BASICS OF EAR

Anatomy of ear

- Pinna
- External Auditory canal
- Tympanic membrane

External Ear → Middle Ear → Internal Ear → Brain

- Ossicles: Malleus, Incus, Stapes
- Labyrinth (membranous and bony part)
- Ventilation (middle ear pressure maintained)

Eustachian Tube → Pharynx

Internal Acoustic meatus (Base of Skull)
Base of the skull and cranial nerves

Cranial Nerves: Passing through (structures)
- Olfactory Nerve (CN I) → Cribiform Plate → Nose
- Optic Nerve (CN II) → Superior orbital fissure → Optic canal → Eye
- Oculomotor Nerve (CN III)
  - Trochlear Nerve (CN IV) → Superior orbital fissure → Eye
  - Abducens Nerve (CN VI)
  - Ophthalmic branch of Trigeminal (CN V → V_1)
- Maxillary branch of Trigeminal (CN V → V_2)
- Mandibular branch of Trigeminal (CN V → V₂) → Foramen ovale
- Lesser petrosal nerve
- 5th cranial nerve ganglion: Gasserian ganglion
  - Branches
  - Ophthalmic
  - Maxillary
  - Mandibular
- Depression formed by Gasserian ganglion → Meckel's cave
  - Abducens nerve (CN VI)
    - Petrous apex
    - Superior orbital fissure
  - CN involved in most intracranial pathologies
- Cranial nerves involved in any petrous apex pathology: V and VI CN

Cranial nerves: VII, VIII, IX, X

- Facial nerve (CN VII)
  - Vestibulocochlear nerve (CN VIII)
  - Internal acoustic meatus → Ear
- Inner ear
  - Present in petrous part of temporal bone
  - Connects to brain through internal acoustic meatus
- Glossopharyngeal Nerve (CN IX)
- Vagus Nerve (CN X)
- Accessory Nerve (CN XI)

- Acoustic Neuroma

Tumour arises from

Internal Acoustic Meatus (CN VII, VIII)

- Grows intracranially
  - Upper pole → CN V, VI
  - Lower pole → CN IX, X, XI

- Hypoglossal Nerve (CN XII)

Hypoglossal canal
Sigmoid sinus

- Posterior boundary of mastoid
- Any infection from mastoid goes to sigmoid sinus → Sigmoid sinus
  Thrombosis
- Sigmoid sinus $\xrightarrow{\text{Jugular Foramen}}$ Jugular bulb $\xrightarrow{\text{neck}}$ Internal jugular vein

[CN IX, X, XI]
EMBRYOLOGY OF INNER EAR

Basic structure of inner ear

- Membranous labyrinth - Has endolymph

  Surrounded by

  →

  Bony labyrinth - has perilymph

  Bony Labyrinth

  Perilymph

  Membranous labyrinth

  Endolymph

Embryology of membranous labyrinth

- Membranous labyrinth - develop from otic placode

  (surface ectoderm)

  →

  Otic pit

  Otic vesicle
From otic vesicle → Outpouchings develop
Form membranous labyrinth

Part of membranous labyrinth

Three semicircular canals

Superior / Anterior scc

Posterior scc

Lateral / Horizontal scc

Through 5 openings

Common opening

Through endolymphatic duct

Open into

Endolymphic sac

It takes 2.5–2.75 turns around modiolus (bony pyramid)

Membranous labyrinth - Functions
- Some parts of membranous labyrinth
  ↓
  undergo specialisation
  ↓
  To form sensory end organs of hearing, balance

Function
- Hearing
- Balance

Sensory end organ of hearing
- The sensory end organ of hearing is present in
  ↓
  Scala media.
  ↓
  Organ of corti
  ↓
  Apex
  ↓
  Base
  ↓
  Low frequency
  ↓
  High frequency
  ↓
  Affected early in meniere's

Sensory end organ of balance
- Present in
  ↓
  Semicircular canal
  ↓
  Utricle & saccule
  ↓
  Cristae
  ↓
  Macula.
  ↓
  For rotational acceleration
  For linear acceleration
Bony labyrinth

- Bony labyrinth formation - mesenchyme around membranous labyrinth
  ↓
  Differentiate into
  ↓
  Cartilage
  ↓
  Bone
  ↓
  Enchondral ossification

![Diagram of the bony labyrinth]

Parts of bony labyrinth

- **Three** semicircular canals - (scc)
  ↓
  Superior scc  Posterior scc  Lateral scc

- Superior SCC - Bulges on the anterior slant of petrous bone into base of skull.

  Clinical significance - Superior scc dehiscence syndrome or 3rd window syndrome

- Around utricle & sacule - vestibule
Around scala media - bony labyrinth - 2 parts

- Above scala media
- Scala vestibule
- Opens into vestibule
- Below scala media
- Scala tympani
- Connected through helicotrema

Oval window

- Oval window connects middle ear to inner ear
  - Into vestibule

  - Function - Sound transmission
    - Vibration of foot plate of stapes
    - Vibrates inner ear

- Abnormalities of oval window

  - Fixed stapes
    - Foot plate
  - If foot plate of stapes touches utricle
    - If saccule - cause vertigo
      - Otosclerosis
      - Dilated utricle
      - Saccule
      - In meniere's
        (tullio's phenomenon)
      - Foot plate is hypermobile
      - Hennebert sign
Round window

- Round window - connects middle ear to inner ear
  ↓
  Through scala tympani

- Round window - closed by
  ↓
  Secondary tympanic membrane
  (very thin, 3 layered, tympanic membrane
  like structure)

- Function - sound transmission
  ↓
  Sound waves
  ↓
  Ossicles vibrate
  ↓
  Foot plate of stapes vibrate
  ↓
  Vibrations through oval window goes into vestibule
  ↓
  Vibrations passed into scala vestibuli
  ↓
  Helicotrema
  ↓
  Scala tympani
  ↓
  Vibrations through round window goes out
  ↓
  This opposite direction of vibrations
  ↓
  Creates shearing force
  ↓
  Stimulates organs of corti in scala media.
  ↓
  Perception of sound
Clinical significance of round window

- Cochlear implant
  - Drugs injected
    - Electrodes passed into
      - Mastoid cavity
        - Into middle ear
          - Through round window
            - Placed in scala tympani
              - Electrode replaces organ of corti function
                - Stimulates 8th nerve

Cochlear aqueduct

- Cerebrospinal → Cochlear duct → Enters scala tympani
  - Fluid (CSF)
    - Forming perilymph

- Inner ear connected to brain by
  - Cochlear aqueduct
    - Internal acoustic meatus
      - Through which 1st, 8th cranial nerve pass
        - So infections can spread from brain to inner ear or vice versa
          (meningitis = labyrinthitis)
• In a child, who has developed labyrinthitis due to meningitis
  ↓
  If hearing loss is seen
  ↓
  Should do cochlear implant as early as 1 year
  ↓
  Because speech area is maximally active at 3 yrs
  ↓
  Only if listening is good
  ↓
  Speech / Language development is good

Fissula ante fenestrum

• The area anterior to fenestra of oval window
  ↓
  Remains cartilaginous – so keeps dividing on external stimuli
  ↓
  Called – Fissula ante fenestrum – fixes foot plate of stapes
  ↓
  m.c site of origin of otosclerosis

• Foot plate fixation – m.c congenital anatomy of middle ear

Congenital anomalies of inner ear

↓
≥
≥
≥

Michel aplasia

↓

Alexander aplasia

↓

Mondini aplasia

↓

Scheibe aplasia

Total non development of inner ear

↓

Dysplasia of basal turn

↓

Cochlea has 1½ turns anomaly of saccule & cochlea

No cochlear nerve organ of corti

↓

High frequency sound affected

↓

Contraindication – for cochlear implant
Endolymphatic SAC

- Utricle & saccule is drained
  ↓
  by endolymphatic duct
  ↓
  into endolymphatic sac
  ↓
  It lies in between two layers of dura.
  (meningeal & periosteal layer of dura)
  - Intradural

[Diagram of the inner ear highlighting various structures, including semicircular canals, utricle, saccule, ampulla, vestibule, cochlea, vestibular nerves, cochlear nerve, oval window, and anterior semicircular canal.]
EMBRYOLOGY OF EXTERNAL EAR AND MIDDLE EAR

- Neck of developing fetus
  - 6 Branchial arches
  - 5th one disappears

- Extodermal lining (on outside)
  - Extodermal (cleft)
  - External Auditory canal (EAC) develops from
  - 1st cleft

- Mesoderm (in middle)
  - Mesodermal Arch (branchial Arch)

- Endodermal lining (on inside)
  - Endodermal Pouch
  - 1st Endodermal Pouch
  - 2nd Endodermal Pouch (small part)

- Tubotympanic Recess
  - Mastoid antrum
  - Tubotympanic Recess
  - Eustachian Tube
  - Middle ear
  - Mastoid Antrum

- Cartilaginous part (present at birth)
  - Bony part (develops later)

- Bilateral defect in formation of Pinna (Anotia, Microtia) is associated with meatal atresia.

- Till 6 years, bone-anchored hearing aids (BAHA) used for hearing.

ENT * v2.0 * Marrow 4.0 * 2020
Reconstruction of pinna can be done with rib cartilage after 6-7 years of age (rib cartilage matures at 6 years of age)

- Meeting point of 1\textsuperscript{st} Endodermal cleft and Tubotympanic recess forms tympanic membrane (contains all 3 germ layers)
- Foot plate of stapes → Bony Labyrinth / Otic capsule

Warning: Not all points are covered in the notes, especially conceptual explanations. Please use the notes in conjunction with Marrow Edition 4 videos.
Development of pinna

mesodermal arch
↓
6 mesodermal thickenings
(Hillocks of His)
↓
From 1st Arch → 1st Hillock
Forms tragus
↓
From 2nd Arch → Remaining 5 Hillocks
Forms remaining cartilage

Preauricular Sinus
→ Fusion defect of 1st and 2nd Arch
→ Location in between tragus and ascending helix
ANATOMY OF EXTERNAL EAR

Anatomy of pinna

- Pinna: A folded piece of cartilage
- Structures:
  1. Helix
  2. Antihelix
  3. Tragus
  4. Antitragus
  5. Concha
  6. Cymba concha

Cymba concha: Cartilagenous landmark for Antrum
Macewen’s Triangle: Bone landmark for Antrum

Boundaries of mastoid antrum
- Superiorly: Dural plate
  - Separates antrum from temporal lobe
  - Temporal lobe abscess is the most common infection after middle ear infection

- Posteriorly: Sinus plate
  - Separates antrum from sigmoid sinus

- Inferiorly: Stylomastoid foramen with facial nerve

- Citelli's Angle
  - A/A/A sinodural angle
  - Angle between sinus plate and Dural plate

- Macewen's Triangle
  - Mastoid is drilled only in this particular area

- Skin → medial side → Loose skin
  → Lateral side → Tight skin

---

### Approach to middle ear and Darwin's tubercle

Approach to TM or middle ear
↓

EAC is not used:
- Cartilage (tend to collapse)
- Perichondritis (risk)
↓

Endaural Approach:
- Incision is made on Incisura terminalis (between tragus and helix)

---

**Darwin's Tubercle**

- Prominence at junction of upper ⅔ and lower ⅔, on the postero superior margin of pinna.
- Atavistic feature (primates also have prominence on pinna)
Anatomy of external auditory canal

Cartilaginous

- Lateral 1/3 → 8 mm
- Direction: upward and backwards
- Lining epithelium: same as skin
  (hair & ceruminous glands +)
- Funnce of ear: localized to cartilaginous part
- Fissure of Santorini

EAC (24 mm)

Cartilaginous - Bony

- Medial 2/3 → 16 mm
- Narrowest part: isthmus (6 mm lateral to TM)
- Direction: downwards and forward
- Lining epithelium: stratified squamous epithelium (without skin appendage)
- If foreign body lodges in isthmus, surgical instruments required for removal
- Foramen of Huschke
- Infection
  - Parotid → Base of skull
  - Usually closes by 5-6 years of age

Parotid

Base of skull

- Roof and Floor of EAC

ENT • v2.0 • Marrow 4.0 • 2020
Anatomy of tympanic membrane

- Window of middle ear
- Derived from all 3 germ layers
- Normal colour: Pearly grey / Pearly white
- Makes an angle of 55° with Horizontal
- Borders:
  - Anterior: Tilted towards middle ear (more medial)
  - Posterior: Tilted towards EAC (more accessible)
- Divided into:
  - Upper ⅓: Pars Flaccida
    (sharpnelli's membrane)
  - Lower ⅔: Pars Tensa

- M.C. site of Retraction pocket → Pars Flaccida.
- Centre of Pars Tensa is dent by tip of handle of malleus, where it attaches and forms umbo
- Cone of Light emerges from umbo, due to:
  - Obliquity of tympanic membrane.
  - Reflection of Handle
- Most mobile part: Paramedian part (periphery > central)
- Cone of light is present in antero-inferior quadrant

Identification of ear from tympanic membrane

- By observing:
  1. Cone of Light
  2. Upper tilt of tympanic membrane
Surface Area → Total 90 mm²

Effective vibrating surface Area → 55 mm²
ANATOMY OF MIDDLE EAR

Walls of middle ear

- The middle ear is a 6 walled cavity:
  - Medial wall: Common wall between middle ear and inner ear
  - Lateral wall: Common wall between external ear and middle ear
  - Anterior wall: Separates middle ear from pharynx
  - Posterior wall: Separates middle ear from mastoid
  - Roof: Separates middle ear from middle cranial fossa.

medial wall of middle ear

- Promontory
  - Bulge produced by basal turn of cochlea
  - Senses sounds of high frequency.
- Oval window
  - Covered by foot plate of stapes
  - Connects to vestibule

- Round window
  - Covered by tympanic membrane
  - Connects to scala tympani

- Lateral semicircular canal
  - Forms a bulge on medial wall

- Processus cochleariformis
  - Projection of bone from cochlea
  - Tensor tympani muscle takes a turn, move laterally & attach to handle of malleus
  - Acts as landmark for 1st genu of facial nerve
  - The geniculate ganglion is present on the 1st genu

- Facial Nerve
  - Enters medial wall: 1st Genu
  - Horizontal/Tympanic segment: Above oval window, below lateral semicircular canal
Posterior wall of middle ear

- **Aditus**
  - Ventilation of mastoid
  - Spread of infections

- **Facial Nerve**
  - Horizontal segment from medial wall
  - Takes a turn at junction of medial and posterior wall
  - Runs vertically (vertical/mastoid segment of facial nerve)

- **Chorda tympani**
  - Has its opening at posterior wall
  - Passes through middle ear → Exits through anterior wall

- **Fossa incudis**
  - Depression produced by short process of incus
- **Facial Recess (supra-pyramidal recess)**
  - Medially: Vertical segment of Facial Nerve
  - Laterally: Entry for chorda tympani
  - Superiorly: Fossa incudes
  - Placement of cochlear electrodes:

    mastoid → **Facial recess** → Middle ear → Round window → Inner ear

- **Sinus tympani (infra-pyramidal recess)**
  - Hidden area of middle ear
  - MC site of residual disease (cholesteatoma)

- **Pyramid**
  - Stapedius muscle originates from pyramid

- **Stapedius muscle**
  - Inserted into neck of stapes

**Stapedial reflex / acoustic reflex**

```
Sound 70-100 db above threshold
↓
External Auditory canal
↓
middle Ear
↓
Organ of Corti
↓
Auditory pathway
↓
Ipsilateral ear
Facial nerve
↓
Stapedius
↓
Stapedial Reflex (SR)

Contralateral ear
Facial nerve
↓
Stapedius
↓
Stapedial Reflex (SR)
```

Afferent

**Efferent (bilateral reflex)**
- Protects inner ear from noise trauma.
- Afferent → 8th Nerve
  Efferent → 7th Nerve
- 6th nerve complete palsy → Stapedial reflex absent in both ears
  7th nerve palsy of an ear → Stapedial reflex absent in that ear
- Used for NOHL (Non Organic Hearing Loss); i.e. malingering

- Pars Flaccida
  → Most mobile part of tympanic membrane.
  → MC site for retraction pocket
- Narrowest part of middle ear: mesotympanum (2 mm)
  Widest part of middle ear: myotympanum (6 mm)

Anterior wall, roof and floor of middle ear

Anterior wall

- 8/11/14 carotid wall
- 3 major structures:
  1. Opening of eustachian tube
  2. Opening for chorda tympani [Canal of Huguier]
  3. Opening for tensor tympani
ROOF

- Tegmen Tympani: Separates middle ear from temporal lobe

Floor:

- Jugular bulb
- 9th, 10th, 11th cranial nerve
- Jacobson's nerve enters middle ear through floor → Forms tympanic plexus on promontory
Middle ear cavity

- Divided into 3 parts
  - Epitympanum / Attic
  - Meso tympanum
  - Hypotympanum

- Scutum:
  - The bone above pars flaccida forming lateral wall of attic

- Prussak’s space
  - The most common site of primary cholesteatoma.
  - It is the area medial to Pars flaccida.
ANATOMY OF INNER EAR

Structure of Scala media

- **Reissner’s membrane** - Separates scala vestibule & scala media.
- **Basilar membrane** - Separates scala media & scala tympani.
- **Stria vascularis** - Present on lateral surface of scala media, site of production of endolymph.
- **Perilymph** - Present in scala vestibule & scala tympani.

Production of endolymph
- **Endolymph production** - by stria vascularis
  
  ↓

  Has Na⁺/K⁺ 2Cl⁻ channels
  
  ↓

  which maintains electrochemical gradient of endolymph
  
  ↓

  Electric potential + 65 mV
• In Perilymph - Na\(^+\) is high (like extracellular fluid)
• In Endolymph - K\(^+\) is high (like intracellular fluid)
• Furosemide (loop diuretic) - Acting on Na\(^+\)/K\(^+\) 2Cl\(^-\) channel in kidney acts in stria vascularis also
• The endolymph electro chemical gradient in utricle & saccule is maintained by Dark Cells

**Ototoxic drugs**

• Organ of Corti - Present on basilar membrane has two types of hair cells (inner & outer hair cells)

• In cristae & maculae - Type I & II hair cells are present

**Drugs**

A - Aminoglycosides  
Analygesics  
Antimalarials  

\(\rightarrow\) Side effect

\(\downarrow\) Irreversible hearing loss

V - Vancomycin  
C - Cisplatin  
D - Loop Diuretic (Furosemide)

**Aminoglycosides**

\(\downarrow\) Cochleotoxic drugs  
\(\downarrow\) Vestibulotoxic drugs

\(\downarrow\) Act on inner & outer hair cells

K - Kanamycin  
A - Amikacin  
N - Neomycin - most ototoxic

**Topical use only**

\(\downarrow\) Act on Type I & II hair cells

- Streptomycin
- Gentamycin - most specific

\(\downarrow\) To destroy the vestibule part

ENT • v2.0 • Marrow 4.0 • 2020
Organ of corti – inner and outer hair cells

Organ of corti

- Inner hair cell
  - Arranged in one row
  - Less in number
  - Less prone to damage
  - Afferent nerves are present

- Outer hair cell
  - Arranges in 2-3 rows
  - More in number
  - More prone to damage by drugs, noise
  - Efferent nerves are present
  - Produces OAE (oto acoustic emission)

Transduction

- When basilar membrane moves → Hair cells moves

  Hair cells has stereocilia.

  Sterocilia – arranged in order of their size (lowest to highest)

  The highest (upper most) stereocilia is attached to

  Tectorial membrane does not move

  Creates shearing force

  All the stereocilia moves towards highest stereocilia.

ENT * v2.0 * Marrow 4.0 * 2020
The tips of stereo cilia are connected to each other called - Tip links
↓
The tips have $K^+$ channels and they open
↓
$K^+$ enters into hair cell from endolymph
↓
Voltage gated $Ca^{2+}$ channels open - $Ca^{2+}$ influx
↓
Causes release of excitatory neurotransmitter glutamate

Auditory pathway

Inner hair cell
↓
Afferent nerve
↓
Cochlear nerve
↓
Spiral ganglion
(exits through internal acoustic meatus)
↓
Brain stem (ventral and dorsal cochlear nuclei)
  S - Superior olivary complex
  ↓
  L - Nucleus of Lateral lemniscus
  ↓
  I - Inferior colliculus
  ↓
  m - Medial geniculate body
  ↓
Auditory cortex (area-4) - Superior temporal gyrus
* The auditory pathways of both ear are interconnected
  ↓
  Important for localization of sound
  ↓
  Interconnection start at the level of superior olivary complex

* Superior olivary complex
  ↓
  Starting point for localization of sound
  ↓
  Centre for stapedial reflex (interconnection of VIII nerve to VII nerve)
  ↓
  Response to high intensity sound

Structure of cristae and maculae

Cristae & Maculae

- Type I hair cells
- Type II hair cells
• In maculae - Hair cells project into gelatinous matrix
  ↓
  Covered by calcium carbonate crystals (CaCO₃)
  ↓
  Called - otoliths
  ↓
  Responsible for linear acceleration

• Following trauma / pathology - Otolith gets dislodged
  ↓
  Goes into semicircular canal (SCC) (m.C-Posterior semicircular canal)
  ↓
  Stimulates SCC and causes vertigo
  ↓
  After sometime, CaCO₃ crystals settle due to gravity - ↓ res vertigo

  ↓
  Head movement causes vertigo again (cycle repeats)
  ↓
  BPPV - Benign Paroxysmal Positional vertigo (m.C - vertigo)
  ↓
  C/F - vertigo on changing posture Lasting for few seconds
Vestibular pathway and internal acoustic meatus

Cristae & maculae
\[ \downarrow \]
vestibular nerve arises
(superior & inferior vestibular nerve)
\[ \downarrow \]
Exits through internal acoustic meatus
\[ \downarrow \]
maintain balance of a person

Structures passing through internal acoustic meatus

<table>
<thead>
<tr>
<th>Anterior</th>
<th>Posterior</th>
</tr>
</thead>
<tbody>
<tr>
<td>Facial nerve</td>
<td>Superior vestibular nerve</td>
</tr>
<tr>
<td>Bill bar</td>
<td>Inferior vestibular nerve</td>
</tr>
<tr>
<td>Cochlear nerve</td>
<td>Singular nerve</td>
</tr>
</tbody>
</table>
Internal acoustic meatus - An opening on petrous bone (posterior slant) facing posterior cranial fossa.

- Facial nerve and vestibulocochlear nerve passes through it.

Internal acoustic meatus

- Superior part
  - Bill bar - crest of bone divides superior part into two
  - Nerves - Facial nerve superior vestibular nerve

- Inferior part
  - Nerves - Inferior vestibular nerve singular nerve (branch of inferior vestibular nerve cochlear nerve)

- Singular nerve - Supplies posterior semicircular canal
- Acoustic neuroma - M.C benign tumor of cerebello pontine angle

It Arises from inferior vestibular nerve in internal acoustic meatus

The upper pole of the tumor involves - 5, 6 cranial nerves

The lower pole of the tumor involves - 9, 10, 11 cranial nerves
Nerve supply of pinna

- In inner ear - no sensory nerve supply - no pain
- In pinna/external auditory canal (EAC)
  - Nerve supply is along with the origin of skin covering the cartilage

C₁ - Greater Auricular nerve
  - Branch of cervical plexus - C₂ - C₃
  - Supplies greater part of pinna (both lateral & medial surfaces)

C₂ - Lesser occipital nerve
  - Supplies small part posteriorly

A - Auriculo temporal nerve
  - Branch of mandibular division of trigeminal nerve
  - Supplies tragus and small part of helix

A - Arnold's nerve / Alderman's nerve
  - Auricular branch of vagus (X) nerve

- Facial nerve - Supplies postero superior part of external acoustic meatus

Nerve supply of external auditory canal
- A - Auriculo temporal nerve
- A - Arnold nerve

Warning: Not all points are covered in the notes, especially conceptual explanations. Please use the notes in conjunction with Marrow edition 4 videos.
Nerve supply of Tympanic membrane

Lateral surface - A - Auriculo temporal nerve
   A - Arnold's nerve

Medial surface - Jacobson nerve
   ↓
   Branch of Glossopharyngeal nerve

**Referred pain of pinna**

Greater Auricular nerve (C3, C3) → Referred pain from
Lesser occipital nerve (C2)  ↓ Spine

Auriculo temporal nerve - Temporo mandibular joint, dental conditions, anterior 2/3 tongue (lingual nerve)

Arnold's nerve - Larynx

Glossopharyngeal nerve - Posterior 1/3 tongue, tonsils

Alderman's nerve

* A/W/A Arnold's nerve
* Before any party/Concert
  ↓
  spirit is instilled into ear
  ↓
  Stimulates vagus nerve
  ↓
  ↑appetite (↑sed gastric secretions)
Audiology and Evaluation - Tuning Fork Tests

Normal pathway

\[
\text{Pinna} \downarrow \quad \text{External auditory canal} \downarrow \quad \text{Tympanic membrane} \downarrow \quad \text{Ossicular chain} \downarrow \quad \text{Foot plate of stapes} \downarrow
\]

\[
\text{Organ of corti} \rightarrow \text{Inner ear} \rightarrow \text{Sensory pathway} \rightarrow \text{Neural pathway} \rightarrow \text{Auditory cortex}
\]

Functions of middle ear

1. Dampening of loud sounds - by stapedial reflex
2. Impedance matching a/k/a Transformer action
   * Amplification of sound - Overcome reflection of sound due to change in medium (air \rightarrow Fluid)

Amplification of sound due to:

- Areal or Hydraulic ratio:
  Sound travels from tympanic membrane (larger surface area) to foot plate of stapes via ossicular chain (smaller surface area)
Normal surface area of Tm: 90 mm²
Effective vibratory area of Tm: 55 mm²
Foot plate area: 3.2 mm²

\[
\text{Areal ratio} = \frac{\text{Vibratory area of Tm}}{\text{Foot plate area}}
\]

= 17 : 1

b) Lever ratio
Long process of incus \(\rightarrow\) 1.3 times smaller than handle of malleus

\[
\text{Lever ratio} = 1.3
\]

\[
\text{Total transformer ratio} = 17 \times 1.3
\]

= 22

Phase difference:
Difference in phase (compression or rarefaction) at oval and round window
Prevents noise cancellation and hearing loss

**Tuning fork tests**

- m.C frequency used: 512 Hz (better heard)
- Ear sensitive to: 20 Hz to 20,000 Hz
- Speech frequencies: 500 Hz, 1000 Hz, 2000 Hz
- Tuning fork of frequencies less than 512 Hz \(\rightarrow\) used to assess vibration

**Air conduction and bone conduction**

Air conduction (AC)
- measure of conductive + Sensory + Neural pathway
- most important part of conductive pathway: middle ear
Bone conduction (BC):

- measure of sensory + neural pathway
  - vibrating tuning fork placed on mastoid
    - Bony cochlea and cochlear fluids stimulated
      - Basilar membrane and hair cells stimulated
        - Neurotransmitter (glutamate) released
          - Auditory Pathway
            - Auditory cortex

Air conduction → Better than bone conduction because amplified sound reaches sensory neural pathway

**Rinnes test**  
Air conduction (AC) and Bone conduction (BC) tested and compared

- AC > BC  
  - Rinnes  
    - Normal ear
    - Sensorineural hearing loss (SNHL)

- BC > AC  
  - Rinnes  
    - Conductive hearing loss (CHL) (>15-20 dB)
    - Severe SNHL
      - BC > AC due to stimulation of opposite cochlea. (False negative Rinnes)
      - Sudden severe SNHL due to viral infection/idiopathic. (sudden edema, ischemia, compression of inner ear is seen)
Weber's test

- To differentiate between conductive and severe SNHL.
- Tuning fork placed on forehead, vertex or upper teeth.
- Tests bone conduction.

\[\text{weber's test} \downarrow\]
\[\text{Assess lateralization of sound} \downarrow\]
\[\begin{align*}
\text{In SNHL} & \quad \text{In conductive loss} \\
\downarrow & \quad \downarrow \\
\text{Sound lateralized to} & \quad \text{Sound lateralized to} \\
\text{opposite side} & \quad \text{same side (defective} \\
\text{of defect} & \quad \text{ear)} [\text{external noise} \\
& \quad \text{not heard} . . . . . . \text{Tuning} \\
& \quad \text{fork conduction better}] \\
\end{align*}\]

"SOCS"

SNHL- lateralized to opposite side
Conductive- lateralized to same side

- Serous otitis media.
  Otosclerosis \{Conductive hearing loss\}
- Meniere's disease
  Acoustic neuroma \{Sensorineural hearing loss\}

Absolute Bone Conduction (ABC) test

- Bone conduction of patient compared to that of examiner (assumed to be normal).
- Tragus occluded \(\rightarrow\): conductive pathway excluded.
- Assess only bone conduction (for SNHL assessment).

\[\begin{align*}
\text{Absolute bone conduction} & \downarrow \\
\downarrow & \downarrow \\
\text{Same as examiner} & \text{\(\downarrow\) / shortened} \\
\downarrow & \downarrow \\
\text{Normal} & \text{Bone conduction} \\
\text{Conductive hearing} & \downarrow \\
\text{loss not assessed} & \text{SNHL} \\
\end{align*}\]
Schwabach test

- Reconfirm conductive and SNHL
- Similar to AEC test, but tragus not covered

\[\text{Compare bone conduction of patient to that of examiner}\]

- Same as examiner
  - Normal

- Shortened
  - BC decreased
  - SNHL
  - Conductive hearing loss

- Lengthened
  - (patient BC > Examiner)

Gelles test

- Test for ossicular fixation (otosclerosis)
- Siegel's speculum used \(\rightarrow\) Alters the external auditory canal pressure

- Hearing varies with change in pressure \(\rightarrow\) Positive Gelles
- No change in hearing with pressure variation \(\rightarrow\) Negative Gelles test \(\rightarrow\) Ossicular chain fixation
### AUDIOLOGY & EVALUATION – AUDIOGRAM (PTA)

<table>
<thead>
<tr>
<th>Conditions</th>
<th>Hearing loss</th>
</tr>
</thead>
<tbody>
<tr>
<td>* Complete obstruction of External Auditory canal (EAC)</td>
<td>→ 30 db</td>
</tr>
<tr>
<td>* Tympanic membrane (TM) Perforation</td>
<td>→ 10-40 db depending on the site &amp; size of perforation</td>
</tr>
<tr>
<td>&quot;larger the perforation, more is the hearing loss&quot;</td>
<td></td>
</tr>
<tr>
<td>* Perforation exposing both oval window &amp; round window at the same time</td>
<td>→ Hearing loss ↑, due to loss of phase difference</td>
</tr>
<tr>
<td>* Tympanic membrane perforation with ossicular discontinuity</td>
<td>→ 40 db</td>
</tr>
<tr>
<td>* Ossicular discontinuity without TM perforation</td>
<td>→ 55 db due to loss of alternate pathway</td>
</tr>
<tr>
<td>* Complete foot plate of stapes fixation (FPS)</td>
<td>→ 60 db (Maximum CHL that one can have)</td>
</tr>
</tbody>
</table>

### Pure Tone Audiometry (PTA)

- PTA – subjective test
- Tuning fork test – subjective test
- Speech frequency = \( \{500, 1000, 2000\} \) Hz

---

ENT • v2.0 • Marrow 4.0 • 2020
### Symbols in Audiogram:

<table>
<thead>
<tr>
<th>Modality</th>
<th>Right</th>
<th>Left</th>
</tr>
</thead>
<tbody>
<tr>
<td>Air conduction masked</td>
<td>○</td>
<td>×</td>
</tr>
<tr>
<td>Air conduction unmasked</td>
<td>△</td>
<td>□</td>
</tr>
<tr>
<td>Bone conduction unmasked</td>
<td>&lt;</td>
<td>&gt;</td>
</tr>
<tr>
<td>Bone conduction masked</td>
<td>□</td>
<td>▼</td>
</tr>
<tr>
<td>No response</td>
<td>▼</td>
<td>▼</td>
</tr>
</tbody>
</table>

During localization of sound, ear along direction of sound hears it louder → Loss of 40 db on the opposite ear (sound is less loud)

Mode of assessment of direction of sound

---

### Audiogram

- Right Ear Bone conduction (BC)
  - Unmasked
  - Right ear Air conduction (AC)
    - Masked
      - ⋮ющая → BC
      - → AC
  - Impression → Right ear

In bone conduction → Subject responding at 0 dB [means no hearing loss]

- Threshold of hearing = 0 db − 25 db

Interpretation: Hearing of patient is same as reference hearing, which we consider as reference of hearing.
Grades of hearing loss

- ASHA [American Speech & Hearing Association]
  - Commonly followed
    - Normal → 25 dB
    - Mild hearing loss → 26-40 dB
    - Moderate → 41-55 dB
    - Moderately severe → 56-70 dB
    - Severe → 71-90 dB
    - Profound → > 90 dB

Indication for cochlear implant

- WHO - Grading of hearing loss:
  - Normal → 0-25db
  - Mild → 26-40db
  - Moderate → 41-60db
  - Severe → 61-80 db
  - Profound → > 81 db

Note: if bone conduction is O → Sensory - Neural Pathway O
  if air conduction is O → Conductive & sensory - neural pathway

Audiogram: discussion

- O ear: Air conduction with 10 dB hearing loss = O
  Conductive + sensory + Neural pathway → O
  i.e., if air conduction O
  BC is also considered normal, if no BC curve is given
- Left ear:
  Air conduction → ✔
- Right ear:
  AC → ☐
  Both ears — Normal

- Left ear:
  BC — ☐
  AC — 50 db hearing loss

- Gap between AC & BC → Conductive hearing loss
  AB Gap → > 15 db (indicates CHL)

Impression: Rt ear otosclerosis (conductive hearing loss seen)
- **Ear:**
  - BC - defective
  - AC - defective

Has equal hearing loss

\[ \implies \text{R SNHL} \]

In sensorineural hearing loss, AB gap < 15 db

- **Ear:** AC → BC
  - Ear: AC → 

If BC curve not given

\[ \implies \text{Assume to be } \frac{\text{Impression - R conductive hearing loss}}{\text{SNHL}} \]

- **Ear:** SNHL at high frequencies
  - No AB gap
  - Down sloping / Descending Audiogram: Seen in high frequency hearing loss Causes - Presbycusis, Noise Trauma, Ototoxicity

(These 3 are B/L high frequency hearing loss)
- ⚫ Ear \(\rightarrow\) SNHL

  * Up sloping Audiogram:
    
    Seen in low frequency hearing loss
    cause - meniere's disease

**Audiogram: How to read?**

- which ear \[\begin{array}{c}
  R \\
  L \\
  Both
\end{array}\]

- \(\%\) or Not \(\rightarrow\) \(\%\) = \(<25\)db Hearing loss

- AB Gap \(\rightarrow\) \(\bigcirc\) in conductive HL

  \(\bigcirc\) in SNHL

- If SNHL \(\rightarrow\) up sloping \(\rightarrow\) low frequency hearing loss (meniere's)

  \(\rightarrow\) down sloping \(\rightarrow\) High frequency hearing loss

  (Presbycusis, NIHL, ototoxicity)

- Mix at 4000 Hz = NIHL (Noise induced Hearing loss)

- Dip at 2000 Hz = Otosclerosis [carharts notch]

- In mild frequency Hearing loss /speech frequency hearing loss-

  "U" shaped Audiogram (or) Trough/
  cookie bite (seen in congenital SNHL)

- ⚫ ear: AC - \(\%\)

- ⚫ ear: SNHL

  up sloping Audiogram \(\bigcirc\)

  Low frequency Hearing loss

  meniere's

**Frequency in Hertz**

**Hearing loss in db**

ENT \(\cdot\) v2.0 \(\cdot\) Marrow 4.0 \(\cdot\) 2020
Area of 4000Hz → Dip
For both ears, indicating NNL

- Seen in NNL
- This is the first change in NNL, later at high frequencies, it becomes downsloping

Audiogram: Otosclerosis

- Ear BC → , indicating S+P pathway is normal but dips at 2000Hz
  Carhart's Notch
  Due to fixation of Foot plate of stapes(FPS)
  Otosclerosis

AC → No notch, all frequencies are defective
AB gap is present (> 15) indicates CHL

- Dip only in BC, Not in AC:
  - Normally additional vibration reach cochlea when the FPS vibrates along with skull via BC
  - Fixed FPS → No additional vibrations reach cochlea
    Frequency of FPS is 2000 Hz → Carhart's notch seen at 2000 Hz
    In air conduction → Conduction affected across TM to FPS
      : all frequencies affected
Audiogram: Bilateral conductive hearing loss

- Ear:
  - BC → \( \equiv \)
  - AB gap → \( \oplus \) = CHL (conductive Hearing loss)
  - AC → Defective

Impression: Bilateral CHL
AUDIOLOGY & EVALUATION – TYPANOMETRY, BERA & OTHERS

Defect in middle ear
+ Tympanic membrane (TM)
③ appearance

- Ossicular discontinuity
- Ossicular fixation
- Serous otitis media (som)

Impedance audiometry

- used to assess middle ear condition when TM is appearing normal & intact
- 3 modalities
  → Tympanometry
  → Stapedial Reflex → Test for malingering

- Tympanometry:
  Probe A: Passes sound across middle ear
  Probe B: Changes the pressure of external auditory canal
  ↓
  To see movements of tympanic membrane
  Probe C: Pick up the reflected sounds

- Excessive movement of TM: Ossicular discontinuity
- ↓ movement of TM: Ossicular fixation, fluid in middle ear
- Normal middle ear pressure = Normal functioning of Eustachian Tube (ET)
  ↓
  Not obstructed
Tympanometry: graph

- Compliance = Ease of mobility of Tm
  \(\downarrow\) resistance = \(\uparrow\) compliance

Graph A \(\rightarrow\) N middle ear
- \(A_0\) \(\rightarrow\) Ossicular discontinuity
- \(A_s\) \(\rightarrow\) Stiffness \(\rightarrow\) Ossicular fixation \(\{\text{Causes: Otosclerosis, Tympanosclerosis}\}\)
- \(B\) \(\rightarrow\) Fluid in middle ear - Serous Otitis Media (SOM)
- \(C\) \(\rightarrow\) Early stage of ET obstruction

<table>
<thead>
<tr>
<th>Type of curve</th>
<th>A</th>
<th>As</th>
<th>Ad</th>
<th>B{dome shape}</th>
<th>C</th>
<th>Flat</th>
</tr>
</thead>
<tbody>
<tr>
<td>Compliance</td>
<td>N</td>
<td>(\downarrow)</td>
<td>(\uparrow)</td>
<td>(\downarrow)</td>
<td>N</td>
<td>(\Theta)</td>
</tr>
<tr>
<td>middle ear pressure</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>(\downarrow/\uparrow)</td>
<td>(\Theta)</td>
<td>(\Theta)</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>N</td>
<td>oto/ tympanosclerosis</td>
<td>ossicular discontinuity</td>
<td>SOM</td>
<td>early ET obstruction</td>
<td>Tm perforation Extreme SOM</td>
</tr>
</tbody>
</table>

Warning: Not all points are covered in the notes, especially conceptual explanations. Please use the notes in conjunction with Marrow Edition 4 videos.
Test of ET functioning

Tympanometry:
- most reliable test for ET function
- Objective test

Valsalva maneuver:
- Test for ET function
- Close nostril → Blow air into mouth → Try to exhale against closed nostrils

→ ○ = Feel popping of eardrum

Pilitzerisation:
- Close 1 nostril & via. other nostril with a politzer bag ↑ pressure
→ If ET ○ = Feel popping of Tm

Eustachian Tube

Catheterization:
- Usually not done to assess ET functioning
- Used for foreign body removal
Test to differentiate: Sensory v/s Neural hearing loss

- **Recruitment:**
  - It is abnormal ↑ in loudness of loud sound
  - In B:
    - Sound heard in left ear at 15db will be heard in right ear at 10db.
    - 40db, at 80db it is heard equally at both ears at the same time.

### Table:

<table>
<thead>
<tr>
<th></th>
<th>RT.</th>
<th>Lt.</th>
<th>RT.</th>
<th>Lt.</th>
</tr>
</thead>
<tbody>
<tr>
<td>90</td>
<td>90</td>
<td>90</td>
<td>80</td>
<td>80</td>
</tr>
<tr>
<td>80</td>
<td>70</td>
<td>70</td>
<td>60</td>
<td>60</td>
</tr>
<tr>
<td>70</td>
<td>60</td>
<td>60</td>
<td>50</td>
<td>50</td>
</tr>
<tr>
<td>60</td>
<td>50</td>
<td>50</td>
<td>40</td>
<td>40</td>
</tr>
<tr>
<td>50</td>
<td>40</td>
<td>40</td>
<td>30</td>
<td>30</td>
</tr>
<tr>
<td>40</td>
<td>30</td>
<td>30</td>
<td>20</td>
<td>20</td>
</tr>
<tr>
<td>30</td>
<td>20</td>
<td>20</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>20</td>
<td>10</td>
<td>10</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>10</td>
<td>0</td>
<td>0</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Impression:** as loudness ↑, patient hears better in right ear.
**Recruitment** (+) → Cochlear deafness → Meniere's

- **SISI:** Short Increment Sensitivity Index
  - If 70-100% of 1db increments is identified correctly → Abnormal ear → Cochlear deafness, Meniere's

**Oto – Acoustic Emission (OAE):**

- Due to activity of outer hair cells
- If OAE (+) → Normal cochlea.
  - **Retrocochlear hearing loss (neural):**
    - Outer hair cell more in number
    - Outer hair cell more prone to damage by noise trauma / Ototoxicity

- Use:
  * Monitor ototoxicity
  * Finding early noise induced hearing loss
- Screening hearing in neonates → Cochlear implants
  → Best modality = OAE

**Types of OAE:**
- Spontaneous OAE → Activity of outer hair cells due to noise in the surroundings
  * Absent in 50% of individuals
- Evoked OAE (EOAE)
  
  Transient EOAE
  Distortion product OAE

  used for screening in neonates
  monitor ototoxicity

- Pre requisite for OAE →
  * No conductive hearing loss > 35db
  * No defect in External / Middle ear
  * Wax free ear

**BERA**

Brain stem Evoked Response Audiometry:
- Best investigation:
  * To differentiate cochlear & retro cochlear hearing loss
  * Find out hearing loss in infants
  * Screening neonates in ICU
  * Retro cochlear hearing loss
    → Acoustic neuroma
  * Malingering (Non Organic hearing loss)

**Waves of BERA:** 7 in number
1 → Cochlear nerve (distal part)
2 → Cochlear nerve (proximal part)
   → Towards brain stem
3 → Cochlear nuclei
4 → Superior olivary nucleus
V → Lateral lemniscus → Largest / most prominent wave
VI → Inferior colliculus

Latency = time at which each wave appear
- Defect in inner ear → wave I appears late
- Latency between I & III ↑ → wave propagation ↓
  ↓
  lesions like tumor
- MC site of Acoustic Neuroma → Inferior vestibular nerve (Internal Acoustic meatus)
- Acoustic Neuroma on BERA:
  - ↑ latency between wave I & V
  - ↑ Interaural latency of wave V → > 0.2ms

Electro cochleography (ECOG)

- Invasive test
- Best investigation for Cochlear hearing loss / Lesion → Meniere's
- Electrical activity of whole or organ of corti
- Probe passed through TM & placed at medial wall (promontory area)
  • 1st to detect → Activity of outer hair cell
  • 2nd to detect → Summating Potential (SP) →
    ↓ Sum of inner & outer hair cell activity
  • 3rd to detect → Action potential in the nerve (AP)
  If \[ \frac{SP}{AP} \] ⇒ > 45% or 0.45 → Meniere's

Speech audiometry

- To determine candidacy of hearing implants
  speech reception threshold - loudness at which patient hears speech properly
  
  IE: Correctly identify ≥ 50% of correctly spoken words
  It is 30dB ± 10db PTA Threshold
Discrimination score - Function of the nerve to discriminate between words easily

If poor → Suggestive of neural deafness

Area → Word recognition score

Graph of loudness v/s % of words correctly recognized by subject

Roll over phenomenon:

Indicate nerve fatigue

↓

Neural deafness

% of correctly identified words

\( N = 100\% \)

Conductive hearing loss

Cochlear deafness

Roll over phenomenon

Loudness

ENT • v2.0 • Marrow 4.0 • 2020
VESTIBULAR PHYSIOLOGY AND LOCALISATION OF LESION – 1

Vertigo

![Vertigo Diagram]

Fistula test

- Normal condition → small changes in pressure is not transmitted from middle ear to inner ear → No vertigo
- Fistula between middle ear and inner ear → small changes in pressure → vertigo

Fistula test

- Positive
  → If there is a fistula in 1, 2, 3 or 4
  → Fenestration operation
    - A fenestra is made over lateral semicircular canal

- False negative fistula test
  → Due to:
    1. Fistula covered by cholesteatoma
    2. Dead labyrinth
• False Positive Fistula Test
  → Hennebert sign
  - Hypermobility of foot plate of stapes
  - Seen in:
    * Congenital syphilis
    * Superior semicircular dehiscence

Circumscribed Labyrinthitis
• Fistula of medial wall → Inner ear infection localized in a circumscribed area around fistula.
• Fistula Test → Positive

Benign paroxysmal positional vertigo
• m.C cause of peripheral vertigo
• Should be ruled out 1" if a patient presents with only vertigo

Otolith (CaCO₃ crystals of macula) in utricle
↓
lodges into posterior semicircular canal
↓ on movement
semicircular canals are activated
↓ experiences vertigo
↓ Later otolith settles down (gravity)
vertigo disappears
↓ when head is moved again, cycle repeats
• Diagnosis:
  → Dix Hallpike manoeuvre
    - Nystagmus seen

  - Change of posture causes vertigo

• Treatment:
  → Epley’s maneuver
    - Brings back otolith from posterior semi circular canal by positional manoeuvre
    - Series of 5 sequential positions of head movements.
VESTIBULAR PHYSIOLOGY AND LOCALISATION OF LESION –2

**Nystagmus**

* Nystagmus: Involuntary oscillatory movements at eye ball

<table>
<thead>
<tr>
<th>Nystagmus</th>
<th>Central</th>
<th>Peripheral</th>
</tr>
</thead>
<tbody>
<tr>
<td>* Latency*</td>
<td>➤</td>
<td>➤</td>
</tr>
<tr>
<td>* Duration*</td>
<td>Not limited</td>
<td>limited to 1-1½ min</td>
</tr>
<tr>
<td>* Fatigability*</td>
<td>➣</td>
<td>➤</td>
</tr>
<tr>
<td>* Optic fixation*</td>
<td>Does not disappear</td>
<td>Disappear</td>
</tr>
<tr>
<td>* Torsional component*</td>
<td>Pure - Horizontal / vertical / Torsional</td>
<td>➤ with horizontal/ vertical</td>
</tr>
<tr>
<td>* Direction*</td>
<td>Changing</td>
<td>Fixed</td>
</tr>
</tbody>
</table>

**Inner ear function: balance**

- Cristae
- Vestibular Nerve
- Macula

Centre
- Cortex (cerebellum)
- Vestibular Nucleus
- Eye
- Spinal system
  - Vestibuloocular Reflex
  - Vestibulospinal Reflex

In UMN lesion, central lesion ➤, reflexes are hyperactive (brisk)
- Direction of nystagmus is always towards the fast component
• In left vestibular lesion [destructive type of lesion]
  → Right vestibular system, push eyeball to left side = Slow component
  • Brain will bring eye back to centre = Fast component

• In right vestibular lesion [irritative type of lesion]
  → Right vestibular system, push eyeball more to the left = Slow component
  • Brain will bring it back to centre = Fast component

**Direction of nystagmus**

• Direction of nystagmus is towards the fast component
• Direction is always towards the active/hyperactive ear
• If nystagmus is towards right side, \( \bigcirc \) side active
  ie: \( \bigcirc \) side hypoactive
  \( \bigcirc \) side hyperactive

• Differential diagnosis for \( \bigcirc \) side nystagmus:
  - Destructive/hypoactive lesion of \( \bigcirc \) side
  - Irritative/hyperactive lesion of \( \bigcirc \) side

**Hallpike's test / Dix - Hallpike manoeuvre**

• Patient is in sitting position, then made to lie down with head tilting to one side
• The labyrinth on the side of the tilt gets stimulated
• Nystagmus \( \bigcirc \) when the defective canal is in the dependent position

**Vestibular neuritis**

• Hyperactive lesion → Nystagmus towards the same side
• Alexander's law →
  * "Nystagmus increases as the person looks towards side of lesion"
  * For peripheral nystagmus only
- Nystagmus disappears if patient looks to the opposite side or fixes the eye
- Torsional component

**Gaze evoked nystagmus**

- Direction changing nystagmus; due to defect in vestibulo ocular reflex (central or peripheral pathology)
- Spontaneous nystagmus - differentiate between vestibular neuritis (peripheral) and cerebellar infarct (central)

**Frenzel Glasses:**
- Used to remove optic fixation
  - Peripheral nystagmus gets enhanced
  - High diopter glasses
  - Gives a magnified look of the eye

**HINT test**

- Head Impulse Nystagmus Test (HINT)
- To test vestibulo ocular pathway in relation to head movements
- Specific test for periphery / inner ear functioning
- brisk movement of patient head to one side with their eyes fixed onto the doctor's nose.
  - = eye is fixed to the doctor's nose → HINT
  - Vestibulo ocular function if abnormal = Catch-up

**Catch-up saccades = Fast movement of the eye towards the end**

**Caloric test**

- Specific test for inner ear functioning
- Water of different temperature is instilled inside the ear
  - Induces conventional currents in inner ear
  - Stimulate inner ear
- Test for "lateral semicircular canal" (LSC)
- LSC maximally responsive in vertical position
- Maximally responsive positions of LSC:
  - Patient in lateral supine position and the head elevated to 30°
  - Patient is made to sit straight and tilt his head back by 60°

Hence by above positions, LSC become vertical

Types of caloric Test:

i) Fitzgerald Hallpike maneuver / bithermal caloric test
ii) monothermal / cold caloric test
↓
Done to check brain stem function in comatose patients

Caloric test: Fitzgerald Hallpike maneuver

* Temperature used is ± 7° of body temperature (37°)
- **Response** is Cold stimulus → Labyrinth hypoactive
  warm stimulus → Labyrinth hyperactive

- **Order of testing:**
  1. ear warm → ② ear warm
  2. ① ear cold ← ② ear cold

- If cold water in ② ear → Nystagmus to left
  ie: - Fast component of nystagmus - left
  - Slow component - Right
  - Direction of nystagmus - left

**VEMP** 00:54:33

Vestibular evoked myogenic potential (VEMP):

- **Eye**
  - **vEMPω**
  - **Foot plate of stapes**
  - **Oval window**
  - **VEMPω = Ocular vEMP**
  - **VEMPγ = Cervical vEMP**

Loud noise → sudden movement of foot plate → saccule ← utricle

- sternocleidomastoid
- Eye
- Relaxes
- Blink

**vEMP**

Significance: Helps to know the nerve functioning
- mc site of origin of acoustic neuroma → inferior vestibular nerve
- vestibular destruction → VEMP
- Acoustic neuroma from IVN → VEMP (cervical)
DISEASES OF EXTERNAL EAR

Diseases of pinna

Cauliflower ear
- **Hematoma of pinna**
- **Cause** - Trauma (MC in Boxer's)
- **A/r A Boxer's ear or wrestler's ear**
- **Treatment (Rx)**
  - Incision & Drainage
- **If the hematoma is fibrosed**
  - No Rx needed
  - Rx only for cosmetic purpose

Perichondritis
- **Inflammation of perichondrium**
  - In the cartilage of pinna except Lobule
- **Cause** - Infection of traumatic/surgical wound by **Pseudomonas**
- **To avoid cartilage damage in surgery**
  - Incision is given at Incisura terminalis
- **C/F** - Red, hot, painful pinna
- **Rx** - Ciprofloxacin, Cefoperazone, Ceftazidime, newer penicillins

Infections of external auditory canal - Otitis externa

Malignant otitis externa
- **Infective condition caused by Pseudomonas**
  (Not a malignancy but mimicking malignancy)
- **C/F** - Severe pain
On examination - Granulations
- Necrosis (erosion)
- Cranial nerve involvement
  (7, 9, 10, 11)

Earliest to be involved
↓
→ nerve (Facial nerve)

Osteomyelitis of base of skull, can occur through fissure of Santorini
↓
(Skull base osteomyelitis)

Malignant otitis externa – Investigations & treatment

• Seen in - Elderly, immunocompromised (diabetic, on steroids or immunosuppressive drugs)

Investigations
• For early diagnosis - Bone Scan
  ↓
  Technetium $^{99m}$ Sca.
  ↓
  Shows increased uptake due to osteoblastic activity

Biopsy - No mitotic activity seen (only granulation tissue is seen)

Management
• Antibiotics against pseudomonas
  ↓
  • Ciprofloxacin (ora)
  • 3rd generation cephalosporins
  • Newer penicillins
  \{ For 6 weeks \}
  ↓
  Stopped only after infection is resolved

• Treat for diabetes also

• Resolution of infection seen by→ Gallium / Indium Scan
  (not commonly done)
  ESR (easy and commonly used)
**Diffuse otitis externa**

- **A/H/A Tropical ear/Swimmer's ear/Telephonist ear**
- **Occurs in immunocompetent patients**

**Cause**
- Sweating
- Changes pH — Acidic to Alkaline
- Itching
- Abrasion

**Clinical Presentation**
- Diffuse edema of external auditory canal
- Erythema
- Discharge
- Severe pain (movement of jaw & pinna increases pain)

**Rx**
- Antibiotics against pseudomonas
- Local (eardrops) & Systemic antibiotics
- Ear toileting before eardrops
Localised otitis externa / furuncle

- Folliculitis
- Localized to outer 1/3 part of External auditory canal (Cartilagenous)
  - C/F
  - Rx
  - Pain - increases on jaw & pinna movement
  - Tragus sign positive (if tragus sign negative - referred pain)
  - Organism - Staphlococcus
  - Antibiotics against staphylococcus e.g. Amoxiclav

Otomycosis

- Cause
- C/F
- Rx
- Fungus (due to increased humidity in EAC)
  - Aspergillus niger
    - Wet newspaper appearance (when mixed with discharge)
  - Candida
    - Cotton wool appearance
    - Ear toileting
    - Local antifungal ear drops
    - Otitis externa - m/c Bacterial (Pseudomonas)
Herpes zoster oticus

- Varicella zoster infection of EAC
  - C/F
  - Rx
  - Ramsay Hunt syndrome
    - Vesicles in the distribution of nerve (greater auricular nerve & part of facial nerve)
    - Discharge
    - Acyclovir
    - Ear toileting
    - Local antibiotic ear drops – for secondary infection due to rupture of vesicles
    - Varicella zoster reactivation in geniculate ganglion
      - Herpes zoster + Facial N Palsy
      - Rx-Acyclovir immediately + steroids

- Discharge
Bullous myringitis

A/V/A myringitis bullosa

- **Cause**
  - Streptococcus Pyogenic
  - Influenza virus
  - Mycoplasma pneumonia

- **Examination**
  - Bullae deep in EAC
  - Tympanic membrane
  - Bloody ear discharge

- **Rx**
  - Ear toileting
  - Local antibiotic – if bullae ruptures resulting in secondary infections
  - Systemic antibiotics against Streptococcus pneumonia

---

Ear wax

**Ear wax**

- **C/F**
  - It is ceruminous secretions + desquamated epithelium

- **Blocked ear**
- **Pain** (when wax swells after swimming or shower)
Ear wax – Management & complications

wax solvents (loosen the wax)

↓

Syringing

↓

water at body temperature

↓

posterolateral direction

↓

at moderate pressure

Instruments for syringing

Complications

1. Vertigo (if water is not at body temperature)

2. Perforation of tympanic membrane (if water injected with high pressure)

Ear wax – contraindications of syringing

1. Tympanic membrane perforation

   - Wax or foreign body removed by instrumentation

   - Wax is removed by passing the probe behind it

   - Foreign body is held with forceps & removed
11) **Battery** (exposure to water - leakage of contents

\[ \downarrow \]

Causes necrosis of tissues

11i) **vegetative Foreign body** (swells up on exposure to water

\[ \downarrow \]

becomes impacted

Foreign body - insect removal

Foreign body - insect in EAC

\[ \downarrow \]

1st - Kill it by instilling oil drop

\[ \downarrow \]

Remove it by syringing
CONDITIONS OF TYMPANIC MEMBRANE

Traumatic perforation

- Due to sudden pressure variations, trauma.

- Management:
  → Keep ear dry
  → If infection or ear disease present: Local and systemic antibiotics given

- Tympanic membrane perforation heals by 3 months (normally)
  → Only 2 layers heal (fibrous layer will not heal)

- If it doesn't heal by 3 months
  ↓
  Permanent perforation
  ↓
  Myringoplasty

Tympanosclerosis

- Follows:
  → Chronic infections: CSOM, serous otitis media.
  → During healing of perforation

- Deposition of calcium in fibrous layer

- Management: conservative
Retraction of tympanic membrane: Stage I & Stage II

- Based on Sade classification of pars tensa retraction

Stage I retraction of tympanic membrane
- Not in contact with Incus
- Tympanic membrane:
  - Sickling of malleolar folds
  - Cone of Light: Absent or distorted
    - Dull Tympanic Membrane
      - Handle of malleus gets shortened
      - Lateral process appears prominent
      - Sickling of malleolar folds
Stage II Retraction of Tympanic membrane
• Tympanic membrane comes in contact with incus

Retraction of tympanic membrane: Stage III & Stage IV

Stage III Retraction of Tympanic membrane
• Atelectasis
• Tympanic membrane is in contact with promontory
• No air in the middle ear

Stage IV Retraction of Tympanic membrane
• Adhesive otitis media
• Tympanic membrane is adherent to promontory

Image of a retracted TM as seen in otoscopy
ACUTE OTITIS MEDIA

Etiology

- MC causative organism → Streptococcus pneumoniae / Pneumococcus
- Infection of pharynx affects the Eustachian tube:
  - Children
    - Eustachian tube is shorter & wider
    - Infection spreads very fast
  - Adults
    - On forceful blowing of nose in case of sinusitis, pharyngitis
  - Tubal blockage (edema)
  - Tympanic membrane is retracted
    - Hyperemia of middle ear mucosa [pre-suppurative]
    - Red congested tympanic membrane
    - Cart wheel appearance of tympanic membrane.

ENT • v2.0 • Marrow 4.0 • 2020
Exudation of fluid in middle ear

\[ \downarrow \]

Pus collects in middle ear cavity

\[ \downarrow \]

Tympanic membrane bulges out

\[ \downarrow \]

Tympanic membrane perforates [suppuration]

\[ \downarrow \]

Pulsatile Otorrhea

(Light house sign)

Clinical approach to Acute Otitis Media [AOM]

- Case of 3 year old child brought with severe pain in right ear & throat (suspect upper respiratory tract infection)

On Examination of the tympanic membrane (Tm)

\[ \downarrow \]

- Hyperemic
- Cart wheel appearance

\[ \rightarrow \]

Red, congested

Full bulging

\[ \downarrow \]

Diagnosis: AOM

Management:
- Antibiotics
- Analgesics
- Decongestants

\[ \rightarrow \]

Diagnosis: AOM

Management:
- Myringotomy
Myringotomy

- Type of incisions
  - Radial
  - Curvilinear
    - Not preferred (Incision closes fast
      → Fibers of Tm run radially)
    - Preferred

Management of ASOM

1. Medical
   - Antibiotics
   - Analgesics
   - Decongestants
2. Impending Rupture
   - Myringotomy

   - Curvilinear J-shaped incision in posteroinferior quadrant (most easily accessible part)
   - Never done in posteroinferior quadrant because of chance of injury to:
     * Chorda tympani
     * Facial nerve
     * Incudostapedial joint
     * Oval window

   → Bullous myringitis
   * Caused by Streptococcus pneumonia.
   * Bullous appearance to a localized area on tympanic membrane.
Acute Necrotizing Otitis Media (ANOM)

- Causative organism: β Hemolytic streptococcus
- Perforation: marginal

Types of perforation:

- Central
  - Thin rim of normal
  - Pars tensa present
  - Annulus normal
    - (not eroded)
  - Includes: small, medium, large, subtotal perforations

- Marginal
  - Annulus is eroded

- Small (1 quadrant)

- Medium (2 quadrants)

- Large (3 quadrants)

Annulus
  - (Thickened fibrous layer of TM)

Subtotal preparation
  - (Thin rim of pars tensa is only present)
SEROUS OTITIS MEDIA

Serous otitis media

Collection of sterile fluid in middle ear

Causes:
- decreased drainage
  - mc - Bilateral hypertrophy of adenoid (children)
  - eustachian tube dysfunction - in cleft palate
  - unilateral nasopharyngeal carcinoma (in adults)
- increased production
  - in allergic conditions

→ Adenoids starts atrophy by puberty and disappears at 20 yrs of age

Fluid collection in middle ear is not under any pressure - painless condition.
Tympanic membrane in SOM

1. Dull
2. Retracted tympanic membrane (not bulging)
3. Fluid level seen
4. Air bubbles present
5. Glue ear
   ↓
   Due to precipitation of fluid inside tympanic membrane

6. Bluish colour of Tympanic membrane
   Features of retracted tympanic membrane:
   - Prominent lateral process
   - Handle of malleus is shortened
   - Sickling of malleolar folds
   - Complete cone of light not seen

Audiological examination

1) Rinne’s test : Negative
2) Weber’s test : Lateralised to worst ear
3) ABC test : Normal (same as examiner)
4) Schwabach test : Lengthened
5) AB gap : Present
6) Tympanogram : B type of curve (best investigation)
Management of serous otitis media

Medical management:
1) Anti allergies
2) Decongestants
3) Steroid nasal spray
   ↓
   3 months of treatment
   ↓
   no response
   ↓
   Surgical management
   ↓
   Adenoidectomy + myringotomy
   with grommet insertion

→ To remove fluid from middle ear
   ↓
   Incision over tympanic membrane
   ↓
   myringotomy

→ Tympanic membrane
   \[\rightarrow\] \[\rightarrow\] “Grommet / ventilation tube”

Warning: Not all points are covered in the notes, especially conceptual explanations. Please use the notes in conjunction with Marrow Edition 4 videos.

→ Radial incision preferred
   ↓
   Tightly holds the grommet
   ↓
   In anteroinferior quadrant of tympanic membrane
   (because it replaces the eustachian tube)
Incision in posteroinferior quadrant not preferred (to avoid injury to important structures).

**Beer can technique**

- Incision at anterosuperior quadrant
  - Air enters the middle ear
  - Pushes fluid out through the lower opening
  - “beer can technique”

Done in cases of serous otitis media where it's difficult to remove fluid via radial anteroinferior incision.

---

**Short term grommet:**
- Stays for <6 months
- Gets extruded on its own
  - Once eustachian tube starts functioning
- Most commonly used
mid term grommet:
- Stays for 6-12 months

Long term grommet:
- "T" tube
- Stays for > 2 years
- In cases of:
  * Atelectasis
  * Adhesive otitis media

**Barotrauma of ear**

→ Pressure in external auditory canal, nasopharynx and inner ear

[Diagram: Automatically regulated according to external pressure]

→ middle ear pressure equal to external pressure

[Diagram: Air from nasopharynx enters into middle ear]

During ascent:

→ \( \downarrow \) pressure externally (negative pressure)

[Diagram: Diagram of ear anatomy, including EAC, bulge, Tm, and Eustachian tube]

\( \Theta \) Nasopharynx
→ If middle ear pressure exceeds +15 mm H₂O
  ↓
  Passive opening of eustachian tube
  ↓
  Pressure relieved

→ In case of eustachian tube block
  ↓
  ↑ pressure in middle ear
  ↓
  tympanic membrane pushed outwards (ossicles pulled laterally)
  ↑↑ pressure
  ↓
  Rupture of tympanic membrane

Barotrauma of ear during descent

Blood vessels of
Inner ear
Exudate
Ossicles
Retraction of EAC
Retraction of Tm
TM
Round window
Blood vessels of
Nasopharynx
Eustachian tube

→ ↑ pressure in middle ear
  ↓
  Pressure over round window
  ↓
  Bulges out into middle ear
  ↓
  Pressure relieved on opening of eustachian tube activity (swallowing, chewing, Valsalva maneuver)
If eustachian tube blocked

Pressure difference \( > 90 \text{ mm H}_2\text{O} \)

Pushes muscles of eustachian tube

Gets blocked

\( \uparrow \) Intracranial pressure

\( \downarrow \)

Rupture round window

\( \uparrow \) Pressure in blood vessels

Exudation of fluids into middle ear

\( \downarrow \)

\( \rightarrow \) Pain

\( \rightarrow \) Tympanic membrane rupture

Management: mostly preventive

\( \rightarrow \) Nasal decongestants

\( \rightarrow \) Do not sleep during descent
SAFE CSOM

Otitis media

→ Tympanic membrane perforation
  ↓
  Heals by 2 layers (maximum of 3 months)
  ↓
  • Outer epithelial layer grows—meets opposite side
  • Inner endothelial layer grows—meets opposite side

→ Outer epithelial layer joins inner endothelium
  ↓
  Perforation becomes permanent

→ Any perforation with h/o ear discharge < 1 month
  ↓
  Acute otitis media

→ Any perforation with h/o ear discharge < 3 months
  ↓
  Acute suppurative otitis media

→ Any perforation with h/o ear discharge > 3 months
  ↓
  Chronic otitis media / Chronic suppurative otitis media

Safe
  ↓
  Mucosal

Unsafe
  ↓
  Squamosal
Mucosal CSOM (Safe)

→ Permanent perforation of tympanic membrane.
  ↓
  On pars tensa.

→ Perforation can be central-small /medium/large or subtotal.

→ Clinical presentation:
  i) Hearing loss (10-40 db)
    a) Discharge
       - From infection of mucosal epithelium
       * middle ear infection through Eustachian tube from pharynx
         ↓
       ↓

       A.H.A. “Tubo tympanic CSOM”

→ Discharge from middle ear – mucoid/mucopurulent

→ Discharge from external auditory canal – Purulent discharge
  (no mucosal glands)
Treatment of otitis media

Medical management:
- Antibiotics:
  - Local ear drops
  - Oral Antibiotics
  \{ 6 weeks \}
- To reduce ear discharge (make ear dry)
- Surgery is contraindicated in acute infections

Surgical management: (Dry ear)
- Myringoplasty: Repair of perforation
- Ossiculoplasty: For ossicular necrosis
- Tympanoplasty: For safe CSOM

Ossicular necrosis

- Patients presents:
  - Discharge from ear
  - Perforation
  - Deafness (> 40 db hearing loss)

- Most common ossicle to get necrosed/eroded
  \downarrow
  Lenticular process of incus
  (Poor blood supply)
  \downarrow
  Followed by
  Long process of incus
  \downarrow
  Followed by
  Stapes supra structure

ENT • v2.0 • Marrow 4.0 • 2020
In TM perforation
- Sound directly goes into oval window
- Alternative pathway disrupted

Due to necrosis
- Ossicular pathway disrupted

Disruption of alternative and ossicular pathway
- Hearing loss (Sotic)

Rx of ossicular necrosis: repair perforation (myringoplasty) and reconstruct ossicles - (ossiculoplasty)

Grafts used:
1) Temporalis fascia (mo)
2) Perichondrium of ear cartilage
3) Fat

Tympanoplasty

- Myringoplasty + Ossiculoplasty
- Repair perforation of tympanic membrane
- Reconstruction of ossicles

Type 1:
- Graft over malleus
- Myringoplasty same as type 1 tympanoplasty
Type 2:
  Graft over incus

Type 3:
  Graft over stapes head
  ↓
  myringostapediopexy
  Aka columella tympanoplasty

Type 4:
  Maintain phase difference

→ Oval window kept open (for compression)
  ↓
  Round window closed (for rarefaction)
  ↓
  Sound reaches organ of corti

→ If both windows closed
  ↓
  Compression of both windows
  ↓
  Sound waves cancel each other

Type 5
→ Not done (vertigo)
→ Footplate completely fixed-otosclerosis
→ Fenestration operation
Myringoplasty

- Overlay technique
- Underlay technique (preferred)
  - Outer epithelial layer of Tm is raised
  - Graft placed under the malleus (over the annulus)
  - Outer epithelial layer replaced back

→ Subtotal perforation
→ Small margin of pars tensa seen
→ Rx Tympanoplasty (After dry ear is achieved)

Partial ossicular and total ossicular replacement prosthesis (PORP/TORP)

PORP:
→ Prosthesis over head of stapes
  ↓
  Graft placed over stapes (head)
→ To prevent narrowing of middle ear cavity

TORP:
→ Graft over foot plate of stapes
→ Stapes head should be absent
→ Both PORP, TORP Corresponds to Type 3 tympanoplasty
UNSAFE CSOM

Unsafe chronic suppurative otitis media

- If infection of middle ear is not confined
  - Spread to adjacent areas
  - Unsafe CSOM

  - Medially
    - Inner ear
    - Labrynthitis
  - Superiorly
    - Brain
  - Posteriorly
    - Mastoid
      - Intracranial complications
      - Mastoiditis
  - Further posterior
    - Sigmoid sinus
      - Sigmoid sinus Thrombosis

Cholesteatoma

- Bony boundaries in walls of middle ear
  - Prevent the spread of infection

- Walls must be eroded
  - For infection to spread
  - Done by cholesteatoma.

- Stratified squamous keratinizing epithelium
  - EAC
    - Keratin accumulation
      - Keratosis obturans
  - Any part of temporal bone except EAC
    - Cholesteatoma
      - Macrophage activated
        - Transform into osteoclast
        - Enzymes
        - Bone erosion
Cholesteatoma types

Congenital
- Behind intact tympanic membrane (TM)
- No history of perforation

Acquired
- Primary cholesteatoma
  - Infection from EAC into middle ear without TM perforation
- Secondary cholesteatoma
  - Infection from middle ear secondary to TM perforation

- Perforation can be following:
  - Traumatic cause
  - ASOM
  - Safe CSOM

Secondary cholesteatoma

- A° cholesteatoma occurs due to marginal perforation with endothelial area erosion
- marginal perforation of Tm
  ↓
  epithelial layer grows towards middle ear
  ↓
  Keratin flakes accumulate
  ↓
  macrophages
  ↓
  erosion of normal endothelium
  ↓
  epithelium grows further-medially
  ↓
  a° cholesteatoma.

Route:
- migration through marginal perforation (Acute necrotizing otitis media—ANOM)
  ↓
  “Haberman theory.”

Primary cholesteatoma

- Infection from EAC into middle ear without Tm perforation
- Eustachian tube obstruction for long period
  ↓
  Tm retraction
  ↓
  Fluid accumulates in middle ear (gradually thicken)
  ↓
  Retraction pocket
  ↓
  Keratin accumulates
  ↓
  Deepening of retraction pockets
  ↓
  Erosion of normal epithelium

- Bony erosion
  ↓
  Complication

- **m/c site of retraction of Tm**: Pars flaccida or Attic or prussak's space
- **m/c route**: via retraction pockets
  ↓
  "wittmaack theory"

### Theories of cholesteatomas

- **Wittmaack theory**
  - **m/c**
  - **m/c route**: retraction pocket in pars flaccida or prussak's space.

- **Sade theory**
  - Due to repeated infections from Eustachian tube
  ↓
  metaplasia

- **Ruedis theory** - Basal cell hyperplasia
  - repeated infections of Tm Basal epithelium grows inwards
Congenital and primary cholesteatoma

Congenital cholesteatoma:
- mc site: Anterior
  part of middle ear
- white (cholesteatoma) seen
  behind normal Tympanic membrane

Primary cholesteatoma:
- Epithelium from External Auditory canal
  ↓
  Via retraction pocket
  ↓ into
  middle ear-prussak space

- Otoscopic picture
- Retraction in pars flaccida.
- Cholesteatoma flakes seen

1° cholesteatoma.
Atticoantral CSOM and cholesteatoma

Atticoantral CSOM:

From pars flaccida retraction
pockets ® cholesteatoma.
  ↓ grows
  Into epitympanum
  ↓ through
  Attic into mastoid antrum.

1° cholesteatoma:

2° cholesteatoma.
Annulus is eroded
  ↓
  marginal perforation
  ↓
Epithelium migrates
  into middle ear
  ↓
Cholesteatoma in
  middle ear

2° cholesteatoma
Management of unsafe CSOM

- No medical line of management

  Surgeries of mastoid

  \[ \text{Intact canal wall procedure} \quad \text{canal wall down procedure} \]

  "Fascial recess approach"

Intact canal wall surgery - "Posterior Tympanotomy"

To remove disease part from middle ear & mastoid

Opening made in fascial recess area

‘Intact canal wall surgery’

Canal wall down surgery
- mastoid filled with cholesteatoma sac
  ↓
  Removed until antrum
  ↓
  Remove wall between mastoid and middle ear
  ↓
  middle ear and mastoid area converted into common cavity
  ↓
  mastoid

Canal wall down procedure classification

Radical mastoidectomy
↓
① Exenteration of mastoid air cells
↓
② Posterior canal lowered
↓
③ All mucosa and ossicles except foot plate of stapes removed (avoid fistula)
↓
④ Eustachian tube closed
Done only when there is Dead ear

modified Radical mastoidectomy
↓
① Exenteration mastoid air cells
↓
② Posterior canal lowered
↓
③ Only unhealthy tissues are removed and reconstruction done (Tympanoplasty)

- Eustachian tube closed to prevent further infections
Aim of surgery in unsafe CSOM

1) Safe ear and dry ear
2) Hearing ear (MRR surgery)

Procedure:
- 1st step: post auricular incision
  ↓
  “Wildes incision”
  ↓
  - Pinna retracted forward
  ↓
  - Drill mastoid at Macewen’s triangle area.
  ↓

Done by
- Cutting Burr → used to drill bones (mastoid)
- Diamond Burr → Blunt burr used to prevent injuries to important structures
- Mastoid antrum filled with cholesteatoma flakes
- Cholesteatoma flakes in pars flaccida (attic area)

- Remove cholesteatoma flakes
- Remove cholesteatoma flakes from attic area

- Mastoid and middle ear cavity clear from cholesteatoma flakes.

**Citelli’s angle and Trautman triangle**

*Citelli’s angle: (sinodural angle)*
- Angle between middle fossa, dural plate, and sigmoid sinus
Trautman triangle:
- Inner ear (anteriorly)
- Sigmoid sinus (posteriorly)
- Superior petrosal sinus (superiorly)

Removal of disease from middle ear

- Through facial recess
  Remove diseased part of middle ear
- Structures seen:
  - Oval window
  - Stapes
  - Round window

- Electrode passed into middle ear 1st
  \[\downarrow\]
  Via round window
  \[\downarrow\]
  Cochlea.

- passed through Facial recess approach

- Canal wall down procedure:
  - Remove complete wall between mastoid and middle ear
    \[\downarrow\]
    Remove diseased part
    \[\downarrow\]
    Reconstruct posterior wall of EAC (cartilage or bone)
COMPLICATIONS OF UNSAFE CSOM

Routes for complication:

1) Bony erosion (mc)
2) Natural openings (oval window, round window, cochlear aqueduct)
3) Congenital dehiscence
4) Traumatic / Surgical (iatrogenic) openings

Classification of complications of unsafe csom/asom and mastoiditis

- Intra temporal (extra cranial)
  - Mastoid antrum
    - Mastoiditis (mc)
      - Labyrinthitis
        - Fever
        - Vertigo
        - Sensorineural hearing loss
      - Facial nerve palsy
  - Inner ear
    - Facial nerve
    - Petrous part of temporal bone
      - Petrositis

- Intra cranial

ENT • v2.0 • Marrow 4.0 • 2020
Pharyngeal infections (MC: streptococcus pneumoniae)

Infection and edema of Eustachian tube → Blockage

Hyperemia of middle ear

Collection of fluid in middle ear → Suppuration

Spread of infection through aditus to mastoid air cells

Hyperemia of mastoid air cells (mucosal lining)

Suppuration

Pus filled mastoid air cells

Walls of mastoid cells undergo dehiscence
(Comes out through Aditus to middle ear, and then to EAC)

Coalescent mastoiditis

Pus filled mastoid cavity

Comes out through Aditus to middle ear, and then to EAC.

Reservoir sign and spread of pus from mastoid cavity

Reservoir sign:
- Rapid refilling of pus in EAC after it is removed
- Mastoid acts as reservoir of pus
Spread of pus from mastoid cavity

**Zygomatic abscess**

\[ \uparrow \]

Superiorly

\[ \uparrow \]

Postauricular abscess

\[ \leftarrow \text{Posteriorly} \rightarrow \]

Pus in mastoid cavity

\[ \rightarrow \text{Anteriorly (EAC)} \rightarrow \text{Luc's abscess} \]

\[ \downarrow \text{Inferiorly} \]

Sternocleidomastoid

\[ \downarrow \text{Bezold's abscess} \]

Post belly of digastric

\[ \downarrow \text{Citelli's abscess} \]

**Clinical features of mastoiditis**

- **MC organism**: *Streptococcus pneumoniae*

1. Pinna pushed - Outwards and laterally

2. Post auricular area:
   - Edema
   - Congested
   - Tenderness
   - Ironed out appearance

3. EAC: Reservoir sign
   - Sagging of posterior wall of EAC

Investigation: “HRCT temporal bone”
Management of mastoiditis

→ First line management
  ↓
  IV antibiotics
  ↓ No response for 48hrs
  Simple mastoidectomy /
  Cortical mastoidectomy /
  Schwartz operation

Petrositis

- In 30% population petrous part of temporal bone contains pneumatised air cells
- Infection spread from mastoid
  ↓ Petrous part of temporal bone
  mastoid infection
  ↓ Spread into petrosal air cells
  ↓ Hyperemia of petrosal air cells
  ↓ Suppuration
  ↓ Single pus filled petrosal cavity
→ Presents as persistence of ear discharge after mRM
  Petrous apex involvement
  ↓
  1. Involvement of 5th cranial nerve
     a. Palsy of 6th cranial nerve

Gradenigo's triad:
- Persistence of ear discharge after mastoid surgery
- Involvement of 5th, 6th cranial nerve

Clinical features:
* Retroorbital pain (5th cranial nerve)
* Diplopia (6th cranial nerve)

**Intracranial complications:**

Unsafe CSOM:

1. Extra dural abscess
   ↓
   Silent complications

a. Subdural abscess
3. meningitis
   ↓
   MC intracranial complication

4. Temporal lobe abscess
   ↓
   MC brain abscess

   Features:
   → Nominal aphasia
   → Supraquadranctic Homonymus Hemianopia
      (Pie in the sky appearance)
   → Convulsions

5. Cerebellar abscess
   → Ataxia (drunken gait)
   → Dysmetria.
   → Dysdiadochokinesia.
   → Nystagmus (direction changing)

6. meningitis (MC)
   → Kernig’s sign +
   → Brudzinski sign +
   → Neck rigidity +
   → ↑ ICT

7. Lateral sinus / Sigmoid sinus thrombosis

8. Otitic Hydrocephalus – Pseudotumour cerebri (Benign – IC HTN)

Sigmoid sinus thrombosis

![Diagram of Sigmoid sinus thrombosis]

ENT • v2.0 • Marrow 4.0 • 2020
unsafe CSOM
↓
Spread into sigmoid sinus
↓
Perisinus abscess
↓
Activation of co-agulation cascade
↓
Sigmoid sinus thrombosis
[lateral sinus thrombosis]
{Thrombus → Released into blood
↓
Fever ↑ which follows,
“picket fence fever or hectic fever”
↓
Thrombus
↓
mastoid emissary vein
↓
Non tender edema
of mastoid
(Griesinger’s sign)
↓
In mastoiditis, tender, ironed out
mastoid seen
↓
Internal jugular vein
↓
Other IJV taken up the function
↓
when normal IJV is compressed
↓
↑ ICT
(Otic Hydrocephalus)
↓
Seen as papilledema
↓
“Crowe-beck sign”
↓
Due to compression of Normal IJV
↓
Seen in lumbar puncture
↓
“Toby Ayer test”
↓
↑ in CSF pressure on lumbar puncture
or pressing normal IJV “Queckenstedt Test”
→ In CECT - MRI, thrombus does not take up contrast & walls appear empty: "empty triangle sign"
   "delta sign"
**OTOSCLEROSIS**

**Features of otosclerosis**

- **Site of origin:** Fissula Ante Fenestrum  
  (part of bony labyrinth; remains cartilaginous throughout life)
- **Hearing loss:** → Conductive  
  → Slowly progressive
- **Autosomal Dominant inheritance**
- **Female : male → 2 : 1**
- **MC in 3rd decade**
- **In pregnant women → aggravated by measles**
- **bilateral**
- **progressive conductive hearing loss**
- **Paradoxical Hearing that is better in noisy surrounding: Paracusis willisi**

**Examination of tympanic membrane**

- **Tympanic Membrane**
  - Pearly white (90%)  
  - Flamingo Pink (10%)
  - → Active cases  
  - → A/V/A Schwartz sign

**Tuning fork tests and pure tone audiometry**

- **Tuning Fork Tests:**
  1. Rinne → Negative
  2. Weber → Lateralised to worst Ear
  3. Absolute Bone conduction (ABC) → Normal
  4. Schwabach → Lengthened
  5. Gelles → Negative
Pure Tone Audiometry

Frequency in Hertz

Hearing loss in dB

→ AB Gap: Conductive Hearing Loss
→ Dip at 2000 Hz in Bone conduction curve: Carhart notch
→ Bone conduction:
  - Bony labyrinth vibrates → Membranous Labyrinth
  - Foot plate of stapes (derivative of Bony Labyrinth) vibrates → Additional vibrations
→ Fixation of Foot plate:
  - Absence of additional vibration → Dip at 2000 Hz
→ Air conduction:
  - Dip at all frequencies (vibration reach inner ear through ossicles alone)

Impedance audiometry

Tympanometry

Stapedial Reflex

→ Absent (fixed foot plate)

Active space

Compliance

Middle ear pressure

→ Rs - sclerosis type of curve
→ Best investigation

ENT v2.0 • Marrow 4.0 • 2020
Management of otosclerosis

- Flamingo Pink Appearance → Active

Medical management: Sodium Fluoride (NaF)

- Stops destruction
- ↓ Osteoclast
- ↓ proteolytic enzyme
- ↑ bone maturation
- ↑ osteoblast

Management of mature cases

Mature cases

- Whole stapes removed
- Teflon prosthesis used
- ↑ Risk of fistula
- ↓ Risk of fistula

- Teflon prosthesis used
- ↓ Risk of fistula
- ↑ Teflon prosthesis used
- Preferred
- Worst ear is operated first

No complication for 1 year
- Other ear operated
- Hearing aid
- Contraindication for surgery → Only hearing ear
Postoperative complications of otosclerosis

1. Conductive hearing loss
   - Fixation
   - Dislodged prosthesis
   - Necrosis of incus

2. Perilymph Fistula → Sensorineural Hearing Loss

Types of incisions for surgery

- Post auricular incision
  - A/H/A wilde's incision

- Lempert incision
  - Endaural approach
    - Incision along incisura terminalis

- Transcanal incision
  - A/H/A Rosen’s Incision

- Post surgery Finding:
  - Tympanometry → Ad curve
MENIERE’S DISEASE

Meniere’s disease-etiology

- N/A Endolymphatic hydrops - Idiopathic

Endolymphatic hydrops

↑ Production

↓ Absorption

Due to
Na⁺ / H₂O retention
(Rx - Salt restriction)

Dilation of membranous Labrynth

↓

1st at Apex of cochlea
(Scala media)

Dilation of saccule

Dilation of utricle
Dilation of scala media

↑ endolymph
↓
↑ dilation
↓
Rupture of Reissner's membrane

- Sudden gush of endolymph into perilymphatic compartment
  - Sudden change in pressure
    - Causes - vertigo 1st symptom
      - 20min-24 hrs vertigo subsides

- Mixture of perilymph and endolymph
  - Damages hair cells
    - Hearing loss - SNHL (sensorineural hearing loss)
      - Fluctuating hearing loss (after pressure equilises Reissner's membrane regenerates - Hearing loss subsides)
      - Low frequency affected 1st
        - Unilateral condition - Binaural diploacusis
          (one sound heard as two)

- Tinnitus
  - Ringing in the ear without external stimulus
  - Non-specific symptom
Dilation of utricle and saccule

Dilation of utricle & saccule

Dilation inside

Dilation outside

Macular membrane distortion

Drop crisis / Tumarkin crisis
(Patient suddenly falls without loss of consciousness)

Stapes touches utricle & saccule

Vertigo on loud sound – Tullio’s phenomenon

Meniere’s – Investigations

Rinne test Weber test ABC Schwabach

Positive SNHL Towards opposite ear Shortened Shortened

- Pure tone audiometry – No Air-bone gap
  Upsloping audiogram – Low frequency

- Recruitment – Present
  Tested by – SISI (short increment sensitivity score)
  - even 1 db increment can be differentiated

- Stapedial reflex – Threshold decreases (Present at low decibel of sound)
• Electrocochleography

\[
\frac{SP}{AP} > 0.45\%
\]

- \(SP\) - Summation potential of hair cells - ↑ due to irritation
- \(AP\) - Action potential of nerve

Glycerol test - Glycerol is hygroscopic

- ↓ endolymph
- ↓ vertigo

- Electrocochleography + Glycerol test = Diagnostic test

Meniere’s - Investigation: MRI

- MRI + gadolinium enhancement (contrast)

  Can be given transtympanically/IV

  If given IV → Normally

    Contrast stays only in
    Perilymphatic compartment

    → In meniere’s

      Due to mixture of perilymph and endolymph

      Contrast seen in both the compartments

Lermoyez’s syndrome

- Lermoyez’s syndrome - Hearing loss occurs before vertigo
Meniere's - Management

management

1st - Labyrinthine sedatives
- Promethazine
- Prochlorperazine
- Cinnarizine

Vasodilators
- Beta histine
  (histamine derivative)

Prevention
- Salt / Caffeine restricted diet

Meniere's - intractable vertigo

Intractable vertigo (not responding to medications)

If hearing is good

Selective vestibular destruction (to ↓ vertigo)

Gentamycin (chemical labyrinthectomy)

Vestibular neurectomy

- Given by microwick through round window
- Mechanism of action
  - Act on Type 1 & II hair cells of crista & macula.

- If no hearing left - Total Labyrinthectomy
Meniere's – management & Endolymphatic sac decompression

- **Endolymphatic sac**
  - Is present in between periosteal layer and meningeal layer of dura mater (intradurally) in posterior cranial fossa.

- **Endolymphatic sac decompression**
  - A small amount of bone is removed above the sac
    - So that sac can expand

- Approach → Wilde's incision given in Macewen triangle
  - Reach mastoid antrum
    - In mastoid antrum – visible parts
      - Descending part of facial nerve – in posterior wall of middle ear Some part of lateral semicircular canal (SCC)
        - On further drilling – reach posterior cranial fossa
          - In posterior cranial fossa.
            - lateral, posterior, superior semicircular canal are visible
              - Draw an imaginary line from Lateral SCC, bisecting
Called Donaldson’s line

Endolymphatic sac lies inferior to Donaldson’s line

- For approaching posterior cranial fossa
  
  Go through - Trautmann’s triangle (lies above Donaldson’s line)

Boundaries of Trautmann’s triangle

  Superiorly - Superior petrosal sinus
  Posteriorly - Sigmoid sinus
  Anteriorly - Inner ear
Trautmann's triangle,
donaldson's line
SUPERIOR SEMICIRCULAR CANAL DEHISCENCE

Superior semicircular canal dehiscence syndrome

* A/V/A Third window
* Bulge of superior semicircular canal present on petrous bone
  ↓
  Arcuate Eminence
* Mechanism of Hearing:

ENT • v2.0 • Marrow 4.0 • 2020
In case of opening in superior semi-circular canal (communicating cranium to inner ear)

Hearing loss: Conductive Hearing Loss

Sound vibrations move from oval window → Inner ear

Some sound is lost due to dehiscence

→ movement of foot plate of stapes

Conductive hearing loss

Bone conduction:

vibrations from skull go to inner ear due to dehiscence

Bone conducted sound is heard better

Autophony

Symptoms of superior semicircular canal dehiscence

1. Cochlear symptoms
   → conductive Hearing Loss: Air-born gap
   → Bone conduction sound heard very well
      (Foot steps, movement of Eyeball → Autophony)

2. Vestibular symptoms
   → Tullio's phenomenon: Vertigo on loud sound
   → Hennebert sign: External pressure changes leading to vertigo
Diagnosis of superior semicircular canal dehiscence

- Audiometry
  - PTA → Air-borne gap → Conductive hearing loss
  - Impedance
    - Tympanometry
    - Stapedial reflex
      - Normal

- HRCT

Note
vestibular neuritis
  - vestibular nerve inflammation
    - Only vertigo
    - No Hearing Loss
PRESBYCUSIS

- Age related hearing loss
- Gradually progressive SNHL

Types:
1. Basilar membrane stiffening → mechanical / Cochlear conductive
2. Hair cells affected → Sensory presbycusis
3. Stria vascularis → vascular / metabolic presbycusis
4. Nerves affected → Neural (mc)

Diagnosis and management of presbycusis

Audiogram (Pure tone Audiometry)

- B/L Symmetrical, Progressive sensorineural hearing loss

![Graph showing hearing loss pattern]

- Downsloping (high frequencies affected)

  Also in: Noise induced hearing loss
  Ototoxicity

Treatment:
- Hearing aids
- Cochlear implants
SUDDEN SENSORINEURAL HEARING LOSS

Sudden sensorineural hearing loss (SNHL)

Definition
- > 30 dB hearing loss in 3 consecutive frequencies within 3 days
- Rinne’s test - Positive (Air conduction > bone conduction)
- Weber’s test - Towards opposite ear
- Pure tone audiometry (PTA) - Show SNHL
- Common in young individuals

Causes
- Meniere’s disease
- Presbycusis
- Ototoxicity
- Noise trauma
- Idiopathic

Negative Rinne test in SNHL

- In Severe SNHL
  - > 70 dB hearing loss, in 3 consecutive frequencies, occurring suddenly
  - Rinne’s test - Negative (Bone conduction > air conduction)
    - False negative

Rinne test - negative

To differentiate

Conductive hearing loss
- Weber - towards same ear

Severe-SNHL
- Weber - towards opposite ear
SNHL - Management

- **Steroids** - Started immediately on high doses
  - If no response in 5 days
  - Transtympanic steroids
    (Introduced into inner ear through round window by microwick)
- **Antivirals**
- **Vasodilators**
- **Carbogen** - 5% CO₂ + 95% O₂
- **Hyperbaric O₂**
NOISE INDUCED HEARING LOSS

Noise induced hearing loss

- It was an occupational hazard, but nowadays common problem due to earphones usage
- Recommended noise level - < 85 db for 8 hrs/day for 5 days

Normal decibel levels of different sounds
- Whisper - 30 db
- Normal Conversation - 60 db
- Shout - 90 db

Decibels above normal range
100 - 130 → Causes discomfort in ear
130 - 150 → Pain in ear
> 140 db → Gun shot / Bomb explosion
   ↓
   Permanent hearing loss
   ↓
   Permanent threshold shift

- Temporary threshold shift - Becomes normal within 24 hours

Noise induced hearing loss – Investigations

- Pure tone audiometry
  - Bilateral
  - Sensorineural hearing loss
  - A dip in the graph at 4000 Hz - Acoustic dip
  - These changes - seen in - Permanent hearing loss (cannot revert the hearing loss)

- High frequency audiometry - Tests from 8000 Hz - 20,000 Hz
  ↓
  If no changes seen on PTA
- Oto Acoustic emission
  ↓
  outer hair cell function
  ↓
  Early diagnosis (can revert the hearing loss)

**Prevention**

Ear muffs - give protection of 40 db

Ear plugs - give protection of 30 db
# TUMORS OF EXTERNAL AND MIDDLE EAR

## Tumors of external auditory canal

- Bony outgrowths of External Auditory Canal (EAC)

<table>
<thead>
<tr>
<th>Outgrowths</th>
<th>Exostosis</th>
<th>Osteoma</th>
</tr>
</thead>
<tbody>
<tr>
<td>Location</td>
<td>multiple</td>
<td>Single</td>
</tr>
<tr>
<td></td>
<td>Deep EAC</td>
<td>Superficial, Lateral EAC</td>
</tr>
<tr>
<td></td>
<td>A/h/A surfer's ear</td>
<td></td>
</tr>
<tr>
<td></td>
<td>In surfer's - exostosis is present normally</td>
<td></td>
</tr>
<tr>
<td></td>
<td>To protect from cold exposure to tympanic membrane</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Which prevents vertigo</td>
<td></td>
</tr>
<tr>
<td>m.c benign tumor of EAC</td>
<td>Only if patient has hearing loss / wax impaction</td>
<td></td>
</tr>
</tbody>
</table>

**Exostosis**

**Osteoma**

---

Exostosis

![Exostosis](image1.png)

Osteoma

![Osteoma](image2.png)
Tumors of middle ear – Glomus tumor

Glomus tumor

↓

Arises from paraganglion cells

(neural crest cell origin – Present in relation with cranial nerves)

↓

Paraganglioma

↓

If arises from Tympanic plexus in the promontory area

↓

Glomus tympanicum

• MC benign tumor of middle ear

• Encapsulated and locally invasive

If arises from jugular bulb area–floor of middle ear in relation with 9, 10, 11 cranial nerves

↓

Glomus jugulare

Glomus tumor – property of hormone secretion

• Usually paragangliomas are hormone secreting

↓

Eg – Pheochromocytoma

↓

Releases catecholamines

↓

Causes hypertension

• The Glomus tumors are usually non-hormone secreting

↓

Non chromaffin cells

• Usually – Chromaffin cells

↓

The hormone secreted stain positive with chromium salts
• To differentiate between chromaffin cells and non-chromaffin cell
  ↓
  Check - urinary vanillylmandelic acid (VMA)

• 10% of glomus tumors are multicentric
  ↓
  So should check urine VMA for two reasons
  ↓
  i) If urine VMA is ↑ - paraganglion tumor is present elsewhere
  ↓
  ii) To prepare the patient before surgery (Treating hypertension)

Glomus tumor - Clinical presentation

  * Tumor arises from jugular bulb enters hypotympanum, mesotympanum
  * Conductive hearing loss (CHL)
    * Pulsatile tinnitus
      - Pulsations from internal carotid artery are transmitted
        ↓
        Perceived as tinnitus
        ↓
        Compression of ICA - relieves tinnitus
  * On examination - Rising sun appearance / Red reflex of tympanic membrane
Glomus tumor – diagnosis

* Some time tumor can protrude into EAC as Polyp
  ↓
  should not be avulsed
  ↓
  Because of relation with important structures (cranial nerves, vessels)

* Diagnostic sign – Brown’s sign / Pulsation sign
  ↓
  Elicited with Siegel’s speculum
  ↓
  Speculum is attached to otoscope
  ↓
  On compressing the bulb – Pressure ↑ses in EAC – tumor blanches
  ↓
  On releasing the bulb – Pressure ↓ses in EAC – tumor pulsates again

* Best investigation – CECT
  ↓
  Absence of crest of bone between Jugular bulb & internal carotid artery
  ↓
  Phelp’s sign
Glomus tumor – Fisch classification

A - middle ear
B - middle ear + mastoid
C - inner ear + infralabrynthine involving carotid canal
  C_1 - Limited vertical carotid canal involvement
  C_2 - Extensive vertical carotid canal involvement
  C_3 - Horizontal carotid canal involvement
D - Intracranial extension

Glomus tumor – management

* It is a benign tumor and locally invasive
  \[\text{\downarrow}\]
  Grows very slowly

* If patient is elderly, tumor is slowly growing, small tumor, asymptomatic
  \[\text{\downarrow}\]
  Wait and watch

* If symptomatic
  \[\text{\downarrow}\]
  * Gamma knife therapy – Only if size is < 3cm
    (Radio surgery – Retards the growth of tumor)
  * Surgery – if size > 3cm
ACOUSTIC NEUROMA

- MC type of CP angle
- R/H/A vestibular Schwannoma
- Locally invasive
- MC arises from Inferior Vestibular Nerve in Internal Acoustic meatus
- unencapsulated
- MC in elderly
- In young patients:
  - → NF - 2 : Bilateral Acoustic Neuroma
  - → 5% NF - 1 : Unilateral Acoustic Neuroma

Clinical features

- Internal Acoustic Meatus (IAM)
  - Facial nerve
  - Bill bar
  - Superior vestibular nerve
  - Transverse crest
  - Singular nerve
  - Inferior vestibular nerve
  - Cochlear nerve

- Cochlear nerve gets compressed → Unilateral SNHL + Tinnitus
- Superior vestibular nerve gets compressed → No Vertigo
- Hitzelberger Sign (Hypoesthesia of IAM posterosuperiorly)
  → Facial Nerve

ENT * v2.0 * Marrow 4.0 * 2020
Tumor in cerebellopontine angle

- Upper pole involvement (Tumor > 2.5 cm)
  - $5^{th}$ CN → Loss of Corneal Reflex
  - $6^{th}$ CN → Diplopia

- Lower pole involvement
  - $9^{th}$, $10^{th}$, $11^{th}$ CN affected

- Earliest nerve to be involved → $8^{th}$ CN >>> $5^{th}$ CN
- Earliest ocular sign → Loss of corneal reflex ($5^{th}$ CN)
- Presenting feature: Unilateral SNHL + Tinnitus

Tuning fork tests and audiometry

Tuning Fork Tests
1. Rinne → ☑
2. Weber → Lateralis to better ear
3. ABC → Shortened
4. Schwabach → Shortened

Audiometry
- PTA → No AB gap
  - SNHL
- Stapedial Reflex Decay: Present
- Best Audiometry test: BERA

1. ↑ Latency between waves I and V
2. Internal latency difference of wave V > 0.2 ms
Best investigation: Gadolinium enhanced MRI

MRI: Ice Cream on a Cone Appearance

Management and histopathology of acoustic neuroma

Management
1. Wait and Watch
2. Gamma Knife (< 3cm)
3. Surgical Excision

Histopathology
- Types of tumours

→ Compact
  → Antoni Type A
→ Loose arrangement
  → Cystic spaces
  → Antoni Type B
  ↓ Excision done usually
Auditory brainstem implant

* Placed in: Lateral Recess of 4th ventricle
* Indication: NF-2 patients operated for Bilateral Acoustic neuroma.
* Replaces nerve (stimulates cochlea directly)
FACIAL NERVE AND ITS DISORDERS - 1

Facial nerve

Intracranial Part
- From origin to internal acoustic meatus (IAM)

Intratemporal Part
- From internal acoustic meatus to stylomastoid foramen

Extracranial Part
- From stylomastoid foramen to structures supplied by it

→ Facial Nerve → mixed nerve (mainly motor)

Sensory component: Nerve of Wrisberg
Nerve of intermedius

Divisions of intratemporal facial nerve

1) Meatal segment (in internal acoustic meatus)

2) Labyrinthine segment (in inner ear)
   - Covered by narrowest part of fallopian canal
   - Susceptible to compression and ischemia and palsy

Enters into medial wall of middle ear

Takes turn 1st genu of facial nerve
3) Tympanic / Horizontal Segment

- Landmarks:
  a) Processus cochleare formis
  b) Oval window (below facial nerve)
  c) Lateral semi circular canal (above facial nerve)

**Mastoid segment**

4) Mastoid segment: (vertical segment)
- Runs in the posterior wall of middle ear

→ Disease of middle ear
  ↓
  Removed via facial recess area
    ↓
    Medial segment

Sinus tympani area should be examined (hidden area)

→ If disease removed via

  Facial recess:
     (lateral segment)
        ↓
        Posterior tympanotomy
          Or
          Intact canal wall surgery
          Or
      Combined approach tympanoplasty

ENT v2.0 • Marrow 4.0 • 2020
→ mc part to be injured in mastoid surgery

(mc part 2nd genu)

Branches of facial nerve

1) From the 1st genu — Greater Superficial Petrosal Nerve

    ↓

    Lacrimation Nasal Palatine Secretion Secretion

→ if damaged → Dry eye

"Schirmer's test"

a) In the middle ear — Nerve to stapedius

    ↓ injured

Absence of stapedial reflex — Hyperacusis

3) Above stylomastoid foramen

    ↓

Sensory branch of posterior auricular nerve

→ involved in Hitzelberger sign in Acoustic Neuroma.

4) Just above stylomastoid foramen — Chorda tympani

    ↓

Exits middle ear enters middle ear through anterior wall through posterior wall

→ Supplies Anterior ⅔rd of tongue — Taste

→ Salivation from Sublingual and Submandibular Salivary glands

Topodiagnostic test & prognostic tests

Topodiagnostic test:

1) Schirmer's test
2) Stapedial reflex
3) Taste (Electrogustometer)
4) Salivation

→ Topodiagnostic tests done to locate site of lesion
Prognostic tests

Electrophysiological test

Electroneuromography  
- Nerve injured  
  - Injury  
  - Wait for 2 days (wallerian degeneration)

Electromyography
- Fibrillation Potential  
- Action Potential  
- Flat Curve
- Bad Prognosis  
- Good Prognosis  
- Re-examination needed

a sides of facial nerves are compared to identify the degree of dysfunction in injured nerve
FACIAL NERVE AND ITS DISORDERS - 2

Bells palsy

- MC idiopathic facial nerve palsy
- 60% cases → Infection with HSV - 1
- Site: Labyrinthine segment (narrowest)
- Patients presents with:
  → Dry eye
  → Hyperacusis
  → Loss of taste and salivation in anterior 2/3rd of tongue
  → Motor palsy of that 1/2 of face

  Lower motor Neuron (LMN) palsy
  → Acute onset

- Management
  1. Antiviral → < 72 hrs/3 days
  2. Steroids → 1 mg/kg
  3. Physiotherapy with facial exercise and nerve stimulation
  4. Eye → Lubricants (dry eye)
     Close the eye with pad

Facial nerve Palsy

- Recurrence is present : < 10%
Melkersson–Rosenthal syndrome

- Idiopathic
- Recurrent
- Swelling of lips + Fissuring of tongue

Infections causing facial nerve palsy

- Viral
  - Ramsay Hunt Syndrome
- Bacterial
  - malignant otitis externa
  - ASOM
  - Unsafe CSOM
  - Dehiscence
  - m.C site
  - Horizontal part of facial nerve above oval window

ENT v2.0 • Marrow 4.0 • 2020
Injury causing facial nerve palsy

- Surgical (iatrogenic)
  - m/c: Parotid
  - Followed by mastoid
- Fracture of temporal bone
  - Longitudinal fracture (↑ common)
    - Conductive hearing loss
    - CSF Otorrhea
    - Facial nerve palsy (Not common)
    - No vertigo
  - Transverse fracture
    - SNHL
    - Vertigo
    - Facial nerve palsy (very common)
    - CSF otorhinorrhea

- Best investigation: HRCT

Management of complications (post - surgery)

- Sudden onset palsy
  - Immediate exploration

- Late onset palsy
  - Steroids

- m/c graft: Greater Auricular
- Longer graft: Sural

Complications following nerve regeneration

- A nerve gets cut
  - It regenerates and connects with other nerves
    (leads to complication)
• Complications:
  1. Synkinesis
  2. Crocodile Tears: Tearing with salivation
     → Injury is before origin of greater superficial petrosal nerve in labyrinthine segment.
     → Management: Botulinum toxin in lacrimal gland

Frey’s syndrome

• Gustatory Sweating
• Injury to Auriculotemporal nerve in parotid surgery
  ↓
  Aberrant regeneration
  ↓
  Supplies sweat glands overlying parotid

• Not due to regeneration of Facial Nerve

• Management:
  → BOTOX
  → Implantation / Sternocleidomastoid below skin
Tests to find location of nerve injury:

- **Topo - diagnostic Tests**
  - Schirmer's Test
  - Stapedial Reflex
  - Taste and Salivation
COCHLEAR & BRAINSTEM IMPLANTS, BAHA

Cochlear implant:

→ Invented by William F. House
   ↓
   Father of Neuro otology

→ Replace organ of corti
→ Stimulates cochlear nerve
→ Indications
   1) Bilateral severe hearing loss > 70db
      a) No benefit from hearing aid trial of atleast 3 months

→ Children who are born deaf – Prelingual deafness

Prelingual & postlingual deafness

→ For prelingual deafness
   ↓
   Hearing aid trial of → No benefit
   ↓
   6 months
   Cochlear implantation
   ↓
   - Done best at 1 yr
   - Speech center maximally developed at 3 yrs

→ Postlingual deafness:
   - Trauma (noise)
   - Presbycusis
   - Ototoxicity
   - Labrynthitis
   } * Done as early as possible
   } * No age limit

3) Prelingual deafness – Rx with in 1 yr
4) • Cochlear nerve has to be functional
    • Central auditory pathway has to be functional
5) No evidence of absence of cochlea
6) Mentally and physically stable children
Components of cochlear implant

External component

- Microphone - Picks up sound
- Speech processor
  ↓
  Converts sound → Digital signals
- Transmitter coil

Internal component:
- Receiver stimulator
  ↓
  Receives signal from transmitter coil
  ↓
  Transmits signal to electrodes

Transmitter

Microphone

Receiver

Eardrum

Electrode (in cochlea)
- Electrodes
  ↓
  mastoid drilled to
  insert electrodes
  ↓
  Pass through Facial recess into middle ear
  ↓
  Via roundwindow
  ↓
  Into inner ear
  (Scala tympani)
  ↓
  Replace organ of corti
  (stimulate cochlear nerve)

Auditory brain stem implant (ABI)
Bone anchored hearing aid [BAHA]

Indications:

1) Deformity of pinna and EAC
2) Large mastoid cavities following modified radical mastoidectomy
3) Chronically discharging ear
   → Replaces EAC, pinna, middle ear
   → Directly stimulate inner ear
   ↓
   Bone conduction threshold < 45db
4) Single sided deafness

Components of BAHA

- Titanium screw - send the vibrations to cochlea through skull
  ↓
  (bone conduction)
  Integrated with bone
  ↓
  "Osseointegration" takes place
- Abutment: Fits into titanium screw
  ↓
  Holds speech processor in its place & passes
- Speech processor - captures sound via microphone
  ↓
  BAHA transducer passes the vibrations to implant via abutment

→ Age for BAHA according to USFDA - 5yrs
→ Can be put as early as 3yrs
→ Skull thickness > 3mm
HRCT TEMPORAL BONE

Indications of HRCT

1) Complications of unsafe CSOM
2) Fracture of temporal bone
   a) Tumours (glomus)

mastoid - Superficial - squamous part
   Deep - petrous part

Suture in between: Petrosquamous suture/ Korner's septum

→ Petrosquamosal suture (rare)
   ↓ if present
   Confused with petrosal part during surgery
Structure seen:

- mastoid air cells
- EAC
- middle ear (connected to mastoid via additus)

"Ice Cream Cone" appearance (roof of middle ear on transverse section)

- Head of malleus
- Short process of incus
  Runs posteriorly
Fractures passing through mastoid

- Hazy mastoid air cells
- Fracture line seen

Longitudinal Fracture passing through mastoid via middle ear

- Longitudinal fractures run - parallel to petrous part
- Transverse (vertical) fractures run - perpendicular to petrous part

- Clinical features of longitudinal fracture:
  - Conductive hearing loss
  - Perforation of tympanic membrane
  - CSF Otorrhea

Transverse/ Vertical fractures:
\[ \rightarrow \text{No fracture of skull} \]
\[ \rightarrow \text{Fracture line passing through internal acoustic meatus} \]
\[ \rightarrow \text{Clinical features:} \]
\[ \rightarrow \text{Sensorineural hearing loss (Cochlear nerve)} \]
\[ \rightarrow \text{Facial nerve palsy} \]
\[ \rightarrow \text{Vertigo (Vestibular Nerve)} \]

\[ \rightarrow \text{Hazy mastoid air cells} \]
\[ \downarrow \]
\[ \text{Fluid / Pus accumulation} \]
\[ \downarrow \]
\[ \text{Mastoiditis / Unsafe CSOM (Cholesteatoma)} \]

\[ \rightarrow \text{Post operative} \]
\[ \rightarrow \text{(Canal wall down surgery)} \]
\[ \downarrow \]
\[ \text{Middle ear and mastoid are converted into common cavity} \]
CHOANAL ATRESIA & ANATOMY OF NOSE

Embryology

- Nasal pits grow medially → Nasal cavity
  Choana (posterior nares)
  Nasopharynx

- Stomatodeum → Oral cavity
  Oropharynx

- Buccal membrane: Separates nasopharynx & oropharynx
  → Disappears at birth
  If buccosal membrane is persistent "Choanal Atresia"

Choanal atresia

- Persistence of buccosal membrane
  → 70% mixed
  → 30% bony

  - Child born with respiratory distress
    Nasogastric tube cannot be passed

  - Respiratory distress is because infants are obligate nasal breathers

  - Immediate management:
    * Mc Govern's technique: wide - bored nipple is placed in the mouth to keep it open

  - Late management:
    Surgical excision
    +
    Mitomycin C application (to prevent synechiae)

- Associated with CHARGE syndrome
  C → Coloboma of eye
  H → Heart defects
  R → Choanal Atresia
  G → Retarded growth & development
  E → Ear abnormalities
• Child will be normal while crying
  Develops cyanosis & breathlessness when quiet

Anatomy of nose

• External nose = Bony + Cartilaginous
  upper 3/4th  lower 1/4th

• Bony ⇒ Nasal bones
  Frontal process of maxilla.

  Cartilaginous ⇒ upper lateral
  { Paired
  Lower lateral (alar)
  Sesamoid (lesser alar)
  Septal cartilage
  → unpaired

• Rhinoplasty – Surgical correction of defects in external
  framework of nose

• Important junctions:
  1) Frontonasal suture nasion
  2) Between bone & Cartilage rhinion
  3) Between upper & Lower lateral cartilage limen nasi

• Limen nasi – External dip in nose corresponds to narrow part of
  nasal cavity
Nasal valve area

- Narrowest part of nasal cavity
- Boundaries:
  Laterally: limen nasi
  Upper border of lower lateral cartilage
  Lower border of upper lateral cartilage
  Inferior turbinate
  Medially: septum

- Offers maximum resistance to airflow during expiration
  - Reverse/eddy currents are formed
    - Ventilates anterior sinuses
    - Frontal
      - Anterior ethmoid
      - Maxillary

Interior of nose

- Divided by septum into 2 nostrils
- Lined by respiratory epithelium
  - Ciliated columnar pseudostratified epithelium
- Lower part of nostril is lined by skin - vestibule
  - Stratified squamous epithelium
- Upper most part of nostril ie, lateral wall + septum + roof of nose
  - Olfactory epithelium - responsible for smell
• when breathing normally ~90% air passes through middle part of the nose - 10-15% reaches olfactory epithelium
• when sniffing, more air reaches olfactory epithelium and improves perception of smell

Lining epithelium

- Upper 1/3rd
  - Olfactory epithelium
    - Ciliated columnar
  - Respiratory epithelium
    - Pseud stratified ciliated columnar epithelium

- Main lining epithelium

- Vestibule
  - Skin
    - Stratified squamous MC site for furuncles (A/V/A nasal vestibulitis)
ANATOMY OF LATERAL WALL OF NOSE 1

Concha bullosa v/s hypertrophied turbinate

Concha bullosa
→ Pneumatised turbinate
→ MC in middle turbinate
→ Confused with polyp
→ Sinusitis

Hypertrophied turbinate
→ External Hypertrophy
→ MC in inferior turbinate
→ Mulberry Appearance
→ Hypertrophic rhinitis

Management: Partial turbinectomy
endoscopic view

→ Management: Partial turbinectomy

**Ethmoid bone**

1. **Lamina papyracea**
   → Thin papery part of Ethmoid bone
   → Separates orbit from Nasal Cavity
   → Forms medial wall of orbit

2. **Cribriform plate of ethmoid bone**
   → Forms roof of nasal cavity

3. **Perpendicular plate of ethmoid bone**

4. **Superior turbinate, medial turbinate**

5. **Uncinate process**

   • Paranosal sinuses develop as an outpouching from lateral wall of nose into maxilla, ethmoid, Frontal and Sphenoid to form respective sinuses
- Function of paranasal sinuses:
  → Lightening of skull bone
  → Resonating chambers for voice

- Ethmoidal sinusitis → m/c orbital complications

**Eustachian tube**

- The eustachian tube opening is 1.25 cm on behind posterior margin of inferior turbinate.
- From middle ear when it drains into nasopharynx, its at an angle of 45° with horizontal.
- The opening of ET to nasopharynx, is surrounded by fat called as ostmann's pad of fat (helps in proper closure of ET)

**Sphenopalatine fossa**
• **Boundaries:**
  - Anterior → Posterior wall of maxillary sinus
  - Posterior → Pterygoid plate
  - Medial → Lateral wall of nasopharynx
  - Lateral → Infratemporal fossa through pterygomaxillary fissure

• **N/K/A pterygopalatine fossa**

• **Caldwell Luc Approach**
  - → To maxillary sinus
    - (done earlier → To remove polyps)
  - → Nowadays done by endoscopic sinus surgery
  - → For sphenopalatine fossa

**Lateral wall of nose:**

<table>
<thead>
<tr>
<th>Turbinates</th>
<th>Meatus</th>
<th>Openings</th>
</tr>
</thead>
<tbody>
<tr>
<td>→ Inferior turbinate (largest)</td>
<td>→ Inferior meatus (Largest)</td>
<td>→ Nasolacrimal Duct (NLD)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Direction: DBL</td>
</tr>
<tr>
<td></td>
<td></td>
<td>→ Downwards</td>
</tr>
<tr>
<td></td>
<td></td>
<td>→ Backwards</td>
</tr>
<tr>
<td></td>
<td></td>
<td>→ Lateral</td>
</tr>
<tr>
<td></td>
<td></td>
<td>→ Opens into junction of anterior ⅔rd and posterior ⅓rd of inferior turbinate</td>
</tr>
<tr>
<td></td>
<td></td>
<td>→ NLD is bounded by valve of Hasner</td>
</tr>
</tbody>
</table>

• **Blockade of NLD is managed by**
  1. Massaging
  2. Endoscopic Dacrocystorhinostomy
ANATOMY OF LATERAL WALL OF NOSE – 2

Middle meatus

![Diagram of the middle meatus with labeled structures: ethmoidal sinuses, frontal sinus, superior turbinate bone, middle turbinate bone, inferior turbinate bone, maxillary sinus, orifice of maxillary sinus.]

![Diagram showing a cross-sectional view of the middle meatus with labels: middle turbinate, hiatus semilunaris, bulb ethmoidalis, septum, uncinate process, infundibulum, inferior turbinate.]
Lateral wall of nose:

<table>
<thead>
<tr>
<th>Turbinates</th>
<th>Meatus</th>
<th>Openings</th>
</tr>
</thead>
<tbody>
<tr>
<td>midae Turbinate</td>
<td>middle meatus</td>
<td>Osteomeatal complex</td>
</tr>
<tr>
<td>post ½</td>
<td>(most congested)</td>
<td>→ Anterior Ethmoid</td>
</tr>
<tr>
<td></td>
<td></td>
<td>→ Frontal sinus (Frontal Recess)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>→ maxillary sinus (opens in infundibulum)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>infundibulum (between uncinate and lateral wall)</td>
</tr>
</tbody>
</table>

- maxillary sinus (mC → sinusitis)
- mC sinusitis in children → Ethmoid sinus

**Functional endoscopic sinus surgery**

- Rigid Endoscope is used
- 1st step → uncineotomy
- Complication: Adhesion / Synechiae Formation (prevented by mitomycin c pack following surgery)
→ Bilateral osteomeatal complex block
→ Infection and secretion in maxillary sinus

Endoscopic Images:

Pus from middle meatus in a patient of sinusitis

Anterior ethmoidal cells

Anterior Ethmoidal Cells

- Bulla Ethmoidalis → most prominent
- Haller cells → extends to floor of orbit
- Roof of maxillary sinus
- Agger Nasi → anterior most anterior ethmoidal cell

ENT • v2.0 • Marrow 4.0 • 2020
**Superior turbinate and posterior ethmoidal cells**

**Lateral wall of Nose:**

<table>
<thead>
<tr>
<th>Turbnate</th>
<th>Meatus</th>
<th>Openings</th>
</tr>
</thead>
<tbody>
<tr>
<td>→ Superior turbinate</td>
<td>→ Superior meatus</td>
<td>→ Posterior ethmoids onodi cell</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Posterior ethmoidal cell</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- In lateral wall of sphenoid sinus</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- While operating, chance of injury to:</td>
</tr>
<tr>
<td></td>
<td></td>
<td>* ICA</td>
</tr>
<tr>
<td></td>
<td></td>
<td>* Optic nerve</td>
</tr>
</tbody>
</table>

- Sphenoid sinus → spheneno ethmoidal recess
ANATOMY OF NASAL SEPTUM AND ITS DISEASES

Anatomy of nasal septum

- Nasal septum forms medial wall of nostril
- Osteo cartilagenous structure (mainly - bony)
- Divided into:
  1. Columellar Septum
  2. Membrane Septum
  3. Septum Proper

Diagram of nasal septum

- Frontal bone
- Frontal sinus
- Nasal bone
- Septal cartilage
- Sphenoid sinus
- Nasal septum:
  - Vertical plate of ethmoid bone
  - Vomer bone
- Sphenoid bone
- Horizontal plate of palatine bone
- Sagittal section
- Nasal Septum:
  - Bony
  - Cartilagenous
  - Perpendicular Plate of Ethmoid
  - Vomer
  - Rostrum of Sphenoid
  - Nasal Spine of Frontal bone
Deviated nasal septum

- MC Cause: Birth trauma → 60%
- Clinical Features:
  → Nasal Obstruction
  → Recurrent Sinusitis
  → Acute Otitis Media
  → Headache
  → Hyposmia
  → Epistaxis
  → Cosmetic deformity

Endoscopic image - deviation

- Surgery is not done till 17 years of age (Development of Secondary ossifying centers of face complete by 17 years)

- Historically, SMR (Submucosal Resection of Septum) was done to correct Deviated Nasal Septum
  ↓
  Led to: SADDLE NOSE DEFORMITY
  ↓
  Corrected by AUGMENTATION RHINOPLASTY
  (TRAGAL CARTILAGE IS USED)
* Septoplasty is preferred now days

![CT Image]

→ Deviated Nasal Septum
→ Nasal Obstruction Right Side (with respect to patient)
→ Inferior turbinate Hypertrophy on left side

* Diagnosis
  → Spatula Test
  → Best: Nasal Endoscopy

* Cottle’s Test
  → Test for patency of nasal valve area

Submucosal resection of septum v/s septoplasty

<table>
<thead>
<tr>
<th>SMR (Submucosal Resection of Septum)</th>
<th>Septoplasty</th>
</tr>
</thead>
<tbody>
<tr>
<td>↓ Killian’s Incision</td>
<td>→ management for Deviated Nasal Septum</td>
</tr>
<tr>
<td>↓ Not done</td>
<td>↓ Freer’s Incision</td>
</tr>
<tr>
<td></td>
<td>↓ Done mostly after 17 years of age</td>
</tr>
</tbody>
</table>
Septal hematoma

- Cause: Trauma
  - Septal cartilage
    - Bleeding (Bilateral)
    - Collects between septal cartilage and perichondrium
    - Septal Necrosis
    - Saddle Nose Deformity

- On examination: Bilateral fluctuant swelling
- Management: Immediate Incision and Drainage

Septal perforation

- M.C Cause: Trauma
- Other causes: Cocaine abuse, Rhinolith (children)
- If no history of trauma
  - Look for granulomatous conditions of nose

1. Syphilis  
2. Wegener's  
  - (Granulomatosis Polyangitis)  
  - Can lead to bony perforation

- Clinical Features:
  → Whistling sound
  → Crusting

- Management
  → Small perforation → Flap
  → Large perforation → Sialotic button
ARTERIAL SUPPLY OF NOSE AND EPISTAXIS

Arterial supply of nose

- Internal carotid artery
- External carotid artery
- Ophthalmic branch
- Optic canal
- Anterior ethmoidal artery
- Posterior ethmoidal artery
- Pierce lamina papyracea
- Enters and supplies the nasal cavity
- Incision at lamina papyracea
- To reach ethmoidal artery
- Curvilinear incision: "Lynch Howarth incision"
External carotid artery

Maxillary artery

Facial artery (Superior labial artery)

Sphenopalatine Artery

Greater Palatine artery

Sphenopalatine

Foramen

Supplies Nasal Cavity

Enter oral Cavity via Palate

Comes up again and supplies Nasal cavity

Kiessellbach's Plexus

Anterior ethmoidal artery

Posterior ethmoidal artery

Sphenopalatine artery

Superior labial artery

Greater palatine artery

Little areas:
- Anterior ethmoidal artery + Sphenopalatine artery + Greater palatine artery + Superior labial branch of facial artery
- "Kiessellbach's Plexus" (MC site of epistaxis)
→ major artery of Kiesselbach’s Plexus
  ↓
  Artery of epistaxis
  ↓
  Sphenopalatine artery
→ mc site of epistaxis in children
  ↓
  Little’s area
→ Injury to anterior ethmoidal artery during FESS
  ↓
  Orbital hematoma.

Epistaxis

Causes:
1. mc over all - Idiopathic
2. mc in children - Trauma (nose picking)
3. mc in young adults - Trauma (nose picking)
4. mc in recurrent epistaxis in young male
  ↓
  Angiofibroma (malignancy)
5. Drugs: Anticoagulant, Antiplatelets
6. Bleeding disorders:
   Hemophilia
   Thrombocytopenia
   Von willebrand disease
8. mc in elderly : Hypertension
9. Infection - Granulomatous conditions of nose

Common site of epistaxis

(mc) arterial
  ↓
  Kiesselbach’s plexus (mc)
  ↓
  Retrocolumellar vein
→ Woodruff’s plexus (venous)
  ↓
Does not cause epistaxis

→ MCC for Anterior & Posterior epistaxis
  ↓
Sphenopalatine artery

→ Based on maxillary sinus opening / Nasal aperture
  ↓
Anterior epistaxis  Posterior epistaxis

Management of epistaxis

1) Trotter’s / Hippocratic method: (Pinch nose and lean forward)
  ↓
2) Local cautery
  ↓
3) Anterior nasal packing (both nostrils)
    → Absorbable pads used for
    ↓
    Anterior nasal packing
    ↓
4) Posterior nasal packing
  ↓
5) Ligation of sphenopalatine artery (Maxillary artery branch)
    TESPAL (Not used)
  ↓
6) Ligation of anterior ethmoidal artery
    → TESPAL (Transnasal Endoscopic Sphenopalatine Artery Ligation)
    - Not used now-a-days
NERVE SUPPLY OF NOSE, ALLERGIC AND VASOMOTOR RHINITIS

Nerve supply to nose - Olfactory nerve

- Olfactory nerve
  - Upper 1/3rd of nose

- Sensory supply
  - Ophthalmic nerve (dorsum & tip of nose)
  - Maxillary nerve

- Parasympathetic supply
  - Nasal secretions ( vidian nerve)

Olfaction:
- Olfactory mucosa: Only in upper 1/3rd of nose
  - a) Hyposmia: ↓ olfaction due to ↓ air conduction
  - b) Anosmia: Absent sense of smell,
    - Destruction of olfactory mucosa/nerve

Kallmann Syndrome:
- Congenital anosmia with hypogonadotropic hypogonadism

- c) Dysosmia / Parosmia / Cacosmia:
  - Altered or perverted sense of smell
- d) Presbyosmia: ↓ olfaction due to aging

Sensory supply of nose – Ophthalmic nerve

- Trigeminal nerve
  - Ophthalmic, maxillary, mandibular

Angle of mandible: Supplied by
- Greater Auricular nerve
Ophthalmic nerve - course

Trigeminal / Gasserian ganglion

↓

Ophthalmic nerve

↓

Enters superior orbital fissure

↓

3 branches

Nasociliary nerve, Lacrimal nerve, Frontal nerve

↓

Supplies dorsum and tip of nose

Sensory supply – Maxillary nerve

Contents of Sphenopalatine fossa:

- maxillary artery
- maxillary nerve
- Sphenopalatine ganglion (Largest parasympathetic ganglion)

Warning: Not all points are covered in the notes, especially conceptual explanations. Please use the notes in conjunction with Marrow Edition 4 videos.
maxillary nerve -
course

\[
\text{Trigeminal ganglion} \\
\downarrow \\
\text{maxillary nerve} \\
\downarrow \\
\text{Exits cranial fossa via Foramen Rotundum} \\
\downarrow \\
\text{Directly enters Sphenopalatine fossa} \\
\downarrow \\
\text{Enters sphenopalatine foramen} \\
\text{(lies 1cm behind middle turbinate)} \\
\downarrow \\
\text{Gives sensory branches} \\
\]

\[
\begin{align*}
\text{Infra- orbital nerve} & \quad \text{Nose and} \\
\text{terminal branch of maxillary) } & \quad \text{sinus} \\
\text{Lies in infra-orbital groove} & \quad \text{Palate and} \\
\text{(above maxillary sinus) } & \quad \text{Teeth (alveolar} \\
\downarrow & \quad \text{branch)} \\
\text{Exits via infra-orbital foramen} & \quad \text{Supplies skin of cheek and} \\
\downarrow & \quad \text{ala of nose} \\
\end{align*}
\]

- Fracture of orbital floor / Zygomatic bone / Maxillary bone

\[
\downarrow \\
\text{Infra-orbital nerve injury} \\
\downarrow \\
\text{Anesthesia of cheek}
\]
Parasympathetic / vasomotor supply of nose

- by vidian nerve (Greater petrosal + Deep petrosal)

Course of vidian nerve:

- Facial nerve (in middle ear cavity)
  ↓

- Greater petrosal nerve (given off at first genu)
  ↓

- Enters cranial fossa via. roof of middle ear
  ↓

- Joins Deep petrosal nerve (from plexus around internal carotid artery)
  ↓

- Forms vidian nerve (nerve of pterygoid canal)
  ↓

- Exits cranial cavity via. pterygoid canal
  ↓

- Enters sphenopalatine fossa
  ↓

- Sphenopalatine Ganglion
  ↓

- Lacrimation, Nasal secretions, Palatine secretions

Vasomotor rhinitis and allergic rhinitis

Vasomotor Rhinitis

↑ Parasympathetic activity
↓

vasodilatation
↓

↑ nasal secretions
↓

Vasomotor Rhinitis
(Paroxysmal sneeze, nasal secretions, post-nasal secretion)
**Allergic Rhinitis: Similar presentation**

<table>
<thead>
<tr>
<th>Allergic Rhinitis</th>
<th>Vasomotor Rhinitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>History of allergy ✧</td>
<td>✧</td>
</tr>
<tr>
<td>IgE ↑↑ (Type I hypersensitivity)</td>
<td>✧</td>
</tr>
<tr>
<td>Eosinophils ↑ (Absolute Eosinophil Count ↑)</td>
<td>✧</td>
</tr>
<tr>
<td>Scratch / Patch / Prick Test ✧</td>
<td>✧</td>
</tr>
<tr>
<td>On examination, Pale, edematous, boggy nasal mucosa</td>
<td>Congested mucosa</td>
</tr>
</tbody>
</table>

**Management of allergic and vasomotor rhinitis**

**Allergic Rhinitis:**
- Avoid allergens
- Anti-allergic medication - Cetrizine
  - Levocetrizine
  - Fexofenadine
  - Leukotriene inhibitors
  - Mast cell stabilizers

- Steroids (nasal spray)
- Nasal decongestants

**Vasomotor Rhinitis:**
- Primary management: Medical (similar to allergic rhinitis).
  - Steroid nasal sprays
  - Nasal decongestants
  - No role of anti-allergic medication

- Vidian neurectomy (in failure of medical management)

**Rhinitis medicamentosa**

- Due to over-use of nasal decongestants
  - Oxymetazoline, xylometazoline
- Leads to rebound congestion
  ↓
  Nasal obstruction
  ↓
  Ischemia
  ↓
  Necrosis

- Nasal decongestants: Not to be used for >7 days

Management:
- Withdrawal of decongestants
  +
  Local steroid therapy

If required: Systemic steroids may be given
ANATOMY OF PARANASAL SINUSES, SINUSITIS AND ITS COMPlications

Maxillary sinus

- Largest sinus
- A/K/A Antrum of Highmore

Ethmoid sinus

- Present at birth
- More pneumatized

\[ \text{m.C infected in infants and children} \]

\[ \text{Ethmoid air cells} \]

\[ \text{Anterior group} \quad \text{Posterior group} \]

\[ \text{Opens to} \quad \text{Opens to} \]

\[ \text{middle meatus} \quad \text{Superior meatus} \]
• Pain ↑ with eye movement
• Orbital complications result most commonly following ethmoid sinusitis

**Sphenoid sinus**

• Present at birth
• Opens into sphenoid ethmoidal recess
• Least common sinusitis
• Type of sphenoid sinus pneumatization

![Conchal Pre-sellar Sellar (m.C)]

**Frontal sinus**

• Not present at birth
• Last to complete its development
• Last to be visualized on x-ray
• Drainage is helped by gravity
  ↓
  **Early morning / Office / Periodic Headache**

**Rhinosinusitis**

• ≤3 months → Acute, >3 months → Chronic
• m.C → Viral
• m.C Bacteria → *Streptococcus pneumoniae*
• Important features:
  → Nasal obstruction
  → Nasal discharge
  → Post nasal drip
  → Headache
  → Facial pain
  → Anosmia

• Examination and management of **Acute rhinosinusitis**
  → Anterior rhinoscopy: Congested mucosa.
• Management:
  → Anti allergics
  → Nasal decongestants
  → Antibiotics (bacterial infection)

Investigation and management of chronic rhinosinusitis

Investigation:
  → Nasal Endoscopy: rigid endoscope
    1<sup>st</sup> pass: Inferior meatus → Nasopharynx
    2<sup>nd</sup> pass: Superior meatus and sphenoethmoidal recess
    3<sup>rd</sup> pass: Middle meatus
  • Congestion
  • Mucopus

  → Best investigation for all chronic sinusitis: NCCT

Management
  → Medical management: (at least 1 month)
    Antibiotics
    Decongestants (<7 days)
    Steroid nasal sprays

  → Best surgical management: FESS (Functional Endoscopic Sinus Surgery) → 1<sup>st</sup> step of FESS:
    Uncinectomy
    - Restores the function of sinuses
    - Rigid endoscope:
      → Tips of different angles (0°, 30°, 45°, 70°)

Indications of FESS

• Chronic sinusitis
• Polyps
• Mucocoele
• Endoscopic surgeries of nose:
  → Deviated nasal septum
  → Ligation of sphenopalatine artery in epistaxis
  → Removing sinonasal tumours
  → Choanal atresia.
  → To access structures around nose:
    - Endoscopic dacryocystorhinostomy (DCR)
    - Orbital decompression
    - CSF leak repair
    - Transphenoidal hypophysectomy

**Complications of FESS**

• Hemorrhage (m.c.)
• Injury to lacrimal duct
• Injury to orbit
• CSF leak,
• Injury to internal carotid artery (most dreaded complication)
• Injury to optic nerve
• Synechiae → mitomycin C applied to prevent this

→ Dangerous area of face: veins communicate with cavernous sinus
→ Dangerous area of nose: Cribriform plate (infection can go intracranially)
→ Venous drainage of sinuses and nose.
Complications of sinusitis

- MC is orbital cellulitis
  - Peri orbital Edema
  - Orbital cellulitis
  - Sub periosteal abscess
  - Orbital abscess
  - Cavernous sinus thrombosis

- Orbital cellulitis:
  - Concurrent involvement of 3, 4, 6 ophthalmic nerves
  - Unilateral
  - Slowly progressive

- Cavernous sinus Thrombosis:
  - 1st nerve to be involved is 6th nerve
  - Bilateral
  - Fulminant presentation

Chronic complications of sinusitis
- Mucocoele
  - MC in frontal sinus

- Management: Antibiotics + FESS
ATROPHIC RHINITIS & GRANULOMATOUS DISEASE OF NOSE

Atrophic rhinitis – Etiology and clinical presentation

- Wide nasal cavities
- Also known as ozaena (foul smell)

Etiology

1) 
2) A\* to granulomatous condition of nose
   a) A\* to tissue destructive surgery ⇒ Empty nose syndrome
   b) 1\* atrophic rhinitis following an infection by Klebsiella ozaenae

Clinical Presentation

1) Patient:
   - Nasal obstruction (because of excessive crusting)
2) Relatives:
   - Complaints of foul smelling nasal discharge from the patient
     ↓
     Merciful (relative) / Blissful (patient) anosmia

Atrophic rhinitis – Management

1) Alkaline nasal pouchoing
   - To remove the nasal crust
   - Method:
     - NaCl, NaHCO₃,
     sodium bicarbonate in a ratio of 2:1:1
     +
     Distilled water
     - Filled in 15 - 20ml syringe and pushed into the nose
       ↓
       Loosens the crust
**Medical Management:**

1) Treat local infection
   
   - **Memicetine solution:** Contains
     - Chloramphenicol
     - Estradiol
     - Propylene Glycol
     - Vitamin D
   
   - 25% glucose in glycerine solution
     - Glycerine: lubricates the nose
     - Glucose: Converts to lactic acid (proteolytic)
       
       Destroys Bacterial Proteins

**Surgical Management:**

2) **Young's operation**
   
   - Closure of nasal cavity
   
   - Method: Raise a flap of mucosa from lateral wall and septum
     
     Structure the flaps

**Modified Young's Operation**

A gap of 2mm is left in the centre

**Granulomatous disease of the nose**

- All granulomatous conditions of the nose
  
  Can cause: Perforation in cartilaginous part of septum,

- Except - Syphilis and Wegener's granulomatosis
  
  Perforation in both bony and cartilaginous part
<table>
<thead>
<tr>
<th><strong>Rhinocleroma</strong></th>
<th><strong>Rhinopidiosis</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Causative agent</strong></td>
<td><strong>Causative agent</strong></td>
</tr>
<tr>
<td><em>M. Rhinoscleromatis</em></td>
<td><em>Rhinopidium seeberi</em></td>
</tr>
<tr>
<td><em>A/K/A Frisch bacillus</em></td>
<td><em>Aquatic protozoa</em></td>
</tr>
<tr>
<td><em>Bacterial granuloma</em></td>
<td></td>
</tr>
<tr>
<td><strong>Clinical Presentation</strong></td>
<td><strong>Clinical Presentation</strong></td>
</tr>
<tr>
<td>- Presents typically as atrophic rhinitis</td>
<td>- More common in Tamil Nadu</td>
</tr>
<tr>
<td>- Hard / woody nose</td>
<td>- H/O taking bath in ponds frequently by animals</td>
</tr>
<tr>
<td>- Hebra nose</td>
<td>- Not confined to nose / nasopharynx (skin nodules)</td>
</tr>
<tr>
<td><strong>On Examination</strong></td>
<td><strong>On Examination</strong></td>
</tr>
<tr>
<td><strong>Diagnosis</strong></td>
<td><strong>Diagnosis</strong></td>
</tr>
<tr>
<td><em>Biopsy (Diagnostic)</em></td>
<td><em>Nasal swab (multiple sporangia)</em></td>
</tr>
<tr>
<td>i) Macrophages</td>
<td><em>Biopsy</em></td>
</tr>
<tr>
<td>- Cytoplasm with phagocytosed bacteria</td>
<td></td>
</tr>
<tr>
<td>mikulicz cells</td>
<td></td>
</tr>
<tr>
<td>i) Plasma cells</td>
<td></td>
</tr>
<tr>
<td>Russel bodies (inclusion body)</td>
<td></td>
</tr>
<tr>
<td><strong>Management</strong></td>
<td><strong>Management</strong></td>
</tr>
<tr>
<td><em>Rifampicin</em></td>
<td><em>Wide excision with cautery of base + Dapsone</em></td>
</tr>
<tr>
<td><em>Streptomycin</em></td>
<td></td>
</tr>
<tr>
<td><em>Tetracycline</em></td>
<td></td>
</tr>
<tr>
<td><em>Ciprofloxacin</em></td>
<td></td>
</tr>
<tr>
<td>{6 weeks}</td>
<td></td>
</tr>
<tr>
<td><strong>Differential Diagnosis</strong></td>
<td><strong>Differential Diagnosis</strong></td>
</tr>
<tr>
<td><em>Hypertrophic rhinitis (mulberry turbinate)</em></td>
<td><em>Angiofibroma</em></td>
</tr>
</tbody>
</table>
Rhinoscleroma.
NASAL POLYP AND FUNGAL SINUSITIS

Nasal polyps

- Mucosal outpouching from paranasal sinuses
- Most acceptable theory → Bernoulli’s theorem:
  (air passes through pathway → velocity increases)

  Infection / Allergy
  ↓

  Edema in middle meatal area
  ↓

  Nasal narrowing
  ↓

  ↑Velocity
  ↓

  Negative pressure
  ↓

  Pulling of mucosa into pathway of air

  Nasal Polyp

  ↓

  From Maxillary
  Antrum

  ↓

  Antrochoanal
  Polyp

  → Multiple
  → Bilateral

  From Ethmoid
  Sinus

  ↓

  Ethmoidal
  Polyp

  → Single
  → Unilateral

  Rarely from
  Frontal sinus

Bilateral multiple polyps

- Follows systemic conditions

Allergic polyps

- Middle age
- Type I Hypersensitivity → ↑ Ig E
- Absolute Eosinophil Count ↑
- Eosinophils ↑
- Management:
  - Antihistamines
  - Leukotriene Inhibitors
  - Mast cell stabilizers
  - Steroid nasal spray ↓ If no benefit
    - FESS (Functional Endoscopic sinus surgery)
  - If it recurs → Management: Same as above

- Samter’s Triad:
  - Allergic rhinitis
  - Asthma
  - Aspirin sensitivity

Bilateral multiple polyps – systemic conditions

Churg Strauss syndrome
- Autoimmune condition
- Allergic manifestation to unknown allergen
- Activation of Eosinophils and vasculitis
- AKA Eosinophilic Granulomatosis
- Adult onset asthma
- Allergic rhinitis + Polyps
- Lung infiltrates seen on chest x-ray
- Eosinophils > 10%
- Management: → Steroids

Cystic Fibrosis
- AKA mucoviscidosis
- Young with multiple bilateral polyps
- Sweat – Chloride Test

Ciliary Dysfunction
- Kartagener’s Syndrome
  - Recurrent Sinusitis
  - Bronchiectasis
  - Situs inversus or Dextrocardia
• Young’s Syndrome
  → Recurrent Sinusitis
  → Bronchiectasis
  → Infertility

Unilateral single polyp

Unilateral Single Polyp

↓

Bacterial  Fungal

↓

Localised  Diffused

→ Infection of only maxillary sinus opening

↓

Antrochoanal Polyp

↓

Diffuse Polyps

main management: FESS

 ENT  •  v2.0  •  Marrow 4.0  •  2020
Fungal sinusitis

- Fungal Ball
  - Aspergillus Niger
  - Immuno-compromised
  - Non-atopic
  - Chronic sinusitis
  - Cheesy secretion
  - Management:
    - FESS
    - No antifungal needed
  - CT: Heterogeneous appearance due to entrapment of metals
  - Double density sign

- Allergic Fungal Sinusitis
  - Fungus entrapped in sinus
  - Aspergillus Niger
  - Immuno-compromised
  - Atopic, allergic to fungus
  - Management:
    - FESS
    - Mucinous secretion (peanut butter)
    - Axial thickening

- Invasive Fungal Sinusitis
  - Mucormycosis
  - Immuno-suppressed (diabetic, on steroids)
  - CT: Entrapment of metals
  - Heterogeneous appearance
  - Double density sign
  - Spreads rapidly
  - Ischemia
  - Necrosis
  - Black discoloration of the area involved
  - Management:
    - IV Amphotericin B
    - Local Debridement

Unilateral polypoidal mass – exceptions

Concha bullosa
- Probe Test: Cannot pass
- Probe all around the mass
- Bleed on probing
- Pain +

Polyp
- Probe Test: can pass
- Probe all around the mass
- No bleeding
- Pain absent

ENT v2.0 • Marrow 4.0 • 2020
Infant
• Polypoidal mass
  ↓
  Rule out meningocele/
  meningoencephalocele
  by (CT)

• Furstenberg Test
  (when child cries → size of mass ↑ due to ↑ ICT)
• Trans illumination

Pubertal Age group
• male, Polypoidal mass, recurrent epistaxis
  ↓
  Angiofibroma

Elderly age group
• Polypoidal mass → Rule out malignancy

Age group based classification of presentation in polyps

1. Infant → meningocele
   → meningoencephalocele

2. Young pubertal age group
   → Unilateral Single: Antrochoanal polyp
   → Bilateral Multiple: Cystic fibrosis
   → Unilateral; Recurrent epistaxis: Angiofibroma
3. Middle age group
   - Bilateral multiple → Allergic ethmoidal polyp
   - Unilateral infection → Bacterial
   → Allergic Fungal Sinusitis

4. Elderly → malignancy
FRACTURES OF FACE AND CSF RHINORRHOEA

Fracture of face

- MC Fracture of face: Fracture of nose
  - Fracture of Nose
    - Class I
      → Following lateral blow
      → Fracture of:
        - Nasal bone + Septum (Septum not deviated)
        - Septum has vertical Fracture
        - Chevallet Fracture
    - Class II
      → Frontal or Lateral blow
      → Fracture of:
        - Nasal bone + Septum (Septum deviated)
        - Septum has Horizontal or C shaped Fracture
      → Jarjaway Fracture
    - Class III
      → Naso-Orbital ethmoid Fracture
      (Pig Nose)

→ management:
  - Closed reduction
    ↓ 1 week (after edema subsides)
    re-impacted/realigned
    ↓
    a nasal splint is applied

* using:
  → Ashs Forceps (Septum)
  → Walsham Forceps (Bony Wall)
• After 3 weeks; Management
  → Rhinoplasty
  → Septorhinoplasty (>17 Years)

CSF rhinorrhoea

• m.C. Cause → Trauma (95%)
  Spontaneous (5%)

  Infective  
  tumor  
  Congenital  
  erosion  
  erosion  
  dehiscence

• m.C. Cause → Fracture of Cribiform plate of Ethmoid
• Diagnosis:

  Clinical Examination  
  → Sniff Test (can't sniff back)
  → Handkerchief Test (do not stiffen)
  → Halo / Target / Double Ring Sign

  Biochemical Examination  
  → β₁ Transferrin
  → β Trace Proteins

  Radiological Examination  
  → HRCT Base of the Skull (Best Investigation)
  → Active
  → CT Cisternography

1. Reservoir sign - make the patient lie down and let him sit suddenly and flex his neck
   gush of fluid from nose

• management: Conservative x 2 weeks
  → ↓ CSF Pressure: - Mannitol
    - Glycerol
    - Diuretics
    - Propped up Position
If no improvement (in 2 weeks) 
Preferably Endoscopic repair or External repair 
(Flushcein Localising Test)

Zygomatic bone fracture

• **Tripod Fracture** = Infraorbital fracture + Zygomatico-
  Temporal fracture + Zygomatico-
  Frontal fracture

• **Infra Orbital fracture / Zygomaticomaxillary fracture**: 
  → Anaesthesia of cheek (infraorbital nerve injury)
  → Flattening of cheek
  → Step deformity of infra orbital margin
  → Periorbital emphysema
  → Diplopia
  → Enophthalmos

• **Zygomatico – Temporal fracture** 
  → Trismus

• **Zygomatico – Frontal fracture** 
  → Step deformity of lateral orbital rim
Blowout fracture

- Fracture of floor of orbit
- Presentation:
  - Anaesthesia of cheek
    - (involvement of infra-orbital nerve)
  - Step deformity of infraorbital margin
  - Periorbital emphysema
  - Restricted Ocular movement
  - Diplopia
  - X-Ray/CT: Tear drop sign

Maxillary bone fracture

- A/A/A Le Fort fracture
- Types:
  - Hanging palate/Hanging Teeth
  - Pyramidal fracture
  - Involvement of infraorbital nerve
  - Guerin Fracture

Mandibular fracture

- M.C site of fracture → Neck/Sub condylar area (weakest part of mandible)
TUMORS OF NOSE

Tumors of nose - benign

Benoign tumors

- External nose
  - Rhinophyma
    - Hypertrophy of sebaceous glands of tip of nose
    - Potato nose appearance seen in - Acne Rosacea
    - Management - Excise skin with knife / Laser

- Nasal cavity
  - Inverted Papilloma
    - mC a/k/a Ringertz tumor / Schneiderian papilloma
    - Cause - Human papilloma virus
      - Arises from lateral wall of nose
      - Locally invasive
      - Recurrent
      - Premalignant
    - Management - Surgical Excision

- Paranasal Sinuses
  - Osteoma - mC
    - mC in frontal sinus
    - Fibrous dysplasia
      - mC in maxilla.
Tumors of nose – malignant

- External nose
  - Basal cell carcinoma
    - Rodent ulcer
- Nasal cavity
  - Squamous cell carcinoma
- Paranasal sinuses
  - Squamous cell carcinoma
    - MC in maxillary sinus
    - MC in Hardwood workers
      - MC is adenocarcinoma of ethmoid

Classification of maxillary sinus carcinoma – Ohngren’s

- Ohngren’s line
  - Passes from the root of nose to the angle of mandible
  - Dividing the maxillary sinus into
    - Suprastructure
      - Carcinoma of suprastructure
      - Poor prognosis
        - Posterior, Superior, Lateral wall of sinus
    - Infrastructure
      - Medial, Inferior, Anterior wall of sinus
Tumor (T) classification of carcinoma of maxilla

*T*₁ - Only mucosa is involved, no bone involvement
*T*₂ - Bony erosion of palate, lateral wall of nose
*T*₃ - Bony erosion of posterior wall of maxillary sinus
    Pterygoid plate, infraorbital margin, medial wall of orbit, infratemporal fossa, skin of cheek
*T*₄ - Involves orbital contents
    Orbital apex
    Frontal / Sphenoid Sinus
    Cribriform plate
    Intracranial
    Nasopharynx

Management of carcinoma of maxilla

- For all the stages → **Surgery** → Followed by → **Radiotherapy**
  ↓
  Partial / Total maxillectomy
  ↓
  with Weber Fergusson incision
  Or
  moure’s incision
  Or
  mid facial degloving incision(sublabial incision,
  (most preferred) - Scar
  not visible externally
ANATOMY OF PHARYNX

Mandibular nerve – divisions

- Sensory branches:
  - External – Auriculotemporal nerve (ATN)
  - Middle – Inferior Alveolar Nerve
  - Inner – Lingual Nerve
  - Buccal branch

- Motor supply:
  - Muscles of mastication
  - Tensor palati
  - Tensor tympani
  - Mylohyoid
  - Anterior belly of digastric

- Conveys parasympathetic supply:
  - IXth nerve
  - Through auriculotemporal nerve
  - To parotid
  - VIIth nerve
  - Through lingual
  - To sublingual
  - Submandibular

Anatomy of pharynx

1. Anterior 2/3rd of tongue
2. Hard palate
3. Floor of mouth
4. Lip, Gingiva, Retromolar trigone
5. Buccal mucosa
6. Vestibule
except
   a) Soft palate
   b) Posterior 1/3rd of tongue \{ Contents of oropharynx \)

b) Pharynx - extent and parts
   * Extent:
     Base of skull to lower border of cricoid
   * Parts:

Based on the structures anterior to it
   - Nasopharynx / Epipharynx
   - Oropharynx
   - Laryngopharynx / Hypopharynx

<table>
<thead>
<tr>
<th>Parts</th>
<th>Extent</th>
<th>Vertebrae</th>
</tr>
</thead>
<tbody>
<tr>
<td>* Nasopharynx</td>
<td>Base of skull to hard palate</td>
<td>( c_1 )</td>
</tr>
<tr>
<td>* Oropharynx</td>
<td>Hard palate to hyoid bone</td>
<td>( c_2, c_3 ) (upper part)</td>
</tr>
<tr>
<td>* Laryngopharynx</td>
<td>Hyoid bone to lower border of cricoid</td>
<td>( c_4 ) (lower) - ( c_6 )</td>
</tr>
</tbody>
</table>

**Posterior pharyngeal wall**

a) Lining Epithelium
   - Nasopharynx: Pseudostratified ciliated columnar (adenoids)
   - Oropharynx: Stratified squamous (Tonsils)
   - Hypopharynx: Stratified squamous
   - Soft palate: Lined by both - Oropharyngeal surface:
     Stratified squamous
- Nasopharyngeal surface:
  - Pseudostratified ciliated columnar

b) Waldeyer’s Ring

- Composed of a collection of lymphoid tissue
- Function: Provide local / systemic immunity in children
- Gets atrophied in the later age

Contents:
1. Adenoids
   - Also known as Luschka’s tonsils / Nasopharyngeal tonsils
   - Located in nasopharynx

2. Tubal tonsils
   - Also known as Gerlach’s tonsils
   - Located behind Eustachian tube

3. Palatine tonsils
   - Also known as Faucial tonsils
   - Located in oropharynx

4. Lingual tonsils
   - Located in the base of the tongue

<table>
<thead>
<tr>
<th>Parts</th>
<th>Tonsils &amp; Epithelium</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nasopharynx</td>
<td>Adenoids, tubal tonsils (ciliated columnar)</td>
</tr>
<tr>
<td>Oropharynx</td>
<td>Palatine, lingual tonsils (stratified squamous)</td>
</tr>
<tr>
<td>Hypopharynx</td>
<td>No Waldeyer’s ring</td>
</tr>
</tbody>
</table>
Muscles of pharynx

- Muscles of pharynx are bounded,
  - Anteriorly by Pharyngobasilar fascia
  - Posteriorly by Buccopharyngeal fascia
- Pharyngobasilar fascia ⇒ Forms capsule of tonsils

**Longitudinal muscles (Dilators)**
1. Stylopharyngeus (Styloid)
2. Salpingopharyngeus (Eustachian tube)
3. Palatopharyngeus (Palate)

**Circular muscles**
1. Superior constrictor
2. Middle constrictor
3. Inferior constrictor

Attaches to posterior border of Thyroid

Superior and inferior constrictors

1. Superior constrictor
   - Free upper border (not attached to base of skull)
- Foramen of Morgagni
  - Gap between base of skull and superior constrictor
  - Filled with pharyngobasilar fascia
  - Structures passing:
    T - Tensor palati
    A - Ascending palatine artery
    A - Palatine branch of ascending pharyngeal artery
    L - Levator palati
    A - Auditory tube

a. Inferior Constrictor

- Thyropharyngeus
- Cricopharyngeus

Potential area of weakness between the two muscles

- Killian's Dehiscence / Gateway of Tear (Posterior Gap)

- Zenker's Diverticulum
  (Due to neuromuscular incoordination between Cricopharynx and Hypopharynx)

- Pulsion diverticulum (No muscle layer, \( \therefore \) False diverticulum)

Zenker's diverticulum – clinical presentation and management

- Clinical Presentation:
  (i) Dysphagia
  (ii) Halitosis
(iv) Hoarseness
(v) Cough
(vi) Lung complications

a) Swelling in the neck - anteriorly
   (most commonly on left side)

   ↓

   On palpation: Gurgling sound +
   ↓
   Boyce Sign

b) Seen only in elderly

• management:

   Barium swallow

Investigations:
• Endoscopy
• Barium swallow - lateral view (best)

Treatment:
• Excision (external procedure) ⇒ incase of a large pouch
• Cricopharyngeal myotomy (cutting of the common wall)

   ↓

   Laser technique
   (for pouch < 2 cm)
   ↓
   Dohlman's procedure

   Endoscopic diverticulotomy
   (recommended)
   (for pouch > 2 cm)
   ↓
   Cuts the wall and staples the edges simultaneously
Pharynx – nerve supply

<table>
<thead>
<tr>
<th>Part</th>
<th>Constrictors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nasopharynx</td>
<td>Superior constrictor</td>
</tr>
<tr>
<td>Hypopharynx</td>
<td>Inferior constrictor</td>
</tr>
<tr>
<td>Oropharynx</td>
<td>Most of lateral wall by Superior constrictor</td>
</tr>
</tbody>
</table>

- Pharynx is supplied by 9th and 10th nerve

Nerves passing:
- Gap between Superior and middle constrictor ⇒ 9th nerve
- Gap between middle and inferior constrictor
  ↓
  Internal laryngeal nerve, branch of superior laryngeal nerve
- Gap between inferior constrictor and esophagus
  ↓
  Recurrent laryngeal nerve

- Glossopharyngeal nerve
  - Sensory supply
  - Supplies oropharynx, tonsils, base of tongue
  - Enters through lower pole of tonsil
ANATOMY: SPACES OF PHARYNX

• Behind the pharyngeal muscles:
  - G → Buccopharyngeal Fascia
  - A → Alar Fascia
  - P → Prevertebral Fascia.

• Capsule of tonsil is formed by pharyngobasilar fascia

• Peritonsillar space
  → Space between capsule of tonsil medially and superior constrictor muscle laterally

• Peritonsillar abscess (Quinsy)
  ↓
  Pushes tonsil medially

• Retropharyngeal space
  → Space between Buccopharyngeal fascia and Alar fascia.

• Dangerous space
  → Space between Alar fascia and prevertebral fascia.

• Prevertebral space
  → Space between prevertebral fascia and vertebra.
# Spaces of Pharynx: Boundaries and Contents

<table>
<thead>
<tr>
<th>Spaces</th>
<th>Upper</th>
<th>Lower</th>
<th>Anterior</th>
<th>Posterior</th>
<th>Contents</th>
</tr>
</thead>
<tbody>
<tr>
<td>Retropharyngeal space of Gillette</td>
<td>Base of skull</td>
<td>T&lt;sub&gt;n&lt;/sub&gt;</td>
<td>Buccopharyngeal Fascia</td>
<td>Alar Fascia</td>
<td>Lymph nodes of Rouvier</td>
</tr>
<tr>
<td>Danger space</td>
<td>Base of skull</td>
<td>Diaphragm</td>
<td>Alar Fascia</td>
<td>Prevertebral Fascia</td>
<td></td>
</tr>
<tr>
<td>Prevertebral space</td>
<td>Base of skull</td>
<td>T&lt;sub&gt;n&lt;/sub&gt;</td>
<td>Prevertebral Fascia</td>
<td>Vertebrae</td>
<td></td>
</tr>
<tr>
<td>Parapharyngeal space</td>
<td>Base of skull</td>
<td>Hyoid</td>
<td>Buccopharyngeal Fascia</td>
<td>→ medial</td>
<td>ICA</td>
</tr>
<tr>
<td>(smallest)</td>
<td></td>
<td></td>
<td></td>
<td>pterygoid</td>
<td>IJV</td>
</tr>
<tr>
<td>(infected)</td>
<td></td>
<td></td>
<td></td>
<td>mandible</td>
<td>9&lt;sup&gt;th&lt;/sup&gt; CN</td>
</tr>
<tr>
<td>(A/V/A Pharyngo-maxillary space)</td>
<td></td>
<td></td>
<td></td>
<td>masseter</td>
<td>10&lt;sup&gt;th&lt;/sup&gt; CN</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Parotid</td>
<td>11&lt;sup&gt;th&lt;/sup&gt; CN</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>12&lt;sup&gt;th&lt;/sup&gt; CN</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Cervical sympathetic chain</td>
</tr>
<tr>
<td>Peritonsillar space</td>
<td>Base of skull</td>
<td>Capsule of Tonsil</td>
<td>→ pharyngobasilar Fascia</td>
<td>Superior constrictor muscle</td>
<td>Fat</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Bulge in posterior pharyngeal wall**

- **Midline Bulge**
  - Collection in prevertebral space (m.c. in children)
  - Space of Gillette (nodes of Rouvier)
  - Drains naso and oropharynx
Parapharyngeal space

- Abscess → Trismus
- Styloid process with its muscle attachments divides parapharyngeal space into:
  - Anterior compartment (pre-styloid compartment)
  - Posterior compartment (post-styloid compartment)
- Structures passing through posterior compartment:
  -ICA
  -IJV
  -9, 10, 11, 12 CN
  -Cervical sympathetic chain
- Ca. Nasopharynx → Parapharyngeal space → Cervical sympathetic chain → Horner syndrome
- Abscess in anterior compartment → Tonsil is pushed medially
Adenoids & structures of lateral wall of nasopharynx

Adenoids:

- Nasopharynx is also known as Epipharynx
- Roof of Nasopharynx
  ↓
  Collection of lymphoid tissue in midline
  ↓
  Adenoids / Luschka tonsil / Nasopharyngeal tonsil

Structures of lateral wall of nasopharynx:
1) 1 cm posterior to posterior end of inferior turbinate
  ↓
  Opening of eustachian tube
- Eustachian tube is cartilaginous

a) Bulge produced by eustachian tube opening: "Torus tubarius"

b) Fossa behind torus tubarius → "Fossa of Rosenmuller"
  ↓
  MC site of origin for Nasopharyngeal Carcinoma
Pterygomaxillary fascia:
Boundaries:
- Anteriorly - Posterior wall of maxillary sinus
- Posteriorly by pterygoid plate
- Medially by lateral wall of nasopharynx
- Laterally - open

Pterygomaxillary fossa

Communicates with infratemporal fossa.

4) 1 cm posterior to posterior end of middle turbinate

Opening of sphenopalatine foramen

MC site of origin of Angiofibroma.

- In case of unilateral serous otitis media.

Rule out Nasopharyngeal cancer

- Adenoid hypertrophy → Bilateral obstruction of eustachian tube

- Nasopharyngeal Ca. → Unilateral obstruction of eustachian tube

Anatomy of oropharynx

- Bounded superiority by hard palate
- Inferiorly between hard palate and hyoid bone
Lateral wall of oropharynx:
- Superiorly - Soft palate
- Anteroinferiorly - Base of tongue
- Laterally - Tonsils
  ↓
  - Anterior Pillar - Extends from palate to tongue
  ↓
  Palatoglossus muscle
  - Posterior pillar - Extends from palate to pharynx
  ↓
  Palatopharyngeus muscle
  ↓
  Longitudinal fibers  Circular fibers
  ↓
  1) Stylopharyngeus  Fuse with fibers of
  2) Salpingopharyngeus  superior constrictor
  3) Palatopharyngeus  muscle
  ↓
  Passavant's Ridge
- During swallowing: Soft palate moves posteriorly and Passavant's ridge formed, which moves anteriorly
  ↓
  Preventing food entry to nasopharynx
- Velopharyngeal insufficiency:
  Food is regurgitated nasally
  ↓
  Producing hypernasal voice (Rhinolalia aperta)

Rhinolalia aperta and rhinolalia clausa

<table>
<thead>
<tr>
<th>Rhinolalia aperta (Hypernasality)</th>
<th>Rhinolalia clausa (Hyponasality)</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Causes:</td>
<td>- Obstruction of Nasopharynx</td>
</tr>
<tr>
<td>1) Cleft palate</td>
<td>- Causes:</td>
</tr>
<tr>
<td>2) Sub mucosal cleft</td>
<td>1) Adenoid hypertrophy (mc)</td>
</tr>
<tr>
<td>3) Palatal paralysis</td>
<td>2) Nasopharyngeal Ca.</td>
</tr>
<tr>
<td>4) Bifid uvula</td>
<td>3) B/L Nasal polyps</td>
</tr>
</tbody>
</table>

ENT • v2.0 • Marrow 4.0 • 2020
Anatomy of hypopharynx

Boundary:
- Superiorly at the level of hyoid
- Inferiorly at the level of cricoid
- Hypopharynx situated behind larynx

"laryngopharynx"

- Endoscopic picture showing
  Eustachian tube opening
  (Torus tubarius)

- Endoscopic picture showing
  - Aryepiglottic fold
  - Pyriform fossa
  - Posterior pharyngeal wall

- Pyriform fossa
  1) Lateral channel for food
  2) MC site for foreign body lodgement
Nerve supply

Sensory supply

- Nasopharynx
  - Maxillary nerve
- Oropharynx
  - Glossopharyngeal nerve
    - Except for base of tongue
    - Superior laryngeal Nerve
- Hypopharynx
  - Upper
  - Lower
    - Recurrent laryngeal Nerve

Motor nerve supply - vagus [carries fibers of XI]

- Longitudinal muscle
  - Except Stylopharyngeus
    - 3rd arch
- Palatine muscle
  - Except Tensor palati
- Constrictor muscle
  - (Superior, middle and inferior)
- Mandibular Nerve

ENT • v2.0 • Marrow 4.0 • 2020
ADENOID HYPERTROPHY AND
THORNSWALDT BURSITIS

Adenoid hypertrophy

- Symptoms:
  1. Nasal obstruction
     → Pinched up nose
     → Nasolabial crease absent
     → Mouth Breathing
        - Palate → High arched
        - Crowding of teeth anteriorly
  2. Eustachian Tube obstruction
     → Serous otitis media (SOM) → CHL → Dull look on face
     → Recurrent Acute otitis media (AOM)
  3. Rhinolalia clausa
  4. Sleep apnea

- Investigation:
  → X-ray Nasopharynx (lateral view)
  → Endoscopic grading:
    Grade I → ⅓ rd obstruction
    Grade II → ⅓ - ⅔ rd obstruction
    Grade III → ⅔ - complete obstruction
    Grade IV → Complete obstruction
Management of adenoid hypertrophy

→ medical management
   Antiallergics
   Antibiotics
   Steroid Nasal Spray

→ Surgery done only when the symptoms become chronic
→ Indications for Adenoidectomy
  1. Nasal obstruction → Recurrent Sinusitis
  2. Eustachian tube obstruction → Chronic OM Recurrent AOM
  3. Sleep Apnea → >5 apneas/hr or >30 apneas/7 hrs

Surgery in adenoid hypertrophy

• Position: Rose’s position
  ↓
  Extension at atlanto occipital position

• Boyle’s and Davis mouth gag is used
  → They are connected using a device: Draf tin Bipod stand

• Coablation
  → uses radio frequency waves

• Cauterization
  → Diathermy

• Curettage
• microdebrider

Hot methods
Cold methods

Visualisation under endoscopy
Contraindications for adenoidectomy and post-operative complications

Contraindication:
* velopharyngeal insufficiency
  (cleft palate)

Post-operative complications:
1. Hemorrhage
2. Injury to Eustachian tube
3. velopharyngeal insufficiency
4. Coroner’s clot
5. Grisel syndrome
   → Inflammatory subluxation of Atlanto-occipital joint

Thornwaldt’s bursitis

* Thornwaldt’s Bursa (A/V/A pharyngeal bursa)
  → Remnant of communication between primitive pharynx and primitive notochord
  → (roof of nasopharynx)

* Infection of Thornwaldt’s Bursa
  ↓
  → Recurrent post nasal drip
  → Head ache (occipital)
• Investigation: CT/MRI
• Management:
  → Antibiotics
  → Excision
  → If cyst formation
  → Marsupialization
ANGIOFIBROMA

- MC benign tumour of nasopharynx
- Locally invasive
- Arises from Sphenopalantine foramen
- In males → Starts during puberty
  * Spread:
    1. Medial spread
       → Nasopharynx and nose
       → Sinuses
    11. Lateral spread
       → Sphenopalatine fossa
       ↓
       → (completely fill) + orbit (proptosis)
       Frog Facies
       → Infratemporal fossa
       (swelling cheek)
    111. Intracranial spread
       → Limited
       → Extensive

----

Radkowski’s staging:

* Stage I
  → 1a
  → 1b

* Stage II
  → 1la

* Stage III
  → 1lb
  → 1lc
  → 111 a
  → 111 b

Investigation and management of angiofibroma

* Blood supply of Angiofibroma → maxillary artery
* Clinical presentation: Recurrent epistaxis in young patient
* Best investigation: CECT
  → Holliman Miller Sign / Antral Sign

![Normal CECT](image1)

![Antral sign](image2)
management

- Surgical Excision
  - Pre-operative Embolisation (arterial supply → maxillary artery)
- Radiotherapy: Unresectable intracranial extension (Stage III b)
- Approach
  - Endoscopic
  - Transpalatal
  - Transmaxillary → Lateral Rhinotomy
    → Mid facial degloving
  → Infratemporal
- Biopsy is contraindicated (no muscular coat)

Crescent of air seen in between mass and pharyngeal wall
↓
Not Angiofibroma.
↓
But Antrochoanal polyp (crescent sign/Dodd sign)

No crescent of air seen
↓
Angiofibroma.
NASOPHARYNGEAL CARCINOMA

- MC cancer of Nasopharynx
- Arises from fossa of Rosenmüller
- MC in south east Asia - MC in south China
  
  A/H/ A Guangdong carcinoma
  (seen in Guangdong province)

- MC in mongolians

  **Etiology**

  ![Etiology Diagram]

  Genetic
  Runs in families
  All family members should be screened

  Genetic
  Runs in families
  All family members should be screened

  **Ebstein barr virus**
  Antigen used for screening
  Capsid antigen
  Early antigen
  IgA/VCA
  (viral capsid antigen)

  **Nitrosamines**
  used in food preservatives

Nasopharyngeal carcinoma – Symptoms

- Does not present with nasopharyngeal symptoms usually
- In 70% – upper deep cervical lymph node swelling – 1st symptom
• Nasal obstruction
• Eustachian tube obstruction → unilateral serous otitis media.

• If the tumor grows
  \[ \text{Laterally} \quad \text{Superiorly} \]
  \[ \text{Para pharyngeal space} \quad \text{Intracranial cavity} \]
  \[ \text{9, 10, 11, 12 cranial Nerves} \quad \text{Sinus of Morgagni} \]
  \[ \text{(sympathetic chain)} \quad \text{called Sinus of Morgagni syndrome or Trotter's triad} \]
  \[ \text{Involved} \quad \text{N - Neuralgia, in 5th nerve distribution}} \]
  \[ \text{Causes - Horner's syndrome} \quad \text{P - Palatal palsy} \]
  \[ \text{C - Conductive hearing loss} \]
  \[ \text{(B curve on tympanometry)} \]

Nasopharyngeal carcinoma – Management 00:13:46

• Diagnosis – Endoscopy
  \[ \text{Biopsy} \quad \text{CT/MRI} \]

management
• Radiosensitive tumors, poor prognosis
  Stage I  – Radiotherapy
  Stage II – Radiotherapy
  Stage III – Concurrent chemoradiation
  \[ \text{Radiotherapy + Chemotherapy given in cycles} \]
  Stage IV – Concurrent chemoradiation
WHO Classification

1. Keratinizing squamous cell carcinoma
2. Non keratinizing squamous cell carcinoma

\[ \text{Differentiated} \quad \text{Undifferentiated} - \text{mC variant in endemic regions} \]
INFECTIONS OF TONSILS AND TONSILLECTOMY

**Tonsils**

- Arise from lymph node tissue
- Component of "Waldeyer's ring"
- Develop from 2nd pharyngeal pouch
- Remnant of and pharyngeal pouch - "Crypta magna"
- Lined by squamous epithelium (stratified)
- Capsule is formed by pharyngobasilar fascia

![Diagram of Tonsils]

- Sensory nerve supply to pharynx
  - Glossopharyngeal Nerve
- Lymphatic drainage: upper deep cervical LN
  - Tonsilar LN

**Vascular supply to tonsil**

- In peritonsilar space:
  - Peritonsilar vein / External palatine vein
  - Most cause for Hemorrhage following tonsillectomy
Arterial supply
Branches of External carotid Artery
1) Ascending pharyngeal artery
2) Lingual artery
3) Facial artery
   - Ascending palatine branch
   - Tonsilar artery (main artery)
4) Maxillary artery

Acute tonsillitis

Clinical presentation:
- Sore throat
- Fever
- Odynophagia
- Pain in ear (IX nerve)
- Enlarged upper deep cervical lymph nodes

- Most Organism: β Hemolytic streptococci
- Management:
  1) Antibiotics - Amoxicillin (mc)
  2) Analgesics
DD for membrane over tonsil:

"ALL VITAMIN D"

A - Agranulocytosis
L - Leukemia
V - Vincent's angina
I - Infectious mononucleosis
↓
Paul Bunnel monospot test
↓
Atypical lymphocytosis

T - Trauma
A - Aphthous ulcer
M - Moniliasis (Candidiasis)
N - Infections of throat
N - Neoplasia
D - Diphtheria
↓
- Caused by: Corynebacterium diphtheriae
  - Unimmunised child
  - Bull neck
  - Pseudomembrane (+) → Extend into soft palate
  - Not limited to tonsil
  - Bleeds on removal
  
  - Management:
    1) Antitoxin
    a) Antibiotics - β lactams
       macrolides

Vincent's angina
- Membrane over tonsil
- Swollen, bleeding
  dirty gingiva
  ↓
  "Necrotising gingivitis"
- Caused by:
  "Borrelia Vincenti"

"F. Fusiformis" (anaerobe)

**Tonsillectomy**

Indications of tonsillectomy
1) Recurrent tonsillitis:
   \[ \downarrow \]
   3/yr x 3 years
   or
   5/yr x 2 years
   or
   7 x 1 year

2) Peritonsillar abscess (1 episode - child, 2 episodes - adult)
3) Febrile seizures
4) Chronic tonsillitis
   - CF:
     - Flushing of anterior pillar
     - Enlarged upper cervical lymph node
     - "Irwin Moore sign"
5) Approach to:
   - Styloid process excision - in "Eagle's syndrome"
     or
   "Stygalgia"

**Spaces of pharynx**

**Diagram**
- Glossopharyngeal neurectomy
  ↓
  In glossopharyngeal Neuralgia.

Position: “Rose’s position”

- Extension at
  1) Cervical joint
  2) Atlanto-occipital joint

- Davis mouth gag ‘used – To keep mouth open

- Mouth kept open
  ↓
  Tongue retracted
  ↓
  Upper jaw retracted
  ↓
  Fixed

Cold method

1) ‘Dissection and snare’
   - Snare is used to crush the pedicle and cut
2) **Cautery - Diathermy**
   - Both bipolar and unipolar cautery used

3) **Coblation** by coablation wand

4) **Laser**

- Tonsillectomy
  - Intracapsular
    - Tonsil not completely removed
      - Obstructive causes (obstructive sleep apnoea)
  - Extracapsular
    - Tonsil + Capsule removed
      - Infective causes
Complications of tonsillectomy

Hemorrhage

- Most common cause
- Route: Venous → "External Paratonsillar Vein"

Primary
- During surgery

Reactionary
- After surgery within 24hrs

Secondary
- >24hrs - 10 days
  - 2° to injections
  - Most common: 5 - 6 days
ABSCESS OF PHARYNX

Spaces of pharynx

Posteriorly

Laterally

Retropharyngeal space

Peritonsillar space

Parapharyngeal space

Dangerous space

Prevertebral space

Peritonsillar abscess

- Abscess in Peritonsillar Space
- mC in adults — the crypta magna—near the capsule of tonsil (due to atrophied tonsil)
  
  So infection reaches the space easily

- Occurs through crypta magna
Clinical presentation
- Fever
- Odynophagia
- Pain in ear
  - Due to 9th nerve involvement
- Muffled speech
  - Hot potato voice
- Trismus

Peritonsilar abscess – Management

1. IV Antibiotics – Against streptococcus
   + Anaerobic
2. Aspiration of pus
3. Incision & Drainage (I & D)
4. Interval tonsillectomy – Done after 1 episode in children
   ↓
   After 2 episodes in adults
   After 6 weeks of last episode
Quinsy and parapharyngeal abscess

→ Parapharyngeal space anatomy

<table>
<thead>
<tr>
<th></th>
<th>Quinsy</th>
<th>Parapharyngeal Abscess</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tonsil pushed medially</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Trismus</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Bulge at angle of jaw</td>
<td>-</td>
<td>+</td>
</tr>
</tbody>
</table>
| Management             | IV Antibiotics ± 1 9 D by intra oral incision | IV Antibiotics and 1 9 D by external incision below lower border (a finger below) ↓
|                        |        | To avoid damage to mandibular branch of facial nerve |

ENT • v2.0 • Marrow 4.0 • 2020
Lugwig's angina

- Submandibular space
  Divided by mylohyoid into
  Sublingual space
  Submental space

Ludwig's Angina

- Cellulitis of submandibular space
- MC cause
  Dental caries
- C/F
  - Difficulty in eating
  - Respiratory distress
  - Difficulty in talking
  - Woody/branzy induration (skin under chin)

Management
- Antibiotics + 19 D
  - 19 D to release tissue pressure
  - Bilateral incision (from one angle of mandible to other)
Retropharyngeal abscess and prevertebral abscess

Unilateral bulge - Retropharyngeal
Bilateral bulge - Prevertebral

Normal prevertebral shadow
At C4 - 7 mm
At C6 - in children - 14 mm
In adults - 21 mm

<table>
<thead>
<tr>
<th></th>
<th>Retropharyngeal abscess</th>
<th>Prevertebral Abscess</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prevertebral shadow</td>
<td>(+)</td>
<td>(+)</td>
</tr>
<tr>
<td>on x-ray - ↑</td>
<td></td>
<td></td>
</tr>
<tr>
<td>unilateral</td>
<td>(+)</td>
<td>midline diffuse</td>
</tr>
<tr>
<td>paramedian bulge of</td>
<td></td>
<td>bulge of posterior</td>
</tr>
<tr>
<td>posterior pharyngeal</td>
<td></td>
<td>pharyngeal wall</td>
</tr>
<tr>
<td>wall</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Retropharyngeal Abscess

- Acute
  - In children
    - Cause: trauma
  - In adults
    - Cause: infection of lymph nodes of Rouviere
    - Management: Antibiotics + 1 D

- Chronic
  - MC in adults
  - Cause: TB spine
  - Management: Anti-tubercular therapy (ATT)
RANULA AND TUMORS OF PHARYNX

**Ranula**

- Ranula → Swelling in the floor of mouth
  - Appears like frog's belly (Rana-Frog)

- Extravasation cyst of the sublingual salivary gland
  - Due to obstruction of the salivary duct

- Transilluminant

- Management - Marsupialisation - Recurrence is ↑
  - Best method - Excision of cyst + sublingual gland

- There can be injury to

  - Lingual nerve
  - Submandibular duct

- Ranula - if it grows (mylohyoid - Divides the floor of mouth in two)

  - Into sublingual space
    - Presents as intraoral ranula

  - Into submental space
    - Pierces mylohyoid and presents as external swelling
    - Plunging ranula
Ranula

Intraoral ranula and Plunging ranula.

Premalignant conditions of oral cavity

Leukoplakia
- Whitish patch
- MUC in smokers
- Management - Close follow up and smoking cessation

Erythroplakia
- Reddish plaque
- More premalignant
- Management - Excision → Biopsy

ENT • v2.0 • Marrow 4.0 • 2020
Submucous fibrosis
- Following chewing of betel nut
- C/F: Trismus — initially blisters → Rupture → Fibrosis
- On examination — white blanched appearance
- Management — Intralesional injection of steroids

Tumors of pharynx

m.C — Squamous cell carcinoma

Site

<table>
<thead>
<tr>
<th>Site</th>
<th>Lip-m.C</th>
<th>Oral cavity</th>
<th>Oropharynx</th>
<th>Hypopharynx</th>
</tr>
</thead>
<tbody>
<tr>
<td>98%</td>
<td>2%</td>
<td>Lateral border of tongue</td>
<td>Tonsil</td>
<td>m.C</td>
</tr>
<tr>
<td>Lower lip</td>
<td>Upper lip</td>
<td>In India</td>
<td>Pyriform fossa</td>
<td>Plummer Vinson syndrome</td>
</tr>
<tr>
<td>Basal cell carcinoma</td>
<td>Buccal alveolar sulcus</td>
<td></td>
<td>Post cricoid carcinoma</td>
<td>In 10-15%</td>
</tr>
</tbody>
</table>

Plummer Vinson syndrome
- In females — age 40-50 yrs
- Iron deficiency anemia
- Post cricoid web
- Upper esophageal dysphagia / Post cricoid dysphagia
Staging of carcinoma of pharynx

T-tumor

$T_1$ $T_a$ $T_3$ $T_4$

Size - $< 2$ cm $> 2$ cm $> 4$ cm Locally
Depth - $< 5$ mm $> 5$ mm $> 10$ mm $> 10$ mm advanced

Staging of carcinoma of pharynx - lymph node

$N_0$ $N_1$

No lymph node Ipsilateral

$N_1 a$ $N_1 b$

Ipsilateral multiple Contralateral

$N_2 a$ $N_2 b$

$> 6$ cm Extra nodal invasion Present

Without extranodal invasion
ANATOMY OF LARYNX, LARYNGOMALACIA, VOCALNODULE, REINKE’S EDEMA AND CONTACT ULCER-I

Anatomy of larynx

Larynx

3 Unpaired cartilages

- Thyroid
  - Largest
  - The two-ala. meet at an angle
    - In males-90° (Adam's apple)
    - In female-120°
    - Ala meets in Centre
      - Thyroid notch
  - Ala -ends Project as
    - Superior cornu
    - Inferior cornu

- Epiglottis
  - Leaf shaped
- Cricoid
  - Only complete ring of larynx (signet ring cartilage)

3 Paired cartilages

1) Arytenoid
  - Pyramid in shape
  - 2 process
    - Vocal
    - Muscular
    - True vocal cord attaches

2) Corniculate
  - Sites on apex of Arytenoid
  - A/V/A cartilage of Santorini

3) Cuneiform cartilage
  - Next to corniculate
  - A/V/A wisberg Cartilage

• A - Arytenoid cartilage
  C - Cricoid cartilage
  T - Thyroid cartilage

• Epiglottis - elastic cartilage

Hyaline cartilage
Extrinsic membranes

- Extrinsic membrane - Connects laryngeal cartilage with nearby structures

  - Hyoepiglottic Ligament
    - Connects
      - Hyoid bone
      - To epiglottis

  - Thyrohyoid membrane
    - Connects
      - hyoid bone to thyroid cartilage

  - Cricotracheal membrane
    - Connects
      - Trachea to cricoid cartilage

    - Internal branch of superior laryngeal nerve pierces & enters larynx
Intrinsic membranes-quadrangular

- Intrinsic membranes - Connects two laryngeal cartilage and quadrangular membranes

  Attached on both sides of epiglottis

  \[\begin{align*}
  & \text{upper border} \\
  & \text{Aryepiglottic fold} \\
  & \text{false vocal cord} \\
  & \text{Connects epiglottis to arytenoid}
  \end{align*}\]

Intrinsic membrane – Conus elasticus

- Conus elasticus - Arises from upper border of cricoid cartilage

  A/V/A cricovocal membrane

  \[\begin{align*}
  & \text{upper border} \\
  & \text{Forms} \\
  & \text{Anterior part between cricoid and thyroid} \\
  & \text{Cricothyroid membrane} \\
  & \text{At the level} \\
  & \text{above} \\
  & \text{below} \\
  & \text{of true vocal cord} \\
  & \text{true vocal cord} \\
  & \text{true vocal cord} \\
  & \text{Glottis} \\
  & \text{Supraglottic} \\
  & \text{Subglottic}
  \end{align*}\]

- Rima glottidis – Space in between two true vocal cords
- Ventricle - Space in between the false and true vocal cords
Subsites of larynx

Supraglottis
- Above true vocal cord (TVC)
  - Components
    - Epiglottis
    - Aryepiglottic fold
    - Arytenoid cartilage
    - Quadrangular membrane
    - Ventricles
    - False vocal cord

Glottis
- At true vocal cord till 1 cm below it

Subglottis
- Below true vocal cord (glottis)

Laryngomalacia – lesion of supraglottis

- Mal Congenital anomaly of larynx
- Congenital laryngeal stridor
  - Clinical presentation – Inspiratory stridor
    - In prone position
    - In supine position
• Diagnosis - FOL (Fibre optic laryngoscopy)
  └── Inlet-large & Floppy
  └── Excessive tissue in supraglottis
      └── Large, curled up, omega-shaped epiglottis
      └── Short & Floppy aryepiglottic folds
      └── Large arytenoids

• Management - Reassurance
  └── Disappears by a year or more

Subsites of larynx - Glottis

Glottis
  └── Components - Two true vocal cords
      └── Attach anteriorly to thyroid cartilage
          └── Attaches posteriorly to vocal process of arytenoid
              └── Anterior commissure
                  └── Posterior commissure

• Anterior 2/3 part - Membranous
  └── Posterior 1/3 part - Cartilaginous
* maximum vibration is at midpoint of membranous vocal cord (junction of anterior ⅔rd and middle ⅓rd of true vocal cord)

So, MC site of vocal nodule/vocal polyp

---

**Vocal nodule and vocal polyp**

<table>
<thead>
<tr>
<th>Vocal nodule</th>
<th>Vocal polyp</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bilateral sessile</td>
<td>Unilateral pedunculated</td>
</tr>
<tr>
<td>Site-midpoint of membranous part/junction of anterior ⅔rd posterior ⅓rd/junction of anterior ⅔rd and middle ⅓rd</td>
<td>→ Same</td>
</tr>
<tr>
<td>A/V/A - Screamers/singers/teachers nodule due to prolonged voice abuse</td>
<td>Sudden episode of voice abuse</td>
</tr>
<tr>
<td>management - voice rest speech therapy</td>
<td>Excision by MLS (microlaryngeal surgery)</td>
</tr>
</tbody>
</table>
Conditions affecting posterior true vocal cord (GLOTTIS)

- Intubation granuloma
- Laryngopharyngeal reflux
- Contact ulcer - when there is abuse of the voice with forceful speech especially at the initiation of speech

\[ \downarrow \]

The two vocal process of arytenoid slam into each other

\[ \downarrow \]

Causes Heaping up of epithelium on one side and on contralateral side causes depression

\[ \downarrow \]

Appear like ulcer - not a true ulcer called contact ulcer/kissing ulcer/ Pachydermia laryngis

- Carcinoma
ANATOMY OF LARYNX, LARYNGOMALACIA, VOCAL NODULE, LARYNGOCELE, REINKE’S ODEMA AND CONTACT ULCER-2

Cavity of larynx

Inlet - Components - Epiglottis

Aryepiglottic folds
Arytenoid cartilage

Vestibule
(cavity between Quadrangular membranes)

Rima vestibuli
(Space in between two false vocal cords)

Ventricle (between false and true vocal cords) → Saccule
(lateral extension of ventricle)

Rima glottidis
contains mucus glands
(Lubricates the vocal cord)

Hyoid bone

Thyroid cartilage

Paraglottic space

Cricoid cartilage

Inlet

Quadrangular membrane

Ventricle

Cricovocal membrane

ENT • v2.0 • Marrow 4.0 • 2020
Laryngocoele

- If the saccule is enlarged

Seen in

- Glass blowers
- Trumpet players
- Carcinoma of ventricle

Diagnosis

- On valsala maneuver swelling increases
- On compressing the swelling sudden gush of air into larynx

Presentation

- Can present as
  - Intrinsic laryngocoele
  - Extrinsic laryngocoele

Management

- Excision

Produces sound

Bryce Sign (hissing sound)
Spaces of larynx

- Lateral space
  - Paraglottic space
    - Of Tucker
  - Boundaries
    - Laterally-Thyroid cartilage
    - Medially-Quadrate membrane - Conus elasticus
    - Posteriorly-pyriform fossa

- Anterior space
  - Pre-epiglottic space
    - Of Boyer

- Reinke's space
  - Submucosal space of true vocal cord

Epithelium in larynx
- True vocal cord is lined by stratified squamous epithelium
- Rest of the larynx - lined by - Ciliated columnar epithelium

Lymphatics of larynx

- Supraglottis
  - Upper deep cervical lymph node
- Glottis
  - No Lymphatics
- Subglottis
  - Lower deep cervical lymph node (LN)
  - Carcinoma has best prognosis as no lymphatic metastasis
Reinke's space and Reinke's edema

Reinke's space - Space between vocal ligament and mucosa

Reinke's edema
- Edema in Reinke's space
- Bilateral
- M.C in smokers - A/K/A Smokers larynx
- Management - In early cases - Stop smoking
  In late cases - Reduction Glottoscopy

Muscles of larynx - acting on inlet

- Epiglottis
- Hyoid (cut)
- Aryepiglottic fold
- Thyroid cartilage (cut)
- Posterior cartilage
- Vocal cord
- Interarytenoid notch
- Cricoid cartilage
- Tracheal rings
Muscles of larynx-acting on true vocal cord

Muscles acting on
True vocal cord (TVC)

- Adduct TVC
  - All the muscles except PCA
    - Thyroarytenoid
      - Runs along TVC-inner most part vocalis
    - Cricoarytenoid muscle
    - Interarytenoid
    - Oblique arytenoid
    - Runs oblique in between two arytenoids
    - Cricoarytenoid
  - Posterior crico arytenoid (PCA)
    - Cricothyroid
      - Only intrinsic muscle lying externally
    - Vocalis
      - Inner part of thyroarytenoid muscle

- Abduct TVC
- Adduction + tensions

Muscles acting on inlet

- To open inlet
  - Epiglottis moves towards thyroid cartilage
    - Thyroepiglottic muscle
- To close inlet
  - Epiglottis moves down towards arytenoid
    - Aryepiglottic muscle
### Difference between larynx in children and adults

<table>
<thead>
<tr>
<th>Infants</th>
<th>Adults</th>
</tr>
</thead>
<tbody>
<tr>
<td>Epiglottis is <strong>omega</strong> shaped</td>
<td>Epiglottis is <strong>leaf</strong> shaped</td>
</tr>
<tr>
<td>Larynx is funnel-narrowest Part is <strong>– subglottis</strong></td>
<td>Larynx is cylindrical narrowest is <strong>glottis</strong></td>
</tr>
<tr>
<td>Larynx is higher placed at C₂ level</td>
<td>Larynx is placed at Cᵥ, Cᵥ, Cᵥ, Cᵥ level</td>
</tr>
<tr>
<td>So that infant can Suckle and breath at the Same time</td>
<td></td>
</tr>
</tbody>
</table>

---

![Diagram of larynx in infants and adults]
INFECTIONS OF LARYNX

Clinical presentation – voice

<table>
<thead>
<tr>
<th>Supraglottis</th>
<th>Glottis</th>
<th>Subglottis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Voice - Normal</td>
<td>Hoarse / Dysphonia / Aphonia</td>
<td>Normal</td>
</tr>
</tbody>
</table>

Clinical presentation – Stridor

<table>
<thead>
<tr>
<th>Supraglottis</th>
<th>Glottis</th>
<th>Subglottis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stridor ↑ - inspiratory</td>
<td>Inspiratory</td>
<td>Biphasic</td>
</tr>
<tr>
<td>Stridor ↑ - in supine (+)</td>
<td>(-)</td>
<td>(-)</td>
</tr>
<tr>
<td>Stridor ↓ - in prone (+)</td>
<td>(-)</td>
<td>(-)</td>
</tr>
</tbody>
</table>

Stridor ↓ Obstruction in Larynx, trachea, until secondary bronchi
  
  Inspiratory
  
  • Supraglottis
  • Glottis

Stertor ↓ Obstruction in beyond secondary bronchi
  
  Inspiratory
  
  • Subglottic
  • Cervical trachea
  • Sometimes in glottis

Wheeze ↓ Oropharynx
  
  Expiratory
  
  • Intrathoracic
**Clinical presentation – dysphagia**

<table>
<thead>
<tr>
<th></th>
<th>Supraglottis</th>
<th>glottis</th>
<th>subglottis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dysphagia/odynophagia</td>
<td>(+)</td>
<td>(−)</td>
<td>(−)</td>
</tr>
<tr>
<td></td>
<td>Epiglottis</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>involved</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Drooling of saliva – in</td>
<td>(+)</td>
<td>(−)</td>
<td>(−)</td>
</tr>
<tr>
<td>children</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Examples:</td>
<td>Laryngomalacia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>infective</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Acute</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>epiglottitis</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Group</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(Laryngotracheo-</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>bronchitis)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(−)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(−)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Acute epiglottitis**

- **Cause**
  - m. C
  - Group-A beta hemolytic streptococci

- **C/F**
  - In child
  - Acute onset fever
  - Inspiratory stridor (↑ in supine ↓ in prone)
  - Tripod position sits bending

- **Diagnosis**
  - Fibre optic laryngoscopy
  - X-ray (Lateral view)

- **Management**
  - Antibiotics
  - Steroids
  - Adrenaline
  - Nebulisation
  - Humidification

- **Thumb sign**

**Radiographs**

- Normal X-ray
- Thumb sign – Acute epiglottitis
Croup – laryngotraceobronchitis

- **Cause**
  - m.C
  - Parainfluenza virus

- **C/F**
  - Fever
  - Hoarseness
  - Stridor ➔
    - Biphasic
    - Seal like / Barking cough

- **Diagnosis**
  - X-ray (AP view)
    - Steeple sign / Pencil tip sign

- **Management**
  - Steroids
  - Adrenaline
  - Nebulisation
  - Humidification

---

**Tuberculosis of larynx**

- TB of larynx - rare condition - following TB of lung
- All granulomatous conditions affect anterior part of larynx
  - Except TB - affects posterior part of larynx

**TB larynx findings**
1. Mammillated arytenoids - 1st affected
2. Mouse nibbled vocal cord
3. Turban epiglottis
4. Very painful condition

**Management** - Anti Tubercular Therapy (ATT)
CONGENITAL CONDITIONS OF LARYNX
AND SPEECH DISORDERS

mc congenital anomaly of larynx: Laryngomalacia.
  2nd mc: Vocal cord palsy
  3rd mc: Sub-glottic stenosis

Sub-glottic stenosis

Larynx in children:
  • Narrowest part: Sub-glottis
  • Funnel shaped

Causes of sub-glottic stenosis:
  a) Congenital
  b) Prolonged intubation (premature infants, ARDS)

Symptoms:
  - Biphasic stridor
  - No associated fever, inflammatory symptoms (differentiate from croup)

Stenosis present if:
  Diameter of subglottis: ≤ 4mm in normal infant
  ≤ 3mm in premature infant

meyer-cotton classification

4 grades

Grade I  Grade II  Grade III  Grade IV
0-50% 51-70% 71-99% no detectable stenosed stenosed lumen
manage:
1. Grade I and early Grade II
   - Asymptomatic
     - Conservative management
   - Symptomatic
     - Soft stenosis
       - Balloon dilatation
     - Hard/Fibrosed
       - Cricoid split + graft
         (costal cartilage used)
2. Grade III
   - Anterior split + Graft
   - Anterior and posterior split + graft
3. Grade IV
   - Cricotracheal resection of stenosed part
   - Mitomycin-C: Prevent re-stenosis and synechiae formation
     Also used in:
     - FESS (nasal packs)
     - Choanal atresia
     - Laryngo-tracheal stenosis

Laryngeal web

- Incomplete canalization of larynx at level of glottis, between true vocal cords

Presentation:
- Weak cry (web limited to anterior commissure)
- Stridor (inspiratory)

Management:
- Excision of web + Sialistic Keel (to keep edges apart)
Speech disorders - dysphonia plica ventricularis

- Altered voice: Voice produced by false vocal cords/vestibular or ventricular folds
- Hoarse, husky voice
- Management: Treat the cause

Functional/ hysterical aphonia

- Females > males
- Presentation: Talks like whispering
- On examination:
  - Vocal cord, abducted
  - Do not adduct on phonation
  - Adduct on coughing
- Management → Psychotherapy

Puberophonia / mutational falsetto

- At puberty → vocal cord lengthens → low pitched voice of adult male
- Adult male with high-pitched voice → Puberophonia.
- Evaluation: Gutzmann pressure test
  - Pressure on thyroid → Low pitched voice
  - Management
    - Type III thyroplasty
    - Shortening of vocal cords (relaxation)
    - By compression of laryngeal framework (Thyroid cartilage)
Spasmodic dysphonia (SD)

- Systemic disorder (other dystonias may be present)
- Dystonia of larynx +

Adductor SD
- Strangled, scratchy
- Croaky voice
  - Management:
    - Botulinum toxin (BOTOX)
    - In thyroarytenoid muscle

Abductor SD
- Breathy (whisper) voice
  - BOTOX in posterior cricoarytenoid
**VOCAL CORD PALSY**

**Nerve supply of larynx**

- **Vagus nerve**
  - **Superior Laryngeal Nerve (SLN)**
    - At Hyoid
      - Internal laryngeal Nerve
        - Pierces Thyrohyoid membrane
        - Supplies cricothyroid muscle
          - Sensory Supply to:
            - Supraglottis
            - Hypopharynx
      - Recurrent Laryngeal Nerve (RLN)
        - Right
          - Takes a turn at
            - Right Subclavian Artery
        - Left
          - Takes a turn at
            - Arch of Aorta
            - Sensory supply to glottis and sub glottis
            - Motor supply to all laryngeal muscles except cricothyroid

- To take biopsy from supra glottis; Superior laryngeal nerve is anesthetised at:
  1. Thyrohyoid membrane
  2. Pyriform fossa

- More prone to injury: **Left RLN (long Course)**

- Etiology of RLN Palsy:
  1. Neck conditions: Involves both right and left RLN
  2. Mediastinal Conditions: Involves only **Left RLN**
    - Lymphadenopathy
    - Bronchogenic carcinoma
    - Ortner’s syndrome
      - Cardio vagal syndrome
→ MC Cause of left RLN Palsy: Bronchogenic carcinoma.
→ MC Cause of Bilateral / Right RLN Palsy: Thyroid Surgery

Position of vocal card

1. Median (midline)
2. Paramedian (0.5 mm from midline) → whispering
3. Intermediate/cadaveric (3.5 mm from midline)
   → Neither abducted nor adducted
   → Seen in complete palsy of nerves
4. Slight Abduction (1 mm from midline) → Normal Respiration
5. Full Abduction (9.5 mm from midline) → Forced Respiration

Vocal Cord Palsy

- Complete (SLN + RLN) (Adductor Palsy)
- Incomplete (only RLN) (Abductor Palsy)

- Cadaveric position
- Median / Paramedian position

- Wagner’s and Grossman theory → RLN Palsy
- Semen’s theory → Abductor muscles (Phylogenetically newer)

Management of vocal cord palsy

<table>
<thead>
<tr>
<th>Function of Larynx</th>
<th>Unilateral RLN Palsy</th>
<th>Bilateral RLN Palsy</th>
<th>Unilateral complete palsy</th>
<th>Bilateral complete palsy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Speech</td>
<td>Normal/mild Hoarseness</td>
<td>Normal</td>
<td>Aphon. Dysthonia</td>
<td>Aphonia</td>
</tr>
<tr>
<td>Respiration</td>
<td>Normal</td>
<td>Stridor</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>Respiration</td>
<td>-</td>
<td>-</td>
<td>+ (occasional)</td>
<td>Chronic</td>
</tr>
<tr>
<td>Management</td>
<td>Conservative</td>
<td>Tracheostomy</td>
<td>Medialisation of vocal cord</td>
<td>Tracheostomy + Epiglottoplasty/ vocal cord plication</td>
</tr>
</tbody>
</table>
<pre><code> |                      | Followed by vocal cord Laterisation |                      |                          |
</code></pre>
Superior laryngeal nerve palsy

SLN Palsy

\[
\begin{align*}
\text{Internal Laryngeal} & \quad \text{External Laryngeal} \\
\text{Nerve Palsy} & \quad \text{nerve palsy} \\
\downarrow & \quad \downarrow \\
\text{Absence of:} & \quad \text{Absence of:} \\
\text{Sensation above} & \quad \text{Function of cricothyroid} \\
\text{true vocal cord} & \quad \downarrow \\
\downarrow & \quad \text{Inability to \textbf{↑} pitch} \\
\text{Occasional} & \quad \text{of the voice} \\
\text{aspiration} & \\
\end{align*}
\]

- Indirect laryngoscopy examination: \textbf{Skewed vocal cord}
- Management: Speech Therapy

Medialisation and laterlisation of cord

Medialisation of cord
\[
\rightarrow \text{Injection of:} \\
\text{Teflon} \\
\text{Collagen} \\
\text{Fat}
\]

Laterisation of Cord:
\[
\rightarrow \text{Cordectomy + Arytenoidectomy} \\
\rightarrow \text{Kashima's operation} \\
\rightarrow \text{Woodman's operation}
\]

Thyroplasty

- Thyroid frame work surgery (nothing to do with vocal cord)
- AR/KA Phonosurgery
- Thyroplasty Types:
  \[
  \rightarrow \text{Mnemonic: Plasty}
  \]
→ Types

I : P → Proximalisation (medialisation)
II : L → Lateralisation (B/L RLN Palsy)
III : S → Shortening (Done for Auberphonia)
IV : T → Tightening (Lengthening)

↓

Androphonia.

Type I

Type II

Active space
CARCINOMA OF LARYNX

Juvenile laryngeal papilloma

- mc benign tumour of larynx in children
- HPV-6, 11 (11 → more virulent)
- Transmitted through mother
- Presentation:
  - Hoarseness of voice
  - Stridor

- Management
  - Microdebrider (main management)
  - CO₂ Laser
  - KTP Laser
  - In post-operative period:
    - A → α Interferon
    - B → Bevacizumab
    - C → Cidofovir

Carcinoma of larynx

- mc → Squamous cell carcinoma.
- mc site → Glottis
- Best prognosis → Glottis (least lymphatic metastasis)
- Earliest presentation → Glottic carcinoma (hoarseness)
- Maximum lymphatic metastasis → Supraglottic

Tumour staging:

T₁ →
- Localised to one subsite
  - Glottic:
    - T₁,a → Only one vocal cord
    - T₁,b → Both vocal cords
\( T_a \rightarrow 
- \text{spread to adjacent subsite}
- \text{vocal cord mobility may be impaired}

\( T_s \rightarrow 
- \text{Fixed vocal cord}
- \text{Involvement of: Pre epiglottic space}
  - \text{Para epiglottic space}
  - \text{Post cricoid}
  - \text{Inner cortex of thyroid}

\( T_{sa} \rightarrow 
- \text{Intrude Thyroid cartilage}
- \text{Involvement of strap muscles}
  - \text{Hypopharynx}
  - \text{Trachea}
  - \text{Base of tongue}

\( T_{sb} \rightarrow 
- \text{Prevertebral space}
- \text{Carotid sheath}
- \text{Base of skull}
- \text{mediastinum}

\text{Post laryngectomy speech}

\text{1. Esophageal speech}
\rightarrow \text{Patient swallows air and use it for speech}
\rightarrow \text{many patients fail to achieve esophageal speech}

\text{a. Tracheoesophageal speech}
\rightarrow \text{Best}
\rightarrow \text{A/V/A voice prosthesis (no prosthesis used)}
\rightarrow \text{A valve is placed, which on closure during expiration leads to air in hypopharynx used for speech}
\rightarrow \text{Blom singer valve}
3. Electrolarynx
   → Vibrates tracheo-oesophageal segment which can be used for speech

→ The quality of life after total laryngectomy is very poor
   ↓ Conservative management is preferred
Management of carcinoma of larynx

- $T_1$, $T_2$ → Radiotherapy, TLM (Transoral Laser Microsurgery)
- $T_1$, $T_2$ Glottis midcord lesion → TLM
- $T_3$ → Concurrent chemoradiation
- $T_4$, $a$ → Total laryngectomy + radiotherapy
- $T_4$, $b$ → Palliative management

Neck node management
- Neck Dissection is done (surgery)
- Types:
  - **Radical**
    - Remove lymph node groups I-V
    - Spinal accessory nerve
    - Int. jugular vein
    - Sterno cleido mastoid muscle
  - **Modified Radical**
    - Remove lymph node groups I-V
    - Types:
      - I → Preserve nerve
      - II → Preserve nerve + IJV
      - III → Preserve all 3 non-lymphatic structures
  - **Selective**
    - Only in $N_0$, neck
    - Only lymph node groups removed
  - Only in $N_0$, neck
  - Oral: I, II, III
  - Larynx: II, III, IV

Recurrence Following Radiotherapy:
- $T_1$, $T_2$ → Conservation surgery (Partial laryngectomy)
  - Horizontal
  - Vertical
  - Better

- $T_4$, $T_5$ → Total laryngectomy
TRACHEOSTOMY

Indications of tracheostomy

• **MNEMONIC: Occupy Most Seats In Medical Assocation**
  1. Obstruction above tracheal rings 2, 3, 4
     - Infections, tumors, trauma, vocal cord palsy
  2. Prolonged mechanical ventilation
     - Cervical spine injury, Phrenic nerve injury
     - COPD with respiratory insufficiency
     - ARDS
     - Guillain Barre syndrome
  3. Suctioning of secretions
  4. Maxillofacial Surgeries
  5. Prevent Aspiration
     - Bilateral Complete palsy

Procedure

• Positioning: Rose’s Position
  - Same position as adenoidectomy, tonsillectomy
  **MNEMONIC** - A ton of Roses in a tray
  - Extension at cervical and atlanto-occipital joint
  - Prevent aspiration

  elective tracheostomy
  
  emergency tracheostomy

  Skin crease incision
  2.5 cm above
  suprasternal notch

  vertical incision
  from below cricoid to
  suprasternal notch

• Retraction of strap muscles
* Opening made at tracheal rings T₁, T₂, T₃.
  - In high tracheostomy, opening is made on T₁.
  - Only indicated in laryngeal carcinoma.
  - Not usually done as it may cause laryngeal stenosis.
  - Before incision, air bubbles are aspirated with syringe to confirm location.
  - A horizontal or vertical incision is made over trachea.

* Insertion of tube
  - Jackson's metallic tube
    - Consists of an outer and inner tube.
    - Inner tube can be removed & cleaned at home.
    - Not used anymore, because it causes pressure necrosis of mucosa.
  - Portex tube
    - Initially cuffed tube is used - provides high volume low pressure air tight seal.
    - Prevents aspiration of blood.
    - Does not cause pressure necrosis.
    - After 72 hrs, changed to non-cuffed tube.

* Removal of tube
  - Tracheostomy reduces dead space by 30 - 50%.
  - Patient gets used to decreased respiratory effort.
  - Test to confirm if patient can breathe normally:
    * Fenestra (opening) made at the bend of the tube.
    * External opening is temporarilu closed.
    * If patient is able to breathe through fenestrated tube, for 24 hours, then tube is removed.
Complications

- Immediate:
  - H → Hemorrhage
  - A → Aspiration
  - A → Apnoea, due to sudden washout of CO₂ treated with carbogen [95% O₂ + 5% CO₂] (for 5-10 minutes)
  - P → Pneumothorax, in infant’s lung apices may extend to neck and be prone to injury to lung
  - I → Injury to adjacent structures like inferior thyroid vein

- Intermediate:
  - Tube obstruction / Displacement
    - If obstructed, immediately removed
    - Prevented by suctioning with saline or NaHCO₃ for 5-10 seconds every 1-2 hours

- Late:
  - → Tracheoesophageal fistula.
  - → Tracheal stenosis
  - → Difficult decannulation (removal of tube)

Boyce position & Indirect laryngoscopy

- For direct laryngoscopy, oesophagoscopy, bronchoscopy
  - Aka barking dog or sniffing the morning air position
  - Flexion at cervical spine and extension at atlanto-occipital joint
  - Brings the laryngeal axis in a straight line
Indirect laryngoscopy

- Done to visualize structures of larynx
- Straight handle
- Warmed before inserting to prevent fogging
- Mirror image of larynx is seen