TOPICS TO BE DONE FROM JOGI (IF YOU HAVE STUDIED JATOI)

BY MARYAM MALIK

RAWALPINDI MEDICAL COLLEGE

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TOPICS

1) SQUINT
2) EYELIDS
3) LACRIMAL
4) CONJUNCTIVA
5) CORNEA
LATENT SQUINT (HETEROPHORIA)

Types

*Esophoria*—There is a tendency for deviation of the eyeball inwards.

*Exophoria*—There is a tendency for deviation of the eyeball outwards.

*Hyperphoria*—There is a tendency for deviation of the eyeball upwards.

*Cyclophoria*—There is a torsional deviation of the eye.

*Anisophoria*—The deviation of the eyeball varies with the direction of gaze.

*Orthophoria*—There is no deviation of the eyes even when the fusion is broken.

Etiology

1. Increased requirement for accommodation and convergence as in hypermetropia results in esophoria.
2. Decreased requirement for accommodation and convergence as in myopia results in exophoria.
3. Occupations requiring too much close work such as goldsmith, watchmakers.
4. General poor health, fatigue and advancing age.

1. *Cover Test*
**Principle**
Fusion of the two eyes is abolished by covering one eye.

**Method**
The patient looks at a distant object.
• While observing one eye, cover and uncover the other eye. The movements of the observed eye and the eye under cover are noted.
• Repeat this process with the other eye and then alternately.

**Interpretation**
1. If there is no movement, patient has orthophoria.
2. If there is inwards movement on removing the cover the patient has exophoria.
3. If there is outwards movement on removing the cover the patient has esophoria.

**2. Maddox Rod Test**

**Principle**
This test is done to find out heterophoria for distance. It alters the appearance of the retinal image in one eye. There is no stimulation given to fusion.

**Method**
• The patient is seated 6 m from a spot of bright light in a dark room.
• A Maddox rod consisting of 4-5 cylinders of red glass fused side by side in a supporting disc is
placed in front of one eye. The same effect is given by a disc of deeply grooved red glass (Maddox groove).

• The spot of light appears as a red line. If the cylinders are placed with their axis horizontal, the red line will appear vertical and *vice versa*.

**Interpretation**

If there is orthophoria, the bright spot will appear in the centre of the vertical red line.

1. *Type of heterophoria*: By the position of the vertical or horizontal line in relation to the spot of light, exact type of heterophoria is detected.
2. *Angle of deviation*: The strength of prism which is necessary to be placed in front of the Maddox rod or the other eye so that the red line and spot appear together; indicates the angle of deviation.
3. *Nature of deviation*: It is indicated by the position of the prism whether base in or base out.
4. *Amount of deviation* can be measured on a graduated tangent scale set on a wall.

**3. Maddox Wing Test**

**Principle**

The Maddox wing is an instrument that dissociates the two eyes for near fixation (one-third of a meter) and measures the amount of heterophoria.

**Method**
The patient is asked to hold the Maddox wing and look through the two observation slits with both eyes open.
• The right eye sees a white arrow pointing vertically and a red arrow pointing horizontally to the left.
• The left eye sees the white figures in the horizontal lines and red figures in the vertical line. The figures are calibrated in degrees to read deviation.
• Ask the patient to read the figures corresponding to red and white arrows.

Interpretation
Any deviation indicates an esophoria, exophoria or hyperphoria which can be read on the scale.

4. Prism Vergence Test

Principle
The actual measurement of the deviation and strength of the muscles involved are tested. The muscles are forced to act with maximum effort against prisms.

Method
The patient is seated 6 m from a light source and looks at the Maddox tangent scale.
• The highest prism which can permit single vision gives the verging power.
• It is tested in different directions.
TREATMENT

Exercises to increase the fusional reserve and convergence are advised.

i. **Pencil exercise**: A pencil is held in the hand and brought slowly towards the nose until the tip appears double. The two images are then fused into a single image by an effort 8-10 times. This is repeated 3-4 times a day for several weeks.

ii. **Exercise the weak muscles against prisms**.

iii. **Exercise the weak muscle by the use of the synoptophore**

HIRSCHBERG TEST:-

Hirschberg test—It is a quick and useful method
to find out the angle of squint by the position of the corneal reflex when the light is thrown into the eye from a distance of about 50 cm.

Worth’s four dot test:
- The patient wears a red lens in front of his right eye which filters all colours except red. A green lens is placed in front of his left eye which filters all colours except green. Thus, he sees red and green colours with right and left eyes respectively.
- He views a box with four lights—one red, two green and one white.

If the patient sees all four lights, he has normal fusion.
- If he sees two red lights, he has left suppression.
- If he sees three green lights, he has right suppression.
- If he sees that the green and red lights alternate, he has alternating suppression.
• If he sees two red and three green lights, he has diplopia.

7. **Hess screen:**

*Principle*: Dissociation of retinal images of two eyes is carried out by red-green goggles. The Hess screen test provides the following information:

- A record of primary and secondary deviation.
• In paralytic squint, it provides information about the progress of the case if taken at suitable intervals.

Method: The patient wears red-green filter goggles and holds a green light projection pointer.
• The surgeon holds a red light projection pointer which is used as a point of fixation.
• The surgeon projects the red light onto the Hess screen.
• The patient is asked to superimpose his green light onto the red light.
• In normal condition, the two pointers should be nearly superimposed in all nine positions of gaze.

Interpretation:
• The two charts are compared.
• The smaller chart indicates the eye with the paralysed right lateral rectus muscle. It shows greatest restriction in the main direction of action of the muscle.
• The larger chart indicates the eye with the overacting muscle, i.e. medial rectus muscle.
8. **Synoptophore (Amblyoscope):** It tests the sensory status of the eyes which includes grade of binocular vision, presence of suppression, amblyopia and retinal correspondence. The instrument consists of two cylindrical tubes with a mirrored right-angled bend. A +6.5 D lens is fixed in each eyepiece. The adjustments of the tubes are indicated on a scale.

**NYSTAGMUS**
It is the involuntary, symmetrical, synchronous, rapid oscillatory movements of the eyes. It is independent of the normal movements of the eyes which are not affected.
Etiology
It is a disturbance of ocular posture. The factors responsible for maintenance of ocular posture are the visual sensory pathway, the vestibular apparatus and the motor mechanisms which coordinate the sensory and motor functions.

1. Ocular Nystagmus
It is due to defect in maintaining fixation.
   a. Physiological:
      i. Optokinetic nystagmus: It is seen when a person travels in a train and keeps on looking outside.
      ii. Latent nystagmus: Nystagmus is not present when both the eyes are open. It becomes manifest on closing either eye.
   b. Spontaneous:
      i. Amaurotic nystagmus: It is jerky or pendular type occurring in infants who are born blind and in whom macular fixation has not developed.
      ii. Amblyopic nystagmus: It is due to interference with the development of macular fixation within the first 4 to 6 months of life, e.g. as in albinism, congenital total colour blindness or any opacity in the media.
      iii. Spasmus nutans: Head nodding movement may occurs in children brought up in a very dim...
iv. **Miner’s nystagmus**: It is an occupational disease occurring in coal mine workers due to dim illumination. It is of rapid rotatory type.

### 2. Vestibular Nystagmus

i. It occurs in diseases of the internal ear, i.e. semicircular canals are involved.

ii. It can be produced in normal persons by rotatory movement, syringing the ear with cold water, etc.

iii. The nystagmus is jerky, fine, rapid and horizontal-rotatory.

### 3. Central Nystagmus

It is caused by lesions of the:

i. **Midbrain**: Disseminated sclerosis, encephalitis, vascular lesions.

ii. **Cerebellum**: Tumour, abscess. The nystagmus is jerky and is most commonly elicited on the lateral deviation of the eye.

### 4. Congenital Hereditary

It is hereditary and the cause is unknown.

**Treatment**

It is palliative like correction of refraction, use of smoked or tinted glass or contact lens in albinism and the treatment of any underlying disease.

**Prognosis**
Nystagmus tends to diminish with advancing age.

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<td>efferent pathway breaks down</td>
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<td>• Traumatic or inflammatory lesion at the level of</td>
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<td>muscle of the same eye</td>
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<td>4. Operative measures—It consists of recession or</td>
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<td>resection of appropriate muscles</td>
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Parasites such as *Demodex folliculorum*, *Phthiriasis palpebrarum*, crab louse, head louse also cause blepharitis
Squamous blephritis is due to abnormal metabolism and seborrhoea. It is usually associated with the dandruff of the scalp.

Tylosis—There is thickening or hypertrophy of the lid margin

**DIFFERENCES BETWEEN SQUAMOUS AND ULCERATIVE BLEPHARITIS**

<table>
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<th>1. Clinical features</th>
<th>SQUAMOUS BLEPHARITIS</th>
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<tr>
<td>i. Scales</td>
<td>White, fine and dry</td>
<td>Yellowing, coarse, and sticky</td>
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<td>ii. Ulceration</td>
<td>Absent</td>
<td>Present</td>
</tr>
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<td>iii. Bleeding</td>
<td>Absent</td>
<td>Present</td>
</tr>
<tr>
<td>iv. Loss of eyelashes</td>
<td>Few and temporary</td>
<td>Permanent and almost all lashes are involved</td>
</tr>
<tr>
<td>2. Course</td>
<td>Mild</td>
<td>Progressive</td>
</tr>
<tr>
<td>3. Complications</td>
<td>Occasional</td>
<td>Usual and serious</td>
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**DIFFERENCES BETWEEN STYE (HORDEOLUM), CHALAZION AND INTERNAL HORDEOLUM**

<table>
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<tr>
<th>1. Onset</th>
<th>STYE (HORDEOLUM)</th>
<th>CHALAZION</th>
<th>INTERNAL HORDEOLUM</th>
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<td>2. Gland</td>
<td>Acute Zeis’s gland Suppurative</td>
<td>Chronic Meibomian gland Granulomatous</td>
<td>Acute meibomian gland Suppurative Severe pain</td>
</tr>
<tr>
<td>3. Type of inflammation</td>
<td>Acute pain and swelling Localised, hard (pus) and tender swelling near the lid margin</td>
<td>Painless Disfigurement Hard swelling away from lid margin</td>
<td>Yellow point seen on evertting the lid Vertical incision and drainage</td>
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<tr>
<td>4. Symptoms</td>
<td>Hot fomentation Antibiotic and Removal of eyelash</td>
<td>Vertical incision and drainage</td>
<td>Antibiotic and analgesic</td>
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<td>5. Signs</td>
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<td>6. Treatment</td>
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Section of the upper eyelid showing normal and abnormal position of tarsus and eyelashes
SYMBLEPHARON
It is a condition of adhesion of the lids to the globe.

Etiology
It is due to the formation of raw surfaces upon two opposite spots of the palpebral and bulbar conjunctiva, causing adhesion during the healing process. It is often due to:
i. Burns due to heat or caustics
ii. Ulcers
iii. Diphtheria
iv. Operations.

Types
1. Anterior symblepharon—The lid margin is usually implicated.
2. Posterior symblepharon—The fornix is implicated so that the conjunctival surfaces are adhered to each other.
3. Total symblepharon—The fornix and lid margins are involved together. The lids are completely adherent to the eyeball. It is a rare condition.

Symptoms
1. Lagophthalmos, i.e. inability to close the lids properly is often present.
2. Diplopia—There is restricted mobility of the eye due to marked
conjunctival adhesions.
3. Cosmetic disfigurement may be the presenting complaint.

**Signs**

Broad or narrow bands of fibrous tissue are seen stretching between lid and globe.

**Treatment**

1. *Prophylaxis*—Prevention is most important. It is achieved by applying eye ointment and moving a glass rod in the fornices several times a day. A therapeutic contact lens may be helpful.
2. *Mucous membrane graft*—The raw surfaces are covered by buccal mucous membrane graft or conjunctiva from the upper temporal quadrant of the same or opposite eye. It is difficult in cases of posterior symblepharon and broad bands. Therefore, great care is taken to prevent perforation of the globe.
3. *Z-plasty* operation can also be done.
ANKYLOBLEPHARON
It is a condition of the adhesion of the margins of the two eyelids. The adhesion may be partial or complete. It is usually associated with symblepharon.

Etiology
It may be congenital or acquired due to chemical burn, e.g. acid, alkali.
**Treatment**
Separation of the lid margins along with mucous membrane or conjunctival grafting is recommended.

**BLEPHAROPHIMOSIS**
It is a condition where the palpebral fissure appears to be contracted at the outer canthus.

**Etiology**
It may be congenital or acquired due to prolonged blepharospasm or epiphora.

**Treatment**
Canthoplasty, i.e. incision of the outer canthus is the treatment of choice. Apply a small artery forceps to the outer canthus. Wait for 2-3 minutes in order to achieve haemostasis. Then cut the outer canthus with a fine scissors or blade.

**LAGOPHTHALMOS**
It is a condition of incomplete closure of the palpebral aperture when eyes are shut

**Etiology**
1. Congenital deformity of the lids.
2. Ectropion.
3. Proptosis.
4. Paralysis of orbicularis oculi muscle.
5. Absence of reflex blinking in extremely ill patients.

**Complication**
Exposure keratitis develops usually in the lower part of the cornea due to incomplete closure of lids.

**Treatment**

1. Application of antibiotic eye ointment and bandage during sleep is recommended.
2. Lateral tarsorrhaphy or paramedian tarsorrhaphy is done in neuroparalytic cases.

**BELL’S PHENOMENA**

*Bell’s phenomenon*—The upwards and outwards rolling up of the eye during sleep or on
forcibly closing the lids is known as the Bell’s phenomenon.

**Principle of correction of ptosis**

There are three main techniques available for the correction of ptosis:

i. If the levator muscle action is good, it may be shortened.

ii. If the levator muscle is paralysed, the superior rectus muscle is used to lift the lid.

iii. If both levator and superior rectus muscles are paralysed, the action of frontalis muscle is utilized.

**Technique**

1. *Resection of levator muscle*—If the levator muscle is not completely paralysed, the levator muscle may be shortened by the resection of the muscle.

   i. Blaskovics operation is done from the conjunctival side.

   ii. Everbusch operation is done from the skin surface.

   iii. Fasanella-Servat operation—The levator muscle is shortened along with excision of 4-5 mm
of the tarsal plate. Muller’s muscle and palpebral conjunctiva.

2. Motais operation—If the levator muscle is paralysed, the superior rectus is pressed into service to elevate the lid.

3. Hess’s operation—If both levator palpebrae superioris and superior rectus muscles are paralysed, action of frontalis muscle is used in raising the lid by passing silk mattress sutures in tarsal plate.

4. Fascia lata sling operation—Three incisions are made in the upper lid about 4 mm from the lid margin. Three more deep incisions are made above the eyebrow as shown in the diagram. The fascial strips are drawn through the lid openings and secured tightly by 5.0 chromic catgut at each eyebrow incision.
Coloboma
There is a triangular notch in the upper lid margin near the nasal side usually. Coloboma of the iris or accessory auricle may be associated.

Epicanthus
It is a bilateral condition which may be associated with ptosis. A triangular fold of skin covers the medial canthus. The eyes appear to be far apart. It can be corrected by plastic surgery.
LACRIMAL SYSTEM

TEARS
Tear is a secretion from the lacrimal gland. It is slightly alkaline and consists mainly of water, small quantities of salts, such as sodium chloride, sugar, urea, protein and lysozyme, a bactericidal enzyme. The secretion of tear does not begin before 3-4 weeks after birth. The average normal secretion of tears is 0.5-2.2 ml. The normal pH of tear is 7.5

DACRYOPS
It is a cystic swelling of the lacrimal gland due to retention of lacrimal secretion as a result of blockage of one of the lacrimal ducts.

MIKULICZ’S SYNDROME
There is symmetrical enlargement of the lacrimal and salivary glands (parotid glands) usually with lymphoid tissue hyperplasia. The etiology is unknown but it is seen in uveoparotid inflammations

TUMOURS
Benign Tumour
The most common tumour is pleomorphic adenoma (mixed tumour). The benign mixed tumour
usually occurs in middle life. It presents as a slowly progressive painless swelling in the upper lid. It may result in mechanical ptosis. It should be excised.

**Malignant Tumour**
The malignant tumour presents with a short history and pain. If malignant, radical surgical removal is necessary.

**TREATMENT OF CONGENITAL NASOLACRIMAL DUCT OBSTRUCTION**

*Massage over the lacrimal sac area* and clean the discharge several times a day. This constitutes the treatment of congenital nasolacrimal duct block up to 6-8 weeks of age.

**COMMON CAUSES OF EPIPHORA**

2. Congenital non-communication or delayed communication of nasolacrimal duct.
3. Occlusion of puncta—foreign body, hair, etc.
4. Chronic dacryocystitis with blockage of nasolacrimal duct.
5. Growth or inflammation of inferior meatus.
6. Functional insufficiency for draining the tear into the nose, e.g. Bell’s palsy, ectropion.

**METHODS OF DCR**

*Method*

- The nasal fossa of the same side is packed with cocaine or xylocaine and adrenaline.
- The canaliculi are dilated and lacrimal sac is irrigated with warm saline.
• The early steps are same as for excision of the sac.
The periosteum over the lacrimal crest is incised and lacrimal bone is exposed.
• The bony crest is removed with a gouge and hammer and nasal mucosa is exposed.
• The nasal mucosa of the middle meatus is anastomosed with the medial wall of the sac by making vertical incisions in them.
• Syringing is done to test the patency of the passage after 1-2 days postoperatively.

Complications
i. Haemorrhage—Intranasal bleeding may occur from the nasal mucosa which requires nasal packing for 24 hours.
ii. Failed DCR—Small bony opening is the most important cause. Other causes include, improper suturing, postoperative infection, nasal pathology such as polyp, etc.
Angular Conjunctivitis (Diplobacillary Conjunctivitis)
The reddening of the conjunctiva is confined exclusively to the intermarginal strip of the bulbar conjunctiva.

**Etiology**
It is caused by Morax-Axenfield diplobacillus. It produces proteolytic ferment which macerates the conjunctival epithelium. It is often found in the nasal cavity and nasal discharge in case of angular conjunctivitis.

**Symptoms**
1. Red eye is the most common feature.
2. There is discomfort and frequent blinking.
3. Mild mucopurulent discharge may be present.

**Signs**
1. Reddening of the bulbar conjunctiva is seen limited to the intermarginal strip specially at the inner and outer canthi.
2. There is excoriation of skin at the outer and inner canthi.

**Complications**
1. Blepharitis occurs in chronic untreated cases.
2. Marginal, central or hypopyon corneal ulcer may occur.
3. Recurrences are common.

**Treatment**
1. Oxytetracycline ointment is the drug of choice (bacteriostatic action).
2. Zinc sulphate lotion though less effective acts by inhibiting the proteolytic enzymes produced by Morax-Axenfeld bacillus. It forms a coagulum in which the bacilli get enmeshed.
3. Zinc oxide ointment may be applied to the lids at night.
Keratopathy in VKC
Buckley has classified the corneal involvement into 5 clinical stages:
i. *Superficial punctate keratitis*—These are tiny microerosions in upper cornea.
ii. *Epithelial macroerosion and ulceration* occurs due to epithelial loss.
iii. *Plaque*—There is bare area caused by macroerosion of epithelium which becomes coated with mucus.
iv. *Ring scar* is formed as a result of subepithelial corneal scarring.
v. *Pseudogeron toxon*—It resembles arcus senilis with appearance of ‘cupid’s bow’.

### Differential Diagnosis of Nodule at the Limbus

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<td>Cystoid cicatrix following glaucoma surgery</td>
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Concretions [Lithiasis]

**Incidence**
It is common in the elderly persons. There is accumulation of epithelial cells and inspissated mucus in Henle’s glands. They never become calcified so the term ‘lithiasis’ or ‘stone’ is a misnomer.

**Symptoms**
Foreign body sensation and irritation are common complaints.

**Signs**
1. There are minute hard yellow spots seen in the palpebral conjunctiva.
2. They project from the surface rubbing against the lid or the cornea.

**Treatment**
Concretions are removed with a sharp needle.
VISUAL DISPLAY TERMINAL SYNDROME (VDTS)

Nowadays an important cause of dry eyes is use of contact lenses and computers.

*Computers*—Many studies have shown that computer screens kept at or above the level of the eyes enhance the evaporation of the tears. This is because the palpebral fissure is widened and blink rate is decreased while using computer.

*Contact lens*—Use of contact lenses also contribute to the development of dry eyes due to following reasons,

i. Rigid lenses disrupt the lipid layer enhancing evaporation of the tear film.
ii. Soft contact lenses actively deplete the mucus layer to maintain their hydration level.
iii. Contact lenses also decrease the corneal sensation, a factor which may be necessary for the tear secretion.

**Symptoms**

1. Burning, discomfort and irritation are common complaints.
2. Photophobia and lacrimation are present in corneal involvement.
3. Impaired vision is present in cases of corneal opacity formation.
4. Night blindness is present in cases of vitamin A deficiency.

**Signs**
1. Bitot’s spot—These are small, triangular, shiny, silver white patches seen on the bulbar conjunctiva near the outer canthus usually.
2. The conjunctival epithelium becomes epidermoid like that of skin.
3. There may be excessive mucus secretion (white coloured) due to deficiency of aqueous layer.

**Complications**
- Corneal stromal ulcers are common.
- Conjunctivitis and blepharitis occur due to loss of defence mechanism

**Argyrosis**
There is staining of the conjunctiva a deep brown colour due to prolonged application of silver salt (nitrate, proteinate, etc.) for the treatment of chronic conjunctivitis

**Tumours**
1. Congenital
   i. *Dermoid*—Dermoids are choristomas. It is yellow-grey in colour.
   - They are smooth, solid round lesions
• It is situated astride the corneal margin on the outer side of limbus.
• Epibulbar dermoid may be associated with other congenital anomalies of the body.
• It consists of epidermoid, epithelium, sebaceous glands and hair.
• It is usually stationary in growth.
• Dermoids when large may cause corneal astigmatism.
• It is dissected off and replaced by lamellar corneal graft for cosmetic region
ii. Dermolipoma is situated at the outer canthus usually.
• It consists of fibrous tissue and fat.
• It should be removed surgically.
2. Papilloma
• It occurs at the inner canthus, fornices and the limbus.
• It should be removed as it may turn malignant.
3. Simple Granuloma
• It consists of exuberant granulation tissue.
• It is polypoid and is usually seen at the chalazion site when chalazion is insufficiently scraped.
• It should be completely removed by scissors.
4. Squamous Cell Carcinoma
• It occurs at the limbus or lid margin (transitional zone).
• It spreads over the surface and into the fornices.
• It may penetrate the eyeball.
• It is removed and the base is cauterized by diathermy.
• If it recurs, or in extensive lesions the eye is enucleated.

5. Pigmented Tumours
i. Naevi or congenital mole is rarely malignant.
ii. Precancerous melanosis is a diffusely spreading pigmentation of the conjunctiva seen in elderly persons.
iii. Malignant melanoma occurs typically at the limbus in old people. It spreads over the surface of the eyeball. Recurrences and metastases occur elsewhere in the body commonly. It is treated by enucleation of the globe or exenteration of the orbit in cases of extraocular extension.

CORNEA
### DIFFERENCES BETWEEN ANTERIOR SYNECHIA, LEUCOMA, ADHERENT LEUCOMA AND ANTERIOR STAPHYLOMA

<table>
<thead>
<tr>
<th></th>
<th>ANTERIOR SYNECHIA</th>
<th>LEUCOMA</th>
<th>ADHERENT LEUCOMA</th>
<th>ANTERIOR STAPHYLOMA</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Definition</strong></td>
<td>Adhesion between iris and cornea</td>
<td>Dense, white opacity of cornea</td>
<td>Adhesion between iris and leucoma</td>
<td>Adhesion between iris and leucoma.</td>
</tr>
<tr>
<td></td>
<td>• Perforated corneal ulcer</td>
<td>• Healed corneal ulcer</td>
<td>• Perforated corneal ulcer</td>
<td>• Perforated corneal ulcer due to secondary</td>
</tr>
<tr>
<td></td>
<td>• Iridocyclitis</td>
<td>• Healed keratitis</td>
<td>• Penetrating injury</td>
<td>glaucoma usually. Same as adherent leucoma</td>
</tr>
<tr>
<td></td>
<td>• Closed angle glaucoma</td>
<td>• Penetrating injury</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Foreign body</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Corneal dystrophy</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Symptoms</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. <strong>Visual acuity</strong></td>
<td>Normal</td>
<td>Impaired if situated over pupillary area</td>
<td>Impaired if situated over pupillary area</td>
<td>Impaired</td>
</tr>
<tr>
<td><strong>Signs</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. <strong>Corneal surface</strong></td>
<td>Flat</td>
<td>Flat</td>
<td>Flat</td>
<td>Ectatic</td>
</tr>
<tr>
<td></td>
<td>Nil</td>
<td>Fine yellowish brown lines in the epithelium</td>
<td>Brown pigment from iris are seen</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>(Hudson-Stahl) haemostiderin, melanin</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. <strong>Anterior chamber</strong></td>
<td>Normal or shallow</td>
<td>Normal</td>
<td>Irregular or shallow where iris comes forwards</td>
<td>Usually absent or very shallow</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Drawn towards adhesion</td>
<td></td>
</tr>
<tr>
<td>4. <strong>Pupil</strong></td>
<td>Normal</td>
<td>Normal</td>
<td>Impaired over opacity</td>
<td>Not seen</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Normal over opacity</td>
<td></td>
</tr>
<tr>
<td>5. <strong>Corneal sensation</strong></td>
<td>Normal</td>
<td>Impaired over opacity</td>
<td>Impaired</td>
<td></td>
</tr>
<tr>
<td>6. <strong>Intraocular tension</strong></td>
<td>Normal or raised when more than 3/4 circumference is involved (closed angle glaucoma)</td>
<td>Normal</td>
<td>Normal or raised (secondary glaucoma)</td>
<td>Raised usually</td>
</tr>
</tbody>
</table>

**ULCUS SERPENS**

It is the most common type of hypopyon ulcer.
It occurs in adults due to pneumococcus bacteria usually.
It has a tendency to creep over the cornea in a serpiginous fashion.

**Symptoms**
1. There is marked pain in the eye and lacrimation.
2. A variable amount of photophobia is present.

**Signs**
1. Cornea is lustreless and hazy. A greyish white or yellow disc is seen in the centre.
2. The opacity is greater at the advancing edge in one particular direction than centre.
3. The tissues breakdown on the side of the densest infiltration (yellow crescent) and ulcer spreads in size and depth.
4. Often there is infiltration anterior to Descemet’s membrane at the floor of the ulcer while the intervening stroma is normal.
5. Marked iritis with cloudy aqueous (hypopyon), conjunctival and ciliary congestion is usually present.
6. The lids are red and swollen.

**Complications**
10. Perforation with iris prolapse may occur due to thinning of cornea.
11. Panophthalmitis may occur due to rapid growth and spread of the virulent organisms.
12. Perforation may heal resulting in leucoma, adherent leucoma, anterior staphyloma or occlusiopupillae causing marked visual impairment.
13. Secondary glaucoma usually follows perforation due to synechia formation.

15. Treatment
16. It is a well-known surgical rule that pus anywhere in the body has to be removed. However, this is not true in case of hypopyon ulcer. The fact that the hypopyon is sterile has great practical importance.
17. When the ulcer is treated properly, the hypopyon gets absorbed automatically.
18. Early and intensive treatment of corneal ulcer as mentioned earlier is started at once after culture and sensitivity.
19. Broad-spectrum antibiotic drops are instilled every few minutes for the first hour. Later it is instilled hourly and then 2 hourly.
20. Topical atropine is applied even if the tension is raised.
24. • Antibiotic and atropine eye ointment are applied at bedtime.
25. • Subconjunctival injection of antibiotic and atropine may be given.
26. • Cauterization if done skillfully may be helpful.
27. 2. Secondary glaucoma is the most common cause of failure of treatment in elderly persons. It affects the nutrition and resistance of the cornea. It is treated by • Topical atropine 1%
• Oral acetazolamide (carbonic anhydrase inhibitor)
• Intravenous mannitol 20%, 200 ml (hyperosmotic agent)
• Paracentesis helps in lowering the tension and brings fresh aqueous and nutrient. It is done only in cases of markedly raised intraocular tension.
1.3. If there is associated chronic dacryocystitis, dacryocystorhinostomy (DCR) is performed
MARGINAL ULCER

Etiology

It is caused by Morax-Axenfeld bacillus, *Staphylococcus*, *H. aegyptius*, etc. It is often associated with chronic blepharoconjunctivitis.

Incidence
It is seen in old debilitated people usually. Deep marginal ulcer may occur rarely in cases of polyarteritis nodosa, systemic lupus erythematosus due to antigen-antibody complexes.

**Symptoms**
There is neuralgic pain in the face and head. Recurrence is common.

**Signs**
1. Shallow, slightly infiltrated, multiple ulcers are seen near the limbus.
2. The ulcers are often vascularised.

**Complications**
1. Deep marginal ulcers—These are seen in autoimmune diseases.
2. There may be formation of ring ulcer.
3. This may be followed by necrosis of the whole cornea.

**Treatment**
- Suitable antibiotic eyedrops and ointment are applied.
- Chemical cautery may be done with 1% silver nitrate in mild recurrent ulcers.
- Steroid drops and ointment may give temporary benefit.
- In severe cases, systemic steroids and cytotoxic drugs may be useful.
## Clinical Features of Different Corneal Ulcers

<table>
<thead>
<tr>
<th>Clinical Features</th>
<th>Bacterial</th>
<th>Viral</th>
<th>Fungal</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Symptoms</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Discharge</td>
<td>Mucopurulent ++</td>
<td>Watery</td>
<td>May be present</td>
</tr>
<tr>
<td></td>
<td>Severe</td>
<td>Moderate ++</td>
<td>Mild</td>
</tr>
<tr>
<td></td>
<td>—</td>
<td>++</td>
<td>—</td>
</tr>
<tr>
<td>3. Systemic-Fever</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>headache, etc.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Recurrence</td>
<td>—</td>
<td>++</td>
<td>—</td>
</tr>
<tr>
<td>5. History of trauma</td>
<td>Common with</td>
<td>—</td>
<td>Vegetative matter</td>
</tr>
<tr>
<td></td>
<td>penetrating injury</td>
<td></td>
<td>injury</td>
</tr>
<tr>
<td><strong>Signs</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Injection</td>
<td>Marked</td>
<td>Moderate +</td>
<td>Marked</td>
</tr>
<tr>
<td>2. Follicles</td>
<td>—</td>
<td>+</td>
<td>—</td>
</tr>
<tr>
<td>3. Ulcer</td>
<td>Central disc with</td>
<td>Typical dendritic</td>
<td>Dry yellow-grey</td>
</tr>
<tr>
<td></td>
<td>necrotic material</td>
<td>and geographical</td>
<td>with satellite</td>
</tr>
<tr>
<td></td>
<td>May be deep</td>
<td>pattern</td>
<td>lesions</td>
</tr>
<tr>
<td>5. Corneal sensations</td>
<td>Present</td>
<td>Usually superficial</td>
<td>Deep</td>
</tr>
<tr>
<td>6. Preauricular</td>
<td>+</td>
<td>Absent</td>
<td>Present</td>
</tr>
</tbody>
</table>

## Differences Between Herpes Simplex and Herpes Zoster

<table>
<thead>
<tr>
<th></th>
<th>Herpes Simplex</th>
<th>Herpes Zoster</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>1. Incidence</strong></td>
<td>Children and young adult</td>
<td>Elderly with depressed cellular</td>
</tr>
<tr>
<td></td>
<td>usually.</td>
<td>immunity.</td>
</tr>
<tr>
<td></td>
<td>Herpes simplex virus.</td>
<td>Varicella zoster virus.</td>
</tr>
<tr>
<td><strong>2. Etiology</strong></td>
<td>There is recurrence in,</td>
<td>It is identical with that</td>
</tr>
<tr>
<td></td>
<td>• Febrile cold</td>
<td>causing chickenpox.</td>
</tr>
<tr>
<td></td>
<td>• Pneumonia, malaria</td>
<td>Associated with chickenpox</td>
</tr>
<tr>
<td></td>
<td>• Exposure to sunlight</td>
<td>infection in youth.</td>
</tr>
<tr>
<td></td>
<td>• Physical exhaustion</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>3. Clinical features</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>a. Systemic</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• There are vesicles on</td>
<td>Fever, malaise, severe pain</td>
</tr>
<tr>
<td></td>
<td>lips, nose and</td>
<td>along 5th nerve fibres.</td>
</tr>
<tr>
<td></td>
<td>genitals.</td>
<td>• Unilateral always.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>b. Ocular</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Follicular keratoconjunctivitis.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Superficial punctate keratitis.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Dendritic ulcer.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Confluent ulcer.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Disciform keratitis.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Eruption of rows of vesicles</td>
<td></td>
<td></td>
</tr>
<tr>
<td>along ophthalmic division of 5th nerve.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Vesicles suppurate and bleed</td>
<td></td>
<td></td>
</tr>
<tr>
<td>forming small scars.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Superficial punctate keratitis.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. Subepithelial coarse punctate</td>
<td></td>
<td></td>
</tr>
<tr>
<td>keratitis.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. Stroma may be involved in late stages.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
PHOTOPHTHALMIA

Etiology
It commonly occurs due to:
1. Exposure to ultraviolet rays by the bright flash of a short circuit or exposure to naked arc light in welding and cinema studio results in photophthalmia.
2. Snow blindness—The ultraviolet rays are reflected from snow surface.

Symptoms
There is extreme burning pain, photophobia, lacrimation and blepharospasm due to desquamation of corneal epithelium.

Signs
There are multiple epithelial erosions associated with blepharospasm and swelling of the palpebral conjunctiva and retrotarsal folds.

**Treatment**
- Cold compresses, astringent lotions and atropine ointment are effective.
- Bandage both eyes for 24 hours. This helps in regeneration of the epithelium.

**Prophylaxis**
Wearing of dark glasses (Crooke’s glasses) made of such materials which cut off practically all the infrared and ultraviolet rays when such exposure is anticipated.

**DEEP KERATITIS**
The deep forms of keratitis affect the stroma of the cornea.

**Etiology**
1. *Congenital syphilis*—It is characterised by bilateral interstitial keratitis, vascularization (Salmon patches) and uveitis. It affects children between the age of 5-15 years.
2. *Tuberculosis*—There is presence of interstitial keratitis in this condition.
3. *Viral infections (disciform keratitis)*—A central grey disc is seen in the stroma. It is unilateral
and seen in adults usually.

4. **Sclerosing keratitis**—It spreads from scleritis involving the corneal stroma.

**Treatment**
The basic cause of deep keratitis is treated along with routine treatment of corneal ulcer.

**Megalocornea**
It is a bilateral condition in which the corneal diameter is more than 14 mm. The cornea is usually clear with normal thickness and vision. It is often associated with Marfan’s syndrome.

**Differential diagnosis**
Megalocornea can be differentiated from buphthalmos and keratoglobus

i. **Buphthalmos**—In this condition IOP is raised and eyeball is enlarged as a whole. Enlarged cornea is associated with Descemet’s membrane tears.

ii. **Keratoglobus**—There is congenital bilateral hemispherical protrusion of the whole cornea.

**Keratoglobus**
In this condition there is thinning and excessive protrusion of cornea which seems enlarged but its diameter is usually normal.

**Microcornea**
The corneal diameter is less than 10 mm with decreased radius of curvature. Hypermetropia and
narrow angle glaucoma may be found in later years. The condition may occur as an isolated anomaly or in association with microphthalmos.

**Cornea Plana**

It is a rare anomaly in which cornea is comparatively flat since birth. It may be associated with microcornea.

**Posterior Embryotoxon**

There is an unusual prominence of Schwalbe’s line which is peripheral termination of Descemet’s membrane. It appears as a ring opacity in deeper layer of cornea.

---

### Classifications of Corneal Degenerations

**1. Central degenerations**
- Cornea farinata
- Salzman’s Nodular degeneration
- Hyaline degeneration
- Lipid degeneration
- Pigmentary degeneration

**2. Peripheral degenerations**
- Arcus senilis
- White limbal girdle of Vogt
- Terriens marginal degeneration
- Pellucid marginal degeneration
- Mooren’s ulcer
- Hepatolenticular degeneration

---

**Corneal Degenerations**

These are non hereditary and usually unilateral. They can be divided into three categories:

i. Primary degeneration
ii. Secondary degeneration
iii. Infiltration associated with metabolic disturbance, e.g. fatty degeneration, hyaline degeneration,
amyloid degeneration, calcific degeneration (Band shaped keratopathy) etc.
The basic difference between degenerations and dystrophies are as under:

**Arcus Senilis**
There is bilateral annular lipoid infiltration of cornea in old persons with no symptoms. It does not require any treatment as it does not affect the vision or vitality of the cornea.
There are concentric grey lines in the upper and lower part of the cornea.
• The lines join to form a ring 1 mm broad which is separated from the margin by a rim of clear cornea about 1.5 mm. It is also known as lucid interval of Vogt. The outer border of the arcus is sharp but the inner border appears faint. It is found in approximately 60% of population between the age of 40 to 60 years and almost in all persons above the age of 80 years. The arcus is formed by deposition of cholesterol, cholesterol esters, phospholipids and triglycerides in the substantial propria layer.

**Arcus Juvenilis**
It usually occurs below 40 years of age. It is a rare condition. A serum lipid profile is indicated to rule out hereditary anomaly which has a serious prognosis. 

**White Limbal Girdle of Vogt**
It is seen mostly in the age group of 40-60 years. It is seen as chalky line in the nasal and temporal periphery of inter-palpebral area of cornea. The opacity is at the level of Bowman’s membrane. It is due to elastotic degeneration of tissues.

**Amyloid Degeneration**
Amyloid degeneration of cornea is characterized by deposition of amyloid material underneath its epithelium. It could be either primary or secondary due to some disease.

**Pigmentary Degeneration**
Pigment deposition in cornea could be iron, blood pigment, melanin and other metallic pigments like cooper, silver, gold etc.

a. Hudson-Stahli-line—it is a horizontal line at the lower half of the cornea due to deposition of hemosiderin pigment
b. Fleischer’s ring is seen at the base of keratoconus
c. Stocker-Busaca line—it is seen in front of a Pterygium.

**Band-shaped Keratopathy**
• It is common in old, blind, shrunked eyes and in Still’s
disease of children.
• It is associated with hyperthyroidism, vitamin D
poisoning
or sarcoidosis.
• It could be either primary or secondary to
hypercalcaemia, chronic uveitis, chronic glaucoma,
interstitial keratitis etc.
• A continuous band lies in the interpalpebral area
starting in the inner and outer side.
• It is due to hyaline infiltration in the superficial
stroma followed by calcareous salt deposition
1. **Reis-Buckler’s Dystrophy**

   It is bilaterally symmetrical dystrophy, which starts in early childhood as recurrent corneal erosions. Later there is diffuse scarring of Bowman’s membrane. Corneal surface becomes rough with diminished sensation. The opacities have typical ring like appearance. It is an autosomal dominant condition. It starts near the Bowman’s membrane. There are subepithelial grey opacities arranged in a fish net pattern.

2. **Cogan’s Microcystic Dystrophy**
It is the commonest epithelial dystrophy with dot, map or fingerprint opacities characterized by recurrent attacks of severe pain, watering, photophobia and blepharospasm. There is increased hydration of cornea and formation of microcysts under the epithelium.

3. **Messman’s Juvenile Epithelial Dystrophy (Recurrent Corneal Erosion Syndrome)**
   Characterized by appearance of small vesicles between epithelium and Bowman’s membrane. It is an autosomal dominant inherited bilateral symmetrical condition. There are minimum symptoms and visual loss is very less, hence it does not require any treatment.

**STROMAL DYSTROPHIES**

1. **Granular Dystrophy**
   - It is autosomal dominant dystrophy.
   - There is milky granular hyaline deposits in anterior stroma.
   - There is clear cornea between opacities.
   - It develops in first decade of life and vision remains good until 40 years of life.

2. **Macular Dystrophy**
   - It is autosomal recessive dystrophy.
   - There is dense opacity in central cornea.
   - There is deposition of mycopolysaccharides.
• It starts in first decade and vision lost early in life.
• It requires penetrating keratoplasty.

3. Lattice Dystrophy
• There is autosomal dominant inheritance.
• There are amyloid deposits in corneal stroma.
• Spider like opacities are seen in cornea.
• It starts early in life.
• Cornea becomes hazy by the age of 20 years.
• It requires penetrating keratoplasty.

ENDOTHELIAL DYSTROPHIES
1. Fuch’s Endothelial Dystrophy
• It was described by Fuch’s in 1910.
• It usually occurs after 50 years of age.
• The female to male ratio is 4:1.
• There is atrophy of the endothelial cells along with oedema and formation of vesicles. Grey punctate opacities are seen in the stroma.
• The clinical features are divided into four stages
  a. Stage of cornea guttata
  b. Oedematous stage
  c. Stage of bullous keratopathy
  d. Stage of scarring
  e. Treatment
  f. a. 5% sodium chloride ointment or solution (hypertonic saline) is useful.
  g. b. Hydrated soft contact lenses may be useful.
h.c. Penetrating keratoplasty can be done.

**i. 2. Cornea Guttata**

j. • It manifests are middle age.
k. • Females are most affected.
l. • It has autosomal dominant inheritance.
m. • There are bilateral symmetric lesions, which appear as golden hue on the posterior surface of cornea.
• It rarely affects vision.

---

**PIGMENT DEPOSITION IN THE CORNEA**

<table>
<thead>
<tr>
<th>TYPE OF PIGMENT</th>
<th>DISORDERS</th>
<th>CORNEAL LOCATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>IRON</td>
<td>• Keratoconus (Fleischer’s ring)</td>
<td>Epithelium</td>
</tr>
<tr>
<td></td>
<td>• Pterygium (Stocker’s line)</td>
<td>Epithelium</td>
</tr>
<tr>
<td></td>
<td>• Filtering bleb (Ferry’s line)</td>
<td>Epithelium</td>
</tr>
<tr>
<td></td>
<td>• Old opacity (Hudson-Stähli line)</td>
<td>Epithelium</td>
</tr>
<tr>
<td></td>
<td>• Siderosis</td>
<td>Mainly stroma</td>
</tr>
<tr>
<td></td>
<td>• Blood staining of the cornea</td>
<td>Mainly stroma</td>
</tr>
<tr>
<td>COPPER</td>
<td>• Wilson’s disease</td>
<td>Descemet’s membrane</td>
</tr>
<tr>
<td></td>
<td>• Chalconosis</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• (Kayser-Fleischer ring)</td>
<td></td>
</tr>
<tr>
<td>MELANIN</td>
<td>• Pigment dispersion syndrome</td>
<td>Endothelium</td>
</tr>
<tr>
<td></td>
<td>• (Krukenberg’s spindle)</td>
<td></td>
</tr>
</tbody>
</table>

---

**TARSORRHAPHY**

The aim is to achieve lid closure so that palpebral aperture is narrowed. It is not strictly a corneal operation, but it is performed for corneal conditions.

**Indications**

• Neuroparalytic keratitis as a result of 5th nerve paralysis.
• Exposure keratitis due to inadequate closure of palpebral aperture as a result of 7th nerve paralysis or proptosis.

**Method**

Palpebral aperture is narrowed by placement of mattress sutures through the small raw areas in the lid margins and skin. The sutures are tied over rubber sheet in the skin.

**Types**

1. *Lateral tarsorrhaphy*—The suture is placed at the junction of middle and lateral third of lid margin.
2. *Paramedian tarsorrhaphy*—Two sutures are placed on either side of the middle line as shown in the diagram.

**EYE BANK**

The primary function of an eye bank is to collect, store good quality donor’s cornea and make it available for cornea transplantation for therapeutic use and research.

**Objective of Eye Bank**

The main objectives of an eye bank can be summarized as follows,

1. Collection of donor eyes.
2. Preservation of donor cornea.
3. Distribution of highest quality of donor tissue for cornea transplantation.
4. Promotion, awareness about eye donation from potential donors.

A person makes a pledge to donate his eyes after death. No living individual can donate his eye because the law does not permit it and moreover it is not practical. The eye cannot be sold or purchased. Eye bank personnel collect the eyes after getting information about death and proper written consent from close relative. It is important to know the age of the donor, cause of death and time of death. Eyes should be removed as early as possible or at least within 5-6 hours after death.

**Equipments for an Eye Bank**

The equipments required for any eye bank are listed below,

<table>
<thead>
<tr>
<th><strong>COMPULSORY</strong></th>
<th><strong>DESIABLE</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>• Refrigerator with temperature recording device</td>
<td>• Incubator</td>
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<td>• Operation theatre</td>
<td>• Specular microscope</td>
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<td>• Slit-lamp</td>
<td>• Ice machine</td>
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<td>• Sterilization facilities</td>
<td>• Microbiology facilities</td>
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<td>• Enucleation and corneal excision instruments</td>
<td>• Centrifuge</td>
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**Enucleation**

Eyes should be enucleated soon after death. A relatively longer interval of 4-6 hours may be allowed
in winter months but in summer, not more than 2-3 hours should elapse between death and enucleation. The eyes should carry the following information about the donor:
1. Age and sex.
2. Cause of death.
3. Time and date of death.
4. Time and date of enucleation.
Enucleation should be done aseptically and the eyeballs should be transported to the eye bank in a wide mouth sterile glass bottle in an ice box or thermos flask. The eyeballs are washed with normal saline, antibiotic drops instilled and the cornea is examined with good illumination and magnification, preferably with slit-lamp. Clinical viability is graded depending upon the degree of stromal oedema and folds. Usable eyeballs are then transferred to autoclaved wide mouth bottles containing sterile cotton gauze pad. Adequate antibiotic solution is instilled to moisten the pad. The eyeball rests on the pad with cornea straight up and without touching any part of the bottle. It is better to have a mouldable clamp in the bottle to hold the eyeball erect to protect the cornea. This is particularly important when these eyes are to be transported to distant corneal surgeons. The bottles should have tight screw
caps so that even if ice has melted off in the container, fluid does not enter the bottle. The Eye Bank Association has designed a thermocol container which has provision for carrying one or two pairs of eyeballs with adequate amount of ice for 18 to 24 hours transport. If Descemet’s membrane folds and stromal oedema exceed acceptable limits, the cornea is designated unusable for therapeutic purpose but can be used for surgical training and experimental purposes. One may conclude that when media preservation facilities are not available, eyeballs enucleated from a relatively younger person who dies after an acute episode such as accident, suicide, homicide, etc. and preserved in moist chamber at 4o C provide the best donor cornea.

**Contraindications for Collection of Donor Eyes**

There are certain conditions when the donor’s eye are not suitable for corneal transplant.

1. **Systemic causes**—These include death due to:
   - AIDS (HIV positive)
   - Hepatitis B
   - Rabies
   - Poison
   - Severe burn
   - Malignancy, leukaemia, lymphoma
• Death from unknown cause.

2. Ocular causes
• Corneal opacities and dystrophy
• Retinoblastoma, malignant melanoma
• Active inflammatory diseases, e.g. conjunctivitis, iridocyclitis, endophthalmitis
• Congenital abnormalities, e.g. keratoconus, keratoglobus
• Prior refractive procedures, e.g. radial keratotomy, laser photoablation
• Anterior segment surgical procedures, e.g. cataract, glaucoma.

Evaluation of Donor Tissue
1. Gross examination with torch and loupe.
2. Slit-lamp examination.
3. Specular microscopy (for endothelial cell count and morphology).

A careful slit-lamp examination provides an overall status of the endothelium. The normal endothelium shows a pattern of cells of similar size and shape with no abnormal structures. The normal cell density is usually between 2000 to 3500 cells per sq. mm.
Preservation of the Donor Eye

Traditionally corneal preservation is described under short-term, intermediate-term and long-term preservations.

1. **Short-term Preservation (up to 96 hours)**
   i. **Moist chamber method**—Whole globe is preserved in a moist chamber at 4°C in a refrigerator for 24 hours.
   ii. **M.K. (McCarey-Kaufman) medium**—It consists of tissue culture (TC)-199, 5% Dextran-40, HEPES buffer to adjust pH at 7.4, Gentamicin 0.1 mg/ml, colour-pink. Corneoscleral button can also be preserved in M-K medium at 4°C for up to 96 hours. It is superior than conventional moist-chamber method and practised widely.

2. **Intermediate-term Preservation (upto 2 weeks)**
   K-SOL medium, *dexol* medium, *optisol* medium, etc. are used for preservation.

3. **Long-term Preservation (months to years)**
   i. **Viable**—Organ culture method, cryopreservation.
ii. *Non-viable*—Glycerine preservation.