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Ear

(Fundamentals of Ear)

Osteology of Temporal Bone

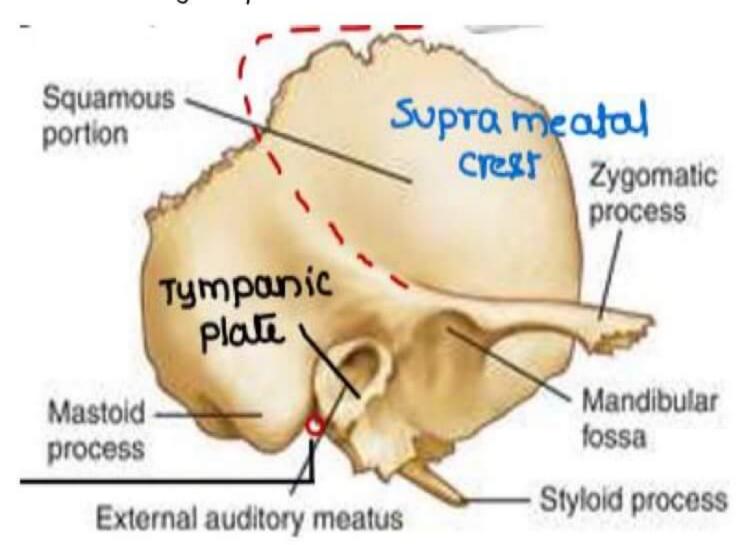
> Most complex bone in the body [20 ossification centers]

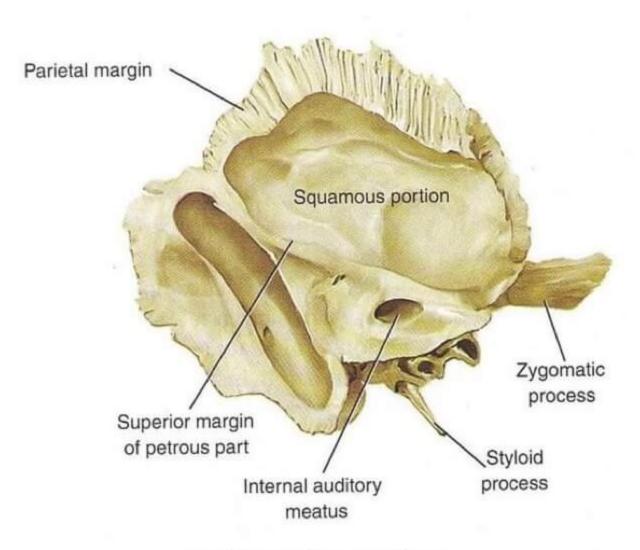
Parts of Temporal Bone

- 1. Squamous
- 2. Petro mastoid Petrous

 Differentiates after birth

 Mastoid
- 3. Tympanic
- 4. Styloid process





medial view of temporal bone.

→ Squamous part

- Covers temporal lobe of brain
- Forms zygomatic process of temporal bone
- Supra meatal crest: Part of inferior temporal line

→ Mastoid process

- Starts developing 6 months after birth (Due to pull of Sternocleidomastoid muscle)
- Mastoid tip appears by 2 years of Age
- Reaches adult size by 18-19 years of age
- Cancellous bone: consist of air cells
 - Largest → mastoid antrum
- Antrum is of adult size at the time of birth

→ Petrous part

- On medial side of Temporal bone inner ear is present
- Most complex part of Temporal bone (14 ossification centers)
- Densest / hardest bone in body
- Internal Auditory Canal / IA Meatus opens into posterior surface

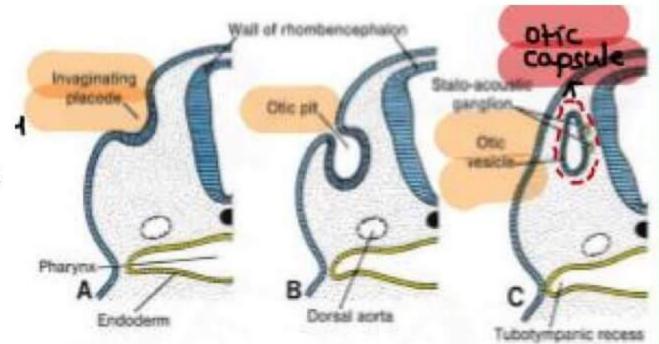


Contents of Internal Auditory Canal:

- 7th & 8th cranial nerve
- · Labyrinthine artery (Branch of AICA)
- Vestibular ganglion (Superior and inferior vestibular nerves)
- Nerve of wrisberg (Nervus Intermedius) carries sensory and secretomotor fibers for facial nerve

Embryology Of Inner Ear Formation

- ightarrow In rhombencephalon stage, near 1st and 2nd Pharyngeal arches there is formation of Otic placode
- → Otic placode formed from surface ectoderm
- → Otic placode converts to Otic pit and leads to formation of Otic vesicle / Auditory vesicle /Otocyst



Rhomben cephalon stage

- → Otic vesicle forms → Membranous labyrinth (Sensory part of Inner ear)
- → Otic capsule is the embryological cartilaginous structure developed from 2° mesoderm It is cartilaginous framework for inner ear

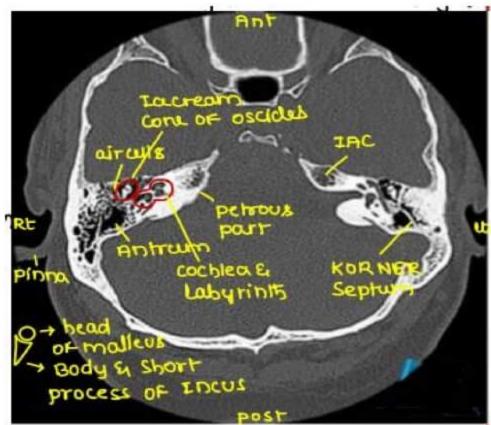
Calcifications by

→ Enchondral Ossification

★ 14 ossification centers
 Inner ear [Bony Labyrinth]

→ Enchondral Bone

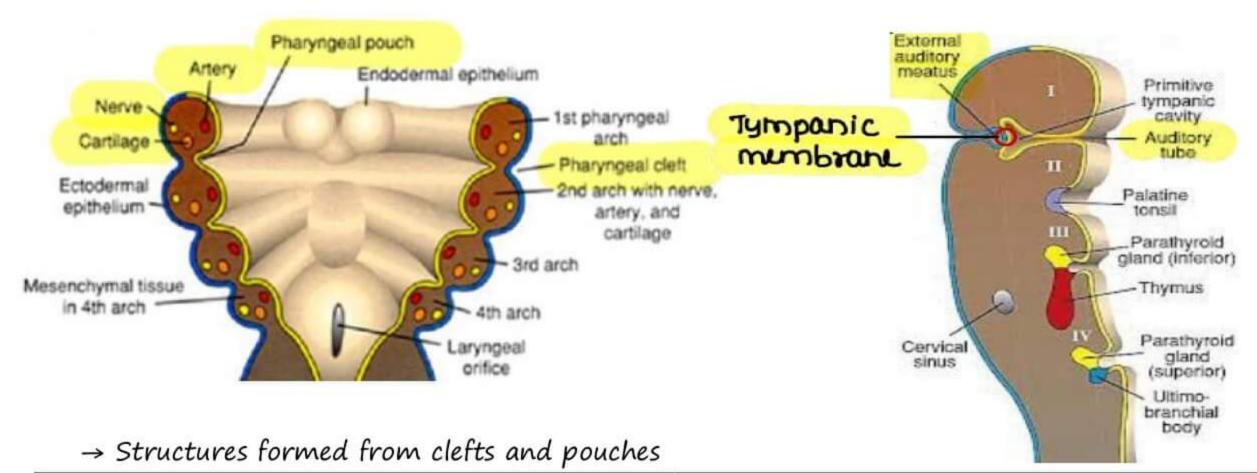
- → Stylomastoid foramen
 - Facial nerve [VII CN] comes out
 - Stylomastoid artery goes in
 - Post aural incision is not given to a child < 2yrs of age
- ightarrow Between squamous & petrous part, Petro squamous suture present in embryo, disappears after birth
 - Koerner's Septum
 - · Persistent petro squamous suture
 - · Significant in mastoid Surgery
 - Koerner's septum is lateral to antrum





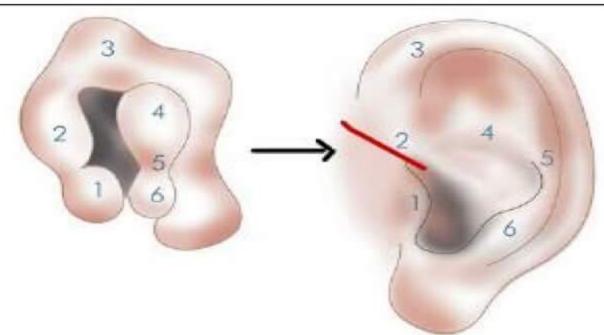
Pharyngeal / Branchial Arches

- → 5 on each side [1, 2, 3, 4, 6]
- → All 3 germs layers are present
- → Own blood & nerve supply and forms its own Musculo-cartilaginous structure

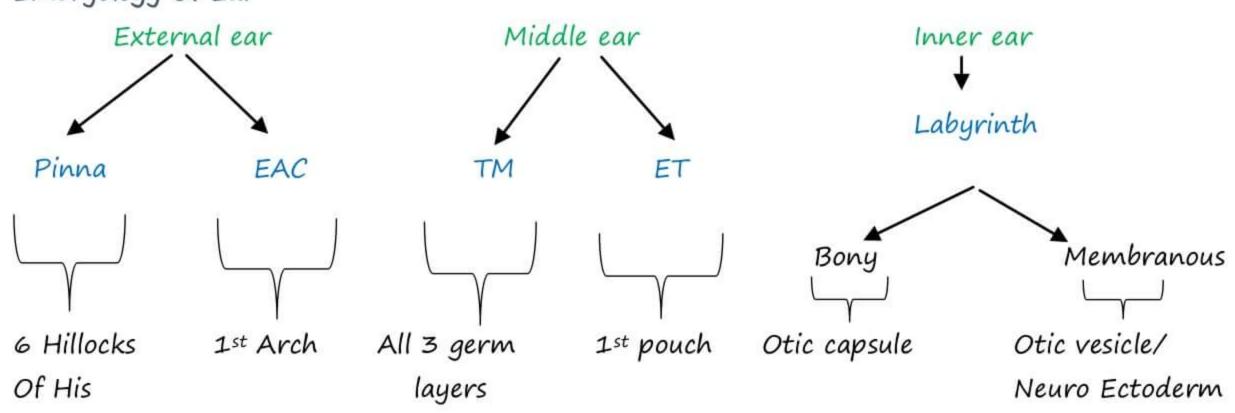


Clefts	Pouches
1st arch – EAC	1st pouch -Tubotympanic recess
2 nd to 4 th clefts are obliterated	2 nd pouch – Tonsillar fossa, Palatine fossa
	3rd pouch – Inferior Parathyroid gland
	4th pouch – Superior Parathyroid gland
	5th pouch – Ultimobranchial body
	(Parafollicular 'C' cells)

- → Around the EAC, the 1st & 2nd arch forms
 - → HILLOCKS OF HIS
 - 1ST HILLOCK forms from 1st arch Remaining Hillocks forms from 2nd
 - 1st hillocks forms tragus



Embryology Of Ear



Anatomy of External Ear

1) Pinna

- → Formed from single piece of yellow elastic cartilage expect lobule
 - Never ossifies
 - Also seen in tip of nose and epiglottis

→ Incisura Terminalis

- Place of fusion of 1st and 2nd hillock.
- End-aural approach done by giving LEMPERT'S INCISION here.

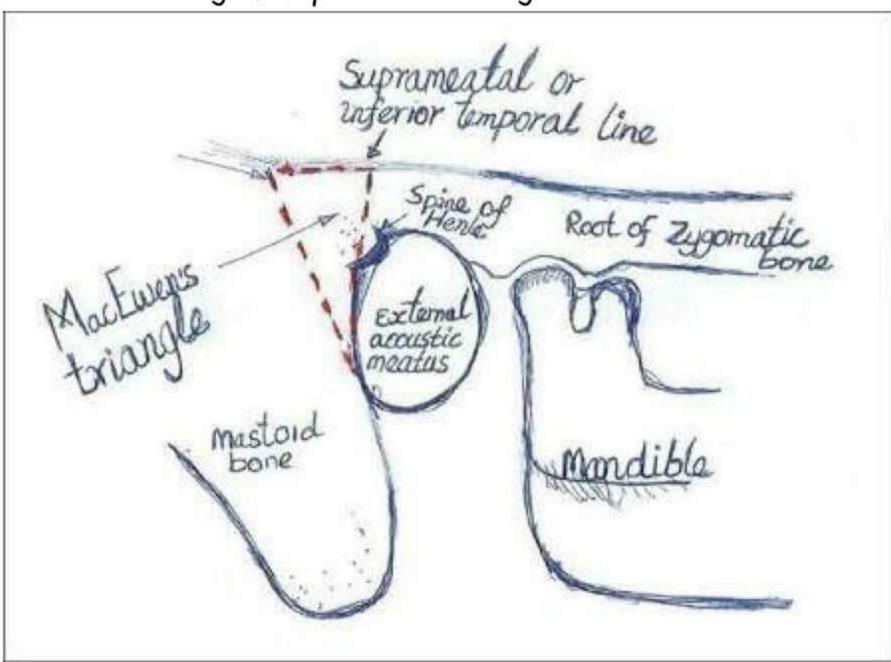


(Post aural approach by William Wilde's approach behind pinna

Trans canal approach / Permeatal approach / Endomeatal approach by Rosen's Incision)

→ Cymba concha is the anatomical landmark of mastoid antrum. (Surface Landmark)





Surgical landmark of Mastoid Antrum

Mastoid antrum is present 1.25-1.5 cm deep to MacEwen's triangle

Boundaries:

- 1. Suprameatal crest / Inferior Temporal line
- 2. Postero-superior wall of EAC
- Tangent between above (2)
- Antrum is 1.25 1.5 cm deep to MacEwan's triangle

2) External Auditory Canal

Length = 24mm

Lateral ¹/3rd = Cartilaginous

Medial/Inner ²/3rd = Bony



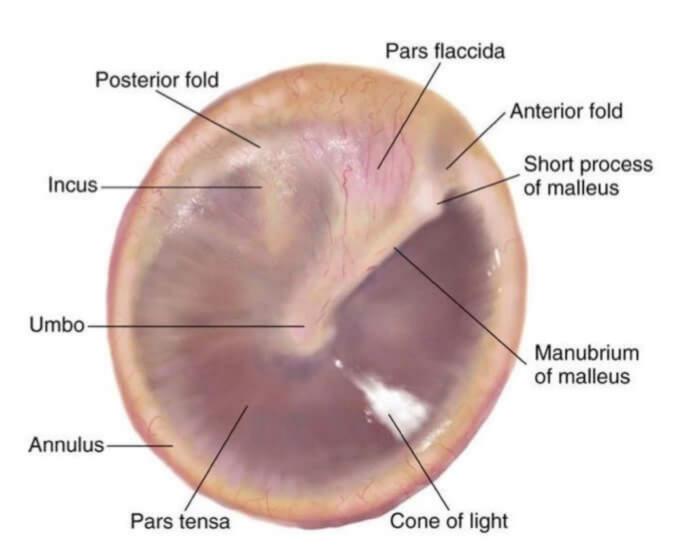
Isthmus - Narrowest point, 6mm lateral to Tympanic membrane

- → Cartilaginous part → Lateral / outer 1/3rd (8mm) of EAC
- → Cartilaginous part has sweat glands, Sebaceous, Ceruminous glands & hairs
- → Ceruminous glands → are modified sweat gland secretes cerumen.

Sweat Glands		
Eccrine / Typical	Apocrine / Atypical (AAA)	
- Present all over the body	- Only at axilla, nipple / areola, pubic area,	
- Opens on skin surface	perianal or circumanal area	
- Supplied by cholinergic nerve fibers	- Open inside hair follicles	
	- Supplied by adrenergic nerve fibers	
	- Modifications	
	Ceruminous – EAC	
	Ciliary (Moll's) glands – Eye lids	
	Mammary glands	

- \rightarrow All secretory glands in the body are supplied by parasympathetic system expect SWEAT glands (By Sympathetic system)
- → Ceruminous glands secrete cerumen
- → Wax is formed by mixture of all secretions + dead epithelial cells & hairs.
- → Wax has pH of 4

Tympanic Membrane



TYMPANIC MEMBRANE

- → Trimeric structure formed from all three germ layers
 - Epithelial layer from ectoderm
 - Fibrous layer from mesoderm
 - Mucosal layer from endoderm
- → Pearly grey / Translucent grey in color
- → TM is divided into two parts: 1. Pars Flaccida (Shrapnel's membrane)
 - 2. Pars Tensa
- → Fibrous layer is scanty and unorganized in Pars flaccida.



- → Fibrous layer forms Annulus tympanicum.
 - It attaches the TM to bony EAC. [Bony annulus]
 - Is fibrocartilaginous in nature
 - Notch of RIVINUS → Deficiency in bony annulus superiorly covered by P. Flaccida
- \rightarrow TM is attached at an angle of 55° with Anteroinferior wall of EAC.
 - At birth TM is almost horizontal
 - At 4 yrs of age it attains 55° angle with Anteroinferior [Floor] wall of EAC
 - Anteroinferior wall is longest wall of EAC
 - Posterosuperior is shortest wall.

Foreign body in EAC removal

- 1) Probing
- 11) Micro forceps
- III) Syringing Posterosuperior direction
 - Water at 37° C
 - C/I
 - 1. FB beyond isthmus
 - 2. Acute infection of external and middle ear
 - 3. Big impacted foreign body
 - 4. Vegetative Foreign body

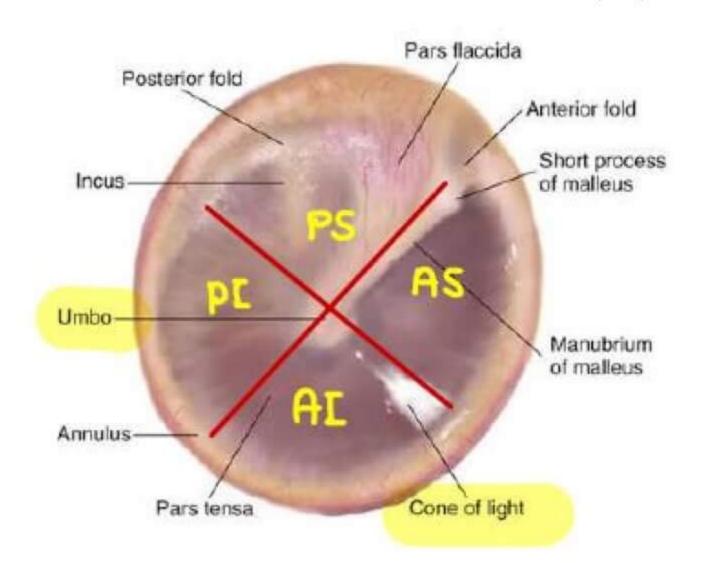
IV) Micro suction

- Best way of foreign body removal
- Creates negative pressure, minimum damage.
- Removal of live insects: With Lukewarm oil

→ UMBO

- Most visible anatomical landmark of Tympanic membrane
- Divides TM into 4 regions.
- Most mobile part of malleus → UMBO
 - Not a reliable landmark
- Most reliable landmark of TM → Lateral process of Malleus
- → Cone of light → in Antero-inferior quadrant of Pars Tensa

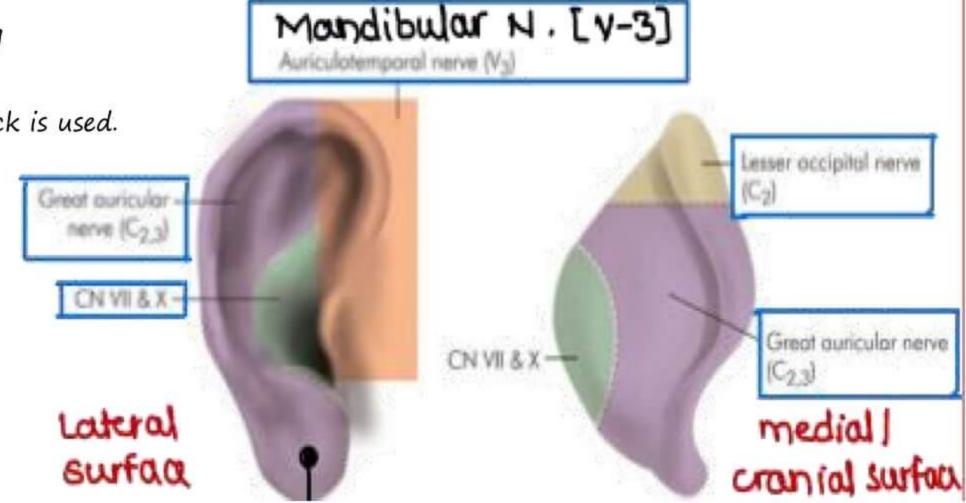
Because pulling of handle of malleus, AI quadrant becomes perpendicular → cone of light.





Nerve Supply to Pinna

- → Split lobule due to Ear piercing
 - Rx by Lobuloplasty
 - Greater Auricular Nerve block is used.



Rhytidectomy

- → Removal of rhytids (Facial wrinkles)
- → Incisions done behind pinna and mastoid
- → M/C nerve involved Greater Auricular nerve
- → M/C motor nerve involved Zygomatic br. of facial nerve

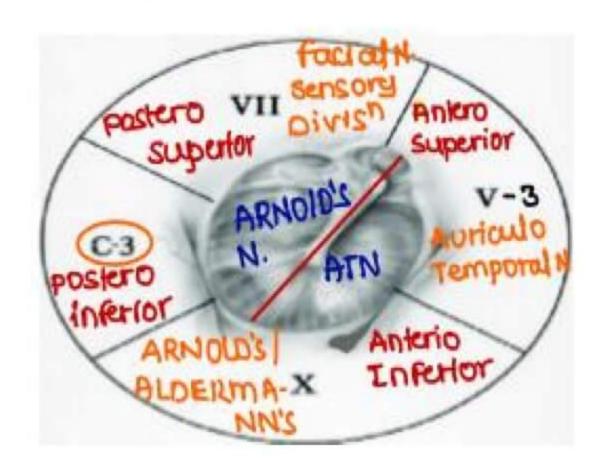


Rhytidectomy Incision (Facelift Incision)

Nerve Supply to EAC

- 1) Antero-superior -> Auriculotemporal nerve
- II) Floor/Antero-inferior → ARNOLD'S branch of Vagus
 - → Cough reflex is due to Internal laryngeal nerve (Branch of Vagus)
 - \rightarrow Cough reflex during syringing is $d/t \rightarrow$ Arnold's nerve
- III) Posterior-superior → Facial nerve

Loss of sensory sensation in this part → HITZELBERGER sign seen in vestibular schwannoma





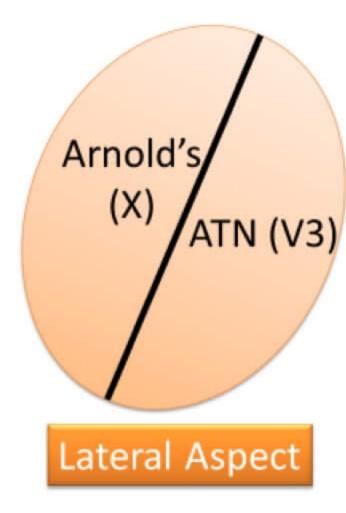
Nerve Supply to Tympanic Membrane

Lateral Aspect

Anterior half - Auriculotemporal Nerve Posterior half - Arnold's Nerve

Medial Aspect

Jacobson's nerve (Branch of CN)
[Sensory supply to middle ear also.]





Medial Aspect

Diseases of External Ear

Congenital Disorders

Anotia/Microtia

- \rightarrow Anotia \rightarrow Absent pinna \rightarrow R_x \rightarrow Pinnaplasty
- \rightarrow Microtia \rightarrow Small pinna \rightarrow R_x \rightarrow Pinnaplasty
- → Pinnaplasty
 - Done by rib / costal cartilage
 - Rib cartilage develops by 4-5 years of age
 - Pinnaplasty done at 5-7 years age.





Anotia

Microtia

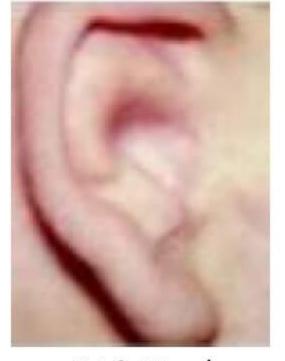
EAC Atresia

- → Defect in IST Arch
- $\rightarrow R_X \rightarrow Canaloplasty$
- → U/L EAC Atresia + Anotia
 Rx- Pinnaplasty first then Canaloplasty
- → B/L EAC Atresia + Anotia
 - Rx → BAHA (Bone Anchored Hearing Aid)
 - Titanium screw has Osseo-integration property
 - Requires 2.5 3 mm bone thickness achieved around 5 yrs of age

FDA norms → Can't implant < 5yrs

NHS norms \rightarrow Allow after 3 years after doing CT scan [2.5 mm \oplus]

Before prescribed age, we can Rx with SOFT BAND HEARING AID



EAC Atresia

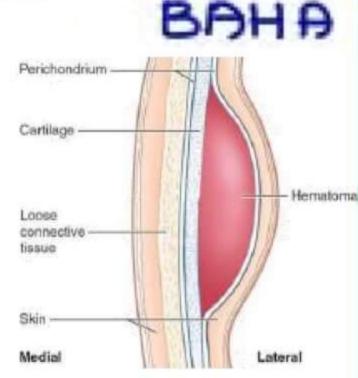


Trauma

PINNA HEMATOMA (CAULIFLOWER EAR / BOXER'S EAR)

- → Due to blunt trauma to pinna
- → Collection of blood between cartilage and perichondrium
- $\rightarrow R_X \rightarrow$ needle aspiration pressure dressing





caulif Lower Ear

Perichondritis of the Ear

Perichondritis of Pinna

M/c causative organism → Pseudomonas

- Rx → 1. Ciprofloxacin (Antibiotic of choice)
 - 2. Analgesics
 - 3. Incision and Drainage

Keratosis Obturans

- → Deposition of wax along with dead epithelial cells in EAC
- → Can erode the bone & can even cause facial palsy

ACUTE OTITIS EXTERNA / TROPICAL / SWIMMER'S EAR

- → Acute infection of EAC
- -> More common in hot and humid climate
- → Types
 - Localized / Furunculosis
 - Diffuse
- → Localized / Furunculosis
 - Staph. aureus infection of hair follicles → furuncle
 - Localized to outer 1/3rd





FURUNCULOSES



DIFFUSE

→ Diffuse

Mc causative organism → Pseudomonas

Presentation: Severe pain/ excruciating pain

O/E: Tragal sign → POSITIVE

Patient moves always when pressure is applied on tragus Present in both localized & Diffuse Otitis externa

$\rightarrow Rx$

- Antibiotics
- Analgesics
- 10% Ichthammol glycerin packing
 Ichthammol → Local antiseptic
 Glycerin → Hygroscopic

Otomycosis (Singapore Ear)

- → Fungal infection of EAC
- → Causative organism

Aspergillus Niger (M/C) – Forms black colonies

Aspergillus Fumigatus – Forms green colonies

Candida albicans – Forms white cottony colonies

- → Severe itching present, & Hearing, discharge, mild pain.
- \rightarrow O/E \rightarrow Wet blotting paper appearance of TM
- \rightarrow Rx \rightarrow Aural toilet by micro suction

Topical antifungal ear drops × 4 weeks

Keratolytic agents → Salicylic/ Acetic Acid









candicla albicans

Gentian Violet → Prevent biofilm formation SODIUM BICARBONATE DROPS NOT USED

Exostosis (Surfer's Ear)

- → Benign growth of bony EAC
- → Found mainly in surfers
- → Broad based, multiple growths
- → Way of body defense mechanism

MYRINGITIS BULLOSA HAEMORRHAGICA (BULLOUS MYRINGITIS)

- → Formation of bleeding blebs on TM Blood mixed discharge present, painful & & hearing "Sago-grain appearance of TM" in healing phase.
- → Caused by pneumococcus (Streptococcus pneumoniae) {Earlier believed d/t Virus/ Mycoplasma}
- → Rx Topical antibiotics+ topical steroids ear drops



SURFER'S EAR



MALIGNANT OTITIS EXTERNA / ACUTE NECROTISING OTITIS EXTERNA

Term Malignant → Misnomer (Mitotic figures are not high) Caused by "Pseudomonas Aeruginosa"

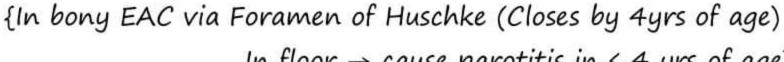
- → Rapidly spreading in (Predisposing factor)
 - 1) Immunocompromised patients
 - 11) Elderly uncontrolled diabetics
 - III) Chemotherapy

C/F - Pain, greenish black discharge

O/E: Granulations in EAC

It spreads to Skull Base: Lateral skull base osteomyelitis

- Multiple CN palsies (M/C CN involved is facial nerve)
- Spread to skull bone through Fissures of Santorini in cartilaginous part of EAC



In floor → cause parotitis in < 4 yrs of age}

Investigation

- Confirmed by bone scan (Tc -99m)

(Diagnostic, IOC - Specific)

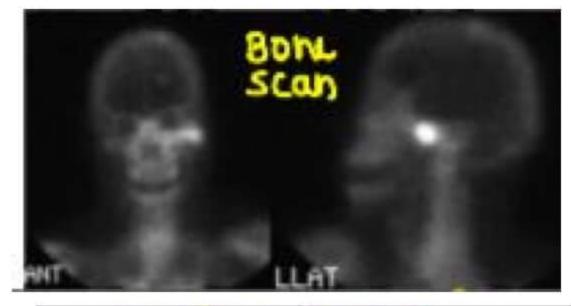
- $\rightarrow R_x$
- → DOC is Ciprofloxacin

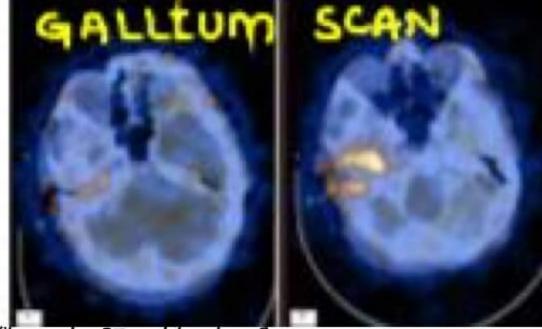
Anti-pseudomonal antibiotics

- 1) Ceftazidime
- 3rd gen Cephalosporins
- 11) Cefoperazone + Sulbactam
- III) Piperacillin + Tazobactam
- IV) Carbapenem → Imipenem, Meropenem



MOE





 \rightarrow After 6 weeks, check ESR \rightarrow (N) \rightarrow Discharge with Tab. Ciprofloxacin [Double dose] Gallium scan [Ga-67] [Prognostic Investigation of Choice] - Non specific

Anatomy of Middle Ear

Middle Ear Cleft

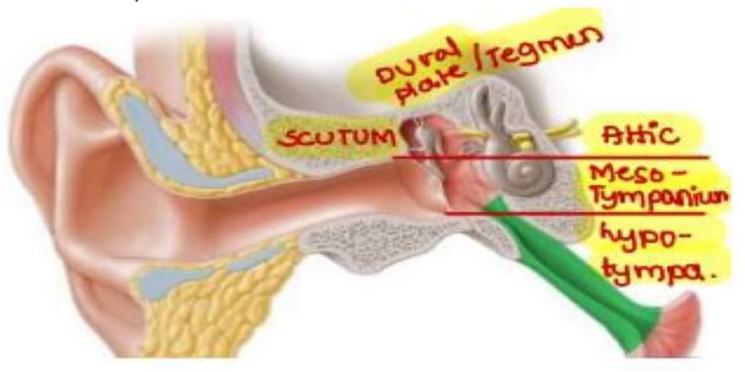
- → Mastoid + Middle ear + Eustachian tube
- \rightarrow Volume = 5ml (Mastoid) + 1 ml (ME) \rightarrow 6ml

Eustachian tube is closed in resting position

T. Tegmun Tegmun

Middle Ear

- → Lateral wall formed by Tympanic Membrane
- → TM divides middle ear into
 - 1) Epitympanum/Attic
 - II) Mesotympanum: Narrowest part
 - III) Hypotympanum



- → Bony lateral wall of Attic → Scutum
 - Erosion of scutum is characteristic CT scan finding of cholesteatoma.
- → Roof separates ME from Middle cranial fossa.
- → Bony plate above the Middle ear & Mastoid TEGMEN / DURAL PLATE.

Part above the ME

- Tegmen Tympani.

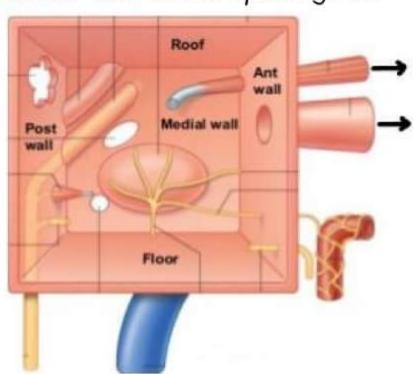
Part above the antrum

- T. Antri.

- → Aditus connects ME/Attic to antrum.
- → Arcuate Eminence

Bulge in Tegmen due to superior semicircular canal Landmark for Facial Nerve in Middle Cranial Fossa Approach

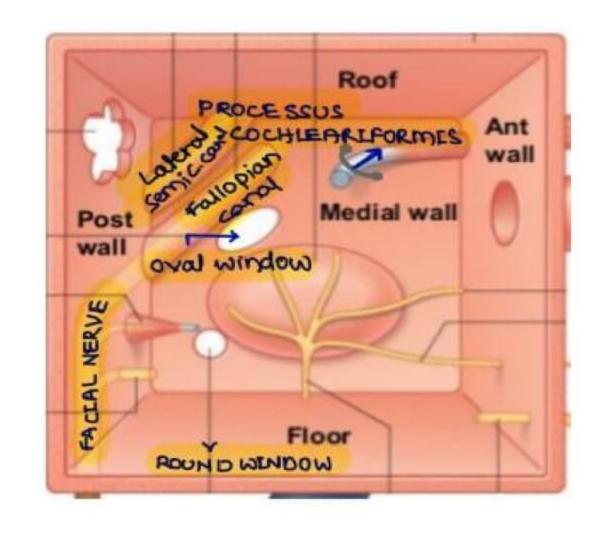
→ Anterior wall has 2 openings for



- 1. Tensor tympani (upper small opening)
- 2. Eustachian tube / Auditory tube (Lower big opening)



- → Medial wall → Has 2 windows
 - 1. Oval window / Fenestra Vestibuli
 - 2. Round window / Fenestra Cochlea
- → Oval window is covered by Foot plate of stapes.
- → Round window by secondary tympanic membrane



- → Processus cochleariformis is a hook
 - Tensor Tympani hooks around Processus cochleariformis and comes out and attaches to handle of malleus.
 - Tensor tympani and Tensor veli palatini both supplied by Mandibular nerve (br. of Trigeminal nerve)
- → Horizontal / Tympanic segment of Facial nerve present in fallopian canal (Intra Temporal part) (Bony canal)
- → Fallopian canal is longest canal for any cranial nerve.

Length = 27 mm

Above it dome of lateral semicircular canal is present.

M/c site of labyrinthine fistula → Dome of lateral SC canal

- → Promontory Outward bulge due to 1st turn / Basal turn of Cochlea.
- → Narrowest part of ME Mesotympanum
- → Surgical floor of ME Medial wall
- → Shallowest part of Middle ear in Surgical position Meso tympanum

→ Posterior wall

- → Vertical segment / Mastoid segment of facial nerve present.
- \rightarrow Pyramid \rightarrow From pyramid, arises stapedius (Smallest named muscle in body), goes and attaches neck of stapes.
- → Opens into mastoid through Aditus.
- → Chorda Tympani Nerve Branch from Mastoid segment, hangs like a cord in the ME, and exits from anterior wall through Canal of Huguier

→ Facial Recess [2D area]

- Lateral to Facial Nerve
- Boundaries

Medial → Facial N

Inferior -> Chorda Facial angle

- Significance - Site for posterior tympanotomy [Opening the ME from Mastoid]



→ Sinus Tympani [3D space]

- Lies medial to Facial Nerve, hidden place in Middle ear
- Boundaries

Lateral → Facial nerve

Posterior → Posterior wall

Medial → Medial wall

Ponticulus - Superior bony ridge

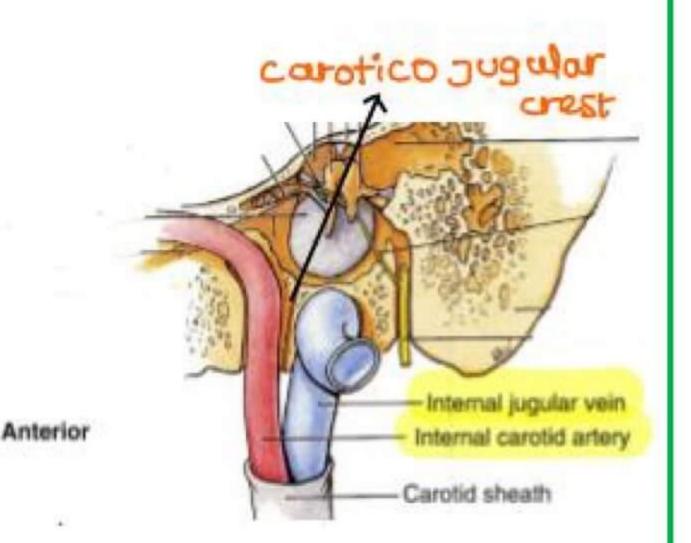
Subiculum - Inferior bony ridge

- Mc site for recurrent/ residual cholesteatoma

→ Floor

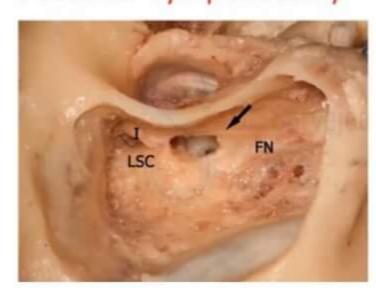
- → Separates ME from jugular bulb and ICA.
- → Carotico Jugular Crest
- ICA present anterior to it
- Jugular bulb present posterior to it.
- Helps in distinguishing b/w ICA & Jugular bulb

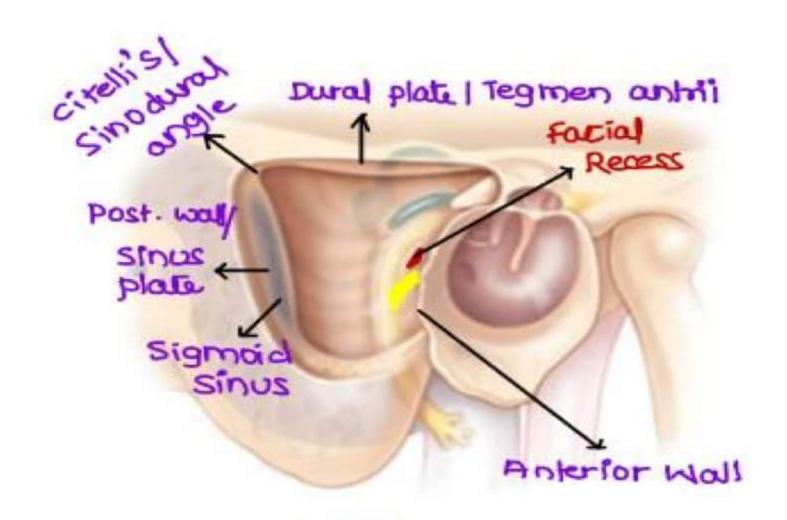
PHELP SIGN: In ability to distinguish b/w ICA & Jugular bulb d/t erosion of Carotico jugular crest Seen in CECT scan of Glomus tumor



Mastoid Process

Posterior Tympanotomy





→ TRAUTMANN'S Triangle

→ Boundaries

MASTOLD PROCESS

Anteriorly → Bony labyrinth

Posteriorly → Sigmoid sinus

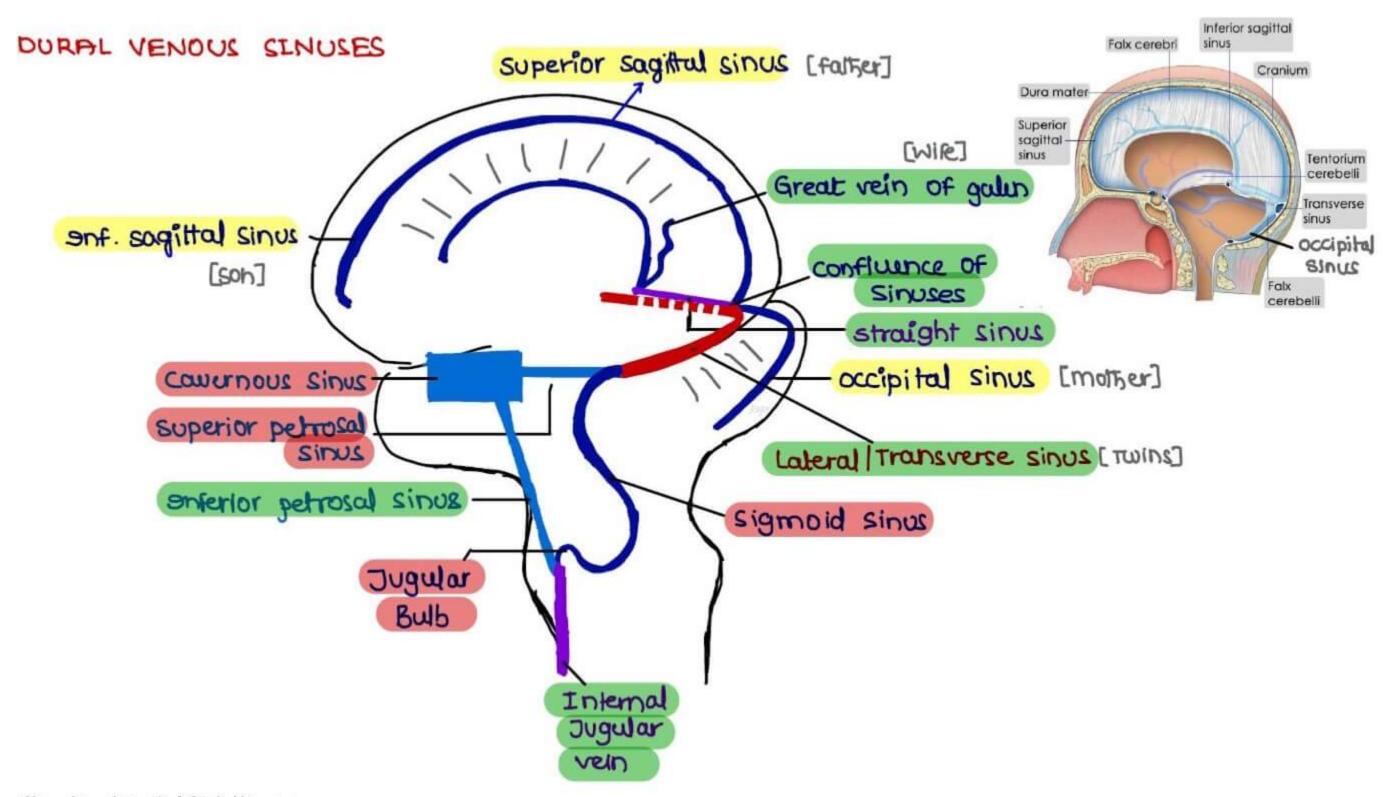
Superiorly → Superior Petrosal sinus

→ Cholesteatoma erodes the bone and goes into posterior cranial fossa from here.



- → Donaldson's line is a surgical landmark for endolymphatic sac
 - It is a line that goes along the lateral semicircular canal, bisecting posterior semicircular canal
 - Endolymphatic sac is situated just below this point where the line meets the sigmoid sinus

Dural Venous Sinuses



Contents of Middle ear

1) 2 Muscles

Tensor Tympani

Origin

At canal for Tensor Tympani

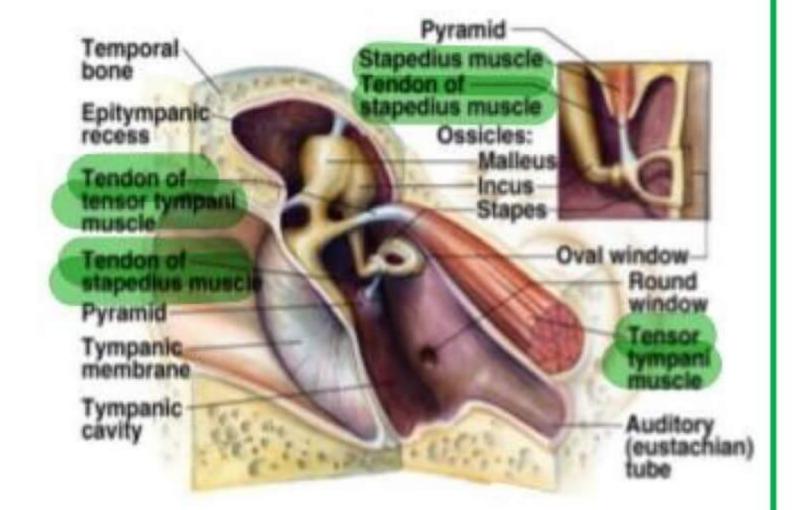
Insertion \rightarrow Handle of Malleus Nerve supply \rightarrow Mandibular nerve

Stapedius

Origin → Pyramid

Insertion → Neck of stapes

Nerve supply → Facial nerve





- 2) 3 Ossicles
- Malleus and Incus formed by (1) Mandibular arch → Meckel's cartilage
- Stapes by (II) Hyoid arch, expect the medial surface of stapes foot plate (From otic capsule)

Ossicles reach adult size at

→ 15 weeks of IUL

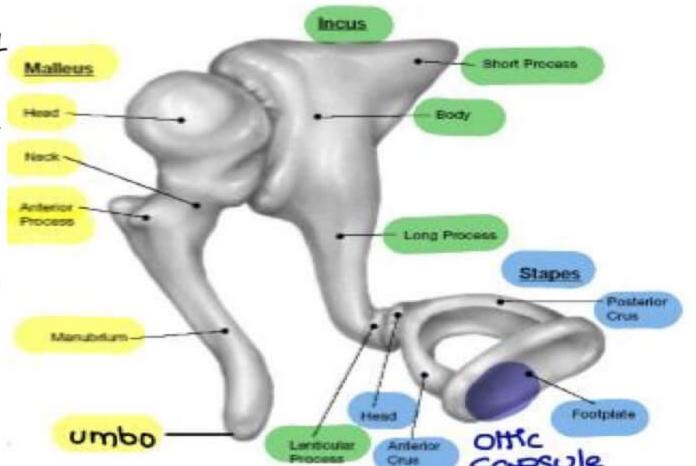
Adult configuration at

→ 20 weeks of IUL

Organ of Corti starts hearing at

→ 20 weeks of IUL

Organ of Corti adult configuration → 25 weeks of IUL



→ Structure attaining adult size at the time of birth

- 1) Ossicles
- 11) ME/Tympanic cavity
- III) Cochlea/labyrinth
- IV) Mastoid Antrum

Mastoid process/ Maxillary Antrum/ Orbital Cavity: Not of Adult Size

Types of Joints of Ossicles → Synovial joint

B/w Malleus & Incus → Saddle type

B/W Incus & Stapes → Ball & socket type

M/c or 1st part to be eroded -> Lenticular process of stapes

2nd M/c part to be eroded → Long process of Incus

Last to undergo erosion is → Foot plate of Stapes



Bony portion

Bony-cartilaginous

junction (isthmus)

Cartilaginous

Nasopharynx

EUSTACHIAN TUBE

→ Eustachian tube / Auditory tube / Pharyngo tympanic tube Connects anterior wall of middle ear to lateral wall of nasopharynx.
36 mm length

Lateral 1/3rd is bony - 12 mm

Medial 2/3rd is cartilaginous - 24 mm

Isthmus → Narrowest part of ET, lies at bony cartilaginous junction

Lies at an angle of 45° with horizontal

Functions

- 1. Maintains the Middle ear air pressure = Atm. air pressure
- 2. Drains the secretions of ME
- 3. Prevents infections from going to Middle ear
 - Generally, ET is closed by OSTMANN'S PAD OF FAT
 - During swallowing / yawning, it opens b/c of medial fibers of TENSOR VELI PALATINI [Dilator tubae]

INFANT ET	ADULT ET
- 16-18mm in length	- 36mm in length
- Almost horizontal	 At an angle of 45° with the horizontal

Eustachian tube function tests:

Passive test or non-physiological tests: Tensor veli palatini is not involved in it.

1. Valsalva test/maneuver:

Principle- Positive pressure in the nasopharynx causes air to enter the eustachian tube and reach middle ear which causes pop up sound d/t bulging of TM.

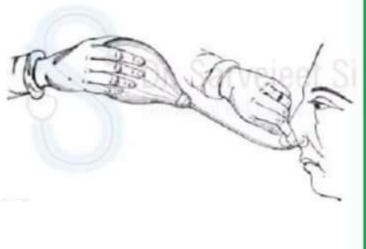
TM Movement can also be seen by examiner with otoscope

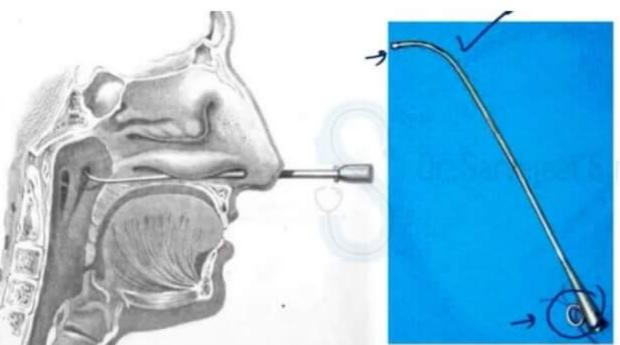
- Politzer test: designed for those who can't perform Valsalva test eg. Children
- Politzer bag is connected to rubber tube
- Tube is placed in one nostril and other is closed
- Air is blown with pressure by pressing the bulb
- Air enters nasopharynx → opens eustachian tube
 & enters middle ear→ pops out TM



Done using ET Catheter - curved tube/catheter with ring attached which tells about the direction of curve







ET catheter is inserted into nose and nasopharynx

1

rotated by 900 to medial side and pulled back

1

catheter engages behind nasal septum

1

now catheter rotated at 180°

1

enters eustachian tube

1

air blown using syringe or Politzer bag

1

air will enter middle ear and pops out TM

1

Can be seen using otoscope or pt. informs pop up sound in ear

Active test or Physiological test:

1. Toynbee test:

Ask the pt. to close the nose and swallow small amount of liquid

1

Negative pressure is created in nasopharynx

1

Eustachian tube opens up d/t action of Tensor veli palatini

1

Air is sucked out of middle ear

1

TM gets retracted

J.

Movement can be seen with otoscope by examiner

2. Tympanometry:

A.k.a. Inflation Deflation test

Tympanometry device can measure the pressure changes in EAC It has 2 parts: 1st is non physiological and 2^{nd} is physiological

a. Device is placed in EAC and pt. is asked to perform Valsalva

1

Air enters middle ear and TM bulges out

1

Pressure in EAC rises and is noted

b. Deflation part of test is done using Toynbee test

1

Pt. is asked to swallow against closed nose



23 928

Negative pressure is created in nasopharynx

 \downarrow

Air moves out of middle ear

1

TM gets retracted and pressure can be measured

Test for perforated Tympanic membrane

1. Radiological test: Instill radio-opaque dye into middle ear against perforated TM

↓ Take x-ray

Dye goes through whole eustachian tube to reach nasopharynx

1

Eustachian tube anatomy can be seen

2. Saccharine or Methylene blue test:

Instill methylene blue dye against perforated TM into middle ear

1

Dye comes into nasopharynx and oropharynx through eustachian tube & can be seen coming out

Saccharine is a sweet agent so, sweet taste sensed

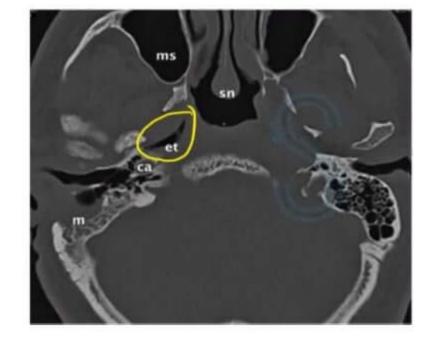
3. CT scan:

- Patulous ET- when eustachian tube has air in it
- Predisposing factors: Thyroid disorder

Pregnancy

Rapid wt. loss

- Pt. can have autophony i.e hears own voice
- Rx: inject silicon paste



4. Sono-tubometry:

Sound signal is given in nose and nasopharynx, through eustachian tube it reaches middle ear and a microphone is placed in EAC to detect it

So, we can detect the functioning of Eustachian tube

Eustachian tube balloon dilatation:

- To open the eustachian tube in case of chronic obstruction
- A balloon catheter is inserted through nose till nasopharynx against endoscopic vision and is
 rotated so that balloon can be inserted into ET and is then blown up. This produces micro
 fractures in eustachian tube and open up eustachian tube which remain open afterwards.



Anatomy of Inner Ear

- → Consist of 2 parts
 - 1) Bony labyrinth (Outer part)
 - derived from the Otic capsule which itself formed from Neural crest cells
 - 2) Membranous labyrinth (Inner part)
 - derived from Otic vesicle which itself formed from surface ectoderm
- → Based on function inner ear has 2 parts
 - 1) Auditory part

Cochlea

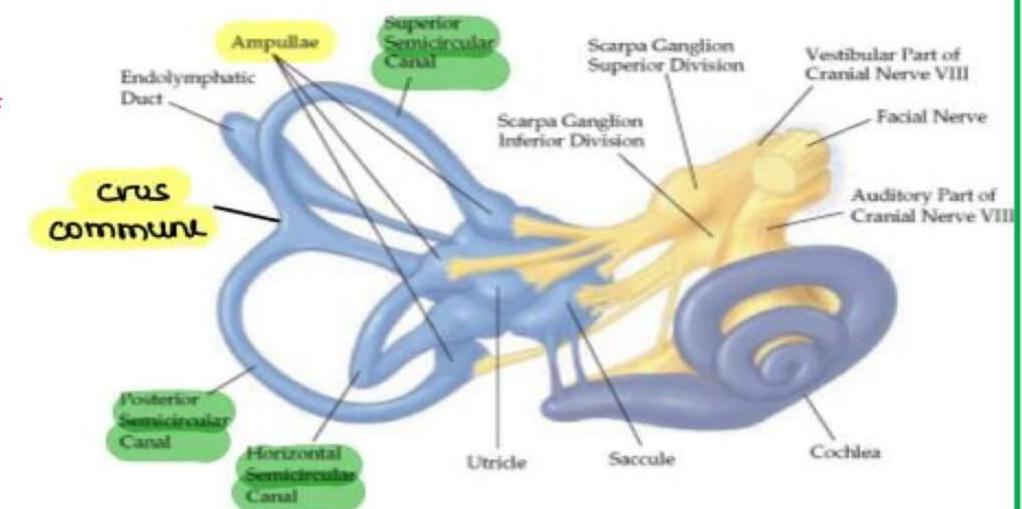
- Spiral structure
- 2.75 turns around central bony axis MODIOLUS
- 2) Vestibular part
 - 3 Semicircular canals &

2 sacs

- Utricle
- Saccule/ Sacculus

→3 semicircular canals

- Posterior / Vertical
- Lateral/ Horizontal
- Superior / Anterior



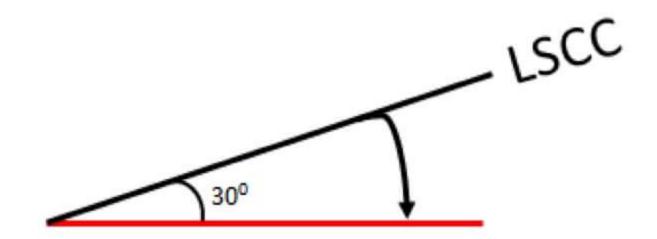
- → The space between the bony labyrinth and the membranous labyrinth is filled with fluid known as Perilymph; therefore, space is known as Peri lymphatic space.
- → Whereas; the membranous labyrinth is filled with Endolymph; therefore, space is known as endolymphatic space.

Angles of Lateral Semicircular Canal

- → The Lateral SCC may be known as Horizontal canal but it is not horizontal
- \rightarrow It is at an angle of 30° to the horizontal line

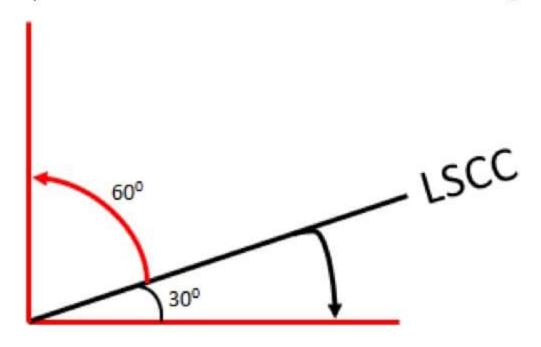
→ How to make Lateral SCC to Horizontal??

Ans: Ask the patient to bend his head forwards by 300



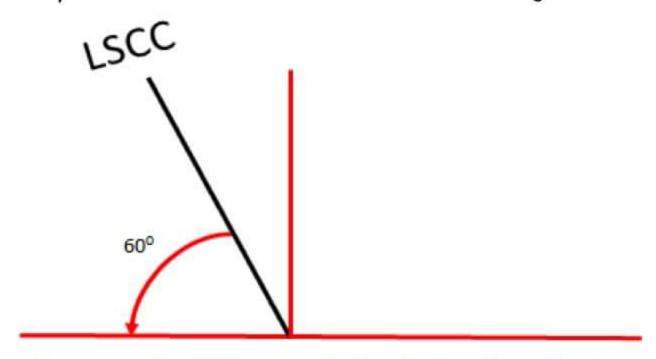
→ How to make Lateral SCC to vertical??

Ans: Ask the patient to turn his head backwards by 600



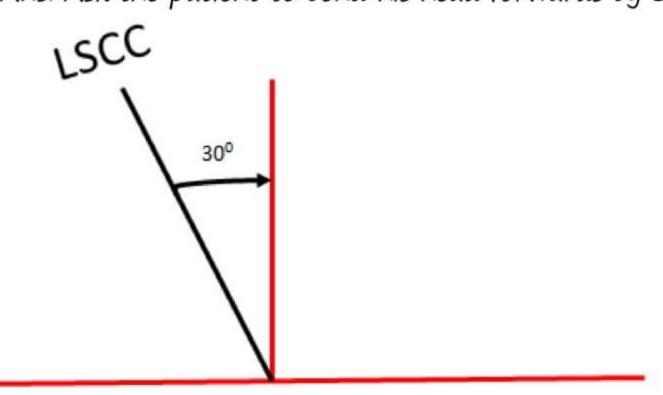
- \rightarrow If the patient is in supine position, that means Lateral SCC has turned backwards by another 90°
- → How to make Lateral SCC in horizontal position if patient is supine??

Ans: Ask the patient to turn his head backwards by 600



→ How to make Lateral SCC in vertical position if patient is supine??

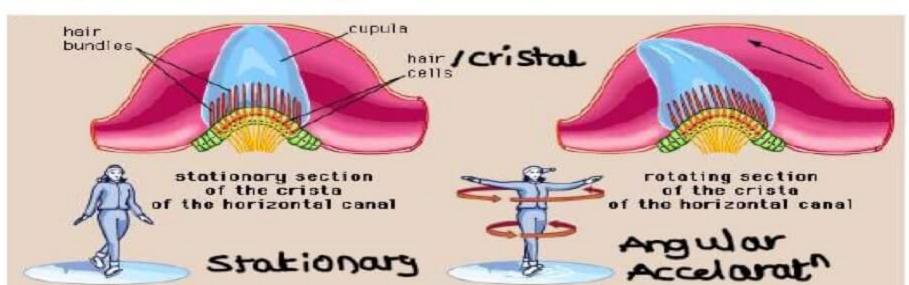
Ans: Ask the patient to bend his head forwards by 30°



- → Each canal has one ampullated end and one non-ampullated end.
- → The non-ampullated ends of superior and posterior SCC unites to form "CRUS COMMUNE" (Total 5 opening)
- → Ampulla-

Cristae Ampullaris → Individual hair cells

Cupula Ampullaris → Cristae together





Function of SCC → Angular acceleration by cristae OR cupula

- → Irritation to SCC produce Nystagmus
- → Lateral SCC Horizontal nystagmus
- → Posterior SCC Vertical nystagmus
- → Superior SCC Torsional nystagmus

Nystagmus

- → Involuntary, Rapid, To & Fro, movement of eyeballs
- → 2 types
 - Peripheral
 - Central
- →Peripheral nystagmus characteristics 5D's
 - 1) Delay
 - 2) Duration
 - 3) Decay (On repeated stimulus)
 - 4) Decreases on gaze fixation (Frenzel glasses +20 D lenses)
 - 5) Direction
 - Fast & Slow Components
 - Fast component gives the direction
 - Vertical nystagmus can be Geotropic or Ageotropic nystagmus
 - Torsional nystagmus can be clockwise Or anticlockwise

Utricle and Saccule:

- → Detects Linear acceleration
- → Macula Sensory epithelium of utricle and saccule
 - Consist of hair cells
 - Otoconia (Calcium carbonate crystals)
- Otoconia direction of movement
- → Otricle Detects horizontal linear acceleration
- → \$accule Detects vertical linear acceleration

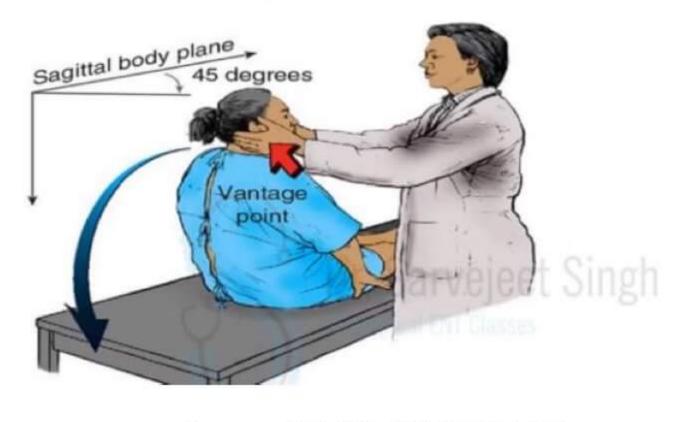
BPPV: /Benign Paroxysmal Positional Vertigo OR Otolithiasis/Canalolithiasis/Cupulolithiasis

- → Vertigo: Few seconds to minutes
- → Positional: Due to change in head position
- → Due to displacement of otoconia into Posterior SCC → OTOLITH
- → Diagnosis confirmed by → DIX-HALLPIKE'S MANOEUVRE

5D's of Peripheral Nystagmus are observed during Dix-Hallpike manoeuver, Direction of Nystagmus is mainly vertical (Geotropic / ageotropic with torsional component present)



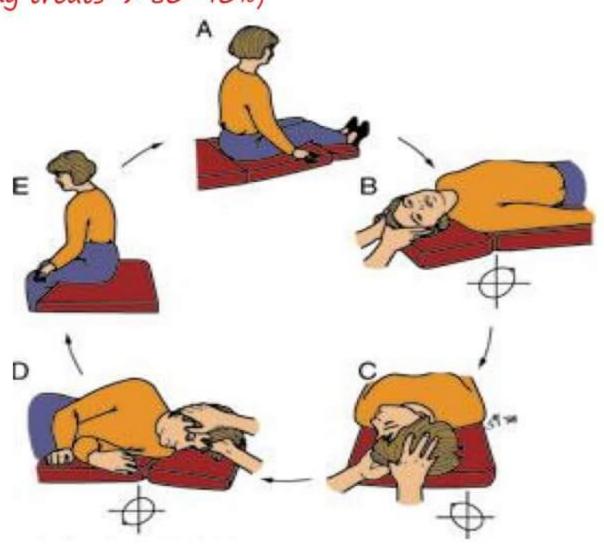
Dix-Hallpike's Maneuver





→ Rxoc → EPLEY'S MANOEUVRE

(1st sitting treats → 80-90%)



Epley's

Other Maneuvers for Rx

1. SEMONT'S / LIBERATORY MANEUVER





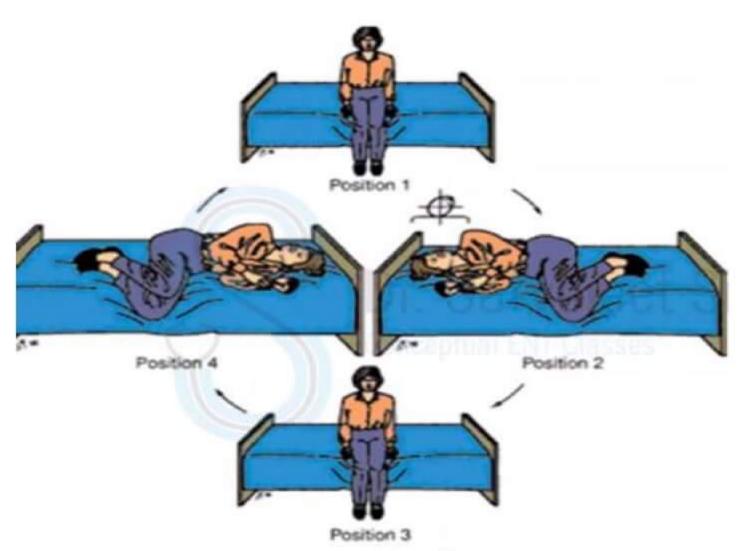
 \rightarrow After any of these 2 manoeuver ask the pt. to lay down in FOWLER'S position for 24hrs

Fowler's Position



→ Repeated BPPV attacks recommend the patient for some home exercises i.e BRANDT-DAROFF

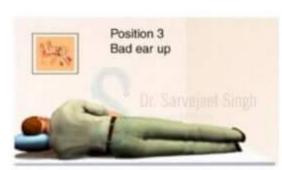




- \rightarrow Lateral canal BPPV patients we do 2 maneuvers for R_X
 - 1. LEMPERTS MANEUVER
 - Complete rotation
 - 2. LOG ROLL (BARBECUE)

Log Roll (barbecue)







- → Tests of Vestibular Labyrinth
- 1) Caloric testing -

Fitzgerald Hallpike test → Syringing at 37 ± 7° c done

30° c → Cold stimulus

44° c → Warm stimulus

- COWS

Cold → Opposite

– Nystagmus d/t irritation

Warm → Same side of lateral sec.

Modified Kobrak test (Cold Caloric Test)

Syringing with O°C water

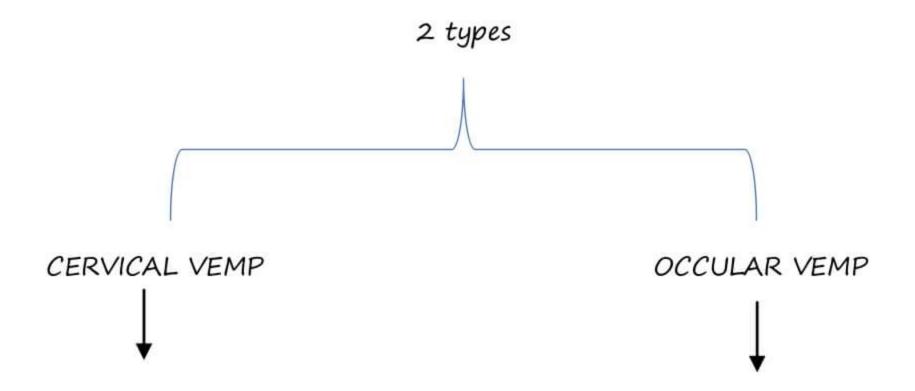
Dundas Grant test

- → Syringing is C/I in perforated Tympanic membrane
- → In such cases, cold air blown through a Copper coil tube



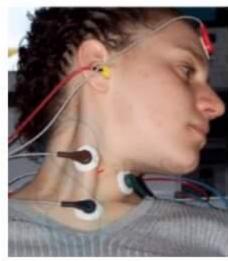
2) VEMP (Vestibular Evoked Myogenic Potential)

- Give stimulus in vestibular system and check the response in muscles



Evaluates saccule and inferior vestibular nerve

Evaluates saccule and superior vestibular nerve Cervical VEMP Ocular VEMP

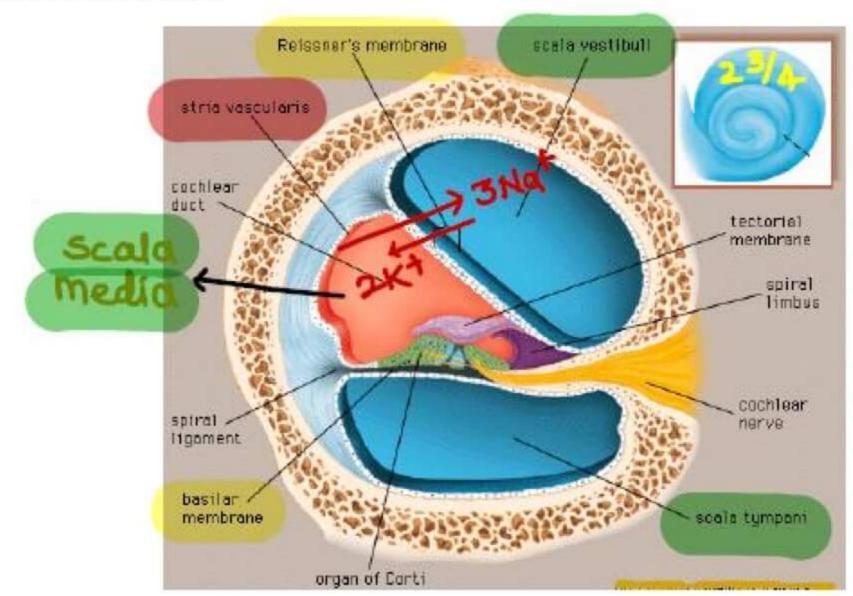


Galvanic Vestibular Stimulation

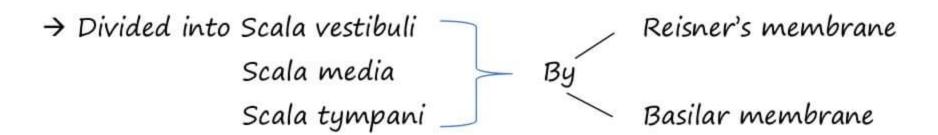
- Test of vestibular nerve with small voltage of current



COCHLEA (Auditoru Laburinth)







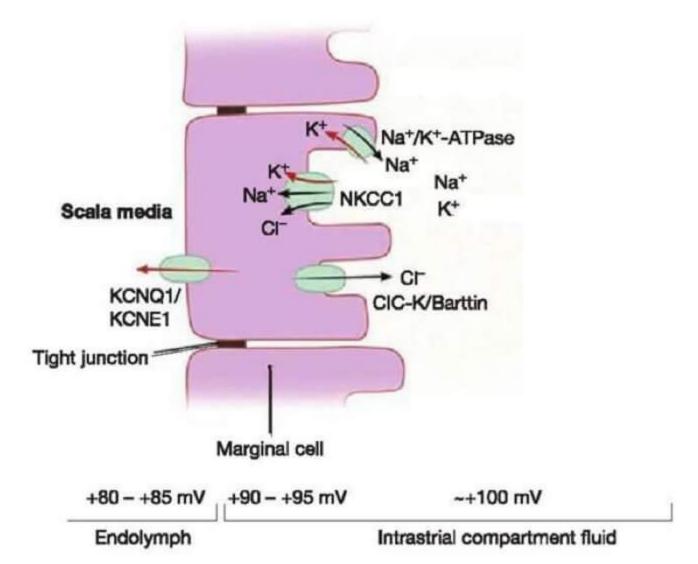
ightarrow Scala vestibuli & Scala tympani filled with ightarrow Perilymph Scala media filled with ightarrow Endolymph

Endolymph secreted by stria vascularis [Na+ K+ ATPase pump]

 \rightarrow

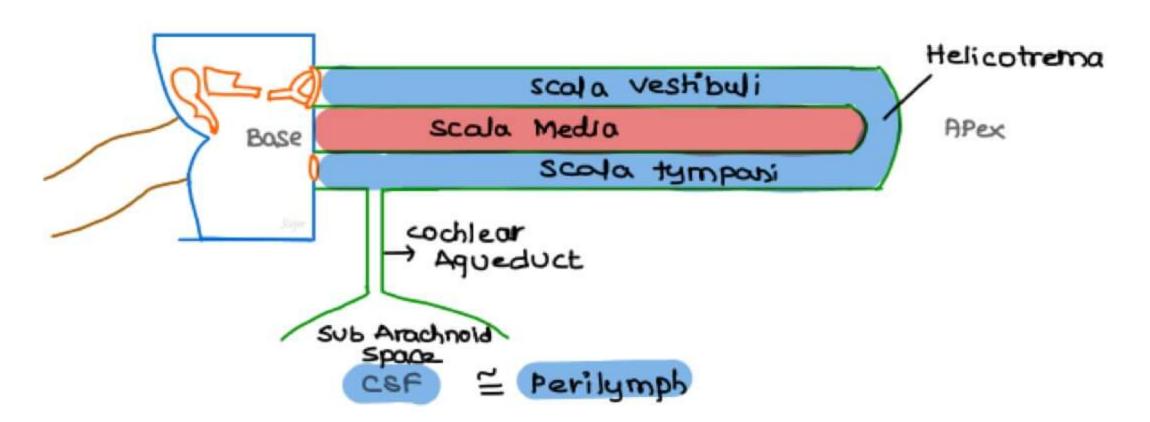
Exchange 3Na+ T 2K+

responsible for ENDO CHOCHLEAR POTENTIAL



ENDO CHOCHLEAR POTENTIAL

- Normal physiological potential
- +80-85 mV
- Not dependent on sound
- DC potential



Endolymph is also produced by Dark cells of Macula

Cochlear Aqueduct: Connects Scala Tympani to Subarachnoid space, Perilymph is same as CSF During meningitis, M/C route of infection from brain to inner ear \rightarrow Cochlear aqueduct





Labyrinthitis

Infection of Inner ear. It can be of 2 types:

- 1. Serous d/t toxins
 - 2. Suppurative d/t bacteria itself
- $\rightarrow C/F$
- a. Sensory Neural Hearing Loss (SNHL)
- b. Vertigo
- c. Tinnitus
- → SNHL is reversible in serous labyrinthitis
- → LABYRINTHITIS OSSIFICANS
 - D/t Suppurative labyrinthitis (Bacteria) → fibrosis → calcification
 - Progressive SNHL
 - IXOC : MRI
 - R_X by cochlear implant

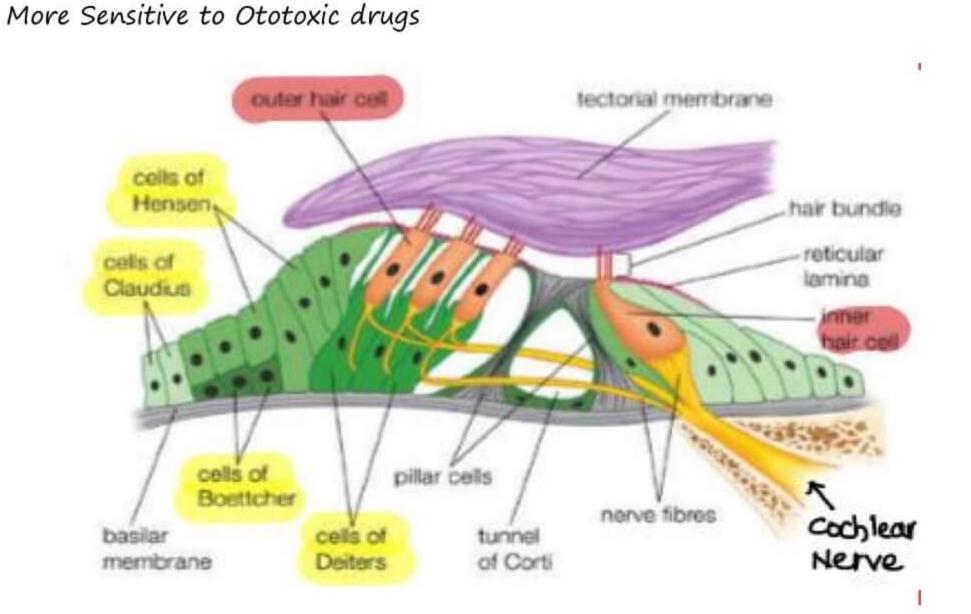
Organ of Corti:

Present on basilar membrane

→ Outer hair cells	Inner hair cells
13-14k in no	3500 in no
More in rows (3-5)	Single row
Tubular	Flask Shaped
Develop late	Develop early

More sensitive to NIHL

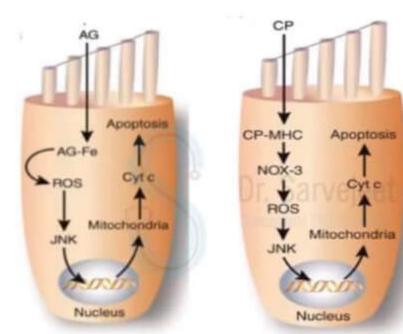
(3000-6000Hz; Max. peak at 4000Hz)





Ototoxic Drugs

- 1. Aminoglycosides (Irreversible)
 - → Streptomycin
 - → Gentamycin Preferential vestibulo toxic
 - → Kanamycin
 - → Amikacin More cochleotoxic



- 2. Loop Diuretics: Furosemide, Ethacrynic Acid → Dose dependent
- 3. Platinum based Chemotherapy drugs: Cisplatin/ Carboplatin [Reversible → Irreversible]
- 4. Macrolides → Erythromycin
- 5. Antimalarial: Quinine / Chloroquine
- 6. NSAIDS Ibuprofen, Naproxen
- 7. Chemicals Alcohol, Tobacco, Marijuana, Carbon monoxide poisoning

Outer hair cells produce - Oto-Acoustic emissions - objective test

- Low intensity sounds in response to a sound stimulus
- Spontaneous OAE: No clinical Significance
- Evoked OAE
 - 1. Transient Evoked OAE (TE OAE) single frequency sound
 - 2. Distortion Product OAE (DP OAE) double frequency sound

OAE is the most sensitive test to detect NIHL (TE-OAE)
OAE is also used as a screening test for Neonatal Deafness

Ototoxicity first affects high frequency sounds, so earlier High Frequency Audiometry was done for Ototoxicity. Now OAE is the most sensitive test to Detect Ototoxicity



Auditory Neural Pathway

E-COLI-MA

E - Eighth nerve (Vestibulocochlear nerve)

C - Cochlear nucleus (Spiral ganglion)

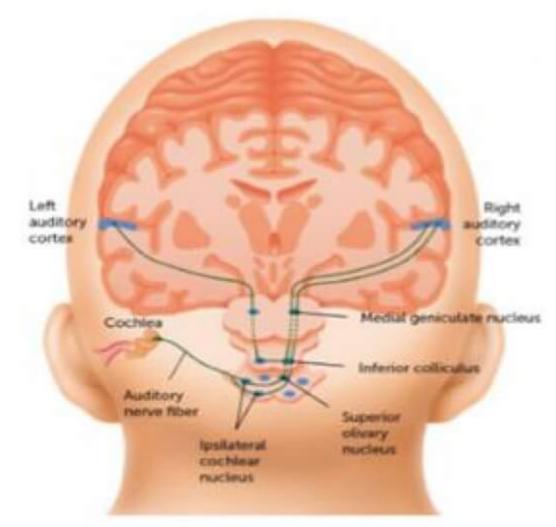
O - Superior Olivary complex (Opposite)

L - Lateral lemniscus (Largest)

1 - Inferior colliculus

M - Medial geniculate body

A - Auditory cortex (Brodmann area 41)



E - 8th nerve COLI → Brainstem MA → Cerebrum

Cochlear nucleus is in brain stem

Cross over of sound takes place at superior olivary nucleus (Through TRAPEZOID BODY)

BRAINSTEM EVOKED RESPONSE AUDIOMETRY (BERA/ABR)

→ Study of electric waves produced along the neural pathway of sound.

→ Produced by distal part of Eighth nerve E - Wave 1

→ Produced by proximal part of Eighth nerve E - Wave 11

> Distal → away from the brain Proximal → near the brain

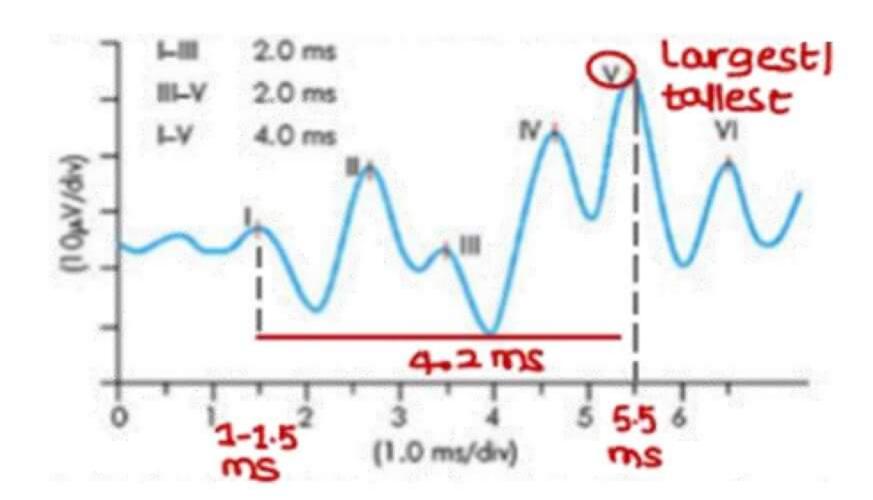
→ Produced from cochlear nucleus C - Wave III

→ Produced from superior olivary complex 0 - Wave IV

→ Produced from Lateral lemniscus L - Wave V

→ Produced from Inferior colliculus 1 - Wave VI

Largest / Tallest/ Most stable / Most significant wave → Wave V





Interpretation of BERA:

- \rightarrow If no waves /not identifiable waves/wave I absent / all waves absent \rightarrow pt. has cochlear hearing loss
- \rightarrow If wave I is present and wave V is absent \rightarrow Retro-cochlear hearing loss and even delayed wave V (Wave I-V interpeak Latency \geq 4.4 ms) \rightarrow Retro-cochlear hearing loss.
- → BERA is used as confirmatory test for neonatal deafness.
 Best test for neonatal deafness → BERA/ABR
- → Also used to detect Malingering
- → BERA is an objective test

Hearing Devices

Hearing Aids:

- 2 types:
- (i) Analog
- (ii) Digital: These can be further classified based upon their placement:
- a. BTE: Behind The ear



b. RIC: Receiver In Canal







c. ITC: In the Canal







d. CIC: Completely in Canal





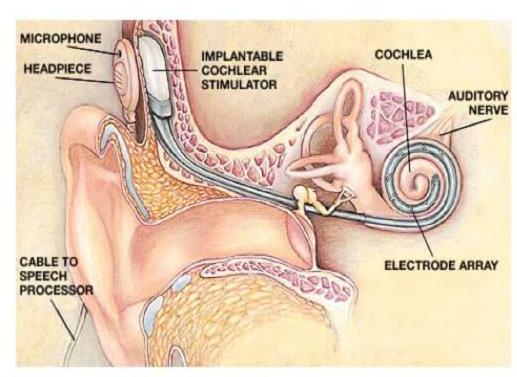
e. IIC: Invisible in Canal





Cochlear Implant





- → Implant an electrode in Scala tympani [Nearest to VIIIth nerve]
- → We enter Scala tympani through cochleostomy near round window or through round window

Parts

1) External body worn part

- 1) Microphone → Receives sound
- II) Speech processor → Converts sound to electromagnetic waves
- III) Transmitter -> Transfers EMW across the layer of skin

2) Internal/ Implantable part -

- → Receiver stimulator → Stimulates electrode array
- → Electrode array: Implanted inside Cochlea (Scala Tympani) and stimulate Eighth Nerve

Indications

B/L SNHL

≥70dB in Adults & ≥90dB in Children (Severe to Profound SNHL)

Condition: - Patient should have tried hearing aid for atleast 6 months



Preference

Post-Lingual Deaf Child is given maximum preference

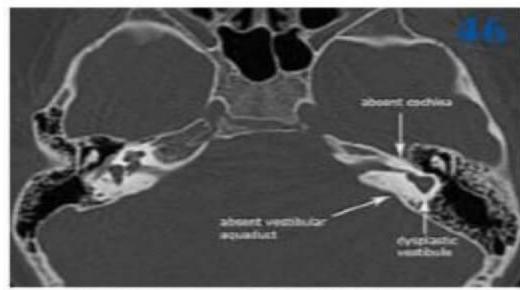
Contraindications

Absolute C/I for cochlear implant

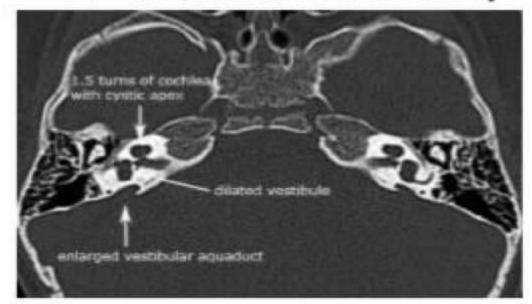
- Michel's aplasia
- Absence of cochlea
- 11. 8th nerve aplasia
- III. 8th nerve tumor
- IV. Scheibe dysplasia (Cochleosaccular dysplasia)
 - M/C congenital anomaly of inner ear

Relative C/I for cochlear implant

- V. Mondini's dysplasia Cochlea has only 1.5 turns Can do cochlear implantation
 - M/C congenital anomaly of Cochlea



MICHEL'S APLASIA



MIZALIASYO 2°INIONOM

2. Auditory Brainstem Implant (ABI)

- → Implantation on brain stem
- → Cochlear nucleus is in 4th ventricle
- → 4th ventricle is entered through Foramen of Lushka



Physiology of Hearing

Binaural hearing \rightarrow Hearing by 2 ears \rightarrow help in sound localization \rightarrow d/t head shadow effect Monoaural hearing \rightarrow Hearing by 1 ear

→ Total surface area of Tympanic membrane = 90 mm²

Effective vibratory area of Tm = 55 mm²

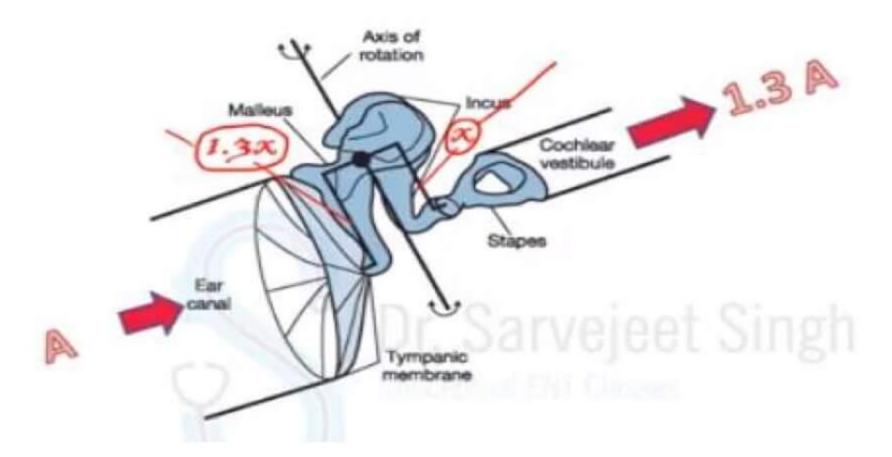
Surface area of stapes foot plate = 3.2 mm²

Areal ratio = 17: 1

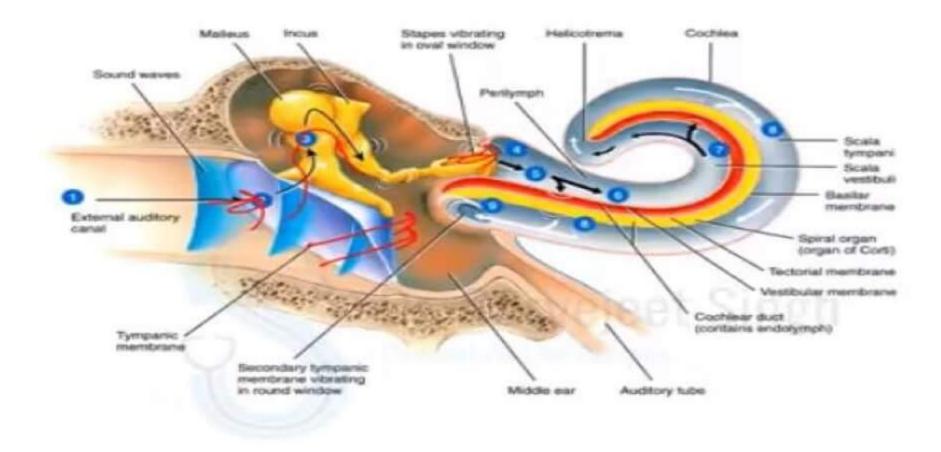
Lever ratio = 1.3:1

(Between length of Handle of Malleus & long process of Incus)

→ TORQUE = Force X length



- → Middle ear transformer ratio (Ossicular Coupling) = 22:1
- → Most mobile part of Tympanic Membrane is periphery of Pars Tensa.
- → Sound travels faster in solids than air due to which the 2 windows are in phase difference
- \rightarrow Middle ear \rightarrow Also Amplifies sound by creating phase difference between Oval & Round Window (Acoustic Coupling)

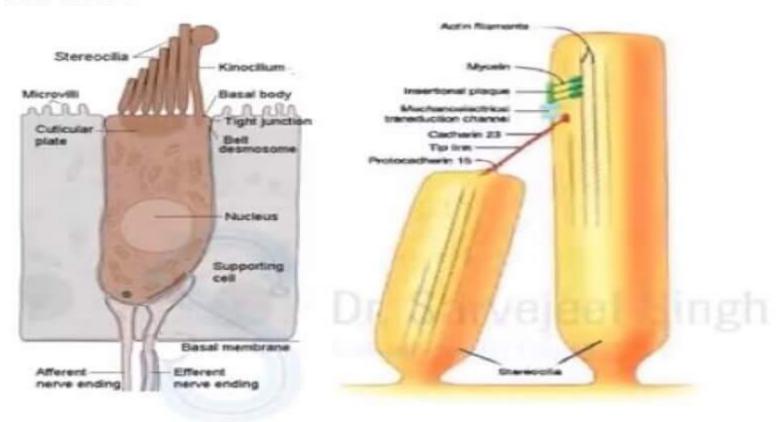


Middle Ear Mechanics: Impedance Matching
Ossicular Coupling + Acoustic Coupling



In inner ear – Sound waves move perilymph in Scala vestibuli → this movement reaches Scala tympani

- → Above Scala tympani there is basilar membrane which has Organ of Corti
- → The stereocilia on Hair cells in Organ of Corti have "tip link" which join one stereocilia to next bigger stereocilia

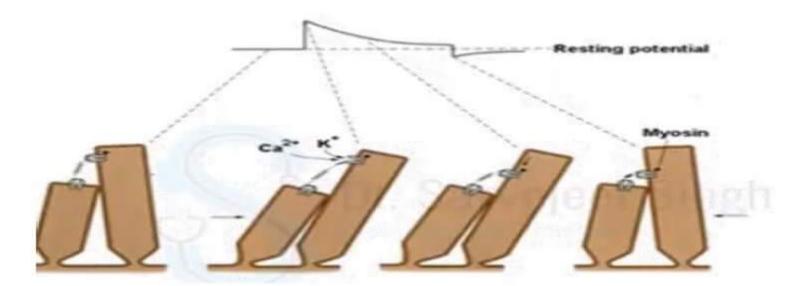


ightarrow Sound waves move the Basilar membrane which in turn move the stereocilia of the hair cells

This leads to stretching of tip link and opening of cation (Positive ion) Channels

Movement of K+ and Ca2+ from Scala media into Stereocilia (Hair cells)

This movement of ions create a potential inside the inner ear called as Cochlear microphonics. Ca²⁺ ions stimulate Myosin protein inside the stereocilia and close the cation channel by moving it down and relaxing tip link.



→ Usher Syndrome

- SNHL + Vision loss (Retinitis pigmentosa)
- Mutation of gene encoding: Cadherin 23 or Protocadherin 15
- Autosomal recessive inheritance
- Finnish population and Ashkenazi Jewish Heritage

→ Cochlear Microphonics

- Electrical potential in the inner ear
- Produced d/t influx of K+ due to opening of Ion channels in response to a sound stimulus.
- This is an AC potential.



→ Summating Potential

- Potential produced inside the outer hair cells in response to movement of K+ ions
- Have higher latency period than cochlear microphonics
- DC potential

→ Eighth Nerve Action Potential

- All or None Phenomenon: Produced Only when Sound stimulus is above hearing threshold

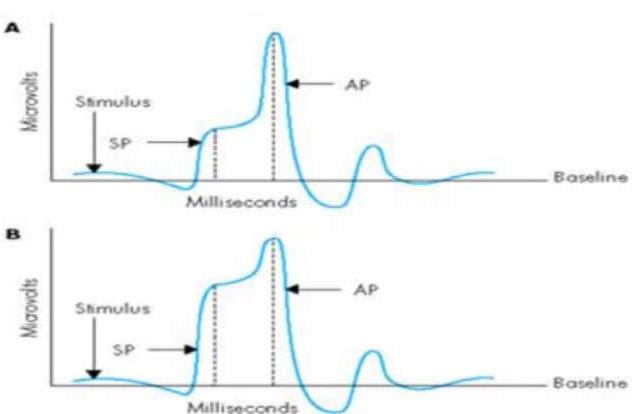
Electrocochleography

→ Summating Potential vs Action Potential

In normal ear \rightarrow SP < 30% AP

In Meniere's disease → SP > 70% AP is confirmatory

→ SP > 45% AP is Indicative



- → Electrocochleography is the confirmatory test for Meniere's disease
- → Invasive investigation: have to make a hole in Tympanic Membrane to place electrode on Round Window

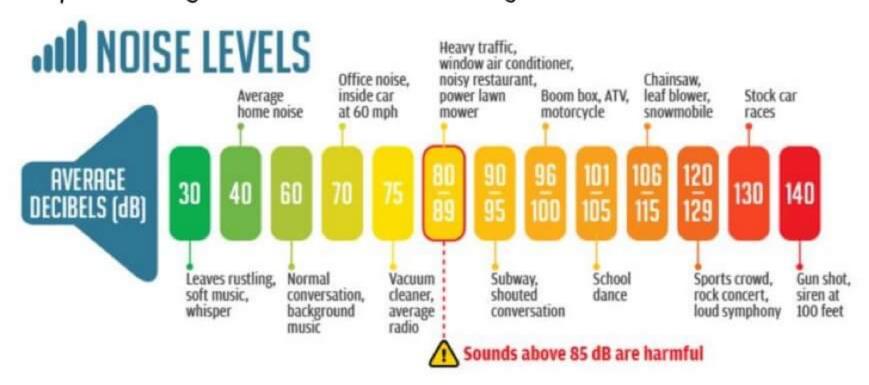
Frequency Localization In Cochlea

- → Normal hearing range → 20-20000 HZ
- → 20Hz is heard at apex of Cochlea, 20,000Hz is heard at Base of Cochlea
- → From every part of cochlea, a neuron comes which carries a different frequency
- → Travelling Wave Theory
 - Peak at which sound heard maximum
 - As frequency increases, the peak shift towards base
 - As frequency decreases, the peak shift towards apex
 - This theory given by Von Bekesy who got noble prize in physiology or medicine [1961]
 - → Neural Pathway is Frequency coded.



Speech Intensity

- → It is measured in dB
- → Sound of whisper 30 dB
- → Normal conversation 45-60 dB
- → Noisy market 60 dB
- → > 80 dB sound is dangerous
- → As per WHO guidelines, sound intensity > 140 dB can lead to deafness even if for few usec



Q. Maximum audible tolerance is??

- A. 90 dB for 6hr
- B. 90 dB for 8hr
- C. 85 dB for 6hr
- D. 85 dB for 8hr

Ans: D

- \rightarrow Maximum audible tolerance of sound according to WHO guidelines and "Factory act 1948" (G.O.I) guidelines is 85 dB for 8 hrs
- → After this WHO follows 3 dB exchange rate i.e if you ↑ the sound intensity by 3 dB you ↓ the time duration by half

Eg. 88 dB - 4hrs

91 dB - 2hrs

→ For G.O.1 there is 5 dB exchange rate

Eg. 90 dB - 4 hrs

- → According to G.O.I there should not be any sound exposure to the factory worker above 110 dB
- → How to protect yourself??
 - Use earphones
 (This protect noise upto 20 dB)
 - 2. Ear plugs
 - Sound protectors
 (This protect noise upto 35 dB)
 - 4. Sound barriers



Tests of Hearing

(1) Tuning fork tests

Mc used \longrightarrow 512 Hz

Not used \longrightarrow 128 Hz [Used in neurological hearing]

Rinne's Test

AC > BC Rinne's +ve \rightarrow Normal, SNHL BC > AC Rinne's -ve \rightarrow CHL

- Disease of Middle ear

- Diseases of External ear



$$AC > BC$$
 \longrightarrow SNHL (U/L)
 $BC > AC$ \longrightarrow Severe SNHL (Dead ear)/ False -ve Rinne's
(Bone conduction by other ear)

256 Hz	512 Hz	1024 Hz	CHL
	±	+	20-30 dB
_	_	+	30-45 dB
s=2	3 - 2	- 2	> 45 dB

→ Most sensitive tuning fork – 256 Hz
Min CHL required to make at least one tuning fork -ve: 20 Hz
(First one to become -ve is 256Hz)
M/C used – 512 HZ

Weber's Test

- → Tuning fork placed in the midline of vertex.
- → Sound heard in the better ear SNHL
- → Sound heard in the bad ear CHL
- → Normal is heard in the center.
- → More sensitive than Rinne's test. (5 db)

Simple funda: Always check the weber's first in tuning fork test questions





4 step method of Tuning fork test

Step 1: Check weber's

Step 2: Check patient's complaints

Step 3: Check Rinne's negative side

Step 4: Rinne's positive both sides (Then it is SNHL)

Absolute Bone Conduction Test (ABC)

→ Comparison test between pt. & examiner Examiner is assumed to be normal Press Tragus to close the air conduction

 \rightarrow Patient BC < Examiner's BC \longrightarrow SNHL Patient BC = Examiner's BC \longrightarrow Normal



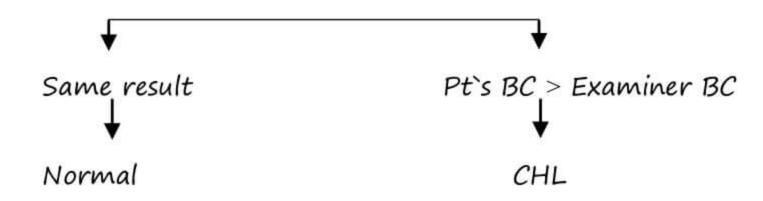
Schwabach Test

→ Comparison test between pt. & examiner Examiner is assumed to be normal No pressing of Tragus

→ Patient BC < Examiner BC — SNHL

Patient BC = Examiner BC

Repeat test \bar{c} reverse order



Siegel's Speculum (M3GF)

Used for Magnification

Mobility

Medication of Tympanic membrane

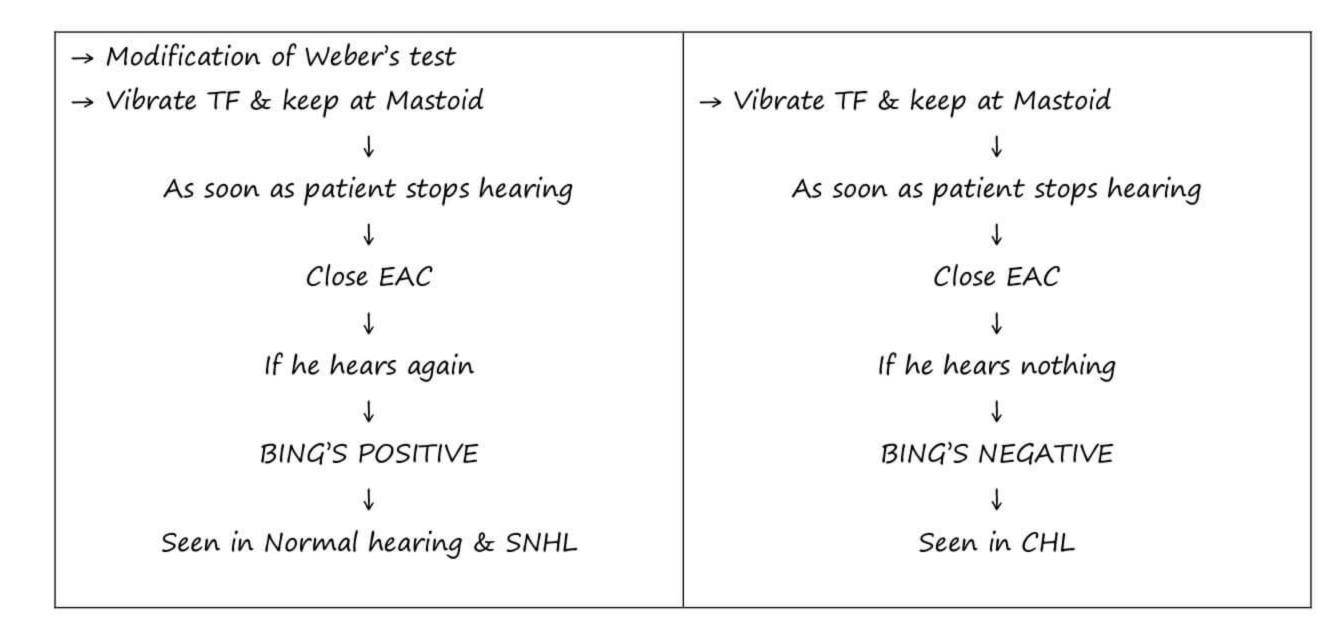
Galle's Test

Fistula Test

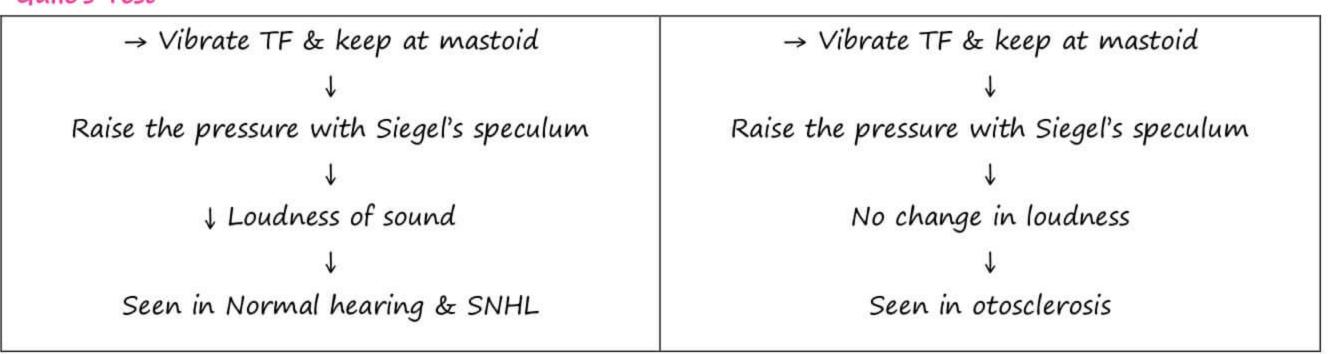
→ Cannot remove F.B.



Bing Test



Galle's Test



For Malingering

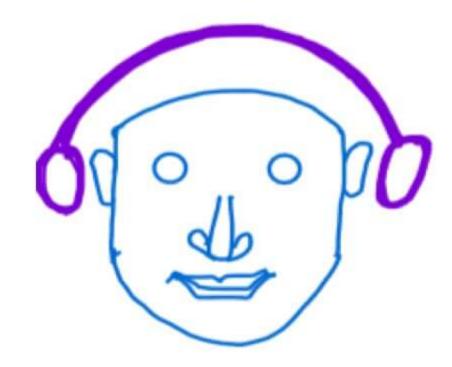
- 1. Chimani- Moos test
- 2. Stenger's test
- 3. Lombard test [B/L Malingering]

(2) Pure Tone Audiometry (PTA)

Can determine

- → Degree/Amount of hearing loss
- → Type of hearing loss
- → Calculates the hearing threshold
- → Minimum intensity at a particular frequency at which a person starts hearing → Hearing/ Auditory Threshold
- → Uses single frequency sounds
- \rightarrow 'O' (Zero) values doesn't mean 'O' in true sense, it is hearing threshold of a normal person. It is different for different frequencies
- \rightarrow AC & BC = 0 for Normal person





Right sideLeft sideRedBlueOOOOAC $\times \times \times$ <<<<<BC>>>>[[[[BC \bar{c} masking]]]]](Sound in non test ear)

Range of SNHL: Clarke's (1981)

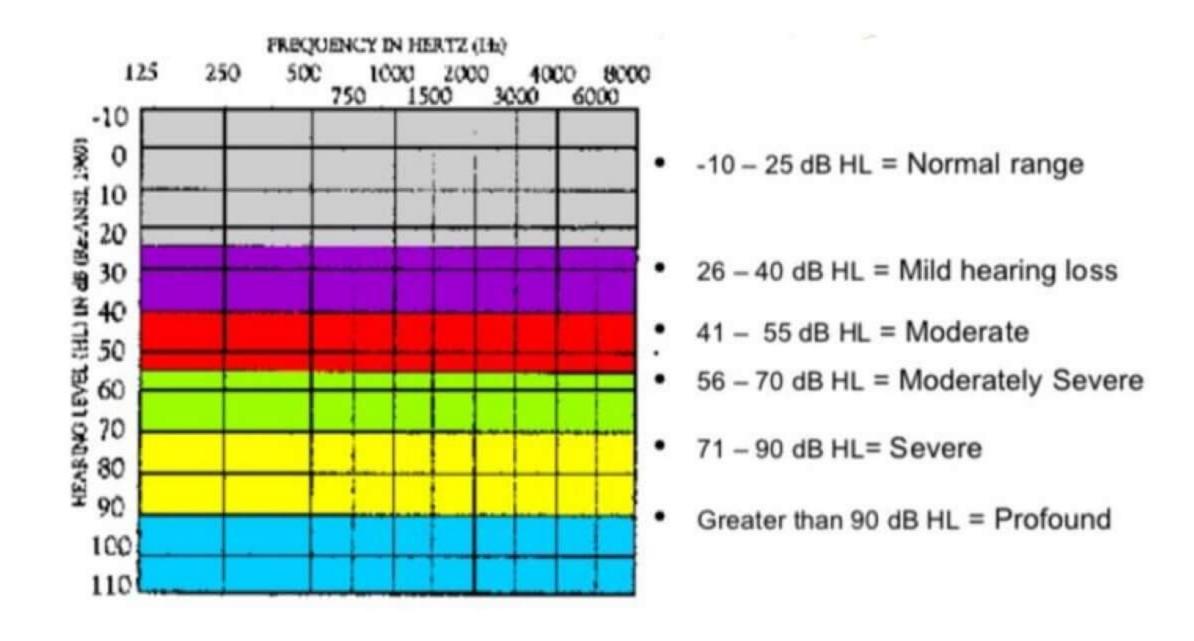
• -10 - + 25 dB $\rightarrow Normal$

• $26 - 40 dB \rightarrow Mild$

• $41 - 55 dB \rightarrow Moderate$

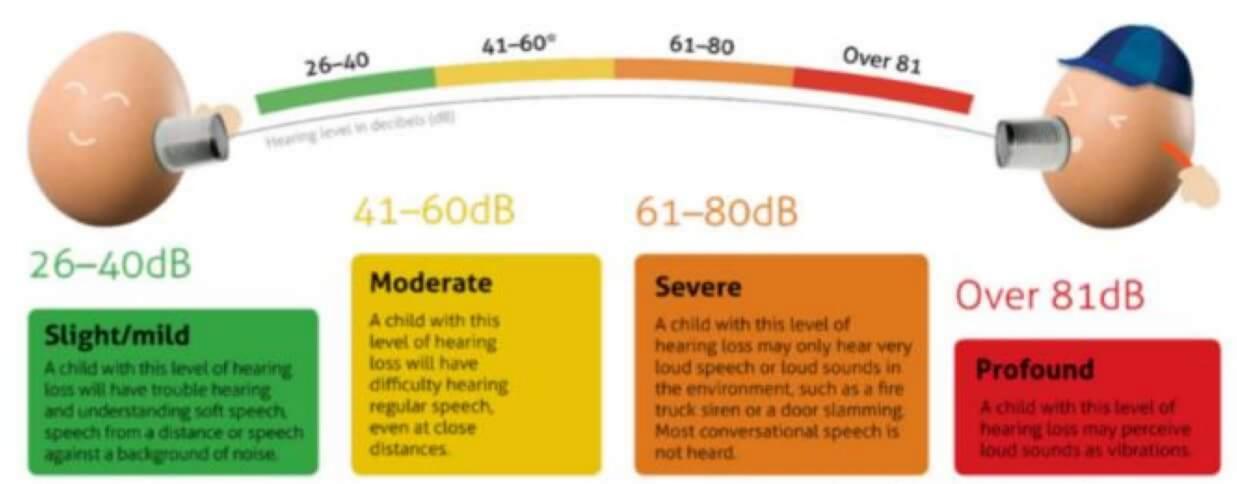
56 - 70 dB → Moderate severe
 71 - 90 dB → Severe

• $\geq 91 \text{ dB}$ $\rightarrow \text{Profound}$



According to WHO

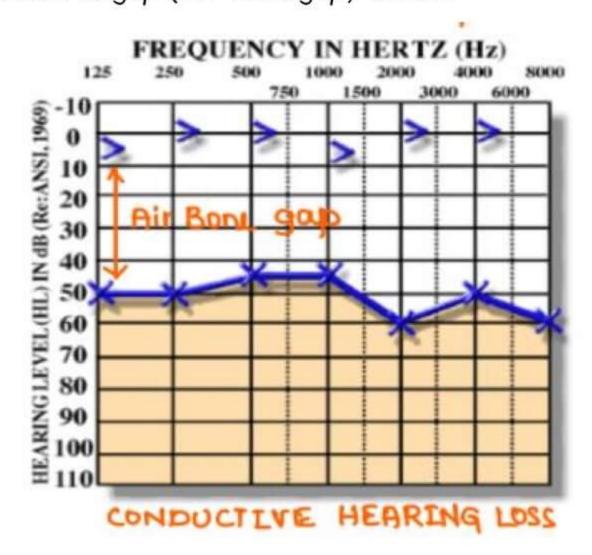
Hearing loss grades



"in the case of moderate hearing loss, the range for children is from 31-60 dB.

Types of Hearing Loss

a. Conductive Hearing Loss: AB gap (Air Bone gap) is seen



b. Noise Induced Hearing Loss (NIHL)

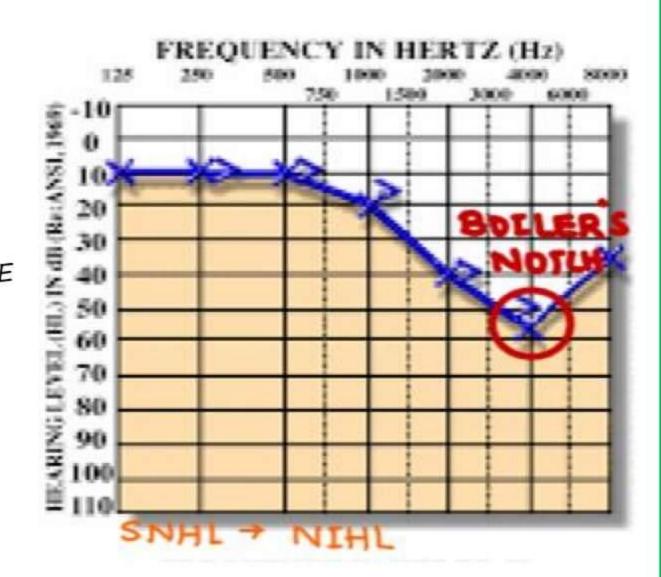
- → Boiler's notch at 4000 HZ
- → Max. Seen at 3000 6000 HZ

Sounds causing Max. Noise induced HL: 2000-3000 HZ

- → Stereocilia of outer hair cells are 1st to be affected
- → Test to find out NIHL even before on audiometry → OAE
- → NO AB gap

Notch is seen only in BC curves

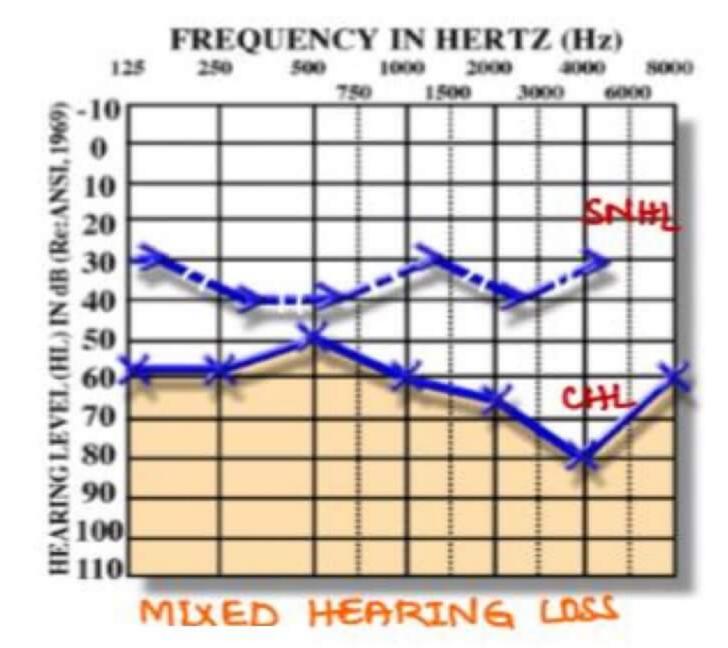
(AC curve Notch are insignificant)



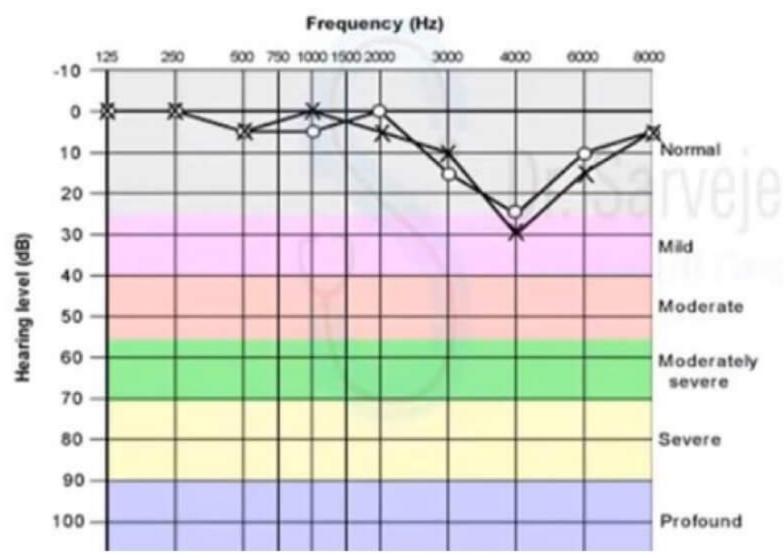


c. Mixed Hearing loss

- → AB gap present
- → Both SNHL & CHL



Q Identify the cause of hearing loss??

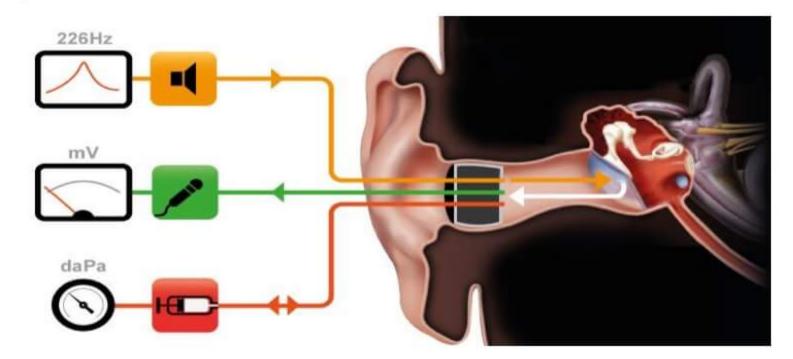


→ B/L Noise Induced Hearing Loss

(3) Impendence Audiometry

Consist of \rightarrow (1) Tympanometry

(II) Stapedial reflex / Acoustic reflex



Types of Curves seen in Tympanometry:



Type A → Seen in Normal Individual

Type As → Seen in Stiffness/ Small/ Sclerosis

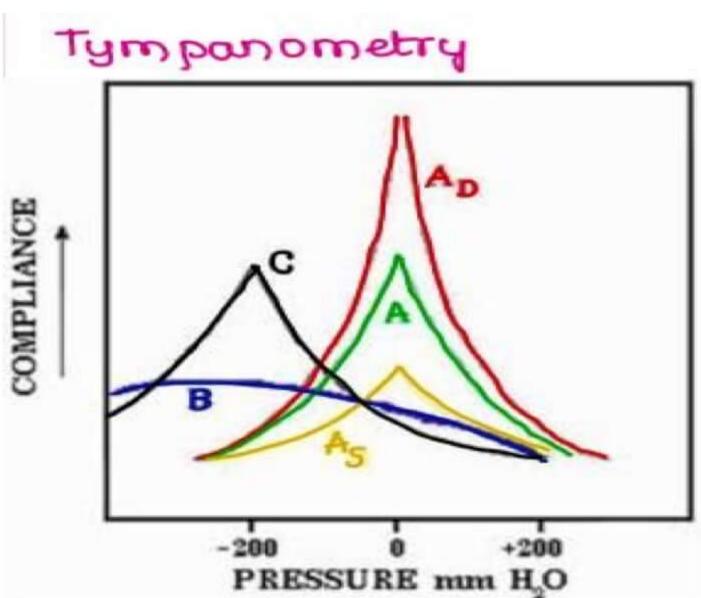
- Otosclerosis [Stapes becomes stiff]
- Tympanosclerosis

Type AD → Seen in Ossicular Discontinuity/ Decrease in stiffness/ Dimeric

Type B → Seen Fluid in the ME

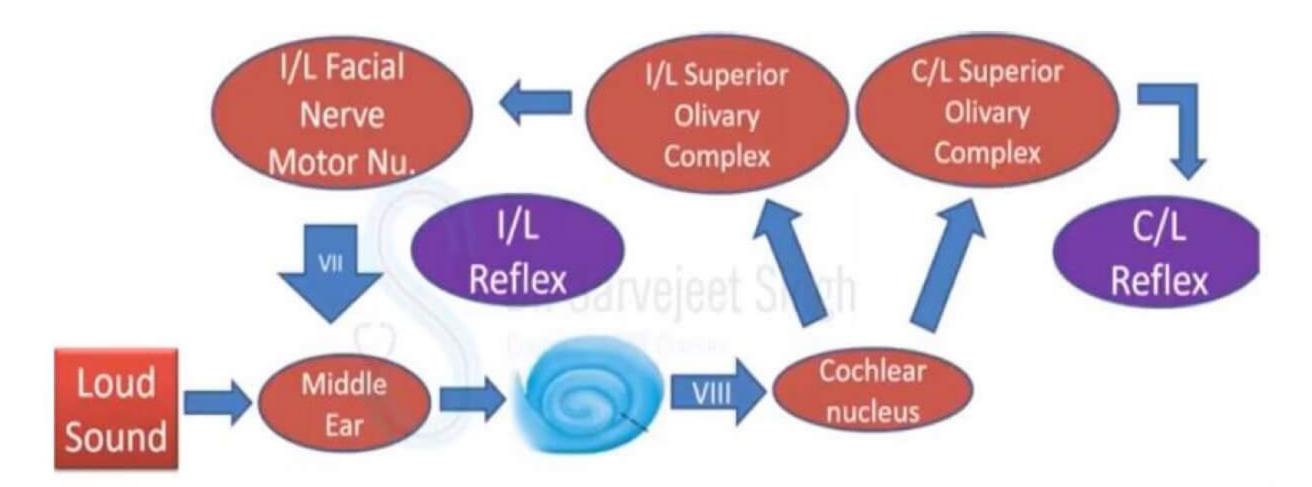
- Glue ear (Serous otitis)

Type $C \rightarrow$ Seen in Retracted Tympanic membrane [We will apply same negative pressure first as TM is retracted]



Stapedial Reflex:-

- → Protects inner ear from noise trauma
- → Stapedius muscle contracts on hearing loud sound



Age wise hearing assessment

Subjective **PURE TONE** AUDIOMETRY PLAY AUDIOMETRY

BOA

Need to consider individual's functional age

Behavioural Observation Audiometry (BOA)

Baby moves head in towards the sound





Very young babies (under 6 months)

Visual Reinforcement Orientation Audiometry (VROA)

Signal can be reinforced to child with visual clues





Infants: 7 months-3 years

Play Audiometry (3-9YEARS)

On hearing sound child has to pick a toy and put it in a bucket



Subjective Objective SCHOOL-AGED + Request responses Tympanometry Acoustic reflex • ECochG BIRTH TODDLER • OAE Condition responses BERA/ABR Observe responses

Need to consider individual's functional age

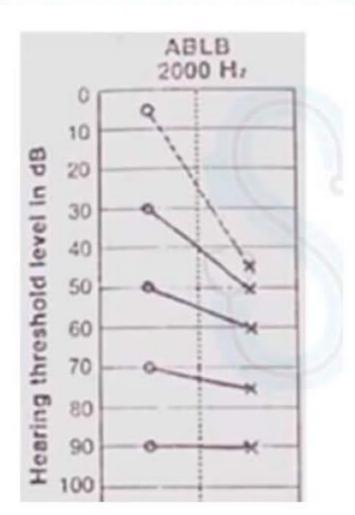


Special Audiometry Tests:

Recruitment

- → Present in cochlear hearing loss
- → The sound that is given appears louder than actual

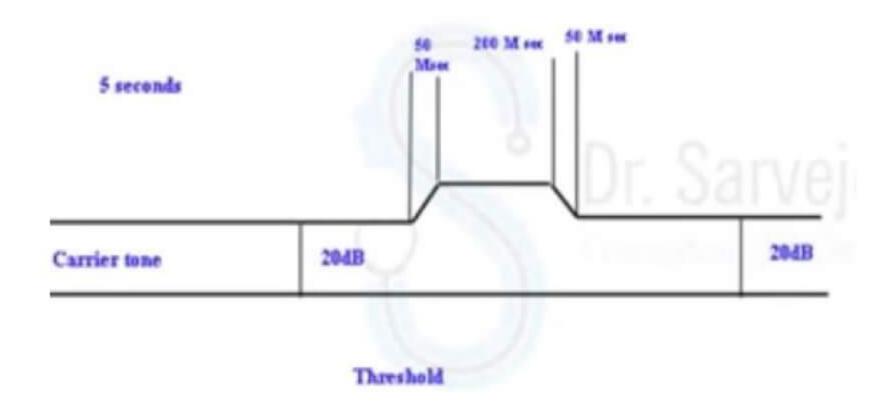
ABLB LADDERGRAM (Alternate Binaural Loudness Balance Test)



Laddergram is converging in Meniere's Disease (Cochlear deafness) due to recruitment

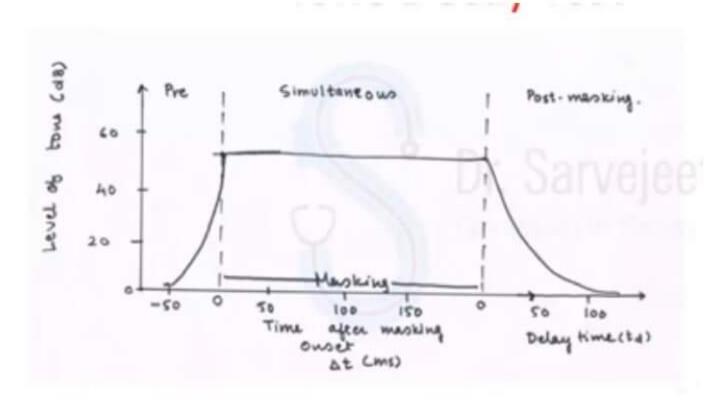
SISI (Short Increment Sensitivity Index) -

- ightarrow Pt. is given 1 dB increasing clicks above the 20 dB of his hearing threshold, and observed how many clicks he can be able identify
- → Test for cochlear hearing loss (Recruitment phenomenon) >70% correct identification



Tone Decay Test

→ If a normal individual is given a sound tone within his hearing threshold or 5 dB within hearing threshold, he should be able to hear the sound for 60 sec



Tone Decay		Pathology
dB	Туре	
0-5	Absent	Normal
10-15	Mild	Cochlear
20-25	Moderate	Cochlear
>25	Severe	Retrocochlear

→ Identifies retro cochlear hearing loss > 25 dB increase in sound intensity so that patient can hear tone for 60 seconds.

Speech Audiometry

Speech Reception Threshold (SRT)

- Minimum intensity at which 50% of spondee (Disyllable with equal stress) words are correctly identified
- Spondaic words

Pancake

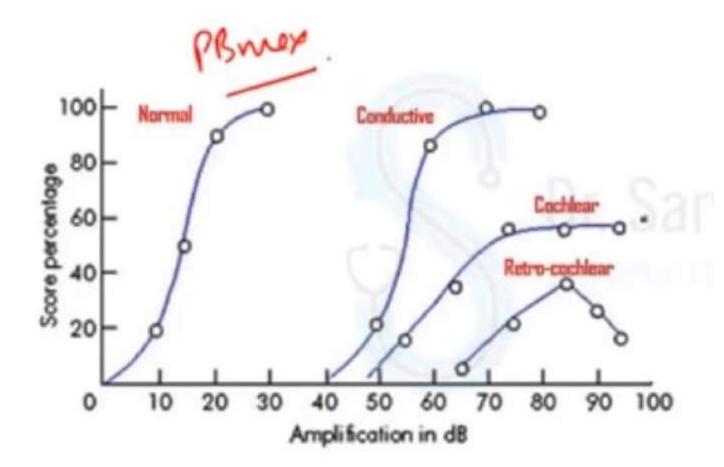
Hardware

Playground

Bat ball

Speech Discrimination Score (SDS)

- Percentage of potentially balanced (Single syllable) words correctly identified at 40dB above
 SRT
- Phonetically balanced words Hit, Pin, Tin, Bin
- PB max score normally reaches 100% 40dB above SRT
- IDENTIFIES RETROCOCHLEAR HEARING LOSS by ROLLOVER Phenomenon



Cochlear Vs Retro-Cochlear

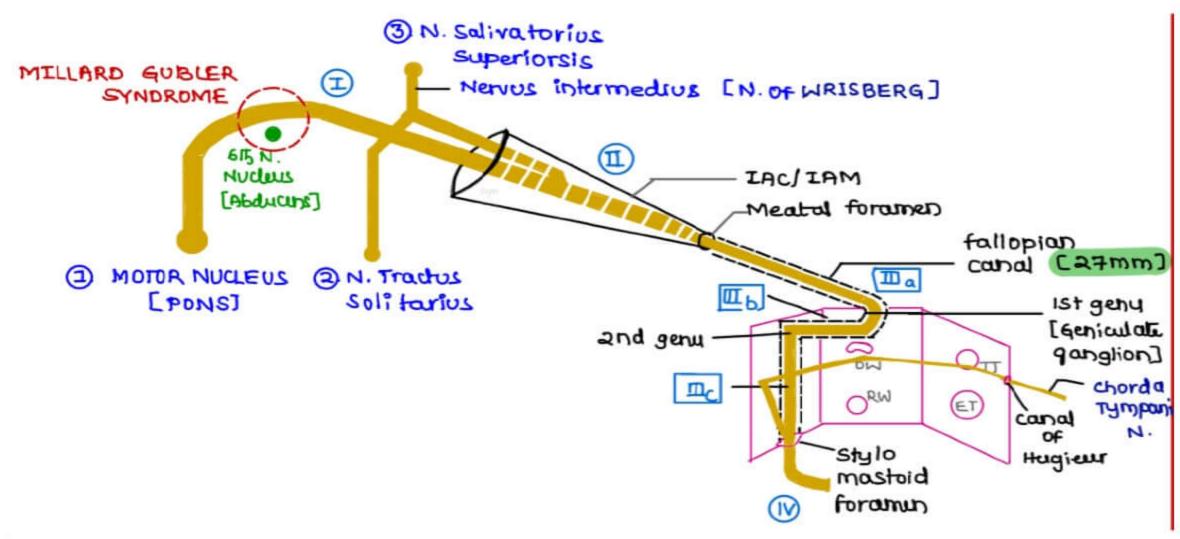
Test	Cochlear	Retro-Cochlear
Speech Audiometry	SDS = 60-80%	<40%, Roll over phenomenon
Tone Decay	Negative(<25 dB)	Positive (>25dB)
S.I.S.I	Positive (>70%)	Negative
A.B.L.B Laddergram	Converging	Diverging
B.E.R.A (Wave V Latency)	= 4.2 msec</td <td>>4.2 msec</td>	>4.2 msec



Facial Nerve

→ Mixed CRANIAL NERVE: Motor, sensory, secretomotor

Sensory part → N. of wrisberg



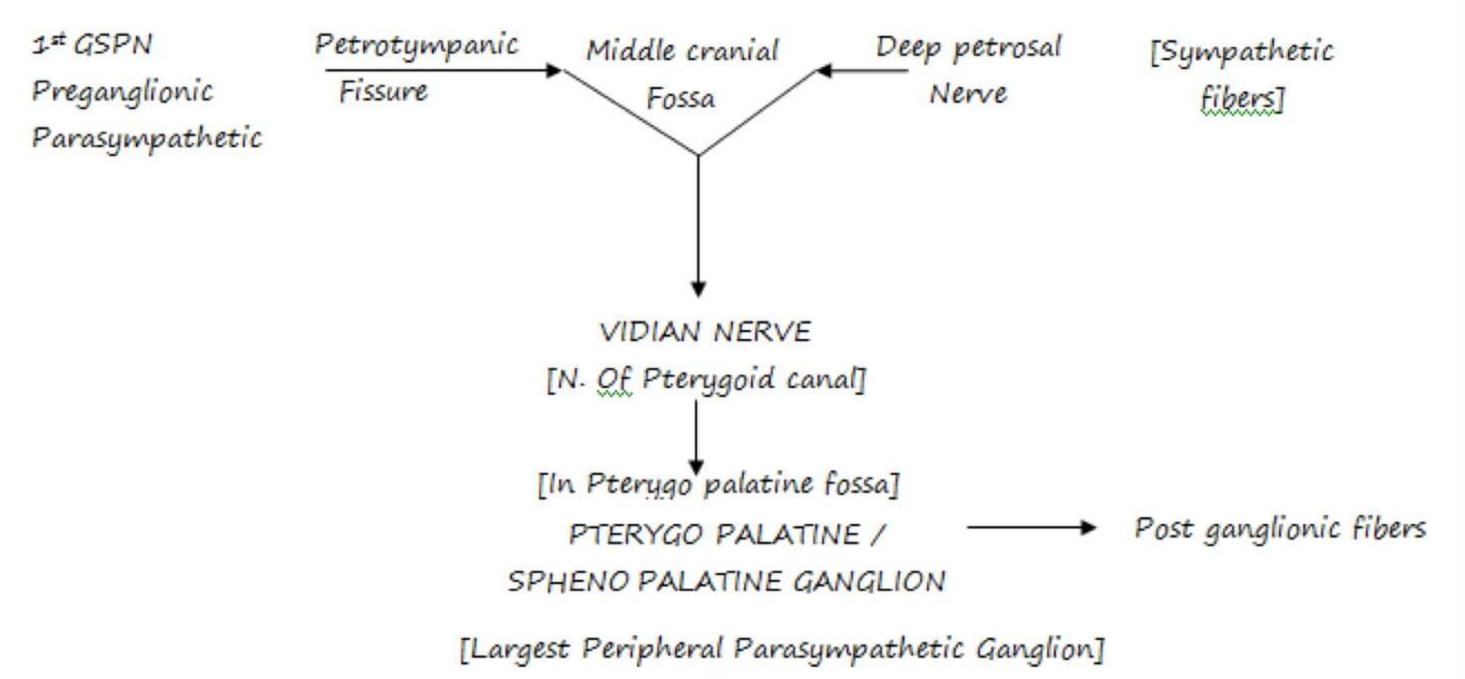
Segments

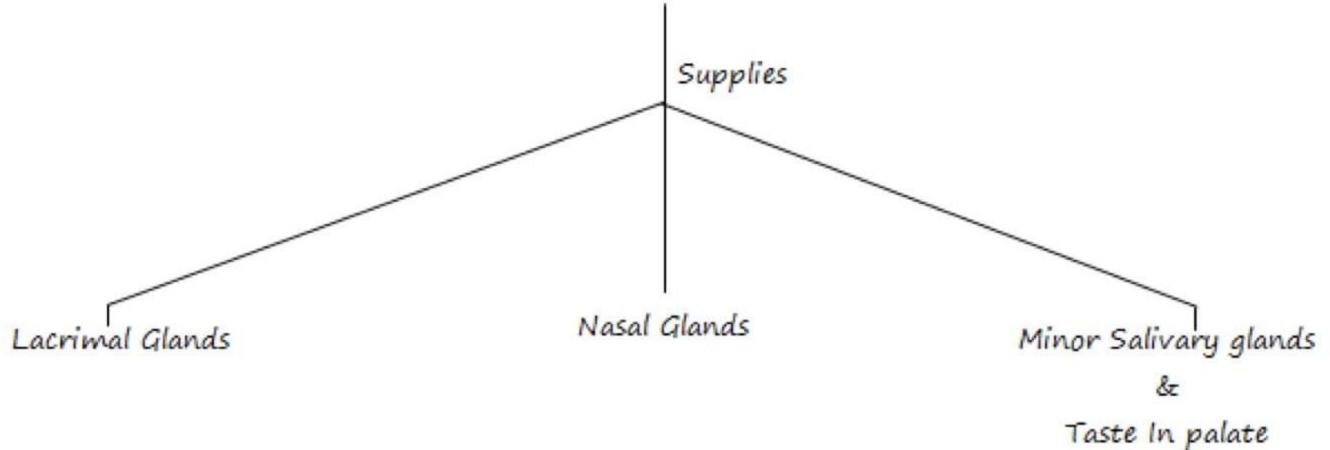
- 1) Intracranial segment (15 20 mm)
- 2) Intra meatal segment (8 10 mm)
- 3) Intra temporal segment / Fallopian canal
 - 3a) Labyrinthine segment [Shortest (3mm) / Narrowest (0.68mm)] 1st genu (3 5 mm)
 - 3b) Tympanic / Horizontal segment 2nd genu (8 12mm)
 - 3c) Mastoid / vertical segment stylomastoid foramen
- 4) Extra temporal segment (15 20 mm)
 - → Fallopian canal 27 mm
 Longest bony canal of any Cranial Nerve.
 - → Facial N. is accompanied by 8th nerve in Intra Auditory Meatus.
 - \rightarrow Millard Gubler Syndrome: Lesion around 6th Nerve nucleus along \bar{c} 7th Nerve nucleus and facial nerve
 - \rightarrow 1st & 2nd genu present in intra temporal segment. Ist genu has Geniculate ganglion

Branches of Facial Nerve

- → No branches from segment 1, 11 & 111a
- → From 1st genu 3 branches
- i) Greater Superficial Petrosal N. → carries preganglionic parasympathetic fibers.
- ii) Lesser Petrosal Nerve
- iii) External Petrosal Nerve



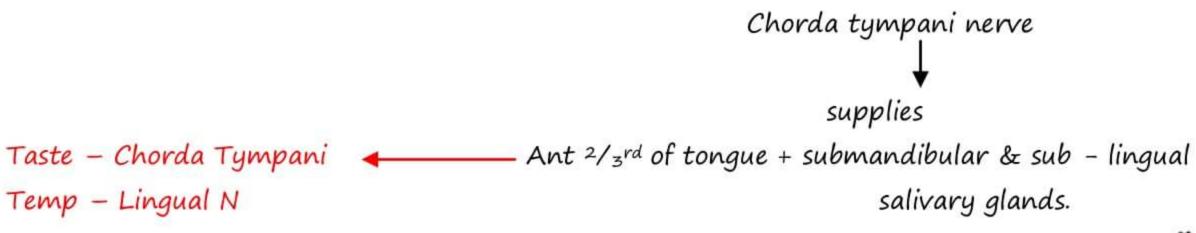




No Branches for III b

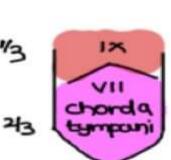
 \rightarrow Just after 2nd genu, Facial N. gives a branch \rightarrow N. to Stapedius [1st motor branch of Facial Nerve]

- → Before Facial N. goes into stylomastoid foramen it gives a branch→ Chorda Tympani nerve (First embryological branch)
- comes in from the posterior wall and comes out from anterior wall through Canal of Hugier



 \rightarrow Sub mandibular & Sub lingual salivary glands supplied by \rightarrow Chorda tympani [VII] Parotid gland supplied by \rightarrow Glossopharyngeal N. [IX]

ightarrow After coming out of stylomastoid foramen, Facial nerve goes into the Parotid gland and divides Parotid gland into 2 lobes





- 1. Superficial lobe
- 2. Deep lobe.
- → In parotid gland, facial Nerve. divides, into 5 terminal branches
- 1. TEMPORAL
- 2. ZYGOMATIC
- 3. BUCCAL
- 4. MARGINAL MANDIBULAR
- 5. CERVICAL

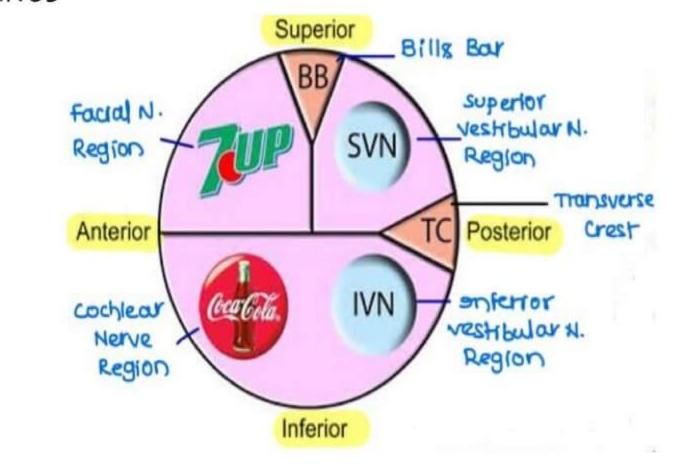
GOOSE FEET ARRANGEMENT

(or)

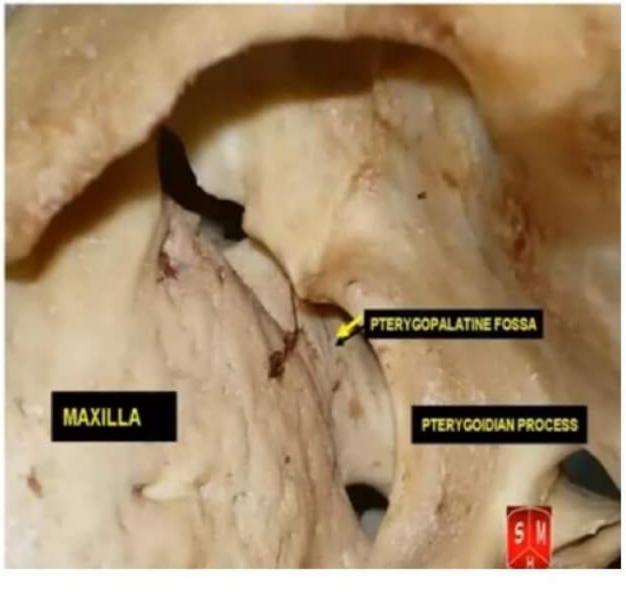
PES ANSERINUS

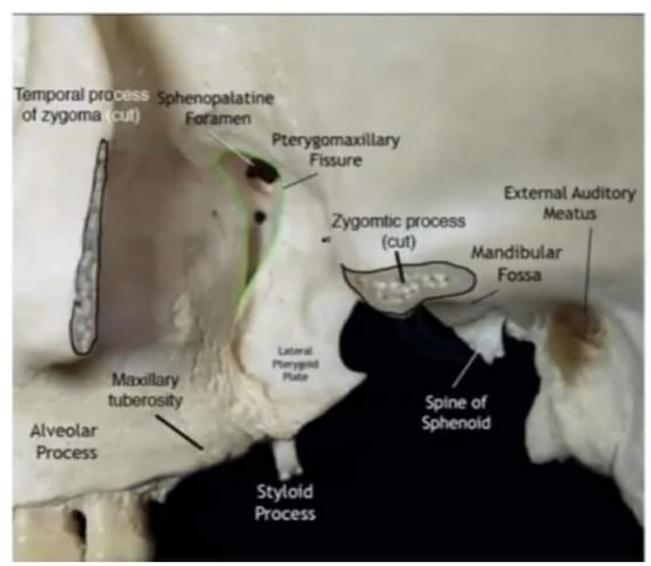
- → Bill's Bar
- -Vertical edge of bone separates 7th CN & Sup. Vestibular N.
- -Named after Dr. William House.
- -Important surgical landmark for Facial N.

(Tympano-mastoid suture)



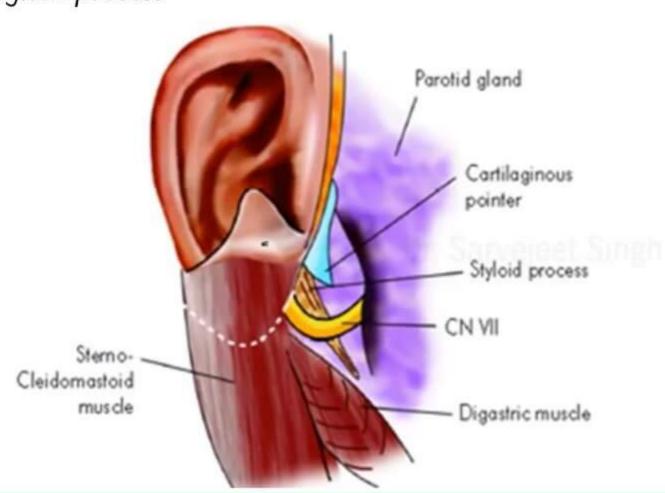
Pterygo Palatine Fossa





SX LANDMARKS OF FACIAL NERVE FOR PAROTID SX

- ightarrow Facial nerve lies 2.5cm deep and internal to the cartilaginous tragal pointer
- → Facial nerve is anterior and superior to the digastric muscle
- → Facial nerve is superficial and anterior to the styloid process
- → Tympanomastoid suture



Test and Diseases of Facial Nerve

Topo Diagnostic Tests

- → Used to identify the location of injury to the Facial nerve
 - 1) Schirmer's Test / Tear Test / Lacrimation Test
 - → ve → Both sides lacrimation is equal → Normal
 - \rightarrow GSPN is Normal \rightarrow Facial nerve is normal till
 - 1st genu.
 - \rightarrow + ve \rightarrow One side lacrimation is decreased
 - → GSPN is affected → injury to Facial nerve is at (or) above 1st genu



2) Stapedial Reflex Test

- → Protective reflex
- → Positive in Normal ears Facial N. is Normal till 2nd genu
- → If it is ve Injury may be at or above 2nd genu

3) Taste Test

- → Ant 2/3rd of tongue → by Chorda tympani nerve
- → Taste present → + ve → Facial N. is Normal till stylomastoid foramen
- \rightarrow Taste absent \rightarrow ve \rightarrow Injury is above the stylomastoid foramen

4) Submandibular Salivary Flow Rate

- → Supplied by chorda tympani
- \rightarrow Salivation on both sides Normal \rightarrow ve \rightarrow Normal \rightarrow Facial N. Normal till

Stylomastoid foramen

- \rightarrow Salivation on both sides different \rightarrow + ve \rightarrow Abnormal \rightarrow Injury is above stylo mastoid foramen
- Q. Patient of RTA, C/o Rt sided VII Nerve palsy.

Schirmer, Stapedial reflex, Taste test → - ve

Site of Injury?

A. Seg IIIa

C. Seg IIIc

B. Seg IIIb

D. Seg IV

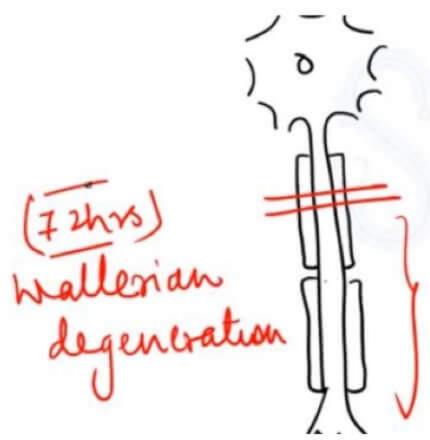
Answer: B- Seg IIIb

Electro Physiological Nerve Testing

- → Electrical stimulus is given proximal to injury Response is recorded distal to stimulus
- → For Facial N.

Normally



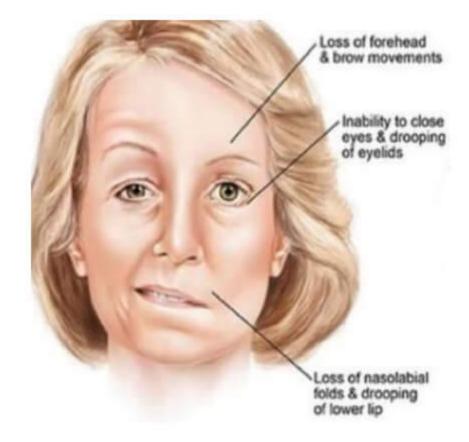


- Test is done after 72 hrs
- Prognostic test
- ightarrow Sometimes, stimulus can be given proximal to injury a response is recorded distal to stimulus even in facial Palsy
- Q. Not a Topo diagnostic test of VII nerve
- a. Schirmer's test
- b. Taste test
- c. Impedance Audiometry
- d. Electro Physiological Testing
- Q. Not a Topo diagnostic test of VII nerve
- a. Schirmer's test
- b. Taste test
- c. Tympanometry
- d. Electro Physiological Testing

Diseases of Facial Nerve

1) Bell's Palsy

- → MCC of Facial Nerve Palsy
- → Ipsilateral
- → Idiopathic (MCC)
- → LMN palsy



LMN

Vs

UMN

- Ipsilateral

- Contralateral
- Forehead not spared
- Lower half of face involved
 (Forehead spared)



Theories

- 1) Viral infection (Herpes)
- 2) Autoimmune theory
- 3) Hypersensitivity / Allergy theory

Edema of facial nerve

But fallopian canal doesn't expand \rightarrow compresses FN \rightarrow Bell's Palsy

Ramsay Hunt Syndrome / Herpes Zoster Oticus

→ LMN Facial palsy + Vesicular rash in external ear + otalgia

70 % of Bell's palsy — Complete recovery
15 % of Bell's palsy — Incomplete recovery

85% recovery without treatment

- → 50% of HZ Oticus → 50% partial Recovery [Poor prognosis]
 - → Involves other nerves also.

Treatment of Bell's palsy

- 1. Steroids
- → Prednisolone for 7 days

No improvement

7 days more

No improvement

Taper the dose & stop

Electrophysiological N. testing

Surgical compression of the nerve

- 2. Antiviral
 - \rightarrow Acyclovir 800mg, 5 times/day, 5 days (Only \bar{c} in 72 hours)
- 3. Eye protective
- → Artificial Tear drops
 Wear goggles, avoid sunny areas, avoid windy areas
 Pad the eye in night times & tape it
- 4. Facial physiotherapy

 \rightarrow 1234 X 7 Days \rightarrow NO improvement

- 1. Steroids
- 3. Eye protection

To be continued for 7 days more

4. Physiotherapy,



- 2. Antivirals → stopped
- → After 2 wKs → No improvement
- 1. Steroids → Taper the dose & stop

 \downarrow

Electrophysiological Nerve testing \rightarrow Not much damage \rightarrow Electro physiotherapy

If Bad Prognosis \rightarrow S_x [Labyrinthine decompression of Facial

Nerve]

Temporal Bone Fracture

Longitudinal Fracture	Transverse Fracture	
→ Along the axis of temporal bone	→ Perpendicular to the axis of temp. bone	
→ Cause → parietal or temporal blow	→ D/t occipital blow	
→ 80% [Mc]	→ 20%	
 Only 10% have Facial N. palsy 	• 50% have Facial palsy	
→ More chances of bleeding from EAC	→ Less chances of bleeding from EAC	
→ More chances of CSF otorrhea	→ Less chances of CSF otorrhea	
Langitudinal fracture Foramen ovale Geniculate ganglion Clivus Jugular foramen Semicircular canala	Transverse fracture Foramen spinosum Foramen magnum External auditory canal	

- → MC Temporal bone fractures → Mixed [54%]
- → Now a days, Temporal bone # are known as

Otic capsule sparing → less complications

Otic capsule involving - more complications

 R_x

RTA, sudden & complete palsy — Facial N decompressing

Relayed & complete — Steroids

Sudden & incomplete — Steroids



House Brackmann Grading Of Facial Nerve Palsy

Grade	Description	Characteristics
	Normal	Normal facial function in all areas
II	Mild dysfunction	Slight weakness noticeable on close inspection; may have very slight synkinesis
III	Moderate dysfunction	Obvious, but not disfiguring, difference between 2 sides; noticeable, but not severe, synkinesis, contracture, or hemifacial spasm; complete eye closure with effort
IV	Moderately severe dysfunction	Obvious weakness or disfiguring asymmetry; normal symmetry and tone at rest; incomplete eye closure
V	Severe dysfunction	Only barely perceptible motion; asymmetry at rest
VI	Total paralysis	No movement

Lesser Petrosal Nerve:

→ Lesser Petrosal Nerve is majorly contributed from Glossopharyngeal Nerve (IX) CN. But some fibers also come from Facial Nerve

Carries preganglionic parasympathetic fibers

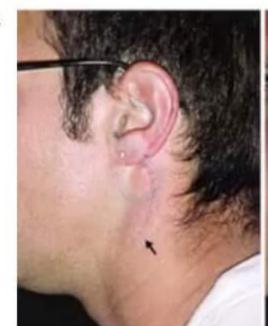
Goes to otic ganglion

[Also taken by 5th nerve (V3) Auriculotemporal nerve]

Supplies the Parotid gland

FREY'S Syndrome (BAILLARGER'S SYNDROME, DUPUY'S SYNDROME, AURICULOTEMPORAL SYNDROME, OR FREY-BAILLARGER SYNDROME) Frey's Syndrome

- \rightarrow Injury to Auriculotemporal nerve which is caring fibers of 9th nerve
- → Injury can take place in Parotidectomy, Rhytidectomy
- → C/F: Flushing and redness on the face while having food





Crocodile Tears Syndrome/Bogorad's Syndrome

- → Injury to Facial nerve before 1st genu of Facial nerve
- \rightarrow C/F: lacrimation while eating.



Diseases of Middle Ear

(Otitis Media)

1) ASOM (Acute Suppurative Otitis Media)/AOM (Acute Otitis Media)

Causes

- → MC causative organism Streptococcus pneumonia
- → Others: H. influenza

Moraxella catarrhalis

- → These bacteria come from nose and pharynx through the Eustachian tube
 - · Functions of Eustachian tube
 - 1. Maintains the middle ear pressure equals to the outside atmospheric pressure.
 - 2. It drains the secretion of middle ear
- → M/C seen in children

Stages

Stage I → Stage of Tubal Occlusion

- → Nasopharyngeal end (or) cartilaginous end of Eustachian tube is blocked
- → ME pressure decreases

1

Tympanic membrane is pulled / retracted

1

Pain, ↓ Hearing O/E Dull & Luster less, Non–shiny No cone of light

Stage II → Stage of Pre-Suppuration

- → Sterile, serous, mucoid secretion
- → TM bulges out, Blood vessels become prominent → CARTWHEEL APPEARANCE
- → O/E pain tes, Hearing les

Stage III → Stage of Suppuration

- → Infection reaches ME
- → Fluid → pus
- → Severe pain + nt
- → Tragus sign ive
- → Red congested bulging TM ready to burst



Stage 1



Stage 2



Stage 3



Stage IV → Stage of Resolution

- → TM burst
- → Pus comes out, pain les
- → M/c site of TM Perforation → Antero inferior quadrant of Pars Tensa
- → After few weeks, perforation heals within 3-6 weeks & hearing becomes normal
- → A healed TM → Dimeric [No fibrous layer]
- → LIGHT HOUSE SIGN → Pus is coming out constantly





Stage 4

RX

Stage I and II → Antibiotics, Analgesics, Nasal decongestant drops

Stage III → Myringotomy (performed in Posteroinferior quadrant)

Rate of growth: PS > PI > AI > AS

Stage IV → 90% → leave alone

Complications

1) Acute Mastoiditis

- → Mc complication
- → Sign Pain & tenderness on / Cymba concha
- → Ironed out mastoid

Rx → IV Antibiotics - 7 days

Battle sign \rightarrow Hematoma over mastoid d/t # of middle cranial fossaGrisseinger sign \rightarrow Pitting edema over mastoid due to Sigmoid Sinus Thrombosis.

2) Coalescent Mastoiditis

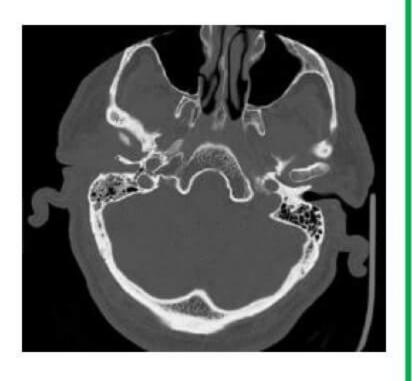
- ME inflammation resolved
- But there is clouding of Mastoid air cells [Filled with fluid]
- Rx IV Antibiotics x 3 weeks (Rxoc)

Sx

groned out







2) Glue ear / Serous Otitis Media (SOM)/Secretory Otitis Media

- → Long standing collection of serous (or) mucoid fluid in the middle ear.
- → Mucoid otitis media or secretory otitis media / Non suppurative otitis media (or) Glue ear
- → Due to tumor, adenoids etc.
- → No pain







Effusion (full of fluid)

- → Conductive hearing loss (MC cause)
- \rightarrow O/E fluid filled middle ear thin bulging TM \bar{c} fluid behind.

Blue TM (Sometimes) → Haemotympanum

Laugier's sign >

Seen in 1. Glue ear [SOM]

- 2. Blood
- 3. Cholesterol granuloma

Glomus tumor (Reddish blue)

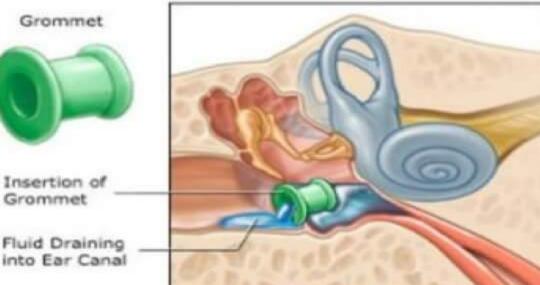
→ Pure Tone Audiometry
AB gap (25 – 30 dB) → CHL
Tympanotomy – B curve



- → Treat the cause (Tumor or Adenoids)
- \rightarrow Myringotomy & drain the fluid (In Anteroinferior quadrant) With grommet insertion.







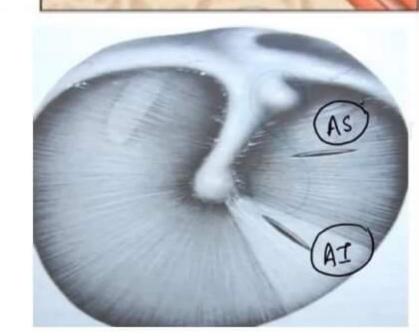
Beer Can Principle

→ 2 Incisions, 1 in Antero superior quadrant for air entry Other in Antero inferior quadrant for pus drainage

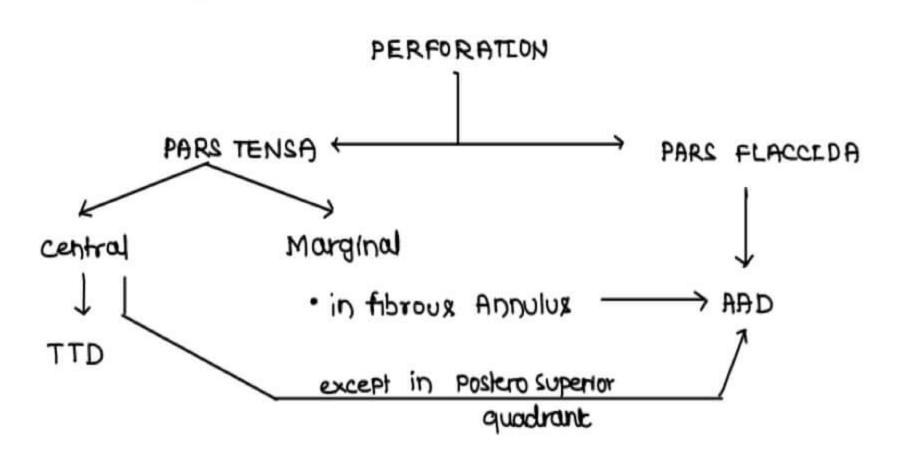


- → Tubo tympamic disease (TTD) / safe CSOM
- → Attico-antral disease (AAD) / unsafe CSOM

Perforation present



Tympanic membrane perforations





Tubo Tympanic diseases / Safe CSOM

- → CHL [AB gap -10-40 dB] [Depends on site & size of perforation]
- → Ear discharge
 - Mucoid (or) mucopurulent
 - Profuse
 - Non foul smelling or foul smelling
 - Continuous or intermittent
- \rightarrow Central perforation +nt
 - small \rightarrow 1 quadrant
 - medium → 2 quadrants
 - large \rightarrow 3 quadrants
 - subtotal \rightarrow 4 quadrants
- → Marginal perforation
 - Total → subtotal + fibrous annulus perforation
- $\rightarrow Rx$
 - 1. Active Antibiotics (For discharge)
 - Tympanoplasty [Myringoplasty +repair of ossicles] → RxOC Myringoplasty [repair of TM]



small perforation



medium perforato



Large perforation



sub total perforato



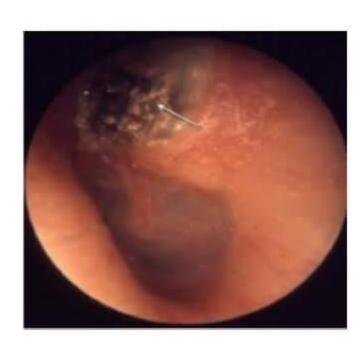
Total perforati

Total per		
Traumatic Perforation	Diseased Perforation	
→ Irregular, rough margins	→ Circular, kidney shaped margins	
→ Blood clots around perforation	→ Smooth margins	
Traumatic	Sieve like perforation [Multiple perforations]	
	→ seen in TB	

Atticoantral Disease / Unsafe CSOM.

Cholesteatoma [Keratoma]

- → Normal keratinizing Stratified squamous epithelium
- → In abnormal place [Middle ear cleft]
- → Types



- 1. CONGENITAL (Mckenzie)
- 2. ACQUIRED
 - a. PRIMARY ACQUIRED
 - b. SECONDARY ACQUIRED

Congenital Cholesteatoma

→ Epithelium is trapped inside, during or before the formation of Middle ear cleft

Acquired Cholesteatoma

→ Epithelium goes into the middle ear cleft after birth

2º Acquired cholesteatoma

- \rightarrow 2° to perforation
- → Migration of squamous epithelium
 - Squamous epithelium From EAC migrates / invades into ME along the marginal perforation
- → Squamous metaplasia
 - Due to chronic infection / inflammation, mucous epithelium in ME transforms into squamous epithelium

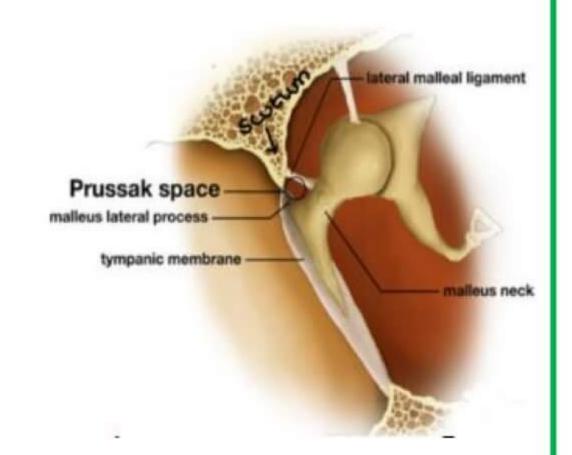
1º Acquired Cholesteatoma

- 1) WITTMACK 'S Invagination / Retraction Pocket Theory
- → PRUSSACK'S SPACE BOUNDARIES
 - → SUPERIOR → Lateral malleal ligament
 - → MEDIAL → Neck Of malleus
 - → INFERIOR → Lateral process of malleus
 - → LATERAL → Pars Flaccida & Scutum



Stage 1

- → Negative pressure in ME +nt → TM pulled in
- → Forms RETRACTION POCKET
 - Has epithelial & mucus layers
 - Epithelium shed off subsequently & get infected
 - → Pus formation occurs
 - Exerts pressure & pocket grows in size & more epithelium dies a cycle continues
 - After a point, it starts eroding surrounding bones
 - Erosion of bones is d/t release of enzymes from lysosomes of dead cells, whichactivates osteoclasts
 - Erosion of scutum is characteristic feature of 1° acquired cholesteotoma
- → Subsequently erodes antrum → ATTICO ANTRAL DISEASE





- → Most accepted theory
- → MC site of 1° acquired cholesteatoma
 Formation → Prussack's Space
- → Prussack 's space located in Epitympanum

Clinical Features

- → Conductive hearing loss
- → Discharge
 - Scanty
 - Purulent
 - Foul smelling
 - Blood stained

Management

- → Pure tone audiometry
- → HRCT in temporal bone
- → Indications for HRCT
 - 1. Complications of CSOM
 - 2. Pediatric patients
 - 3. EAC Polyp
 - 4. EAC Atresia [Congenital abnormalities]
 - 5. Revision Sx
 - 6. Medico Legal case
- → HRCT and MRI both are indicated for → COCHLEAR IMPLANT

Treatment → Modified Radical Mastoidectomy

Mastoidectomy - Types

SIMPLE / CORTICAL / SCHWARTZ MASTOIDECTOMY

- → We don't touch middle ear
- → Just drill in the mastoid
- → Indicated for
 - → Coalescent mastoiditis
 - → Cochlear implant
 - After cortical mastoidectomy, posterior tympanotomy done
 - MC site for cholesteatoma → Ant. inf. to round window
 - KORNER 'S SEPTUM → Persistent petrous squamous suture
 - → Makes difficult to find antrum
 - -> Lateral semicircular canal identification confirms the antrum

Radical Mastoidectomy

- 1. We open the mastoid and middle ear and common wall b/w the two is removed \rightarrow Created common cavity
 - → Preserve Facial nerve



RETRACTO POCKET 1º ACq. cholesteatoma



- 2. We will remove conductive apparatus except stapes
- → Mcc of iatrogenic Facial N. palsy
- → Mastoidectomy

- Mc site

- → Vertical segment just after 2nd genu
- → Indication of Radical Mastoidectomy
- → Malignancy
- → Aim → complete eradication of disease

Modified Radical Mastoidectomy

- → Same as Radical Mastoidectomy but conductive apparatus also preserved, if conductive apparatus has been damaged, it will be repaired
- \rightarrow Primary Aim \rightarrow Complete eradication of disease Secondary Aim \rightarrow Preservation of Conductive hearing never at the cost of primary aim

Canal Wall Down Mastoidectomy

→ Classical MRM

Canal Wall Up Mastoidectomy

→ Common wall (Posterior wall of ME)
b/w mastoid and ME is preserved

→ Advantages

- Hearing results are better d/t normal positioning of TM during Tympanoplasty
- Can go for swimming

→ Disadvantages

- Rate of recurrence is higher
- Advised to follow up by relook
 2nd stage Sx after 6 months

Complications of Otitis Media

- → Acute Otitis Media [AOM] Complications
 - M/C complication of AOM is TYMPANIC MEMBRANE PERFORATION
 - Acute Mastoiditis [2nd mc complication of OTITIS MEDIA]
- → Chronic Suppurative Otitis Media [CSOM/COM] Complications
 - → Ossicular Chain Erosion [Mc complication of CSOM] → CHL
 - → INTRA CRANIAL

EXTRA CRANIAL

INTRA TEMPORAL

EXTRA TEMPORAL

Intra Temporal Complications

- 1. Ossicular Chain Erosion
- 2. Facial Nerve palsy → Rx by Facial nerve decompression
- 3. Labyrinthitis
 - → Serous labyrinthitis → SNHL reversible
 - → Suppurative labyrinthitis → SNHL Irreversible

- \rightarrow Rx by
 - 1. Labyrinthine sedative drugs
 - 2. IV antibiotics (Which can cross Blood brain barrier) like cephalosporins ceftriaxone
- 4. Labyrinthine Fistula
- → Mc site → dome of lateral Semicircular canal

FISTULA SIGN /TEST → Pressing on tragus with finger vertigo or nystagmus occurs

SEIGELIZATION → Tragus pressure with Seigel speculum

→ 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 & Meniere's Disease

HENNEBERT'S SIGN



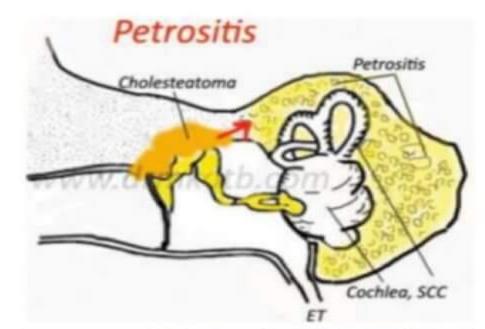
5. Petrositis

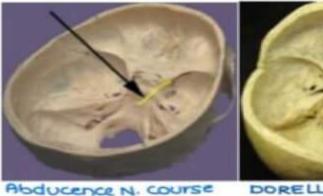
Clinical Features

- → Long standing discharge
- → Deep seated retro orbital pain
- → Diplopia of lateral gaze
 - D/t Lateral Rectus palsy
 D/t Abducent nerve palsy [6th CN]
 [D/t inflamed DORELLO's CANAL [6th n. canal]
- → Gradenigo syndrome

Grade D syndrome

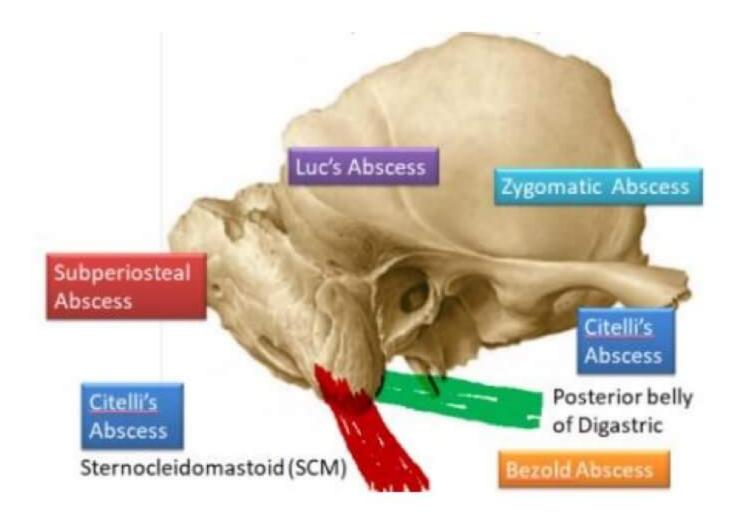
- D → Long standing Discharge
- D → Deep seated retro orbital pain
- D → Diplopia
- D → Dorello's canal







Extra Temporal Complications



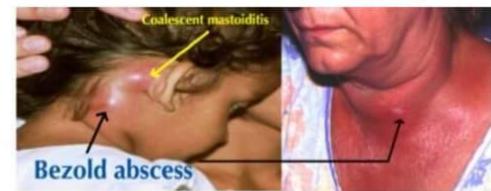
GRADENIGO

SYNDROME

- I. POST AURAL / SUB PERIOSTEAL ABSCESS
- 2. BEZOLD ABSCESS
 - → Present with torticollis [Spasm OF Scm]
- 3. LUC's Abscess
- 4. Zygomatic abscess
- 5. CITELLI's abscess \rightarrow Anterior to mastoid along the posterior belly of digastric or
 - → Posterior to mastoid









LUC'S Abscess





Zygomatic Abscess

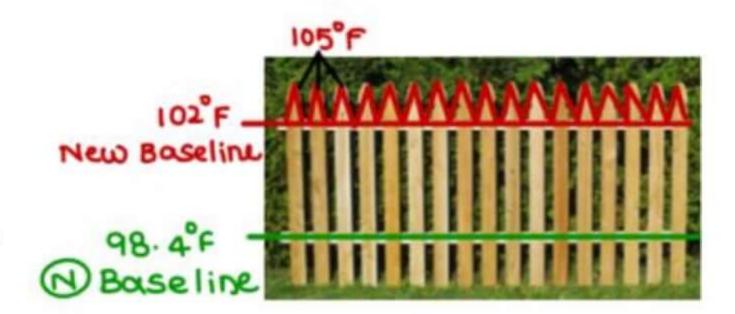


Intra Cranial Complications

- \rightarrow Meningitis (M/C)
- → Extra dural abscess
- → Sub dural abscess
- → Brain abscess
- → Otitic hydrocephalus
- → Sigmoid / lateral sinus thrombosis

Sigmoid / Lateral Sinus Thrombosis

- $\rightarrow C/F \rightarrow Pallor$
 - → Headache
 - → Picket Fence Fever
- · Temperature does not come to normal base line
- Remittent fever
 [Intermittent fever seen in malaria]



Management

1) Tobey Ayer / Queckenstedt test.

- → Compression over IJV on normal side, increases CSF pressure in lateral sinus thrombosis
- → Invasive test

2) Crow beck test

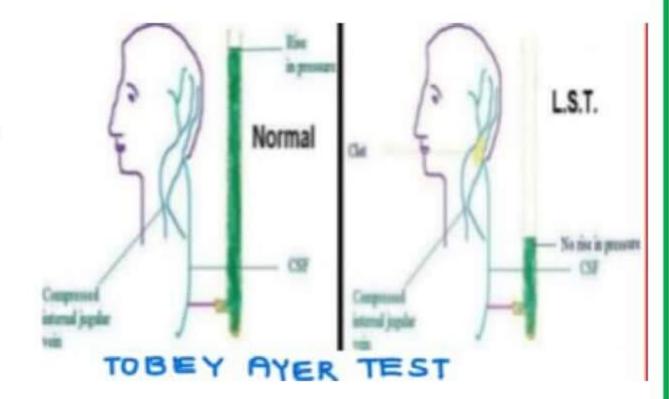
→ Compression over IJV on same side leads to engorgement of Retinal veins.

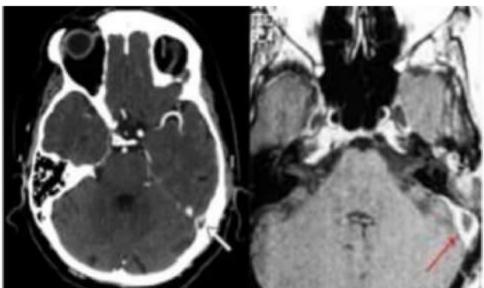


- → Empty Delta sign
- → Confirmatory

Rx

Surgery + IV Antibiotics







Empty delta Sign

crow beck Test



Otosclerosis

ENCHONDRAL BONE

 \longrightarrow

SPONGY BONE

[Most commonly at Fissula ante Fenestram]

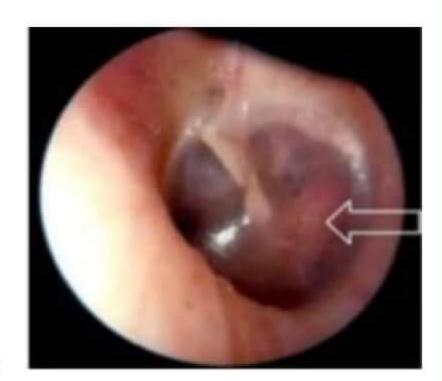
Unorganized Bone

Engulfs the Stapes foot

plate

↓ Hearing

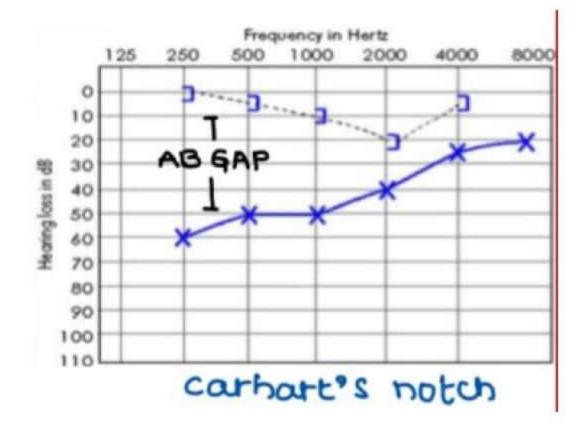
- → Predominant in Females (20-30 yrs).
- → Increased Incidence in pregnancy.
- → AD (50% pts have +ve family history)
- \rightarrow B/L hearing loss.
- → Tympanic membrane
 - Usually normal
 - Sometimes, show flamingo pink colour → Schwartz sign



Seen when enchondral bone changes to spongy bone (Active/Early phase of disease) > Otospongiosis

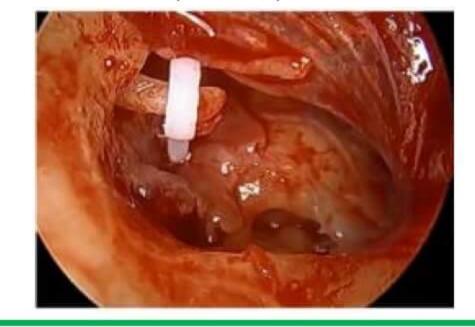
- → DX
- 1) Pure Tone Audiometry
- → Otosclerosis is a disease of conductive hearing loss

DISEASE		AB GAP
Som	\rightarrow	25 - 30 dB
Tm Perforation	\rightarrow	10 - 40 dB
Tran Perforat + ossicular chain erosion	\rightarrow	45 dB
Tm intact + ossicular discontinuity	\rightarrow	54 dB
Oto sclerosis	\rightarrow	Upto 60 dB



CARHART'S NOTCH \rightarrow Dip present at 2000 Hz in bone conduction seen [BOILER 'S NOTCH \rightarrow (In NIHL) dip at 4000 Hz in bone conduction seen

- 2) Impedance Audiometry: As curve seen
- 3) Stapedial reflex is absent b/c stapes footplate is fixed.
- → Paracussis willisii Pt. hears better in noisy places
 Also seen in pts. with B/L CHL.
- → Tx :-
- Stapedectomy → Remove stapes & graft is placed at the oval window & piston is placed.
 → Risk of SNHL present
- 2) Stapedotomy (R_xOC) \rightarrow Hole made in stapes footplate and anchored with piston





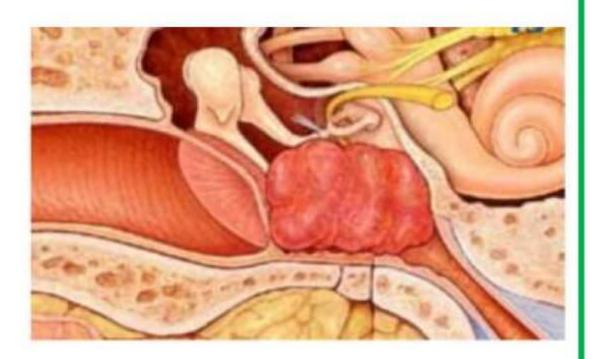
Glomus Tumor

- → MC benign tumor of middle ear.
- → Arises from jugulotympanic paraganglions (In association with Jacobson's and Arnold's nerves)

Paraganglionic cells

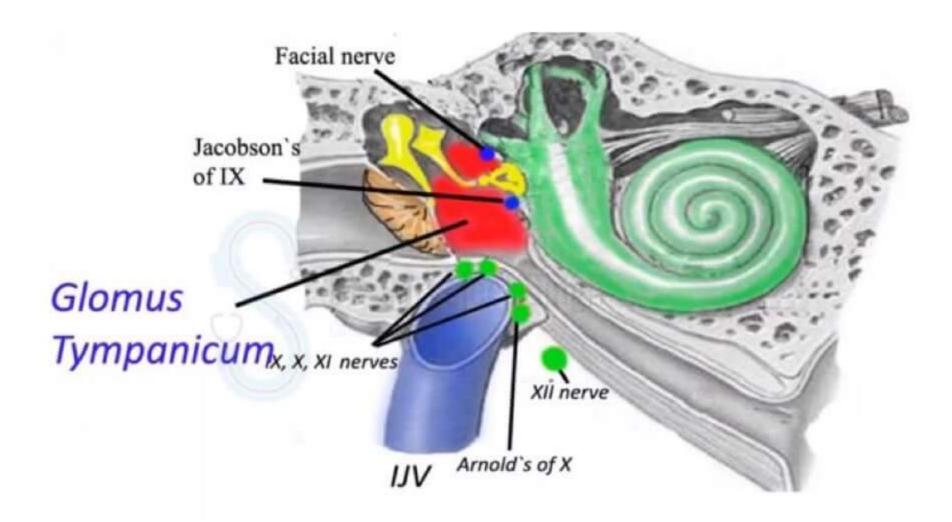
Glomus Jugulare

Glomus Tympanicum



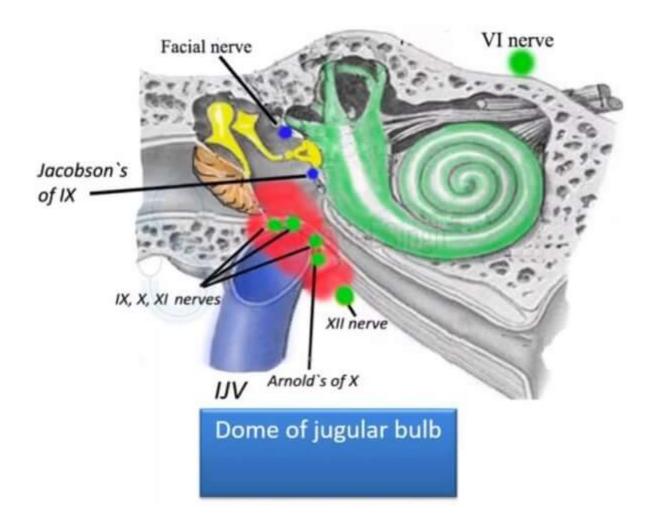
→Glomus Tympanicum

- Arises from the promontory along the course of jacobson's nerve
- Leads to facial nerve palsy most commonly



→Glomus Jugulare

- Arises from the dome of jugular bulb
- Leads to palsy of IX , X , XI , XII cranial nerves



→ Bleeding Aural Polyp

- If there is a bleeding polyp in EAC then, biopsy is absolutely contra indicated
- → Glomus tumor is locally invasive & highly vascular
- ightarrow MC blood supply of glomus tumor Inferior tympanic branch of the Ascending pharyngeal artery

→ C/F of glomus tumor

- Age: 40-50yrs

- Female: Male ratio - 5:1

- Symptoms : CHL

Pulsatile tinnitus present

· Profusely bleeding polyp in EAC

→ O/E - RISING SUN / AQUINO / BROWN SIGN

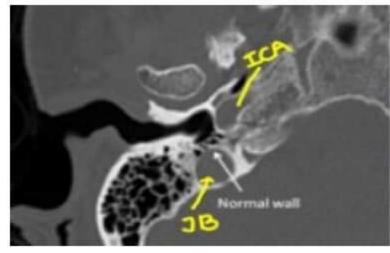
Rising sun sign → Reddish blue rising from hypotympanum

Aquino sign \rightarrow Jugular pressure \bar{c} finger leads to \downarrow Tinnitus.

Brown sign -> On seigelization, tumor blanches & becomes white



- → Sometimes Blue Tympanic membrane is seen.
- → CECT (IOC)
 - Confirmatory Test.
 - Phlep sign seen on CT. (Inability to distinguish b/w internal carotid Artery & jugular bulb d/t
 erosion of caroticojugular crest.)





→ Staging of glomus tumor

Fisch Classification (Glomus Tympanicum)

TYPE A - Middle ear cleft

TYPE B - Tympano mastoid area

TYPE C -Infra labyrinthine compartment / petrous apex

C1: Limited involvement of vertical portion of carotid canal

C2: Invasion of vertical portion of carotid canal

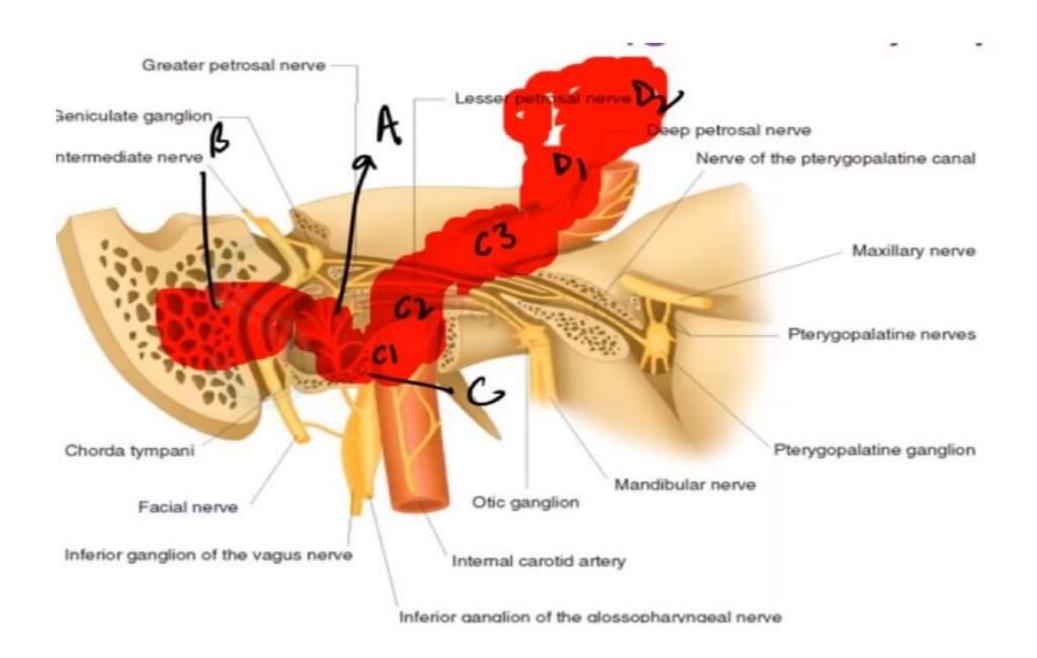
C3: Invasion of horizontal portion of carotid canal

TYPE D -Intra cranial involvement

D1: I/C extension (< 2cm in diameter)

D2: I/C extension (> 2cm in diameter)





RX

- → Surgical excision (TOC)
- → 10% Rule → 10% multicentric
- → 10% familial
- → 10% secretory (Secretes catecholamines)

Diseases of Inner Ear

Congenital Deformities

1. Michel's Aplasia

- → Absence of cochlea
- → Absolute C/I of cochlear implant

2. Scheibe's Dysplasia (Cochleosaccular Dysplasia)

- → M/C congenital anomaly
- → Absolute C/I of cochlear implant

3. Mondini's Dysplasia

- → M/C congenital anomaly of cochlea
- → Cochlea have only 1.5 turns
- → Can do cochlear implantation

4. Alexender's Dysplasia

- → Basal turn of cochlea is absent
- → Absolute C/I for cochlear implant

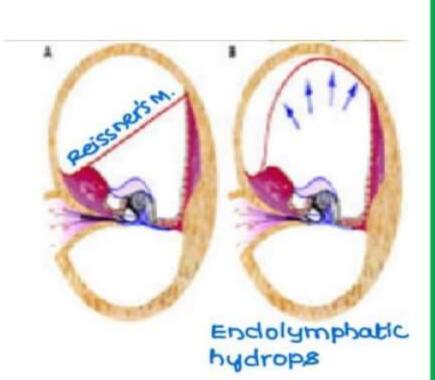
Meniere's Disease / Endolymphatic Hydrops

- → Endolymph Normal Physiology
 - → Secreted by stria vascularis
 - → Transported to endolymphatic sac by endo lymphatic duct
 - → Endolymph absorbed by endolymphatic sac

→ Endo Lymphatic Hydrops

Collection of endolymph in inner ear due to

- Excessive production
- Blockage of duct
- Defective absorption
- → D/t ting pressure, at some point Reissner's membrane breaks, & Causes
 - → VERTIGO [d/t K+ entry into perilymph and causes irritation]
 - \rightarrow SNHL [d/t ion gradient imbalance]
 - → Tinnitus / Aural fullness



 \rightarrow After some time, Reissner's membrane heals, ion gradient returns \rightarrow Hearing comes back to normal

K+ restores in endolymph → Vertigo subsides

- → And again the above cycle continues, leading to
- 1. Episodic Vertigo
- 2. Fluctuating SNHL

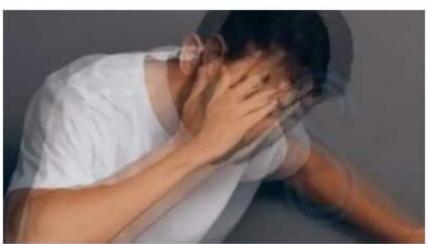
TRIAD

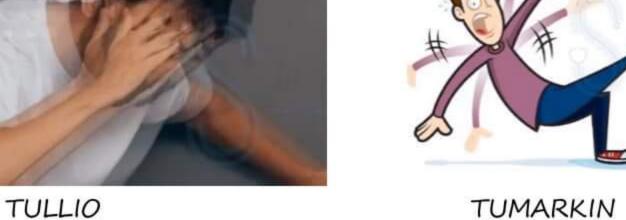
- 3. Tinnitus [Remains even btw 2 episodes]
- 4. Aural Fullness
- → U/L disease, Common in 35 40 yr, Male

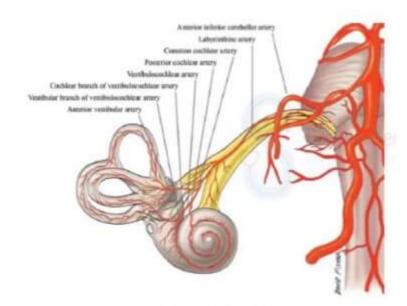


Other Phenomenon:

- 1. Tullio's Phenomenon: Loud Nose Precipitates Vertigo
- 2. Tumarkin's Crisis: Sudden Fall Attack
- 3. Lermoyz syndrome: Vertigo Relieves SNHL
- 4. Hennebert's Sign: Fistula Test False +ve







LERMOYEZ

Diagnosis

1. Pure Tone Audiometry → SNHL

IV glycerol [1.5 ml/kg] [Glycerol test]

Repeat Puretone audiometry after 30 min

Hearing improved > 10 dB

MENIERE'S disease

2. Electro cochleography → Confirmatory test

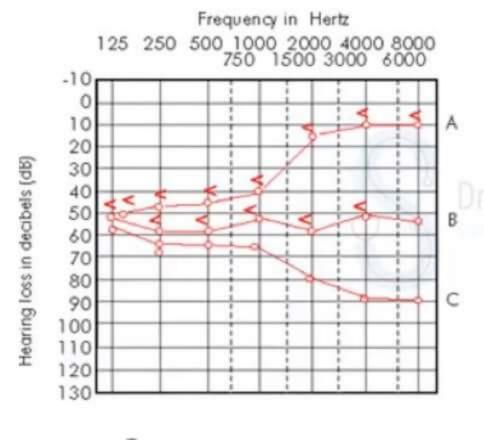
Pure Tone Audiometry

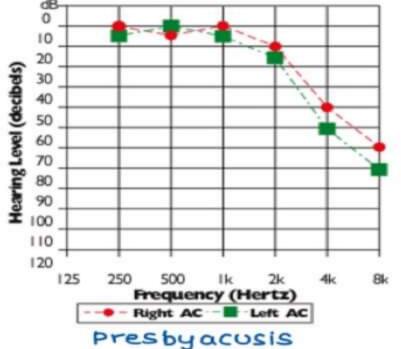
- A. Early Meniere's disease → Rising curve
- → More hearing loss at low frequencies
- → High hearing loss at less frequencies

B. Late Meniere's disease → Sloping curve → More hearing loss at high frequencies → U/L sloping curve

[B/L Sloping curve → Presbyacusis

→ Ototoxicity]

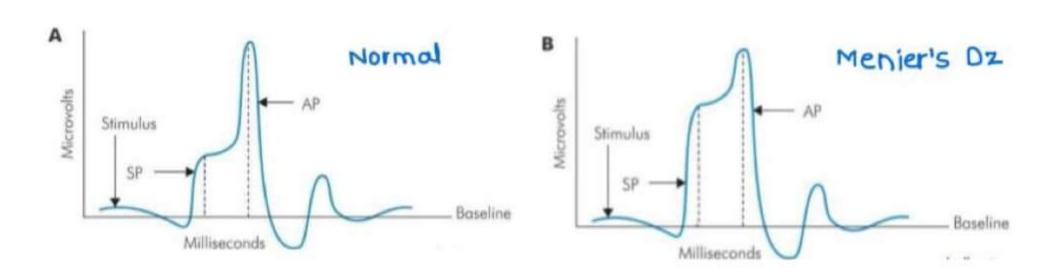






Electro Cochleography

- → Confirmatory test
- → In Normal ear, summating potential (SP) < 30 % AP
- → In Meniere's disease, SP > 70% AP
- → Invasive procedure



TX

- 1) Acute episode: Labyrinthine sedatives
- II) Maintenance phase
- a) Medical
- → k⁺ sparing diuretics
- → B Blockers
- → Antihistamines cystaminics
- b) Surgical
- → Conservative: Endolymphatic sac decompression, vestibular neurectomy
- → Radical: Surgical labyrinthectomy

 \downarrow

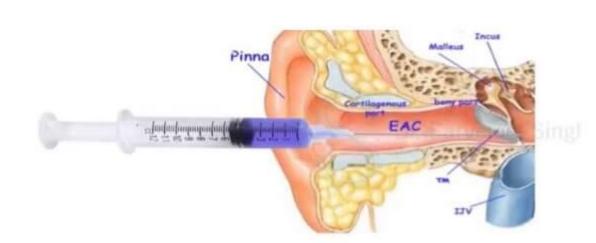
Surgical landmark's Donaldson's line

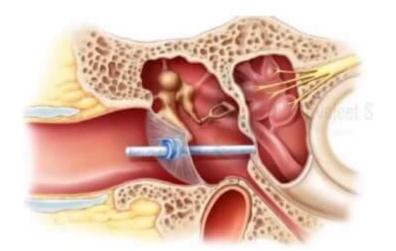
Surgical labyrinthectomy is done in a patient with chronic Menier's disease with sever SNHL because of which U/L ear is not serviceable for hearing and patient has vertigo

Q. Gold standard treatment for intractable vertigo in a patient of Menier's disease??

Ans: Surgical labyrinthectomy

- C) Intra Tympanic Gentamycin Therapy
 - → Chemical Labyrinthectomy
- D) Silverstein Microwick Microcatheter
 - → Drug delivery system to the inner ear

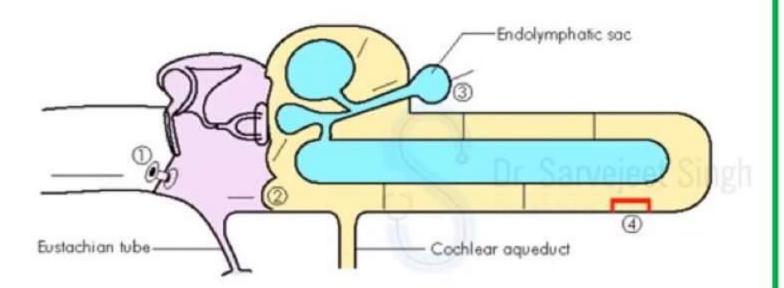






E) Meniett's Device

- → Intermittent low-pressure therapy device
- → US FDA approved



Note: 1. Ds of external ear + Ds of middle ear cause CHL

2. Ds of inner ear +8th nerve cause SNHL (Cochlear / Retro cochlear)

Superior Semi-Circular Canal Dehiscence /3rd Window Syndrome

- → Disease of inner ear leading to CHL
- → Superior SCC dehiscence creates a 3rd window
 - when oval window goes in some part of energy is lost via 3rd window
 - leads to CHL
- → C/F: Vertigo

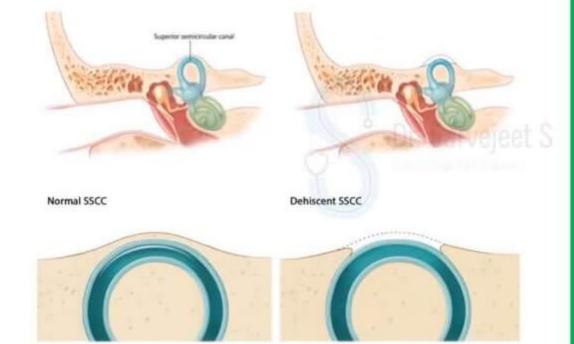
Oscillopsia

Autophony

Tullio's phenomenon

Fullness / pressure in the ears

- → Patients bone conduction is super normal
- → Diagnosis HRCT temporal bone
- → Rx Repair the SCC dehiscence through middle cranial fossa approach





Vestibular Schwannoma / Acoustic Neuroma

- → Earlier name Acoustic neuroma (Obsolete now)
- → MC Benign tumor of cerebellopontine angle. (CP angle)
- → Usually U/L But B/L in Neurofibromatosis II.
- → Site of origin
 - Inf vestibular Nerve in IAC (60-90%) \rightarrow M/C
 - · Sometimes sup vestibular N.
 - · Rarely from cochlear N.
- → Arises from Schwann cells in the myelin sheath, not from the nerve → SCHWANNOMA



- → SNHL
 - Slow & progressive
 - Most common presentation
- → Tinnitus
- → Vertigo/Dizziness (Not prominent)

→ Signs

- → Earliest: Loss of corneal reflex (Due to 5th nerve Involvement)
- → Hitzelberger sign: Loss of sensory supply by the facial Nerve in the postero-superior
 Wall of EAC

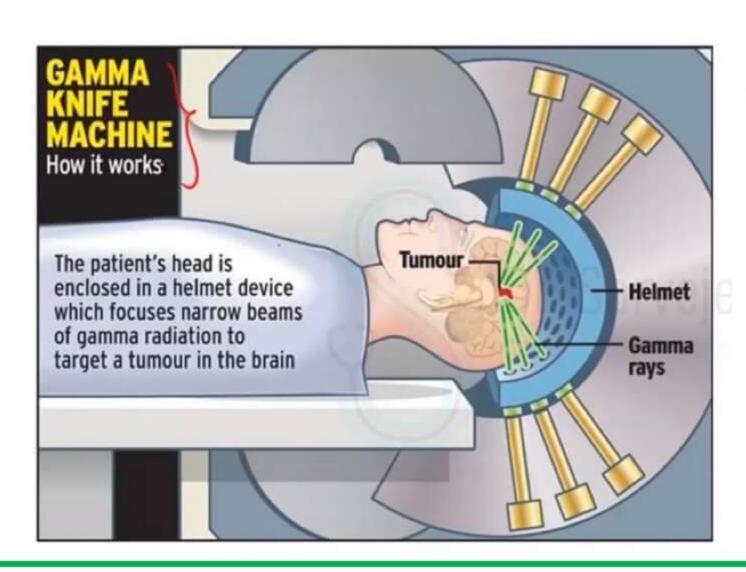
→ DX

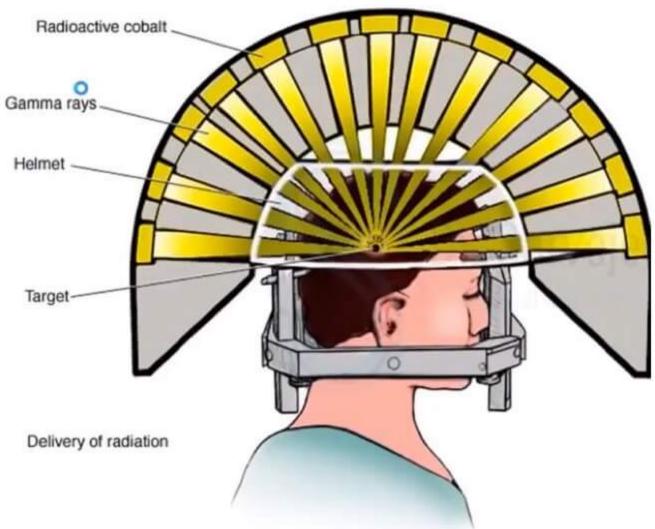
→ Gadolinium Enhanced MRI→ (10C)

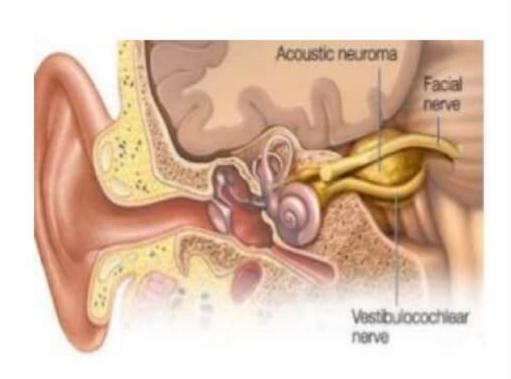
Treatment

- 1) Large Tumor Surgical excision
- 2) Small Tumor
 - → Old patient, slow growing tumor Serial MRI every 6M
 - → Young patient, fast growing tumor Gamma knife excision









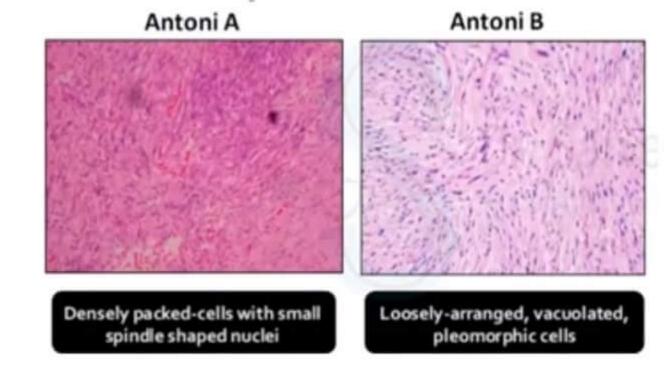
Approaches For Surgical Excision

- → When there is no hearing present
 - TRANSLABYRINTHINE approach
- → If hearing present
 - Middle cranial fossa approach (Limited access)
 - Retro sigmoid / sub occipital approach
- → After Sx excision, tumor is sent for histopathology
 - ANTONY A CELLS

More common

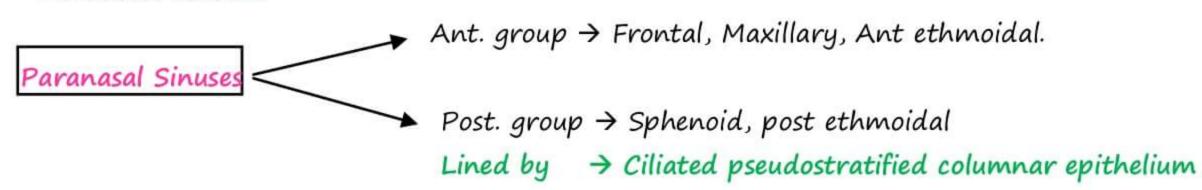
Densely packed cells with small spindle shaped nuclei

ANTONY B CELLS
 Loosely arranged, vacuolated pleomorphic cells



Nose & Paranasal Sinuses

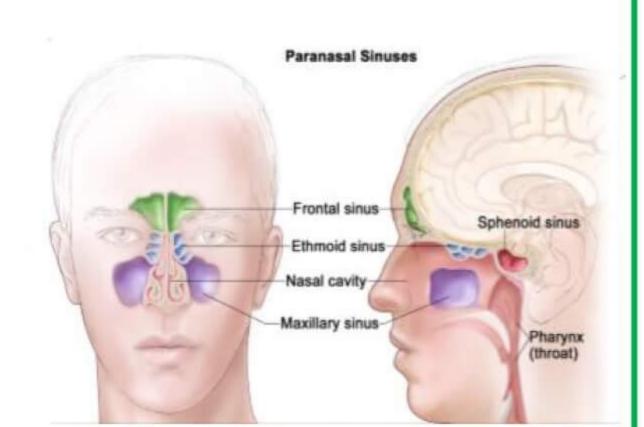
Paranasal Sinuses



1) Maxillary Sinus

- → 1st to develop at 12 wks (3M): present at birth.
- → Reaches adult size 15 18 years
- → Largest Paranasal sinus adult volume 15ml
- → AKA Antrum of Highmore / Maxillary Antrum
- → Visible on X Ray → 4 5 M after birth

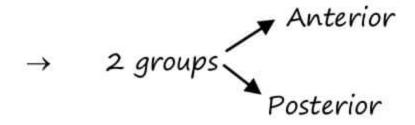
 CT Scan → at Birth



2) Ethmoid Sinus

- → Location Ethmoid bone (Unpaired)
 Cribriform plate Roof of nasal activity
 Thinnest bone in the body. Danger area of Nose
- → Contain multiple small air cells
- → Present at Birth, Adult size 12 years.
- \rightarrow Visible on X Ray \longrightarrow 1 years.

 CT Scan \longrightarrow at Birth



3) Sphenoid Sinus

Location: Sphenoid Bone - Unpaired

Body

Right sphenoid sinus

Inter sinus septum

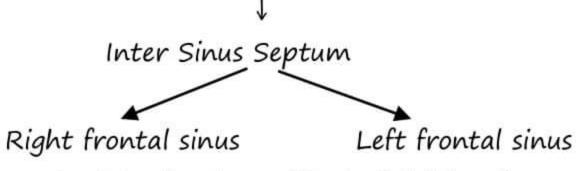
Left sphenoid sinus

- → Present at Birth as a small cavity.
- → Pneumatization 2 years
- → Adult size 15 years



4) Frontal Sinus

→ Location: Frontal bone - Unpaired



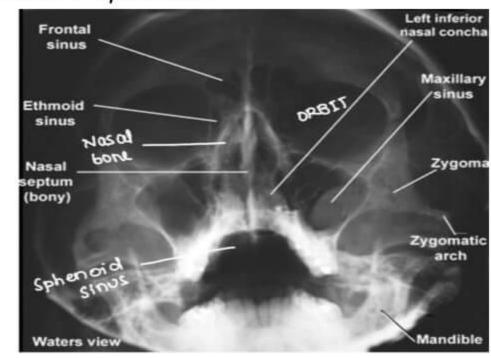
- → Last to develop (Early Adulthood)
- → Present as a small cavity at Birth, but indistinguishable from Anterior Ethmoid. (Not present at Birth)
- → Pneumatization After 2 years

Sinuses		Present (at Birth)	First X-Ray Appr.	Adult size
Maxillary	(M)	Yes	4 – 5 M after birth	15 – 18 years
Ethmoid	(E)	Yes	1 year	12 years
Sphenoid	(S)	Yes	6 -7 years	15 years
Frontal	(F)	Yes (NOT in CT Scan)	4 – 5 years	18 years

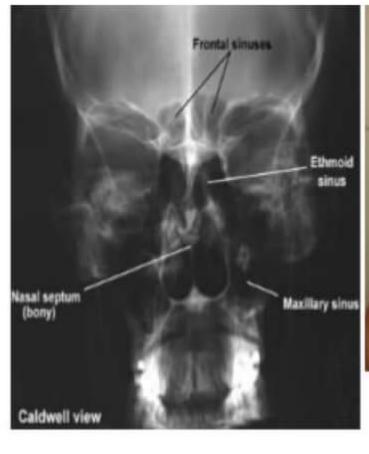
Plane X - Ray views for PNS

- → Mc done: Water's view / occipitomental view / Nose chin position
- 1) Sinuses best seen: Maxillary sinus, Anterior Ethmoidal
- 11) Sinuses not seen: Posterior Ethmoidal > Sphenoid





- > Sphenoid sinus is seen in water's view with open mouth: Pierre's view
- → Best view for Frontal sinus Caldwell's view / occipitofrontal view / nose forehead position





→ Lateral view: - All the paranasal sinuses are seen



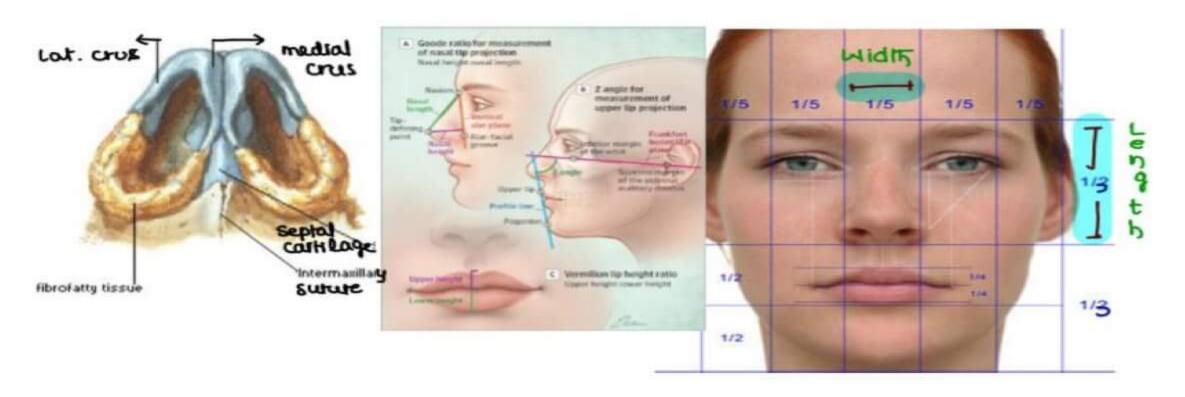
External Nose

Anatomy

Good Nose

→ Lateral profile: -

GOODE RATIO: _____ Ideal: 0.55 - 0.6



External Nose

- → Upper 1/3rd → Bony, formed by nasal bone supported by frontal process of maxilla
- → Lower 2/3rd Cartilaginous (3 paired & 1 unpaired)

- Paired

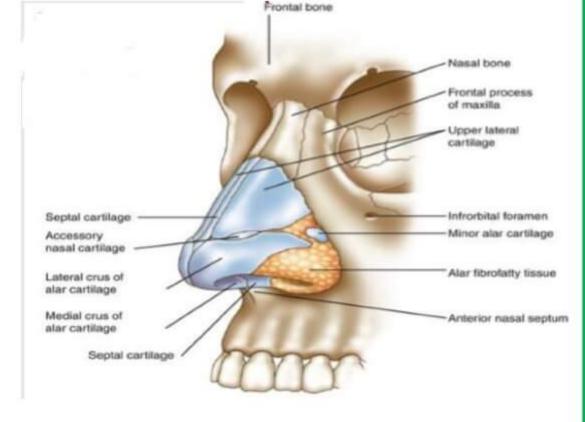
- 1. Upper Lateral
- 2. Lower Lateral [Alar] mainly
- 3. Lesser Lateral /Alar /Minor Alar
- Unpaired → Septal/Quadrilateral/ Quadrangular cartilage
- → Main Component of Alar Cartilage Fibrofatty Tissue Alar crus have 2 parts: Lateral crus (forms Ala of nose)
 Madial Cartilage – Fibrofatty Tissue

Medial Crus of Alar Cartilage forms Columellar Septum

Deformities & Diseases of External Nose

1) Deviated Nose

- → External deformity
- \rightarrow R_x Rhinoplasty
- 2) Crooked Nose / C shape deformity
- → R_x Rhinoplasty
- 3) Nasal Hump
- → Rx: Reduction Rhinoplasty
- 4) Saddle NOSE (Dorsum has collapsed inside)
- → R_x Augmentation Rhinoplasty

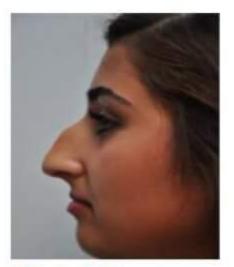








Crooked Nose



Nasal Hump



saddle Nose

5) Rhinophyma / potato tumors

- \rightarrow MC seen in males, 35 40 years.
- → A/W long standing Acne Rosacea (Pilosebaceous duct is blocked)
- → Benign hypertrophy of sebaceous glands.
- → Presence of lobulated mass
- → Not a tumor (No hyperplasia)
- $R_x \rightarrow Dermabrasion \bar{c} co_2 laser (Rx of choice)$



Rhinophyma

6) Rodent ulcer / basal cell carcinoma

- → MC malignancy of the skin
- → Sun exposed Areas Face, Dorsum of hands
- → Locally invasive malignancy
- → Distant metastasis are rare
- → More common in fair people

Rx: - Wide local excision









Nasal Cavity

Boundaries of Nasal Cavity

Roof - Cribriform plate of Ethmoid.

Floor - Hard Palate -> Anteriorly - Maxilla

Posteriorly - Palatine bone

Medial wall - Nasal Septum

Lateral wall - 1) Anterior openings - Anterior nares

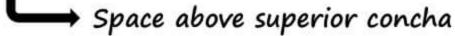
11) Posterior openings – Posterior choana

111) 3 projection / concha's / Turbinate

- Superior concha → Supreme

Turbinate

- Middle concha



- Inferior concha

IV) Space between turbinate's is meatus

- Superior meatus
- Middle meatus
- Inferior meatus (Largest)

SUPREME TURBLINATE [60multimus] Corporate Antenna Indian Soft palara Suprement Su

Bones of Nasal Cavity & Septum

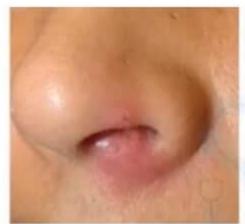
vomer

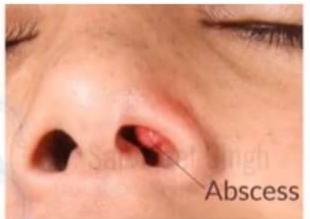
frontal

Lateral Wall Of The Nose

Nasal Vestibulitis

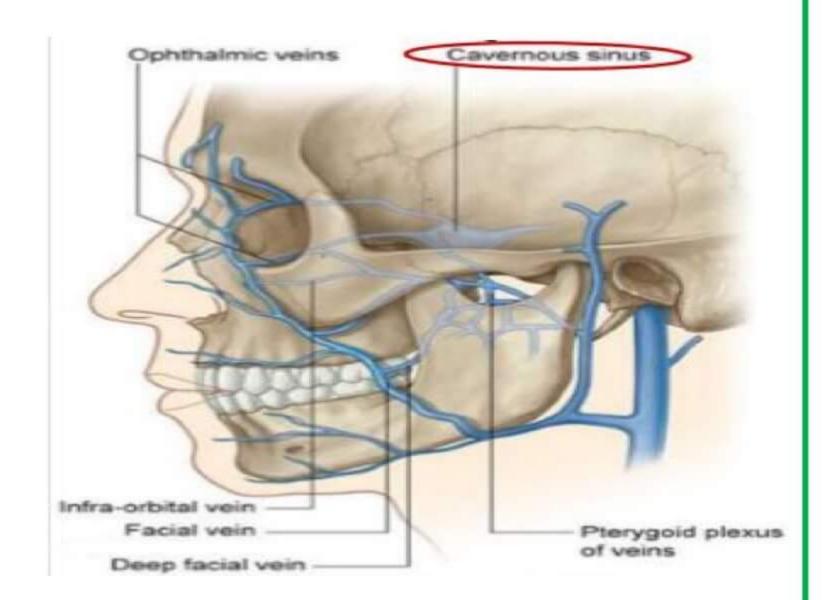
- → Any Furuncle in nasal vestibule
- → Presents with Pain & swelling (Red & Hard), Abscess
- → Infection by Staph aureus.
- → Rx: systemic Antibiotics & Analgesics.





Dangerous Area of Face

- → Area between upper lip & lower part of nose.
- → Veins drain into cavernous sinus through pterygoid plexus.
- → Infection of this area causes cavernous sinus thrombosis.





Nasolacrimal Duct (NLD)

- → Opens into inferior meatus through valve of Hasner
- → Direction
 - $N \rightarrow Inwards$
 - L → Laterally
 - D > Downwards
 - → After DCR (Dacryocystorhinostomy)
 [Done for chronic dacryocystitis]

NLD opens into middle meatus.

Osteo Meatal Complex (OMC)

- > Present in middle meatus
- → 3 sinuses open Frontal

 Anterior ethmoidal

 Maxillary sinus

Infection / Mass will block all 3 sinuses

Lateral Wall-Bony Anatomy

- 1) Total 8 Bones
- 2) (4) Frontal bone

 Ethmoid bone

 Sphenoid bone

 Maxilla
- 3) (4) Nasal bone Inferior Turbinate* Individual bone Palatine bone → L shaped Lacrimal bone (exclusive)

Medial Wall / Nasal Septum

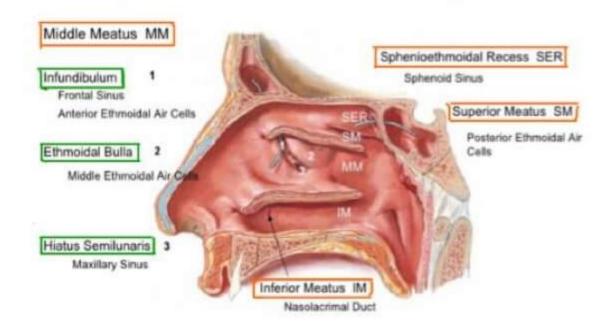
3 parts

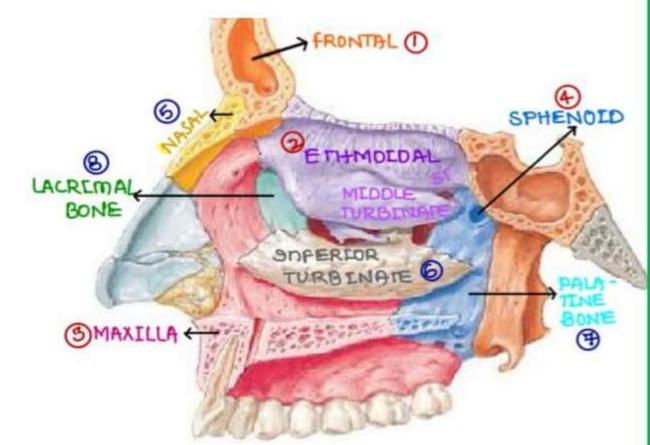
- 1) Columellar septum Medial crus of alar cartilage
- 2) Membranous septum Septal piercing
 - No bone & cartilage
- 3) Septum proper

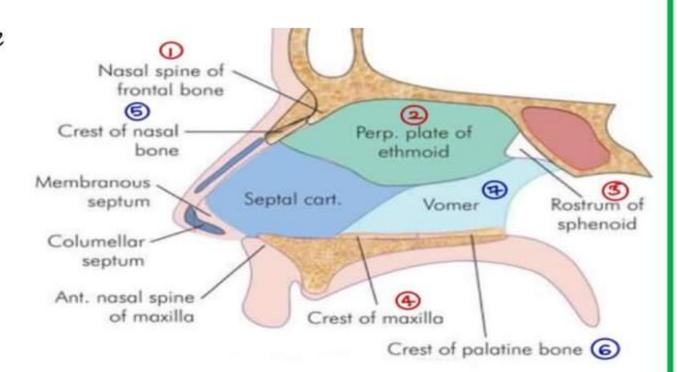


- 4 → Nasal spine of frontal bone Perpendicular plate of Ethmoid Rostrum of sphenoid Maxilla
- 3 → Nasal bone Crest of palatine bone Vomer (Doesn't contribute to lateral wall)

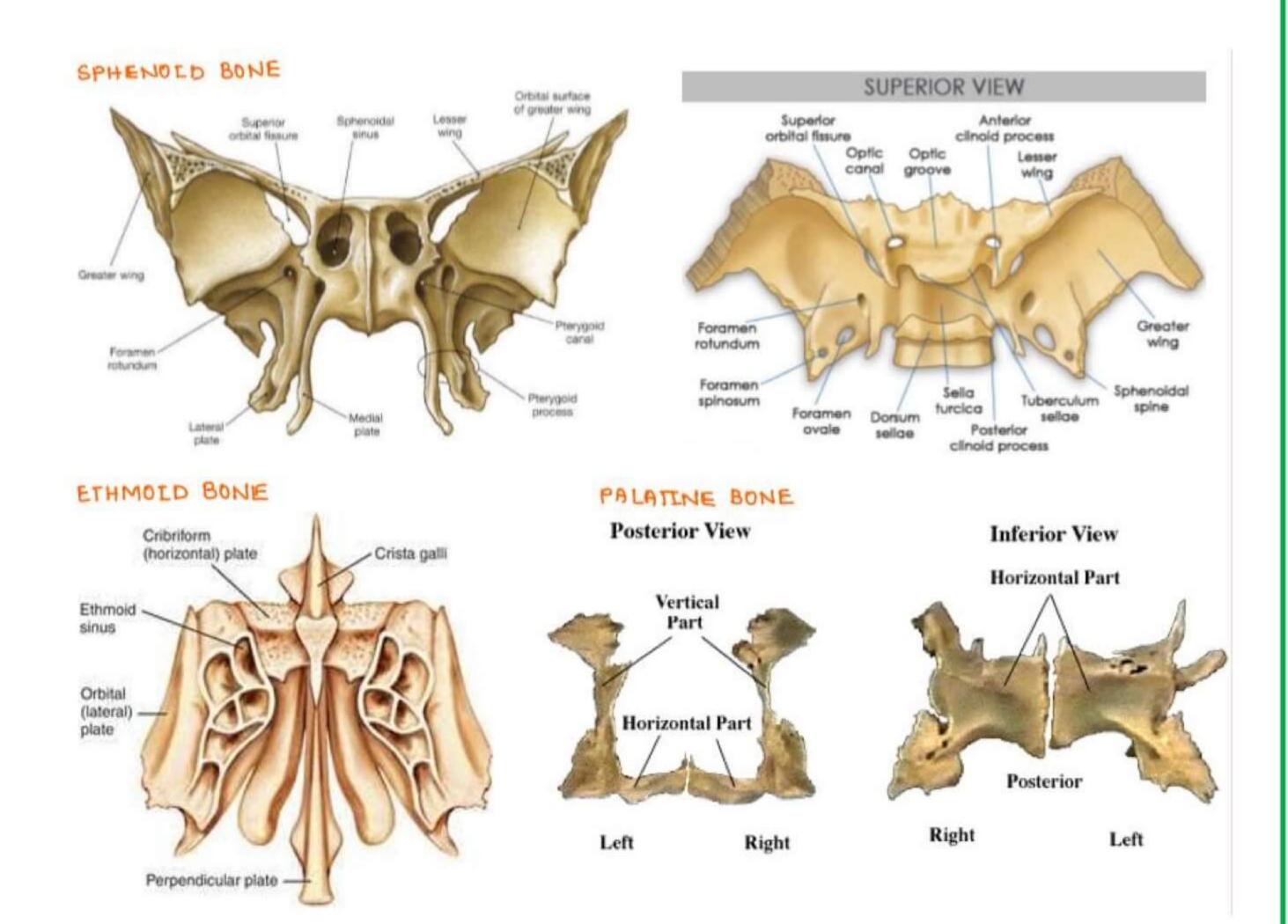
Openings in the Lateral Wall of Nasal Cavity



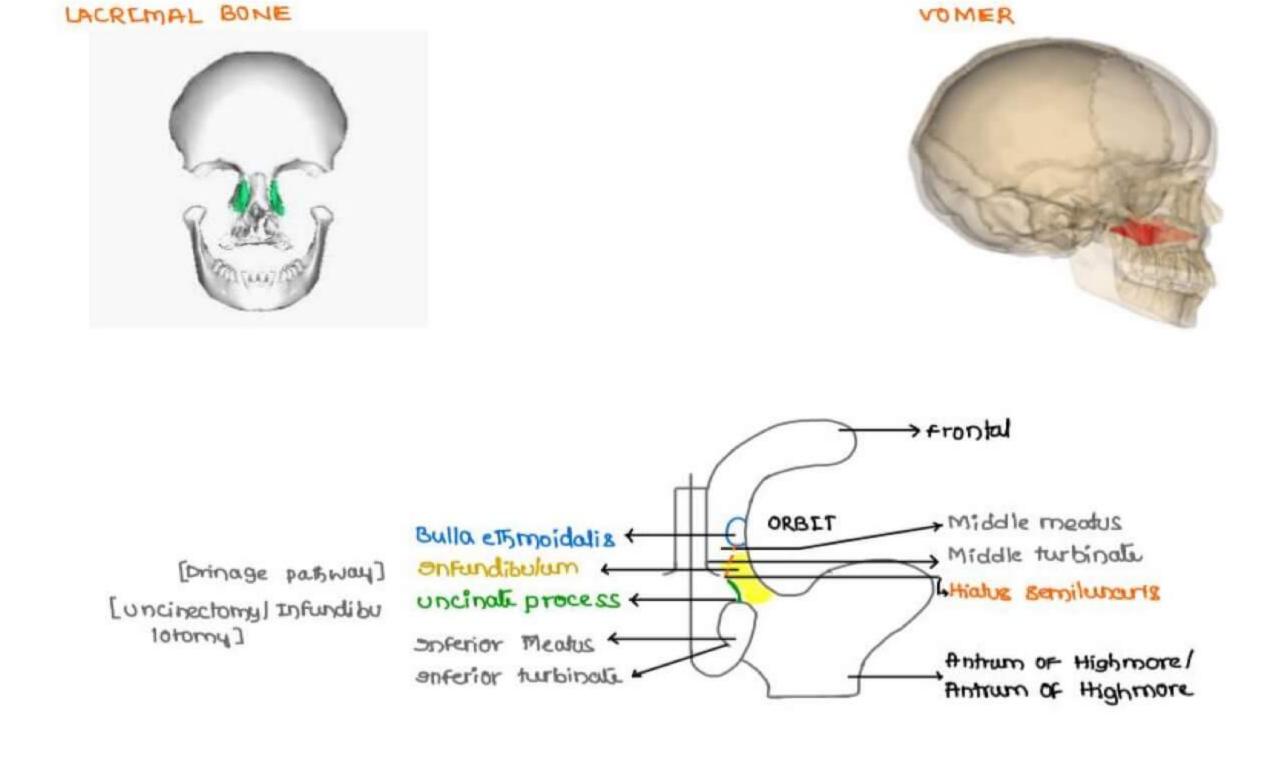






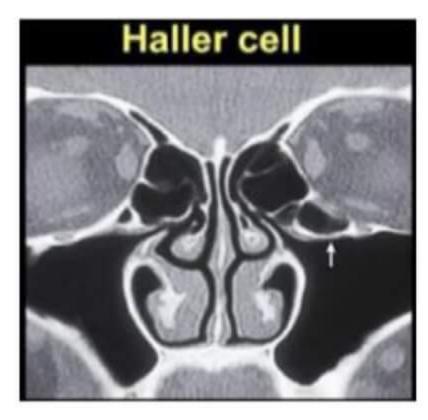


 \rightarrow Dangerous area of NOSE \rightarrow Cribriform plate of ethmoid [Olfactory nerve fibers]

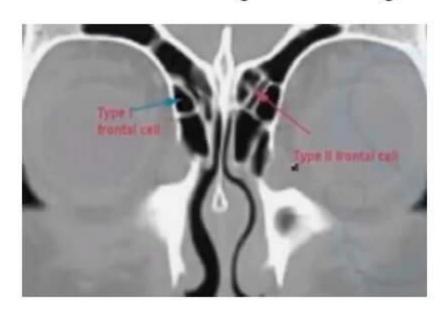


Anterior Ethmoidal Cells

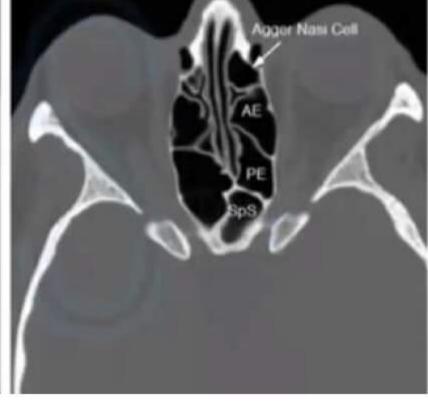
- 1) Bullae Ethmoidalis
 - > Most consistent cell of Ethmoid.
 - > Drains into middle meatus. (Anterior ethmoid)
- 2) Supra bullar cell
- 3) Infraorbital cell / Haller cell
 - → Infection can easily block drainage pathway.
- 4) Agar Nasi cells
 - → Anterior most cell of ethmoid.
 - → Close relation with lacrimal bone.



→ Left haller cell is larger than Right. Any inflammation can easily block drainage pathway.



Type 1 & 2 Frontal Cell



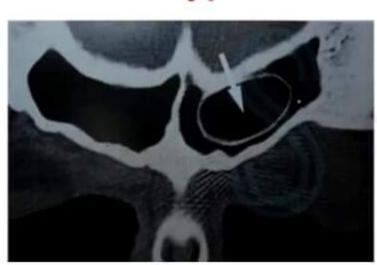
Type 3

Supraorbital cell



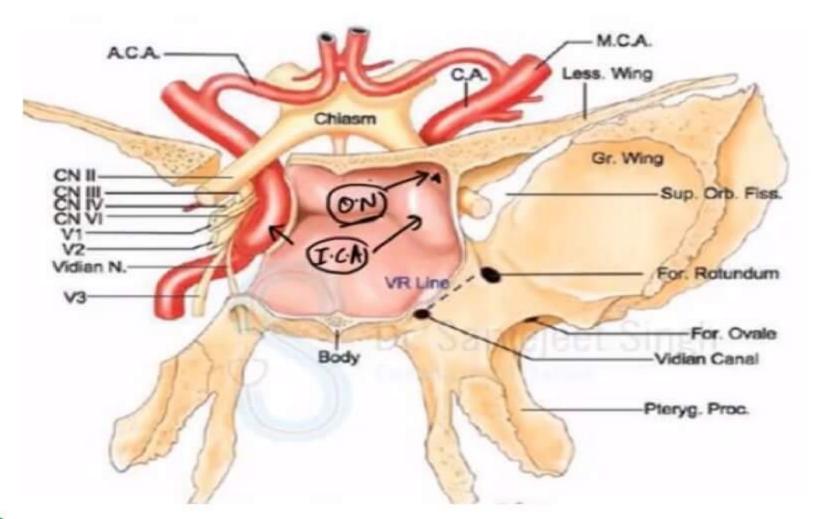


Type 4: Loner cell



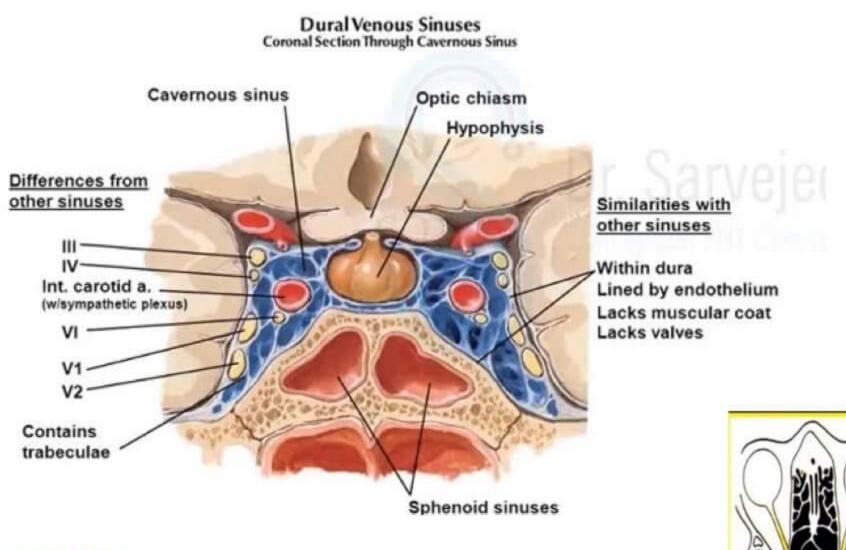
KUHN'S Classification Of Frontal Cells

- → 4 Types
- → Type 1 Single cell above agger nasi
 - Type 2 Multiple cells above agger nasi
 - Type 3 Inside the frontal sinus causes frontal headache (Office headache)
 - Type 4 aka loner /satellite cell
- → Type 1, 2 cells present outside the frontal sinus
- → Optic nerve is present in the lateral wall of sphenoid postero superior aspect.
- → ICA [Internal carotid artery] also present in Lateral wall of sphenoid sinus.



Cavernous Sinus

→ Only cavernous sinus has nerves and arteries compared to all other Dural venous sinuses



Posterior Ethmoid Cells

- 1) Onodi cell
 - → Posterior most cell of ethmoid
 - → Present lateral to sphenoid sinus
 - → Closely related with optic N. (Surgical significance)

ONODI CELL

Sphenoid Pneumatization

- 1. CONCHAL PNEUMATIZATION [LC, 1-4%]
- 2. PRE SELLAR PNEUMATIZATION
- 3. SELLAR PNEUMATIZATION [M/c, 54%]
- 4. MIXED PNEUMATIZATION [2nd M/c] [27%]
- → Has surgical importance during pituitary Sx, sophisticated instruments are required.
- Requires more time.
- More rate of complication in Conchal Pneumatization.
- Experience surgeon needed
- Care to be taken for not to damage optic nerve and ICA



Epistaxis

Ophthalmic A.

Interior maxillary A.

Blood Supply Of Nasal Septum

- → Supplied by 5 Arteries
 - 1) Anterior Ethmoidal A.

2) Posterior Ethmoidal A.

3) Sphenopalatine A.

4) Greater palatine A.

5) Superior labial A.

Facial A.

ECA

MCC of Epistaxis → Nose Picking

Little's Area / Kiesselbach's plexus

- → Formed by 4 branches anastomosis.
- → Posterior Ethmoidal Artery does not contribute
- → Mc site of Epistaxis (Nasal bleeding)
- → Ant. Epistaxis present from Little's area
- → Present On the medial wall

Posterior Epistaxis

- → MC Site woodruff's plexus
- →Woodruff's plexus
 - Venous plexus
 - Posterior to inferior turbinate
 - Supplied by Sphenopalatine A.
 Posterior Pharyngeal A
 - → MCC Idiopathic
 - → On lateral wall.
 - \rightarrow HTN is not a cause.
 - → Causes In elderly → winter / autumn season.
 - NSAIDs
 - Alcohol (last 24 hours)

Management of Epistaxis

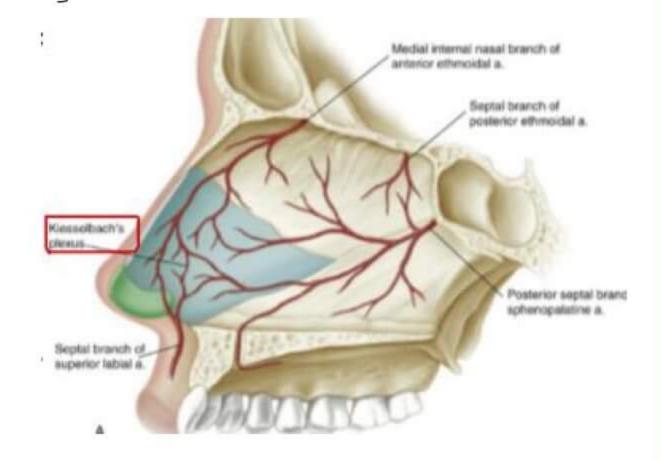
1) Anterior Epistaxis

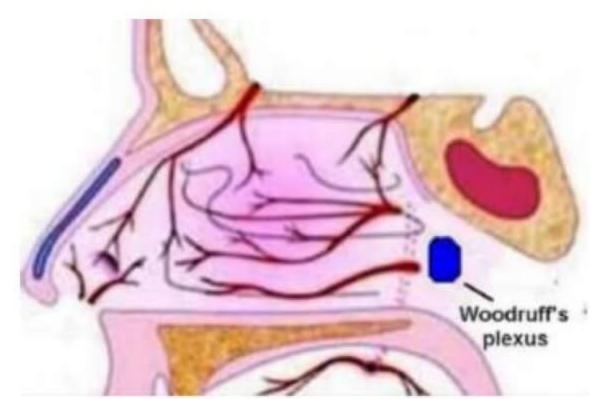
- → Anterior to piriform aperture.
 - $R_x \rightarrow Nose pinching for 3 5 minutes$

Trotter's method

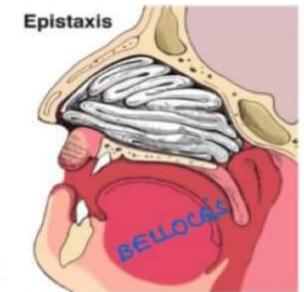
- \rightarrow Child \bar{c} recurrent epistaxis
 - Chemical cautery [AgNO3, TCA]
 - Electrocautery

Under local anesthesia









Anterior Nonal packing

2) Posterior Epistaxis

→ Bleeding point not visible.

TOC → Endoscopic electrocautery/ligation (80%) RxOC

→ Anterior Nasal packing in case of profuse bleeding Not responding.

B/L Nasal Packing

Posterior Nasal packing \bar{c} foley's catheter under local anesthesia. Not responding

Combined classical anterior + posterior nasal packing

- → Epistaxis catheters / Balloon catheters
- -> Artery of Epistaxis Sphenopalatine Artery

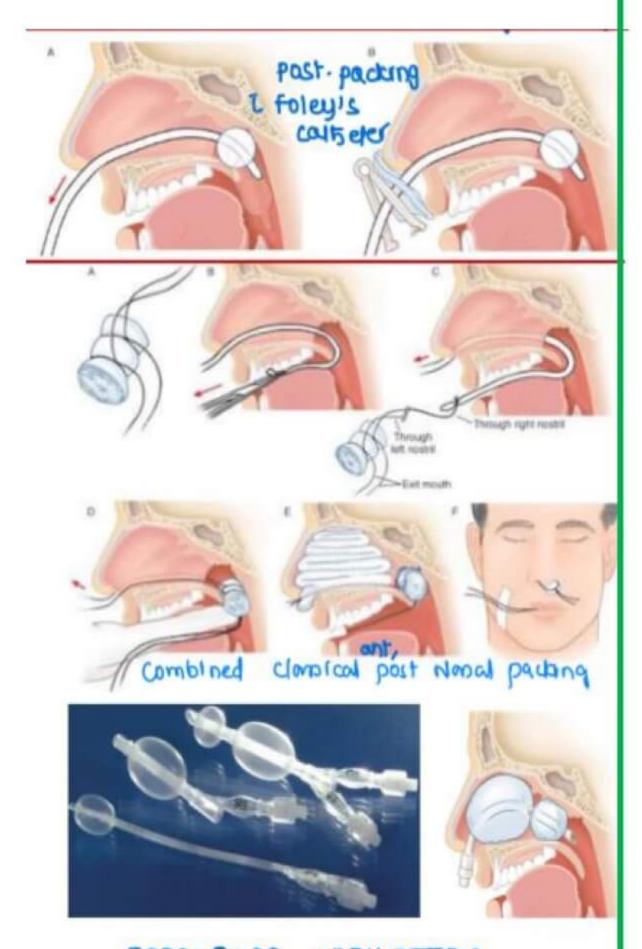
Endoscopic sphenopalatine A. ligation. (ESPAL)

Done in recurrent epistaxis

Not responding

Int. Maxillary ligation / ECA ligation.

→ ICA is never ligated.

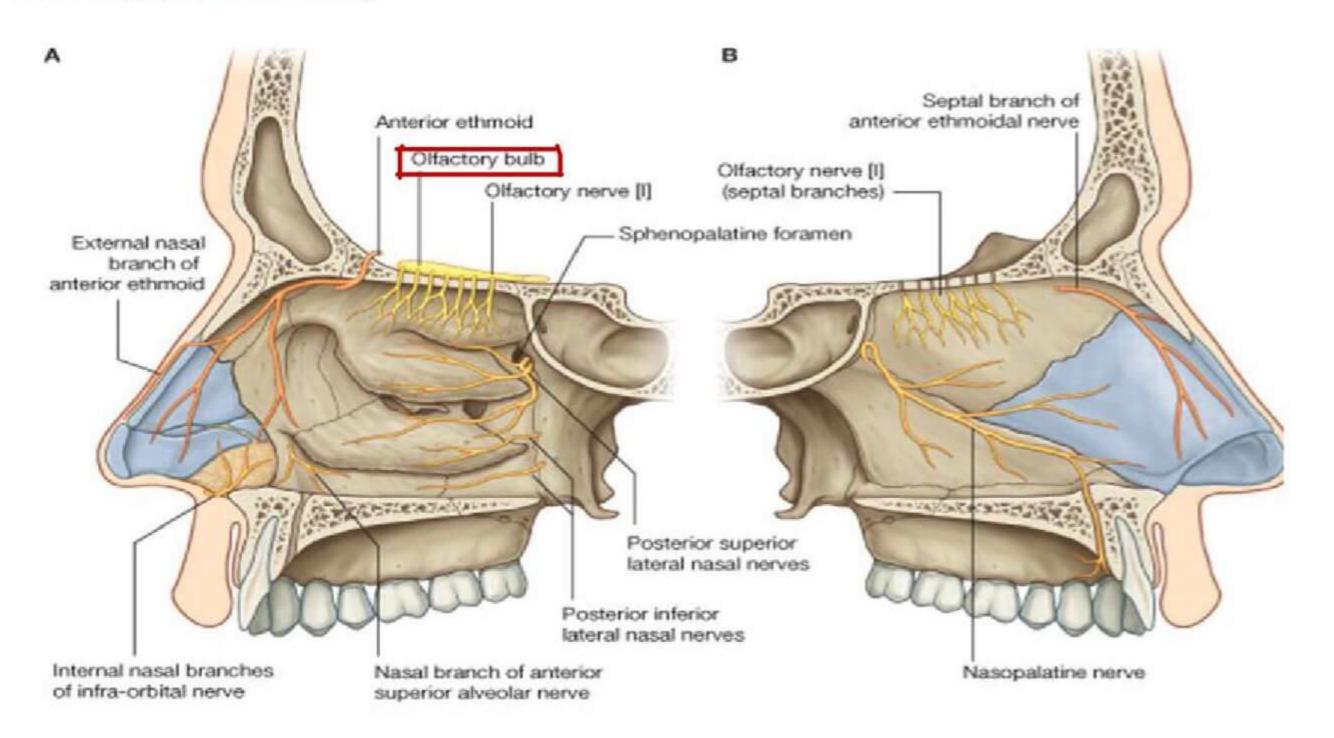


EPISTAXIS CATHETERS



Physiology of Nose

Nerve Supply of Nasal Cavity



→ Inspiration

During normal tidal inspiration, the maximum air passes through middle meatus in parabolic curve.

→ Expiration

During expiration, the paranasal sinuses are aerated & air comes out during inspiration

- BERNOULLI'S PHENOMENON: During inspiration it creates -ve pressure.
- PNS role: Air conditioning during inspiration and helps preserving heat & moisture during expiration.
- → Olfaction Olfactory bulb is present in anterior cranial fossa above cribriform plate. Olfactory nerve fibers are present in upper $1/3^{rd}$ of nasal cavity.

YA WALAA

CHEVALLET #

Diseases of Nasal Septum

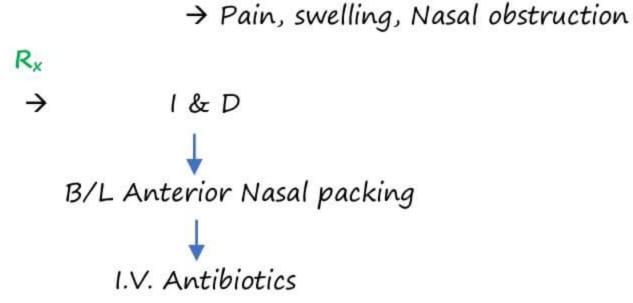
- 1) JARJAWAY # > # Line parallel to cartilage vomer junction.
- 2) CHEVALLET # → # Line starts from anterior most point to maxillary crest (Nasal spine of maxilla) to the nasal spine of frontal bone
 - > Punch / impact from below



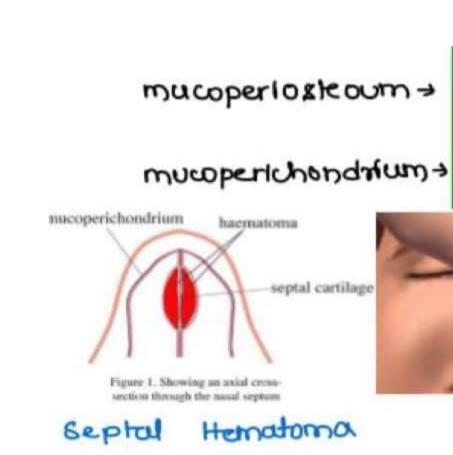
- → B/L Nasal obstruction
- → Collection of blood between septal cartilage & Mucoperichondrium

Rx

- → 1 & D → Prophylactic Antibiotics
- → B/L Anterior Nasal packing to stop the bleeding.
- 4) Septal Abscess → Collection of pus between cartilage & its mucoperichondrium



→ M.C. complication: - Septal perforation



5) Septal Perforation

- → Hole in the septum
- → Causes
- 1) Trauma MCC
 - Digital
 - S_x
- 11) Cocaine Abuse
- III) Granulomatous:
 - TB
 - Leprosy
 - Lupus vulgaris → Apple jelly nodules in nose
- → Bony: Syphilis Vulgaris
- → Bony & Cartilage: Wegner's Granulomatosis



- IV) Septal Piercing
- → Septal Surgeries







Septal Perforati



SMR (Sub Mucus Resection)		Septoplasty	
 Rise the mucoperichondrium & Mucoperiosteal flaps on both sides & remove bone & cartilage leaving 'L'	-	Rise both flaps on one side & remove only deviated septum More conservative S _x	
Shaped cartilage.		Low chances of perforation	

COTTLE'S Line

- Ant to line septoplasty
- Post to line SMR

COTTLE'S Test

- Test of nasal valve / Limen nasi/ Limen vestibuli

Indications of Septal Sx

- 1. DNS causing nasal obstruction
- 2. DNS causing recurrent epistaxis DNS itself is not an indication

- 3. DNS causing chronic infection
- 4. As a part of other S_x
- 5. To give access to other S_x

Diseases of Nose & Paranasal Sinuses

Inflammations

Rhinitis	Sinusitis	
→ Nose is lined by Pseudo stratified ciliated	→ PNS lined by pseudo stratified ciliated	
columnar epithelium [Respiratory epithelium]	columnar epithelium	
columnar epithelium [Respiratory epithelium]	columnar epithelium	

> Common term - Rhinosinusitis

1) Acute Rhinosinusitis / Common Cold

MC causative agent - Rhino virus

- Mucoid discharge

Rx - Symptomatic

2) Acute Bacterial Rhinosinusitis

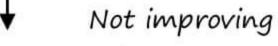
→ Mcc: Strep. pneumonia
Moraxella catarrhal
H. Influenza

- → Yellowish / mucopurulent discharge
- → Mc sinus involved Maxillary sinus Rx: - Antibiotics

3) Chronic Rhinosinusitis

- → Infection >12 wks (3m)
- → 2w ->3m: subacute
- → Mc causative: Staph Aureus
- → Chronic infection + chronic hypertrophy & obstruction
- → Management

 $TOC \rightarrow 1$) Culture directed antibiotics + Nasal decongestant.



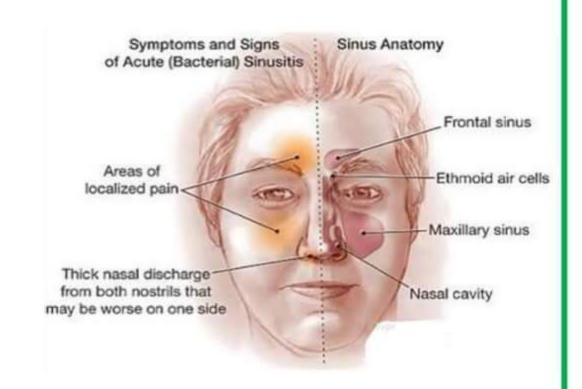
Antral puncture & lavage / Antral wash (Obsolete)

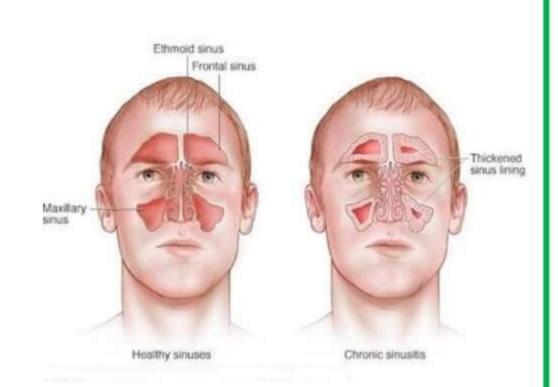


FESS (Functional endoscopic sinus sx)

Frontal sinusitis: Office headache / frontal headache

Sphenoid sinusitis: Occipital headache



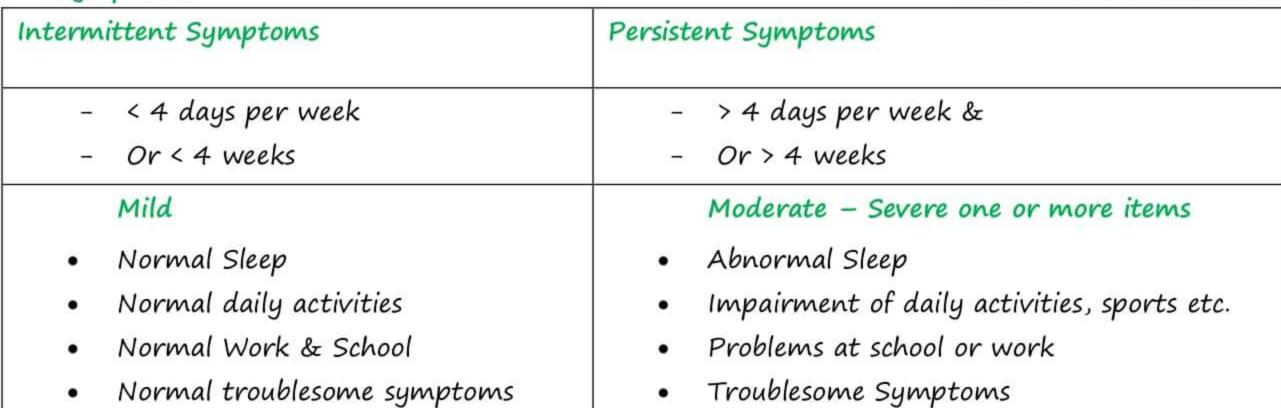




4) Allergic Rhinosinusitis

- → Mc allergen House dust mite / carpet dust
- → Mc in urban areas & developed countries
- → Mc in snow covered areas

Symptoms



On Examination

- → Lethargic
- → Allergic / Atopic facies
- → Allergic shiners / Denni Morgan lines
- → Allergic salute / Nasal crease

On Nasal Examination

- → Mucoid secretion in nasal cavity
- → Mulberry appearance of Nasal mucosa
- → Nasal mucosa is hypertrophied

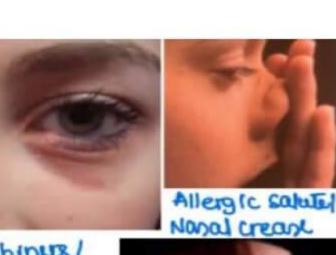


Allergic Atopic tacles



Allergic Shinus/ DENNI MORGAN LINES





→ Confirmatory Test

- → Skin prick test 10C
 - 1 is histamine used (As positive control)
 - 2 is normal saline (Negative control)

→ Nasal allergen challenge test/provocation test (Gold std.)

- Not commonly done

Rx

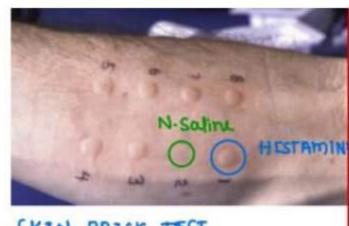
- → Steroids (Intra nasal spray) DOC
- → Non respondent to pharmacotherapy → Immunotherapy

5) Vasomotor Rhinosinusitis

- → d/t ↑ Parasympathetic discharge
- → MC in emotional females
- → Non allergic non infective perennial Rhinosinusitis (NANIPER)

Rx → Anticholinergic spray (Ipratropium Bromide)

→ Vidian Neurectomy - Gold std.



SKIN PRICK TEST



6) Rhinitis medicamentosa

→ Excess usage of Nasal decongestant drops

Xylo / oxymetazolines

REBOUND PHENOMENON

Rx

-Stop decongestants

DOC - Intra nasal corticosteroid spray (Topical)

Atrophic Diseases

1) Atrophic rhinosinusitis (Ozaena)

- → Causative organism: Klebsiella ozaena / Perez Bacillus
- → Young females, low socioeconomic status.
 - Iron deficiency.
 - Multivitamin def/vit D deficiency.
- → Pseudostratified ciliated columnar epithelium is replaced by stratified squamous Epithelium.
- → M.C. complaint → Nasal obstruction (Crust formed)
 - > Foul smell
 - → Anosmia (Merciful Anosmia)
- → 0/E
 - Large roomy nasal cavity \overline{c} foul smelling crusts.

Rx

- 1. Alkaline nasal douching (Nacl + NaHCO3 + Na biborate)
- 2. 25% glucose (Nourishment) in glycerin (Hygroscopic)
- 3. Iron & vitamin D supplements
- 4. Antibiotics
- 5. Estrogen spray
- 6. Kimecetine nasal soln / Anti Ozaena solution
 - a. Chloromycetin / Chloramphenicol
 - b. Vitamin D
 - c. Estradiol
- 7. Sx → Young's operation → Alternative closure of each nasal cavity for 6 months

Modified Young's Operation (1 - 3 mm)

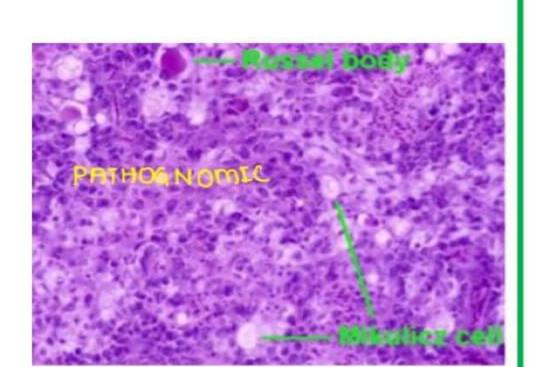
2) Rhinoscleroma / Respiratory Scleroma

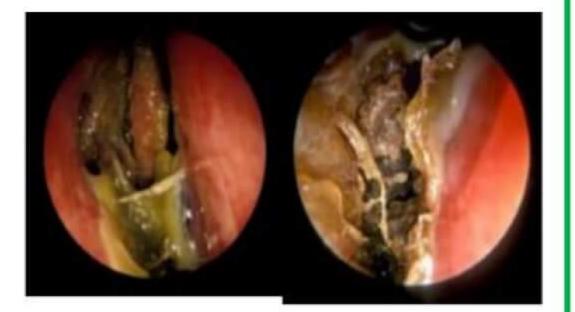
→ Caused by klebsiella Rhinoscelromatis / Frisch Bacillus

Stages

- 1) Stage of Atrophy
- II) Stage of granuloma formation









→ Miculicz Cells & Russell Bodies Are Seen

- III) Stage of sclerosis / fibrosis/cicatrization
 - AKA woody nose / Hebra nose / Tapir nose



- → Seen in stage 3
- → Woody induration

(Seen in stage 2)

Rx

- DOC → Rifampicin
- Rx OC → Laser excision + Base electrocautery



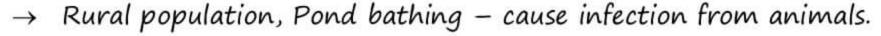
- → Rhinoscleroma → Disease of Respiratory, Epithelium
 - New Name → Respiratory scleroma

3) Rhinosporidiosis / Strawberry Granuloma

→ Caused by Rhinosporidium seeberi



- 1) Aquatic protozoa
- 11) Affects only mucosal surfaces
- → Seen in southern Eastern coast of India, Sri-lanka, Bangladesh.



- → Strawberry granuloma
- → Nasal obstruction present
- → Confirmatory tests: Bx + HPE

Multiple thick-walled sporangia

 R_{x} DOC \rightarrow Dapsone

Amphotericin - B

 $TOC \rightarrow Laser excision \bar{c}$ Base electrocautry



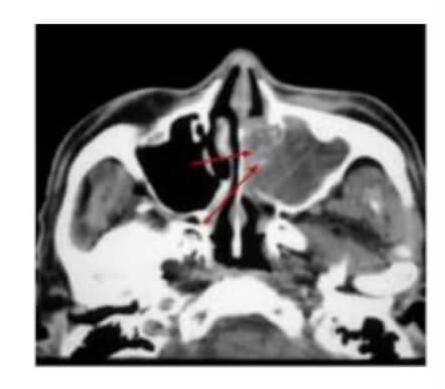


Fungal Rhino Sinusitis

1) Fungal Ball / Mycetoma / Aspergilloma

- → Mc : Aspergillus fumigatus
- → Immuno competent patients; No mucosal sensitivity
- → No invasion by fungus, No reaction from nasal mucosa

 R_x : - Evacuation \bar{c} FESS No role of Antifungal



2) Allergic Fungal Rhinosinusitis (AFRS)

→ Mc causative organism - Bipolaris

Others - Curvilaria

- A. fumigatus
- → Criteria Bent & Kuhn Diagnostic criteria

Bent and Kuhn Diagnostic criteria

Major	Minor
Type I hypersensitivity	Asthma
Nasal polyposis	Unilateral disease
Characteristic CT findings	Bone erosion
Eosinophilic mucin without invasion	Fungal cultures
Positive fungal stain	Charcot-Leyden crystals
	Serum eosinophilia

- \rightarrow CT findings
- U/L generally, but can be B/L
- Bone erosion w/o invasion
- Hyper densities [due to ca2+ deposition in dense fungal hyphae.]
- → Type 1 Hyper Sensitivity Allergic mucin release
- → Patient is immunocompetent
- → Multiple nasal polyps, No invasion
- \rightarrow +ve fungal stain
- → Characteristic of CT scan Double dense appearance

Rx

- FESS & removal of fungus
- Steroids oral → short course & prolonged nasal steroids
- Immunotherapy





3) Rhinocerebral Mucormycosis

- → Mucor / Rhizopus (Saprophytic fungus)
- → Acute invasion fulminant fungal disease
- → Immunocompromised patients (Diabetic patients with uncontrolled sugar)
- → Necrotizing vasculitis due to invasion of arteries.
 Ophthalmic involvement → Sudden blindness
 ICA involvement → Stroke
- → DOC/ TOC → Amphotericin B Surgical debridement Diabetes control





SHVASIVE FUNGAL RHINOSINUSTITS

Nasal Polyps

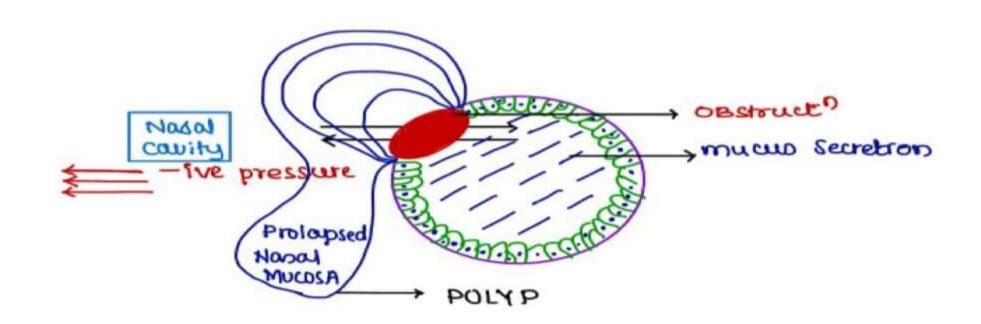
Antrochoanal Polyp [AC Polyp]

- → Starts from maxillary antrum
- → Single, large, U/L
- → Grows posteriorly towards choana
- \rightarrow Children \bar{c} an infection
- → C/F: Both U/L & B/L obstruction

Ethmoidal Polyp

- → Starts from ethmoidal air cells
- → Small, multiple, B/L
- → Comes out anteriorly
- \rightarrow Adults \bar{c} allergy
- → B/L Nasal obstruction

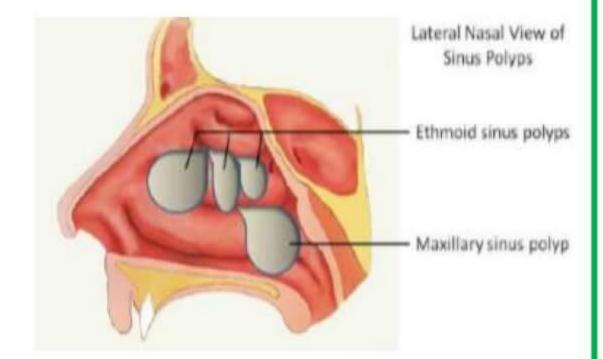
Pathophysiology of a Polyp



- → Prolapsed sinus mucosa due to -ve pressure in Nasal cavity.
- → No Nerve Supply, No Blood supply
 - No bleeding or pain on touch
 - Pale & Glistening polyps

IOC → NCCT of Nose & PNS

Rx → FESS (TOC)





Tumors of Nose & PNS

Benign Tumors

1) Osteoma

- M/C benign tumor of PNS
- M/C location Frontal sinus > Ethmoid sinus

→ Presentation:

- Sinus Blockage c obstructive symptoms
- Mass pressing on the orbit
- Rx -> Surgical Excision by endoscopic approach

2) Inverted Papilloma / Ringertz Tumor

- → M/C benign tumor of Nasal cavity
- → M:F 3.5:1

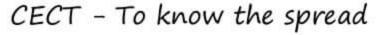
50-60 years

→ Arises from Schneiderian Membrane

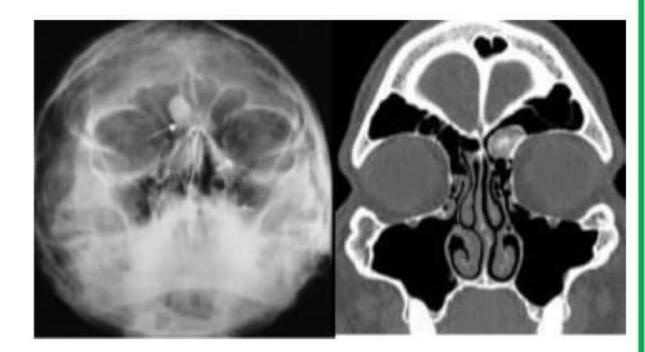
(Transitional cell epithelium)

- → Presentation
 - Polypoidal mass arising from lateral wall of nasal cavity
 - Nasal obstruction
 - Bleeding
- → Investigations

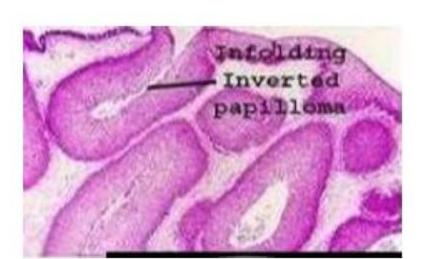
 $IOC \rightarrow B_x \rightarrow HPE$: Shows inverted Infolding papilloma (Confirmatory).

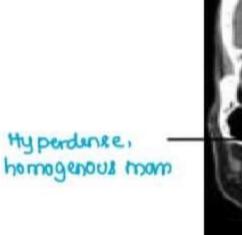


- Hyperdense homogenous mass
- $\rightarrow R_x$
 - Endoscopic Excision (TOC)
- → Chances of Recurrence & conversion into sq. cell carcinoma.







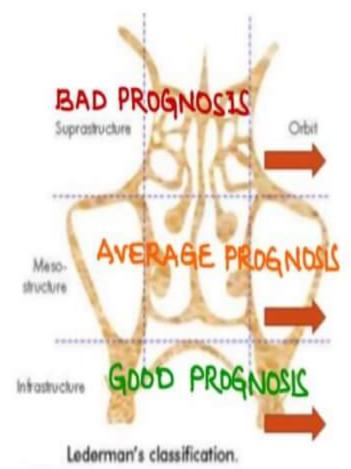




Malignant Tumors

1) Squamous Cell Carcinoma (80%)

- → M/C PNS: maxillary sinus
- → M/C malignancy of PNS & Nasal cavity
- → Occupational malignancy
 - Furniture industry Adeno CA
 - Nickel Industry Sq. CA
- → M.C. in males
- → sq. cell CA of the septum: Nose Picker's tumor
- → MC lymph Nodes involved: Submandibular



Ethmoid, sphenoid, frontal sinuses & olfactory area of nose.

Maxillary & respiratory part of nose.

Alveolar process



Classification

- 1) Ohngren's line: Hypothetical line from medial canthus to angle of mandible
 - Above the line / Suprastructure: poor prognosis
 - Below the line / Infrastructure: Good Prognosis
- 2) Lederman's classification
 - Uses two lines of sebileau.

TNM Staging

T

- $T_1 \rightarrow Involvement of sinus mucosa$
- $T_2 \rightarrow Bone$ (Except the superior wall of maxillary sinus)
- $T_3 \rightarrow$ superior wall or Ethmoid air cells or subcutaneous tissue.
- $T_4 \rightarrow Orbital content + skin + eyeball$

Investigation → Biopsy (Confirmatory)

 $T_x \rightarrow S_x + Radiotherapy \pm Chemo$

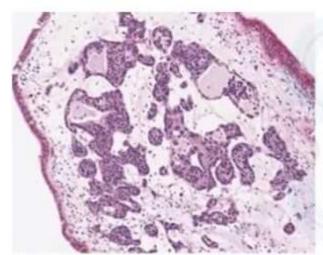


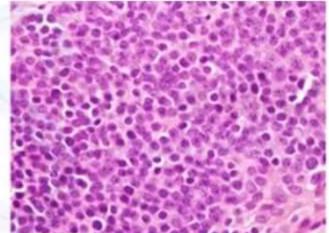
Esthesio-Neuroblastoma/Olfactory Neuroblastoma

- → Rare
- → Aries from olfactory neuroepithelium
- → M/C site Roof of nasal cavity
- > Histology shows neurofibrillary stroma and neurosecretory granule
- → Rx Endoscopic excision

f/b

Radiotherapy

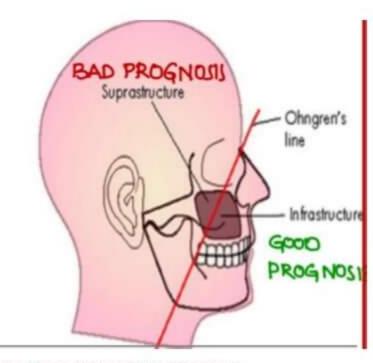




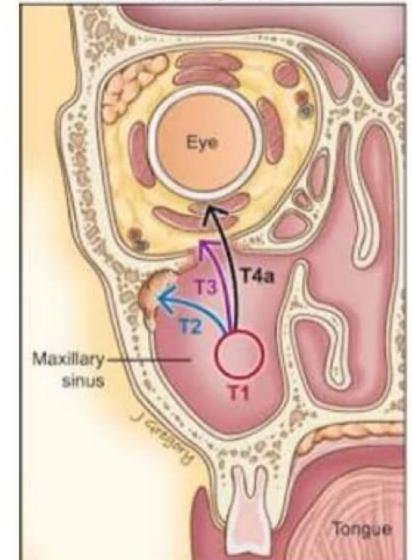
neurofibrillary stroma and neurosecretory granules

Midline Lethal Granuloma/ Stewart's Granuloma

- → T cell /NK cell lymphoma
- → Rapidly spreading, destructive lymphoma
- → Rx chemotherapy



Tumors of the Maxillary Sinus







Miscellaneous Topics

1) Nasal Myiasis / Maggots

Causative - Chrysomia fly larvae

Tx - 10 % chloroform → (To anesthetize maggots)

▼ Removal

2) Foreign Body in NOSE

- → MC seen in children
- → U/L mucopurulent discharge or blood-stained discharge in child
- \rightarrow Investigation
 - Endoscopy (For confirmation)
 - X-Ray
- → Rhinolith formation due to calcification [stone in nose]
- \rightarrow R_x: Removal \bar{c} a probe / eustachian tube catheter
- → Posterior fb → Endoscopic removal under general anesthesia

3) Facial Trauma

- → MC bone to undergo # Nasal Bone
- → Open book deformity

Due to the fracture

- → Closed book deformity
- → C/F
 - → Cosmetic deformity
 - → Nasal obstruction

Rx

- > Closed reduction with external fixation
- Time $\rightarrow \bar{c}$ in 24 hours (Before onset of edema)
 - → After 4 -5 days (After reduction)
 - → Not done between 2 weeks 3 weeks (Callus is formed)
 - → Complete rhinoplasty after 3M (Complete bone is formed)

4) Zygomatic Bone # / Tripod

- → 2nd mc facial bone #
- → Now known as Quadripod #
- → Malar prominence is lost
- → Step deformity on palpation
- $\rightarrow R_x \rightarrow ORIF$









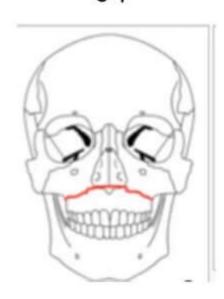


5) Blowout # / Orbit

- → Weakest wall of orbit: Floor
- → Thinnest: Medial wall / Lamina papyracea
- → Orbit hangs in the maxillary antrum
 - Tear drop sign
- → Diplopia due to Inferior Rectus intrapment
- \rightarrow R_x: Mesh application & reduction

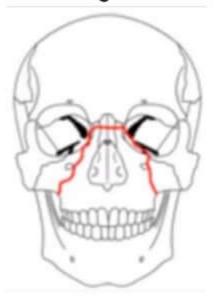
5) Maxillary # / Le fort

- → Le fort 1 # / Transverse #
 - Line parallel to hard palate
 - Floating palate

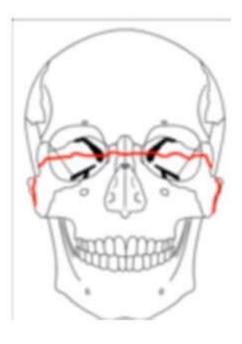


→ Le fort 2 # / Pyramidal #

- Floating maxilla



→ Le fort 3 # / cranio facial dysfunction



- Most dangerous, Morbidity

6) CSF Rhinorrhea

- → MCC: Head Trauma
- → Clear, watery discharge





- ↑ On coughing, sneezing, straining, bending forward (Tea pot sign)
- Reservoir sign
- → MC site :- # Cribriform plate of Ethmoid
- → Sniff test: Cannot sniff back
- → Handkerchief test: Mucoid discharge will make it stiff.
- → Glucose test: 2/3rd of blood glucose levels present in CSF (40 60 mg/dl)
- → In case of blood in CSF
 - 1) Halo sign / Target sign / double ring sign
 - β₂ Transferrin test (Gold std)
 Only found in CSF
- → IOC :- HRCT →

→ Gold standard investigation in CSF rhinorrhea??

- a. β2 transferrin levels (Not done because diagnose already done)
- b. HRCT of nose and PNS (To find site of leak) 1st inv./10C
- c. MRI T2 weighted images (Can't see both # the CSF at once
- d. CT cisternography (Can see both #and the CSF)

Management

- 1) Traumatic
- Q. 38 yrs old pt presented after RTA few hrs back with CSF Rhinorrhea. Next line of $M_{\rm x}$:-
- a. Conservative Mx [No active Surgical mid → heal on itself
- b. B/L Ant. nasal packing to stop CSF leak → Nasal packing C/I
- c. Immediate endoscopic repair of Skull base
- d. Open neurosurgical Repair of skull base

→ Conservative Management

→ Prophylactic Antibiotics (Wait for 7 days)

Acetazolamide

- → Stool softners & bed rest
- → B/L anterior Nasal packing C/I (Causes meningitis)
- → No relief: Lumbar drain for 2 wks
- → No relief :- Endoscopic repair

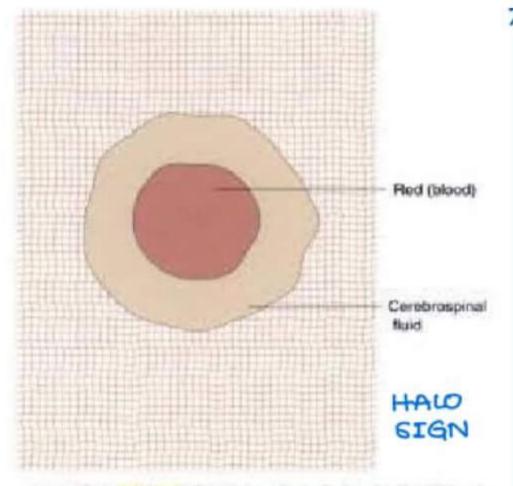
2) Spontaneous CSF Rhinorrhea

→ No injury

Causes → Brain tumor - Do MRI for R/O cause of ICT

- → Repair (Endoscopic)
 - 3) Paradoxical CSF Rhinorrhea
- → # Temporal bone (Petrous part), can cause CSF otorrhea





Halo sign. Clear drainage that separates from bloody drainage suggests the presence of cerebrospinal fluid.

Pharynx

Extends from skull base to C6 Vertebrae divided into 3 parts

- 1) Nasopharynx
- → From skull base to hard plate
- 2) Oropharynx
- → From hard palate to hyoid bone
- 3) Hypo / Laryngopharynx → From hyoid bone to esophagus

MUSCULAR LAYER

- → All supplied by superior laryngeal nerve of vagus except cricopharyngeus (By recurrent laryngeal nerve of vagus)
- → Sinus of Morgagni
 - Structures passing are
 - 1. Eustachian tube
 - 2. Levator veli palatini
 - 3. Ascending palatine artery
- → Stylopharyngeus muscle with glossopharyngeal nerve (9th)
 Pass b/w superior and middle constrictor

Deep Layer

- → Salpingopharyngeus
- → Palatopharyngeus
 - Forms a ridge PASSAVANT'S RIDGE
 Prevents regurgitation
- → Stylopharyngeus
- → Superior constrictor forms bed of palatine tonsils

Position of palatopharyngeal sphincter

Structures That Pass

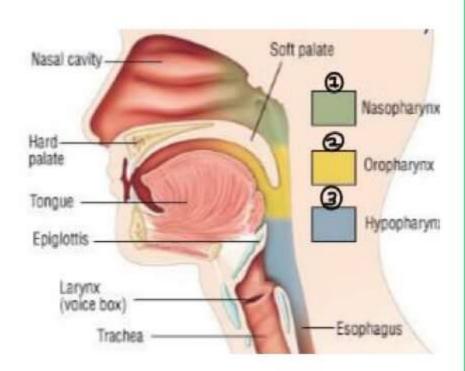
a. Through sinus of Morgagni

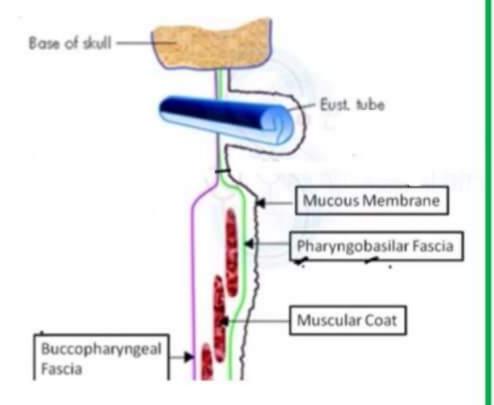
PA - Ascending Palatine Artery

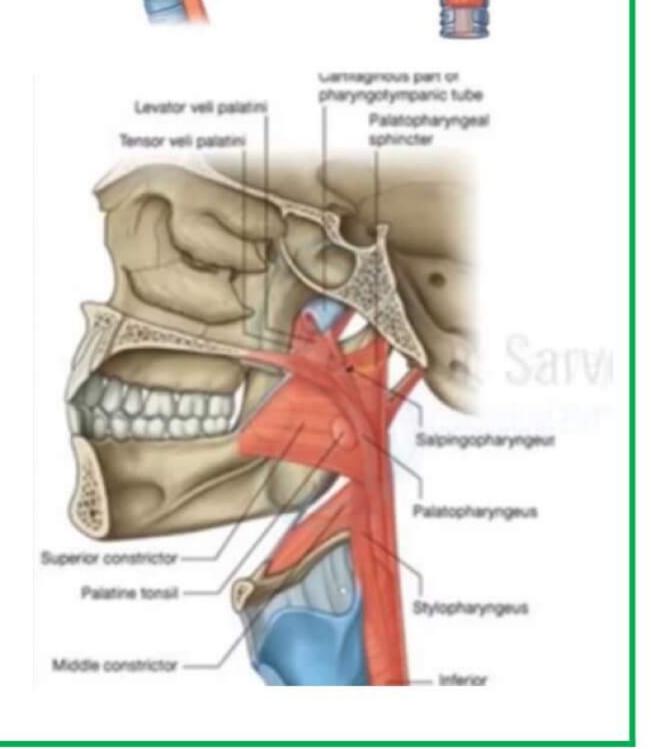
L - Levator veli palatini

AT e - Eustachian tube (Auditory Tube)

- b/w middle and inferior constrictor
 Internal laryngeal nerve
 Superior laryngeal vessels
- c. Below the inferior constrictor
 Recurrent laryngeal nerve
 Inferior laryngeal vessels







Nasopharynx / Epipharynx

Eustachian Tube

- \rightarrow Opens on lateral wall
- → Torus tubarius cartilagenous protrusion

Adenoids

- Lymphoid tissue on posterior superior wall
- Nasopharyngeal bursa → Mid line recess.

Represents the attachment of notochord in embryonic life

Pharyngeal chordoma arises from here.

Rathke's pouch

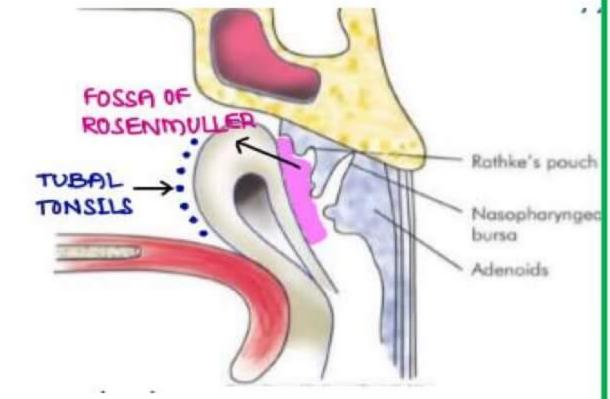
- Place from where pharyngeal mucosa invaginates to form pituitary gland
- Craniopharyngioma arises from here

Fossa of Rosenmuller

Blind recess in posterior superior to Eustachian tube opening. M/C site for origin of nasopharyngeal carcinoma.

Tubal Tonsils

- Lymphoid nodules around ET





Diseases of Nasopharynx

1) Thornwaldt's Disease

- Abscess or cyst formation in nasopharyngeal bursa due to blockage.
- B/L nasal obstruction

 R_x - Incision and drainage (Cruciate incision)

2) Adenoids - Chronic Adenoiditis / Chronic Adenoid Hypertrophy

- Normal physiological growth pattern

3-5 years age = Growth starts

5-7 years age = Max size

>7 years age = Growth size decreases

→ B/L nasal obstruction → Result in mouth breathing

High arched palate

Elongated face Pinched nose Overcrowding of anterior teeth

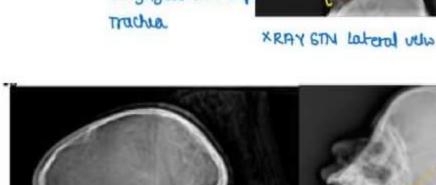
ADENOID FACIES

- B/L serous otitis media → B/L conductive hearing loss
- → Recurrent upper respiratory infection
- → Obstructive sleep apnea

Pulmonary HTN Rt ventricular hyper trophy

Indication of adenoidectomy -> all above

soft palate & uvula Hyoid Bone Epiglottes Cor pulmonale



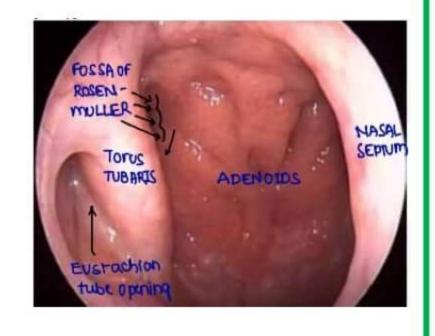
X-Ray STN (Soft tissue of neck)

- → Adenoid hypertrophy
- → Crescent sign / dodd's sign
 - Air in postero superior to mass.

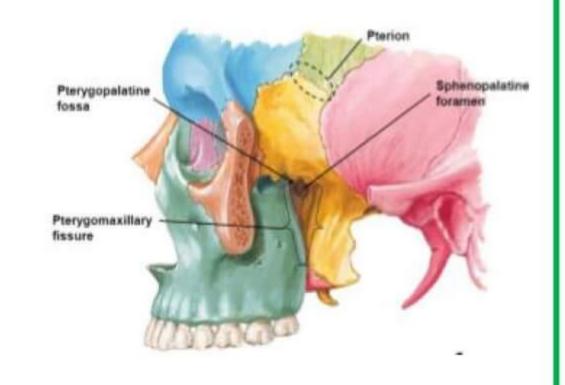
 $R_x \rightarrow Adenoidectomy$

3) Juvenile Nasopharyngeal Angiofibroma / JNA

- → M/C benign tumor of nasopharynx
- → M/C in young male (Androgen dependent tumor) (8-22 years)
- → Site of origin
 - 1. Sphenopalatine foramen
 - 2. Vidian canal
 - 3. Basi sphenoid









- → Sphenopalatine foramen connects the nasal cavity with pterygopalatine fossa
- → Inside pterygopalatine fossa/ sphenopalatine fossa, pterygopalatine ganglion (Largest peripheral parasympathetic ganglion)

→ PTERYGOMAXILLARY TUNNEL

MEDIAL

PTERYGOPALATINE FOSSA

LATERAL

NASAL CAVITY → SPENOPALATINE FORAMEN

→ PTERYGO MAXILLARY FISSURE

INFRATEMPORAL FOSSA

- → Vascular tumor
 - MC blood supply or JAF → Sphenopalatine artery (Br. Of internal maxillary Artery)
- → Exclusively seen in adolescent males (Androgen dependent tumor) → 8-22 yrs
- → Symptoms: No tunica media → Profuse recurrent epistaxis → anemia

Nasal obstruction

Swelling in cheek⊕

O/E → Reddish polypoid mass → Bleed on touch

→ Proptosis

 \rightarrow Tele canthus (Intercanthus length \uparrow , eye ball pushed laterally) \int

 D_X

CECT -

- → Anterior bowing of posterior maxillary wall = ANTRAL/ (HOLMAN MILLER sign)
- → Hyperdense, spindle shape tumor DUMBELL SHAPED TUMOR
- → Widening of pterygo maxillary tunnel HONDUSA SIGN
- → Biopsy is C/I
- $\rightarrow R_x R_xOC$ Endoscopic excision
 - → Surgical techniques
 - Lateral rhinotomy approach

 \downarrow

Medial maxillectomy

- Wide exposure present but scar present
- Transpalatine approach

Exposure limited (Limited by teeth)

- SARDANA'S APPROACH → Transpalatine + sublabial approach
- Midfacial degloving approach (better exposure)
- Endoscopic excision (Preferred method (RxOC) now) blood loss is minimum





4) NASO Pharyngeal Carcinoma (NPC)

- → Male, 8-12 years or 60-70 year age
- → Common in Chinese dry salted fish consumption
- → Cause EBV Association
 - 1. Hoggikin's lymphoma
 - 2. Burkitt's (Non-hodgkin's lymphoma)
 - 3. Gastric adeno ca
 - 4. NPC
 - 5. Immune deficiency associated non-hodgkin's lymphoma
- → M/c site → Fossa of Rosenmuller
- \rightarrow M/c type \rightarrow sq. cell ca (>85%)
- \rightarrow M/c presentation
 - Neck mass (Cervical Lymphnode Metastasis) [75%]
- → 2 malignancies where LN metastasis is early but prognosis is good are
 - 1) NPC
 - 2) Thyroid CA
- → U/L serous otitis media U/L conductive hearing loss
- → Multiple CN palsy -

Earliest CN → 6th nerve

Olfactory nerve is spared

Trotter's Triad - [Sinus of Morgagni Syndrome]

Ipsilateral trigeminal neuralgia -5th CN involvement
Ipsilateral palatal palsy - 10th CN involvement
U/L SOM

TNM Staging

T

T 1 → Limited to Soft tissues of NP

T2 → To nasal cavity or oropharynx

 $T-3 \rightarrow To bone / PNS$

 $T-4 \rightarrow To$ cranial cavity or hypo pharynx or cranial nerves

M

Mo → No Distant metastasis

M1 → Distant metastasis

N For all Head & Neck malignancies except CA NASOPHARYNX

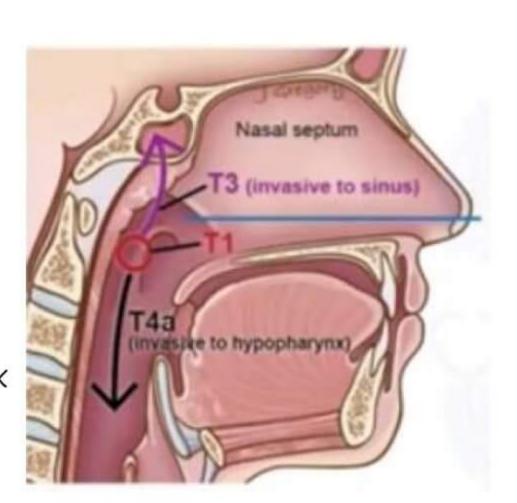
N1 → < 3cm Ipsilateral, Single

 $N_2 \rightarrow 3 - < 6 \text{ cm}$

 $N_{2a} \rightarrow Ipsilateral / single$

N2b → Ipsilateral / multiple

 $N_{2c} \rightarrow B/L$ Or contra lateral





 $N_3 \rightarrow 26cm$

N

 $N = \rightarrow < 6$ cm, Any one Side

 $N_2 \rightarrow < 6$ cm, B/L

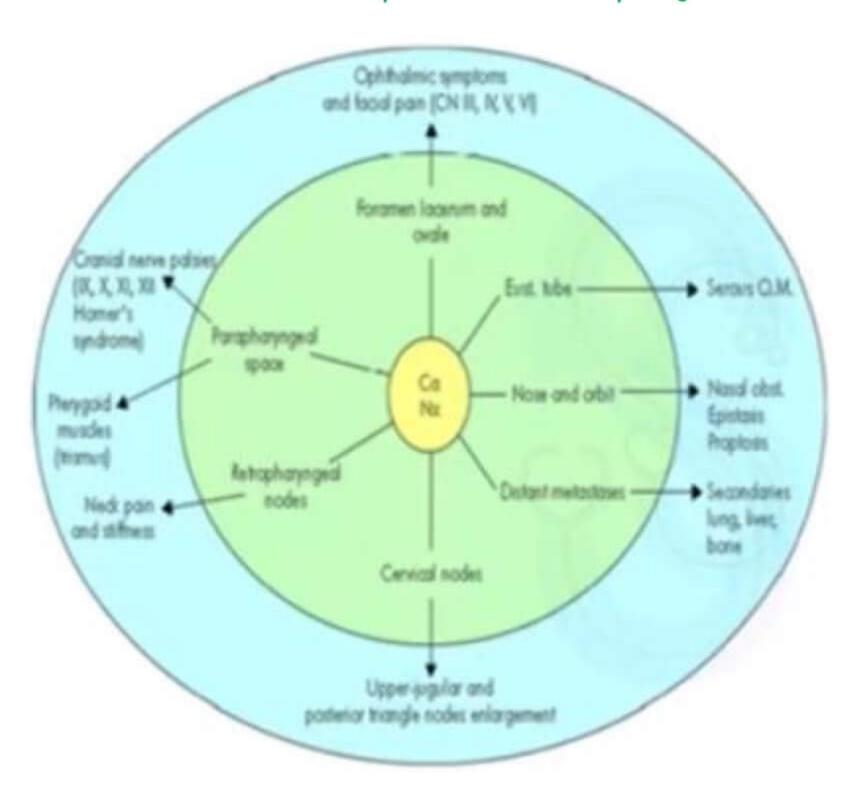
 $N_3 \rightarrow N_{3a} \rightarrow \geq 6$ cm

N3b → HO 'S Triangle or Supraclavicular fossa

Supraclavicular Fossa / Ho's Triangle



Spread OF CA Nasopharynx



Dx 10C→ Endoscopic Biopsy

 $R_x \rightarrow Rxoc - Radiotherapy$

→ Chemotherapy in advanced cases.

Prognostic Markers

- → EBV
- Viral capsid Antigen [VCA]
- Early Antigen [EA]
- → IgA
- IgA to VCA 97% Specific, 95 % sensitive [Screening Test]

 IgA to EA 99% Specific, 90% Sensitive

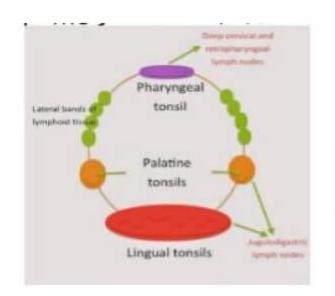
Oropharynx

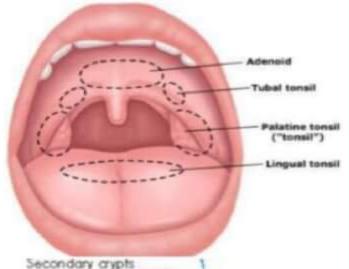
WALDEYER'S Ring

- 1st line of defense

Tonsils

- → B/L, covered by fibrous capsule and forms crypts
- → Tonsillar bed formed by superior constrictor muscle





→ Peritonsillar Space → Between fibrous capsule and superior Constrictor muscle.
Infection of this space is known as — Quinsy / peritonsillar Abscess
Presentation → Pain in throat, Fever, dysphagia, odynophagia
Red enlarged congested tonsil pushing soft palate and uvula.

"Hot potato voice "

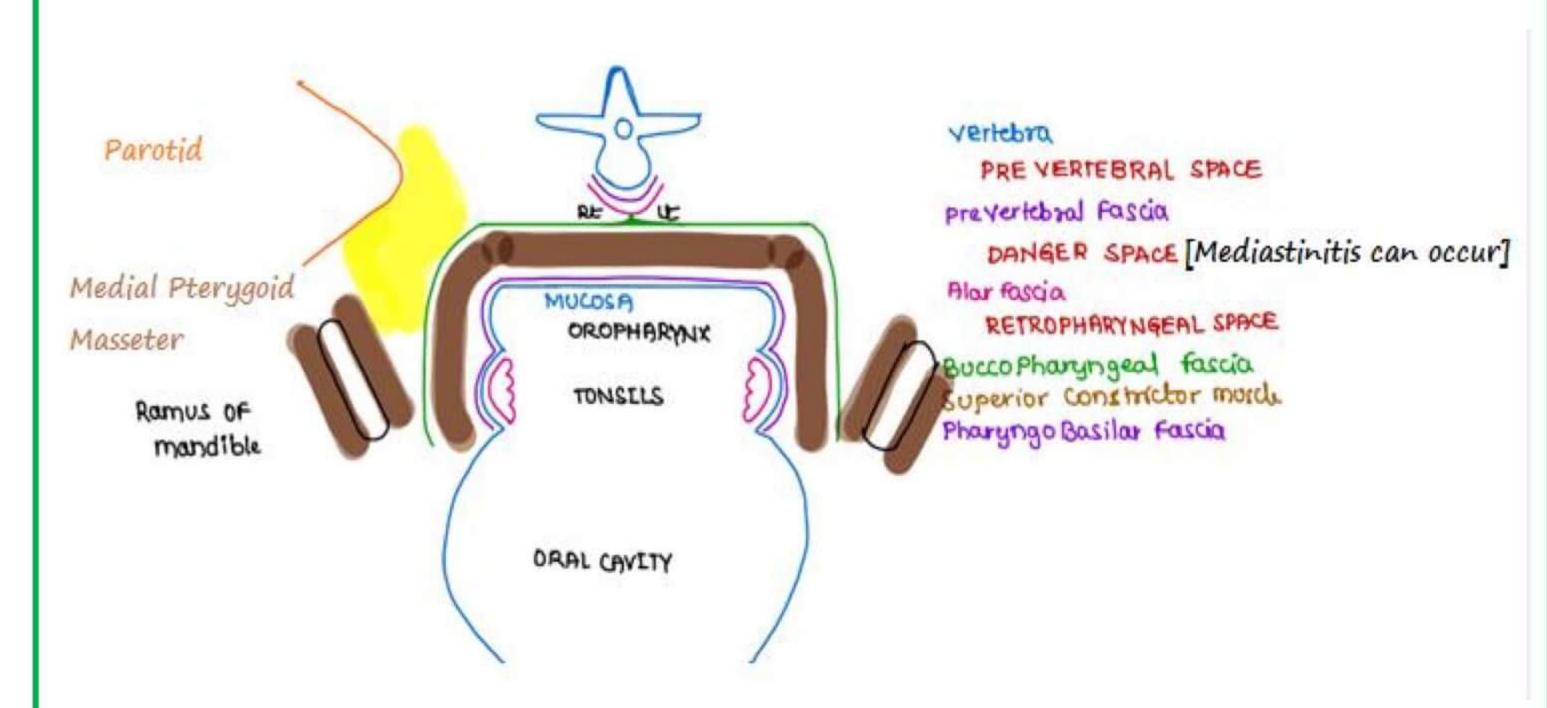
Rx

- → Incision and drainage
- → IV Anti-biotics
- → Interval tonsillectomy done 6 weeks after quinsy
- → Hot tonsillectomy (Tonsillectomy at the time of quinsy) is not advisable

Quinsy Deviated uvula Right tensil

Retropharyngeal Space

- → Divided into 2 parts by midline band which are called space of gilette
- → Infection of LN of Rouviere cause acute retropharyngeal abscess.
- → Retropharyngeal abscess will be on one side (RT.<.)
 Prevertebral abscess will be in midline.





Boundaries

Superior → Skull bone

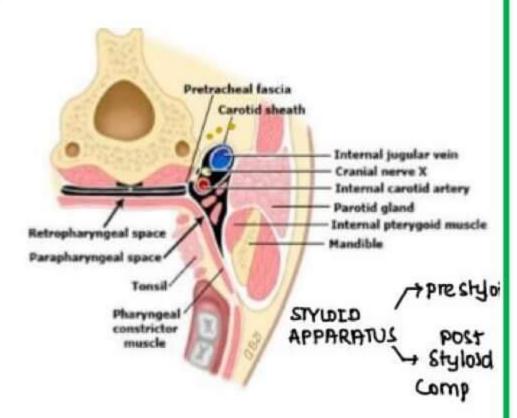
Lateral → Parotid gland, ramus of mandible, medial pterygoid muscle

Medial \rightarrow Oropharynx with superior constrictor muscle, palatine tonsil

Inferior → Hyoid bone

Contents

- Styloid process along with muscles STYLOID APPARATUS
- Styloid process divides PPS into
 - · Pre styloid compartment Consist of fat Trismus
 - Post styloid compartment –
 ICA (Internal carotid artery)
 Internal jugular vein
 CN X, IX, XI, XII



Q 7 YR OLD CHILD WITH QUINSY AND SEVERE TRISMUS, NEXT LINE OF MANAGEMENT /RXOC?

A. Intra oral drainage

B. IV antibiotics for 48hrs

C. Ext. drainage

D. Tracheostomy

Blood Supply of Tonsils

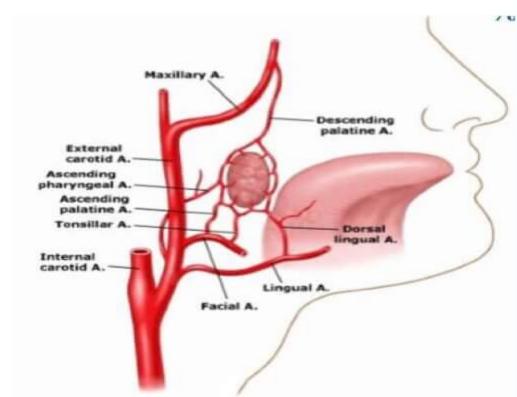
- 1) Tonsillar artery (Branch of facial artery)
 - Main blood supply
- 2) Ascending palatine artery
- 3) Descending palatine artery
- 4) Ascending pharyngeal
- 5) Dorsal lingual artery
- ightarrow Venous drainage by PARA TONSILLAR VEIN.

Acute Tonsillitis

- → Common in children
 - 1. Acute catarrhal tonsillitis
 - Red, congested, seen in viral infection
 - 2. Acute follicular tonsillitis
 - 3. Acute parenchymal tonsillitis
 - 4. Acute membranous tonsillitis
- → M/C organism Streptococcus
- ightarrow Symptoms ightarrow Enlarged and tender jugular digastric node
- → Rx Penicillin → Doc

Chronic Tonsillitis

- → Bacteria grow and resides in crypts
- → Recurrent sore throat
- → IRWIN MOORE Sign: Pus comes out on pressing the anterior pillar.
- $\rightarrow R_x$ Tonsillectomy







Tonsillectomy

Indication

- 1. Recurrent sore throat PARADISE CRITERIA OF SORE THROAT (Detailed one)
 - ≥ 7 episodes in one yr or
 - > 5 episodes / yr for last 2 yrs or
 - > 3 episodes / yr for last 3 consecutive yrs or if a child misses school
 - for ≥ 14 days in 1 yr because of sore throat

- 2. Quinsy
- a. Episode in child
- b. Episode in an adult

Absolute indication

- 3. Obstruction
- 4. Malignancy
- 5. Febrile seizures
- 6. Rheumatic Tonsillitis (GABHS Gr. A β haemolytic Streptococcus) infection
- → Position
 - 1. Rose's position [For adenoidectomy too]
 - Thoraco cervical joints are extended.

Advantage

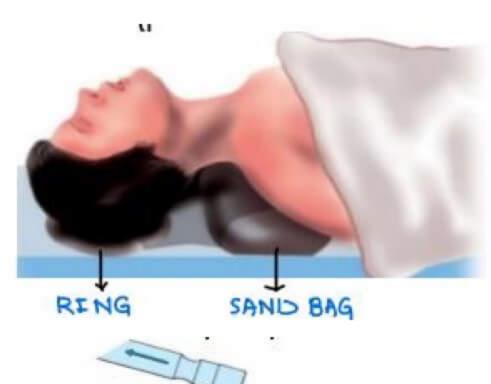
- Prevents the entry of blood in airways
- 2. Boyce position / Morning sniff / BARKING DOG

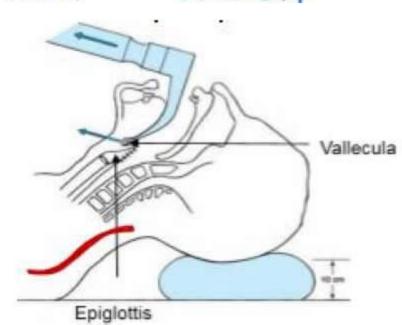
Cervical joint - Extended

Thoracic joint - Flexed

Used for laryngoscopy, esophagoscopy, Bronchoscopy

Complication of tonsillectomy \rightarrow Bleeding (m/c)





Primary	Reactionary	Secondary
 Intra operative Venous bleeding Mc source → para tonsillar vein Rx → ligation 	 Within 24 hrs Slippage of ligature Most dangerous Rx → Re ligation 	 5-7 days d/t secondary infection Warning bleeding ⊕

- Referred pain to ear (D/t glossopharyngeal N.)



Classical Method - DISSECTION & SNARE METHOD / COLD STEEL INSTRUMENTS

- · Crushed & cut with EVE's Tonsillar snare
- Pain minimal (No heat used)
- Bleeding is maximum

Electro Cautery

- No bleeding
- Post op pain is maximum

CO- Ablation

- Uses radiofrequency to break Nacl to Na+ & cl- ions
- Na× ions used to dissect the tissue → no bleeding
- Minimal post op. pain no heat used
- High cost & healing is slow

Hypopharynx / Laryngo Pharynx

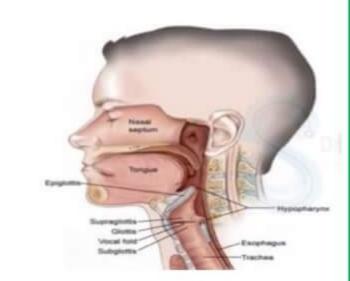
→ Thyropharyngeus Supplied by SLN Cricopharyngeus by RLN

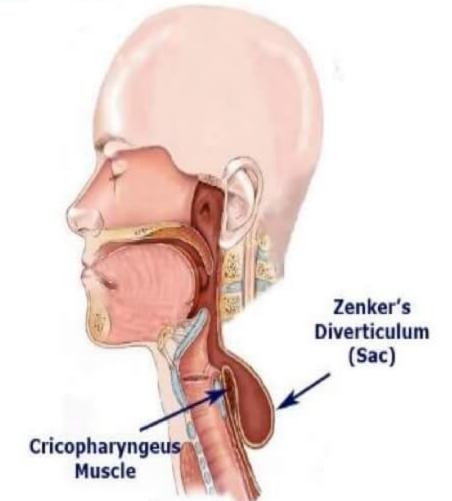
ZENKER'S / Pulsion Diverticulum

→ M/C SITE → KILLIAN's Dehiscence Also develop from laimer's dehiscence and Killian – Jameison diverticulum

- → C/F
- Regurgitation of old eaten food at night times
- Dysphagia (M/c)
- Halitosis
- $\rightarrow Dx$
- Barium swallow
 - In case of malignancy
 - Irregular margins present
 - Filling defect present
 - It is also present in case of food particles
 - To d/t repeat barium swallow after some time
 - Video fluoroscopy
 - Can check filling detects
 - Esophagus copy or Endoscopy are c/I [Risk of perforation]
 - To take Biopsy, it is indicated [Not for Dx].











- Excision
- DOHLMAN 'S Operation / Endoscopic Diathermy → RxOC
 - Common wall btw diverticulum & esophagus removed
- ightarrow ZD develops above the upper esophageal sphincter epiphrenic diverticulum develops above the

lower esophageal sphincter

- → ZD is a pseudodiverticulum
- → TRACTION DIVERTRTICULUM is a true diverticulum







Plummer Vinson Syndrome/ Patterson Brown Kelly Syndrome

- 1. Post corticoid webs
- 2. Fe-deficiency anaemia → More common in Females, low socio economic status &

developing countries

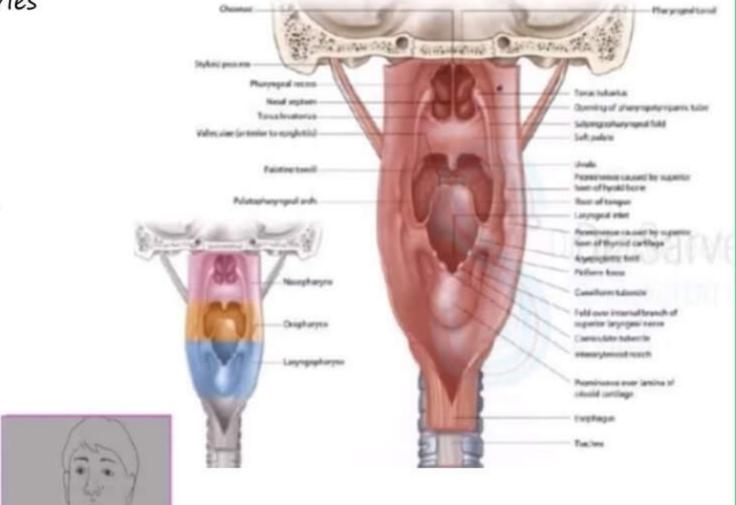
- 3. Koilonychias
- → Post cricoids webs → Pre malignant
 - → More common in Q → post cricoid cancer

$\rightarrow O/E$

- Post cricoid crepitus absence Moure sign
 Moure sign is that in hypopharyngeal CA
- CHEVALIER JACKSON'S SIGN
 Collection of secretion in pyriform fossa

$\rightarrow Rx$

Depends on stage of CA
 Early stages – Sx
 Late stages – Chemo radiation



Larynx

Embryology of Larynx

- → Formed from fusion of 4th and 6th branchial arches (Cartilaginous structure)
- ightarrow 4th arch blood supply ightharpoonup Rt. Rt. Subclavian artery Lt. Arch of aorta
- ightarrow 6th arch blood supply ightharpoonup Rt. Rt. Pulmonary artery Lt. Ductus Arteriosus
- \rightarrow Nerve supply by vagus nerve (X) SLN -4^{TH} arch RLN -6^{th} arch
- → Muscles formed by
 - 4th arch All constrictor muscles of pharynx except cricopharyngeus
 - 6th arch Cricopharyngeus
 - All intrinsic muscles of larynx except cricothyroid
- → Cartilages
 - 4th arch Cu Cuneiform
 - T Thyroid
 - E Epiglottis
 - 6th arch Co Corniculate
 - C Cricoid
 - Aine Arytenoid

Cartilage of Larynx

LARYNX is placed at the level of C3 - C6 vertebra

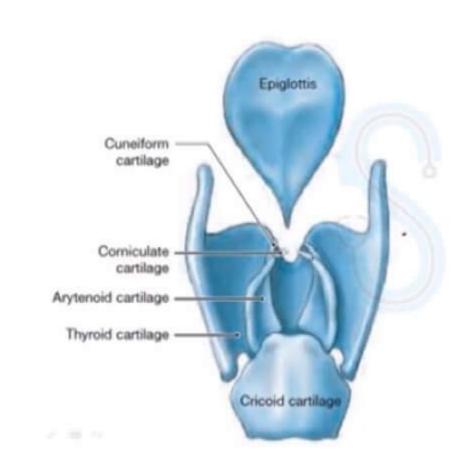
Larynx is placed a little higher in children's and females

- → Formed by 3 unpaired and 3 paired cartilage
- Unpaired 1) Epiglottis
 - 11) Thyroid
 - III) Cricoid
- Paired-
- 1) Arytenoid Make posterior 1/3 of vocal cord
- 2) Corniculate
- 3) Cuneiform

Epiglottis → Elastic cartilage

In adult \rightarrow Leaf shaped

Children → Omega-shape





Thyroid Cartilage – 'v' shape with right and left lamina, both lamina meet $\rightarrow C_3 - C_4$ / upper border of C_4

- Thyroid cartilage Anteriority from 90° angle in male 120° angle in female

- Prominence of thyroid cartilage in male - Adam's apple

Cricoid Cartilage – It is combined to the trachea with the help of crico-tracheal ligament

- Is only cartilage forming a complete ring.
- Synovial joints in cricoid cartilages are 1. Crico-arytenoid

2. Crico-thyroid

Arytenoid Cartilage – Has 2 important processes 1. Vocal process 2. Muscular process

Corniculate Cartilage - They are fibroelastic in nature

Cuneiform Cartilage - They are club shaped cartilages

- Present in the free fold of aryepiglottic folds
- They are fibroelastic in nature
- → Any muscle/ membrane / ligament that present in b/w 2 cartilages → INTRINSIC
- → Any muscle/membrane/ligament that connects Cartilages to external structure → EXTRINSIC
 - Thyrohyoid membrane
 Thyrohyoid ligament
 Cricotracheal ligament

EXTRINSIC

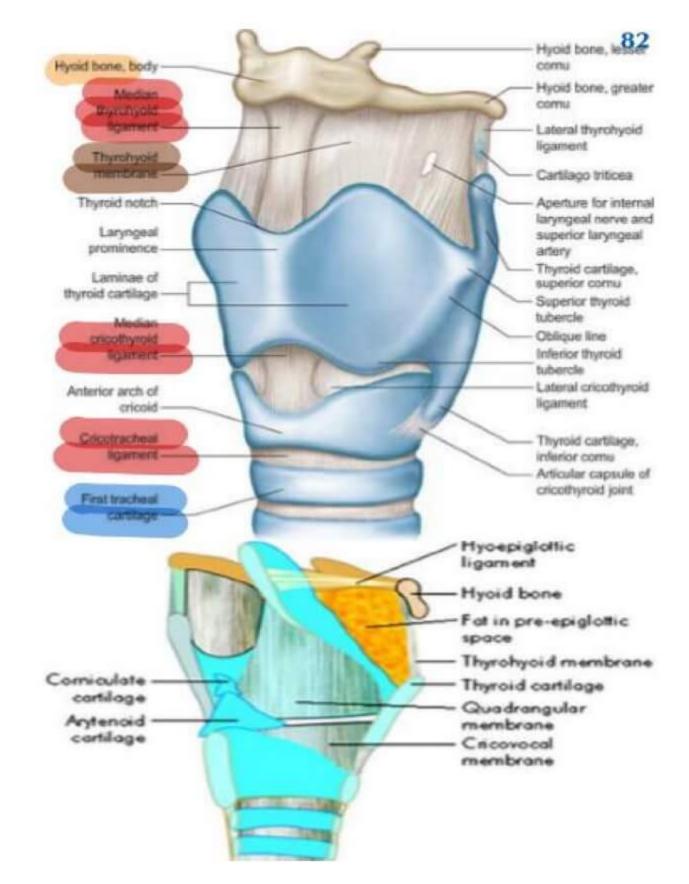
Cricothyroid membrane

Cricothyroid ligament

Cricothyroid muscle

INTRINSIC

[Only intrinsic muscle of larynx]





Palatine tonsil

Hyoid bone

Epiglottis

Thyroid cartilage

Uvula

→ PRE Epiglottic Space

- Boundaries
 - 1. Superior → Hyo epiglottic ligament
 - 2. Posterior → Epiglottis

Thyro epiglottic ligament

3. Anterior → Body of Hyoid

Thyrohyoid ligament

Thyroid cartilage [Small part]

Space is open on 2 sides & communication with paraglottic space

- Fat filled space
- Potential space [Not a actual space]
 - Spread of malignancy into it → T3
- Epiglottis is divided into 2 parts:
 - 1. Suprahyoid epiglottis
 - For malignancy purposes 2. Infrahyoid epiglottis

[Suprahyoid malignancy goes to tongue base Infrahyoid malignancy goes to pre epiglottic space]

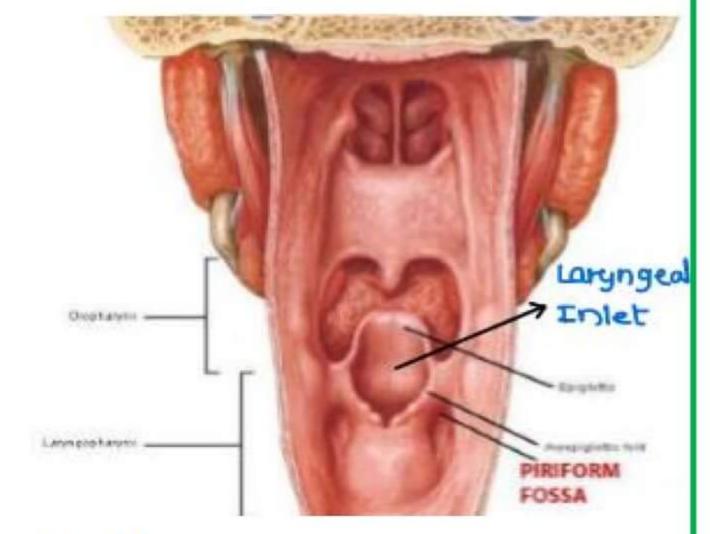
Laryngeal Inlet

→ Anteriorly → Epiglottis Posteriorly → Arytenoids [Pair] Ary Epiglottic fold in B/w them

> Part of larynx that contains vocal fold is Glottis

Supra glottis → Part above the glottis

Sub glottis → Part below the glottis



Pharyngeal tonsil

Nasopharynx

epigloms

Infrahyoid epiglottus – Laryngopharynx

Pre - epiglottic

Space

suprabyoid

cross sectional anatomy of Larynx

Largngeal Inlet Supra glotte ventricle Glottes

sub glotte

Ary epiglottic fold Epiglottis Arytenoid cartilage

false vocal corde/vestibular fold/ventricular folds

vocal fold [True vocal cords]

Largngeal vestibule

Ventricle

- → Forms ventricle
- → Only true space of larynx

PrepLadder

False Vocal Cords / vestibular Folds / Ventricular Folds

Basement membrane

oran.

→ In glass blowers, wt lifters, trumpet players,

Pressure produced by glottis closure

Ventricles starts increasing in size [Upside]

Laryngocele

Laryngocele

- → Air filled cystic swelling in neck
- > Arises from anterior most part of ventricle SACCULE

Types → 1) External - BRYCE sign

[On compressing, the swelling & size with a gurgling sound of air escape]

(BOYCE SIGN - seen in zenker's diverticulum (Sound of fluid escape)

II) Internal – Foreign body sensation in throat cough, noisy breath. $O/E \rightarrow Smooth swelling of vestibular fold & Ary epiglottic fold$

Dx - CT - Scan in Valsalva procedure

R_x External → Sx excision

Internal → Transoral micro laryngeal Sx

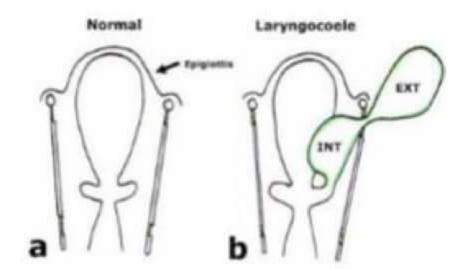
Combined → Sx excision

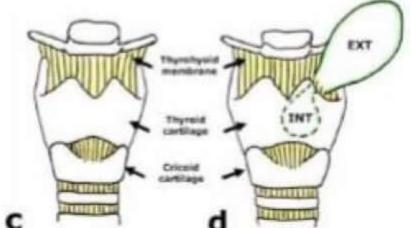
Glottis

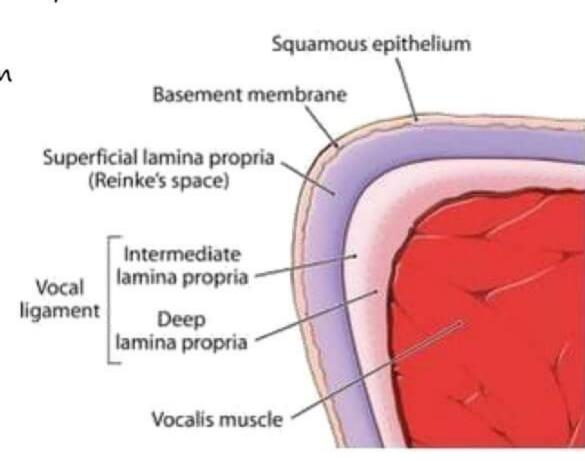
- Consist of 2 vocal folds (One on each side)
- Earlier they were named as (True vocal cords)
- Vocal folds are lined by non-keratinizing stratified squamous epithelium
- Sub epithelium space is known as Reinke's space
- Junction between columnar epithelium and squamous epithelium is known as arcuate line
- 5 layers in the vocal folds
 - 1. Non-keratinizing stratified squamous epithelium
 - 2. Lamina propria
 - Superficial
 - Intermediate
 - Deep
 - 3. Vocalis muscle
- → Narrowest part of larynx in child Sub glottis
- → Narrowest part of larynx in adult Glottis



Quadrangular membrane







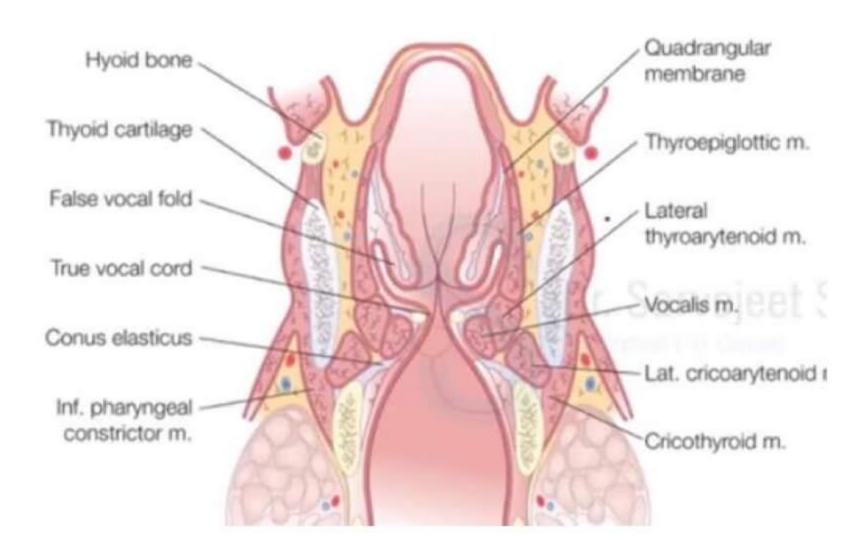
Sub Glottis

→ Consists of ring of cricoid cartilage

Para Glottic Space

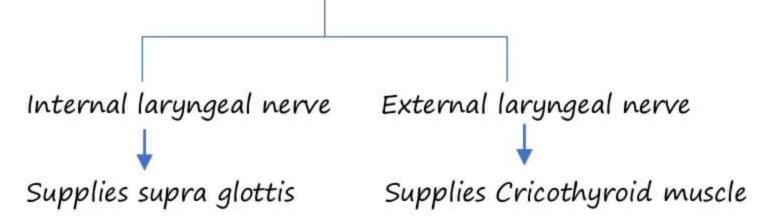
Boundaries

- 1. Antero laterally Thyroid cartilage
- 2. Supero medially Quadrangular membrane and vestibular fold
- 3. Infero medially Conus elasticus
- 4. Posteriorly Pyriform fossa
- → Para glottic space is communicating anteriorly with pre epiglottic space



Nerve Supply of Larynx

- → Xth Cranial nerve
 - 1. Superior laryngeal nerve



2. Recurrent laryngeal nerve → supplies sub glottis

All intrinsic muscles of larynx except cricothyroid

→ At level of glottis ILN (above) and RLN (below) supplies via Galen's anastomosis

Functions of Larynx -

Primary function – Protection of lower airways
Other – Voice production.



Muscles of Larynx -

- 1. Abductor Posterior cricoarytenoid
- 2. Adductors Inter arytenoid

Thyroarytenoid Lateral cricoarytenoid

 Tensor muscle – Cricothyroid Vocalis

All muscles lie inside larynx expect cricothyroid.

All muscle of larynx are supplied by Recurrent laryngeal nerve (RLN) expect cricothyroid. Which is supplied by External branch of superior laryngeal nerve.



Congenital Disease of Larynx

1. Laryngomalacia

- → M/C congenital abnormality of larynx
- → Soft laryngeal cartilage

C/F - Inspiratory stridor → from birth cry is normal [Expiratory phenomenon] crying ↑ stridor

- In prone position → stridor improve
- → O/E Omega shaped epiglottis
- → R_x Reassurance Conservative

Laryngomalacia disappear by 2 years of age.



2. Subglottic Hemangioma

- → Vascular malformation
- → C/F stridor at 3 -6 months of age
 - · Normal
 - Glottis and sub glottis → Biphasic stridor
 - Supraglottis and above → Inspiratory stridor
 - Below sub glottis → Expiratory stridor
 - In sub glottic hemangioma has inspiratory stridor (Because of vascular

malformation)

- \rightarrow Dx Fiberoptic / direct laryngoscopy \rightarrow Reddish blue mass in sub glottis
- \rightarrow Rx (1) Save the airway \rightarrow Tracheostomy (First)
 - (11) CO2 laser excision or injection of steroid or sclerotherapy

3. Larnygeal WEB

- → Membrane formed d/t incomplete canalization of airway
- → M/C site → Anterior glottis
- → C/F → Biphasic stridor from birth
- $\rightarrow O/E \rightarrow Web$ is seen
- → Types congenital → cartilaginous

Acquired → membranous







- $\rightarrow R_x$
- \rightarrow CO₂ laser excision \bar{c} silicon is placed between vocal folds for few weeks to months to prevent recurrence.



Acute & Chronic Inflammation of Larynx

Acute Epiglottitis / Supra Glottitis

→ Mcc: - Hemophilus influenza type B (HIB)

in developed countries - streptococcus pneumoniae

 \rightarrow Children = 2 - 7 years

C/F →

Drooling-> Not able to swallow even saliva

Dyspnea

Tachypnea

Tachycardia

Tripod sign

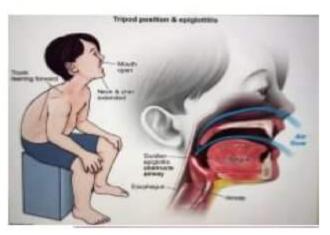
Rising sun sign' → red inflamed epiglottis

→ Instrumentation is C/I



- X Ray
- Thumb sign
- Thickening of epiglottis

→ R_x → Doc → ceftriaxone Steroid nebulization Tracheostomy (If stridor is present)







Cricothyrotomy

→ Used to open up the airway in emergency situation (Done outside the hospital)

Tracheostomy

Indications

5R's

Respiratory obstruction

→ High tracheostomy

· b/w 1/2nd rings

Respiratory collapse

Respiratory secretions

Respiratory insufficiency → Low tracheostomy

· b/w 3/4th rings

To secure the Respiratory pathway



4. Acute Laryngo-Tracheo Bronchitis [CROUP]

- → Mcc Para influenza virus type 1, 2
- → Children 3 months 3 years

C/F → Fever, cough (Barking cough)
Expiratory stridor

O/E → Sub coastal, inter coastal retraction
B/L conducted sound on Auscultation

 $C - X - Ray \rightarrow Steeple sign$

Rx → Symptomatic



5. Laryngeal TB

- → Always associated with pulmonary TB
- → Submucosal nodules
 - M/C site → Posterior larynx
 - I^{st} sign \rightarrow Hyperemia of vocal fold \bar{c} Incomplete adduction
- → Mamillated appearance of nodules Mouse nibbled appearance of vocal folds Turban epiglottis
- $\rightarrow R_x$
 - Rx pulmonary TB

6. Contact Ulcer / Granuloma

- → M/C site → posterior ^{1/3} of vocal folds MCC → LPR (Laryngo – pharyngeal reflux) Other → vocal abuse
- → C/F → Hoarseness of voice
- → In long term intubation Intubation granuloma
- $\rightarrow R^{\times}$
 - PPIs ā speech therapy (For LPR)
 - Intubation granuloma
 - $-R_x \rightarrow Antibiotics$ and steroids













Benign Lesions of Larynx

1. REINKE'S Edema

- → Mcc Smoking Other- Faulty speech
- → Boggy spindle shaped vocal folds → SMOKER's POLYPS
- → Females
- → Rx Cessation of smoking

 $R_x \rightarrow Stripping of epithelium from vocal folds$ 2 stages $S_x \rightarrow do$ one side at a time

2. Vocal Nodule / Singer's Nodule / Screamer's Nodule

- → Due to Chronic misuse of voice
- → Seen in Singer's teachers
- → B/L, Sessile (Fixed on free margins)
- \rightarrow M/C site at the junction of anterior $^{1/3}$ rd and posterior $^{2/3}$ rd. [Area of maximum vibration]
- \rightarrow C/F \rightarrow Hoarseness of voice
- $\rightarrow R_x$
- Speech therapy
- PPI (LPR's is a major contributor).
- Early / soft nodules
 - With speech therapy & PPI
- Late/ hard nodules
 - RxOC speech therapy & PPI
 - S_x

3. Vocal Polyp

- \rightarrow Single, U/L, pedunculated, moves \bar{c} Respiration
- → M/C site → Anterior 1/3 rd and post 2/3 rd
- → Cause Sudden vocal abuse
- → C/F Hoarseness, diplo phonia

Rx → Micro laryngeal Sx

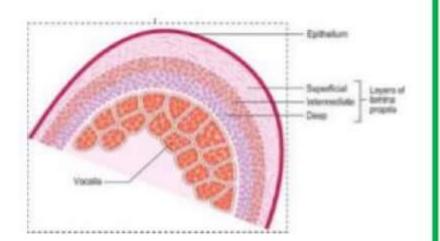
4. Recurrent Respiratory Papilloma

- \rightarrow A/W \rightarrow HPV 6, 11
- → Types

Adult \rightarrow Limited, single, U/L, doesn't recur after S_x Juvenile \rightarrow Multiple, B/L, stridor positive, highly recurrent vertical transmission positive

→ M/C → Site anterior part of glottis Juvenile → Tracheostomy is C/I Risk of spread

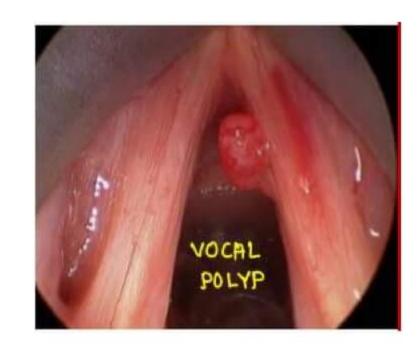
But in emergency → Low tracheostomy





Vocal Nodules









ADULT JUVENILE



$R_x \rightarrow Co_2$ laser excision

Micro debrider excision→ Now preferred.

Medical Rx

- Chemotherapy
- Newer photodynamic therapy
- Interferon α (↓ recurrence)

Vocal Cyst

- → Collection of fluid in vocal fold
- → Rx Micro laryngeal Sx
- → D/D- Lipoma

Dx made during Sx

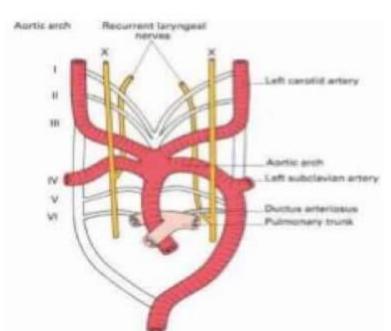
Vocal Cyst

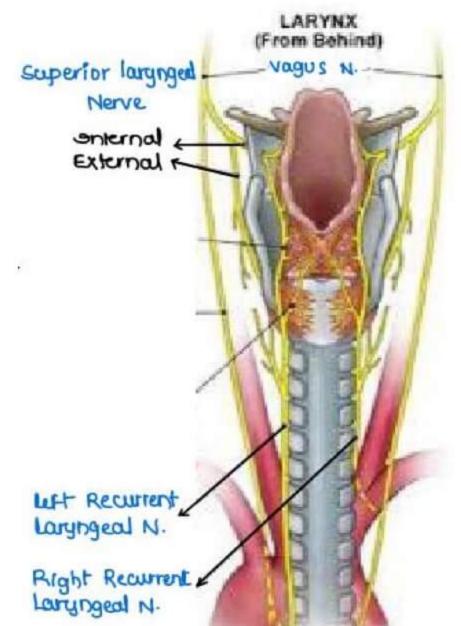


Vocal Fold Palsy

Nerve Supply of larynx

- → It RLN, longer in course in thorax [Takes a turn around arch of aorta]
- → In 10% of cases, Rt side may be Non-Recurrent Also
- → MC nerve injured in total thyroidectomy Ext. laryngeal nerve
- → Injured during CT Sx → Lt. RLN
- → More commonly injured in total thyroidectomy Among RLN → Rt. RLN [superficial]





Semon's Law

 \rightarrow In case of any progressive neurological disorders, the Abductor fibres of RLN are 1st to be affected [Phylogenetically new] \rightarrow Vocal folds lie in adduction

Wagner & Grossman Hypothesis

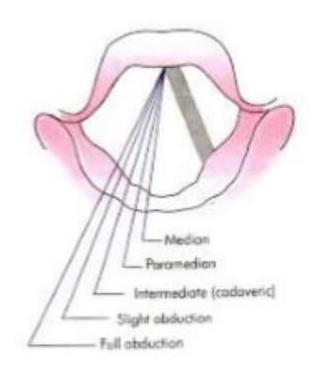
→ All muscle are causing adduction in larynx, In RLN palsy all undergo palsy except cricothyroid [Supplied by external laryngeal Nerve] which is the reason for unopposed adduction

Positions Of Vocal Folds

- 1. U/L ELN palsy
 - > Production of high pitched voice problem only
 - → Go unnoticed
- 2. B/L ELN palsy
 - → Go unnoticed
 - > Severe problem in production of high pitched voice



- > Para median position d/t incomplete adduction
- → No problem in airway
- → Compensatory hypertrophy of other vocal fold in few weeks-months → voice with will improve
- 4. U/L RLN + U/L ELN injury
 - → Cadaveric position of vocal fold on one side
 - → No problem in breathing
 - → Voice will not improve





- \rightarrow R₁ \rightarrow Push the paralytic vocal fold to midline
- → U/L vagus Nerve injury [ILN also included]
 - Problem of aspiration also present
- → Manual Compression test is done
- > Type I ISSHIKI THYROPLASTY
 - 4 types I, II, III, IV present
 - Aka medialization thyroplasty / Approximation laryngoplasty
 - Benefits :-
 - · Improves the voice
 - In X nerve palsy, it prevents aspiration
- 5. B/L SLN injury
 - \rightarrow Cough reflex on both sides gone \rightarrow Aspiration Gold std R_x in intractable aspiration \rightarrow Tracheal separation & permanent Tracheostomy
- 6. B/L RLN Palsy / B/L Abductor palsy
 - → Both vocal cords in para median position
 - → Only 2-3 mm airway to breath
 - → Biphasic stridor (+)
 - → Voice is good
 - → Rx Type 2 Thyroplasty / Lateralization Thyroplasty
 - → Laryngo fissure

Type III Thyroplasty

- → Done for Puberphonia [Female like voice in a male]
- ightarrow GUTZZMANN TEST done to confirm whether the patient will improve with type 3 thyroplasty

Type 4 Thyroplasty

→ Done for Androphonia [Male like voice in a female]



Carcinoma Larynx

M/C in a male - smokers, Alcoholics

M/C type \rightarrow sq. cell ca

M/C site \rightarrow ca glottis

M/C presentation - Hoarseness

Best prognosis - Early presentation, late spread

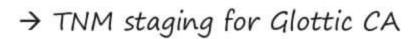
 \rightarrow M/c presentation of ca supra glottis \rightarrow Foreign body sensation

Pain of swallowing

- → M/c presentation of ca sub glottis Stridor [Poor prognosis]
- $\rightarrow D_x + 10C$ direct larynogoscopic biopsy

CT scan of neck - 1) extent of neck (T)

X - Ray chest - distant metastasis (M)



T₁ → Normal vocal fold mobility

 \rightarrow T₁a \rightarrow one

 $\rightarrow T_1b \rightarrow two$

T2 > Impaired vocal fold mobility

T₃ → Fixed vocal folds [Palsy]

3P → Pre epiglottic

Para glottic

Post cricoid area

T₄ → Outside larynx

T4a

T4b → Superior mediastinum

Prevertebral space

Encases carotid artery

→ TNM staging for supra glottic CA

5 sub sites

- Supra hyoid epiglottis
- Infra hyoid epiglottis
- Arytenoids
- Vestibular folds
- Aryepiglottic fold

 $T_1 \rightarrow 1$ subsite

 $T_2 \rightarrow > 1$ subsite

Mucosa of base of tongue or glottic spread (No vocal fold palsy)

 $T_3 \rightarrow Vocal fold palsy$

 $T_4 \rightarrow Outside larynx (Same as glottic CA)$



CA GLOTITIS



→ TNM staging for CA sub glottis

T₁
 T₂ Not considered (Present late)
 T₃ T₄ Same as glottic CA

 $\rightarrow R_x$

- T1 / T2 Stage of CA Glottis
 - Radiotherapy > R1OC [Follow up of 6 Wks required]
 - T1a → Laser cordotomy
 - Poor voice results
 - · Single sitting is enough
- > T3 Lesions
 - Concurrent chemo radiotherapy [CCRT] R10C
 - Exception → Bulky lesion
 Associated lymph nodes
 Multiple sites involvement

 S_x is the R_1

- Perichondritis is absolute C/1 for radiotherapy $\rightarrow S_x$ is R_4
- → T4a
 - Combined modality

S× → Radiotherapy & chemotherapy

→ T₄b - Palliative R_x

Total Laryngectomy

→ High tracheostomy done in total laryngectomy

Post Laryngectomy Voice Rehabilitation

1) Electro Larynx

- > Small hand held device mechanical vibration
- → Disadvantage → Produces monotonous sound

2) Oesophageal Speech

→ Airway is separated from digestive pathway.
Disadvantage → Only speaks few words at a time





ELECTROLARYNX

3) Tracheo-Oesophageal Speech

- → Consist a one way valve Placed between trachea and esophagus—
- → Can speak complete sentences
- → Best way of voice rehabilitation
- → Disadvantage Has to be replaced once from 6 months 2 years
 Costly

