ENT

EAR

Ear consists of 3 parts

→ External ear
→ Middle ear
→ Inner ear

Embryology of Ear

External Ear

→ Pinna is formed from 6 hillocks of his (1and 2nd Arch)
→ External Auditory Canal: develops from 1st Arch

Middle ear

→ Tympanic membrane develops from all 3 germ layers
→ Middle ear cavity and Eustachian Tube develops from 1st Pouch. (Endodermal in origin)
Ossicles: It reaches **adult** size by 15 weeks, and adult configuration by 20 weeks.
→ Malleus and Incus develop from **1st Arch (Meckel's cartilage)**
→ Stapes supra structure develops form **2nd Arch (Reichert's Cartilage)**
Stapes footplate (Medial surface) develops from **otic capsule**.

→ Inner ear has 2 parts:
1. **Bony labyrinth** which develops Otic capsule (secondary mesoderm)
2. **Membranous labyrinth** which develops from the otic vesicle (Surface ectoderm)

Osteology of Temporal Bone

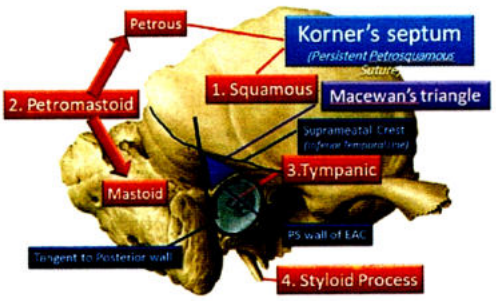
Parts of Temporal Bone

1. Squamous → Covers temporal lobe of brain
2. Petro mastoid → After Birth differentiate into **Petrous and mastoid parts**
Petrous part which is on medial surface contains Inner ear
Mastoid part consist of Mastoid air cells

→ Largest Mastoid cell is K/as mastoid Antrum
→ Mastoid antrum is of adult size at the time of birth
→ Mastoid process start developing 6 months after birth
→ Mastoid Tip is formed by 2 year of age
→ Mastoid reaches adult size by 18-19 years.

3. Tympanic Plate – forms EAC and middle ear.
4. Styloid process

Temporal Bone



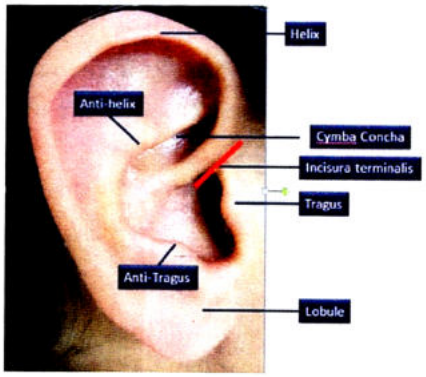
→ Petrous part and the squamous part in embryonic period has a suture in between than k/as Petrous squamous suture. This suture disappears, after birth if it does not disappear it forms a Korner's septum

→ Above EAC there is bony elevation k/as **Supra meatal crest** (a/k/as Inferior Temporal line)
→ Supra metal crest, Postero-superior wall of EAC, tangent to posterior wall of EAC and in between there is a Triangle k/as Macewen's Triangle (surgical landmark for mastoid antrum)

Anatomy of Ear

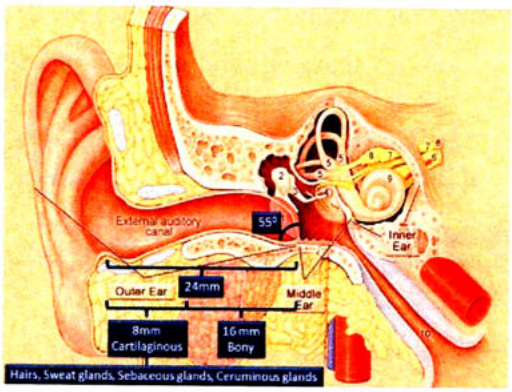
External ear

Pinna



- → **Tragus**: Embryologically develops from 1st Arch
- → Lobule consist of only Fat, no cartilage in the lobule
- → Cymba concha is the anatomical landmark of mastoid antrum (Surface landmark)
- → Incisura terminalis: Place of Fusion of 1st and 2nd Arch
- → If Incision is given at Incisura terminals we reach bone without cutting cartilage This incision is k/as Lampert's Incision (endaural approach)

External Auditory canal



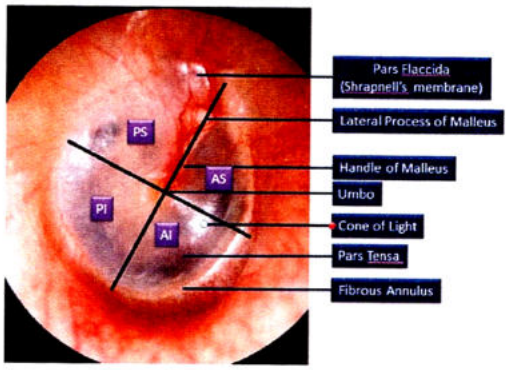
24 mm in length lateral 1/3rd is cartilaginous(8mm) & inner 2/3rd (16 mm) is Bony.

Cartilaginous Part has hairs, Sweat Gland, sebaceous glands and ceruminous glands

Ceruminous glands are modified apocrine sweat glands.

- → Medial end of EAC has tympanic membrane which form an angle of 55 with the floor of EAC.
- → Antero-inferior wall is longest part of EAC and postero superior wall is shortest wall of EAC.

Tympanic Membrane



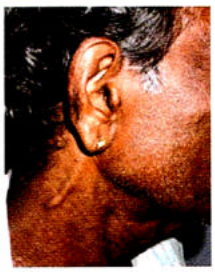
- → Trimeric structure
  - upper part is pars flaccida is Shraphell's membrane
  This part has minimum fibrous layers
  Lower part is pars tensa – It has all the Germ Layers.
- → Fibrous annulus is formed by fibrous layer of Pars tensa.
- → Umbo: It is most visible anatomical landmark of tympanic membrane
- → It Divides Tympanic membrane into 4 quadrants antero superior; Antero inferior; Postero superior and Postero inferior
  Cone of light is seen in antero inferior quadrant of Pars tensa
- → Cone of light is present due to handle of Malleous, Antero inferior part becomes Perpendicular therefore cone of light is visible
  Most reliable landmark of Tympanic Membrane – Lateral Process of malleus

Nerve Supply of Pinna

- → Auriculo temporal Nerve – Branch of Mandib ular Nerve
- → CN VII, X Nerve (C2, C3) – Supplies choncha
- → Greater auricular Nerve (C_2 $C)_3$is a Spinal Nerve
- → Supplies majority of EAR in medial as well as lateral part of ear.
- → Split lobule is seen due to ear Piercing **Rx by Lobuloplasty** (Greater Auricular Nerve Block is used)

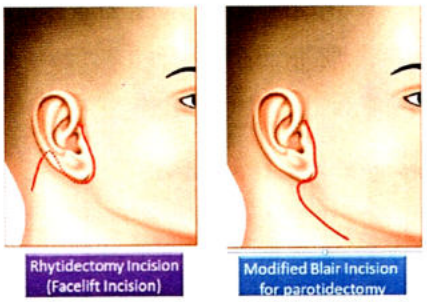
Greater auricular Nerve is the most common Nerve to get injured during rhytidectomy incision (Face lift incision)

→ It can also be injured during **Parotidectomy** (modified blare incision) and patient a will have anesthesia in shaving area, lobule, Pinna.



ThickenedGreater Auricular Nerve in Leprosy

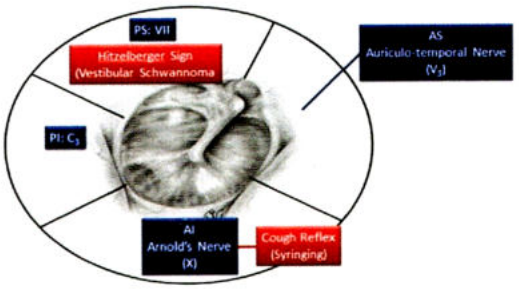
→ Parotidectomy can also lead to injury to Auriculo – Temporal Nerve which can cause **Frey's syndrome**



Nerve Supply of EAC

→ Anterio superior quadrant is supplied by Auriculo Temporal Nerve (Branch of mandibular Nerve)
→ Anterio Inferior Quadrant supplied by Arnold's Nerve – Branch of Vagus. Cough reflex during syringing is due to arnold's Nerve
→ Posterio inferior is supplied by c_3
→ Postero – superior is supplied by Facial Nerve in this region

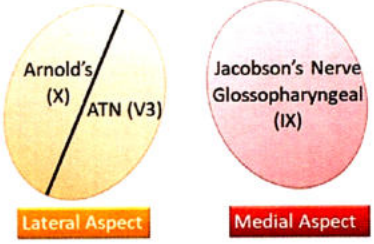
Nerve Supply of EAC



Absence of sensory supply facial Nerve is k/as hitzelberger sign
It is seen in **vestibular schwannoma**

Nerve supply of Tympanic Membrane
Lateral Aspect

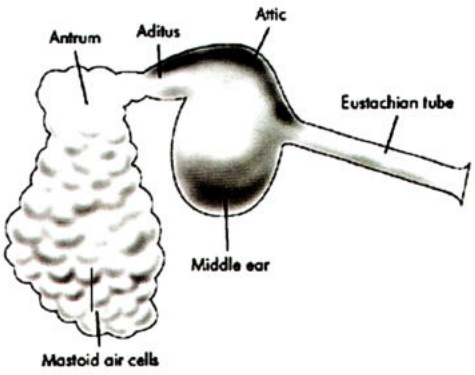
Anterior Half: Aurico–Temporal Nerve
Posterior Half: Arnold's Nerve
Medial Aspect: Jacobson's Nerve (Brach of IX Nerve)
Jacobson nerve is Sensory supply to middle ear.



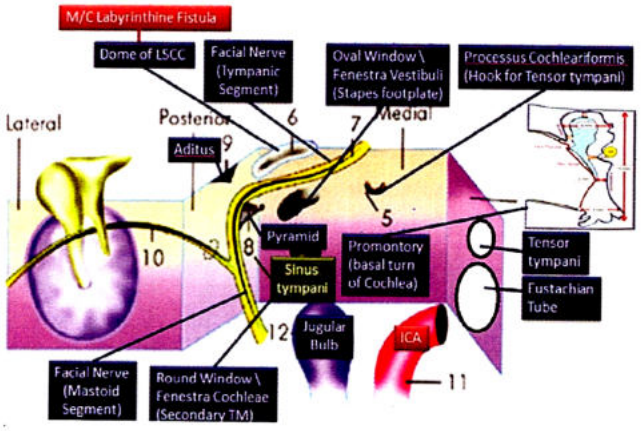
Middle ear

Middle ear cleft: Volume 6 ml
Formed by 3 Parts
Middle ear + Eustachian Tube + Mastoid

Volume: 5 ml (mastoid) + 1 ml (middle ear) = 6 ml



Middle ear



Lateral wall is formed by: Tympanic Membrane + Bony lateral wall of actic Scutum is present

→ Superior wall is k/as Tegmen

Superior Buldge on superior wall is arcuate eminence (due to superior semi-circular canal.)

Anterior wall has 2 openings

→ Tensor Tympani

→ Eustachian Tube

Medial wall

→ Oval window/ Fenestra Vestibule covered by Stapes foot plate.

Round window/ Fenestra conchae covered by secondary tympanic membrane

→ Anterior to oval window there is hook for tensor tympani – k/as processus cochleariformis
Tensor Tympani is supplied by mandibular Nerve
Dome of Semi circular Canal → M/c site for labyrinthine fistula

→ Horizontal / tympanic segment of facial Nerve is covered in a bony canal c/as fallopian canal

→ **Promontory**: Due to Basal (1st) turn of cochlea.

→ Meso tympanum (2 mm) is narrowest part of middle ear

Posterior wall

i. Auditus connects middle ear to mastoid

ii. Vertical segment of facial Nerve

Pyramid: Stapedius is smallest named muscle arizes from Pyramid inserts on stapes and is supplied by 7th nerve

→ Area medial to facial Nerve is k/as **sinus Tympani** (M.C site for residual cholesteatoma)

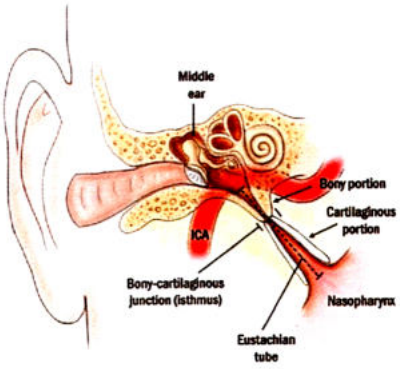
→ Area lateral to Facial nerve and above cauda facial angel → k/as facial recess (This is area of Posterior tympanostomy)

Floor / inferior wall

Jugular bulb and internal carotid Artery are present below the floorIn between them there is bony crest k/as Carotico Jugular Crest

Phelp sign: Inability to distinguish b/w juglar bulb & carotid artery due to erosion of cortico-Jugular crest seen in **CECT of tumor of** Jugular bulb (glomus tumor).

→



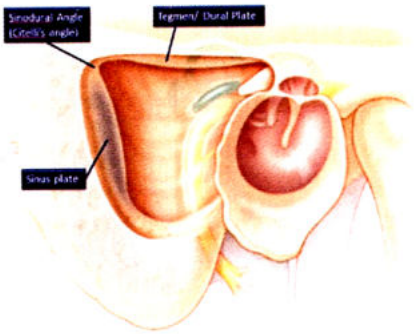
Eustachian Tube (a/k/as Pharyngo-Tympanic Tube /Auditory tube)

→ Connect the anterior wall of middle ear to lateral wall of Nasopharynx
36 mm in length
Lateral 1/3rd is bony
Medial 2/3rd is cartilaginous
Isthamus: Narrowest part of eustachian tube
Eustachian Tube make angle of 45 to the horizontal line

In case of Neonates Eustachian tube is almost flat and ½ the length (16-18 mm)
Therefore Middle ear infection is most common in the children as it can travel from Nasopharynx to middle ear .

Function of Eustachian Tube
i. It maintain the middle ear air pressure equal to outmost pressure
ii. It drains the secretions of middle ear
Eustachian tube is normally in a close position and that is because of ostmann of fat

It opens due to action of Tensor Veli palatini
(Main dilator of Eustachian tube)

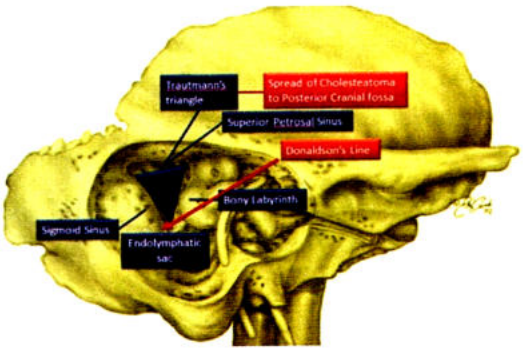


Mastoid Anatomy

3 walls

Ant wall: is same as post wall of middle ear
Superior wall of mastoid k/as Tegmen / Dural plate
Posterior wall is k/a sinus plate as it cover sigmoid sinus
Angle between tegmen and sinus plate is k/as sinodural angel a/k/a citelli angle.

Medial Wall Of Mastoid



Trautmann's Triangle is seen:

Boundaries:

→ Posterior boundary – Sigmoid sinus
Anterior Boundary – Bony labyrinth
→ Superior Boundary – Superior petrosal sinus

Significance:

This is place where cholesteatoma erodes the Bone and goes into the Posterior cranial fossa.
(Bony labyrinth is formed from otic capsule it is hardest bone in body)

→ **Donaldson's line** Line drawn along semi circular canal – It bisects the Posterior canal
→ It is landmark for endolymphatic sac.

Middle ear ossicles

Type of joint between ossicles: synovial joint b/w Incus &

b/w malleus and Incus → Saddle Joint stapes

b/w malleus and Stapes → Ball & socket joint

→ First/ M.C common part to undergo erosion – lenticular Process of Incus.

2nd m/c → Long Process of incus

Last to undergo erosion is foot plate of stapes.

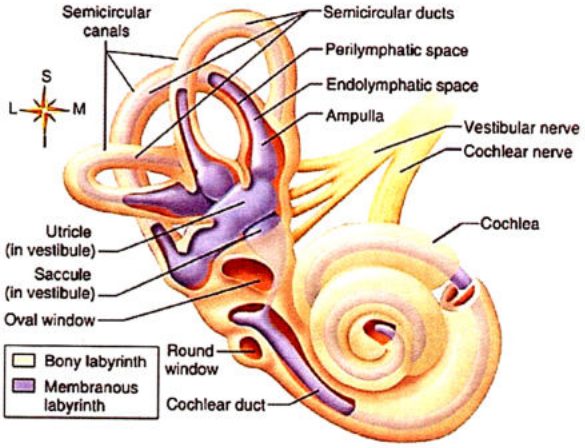
Anatomy of Inner ear

Inner ear

1. **Bony labyrinth** formed from otic capsule.
2. **Membranous labyrinth** formed from optic vesicle.

Between Bony labyrinth and Membranous labyrinth there is space filled with Perilymph → K/as Peri lymphatic space.

Inside Membranous labyrinth → Endolymph fluid is present – k/as Endolymphatic space

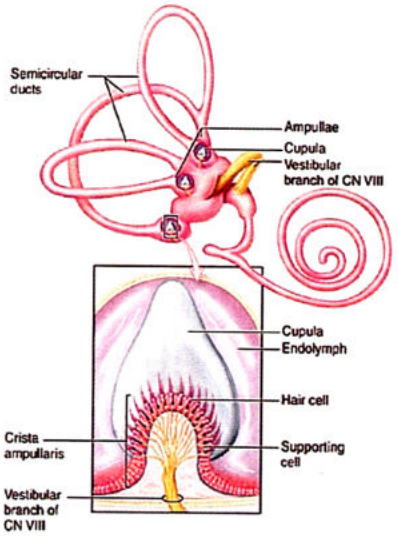


It can also be divided on basis of function

→ Auditory (Hearing)

Cochlea consists of 2.75 turns around a central Bony axis k/as **modulus.**

→ Vestibular (balancing)



→ 3 semicircular canals – Superior, Posterior and lateral each canal has opening c/as Ampulla Inside Ampule there are hair k/as crista ampullaris

→ Functions of cristae ampularis/ cupla ampullaris (semi circular canals) is to detect the **Angular Acceleration**

Function of utricle and saccule

→ Inside utricle and saccule, there is macula.This macula help in detecting the **linear acceleration**

→ **Utricle** help in detecting horizontal linear acceleration

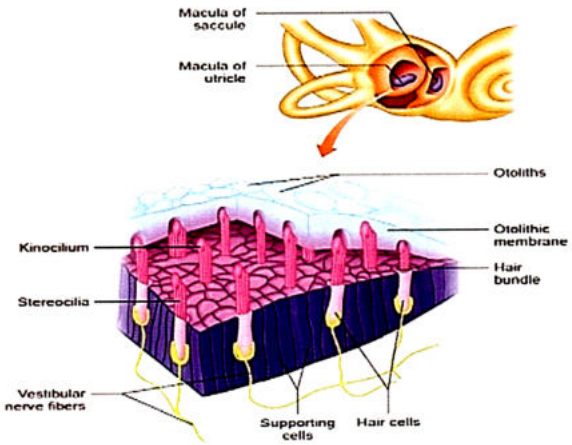
→ **Saccule** help in detection vertical linear acceleration.

Macula consist of calcium carbonate Crystal on it and forms a layers on hair cells k/as "otoconia"

Some otoconia goes in Posterior semi circular canal which leads to development of BPPV.

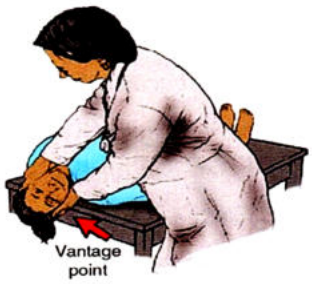
Benign Paroxysmal Positional vertigo (BPPV)

→ Positional vertigo
→ Last for few seconds to minutes
→ It is due to otoconia displaced to Posterior semi circular canal where they are k/as otolith – Condition is k/as otolithiasis



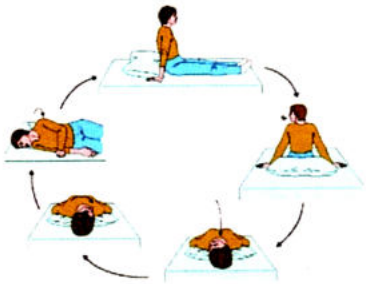
Diagnosis is made by Dix – hallpike maneuver

In this we move the patient in vertical direction check whether patient has nystagmus or not.



Dik hallpike maneuver

Treatment is by **Epley's Maneuver**
Epley's Maneuver consists of series of steps
First step is same as Dix. Hallpike's make patient go supine
Turn head other side
↓
Again roll the body once again by 90 C
↓
Take patient back to sitting position

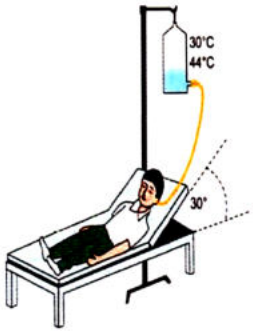


Epleys maneuver

Test for Vestibular functioning

i. **Caloric Test**

→ **Fitzgerald Hallpike Test:** Syringing is done with hot (44 C) and cold (30 C) wat (37 ± 7 C)
It produces nystagmus by stimulating lateral semi circular canal(horizontal nystagn



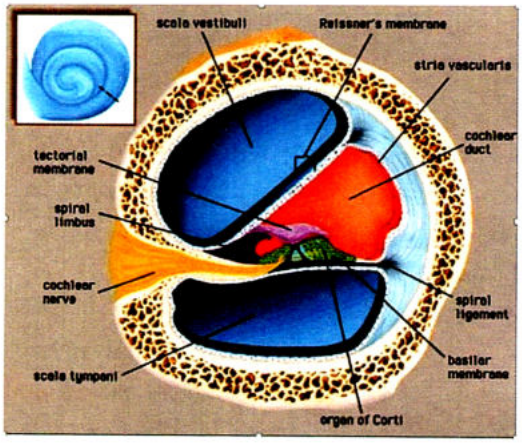
Syringing done with (COWS)

i. Cold water → Opposite side Nystagmus
ii. Hot water → Same side Nystagmus

2. **Modified Kobrak Test**

Instead of Hot and cold water at O c is used because cold stimulus is strongest stimulus than Warm Temperature.

3. **Dundas Grant Test**
   → Cold air is used when Syringing is C/I in perforated Tympanic membrane



Cochlea:

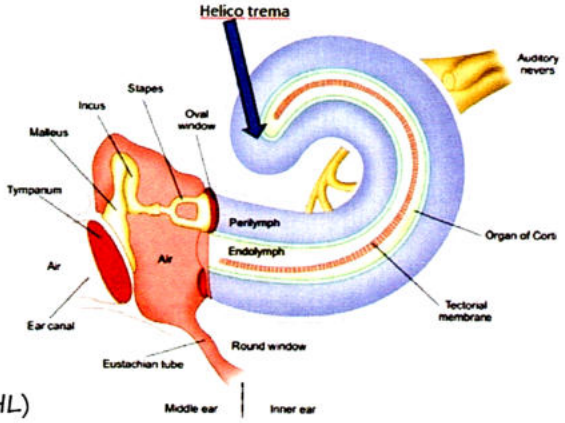
Divided into

a. scala vestibuli
b. Scala Media a/k/as cochlear duct
c. Scala Tympani

By 2 membrane
→ Reissner's Membrane
→ Basilar Membrane

Scala vestibule and scala tympani are filled with **perilymph**
→ In scala Media it is filled with **Endolymph**
Endolymph is secreted by stria vascularis with help of Na+ K+ ATPase pump the endolymph
→ In endolymph there is ↑ K and Less Na

Unspiralled Cochlea



Due to high K+ ions, endolymph as Positive potential inside it k/as **endocochlear potential.**
Scala vestibule and scala tympani communicates with each other at the apex and this communication is k/as **Helico-Trema.**

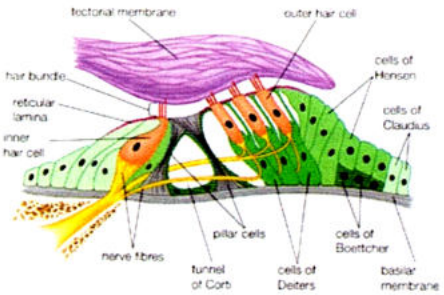
trauma (NIHL)

→ Scale tympani also communicates with the sub-arachnoid space through cochlear aqueduct so Perilymph is same as CSF/ECF.
→ Endolymph which is produced by stria vestibularis it is absorbed by the endolympatic sac.

Endolymph is same as Intra cellular fluid.

Organ of Corti

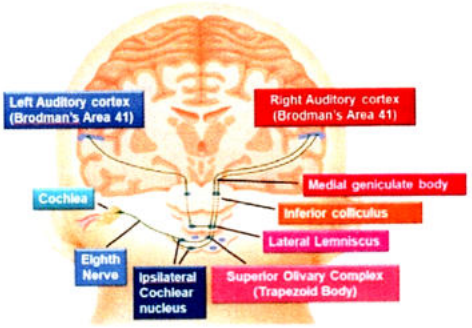
Inside the cochlea there is sensory organ resting on basilar membrane known as organ of Corti.
There are 2 hair cells k/as outer hair cells & Inner hair cells.



Outer Hair cells (mOre)	Inner Hair cells
More in number (13-14 k)	Less in Number (3500)
More in Rows (3-5)	Less in rows (single)
Late development (more time)	Early to develop
More sensitive to ototoxic Drugs, Acoustic trauma (NIHI)	Less sensitive
It produces OAE (OHO-Acoustic emission)	

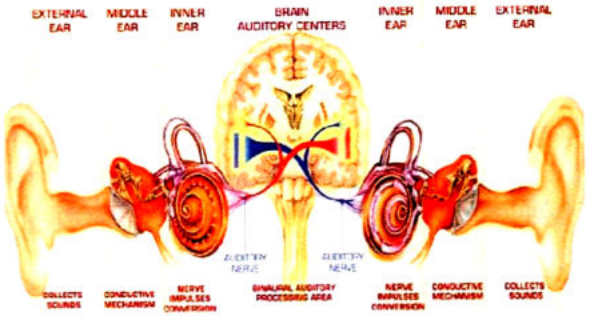
Neuronal Pathway of Sound

E → Eight Nerve } Nerve
C → Cochlear Nerve (Spiral Ganglion)
O → Superior olivary complex (opposite side) } Brain stem
L → Lateral lemniscus (Largest)
I → Inferior colliculus
M → Medial Geniculate body } Cerebrum
A → Auditory cortex (Brodmann area 41)



→ One cochlea stimulates both the auditory cortex maximum to opposite side but some goes to same side.

Binaural hearing: sound localization (Head shadow effect)



BINAURAL HEARING

Middle ear Mechanics:

It is concerned with impedence matching, transferring sound from external environment into the inner ear.

Total surface area of tympanic membrane =90mm2
Effective vibratory area of TM = **55 mm²**
Surface area of stapes foot plate = **3.2 mm2**
Area ratio: 17:1
Lever ratio: 1.3:1
(Handle of malleus: long process of Incus)
Middle ear transformer ratio: (22:1)
Total Amplification by Middle ear k/as **ossicular coupling**

→ Another Function of middle ear is to create a phase difference b/w 2 windows oval window and round window k/as **Acoustic coupling**
Ossicular coupling and Acoustic coupling together help the middle ear to transfer the sound into the inner ear → Impedence Matching

Inner ear Potentials

1. **Endocochlear Potential**
   DC potential
   Due to Na^+ - K ATP Pump in stria vascularis
2. **Cochlear Microphonics**
   AC potential
   Due to opening of cation channels.
3. **Summating potential**
   DC potential
   Inside hair cells
4. **Action Potential**
   8th Nerve Action potential and **All or None phenomenon** is seen .

Mechanical energy of sound it is converted into electrical energy of the nerve impulse.

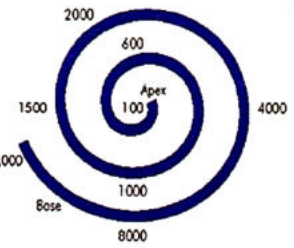
Frequency localization in Cochlea

Hearing range (frequency) 20 Hz – 20,000 Hz But each
Each part of cochlear has different frequency

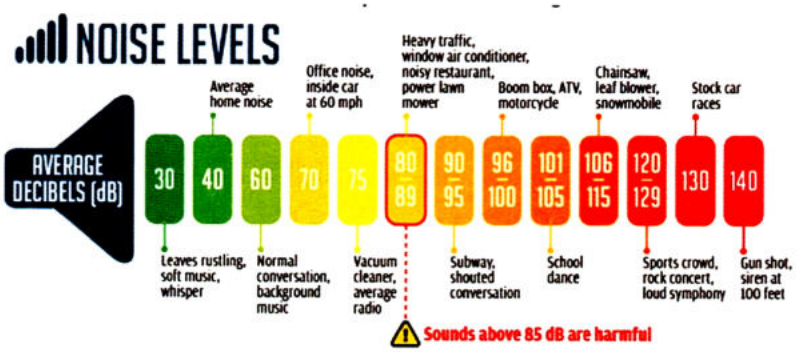
→ At base 20,000 it picked & at apex 20 Hz is picked.

→ It is explained by Travelling wave theory .
given by Von bekesy.

→ (Nobel Prize in physiology or Medicine in 1961 for this travelling wave Theory



Speech Intensity



Intensity of sound measured in Decibel.

→ Leaves, rustling soft music: 30 Db.

→ Normal conversation between & 45 – 60 Db.

→ Background music: 60 Db (market noise).

→ Sounds > **85 Db** becomes harmful.
85 Db x 8 hrs is maximum audible tolerance limit set for industry workers.
Industry workers work for **8 hours/day, 5 days/week** Otherwise it will cause Noise Induced hearing loss.

If noise is more than 85 db then worker should be provided noise Maskers or protectors

Any exposure > **140 Db** even for few milliseconds will cause NIHL

Test of Hearing

1. Tuning Folk Test:

→ The Most Commonly used tuning folk is **512 Hz.**

Rinne's Test:

Comparison of air conduction with Bone conduction of same side

AC > BC → **Rinne's Positive** Seen in Normal ear , **Sensineural Hearing loss.**

BC > AC → **Negative Rinne's** Seen in Conductive Hearing loss

False -ve Rinne's: U/L Severe to Profound sensineural hearing loss

3 tuning folks can be Used for conducting Hearing – 256 Hz; 512 Hz, 1024 Hz

256 Hz	512 Hz	1024Hz	CHL
•	⊕	⊕	20-30 dB
⊖	⊖	⊕	30-45 dB
⊖	⊖	⊖	> 45dB

M.C used tuning folk = 512 Hz
Most sensitive tuning folk = 256 Hz

Weber's Test : Most sensitivity Tuning folk test
It changes direction even with 5 Db change in sound levels.

→ **1st test to be checked in Question**
Normal = Centralized
It Goes Away in Sensorineural Hearing loss
It Goes Toward in Conductive Hearing loss
eg if Weber Test Sound lateralize to Right
Right CHL / Left SNHL

Absolute bone Conduction / Test
* This Test is Comparison of patient with examiner.
Press Tragus to close air conduction
(Examiner is Normal)
Patient BC = Examiner BC = **Normal**
Patient BC < Examiner BC: **SNHL**
Modification of ABC Test is **Schwabach's test**
In this test external auditory Canal is not closed so it is not absolute bone conduction
→ Air conduction plays a role in this. Schwabach's test can also have 2 similar results as of absolute bone conduction ,but there can be 3 finding also.

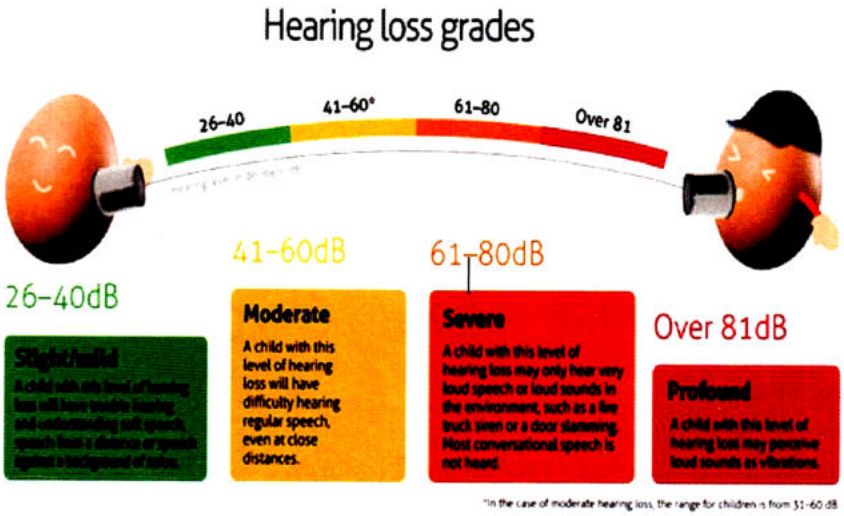
Patient BC > Examiner Bone conduction: the patient is having conducting Hearing loss.

Pure tone Audiometry (PTA)
It is used to find out
(i) Amount (degree of Hearing loss)
(ii) Type of Hearing loss
Normally, Both AC and BC Curve are near the Zero decibel line.
In conductive Hearing loss, BC is still normal (near zero line) but AC is reduced(air bone gap)
In Sensineural Hearing loss both air conduction and Bone conduction will be reduced.

Ranges of Hearing loss:

Clarks Classification
-10- +25 dB HL = Normal range
26-40 dB HL = Mild Hearing loss
41-55 dB HL = Moderate Hearing loss
56-70 dB HL = Moderately – Severe Hearing loss
71-90 dB HL = Severe Hearing loss
> 90 dB HL(91) → profound hearing loss

WHO Classification



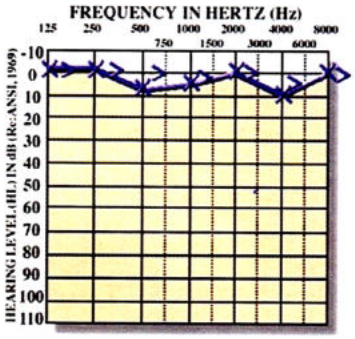
Hearing loss formula

Average Hearing loss : [500 Hz + 1000 Hz + 2000 Hz] ÷ 3

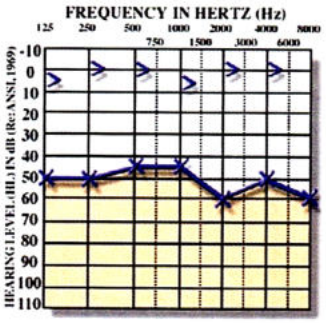
Percentage Hearing loss / Hearing Impairment → [Average Hearing loss - 25] x 1.5 = % of loss

Hearing disability = [(% better ear x 5) + (% worst ear)] ÷ 6 = % of disability

Hearing graphs

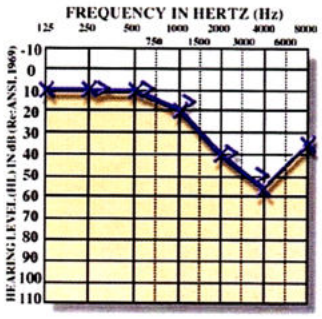


Normal Hearing



Conductive Hearing Loss

Air Bone Gap present

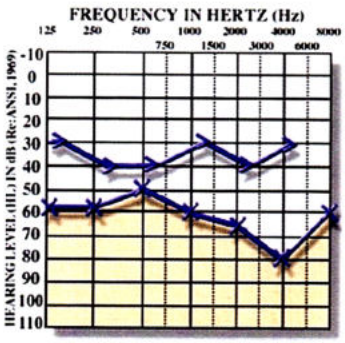


Sensorineural Hearing Loss

Dip @ 4000 Hz → **Boiler's Notch**

Seen in Noise induced Hearing loss

This Notch is seen in Bone Conduction



Mixed Hearing Loss

Both air conduction and Bone conduction has come down but there is also AB Gap.

Both components of CHL & SNHL

Impedence Audiometry

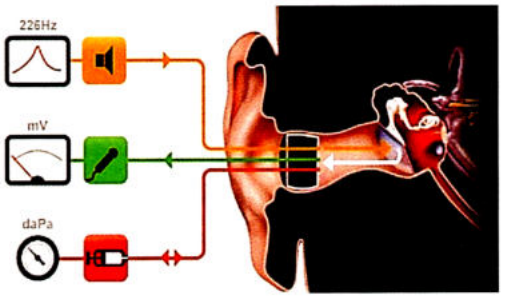
It consists of 2 test → **Tympanometry** and **Stapedial reflex** / Acoutic reflex Test

1. **Tympanometry**

Probe is Placed inside the ear

This Probe consists of 3 Things:-

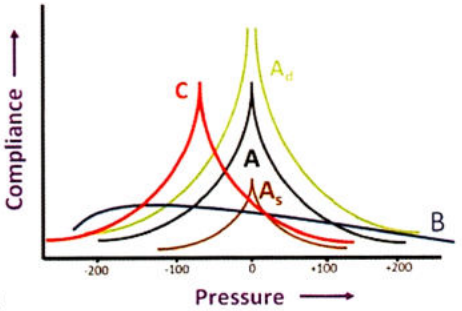
- **Sound ossilator** produces sound @ 226Hz
- **Microphone** – which can detect sound waves coming after reflecting for Tympanic Membrane.
- **Pressure probe** by which Pressure can be ↓ or ↑



Different curves are obtained

- Normal Curve is K/as **A Type curve** (maximum Compliance of Tympanic Memb. @ O Pressure)
- In case of otosclerosis stapes foot plate get fixed Compliance ↓ ; **As Type of curve is obtained**
- In case of ossicular discontinuity Tympanic Membrane is able to move freely

Ad Type of curve is seen(Compliance ↑)



- In case of fluid in middle ear tympanic Membrane is not able t Flat Curve is obtained **B Type**
- In case of retracted Tympanic Membrane, → Curve Peek is shifted towards Negative Pressure. **C type curve**

2. **Acoustic reflex A/K/a Stapedial reflex** ; middle ear reflex, Attenuation reflex or Auditory reflex
   → Spadial muscle Contracts in response to loud sound and stops loud sound going to ear and causing Noise induced Hearing loss. It is Protective reflex.

Mechanism:

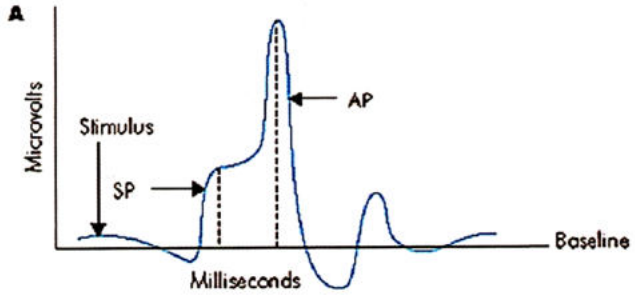
Loud Sound → middle ear → inner ear(cochlea) → VIII Nerve → Cochlear Nucleus → I/L and C/L Superior olivary complexes

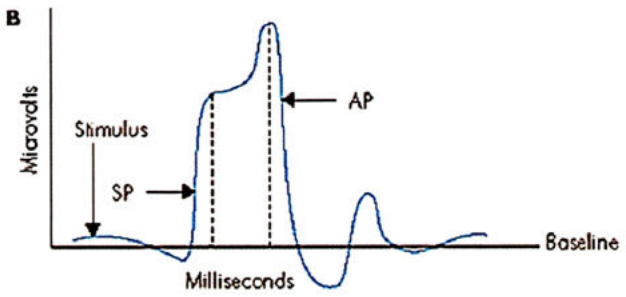
I/L Superior olivary complex → I/L facial Nerve Nucleus →middle ear (Stapedial muscle contracts) → I/L reflex.

C/L Superior olivary Nucleus → C/L facial Nerve Nucleus → C/L reflex.

Electro cocheography

Normally Summating Potential is less than 30 % of Action Potential SP/AP ratio is < 0.3





In case of Mennier's disease, the Summating Potential is > 70% of A.P

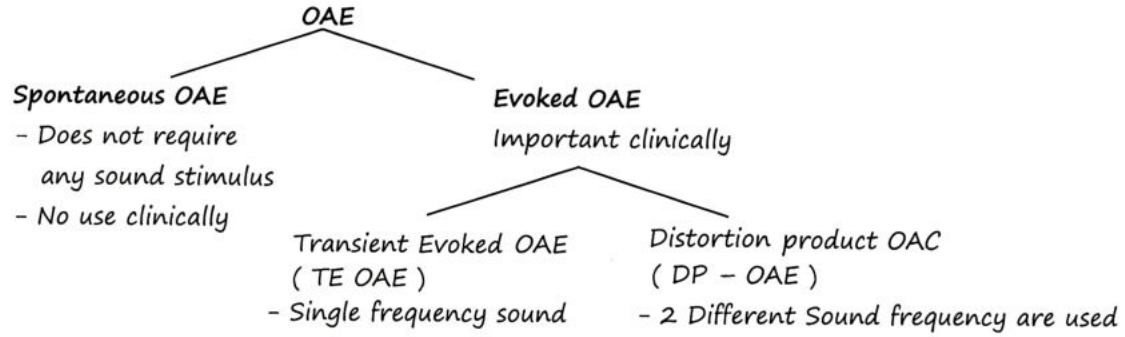
SP/AP ratio > 0.7

Most Specific Investigation for Meinner's disease

However, it is invasive Investigation, make a hole in tympanic Membrane & Mesaure Potential from round window.

OAE [Oto Acoustic emissions]

Low Intensity sound produced by Outer hair cells in response to sound stimulus.



Transient Evokes OAE is most sensitive test of outer Hair cells.

- OAE Directly coming from outer Hair cells so most sensitive test for detecting Noise Induced Hearing loss as well as ototoxicity
- **OAE are used as Screening test for Neonatal Deafness.**

BERA (Brain Stem Evoked Response Audiometry) / ABR (Auditory Brain stem response)

-**Wave I** → Produced by Distal Part of 8th Nerve

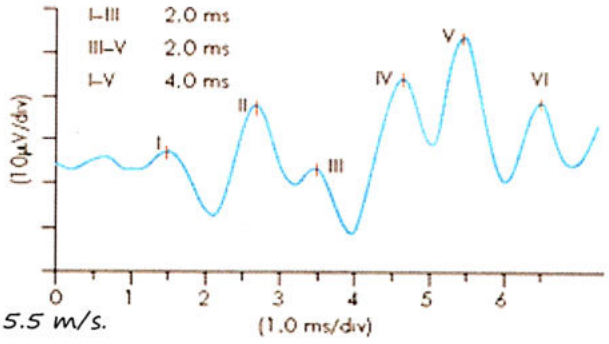
-**Wave II** → Produced by Proximal Part of 8th Nerve

-**Wave III** → Produced by Cochlear Nucleus

-**Wave IV** → Superior Olivary Complex

-**Wave V** → Lateral Lemniscus

-**Wave VI** → Inferior Colliculus



Wave V: Largest wave, Most stable Wave Produced at 5.5 m/s.

I, III, V are stable waves

Wave I @ 1.5 m/s

Difference b/w wave I & wave V = 4 m/s. If >, 4.4 then **Retrocochlear Hearing loss.**

Confirmatory test for Neonatal Deafness (Best)

-**Wave VI** → Inferior Colliculus

-**Wave VI** → Inferior Colliculus

Age wise Hearing Assessments Subjective test

Subjective test from Birth – 6 Months = Behavioral Observational Audiometry

6M – 3yrs – Visual reinforcements orientation Audiometry

3yrs- 9 yrs – Play Audiometry

Later - Pure tone Audiometry

Objective test:

- Tympanometry
- Acoustic reflex
- Electrocochleography
- OAE
- BERA / ABR
- BERA is also the best test for malingering

Cochlear vs Retero Cochlear Hearing loss

Test	Cochlear	Retro- Cochlear
S.I.S.I	Positive (>70%)	Negative
A.B.L.B Laddergram	Converging	Diverging
Tone Decay	Negative(<25 dB)	Positive (>25dB)
Speech Audiometry	SDS = 60-80%	<40%, Roll over phenomenon
B.E.R.A (Wave V Latency)	</= 4.2 msec	>4.2 msec

S.I.SI. – Short Increment Sensitivity Index
A.B.L.B. – Alternal Binaural Loudness Balace test.
Both these tests are test of **cochlear hearing loss.**
They are based on Recruitment phenomenon

Tone Decay, Speech Audiometry, BERA are Reterocochlear Hearing loss

Facial Nerve

3 Nuclei :-

a. Motor Nucleus (Pons)
b. Nuclear Tractus Solitarius: Touch
c. Nucleus Salivatories Superior is: Secretomotor
   nerve fibers from Nucleus Tractus Solitaries and Salivatorius Superioris forms Nerve of Wrisberg

(Nerves Intermedius)

4 Segments:

a. Intracranial
b. Intra Meatal

c. Intra temporal – Fallopian canal (27 mm) Longest Canal for any cranial Nerve
d. Extra temporal

3rd Segment: Parts

a. Labyrinthine Shorter(3mm) & Narrowest (.68mm)
b. Tympanic / Horizontal
c. Mastoid / Vertical

Branches of facial Nerves:

1st Genu 3 Branches

a. Greater superficial petrosal Nerve (GSPN) :1st branch: Preganglionic Parasympathetic
b. Lesser Petrosal
c. External Petrosal

GSPN Combines with Deep Petrosal Nerve (Sympathetic fibres)
↓ forms
Vidian Nerve
(Nerve of Pterygoid Canal)

1st Branch (GSPN) Supplies & places

a. Lacrimal Glands
b. Nasal Gland
c. Minor Salivary
d. Taste in palate

3 Segments gives 3 Important (1st Branch)

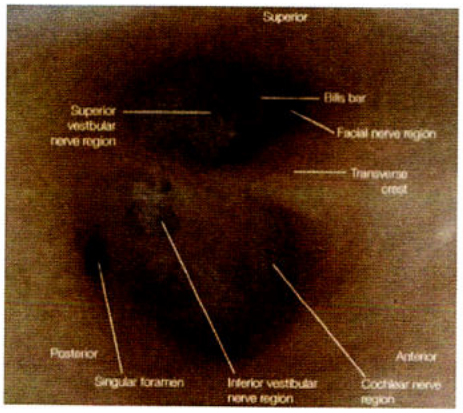
a. 1st Branch: GSPN
b. 1st Motor Branch: Nerve to stapedius
c. 1st Embryonic Branch: Chord Tympani

5 terminal Branches

a. Temporal
b. Zygomatic
c. Buccal
d. Marginal Mandibular
e. Cervical

Meatal Foramen

Meatal Foramen divided into 2 parts Transverse Crest
Upper Crest is further divided into 2 Parts – ant & Post by a
Verticle ridge of Bone – Bill's Bar
Named after Dr. William House
Dr. William House (father of Neuro-otology)
Anterior to Bill's Bar – Facial Nerve
Posterior to Bill's Bar – Superior Vestibular Nerve



In Inferior part - Posterior side – Inferior Vestibular Nerve & in Anterior side - Cochlear part

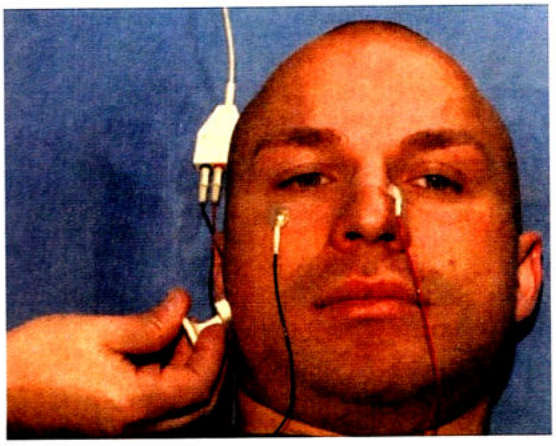
Topodiagnostic Tests

1. **Schirmers test / Lacrimation / Tear test**
   Normal: ⊖ve Facial Nerve is Normal till 1st Genu.
   Abnormal: ⊕ve : Injury at or above 1st Genu
2. **Stapedial Reflex test / Acostic reflex test**
   Normal: ⊕ve: Facial Nerve is Normal till 2nd Genu
   Abnormal ⊖ve : Injury at or Above 2nd Genu.

3 **Taste Test:**
Normal: + ve F.N. normal till Stylomastoid foramen
Ab Normal: -ve Injury above stylomastoid foramen

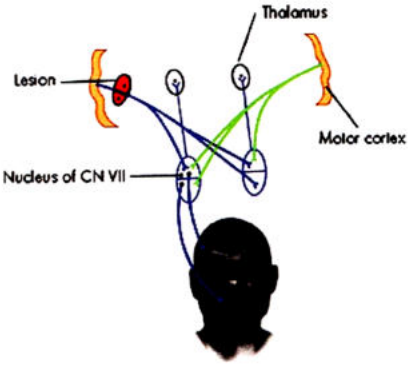
4 Submandibular Salivary flow Rate:

Normal : -ve F.N N till stylomastoid formen
Abnormal :+ ve injury above stylomastoid foramen



Electrophysiological Testing of facial Nerve
Based on Wallerian Degeneration.
Wait for 72 hours before doing Electrophysiological test
(Prognostic Test)
if Injury is on face it can be used as topo Diagnostic test.

UMN vs LMN of facial Nerve
LMN lesion: I/L Complete facial palsy
UMN Lesion :C/L lower half facial palsy



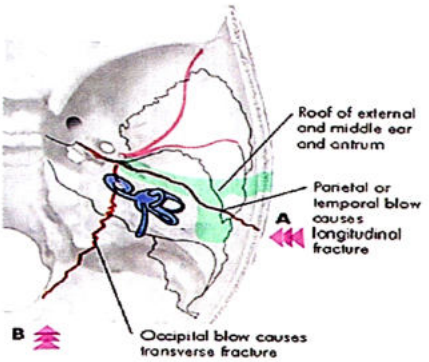
Disorders of facial Nerve
Bell's Palsy (M/C)
Idiopathic I/L LMN palsy
Viral Infection theory : Compression of labyrinthine segment
Tratment:
Steroids, Antiviral (72 hrs) Eye Protection, Physiotherapy
Herpes Zoster Oticus / Ramsay Hunt Syndrome
LMN Facial Palsy + Vesicular rash + Otalgia
Poor Prognosis as compared to Bell's palsy.

Crocodile tears Syndrome / Bogorad's Syndrome

Injury above 1st Gene
There is criss-crossing of fibres and whenever patient tries to eat, lacrimal gland are stimulated instead of minor salivary glands leading to lacrimation on eating food.
Iatrogenic Facial Palsy: M/C Cause: Mastoidectomy (vertical segment just after 2nd Gene)

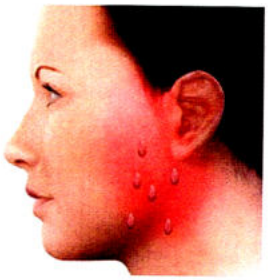
Temporal Bone Fractures

Longitudinal fracture: M.C Common (80%)due to blow from side
Transverse fracture: Occipital blow causes transverse fracture(20%)
Only 10% of Longitudinal fracture have facial Nerve Palsy
In Transverse fracture – 50% have facial Nerve Palsy
Mixed fracture (54%)



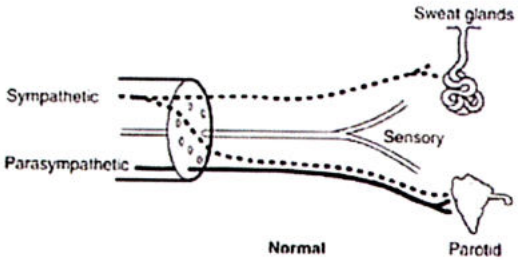
Frey's Syndrome :-

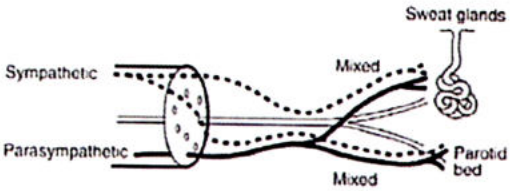
(Baillarger's Syndrome, Dupuy's Syndrome, auricotemporal Syndrome, Frey-Baillarger Syndrome)
There is criss crossing of fibres, Aurico temporal Nerve serves as Pathway of travelling of GlossPharyngeal Nerve & Supply Parotid Gland (Parasympathetic fibres)



After the Injury Parasympathetic fibres they get Inter changed and they also start supplying the sweat glands.

So whenever patient tries to eat there is sweating k/a freys syndrome.
This is due to injury to Auriculotemporal nerve
→ Nerve fibres of Glossopharyngeal Nerve (IX CN) are Getting damaged.
Auriculotemporal nerve is a branch of mandibular nerve but it carring the fibres of glossopharyngeal nerve.

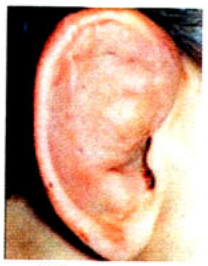


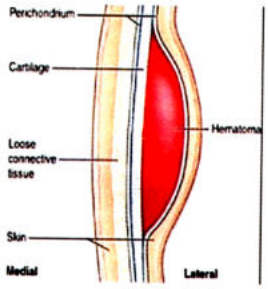


Diseases of External EAR

Pinna Hematoma (Cauliflower EAR) (Boxer's EAR)

→ D/t blunt trauma to pinna
→ Collection of blood b/w cartilage & perichondrium
RX → Needle Aspiration → Pressure dressing



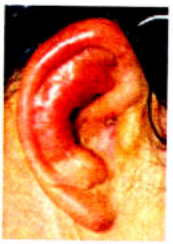


cauliflower ear

Perichondritis of Pinna

→ mc causative organism → Pseudomonas

Rx
1. Ciprofloxacin (Antibiotic of choice)
2. Analgesics
3. I & D (Incision & Drainage)



Perichondritis of Pinna

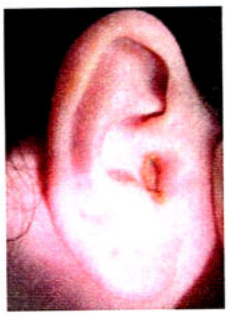
Acute Otitis External/ Tropical/ Swimmer's ear

→ Types
1. Localised/ furunculosis
2. Diffuse

1. Localised/ furunculosis

→ Staph. Aureus infection of hair follicles → furuncle

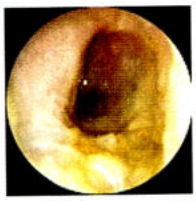
→ Localised to outer 1/3rd



localized/furunculosis type

2. Diffuse type:

→ mc causative organism → Pseudomonas
→ Severe pain/ excruciating pain
→ Tragal sign → Positive



Diffuse type

Rx

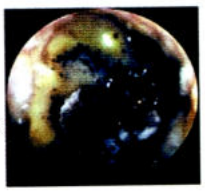
→ Antibiotics
→ Analgesics
→ 10% Ichthymol glycerine packing

Otomycosis/ Singapore EAR

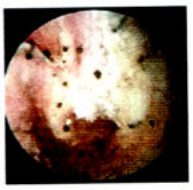
→ **Causative organism**
Aspergillus niger (mc) → forms black colonies
Aspergillus fumigatus → forms green colonies
Candida albicans → forms white colony colonies

O/E→ Wet blotting paper

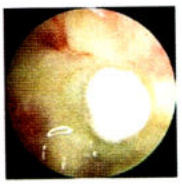
Rx → Aural toilet by micro suction
→ Topical antifungal ear drops for 4 weeks



Aspergillus niger (M/C)



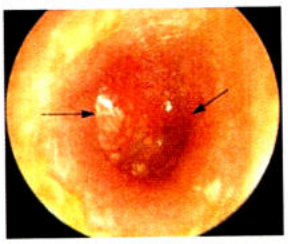
Aspergillus fumigatus



Candida albicans

Myringitis Bullosa Haemorrhagica (Bullous Myringitis)/ Hemorrhagic Otitis Externa

→ Formations of bleeding blebs on tympanic membrane
→ Blood mixed discharge +nt painful, conductive hearing loss
→ Caused by pneumococcus (Strep. pneumoniae)

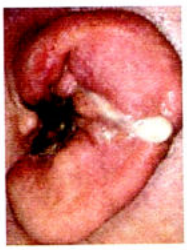


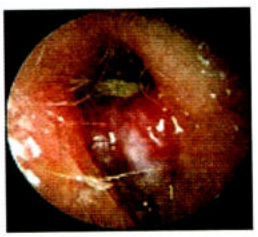
Rx

Topical Antibiotic
Topical Steroids

Malignant Otitis Externa/ Acute Necrotizing Otitis Externa

→ Malignant → misnomer
→ Rapidly spreading pseudomonas infection in a immune compromised patient

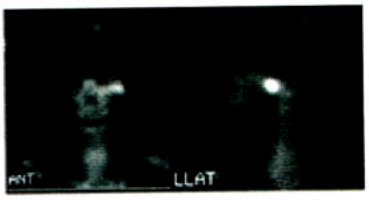




C/F

→ Pain
→ Greenish Black discharge
→ Lateral skull base osteomyelitis
- Multiple ca palsies (mc → facial N. palsy)
- Spread to skull bone

via fissures of Santorini (in cartilage)
- Diagnostic IOC - bone scan (TC - 99m)



Rx → Ciprofloxacin

Gallium scan (Ga-67) (Prognostic IOC) – non specific

ASOM (Acute Suppurative otitis media)/ AOM (Acute Otitis Media)

Causes

→ Streptococcus pneumonia (mc)

Stages

Stage 1 → Stage of Tubal occlusion

→ Nasopharyngeal end or cartilaginous end of Eustachian tube is blocked

→ ME pressure decrease

↓

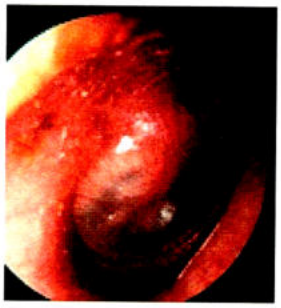
Tympanic membrane is pulled/ retracted

↓

Pain, ↓ Hearing

Dull & Luster less, non-shiny TM

cone of light absent

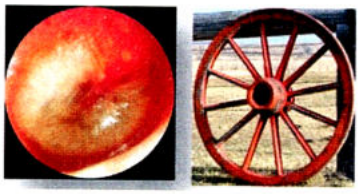


stage 1

Stage II →Stage of pre suppuration

→ serous, mucoid secretion accumulate in ME → bacteria is present in ME

→ TM bulges out, Blood vessels becomes prominent → Cart wheel Appearance



stage 2

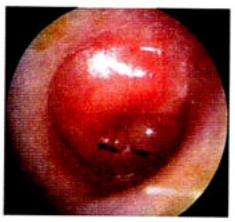
Stage III → stage of suppuration

→ Fluid → PUS (bacteria; macrophages)

→ Severe pain +nt

→ Tragus sign –ve

O/E: Red congested bulging TM ready to burst



stage3

Stage 4: Stages of Resolution

→ MC site of tm perforation → Antero inferior quadrant of pars tensa

→ After few weeks, perforation heals & hearing becomes Normal

→ A healed TM → Dimeric (No fibrous layer)

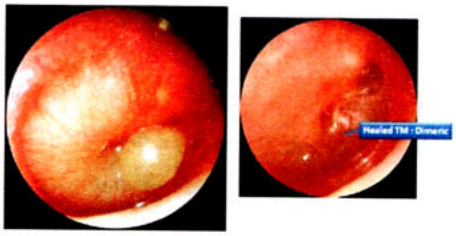
Rx

Stage I & II → Antibiotics, analgesics, Nasal decongestant drops

Stage III → Myringotomy (Performed in Postero inferior quadrant)

Stage IV → 90% → leave alone

M/C Complications – Perforation (According to latest Guideline's)



Stage 4

Acute Mastoiditis

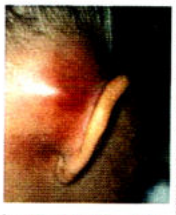
→ 2nd mc complication (Infection from middle ear has goes to mastoid)

O/E

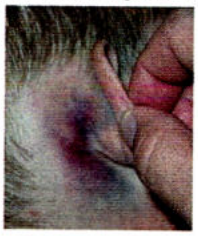
→ Ironed out mastoid :
Mastoid is red, hot and smooth

Battle Sign

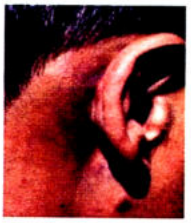
→ Hematoma over mastoid
d/t fracture middle cranial fossa



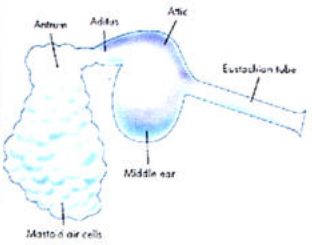
Ironed Out Mastoid: Acute Mastoiditis



Battle Sign: # Middle Cranial fossa

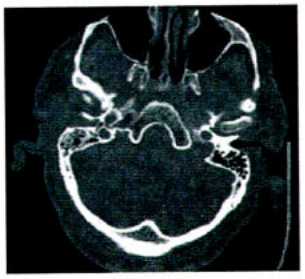


Griesinger's sign: Sigmoid Sinus Thrombosis



Gresseinger Sign

→ Pitting edema over mastoid
d/t sigmoid sinus thrombosis



2. **Coalescent mastoiditis** chronic infections in mastoid, leading to
Clouding and destruction of mastoid air cells (filled with fluid)

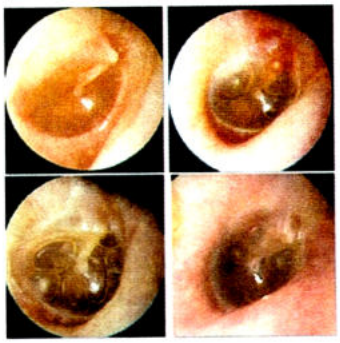
Rx → IV antibiotics X 3 weeks (DOC)

Glue EAR/ SOM (Serous otitis Media) (Secretory Otitis media)/ Non-suppurative O.M

→ Long standing collection of serious mucoid fluid middle ear

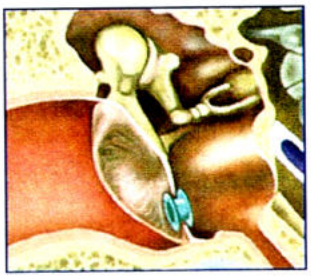
→ Hearing loss present (CHL – 25-30 db AB Gap ↓)

O/E → Fluid filled in middle ear → thin bulging TM with fluid behind TM. Air bubbles, airfluid levels may be seen. Blue TM (Sometimes)
also seen in hemotympanum → Laugier's sign
(middle cranial fracture; fossa fracture) cholesterol Granuloma
Glomus has reddish Blue T.M.



Mx

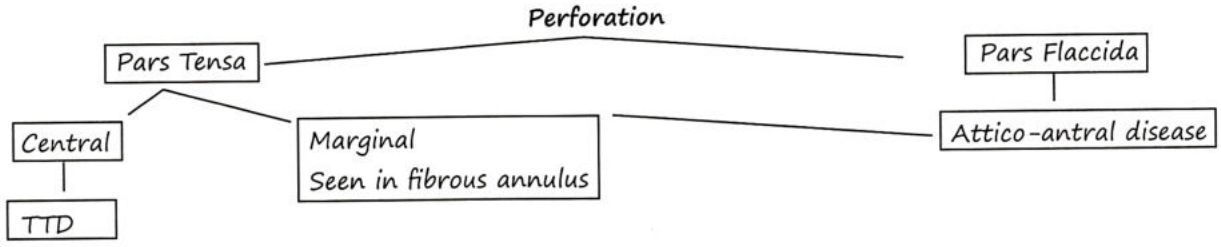
1. Treat the cause
2. Myringotomy & grommet insertion in Antero inferior Quadrant



CSOM(Chronic Suppurative Otitis Media) / COM (chronic Otitis media)

→ Tubo Tympanic Disease (TTD)

→ Att o antral disease(AAD)



Tubo Tympanic Disease

Central perforation not involving fibrous annulus.

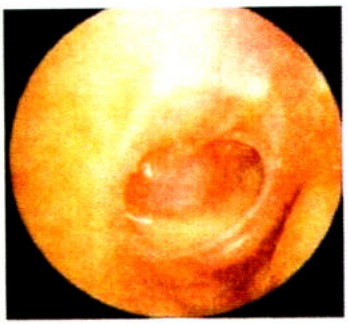
→ CHL (AB gap → 10-40 dB) (depends on site & size of perforation)

→ Ear Discharge

- Mucoid (or) Mucopurulent
- Non foul smelling
- Continuous or intermittent
- Copious in Quantity

It is also called as Mucoid disease

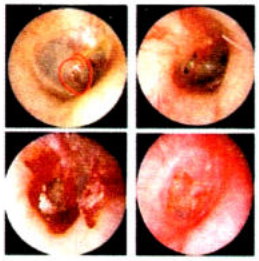
1. Active (Discharge coming)
2. Inactive discharge (Not coming)



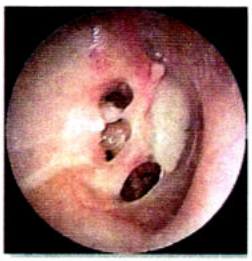
Tubo-tympanic Disease (TTD)

Rx

Active → Antibiotics (for Discharge)
Myringoplasty (Repair of TM)

Traumatic Perforation	Diseased Perforation
Irregular, rough margins Blood clots around perforation  traumatic perforation	Circular or kidney shaped with Smooth margins

→Sieve like perforation (Multiple perforations): Seen in TB



Types of Tympanoplasty

Type 1 Tympanoplasty/ Myringoplasty
→ mc used graft → Temporalis fascia

Type II Tympanoplasty
→ Myringoplasty + Ossiculoplasty (Normal middle ear physiology maintained)

Type III Tympanoplasty/ Myringostapediopexy (columella effect)

Type IIIa → Graft directly placed on stapes head

Type IIIb → PORP (Partial ossicular Reconstruction prosthesis)

Type IIIc → TORP (Total ossicular Reconstruction prosthesis)
→ A real ratio is maintained
→ Lever ratio is not present

Type IV Tympanoplasty (Baffle effect)
→ Graft is kept over round window
→ Phase difference present but no amplification

Type V Tympanoplasty/ fenetration operation
Earlier done for Otosclerosis, now obsolete

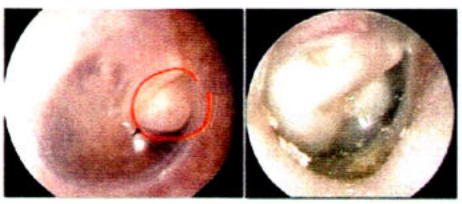
Type VI Tympanoplasty/ Sono inversion
→ Graft is placed over oval window phase difference is created, reverse of type IV Tympanoplasty

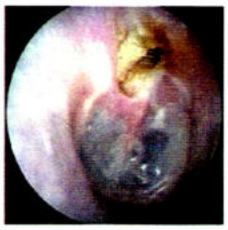
Attico Antral Disease

Cholesteatoma (Keratoma)
→ Normal keratinized stratified
→ squamous epithelium
→ In abnormal place (middle ear cleft)

Type
1. Congenital
2. Acquired

Congenital Cholesteatoma





Congenital Cholesteatoma
→ Epithelium is trapped inside, during or before the formation of middle ear cleft. It look like a bright pearly mass, behind an intact tympanic membrane (No h/o of Tympanic memes perforation)

Acquired cholesteatoma

→ Epithelium goes into the middle ear cleft after birth

2 Acquired Cholesteatoma

→ 2 to perforation

→ Migration of squamous epithelium

- Marginal perforation

→ Squamous metaplasia

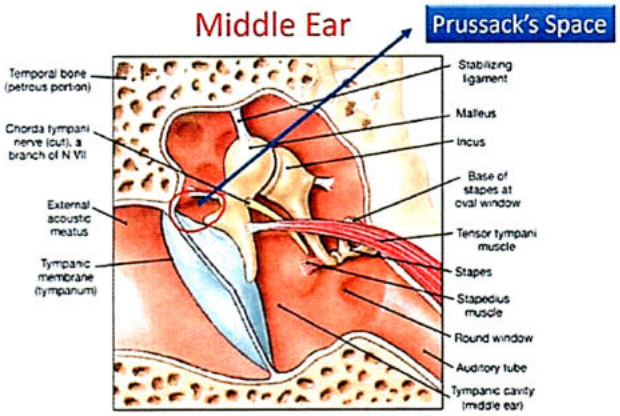
1°Acquired Cholesteatoma

Wittmack's Retraction Pocket Theory/theory of Invagination

→M/C formed in Prussack's space in epitympanum

boundaries - Superior → Lateral malleal ligament

- Medial → Neck of malleus
- Inferior → Lateral process of malleus
- Lateral → Pars flaccida & Scutum



Clinical Features

→ Conductive hearing loss (AB Gap 45 db)

→ Discharge → Scanty

→ Purulent

→ Foul smelling

→ Blood stained (due to bone erosion)

→ intermittent/ continous

Management

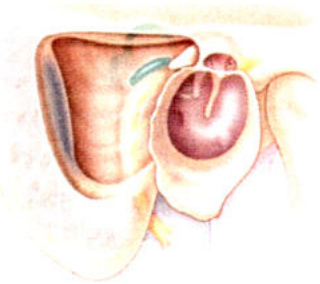
Atticoantral disease is classified as squamous disease

→ Active (Discharge)

→ Inactive (Discharge Not present)

1. **Treatment**

→ Modified Radical mastoidectomy



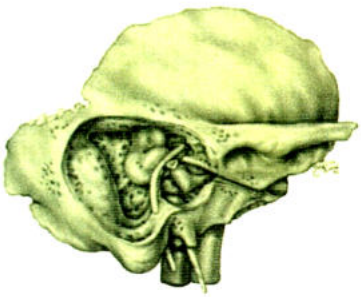
Mastoidectomy – Types

Simple/ Cortical/ Schwartz Mastoidectomy

→ Just drill in the mastoid

→ Indicated for

- Coalescent mastoidectomy
- Cochlear implant



Radical mastoidectomy

Common cavity of mastoid & middle ear is created by removing common wall between the two.

Indication → Malignancy

Modified Radical Mastoidectomy

Same as radical mastoidectomy, however conductive hearing apparatus is also preserved, if conductive apparatus has been damaged, it is repaired

Rx of choice for Attico-antral disease

Complications

Chronic Suppurative otitis media (CSOM/SOM) complications

→ Intra cranial

→ Extra Cranial

Intra Temporal

Extra Temporal

Intra Temporal Complications

1. Ossicular chain Erosion (mc complication) –CHL.
2. Labyrinthitis
   - Serous Labyrinthitis → SNHL reversible
   - Suppurative labyrinthitis → SNHL Irreversible
3. Labyrinthine Fistula

   mc site → done for lateral semicircular canal

   Fistula Sign/ test → pressing on tragus with finger vertigo or nystagmus occurs

   True +ve Fistula test → Fistula present, Fistula sign +ve

   False –ve fistula test → Fistula present, Fistula sign –ve

   Seen in

   → Dead ear

   → Cholesteatoma covering fistula

   → False +ve Fistula test → No fistula

   → Fistula sign +ve

   Seen in

   → Congenital Syphilis
   → Menier's disease

   } Hennerbert's Sign

4. **Petrositis**

   → Long standing discharge
   → Deep seated retro orbital pain
   → Diplopia of lateral gaze

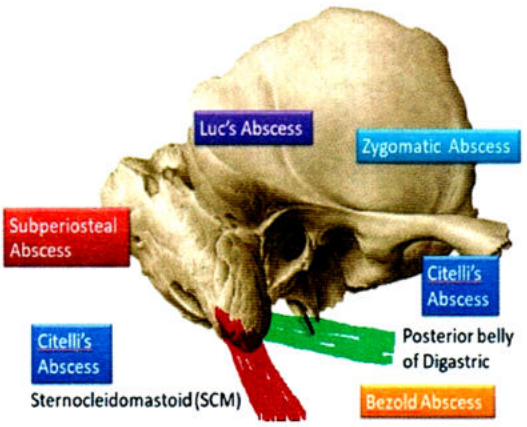
   } Gradenigo Syndrome (Grade "D")

   - d/t lateral rectus palsy
   - d/t abducent nerve nerve palsy (6th canal) in Dorello's canal (6th n. canal)

Extra Temporal Complications:

When infection come out of temporal bone it leads to extra Temporal complications.

Temporal bone on mastoid process there are 2 muscles attached,



on lateral aspect sternocleidomastoid is attached, on medial aspect posterior belly of digastric is attached

1. **Post Aural/ Sub Periosteal Abscess** IF the cholesteatoma breaks
   The mastoid bone and comes out behind the pinna this is k/a
   Sub periosteal abscess.
2. **Bezold Abscess** if choleasteatoma breaks the bone at the tip of mastoid
   And goes along sternocledomastoid into the neck it is called as bezold abscess
3. **Luc's Abscess** if cholesteatoma breaks the wall of external auditory canal and forms abscees in
   External auditory canal it is called as luc's abscess.
4. **Zygomatic abscess** if cholesteatoma breaks the root of zygomatic process it is called
   Zygomatic abscess . It lies anteriosuperior to external auditory canal.
5. **Citellis's Abscess** → IF cholesteatoma breaks the bone medial to mastoid where posterior belly of
   1.digastric is attached, it can spread along posterior belly of digastric or spread behind the mastoid

Intra Cranial Complications

1. Meningitis (mc)
2. Extra Dural Abscess or epidural abscess.
3. Sub Dural Abscess
4. Brain Abscess: abscess inside brain parenchyma
   types
   → Temporal abscess
   → Cerebellar Abscess

   Both are seen with csom but temporal abscess is more common
   Most common cause of brain abscess is otogenic(ear infections)
   Brain abscess can have different stages it starts with stage of encephalitis and then
   Stage of latency for next 1-3 days and after that it start expanding in next 3-10 days and there
   after it undergoes rupture
5. Otitic Hydrocephalus (Rarest)
6. Sigmoid/ Lateral Sinus Thrombosis

Sigmoid/ Lateral Sinus Thrombosis

Clinical Features

→ **Picket Fence Fever**
Remittent Fever (Fever rises, goes down but never come to Normal)

→ **Grieseinger sign** → Odema over Mastoid (Emissary Vein Thrombosis)

→ **Tobey Ayer/ Queckenstedt Test**
Rise in intra cranial pressure when IJV is pressed over normal side

Crow Beck Test

→ Whenever IJV is pressed normal side there is engorgement of retinal veins

CECT/MRI

→ Empty delta Sign elicited

→ Most important sign seen in sigmoid sinus thrombosis.

Otosclerosis

→ Enchondral Bone changes to Spongy bone.

M.C site of formation of spongy bone is at Fistula Ante Fenestrum
It is just anterior to the oval window and that leads to fixation of stapes.

- M.C site for fixation of stapes is Anterior 1/3rd or anterior crura
  This spongy bone grows and completely fixes the stapes foot plates & results in decreased hearing.

Presentation

- Female Male → 2:1
- 20-30yrs
- The incidences increases during pregnancy
- Autosomal Dominant (50 % positive family history)

→ **Syndrome Associated with otosclerosis: -**

Vander Hoeve syndrome: -

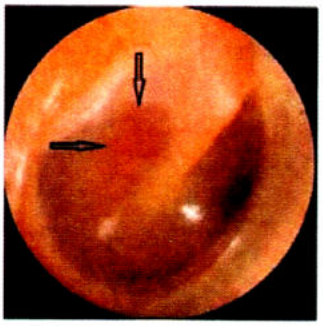
(Otosclerosis + osteogenesis imperfecta + Blue sclera)

- B/L disease
- Conductive Hearing loss
- Paracusis Willisi (Hear better in a Noisy environment)

O/E

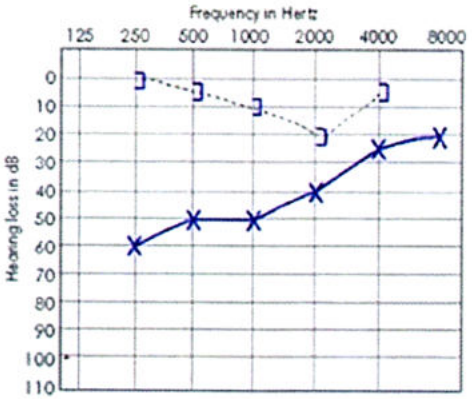
Schwartz sign

→ Flamingo Pink coloured Tympanic Membrane, Seen in early or active phase of disease. a/K/a phase of otospongiosis.



Schwartz sign

Pure tone audiometry:-



- Ab gap into 60 db seen
- Maxium possible conductive hearing loss that can be seen
  Dip in bone conduction @2000 Hz k/as Carhart's notch

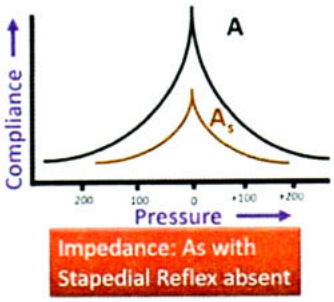
Note:-

- This is disease of air conduction, but **carhart's notch** is in bone conduction @2000 Hz
- Similar dip in bone conduction @ 4000 Hz is seen in **Noise induced hearing loss**

Impedence audiometry →

- As types of curve

→ Spapedial reflex is absent because stapes is fixed

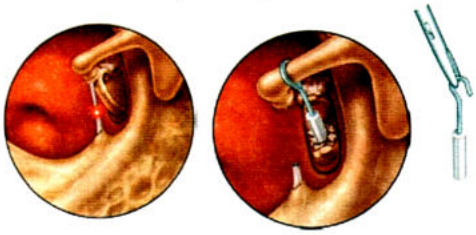


Impedance: As with Stapedial Reflex absent

Rx Laser Stapdotomy

A hole is made in stapes footplate & piston placed in it & anchored to Long Process of Incus

Laser Stapedotomy

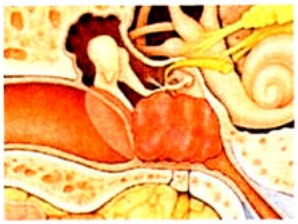


Glomus tumour

M/C benign tumour of middle ear

Age: 40-50 yrs of age group

Female: Male → 5:1



It arises from Para ganglionic cells (Glomus Body)

Glomus tumor can be divided into 2 types

Globus Tympanium

↓

It is arising from the paraganglionic cell which are on the promontory along the course of Jacobson's Nerve (branch of IX Cranial Nerve)

Limited to Middle Ear

It causes palsy of VII Nerve

Glomus Jugulare

↓

Arises from Dome of Jugular

It involves Juglar foramen

Spreads to skull base

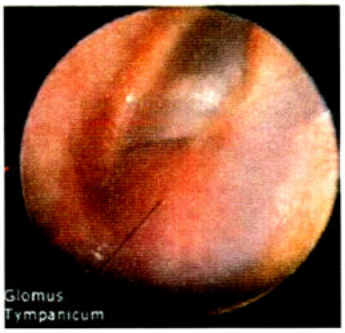
Can cause multiple cranial nerve Palsy (IX, X, XI, Cranial Nerve) jugular foramen syndrome (a/k/a Vernet's syndrome)

Clinical Presentation:

Symptoms:

- Conductive Hearing loss
- Pulsatile tinnitus

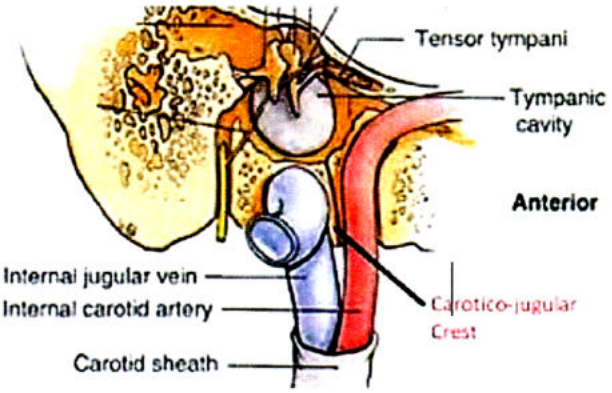
O/E: Profusely bleeding polyp



Signs:

- **Rising sun sign**
  Reddish blue tumor rising from hypotympanum
- If juglar vein is pressed the tinnitus will be Improved K/a **Aquino sign**

Brown Sign: On seigalization, tumour blanches and becomes white.
IOC → CECT



Phelp Sign Seen:
Inability to distinguish b/w Internal carotid artery and Juglar bulb due to erosion of Cartico juglar Crest

10% Rule of Glomus:
- 10% MultiCentric
- 10% Familial
- 10% Secretory (Secrete Catecholamines)

Rxoc-Surgical Excision

Meniere's disease: Endolymphatic hydrops

- disease of cochlea.
- Endolymphatic hydrops: Endolymph Accumulates inside Cochlea leading to bulging of Reissner's membrane. It can burst and there can be mixing of endolymph with Perilymph

Presentation:
- U/L disease
- Affects Male more
- 35-40yrs
- Meniere's disease is **triad** of mainly 3 symptoms.

  Episodic Vertigo
  Fluctuating Sensineural Hearing loss
  Tinnitus

Other than this Patient Can also complain of aural fullness

- **Tullio Phenomenon:**
  Loud noise precipitate vertigo

- **Lermoyez syndrome**: variant of Meniere's disease: vertigo relieves sensorineural hearing loss

- **Tumarkin crisis**
  Sudden fall on the Ground because of the severity of meniere's disease

Investigations:

1. **Glycerol test:** Glycerol is hygroscopic agent which will absorb water and it will lead to a 10 db ↑ in sensineural hearing loss on pure tone audiometry or 10% improvement in speech speech discriminations score on speech audiometry

It is done Generally in acute attack of **Meniere's disease**

Pure tone audiometry

In Early meniere's disease, there is rising curve.

In case of late meniere's disease it shows a Sloping curve

Gold Standard investigation → Electrocochleography

In this Summating Potential is compared with action potential

Sp - > 70% of A.P.

SP/AP ratior > 0.7

Rx: Acute attack of meniere's disease

- Labyrinthine Sedative P.O.C Prochlorperazine, Cinnarizine

Maintain Phase

Medical Management:

i. Diuretics
ii. β-Blockers
iii. Histaminics

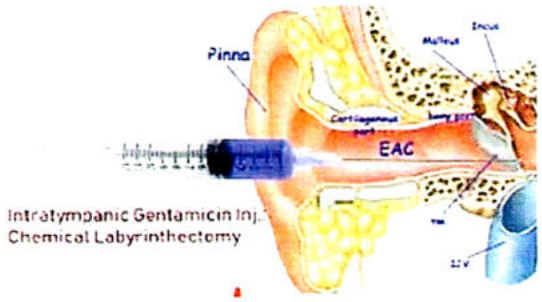
Surgical management:-

Conservation Surgery: Endolymphatic Sac decompression

Radical Surgery: Surgical Labyrinthectomy

Gold Standard Treatment in intractable vertigo

(No relief despite all treatment) – Surgical Labyrinthectomy



Nowadays New treatment is available, before going from medical to surgical management

→ Intra tympanic Gentamycin therapy (chemical labyrinthectomy)

→ New method of delivery of Gentamicin to inner ear is **Micro wick Microcatheter system** (Drug delivery system)

→ Meniett's device: New home treatment device, it is USA FDA device

→ Intermittent low pressure pulse therapy

Vestibular schwannoma:

Earlier k/as Acoustic Neuroma.

→ M/C Benign tumor of CP angle (cerebello pontine angle)

M.C site of origin

→ Schwann cells of inferior vestibular Nerve inside the

→ Internal auditory Canal.

Symptoms

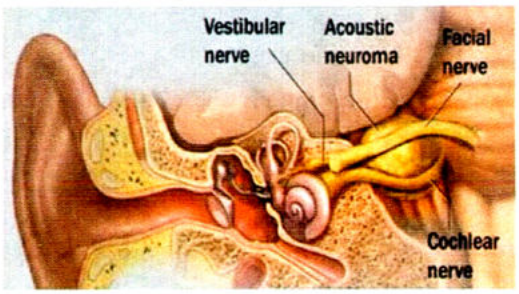
→ U/L slow progressive SNHL (M/C)

→ Tinnitus

Signs

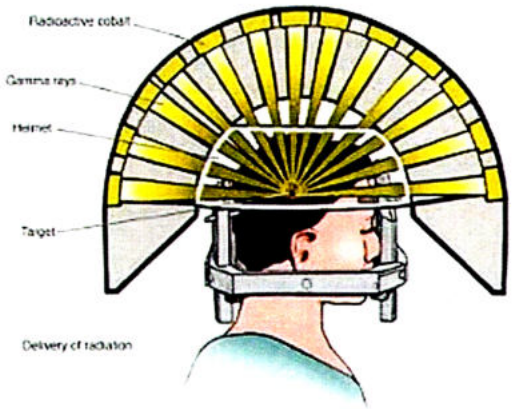
→ Loss of corneal reflex (Vth Nerve involvement)

→ M/C cranial Nerve involve 8th Nerve.



Hitzelberger Sign: Involvement of 7th cranial Nerve where sensory supply to Postero superior wall of EAC is lost

IOC: Contrast enhanced MRI (Gadolinium contrast)



Treatment

Treatment of Large Tumour

Surgical excision

Treatment of small tumour.

→ **1. Old patient, slow growing tumor**

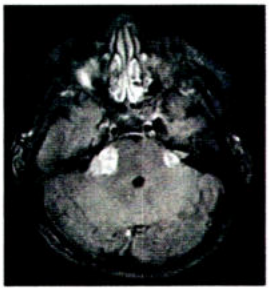
Rx: is Wait and watch

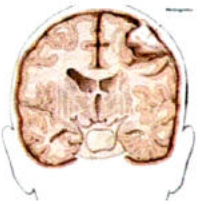
MRI is done every 6th months and observation

→ **2. Young Patient, Fast Growing Tumour**

Rx: Gamma Knife Excision/ Cyber knife excision

This is targeted radiotherapy technique a/k/a stereotactic radiotherapy





Meningiomas

Neurofibromatosis 2

→ NF – 2 Gene mutation

→ Produces a tumour suppressor NF-2 Gene k/as Merlin

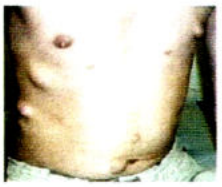
Presentation

→ B/L vestibular schwannoma

→ Meningioma's

→ Multi Fibromas

→ Vestibular schwannoma are generally seen in young

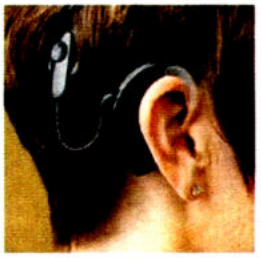


Rx: B/L surgical excision

Rehabilitation: Auditory Brainstem Implants (ABI)

Hearing Devices/ Implants

1. **Cochlear Implants (Bionic sense organ)**



Components

1. External Body worn consist of Microphone, speech processor, transmitter.
2. Internal implantable component /Surgically implanted Part

consist of receiver/ stimulator and electrode array

Surgically implanted Part consist of receiver/ stimulator and electrode array

→ This electrode array is implanted in the cochlea in scala tympani through round window and stimulate 8th Nerve

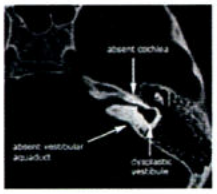
Indications

1. Severe to profound SNHL: adult with 70 db hearing loss & children > 90 db
   → Implanted in U/L or B/L profound SNHL
2. Poor speech perception: SDS < 20-30%
3. No improvement with hearing aids
4. Age 1 year or older

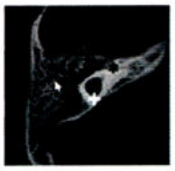
Maximum Priority is given to post – lingual deaf – children

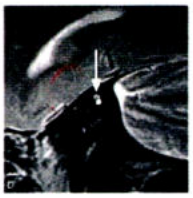
Absolute contraindications of Cochlear implants

1. **Michel Aplasia**: No cochlea
2. **8 th Nerve aplasia**
3. **Scheibe Dysplasia**: a/k/a cochlea saccule dysplasia.
   Part of cochlea is damaged.
4. Neurofibromatosis Type II with Bilateral vestibular schwannoma
5. **Pre lingual deaf Patient ≥ 7 Years**



Michel Aplasia


Scheibe dysplasia


8th Nerve aplasia

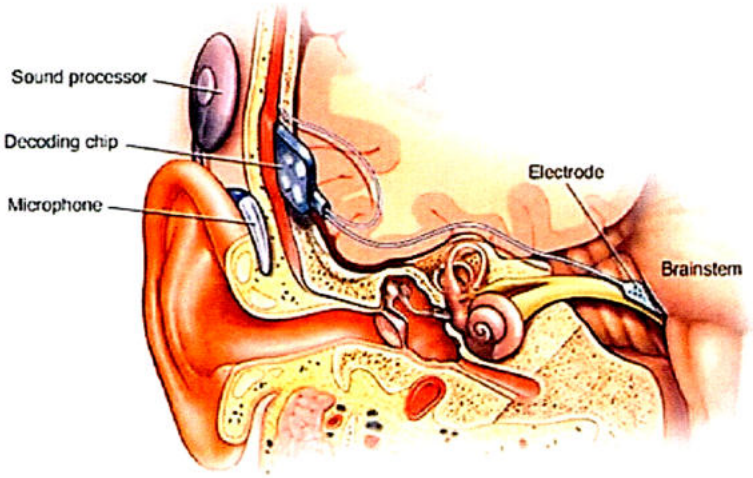
Mondinis' dysplasia is not a absolute C/I
Mondini's dysplasia is a relative contra indication

Auditory Brain Stem implant (ABI)

→ It is used when cochlear implant cannot be used All C/I of Cochlear Implant becomes indications of Auditory Brains stem Implant

→ ABI is similar to cochlear implant, only difference is that electrode array is shaped in different way and this is implanted on Cochlear Nucleus (4th Ventricle)

4th ventricle is reached through the foramen of Luschka in the brain stem.



Nose

Externally Nose Consists Of

Upper 1/3rd → Bony, made up of nasal bone which is supported by frontal process of maxilla.

Lower 2/3rd → Cartilaginous, consists of 3 paired and 1 unpaired cartilage

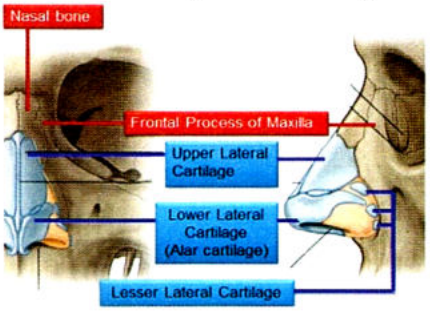
Paired Cartilages

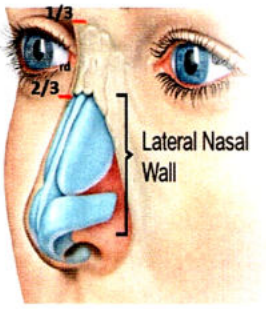
→ Upper lateral

→ Lower lateral (Alar)

→ Lesser lateral / Lesser Alar Cartilage

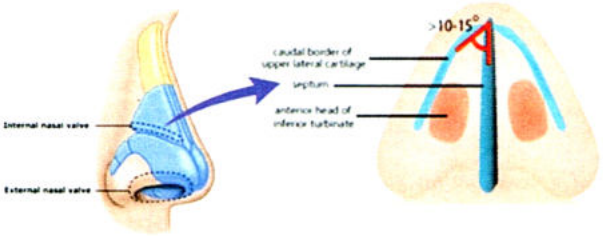
Unpaired → Septal cartilage

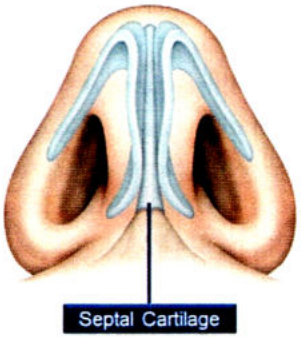




Medial crus of alar cartilage goes inside the septum and it form columellar septum.
Just behind the columellar septum, there is membranous septum, where there is no bone or cartilage and septal piercing is done in membranous septum. lower end of upper lateral cartilage it forms internal nasal valve along with nasal septum.

Internal Nasal Valve

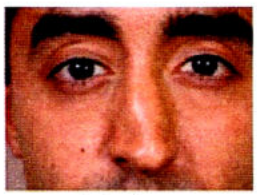




→ The angle b/w the caudal end or lower end of upper lateral cartilage with the septum should be ≥ 10-15° for good nasal patency and is k/a minimum angle.

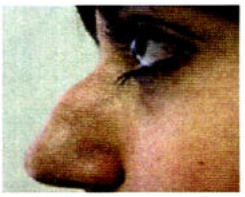
→ Internal nasal valve aka Limen Nasi or Limen vestibuli this leads to 50% of total nasal airway resistance.

Deformities and Diseases of External Nose

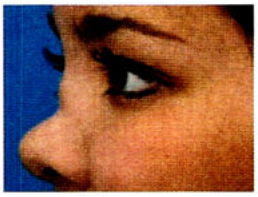


Deviated Nose
Rx by Rhinoplasty

Crooked Nose
'C' shaped deviation
Rx by Rhinoplasty



Nasal Hump
Rx by reduction
Rhinoplasty



Saddle Nose
Rx by Augmentation
Rhinoplasty

Rhinophyma (Potato Tumor)

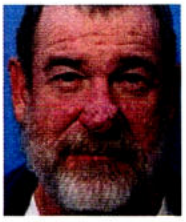
→ Tumor word is misnomer because there is no hyperplasia.

→ More common in males, 35 – 40 yrs

→ There is benign hypertrophy of sebaceous glands

→ There is Lobulated mass of Nose

Rx → Derma Abrasion with CO laser



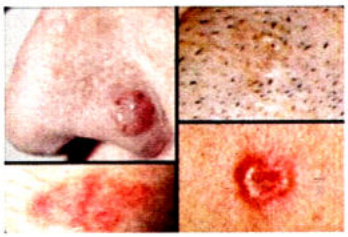
Rhinophyma

Rodent Ulcer (Basal Cell Carcinoma)

Most common skin malignancy

It is generally on exposure to UV rays on skin exposed area such as Dorsum of hand and face
It is k/a rodent ulcer because outside appearance is less than what is inside.

→ Locally invasive tumor
→ Distant metastasis is rare
Rx → Wide Local Excision



Rodent Ulcer

Nasal Cavity

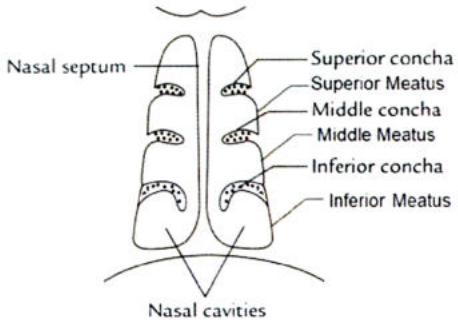
→ Medial wall of nasal cavity is formed by nasal septum.

Lateral Wall

There are 3 Turbinates

→ Superior turbinate / Concha
→ Middle turbinate / Concha
→ Inferior Turbinate / Concha

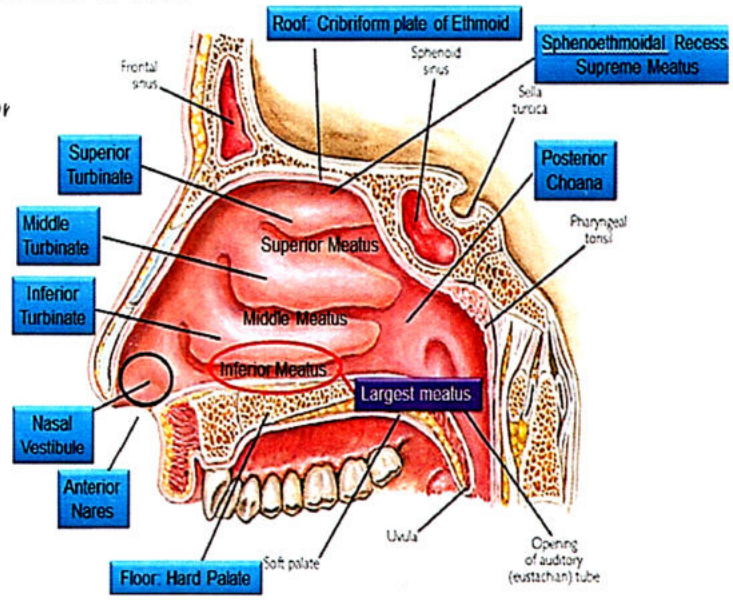
The space below each turbinate is k/a meatus
Below Superior Turbinate there is superior meatus
Below Middle Turbinate there is middle meatus
Below Inferior Turbinate there is Inferior meatus



Lateral Wall of Nose

→ Roof of Nasal Cavity Is formed by cribriform plate of ethmoid bone
→ Cribriform plate of Ethmoid bone is thinnest bone of body. It is called cribriform because it has holes and through these holes the olfactory nerves fibres pass through.
→ Olfactory nerve fibres are bipolar neurons.
→ Cribriform plate of ethmoid is k/a dangerous area of nose.

Floor of Nasal Cavity is formed by hard palate.
Hard palate is formed by 2 bones, maxilla anterior and palatine bone posteriorly.



Anterior Opening of nasal cavity is known as nares through which air enters into nasal cavity.
Posterior Opening is k/a choana

Nasal Vestibular

→ In nasal vestibular lot of hairs are present.
→ Any infection by Staph. Aureus is K/a as Nasal Vestibulitis.

Treatment → Systemic antibiotics

→ The lower part of nose between 2 naso labial folds and upper lip is k/a dangerous triangle of face.

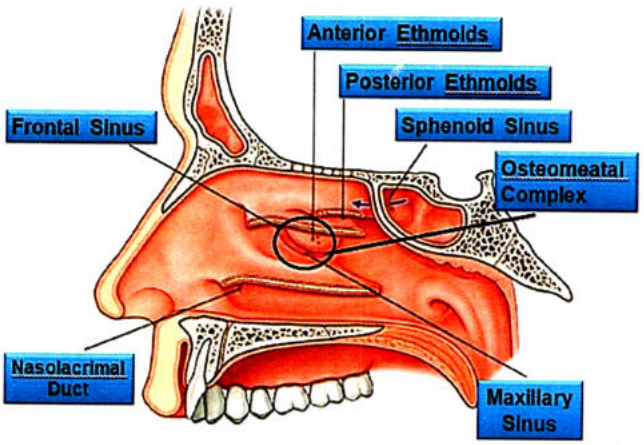
Infection from dangerous area of face can quickly go to cavernous sinus and can cause cavernous sinus thrombosis

→ The space above the superior turbinate is k/a sphenoethmoidal recess / supreme turbinate.
→ Sometimes 4th turbinate is present in sphenoethmoidal recess k/a supreme turbinate.

Inferior meatus is largest meatus.
Normally NLD Opens into inferior meatus; but after DCR (Dacryocystorhinostomy) NLD opens into middle meatus.

Direction of NLD is

N → Inwards
L → Laterally
D → Downwards



Middle Meatus

There Is Opening Of

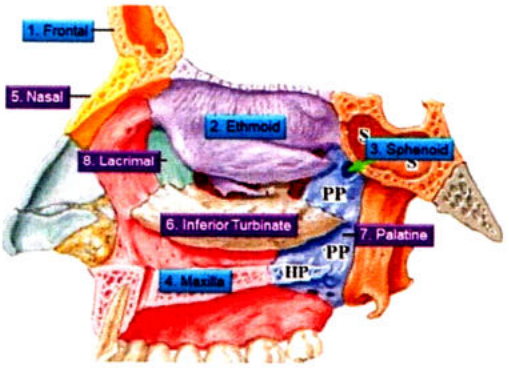
(i) Maxillary sinus
(ii) Frontal sinus
(iii) Anterior ethmoids

→ Ethmoids opening in middle meatus are k/a anterior ethmoids.
→ Ethmoids opening in superior meatus are k/a posterior ethmoids.

Sphenoid Sinus

→ Sphenoid Sinus opens in area that is above the superior turbinate which is k/a Spheno ethmoidal recess / supreme meatus.

Osteomeatal Complex (OMC) → Key area on lateral wall where there is opening of maxillary sinus, frontal sinus and anterior ethmoids this is known as '**OMC**'.



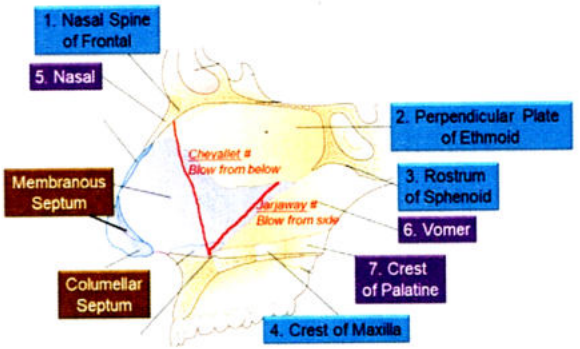
Lateral Wall – Bony Anatomy

Total 8 Bones < 4 + 4

Para Nasal Sinus Bones	Individual Bon
e→ Frontal Bone	→ Nasal Bone
→ Ethmoidal Bone	→ Inferior Turbinate
→ Sphenoid Bone	→ Palatine Bone
→ Maxillary Bone	→ Lacrimal Bone

Bones which do not contribute to nasal septum
"**Lacrimal Bone**" or "**Inferior Turbinate**"

Bones of Nasal Septum
total 7 Bones = 4+3



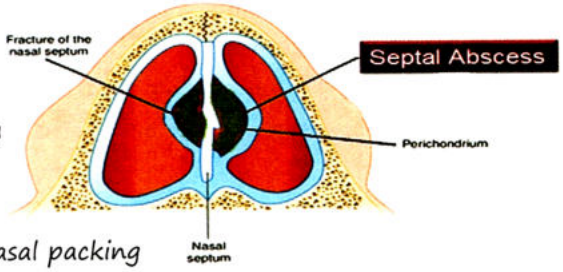
Para Nasal Sinus Walls	
→ Nasal Spine of frontal bone	→ Nasal bone
→ Perpendicular plate of ethmoid	→ Vomer
→ Rostrum of sphenoid	→ Crest of palatine bone
→ Crest of maxilla	

Vomer → It is exclusive to septum it does not contribute to the lateral wall.

Jarjaway fracture. Any Injury of nasal septum such as blow from side cause fracture running parallel to the cartilage vomer junction

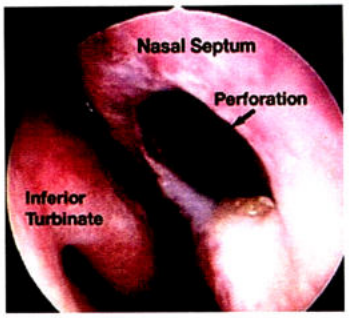
Chevallet fracture → If the punch is landed on the nose from below there can be fracture running from anterior most point of maxillary crest (Nasal spine of maxilla) upto nasal spines of frontal bone.

Septal hematoma
Collection of blood B/W the cartilage and its mucoperichondriu
→ B/L nasal obstruction



Treatment → Incision and drainage followed by B/L anterior nasal packing
Septal Abscess: If this haematoma get infected, there can be pus between cartilage and mucoperichondrium, it will be k/a septal abscess.

Rx → Incision and drainage B/L anterior nasal packing along with IV antibiotics
Most common complication of septal abscess is septal perforation.



Septal Perforation

Most common cause → Trauma
Trauma can be Nose Picking, Iatrogenic due to chemical cautery or Electro cautery on septum or septal surgery.
Nose picking is type of trauma but however nose picking itself is not a most common cause of septal perforation.
(Nose picking is most common cause for Anterior Epistaxis)

Other Causes

→ Septal abscess

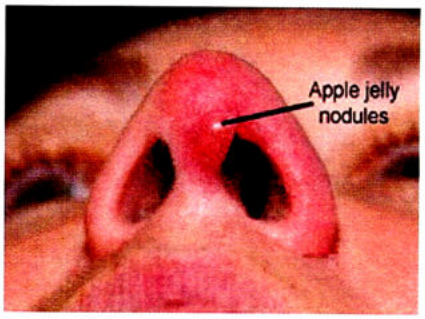
→ Septal piercing

→ Cocaine abuse

→ Granulomatous disorders

Granulomatous disorders are of various types

Cartilaginous	Bony	Both (Cartilaginous Bony)
E.g. TB, Leprosy	E.g. Syphilis	E.g. Wegner's Granulomotosis
Lupus vulgaris		



Apple jelly nodules are seen in lupus vulgaris.
Lupus vulgaris is mild form of tuberculosis skin lesions.

Blood Supply of Nasal Septum

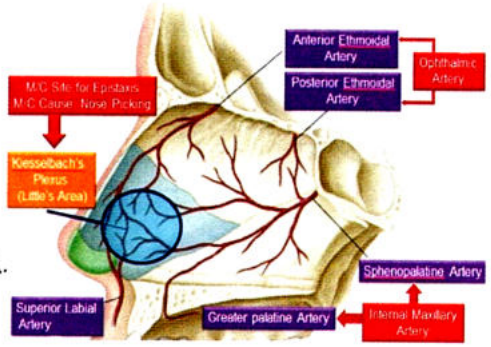
Septum Is Supplied By 5 Arteries

→ Anterior Ethmoidal Artery
→ Posterior Ethmoidal Artery
} Branches of Ophthalmic Artery

↑

Branch of ICA

→ Sphenopalatine Artery
→ Greater Palatine artery
} Branches of Internal Maxillary Artery



→ Septal branch of superior labial artery → Branch of Facial Artery

Both facial artery and internal maxillary artery is branch of ECA.

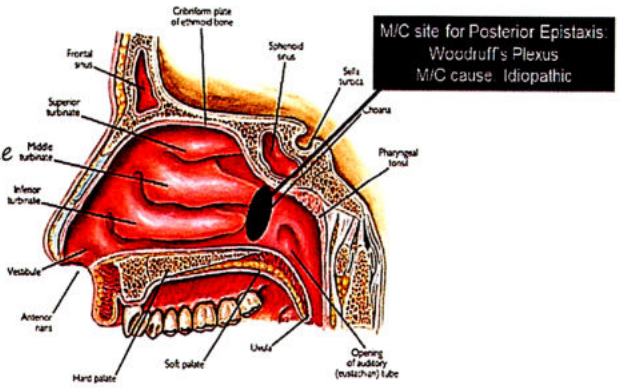
Nose is being supplied by Both ICA and ECA systems.

Little's Area

→ In the anterior inferior part of nasal septum there is an arterial plexus formed by 4 arteries.
→ This plexus is k/a Kiesselbach's plexus in the area k/a Little's area.
→ Posterior Ethmoidal Artery does not contribute to Kiesselbach's plexus.
→ Branch of ICA which contribute to the Kiesselbach's Plexus → Anterior Ethmoidal Artery
→ Most common site for epistaxis is Kiesselbach's plexus.
→ Most common cause → Nose picking.

Posterior Epistaxis

→ Most common for posterior epistaxis is area behind the posterior of middle and inferior turbinate on the lateral wall → k/a Woodruff's plexus
→ Most common cause → Idiopathic



Management strategy for epistaxis

↓

Resuscitation

↓

Bleeding point visible on anterior Rhinoscopy **YES** → **Anterior Epistaxis Chemical / Electro cautery**

↓ NO

Posterior Epistaxis
Treatment of Choice → Endoscopic Electrocautery

↓ Fails/Diffuse bleeding on endoscopy

B/L anterior nasal packing

↓ Fails

B/L Anterior packing + Posterior Nasal Packing (Foley's catheter, Bellocq's Pack,gauge peice)

↓

Vessel Ligation

Endoscopic sphenopalatine artery ligation

↓ Not respond

Internal maxillary / Artery ligation

↓ Not respond

External carotid artery ligation

→ Sphenopalatine artery is artery of epistaxis. It is most common artery involved epistaxis and it is first vessel to ligated.

→ Even after EC ligation there is bleeding that is most likely from anterior ethmoidal artery which can be ligated.

→ We never Ligate internal carotid artery.

Hereditary Hemorrhagic Telangiectasia (HHT)/ Osler Weber Rendu disease

Autosomal Dominant Disease. Diagnosis is done by "**Curacao Criteria**"

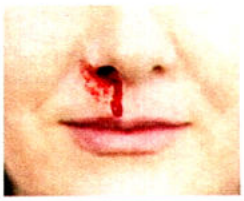
Curacao Criteria Consists Of 4 Things (At least 3 criteria out of 4 criteria should be Present to confirm it as a case of Osler Weber Rendu Disease)

→ Spontaneous recurrent epistaxis (H/O of bleeding without any cause, trauma)

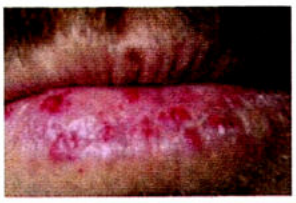
→ Multiple Telangiectasia at Lips / Oral Cavity / Figures / Nose

→ Arteriovenous malformation in liver, gut

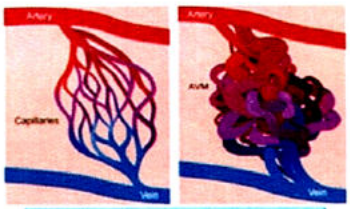
→ Family history in first degree relatives



Spontaneous Recurrent Epistaxis



Telangectasias at Lips/ Oral cavity/ Fingers/ Nose



Visceral lesions: AVMS

Family History

Paranasal Sinus

Development Of Paranasal Sinus

Sinus	Present at Birth	First X-ray appearance	Adult Size
Maxillary Antrum (Antrum of Highmore) Largest 15ml	Yes Earliest 12 weeks IUL	4-5 months after birth	15- 18 yrs
Ethmoid	Yes	1 yr	12 yrs
Sphenoid	Yes	6- 7 yrs Last	15 yrs
Frontal	Yes Last	4- 5 yrs	18 yrs Last

Sequence of development of paranasal sinus

M → Maxillary

E → Ethmoid

S → Sphenoid

F → Frontal

→ Maxillary sinus, Ethmoid sinus and Sphenoid sinus can be seen at birth on "**CT Scan**"

→ At time of birth frontal is indistinguishable from anterior ethmoids.

On CT scan Frontal sinus can be seen by 1 yr.

Anatomy Of Paranasal Sinuses

→ Bulla ethmoidal belongs to anterior ethmoids.

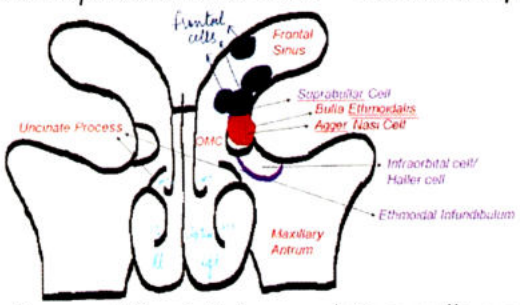
→ Together frontal, maxillary and anterior ethmoids drains in a space called as **Osteo – Meatal Complex.**

→ Uncinate process is a part of ethmoid bone and it creates a drainage pathway of maxillary sinus which is K/a **Ethmoidal Infundibulum**

→ There are cells above and beyond the bulla ethmoidals k/a suprabullar cell.

→ Cells anterior to bulla k/a Agar Nasi Cells

→ It is in close relation to the lacrimal bone.



The cells above the Agar Nasi they start blocking the drainage pathway of frontal sinus and these cells are k/a frontal cells.

There is classification of these frontal cells k/a "**Kuhn's Classification**":

Type I → A single cell above Agar Nasi

Type II → Multiple cells above Agar Nasi

Type III → Cells which goes into frontal sinus
Causes frontal headache (office head ache)

Type IV → Single loner cell deep inside frontal sinus

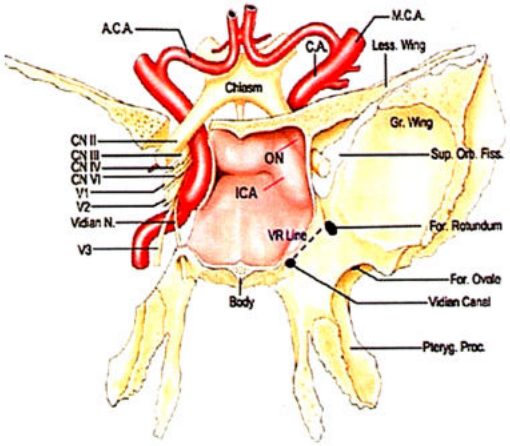
Anatomy of Sphenoid Sinus

→ Body of sphenoid bone is divided into 2 parts by inter sinus septum

→ In lateral wall of sphenoid sinus there is optic nerve, ICA.

→ Posterior most cell of ethmoid k/a onodi cell lies lateral to sphenoid sinus.

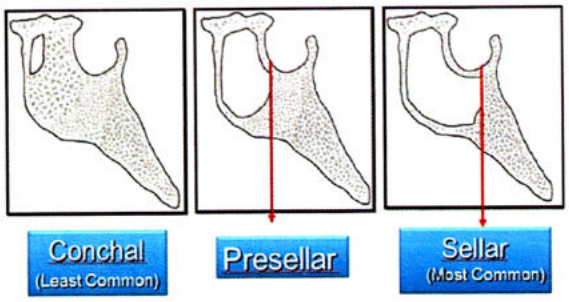
→ So it will lie b/w the sphenoid sinus and optic nerve and that can lead to a damage through optic nerve during surgery.



Sphenoid Pneumatization

4 Types

1. **Conchal Pneumatization** (Least Common Type)
   → Seen in 1-4 % individuals
2. **Presellar Pneumatization**
   The Pneumatization reaches upto anterior wall of sella.



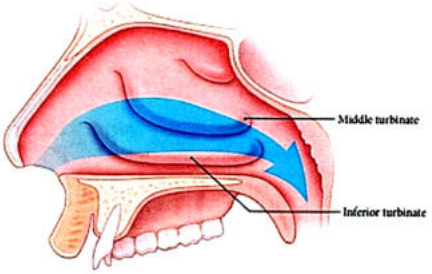
3. **Sellar Pneumatization/ Sphenoid Pneumatization (MC)**
   when Pneumatization goes through floor almost upto posterior wall
   → Seen in 54% cases
   → It is surgical importance during pituitary surgery.
4. **Mixed Pneumatization**

X Ray Of Paranasal Sinus

1. **Water's View** → Most common view done for Paranasal Sinuses.
   Aka occipito mental view (Nose Chin Position)
   → Best for Maxillary sinus and anterior ethmoids
2. **Pierre's View** → Water's view done with **open mouth**.
   → Sphenoid Sinus also seen
   → Posterior ethmoids are not seen in water's view both with open & close mouth.
3. **Caldwell's View** → aka occipito frontal (Nose forehead position).
   → Best for frontal & Ethmoidal sinus
4. **Lateral View** → All Sinus are visible

Airflow during inspiration to Nasal Cavity

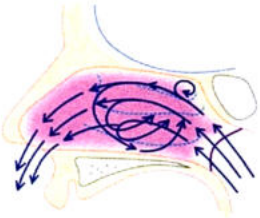
→ Max^{m} Air flow is through middle meatus is in a parabolic curve
→ During the flow of air in inspiration there is olfaction called as orthonasal olfaction which is not very developed due to missing of olfactory fossa



Airflow During Inspiration

Bernoulli's Phenomenon
Negative pressure in Nasal cavity due to lamellar flow

Air flow during expiration to nasal cavity
During expiration, this air flow is rerotated back and there is creation of Eddie current around the middle Turbinate → This help in Aeration of Para-nasal sinus as well as Retero nasal olfaction
Retronasal olfaction is also due to Cranial Nerve 5, 7, 9, 10.


Airflow During Expiration

Rhinosinusitis

1. **Acute Rhinosinusitis**
→ Mc causative agent: Rhino virus
→ Rx: Symptomatic
2. **Acute Bacterial Rhinosinusitis**
→ Mc cause: Streptococcus pneumoniae
→ Rx: Symptomatic + Antibiotics
3. **Chronic Rhinosinusitis**
→ > 12 weeks → Chronic Rhinosinusitis
→ Mc causative organism → Staph. Aureus

→ Rx → Culture directed Antibiotics + Nasal Decongestants (Rx of choice)

↓ Not responding

FESS (Functional endoscopic Sinus Surgery)

4. **Vasomotor Rhinosinusitis**

→ Due to increase parasympathetic discharge there is Non allergic Non infective perineal Rhinosinusitis (Naniper)

→ More common in emotional female

Rx → Vidian Neurectomy – Gold standard Treatment

5. **Rhinitis Medicamentosa**

→ Excess usage of Nasal decongestants for long period of duration which leads to **Rebound phenomenon**

→ Rx → Intra nasal corticosteroid spray (D.O.C)

6. **Allergic Rhinosinusitis**

M/c allergen → Carpet dust/ House dust mite

Symptoms:

Intermittent Symptoms (< 4days/ week or < 4 weeks) → Mild

Intermittent Symptoms (< 4days/ week or < 4 weeks) → Moderate - Severe

Persistent Symptoms (> 4 days/week & > 4 weeks)

On Examination

Signs

→ Allergic or Atopic facies- mouth breathing

→ Allergic Shiner and Denni morgan lines

→ Allergic salute → Nasal Crease

→ **Pale bluish hypertrophied mucosa**

→ Confirmed by **Skin Prick Test**

→ **Nasal Allergen challenge/ Provocation test/Inhalation test (Gold Standard)**

- Not commonly done

→ Rx → Mild – Intermittent → 2nd generation Non sedative Antihistaminic

- Moderate to severe/ Persistent → Intranasal steroids spray is given and if not responds to pharmacotherapy, immunotherapy is given.

Nasal Polyps

Antrochoanal Polyp (AC Polyp)	**Ethmoidal Polyp**
Starts from Maxillary antrum and goes up to choana	Starts from ethmoidal air cells and goes to Nares
Single, large, Unilateral	Small, multiple, bilateral
Grows posteriorly towards choana	Comes out anteriorly
Seen in children	Seen in Adults

Polyps with Syndrome

1. **Samter's Traid** → Asthma + Aspirin intolerance + Polyps
2. **kartagener Syndrome** → Situs Inversus + Bronchiectasis + Polyp
3. **Young Syndrome** → Azoospermia + Bronchiectasis + Polyps
4. **Churg strauss Syndrome** → Eosinophilic granulomatosis with Polyangiitis
5. **Nares** → Non allergic Rhinitis with Eosinophilia also has Polyps
6. **Allergic fungal Rhinosinusitis (AFRS)** → also has Nasal Polyps

Atrophic Rhinosinusitis (ozaena)

→ Causative agents → **Klebsiella ozaenae (Perez bacillus)**

→ Young Females of poor socio economic status are affected

- Iron deficiency
- Multi Vitamin (Vitamin D) deficiency

→ Pseudostratified Ciliated columnar epithelium is converted to Stratified squamous epithelium

→ Mc complaint → Nasal obstruction [crust formation]

- foul smell
- Anosmia → Merciful Anosmia

Rx

1. Alkaline Nasal Douching [Nacl + $NaHco_3$ + Na biborate] → Crust softens Klebsiella does not grow
2. 25% Glucose in Glycerin (Hygroscopic effect)

   ↓

   Nourishes the columnar cells
3. Antibiotics
4. Multivitamins (Vitamin D), Fe supplements
5. Estrogen Spray
6. Kemicetine/ Anti ozaena solution
   → Chloromycetin/ Chloramphenicol
   → Vitamin D
   → Estradiol

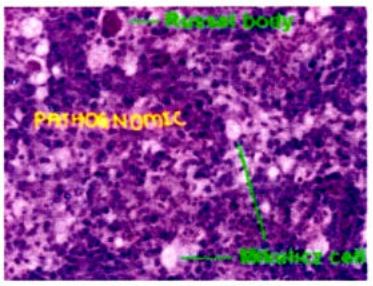
6 Sx → **Young's Operations** → alternative closure of each nasal cavity for 6 months
Modified Young's Operations → Partially closing both Nasal Cavities.

Rhinoscleroma (Respiratory Scleroma) aka hebra nose

→ Caused by Klebsiella rhinoscleromatis (Frisch Bacillus)

Stages

1. Stage of Atrophy (Behaves like Atrophic Rhinitis)
2. Stage of Granuloma formation
   On histopathological examination **Mukulicz cells and Russel bodies** are seen which are diagnostic of Rhinoscleroma

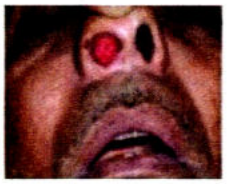


3. Stage of Sclerosis/ fibrosis/ Cicatrization
   → Woody Nose/ Hebra Nose/ Tapir Nose
   → Woody nose formed in stage 3
   → Woody induration initially seen in stage 2

Doc → Rifampicin

Rx of choice → Laser Excision + Base Electro cautery
→ Rhino Scleroma → Disease of Resp. Epithelium
→ New Name – Respiratory Scleroma

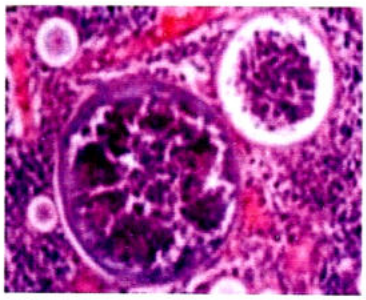
Rhinosporidiosis



→ Caused by Rhinosporidium Seeberi (It is an Aquatic Protozoa)

- Affects only mucosal surfaces

→ Seen in southern Eastern coast of India, Srilanka & Bangladesh mainly in rural population (Pond Bathing) → Cross infection from animals



→ **Strawberry Granuloma** is seen
→ Humans are accidental host
  - On HPE – Multiple thick walled sporangia

DOC → Amphotericin B/ Dapsone
Rx of choice → Laser Excision + Base electro cautery (Fungal Rhino sinusitis)

Fungal Rhino Sinusitis

Fungal Ball/ Mycetoma/ Aspergilloma

→ **MC** → Aspergillus Fumigates
→ No invasion, No reaction from nasal mucosa
Rx → Evacuation with FESS

Allergic Fungal Rhinosinusitis (AFRS)

→ Causative agent - Bipolaris (M.C)

Criteria

Bent and Kuhn diagnostic criteria

Major	Minor
Type I Hypersensitivity	Asthma
Characteristic CT finding	Unilateral disease
Eosinophilic mucin without invasion	Bone erosion
Positive fungal stain	Fungal Cultures
Nasal polyposis	Charcot leyden crystals
	Serum eosinophilia

CT Findings

Double Dense Appearance/ Heterogeneous appearance is most characteristic features of Allergic fungal Rhinosinusitis

→ It is due to calcium deposits in fungal hyphae
→ Generally it unilateral but may be Bilateral
→ Bone erosion are seen without invasion

Treatment

→ Functional endoscopic sinus surgery and removal of fungai **Double Dense appearance**
→ Steroids
→ Immunotherapy

Rhino Cerebral Mucormycosis

→ Caused by Mucor/Rhizopus it is saprophytic organism
→ Acute invasive fulminant fungal disease
→ Seen in Immuno compromised patient especially in Diabetic Patients
→ Necrotizing Vasculitis present

C/C

→ Sudden Blindness
→ Stroke

DOC → Amphoterecin B
→ Surgical Debridement
→ Diabetic Control

Tumors of Nose and Paranasal Sinus

Osteoma

→ Mc benign tumor of PNS

→ Mc site is frontal sinus > Ethmoidal sinus

Rx is endoscopic excision

Inverted Papilloma/ Ringertz Tumor

→ mc Benign tumor of Nasal Cavity

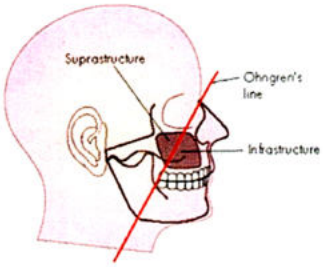
→ Arises from Schneiderian Membrane (specially epithelium on lateral wall) transitional cells epithelium

HPE – Shows Infolding inverted papilloma (IOC).

Rx

→ Endoscopic Endonasal Excision → Rx of choice

→ High rate of Recurrence – more the recurrence, more the chances of squamous cell CA (- 10% can harbor squamous cell carcinoma)



Ohngren's Line

Squamous Cell CA

→ Mc Para nasal sinus involved → maxillary Sinus

→ Mc malignancy of PNS & Nasal Cavity

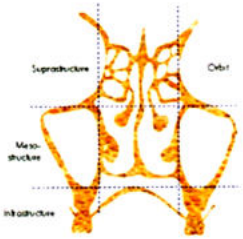
Classification

→ **Ohngren's line** → from canthus to angle of mandible dividing it into supra and Infra structure

→ Supra structure malignancy → Bad prognosis

→ **Lederman's classification** uses two lines of Sibileau divide into 3 parts

- Supra structure
- Meso. Structure
- Infra structure



Lederman's Classification (Lines of Sebileau)

TNM Classification

T_1 → Involvement of Sinus mucosa

T_2 → Involvement of Bone except superior wall of max. Sinus

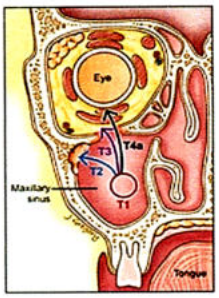
T_3 → Involves the superior wall or ethmoidal air cells or into subcutaneous tissue

T_4 → Involves the eye ball, orbit, skin

Rx → Sx + Radiotherapy stage

→ T1 – T2 stage, surgery only

→ T3 – T4 a, b, surgery + radiotherapy



TNM

Facial trauma

→ Mc bone to undergo fracture in facial trauma → Nasal Bone

- Open book deformity
- Closed book deformity

Rx → Closed reduction, either done immediately i.e. before the onset of edema or after 4 – 5 days (When the edema subsides).

Zygomatic fracture/ Tripod fracture

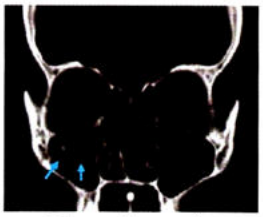
→ 2nd most fractured bone in facial trauma

→ Now it is known as Quadripod fracture

→ Malar prominence is lost a/k/a **step deformity**

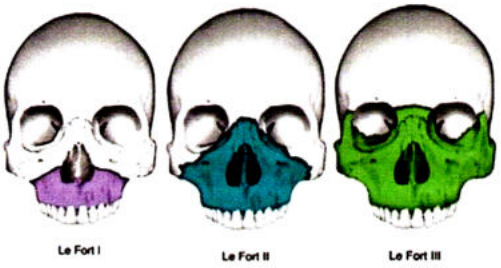
Rx → Open reduction internal fixation

Blow Out Fracture


Tear drop sign

- → Weakest wall of orbit is floor of orbit.
- → Orbital contents hangs in max. antrum is **Tear Drop Sign**
- → There is entrapment of Inferior rectus muscle so Patient not able to look up confirmed it by forced duction test.
- → Treatment mesh application and reduction of contents

Maxillary fracture/ Le Fort fracture



- → **LeFort I fracture**/ Transverse fracture (floating Palate Seen)
  - Fracture line runs parallel to hard palate
- → **LeFort II**/ Pyramidal Fracture (Floating maxilla is seen)
- → **Lefort III**/ Cranio Facial Dysjunction
  - Most dangerous associated with maximum morbidity
  - Lower part forms **Pigs snout Deformity**
- → Treatment of LeFort Fracture is **open reduction internal fixation**

CSF Rhinorrhea

- → M/C/C → head injury
- → M/c site → fracture cribriform plate of ethmoid
- → Clear watery discharge
  - ↑ on coughing, straining, sneezing, bending forward → **Tea Pot Sign/ Reservoir Sign**

Test to differentiate Mucosal discharge from CNS rhinorrhea

- → **Sniff Test** → can not sniff back CSF
- → **Handkerchief Test** → not stiffens
- → **Glucose Test** → 2/3rd of N Blood Glucose level
  → 40-60 mg/dL
- → In case of blood mixed with CSF

1. **Halo Sign/ Double Ring Sign/ Target Sign**
2. **β_2Transferrin test**
   - Gold Standard for CSF
   - Only found in CSF

- → **Gold Standard investigation in CSF Rhinorrhoea**

1. HRCT of Nose & PNS (to find site of leak) → **1st inv/IOC**
2. MRI – T_2weighted images [MR - Cisternography] → can't see both fracture and the CSF at once
3. CT cisternography → can see both fracture and the CSF (most specific Investigation)

Management

Conservative Mx

- → Prophylactic Antibiotics
- → Acetazolamide & Wait for 7 days
- → Stool softeners, anti tussive
- → No relief → Lumbar Drain for 2 weeks (Total 3 weeks)
- → No relief → Endoscopic Repair

Spontaneous CSF Rhinorrhea

- → Do MRI, rule out any cause of ICT
- → Endoscopic Repair

Paradoxical CSF Rhinorrhea

→ Fracture in temporal bone (petrous part)

→ Can cause CSF Otorrhea also

Rx →Treatment of fracture of Temporal Bone

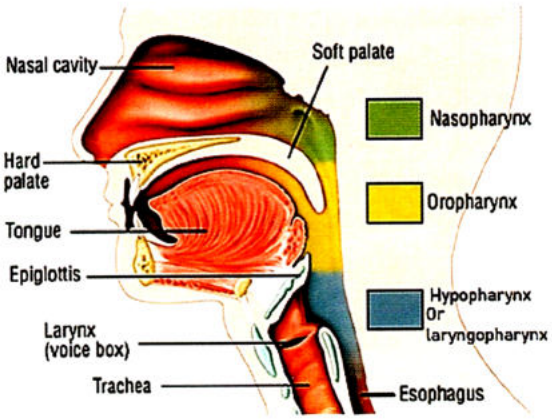
Pharynx

Extends from skull base to the Esophagus

Nasopharynx → From skull base to hard palate

Oropharynx → From hard palate to hyoid bone

Hypopharynx → From hyoid bone to esophagus



Nasopharynx/ Epipharynx

Eustachian Tube

→ Opens on lateral wall

→ Torus Tubarius – Cartilaginous Protrusions

Adenoids

→ Lymphoid tissue on posterolateral wall

→ Nasopharyngeal bursa → midline recess represents the attachment of Notocord in embryonic life **Pharyngeal chordoma** arises from here (M.C. Site). Blockage of Nasopharyngeal bursa leads to collection of secretion in it and forms **Thornwaldt's Disease.**

Rathke's Pouch

→ Place from where pharyngeal mucosa invaginates to form pituitary gland

→ Craniopharyngioma arises from here

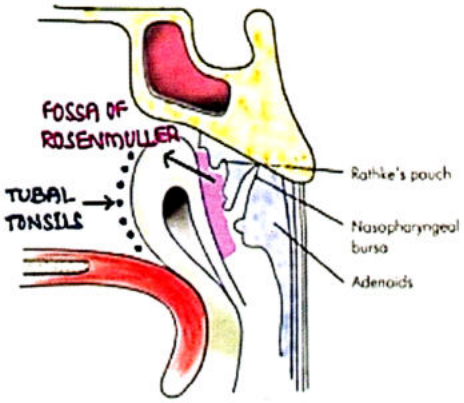
Fossa of Rosenmuller

→ Blind recess in posterior superior to Eustachian tube openir

→ Mc site for origin of Nasopharyngeal carcinoma

Tubal Tonsils of Gerlach

→ Lymphoid nodules around ET

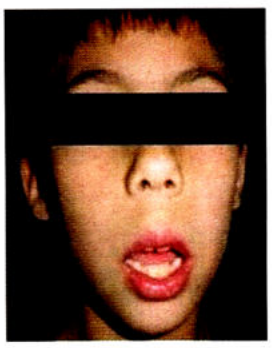


Diseases of Nasopharynx

Chronic Adenoiditis/ Chronic Adenoid Hypertrophy

→ 3 – 5 years → Growth Starts
5 – 7 years → Max. growth
> 7 years → Growth ↓

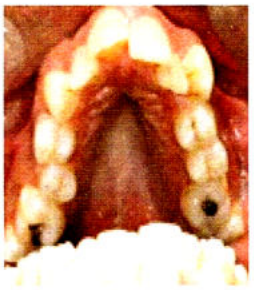
} Normal Physiological growth pattern of adenoids



Elongated face with open Mouth pinched nose

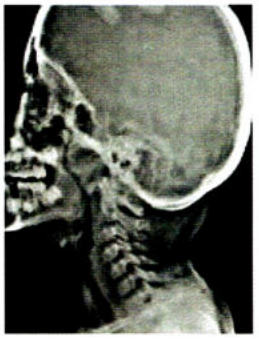
Clinical Features

→ B/L Nasal obstruction – Mouth breath → High Arched Palate
→ Adenoid Facies



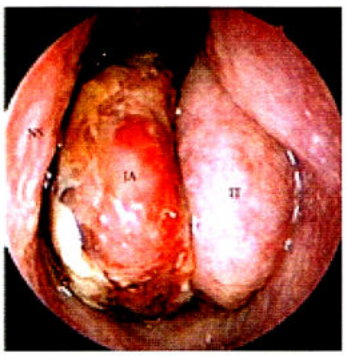
High Arched Palate
Overcrowding of anterior teeth

X – Ray
→ Mass arising from Postero-superior wall of Nasopharynx and Completely blocked Nasopharynx
Rx Management: Adenoidectomy



Indications of Adenoidectomy

1. Obstruction
2. Adenoid facies
3. B/L serous otitis media is seen in some patients
4. Recurrent attack of acute otitis media.
5. CSOM Associated with chronic adenoiditis
6. Recurrent & Rhino Sinusitis

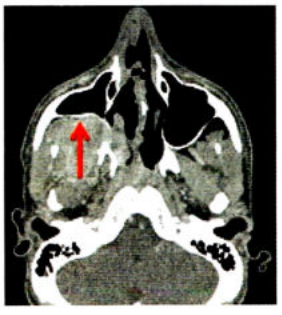


Juvenile Nasopharyngeal Angiofibroma/ JNA
→ mc benign tumor of nasopharynx
→ Site of origin → Lateral nasal wall near spheno palatine foramen & pterygoid base
→ Vascular Tumor
- mc blood supply of JAF → Sphenopalatine Artery (br. Of Internal max. A).

→ Exclusively seen in adolescent males (Androgen dependent tumor) → 8-22 years
→ No tunica media M/C presentation profuse recurrent epistaxis → anemia
- Nasal obstruction⊕
- Swelling in Cheek ⊕

Session Staging

Stage i → Limited to Nasal Cavity/ Nasopharynx
Stage ib → Extension into one or more Paranasal Sinuses.
Stage ii a → Lateral extension goes in Pterygopalatine fossa.
Stage ii b → Antral sign or extension into orbit
Stage ii C → Goes in Infra temporal fossa
Stage iii → Goes intra cranially



HOLMANN MILLER SIGN

Dx
→ CECT (I.O.C)
- Anterior bowing of posterior maxillary wall → ANTRA /HOLMANN MILLER SIGN
- Widening of pterygo palatine tunnel → HONDUSA SIGN

Rx of choice – Endoscopic excision

Nasopharyngeal Carcinoma (NPC)

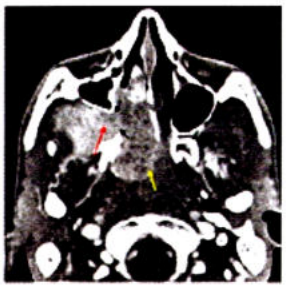
→ Male, 8 – 12 years & 60 – 70 years

→ Common in Chinese

- EBV association

→ Mc site → fossa of Rosenmuller

→ Mc type → sq. cell CA (> 85%)



HONDUSA SIGN

Presentation

→ Mc - Cervical Lymph Node Metastasis → associated with good prognosis

→ U/L serous otitis media → u/l conductive hearing loss

→ Multiple cranial nerve palsy (earliest – 6th nerve) (OI factory N. spared)

- 5th CN involvement → ipsilateral Trigeminal Neuralgia
- 10th CN involvent → ipsilateral palatal palsy

} Trotter's Traid or sinus of morgagni Syndrome

TNM Staging

T_1 → Soft tissue involve of Nasopharynx/ Nose/ Oropharynx

T_2 → Involvement of parapharyngeal space

T_3 → Skull Base involvement/ involves Para- nasal sinus

T_4 → Intracranial extension

N_1 → Tumour < 6 cm any one side

N_2 → < 6 B/L

N_3a → > 6 cm (Single Node)

N_3b → Tumor in ho's triangle or supraclavicular fossa

Treatment of NPC

Radio sensitive Tumor: Radio therapy is mainstay t/t in all stages.

Stage 1 & Early Stage 2 → Radiotherapy

Stage 2 late, stage 3 & 4 → Chemo radiation

Prognostic Markers → IgA/VCA & IgA/EA

(VCA: Viral Capsid Antigen; EA: Early Antigen of EBV)

IgA/ vCA: Serological screening of NPC

OROPHARYNX

Palatine Tonsils

→ B/L

→ Covered by fibrous capsule & forms crypts

→ Tonsillar bed formed by superior constrictor muscle

→ Peri Tonsillar space → b/w fibrous capsule & sup. Constrictor muscle

Infection of Peritonsillar Space → Peritonsillar abscess/ Quinsy

Present with

- Pain in throat
- Fever
- Dysphagia, Odynophagia

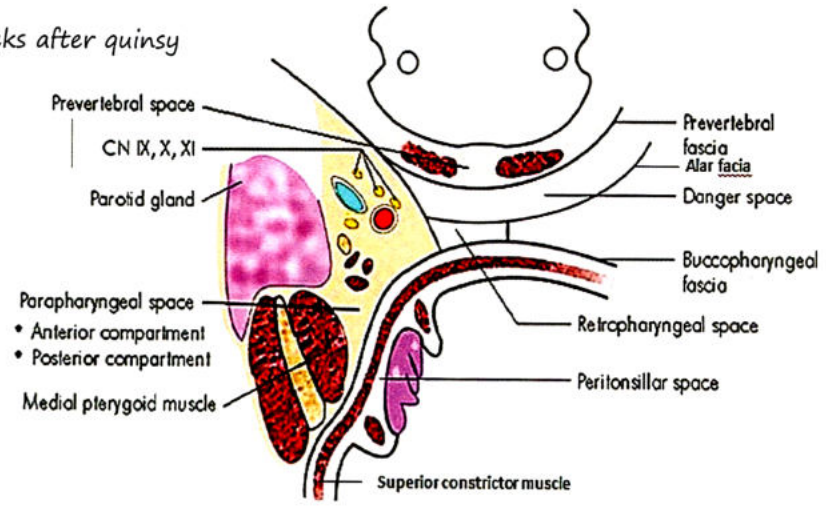
O/E:

- Red enlarged congested tonsil pushing soft palate
- Hot potato voice / Muffled voice

Treatment

1. Incision & Drainage
2. Antibiotic iv
3. Interval Tonsillectomy done 6 weeks after quinsy

DEEP NECK SPACES



Prevertebral space: Space between Vertebra & Pre vertebral fascia. Infection is called as Prevertebral space abscess. It forms a midline swelling in Post Pharyngeal wall.

→ **Danger space:** Space between Pre vertebral fascia and alar fascia. The Infection from here quickly spread to the mediastinum.

Retropharyngeal Space It is space between Buccopharyngeal fascia and Alar fascia it formed by condensation of buccopharyngeal fascia in midline that leads to formation of Retropharyngeal space on both sides.
Abcess in Reteropharyngeal space will be on posterior pharyngeal wall and it may be on Right side or left side

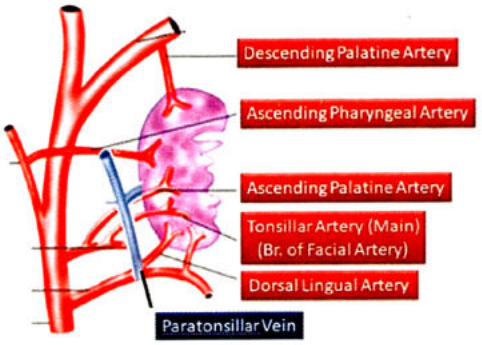
Parapharyngeal Space

Medial Boundary – Palatine Tonsils, Superior Constrictor muscle, buccopharyngeal fascia
Posteriorly: Pre Vertebral fasia
Lateral: Mandible, medial Pterygoid muscle, Parotid Gland.
If there is Deep lobe Parotid Gland Tumor, it will come in parapharyngeal space.
Parapharyngeal space is divided into 2 compartment.

- Anterior Compartment and posterior compartment
- Anterior compartment has only fat, adipose tissue
- Posterior compartment has important Neurovascular structure Such as internal Juglar vein, Interval Carotid artery, X Cranial Nerve in Carotid Sheath IX, XI, XII cranial Nerve

Anterior compartment abscess → it will push tonsil medially
→ Tonsils are enlarged and they reach midline → Quinsy
→ Tonsils are pushed to midline → Δsis Parapharyngeal space abscess
→ Parapharyngeal space abscess can have Trismus because of involvent of medial Pterygoid muscle.
→ Generally seen in Anterior compartment
Quinsy can also spread to Parapharyngeal space also cause trismus by involving medial Pterygoid muscle.

Blood Supply of Tonsils



Tonsils are supplied by S. Arteries.

i. Tonsillar Artery (main) → Branch of Facial Arteries
ii. Ascending Palantine Artery
iii. Descending Palantine Artery
iv. Ascending Pharyngeal Artery
V. Dorsal Lingual Artery

Venous drainage of tonsillitis by Para tonsillar vein

Acute Tonsillitis

1. **Acute Catarrhal Tonsillitis** When there is viral infection, tonsils are red, congested but they are inside tonsillar fossa.
2. **Acute follicular Tonsillitis** Due to Bacterial infections, Bacteria goes in crypts and there is presence of Pus in crypts openings.
3. **Acute Parenchymal Tonsillitis** Infection in Parenchyma which result in Tonsillar Hypertrophy Grade 4 Tonsillar Hyper trophy is called Kissing Tonsils
4. **Acute Membranous Tonsillitis** When Pus in Crypt form a Membrane over tonsils.

Causes:

1. Acute Tonsillitis
2. Faucial Diphtheria
   → Seen in child 2 – 3 year age, unimmunized

C/C

→ Fever
→ Throat Pain
→ Bull neck → B/L Cervical Lymphadenopathy
Psudomembrane over tonsils – Bleeds on Peeling, dirty Greyish membrane

3. **Infectious Mononucleosis:** Similar Appearance is also seen → Age 20 – 30years
   - B/L cervical lymphadenopathy
   - Palatal petechiae
4. **Candidiasis** Immunocompromised/ on inhalational steroids
5. **Vincent Angina** is caused by Borrelia Vincentii (Trench Mouth)
6. **Aphthous Ulcers** – Painful, red margins

Chronic Tonsillitis

Irwin Moore Sign

→ Press the anterior tonsillar Pillar, Pus come out of crypts shows there is chronic Bacterial infections inside the tonsils

Management

→ Tonsillectomy
→ Position used – Rose Position
→ Cervical Joint & Thoraco – Cervical, Joint, both are in extension

Indications of Tonsillectomy

1. Recurrent sore Throat:
a. ≥ 7 episodes/ 1 year

b. ≥ 5 episodes/ year for 2 years
c. ≥ 3 episodes/ year for last 3 years

2. **Quinsy**: Single episode in child
   - 2nd episode in adult
3. Obstruction
4. Febrile seizures
5. Biopsy for Malignancy

Coblation Tonsillectomy (In which radio frequency is used to ionize the Sodium Chloride)
Bloodless and Painless

Complication of Tonsillectomy

M.C complication – Hemorrhage

3 Types

→ Primary (Intra operative): M.C source of Primary Haemorrhage is Paratonsillar Vein. It is controlled by suture Ligation

→ Reactionary (Within 24 hours): Reactionary Haemorrhage: because of Slippage of suture **Rx:** Repeat Ligation. Most dangerous type of Haemorrhage

Secondary (5 – 7 days): Due to Secondary Infection **Rx:** I/V Antibiotics

2. Pain in ear: due to glossopharyngeal Nerve involvement in tonsillar fossa

Hypopharynx/ Laryngopharynx

→ Wall is made up of inferior constrictor muscle consisting of Thyropharyngeus and Cricopharyngeus

→ Thyropharyngeous is supplied by Superior laryngeal nerve

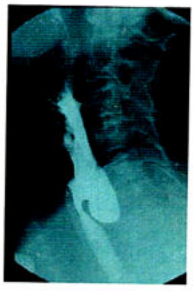
→ Cricopharyngeus is Supplied by Recurrent laryngeal Nerve

→ Oblique fibres of thyropharyngeus and Transverse fibres of Cricopharyngeus form a Δ space → Killians Dehiscence/ Gateway of Tears
M.C site for Oesopharyngeal Perforation

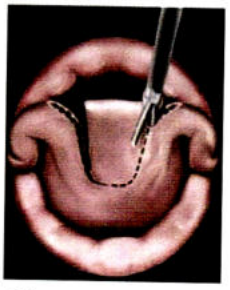
Zenkers/ Pulsion Diverticulum

M/C Site: Killian's Dehiscence

Presents with Dysphagia, Halitosis, Regurgitation of old eaten food.
It is Pseudo diverticulum



Barium Swallow



Dohlman's Operation

Investigation of choice → Barium Swallow

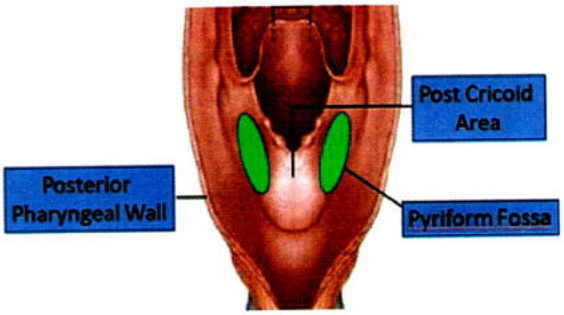
Treatment

Treatment of Choice → (Endoscopic Diathermy/ Dohlman's Operation)

Hypopharynx

3 components

1. Posterior Pharyngeal
2. Post Cricoid Area
3. Pyriform Fossa



Plummer Vinson/ Patterson Brown Kelly syndrome

→ Post Cricoid Webs
→ Iron Deficiency Anemia
→ koilonychia

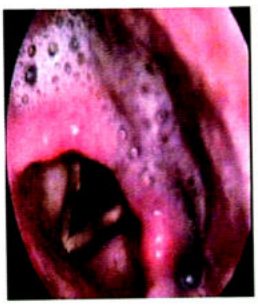
Post Cricoid Webs lead to development of Squamous Cell Carcinoma

Post Cricoid Carcinoma

2 Signs

1. **Moure/ Broca's Sign** – Move the larynx with hand, Crepitus is felt normally. But in Post Cricoid Carcinoma Crepitus ⊖nt → Muir's Crackle ⊖ nt
2. **Chevalier Jackson's Sign** – Swelling in Pyriform fossa

Moure /Broca's Sign



Chevalier Jackson's Sign

Larynx

Embryology of larynx

Larynx is formed by 2 arches –

→ 4th Branchial arch and 6th Branchial arch

4th Branchial arch:

Nerve supply: Superior laryngeal Nerve branch of vagus nerve.

It supply cricothyroid muscle – only intrinsic muscle of larynx which is supplied by superior laryngeal nerve

4th Branchial arch + 3rd Branchial arch they together they form a Hypobranchial eminence.

Hypobranchial eminence form posterior 1/3rd of tongue and epiglottis

4th branchial arch also forms thyroid, corniculate, cuneiform cartilage

6th Branchial arch

Nerve supply: Recurrent laryngeal nerve branch of vagus nerve

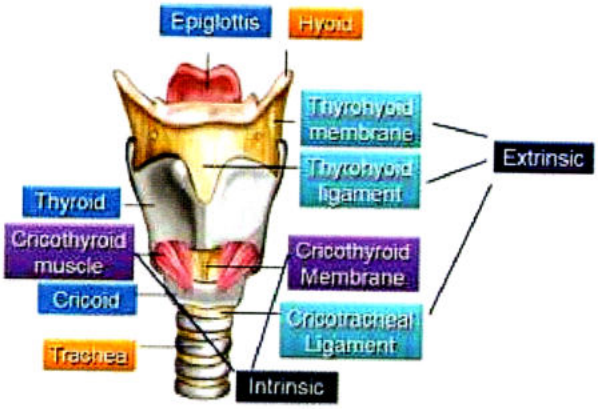
→ It supplies all intrinsic muscle except cricothyroid muscle.

6th Branchial arch forms the cricoid and arytenoid cartilag

Cartilages of larynx

3 unpaired cartilage	3 paired cartilage
Thyroid	Arytenoid
Epiglottis	Corniculate
Cricoid	Cuneiform

Front view of larynx



→ Any muscle / Membrane/ ligament that is present in b/w 2 cartilage → Intrinsic

Any muscle/ membrane/ligament that connects cartilage to external surface = Extrinsic

Thyroid membrane
Thyrohyoid ligament
Cricotracheal ligament
} extrinsic structure

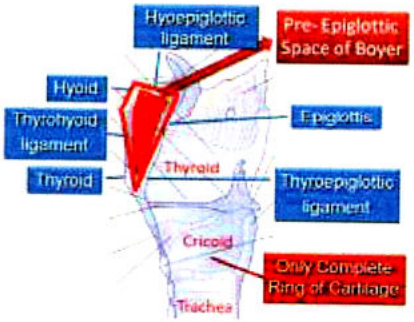
Cricothyroid membrane
Cricothyroid muscle
Cricothyroid ligament
} Intrinsic Structure

Cricothyroid in only intrinsic muscle of larynx which lies on external surface of larynx

Cross section of larynx

→ Cricoid cartilage – only complete ring of cartilage in whole human airway

→ Anterior to epiglottic there is space k/a pre epiglottic space of Boyer

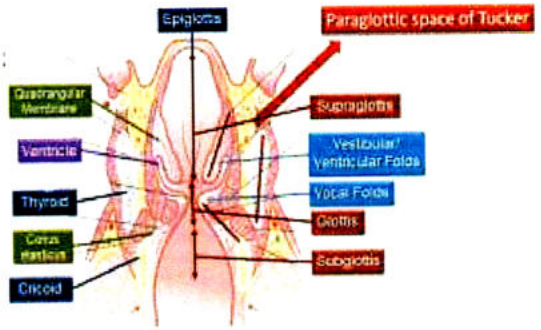


Boundaries:

Superior: Hyoepiglottic ligament
Posterior: Epiglottis and thyroepiglottic ligament
Anteriorly: Body of Hyoid, Thyrohyoid ligament and small part of thyroid cartilage
Space is open on 2 sides and communicate with paraglottic space

3 Parts of Larynx:

1. Glottis: part that contain vocal folds
2. Supraglottis: part above the glottis
3. Sub Glottis is formed by a complete ring of cricoids cartilage.
   In supraglottis there is vestibular/ **ventricular folds**
   They are called ventricular folds because they enclose a space k/a ventricle.



→ Ventricle is only true space of larynx Rest all are potential spaces.
Inside Vestibular folds there is membrane of k/a Quadrangular membrane.

Boundaries of paraglottic space

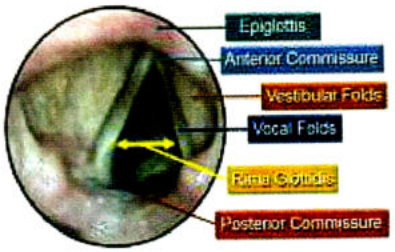
Anterior lateral: Thyroid cartilage
Superomedial: Quadrangular membrane
Antero medial boundary – conus elasticus
Any Malignancy seen in Paraglottic space of Tucker is Transglottic Malignancy and is considered as t3 stage

Indirect vs direct laryngoscopy

Image: virtual, smaller and inverted	Actual larynx seen
Ease of procedure can be done in OPD	Requires OT
Phonation possible (vocal fold movements can be seen)	Not Possible (movements cannot be seen)
Patient cooperation is required (cannot be done in children)	Not required (can be done)
Additional procedures like Biopsy cannot be done	Can be done

Indirect larygoscopy

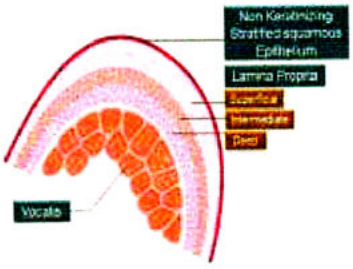
→ Glottis is Narrowest part of airway
however, in Neonates subglottis is Narrowest
In Neonates the larynx is funnel shaped,
so subglottis is Narrowest



Layers of vocal folds

layers of vocal fold

i Non keratinizing stratified squamous epithelium shines, reflects back light, stands out so vocal fold are looking different from remaining tissues
ii **Lamina propria**



Divided In 3 further layers:

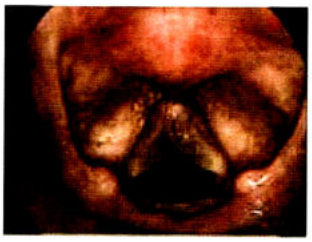
- Superficial
- Intermedial
- Deep

iii. Vocalis Muscle

Superficial layer of lamina propria → k/a Reinke's space

Edema of Reinke's space – Reinke's oedema

Intermediate and deep layers combines to form vocal ligament.



Laryngeal Inlet

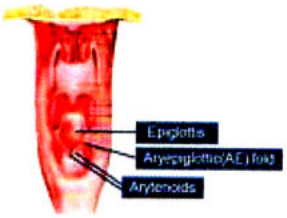
Reinke's oedema

Boundaries

Anteriorly – Epiglottis

Posteriorly – arytenoids cartilage

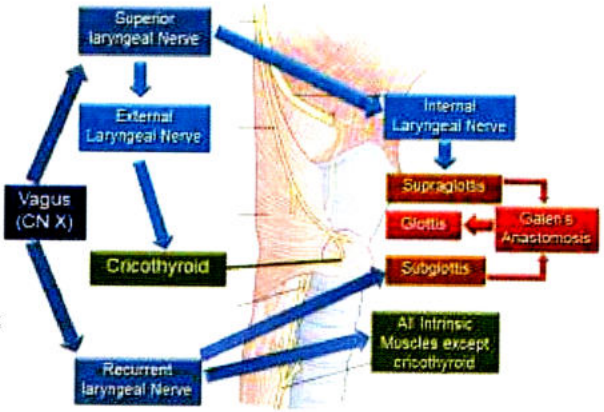
Aryepiglottic fold in between



Laryngeal Inlet

Nerve supply of larynx

Larynx is supplied by vagus verve (X C.N)



2 Terminal branches

1. Superior laryngeal nerve (SLN)
2. Recurrent laryngeal nerve (RLN)

Superior laryngeal nerve further divides into 2 Branches

- External laryngeal nerve
- Internal laryngeal nerve

External laryngeal nerve supplies cricothyroid muscle

(Only intrinsic muscle, supplied by External laryngeal Nerve)

→ Internal laryngeal nerve give sensory supply to supraglottis

Recurrent laryngeal nerve supplies all intrinsic muscles except **cricothyroid**.

- Also give sensory supply to subglottis
- At level of glottis internal laryngeal nerve from supraglottis & recurrent laryngeal nerve from subglottis both comes & from **Galen's anastomosis**

Galen's Anastomosis supply glottis

Muscles of Larynx

All the muscles are adductors except posterior cricoarytenoid (only abductor of Larynx)

Safety muscle of Larynx: Posterior cricoarytenoid

Tensor of vocal folds: Cricothyroid

Relaxer of vocal fold: vocalis (medial fibres of thyroarytenoid)

Diseases of Larynx

1. **Laryngomalacia**

→ **Congenital lesions of larynx**

→ M.C congenital lesion: Laryngomalacia

→ Child is born with soft cartilages.

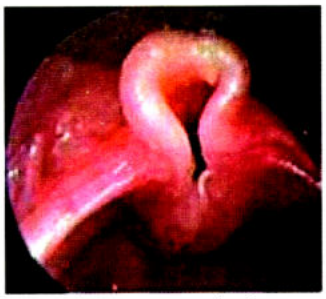
→ Inspiratory stridor at Birth (Improves in Prone position)

→ Cry is Normal as expiration is normal

O/E– omega shaped epiglottis

→ RX Conservative Treatment

→ Disappears by 2 yrs of age



Laryngomalacia

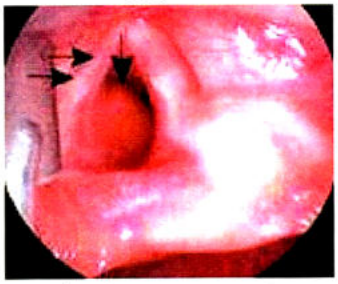
2. **Sub Glottic haemangioma**

→ C/F: Inspiratory stridor at 3-6 months of age

50% of patients have cutaneous haemangiomas

Rx - Tracheostomy.

- Co_2 laser excision
- Injection of steroids



Sub Glottic hemangioma

3. **Laryngeal Web's**

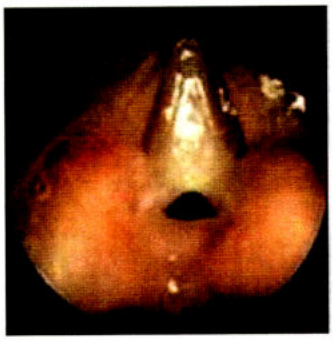
Membrane formed due to incomplete canalization of airway

C/F: Biphasic stridor from birth

Stridor	
supra glottis	- inspiratory stridor
Glottis & subglottic	- Biphasic stridor
trachea & bronchi	- expiratory stridor

M/C Site = Anterior Glottis (1/3rd)

Rx CO_2 laser Excision



Infections of Larynx

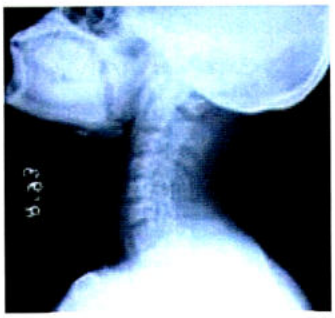
1. **Acute epiglottitis**/ Acute supraglottitis

M.C causative organisms → Haemophilus influenza B

C/C: Fever, Sorethroat, dysphagia, Dyspnoea.

O/E: Tripod sign/ Rising sun sign

Tripod sign



Steeple sign

IOC : X Ray Lateral View : Thumb sign

Rx: I/V Antibiotics

Ceftriaxone (D.O.C)

Steroid Nebulisation

2. **Acute laryngo Tracheo Bronchitis** (Croup)

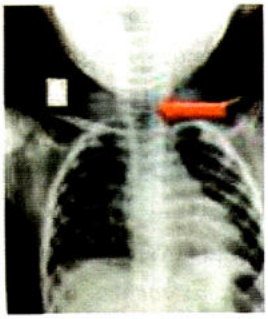
Viral infection caused by parainfluenza type 1 & 2

C/C: fever, Barking cough (croup)

Stridor: Initially it is inspiratory later on become biphasic, some have expiratory stridor.

IOC X-ray Steeple Sign:

Rx Symptomatic



Steeple sign

3. **Laryngeal T.B**

- Always associated with pulmonary T.B
- submucosal nodules.

Signs:

1. Hyperemia of vocal folds along with incomplete adduction (first/ earliest sign)
2. Mammillated appearance
3. Mouse nibbled appearance
4. Turban epiglottis
   (Pseudo edema of epiglottis)
   Rx ATT

Vocal fold palsy

1. U/L external laryngeal nerve palsy:
   M.C Cause: Thyroid surgery
   M.C Nerve Injured in thyroid surgery is external laryngeal nerve
   - There is loss of pitch
   Rx: **Conservative**

2. **U/L Recurrent laryngeal nerve palsy**
   - Right is M.C Injured during thyroid surgery
   - Left is M.C injured during cardio-thoracic surgery as it has longer course in thorax

O/C – hoarseness
Vocal ford lie in Paramedian position
Rx: conservative

3. **U/L X cranial Nerve palsy**
   (ELN + ILN + RLN) palsy
   → M.C cause = Surgery
   C/C – hoarseness, Aspiration
   Vocal folds lie in cadaveric position
   Rx Thyroplasty Type I
4. **B/L recurrent LN palsy**
   M.C cause: Thyroid surgery
   C/C: Stridor but voice Quality is good
   Both vocal folds lie in paramedian position
   ↓
   So airway is inadequate
   A/k/as B/L Abductor cord Palsy
   Rx Type 2 Thyroplasty
5. **B/L superior Laryngeal Nerve Palsy**
   - MC cause Thyroid surgery
   - C/C: severe Aspiration

   Gold standard Rx. Intractable aspiration → Tracheal separation & Permanent Tracheostomy

Thyroplasty

Given by Isshiki

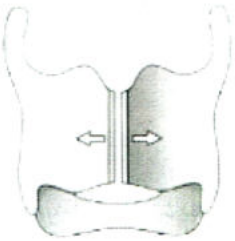
Type I Thyroplasty: a/k/as medialization/ Approximation

Done in RLN + SLN palsy

Type I Thyroplasty

Type II **Thyroplasty**: a/k/as lateralization/ Expansion

→ Done is B/L RLN Palsy



Type II Thyroplasty

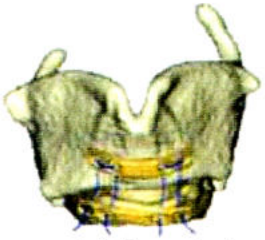
Type III **Thyroplasty**: a/k/as shortening/ Relaxation

Done in puberphonia

Type III Thyroplasty

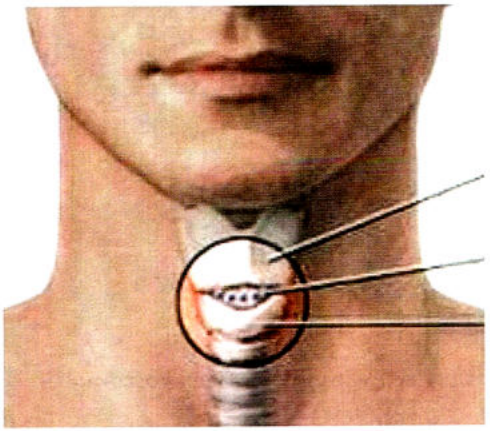
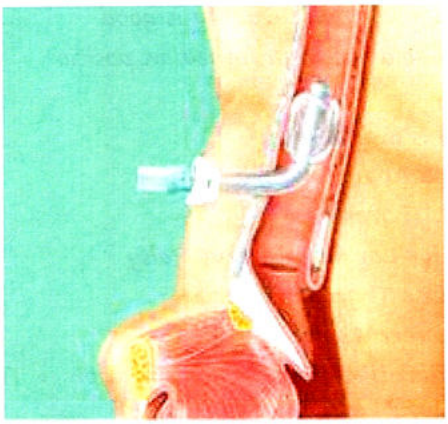
Type IV **Thyroplasty**: a/k/as lengthening / tensioning thyroplasty

→ Done in Androphonia



Type IV Thyroplasty

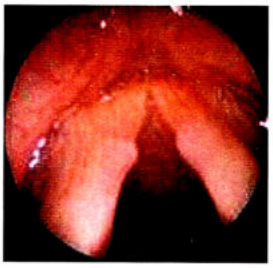
Cricothyrotomy vs Tracheostomy

Cricothyrotomy	Tracheostomy
Done in emergency	Done in emergency / planned
Outside the hospital	In the hospital
Incision is given in cricothyroid membrane	Opening b/w 2nd and 3rd Tracheal ring
	

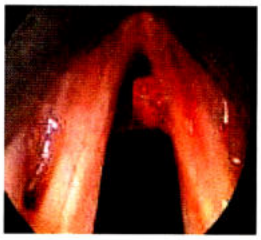
High Tracheostomy

Tracheostomy done B/W 1st and 2nd Tracheal ring
Done in Ca larynx

Benign lesions of larynx

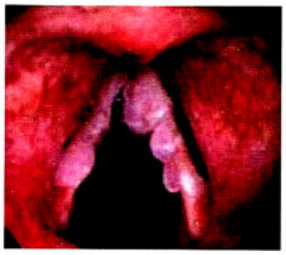


1. **Vocal Nodule / singer's nodules/ screamer's nodule.**
   - It is due to chronic misuse of voice
     C/C – Hoarsness of voice
     O/E: B/L symmetrical,
     Vocal nodule is sessile on free margin
     It is present at Junction of Anterior 1/3rd and posterior 2/3rd (area of maximum vibration)
     Rx speech therapy
   - Proton pump inhibitors



Vocal polyp

2. **Vocal polyp**
   It is due to sudden vocal abuse
   C/C hoarseness, Diplophonia
   O/E: U/L Pedunculated, mobile at
   Anterior 1/3rd and posterior 2/3rd of vocal cords
   Rx: Microlaryngeal surgery



JORRP

3. **Juvenile onset Recurrent Respiratory Papillomatosis(JORRP)**
   Causative organisms: HPV 6 and 11
   Acquires HPV during birth from
   mother's Genito urinary Tract.
   Age 3-4 year child born to primi gravida mother
   C/C: Stridor/ hoarseness
   O/C: B/L Multiple Papillomas, very high recurrence after surgical exision
   Rx: Excision by microdebrider
   Medical Rx: Interferon α (to reduce the recurrence)

Carcinoma larynx

M/C Type – Squamous cell carcinoma
M.C site: Glottis

Best prognosis

Ca Glottis: M.C presentation – Hoarseness (best prognosis)
Ca supraglottis: M.C presentation – Foreign body sensation
Ca Subglottis: M.C presentation - Stridor, Poor prognosis

TNM Staging for glottis Carcinoma

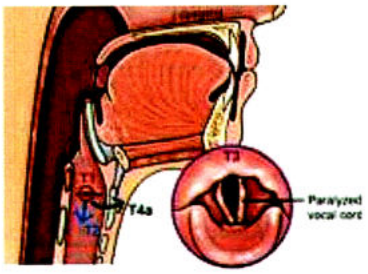
T_1- Normal vocal fold
T_2- Impaired vocal fold motility

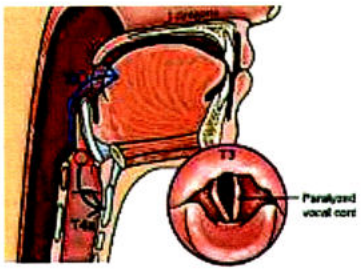
T_3- Fixed vocal folds or 3p - pre- epiglotic
- Para epiglotic
- Post Cricoid area

T_4-Outside larynx

T_{4a}- Anywhere in neck except T_{4b}

T_{4b}- - Superior mediastinum
- Prevertebral space
- Encases carotid Artery





TNM staging for supra glottic carcinoma

T_1 → 1 subsite

T_2 → > 1 Subsite mucosa of base of tongue or

T_3 → vocal fold palsy

T_4 → outside larynx

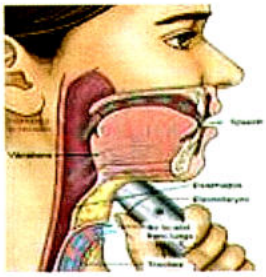
Treatment for Ca Glottis:

- $T1/T_2$- Stage – Radiotherapy
- T_3 – CCRT (concurrent chemo radiotherapy)
- T_{4a} Stage combined (surgery + Radiotherapy ± chemotherapy)
- T_{4b} Stage: Palliative therapy

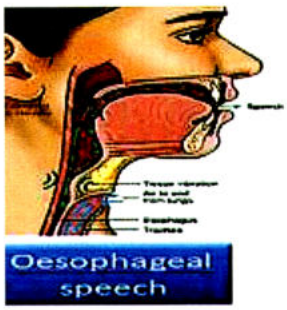
In T_1Stage if malignancy is on Anterior commisure → Staged as $T._1$But it is treated as T4a

Post laryngectomy voice rehabilitation:

1. **Electrolarynx**
   Small hand held device – easy to use



2. **Oesophageal speech**
   Patient swallows the air in to esophagus
   and brings it out to phonate



3. **Tracheo-oesophageal speech**
   Consists of one way valve c/as trachea
   esophageal voice prosthesis (TEP)
   Placed b/w Trachea and esophagus
   Best way for voice rehabilitation