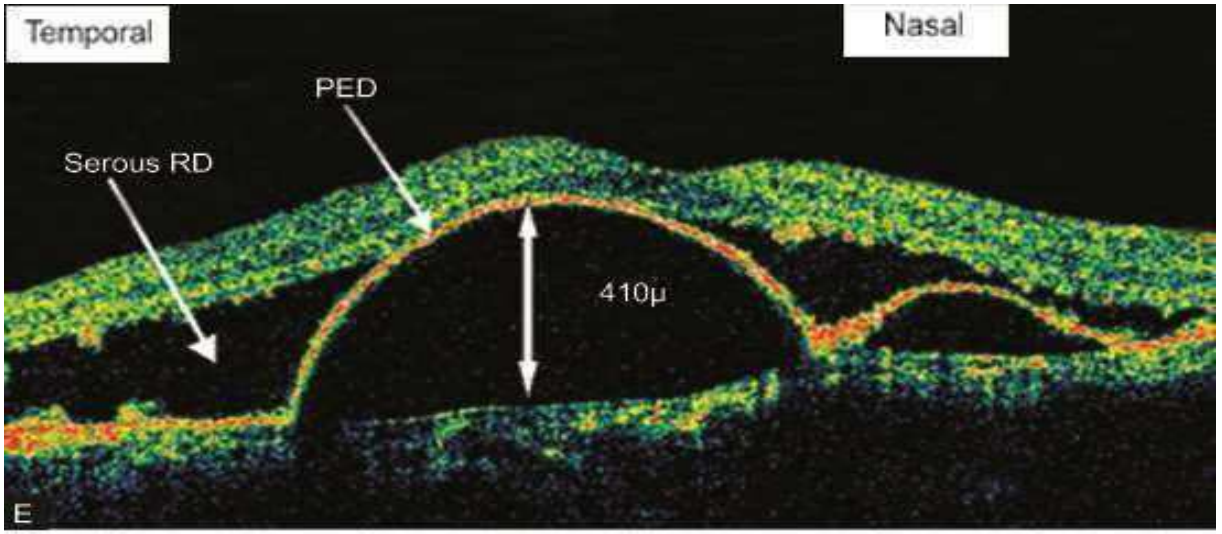


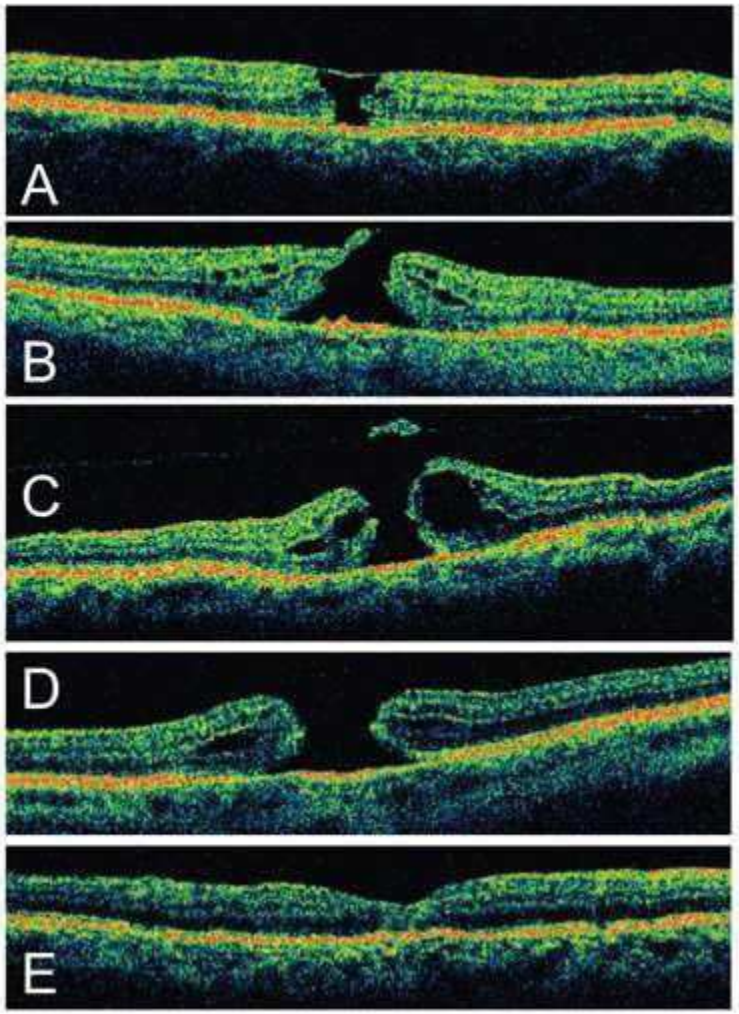
Fig. 6-2e

- Scan of posterior segment pathology

1.Macular Hole

•confirmation of diagnosis and differentiates it from lamellar hole, foveal pseudo cyst.

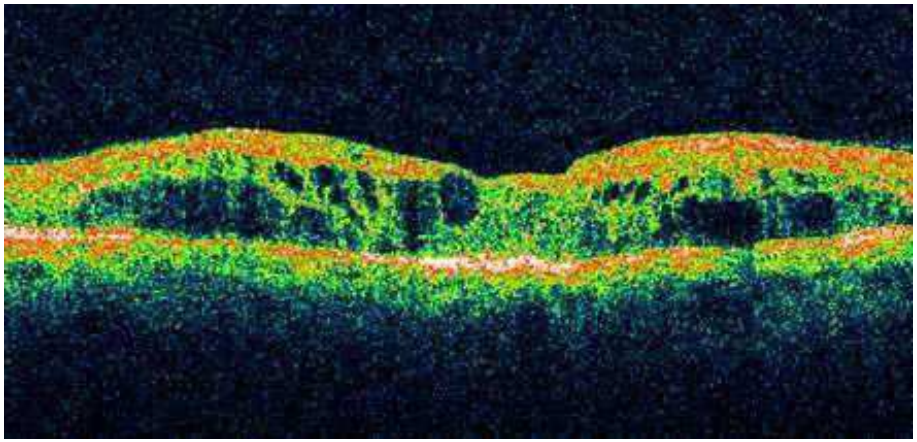
•monitoring the course of the disease and the response to surgical intervention.



2.Macular Edema

•: intraretinal areas of decreased reflectivity and retinal thickening.

•Round, optically clear regions within the neurosensory retina are noted in cystoid macular edema.



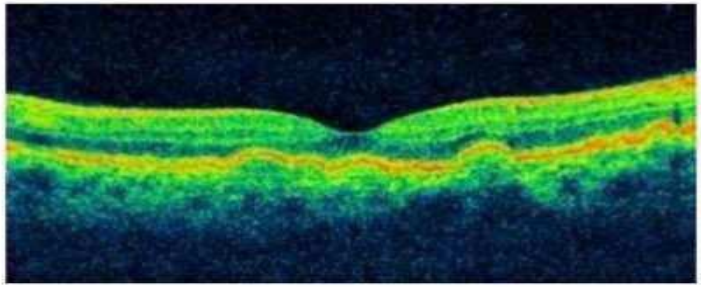
3. ARMD

•Morphological changes in the non exudative ARMD.

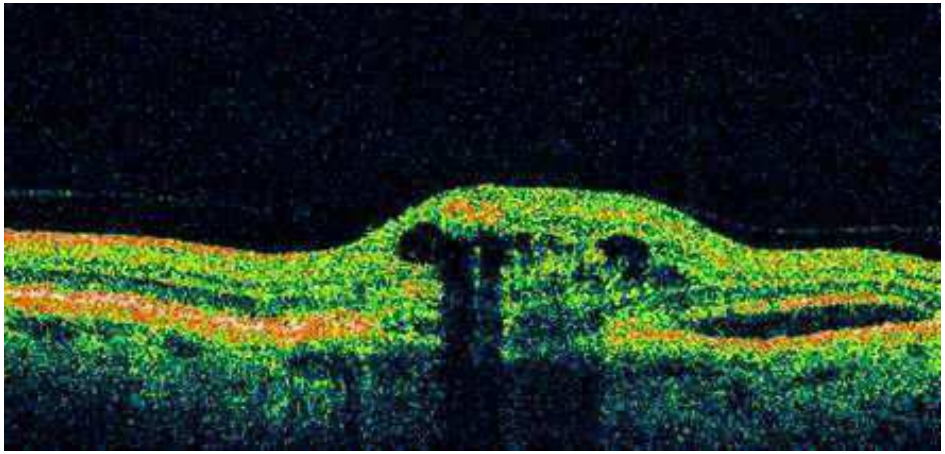
•Subretinal fluid, intraretinal thickening and

•sometimes, choroidal neovascularization in exudative ARMD.

Drusens- seen between bruch's membrane and RPE

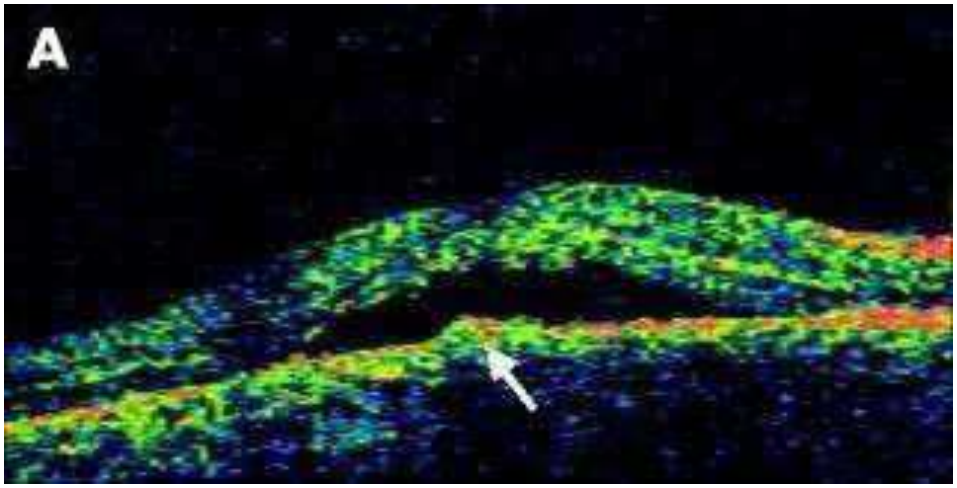


• hyperreflective bumpy RPE with localised PED

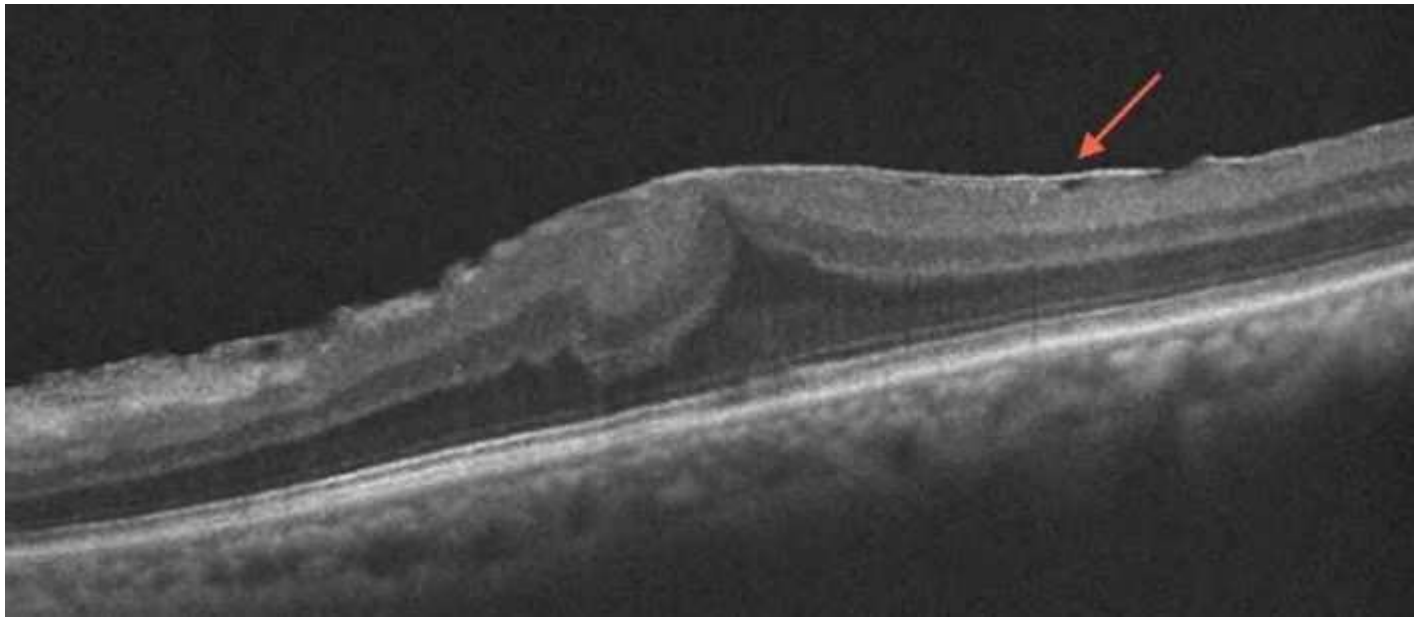


4. Central serous retinopathy

- area of decreased reflectiv ity between two hyper reflective areas



5. Epiretinal membrane:
highly reflective diaphanous membrane over the surface of retina.

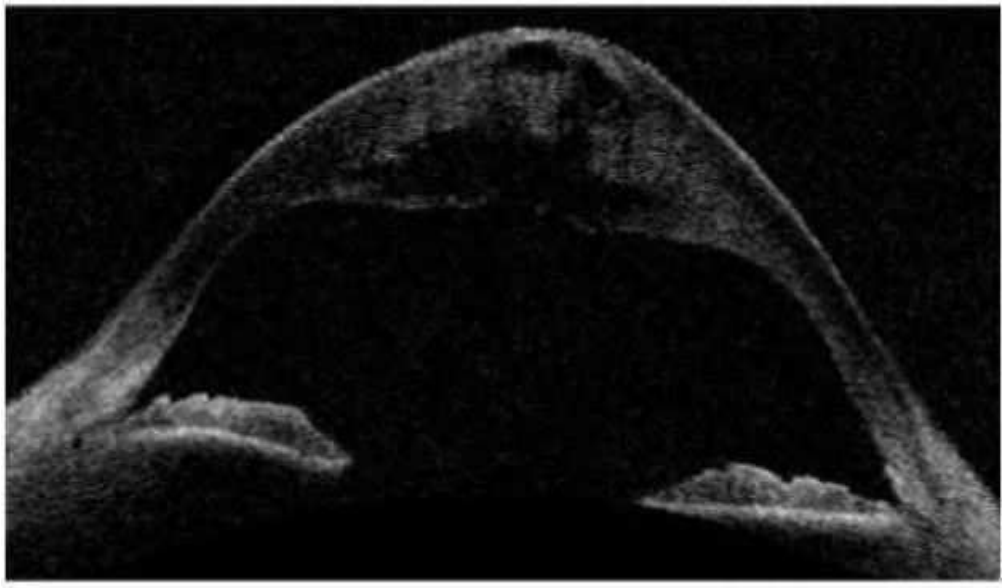


OCT IN GLAUCOMA

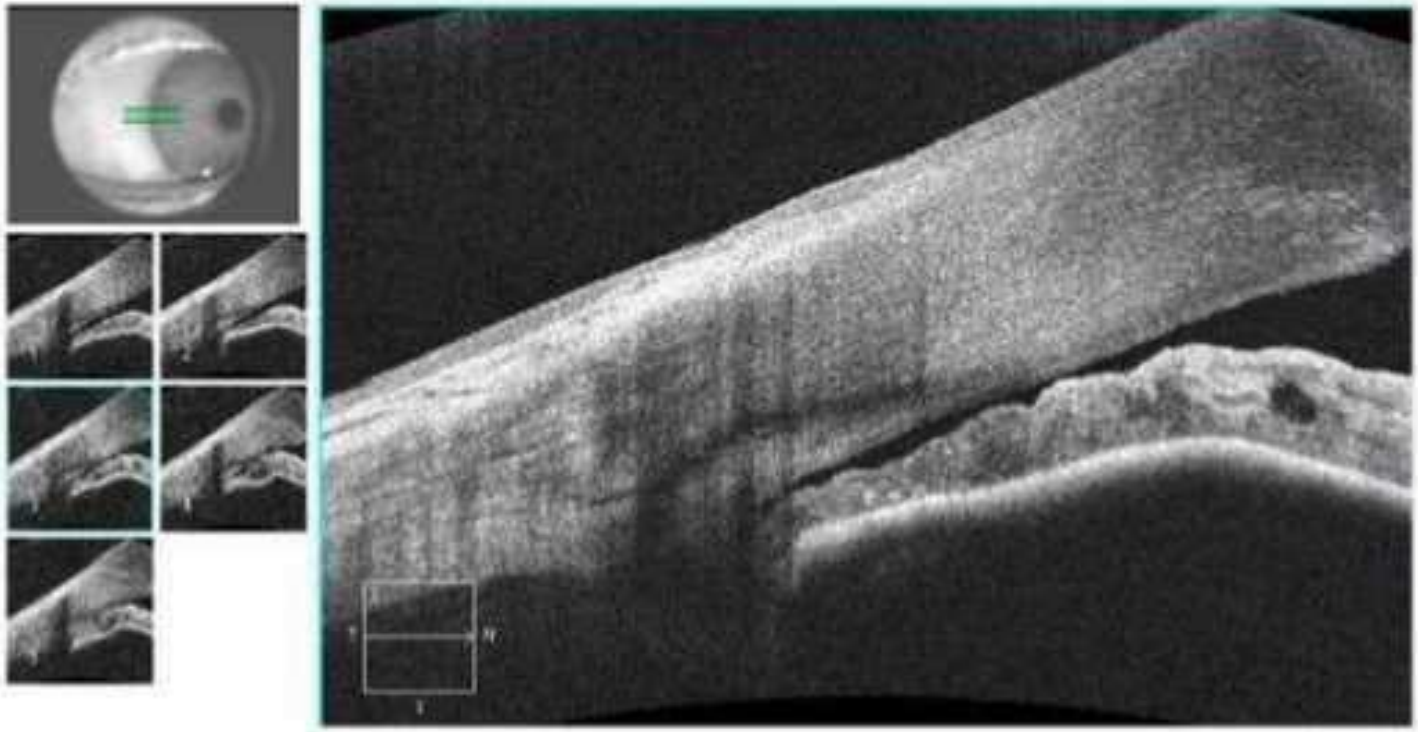
- Diagnosing and monitoring the glaucomatous change.
- Evaluating the RNFL for early (pre- perimetric) glaucoma detection.
- Evaluation of cystoid macular edema after combined cataract and glaucoma surgery.

ANTERIOR SEGMENT OCT

- Corneal thickness and keratoconus evaluation
- Anterior chamber angle
- Assessing the fit of intraocular lens implants



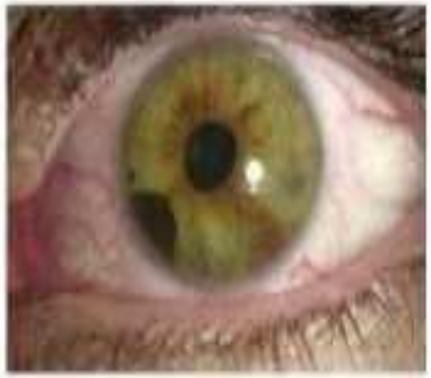
- Results of corneal implants



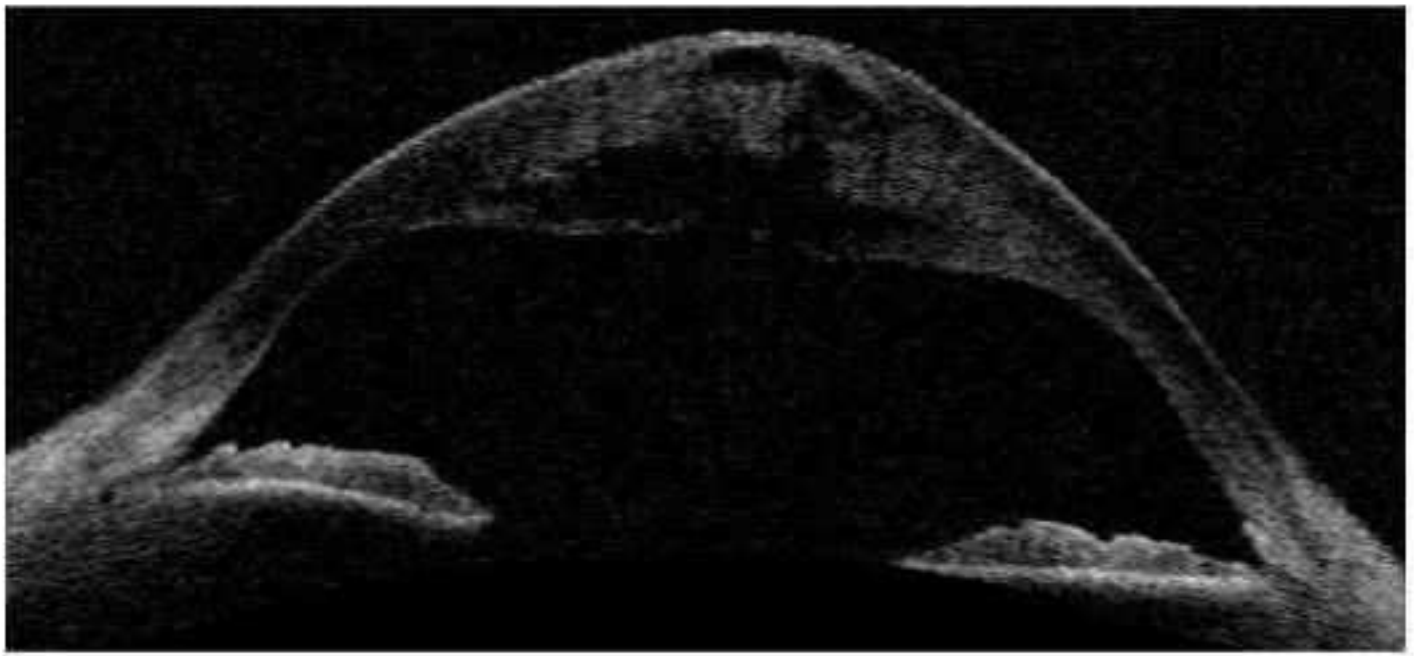
Images courtesy of Martha Leen, M.D. & Paul Kreiner M.D. Achieve Eye and Laser Specialists, Silverdale, WA

Narrowing of angle of anterior chamber

Tumor of the iris



Keratoconus



- Conical cornea with central stromal thinning

Limitations

- Quality of OCT depends on the transparency of the ocular media
- OCT is operator dependent
- The statistical analysis is based on a control population, which may not be accurate depending on the population studied

Thank you

Uses of visual field

Dr samina

Definition

- Island of vision in sea of blindness.
- The peak of the island represents the point of highest acuity, the fovea
- while the “bottom less pit” represents the blind spot, the optic disc.

Normal Monocular & Binocular Field

- Monocular ;
  - Nasal side -50° to 60°
  - Superior side - 60° to 70°
  - Inferior side - 70° to 80°
  - Temporal side - 100°to110°
- Binocular:
  - Vertical field -110 °
  - Horizental field - 200 °

Visual Field Testing

- 1.Stimuli: Testing the island of vision at various levels requires targets that vary in
  - (a) Size
  - (b) Intensity
  - (c) Colour
- 2. Field Test Methods:
  - Kinetic; Mapping the contours of the island at different levels, resulting in one Isopter for each level tested.
  - Static; Vertical contours of the island along a selected meridian.

Clinical testing methods

- Central fields - Below 30°
- Peripheral fields – Above 30° to 360°
- **Central Fields:**
  - *Confrontation method*
  - *Amsler's grid*
  - *Bjerrum's screen*
- **Peripheral Fields:**
  - *Listen perimeter*
  - *Goldmann perimeter*

Uses of visual field

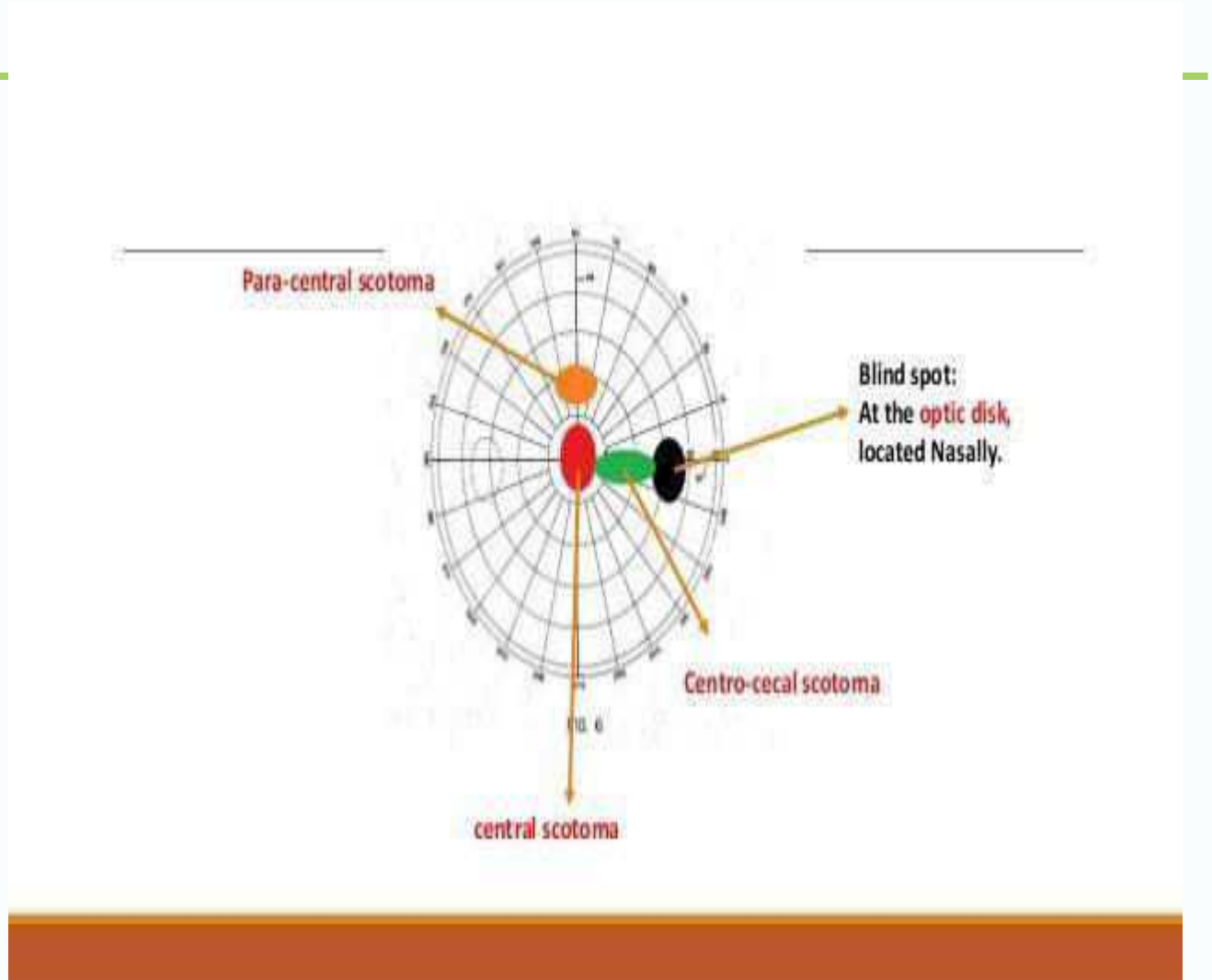
- Uses of visual field in Retina
- Uses of visual field in Glaucoma
- Uses of visual field in Neuro

Visual Field defect

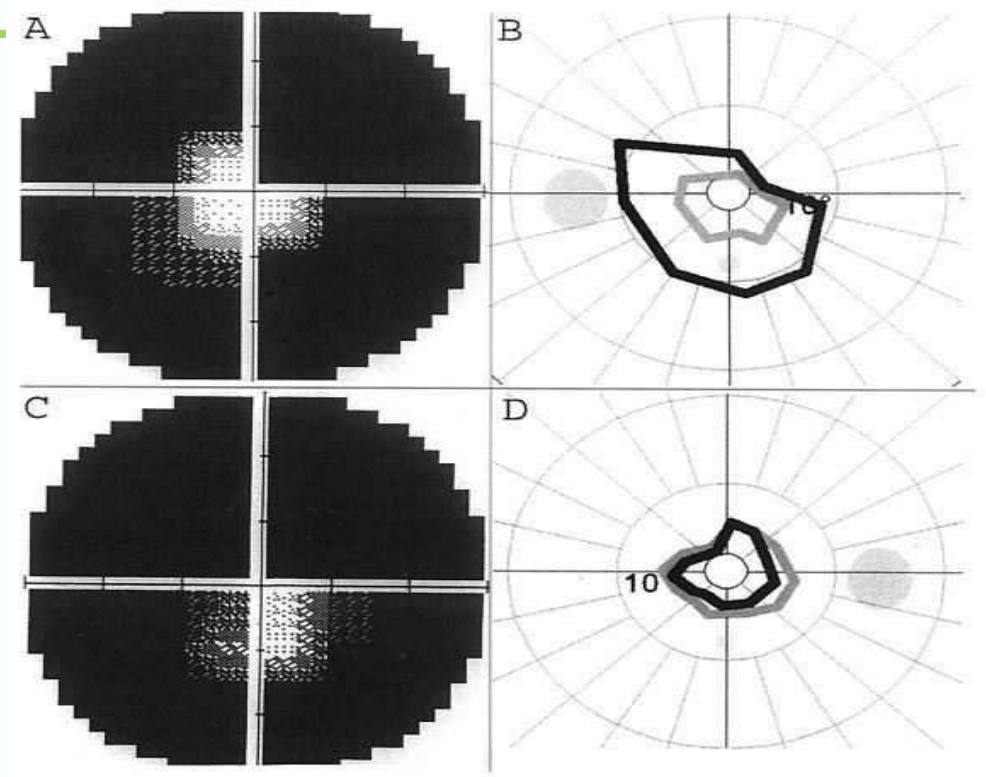
- **Central Scotoma:**
  - *Central serious Retinopathy*
  - *Macula Degeneration*
  - *Macular Oedema (any macular disease)*

- **Centro-cecal scotoma**
  - *Toxic Amblyopia*
  - *Stargadits*

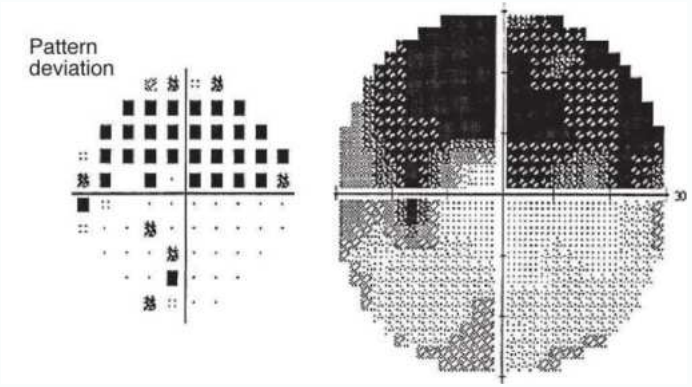
- Central Scotoma
- & Centro-cecal



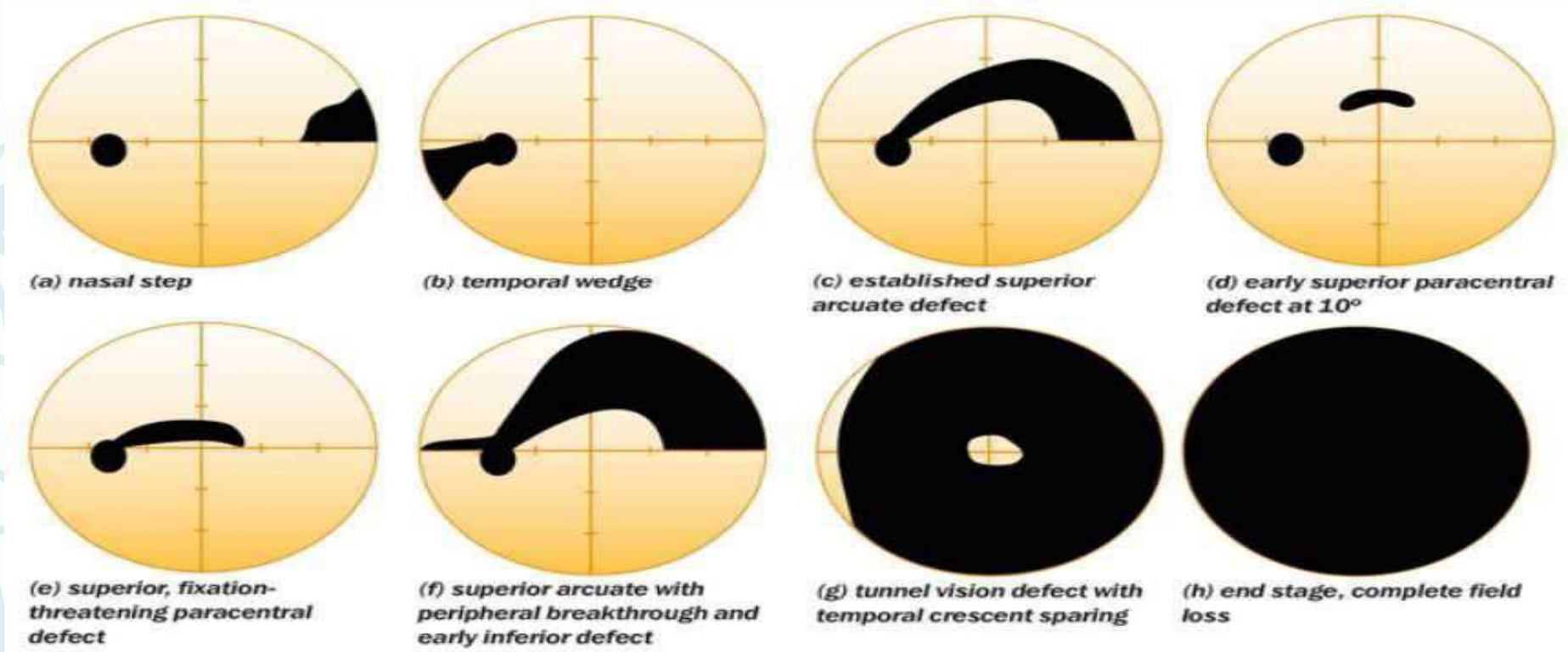
- **Contraction & Tubler field defect**
  - Retinitis pigmentosa
  - High myopia
  - CRAO with Sparing of cilioretinal artery



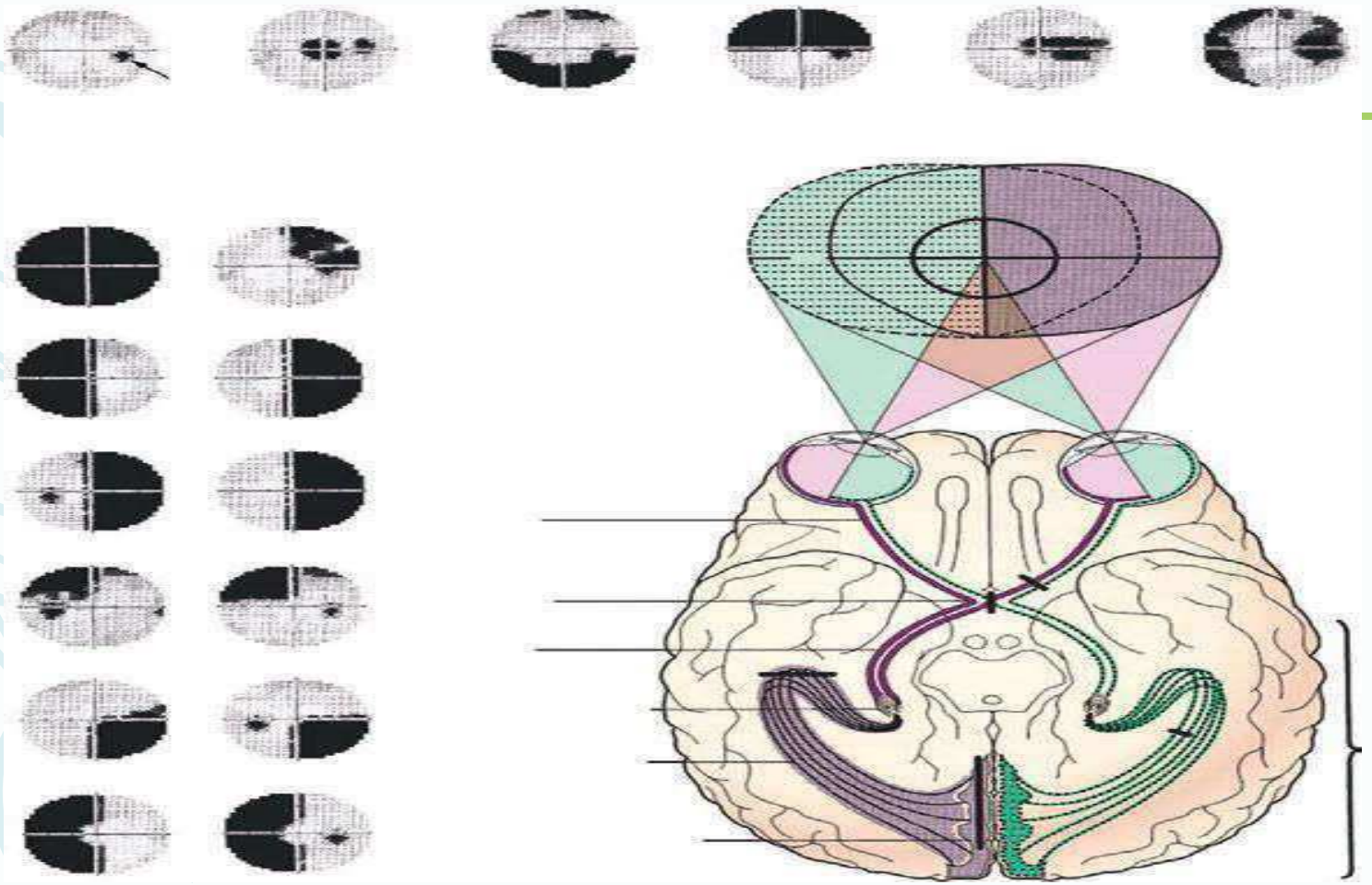
- **Altitudinal defect:**
  - *AION Depression:*
- **Depression;**
  - *Retinal Detachment*
- **Ring Scotoma:**
  - *High myopia*
  - *Aphakic spectacle correction*
  - *Retinitis pigmentosa*
  - *Pan retinal photocoagulation*



Visual Field Defects in glaucoma



Neurological Visual Field Defects



Diseases of the Optic nerve

- Congenital &
- Hereditary
- Traumatic
- Tumour
- Inflammatory
- Toxic
- Vascular Lesion

Congenital optic nerve defect

- Mylinated nerve fiber defect
- Coloboma
- Hypoplasia
- Drusen
- Optic nerve pits
- Tilted disc

- **Mylinated nerve fiber defect**
  - *Blind spot enlargement*
  - *Paracecal scotoma*
  - *Ring scotoma*
  - *Central scotoma*
- **Coloboma**
  - *Superior nasal depression*
  - *Contraction*
  - *Superior altitudinal hemianopia*

- **Hypoplasia:**
  - *Central Scotoma*
  - *Binasal & Bitemporal hemianopia*
  - *Bilateral inferior extension of blind spot*
- **Drusen:**
  - *Blind spot enlargement*
  - *Irregular nerve fiber bundle scotoma*

- **Optic nerve pits:**
  - *Blind spot enlargement with or without macula involvement.*
  - *Central scotoma*
  - *Altitudinal hemianopia*
  - *Upper temporal field defect*
- **Tilted disc**:
  - *Upper temporal defect which may be mistaken for chiasmal compression.*

Hereditary Field defect

Optic atrophy ;

Contraction

Tubler Field

Retinitis Pigmentosa

contraction

Ring scotoma

Tubler field

Traumatic Field defect

- **Traumatic optic neuropathy:**
  - *Superior altitudinal defect*
  - *Total Blind*

Optic nerve tumour

- **Glioma:**
  - *Blind spot enlargement*
  - *Contraction*
  - *Tubler field*
- **Meningioma:**
  - *Junctional scotoma*
  - *Central scotoma*
  - *Upper temporal field defect*

Inflammatory Field defect

- **Papillitis:**
  - *Central scotoma*
  - *Centro-cecal scotoma*
  - *Para central scotoma*
  - *Blind spot enlargement*
- **Retro bulbar neuritis:**
  - *Sectoral scotoma*
  - *Ring scotoma*
- **Neuro retinitis:**
  - *Central Scotoma*
  - *Centro- cecal scotoma*

Toxic Field defect

- **Mild Toxic :**
  - *Central scotoma*
  - *Centro- cecal scotoma (BE)*
- **Severe Toxic** :
  - *Peripheral contraction*
  - *Total blind*

Vascular lesion Field defect

- Anterior Ischemic optic neuropathy:

- Altitudinal hemianopia(mainly involving the inferior half)

Lesion due to pressure

- **Papilloedema:**
  - *Blind spot enlargement*
  - *Peripheral contraction*
  - *Total loss of visual field*

Chiasma Field defect

- **Infra chiasmatic lesion:**
  - *Bitemporal hemianopia*
- **Supra chiasmatic lesion:**
  - *Central hemianopic scotoma*
  - *Junctional scotoma*
  - *One eye is more field defect and other is less field defect (inferior temporal)*

- **Posterior side:**
  - Infra Temporal field defect
  - Bilateral inferior quadrant scotoma (near fixation point)
  - Bitemporal hemianopia

- **Optic Tract Field defect**
  - Part I: Homonymus hemianopia
  - PartII: Incongruous hemianopia
- **LGB Field defect**
  - Congruous Homonymus lower Quadrantropia
  - Congruous Homonymus upper Quadrantropia
- **Optic radiation Field defect**
  - Pie in the Sky (superior homonymus quadrantropia)
  - Pie in the floor (Inferior homonymus quadrantropia)

Visual Cortex Field defect

- Congruous homonymus hemianopia
- Congruous quadrantropia
- Homonymus hemianopia with macula sparring
- Homonymus hemianopia with macula splitting
- Altitudinal hemianopia
- Tubler field

thanks

FUNDUS FLUORESENCIN ANGIOGRAPHY

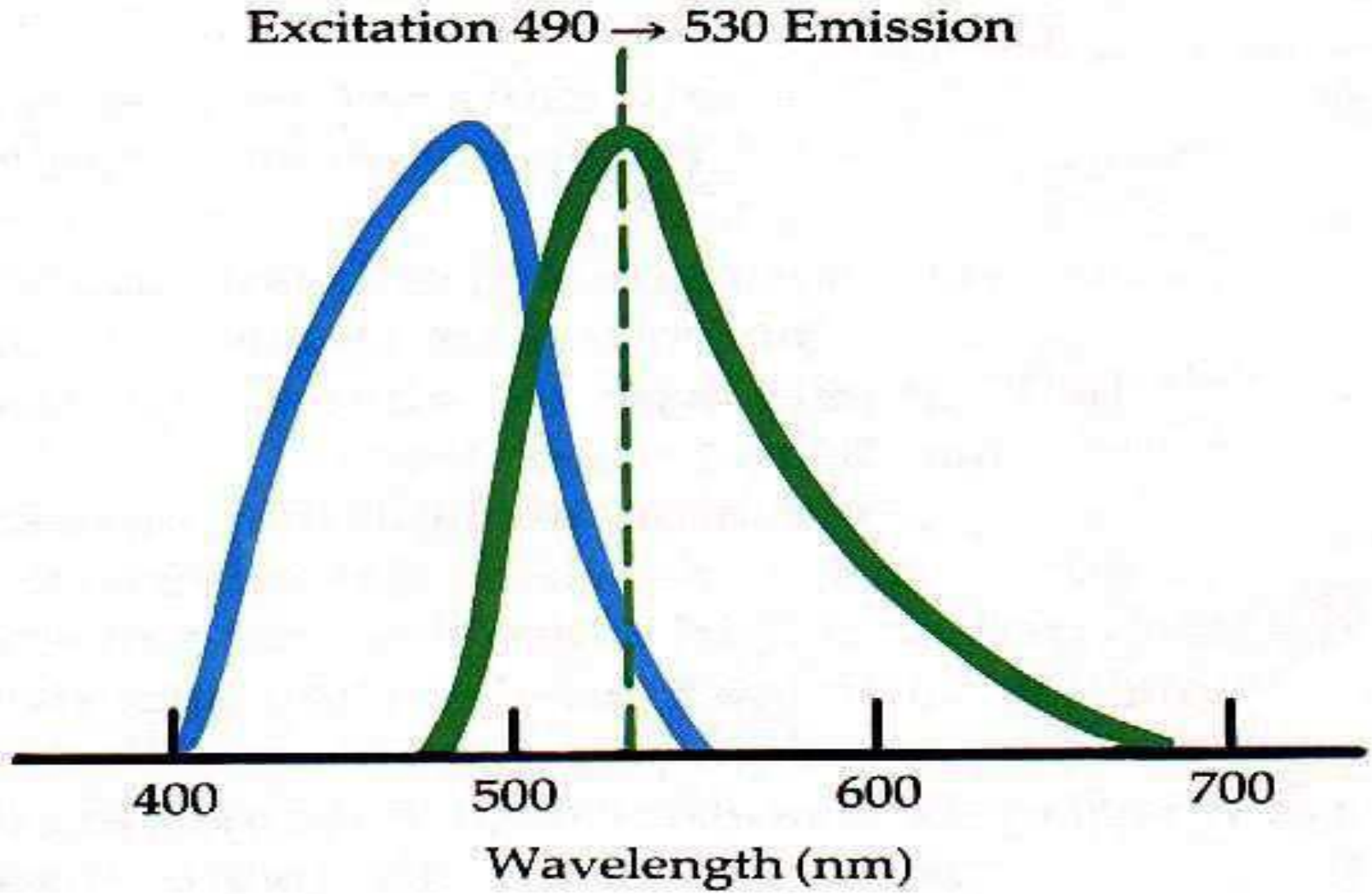
Dr Samina karim

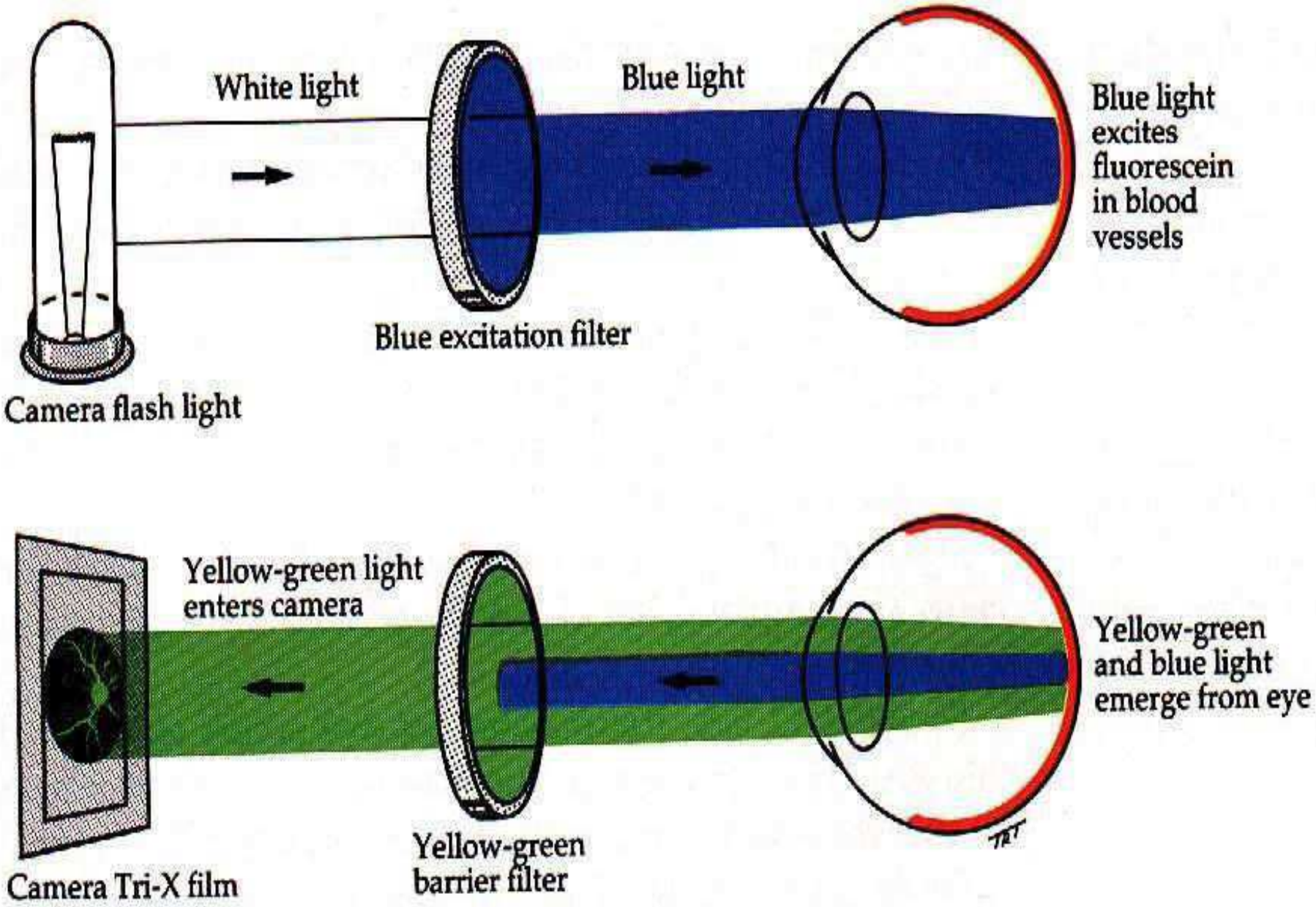
Assistant professor ophthalmology, HMC

Principle

- ***FLUORESCENCE*** :- Property of the certain molecules to emit light energy of longer wave length when stimulated by a shorter wavelength.
- Absorbs light in the blue range peaking at **465-490 nm**
- Emits light of yellow-green range of visible spectrum peaking at **520-530nm.**

EXCITATION AND EMISSION




White light
Blue light
Blue light excites fluorescein in blood vessels
Blue excitation filter
Camera flash light
Yellow-green light enters camera
Yellow-green and blue light emerge from eye
Yellow-green barrier filter
Camera Tri-X film

sodium fluorescein

- Fluorescein(sodium fluorescein) is an orange water-soluble dye that, when injected intravenously,
- Remains largely intravascular (>70% bound to serum proteins).
- It is excreted in the urine over 24–36 hours.
- FFA involves photographic surveillance of the passage of fluorescein through the retinal and choroidal circulations following intravenous injection.

PROCEDURE

- Patient is informed of the normal procedures, the side effects and the adverse reactions.

- Dilating the pupil
- Made to sit comfortable.
- 3-4 red free photographs taken.(**control photographs**)

- 5ml of 10% or 3ml of 25% fluorescein dye injected through the anticubital vein
- wait for 10 – 12 seconds(normal arm-retina time)
- Photos are taken at 1 second interval for 10 seconds
- Then every 2 seconds interval for 30 seconds
- Late photographs are usually taken after 3 ,5 and 10 minutes

TECHNIQUE

CIRCULATION

Peripheral vein

↓

venous circulation

↓

heart

↓

arterial system

↓

INTERNAL CAROTID ARTERY

↓

Ophthalmic artery

↙ ↘

Short posterior ciliary artery (choroidal circulation)

Central retinal Artery (retinal circulation)

PHASES OF NORMAL ANGIOGRAM

- Prearterial phase (choroidal phase)
- Arterial phase
- Arterio -venous phase
- Venous phase
    - early venous
    - mid venous
    - late venous
- Late phase

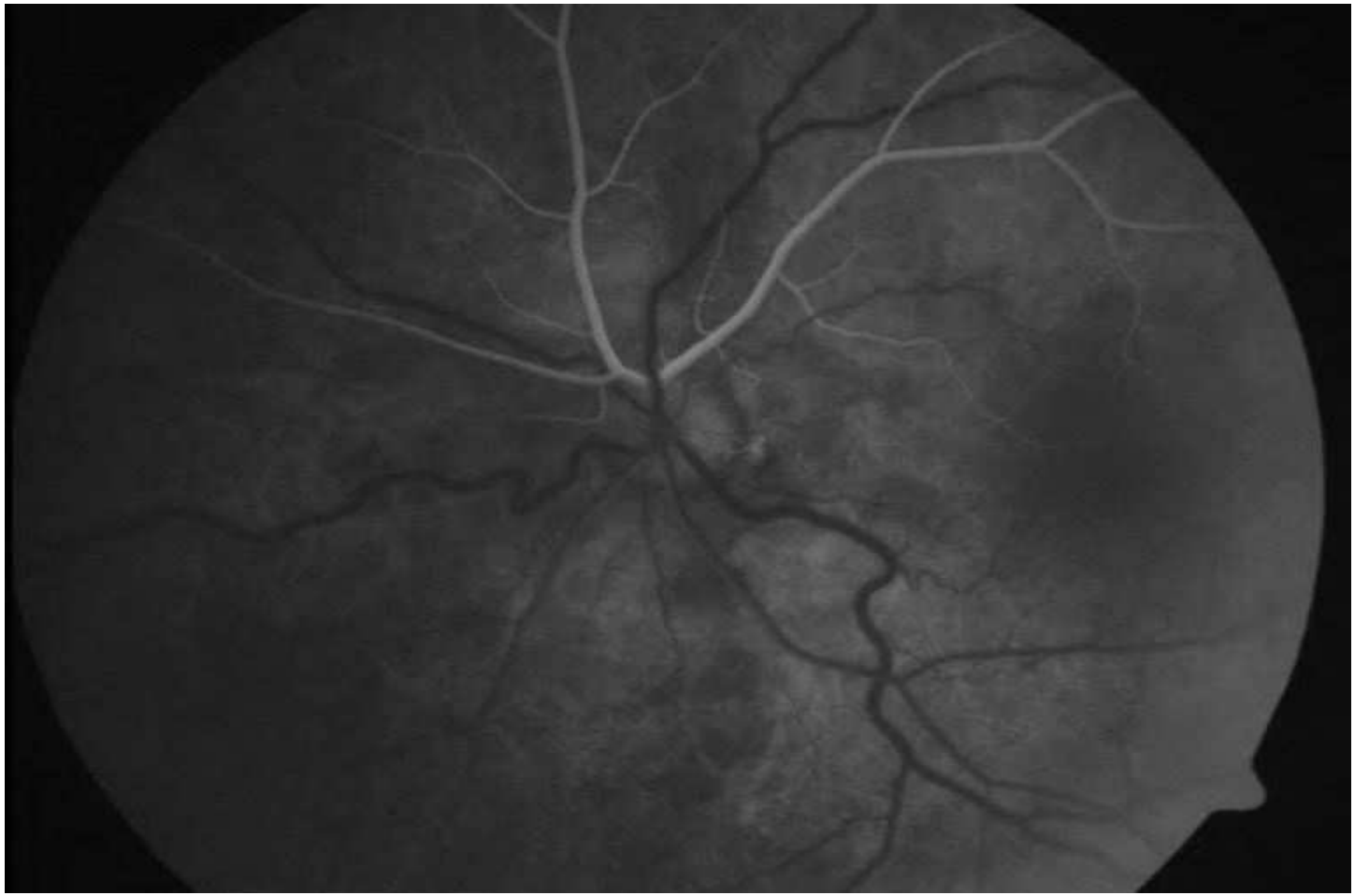
Choroidal phase

- 10 -12 seconds
- Initially patchy filling → diffuse filling → dye leaks from choriocapillaris

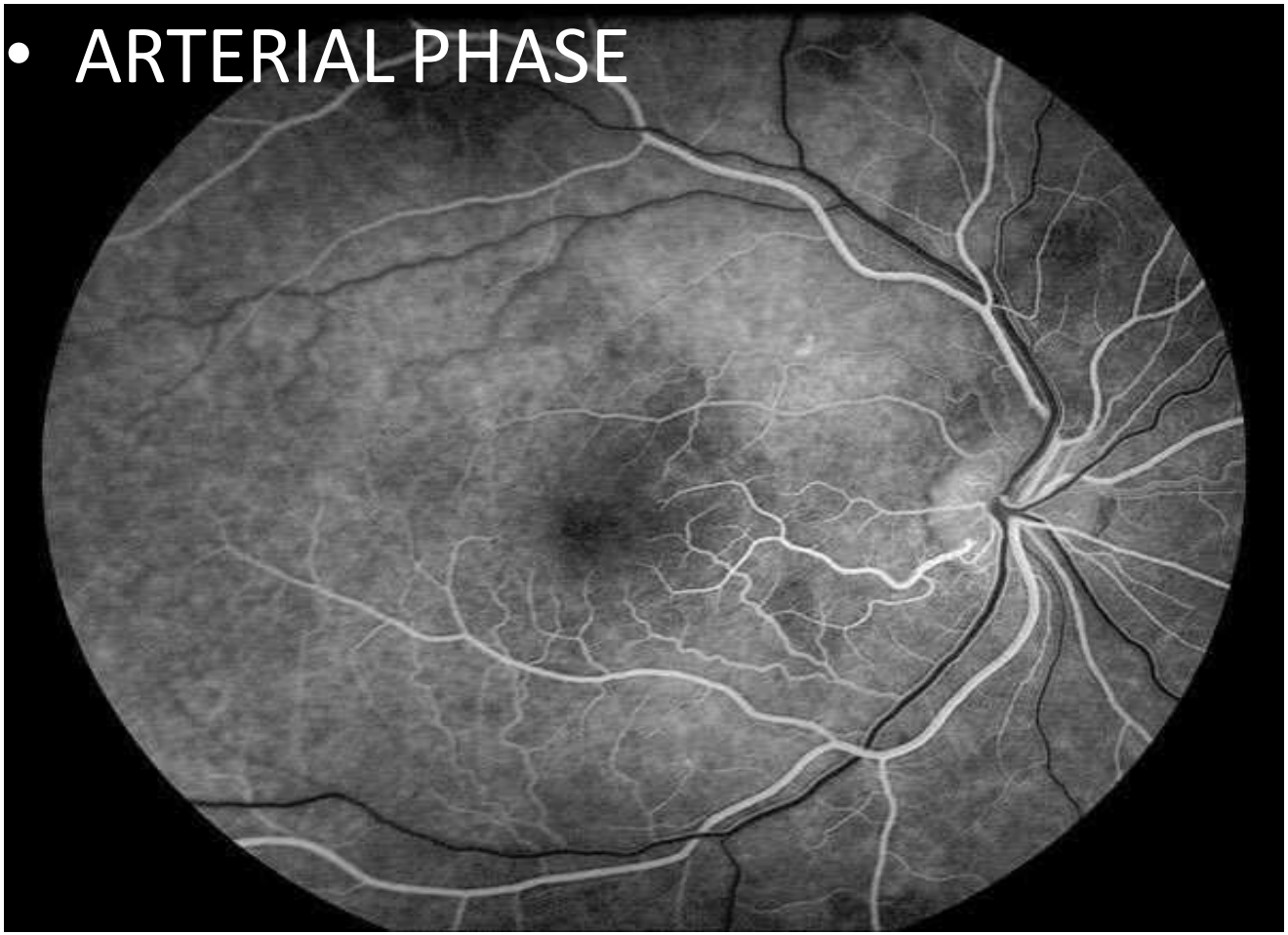
- No dye reaches retinal arteries

- Cilioretinal artery if present fills in this phase

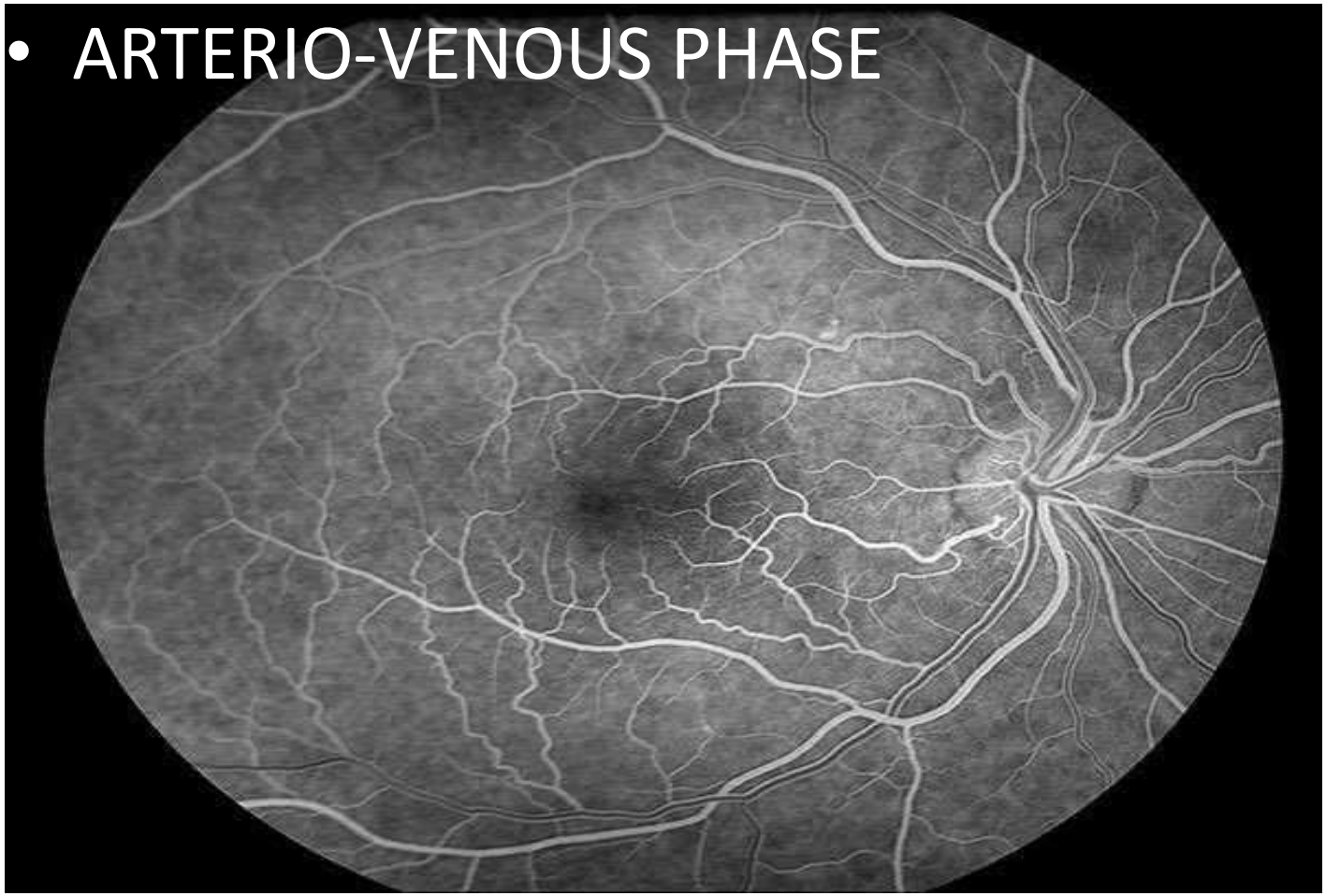
CHORIDAL PHASE



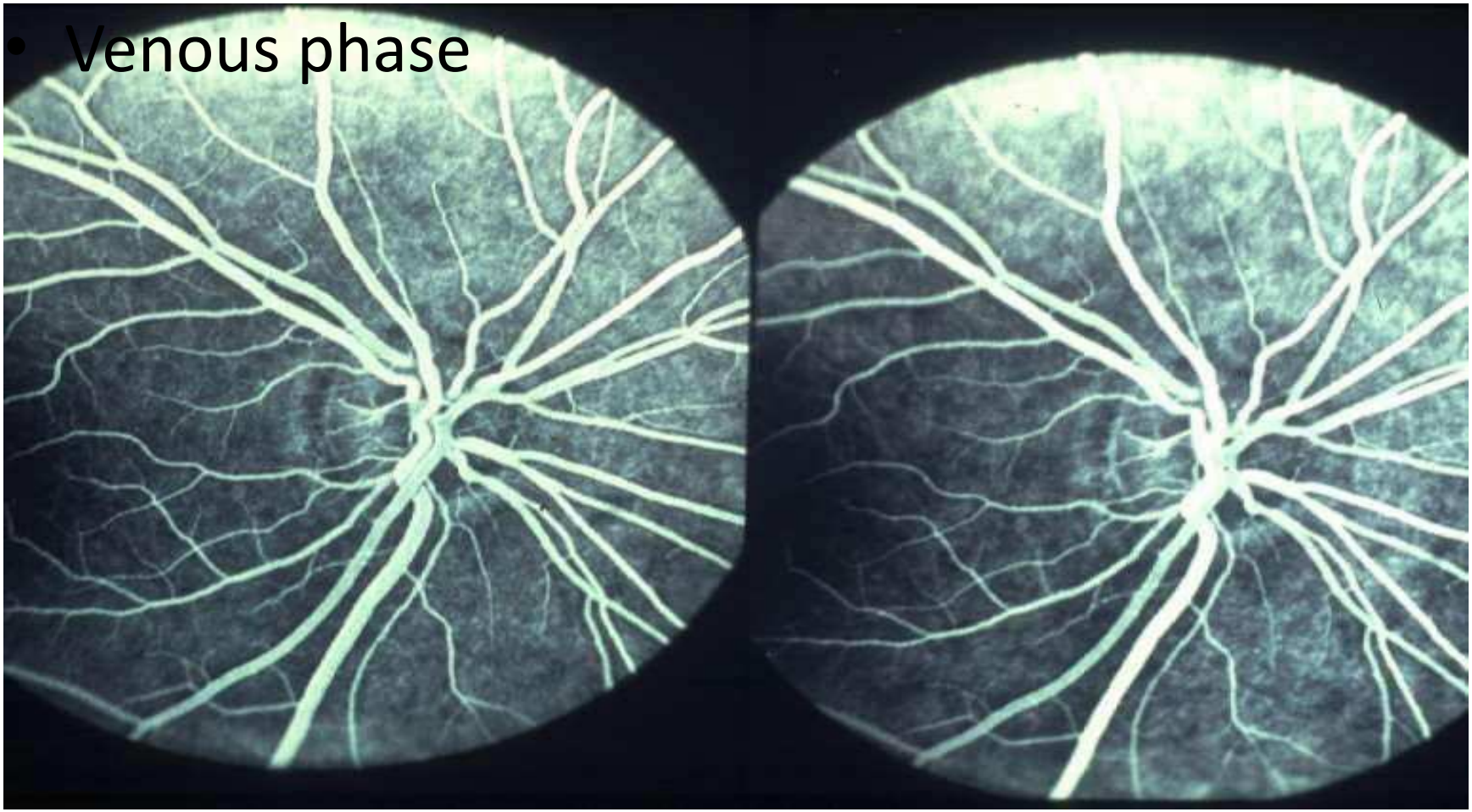
- ARTERIAL PHASE



- ARTERIO-VENOUS PHASE



- Venous phase



Outer blood–retinal barrier

- The major choroidal vessels are impermeable to both bound and free fluorescein.

- However, the walls of the choriocapillaris contain fenestrations through which unbound molecules escape into the extravascular space, crossing Bruch membrane but on reaching the RPE are blocked by intercellular complexes termed tight junctions or zonula occludentes

Inner blood–retinal barrier

- composed principally of the tight junctions between retinal capillary endothelial cells, across which neither bound nor free fluorescein can pass;
- the basement membrane and pericytes play only a minor role in this regard.
- Disruption of the inner blood–retinal barrier permits leakage of both bound and free fluorescein into the extravascular space

Features of FFA

1. Hyperflourescence----an area of abnormally high fluorescence due to increase density of dye molecule
2. Hypoflourescence -----an area of abnormally poor fluorescence

Causes of Hyperflouresence

- ✓Window defect
- ✓Pooling of dye
- ✓Leakage of dye
- ✓Staining of dye

WINDOW DEFECT

- Caused by atrophy or absence of the RPE
- e.g
  - AMD
  - Full thickness macular hole
  - RPE tears
  - Drusen

POOLING

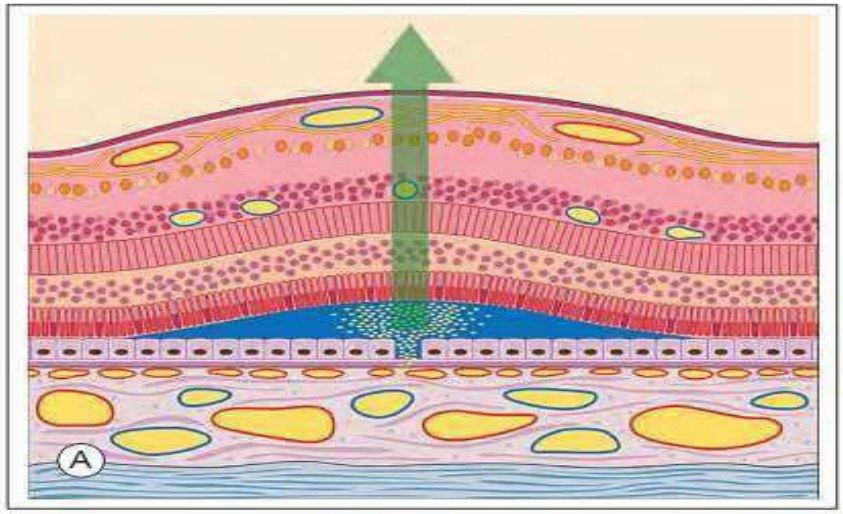
- Accumulation of dye in closed space

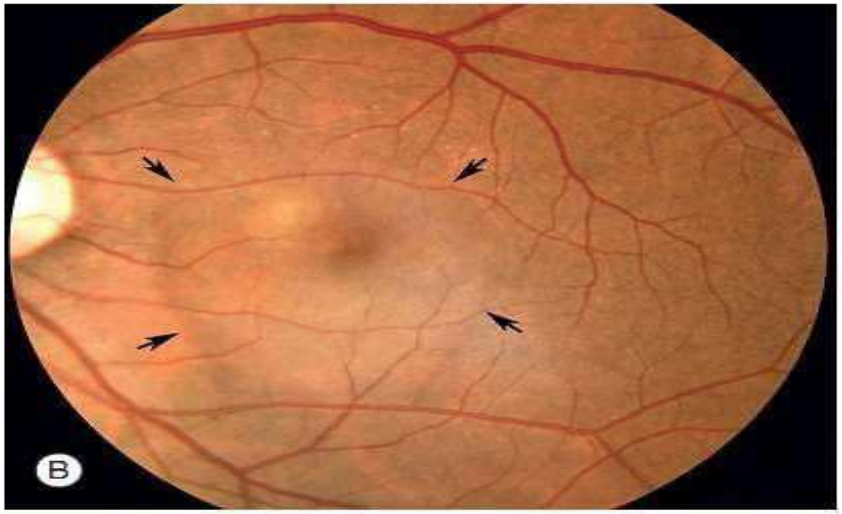
- e.g.

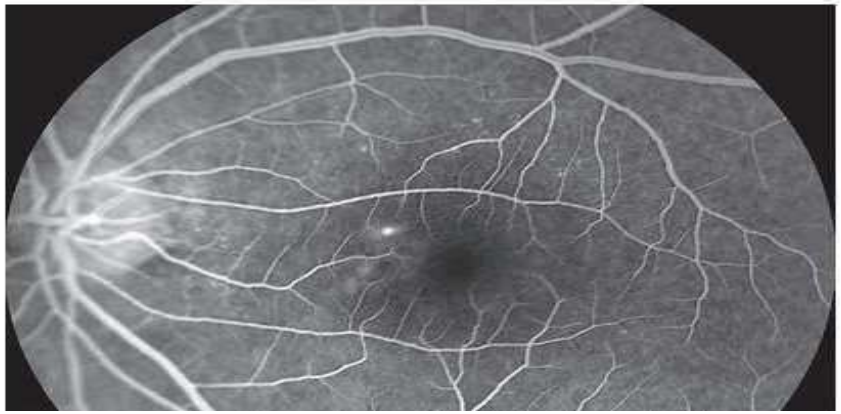
  RPE detachment,

  CSR

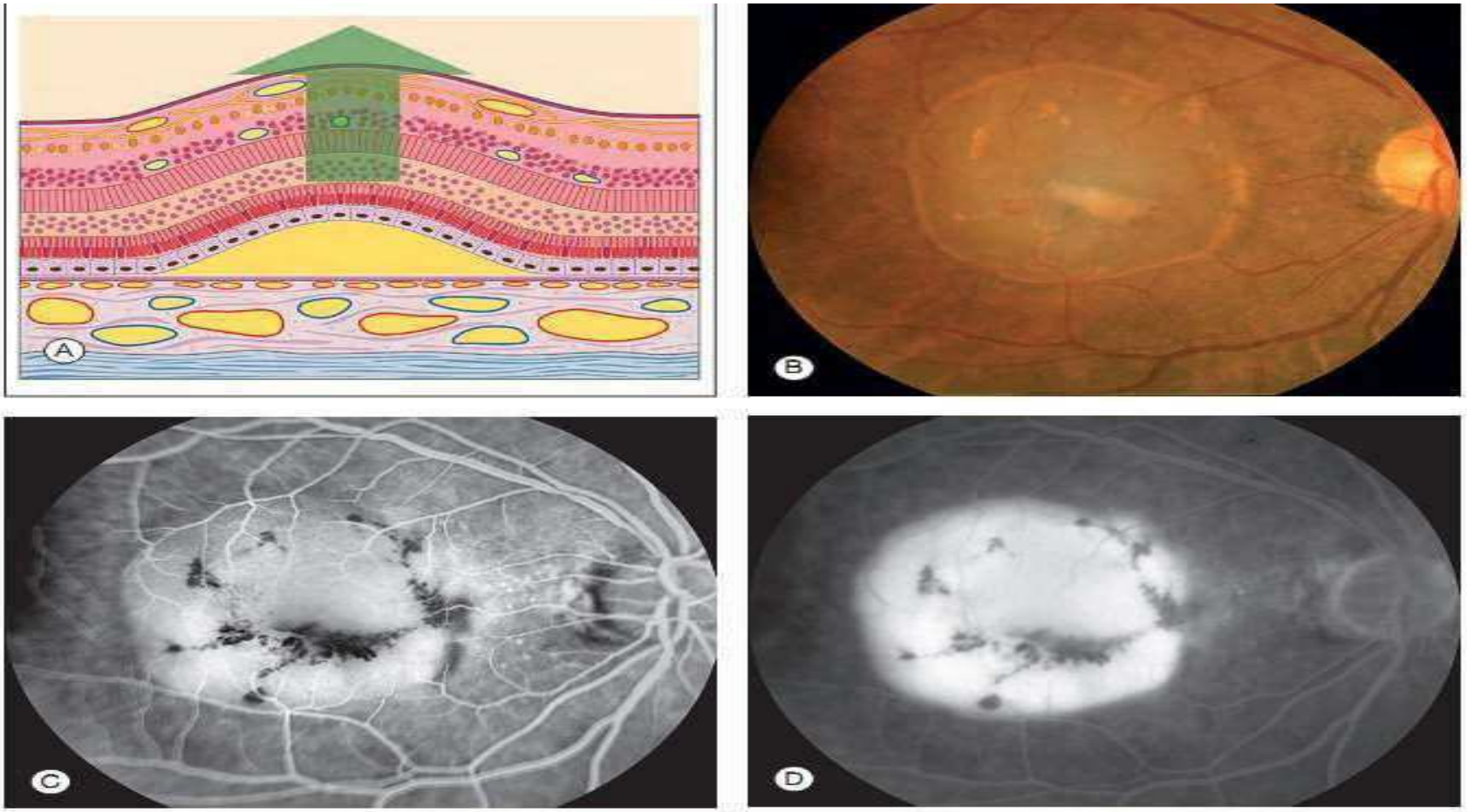
CENTRAL SEROUS CHORIORETINOPATHY(CSR)







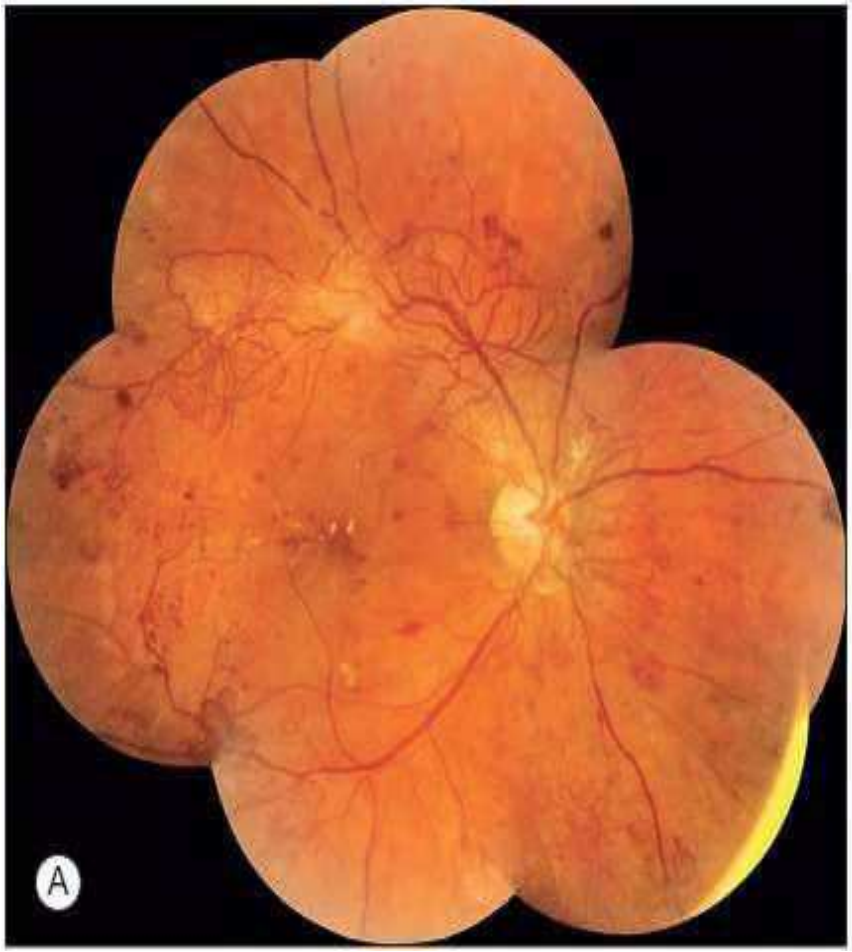
PED

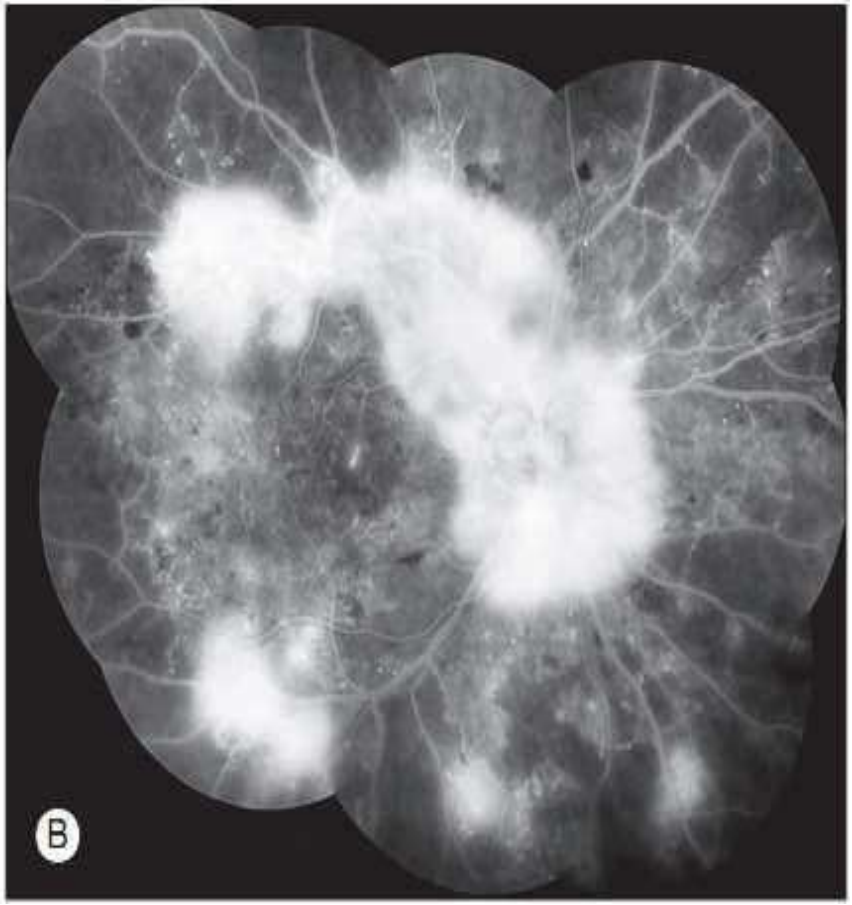


Leakage

- Leakage of dye is characterized by fairly early hyperfluorescence, increasing with time in both area and intensity.
- It occurs as a result of breakdown of the inner blood–retinal barrier due to:
  - Dysfunction or loss of existing vascular endothelial tight junctions as in
    - background diabetic retinopathy (DR),
    - retinal vein occlusion (RVO),
    - cystoid macular oedema and
    - papilloedema.
  - Primary absence of vascular endothelial tight junctions as in
    - CNV,
    - proliferative diabetic retinopathy ,
    - tumours and
    - some vascular anomalies such as Coats disease.

Neovascularization

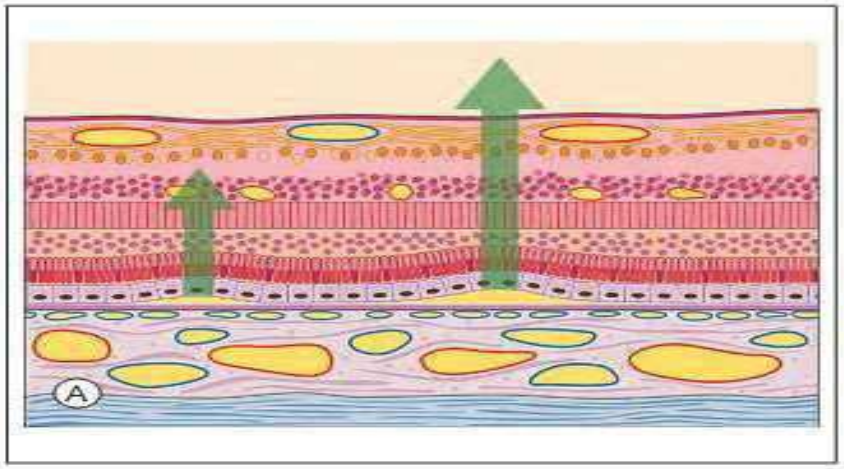


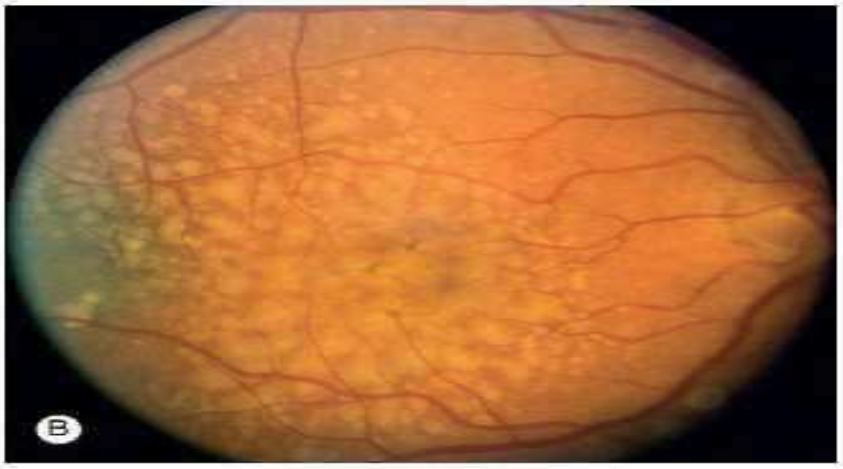


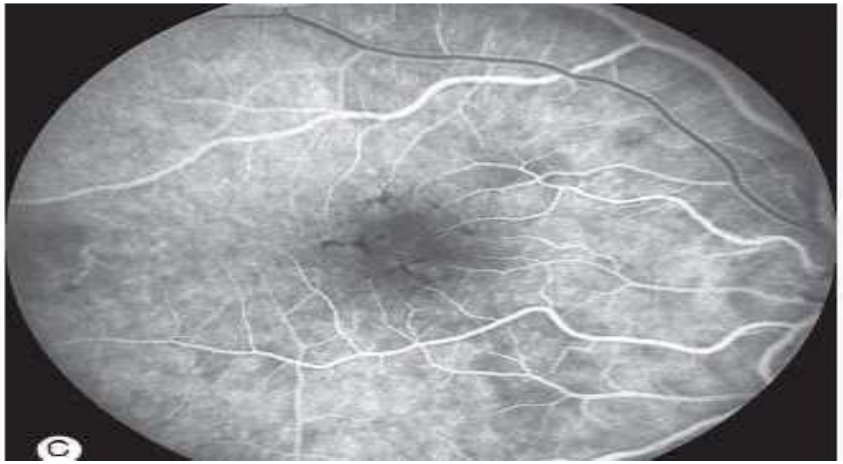
STAINING

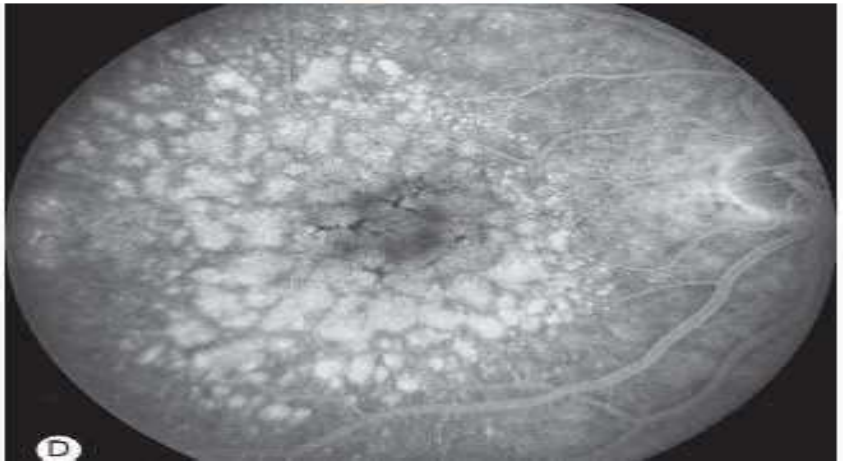
- It is a late phenomenon consisting of the prolonged retention of dye in entities such as
  - drusen
  - fibrous tissue
  - exposed sclera and
  - the normal optic disc
- it is seen in the later phases of the angiogram, particularly after the dye has left the choroidal and retinal circulations.

STAINING





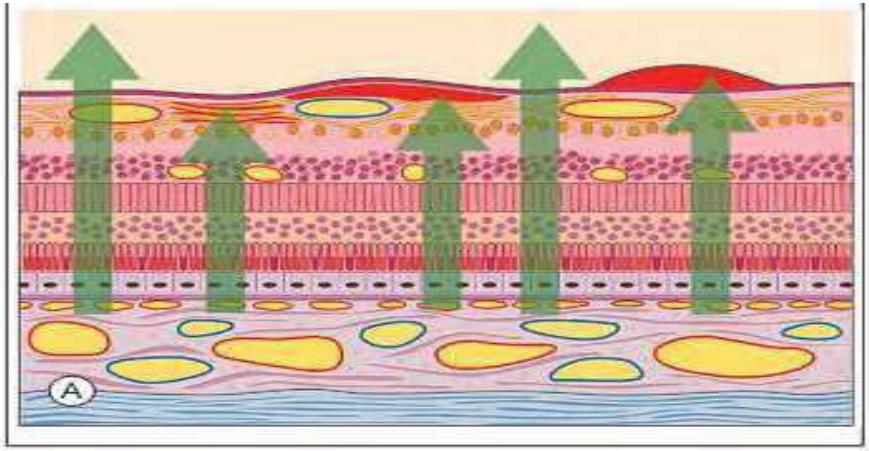


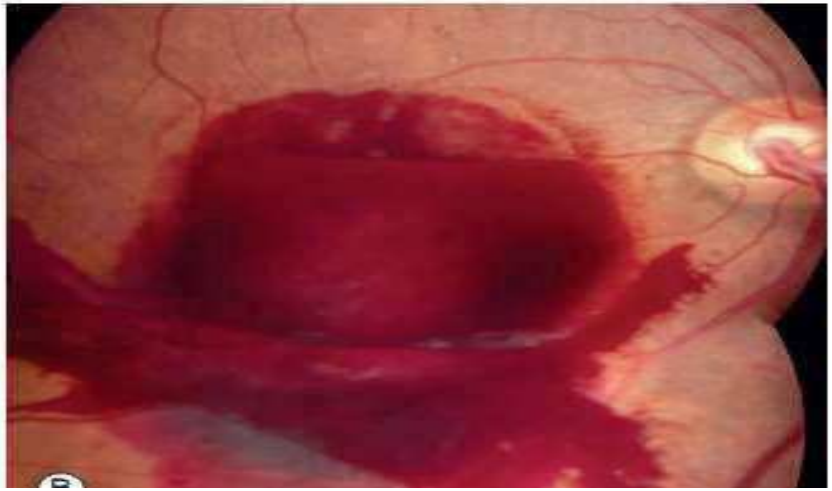


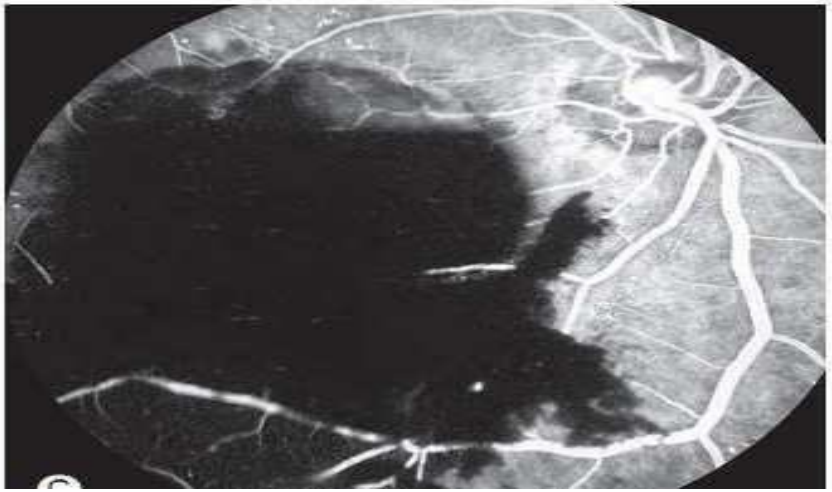
CAUSES OF HYPOFLUORESENCE

- BLOCKAGE
- VASCULAR FILLING DEFECT

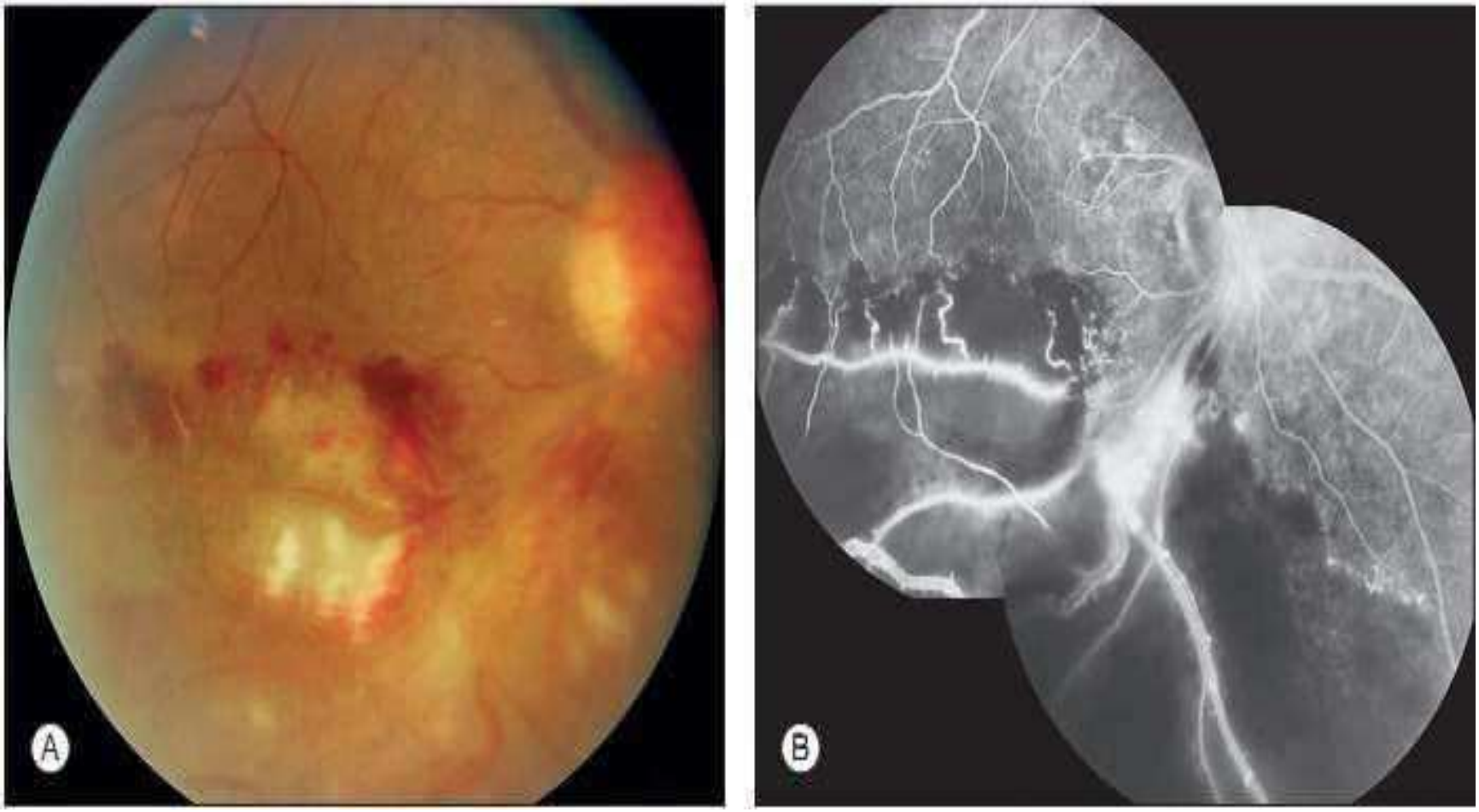
BLOCKAGE







VASCULAR FILLING DEFECT (BRVO)



USES OF FFA

- Evaluation of vascular integrity of retinal & choroidal vessels
- Disease process affecting macula
- Integrity of the Blood Ocular Barrier.
  - outer blood retinal barrier breaks in :- CSR
  - inner blood retinal barrier breaks in:- NVD ,NVE

- Determining the extent of damage
- Formulating the treatment strategy for retinal & choroidal disease.
- Monitoring the result of treatment.

COMPLICATIONS

MILD	MODERATE	SEVERE
Staining of skin, sclera and mucous membrane	Nausea ,vomiting	laryngeal edema bronchospasm
Stained secretion Tear, saliva	Vasovagal response	Circulatory shock, MI, cardiac arrest
Orange-yellow urine	urticaria	Generalized convulsion
Skin flushing, tingling lips, pruritus	fainting	Skin necrosis
	periphlebitis	

Table 6-2

List of Must-Have Pharmacologic Agents in an FA Facility

Adrenaline/epinephrine (1/10000) 1 mg/10ml *Preferably preloaded syringes (AnaKit/EpiPen)*
Atropine (1 mg/10 ml) (Atropair, Atropisol)
Atenolol (5 mg/10 ml) 0,4% IV **Lidocaine**
Diazepam (oral 5 to 10 mg, and 10mg/2ml ampoules)
Verapamil (5 mg/2 ml) or 50 mg/10ml IV **Urapidil** (Elgadil, in Europe)
Hydrocortisone hemisuccinate (100 mg/ml) or IV **Methylprednisolone** (20, 40, and 125 mg)
Methylxanthines (eg, **Aminophylline** 200mg/10 ml ampoules) **Salbutamol** (500 microg/1 ml ampoules and spray)
5mg/1ml IV **Dexchlorpheniramine** (Polaramine)
Nitroglycerin (1 mg sublingual, spray, and transdermal discs)
Promethazine hydrochloride (Phenergan) or 10 mg/2 ml **Metoclopramide** (IV and oral solution) in Europe.
Oral **glucose** (eg, oral gel, Glutose 15) Glucagon (Glucagen in Europe, acts faster than oral glucose in case of severe hypoglycemia if sufficient glucose in liver exists)
IV **Sodium bicarbonate** (1/6 M)
IV **Normal saline, Glucose saline** and **Ringer Lactate** (50, 100, and 500 ml, preferably in plastic bottles) 500 mL **Haemocel**

Table 6-3

Typical Contents of a Standard First Aid Kit in an FA Facility

- Examination gloves (small, medium, and large): sterile and nonsterile
- Stainless steel basins
- Emesis bags
- Cold/hot packs (in the refrigerator)
- 4 x 4 gauze packs
- Small bandages
- Scissors
- Gauze pads
- Adhesive tapes
- Skin cleanser (Alcohol, Betadine)
- Syringes (insulin, 2, 5, and 10 ml) and needles (different sizes)
- Tourniquets (Smark)

- Stethoscope
- Sphygmomanometer with several size cups
- Thermometer (fast reading, digital)
- Glucometer (eg, Accu-Trend)
- Automated external defibrillator

- Ambu bag
- Nasal cannulas
- Mayo cannulas (different sizes)
- Emergency tracheostomy cannula (kit)
- Pocket face mask (with one way valvula)
- O_2 portable cylinder with a low flow regulator

Contraindications of FFA

ABSOLUTE

1) known allergy
2) H/O adverse reaction in past

RELATIVE

- Asthma
- Hay fever
- Renal failure
- Hepatic failure
- Pregnancy (especially 1st trimester

Optics & Eye

Dr samina karim

AP Ophthalmology

KGMC, HMC

objectives

- Discuss visual functions(visual acuity, color vision, contrast sensitivity, light brightness).

- Discuss refraction, aphakia, pseudophakia and anisometropia.

Optics of the eye

- Light rays enter the eye through the clear cornea, pupil and lens.

- These light rays are focused directly onto the retina, the light-sensitive tissue lining the back of the eye.

- The retina converts light rays into impulses, sent through the optic nerve to the brain, where they are recognized as images.

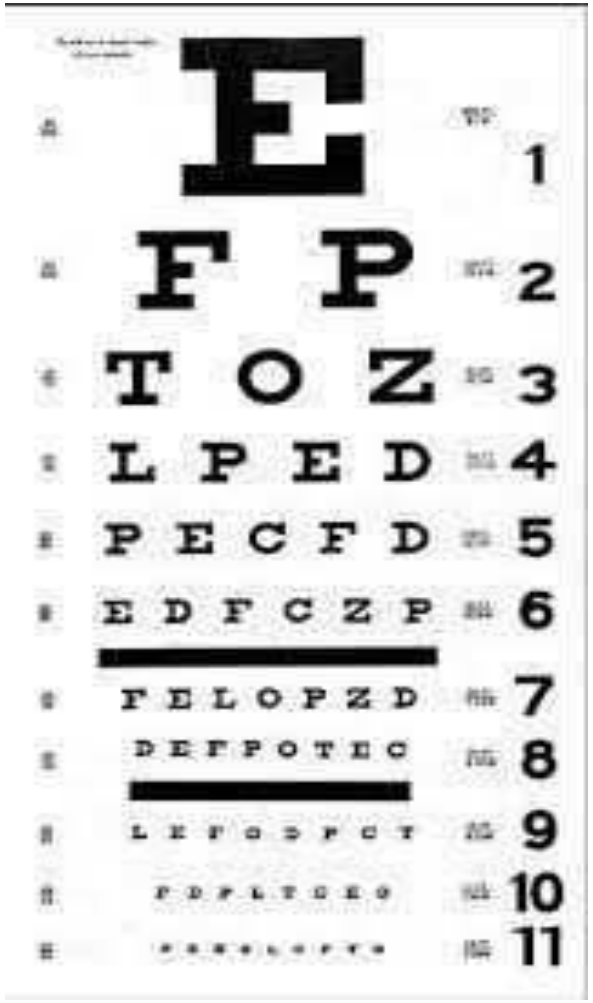
- 70% of the eye's focusing power comes from the cornea and 30% from the lens.

Visual acuity

- Visual acuity (VA) is **a measure of the ability of the eye to distinguish shapes and the details of objects at a given distance**.
- It is important to assess VA in a consistent way in order to detect any changes in vision.
- One eye is tested at a time.

Test for Visual acuity

- **Snellen visual acuity**
  - Distance visual acuity (VA) is directly related to the minimum angle of separation (subtended at the nodal point of the eye) between two objects that allow them to be perceived as distinct.
  - In practice, it is most commonly carried out using a Snellen chart, which utilizes black letters or symbols (optotypes) of a range of sizes set on a white chart with the subject reading the chart from a standard distance.
  - Distance VA is usually first measured using a patient's refractive correction, generally their own glasses or contact lenses.


E
1
F P
2
T O Z
3
L P E D
4
P E C F D
5
E D F C Z P
6
F E L O P Z D
7
D E F P O T E C
8
9
10
11

- For completeness, an unaided acuity may also be recorded.
- The eye reported as having worse vision should be tested first, with the other eye occluded.
- It is important to push the patient to read every letter possible on the optotypes being tested.

- Normal monocular VA
  - equal to 6/6 (metric notation; 20/20 in non-metric ‘English’ notation) on Snellen testing.
  - Normal corrected VA in young adults is often superior to 6/6.
- Best-corrected VA (BCVA)
  - denotes the level achieved with optimal refractive correction.

- Pinhole VA:
  - a pinhole (PH) aperture compensates for the effect of refractive errors, and consists of an opaque Occluder perforated by one or more holes of about 1 mm diameter.
  - However, PH acuity in patients with macular disease and posterior lens opacities may be worse than with spectacle correction.
  - If the VA is less than 6/6 Snellen equivalent, testing is repeated using a pinhole aperture.
- Binocular VA
  - usually superior to the better monocular VA of each eye, at least where both eyes have roughly equal vision.

Very poor visual acuity

- Counts (or counting) fingers (CF)
- Hand movements (HM
- Perception of light (PL)

Log MAR acuity

- Log MAR charts address many of the deficiencies of the Snellen chart and are the standard means of VA measurement in research and increasingly in clinical practice.

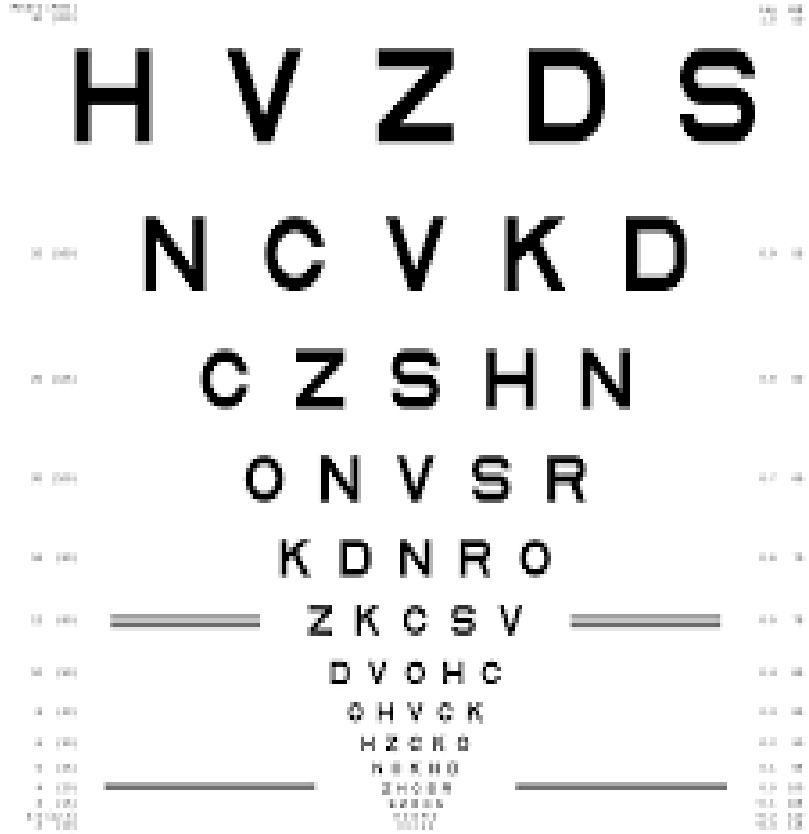
- Log MAR is an acronym for the base-10 logarithm of the minimum angle of resolution, and refers to the ability to resolve the elements of an opto type.

LogMAR charts

- The Bailey–Lovie chart.
  - ✓ Used at 6 m testing distance.
  - ✓ Each line of the chart comprises five letters and the spacing between each letter and each row is related to the width and the height of the letters. A 6/6 letter is 5′ in height by 4′ in width. The distance between two adjacent letters on the same row is equal to the width of a letter from the same row, and the distance between two adjacent rows is the same as the height of a letter from the lower of the two rows.
  - ✓ Snellen VA values and logMAR VA are listed to the right and left of the rows respectively.

- Other charts are available that are calibrated for 4 m. The Early Treatment Diabetic Retinopathy Study (ETDRS) charts utilize balanced rows comprising Sloan optotypes,.

- Computer charts are available that present the various forms of test chart on display screens, including other means of assessment such as contrast sensitivity


H V Z D S
N C V K D
C Z S H N
O N V S R
K D N R O
Z K C S V
D V O H C

Comparison of Snellen and logMAR visual acuity testing

Snellen

- Shorter test time
- More letters on the lower lines introduces an unbalanced 'crowding' effect
- Fewer larger letters reduces accuracy at lower levels of VA
- Variable readability between individual letters
- Lines not balanced with each other for consistency of readability

Log MAR

- Longer test time
- Equal numbers of letters on different lines controls for 'crowding' effect
- Equal numbers of letters on low and higher acuity lines increases accuracy at lower VA
- Similar readability between letters
- Lines balanced for consistency of readability

- 6 m testing distance: longer testing lane (or a mirror) required
- Letter and row spacing not systematic
- Lower accuracy and consistency so relatively unsuitable for research
- Straightforward scoring system
- Easy to use

- 4 m testing distance on many charts: smaller testing lane (or no mirror) required
- Letter and row spacing set to optimize contour interaction
- Higher accuracy and consistency so appropriate for research
- More complex scoring
- Less user-friendly

color vision

- Colour vision is the ability of the eye to discriminate between colours excited by lights of different wavelengths.
- Colour vision is a function of cone .
- Better appreciated in photopic condition

THEORIES OF COLOUR VISION

- TRICHROMATIC THEORY:

- Also called as young - helmholtz theory Thomas Young Helmholtz

- It postulates the existence of three kinds of cones

- Each cone containing a different photopigment and maximally sensitive to one of three primary colours i.e. Red, Green and Blue.

- Human eye can see any colour due to a combination of red, green and blue monochromatic light in different proportions.

- Humans are considered trichromats –

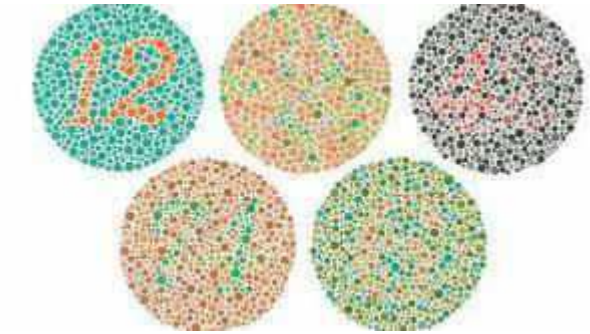
  - Blue, Red, and Green Cone Photoreceptors
  - Rod photoreceptors – are important for vision in dim light

- Colour vision depends on three populations of retinal cones, each with a specific peak sensitivity;
  - blue (tritan) at 414–424 nm,
  - green (deuteran) at 522–539 nm
  - red (protan) at 549–5

- Trichromats possess all three types of cones (although not necessarily functioning perfectly), while absence of one or two types of cones renders an individual a dichromat or monochromat, respectively.

Colour vision tests

- The Ishihara test
- The Hardy–Rand–Rittler test
- The City University test
- The Farnsworth–Munsell 100-hue test



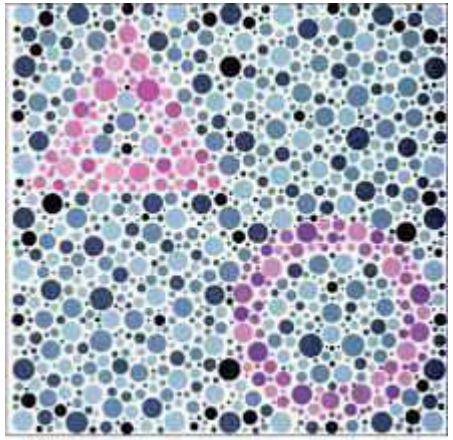


FIGURE 1: A plate from bandy-randolph pseudoisochromatic test

contrast sensitivity

- Contrast sensitivity is a measure of the ability of the visual system to distinguish an object against its background.

- A target must be sufficiently large to be seen, but must also be of high enough contrast with its background; a light grey letter will be less well seen against a white background than a black letter.

- Contrast sensitivity represents a different aspect of visual function to that tested by the spatial resolution tests such as visual acuity, which use high-contrast optotypes.

- Many conditions reduce both contrast sensitivity and visual acuity, (e.g. amblyopia, optic neuropathy, some cataracts, and higher-order aberrations),
- but under some circumstances visual function measured by contrast sensitivity can be reduced whilst VA is preserved.

- Hence, if patients with good VA complain of visual symptoms (typically evident in low illumination), contrast sensitivity testing may be a useful way of objectively demonstrating a functional deficit.

- Despite its advantages, it has not been widely adopted in clinical practice

Test for contrast sensitivity

- The Pelli–Robson contrast sensitivity letter chart
  - It is viewed at 1 metre and consists of rows of letters of equal size (spatial frequency of 1 cycle per degree) but with decreasing contrast of 0.15 log units for groups of three letters.
  - The patient reads down the rows of letters until the lowest resolvable group of three is reached.
- Sinusoidal (sine wave) gratings
  - require the test subject to view a sequence of increasingly lower contrast gratings

Pelli–Robson contrast sensitivity letter chart

light brightness

- Brightness is **an attribute of visual perception in which a source appears to be radiating or reflecting light.**

Refraction

- A refraction is **an eye exam that measures a person's prescription for eyeglasses or contact lenses**.

- Normal vision occurs when light is focused directly on the retina rather than in front or behind it.

Aphakia

- Absence of crystalline lens.

- Lens is absent from the pupillary line and does not take part in refraction.

CAUSES

- Congenital absence of lens.
- Surgical aphakia.
- Aphakia due to absorption of lens matter.
- Traumatic extrusion of lens.
- Posterior dislocation of lens.

Sign and symptoms of aphakia

- Blurred vision.
- Problems seeing things that are close and far away.
- Problems seeing the brightness of colours. Colours may seem to be faded. ...
- An iris that jiggles. This is called iridodonesis.
- Problems adjusting to differences in how far away or close something.

Treatment of aphakia

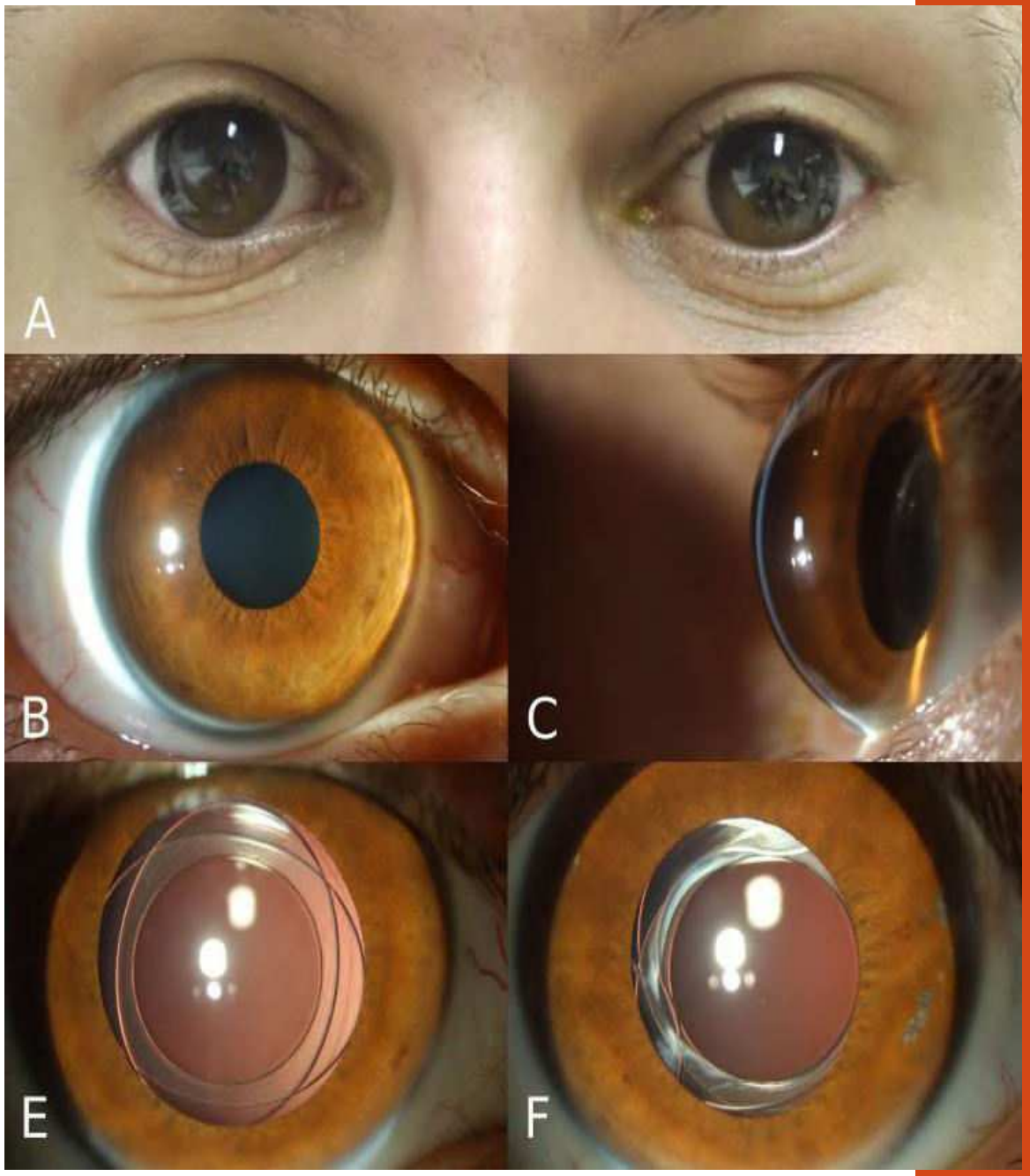
- infants with unilateral aphakia are treated with contact lenses for the first few years of life, after which an IOL can be implanted as a secondary procedure.

- Aphakic glasses are also an option for replacing focusing power, but are very thick, causing distortion.

Pseudophakia

- Pseudophakia is a Latin word for **false lens**.
- This term use after placing an artificial lens into the eye. Also known as intraocular IOL, lens implants, or “fake eye lenses,”
- This procedure can significantly improve vision after removing cataracts and replacing them with a new lens

- Pseudophakic lenses can be made of
  - plastic composites,
  - silicone,
  - acrylic material.
- **Types of lenses**
  - Monofocal IOLs:
  - Multifocal IOLs:
  - Accommodative IOLs
  - Toric IOLs:



Anisometropia

- Anisometropia means that **vision in one eye is worse than the vision in the other due to a difference in refractive error.**

types of anisometropia

- There are six clinical types of anisometropia:
  - **Simple;**

    One eye sees normally, while the other is myopic or hypermetropic
  - **Compound;**

    Compound anisometropia: Both eyes are myopic or hypermetropic (also called ametropic
  - **Mixed;**

    One eye is myopic; the other is hypermetropic.
  - **simple astigmatic**
  - **compound astigmatic**
  - **mixed astigmatic.**

Symptoms of anisometropia

- Double vision (dipoplia).
- Blurred vision.
- Headaches.
- Poor depth perception.
- Dizziness.
- Eye pain or discomfort.

- Anisometropia is treated by correcting eyesight through glasses, contact lenses or surgery.

- If a child developed amblyopia (one eye is weaker than the other), the treatments may include forcing the brain to use the weaker eye by:
  - Patching the stronger eye.
  - Using eye drops that blur vision in the stronger eye.
  - Using filtered glasses that block vision in the stronger eye.

Thank you

REFRACTIVE ERRORS

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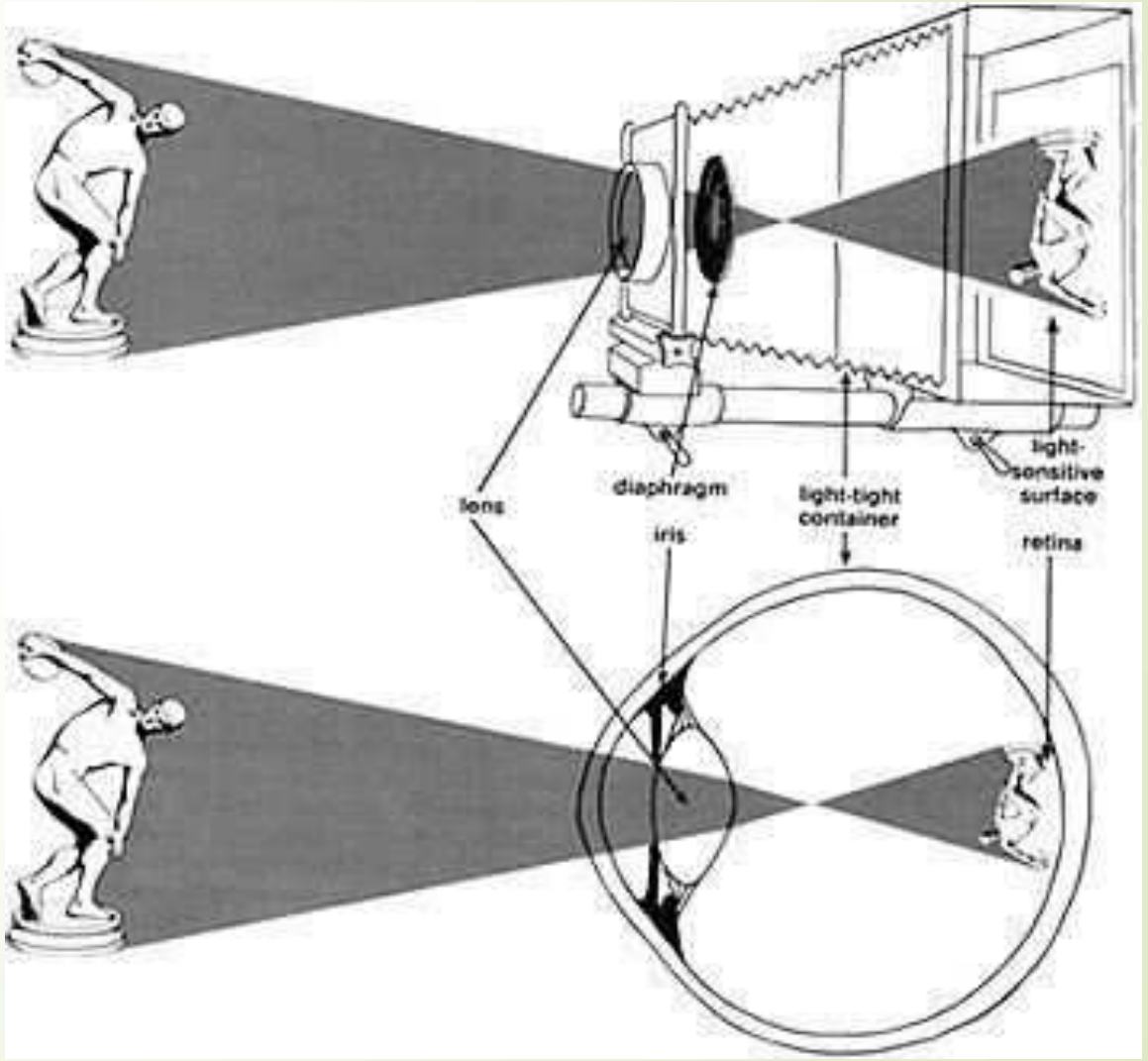
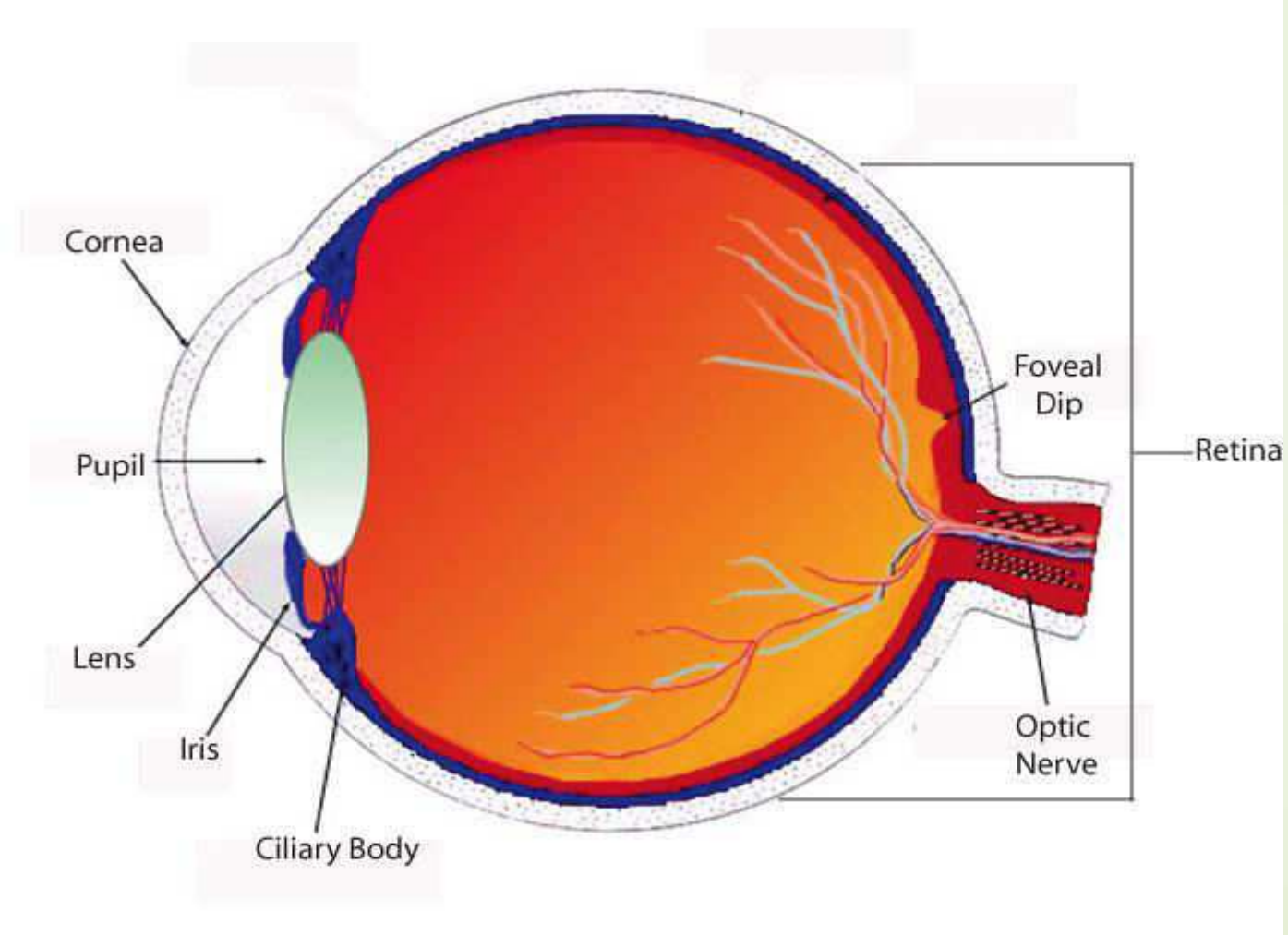
Objectives:

- Define and enumerate treatment options of
  - Emmetropia
  - Myopia
  - Hypermetropia
  - Astigmatism
  - Presbyopia

- **Eyelids**- shutter
- **Cornea**- focusing system
- **Lens**- focusing system
- **Iris**- diaphragm
- **Choroid**- dark chamber
- **Retina**-light sensitive film

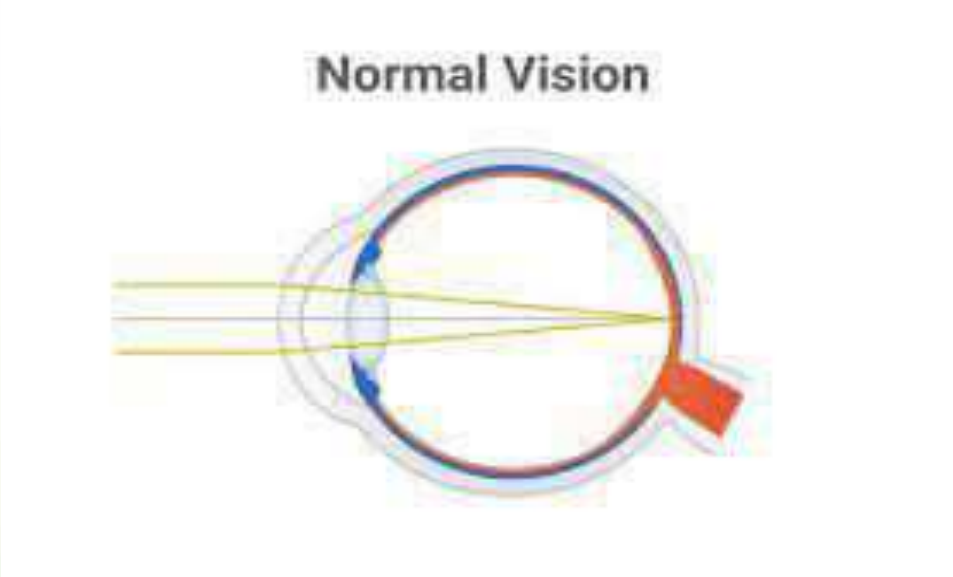



Cornea
Pupil
Lens
Iris
Ciliary Body
Foveal Dip
Retina
Optic Nerve

- Emmetropia
- Ametropia
  - Myopia
  - Hypermetropia
  - Astigmatism
  - Presbyopia

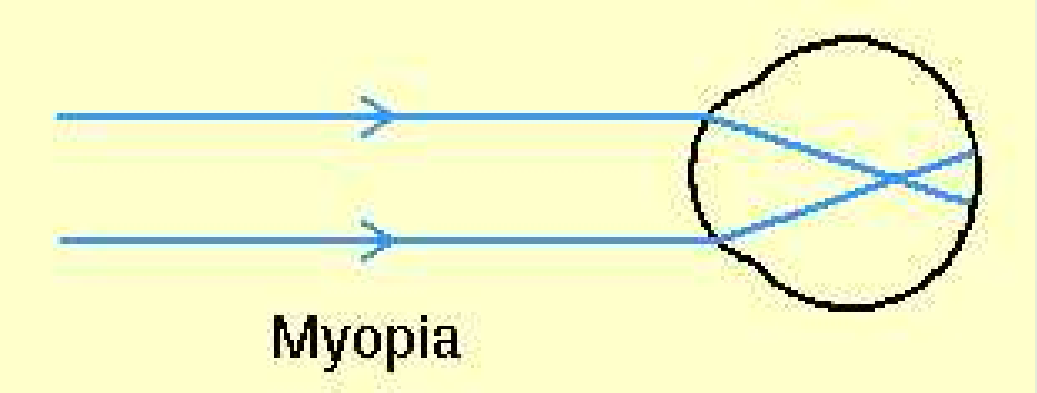
Emmetropia

- Parallel rays of light focus on retina with accommodation at rest



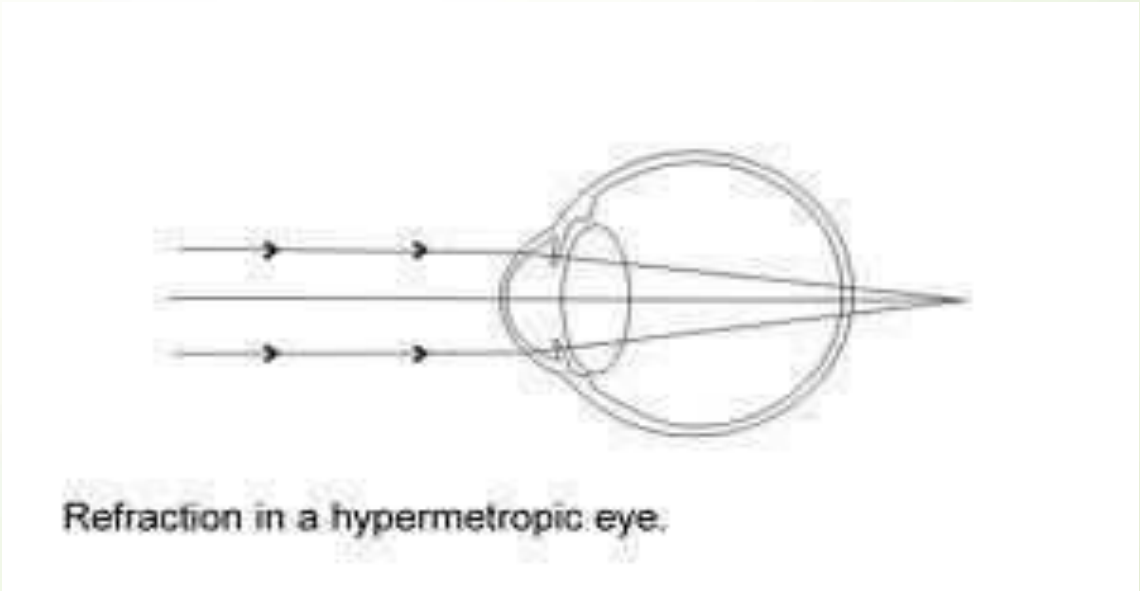
Myopia

- A form of refractive error in which parallel rays of light entering the eye are focused in front of retina with accommodation being at rest.



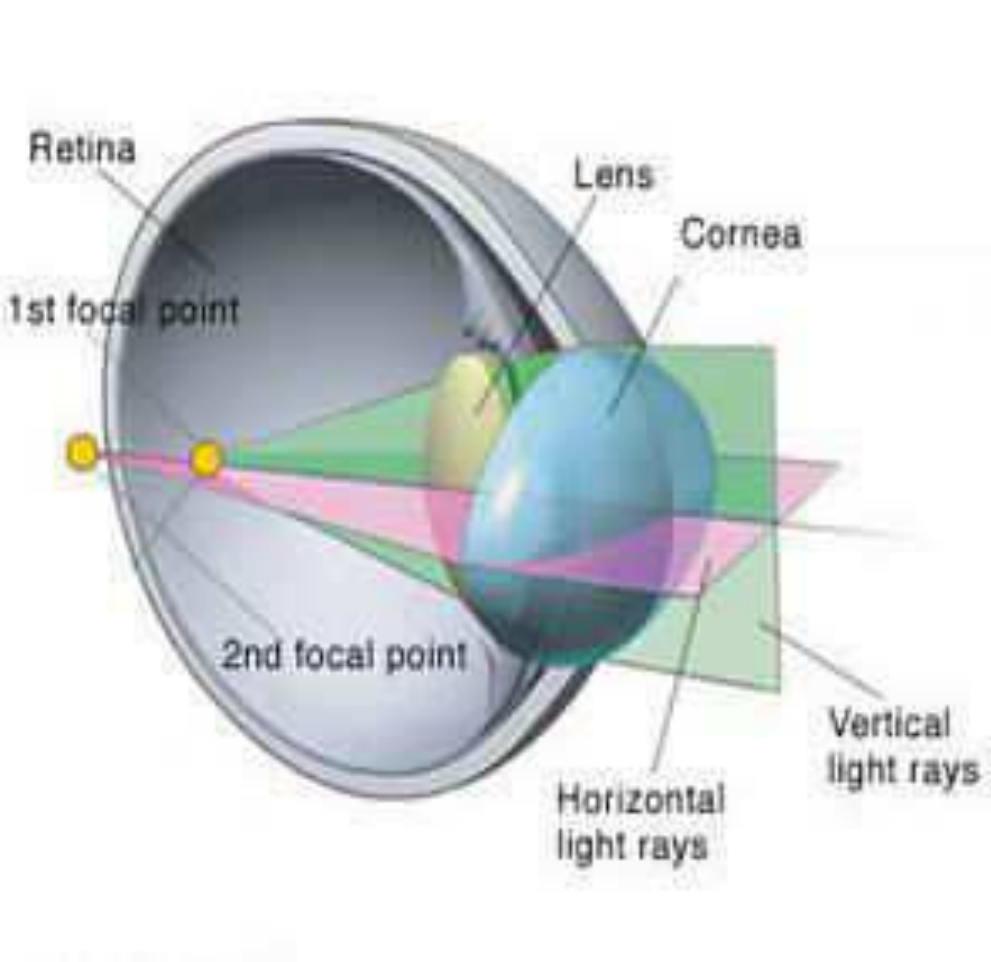
Hypermetropia

- Parallel rays of light coming from infinity are focused behind the retina with accommodation at rest



Refraction in a hypermetropic eye.

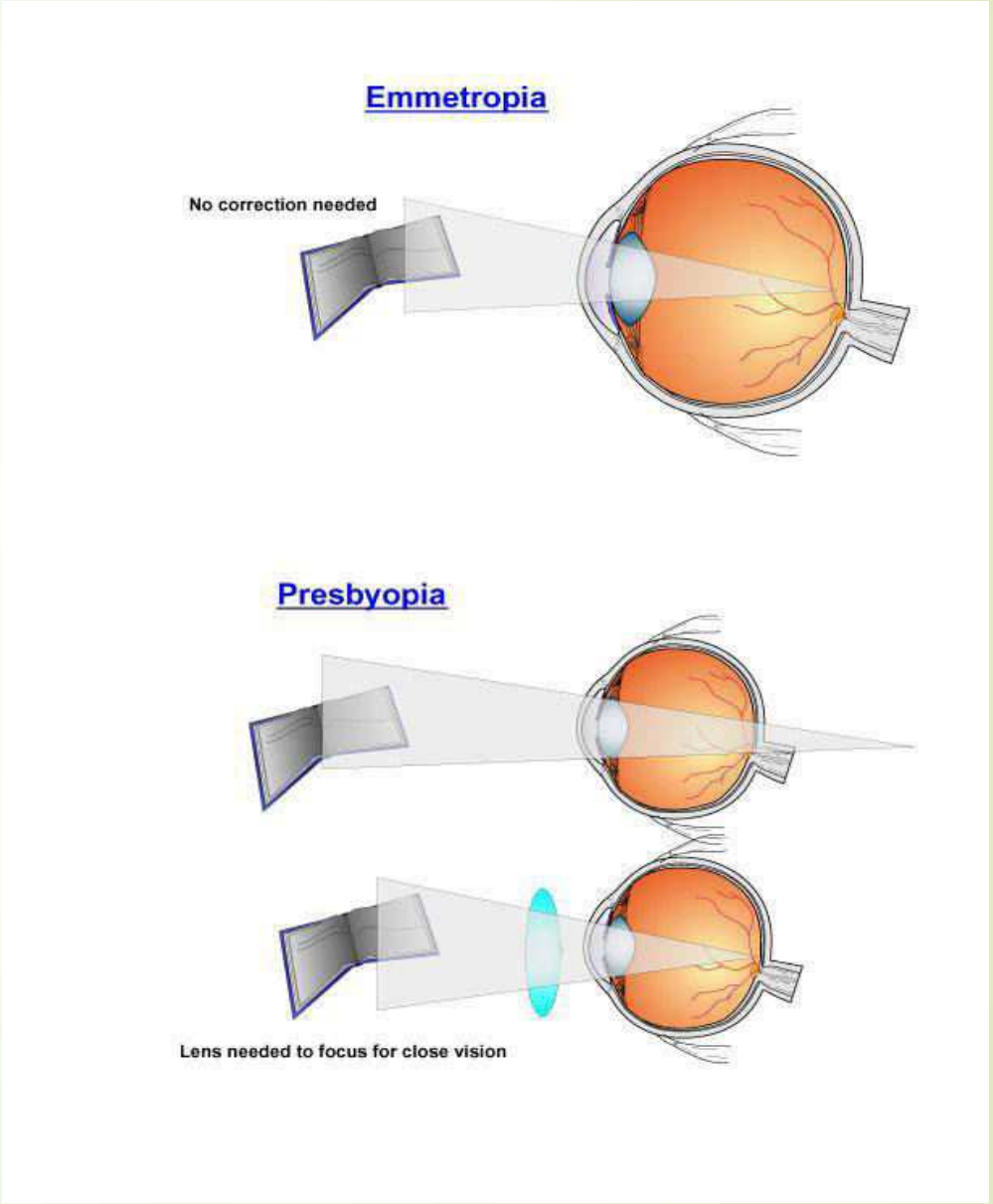
ASTIGMATISM



- A-Stigma = Not a point
- A defect of an optical system causing light rays from a point source to fail to meet in a focal point resulting in a blurred and imperfect image

PRESBYOPIA

- The physiologic loss of accommodation in the eyes with advancing age



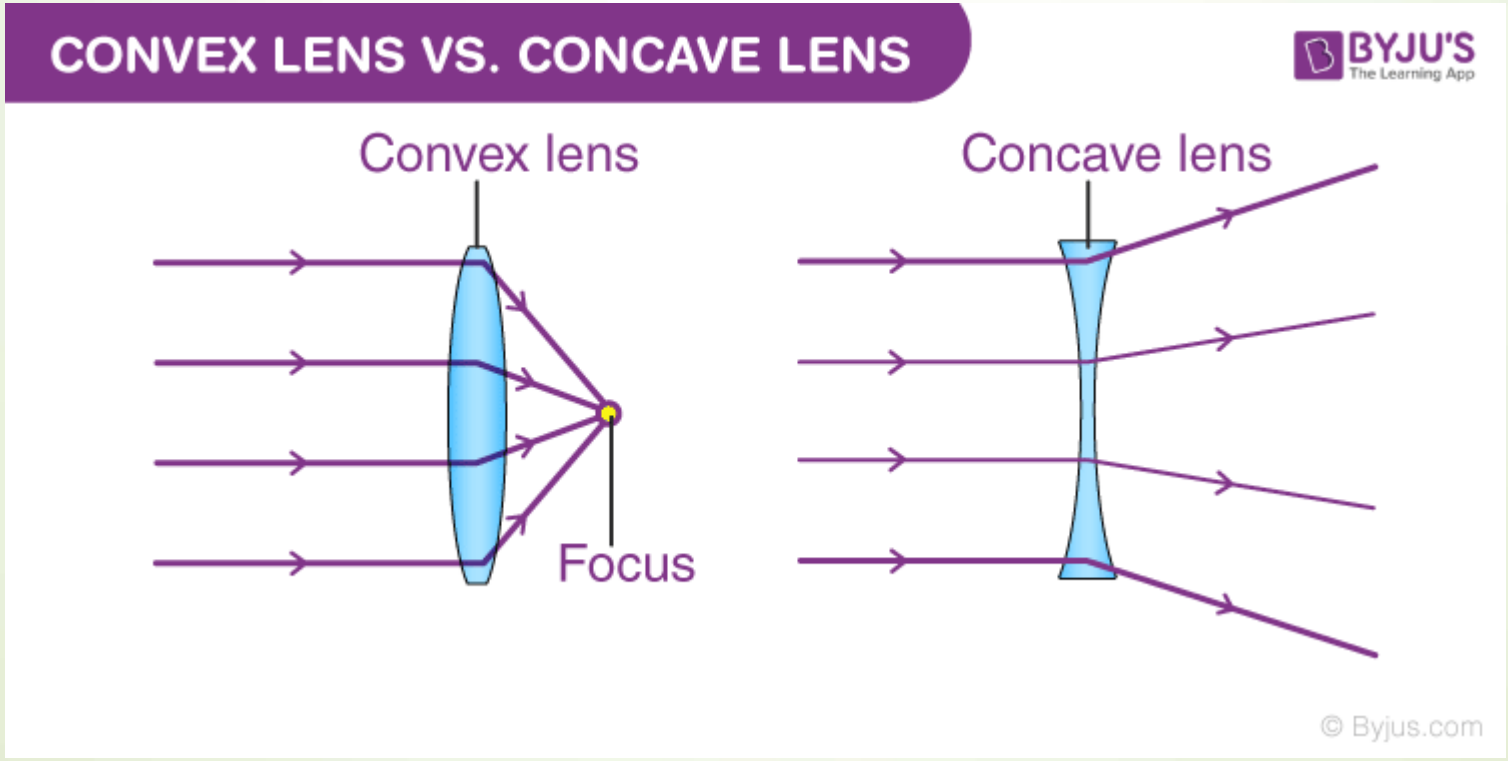
Treatment

GLASSES

- *Myopia*
  - Concave = - (minus) lenses

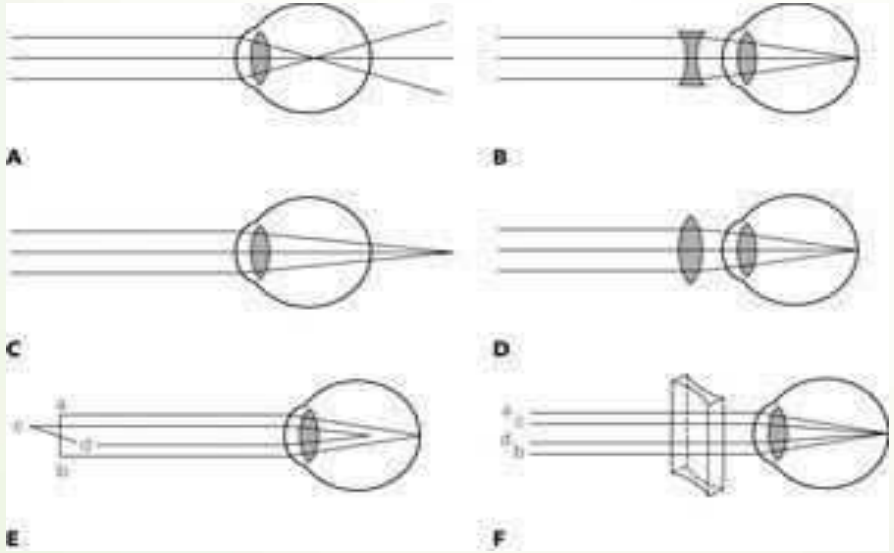
Hypermetropia

- Convex = + (plus)



ASTIGMATISM

- Cylindrical lenses



Contact lenses

why opt for refractive surgery

Previously:

- Profession
- Sport
- Spec/CL intolerance

Now:

- Cosmesis
- Frustration with use of spec
- Improved Unaided VA

- Keratorefractive procedures
- Lens based refractive procedures
- Combined procedures

- **Incisional**
  - Radial keratotomy
  - Astigmatic keratotomy
  - Limbal relaxing incisons
- **Laser ablation**
  - PRK
  - LASIK
  - SMILE
- **Corneal implants**
  - ICL
  - Intrastromal corneal ring segments (INTACS)

Preoperative evaluation

- History Taking
- Screening
- Examination and counselling

Ophthalmic Examination

- VA
- Refraction
- Gross External Examination
- SLE
- Fundus Examination
- Jones' Basal tear secretion rate

Patient selection

- Age: 18 or more
- Stable refraction: at least 6-12 months
- Normal eye exam
- Normal screening tests

Systemic contraindications

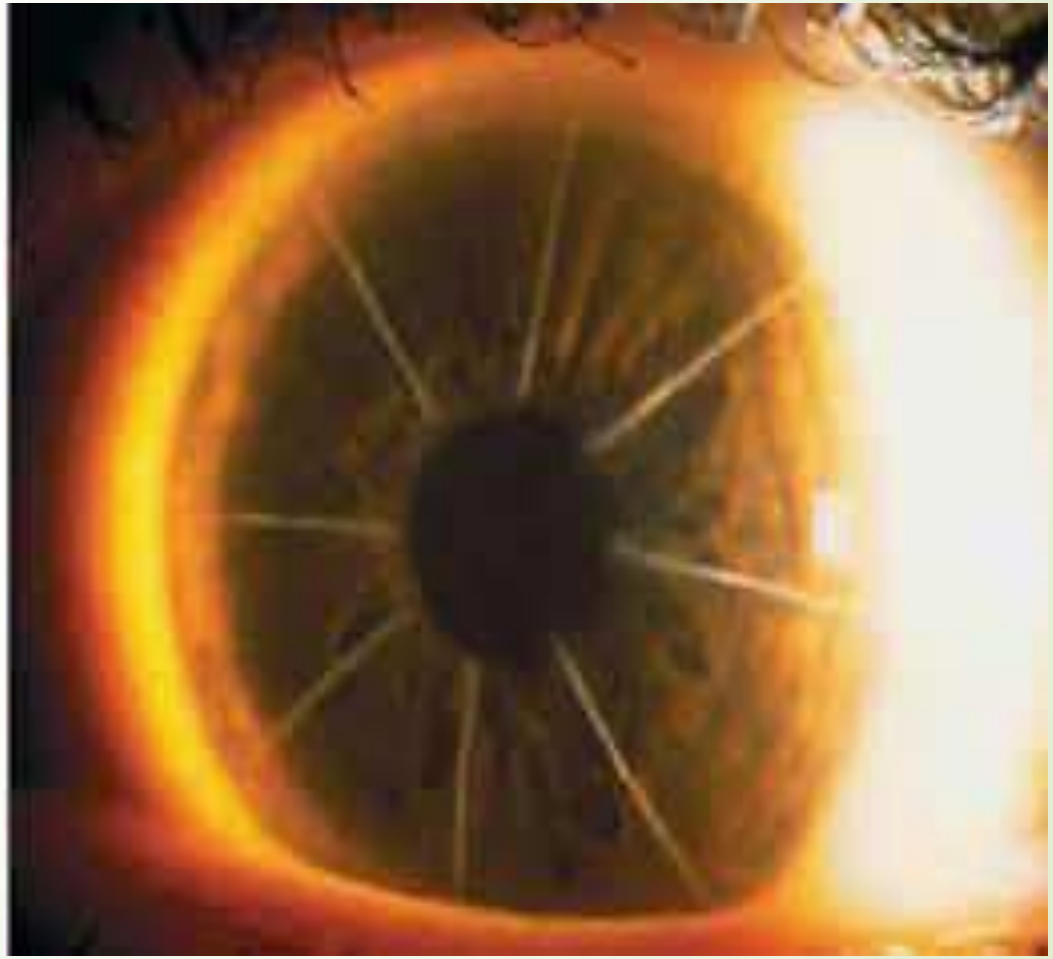
- DM
- Pregnancy/Lactation
- Autoimmune disorders(RA,SLE,PAN)
- Immunodeficiency
- Abnormal wound healing(Keloid)

Ophthalmic Contraindications

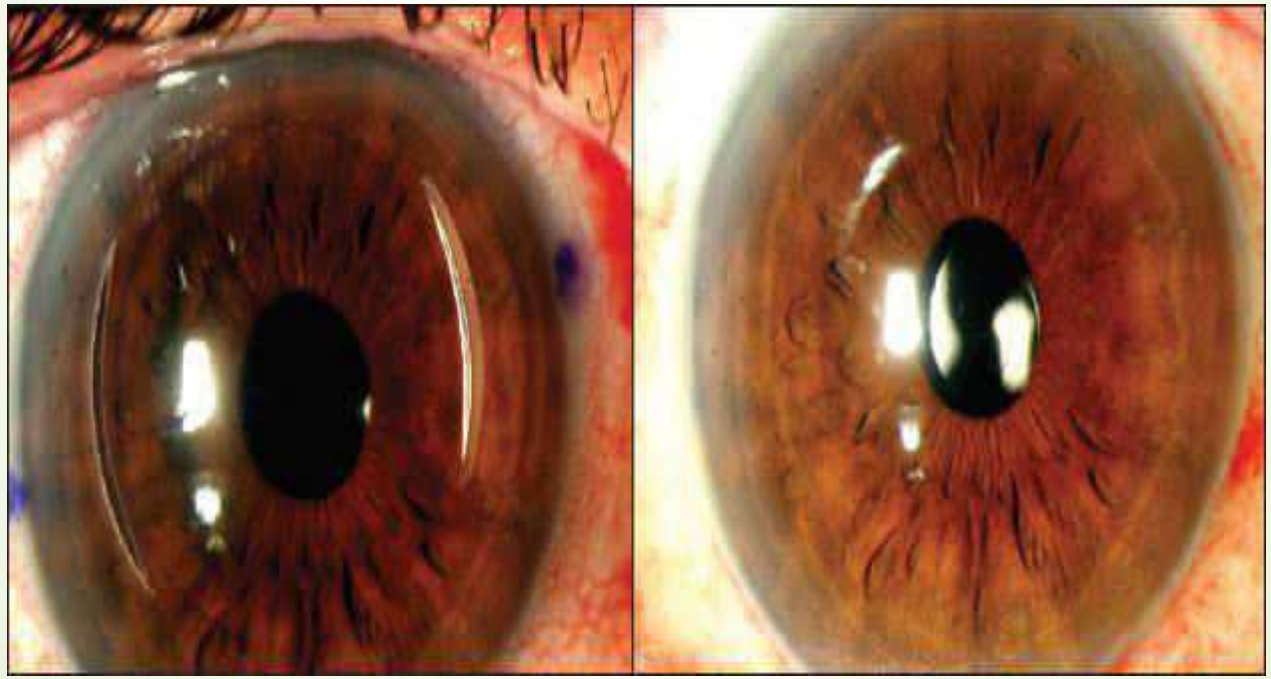
- HZO, HSK
- Glaucoma
- Dry eyes
- High irregular astigmatism
- Corneal ectasia i,e KC, PMD
- Uveitis, Progressive retinal degenerations

Radial Keratotomy

- Russian surgeon svyatoslav Nikolay Fyodrov. 1970



Astigmatic Keratotomy



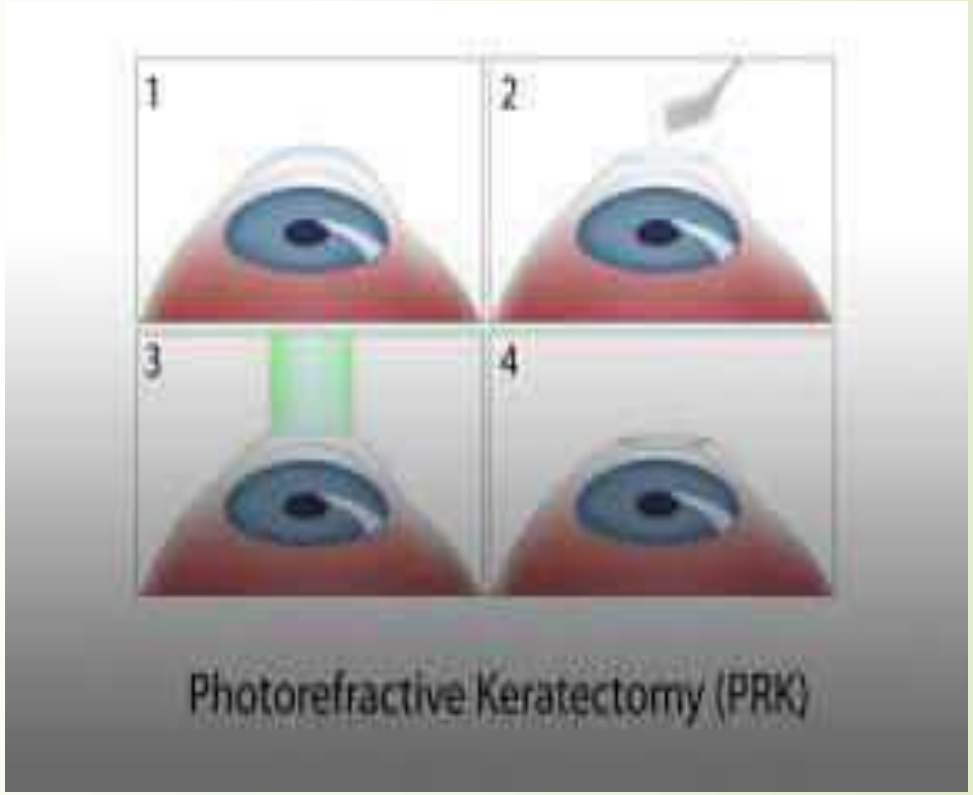
complications

- Under/overcorrection
- Instability of correction
- Astigmatism
- Perforation
- Traumatic globe rupture
- Bacterial keratitis
- Glare

- Blind eye, partially sighted and fully sighted clinical trials
- LASIK was developed by Ioannis Pallikaris

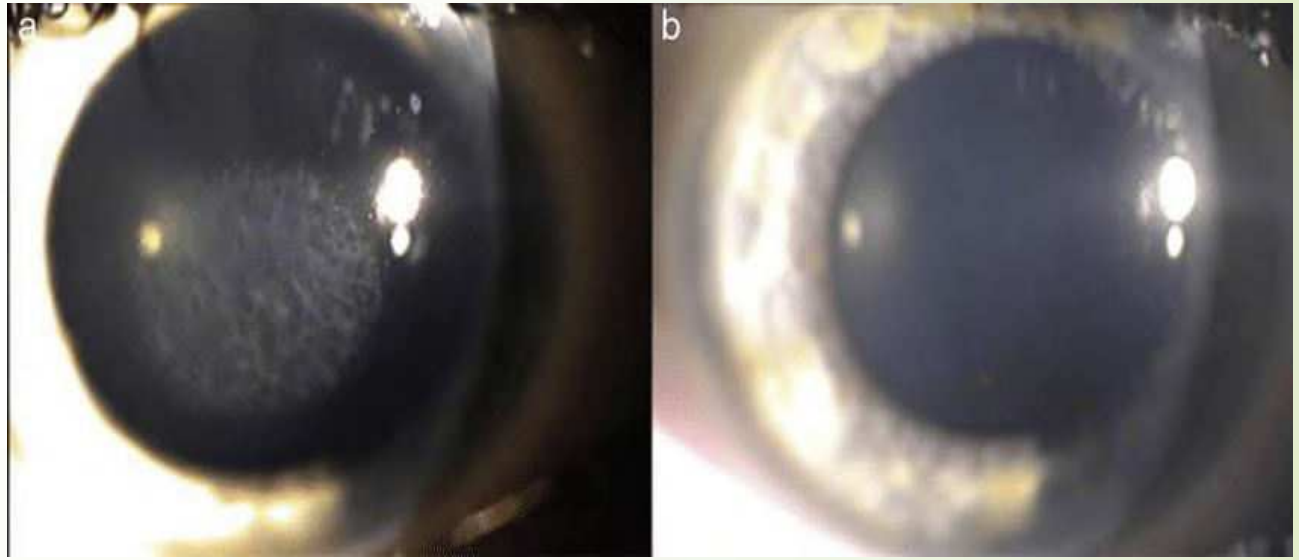
Photorefractive Keratectomy (PRK)

- Epithelial removal
- Patient fixate on aiming beam
- Photoablation of cornea
- MMC
- BCL



Complications

- Pain
- Decentration
- Corneal haze
- Keratitis
- Dryness



LASIK (Laser Assisted In Situ Keratomileusis)


BASIC LASIK PROCEDURE
2
3
Under L/A, cornea is marked
Suction ring applied (65mmHg)
Microkeratome cut hinged flap
4
5
6
Flap folded to expose stromal bed
Stromal ablated
Flap repositioned

Advantages

- No or minimal pain
- Early recovery
- Residual haze is unlikely
- High degree of correction
- Less regression

Disadvantages

- Flap complications
  - Incomplete
  - Thin flap
  - Button hole
  - Free cap
- Army personnel
- Contact sports

SMILE(SMall Incision Lenticule Extraction)

- Patient raised to contact glass
- Suction is applied

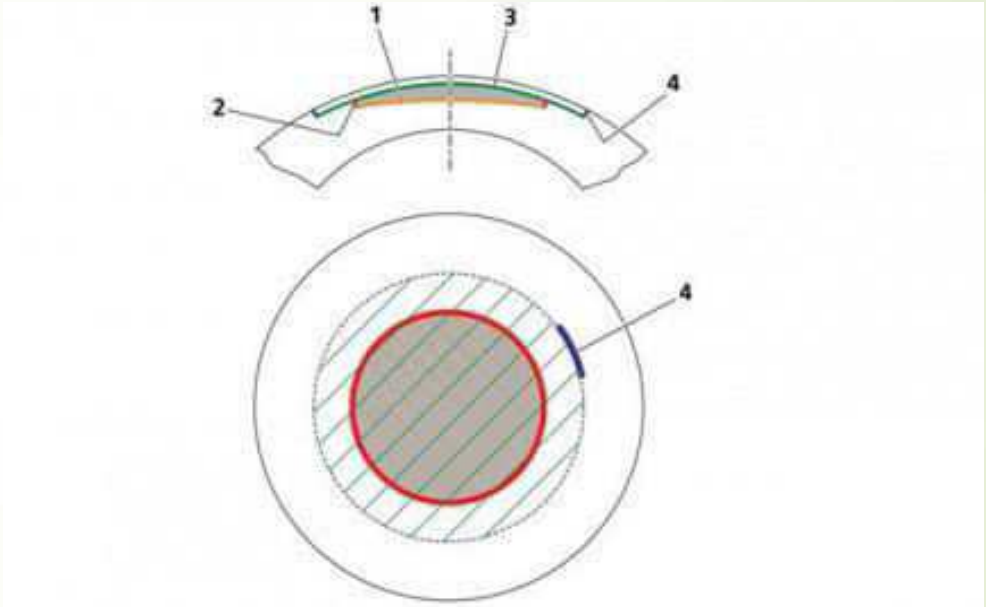
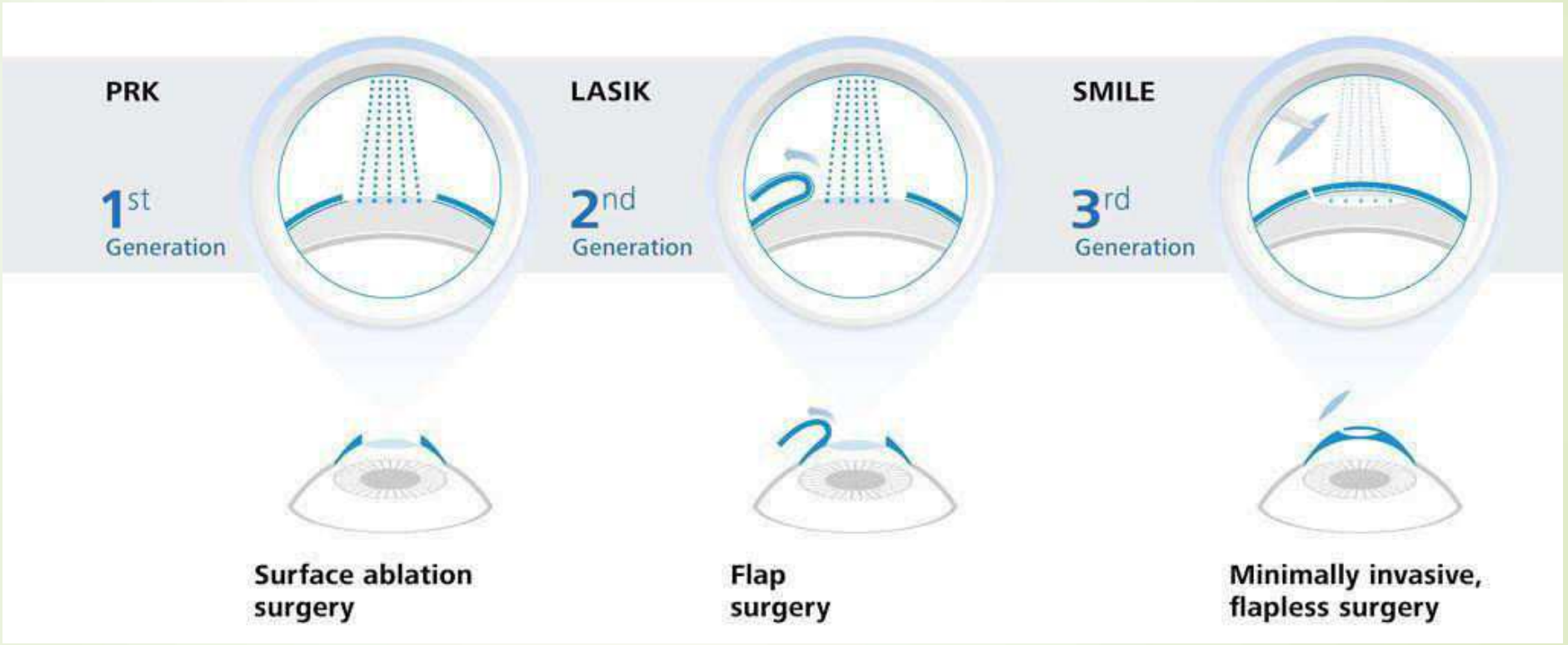


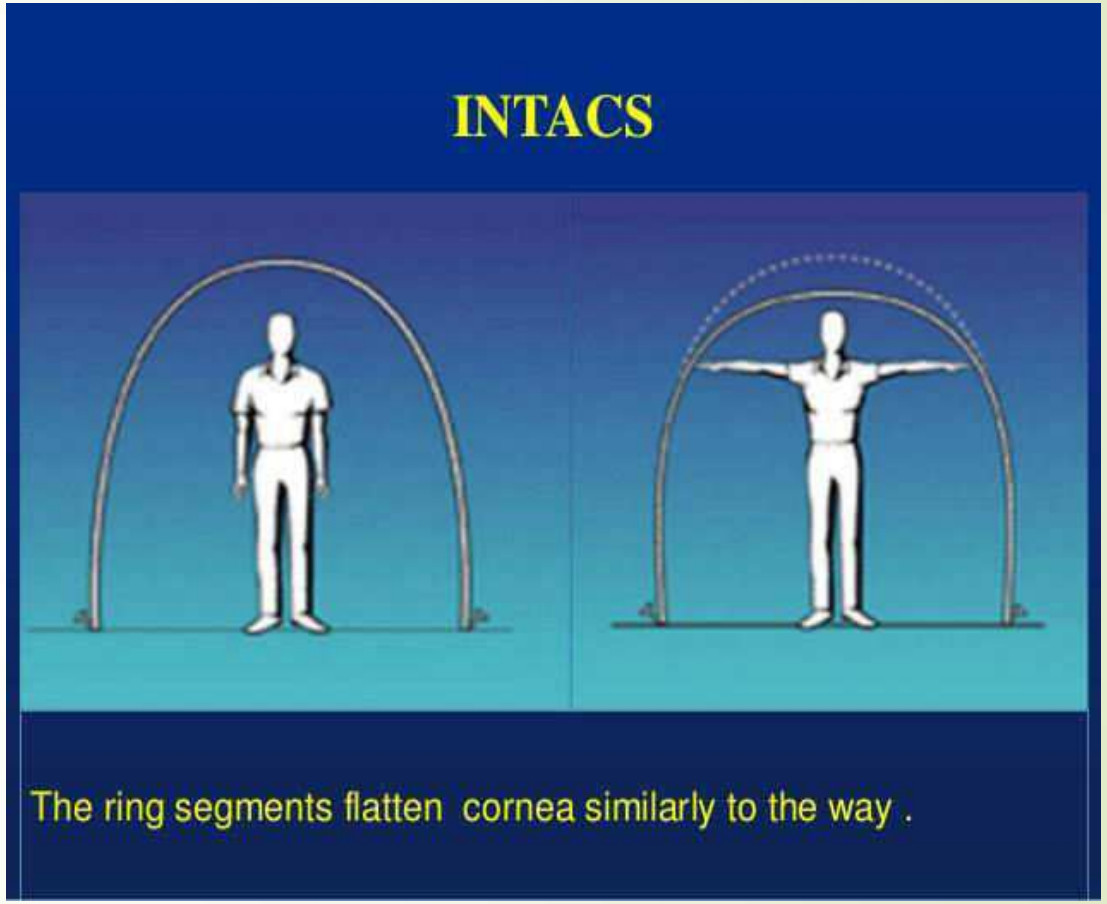
Fig. 1 Incision geometry of the SMILE Procedure

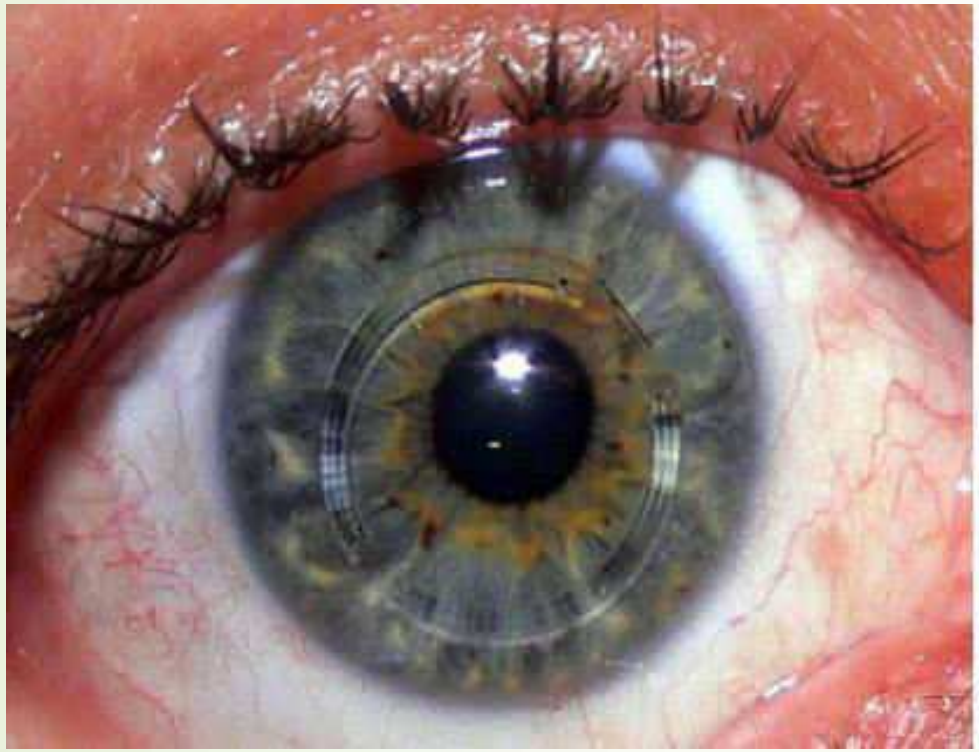
Reinstein *et al.*: Small incision lenticule extraction (SMILE) history, fundamentals of a new refractive surgery technique and clinical outcomes. *Eye and Vision* 2014 1:3. Web 10 Mar. 2015. http://www.eandv.org/content/pdf/s40662-014-0003-1.pdf

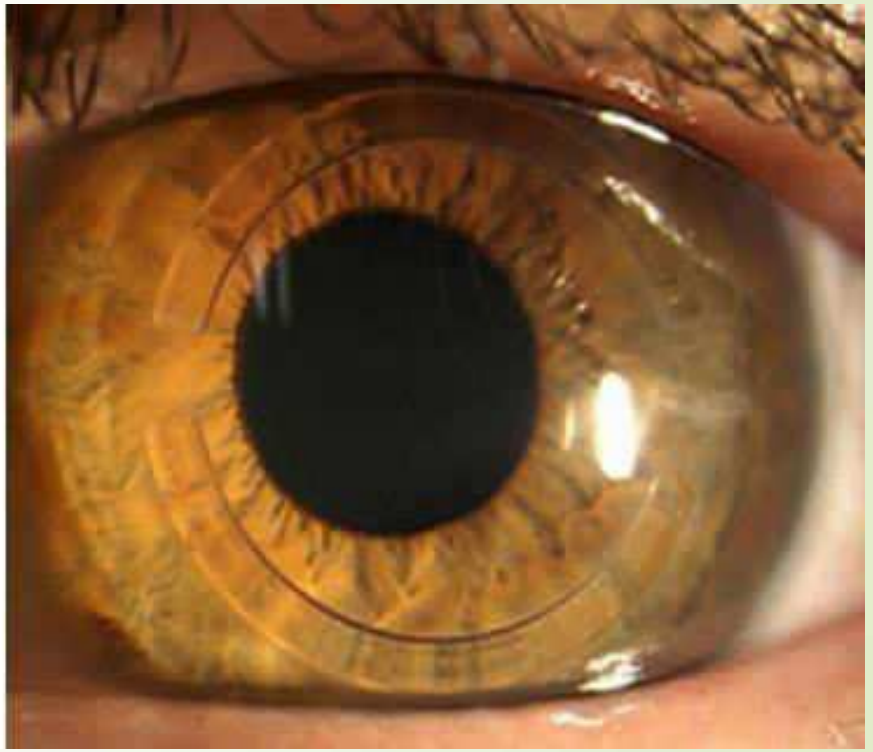

PRK
1st Generation
LASIK
2nd Generation
SMILE
3rd Generation
Surface ablation surgery
Flap surgery
Minimally invasive, flapless surgery

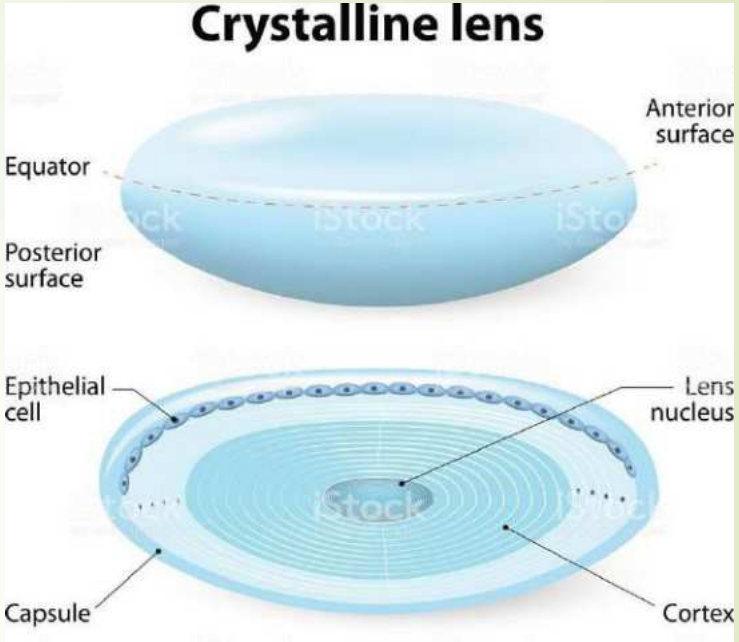
Intracorneal Ring Segments

- Intacs
- Keratoconus
- Flatten central cornea









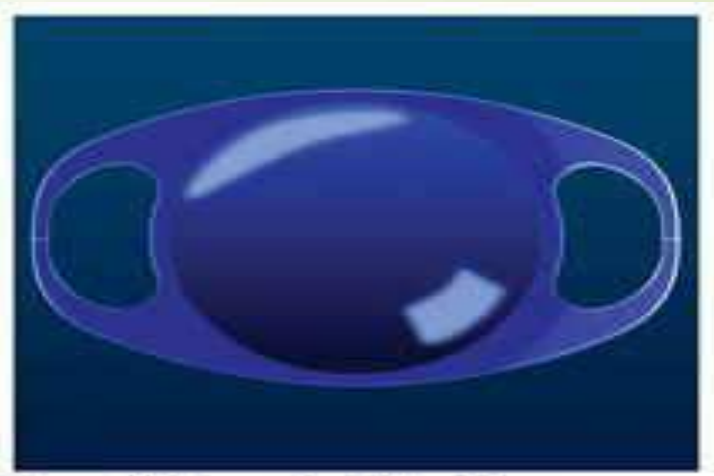
LENS BASED SURGERY

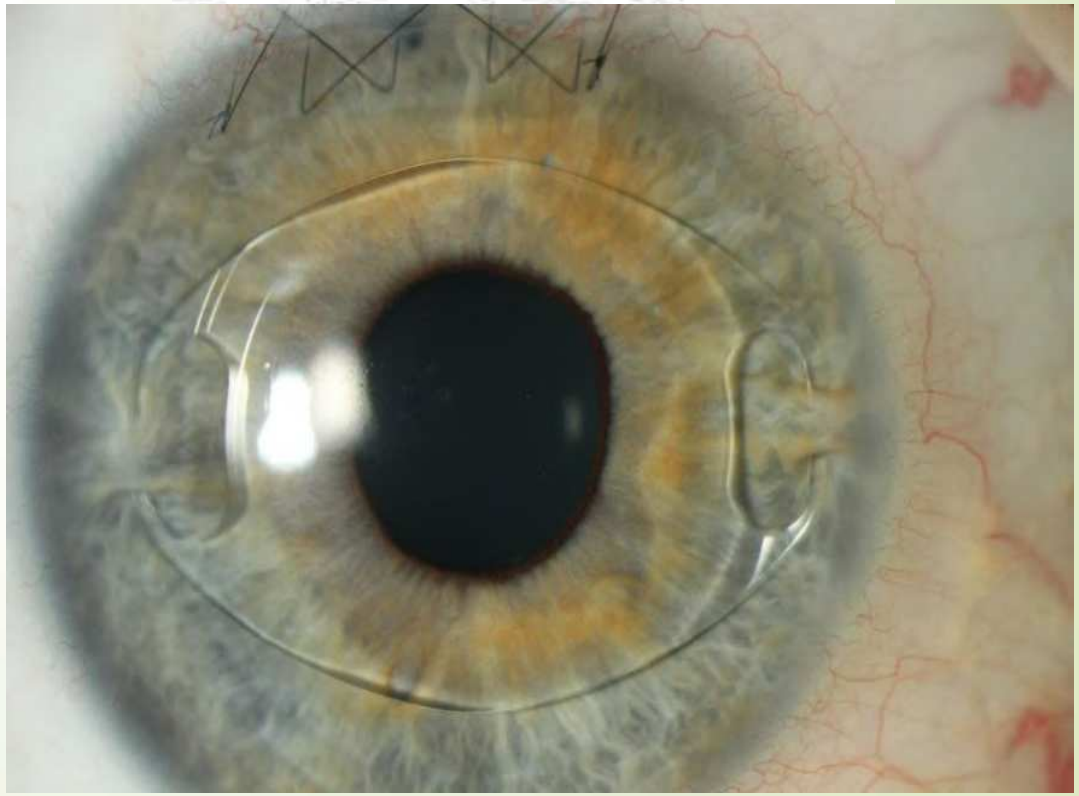
Phakic IOLs

- 1953 by Dr. Strampelli
- High refractive error
- Not suitable for corneal procedures

Artisan

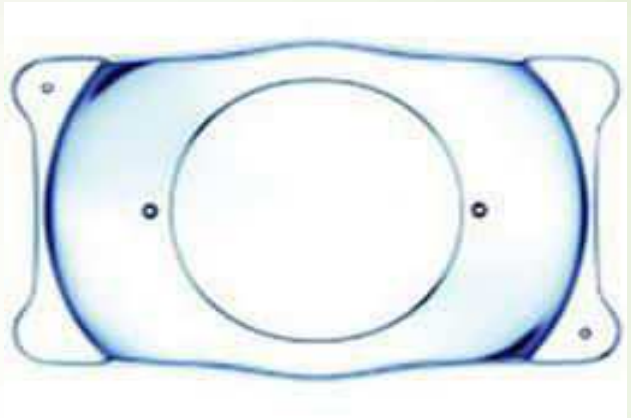
- Iris claw
- AC IOL
- Complications
  - Inflammation
  - Glaucoma
  - Decompensations

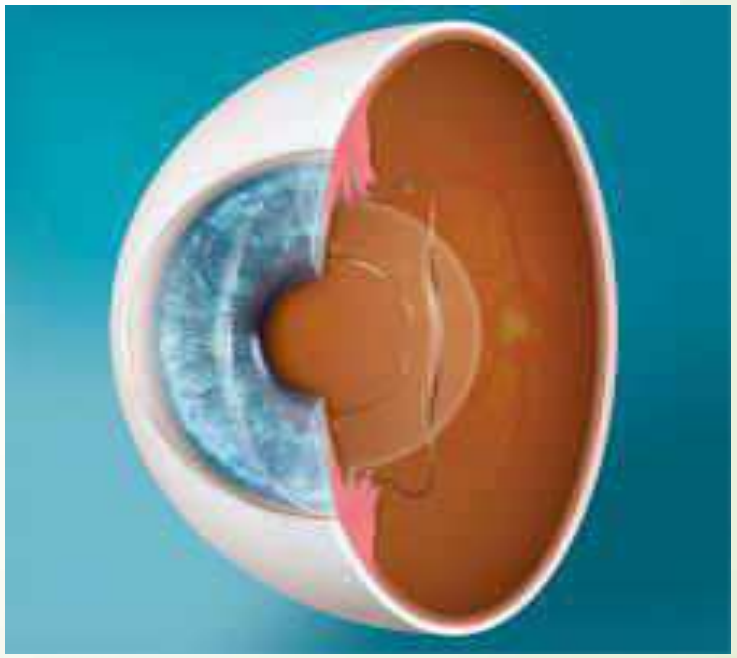




visian

- PC pIOL
- b/w iris and lens
- Complications
  - Cataract
  - Glaucoma
  - Inflammation





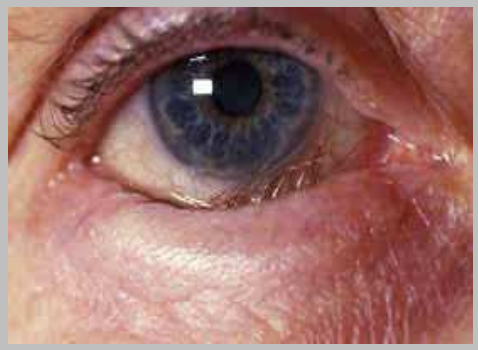
Refractive Lens Exchange

- Clear lens exchange
- Presbyopic age
- High Hypermetrope

Thank you for your cooperation

Eyelid Abnormalities

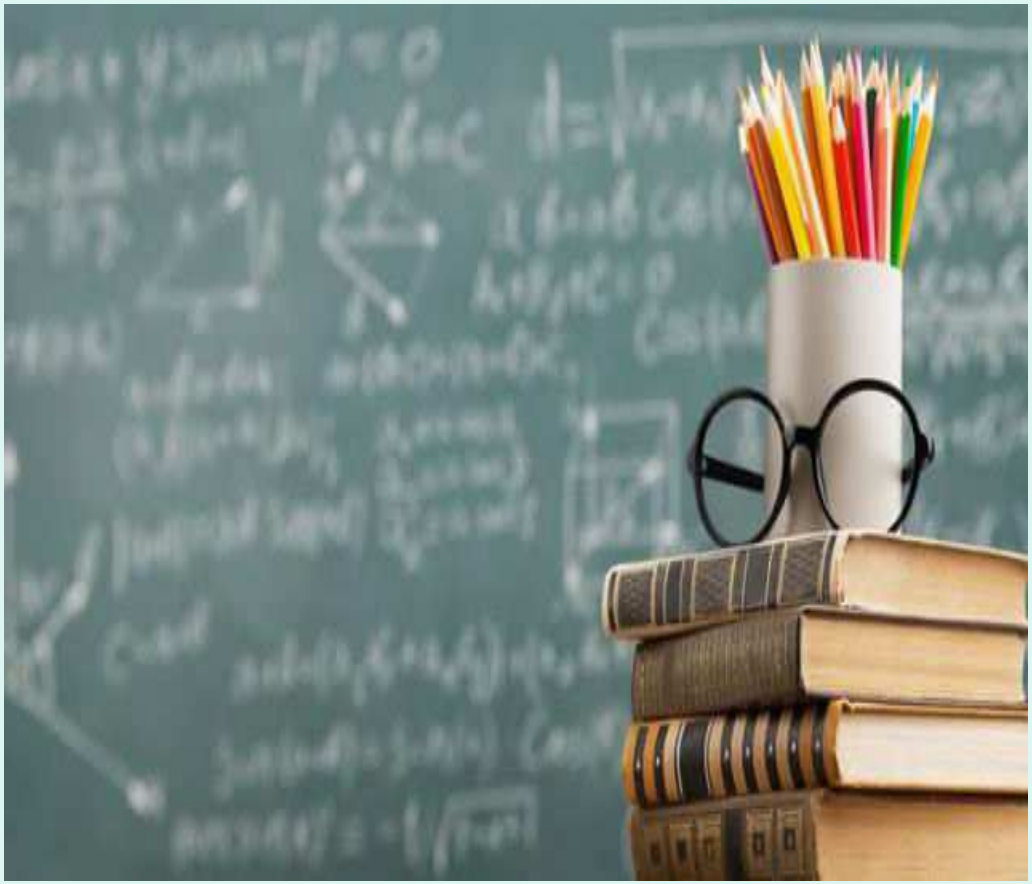
ENTROPION



ECTROPION

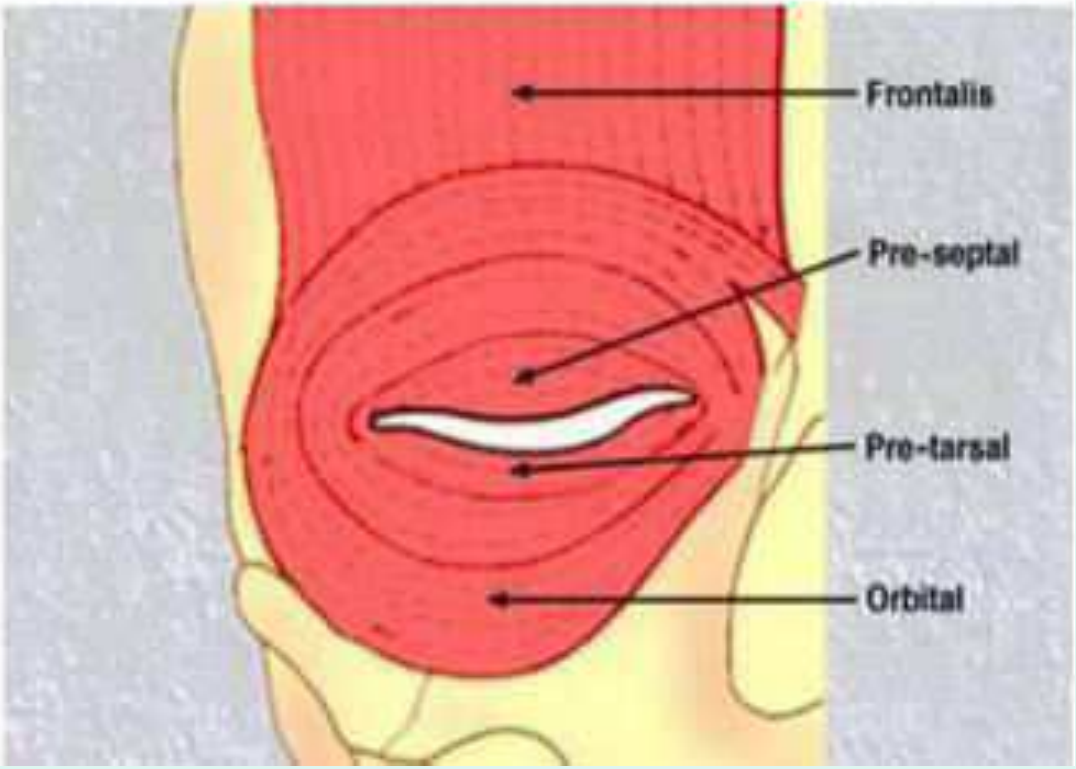
Presentation lay out

- ***Anatomy of eyelids***
- ***Factors responsible for maintaining lower eyelid position***
- ***Definition***
- ***Classification***
- ***Etiology***
- ***Clinical features***
- ***Management***
- ***References***

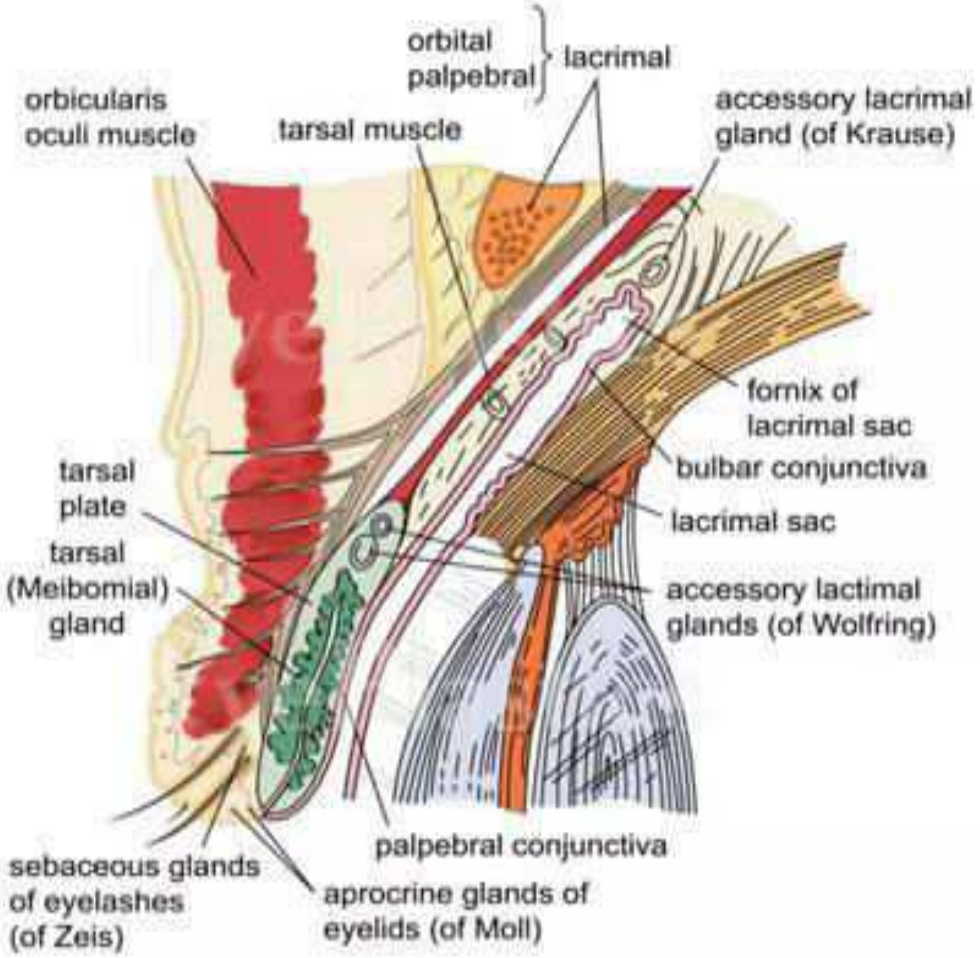


ANATOMY OF EYELIDS:-

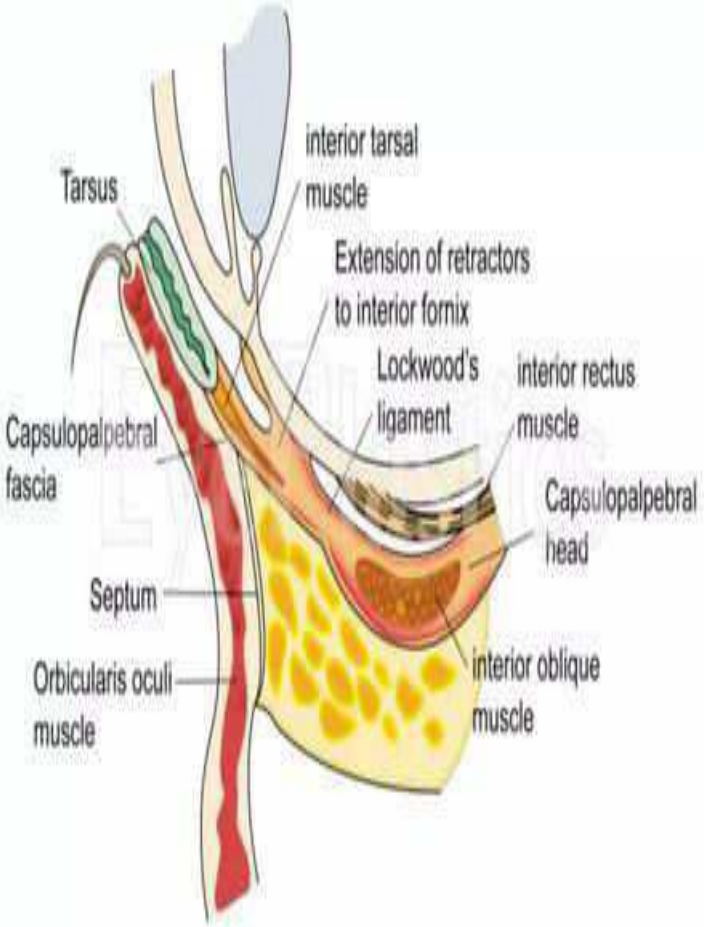
- SKIN- thin,stretches with age & there is usually excess available for a full thickness skin graft.
- ORBICULARIS MUSCLE:-



- ***UPPER EYELID ANATOMY:-***



LOWER EYLID ANATOMY:-



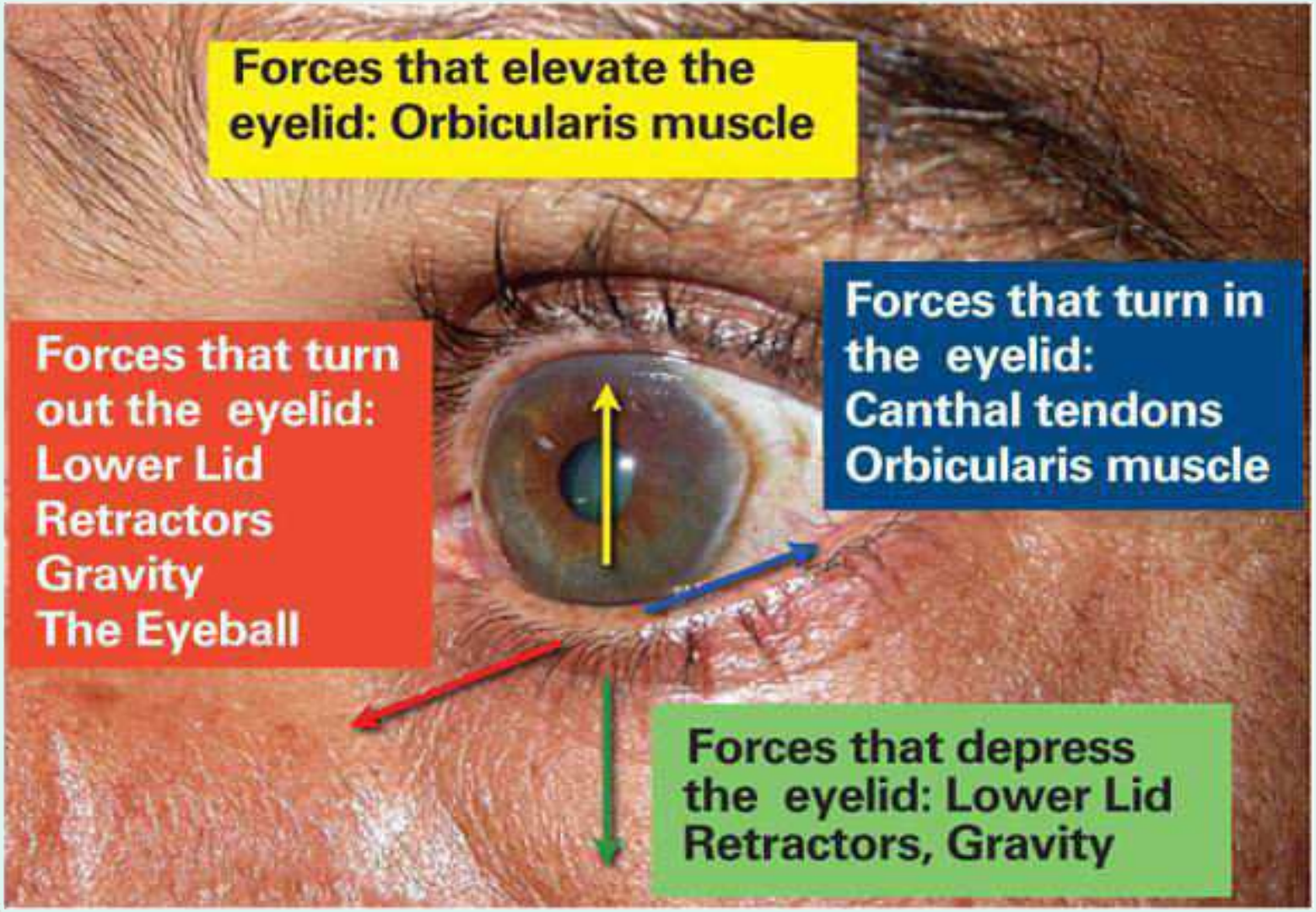
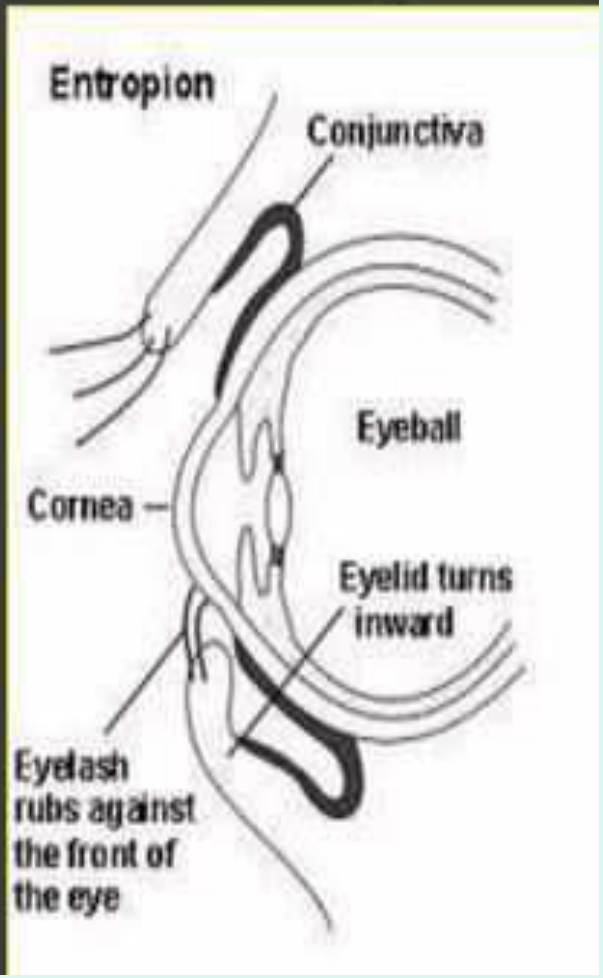


Figure 1. Forces acting on the lower eyelid to maintain normal position.


entropion

Definition

- Rolling inwards of lid margin is called entropion
- Produced by disparity in length and tone between anterior skin muscle,and posterior tarsoconjunctival laminae of eyelid.



Classification

- INVOLUTIONAL
- CICATRICIAL
- SPASTIC
- CONGENITAL

Clinical picture

SYMPTOMS

- Foreign body sensation
- Photophobia
- Irritation
- Pain
- Lacrimation

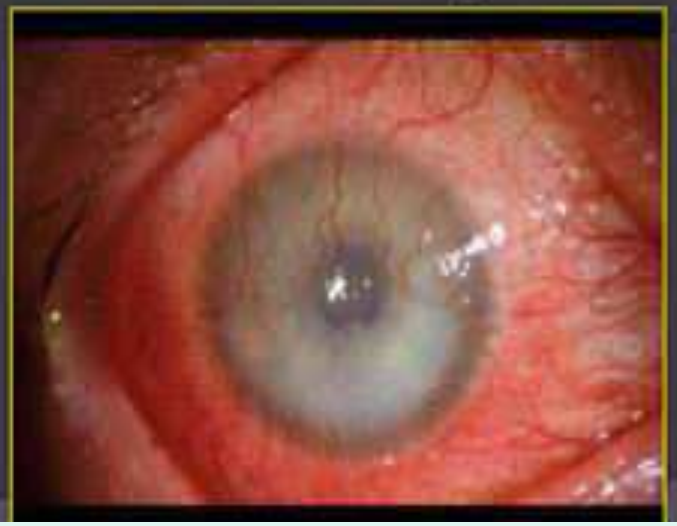
Clinical picture

SIGNS

- Lid margin is **inturned**
- Depending on degree of in turning,dividing into three grades.
- **Grade 1** only posterior lid border is inrolled.
- **Grade 2** inturning of intermarginal strip.
- **Grade 3** whole lid margin including anterior border inturned

Clinical picture

- **COMPLICATIONS**
- Recurrent corneal abrasions
- Superficial corneal opacities
- Corneal vascularisation
- Non healing corneal ulcer

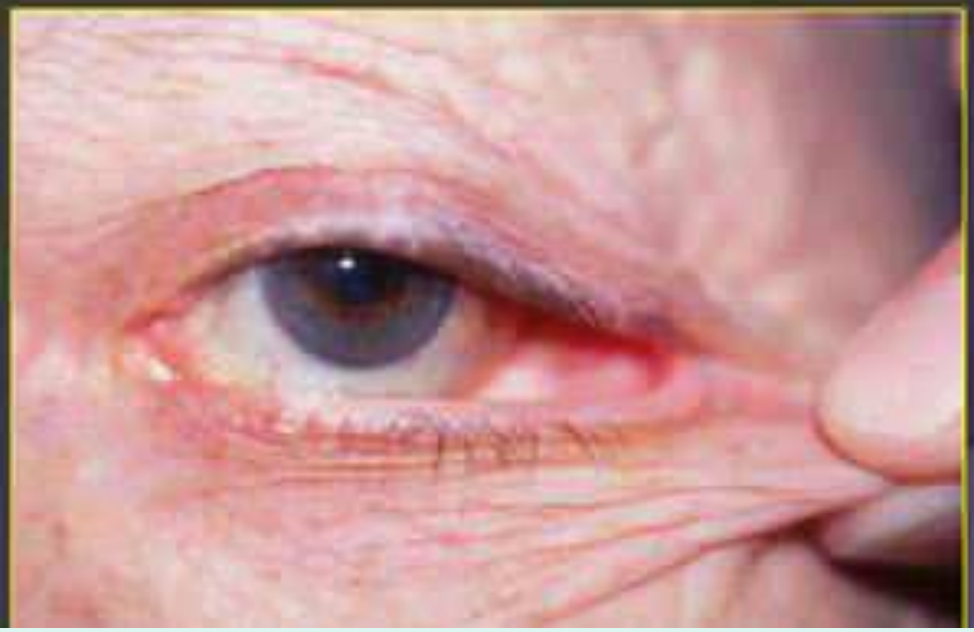


Involutional entropion

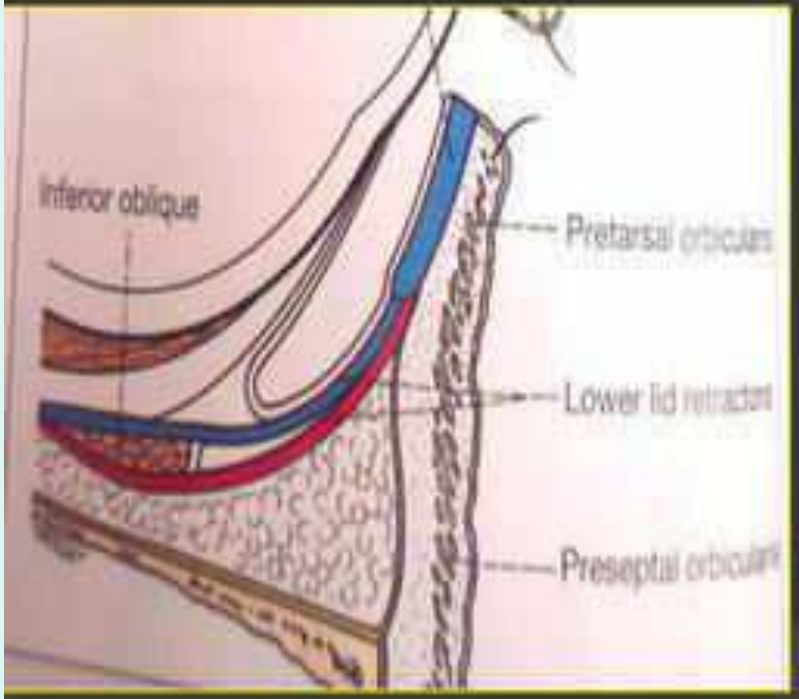
- Age related entropion.
- Affects mainly the lower eye lid.

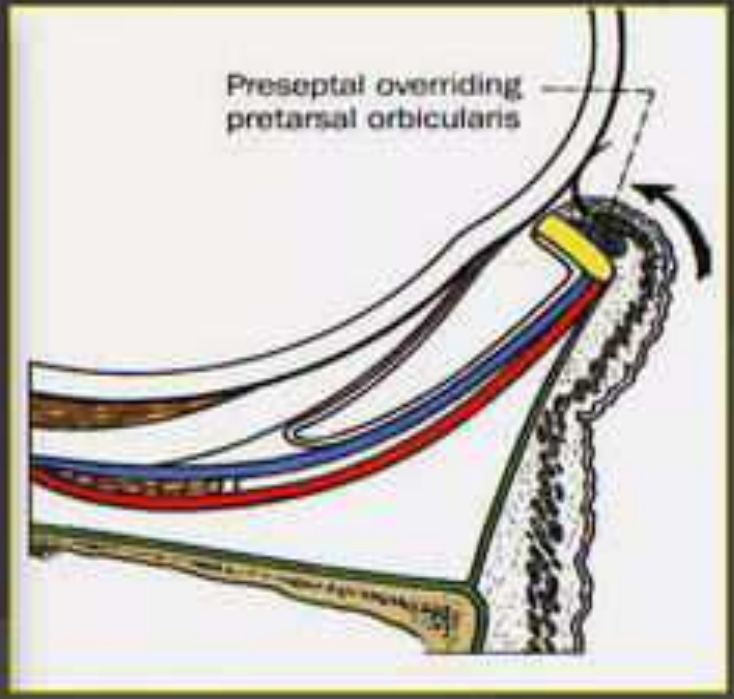
Pathogenesis

- Age related degeneration of elastic fibrous tissue within the eyelid results in following
- **HORIZONTAL LID LAXITY** caused by stretching of canthal tendons and tarsal plate.

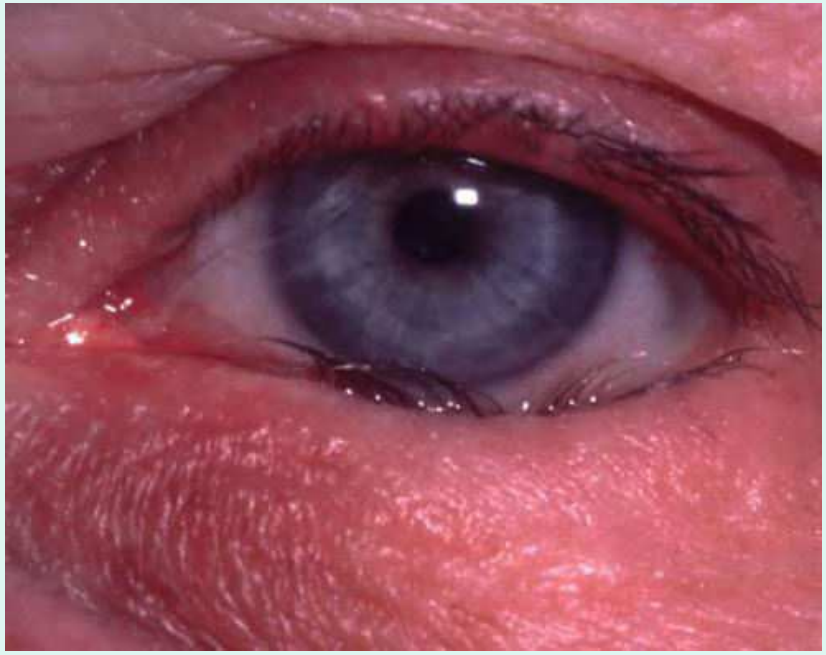


- **VERTICAL LID INSTABILITY** caused by disinsertion of lower eyelid retractors.
- **OVERRIDING** of the pretarsal by the preseptal orbicularis

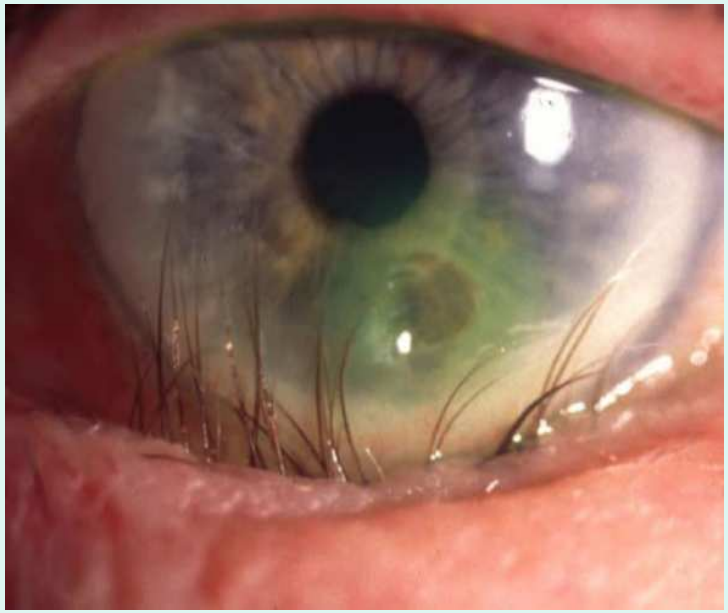




Involutional entropion



Affects lower lid because upper lid has wider tarsus and is more stable



If longstanding may result in corneal ulceration

Treatment

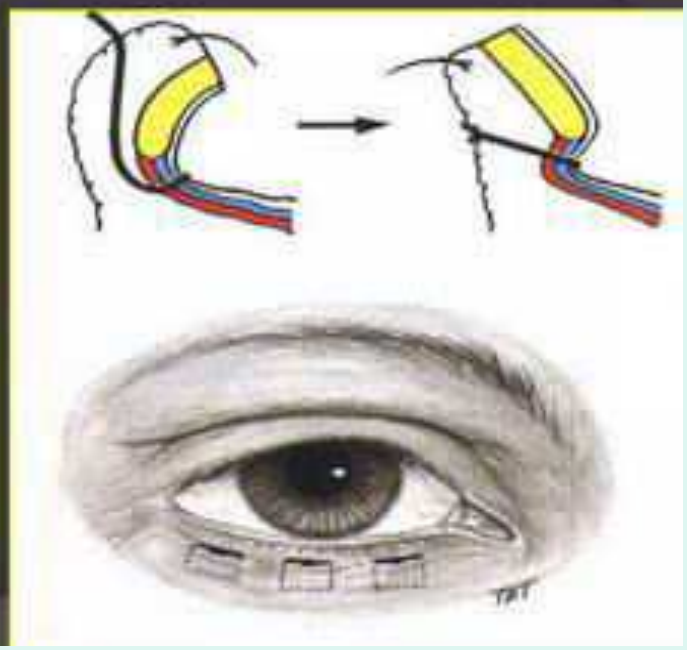
MEDICAL

- Lubricants
- Taping
- Soft bandage contact lenses
- Adhesive tape-pulling the skin outward with strip of adhesive tape
- injection of botulinum toxin

SURGERY

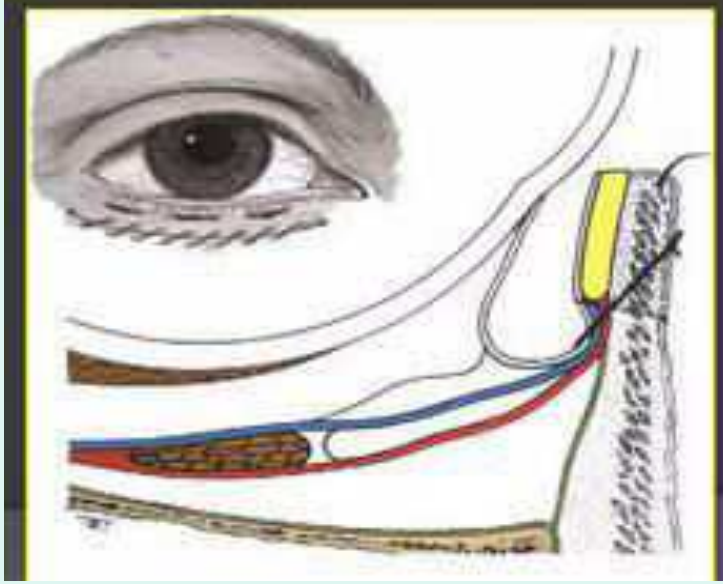
TRANSVERSE EVERTING SUTURES

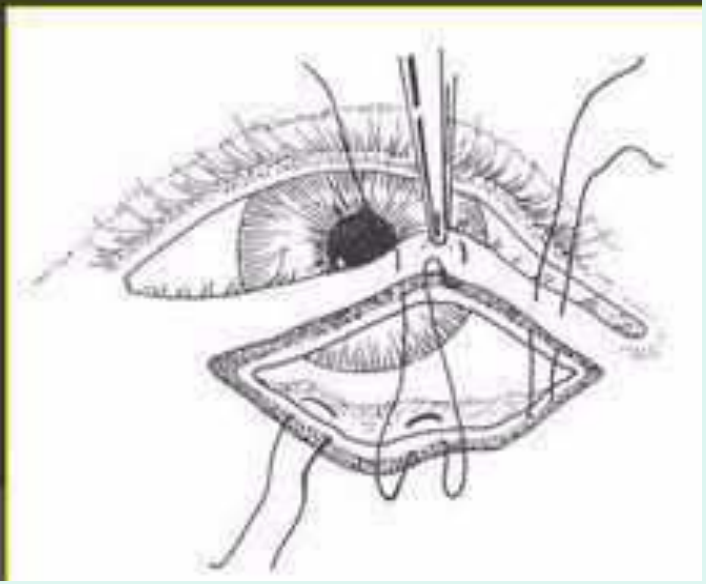
- Prevents overriding and provide temporary corrrection lasting several months.



WEIS PROCEDURE

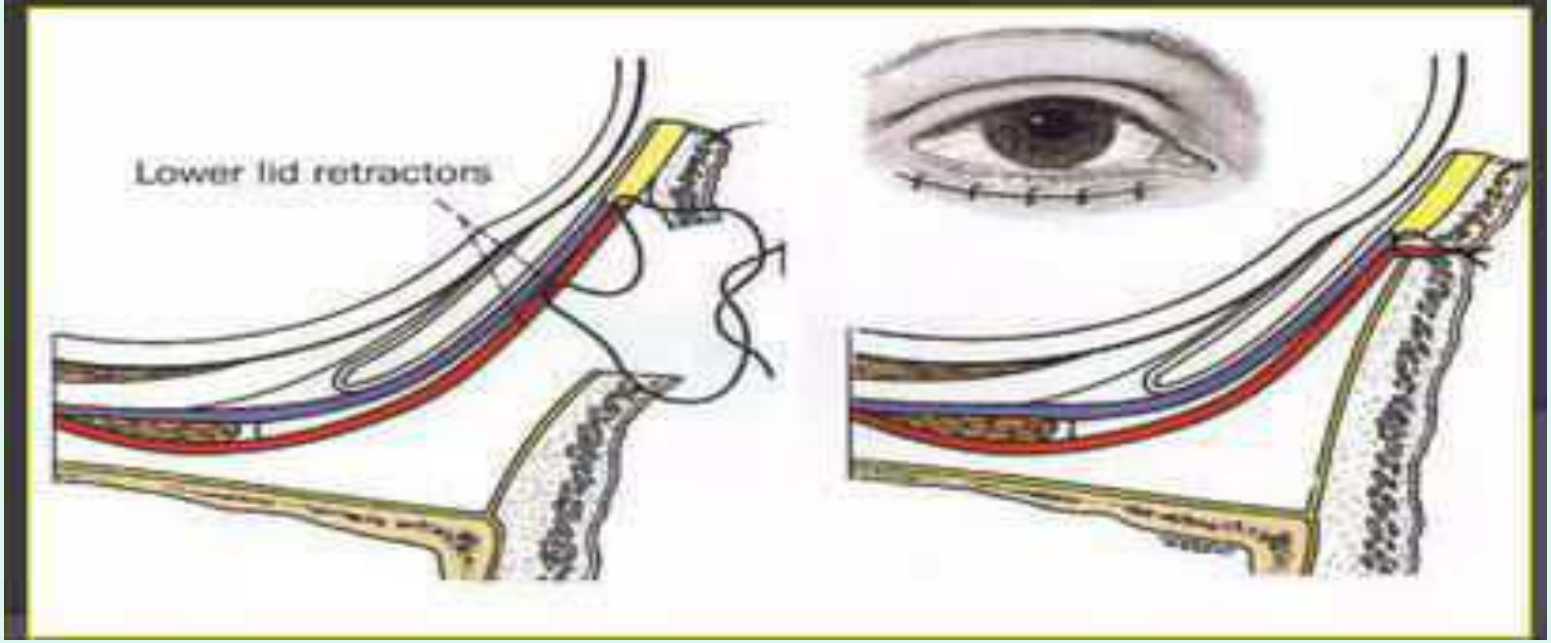
- Full thickness horizontal lid spliting and insertion of everting sutures .
- Scarring prevents overriding of preseptal and pretarsal parts of orbicularis.





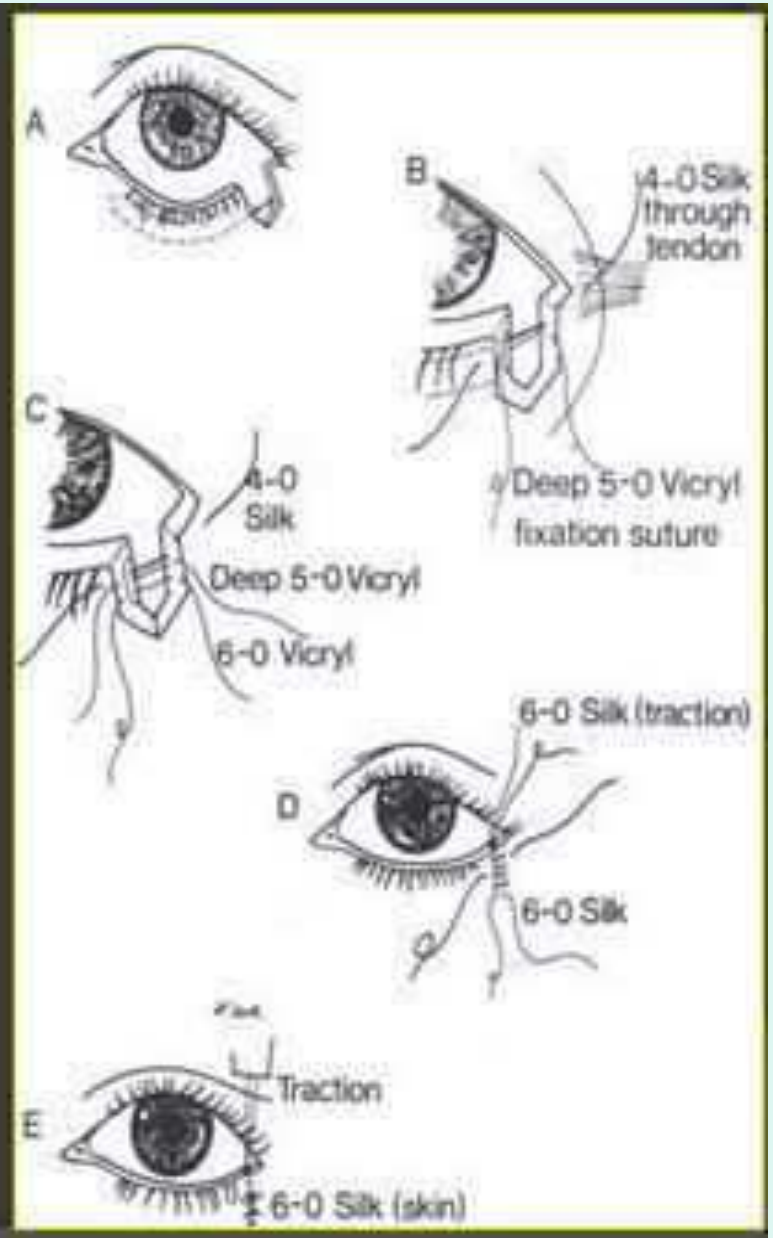
JONES PROCEDURE

- Plication of lower eyelid retractors thus increasing their pull and creating the barrier between preseptal and pretarsal portions of orbicularis
- Performed in recurrent cases.



BICKS PROCEDURE WITH REEH'S MODIFICATION

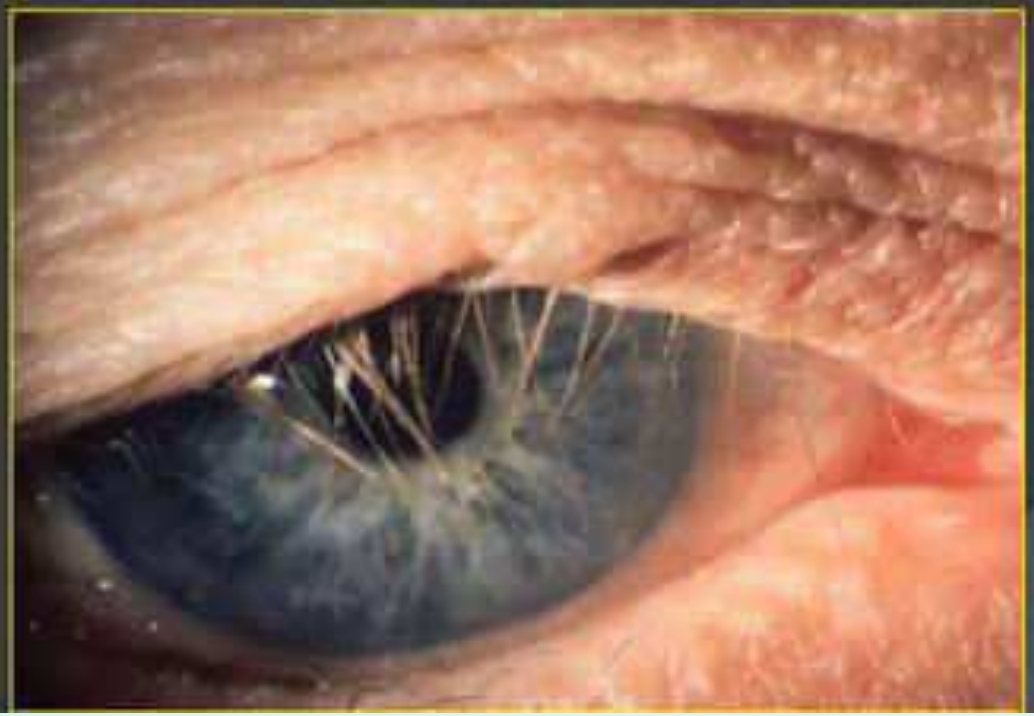
- Useful in patient with horizontal laxity.



Cicatricial entropion

PATHOGENESIS

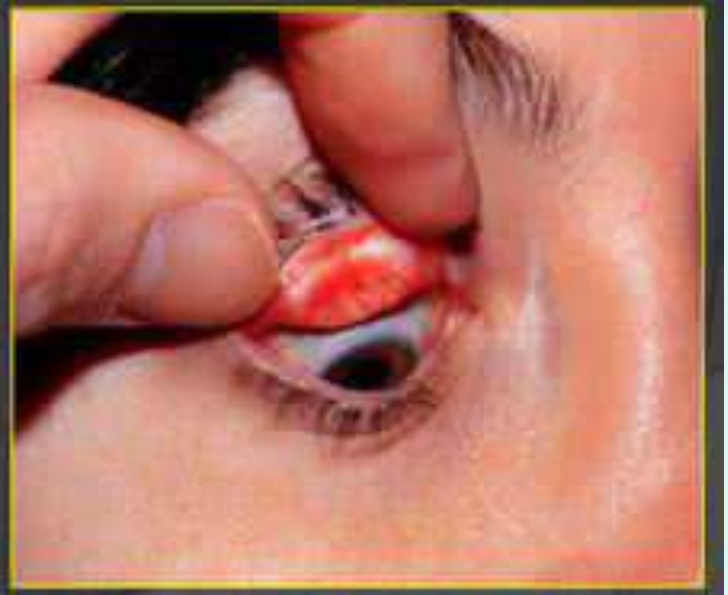
- It is caused by severe scarring of the palpebral conjunctiva which pulls the upper or lower lid margin towards the globe.



CAUSES

- Cicatrizing conjunctivitis
- Trachoma
- Trauma
- Chemical injuries

Cicatricial entropion of upper lid.

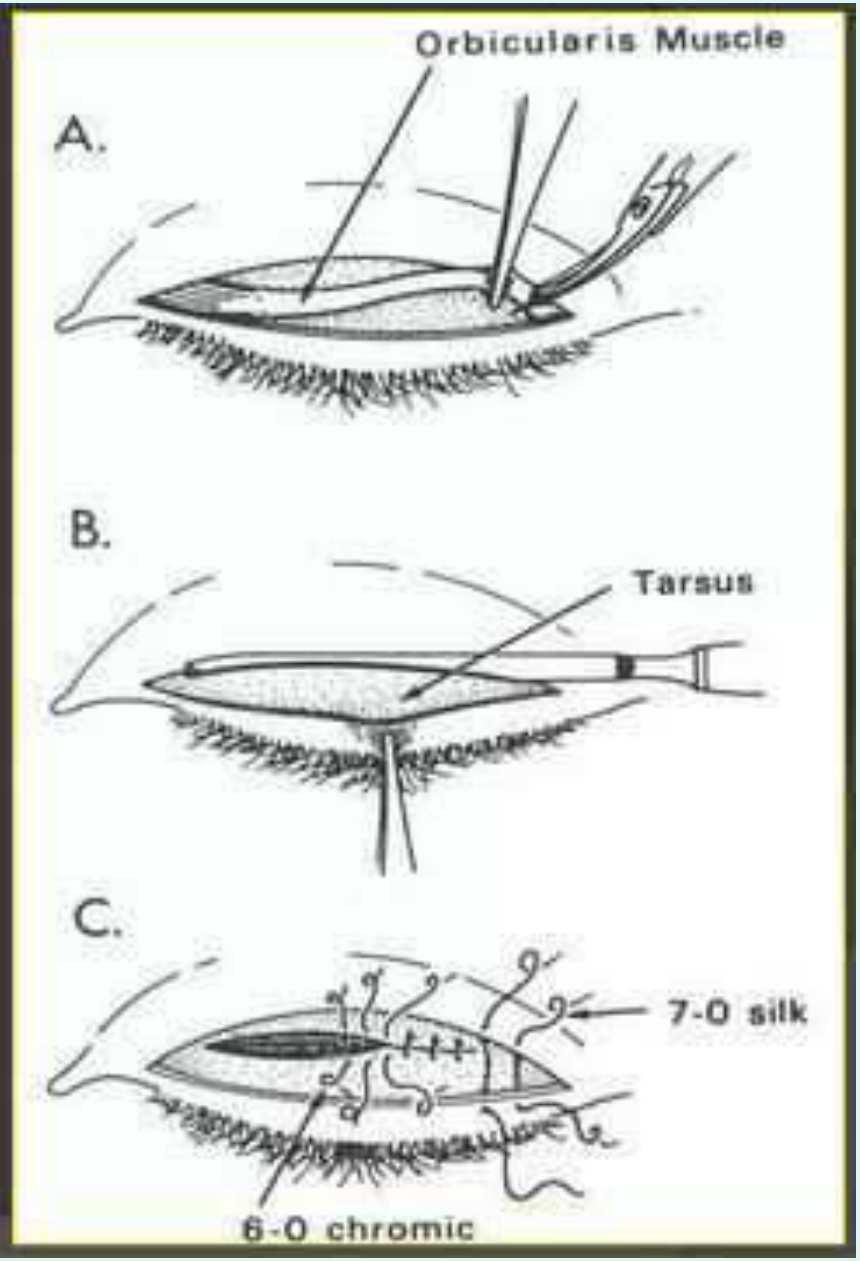


scar tissue involving tarsal conjunctiva

Treatment

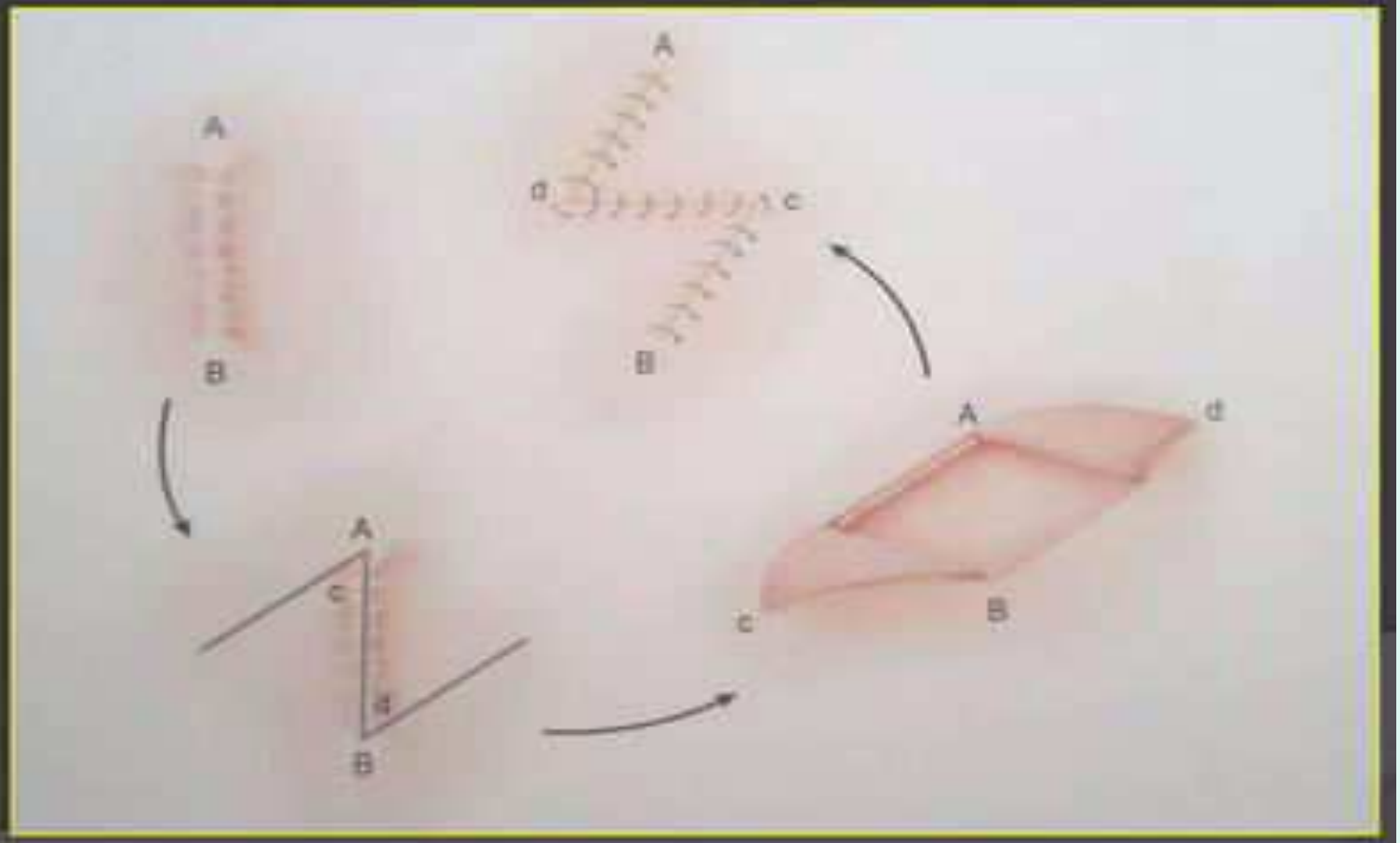
SURGERY

- Mild to moderate cases
- Wedge resection of tarsus



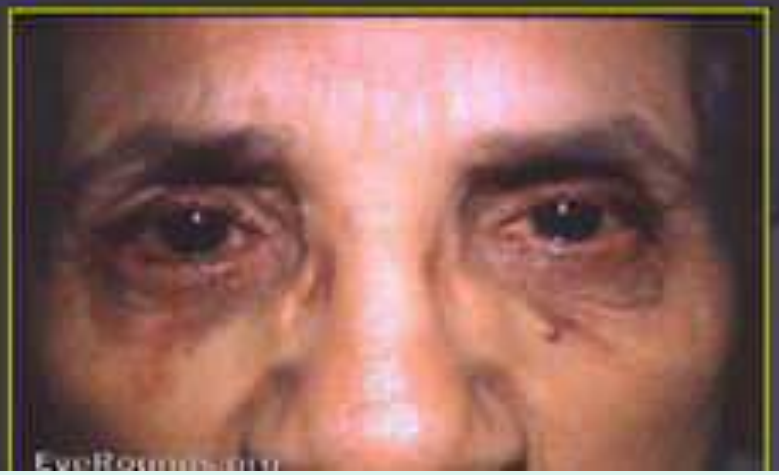
Linear Scar

Z PLASTY



Spastic entropion

- Spasm of orbicularis muscle in presence of degeneration of palpebral connective tissue separating orbicularis muscle fibres.
- Degeneration of aponeurosis of orbicularis muscle tends to approximate lid margins and turns them inwards on contraction.
- Horizontal lid laxity



CAUSES

- Ocular irritations causing inflammation and trauma

  Chronic conjunctivitis

  Keratitis

- Tight bandage post operatively
- Blepharophimosis

Treatment

MEDICAL

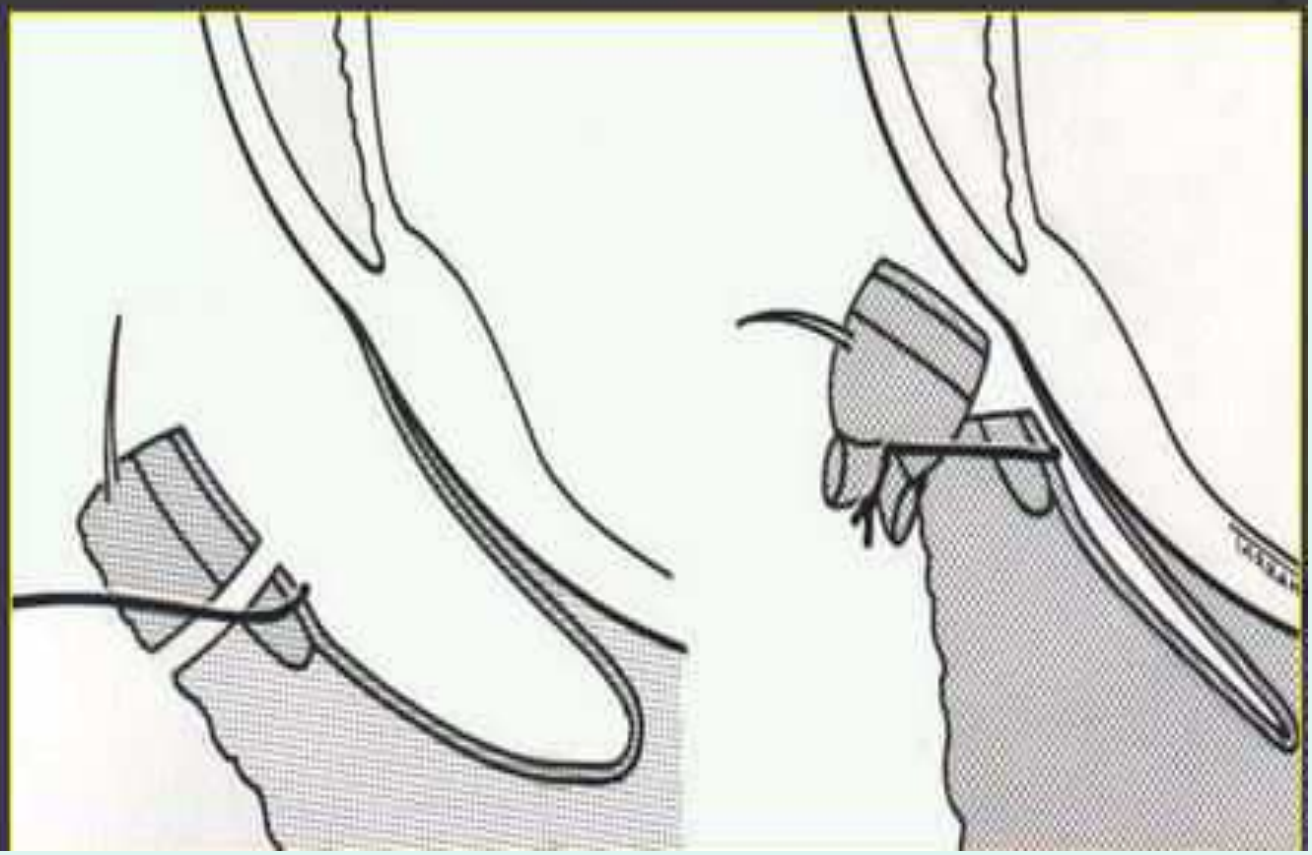
- Lubricants for surface disorders
- Antibiotics for conjunctival or lid inflammation.
- Removal of bandage.
- Injection of botulinum toxin.
- In elderly ,eversion of lid margin with adhesive plaster.

SURGERY

- A ridge of fibrous tissue in orbicularis muscle is made for preventing the sliding of fibres vertically.
- If the spasm is not relieved then the following procedures can be done.

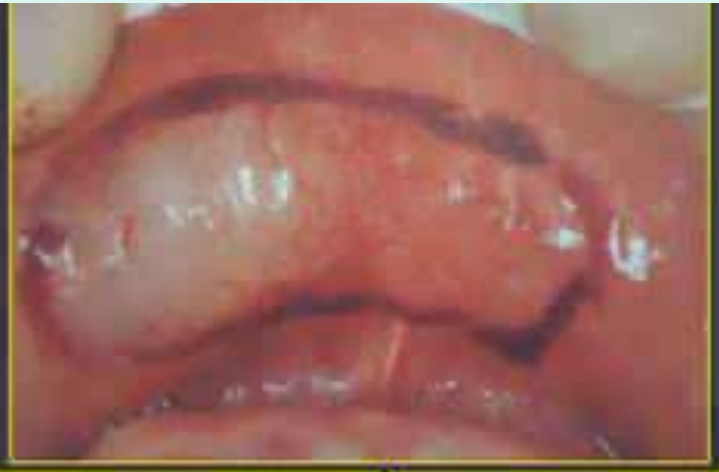
- **Weis procedure**
- **Jones procedure**
- **Bicks procedure**

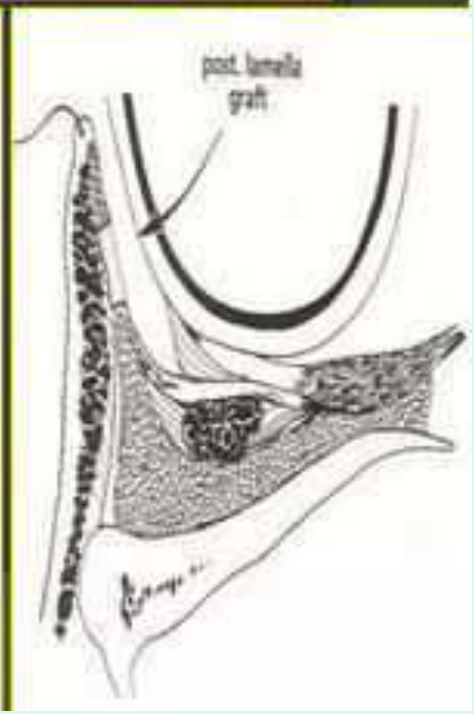
- Severe case
- Burrow operation or tarsal fracture



Extensive scarring

- Needs replacement of conjunctiva. Posterior lamella grafting
  - Conchal cartilage
  - Nasal chondromucosa
  - Palatal mucoperichondrium
  - Buccal mucosa





Congenital entropion

- Rare condition due to the dysgenesis of lower eyelid retractors or developmental abnormality of tarsal plate.
- Associated with microophthalmos.

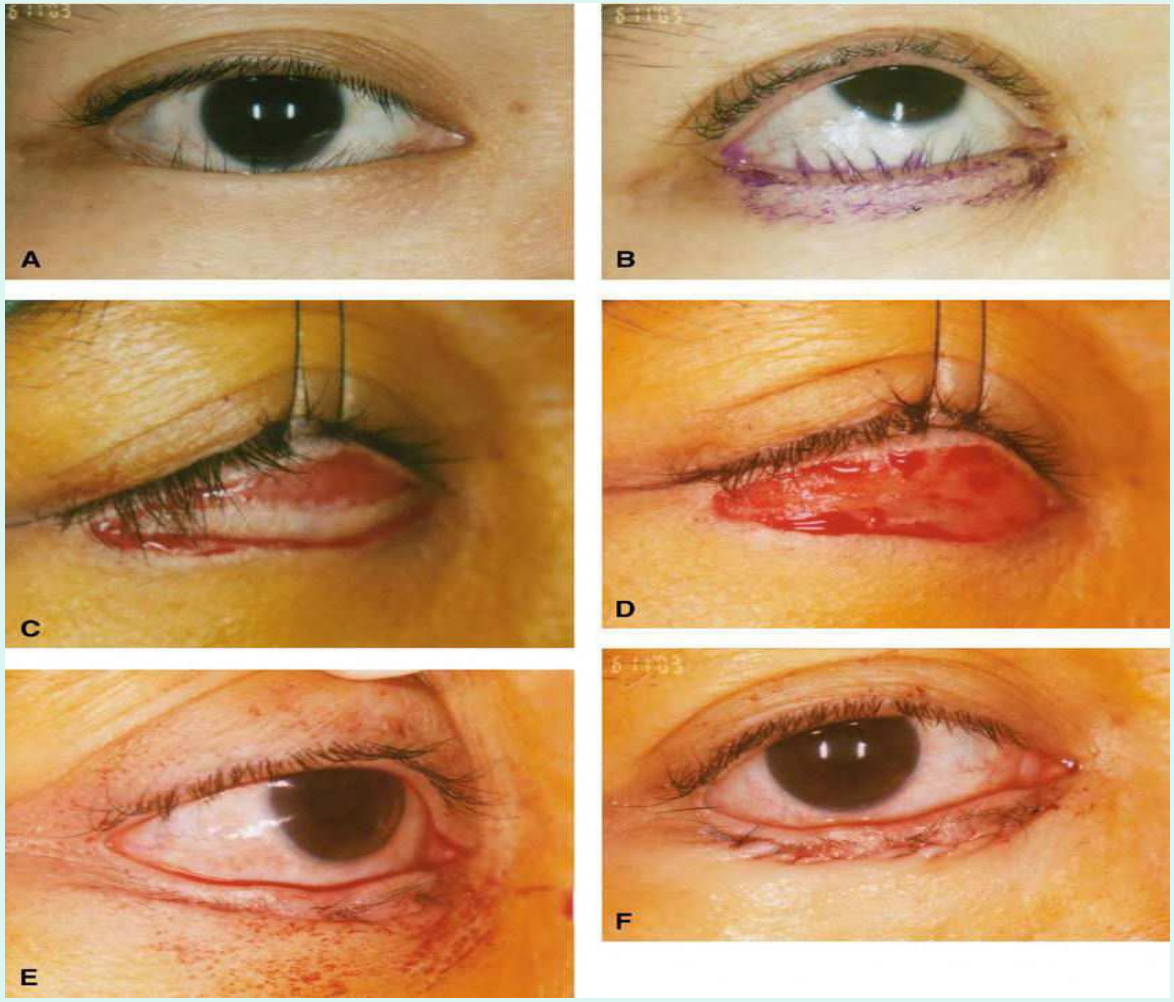
Epiblepharon

- Extra horizontal row of skin across lid margin
- When fold of skin is pulled down lashes turn out but lid remains in apposition to the globe.

Congenital entropion	Epiblepharon
Rare	Common
Inturning of entire lower eyelid and lashes	Extra horizontal row of skin across lid margin
Absence of lower lid crease	Presence of lowerlid crease
When skin is pulled down lid also pulls away from globe	Skin remains in apposition with the globe
Does not resolve spontaneously	Resolve spontaneously

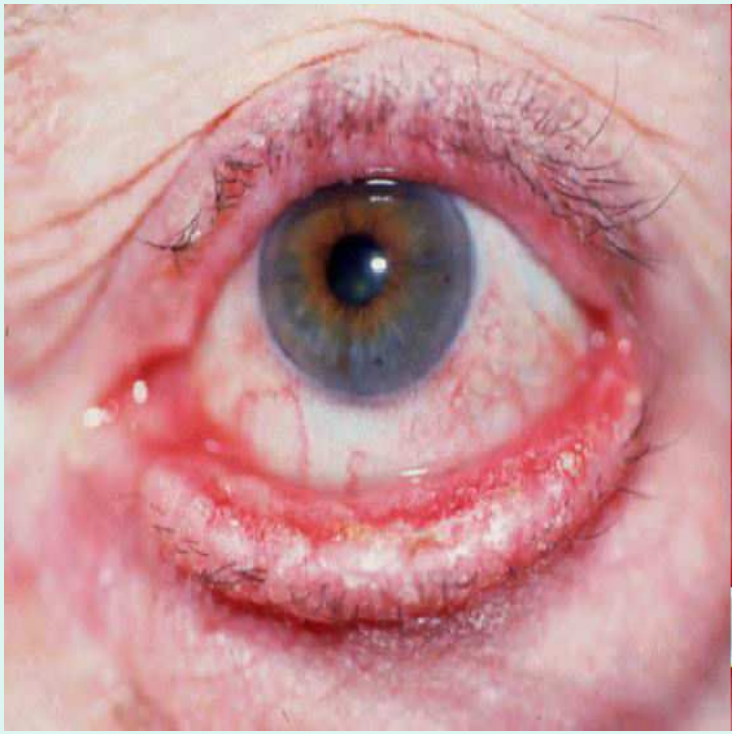
Treatment

- Resection of abnormal portion of tarsus
- Plastic reconstruction of lid crease



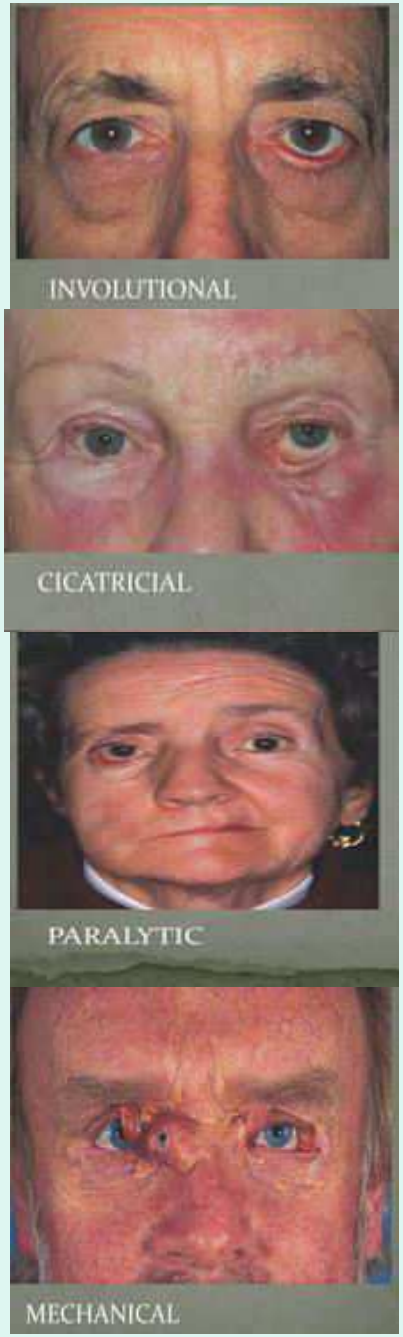
ECTROPION

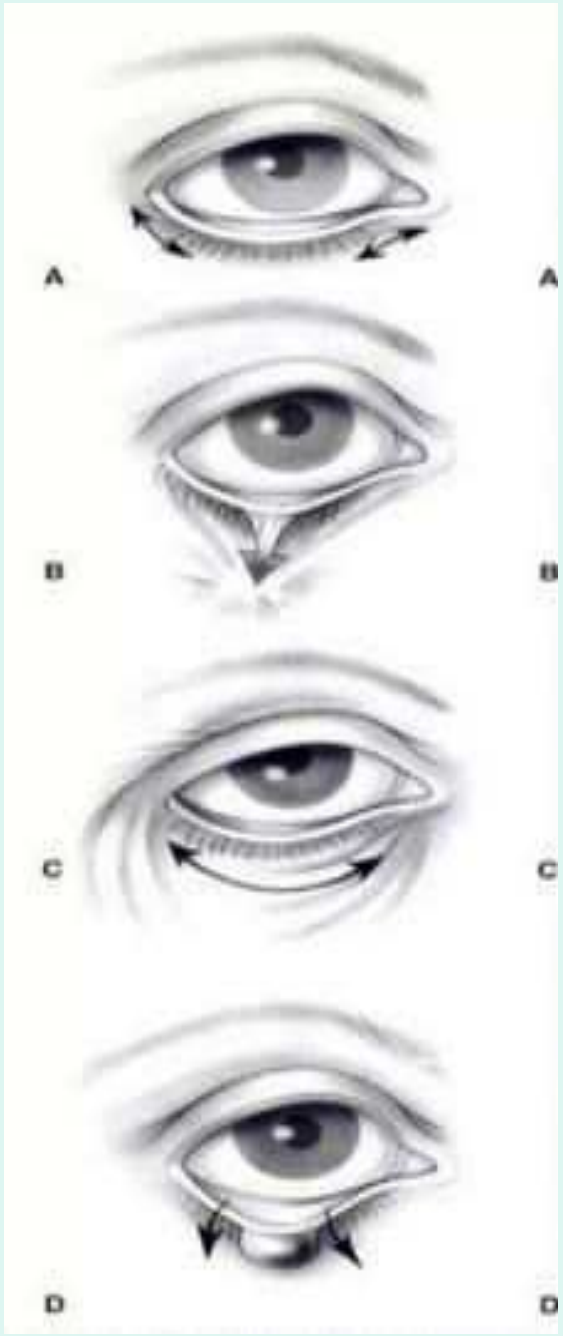
- ***It is turning outward of the eyelid margin***
- ***More common in the lower eyelid***
- ***Congenital ectropion is rare***
- ***Involutional ectropion is common among other types***



- ***TYPES***

- ***Involutional***
- ***Cicatricial***
- ***Paralytic***
- ***Mechanical***

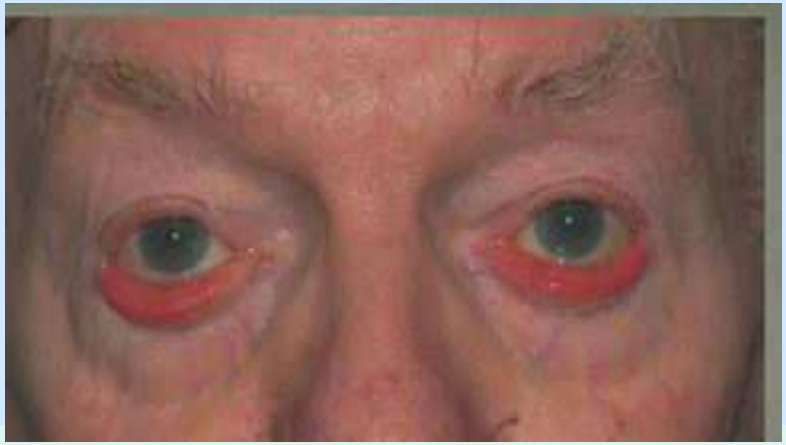




Clinical Features

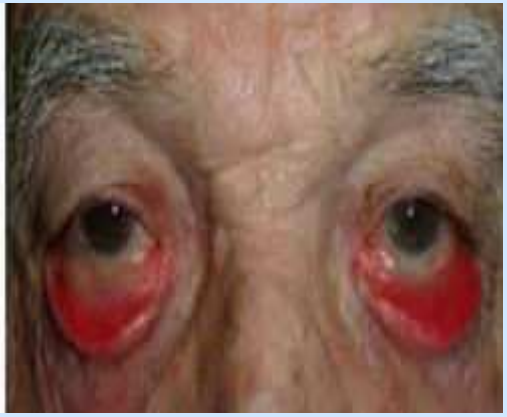
- Epiphora
- Photophobia
- Keratinization of the conjunctival epithelium
- **Corneal exposure**

Corneal dryness, FB sensation corneal ulceration, exposure



PATHOGENESIS

- ***Senile or involutional***
- *lid laxity is responsible caused by a horizontal lid laxity*
- *Lengthening of the MCT and LCT*
- *Most common type*
- *There is chronic epiphora and conjunctivitis*



Classification of Involutional Ectropion

- *Punctal ectropion*
- *Medial ectropion without horizontal lid laxity*
- *Medial ectropion with horizontal lid laxity*
- *Medial ectropion with MCT laxity*
- *Ectropion of the whole length of the eyelid*
- *Complete tarsal ectropion*

- ***Cicatricial ectropion***

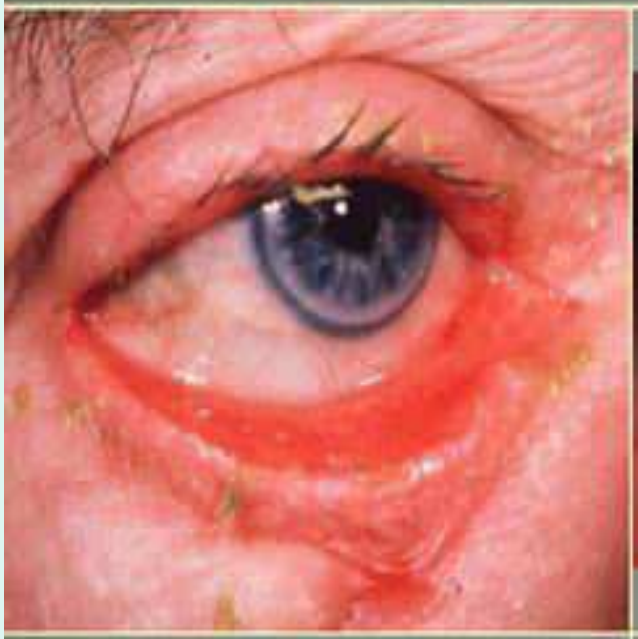
Shortening of the anterior lamella is either postoperatively, trauma(burns or injuries)

- ***Paralytic ectropion***

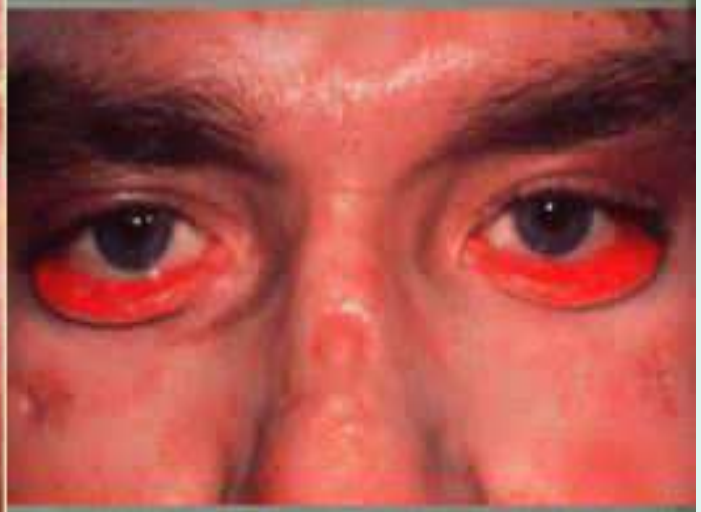
Support of the lower eyelid depends on the tone of the orbicularis and loss of this support lead to paralytic ectropion-----facial nerve palsy

Causes of cicatricial ectropion

- Contracture of skin pulling lid away from globe
- Unilateral or bilateral, depending on cause



Unilateral ectropion due to traumatic scarring



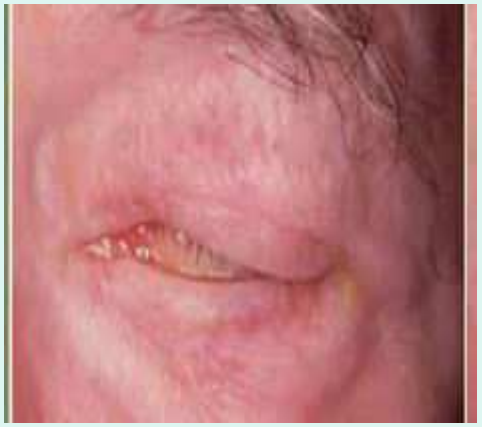
Bilateral ectropion due to severe dermatitis

PARALYTIC ECTROPION

- Caused by facial nerve palsy
- Lagophthalmos leads to exposure keratopathy
- Epiphora is caused by

  Failure of lacrimal pump

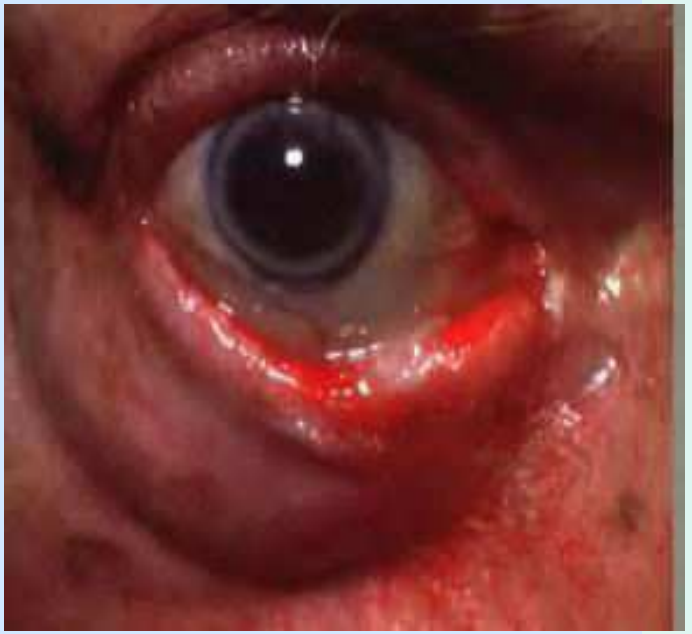
  increased tear production resulting from exposure



MECHANICAL ECTROPION

- **Mechanical lid eversion by tumor**

- **Treatment**
  removal of the cause
  correction of lid laxity

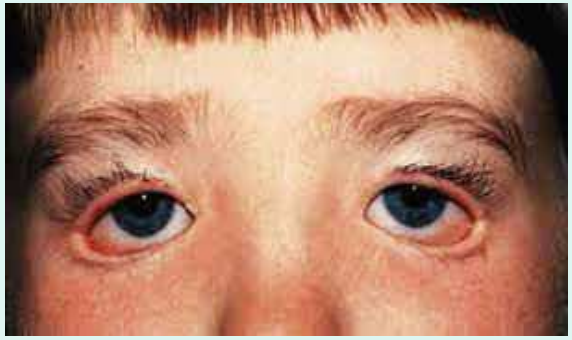


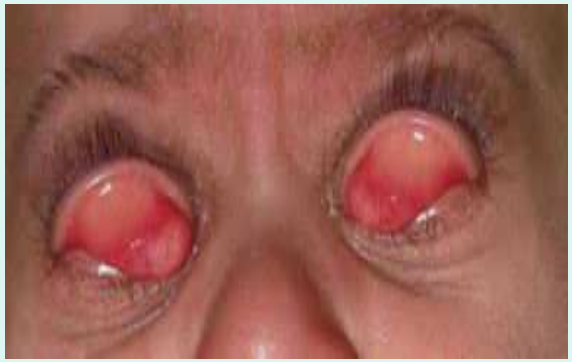
CONGENITAL ECTROPION

- **Rare**
- **Associated with other anomalies…..Euryblepheron, Blepherophimosis**

  Congenital icthyosis

  Down syndrome
- **It is caused by shortage of skin**





PATHOGENESIS

- The initial sign of a lower lid ectropion is inferior punctal eversion
- lead to a vicious cycle of secondary events
- Eversion of the inferior punctum → exposure and drying of the punctum → stenosis → Epiphora → excoriation and contracture of the skin of the lower eyelid that further exacerbates the ectropion.
- patient tends to continually wipe the tears

↓

eyelid and medial canthal tendon laxity that further exacerbates the lower eyelid ectropion.

- If the condition is neglected, the tarsal conjunctiva becomes exposed and eventually thickened and keratinized.
- Lower lid ectropion often results in a corneal epitheliopathy, especially in the inferior third of the cornea

Patient evaluation

It should be directed towards recognition of the ectropion and its severity .

1. Severity of ectropion:
    - *Mild : The lower punctum is everted*
    - *Moderate : The tarsal conjunctiva is exposed*
    - *Severe : The lower fornix is exposed*

2. Extent of ectropion: Medial or lateral or involving the entire lower eyelid.

3. Presence of any traumatic or surgical scar tissue.

4. Presence of a horizontal lid laxity. Which is demonstrated by:

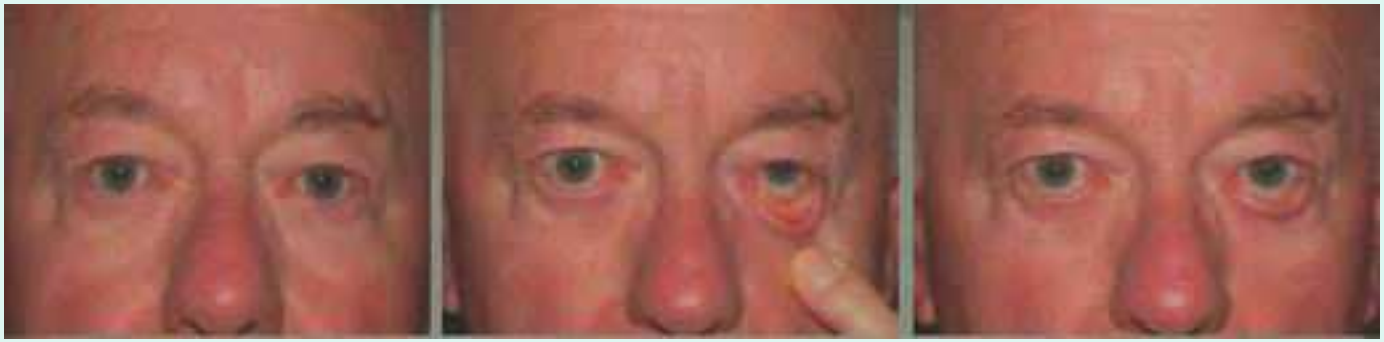
a. Eyelid snap test: Pull the eyelid inferiorly.

- If the eyelid springs to its normal position without a blink it means no lid laxity.
- If it remains away from the eye for a time; it means a lax lid. Then the degree of lid laxity will be determined by the Number of blink required to bring the lid on contact to the eye.

b. Lateral distraction test: By pulling the eyelid laterally from the eye, the punctum can be drawn lateral to medial limbus, suggest medial canthal tendon laxity

SNAP TEST

- **Positive when the eyelid fails to return to the globe without a blink**



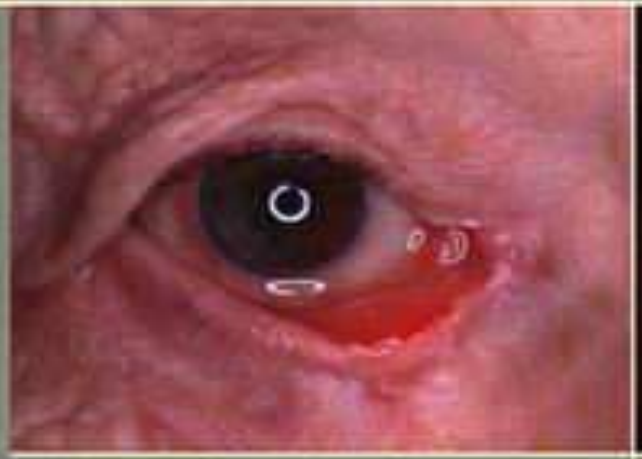
- ***Medial tendon laxity***

- 5. Signs of lower facial nerve palsy as brow ptosis, lid retraction with incomplete blink, lagophthalmos and absence of nasolabial fold.

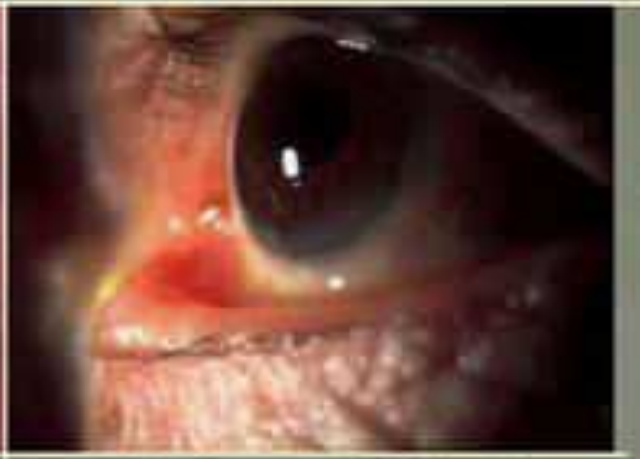
- 6. Weakness of the preseptal orbicularis oculi is tested by closure of eyelids.

- 7. Examination of corneal sensation is a must

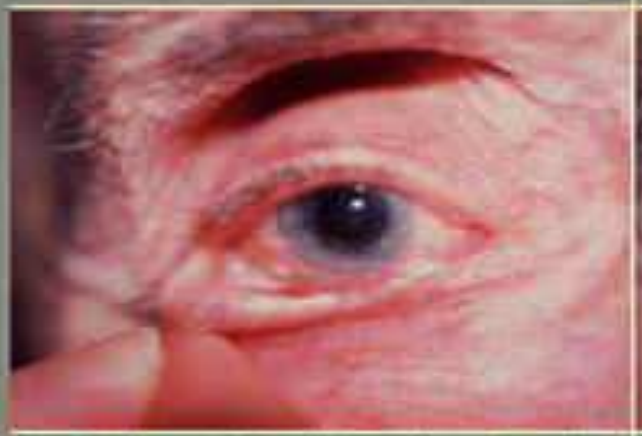
Preoperative assessment



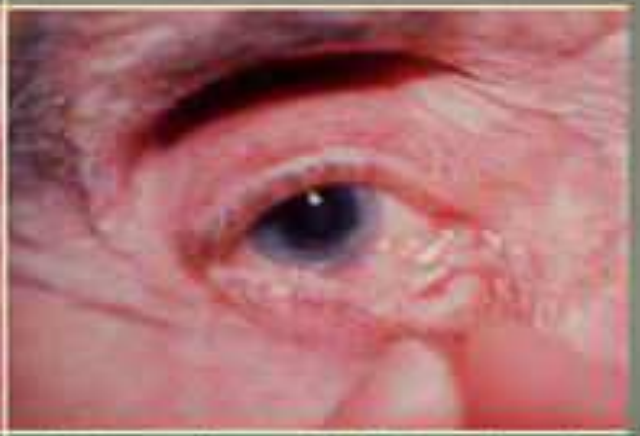
Postition of maximal ectropion



Horizontal lid laxity



Medial canthal tendon laxity



Lateral canthal tendon laxity

Retropunctal cautery

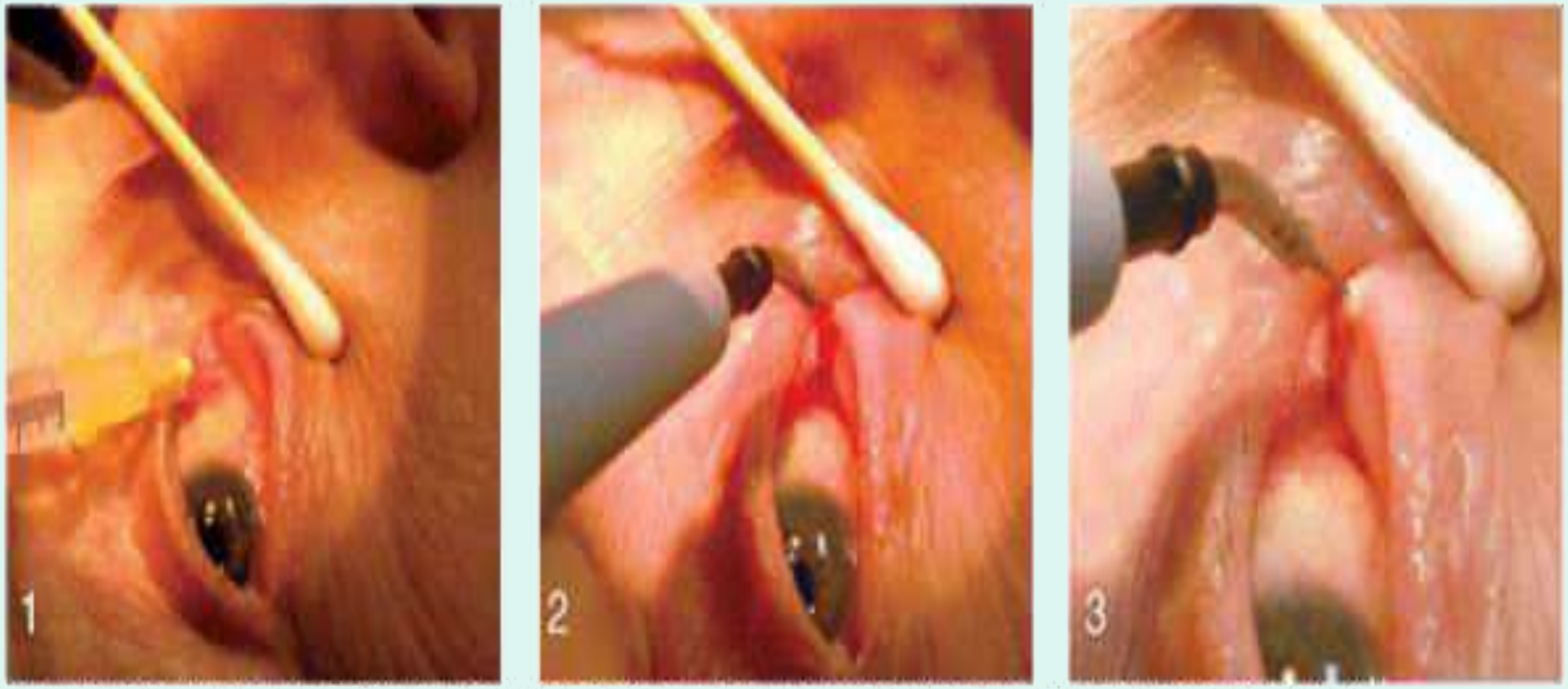


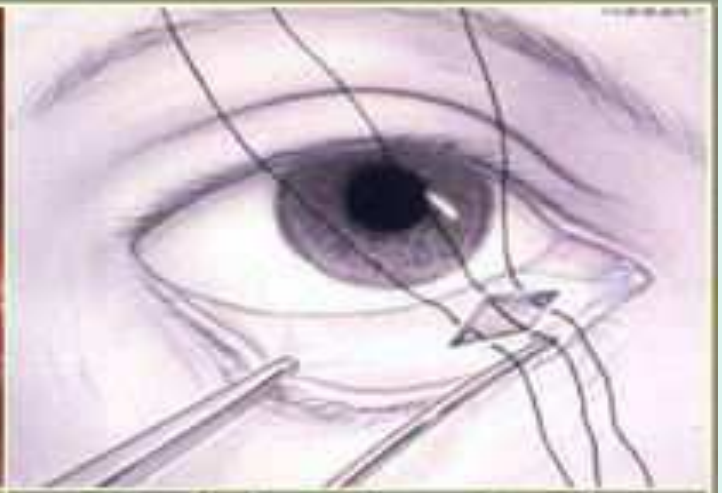
Figure 1. A 30-ga. needle with 0.4 mL of 2% lidocaine is placed 2 mm posterior to the punctum to anesthetize the plug.

Figure 2. Insert the hyfrecator tip of the cautery device deep into the punctum and horizontal canaliculus.

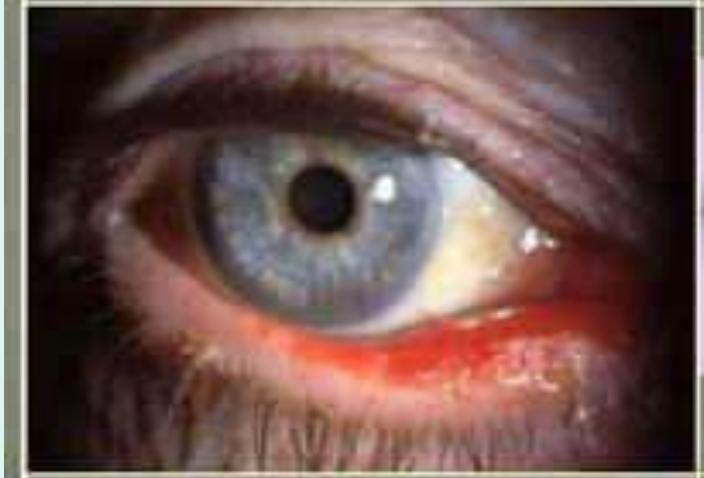
Figure 3. The endpoint of the cautery tip should produce a brisk white bubble at the punctum.

Treatment of medial ectropion

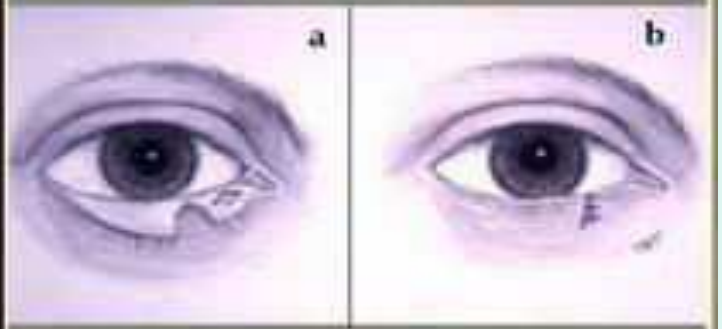
Mild



Medial conjunctivoplasty

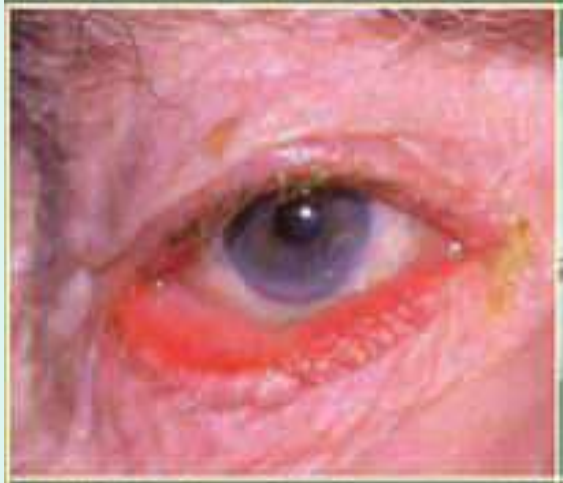


Severe

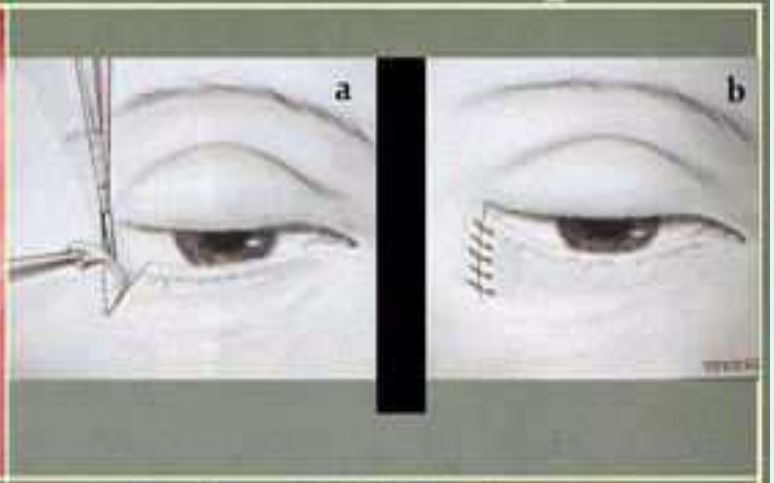


Lazy-T procedure

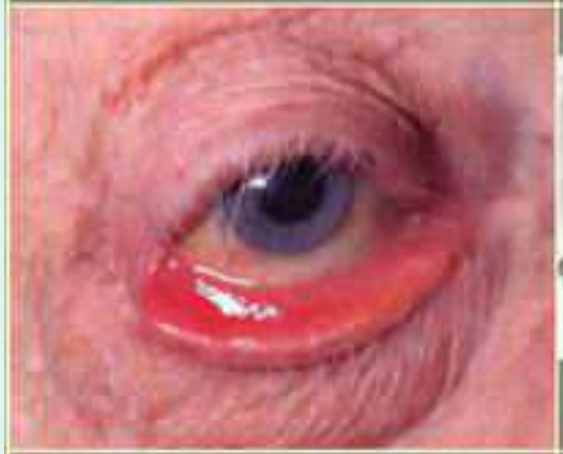
Treatment of extensive ectropion



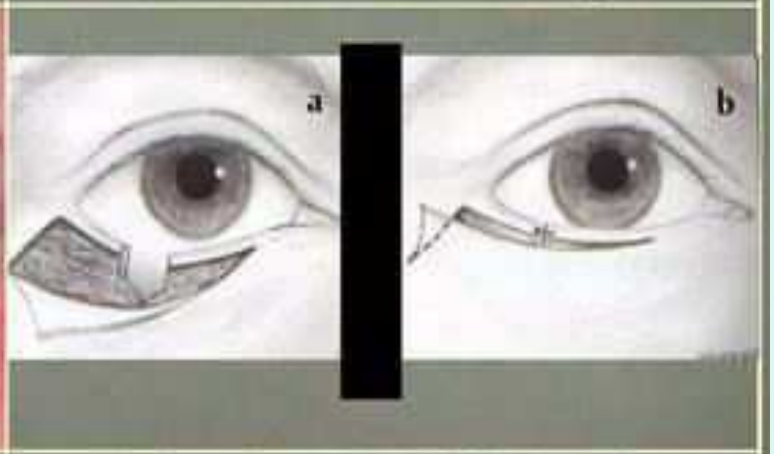
Without marked excess skin



Horizontal lid shortening



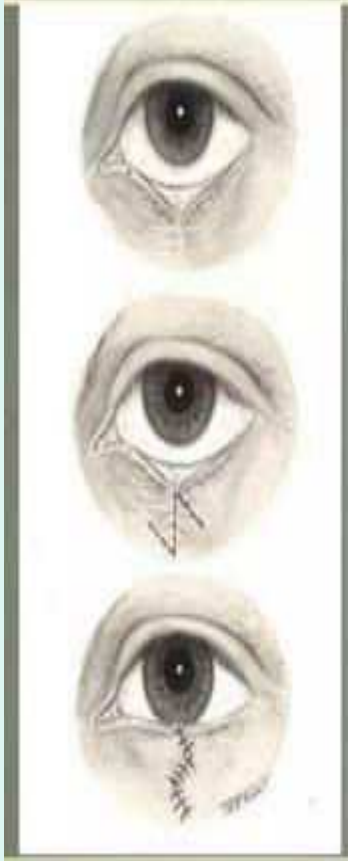
With marked excess skin



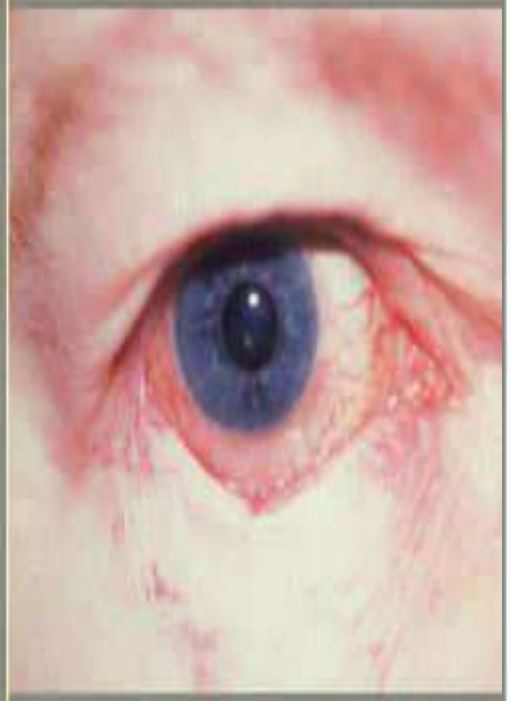
Kuhnt-Szymanowski procedure

Treatment of cicatricial ectropion

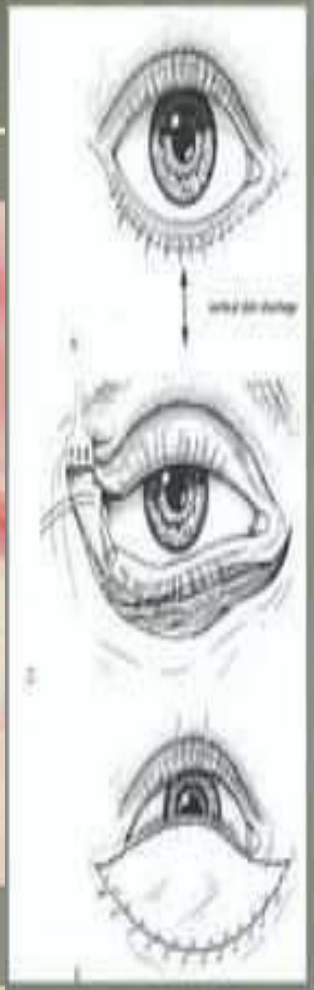
Method depends on severity



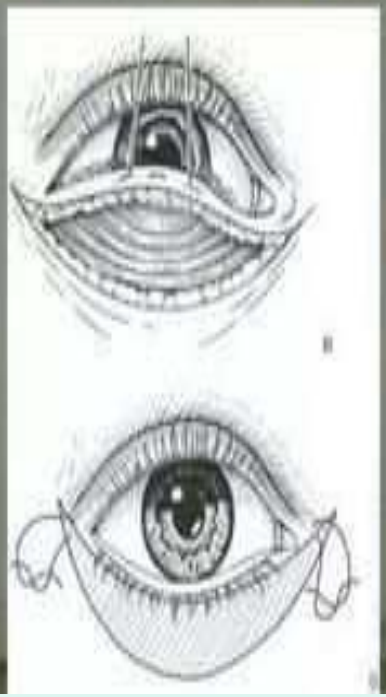
Mild localized cases are treated by excision of scar tissue combined with 'Z'-plasty



Severe cases require transposition flaps or free skin grafts



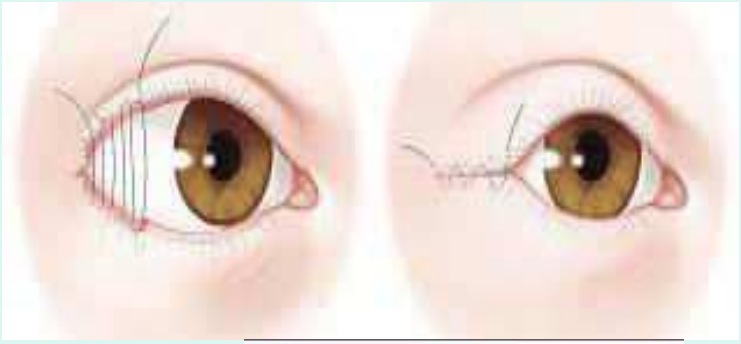
- Correction of cicatricial ectropion
  - Horizontal tightening
  - Fascial sling
  - Full thickness skin graft



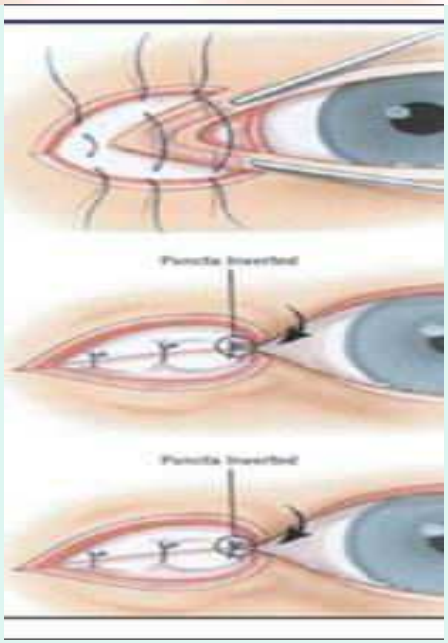
Treatment options for Paralytic Ectropion

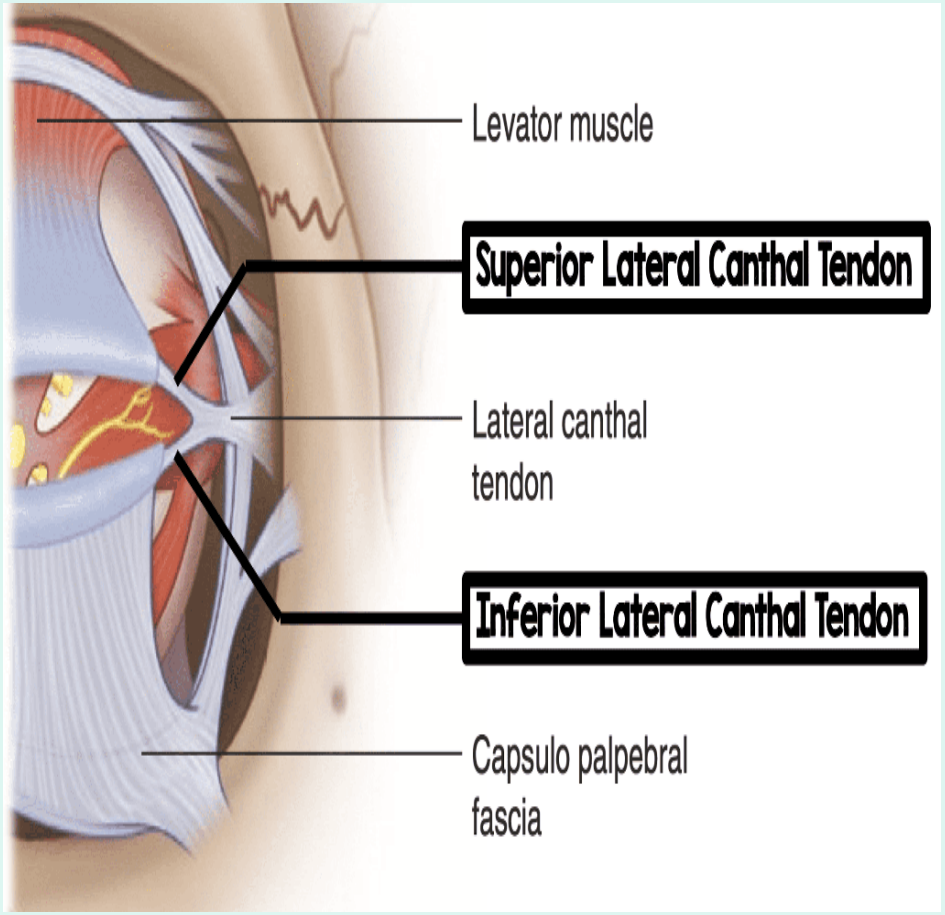
- **TEMPRORAY**
- Lubricants
- Botulinum toxin injection
- Temporary tarsorraphy in patients with poor bells phenomena
- **PERMANENT**
- Medial canthoplasty
- Medial wedge resection to treat MCT Laxity
- Lateral canthal sling to control residual ectropion

Tarsorrhaphy *is a safe and relatively simple procedure in which part, or all the upper and lower eyelids are joined together to cover the eye partially or completely*

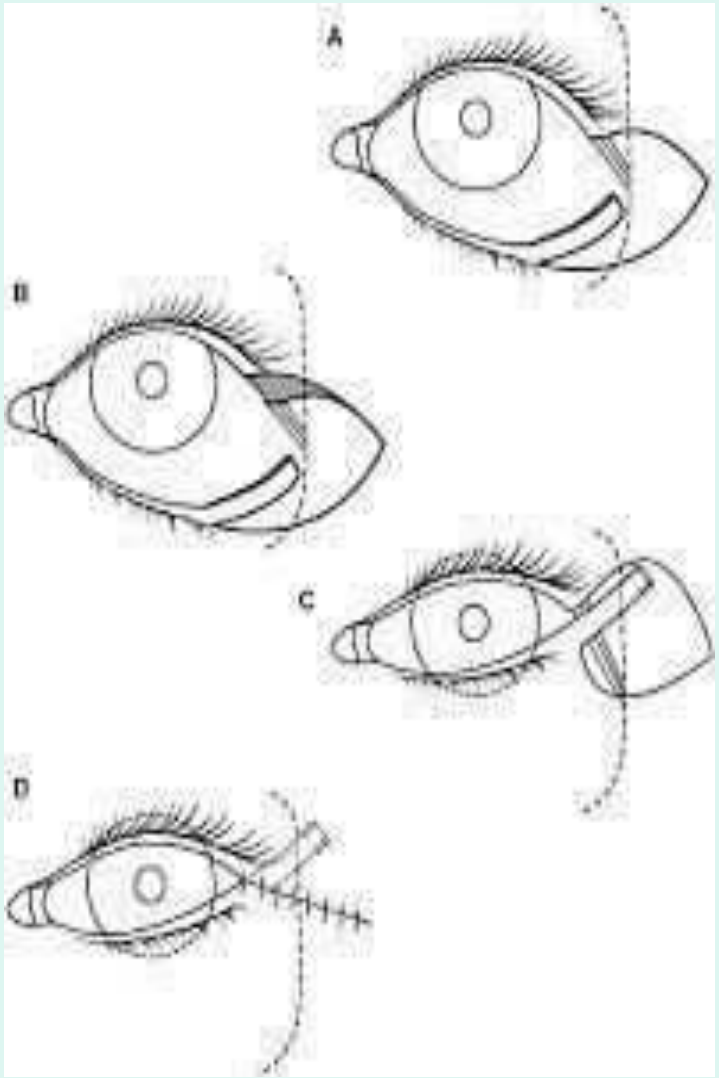


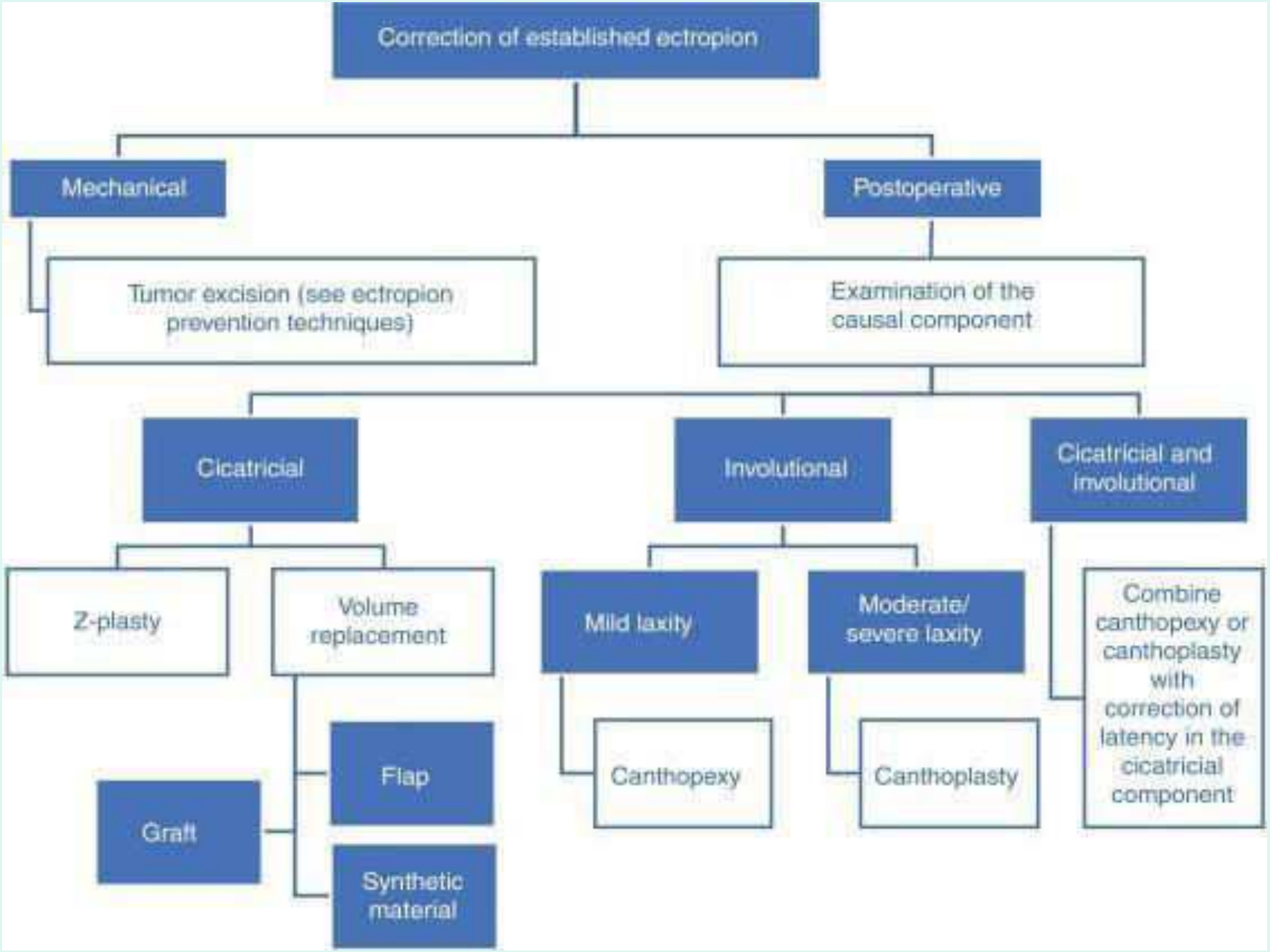
Medial canthoplasty





Lateral canthal sling



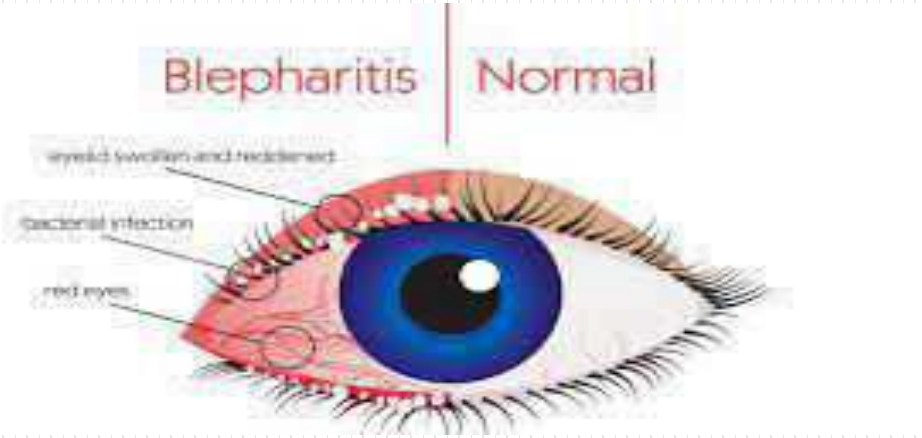

Correction of established ectropion
Mechanical
Postoperative
Tumor excision (see ectropion prevention techniques)
Examination of the causal component
Cicatricial
Involutional
Cicatricial and involutional
Z-plasty
Volume replacement
Mild laxity
Moderate/ severe laxity
Combine canthopexy or canthoplasty with correction of latency in the cicatricial component
Flap
Graft
Synthetic material
Canthopexy
Canthoplasty

Bibilography

- Kanski clinical opthalmology 6th edition
- Parsons diseases of eye 21st edition
- A K Khurana opthalmology 4th edition
- Collins occuloplasty


Thank
you!

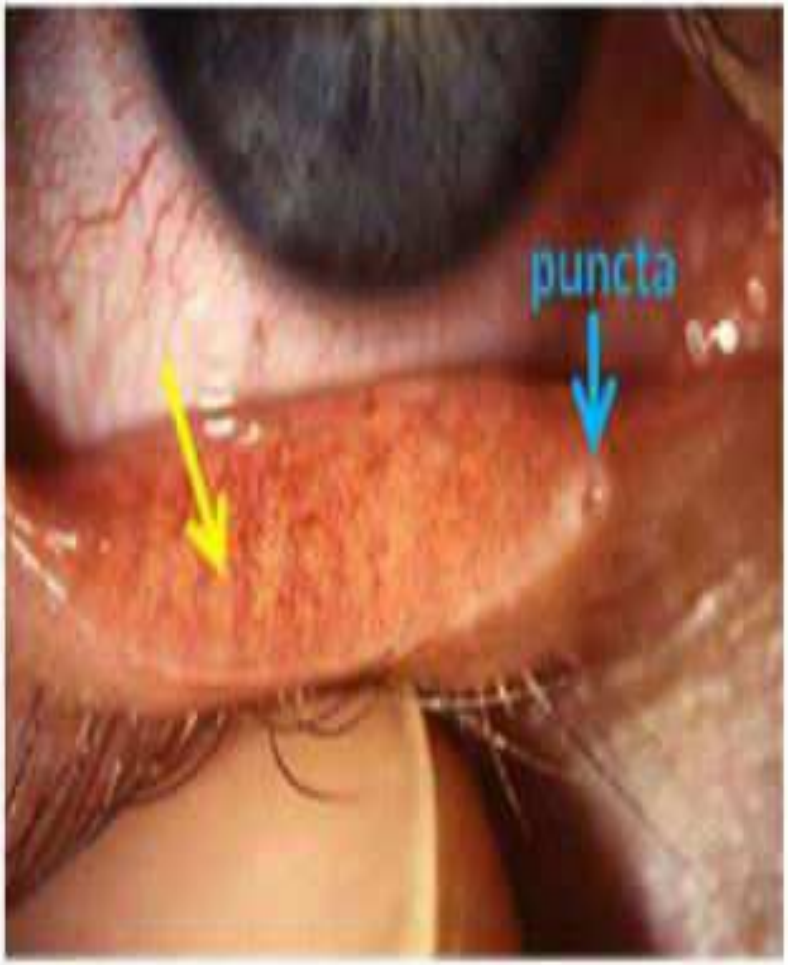
BLEPHRITIS
CHALAZION
ABORMALITIES OF EYELASHES

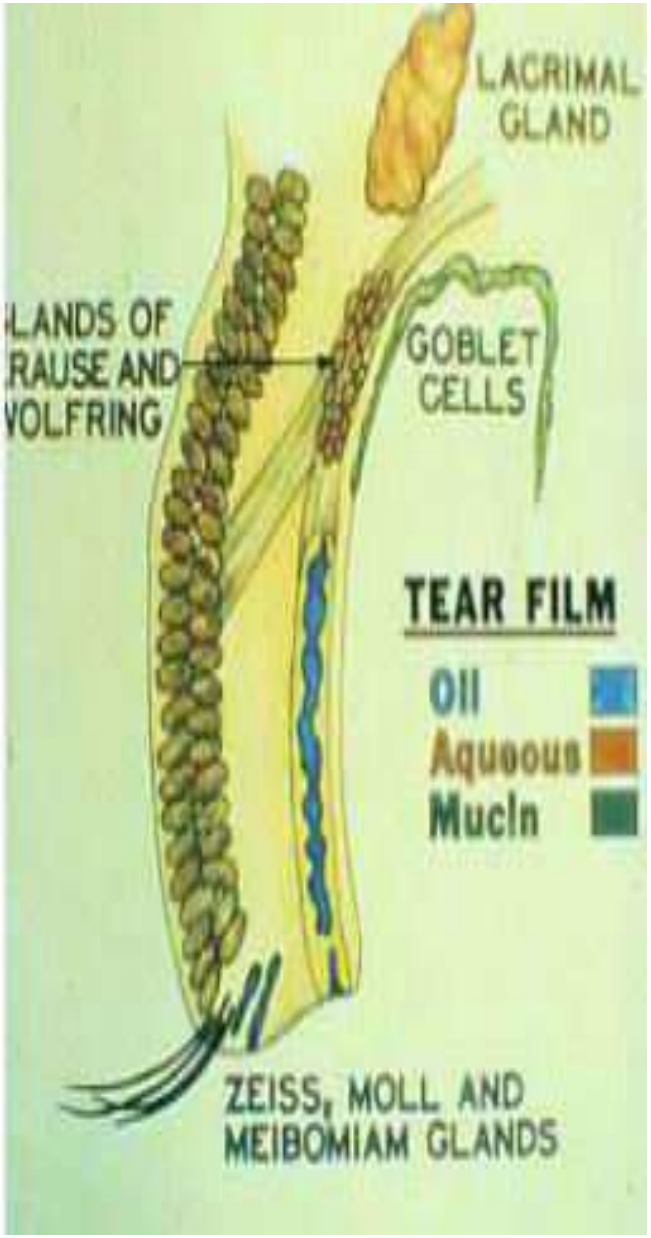


Prof SofiaIqbal
FRCS, MRCOphth
Fellowship Orbit/Oculoplastics
Fellowship Refractive surgery

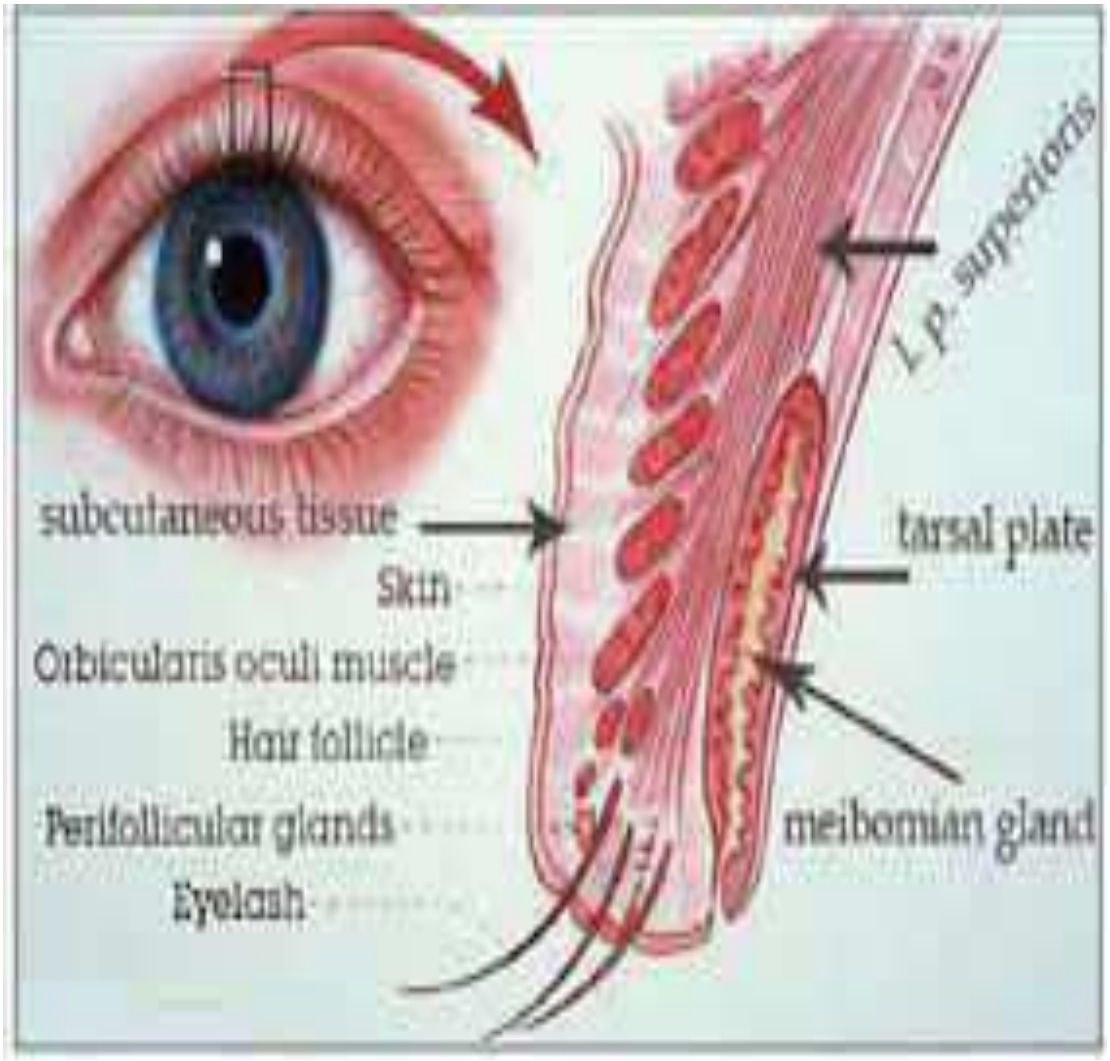
Presentation lay out

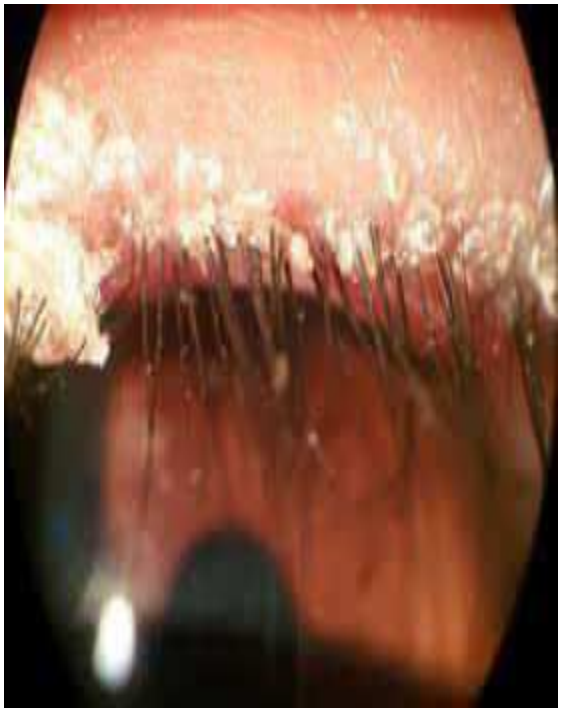
- Lid anatomy
- Definition, etiology, types, clinical features, complications and treatment of blepharitis
- Infection of eye lid margins
- Chalazion
- Eye lash abnormalities


puncta

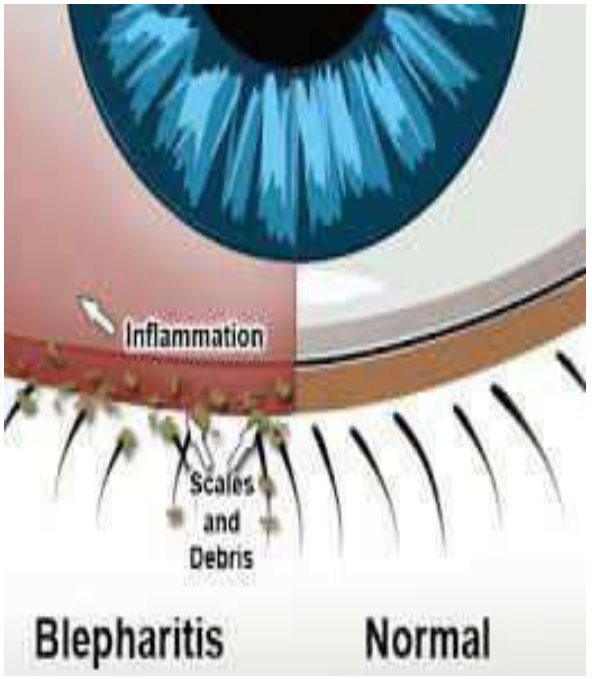

LACRIMAL GLAND
LANDS OF
RAUSE AND
VOLFRING
GOBLET CELLS
TEAR FILM
Oil
Aqueous
Mucin
ZEISS, MOLL AND
MEIBOMIAM GLANDS

BLEPHRITIS


L.P. superioris
subcutaneous tissue
Skin
Orbicularis oculi muscle
Hair follicle
Perifollicular glands
Eyelash
tarsal plate
meibomian gland



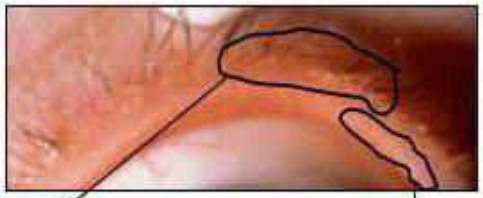
INTRODUCTION



- **Blepharitis** is inflammation or infection of the eyelid margins .

- **Blepharitis** is one the most common ophthalmological complications as well as one of the most difficult conditions to treat.

Blepharitis is a common eyelid inflammation that sometimes is associated with a bacterial eye infection, symptoms of dry eyes or certain types of skin conditions such as acne rosacea.



BLEPHARITIS

Anterior Blepharitis

- **Bacterial** (Staphilococcal)
- **Seborrhehic**

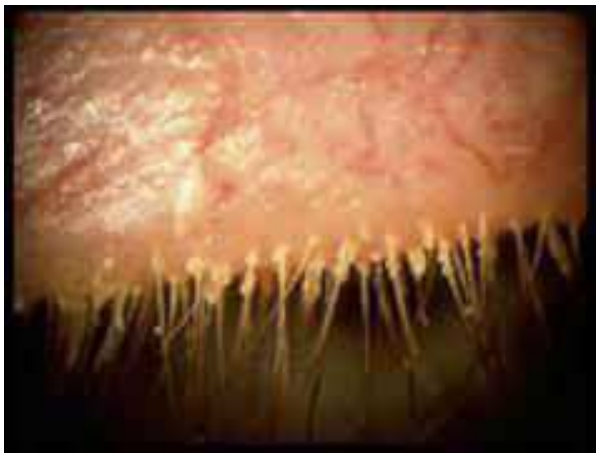
Posterior Blepharitis

- **Meibomian Gland Dysfunction**

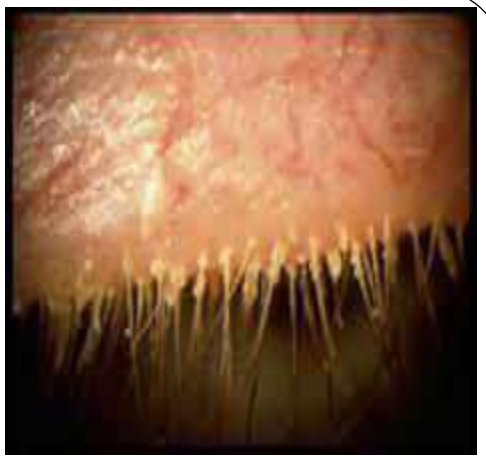
- **Eye Lid Irritation**
- **Chronic Red Eye**
- **Dry Eye**

Classification

- *Anterior*
  - *Staphylococcal*
  - *Seborrhoeic*
  - *Mixed*
- *Posterior*
  - *Meibomian seborrhoea*
  - *Meibominitis*
- *Mixed*



ANTERIOR BLEPHRITIS



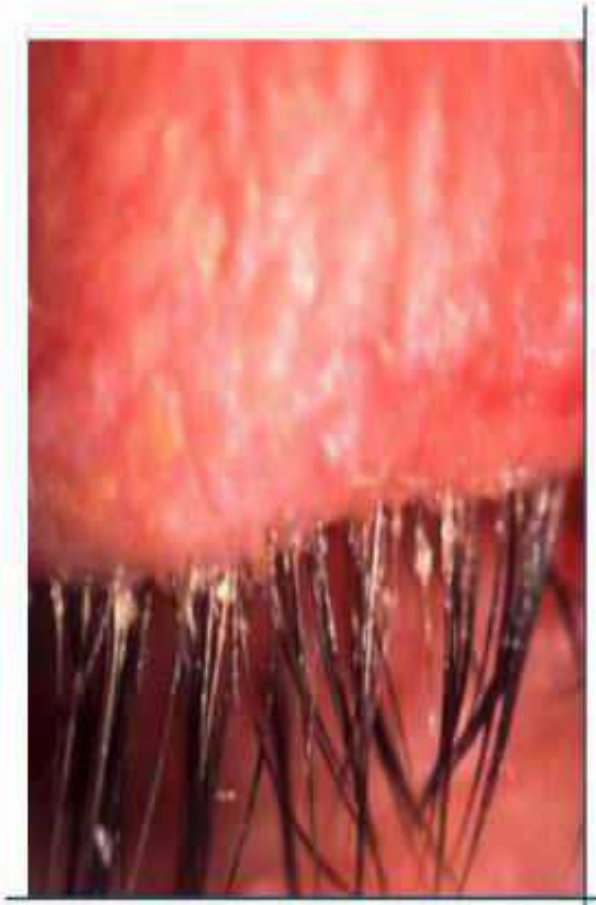
Anterior blepharitis is characterized by inflammation at the base of the eyelashes .

Two variants of anterior blepharitis are identified: staphylococcal and seborrheic.

- *In staphylococcal anterior blepharitis, colonization of the eyelids by staphylococci leads to formation of fibrinous scales and crust around the eyelashes.*

- *The seborrheic variant is characterized by dandruff-like skin changes around the base of the eyelids, resulting in greasy scales around the eyelashes.*

Staphylococcal blepharitis



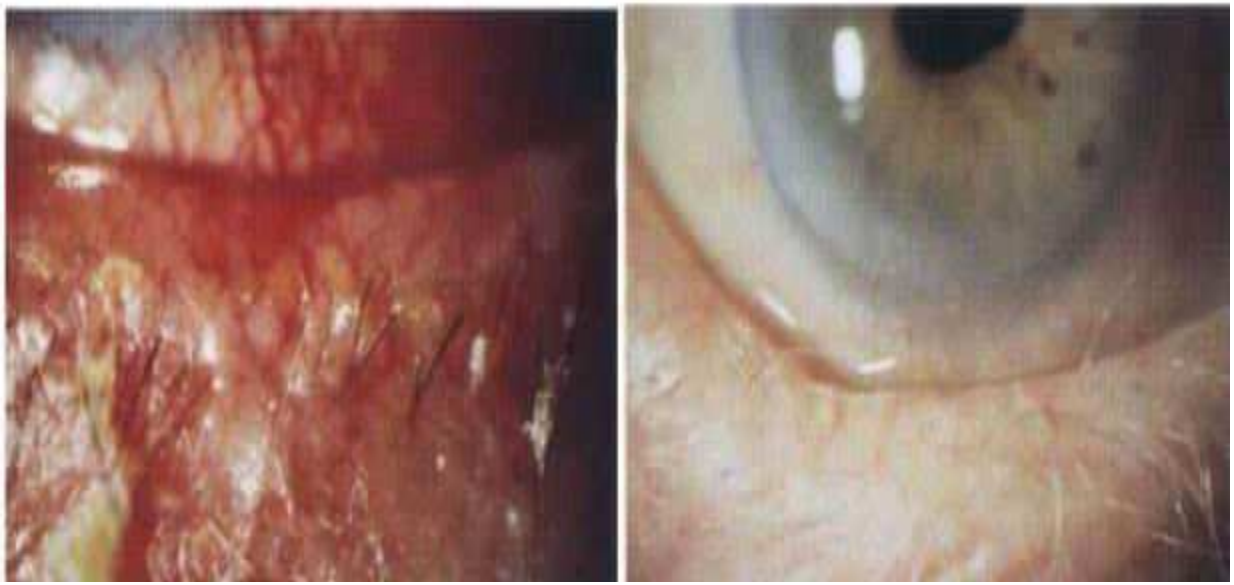
Chronic irritation worse in morning.

Scales around base of lashes . (collarettes)

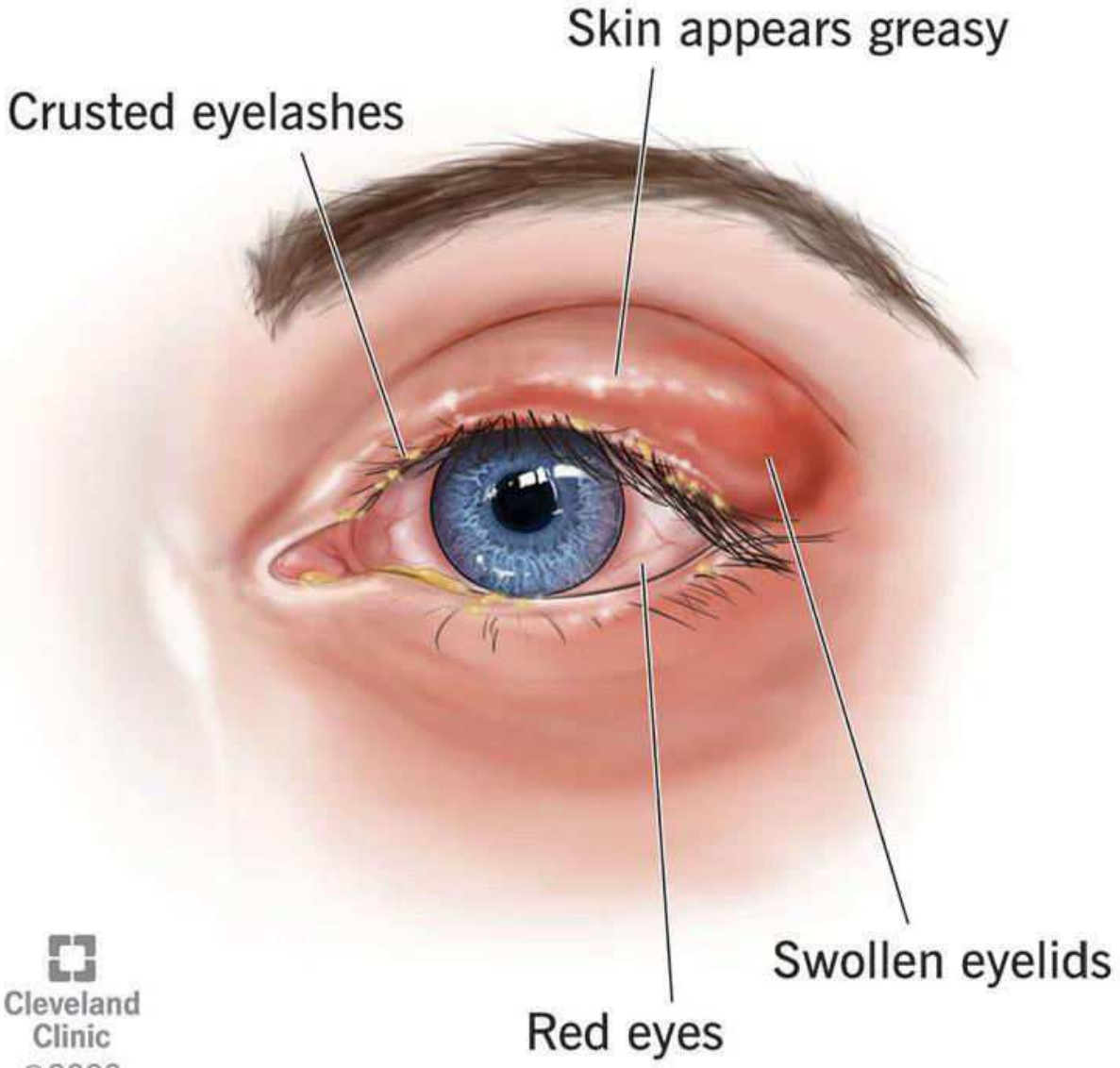
Hyperaemia and telangiectasia of anterior lid margin

Scarring and hypertrophy if . longstanding

Cont. Signs of Staphylococcal blepharitis



Blepharitis

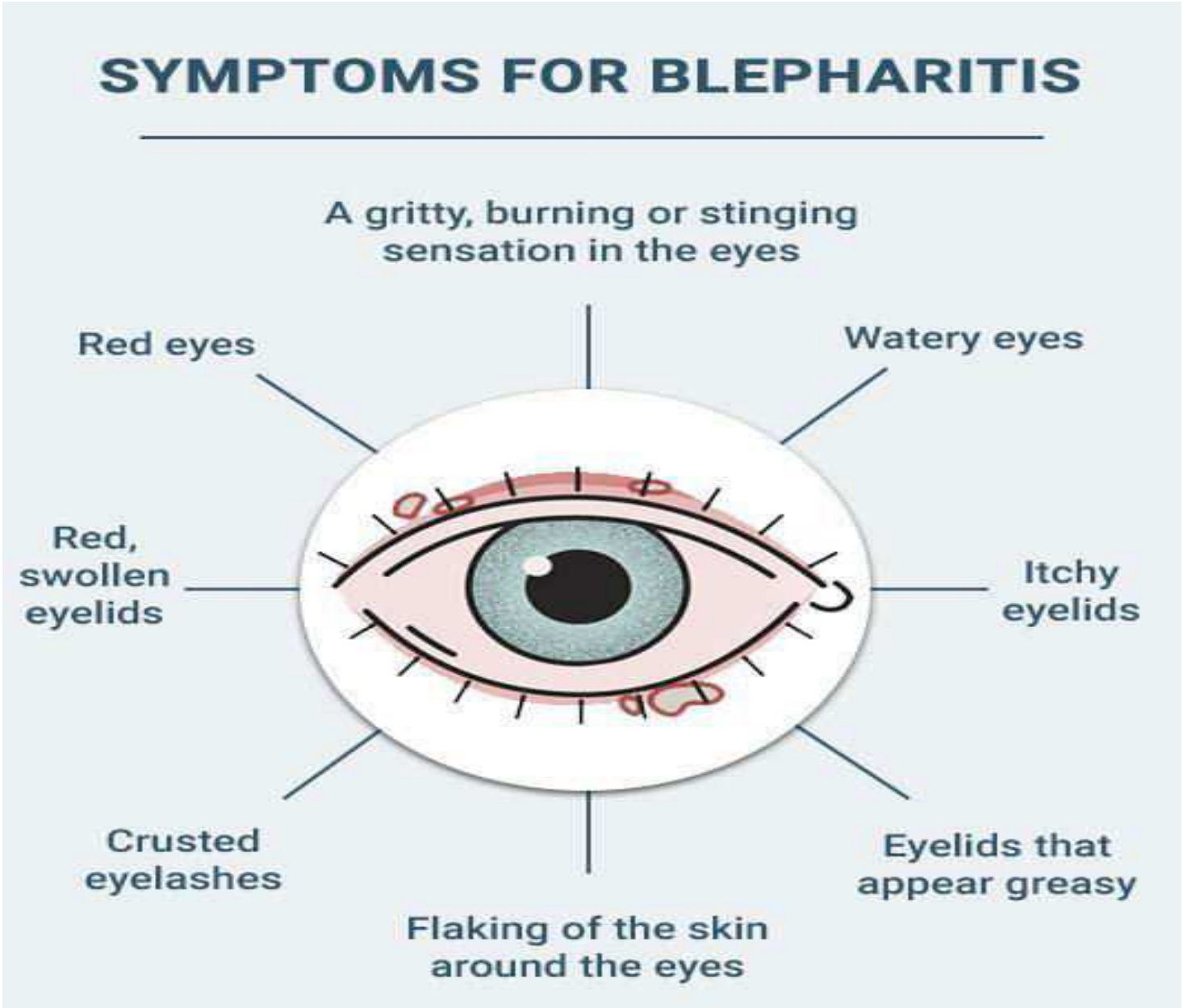


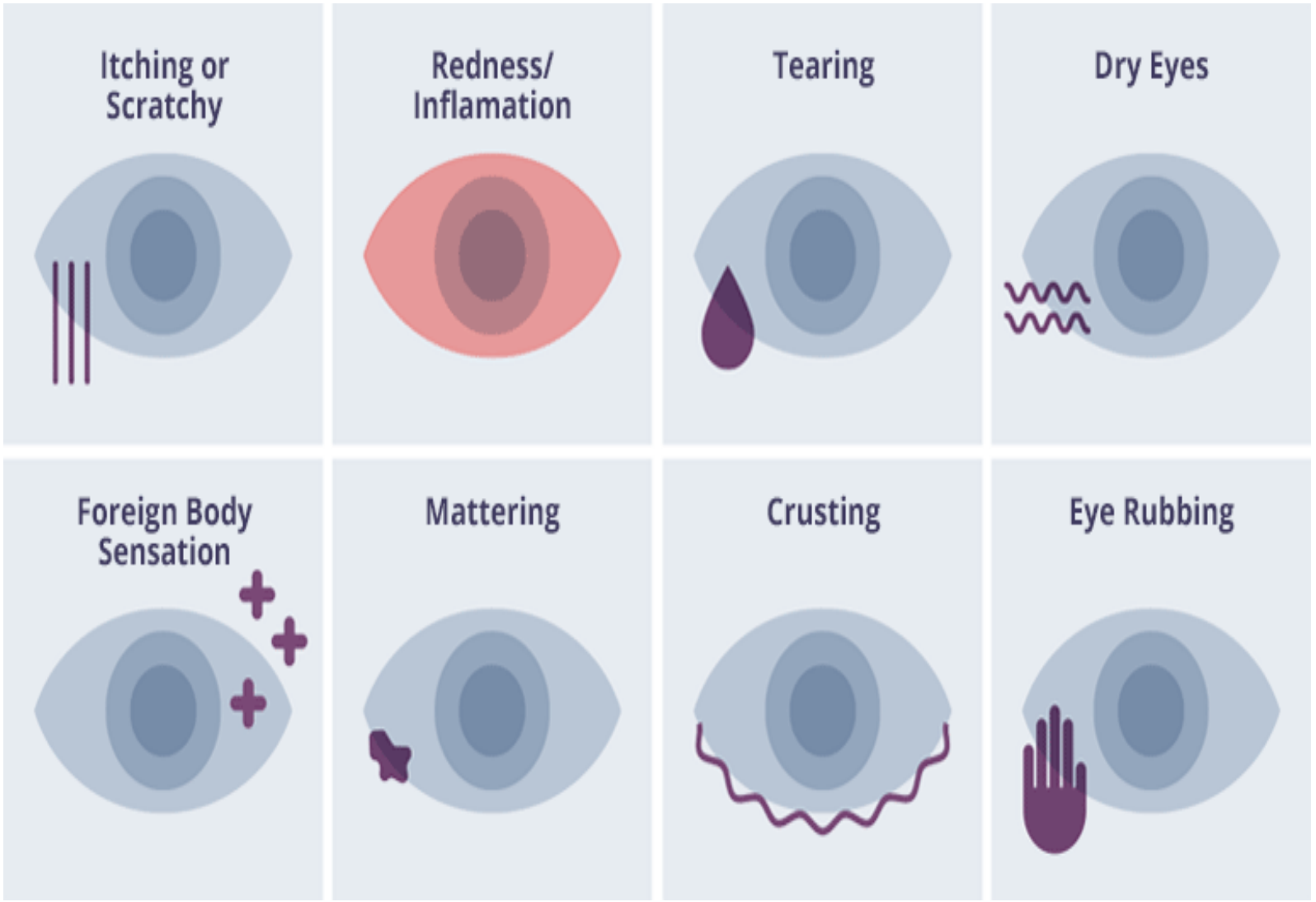
PATHOPHYSIOLOGY

The pathophysiology of blepharitis is not completely understood. A role for lid-colonizing staphylococcal bacteria was first noted in 1946 . Several mechanisms by which staphylococci may alter meibomian gland secretion and cause blepharitis are supported by many studies .

- Direct infection of the lids
- Evoke reaction to staphylococcal exotoxin
- Provoke allergic response to staphylococcal antigens .
-

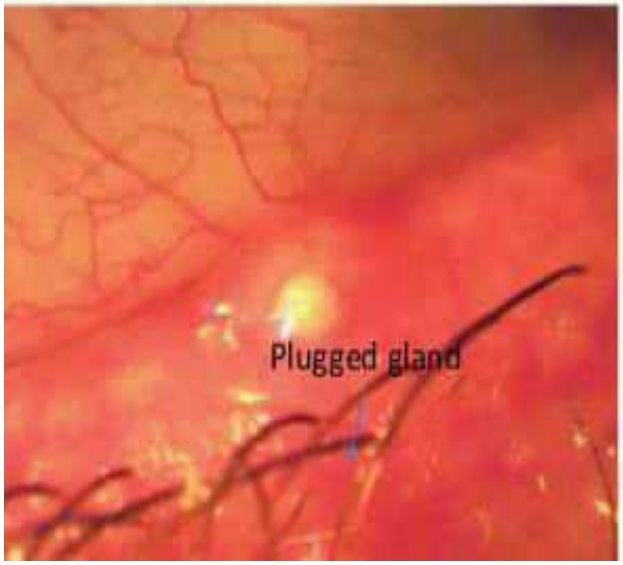
It is likely that a combination of these is responsible for the clinical manifestations of staphylococcal blepharitis.

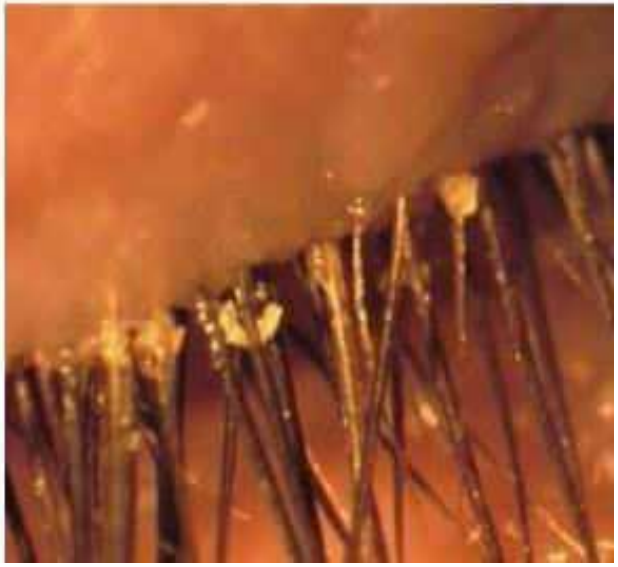

SYMPTOMS FOR BLEPHARITIS
A gritty, burning or stinging sensation in the eyes
Red eyes
Watery eyes
Red, swollen eyelids
Itchy eyelids
Crusted eyelashes
Eyelids that appear greasy
Flaking of the skin around the eyes


Itching or Scratchy
Redness/ Inflamation
Tearing
Dry Eyes
Foreign Body Sensation
Mattering
Crusting
Eye Rubbing

SLIT LAMP FEATURES

The lashes should be examined for abnormalities such blepharitis. With blepharitis there will be collarettes found at the base of the lashes.



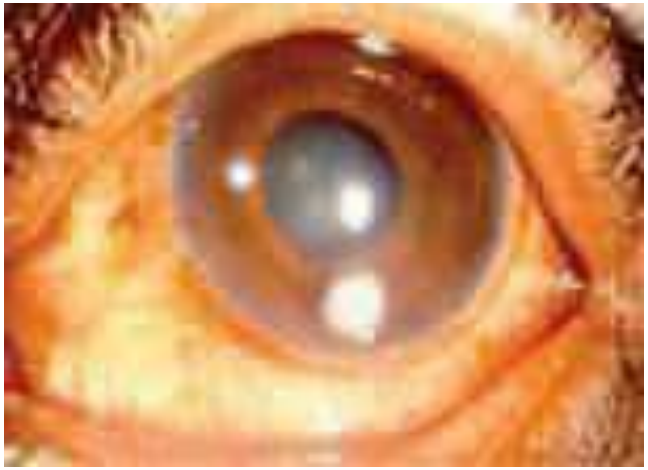


ASSOCIATIONS

secondary changes include :

- *stye formation*
- *marginal keratitis and occasionally phlyctenulosis (Corneal nodulesthat developed near the limbus and then spread onto the cornea, carrying behind them a leash of vessels)*

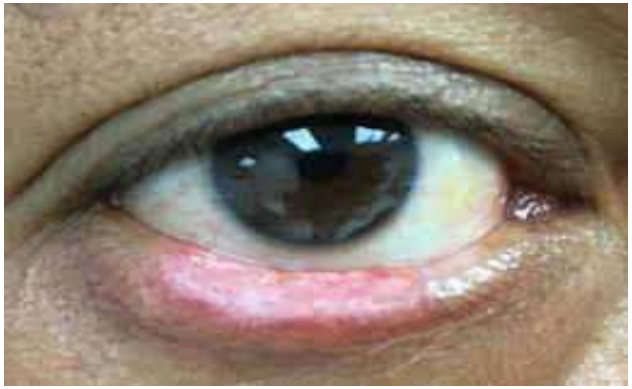
- *associated with tear film instability and dry eye*



Complications

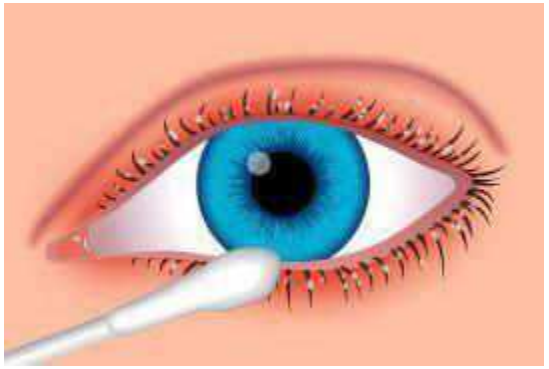
- Trichiasis
- Poliosis
- Madarosis
- Tylosis
- Epiphora
- tear film instability _ dry eyes
- Marginal keratitis
- Recurrent styes
- Conjunctivitis
- Chalazion

COMPLICATIONS



Treatment

- Lid hygiene
- Tear film substitutes
- Antibiotic ointment
- Weak topical steroids

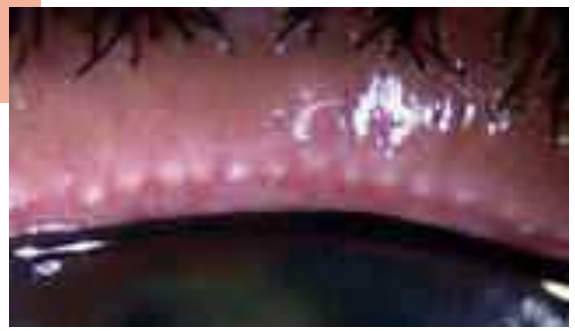


Lid hygiene

- Wash hands, clean fingertips with Sterilid prior to use, rinse.
- Pump Sterilid foam onto clean fingertips.
- Close eyes and gently massage foam onto lids and lashes.
- Avoid touching eyes directly.
- Leave in place for 60 seconds for maximum effectiveness

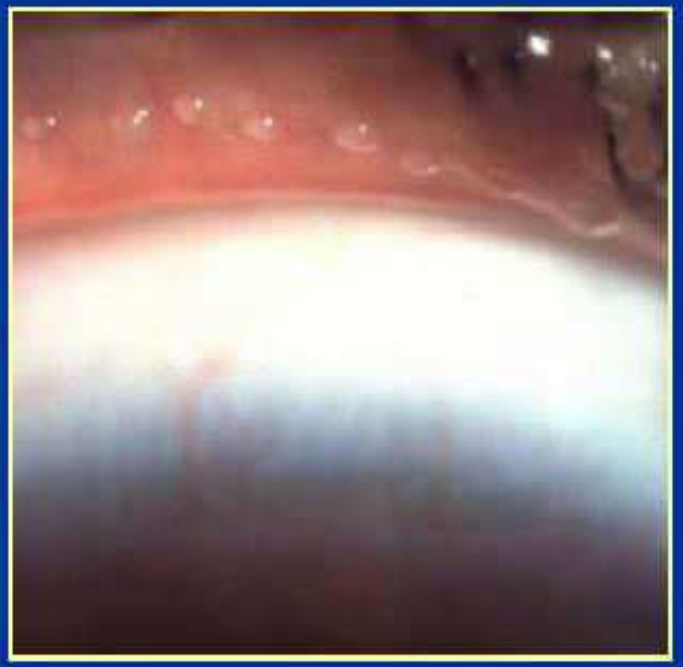
Posterior blephritis

- Meibomian seborrhoea
- Meibomianitis
- Obstruction of orifices

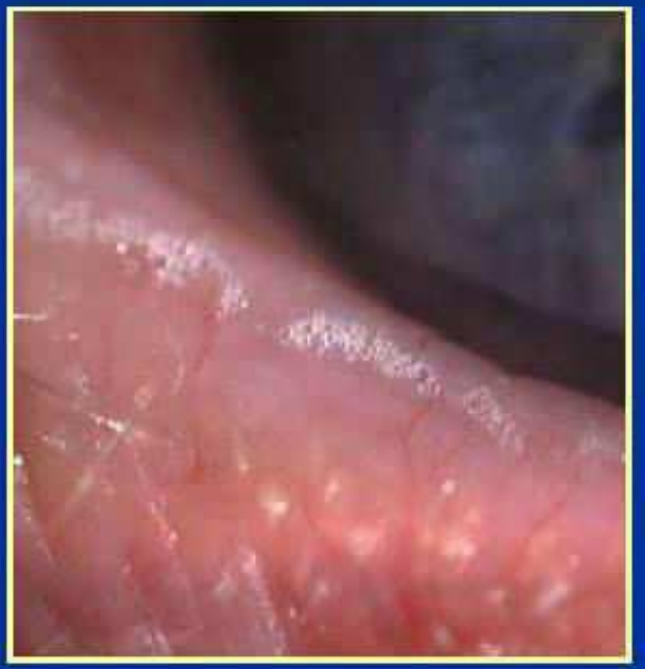


Posterior blepharitis

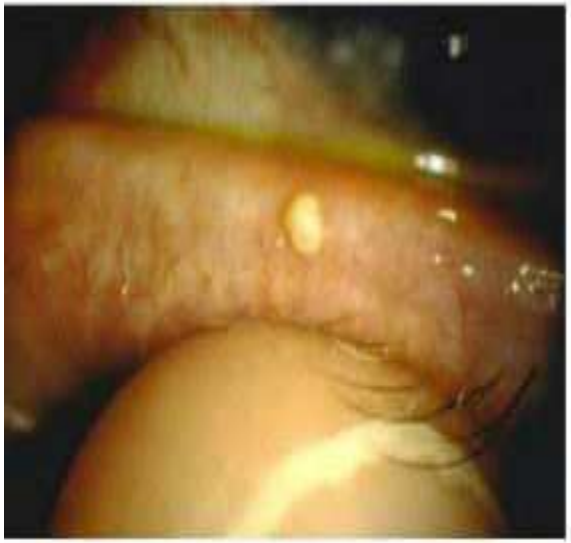
1.Meibomian gland dysfunction

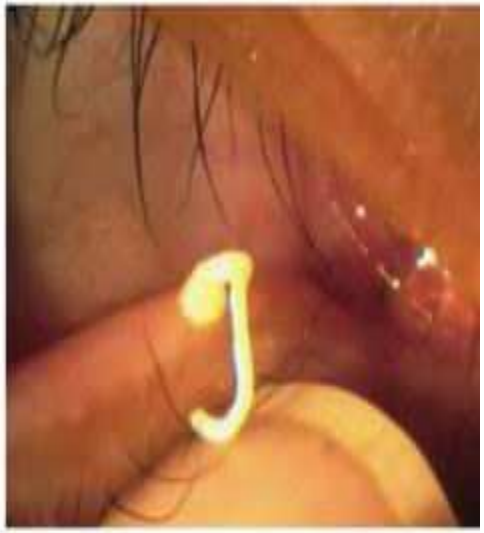


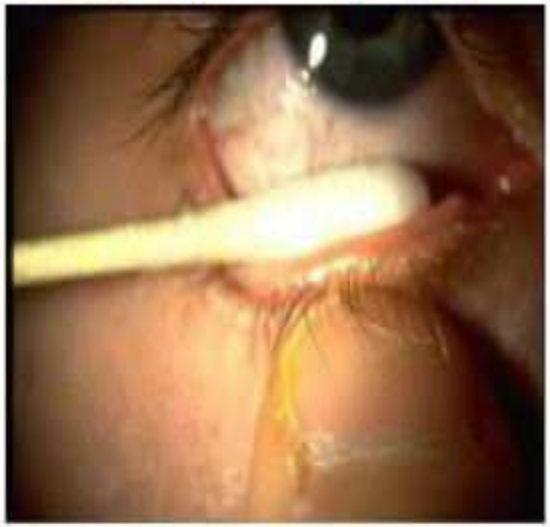
- Oil globules over meibomian gland orifices



- Oily and foamy tear film







Complications of Posterior Blephritis

- Chalazion formation

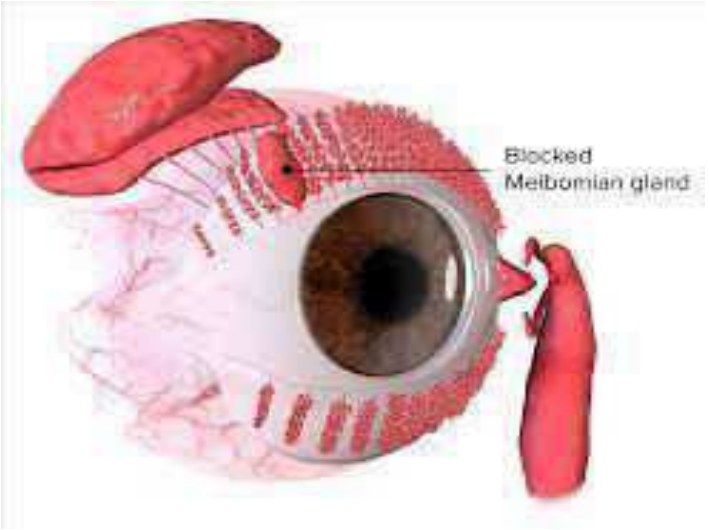
- Tear film instability
- papillary conjunctivitis
- inferior corneal epithelial erosions
- marginal keratitis

Treatment

- **Systemic tetracyclines**
- **Azithromycin or erythromycin**
- **Lid hygiene**
- **Warm compresses**
- **Tear substitutes**
- **Weak steroids**

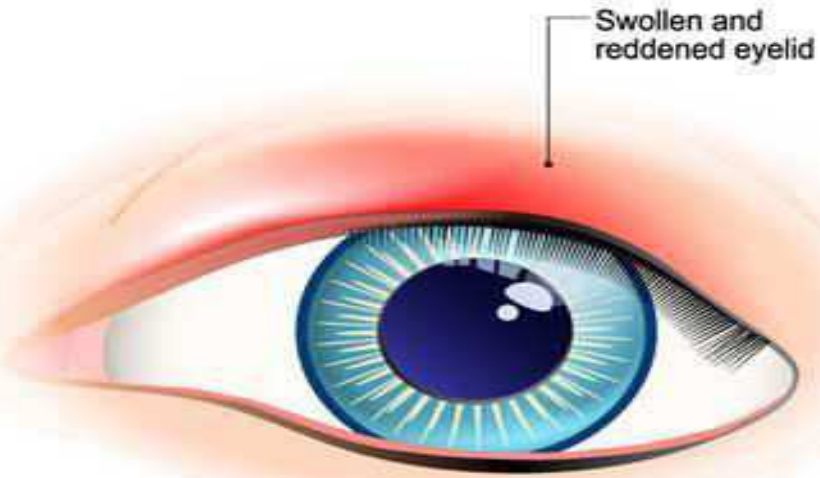
CHALAZION

- Chronic, sterile, lipogranulomatous inflammatory lesion caused by blockage of meibomian gland orifices and stagnation of sebaceous secretions
- Meibomian cyst
- Painless ,round nodule

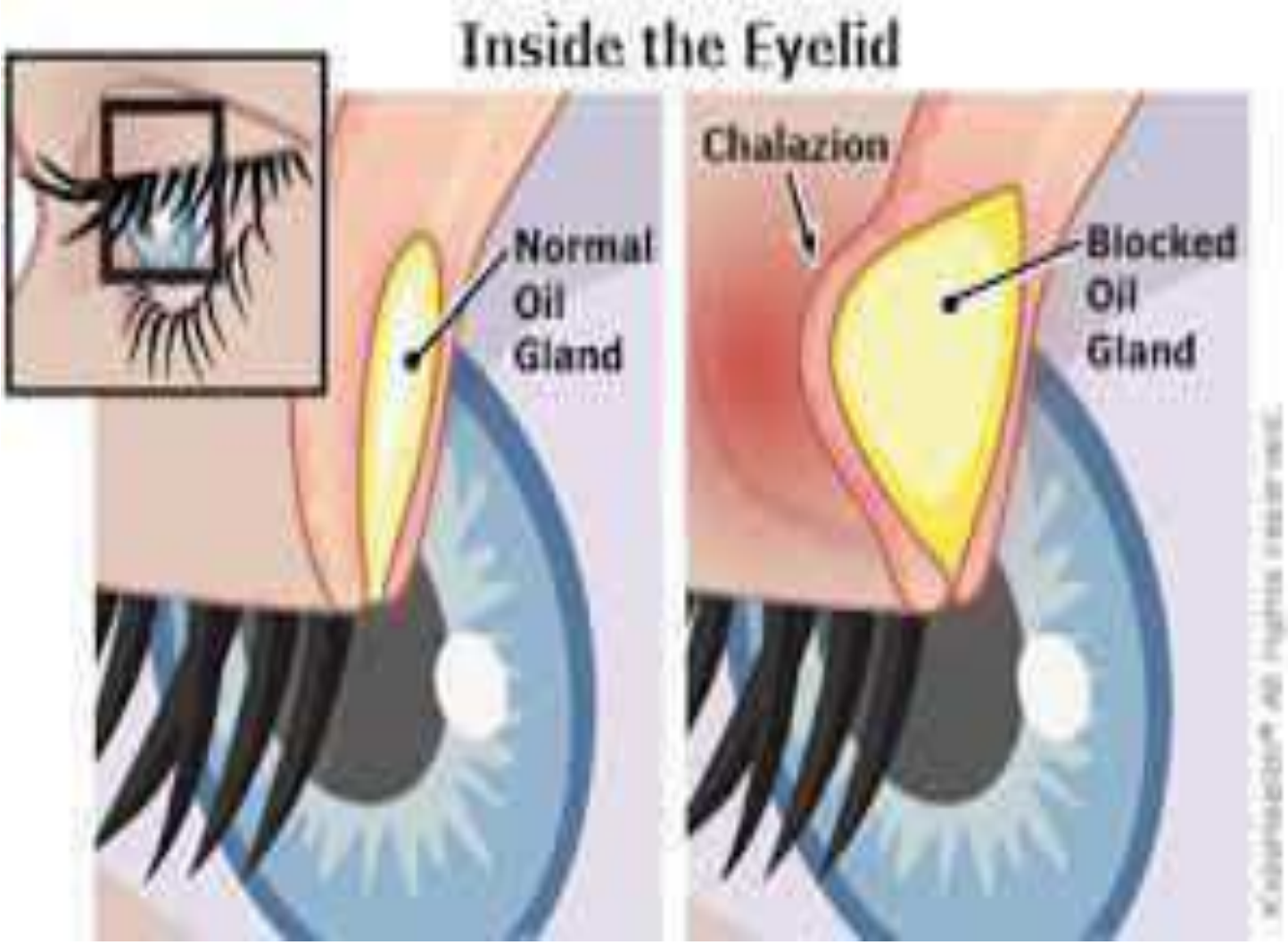


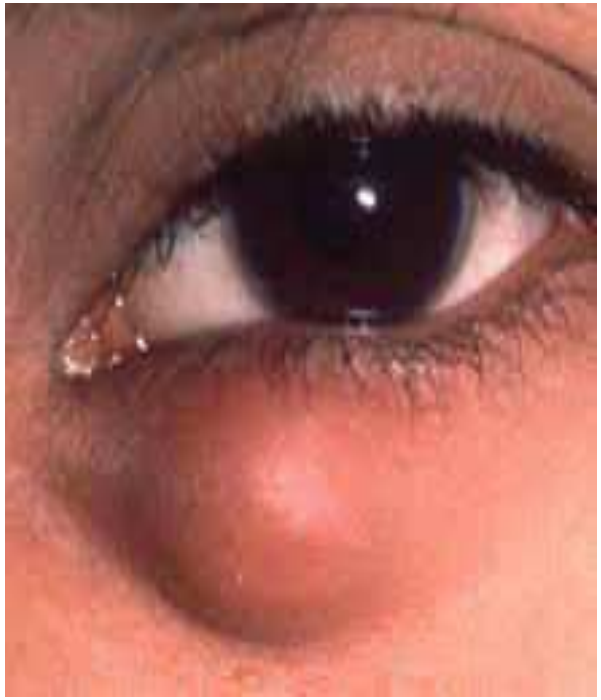
- May press on cornea and can cause astigmatism and blurred vision

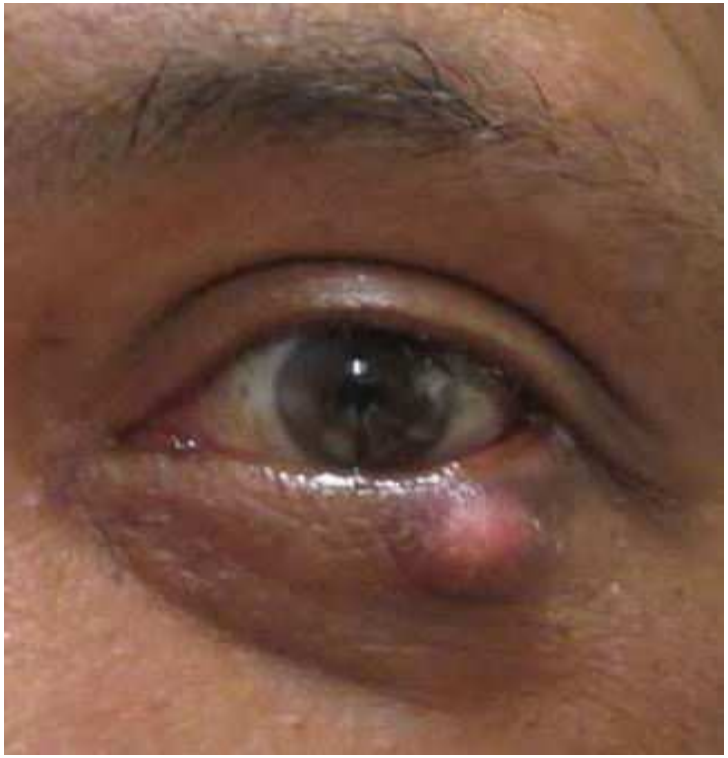
Risk Factors for Chalazion



- Chronic Blepharitis
- Acne Rosacea
- Seborrhea


Inside the Eyelid
Normal
Oil
Gland
Chalazion
Blocked
Oil
Gland





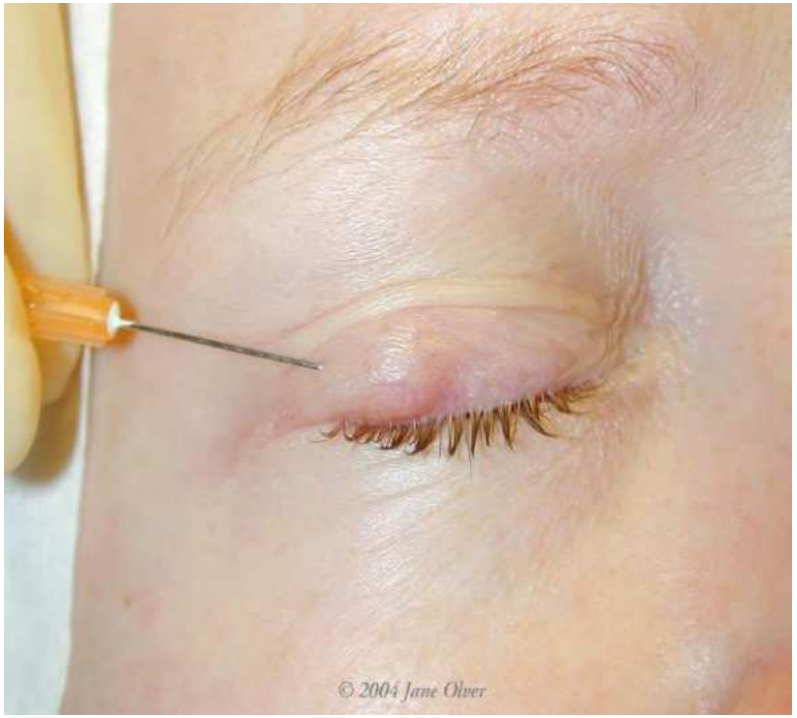
COMPLICATIONS

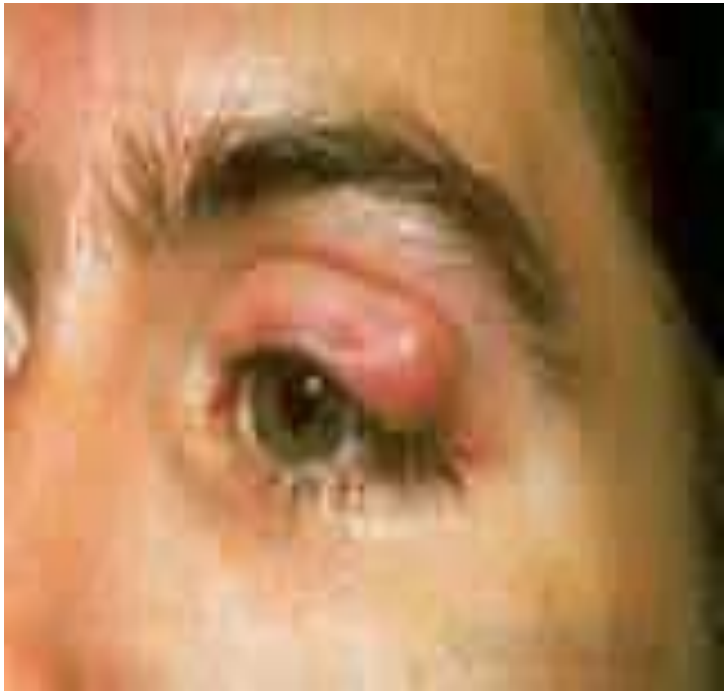
Conjunctival granuloma

Treatment

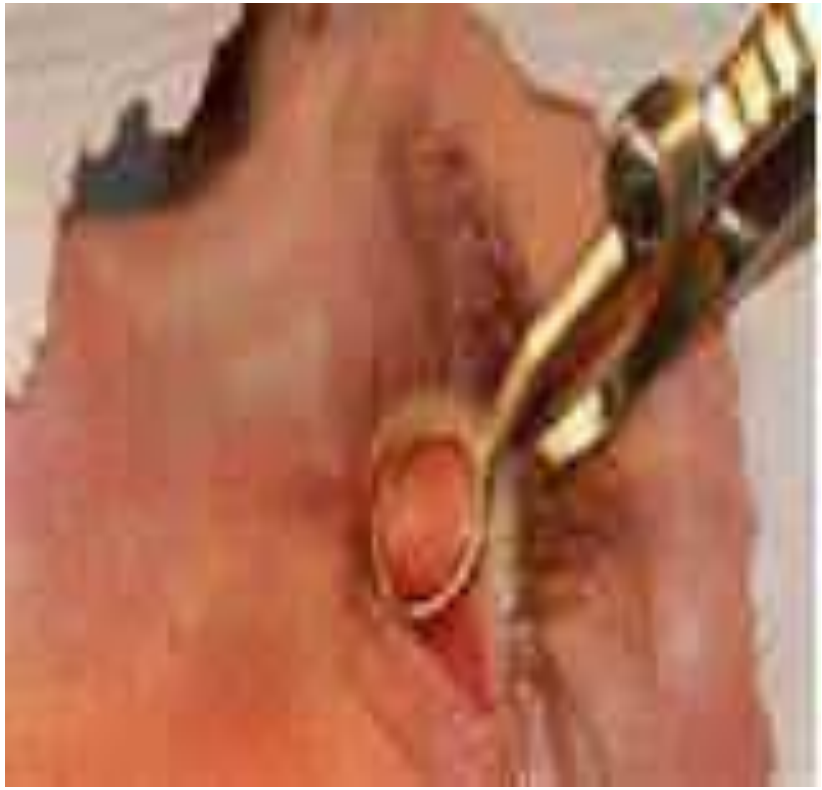

4 Ways to Get Rid of a Chalazion
Warm compress
Medication
Surgery
Corticosteroid injection
ALL ABOUT VISION

Surgery





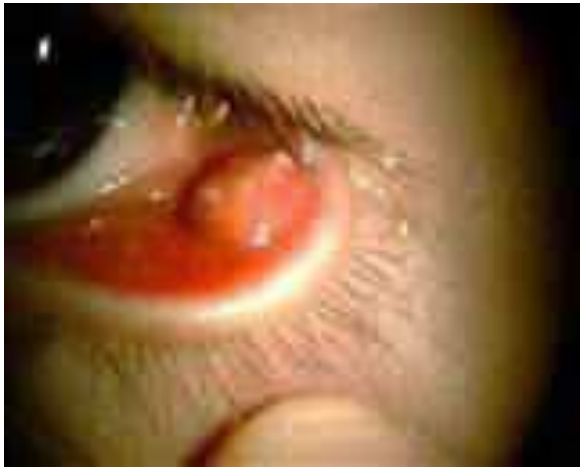
Incision and Curettage



INFECTIONS OF LID MARGINS

Internal hordeolum

an acute staphylococcal infection of meibomian glands



- **External hordeolum**

- an acute staphylococcal infection of lash follicle and its gland of zeiss or moll

EXTERNAL HORDEOLUM (STYE)

- Painful stye.

- External hordeolum=Infection of Zeis gland=stye
- Compared with seborrheic blepharitis patients, patients with S. blepharitis are younger and more frequently female. During acute S. blephararitis, perifolliculitis can lead to ulceration and fibrinous exudates on the lid margin.

Typical changes of chronic blepharitis include crusting and hard brittle scales on the base of the lashes.

Etiology

- Bacterial infection by staphylococcus
- Excessive use of cosmetic
- Poor nutrition
- Sleep deprivation
- Lack of hygiene
- Lack of water
- Rubbing of the eyes.

SIGNS AND SYMPTOMS

- Localized swelling of the eyelid
- Localized pain
- Redness
- Tenderness
- Crusting of the eyelid margins
- Burning in the eye
- Droopiness of the eyelid
- A lump on the top eyelid
- Itching

Internal Hordeolum : It is an acute Supportive inflammation (formation of pus)of mei-bomian glands

Etiology: Occurs due to secondary infection (occurs during or after treatment for another **infection**.) of chalazion.

Symptoms : More violent than stye because the gland is larger & embedded deeply in the dense fibrous tissue.

Sign : Yellow spot (pus) seen shining through the conjunctiva on averting (remaining) the lid;

TREATMENT

Warm compresses an d massages of the lesions for 10 minutes 4 times per day

Tropical anti biotic ointment

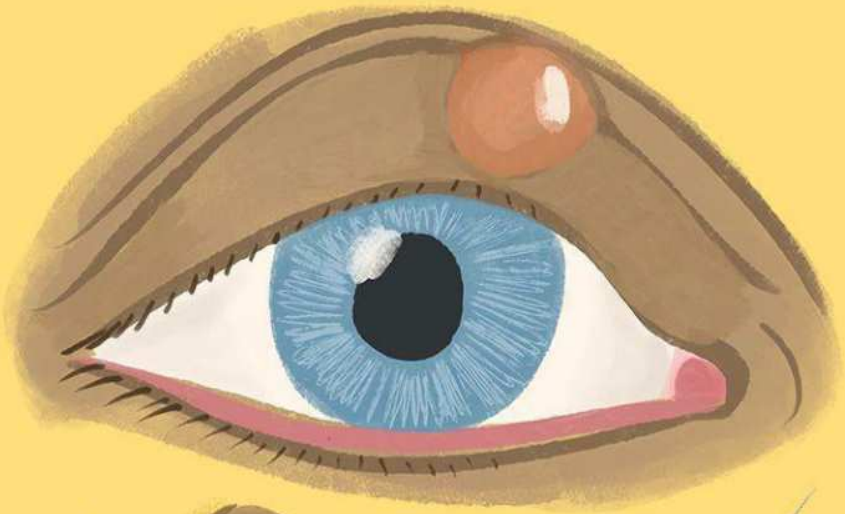
Amoxicillin

Doxycycline

Erythromycin

Chalazion vs. Stye

Chalazion:

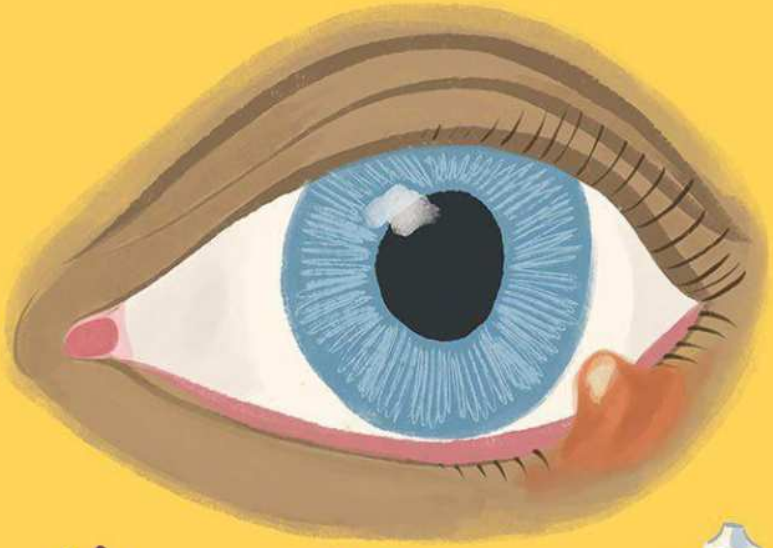


Caused by a blocked oil gland

Resolved by warm compresses, surgery, or steroid shots

Defined by swelling, blurry vision, and a hard bump

Stye:



Triggered by an infection

Treated with antibiotics and OTC solutions

Marked by pain, tearing, and sensitivity to light

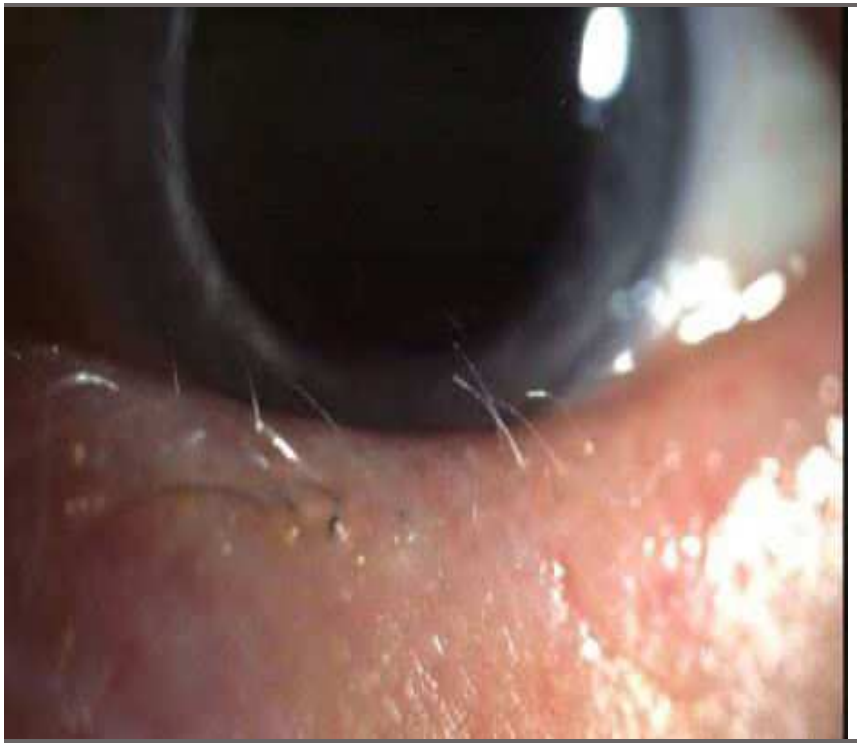
verywell

DISORDERS OF LASHES

1. Trichiasis
2. Metaplastic lashes
3. Distichiasis
4. Phthiriasis palpebrarum
5. Madarosis
6. Poliosis

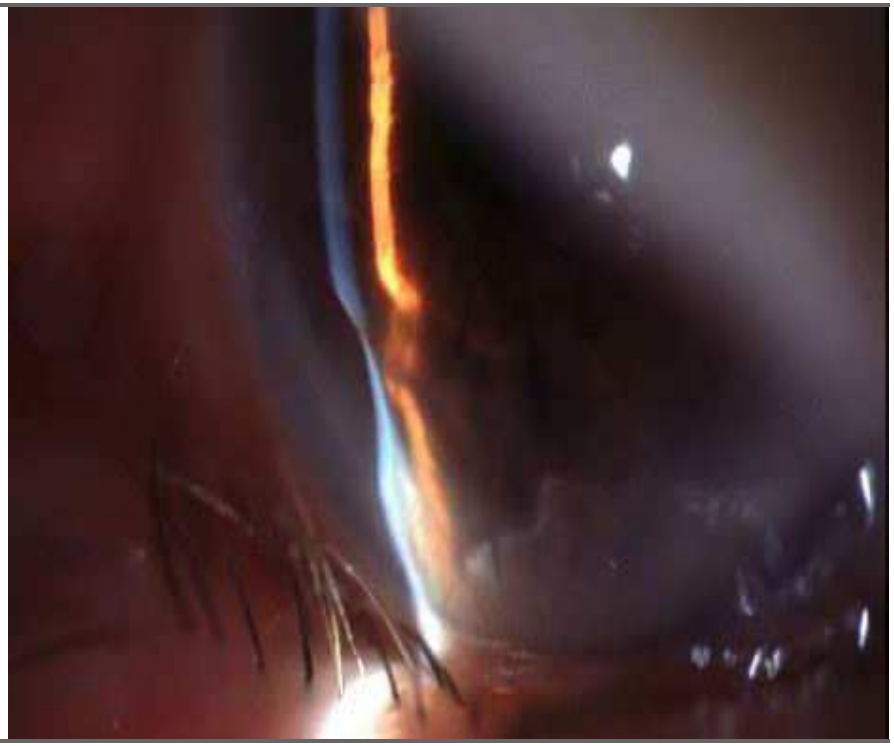
Trichiasis

Signs



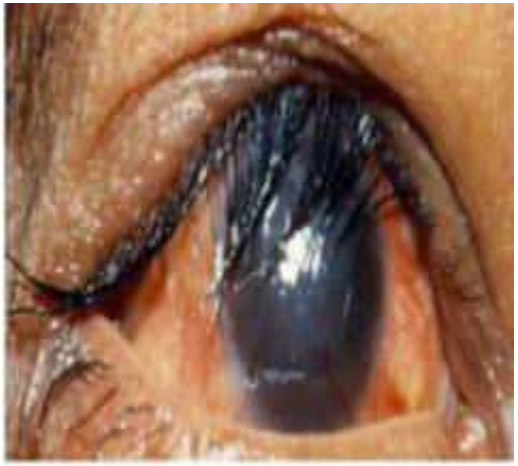
- Posterior misdirection of normal lashes
- Most frequently affects lower lid

Complications



- Inferior punctate epitheliopathy
- Corneal ulceration and pannus

Common Causes of Trichiasis



Etiology: common causes:

- 1. trachoma
- 2. spastic entropion

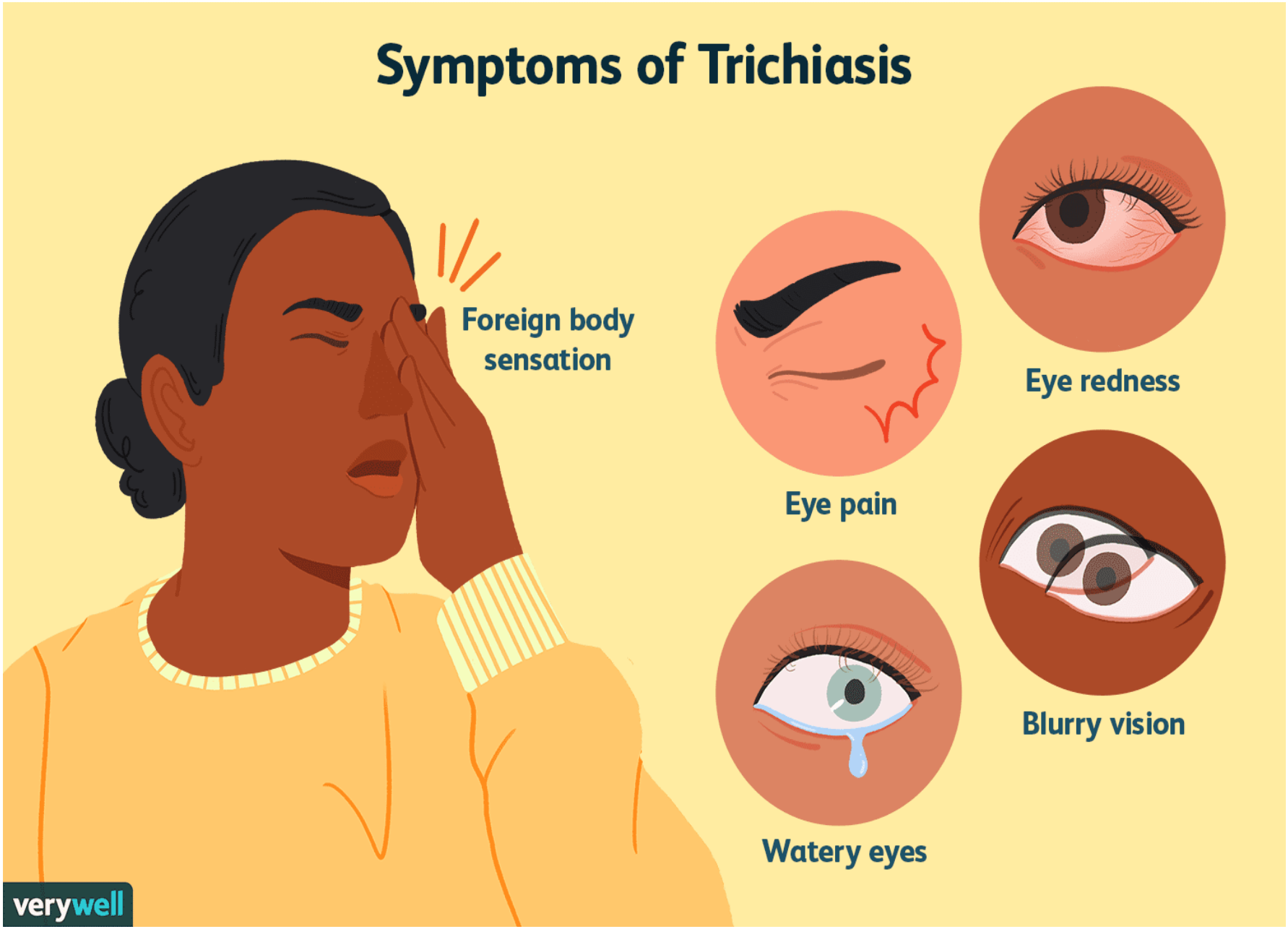
Other causes:

- 1. Blepharitis
- 2. ocular pemphigoid
- 3. scars resulting from injuries
- 4. chemical burns
- 5. destructive inflammations such as stevens johnson syndrome
- 6. congental distichiasis

Symptoms: FB sensation with irritation in the eye, pain ,conjuntival congestion,reflex blepharo spasm, and lacrimation.

Complications : Recurrent erosions, superfecial corneal opacities, recurrent corneal ulcers, corneal vascularization. Sometime it may threaten the Vision.

Symptoms of Trichiasis



Treatment Options for Trichiasis

Epilation - but recurrences within few weeks

Electrolysis - but frequently repeated treatments required

Cryotherapy - for many lashes

Laser ablation - for few scattered lashes

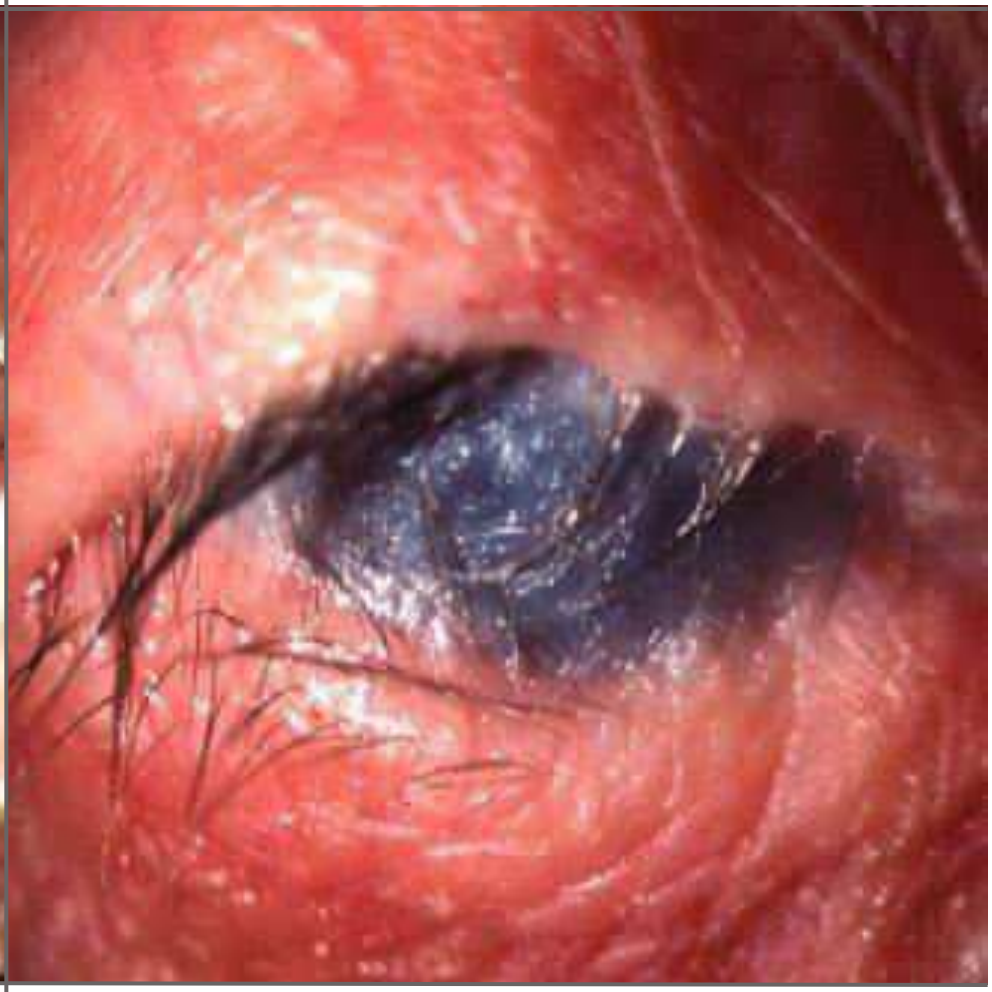
Surgery - for localized crop resistant to other methods

Metaplastic lashes

Signs

Aberrant lashes arising from meibomian gland orifices

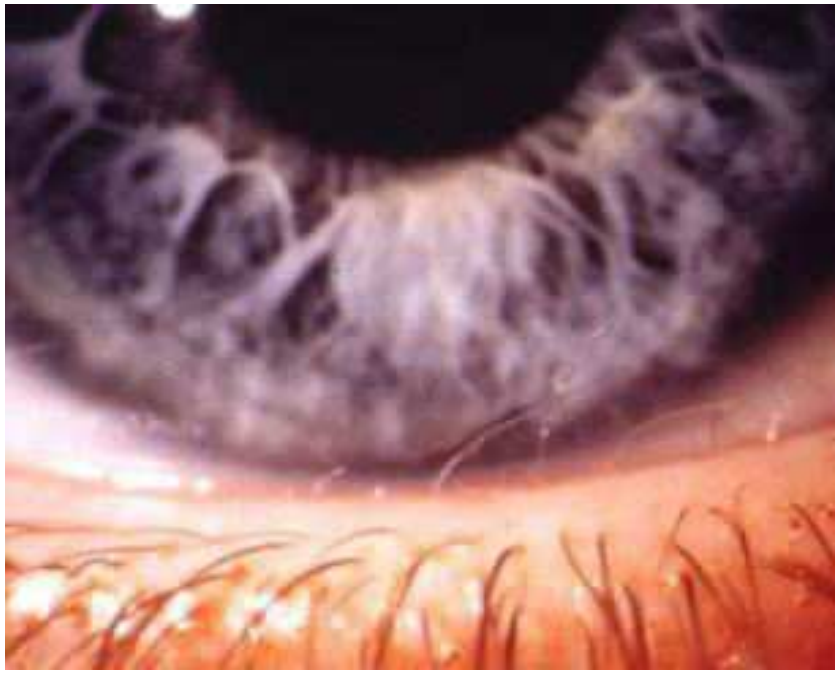
Causes



Cicatrizing conjunctivitis (ocular pemphigoid, Stevens-Johnson, chemical burns)

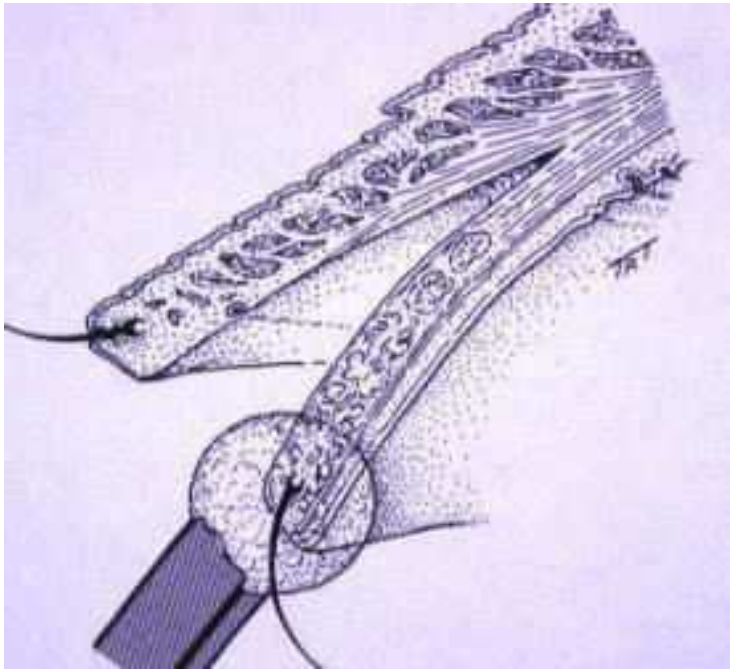
Distichiasis

Signs



- Second row of lashes arising from meibomian gland orifices
- Congenital
- Occasionally dominantly inherited

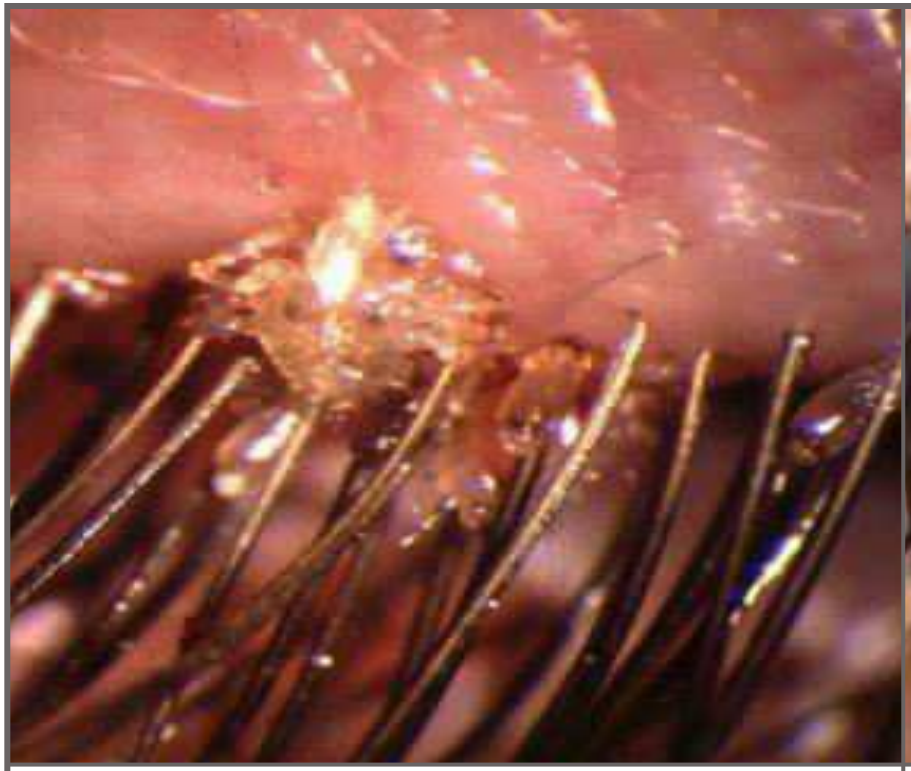
Treatment



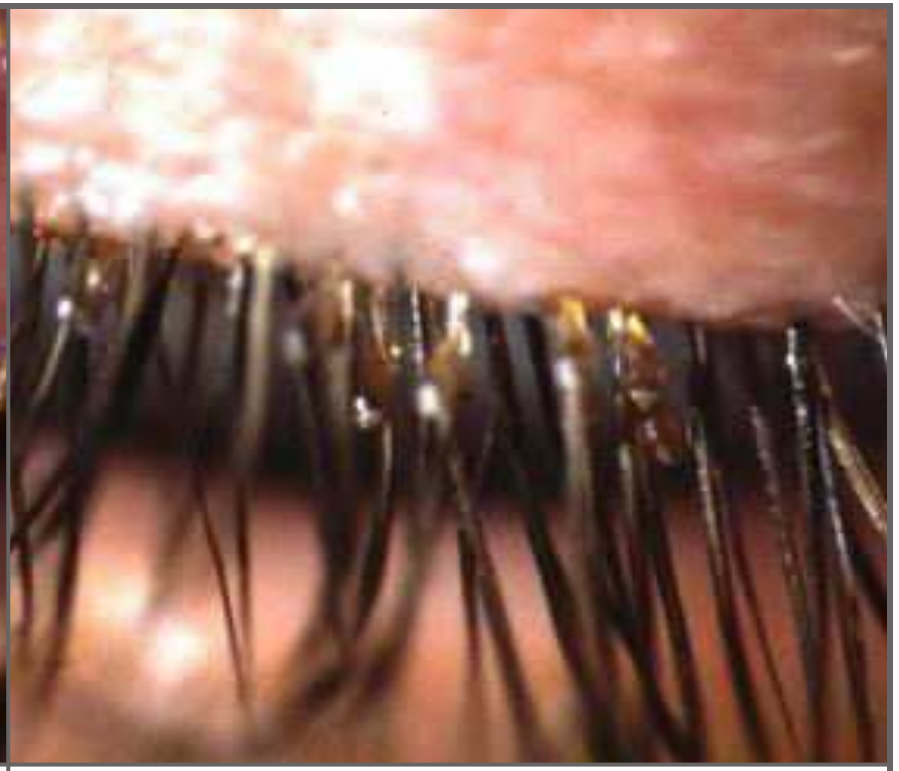
- Division into anterior and posterior lamellae
- Cryotherapy to posterior lamella
- Reapposition of lamellae

Phthiriasis palpebrarum

- **Infestation of lashes by pubic crab louse and its ova (nits)**
- **Typically affects children in poor hygenic conditions**



Lice gripping base of lashes



Nits and empty shells adhere to base of lashes

Treatment - removal, destruction and delousing

Phthiriasis palpebrarum

Infestation of lashes by pubic crab louse and its ova (nits)

Typically affects children in poor hygenic conditions

Lice gripping base of lashes

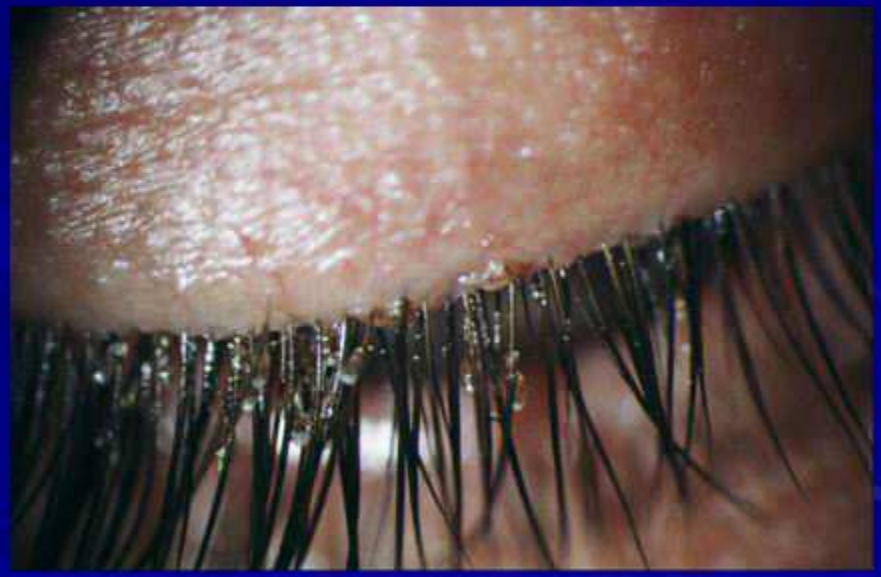
Lice, Crabs (pediculosis, phthiriasis)

Treatment

- Mechanical removal
- Bland ophthalmic ointment

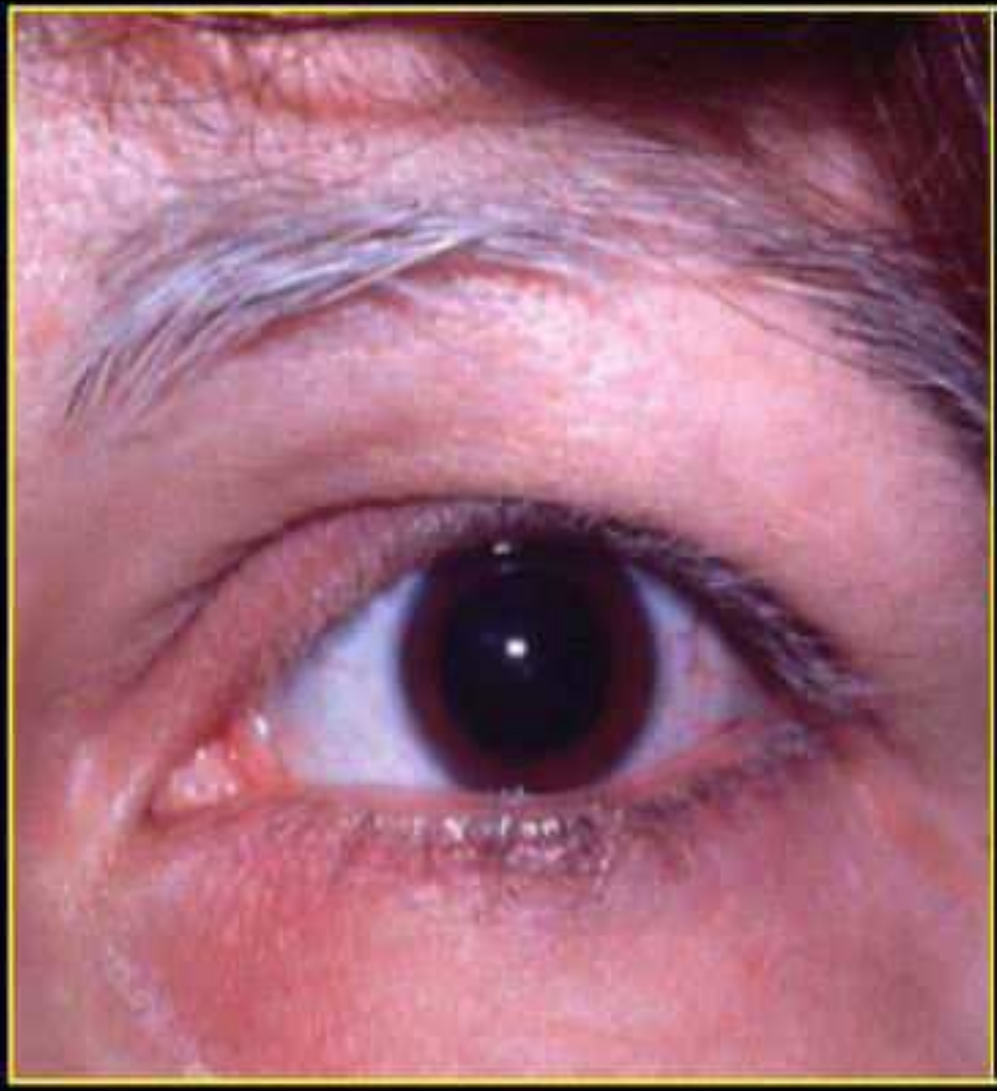
Pearls

- Anti-lice lotion to other involved body parts
- Sexual partners
- R/o other STDs



Poliosis

Premature localized whitening of hair



Ocular associations

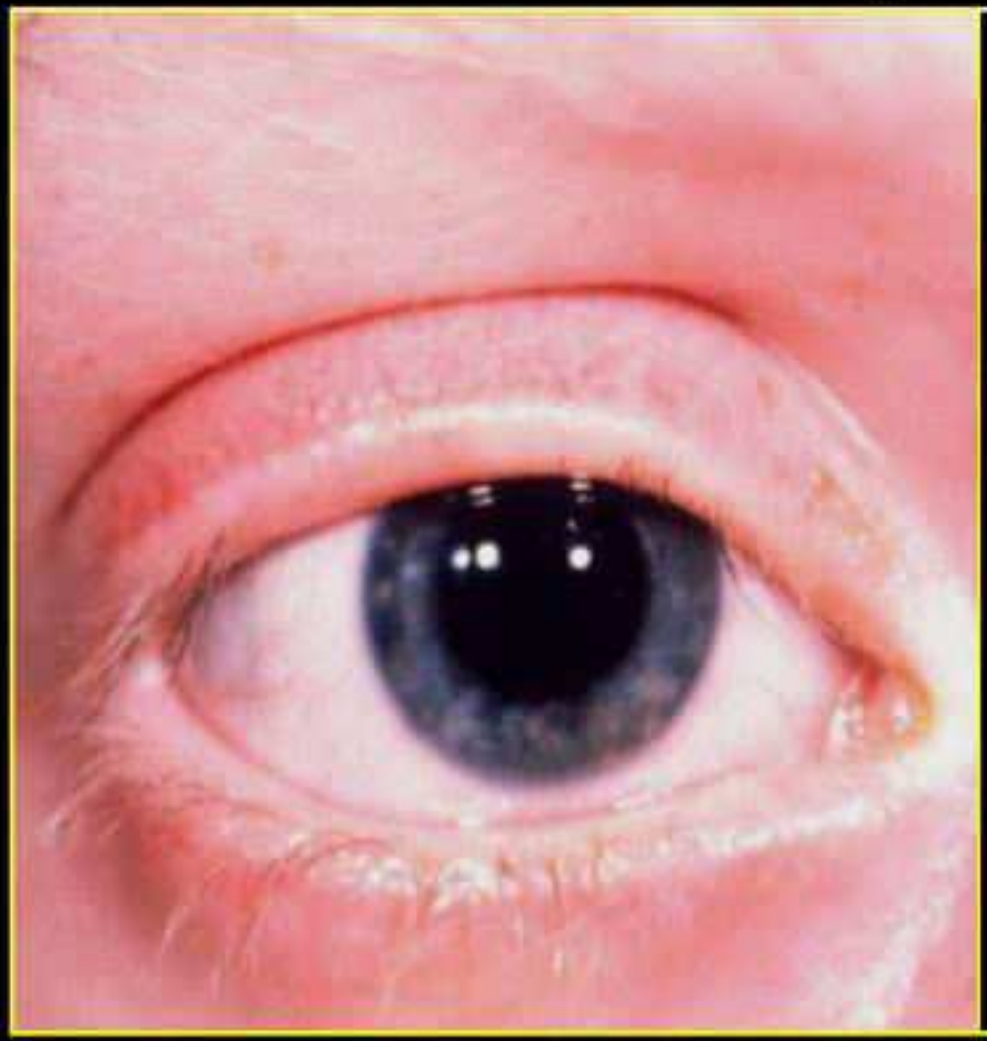
- Chronic anterior blepharitis
- Sympathetic ophthalmitis

Systemic associations

- Vogt-Koyanagi-Harada syndrome
- Waardenburg syndrome

Madarosis

Decrease in number or complete loss of lashes



Local causes

- Chronic anterior lid margin disease
- Infiltrating tumours
- Burns, radiotherapy or cryotherapy

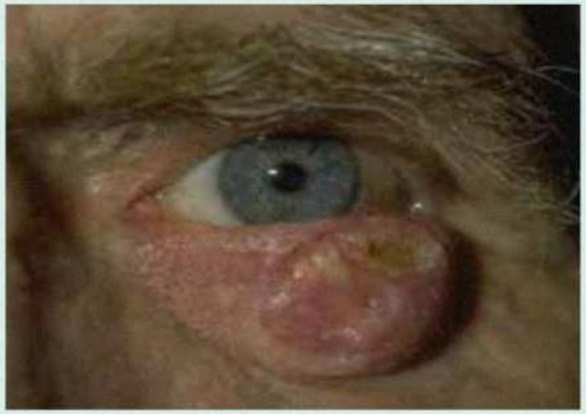
Systemic causes

- Generalized alopecia
- Myxoedema
- SLE
- Syphilis
- Leprosy

Following removal

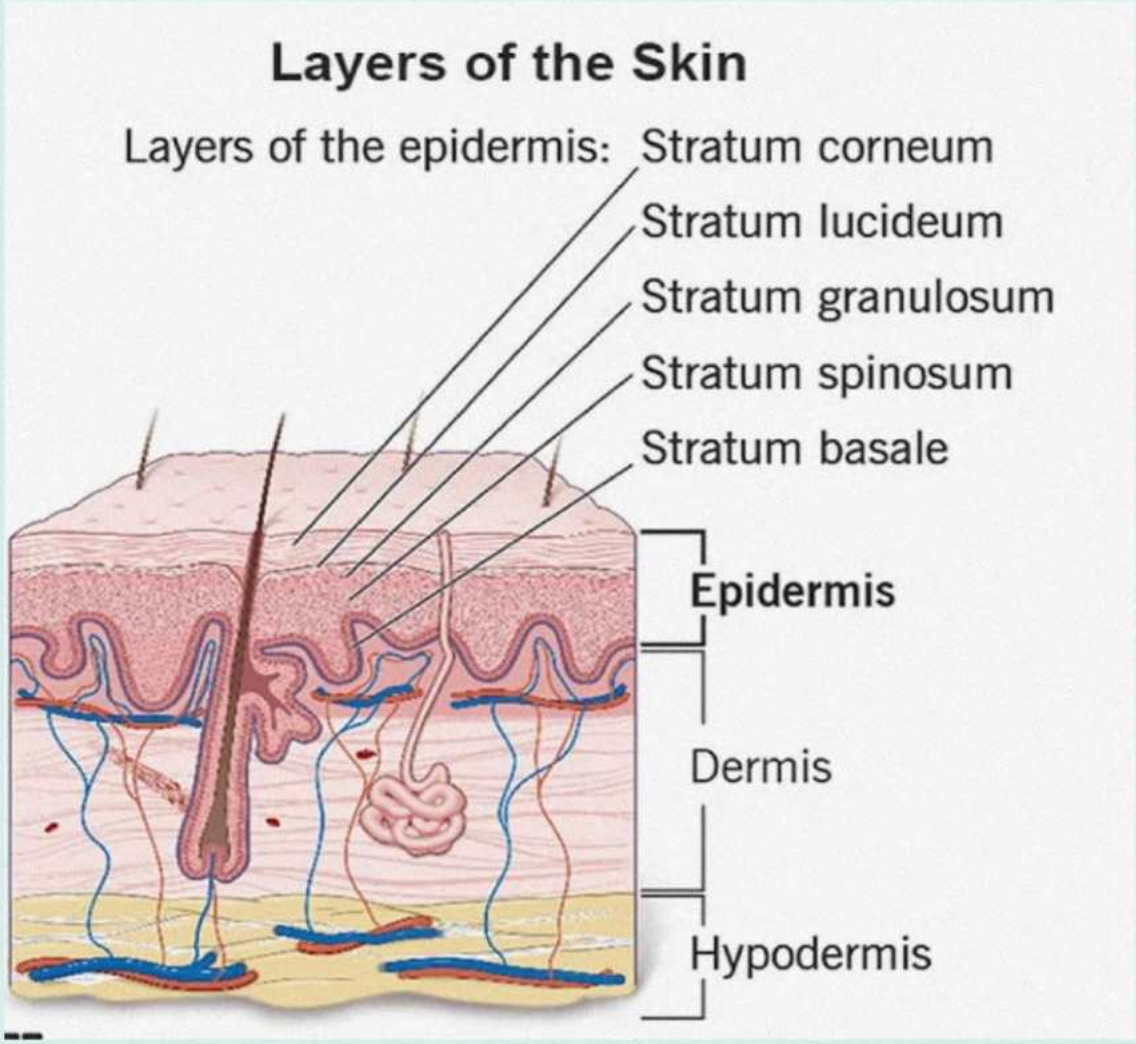

ANY
QUESTIONS

EYELID TUMORS

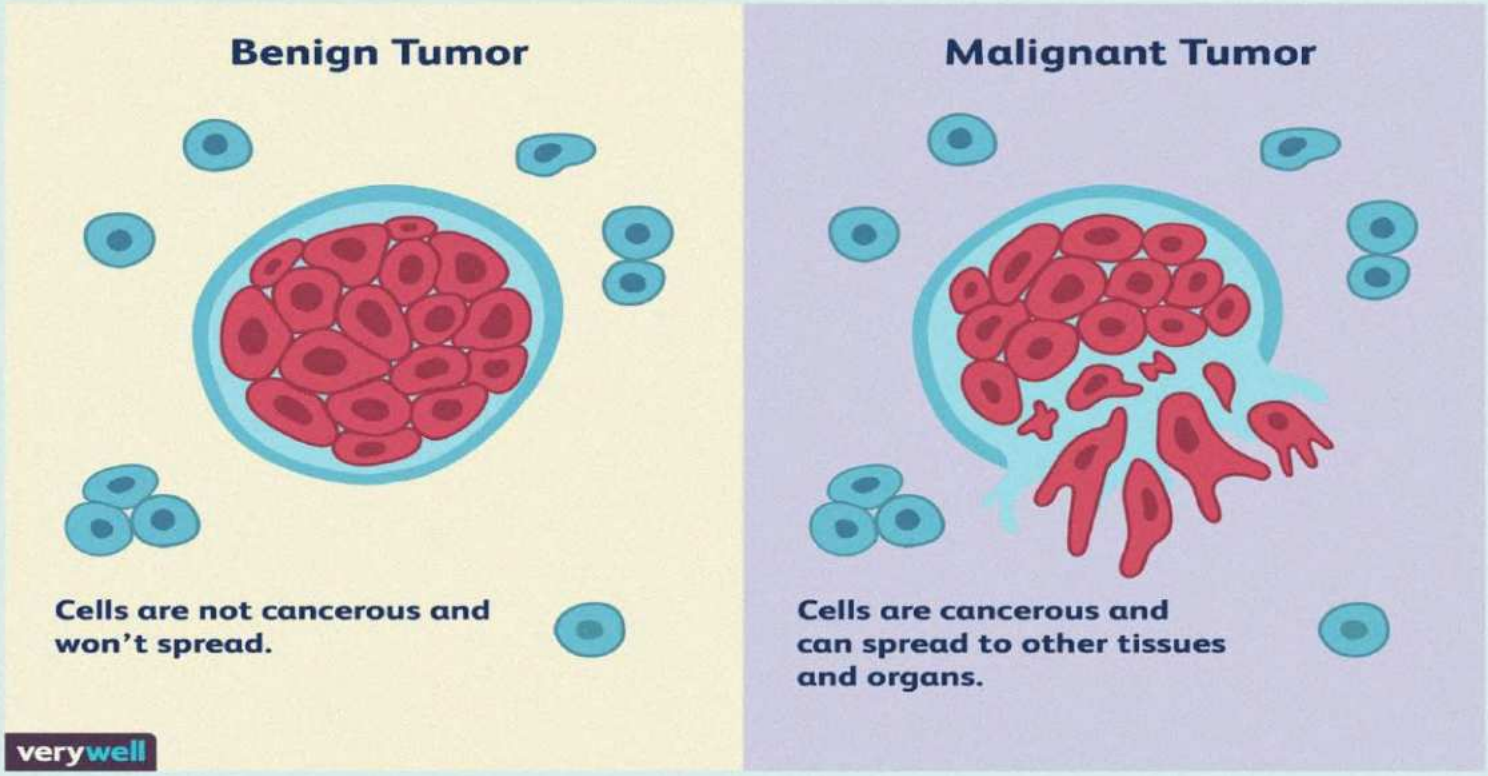


Prof Sofialqbal
FRCS, MRCOphth
Fellowship Orbit/Oculoplastics
Fellowship Refractive surgery

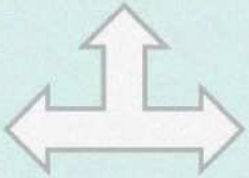
Layers of the Skin



- ***A swelling of a part of a body generally without inflammation resulting in an abnormal growth of tissue***



Classification



BENIGN

- NODULES
- CYSTS
- TUMORS

MALIGNANT

BENIGN

NODULES

- CHALAZION
- HORDEOLUM
- XANTHELASMA
- MOLLUSCUM CONTAGIOSUM

CYSTS

- HIDROCYSTOMA
- SEBACEOUS CYST
- CYST OF ZIESS
- CYST OF MOLL

TUMORS

- KERATOACANTHOMA
- CAPILLARY HEMANGIOMA
- PORT WINE STAIN
- CUTANEOUS HORN
- PYOGENIC GRANULOMA
- VIRAL WART

Benign tumors

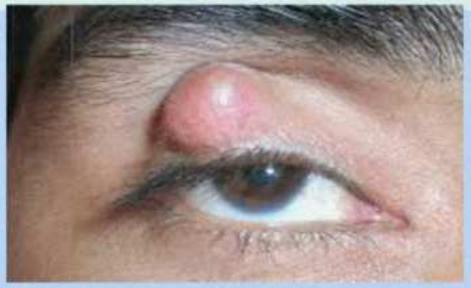
- Epithelial tumors
- Melanocytic tumors
- Adnexal cystic lesions
- Sweat gland origin
- Hair follicle origin
- Miscellaneous lesions

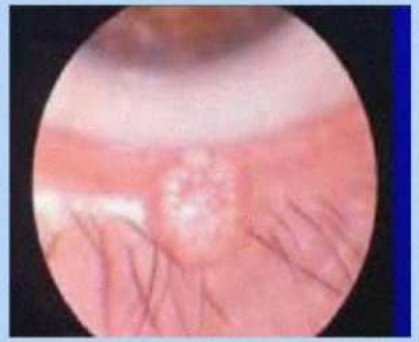
MALIGNANT

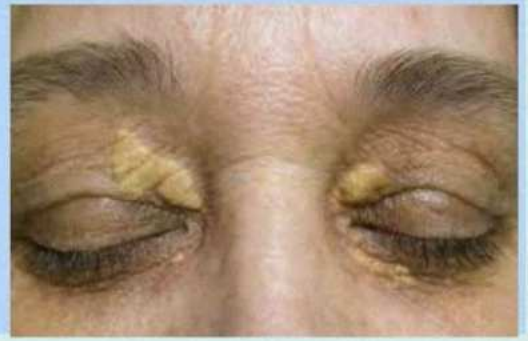
- Basal cell carcinoma
- Sebaceous gland carcinoma
- Melanoma
- Kaposi sarcoma
- Merkel cell carcinoma

NODULES

- *Chlazion*
- *Acute hordeolum*
- *Xanthelasma*
- *Molluscum contagiosum*

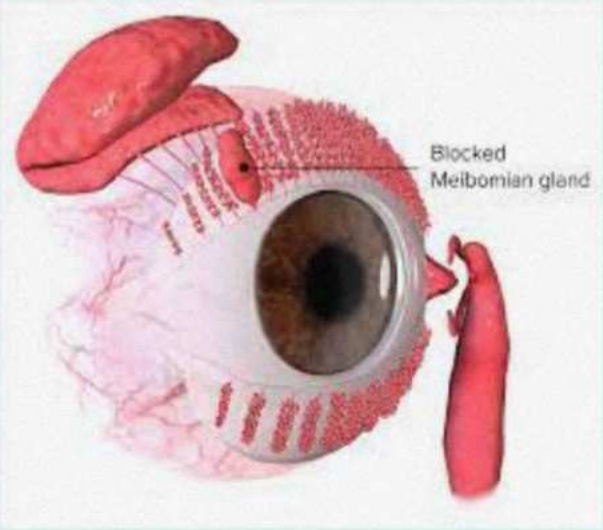






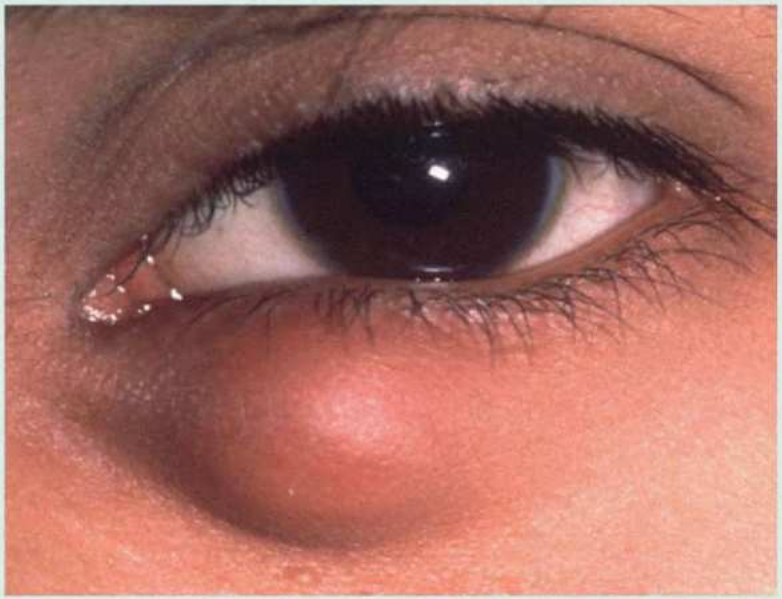
CHALAZION

- Chronic, sterile, lipogranulomatous inflammatory lesion caused by blockage of meibomian gland orifices and stagnation of sebaceous secretions
- Meibomian cyst
- Painless ,round nodule

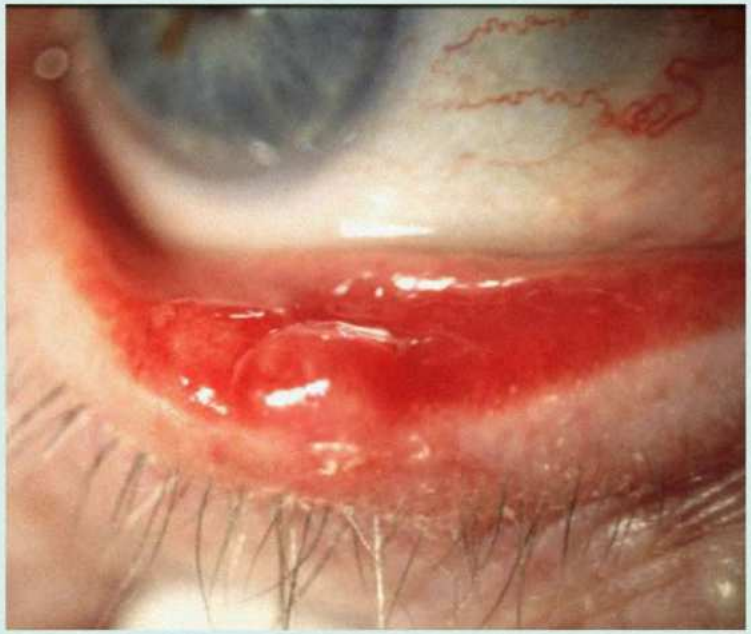


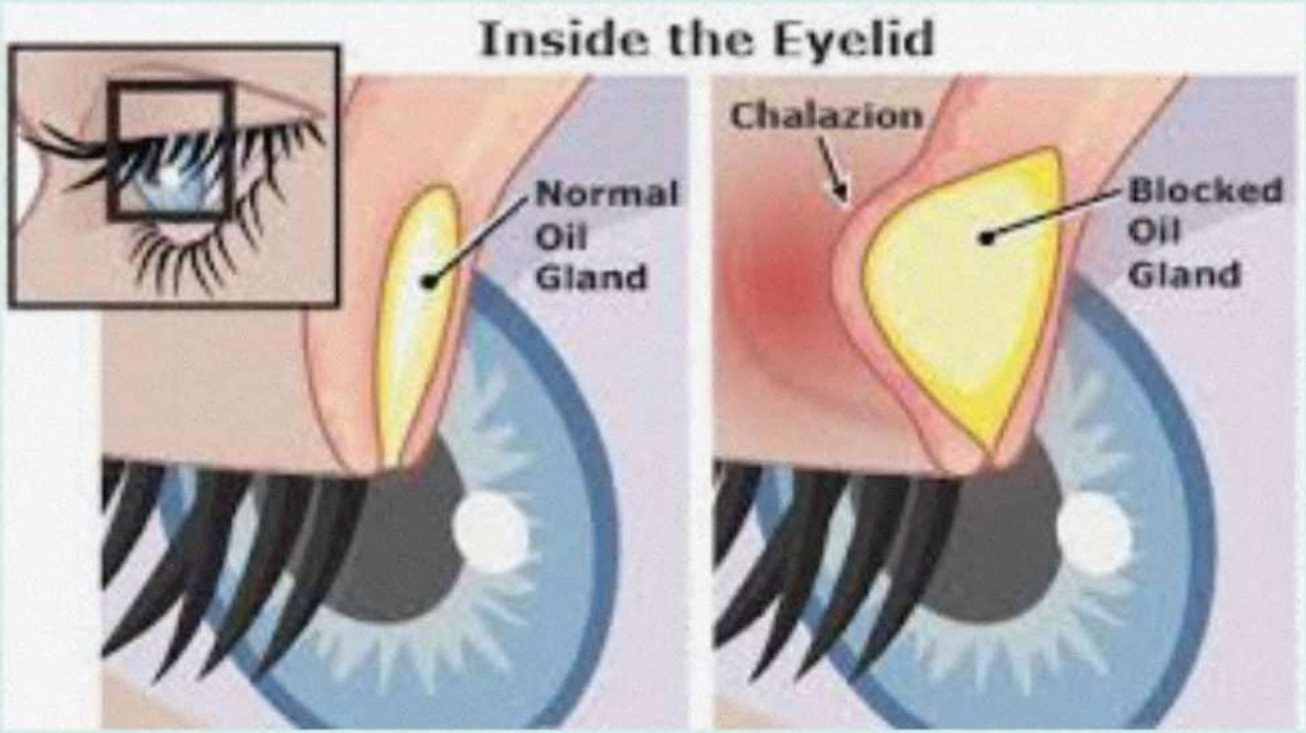
- May press on cornea and can cause astigmatism and blurred vision

Painles,firm roundish nodule within the tarsal plate

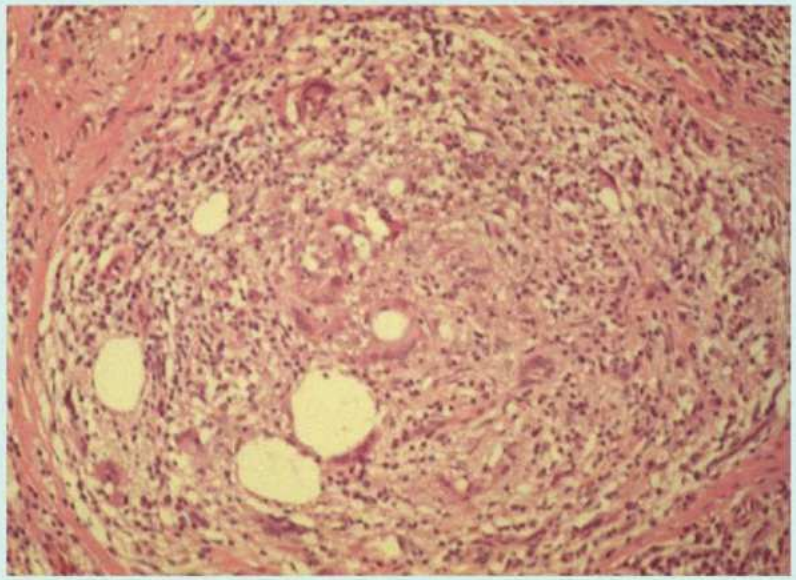


May rupture through the conjunctiva and cause granuloma

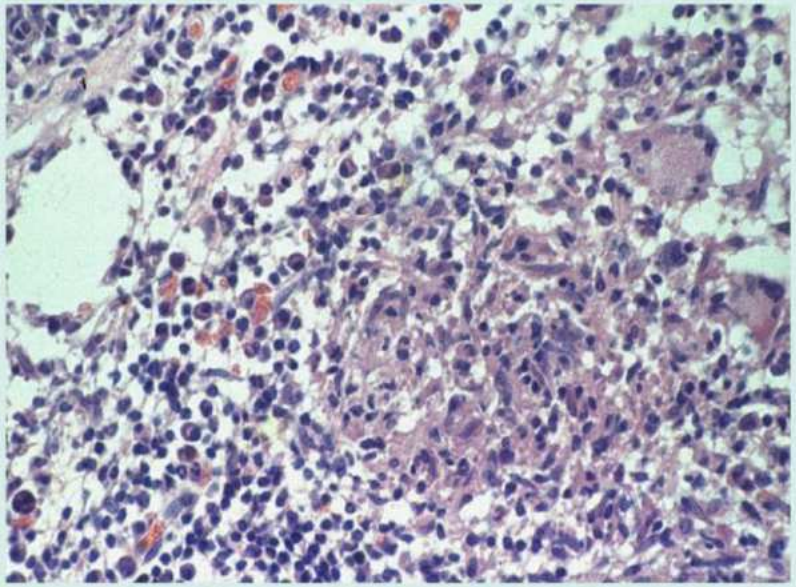




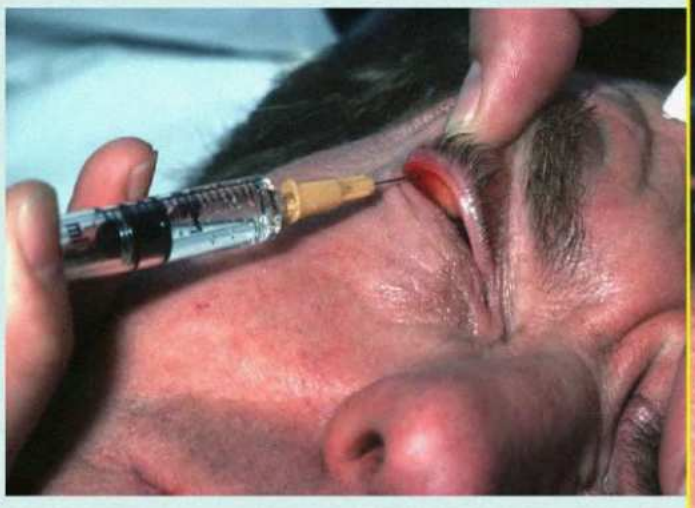
Multiple round spaces previously containing fat surrounded by granulomatous reaction



Epitheliod and multinucleate giant cells

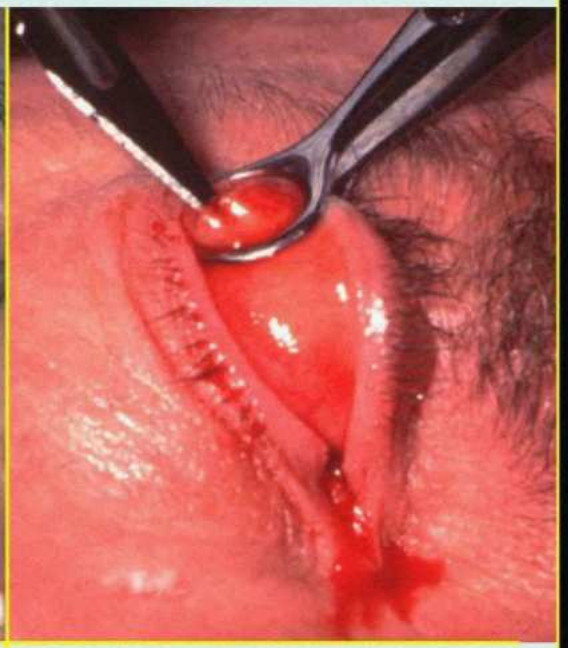


Treatment of chalazion



Injection of local anaesthetic

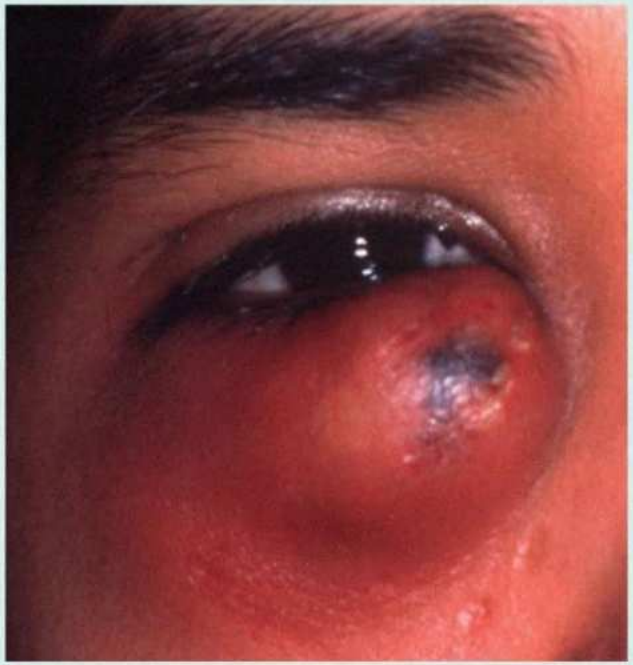
Insertion of clamp



Incision and curettage

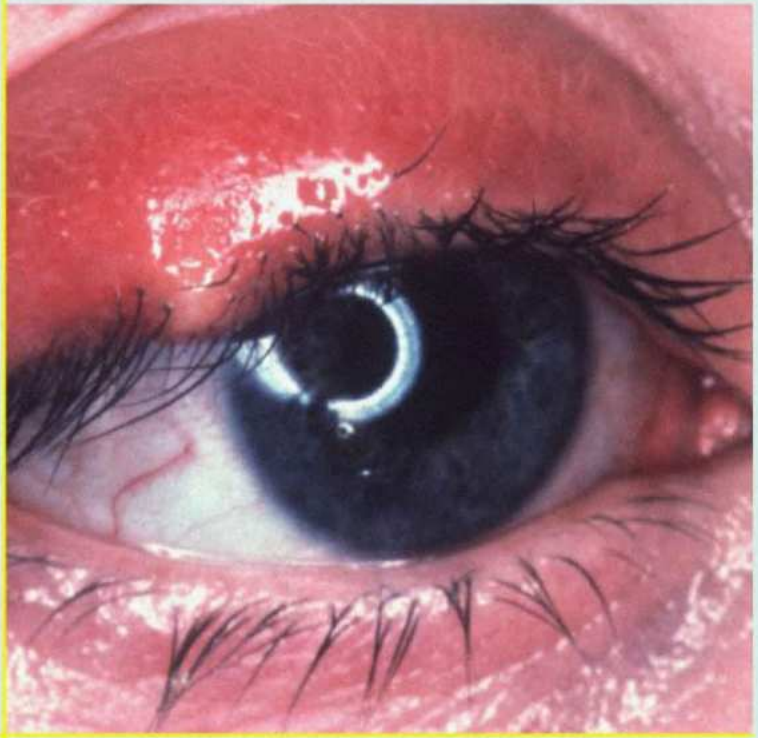
Acute hordeola

Internal hordeolum (acute chalazion)



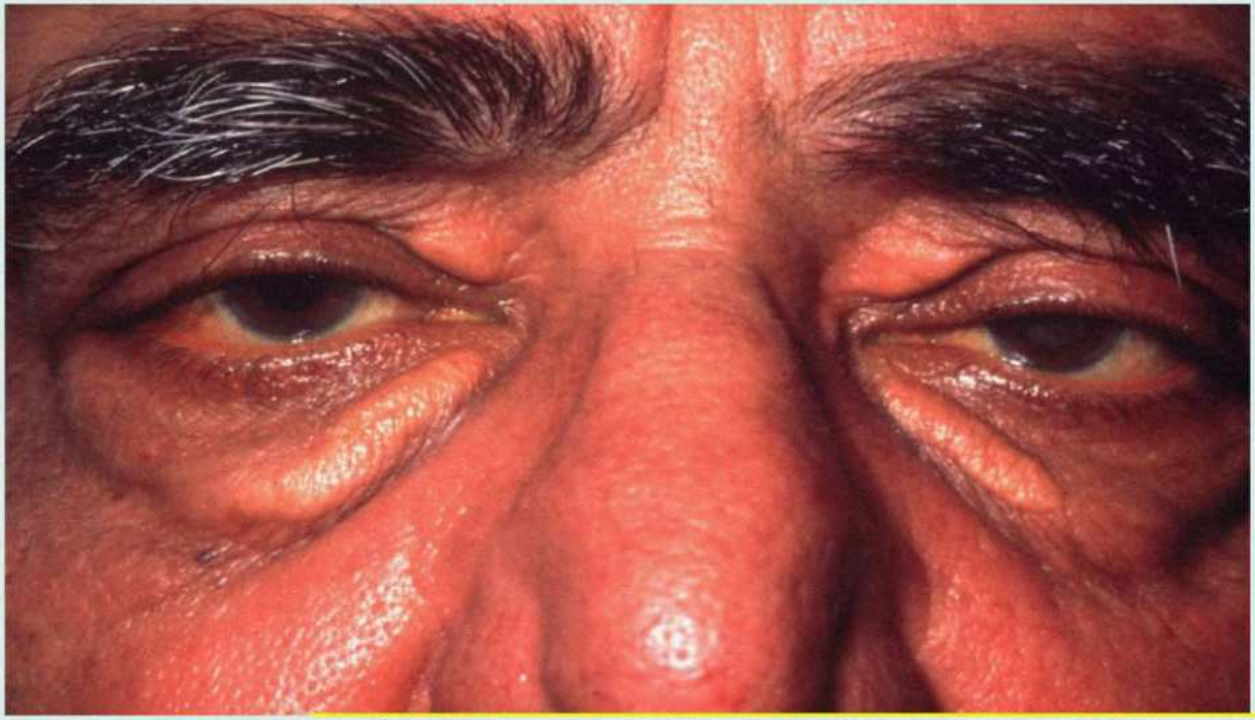
- *Staph.* abscess of meibomian glands
- Tender swelling within tarsal plate
- May discharge through skin or conjunctiva

External hordeolum (stye)



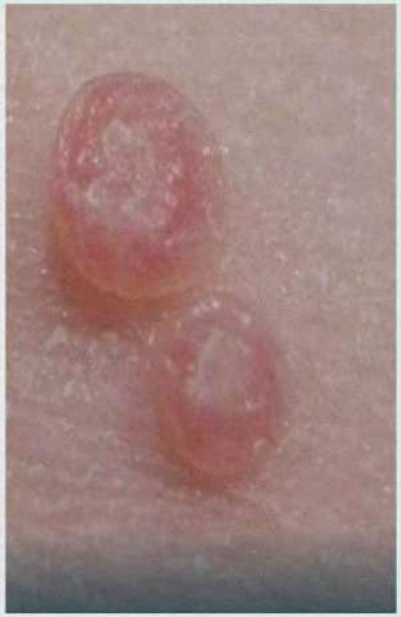
- *Staph.* abscess of lash follicle and associated gland of Zeis or Moll
- Tender swelling at lid margin
- May discharge through skin

Xanthelasma



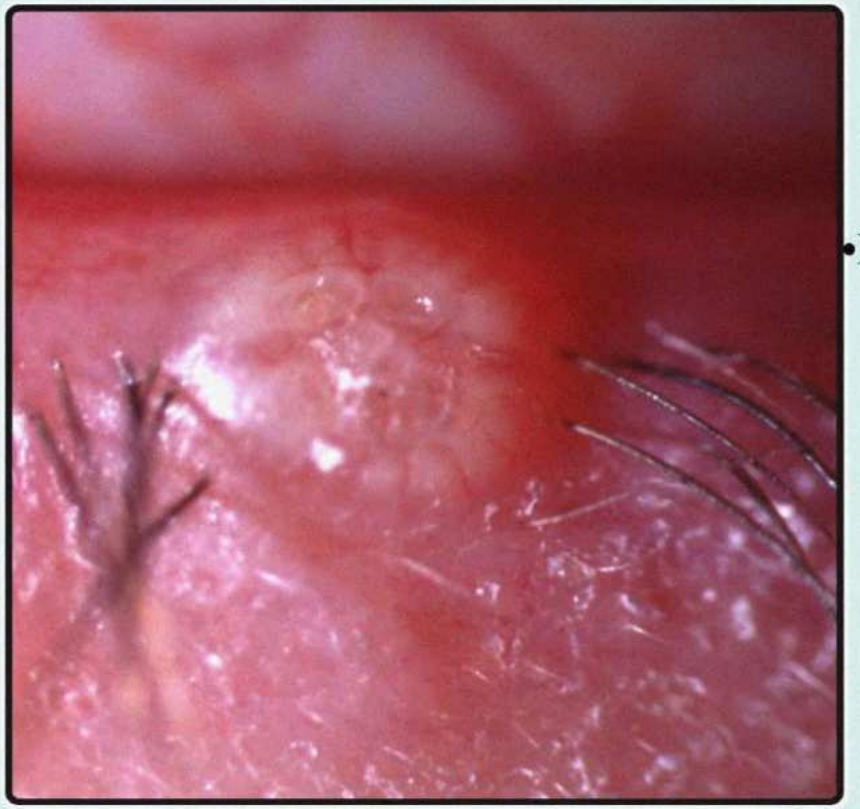
- Common in elderly or those with hypercholesterolaemia
- Yellowish, subcutaneous plaques containing cholesterol and lipid
- Usually bilateral and located medially

Molluscum contagiosum



It is caused by a DNA poxvirus called the molluscum contagiosum virus (MCV). MCV has no nonhuman-animal reservoir (infecting only humans). There are four types of MCV, MCV-1 to -4; MCV-1 is the most prevalent and MCV-2 is seen usually in adults. The virus that causes molluscum is spread from person to person by touching the affected skin. The virus may also be spread by touching a surface with the virus on it, such as a towel, clothing, or toys.

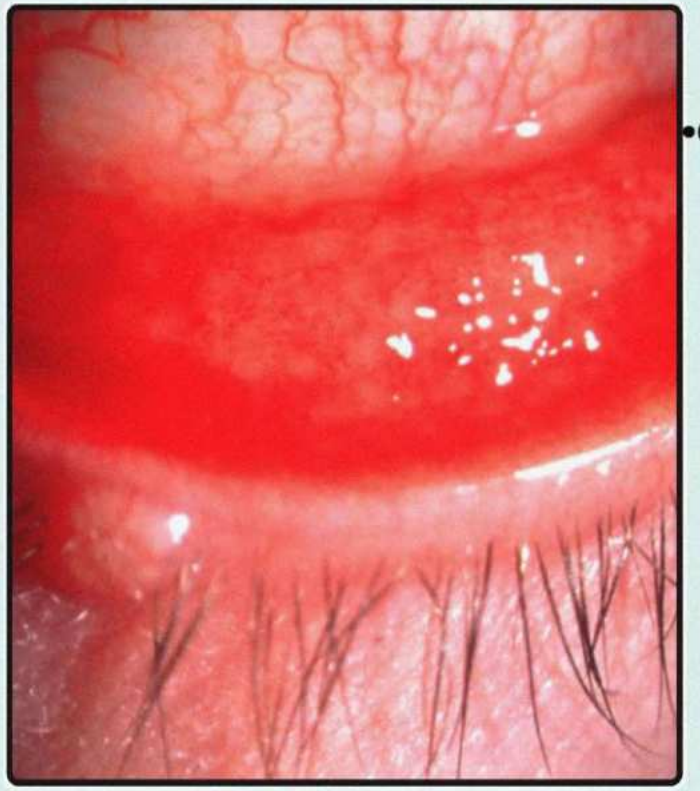
SIGNS



- **Painless, waxy, umbilicated nodule**

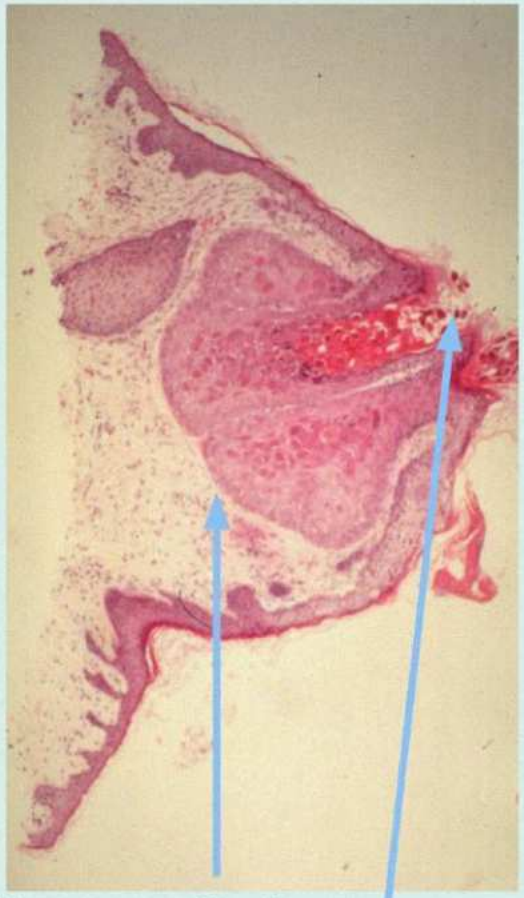
  May be multiple in AIDS patients

Complications

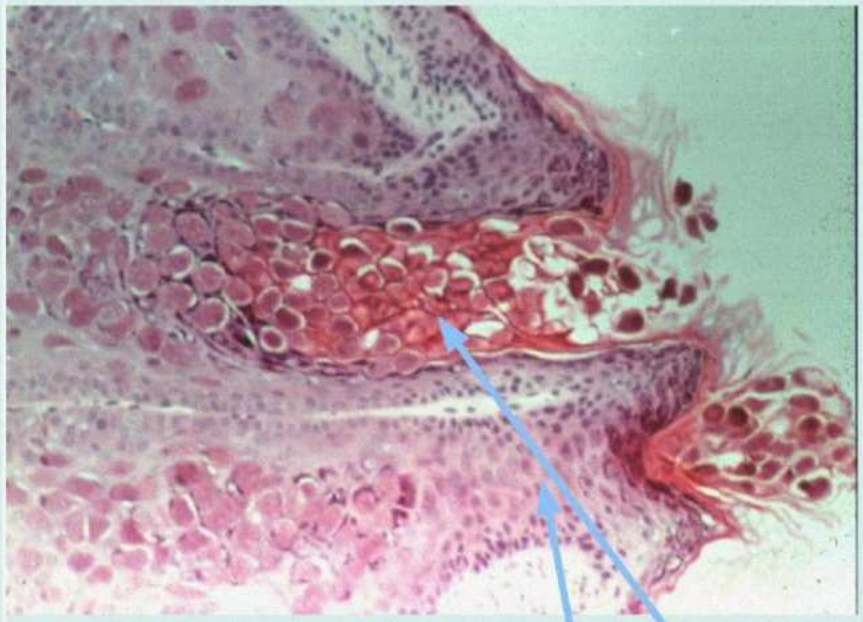


- Chronic follicular conjunctivitis
- Occasionally superficial keratitis

Histology of molluscum contagiosum



- Circumscribed lesion
- Surface covered by normal epithelium except in centre

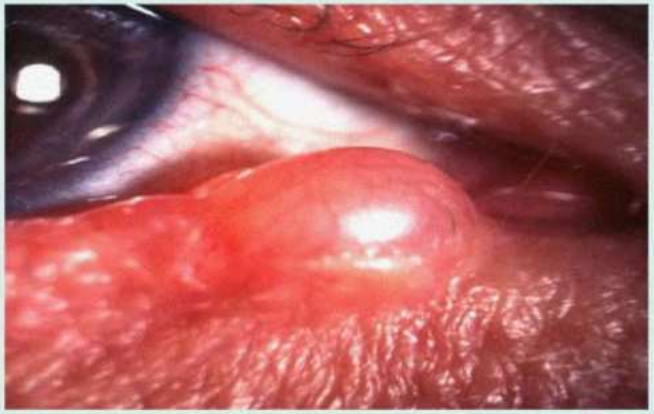


- Lobules of hyperplastic epithelium
- Intracytoplasmic (Henderson-Patterson) inclusion bodies
- Deep within lesion bodies are small and eosinophilic
- Near surface bodies are larger and basophilic

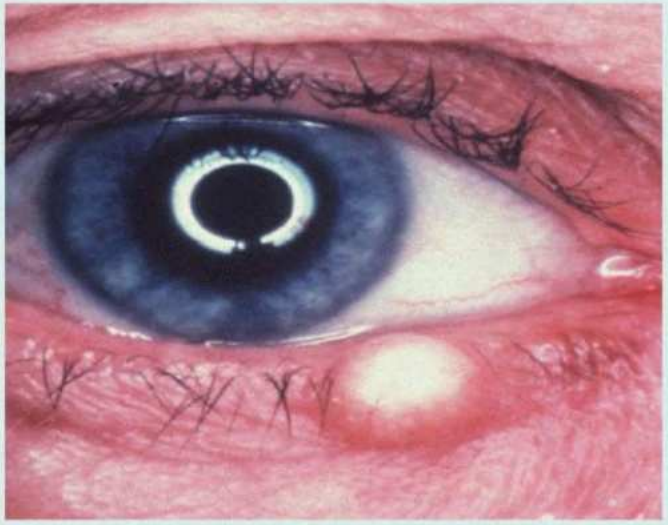
CYSTS

- *Hidrocystoma*
- *Sebaceous cyst*
- *Cyst of Zeiss*
- *Cyst of Moll*

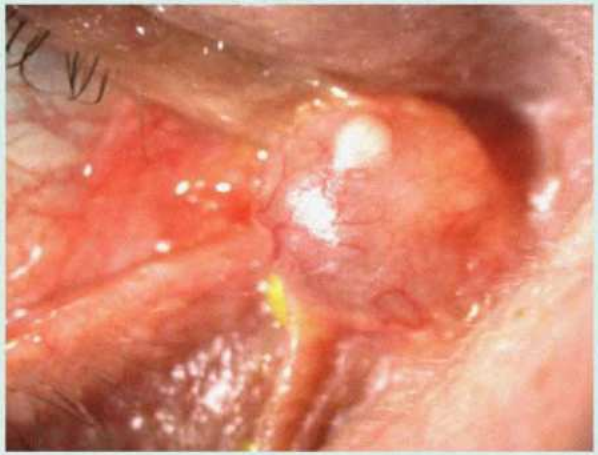
CYSTS

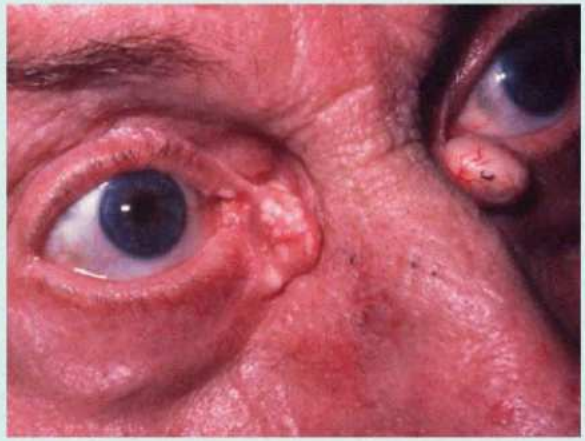


- **CYST OF MOLL**
- **Translucent**
- **On eyelid margin**



- **Cyst of Ziess**
- **Opaque**
- **On lid margins**





- **ECCRINE SWEAT GLAND HIDROCYSTOMA**

- *Similar to cyst of Moll*
- *Not confined to lid margin*

SEBACEOUS CYST

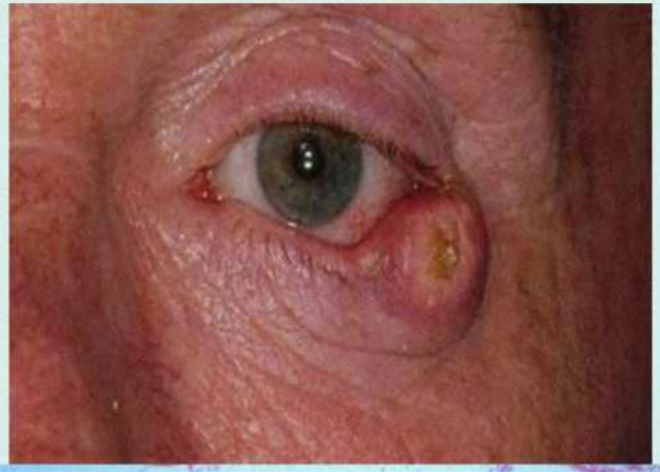
- *Cheesy content*
- *Often on inner canthus*

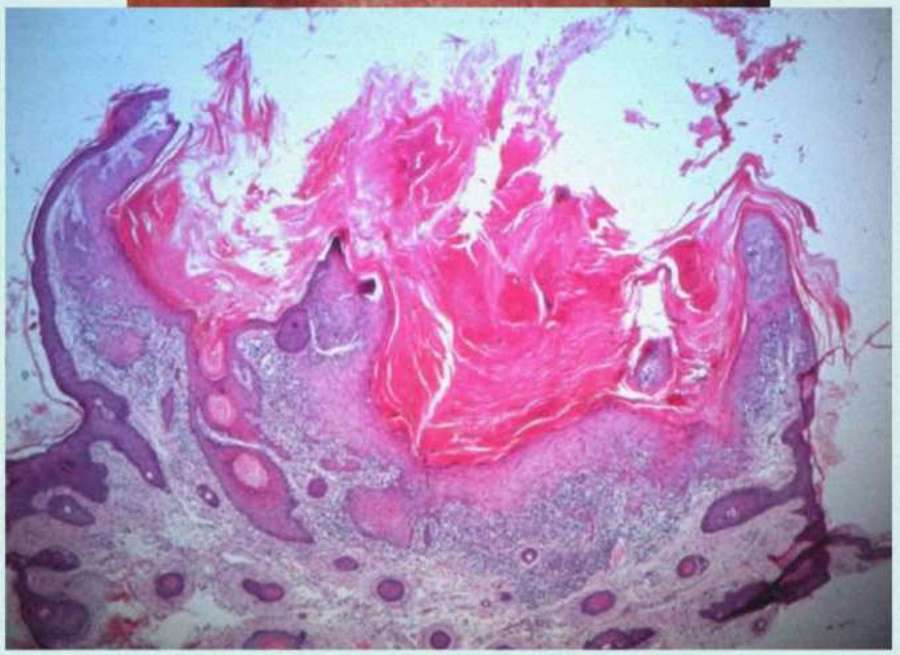
Tumours

- *Keratoacanthoma*
- *Capillary haemangioma*
- *Port wine stain*
- *Pyogenic granuloma*
- *Cutaneous horn*
- *Viral wart*

Keratoacanthoma

- Uncommon , fast growing nodule
- Involutes spontaneously with one year
- Rolled margins with a central keratin filled crater
- There may be underlying SCC





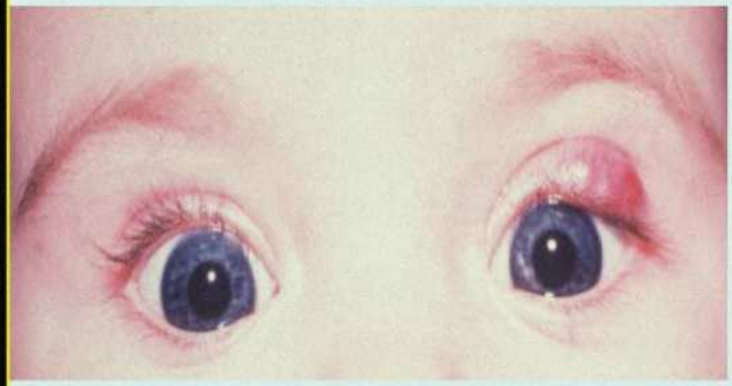
Hemangioma

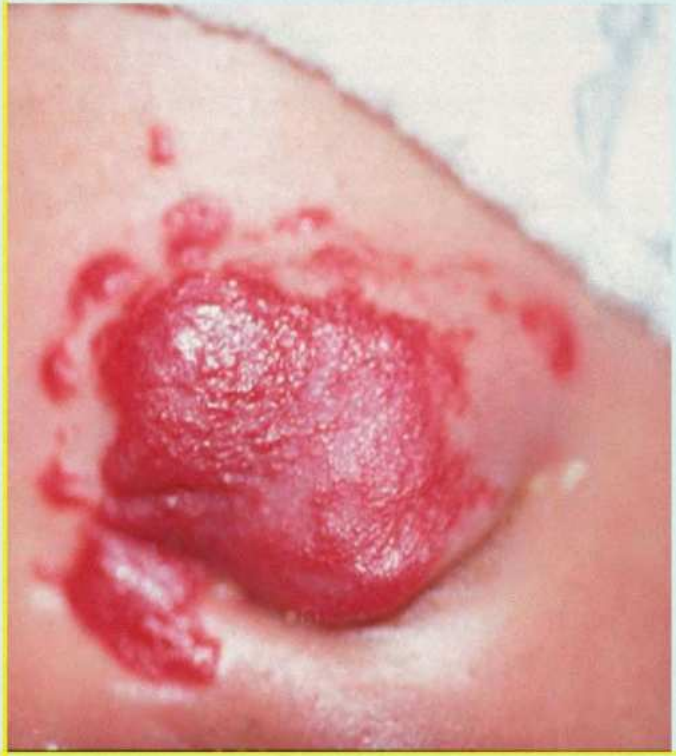
- The most common tumor of infancy and childhood (4-10%)
- 3-5 times more seen in girls
- More seen in premature infants (<1200 grams% 23)
- Not frequent in darker-skinned babies
- Usually occurs in first 2 weeks after birth
- Initially, a pale-colored, telangiectatic or macular red stain or purple-colored stain
- Single lesion in 80%, 20% more than one lesion
- In patients with more than one lesion accompanies other system hemangiomas (liver etc.)

Types

- **Central**
  - In bone
- **Capillary**
  - Intercommunication capillary vessels
    - Strawberry angioma
    - Port wine stain
    - Salmon's notch
- **Cavernous**
  - Dilated blood containing spaces lined by endothelium
- **Arterial**

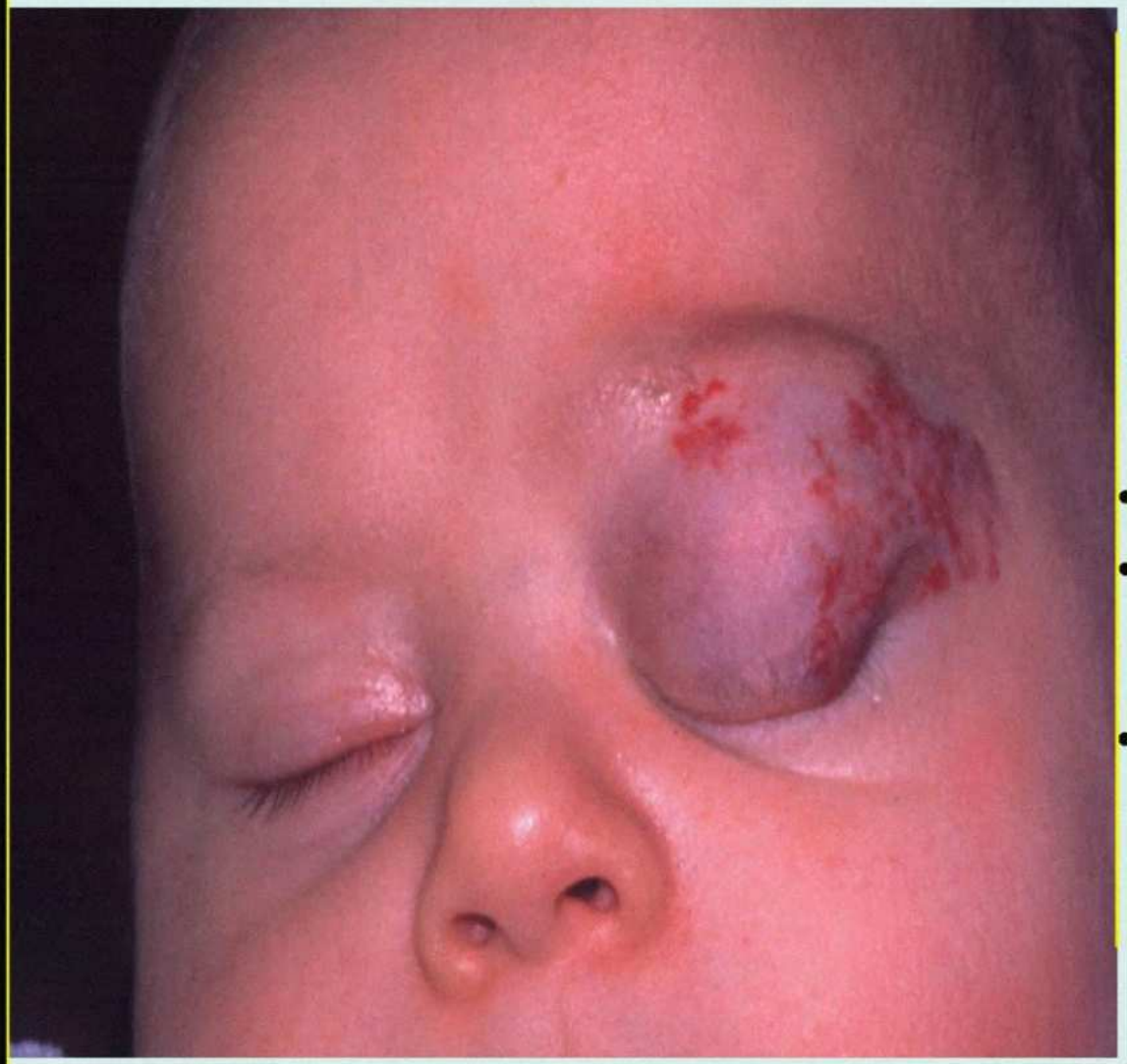
Capillary haemangioma





- Rare tumour which presents soon after birth
- Starts as small, red lesion, most frequently on upper lid
- Blanches with pressure and swells on crying
- May be associated with intraorbital extension
- Grows quickly during first year
- Begins to involute spontaneously during second year

Periocular haemangioma



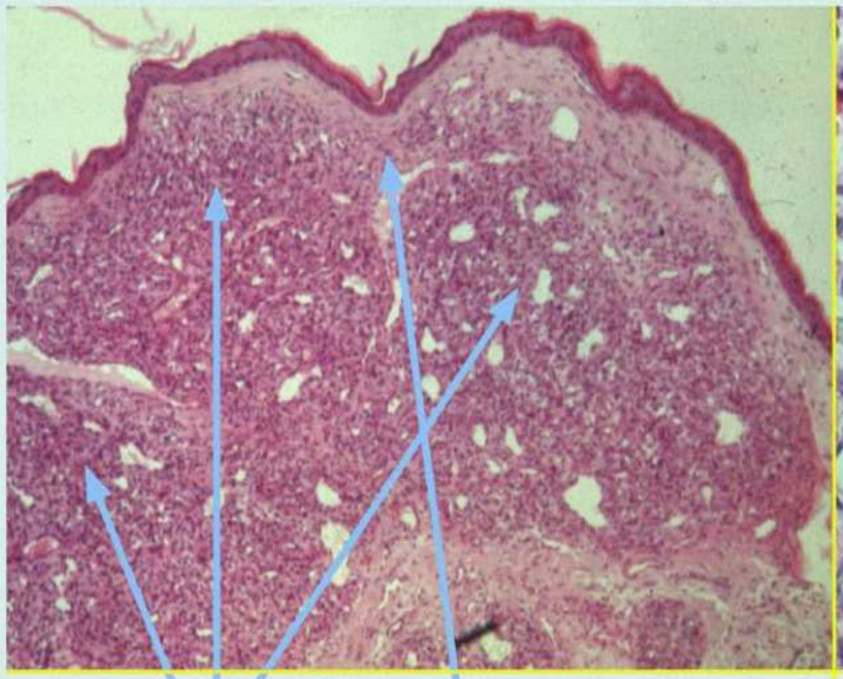
Treatment options

- Steroid injection in most cases
- Surgical resection in selected cases

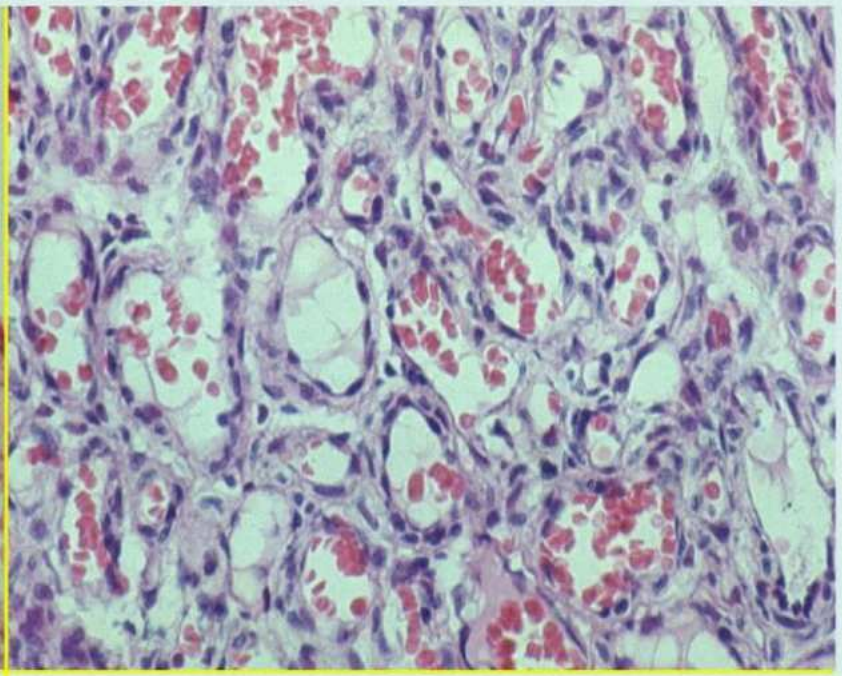
Occasional systemic associations

- High-out heart failure
- *Kasabach-Merritt syndrome* - thrombocytopenia, anaemia and reduced coagulant factors
- *Maffuci syndrome* – skin haemangiomas, endrochondromas and bowing of long bones

Histology of capillary haemangioma

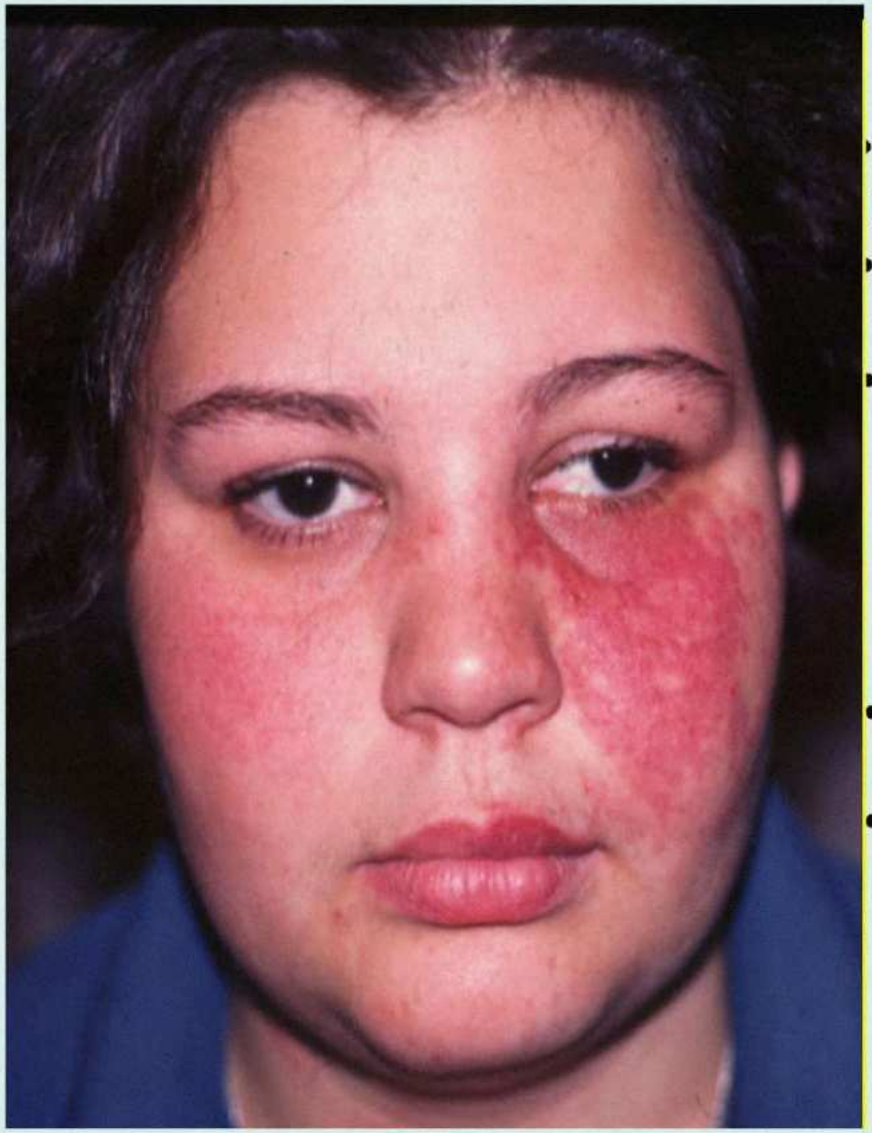


Lobules of capillaries Fine fibrous septae



Lobules under high magnification

Port-wine stain (naevus flammeus)

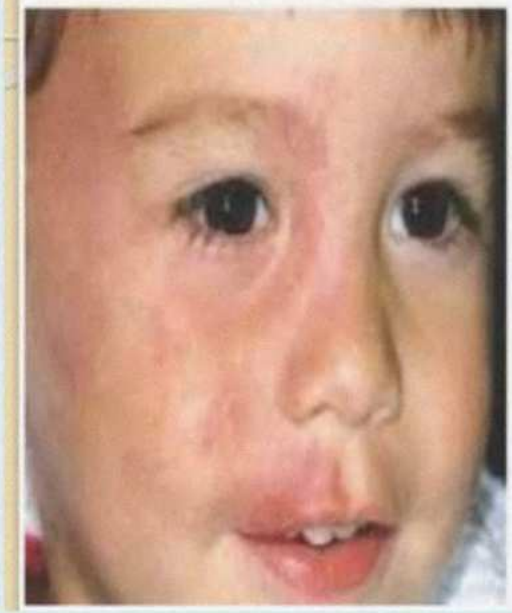


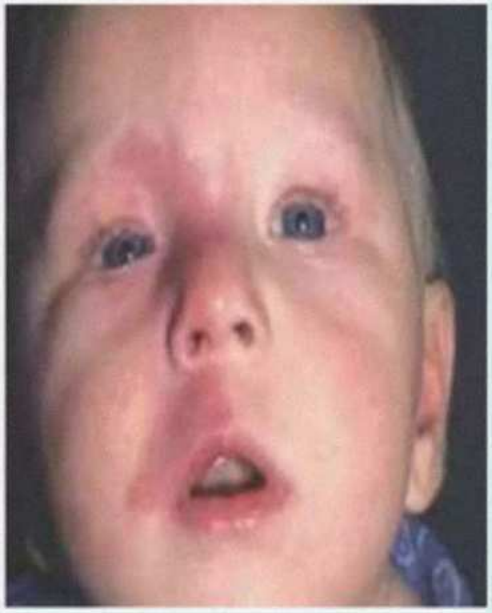
- Rare, congenital subcutaneous lesion
- Segmental and usually unilateral
- Does not blanch with pressure

Associations

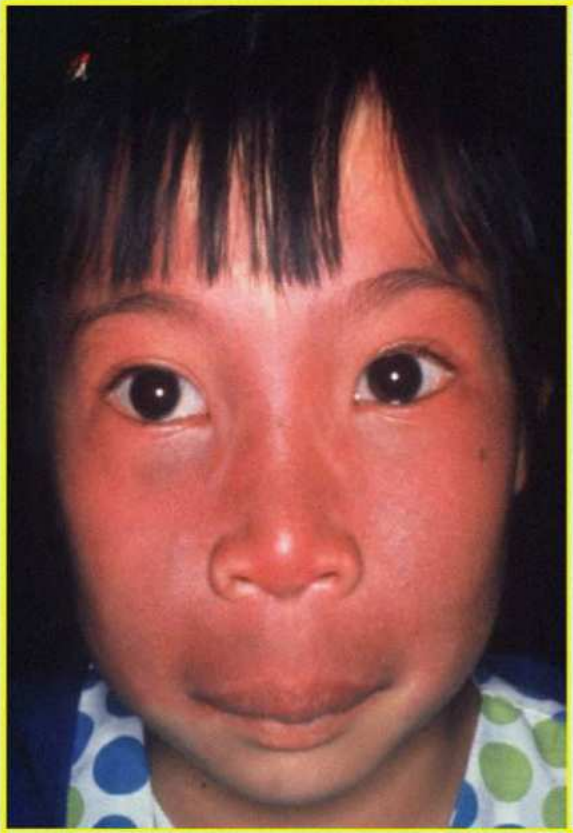
- Ipsilateral glaucoma in 30%
- Sturge-Weber or Klippel-Trenaunay-Weber syndrome in 5%

NAEVUS FLAMMEUS

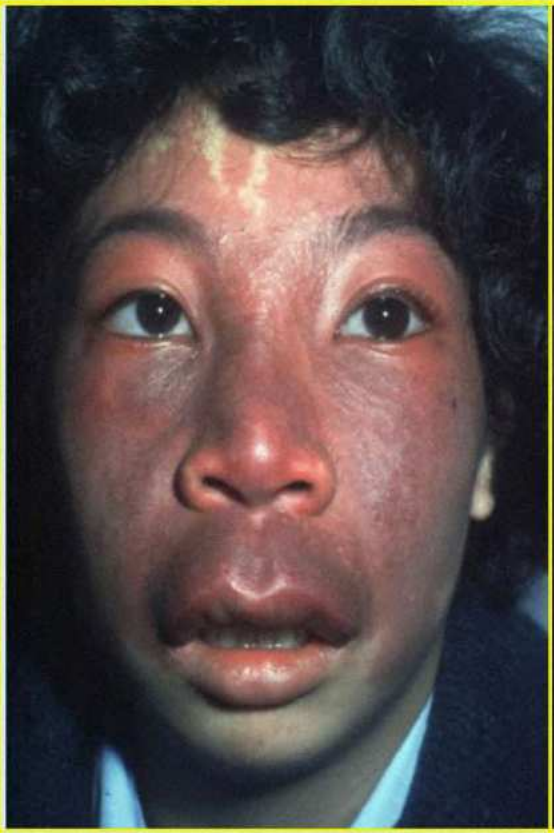




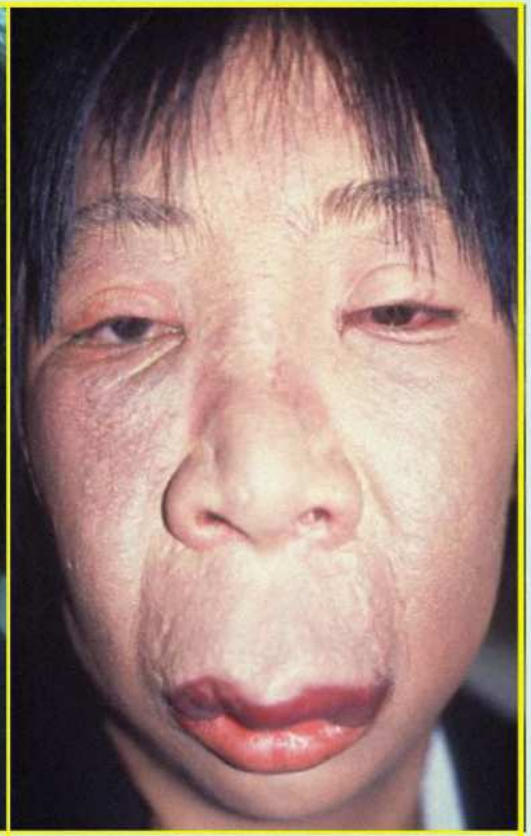
Progression of port-wine stain



Initially red and flat

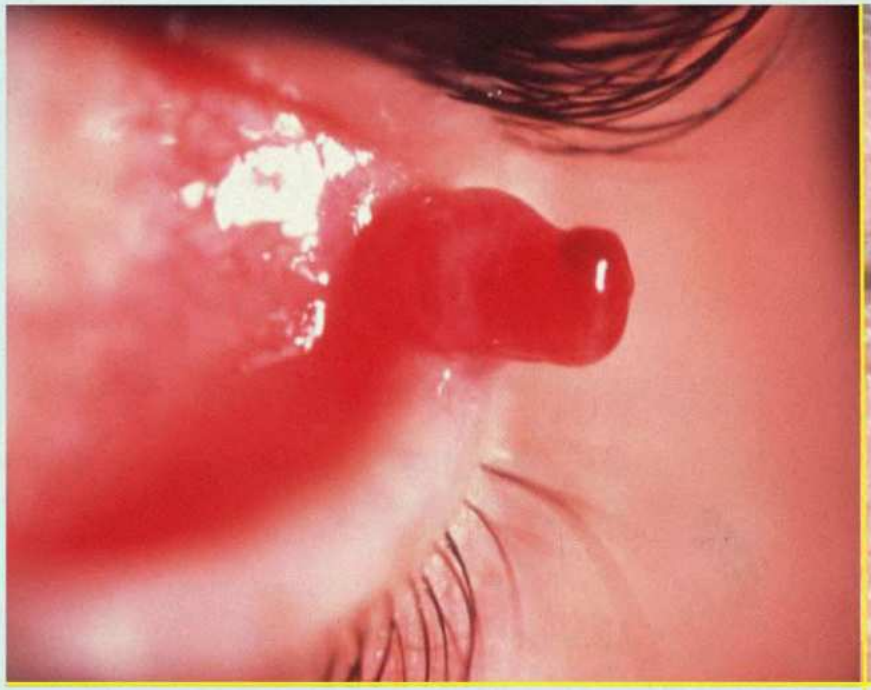


Subsequent darkening and hypertrophy of skin



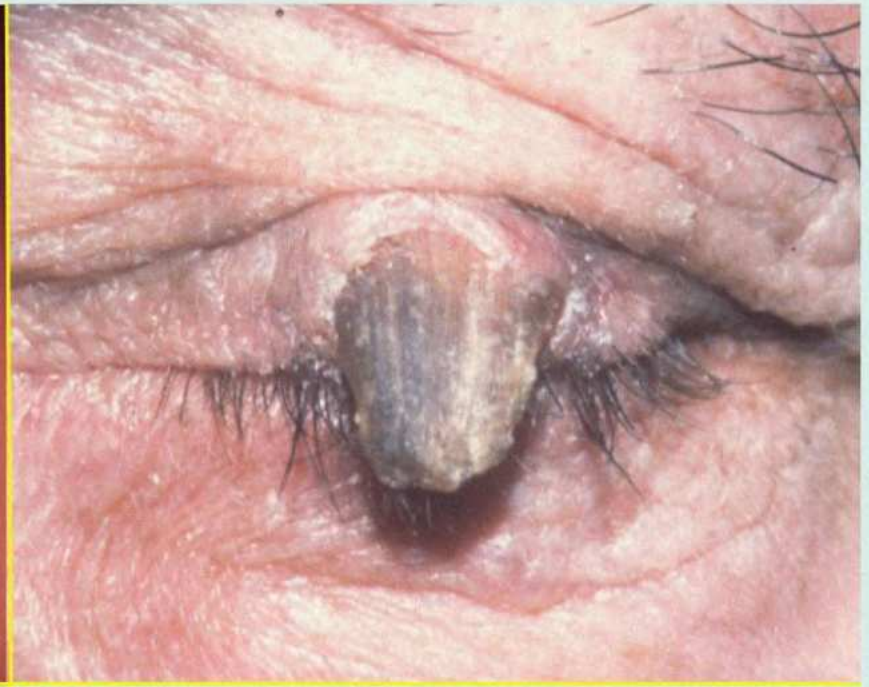
Skin becomes coarse, nodular and friable

Pyogenic granuloma



- Usually antedated by surgery or trauma
- Fast-growing pinkish, pedunculated or sessile mass
- Bleeds easily

Cutaneous horn

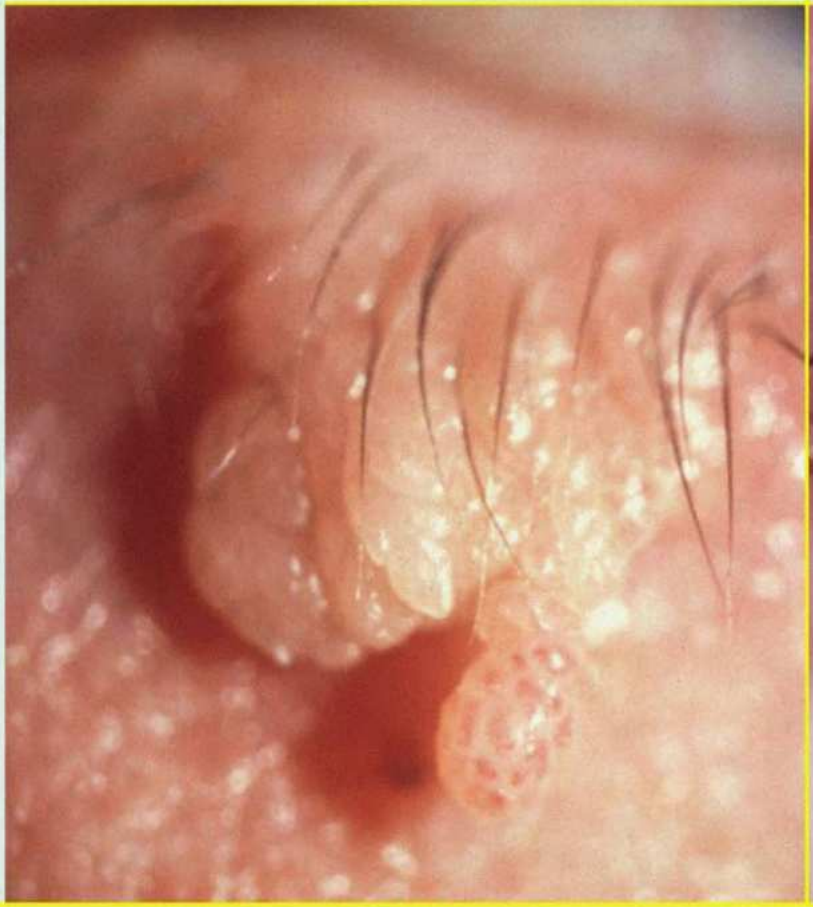


- Uncommon, horn-like lesion protruding through skin
- May be associated with underlying actinic keratosis or squamous cell carcinoma

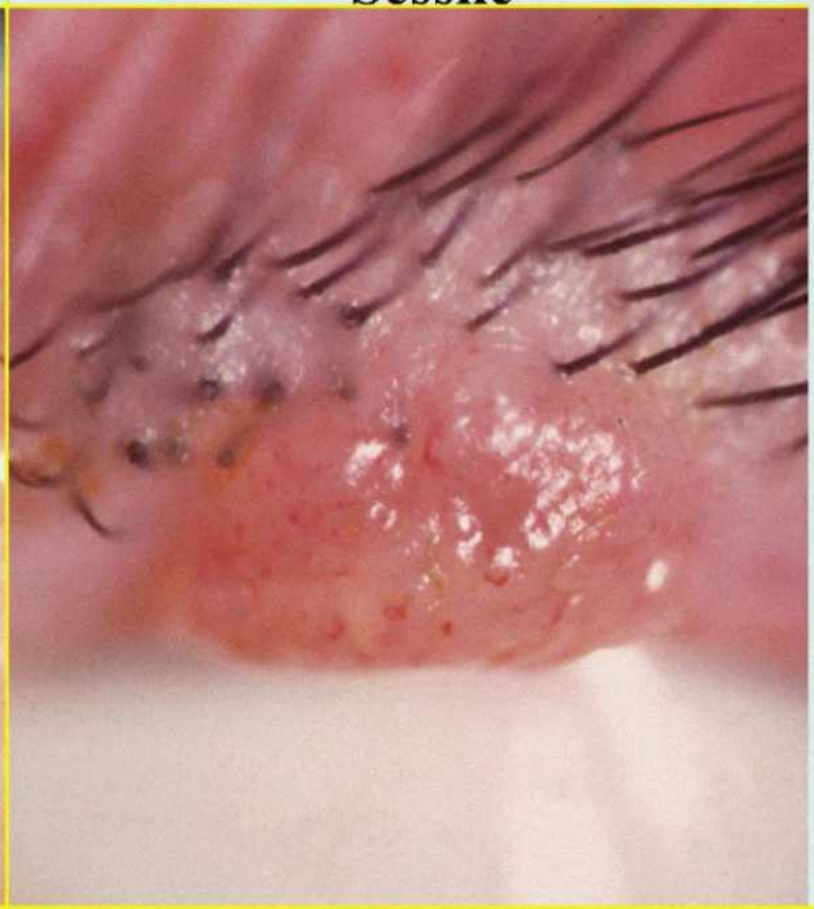
Viral wart (squamous cell papilloma)

- **Most common benign lid tumour**
- **Raspberry-like surface**

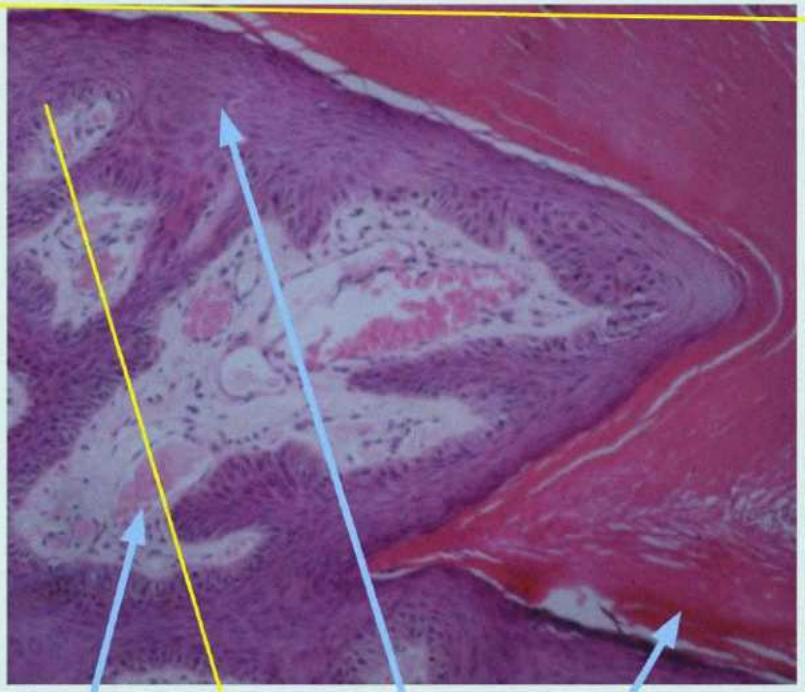
Pedunculated



Sessile



Histology of viral wart

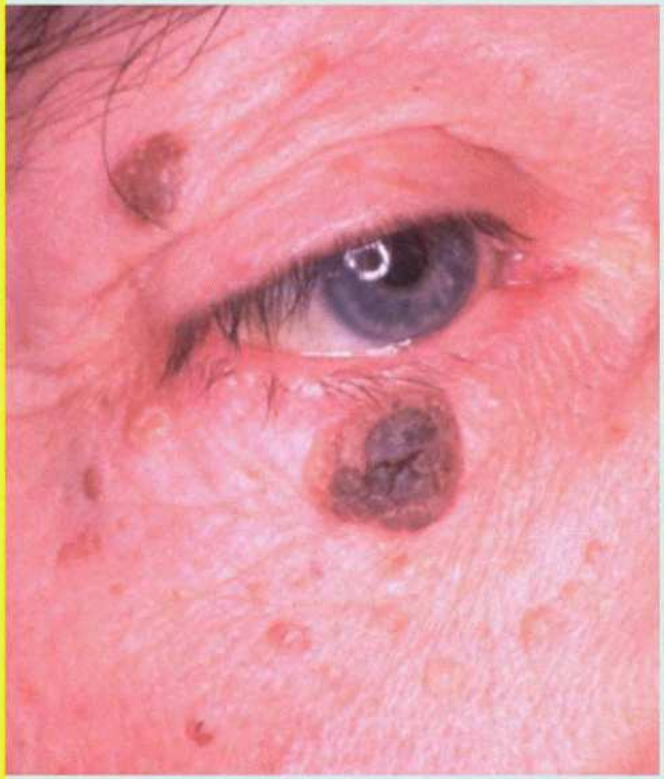


Finger-like projections of fibrovascular connective tissue

Epidermis shows acanthosis (increased thickness) and hyperkeratosis
Rete ridges are elongated and bent inwards

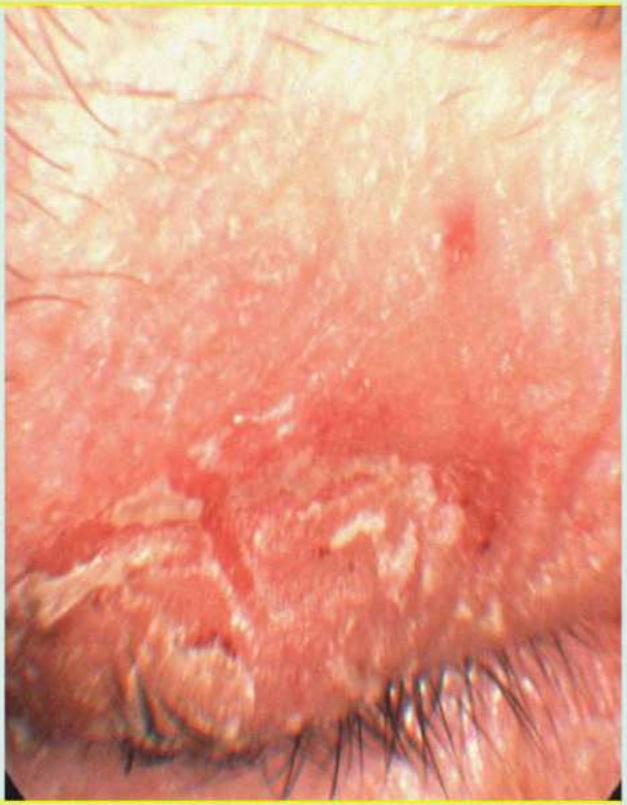
Keratoses

Seborrhoeic



- Common in elderly
- Discrete, greasy, brown lesion
- Friable verrucous surface
- Flat ‘stuck-on’ appearance

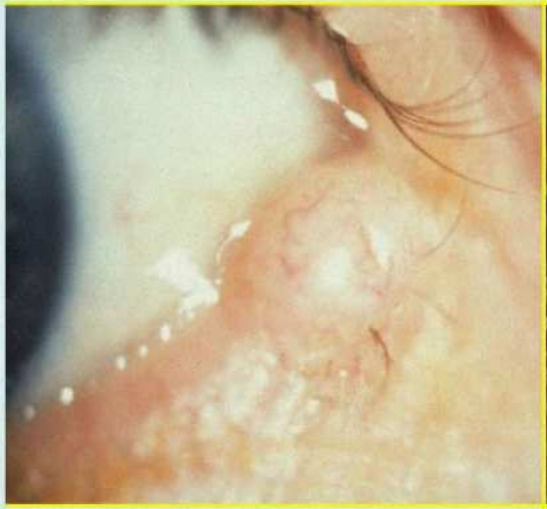
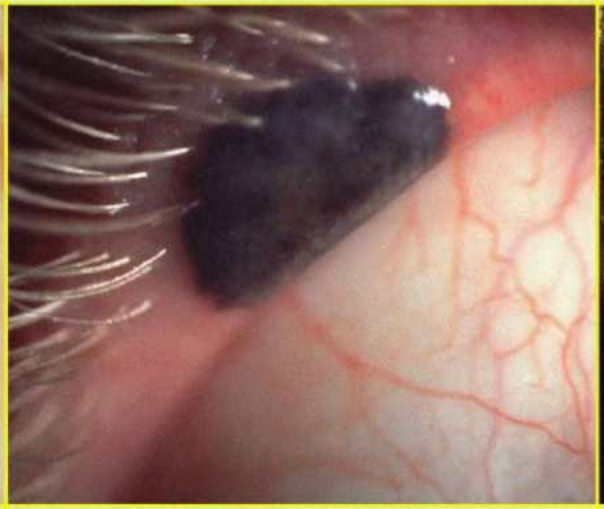
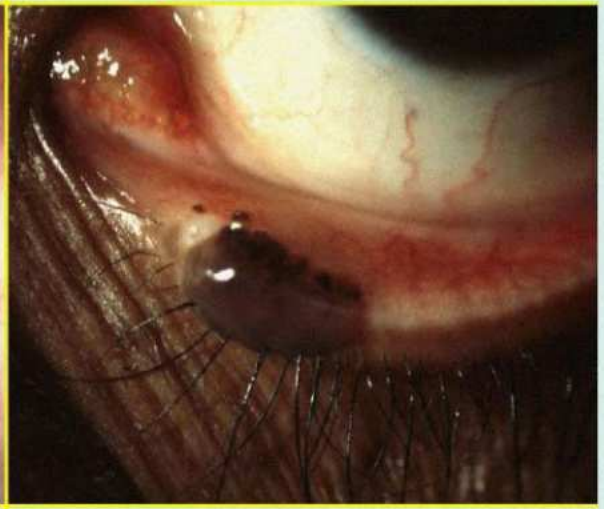
Actinic



- Affects elderly, fair-skinned individuals
- Most common pre-malignant skin lesion
- Rare on eyelids
- Flat, scaly, hyperkeratotic lesion

Naevi

- Appearance and classification determined by location within skin
- Tend to become more pigmented at puberty

Intradermal	Junctional	Compound
		
• Elevated • May be non-pigmented • No malignant potential	• Flat, well-circumscribed • Pigmented • Low malignant potential	• Has both intradermal and junctional components

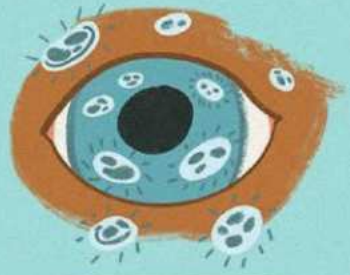
MALIGNANT EYELID TUMORS

1. Basal cell carcinoma
2. Squamous cell carcinoma
3. Meibomian gland carcinoma
4. Melanoma
5. Kaposi sarcoma
6. Merkel cell carcinoma

Eyelid Cancer Symptoms

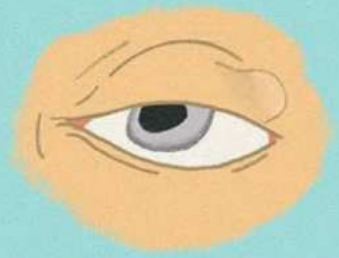
A change in eyelid appearance

Eyelid swelling/ thickening



Chronic eyelid infections

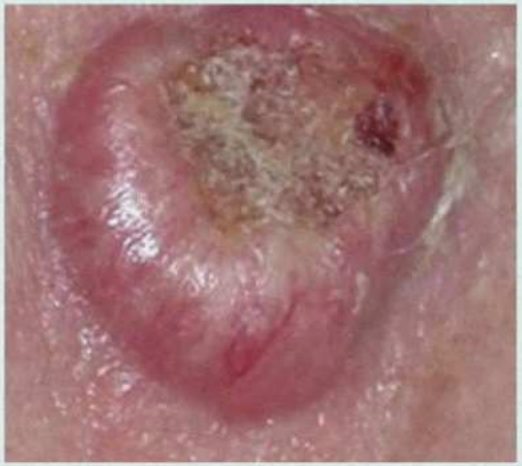
A non-healing eyelid ulceration



A spreading, colorless mass on eyelid

The first detailed description of BCC was that of an eyelid tumor (rodent ulcer, Jacobs 1827)

Jacob's ulcer, chancroid ulcer, ulcus exedens, benign skin cancer, rodent ulcer, and basal cell epithelioma, and noli me tangere (don't touch me or touch me not)

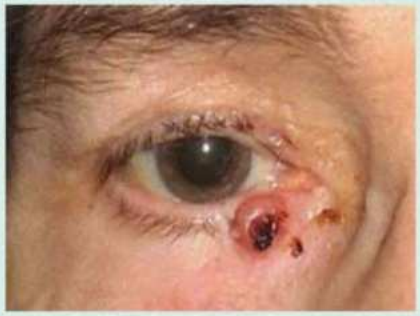


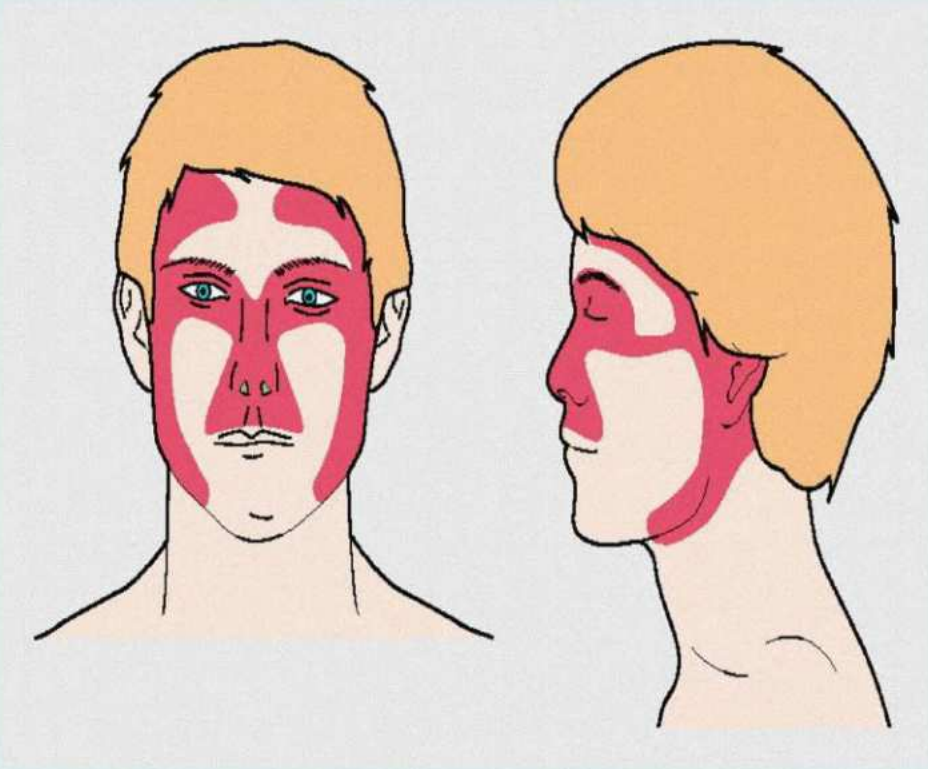
The most common malignancy in humans

Most frequent periocular malignancy accounting for 90% of eyelid malignancies

A slow-growing tumor, and rarely metastasizes but can lead to significant morbidity in the periocular region as a result of orbital invasion or if neglected and treated inadequately

Disease of elderly





90% affect the Head and Neck area

Incidence

- Australia has the highest rate in the world (884/100,000 population/ year)
- The incidence is increasing worldwide by up to 10% a year
- A study of the white population in North America has estimated a lifetime risk of 30% of developing BCC
- 95% of BCCs occur in people aged between 40 and 79 years

References

- *Staples MP, Elwood M, Burton RC, et al: Non-melanoma skin cancer in Australia: the 2002 national survey and trends since 1985. Med J Aust 184: 6-10, 2006*
- *Wong CS, Strange RC, Lear JT: Basal cell carcinoma. BMJ 327: 794-8, 200*
- *Miller DL, Weinstock MA: Nonmelanoma skin cancer in the United States: incidence. J Am Acad Dermatol 30: 774-8, 1994*
- *Kopf AW: Computer analysis of 3531 basal-cell carcinomas of the skin. J Dermatol 6: 267-81, 1979*

ENVOIRNMENTAL

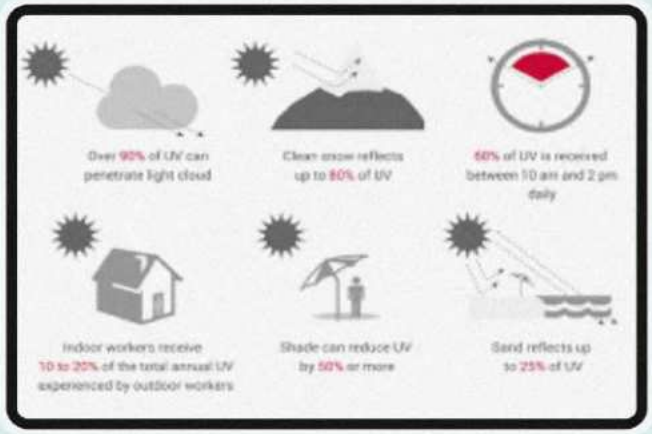
PATIENT FACTORS

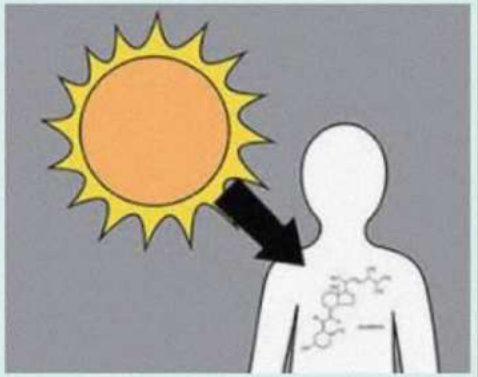
HERIDITARY

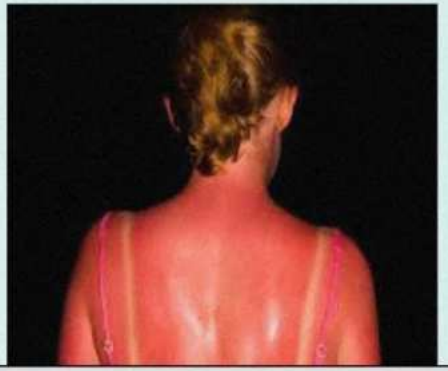
IMMUNOSUPPRESSION

ENVOIRNMENTAL FACTORS

- Exposure to ultraviolet (UV) light
- Exposure to ionizing radiation
- Arsenic
- Therapeutic combination of oral methoxasalen (psoralen) and ultraviolet radiation A (UVA) (known as PUVA)



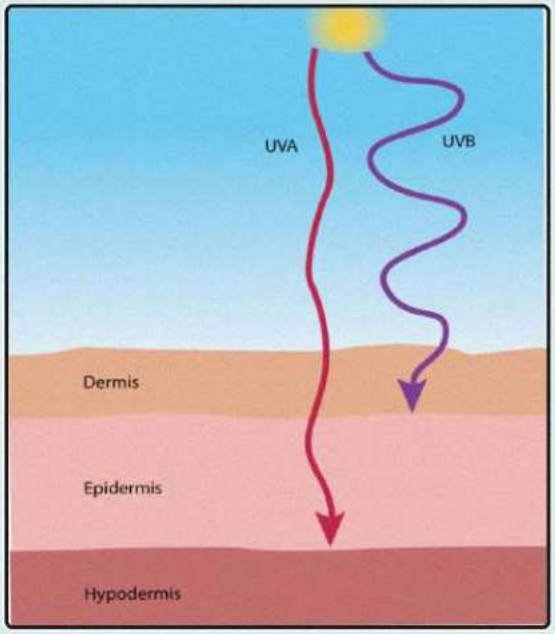


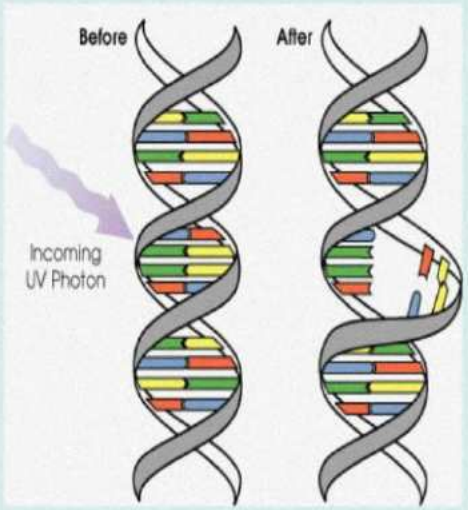


Short-wavelength UVB radiation (290–320 nm, sunburn rays) plays a more important role in BCC formation than long-wavelength UVA radiation

UVB radiation damages DNA and its repair system, and changes the immune system resulting in progressive genetic alterations that lead to the formation of neoplasms

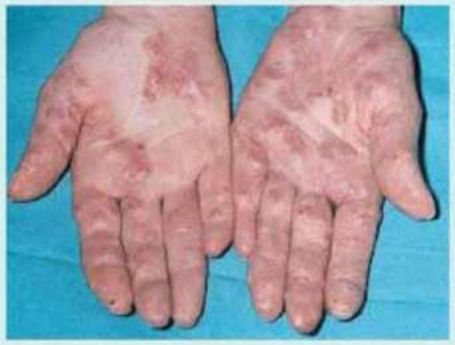
Mutations in the TP53 tumor-suppressor gene induced by UV have been found in about 50% of BCC cases

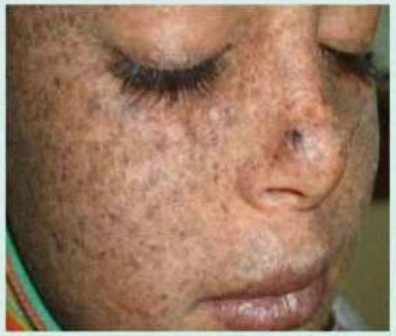


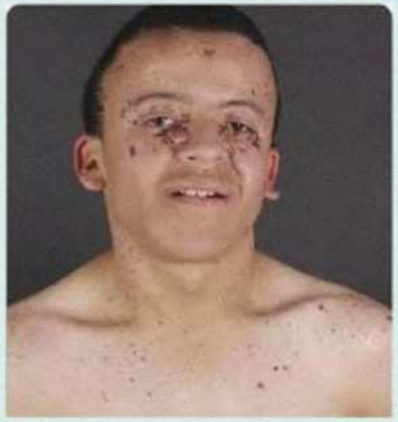


HERIDITARY FACTORS

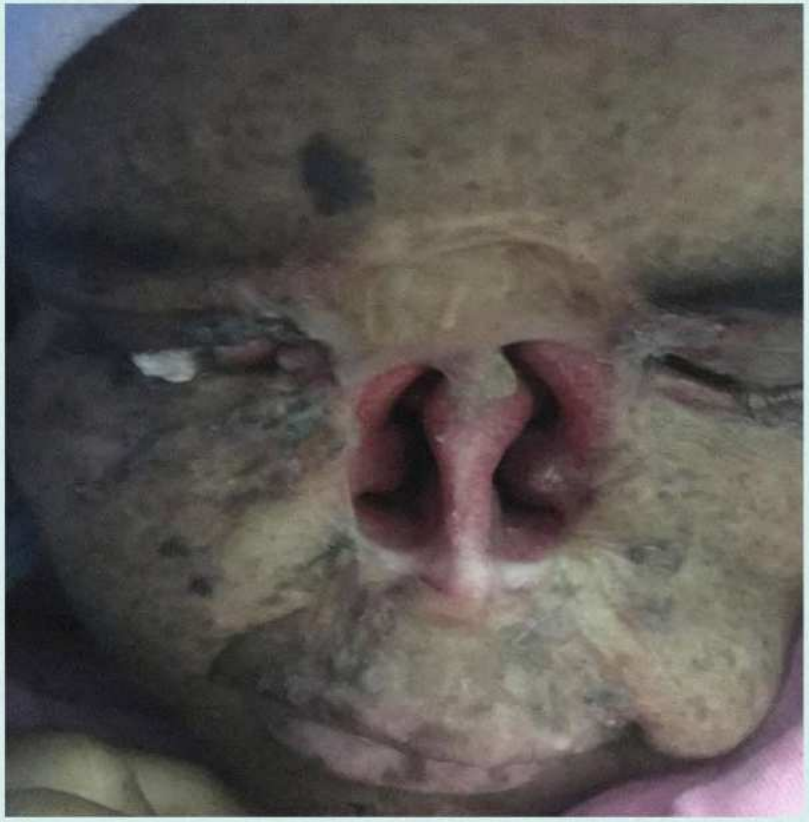
- ***Albinism***
- ***Xeroderma pigmentosum***
- ***Gorlin syndrome***
- ***Rombo syndrome***
- ***Basex syndrome***







XERODERMA PIGMENTOSUM



RISK FACTORS

- ***Immunosuppression***

  HIV, Recipients of solid organ transplants

- ***Patient factors***

  Fair complexion
  Skin that burns and does not tan
  childhood freckles
  cutaneous scars, following burns

GENETICS

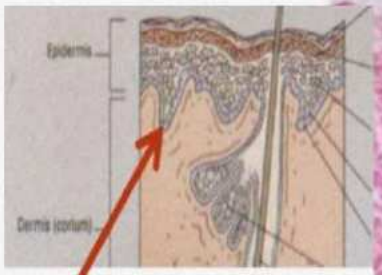
Many BCCs have mutations in the PTCH1 gene, a member of the Hedgehog (Hh) signaling pathway

Hedgehog pathway deregulation results in nuclear accumulation of **beta-catenin**

which in turn increases the transcription of MYC and cyclin D1 genes (involved in cell cycle control) and matrix metalloproteinase 7 gene (involved in stromal degradation).

Beta-catenin accumulation play a role in tumor proliferation and tumor invasion

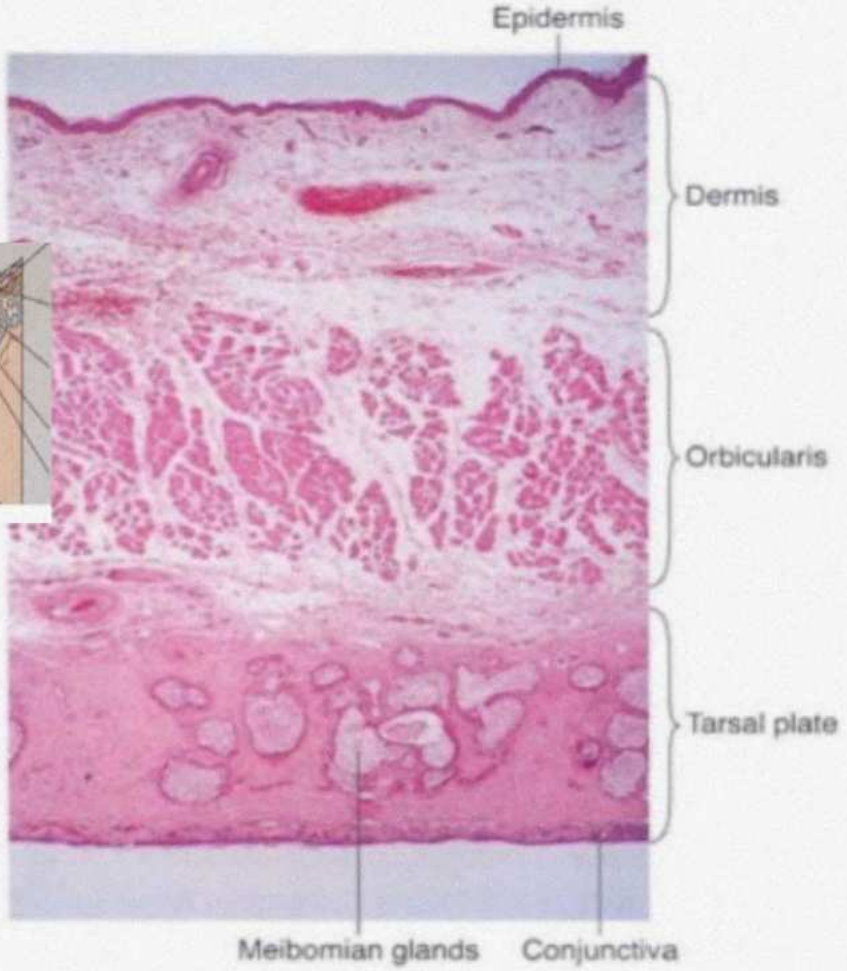
Normal Histology of the Eyelid



Arises from

- Stratum basale
- Outer root sheath of the hair follicle

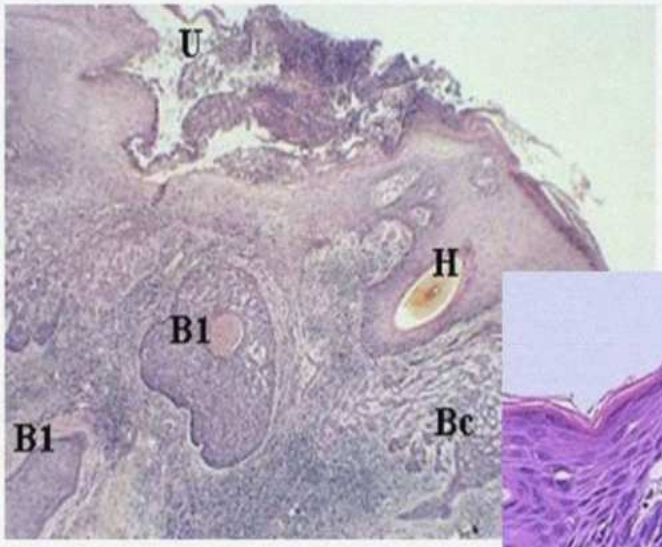
Only in hair-bearing tissue



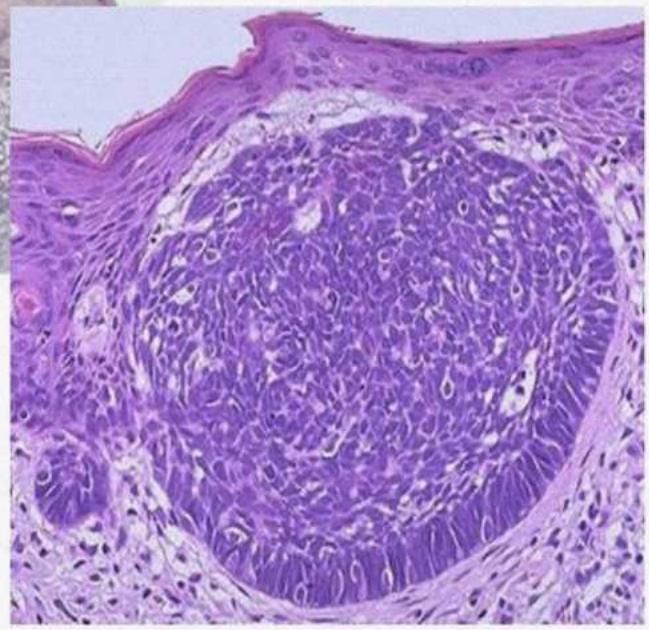
- ***Basal cell carcinoma is characterized by proliferation of basaloid cells arising from the epidermis and invading the dermis***

- ***Classically, palisading of peripheral tumor cells is seen and a clear space is noted between the tumor nodules and the adjacent stroma***

- ***This crack/retraction artefact is considered to be a pathognomonic histological feature of BCC and is thought to be secondary to absence of normal adhesion molecules, such as the bullous pemphigoid antigen in the tumor cells***



Most common malignancy of the eyelid



Blue basaloid tumor cells arranged in nests and cords

www.mrcophth.com

CLASSIFICATION

- No universally agreed classification for BCC
- Two main classification systems exist:

Histopathological growth pattern (morphological)

Histological differentiation

Morphological classification has the greatest clinical significance

MORPHOLOGICAL CLASSIFICATION

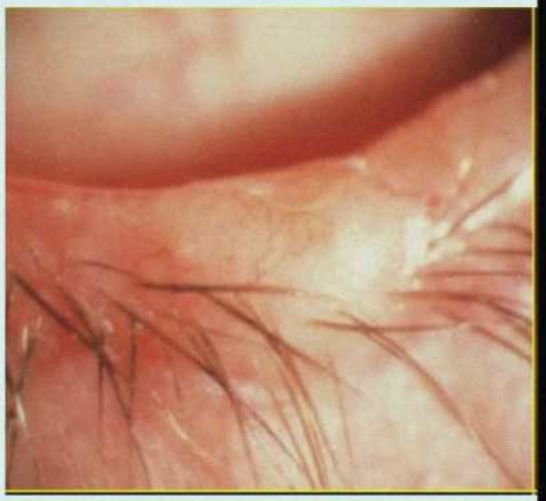
- ***Nodular, including micronodular***
- ***Superficial***
- ***Infiltrative, including morpheic***
- ***Mixed***

- The morphological classification aids in stratifying BCC into low- and high-risk histological subtypes

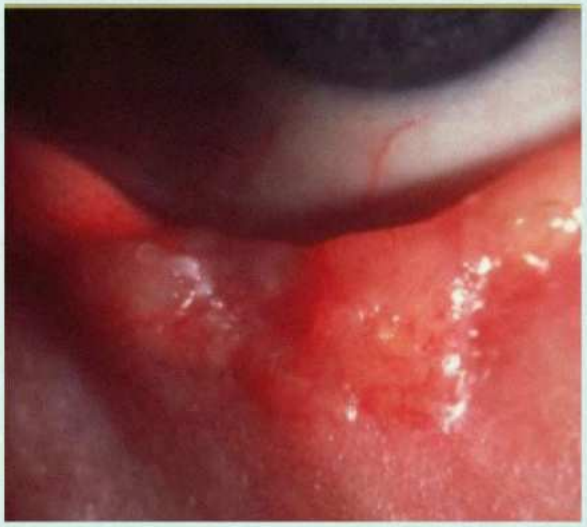
- The high-risk BCCs (infiltrative, morpheic, and micronodular) are characterized by an increased probability of subclinical extension, incomplete excision, aggressive local behavior, and recurrence

NODULAR BCC

- **EARLY**
- ***Shinny nodule with surface vascularization***

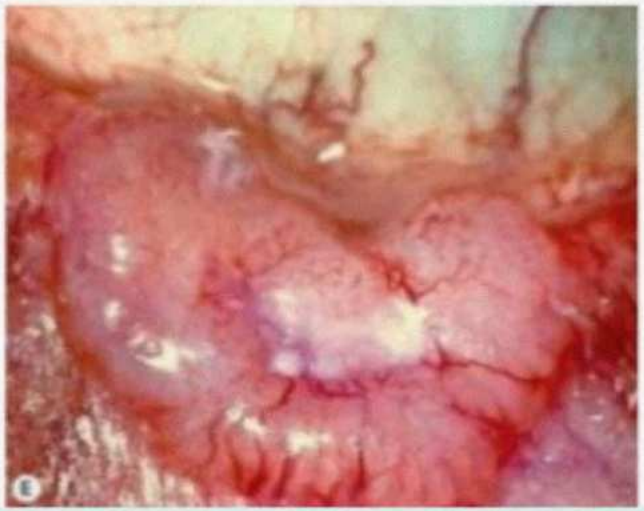


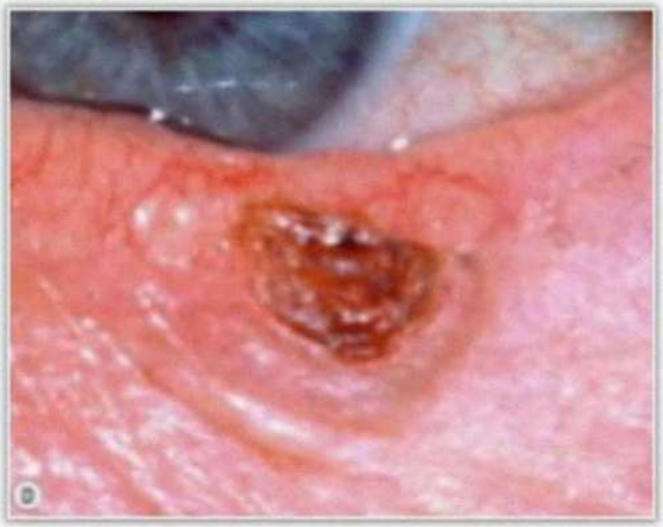
- **LATE**
- ***Slow progression and may destroy a large portion of the eyelid***



RODENT ULCER

- Central ulceration
- Pearly raised rolled edges
- Dilated vessels over its margins
- Telangectasis

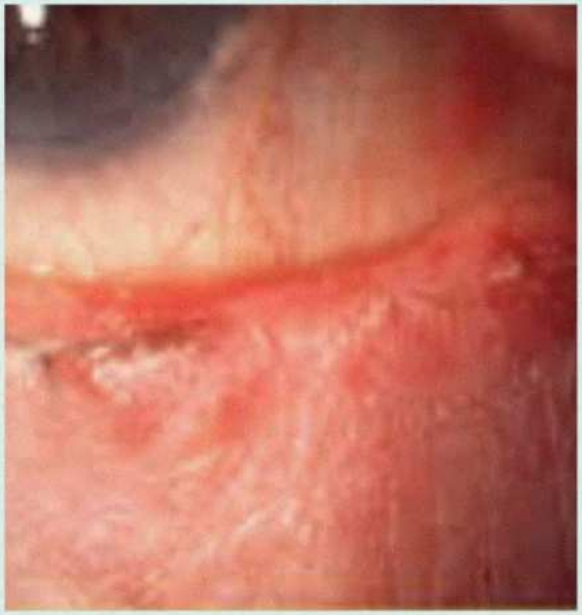




SCLEROSING BCC

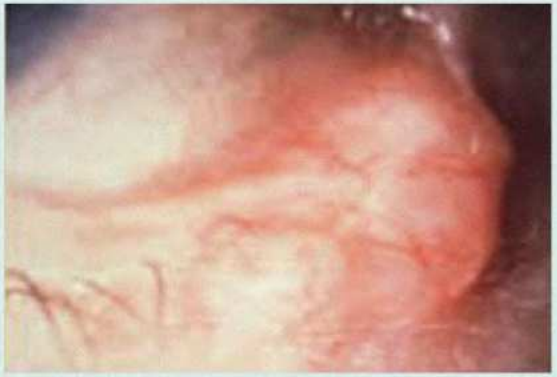
indurate plaque with loss of eyelashes

May mimic chronic blephritis



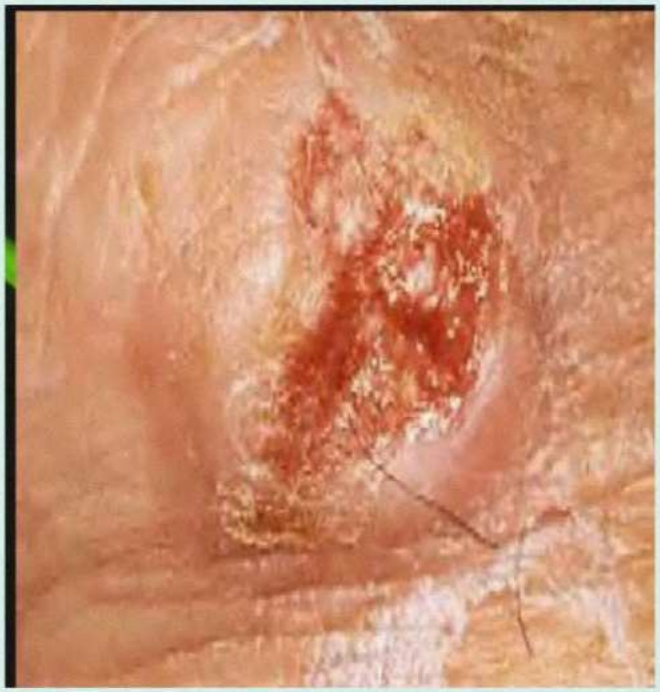
Spread radially beneath the epidermis

Margins are difficult to delineate



NODULO-ULCERATIVE BCC

- Small,slow growing
- Firm
- Telangectasias
- Ulceration



HISTOLOGICAL CLASSIFICATION

- 20 subtypes have been described

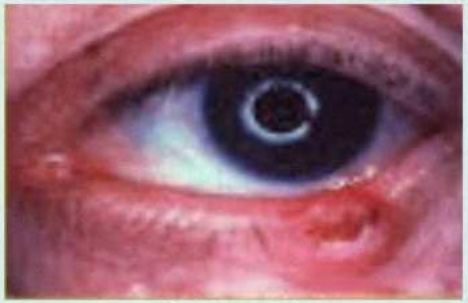
  Except for squamous differentiation the other subtypes have no clinical significance

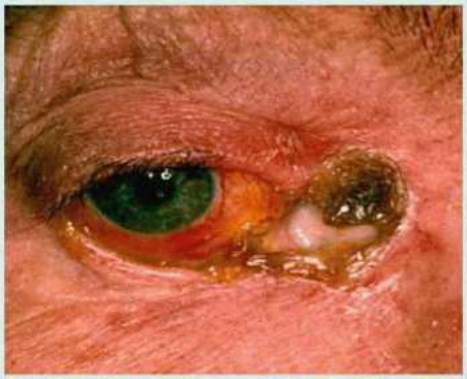
  The term ***basosquamous carcinoma or metatypical*** ,is there is presence of squamous differentiation in a BCC and is associated with a higher incidence of recurrence and metastasis

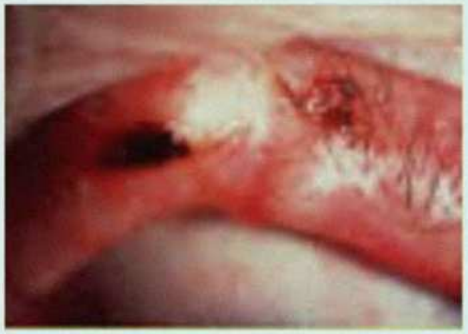
- The presence of perineural invasion on histopathology is associated with a poorer prognosis

Frequency of location of BCC

- Lower lid 70%
- Medial Canthus 15%
- Upper lid 10%
- Lateral Canthus 5%





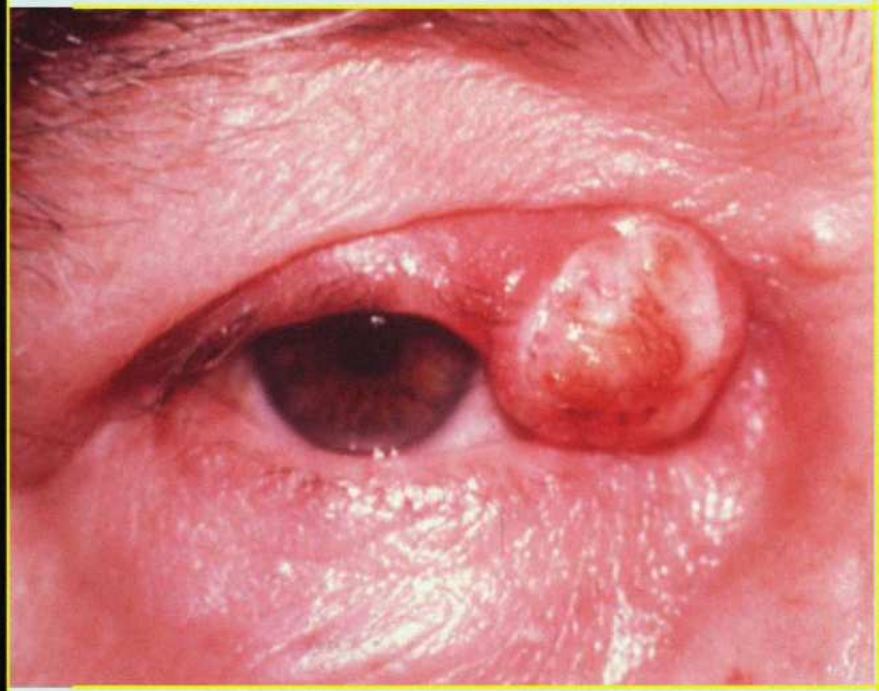
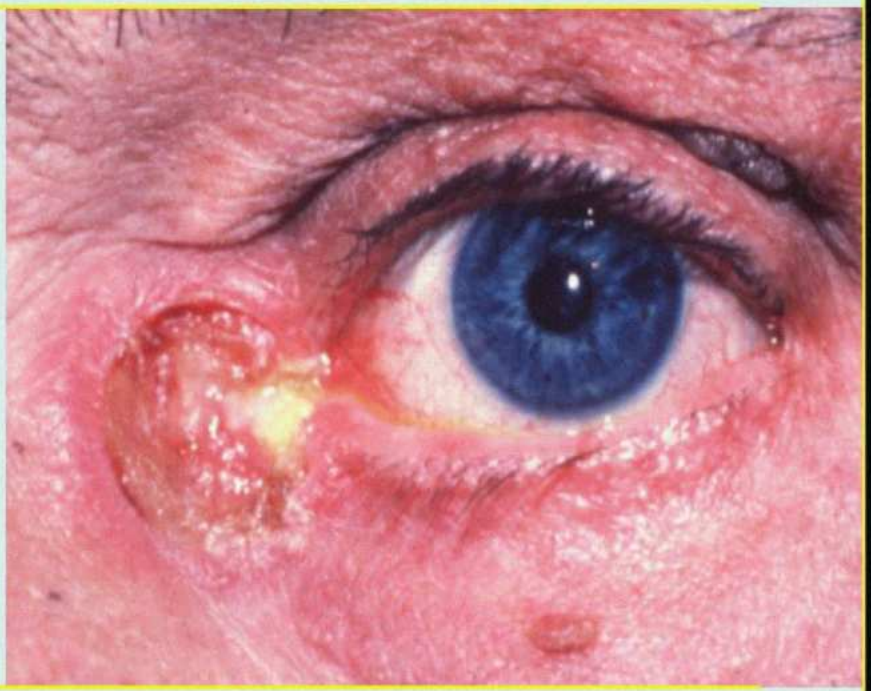


Squamous Cell Carcinoma (SCC)

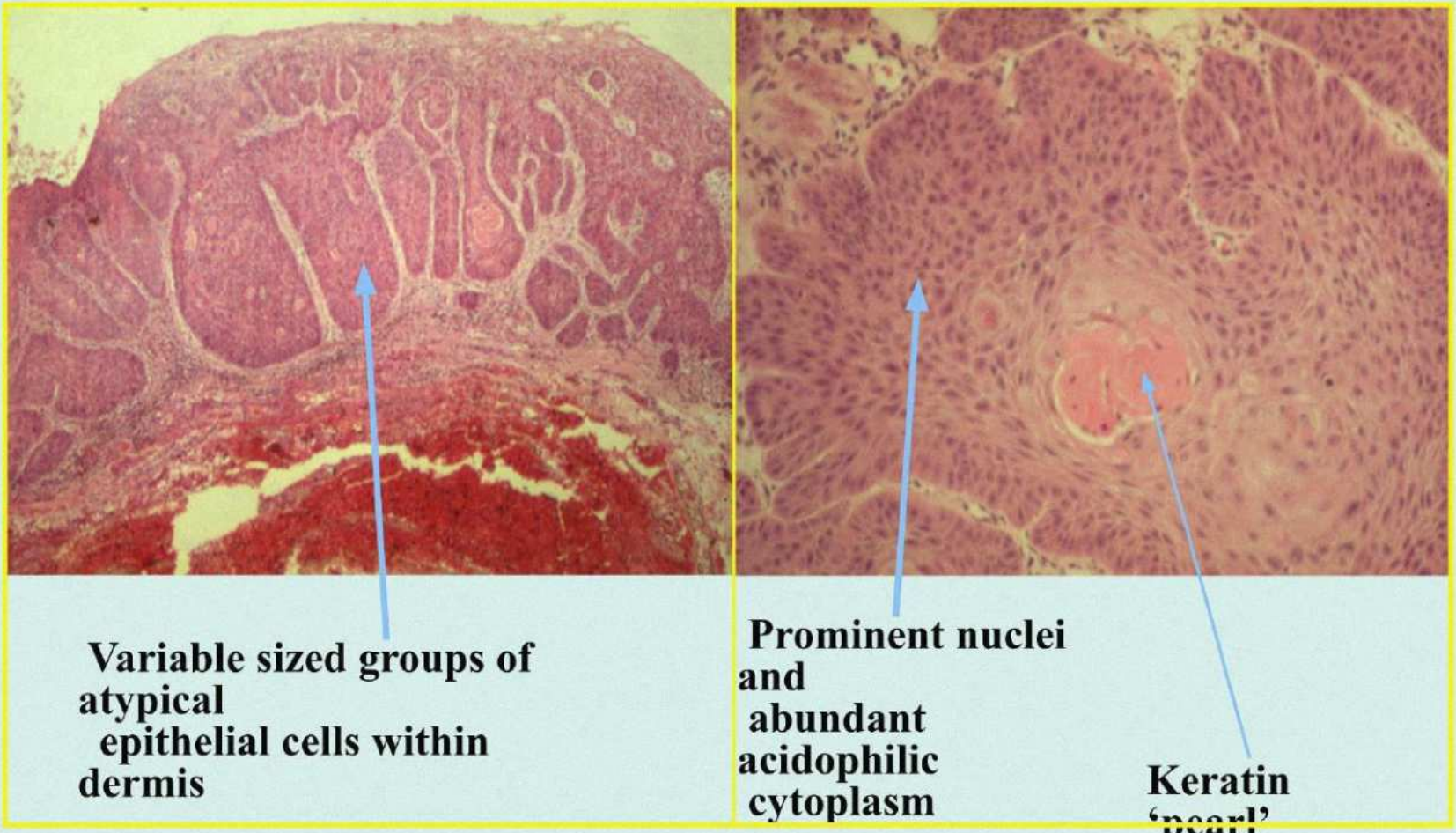
- **Squamous cell carcinoma (SCC)** is a malignant neoplasm of keratinizing epidermal cells.
- It frequently occurs on sun-exposed skin or at the base of skin lesion.
- SCC is less common than BCC.
- SCC can be highly aggressive, has the potential to metastasize, and may lead to death if not treated early and correctly.

Squamous cell carcinoma

- Less common but more aggressive than BCC
- May arise *de novo* or from actinic keratosis
- Predilection for lower lid

Nodular	Ulcerative
	
• Hard, hyperkeratotic nodule • May develop crusting • Surface vascularization	• Red • Borders sharply defined, indurated and elevated

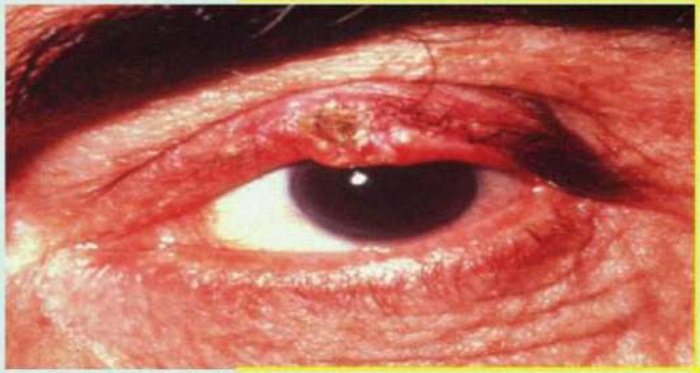
Histology of squamous cell carcinoma



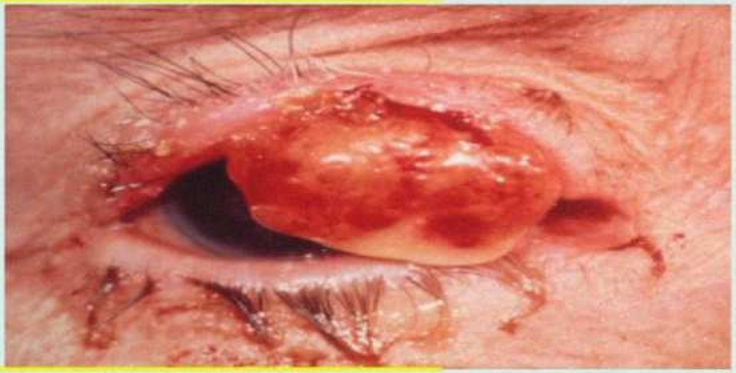
Meibomian gland carcinoma

- Very rare aggressive tumour with 10% mortality
- Predilection for upper lid

Nodula

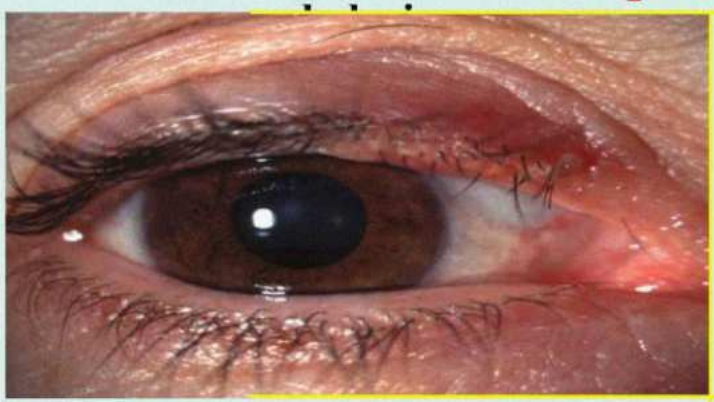


Hard nodule; may mimic a

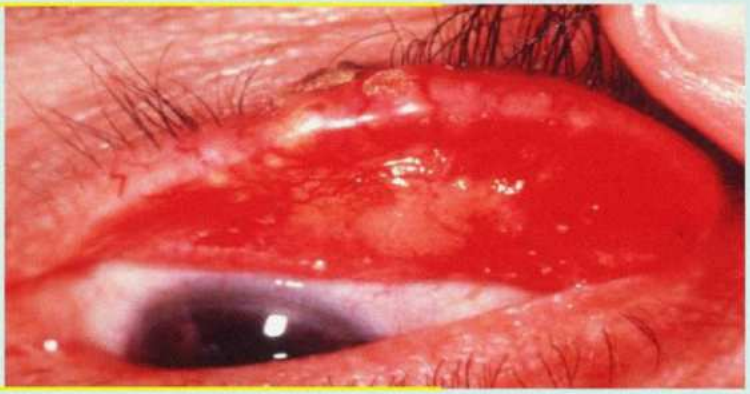


Very large tumour

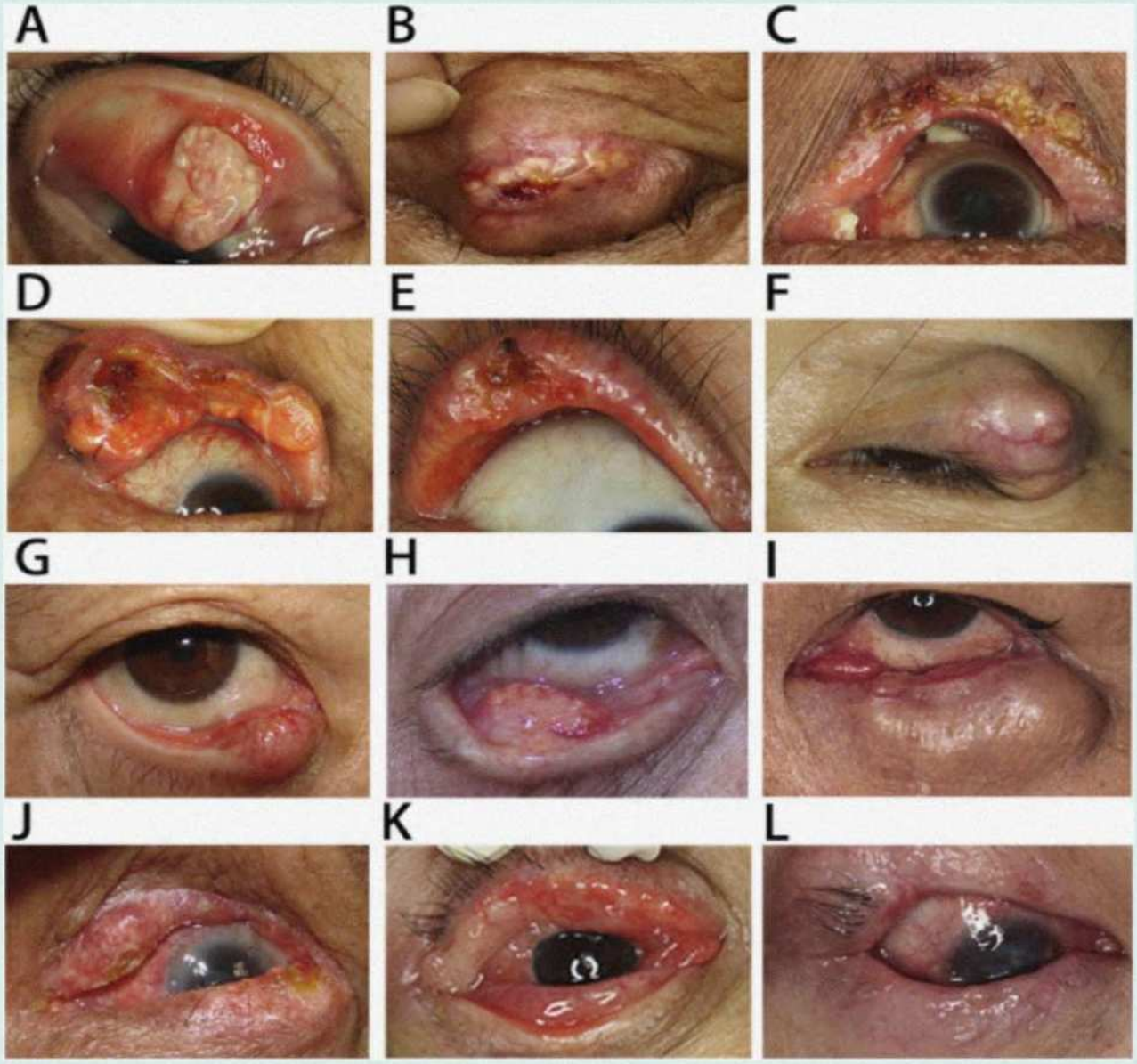
Spreadi



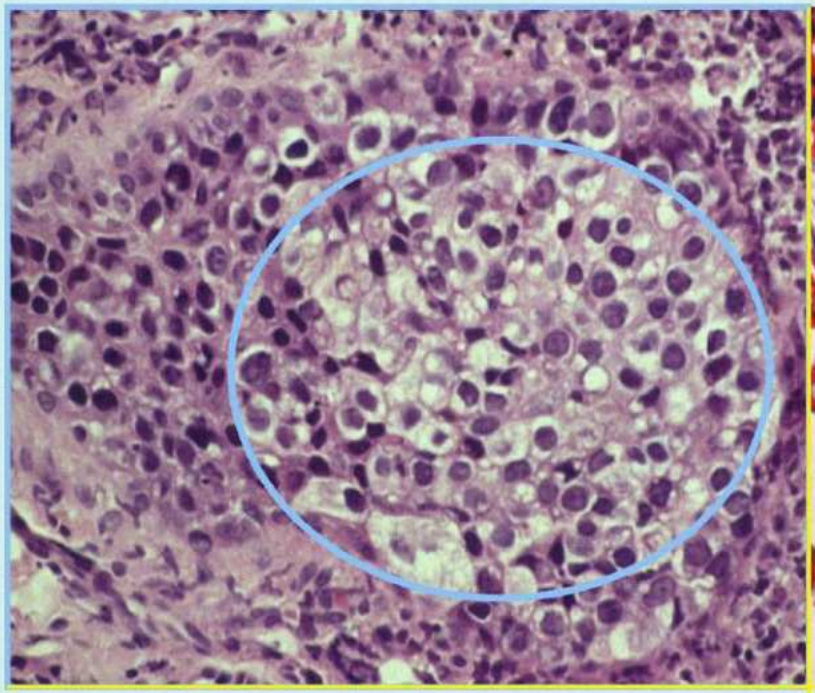
Diffuse thickening of lid margin and loss of lashes



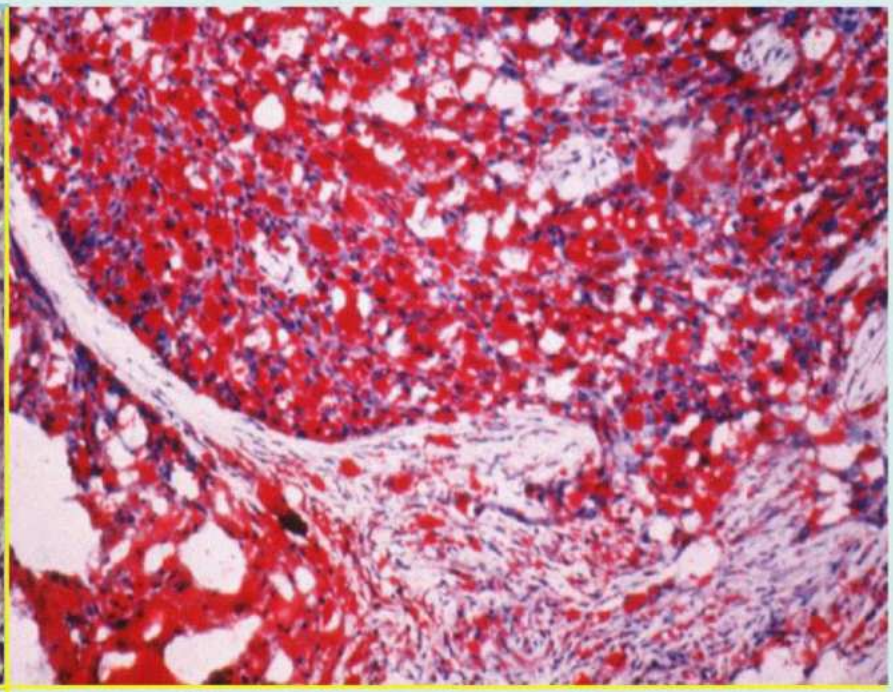
Conjunctival invasion; may mimic chronic conjunctivitis



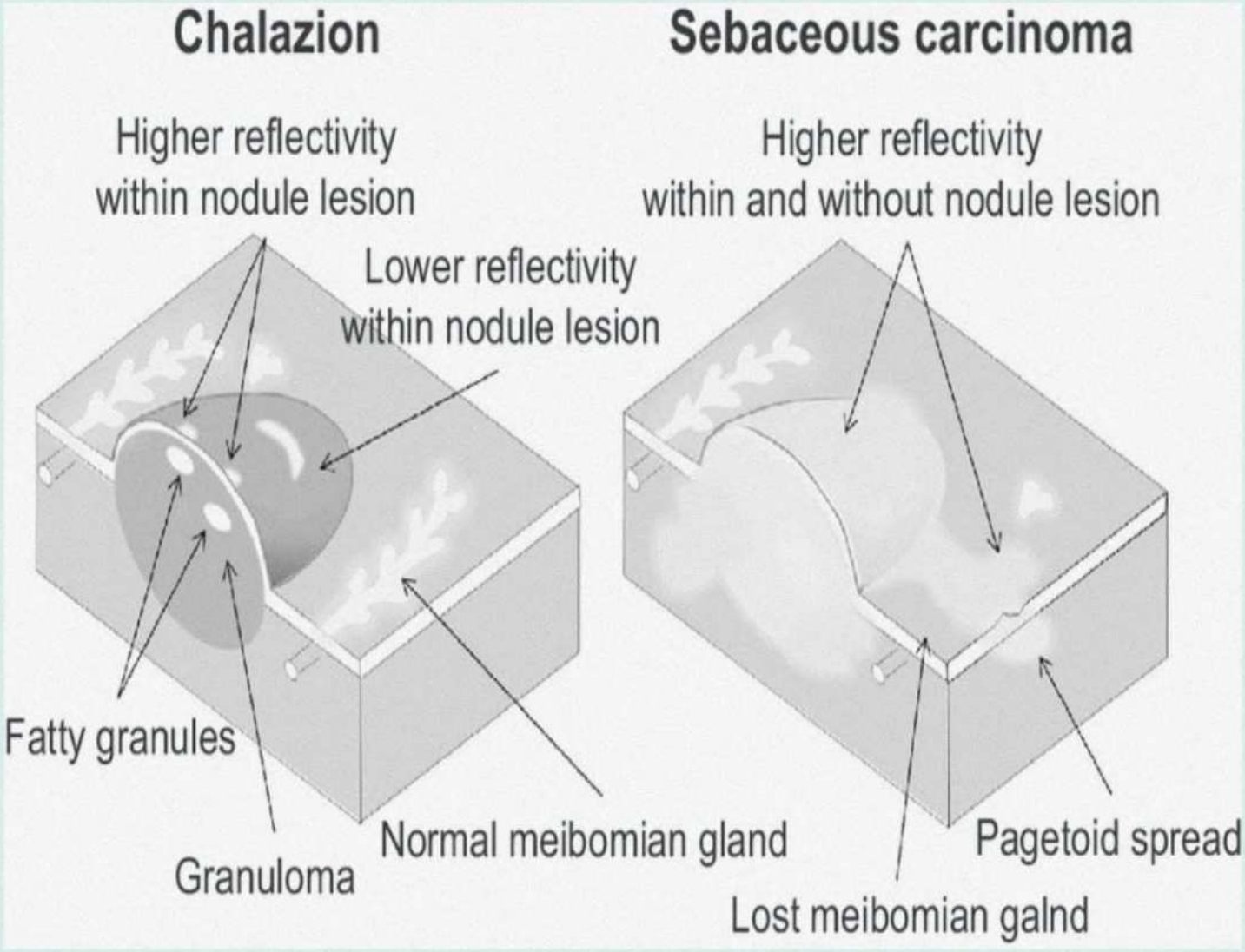
Histology of meibomian gland carcinoma



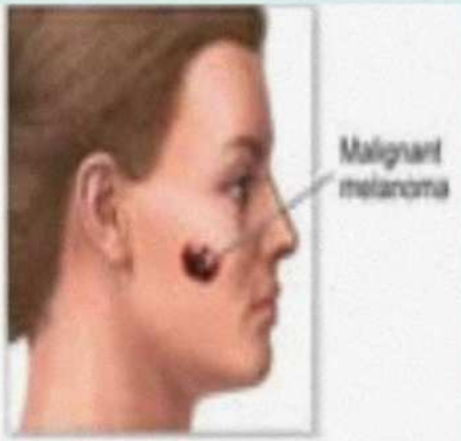
Cells contain foamy vacuolated cytoplasm and large hyperchromatic nuclei

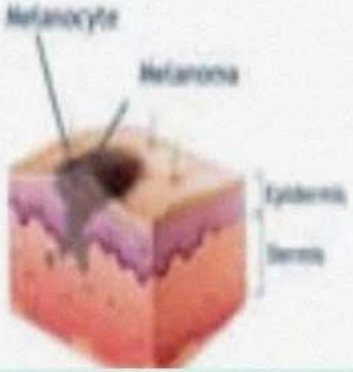


Cells stain positive for fat

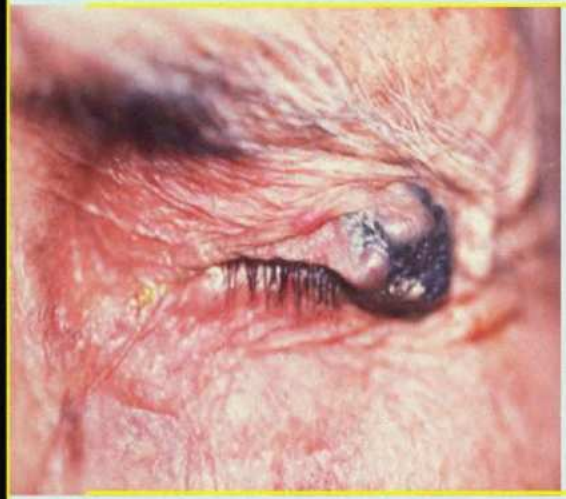
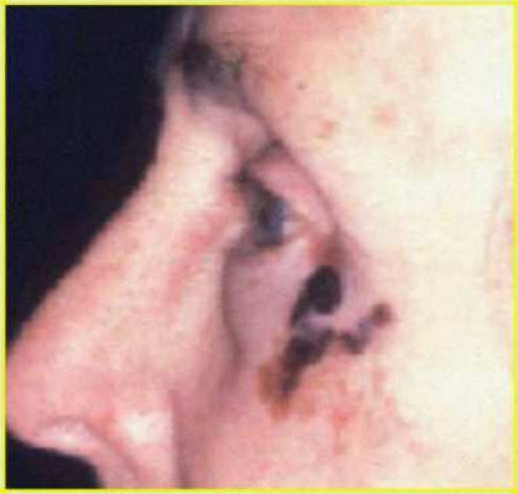


- *Melanoma* is a very serious form of skin cancer.
- Melanoma is cancer of the *melanocytes*.
- Melanocytes are located in the *Stratum Basale* and produce *melanin*.



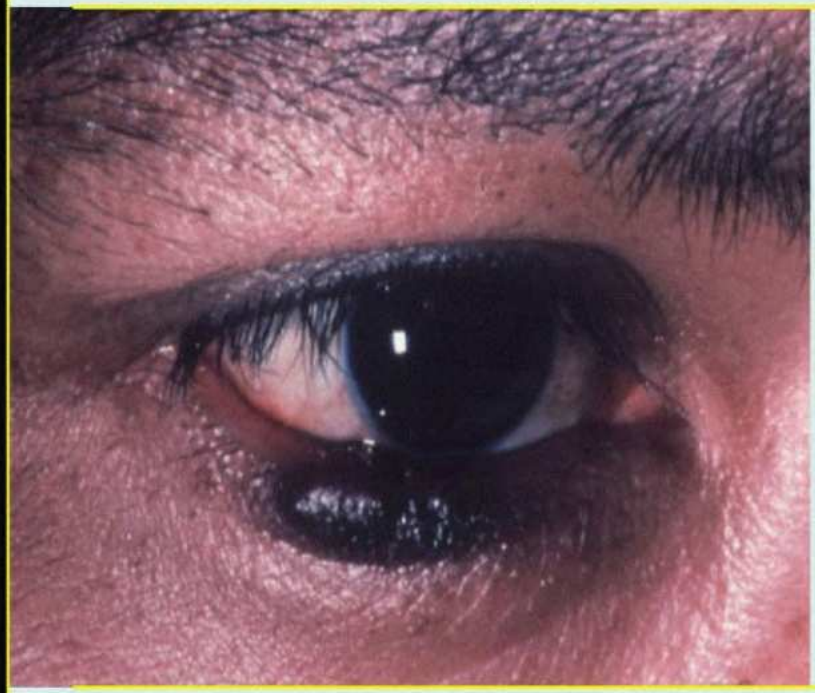
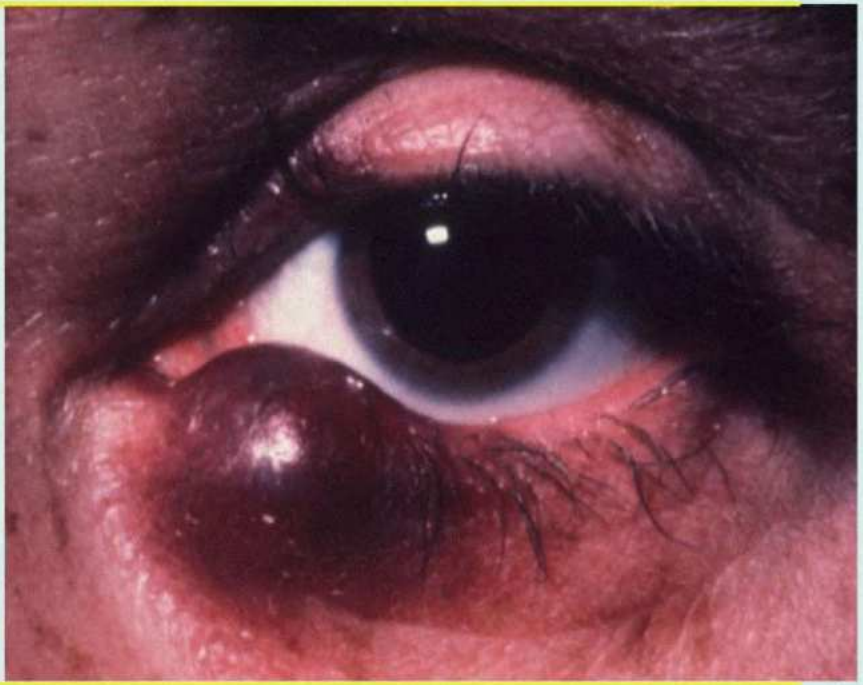


Melanoma

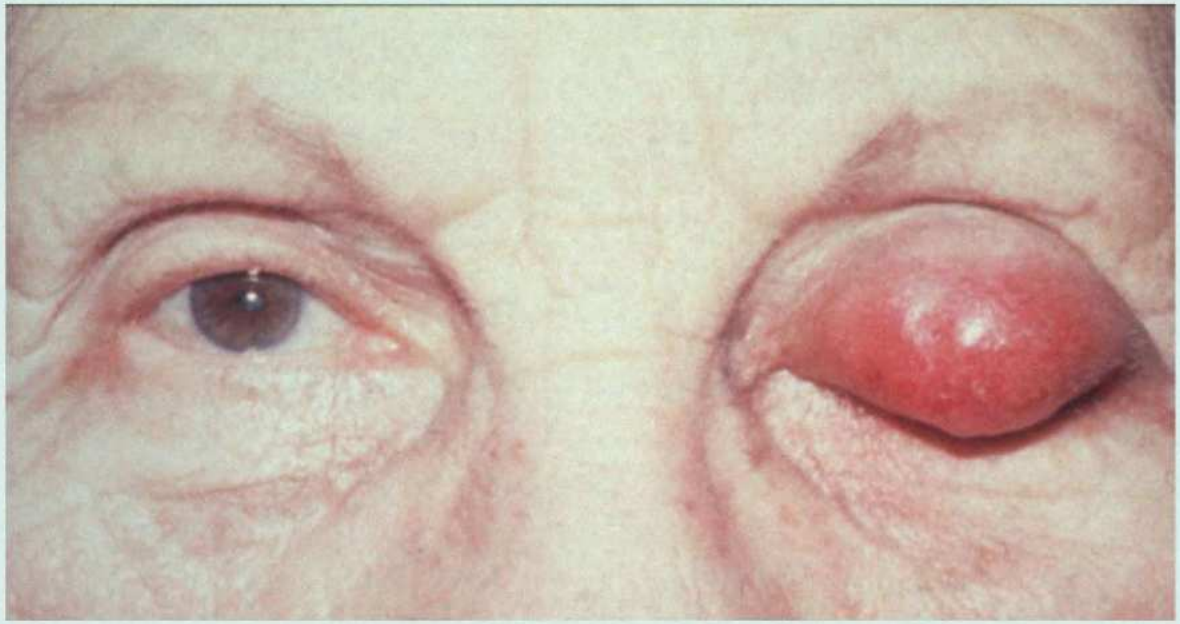
Nodular	Superficial spreading	From lentigo maligna (Hutchinson freckle)
		
• Blue-black nodule with normal surrounding skin • May be non-pigmented	• Plaque with irregular outline • Variable pigmentation	• Affects elderly • Slowly expanding pigmented macule

Kaposi sarcoma

- Vascular tumour occurring in patients with AIDS
- Usually associated with advanced disease
- Very sensitive to radiotherapy

Early	Advanced
	
Pink, red-violet lesion	May ulcerate and bleed

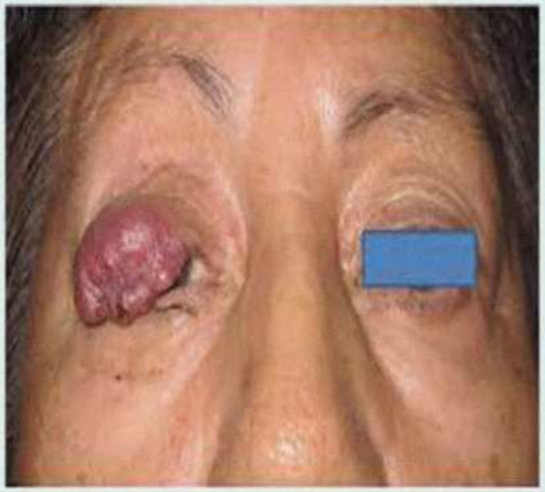
Merkel cell carcinoma



- Highly malignant with frequent metastases at presentation
- Fast-growing, violaceous, well-demarcated nodule

- Intact overlying skin
- Predilection for upper eyelid

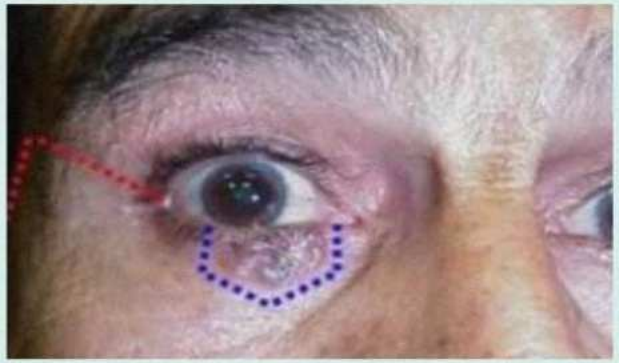
MERKEL CELL CARCINOMA



DIAGNOSIS

- ***The gold standard of diagnosis is surgical excision followed by Histopathology***

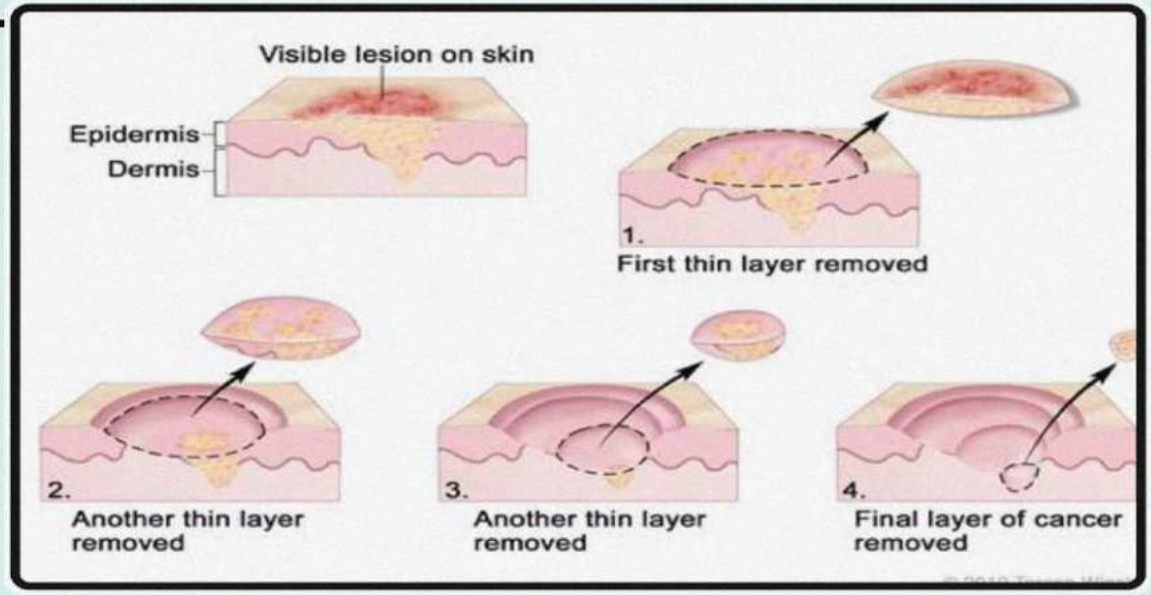
- *Surgical excision is done with 3-5mm of normal skin from the outer edge of tumor using Mohs micrographic surgery or wide surgical excision with frozen section margin control*



Mohs micrographic surgery

Involves removing a skin cancer one layer at a time and examining these layers under a microscope immediately after they are removed

This procedure allows for a close examination of each layer of skin to detect cancer cells



Management

"Ulcers lasting a year or longer cause the underlying bone to be eaten away and the resulting scars are depressed.' 'What drugs will not cure, the knife will; what the knife will not cure, the cautery will; what the cautery will not cure must be considered incurable."

Hippocrates' book of Aphorisms **(46 BC)**

MANAGEMENT

- ***Surgery followed by reconstruction***
- ***Radiotherapy***
- ***Cryotherapy***
- ***Chemotherapeutic and immune-modulating agents***

Treatment Options

1. **Surgical excision**
   - Method of choice
2. **Radiotherapy**
   - Small BCC not involving medial canthus
   - Kaposi sarcoma
3. **Cryotherapy**
   - Small and superficial BCC irrespective of location
   - Adjunct to surgery in selected cases

Chemotherapeutic and immune modulating agents

Topical fluorouracil is approved by the FDA for the treatment of superficial BCC

The first Hh pathway inhibitor

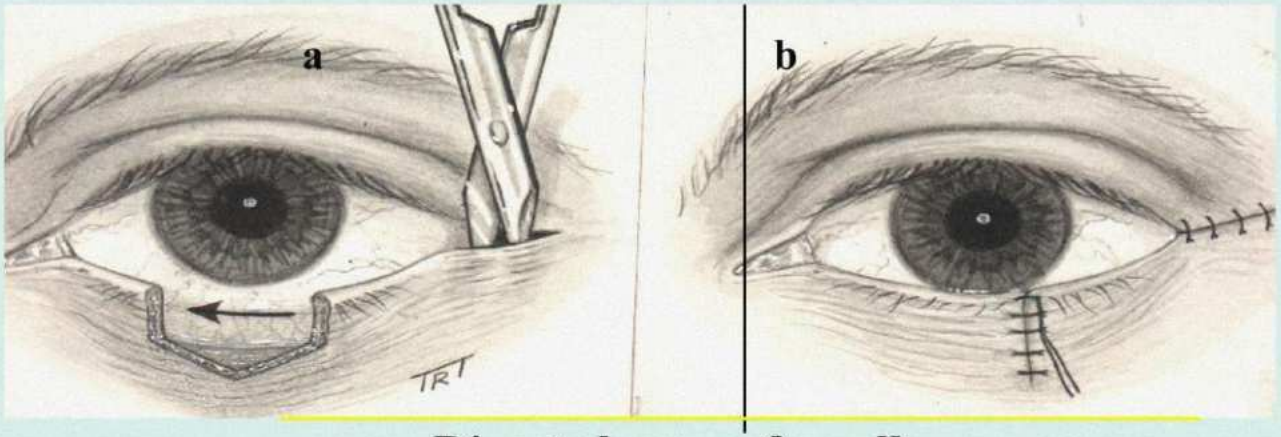
Used topically for prophylaxis or maintenance in patients who are prone to having many BCCs, likely by treating subclinical tumors especially on the trunk and extremities

SURGICAL TREATMENT

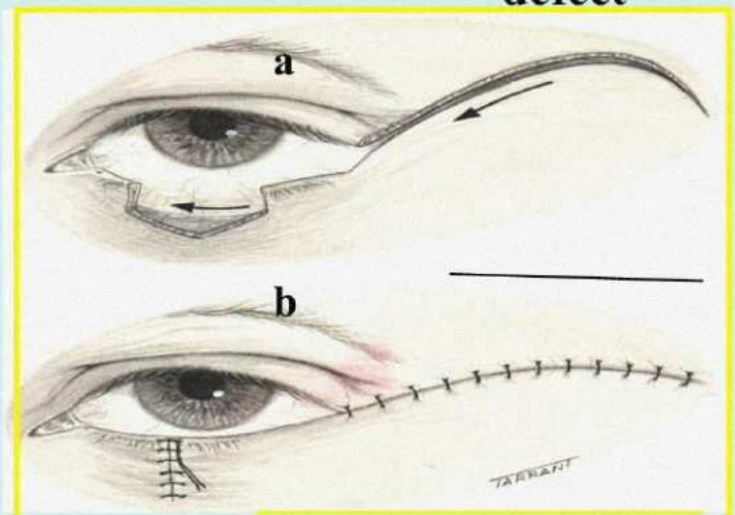
- ***Eyelid reconstruction should be carefully considered as both function and aesthetic outcome in patients are important after clear excision of tumors***

- ***Exenteration is considered in the case of extensive orbital invasion or high-risk aggressive tumors in order to reduce the rate of recurrence***

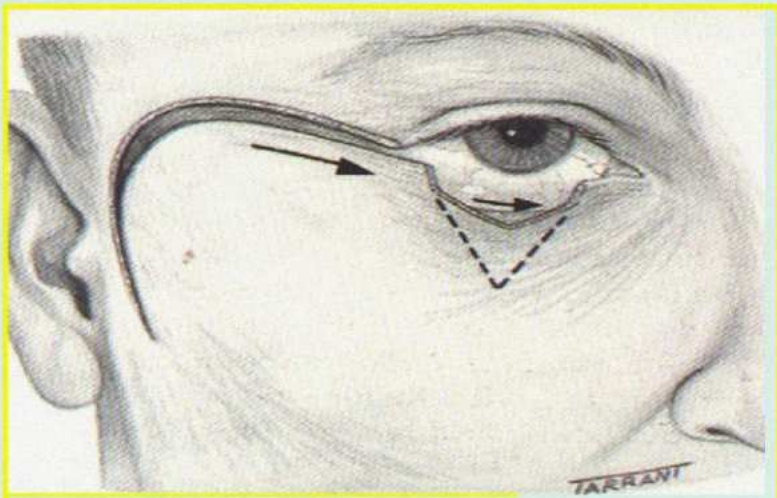
Lower eyelid reconstruction following tumour excision



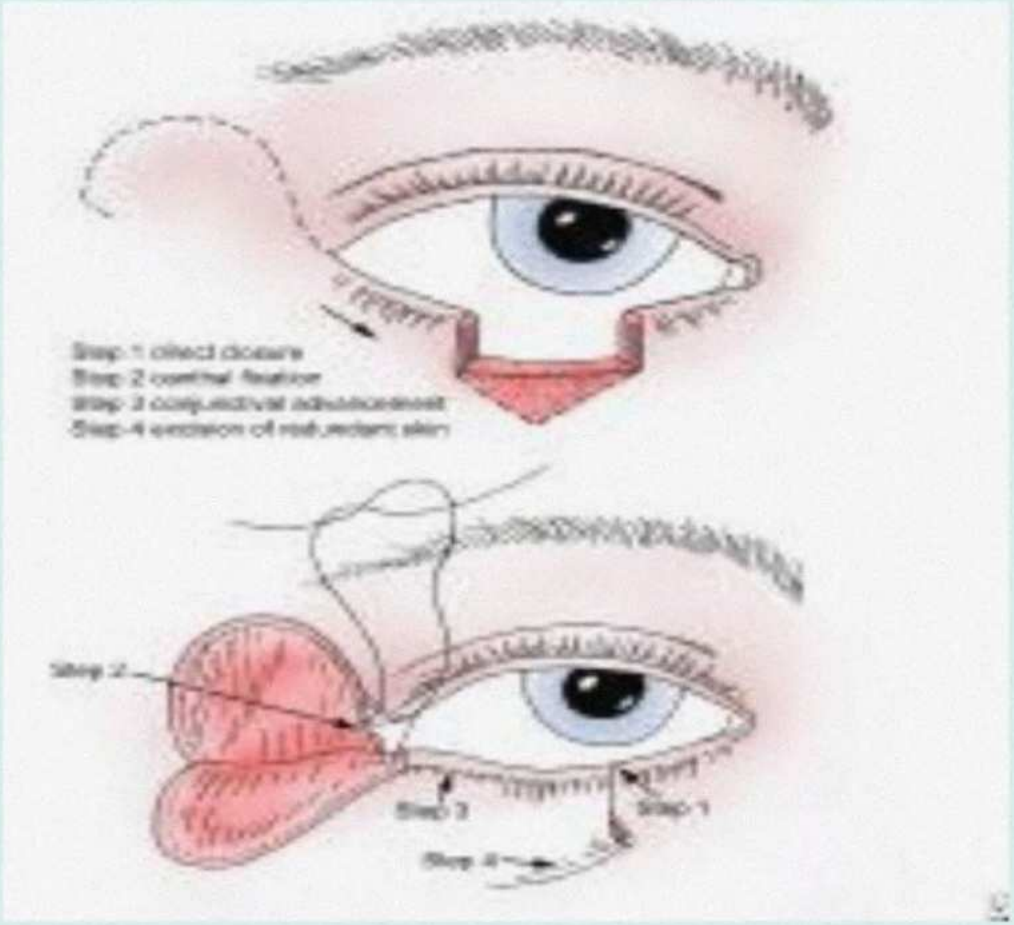
Direct closure of small defect

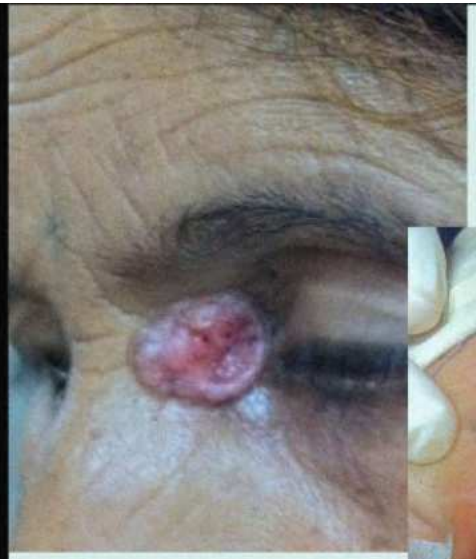
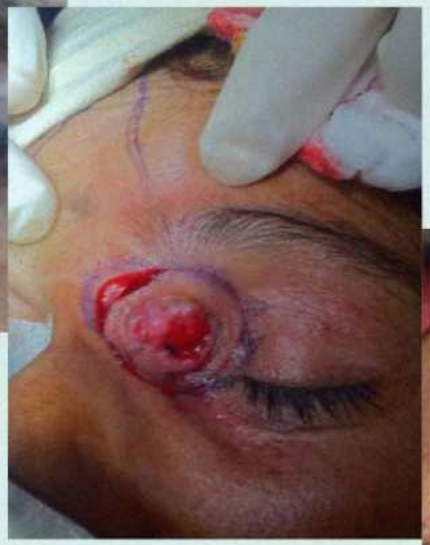
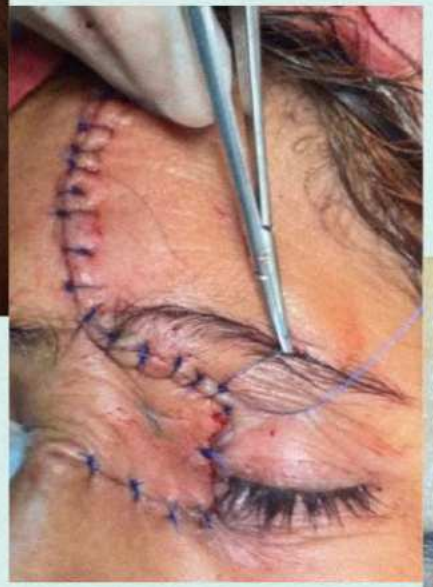
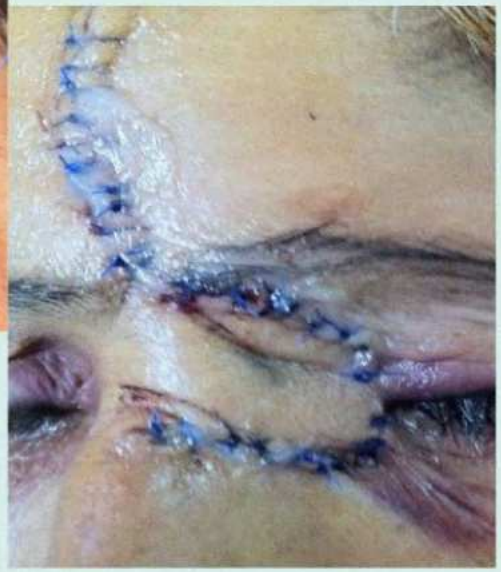


Tenzel flap for moderate



Mustarde cheek rotation flap for large defect

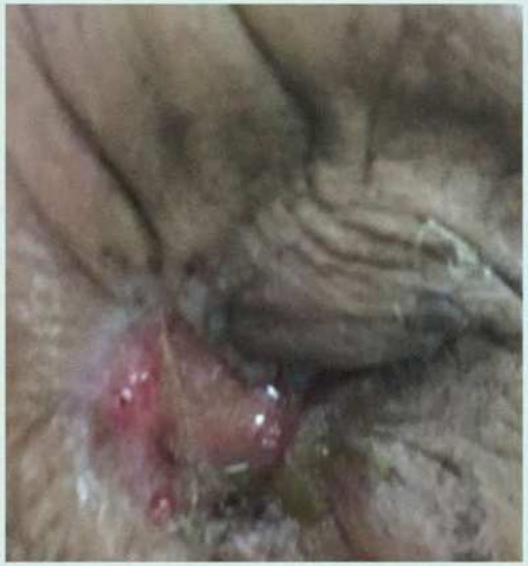

Step-1: direct closure
Step-2: central fixation
Step-3: conjunctival advancement
Step-4: excision of redundant skin
Step-2
Step-3
Step-1
Step-4

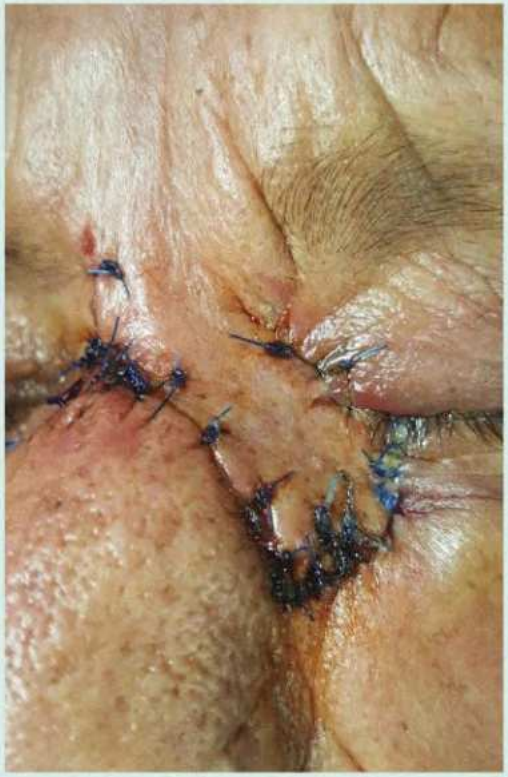





BCC and Mustarde cheek rotation flap

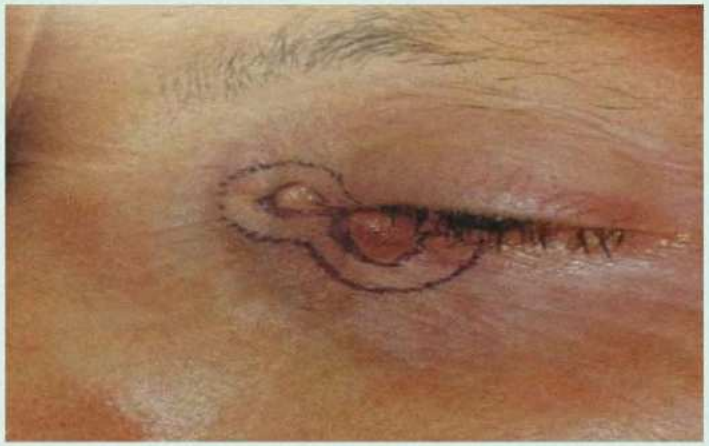


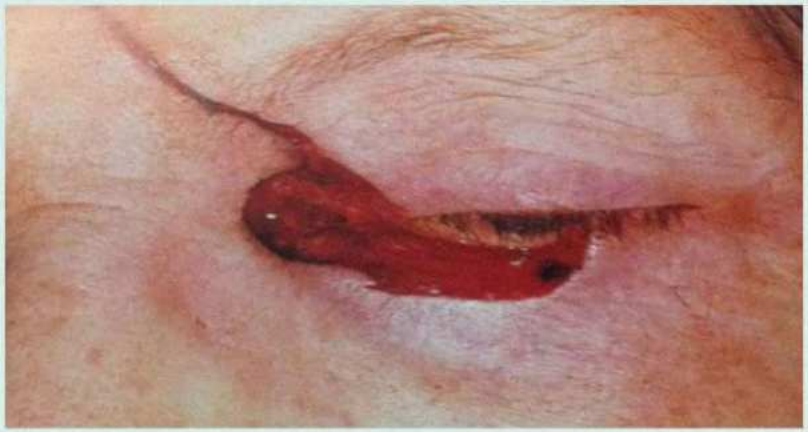

MEDIAL CANTHUS BCC AND RHOMBOID FLAP

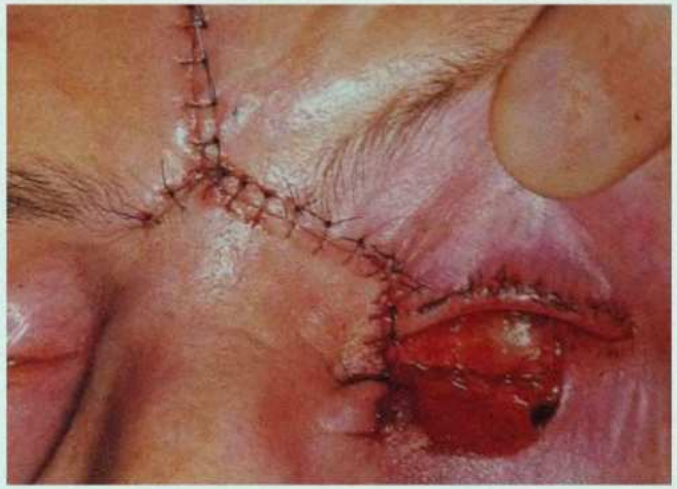


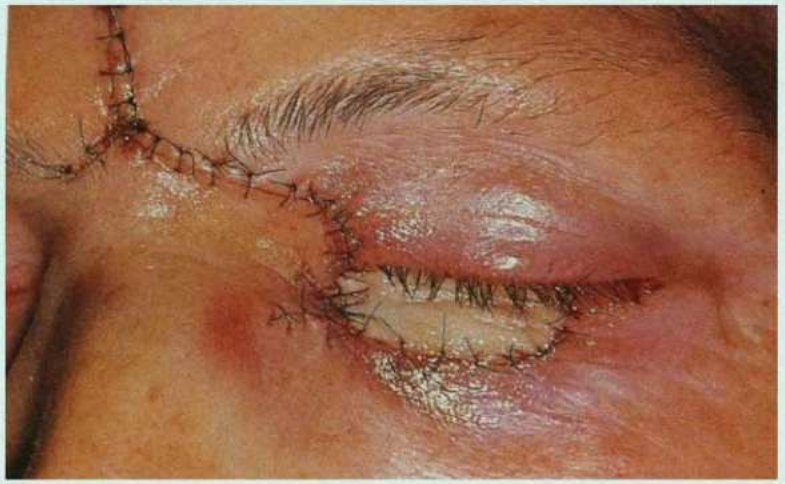


Glabellar flap and Hughes tarsconjunctival flap combined

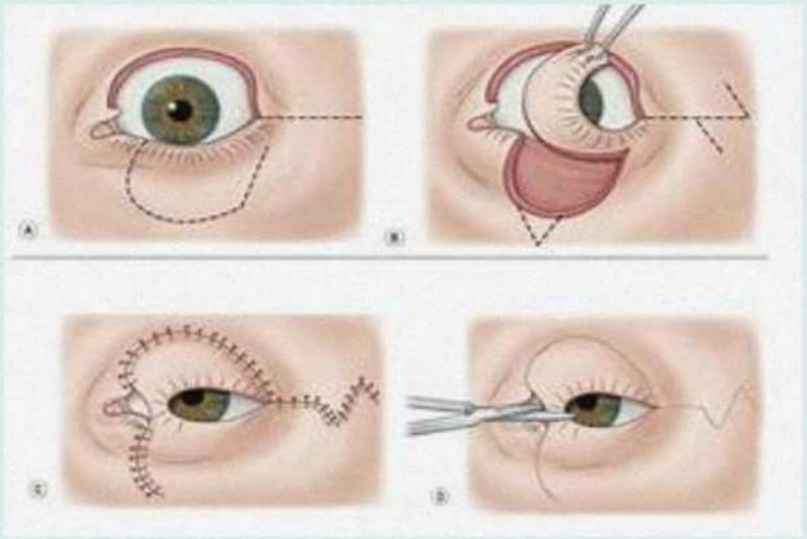


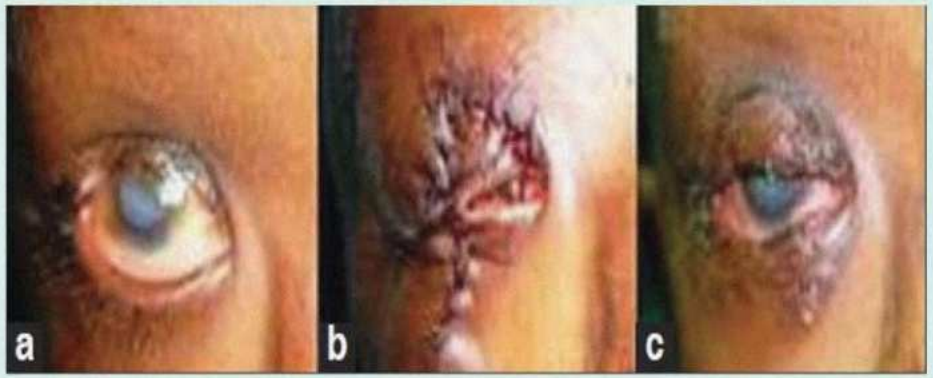




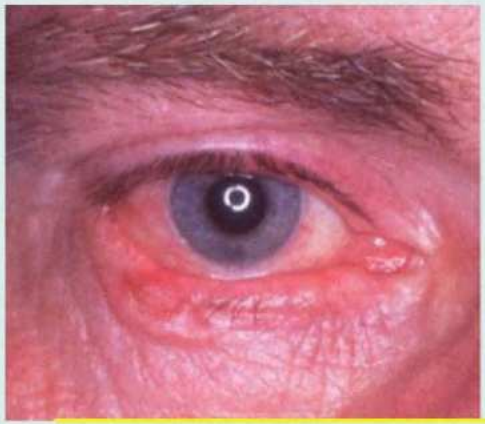


LID SWITCH FLAP



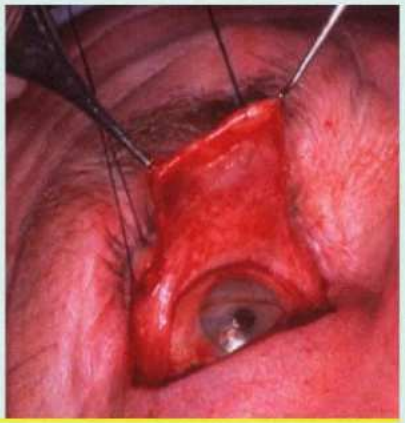


Eyelid-sharing procedure

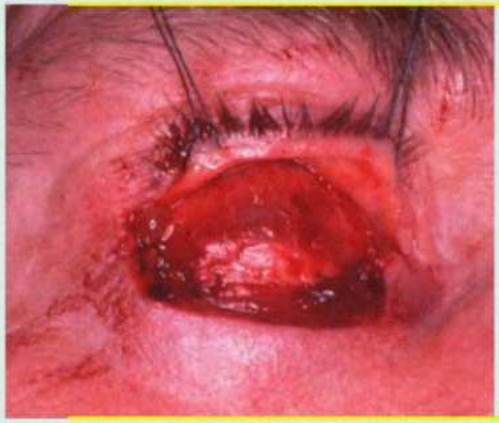


Extensive sclerosing BCC

Total excision of lower lid

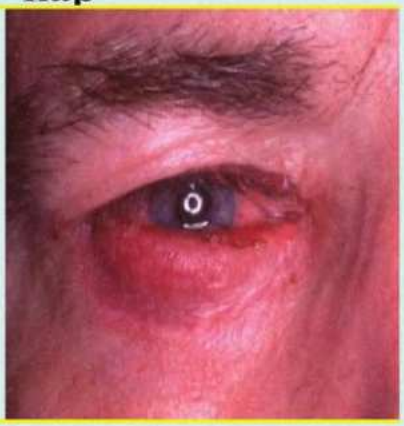


Tarsoconjunctival flap



Reconstruction of posterior lamella

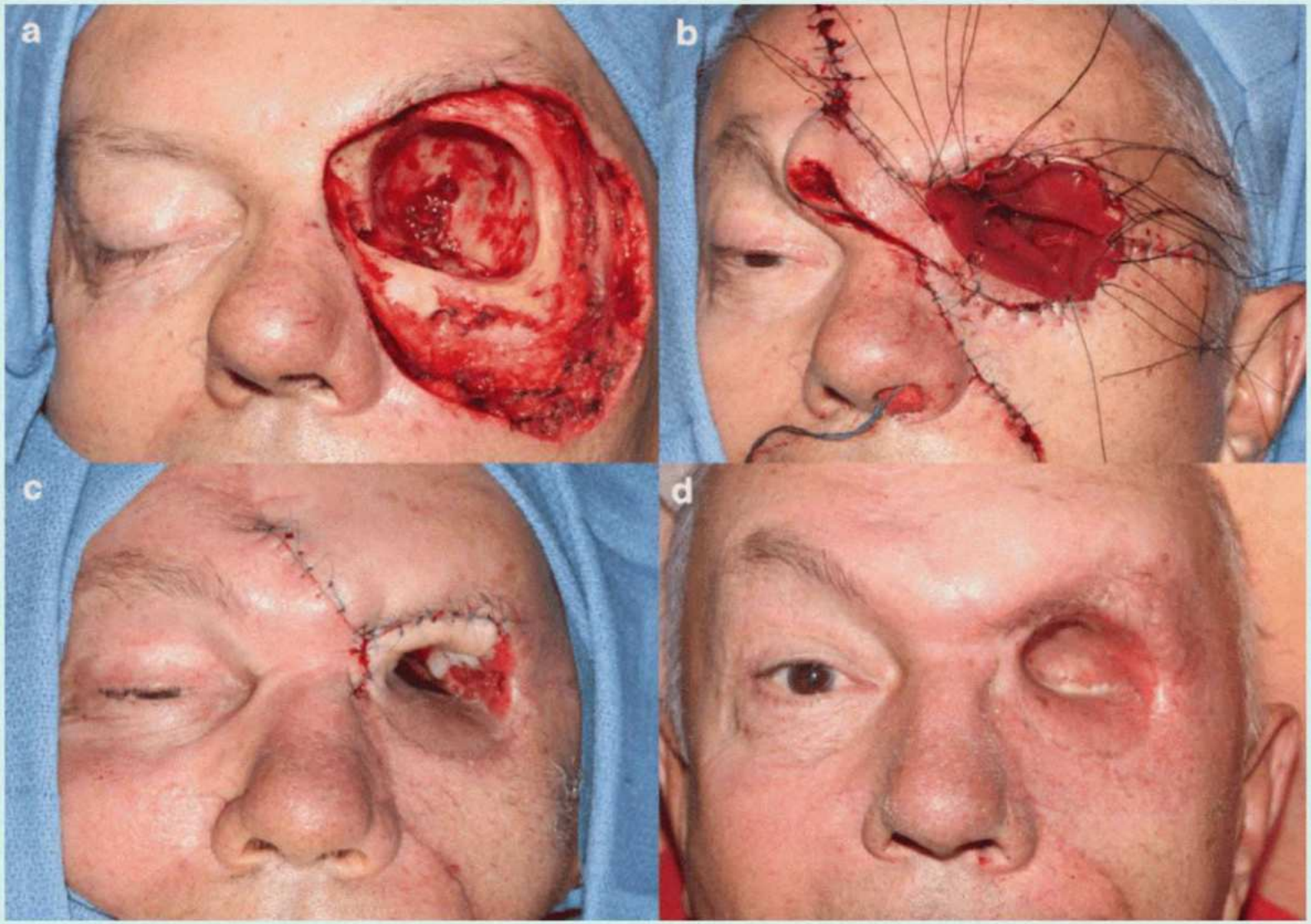
Reconstruction of anterior lamella with skin graft



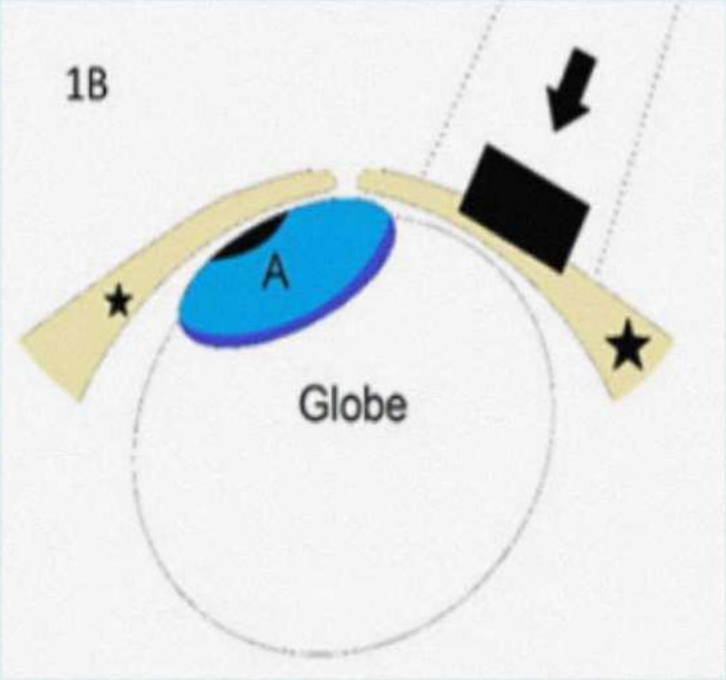
Appearance after healing

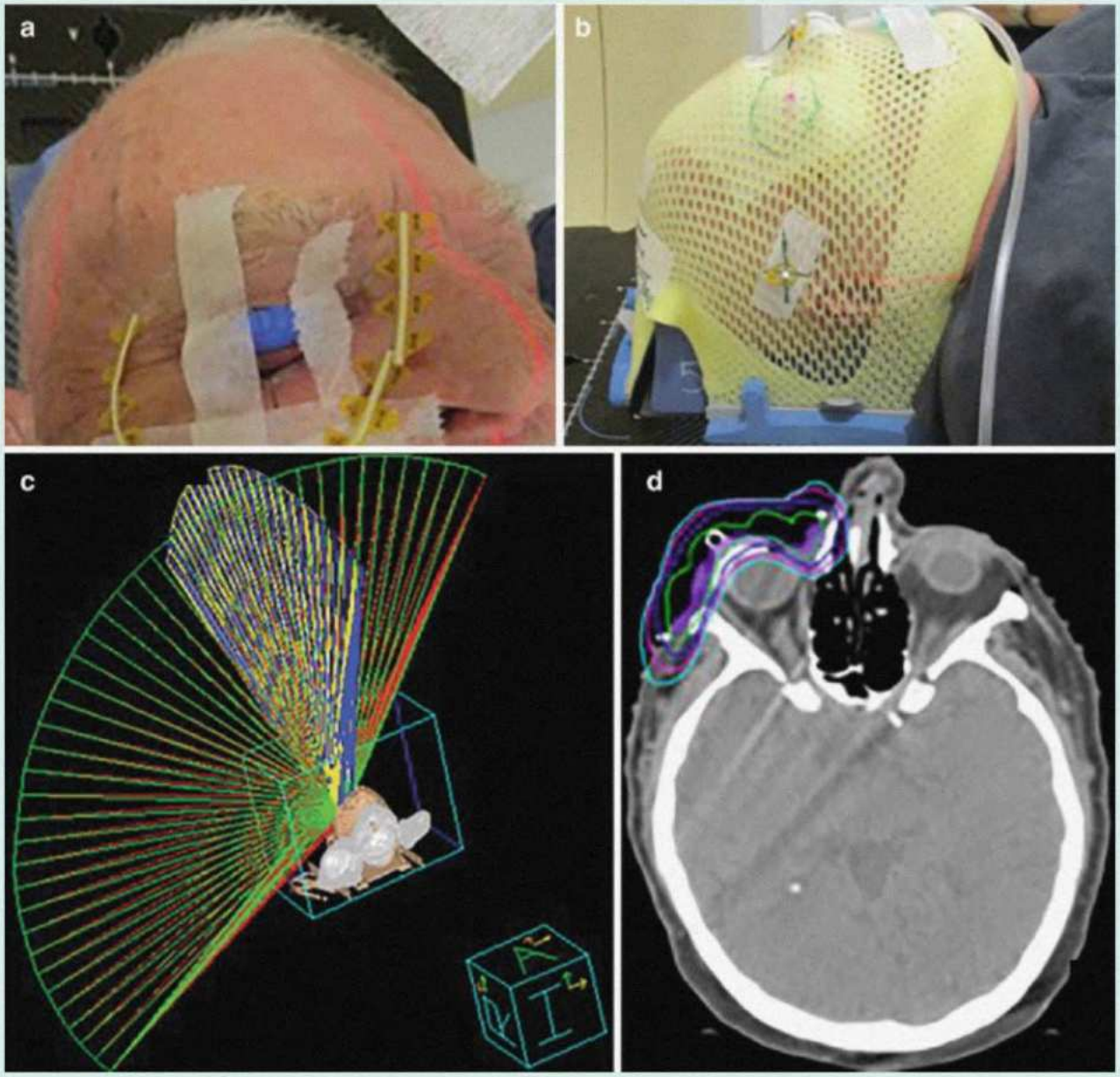
Exenteration

- a surgical procedure involving removal of the entire globe and its surrounding structures including muscles, fat, nerve
- Total and subtotal


a
b
c
d

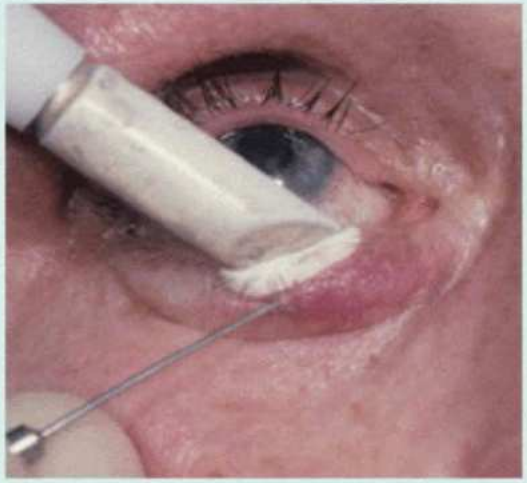
Radiotherapy for eyelid lesions

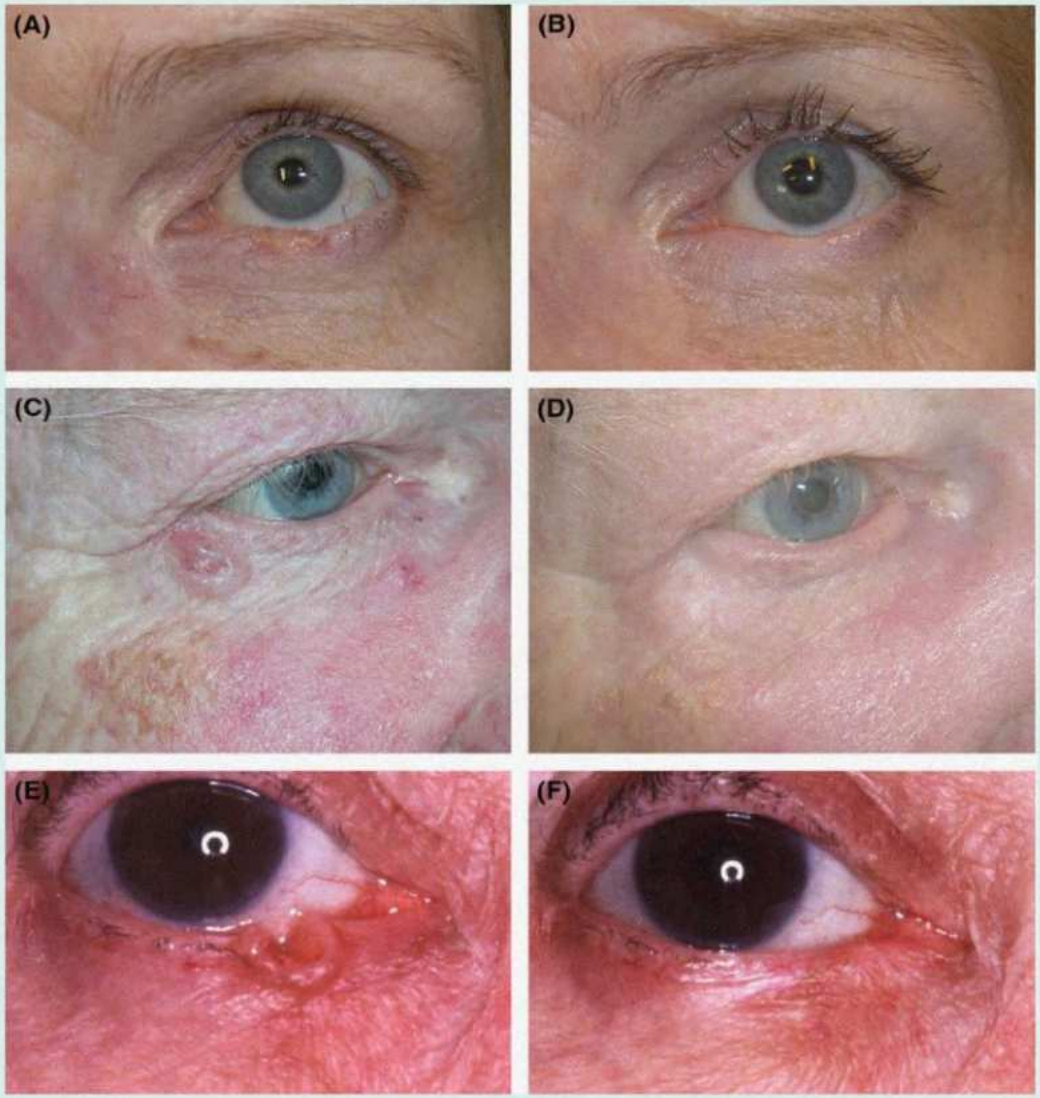



a
b
c
d

Cryotherapy

- **uses a special probe to apply very low temperatures to the eyelids**





Poor prognostic factors

Treatment history

- Recurrent tumor
- Incomplete excision
- Previous non surgical treatment

Tumor site size and location

- Size more than 2cm
- Medial canthus location
- Poorly defined margins
- Located in H zone of the face

Histological factors

- Infilterative morphea and micronodular type
- Perineural invasion

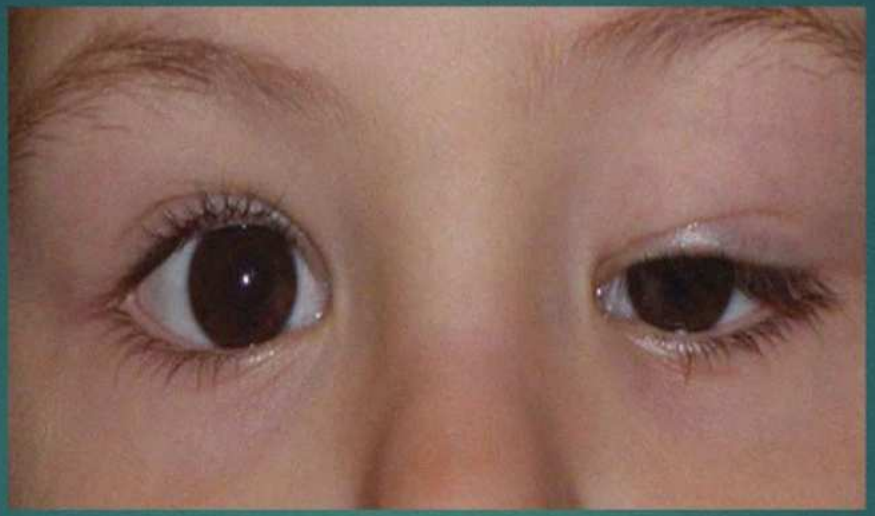
Patient factors

- immunosuppression

Dr Sofia Iqbal

Professor of Ophthalmology

KGMC/Hayatabad Medical Complex



"Blepharoptosis is a more accurate term than ptosis alone"

Presentation layout

By the end of this presentation you should be able to know about the following aspects of PTOSIS

- Causes
- Types
- Evaluation
- Management

Presentation layout

By the end of this presentation you should be able to know about the following aspects of PTOSIS

- Causes
- Types
- Evaluation
- Management

What is Ptosis?

- The term is from Greek πτῶσις – "a fall, falling
- Normally upper lid covers about upper one sixth of the cornea i.e. about 2mm
- In ptosis it covers more than 2mm

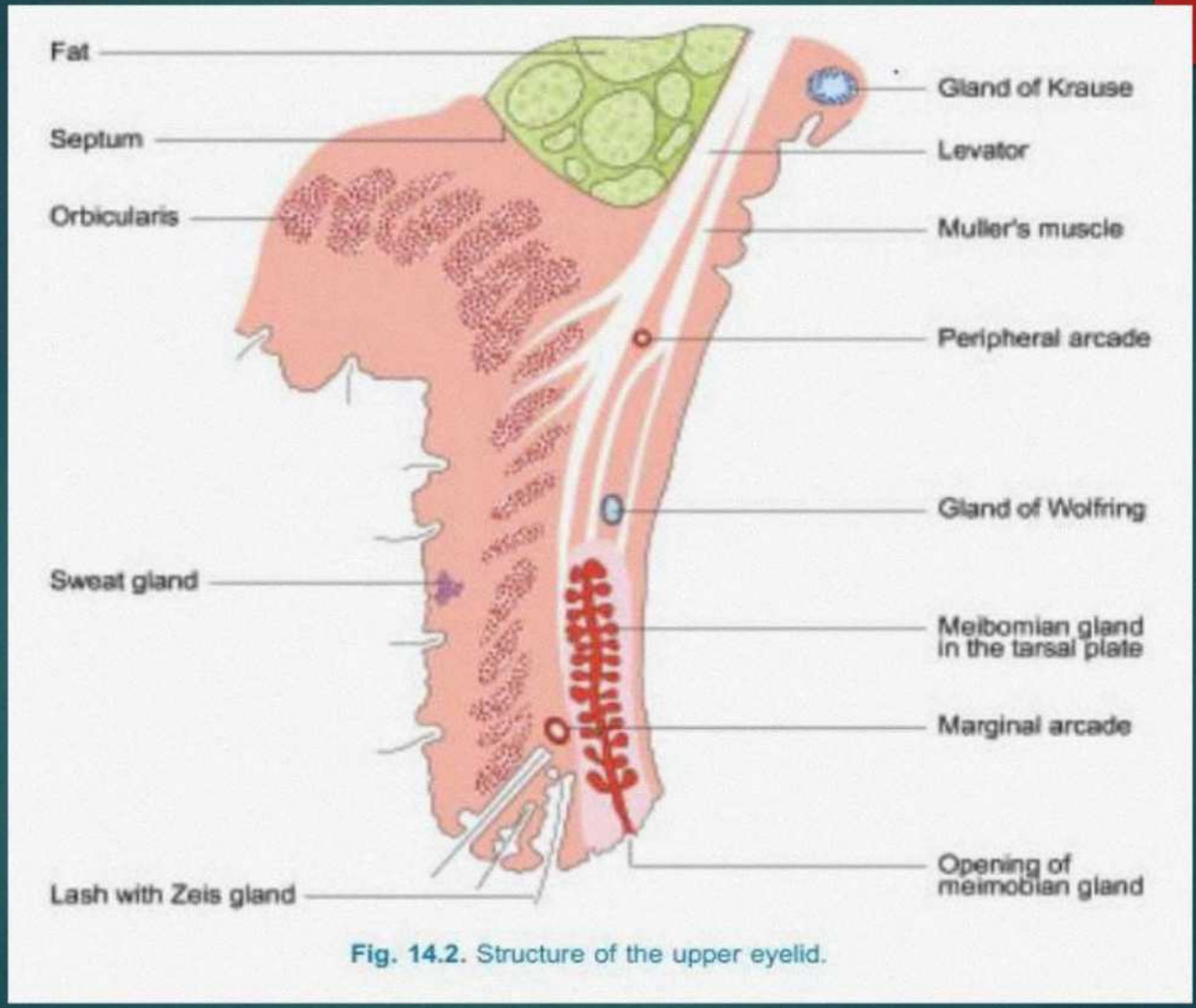
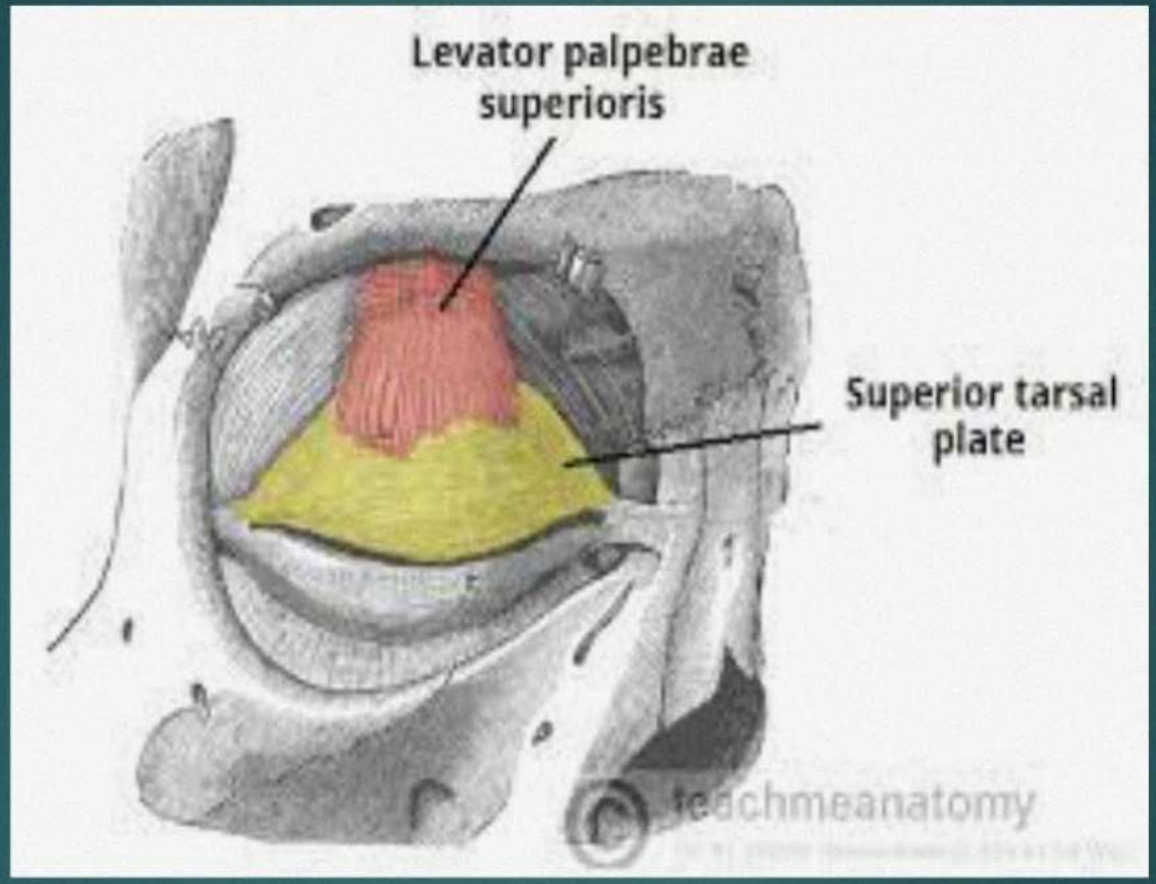


Fig. 14.2. Structure of the upper eyelid.


Levator palpebrae superioris
Superior tarsal plate
teachmeanatomy

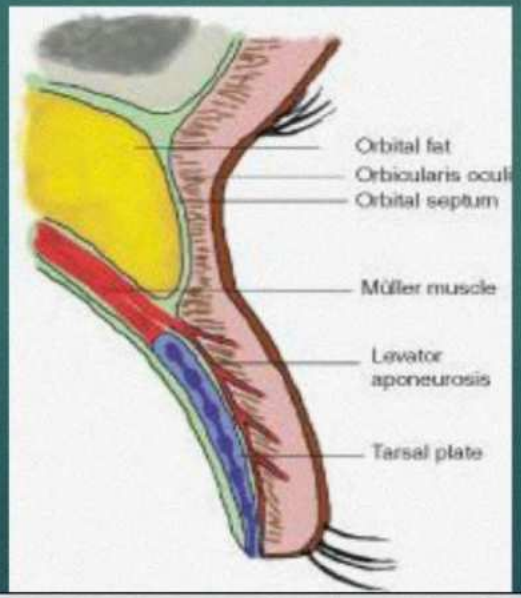
LPS

- Primary muscle responsible for lid elevation
- It arises from the back of the orbit and extends forwards over the cone of eye muscles
- It inserts into the eyelid and the tarsal plate, a fibrous semicircular structure which gives the upper eyelid its shape
- Supplied by the superior division of 3rd CN

Muller's muscles:

- The way that the LPS attaches to the tarsal plate is modified by the underlying Müller's muscle

- Involuntary muscle, with sympathetic innervation, has the capacity to 'tighten' the attachment and so raise the lid a few millimeters

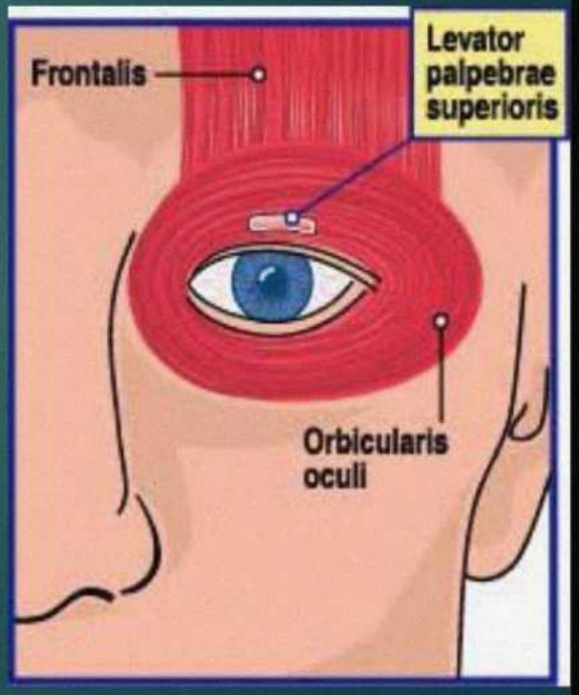


• Frontalis & Orbicularis Oculi

- Supplied by facial nerve

- Frontalis contraction helps to elevate the lid by acting indirectly on the surrounding soft tissues

- OO contraction depresses the eyelid



CLASSIFICATION OF PTOSIS

A. **Congenital**

B. **Acquired**
   1. Neurogenic
   2. Myogenic
   3. Aponeurotic
   4. Mechanical
   5. Neurotoxic

C. **Pseudo Ptosis**

Congenital Ptosis

1-Simple congenital Ptosis

2- Congenital ptosis with associated weakness of superior rectus muscle

3- Blepharophimosis syndrome

4- Congenital synkinetic Ptosis

(Marcus Gunn jaw winking ptosis)

Congenital Ptosis

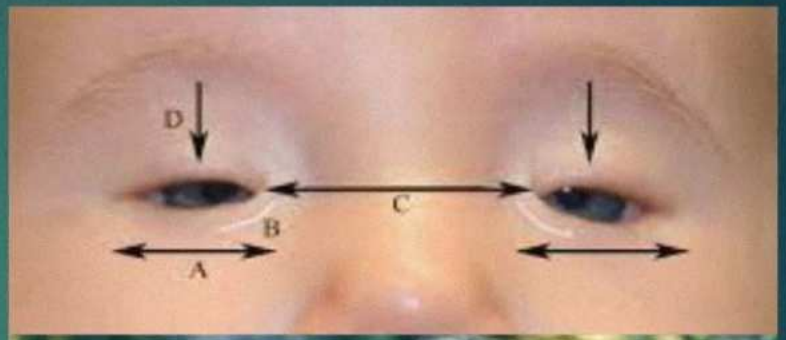
- Maldevelopment of the levator palpebrae superioris (LPS)
- May be associated with SR weakness
- If visual axis is covered - risk of amblyopia
- Absent skin crease
- Upward position of ptotic lid in down gaze

Congenital Ptosis

What do u see here?

Blepharophimosis syndrome

- Moderate to severe symmetrical ptosis
- Short horizontal palpebral aperture
- **Telecanthus** (lateral displacement of medial canthus)
- **Epicanthus inversus** (lower lid fold larger than upper)
- Poorly developed nasal bridge and hypoplasia of superior orbital rims



Type 1 : associated with primary ovarian failure
Type 2: no systemic association

- Mutation in FOXL2 gene
- Controls the production of the FOXL2 protein
- (involved in the development of the muscles in the eyelids as well as the growth and development of ovarian cells)
- Females should be referred to an endocrinologist or gynecologist to assess for primary ovarian insufficiency

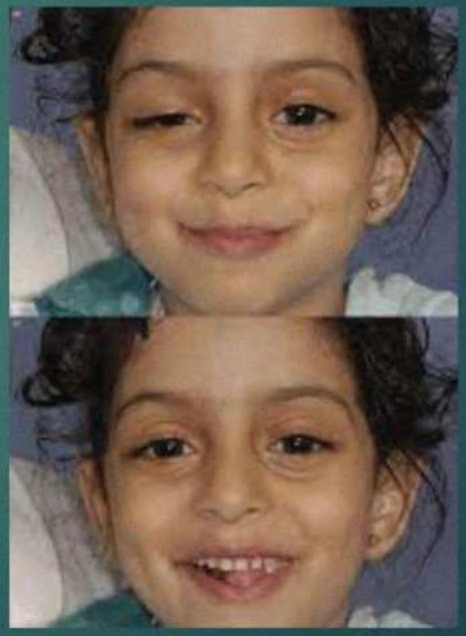
Congenital Synkinetic ptosis

IN MARCUS GUNN PHENOMENON

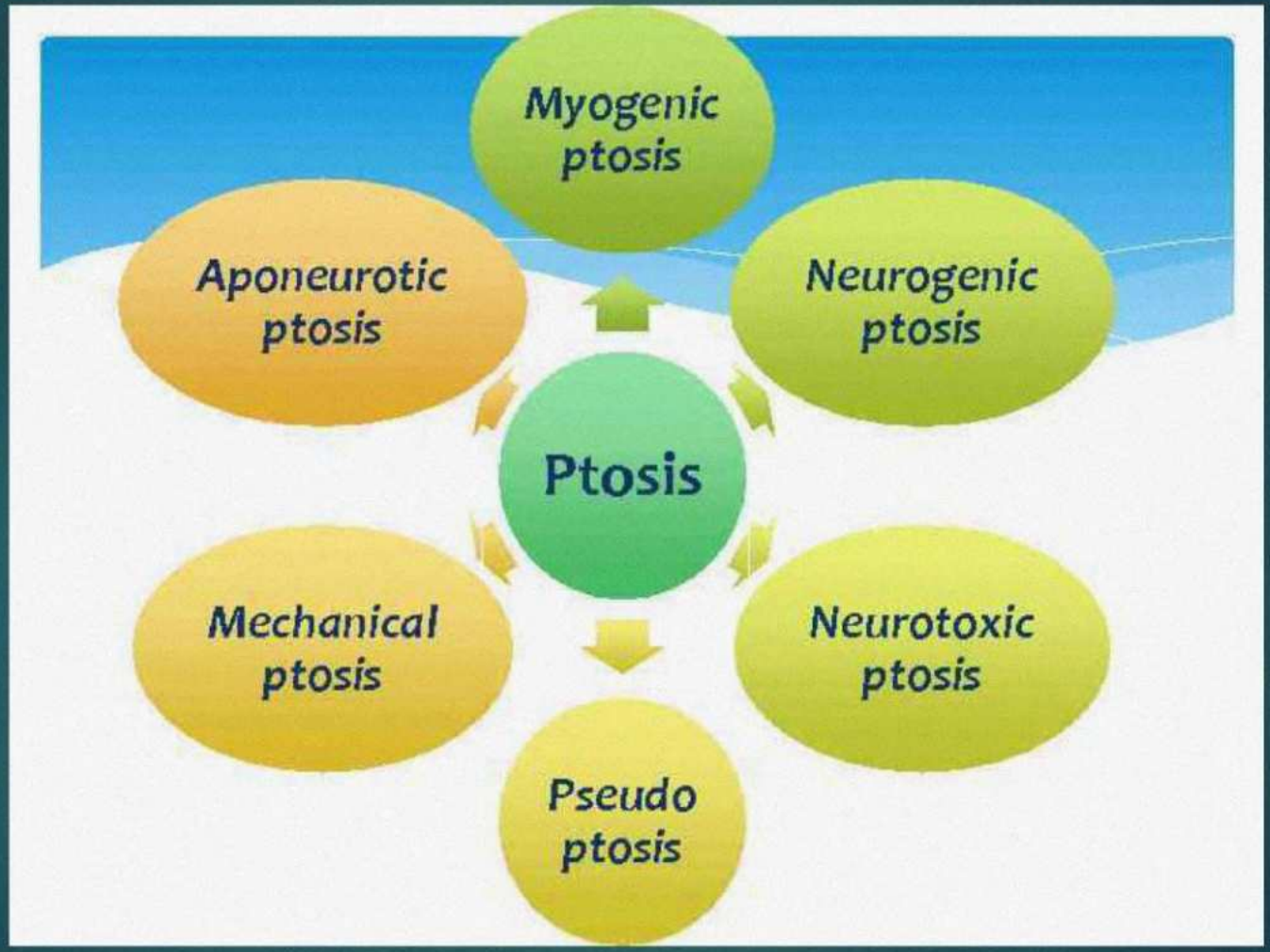
- The stimulation of the trigeminal nerve by the contraction of the pterygoid muscles results in the excitation of the branch of the oculomotor nerve that innervates the LPS ipsilaterally, so the patient will have rhythmic upward jerking of their upper eyelid

- Accounts for about 5% of all cases of congenital Ptosis

Marcus Gunn jaw-winking syndrome



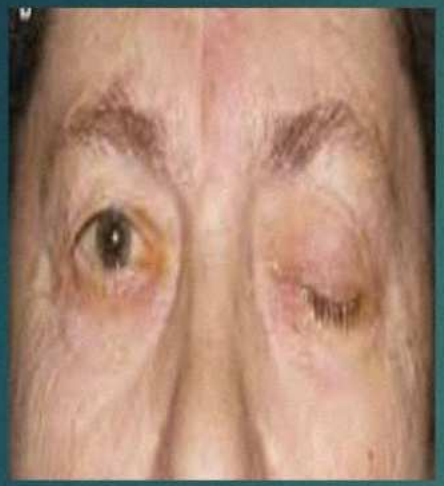
ACQUIRED PTOSIS

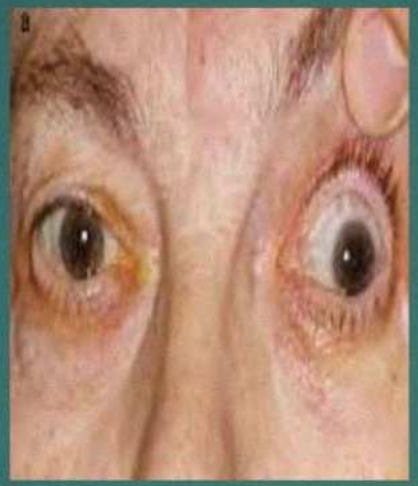


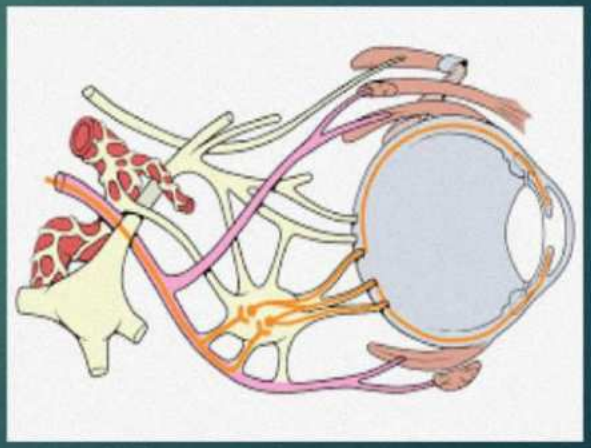
1-Neurogenic ptosis

- Third nerve palsy
- 3rd nerve misdirection
- Horner's syndrome
- Ophthalmoplegic migraine
- Cerebral ptosis
- Multiple sclerosis

Third nerve palsy

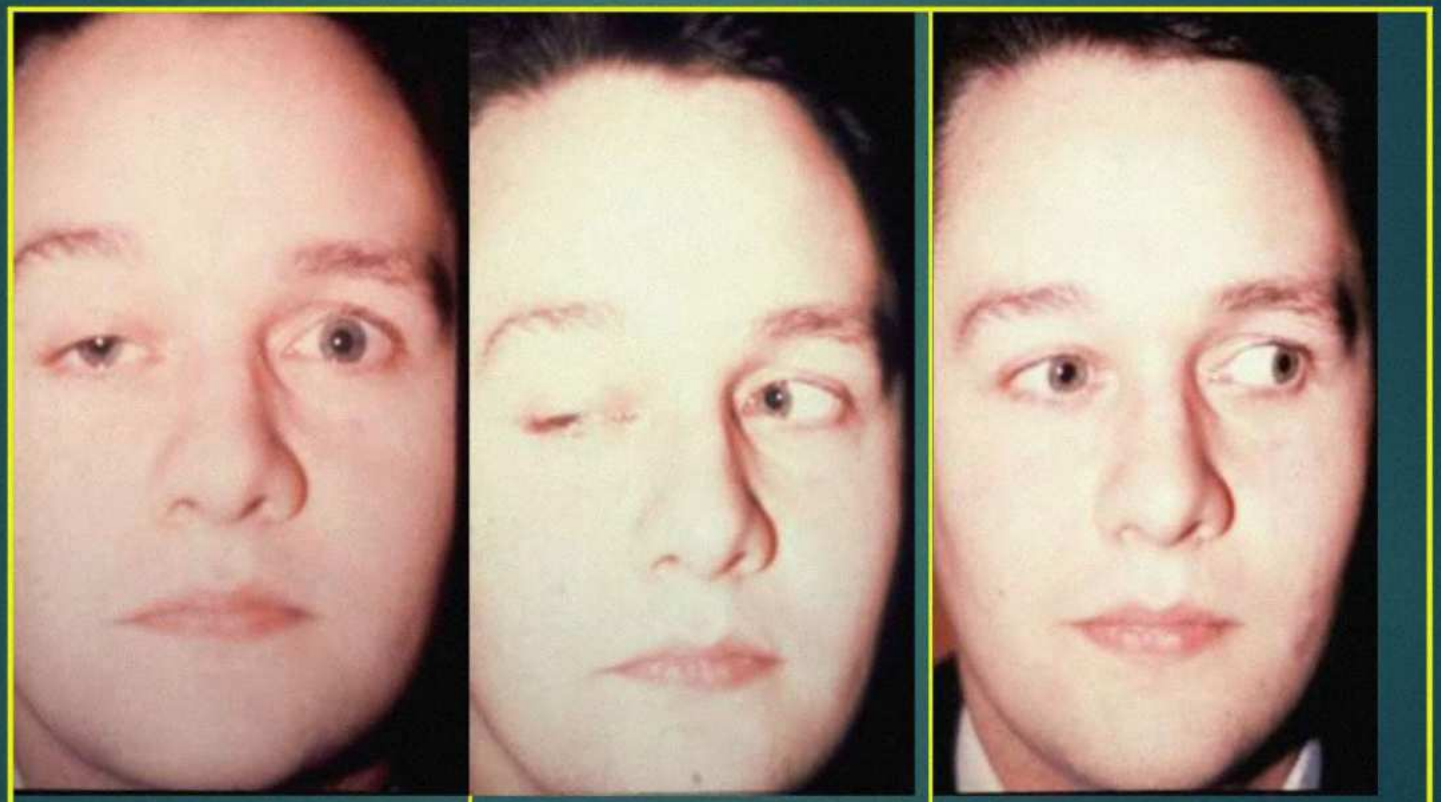






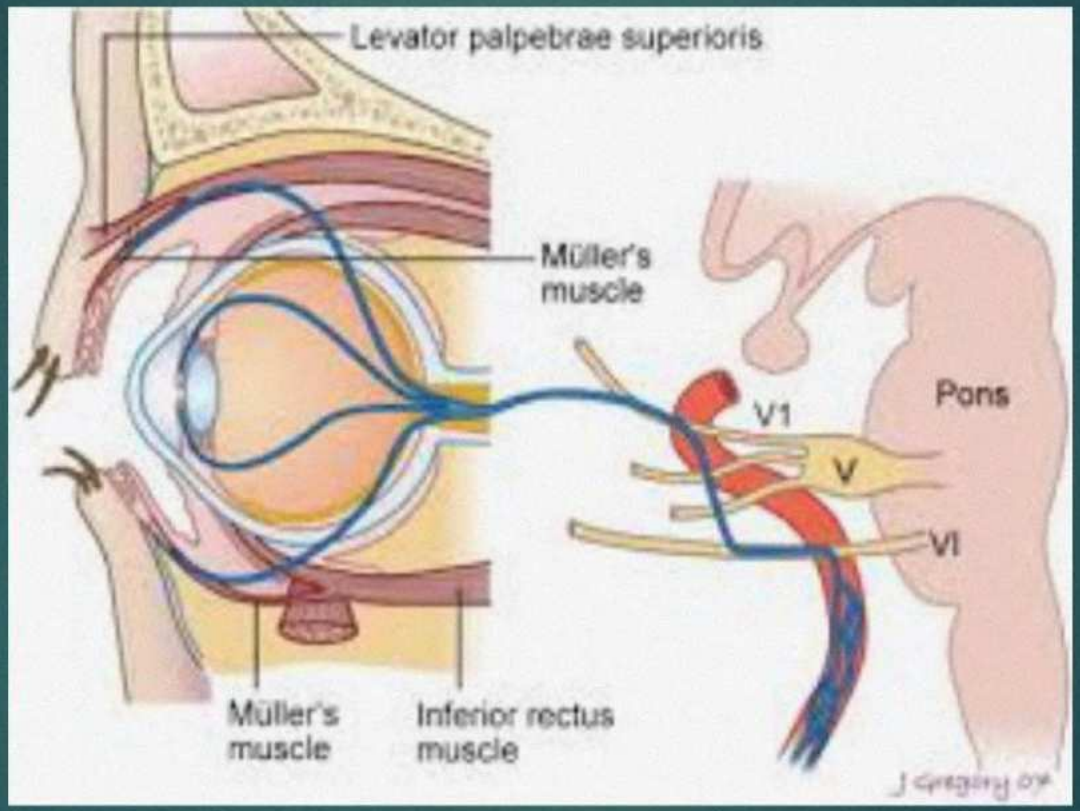
Right third nerve misdirection

- Aberrant regeneration following acquired third nerve palsy
- Pupil is occasionally involved
- Bizarre movements of upper lid accompany eye movements



Right ptosis in primary position	Worse on right gaze	Normal on left gaze

HORNER'S SYNDROME



HORNER'S SYNDROME

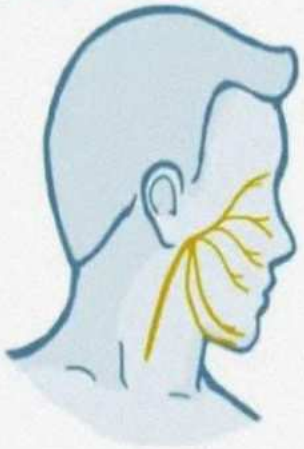
JOHANN FRIEDRICH HORNER

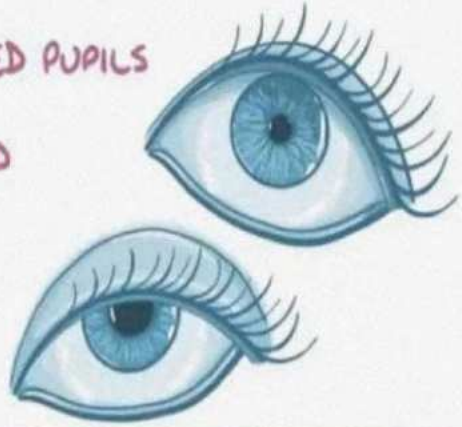
~PROBLEM WITH SYMPATHETIC NERVE SUPPLY TO ONE SIDE of FACE

↓

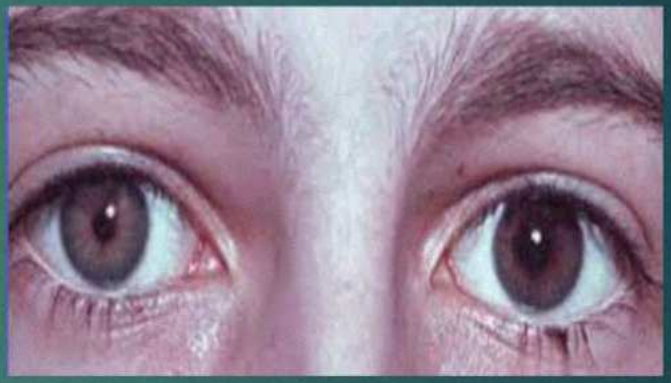
RESULTS IN:

- MIOSIS ~ CONSTRICTED PUPILS
- PTOSIS ~ DROOPY EYELID
- ANHIDROSIS
  - FAILURE TO SWEAT

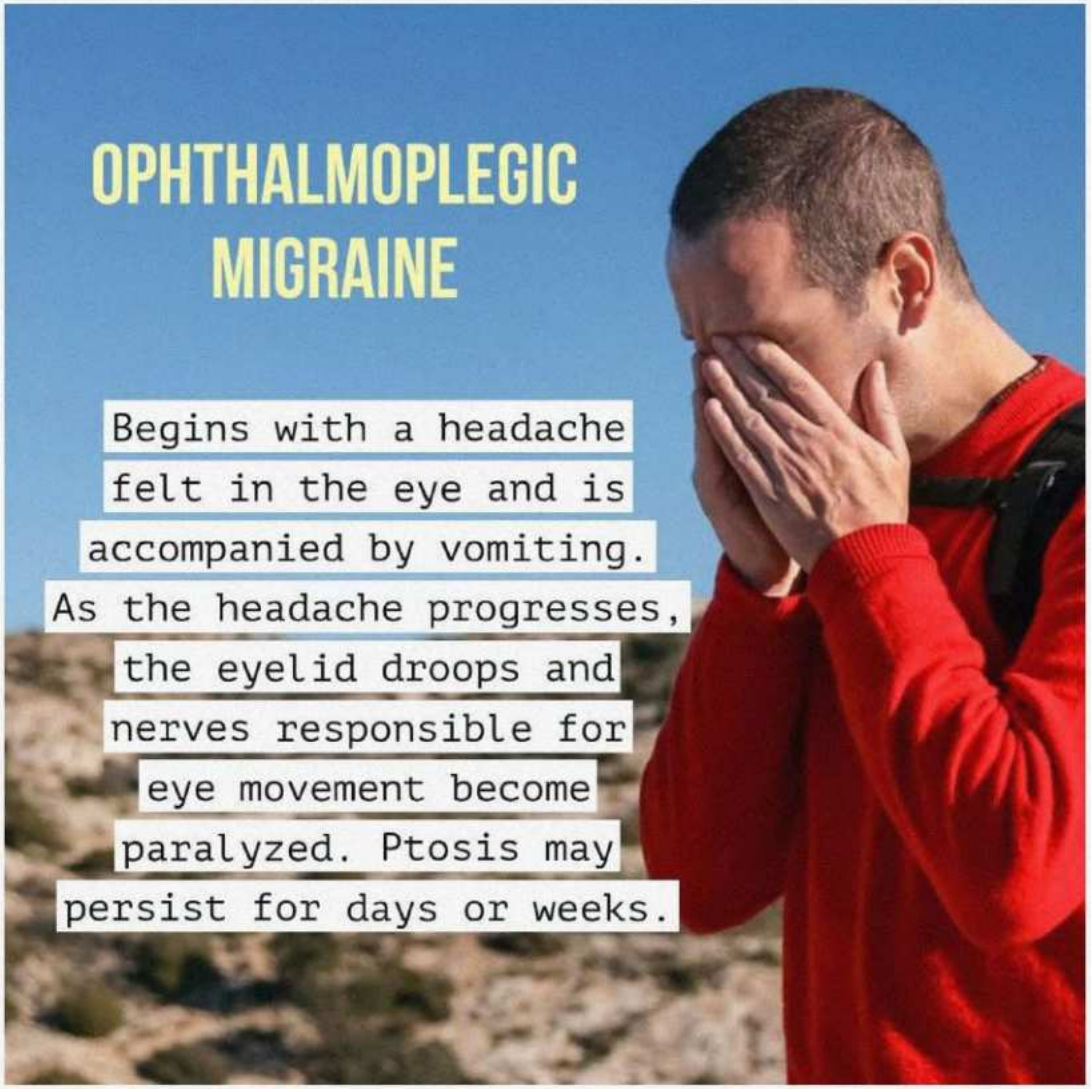




Horner's syndrome



OPHTHALMOPLEGIC MIGRAINE



Begins with a headache felt in the eye and is accompanied by vomiting. As the headache progresses, the eyelid droops and nerves responsible for eye movement become paralyzed. Ptosis may persist for days or weeks.

Cerebral ptosis

- Due to supranuclear lesions
- Unilateral cerebral ptosis occurs with lesions, usually ischemic, of the opposite hemisphere, and is more common with right hemisphere lesions
- Bilateral supranuclear ptosis may occur with unilateral or bilateral hemispheric lesions
- Ptosis has been reported in as many as 37.5% of patients with hemispheric strokes

2-MYOGENIC PTOSIS

- It is due to acquired disorders of the LPS muscle or of the myoneural junction

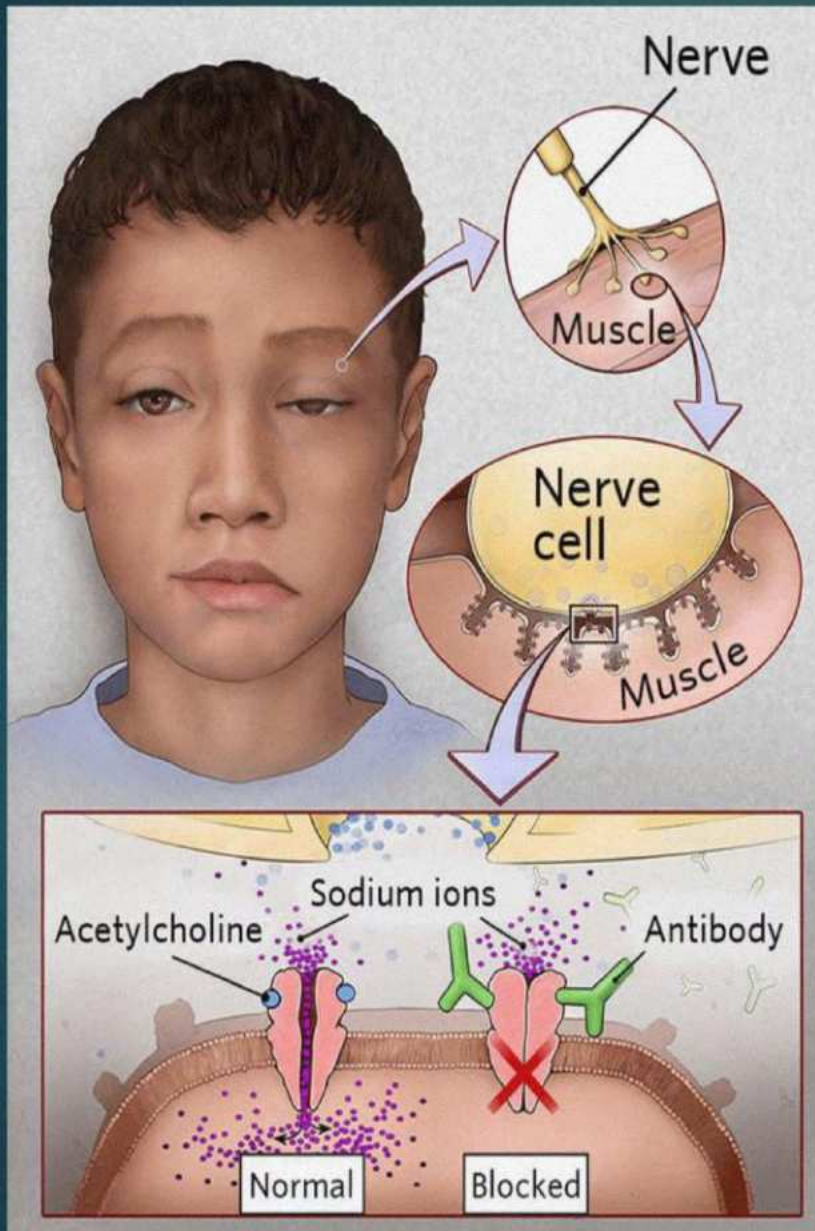
- **Myasthenia gravis**
- **Myotonic dystrophy**
- **Ocular myopathies**
- **Oculo-pharyngeal muscular dystrophy**
- **Following trauma to the LPS muscle**

Myasthenia Gravis

Disease of Neuromuscular Junction

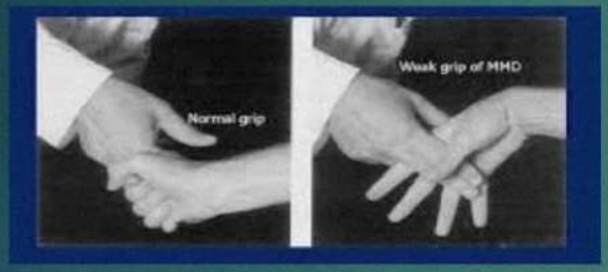
Features

(1) Drooping of eyelids
(2) Weakness in arms legs
(3) Change of Voice
(4) Swalllowing Difficulty



Myotonic dystrophy

Release of grip difficult

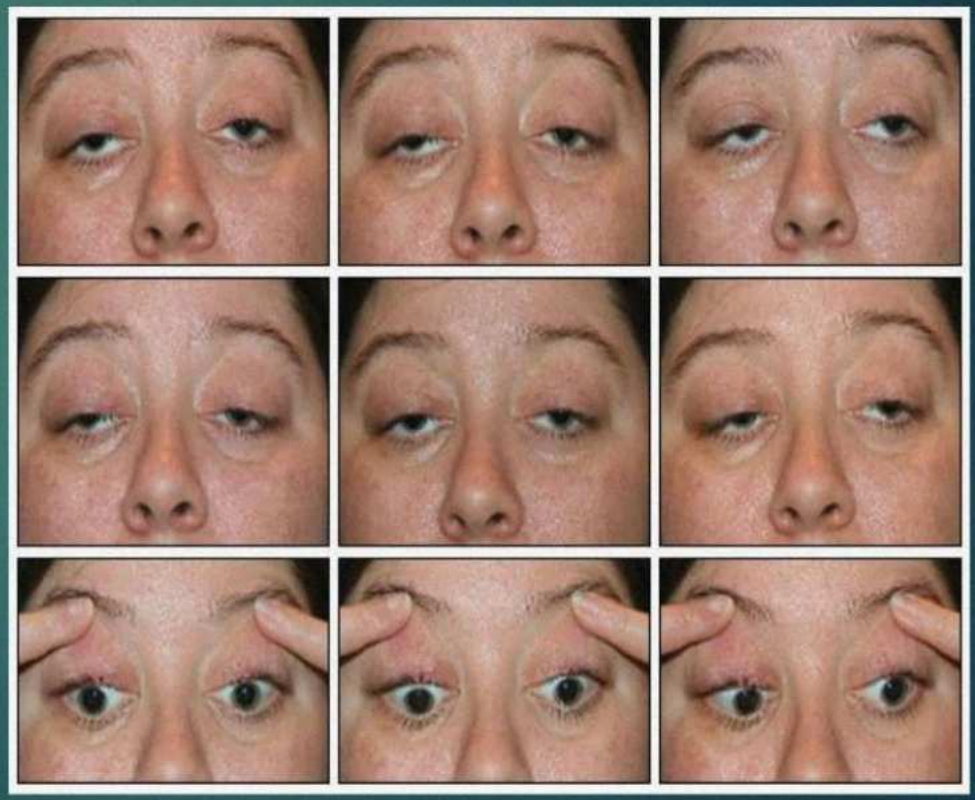


- Muscle wasting
- Involvement of tongue and pharyngeal muscles
- Ophthalmoplegia – uncommon
- Hypogonadism
- Frontal baldness in males
- Intellectual deterioration
- Presenile stellate cataracts

Ocular myopathies

Clinical types

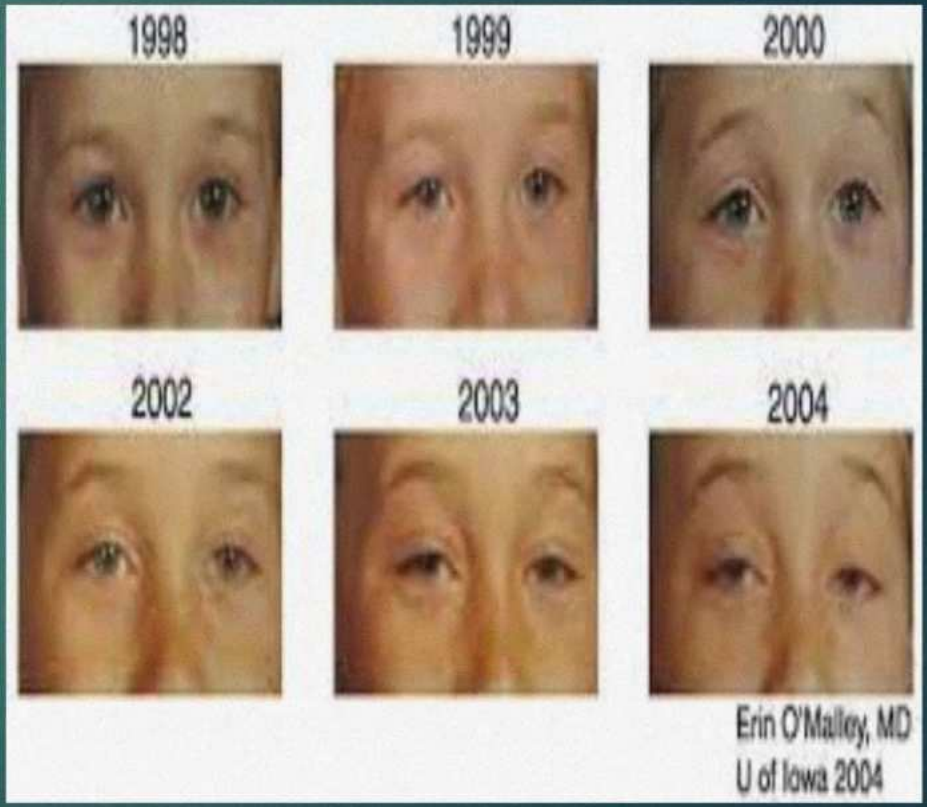
- Isolated
- Oculopharyngeal dystrophy
- Kearns-Sayre syndrome (pigmentary retinopathy)



Ocular features

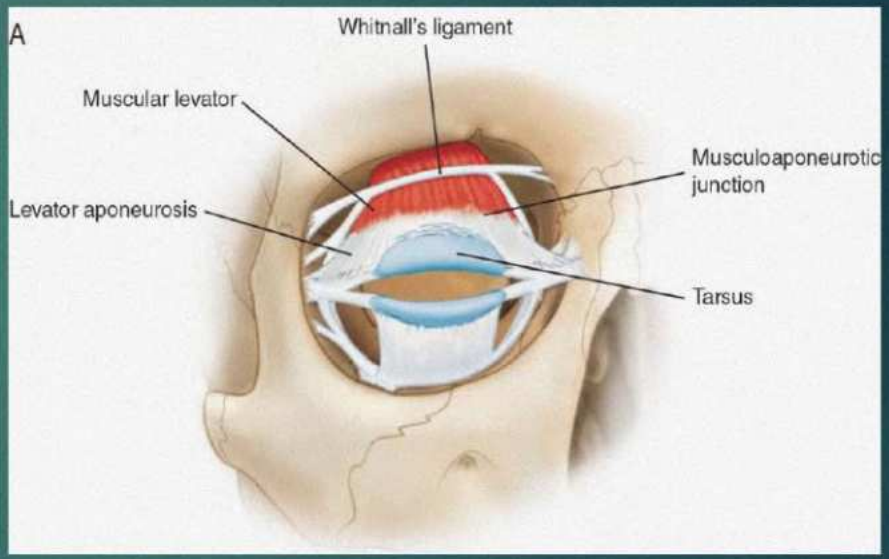
Ptosis – slow, progressive and symmetrical

Ophthalmoplegia slow, progressive and symmetrical no diplopia

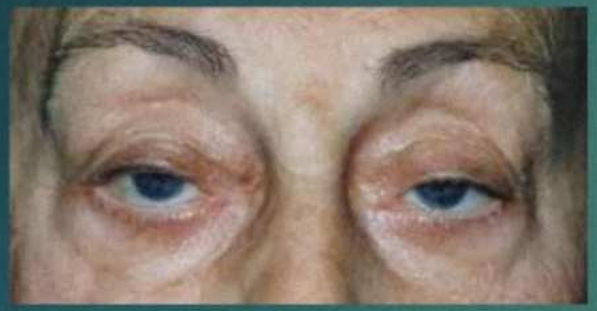


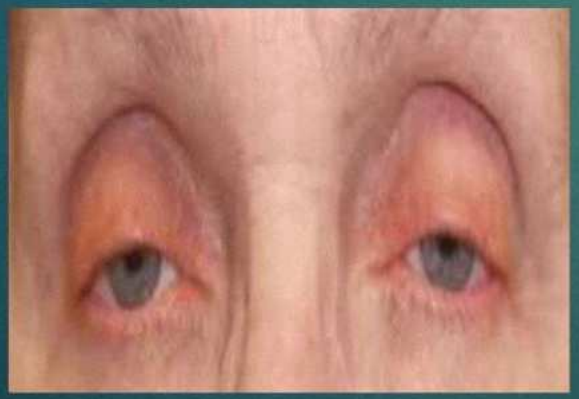
3. APONEUROTIC PTOSIS

- It develops due to defects of the levator aponeurosis in the presence of a normal functioning muscle
- Involutional (senile) ptosis
- Post operative ptosis
- Posttraumatic dehiscence or disinsertion of the aponeurosis



Involutional ptosis

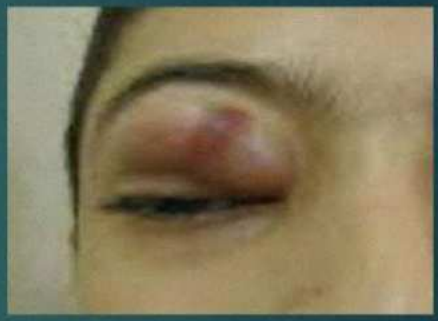


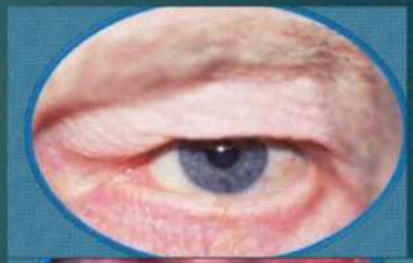


- High upper lid crease
- Good levator function
- Absent upper lid crease
- Deep sulcus

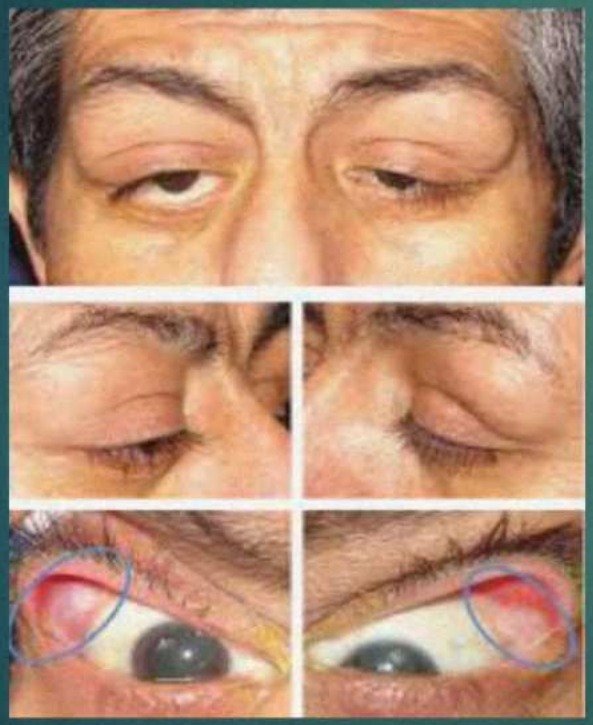
4. Mechanical ptosis

- **Due to excessive weight** on the upper lid
  - lid tumors
  - Chalazion
  - lid edema
- **Cicatricial Ptosis**
  - Ocular pemphigoid
  - Trachoma

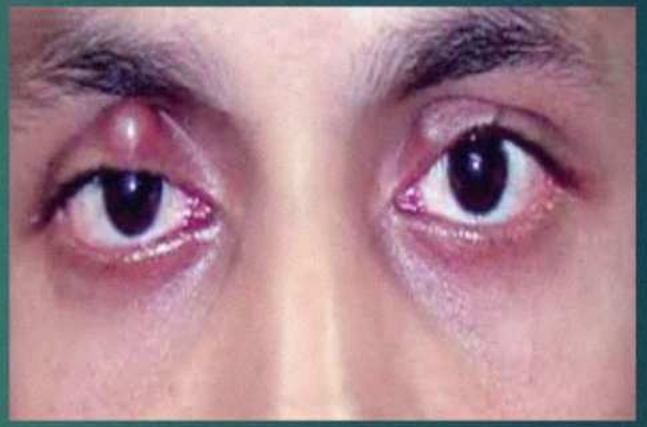




- Orbital tumors



- Chalazion



5. NEUROTOXIC PTOSIS

- Envenomation by elapids such as cobras, or kraits
- Bilateral ptosis is usually accompanied by diplopia, dysphagia and/or progressive muscular paralysis
- Neurotoxic ptosis is a precursor to respiratory failure and eventual suffocation caused by complete paralysis of the thoracic diaphragm
- Medical emergency

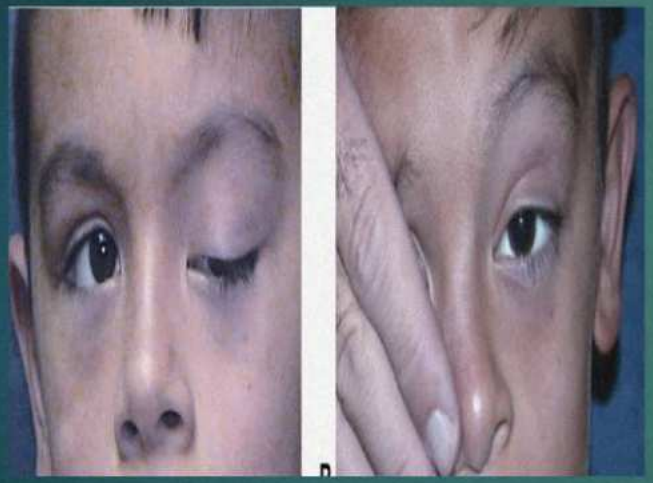
PSEUDOPTOSIS

- Pseudoptosis is the appearance of ptosis in the absence of LPS abnormality
- Exclude pseudoptosis (simulated ptosis) on inspection

- **Microphthalmia**
- **Anophthalmia**
- **Phthisis bulbi**
- **Blepharochlalasis**
- **Contralateral proptosis**
- **Hypotropia**

Pseudo ptosis

HYPOTROPIA

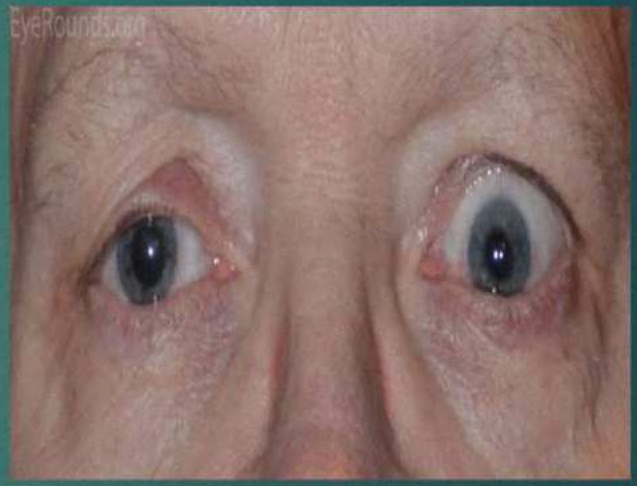


DERMATOCHALASIS

Pseudo ptosis

Anophthalmia

Contralateral lid retraction



EVALUATION OF PTOSIS

- Detailed History
- Ocular examination
- GPE
- Ptosis measurements
- Investigations
- Treatment plan

HISTORY

Ptosis

- Age of onset
- Duration — *SUDDEN ONSET MEANS EMEREGENCY*
- Trauma
- Diurnal variability
- Previous surgery
- Poisoning
- Allergy
- Malignancy /treatment
- Any reaction with anesthesia
- Bleeding tendency

Association with

- Jaw movements
- Abnormal ocular movements
- Abnormal head posture
- Diplopia
- Dysphagia /dysarthria
- Muscle weakness/neurological features

- Previous photographs may prove to be of great help
- Is there a family history of ptosis or of other muscle weakness?

Examination:

- Inspection of the patient as whole

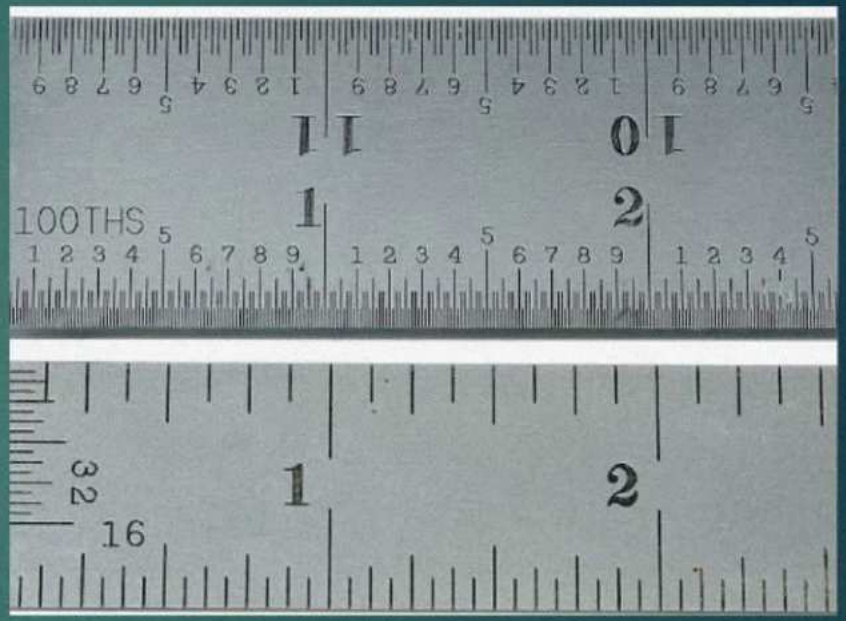
Face, chin position, head posture

The normal upper eyelid in primary position

- VA
- PUPILS
- EOM
- Squint assessment
- Dilated fundoscopy

Measurements

- Margin reflex distance
- Vertical fissure height
- LPS action
- Lid crease level

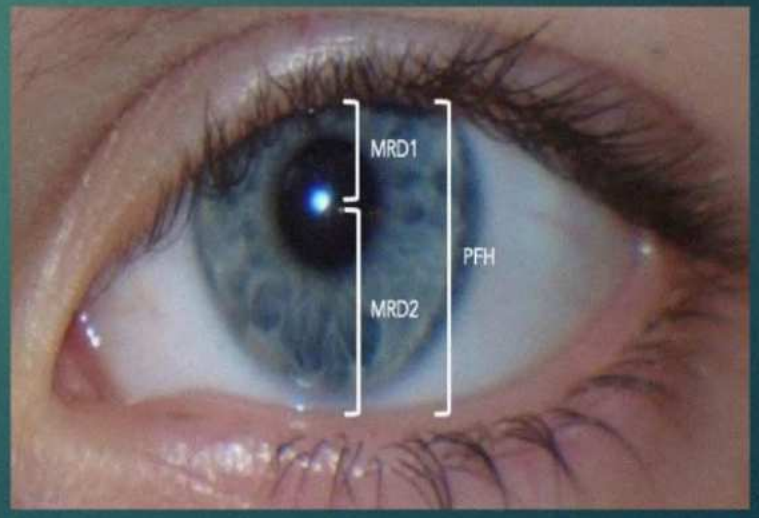


1. MARGIN REFLEX DISTANCE

- Margin-to-reflex distance 1 (MRD1)

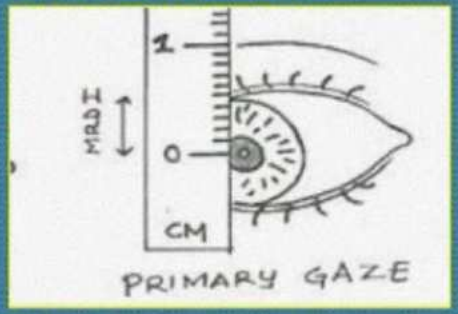
- When light is thrown on the cornea a reflection occurs the distance from the central pupillary light reflex to the upper eyelid margin with the eye in primary gaze

- NORMAL : 4 - 5 mm

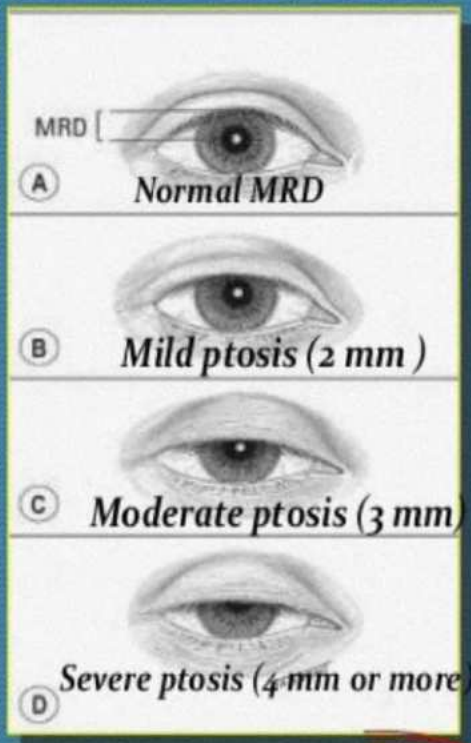


Measurements Of MRD

Margin-reflex distance (MRD).



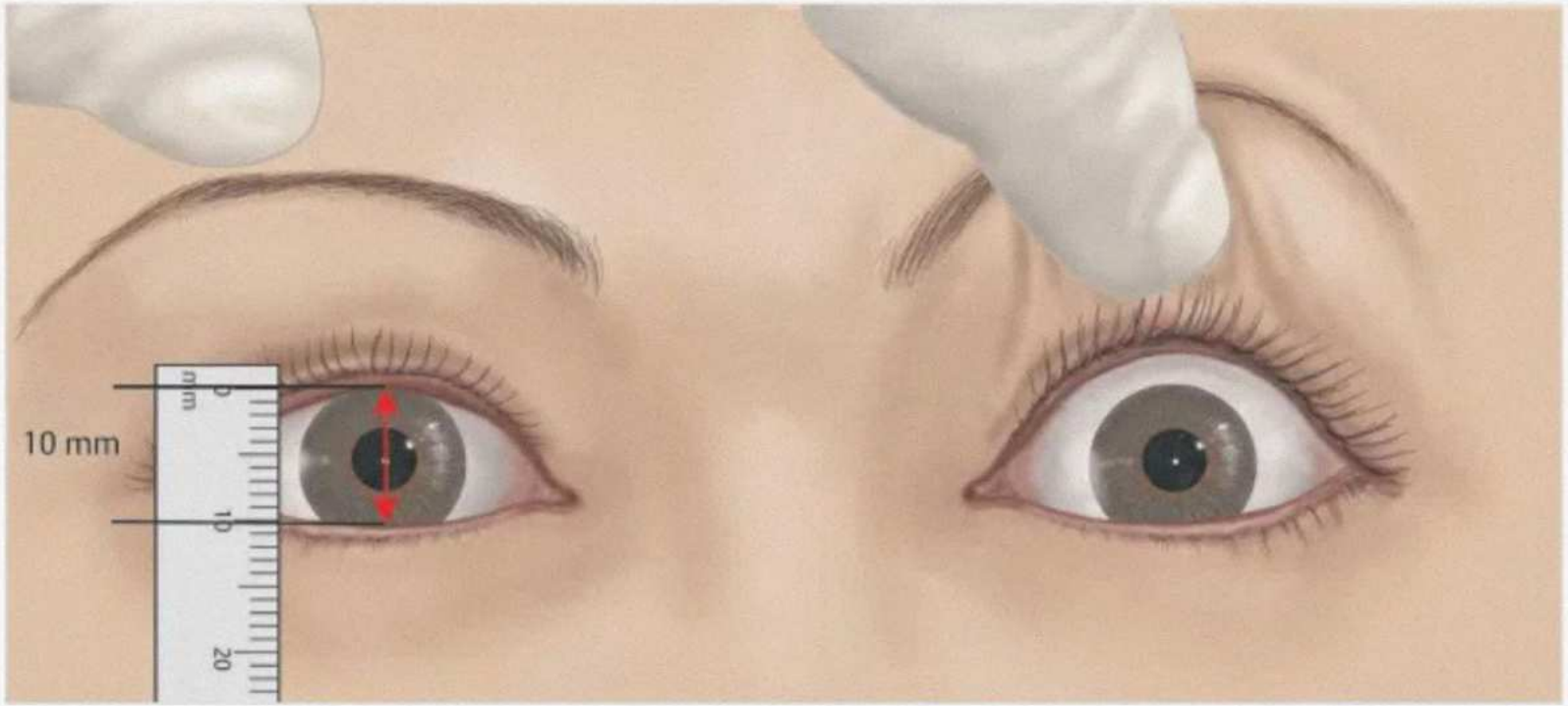
- MRD1: distance between upper lid margin and CLR. N: 4-4.5 mm
- MRD2: distance between lower lid margin and CLR. N: 5-5.5 mm



IIEI&H

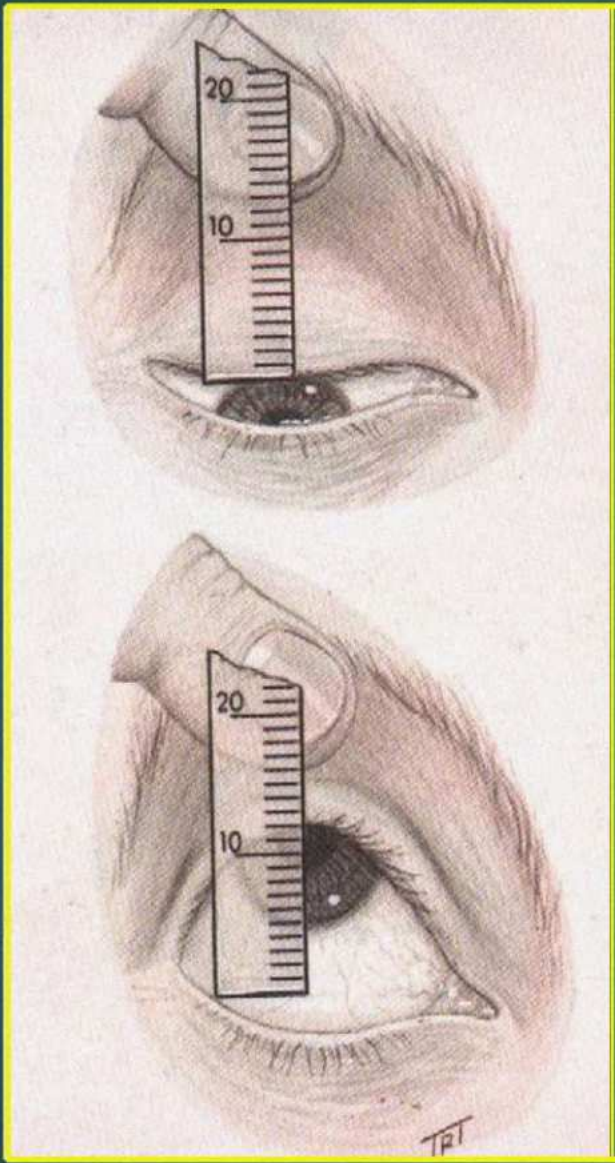
2. Palpebral fissure Height

- The distance between the upper and lower eyelid with the center of the pupil in primary gaze, with the patient's brow relaxed
- Normal – 9-10mm in primary gaze
- Amount of ptosis = difference in palpebral apertures in unilateral ptosis or Difference from normal in bilateral ptosis



3. Levator Function assessment

- It is determined by the lid excursion caused by LPS muscle (Burke's method)
- Patient looks down, and thumb is placed firmly against the eyebrow (to block the action of frontalis muscle)
- The patient looks up and the amount of upper lid excursion is measured with a ruler



- Reflects levator function
- Normal (15 mm or more)
- Good (8 mm or more)
- Fair (5-7 mm)
- Poor (4 mm or less)

Upper lid crease

- Distance between lid margin and lid crease in down-gaze
- Females 10 mm, males 8 mm
- Absence in congenital ptosis indicates poor levator function
- High crease suggests an aponeurotic defect

- Bells Phenomenon
- Jaw Winking and Lid Lag
- Fatigability
- Cogan twitch
- Corneal sensation
- Tear film
- Increased innervation

Should be noted

Normal values

Tests done for ptosis evaluation	Normal values
Palpebral fissure height	7-10mm(male) 8-12mm(female)
Margin reflex distance(MRD)1	4-5mm
Margin reflex distance(MRD)2	>5mm
Lid crease height	5-7mm(male) 8-10mm(female)
Levator function	13-17mm
Margin limbal distance	9mm
Bell's phenomenon	Upward rotation of eyeball with closure of eyelid

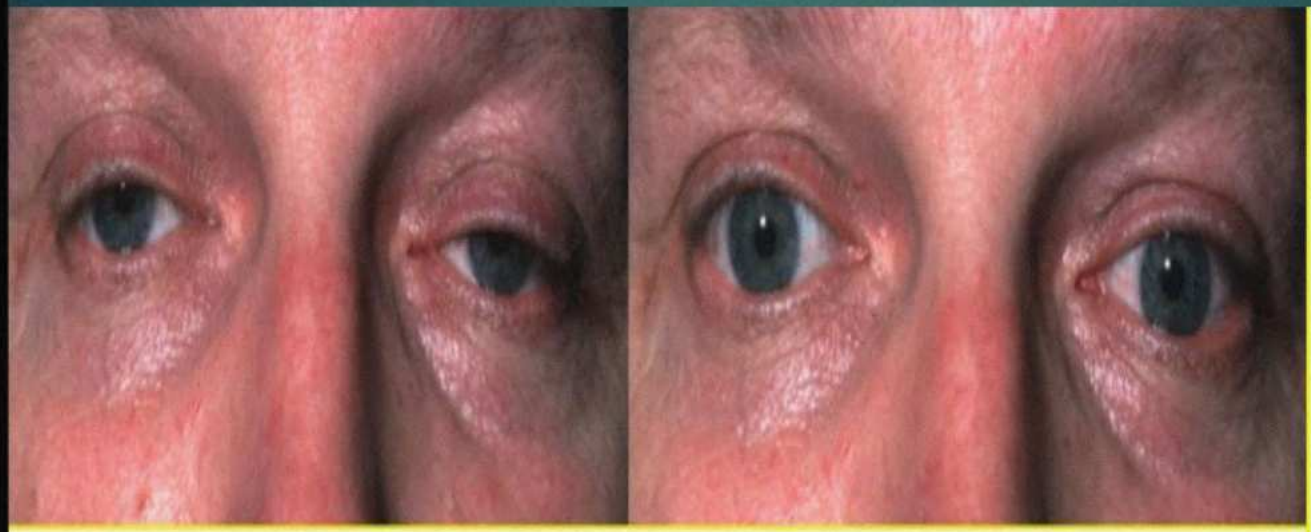
ICE Test

- Ice pack is applied on eyelid for five minutes
- Positive test is improvement of ptosis by 2mm or more
- Cold decreases the acetyl cholinesterase breakdown of acetyl choline

Investigation

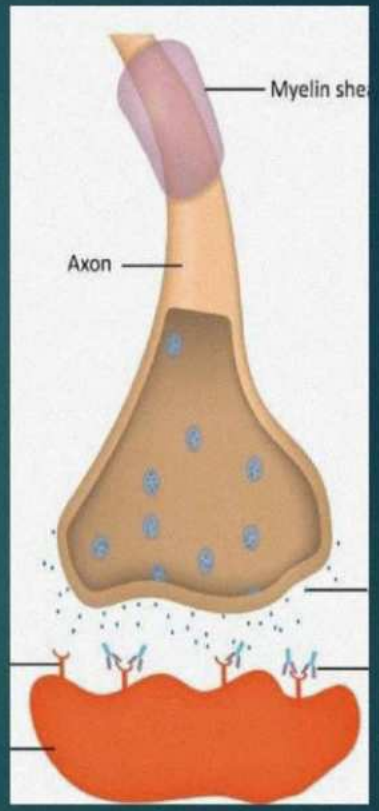
- Serum acetylcholine receptor assay
- Tensilon test
- EMG
- ECG
- T3, T4, TSH
- Imaging studies

TENSILON TEST



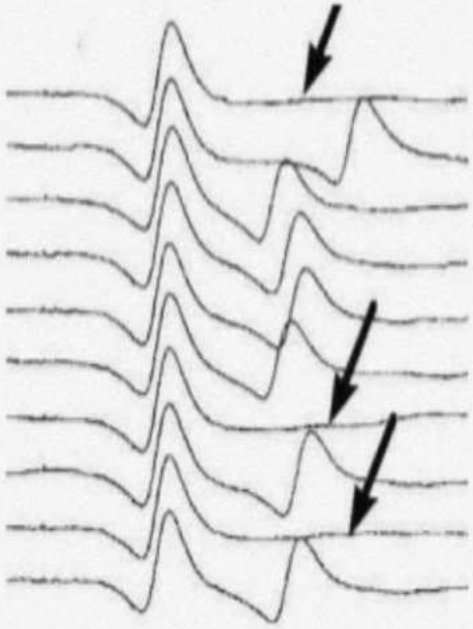
- Measure amount of ptosis or diplopia before injection
- Inject i.v. test dose of edrophonium
- Inject remaining dose if no hypersensitivity

MYASTHENIA GRAVIS(paradoxical reversal)



Single-fiber electromyography (EMG)

- It considered the most sensitive test for myasthenia gravis, detects impaired nerve-to-muscle transmission.



Neuro-imaging

- Sudden onset
- Vague history
- Post trauma
- Horner syndrome
- Associated neurological findings
- Third nerve palsy
- Tumors/ suspicion of malignancy

MANAGEMENT

- MEDICAL PTOSIS
- SURGICAL PTOSIS
- Pediatrician/internist/ Anaesthetist

Ptosis Surgery

5 important Factors

1. Aetiology
2. Age
3. Levator function
4. Severity of ptosis
5. Bell's phenomenon

Absence- contraindication for ptosis surgery

Congenital ptosis

Indications for IMMEDIATE surgery

- Amblyopia
- Abnormal head posture
- Significant astigmatism
- Strabismus

NO → Wait until 4 y/o

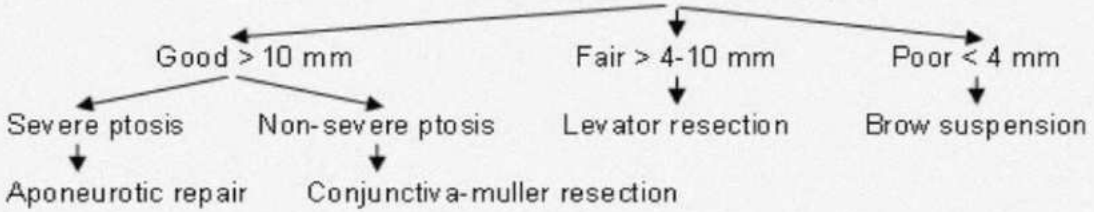
1. Accurate VA measurement
2. Accurate Ptosis assessment
3. Maturation of fascia lata
4. Normal facial development

YES

Order of Ptosis Surgery

1. Strabismus surgery
2. Lid margin abnormalities correction
3. Ptosis surgery

Levator function



Aponeurotic repair

Suturing of aponeurosis to the tarsal plate- through anterior / posterior approach

Conjunctiva-muller resection

Transconjunctival resection of Muller muscle together w underlying conjunctiva
Reattachment of resected end to the TARSAL plate
Maximum lid elevation- 3 mm

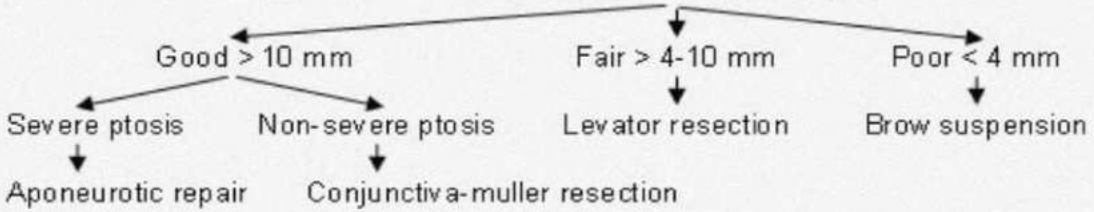
Levator resection

Shortening of levator complex- thro anterior / posterior approach

Brow suspension

Elevation of eyelid w Frontalis muscle via a sling

Levator function



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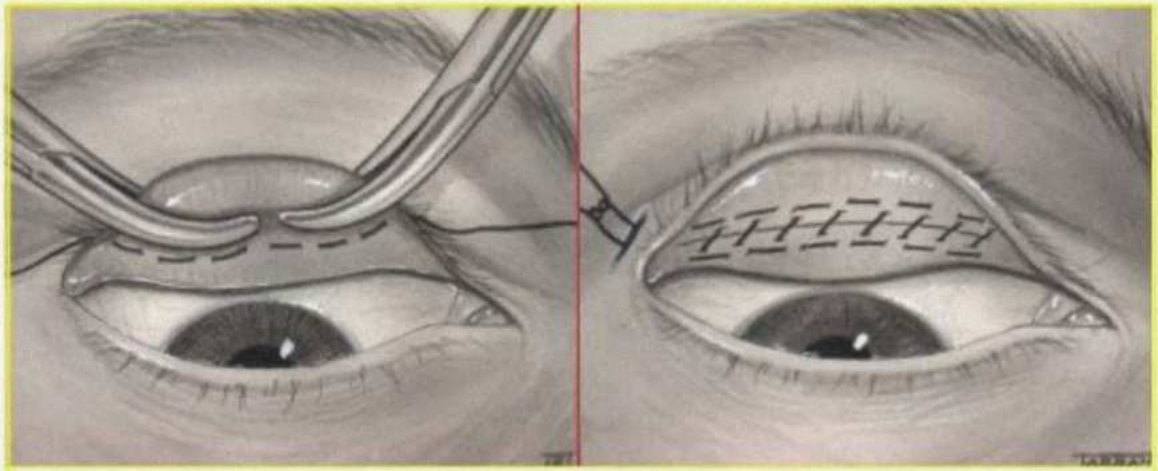
Fasanella-Servat procedure

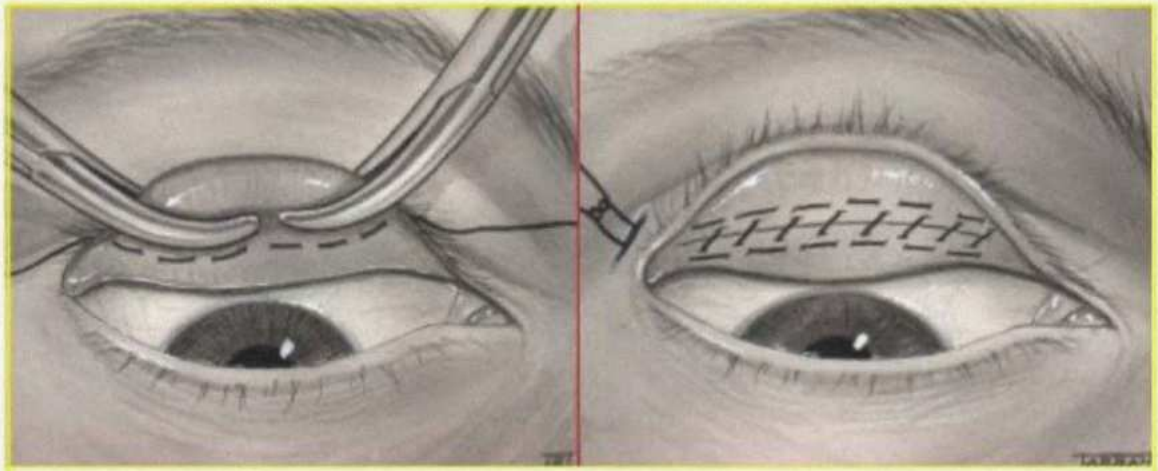
Indicated for mild ptosis(1.5-2mm) with good levator function



Excision of upper border of tarsus, lower border of Müller muscle and overlying conjunctiva

Fasanella-Servat procedure

Indicated for mild ptosis(1.5-2mm) with good levator function



Excision of upper border of tarsus, lower border of Müller muscle and overlying conjunctiva

Levator Resection

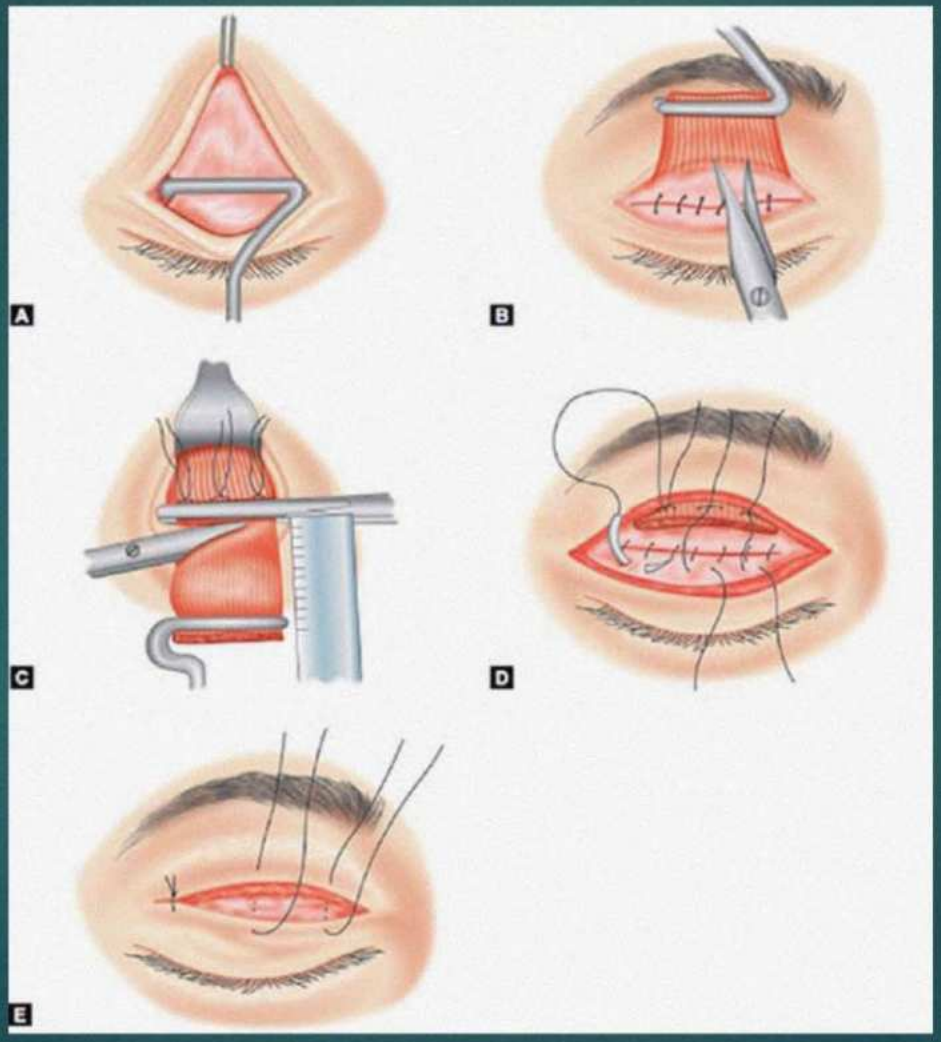
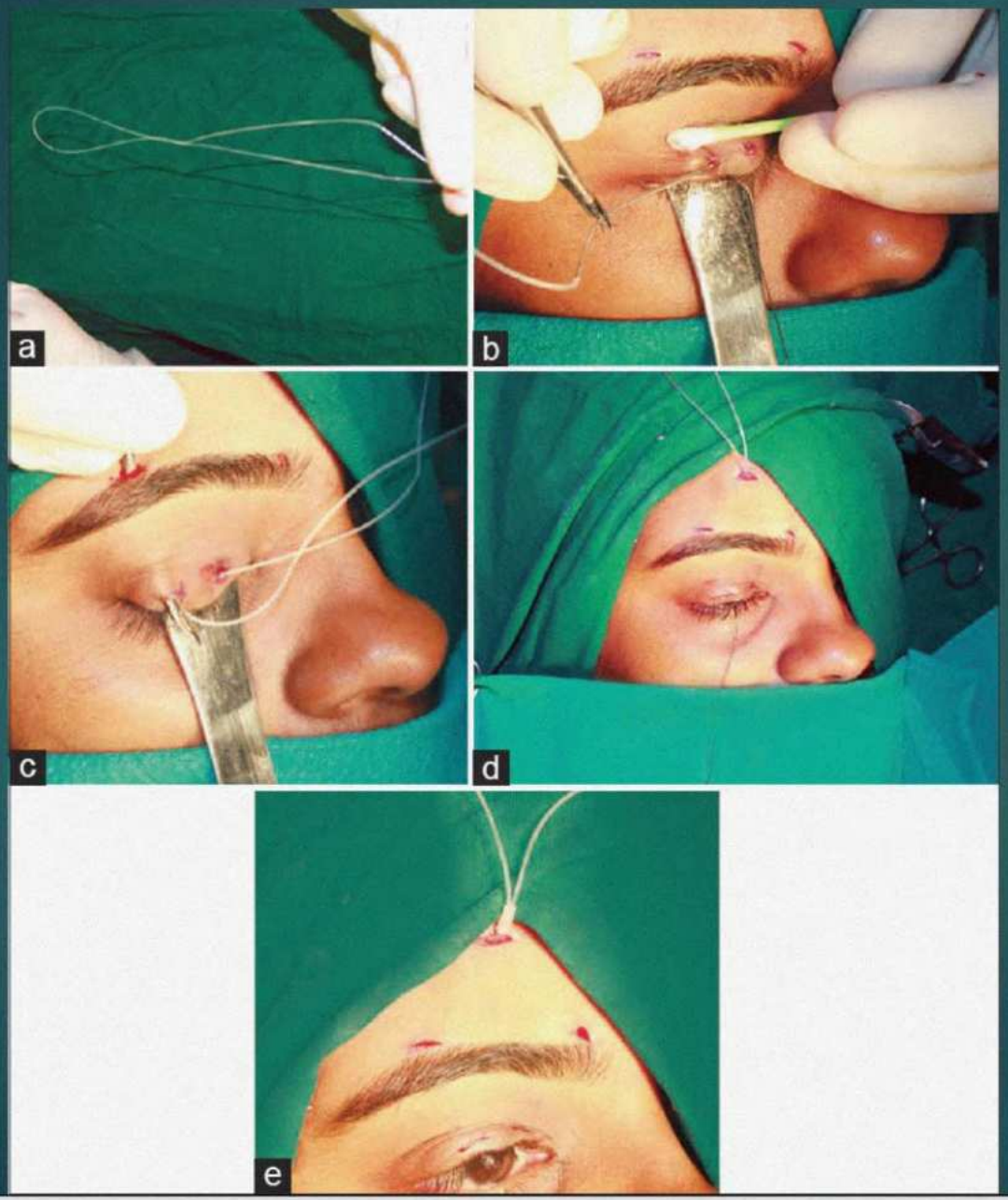


Table 3 Amount of levator resected based on LF

LF	Amount of resection
8–12 mm	10–13 mm
6–8 mm	16–18 mm
4–6 mm	22+ mm

LF, levator function.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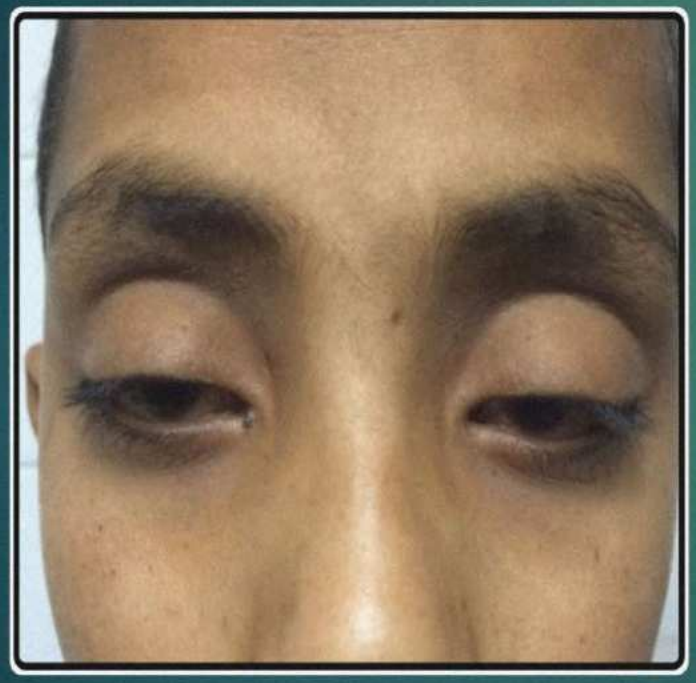
Ptosis props / crutches

Bilateral congenital Ptosis ALR

Bilateral congenital Ptosis
PLR



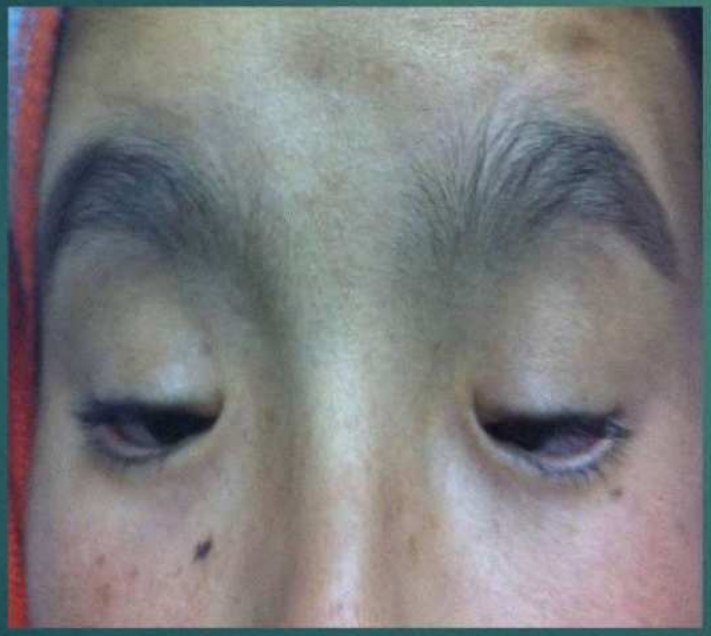
Unilateral Ptosis

Traumatic Ptosis

Bilateral FS (silicone)

Frontalis sling (Autologous fascia lata)

MCT shortening double Z Plasty FSS



- A 45 years old patient underwent cataract surgery. Surgery was complicated and prolonged. Patient developed ptosis postoperatively, was observed for 6 months but no improvement observed.

The procedure of choice in a patient with ptosis following cataract surgery who exhibits good levator function and a high upper eyelid crease is:

- Fasanella Servat procedure
- levator muscle resection
- Muller's muscle resection
- Reinsertion of levator aponeurosis
- Frontalis suspension

- An 82-year-old lady presents having had previous upper eyelid surgery in both eyes for involutional ptosis. She complains of left eye irritation and asymmetry of the lid appearance. On examination, she has 7mm of lid retraction on the left

What is the most suitable management?

- Aponeurosis recession
- Conservative treatment with lubricants
- Skin and hard palate graft to upper lid
- Mullerotomy

- A 38-year-old man presents with recurrent, unilateral, episodic temporal headache and periocular pain over 6 weeks. The pain lasts for up to an hour. His nose is congested during an attack. He has anisocoria and ptosis on the same side as the pain during an attack. He had a recent MRI head which was normal

- What is the most likely diagnosis?

- Central Horner's syndrome
- Hutchison's pupil
- Herpes zoster ophthalmicus
- Post-ganglionic Horner's
- Pre-ganglionic Horner's

- The history suggests cluster headache, which can cause a post-ganglionic Horner's syndrome

Topic: Proptosis - Basics

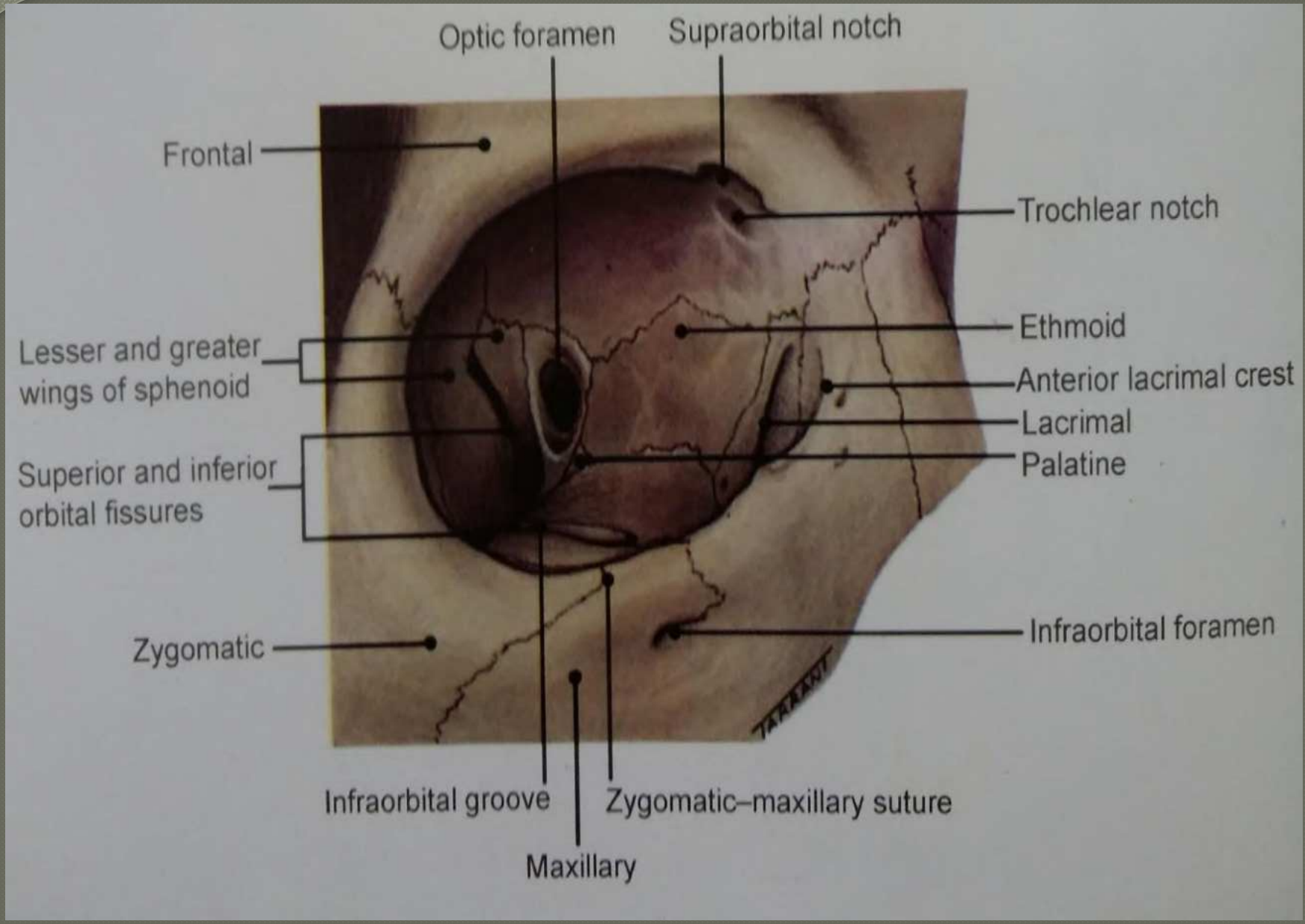
Learning objectives:

Discuss anatomy of orbit, definition enumerate differential diagnosis/causes etiology of proptosis in children and adults

Prof. Dr. Tariq Farooq Babar
Head & Incharge Eye "A" Unit
Khyber Girls Medical College,
MTI/Hayatabad Medical Complex,
Peshawar

Clinical anatomy of orbit

Orbit is a pear shaped cavity stalk of which is the optic canal



Anatomy of the orbit

Orbit has:

- Roof
- Lateral Wall
- Floor
- Medial Wall

Roof:

- Consist of two bones
  - Lesser wing of sphenoid
  - Orbital plate of frontal bone
- Defect in orbital roof – pulsatile proptosis
- Due to transmission of CSF pulsation to orbit.

Lateral wall:

- Consists of two bones
  - Greater wing of sphenoid
  - Zygomatic
- Anterior half of globe vulnerable to lateral trauma – it protrudes beyond the lateral orbital margin.

Floor:

- Consists of three bones
  - Zygomatic
  - Maxillary
  - Palatine
- Posteromedial portion of maxillary bone relatively weak – involved in a blow out fracture.

Orbital floor

- Forms roof of maxillary sinus
- Maxillary carcinoma invading orbit, displace globe upwards.

Medial wall:

Consists of four bones

- Maxillary
- Lacrimal
- Ethmoid
- Sphenoid

Lamina papyracea forms part of medial wall

Paper thin, perforated by numerous foramina for nerves and blood vessels.

Orbital cellulitis is therefore secondary to ethmoidal sinusitis.

Proptosis / Exophthalmos

Definition

- Abnormal protrusion of globe
- Displacement of globe relative to orbital rims
- Proptosis of more than 21mm or
- More than 2mm asymmetry between the two eyes is abnormal

Exopthalmos

Specially used to describe the proptosis of eyes associated with thyroid eye disease

Enophthalmos

Defined as retro displacement of eye into orbit

Conditions that mimic proptosis (Pseudo proptosis)

Ipsilateral large globe

- Megalophthalmos
- Buphthalmos
- High myopia

Ipsilateral lid retraction

- Contralateral anophthalmos

Dystopia

Displacement of globe in coronal plane

Differential diagnosis of Proptosis

Vascular

- Carotid cavernous fistula
- Cavernous sinus thrombosis
- Arteriovenous malformations such as
  - Hemangioma
  - Aneurysm
  - Varix

Trauma

- Retrobulbar hemorrhage
- Post-traumatic mucocele
- Encephalocoele (due to orbital roof fracture)

Endocrine

- Thyroid associated ophthalmopathy
- Grave's disease

Infective

- Orbital cellulitis
- Mucormycosis
- Granuloma

Inflammatory

- Orbital pseudotumor
- Myositis
- Granulomatous disease
- Sarcoidosis

Tumor

Primary like

- Schwannoma
- Lymphoma
- Optic nerve glioma

Metastases from distant sites commonly

- Leukemia
- Sarcomas

Pseudoproptosis

- Contr-lateral enophthalmos
- Contra-lateral globe rupture

Causes of proptosis in children & adults
Childhood proptosis

Congenital
- Exorbitism
- Cranio synostosis
- Skull anomalies
- Meningocele / encephalocele
- Dermoid cyst

Traumatic
- Orbital hematoma
- Traumatic hemorrhage in existing neoplasm

Inflammatory
- Orbital cellulitis
- Abscess
- Pseudo tumour
- Mucocele

Neoplastic

Ophthalmic / orbital

- Hemangioma
- Optic nerve glioma
- Rhabdomyosarcoma
- Orbital retinoblastoma
- Teratoma

Non ophthalmic

- Granulocystic sarcoma
- Metastatic neuroblastoma
- Lymphoma lymphosarcoma
- Histiocytosis x

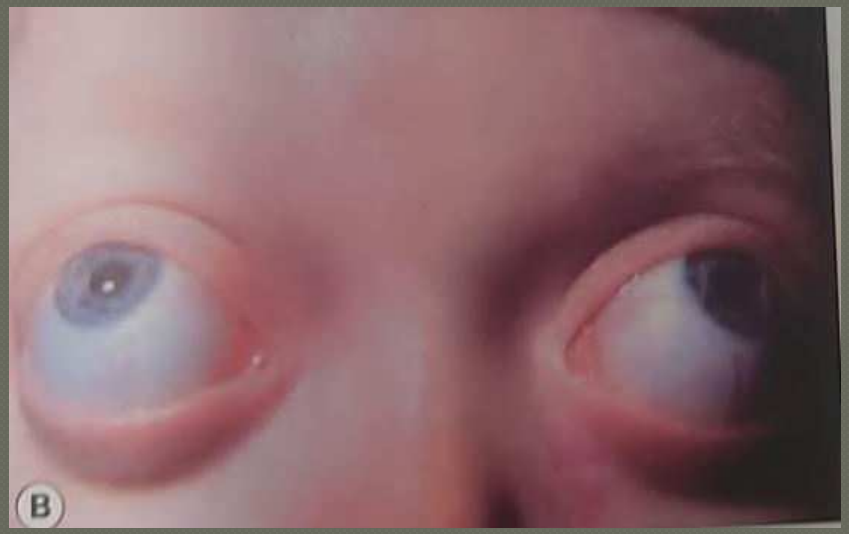
Congenital

Exorbitism

e.g Craniosynostosis

- Group of congenital conditions
- Abnormally shaped skull
- Cause being premature closure of skull sutures. e.g Crouzon syndrome and Apert syndrome and Pfeiffer syndrome


A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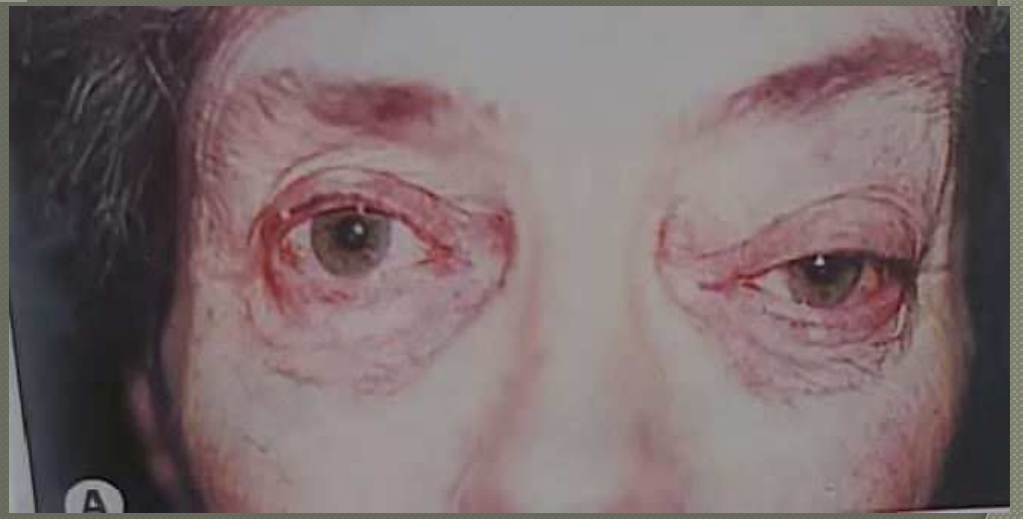
Encephalocoele

- Formed by herniation of intracranial contents through a congenital defect of the base of the skull
- Located at the front or back of the head
- A meningocoele contains only dura
- Meningo – encephalocoele also contains brain tissue.

Dermoid cyst

- A choristoma is a mass of histologically normal tissue in an abnormal location.
- It is derived from displacement of ectoderm to a subcutaneous location along embryonic lines of closure.
- Dermoids are lined by keratinized stratified squamous epithelium.
- They are smooth, non-tender 1-2cm in diameter


A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Traumatic

Orbital hematomas

- Can occur spontaneously
- Result of vascular anomalies
- Induced by trauma
- Following paranasal sinus surgery

Risk

Retrobulbar hematoma can compress optic nerve

Inflamatory

Orbital cellulitis

- In children usually secondary to ethmoiditis
- Child very unwell with high fever
- Rapid onset of proptosis usually down and out
- Pain, chemosis, lid oedema with restricted ocular motility
- In severe cases – there may be signs of optic nerve dysfunction


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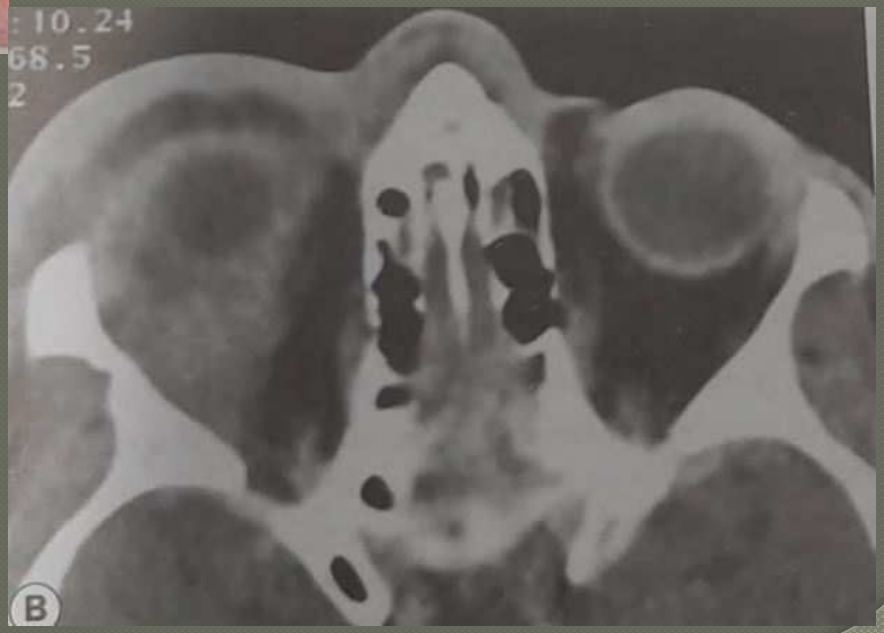
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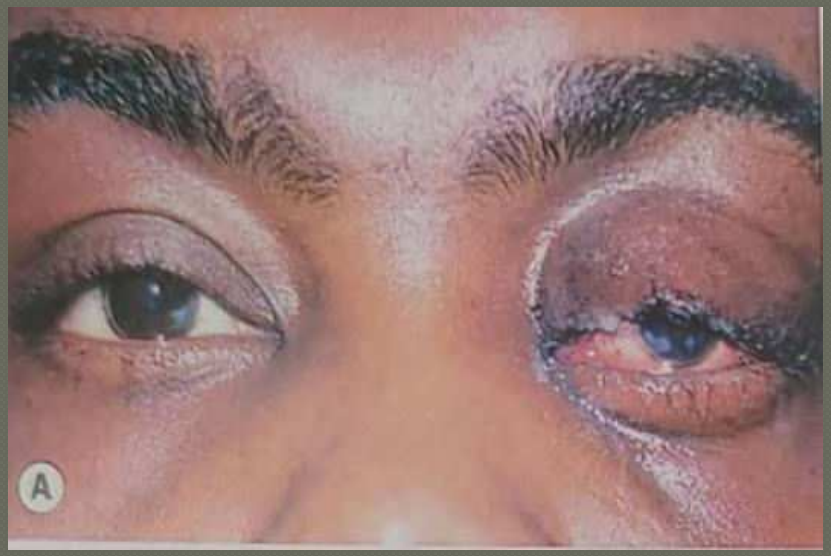
68.5
2
B

Pseudotumor (Idiopathic orbital inflammatory syndrome)

- Presentation between ages of 6 & 14 years
- 1/3rd of patients have bilateral involvement
- Subacute onset, axial proptosis associated with chemosis and lid oedema
- Look for Wegener granulomatosis in bilateral cases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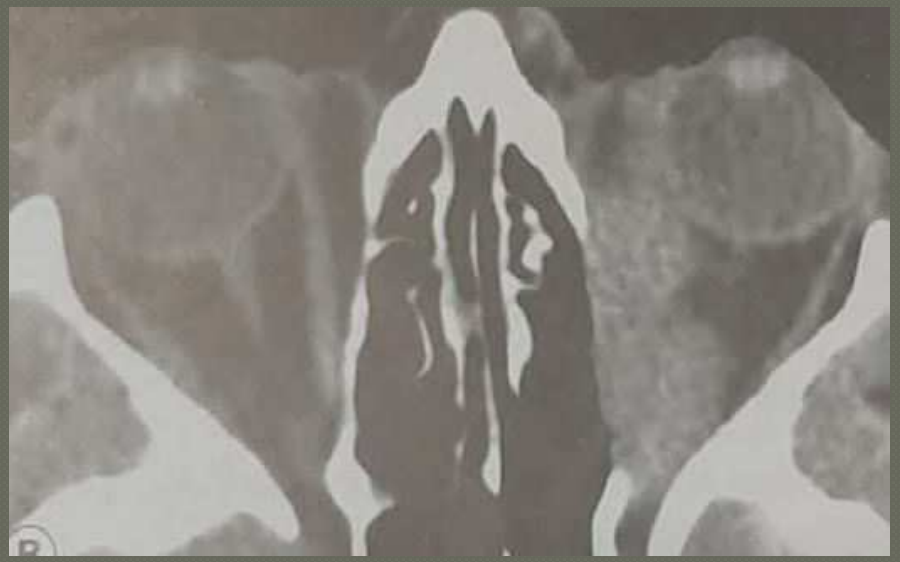
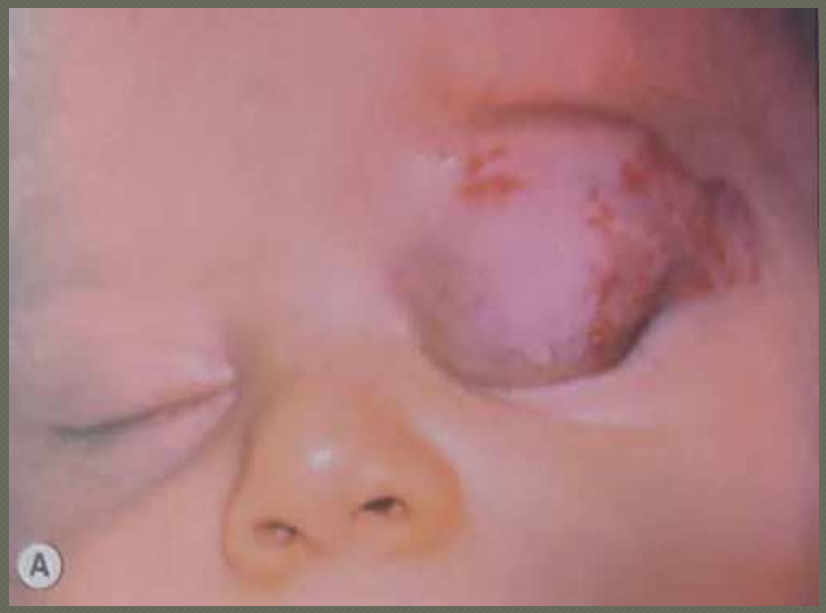



C

Neoplastic

Capillary hemangioma

- Presents usually at birth or early infancy
- Slowly progressive proptosis associated with an upper anterior orbital mass
- The mass becomes engorged and sligtly increases in size when the child cries
- The child may have capillary skin hemangiomas on the eyelids or else where.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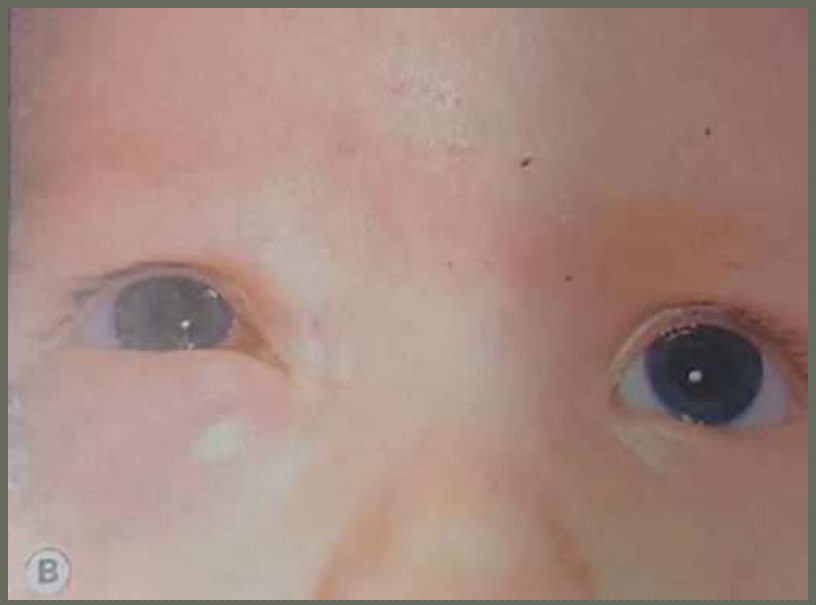
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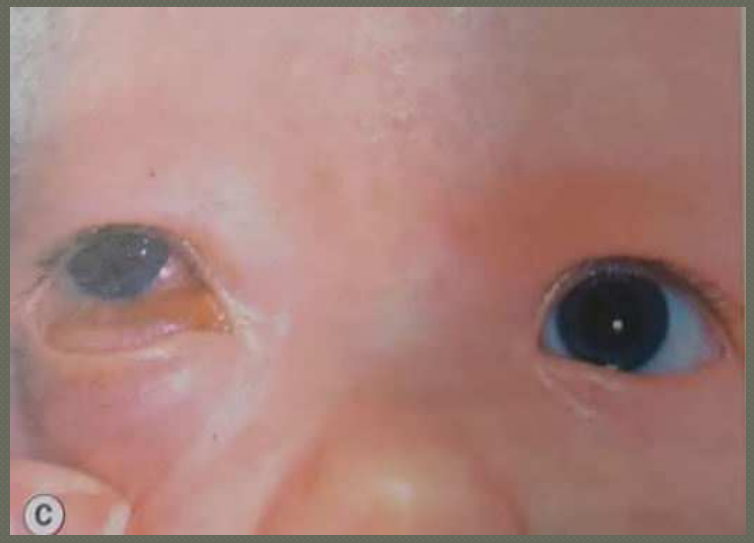
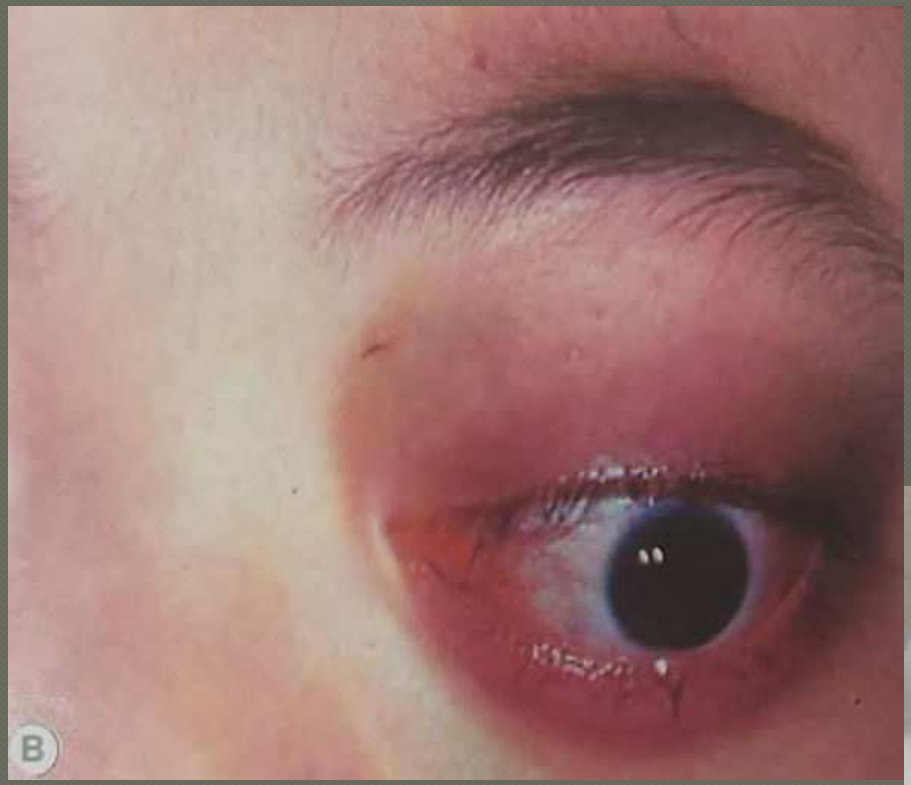

C

Optic nerve glioma

- Presentation between age of 2 and 7 years
- NF-I present in 50% of unilateral tumors and 100% in bilateral tumors
- Slowly progressive axial or non axial proptosis
- Decrease VA and an APD
- Optic disc – swollen, atrophic, optico cilairy shunts
- Disproportionate loss of VA compared to proptosis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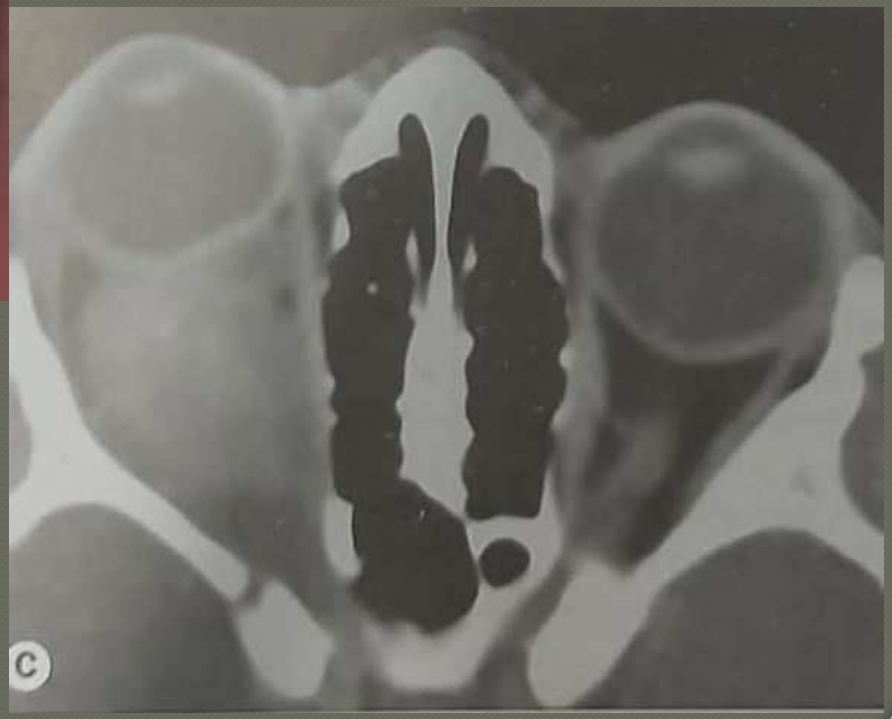
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Rhabdomyosarcoma

- Presentation at age 7 years
- More common in boys than in girls
- Rapid onset of progressive painful proptosis with chemosis and lid oedema
- Location – retrobulbar followed by superior and inferior.

Metastatic neuroblastoma

Presents during 1st 5 years of life

Primary tumor

usually develops in the abdomen

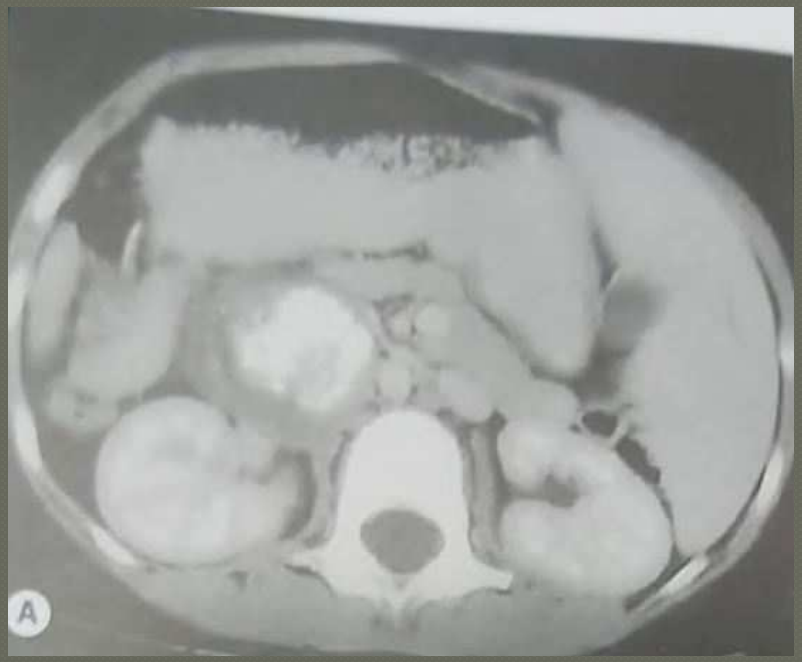
Metastatic tumor

Arising from neck or medinastinum may cause Horner syndrome

40% of metastasis involve both orbits

Sudden onset of rapidly progressive proptosis

Associations – echymosis and superolateral orbital mass



Histiocytosis x / Langerhans cell histiocytosis

A rare multisystem disorder consisting of three related and overlapping conditions

- Eosinophilic granuloma – lesions confined to bone
- Hand – Schuller – Christian disease - a triad of diabetes inspidus, proptosis and bony skull defects
- Letterer – Siwe disease – aggressive visceral involvement.

- Orbital involvement in 25% of cases with either Eosinophilic granuloma or Hand Schuller Christian disease.
- Bilateral or unilateral bony lysis and soft tissue growth
- Typically involves superolateral orbit

Proptosis in adults

Table 2: Common causes of proptosis in adults

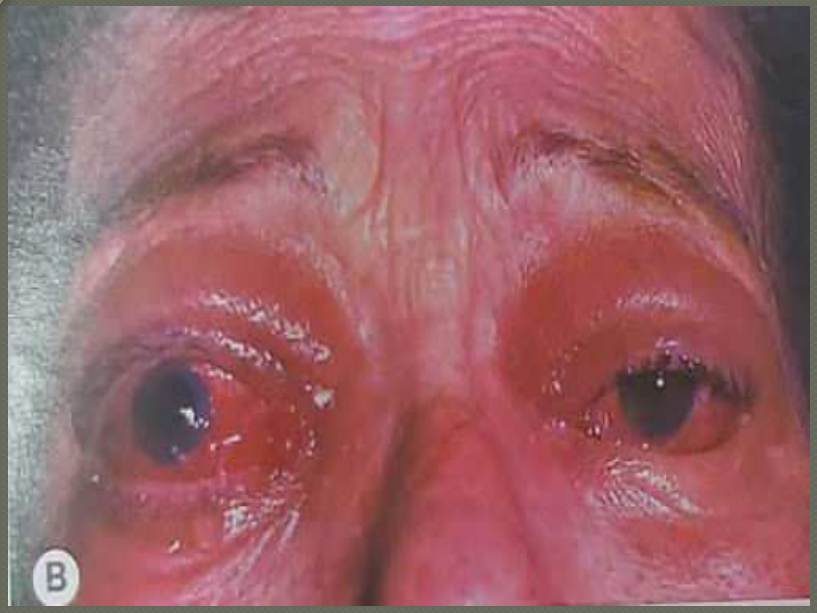
Category	Specific conditions
Endocrine	Thyroid eye disease
Trauma	Facial fracture, soft tissue swelling, retrobulbar haemorrhage
Vascular	Carotid-cavernous fistula, cavernous sinus thrombosis, cavernous haemangioma
Inflammatory	Inflammatory orbital pseudotumor, dacryoadenitis, orbital myositis, Tolosa–Hunt syndrome, Wegener's granulomatosis, sinus mucocele, sarcoidosis, Churg–Strauss syndrome
Infective	Orbital cellulitis, mucormycosis
Tumours	Lymphoma, schwannoma, sinonasal tumour, lacrimal gland tumour, meningioma, neurofibroma, optic nerve glioma, metastasis, myeloid sarcoma, ossifying fibroma, orbital osteoma, haemangioblastoma, neuroblastoma, neurofibroma, acute leukaemia
Other	Paget's disease, fibrous dysplasia, Langerhans cell histiocytosis, Erdheim–Chester disease

Endocrine

Thyroid eye disease

- Most common cause of proptosis in adults
- Axial proptosis
  - Unilateral
  - Bilateral
- Lid retraction and lid lag
- Injection over horizontal recti

- Superior limbic kerato conjunctivitis
- Restricted ocular motility defects
- Signs of optic nerve compression
- Chorio retinal folds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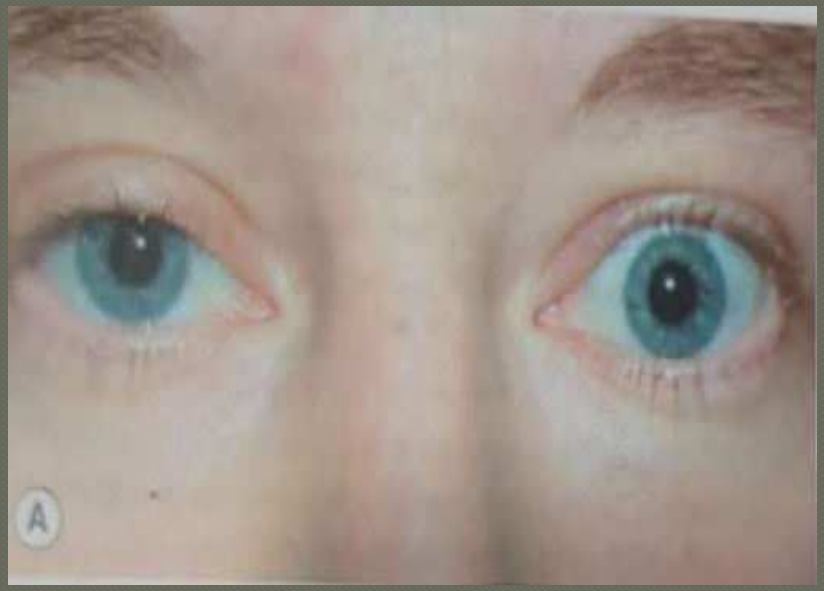
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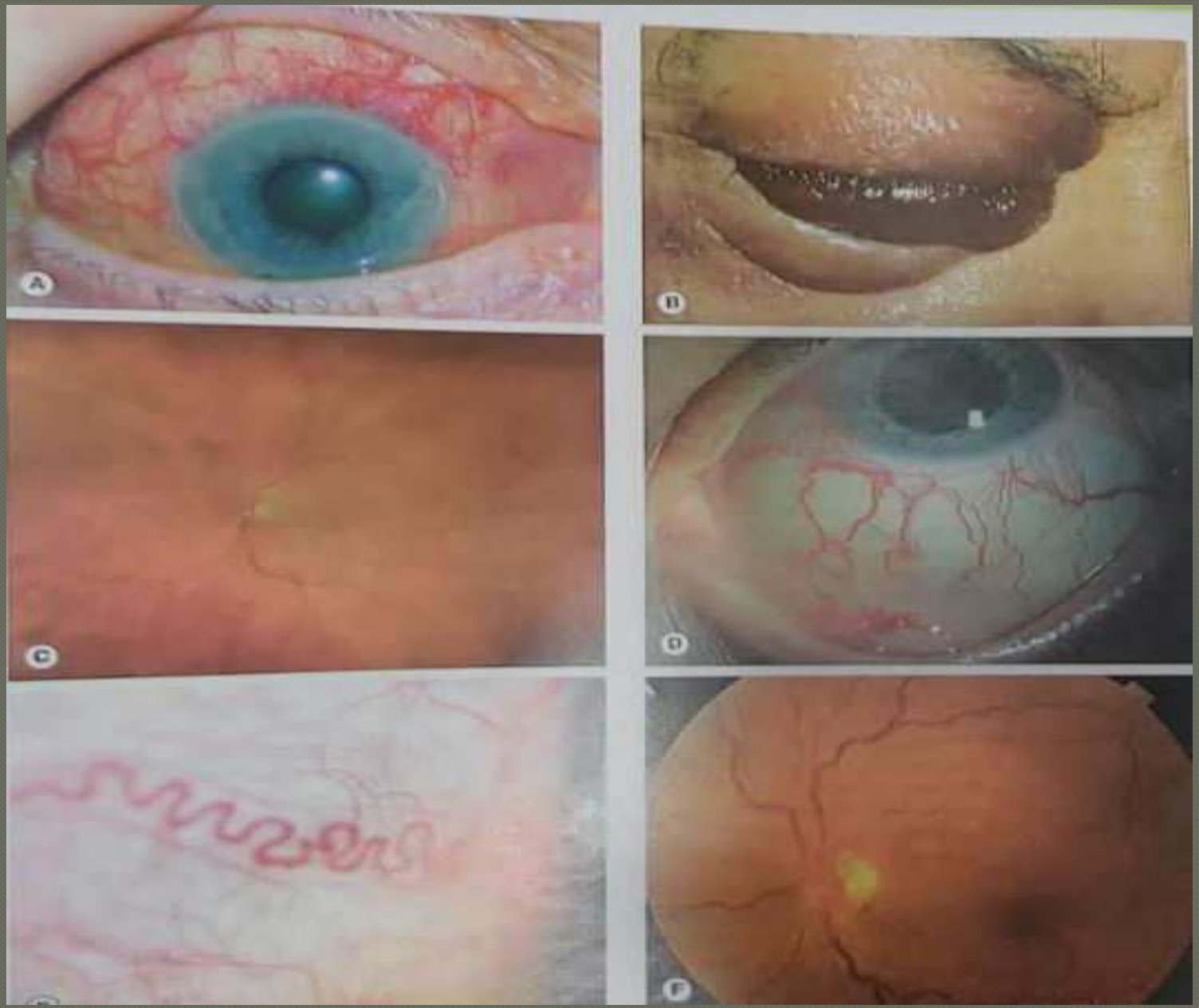
Vascular

Carotid – cavernous fistula

- Caused by either head trauma or a spontaneous rupture of an intracavernous aneurysm
- Unilateral painful pulsatile proptosis associated with a bruit
- Grossly dilated epibulbar vessels
- Ophthalmoplegia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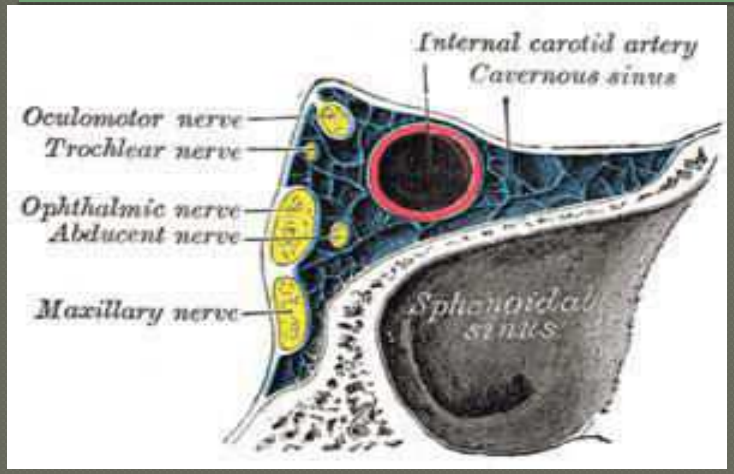
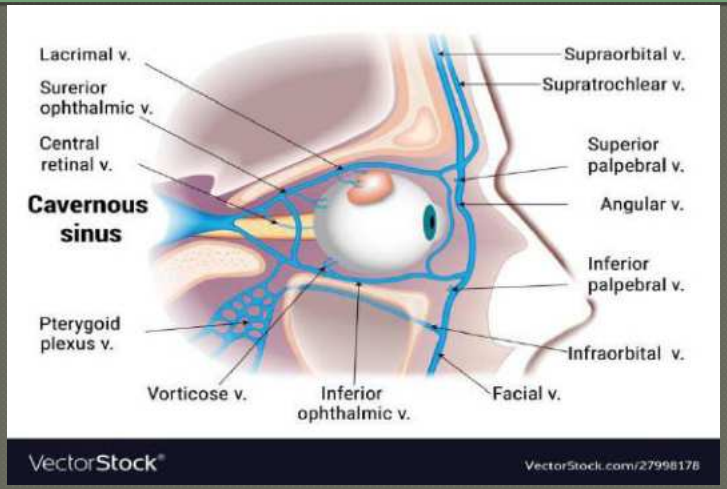
Cavernous sinus thrombosis

- A serious condition
- Most commonly secondary to skin or paranasal sinus infection such as sinusitis, orbital or preseptal cellulitis or otitis.
- Similar to a CCF except that the patient is usually more ill because of systemic infection.
- Patient have severe headache, vomiting, unilateral or bilateral proptosis.
- Congestion of conjunctival or retinal veins, reduced vision, 3rd to 6th CN palsy.





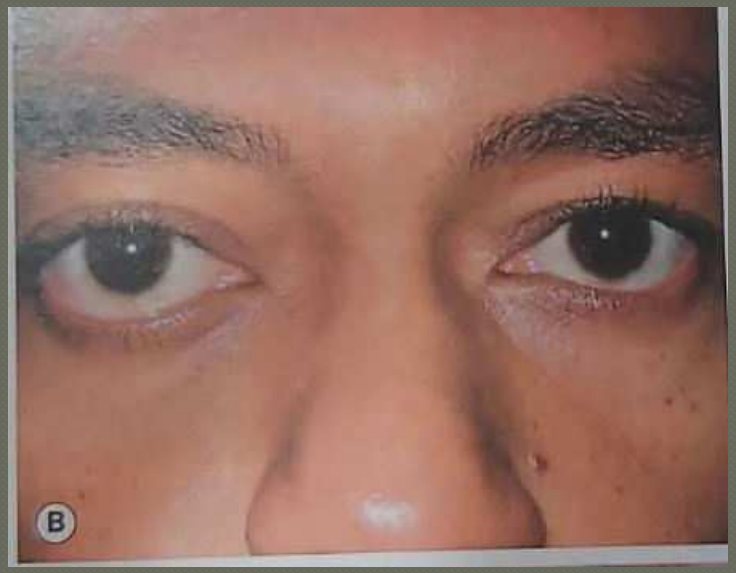
Clinical features

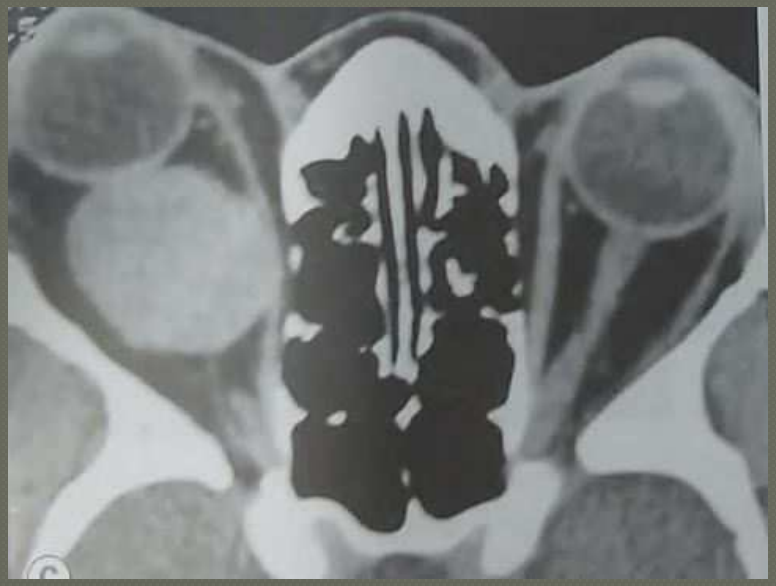
- Infections from face, orbit, sphenoid sinus can cause thrombosis.
- III,IV,V1,V2,VI can be involved resulting in various diplopias
- Rupture of ICA can cause pulsatile exophthalmos

Caput medusae in cavernous sinus thrombosis

Cavernous hemangioma

- Presents during the 4th & 5th decades of life.
- Most commongly encountered benign orbital tumor in adults.
- Unilateral, usually axial, slowly progressive proptosis.
- Look for hypermetropia and chorioretinal folds.



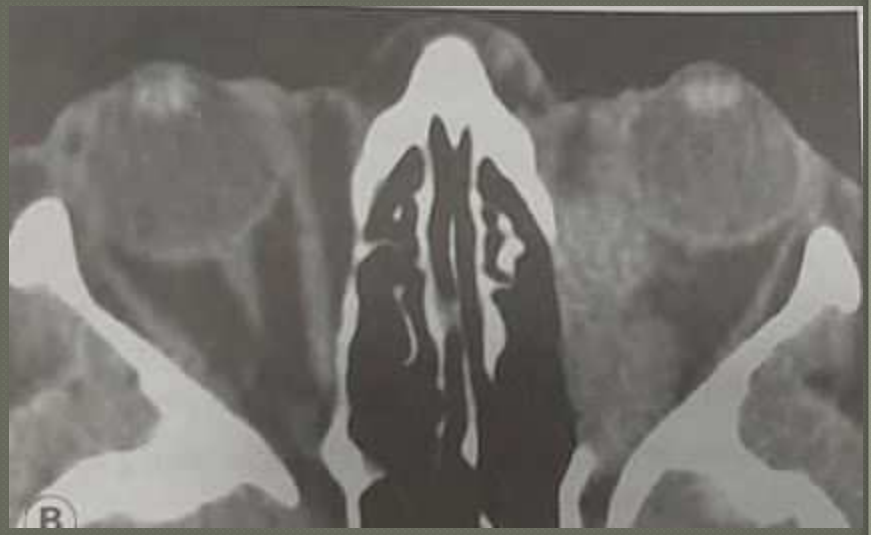


Inflammatory

Orbital Pseudo-tumor

- Presents between ages of 20 & 25 years
- Acute orbital myositis is a type of pseudotumor which primarliy affects one or more of the extraocular muscles.
- Usually unilateral axial proptosis
- In patients with acute orbital myositis, pain and diplopia increases on attempted gaze into the field of the affected muscle


A


B

Mucoceles

- Presents with a combination of ptosis, proptosis and globe displacement.
- The proptosis may fluctuate when the walls of the mucocele become inflammed.
- Frontal mucocele displaces the globe downward.
- Ethmoidal mucocele causes lateral displacement.

Infective

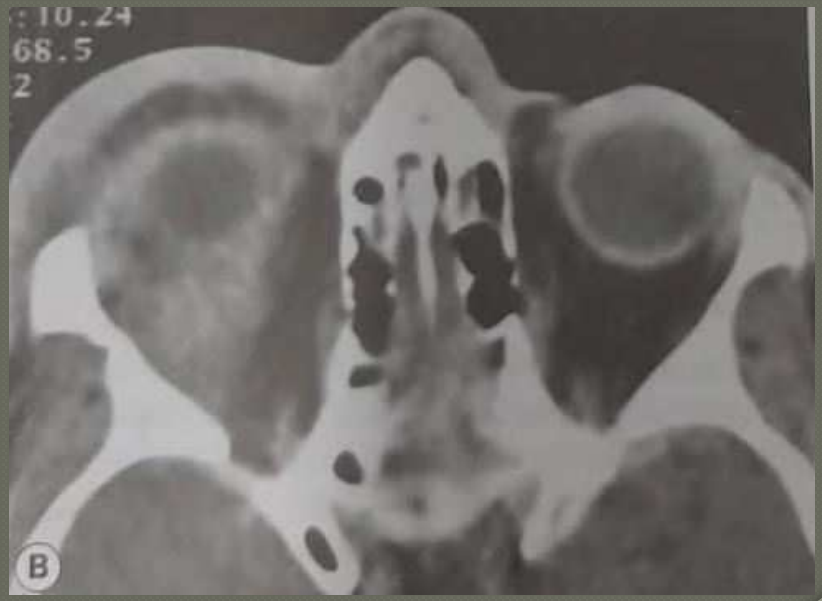
Orbital cellulitis

- There are four types of orbital cellulitis
  - Associated with sinus infection
  - From adjacent structures (e.g dacryocystitis, midfacial septum
  - Post-traumatic in injuries which penetrate the orbital septum.
  - Post-surgical (e.g retinal detachment, strabismus, lacrimal and orbital surgery).

- Unilateral painful proptosis and severe lid oedema in a very unwell patient.

Ophthalmoplegia

Look for signs of optic nerve compression.



Tumours

Lymphoma

- Presentation of lymphoid tumors is usually in old age.
- Unilateral or bilateral involvement with periorbital puffiness
- Anteriorly located lesion have rubbery consistency
- Conjunctival extensions may be present.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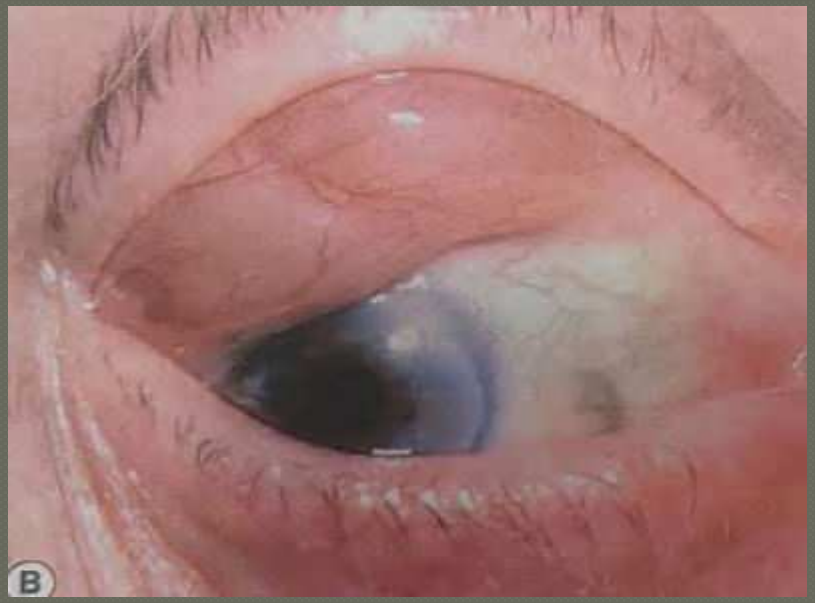
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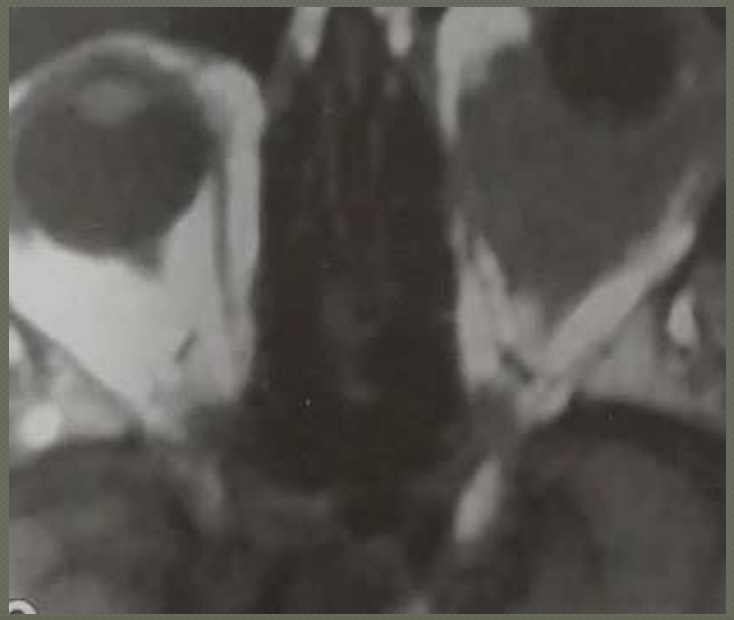
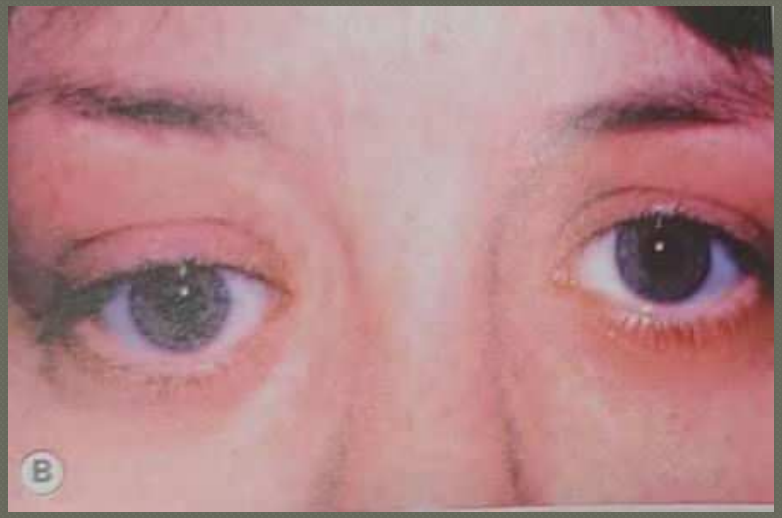


Lacrimal galnd tumors

Pleomorphic adenoma

- Presentation is usually in middle age
- Chronic, painless fullness of the eyelid
- Displacement of the globe downward and inward.
- Diplopia when gaze is directed towards the lesion i.e upward and outward.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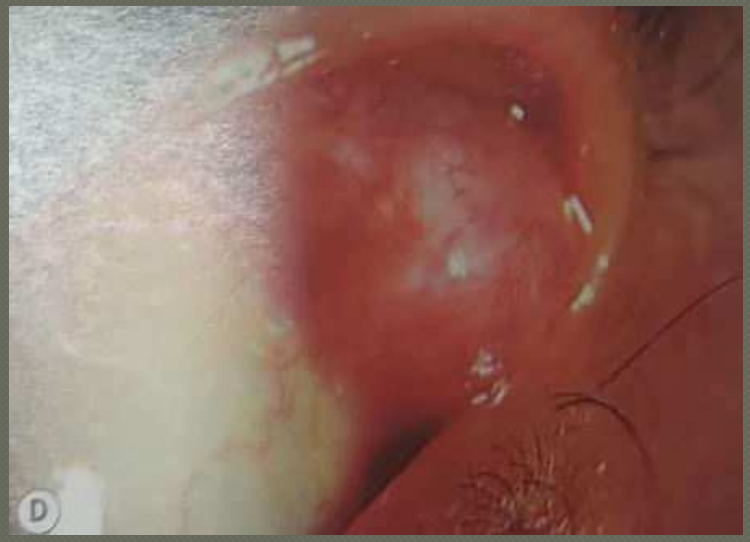
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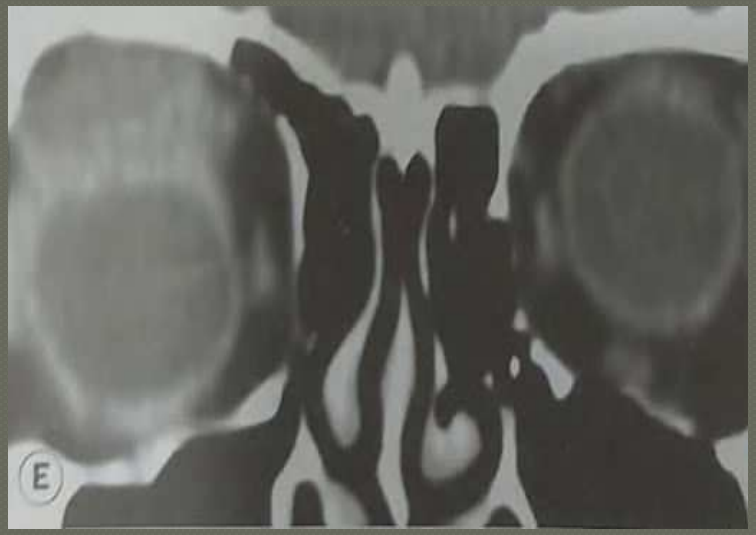
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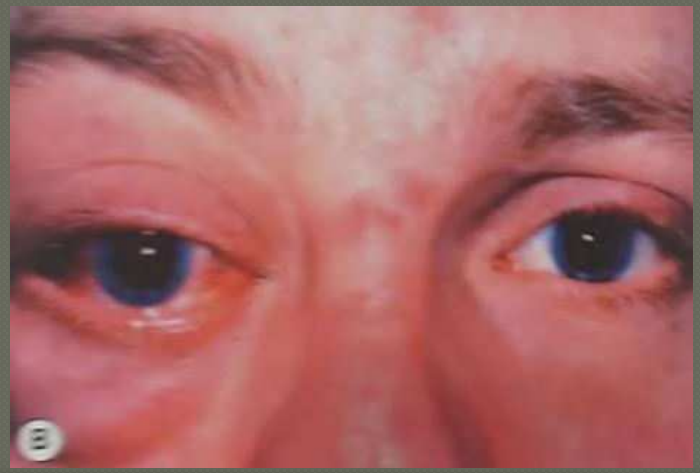
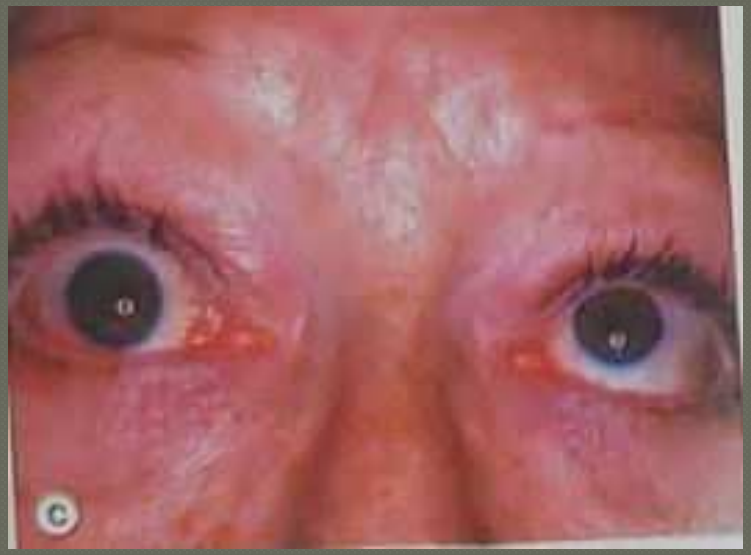
Malignant lacrimal gland tumors

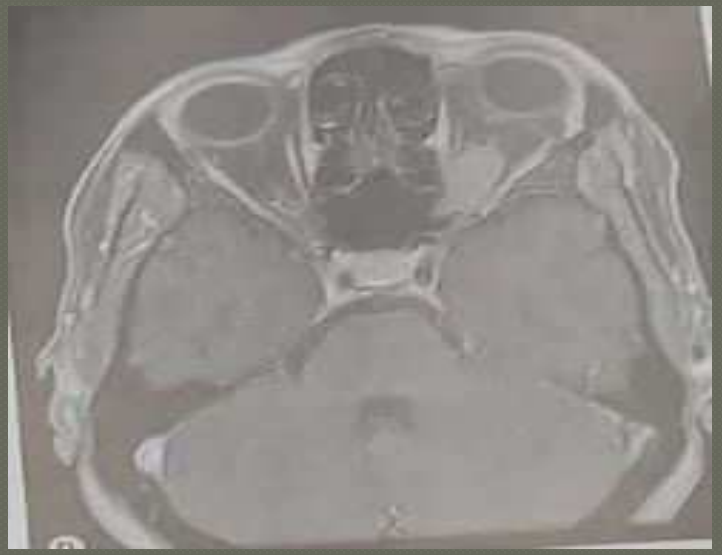
- Have shorter history than benign tumors
- Associated with pain and diplopia
- Pleomorphic adenocarcinoma occur in old age
- Adenoid cystic carcinoma occur around 40 years.

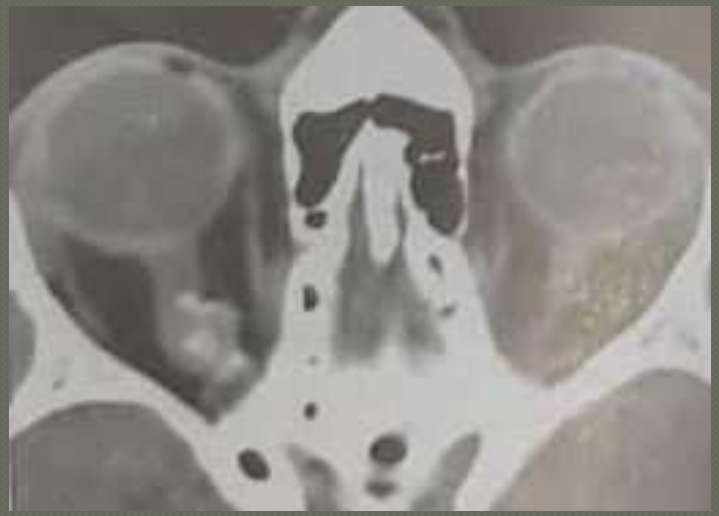


Optic nerve sheath meningioma

- Presents usually in the middle age
- Female to male ratio is 3:1
- Unilateral, slowly progressive axial proptosis
- Early decrease in VA
- There may be optic atrophy and opticociliary shunts

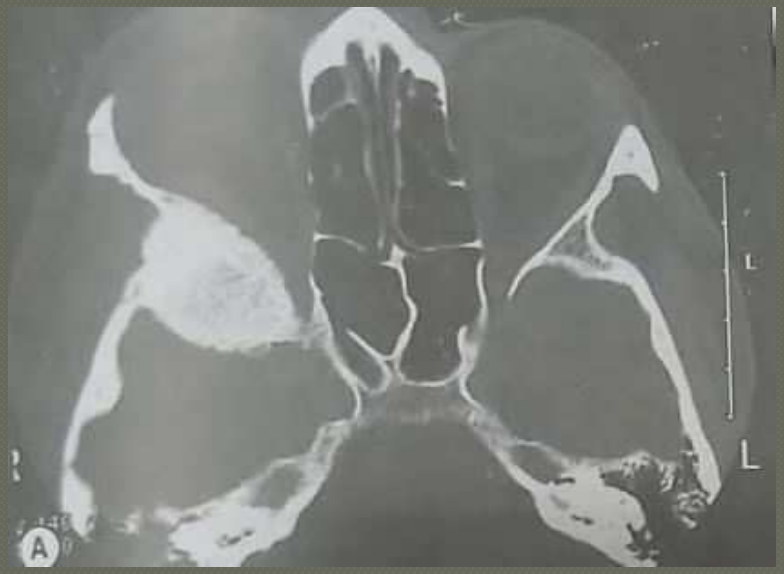






Sphenoidal ridge meningioma

- Presents in middle age with proptosis and reactive hyperostosis
- Slowly progressive painless, downward and outward proptosis
- Fullness of temporal fossa
- Optic nerve dysfunction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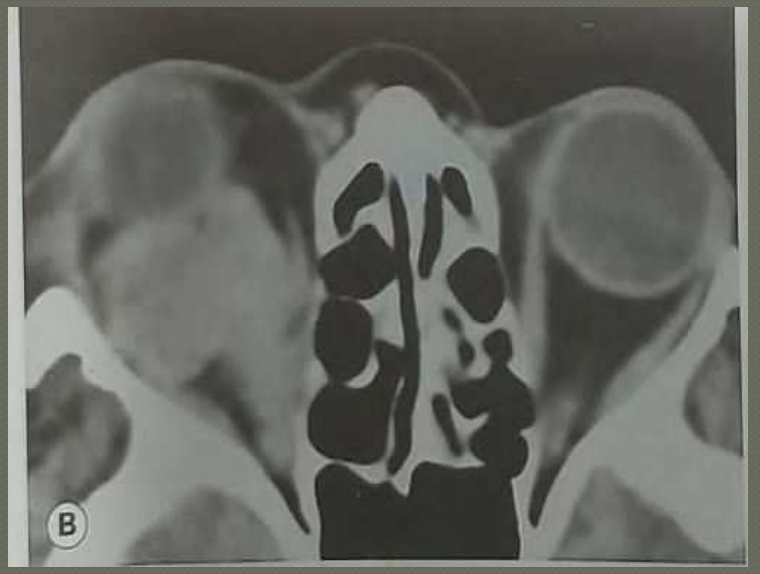
Metastatic tumors

- Presentation is usually with a rapid onset of diplopia, lid edema and pain
- Most metastases from tumors cause proptosis while secondaries form scirrhous breast carcinoma give rise to enophthalmos.


A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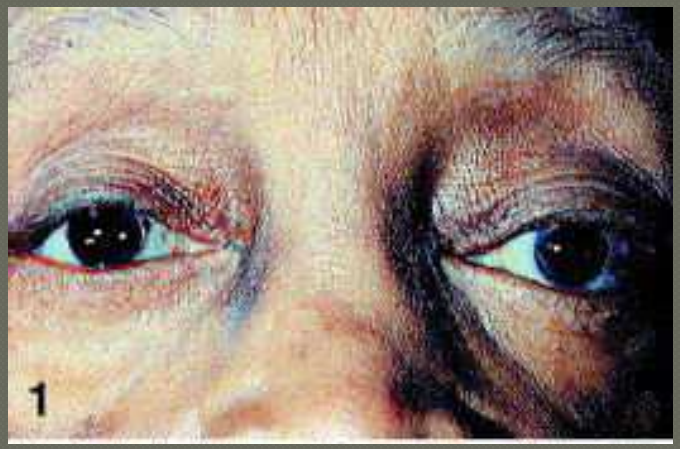
Isolated neurofibromatosis

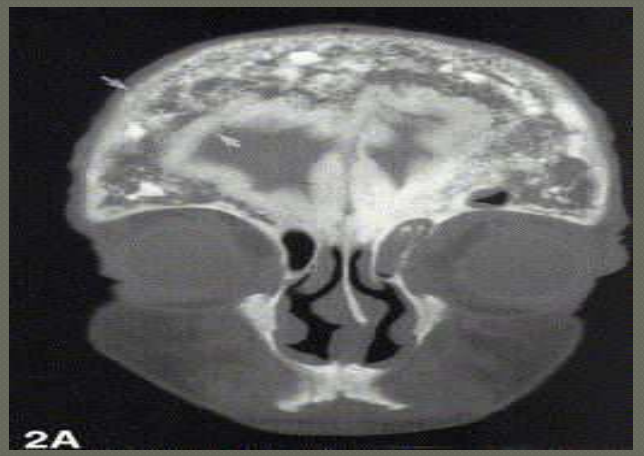
- 10% of patients have NF1
- Presentation is in the 3rd to 4th decade
- Insidious mild painful proptosis
- Usually not associated with visual impairement or ocular motility dysfunction.

Other

a) Paget disease of the skill

- May cause slowly progressive unilateral or bilateral proptosis.
- Ocassionaly patients develop osteosarcoma





Thanks

Topic: Proptosis Basics

Learning objectives: Discuss the clinical features, investigations and management of proptosis in children and adults

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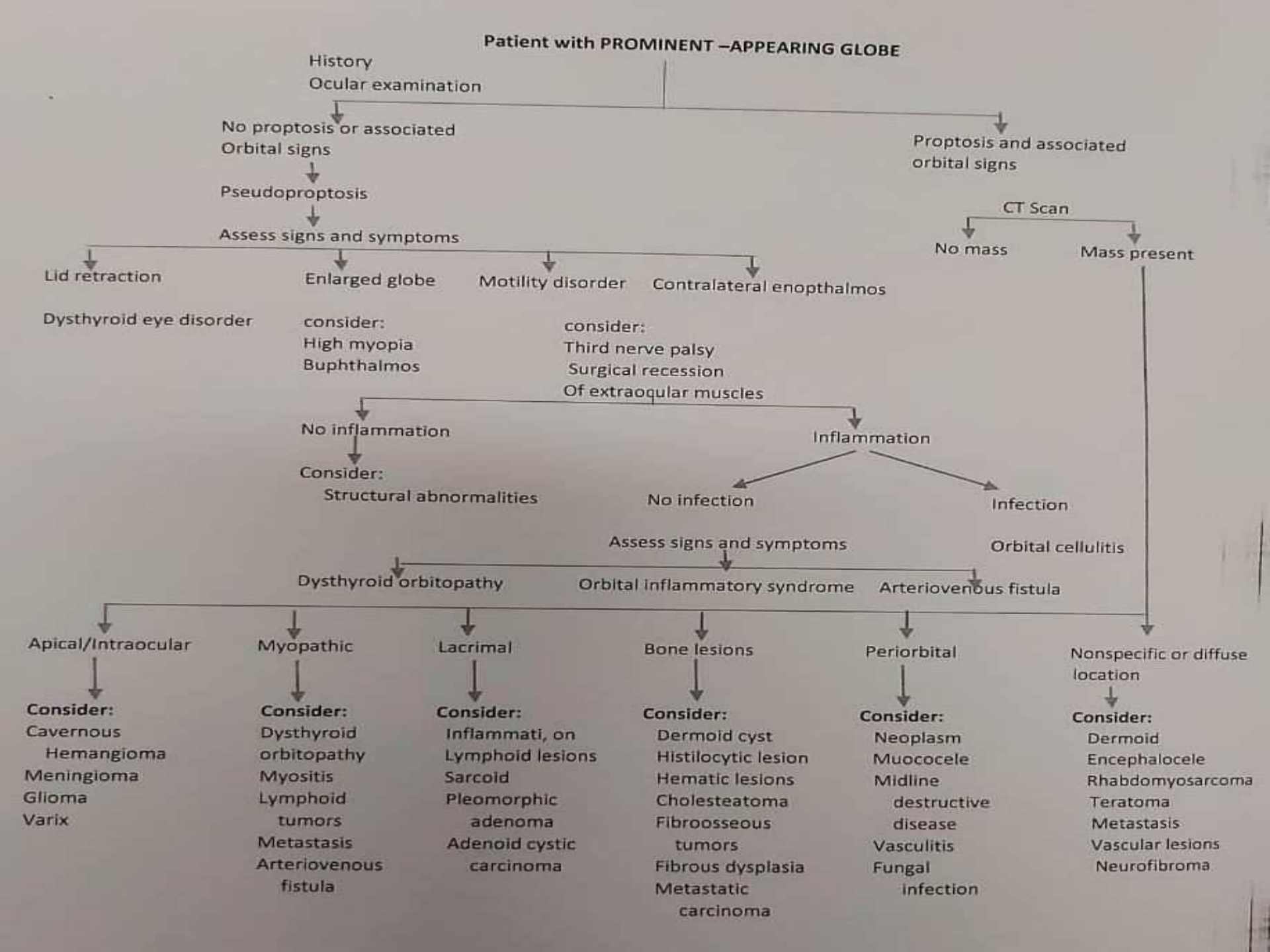

Patient with PROMINENT –APPEARING GLOBE
History
Ocular examination
No proptosis or associated Orbital signs
Pseudoproptosis
Assess signs and symptoms
Lid retraction
Dysthyroid eye disorder
Enlarged globe
consider:
High myopia
Buphthalmos
Motility disorder
Contralateral enopthalmos
consider:
Third nerve palsy
Surgical recession
Of extraoqular muscles
No inflammation
Consider:
Structural abnormalities
Inflammation
No infection
Infection
Orbital cellulitis
Assess signs and symptoms
Dysthyroid orbitopathy
Orbital inflammatory syndrome
Arteriovenous fistula
Proptosis and associated orbital signs
CT Scan
No mass
Mass present
Apical/Intraocular
Consider:
Cavernous Hemangioma
Meningioma
Glioma
Varix
Myopathic
Consider:
Dysthyroid orbitopathy
Myositis
Lymphoid tumors
Metastasis
Arteriovenous fistula
Lacrimal
Consider:
Inflammati, on
Lymphoid lesions
Sarcoid
Pleomorphic adenoma
Adenoid cystic carcinoma
Bone lesions
Consider:
Dermoid cyst
Histilocytic lesion
Hematic lesions
Cholesteatoma
Fibroosseous tumors
Fibrous dysplasia
Metastatic carcinoma
Periorbital
Consider:
Neoplasm
Muococele
Midline destructive disease
Vasculitis
Fungal infection
Nonspecific or diffuse location
Consider:
Dermoid
Encephalocele
Rhabdomyosarcoma
Teratoma
Metastasis
Vascular lesions
Neurofibroma

Clinical features of orbital disease

Symptoms:

- Eyelid and conjunctival swelling
- Redness, watering, pain
- Ocular prominence, displacement or a sunken impression of the eye
- Double vision, blurring
- Pulsating sensation or audible bruit.

a) Soft tissue involvement:

- Eyelid and periocular oedema
- Skin discoloration, ptosis
- Chemosis, which may involve plica and caruncle
- Epibulbar injection

Causes:

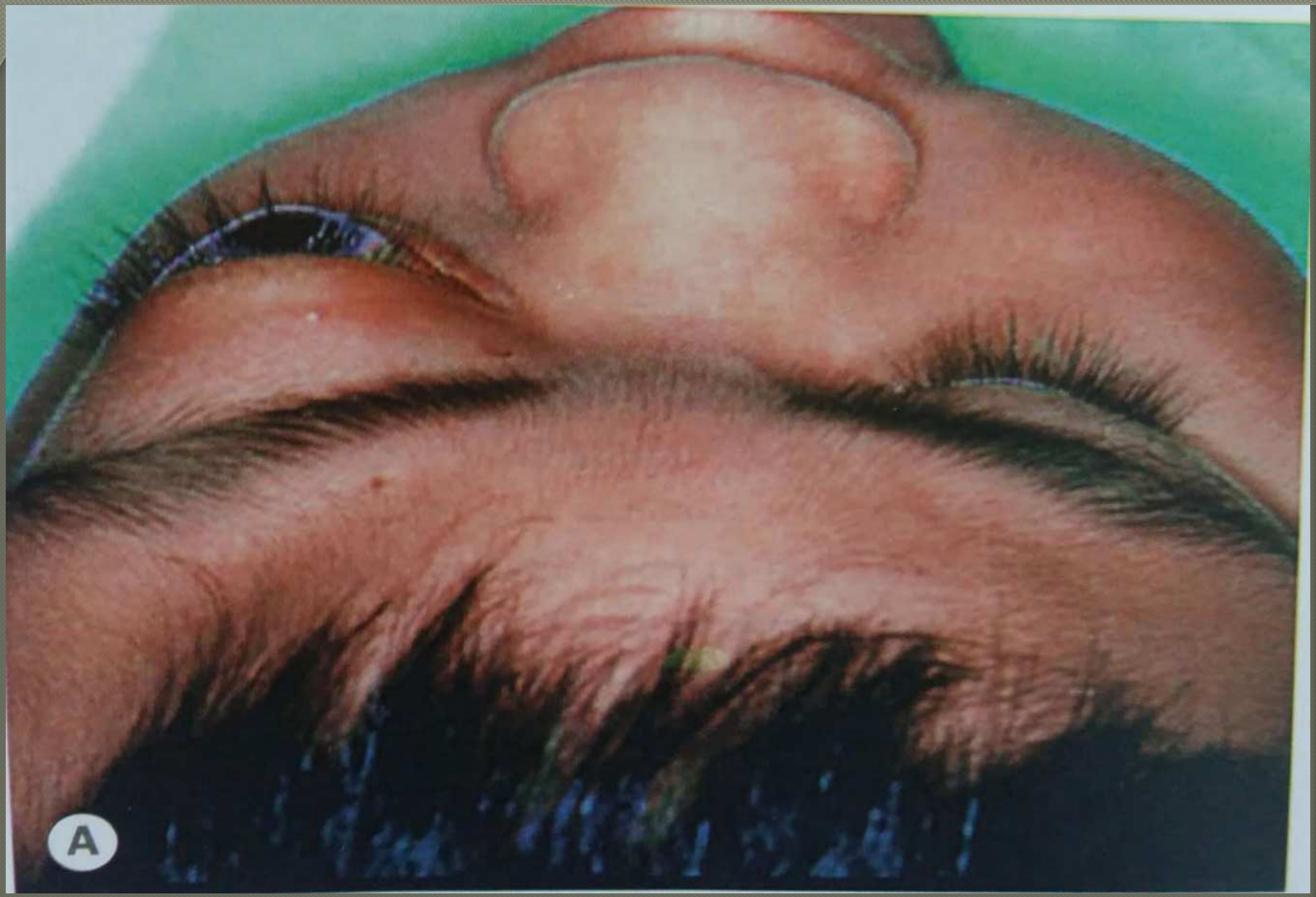
Thyroid eye disease, orbital inflammatory disease and obstruction to venous drainage.

b) Proptosis:

- Abnormal protrusion of an organ
- Caused by retrobulbar lesions or a shallow orbit.
- The intra orbital portion of the optic nerve is longer than the distance between the back of globe and optic canal.
- Allows for significant forward displacement of the globe without excessive stretching of the nerve.

Asymmetrical proptosis:

Detected by looking down at the patient from above and behind.



Left proptosis visualized from above

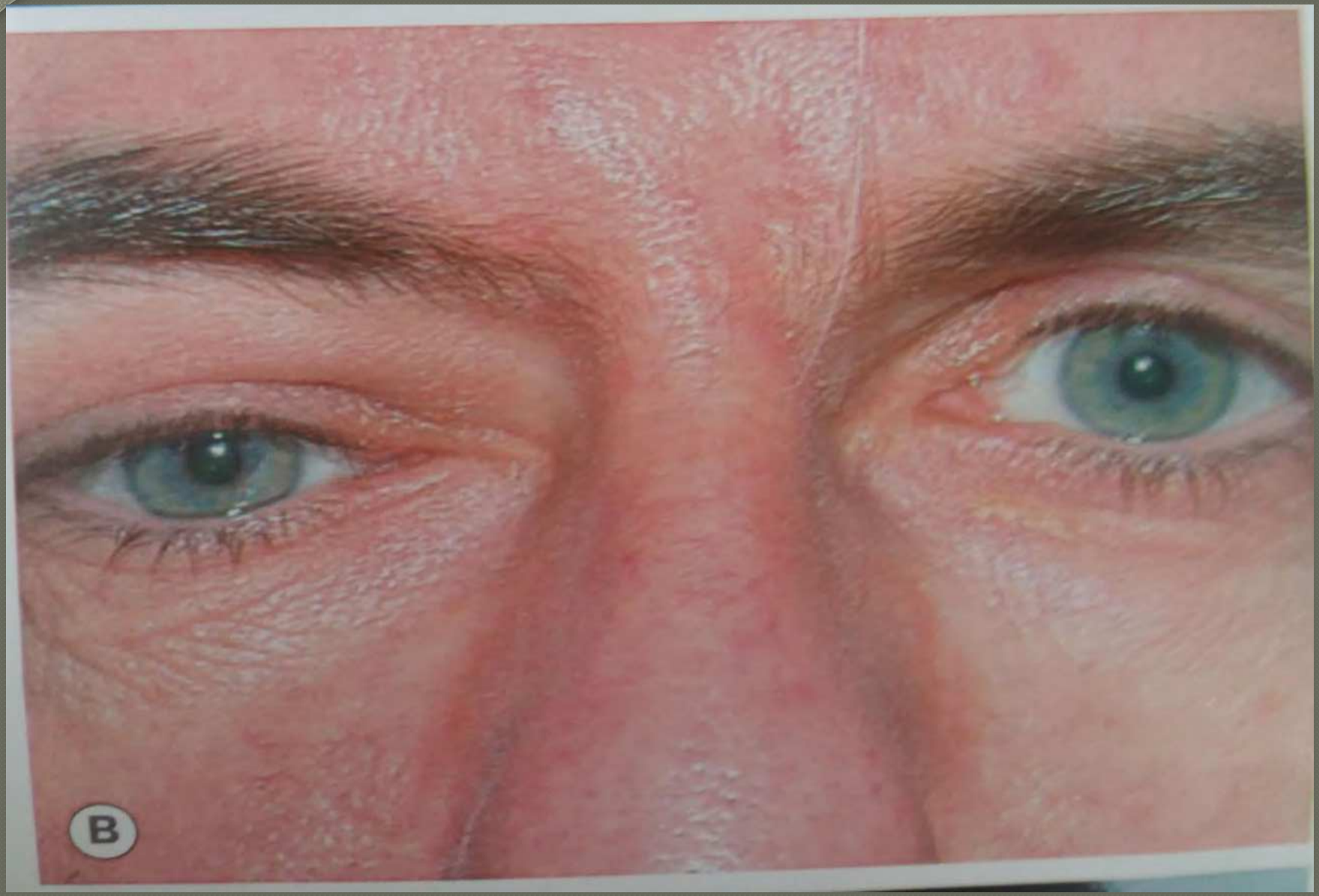
The direction of proptosis:

- Indicate the likely pathology
- e.g space occupying lesions within muscle cone such as a cavernous haemangioma or optic nerve tumours cause axial proptosis.
- External lesions give rise to combined proptosis and dystopia.

Dystopia:

Displacement of globe in coronal plane

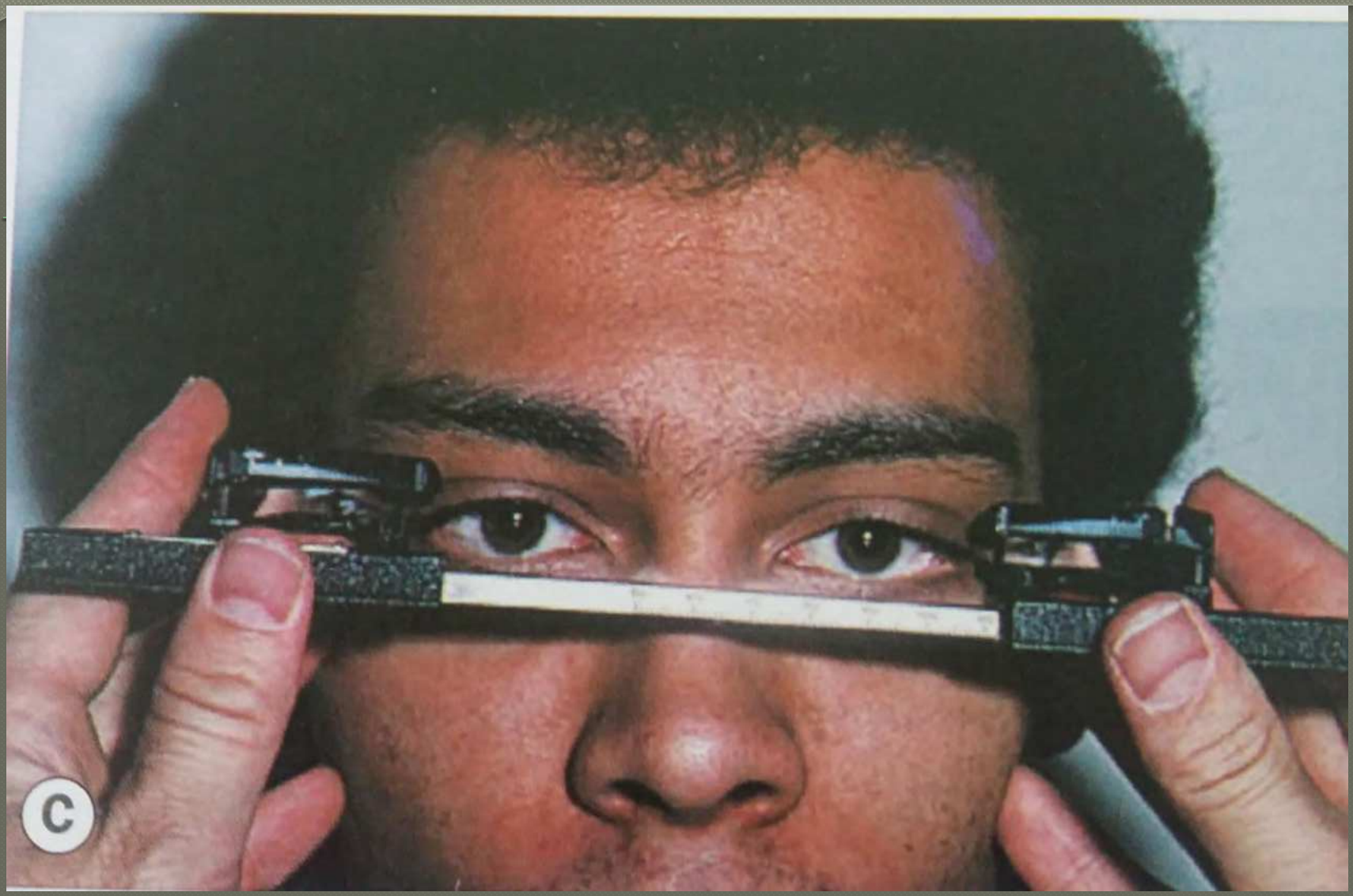
- Usually due to extraconal orbital mass such as a lacrimal gland tumour.
- Horizontal displacement is measured from the midline (nose) to centre of pupil.
- Vertical displacement is read on a vertical scale perpendicular to a horizontal rule placed over bridge of the nose.



Right inferior dystopia

Severity of proptosis:

- ✓ Measured by plastic rule resting on the lateral orbital margin.
- ✓ A binocular exophthalmometer e.g Hertel is employed for this purpose.



Measurement of proptosis with an exophthalmometer

- ✓ Reading greater than 20 mm indicative of proptosis.
- ✓ Difference of 2 – 3 mm or more between two eyes is suspicious.

Pseudoproptosis:

- ✓ False impression of proptosis
- ✓ Due to facial asymmetry
- ✓ Enlargement of globe e.g high myopia or buphthalmos
- ✓ Lid retraction
- ✓ Contralateral enophthalmos.

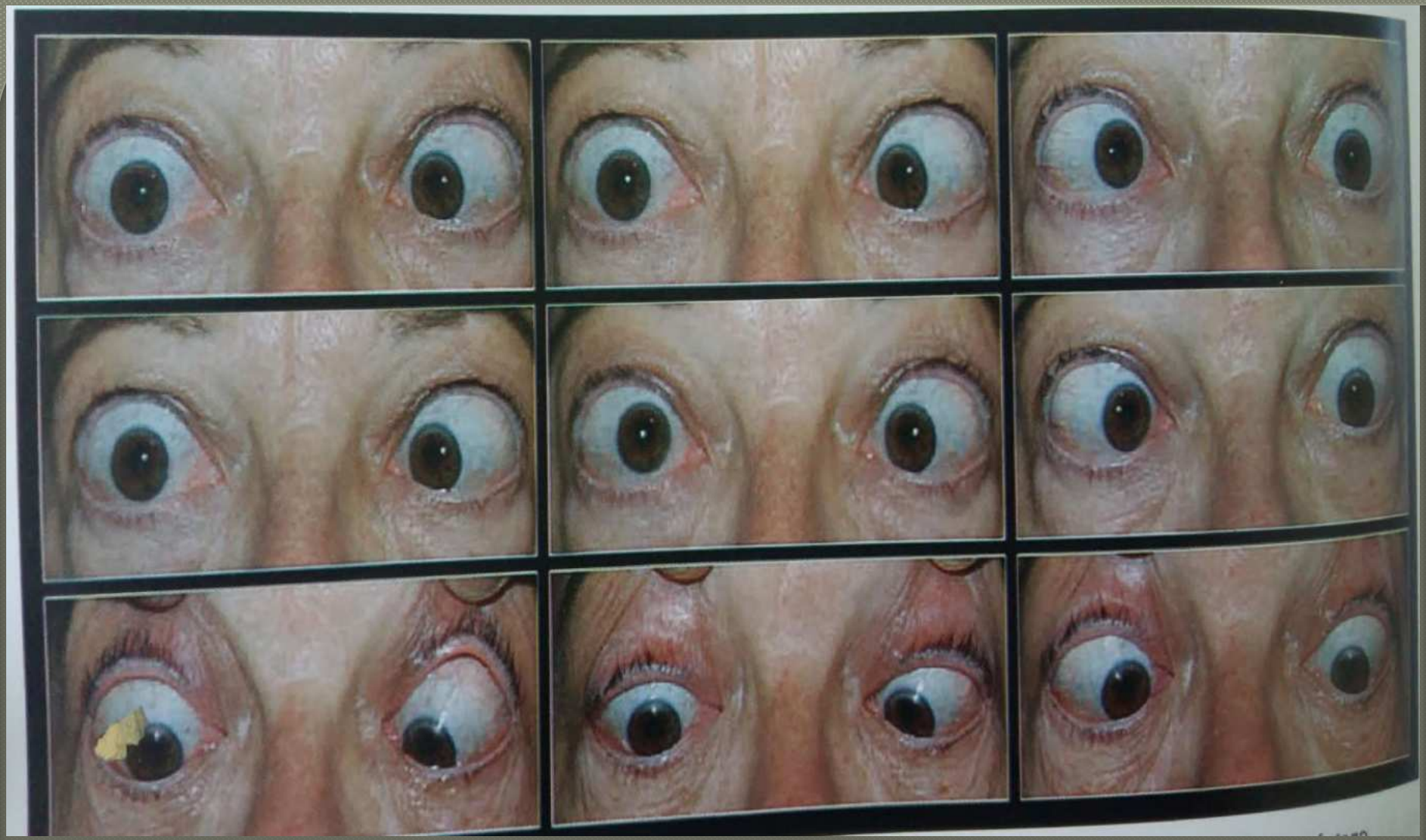
c) Enophthalmos:

- Implies recession of the globe within the orbit
- Causes include congenital/traumatic orbital wall abnormalities.
  - Atrophy of orbital contents
    e.g radiotherapy
    Scleroderma
    Sclerosis(e.g metastatic scirrhous carcinoma)

- Psuedoenophthalmos
  - Small or shrunken eye (microphthalmos phthsis bulbi)
- Ptosis
  - Contralated proptosis
  - Pseudoproptosis

d) Ophthalmoplegia:

- Defective ocular motility
- Causes – orbital mass
  Restricted myopathy (e.g TED)
  Orbital myositis
  Ocular motor nerve involvement associated with lesions in cavernous sinus, orbital fissures or posterior orbit (e.g CCF, THS)



Restrictive myopathy and bilateral lid retraction and proptosis in thyroid eye disease – nine positions of gaze

- Tests to differentiate a restrictive from neurological motility defect:

i) *Forced duction test (FDT):*

- Under topical anaesthesia, insertion of muscle in an involved eye is grasped with forceps.

- Globe is rotated in the direction of reduced mobility
- Checked movement of globe – indicate restrictive problem
- No resistance encountered with a neurological lesion.

ii) Differential IOP test:

- Involves less discomfort than FDT.
- An objective rather than subjective end point.
- IOP measured in primary position of gaze and then with patient attempting to look in direction of limited mobility.
- An increase of 6 mm or more denotes resistance transmitted to the globe by muscle restriction.

iii) Saccadic eye movements:

- In neurological lesions are reduced in velocity.
- In restrictive defects manifest normal saccadic velocity with sudden halting of ocular movements.

e) Dynamic properties:

Increasing venous pressure

By dependent head position – the valsalva manoeuvre or jugular compression may induce or exacerbate proptosis in patients with orbital venous anomalies or infants with orbital capillary haemangioma.

Pulsation:

Caused either by

- Arteriovenous communication or
- Defect in orbital roof

In arteriovenous communication

- Pulsation associated with a bruit depending on size of communication

In orbital roof defect

- Pulsation transmitted from brain by CSF. There is no associated bruit.

A bruit:

Sign found with a larger carotid cavernous fistula.

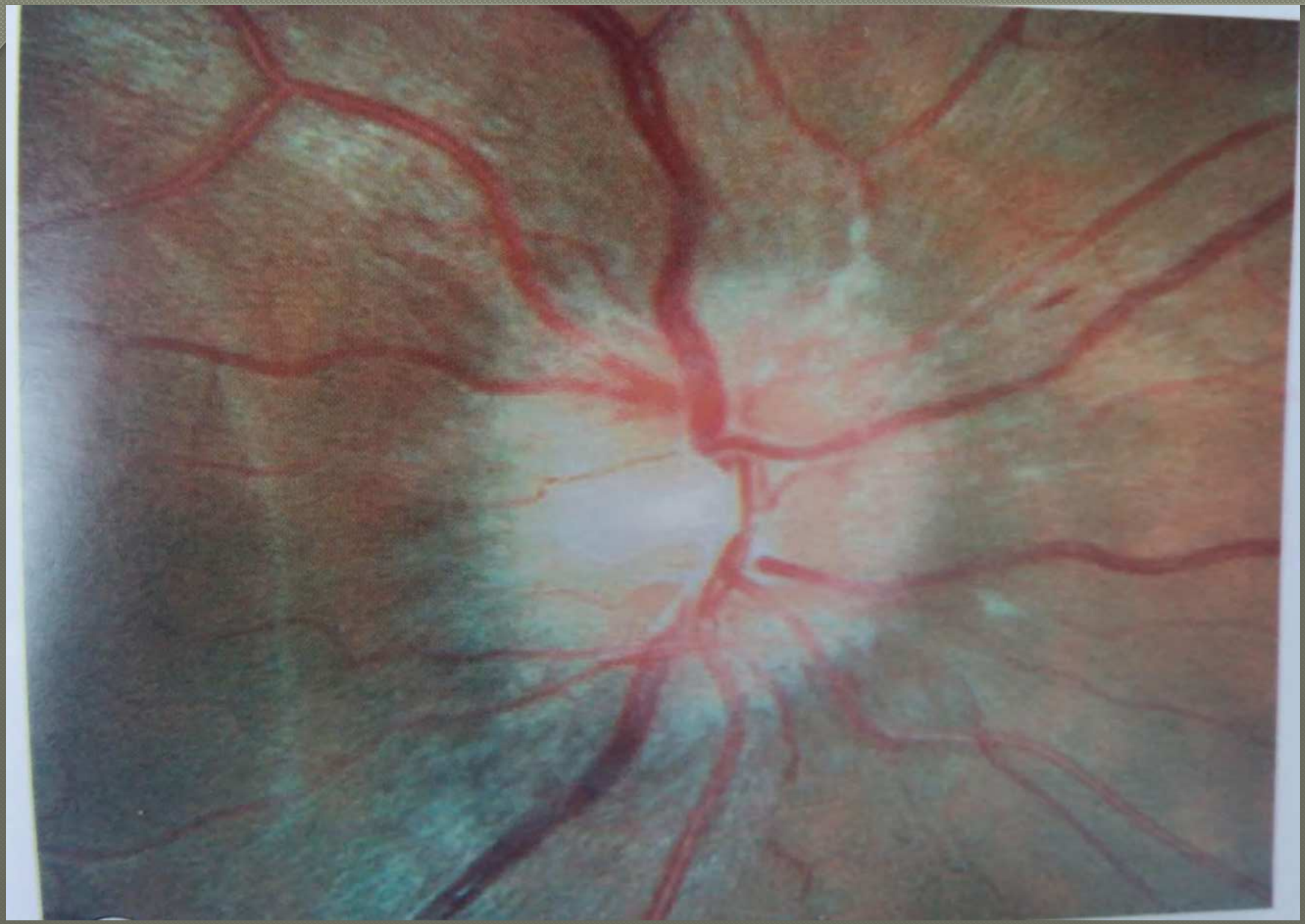
Best heard with bell of stethoscope.

Bruit Lessened or abolished by gently compressing ipsilateral carotid artery in neck.

f) **Fundus changes:**

Optic disc swelling

Initial feature of compressive optic neuropathy.

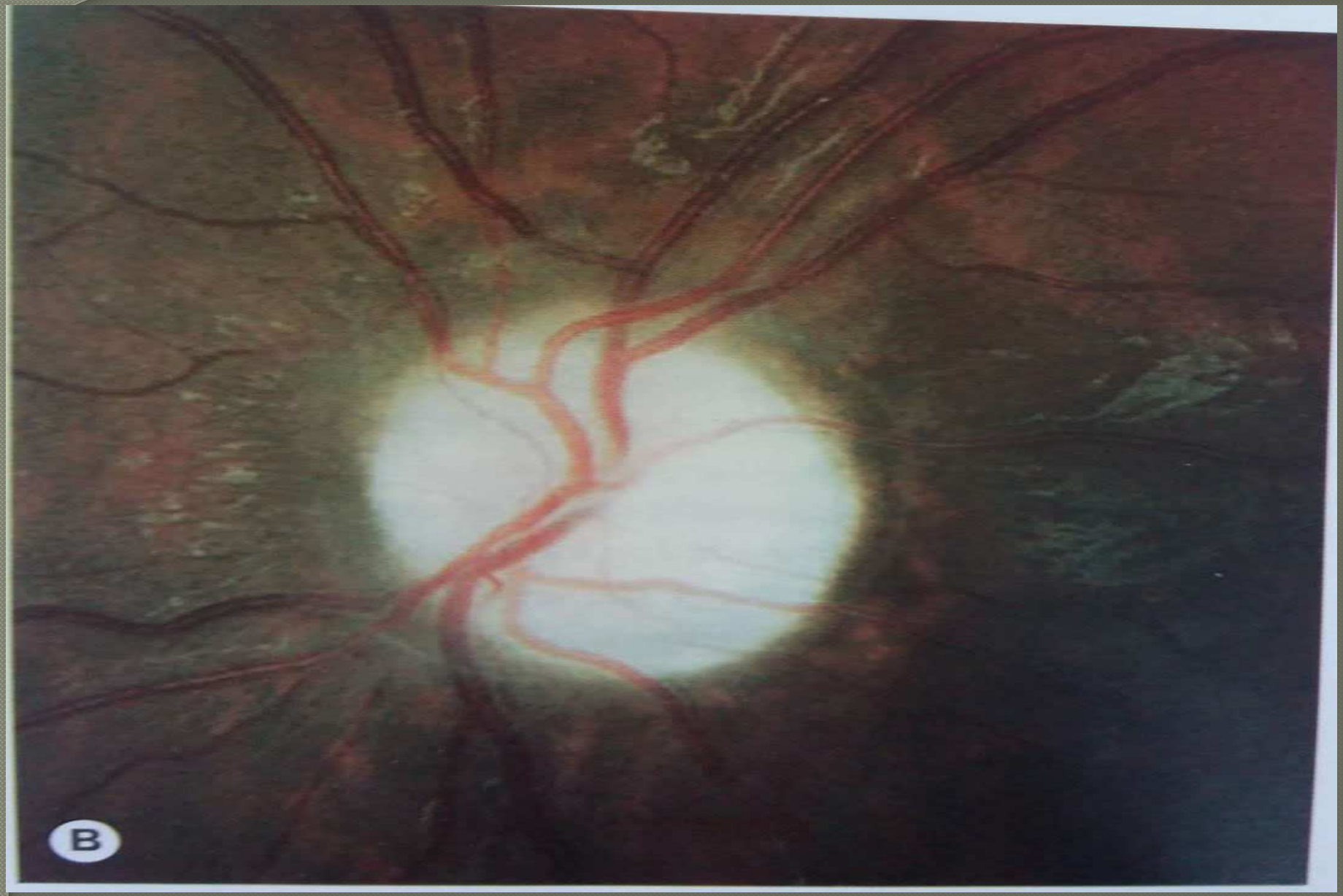


Disc swelling

Optic atrophy

Preceded by swelling

Feature of severe compressive optic neuropathy, TED and optic nerve tumours.



Optic atrophy

Opticociliary collaterals

Consist of enlarged pre-existing peripapillary capillaries.

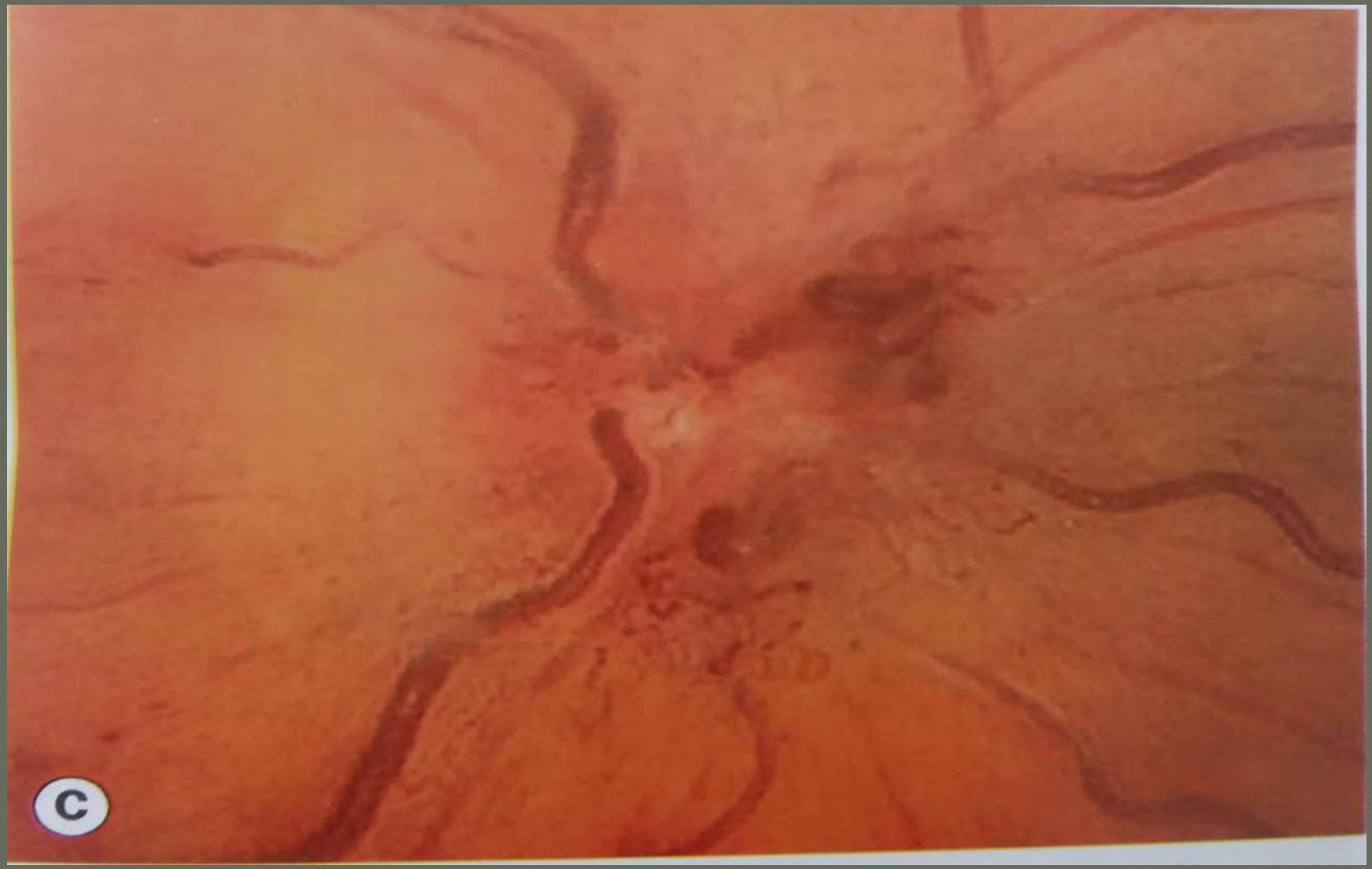
Divert blood from central retinal venous circulation to peripapillary choroidal circulation when there is obstruction of normal drainage channels.

On Ophthalmoscopy:

Vessels appear as large tortuous channels

Most frequent location temporally.

Disappear at disc margin.



Opticociliary vessels on a chronically swollen disc

Collaterals:

Associated with any orbital or optic nerve tumour.

Compresses intra orbital optic nerve and impairs blood flow through central - retinal vein.

Most common tumour associated with shunts is an optic nerve sheath meningioma.

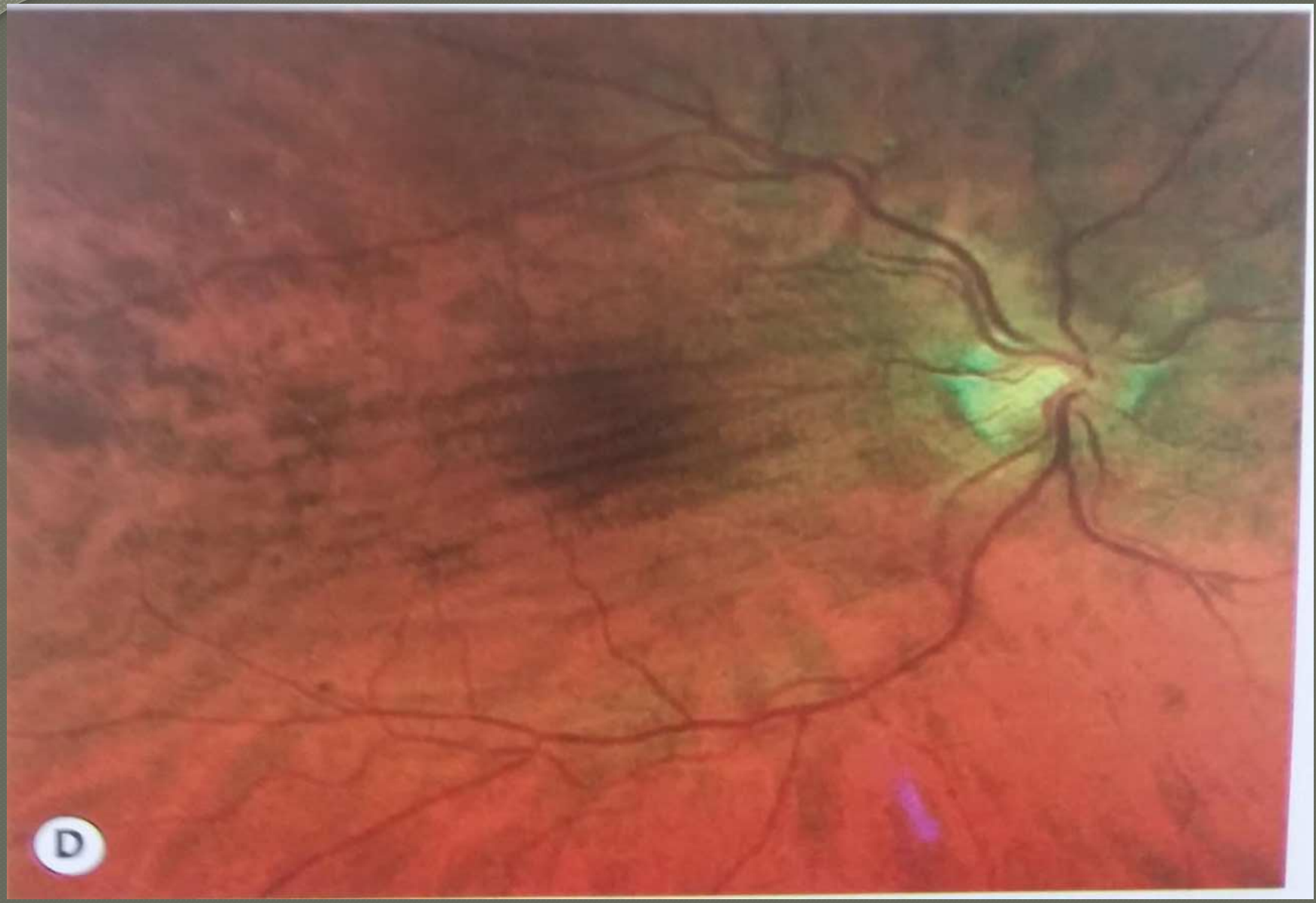
Other causes – optic nerve glioma
CRVO
Idiopathic intracranial - hypertension
Glaucoma

Choroidal folds

Are parallel grooves or striae involving inner choroid, Bruch membrane, RPE and retina.

Causes include:

- Idiopathic
- Papilloedemia
- Orbital disease e.g retrobulbar tumours and thyroid ophthalmopathy.
- Ocular disease such as choriodal tumours, inflammation such as posterior scleritis and hypotony.



Choroidal folds

Investigations

1) Computed tomography (CT):

- Useful for depicting bony structures and location and size of space occupying lesions.
- Particular value in patients with orbital trauma.
- It can detect small fractures, foreign bodies, blood, herniation of extraocular muscle and emphysema

2) Magnetic resonance imaging (MRI):

- Demonstrate orbital apex lesions and intracranial extension of orbital tumours.
- Useful for imaging orbital inflammatory disease.

3)Plain X – rays:

- Little useful
- Rarely used for the initial diagnosis of traumatic bony injury.

4) Ultrasonography:

- Provide useful information
- Helpful for posterior segment and orbital lesions.
- High grade apparatus and operator is required.

5) Fine needle biopsy:

- Sometimes performed
- Helpful in suspected neoplastic disease.
- Complications include haemorrhage and ocular penetration.

Management

- Depends on underlying cause

Medical

- Artificial tears
- Antibiotics for oribtal cellulitis
- Medical treatments for underlying conditions such as medications for hyper thyroidism
- IV medication teprotumumab for thyroid eye disease

Other non surgical treatments

- Double vision treatments
- Immuno suppressive drugs
- Corticosteroids

Surgery (orbitotomy or biopsy)

Indicated for

- Remove a tumor
- Create more space behind the eye in the eye socket
- To perfect cornea

Prevention

- Keeping thyroid levels in check
- Quitting smoking

Thanks

PROPTOSIS IN CHILDREN

Prof SofiaIqbal
FRCS, MRCOphth
Fellowship Orbit/Oculoplastics
Fellowship Refractive surgery

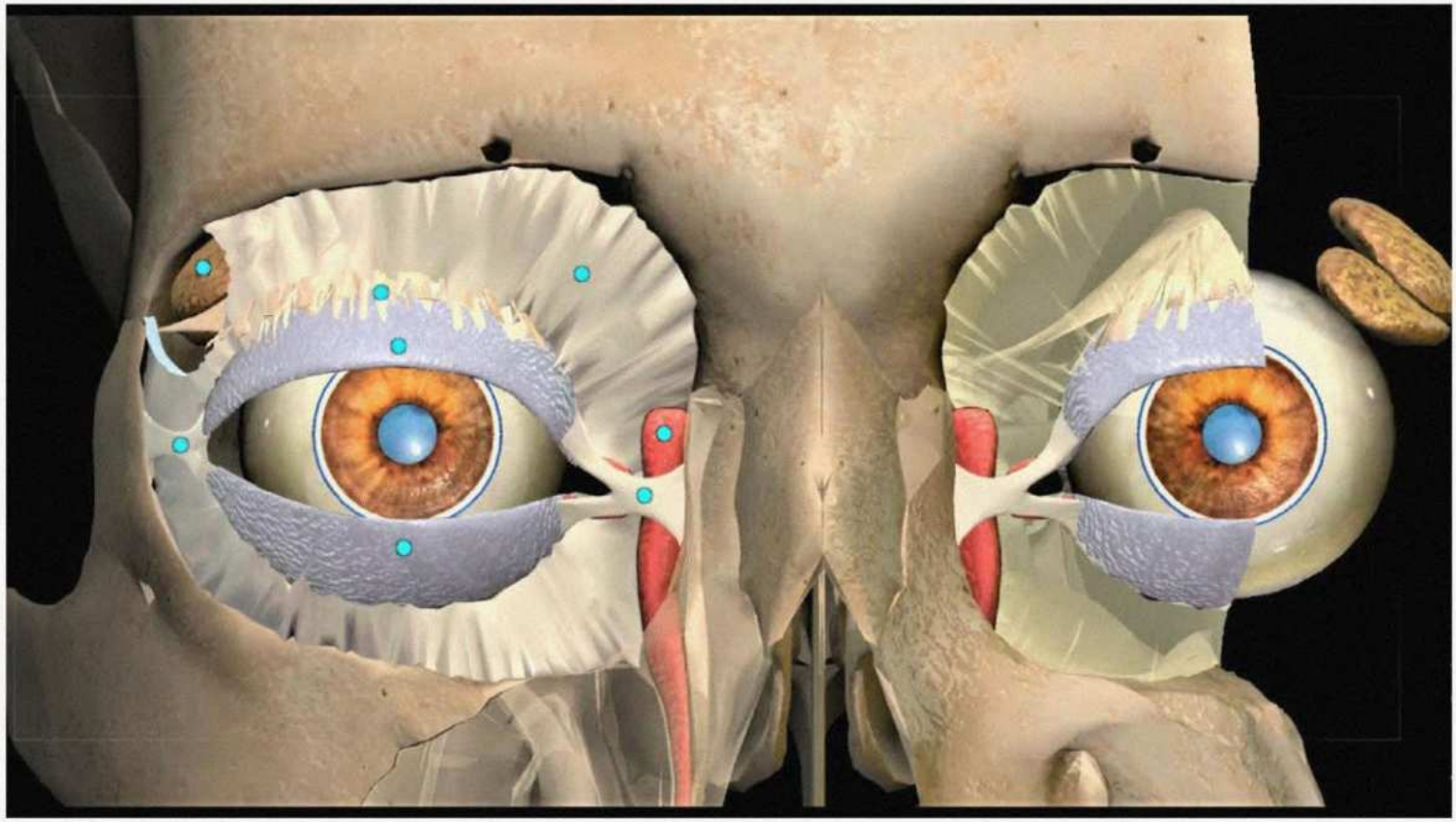
CAUSES

- Orbital cellulitis
- Dermoid cyst
- Capillary hemangioma
- Optic nerve glioma
- Rhabdomyosarcoma
- Retinoblastoma
- Leukemia
- Lymphangioma
- Metastasis
  - Metastatic neuroblastoma
  - Ewing's sarcoma

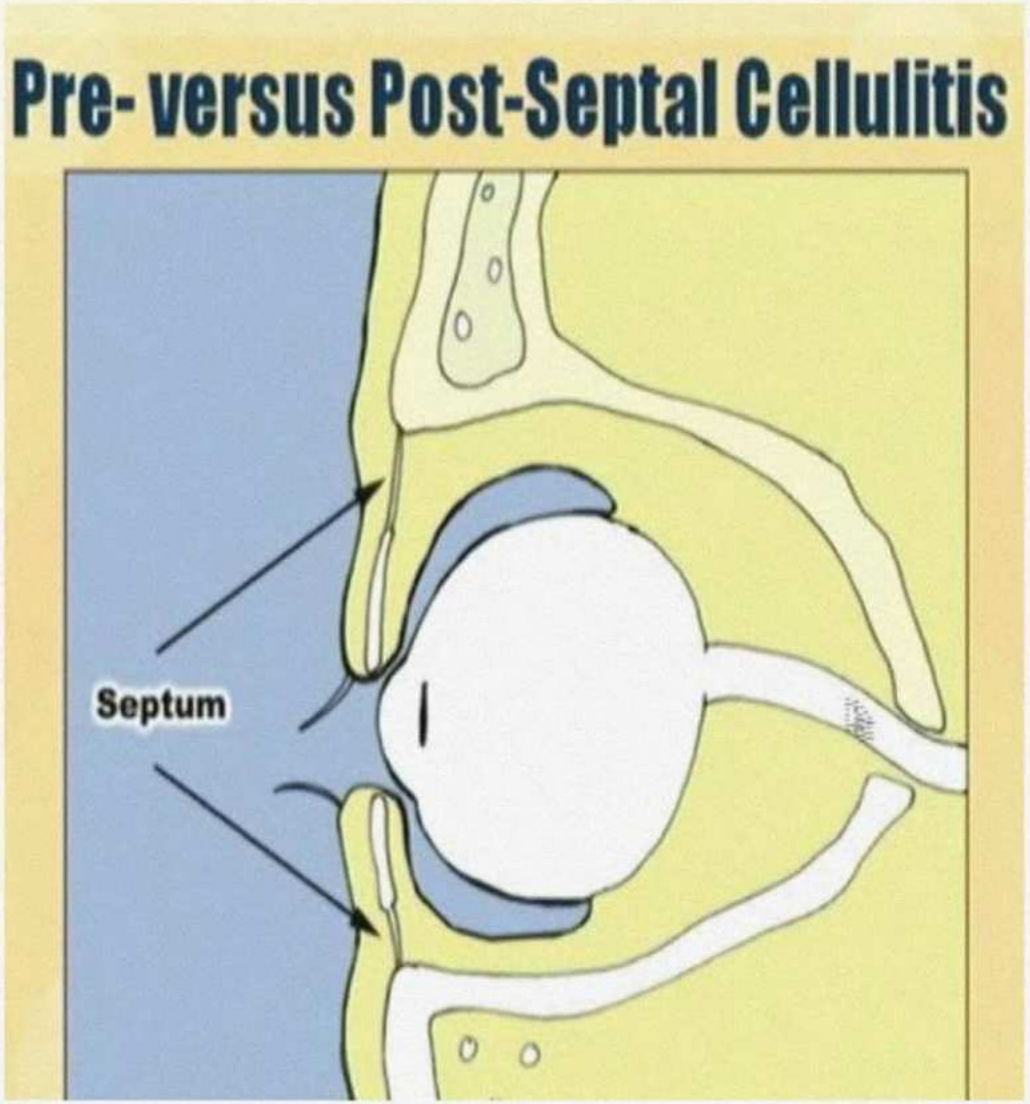
BIRTH-4YEARS	4-10 YEARS	OVER 10 YEARS
Orbital cellulitis	Lyphangioma	Orbital cellulitis
Dermoid cyst	Rhabdomyosarcoma	NSOI
Capillary haemangioma	Leukemia	Thyroid ophthalmopathy
Lymphangioma	Optic nerve glioma	Dermoid cyst
Structural abnormality	Langerhan cell histiocytosis	Leukemia
Metastatic (neuroblastoma)	NSOI	Fibrous dysplasia
Rhabdomyosarcoma	Orbital cellulitis	
Langerhan cell histiocytosis		
Teratoma		
Optic nerve glioma		

Orbital Septum

- *The orbit is separated from the soft tissue of the eyelid by the orbital septum*
- *A facial plane that is continuous with the periosteum of the facial bones*
- *It inserts into the tarsal plate of the upper and lower eyelids*
- *It is a barrier which prevents the spread of infection from the eyelids posteriorly to the orbit*

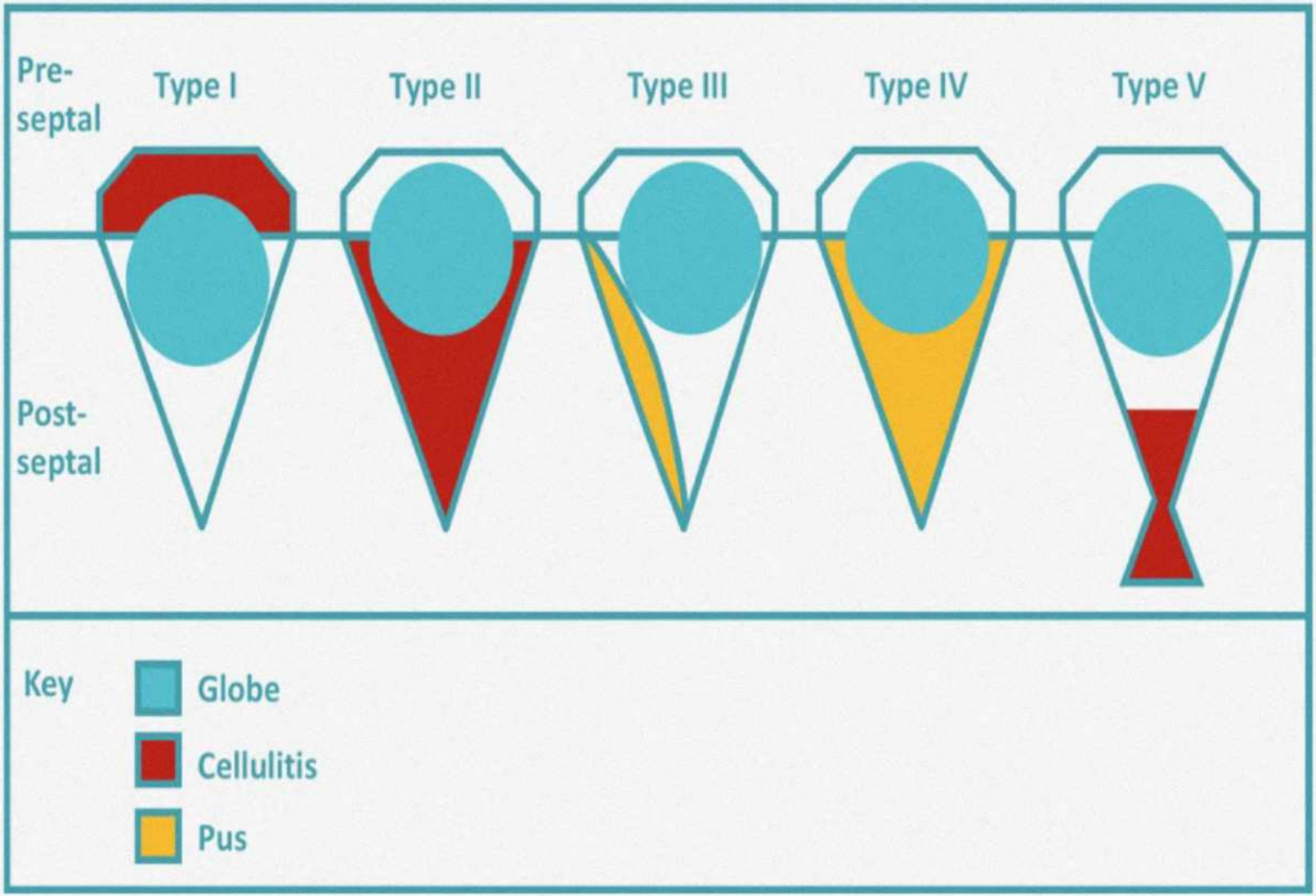


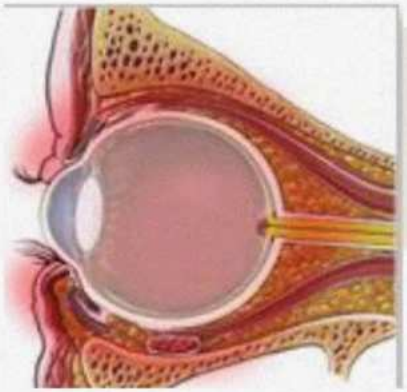
Pre- versus Post-Septal Cellulitis



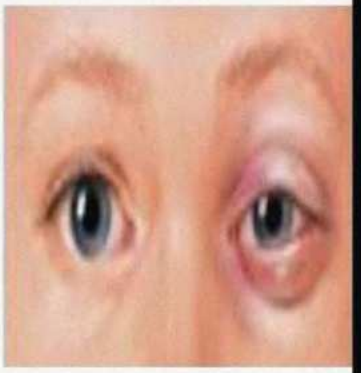
CHANDLER CLASSIFICATION

GROUP 1	Preseptal cellulitis
GROUP 2	Orbital cellulitis
GROUP 3	Sub periosteal abscess
GROUP 4	Orbital abscess
GROUP 5	Cavernous sinus thrombosis





PRE-SEPTAL CELLULITIS

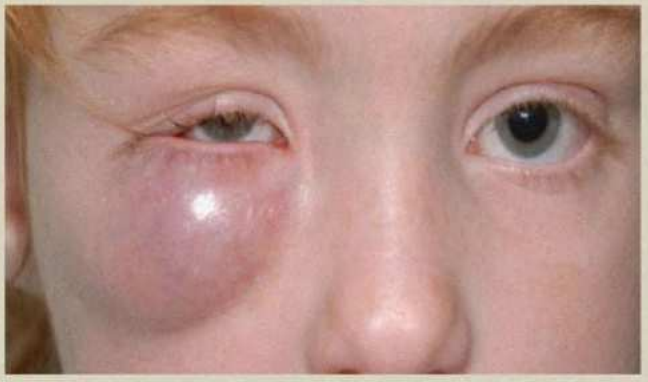


- Infective /inflammatory process anterior to the orbital septum
- Eyelid is swollen and can be non tender
- The VA and EOM are normal and there is no chemosis

ETIOLOGY

- Bacteria (staph aureus,strep pneumo,anaerobes)
- Local spread from an adjacent sinusitis or dacryocystitis, from an external ocular infection, or following trauma to the eyelid

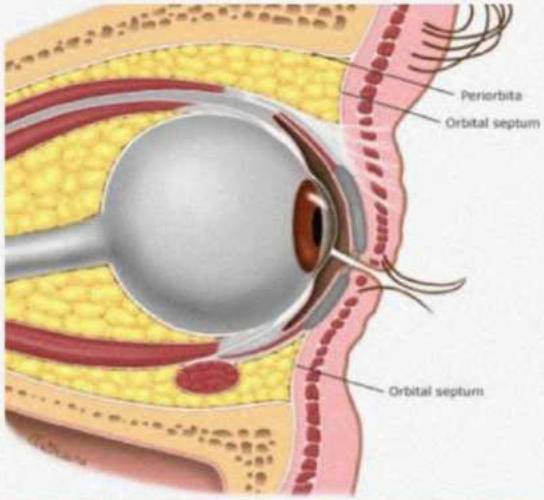
Management



- **Antibiotics**

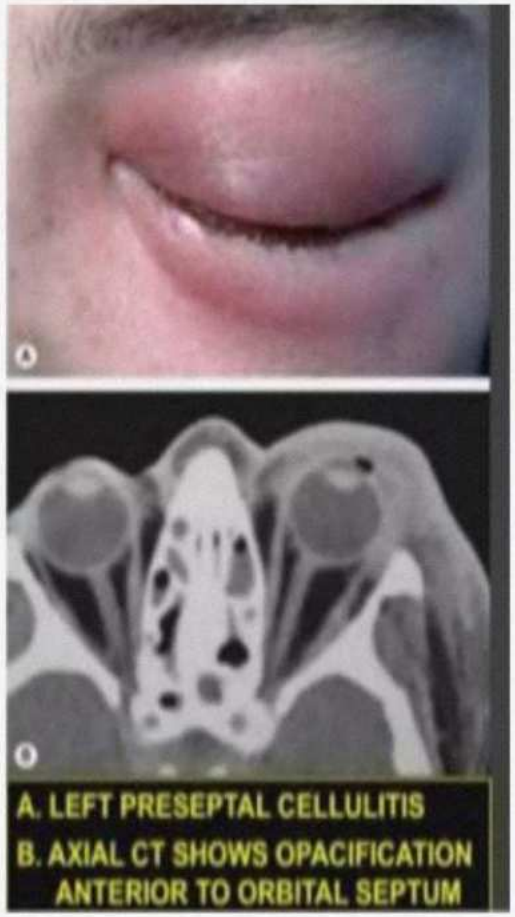
  Amoxicillinclavulanate,Cefuroxime,Gatifloxacin,Moxifloxacin, Levofloxacin
- **Pain killers**
- **Supportive therapy**

Infection can spread as Orbital cellulitis which is vision and life threatening

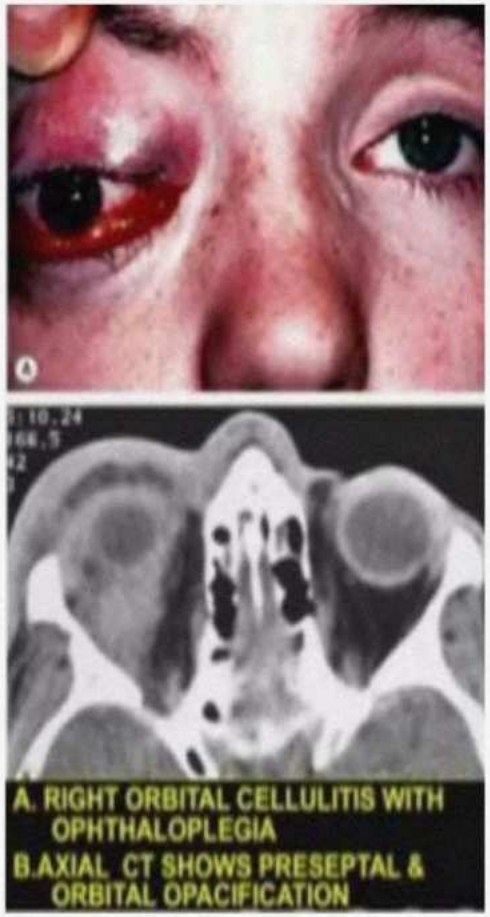


Preseptal Cellulitis	Orbital Cellulitis
✅ Unlikely to have complications	❌ Can progress to loss of vision, brain abscess
Due to external source	Associated with paranasal sinusitis
Staph aureus Strep pneumo + other Strep Anaerobes	Same micro as preseptal cellulitis plus fungal, mycobacterial

Clinical Feature	Preseptal Cellulitis	Orbital Cellulitis
Eyelid swelling with or without erythema	Yes	Yes
Eye pain, tenderness	+/-	Yes, can have deep eye pain
Pain with EOM	No	Yes
Proptosis	No	Usually, can be subtle
Ophthalmoplegia, diplopia	No	May be present
Vision impairment	No	May be present
Chemosis	Rare	May be present
Fever	May be present	Usually
Leukocytosis	May be present	May be present



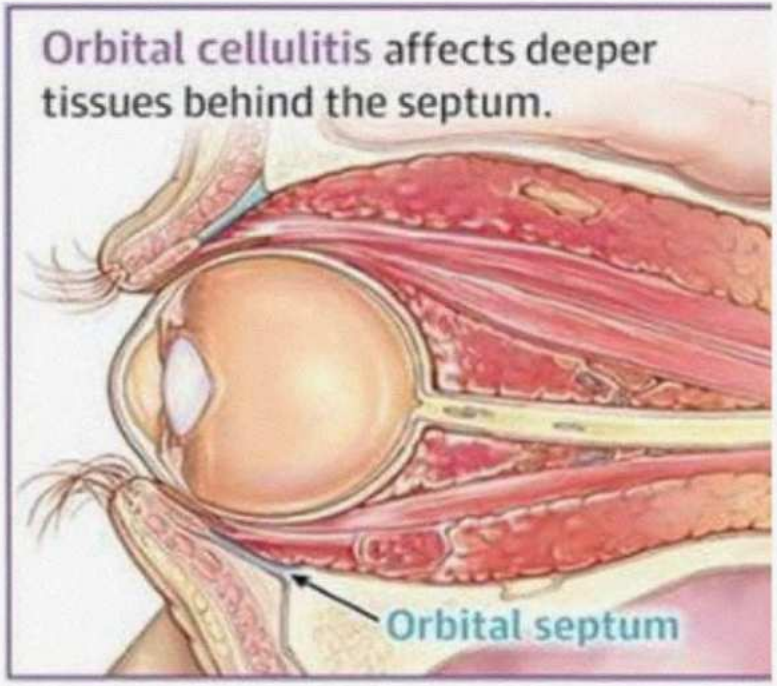
A. LEFT PRESEPTAL CELLULITIS
B. AXIAL CT SHOWS OPACIFICATION ANTERIOR TO ORBITAL SEPTUM



A. RIGHT ORBITAL CELLULITIS WITH OPHTHALOPLEGIA
B.AXIAL CT SHOWS PRESEPTAL & ORBITAL OPACIFICATION

ORBITAL CELLULITIS

- Infection of the eye tissue behind the orbital septum



CAUSES OF ORBITAL CELLULITIS

Extension from periorbital structures

- Paranasal sinuses (sinusitis)
- Face and eyelids, infection of
- Lacrimal sac (dacryocystitis)
- Dental (odontogenic infection)

Exogenous causes

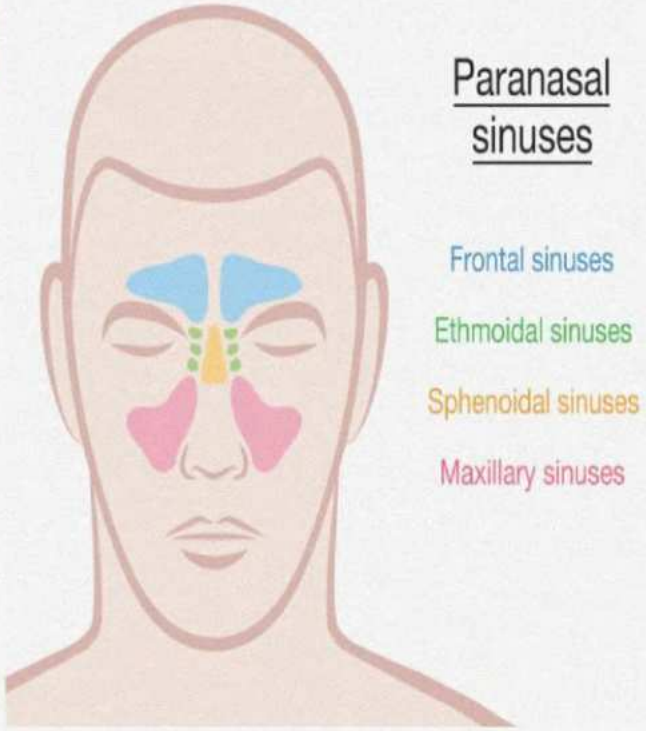
- Trauma (rule out foreign bodies)
- Surgery (after any orbital or periorbital surgery)

Endogenous causes

- Bacteremia with septic embolization

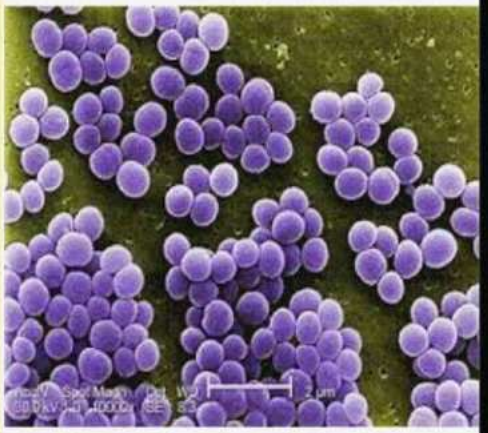
Intraorbital causes

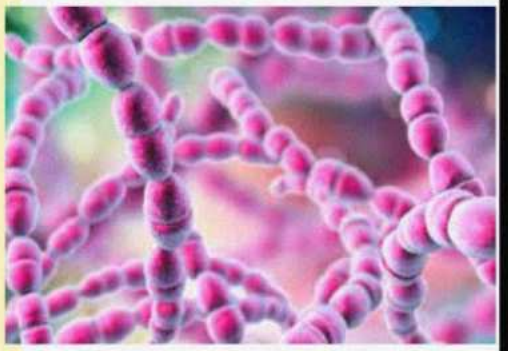
- Endophthalmitis
- Dacryoadenitis

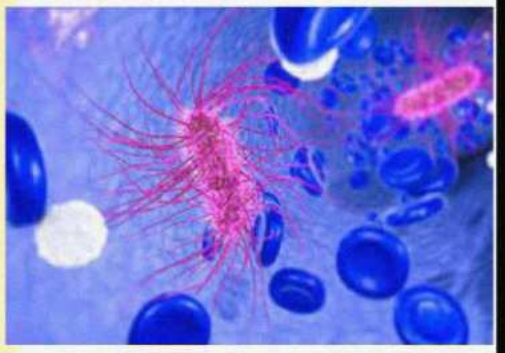


Bacterial isolates from orbital cellulitis (abscesses), ocular surface or blood

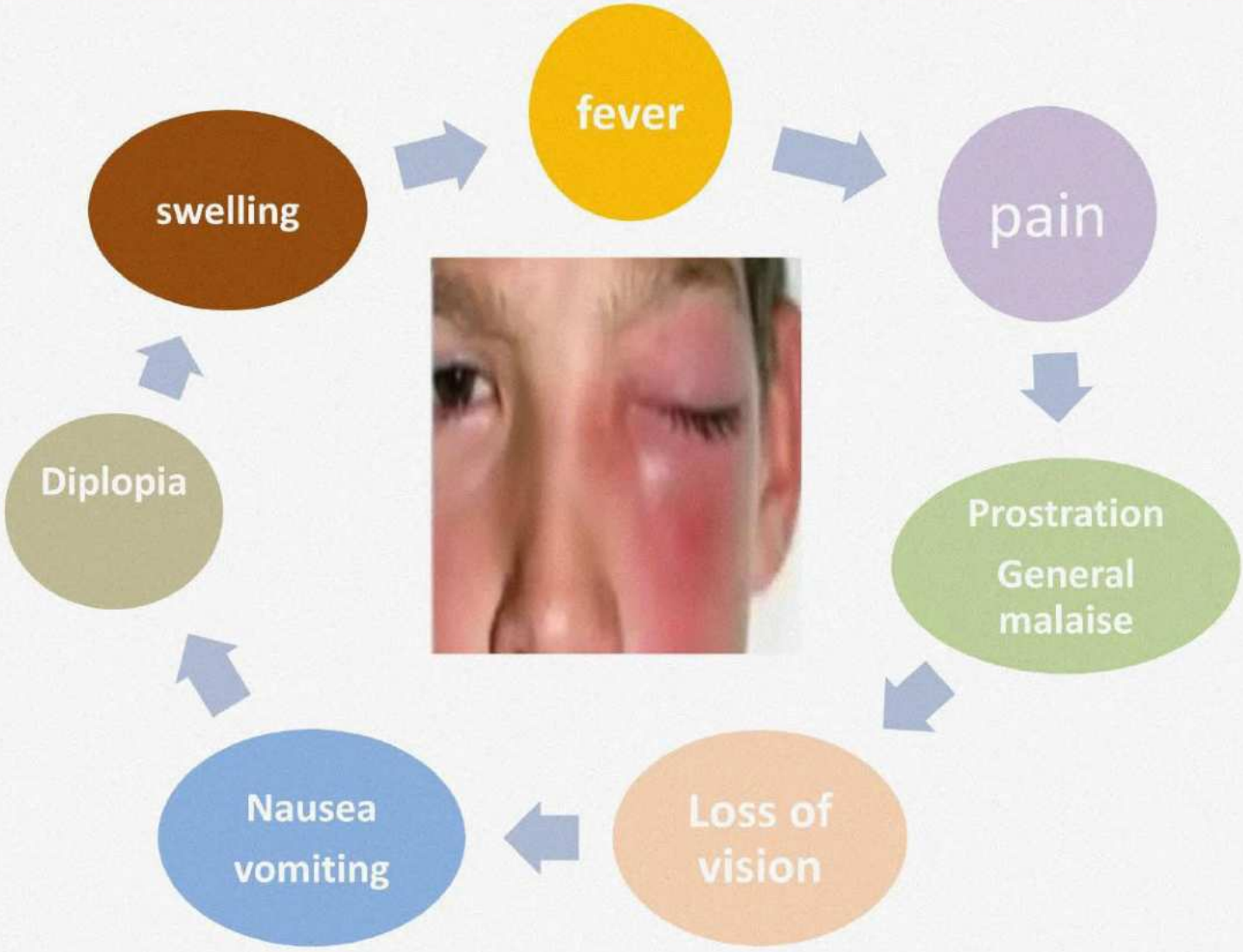
1. *Staphlococcus aureus*, *Staphlococcus epidermidis*
2. *Streptococci pneumoniae*, *Streptococci pyogenes*, *Streptococci sangunis*, *Streptococci fecalis*, *Streptococci mitis*
3. Diphtheroids
4. *Haemophilus influenza*
5. *Escherchia coli*
6. *Moraxella catarrhalis*
7. *Neiserria* sp
8. *Bacillus thuringiensis*
9. *Arcanobacterium haemolyticum*
10. *Pseudomonas aeroginosa*
11. *Pasturella multocida*
12. Atypical Mycobacteria and *Mycobaterium tuberculosis*
13. *Fusobacterium necrophorum*

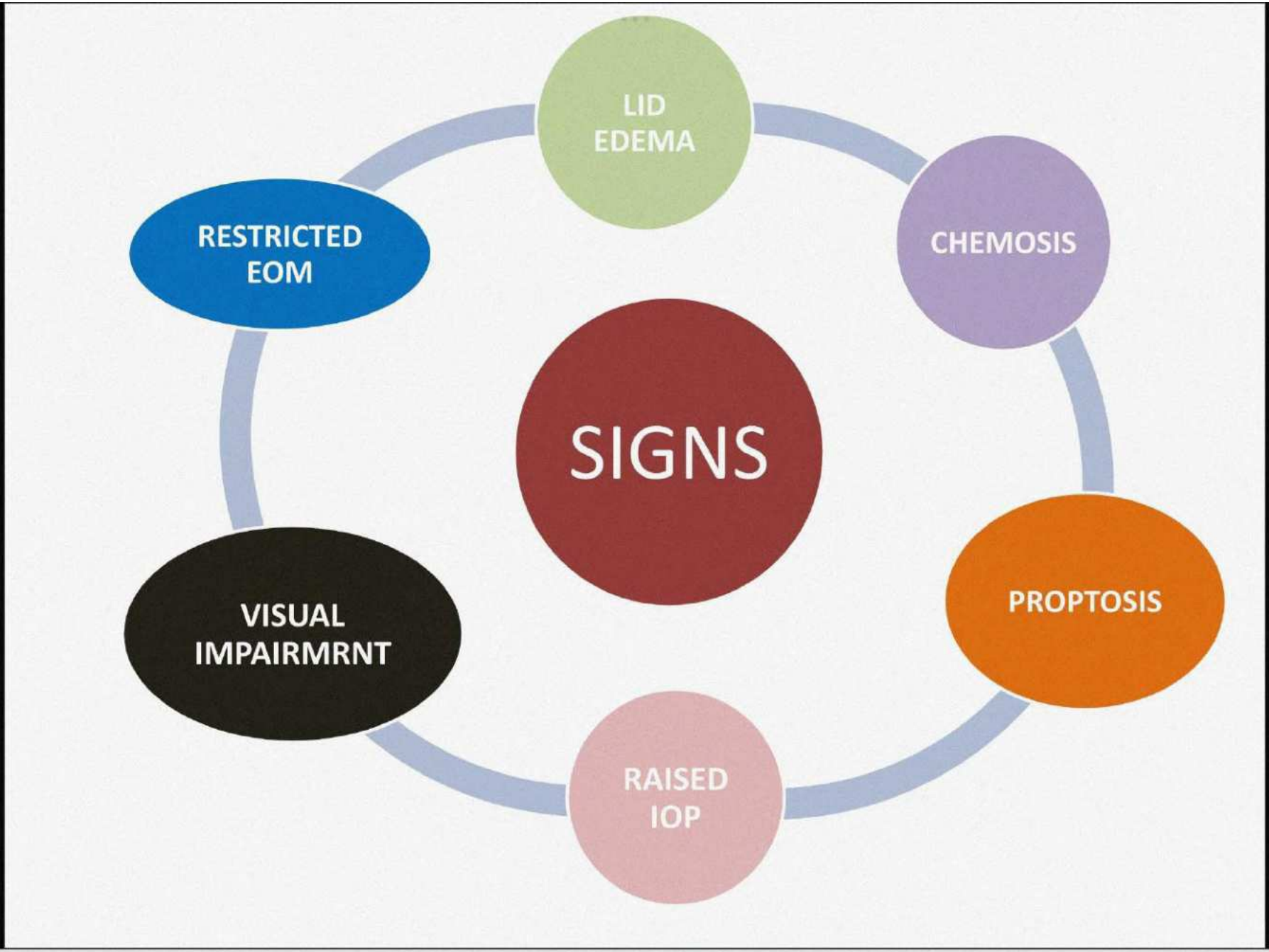






SYMPTOMS





Differential diagnosis of orbital cellulitis in children

(A) Idiopathic nonsupurative inflammation of the orbit
  (i) Nonspecific orbital inflammatory disease
  (ii) Inflammatory thyroid eye disease (rarely seen in children)
  (iii) Wegners granulomatosis
  (iv) Sarcoid related inflammatory disease

(B) Benign and neoplastic disease
  (i) retinoblastoma and its treatment
  (ii) Lymphoma
  (iii) Lymphangioma
  (iv) Eosinophilic granuloma (histiocytosis)
  (v) Rhabdomyosarcoma
  (vi) Leukaemic deposits
  (vii) Dermoid cyst

(C) Systemic disease
  (i) Kawasaki disease
  (ii) Sickle cell anaemia

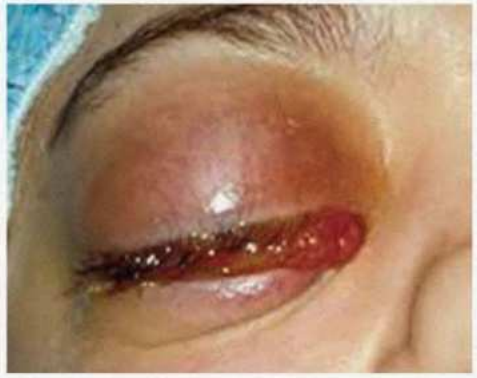
PROPTOSIS

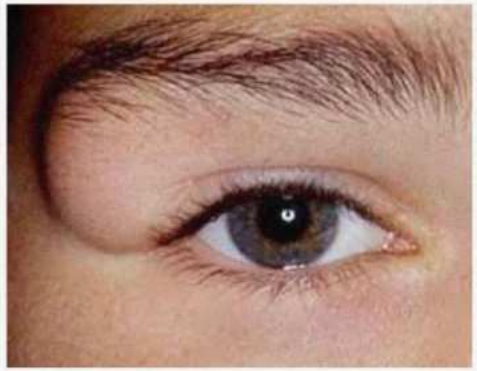
Lid edema
Sinus disease
Restricted motility
Pain/tender
Lid trauma
fever

ORBITAL CELLULITIS

Little or no inflammation
Orbital lesion
Young child/adult
Slow growing
Located near bone sutures

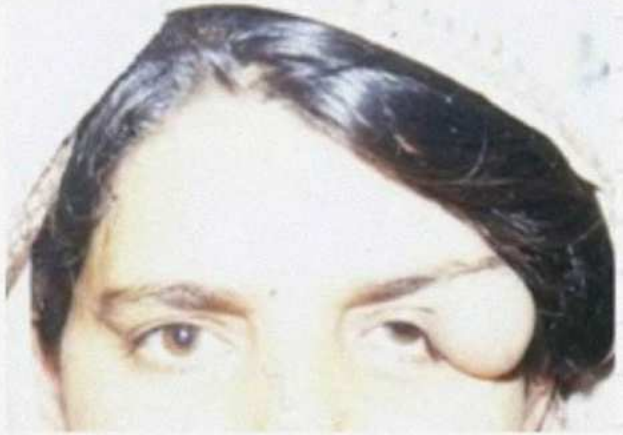
DERMOID CYST

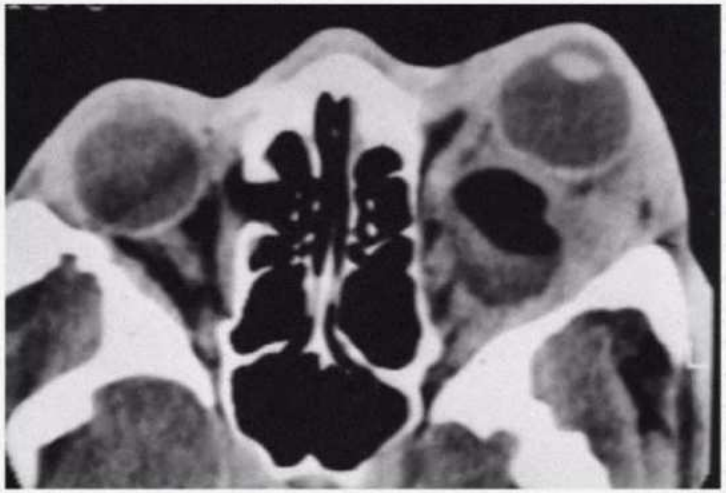




Dermoid Cyst

- A choristoma found adjacent to suture lines
- Slow growing,nontender mass usually superotemporal mostly in children
- Consist of keratinized stratified squamous epithelium, blood vessels,collagen,sebaceous and sweat glands and hair follicles
- Inflammation if ruptures
- Excision should be in toto





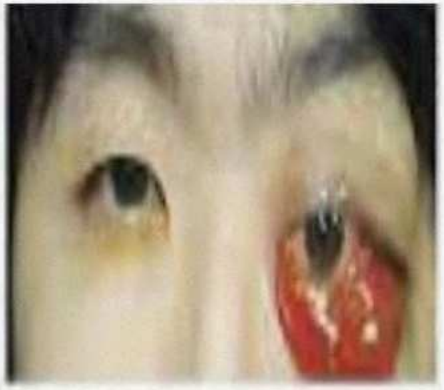
PROPTOSIS

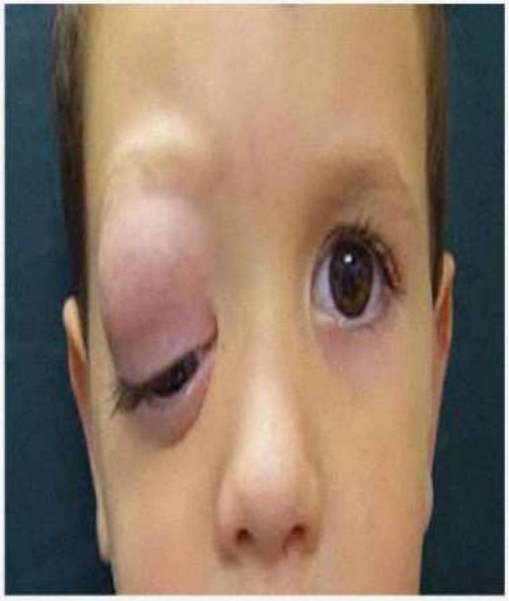
Age ≥5 years
Painful
Upper nasal quadrant mass
Few episodes of proptosis
MRI shows fluid levels

LYMHANGIOMA

Age 7-8 years
Unilateral
Hx of nose bleeds
Bluish color lids
Painful
Lymph node involvement

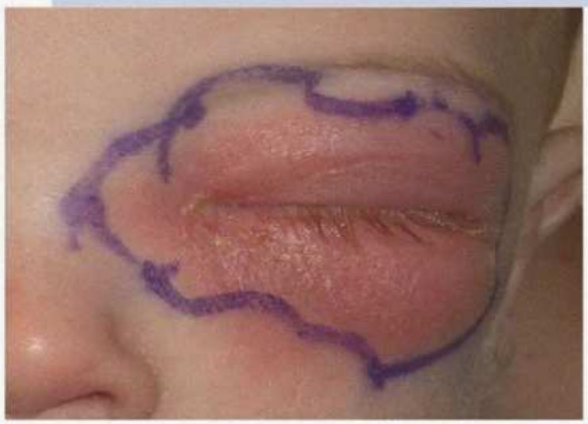
RHABDOMYOSARCOMA

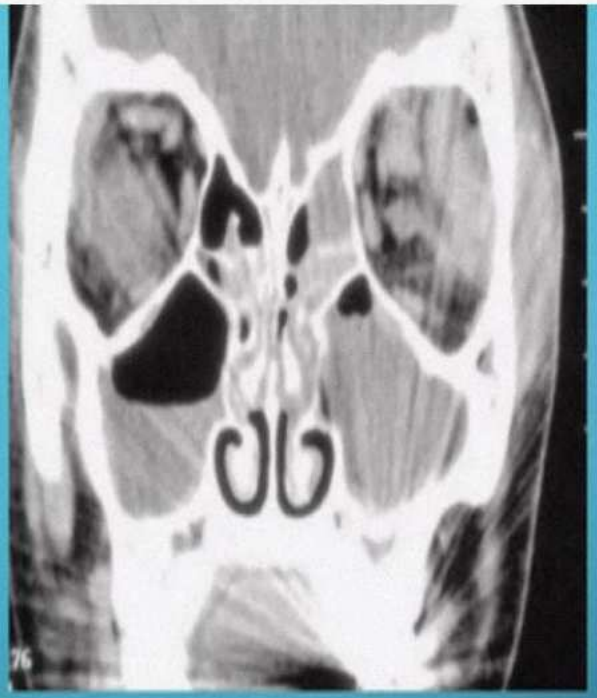




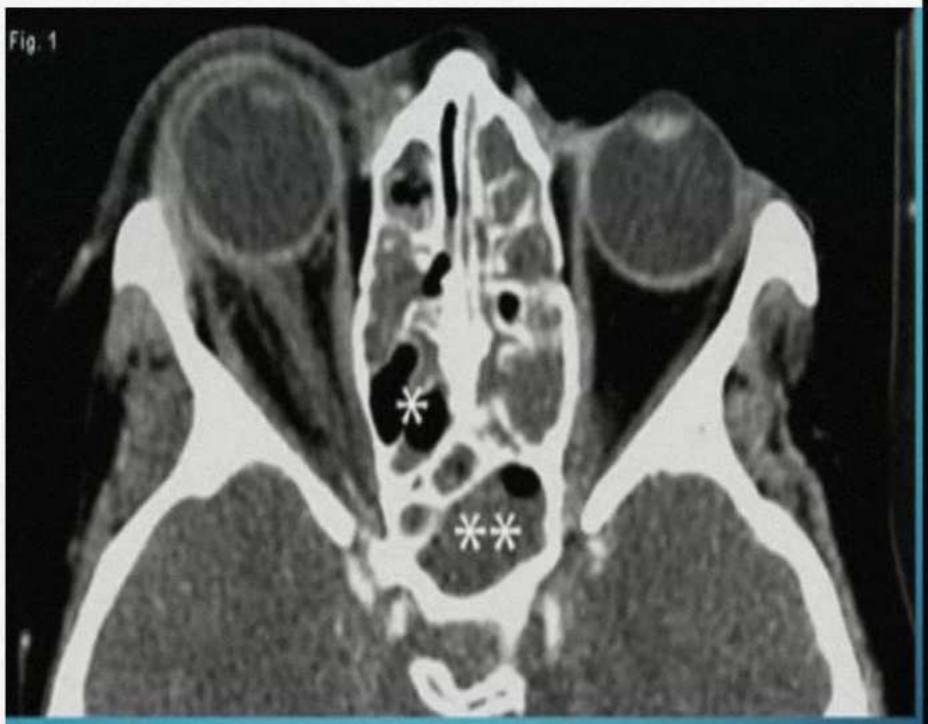
INVESTIGATIONS/WORK UP

- Full blood count
  Blood cultures
- Nasal swabs
- XRAY PNS
- BSCAN
- CT SCAN
- **MRI**
- LUMBER PUNCTURE



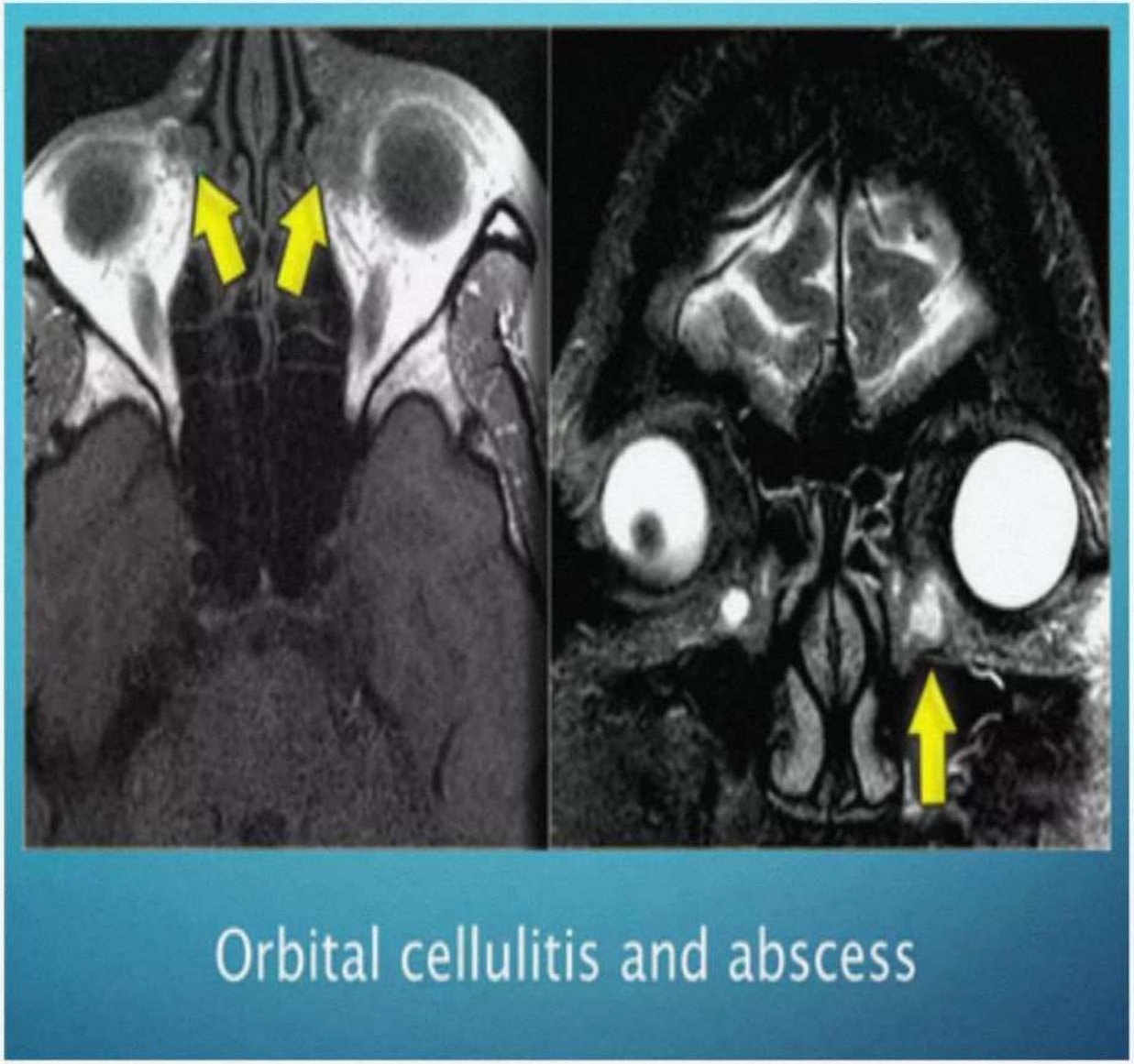


Coronal CT scan in a pediatric patient with sinusitis as well as an orbital and subperiosteal abscess (Left Side)



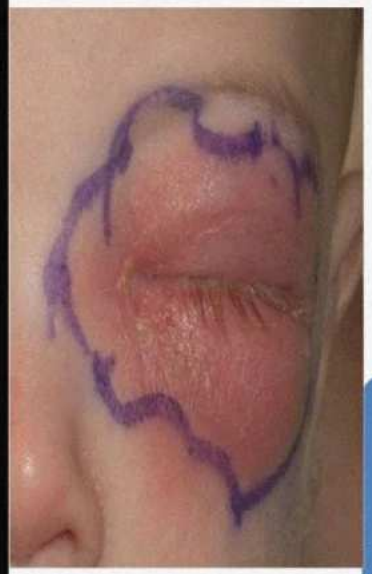
AXIAL VIEW CT scan of the orbit with contrast

- There is Proptosis and Retrobulbar fat stranding.
- Note the mucosal thickening and fluid in the ipsilateral ethmoidal (single asterisk) and sphenoidal sinuses (double asterisk) consistent with acute



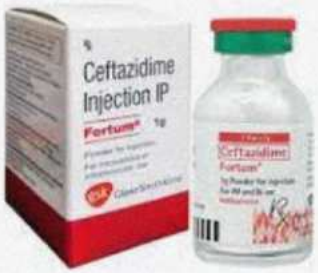
Orbital cellulitis and abscess

MANAGEMENT



- Hospitalization/ENT opinion
  - Proper work up
    - 4 hourly monitoring of vitals and visual functions/IOP/Pupils
    - Indications for surgical intervention
      LP for meningeal signs
  - Intensive antibiotic therapy
    - Analgesics
      Nasal decongestants
      Anti-inflammatory

IV ANTIBIOTICS



Ceftazidime
Ceftriaxone
Cefotaxime

Flucloxacillin
Vancomycin
Ampicillin -Salbactam

Ciprofloxacin/Levoflloxacin
metronidazole

- Ceftazidime (Fortum®)
  - Adults=1-2g/8hr I.V.
  - Pediatric
    - Neonates=30mg/kg IV
    - 1 month-12 years=30-50mg/kg IV
- Ceftriaxone (Rocephin®)
  - Adults=2g/12hr IV
  - Pediatric
    - 20-50mg/kg/day IV

- Cefotaxime (Claforan®)
  - Adults=1-2g/10ml/4hr IV in 3-5 min (max=12g/day)
  - Pediatric
    - 0-1 week=50mg/kg/12hr IV
    - 1-4weeks=50mg/kg/8hr IV
    - 1month-12years=50-80mg/kg/day IV in 4-6 doses

DOSES

Oral Drugs

- Amoxicillin-clavulanate (Augmentin®)
  - Adults=500mg TDS
  - Children=20-40mg/kg/day 3 divided doses
- Cefaclor (Ceclor®)
  - Adults=500mg TDS
  - Children=20-40mg/kg/day 3 divided doses
- Metronidazole

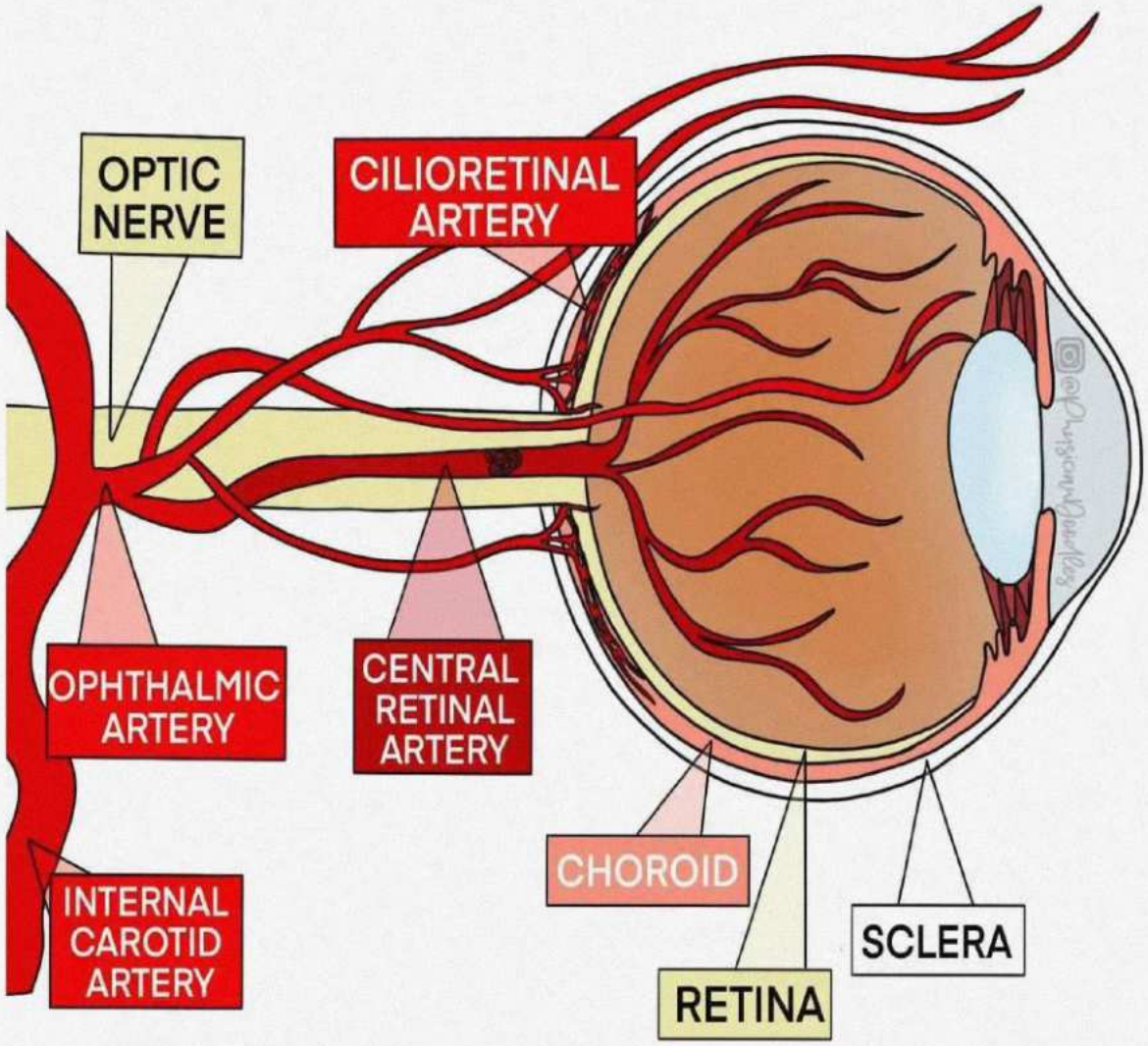
INDICATIONS FOR SURGICAL INTERVENTION

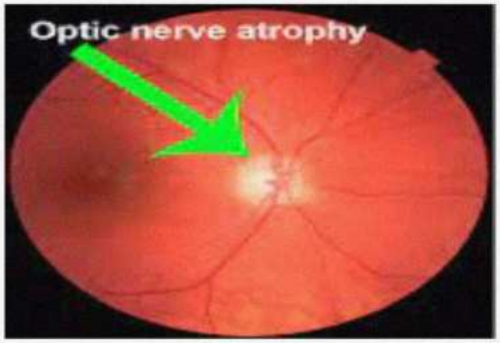
- Suspicion of orbital abscess/foreign body
- Decrease in VA
- EOM defects
- Worsening of proptosis
- Non reduction in abscess size on CT scan after antibiotic therapy

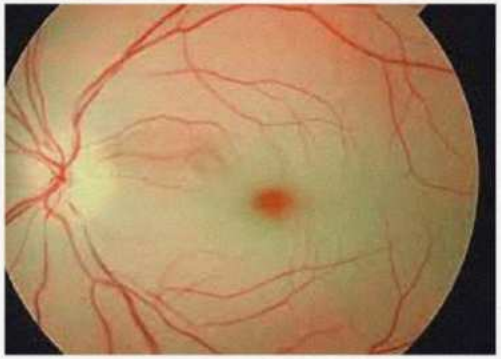
- Canthotomy/Cantholysis in emergency
- Surgical drainage of fluid
- Orbital surgery ± sinusotomy
- For fungal
  - Debridement
  - Exenteration

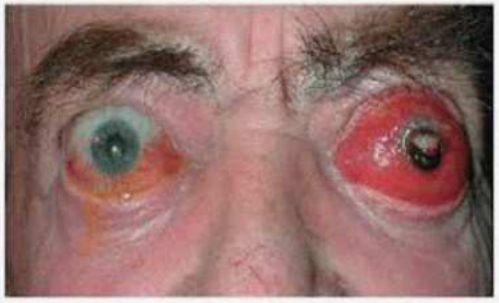
COMPLICATIONS

Exposure Keratopathy
Orbital abscess
Sub periosteal abscess
Central retinal artery occlusion/CRVO
Endophthalmitis
Increase IOP
Optic neuropathy
Cavernous sinus thrombosis
septicemia
Hearing loss
Intracranial spread of infection brain abscess/Meningitis
Increased mortality

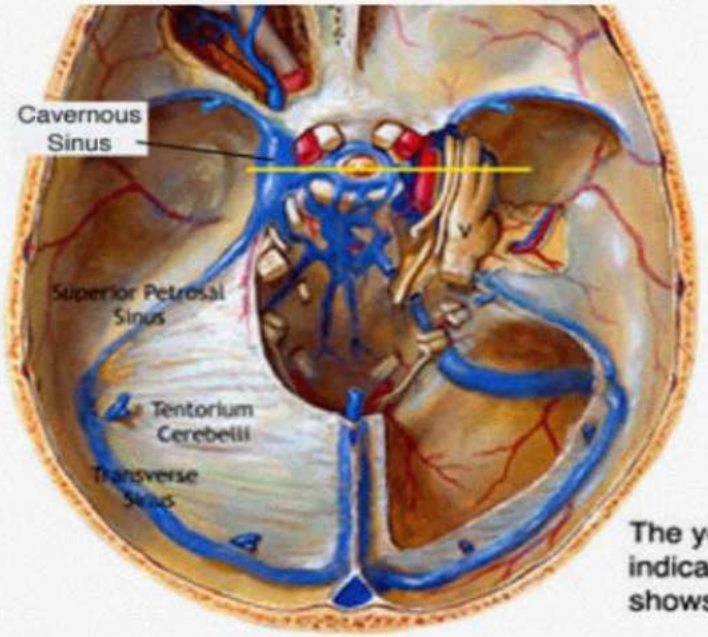


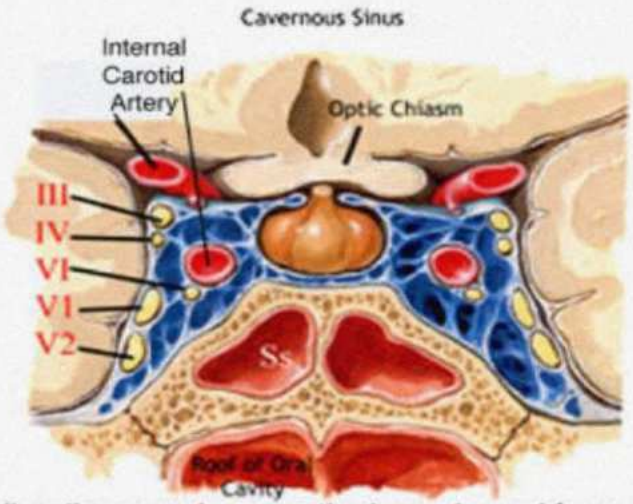






CAVERNOUS SINUS THROMBOSIS





The yellow line crossing over the hypophyseal fossa indicates the plane of section of th image above. It shows the cavernous sinus and its contents.

Clinical

- High **fever**
- **Periorbital edema** and **chemosis** (conjunctival edema)
- **Cranial nerve palsies** (CN VI most common)
- Decreased **visual acuity**

Dx

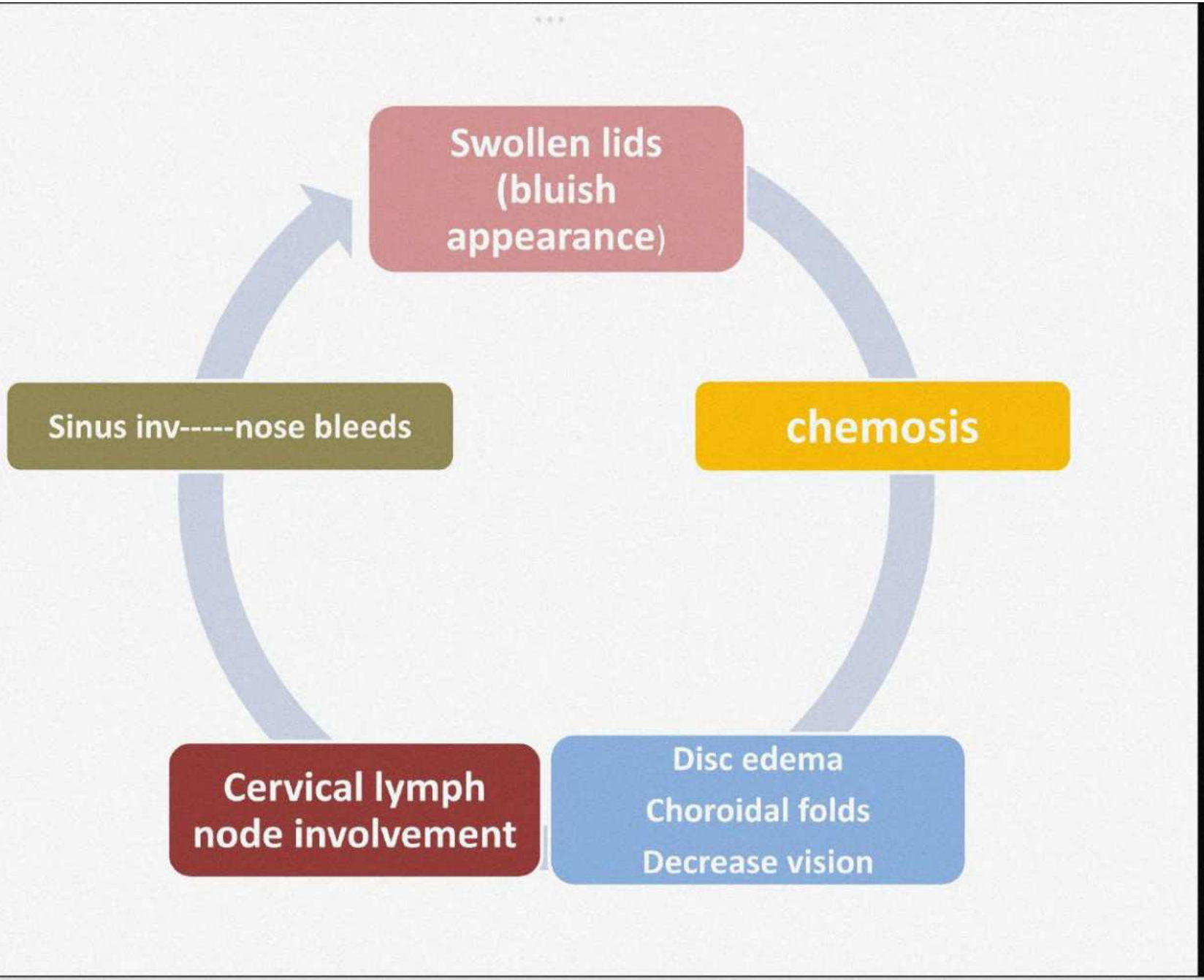
- CT scan
- **MRI**

Rx

- IV **ABX**
- **Heparin**

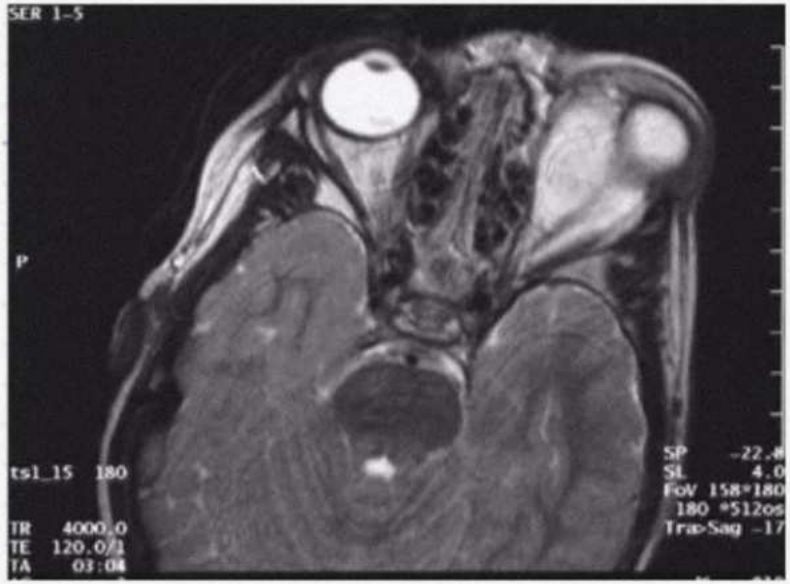
Embryonal Sarcoma

- Most common primary orbital malignancy of childhood
- Origin……undifferentiated mesenchyme cells
- Called Rhabdomyosarcoma if differentiate into striated muscles
- Usually first decade ……7years
- Boys more than girls
- Rapid onset, unilateral non axial painful proptosis mimics ….orbital cellulitis
- Location….. Superonasal,retrobulbar,superior inferior
- Histology …….Embryonal alveolar and pleomorphic



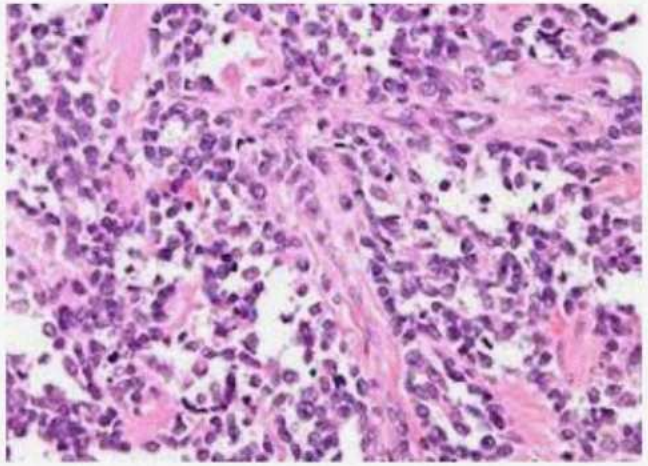
Work up

- USB,CT,MRI
- Biopsy
- Systemic inv



Management

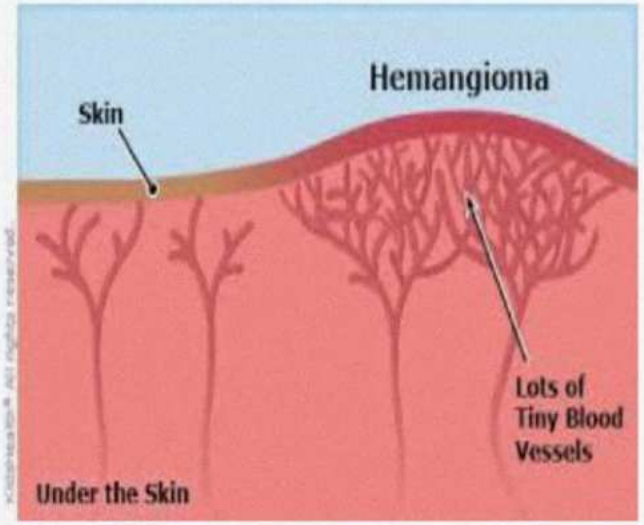
- Refer to pediatric oncologist
- Radiotherapy
- Chemotherapy
- Surgery



	RHABDOMYOSARCOMA	ORBITAL CELLULITIS
Pathology	Undifferentiated mesenchymal cells, most common primary orbital malignancy of childhood	Infection of soft tissues behind the septum
Demographics	Boys > girls (avg 7 yrs),	Commonest cause of proptosis in children, adults also affected
Associated	Cervical L. nodes involved	Usually after trauma, sinus related
Presentation	Nonaxial, painful, unilateral, rapid onset, swollen bluish lids, nose bleed, Ptosis, chemosis	Painful, lids edema with reddish color, Ptosis, malaise, fever, life threatening, VA impaired, Ophthalmoplegia,
Radiology	Moderately well-defined homogenous mass, Bony destruction	CT-(orbit, sinuses, brain): diffuse orbital infiltrate, sinus opacity, proptosis MRI for cavernous sinus thrombosis.
Tx	Radiotherapy, chemotherapy, Exenteration for resistant cases	Medical, surgical

CAPILLARY HEMANGIOMA

- Benign endothelial cell neoplasms
- Rapid growth in infancy and involutes later(75%-7years,30-50%-5years)
- Most common orbital tumor of infancy
- Usually located superonasal, brow and eyelid
- Blanches on pressure
- Ptosis and proptosis on posterior extension
- Cutaneous, preseptal,extraconal and intraconal



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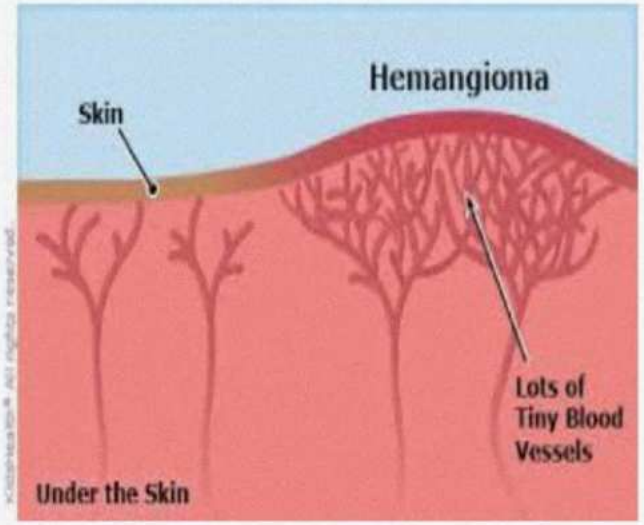
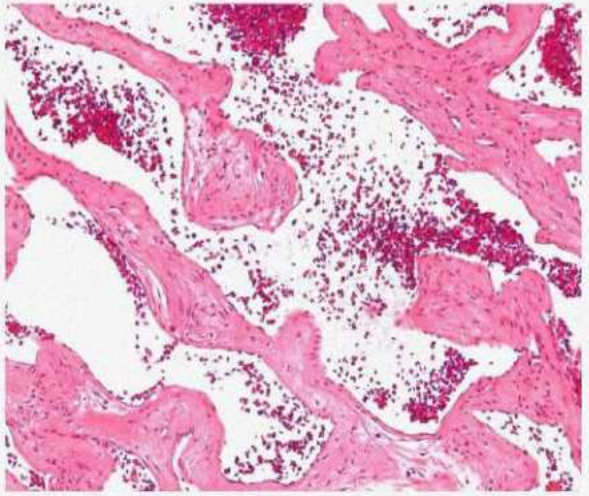
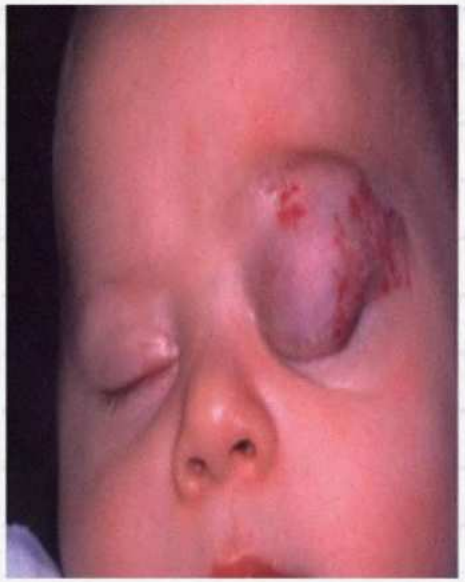
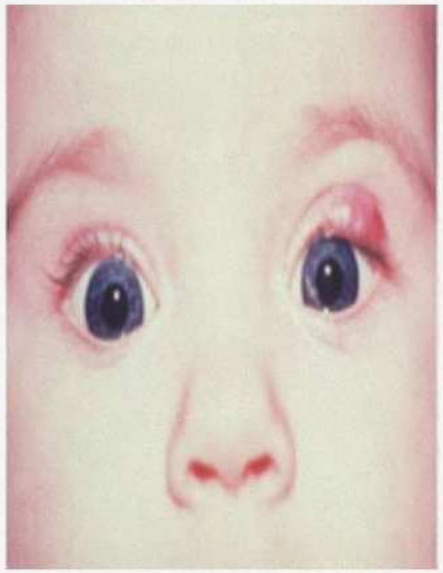




Mortality/Morbidity

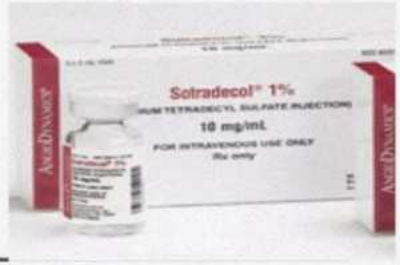
- Kasabach-Merritt syndrome
  - Coagulopathy, thrombocytopenia
  - large visceral / Nasopharyngeal hemangiomas
  - DIC may occur, high output CCF
  - Mortality...30-50%
- Ophthalmic morbidity
  - Space occupying
  - Amblyopia

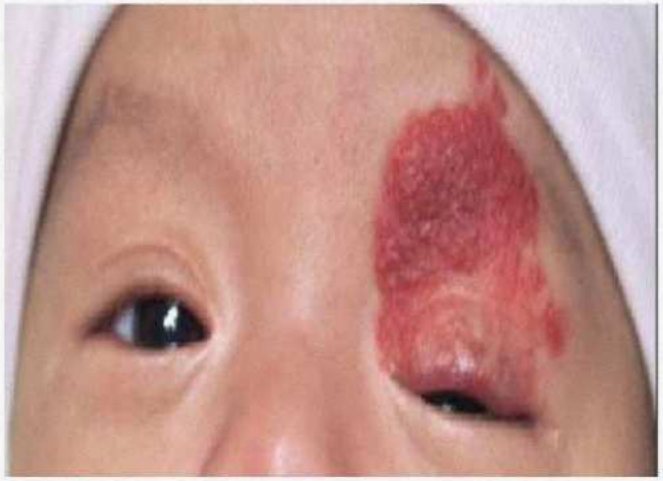


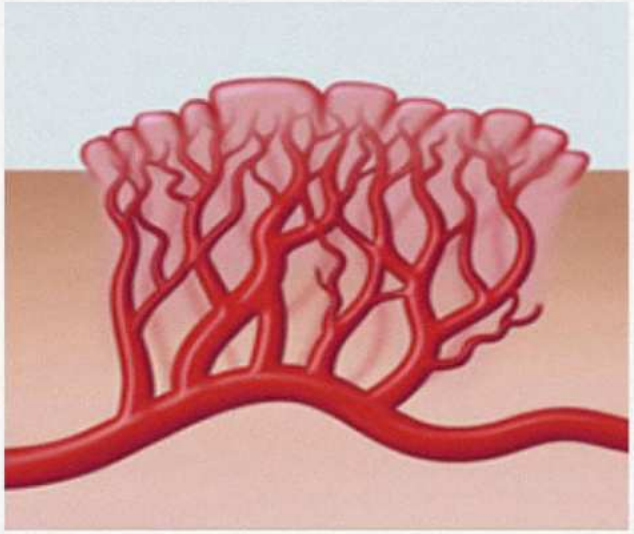


MANAGEMENT

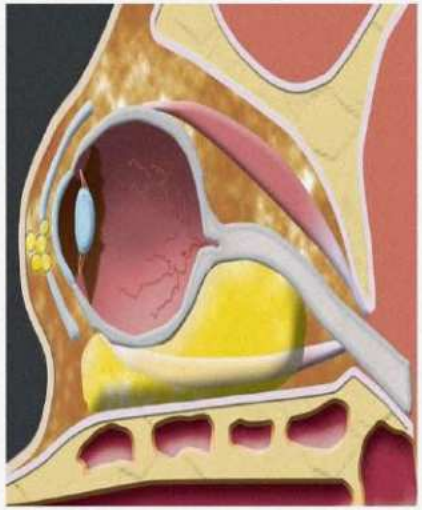
Medical	Surgical
Topical	
Topical corticosteroids under occlusion	Laser therapy
Intralesional corticosteroids	Cryotherapy
Becaplermin	Radiotherapy
Imiquimod	Surgical excision
Systemic	Compression
Systemic corticosteroids	Embolization
Vincristine	Sclerosant injection
Interferon-alpha	
Bleomycin	
Cyclophosphamide	

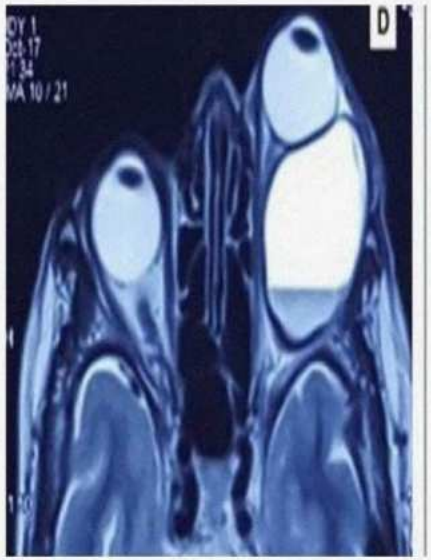




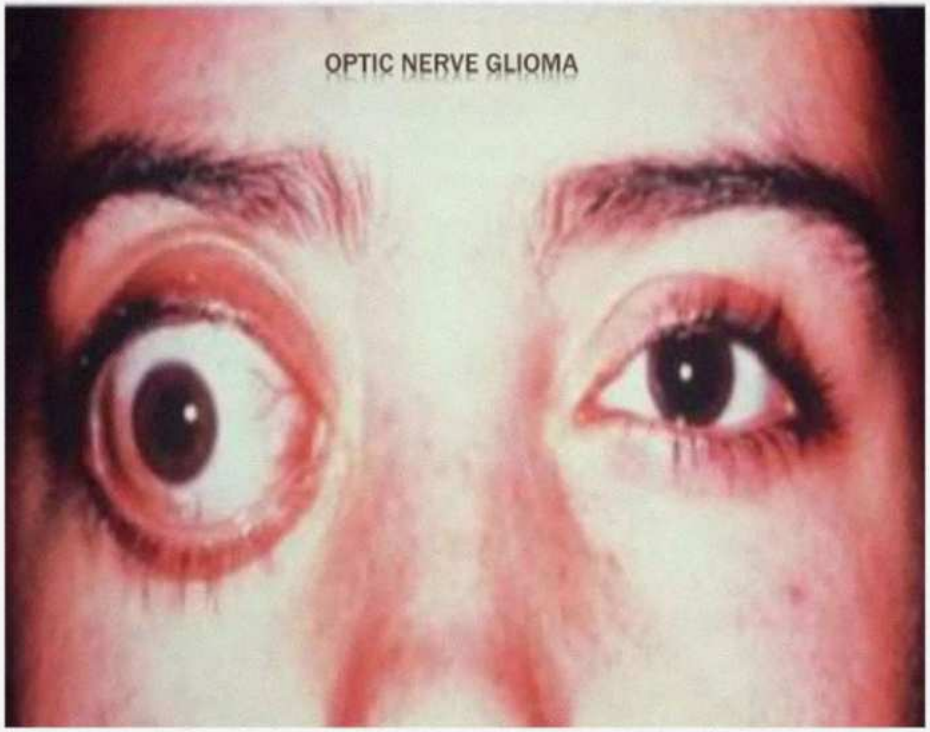


	CAPILLARY HEMANGIOMA	ORBITAL LYMPHANGIOMA
Pathology	Vascular hemartoma	Isolated vascular hemartoma
Demographics	Infant, commonest benign orbital tumor in childhood, spontaneous involution	Early childhood
Associated	Visceral, nasopharyngeal hemangioma, kasabach-merit syndrome	-
Presentation	Nonaxial, +ve Valsalva, superficial/deep	Nonaxial, -ve Valsalva, acute episodes of spontaneous hemorrhage, may be superficial/deep
Radiology	Intra/extraconal mass, poorly defined	Low density cyst-like mass, enlargement of bony orbit
Tx	Observation, surgery	Guarded prognosis, surgery, drainage





OPTIC NERVE GLIOMA



Classification

- **Primary Neural Tumors**

  Optic nerve
  Peripheral nerves

- **Secondary Neural Tumors**

  Metastatic and infiltrative neoplasms
  Breast cancer, prostate cancer, melanoma, lung cancer

  ON may be affected by peripheral nerve tumors
  in the orbit

OPTIC NERVE GLIOMA

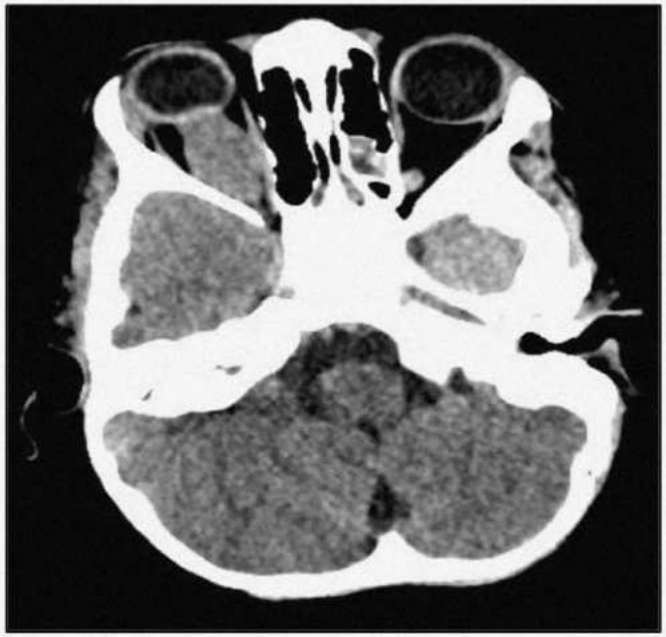
- Most common ON neoplasm, arising from astrocytes within the ON

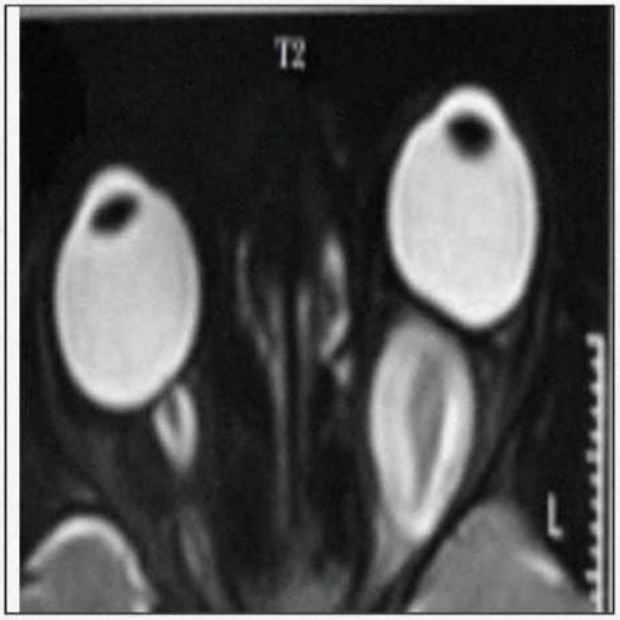
- BENIGN_ common in children (juvenile pilocytic astrocytomas)
- MALIGNANT in adulthood (malignant glioblastoma)

- 90% diagnosed during the first 2 decades of life (median age 5 years)
- Females preponderance (60%)

- 10-70% of patients diagnosed with juvenile pilocytic astrocytoma have associated (NF-1)
- In patients with NF-1, 8-31% are diagnosed with associated ON glioma

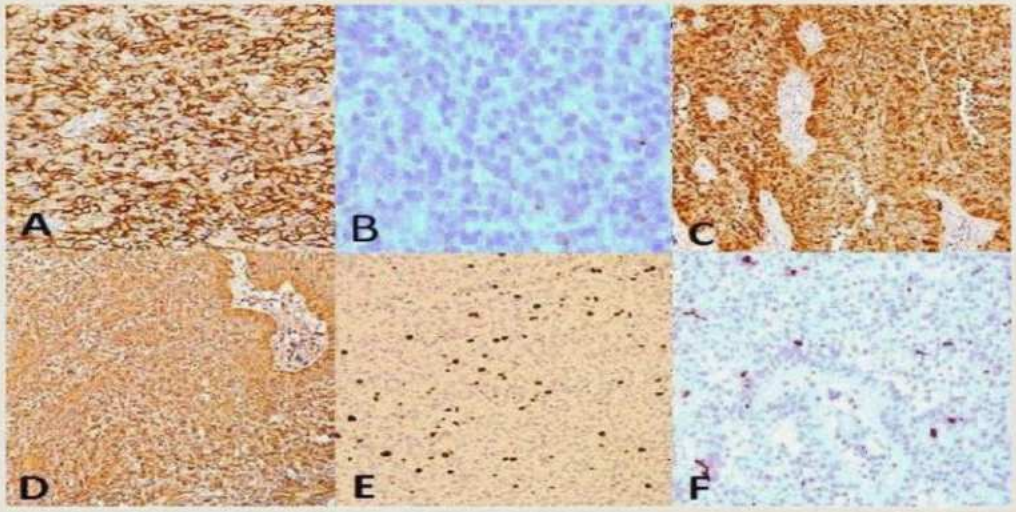
	NON –NEUROFIBROMATOSIS TYPE 1	NEUROFIBROMATOSIS TYPE 1
Presentation	Visual loss, strabismus,proptosis	Asymptomatic, visual loss
Progression	63%	12%
Visual outcome	Poor	Good
Growth rate	Faster- occasionally rapid	Stable, slow growing
Location	Discrete, unilateral	Multifocal, diffuse, bilateral
Survival	5 year =83% 10 years =63%	5 year =93% 10 years =81%
Radiographic findings	Fusiform ON enlargement , loss of perineural space	Fusiform ON enlargement , kinking of intraorbital nerve
Associated features	None	Café au lait spots, lisch nodules
Hydrocephalus	79% (radiologically)	Very rare
Follow up	Regular imaging	Not routine unless symptomatic





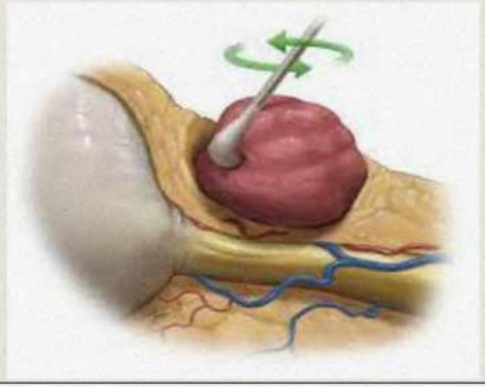
Cellular morphology
Immunohistochemistry

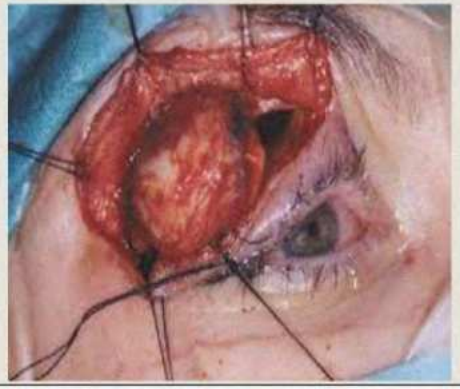
- Meningiomas are positive for EMA and S-100
- Gliomas are positive for GFAP and negative for S-100 and EMA
- Peripheral nerve sheath tumors contain Schwann cells that stain positively for S-100
- Perineural cells may also be positive for EMA
- Granular cell tumors stain positively for S-100 and may be positive for CD68 and leu-7

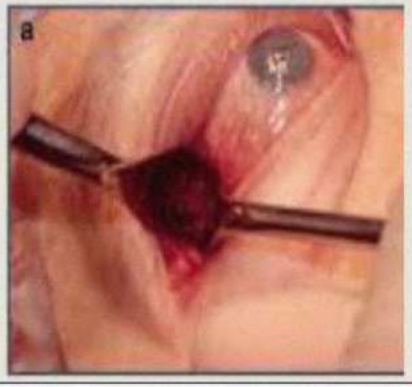


Treatment Options

- Observation
- Radiotherapy
- Chemotherapy
- Surgical excision





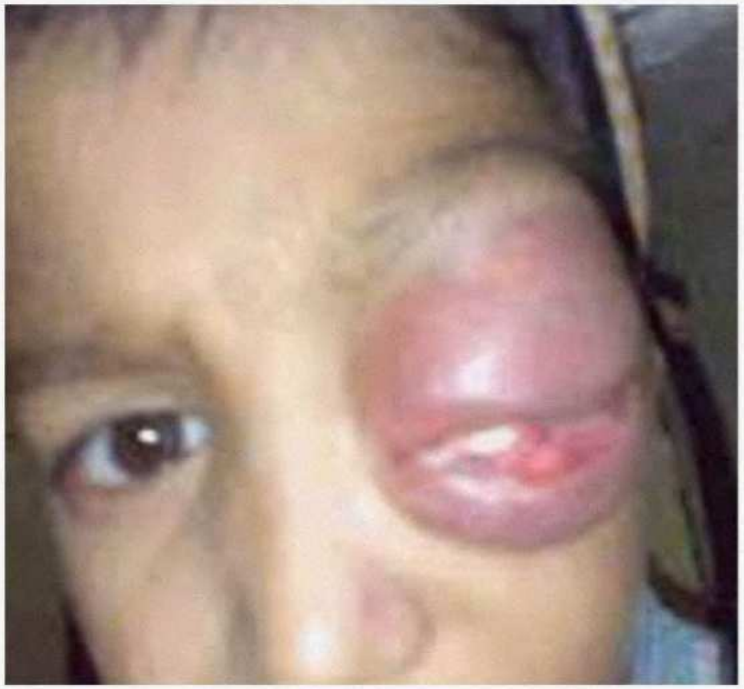


CHILDHOOD METASTATIC TUMORS

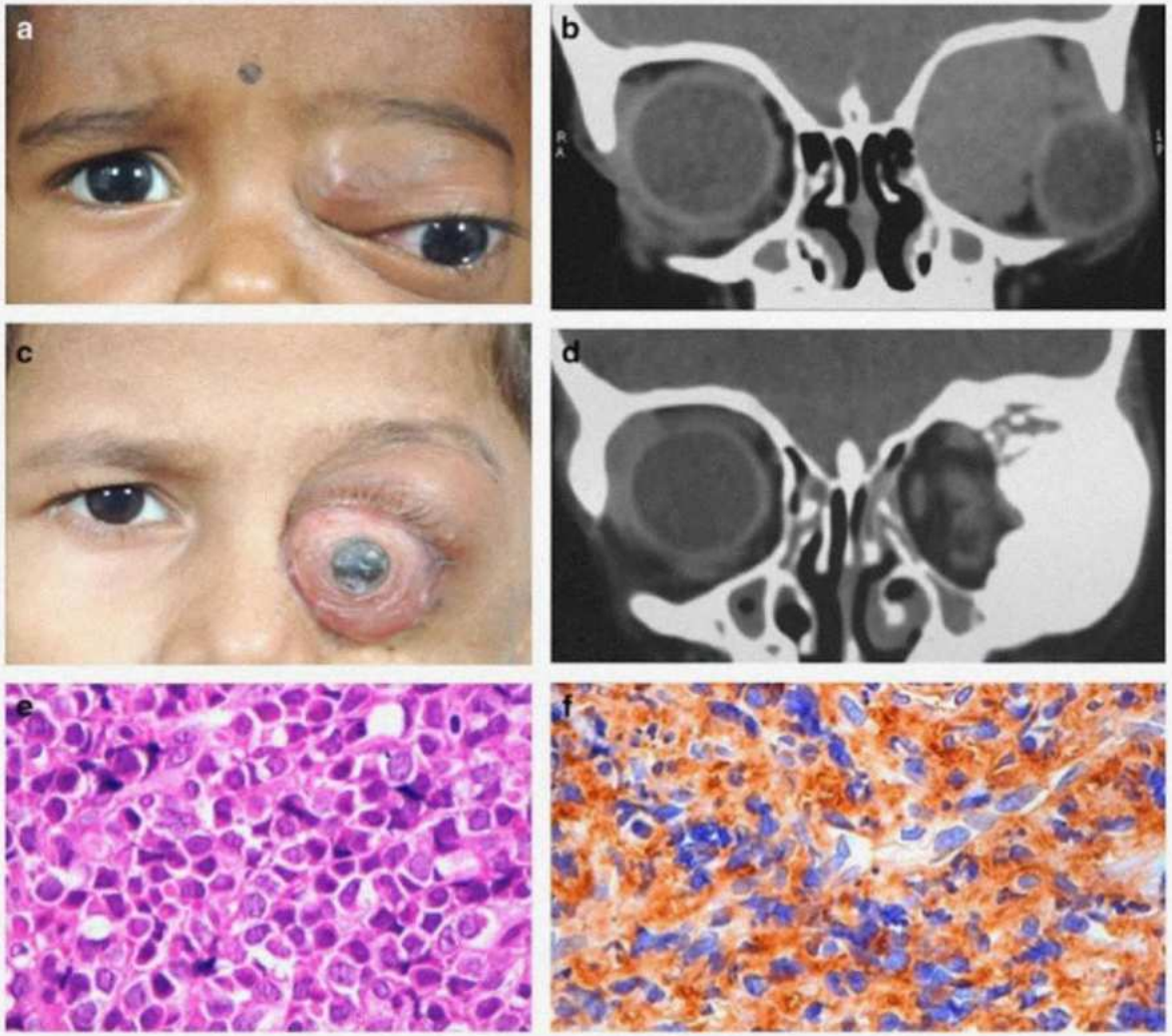
Neuroblastoma

- Most common childhood cancer.
- Frequent source of orbital metastasis.
- Originates in either adrenal gland or sympathetic ganglion chain in the retroperitoneal mediastinum.
- Metastatic Neuroblastoma in the orbit typically produces proptosis, & periorbital ecchymosis.
- CT – shows evidence of bone destruction.
- With intensive treatment including radio & chemotherapy prognosis is considerably better in infants under the age of 1 yr. than older children.

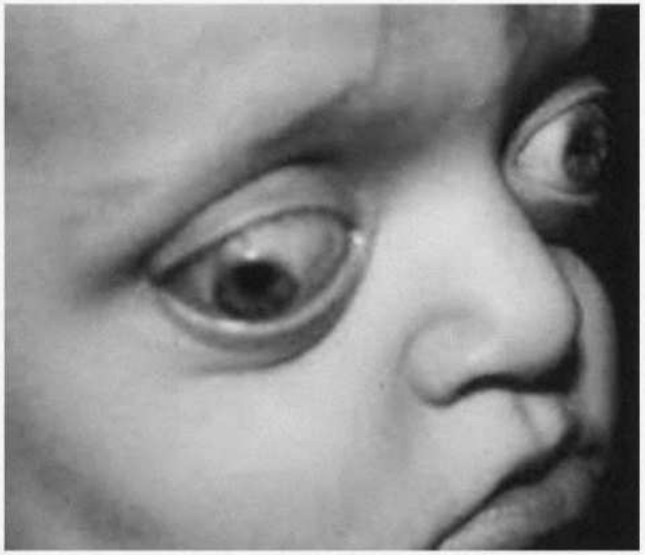
NEUROBLASTOMA



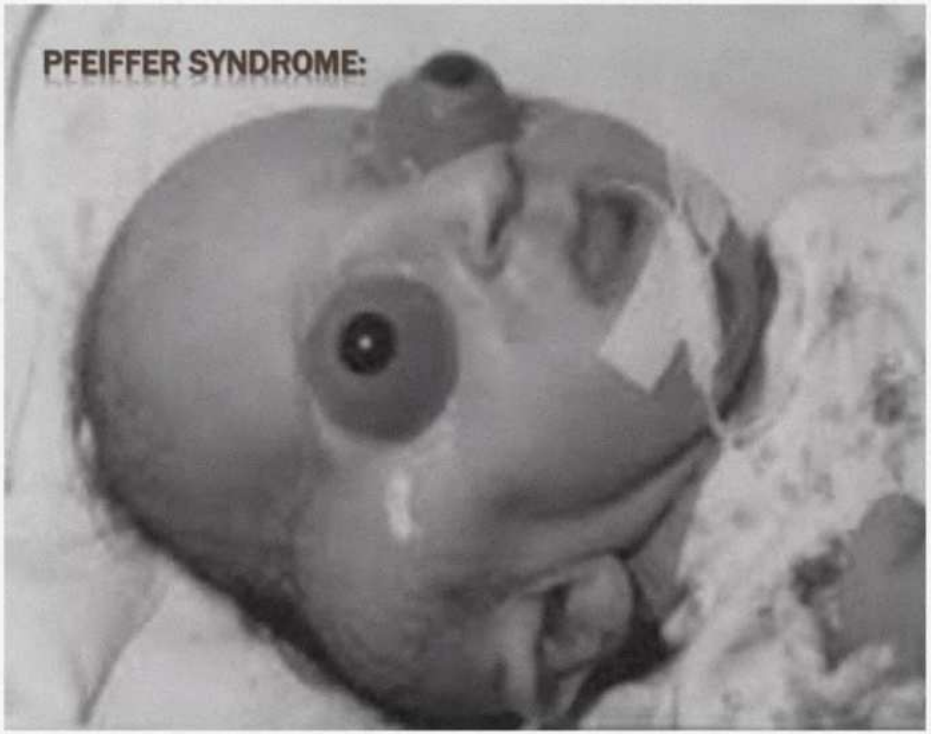
EWINGS SARCOMA



DEVELOPMENTAL ANOMALIES



CROUZEN SYNDROME



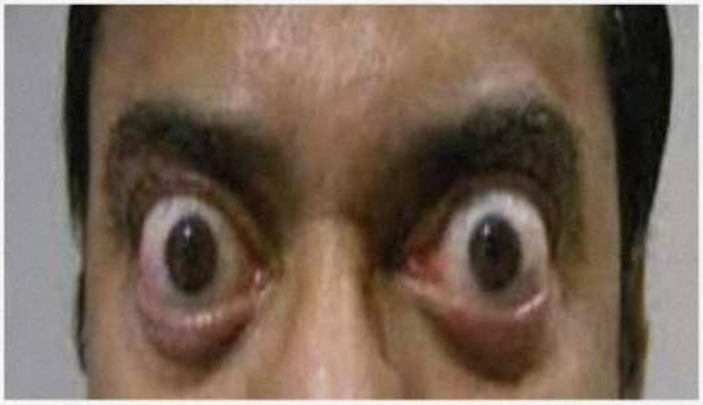
CONCLUSION

- General examination, CBC, peripheral smear and local imaging study is mandatory to rule out malignant conditions

- Histopathology aids in the precise diagnosis

Unilateral proptosis in a child-need for prompt diagnosis

PROPTOSIS in ADULTS



Prof SofiaIqbal
FRCS, MRCOphth
Fellowship Orbit/Oculoplastics
Fellowship Refractive surgery

ETIOLOGY

- *Endocrine*
- *Trauma*
- *Vascular*
- *Infective*
- *Inflammatory*
- *Neoplastic*
- *Others*

CATEGORY	SPECIFIC CONDITIONS
Endocrine	Thyroid eye disease
Trauma	Facial fracture, soft tissue swelling, retro bulbar hemorrhage
Vascular	Carotid cavernous fistula
	Cavernous Haemangioma
	Cavernous sinus thrombosis
Infective	Orbital cellulitis
	Mucormycosis
Inflammatory	IOID/Dacryoadenitis/Orbital myositis
	Tolosa hunt syndrome / Wegner granulomatosis
	Sarcoidosis
	Churg Strauss syndrome
Other	Paget disease/ fibrous dysplasia
	Langerhans cell Histiocytosis

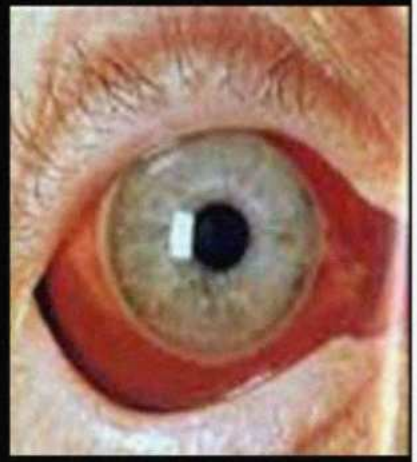
NEOPLASTIC DISORDERS

1.	Lacrimal gland tumors
2.	Meningioma
3.	Optic nerve Glioma
4.	Schwannoma
5.	Neurofibroma
6.	Lymphoma
7.	Sino-nasal tumors
8.	Ossifying fibroma
9.	Orbital Osteoma
10.	Neuroblastoma
11.	Haemangioblastoma
12.	Acute leukemia
13.	Myeloid sarcoma
14.	Metastasis

ORBITAL TRAUMA

- Orbital trauma can damage the facial bones and adjacent soft tissues. Fractures may be associated with injuries to orbital contents, intracranial structures, and paranasal sinuses.

Assessment of Orbital Trauma



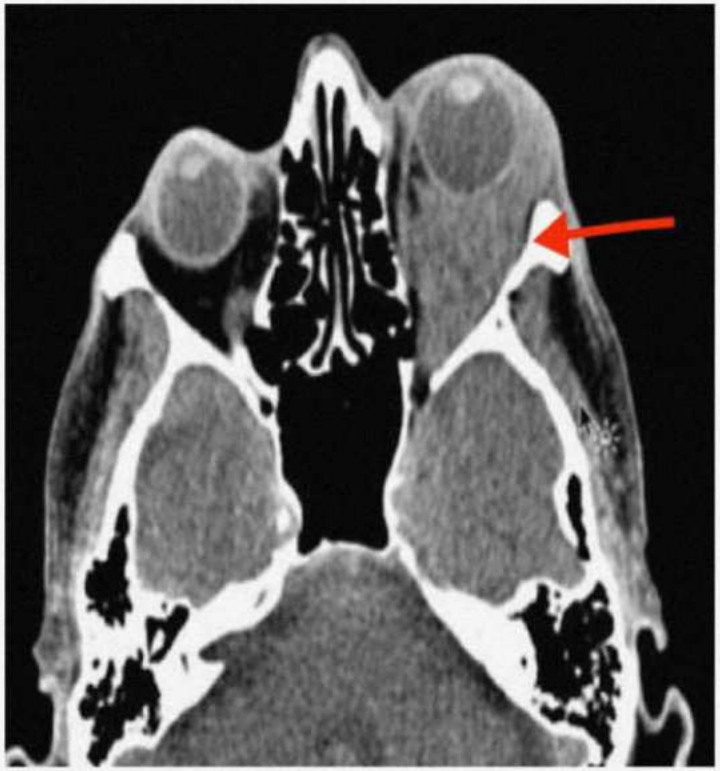
- Check ABC's
- History of mechanism of injury
- Signs & Symptoms:

  Varies but look for s/s suggestive of severe injury, including diplopia, visual loss, ptosis, lid laceration, subconjunctival hemorrhage, periorbital ecchymosis or infraorbital anesthesia
- Ophthalmologic examination:

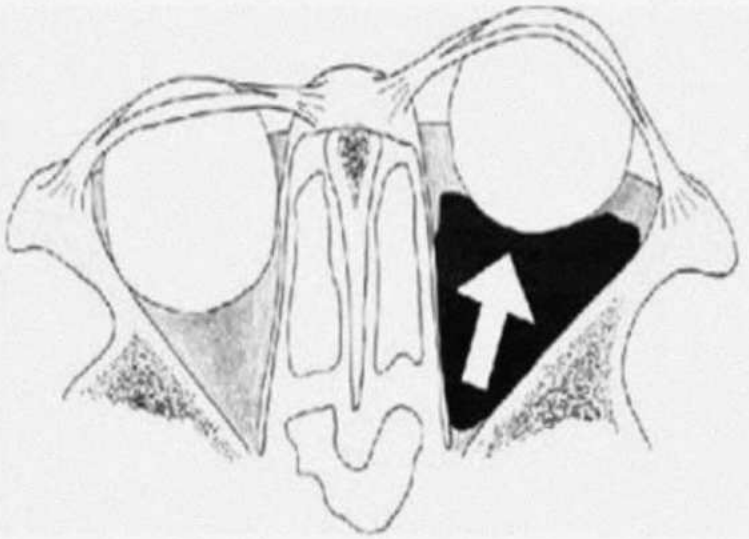
  visual acuity, pupil reaction, motility, sensation, globe position, lid function, integrity of globe and fundoscopy
- Imaging…

TYPES OF ORBITAL TRAUMA

- Blow out orbital floor fracture
- Blow out medial wall fracture
- Roof fracture
- Lateral wall fracture



•*Proptosis*



Without pulsation

-Orbital hemorrhage

-Emphysema

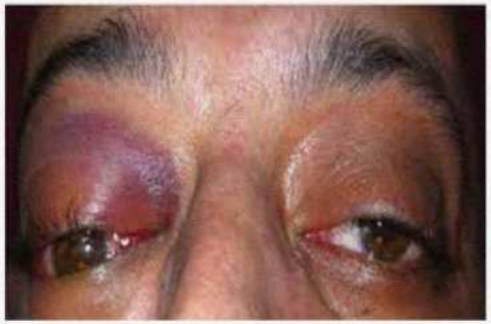
With pulsation

-High flow CCF

Orbital roof fracture with secondary herniation of anterior cranial fossa contents

Retro bulbar Hemorrhage (RBH)

- Rapidly progressive, sight-threatening emergency that results in an accumulation of blood in the retro bulbar space due to a rupture of an artery /vein
- The pressure increases and leads to the damage of the structures
- Treatment: blood evacuation, canthotomy, cantholysis



VASCULAR LESIONS

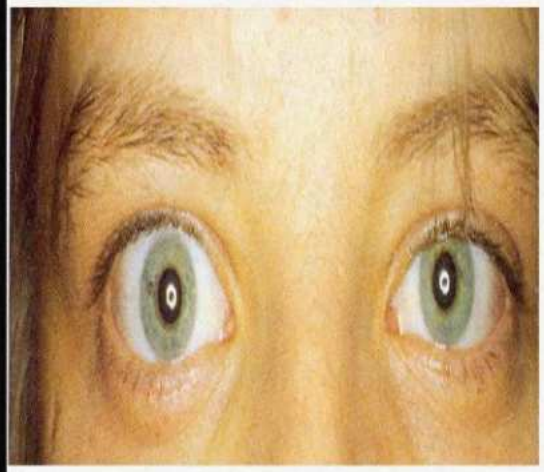
1. Cavernous Haemangioma
2. Orbital venous anomalies
3. Carotid-cavernous fistula
4. Cavernous sinus thrombosis

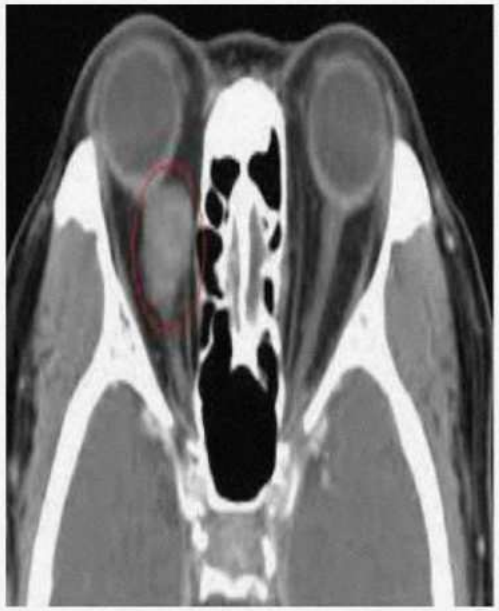
CAVERNOUS HEMANGIOMA

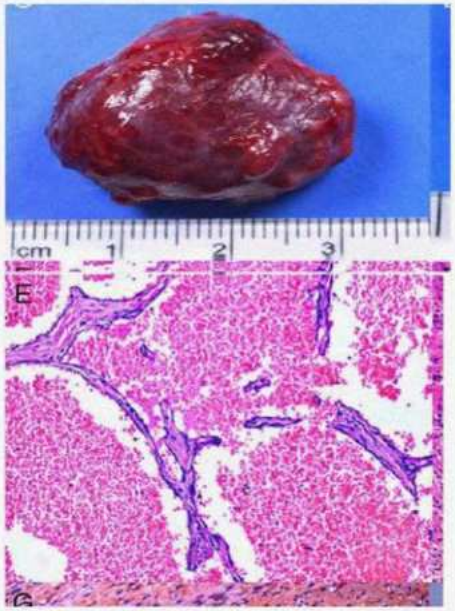
- It is the most common benign orbital tumor in adults
- Frequently occurs within the muscle cone
- Female preponderance is 70%
- 4^{th} to 5^{th} decades with slowly progressive unilateral proptosis
- May compress the optic nerve at orbital apex
- Gaze evoked temporary blurring of vision is an occasional feature

Treatment Surgical excision

- **Benign, noninfiltrative, slowly progressive vascular neoplasm composed of endothelial-lined spaces surrounded by a well-delineated fibrous capsule.**

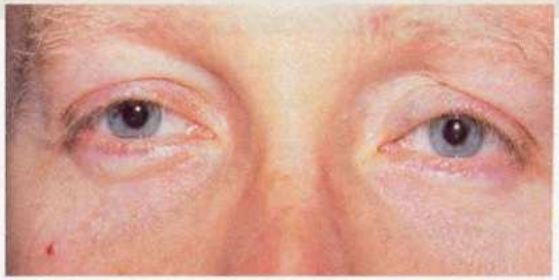


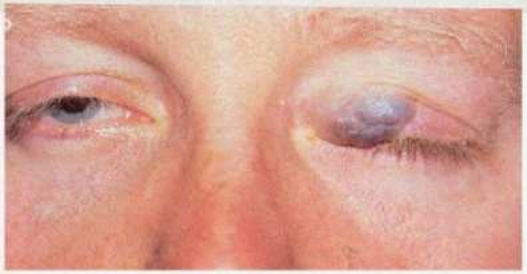


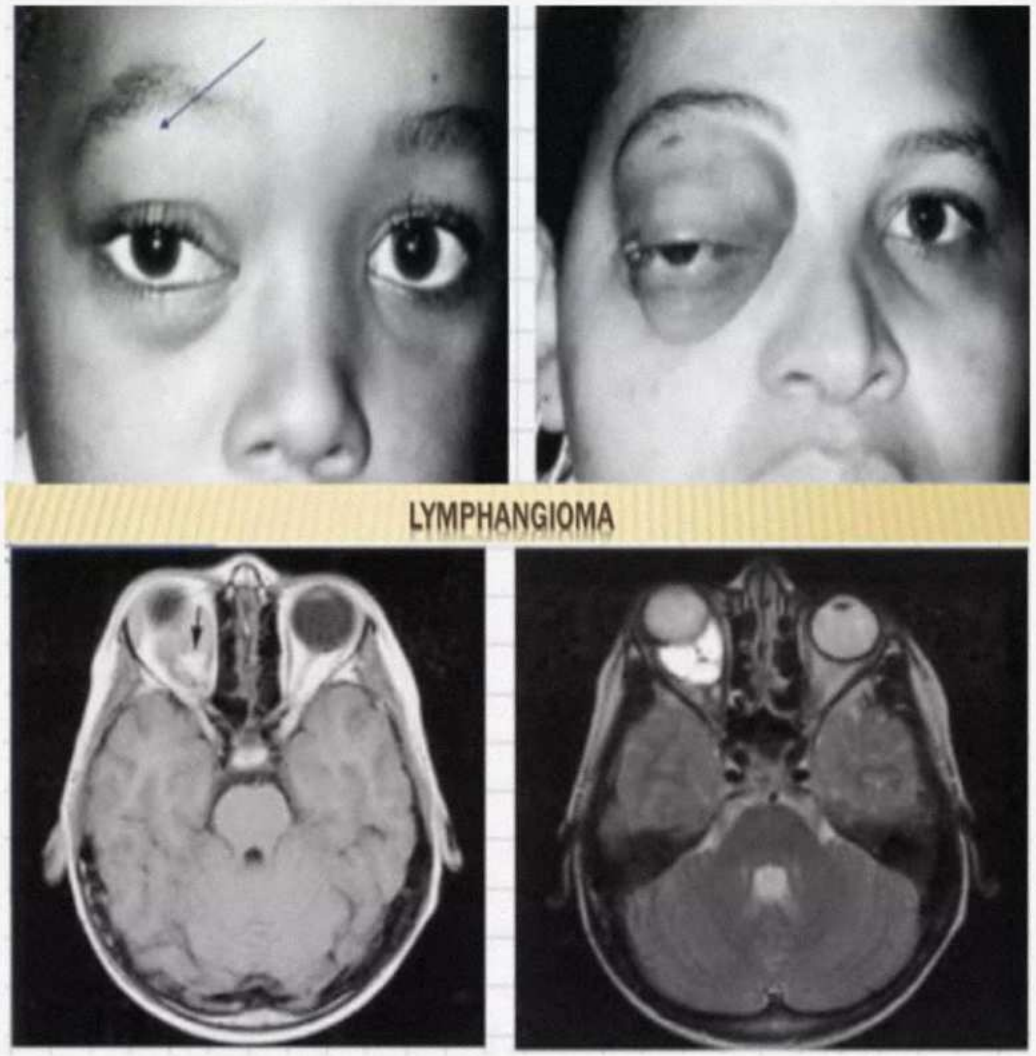


Orbital Varices/OLM

- Venous malformations of the orbit caused by vascular dysgenesis
- They consist of a plexus of thin-walled distensible low flow vein-like vessels that are commonly intrinsic to the normal circulation
- They are considered to be hamartomatous, often located in the intra conal area of orbital apex
- They cause intermittent proptosis when pressure increase due to certain maneuvers

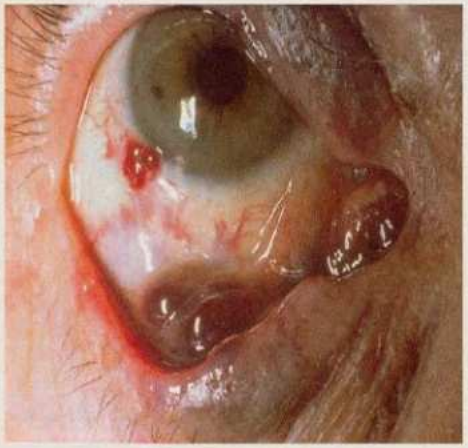


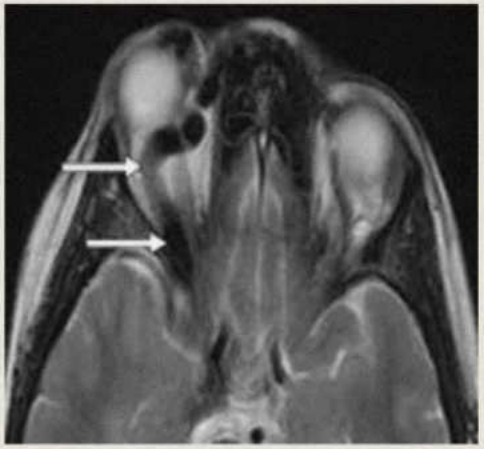


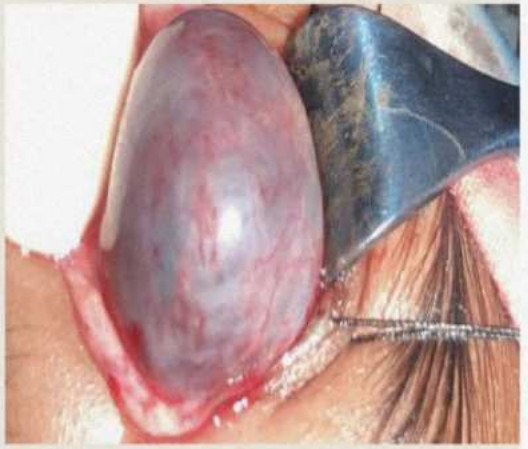


Treatment

- **Excision, Aspiration of chocolate cysts, Sclerosing agents (Bleomycin)**
- ***Indications*** **for treatment are proptosis,hemorrhage,thrombosis**

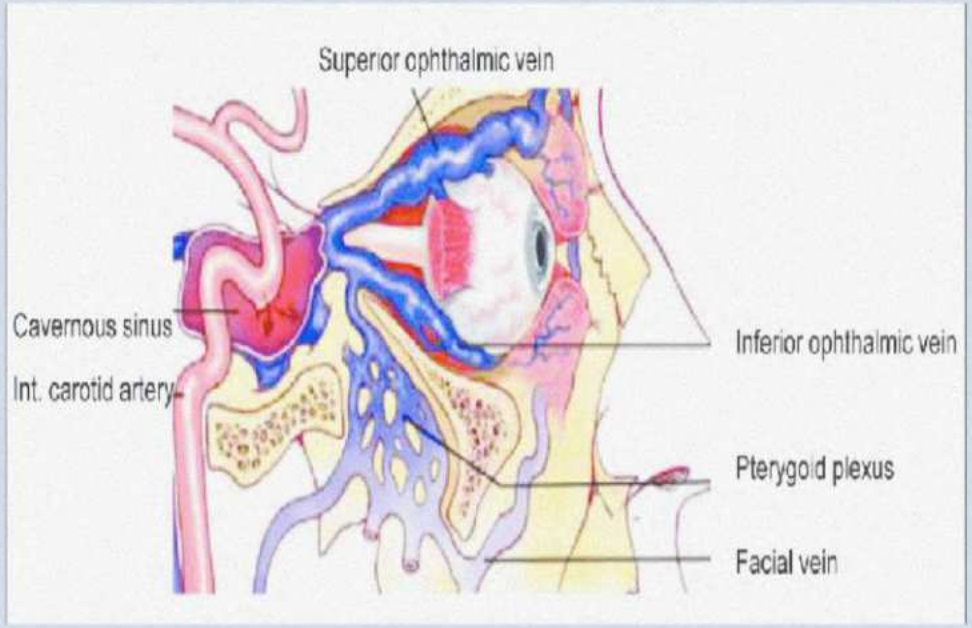






Carotid-cavernous fistula

- CCF is the result of an abnormal vascular connection between the internal carotid artery (ICA) or external carotid artery (ECA) and the venous channels of the cavernous sinus leading to a compromise in venous drainage from the eye
- Direct type…….Trauma/ruptured anuerysm
- Indirect type……..Hypertensive old ladies /postmenopausal/insidious



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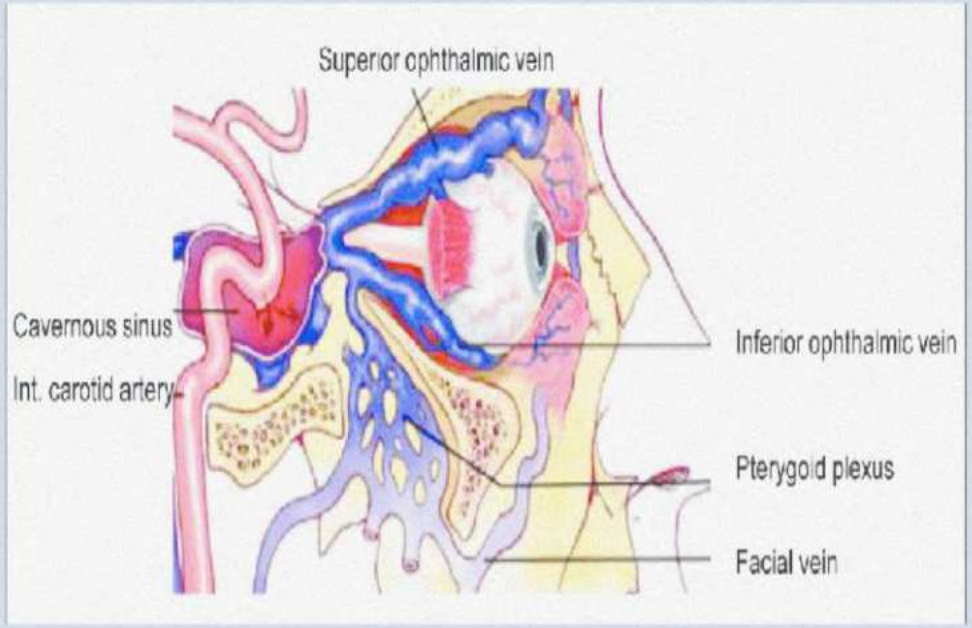
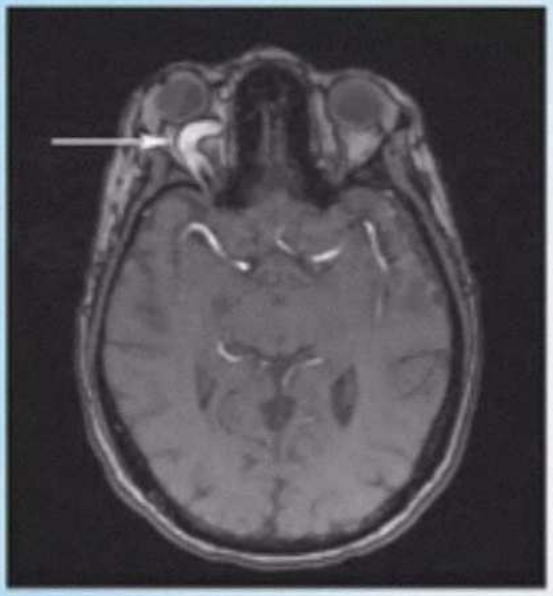
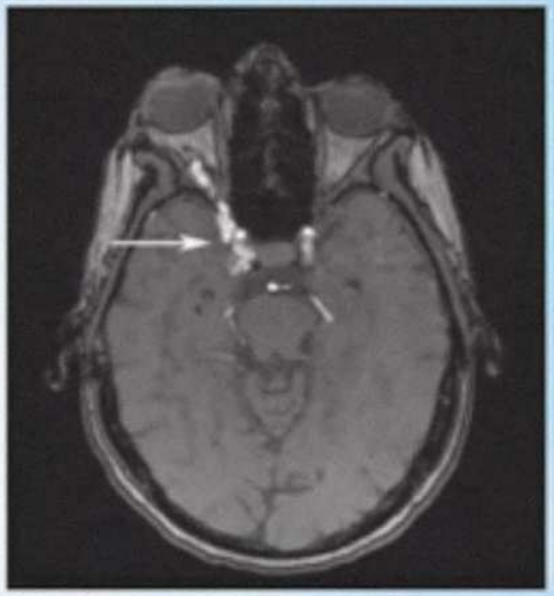


MRI

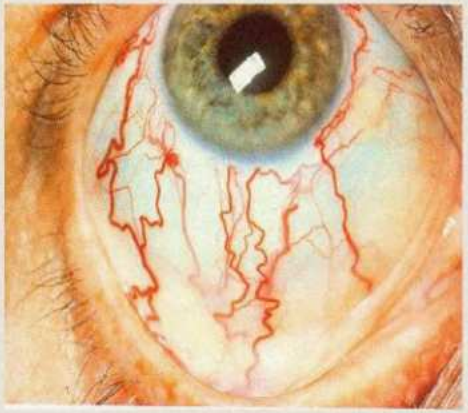


Magnetic Resonance Angiogram (MRA) image demonstrating an enlarged superior ophthalmic vein

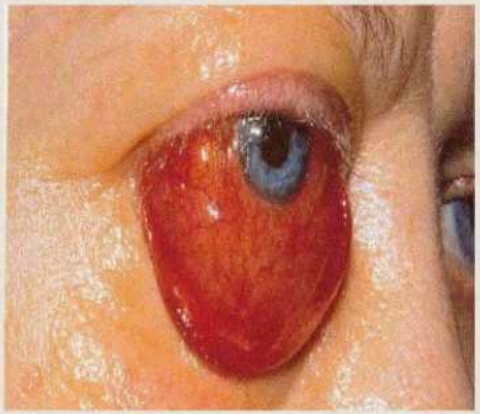


MRA demonstrating a right carotid cavernous fistul

- Congestion, headaches, cranial nerve palsies, intermittent proptosis
- Treatment -------endovascular embolization



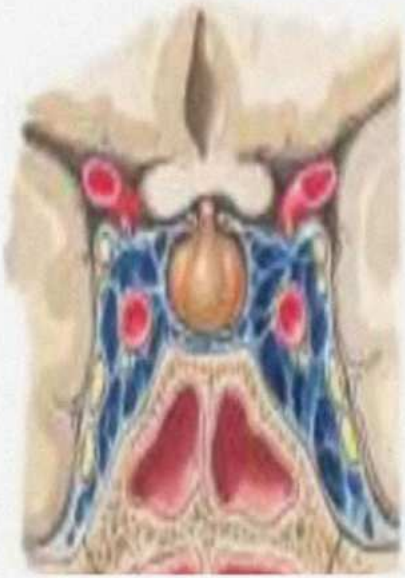
LOW FLOW CCF



HIGH FLOW CCF

CAVERNOUS SINUS THROMBOSIS

- **Anterior -** extends into medial end of superior orbital fissure.
- **Posterior -** upto apex of petrous temporal bone.
- **Medial –** Pitutary above and sphenoid below
- **Lateral –** temporal lobe and uncus



Causes

Septic CST

- Infectious

Aseptic CST

- Trauma
- Post surgery

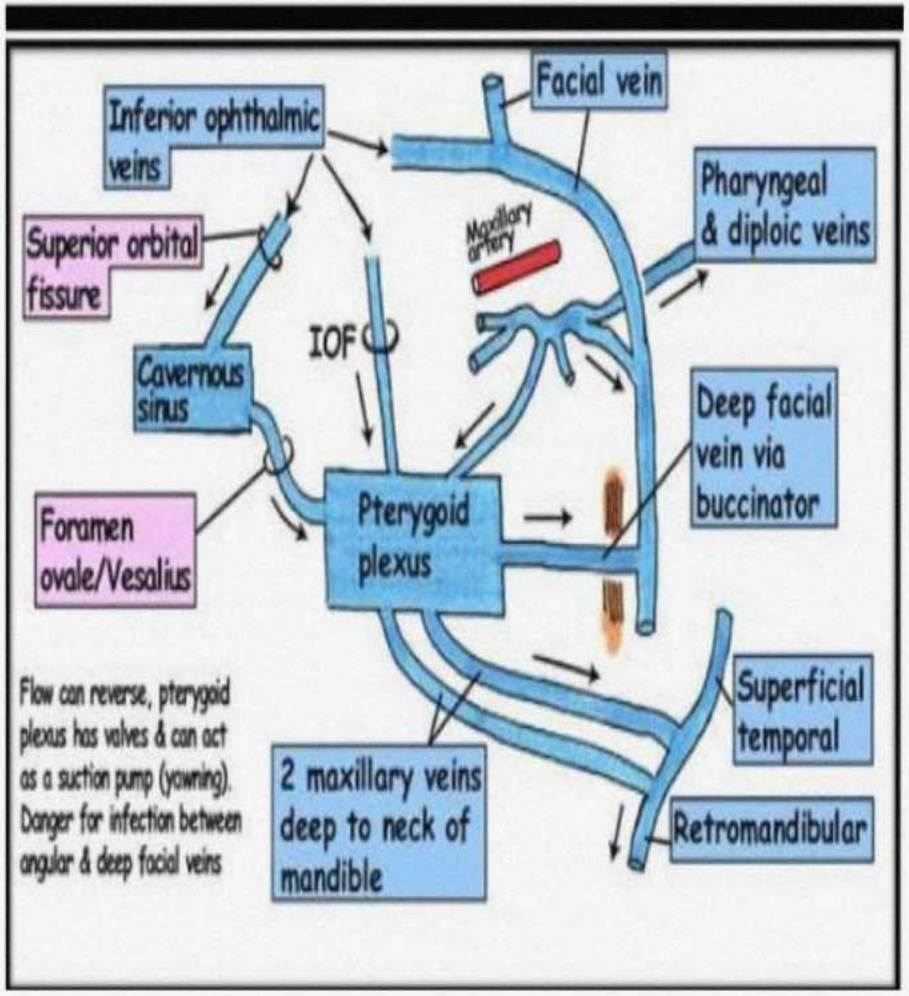
Rhinoplasty
Base of skull
Tooth extraction

- Hematologic
- Malignancy

Nasopharyngeal Ca.

- Dehydration

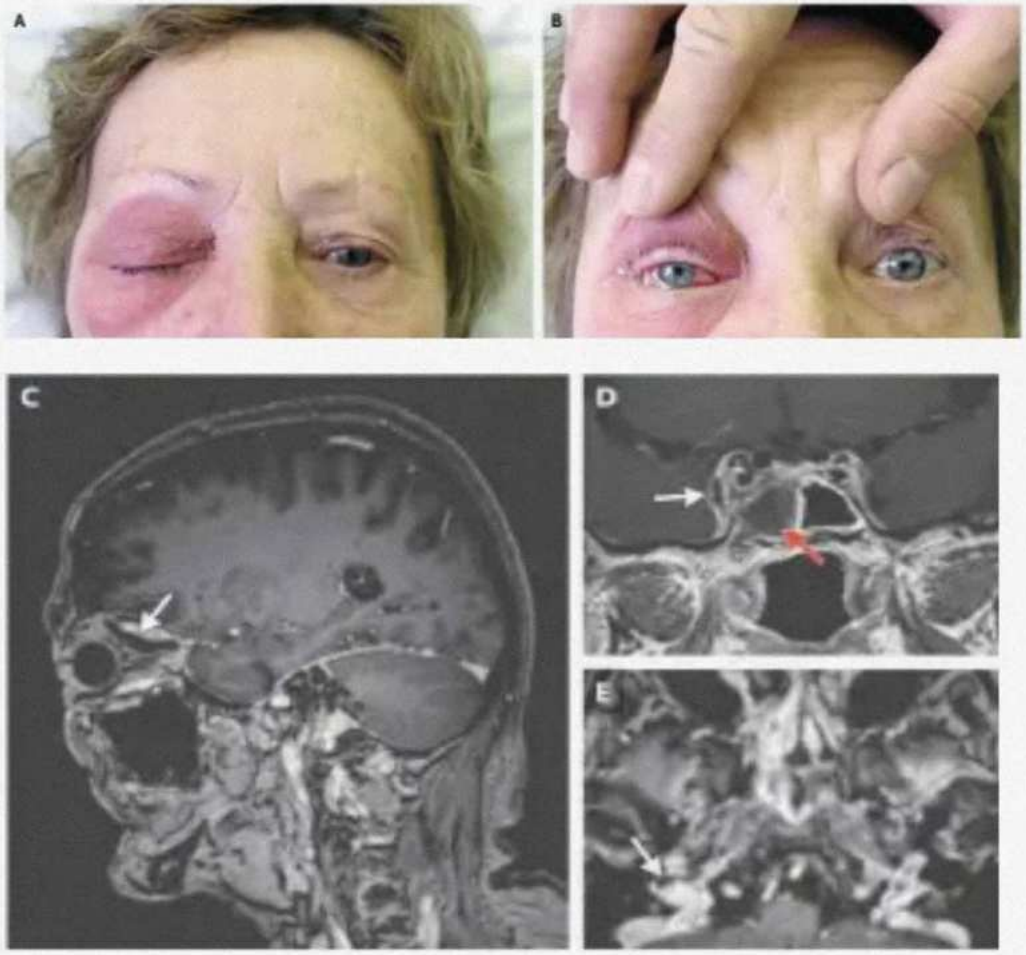
Venous connections of cavernous sinus

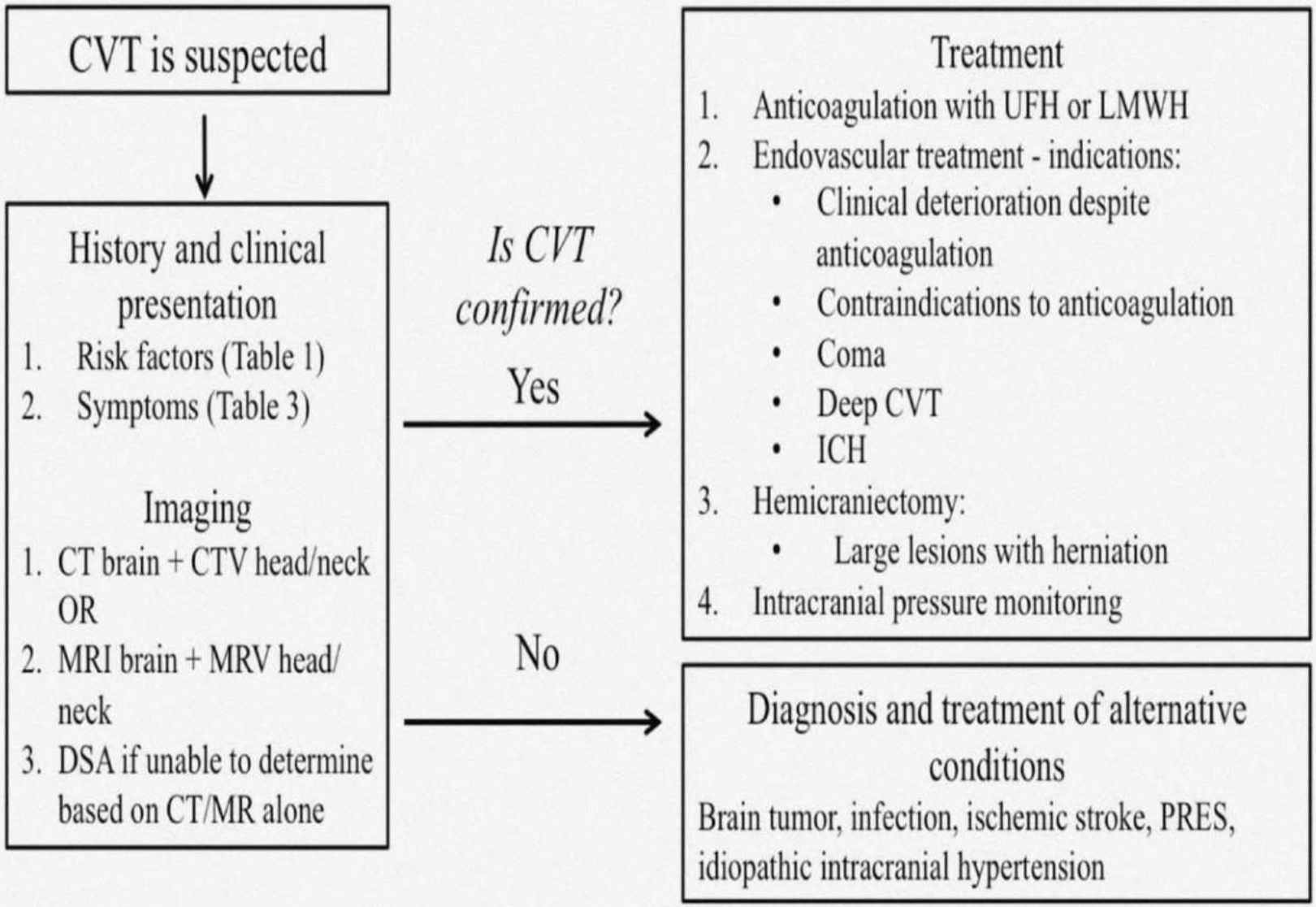


Venous obstruction

- Proptosis (first oedema & chemosis)
- Oedema of eyelids and bridge of nose
- Dilatation and tortuosity of retinal veins
- Retinal hemorrhages
- Involvement of the contralateral eye - (48 hours)
- When pterygoid plexus is occluded along with sinus, - oedema of the pharynx or tonsil

Septic Cerebral Venous Thrombosis



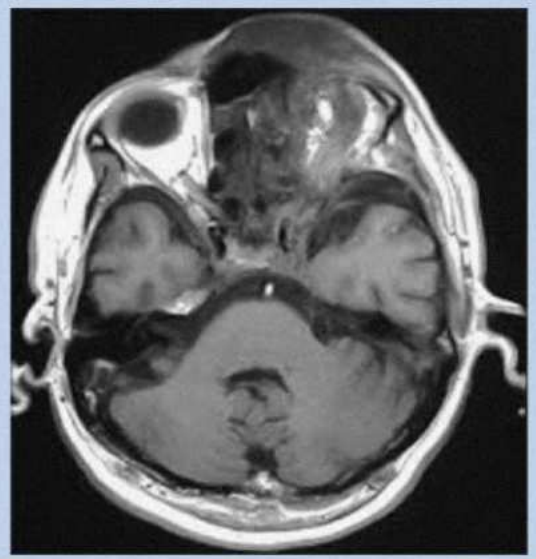


INFECTIVE

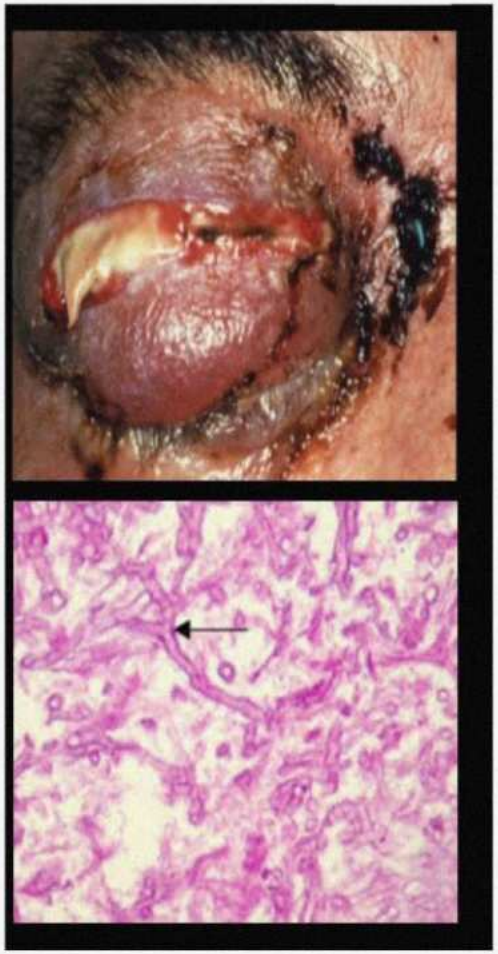
- Orbital cellulitis
- Orbital Mucormycosis

Rhino-orbital Mucormycosis

- An **aggressive invasive fungal infection** which tends to affect patients with a history of diabetes (especially with ketoacidosis), chronic steroid use, and immunosuppression
- COVID Pandemic

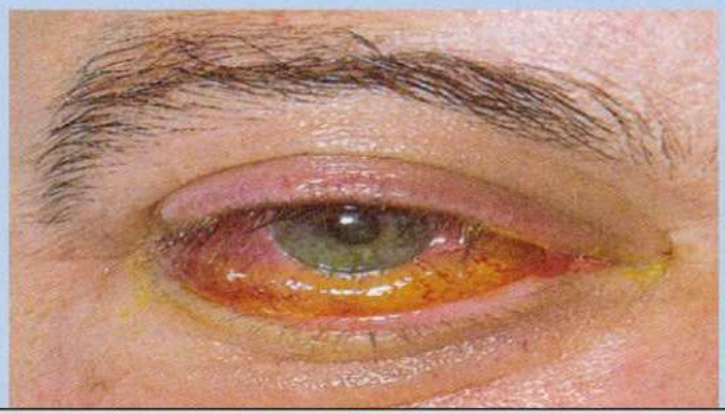


- Inhalation of spores → upper respiratory infection → Spread to contiguous sinuses → spread to orbit and brain.
- **PATHOGENESIS** : Invasion of blood vessels by the hyphae results in occlusive vasculitis with infarction of orbital tissues.
- **CLINICAL FEATURES** : Facial and periorbital swelling, diplopia , visual loss ,proptosis , ophthalmoplegia , black eschar on the palate, turbinates, nasal septum, skin, eyelids.
- **INVESTIGATIONS** : Tissue biopsy from nasopharynx/orbit. Histology: Non-septate , large , branching hyphae .
- **COMPLICATIONS** : Retina vascular occlusions, cranial nerve palsies, cerebrovascular occlusions.
- **TREATMENT** : Treatment of underlying conditions, wide excision of necrotic issues , intravenous amphotericin B (1mg/kg/day) or Liposomal amphotericin B for three weeks , Oral Posaconazole(300 mg/day) and hyperbaric oxygen therapy.



ORBITAL INFLAMMATORY DISEASE

- Characterized by non-neoplastic and noninfectious space occupying orbital lesion
- Unilateral disease is the rule in adults but in children bilateral involvement occurs in 30%
- Presentation is usually b/w 20 and 50 years with abrupt painful onset

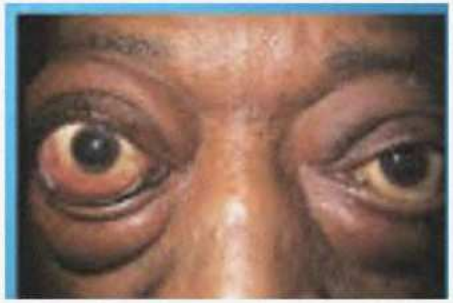


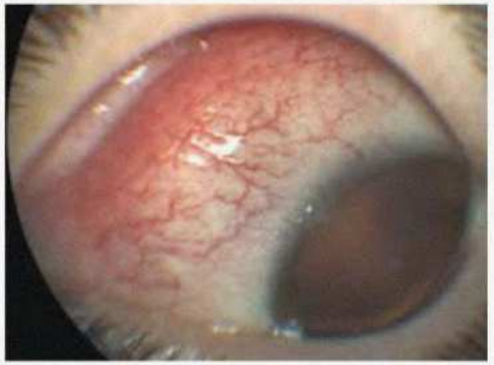
Classification of Orbital Inflammation

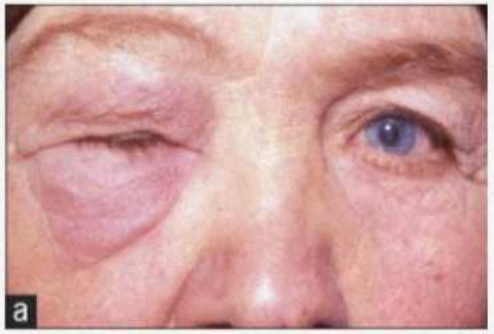
- **Idiopathic inflammatory disease**
- **Neoplasm**
  - Lymphoma
  - Rhabdomyosarcoma
  - Choroidal Malignant melanoma with extrascleral spread
  - Metastatic disease
- **Conenital malformation**
  - Dermoid cyst
  - Lymphangioma
- **Infectious disease**
- **Systemic inflammatory disease**
  - Thyroid associated ophthalmopathy
  - Wegener's granulomatosis
  - Churg strauss disease
  - Giant cell arterirtis
  - PAN
  - Sarcoidosis
  - Crohn's disease
  - SLE, RA, Scleroderma
  - Erdheim chester syndrome
  - Histiocytosis X
  - Idiopathic fibrosclerotic disorders

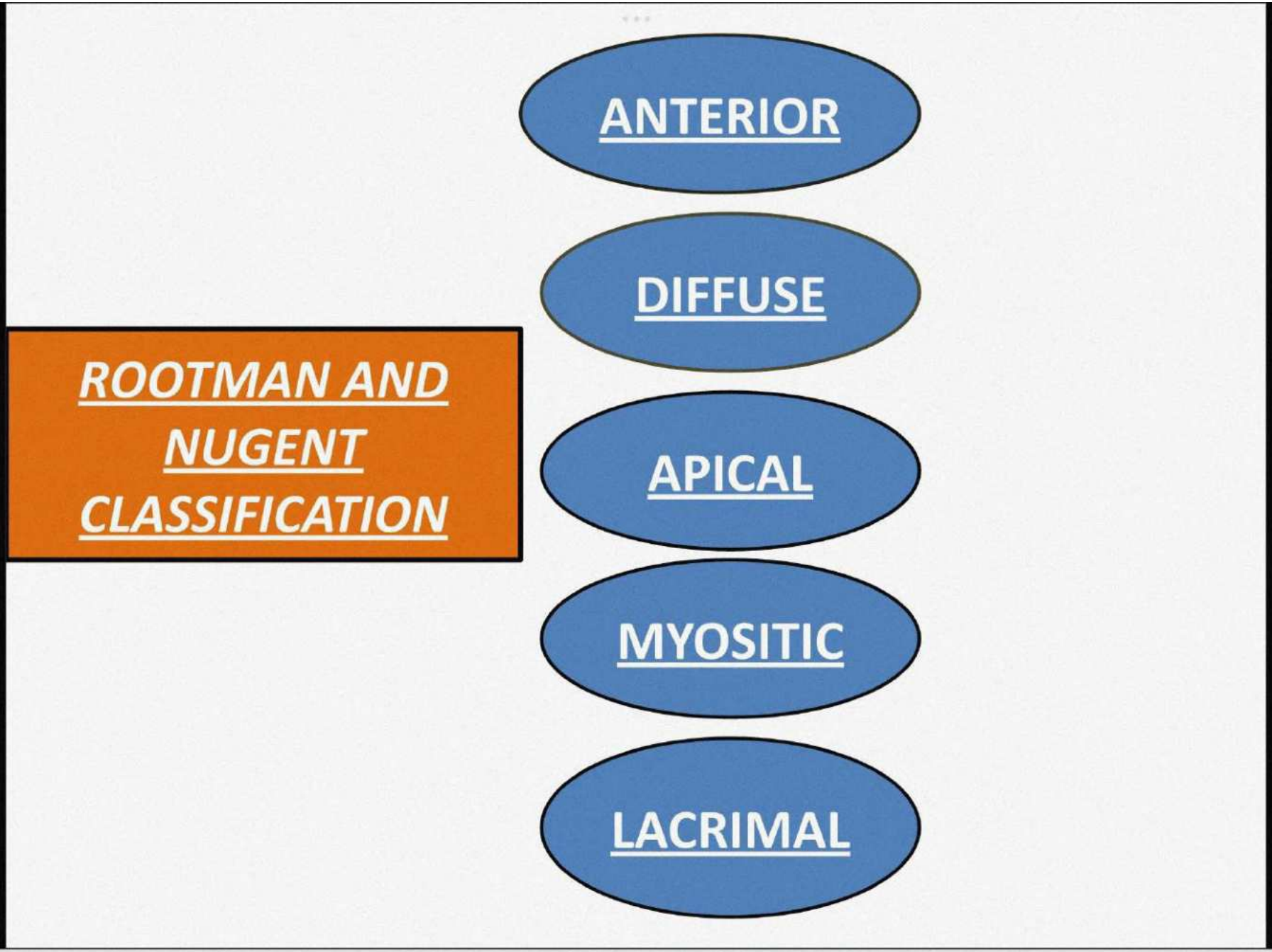
SIGNS

- CONJUNCTVAL CHEMOSIS /CONGESTION
- EYELID SWELLING
- RESTRICTED EOM
- PROPTOSIS
- IMPAIRED VISION
- ↓corneal sensitivity
- Hi IOP
- INTRAOCULAR uveitis/ RD



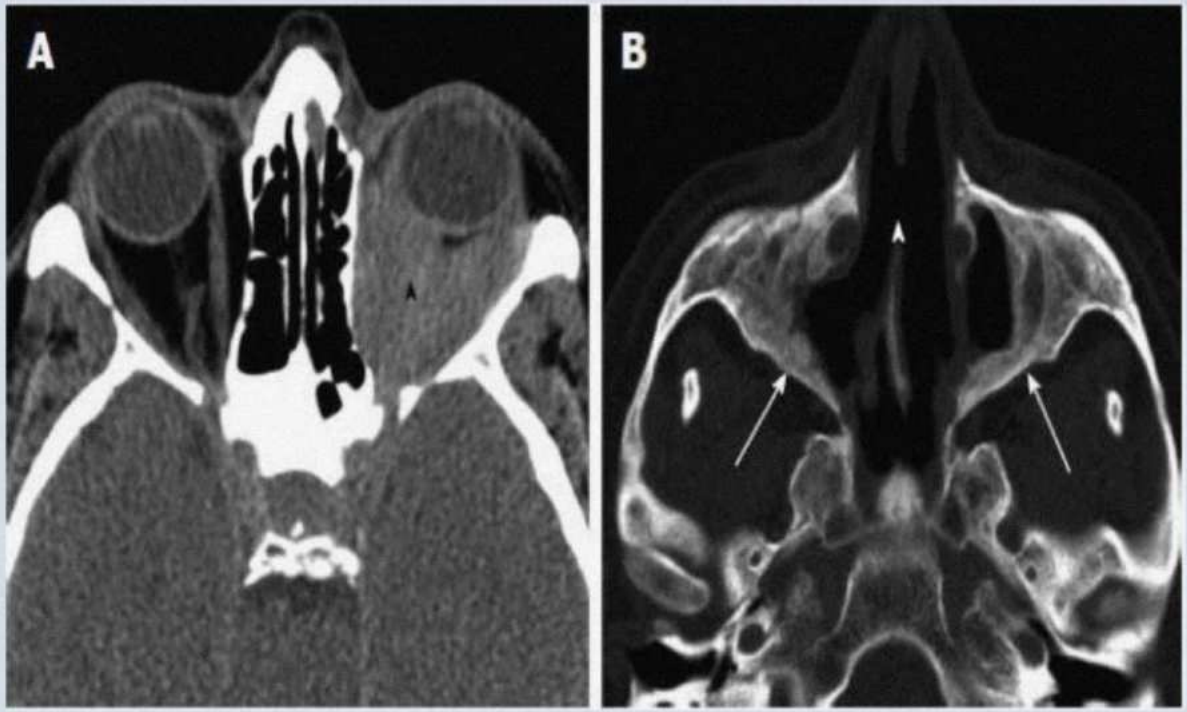






Diffuse OID

- 4-11% of all OID

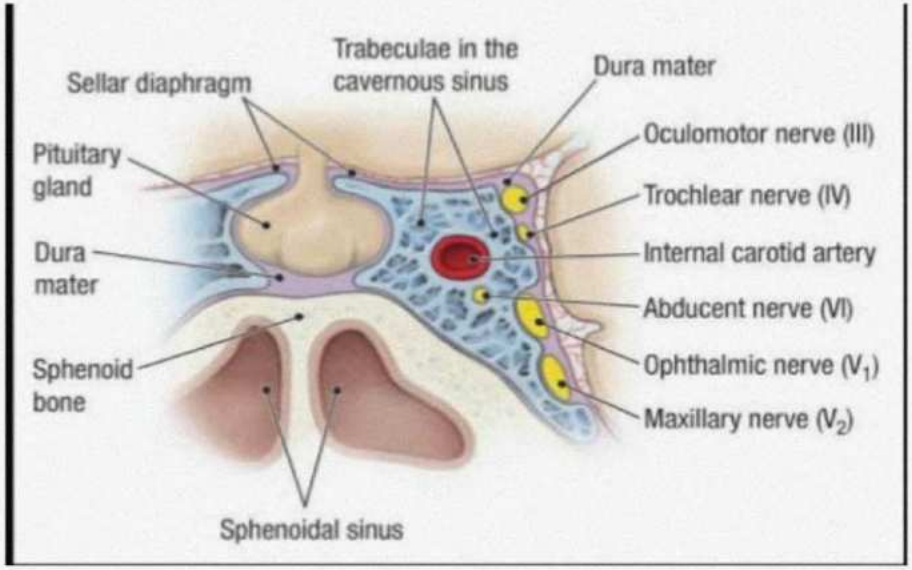


ORBITAL APICITIS

Less common and with poor outcome

Risk of invading ON and extension into Cavernous Sinus

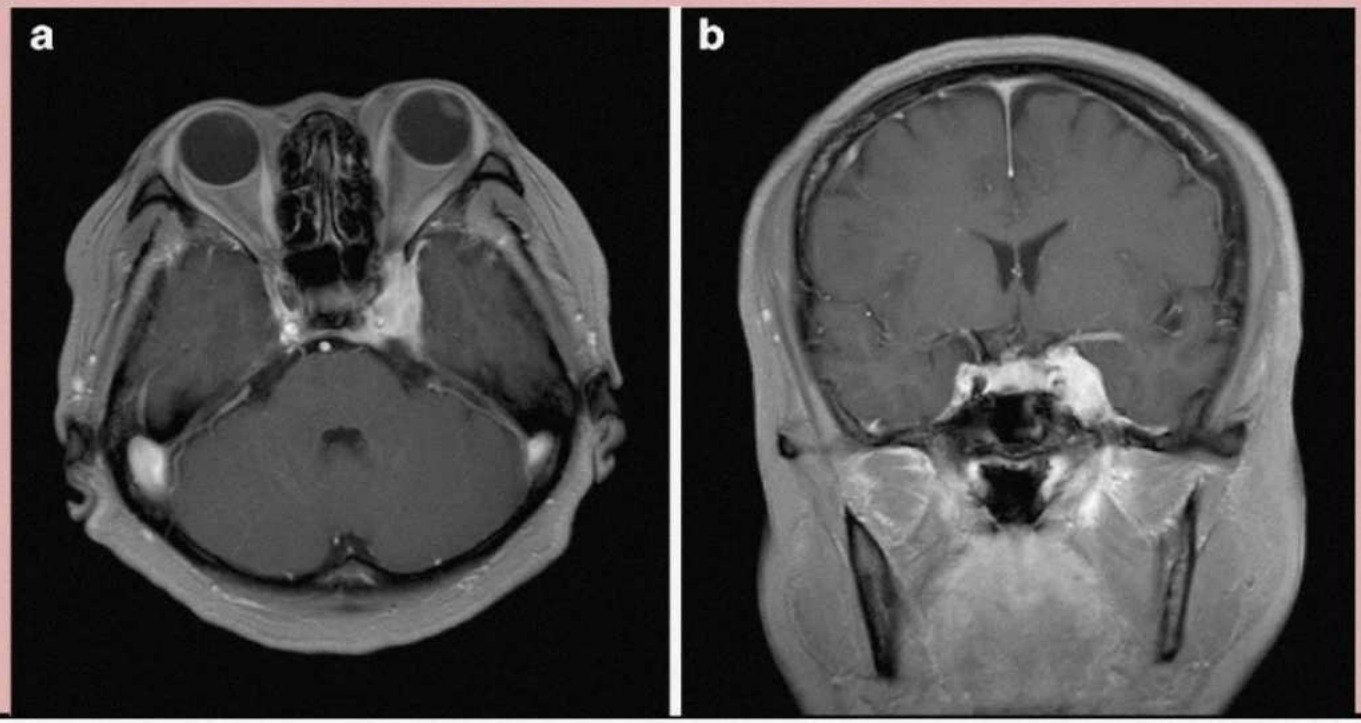
TOLOSA HUNT SYNDROME is inflammation of cavernous sinus with remitting relapsing orbital pain and invol of 3rd 4th 5th and 6th CNs



T1 MRI shows intermediate intensity as inflammatory tissue replaces the normal high intensity fat at the orbital apex

T2 MRI shows low intensity with darker signal indicating high degree of fibrosis

CNS involvement include abnormal soft tissue extension into the MCF, expansion of the ipsilateral cavernous sinus walls and post gadolinium enhancement of the meninges and dura



Corticosteroids

First-line therapy

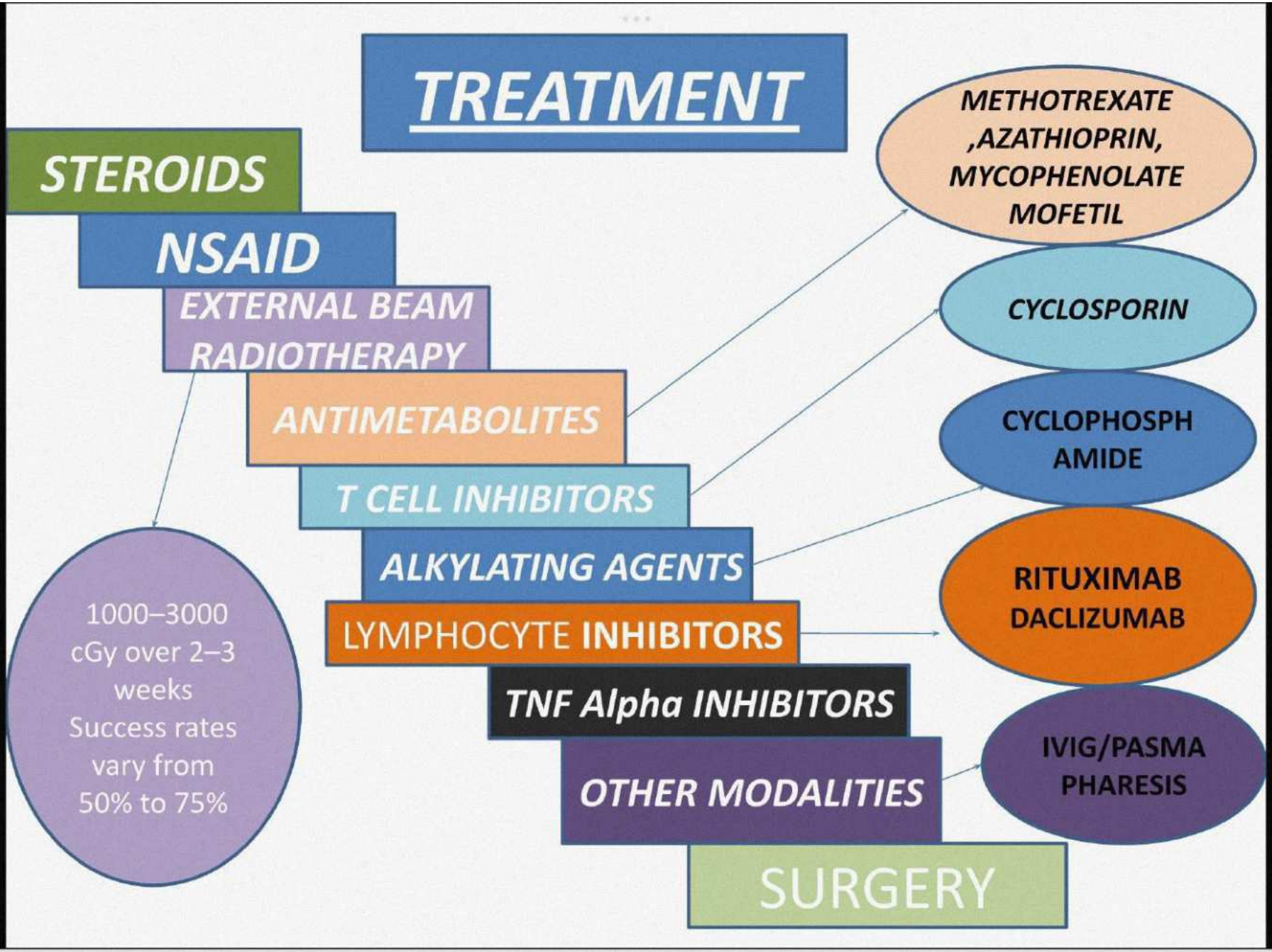
Anti-inflammatory

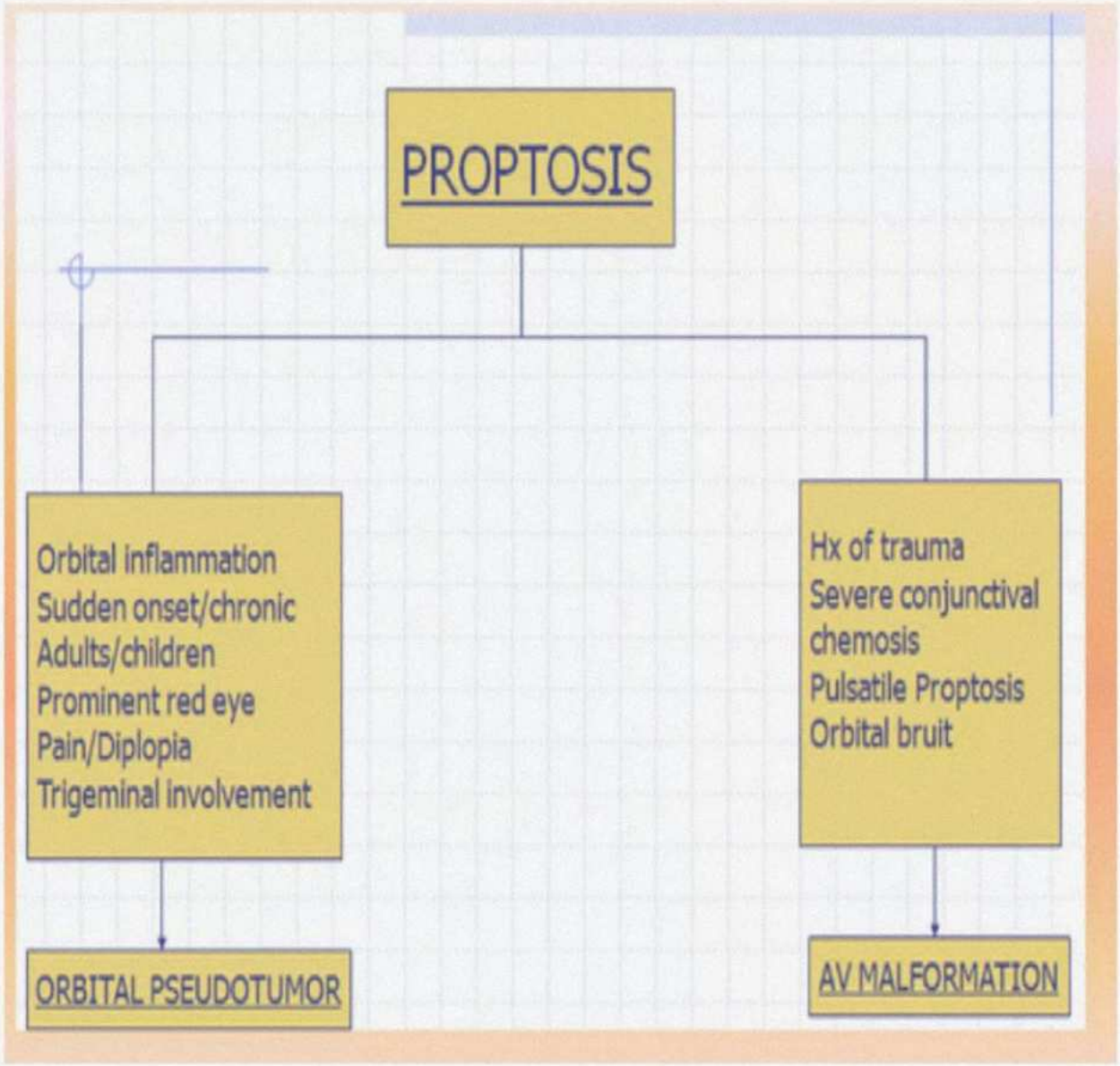
(inhibition of Phospholipase A2 and Cyclooxygenase pathways)

Immunosuppressive **(inhibition of IL and IFN synthesis, inhibition of major histocompatibility antigen expression, and cytotoxic effect on T lymphocytes)**

Over 75% of patients show dramatic improvement

Starting dose of 1 mg/kg/day of Prednisone
with a slow taper over 6–8 weeks


TREATMENT
STEROIDS
NSAID
EXTERNAL BEAM RADIOTHERAPY
ANTIMETABOLITES
T CELL INHIBITORS
ALKYLATING AGENTS
LYMPHOCYTE INHIBITORS
TNF Alpha INHIBITORS
OTHER MODALITIES
SURGERY
1000–3000 cGy over 2–3 weeks Success rates vary from 50% to 75%
METHOTREXATE ,AZATHIOPRIN, MYCOPHENOLATE MOFETIL
CYCLOSPORIN
CYCLOPHOSPHAMIDE
RITUXIMAB DACLIZUMAB
IVIG/PASMA PHARESIS


PROPTOSIS
Orbital inflammation
Sudden onset/chronic
Adults/children
Prominent red eye
Pain/Diplopia
Trigeminal involvement
Hx of trauma
Severe conjunctival chemosis
Pulsatile Proptosis
Orbital bruit
ORBITAL PSEUDOTUMOR
AV MALFORMATION

Neoplastic Disorders

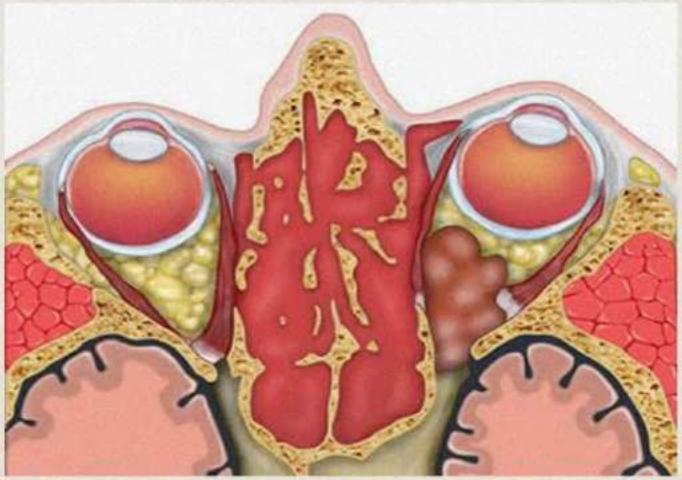
- Orbital lymphoma
- Lacrimal gland tumors
- Neural tumors
- Metastasis

Orbital lymphoma

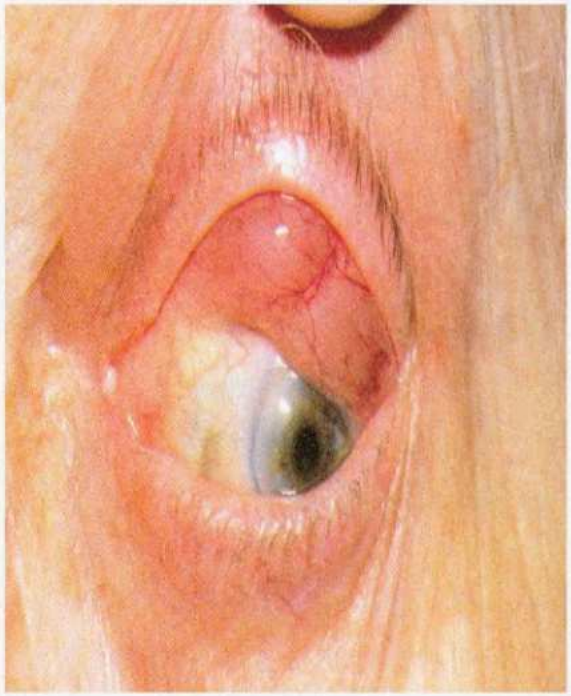
- **lymphoma occurring in the conjunctiva, lacrimal gland, eyelid and ocular musculature**
- Primary non-Hodgkin's lymphoma (NHL) of the orbit is a rare presentation with an indolent course

• ***ETIOLOGY***

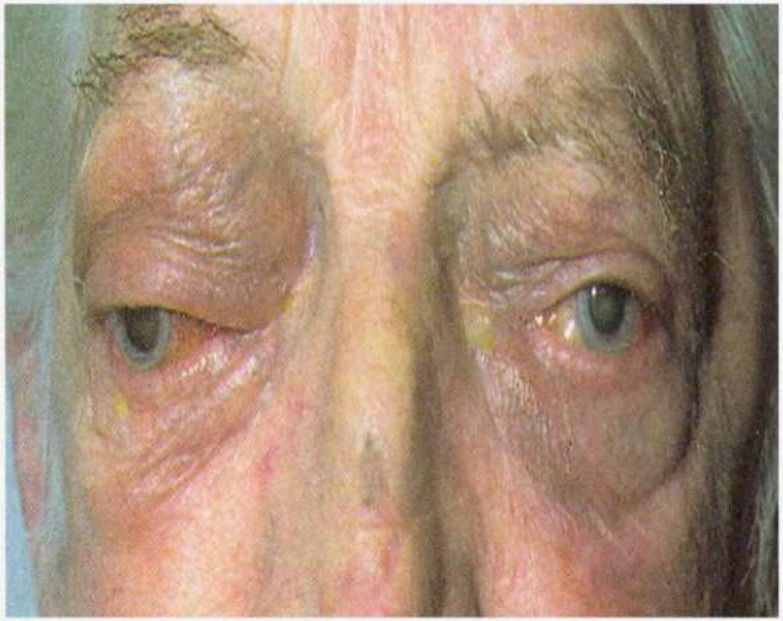
- **Increasing age, immunosuppressive drugs, or autoimmune disorders such as rheumatoid arthritis, lupus, anemia, and HI**
- **highly curable** with RT



ANTERIRO ORBITAL LYMPHOMA



BILATERAL ORBITAL LYMPHOMA



Lacrimal Gland Tumors

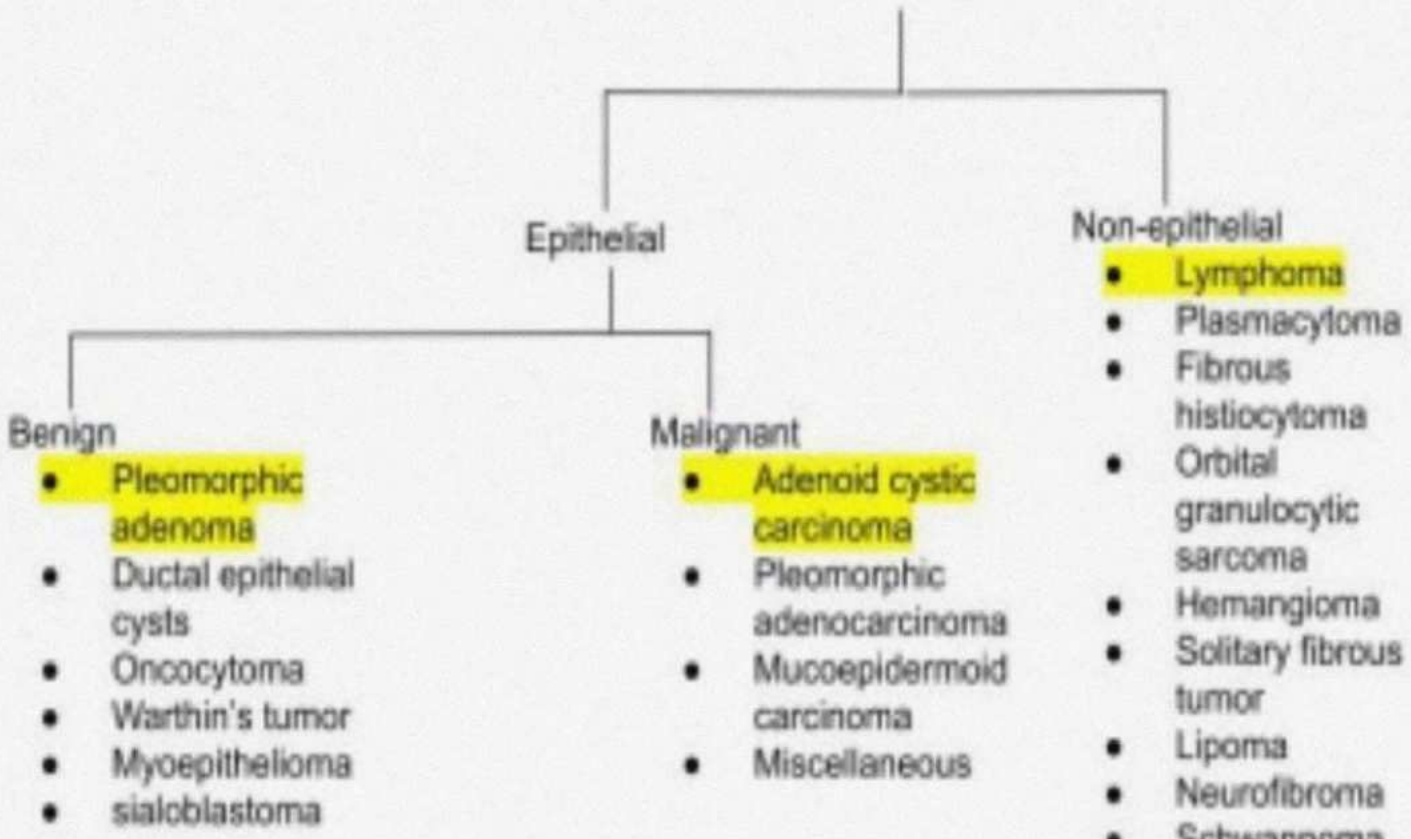
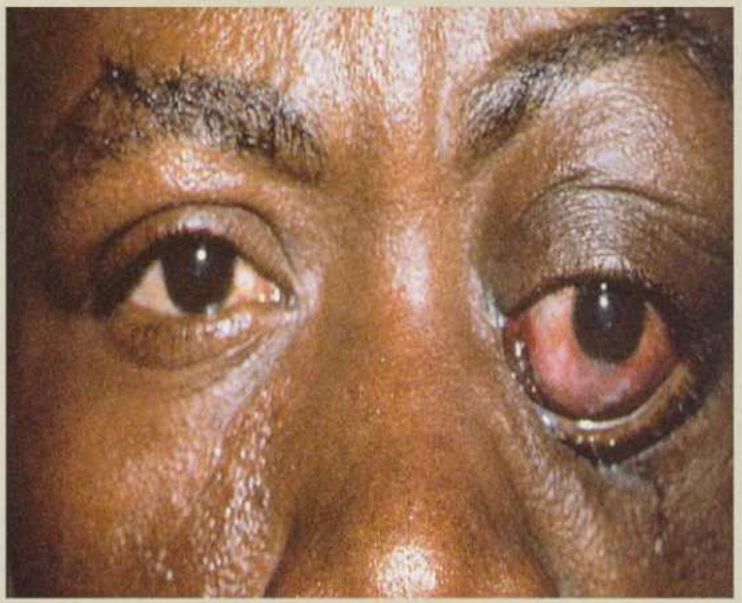
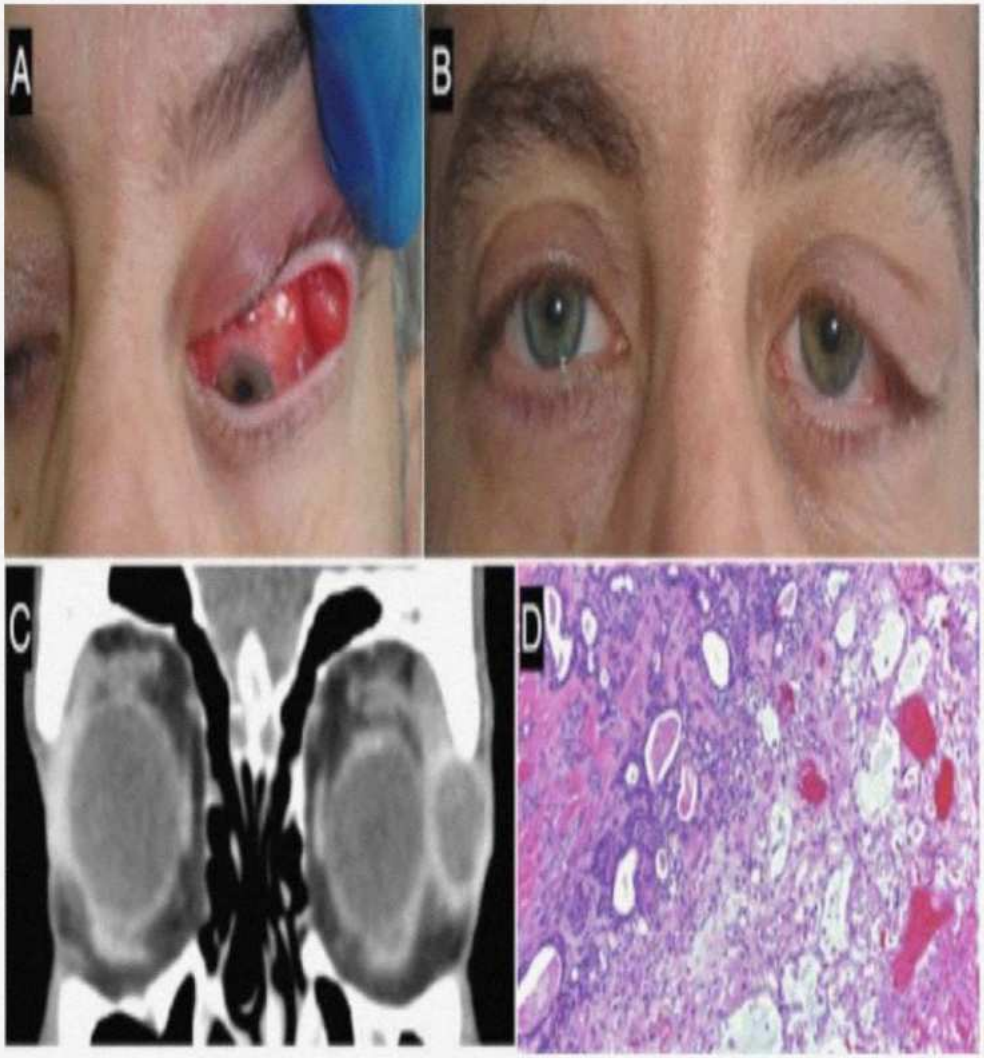


Image created by Hiten Doshi

Pleomorphic adenoma

- **Most common epithelial tumor of the lacrimal gland from ducts, stroma and myoepithelial cells**
- **5^{th} decade of life**
- **Painless slow growing lesion in the upper outer quadrant for ≥ 1 year**



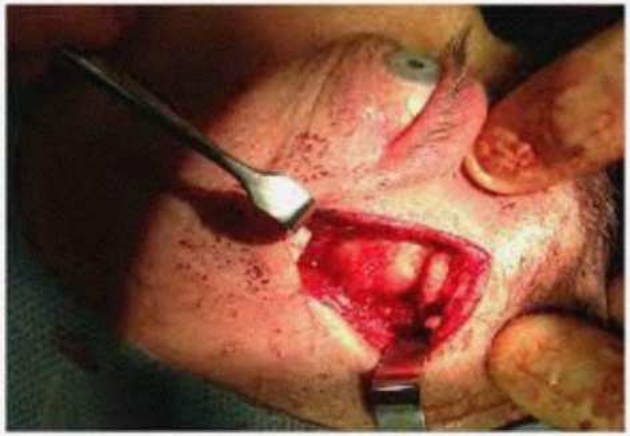

A
B
C
D

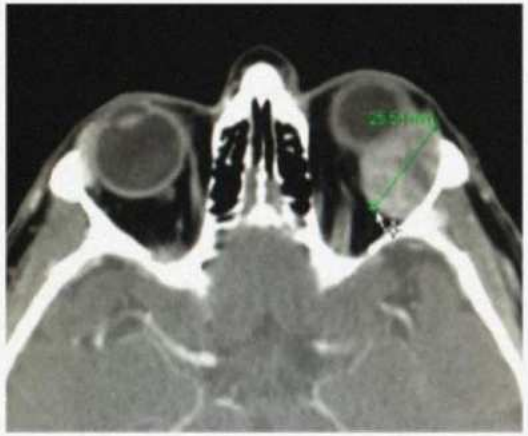
- Smooth,firm,non tender mass in the lacrimal gland fossa
- Tends to extend backwards may cause proptosis opthalmoplegia and choroidal folds

CT scan –round or oval smooth outline with excavation of the lacrimal gland fossa without destruction

Treatment .....complete excision without capsule disruption

Incomplete excision .....recurrence and malignant transformation



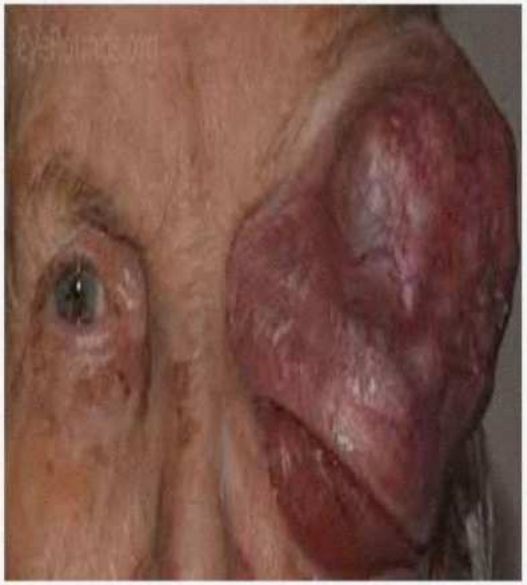


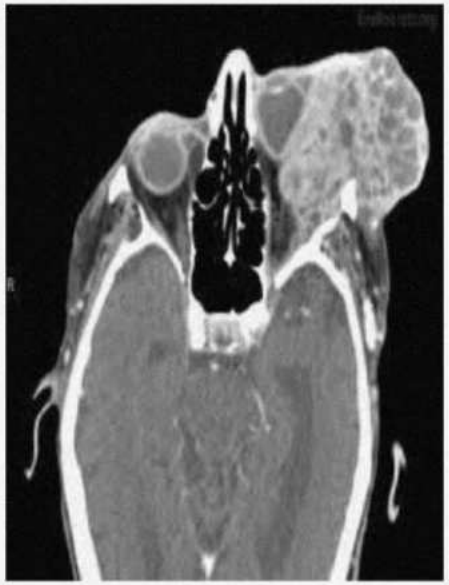
Lacrimal gland carcinoma

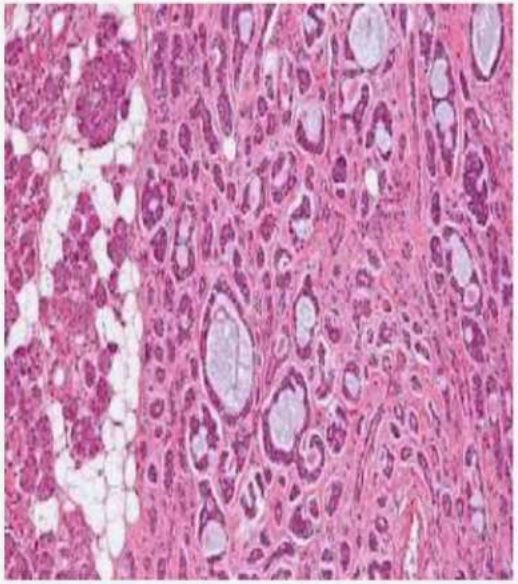
- Rare ,high morbidity and mortality
- 4^{th} and 6^{th} decade of life with shorter history

Histologic types

- **Adenoid cyst carcinoma**
- **Pleomorphic adenocarcinoma adenocarcinoma**
- **Mucoepidermoid carcinoma**
- **Squamous cell carcinoma**

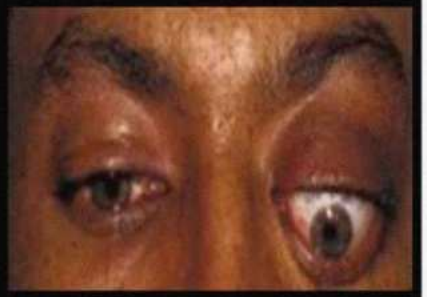


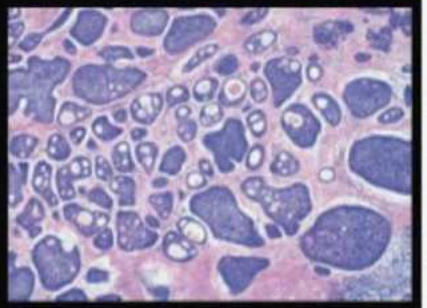




ADENOID CYSTIC CARCINOMA OF THE LACRIMAL GLAND

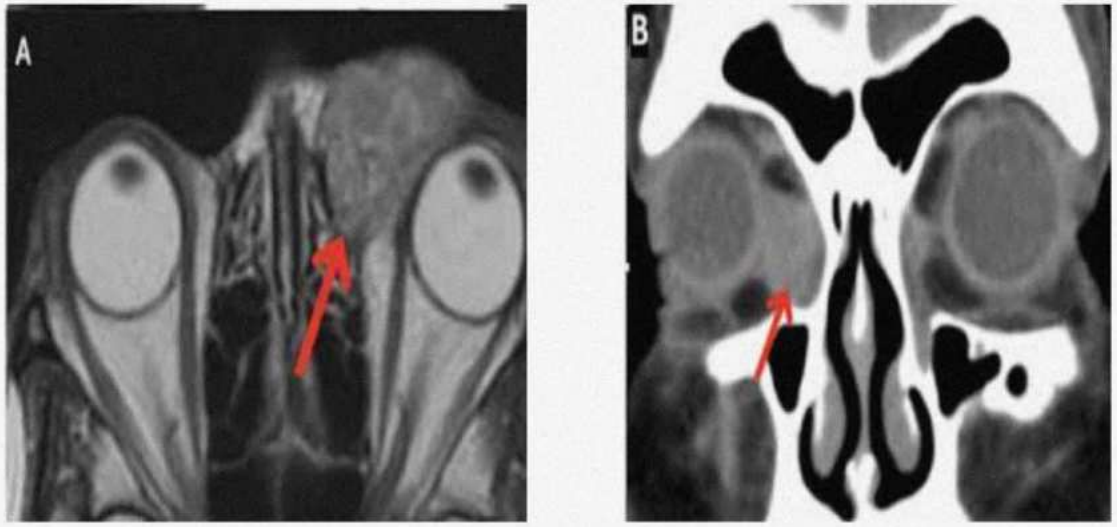
- Most common malignant epithelial tumour of lacrimal gland.
- Presents in young or middle-aged adults , slight predilection in women.
- SYMPTOMS – Proptosis , pain due to perineural invasion is characteristic.
- SIGNS - Inferior and nasal displacement of globe, hypoaesthesia in the region supplied by lacrimal nerve, optic disc swelling and choroidal folds. Extension of the tumour posteriorly with involvement of the superior orbital fissure may cause epibulbar congestion, proptosis, periorbital oedema and ophthalmoplegia.
- INVESTIGATIONS - CT ORBIT - Ovoid soft tissue mass in the superotemporal quadrant of orbit with irregular margins and bony erosion . Focal calcifications may also be present.
- HPE – The histological patterns seen on biopsy include - cribriform , basaloid, sclerosing, comedocarcinoma and tubular. The cribriform pattern is most common and consists of lobules of tightly packed basaloid cells with intervening circular pools of mucin – swiss-cheese pattern.
- TREATMENT – Surgical resection of tumour with postoperative radiotherapy. For advanced tumors with high risk of recurrence - neoadjuvant intraarterial chemotherapy with cisplatin and doxorubicin used with exenteration and irradiation.
- Mortality is due to intracranial spread as a result of perineural invasion and pulmonary metastasis.





Dr.Harinikrishna B
Aravind eye hospital
Madurai

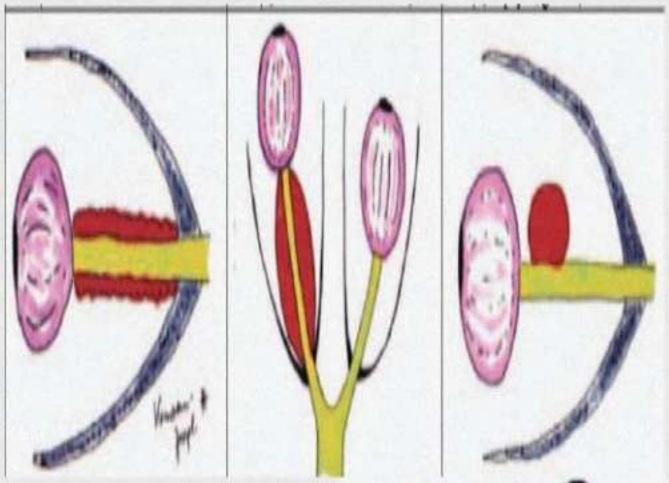
LACRIMAL SAC TUMORS



Classification

- **Primary Neural Tumors**

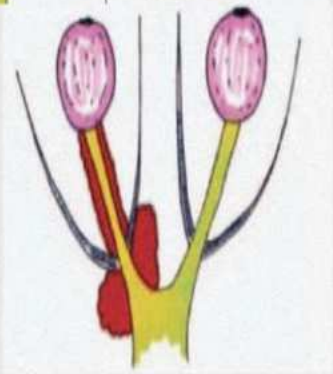
  Optic nerve
  Peripheral nerves



- **Secondary Neural Tumors**

Metastatic and infiltrative neoplasms
Breast cancer, prostate cancer, melanoma, lung cancer

ON may be affected by peripheral nerve tumors in the orbit



ON sheath Meningioma

- Benign tumors
- Meningothelial cells of the arachnoid layer within the optic nerve sheath
- Mean age - 41 to 48 years
- Higher incidence in females (61%)
- Unilateral (95%)
- Neurofibromatosis type 2 (NF-2) has an incidence of 9%, present at younger age, may be bilateral

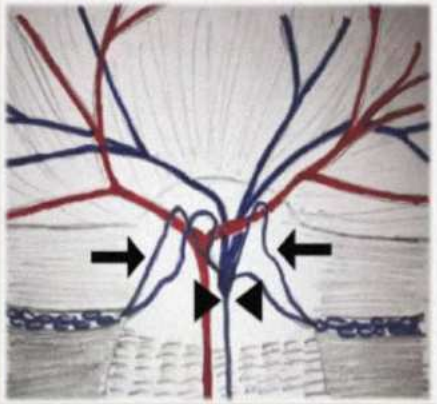
- Intracranial Meningiomas of sphenoid wing invade the orbit

CLINICAL FEATURES

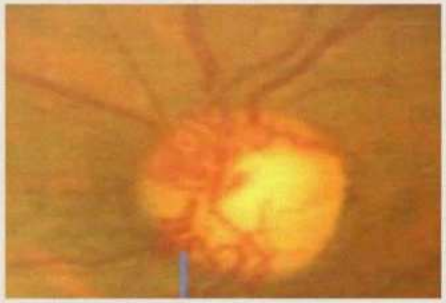
- **Visual loss**

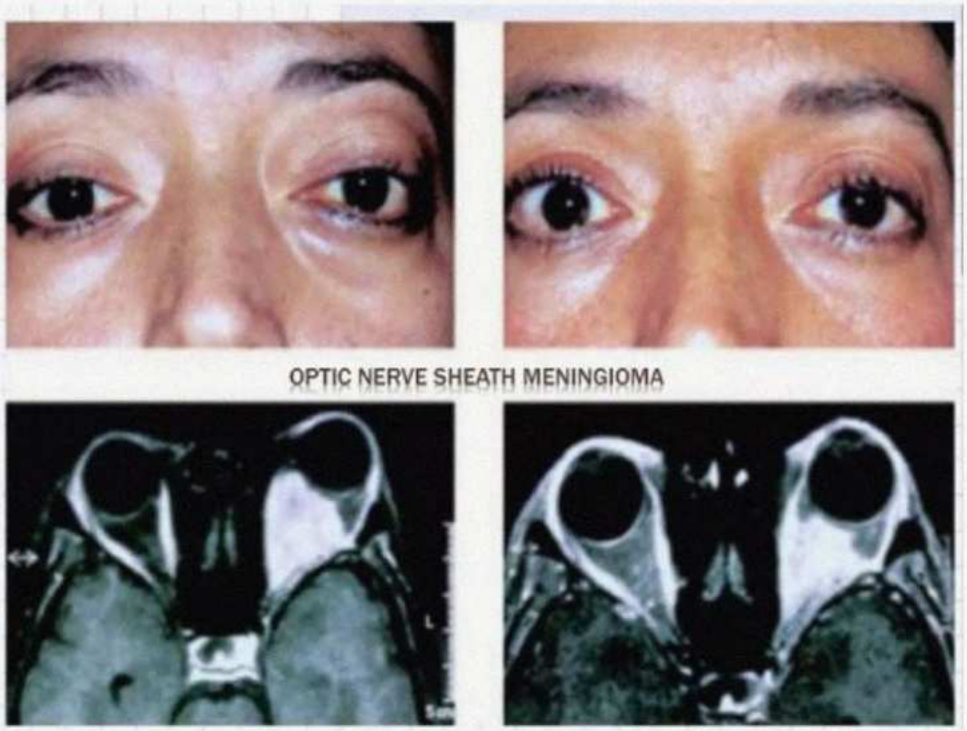
  gradual, painless, and usually 1 to 5 years before presentation
- **Proptosis**

  rarely the initial symptom, (59%) upto 2 to 5 mm
- **Chronic optic disc swelling**
- Longstanding CRVC - optociliary shunt vessels
- Optic atrophy



classic triad of gradual visual loss, optic atrophy, and optociliary shunt vessels is consistent for optic nerve sheath meningioma





OPTIC NERVE SHEATH MENINGIOMA

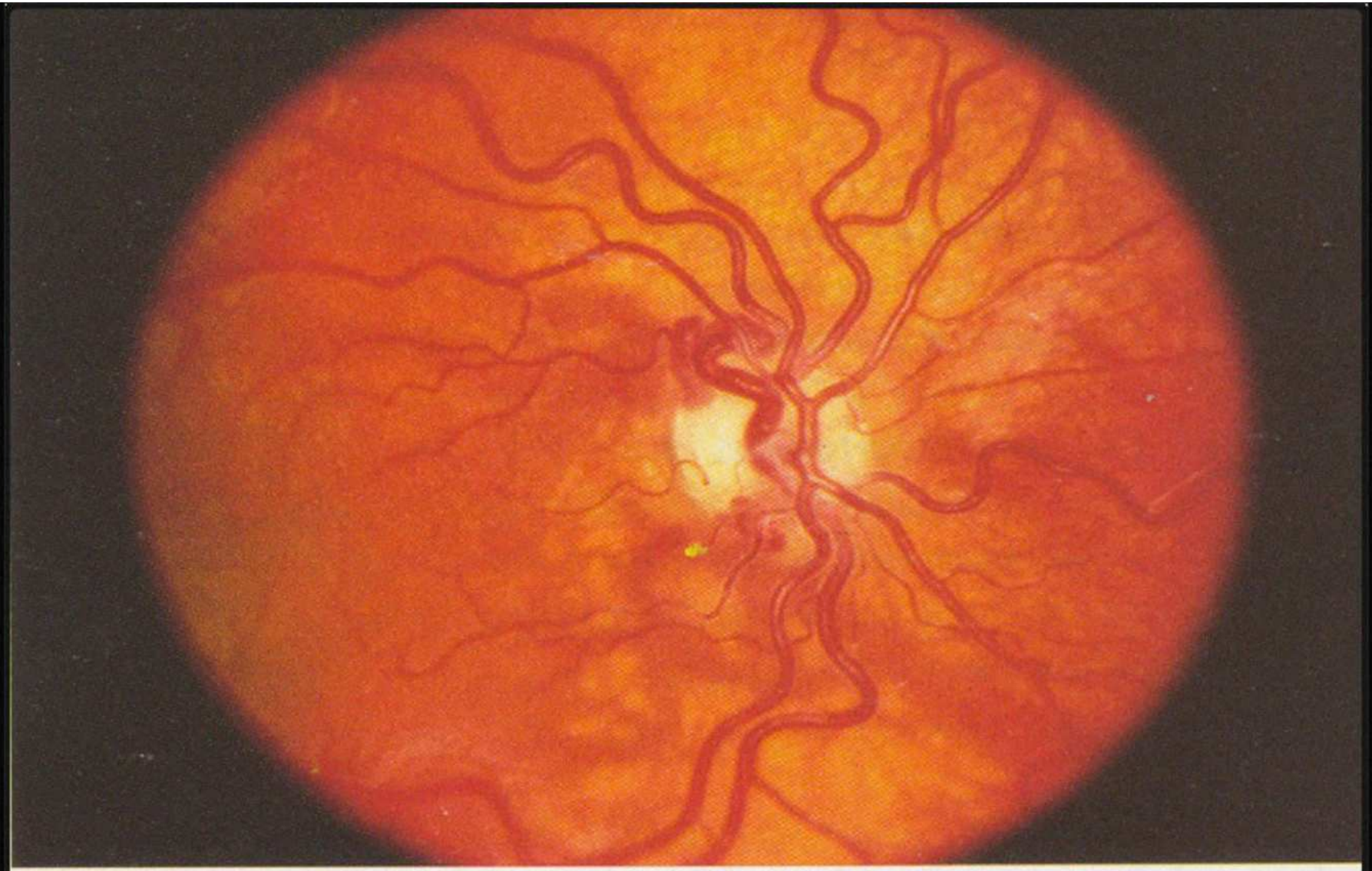


Figure 14.10 Optociliary shunt vessels

Diagnostic Approaches

CT SCAN

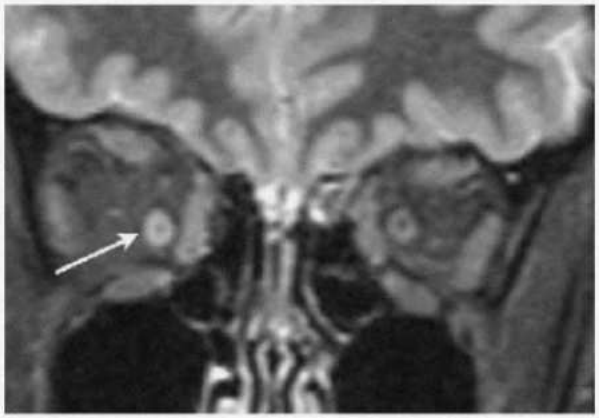
- ***Tram-track" signal*** An enlarged optic nerve with increased peripheral intensity and decreased central intensity"
- calcifications within the optic nerve sheath

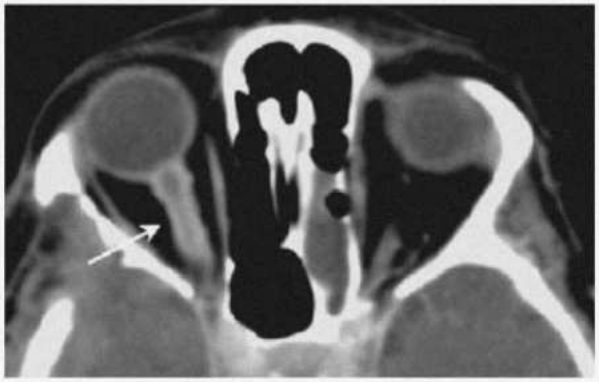
MRI most sensitive and specific imaging modality

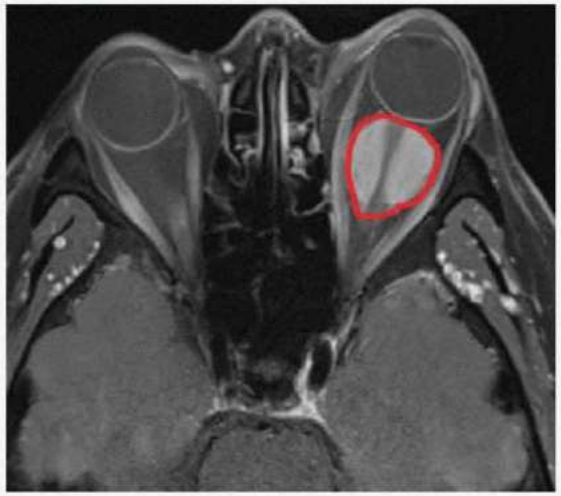
- T1-weighted MRI with contrast and fat suppression outlines the true anatomic borders of the optic nerve
- Homogenous enhancement of the lesion with gadolinium, though the optic nerve itself does not enhance

Biopsy

- often not needed except, if the patient has atypical sudden or rapidly progressive visual loss



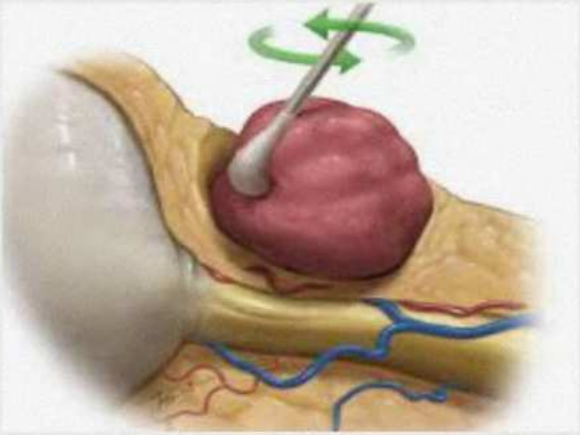




TRAM-TRACK Signal

SURGICAL EXCISION

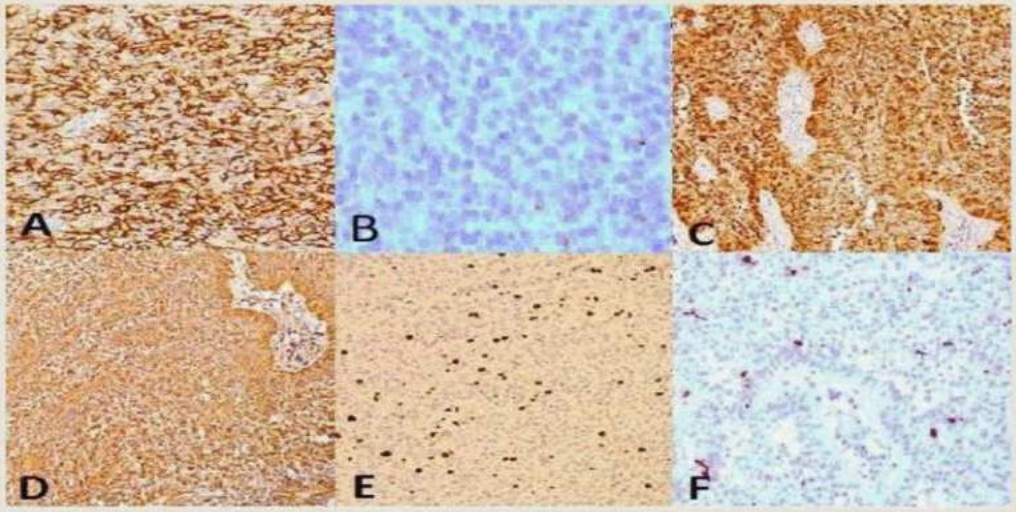
- **Progressive visual loss**
- **Disfiguring proptosis**
- **To relieve pain**
- **in children with more aggressive tumors**



- likelihood of blindness / damage to the pial vessels shared by the ON and the meningioma

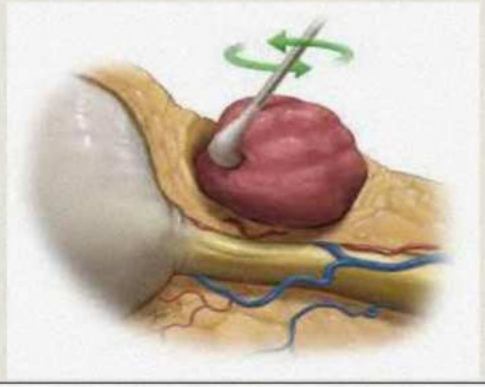
Cellular morphology
Immunohistochemistry

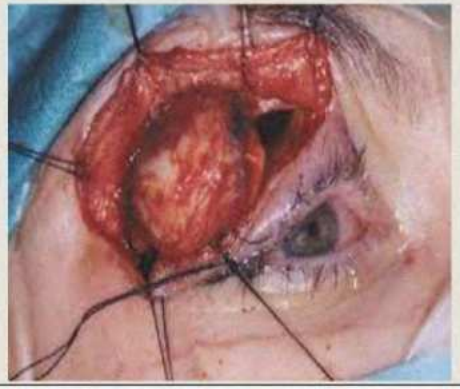
- Meningiomas are positive for EMA and S-100
- Gliomas are positive for GFAP and negative for S-100 and EMA
- Peripheral nerve sheath tumors contain Schwann cells that stain positively for S-100
- Perineural cells may also be positive for EMA
- Granular cell tumors stain positively for S-100 and may be positive for CD68 and leu-7

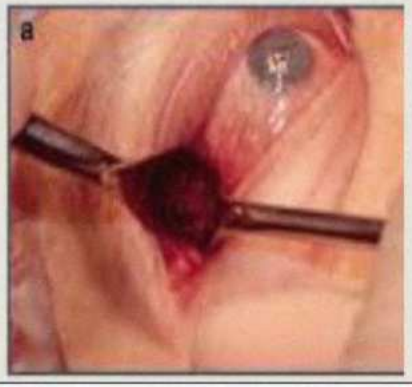


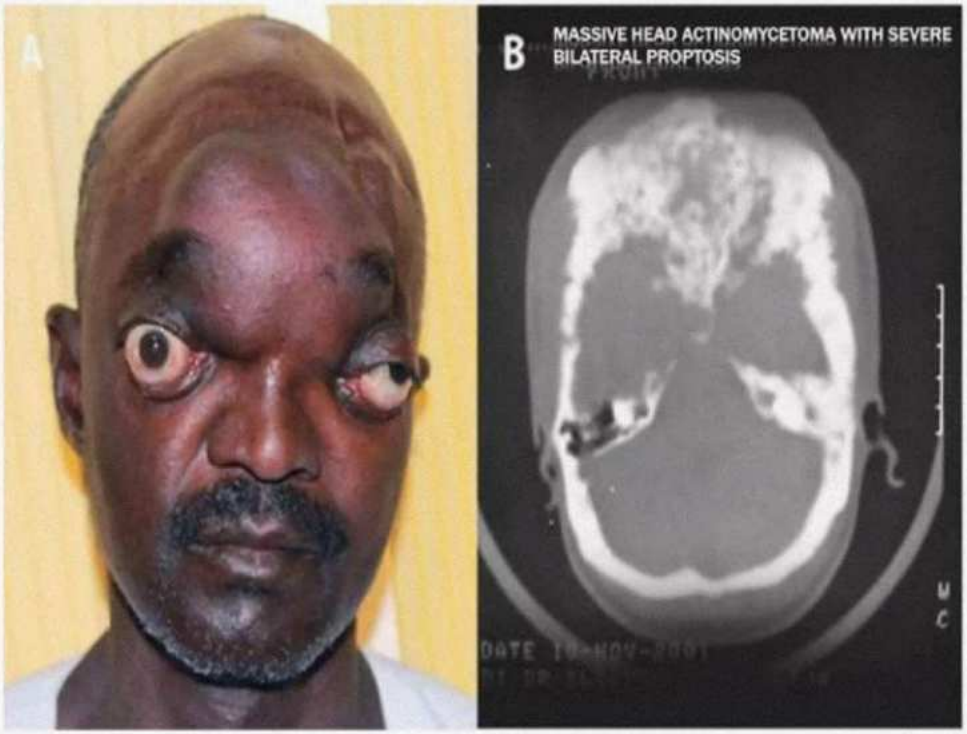
Treatment Options

- Observation
- Radiotherapy
- Chemotherapy
- Surgical excision



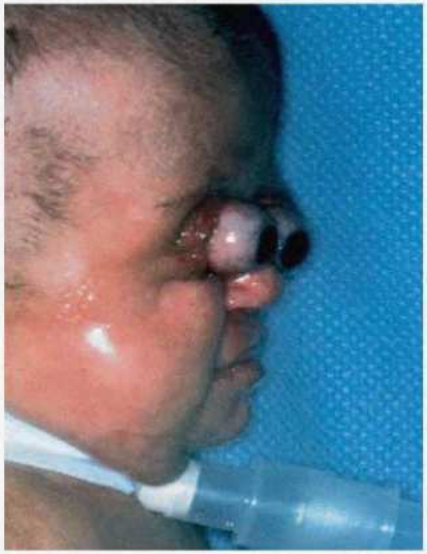


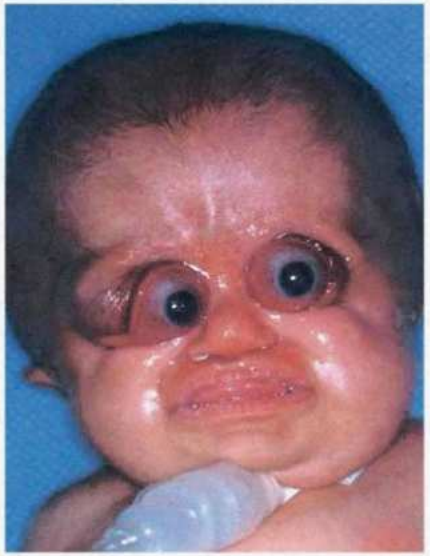



A
B
MASSIVE HEAD ACTINOMYCETOMA WITH SEVERE BILATERAL PROPTOSIS

Congenital malformations

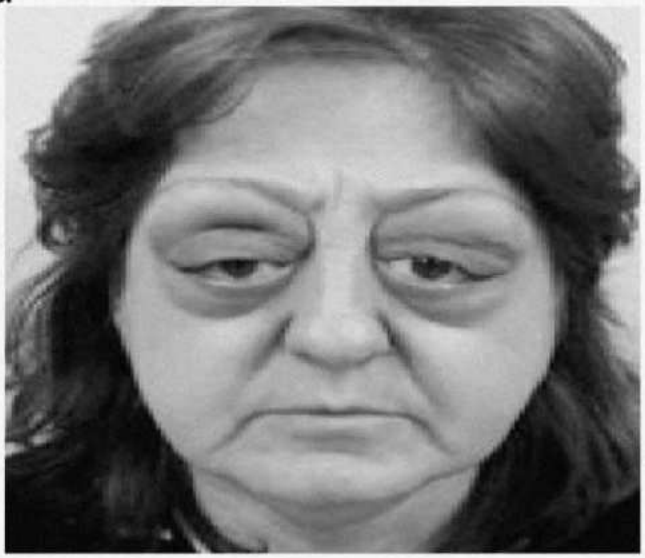
- ***CRANIOFACIAL SYNOSTOSIS***





Mikulicz syndrome

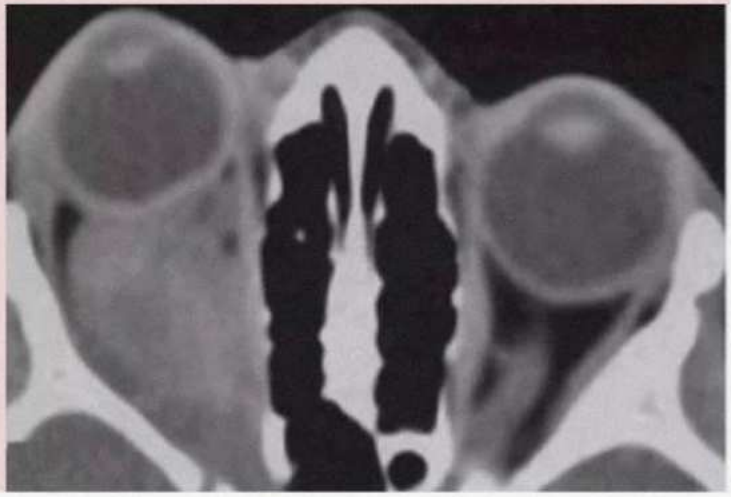
- **Chronic autoimmune disease in which the glandular tissue of the head and neck are excessively enlarged, usually bilaterally**. Mostly, the salivary (parotid) and lacrimal (tear-duct) glands are affected



AXIAL PROPTOSIS

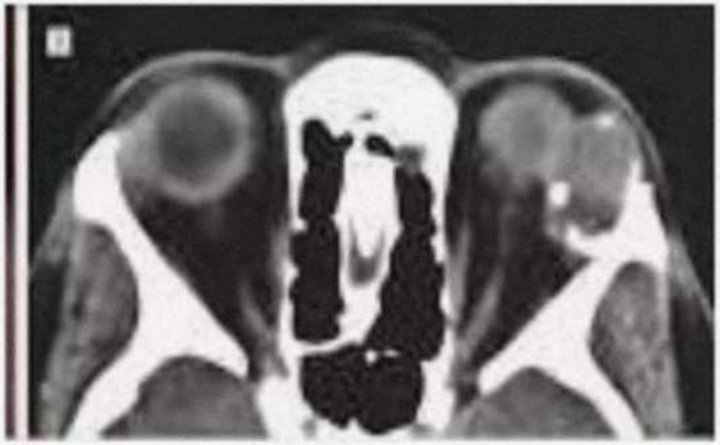
- ***Lesions of intra conal space arising from the optic nerve and central space***

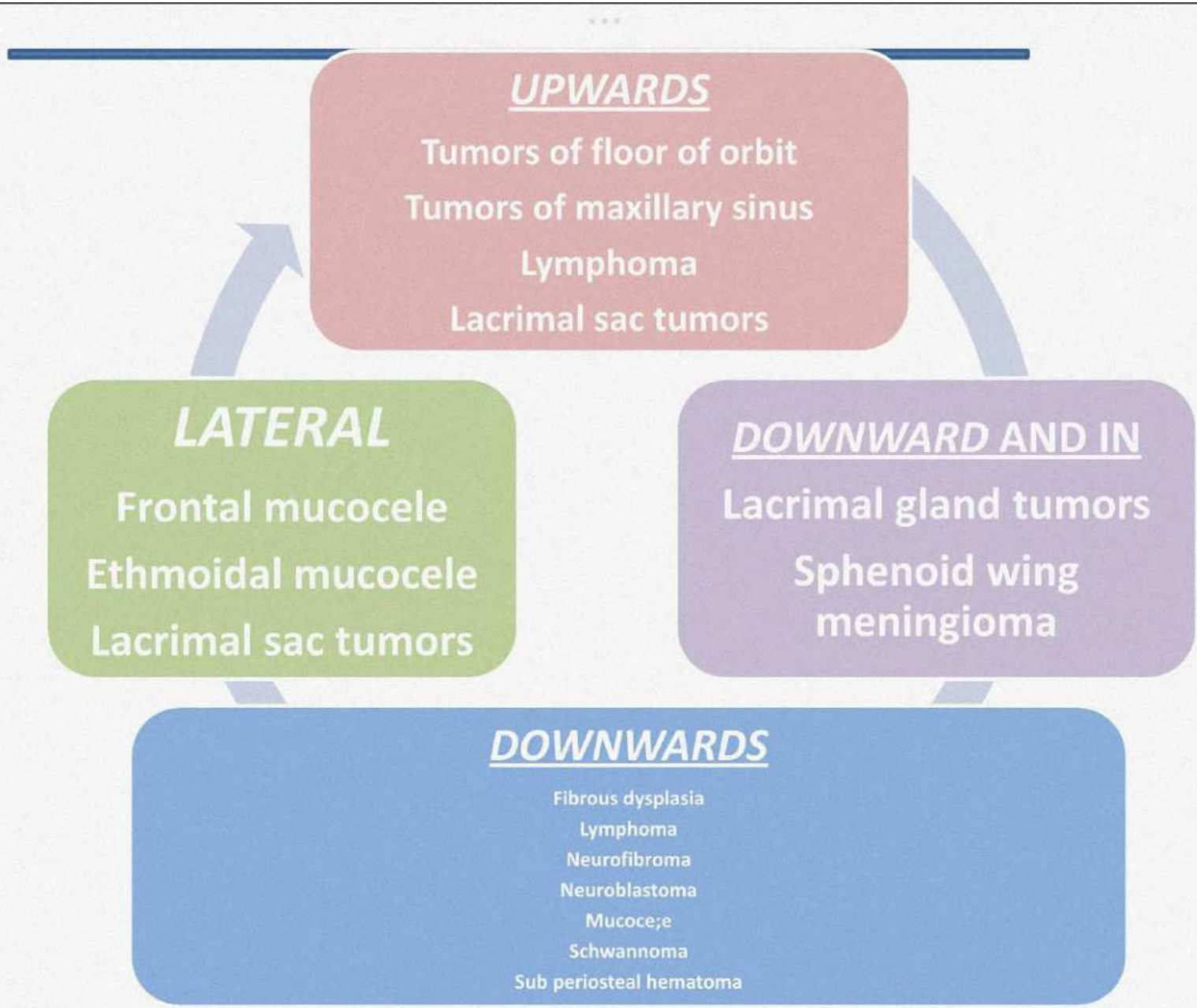
Optic nerve Glioma
Optic nerve sheath Meningioma
Cavernous hemangioms
Schwannoma
Neurofibroma
Orbital varices
Hydatid cyst



NON AXIAL PROPTOSIS

Proptosis caused by any extraconal lesion or fracture displacement of orbital bones protruding inwardly.

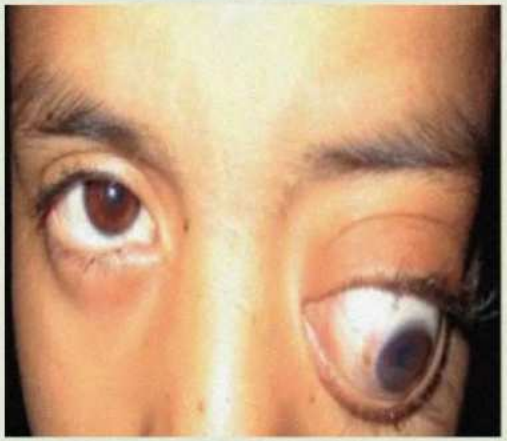


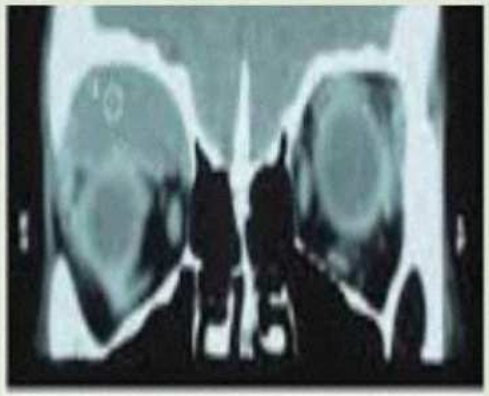


NON AXIAL PROPTOSIS

- ***Downward Displacement***

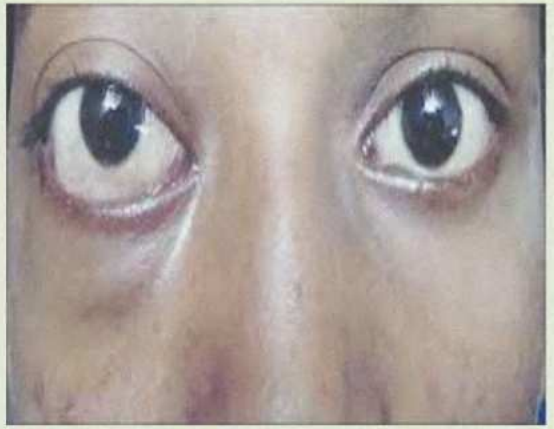
Fibrous dysplasia
Frontal mucocele
Subperiosteal hematoma
lymphoma
Neurofibroma
Neuroblastoma
Schwannoma
Thyroid Orbitopathy





- ***Upward displacement of the Globe***

  lacrimal sac tumor
  lymphoma
  Metastatic tumor
  Maxillary sinus tumor



- ***Lateral displacement of the Globe***

  lacrimal sac tumor
  Rhabdomyosarcoma
  Nasopharyngeal tumors
  Ethmoid mucocele
  Metastatic tumor

COMMON ORBITAL SPACE OCCUPYING LESIONS

ORIGIN	CHILDREN	ADULTS
CONGENITAL	Dermoid cyst Teratoma	
VASCULAR	Capillary haemangioms Lymphangioma	Cavernous haemangioma Orbital varices Haemangiopericytoma
NEURAL	Optic nerve Glioma Plexiform neurofibroma	Optic nerve meningioma Schwanoma Neurofibroma
MESENCHYMAL	AML Rhabdomyosarcoma	Fibrous Histiocytoma
HAEMPOIETIC	Histiocytosis Neuroblastoma	Lymphomas
METASTATIC	Neuroblastoma	Breast/Lung

- Proptosis with white reflex – retinoblastoma
- U/L fast progressing proptosis , fever , toxic child , pain – orbital cellulitis
- U/L axial proptosis with early vision loss – optic nerve glioma
- B/L proptosis , fever and toxemia – cavernous sinus thrombosis
- Pale child , bleeding from gums , U/L or B/L proptosis – leukemia
- U/L proptosis ,pain ,fever,hazy cornea and loss of vision - panophthalmitis

LATERALITY ON INSPECTION:

UNILATERAL PROPTOSIS:

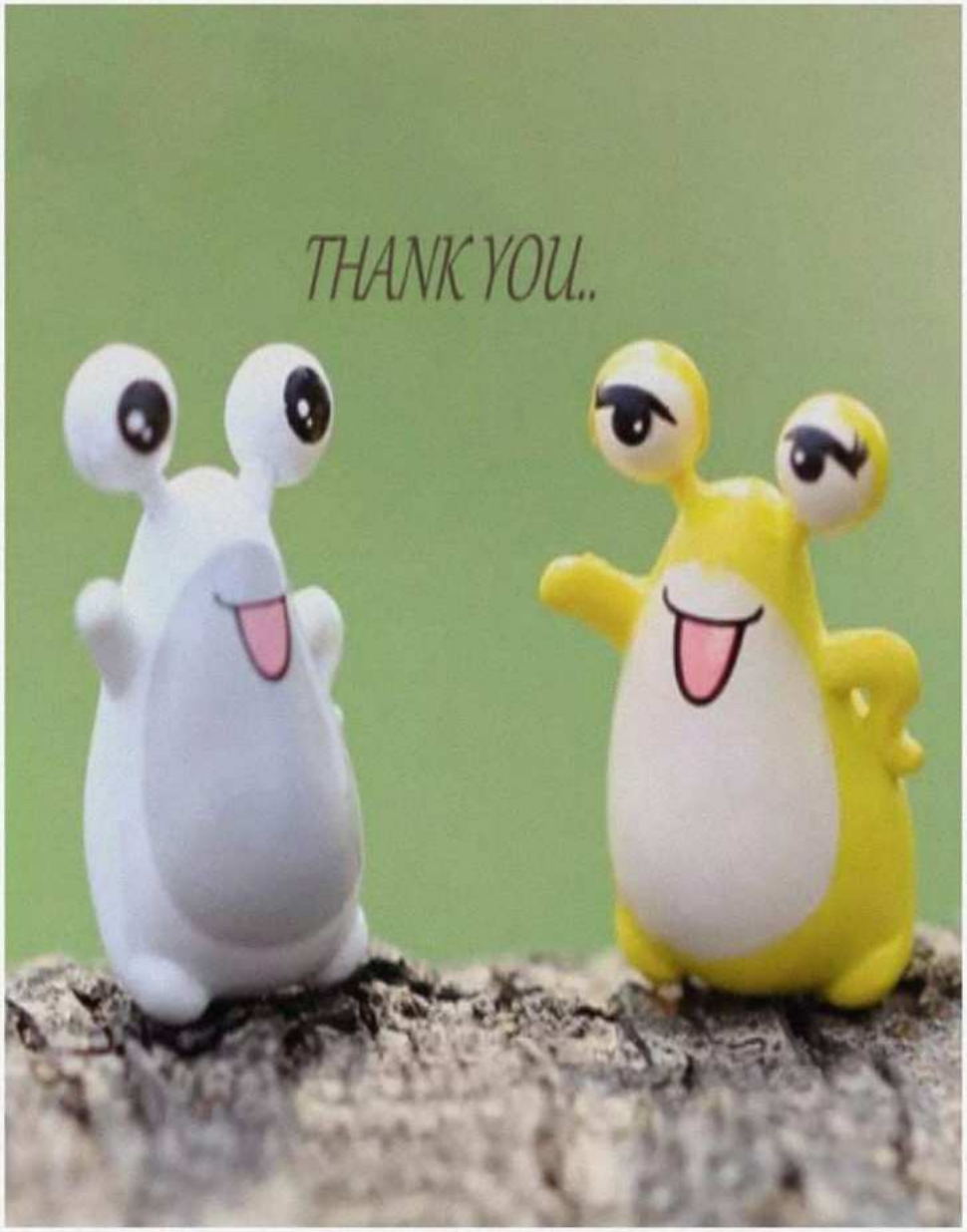
- dermoid cysts,orbital teratoma,congenital cystic eyeball,
- Orbital hemorrhage,traumatic aneurysm,
- Cellulitis/abscess, cavernous sinus thrombosis
- TED,pseudotumour
- Varices,tumors,cysts.

BILATERAL PROPTOSIS:

- craniofacial synostosis,osteitis deformans,rickets
- TED,mickulicz syndrome
- Histiocytosis,amyloidosis,wegener's granulomatosis,
- Tumors.

TAKE HOME MESSAGES

- *The most common cause of bilateral/unilateral proptosis is GRAVES DISEASE*
- *Acute unilateral Proptosis suggests infection or vascular disorder (hemorrhage/CCF/CST)*
- *Chronic unilateral proptosis suggest tumor*
- *Role of imaging*
- *Care of exposed cornea*



PROPTOSIS

Prof SofiaIqbal
FRCS, MRCOphth
Fellowship Orbit/Oculoplastics
Fellowship Refractive surgery

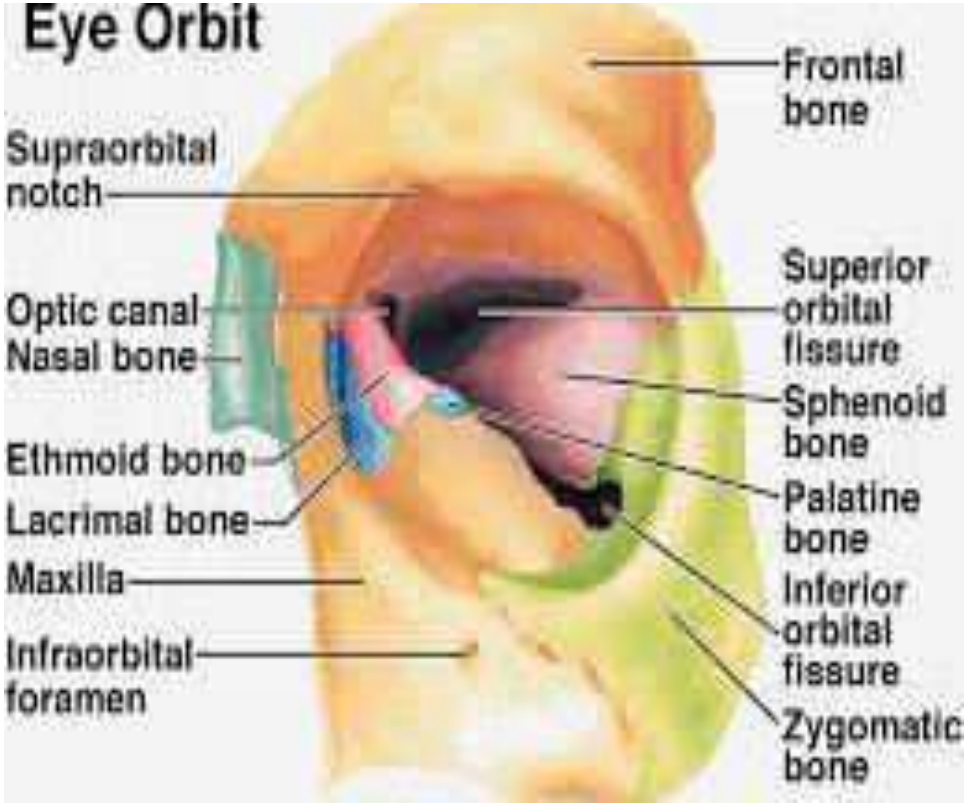
PRESENTATION LAY OUT

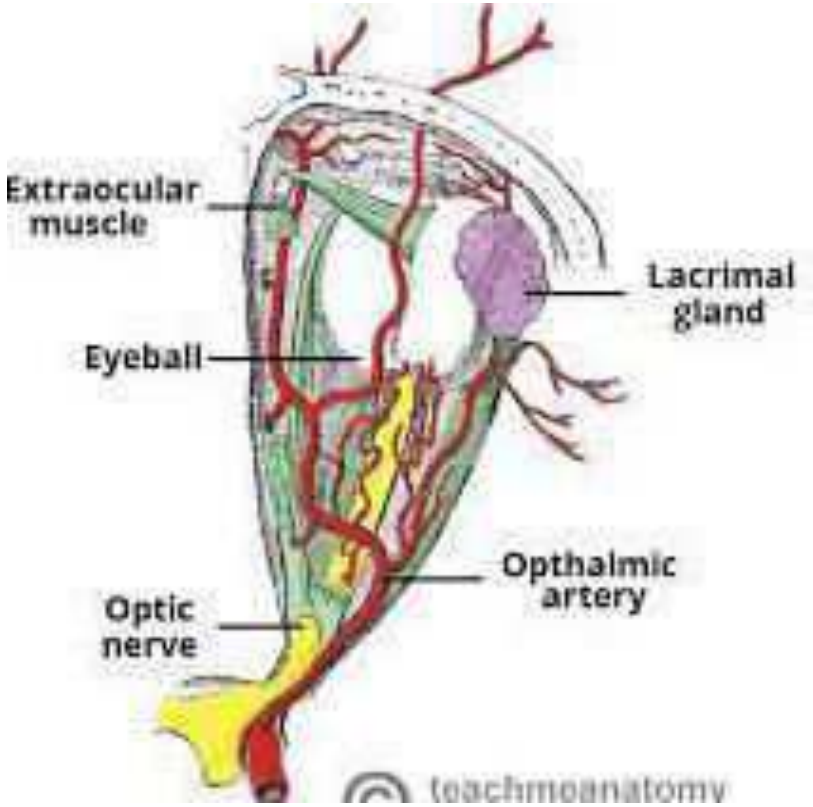
- Anatomy
- Definition
- Presenting features

Approach to a patient with Proptosis

History
Ocular/systemic examination
Local examination
Measurements
Lab investigations
Imaging
Histopathology
Conclusion

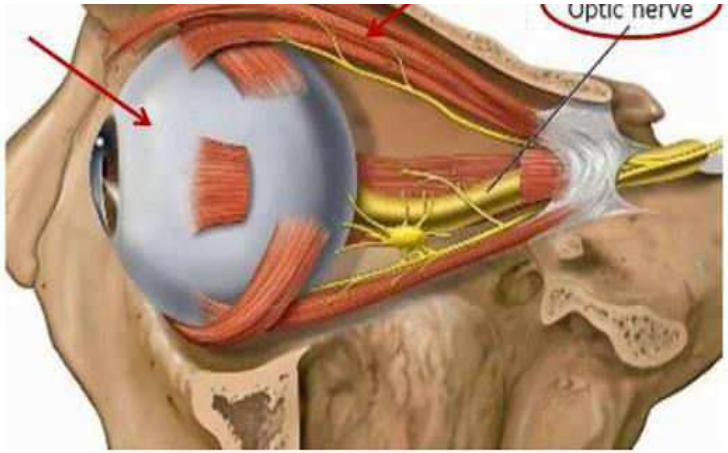
ANATOMY OF THE ORBIT

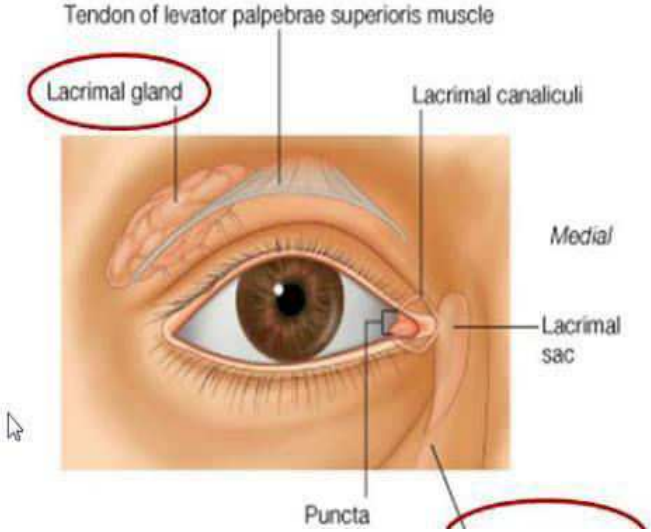


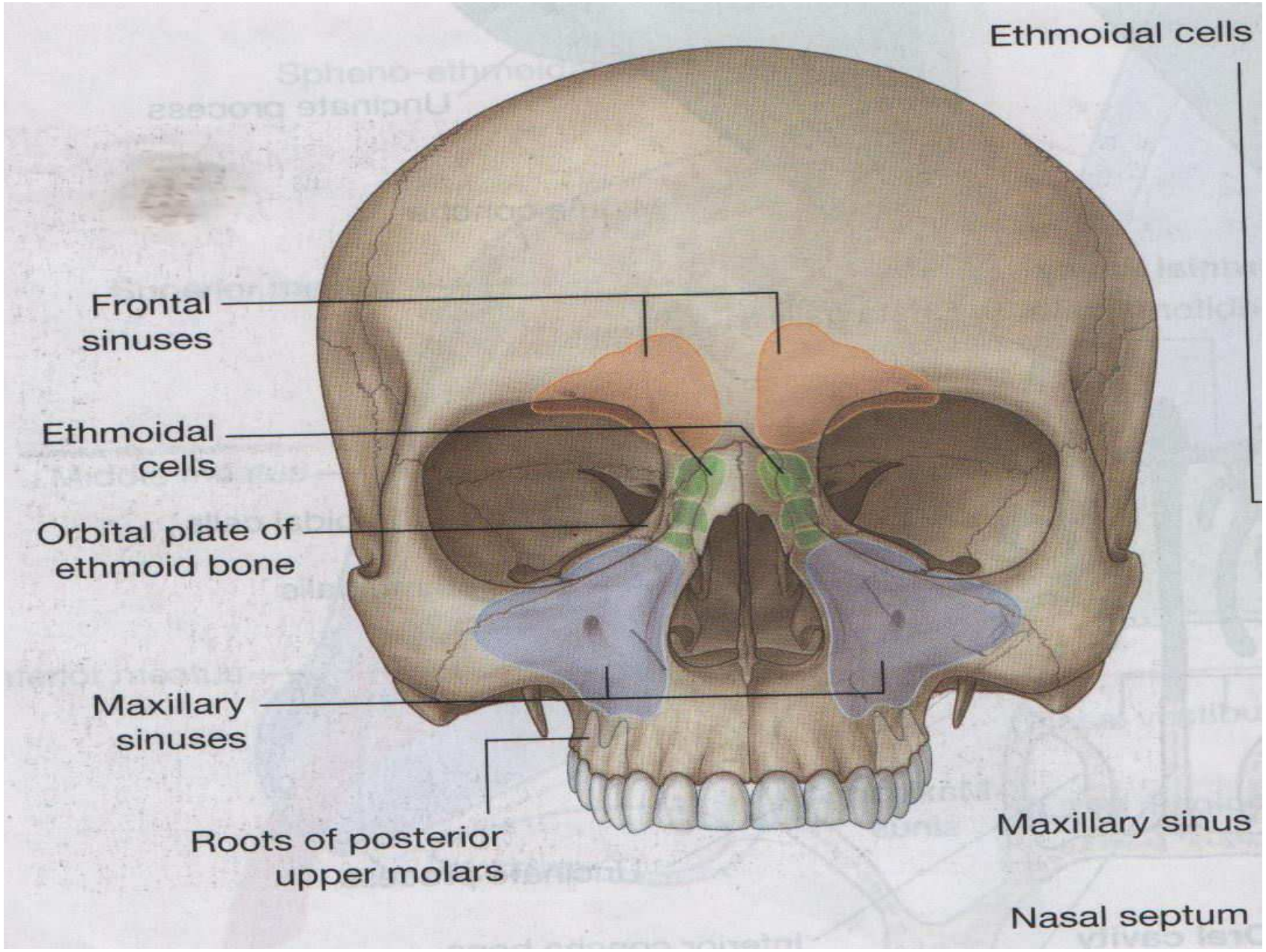


Contents of the orbit:

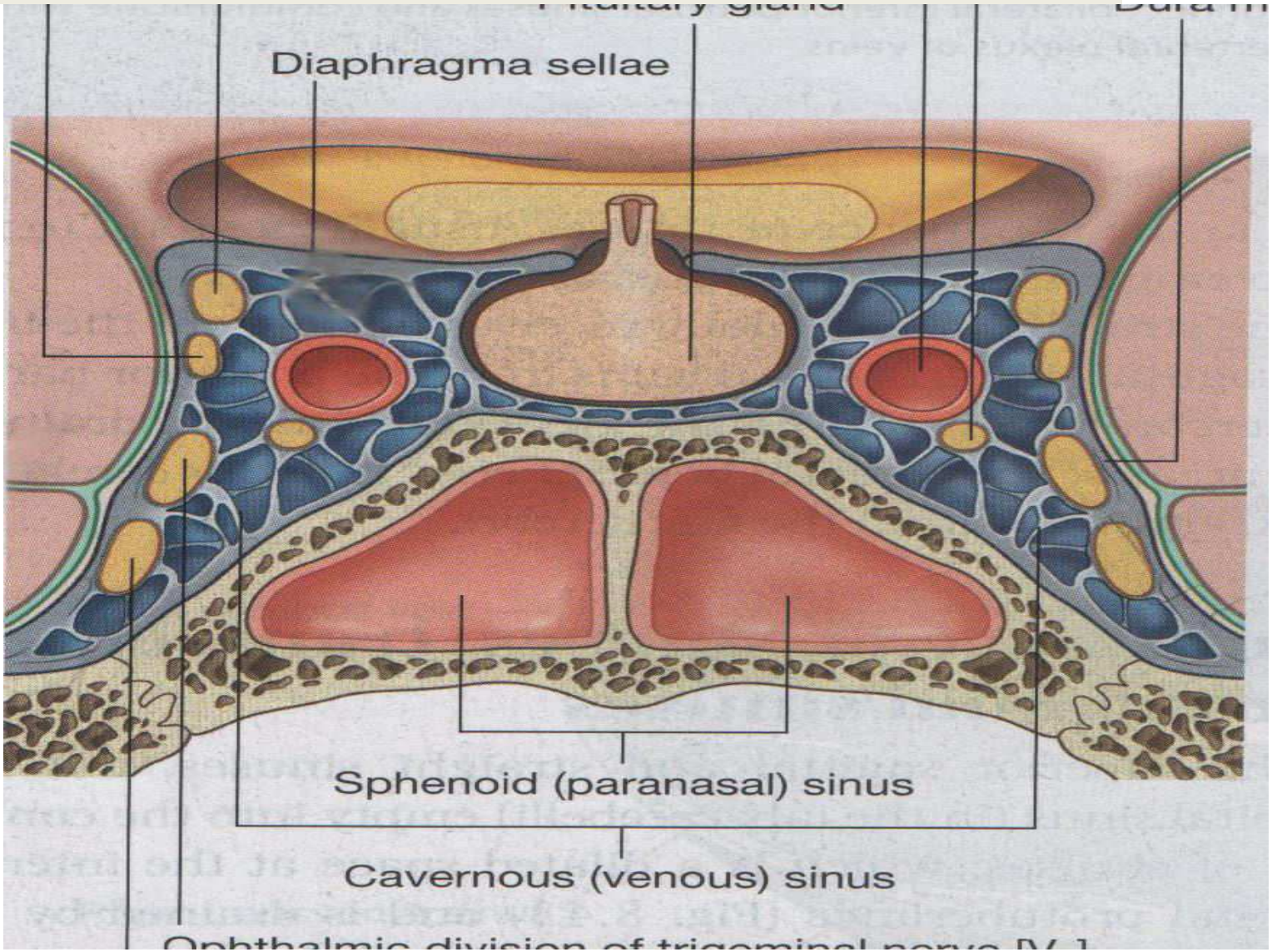
- Eye ball.
- Fascia.
- Ocular muscles: intra-ocular and extra-ocular muscles.
- Nerves: sensory and motor nerves.
- Blood vessels: ophthalmic artery and ophthalmic veins.
- Lacrimal apparatus: lacrimal gland and nasolacrimal duct.





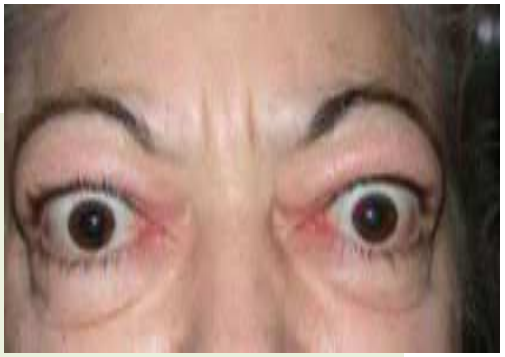

Ethmoidal cells
Frontal sinuses
Ethmoidal cells
Orbital plate of ethmoid bone
Maxillary sinuses
Roots of posterior upper molars
Maxillary sinus
Nasal septum

POSTERIOR RELATION



- **PROPTOSIS** is defined as forward protrusion of the eyeball

➡ Proptosis of more than 21mm or more than 2mm asymmetry between the two eyes is abnormal

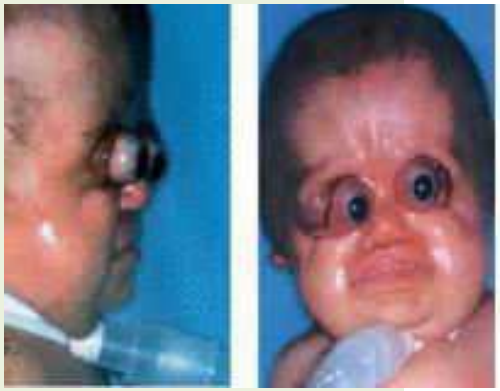


- ***EXOPHTHALMOS***

  Prominence of the eyeball secondary to thyroid disease

- ***PROPTOSIS***

  Prominence of the eyeball due to all other causes



- ***DYSTOPIA***

  Displacement of the globe in coronal plane

  It may coexist with proptosis or exophthalmos

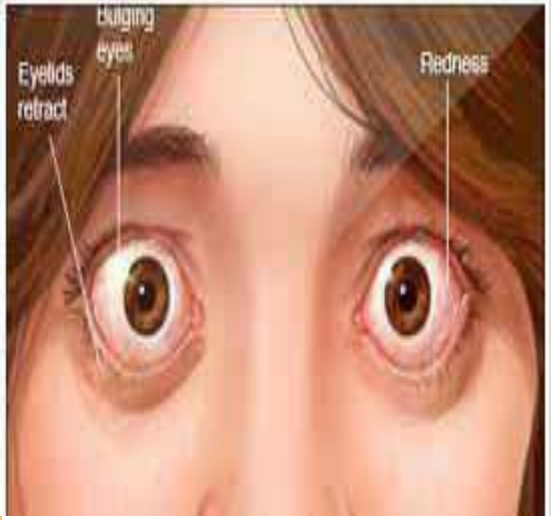
- ***EXORBITISM***

Due to decrease in the volume of orbit causing the contents to protrude forwards, Should be differentiated from proptosis and exophthalmos

PSEUDOPROPTOSIS

- It is the false impression of proptosis
- Seen in conditions like

1. Buphthalmos
2. High myopia
3. Contralateral ptosis
4. Contralateral enophthalmos



PRESENTING FEATURES

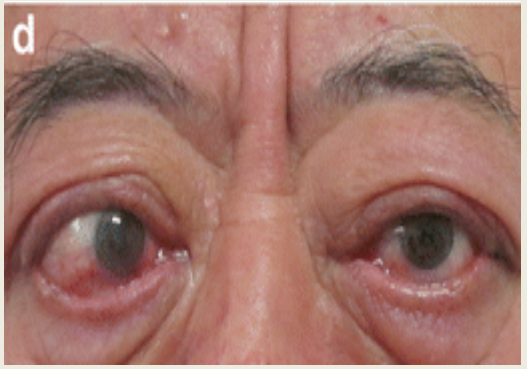
Disfigurement

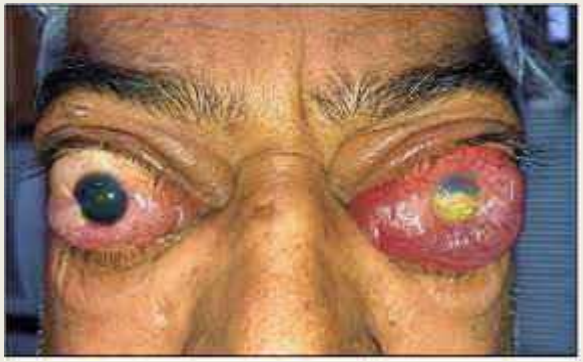
Gradual vision loss

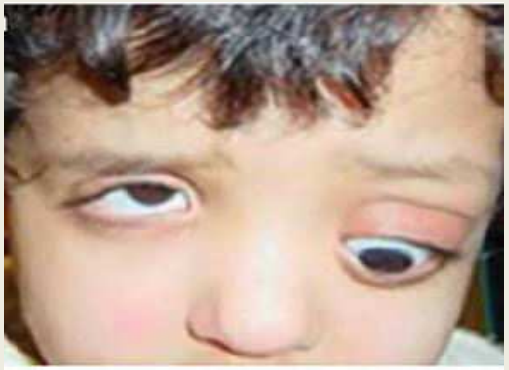
Diplopia

Pain

Reddness/chemosis

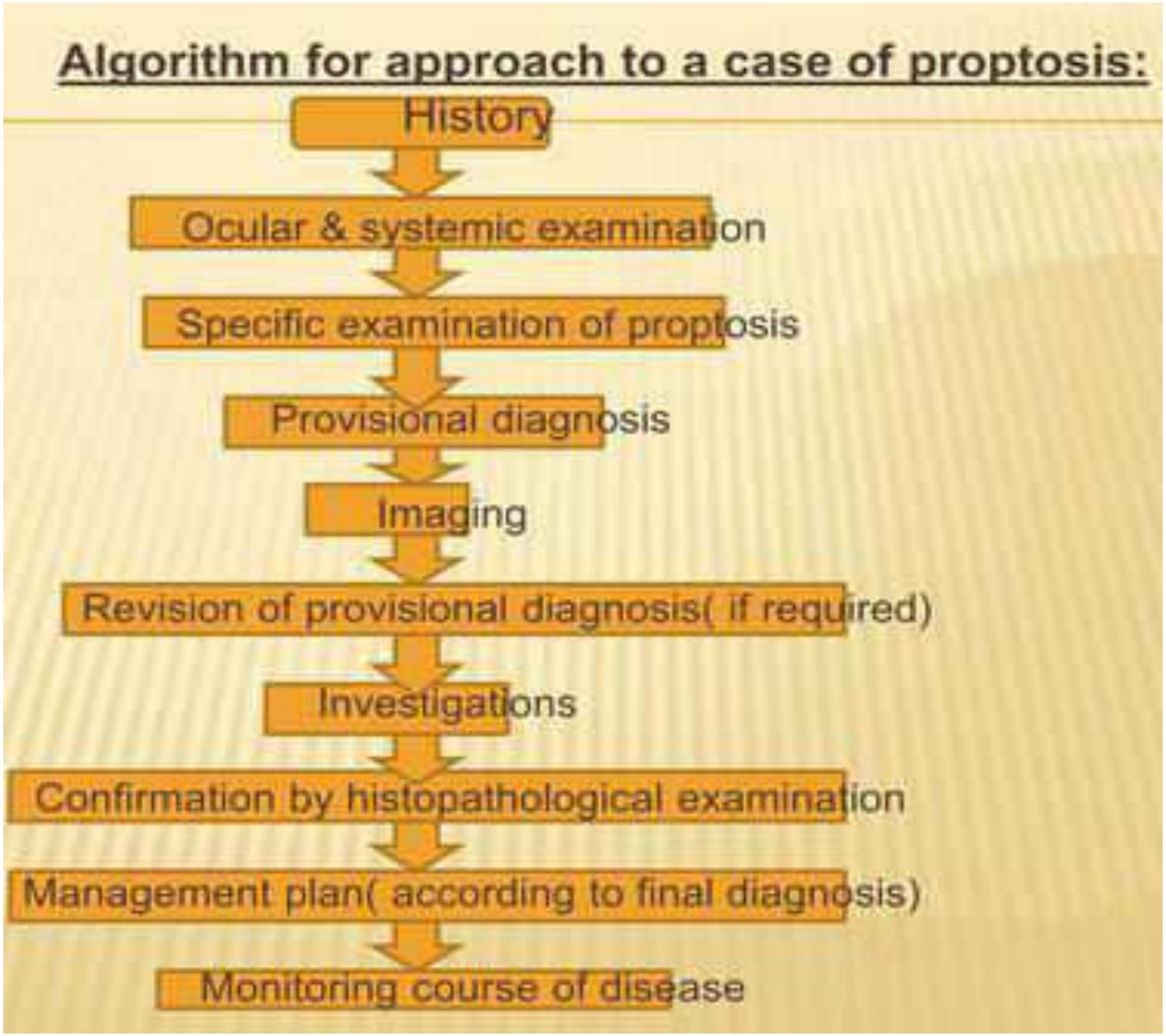






APPROACH

Algorithm for approach to a case of proptosis:



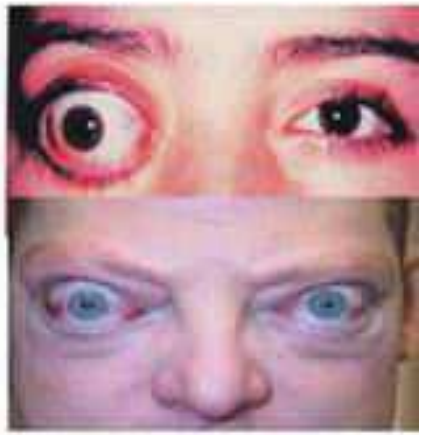
HISTORY

(A Thorough Medical /Ophthalmic History)

- Protrusion of eyeball - Age of Onset , duration , progression
- Constant or intermittent
- Variation with posture / strain
- Decreased vision – preceded/followed
- Stationary/progressive
- Associated field defects

- h/o Pain
- h/o Double vision
- h/o Trauma
- h/o fever , chills ,systemic symptoms
- h/o cancer
- h/s/o thyroid disease ,TB , DM ,HTN ,HIV , Syphilis

CLASSIFICATION



LATERALITY UNILATERAL & BILATERAL

TYPE AXIAL OR ECCENTRIC

DURATION ACUTE ,CHRONIC & INTERMITTENT

NATURE PULSATILE OR NON PULSATILE

COURSE OF THE DISEASE/ONSET

ACUTE	SUBACUTE	CHRONIC
Hours-Days	Weeks	Months/years
Traumatic –orbital hematoma Orbital emphysema	Inflammatory-OID	Neoplastic – benign/malignant
Infective (orbital cellulitis)	Thyroid eye disease	Inflammatory
	Neoplastic	

PROGRESSION OF PROPTOSIS

ACUTE (HOURS-WEEKS)	SUBACUTE (1-4 WEEKS)	CHRONIC (≥ 1MONTH)
Infection	Inflammation	TAO
Inflammation	Parasitic infections	ORBITAL VARICES
Parasitic infections	Metaplastic neoplasia	Cavernous Hemangioma
Trauma		Schwannoma
Metastatic lesions /Haemangioma		Optic nerve Glioma

TEMPORAL ONSET OF COMMON ORBITAL DISEASES

Hours	**Days**	**Weeks**	**Months**	**Years**
Traumatic	Inflammatory	Inflammatory	Neolpastic	Neoplastic
Hemorrhagic	Infections	Neolpastic	Lymphoid	Degenerative
Infectious	Traumatic	Traumatic	Vascular	Lymphoid
	Hemorrhagic	lymphoid	Inflammatory	Vascular
	Vascular	Vascular	Degenerative	Inflammatory

NATURE OF PROPTOSIS

Intermittent proptosis

1. Orbital Varices
2. Periodic orbital edema
3. Recurrent orbital hemorrhage

Pulsating proptosis

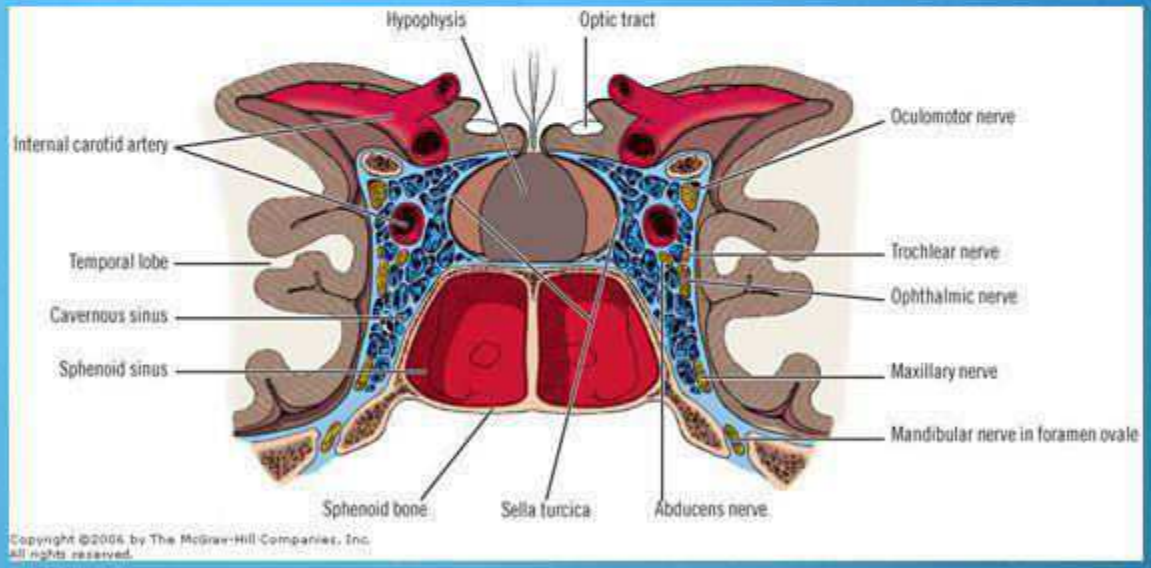
1. Carotid cavernous fistula
2. Congenital meningocele
3. Meningo encephalocele
4. Traumatic/Operative hiatus in orbital roof
5. Sacular aneurysm of ophthalmic artery

Carotid-Cavernous Fistula (CCF)

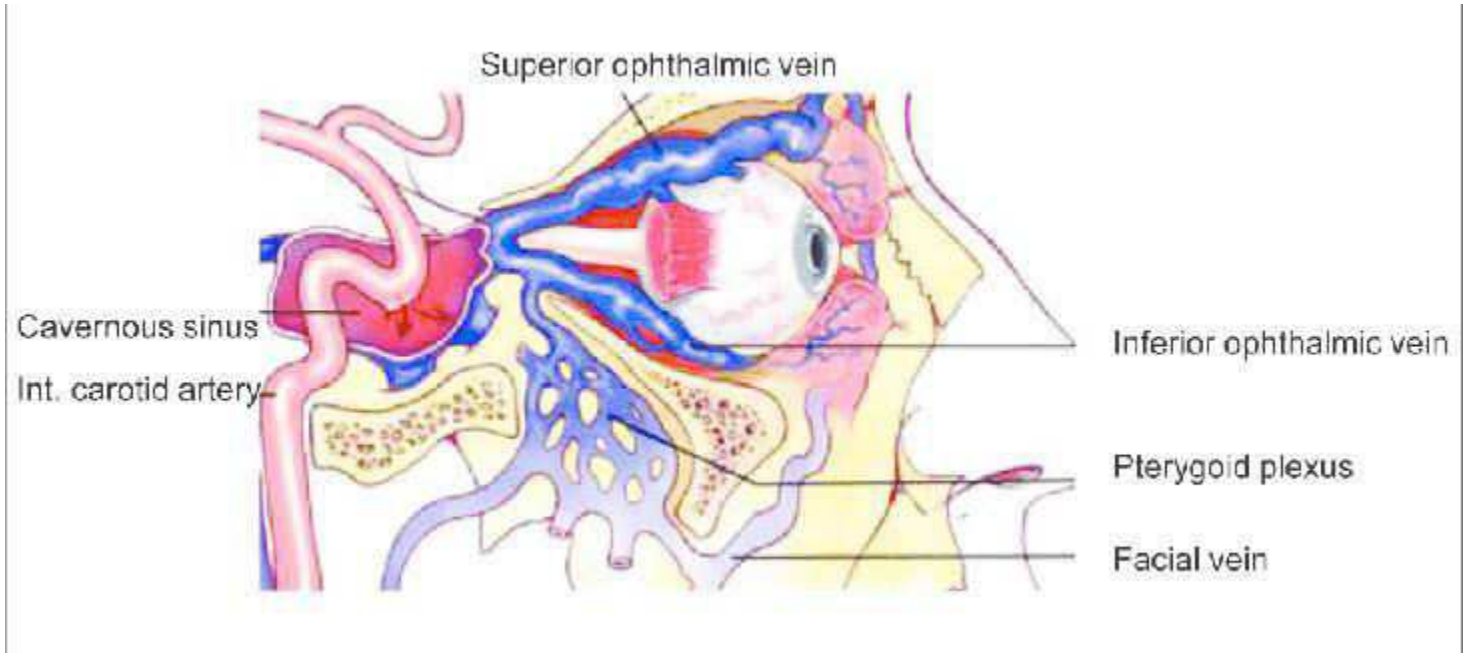
- Abnormal artery-vein communication
- #1 cause – trauma
- Chemosis, pulsatile proptosis, ocular bruit
- Cavernous sinus:

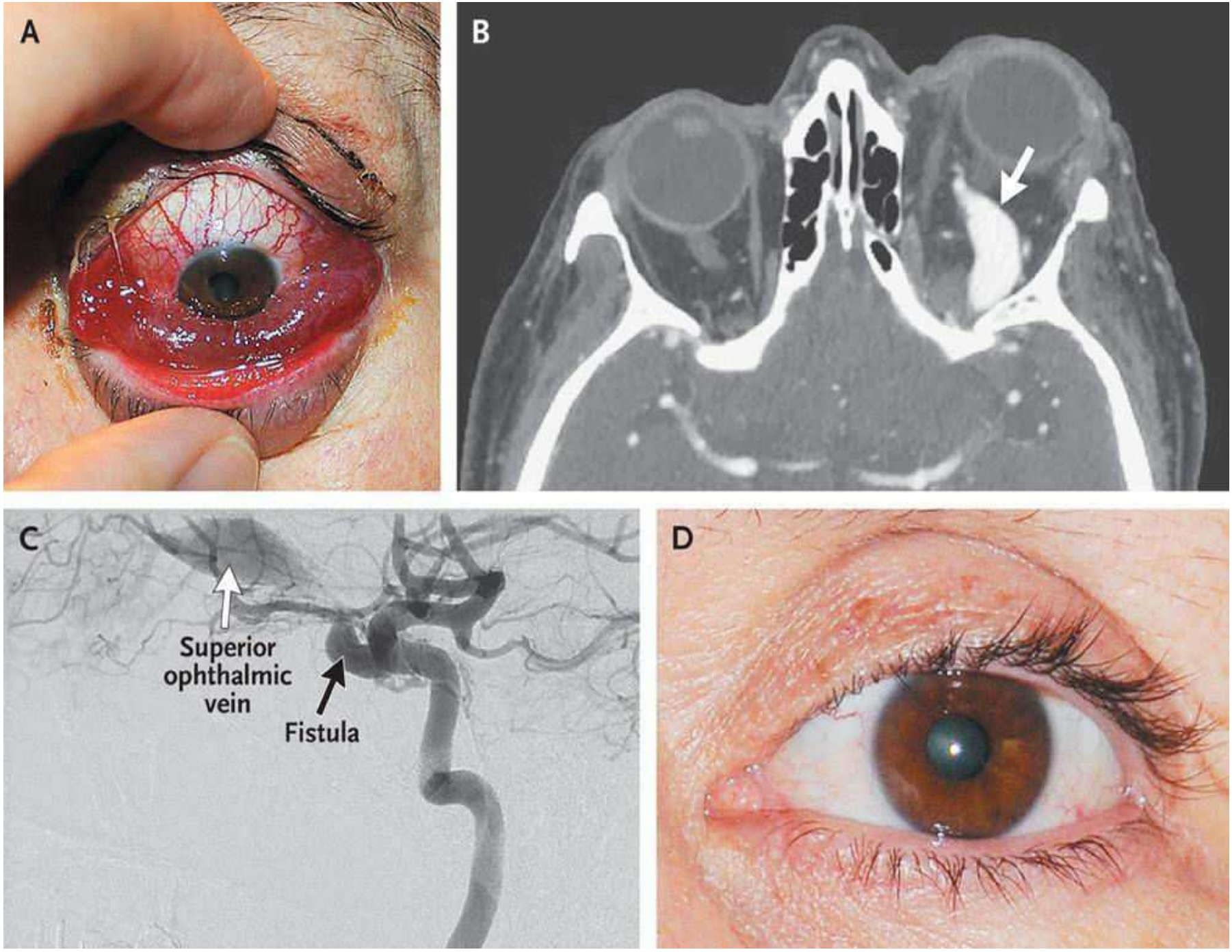
  3, 4, 6, V1, V2

  V3 & 7 do not go through

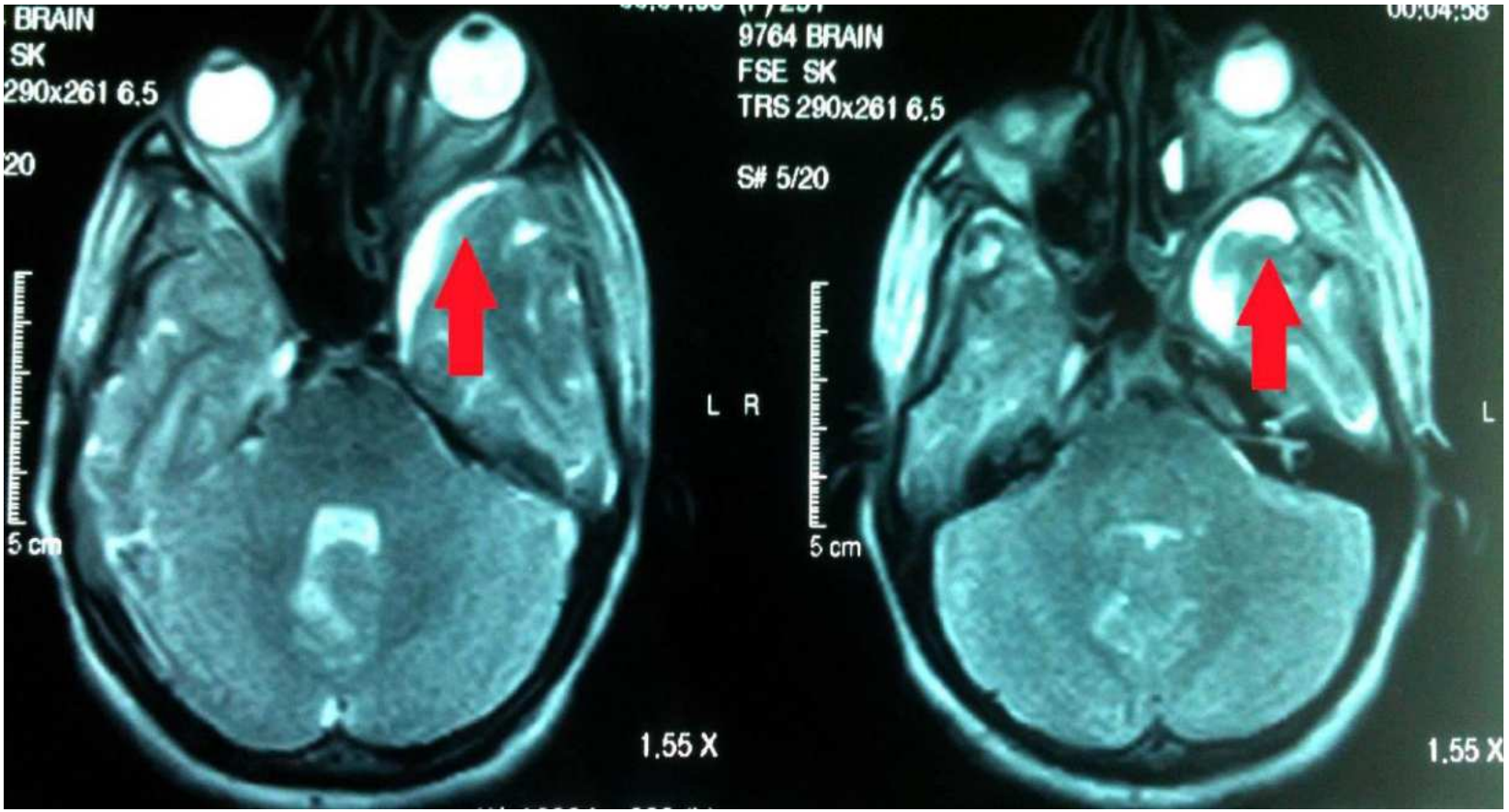


DIRECT CAROTID CAVERNOUS FISTULA

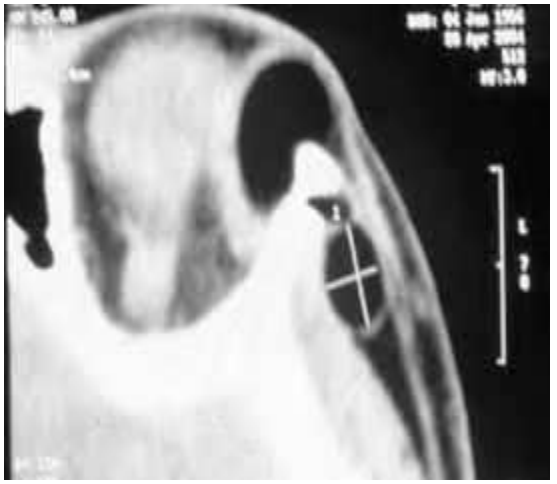


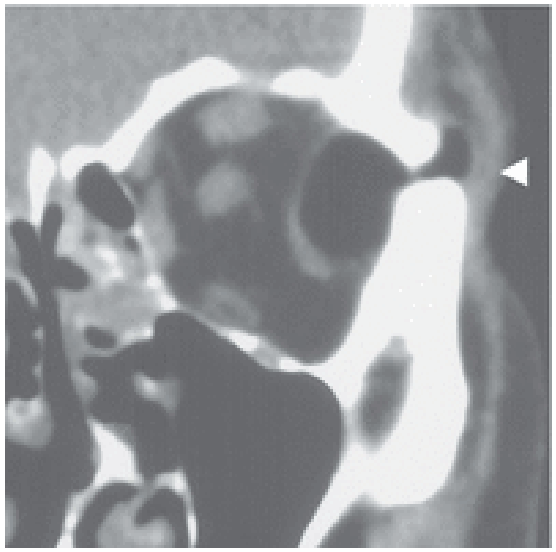

A
B
C
Superior ophthalmic vein
Fistula
D

PULSATILE PROPTOSIS



- **Progression:** The proptosis my be progressive,static or waxing-waning.
- Rare cases of ***intermittent proptosis*** are caused by ***dumb-bell dermoids***,with components in the orbit & the temporal fossa.
- **Medical & systemic history**: pt asked for h/o malignancy,weight loss,smoking.
- **Biological effects of disease**: ***pain***, swelling around the eye,diminished vision,watering, diplopia.



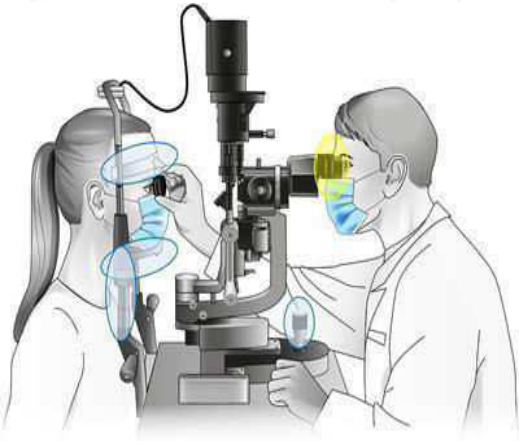


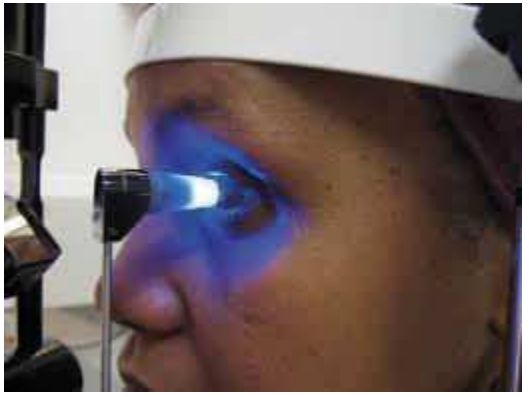
PAIN

SEVERE	MODERATE	DULL BORING PAIN
INFECTIONS	IOID	BONE EROSION DUE TO NEOPLASTIC TUMOR
INFLAMMATIONS	RUPERED DERMOID CYST	
ORBITAL ABCESS	TRAUMA	
METASTATIC LESIONS	MYCOCYSTICERCOSIS	
ACUTE ONSET TAO		
LYPMHANGIOMA		
HIGH FLOW CCF		

OCULAR EXAMINATION

- VISION
- PUPIL
- IOP
- OCULAR-MOTILITY & ALIGNMENT
- PROPTOSIS
- PALPRABERAL FISSURE HEIGHT
- CONJUNCTIVAL CHEMOSIS
- CORNEA
- FUNDUS

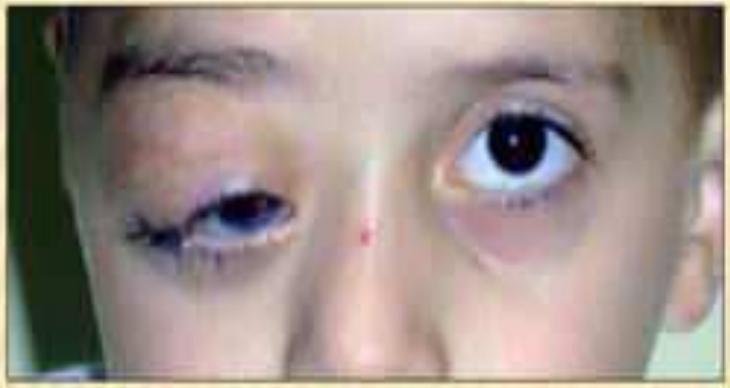


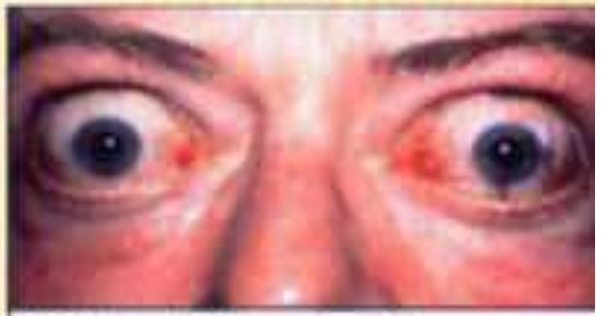


OCULAR EXAMINATION

- *Visual acuity*: diminution d/t optic nerve compression, corneal exposure.
- *Refraction:* acquired hyperopia d/t mass indenting the posterior pole of globe, high myopia causing pseudoproptosis.
- *IOP:* thyroid orbitopathy(d/t restriction of movt.), aretriovenous fistula (d/t elevated venous pressure)
- *Conjunctiva:* chemosis (in severe inflammation salmon colored patch (in lymphoma), dilated episcleral vessels (carotid cavernous fistula)

Eyelids: lid retraction,lid lag in thyroid orbitopathy,
S-shaped lid thickening (neurofibromatosis),lagophthalmos

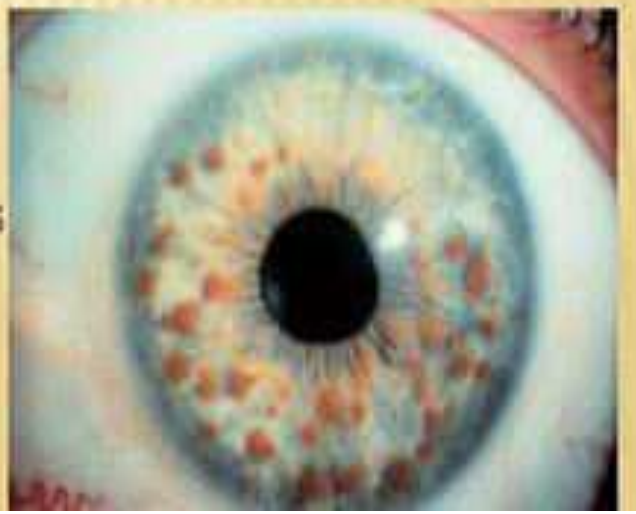




Bilateral lid retraction
- Bilateral proptosis

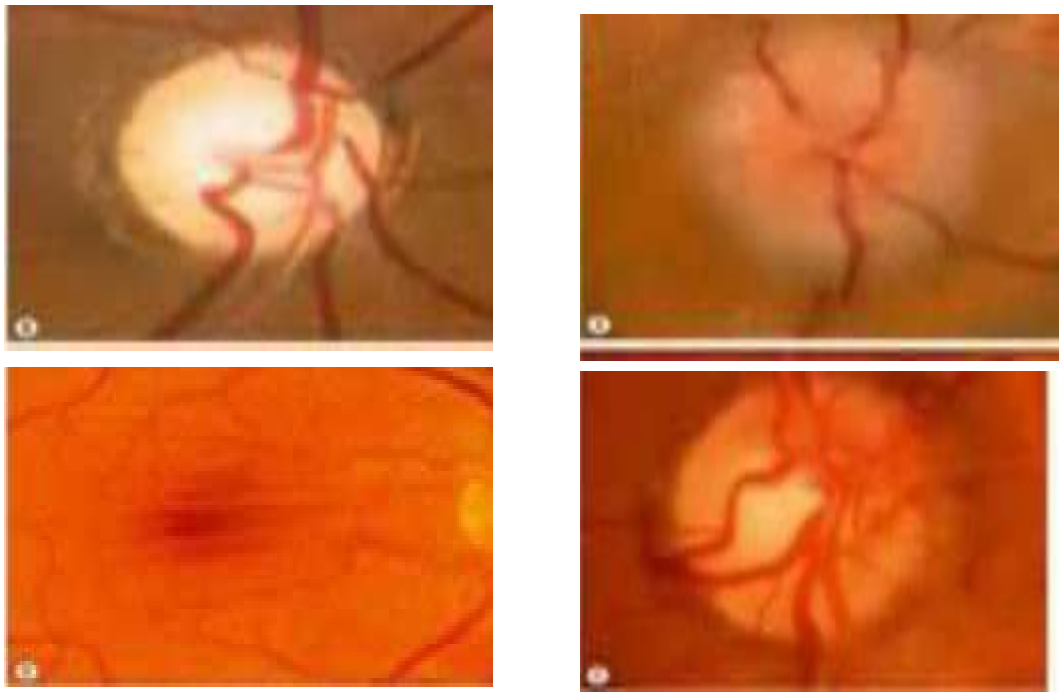
Cornea: exposure keratopathy.

Iris: lisch nodules. (neurofibromatosis



- Dilated episcleral vessels – AV shunt
- Optociliary shunt vessels- optic nerve sheath meningioma
- Salmon patch beneath upper eyelid – orbital lymphoma
- Eversion of upper lid – waxy yellow infiltrate with tortuous vessels- amyloid
- S shaped deformity of upper lid – plexiform neurofibroma
- Lid retraction or lidlag - thyroid ophthalmopathy

- ***PUPILS-RAPD***
- ***EOM*****-direct muscle involvement by the disease, mechanical limitation,compression of nerves, cavernous sinus thrombosis**
- ***FUNDUS exam*****-Swollen disc, optic atrophy,optociliary shunt vessels, choroidal folds**



SYSTEMIC EXAMINATION

- Thyroid examination
- Primary tumors elsewhere in the body – CVS/RS/Abdomen/PV/Rectal
- ENT examination

LOCAL EXAMINATION

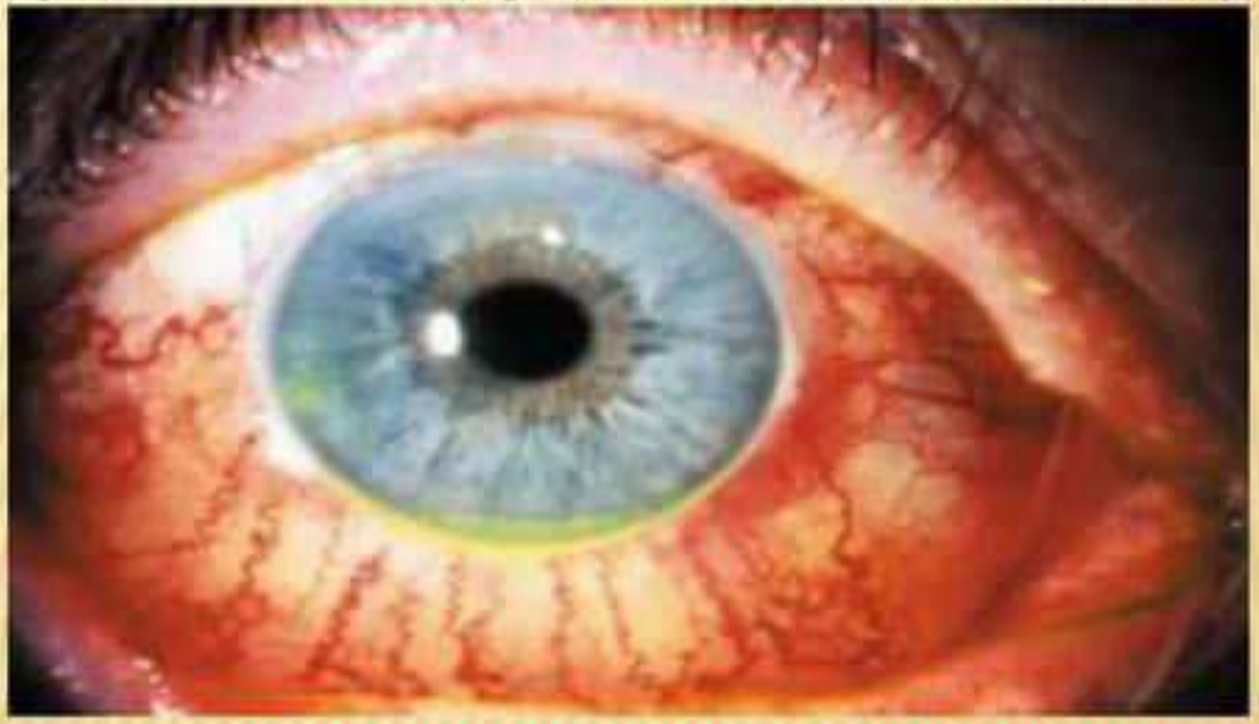
- 1) INSPECTION –
  - ★ Proptosis or pseudoproptosis
  - ★ Unilateral or bilateral
  - ★ Axial or eccentric
- 2) PALPATION – size ,shape,surface,margins consistency , tenderness , compressibility

  Thrill /increase with valsalva/ orbital rims / regional lymph nodes
- 3)AUSCULTATION - bruit

PULSATION:

-best detected on lateral view/ while using applanation tonometer.

-e.g arterio-venous fistula (high flow carotid-cavernous fistula), Aneurysms.



(dilated episcleral vessels in arterio-venous fistula)

OR

Due to transmitted pulsation through a defect in the bony orbital wall.

e.g : Sphenoid wing dysplasia (in neurobibromatosis),

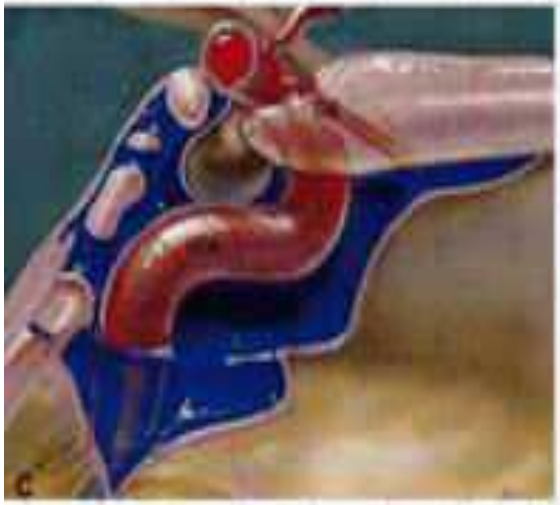
Meningo encephalocele,

Herniation of frontal lobe of brain into orbit following trauma

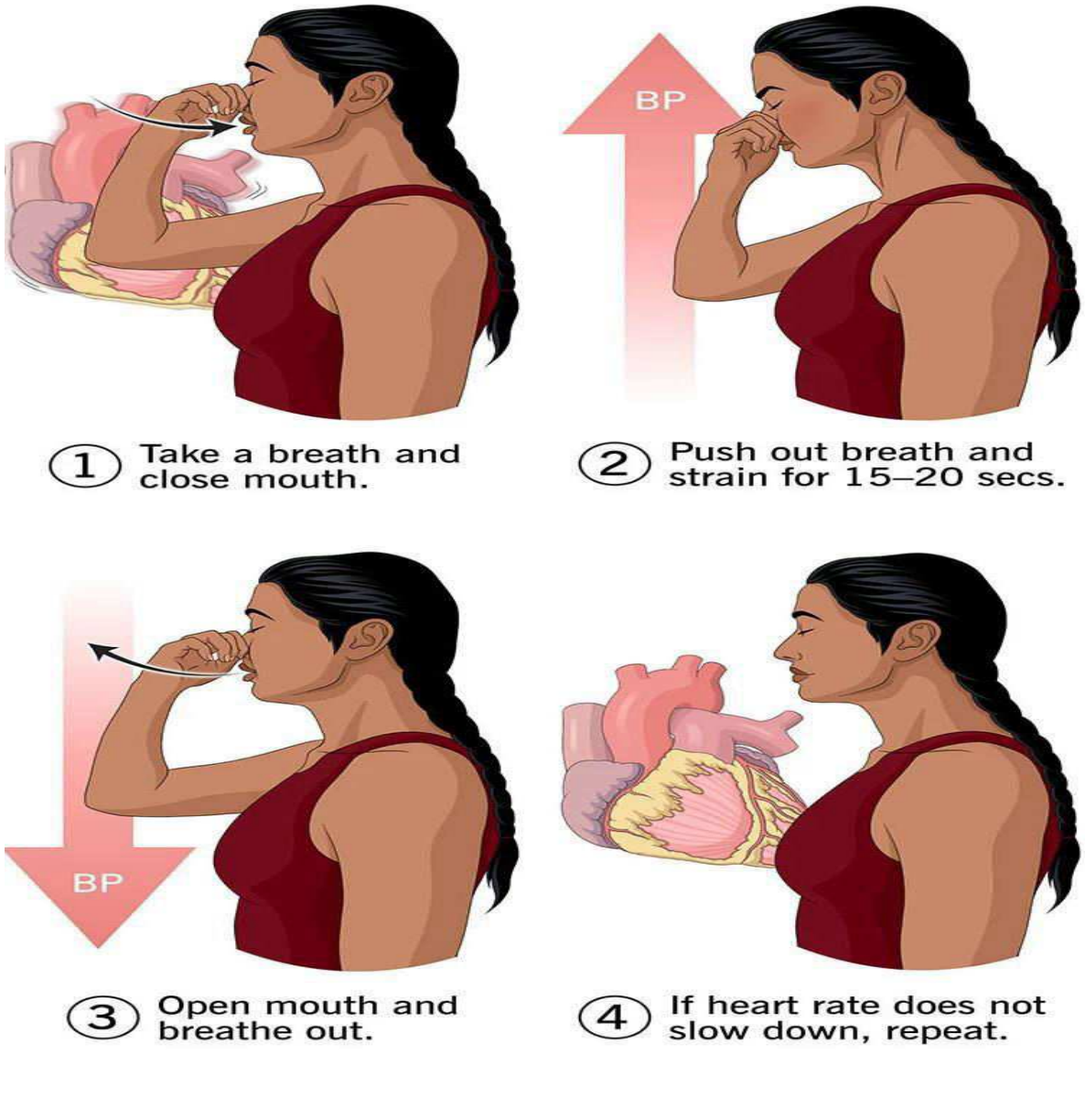
AUSCULTATION:

carotid-cavercous fistula→ bruit heard best by the bell of

- Globe/temporal region for bruit



Valsalva maneuver



PROPTOMETRY

It is the measurement of the distance between apex of the cornea and the

bony point usually taken as deepest portion of the lateral orbital rim with

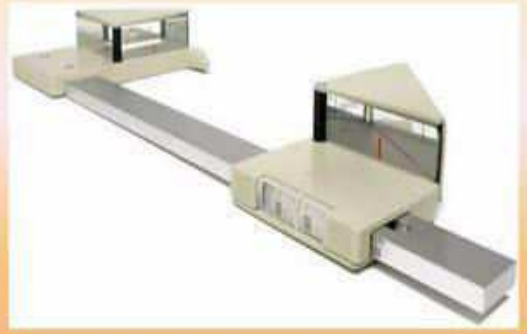
the eye looking in primary gaze.

MEASUREMENTS:

Asymmetry > 2mm or more b/w the eyes.

OR

Protrusion greater than
-***13-15mm*** in east asians
-***21mm*** in caucasian adults.
-***23mm*** in adult african-americans



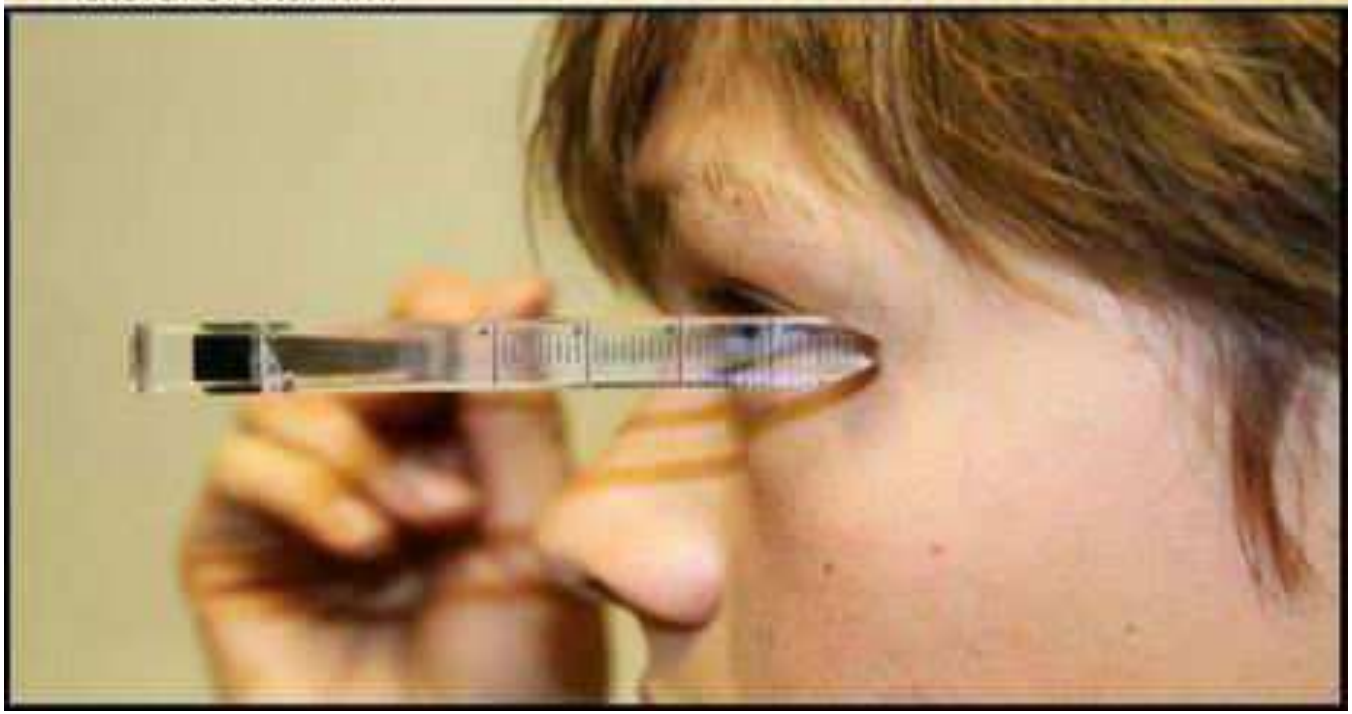
Clinical methods for measurement of proptosis:

- A) **PLASTIC RULER**: can measure proptosis from the lateral orbital rim to the corneal apex,holding the ruler parallel to ground.

- B)**LUEDDE'S EXOPHTHALMOMETER**: has several advantages

-notch confirms to lateral orbital rim.

-the scale starts from tip of instrument,where the notch meets the lateral orbital rim.



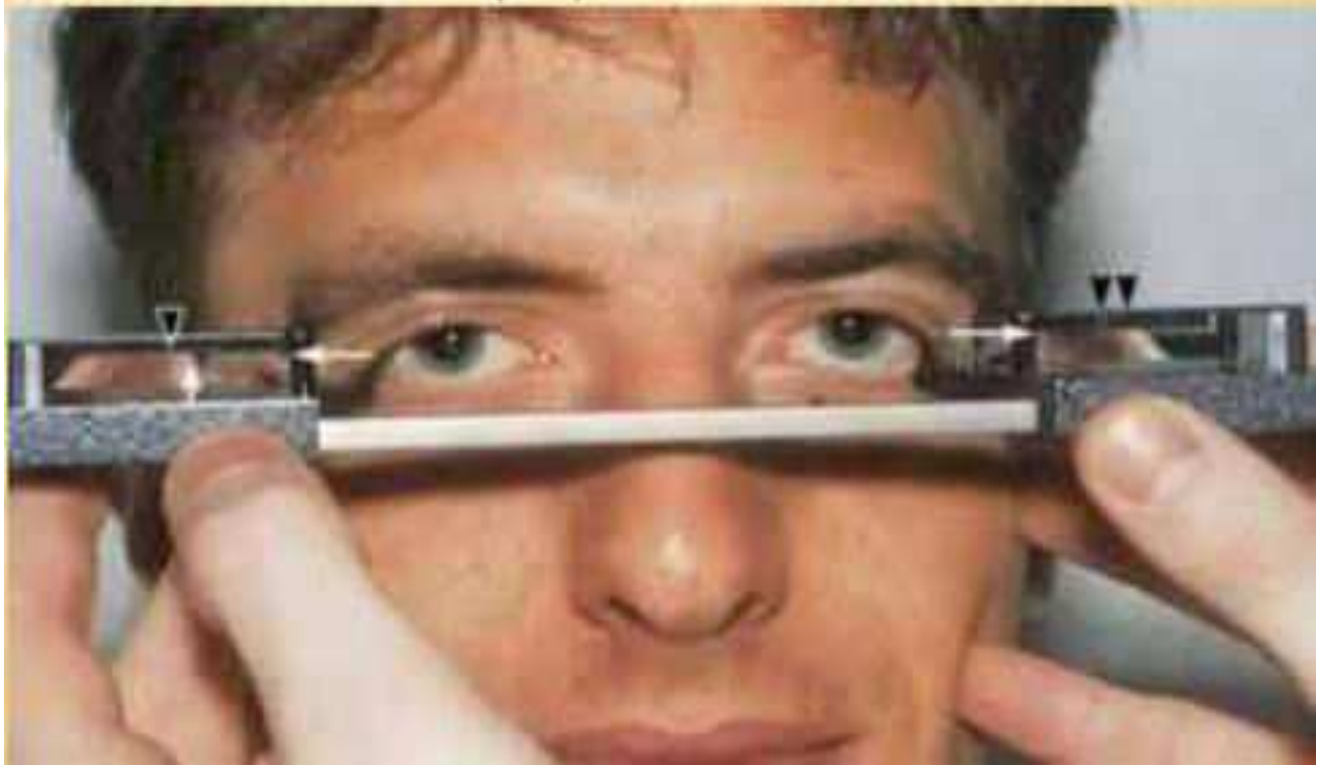
-markings on both sides help to avoid parallax error.

-luedde's exophthalmometer is better than hertel's if there is facial asymmetry.

C) **HERTEL'S EXOPHTHALMOMETER**: m/c used.

-it may use prisms or mirrors set at 45 degree angles.

-it is best for serial follow up of patients.

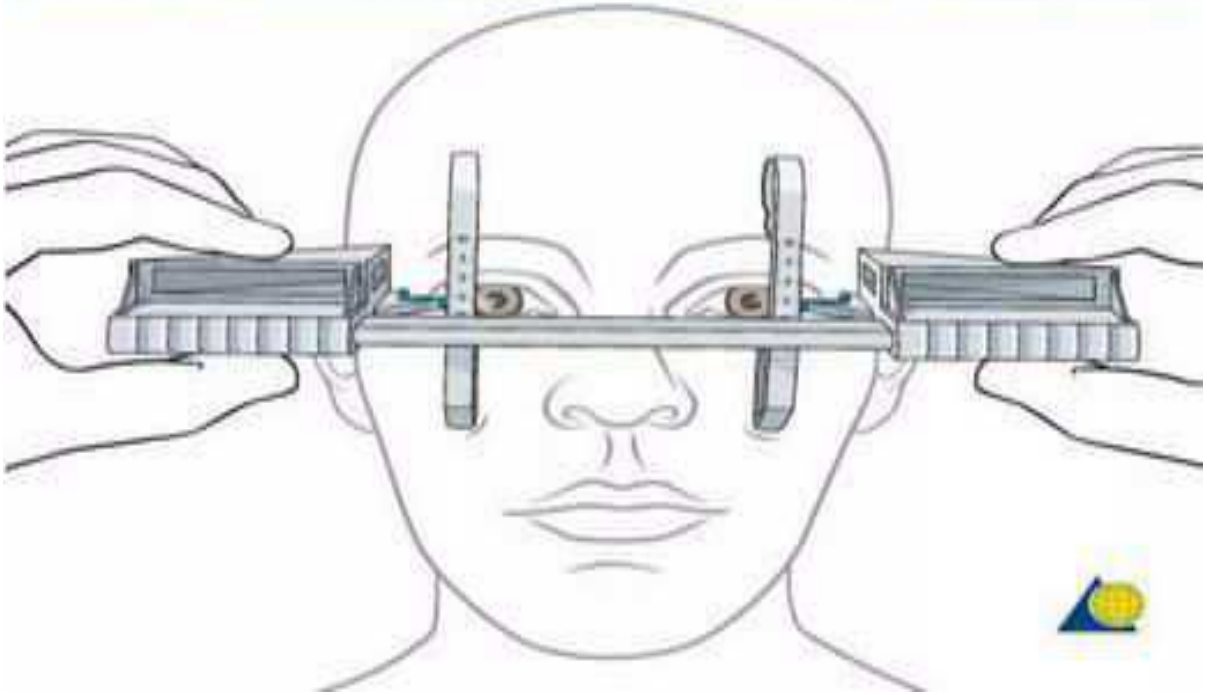


D) NAUGLE'S EXOPHTHALMOMTER:

-In case of acquired or congential asymmetry of the lateral orbital rims a Hertel exophthalmometer is misleading

-This is an inferior & superior rim based instrument.

-may be used when the lateral orbital rim is not intact.



MEASURING PROPTOSIS ON A CT SCAN

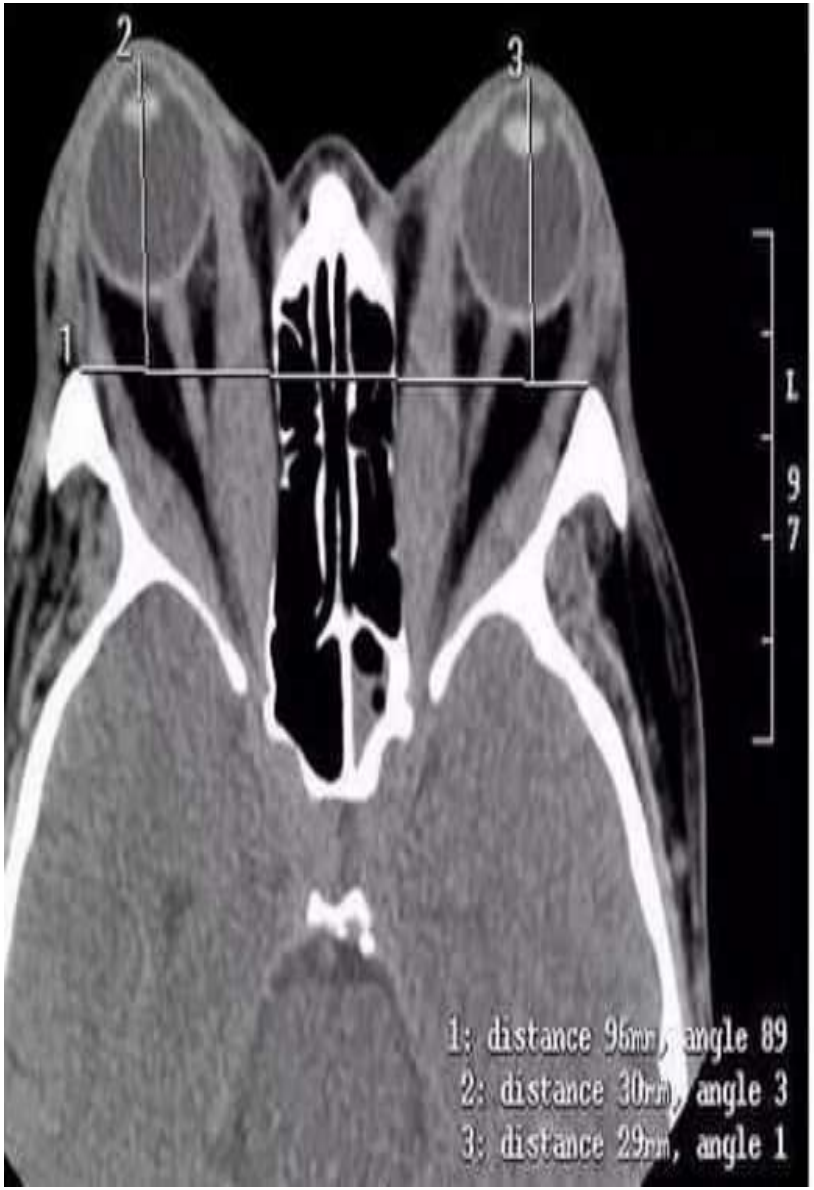
HILAL AND TROKEL METHOD:

-In a mid axial CT scan image,a baseline between the tips of lateral orbital rims is drawn.

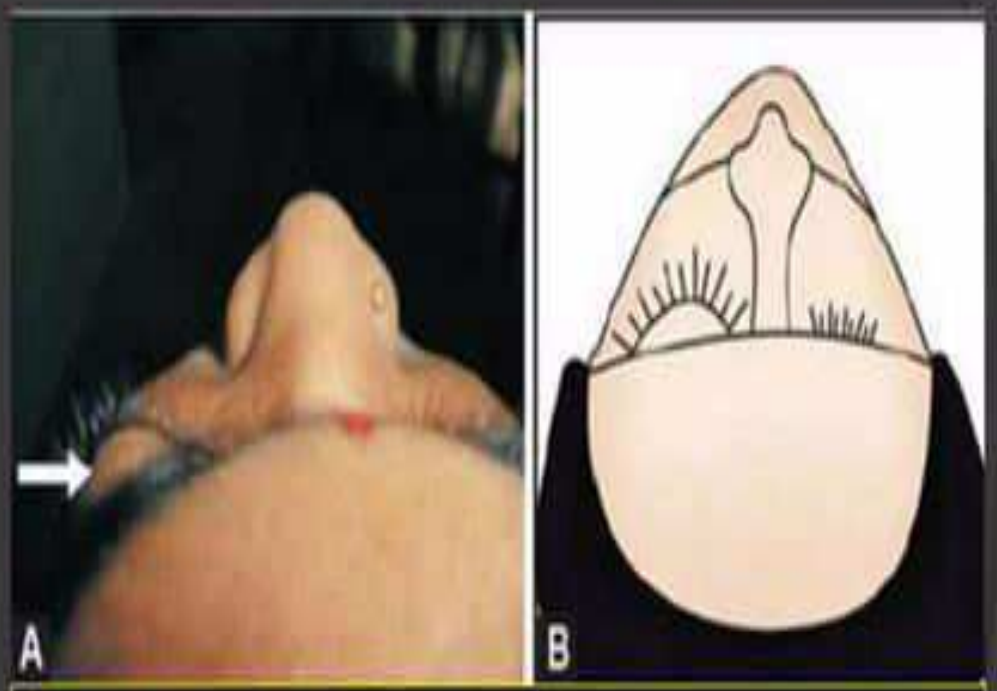
-a perpendicular from each corneal apex to this line is dropped & measured to scale.

if each line > 21mm
or
if asymmetry >2mm b/w two

indicates abnormality.

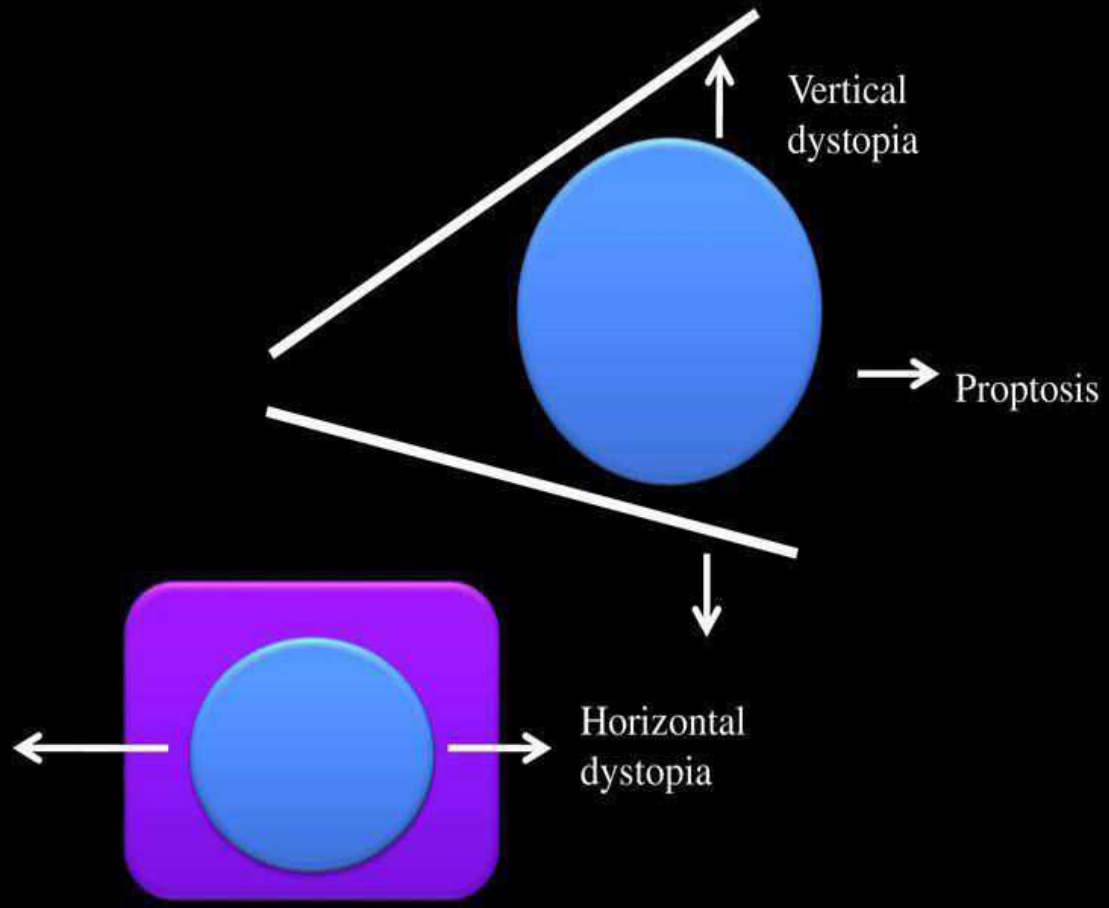


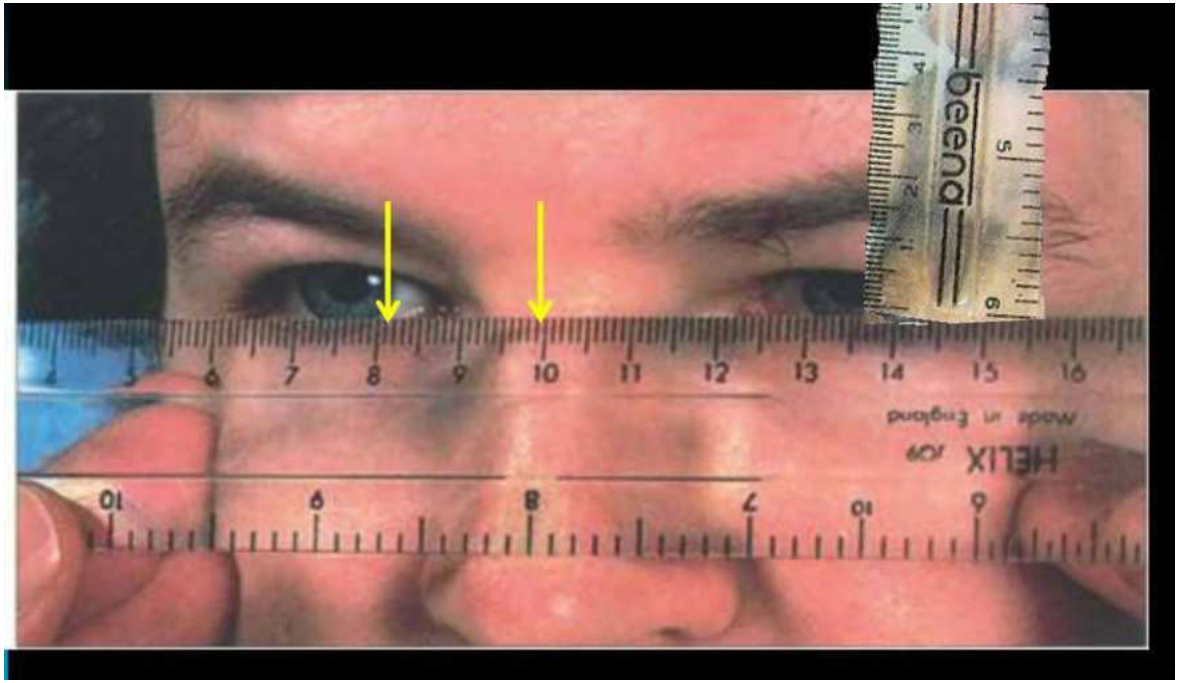
ASSESSMENT & MEASUREMENT OF PROPTOSIS



NAPHZEIGER'S TEST: NOTE THE PROMINANCE OF LEFT EYE. THE RIGHT IS NOT VISIBLE

Proptosis & dystopia



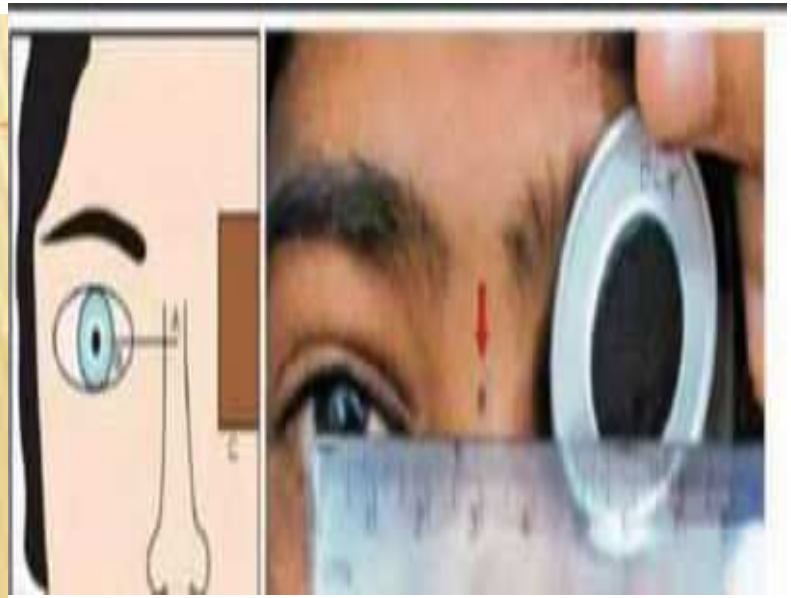

beena
HELIX
Made in England

Measurement of dystopia:

-In an eccentric or non axial proptosis, the horizontal & vertical dystopia of globe is to be measured.

-***Horizontal dystopia*** :is measured by the distance from the midline of bridge of nose to the nasal limbus, compared bilaterally.

-***Vertical dystopia*** :is measured by the superior or inferior deviation of the central corneal reflex of the proptotic eye from a horizontal line passing through the centre of normal eye.



LAB INVESTIGATIONS

- Hematological - CBC , ESR, VDRL
- Thyroid function tests
- Serum ANA , c- ANCA , ACE
- BUN , Creatinine
- C-XRAY , Mantoux test
- Casonis test – r/o hydatid cyst
- Stool examination – cysts /ova
- Urine analysis – bence jones proteins - MM

IMAGING

- XRAY –
- Calcification/hyperostosis – Meningiomas
- Waters view – blow out fractures
- Rhese view – optic foramen and SOF

- CT- SCAN
- USG
- MRI

→ Size , position and shape of lesion

- ORBITAL VENOGRAPHY – Orbital varix
- CAROTID ANGIOGRAPHY – Aneurysms /AV communications

HISTOPATHOLOGICAL STUDIES

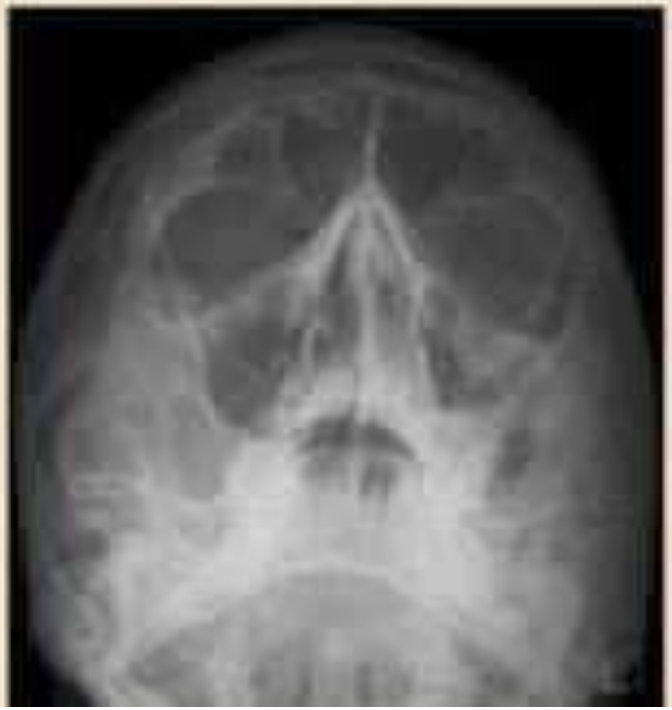
- FNAB
- Incisional biopsy
- Excisional biopsy

XRAYS

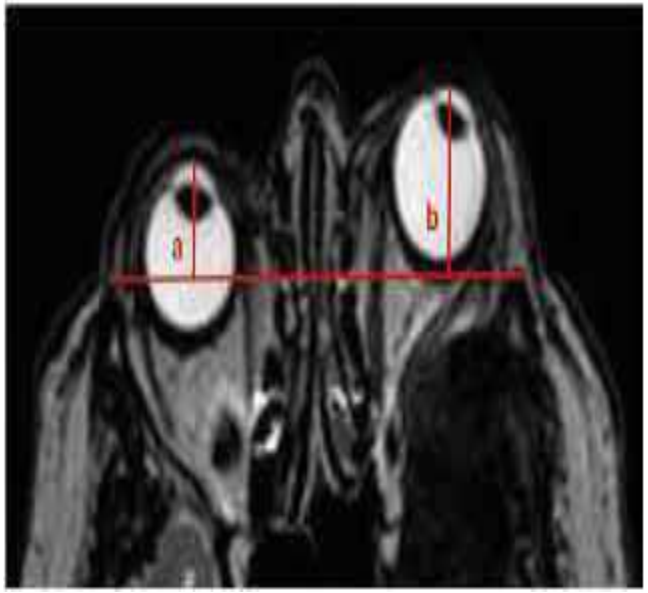
VIEW	STRUCTURES APPRECIATED
Caldwell view:	greater and lesser wing of sphenoid. Superior orbital fissure, most of the paranasal sinuses
Water's view:	orbital rim, orbital roof and floor and maxillary sinuses
Lateral view:	sphenoid, sphenoid air sinuses, anterior clinoid and sella turcica
Townne's view:	Infraorbital fissure , Superior orbital fissure
Axial	Basal view

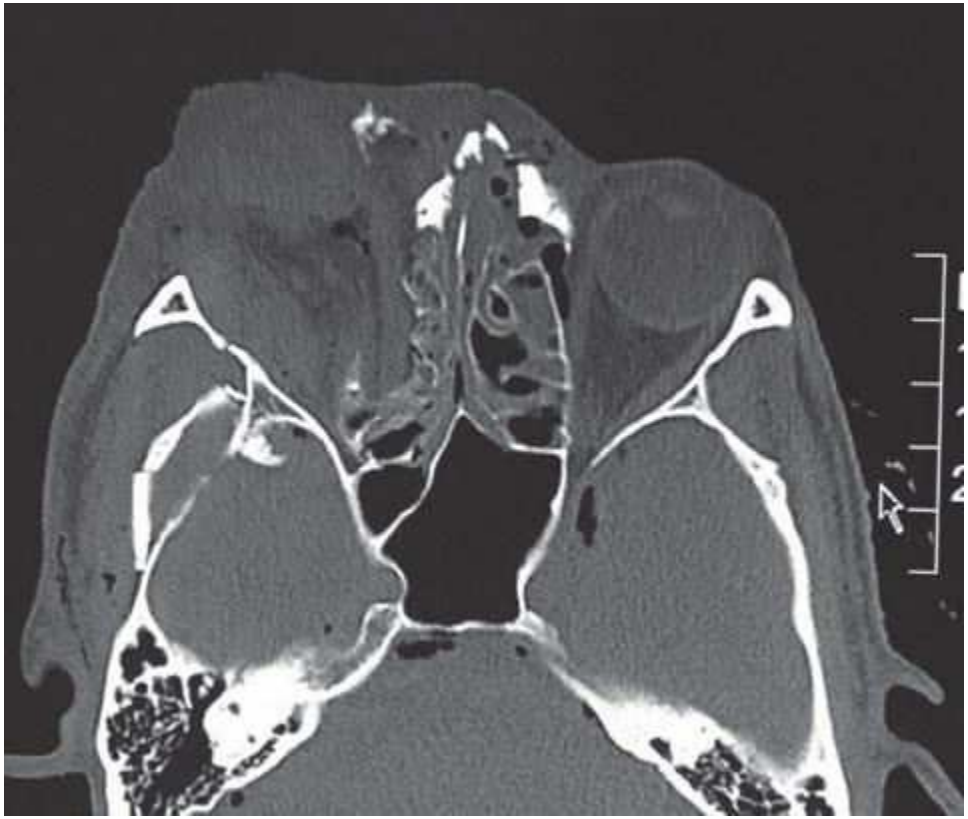
Water's view/Orbital view

Structures seen- anterior 2/3rd of orbital floor

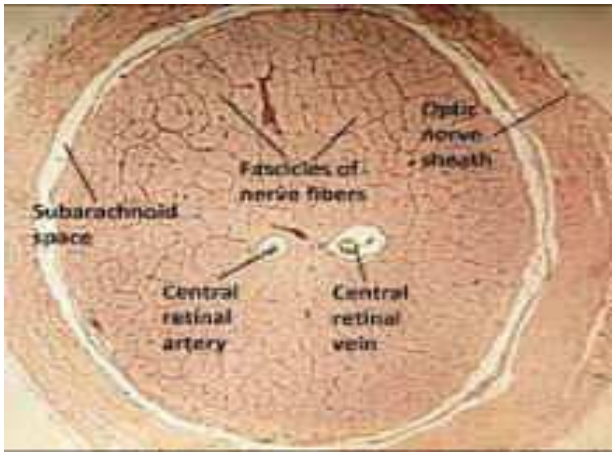


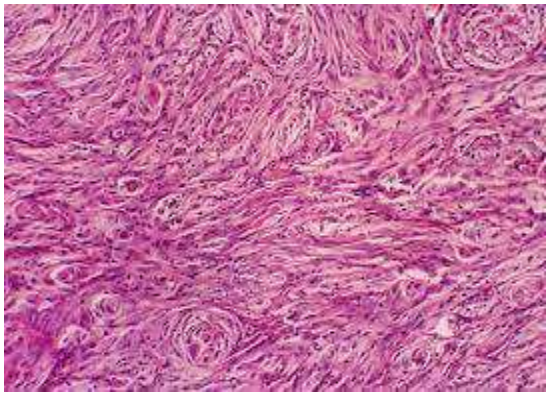
CT SCAN/MRI SCAN IN PROPTOSIS



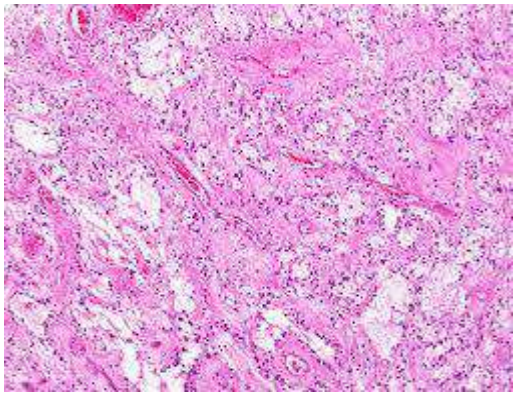


Biopsy for definitive diagnosis

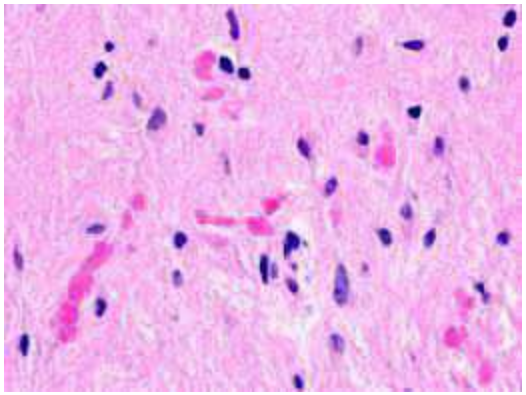




Meningioma



Glioma

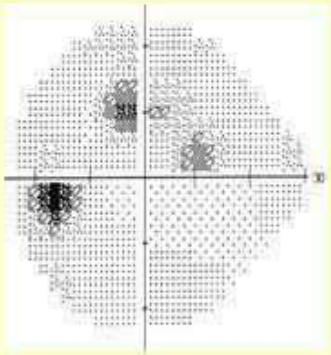


Pilocytic astrocytoma

CONCLUSION

- ***Orbital diseases have sight threatening and at times life threatening sequelae***
- ***Commonest presentation is proptosis***
- ***Sight threatening signs are corneal exposure, disc swelling, pupillary abnormalities***
- ***A proper work up would lead to the definitive diagnosis***

What do we do in eye clinic

	Note symptoms/appearance Measure exophthalmos/TED group
	Afferent pupillary defect Test visual fields, colour vision, eye pressure, examine optic disc
	CT scan..diagnosis, especially if unilateral

KEY POINTS

- The most common cause of bilateral proptosis is Graves disease.
- Acute unilateral proptosis suggests infection or vascular disorder (eg, hemorrhage, fistula, cavernous sinus thrombosis).
- Chronic unilateral proptosis suggests tumor.
- Do CT or MRI and thyroid function testing when Graves disease is suspected.
- Apply lubrication to exposed cornea.


THANK YOU..

Topic: Preseptal & Orbital Cellilitis

Learning Objectives: Clinical Features and management of Preseptal and orbital cellulitis

Prof. Dr. Tariq Farooq Babar
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Orbital inflammatory diseases

- Cellulitis
  - Preseptal
  - Orbital
- Mucormycosis
- Idiopathic orbital inflammatory disease
- Myositis
- Dacryoadenitis
- Tolosa – Hunt Syndrome
- Wegener's granulomatosis

- Orbital cellulitis – an ophthalmic and medical emergency
- May cause loss of vision, even death
- Management under the combined care of:
  - ✓ Ophthalmologist
  - ✓ ENT Specialist
  - ✓ Pediatrician in children
  - ✓ Medical Specialist in adults and elderly

Preseptal cellulitis

- Not truly an orbital disease
- Much common than orbital cellulitis
- Commoner in children
- 80% cases under 10 years of age.

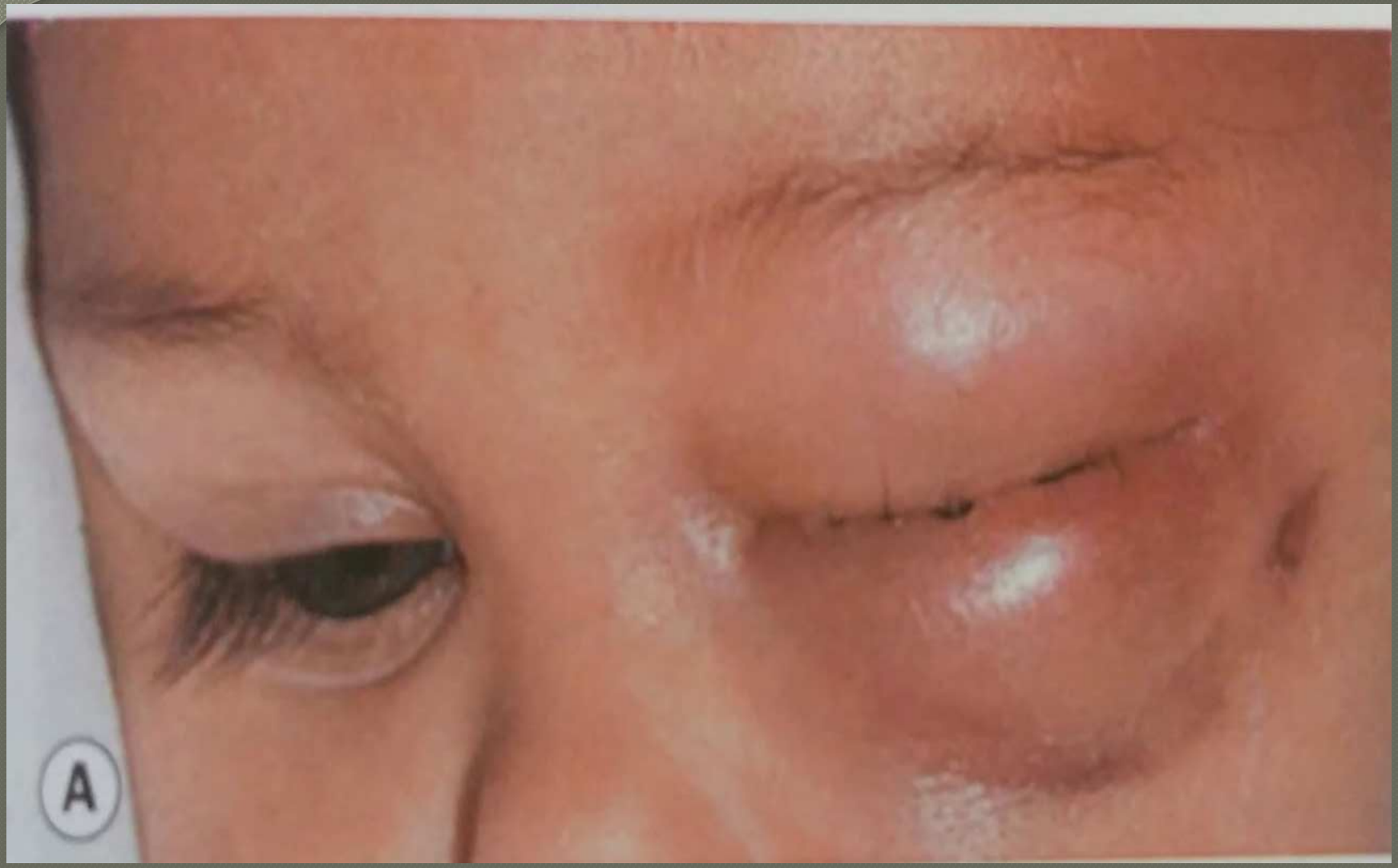
- Main causative organisms
  - Staphylococci
  - Streptococci
- Less severe disease, at least in adults and older children.
- In younger children, orbital septum not fully developed.
  - High risk of progression
  - Treated similarly to orbital cellulitis

Risk factors:

- Infection of adjacent structures
  - Dacryocystitis
  - Hordeolum
- Systemic infections
  - Upper respiratory tract infections
- Trauma
- Laceration

Clinical features:

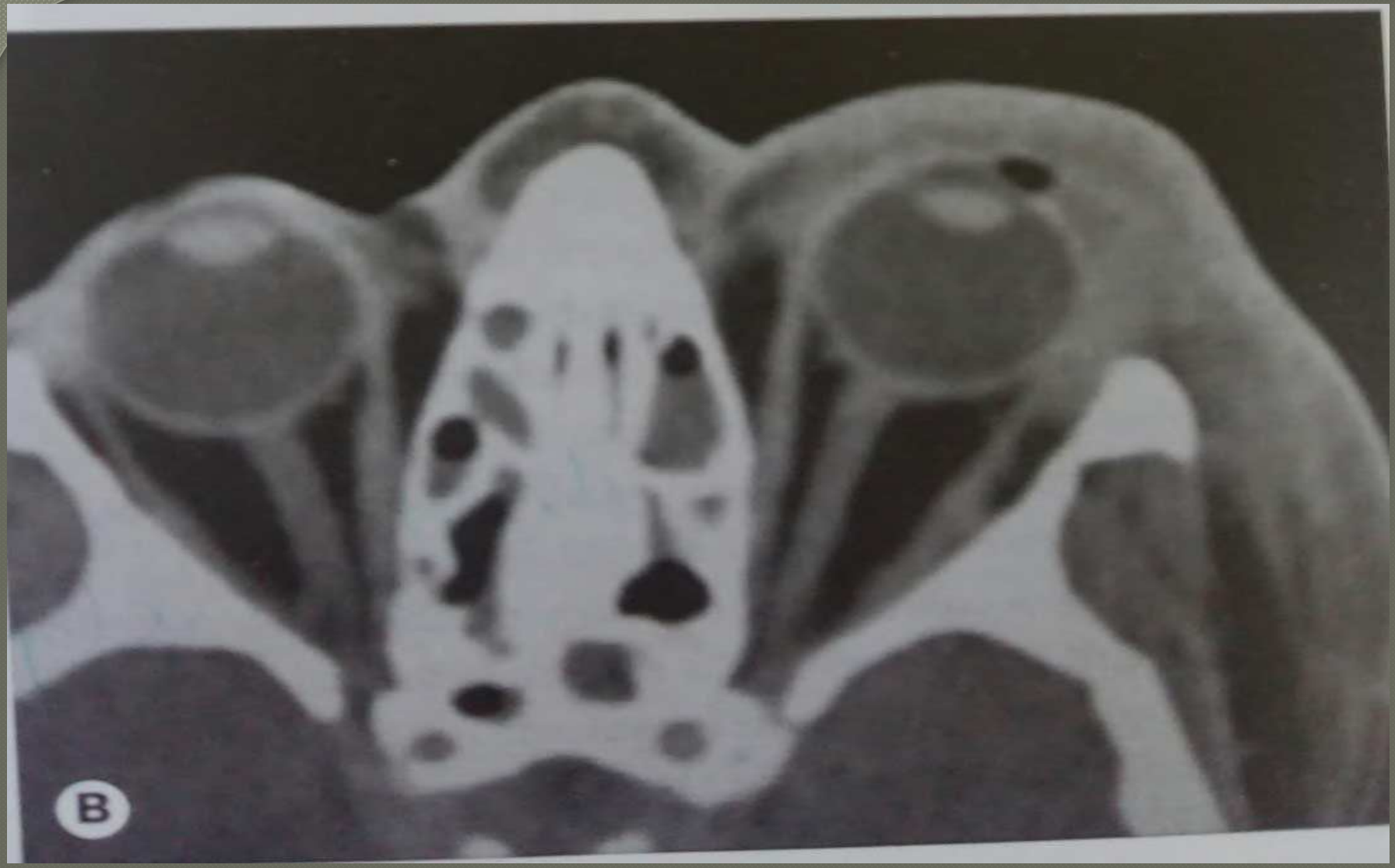
- Fever, malaise painful, swollen lid/periorbital inflamed lids.
- No proptosis, normal eye movements
- White conjunctiva, normal ON function.



Left preseptal cellulitis resulting from an infected eyelid abrasion

Investigations:

- Not usually required
- Needed when there is:
  - Orbital or sinus involvement
  - (opacification anterior to orbital septum)



Axial CT shows opacification anterior to the orbital septum

Treatment:

- Daily review until resolution
- Admit young or unwell children
- Treat with oral antibiotics (e.g. flucloxacillin 500mg 4x/d for 1 wk

Table - Orbital vs preseptal cellulitis

	Orbital	Preseptal
Proptosis	Present	Present
Ocular motility	Painful + restricted	Normal
VA	↓ (in severe cases)	Normal
Colour vision	↓ (in severe cases)	Normal
RAPD	Present (in severe cases)	Absent (i.e normal)

Orbital cellulitis

- Infective organisms include:
  - Streptococcus pneumoniae
  - Staphylococcus aureus
  - Streptococcus pyogenes
  - Haemophilus influenzae (common in children)

Risk factors:

- Sinuses disease
  - Ethmoidal sinusitis
  - Maxillary sinusitis
- Infection of adjacent structures
  - Preseptal
  - Facial
  - Dacryocystitis
  - Dental abscess

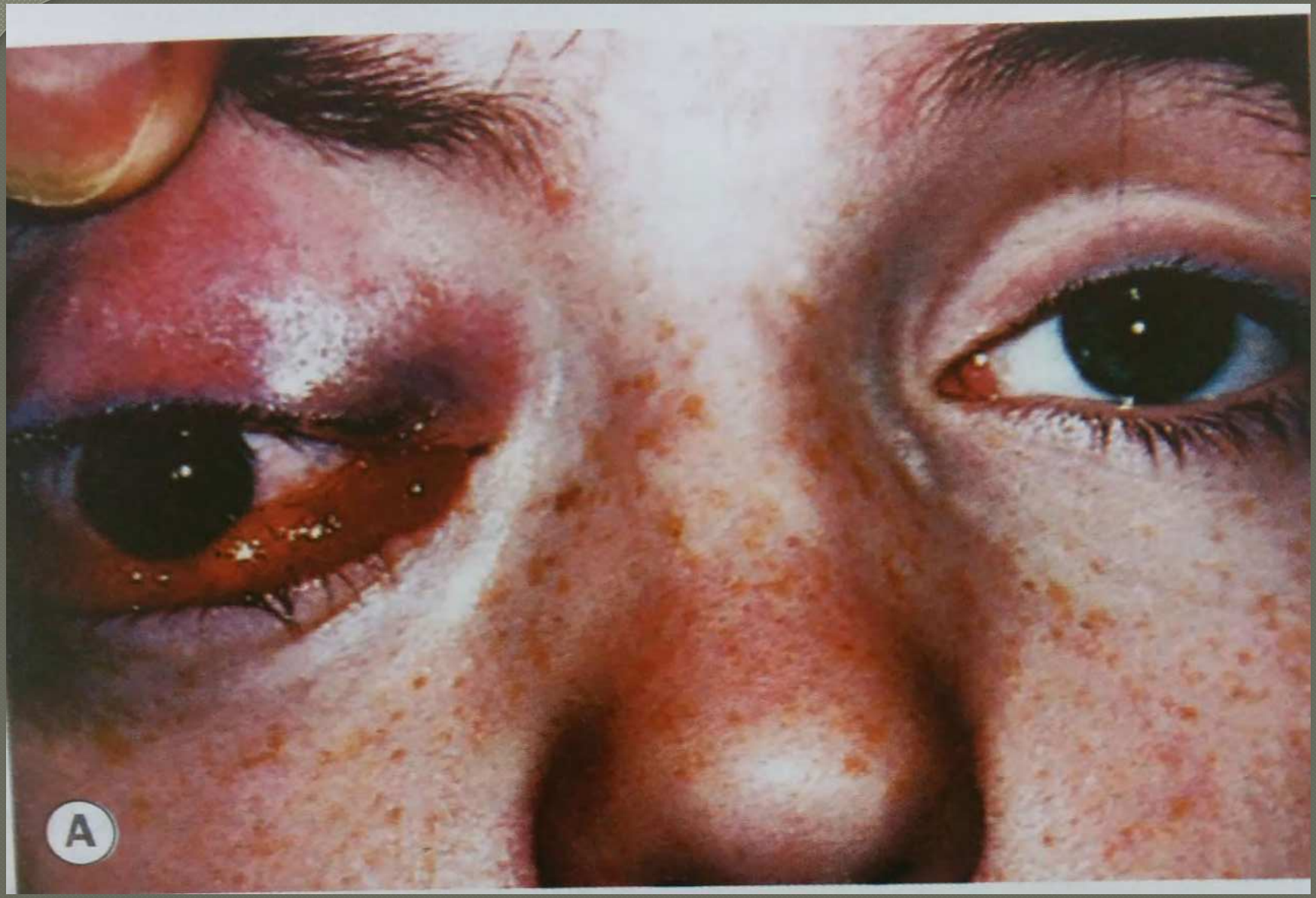
Trauma : Septal perforation
Retained FB

Surgical : Orbital
Lacrimal
VR Surgery

Endogenom spread: In immuno compromised patients

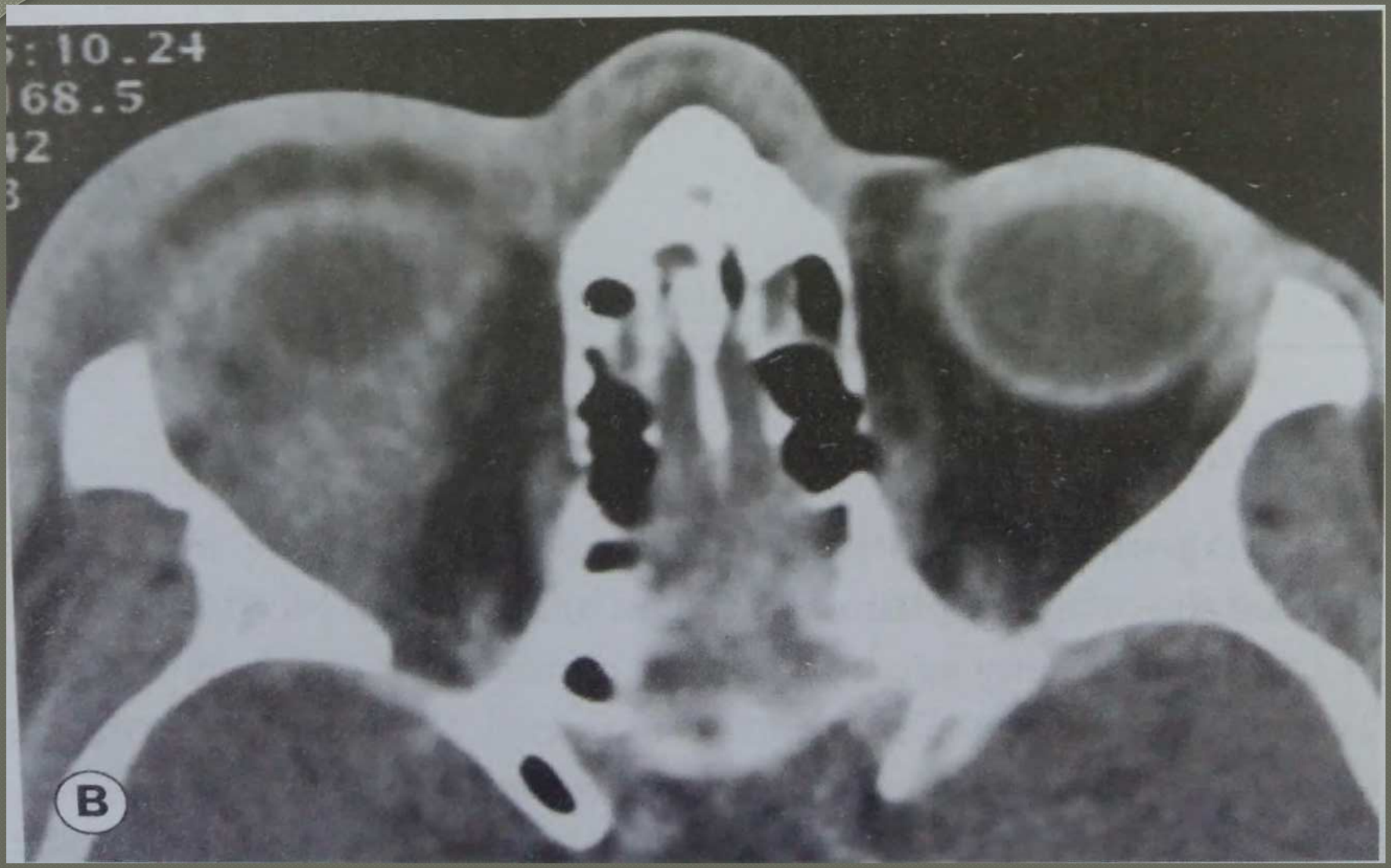
Clinical features:

- Fever, malaise and periocular pain
- Informal lids (swollen, red, tender, warm) ± chemosis, proptosis. Painful restricted eye movements, diplopia, lagophthalmos, optic nerve dysfunction (↓VA,↓colour Vision, RAPD)



Right orbital cellulitis with ophthalmoplegia

- Complications
  - Exposure keratopathy
  - ↑IOP, CRAO, CRVO
  - Inflammation of optic nerve
- Systemic
  - Orbital or paranasal abscess
  - Cavernous sinus thrombosis
  - Meningitis, cerebral abscess



Axial CT shows both preseptal and orbital opacification

Investigation:

- Temperature
- FBC, blood culture (but yield is low)
- CT (orbit, sinuses, brain)

  Orbital abscess

  Diffuse orbital infiltrate, proptosis ± sinus opacity

Treatment:

- Admit for IV antibiotics
- e.g either cefuroxime 750mg – 1.50g 3x/d or ceftriaxone 1 – 2g 2x/d with metranidazole 500mg 3x/d if history of chronic sinus disease
- Mark extent of skin inflammation to monitor status.

- Regular review orbital and visual functions
- ENT to assess for sinus drainage
- If any dereioration repeat CT to exclude abscess formation.

Mucormycosis

- Rare, very aggressive life threatening fungal infection
- Caused by Mucor syp or Rhizopus
- Disease of immunosuppressed
- Seen in patients who are acidotic such as in DKA or renal failure.

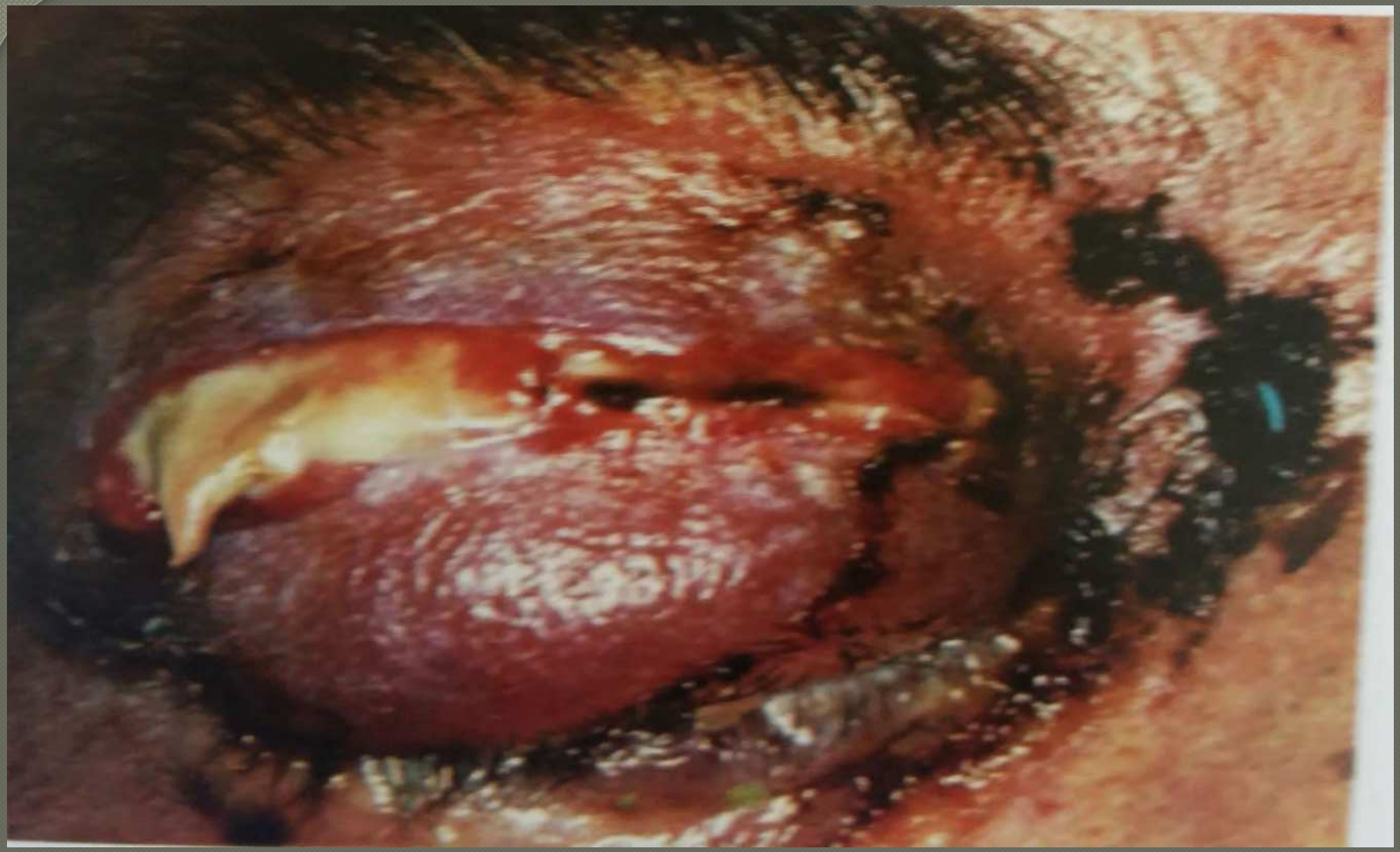
- Also occur in
  - Elderly
  - Malignancy
  - HIV/AIDS

- Immumosuppression
  e.g organ transplant recipients

- Represents fungal septic necrosis and infarction of tissues of nasopharynx and orbit.

Clinical features:

- Black crusty material in nasopharynx
- Acute evolving of eranial neive palsies (III, IV, V, VI, IIn)
- Obvious orbital inflammation.



Necrosis of the eyelid in rhino – orbital mucormycosis

Investigation:

Biopsy: Fungal stains show non septate branching hyphae

FBC : U + E, Glucose

Treatment:

Admit and coordinate care with

- Microbiologist
- Infectious disease Specialist
- ENT Specialist
- Physician

- Correct underlying disease e.g DKA
- IV antifungals (as guided by microbiology e.g high dose amphotericin
- Hyperbaric oxygen therapy
- Aggressive surgical debridement by
  - ENT specialist
  - Orbital exenteration (for unresponsive disease)

Idiopathic orbital inflammatory disease

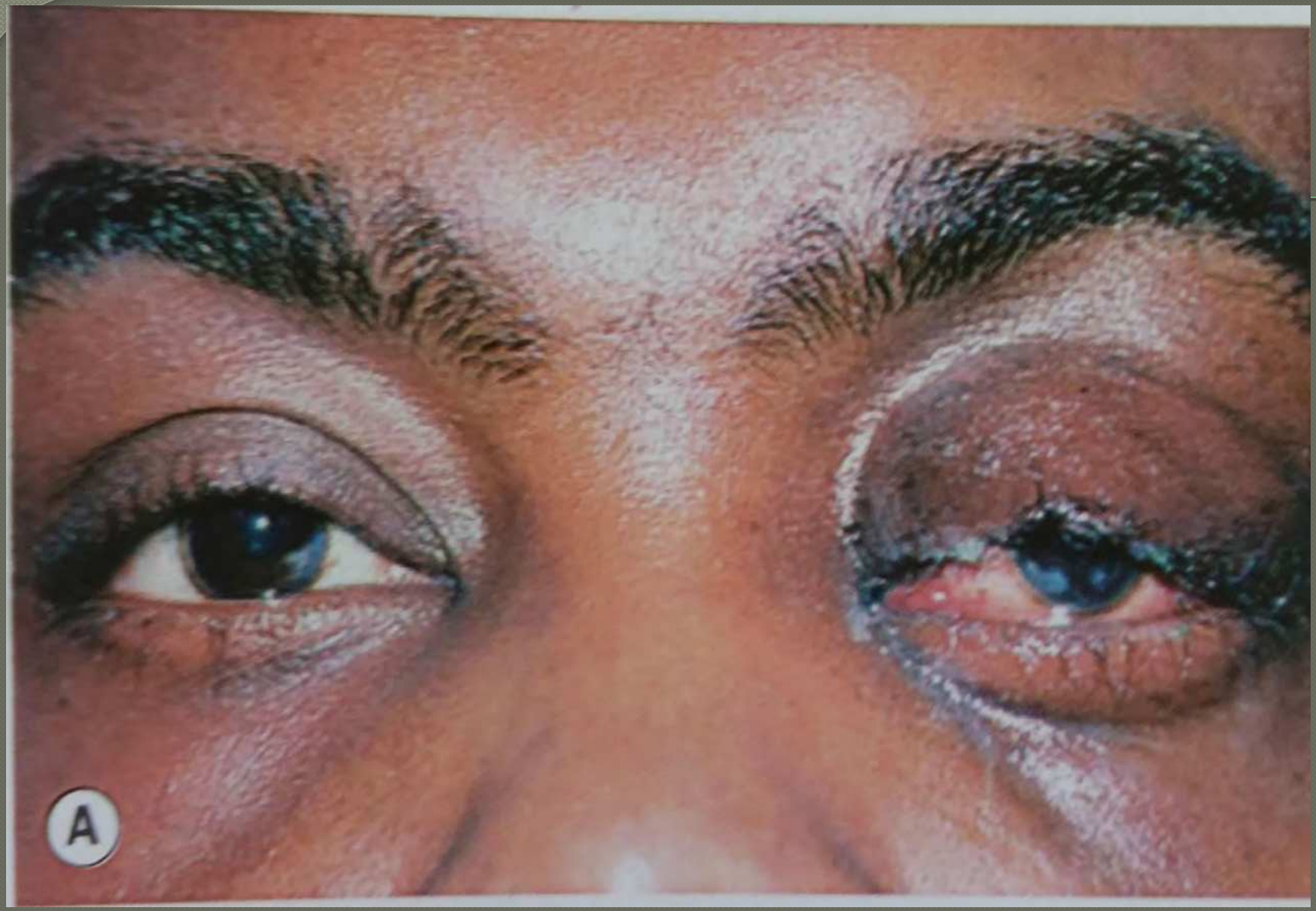
- Chronic inflammatory process of unknown aetiology.
- Inflammation may be predominantly
  - anterior orbit
  - Diffuse
- It may simulate a neoplastic mass.

Histology:

- Pure inflammatory response
- No cellular atypia
- Diagnosis of exclusion
- May represent a number of poorly understood entities
- Occur at any age, usually unilateral.

Clinical features:

Acute pain, redness, lid swelling, diplopia, conjunctival injection, chemosis, lid oedema, proptosis, restrictive myopathy, orbital mass.

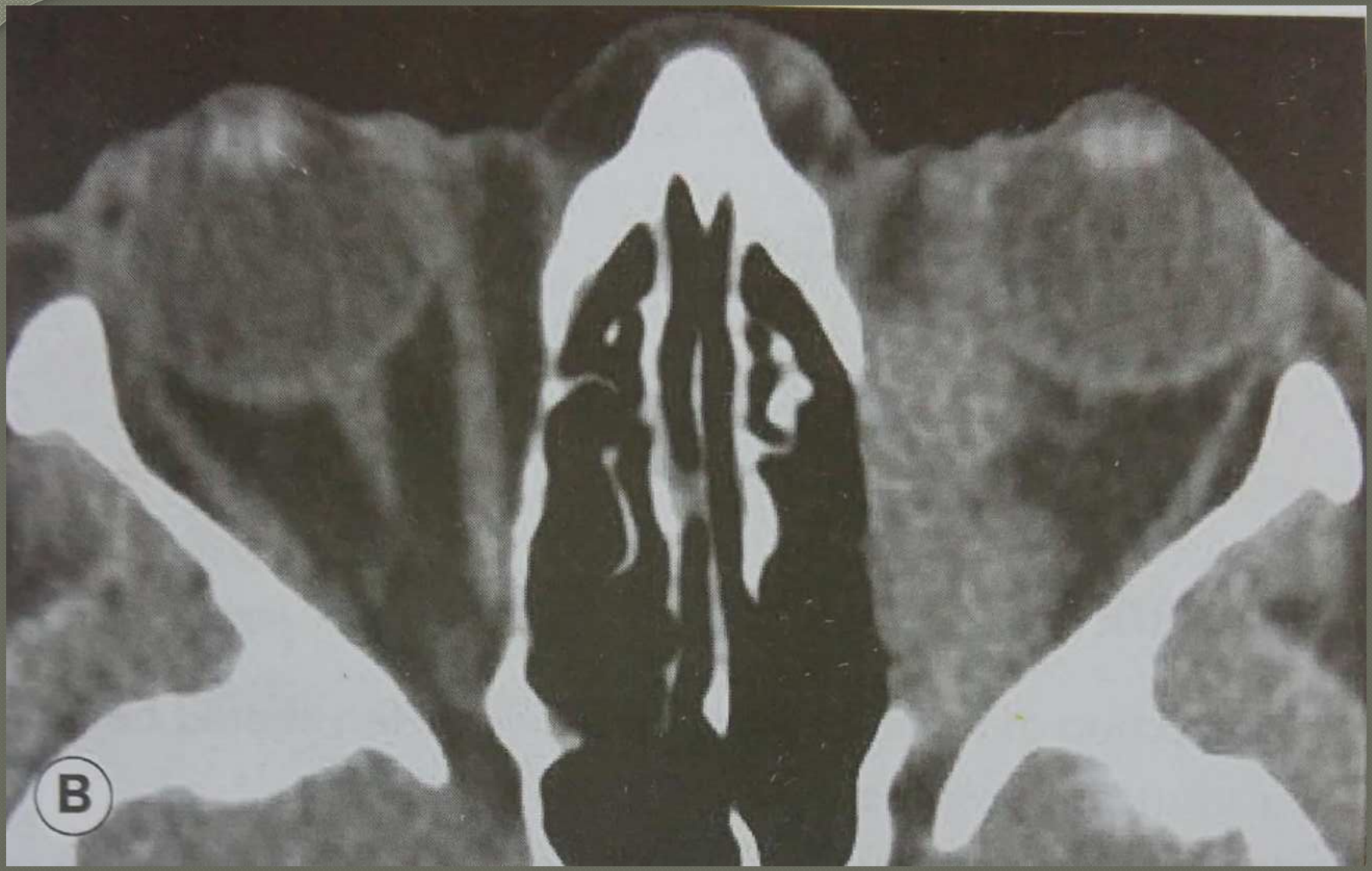


Left idiopathic orbital inflammatory disease

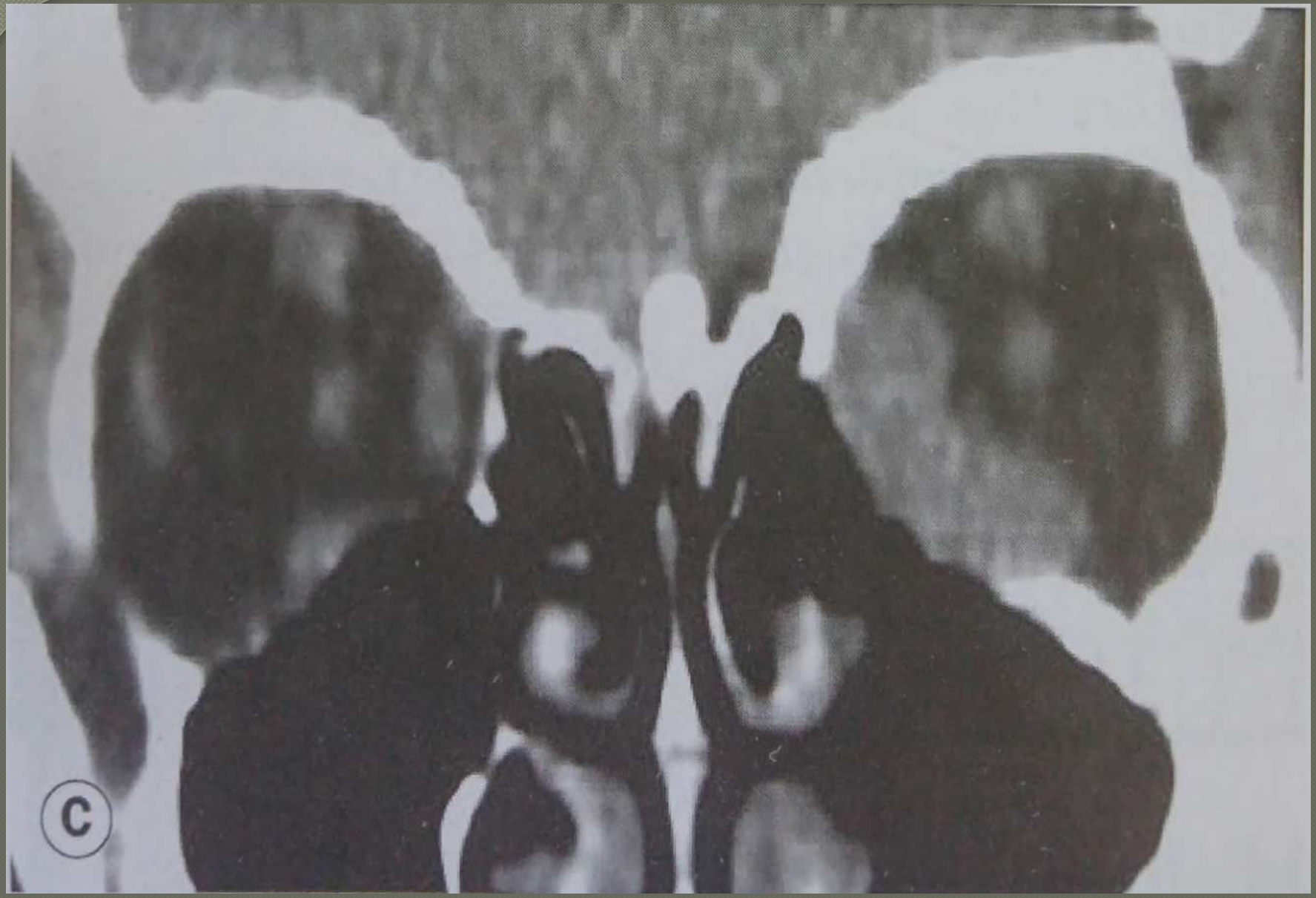
Investigations:

Orbital imaging

- B – Scan → low medium reflectivity
  acoustic homogeneity
- MRI → hypo intense-CF muscle on T1
  hypo intense-CF muscle on T2
- Biopsy → Required to confirm diagnosis



CT axial view shows ill – defined orbital opacification



Coronal view

Treatment:

Immumosuppression

- Usually systemic corticosteroids
- May need cytotoxics e.g
  - Cyclophosphamide
  - Radiotherapy

Differential Diagnosis:

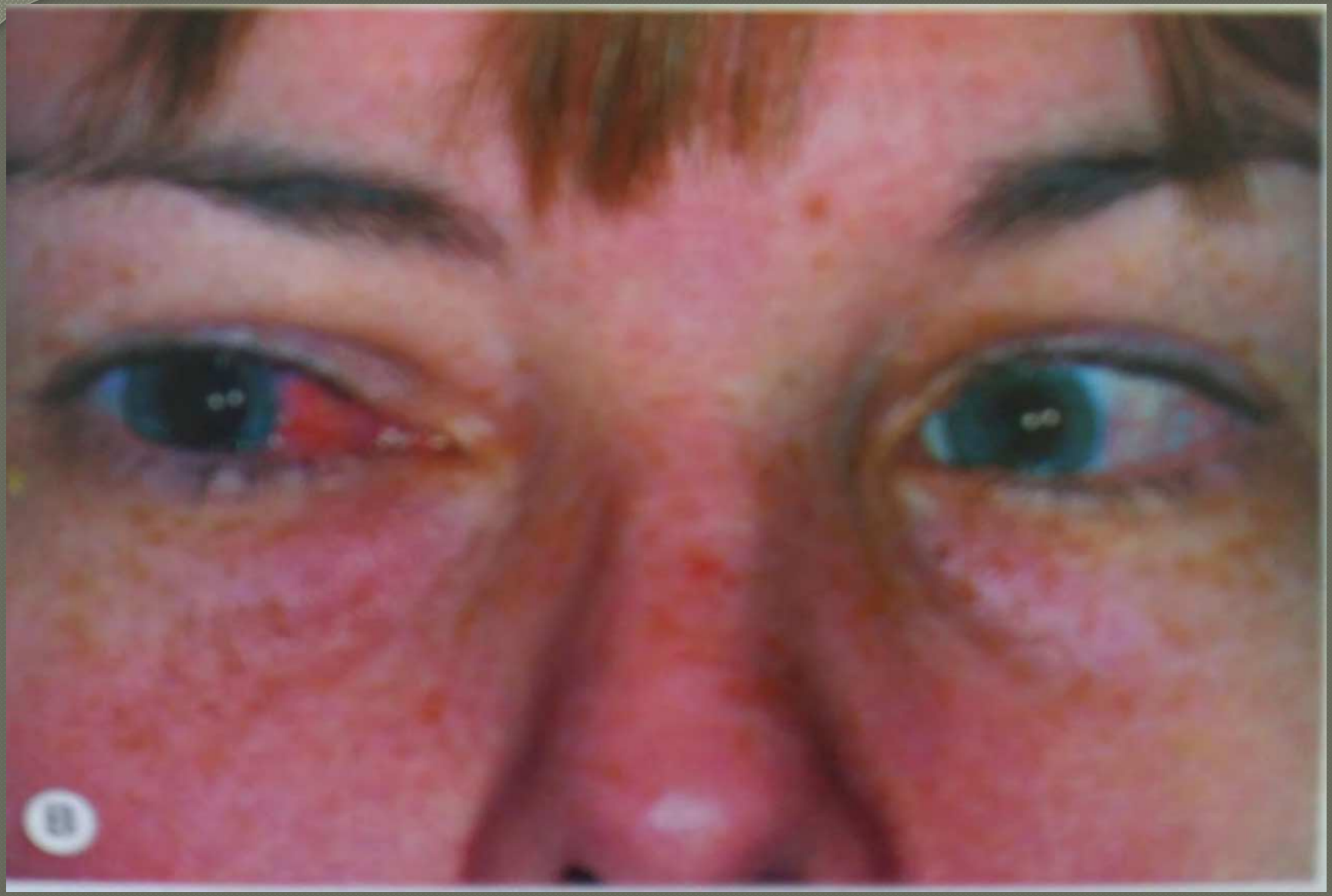
- Orbital cellulitis
- TED
- Wegener's granulomatosis
- Rhabdomyosarcoma
- Metastatic neuroblastoma
- Leukemic infiltration

Myositis

- Idiopathic inflammatory process
- Restricted to one or more extra ocular muscle superior or lateral
- Can occur at any stage
- Unilateral

Clinical features:

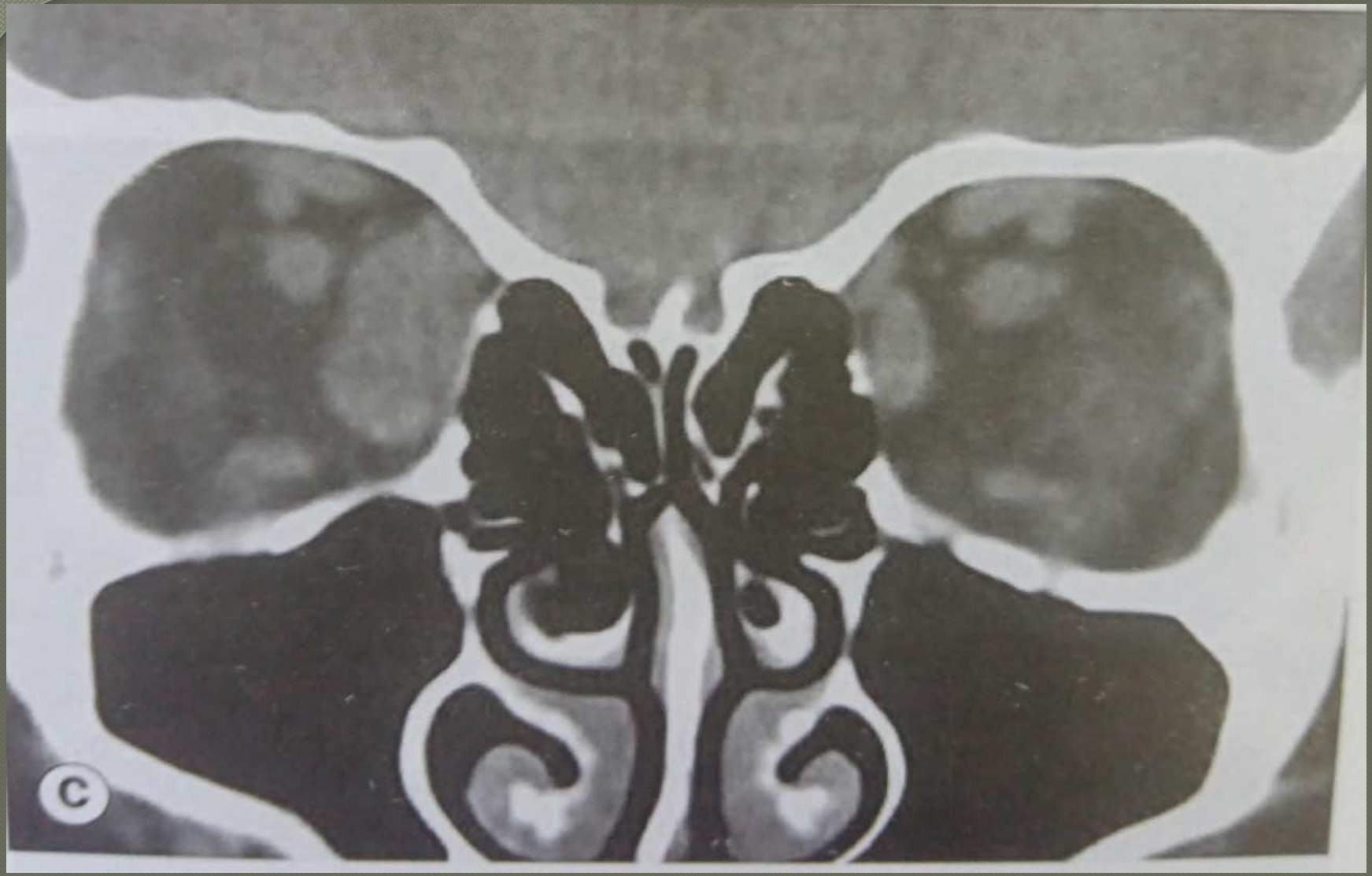
- Acute pain (on movement in direction of involved muscle.
- Injection over muscle
- Mild proptosis
- Repeated episodes – EOM fibrosis, squint



Vascular injection over the insertion of the right medial rectus

Investigations:

- Orbital imaging
  - CT Scan – show enlargement
  - MRI – better soft tissue resolution
- The whole of the muscle and tendon insertion – enlargement and inflammation.



Coronal CT shows enlargement of the right medial rectus

Treatment:

Immumosuppression:

- very sensitive to systemic steroids
- Radiotherapy if recurrent / chronic or poor response to steroids.

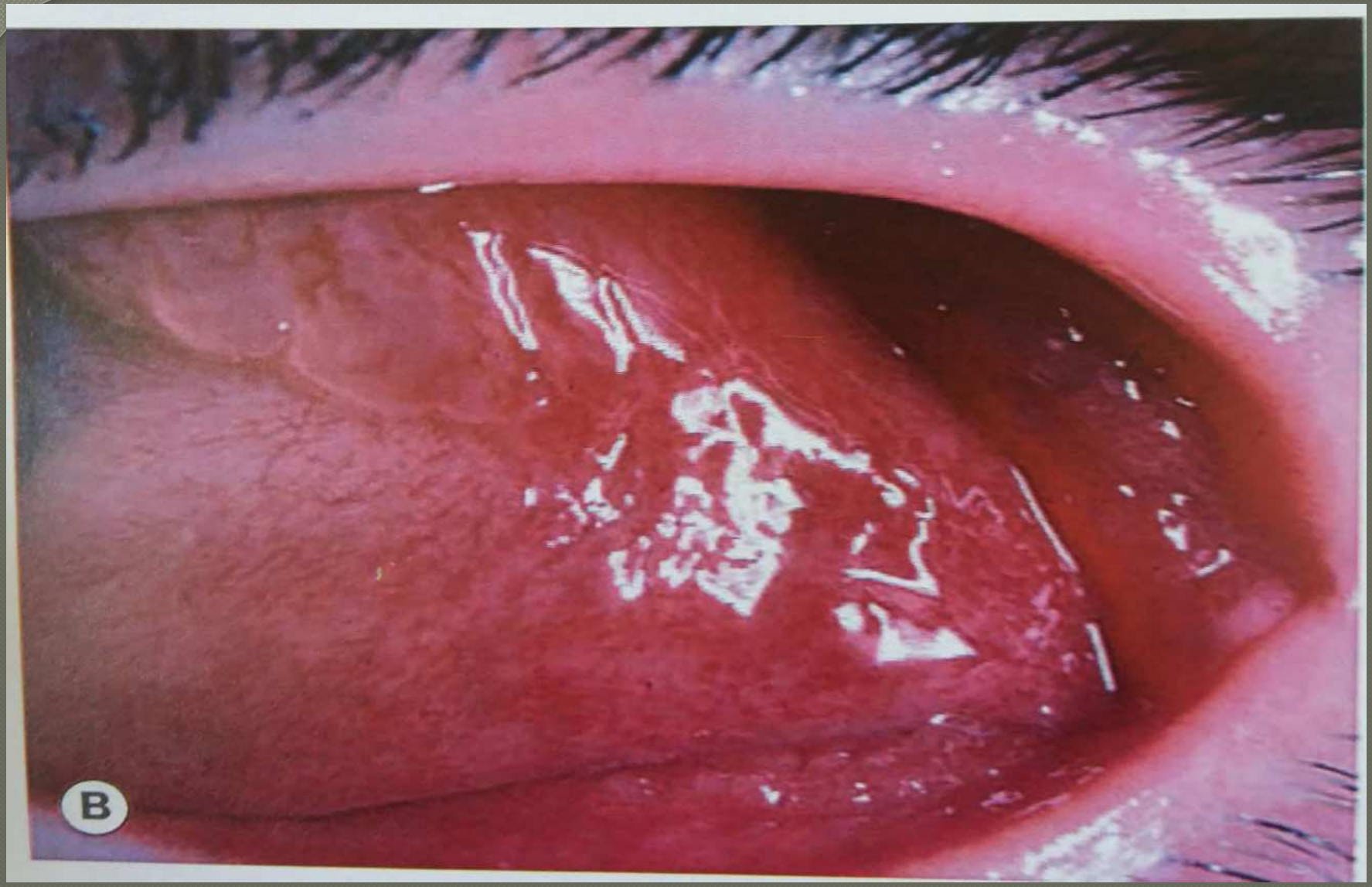
Biopsy – if treatment responsive poor / Persistent symptoms

Dacroyoadenitis

- Lacrimal gland inflammation
- Isolated
- Occur as part of diffuse idiopathic orbital inflammatory disease.

- Presents with acutely painful swollen lacrimal gland – tender to palpation
- Has reduced tear production
- S – shaped deformity to the lid and upper lid causing ptosis.

Swelling on the lateral aspect of the eyelid and an S – shaped ptosis



Injection of the palpebral portion of the lacrimal gland and adjacent conjunctiva

Differential diagnosis:

- Infection – e.g Mumps, EBV, CMV
  Sarcoidosis, Sjogren's syndrome

- Isolated dacryoadentis – responds to
  oral NSAIDS (flurbiprofen 100mg3x/d
  oral steroids.

- Complete resolution – 3 months
- Orbital imaging & biopsy – indicated if inflammation persists.

Tolosa – Hunt syndrome

- Rare idiopathic condition
- Focal inflammation of superior orbital fissure ± orbital apex ± cavernous sinus involvement.

- Presents with orbital pain cranial nerve palsies, proptosis.
- Neuro – imaging required for diagnosis.
- Very sensitive to steroids.

Differential diagnosis:

- Other causes of superior orbital fissure syndrome.
  - GPA
  - CCF

- Cavernous sinus thrombosis
- GPA, Pituitary apoplexy
- Sarcoidosis, mucormycosis

Wegener's granulomatosis

- Uncommon, severe necrotizing granulomatosis vasculitis
- Have ophthalmic involvement in upto 50% of cases
- Orbital involvement in 22%
- Common in males and in middle age.

Clinical features:

Ophthalmic

- Orbital disease – Pain, proptosis
  Restricted myopathy
  Disc swelling, ↓ VA
- Other ocular disease – Episcleritis, scleritis, PUK, uveitis and Vasculits

Systemic:

- Preumonitis
- Glomerulonephritis
- Sinusitis
- Nasopharyngeal ulceration

Investigations:

- **ANCA** – Most cases- cANCA positive

- **CT Scan** – Obliteration of orbital fat planes by a plaque like infiltrative mass. Erosion and destruction of sinus and nasal bones.

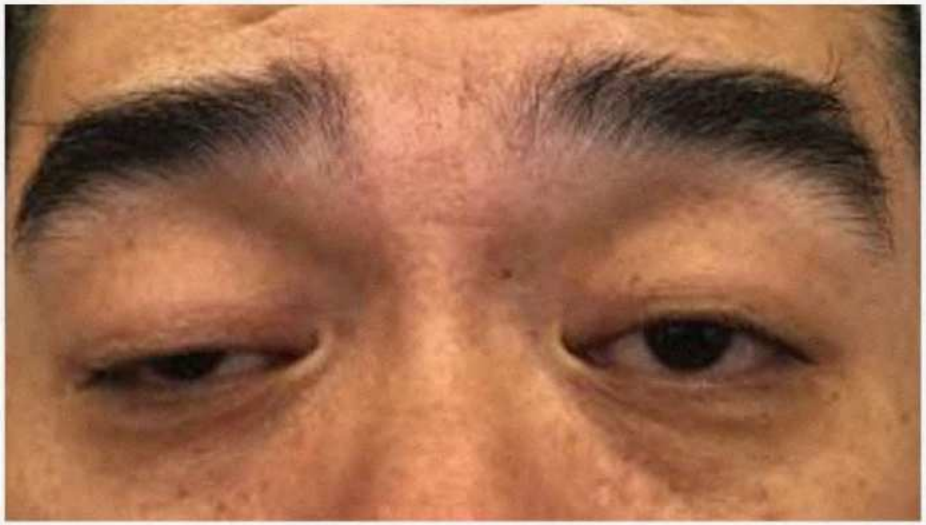
Treatment:

Coordinated by Rheumatologist & Physician

Usually combined corticosteroids, cyclophosphamide or retuximab.

THANKS

Myasthenia Gravis



Prof SofiaIqbal
FRCS, MRCOphth
Fellowship Orbit/Oculoplastics
Fellowship Refractive surgery

Presentation lay out

- Introduction
- Definition
- Etiology
- Pathophysiology
- Role of thymus gland
- Types
- Classification
- Diagnosis
- Differential diagnosis
- Management

INTRODUCTION

- **A neurological / neuromuscular autoimmune disorder**
- **Error in the transmission of nerve impulses to muscles at the neuromuscular junction—the place where nerve cells connect with the muscles they control**
- Antibodies to the **acetylcholine receptor (AChR)**,nicotinic receptors found in the serum of 85% of patients
- **Affects 1 in 10,000 population**
- **Leads to weakness and fatigability**

Potential Risk Factors for Developing Myasthenia Gravis

Women 20-40 years old and men 50-80 years old

People who have rheumatoid arthritis or lupus

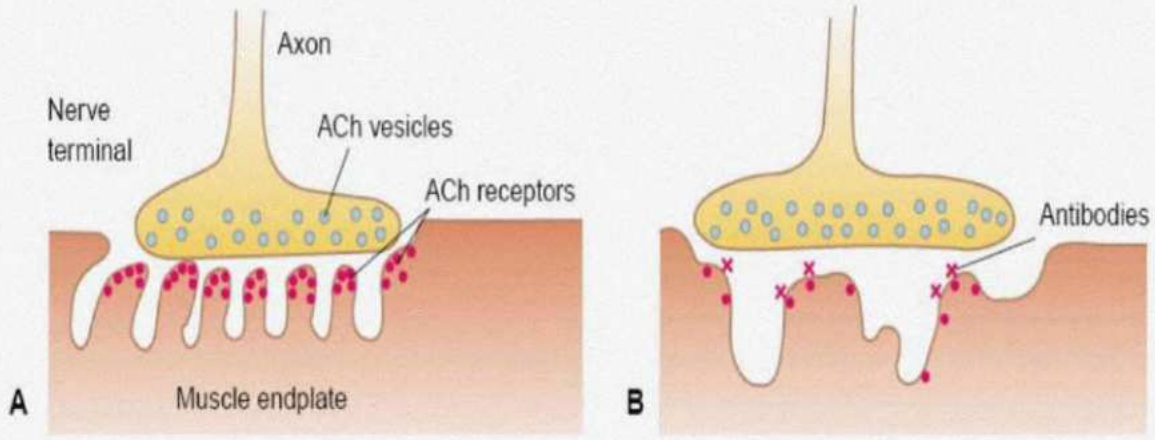
Taking certain medications for malaria, heart arrhythmia, antibiotics and psychiatric drugs

Having undergone extensive surgeries in the past

Issues with the thyroid gland

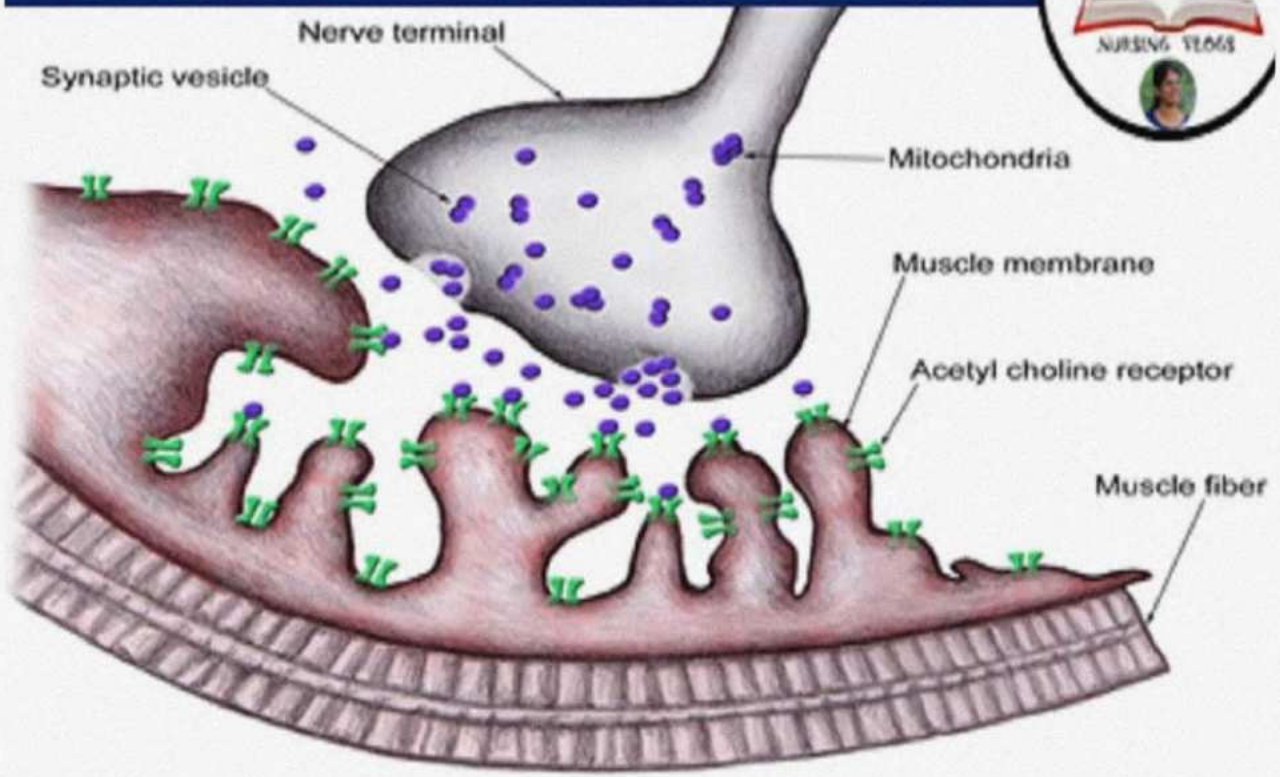
PATHOPHYSIOLOGY

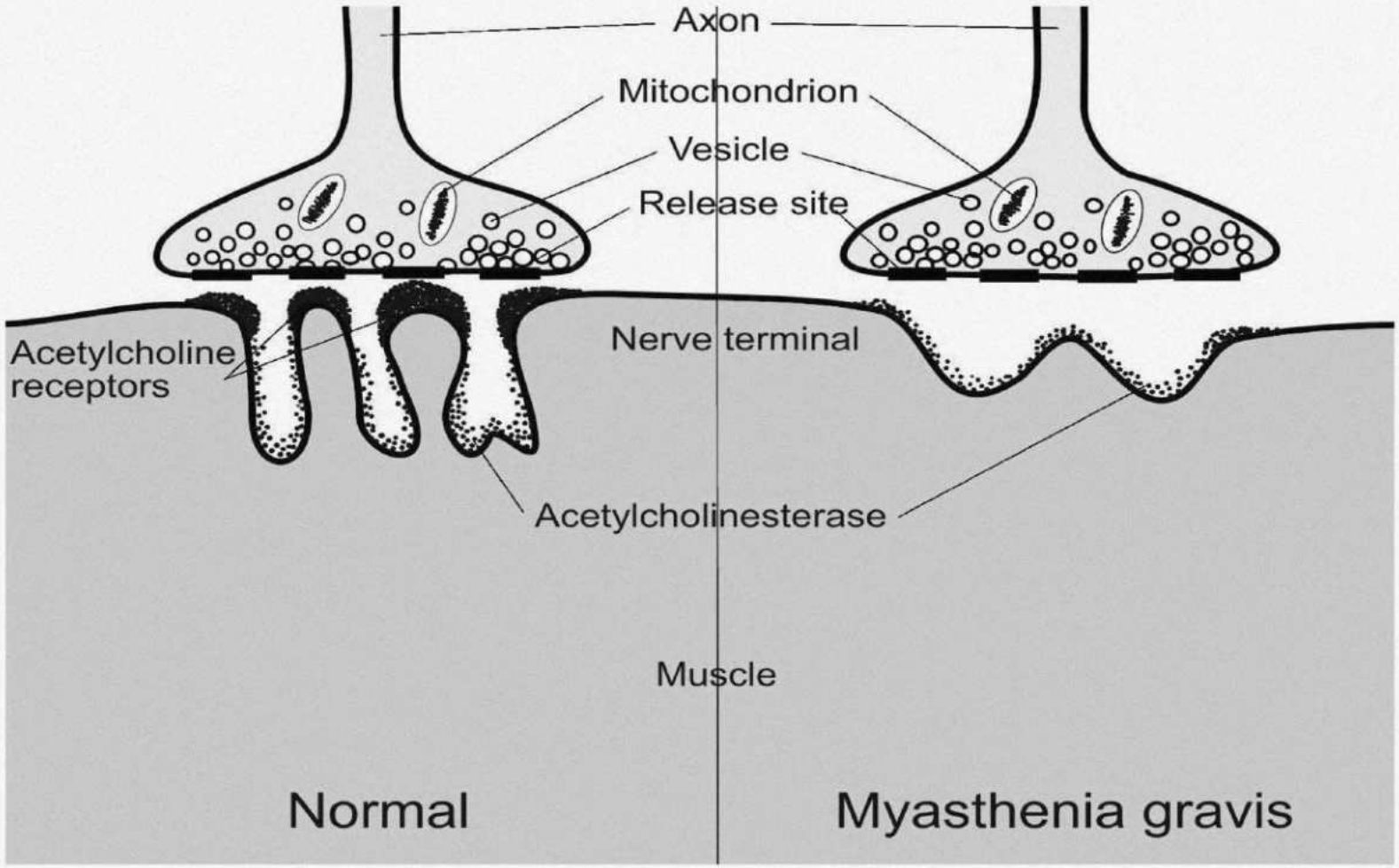
- Normally, a chemical impulse precipitates the release of acetylcholine from vesicles on the nerve terminal at the myoneural junction. The acetylcholine continuously bind to the receptor sites on the motor end plate, for muscle contraction to sustain.

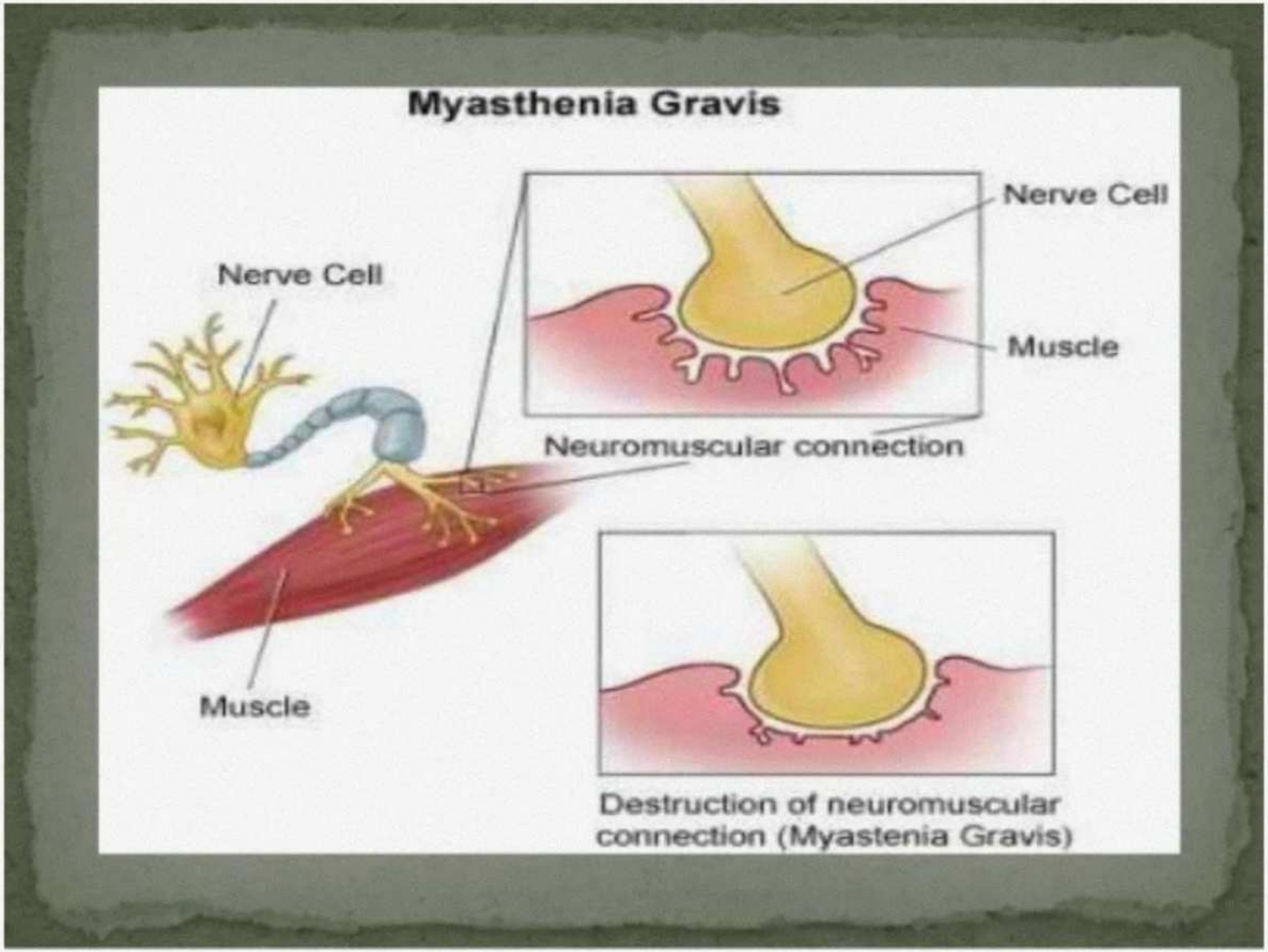


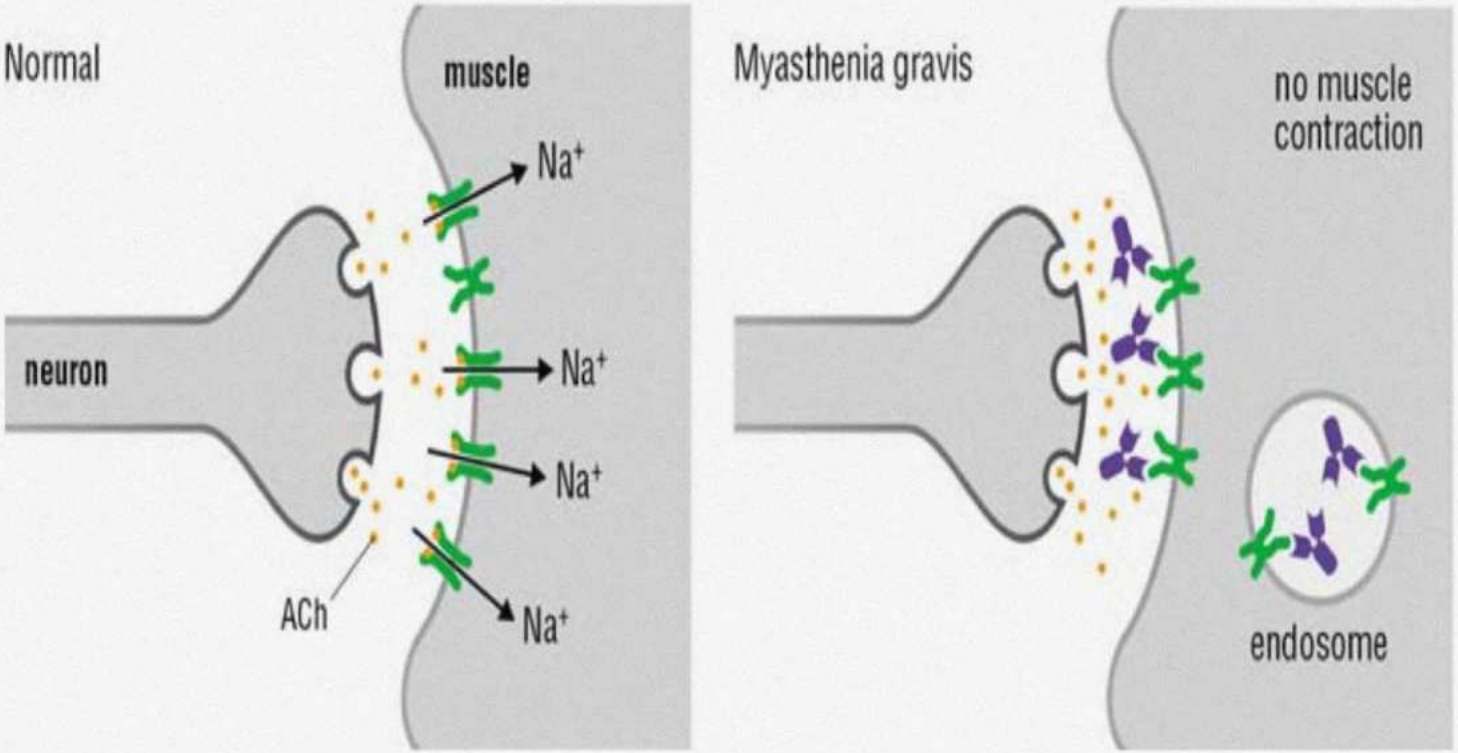
Myasthenia gravis. (A) Normal ACh receptor site. (B) ACh receptor site in myasthenia gravis.

MYASTHENIA GRAVIS



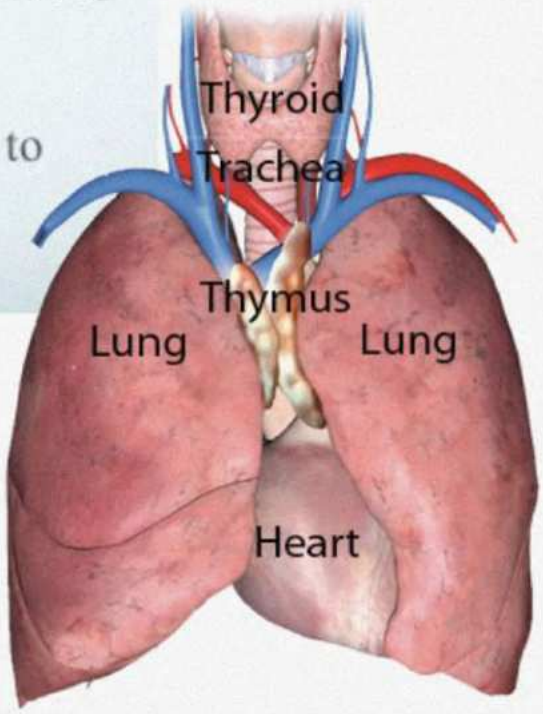

Axon
Mitochondrion
Vesicle
Release site
Acetylcholine receptors
Nerve terminal
Acetylcholinesterase
Muscle
Normal
Myasthenia gravis

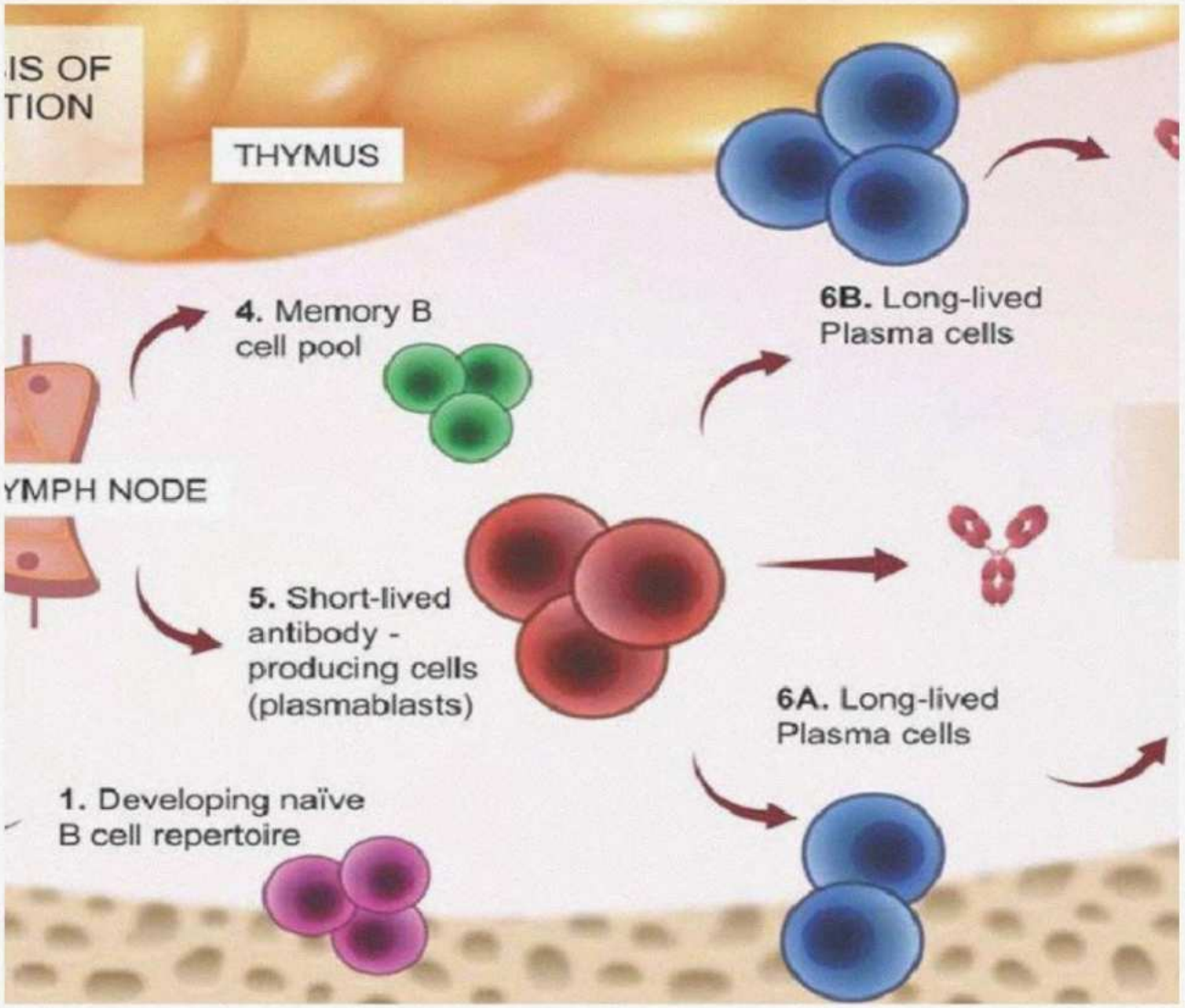

Myasthenia Gravis
Nerve Cell
Nerve Cell
Muscle
Neuromuscular connection
Muscle
Destruction of neuromuscular connection (Myastenia Gravis)



ROLE OF THYMUS GLAND

- The factors that trigger the autoimmune process are not known, but the thymus gland is involved.
- The thymus lies behind the sternum and may extend down to the diaphragm or up to the neck.
- This gland plays a role in the responsiveness of T cells to foreign antigens.





Pathophysiology:-

- Due to etiological factors

↓

- Lymphocyte produce acetylcholine receptor antibodies that attack the post synaptic muscle membrane

↓

- Depletion of acetylcholine receptor of the neuromuscular junction

↓

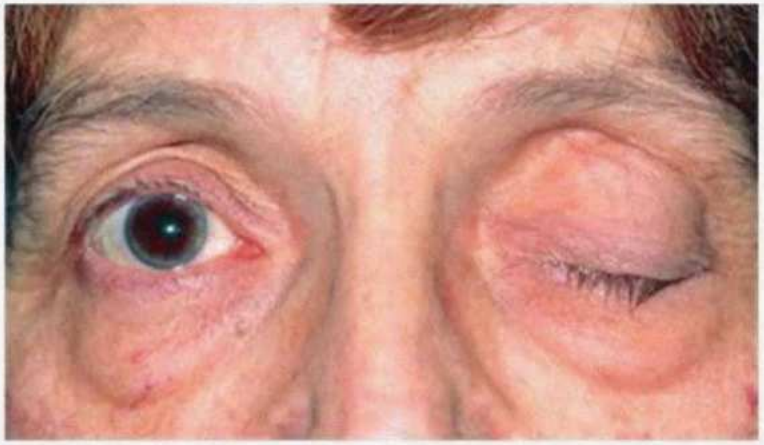
Defect in the transmission of impulse from nerve to muscle cell

↓

- Myasthenia gravis

Clinical Manifestations

- Muscle weakness
- Double vision (diplopia)
- weak eyelids (unilateral ptosis)
- Difficulty speaking or smiling
- Difficulty chewing and swallowing



TYPES OF MG

- **OCULAR/BULBAR/GENERALIZED**

OCULAR

- Diplopia
- Ptosis
- Ophthalmoplegia

RESPIRATORY

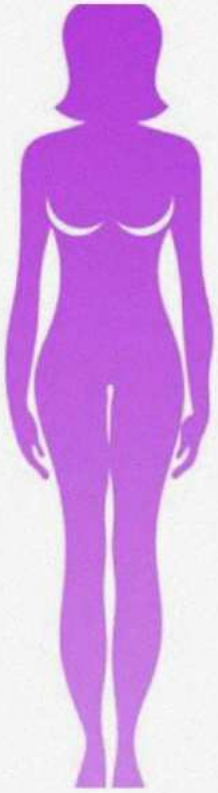
- Breathlessness
- Weak breathing
- Respiratory failure

BULBAR

- Fatiguable chewing
- Dysarthria
- Dysphagia

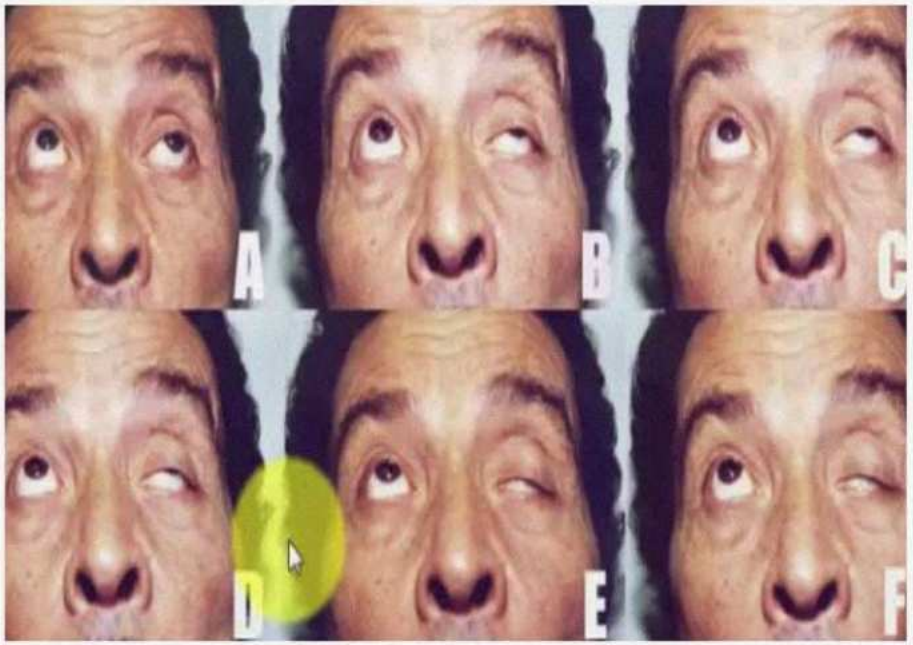
LIMBS, NECK

- Dropped head
- Proximal > distal
- Arms > legs



OCULAR MYASTHENIA

- **Ocular myasthenia gravis (OMG) can mimic isolated cranial nerve palsies, gaze palsies, internuclear ophthalmoplegia, blepharospasm, and even a stroke**



Strabismus types	Number of patients (%)
Vertical deviation	6 (28.6)
Exotropia and vertical deviation	5 (23.8)
Esotropia	4 (19.0)
Esotropia and vertical deviation	3 (14.3)
Exotropia	3 (14.3)
Total	21 (100.0)

Classification

- Class I: Eye muscle weakness only
- Class II: Eye muscle weakness
  + mild weakness of other muscles
- Class III: Eye muscle weakness
  + moderate weakness of other muscles
- Class IV: Eye muscle weakness
  + severe weakness of other muscles

  OR need for nasogastric feeding
- Class V: Intubation needed to maintain airway

Diagnosis: CLINCAL, SEROLOGIC AND EMG FINDINGS

1.Clinical DX:

-Bedside: ice pack test/ Edrophonium test

- Cogan sign
- Peek sign

Imaging:

CT CHEST: Evaluate for thymoma

2.Electrophysiologic confirmation:

-Repetitive nerve stimulation

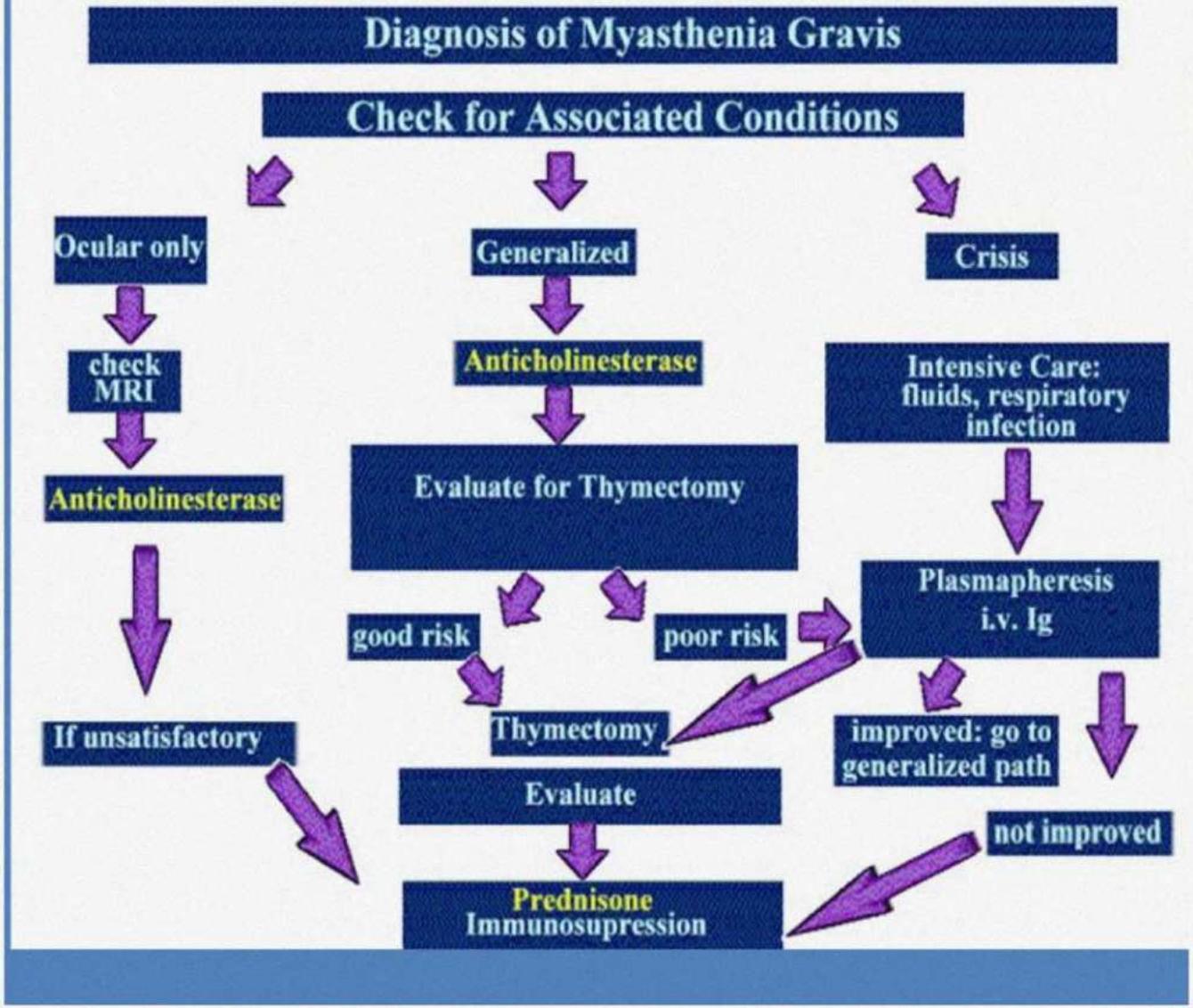
-Single fiber electromyography

3.Labs:

AchR antibodies- first step in immunologic assay

MuSK antibodies

LRP4 antibodies



Test	Positive Result
Fatigue test	Worsening of symptoms after prolonged use
Ice test or sleep test	Improvement of ptosis after ice pack application or period of rest
Edrophonium (Tensilon or Enlon)	Improvement in symptoms within 30-60 seconds
Serologic screening	Identification of circulating AchR, MuSK or LRP4 antibodies
Electrophysiologic testing (RNS, SFEMG)	Decrease in action potential of stimulated nerves
Thyroid panel, thoracic imaging	Used to identify coexisting conditions

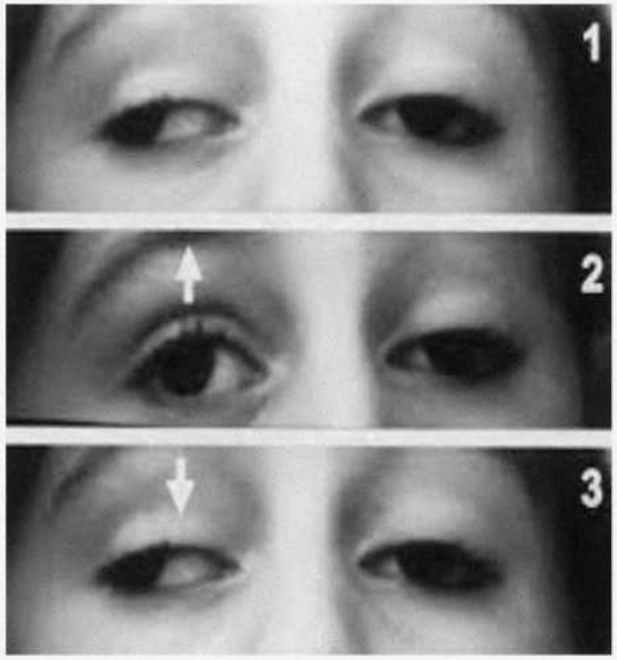
RNS = repetitive nerve stimulation; SFEMG = single-fiber electromyography

ICE PACK TEST

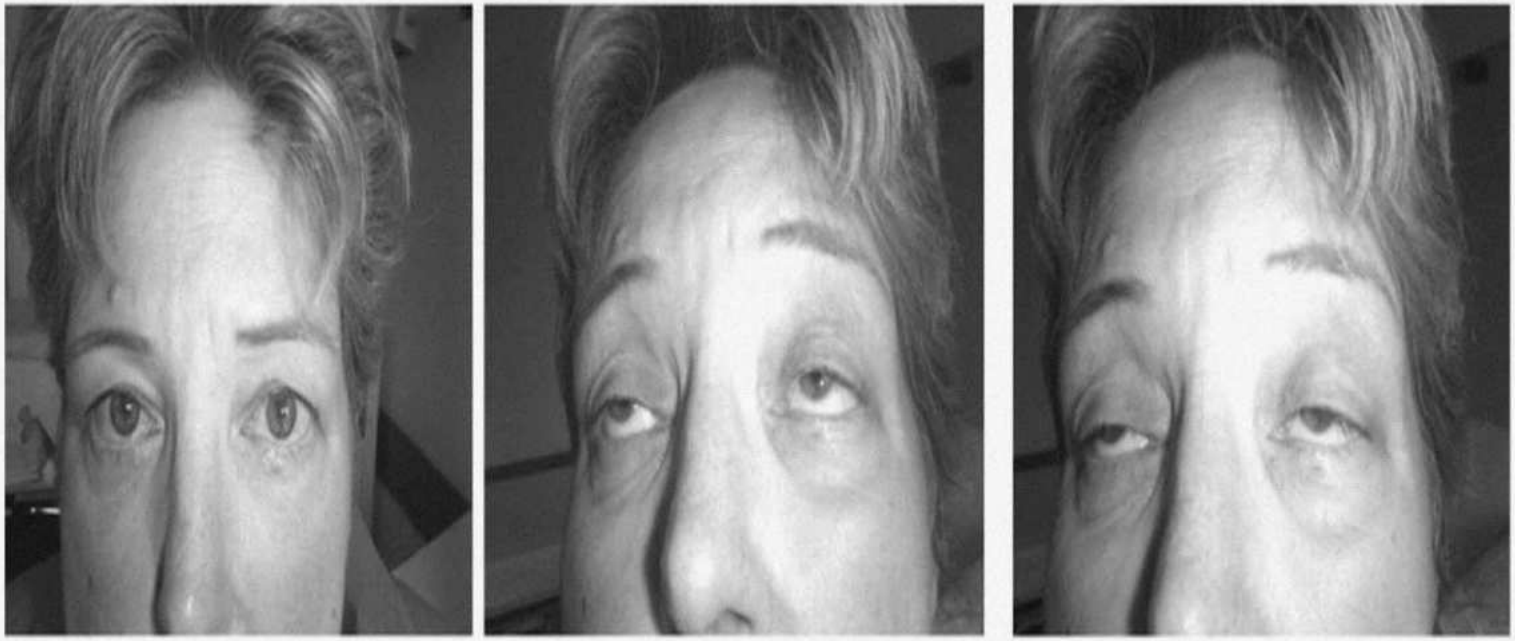
- Apply ice pack for 3 to 5 minutes
- Bed side test
- **Cold improves neuromuscular transmission**
- **Sensitivity of 85%**

Cogan's sign

- Ask the patient to gaze downward for 10–15 seconds and then returning to primary gaze
- Cogan's sign is present **when the affected lid briefly "twitches" upward on returning to primary gaze**

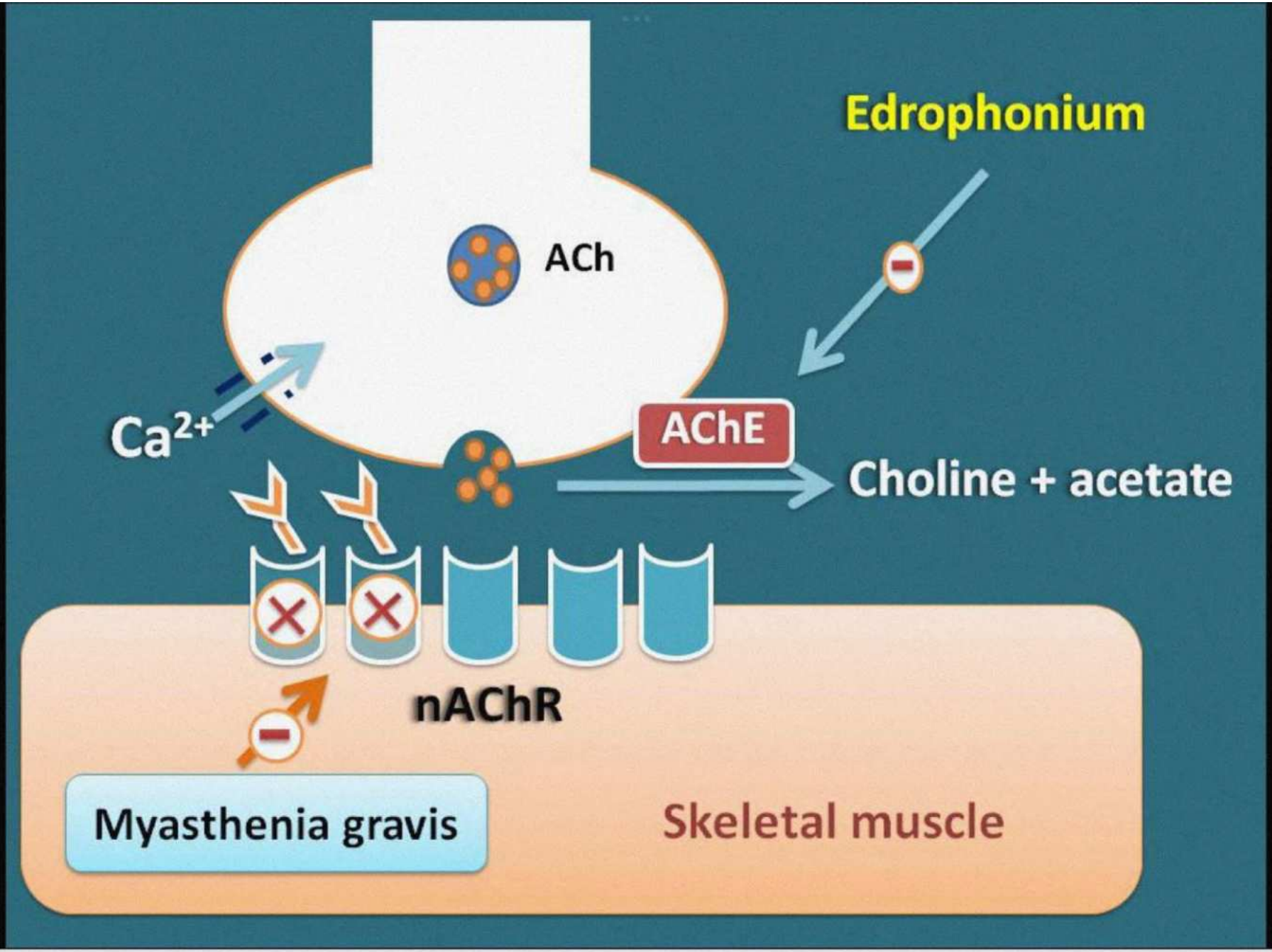


FATIGUIBILITY TEST



Tensilon Test

- Edrophonium chloride
  - Inhibits acetylcholinesterase
- Onset 30 seconds; duration 5-10 min
- NEED A CLEAR OBJECTIVE ENDPOINT
  - Works best with complete ptosis
- Compare to placebo (saline)
- Prepare atropine
- Give test dose 1-2 mg then up to 10 mg total
- SFX:
  - salivation, sweating, nausea, abdo cramping, fasciculations; hypotension & bradycardia are rare (may be as low as 0.16%)
- Sensitivity 71.5- 95%
- Specificity: not clear but can be positive in many other conditions (even ALS or normal controls)
- Not availible



How it is given?

Initial safety check

Edrophonium

2 mg by IV

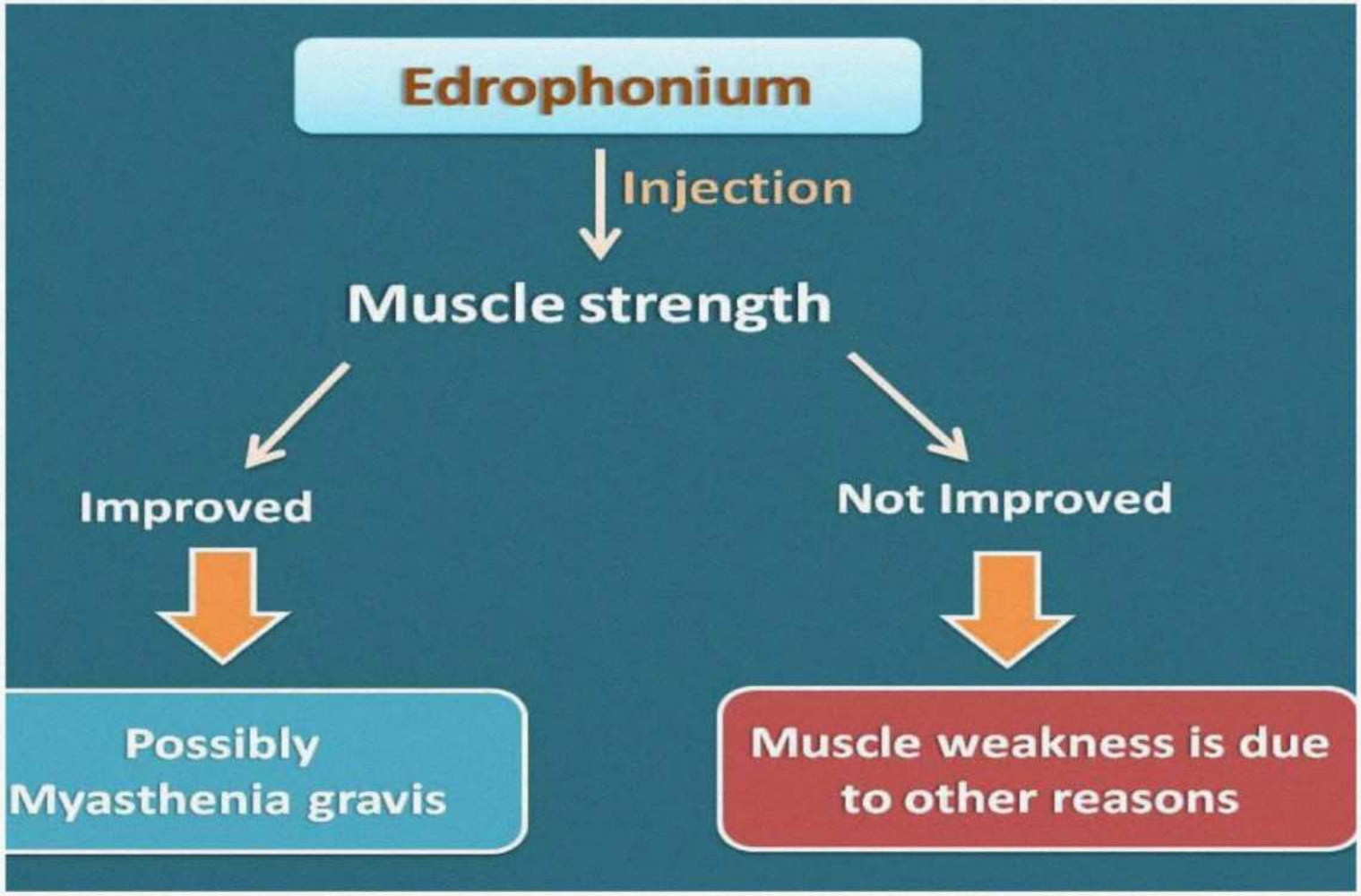
Check for any side effects

Diagnosis step

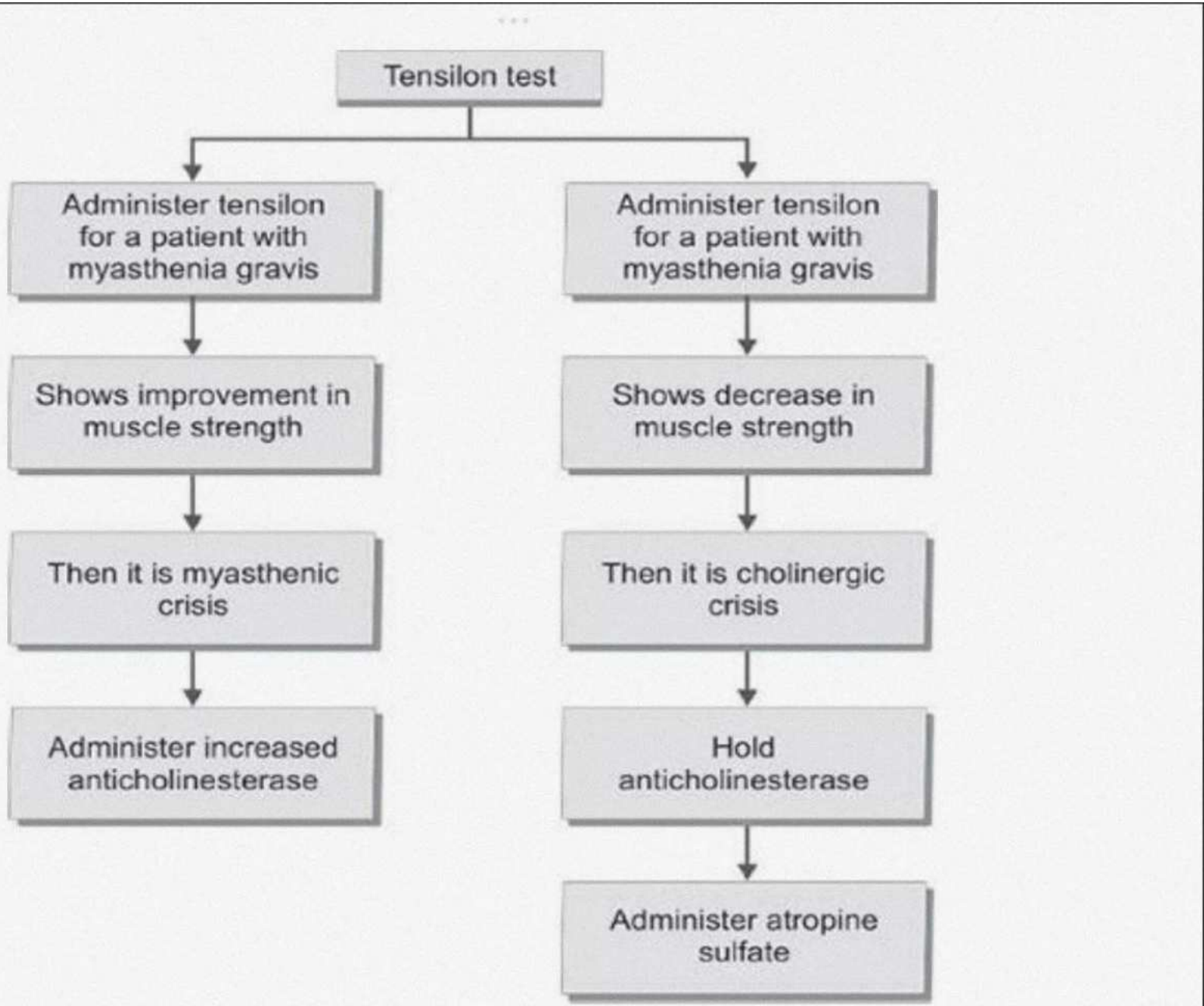
Edrophonium

8 mg by IV

Check for improvement in muscle strength



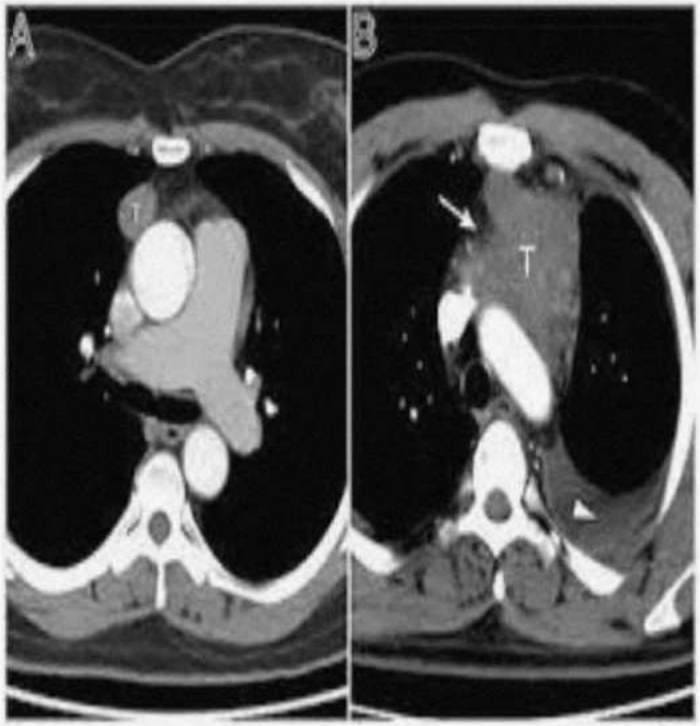
Tensilon test

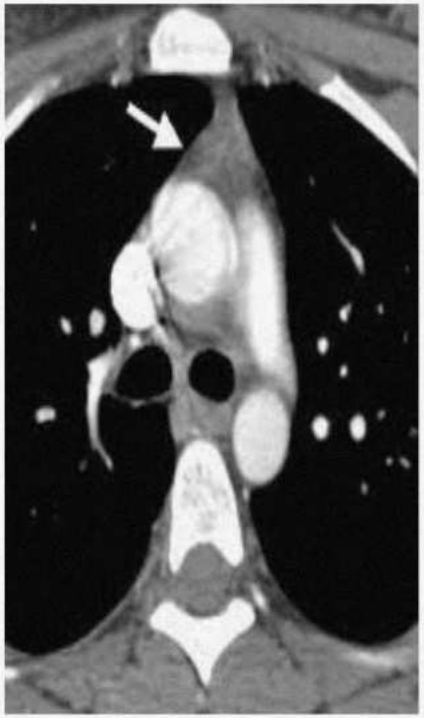


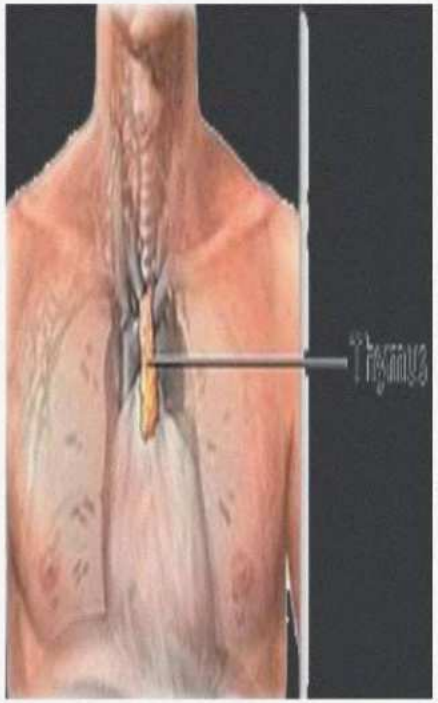
Myasthenic Crisis vs. Cholinergic Crisis

- Mysathenic Crisis
  - Serious complication where patients are unable to breathe adequately and possibly develop respiratory failure
  - Impaired swallowing and managing of secretions leading to aspiration
  - Monitory NIF, vital capacity, tidal volume
- Cholinergic Crisis
  - Due to and excess of acetylcholine at the NMJ as seen in organophosphate poisoning
  - Fasiculations, sweating, myosis, abdominal pain, bradycardia
  - Flaccid paralysis and respiratory failure
- Differentiate with edrophonium test

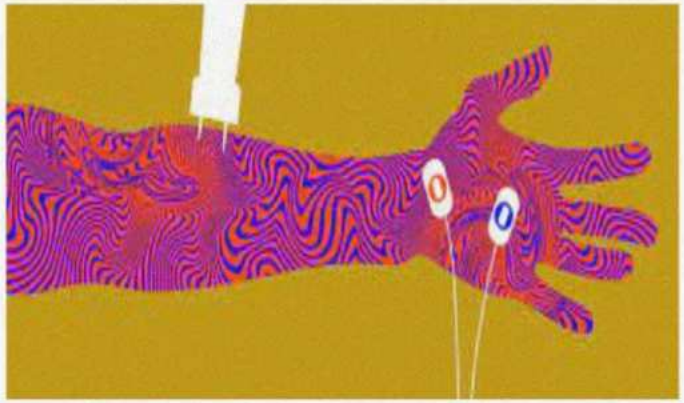
THYMOMA RADIOLOGY

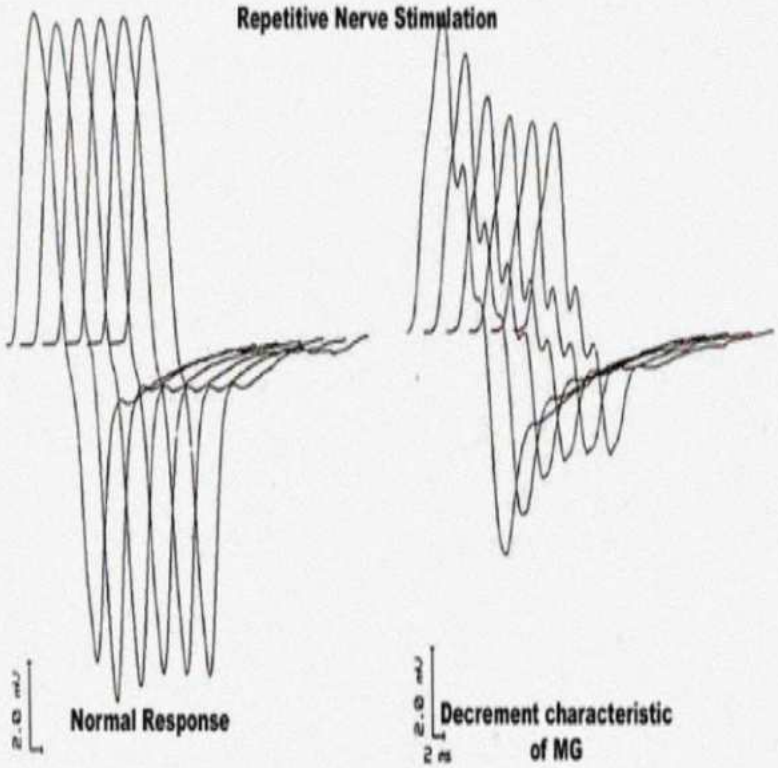




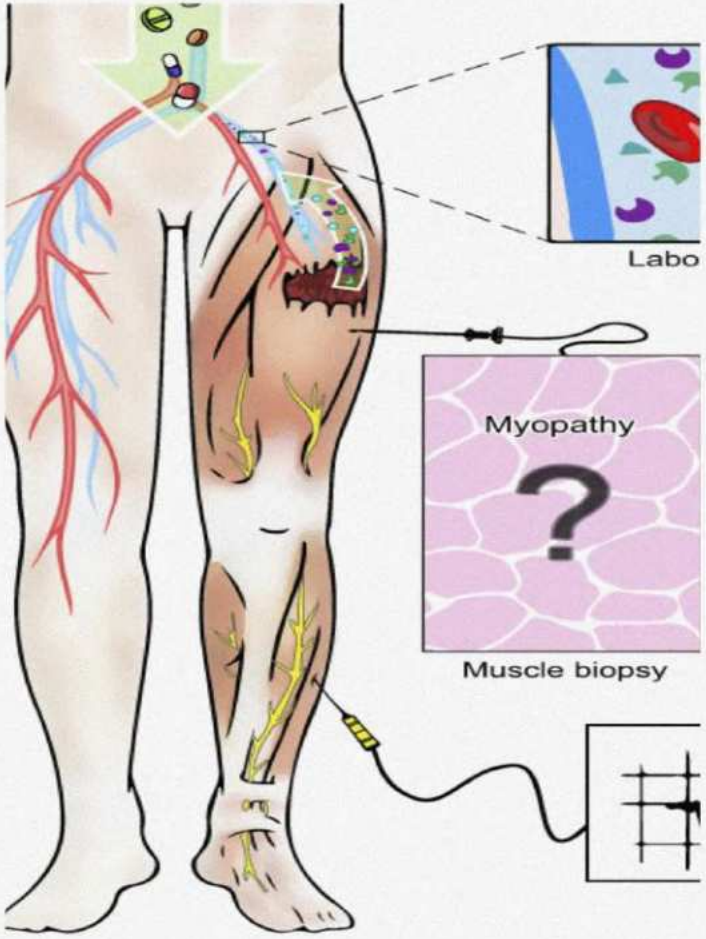


ELEECTROMYOGRAPHY
EMG Studies





MUSCLE BIOPSY



DIFFERENTIAL DIAGNOSIS

Thyroid opthalmopathy
Kearns-Sayre syndrome
Myotonic dystrophy
Brain stem/ Cranial nerve pathology
Generalized fatigue
ALS
Lambert Eaton myasthenia syndrome
Miller Fischer and PCB variants of GBS
Botulism
Penicillamine induced myastheniay

Lambert Eaton myasthenic syndrome:

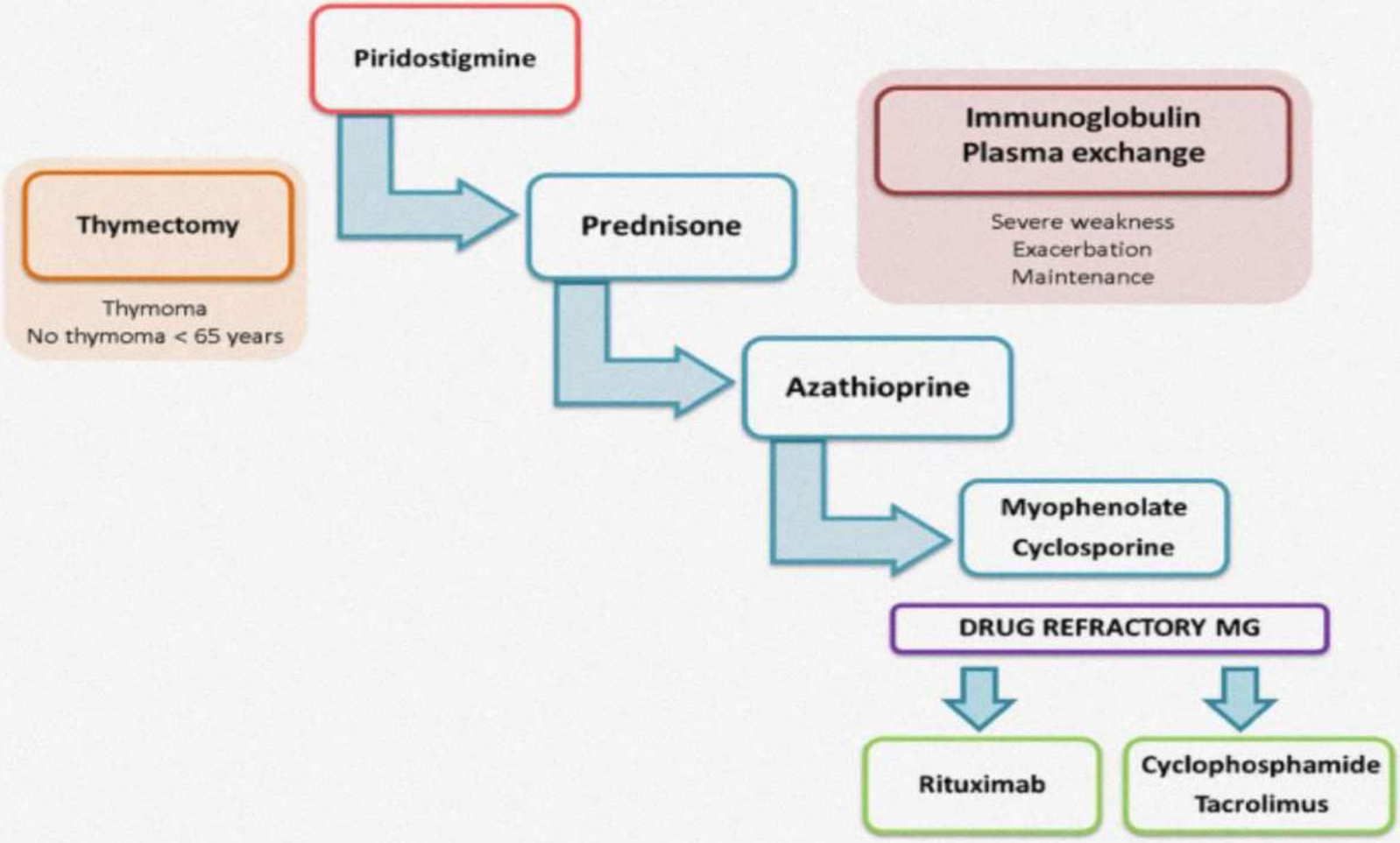
- Rare autoimmune disorder
- The immune system attacks channels that regulate calcium levels in the blood
- This causes insufficient acetylcholine to be released, leading to muscle weakness, fatigue, and other symptoms

Myasthenia gravis	Lambert Eaton syndrome
Antibody against AchR antibody	Antibody against voltage gated calcium channel
Associated with Thymic tumor	Associated with Small cell lung cancer
Weakness worsen on prolonged exercise	Weakness improves on prolonged exercise
Normal Deep tendon reflex	Decreased or absent deep tendon reflex
Autonomic dysfunction is absent	Autonomic dysfunction is present
On repeated nerve stimulation, there is decremental response	On repeated nerve stimulation, there is incremental response

Management plan

- Drug therapy
- Immunomodulation
- Surgical therapy
- Supportive therapy
- Life style modification

MANAGEMENT



DRUGS USED IN MYASTHENIA GRAVIS

1) AChE inhibitors:

- Anticholinesterase inhibit Acetylcholinesterase (AchE), allowing the same Ach molecules to repeatedly interact with the available nicotinic receptors (NRs); frequency of Ach-NR interaction is increased.
- **Drugs:**
  1) Pyridostigmine bromide
  2) Prostigmine

2) Immunosuppressant medicines:

- They inhibit the immunity system, and limiting antibody production.
- **Drug:** Azothiaprine in addition to steroid medication (Prednisolone)

- **Pyridostigmine**
  Anticholinesterase with symptomatic relief

- **Rituximab (Rituxan) and eculizumab (Soliris)** are intravenous medications usually used for those who don't respond to other treatment

- **zilucoplan**, a peptide inhibitor of complement component 5 (C5 inhibitor), for the treatment of generalized myasthenia gravis in adult patients who are acetylcholine receptor antibody positive

Effects of cholinergic drugs

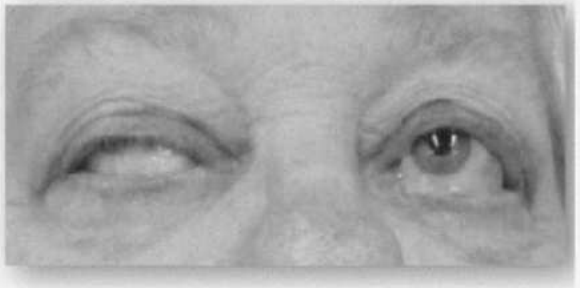
- CNS – enhance cognitive functions such as arousal, attention, & memory encoding – treatment for Alzheimer's disease & dementia
- Eye – pupil constriction – for surgery & treatment of glaucoma
- GI – smooth muscle stimulant – for post-op abdominal distention or paralytic ileus
- GU – urinary bladder stimulant – for post-op or postpartum urinary retention
- Musculoskeletal (indirect acting cholinergic drugs) – improve muscle tone & strength – for myasthenia gravis

Drugs that can Exacerbate Myasthenia Gravis

www.openmed.co.in

Mnemonic - *EXACERBATE*

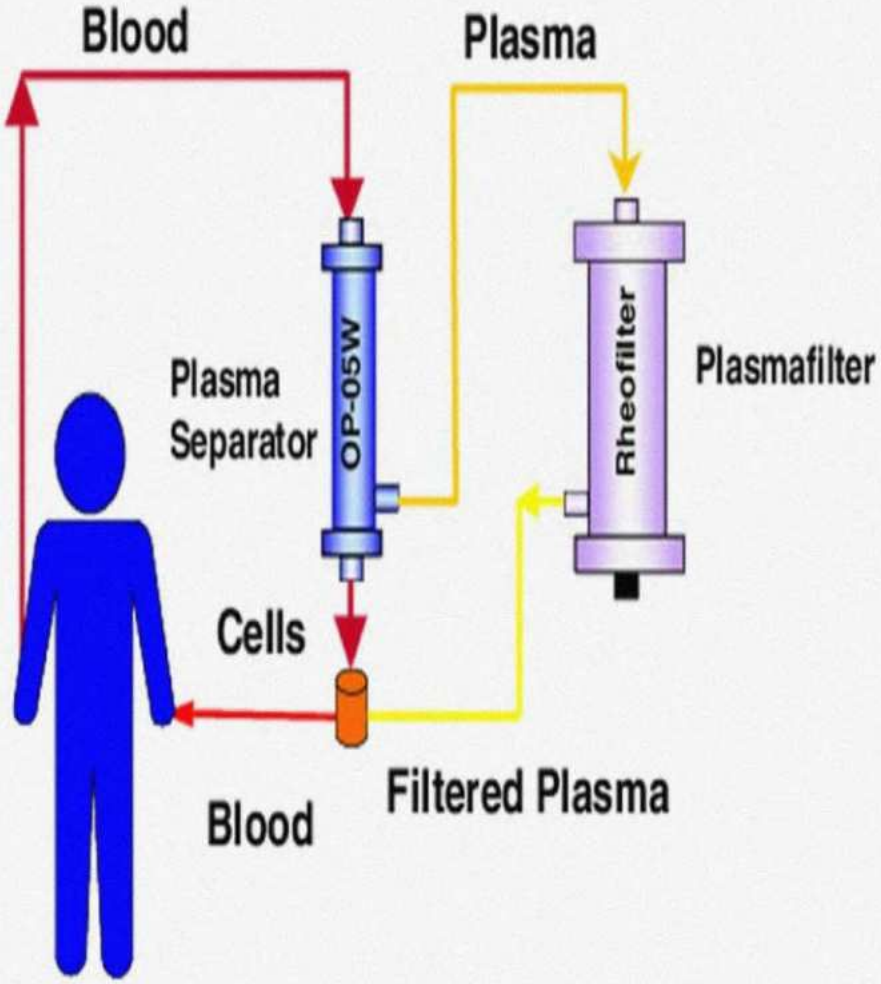
- **E**rythromycin (Macrolides)
- **X**ylocaine (Lignocaine)
- **A**minoglycosides
- **C**iproflox (Quinolones)
- **E**lectrolyte (Mg)
- **R**elaxant (Skeletal Muscle Relaxants)
- **B**otox & Beta Blocker
- **A**nti malarial (Quinine)
- **T**imolol (Eye Drops)
- **E**chothiophate (Eye Drops)

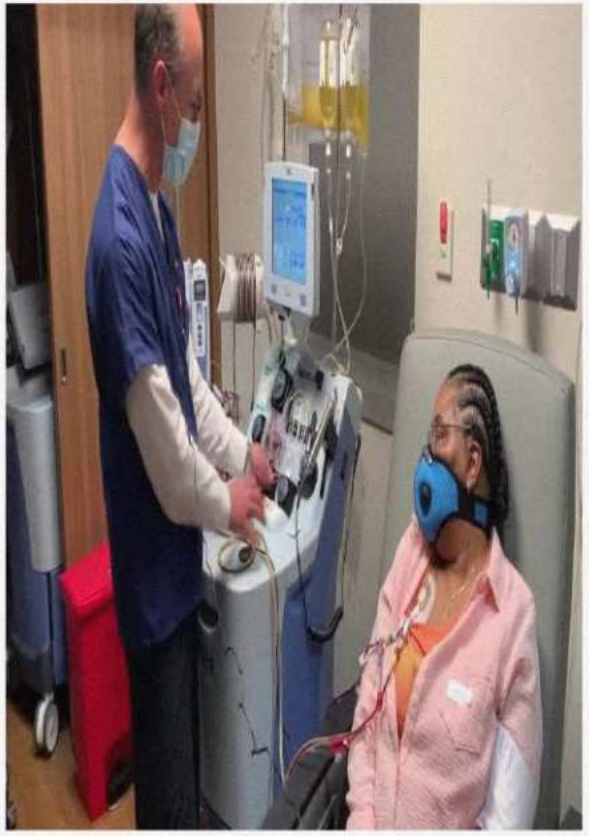


PLASMAPHERESIS

- *A method of removing blood plasma from the body by withdrawing blood, separating it into plasma and cells, and transfusing the cells back into the bloodstream*

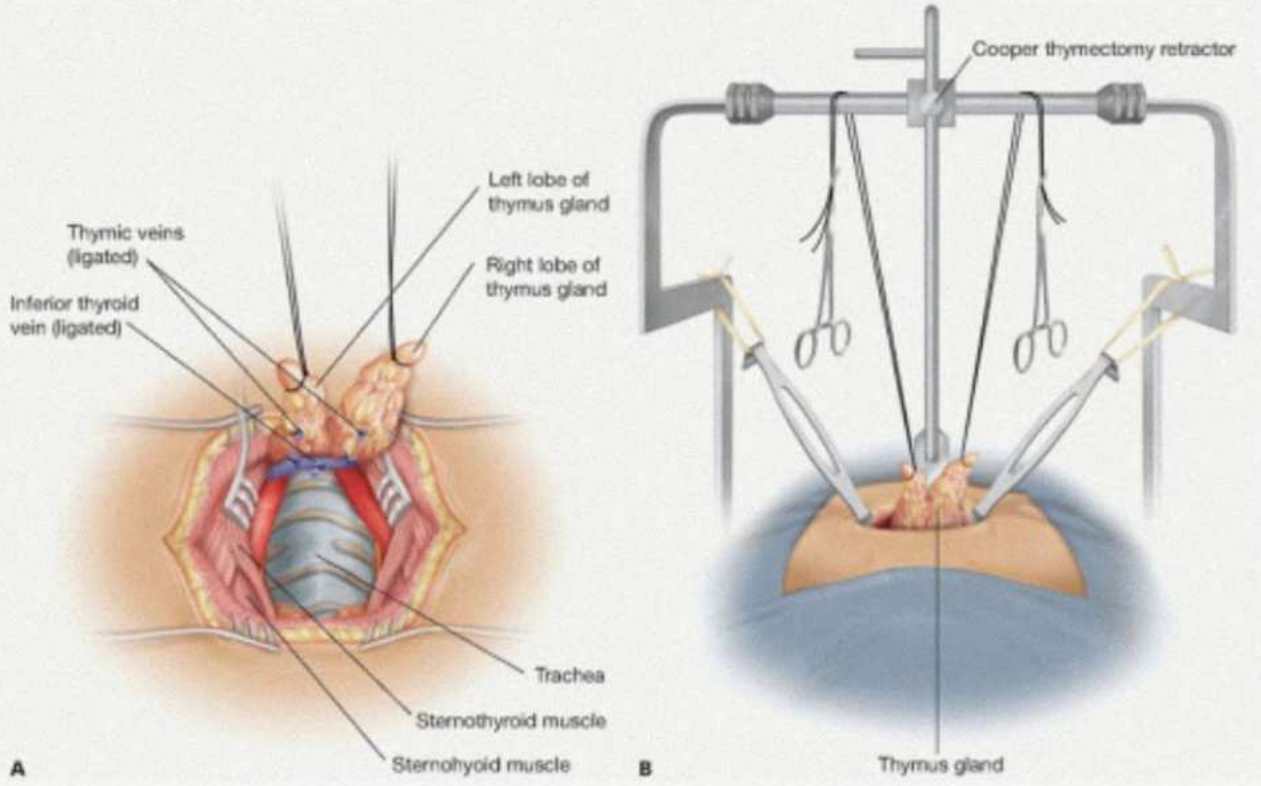
- *It is performed especially to remove antibodies in treating autoimmune condition*



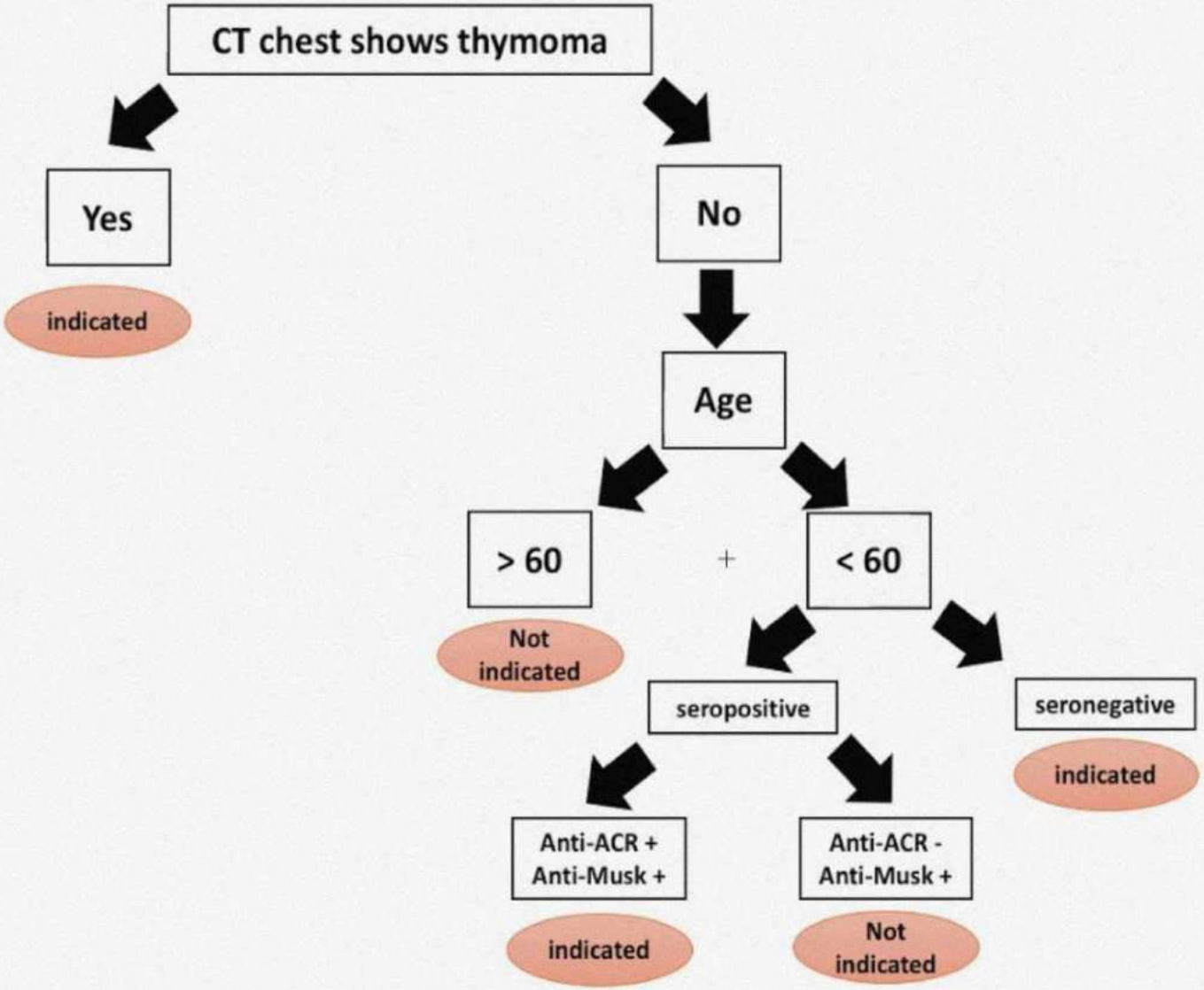


Thymectomy

- The goal is to cause remission of the disease
- To allow dose reduction of harmful immunosuppressive medications



Indications of thymectomy in Myasthenia Gravis



Life style modification

- Avoid physical exertion
- Take Plenty of Rest
- Avoid emotional stress
- Avoid exposure to extreme temperatures
- Continuous positive airway pressure therapy
- If diplopia bothers then occlusion
- Avoid medications such as muscle relaxants
- Avoid pneumonia/respiratory illness
- Avoid Low levels of potassium (diuretics and vomiting)


Myasthenia Gravis Diet Considerations
Aim to eat more small meals frequently rather than large meals
Make foods that are soft and easy to swallow or puree them
Tweak spices and temperature to boost appetite
Try out a liquid diet that includes shakes and smoothies
Incorporate thickening liquids to prevent them from being breathed into the lungs
verywell

Do you know?

AMITABH BACHCHAN

is suffering from

MYASTHENIA GRAVIS

for the past 30 years!

June is....

Myasthenia Gravis Awareness Month

Many MG patients experience drastic changes in their physical appearance. For some, they may only experience changes in relation to symptoms of the MG (ex. dropping eyelids); however, others may experience changes as a side effect of medications and treatments for controlling the MG (ex. Prednisone leading to weight gain or 'moon face')