शालाक्यतंत्र

(अर्वाचीन मतानुसार)

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red'shine
Publication

SHALAKYTRANTRA (ARVACHIN MATANUSAR)

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REDSHINE Publication Pvt. Ltd.

Headquarters (India): 88, Patel Street, Navamuvada,

Lunawada, India-389 230

Contact: +91 76988 26988

Registration no. GJ31D0000034

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ISBN: 978-93-87822-06-1 (Hardcover)

eISBN: 978-93-87822-07-8 (eBOOK)

Price: ₹ 350

DIP: 18.10.061/87822

March, 2018 (First Edition)

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www.redmac.in | info.redmac@gmail.com

In association with **RED'MAC Networks**,

India | Sweden | Canada

Printed in India | Title ID: 87822061

DEDICATED TO MY PARENTS

Mr. Harsukhlal J. Agravat

Mrs. Jyotsanaben H. Agravat

ACKNOWLEDGEMENT

Vd. Nehal Nimavat

Vd. Yogesh Manani

Vd. Himanshu Kanzaria

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CHAPTER 1
DISEASES OF EYE

Anatomy of eyeball

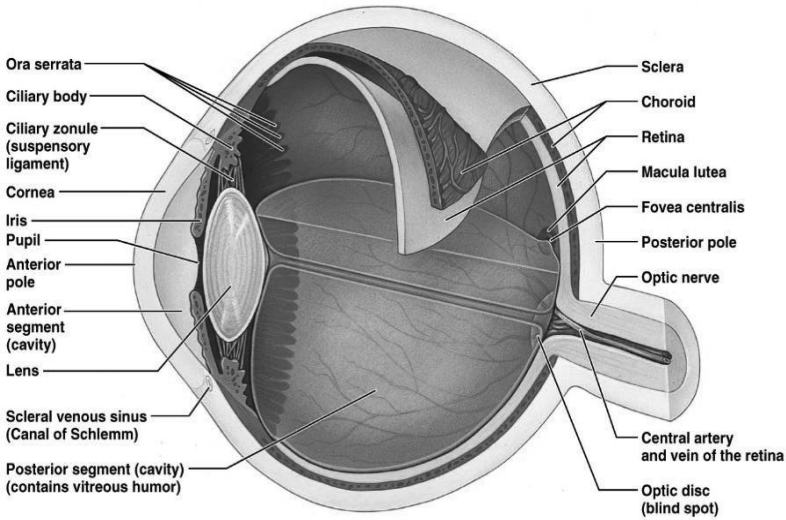
- Each eye ball is a cystic structure kept distended by the pressure inside it.

Dimensions:

- Anteroposterior diameter : 24 mm
- Horizontal diameter : 23.5 mm
- Vertical diameter : 23 mm
- Circumference : 75mm
- Volume : 6.5 ml
- Weight : 7 gm

Coats of eyeball:

- **Outer fibrous coat:**
  - Anterior $1/6^{th}$ is transparent : **cornea**
  - Posterior $5/6^{th}$ is opaque : **sclera**
- **Middle vascular coat (uveal tissue)**:
  - Supplies nutrition to the various structures of the eyeball.
  - It consists of 3 parts: from anterior to posterior:
    1. Iris
    2. Ciliary body
    3. Choroid
- **Inner nervous coat (Retina):**
  - It is concerned with visual functions.

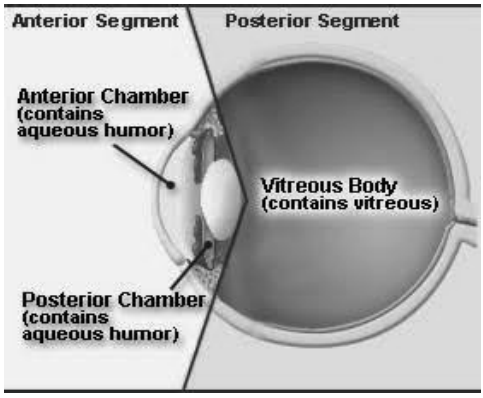


Segments and chambers of the eyeball:

1. **Anterior segment** :
   - Includes crystalline lens and structures anterior to it, viz. Iris, cornea and two aqueous humour filled spaces – anterior and posterior chambers.
     - **I. Anterior chamber:**
       - Bounded anteriorly by back of cornea and posteriorly by the iris and part of ciliary body.
       - About 3 mm deep in the centre
       - Shallower in hypermetropes and deeper in myopes
       - Contains 0.25 ml of aqueous humour
     - **II. Posterior chamber**:
       - A triangular space containing 0.06 ml of aqueous humour
       - Bounded anteriorly by the posterior surface of iris and part of ciliary body and posteriorly by the crystalline lens

and its zonules, and laterally by the ciliary body.

2. **Posterior segment**:
   - Includes the structures posterior to lens, viz. Vitreous humour, retina, choroid and optic disc.



Orbit

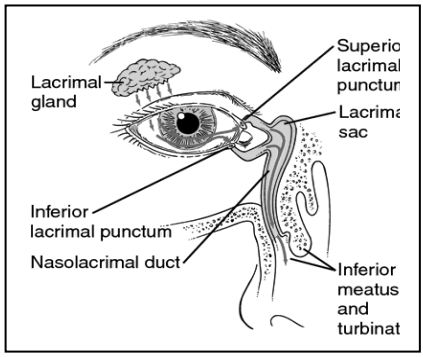
- Each eyeball is suspended by extraocular muscles and fascial sheaths in a quadrilateral pyramid shaped bony cavity called Orbit.

Appendages of the eye

- Each eye is protected anteriorly by two shutters called the ***eyelids.***
- The anterior part of sclera and posterior surface of lids are lined by a thin membrane called ***conjunctiva***.
- For smooth functioning, the cornea and conjunctiva are to be kept moist by tears which are produced by ***lacrimal gland*** and drained by the lacrimal passages.
- These structures (eyelids, eyebrows, conjunctiva and lacrimal apparatus) are collectively called the appendages of the eye.

The lacrimal apparatus

- Comprises the structures concerned with the formation of tears (main lacrimal gland and accessory lacrimal glands) and its transport.
- The lacrimal passage includes:
  - Puncta
  - Canaliculi
  - Lacrimal sac
  - Nasolacrimal duct



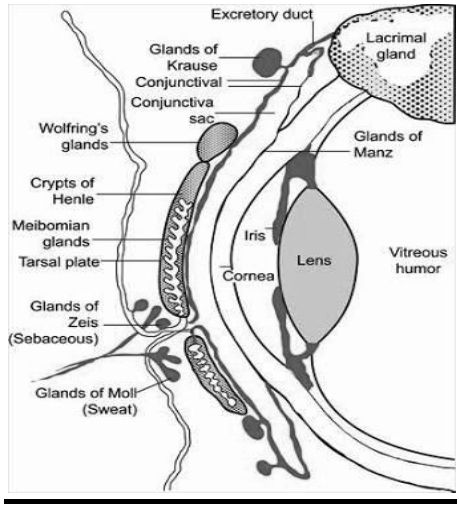
Lacrimal glands

Main lacrimal gland

- Situated in the fossa for lacrimal gland, formed by the orbital plate of frontal bone, in the anterolateral part of the roof of orbit.
- Ducts:
  - 10-12 ducts open in the lateral part of superior fornix
  - 1 or 2 ducts open in the lateral part of inferior fornix

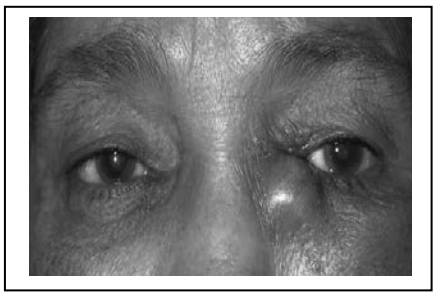
Accessory lacrimal glands

- Glands of Krause:
  - Microscopic glands lying in subconjunctival tissue of fornices
  - 40-42 in upper fornix and 6-8 in lower fornix
- Glands of Wolfring
  - Microscopic glands present along the upper border of superior tarsus and lower border of inferior tarsus.



DACRYOCYSTITIS

- Inflammation of the lacrimal sac.



Chronic Dacryocystitis

- More common than acute dacryocystitis.
- Etiology:
  - Predisposing factors
  - Factors responsible for stasis of tears in the lacrimal sac
  - Source of infection
  - Causative organisms

Predisposing factors:

- Age: more common between 40-60 years of age
- Sex: predominantly in females (80%)
- Lower socio-economic status
- Poor personal hygiene

Factors responsible for stasis of tears in the lacrimal sac:

- Anatomical factors which retard drainage of tears
- F.B. in the sac
- Excessive lacrimation, primary/reflex, causes stagnation of tears in the sac
- Mild grade inflammation of the lacrimal sac due to associated recurrent conjunctivitis may block the NLD
- Obstruction of lower end of NLD by nasal diseases such as polyps, hypertrophied inferior turbinate, marked degree of DNS, tumours etc.

Source of infection:

- Lacrimal sac may get infected from the conjunctiva, nasal cavity or PNS.

Causative organism:

- Staphylococci
- Pnemococci
- Streptococci
- Pseudomonas pyocyanea

Clinical features

- Stage of chronic catarrhal dacryocystitis
- Stage of lacrimal mucocele
- Stage of chronic suppurative dacryocystitis
- Stage of chronic fibrotic sac

Chronic catarrhal dacryocystitis	Lacrimal mucocele	Chronic suppurative dacryocystitis	Chronic fibrotic sac
Watering eye is the only symptom. Sometimes mild redness in inner canthus.	Constant epiphora associated with a swelling just below the inner canthus	Epiphora, associated with recurrent conjunctivitis, swelling at the inner canthus with mild erythema of overlying skin	Low grade repeated infections for a prolonged period ultimately result in a small fibrotic sac due to thickening of mucosa, which is often associated with persistent epiphora and discharge
On syringing the lacrimal sac, either clear fluid or few fibrinous mucoid flakes regurgitate	Milky or gelatinous mucoid fluid regurgitates from the lower punctum on pressing the swelling.	A frank purulent discharge flows from the lower punctum	

Treatment:

- Conservative:
  - Probing or lacrimal syringing
- Balloon catheter dilation(balloon dacryocystoplasty)
  - In patients with partial NLD obstruction
- Dacryocystorhinostomy (DCR)
- Dacryocystectomy (DCT)
- Conjunctivodacryocystorhinostomy (CDCR)

Acute Dacryocystitis

- Acute suppurative inflammation of the lacrimal sac, characterized by presence of a painful swelling in the region of sac.

Etiology:

- It may develop in two ways:
  - As an acute exacerbation of chronic dacryocystitis
  - As an acute peridacryocystitis due to direct involvement from the neighbouring infected structures such as: PNS, surrounding bones and dental abscess or caries teeth
- Causative organism:
  - Streptococcus hemolyticus, pneumococcus, staphylococcus.

Clinical features:

- Stage of cellulitis
- Stage of lacrimal abscess
- Stage of fistula formation

Fistula formation

Stage of cellulitis	Stage of lacrimal abscess	Stage of fistula formation
Painful swelling in the region of lacrimal sac	Occlusion of canaliculi due to oedema. The sac is filled with pus, distends and its anterior wall ruptures forming a pericystic swelling	When lacrimal abscess is left untreated, it discharges spontaneously, leaving an external fistula below the medial palpebral ligament.
Epiphora Constitutional symptoms: fever, malaise		

Treatment:

- Cellulitis stage:
  - Systemic and topical antibiotics
  - Systemic anti-inflammatory analgesics
  - Hot fomentation
- Stage of lacrimal abscess
  - Drainage with a small incision
- External lacrimal fistula
  - Fistulectomy along with DCT or DCR

THE WATERING EYE

- Overflow of tears from the conjunctival sac.
  - Hyperlacrimation: excessive secretion of tears
  - Epiphora: inadequate drainage (outflow) of normally secreted tears.

Epiphora

Causes:

- Physiological causes
- Mechanical causes

- Physiological causes: lacrimal pump failure due to lower lid laxity or weakness of orbicularis muscle.
- Mechanical obstruction in lacrimal passage
  - Punctal causes
  - Causes in the canaliculi
  - Causes in the lacrimal sac
  - Causes in the NLD

1. Punctal causes:
   - Eversion of lower punctum: commonly seen in old age due to laxity of lids. It may also occur following chronic conjunctivitis, chronic blepharitis
   - Punctal obstruction: there may be congenital absence of puncta or cicatricial closure following injuries, burns or infections.
2. Causes in the canaliculi: may be congenital or acquired due to F.B., trauma, canaliculitis.
3. Causes in the lacrimal sac: congenital mucous membrane folds, traumatic strictures, dacryocystitis
4. Causes in the NLD:
   - Congenital: noncanalization, partial canalization
   - Acquired: traumatic strictures, inflammatory strictures, tumours, diseases of the surrounding bones

Clinical evaluation

- Ocular examination with diffuse illumination using magnification: to rule out any cause of reflex hypersecretion located in lids, conjunctiva, sclera, cornea etc.
- Regurgitation test

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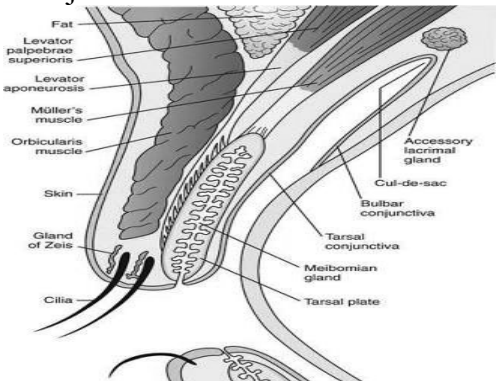
- FDDT
- Lacrimal syringing test
- Jones dye test
- Dacryocystography

ANATOMY OF EYELID

- Mobile tissue curtains placed in front of the eyeballs.
- Act as shutters protecting the eyes from injuries and excessive light.
- Also perform an important function of spreading the tear film over the cornea and conjunctiva.
- **Position**: the upper eyelid covers about one-sixth of the cornea and lower eyelid just touches the cornea
- **Canthi**: The two eyelids meet each other at medial and lateral angles (inner and outer canthi)
- **Eyelashes**: arranged in 2-3 rows.
    - Those in upper lid (100-150 in number) are directed forward, upward and backward
    - Those in lower lid (50-75 in number) are directed forward, downward and backward.
    - The cilia are darker than the scalp hair.
    - Each cilium has a lifespan of some 3-4 months. At the termination of this period, the old cilium drops away, the follicles rests for several weeks and then a new cilium grows out.
- **Structure**
    - Skin
    - Layer of subcutaneous areolar tissue
    - Layer of striated muscles
    - Submuscular areolar tissue
    - Fibrous layer
    - Layer of non-striated muscle fibres.
    - Conjunctiva



STYE

Acute suppurative inflammation of lash follicle and its associated glands of Zeis or Moll.

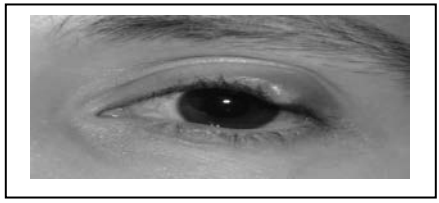
CLINICAL FEATURES:

Symptoms:

- Acute pain associated with swelling of lid
- Mild watering
- Photophobia

Signs:

- Stage of cellulitis: localized firm, red, tender swelling at the lid margin associated with marked oedema
- Stage of abscess formation: a visible pus point on the lid margin in relation to the affected cilia.



Treatment:

- Hot compress 2-3 times a day. Very useful in cellulitis stage
- Evacuation of the pus by epilating the involved cilia, when the pus point is formed. Surgical incision is required rarely for a large abscess.
- Antibiotic eye drops and ointment
- Systemic anti-inflammatory and analgesics to relieve pain and oedema
- Systemic antibiotics for early control of infection.

	Stye	Chalazion
Location	Most commonly found at or near an eyelash follicle, but can be higher or lower than the lashes.	Most commonly found above the eyelashes on the upper lid, but can be found below the lashes on the lower lid.
Cause	Bacterial infection either at the root of the eyelash follicle or in the oil gland of the lids.	A blocked oil gland (Meibomian gland).
Symptoms	Tenderness similar to a pimple; can cause swelling of the lid with subsequent tearing.	Firm lump that does not hurt, however if they get large enough to press on the globe and affect the vision. A chalazion grows slower and takes longer to go away than a stye.
Treatment	Many times styes open and drain on their own in about 3 days, however warm compresses will assist in opening the stye.	Chalazie (plural) typically resolve within a month by use of warm compresses and sometimes eye drops. There are times when lancing by the doctor is required to drain the blockage.

CHALAZION

- Chronic non-infective granulomatous inflammation of the meibomian gland.

Etiology:

- Predisposing factors:
- Habitual rubbing of the eyes or fingering of the lids and nose
- Chronic blepharitis and DM usually associated with recurrent chalazion
- Metabolic factors, chronic debility, excessive intake of carbohydrates and alcohol

Pathogenesis

- First there occurs mild grade infection of meibomian gland by organism of very low virulence.
- As a result, there occurs proliferation of the epithelium and infiltration of the walls of the ducts, which are blocked.
- There occurs retention of secretions (sebum) in the gland, causing its enlargement.

Clinical picture:

Symptoms:

- Painless swelling in the eyelid, gradually increasing in size
- Mild heaviness in the lid
- Blurred vision may occur due to induced astigmatism by a very large chalazion pressing on the cornea.

Signs:

- Nodule slightly away from the lid margin. Firm to hard. Non-tender.
- Upper lid is involved more commonly than the lower lid probably because the upper lid contains more meibomian glands than the lower lid.
- Reddish purple area seen on the palpebral conjunctiva after eversion of the lid.

Treatment:

- Conservative treatment: in small, soft, recent chalazion
- Hot fomentation, topical antibiotic eye drops and oral anti-inflammatory drugs.
- Intralesional injection of long-acting steroid (triamcinolone) is reported to cause resolution in 50% cases
- Incision and curettage
- Diathermy
- Oral tetracycline as prophylaxis in recurrent chalazia

TRACHOMA

- Previously known as Egyptian ophthalmia
- Chronic keratoconjunctivitis primarily affecting the superficial epithelium of conjunctiva and cornea simultaneously.
- Characterized by a mixed follicular and papillary response of conjunctival tissue.
- The infection causes roughening of the inner surface of eyelids.
- Roughening can lead to pain in the eyes, breakdown of the outer surface of cornea and blindness.
- Untreated, repeated trachoma infections can result in a form of permanent blindness when the eyelids turn inwards.

Etiology:

- Causative organism: Chlamydia trachomatis.

Predisposing factors:

- Age: usually contracted during infancy and early childhood
- Sex: more in females
- Climate: dry and dusty weather
- Socioeconomic status: poor classes owing to unhygienic living condition, overcrowding, unsanitary conditions, abundant fly population, lack of material like separate towels and handkerchiefs, lack of education
- Environmental factors: exposure to dust, smoke, irritants, sunlight etc increase the risk

Source of infection:

conjunctival discharge of the affected person.

Modes of infection:

- Direct spread: air-borne or water-borne modes
- Vector transmission through flies

- Material transfer: through contaminated fingers of doctors, nurses, contaminated tonometers. Use of common towels, handkerchiefs, bedding, surma-rods.

SYMPTOMS

In the absence of secondary infection:

- Mild F.B.sensation
- Occ. Lacrimation
- Slight stickiness of the lids
- Scanty mucoid discharge

In the presence of secondary infection:

- Symptoms of acute mucopurulent conjunctivitis.

Signs:

Conjunctival signs	Corneal signs
1. **Congestion** of upper tarsal and forniceal conjunctiva 2. **Conjunctival follicles**: look like boiled sagograins and commonly seen on upper tarsal conjunctiva and fornix. May also be present in lower fornix 3. **Papillary hyperplasia**: papillae are reddish raised areas which give red and velvety appearance to the tarsal conjunctiva. 4. **Conjunctival scarring** 5. **Concretions**: hard looking whitish deposits varying from pin point to 2mm in size. Formed	1. **Superficial keratitis** 2. **Herbert follicles**: present in the limbal area. 3. **Pannus**: infiltration of the cornea associated with vascularisation is seen in the upper part. 4. **Corneal ulcer**: may sometime develop at the advancing stage of pannus. 5. **Herbert pits**: oval or circular pitted scars, left after healing of herbert follicles in the limbal area. 6. **Corneal opacity**: may be present in the upper part. May even extend down and involve the

due to accumulation of dead epithelial cells and inspissated mucus in the depressions called glands of henle.	papillary area. It is the end result of trachomatous corneal lesions.

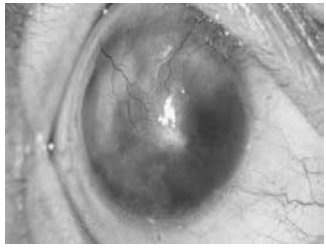
WHO Classification:

1	TF – Trachomatous inflammation – Follicular	Active disease which needs treatment	5 or more follicles of at least 0.5mm diameter on the upper tarsal conjunctiva. Palpebral conjunctival blood vessels are visible.
2	TI – Trachomatous inflammation- Intense	Severe disease which needs urgent treatment	Follicles and papillae are so numerous and inflamed that more than 50% of the palpebral conjuntival blood vessels cannot be seen clearly.
3	TS – Trachomatous Scarring	Old, now inactive infection	Tarsal conjunctival cicatrisation with white fibrous bands
4	TT – Trachomatous Trichiasis	Needs corrective surgery	At least one eyelash rubs the eyeball.
5	TO – Trachomatous Opacities	Cause visual loss	Presence of corneal opacity covering part of the pupillary region.


Scarring

Trachomatous inflammation


Trichiasis


Corneal opacity

Treatment

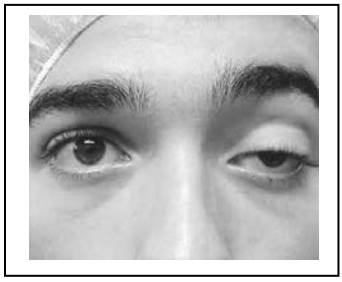
Topical	Systemic
• Tetracycline (1%) or erythromycin (1%) eye ointment 4 times a day for 6 weeks • Sulfacetamide (20%) eye drops three times a day along with 1% tetracycline eye ointment at bed time for 6 weeks	• Tetracycline or erythromycin 250 mg orally, four times a day for 3-4 weeks or • Doxycycline 100mg orally twice daily for 3-4 weeks or • Azithromycin 1gm stat or 250mg OD 4 days.

PTOSIS

- Abnormal drooping of the upper lid to a level that covers more than 2 mm of the superior cornea.
- Elevation is a function of the levator palpebrae superioris, assisted by the frontalis and muller's muscle.
- Ptosis occurs usually due to paralysis or defective development of the LPS.
- A purely mechanical ptosis may be due to deformity and increased weight of the lid brought about by trachoma or tumour.
- An apparent drooping of the lid – pseudoptosis - may be due to lack of support as in phthisis bulbi or enophthalmos.

Classification:

- Congenital
- Acquired:
  - Neurogenic
  - Myogenic
  - Aponeurotic
  - Mechanical



Congenital:

- Simple ptosis due to maldevelopment of the levator muscle
- Ptosis associated with congenital weakness of the superior rectus

Acquired:

- **Neurogenic:**
  - Due to partial or complete 3rd nerve palsy affecting the levator
  - Horner Sydrome: paralysis of the muler's muscle
- **Myogenic:**
  - Trauma to the levator muscle

  - Muscular dystrophy of the levator muscle as in ocular myopathy
  - Myasthenia gravis.
    - *(Painless weakness of muscles*
    - *The weakness becomes progressively worse during periods of physical activity*)
- **Aponeurotic:**
  - Involutional
  - Due to weakness or disinsertion of LPS aponeurosis from the anterior surface of tarsus.
- **Mechanical:**
  - When tumour or inflammation weigh down the lid and cause it to droop.

Degree of Ptosis:
- Mild : 2 mm
- Moderate : 3 mm
- Severe : 4 mm or more

Symptoms:
- There is no symptom, if the pupil is not covered by the lid. Otherwise there is visual disturbance.

Treatment:
- Fasanella – Servat operation : for mild ptosis
- Levator resection: moderate to severe ptosis
  - Blaskovic's operation(conjunctival approach)
  - Everbusch's operation (Skin approach)
- Frontalis sling operation (Brow suspension) : for severe ptosis with no levator function

LAGOPHTHALMOS

Incomplete closure of the palpebral aperture when an attempt is made to shut the eyes

Causes

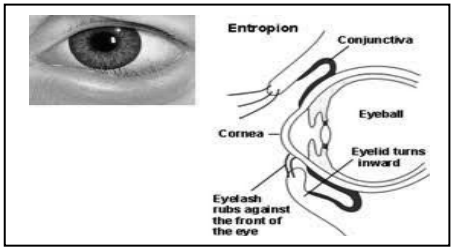
- Contraction of lids from cicatrization
- Congenital deformity
- Ectropion
- Paralysis of the orbicularis
- Proptosis due to exophthalmic goitre
- Laxity of the tissues

Treatment:

- Artificial tear drops to prevent conjunctival/corneal dryness
- Lateral tarsorrhaphy : paralytic lagophthalmos
- Skin graft : in cicatricial cases

ENTROPION

A condition in which lid margin rolls inwards



Classification:

- Involutional
- Cicatricial
- Spastic
- congenital

Involutional / Senile:

- Lack of support of the eyelid due to disappearance of orbital fat and also due to atrophic and inelastic condition of the skin in senility.
- Treated by keeping the lower lid pulled downwards by a strip of adhesive plaster
- Plastic operation – Skin muscle operation

Cicatricial:

- Caused by cicatricial contraction of the palpebral conjunctiva resulting in inversion of lid margin
- Its most severe form is found in trachoma.
- Other causes are trauma, chemical burns, Steven-Johnson syndrome
- Treatment: Plastic operation

Spastic:

- Develops typically in a case of spasm of orbicularis in the presence of degeneration of palpebral connective tissue separating the orbcularis muscle fibres.
- This generally occurs in response to ocular irritation such as inflammation or trauma.
- Also occurs after prolonged bandaging of the eye.
- If the aponeurosis degenerates, strong contraction of circularly arranged orbicularis tends to approximate the lid margins and turn them inwards.
- The lower lid is usually affected.

Congenital:

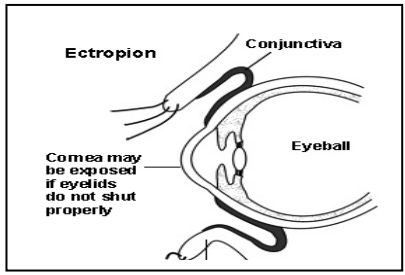
- Rare
- Due to dysgenesis of the lower lid retractors or a developmental abnormality of the tarsal plate, causing the lid margin to turn onto the globe.

ECTROPION

A condition in which the lid margin rolls outwards i.e. becomes everted

Classification:

- Acquired
  - Involutional / senile
  - Cicatricial
  - Paralytic
  - mechanical
- Congenital



Senile:

- As a result of laxity of the tissues of the lid and loss of tone of the orbicularis muscle.

Cicatricial

- Commonly the result of burns, trauma, chronic inflammation of skin which shorten the anterior lamina of the eyelid i.e. the skin-muscle layers.

Paralytic

- As a result of weakness of the orbicularis muscle due to paralysis of the facial nerve.
- The lower lid is only affected.

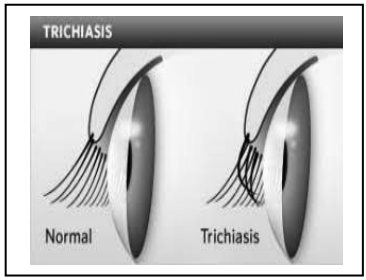
Mechanical

- Caused by weight of a mass in the eyelid e.g. tumour or by pressure on the eyelid from behind as in proptosis.

TRICHIASIS

- Misdirection of cilia so that they are directed backwards and rub against the cornea.
- The position of the lid margin is normal.
- Any condition causing entropion will cause trichiasis.
- Trachoma and spastic entropion are the most common causes.
- Other causes: blepharitis, scars resulting from injuries, chemical burns, operations.
- A few of the lashes may be affected or the condition may be due to entropion involving the whole margin of the lid.

Symptoms:



- F.b. sensation
- Irritation
- Pain
- Conjunctival congestion
- Reflex blepharospasm
- lacrimation
- Recurrent erosions
- Superficial opacities
- Vascularization of cornea
- Frequent, recurrent corneal ulcers
- Trichiasis may prevent corneal ulcers from healing, thereby threatening the vision.

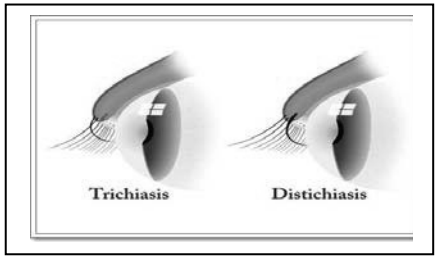
Treatment:

- Epilation : mechanical removal by forcep
- Electrolysis: destroying the lash follicle by electric current
- Surgery
- Lubricants such as artificial tears and ointments, may decrease the irritant effect of lash rubbing.

DISTICHIASIS

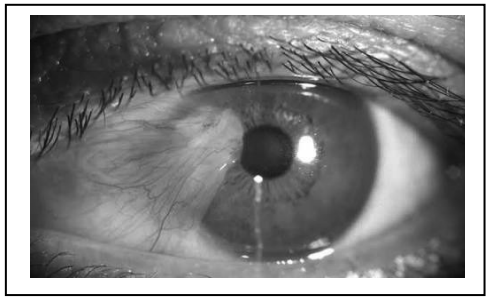
- Congenital abnormality of the eyelid
- Rare

- An extra posterior row of cilia
- The posterior row occupies the position of the meibomian glands.
- These lashes may irritate the cornea



PTERYGIUM

- Degenerative condition
- A wing shaped triangular fold of conjunctiva encroaching upon the cornea from either side within the interpalpebral fissure.



Etiology:

- Not definitely known
- More common in people living in hot climates
- So, it may be a response to prolonged effect of environmental factors such as exposure to sun (UV rays), dry heat, high wind and abundance of dust

Pathology:

- Degenerative and hyperplastic condition of conjunctiva
- Subconjunctival tissue undergoes elastotic degeneration and proliferates as vascularised granulation tissue under the epithelium, which ultimately encroaches the cornea.
- The corneal epithelium, Bowman's layer and superficial stroma are destroyed.

Parts:

- Head: apical part present on the cornea
- Neck: constricted part present in the limbal area
- Body: scleral part, between limbus and canthus
- Cap: semilunar whitish infiltrate present just in front of the head.

Symptoms

- Cosmetic intolerance may be the only issue in otherwise asymptomatic condition in early stages
- F.B. sensation
- Defective vision when it encroaches the pupillary area or due to corneal astigmatism induced by fibrosis in the regressive stage.
- Diplopia may occur occasionally due to limitation of ocular movements.

Signs:

- Triangular fold of conjunctiva encroaching on the cornea in the area of palpebral aperture usually on the nasal side, but may also occur on the temporal side.
- Very rarely, both sides are involved (primary double pterygium)

Types:

- **Progressive**: thick, fleshy and vascular with a few infiltrates in the cornea, in front of the head of the pterygium.
- **Regressive**: thin, atrophic, attenuated with very little vascularity. There is no cap, but deposition of iron (stocker's line) may be seen sometimes, just anterior to head of pterygium. Ultimately it becomes membranous but never disappears.

PTERYGIUM

TRUE PTERYGIUM	PSEUDOPTERYGIUM
Age - ≥ 40 yrs	Any age
Palpebral fissure region	Anywhere
Organisation into head,body and tail	No such organisation
Firm adhesion at limbus	No such adhesion
Probe test : cannot be passed under pterygium	probe can be passed
Always progressive initially	Always stationary
No such history	History of severe conjunctivitis / chemical burns

Treatment:

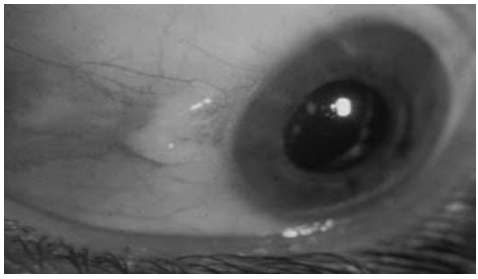
- Surgical excision: is the only satisfactory treatment, which may be indicated for
  - Cosmetic reason
  - Continued progression threatening to encroach onto the papillary area (once the pterygium has encroached the pupillary area, wait till it crosses on the other side)
  - Diplopia due to interference in ocular movements.

Surgical Excision:

- Topical anaesthesia
- Clean and drape
- Apply universal eye speculum to expose the part
- Head of the pterygium is lifted and dissected
- The mass of pterygium is separated from sclera and conjunctiva for about half the distance towards the canthus
- Carefully pterygium is excised
- Haemostasis
- Cauterization of exposed episcleral tissues
- Conjunctiva sutured back to cover the sclera

PINGUECULA

- Common degenerative condition of the conjunctiva
- Triangular patch on the bulbar conjunctiva near the limbus, the apex of the triangle being away from the cornea and affects nasal side first, then the temporal.
- It is yellow in colour and looks like fat, hence the name (pinguis – fat)
- Etiology is unknown
- Usually found in elderly people
- Especially those exposed to strong sunlight, dust, wind etc.
- Usually asymptomatic.
- Requires no treatment.



Pinguecula

EPISCLERITIS

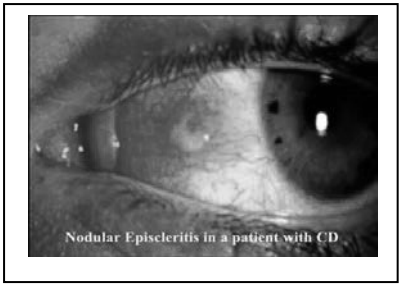
- Inflammation of subconjunctival connective tissues (that lies between conjunctiva and sclera)
- Usually a mild, self limiting, recurrent disease
- Most cases are idiopathic, although up to one third have an underlying systemic condition.

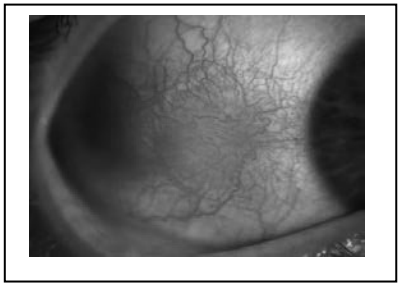
Etiology

- Etiology in most cases is not known
- Occurs in association with gout, psoriasis and as hypersensitivity reaction to an endogenous toxin.
- A history of rheumatoid arthritis is commonly obtained.
- Affects young adults, being twice as common in women than men.

Clinical Types

1. ***Simple / Diffuse :***
    1. Redness of eye mostly due to the engorgement of the large episcleral vessels which run in radial direction beneath the conjunctiva
2. ***Nodular :***
    1. A pink or purple flat nodule surrounded by congestion, usually situated 2-3 mm away from the limbus.
    2. It is hard, tender, immovable
    3. There may be little or no pain, but usually there is feeling of discomfort and tenderness on pressure





Symptoms:

- Pain, ocular discomfort
- No discharge, lacrimation or photophobia
- Nodular episcleritis tends to be more symptomatic and takes longer to resolve.
- Most common type is simple episcleritis
- Intermittent bouts of moderate to severe inflammation that often recur at 1-3 months intervals.
- The episodes usually last 7-10 days and most resolve after 2-3 weeks.
- Prolonged episodes may be more common in patients with associated systemic conditions.
- Patients with nodular episcleitis have prolonged attacks of inflammation that are typically more painful.

Treatment:

- If mild, the disease can be treated with lubricants alone
- If moderate to severe, a mild topical steroid (Fluorometholone) 4 times a day relieves the discomfort and inflammation
- In cases where topical treatment is unsuccessful, oral NSAIDs may help.
-

SCLERITIS

- Pathologically scleritis resembles episcleritis, but extends more deeply.
- Commonly associated with systemic autoimmune disorders
- Usually bilateral disease
- Occurs most frequently in women
- Rarer than episcleritis

Etiology:

- About 50% cases are associated with connective tissue disease
- A thorough investigation is required to eliminate active systemic disease such as polyarteritis nodosa, systemic

lupus erythematous, rheumatoid artheritis, Wegener granulomatosis, gout

- Other known association include acute or previous attack of herpes zoster ophthalmicus, syphilis and recent ocular surgery such as cataract extraction .

Classification

- Anterior Scleritis:
  - Nodular
  - Diffuse
  - Necrotizing
    - With inflammation
    - Without inflammation
- Posterior Scleritis
- Diffuse anterior scleritis
  - Widespread inflammation of the anterior portion of the sclera
  - Most common form of anterior scleritis
- Nodular anterior scleritis
  - One or more erythamatous, immovable, tender inflamed nodules on the anterior sclera.
- Necrotizing scleritis with inflammation:
  - Red, painful eye progressive worsening of symptoms
  - Usually a part of systemic autoimmune disease
- Necrotizing scleritis without inflammation:
  - Called scleromalacia perforans
  - Occurs in patients suffering from seropositive rheumatoid artheritis
- Posterior scleritis:
  - Frequently misdiagnosed
  - Usually not associated with any systemic disease
  - Clinical features include decreased vision, with or without pain, proptosis or restricted ocular movements.

Treatment:

- Diffuse or nodular scleritis:

  - Oral NSAID
  - Oral prednisolone
- Necrotizing scleritis:
  - Systemic steroids
  - Immunosuppressives
- Posterior scleritis
  - systemic NSAIDs
  - Steroids or immunosuppressive therapy

CONJUNCTIVAL XEROSIS

Dry, lustreless condition of the conjunctiva due to deficiency of mucin.

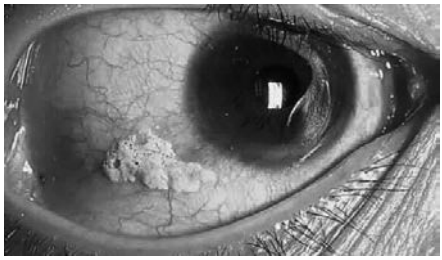
- Conjunctiva contains 2 types of glands:
  - Mucin secretory glands
  - Accessory lacrimal glands
- Goblet cells are unicellular mucous glands which secrete mucin.
- Mucin lubricates and protects the epithelial cells of conjunctiva and cornea
- Destruction of goblet cells, as in xerosis, leads to dessication of the conjunctiva.

Causes:

1. As a sequel of local ocular affection (Parenchymatous xerosis)
   - Cicatricial degeneration of the conjunctival epithelium and glands following trachoma, burns, pemphigoid etc
   - Following prolonged exposure due to ectropion or proptosis
2. Associated with systemic disease (Epithelial Xerosis)
   - Deficiency of Vitamin A in diet
   - Found particularly in children
   - Accompanied by night blindness

- Characterized by small triangular, white patches covered by a material resembling dried foam, which is not wet by tears. (Bitot's Spot)

- These foamy spots are due to gas production by *corynebacterium xerosis* present in epithelium.



Bitot's spot

Treatment:

- Purely symptomatic
- Locally liquid paraffin or methyl cellulose 1% for dryness
- In Vit. A deficiency, nutrition should be restored by administration of vitamin A.

SUBCONJUNCTIVAL HAEMORRHAGE

- Also called subconjunctival ecchymosis
- Due to rupture of small vessels

Causes:

- Direct trauma to eye
- Head injury or injury to the orbit causing fracture of a wall of orbit
- Large extravasations : severe straining, especially in old people, as on lifting heavy weights or vomiting, children with whooping cough, scurvy, blood diseases like purpura
- Arteriosclerosis, hypertension
- Very minute ecchymoses : in severe conjunctivitis

Treatment:

- The blood gradually changes colour and gets absorbed in 1-3 weeks without treatment
- Hot compress may help absorption faster.
- If mild ocular irritation is present, artificial tears can be prescribed 4 to 6 times a day.

Subconjunctival haemorrhage

CORNEA

- Transparent, avascular, watch-glass like structure
- It forms anterior one sixth of the outer fibrous coat of the eyeball.

- **Layers**:
  - **Epithelium** : Stratified squamous type. Corneal epithelium replaces itself about once a week.
  - **Bowman's membrane**: once destroyed does not regenerate.
  - **Stroma**: constitutes 90% of total thickness. Consists of collagen fibres.
  - **Descemet's membrane**: once destroyed, it regenerates. Its prominent peripheral end forms schwalbe's line.
  - **Endothelium**: cell density in a young adult is about 6000 cells/mm^2. Metabolically, it is the most active layer of cornea.
- **Blood supply**
  - Avascular
  - Small loops derived from the anterior ciliary vessels invade its periphery for about 1mm
  - These loops are not in the cornea but in the subconjunctival tissue which overlaps the cornea.
- **Nerve supply**:
  - Supplied by anterior ciliary nerves which are branches of ophthalmic division of the 5^{th} cranial nerve (trigeminal).

CORNEAL ULCER

- Discontinuation in normal epithelial surface of cornea associated with necrosis of the surrounding corneal tissue

Classification:

- Purulent ulcer/ Suppurative keratitis:
  - Ordinary pyogenic ulcer
  - Hypopyon ulcer
  - Mycotic ulcer
  - Marginal ulcer
- Non-purulent ulcer
  - Ulcer in association with trachoma
  - Dendritic ulcer
  - Lagophthalmic
  - Ulcer due to Vit. A deficiency
  - Neurotrophic ulcer
- Allergic ulcer : Phlyctenular ulcer
- Degenerative ulcer
  - Atheromatous ulcer
  - Mooren's ulcer

Suppurative Keratitis (purulent ulcer)

- It is due to organisms that produce toxins which cause tissue death (necrosis) and pus formation in the corneal tissue.
- Normal corneal epithelium cannot be penetrated by any organism except by *Corynebacterium diphtheriae*, *N. gonorrhoea* and *N. meningitidis* but most bacteria are capable of producing ulceration when the epithelium is damaged.

Etiology

- 2 main factors in the production of purulent corneal ulcer:
  - Damage to corneal epithelium
  - Infection of eroded area
- Corneal epithelial damage:

  - Corneal abrasion due to small foreign body, misdirected cilia, contact lens wearers
  - Epithelial drying as in xerosis and exposure keratitis
  - Necrosis of epithelium as in keratomalacia
- Sources of infection:
  - Exogenous infection:
    - Conjunctival sac, lacrimal sac, infected foreign bodies, infected vegetative material, airborne infection
  - From ocular tissue:
    - Owing to direct anatomical continuity, disease of the conjunctiva readily spread to corneal epithelium, those of sclera to stroma, and of the uveal tract to the endothelium of cornea.

- Causative organism: Common bacteria associated with corneal ulceration include
  - *Staphylococci*
  - *Pseudomonas*
  - *Enterobacteriaceae*
  - *Neisseria*

Pathogenesis

- Stage of progressive infiltration
- Stage of active ulceration
- Stage of regression
- Stage of cicatrization

- **Progressive stage:**
  - Marked infiltration of corneal tissue with polymorphoneuclear and/or lymphocytes into the epithelium from peripheral circulation surrounding the ulcer
  - Subsequently, necrosis of the involved tissue may occur, depending upon virulence of offending agent and the strength of host defence mechanism.

- **Active ulceration**:
    - Necrosis and sloughing of the epithelium, Bowman's membrane and involved stroma.
- **Stage of regression**:
    - Regression is induced by the natural host defence mechanisms
    - A line of demarcation develops around the ulcer, which consists of leucocytes that neutralize and eventually phagocytose the offending organisms.
    - The ulcer begins to heal and epithelium starts growing over the edges.
- **Stage of cicatrization:**
    - Healing continues by progressive epithelization which forms a permanent covering.
    - Beneath the epithelium, fibrous tissue is laid down partly by the corneal fibroblasts and partly by the endothelial cells of new vessels.
    - The newly laid fibrous tissue causes an opacity of the cornea

Symptoms

- Pain which may be due to exposure and irritation of sensory nerve endings
- Watering from eyes due to reflex hyperlacrimation
- Photophobia results from stimulation of nerve endings
- Blurred vision results from corneal haze.
- Redness of eyes occurs due to congestion of circumcorneal vessels.

Signs

- Blepharospasm
- Rough and raw yellowish white area on the cornea which stains with fluorescein
- Haziness of cornea surrounding the ulcerated area
- Ciliary congestion with conjunctival hyperemia
- Blood vessels may encroach the cornea from the limbus

Treatment

- Specific treatment
  - Topical antibiotics
  - Systemic antibiotics
- Non-specific treatment
  - Cycloplegic drugs
  - Systemic analgesics and aniinflammatory drugs
  - Vitamins
- Physical and general measures
  - Hot fomentation
  - Dark goggles
  - Rest, good diet, fresh air

CORNEAL OPACITY

Loss of normal transparency of cornea due to scarring

Causes

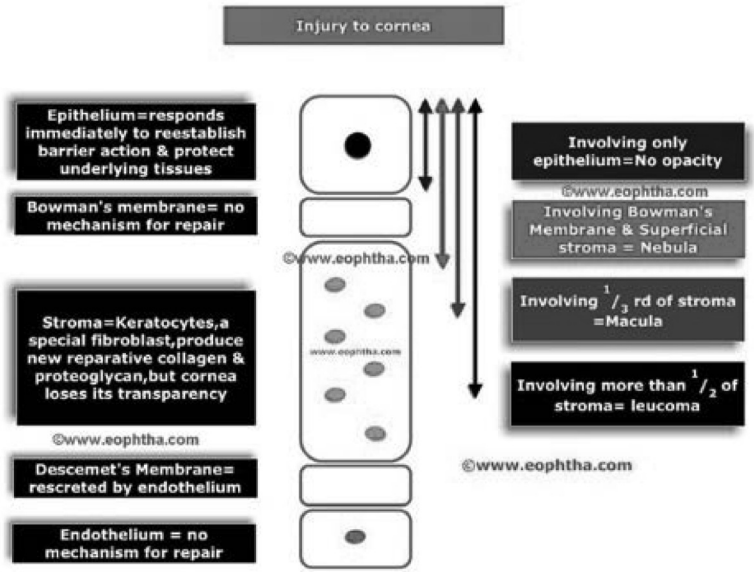
- Congenital opacities may occur as developmental anomalies or following birth trauma
- Healed corneal wounds
- Healed corneal ulcers

Types

1. Nebular
2. Mecular
3. Leucomatous
4. Adherent leucoma
5. Corneal facet
6. Kerectasia
7. Anterior staphyloma

- **Nebula**:
  - Opacity results due to superficial scars involving Bowman's layer and superficial stroma.
- **Macula**:
  - A semi-dense opacity produced when scarring involves about half the corneal stroma.

- **Leucoma**
  - Dense white opacity which results due to scarring of more than half of the stroma.
- **Adherent leucoma**
  - It results when healing occurs after perforation of cornea with incarceration of iris.



- **Corneal facet**
  - Sometimes, the corneal surface is depressed at the site of healing (due to less fibrous tissue), such a scar is called facet.
- **Kerectasia**:
  - Corneal curvature is increased at the site of opacity. (Bulge due to weak scar)
- **Anterior staphyloma**:
  - An ectasia of pseudocornea which results after total sloughing of cornea, with iris plastered behind it is called anterior staphyloma.

Treatment

1. Optical iridectomy
   - In cases with central macular or leucomatous corneal opacities, provided vision improves with pupillary dilatation
2. Phototherapeutic keratectomy (PTK)
3. Keratoplasty
4. Cosmetic coloured contact lens gives good cosmetic appearance in an eye with ugly scar
5. Tattooing of scar

HYPOPYON CORNEL ULCER

- Characteristic ulcer caused by *Pneumococcus*
- also called ulcus serpens
- *Source of infection* for pneumococcal infection is usually the chronic dacryocystitis.

Etiology:

- 2 main predisposing factors:
  - Virulence of infective organism
  - The resistance of the tissue
- Hence hypopyon ulcers are much more common in old debilitated or alcoholic subjects.

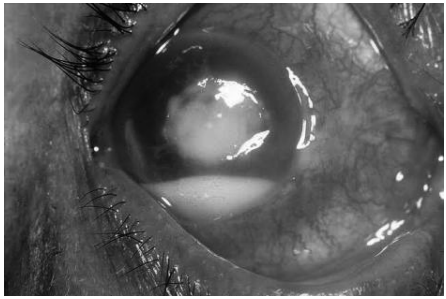
Pathology:

- When the iritis is severe, the outpouring of leucocytes from vessels is so great that these cells gravitate to the bottom of anterior chamber to form a hypopyon
- Once the ulcerative process is controlled, the hypopyon is absorbed.

Signs:

- Greyish white or yellowish disc shaped ulcer near the centre of cornea
- Violent iridocyclitis

- Hypopyon increase in size rapidly and often results in secondary glaucoma

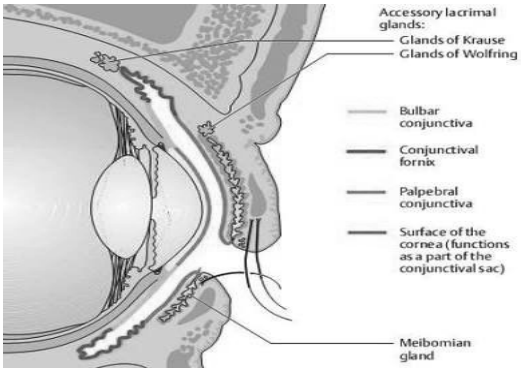


Management:

- Same as for other bacterial corneal ulcer

CONJUNCTIVA

- Translucent mucous membrane which lines the posterior surface of the eyelids and anterior aspect of the eyeball
- The name is given owing to the fact that it joins the eyeball to the lids (conjoin=to join)



1. Palpebral conjunctiva:

- Lines the lids
- Can be subdivided into 3 parts
  - Marginal: extends from lid margin to about 2 mm on the back of lid
  - Tarsal: thin, transparent, highly vascular; firmly adherent to the whole tarsal plate in upper lid;in lower lid, it is adherent only to half width of the tarsus.
  - Orbital: lies loose between the tarsal plate and fornix

2. Bulbar conjunctiva

- Thin, transparent, lies loose over the underlying structures and thus can be moved easily.

3. Conjunctival fornix

- A continuous circular cul-de-sac which is broken only on the medial side by caruncle and plica semilunaris.
- Fornix joins the bulbar conjunctiva with the palpebral conjunctiva.

Structure:

- Consists of 3 layers
  - Epithelium
  - Adenoid layer
  - Fibrous layer

CONJUNCTIVITIS

Conjunctival hyperemia associated with a discharge which may be watery, mucoid, mucopurulent or purulent.

Types:

- Infective
- Allergic
- Cicatricial
- Toxic

Infective:

- Bacterial
- Viral
- Chlamydial
- Ophthalmia neonatorum

Bacterial:

- Commonest type
- Etiology
  - Predisposing factors
    - Flies, poor hygienic conditions, hot dry climate, poor sanitation and dirty habits
  - Causative organism
    - Staphylococcus aureus, staphylococcus epidermidis, streptococcus pyogenes, haemophilus influenzae, Moraxella lacunata
- Mode of infection:
  - Exogenous:
    - Directly through close contact - airborne, waterborne
    - Vector transmission: e.g.flies

    - Material transfer: infected fingers of doctors, nurses, towels, infected tonometers
  - Local spread from neighbouring structures such as infected lacrimal sac, lids, nasopharynx
  - Endogenous: rarely through blood e.g. gonococcal and meningococcal infections
- Clinical types
  - Acute
  - Hyperacute
  - Chronic
  - angular

Acute bacterial conjunctivitis

- Most common type
- Characterized by marked conjunctival hyperemia and mucopurulent discharge
- So, clinically it is called acute mucopurulent conjunctivitis
- Common causative bacteria: Staphylococcus aureus, pneumococcus, streptococcus

Clinical features:

- Discomfort, F.B. sensation, grittiness, blurring and redness of sudden onset
- Mild photophobia
- Mucopurulent discharge
- Sticking together of lid margins with discharge during sleep
- Slight blurring of vision due to mucous flakes in front of cornea
- Coloured halos due to prismatic effect of mucus present on cornea

Signs:

- Flakes of mucopus in the fornices, canthi, lid margins
- Conjunctival congestion
  - Giving the appearance of fiery red eye.

  - Less marked in circumcorneal zone
- Chemosis: swelling of conjunctiva
- Cilia are usually matted together with yellow crusts

Clinical course:

- Usually bilateral, although one eye may become affected 1-2 days before the other.
- Usually reaches its height in 3 to 4 days
- If untreated, in mild cases the infection may be overcome and condition is cured in 10-15 days

Treatment:

- Topical antibiotics:
  - Chloramphenicol(1%) or gentamicin (0.3%) or tobramycin 0.3% or framycetin 0.3% eye drops 3-4 hourly in day
  - If patient does not respond, then quinolone anibiotic drops – ciprofloxacin(0.3%), Ofloxacin(0.3%), gatifloxacin(0.3%)
- Irrigation of conjunctival sac: with sterile warm saline once or twice a day will help removing deleterious material.
  - Frequent eyewash is contraindicated as it will wash away the lysozyme and other protective proteins present in the tears
- Dark goggles to prevent photophobia
- Anti inflammatory and analgesics to provide symptomatic relief
- No bandage, No steroids

<u>Hyperacute conjunctivitis</u>

- Affects adults, predominantly males
- Gonococcal infection spreads from genitals to eye.
- Usually associated with urethritis and arthritis
- Symptoms:
  - Pain: moderate to severe
  - Purulent discharge: usually copious
  - Swelling of eyelids

- Treatment:
  - Systemic therapy: third generation cephalosporin
  - Topical antibiotic therapy
  - Irrigation

Chronic bacterial conjunctivitis

- Also known as chronic catarrhal or simple chronic conjunctivitis
- Characterized by mild catarrhal inflammation of conjunctiva
- Etiology:
  - Predisposing factors:
    - Chronic exposure to dust, smoke, chemical irritants
    - Local cause of irritation : trichiasis, F.B.
    - Eye strain due to refractive errors
    - insomnia
  - Causative organism: Staphylococcus aureus is the commonest cause
  - Source and mode of infection:
    - Continuation of acute bac. Conjunctivitis
    - Chronic infection from ass. Chronic dacryocystitis, chronic rhinitis
    - Mild exogenous infection: direct contact, material transfer etc

- Symptoms:
  - Burning, grittiness in the eyes
  - Difficulty in keeping the eyes open
  - Mild mucoid discharge
  - Watering off and on
  - Feeling of sleepiness and tiredness in eyes
- Treatment:
  - Eliminate predisposing factors
  - Topical antibiotics: chloramphenicol, tobramycin or gentamicin should be instilled 3-4 times a day for about 2 weeks
  - Astringent eye drops such as zinc-boric acid drops

Angular bacterial conjunctivitis

- A type of chronic conjunctivitis
- Characterized by mild grade inflammation confined to the conjunctiva and lid margins near the angles
- Etiology:
  - Causative organism: Moraxella Axenfield (MA)
  - Source of infection: Nasal cavity
  - Mode of infection: infection is transmitted from nasal cavity to the eyes by contaminated fingers or handkerchief.
- Symptoms:
  - Irritation, burning sensation, feeling of discomfort in the eyes
  - Redness in the angles of eyes
- Signs:
  - Hyperemia of bulbar conjunctiva near canthi
  - Foamy mucopurulent discharge at the angles
  - Excoriation of skin around the angles
- Treatment:
  - Prophylaxis: treatment of associated nasal infection and good personal hygiene
  - Curative:
    - Oxytetracycline (1%) eye ointment, 2-3 times a day for 9 tp 14 days

Viral conjunctivitis

- Most of the viral infections tend to affect the epithelium, both of the conjunctiva and cornea; so the typical viral lesion is a 'keratoconjunctivitis'.
- Viral infections of conjunctiva include:
  - Adenovirus
  - Herpes simplex
  - Herpes zoster
  - Molluscum contagiosum
  - Poxvirus
  - Myxovirus
  - Paramyxovirus

Table 1. How to Differentiate Bacterial from Viral Conjunctivitis

Bacterial	Viral
Mucopurulent discharge	Watery discharge
Bilateral	Unilateral
Preschoolers (3.6 yrs)	Older children (7.5 yrs)
Otitis media	Pharyngitis
No adenopathy	Adenopathy
Both can be highly contagious!	

Sources: Bodor FF, et al. *Pediatrics*. 1985;76:26-28; Tarabishy AB, et al. *Cleve Clin J Med*. 2008;75:507-512.

Allergic conjunctivitis

Inflammation of conjunctiva due to allergic or hypersensitivity reactions

Types:

- Simple allergic conjunctivitis
  - Seasonal
  - Perennial
- Vernal keratoconjunctivitis (VKC)
- Atopic keratoconjunctivitis (AKC)
- Giant papillary conjunctivitis (GPC)
- Phlyctenular keratoconjunctivitis (PKC)
- Dermatoconjunctivitis

Simple allergic conjunctivitis

- Symptoms:
  - Intense itching and burning sensation in the eyes
  - Watery mucus, stringy discharge
  - Mild photophobia
- Signs:

  - Hyperemia, chemosis
  - Mild papillary reaction may be seen on palpebral conjunctiva
- Treatment:
  - Elimination of allergens
  - Topical vasoconstrictors like naphazoline
  - Artificial tears like CMC provide soothing effect
  - Mast cell stabilizers such as sodium cromoglycate are effective in preventing the recurrences
  - Systemic antihistaminic drugs are useful in acute cases with marked itching

Table 1. Bacterial vs. Viral vs. Allergic Conjunctivitis

Clinical Finding	Bacterial	Viral	Allergic
Bilateral eyes	50% to 74%	35%	Mostly
Discharge	Mucopurulent in younger children	Mild, watery, or "sleepers" only	Rare
Redness	Common in older children, uncommon in infants and toddlers	Usually	Usually
Acute otitis media	32% to 39%	10%	No
Pruritic	No (but many rub eyes)	No	Major

Sources: Block SL, et al. *Antimicrob Agents Chemother.* 2000;44:1650-1654; Bodor FF, et al. *Pediatrics.* 1985;76:26-28; Gigliotti F, et al. *J Pediatr.* 1981;98:531-536; Tarabishy AB, et al. *Cleve Clin J Med.* 2008;75:507-512.

Vernal keratoconjunctivitis/spring catarrh:

- Recurrent, bilateral, intertitial, self-limiting, allergic inflammation of conjunctiva
- More common in summer
- Etiology:
  - Type-I IgE mediated hypersensitivity reaction to pollen allergens.
- Clinical features:
  - Marked burning and itching sensation usually intolerable and accentuated when patient comes in a warm humid atmosphere

  - Mild photophobia, lacrimation, ropy discharge, heaviness of lids
- Treatment
  - Topical anti-inflammatory therapy with combined steroids, mast cell stabilizers, antihistamines, NSAIDs
  - Topical lubricating and mucolytics
  - Systemic therapy
    - Oral antihistaminics
    - Oral steroids
  - General measures:
    - Dark goggles, cold compress

Atopic conjunctivitis

- An adult equivalent of VKC and is often associated with atopic dermatitis
- Pathogenesis:
  - Both IgE and cell-mediated immune mechanism play role i.e. type –I and type IV hypersensitivity reactions are responsible
- Symptoms:
  - Itching, soreness, dry sensation
  - Mucoid discharge
  - Photophobia
- Treatment
  - Same as that of VKC
  - Less responsive and requires more intensive and prolonged therapy

Giant Papillary Conjunctivitis:

- Inflammation of conjunctiva with formation of very large sized papillae
- Also known as mechanically induced papillary conjunctivitis
- Localized allergic response to a physically rough or deposited surface (Contact lens, prosthesis, exposed nylon sutures etc)

Phlyctenular conjunctivitis :

- Characteristic nodular affection occuring as an allergic response of the conjunctival and corneal epithelium to some endogenous allergens to which they have become sensitized.

GLAUCOMA

- Not a single disease but group of disorders characterized by a progressive optic neuropathy resulting in a
  - characteristic appearance of the optic disc and
  - a specific pattern of irreversible visual field defects **that are associated frequently but not invariably with raised IOP.**
- Thus IOP is the most common risk factor but not the only risk factor for the development of Glaucoma
- *[Cases having constantly raised IOP without any associated glaucomatous damage :* ***Ocular hypertension***
- *Typical cupping of the disc and/or visual field defects associated with a normal or low IOP :* ***Normal or Low tension Glaucoma (NTG/LTG)]***

Classification:

- Congenital:
  - Primary Congenital Glaucoma (Without associated anomalies)
  - Developmental Glaucoma (With associated anomalies)
- Primary Adult Glaucomas
  - Primary Open-angle Glaucomas (POAG)
  - Primary Angle-closure Glaucomas (PACG)
  - Primary Mixed mechanism Glaucoma
- Secondary Glaucomas

Pathogenesis

- All glaucomas are characterized by a progressive optic neuropathy.
- Progressive optic neuropathy results from the death of retinal ganglion cells (RGCs) in a typical pattern which results in characteristic optic disc appearance and specific visual field defects.

Pathogenesis of RGC death

- Some pathologic events blocks the transport of growth factors (neurotrophins) from the brain to the RGCs.
- The blockage initiate a damaging cascade, and the cell is unable to maintain its normal function.
- The RGCs undergo apoptosis and also trigger apoptosis of adjacent cells
- RGC death is associated with loss of retinal nerve fibres.
- As the loss of nerve fibres extends beyond the normal physiological overlap of functional zones, the characteristic optic disc changes and specific visual field defects become apparent over the time.

Etiological factors:

- Primary insults
  - Raised IOP
  - Pressure independent factors (Vascular insufficiency theory)
    - Failure of autoregulatory mechanism of blood flow
    - Vasospasm
    - Systemic hypotension
    - Other factors: acute blood loss, abnormal coagubility profile
- Secondary insults (Excitotoxicity theory)

Congenital Glaucoma

- A group of diverse disorders in which abnormal high IOP results due to developmental abnormalities of the angle of anterior chamber obstructing the drainage of aqueous humour
- Sometimes, glaucoma may not occur until several years after birth, therefore, the term developmental glaucoma is preferred.

Primary congenital

- Abnormally high IOP which results due to developmental anomaly of the angle of the anterior chamber, not associated with any other ocular or systemic anomaly.
- **Newborn glaucoma**: true congenital glaucoma, IOP is raised during intrauterine life and child is born with ocular enlargement.
- **Infantile glaucoma**: menifests prior to the child's 3rd birthday
- **Juvenile glaucoma**: develop pressure rise after 3 years but before adulthood.
- **Buphthalmos**: when the disease menifests prior to age of 3 years, the eyeball enlarges and so the term Buphthalmos (Bull like eyes) is used. As it results due to retention of aqueous humour (watery solution), the term **hydrophthalmos** has also been suggested.

Primary Open-Angle Glaucoma

- A type of primary glaucoma where there is **no obvious systemic or ocular cause of rise in the IOP.**
- Also known as ***Chronic simple glaucoma*** of adult onset
- Characterized by:
  - Slowly progressive raised IOP
  - Open normal appearing anterior chamber angle
  - Characteristic optic disc cupping
  - Specific visual field defects

Etiopathogenesis:

- Not known exactly.
- Predisposing and risk factors:
  - IOP : most common risk factor
  - Age : the risk increases with increasing age
  - Myopes are more predisposed
  - Diabetics have higher prevalence
  - Cigarette smoking
  - Hypertension

Clinical features:

- **Asymptomatic** until it has caused a significant loss of visual field.
- **Headache** and **eye ache** of mild intensity may be experienced
- **Difficulty in reading and close work**, often persistently increasing. This occurs due to increasing accomodative failure as a result of constant pressure on the ciliary muscle and its nerve supply. Therefore patient usually complain of frequent changes in presbyopic glasses
- **Significant loss of vision and blindness** is the end result of untreated cases of POAG.

Investigations:

- Tonometry
- Gonioscopy
- Slit lamp examination
- Perimetry

Management:

- Medical
- Argon or Diode Laser Trabeculoplasty
- Filteration surgery

Medical

- Single drug therapy
  - Prostaglandin analogues
  - Topical beta-blockers
  - Adrenergic drugs
  - Dorzolamide
  - Pilocarpine
- Oral carbonic anhydrase inhibitors
  - Acetazolamide
- Hyperosmotic agents
  - mannitol

Primary Angle-Closure Glaucoma

- Apposition of peripheral iris against the trabecular meshwork resulting in obstruction of aqueous outflow by closure of an already narrow angle of the anterior chamber.
- This condition is not associated with any other ocular or systemic abnormalities.

Predisposing factors

- A short eye
- Smaller corneal diameter
- A shallow anterior chamber
- A relative anterior positioning of the lens-iris diaphragm

Classification:

Angle Closure Staging Classification

DISEASE STAGING	≥180° ITC	⊠ IOP and/or PAS	GLAUCOMATOUS OPTIC NEUROPATHY	
PACS Primary Angle Closure Suspect	+	−	−	Trabecular meshwork at risk
PAC Primary Angle Closure	+	+	−	Trabecular meshwork dysfunction
PACG Primary Angle Closure Glaucoma	+	+	+	Optic nerve damage

ITC – Iridotrabecular contact; PAS – Peripheral anterior synechiae (Figure courtesy of H. George Tanaka, MD)

Primary Angle-closure Suspect:

- These eyes are identified by a shallow anterior chamber associated with an occludable angle

Subacute/intermittent PACG

- Eyes with a shallow anterior chamber and an occludable angle in which physiological factors such as reading in dim illumination, watching a film in a darkened cinema hall have precipitated a pupillary block.
- This causes sharp rise in IOP for a short period of time – a minute to a couple of hours – followed by a spontaneous resolution of the pupillary block, possibly due to

physiological miosis which may occur in sleep or otherwise.

Acute/congestive PACG

- Sudden occlusion of the entire angle with a resultant acute rise of IOP to extremely high levels

Chronic PACG

- The IOP is chronically raised in eyes having synechial closure over at least 180°.

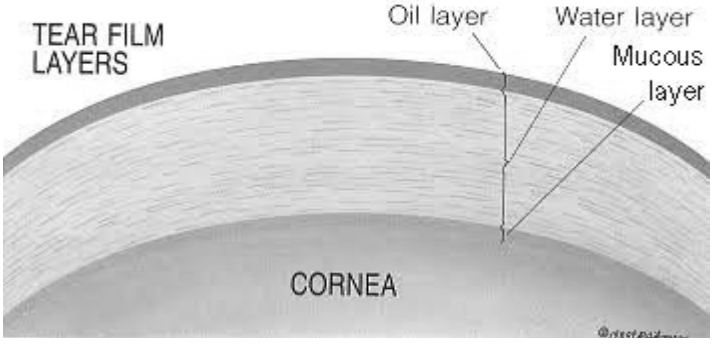
Secondary Glaucoma:

- A group of disorders in which rise of IOP is associated with some primary ocular or systemic disease

Tear film

Layers:

1. Mucus layer:
   - Innermost
   - Consists of mucin secreted by conjunctival goblet cells and gland of Manz
2. Aqueous layer:
   - Intermediate layer consists of tears secreted by the main and accessory lacrimal glands.
3. Lipid or Oily layer:
   - Outermost, thinnest layer formed at air-tear interface from the secretions of meibomian, zeis and moll glands
   - This layer prevents the overflow of tears, retards their evaporation and lubricates the eyelids as they slide over the surface of the globe.



Functions of tear film:

- Keeps moist the cornea and conjunctiva
- Provides oxygen to corneal epithelium
- Washes away debris and noxious irritants
- Prevents infection due to presence of anti-bacterial substances
- Facilitates movements of the lids over the globe

THE DRY EYE

- A symptom complex occurring as a sequelae to deficiency or abnormality of tear film.

Etiology:

1. ***Aqueous deficiency dry eye:***

Also known as Keratoconjunctivitis sicca (KCS)
Its causes include:

- Sjogren's syndrome
- Non-sjogren's KCS
  - Primary age related hyposecretion
  - Lacrimal gland deficiencies
  - Lacrimal gland obstruction
  - Reflex hyposecretion

2. ***Evaporative dry eye:***

- It is caused by conditions which decrease tear film stability and thus increase evaporation
- Causes
  - Meibomian gland dysfunction
  - Lagphthalmos
  - Defective blinking
  - Vit. A deficiency

Symptoms:

- Irritation
- Foreign body sensation
- Feeling of dryness
- Itching
- Nonspecific ocular discomfort

Tear film tests:

- Schirmer I - with anesthetic. Evaluates baseline secretion.
- Schirmer – II – without anesthetic. Measures baseline plus reflex secretion.

- Schirmer-I test: measures total tear secretions.
  - Filter paper strip (5 mm wide, 35 mm long) kept in the lower fornix
  - The patient is asked to look up and not to blink the eyes.
  - After 5 minutes, wetting of filter paper strip is measured.
    - More than 15 mm – normal
    - 5-10 mm – mild to moderate KCS
    - Less than 5 mm – severe KCS

Treatment:

- At present, there is no cure for dry eye.

1. Supplementation with tear substitutes: artificial tears
2. Topical cyclosporins: helps by reducing cell-mediated inflammation of lacrimal tissue
3. Mucolytics: 5% acetylcystine helps by dispersing the mucus threads and decreasing tear viscosity.
4. Preservation of existing tears by reducing evaporation and decreasing drainage: evaporation can be decreased by decreasing room temperature,
5. Treatment of the causative disease
    1. Vit A supplement
    2. Treat the cause of lagophthalmos

ERRORS OF REFRACTION

EMMETROPIA (Optically normal eye):

- A state of refraction, where in the parallel rays of light coming from infinity are focused at the sensitive layer of retina with the accommodation at rest.

AMETROPIA (A condition of refractive error)

- A state of refraction, when the parallel rays of light coming from infinity (with accommodation at rest) are focused either in front or behind the sensitive layer of retina, in one or both the meridians.
  I. Myopia
  II. Hypermetropia
  III. Astigmatism

	Hypermetropia (long sightedness)	**Myopia (short sightedness)**
	Parellel light rays coming from infinity are focused behind the retina with accommodation at rest.	Parellel light rays coming from infinity are focused in front of the retina with accommodation at rest.
Etiology		
Axial	• Axial shortening of the eyeball • About 1 mm shortening of AP diameter of the eye results in 3 dioptres of hypermetropia.	• Increase in AP length of eyeball.
Curvatural	• The curvature of cornea, lens or both is flatter than the normal resulting in a decrease in the refractive power of	• Increased curvature of cornea, lens or both.

	Hypermetropia (long sightedness)	Myopia (short sightedness)
	eye. • About 1 mm increase in radius of curvature result in 6 dioptres of HM.	
Index	• Decrease in RI of lens in old age. • May also occur in diabetics under treatment.	• Increase in RI of crystalline lens associated with nuclear sclerosis.
Positional	• Posteriorly placed crystalline lens.	• Anterior placement of crystalline lens.
	• **Absence of crystalline lens** congenitally / acquired leads to aphakia – a condition of high HM.	• Myopia due to **excessive accommodation** occurs in patients with spasm of accommodation

ASTIGMATISM

- A type of refractive error wherein the refraction varies in the different meridian of the eye.
- Consequently, the rays of light entering in the eye cannot converge to a point focus but form focal lines.

REGULAR ASTIGMATISM: When the refractive power changes uniformly from one meridian to another.

TYPES OF REGULAR ASTIGMATISM:

1. **With the rule Astigmatism**: the two principal meridia are placed at right angles to one another but the vertical meridian is more curved than the horizontal.

Thus, correction of this astigmatism will require the concave cylinder at 180 +/- 20 or convex cylinder lens at 90 +/- 20.

2. **Against the rule Astigmatism:** the horizontal meridian is more curved than the vertical meridian.

   The correction will require convex cylindrical lens at 180+/-20 or concave cylindrical lens at 90+/- 20 axis.
3. **Oblique astigmatism:** the two principal meridia are not the horizontal and vertical though these are at right angles to one another.
4. **Bioblique astigmatism**: the two principal meridian are not at right angle to each other e.g. one may be at 30 and other at 100.

REFRACTIVE TYPES OF REGULAR ASTIGMATISM:
depending upon the position of the two focal lines in relation to retina

1. **Simple astigmatism:** rays are focused on the retina in one meridian, and either in front (simple myopic astigmatism) or behind (simple hypermetropic astigmatism) the retina in the other meridian.
2. **Compound astigmatism:** rays in both the meridia are focused either in front (compound myopic) or behind (compound hypermetropic) the retina.
3. **Mixed astigmatism:** light rays in one meridian are focused in front and in other meridian behind the retina. Thus in one meridian, the eye is myopic and in another hypermetropic. Such patients have comparatively less symptoms as circle of least diffusion is formed on the retina.

Etiology:

1. **Corneal** astigmatism: is the result of abnormality of curvature of cornea. Most common cause of astigmatism.
2. **Lenticular** astigmatism: rare. It may be
   I. Curvatural due to abnormalities of curvature of lens as seen in lenticonus.

II. Positional due to tilting or oblique placement of lens as seen in subluxation.
III. Index astigmatism may occur rarely due to variable RI of lens in different meridian.

3. **Retinal** astigmatism: due to oblique placement of macula. Seen occasionally.

CATARACT

- Development of any opacity in the lens or its capsule.
- Cataract, thus may occur
  - Either due to formation of opaque lens fibres **(Congenital or developmental)**
  - Or due to degenerative process leading to opacification of the normally formed transparent lens fibres **(Acquired cataract)**.

Congenital and developmental cataract:

- Occur due to some disturbance in the normal growth of lens.
- When the disturbance occurs before birth, the child is born with a congenital cataract.
- Developmental cataract may occur from infancy to adolescence.

Etiology

1. Idiopathic: about 33% cases are sporadic and of unknown etiology.
2. Heredity: about one third of all congenital cataracts are hereditary.
3. Maternal factors:
    1. Malnutrition during pregnancy
    2. Infections: infections like Rubella are associated with cataract in 50% cases
    3. Drugs ingestion: certain drugs during pregnancy (thalidomide, corticosteroids)
    4. Radiation: exposure to radiation during pregnancy

4. Foetal/infantile factors:
    - Anoxia owing to placental hemorrhage
    - Birth trauma
    - Metabolic disorders: galactosemia, galactokinase deficiency
    - Cataract associated with other congenital anomalies: Lowe's syndrome, myotonia dystrophica
    - Ocular disease associated with developmental cataract
    - Malnutrition

Clinical types:

- Cogenital capsular cataracts
    - Anterior capsular
    - Posterior capsular
- Polar cataracts
    - Anterior polar
    - Posterior polar
- Congenital nuclear cataracts
    - Cataracta pulverulenta
    - Lamellar cataract
    - Sutural and axial cataracts
- Generalized cataracts
    - Coronary cataract
    - Blue dot cataract
    - Total congenital cataract
    - Congenital membranous cataract.

<u>Acquired cataract</u>

- Degeneration of the already formed normal fibres.
    - Age related cataract
    - Traumatic cataract
    - Metabolic cataract
    - Complicated cataract
    - Radiational cataract
    - Electric cataract

Age related cataract:

- Commonest type
- Usually bilateral, but almost always one eye is affected earlier than the other.
- Morphologically, it occurs in two forms:
  - The cortical (soft cataract)
  - The nuclear (hard cataract)

Etiology:

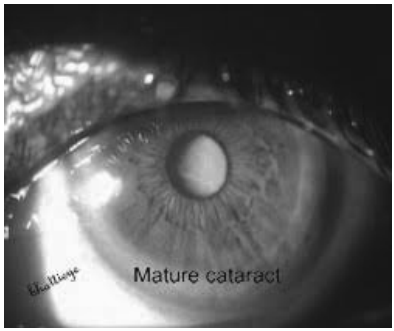
- Senile cataract is essentially an ageing process.
- Its precise etiopathogenesis is not clear.

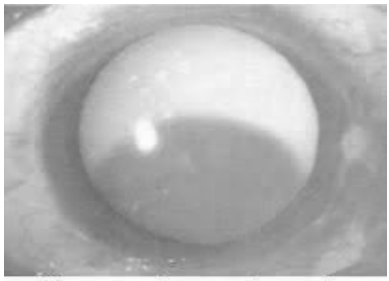
Risk factor:

- **Age**: it usually occurs after the age of 50 years. By the age of 70 years, over 90% of the individuals develop senile cataract.
- **Heredity**
- **UV irradiation**: more exposure to UV irradiation from sunlight
- **Dietary factors**: diet deficient in certain proteins, amino acids, vitamins
- **Dehydrational crisis**: prior episode of severe dehydrational crisis (Due to diarrhoea, cholera etc)
- **Smoking**

Maturation of the cortical type of senile cataract

- Stage of lamellar separation
- Stage of incipient cataract
  - Cuneiform senile cortical cataract
  - Cupuliform senile cortical cataract
- Immature senile cataract
- Mature senile cataract
- Hypermature senile cataract
  - Morgagnian
  - Sclerotic type





Hypermature cataract-
Morgagnian

Maturation of nuclear type of senile cataract

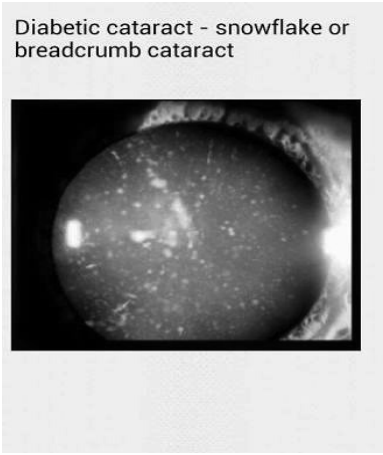
- Progressive nuclear sclerotic process renders the lens inelastic and hard, decreases its ability to accommodate and obstructs the light rays.
- These changes begin centrally and spread slowly peripherally almost up to capsule when it becomes mature.
- The nucleus may become greyish. (Yellow to black).

Clinical features of cataract:

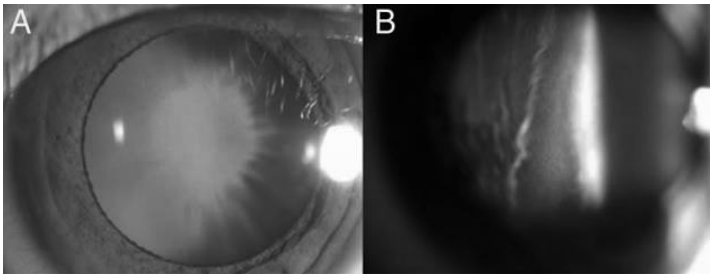
- Opacity may be present without any symptom.
- Glare: intolerance to bright light such as direct sunlight
- Uniocular polyopia: doubling of object. Occurs due to irregular refraction by the lens
- Coloured halos: due to presence of water droplets in the lens
- Deterioration of vision:
    - Central opacity(Cupuliform i.e. PSC cataract): have early loss of vision. These patients see better when pupil is dilated due to dim light in the evening
    - Peripheral opacities (Cuneiform): visual loss is delayed and vision improves in bright light when pupil is contracted.
    - Nuclear sclerosis: distant vision deteriorates due to progressive index myopia.
- As opacification progresses, vision steadily diminishes, until only perception of light.

Metabolic cataracts: due to endocrine disorders and biochemical abnormalities.

1. Diabetic cataract
    a. Senile: appears at an early age and progresses rapidly.
    b. True diabetic: snowflake cataract or snowstorm cataract. Rare, occurring in young adults due to osmotic overhydration of the lens which occurs due to accumulation of sorbitol.



2. Galactosaemic cataract: inborn error of galactose metabolism. Bilateral. Oil droplet central lens opacities. The lens changes may be reversible and cataract may be prevented if milk and milk products are eliminated from diet.
3. Hypocalcaemic cataract: associated with parathyroid tetany, which may occur due to atrophy or inadvertent removal of parathyroid glands during thyroidectomy.
4. Cataract due to error of copper metabolism: Wilson's disease (Hepatolenticular degeneration).
    a. Sunflower cataract
    b. Kayser-Fleischer ring (KE ring): a golden ring due to deposition of copper in the peripheral part of Descemet's membrane of cornea. Commonly found in Wilson's disease.



5. Cataract in Lowe's syndrome: Lowe's (oculo-cerebral-renal) syndrome is rare inborn error of amino acid metabolism. Congenital cataract, glaucoma, blue sclera.

Complicated cataract: Opacification of lens secondary to some other intraocular disease. The lens depends for its nutrition on intraocular fluids.

Therefore, any condition in which the ocular circulation is disturbed or in which inflammatory toxins are formed, will disturb nutrition of the crystalline lens, resulting in development of complicated cataract.

1. Inflammatory conditions: iridocyclitis, choroiditis, pars planitis, endophthalmitis, hypopyon corneal ulcer. Anterior uveitis is the most common cause.
2. Degenerative conditions: RP, myopic chorioretinal degeneration
3. Retinal detachment: long standing cases
4. Glaucoma
5. Intraocular tumours

Clinical features:

- starts as Posterior Subcapsular Cortical cataract (PSC)
- opacity – irregular in outline
- breadcrumb appearance
- polychromatic luster (Rainbow cataract)
- deposition of calcium is common in later stage.

Drug induced Cataract:

1. Corticosteroid induced : PSC opacities

2. Miotics induced cataract: anterior subcapsular granular type
3. Other drug induced: amiodarone, chlorpromazine, busulphan, gold, allopurinol.

Radiational cataract: exposure to all types of radiant energy is known to produce cataract by causing damage to the lens epithelium.

1. Infrared (Heat) cataract: glass-blower's or glass-worker's cataract. Because typically seen in persons working in glass industries. May cause discoid PSC.
2. Irradiation cataract: exposure to X-rays, ɣ-rays or neutrons
3. UV radiation cataract.

Electric cataract:

- After passage of powerful electric current through the body.
- Starts as punctate subcapsular opacities

Syndermatotic Cataract: lens opacities associated with cutaneous disease .

1. Atopic dermatitis: most common.
2. Other skin disorders: poikiloderma, scleroderma etc.

Traumatic cataract: Concussion cataract

- Occurs mainly due to inbibition of aqueous and partly due to direct mechanical effects of injury on lens fibres.
  Shapes:
  - Discrete subepithelial opacities: most common
  - Ealy rosette cataract (Punctate) appears as feathery lines of opacities along the star shaped suture lines. Usually in posterior cortex.
  - Late rosette cataract: develops in posterior cortex 1-2 years after injury.

Complications of cataract:

1. Phacoanaphylactic uveitis:

- Lens protein may leak into the anterior chamber in hypermature cataract.
- These protein may act as antigen and induce antigen-antibody reaction leading to phacoanaphylactic uveitis.

2. Lens induced glaucoma
    - Phacomorphic glaucoma: most common type. Secondary angle closure glaucoma.
    - Phacolytic glaucoma: secondary open angle glaucoma.
        i. Lens proteins are leaked into the anterior chamber in Morgagnian hypermature cataract.
        ii. These proteins are engulfed by macrophages. The swollen macrophages clog the TM leading to raised IOP.
    - Phacotopic glaucoma: hypermature cataractous lens may subluxate/dislocate and cause glaucoma by blocking the pupil or angle of ant. Chamber.
3. Subluxation or dislocation of lens: occur due to degeneration of zonules in hypermature stage.

Management of cataract:

❖ **Non-surgical measures:**

1. Treatment of cause
    - Control of DM
    - Removal of drugs
    - Removal of irradiation
    - Early treatment of diseases like uveitis

2. Measures to delay progression
    - Topical preparations containing iodide salts of calcium and potassium in early stages
    - Vitamin E and aspirin
3. Measures to improve vision in the presence of incipient and immature cataract
    - Prescription of glasses

- In peripheral opacities: Arrangement of illumination
- In central opacities: a dull light placed beside and slightly behind the head; Use of dark goggles
- Mydriatics: Patients with a small axial cataract, frequently may benefit from pupillary dilatation. This allows the clear paraxial lens to participate in light transmission, image formation and focusing. 5% phenylephrine/1% tropicamide 1 drop bid in the affected eye may clarify the vision.

❖ **Surgical management**:
- ICCE (Intracapsular Cataract Extraction)
- ECCE (Extracapsular Cataract Extraction)
  - ECCE
  - SICS (Small Incision Cataract Surgery)
  - Phacoemulsification

- ICCE: the entire cataractous lens along with the intact capsule is removed
- ECCE: major portion of anterior capsule with epithelium, nucleus and cortex are removed leaving behind the intact posterior capsule.

ECCE: Surgical steps

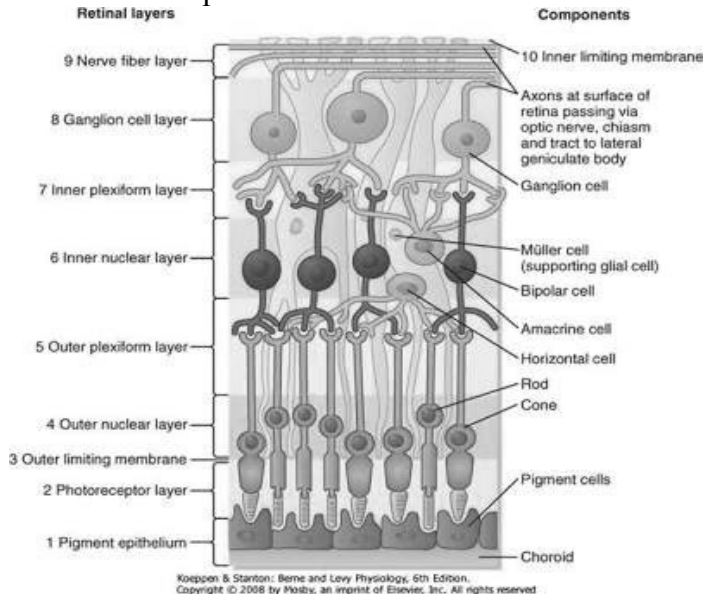
1. Superior rectal suture
2. Conjunctival flap
3. Partial thickness groove/gutter: made through about two-thirds depth of anterior limbal area from 10 to 2 o'clock (120) with the help of a razor blade knife.
4. Entry into anterior chamber: with the razor blade knife or with 3.2mm keratome.
5. Injection of viscoelastic substance in anterior chamber: 2% methylcellulose or 1% sodium hyaluronate. This

maintains the anterior chamber and protects the endothelium.

6. Anterior capsulotomy
    a. Can-opener's technique
    b. Linear capsulotomy
    c. Continuous circular capsulorrhexis (CCC)
7. Removal of anterior capsule: with the help of a Kelman-McPherson forceps.
8. Completion of corneoscleral section
9. Hydrodissection: the balanced salt solution (BSS) is injected under the peripheral part of the anterior capsule. This manoeuvre separates the corticonuclear mass from the capsule.
10. Removal of nucleus
    a. Pressure and counterpressure method
    b. Irrigating wire vectis technique
11. Aspiration of cortex
12. Implantation of IOL
13. Closure of the incision
14. Removal of viscoelastic substance: before tying the last suture the viscoelastic material is aspirated out with 2 way cannula and anterior chamber is filled with BSS.
15. Conjunctival flap is reposited
16. Subconjunctival injection of dexamethasone 0.25ml and gentamicin 0.5ml
17. Patching of eye.

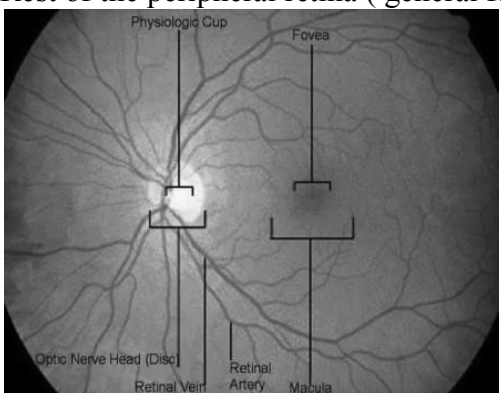
RETINA

- Innermost tunic of the eyeball
- Thin, delicate, transparent membrane.
- Most highly developed tissue of the eye.
- Extends from optic disc to ora serrata.



Three distinct regions on ophthalmoscopic examination:

- Optic disc
- Macula lutea
- Rest of the peripheral retina (general fundus)



Optic disc

- Pale, pink, well defined circular area of about 1.5mm diameter.
- At the optic disc, all the retinal layers terminate except the nerve fibres, which pass through the lamina cribrosa to run into the optic nerve.
- The physiological cup of the optic disc is a depression seen in it.

Macula lutea (yellow spot)

- Comparatively dark area 5.5 mm in diameter
- Situated at the posterior pole of the eyeball, temporal to the optic disc.
- Fovea centralis is the central depressed part of the macula.
- Foveola forms the central floor of the fovea.

DIABETIC RETINOPATHY

- Diabetic retinopathy is a leading cause of blindness.
- Classification:
  - Non-Proliferative Diabetic Retinopathy (NPDR)
  - Proliferative Diabetic Retinopathy (PDR)

Pathogenesis

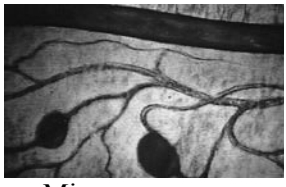
- Cellular damage:
  - Damage to the endothelial cells
  - Loss of pericytes
  - Thickening of basement membrane of capillaries
- Hematological and biochemical changes
  - Pletelet adhesiveness increase
  - Blood viscosity increase
  - RBC deformation
  - Leukostasis increase

Non-proliferative Diabetic Retinopathy(NPDR)

- Earliest stage.
- Damaged blood vessels in retina begin to leak extra fluid and some amount of blood into the eyes
- Sometimes deposits of cholesterol and fats from the blood may leak into the retina

Signs

- Microaneurysms: small bulges in blood vessels formed due to focal dilation of capillary wall following loss of pericytes.
- Retinal hemorrhages
- Hard exudates

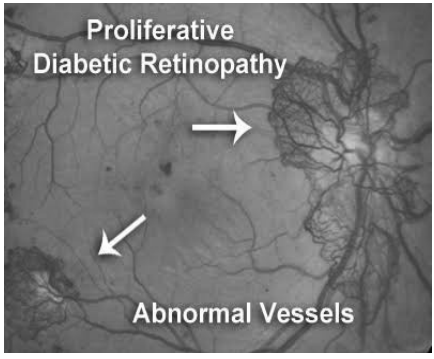


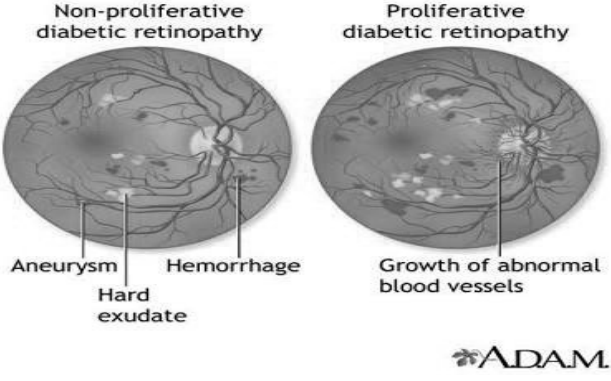
Microaneurysms

Proliferative Diaetic Retinopathy(PDR)

- Occurance of neovascularization: proliferation of new vessels from the capillaries, in the form of neovascularization at the optic disc and/or elsewhere in the fundus.
- In attempt to supply blood to the area where the original vessels closed, the retina responds by growing new blood vessels – neovascularization.
- However, these new vessels are abnormal and do not supply the retina with proper blood flow.
- Proangiogenic factors such as
    - VEGF (Vasculoendothelial growth factors)
    - PDGF (Pletelet derived growth factor)

  are released as a result of ischemia produced by microvascular occlusions.





Treatment:

- Control of DM
- Intravitreal anti-VEGF drugs
- Intravitreal steroids
- Laser therapy
- Surgery

HYPERTENSIVE RETINOPATHY

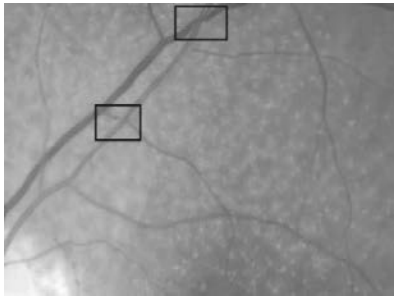
- Hypertensive retinopathy refers to fundus changes occurring in patients suffering from systemic hypertension.
- It is diagnosed based upon its clinical appearance on dilated fundoscopic exam and coexistent hypertension.

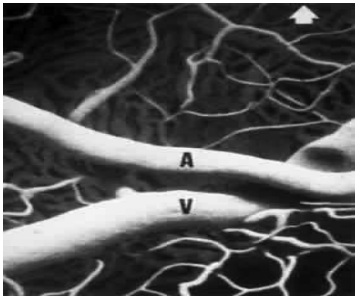
Signs

- AV nicking
- Arteriolar reflex changes
- Superficial retinal haemorrhage
- Cotton wool spots
- Hard exudates

AV nicking

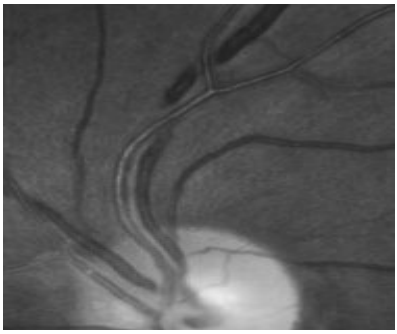
- Arteriovenous nicking is the phenomenon, where, on examination of eye, a small artery (arteriole) is seen crossing a small vein (Venule), which results in the compression of the vein with bulging on either side of the crossing.
- The arteriole's thicker walls push against those of the venule forcing the venule to collapse.

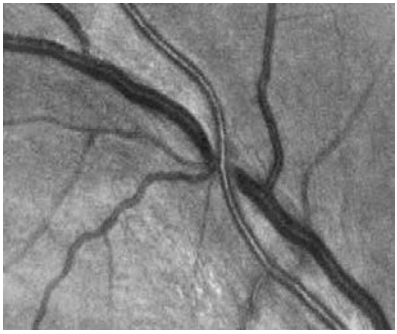




Arteriolar reflex changes

- Grade I & II: more diffuse and less bright reflex is seen due to thickening of vessel wall
- Grade III : **Copper wiring** i.e. reddish-brown reflex occurs due to progressive sclerosis
- Grade IV : **Silver wiring** i.e. opaque white reflex occurs ultimately due to the continued sclerosis





Superficial retinal hemorrhage

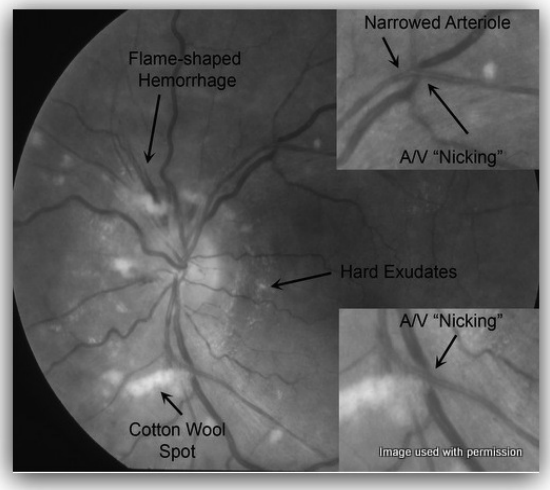
- Due to disruption of the capillaries in the retinal nerve fibre layer.

Hard exudates

- Lipid deposits in the outer plexiform layer of retina which occur following leaky capillaries

Cotton wool spots

- Caused by ischemia to the nerve fibre layer secondary to luminal narrowing.
- Ischemia to the nerve fibres leads to nerve swelling and ultimately fluffy opacification.



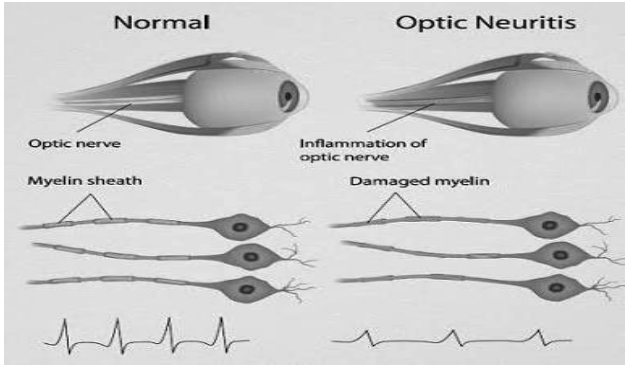
Prevention

- Routine blood pressure monitoring and treatment will prevent hypertensive retinopathy from developing.
- Finding these vascuar changes suggests that chronic hyprertension is producing end-organ damage and must be controlled.

OPTIC NEURITIS

An inflammation of the optic nerve

- Optic neuritis includes inflammatory and demyelinating disorders of the optic nerve.



Anatomical types

- Papilltis: involvement of the optic disc in inflammatory and demyelinating disorders.
- Neuroretinitis: combined involvement of optic disc and surrounding retina in the macular area.
- Retrobulbar neuritis: involvement of the optic nerve behind the eyeball.

Symptoms

- Visual loss: sudden, progressive and profound visual loss
- Dark adaptation may be lowered
- Visual obscuration in bright light
- Impairment of colour vision
- Occasionally, altered perception of moving object (Pulfrich's phenomenon)
- Pain: mild eyeache especially aggravated by ocular movement.
- Worsening of symptoms with exercise or an increase in body temperature (Uhthoff's symptom).

Treatment:

- Treatment of the cause
- Corticosteroid therapy:
  - Intravenous methylprednisolone

OPTIC ATROPHY

Degeneration of the optic nerve

- Death of retinal ganglion cell axons that comprise the optic nerve with the resulting picture of pale optic disc.
- The optic nerve comprises approximately 1.2million axons that originate at the ganglion cell layer. The axons, once damaged, do not regenerate.
- Since the optic nerve transmits retinal information to the brain, optic atrophy is associated with vision loss.
- Primary: simple degeneration of the nerve fibres without any complicating process within the eye
- Secondary: following any pathologic process which produces optic neuritis.

- Primary : lesions proximal to the optic disc without antecedent papilloedema
  - Multiple sclerosis
  - Retrobulbar neuritis
  - Intracranial tumours pressing directly on the anterior visual pathway
  - Traumatic severance
- Consecutive : occurs following destruction or inflammatory lesions of the choroid or retina
  - Retinitis pigmentosa, chorioretinitis
- Post-neuritic : develops as a sequele to long standing papilloedema or papillitis.
- Glaucomatous: long standing raised IOP
- Vascular (Ischemic): conditions producing disc ischemia
  - Severe hemorrhage, severe anemia, quinine poisoning.

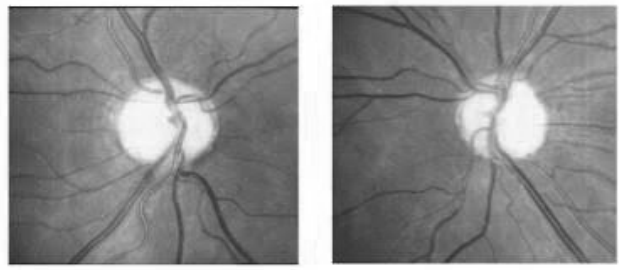
Symptoms

Loss of vision:

- Sudden or gradual (depending upon cause of optic atrophy)
- Partial or total (depending upon the degree of atrophy)

- Optic disc is always pale.
- Decrease in the number of small blood vessels.

Primary Optic Atrophy



Treatment:

- Treatment of cause may help in preserving some vision

- However, once complete atrophy has set in, the vision cannot be recovered.

AGE RELATED MACULAR DEGENERATION (ARMD)/SENILE MACULAR DEGENERATION

- Deterioration or breakdown of the macula.
- Bilateral disease
- Affects persons over 50 years of age
- Leading cause of blindness
- Exact cause of ARMD is still unknown.
- Risk factors which may affect the age of onset/progression :
    - Heredity
    - Nutrition
    - Smoking
    - Hypertension
    - Exposure to sunlight

Symptoms

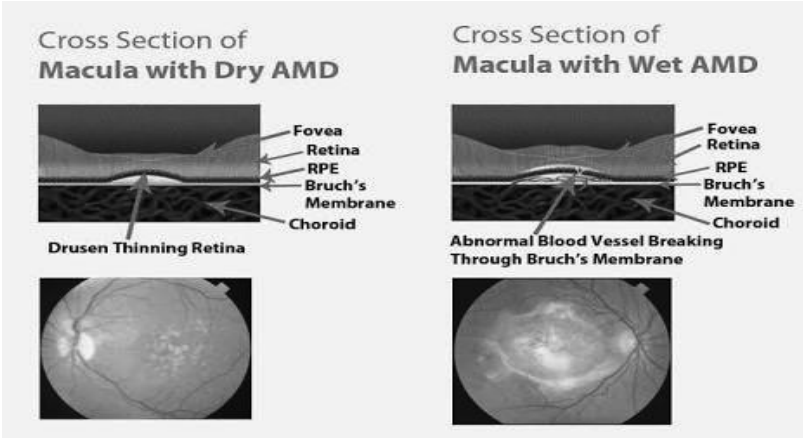
- Bluriness, dark areas or distortion in central vision and perhaps permanent loss of central vision.
- Usually does not affect peripheral vision.

Non-exudative/Dry/Atrophic ARMD

- Responsible for 90% of patients.
- Caused by aging and thinning of the tissue of the macula.
- Begins when tiny yellow or white pieces of fatty protein called drusen form under the retina.
- Eventually, the macula may become thinner and stop working properly.

Exudative/Wet/Neovascular ARMD

- Responsible for only 10% cases
- Occurs when abnormal blood vessels begin to grow underneath the retina.
- These new blood vessels may leak fluid or blood
- Vision loss from this form may be faster and more noticeable than that from dry ARMD.

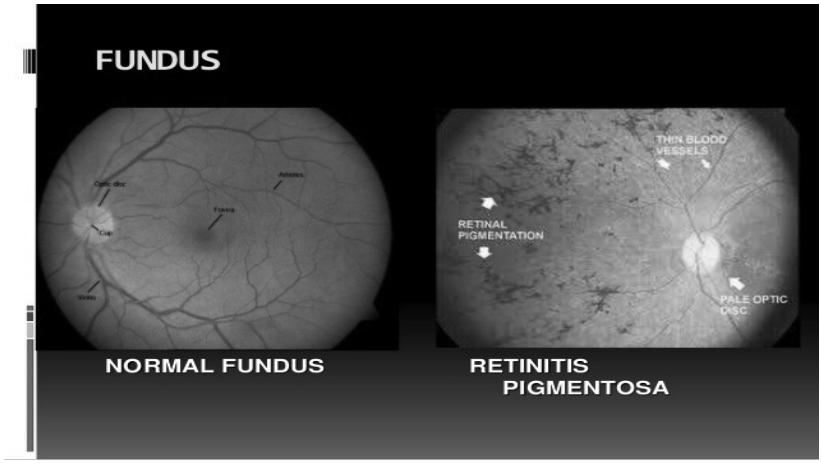


Treatment:

- Currently, there is no effective treatment that stop progression of dry ARMD.
- Dietary supplements and antioxidant vitamins may help to lower the risk for or halt the progression of dry ARMD.
- Laser photocoagulation is effective in sealing leaking or bleeding subretinal vessels in some eyes with exudative macular degeneration.

RETINITIS PIGMENTOSA (PIGMENTARY RETINAL DYSTROPHY)

- Slow, degenerative disease of retina
- The degeneration predominantly affects the rods more than the cones.
- Rods are degenerated early and cones are involved late.
- Occurs in 1 person per 5000 of the world population
- Appears in the childhood and progresses slowly, often resulting in blindness in advanced middle age.
- Disease is almost invariably bilateral and both eyes are equally affected.



Clinical features:

- Night blindness: it is the characteristic and earliest feature. It occurs due to degeneration of rods.
- Tubular vision: i.e. loss of peripheral vision
- Central vision is also lost ultimately after many years. (50-60 years of age)

Treatment:

- Till date, there is no effective treatment for the disease.
  - Correct refractive error
  - Low vision aids in the form of magnifying glasses and night vision devices my be of some help
  - Rehabilitation of the patient should be earned out as per his socioeconomic background.

EALE'S DISEASE (RETINAL VASCULITIS)

- Primary inflammation of the retinal vessels wall.
- Idiopathic inflammation of the peripheral retinal veins.
- Characterized by recurrent vitreous haemorrhage.

Etiology:

- Not known exactly

Clinical features:

- Bilateral disease
- Affects young adult males (20-30 years)
- Symptoms:
  - Sudden appearance of floaters in front of the eyes
  - Sudden painless loss of vision due to vitreous haemorrhage

Clinical course:

- Active inflammation
- Ischemia / vascular occlusion
- Neovascularization
- Sequelae / advance stage

Treatment:

- Medical:
  - Oral corticosteroids for extended periods
- Laser photocoagulation
- Vitreoretinal surgery

CENTRAL SEROUS RETINOPATHY (CSR)

- Spontaneous serous detachment of neurosensory retina in the macular region, with or without retinal pigment epithelium detachment.

Risk factors

- Emotional stress
- Hypertension
- Pregnancy (3^{rd} trimester)
- Cushing's disease

Clinical features:

- Sudden painless loss of vision
- Relative positive scotoma
- Signs:
  - Mild elevation of macular area

- Small yellow elevations may be seen due to RPE detachment.
- Foveal reflex is absent or distorded

Treatment:

- Conservative
  - Reassurance. CSR undergoes spontaneous resolution in 80-90% cases. Visual acuity returns to normal or near normal within 3 to 6 months.
  - Life style changes to reduce stress in life
- Laser photocoagulation
- Photodynamic therapy

STRABISMUS

- Eyes don't look in exactly the same direction.
- A condition which interferes with binocular vision because it prevents a person from directing both eyes simultaneously towards the same fixation point.
- The eyes don't properly align with each other.

Classification of strabismus:

1. Apparent squint/ pseudostrabismus
2. Latent squint (heterophoria): tendency of eyeballs to deviate is kept latent by fusion. Therefore, when the influence of fusion is removed the visual axis of one eye deviates away.
    a. Esophoria: tendency to deviate inwards
    b. Exophoria: tendency to deviate outwards
    c. Hyperphoria: tendency to deviate upwards
    d. Cyclophoria: tendency to rotate around the anteroposterior axis.
3. Manifest squint (Heterotropia)
    a. Concomitant
    b. Incomitant

Latent squint (Heterophoria):

Physiological factors:

1. Age:
    a. Esophoria: common in younger age group
    b. Exophoria: in elderly
2. Role of accommodation:
    a. Increased accommodation (hypermetropes, excessive near work): esophoria
    b. Decreased accommodation(Myopes): exophoria
3. Role of convergence:
    a. Excessive use of convergence(bil. Cong. Myopes): esophoria
    b. Decreased use of convergence(presbyopes): exophoria

4. Dissociation factors:
    a. Prolonged constant use of one eye: exophoria (using uniocular microscope and watch makers using uniocular magnifying glass).

Factors predisposing to decompensation:

1. Inadequacy of fusional reserve
2. General debility and lowered vitality
3. Psychosis, neurosis, mental stress
4. Precision of job
5. Advancing age

Treatment:

1. Correction of R.E.
2. Orthoptic treatment
3. Prescription of prism in glasses
4. Surgical treatment.

Manifest squint (Heterotropia)

- It involves lack of co-ordination between the extraocular muscles which prevents directing the gaze of both eyes at once to the same point.

Types:

- Concomitant
  - Convergent
  - Divergent
  - vertical
- Incomitant
  - Paralytic
  - A and V pattern
  - Restrictive

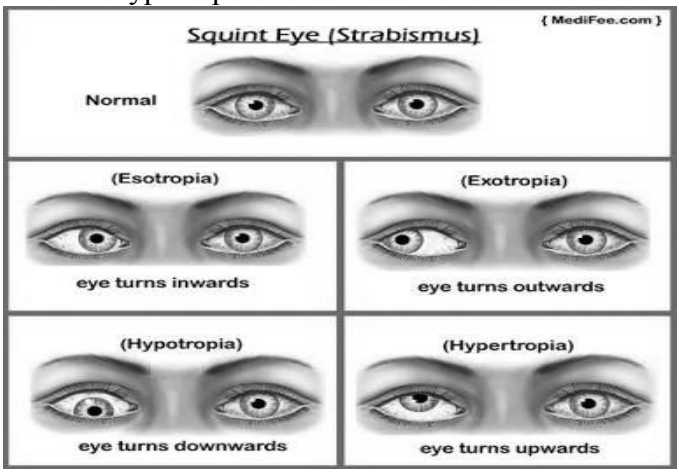
Concomitant:

- The amount of deviation in the squinting eye remains constant in all directions of gaze.
- Although the eyes are misaligned, they retain their abnormal relation to each other in all directions of gaze.

1. Convergent (Esotropia)
- Inward deviation of one eye.
- Most common type of squint in children.

2. Divergent (Exotropia)
- Outward deviation of one eye.

3. Vertical
- Hypertropia: upward deviation
- Hypotropia: downward deviation



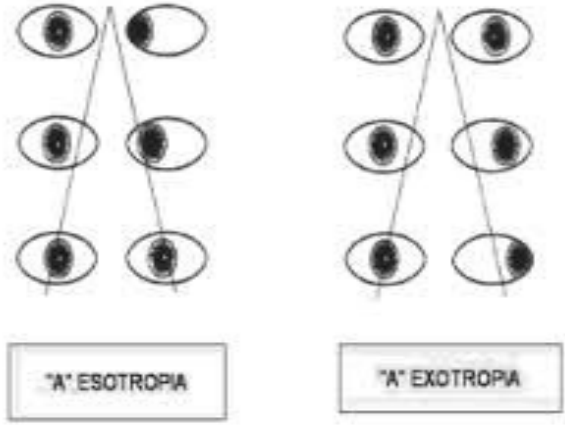
Treatment:
- Spectacles with full correction of RE
- Occlusion therapy: indicated in presence of amblyopia. After correcting RE, the normal eye is occluded and patient is advised to use the squinting eye.
- Squint surgery:
  - To weaken the strong muscle by recession
  - To strengthen the weak muscle by resection.

Incomitant:
- The amount of deviation varies in different directions of gaze.

1. Paralytic:
- Ocular deviation resulting from complete or incomplete paralysis of one or more extraocular muscles.

2. A and V pattern:

- The amount of deviation in squinting eye varies by more than 10 and 15 respectively, between upward and downward gaze.
- A and V esotropia: In A esotropia, the amount of deviation increases in upward gaze and decreases in downward gaze. The reverse occurs in V esotropia.
- A and V exotropia: In A exotropia, the amount of deviation decreases in upward gaze and increases in downward gaze. The reverse occurs in V exotropia.



3. Restrictive:

- The extraocular muscle is not paralysed but its movement is mechanically restricted.

Strabismus surgery

- Techniques:
  - Muscle weakening procedures
  - Muscle strengthening procedures
  - Procedures that change direction of muscle action.

AMBLYOPIA

- A partial reversible loss of vision in one or both eyes, for which no cause can be found by physical examination of the eye, i.e. absence of any organic disease of ocular media, retina and visual pathway.

Pathogenesis:

- It is produced by certain amblyogenic factors during critical period of visual development. (birth to 6-7 years of age)
- During this period the visual pathway continues to develop and brain learns to interpret the signals that come from the eye.
- If for any reason, a young child cannot use one or both eyes normally, then the vision is not developed completely – amblyopia.

- The most sensitive period for development of amblyopia is first six months and it usually does not develop after the age of 6-7 years.

Amblyogenic factors:

- Visual deprivation as in anisometropia
- Light deprivation e.g. due to congenital cataract
- Abnormal binocular interaction e.g. in strabismus.

Types:

- **Strabismic amblyopia**:Prolonged uniocular suppression in children with unilateral constant squint
- **Stimulus deprivation amblyopia**:Develops when one eye is totally excluded from seeing early in life as in congenital or traumatic cataract, complete ptosis etc
- **Anisometropic amblyopia**: occurs in eye having higher degree of RE than the other.
- **Isoametropic amblyopia**: bilateral amblyopia in children with bilateral uncorrected high RE.

- **Meridional amblyopia**: children with uncorrected astigmatic RE.

Treatment:

- Occlusion therapy:
  - Occlusion of normal eye to force the use of amblyopic eye is the main treatment.

COMMUNITY OPHTHALMOLOGY

- Community ophthalmology, over the years, has developed as an important branch of community medicine.
- Its activities emphasize the prevention of ocular diseases and visual impairment; reduction of ocular disability; promotion of ocular health, quality of life and efficiency of a group of people at the community level.
- Thus it can be defined as a system which utilises the full scope of ophthalmic knowledge and skill, methodology of public health and services to other medical and nonmedical agencies to promote ocular health and prevent blindness at the community level with an active, recognised and crucial role of community participation.
- The concept of community ophthalmology has become more relevant and essential to achieve the goal of "vision 2020: the right to sight" and to accomplish the theme behind vision for the future (VFTF)

WHO definition of blindness: "visual acuity of less than 3/60 (Snellen) or its equivalent".

In order to facilitate the screening of visual acuity by nonspecialised persons, in the absence of appropriate vision charts, WHO in 1979 added "the inability to count the fingers in daylight at a distance of 3 metres" to indicate vision less than 3/60 or its equivalent.

Causes of global blindness:

1. Cataract : 39%
2. Glaucoma : 10%
3. Corneal scarring including trachoma : 3%

4. ARMD : 7%
5. Diabetic retinopathy : 4%
6. Childhood blindness including xerophthalmia : 3%

Global initiatives for prevention of blindness:
The major global initiatives are:

1. WHO: Prevention of Blindness Programme (PBP) (1978)
2. Vision 2020: the right to sight (1999)
3. Vision For The Future (VFTF) (2001).

National Programme For Control Of Blindness (NPCB) in India

- India was the first country in the world to launch the National Programme for control of Blindness (NPCB) in year 1976 as 100% centrally sponsored programme with the following goals:
  - To provide comprehensive eye care facilities for primary, secondary and tertiary levels of eye health care
  - To reduce the prevalence of blindness in population from 1.38% (ICMR 1971-1974) to 0.31 by 2000AD.
- Vision 2020 has been adopted in NPCB with the objective to eliminate avoidable blindness by the year 2020.
- Community ophthalmology practice at primary level:

Promotive	Preventive	Curative	Rehabilitative
• Nutrition education • Improved maternal and child nutrition • Health education • Face washing	• Ocular prophylaxis at birth • Vit.A doses • Measles vaccine • Avoid medication in pregnancy	• Vision screening • Treatment for Vit.A deficiency • Referral for surgery • Emergency management • Treatment for trachoma	• Provision of low vision services • Community based rehabilitation • Counselling of the incurable blind

Promotive	Preventive	Curative	Rehabilitative
• Good antenatal care • Safe water • Improved environmental sanitation	• Avoid hypoxia at birth • Examine neonate's eye • Nutrition supplementation	• Treatment for common eye diseases	• Certification of blind by eye surgeon

EYE BANK

- Eye bank is an organisation which deals with the collection, storage and distribution of cornea for the purpose of corneal grafting, research and supply of the eye tissue for other ophthalmic purpose.
- **Functions of an eye bank:**
  - Promotion of eye donation by increasing awareness about eye donation to general public
  - Registration of the pledger for eye donation
  - Collection of donated eyes
  - Receiving and processing the donor eyes
  - Preservation of the tissue for short, intermediate, long or very long term.
  - Distribution of the donor tissues to the corneal surgeons
  - Research activities for improvement of the preservation methodology etc
- **Eye bank personnel:**
  - Eye bank in charge
  - Eye bank technician
  - Clerk-cum-storekeeper
  - Medical social worker or public relation officer
  - Driver-cum-projectionist

EYE DONATION

- Eye donation is an act of donating one's eyes after his/her death.
- It is an act of charity, purely for the benefit of the society and is totally voluntary
- Only corneal blinds can be benefitted through this process not other blinds. Removing the damaged cornea and replacing it with a healthy cornea by surgery can cure corneal blindness.
  (Artificial cornea has not yet been developed and hence the only source for cornea is from human beings.)
- The cornea of the received eye can be used to restore vision to corneal blind person. The other portions of the eye are also used for research and training purposes to develop cures for some of the common eye diseases.

How can one donate eyes?

- The eyes of a dead person can be used only if they are taken out within 6 hours of death.
- When a person who has given consent for the donation of eyes dies, the eye should be kept wet by sprinkling water on them till the eye-surgeon arrives.
- After the removal of the eyes, the eyelids are stitched together and there will be no visible sign of the eyes having been removed.
- The entire process is completed within 10-15 mins.

Who can donate eyes?

- Any person of any age can donate eyes.
- Even if the person has medical history of hypertension, diabetes, asthma, tuberculosis etc and even spectacle wearers and people who have undergone cataract surgery can donate eyes.

Who cannot donate their eyes?

- Patients suffering from rabies, tetanus, AIDS, Hepatitis B and C, jaundice, cancer, gangrene, brain tumour, food poisoning, septicaemia and a person who has died due to drowning etc cannot donate their eyes.

After eye donation:

- The donor's family receives a certificate of appreciation from the eye bank.
- The eyes are taken to the eye bank and evaluated by a trained eye bank staff.
- Tests are carried out and the tissue is sent to the corneal surgeon.

CORNEAL TRANSPLANT

- Corneal transplants are the most common organ and tissue transplants.
- According to the National Eye Institute, about 40,000 people undergo this procedure every year.
- Indications:
  - Thinning of cornea
  - Cornel infection and injury
  - Clouding of cornea
  - Swelling of cornea
  - Corneal ulcers
- Corneal transplantation is done either to restore vision, to relieve pain in a corneal infection or for cosmetic reasons.
- In a cornea transplant (keratoplasty), a portion of the diseased cornea is replaced with healthy cornea from a donor.
- Two types:
  - Full thickness grafting – penetrating keratoplasty
  - Partial thickness grafting – lamellar keratoplasty
- Corneal transplant is usually performed within 4 days after donation, depending upon the method of cornea preservation.

- If transplant fails, it does not mean blindness or loss of the eye. Rather a subsequent transplant may be performed with a good chance of success.
- About one in five people undergoing corneal transplant, eventually reject the donor cornea, making it necessary to transplant another one.

EXAMINATION OF EYE

- Testing of visual acuity
- External ocular examination
- Fundus examination
- Visual field examination

1. Testing of Visual acuity

- Distant visual acuity
  - Snellen's test
- Visual acuity for near
  - Jager's chart
  - Snellen's near vision test

Snellen's test types

- The distant central visual acuity is usually tested by Snellen's test types.
- It consists of a series of black capital letters on a white board, arranged in lines, each progressively diminishing in size.
- The letters of the top line of snellen's chart should be read clearly at a distance of 60 m.
- Similarly, the letters in subsequent lines should be read from a distance of 36, 24, 18, 12, 9, 6 and 5 m, respectively.

Procedure of testing

- The patient is seated at a distance of 6 m from the snellen's chart, so that the rays of light are practically parallel and the patient exerts minimal accomodation.
- The chart should be properly illuminated.

- The patient is asked to read the chart with each eye separately and the visual acuity is recorded as a fraction, the numerator being the distance of the patient from the letters, and the denominator being the smallest letters accurately read.
- When the patient is able to read up to 6 m line, the visual acuity is recorded as 6/6, which is normal.

Visual acuity for near

- Near vision is tested by asking the patient to read the near vision chart, kept at a distance of 35 cm in good illumination.

2. External ocular examination

- Examination for the head posture
- Examination of forehead and facial symmetry
- Examination of eyebrows
- Examination of the eyelids
- Examination of lacrimal apparatus
- Examination of eyeball as a whole
- Examination of conjunctiva
- Examination of sclera
- Examination of cornea
- Examination of anterior chamber
- Examination of the iris
- Examination of the pupil
- Examination of the lens

Examination of head posture

- Position of head and chin should be noted first of all.
- Head posture may be abnormal in a patient with paralytic squint. (head is turned in the direction of the action of paralysed muscle to avoid diplopia) and incomplete ptosis (chin is elevated to uncover the pupillary area in a bid to see clearly)

Examination of forehead and facial symmetry

- Forehead may show increased wrinkling (due to overaction of frontalis muscle) in patients with ptosis.
- Complete loss of wrinkling in one-half of forehead is observed in patients with lower motor neuron facial palsy.
- Facial asymmetry may be noted in patient with Bell's palsy and facial hemiatrophy.

Examination of eyebrows

- Level of the two eyebrows may be changed in a patient with ptosis (due to overaction of frontalis)
- Cilia of lateral one-third of the eyebrows may be absent (madarosis) in patients with leprosy or myxoedema.

Examination of eyelids

- Position:
  - Normally the lower lid just touches the limbus while the upper lid covers about $1/6^{th}$ of cornea.
  - In ptosis, upper lid covers more than $1/6^{th}$ of cornea.
  - Upper limbus is visible due to lid retraction as in thyrotoxicosis and sympathetic overactivity.
- Movements of lids:
  - Blinking is involuntary movement. Normal rate is 12-16 blinks per minute. Blinks are decreased in trigeminal anaesthesia and absent in those with 7^{th} nerve palsy.
  - Lagophthalmos is a condition in which the patient is not able to close his lids. Causes are
    - Facial nerve palsy
    - Extreme degree of proptosis
- Lid margin:
  - Entropion (inward turning of lid margin)
  - Ectropion (outward turning of lid margin)
  - Eyelash abnormalities:
    - Trichiasis
    - Distichiasis
    - madarosis

  - Scales at the lid margins: in blepharitis
  - Swelling at lid margin may be stye, papilloma or marginal chalazion.
- Palpebral aperture:
  - The exposed space between the two lid margins is called palpebral fissure which measures 28-30 mm horizontally and 8-10 mm vertically (in the centre).
  - Ankyloblepharon: usually seen following adhesions of the two lids at angles e.g. after ulcerative blepharitis and burns. It results in horizontally narrow palpebral fissure.
  - Blepharophimosis: all around narrow palpebral fissure (congenital anomaly)
  - Vertically narrow palpebral fissure
    - Ptosis
    - Enophthalmos (sunken eyeball)
    - Anophthalmos (absent eyeball)
    - Microphthalmos (congenital small eyeball)
  - Vertically wide palpebral fissure:
    - Proptosis
    - Large sized eyeball
    - Facial nerve palsy

Examination of Lacrimal apparatus

- Inspection of lacrimal sac area for redness, swelling or fistula.
- Inspection of lacrimal puncta, for any defect such as eversion, stenosis, absence or discharge.
- Regurgitation test
- Lacrimal syringing
- Jone's dye test I and II

Examination of eyeball as a whole

- Position of eyeballs:
  - Normally, the two eyeballs are symmetrically placed in the orbits in such a way that a line

joining the central points of superior and inferior orbital margins just touches the cornea.
  - Abnormalities in position:
    - Proptosis/exophthalmos: bulging of eye
    - Enophthalmos (sunken eyeball)
- Size of eyeball:
  - Increased in : buphthalmos, unilateral high myopia
  - Decreased in : congenital microphthalmos, phthisis bulbi
- Visual axes of eyeball:
  - Visual axes are simultaneously directed at the same object which is maintained in all the directions of gaze.
  - Deviation in visual axis of one eye is called squint.

Examination of conjunctiva

- Bulbar conjunctiva
- Palpebral conjunctiva
  - Lower palpebral
  - Upper palpebral

Conjunctival signs

- Discolouration
  - Brownish in malenosis
  - Greyish due to surma deposits
  - Pale in anaemia
  - Bluish in cyanosis
- Congestion of vessels
- Conjunctival chemosis (oedema) : in allergic or inflammatory conditions
- Follicles
- Papillae
- F.B.
- Scarring
- Pterygium
- Pinguecula : degenerative condition observed in many adult patients.

Examination of sclera

- Discoloration : normally white.
  - Yellow in jaundice
- Inflammation
  - Episcleritis: superficial localised pink or purple circumscribed flat nodule
  - Scleritis: deep, dusky patch with marked inflammation
- Staphyloma: thinned out bulging are of sclera
- Traumatic perforations in blunt trauma are usually seen in the region of limbus or at the equator.

Examination of cornea:

- Size
  - Microcornea: anterior horizontal diameter is less than 10 mm.
  - Megalocornea: when horizontal diameter is more than 13 mm.
- Shape (Curvature)
  - Keratoglobus: an ectatic condition in which cornea becomes thin and bulges out like a globe
  - Keratoconus: an ectatic condition in which cornea becomes cone shaped.
  - Cornea plana: flat curvature of cornea
- Surface :
  - Smoothness is disturbed due to abrasions, ulcerations etc.
  - Placido's keratoscopic disc.
- Sheen (shine / lustre) : Normal cornea is a bright shining structure. Sheen of corneal surface is lost in dry eye conditions.
- Transparency: transparency is lost in corneal oedema, opacity, ulceration, degenerations, vascularization etc.
- Corneal vascularization
- Corneal sensations: the cornea is a very sensitive structure, being richly supplied by the nerves. The sensitivity is diminished in herpetic keratitis, neuroparalytic keratitis, leprosy, DM, absolute glaucoma,

trigeminal block for postherpatic neuralgia. Aesthesiometer.

- Back of cornea: for KP – a sign of anterior uveitis.
- Biomicroscopic examination after staining of cornea with vital stains:
  - Fluorescein staining: the area denuded of epithelium due to abrasions or ulcer when stained with fluorescein appear brilliant green. When examined using cobalt blue light, the stained area appears opaque green.
  - Bengal rose (1%) stains the diseased and devitalised cells red. As In superficial punctuate keratitis. Very irritating. 2% xylocaine should be used before using it.
  - Alcian blue dye stains the excess mucus selectively as in KCS.

Examination of iris:

1. Colour: varies in different races
   a. Heterochromia iridum: diff. colour of two iris
   b. Heterochromia iridis: diff. colour of sectors of the same iris
2. Pattern of normal iris: peculiar due to presence of collarette, crypts. This pattern is disturbed due to muddy iris in acute iridocyclitis.
3. Persistent papillary membrane (PPM): abnormal congenital tags of iris tissue adherent to collarette area.
4. Synechiae: adhesion of iris to other intraocular structure.
   a. Anterior: adherent leucoma
   b. Posterior: iridocyclitis. May be total / annular (ring) / segmental
5. Iridodonesis: tremulousness of iris. Aphakia / subluxation of lens.
6. Nodules on the iris surface:
   a. Granulomatous uveitis: koeppe's, busacca's nodules
   b. Malenoma, tuberculoma, gumma of iris

7. Rubeosis iridis: new vessel formation on iris. DR, CRVO, BRVO, chronic uveitis, chronic retinal detachment
8. A gap or hole in iris: congenital coloboma / due to iridectomy (surgical coloboma). Separation of iris from CB is called iridodialysis.
9. Aniridia / irideremia : complete absence of iris. Rare congenital condition.
10. Iris cyst: seen near papillary margin in patients using strong miotic drops.

Examination of pupil

- Number: normally, there is only one pupil. Rarely, there may be more than one pupil. This congenital anomaly is called **Polycoria**.
- Location: almost in the centre (slightly nasal) of iris.
- Size: normal pupil size varies from 3 to 4 mm depending upon the illumination. It may be abnormally small (miosis) or large (mydriasis).
- Shape: circular.
- Colour: of course, the pupil is a hole in iris, but the pupillary area does exhibit colour depending upon the condition of the structures located behind it.
    - Greyish black – normally
    - Jet black – aphakia
    - Greyish white – immature senile cortical cataract
    - Pearly white – mature cortical cataract
    - Milky white – hypermature cataract
    - Leucocoria (white reflex in pupil): in children
        - Congenital cataract, retinoblastoma
        - Amaurotic cat's eye reflex: yellowish white, semidilated, non-reacting pupil. in retinoblastoma and pseudoglioma.
- Pupillary reactions:
    - Direct light reflex
    - Consensual light reflex
    - Swinging flash light test
        - Marcus gunn pupil/RAPD: earliest indication of optic nerve disease

    - Near reflex
- Abnormal papillary reactions:
    - Amaurotic pupil
    - Efferent pathway defect
    - Wernicke's hemianopic pupil
    - Marcus Gunn pupil
    - Argyll Robertson pupil
    - Tonic pupil

Miosis	Mydriasis
1. Local miotic (Parasympathomimetic) drugs 2. Systemic morphine 3. Senile rigid miotic pupil 4. During sleep pupil is pinpoint 5. Strong light 6. Head injury (Pontine hemorrhage) 7. Iridocyclitis 8. Horner's syndrome	1. Topical sympathomimetic drugs (Adrenaline, phenylephrine) 2. Topical parasympatholytic drugs (Atropine, homatropine, tropicamide, cyclopentolate) 3. Acute congestive glaucoma 4. Absolute glaucoma 5. Optic atrophy 6. Retinal detachment 7. Internal ophthalmoplegia 8. 3rd nerve paralysis 9. Belladonna poisoning

Examination of lens

- A thorough examination of lens can be accomplished with the help of oblique illumination, slit lamp biomicroscopy and distant direct ophthalmoscopy with fully dilated pupils.
- Position: it is positioned in the patellar fossa (space between the vitreous and back of iris) by the zonules.

  - Dislocation of lens
  - Subluxation of lens – lens is partially displaced. Zonules are intact in some quadrant and lens is shifted on that side.
  - Aphakia – absence of lens
  - Pseudophakia
- Shape of lens: normally biconvex
  - Spherophakia : spherical lens
  - Lenticonus anterior : anterior cone shaped bulge
  - Lenticonus posterior : posterior cone shaped bulge.
- Transparency: normal lens is transparent structure. Any opacity in the lens is called cataract.
- Deposits on anterior surface of lens:
  - Vossius ring: a small ring shaped pigment. After blunt trauma.
  - Pigmented clumps: iridocyclitis
  - Dirty white exudates: uveitis / endophthalmitis
  - Rusty deposits: deposition of ferrous ions – siderosis bulbi
  - Greenish deposits: deposition of copper ions – chalcosis.
- Purkinje image test:

The Intraocular Pressure

- The measurement of IOP should be made in all suspected cases of glaucoma and in routine after the age of 40 years.
- The exact measurement of IOP is done by an instrument called tonometer.
- Normal IOP range is 10-21mm of Hg with an average tension of 16 ±5 mm of Hg.

3. Fundus examination

- Media
- Optic disc
- Macula
- Retinal blood vessels
- General background

- For thorough examination of fundus, the pupils should be dilated with 5% phenylephrine and/or 1% Tropicamide eye drops.

Media

- Normally ocular media is transparent.
- Causes of opacities are:
    - Corneal opacity
    - Lenticular opacity
    - Vitreous opacities (may be exudates, hemorrhage, degeneration, F.B.)

Optic disc

- Size: 1.5 mm diameter. Looks 15 times magnified during direct ophthalmoscopy.
- Shape: normal disc is circular
- Colour: normally pinkish with central pallor area. Hyperemia of disc in papilloedema, paler disc is seen in partial optic atrophy, chalky white in primary optic atrophy,
- Cup disc ratio: normal ratio is 0.3.
    - Large cup may be physiological or glaucomatous. Cup becomes full in papilloedema and papillitis.
- Kesten Baum index: ratio of large blood vessels vs small blood vessels on the disc. Normal is 4:16. Decreased in optic atrophy.

Macula

- The macula is situated at the posterior pole with its centre (foveola) being about 2 disc diameters lateral to temporal margin of disc.
- Normal macula is slightly darker than the surrounding retina.
- Its centre imparts a bright reflex (foveal reflex).

Retinal blood vessels

- Normal arterioles are bright red in colour and veins are purplish with a caliber ratio of 2:3.
- Narrowing of arterioles: in hypertensive retinopathy, arteriosclerosis, CRAO.
- Tortuosity of veins: in DM, CRVO

General background

- Normally pinkish red in colour.
- Superficial retinal hemorrhage: hypertension, DM, trauma, venous occlusions
- Deep retinal hemorrhage: in DR
- Cotton wool spots
- Hard exudates
- Microaneurysms
- Neovascularization

4. Visual field

- The total area in which objects can be seen in the side (peripheral) vision as we focus our eyes on a central point.
- Confrontation test
- Perimetry
    - Manual
    - Automated

CHAPTER 2
DISEASES OF HEAD

MIGRAINE

- It is thought that migraine is caused by genetic abnormality that makes the neurovascular system hyper-excitable.

Causes

- Underlying causes are unknown.
- They are believed to be related to a mix of environmental and genetic factors.
- They run in families in about two-thirds of cases.
- A number of psychological conditions are associated, including depression, anxiety etc.

Triggers

- Normal hormone fluctuations which occur with regular menstrual cycles may predispose some women to experience migraine headaches.
- Some oral contraceptives
- Alcohol beverages
- Various foods:
  - Chocolates, dairy products, MSG, artificial sweeteners
- Oversleeping
- Stress
- Exposure to strong stimuli such as bright lights, loud noises, strong smells

Symptoms

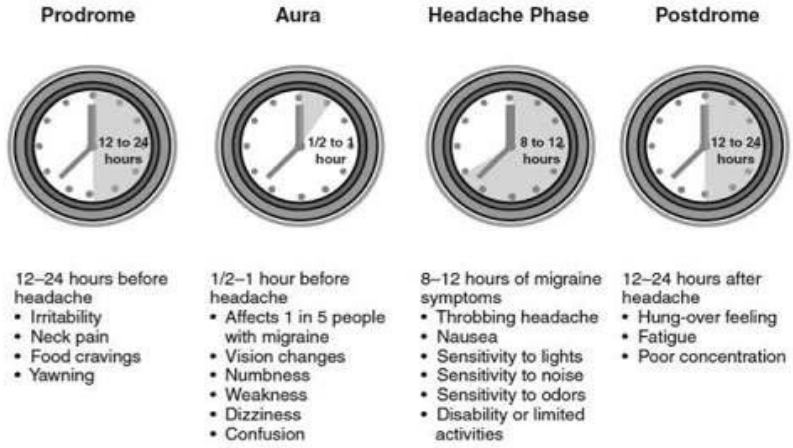
- Severe, often, pounding pain, usually on one side of the head
- Nausea and/or vomiting
- Sensitivity to light (Photophobia)
- Sensitivity to sound (Phonophobia)

- Eye pain
- Headache typically lasts for several hours up to several days.
- Aggravated by physical activity or exertion (walking upstairs etc)

Aura

- Neurological symptoms, usually happens before the headache.
- Due to changes in the cortex area of brain.
- Last from 5 to 60 minutes.
  - Changes in sight, such as dark spots, coloured spots, sparkles etc
  - Numbness or tingling
  - Weakness
  - Dizziness or vertigo
  - Speech and hearing may be disturbed.

Phases:



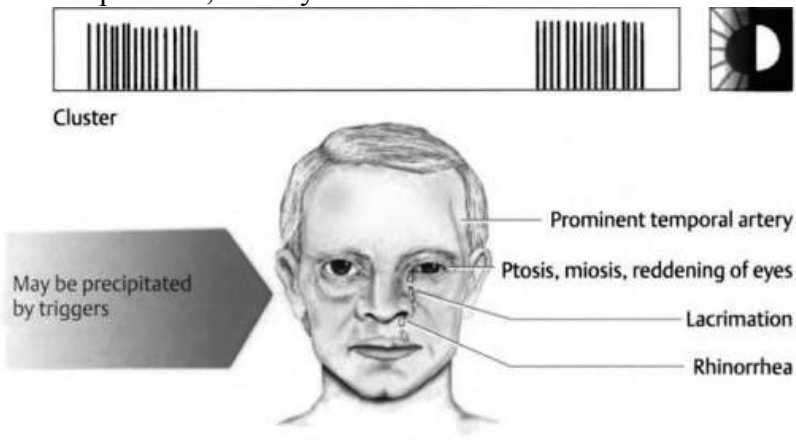
Treatment

- Abortive: to stop migraine once it starts.
  - Triptans (Serotonin receptor agonists)
  - Acetaminophen

  - Dihydroergotamine (DHE) : an ergot alkaloid used to treat migraines.
- Preventive
  - If migraines occur frequently, typically more than one migraine per week

CLUSTER HEADACHE

- Cluster headache is named after the demonstrated grouping of headache attacks occurring together (Cluster).
- Pain is almost always one-sided and it stays on the same side during a period (When a new headache period starts, it might switch to the opposite side, but that's rare)
- Pain is felt behind or around one eye.
- It may spread to forehead, temple, nose, cheek, upper gum on that side.
- Scalp may be tender.
- Restlessness, photosensitivity, phonophobia, photophobia may occur.
- Nausea is rare.
- Physical exhaustion, confusion, agitation, aggressiveness, depression, anxiety.



Cluster headache

- Headache lasts a short time – usually 30 to 90 minutes, but may be as little as 15 minutes or as long as 3 hours.

- They happen generally at the same time each day. **(alarm clock headache)**
- Those with cluster headaches may experience suicidal thoughts during an attack as a result of the pain. (giving the alternative name "**suicidal headache" or "suicide headache"**)
- Cluster headaches are more common in people who smoke or are heavy drinkers.

	Cluster	Migraine
Characteristics		
Frequency	1–6 per day, lasting For several weeks	1–10 per period of attack, 2–5 times per month
Duration	30–90 minutes	4–24 hours
Location	Only one side, around the eyes or back of the head	One or both sides of the head
Time of day	Usually at night and/or at the same time each day	Any
Average age of onset	20	10–50 years
Gender (majority)	Male	Female
Family history	7%	90%
Other symptoms		
Nausea and vomiting	2%–5%	85%
Blurry vision	Uncommon	Common
Watery eyes	Common	Uncommon
Nasal congestion	70%	Rare
Drooping eyelid	30%	1%–2%
Pupil contraction	50%	Absent

SOURCE: Michigan Headache and Neurological Institute

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TENSION HEADACHE

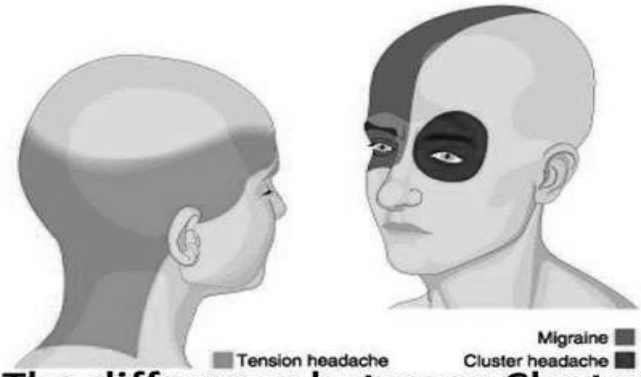
- Dull pain, tightness or pressure around forehead or the back of head and neck.
- There is no single cause.
- Most of the time, it is triggered by stress, whether from work, school, family, friend or other relationships.
- Women are twice as likely to get them as men.
- Some people get them because of tightened muscles in the back of the neck and scalp.
- This muscle tension may come from:
    - Not enough rest
    - Bad posture
    - Emotional or mental stress, including depression
    - Anxiety
    - Fatigue
    - Hunger
    - Low iron level

Symptoms

- Mild to moderate pain or pressure in the front, top, or sides of head
- Headache starts later in the day
- Trouble sleeping
- Trouble focusing
- Feeling very tired
- Irritability
- Mild sensitivity to light or noise.

Prevention

- Avoid the causes or triggers
- Manage stress or learn relaxation techniques
- Better posture



The difference between Cluster Headache and Migraine

CHAPTER 3
DISEASES OF EAR

ANATOMY OF EAR

External ear

- Consists of
  - Auricle / pinna
  - External acoustic canal
  - The tympanic membrane

Auricle

- The entire pinna, except its lobule, and the outer part of EAC are made up of a framework of a single piece of yellow elastic cartilage covered with skin.
- There is no cartilage between the tragus and crus of the helix, this area is called the ***incisura terminalis***.

External Auditory Canal

- About 24 mm long
- Divided into two parts:
  - Cartilaginous:
    - Outer one-third (8mm)
    - The skin covering the cartilaginous canal is thick and contains ceruminous and pilosebaceous glands which secrete wax.
    - Hair is only confined to outer canal.
  - Bony
    - Forms inner two-thirds(16mm).
    - Skin is devoid of hair and ceruminous glands.

Tympanic membrane

- Partition between the EAC and the middle ear.
- Obliquely set.
- Parts:
  - Pars tensa
  - Pars flaccida (Sharpnel's membrane)

- Layers of TM:
  - Outer epithelial layer
  - Middle fibrous layer
  - Inner mucosal layer

Middle ear cleft

- Middle ear cleft: consists of a series of interconnected air filled cavities which are located within the temporal bone.
- Middle ear together with the eustachian tube, aditus, antrum and mastoid air cells is called the middle ear cleft.
- Ossicles in the middle ear:
  - The malleus
  - The incus
  - The stapes
- Intratympanic muscles:
  - Tensor tympani
  - Stapedius

Internal ear

- Consists of:
  - Bony labyrinth
  - Membranous labyrinth
- Membranous labyrinth is filled with a clear fluid called ***endolymph***
- The space between membranous and bony labyrinths is filled with ***perilymph***.

Bony labyrinth

- Consists of three parts:
  - Vestibule
  - Semicircular canals
  - Cochlea

Vestibule:

- Central chamber of the labyrinth.
- In its lateral wall lies the oval window.
- The inside of its medial wall presents two recesses
  - Spherical recess: lodges the saccule

    - Elliptical recess: lodges the utricle
  - In the posterosuperior part of vestibule are the five openings of semicircular canals.

Semicircular canals:

  - Three in number: lateral, posterior, superior
  - Lie in planes at right angles to one another
  - Each canal has an ampullated end which opens independently into the vestibule.

Cochlea:

  - The bony cochlea is a coiled tube making 2.5 to 2.75 turns round a central pyramid of bone called the modiolus.
  - The bony cochlea contains three compartments:
    - Scala vestibuli
    - Scala tympani
    - Scala media or the membranous cochlea.

Membranous labyrinth

- Consists of:
  - Cochlear duct / scala media/ membranous cochlea
  - Utricle and saccule
  - Three semicircular ducts
  - Endolymphatic duct and sac

Cochlear duct

  - Blind coiled tube
  - Triangular on cross section and three walls are formed by:
    - The basilar membrane, which supports the organ of corti
    - The Reissner's membrane: separates it from the scala vestibuli

Utricle and saccule:

  - Utricle lies in posterior part of bony vestibule
  - Receives five openings of the three semicircular ducts

- The Saccule also lies in the bony vestibule, anterior to the utricle opposite the stapes footplate.

Semicircular ducts:

- three in number and correspond exactly to the three bony canals.
- They open in the utricle.

Endolymphatic duct and sac:

- Endolymphatic duct is formed by the union of two ducts, one each from the utricle and the saccule.
- Its terminal part is dilated to form the endolymphatic sac.

OTALGIA (EARACHE)

Referred causes:

1. Via Vth cranial nerve	a. Dental : caries, apical abscess, impacted molar, malocclusion. b. Oral cavity: benign or malignant ulcerative lesions of oral cavity or tongue c. TMJ disorders: bruxism, osteoarthritis, recurrent dislocation, ill-fitting denture. d. Sphenopalatine neuralgia.
2. Via IXth cranial nerve	a. Oropharynx: acute tonsillitis, peritonsillar abscess, tonsillectomy b. Base of tongue: tuberculosis or malignancy c. Elongated styloid process
3. Via Xth cranial nerve	Malignancy or ulcerative lesions of: vallecula, epiglottis, larynx, laryngopharynx, oesophagus.
4. Via C2 and C3 spinal nerves	Cervical spondylosis, injuries of cervical spine, caries spine.

Local causes:

External ear
- Furuncle
- Impacted wax
- Otitis externa
- Otomycosis
- Myringitis bullosa
- Herpes zoster
- Malignant neoplasms

Middle ear
- Acute otitis media
- Eustachian tube obstruction
- Mastoiditis
- Extradural abscess
- Aero-otitis media
- Ca middle ear

Psychogenic causes:

- When no cause has been discovered, the pain may be functional in origin but the patient should be kept under observation with periodic re-evaluation.
- Otalgia is a symptom.
- It is essential to find its cause before specific treatment can be instituted.

ACCUTE SUPPURATIVE OTITIS MEDIA

- Acute inflammation of middle ear by pyogenic organisms.

Etiology:

- More common in infants and children of lower socioeconomic group.

- **Routes of infection:**
  - Via eustachian tube: the most common route.
    - Breast or bottle feeding in a young infant in horizontal position may force fluids through the tube into the middle ear
    - Swimming and diving can also force water through the tube into the middle ear.
  - Via external ear: traumatic perforation
  - Blood borne: uncommon
- **Predisposing factors**: anything that interferes with normal functioning of eustachian tube predisposes to middle ear infection. It could be:
  - Recurrent attacks of common cold, URTI, diphtheria, whooping cough
  - Infections of tonsils, adenoids
  - Chronic rhinitis, sinusitis
  - Nasal allergy
  - Cleft palate
- **Bacteriology**:
  - Streptococcus pneumoniae
  - Haemophilus influenza
  - Moraxella catarrhalis

Pathology

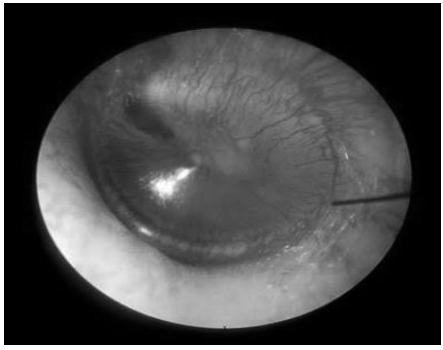
- Tubal occlusion
- Pre-suppuration
- Suppuration
- Resolution
- Complication

1. Tubal occlusion

- Oedema and hyperemia of nasopharyngeal end of eustachian tube blocks the tube, leading to absorption of air and negative intratympanic pressure.
- Retraction of tympanic membrane with some degree of effusion in middle ear but may not be clinically appreciable.
- Symptoms: deafness and earache but not marked.

2. Pre-suppuration

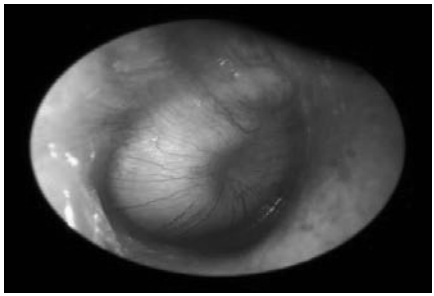
- If tubal occlusion is prolonged, pyogenic organism invade tympanic cavity causing hyperemia of its lining.
- Tympanic membrane becomes congested.
- Symptoms:
  - marked earache which may disturb sleep.
  - Deafness and tinnitus are also present.
- Signs:
  - cart-wheel appearance of tympanic membrane.
- Tuning fork: conductive loss



3. Suppuration

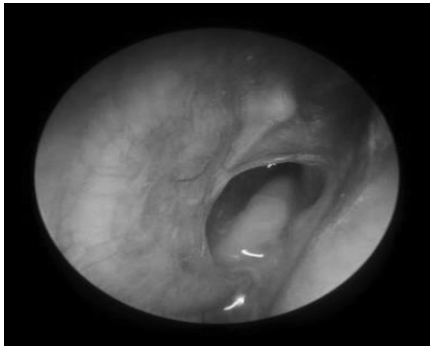
- Formation of pus in the middle ear.
- Tympanic membrane starts bulging to the point of rupture.
- Symptoms:
  - earache becomes excruciating.
  - Deafness increases
  - Fever

- Signs:
  - tympanic membrane appears red and bulging
  - A yellow spot may be seen on the TM where rupture is imminent.



4. Resolution

- The tympanic membrane ruptures with release of pus and subsidence of symptoms.
- Symptoms:
  - with evacuation of pus, earache is relieved.
- Signs:
  - EAC may contain blood-tinged discharge which later becomes mucopurulent.
  - Usually a small perforation is seen in antero-inferior quadrant.



5. Complication

- If virulence of organism is high or resistance of patient is poor, resolution may not take place.
- Acute mastoiditis, facial paralysis, labyrinthitis, meningitis, brain abscess

Treatment

- **Antibacterial therapy**: for a minimum of 10 days, till tympanic membrane regains normal appearance and hearing returns to normal
- **Decongestant nasal drops**: to relieve eustachian tube oedema and promote ventilation of middle ear
- **Analgesics and antipyretics**
- **Ear toilet**: if there is discharge in the ear, it is mopped with sterile cotton bud and a wick moistened with antibiotic may be inserted.
- **Dry local heat** to relieve pain
- **Myringotomy:** incising the drum to evacuate pus, when
  - Drum is bulging and there is acute pain
  - Incomplete resolution despite antibiotics when drum remains full with persistent conductive deafness
  - Persistent effusion beyond 12 weeks.

CHRONIC SUPPURATIVE OTITIS MEDIA (CSOM)

Long standing infection of a part or whole of the middle ear cleft characterized by ear discharge and a permanent perforation

Types:

- **Tubotympanic** (safe/benign):
  - Involves anteroinferior part of middle ear cleft
  - Associated with a central perforation
  - No risk of serious complications
- **Atticoantral** (Unsafe/dangerous):
  - involves posterosuperior part of the cleft
  - Associated with attic or marginal perforation
  - Risk of complications is high

1. Tubotympanic

Aetiology:

- Sequela of ASOM.
- The perforation becomes permanent and permits repeated infection from external ear. Also middle ear mucosa is exposed to the environment and gets sensitized to dust, pollen causing persistent otorrhoea.
- Ascending infection via eustachian tube from tonsils, adenoids, infected sinuses

Clinical features

- Ear discharge:
  - offensive, mucoid / mucopurulent, constant / intermittent.
  - The discharge appears at time of URTI or accidental entry of water into the ear.
- Hearing loss: conductive type
- Perforation: central, may be small, medium or large
- Middle ear mucosa: seen when perforation is large.

Treatment

- Aural toilet: remove discharge from ear.
- Ear drops: antibiotic ear drops combined with steroids
- Systemic antibiotics
- Precautions: patients are instructed to keep water out of the ear during bathing, swimming, hair wash. Hard nose blowing can also push infection from nasopharynx to middle ear and should be avoided.
- Reconstructive surgery: once ear is dry, tympanoplasty can be done.

2. Attico-antral:

- Involves posterosuperior part of middle ear cleft (attic, antrum, posterior tympanum, mastoid)

Clinical features

- Ear discharge: scanty, but always foul smelling due to bone destruction
- Hearing loss
- Bleeding may occur from granulations
- Perforation: attic or posterosuperior marginal type

Treatment

- Surgical
  - MRM (Modified Radical Mastoidectomy)
  - Reconstructive - Tympanoplasty

DEAFNESS

- **Hearing loss** is impairment of hearing and its severity may vary from mild to severe or profound
- The term **deafness** is used when there is little or no hearing at all.
- WHO (1980): the term 'deaf' should be applied to those individuals whose hearing impairment is so severe that they are unable to benefit from any type of amplification.
- The deaf are those in whom the sense of hearing is non-functional for ordinary purposes of life.

Degree of hearing loss (WHO):

- No apparent impairment of hearing from 0-25dB
- Mild : 26-40 dB
- Moderate : 41-55dB
- Moderately severe : 56-70dB
- Severe : 71-91dB
- Profound : more than 91dB
- Total

Types

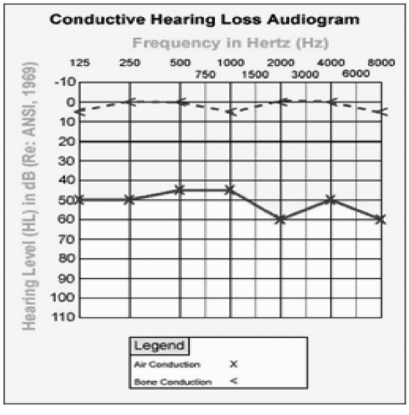
- **Conductive hearing loss**
  - Any disease process which interferes with the conduction of sound to reach cochlea causes conductive hearing loss.

- The lesion may lie in the external ear, TM, middle ear or ossicles.

- **Sensorineural hearing loss (SNHL):**
  - Lesions of cochlea, VIIIth nerve or central auditory pathways

Characteristics

Conductive Hearing loss	**Sensorineural hearing loss**
• Negative Rinne test i.e. BC>AC • Weber lateralized to poorer ear • Low frequencies affected more • Audiometry shows bone conduction better than air conduction with air-bone gap. Greater the air-bone gap, more is the conductive loss. • Loss is not more than 60 dB.	• Positive Rinne test i.e. AC>BC • Weber lateralized to better ear • Bone conduction reduced • More often involving high frequencies • No gap between air and bone conduction curve • Loss may exceed 60dB



Causes of conductive hearing loss:

Congenital causes

- Meatal atresia
- Fixation of stapes foot plate
- Fixation of malleus head
- Ossicular discontinuity
- Congenital cholesteatoma

Acquired causes:

External ear:

- any obstruction in the ear canal e.g. wax, f.b., furuncle, acute inflammatory swelling, benign or malignant tumour or atresia of canal.
- Middle ear:
- Perforation of tympanic membrane – traumatic or infective
- Fluid in the middle ear e.g. acute otitis media, serous otitis media or hemotympanum.
- Mass in middle ear e.g. benign or malignant tumour
- Disruption of ossicles e.g. trauma to the ossicular chain, CSOM, cholesteatoma.
- Fixation of ossicles e.g. otosclerosis, tympanosclerosis, adhesive otitis media.
- Eustachian tube blockage e.g. retracted tympanic membrane, serous otitis media.

Treatment:

- Removal of canal obstructions
- Removal of fluid
- Removal of mass
- Stepedectomy
- Tympanoplasty
- Hearing aid

Causes of SNHL:

- **Congenital**:
  - it is present at birth and is the result of anomalies of the inner ear or damage to the hearing apparatus by prenatal or perinatal factors.
- **Acquired**:
  - Infections of labyrinth – viral, bacterial or spirochaetal.
  - Trauma to labyrinth or 5th nerve, e.g. fractures of temporal bone or concussion of labyrinth or ear surgery.
  - Noise-induced hearing loss
  - Ototoxic drugs
  - Presbycusis
  - Meniere's disease
  - Acoustic neuroma
  - Sudden hearing loss
  - Familial progressive SNHL
  - Systemic disorders, e.g. diabetes, hypothyroidism, kidney disease, autoimmune disorders, multiple sclerosis, blood dyscrasias.

WAX

- Composed of secretion of sebaceous glands, ceruminous glands, hair, desquamated epithelial debris, keratin, dirt.
- Normally a small amount of wax is secreted, which dries up and is later expelled from the meatus by movements of the jaw.
- As some people sweat more than others, the activity of ceruminous glands also varies, excessive wax may be secreted and deposited in the meatus.
- Certain other factors like narrow canal, stiff hair or obstructive lesion of canal may favour retention of wax.

Symptoms

- Impairment of hearing
- Tinnitus

- Giddiness may result from impaction of wax against the TM.
- Reflex cough due to stimulation of auricular branch of vagus may sometimes occur.
- The onset of these symptoms may be sudden when water enters the ear canal during bathing or swimming and the wax swells up.

Treatment

- Removal by syringing or instrumental manipulation.
- Hard impacted wax may require prior softening with wax solvents.

OTOMYCOSIS

- Fungal infection of the ear canal that often occurs due to *Aspergillus niger, A. fumigatus or candida albicans*.
- Seen in hot and humid climate.
- Secondary fungal growth is also seen in patients using topical antibiotics.

Clinical features

- Intense itching
- Discomfort or pain
- Watery discharge
- Ear blockage
- Fungal mass may appear white, brown or black

Treatment

- Thorough ear toilet
- Antifungal agents : Clotrimazole (broad spectrum antifungal agent)
- Ear must be kept dry.

TINNITUS

- Ringing sound or noise in the ear.

- Origin of sound is within patient.
- Usually unilateral, but may also affect both ears.
- It is more annoying in quiet surroundings, particularly at night, when the masking effect of noise from environment is lost.
- **In many cases, the exact cause is never found.**

Types:

- Subjective: can only be heard by patient
- Objective: can even be heard by the examiner.

Cause:

- A common cause of tinnitus is inner ear cell damage.
- Tiny, delicate hair cells move in relation to the pressure of sound waves.This triggers ear cells to release an electrical signal through a nerve from ear to brain.
- If the hairs inside inner ear are bent or broken, they can leak random electrical impulses to brain, causing tinnitus.

Causes of objective tinnitus

- Vascular tumours of middle ear
- Aneurysm of carotid artery
- Palatal myoclonus (rapid spasm of palatal muscles which results in clicking in the ear).

Causes of subjective tinnitus:

Otologic

- Impacted wax
- Fluid in the middle ear
- Acute and chronic OM
- Abnormally patent ET
- Meniere's disease
- Presbyacusis
- Noise trauma
- Tumours of VIII th nerve

Non-otologic

- Diseases of CNS
- Anaemia
- Hypertension
- Hypotension
- Hyoglycaemia
- Migraine
- Epilepsy

- Sometimes tinnitus is psychogenic and no cause can be found in the ear or CNS.

Treatment

- Tinnitus is a symptom, not a disease.
- Where possible, its cause should be discovered and treated.
- When no cause is found, management includes:
    - Reassurance and psychotherapy: many times the patient has to learn to live with tinnitus.
    - Techniques of relaxation and biofeedback
    - Sedation and tranquillizers
    - Masking of tinnitus: at night. Use of a fan, loudly clicking clock or a similar device may mask the tinnitus and help the patient to go to sleep.

MENIERE'S DISEASE (ENDOLYMPHATIC HYDROPS)

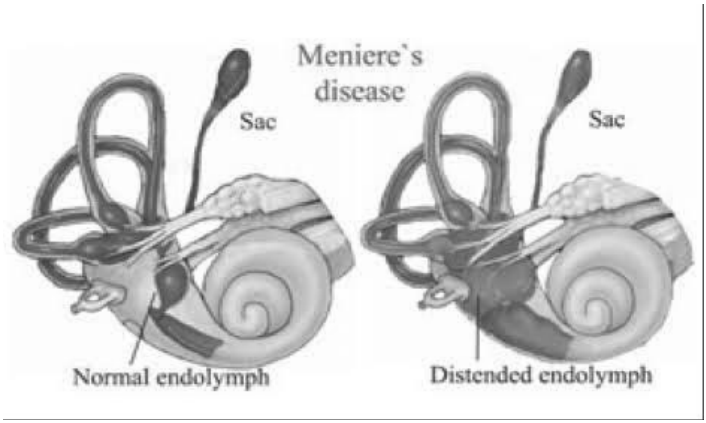
- A disorder of the middle ear where the endolymphatic system is distended with endolymph.
- Characterized by
    - Vertigo
    - Sensorineural hearing loss
    - Tinnitus
    - Aural fullness

Etiology:

- Defective absorption by endolymphatic sac
- Vasomotor disturbance: sympathetic overactivity
- Allergy
- Na and water retention
- Hypothyroidism
- Autoimmune and viral etiologies

Pathology:

- The main pathology is distension of endolymphatic system due to increased volume of endolymph, mainly affecting the cochlear duct (scala media) and the saccule, and to a lesser extent the utricle and semicircular canals.
- The exact cause of Meniere's disease is not yet known.



Clinical features:

- Episodic Vertigo: it comes in attacks. With nausea, vomiting, nystagmus, instability.
  - Visually induced vertigo.
  - Tullio's phenomenon (noice induced vertigo): due to distended saccule against stapes foot plate.
- Hearing loss: progressive, fluctuating SNHL, exacerbates during attacks.
  - Distortion of sound
  - Intolerance to loud sounds
- Tinnitus: fluctuating, low pitched.
- Sense of fullness or pressure
- Emotional upset

Examination:

- Otoscopy: no abnormality is seen in TM.
- Tuning fork tests: indicate SNHL.
- Nystagmus: only during acute attack.

Treatment:

- General measures:
  - Reassurance
  - Cessation of smoking
  - Low salt diet
  - Avoid excessive intake of water
  - Avoid over-indulgence in coffee, tea, alcohol

  - Avoid stress.
  - Avoid activities requiring good body balance
- Management of acute attack:
  - Reassurance
  - Bed rest
  - Vestibular sedatives: to relieve vertigo
  - vasodilators
- Management of chronic phase:
  - Vestibular sedatives: stemetil 10mg thrice a day for 2 months
  - Vasodilators: betahistine (vertin) 8-16 mg
  - Diuretics: furosemide 40mg on alternate days
  - Elimination of allergen
  - Intratympanic gentamicin therapy (chemical labyrinthectomy)
- Sugical
  - Conservative: when vertigo is disabling but hearing is still useful and needs to be preserved.
    - Decompression of endolymphatic sac
    - Endolymphatic shunt operation
    - Sacculotomy (Fick's operation)
    - Section of vestibular nerve
  - Destructive: they totally destroy vestibular and cochlear function.
    - Labyrinthectomy
    - Intermittent low pressure pulse therapy (Meniett device therapy)

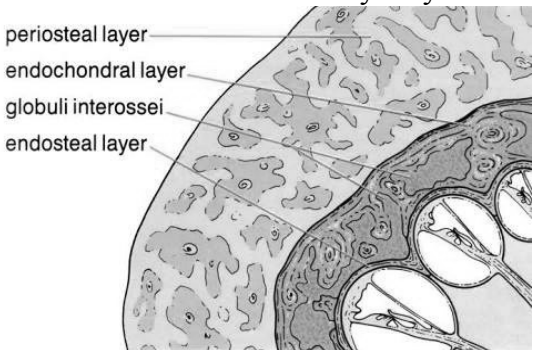
OTOSCLEROSIS

- Otic labyrinth: membranous labyrinth or endolymphatic labyrinth. It is filled with endolymph.
- Periotic labyrinth / perilymphatic labyrinth (or space) : it surrounds the otic labyrinth and filled with perilymph.
- Otic capsule: bony labyrinth.

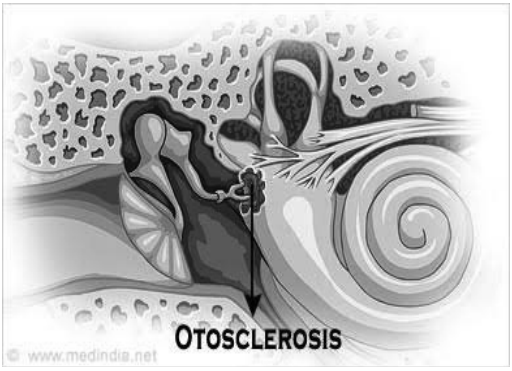
Otic capsule

- It has three layers:
  - Endosteal: innermost

- Enchondral: develops from the cartilage and later ossifies into bone. It is in this layer that some islands of cartilage are left unossified that later give rise to otosclerosis.
- Periosteal: covers the bony labyrinth.



- Primary disease of the bony labyrinth.
- One or more foci of irregularly laid spongy bone replace part of normally dense enchondral layer of bony otic capsule.
- Most often it involves the stapes region leading to stapes fixation and conductive deafness.



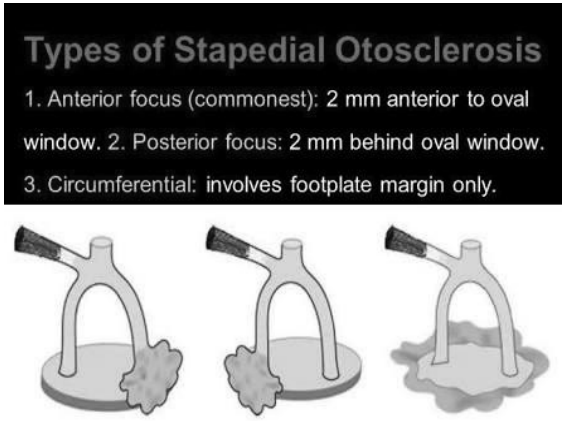
Aetiology

- The exact cause is not known.
- Anatomical basis:
  - Bony labyrinth is made of enchondral bone. Sometimes in this hard bone, there are the areas of cartilage rests which due to certain non-specific factors, are activated to form a new spongy bone.

  - One such area is fissula ante fenestrum lying in front of oval window.
- More common in Indians. Rare among Chinese and Japanese. More common in white than negroes.
- May be associated with Osteogenesis imperfect with h/o multiple fractures.
- Van der hoeve syndrome (Adair Dighton syndrome): otosclerosis, blue sclera, Osteogenesis imperfect.
- Enzymatic theory: imbalance in trypsin/antitrypsin in inner ear fluid.
- Paget's disease
- Measles virus infection

Types

1. Stapedial otosclerosis:
   - Most common variety
   - Causes stapes fixation and conductive deafness



2. Cochlear otosclerosis
   - Involves region of round window or other areas in the otic capsule, and may cause SNHL probably due to liberation of toxic materials into the inner ear fluid.
3. Histologic otosclerosis: Asymptomatic

Symptoms

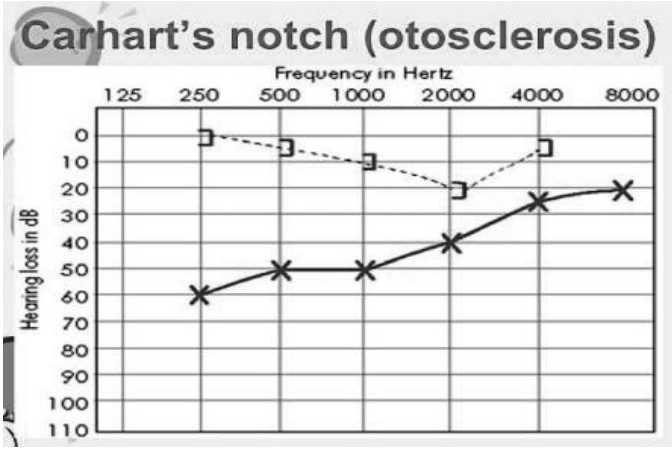
- Deafness: painless, progressive, tends to be bilateral conductive type.
- Paracusis willisii : the patient hears better in noisy than quiet surroundings.
- Tinnitus : more common in cochlear otosclerosis and in active lesions.
- Giddiness: when there is involvement of the bony capsule around the vestibule.
- Speech: monotonous, well modulated speech

Signs:

- Schwartz's sign: reddish hue of tympanic membrane. Indicates active focus with increased vascularity.

Investigations

- Tuning fork tests: reveal conductive deafness.
- Audiometry: audiogram shows a 'Carhart notch' characterised by a slight reduction in the bone conduction at 2000 Hz frequency. This is supposed to be caused by increase in the mass of the foot plate.



Treatment:

- **Medical**: no medical treatment. Sodium fluoride in a dose of 20 mg twice a day with calcium to arrest the rapid progress of otosclerosis.
- **Surgical**: Stapedectomy with a placement of prosthesis. Here the fixed otosclerotic stapes is removed and a prosthesis is inserted between the incus and oval window. Prosthesis employed may be a Teflon piston, stainless steel piston, platinum Teflon or titanium Teflon piston.

VERTIGO

- Vertigo is defined as a false sense of orientation of one's own self in relation to the surrounding environment.
- It is a subjective sensation of imbalance.

Causes:

Peripheral	Central
• Involve vestibular end organs and first order neurons (i.e. vestibular nerve) • The cause lies in the internal ear or the VIIIth nerve.	• Involve central nervous system after the entrance of vestibular nerve in the brainstem.
Peripheral	**Central**
• Meniere's disease • Benign paroxysmal positional vertigo • Vestibular neuronitis • Labyrinthitis • Head trauma • Vestibulotoxic drugs • Syphilis • Acoustic neuroma • Perilymph fistula	• Vertebrobasilar insufficiency • Basilar migraine • Cerebellar disease • Multiple sclerosis • Epilepsy • Tumours of brainstem and fourth ventricle • Cervical vertigo

Investigations: the patient should first be treated symptomatically. After he becomes comfortable, following investigations may be advised.

1. History: past history of injury, diabetes, hypertension, medication, surgery.
2. Examination:
    a. Otoscopy
    b. Blood pressure measurement, examination of CVS and CNS
3. Tests for ear function: Tests of hearing, labyrinthine tests
4. Radiological examination of mastoid, cervical spine and skull.
5. Pathological investigation:
    a. Hemogram
    b. Glucose tolerance test for diabetes
    c. S. cholesterol
    d. V.D.R.L.
6. ECG
7. Neurological investigations like CSF examination, cerebral angiography.

Treatment:

1. Treatment of the cause
2. Assurance: the patient should be assured that giddiness is not dangerous, but it has a nuisance value in majority of cases.

EXAMINATION OF EAR

- Pinna
- Pre-auricular region
- Post-auricular region
- Protrusion of pinna
- Tenderness
- External auditory canal
- Tympanic membrane

Pinna

- Large pinna – macrotia
- Small pinna – microtia
- Absence of pinna – anotia.

- Congenital anomaly:
  - Bat ear (absence of antihelix)
- Thickened or cauliflower pinna is the end result of perichondritis.

Pre-auricular region

- Presence or absence of
  - Pre-auricular sinus
  - Accessory tragus

Post-auricular region

- Any swelling or fistula
- Any lymph node
- Scar of previous surgery
- Palpate the mastoid process:
  - In acute mastoiditis, the periosteum of mastoid process is thickened due to periostitis called ironing of mastoid.

Protrusion of pinna

- Pinna is forward and outward in furunculosis while forward, outward and downward in mastoid abscess (erection of pinna)

Tenderness

- Tenderness at the mastoid process: mastoiditis
- Tenderness of tragus: furunculosis
- Tenderness of pinna: perichondritis

External Auditory Canal

- Pinna is pulled upwards, outwards and backwards to make EAC straight in adults.
- Examine for the presence of wax, fungus, discharge, osteoma, foreign body etc

Tympanic membrane

- Colour: red and congested in ASOM
- Congestion: cartwheel appearance in ASOM.

- Perforation:
  - central/marginal/attic
  - Margin: CSOM has smooth perforation while traumatic perforation has ragged margins.
  - Site:
    - tubotympanic: perforation is due to eustachian tube pathology
    - Atticoantral: pathology lies in attic or mastoid region.
  - Any discharge through perforation

Tuning fork tests

- Rinne's test
- Weber's test
- Absolute bone conduction

TUNING FORK TESTS

Rinne test:

- In this test, the air conduction of the ear is compared with its bone conduction.

- **Procedure**:
  - The tuning fork is set into vibration by striking it against a firm but yielding surface like knee or elbow of the examiner.
  - It is placed near the ear canal for testing AC.
  - When the patient stops hearing, its footplate is transferred to the mastoid behind the ear and BC is tested.
- **Interpretation**:
  - Rinne positive: AC is better than BC. (AC>BC)
  - Rinne negative: BC is greater than AC. (BC>AC)
  - Rinne equivocal: AC and BC are equal.
  - False rinne negative: AC and BC are markedly reduced on diseased side, but the patient may still indicate good BC on the diseased side. It is due to

crossing of the sound from the affected ear to the normal ear through the skull, and the patient really hears through the opposite normal ear.

- Rinne positive indicates normal hearing or sensori-neural hearing loss.
- Rinne negative indicates conductive hearing loss
- Rinne equivocal indicates very mild conductive hearing loss
- False rinne negative indicates severe sensori-neural hearing loss

Weber test:

- It compares the BC of the two ears.
- **Procedure**:
  - A vibrating tuning fork is placed in the middle of the forehead or the vertex or on the upper incisors.
  - The patient is asked in which ear the sound is heard.
  - The sound travels directly to the cochlea via bone.
  - Normally it is heard equally in both ears.

- **Interpretation**:
  - Conductive hearing loss: in unilateral conductive hearing loss, the sound lateralizes to the affected ear as BC is better on that side.
  - Sensori-neural hearing loss: the sound lateralizes to the better ear.

Absolute bone conduction (ABC) test:

- This test is a measure of inner ear function.
- The patient's BC is compared with that of the examiner (presuming that the examiner has normal hearing.)
- Thus one can detect the SNHL in a patient.
- **Procedure**:
  - The ear canal is blocked by a finger.

    - The vibrating tuning fork is placed on the mastoid of the patient.
    - As soon as he stops hearing, it is transferred to the mastoid of the examiner.
- **Interpretation**:
    - Conductive loss: ABC is same as that of the examiner.
    - SNHL: ABC of the patient is less than ABC of the examiner.

CHAPTER 4
DISEASES OF NOSE

ANATOMY OF THE NOSE

External Nose:

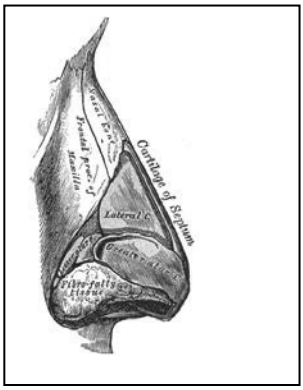
- Pyramidal shape with its root up & base downwards.

OSTEOCARTILAGINOUS FRAMEWORK:

Bony part	• Upper 1/3rd of the external nose. • Consists of 2 nasal bones which meet in the midline and rest on the upper part of the nasal process of the frontal bones and are themselves held between the frontal processes of the maxillae.
Cartilaginous part	1. **Upper lateral cartilages:** Extend from undersurface of of the nasal bones above, to the alar cartilages below. Its lower free edge is seen intranasally as limen vestibule or nasal valve on each side. 2. **Lower lateral cartilages (Alar cartilages):** U-shaped. It has **a lateral crus** which forms the ala and **a medial crus** which runs in columella. 3. **Lesser Alar (sesamoid) cartilages:** 2 or more in number. They lie above & lateral to alar cartilages. 4. **Septal cartilages:** Its anterosuperior border runs from under the nasal bones to the nasal tip. It supports the dorsum of the cartilaginous part of the nose.

NASAL MUSCULATURE:

1. Procerus
2. Nasalis
3. Levator labii superioris alaeque nasi
4. Anterior & posterior dilator nares
5. Depressor septi



NASAL SKIN:

The skin over the nasal bones & upper lateral cartilages : Thin, freely mobile The skin covering the alar cartilages : Thick, adherent, contains many sebaceous glands

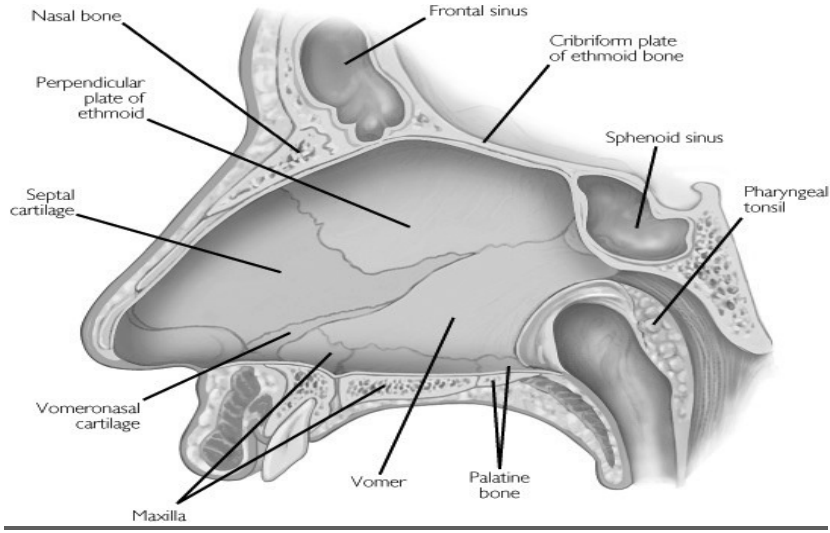
Internal Nose

- Divided into right & left nasal cavities by nasal septum
- Each cavity consists of
  - a skin lined portion- the ***vestibule*** and
  - a mucosa lined portion- the ***nasal cavity proper***

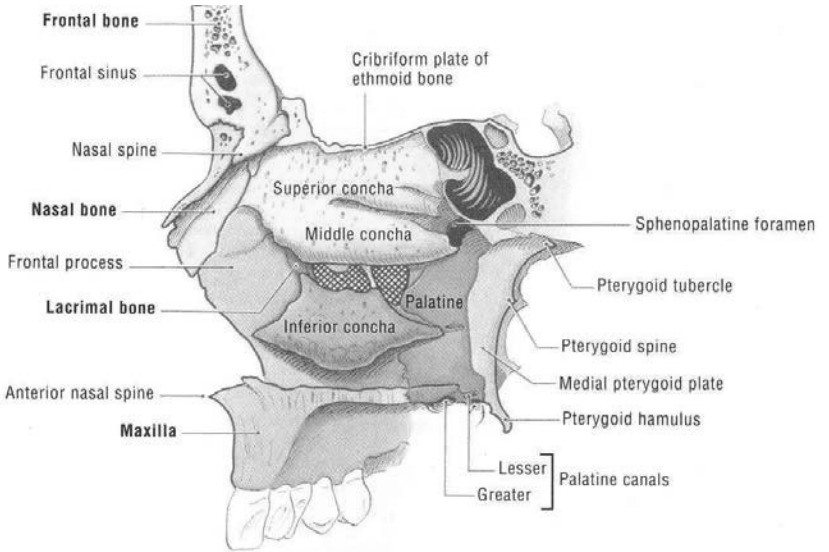
Vestibule	• Anterior & inferior part of nasal cavity • Lined by skin • Contains sebaceous glands, hair follicles & hair called vibrissae. • -Its upper limit on the lateral wall is marked by limen nasi(nasal valve) - Its medial wall is formed by the columella & lower part of the nasal septum upto its mucocutaneous junction
Nasal cavity proper	**Lateral Nasal wall:** • 3 or occasionally 4 turbinates or conchae

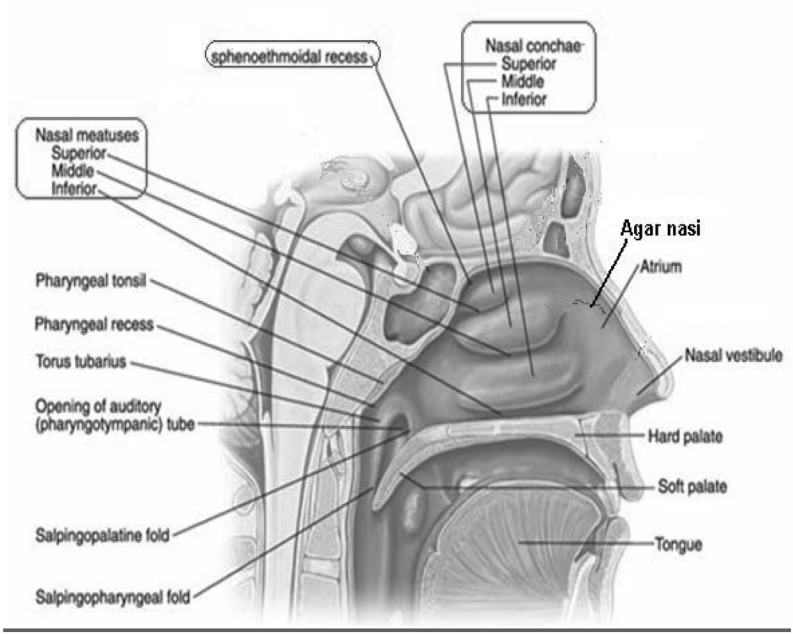
	which are scroll like bony projections covered by mucous membrane. • The space below turbinates are called meatuses. **Medial wall:** • Formed by nasal septum **Roof:** • Anterior sloping part is formed by nasal bones • Posterior sloping part is formed by the body of sphenoid bone • Middle horizontal part is formed by the cribriform plate of ethmoid through which the olfactory nerves enter the nasal cavity. **Floor:** • Anterior 3/4th is formed by palatine process of the maxilla • Posterior 1/4th by the horizontal part of the palatine bone

MEDIAL WALL OR SEPTUM:



LATERAL WALL:





LINING MEMBRANE OF INTERNAL NOSE:

VESTIBULE	LINED BY SKIN CONTAINING HAIR, HAIR FOLLICLES & SEBACEOUS GLANDS.
OLFACTORY REGION	• FORMED BY UPPER 1/3RD OF LATERAL WALL, CORRESPONDING PART OF NASAL SEPTUM AND THE ROOF OF NASAL CAVITY • MUCOUS MEMBRANE IS PALER HERE.
RESPIRATORY REGION	• FORMED BY LOWER 2/3RDS • MUCOUS MEMBRANE SHOWS VARIABLE THICKNESS -THICKEST OVER NASAL CONCHAE - QUITE THICK OVER THE

	NASAL SEPTUM - VERY THIN IN THE MEATUSES AND FLOOR OF THE NOSE. • HIGHLY VASCULAR & ALSO CONTAINS ERECTILE TISSUE • ITS SURFACE IS LINED BY PSEUDOSTRATIFIED CILIATED COLUMNAR EPITHELIUM WHICH CONTAINS PLENTY OF GOBLET CELLS • IN THE SUBMUCOUS LAYER OF THE MUCOUS MEMBRANE, ARE SITUATED SEROUS, MUCOUS, BOTH SEROUS AND MUCOUS SECRETING GLANDS, THE DUCTS OF WHICH OPEN ON THE SURFACE OF MUCOSA.

NERVE SUPPLY:

- Nasal cavity receives sensory & visceral innervation

Sensory innervation

- Olfactory mucosa supplied by olfactory nerves.
- Nerves of general sensation are derived from opthalmic & maxillary nerves
- Anterior part supplied by the anterior ethmoidal nerve (branch of opthalmic nerve)
- Posterior part supplied by nasal, nasopalatine and palatine branches (of maxillary nerve)

Visceral Innervation

- Sympathetic fibers arise from neurons of superior cervical ganglion and are distributed through plexuses around the arteries, supply mainly vascular smooth muscle

- Parasympathetic fibers arise from neurons of the pterygopalatine ganglion that course in the nasopalatine nerve (branch of maxillary) and its branches, supply the mucosal glands.

ARTERIAL SUPPLY:

- Sphenopalatine artery (branch of the maxillary artery) is the main supply
- Alar and septal branches of superior labial artery (branch of the facial artery)
- Anterior & posterior ethmoidal arteries (branches of the ophthalmic artery)

VENOUS DRAINAGE:

- Veins begin as a rich plexus in the submucosa, accompany the corresponding arteries, and drain into the facial, ophthalmic, and sphenopalatine veins.

LYMPHATIC DRAINAGE:

The lymphatics from the:

- Vestibule drain into the submandibular lymph nodes
- Rest of the cavity drains into the upper deep cervical lymph nodes

DEVIATED NASAL SEPTUM (DNS)

Etiology:

- Trauma
    - A lateral blow on the nose may cause displacement of septal cartilage from the vomerine groove and maxillary crest
    - A crushing blow from the front may cause buckling, twisting, fractures, duplication of nasal septum
    - Birth injuries
- Developmental error
    - The septum should grow at the same rate as that of the face. If the septum starts growing at a rapid rate, it becomes buckled to accommodate itself.
    - Unequal growth between the palate and the base of skull may cause buckling of the nasal septum.
    - In mouth breathers, as in adenoid hypertrophy, the palate is often highly arched and the septum is deviated.
- congenital
    - abnormal intrauterine posture.

Types:

- **Anterior dislocation**: septal cartilage may be dislocated into one of the nasal chambers.
- **C-shaped deformity**:
    - Septum is deviated in a simple curve to one side.
    - Nasal chamber on the concave side of the nasal septum will be wider and may show compensatory hypertrophy of turbinates.
- **S-shaped deformity:** in vertical or antero-posterior plane. It causes bilateral nasal obstruction.
- **Spurs:**
    - A shelf like projection often found at the junction of bone and cartilage.
    - A spur may press on the lateral wall and gives rise to headache.

  - It may also predispose to repeated epistaxis from the vessels stretched on its convex surface.
- **Thickening**: it may be due to organised hematoma.

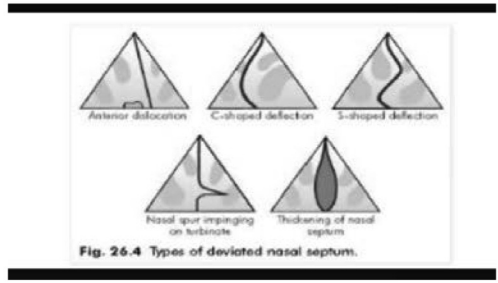


Fig. 26.4 Types of deviated nasal septum.

Symptoms:

- Nasal obstruction: unilateral or bilateral
- Headache : due to different causes:
  - Sinusitis secondary to the obstruction to the drainage
  - Vacuum headache: vacuum created in PNS due to obstruction to the drainage of sinus, resulting in absorption of air by the mucosa of sinus.
  - Neuralgic headache: when septum touches a sensitive part on the lateral wall of the nose.
- Epistaxis : Little's area on deviated side of the septum is exposed to the atmosphere directly, predisposing to drying and crusting of mucosa and tendency to pick the nose.
- Anosmia is rare.
- Middle ear infection
- External deformity

Deviation of the septum occurs very frequently but it requires treatment only if it produces symptoms.

Treatment:

- Submucous resection (SMR)
- Septoplasty
- Septal surgery is usually done after the age of 17 so as not to interfere with the growth of nasal skeleton.

EPISTAXIS

Causes:

Local causes:

In Nose:

- **Trauma**: finger nail, intranasal surgery, fractures of middle third of face and base of skull, hard blowing of nose, violent sneeze
- **Infections**:
  - Acute: viral rhinitis, nasal diphtheria, acute sinusitis
  - Chronic: atrophic rhinitis, rhinitis sicca, tuberculosis, syphilis septal perforation, granulomatous lesion of the nose e.g. rhinosporidiosis.
- **Foreign bodies**
- **Neoplasms** of nose and PNSs
  Benign: haemangioma, papilloma
  Malignant: carcinoma/sarcoma
- **Atmospheric changes**: high altitudes
- **DNS**

In Nasopharynx:

- Adenoiditis
- Juvenile angiofibroma
- Malignant tumours

General Causes:

- **Cardiovascular system**:

Hypertension, arteriosclerosis, mitral stenosis, pregnancy

- **Disorders of blood & blood vessels**:

Aplastic anaemia, leukaemia, haemophilia, scurvy, Vit. K deficiency

- **Liver diseases**: hepatic cirrhosis
- **Kidney diseases**: Chronic nephritis
- **Acute general infection**:

Influenza, measles, chicken pox, whooping cough, rheumatic fever, typhoid, pneumonia, malaria, dengue fever

- **Drugs**:

Excessive use of salicylates and other analgesics (as for joint pains), Anticoagulant therapy (for heart dis.)

- **Mediastinal compression**: tumours of mediastinum.

Common Sites:

1. Nasal septum: little's area accounts for almost 90% of cases.
2. Above the middle turbinate: Anterior ethmoidal vessels may bleed due to hypertension.
3. Below the level of middle turbinate: branches of sphenopalatine artery.

Types:

- Anterior Epistaxis: blood flows out of the nose with the patient in sitting position.
- Posterior Epistaxis: Mainly the blood flows back into the throat. Patient may swallow it and later have a coffee-coloured vomitus.

	Anterior Epistaxis	Posterior Epistaxis
Incidence	More common	Less common
Localization	Easy	Difficult
Common site	Little's area	Woodruff plexus
Age	< 18 yr	> 40 yr
Common Cause	Trauma	Hypertension
Treatment	Anterior pack	Posterior pack

Investigations:

- B.P.
- Haemogram
- Tests for coagulation: to detect bleeding disorder
- Radiography: Acute sinusitis, fracture or malignancy of PNS may be detected

- CT Scan
- Endoscopy of nose and PNS

Treatment:

- First aid: pinching the nose with thumb and index finger for about 5 minutes. Cold compress should be applied to the nose to cause reflex vasoconstriction.
- Cauterization: when bleeding point has been located.
- Anterior nasal packing: the whole nasal cavity is packed by layering the gauze from floor to roof and from before backwards.
- Posterior nasal packing: required for the patients bleeding posteriorly into the throat.
- Ligation of vessels

NASAL POLYP

- Non-neoplastic masses of oedematous nasal or sinus mucosa.
- Hypertrophied, Edematous, prolapsed mucosa of nose and paranasal sinuses.

PROPERTIES OF NASAL POLYPI

Gray in color
Smooth surface
Pedunculated
Mobile
Insensitive to pain
Not bleed on probing

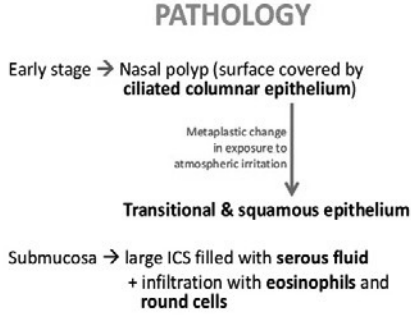
- 2 main varieties:
  - Antrochoanal
  - Ethmoidal

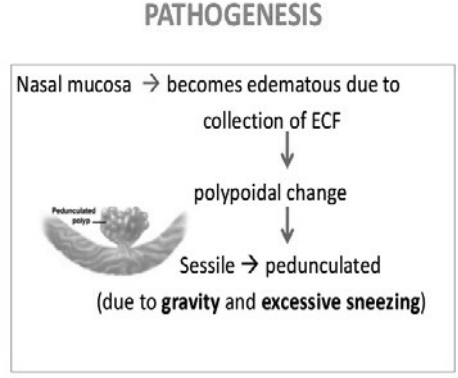
Aetiology:

- Not well understood.
- May arise in
  - inflammatory conditions of nasal mucosa
  - Disorders of ciliary motility

  - Abnormal composition of nasal mucus (Cystic fibrosis).
- Various diseases associated with the formation of nasal polypi are:
  - **Chronic rhinosinusitis**: both allaergic and non-allergic origin
  - **Aspirin intolerance**: 36% of the patients with aspirin intolerance may show polypi. (Sampter's triad: nasal polypi, asthma, aspirin intolerance)
  - **Cystic fibrosis**: 20% of patients with cystic fibrosis form polypi.
  - **Allergic fungal sinusitis**

- Consequence of damage in the epithelium of nasal mucosa.
- Injuries may be caused by bacterial or viral infection as well as prolonged inhalation of irritating substances.
- Damaged mucosa always tends to heal the injury through the migration of the epithelium.
- In some cases, a regeneration of the nasal epithelium is insufficient.
- A regeneration of nasal epithelium releasing cytokines, which activate inflammatory cells, may lead to the formation of nasal polyp.





PATHOGENESIS:

- Nasal mucosa, particularly in the region of middle meatus and turbinate becomes oedematous due causing polypoidal change.
- Polypi which are sessile in the beginning become pedunculated due to gravity and the excessive sneezing.

PATHOLOGY:

- In early stage, surface of nasal polypi is covered by ciliated columnar epithelium like that of normal nasal mucosa but later it undergoes a metaplastic change to transitional and squamous type on exposure to atmospheric irritation.

- Submucosa shows large intercellular spaces filled with serous fluid.
- There is also infiltration with eosinophils and round cells.

Antrochoanal

- Common in children
- Aetiology: Infection
- Number: Solitery
- Unilateral
- Origin: Maxillary sinus near the ostium
- Grows backwards to the choana; may hang down behind the soft palate

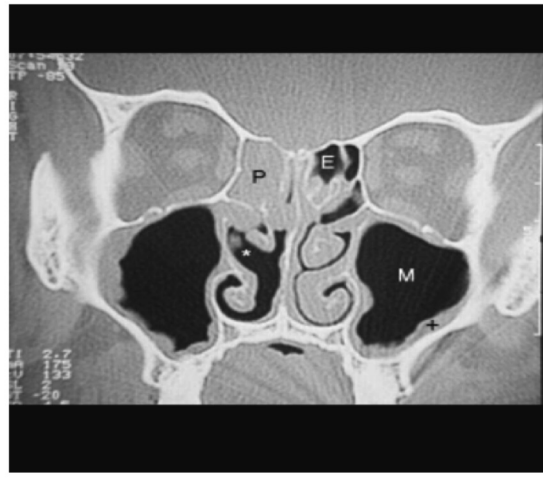
Ethmoidal

- Common in adults
- Aetiology: Allergy
- Multile
- Bilateral
- Origin: Ethmoidal sinuses, middle turbinate, middle meatus
- Mostly grow anteriorly and may present at the nares

Ethmoidal Polyp

- **Symptoms:**
  - Unilateral or bilateral total nasal obstruction
  - Partial or total anosmia
  - Headache due to blockage of sinus osteam
  - Sneezing and watery nasal discharge due to asso. Allergy
  - Mass protruding from nose

- **Signs:**
  On anterior rhinoscopy:
- Smooth ,pale , grape like multiple masses in nasal cavity
- Insensitive to probing or painless
- Not bleed on touch
- Often bilateral



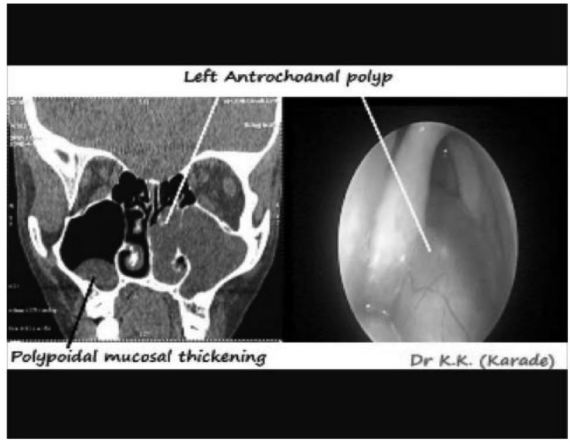
Treatment:

- Polypectomy
- FESS

Antrochoanal polyp

- This polyp arises from the mucosa of maxillary antrum, comes out of it and grows in the choana and nasal cavity.
- **Aetiology:**
  - Exact cause is unknown
  - Chronic maxillary sinusitis
  - Nasal allergy with sinus infection
  - Impaired maxillary sinus drainage.
  - Seen in children and young adults
  - Single, unilateral
- **Symptoms:**
  - Unilateral nasal obstruction. May become bilateral when polyp grows into nasopharynx and starts obstructing the opposite choana.
  - Voice may become thick and dull
  - Nasal discharge: mucoid
- **Signs:**
  - As it grows posteriorly, it may be missed on anterior rhinoscopy
  - When large, a smooth greyish mass covered with nasal discharge may be seen.

- Soft, can be moved up and down with a probe.
- Posterior rhinoscopy may reveal a globular mass filling choana or nasopharynx.



- **Differential Diagnosis:**
  - A blob of mucus often looks like a polyp but it would disappear on blowing the nose.
  - Hypertrophied turbinate is differentiated by its pink appearance and hard feel of bone on probe testing.
  - Angiofibroma: H/o profuse recurrent epistaxis, firm in consistency, easily bleeds on probing.
- Reasons for posterior migration of AC polyp:
  - Inspiratory air current is more powerful than expiratory current, there by pushing the polyp posteriorly
  - The natural slope of nasal cavity is directed posteriorly
  - Cilia beats towards the choana.

Diagnosis:

- DNE
- X-ray PNS
- CT Scan PNS

Treatment:

- Polypectomy
- FESS

RHINITIS

Irritation and inflammation of the mucus membrane inside the nose.

Acute

- Viral
- Bacterial
- Irritative

Chronic:

- Chronic simple
- Hypertrophic
- Atrophic
- Rhinitis Sicca
- Rhinitis Caseosa

Viral Rhinitis
1. Common cold

Aetiology

caused by a virus (adeno virus, picorna virus, rhinovirus, coxsackie)

•Infection is usually contracted through airborne droplets.

•Incubation period : 1-4 days

•Illness lasts for 2-3 weeks.

Clinical features:

•Burning sensation at the back of nose soon followed by nasal stuffiness, rhinorrhoea, sneezing

•Low grade fever with chills

•Initially, nasal discharge is watery, profuse but may become mucopurulent due to secondary bacterial invasion.

The disease is usually self limiting and resolves spontaneously after 2-3 weeks.

Treatment :

•Bed rest

•Plenty of fluids

•Antihistaminics and nasal decongestants

•Analgesics to relieve headache, fever, myalgia

•Antibiotics when secondary infection supervenes.

Bacterial Rhinitis

Non-specific infections:

Primary/Secondry

1. Primary: seen in children and usually the result of infection with pneumococcus, streptococcus or staphylococcus.
   A greyish white tenacious membrane may form in the nose, which causes bleeding on attempt of removal.
2. Secondary is the result of bacterial infection supervening acute viral rhinitis.

Diphtheritic Rhinitis:

•Diphtheria of nose is rare these days.
•A greyish membrane is seen covering the inferior turbinate and the floor of the nose.
•Membrane is tenacious and removal causes bleeding
•Excoriation of anterior nares and upper lip
•Treatment:
- •Isolation of the patient
- •Systemic penicillin
- •Diphtheria antitoxin

Chronic Simple Rhinitis

- Recurrent attacks of acute rhinitis in the presence of predisposing factors lead to chronicity.
  - Persistence of nasal infection due to sinusitis, tonsillitis, adenoids
  - Chronic irritation from dust, smoke, smoking
  - Nasal obstruction due to DNS, synechia leading to persistence of discharge in the nose
  - Vasomotor rhinitis
  - Endocrinal/metabolic factors e.g. hypothyroidism, excessive intake of carbohydrates, lack of exercise.

Symptoms:

- **Nasal obstruction**
- **Nasal discharge** : mucoid/mucopurulent, thick, viscid, often trickles into the throat as post-nasal drip. Patient has a constant desire to blow the nose and clear the throat.
- **Headache** – due to swollen turbinates impinging on the septum
- **Swollen turbinates** – they pit on pressure and shrink with application of vasoconstrictor drops.
- **Post-nasal discharge** – seen on the PPW.

Treatment:

•**Treat the cause** : allergy, personal habits, environment or work situation (smoky or dusty surroundings)
•**Nasal irrigations** with alkaline solution help to keep the nose free from viscid secretions and also remove superficial infection
•**Nasal decongestants** help to relieve the nasal obstruction and improve sinus ventilation.
•**Antibiotic**s help to clear nasal infection.

Hypertrophic Rhinitis

Thickening of mucosa, submucosa, seromucinous glands, periosteum and bone.

Aetiology:
Recurrent nasal infections, chronic sinusitis, chronic irritation of nasal mucosa due to smoking, industrial irritants, prolonged use of nasal drops

Symptoms:
•Nasal obstruction – predominant symptom
•Nasal discharge – thick, sticky
•Headache, heaviness of head, transient anosmia

Signs:
•Hypertrophy of turbinates - turbinal mucosa is thick and does not pit on pressure
•Little shrinkage with vasoconstrictor drugs due to presence of underlying fibrosis

Treatment:
•Discover the cause and remove it.
•Nasal obstruction can be relieved by reduction in the size of turbinates.
- •Linear cauterization
- •Submucosal diathermy
- •Cryosurgery of turbinates.
- •Turbinectomy- partial or total

ATROPHIC RHINITIS

- Chronic inflammation of nose characterized by atrophy of nasal mucosa and turbinate bones.
- The nasal cavities are roomy and full of foul smelling crusts.

Aetiology:

1. Hereditary factors
2. Endocrinal disturbance
3. Racial factors: white and yellow races
4. Nutritional deficiency: deficiency of Vit. A,D or iron or some other dietary factors.
5. Infective: Klebsiella ozaenae, diphtheroids, Perez vulgaris, Esch. Coli, staphylococci, streptococci
6. Autoimmune process: the body reacts by a destructive process to the antigens released from the nasal mucosa.

Pathology:

- Metaplasia of ciliated columnar nasal epithelium into squamous epithelium.
- Mucosa and submucosa undergo atrophy
- Atrophy of cilia and mucosal glands resulting in a pale mucosa with thick scanty secretions.
- Turbinates undergo atrophy producing wide roomy cavity
- Blood vessels are affected by periarteritis and endarteritis obliterans.
- Nerves become atrophied. There is also atrophy of olfactory nerves as well and patient loses the sense of smell.

Clinical picture

- Usually seen in females, starts around puberty.
- Foul smell from the nose, patient himself is unaware of smell due to marked anosmia (merciful anosmia).
- Patient may complain of nasal obstruction inspite of unduly wide nasal chambers. This is due to large crusts filling the nose.
- Epistaxis may occur when crusts are removed.

Examination:

- Nasal cavity full of greenish or greyish black dry crusts covering the turbinates and septum.
- Attempts to remove them may cause bleeding. When removed, nasal cavities appear roomy with atrophy of turbinates so much that the posterior wall of nasopharynx can be easily seen.
- Nasal mucosa appears pale. Septal perforation or dermatitis of vestibule may be present.
- Atrophic changes may be seen in the pharyngeal mucosa and larynx.

Treatment:

- Medical:
  - Nasal irrigation and removal of crusts: soda bicarbonate 1 part, sodium biborate 1 part, sodium

chloride 2 parts in 280ml water, is used to irrigate the nasal cavities. The solution is run through one nostril and comes out from the other. It loosens the crusts and removes the thick tenacious discharge.

- 25% glucose in glycerine: after crusts are removed, nose is painted with 25% glucose in gycerine. This inhibits the growth of proteolytic organisms which are responsible for foul smell.
- Local antibiotics: to eliminate secondary infection
- Oestradiol spray: to increase the vascularity of nasal mucosa and regeneration of ceruminous glands.

- Surgical:
  1. Young's operation: both the nostrils are closed completely just within the nasal vestibule by raising flaps. They are opened after 6 months or later. In these cases mucosa may revert to normal and crusting reduced.

  Modified young's operation: aims to partially close the nostrils.

  2. Narrowing the nasal cavities:
     1. Submucosal injection of teflon paste
     2. Insertion of fat, cartilage, bone or teflon strips under the mucoperiosteum of the floor and lateral wall of the nose and mucoperichondriom of the seeptum.

Rhinitis Sicca:

- Crust forming disease seen in the patients working in hot, dry, dusty surroundings
- Confined to anterior 1/3 of nose particularly of septum
- Ciliated columnar epithelium undergoes squamous metaplasia with atrophy of seromucinous glands.
- Crusts form on the anterior part of septum and their removal causes ulceration and epistaxis, may lead to septal perforation.
- Treatment: correction of occupational surroundings and application of ointment with an antibiotic or steroid to the affected part.

Rhinitis Caseosa

- Uncommon, usually unilateral, mostly affecting males.
- Nose is filled with offensive purulent discharge and cheesy material.
- The disease possibly arises from chronic sinustis with collection of inspissated cheesy material.
- Sinus mucosa becomes granulomatous, bony walls of sinus may be destroyed.
- Treatment is removal of debris and granulation tissue and free drainage of the affected sinus.

Allergic Rhinitis

- An Ig-E mediated immunologic response of nasal mucosa to air-borne allergens
- Characterized by watery nasal discharge, nasal obstruction, sneezing, itching in the nose.
- May be associated with symptoms of itching in the eyes, palate and pharynx.

Types:

- Seasonal: symptoms appear in or around a particular season when the pollens of particular plant, to which the patient is sensitive, are present in the air.
- Perennial: symptoms are present throughout the year.

Pathogenesis

- Inhaled allergens
- Produce specific IgE antibody in genetically predisposed individuals

- This antibody becomes fixed to the blood basophils or tissue mast cells by its FC end.
- On subsequent exposure, the antigen combines with IgE antibody at its Fab end.
- This reaction produces degranulation of mast cells with release of several chemical mediators which are responsible for the symptomatology of allegic disease.

Seasonal

- Paroxysmal sneezing
- 10-20 sneezes at a time
- Nasal obstruction
- Watery discharge
- Itching in the nose
- Itching may also involve eyes, palate or pharynx

Perennial

- Not so severe as that of the seasonal type
- Frequent colds
- Persistently stuffy nose
- Loss of sensation of smell due to mucosal oedema
- Postnasal drip
- Chronic cough
- Hearing impairment due to eustachian tube blockage or fluid in the middle ear.

Signs:

Nasal Signs:

1. Pale and edematous mucosa which may appear bluish
2. Turbinates are swollen
3. Thin, watery, mucoid discharge

Ocular signs:

1. Oedema of lids
2. Congestion and cobble-stone appearance of conjunctiva
3. Dark circles under the eyes

Otologic Signs:

1. Retracted TM
2. Serous Otitis Media due to ET blockage

Pharyngreal Signs:

1. Granular pharyngitis due to hyperplasia of submucosal lymphoid tissue

Laryngeal Signs:

1. Hoarseness of voice
2. Oedema of vocal cords

Treatment:

- Avoidance of allergen
- Treatment with drugs:
  - **Antihistaminics**: they control rhinorrhoea, sneezing and pruritis
  - **Sympathomimetic drugs** (oral/topical): alpha adrenergic drugs constrict blood vessels and reduce nasal congestion and oedema. Pseudoephidrine and phenylpropanolamine
  - **Topical**: for nasal decongestion. Phenylephrine, oxyetazoline, xylometazoline are used to relieve nasal obstruction.
  - **Corticosteroids**: Oral steroids are very effective but their use should be limited to acute episodes which have not been controlled by other measures.
  - **Topical steroids**: beclomethasone dipropionate, budesonide, fluticasone, mometasone
  - **Sodium cromoglycate**: used as 2% solution for nasal drops / spray

VASOMOTOR RHINITIS

- Non-allergic rhinitis but clinically simulating nasal allergy with symptoms of nasal obstruction, rhinorrhoea and sneezing.
- The condition usually persists throughout the year and all the tests for nasal allergy are negative.

- Sympathetic stimulation : Vasoconstriction, shrinkage of mucosa
- Parasympathetic stimulation : Vasodilation, engorgement
- Autonomic nervous system is under control of hypothalamus and therefore emotions play a great role in vasomotor rhinitis.
- Autonomic nervous system is unstable in cases of vasomotor rhinitis.
- Nasal mucosa is also hyperactive and responds to several non-specific stimuli, e.g. change in temperature, humidity, blasts of air, small amounts of dust or smoke.

Symptoms:

- Paroxysmal sneezing: bouts of sneezing start just after getting out of the bed in the morning.
- Excessive rhinorrhoea: profuse and watery.
- Nasal obstruction: alternates from side to side. More marked at night.
- Postnasal drip

Treatment:

- Avoidance of physical factors which provoke symptoms e.g. sudden change in temperature, humidity, dust etc
- Antihistaminics, oral nasal decongestants
- Topical steroids
- Systemic steroids in very severe cases
- Psychological factors should be removed. Tranquillizers may be needed.

SINUSITIS

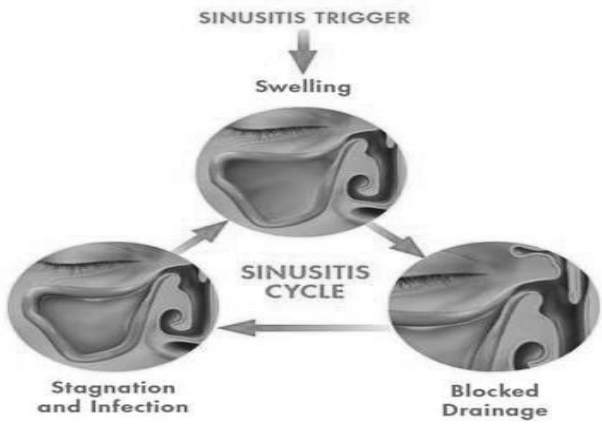
- Inflammation of sinus mucosa
- Maxillary sinus is frequently infected PNS followed in turn by ethmoid, frontal, sphenoid.
- This may be due to its close proximity to teeth and due to inadequate drainage because of higher level of ostium.

Predisposing factors

- Nasal infection:
  - Common cold, influenza. Following factors help the spread of the infection from nose.
    - Nasal obstruction: DNS, turbinate hypertrophy, polyp etc
    - Nasal allergy
    - Habits: Forcible blowing of the nose pushes the infection from the nose into the sinuses.
    - Swimming: when water enters the nose forcibly, infection may spread to the sinuses.
    - Barotrauma
- Dental infections: Infection from upper molar and premolar teeth or their extraction may be followed by acute sinusitis.
- Trauma: results in collection of blood in its cavity and may become secondarily infected.

Pathogenesis:

- Obstruction of natural ostia : most common pathologic process.
- Hypoxygenation
- Ciliary dysfunction
- Retention of secretion



Types:

- Acute Sinusitis : lasting less than 1 month
- Subacute Sinusitis : lasting 1 to 3 months
- Chronic Sinusitis: lasting more than 3 months.

- Acute and subacute sinusitis are usually treated medically
- Chronic sinusitis is considered irreversible by medical therapy alone. Oxygenation of sinuses through the opening of the ostia is the primary treatment.

Acute Maxillary Sinusitis

- Pain in maxillary region: May radiate to teeth, eyes, ears, frontal sinus. Aggravated on bending down, coughing, sneezing.
- Nasal discharge: mucoid initially, soon becomes purulent.
- Post nasal discharge
- Constitutional symptoms: Patient may have malaise, headache, fever

Investigation:

- Posture test
- Radiograph
  - X-ray PNS Water's view
  - CT scan PNS

Treatment:

- General:
  - Antibiotics
  - Nasal decongestant drops : 1% ephidrine or 0.1% xylometazoline
  - Analgesics
  - Steam inhalation
  - Hot fomentation

Proof Puncture (Antral irrigation)

- Lateral wall of inferior meatus is punctured with trocar and cannula near the attachment of concha with lateral wall.

- The antrum is irrigated with NS.
- Syringing is continued till return is clear.

Caldwell-Luc Surgery:

- Process of opening the maxillary antrum through canine fossa by sublabial approach.
- Incision is made below the gingivobuccal sulcus, from lateral incisor to the 2nd molar.
- Elevation of flap
- Opening the antrum: using cutting burr, a hole is made in the antrum.
- Pathology is removed: diseased mucosa is removed with elevators, curettes, forceps. Cysts, polyp, tumour is removed.
- Packing the antrum
- Closure of wound.

Acute Frontal Sinusitis

Clinical features:

- Pain in frontal region: It comes up in the morning , gradually increases and reaches its peak by about mid day, then starts subsiding. Also called Office headache.
- Tenderness: above the inner canthus of eye.
- Oedema of the upper eyelid is rare.

Frontal sinus Trephination

- A 2 cm long incision is made just above the medial end of eyebrow.
- Floor of frontal sinus is exposed and a hole drilled with a burr.
- Plastic drainage tube is inserted and fixed.
- Sinus is irrigated with normal saline 2-3 times daily until frontonasal duct becomes patent

Acute Ethmoid Sinusitis

- Often associated with infection of other sinuses

- Pain : localised over the bridge of the nose, medial and deep to the eye.
- Nasal discharge
- Swelling of middle turbinate.
- Treatment: same as for acute maxillary sinusitis.

Acute Sphenoid Sinusitis

- Isolated involvement of sphenoid sinus is rare.
- Often a part of pansinusitis or associated with infection of posterior ethmoid sinuses.
- Headache : usually localised to the occiput or vertex
- Postnasal discharge.
- Treatment: same as for acute infection of other sinuses.

CHRONIC SINUSITIS

- Sinus infection lasting for months or years is called Chronic sinusitis
- Most important cause of chronic sinusitis is failure of acute infection to resolve.

Pathophysiology:

- Acute infection destroys normal ciliated epithelium impairing drainage from sinus.
- Pooling and stagnation of secretions inside the sinus invites infection
- Persistence of infection causes mucosal changes, such as loss of cilia, oedema and polyp formation, thus continuing the vicious cycle.

Clinical features:

- Similar to those of acute sinusitis but of lesser severity
- Purulent nasal discharge
- Complaint of nasal stuffiness and anosmia may be found

Treatment:

- Conservative : antibiotics, decongestants, antihistaminics, sinus irrigations
- Surgery: endoscopic sinus surgery

EXAMINATION OF NOSE

1. External examination of nose
2. Anterior rhinoscopy
3. Posterior rhinoscopy
4. Examination of paranasal sinuses

External examination

1. **Depresssed nasal bridge** : leprosy, syphilis, tuberculosis of nose, cretinism, thalassemia major
2. **Nasal injury**: after road accidents
3. **Deviated nose**: common in boxers
4. **Congenital swelling**: Glioma present since birth
5. **Benign conditions**: Rhinophyma (potato nose) due to hypertrophy of sebaceous glands
6. **Broadening of nose**: Antrochoanal polyp
7. **Frog face deformity**: juvenile NAF
8. **Crepitus** : fracture nasal bone

Anterior rhinoscopy

- Carried out by Thudicum's nasal speculum.
- First raise the tip of nose by the thumb and examine the vestibule before using nasal speculum.

1. Nasal septum
2. Nasal cavity
3. Turbinates
4. Middle meatus
5. Colour of mucosa
6. Discharge
7. Crusts
8. Polyp
9. Mass
10. Bleeding
11. Pus
12. Maggots (if any)

Septum:

1. Septal spur: sharp angulations, occuring at the junction of septal cartilage with the ethmoid or vomer bone.
2. Septal deviation: C shaped or S shaped.
3. Septal dislocation: lower portion of septal cartilage is displaced from the median position.
4. Septal perforation : causes whistling sound during inspiration while a large septal perforation causes crusting of nose.

Causes:

1. Syphilis
2. Tuberculosis
3. Granulomatous disease of the nose
4. Leprosy
5. Idiopathic
6. Snuff taker's perforation
7. Chromium perforation

Nasal cavity:

- Roomy nasal cavity : atrophic rhinitis
- Greenish crusts : atrophic rhinitis

Turbinates

- Inferior turbinate may be normal or engorged on one side or both sides. If DNS is on left side, there is a compensatory hypertrophy of RIT.
- Middle turbinate has normal medial convexity towards the septum. If it has convexity towards the lateral wall, it is called paradoxically bent middle turbinate.
- Superior turbinate can be seen only by the nasal endoscope.

Middle meatus

- Look for any pus in the middle meatus.
- Pus in the middle meatus means suppuration of ethmoid or maxillary sinus.

Colour of mucosa

- Pinkish red – normal
- Red congested mucosa – acute rhinitis
- Pale oedematous mucosa – allergy
- Pinkish white mucosa – anaemia, tuberculosis

Discharge

- Watery – allergic rhinitis
- Clear fluid discharge – CSF rhinorrhoea
- Mucoid discharge – antrochoanal polyp
- Mucopurulent discharge – chronic maxillary sinusitis
- Blood stained discharge – inverted papilloma or underlying malignancy
- Foul smelling unilateral discharge in children – F.B. in nose unless proved otherwise.

Crusts

- Foul smelling crusts are seen in atrophic rhinitis.

Polyp

- Polyps are edematous mucus membrane. They are not new growth.
- Antrochoanal or ethmoidal polyps.

Mass

- Could be benign or malignant.
- Benign can be JNAF
- Malignant could be arising from nose, sinuses or nasopharynx.

Bleeding

- Ulceration with clotted blood is seen in the little's area in hypertensive patients.
- Epistaxis digitorum (bleeding by nose picking) is another common cause of bleeding in children and adults especially in summers.

Pus:

- Pus in the middle meatus – suppuration of anterior group of sinuses
- Pus in the olfactory cleft – suppuration of posterior group of sinuses.
- Maggots: Occur in nose in atrophic rhinitis.

Posterior Rhinoscopy

- Posterior part of septum, both choanae, common meatus, roof and posterior wall of nasopharynx, opening of nasopharyngeal end of eustachian tube, adenoid in case of children, JNAF, nasopharyngeal carcinoma, antrochoanal polyp.

Examination of Paranasal Sinuses

Inspection:

- In acute sinusitis, there may be swelling and redness of the skin overlying the maxillary sinuses.

Palpation:

- The tenderness of the maxillary sinuses is elicited by applying the firm pressure at the canine fossa.
- The tenderness of ethmoid sinuses are elicited by applying pressure at the lateral side of bridge of nose.
- The tenderness of frontal sinuses is elicited by applying pressure at the floor of frontal sinus just above the orbit.
- **Sinusitis can be confirmed by taking x-ray PNS.**
- **The haziness of the sinuses should be compared with the orbit.**

CHAPTER 5
DISEASES OF THROAT AND ORAL CAVITY

Anatomy of the oral cavity

Divided into

- An outer smaller portion: the **vestibule**
- An inner larger part: the **oral cavity proper**

1. **Vestibule:**
   - A narrow space bounded
     - externally by the lips & cheeks and
     - internally by teeth & gums
   - Except for the teeth, the entire vestibule is lined by mucous membrane.
2. **Oral cavity proper**
   - Bounded anterolaterally by the teeth, gums, alveolar arches of the jaws.
   - The roof is formed by the hard & soft palate.
   - The floor is occupied by the tongue posteriorly, and presents the sublingual region anteriorly, below the tip of tongue.
   - Posteriorly the cavity communicates with the pharynx

Lips

- Fleshy folds lined externally by skin and internally by mucous membrane.
- Each lip is composed of:
  - Skin
  - Superficial fascia
  - The orbicularis oris muscle
  - The submucosa containing labial glands & blood vessels
  - Mucous membrane
- The inner surface of each lip is supported by a frenulum which ties it to the gum.
- The outer surface of the upper lip presents a median vertical groove – the philtrum.

Cheeks (Buccae)

- Flashy flaps forming a large part of each side of the face.
- They are continuous in front with the lips and the junction is indicated by the nasolabial sulcus (furrow) which extends from the side of the nose to the angle of the mouth.
- Each cheek is composed of:
  - Skin
  - Superficial fascia
  - The buccinator covered by buccopharyngeal fascia
  - Submucosa with buccal glands
  - Mucous membrane

Palate

Palate is divided into anterior hard palate and posterior soft palate.

Hard palate:

- It is a partition between nasal & oral cavities.
- Its
  - anterior 2/3 are formed by the palatine processes of the maxillae
  - posterior 1/3 by the horizontal plates of the palatine bones.

Soft palate (Velum or muscular palate)

- It is a movable, muscular fold, suspended from the posterior border of the hard palate.
- It separates the nasopharynx from the oropharynx.
- Muscles:
  - Tensor palati - swallowing
  - Levator palati - swallowing
  - Musculus uvulae – moves the uvula
  - Palatoglossus - swallowing
  - Palatopharyngeus - breathing

Tongue

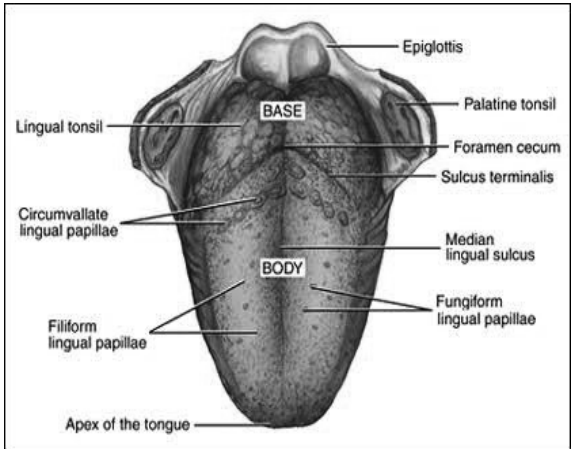
- Root
- Tip
- Body

A body:

Upper surface/dorsum:

- Convex in all directions.
- Divided into
  - An oral part – anterior 2/3
  - A pharyngeal part – posterior 1/3

  By a faint V-shaped groove, the sulcus terminalis.
- The 2 limbs of the V meet at a median pit – foramen caecum
- Posterior most part of the tongue is connected to the epiglottis by three folds of mucous membrane.
- Median; right & left glossoepiglotic folds.



Muscles of the tongue:

- A middle fibrous septum divides the tongue into right and left halves.
- Each half contains 4 intrinsic & 4 extrinsic muscles

Intrinsic muscles:	Extrinsic muscles:
• Superior longitudinal • Inferior longitudinal • Transverse • vertical	• Genioglossus • Hyoglossus • Styloglossus • Palatoglossus

Papillae of the tongue

- These are the projections of mucous membrane or corium which give the anterior 2/3 of the tongue its characteristic roughness.

Filiform/conical:

- Cover the presulcul area of the dorsum of tongue.
- Smallest, most numerous
- Contain less number of taste buds.

Fungiform:

- Numerous near the tip and margins of the tongue.
- Larger than filiform.
- Number of taste buds in each is moderate (up to 10).

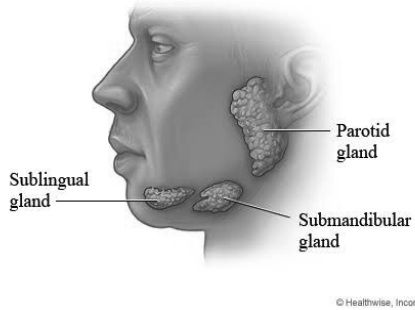
Vallate/circumvallate:

- Situated immediately in front of sulcus terminalis.
- Large in size.
- Each papillae contains many taste buds (up to 100).

Salivary glands

- The saliva is secreted by three pairs of major salivary glands and some minor salivary glands in the oral & pharyngeal mucous membrane.
- The major glands are:
  - Parotid
  - Submaxillary/submendibular
  - Sublingual

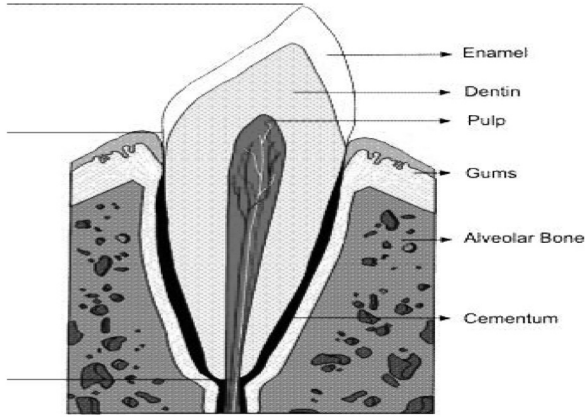
Gland	Location	Duct
Parotid glands	Situated at the side of the face just below and in front of the ear. Largest of the salivary glands	• ***Stensen's duct.*** • Open inside the cheek against the upper second molar tooth. • 35-40mm long.
Submaxillary glands	Located in submaxillary triangle medial to mandible.	• ***Wharton's duct*** • Opens at the side of frenulum of tongue by means of a small opening on the summit of papilla called caruncula sublingualis.
Sublingual glands	Situated in the mucosa at the floor of the mouth.	• ***5-15 small ducts of Ravinus.*** • Open on small papillae beneath the tongue. • One of the ducts is larger and is called Bartholin's duct.



Tooth

- A tooth consists of ***three hard tissues***- enamel, dentine and cementum surrounding ***a soft tissue***- the pulp.
- The pulp is surrounded by dentin on all sides except at the apical foramen, where it is continuous with the periodontal soft tissue.
- The dentin forms the bulk of the tooth, but it is not exposed outside.

- The part of the tooth covered by enamel – **Crown**. The part covered by cementum – **Root**.
- The line of the junction of the crown and the root is known as the **cemento-enamel junction** or the **cervical line**.
- The part of the root area immediately adjacent to the crown is called the **neck of the tooth**.
- The crown is exposed to the oral cavity and the root lies in a socket in the set of fibrous ligament, the periodontal ligament.
- The pulp cavity in the crown area is called the **pulp chamber** and in the root portion it is known as the **pulp canal or the root canal.**
- The roots may be
    - single- incisors and canines or
    - multiple- molars.
- The portion of the jaw bone that anchors and supports the teeth is called the **alveolar bone or the alveolar process** and the hole in which the root lies is called the **socket.**



Gingiva

- That part of mucosa which surrounds the tooth and covers the alveolus.
- The gingiva is divided into two regions depending on the firmness with which it is attached to the underlying tissue.
    - Free gingiva
    - Attached gingiva

- About 1-15 mm of coronal part of the gingiva is loosely attached to the tooth.
- The potential space between tooth and free gingiva is called *gingival sulcus/gingival crevice.*
- Viewed from facial and lingual aspects, the margins of free gingiva appear as a wavy line – *marginal gingiva*.
- The free gingiva in the *interproximal area – papillary gingiva / interproximal gingiva.*

- Gingiva apical to the free gingiva is firmly attached to the underlying structures via gingival fibres of periodontal ligaments.
- The attached gingiva is continuous with alveolar mucosa. This border is called *mucogingival line*. This line is imperceptible on the palate.

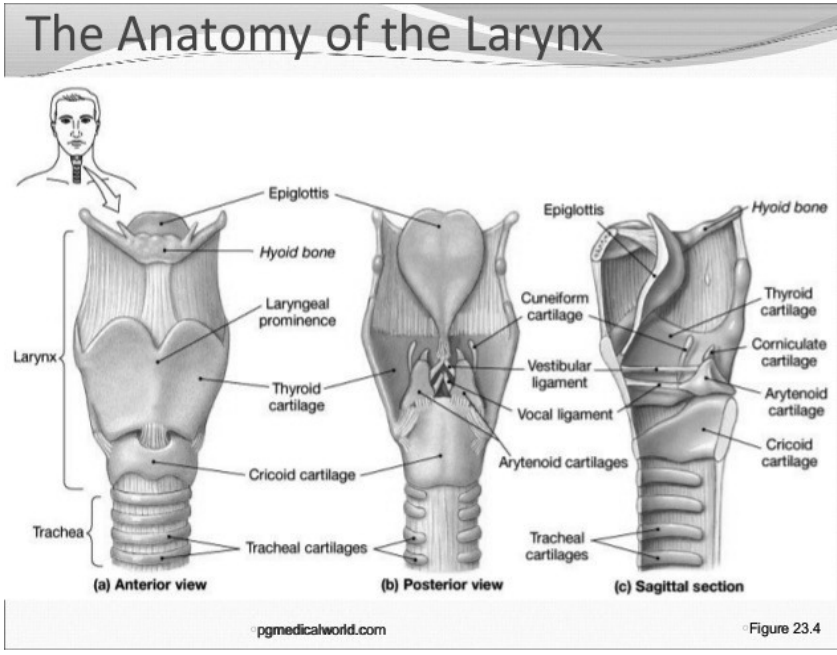
Larynx

- From base of tongue to trachea
- Opposite the 3rd-6th cervical vertebrae
- Higher in females and children.
- The male larynx enlarges after puberty.

Cartilages:

Paired	Unpaired
1. Arytenoid 2. Corniculate (Cartilage of Santorini) 3. Cuneiform (Cartilage of Wrisberg)	1. Thyroid 2. Cricoid 3. Epiglottis

- Thyroid is the largest.
- Its two alae meet anteriorly forming an angle of 90 in males and 120 in females.
- The laryngeal prominence, Adam's apple in males is due to this angle.



Muscles:

Intrinsic (within the framework of larynx.)	**Extrinsic** (connect larynx to hyoid and sternum.)
Abductors: (movement away from each other) 1. Posterior cricoarytenoid Adductors: (approximation with each other) 1. Lateral cricoarytenoid 2. Interarytenoid 3. Thyroarytenoid Tensor of vocal cord: 1. Cricothyroid 2. Vocalis	Elevators: 1. Stylopharyngeus 2. Salpingopharyngeus 3. Palatopharyngeus 4. Thyrohyoid Depressors: 1. Sternohyoid 2. Sternothyroid 3. Omohyoid

Nerve supply:

- Cricothyroid muscle: superior laryngeal nerve
- All other intrinsic muscles: recurrent laryngeal nerve.
- Both of these are branches of Vagus nerve.

Joints: synovial joints.

1. Cricothyroid
2. Cricoarytenoid

Functions:

1. Respiration: vocal cords abduct during inspiration and adduct during expiration.
2. Protection of lower airways: watch-dog of lungs
3. Phonation
4. Fixation of chest

Pharynx

- Base of skull to 6th cervical vertebrae.
- Approx. 15 cm in length. 3.5 cm at base and 1.5cm at cricopharynx.
- From above downwards the nasal cavity, oral cavity and laryngeal inlet open into the pharynx. The corresponding part is named as: nasopharynx, oropharynx, laryngopharynx (hypopharynx).

- The lower end of pharynx is continuous wuth oesophagus. This junction at cricopharyngeal sphincter is the narrowest point in the digestive tract.

Nasopharynx:

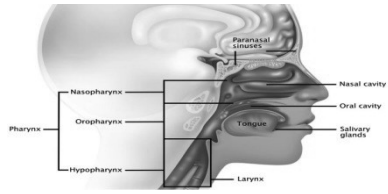
- Posteriorly related to C2.
- Eustachian tube opening: in lateral wall on each side of nasopharynx. The orifice is bounded postero-superiorly by torus tubaris. Just posterior to it, fossa of Rosenmuller. This is one of the hidden areas of body where malignancy can develop and difficult to diagnose early.
- Tubal tonsils (Gerlach's tonsils)
- Sinus of morgagni: a potential space between the base of skull and the upper border of the superior constrictor muscle of pharynx.
- Adenoids
- Rathke's pouch: embryological structure which disintegrates with development. An ectodermal diverticulum from which the anterior part of pituitary gland develops.
- Nasopharyngeal bursa: covers the adenoids
- Passavant's ridge.

Oropharynx:

- Space between the hard palate and hyoid bone.

Laryngopharynx:

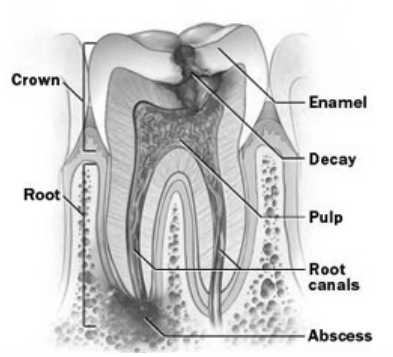
- Extends from hyoid bone to the level of C6 (cricoids cartilage).
- Pyriform sinus: a recess on either side of larynx. The internal laryngeal nerve traverses submucosally through the pyriform sinus and is easily accessible for local anaesthesia for procedures such as direct laryngoscopy and biopsy. This also forms one of hidden areas for malignancy.



Dental caries

- An irreversible microbial disease of calcified tissues of the teeth, characterized by
  - Demineralization of the inorganic portion and
  - Destruction of the organic substance of the teeth

  Which often leads to cavitations.



Etiology:

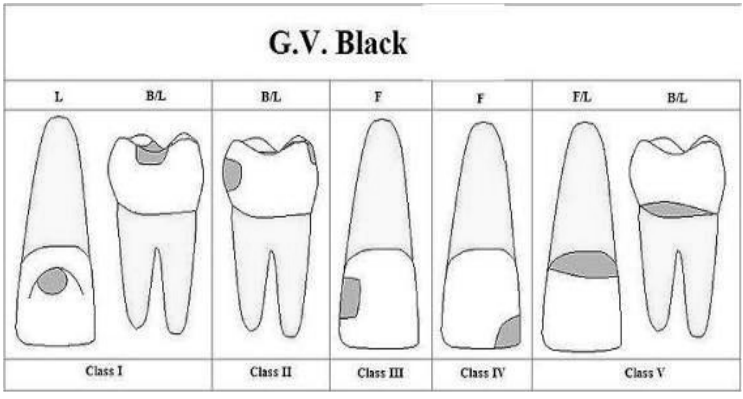
1. Tooth
   - Presence of deep, narrow retentive pits and fissures
   - Malaligned, rotated or out of position teeth
2. Plaque:
   - Plaque has high concentration of streptococcus mutans and lactobacillus acidophilus.
   - Streptococcus mutans can readily ferment dietry carbohydrate to produce acids which cause tooth destruction.

3. Diet:
   - Fermentable dietry carbohydrates play an important role in formation of caries. e.g. glucose, sucrose, fructose.
   - These sugars are easily and rapidly fermented by cariogenic bacteria to produce acid and cause dissolution of hydroxyapatite crystal of enamel and dentin.
   - The risk of caries is increased if sugar is taken repeatedly between two major meals because it provides constant supply of carbohydrates for formation of acids.

- It is also increased when dietry food is sticky in nature which can remain adhere to the tooth surface for a long time after taking the meal.

Classification of caries: G.V.Black classification

- **Class I caries**: occurring in pits, fissures or defective grooves on the tooth surfaces. This usually has three locations:
  - Occlusal surfaces of molars and premolars,
  - occlusal two-thirds of the facial and lingual surfaces of molars and
  - the lingual surfaces of maxillary anteriors
- **Class II caries**: found on the proximal surfaces of molars and premolars.
- **Class III caries:** occurring in the proximal surfaces of anterior teeth without involving the incisal angle.
- **Class IV caries:** found on the proximal surfaces of anterior teeth with involvement of the incisal angle.
- **Class V caries**: seen at the gingival third of the facial and lingual surfaces of anterior and posterior teeth.
- **Class VI caries**: found on the incisal edges of anterior teeth and cusp tips of posterior teeth.

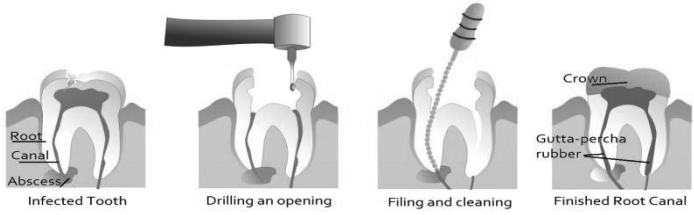


Prevention of caries:

- Use fluorides: fluorides work mainly by slowing down the process of demineralization. It also helps to heal (remineralize) surfaces.
- Reduce frequent consumption of sugars
- Pit and fissure sealants: they create a thin barrier preventing the access of plaque and plaque acids to the enamel surface.

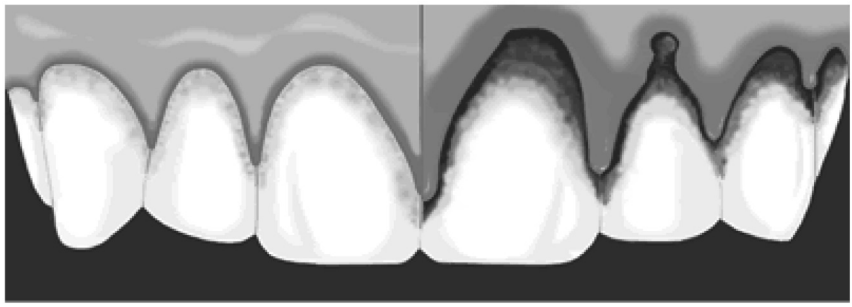
Treatment:

- The main treatment option for tooth cavity is to drill out the decay and put in a filling (Restoration) made from various materials (e.g. composite resins, amalgam, porcelain)
- Extensive tooth decay may necessitate RCT (Root Canal Treatment) or even extraction of the tooth.
- When you undergo a root canal or other endodontic treatment, the inflamed or infected pulp is removed and the inside of the tooth is carefully cleaned and disinfected, then filled and sealed with a rubber-like material called **gutta-percha**.



Tartar (Calculus)

- Calculus is a hard deposit that forms by mineralization of dental plaque, and it is generally covered by a layer of unmineralized plaque.
- Calculus is classified as supragingival or subgingival, according to its relation to the gingival margin.



Dental Plaque Tartar

Supragingival calculus

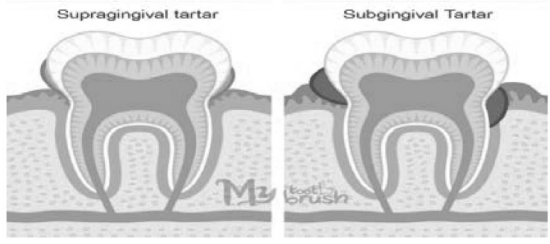
- Located coronal to the gingival margin and therefore is visible in the oral cavity.
- Usually white or whitish yellow in colour, hard with clay like consistency, and easily detached from the tooth surface.
- After removal, it may rapidly recur, especially in the lingual area of the mandibular incisors.

Subgingival calculus

- Located below the crest of the marginal gingiva and therefore is not visible on routine clinical examination.
- The location and extent of subgingival calculus may be evaluated by careful tactile perception with a delicate dental instrument such as an explorer.
- Subgingival calculus is typically hard and dense and frequently appears dark brown or greenish black while being firmly attached to the tooth surface.

Treatment:

Scaling is the process by which plaque and calculus are removed from both supragingival and subgingival tooth surfaces.



Gingivitis

Etiology :

- **Agent factors:**
  - Plaque
  - Calculus
- **Host factors:**
  - **Diet**: Sticky food adheres to the teeth and is difficult to remove, thus interfering with the natural cleansing process of the oral cavity
  - **Tooth anatomy**: poor anatomy, crowding of teeth, mal alignment can cause food impaction and accumulation causing the gingival tissue to become irritated and inflamed
  - **Habits**: unilateral mastication, abnormal habits like biting a pencil, finger nail ; incorrect brushing method
  - **Local irritants:** alcohol, tobacco, condiments lower the tissue resistance; mouth breathing because of obstruction in nasal passage, habit
  - **Systemic factors:** anemia, puberty and pregnancy gingivitis, DM, leukaemia, HIV, radiation etc

Clinical features:

- Erythema and enlargement of gingival tissue
- Interdental papillae become enlarged
- Bleeding while brushing
- Pain may or may not be present

Types

- Localized: confined to the gingiva of a single tooth
- Generalized: involves the entire mouth
- Marginal: involves the gingival margin
- Papillary: involves interdental papille and often extends to the adjacent portion of gingival margin
- Diffuse: affects gingival margin, attached gingiva and interdental papillae.

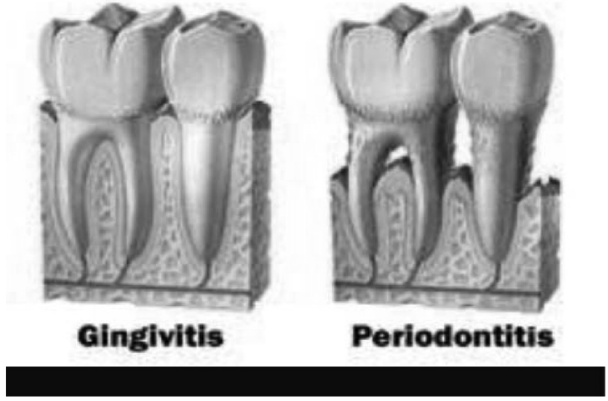
Treatment

- Correction of local contributing factors
- Removal of local irritants
- Initiation and maintenance of oral hygiene

- Treatment of underlying systemic cause

Periodontitis

- Inflammation and infection of the ligaments and bones that support the teeth.
- An inflammatory disease of the supporting tissues of the teeth caused by specific microorganism or group of specific microorganisms resulting in progressive destruction of the periodontal ligament and alveolar bone with pocket formation, destruction or both.
- The clinical feature that distinguishes periodontitis from gingivitis is the presence of clinically detectable attachment loss.
- This is often accompanied by periodontal pocket formation and changes in the density and height of subjacent alveolar bone.

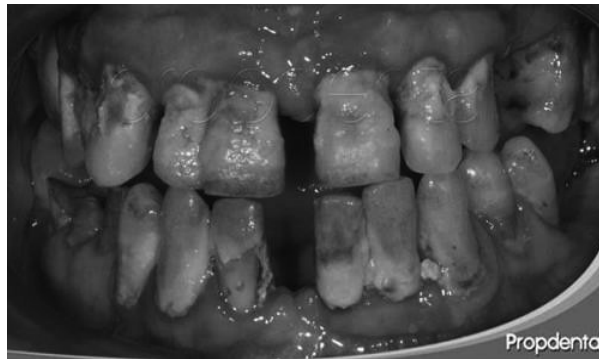


- Occurs when gingivitis is left untreated or treatment is delayed.
- Infection and inflammation spreads from the gums to the ligaments and bone that support the teeth.
- Loss of support causes the teeth to become loose and eventually fall out.

Chronic periodontitis:

- Most common form of periodontitis

- Associated with the accumulation of plaque and calculus
- Localized: less than 30% sites involved
- Generalized: more than 30% sites involved



Symptoms:

- Usually painless
- Pain may be present caused by exposed roots that are sensitive to heat, cold or both.
- Gingival tenderness or itchiness may be found
- Presence of areas of food impaction
- Teeth may be loose

Clinical features

- Supra and subgingival plaque acummulation ass. with calculus formation
- Gingival inflammation
- Pocket formation
- Loss of periodontal attachment
- Loss of alveolar bone

Aggressive periodontitis

- Rapidly progressing type
- Patient do not have large accumulations of plaque or calculus
- The disease appears to be the result of defect in the immune response
- Patient may have functional defect of polymorphonuclear leucocytes, monocytes or both but without any systemic manifestation.

- Causative organisms:
  - Actinobacillus actinomycetemcomitans
  - Porphyromonas gingivalis
  - Mycoplasma

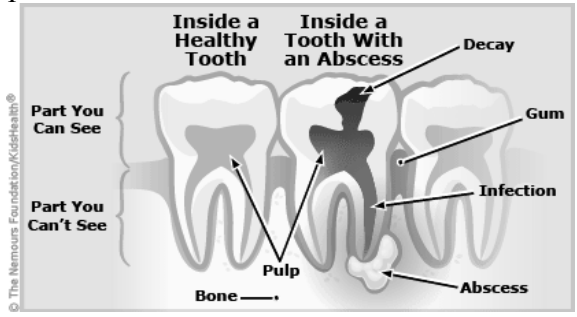
- Localized:
  - Localized to the first molars and incisors
- Generalized:
  - Involves at least 3 teeth other than the first molar and incisor
  - Usually affecting person under 30 years of age

Treatment of periodontitis:

- Proper brushing and flossing
- Professional tooth cleaning more than twice a year
- Rough surfaces of teeth should be repaired
- Deep pockets may need to be opened and cleaned
- Loose teeth may need to be supported or may need to be removed.

Acute alveolar abscess/Acute periapical abscess/ Acute apical abscess

- Localised collection of pus in the alveolar bone at the root apex of a tooth, following death of the pulp, with extension of the infection through the apical foramen into the periradicular tissues.



Causes:

- Extension of pulpal infection into periapical tissue.
- Fracture of tooth with pulp exposure.

- Accidental perforation of the apical foramen during RCT, which results in entry of pulpal microorganisms into periapical area.
- Extension of periodontal infection.

Symptoms:

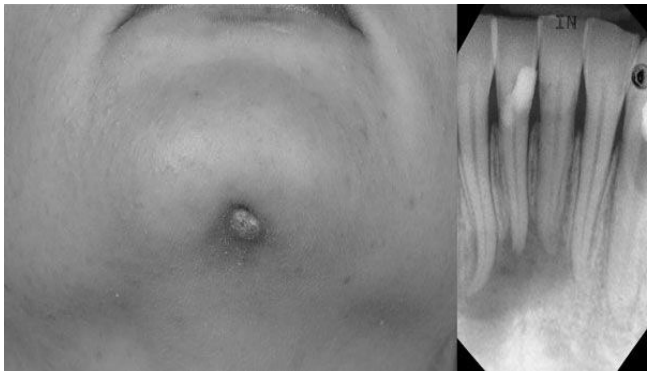
- Severe pain in affected tooth.
- Pain aggravates during percussion or when pressure is applied with the opposing tooth.
- Application of cold relieves pain temporarily.
- Localised swelling and erythematous change in overlying mucosa.
- The affected area of the jaw may be tendered on palpation.
- Localized lymphadenitis
- If left untreated, the infection may progress to chronic apical abscess wherein the pus may break through to form a sinus tract, usually opening in the labial/buccal mucosa.
- It may further progress to osteitis, periosteitis, cellulitis or osteomyelitis.

Chronic alveolar abscess

- Long standing, low grade infection of periradicular alveolar bone characterized by the presence of an abscess draining through a sinus tract.
- The source of infection is in the root canal.
- It is a natural sequel of death of the pulp with extension of the infection periapically.
- It may result from a pre-existing acute abscess.

Clinical features:

- Tooth is generally asymptomatic or mildly painful.
- The sinus tract usually prevents exacerbation or swelling by providing continual drainage of the periradicular lesion.
- At times, such an abscess is detected only during routine radiographic examination or because of the presence of a sinus tract, which can be either intraoral or extraoral.



Treatment of alveolar abscess:

- Drainage is established either through an opening in the tooth or by an incision over the soft tissue swelling at the apex region.
- Antibiotics
- Once the acute phase of disease is brought under control, the affected tooth is treated either by root canal therapy or by extraction.

TONSILLITIS

- **Acute tonsillitis:**
  - Acute catarrhal/superficial
  - Acute follicular
  - Acute parenchymatous
  - Acute membranous

- **Chronic tonsillitis:**
  - Chronic follicular
  - Chronic parenchymatous
  - Chronic fibroid

Acute Tonsillitis

- Acute infections of tonsil are classified as:
  - **Acute catarrhal/superficial tonsillitis**:
    As a part of gen. pharyngitis, mostly seen in viral infection.

– **Acute follicular tonsilltis:**
Infection spreads into the crypts which become filled with purulent material, presenting at the openings of crypts as yellowish spots.
– **Acute parenchymatous tonsillitis:**
Tonsil is uniformly enlarged and red.
– **Acute membranous tonsillitis**:
Stage ahead of acute follicular tonsillitis when exudation from the crypts coalesces to form a membrane on the surface of tonsil.

Aetiology:
- Often affects school going children, but also affects adults.
- *Haemolytic streptococcus* is the most infecting organism.
- Other causes may be *staphylococci*, *pneumococci* or *H. Influenzae*.

Symptoms:
- Sore throat
- Difficulty in swallowing: the child may refuse to eat anything due to local pain.
- Fever : 38-40 C, may be associated with chills and rigors
- Earache: referred pain or due to AOM which may occur as a complication
- Constitutional symptoms: Headache, general body aches, malaise

Treatment:
- Patient is put to bed, encouraged to take plenty of fluids.
- Analgesics (aspirin or paracetamol) to relieve pain and bring down the fever
- Antimicrobial therapy: most of infections are due to streptococcus, and penicillin is the drug of choice.

Chronic Tonsillitis
- Aetiology:
  – May be complication of acute tonsillitis
  – Mostly affects children and young adults

- Chronic infection in sinuses or teeth may be a predisposing factor.

Types:

- **Chronic follicular tonsillitis**:
  - Crypts are full of infected cheesy material
- **Chronic parenchymatous tonsillitis**:
  - Tonsils are very much enlarged and may interefere with speech, deglutition and respiration. Attacks of sleep apnoea may occur.
- **Chronic fibroid tonsillitis**:
  - Tonsils are small but infected, with history of repeated sore throats.

Clinical features

- Recurrent attacks of sore throat or acute tonsillitis
- Chronic irritation in throat with cough
- Bad taste in mouth and foul breath due to pus in the crypts
- Thick speech, difficulty in swallowing

Treatment

- Conservative: general health, diet, treatment of co-existent infection of teeth, nose and sinuses.
- Tonsillectomy when tonsils interfere with speech, deglutition and respiration or cause recurrent attacks.

PHARYNGITIS

Acute Pharyngitis

- Very common
- Occurs due to varied etiological factors like viral, bacterial, fungal or others.
- Viral causes are more common.

Causes:
Viral
- Rhinoviruses
- Influenza
- Measles and chickenpox
- Coxsackie virus
- Herpes simplex
- Infectious mononucleosis

Bacterial
- Streptococcus
- Diphtheria
- Gonococcus

Clinical features:
- Milder infections:
  - Discomfort in throat, some malaise, low grade fever
- Moderate to severe infections:
  - Pain in throat, dysphagia, headache, malaise, high fever.
  - Viral infections are generally mild and are accompanied by rhinorrhoea and hoarseness while the bacterial ones are severe.

Treatment:
- **General measures*:***
  - Bed rest, plenty of fluids, warm saline gargles
  - Local discomfort in the throat in severe cases can be relieved by lignocaine viscous before meals to facilitate swallowing.
- **Specific treatment*:***
  - Streptococcal pharyngitis is treated with penicillin G, 2,00,000 to 2,50,000 units orally four times a day for 10 days

<u>Chronic Pharyngitis</u>
- 2 types:
  - **Chronic catarrhal**:
    - There is a congestion of PPW
  - **Chronic hypertrophic (granular):**

- PPW may be studded with reddish nodules (hence the term granular)
- These nodules are due to hypertrophy of subepithelial lymphoid follicles normally seen in pharynx.

Etiology

- **Persistent infection in the neighbourhood**: in chronic rhinitis and sinusitis, purulent discharge constantly trickles down the pharynx and provides a constant source of infection.
- **Mouth breathing**: exposes the pharynx to air which has not been filtered, humidified and adjusted to body temperature thus making it more susceptible to infections.
- **Chronic irritants:** excessive smoking, chewing of tobacco and pan, heavy drinking, highly spiced food
- **Environmental pollution**: smoky/ dusty environment or industrial fumes
- **Faulty voice production:** excessive use of voice or faulty voice production.

Symptoms:

- Discomfort or pain
- Foreign body sensation in throat
- Tiredness of voice: patient cannot speak for long and has to make undue effort to speak as throat starts aching.
- Cough: throat is irritable and there is tendency to cough

Treatment:

- Etiological factor should be eradicated
- Voice rest,
- Hawking, clearing the throat frequently or any other such habit should be stopped
- Warm saline gargles especially in the morning.

LARYNGITIS

Acute laryngitis

Infectious

- More common
- Follows URTI

- To begin with, it is viral, but soon bacterial invasion takes place with
  - *strept. Pneumoniae,*
  - *H. influenzae,*
  - *hemolytic streptococci or*
  - *staph. Aureus.*

Non-infectious

- Vocal abuse,
- Allergy
- Thermal or chemical burns to larynx due to inhalation or ingestion of various substances
- Laryngeal trauma

Symptoms:

- Hoarseness of voice which may lead to complete loss of voice
- Discomfort or pain in throat, particularly after talking
- Dry, irritating cough which is usually worse at night

Treatment:

- Vocal rest: most important
- Avoidance of smoking, alcohol
- Steam inhalation
- Antibiotics: when infection
- Analgesics: to relieve local pain, discomfort
- Steroids: in laryngitis following thermal or chemical burns

Chronic Laryngitis:

- Diffuse inflammatory condition symmetrically involving the whole larynx

Etiology:

- May follow incompletely resolved acute laryngitis or its recurrent attacks
- Presence of chronic infection in PNS, teeth and tonsils
- Occupational factors e.g. exposure to dust and fumes

- Smoking and alcohol
- Vocal abuse
- Persistent trauma of cough as in chronic lung diseases

Symptoms:

- Hoarseness. The voice becomes easily tired and patient becomes aphonic by the end of the day.
- Constant hawking. Dryness and intermittent tickling in the throat and patient is compelled to clear the throat repeatedly.
- Discomfort in throat
- Cough: dry and irritating

Treatment:

- Eliminate infection of URT or LRT: infection in sinuses, tonsils, teeth
- Avoidance of irritating factor: e.g. smoking, alcohol, polluted environment
- Voice rest and speech therapy: voice rest has to be prolonged for weeks or months.
- Steam inhalation: help to loosen secretions
- Expectorants: help to loosen viscid secretions and give relief from hawking

ADENOIDITIS

Etiology:

- Recurrent attacks of rhinitis, sinusitis or chronic tonsillitis may cause chronic adenoid infection and hyperplasia.
- Allergy of Upper respiratory tract may also contribute to the enlargement of adenoids.

Clinical features:

Nasal symptoms:

- Nasal obstruction: commonest, leads to mouth breathing
- Nasal discharge: due to choanal obstruction as the normal nasal secretions cannot drain into nasopharynx and associated chronic rhinitis

- Sinusitis: chronic maxillary sinusitis is commonly associated with ademoids due to persistence of nasal discharge.
- Epistaxis: when adenoids are acutely inflamed, epistaxis can occur with nose blowing
- Voice change

Aural Symptoms:

- Tubal obstruction: adenoid mass blocks the eustachian tube
- Recurrent attacks of AOM due to spread of infection via eustachian tube
- CSOM
- SOM

General symptoms:

- Adenoid facies: chronic nasal obstruction and mouth breathing lead to characteristic facial appearance called adenoid facies.
  - Elongated face with dull expression, open mouth, prominent and crowded upper teeth

Diagnosis:

- Examination of postnasal space is possible in some young children
- A nasopharyngoscope is also useful
- Soft tissue lateral radiograph of nasopharynx will reveal the size of adenoids and also the extent to which nasopharyngeal air space has been compromised.

Treatment:

- When symptoms are not marked, breathing exercise, decongestant nasal drops, anti histaminics for co-existent nasal allergy
- When symptoms are marked, adenoidectomy is done.

DYSPHAGIA

- Dysphagia : difficulty in swallowing
- Odynophagia: pain while swallowing

Causes:

- Pre-oesophageal causes
  - Oral phase

  - Pharyngeal phase
- Oesophageal causes

1. Pre-oesophageal causes:

Oral phase

- **Disturbance in mastication**
  - Trismus, tumours of upper or lower jaw
- **Disturbance in lubrication**
  - xerostomia
- **Disturbance in mobility of tongue**: paralysis of tongue, tumours of tongue, painful ulcers, total glossectomy
- **Defects of palate**: cleft palate
- **Lesions of buccal cavity and floor of mouth**: stomatitis, ulcerative lesions

Pharyngeal phase:

- **Obstructive lesions of pharynx:** tumours of tonsil, soft palate, pharynx, BOT
- **Inflammatory conditions:** acute tonsillitis, peritonsillar abscess, acute epiglossitis
- **Spasmodic conditions:** Tetanus, rabies
- **Paralytic conditions:** paralysis of soft palate.

2. Oesophageal phase:

- **Lumen**:
  - foreign body, benign/malignant tumours
- **Wall**:
  - Acute/chronic oesophagitis,
  - motility disorders
    - Hypomotility: achalasia
    - Hypermotility: diffuse oesophageal spasm
- **Outside the wall**:
  - Hiatus hernia
  - Thyroid lesions: enlargement, tumours
  - Mediastenal lesions: tumours, aortic aneurysm, lymph node enlargement.

Investigation:

- History:
  - Sudden onset : foreign body or impaction of food

  - Progressive: malignancy
  - Intermittent: spasm
  - More to liquids: paralytic lesion
  - More to solids and progressive even to liquids: malignancy or stricture
  - Intolerance to acid food or fruit juices: ulcerative lesion
- Clinical examination: to exclude most of the pre-oesophageal causes
- Blood examination: for diagnosis of Plummer-vinson sydrome and to know the nutritional status of the patient.
- Oesophagoscopy: direct examination of oesophageal mucosa and permits biopsy specimens.
- Radiography:
  - X-ray chest: to exclude cardiovascular, pulmonary, mediastinal causes
  - Lateral view of chest: to exclude cervical osteophytes and soft tissue lesions of post-cricoid or retropharyngeal space.
  - Barium swallow: in the diagnosis of malignancy, cardiac achalasia, strictures, hiatus hernia or oesophageal spasms.

TONGUE TIE (ANKYLOGLOSSIA)

- It is a condition present at birth that restricts the tongue's range of motion.
- With tongue tie, an unusually short, thick or tight band of tissue (Lingual frenulum) tethers the bottom of the tongue's tip to the floor of mouth.
- Why this happens is largely unknown.

Symptoms:

- Difficulty in lifting the tongue to the upper teeth or moving the tongue from side to side
- Breast feeding problems
- Speech difficulties: it can interfere with the ability to make certain sound such as: t, th, d, s, r

- Poor oral hygiene: tongue tie can make it difficult to sweep food debris from the teeth.
- Challenges with other oral activities: such as licking an ice-cream cone, playing a wind instrument

Treatment:

- Surgery: frenotomy/frenuloplasty.

RANULA

- Derived from the latin word rana, meaning frog.
- It describes blue translucent swelling in the floor of the mouth reminiscent of the underbelly of a frog.

Pathophysiology:

- **Partial obstruction** of sublingual duct can lead to formation of retention cyst.
- **Trauma**: with trauma, if a duct is obstructed, secretory backpressure bulids leading to mucus extravasation.
- Alternately, trauma causes direct damage to the duct or acini leading to mucus extravasation.

Symptoms:

- Most commonly observed as a bluish cyst located below the tongue
- It may fill the mouth and raise the tongue.
- Typically, these are painless masses that do not change in size in response to chewing, eating or swallowing.
- Occasionally, pain may be involved.

Plunging ranula:

- Deep/diving/cervical ranula
- When these extravasation cysts extend into the submandibular or submental space, they are called plunging ranula.
- The extension of ranula is related to the herniation of the mylohyoid muscle by the sublingual gland.

Treatment:

- Marsupialization (the opening of the cyst to the surface)
- Complete excision of cyst and the associated gland – would prevent chances of recurrence

CLEFT LIP AND CLEFT PALATE

- Cleft lip and cleft palate are facial and oral malformations that occur very early in pregnancy.
- Clefting results when there is not enough tissue in the mouth or lip area, and the tissue that is available does not join together properly.
- A cleft lip is a physical split or separation of the two sides of the upper lip and appears as a narrow opening or gap in the skin of the upper lip.
- This separation often extends beyond the base of the nose and includes the bones of the upper jaw and/or gums.
- A cleft palate is a split or opening in the roof of the mouth.
- A cleft palate can involve the hard palate and/or the soft palate.
- In most cases, cleft lip is also present.

Symptoms:

- Eating problems: food and liquids can pass from the mouth back through the nose.
- Ear infections: increased risk of ear infections since they are more prone to fluid build up in the middle ear.
- Speech problems: the voice may take on a nasal sound
- Dental problems: children with clefts are more prone to a larger number of cavities and often have missing, extra, malformed or displaced teeth

Risk factors:

- Smoking during pregnancy
- Diabetes, obesity, an older mother
- Certain medications: anti-seizure/anticonvulsant drugs

Treatment:

- Surgical repair
  - Cleft lip repair is done when the child is 6 to 12 weeks old

- Cleft palate repair is done when the child is older, between 9 months and 1 year old.

SALIVARY GLAND DISORDERS

Mumps (Viral Parotitis)

- Viral infection caused by paramyxovirus
- Disease is contracted by droplet infection and fomites
- Children are most often affected but adults can also contract the disease.
- Incubation period is 2-3 weeks.

Clinical features:

- Fever (up to 103 F)
- Malaise, anorexia, muscular pain
- Parotid swelling may appear only on one side. Other parotid gland may be enlarged simultaneously or after some time.
- Swelling subsides in about a week.

Treatment:

- Proper hydration
- Rest
- Analgesics
- Food which encourages salivary flow should be avoided as they cause pain.
- Parotid swelling persists for about 1 week.

Prevention:

- Immunisation can be given by MMR vaccine at the age of 15 months.

Acute Suppurative Parotitis

- Most commonly seen in the elderly, debilitated and dehydrated patients.
- Dry mouth due to any cause is a predisposing factor
- Staph. aureus is the usual causative organism.

Clinical features:

- Sudden onset with severe pain and enlargement of the gland
- Movements of jaw aggravate the pain
- Opening of the stensen's duct is swollen and red and may be discharging pus.

Treatment:

- Appropriate antibiotics, preferably through i.v. route
- Adequate hydration
- Measures to promote salivary flow
- Attention to oral hygiene

Salivary calculi

- Calculi may form in the ducts of submandibular or parotid glands
- They are formed by deposition of calcium and phosphate on the organic matrix of mucin or cellular debris.
- About 90% of the stones are seen in submandibular and 10% in the parotid.

Clinical features:

- Intermittent swelling of the involved gland
- Pain due to obstruction to outflow of saliva.
- About 80% of the stones are radio-opaque and can be seen on appropriate X-rays.

Treatment:

- Stones in peripheral part of submandibular or parotid ducts can be removed intraorally, while those at the hilum or in the parenchyma require excision of the gland.

Sjogren's Syndrome

- An autoimmune disorder involving exocrine glands of the body.

Primary Sjogren's Syndrome:

- Consists of xerostomia and xero-ophthalmia
- Due to involvement of salivary and lacrimal glands.

- Parotid is most often involved.

Secondary Sjogren's syndrome:

- Consists of 3 major components:
  - Keratoconjunctivitis sicca (due to involvement of lacrimal gland)
  - Xerostomia (due to involvement of salivary glands and mucous glands of oral cavity)
  - autoimmune connective tissue disorder, usually the rheumatoid arthritis.

STOMATITIS

- The word "stomatitis" literally means inflammation of the mouth.
- Stomatitis is the inflammation of the mucous lining of any of the structures in the mouth, which may involve the cheeks, gums, tongue, lips,throat, and roof or floor of the mouth.
- This condition is frequently referred to as mucositis.
- It is characterized by mouth ulcers or sores, and pain in the mouth, associated with redness, swelling, and occasional bleeding from the affected area.
- The inflammation can be caused by conditions in the mouth itself, such as poor oral hygiene, poorly fitted dentures, or from mouth burns from hot food or drinks, or by conditions that affect the entire body, such as medications, allergic reactions, or infections.
- Stomatitis affects all age groups, from the infant to the elderly.

Causative factors:

1. Poorly fitted oral appliances that irritate the tissues of the mouth.
2. cheek biting
3. jagged teeth can persistently irritate the oral structures
4. chronic mouth breathing can cause dryness of the mouth tissues, which in turn leads to irritation

5. Diseases such as herpetic infections ,gonorrhea, measles,mumps, leukemia,AIDS
6. Protozoal infections i.e. malaria, amoebiasis
7. vit. C deficiency
8. Protein deficiency
9. nutritional deficiencies, especially of vitamin B_{12}, folate, or iron
10. Generalized or contact stomatitis can result from excessive use of alcohol, spices, hot food, or tobacco products.
11. Sensitivity to mouthwashes, toothpastes, and lipstick can irritate the lining of the mouth.
12. poor oral hygine
13. chemical irritation
14. Thrush, a fungal infection, is a type of stomatitis.
15. Allergic reactions
16. Stress e.g. oral cavity ulcers seen in dysmenorrhoea by causing dryness of mouth
17. Metabolic disorders like diabetes, hypothyroidism (by xerostomia), hyperthyroidism (due to diarrhoea), atherosclerosis(by causing b complex def.)
18. Any GIT disorders like GORD (gastric oesophageal reflux disease), dysentry etc…
19. Autoimmune disorders like lichen planus, pemphigus , SLE (systemic lupus erythmatosus), multiple sclerosis, rheumatoid arthritis, poly arthritis rumoda etc..
20. cancer treatments such as chemotherapy and radiation therapy

Symptoms

- The first symptoms may be sensitivity to spicy foods and reddened mucous membranes.
- The patient with stomatitis may also experience a dry or swollen tongue,
- difficulty swallowing, and an inability to eat or drink.
- Oral bleeding or blood in saliva
- Bad breath
- Mouth ulcers
- Sore mouth

Signs

- Reddened areas in the mouth may appear .
- The inflammation can range from mild to severe.
- As time goes on, ulceration occurs.
- If such complications as infection do not occur, stomatitis usually heals completely within two to four weeks
- It is usually a short-term condition, lasting from just a few days to a few weeks.

Diagnosis

- Diagnosis of stomatitis can be difficult.
- A patient's history may disclose a dietary deficiency, a systemic disease, or contact with materials causing an allergic reaction.
- A physical examination is done to evaluate the oral lesions and other skin problems.
- Blood tests may be done to determine if any infection is present.
- Scrapings of the lining of the mouth may be sent to the laboratory for microscopic evaluation, or
- cultures of the mouth may be done to determine if an infectious agent may be the cause of the problem.

Treatment

- local cleansing and good oral hygiene
- Avoid sharp-edged foods like tacos, peanuts and potato chips
- use of soft-bristled toothbrush and careful washing of teeth and gums
- amendment of ill-fitting dental appliances or sharp teeth by a dentist
- medication for infectious cause
- measures to overcome vitamin B12, iron or folate deficiency
- use of cotton-tipped applicator for aphthous ulcer
- low-power treatment with a carbon dioxide laser for recurrent aphthae
- use of tetracycline antibiotics or corticosteroids for aphthous stomatitis

- use of valacyclovir for stomatitis due to herpesviruses
- use of topical anesthetics like a 2% lidocaine gel or a protective paste (Orabase) or a coating agent like Kaopectate
- botanical medicine like calendula in tincture form and diluted for a mouth rinse is effective against stomatitis
- extract of leaves of Trichilia glabra (found in South America) kills viruses causing stomatitis
- Chemical or physical cautery relieves pain. Silver nitrate sticks are less effective than low-power, defocused, pulsed-mode carbon dioxide laser treatments.

Prevention:

- Rinsing the oral cavity after meals and before bedtime with a mild salt-water or baking soda and water solution will help keep the mouth clean and free of debris.
- A soft-bristled toothbrush or soft foam tooth-cleaning device should be used to keep the mouth and teeth very clean.
- Maintaining a good nutritional intake and drinking adequate amounts of fluids helps the body heal the stomatitis.
- The use of any tobacco products and alcohol should be avoided, as they can irritate the lining of the mouth.
- Avoid spicy or acidic foods, or very hot foods.
- Patients who wear dentures should remove them at night rather than leaving them in the mouth overnight, and should clean them carefully with an antiseptic solution.

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