بسم الله الرحمن الرحيم

**This file contains the following topics:

1) acute otitis media
2) otitis media with effusion
3) chronic otitis media
4) hearing assessment
5) hearing loss
6) vertigo
7) epistaxis
8) tracheostomy
9) strider & hoarseness of voice

** missed topics:

1- foreign body
2- facial N. Pulsy
3- tinnitus
4-tonsils & adenoids
5-acute rhino-sinusitis
6-allergic & non allergic rhinitis
7-nasopharyngeal cancer
8-laryngeal cancer

Acute Otitis Media

Presented by :
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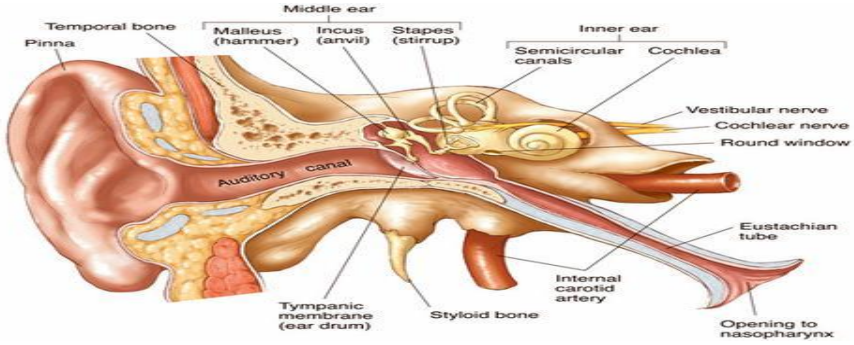


- The ear consists of three major parts: the outer ear, the middle ear, and the inner ear. The outer ear includes the pinna—the visible part of the ear—and the ear canal. The outer ear extends to the tympanic membrane or eardrum, which separates the outer ear from the middle ear.
- The middle ear is an air-filled space that is located behind the eardrum. The middle ear contains three tiny bones, the malleus, incus, and stapes, which transmit sound from the eardrum to the inner ear.
- The inner ear contains the hearing and balance organs. The cochlea contains the hearing organ which converts sound into electrical signals which are associated with the origin of impulses carried by nerves to the brain where their meanings are appreciated

- **Acute otitis media (AOM) :**

is a **short-lived (usually 5-7 days)** infection of the middle ear. If it is **viral** it may last **as little as a day** or so but it can persist, causing pus to accumulate under pressure behind the eardrum, which may perforate .

Before the eardrum perforates, AOM is intensely painful. It mainly occurs in children. Recurrent otitis media (ROM) refers to repeated such episodes, typically more than three in a 6-month period.

- **Otitis media with effusion (OME) :**

is also common in children. **Fluid – often thick sticky 'glue'** – accumulates in the middle ear behind an **intact drum**. Because some fluid in the middle ear is normal for up to several weeks after an episode of AOM, **the term OME requires that the fluid be persistent for at least 3 months.**

- **Chronic otitis media :**

This implies that the eardrum has **perforated,** the perforation has failed to heal and there is ongoing infection. The term chronic suppurative otitis media **(CSOM)** is often used to emphasize the tendency for ears with longstanding perforations to become infected and discharge

Classification of otitis media according to:

- 1) Duration
- 2)Nature of fluid/discharge : suppurative and non-suppurative
- 3)Otitis media with effusion and Aero-otitis media
- 4)Causative organism –viral /bacterial

Clssification of OM according to the duratoin of illness

1) Acute otitis media (AOM):

Rapid onset of symptoms < 3 weeks

2)Subacute otitis media :

Symptoms lasting for 3 weeks to 3 months

3) Chronic otitis media :

Symptoms lasting for 3 months or longer

Epidemiology

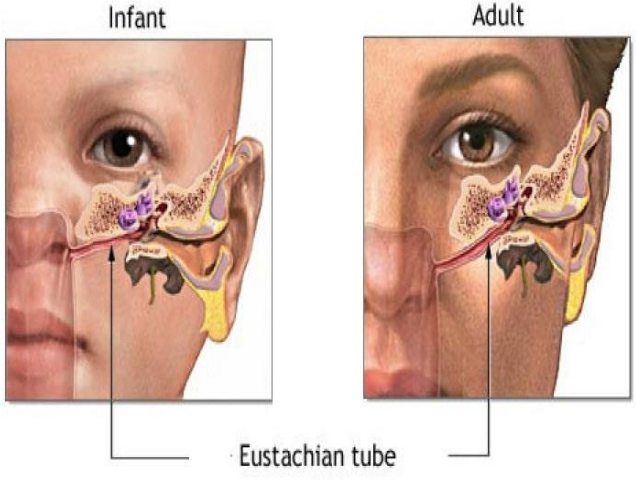
- **Otitis media** (OM) is the second **most common** disease of childhood.
- Most **children** will experience some form of acute otits media during their lifetime , **3 out 4 kids have had at least one ear infection by the time they reach 3 years of age**.
- The peak age of incidence **is 6 months to 3 years** old and it is rarely seen above 5 years of age.
- Otitis media occurs more in the winter than summer months as it is usually associated with a cold.
- It can occur in **adults** **but this is unusual.**

Risk factor of otitis media :

- Boys are more likely than girls to develop **otitis media .**
- Children with older siblings at school or nursery are exposed to infections that may be brought home.
- Children who suffer with many colds or respiratory infections are more likely to develop OME.
- Parent's smoking is thought to be associated with an increase in both acute and chronic otitis media as it is believed to increase S.pneumonia colonization.

Why OM more common in childhood?

1. ET → shorter,wider,more horizontal : making it easier for the bacteria to travel from throat and upper RT .
2. Upper Resp tract infection is more in children
3. regurgitation of milk and vomiting.
4. Adenoid → obstruction of Eustachian tube.



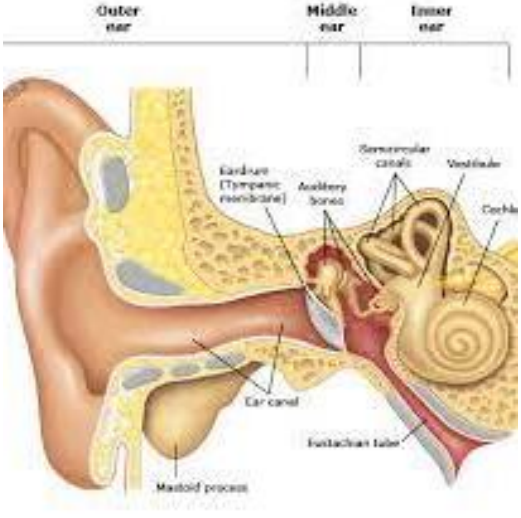
Acute otitis media (AOM)

Acute otitis media is **common and frequently bilateral**. Most children will develop one or more episodes typically before they are 2 years old.

It can follow an acute upper respiratory tract infection and may be viral or bacterial. **A viral infection is short-lived (1 or 2 days)** and often accompanied by some general features of an upper respiratory infection, e.g. pharyngitis and a runny nose.

Acute otitis media (AOM)

- is an acute, suppurative infectious process marked by the presence of infected middle ear fluid and inflammation of the mucosa lining the middle ear space.(Eustachian tube, tympanic cavity mastoid antrum and air cells.)
- The infection is most frequently precipitated by **impaired function of the Eustachian tube**, resulting in the retention and suppuration of retained secretions.
- AOM may also be associated with **purulent otorrhea** if there is a ruptured tympanic membrane.
- AOM usually responds promptly to antimicrobial therapy



pathogenesis

The bacteria responsible for acute otitis media are:

- **Streptococcus pneumoniae 35%**
- **Haemophilus influenzae 25%**
- **Moraxella catarrhalis 15%**
- Group A streptococci and Staphylococcus aureus may also be responsible.

Causes of AOM :

- **More common**

1) Common cold
2) Acute tonsillitis
3) Influenza
4) Coryza of measles, scarlet fever ,whooping cough

- **Less common**

1) Sinusitis
2) Haemotympanum
3) Trauma to the tympanic membrane
4) Barotrauma (air flight)
5) Diving
6) Temporal bone fracture

The sequence of events in acute otitis media is as follows:

more in children → irratable (شكوى الام) ⇝ مجرد perforation & pus discharge ⇓ يهدأ الطفل √√

1) Organisms invade the **mucous membrane of ET** causing inflammation, congestions and oedema

- Oedema **closes** the Eustachian tube, preventing aeration and drainage and lead to **retraction** of drum duo to **–ve** pr

2) Oedema and congestions of **middle ear mucosa** lead to secretion of serous fluid

3) Accumulation of **pus** in M.E

-Pressure from the pus rises, causing the drum to **bulge** and **perforate** lead to **discharge**

bcz mcc is viral 80% infection → resolve spontanously √√

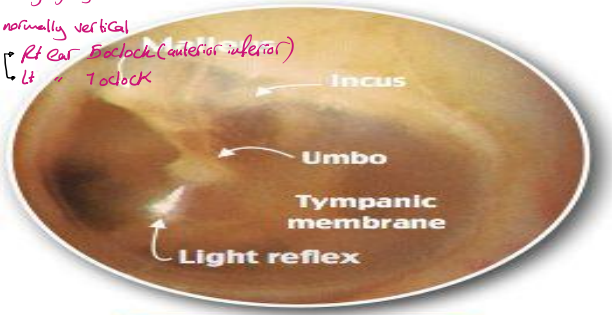
4) Most cases **resolve** completely. A small number cause complications or persistent perforation.

Sequences of acute otitis media

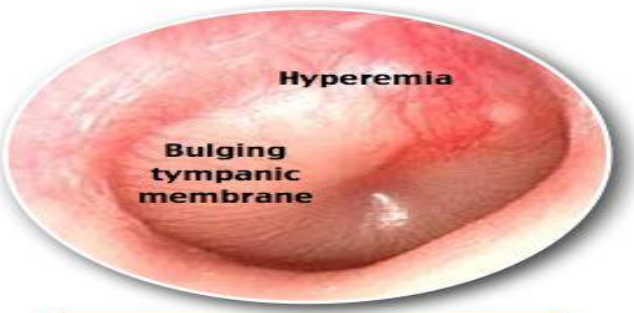
1. Perforation >>> rupture of tympanic membrane >> scar.
2. Inflammation and discharge >>> Spontaneous resolution (common and usually in children).
3. Transform into chronic form.
4. Transform into otitis media with effusion.
5. Resolves with antibiotics before discharge.
6. Pus and discharge.

Normal tympanic mem.:
① intact
② color → pearly gray or semitranslucent
③ handle of malleus → normally vertical
④ Cone of light → Rt ear 5o'clock (anterior inferior)
Lt ear 7 o'clock

Acute Otitis Media



Normal



Acute Otitis Media

The key difference between right and left tympanic membrane is that cone-shaped light reflection of the otoscope light is seen at the 4 o'clock to 5 o'clock position in the right tympanic membrane while cone-shaped light reflection of the otoscope light is seen at the 7 o'clock to 8 o'clock position in the left ...

Symptoms :

- **Deafness** :

is always present in acute otitis media but if the infection is unilateral this can go unnoticed. It is conductive in nature and may be accompanied by tinnitus. In an adult deafness or tinnitus may be the first complaint.

- **Earache (otalgia)** :

may be slight in a mild case, but more usually it is throbbing and severe. The child may cry and scream inconsolably until the ear perforates, the pain is relieved and peace is restored.

- **Discharge** :

Pressure builds up in the middle ear and the drum ruptures. The child gets immediate pain relief but the parents notice a sticky discharge, often purulent. The perforation formed in this way usually heals.

- **Signs**

1) **Pyrexia** :

The child is flushed and ill.

The temperature may be as high as 40 °C.

2) **Tenderness**:

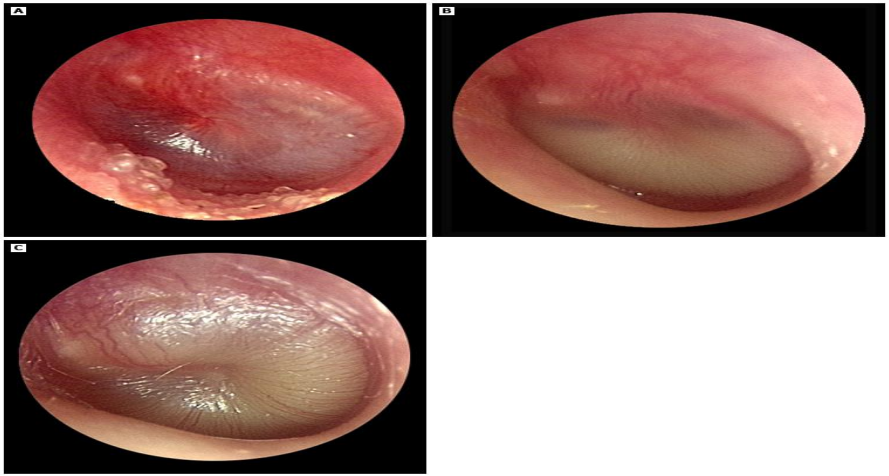
There is usually some tenderness to pressure on the mastoid bone.

3)Mucoid (sticky) discharge from an ear must mean that there is a perforation of the tympanic membrane. There are no mucous glands in the external canal.

Otoscopy and interpreting the findings can be difficult in a fractious child (نكد / مشاكس).

The tympanic membrane varies in appearance according to the stage of the infection:

1. **Loss** of lustre (بريق) and break-up of the light reflex.
2. **Congestion** of the small vessels around the periphery and along the handle of the malleus.
3. Redness and fullness of the drum; the malleus handle becomes **more vertical**.
4. **Bulging**, with loss of landmarks. Purple colour. Outer layer may **desquamate** (متقشر), causing blood-stained **serous** discharge. Early necrosis may be recognized, heralding imminent perforation.
5. **Perforation** with **otorrhoea**, which will often be blood-stained. Profuse and mucoid at first, later becoming thick and yellow



(A) Early acute otitis media with inflammation; subsequently progressed to effusion.
(B) Purulent effusion with air-fluid level.
(C) Bulging purulent effusion filling the middle ear.

Investigations

Usually **no investigation** is required.

Culture of discharge from ear may be indicated in chronic or recurrent perforation.

Audiometry should be performed if chronic hearing loss is suspected; however, not during acute infection.

So diagnosis simply by detect an active infection in the middle ear is to **look** in the child's ear with an **otoscope**, a light instrument that allows the physician to examine the outer ear and the eardrum.

Inflammation of the eardrum indicates infection.

Treatment

- The treatment depends on the stage reached by the infection.
- The following stages may be considered:

1) Early
2) bulging
3) discharging.

- Most cases of AOM improve spontaneously. 80% Viral ... أخف / spontaneous resolve = بشرية
- Cases that require treatment may be managed with antibiotics and analgesics or with observation alone.
- The recommendations offer more rigorous (صارم) diagnostic criteria to reduce unnecessary antibiotic use.

For early stage :

1. Antibiotics

- **Penicillin** remains the drug of choice in most cases, and ideally should be given initially by injection followed by oral medication.
- In children under 5 years, when *Haemophilus influenzae* is likely to be present, **amoxycillin** will be more effective, and should always be considered if there is not a rapid response to penicillin.
  - **Co-amoxiclav** is useful in *Moraxella* infections.
  - Be guided by sensitivity reports from the laboratory.

2. Analgesics

- Simple analgesics, such as aspirin or paracetamol, should suffice.
- Avoid the use of aspirin in children because of the risk of Reye's syndrome.

* Asperin in high analgesic doses not use neither in children nor in adults

3. Nasal vasoconstrictors

- The role of 0.5% ephedrine nasal drops is traditional but its value is uncertain in the presence of acute inflammation of the middle ear (**used when there is upper respiratory tract infection predisposing to otitis media**).

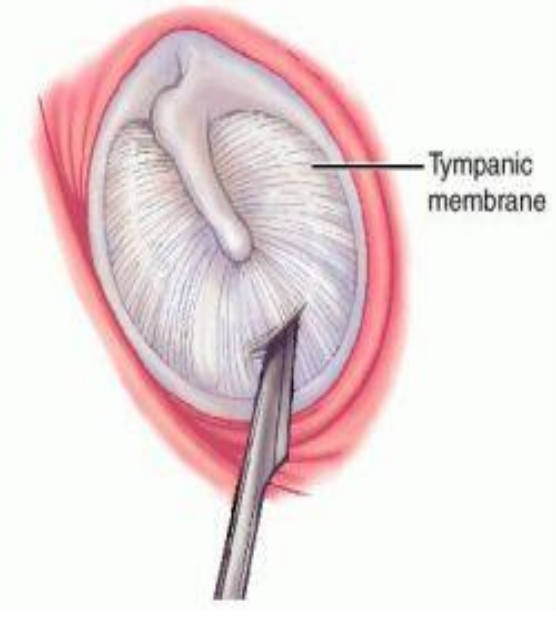
4. Ear drops

- Ear drops are of **no** value in acute otitis media with an intact drum. Especially illogical is the use of drops containing local anaesthetics, which can have **no** effect on the middle-ear mucosa yet may cause a sensitivity reaction in the meatal skin.

In bulging stage:

- **Myringotomy** is necessary when bulging of the tympanic membrane persists, despite adequate antibiotic.
- **Adequate antibiotic therapy:**
  - ✓ Choice of drug (according to the organism and antibiotic resistance).
  - ✓ Dose (according to body weight).
  - ✓ Compliance.
- It should be carried out under general anaesthesia in theatre and a large incision in the membrane should be made to allow the ear to drain. Pus should be sent for bacteriological assessment.
- Following myringotomy, the ear will discharge and the outer meatus should be dry-mopped regularly.

→ what do you mean by this?
① dose (according body wt)
② duration
③ Compliance
④ broad spictrum anti-biotic



In discharge :

- Discharging—nature's myringotomy ☺ ☺
- If the ear is already discharging when the patient is first seen, a swab should be sent for culture of the organism. Antibiotic therapy should be started but modified if necessary when the result of the sensitivities is known.
- Regular aural toilet will be necessary.

Further management Acute otitis media is not cured until the hearing and the appearance of the membrane have returned to normal. This can take several weeks and a persistent effusion of fluid in the middle ear is especially common in children.

If there is no resolution suspect:

1) the nose, sinuses or nasopharynx; infection may be present;
2) low-grade infection in the mastoid cells (mastoiditis).
3) the choice or dose of antibiotic

Recurrent Otitis Media

- Some children are susceptible to **repeated attacks of AOM**.
- There may be an underlying immunological deficit such as **IgA** deficiency or **hypogammaglobulinaemia** that will need to be investigated.
- Long-term treatment with half-dose **cotrimoxazole** (trimethoprim/sulfamethoxazole (combined Abx)) may be beneficial.
- If the attacks persist, **grommet insertion** **Tympanostomy tube** may prevent further attacks but may result in purulent discharge.

A grommet is a small ventilation tube inserted into the eardrum to allow air into the middle ear and prevent a build-up of fluid

Tympanostomy tube indication

1. people who have three or more episodes of acute otitis media in 6 months. (≥3 AOM in 6m)
2. or four or more in a year, with at least one episode or more attacks in the preceding 6 months (≥4 AOM in 1y)

complication :

- most common otorrhea

Grommet insertion

COMPLICATIONS ☹

Otologic complications :

- TM perforation,
- chronic suppurative OM, CSOM
- Cholesteatoma : an abnormal collection of skin cells deep inside your ear
- ossicular necrosis
- chronic otorrhoea

Intracranial complications

- meningitis,
- extradural abscess,
- brain abscess

Other complications include:

- mastoiditis
- labyrinthitis
- sigmoid sinus thrombophlebitis.

OTITIS MEDIA WITH EFFUSION

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INTRODUCTION

OM.E

- also known as AKA; Serous Otitis Media, Secretory Otitis Media, Mucoid Otitis Media, "Glue Ear"
- The condition is due to the accumulation of non-infected fluid within the middle-ear cleft; *(often the effusion is thick & viscid but sometimes it maybe thin & serous)* resulting in conductive deafness.
- It is commonest in small children (6 months- 3 years) and those of primary school age and may cause significant deafness.
- It occurs in adults, usually as a serous effusion and may rarely be a sign of nasopharyngeal malignancy. → very rare
- self-limited , takes 4-6 weeks to resolve

Pathogensesis

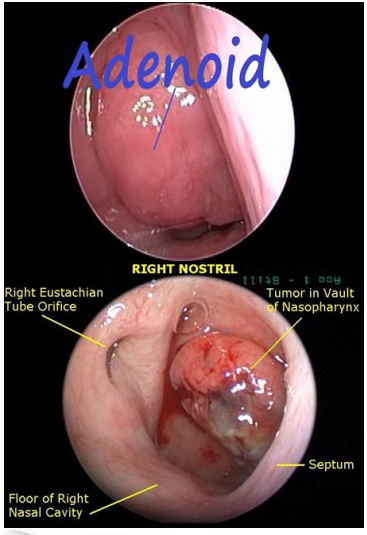
- Two main mechanisms are thought to be responsible:

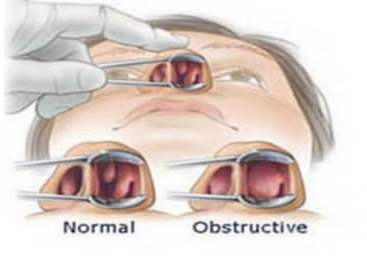
1. Malfunctioning of Eustachian tube → prevents adequate drainage → accumulation of fluid
2. Increased secretory activity of middle ear mucosa;

(Biopsies of middle ear mucosa in these cases have confirmed increase in number of mucus or serous-secreting cells.)

CAUSES OF OME

1. Nasopharyngeal obstruction, e.g. large adenoids(mc cause in children) or tumour resulting in Eustachian tube dysfunction.
2. untreated Acute otitis media, will often give rise to a spontaneous perforation and drainage of the middle ear.
3. Allergic rhinitis, often missed in children, will predispose to middle-ear effusions.
4. parental smoking has been shown to predispose to OME in children.
5. OME is commoner in winter months.
6. In many cases of secretory otitis media, no cause is apparent.





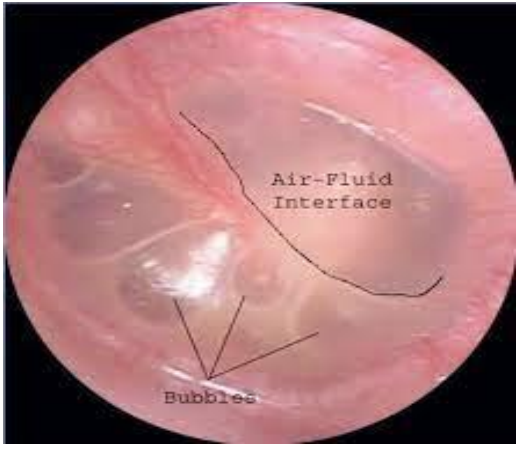
SYMPTOMS

- Not all children with OME have symptoms, painless
- Symptoms are often mild or minimal. They can vary based on the child's age.
- It may be responsible for developmental and educational impairment (delayed speech development) , and if untreated may result in permanent middle-ear changes.
- **Deafness** may be the only symptom.
- **Discomfort** in the ear —rarely severe.
- Occasionally, **tinnitus** or **unsteadiness**.

signs

1) **otoscope:**

- Intact drum and its colour is:
- *amber yellow (in serous)
- Or*dull gray(in mucoid)
- There may be fluid level(hair line)
- There may be air bubbles


Air-Fluid
Interface
Bubbles

signs

Retracted drum:

1. Prominent lateral process.
2. Shortened handle of malleus.
3. Disturbed or absent cone of light.
4. Exaggerated ant. and post. malleolar folds.
5. Limited mobility on siegalization.

Retracted TM

Normal TM

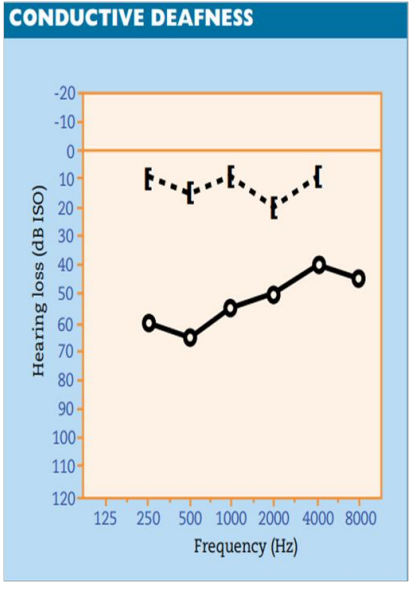
-Tuning fork tests show conductive deafness, i.e. bone conduction > air conduction.

Pneumatic otoscope

investigation

1)PTA(pure tone audiometry)

Conductive hearing loss

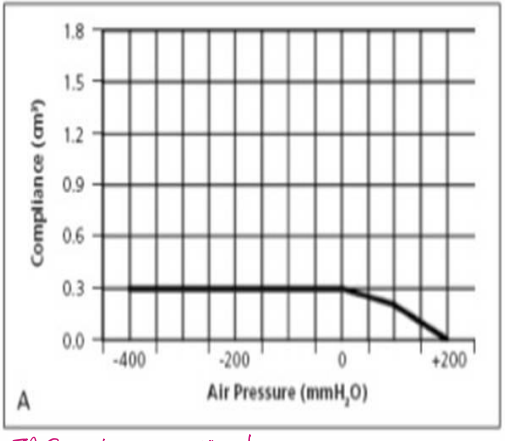


investigation

for children after diagnosed with otitis media with effusion

- 2- **Tympanometry:**

➢Flat, type B tympanogram.



EAC Volume → external Auditory Canal

Cont…

- **X-ray**
- **lateral view nasopharynx for adenoid**

Super imposed bacterial infection → causing pain ✱

TREATMENT

- In children :
- **1** Many cases will **resolve spontaneously**, and the child should usually be observed for 3 months before embarking on surgery.
- **2** The use of nasal anti-histamines , nasal mucolytics glucocorticoids is of no proven benefit.

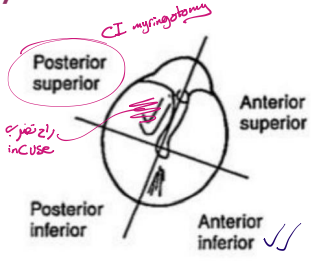
Antibiotic therapy may help in the short term.

Surgery is indicated if hearing loss persists for 3 months or if there is recurring pain.

- **3 Surgical treatment (in 10% of cases).**

Myringotomy and grommet insertion

- Under general anaesthetic, the tympanic membrane is incised antero-inferiorly
- -it's far away from the location of the ossicles
- - this part have the least pressure
- -healing rates thus the groomet can stay for longer duration



- The glue is aspirated and a grommet inserted into the incision.
- The function of the grommet is to ventilate the middle ear and not to drain the fluid.

Indications of grommet insertion

1. 3 or more episodes of AOM in 6 months
2. 4 or more attack in a year with at least one episode in the preceding 6 months
3. OME with conductive hearing loss persists for 3 month or if there is recurrent pain .

Complications of grommet insertion: Otorrhea (most common)& Permanent perforation

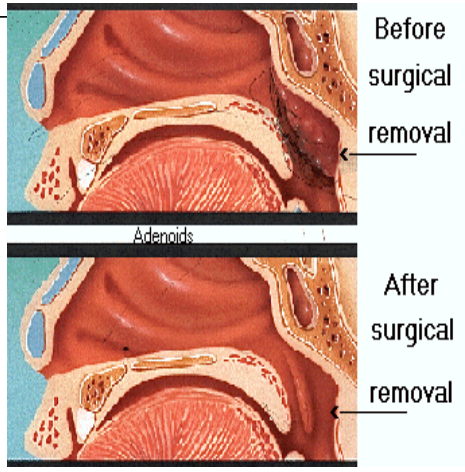
Complications of Myringotomy with Grommet insertion

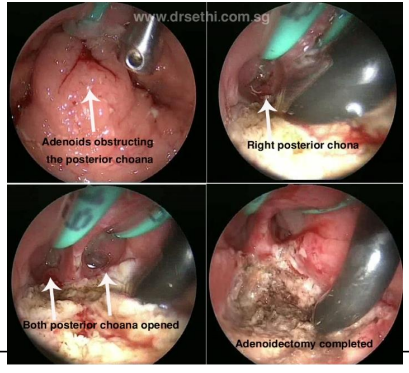
1. Infection.
2. Bleeding.
3. Permanent perforation.
4. Damage to the ossicles.
5. Damage to the facial nerve.

Adenoidectomy

*if hypertrophied بتعمل العملية

- It has been shown that adenoidectomy is beneficial in the long-term resolution of OME.
- The maximum benefit occurs between the ages of 4 and 8 years.





In adults

O.M.E

- Examination of the nasopharynx to **exclude tumour** is essential, especially if the effusion is unilateral.
- Under the same anaesthetic, a grommet may be inserted.
- Secretory otitis media in adults not due to tumour usually follows a cold. Resolution is usually spontaneous, but may take up to 6 weeks.

Complications and Long-Term Effects of OME

- If OME is associated with frequent ear infections, more complications can occur.
- These can include:
  - Acute ear infections .
  - Cysts in the middle ear .
  - Eardrum scarring .
  - Damage to the ear that causes hearing loss .
  - Affected speech or language delay . (Children)

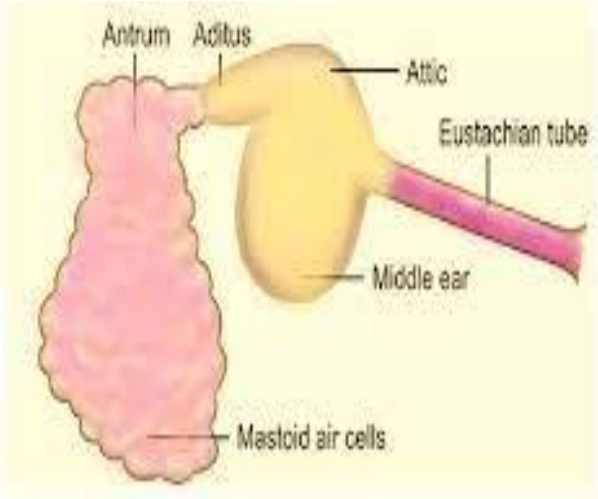
Chronic Otitis media

Introduction

- **Chronic Otitis Media (COM) :- it is an intractable pathology of middle ear cleft with permanent tympanic membrane defect of duration greater than 3 months.**

- **Tympanic membrane defects Includes :**

1. **Retraction pockets (Invagination of tympanic membrane into middle ear).**
2. **Atelectasis (Retraction of tympanic membrane medially).**
3. **Perforation.**



Risk factors

1. Late treatment of acute otitis media.
2. Inappropriate antibiotic.
3. Lowered resistance : malnutrition – anemia.
4. Ascending infections via eustachian tube from tonsils, adenoids, sinuses.
5. Poor socio-economic status.
6. Virulent infection ex . measles : infects Eustachian tube (makes it easy for bacteria to infect the ear).

CHRONIC SUPPURATIVE OTITIS MEDIA (CSOM)

Chronic otitis media with otorrhea for 6-12 weeks.

Pathophysiology :

CSOM is initiated by an episode of acute infection. Irritation and subsequent inflammation of the middle ear mucosa. The inflammatory response creates mucosal edema. Ongoing inflammation eventually leads to mucosal ulceration and consequent breakdown of the epithelial lining . ► The host's attempt at resolving the infection or inflammatory insult manifests as granulation tissue, which can develop into polyps within the middle ear space . ► The cycle of inflammation, ulceration, infection, and granulation tissue formation may continue, destroying surrounding bone margins.

2 types :

1.Tubotympanic.

2.Atticoantral.

1. Pseudomonas aeruginosa 48%-98% of cases.
2. S. aureus is the second most common.
3. Anaerobes make up 20-50% ; tend to be associated with cholesteatoma.
4. To 10% of infections are polymicrobial in etiology, often demonstrating a combination of gram-negative organisms and S. aureus.

Tubotympanic type

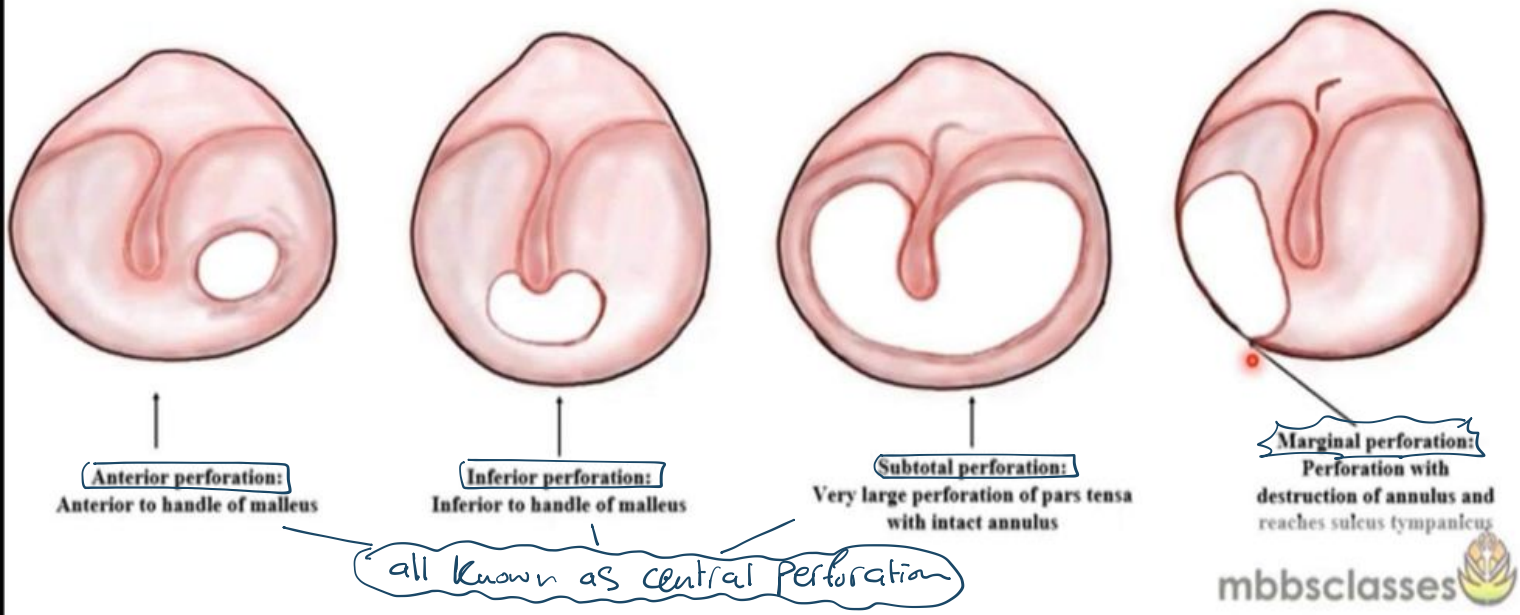
- Also called the safe or benign (mucosal) type ; it involves anteroinferior part of middle ear cleft (Eustachian tube) and is associated with a central perforation . It is less liable to produce complications.

- **Signs :**

1. Profuse (Secretions from ciliated columnar epithelium from Eustachian tube), mucopurulent odorless (Aerobic discharge).
2. Middle ear mucosa : Pale & dry (inactive) or Congested (active).
3. Granulations → polyps.
4. Central perforation.
5. Hearing loss : Conductive type of loss.

Types of perforations in COM without cholesteatoma

Types of perforations in COM without cholesteatoma



Diagnosis

1. **Otoscopic examination:**
   - Type of perforation (Anterior , Inferior , Subtotal , Marginal).
   - Middle ear mucosa (Pale & Dry or Congested).
2. **Microscopic examination:**
   - Status of ossicular chain.
   - Granulation tissue.
3. **Microbiology : culture and sensitivity of ear discharge.**
4. **Audiological tests:**
   - Tuning fork tests (Conductive hearing loss).
   - Pure tone audiometry (Conductive hearing loss).
   - Tympanometry (Type B).
5. **Radiological:**
   - Mastoid x-rays.
   - CT scan for temporal bone.

Management

❖ Medical treatment :

- Indicated in infected tympanic membrane perforation, basis is to eradicate infection and produce dry ear.

1. **Aural toilet** : by dry mopping or suction cleaning under microscopic visualization
2. **Topical antibiotics** (Neomycin).
3. **Systemic antibiotics** : needed in acute exacerbation of disease.
4. **General advice** : avoid swimming and entry of water into the ear.

❖ Surgical treatment :

- When there is a dry perforation, surgery may be considered but is not mandatory.

✔ **Myringoplasty** is the repair of a tympanic membrane perforation.

✔ Success rates for this procedure are very , high repair of the tympanic membrane may be combined with ossicular reconstruction, if necessary , in order to restore hearing— the operation is then referred to as a **tympanoplasty.**

Atticoantral (Bony) type

- It involves a posterosuperior part of middle ear cleft (attic, antrum, posterior tympanum and mastoid) and is associated with cholesteatoma, which, because of its bone eroding properties, causes the risk of serious complications. For this reason, the disease is also called unsafe or dangerous type.

- **Signs** :

1. The discharge is often scanty (squamous epithelium secretes less than ciliated columnar) but usually persistent, and is often foul smelling (Boney part involvement).
2. Osteitis and granulation tissue : A fleshy red polypus may be seen filling the meatus.
3. Cholesterol granuloma : It is a mass of granulation tissue with foreign body giant cells surrounding the cholesterol crystals.
4. Cholesteatoma.
5. Ossicular necrosis : It is common in Atticoantral disease. Destruction may be limited to the long process of incus or may also involve stapes superstructure, handle of the malleus or the entire ossicular chain. Therefore , hearing loss is always greater than in disease of tubotympanic type.

- **Complications** :

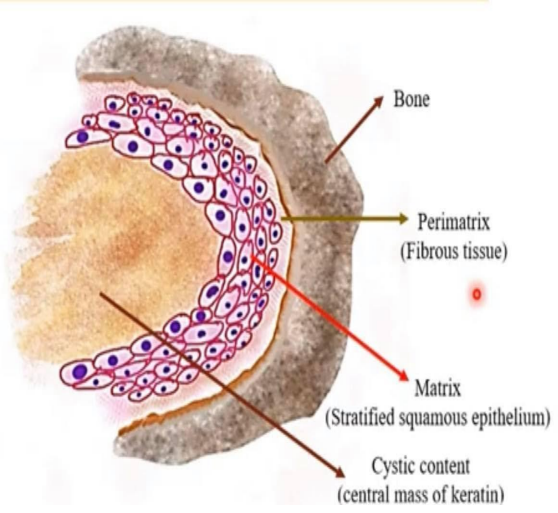
- Intracranial : Extradural abscess , Brain abscess , Meningitis.
- Infratemporal : Mastoiditis , Facial paralysis.

Cholesteatoma

- Is a destructive and expanding growth consisting of keratinizing squamous epithelium in the middle ear and/or mastoid process.
- It's considered lethal if not treated.

- **Histology (from Inner to Outer) :**

1. Central mass of keratin.
2. Matrix - stratified squamous epithelium.
3. Peri matrix - fibrous tissue.
4. Bone part.



Interaction of cholesteatoma with bone

✔ Results in bone erosions of ossicular chain (Mostly Long process of incus and stapes superstructure) and bone wall of middle ear and mastoid.

Other structures involved :

1. Bone covering lateral semi-circular canal (leading to Vertigo).
2. Facial Nerve (leading to facial paralysis).
3. Bone over dura of middle and posterior cranial fossa (leading to meningitis , brain abscess).

- Mechanism :

✔ Physical pressure.

✔ Activation of osteoclasts.

✔ Release of inflammatory mediators such as cytokine interleukin from macrophages and epidermal keratinocytes.

✔ Other factors associated : prostaglandins, cathepsin D.

Cholesteatoma Types

Congenital Cholesteatomas

- ✔ Inclusion cyst in the anterosuperior surface quadrant of middle ear near the Eustachian Tube.
- ✔ Diagnosed by otoscope which reveals white mass behind an intact tympanic membrane with no history of otitis media.

Acquired Cholesteatoma

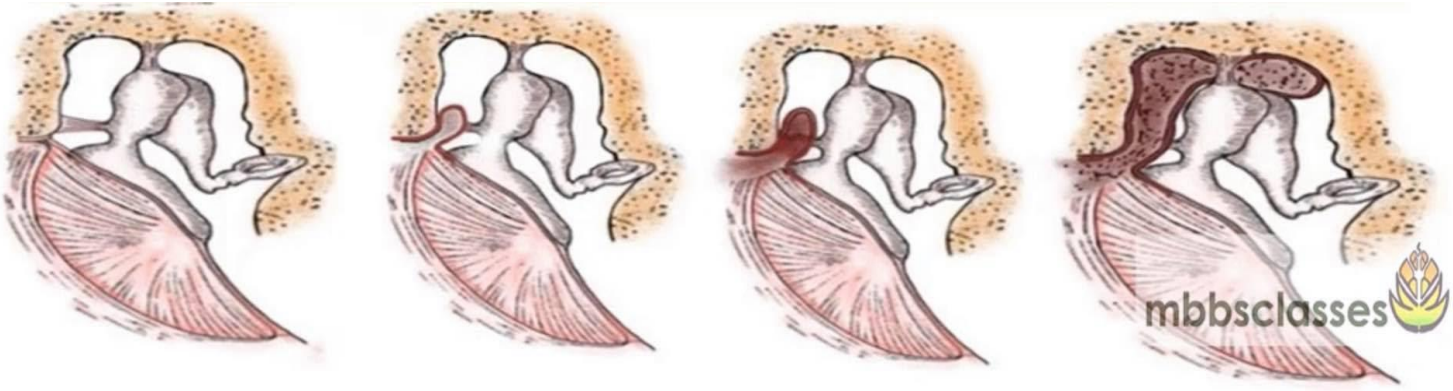
Primary acquired Cholesteatoma

Typical retraction pocket cholesteatoma adjacent to posterosuperior tympanic membrane or pars flaccida and in the center contains keratin (Invagination theory).

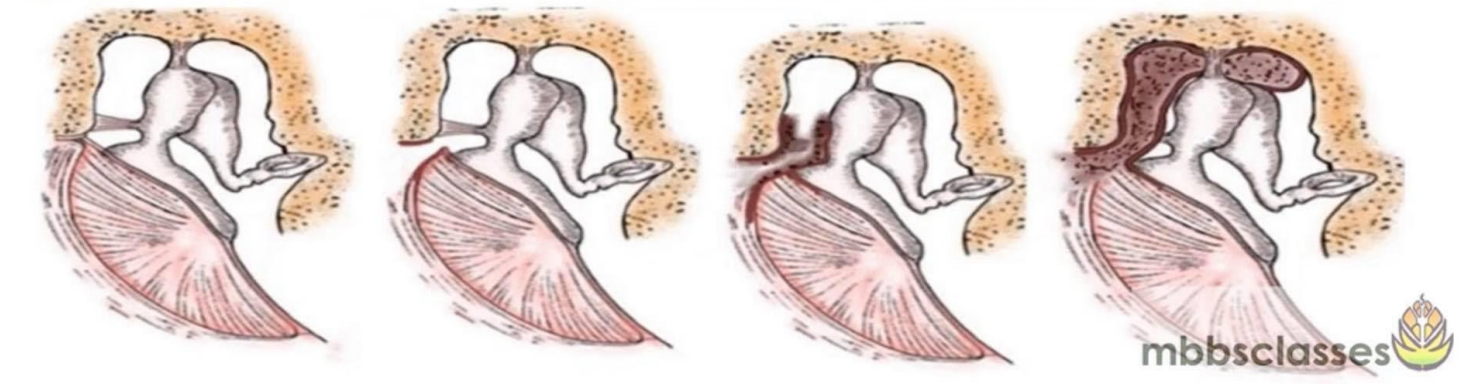
Secondary acquired Cholesteatoma

As the tympanic membrane tries to heal, keratinizing squamous epithelium migrate through the perforation into the middle ear and makes the cholesteatoma (Migration theory).

Invagination theory



Migration theory



Classification of Cholesteatoma based on anatomical sites

1. **Pars tensa cholesteatoma** : results from retraction pocket or perforation in posterosuperior quadrant of pars tensa.
2. **Pars flaccida cholesteatoma** : results from retraction pocket or perforation in pars flaccida.
3. **Occult Cholesteatoma** : often congenital.

- **Pathogenesis of Acquired Cholesteatoma :**

- Invagination of ~~temporal~~ tympanic membrane (retraction pocket Cholesteatoma).
- Basal cell hyperplasia.
- Epithelial ingrowth through perforation (Migration theory).
- Squamous metaplasia (unknown cause).

Clinical presentation

✔ Patient usually have either history of otitis media or ventilation tube or had surgery on the tympanic membrane.

- **Signs and symptoms :**

1. Progressive hearing loss (Conductive hearing loss).
2. In late stages (Sensorineural hearing loss).
3. Otorrhea :

- Purulent or blood stained.
- Foul smelling due to infection with anaerobic bacteria.
- Blood stained due to osteitis and granulation tissue.

4. Otalgia. (not always)
5. Aural fullness.
6. Symptoms of complications : Vertigo, Facial paralysis or intracranial infection.

Approach to patient

1. Otoscopic examination (for Diagnosis) : suction cleaning and canal debridement for better visualization.
2. Microscope examination (For Diagnosis) : better visualization of perforation, retraction pocket, Cholesteatoma and granulation tissue.
3. Radiological tests (For Management) : High resolution CT scan , MRI.
4. Audiological tests : To document preoperative hearing loss.
5. Pneumatic otoscopy : In patients of COM with vestibular symptoms.
6. Positive fistula tests characterized by vertigo and nystagmus suggests erosion of lateral semi circular canal by cholesteatoma.

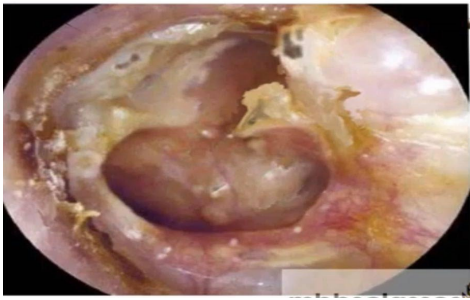
Otoscopic findings of Cholesteatoma

Primary acquired cholesteatoma :

- ✔ Typical attic retraction cholesteatoma appears as pearl white defect of variable size adjacent to posterosuperior portion of tympanic membrane.
- ✔ Contains keratin debris.

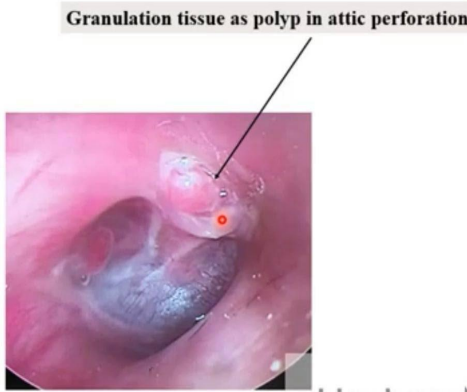
Secondary acquired cholesteatoma :

- ✔ It is found next to TM perforation.
- ✔ Center of the defect contains keratin debris.
- ✔ Keratinized epithelium migrates through a perforation into middle ear.



Aural polyp :

- ✔ Infected cholesteatoma may manifest as an aural polyp.
- ✔ Polyps in chronically infected ear should be considered as cholesteatoma until proved otherwise.
- ✔ Polyps are granulation tissue at the junction of eroding cholesteatoma and bone.



Complications

Extra cranial :

1. Acute mastoiditis.
2. Sub periosteal abscess.
3. Post auricular, Zygomatic, Bezolds , citelli's abscess.

deep neck abscess (deep to SCM muscle)

extratemporal complication of otitis media. It occurs when pus from the mastoid tip trickles down along the posterior belly of the digastric muscle to the occipital and cervical region.

4. Labrynthitis.
5. Facial nerve paralysis.
6. Petrositis and Gradenigo syndrome

infection of petrous part of temporal bone

a clinical triad of otitis media, facial pain and abducens palsy that is most commonly developed from infection in the petrous temporal bone (the full triad of GS may not always be presente)

7. Septicemia.

Intra Cranial :

1. Sub Dural abscess.
2. Meningitis.
3. Peri sinus abscess.
4. Sigmoid sinus thrombosis.
5. Otitis encephalitis.
6. Brain abscess :
   -temporal lobe abscess , cerebellar abscess.

Management of COM with cholesteatoma

- **Principle aim of the treatment :**

✔ **To remove the disease and eliminate the major risk of complications to make the ear safe and if possible dry.**

✔ **Once ear becomes safe , additional objective is to restore the hearing if it is compromised by cholesteatoma.**

 The effective treatment of cholesteatoma requires a surgical intervention.

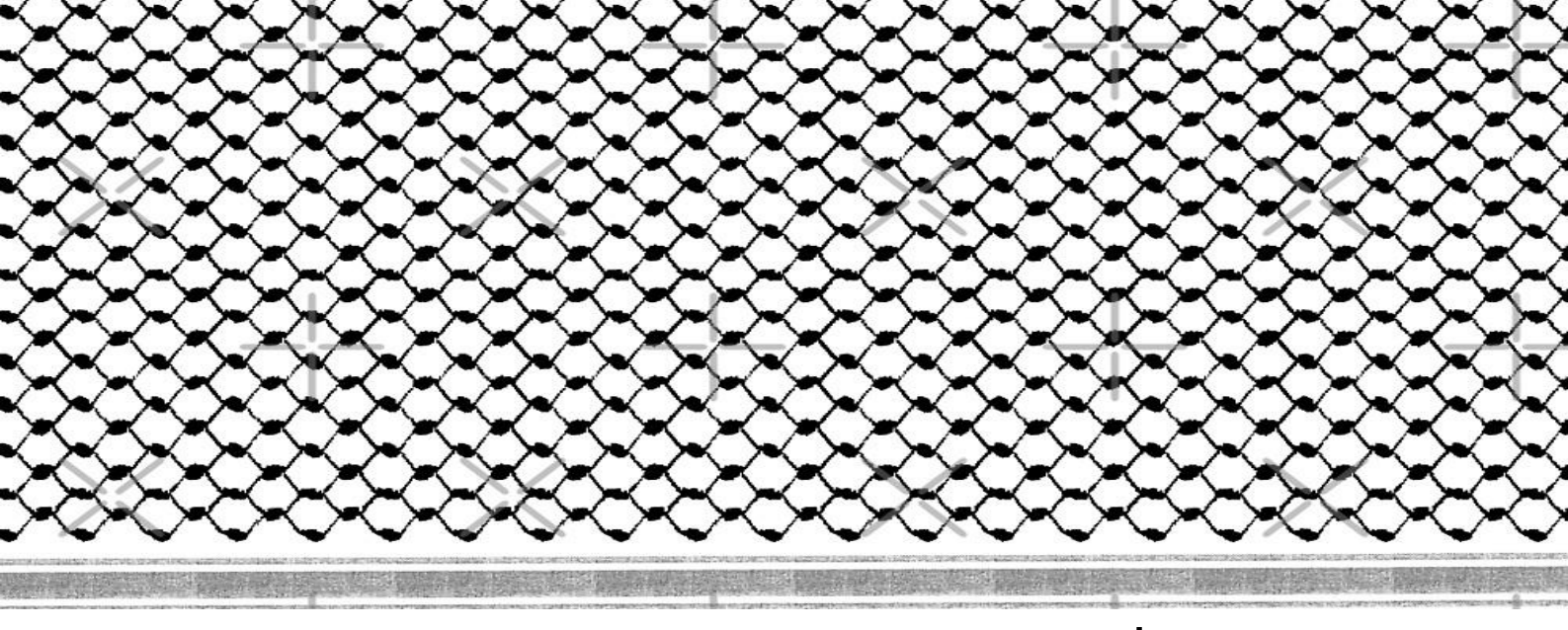
- **Treatment :**

1. **Regular aural toilet.**
2. **Suction toilet : under microscope, we evacuate pocket of cholesteatoma.**
3. **Mastoidectomy : it is always necessary.**
4. **Antibiotics if there is infection.**

Mastoidectomy

1. Simple Mastoidectomy.
2. Radical Mastoidectomy.
3. Modified Radical Mastoidectomy.
4. Mastoidectomy with tympanoplasty.

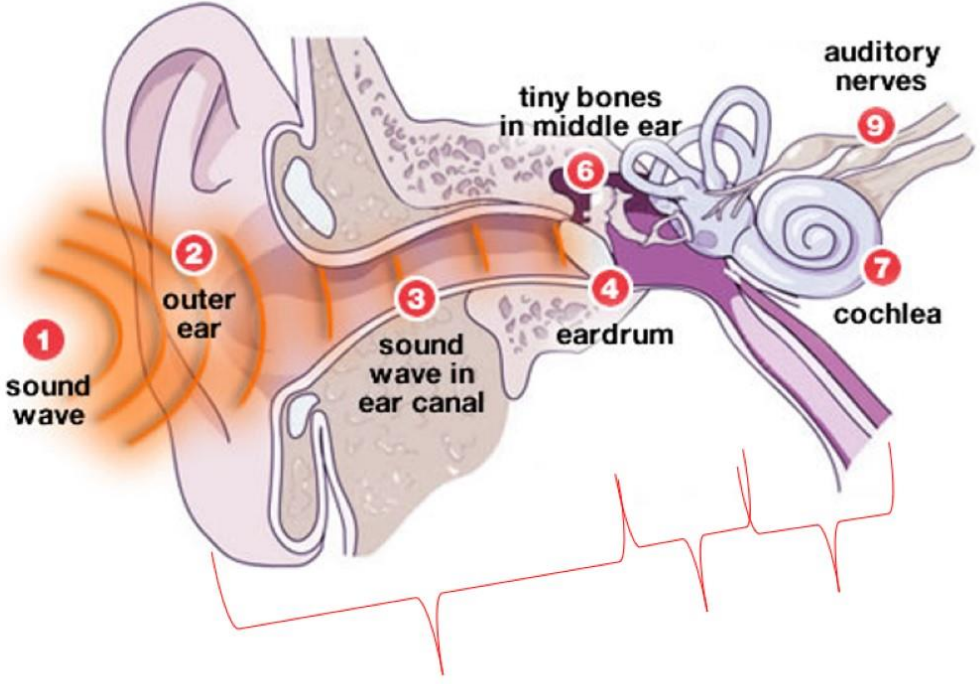
✔ In case of Eustachian Tube dysfunction , it leads to high failure rate, so we use antihistamine and mucolytics to hopefully to restore eustachian tube function but it is controversial.



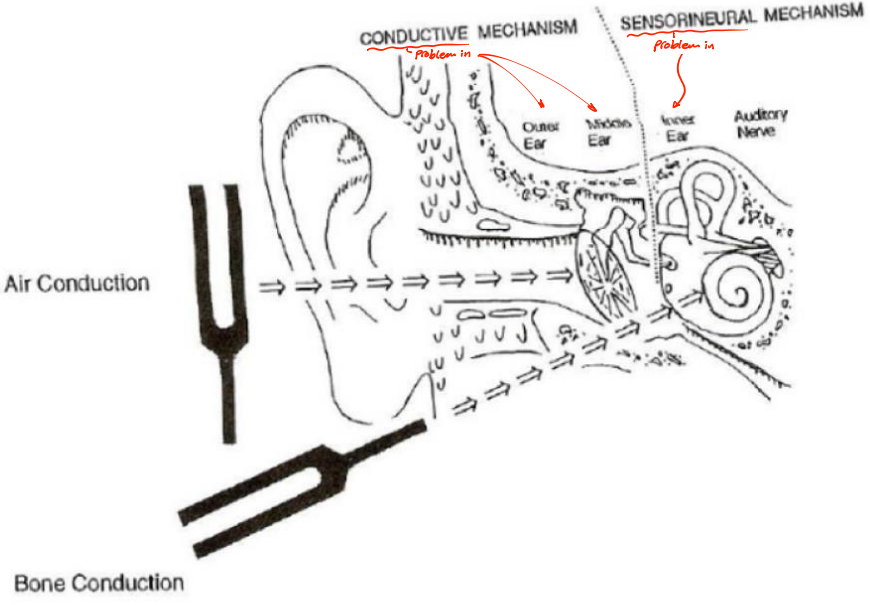
Assessment of Hearing

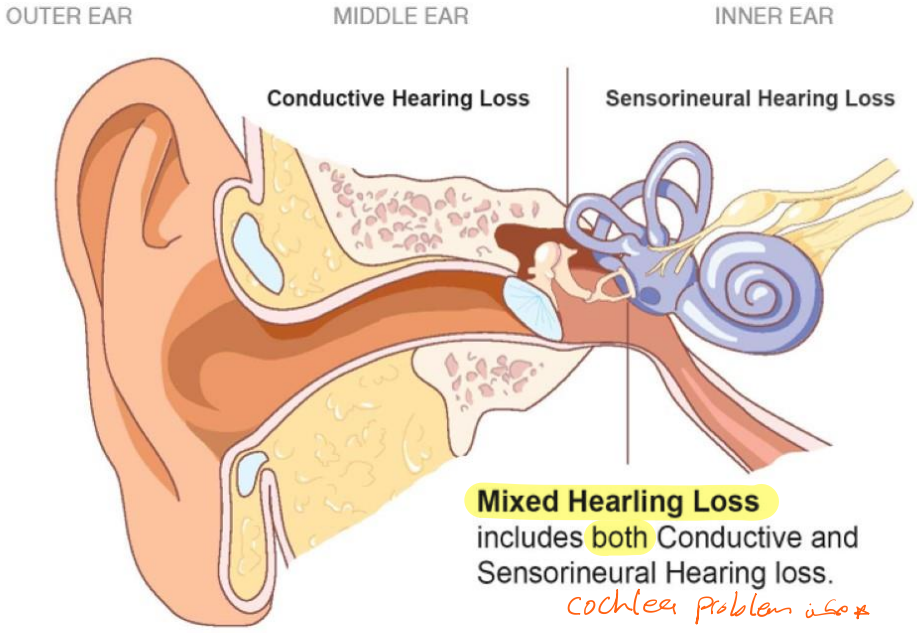
Yazeed Al-Hamaideh
Sara Al-Jaafreh
Rama Al-Shamaileh

Physiology Of Hearing



Types Of Hearing Loss




OUTER EAR
MIDDLE EAR
INNER EAR
Conductive Hearing Loss
Sensorineural Hearing Loss
Mixed Hearling Loss
includes both Conductive and
Sensorineural Hearing loss.
Cochlea problem also

Clinical Assessment Of Hearing

- **Whispered Voice Test**
- **Tuning Fork Tests**
  - ① - Rinne's Test
  - ② - Weber's Test
- **Audiometry**
  - 1 - Pure Tone Audiometer (PTA)
  - 2 - Speech audiometer
  - 3 - Impedance Audiometer
  - 4 - Electric response audiometer

Whispered Voice Test

- Formal Assessment. Ask the patient to repeat words spoken by the examiner at different intensities & distances.
- Examination sequence
  - Stand behind the patient. → عشان صايقرأ شفاهه
  - Start with your mouth about 15 cm from the ear you are testing.
  - Mask hearing in the other ear by rubbing the tragus.
  - Ask the patient to repeat your words. Use a combination of multisyllable numbers and words (1,2,3 or A,B,C). Start with a normal speaking voice to confirm that the patient understands the test. Lower your voice intensity to a clear whisper.
  - Repeat, but this time at arm's length from the patient's ear. People with normal hearing can repeat words whispered at 60 cm.

Interpretation of Results...

Normal

- Patient is able to hear whispered speech accurately.
- Volume is the same in both ears.

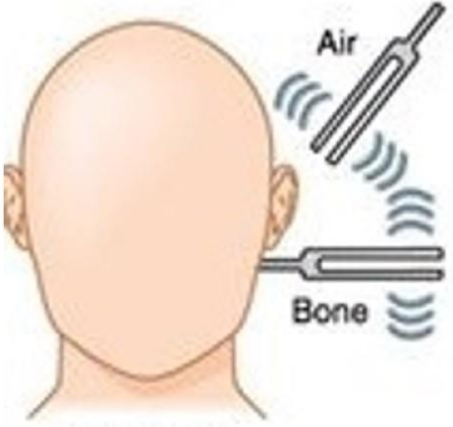
Abnormal

- The patient is unable to hear whispered speech.
- The patient hears speech at a higher volume in one ear.
- The patient hears sounds but does not understand words.

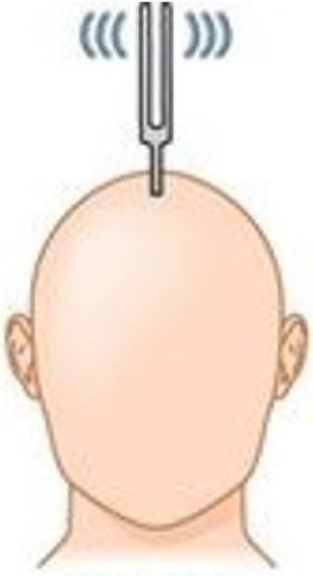
Tuning Fork Tests

- using either a 512 Hz or a 256 Hz tuning fork.

Usually ⅄



Rinne test



Weber test

paracusis willisii definition

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The ability to hear better in noise. The patient experiences better understanding of speech in a noisy environment. This phenomenon is characteristic of all patients with conductive hearing loss; it occurs because people speak louder in noisy surroundings.

+ve in $<^{N}_{SNHL}$

	Rinne's	Weber's
normal	positive	centralized
Conductive	Negative bone > Air	Lateralized to affected ear
sensorineural	positive	Lateralized to the unaffected ear
Profound sensorineural	False Negative	

'false' - the patient has a severe or total sensorineural deficit, but can hear the tone in the "good" ear due to the transfer of sound through the skull base to the functioning cochlear on the other side.

(PSHL) is a specific form of sensorineural hearing loss (SNHL) caused by congenital or acquired lesions of the cochlea, auditory nerve and auditory center.

a person with a profound hearing loss will not hear any speech and only very loud sounds.

pt has a dead ear

Rinne's Test

- Compares the level of air and bone conduction of the same ear.

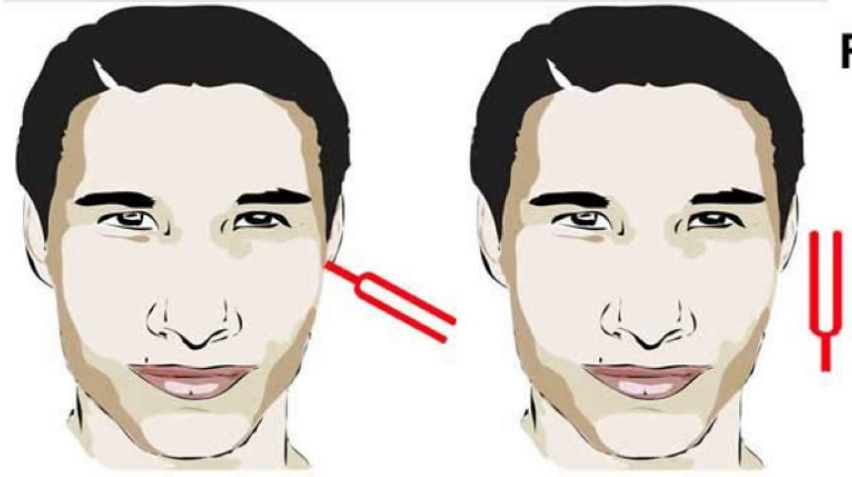
Examination Sequence

- Place the vibrating prongs at the patient's external auditory meatus; ask if he can hear it.
- Now place the still-vibrating base on the mastoid process. Ask: 'Is it louder in front, or behind your ear?'

Alternatively:

- The Rinne's test is performed by placing a vibrating tuning fork (512 or 256 Hz) initially on the mastoid process until sound is no longer heard, the fork is then immediately placed just outside the ear.

Rinne's Test



Rinne's Test

With a 512 Hz tuning fork press against the mastoid bone and then hold it 1cm away from the ear.

'Which is louder, behind the ear or in front?'

Additional Note

- If hearing in one ear is extremely poor there may be a false negative Rinne's.
  - The sound will be conducted through the bone to the opposite ear and give a false impression of better BC. Bone Conduction

Weber's Test

Examination Sequence:

- Place the base of the vibrating tuning fork in the middle of the patient's forehead.
- Ask: 'Where do you hear the sound?'
- Record which side Weber's test lateralizes to if not central.

Finding	Interpretation
No Lateralization (The sound is heard equally in both ears)	Normal/Healthy Patient
	Bilateral symmetrical Conductive Deafness
	Bilateral symmetrical Sensorineural Deafness
Lateralized to better/healthy ear	Unilateral Sensorineural Deafness
Lateralized to worse/affected ear	Unilateral Conductive Deafness

Quiz Tiiime

- Rinne's Rt(-) Lt(+) Weber's lateralizes to Rt → Rt CHL
- Rinne's Rt(+) Lt(+) Weber's lateralizes to Lt → ~~Lt~~ Rt SNHL (better ear)
- Rinne's Rt(+) Lt(+) Weber's is centralized → N or bilateral symmetrical SNHL
- Rinne's Rt(-) Lt(+) Weber's lateralizes to Lt → false -ve rinne ⇒ Dx: Rt profound SNHL

〃 bilateral (+ve) 〃 〃 〃 Rt → Lt SNHL

Clinical Assessment Of Hearing

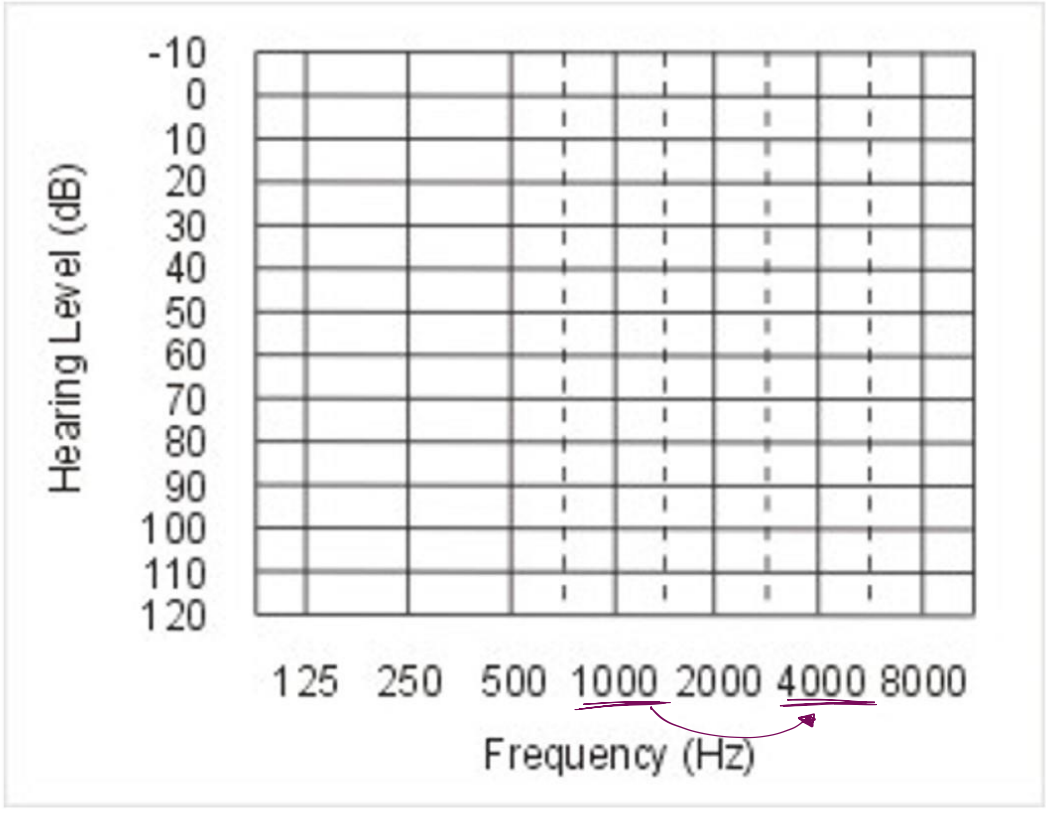
- **Whispered Voice Test**
- **Tuning Fork Tests**
  - **Rinne's Test**
  - **Weber's Test**
- **Audiometry**
  - **Pure Tone Audiometer**
  - **Speech audiometer**
  - **Impedance Audiometer**
  - **Electric response audiometer**

Pure Tone Audiometry

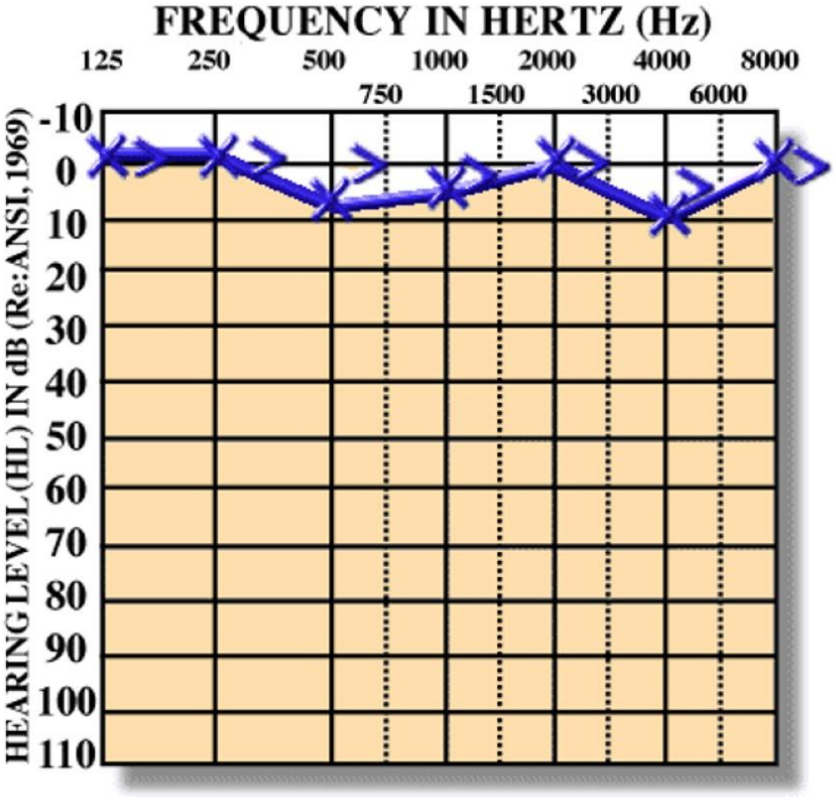
- Electronic device that generates tones for determining hearing thresholds. Displayed on a graphic plot called audiogram.

Audiogram

- The ears are most sensitive to frequencies in the range of 1000-4000Hz.
- range of human speech >(400-3000 Hz)
- The hearing threshold **is the sound level below which a person's ear is unable to detect any sound.** For adults, 0 dB is the reference level.

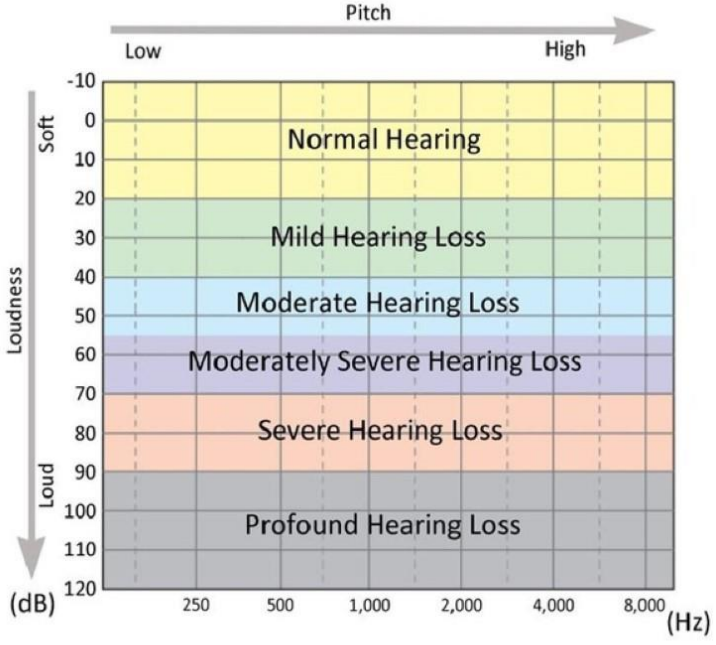


Normal Hearing



Legend	Right	Left
Air Conduction	O	X
•with masking	△	□
Bone Conduction	<	>
•with masking	⊏	⊐
No Response	↖	↗

Ranges of Hearing Loss

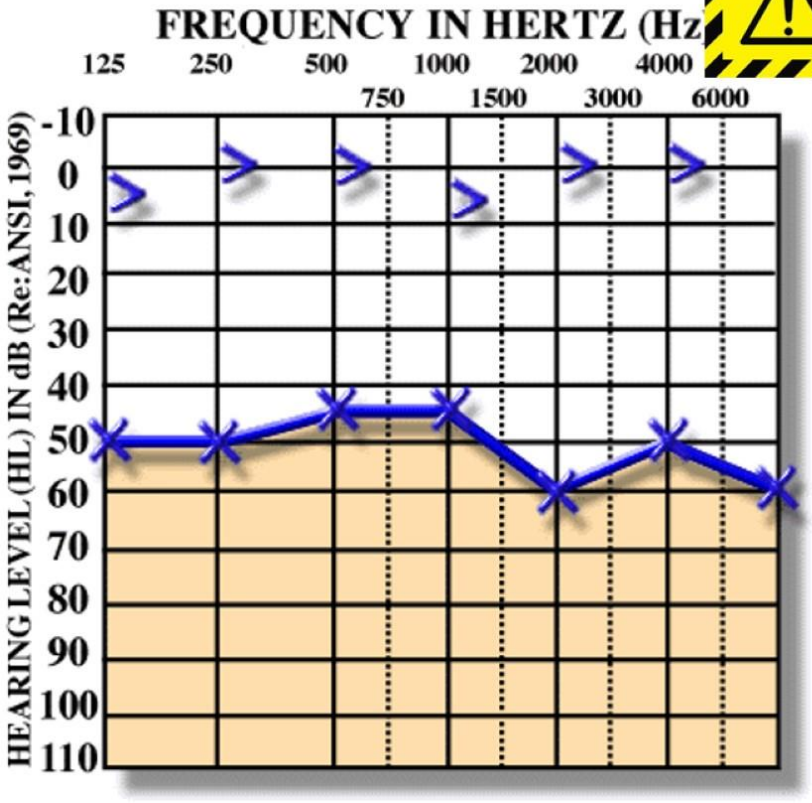


- **-10 – 20 dB HL = Normal range**
- **21 – 40 dB HL = Mild hearing loss**
- **41 – 55 dB HL = Moderate**
- **56 – 70 dB HL = Moderately Severe**
- **71 – 90 dB HL= Severe**
- **Greater than 90 dB HL = Profound**

Conductive Hearing Loss

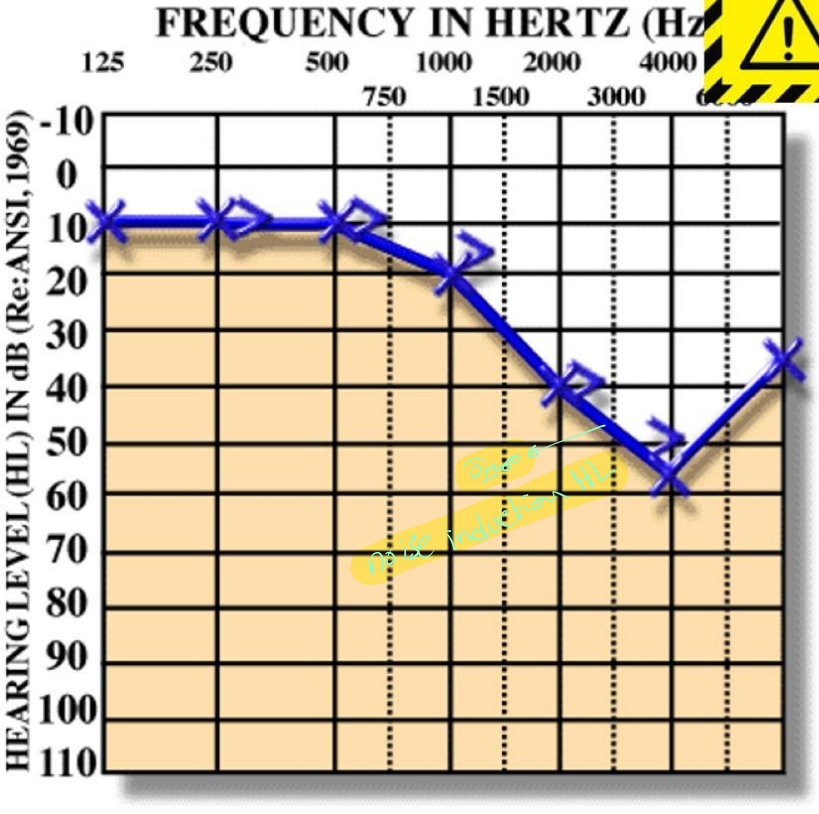
- AC worse than BC
- AC is below normal with at least 10 dB
- outer ear or middle ear abnormality;

1. Occlusion of the external auditory canal by cerumen or a mass
2. Middle ear infection and/or fluid
3. Perforation of the tympanic membrane
4. Ossicular abnormalities



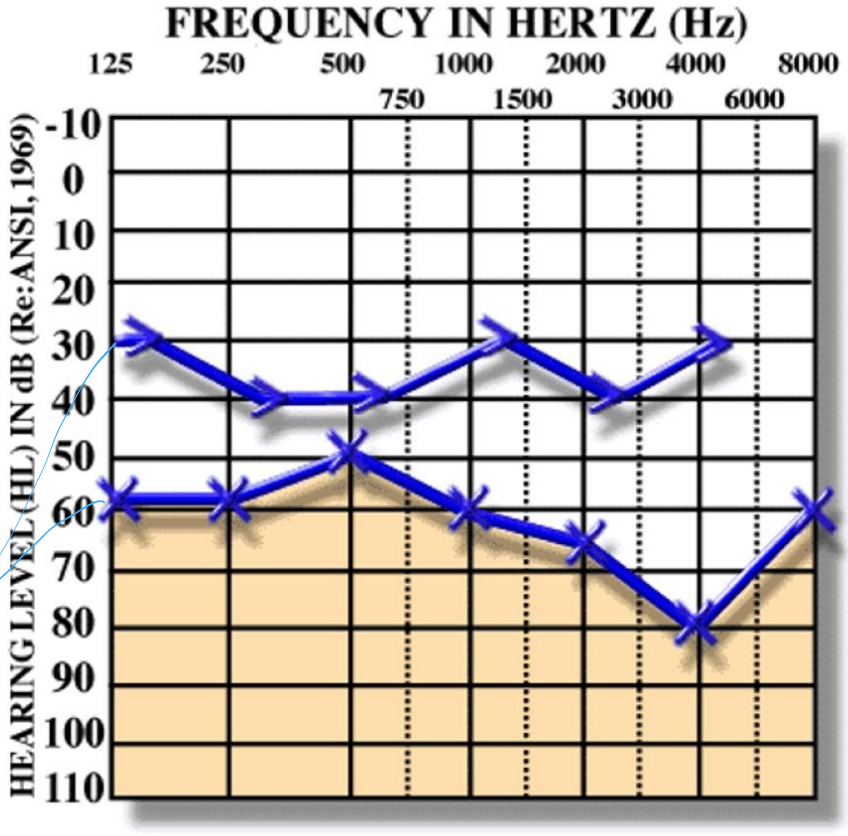
Sensorineural Hearing Loss

- No air-bone gap = normal or SNHL
- and thresholds are higher than 25 dB HL.
- cochlear abnormalities and/or an abnormality of the auditory nerve or central auditory pathways.



Mixed Hearing Loss

- Pure-tone air-conduction thresholds are poorer than bone-conduction thresholds by more than 10 dB.
- Bone-conduction thresholds are higher than 25 dB.



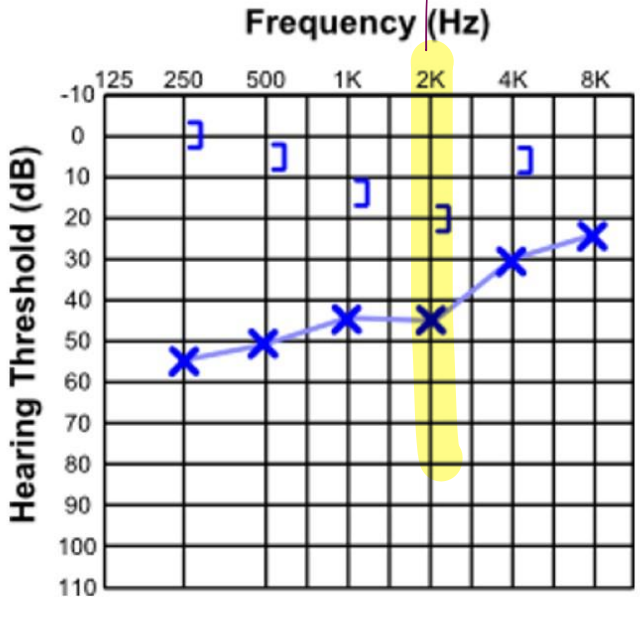
❑**Conductive hearing loss**

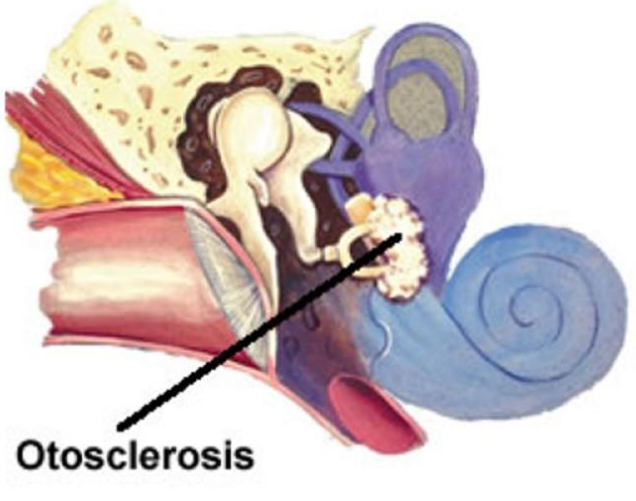
- Otosclerosis

❑**Sensorineural Hearing Loss**

- Examples include:
  - Noise-induced hearing loss
  - Presbycusis (age related)
  - Ménière disease
  - Retrocochlear lesions such as vestibular schwannoma

Otosclerosis

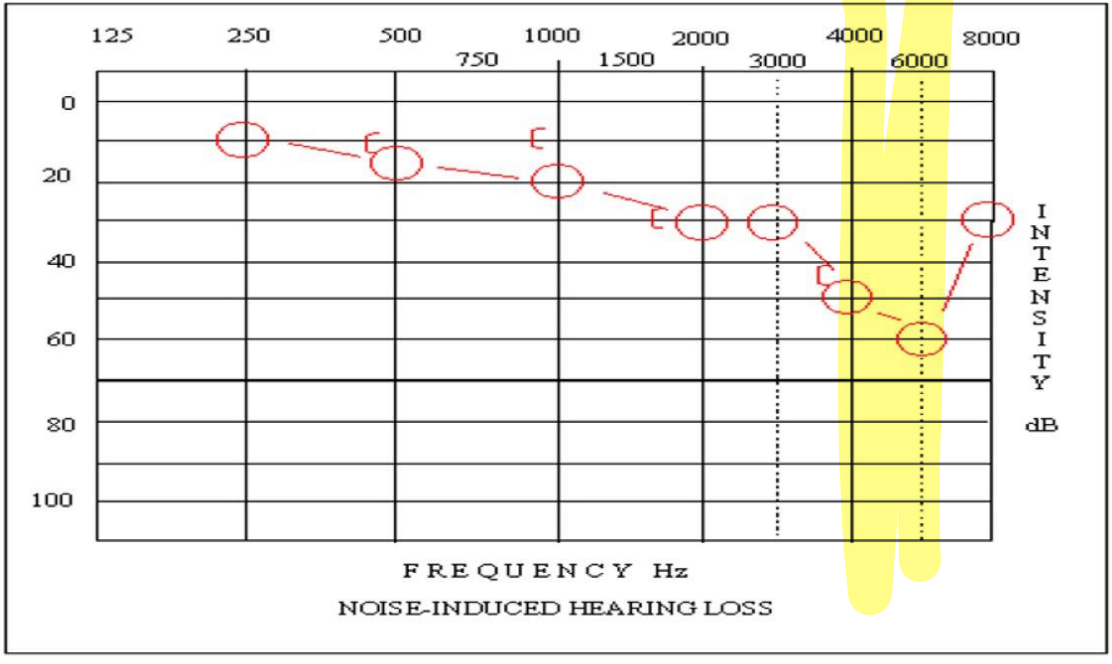




Otosclerosis

- caused by stapedial fixation in the oval window, stiffening the middle ear system.
  - ✓ **Stapes fixation produces an audiometric artifact known as the Carhart notch, (Carhart notch :Isolated depression(20-30 Db) around 2000 Hz in the bone-conduction audiogram of patients with otosclerosis.)**
  - ✓ Onset is 15-45 years, more common in women.
  - ✓ One half of patients report a family history of otosclerosis.

Noise-induced Hearing Loss

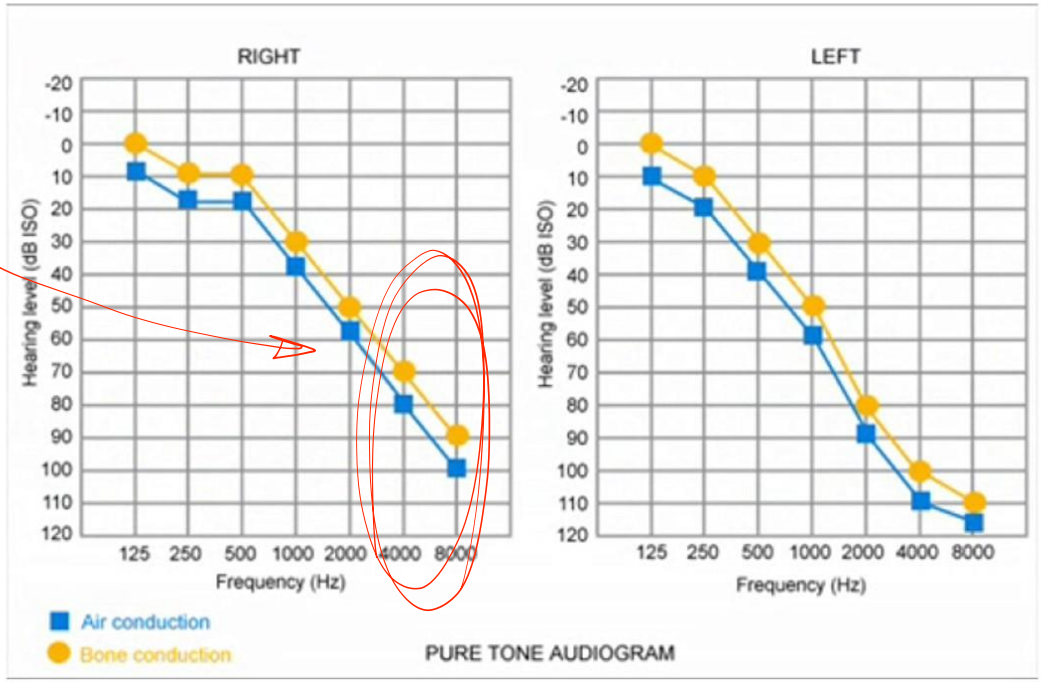


Noise-induced Hearing Loss

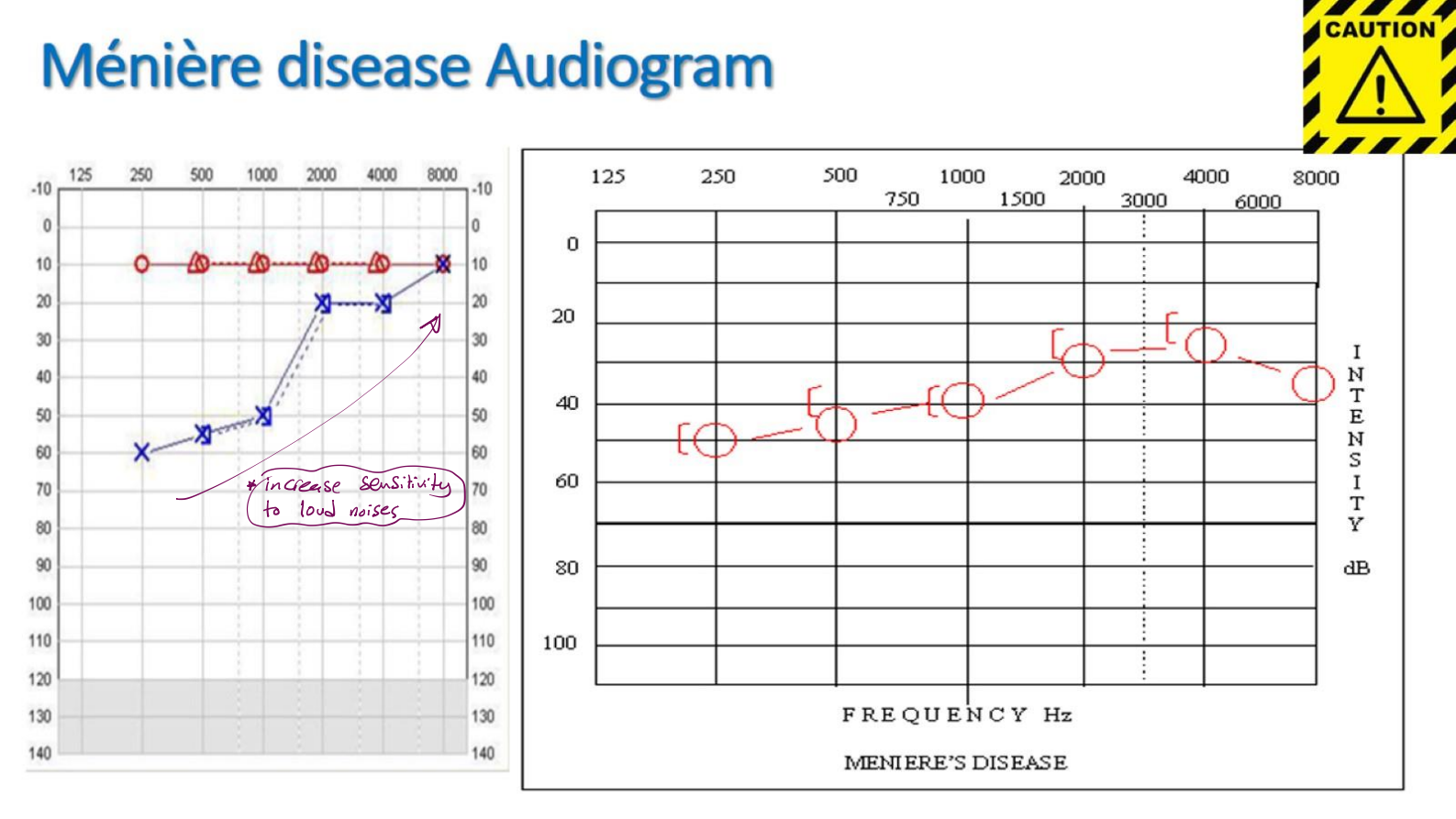
- Exposure to high-intensity noise may cause temporary or permanent hearing loss.
- Noise-induced hearing loss is typically greatest in the 4000- to 6000-Hz region.
- Noise-induced hearing loss is sensorineural except in certain blast injuries with possible tympanic membrane and middle ear damage.

Presbyacusis (Age related)

- bilateral and symmetrical sensorineural hearing loss.
- the higher frequencies are most severely affected.
- Hearing loss is secondary to degeneration of the cochlea, cranial nerve VIII, and/or the central auditory system.



Ménière disease Audiogram



Ménière disease

- It is idiopathic disease ,but it is believed to be linked to **endolymphatic hydrops, an excess of fluid in the inner ear**. include some combination of **vertigo, hearing loss, and tinnitus.**
- Hearing loss is usually **unilateral**, at least in the early stages,
- Many patients report increased sensitivity to loud noises (recruitment) in addition to the listed symptoms.
- Onset for approximately one half of patients occurs when aged 40-60 years. **The disease is rare in children.**

Speech audiometry

Two parameters are studied :

- Speech recognition (إدراك) threshold
- Speech awareness

speech-recognition threshold (SRT)

The speech-recognition threshold (SRT) is sometimes referred to as the speech-reception threshold.

The objective of this measure is to obtain the lowest level at which speech can be identified at least half the time.

Speech-awareness threshold (SAT)

- Speech-awareness threshold (SAT) is also known as speech-detection threshold (SDT).
- The objective of this measurement is to obtain the lowest level at which speech can be detected at least half the time.

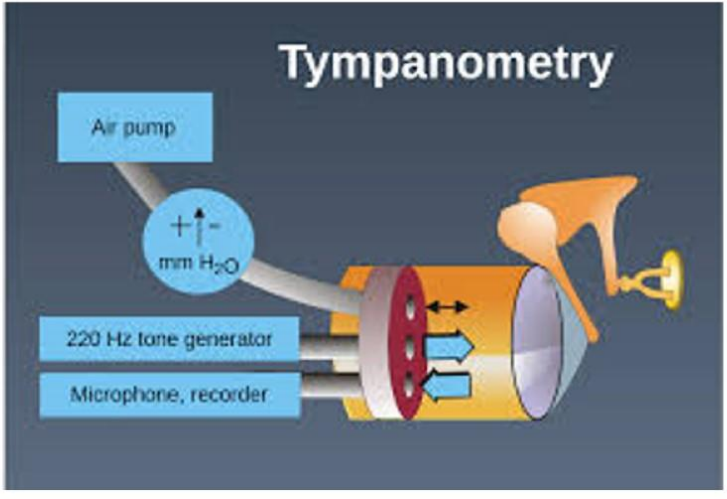
  - The SAT is especially useful for patients too young to understand or repeat words.

  - The SAT may also be used for patients who (1) speak another language or who (2) have impaired language function because of neurological insult.

  - For patients with normal hearing or somewhat flat hearing loss, this measure is usually 10-15 dB better than the speech-recognition threshold (SRT) that requires patients to repeat presented words.

Impedance audiometry (Tympanogram)

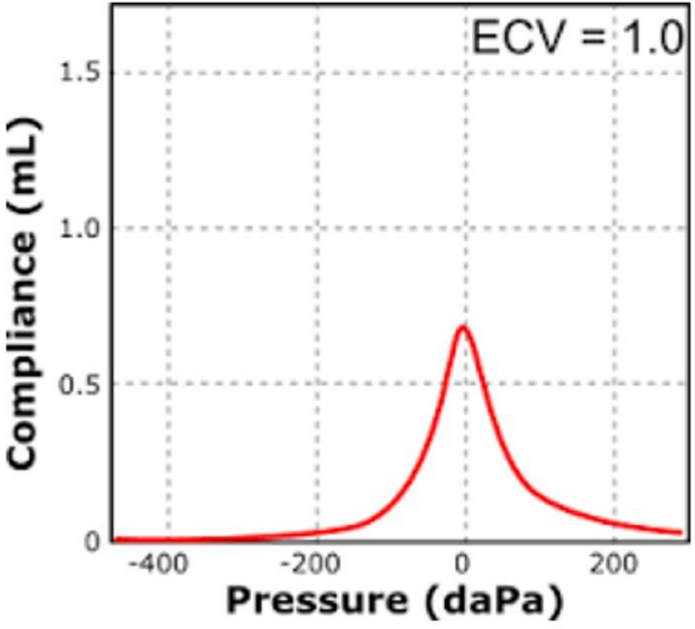
- The primary purpose of impedance audiometry is to determine the status of the tympanic membrane and middle ear via tympanometry



- A **type A** response suggests **normal middle ear function**, but it occurs in some otosclerotic ears, particularly in early stages.

ideal peak 0.7 N

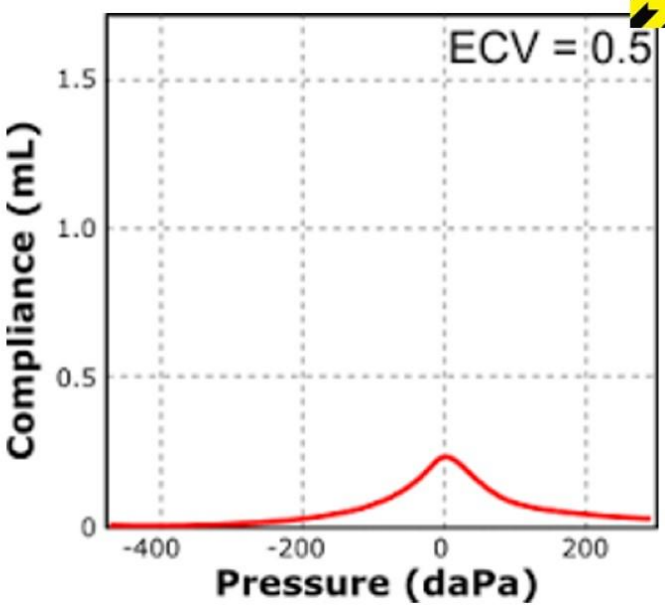
Peak is between +/- 100 daPa
Compliance from 0.3-1.5 ml



Type As (A shallow) suggests a **stiffened** (less compliant) middle ear system.

- Peak is between +/- 100 daPa
- Compliance is less than 0.3 ml

This type may suggest, **otosclerosis and malleus fixation**

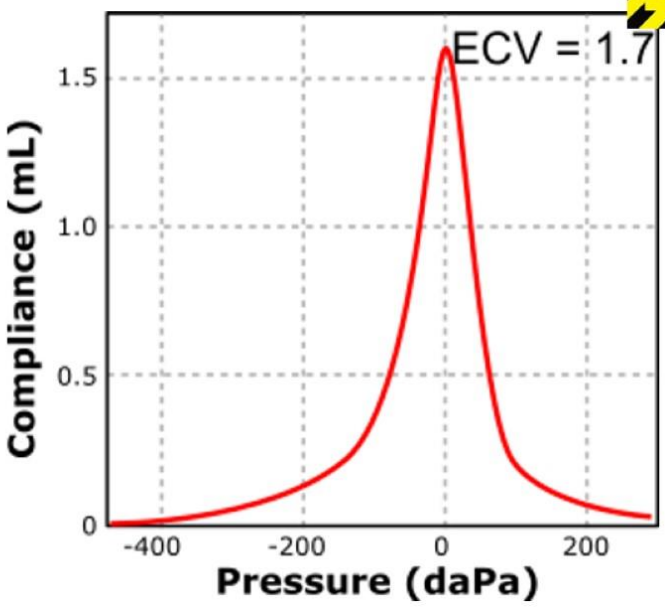


Type Ad High compliance at or near normal pressure.

- Peak is between +/- 100 daPa
- Compliance is more than 1.5 m

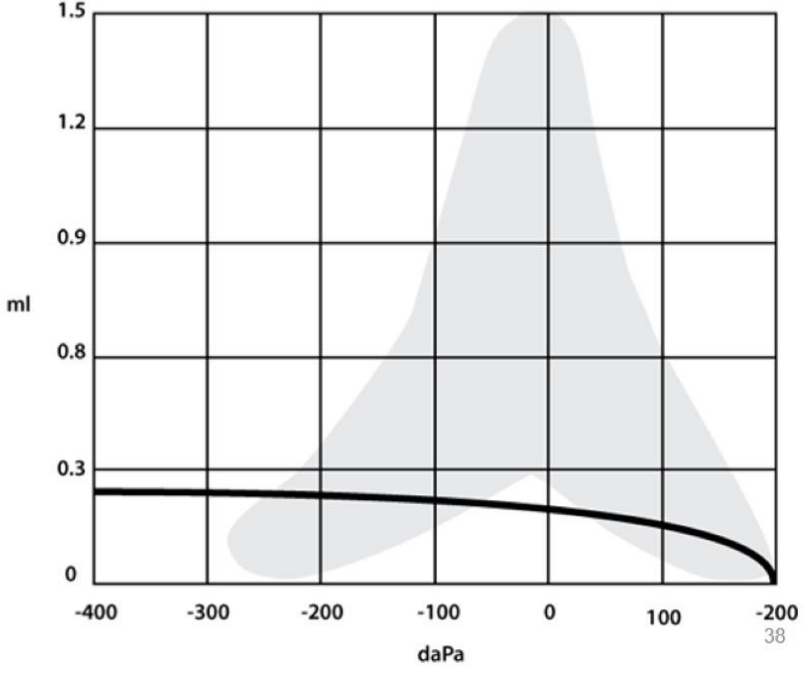
Seen in **ossicular discontinuity** or **thin and lax tympanic membrane**

Post-stapedectomy.



Type B tympanogram

Type B A flat or dome-shaped graph. No change in compliance with pressure changes.



Type B tympanogram

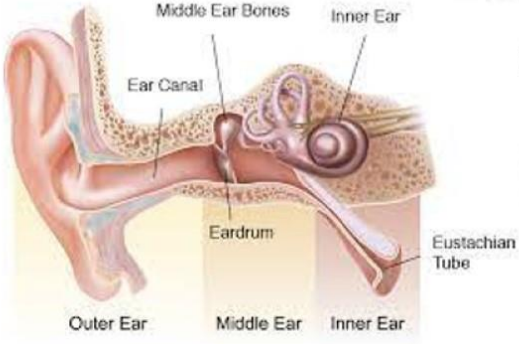
- . Type B tympanograms must be interpreted in conjunction with **ear canal volume** readings.
  - Average ear canal volumes for children are 0.5-1.0 mL.
  - Average adult volumes are 1.0-1.5 mL.
- Type B (**normal** ear canal volume) usually suggests **otitis media.** + OME
- Type B (**small** ear canal volume) may suggest that the ear canal is occluded with **wax/debris** or that the immittance **probe** is pushed against the side of the ear canal.
  - probe against canal

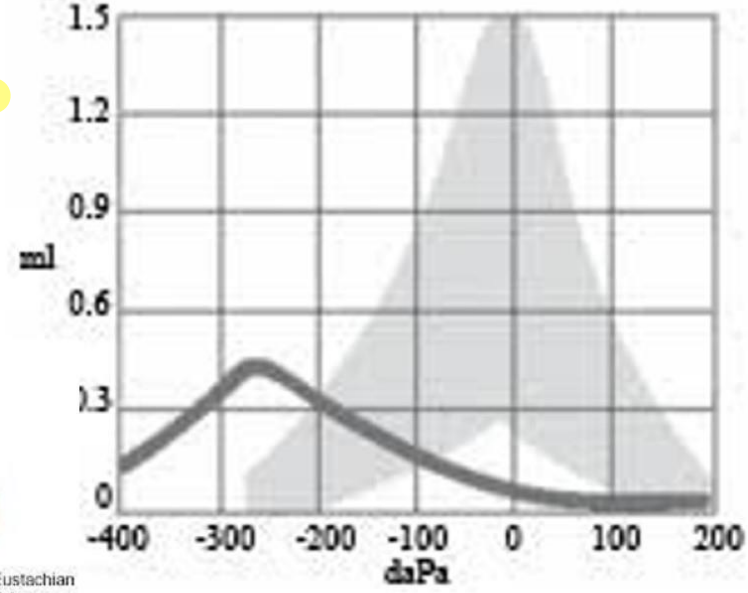
Type B (**large** ear canal volume) suggests a **perforation of the tympanic membrane**. (because middle ear volume is added up to the volume of external ear canal)

Peak is below –100 daPa
Compliance from 0.3-1.5 ml

Type C suggests significant negative pressure in the middle ear system

Additionally, this type indicates a **malfunctioning eustachian tube.**





Etiologies

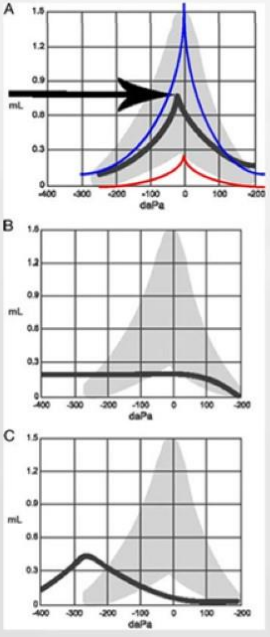


Figure 2. Tympanograms

Type A → Normal

Type A_d → Ossicular Discontinuity (scaling)

Type A_s → TM Scarring, Otosclerosis

Type B "Flat" →
- Otitis Media w/ Effusion
- Occluded Cerumen
- Probe against canal (ECV)
- Perforation/Patent PE tube
- Head Trauma
- Otosclerosis
- Cholesteatoma

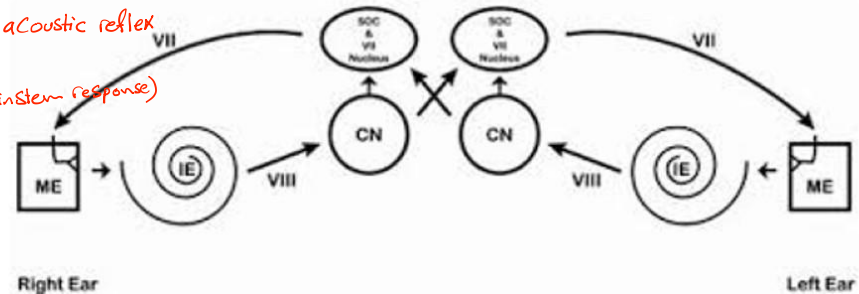
Type C →
- ETD dysfunction
- Pre/Post OM
- Barometric pressure changes

Harlor & Bower, 2009

Acoustic reflex

It is based on the fact that a loud sound, 70 - 100 dB above the threshold of hearing of a particular ear, causes bilateral contraction of the stapedial muscles which can be detected by tympanometry

- A person who feigns total deafness and does not give any response on pure tone audiometry but shows a positive stapedial reflex is a malingerer
- In otosclerosis >decreased reflex



Brainstem Evoked Response Audiometry (BERA)

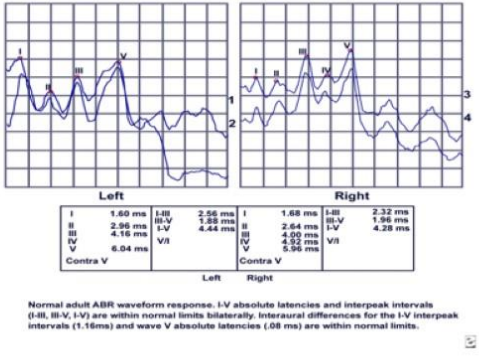
Alternative Name: Auditory Brainstem Response (ABR) Audiometry

ABR Audiometry

تخطيط سمعي دماغي

- Auditory brainstem response (ABR) audiometry is a neurologic test of auditory brainstem function in response to auditory (click) stimuli.
  - It is a test of the ***central pathways***.
- Used for **screening for retrocochlear pathology(**such as **an acoustic neuroma** or **vestibular schwannoma.), universal newborn hearing screening**, and **intraoperative monitoring**.

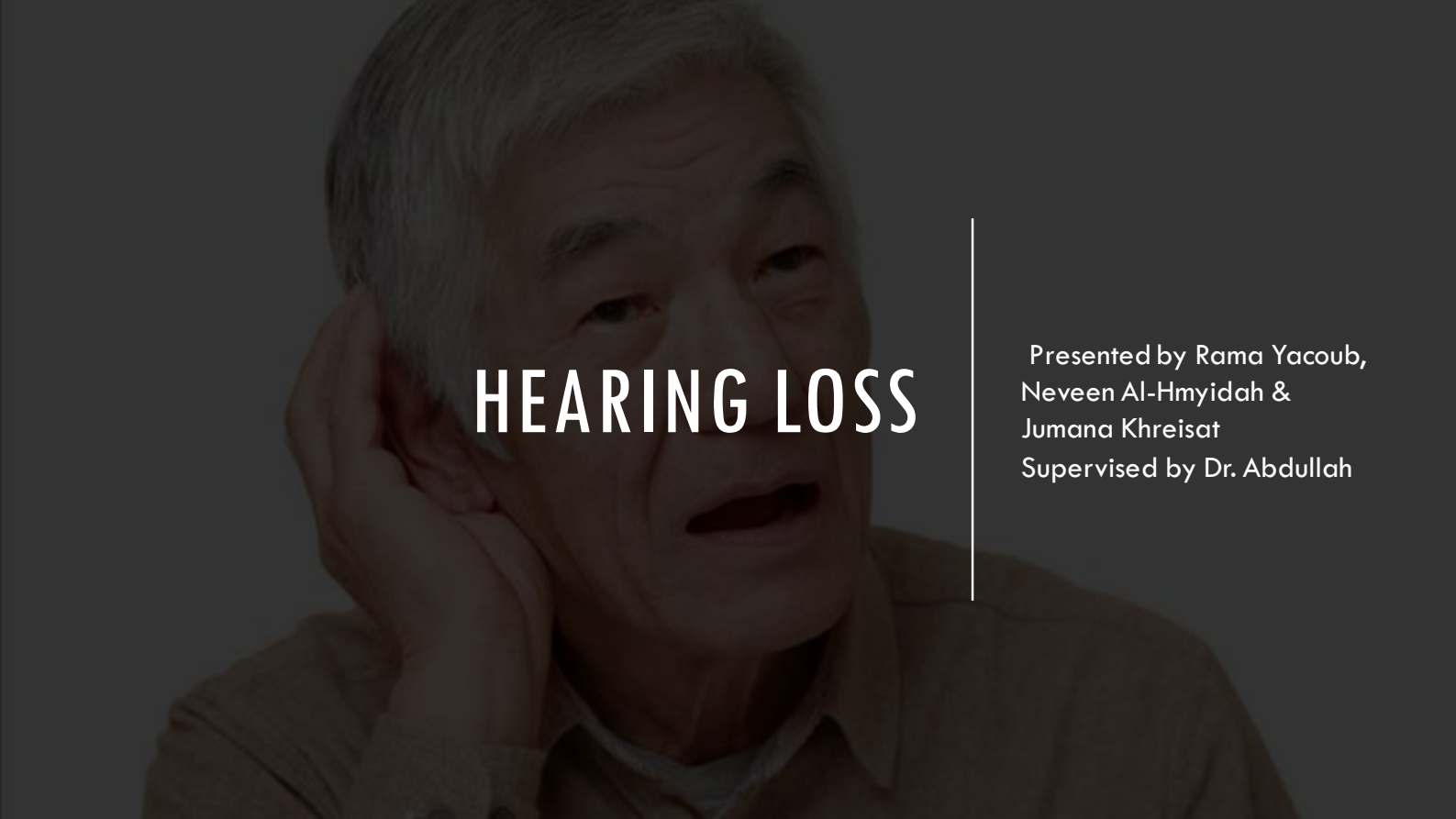
نبرة ي
Acoustic reflex



Normal adult ABR waveform response. I-V absolute latencies and interpeak intervals (I-III, III-V, I-V) are within normal limits bilaterally. Interaural differences for the I-V interpeak intervals (1.16ms) and wave V absolute latencies (.08 ms) are within normal limits.

Otoacoustic Emission

- The primary purpose of otoacoustic emission (OAE) tests is to determine **cochlear status, specifically hair cell function**.
- This information can be used to:
  - Screen hearing (particularly in neonates, infants, or individuals with developmental disabilities)
  - Partially estimate hearing sensitivity within a limited range
  - Differentiate between the sensory and neural components of sensorineural hearing loss.
  - Test for functional (feigned) hearing loss
- Does not measure the central pathway



HEARING LOSS

Presented by Rama Yacoub,
Neveen Al-Hmyidah &
Jumana Khreisat
Supervised by Dr. Abdullah

DEFINITION

- Hearing loss is a decrease in the ability to perceive sound or any degree of impairment of the ability to apprehend sound.
- May be present at birth or acquired at any time.
- May occur in one or both ears.
- Can be temporary or permanent.
- In adults it can create difficulties with social interaction and at work.

*in children "if untreated" can cause verbal, social and emotional development impairment

CONDUCTIVE HEARING LOSS

CONDUCTIVE HEARING LOSS

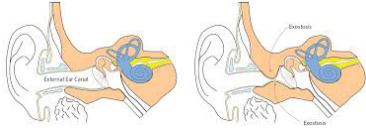
5-15 %

- It is caused by any disease process interfering with the conduction of sound from the external ear to the stapediovestibular joint.
- Causes :

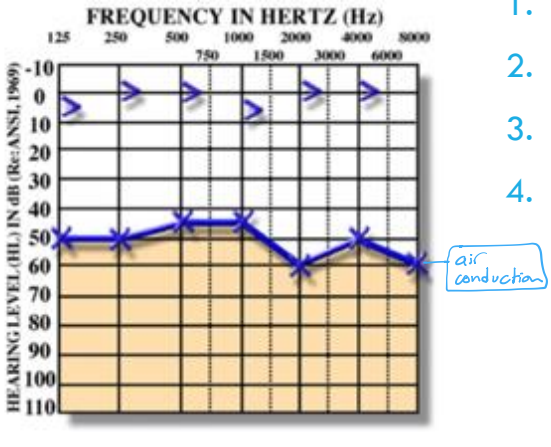
1. The external ear (obstruction). → ear wax, foreign body
2. Tympanic membrane (perforation).
3. Middle ear (fluid). → OME — also → infection < otitis media / otitis externa
4. Ossicles (fixation or disruption). → Otosclerosis
5. The Eustachian tube (obstruction). or dysfunction

* also Cholesteatoma / Exostoses (external) / tumors

Exostoses are bony growths in your ear canal that can occur due to frequent exposure to cold water. "Sufer's ear"



THE CHARACTERISTICS OF CHL ARE:



1. Negative Rinne test.
2. Weber lateralized to the affected ear.
3. Low frequencies were affected more.
4. Audiometry shows bone conduction is better than air conduction with an air-bone gap.

MANAGEMENT OF CONDUCTIVE HEARING LOSS

1. Removal of canal obstruction, e.g. impacted wax, foreign body, benign or malignant tumor.
2. Removal of fluid. → by Myringotomy
3. Removal of mass from the middle ear. → (Cholesteatoma)
4. Tympanoplasty (repair of perforation)
5. Hearing aid, in cases where surgery is not possible, refused, or has failed.

Cholesteatoma is a skin-lined cyst that begins at the margin of the eardrum and invades the middle ear and mastoid

OTOSCLEROSIS

abnormal extension of sponge-like bone growing in the middle ear cavity. This growth prevents the ear bones from vibrating normally in response to sound waves.

abnormal bone remodeling in middle ear → unknown cause, may be congenital

abnormal hardening

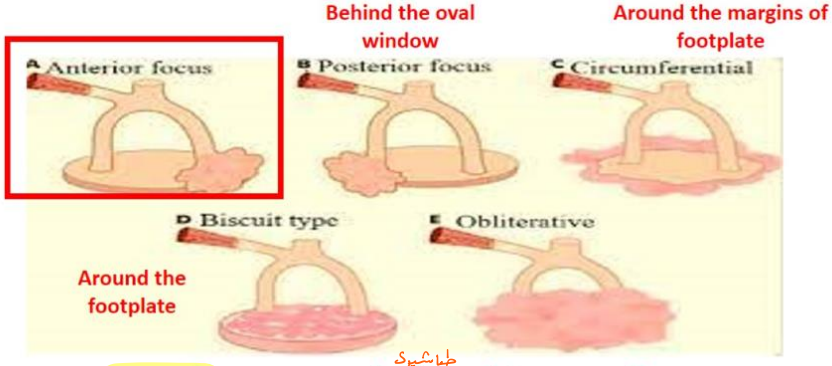
OTOSCLEROSIS (OTOSPONGIOSIS)

- Common middle ear disease. (MCC of middle ear hearing loss in young adults)
- Is a primary disease of the bony labyrinth.
- In this, one or more foci of irregularly spongy bone replace part of the normally dense enchondral layer of the bony otic capsule. (bony labyrinth)
- Most often, otosclerotic focus involves the stapes region leading to stapes fixation and conductive deafness.
- The exact cause of otosclerosis is not known; however, the following facts have been documented:

1. Anatomical basis/heredity /race (white people)/sex. (more in women)
2. Viral infection: electron microscopic and immunohistochemical studies have shown RNA related to measles virus. Otosclerosis is likely a viral disease.

in oval window, Stiffening the middle ear sys.

- **Stapedial otosclerosis**: causing stapes fixation and conductive deafness is the most common variety.
- Here lesion starts just in the front of the oval window in an area called "fistula ante fenestram."(anterior focus).



طباشيري

Grossly appears Chalky white or yellow.

- **Cochlear otosclerosis**: involves a region of round window or other areas in the otic capsule and may cause sensorineural hearing loss probably due to the liberation of toxic materials into the inner ear fluid.

release

OTOSCLEROSIS (OTOSPONGIOSIS)

- Symptoms of otosclerosis:

1. Hearing loss. This is the presenting symptom and usually starts in the twenties.
2. Tinnitus
3. Vertigo

- Deferential diagnosis:

1. Serous otitis media. (OME)
2. Adhesive otitis media. form of chronic otitis media where there is an adhesion of medial ear structures as a result of chronic inflammation. There are often complete or partial adhesions between the thin retracted and atrophic pars tensa and the medial wall of the middle ear.
3. Tympanosclerosis. → results in deposits of dense hyaline tissue or even bone in lamina propria of middle ear mucous mem.
   → scaring of tympanic mem. after injury or surgery
4. Attic fixation of head of malleus. congenital or acquired
   can be ass. with stapes fixation
5. Ossicular discontinuity or congenital stapes fixation.

separation of middle ear ossicles at one of joints or within above (fracture)
↳ basal skull fracture (trauma)
Chronic otitis media
Cholesteatoma

During development, the stapes bone forms adjacent to the cochlea. If the window where the stapes sits in the cochlea fails to form, congenital stapes fixation occurs.

What is the difference between congenital stapes fixation and otosclerosis?

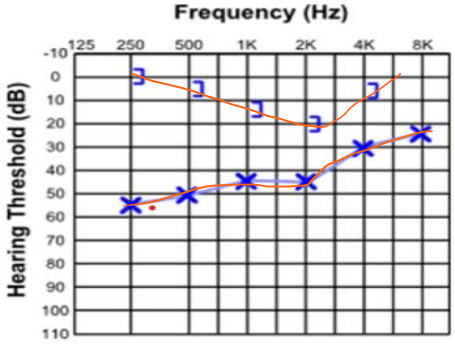
In otosclerosis, an abnormal growth of inner-ear bone leads to stapes fixation. In congenital stapes fixation, the separation between the footplate and the surrounding otic capsule bone does not occur.

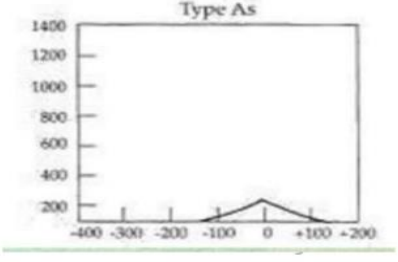
TREATMENT

- Medical: there is no medical treatment that cures otosclerosis
- Surgical: stapedectomy/stapedotomy.

❖ The characteristics of otosclerosis are:

1. Negative Rinne test.
2. Weber lateralized to the affected ear.
3. Audiometry shows the Carhart sign.
4. Tympanogram As





SENSORINEURAL HEARING LOSS

SENSORINEURAL HEARING LOSS

90%

- Sensorineural hearing loss (SNHL) results from a lesion of the cochlea, VIII (8th) nerve, or central auditory pathway. It may be present at birth (congenital) or start later in life (acquired).

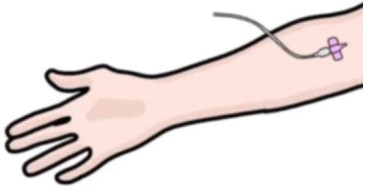
  - Acquired: it appears later in life. The cause may be genetic or nongenetic.

  - The genetic hearing loss may manifest late (delayed onset) and may affect only the hearing or be a part of a larger syndrome affecting other systems of the body as well (syndromal). → as *Pendred syndrome*

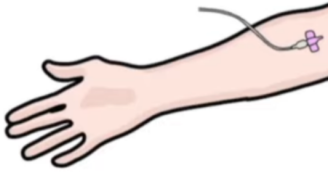
70% non Syndromic

CAUSES OF SENSORINEURAL HEARING LOSS

- SUDDEN SENSORINEURAL HEARING LOSS
  - OVER LESS THAN 72 HOURS
- PRESBYCUSIS → AGE RELATED
- NOISE EXPOSURE
- MÉNIÈRES DISEASE
- LABYRINTHITIS
- ACOUSTIC NEUROMA (vestibular schwannoma)
- NEUROLOGICAL CONDITIONS
  - → STROKE
  - → MULTIPLE SCLEROSIS
  - → BRAIN TUMOUR

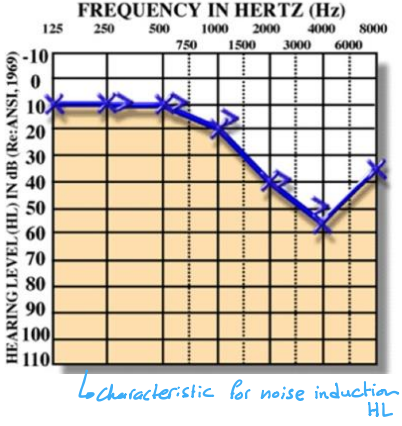


- INFECTIONS → MENINGITIS
- MEDICATIONS
  - LOOP DIURETICS (E.G. FUROSEMIDE)
  - AMINOGLYCOSIDE ANTIBIOTICS (E.G. GENTAMICIN)
  - CHEMOTHERAPY (E.G. CISPLATIN)



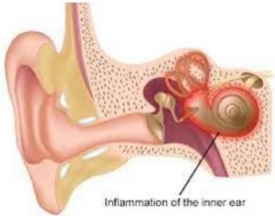
THE CHARACTERISTIC OF SENSORINEURAL HEARING LOSS ARE:

1. Positive Rinne test.
2. Weber lateralized to the better ear
3. More often involving high frequencies
4. No gap between air and bone conduction curve on audiometry.



INFLAMMATION OF LABYRINTH

INFLAMMATION OF THE LABYRINTH



1. Viral labyrinthitis : Measles + Mumps + CMV.
2. Bacterial: bacterial infections reach the labyrinth through the middle ear (tympanogenic) or CSF (meningiogenic).
3. Syphilitic

FAMILIAL PROGRESSIVE SENSORINEURAL HEARING LOSS

It is a genetic disorder in which there is progressive degeneration of the cochlear starting in late childhood or early adult life.

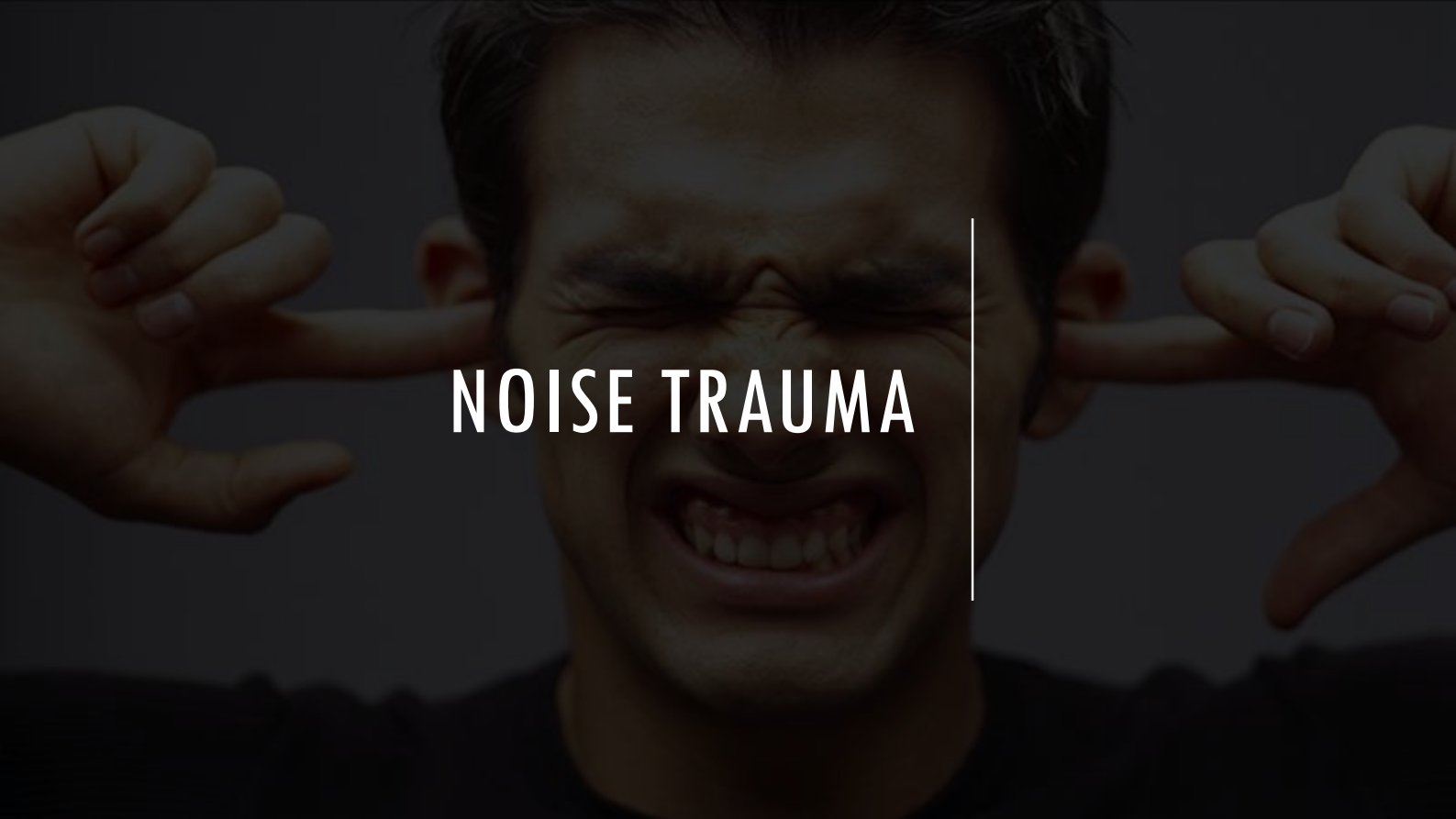
Hearing loss is bilateral with a flat or basin-shaped audiogram.

OTOTOXICITY

OTOTOXICITY

Various drugs and chemicals can damage the inner ear and cause sensorineural hearing loss:

1. Aminoglycoside antibiotic (Gentamycin) 
2. Diuretics. (furosemid)
3. Salicylates.
4. Quinine. (anti malarial)
5. Cytotoxic drugs (cisplatin) Chemo.
6. Topical ear drops.



NOISE TRAUMA

NOISE TRAUMA

Noise trauma has assumed greater significance because it is an occupational hazard, hearing loss caused by excessive noise can be divided into two groups:

1. Acoustic trauma: single brief exposure to very intense sound without this being preceded by a temporary threshold shift. Also called impulse noise, such noise can arise from an explosion, gunfire, or a powerful cracker and may reach or cross 140 dB.
2. Noise-induced hearing loss (NIHL): follows chronic exposure to less intense sounds than seen in acoustic trauma and is mainly a hazard of noisy occupations. Noise of 90 dB (A) SPL, 8 h a day for 5 days per week is the maximum safe limit, No exposure over 115 dB is to be permitted.

NOISE TRAUMA

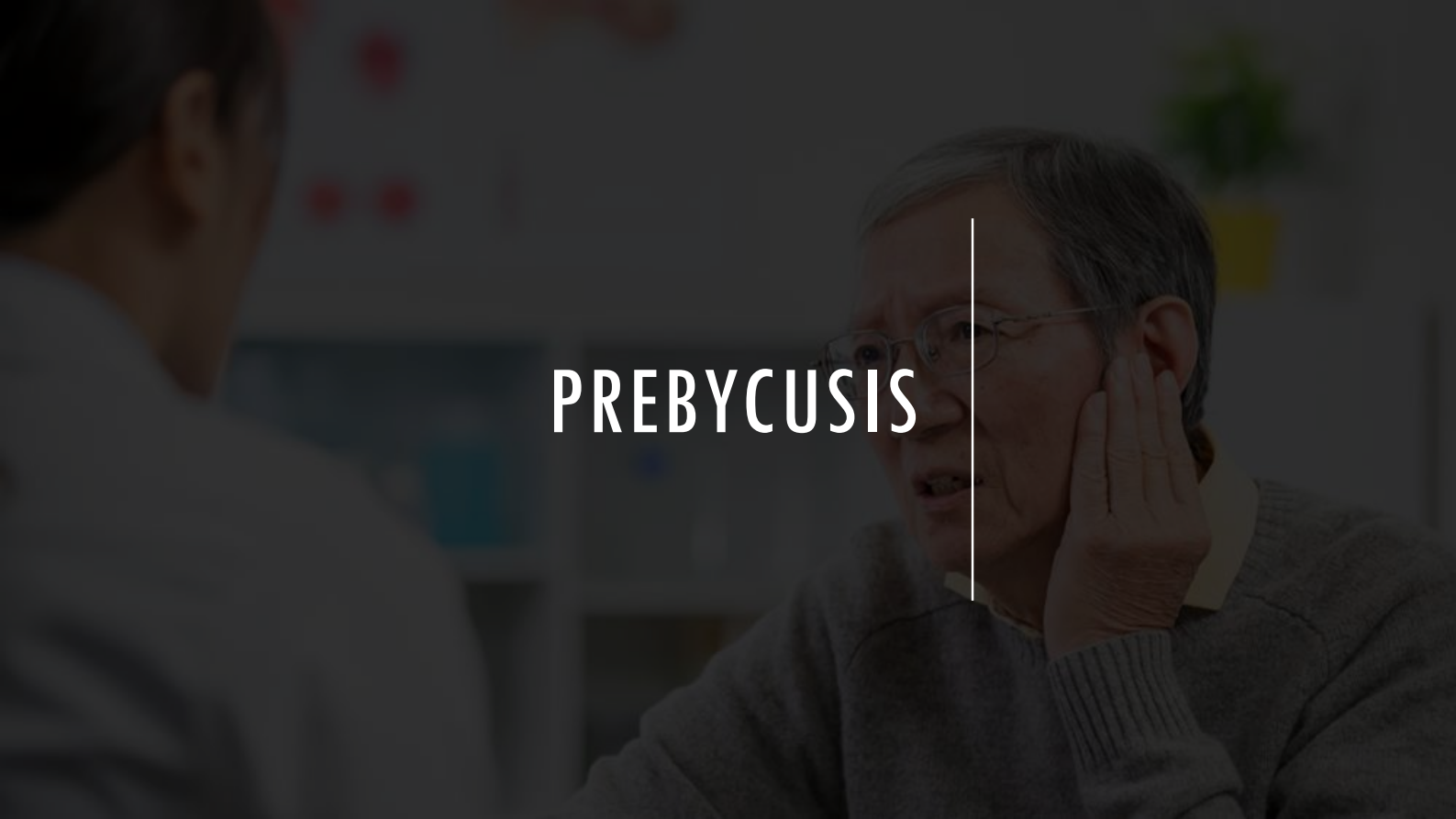
- The damage caused by noise trauma depends on several factors:

1. Frequency of noise.
2. Intensity and duration of noise: as the intensity increases, permissible time for exposure is reduced. Continuous noise is more harmful.
3. Susceptibility of the individual.
4. Pre-existing ear disease.

The "4 P's" of noise-induced hearing loss: Painless① Progressive② Permanent③ Preventable④

- Treatment:

Noise-induced hearing loss is preventable. Persons who have to work at places where noise is above 85 dB (A) should have pre-employment and then annual audiograms for early detection. Ear protectors (ear plugs or earmuffs) should be used where noise levels exceed 85 dB (A). They protect up to 35 dB noise-related hearing loss tends to be permanent, Your doctor may recommend a hearing aid or an implant to improve your ability to communicate with others.



PREBYCUSIS

PRESBYCUSIS

- Sensorineural hearing loss associated with the physiological aging process in the ear.
- It usually manifests at the age of 65 years but may do so early if there is a hereditary predisposition, chronic noise exposure, or generalized vascular disease.
- Four pathological types: sensory, neural, strial or metabolic, and cochlear conductive.
- Patients of presbycusis have great difficulty hearing in the presence of background noise though they may hear well in quiet surroundings.
- They may complain of speech being heard but not understood.
- Tinnitus is another problem and in some, it is the only complaint.
- Treatment:

  Patients of presbycusis can be helped by a hearing aid. They should also have lessons in speech reading through visual cues. Cutting off smoking and stimulants like tea and coffee may help to decrease tinnitus

SUDDEN SENSORINEURAL HEARING LOSS

SUDDEN IDIOPATHIC SENSORINEURAL HEARING LOSS

- This is a medical emergency defined as sensorineural hearing loss that has developed over a period of hours or a few days.
- Loss may be partial or complete.
- Mostly it is unilateral.
- Hearing loss of 30 dB or more at 3 contiguous frequencies over a period of 3 days or less.
- Treatment :

Steroids
very high doses

MENIERE'S DISEASE

MENIERE'S DISEASE

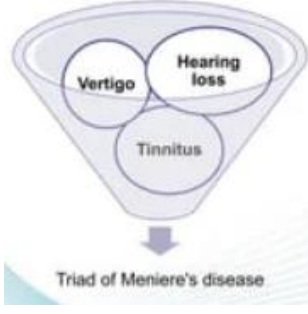
Build up of fluid within a membranous labyrinth in the inner ear

Due to impaired endolymph resorption that results in endolymph hydrops.

The cause of it is unknown.

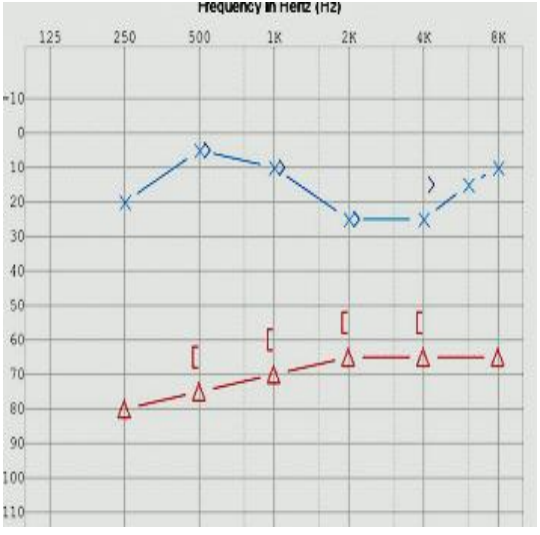
Clinical presentation: **vertigo, tinnitus, sensorineural hearing loss, horizontal nystagmus, ear fullness, nausea, vomiting. The attacks last from 20 min-12 hours**

- Low to mid-frequency hearing loss that progressively worsens with each episode
- May progress to deafness over several years.
- Starts in people between 30-50 years old



CHARACTERISTICS OF MD:

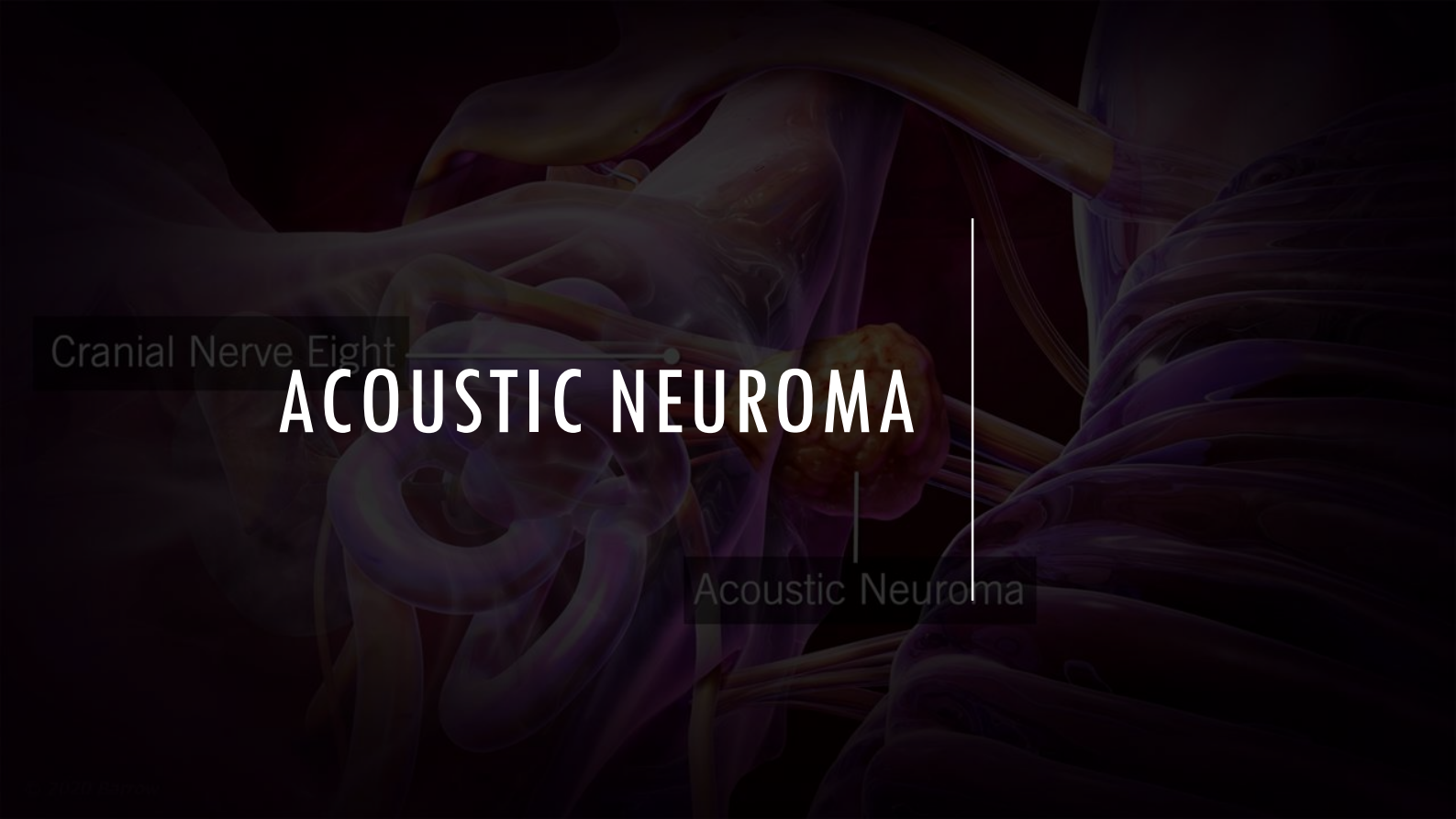
1. Tympanometry: normal (type A)
2. Rinne test: positive
3. Weber test: lateralized to the contralateral side. (healthy)
4. Hearing loss: sensorineural at low frequencies. هي نوع غريب مثل SNHL



TREATMENT

1. Lifestyle modification: limiting of salts. caffeine or alcohol quit smoking.
2. Pharmacological therapy: thiazide diuretics, chemical ablation with intratympanic injection of aminoglycoside like gentamycin (an ototoxic drug that damages the dark cell that produces endolymph). Intratympanic steroid therapy, betahistine hydrochloride (anti-vertigo).
3. Surgical in some cases: labyrinthectomy or endolymphatic sac drainage.

يؤدي إلى permanent HL in this ear!



ACOUSTIC NEUROMA

ACOUSTIC NEUROMA (VESTIBULAR SCHWANNOMA)

specifically vestibular part

It's a benign tumor that arises from Schwann cells of the vestibulocochlear nerve.

Median age: 52 years

Most commonly unilateral.

Clinical features :

First nerve affected: vestibulocochlear nerve

Second nerve: facial nerve

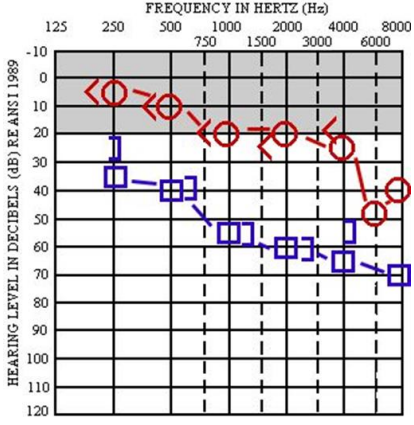
Treatment:

1. Observation
2. Highly focused radiation therapy to stop the growth of tumor (Gamma knife)
3. Surgery.

Diagnoses: MRI, CT, cranial nerve testing.

CHARACTERISTICS OF ACOUSTIC NEUROMA:

1. Tympanometry: normal type A
2. Rinnie test: positive.
3. Weber test: lateralized to the contralateral side.




MANAGEMENT

MANAGEMENT

A hearing aid amplifies sounds electronically and is effective for many people with age-related hearing loss; Hearing aids today are very small, so small that other people do not notice you are wearing them.

Cochlear implants are a small electronic device that electrically stimulates the cochlear nerve. The implant has external and internal parts. The external part sits behind the ear. It picks up sounds with a microphone. It then processes the sound and transmits it to the internal part of the implant.

Vertigo

Presented by :

Jonaid Aljaradat

Mohammad alrawashdeh

Saif alnoor aldherat

Definition

- Vertigo is a subjective sensation or illusion of movement or motion, usually rotatory but sometimes linear. It's often accompanied by pallor, sweating and vomiting

- The objective sign of vertigo is nystagmus (in which eyes make repetitive uncontrolled movements)

What are the key differences between dizziness and vertigo ?

- Dizziness is a vaguer term that, while it includes vertigo, may also refers to a sensation of discomfort in the head such as lightheadedness, disorientation, disequilibrium or imbalance.

Differential diagnosis of vertigo

Peripheral causes	Central causes	Other
Benign paroxysmal positional vertigo (most common)	Vestibular migraine	Psychiatric
Vestibular neuritis	Cerebrovascular disease	Drug induced (ototoxicity)
Meniere disease	Cerebellopontine angle and posterior fossa tumors	Cardiovascular/metabolic
Supurrative labyrinthitis		Orthostatic
Syphilitic labyrinthitis		Post-operative
Geniculate herpes zoster		Trauma to the labyrinth
Perilymph fistula		

Peripheral causes

- Body balance is maintained by the input to the brain from the inner ear, the eyes and the proprioceptive organs, especially of the neck so dysfunction of any of these systems may lead to imbalance.

- Peripheral causes arise from abnormalities in the peripheral vestibular system.

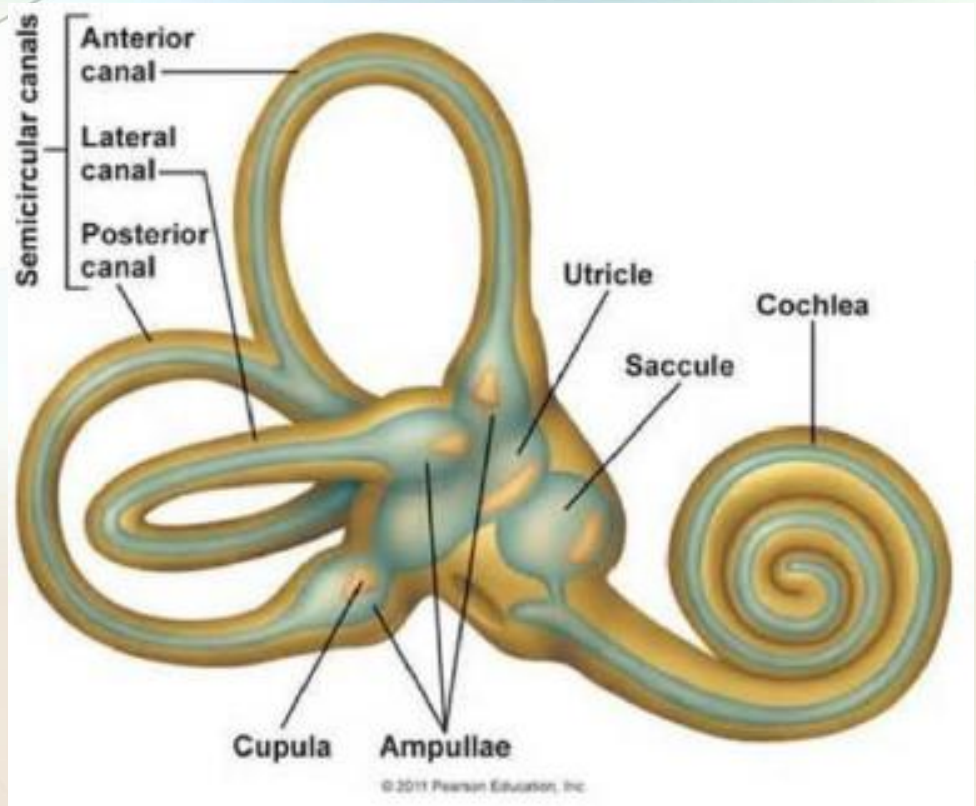
Peripheral causes

- The vestibular system is situated in the petrous part of the temporal bone, in close proximity to the cochlea. It responds to movement of the head relative to space and gravity, using inertial-sensing receptors.

- THE VESTIBULAR SYSTEM CONSISTS OF TWO TYPES OF SENSORS:

1. The two otolith organs (the saccule and utricle) , which sense linear movement .
2. A set of three semicircular canals, arranged at right angles to each other, sensing rotation movement in three planes.

- and the vestibucochlear nerve.


Semicircular canals
Anterior canal
Lateral canal
Posterior canal
Utricle
Cochlea
Saccule
Cupula
Ampullae
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1- Benign paroxysmal positional vertigo (BPPV)

- Benign paroxysmal positional vertigo is due to a degenerative condition of the utricular neuroepithelium in which calcium carbonate crystals, otoconia/canaliths, that normally reside in the utricle of the ear are dislodged and end up in the semicircular canals, usually the posterior canal.
- At any age, but is most common between 50 and 70 years.
- No obvious cause is found in 50% to 70% of older patients, and may occur spontaneously or following head trauma is a possibility in younger persons.
- Brief episodes of intermittent dizziness lasting seconds to hours.
- Common triggers are head motion on change of body position (e.g., rolling over in bed) or looking upward

1- Benign paroxysmal positional vertigo (BPPV)

- There are two main techniques used in the assessment and management of benign paroxysmal positional vertigo (BPPV) :

1- The **Dix-Hallpike Test** is used for the diagnosis of BPPV

2- The **Epley** Manoeuvre can be used for its treatment once diagnosed.

Dix-Hallpike Maneuver

Tests for **canalithiasis** of the **posterior semicircular canal**, which is the **most common cause of benign paroxysmal positional vertigo (BPPV)**

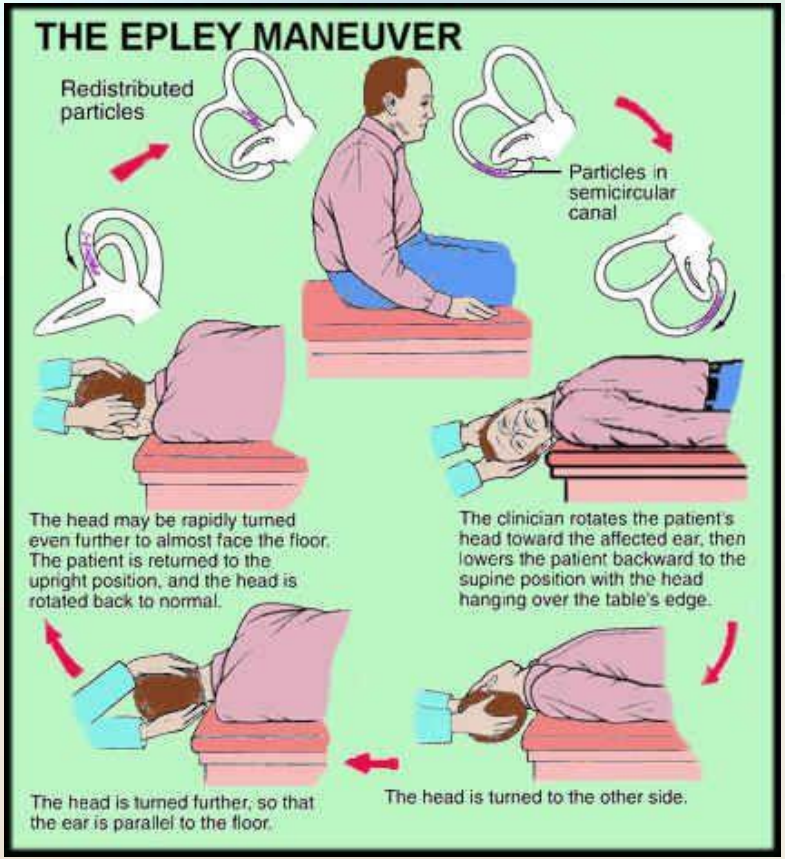
1. With the patient sitting up, turn the head 45 degrees to one side
2. Lie the patient down with head overhanging the edge of the bed and look for nystagmus
3. Repeat on the contralateral side

Positive if the maneuver **provokes paroxysmal vertigo and nystagmus**

1- Benign paroxysmal positional vertigo (BPPV)

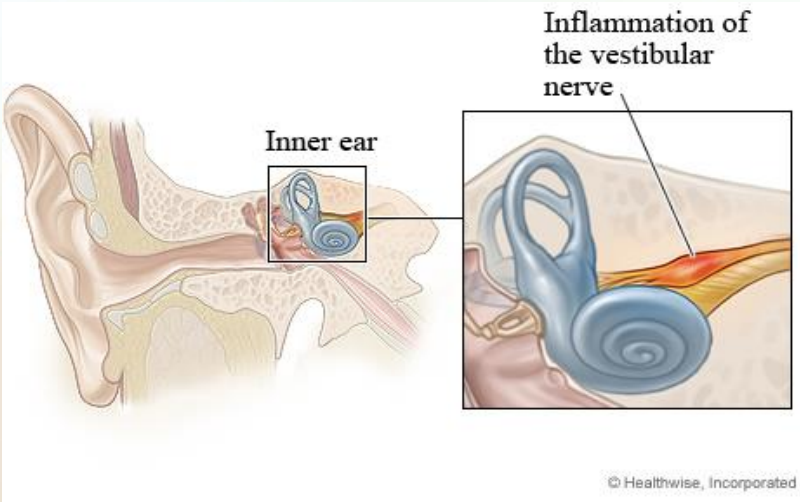
- TREATMENT:

1) canalith repositioning procedure such as the **Epley maneuver** which repositions the canalith from the semicircular canal into the vestibule. *The success rate is approximately 70% 1st attempt, and 100% on successive maneuvers.*
2) Home treatment: **Brandt-Daroff exercises** can also be successful.
3) If there is no improvement with repeated repositioning maneuvers, or if atypical or ongoing nystagmus with nausea is present, another cause should be considered


THE EPLEY MANEUVER
Redistributed particles
Particles in semicircular canal
The head may be rapidly turned even further to almost face the floor. The patient is returned to the upright position, and the head is rotated back to normal.
The clinician rotates the patient's head toward the affected ear, then lowers the patient backward to the supine position with the head hanging over the table's edge.
The head is turned further, so that the ear is parallel to the floor.
The head is turned to the other side.

2- Vestibular neuritis

- This is inflammation of the vestibular nerve, possibly due to **viral infection** which disrupts the transmission of sensory information from the ear to the brain.



2- Vestibular neuritis

- It most commonly affects persons 30 to 50 years of age.

- It can cause severe rotatory vertigo (longer than one day) with severe nausea and vomiting with apparent movement of objects in the visual field (oscillopsia), horizontally rotating spontaneous nystagmus to the non-affected side, or an abnormal gait with a tendency to fall to the affected side.

- Hearing is not impaired in this condition.

bcz (vestibular part of CN VIII only*

2- Vestibular neuritis

- As vestibular compensation occurs, the patient's vertigo resolves slowly over a few days.
- In 50% of patients, the underlying nerve damage may take two months to resolve. disequilibrium may persist for months.
- Reassurance, explanation, and advice are essential, in combination with symptomatic treatment for the first few days.

2- Vestibular neuritis

- TREATMENT:

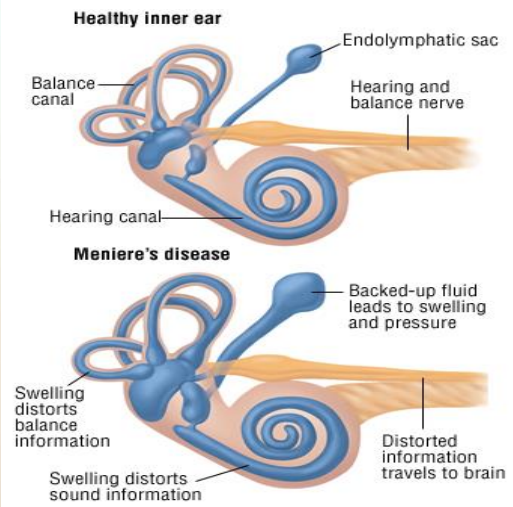
1) Vertigo and associated nausea or vomiting can be treated with a combination of antihistamine, antiemetic, or benzodiazepine.

- Antiemetics and antinausea medications should be used for no more than three days because of their effects in blocking central compensation.

2) Antibiotics or antiviral medications if caused by an underlying infection

3- Ménière's disease

- It's a condition of unknown etiology in which there's increased volume of endolymph in the semicircular canals leading to distention of the membranous labyrinth leading to inner ear dysfunction.



3- Ménière's disease

- Although it can develop at any age, it is more common between 20 and 60 years.

- Episodic spontaneous vertigo (at least two episodes lasting at least 20 minutes) associated with documented low- to medium frequency sensorineural hearing loss by audiometric testing in the affected ear (unilateral), tinnitus or aural fullness, nausea, vomiting and headache.

- Many patients report increased sensitivity to loud noises (recruitment) in addition to the listed symptoms.

- Psychological factors such as stress can act as a trigger mechanism for an attacks.

3- Ménière's disease

- TREATMENT:

1) 1st line : lifestyle changes, including limiting dietary salt intake, reducing caffeine intake, and limiting alcohol intake.

2) Daily thiazide diuretic therapy can be added if vertigo is not controlled with lifestyle changes.

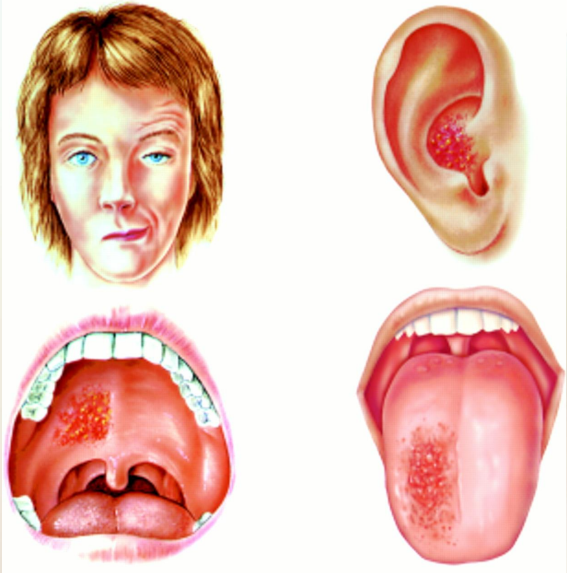
3) Transtympanic injections of glucocorticoids and gentamicin can improve vertigo.

4) Betahistine hydrochloride (anti vertigo)

5) Endolymphatic sac drainage or labyrinthectomy.

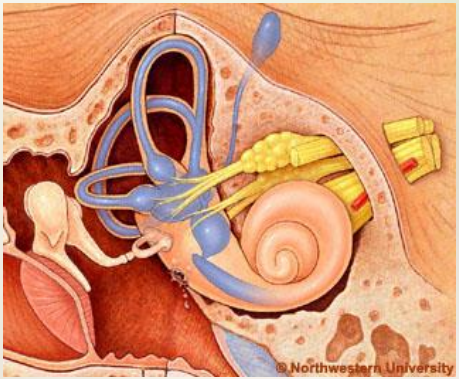
4- Geniculate herpes zoster

- Geniculate herpes zoster (Ramsay Hunt syndrome) usually causes vertigo along with facial palsy and severe pain in the ear.
- Caused by the reactivation of varicella zoster virus in the geniculate ganglion (a nerve cell band of the facial nerve)



5- Perilymph fistula

- It is a result of spontaneous rupture of the round-window membrane or trauma to the stapes footplate.
- Perilymph fistula causes marked vertigo with tinnitus and deafness.
- There is usually a history of straining, lifting or subaqua diving in the spontaneous cases, and treatment is by bed-rest initially, followed by surgical repair if symptoms persist.



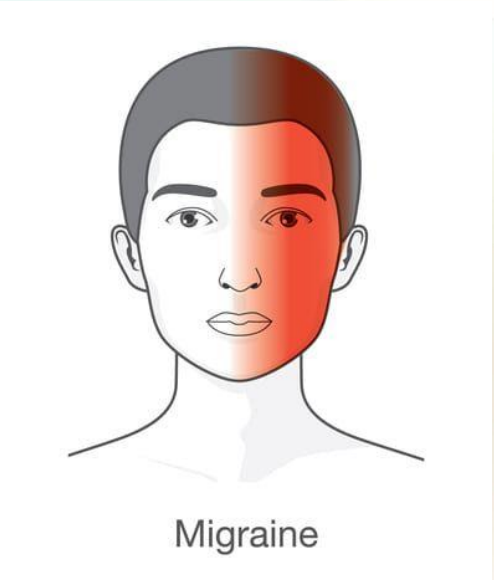
Central causes

- The vestibular nuclei, cerebellum, brainstem, spinal cord, and vestibular cortex make up the central vestibular system.

- May present with disequilibrium and ataxia rather than true vertigo. However, vertigo can be a presenting symptom of an impending cerebrovascular event.

1- Vestibular migraine

- Episodic vertigo in a patient with a history of migraine headaches.



2- Vertebrobasilar insufficiency (ischemia)

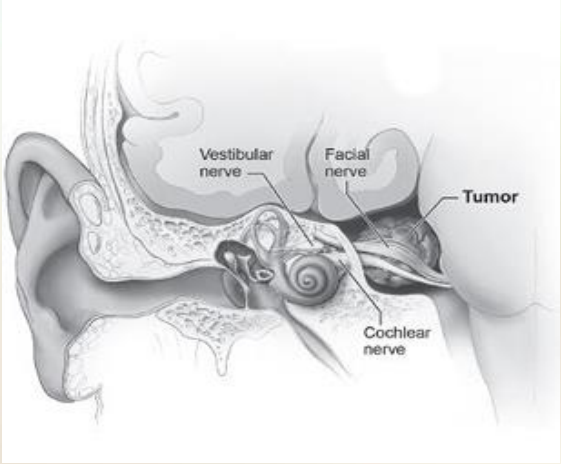
- The blood supply to the brainstem, cerebellum, and inner ear is derived from the vertebrobasilar system.

- Vertebrobasilar insufficiency may cause momentary attacks of vertigo precipitated by neck extension.

- Vertigo is the initial symptom in 48% of patients and diagnosis usually relies on a history of brainstem symptoms, such as diplopia, dysarthria, weakness, or clumsiness of the limbs.

3- Acoustic neuroma(vestibular shwannoma)

- It is a slow-growing benign tumor of the vestibular nerve that causes gradual unilateral sensorineural hearing loss and slow loss of vestibular function and vertigo.

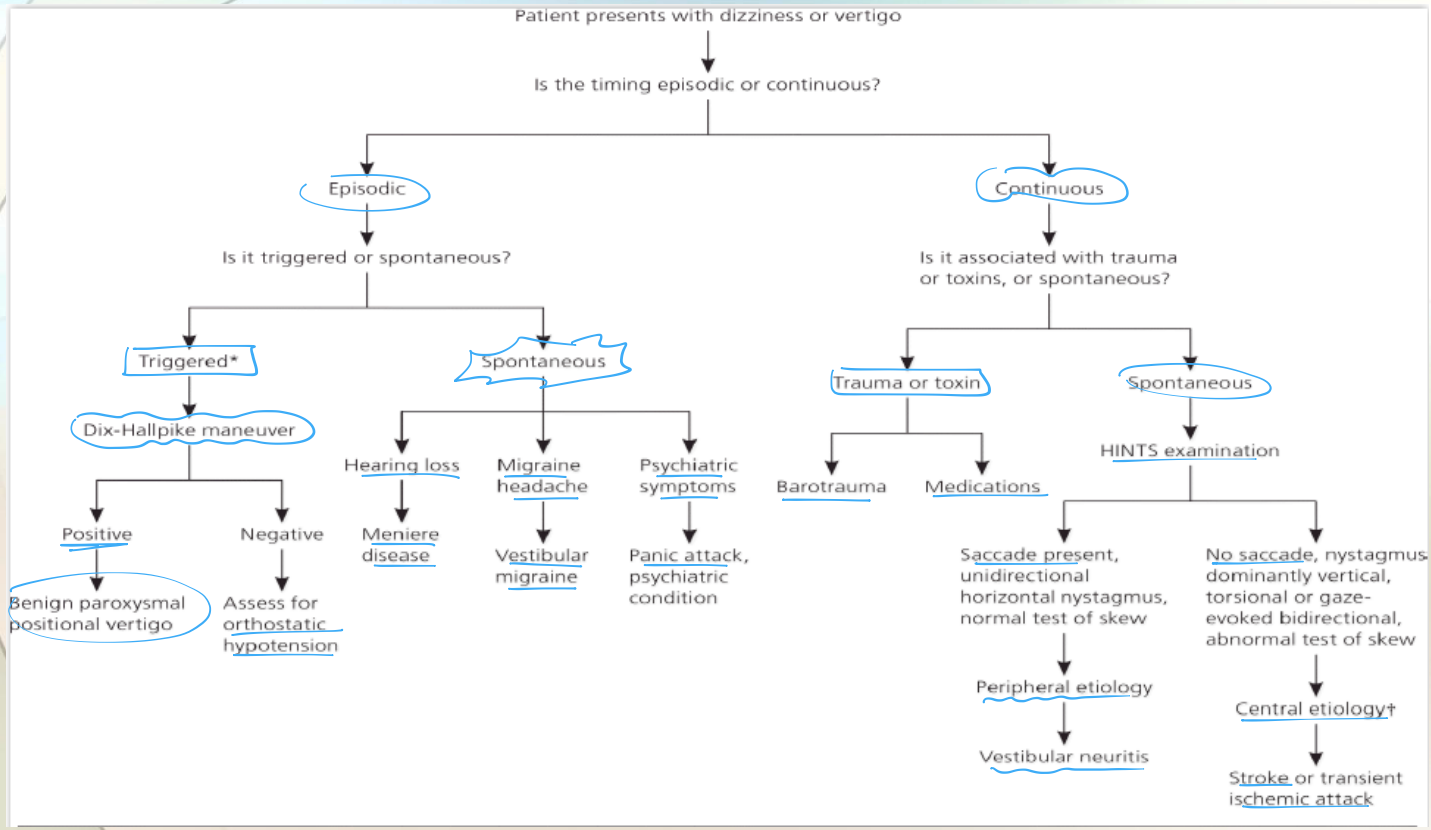


General Approach

- The diagnosis of the cause of vertigo or imbalance depends mostly on history, examination and little on investigation.

A. History

1. **Timing** (onset, duration, and evolution of dizziness)
2. **Triggers** (actions, movements, or situations) that provoke dizziness.
3. **Aural symptoms**: deafness, fluctuating or progressive; tinnitus; earache discharge.
4. **Neurological symptoms**: loss of consciousness, weakness, numbness, dysarthria, diplopia, fitting..


Patient presents with dizziness or vertigo
Is the timing episodic or continuous?
Episodic
Is it triggered or spontaneous?
Triggered*
Dix-Hallpike maneuver
Positive
Benign paroxysmal positional vertigo
Negative
Assess for orthostatic hypotension
Spontaneous
Hearing loss
Meniere disease
Migraine headache
Vestibular migraine
Psychiatric symptoms
Panic attack, psychiatric condition
Continuous
Is it associated with trauma or toxins, or spontaneous?
Trauma or toxin
Barotrauma
Medications
Spontaneous
HINTS examination
Saccade present, unidirectional horizontal nystagmus, normal test of skew
Peripheral etiology
Vestibular neuritis
No saccade, nystagmus dominantly vertical, torsional or gaze-evoked bidirectional, abnormal test of skew
Central etiology†
Stroke or transient ischemic attack

General Approach

B. Physical Examination

- Findings from the physical examination—including a cardiac and neurologic assessment, with attention to the head, eye, ear, nose, and throat examination.
- The use of the **HINTS (head-impulse, nystagmus, test of skew) examination** can help distinguish a possible stroke (central cause) from acute vestibular syndrome (peripheral cause).
- Rotation test and caloric reflex test

Caloric reflex test

- The **Caloric Reflex Test** is used to test the Vestibulo–ocular reflex.

- **HOW IT WORKS:**

Cold (30C) or warm water (44C) is flushed into the external auditory canal via a clean syringe. The difference in temperature between the water and your body generates convection signals in the endolymph of the ear mimicking head rotation.

Caloric reflex test

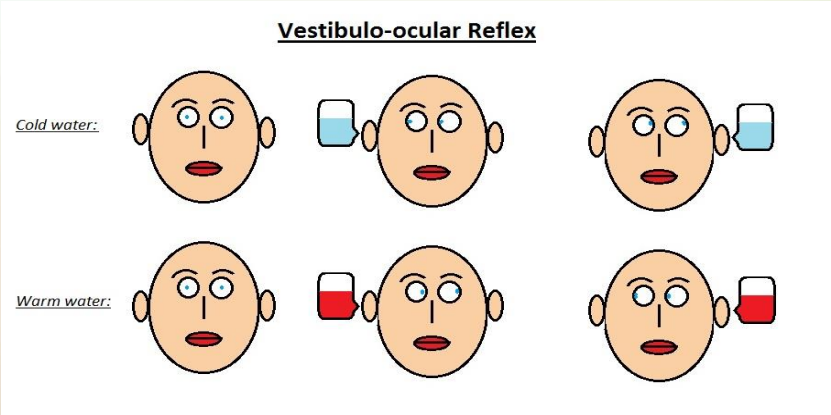
- **Mnemonic= COWS**

The **normal** response is

1- Irrigated with COLD water: Eyes deviate to ipsilateral ear and the nystagmus beats away to the **OPPOSITE** ear.

2- Irrigated with WARM water: Eyes deviate to contralateral ear and the nystagmus beats towards to the **SAME** ear.

- **C**old
- **O**pposite
- **W**arm
- **S**ame



Caloric reflex test

Each ear washed for 40 seconds

- Normal result→ nystagmus & vertigo last 90 sec
- >90 sec→hypofunction
- no response→dead ear

General Approach

C. Investigations

1- Tests of vestibular system (balance) function include

- electronystagmography (ENG),
- computerized dynamic posturography (CDP).

2- Tests of auditory system (hearing) function include

- pure-tone audiometry,
- speech audiometry,
- acoustic-reflex, electrocochleography (ECoG), otoacoustic emissions (OAE), and auditory brainstem response test (ABR).

3- Other diagnostic tests include magnetic resonance imaging (MRI) and computerized axial tomography (CAT or CT).

Epistaxis

Supervised by: DR.OSAMA

PRESENTED BY :

- Siraj Maaitah
-Rashed Malahmeh
-Mohammad Asasfeh

INTRODUCTION

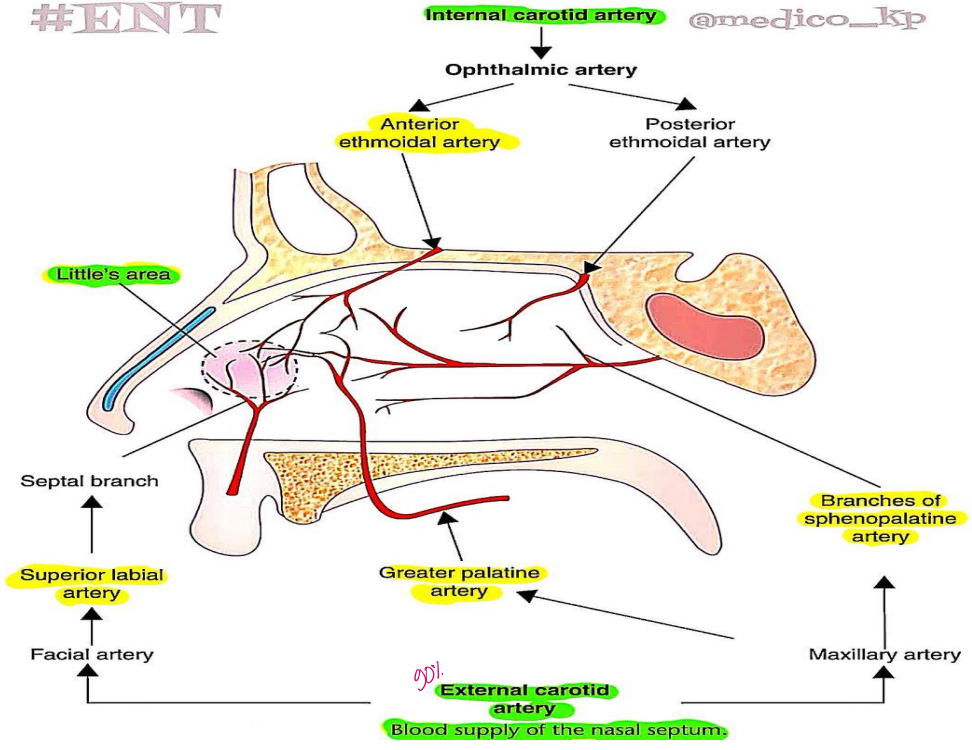
- **Most common Otolaryngological emergency in the U.S.**
- **Presents in 7-14% of general population each year**
- **Estimated lifetime incidence 60%**
- **Most of the time, bleeding is self-limited, but can often be serious and life threatening.**
- **Epistaxis should never be treated as a harmless event.**

• INTRODUCTION

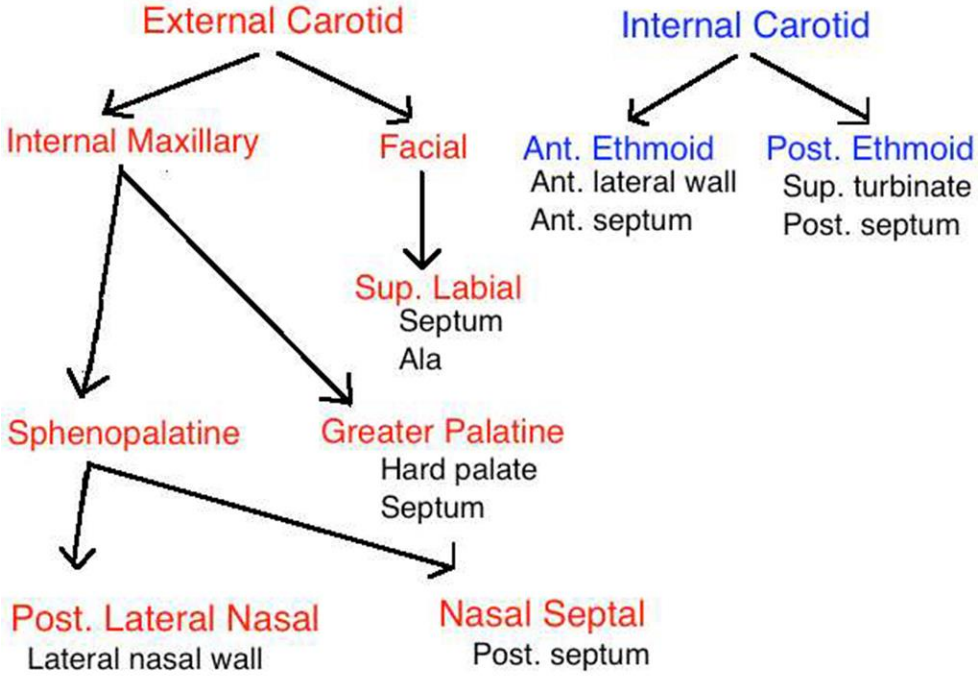
- ✓ Bimodal incidence (2-10)& (60-80) years.
- ✓ Males>females.
- ✓ Winter (due to dryness of mucosa).

Arterial supply of the nasal septum

- **The nasal septum is supplied by the following arteries:**
- **- Anterior and posterior ethmoidal branches of the ophthalmic artery, which is a branch of the internal carotid artery**
- **- Sphenopalatine branch of the maxillary artery, which is a branch of the external carotid artery**
- **- Superior labial branch of the facial artery (not shown here), which is also a branch of the external carotid artery**

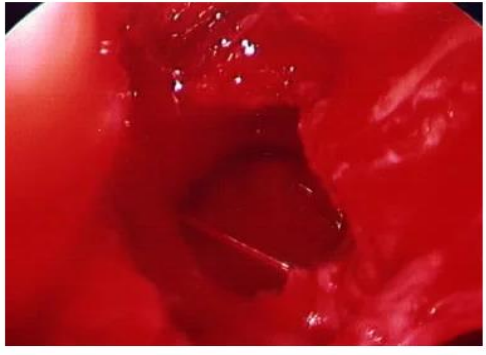


1. Anterior ethmoidal artery (IC).
2. Sphenopalatine artery (EC).
3. Superior labial artery (EC).
4. Greater palatine artery (EC).

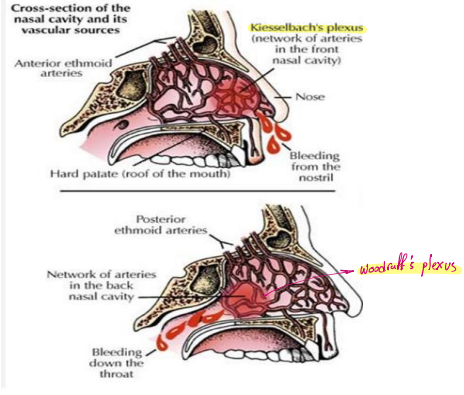

External Carotid
Internal Carotid
Internal Maxillary
Facial
Ant. Ethmoid
Ant. lateral wall
Ant. septum
Post. Ethmoid
Sup. turbinate
Post. septum
Sup. Labial
Septum
Ala
Sphenopalatine
Greater Palatine
Hard palate
Septum
Post. Lateral Nasal
Lateral nasal wall
Nasal Septal
Post. septum

Anterior Vs Posterior epistaxis

- **Epistaxis can be divided into 2 categories, anterior bleeds and posterior bleeds, on the basis of the site where the bleeding originates.**



Posterior epistaxis from the left sphenopalatine artery.



Posterior epistaxis may be a sign of life-threatening hemorrhages.

Anterior bleeding 90%	Posterior bleeding
More common	Less common
Young patient	Older age
Due to mucosal dryness, picking in children	Due to HTN
Less significant	More significant

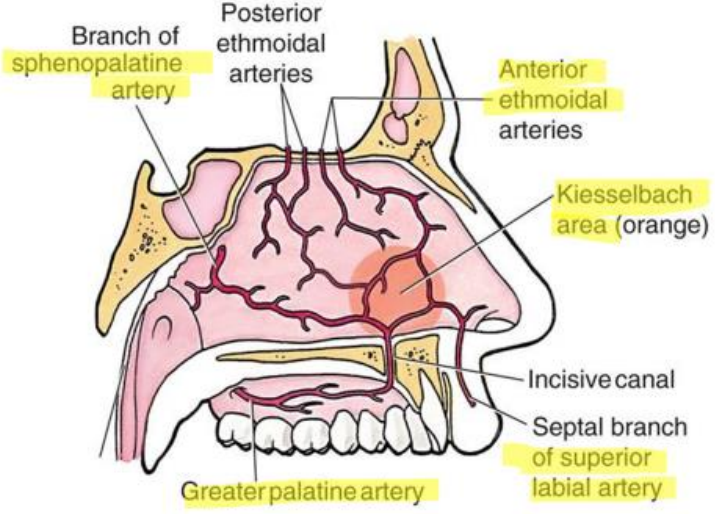
The Kiesselbach's Plexus is formed by :

- the anastomoses between the **anterior ethmoidal, sphenopalatine, greater palatine, and superior labial arteries.** posterior ethmoidal a. ← مش posterior

-It is located in the **antero-inferior region** of the nasal **septum and is the most common site of epistaxis.**

To remember the vessels that form the Kiesselbach plexus, think of **LEGS**: **L**abial (superior), **E**thmoidal (anterior), **G**reater palatine, and **S**phenopalatine arteries.

Kiesselbach's Plexus – (Little's Area)



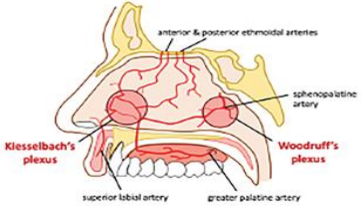
Woodruff's Plexus

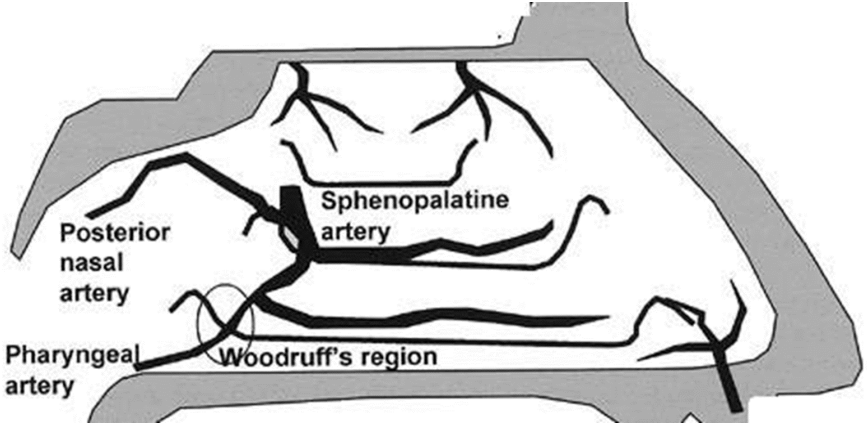
- A collection of arteries located in the postero-inferior region of the lateral nasal cavity, formed by anastomoses of the **sphenopalatine artery (**branch of the maxillary artery) and **pharyngeal artery**

Sites of posterior bleeding :

✓ **Sphenopalatine artery (most common).**

✓ **Woodruff's region.**





90% idiopathic

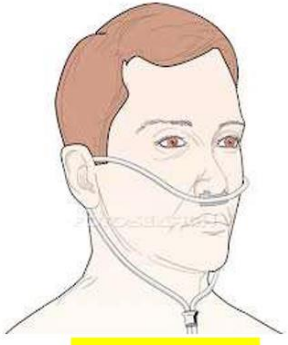
Etiology

Local factors :

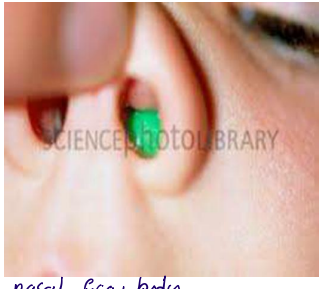
90% idiopathic

1. **Traumatic** (fractures, foreign body, nose picking).
2. **Inflammatory** (rhinitis, sinusitis).
3. **Neoplastic** (tumors of the nose, sinuses and nasopharynx).
4. **Environmental** (high altitude, air conditioning).
5. **Iatrogenic.**
6. **DNS**.(deviated nasal septum)
7. **Chemical irritants.**

mcc in child



nasal cannula

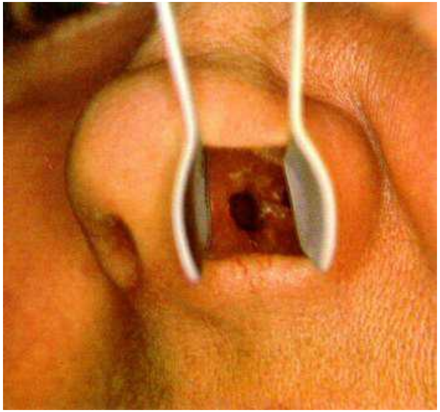


nasal foreign body

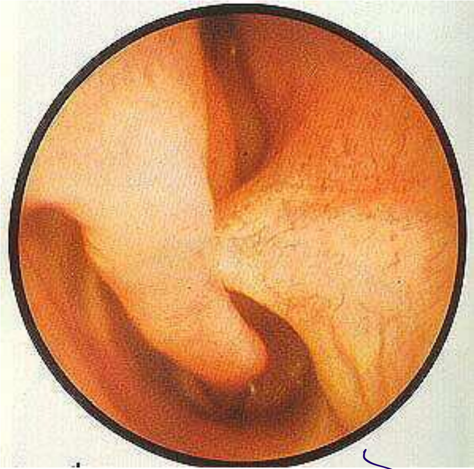
Systemic factors :

1. Coagulopathies (hemophilia, leukemia).
2. Anticoagulant medications
3. ASA & NSAIDS
4. Vascular abnormalities
5. Renal \ liver failure
6. HTN

Aspirin (acetylsalicylic acid)

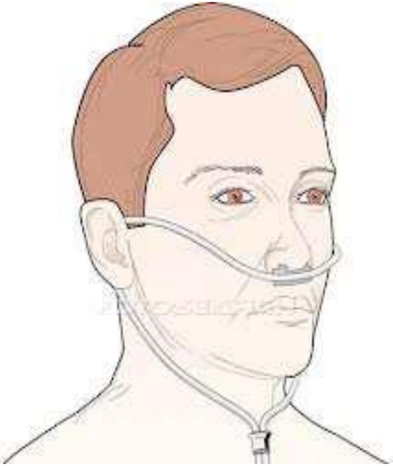


nasal septum perforation



deviated nasal septum

sever hedach
+ epistaxis



nasal cannula

<u>Perforations</u> can occur due to complications in surgery, aggressive cautery for recurrent nosebleeds, nasal trauma or fracture, infection, or from certain autoimmune diseases.

* Septal perforation
SLE
TB
:
Macca position
head down

* decongestion
head up

- **Neoplasms :**

✓**Juvenile nasopharyngeal Angiofibroma.**

✓**Inverted papilloma.**

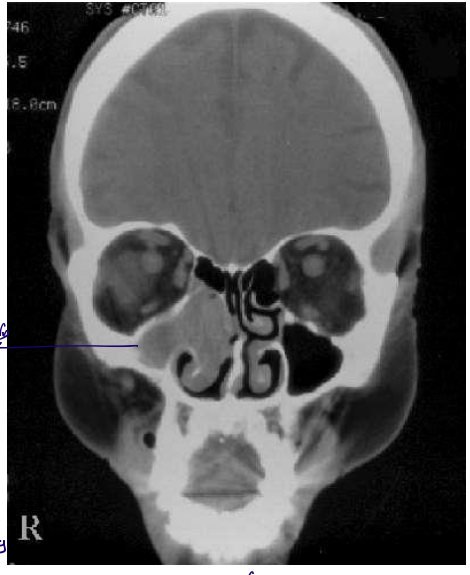
✓**SCCA.**

✓**Adenocarcinoma.**

✓**Melanoma.**

✓**Esthesioneuroblastoma.**

✓**Lymphoma.**



contraquinal polyp
↳surgical ttt
fill maxillary sinuses

HTN as a cause of epistaxis :

- ✓systematic review was done by Kikidis et al. March 2013 -European Archives of Otolaryngology –
- ✓Although multiple studies exist that examine this relationship, no consensus has been achieved.

- ✓The primary issue is that there are multiple confounding factors such as ***age and use of anticoagulation medication*** that may be the cause of epistaxis and not the hypertension itself.
- ✓Increased age induces fibrosis of the tunica media of the arteries, which may lead to inadequate vasoconstriction after rupture of a blood vessel.

• Hereditary Hemorrhagic Telangiectasia (HHT) :

•**Definition:** a hereditary, systemic vasculopathy characterized by telangiectasia on the skin and mucosa, particularly in the area of the face (nose, lips, tongue)

- ✓AKA **Osler-Weber- Rendu disease.**
- ✓**Autosomal dominant.**
- ✓ **Widespread cutaneous, mucosal, and visceral telangiectasias (arteriovenous malformations) in the brain, lungs, liver, and gut.**
- ✓**Manifests in nose as raised Lesions.**
- ✓***Treated by : Septodermoplasty.***

The principle of septodermoplasty is to replace the fragile nasal mucosa with split-thickness skin graft.

HHT

Treated by : Septodermoplasty.

Initial Management

- ABC
- Is your patient in shock?
  IV access
- Hippocratic technique

HISTORY

- Amount, duration
- Previous bleeding episodes
- Nasal trauma, obstruction
- Easy bruise ability
- Family history of bleeding
- HTN– current medications and how tightly controlled
- Use of anticoagulants
- Renal \ Hepatic diseases
- Other medical conditions – DM, CAD, etc.

PHYSICAL EXAMINATION

- Sitting position

- Wear protective clothing

- Equipments :
  - couch or reclining chair,
  - headlight, suction , vasoconstrictor solutions, → Adrenalin CI in HTN Pt ephidrine can be used
  - packs, tampons, cautery, nasal endoscopy


Gelfoam®
absorbable gelatin sponge, USP
Size 50
5"x9"
Xeroform
PETROLATUM DRESSING
Merocel
Standard Nasal Dressing, 8 cm
Rhinall
Comeen

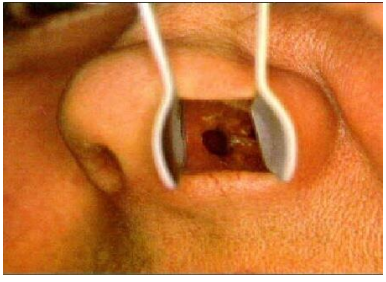
PHYSICAL EXAM

Anterior rhinoscopy :

- Suck out any clots
- Side of bleeding
- Anterior vs. posterior
- Local anesthesia & vasoconstriction

Posterior rhinoscopy :

- Endoscopic visualization



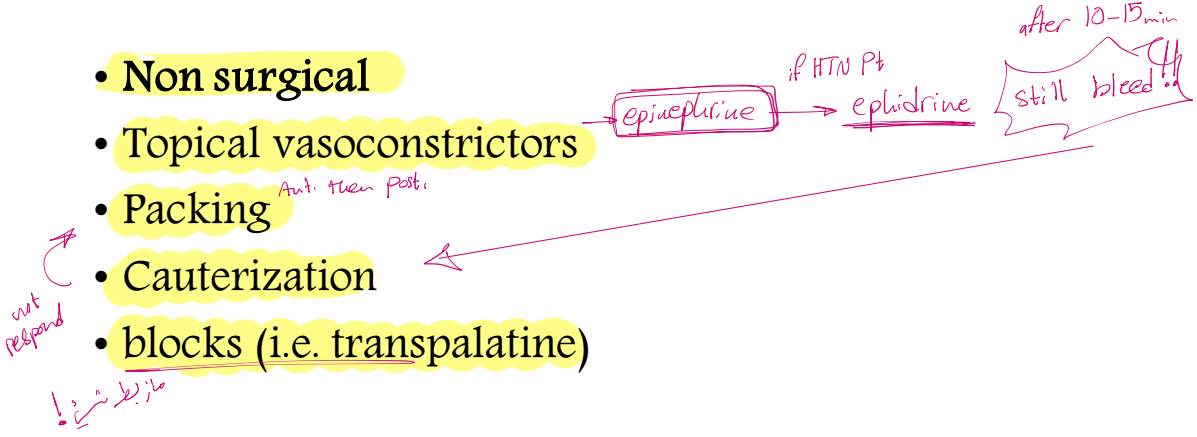
LAB STUDY

- CBC (including platelet)
- X-Match
- KFT
- LFT
- Coagulation profile

Treatment

- **Non surgical**
- Topical vasoconstrictors
- Packing
- Cauterization
- blocks (i.e. transpalatine)

- **Surgery/ interventional radiology (IR)**



Treatment

- Control of hypertension

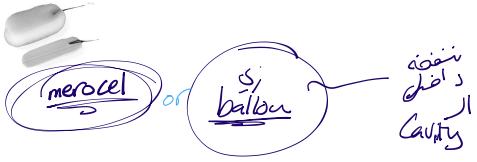
- Correction of coagulopathies/thrombocytopenia

- FFP (fresh frozen plasma) or whole blood/reversal of anticoagulant/platelets

TREATMENT

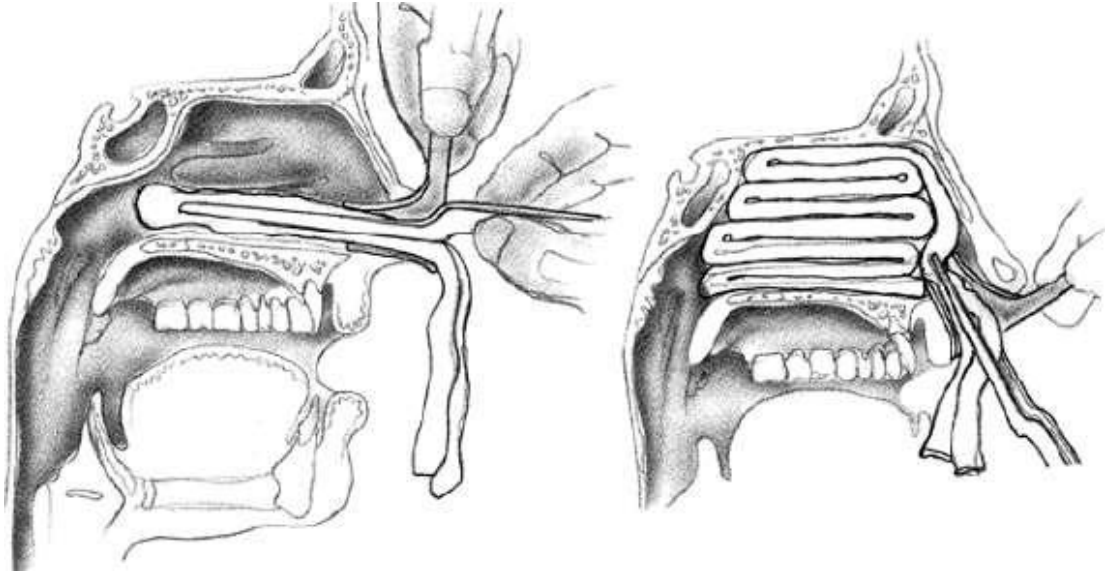
- Topical vasoconstriction: anesthetic solutions (lignocaine & pseudoephedrine or cocaine) 1% phenylephrine or 0.05% oxymetazoline

Treatment

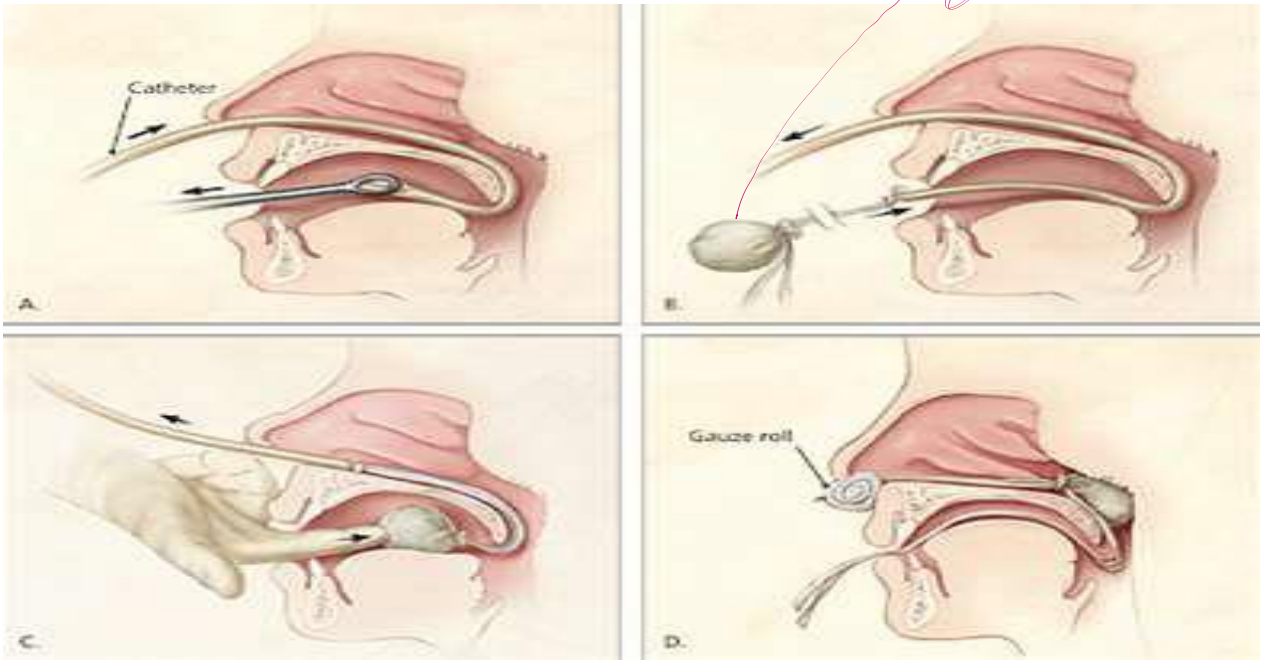


- **Nasal Packs**
- Intranasal device which applies constant local pressure
- Anterior, posterior, or anterior and posterior
- Systemic or topical antibiotics
- Precautions to admit patient with comorbidities to ICU if a posterior pack is placed
- Packs should be removed within 2-3 days of initial placement

Anterior Nasal Packs



Posterior Nasal Packs



Posterior Nasal Packs (Anterio-posterior pack)

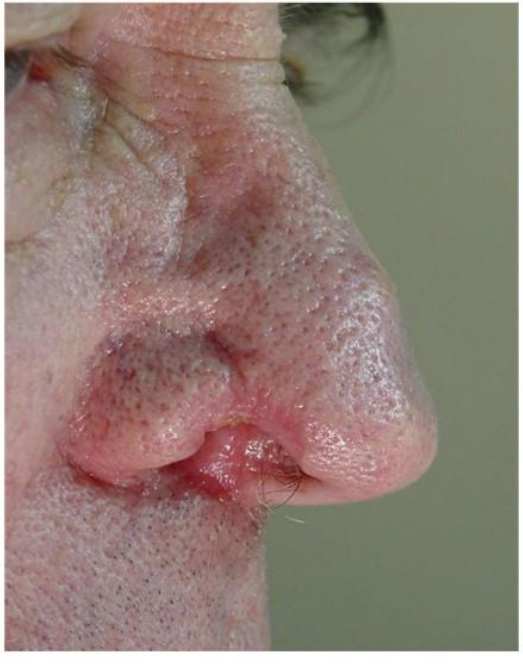
→ Alar necrosis complication

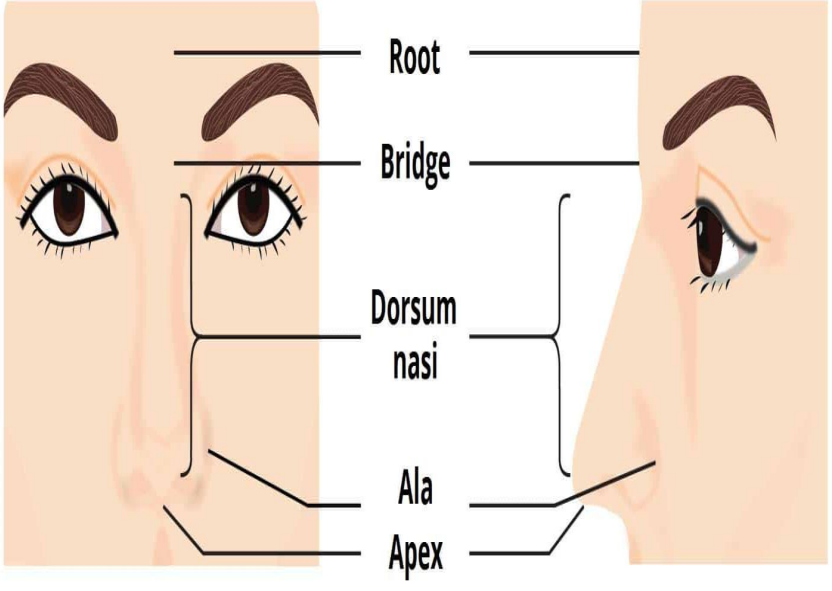
addmition in ICU

Complications of packing

- Sinusitis
- hypoxia
- Septal perforation
- Mucosal pressure necrosis
- Alar necrosis → with posterior one
- Migration of the nasal packing and aspiration into the airway or airway compromise
- Toxic shock syndrome (TTS) is caused by staphylococcal or streptococcal exotoxins → mcc of toxic Shock Syndrom

Alar necrosis from posterior pack placement





- Posterior pack admission :
  - Blood transfusion & FFP if needed
  - IVF
  - Elderly & those with chronic medical illness may need ICU admission.
  - Continuous cardiorespiratory monitoring
  - Oxygen supplment maybe needed
  - Abx for 7-10 dyays to prevent sinusitis
  - Analgesia \mild sedation
  - Deflate the balloon and remove the catheter after 48 to 72 hours

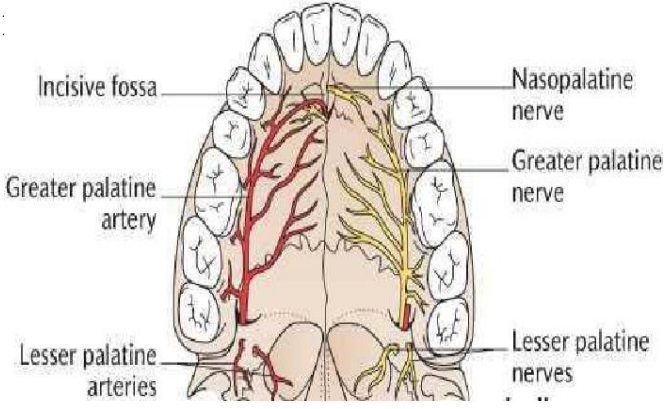
Treatment

CI : Acute infection, large area, both nares

- **Cauterization** ~ packing لو مازبط
- Chemical: Silver nitrate . → $AgNO_3$ or dechrical
- Thermal: Bipolar suction diathermy
- After cauterization advise patient to:
- Avoid nose blowing for at least 1 week
- Apply greasy antiseptic barrier ointment three times a day for 1-2 weeks

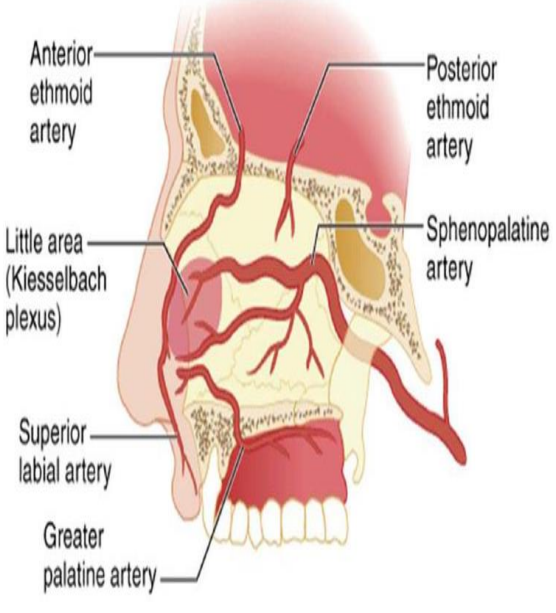
Greater Palatine Injection

- Injection into pterygopalatine fossa through greater palatine foramen
- Particularly effective for posterior epistaxis (origin of bleeding from SPA)
- infiltration a 2 ml of local anaesthetic (lignocaine) and adrenaline into the region of the greater palatine foramen to induce vasospasm



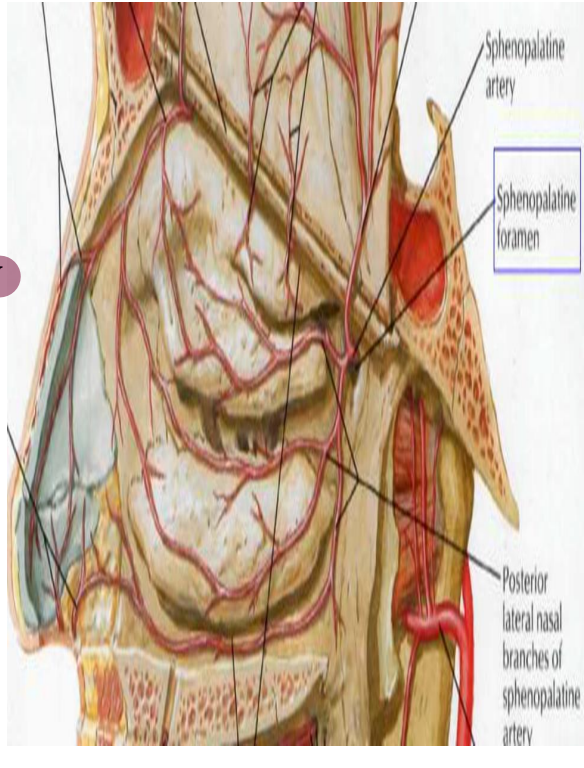
Surgery/Interventional radiology

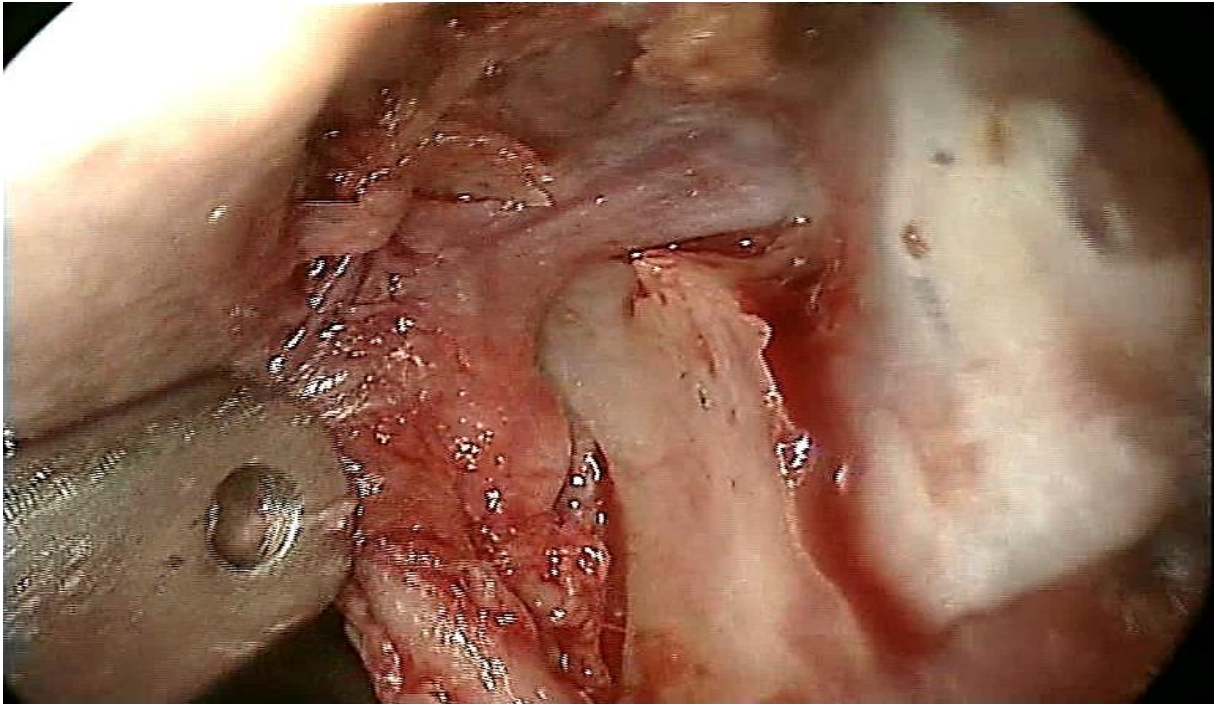
- Endoscopic sphenopalatine artery (SPA) ligation
- Anterior/posterior ethmoidal artery ligation
- Transantral ligation of IMAX (internal maxillary a.)
- Embolization of the IMAX



Endoscopic Cauterization of Sphenopalatine Artery (SPA)

- Newer Method – Endoscopic
- Older method – Caldwell-Luc approach(open)
- Allows direct cauterization of vessels and is highly effective as a second-line treatment
- Low morbidity
- Complications are rare
- Fast, not technically difficult
- Good alternative to embolization
- Highly effective – 96-100%





Anterior/Posterior Ethmoidal Artery Ligation

- Henry Goodyear performed the first anterior ethmoid artery ligation
- Can be performed externally (Lynch incision) or endoscopically

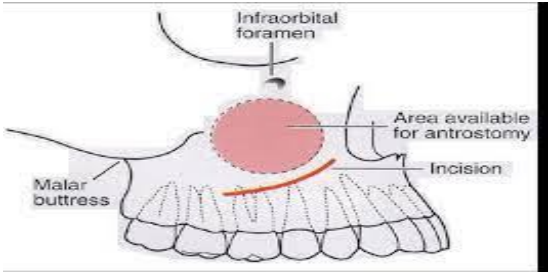
- Anterior ethmoid artery is located 24mm from the anterior lacrimal crest,
- posterior ethmoid artery is 36mm from anterior lacrimal crest

Optic N. → is 42mm

- Complications of procedure include stroke, blindness, ophthalmoplegia, and epiphora

Transantral Ligation of IMAX

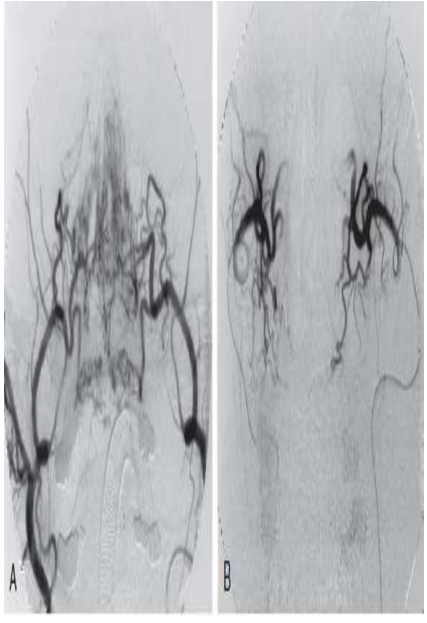
- Older method (more recently replaced by SPA ligation)
- Performed via Caldwell-Luc approach
- expose pterygopalatine fossa
- Tortuous IMAX is identified and ligated
- High failure rate 11%-20%
- High complication rate 14%-20%
- Facial paresthesia, facial pain, dental pain and numbness, hematoma, ophthalmoplegia, blindness



Embolization of IMAX

- Alternative to SPA ligation for control of posterior epistaxis
- Good for poor surgical candidates
- Back-up to unsuccessful surgical ligation

- Requires highly skilled interventional radiologist
- Complications are high (i.e. stroke, facial pain, numbness)
- Higher failure rate than surgical ligation
- Less cost effective than surgical ligation




Tracheostomy
Presented By :
Hisham Al-hamayideh
Ahmad Al-dmour
Auwid Al-shra'ah

Anatomy

- It is a mobile cartilagenous & membranous tube .
- It begins as a continuation of the larynx at the lower border of the cricoid cartilage at the level of the 6th cervical vertebra , it descends in the midline of the neck ...
- In the thorax it ends at the carina by dividing into right & left main bronchi at the level of the sternal angle opposite to the disc between T4/5
- So it extend from **C6-T4/5**.

Cont...

In adult , the trachea is about (11.5cm) in length and nearly (2.5cm) in diameter.

- In infants , the trachea is about (4 to 5 cm) in length and may be as small as 3 mm in diameter

- The blood supply of the trachea is from the **inferior thyroid vessels(upper2/3) & bronchial arteries(lower1/3)** , and its lymphatic drainage is to **paratracheal ,pretrachial , deep cervical lymph nodes.**

Relations

- Lying partly in the neck and partly in the thorax, its **Cervical** relations are:
  - **Anteriorly**— the isthmus of thyroid gland, inferior thyroid veins, sternohyoid and sternothyroid muscles.
  - **Laterally** —the lobes of thyroid gland and the common carotid artery.
  - **Posteriorly** —the oesophagus with the recurrent laryngeal nerve lying in the groove between oesophagus and trachea.

Cont…

- The patency of the trachea is maintained by a series of 15–20 U-shaped cartilages.

Tracheostomy

- is an operative procedure that creates a surgical airway in the cervical trachea just below the larynx through which an indwelling tube is placed thus an artificial airway is created , it provides an air passage to help the pt breathe when the usual route for breathing is obstructed or impaired.

Types

(Temporary Vs. permanent)

Temporary tracheostomy:

This can be removed **when the patient recovers.**

→the upper airway will remain connected to the lower airway if the tracheostomy tube were to be dislodged

Types

(Temporary Vs. permanent)

Permanent tracheostomy:

- This stoma is kept open by the rigidity of the tracheal cartilage. The patient will breathe through this stoma **for the remainder of his/her life.**
- As a result, there is **no connection** between the nasal passages and the trachea.

→It is most often performed in patients who have had difficulty weaning off a ventilator, followed by those who have suffered trauma or a catastrophic neurologic insult

One of the most indection for permanent tracheostomy is total laryngectomy

Indications

- ✓ Acute indications.
- ✓ Chronic indications.
- ✓ Elective setting.

Acute indications

- ✓Maxillofacial injuries.
- ✓Poisoning
- ✓Upper airway obstruction.
- ✓ acute angioedema and inflammation of the head and neck

Upper Airway Obstruction

Cause	Examples
Congenital	Subglottic or upper tracheal stenosis, laryngeal web, laryngeal and vallecular cysts, tracheo-oesophageal anomalies or haemangioma of the larynx.
Infective	Acute epiglottitis, laryngotracheobronchitis, diphtheria or Ludwig's angina.
Malignancy	Advanced tumours of larynx, tongue, pharynx or upper trachea presenting with stridor.
Trauma	Gunshot and knife wounds to the neck, inhalation of steam or smoke, swallowing of corrosive fluid.
Vocal cord paralysis	Post-op complication of thyroidectomy, cardiac or oesophageal surgery, bulbar palsy.
Foreign body	Swallowed or inhaled object lodged in upper airway causing stridor.

Chronic indications

* prolonged intubation → most common cause of tracheostomy

1. Pulmonary Ventilation

- Tracheostomy should be performed in a patient still requiring ventilation through an endotracheal tube for more than tow week.
- Tetanus; Brain-stem stroke , Coma

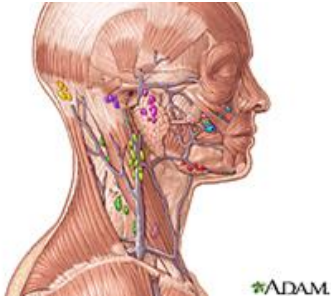
Cont…

2. Pulmonary Toilet

- Those who cannot cough and clear their chest.
- Prevent aspiration by low pressure high volume cuff tracheostomy tube.
- Any condition causing pharyngeal or laryngeal incompetence may allow aspiration of food, saliva, blood or gastric contents.
- In respiratory failure due to central depression of res. Center (Polyneuritis (e.g. Guillain–Barré syndrome);Bulbar poliomyelitis;Multiple sclerosis;)
- Myasthenia gravis

Elective setting

- In head and neck operations.

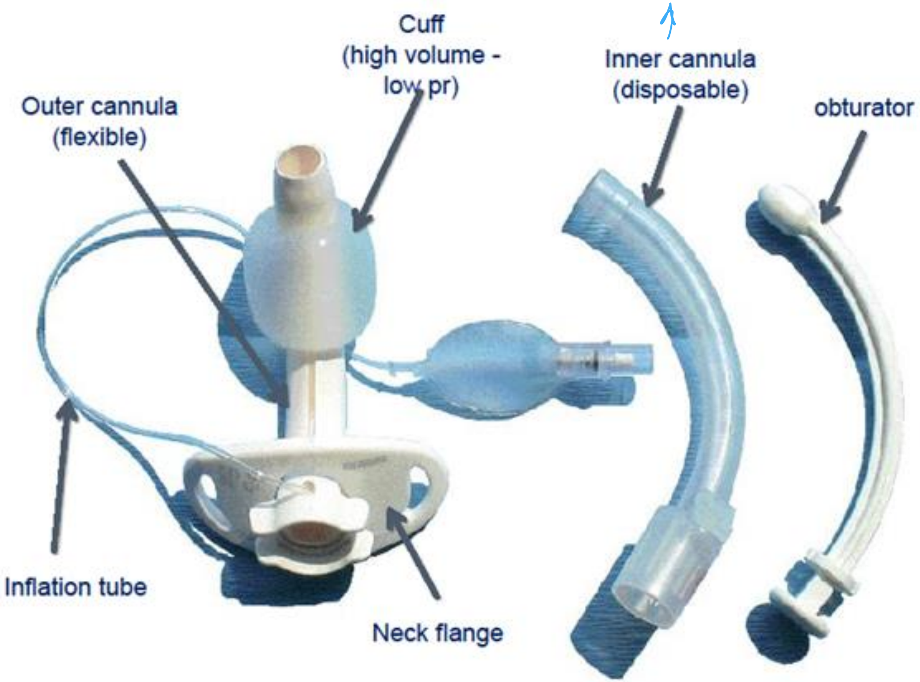


Tracheostomy tube

bilumen

mc use

single lumen



Tracheostomy tubes

Types:

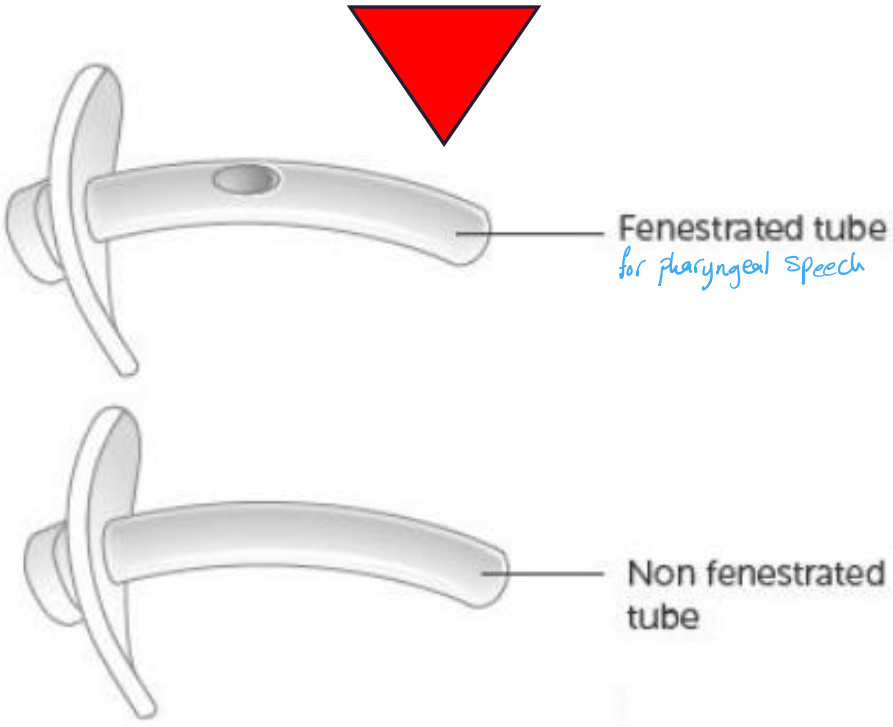
- Material
  - Metal (Silver-Stainless steel)
  - Plastic (Polyvinyl chloride)
  - Silicone
- Cuffed or Uncuffed
- Single or double tubes
- Fenestrated or unfenestrated tubes

Silver tube

Pediatric silicone tube

Cuffed tube

Uncuffed tube


Fenestrated tube
for pharyngeal speech
Non fenestrated tube

Obturator - The obturator is only used during insertion of the tracheostomy tube. It replaces the inner cannula during insertion. **Must always be present at patient bedside in case of accidental decannulation.**

Cuff – Is the balloon around the outer cannula that is inflated to maintain a seal around the tube.
** **Note: not all trachs have cuffs.**

Procedures

- Percutaneous tracheostomy
- Surgical tracheostomy
- Cricothiroidectomy

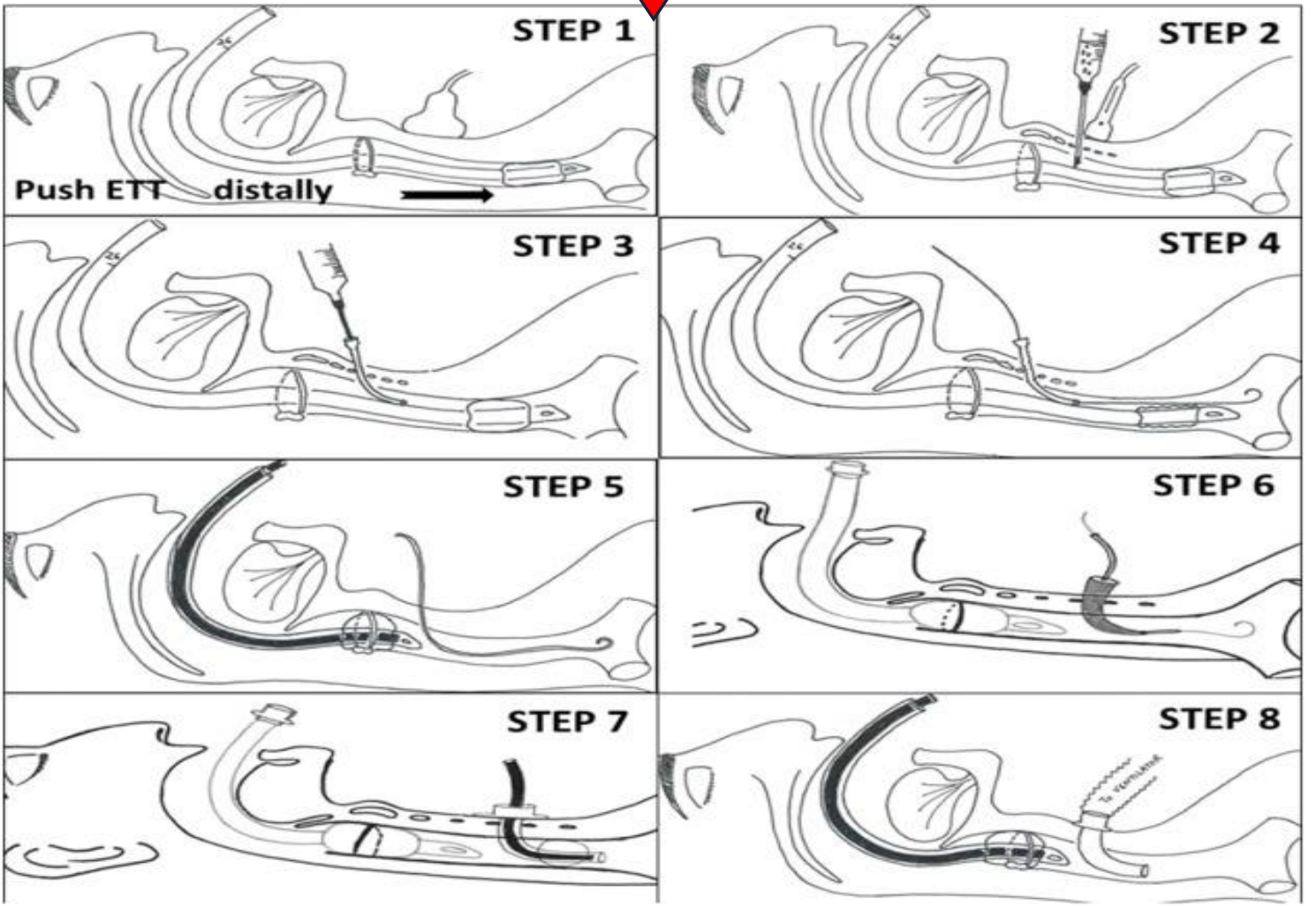
Percutaneous procedure

*by ICU. Dr
* Pt → supine position
*site of incision 2fingers above notch

- Curvi-linear (oblique) skin incision along relaxed skin tension lines between sternal notch and cricoid cartilage.
- Midline blunt dissection down to the trachea.
- Insertion of 14-gauge plastic cannula and needle with fluid filled syringe attached into trachea. Aspiration of air confirms correct placement of the tip in the trachea.
- Removal of needle leaving cannula in place.
- Insertion of soft tipped guide wire into trachea through cannula.
- Removal of cannula leaving guide wire in place.
- Tracheal dilatation
- Insert tracheostomy tube (with concomitant withdrawal of endotracheal tube), inflate cuff, secure with tape around neck or stay sutures.
- Connect ventilator tubing.

بشوف bubbles ← Airway مجرد دخولها

First step is infiltration of local anesthesia


STEP 1
Push ETT distally
STEP 2
STEP 3
STEP 4
STEP 5
STEP 6
STEP 7
STEP 8

- **The advantages of percutaneous tracheostomy (PT) over surgical tracheostomy (ST) are primarily related to :**

- ICU , Bed Side Tracheostomy
- Use of guide wire and Dilators
- Under the vision of Bronchoscope through endotracheal tube
- Less time ,Less Expensive , reduced tissue trauma
- Not suitable for thick neck and in emergency

Contraindications include :

- ***1 .unstable cervical spine***

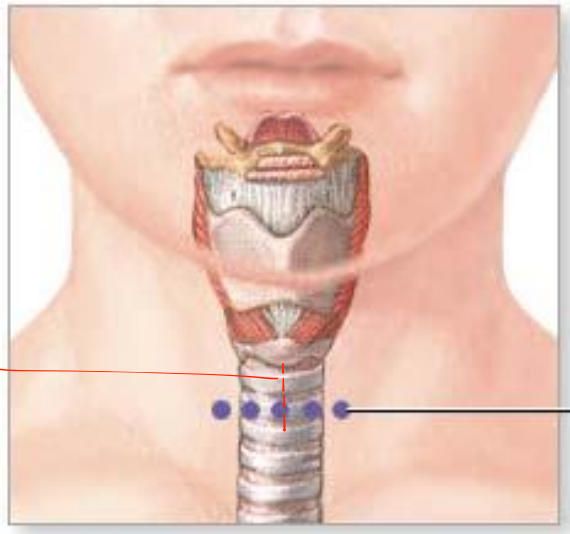
- ***2.inability to identify anatomic landmarks (in obese , thick neck)***

- ***3.refractory coagulopathy*** continued bleeding from multiple sites despite ongoing blood component administration, Grade III or higher

Surgical Procedure

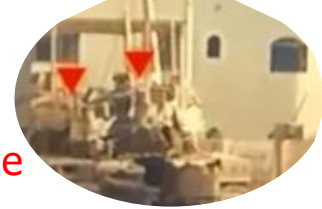
- ✓ Curvi-linear skin incision along relaxed skin tension line (RSTL) between sternal notch & cricoid cartilage.
- ✓ Midline vertical incision from the region of the cricothyroid membrane inferiorly toward the suprasternal notch with dividing strap muscles.
- ✓ Division (or retraction) of thyroid isthmus inferiorly.
- ✓ Divide the 2nd tracheal ring & insert tracheostomy tube (with concomitant withdrawal of ETT), inflate the cuff, then secure with tape or sutures.(**don't do the incision at first ring <to prevent the stenosis)**
- ✓ Connect ventilator tubing.

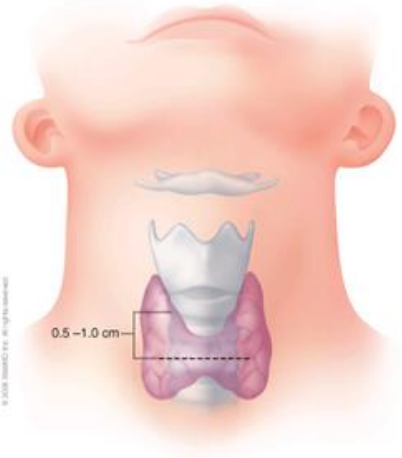
- ❖ Note: the procedure is peformed **under GA with endotracheal intubation.**



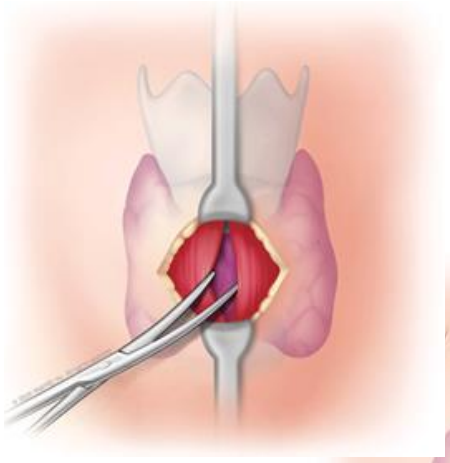
A.D.A.M.

*incision & excision — adults
only — children

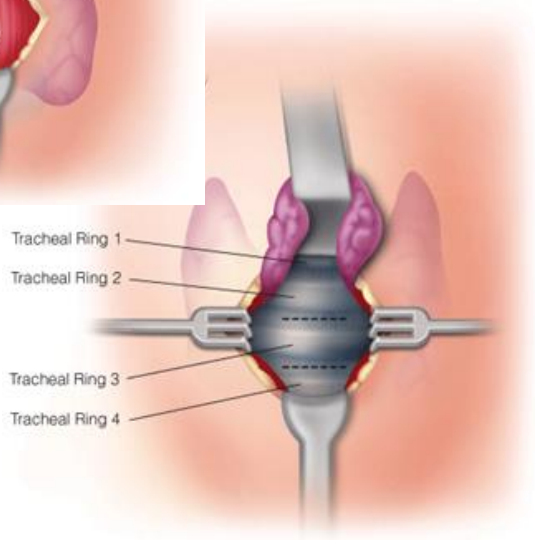




Incision 1 cm below the cricoid or halfway between the cricoid and the sternal notch.

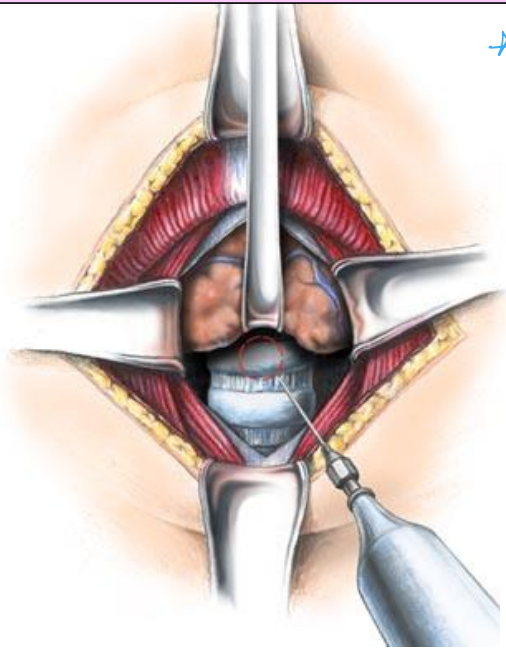


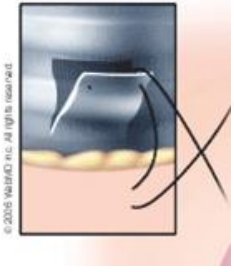
Retractors are placed, the skin is retracted, and the strap muscles are visualized in the midline. The muscles are divided along the raphe, then retracted laterally

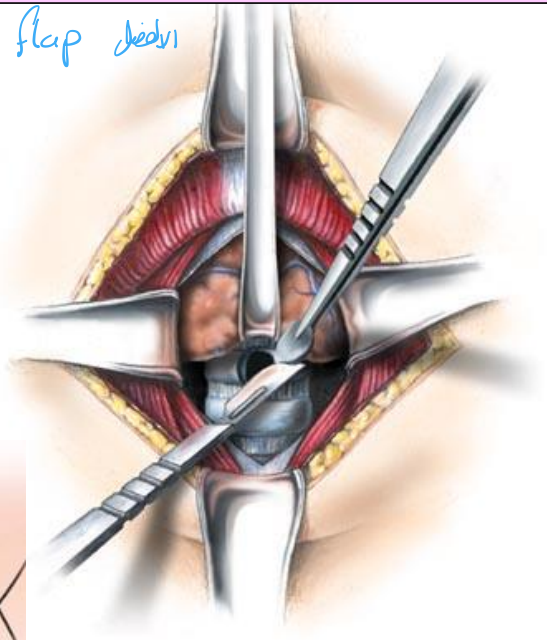


The thyroid isthmus lies in the field of the dissection.
Typically, the isthmus is 5 to 10 mm in its vertical dimension,
mobilize it away from the trachea and retract it,
then place the tracheal incision in the second or third tracheal interspace

* inverted U flap







Pediatric Tracheostomy

*To allow the cartilage to growth

To allow the cartilage to grow

- Vertical incision in trachea BTW 2nd and 3rd ring.
- No excision of ant. Wall of trachea
- Secure the tube with neck by two sutures

cricothyroidotomy

Cricothyroidectomy (mini -tracheostomy)

- It is an emergency incision through the skin & cricothyroid membrane to secure pt's airway during an emergency situation.

- It is only a temporary airway for life-saving situations. It is not suitable for prolonged ventilation due to its small size. A definitive airway (ETT or tracheostomy) must be performed later in hospital for adequate ventilation.

Indications

* Severe facial or nasal injuries that don't allow oral or nasal intubation.
  - ✓Massive midfacial trauma.
  - ✓Possible cervical spine trauma preventing adequate ventilation.
  - ✓Anaphylaxis.
  - ✓Chemical inhalation injuries.

Contra-indications

- ✓Inability to identify landmarks (cricothyroid membrane)
- ✓Underlying anatomical abnormality (tumor)
- ✓Tracheal transection.
- ✓Acute laryngeal disease due to infection or trauma.
- ✓Small children >10 years.
- ✓Patients who have an increased risk of subglottic stenosis, those with inflammatory process in near proximity, epiglottitis and bleeding diathesis.
- ✓anemia

Procedure

- ✓ With a scalpel, create a 1 cm transverse incision through the cricothyroid membrane.
- ✓ Open the hole by inserting the scalpel handle into the wound & rotating 90° or by using a clamp.
- ✓ Insert a 6 or 7 mm internal diameter tracheostomy tube or ETT.
- ✓ Inflate the cuff &secure the tube

- ✓ Provide ventilation via a bag-valve device with the highest available concentration of oxygen.

Rapid four-step technique

the only equipment needed is a #20 scalpel, a tracheal hook with a large radius, and a cuffed tracheostomy tube. The 4 steps are: 1) palpation, 2) stab incision, 3) inferior traction, and 4) tube insertion. This streamlined method is

Step 1: Identify the CTM by palpation

Standard cricothyrotomy: Step 1

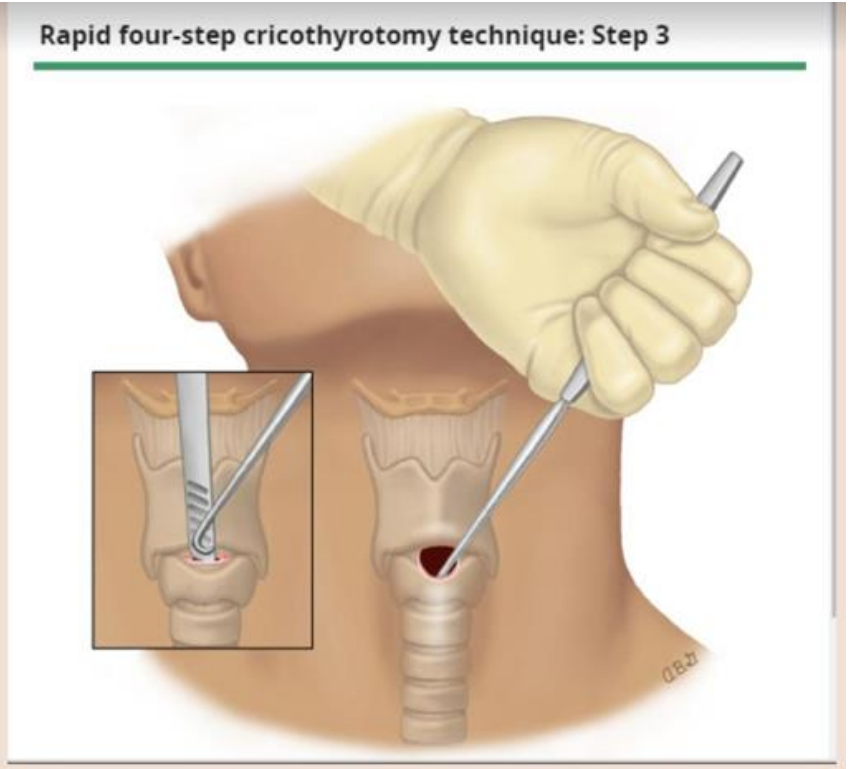
Immobilize the larynx and palpate the cricothyroid membrane with the index finger of the nondominant hand.

Step 2: **Make a single horizontal stab incision through the skin, subcutaneous tissue, and CTM** with the scalpel

The size of the skin incision is approximately 3 cm

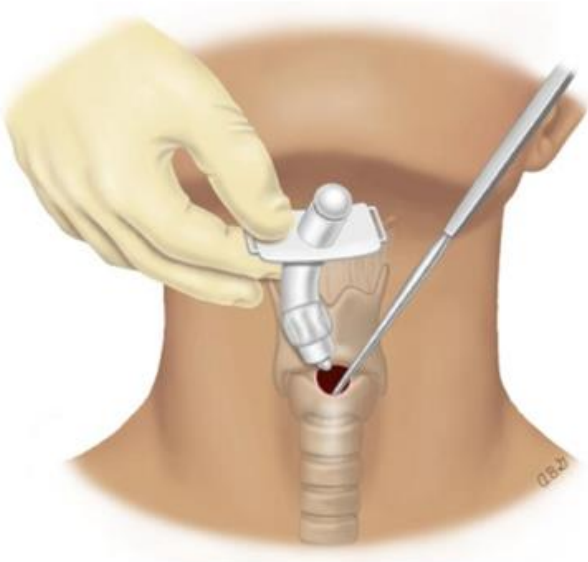
Step 3: Place the hook –
Prior to removal of the scalpel, the hook is placed and directed inferiorly to engage the cricoid cartilage. Caudal traction is used to stabilize the larynx

This marks a significant change from the standard method, in which the tracheal hook is placed under the thyroid cartilage. Also in contrast with the standard technique, this step does not require an assistant to manage the hook



Step 4: Insert the tracheostomy tube into the trachea

Rapid four-step cricothyrotomy technique: Step 4



Postoperative care of tracheostomy

- A **chest x ray** is often taken, especially in children, to check whether the tube has become displaced or if complications have occurred.
- **Antibiotics** may prescribed to reduce the risk of infection.
- If the patient can breathe without a ventilator, the **room is humidified**; otherwise, if the tracheotomy tube is to remain in place, the air entering the tube from a ventilator is humidified.
- **Swallowing** : Evaluate the patient's risk of aspiration before feeding begins.

- **Position**
  - Adult patients in the postoperative period should usually be sitting well propped up; care must be taken in infants that the chin does not occlude the tracheostomy and the neck should be extended slightly.

*** Tube changing**

Tube changing should be avoided if possible for 2 or 3 days, after which the track should be well established and the tube can be changed easily.

Cuffed tubes need particular attention, with regular deflation of the cuff to prevent pressure necrosis.

The amount of air in the cuff should be the minimum required to prevent an air leak.

Closure of tracheostomy

Remain closure of tube for 24/48 h >and lock for o2 sat

- Before we close it we must ensure that there's adequate breathing in the absence of tracheostomy.
- If the tracheostomy is temporary, the tube will eventually be removed.
- Healing will occur quickly, leaving a minimal scar.
- Occasionally a stricture, or tightening, of the trachea may develop, which may affect breathing.
- If the tracheostomy tube is permanent, the hole remains open and further surgery may be needed to widen the opening, which narrows with time.

Tube Occlusion

- Signs of tube occlusion include:

- Difficult or laboured breathing
- Use of accessory muscles
- None or limited expired air from tracheostomy tube
- Pale/Cyanosed skin color
- Anxiety
- Increase Pulse and Respiratory Rate
- Clamminess مرخب
- Cessation of respiration

Complications of Tracheostomy

- Complications 5-40%
- Mortality <2%

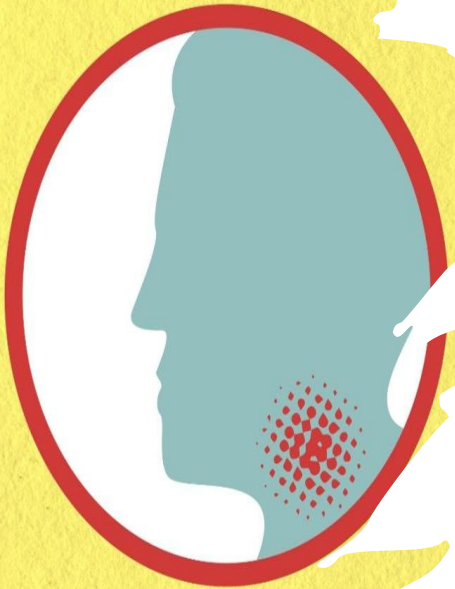
- Complications are more frequent in emergency situations, severely ill patients

Surgical Complications

Stage	Complication
Intra-operative	Haemorrhage Airway fire Injury to trachea and larynx Injury to paratracheal structures Air embolism Apnoea Cardiac arrest
Early post-operative	Subcutaneous emphysema Pneumothorax/pneumomediastinum Tube displacement Tube blockage (crusts) Wound infection Tracheal necrosis Secondary haemorrhage Swallowing problems
Late post-operative	Haemorrhage Granuloma formation Tracheo-oesophageal fistula Difficult decannulation Tracheocutaneous fistula Laryngotracheal stenosis Tracheostomy scar

- Children, post head trauma, burn pts, & seriously debilitated pts (منهك) are more susceptible to complications.

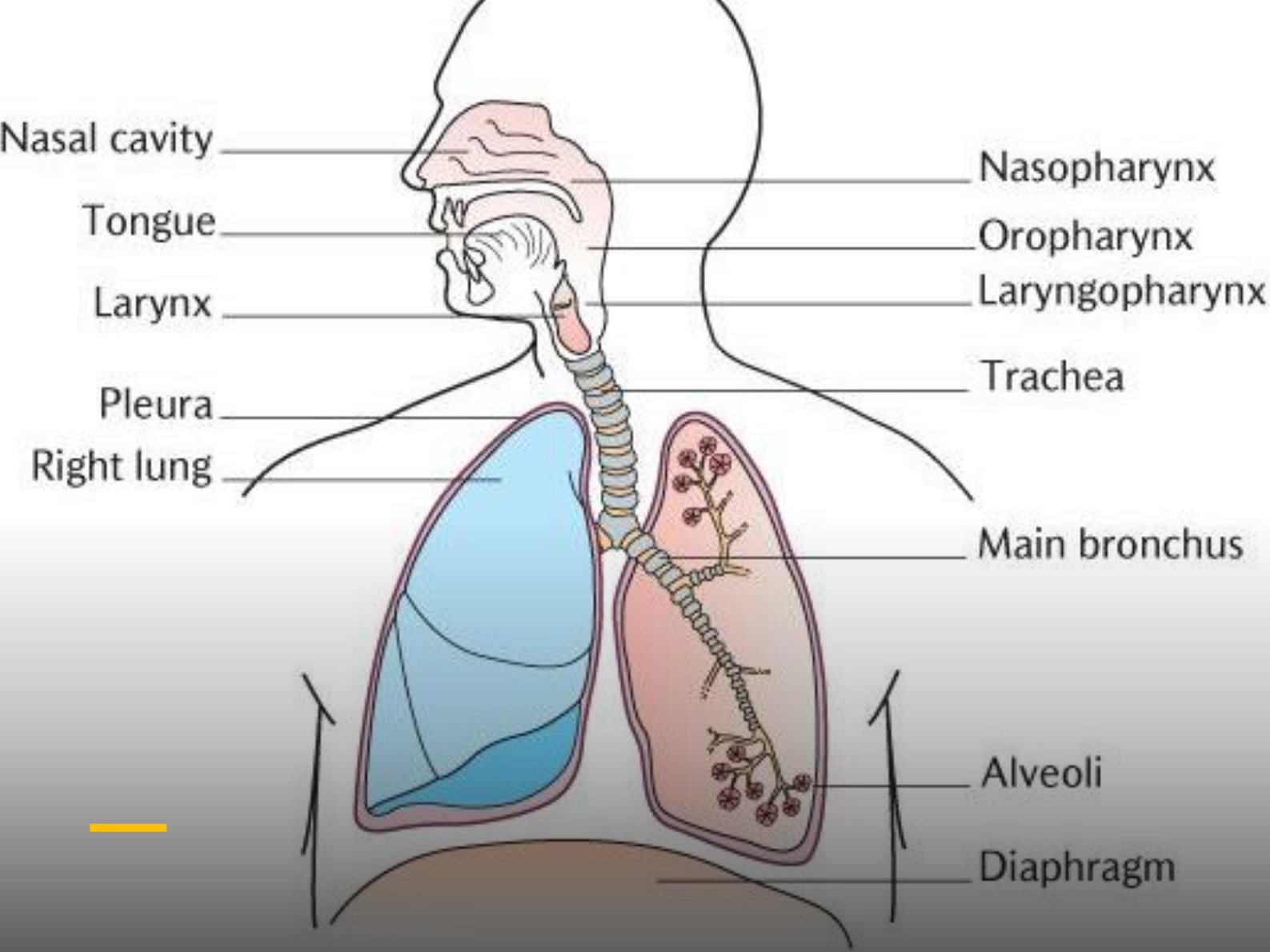
- The most common complications are hemorrhage (1), followed by tube obstruction (2), then tube dislodgement (3).

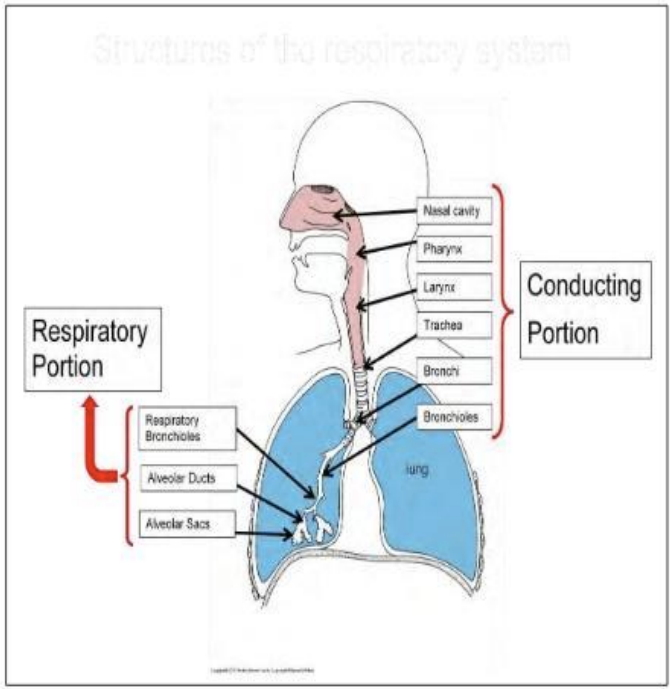


Stridor and Hoarseness of voice

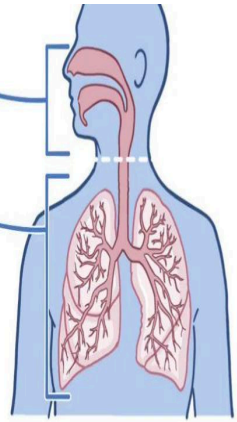
Done by :
Tala Sarayreh
Adan Mohammad
Format Thunibat

Supervised by :
Dr. Osama Btoosh


Nasal cavity
Tongue
Larynx
Pleura
Right lung
Nasopharynx
Oropharynx
Laryngopharynx
Trachea
Main bronchus
Alveoli
Diaphragm



* TWO REGIONS
  - UPPER RESPIRATORY TRACT
    - NOSE, NASAL CAVITY, ORAL CAVITY, PHARYNX, LARYNX, & UPPER TRACHEA
  - LOWER RESPIRATORY TRACT
    - LOWER TRACHEA, BRONCHI, BRONCHIOLES, & LUNGS

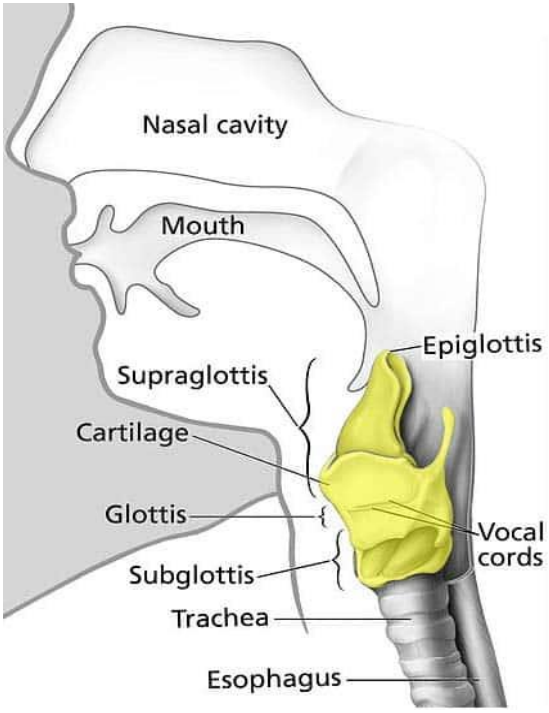


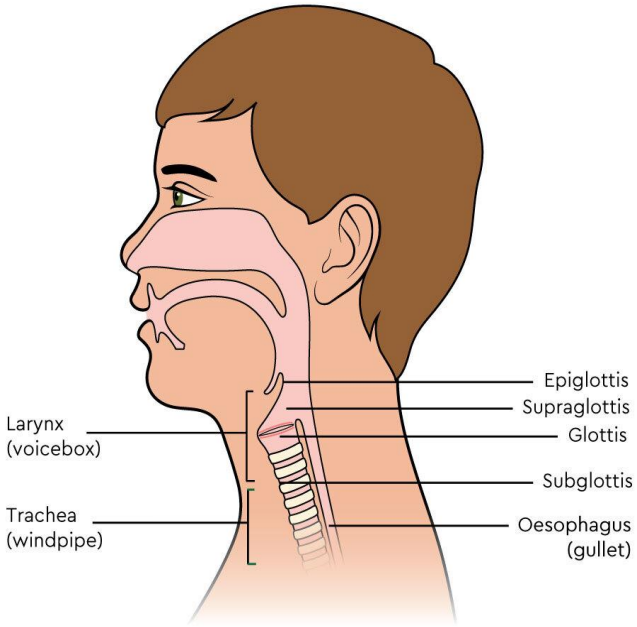
Larynx

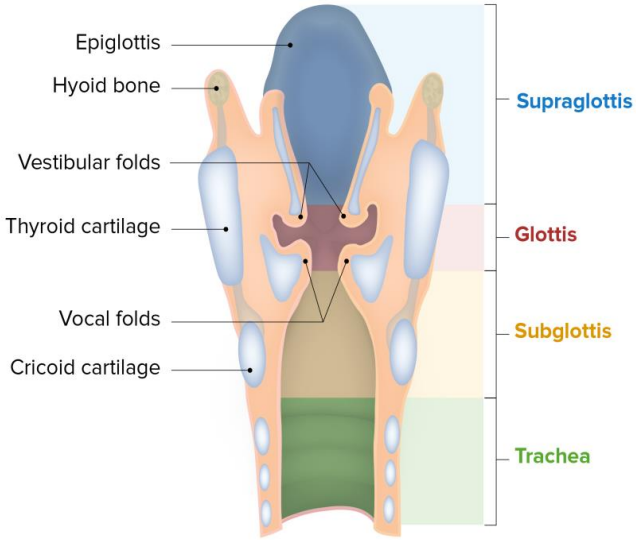
- At the beginning of trachea
- Its beginning is guarded by epiglottis (has elastic cartilage)
- Has 2 functions :

✓production of voice (vocal cords)

✓Prevent food & fluid from entering the Trachea through the (epiglottis)




Epiglottis
Supraglottis
Glottis
Subglottis
Oesophagus
(gullet)
Larynx
(voicebox)
Trachea
(windpipe)


Epiglottis
Hyoid bone
Vestibular folds
Thyroid cartilage
Vocal folds
Cricoid cartilage
Supraglottis
Glottis
Subglottis
Trachea

Vocal cords

● 2 pairs of **mucosal folds** extend in lumen of larynx

1. False vocal cords (Upper pair)

. Called (vestibular folds)

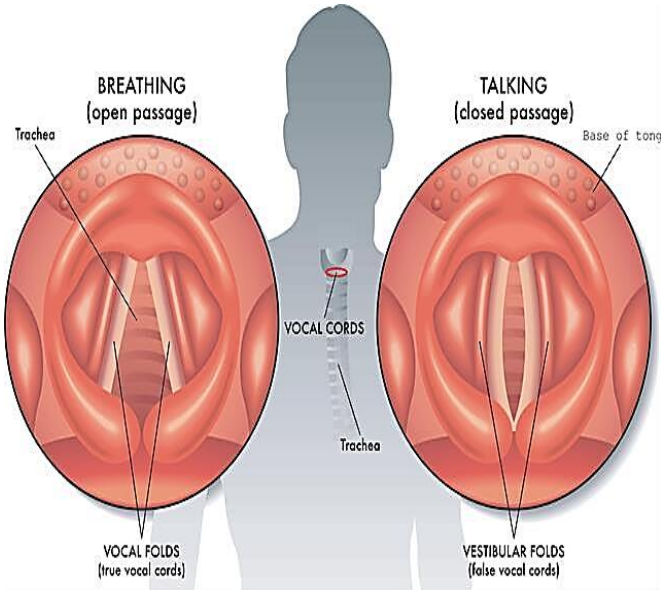
. Made of vestibular ligament

. Guard against entrance of food into larynx

2. True vocal cords (Lower pair)

. Made of vocal ligaments & skeletal (Vocalis Muscles)

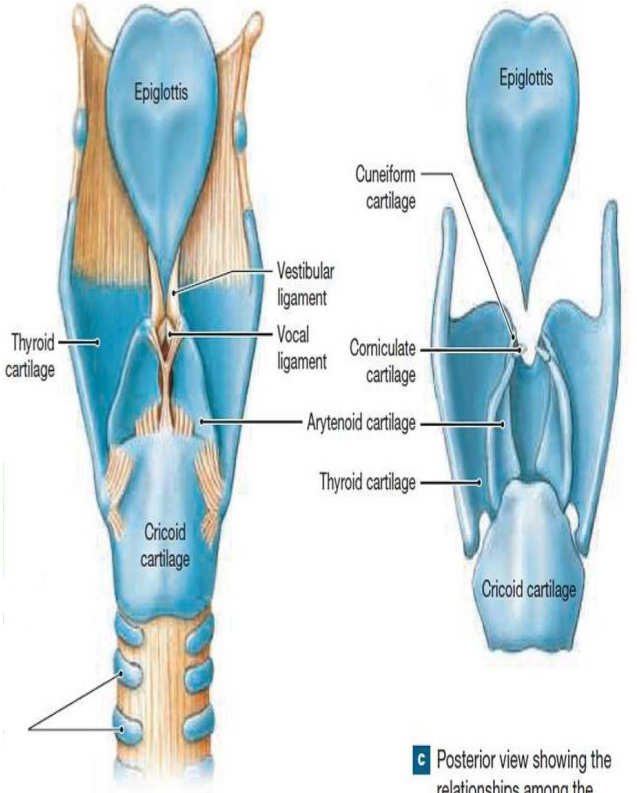
. Tension of cords & distance between them produce sound



vocal cords in
open position

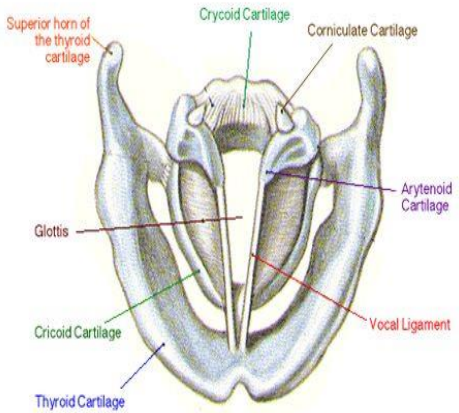
vocal cords in
closed position

vocal cords attempting
closed position
(with one sided palsy)



b Posterior view of the intact larynx

c Posterior view showing the relationships among the individual laryngeal cartilages



The Larynx: viewed from above

STRIDOR

- is an **abnormal**, **high-pitched sound**
- produced by turbulent airflow through a **partially obstructed airway** at the level of the supraglottis, glottis, subglottis, or trachea.

- Any obstruction **above** those levels will cause a **stertor** sound and obstruction at the level of the bronchi or below will cause a **wheeze.**

- Stridor is **a symptom, not a diagnosis** or a disease, and the underlying cause must be determined.

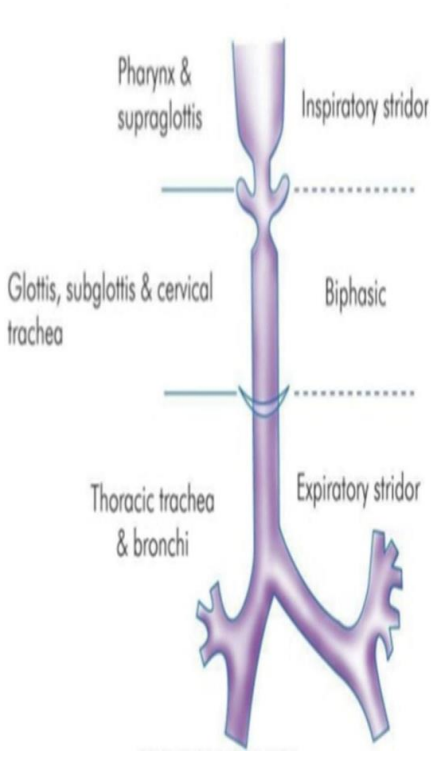
- **A hoarse voice refers to a weak or altered voice.**

Stridor may be inspiratory (most common), expiratory, or biphasic.

Each suggest different causes, as follows:

- **Inspiratory stridor:** supraglottic obstruction
- **Expiratory stridor**: implies tracheobronchial obstruction
- **Biphasic stridor:** suggests a subglottic or glottic anomaly.

- Stridor is mainly classified to **acute** and **chronic** according to the <u>onset</u> and <u>severity</u> of the stridor



CLINICAL PRESENTATION

History

- The **most common** presenting symptom is **loud, raspy, noisy breathing…**

- Age of onset, duration, severity, and progression of the stridor.
- Precipitating events (eg: crying or feeding)
- Positioning (eg: prone, supine, or sitting)
- **Presence** of **aphonia**.
- Other associated symptoms (eg: paroxysms of cough, aspiration, difficulty in feeding, drooling, or sleep-disordered breathing)
- History of color change (cyanosis)

Examination

- On initial presentation, especially if the symptoms are of acute onset, the child should immediately be assessed for severity of stridor and respiratory compromise.

- Special attention should be paid to the following:

- Level of consciousness
- Responsiveness
- Heart and respiratory rates
- Cyanosis
- Use of accessory muscles of respiration
- Nasal flaring , cough , ..

- The patient may prefer certain **positions that alleviate the stridor**.
- It is important to observe the character of the cough, cry, and voice.
- The presence of **fever** and toxicity generally implies serious bacterial infections.

- **Note**: Any form of airway examination should be **avoided** in cases of suspected epiglottitis or croup, as can predispose to **sudden airway closure**

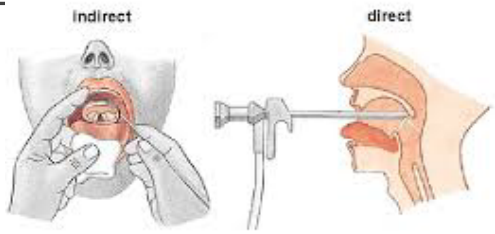
INVESTIGATIONS

- On **initial evaluation**, pulse oximetry may be useful to determine the extent and severity of the stridor and respiratory compromise.

- **Fibreoptic nasal endoscopy**

A quick and minimally invasive method to differentiate where the pathology lies.

- **Note**: Fibreoptic nasal endoscopy is **contraindicated** in patient suspected of croup or epiglottitis.



- **Indirect laryngoscopy**

For local laryngeal causes

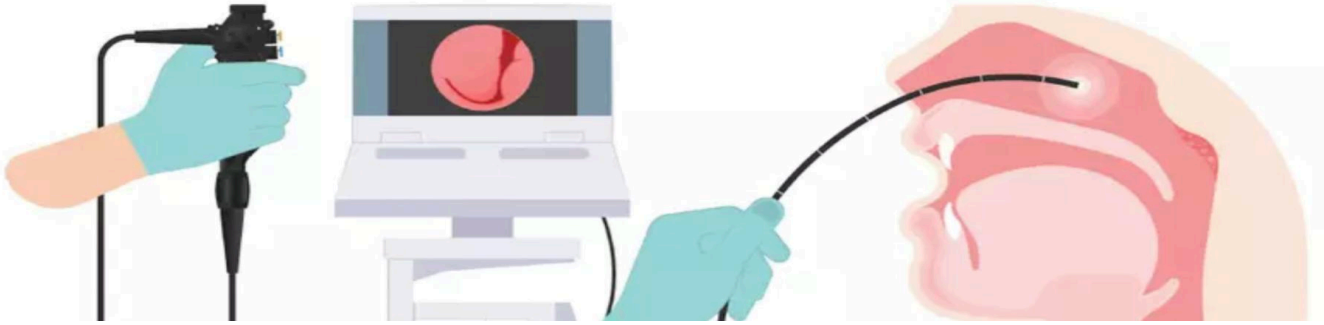
- **Bronchoscopy**

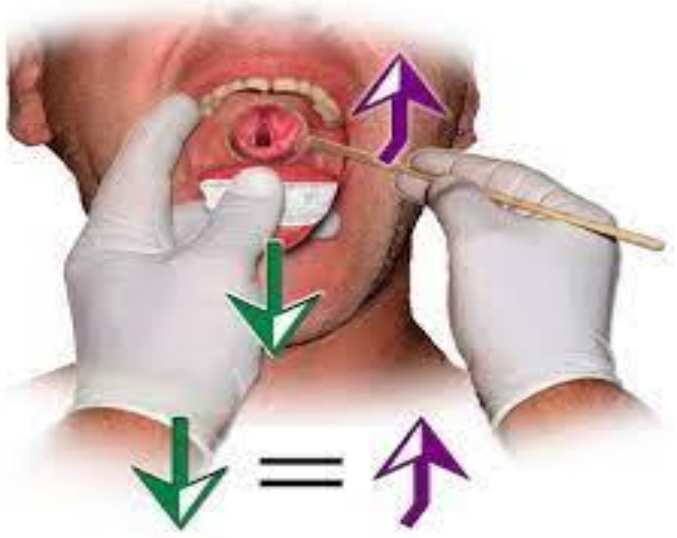
Bronchoscopy can be used if visualisation below the vocal cords is warranted, such as suspected subglottic stenosis

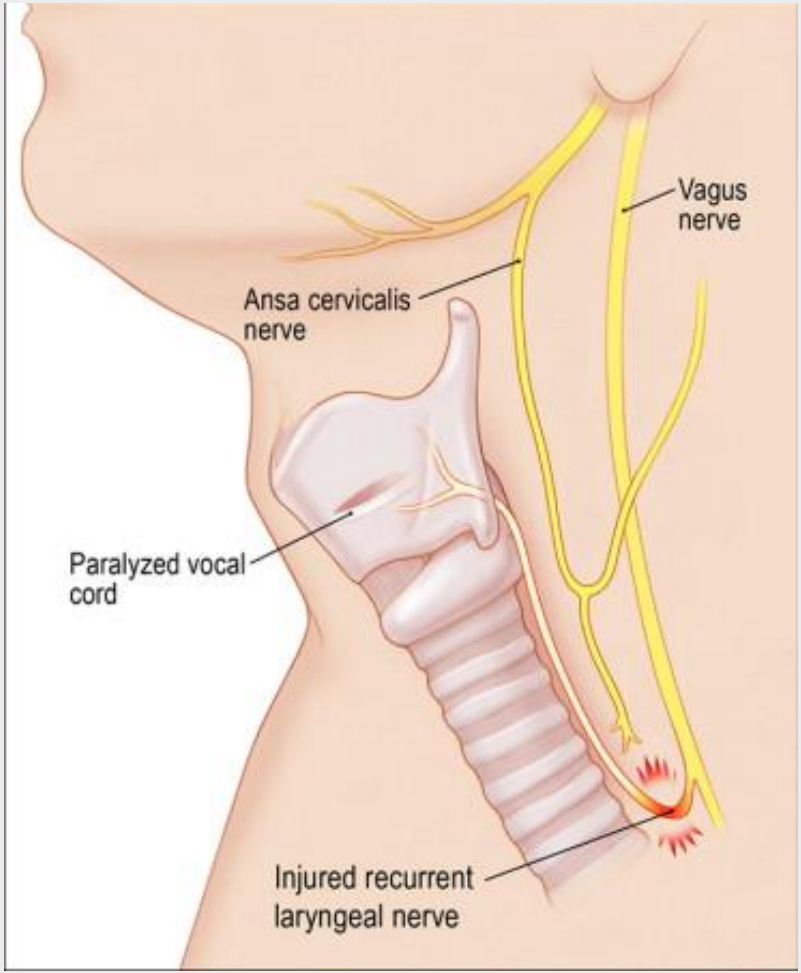
- **CT Scan**

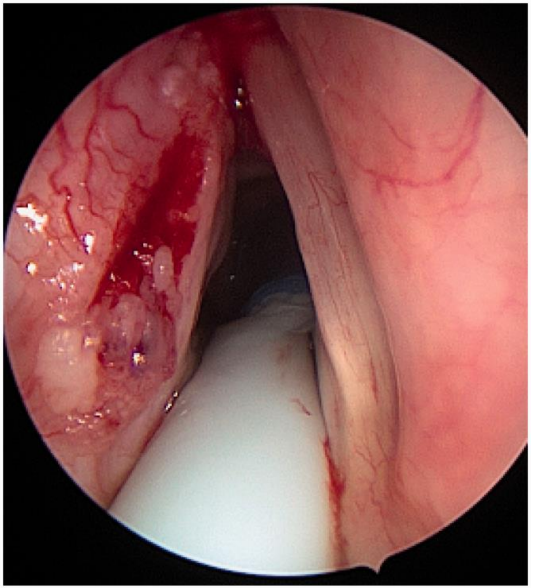
Used in the case of abscess or malignancy.

Nasal Endoscopy: Procedure and Its Importance






Vagus nerve
Ansa cervicalis nerve
Paralyzed vocal cord
Injured recurrent laryngeal nerve



ACUTE STRIDOR

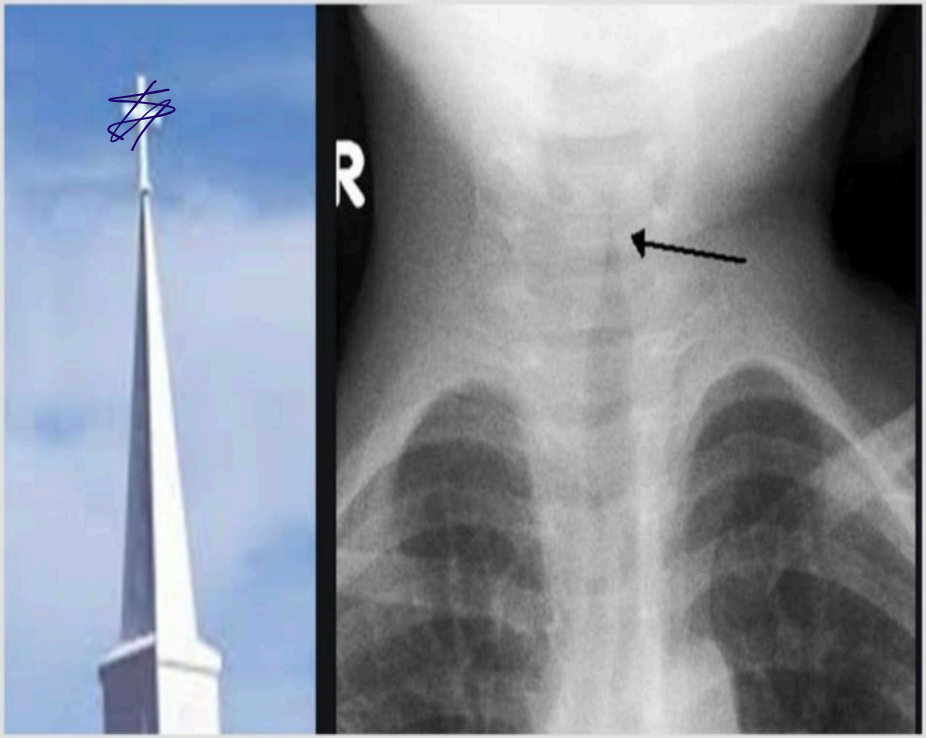
Laryngotracheobronchitis (Croup)
Epiglottitis
Foreign body inhalation
Anaphylaxis
Neck space abscess
Laryngitis

Laryngotracheobronchitis (CROUP)

- It is inflammation of the larynx, trachea, and bronchus, including the vocal cords.
- Croup is the most common cause of acute stridor in children aged 6 months to 2 years.
- The cause of croup in 95% of cases is viral infection, common organisms including parainfluenza, influenza and rhinovirus
- It is typically preceded by an upper respiratory infection, before developing into dyspnea and a characteristic barking cough, with potential fever. Symptoms are usually worse at night.
- Most cases do not require any investigations and can be made as a clinical diagnosis.

Laryngotracheobronchitis (CROUP)

- The steeple sign is a radiologic sign found on an AP neck radiograph where subglottic tracheal narrowing produces the shape of a **church steeple** within the trachea itself



Laryngotracheobronchitis (CROUP)

Management

- all children with croup should receive single dose of oral dexamethasone (0.15mg/kg) to reduce the inflammation and symptoms.
- Pain and fever can be controlled with paracetamol and ibuprofen, as needed.
- In the presence of any of the following signs, hospital admission should be considered:
  - Known structural upper airway obstruction, a history of severe croup, or immunocompromise
  - Uncertain diagnosis
  - An unwell child with inadequate oral intake
  - Less than 6 months old
- In hospital, inhaled corticosteroids can be given, alongside nebulized adrenaline, to decrease airway inflammation. In severe cases, intubation may be warranted

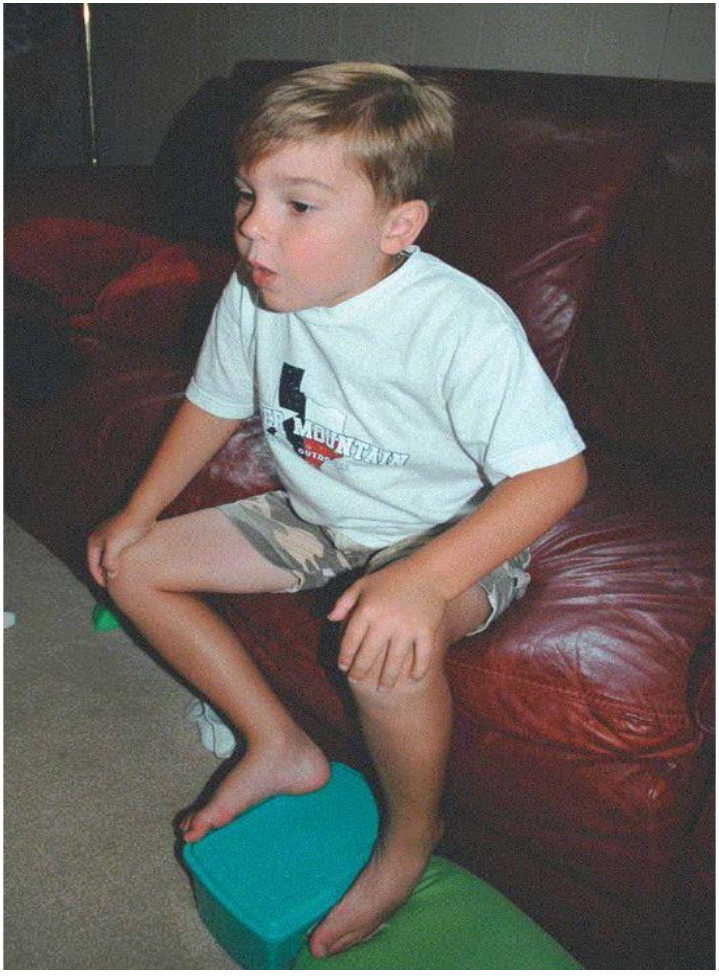
Epiglottitis

- Most commonly caused by H. Influenzae type B infections.
- Typically affects children between 2-7 years.
- Patients will initially present with a sore throat, a fever, and dyspnea, characteristically in the absence of a cough. Late signs of the condition if left untreated include drooling, dysphagia, and stridor.
- Hot potato voice
- The patient will look unwell and is classically seen, in late stages on the disease, sitting in the tripod position (to allow gravity to assist in keeping the airway open).

Epiglottitis

Investigations and management

- All cases of epiglottitis need urgent assessment by senior anesthetist or ENT surgeon, in a HDU or ICU setting (the airway should not be examined without such support due to the high risk of airway narrowing).
- Nebulized adrenaline and IV dexamethasone should be started in all suspected cases.
- Blood and throat cultures should be taken and IV broad-spectrum antibiotics should be given, alongside analgesia and IV fluids.



Tripod position

Epiglottitis

Epiglottitis

- The thumb sign in epiglottitis is a manifestation of an edematous and enlarged epiglottis which is seen on lateral soft-tissue radiograph of the neck, and it suggests a diagnosis of acute infectious epiglottitis.

Management of acute stridor

For acute causes that require urgent management, initial steps should involve:

- Stabilize the patient, start high-flow oxygen, and alert suitable senior specialists (ENT and / or anesthetist)
- Try to suction secretions or clear any foreign body from airway if obvious or visible
- Give adrenaline or steroids (IV or inhaled), as necessary
- Take bloods, including an ABG or cultures if indicated
- In emergency situations, be prepared to perform or assist with an emergency tracheostomy or intubation

CHRONIC STRIDOR

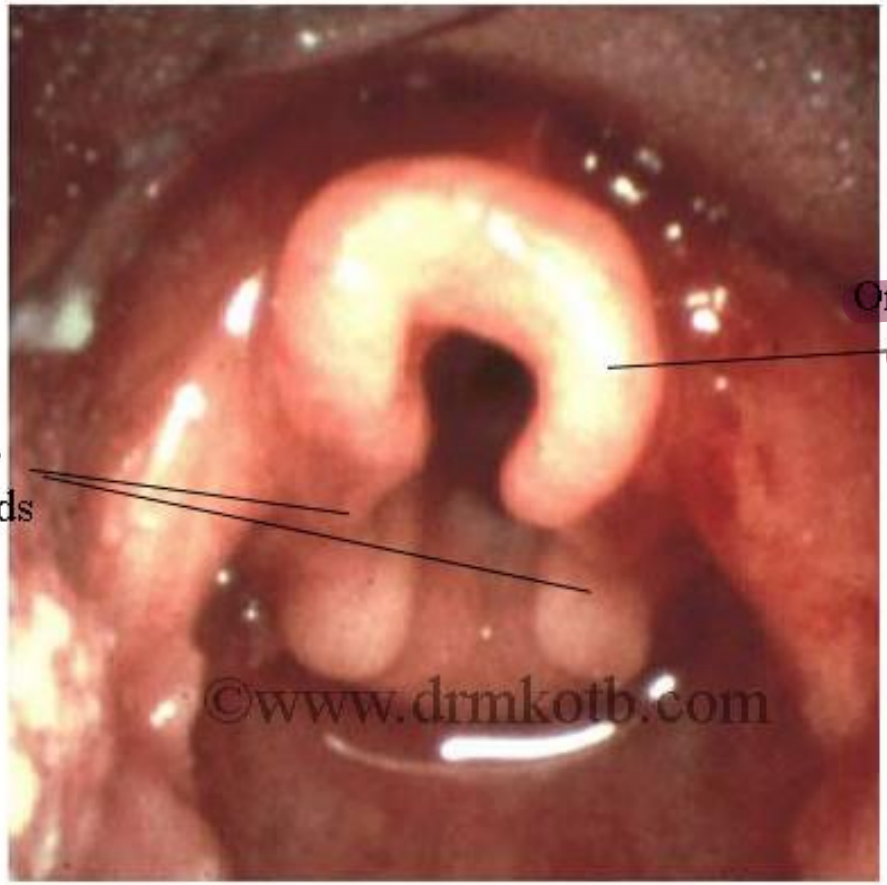
Laryngomalacia
Subglottic stenosis
Vocal cord paralysis
Subglottic haemangioma
Macroglossia or micrognathia
Malignancy

Laryngomalacia

- The most common cause of inspiratory stridor in the neonatal period and early infancy and accounts for as many as 75% of all cases of stridor.
- Stridor may be exacerbated by crying or feeding. Placing the patient in a prone position with the head up alleviates the stridor; a supine position exacerbates the stridor.
- Laryngomalacia is usually benign and self-limiting and improves as the child reaches age 1 year. In cases where significant obstruction or lack of weight gain is present, surgical correction or supraglottoplasty may be considered

Subglottic stenosis

- *Most commonly seen secondary to prolonged intubation.*



Laryngomalacia

Vocal cord dysfunction

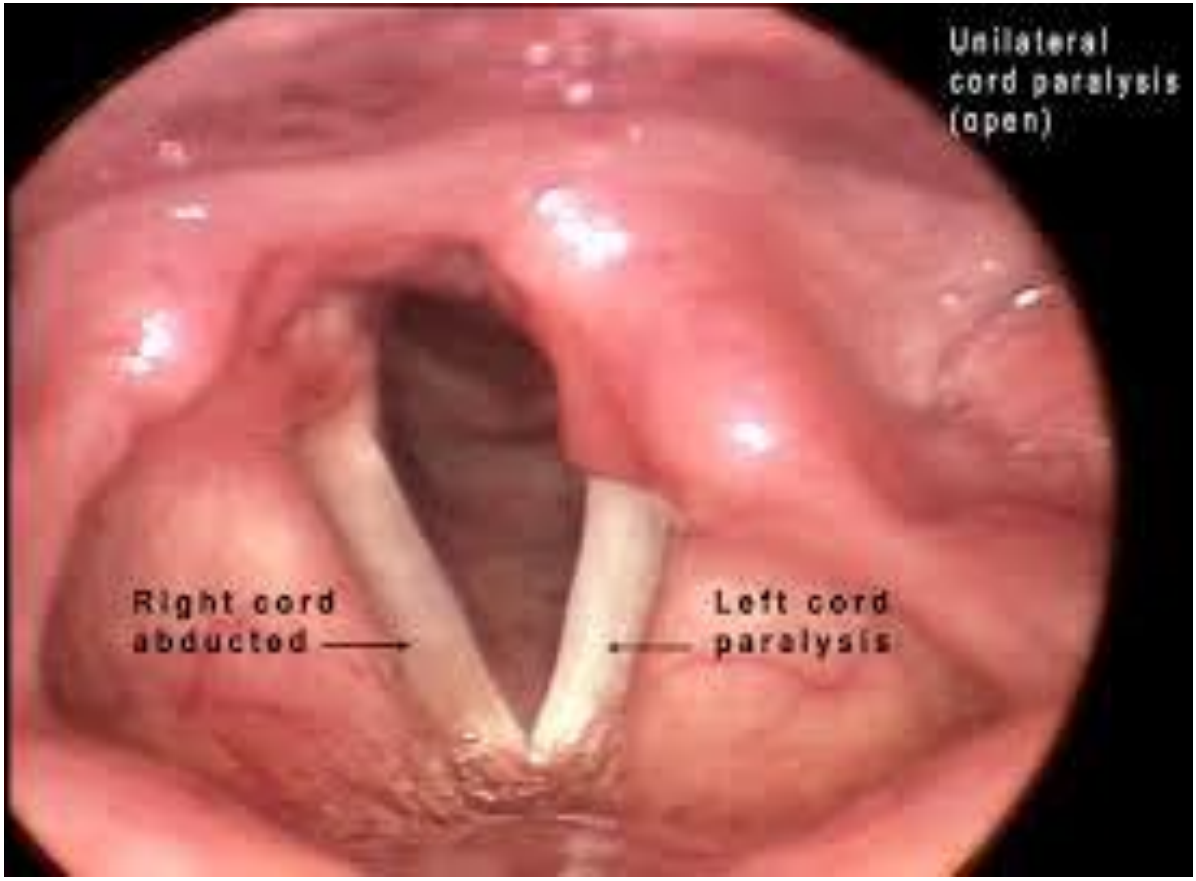
- Probably the second most common cause of stridor in infants. Unilateral vocal cord paralysis can be either congenital or secondary to birth or surgical trauma (eg, from cardiothoracic procedures, thyroid surgery).
- Patients with a unilateral vocal cord paralysis present with a weak cry and biphasic stridor that is louder when awake and improves when lying with the affected side down.
- Bilateral vocal cord paralysis is a more serious entity. Patients usually present with aphonia and a high-pitched biphasic stridor that may progress to severe respiratory distress.

Grade 1
Stenosis 0-50%

Grade 2
Stenosis 51-70%

Grade 3
Stenosis 71-99%

Grade 4
Stenosis 100%


Unilateral
cord paralysis
(open)
Right cord
abducted
Left cord
paralysis

Management of Chronic Stridor

- For non-emergency or chronic cases, visualisation of the upper airway will normally be done via fibreoptic nasal endoscopy.
- Further imaging studies, such as CT scanning, can be used in the case of abscess or malignancy, whilst bronchoscopy can be used if visualisation below the vocal cords is warranted, such as suspected subglottic stenosis.
- **Definitive management steps** will then vary between the underlying causes.

HOARSENESS OF VOICE

- A **hoarse voice** refers to a weak or altered voice. It is a relatively common presentation, and can represent a wide range of pathologies.
- Patients with hoarse voice should undergo a full investigation to identify the cause of the symptom and should be treated accordingly.

INVESTIGATIONS

flexible nasal endoscopy

- Any patient presenting with a hoarse voice should undergo a flexible nasal endoscopy (FNE). FNE allows visualisation of the larynx and the vocal cords

Microlaryngobronchoscopy

- Another procedure that allows for visualize the larynx, vocal cords, and the bronchi. Similar to FNE, however MLB is performed in a theatre setting under general anesthesia.

Stroboscopy

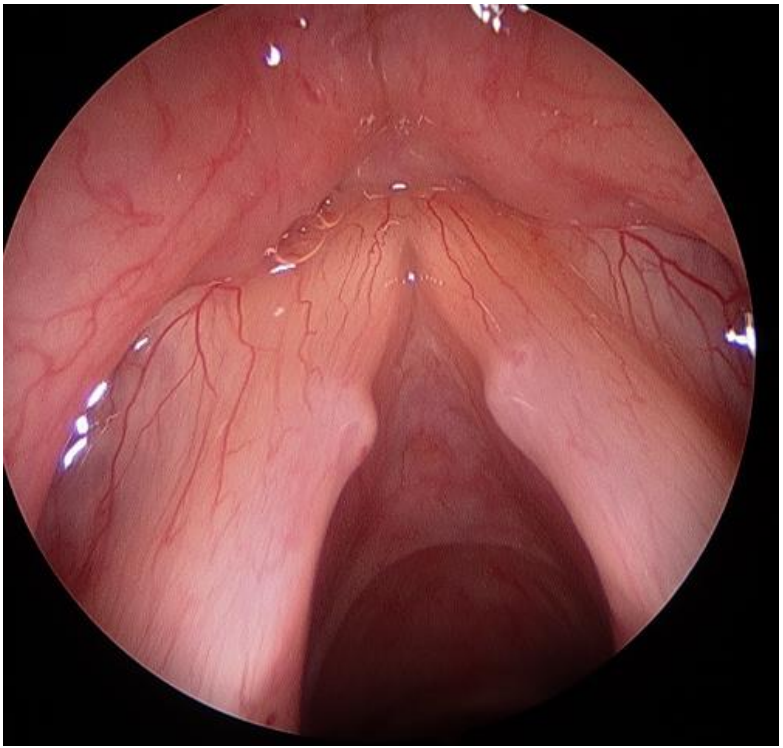
- Used in specialist voice clinics and can be a very useful diagnostic test in vocal cord dysfunction. It involves the use of synchronised flashing lights to make vocal fold movements appear slower, allowing for complete assessment of their movement.

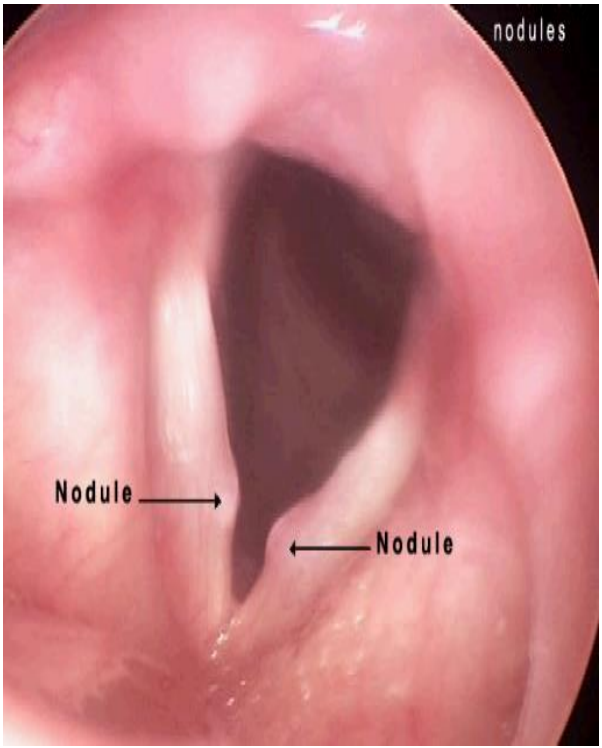
DIFFERENTIAL DIANGOSIS

BENIGN LARYNGEAL CONDITIONS

Vocal cord nodules

- Commonly secondary to chronic phonotrauma (vocal abuse)They are benign lesions that are frequently bilateral, occurring at the junction of the anterior 1/3rd and posterior2/3rd.
- Management is mainly from the Speech and Language Therapy (SALT) team, however in severe or resistant cases, surgical intervention may be warranted.





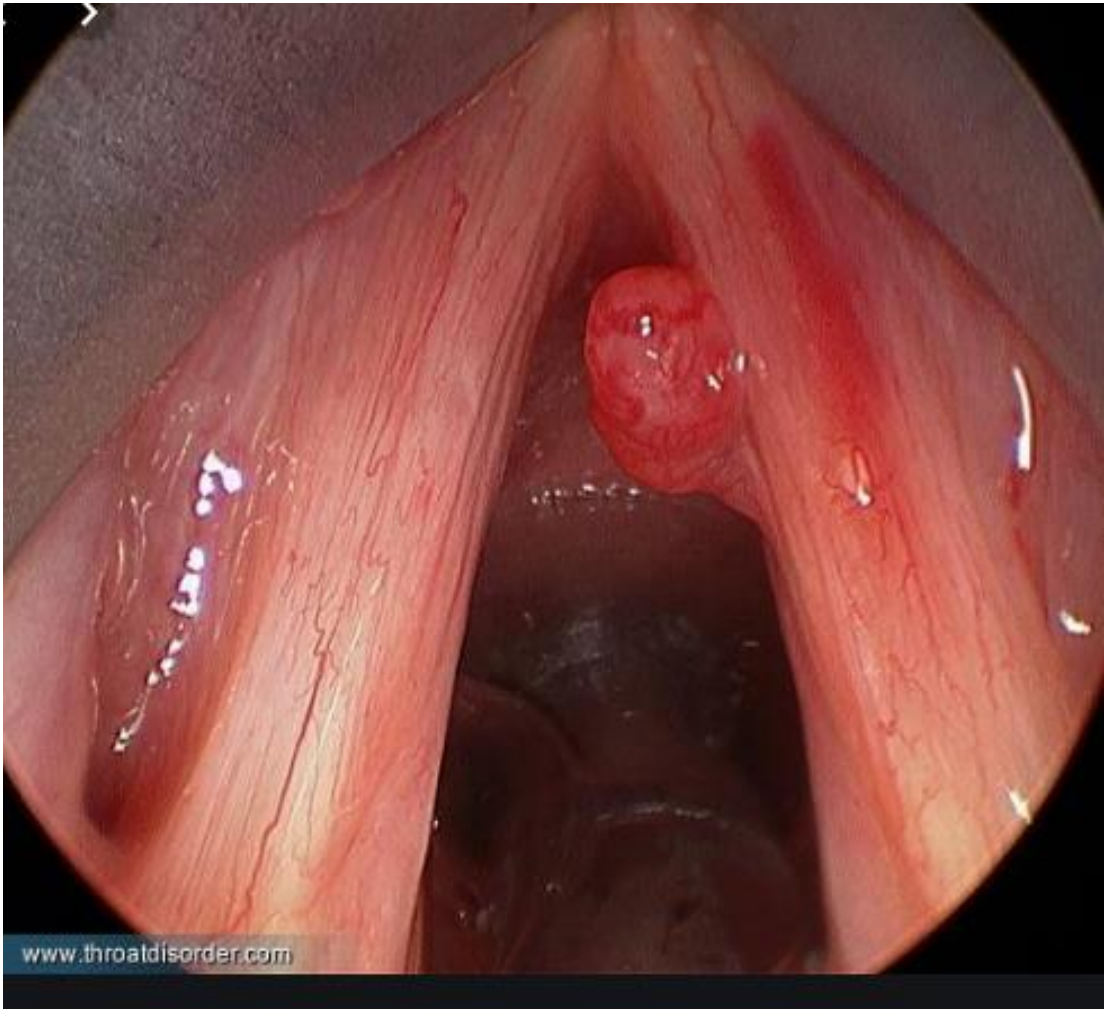
Pathophysiology: abuse (yelling), or misuse (hyperfunction) may produce excessive amounts of mechanical stress by increasing the rate and/or force with which the vocal folds collide. This may lead to trauma that is focalized to the mid-membranous vocal fold and subsequent wound formation. Repeated or chronic mechanical stress is thought to lead to the remodeling of the superficial layer of the lamina propria.
It is this process of tissue remodeling that results in the formation of benign lesions of the vocal folds such as nodules.

Vocal cord polyps

- Benign lesions
- Differ from nodules in that :

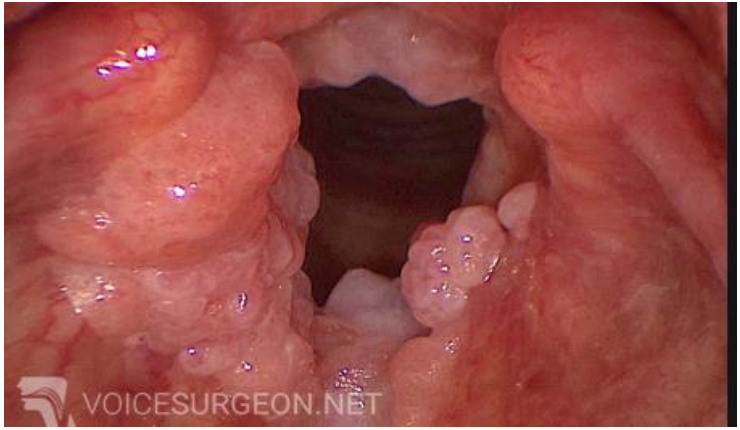
Unilateral, more vascular, usually larger.

- Vocal abuse, smoking, alcohol use, sinusitis, allergies, and rarely, hypothyroidism.
- may need surgical excision to exclude malignancy.


www.throatdisorder.com

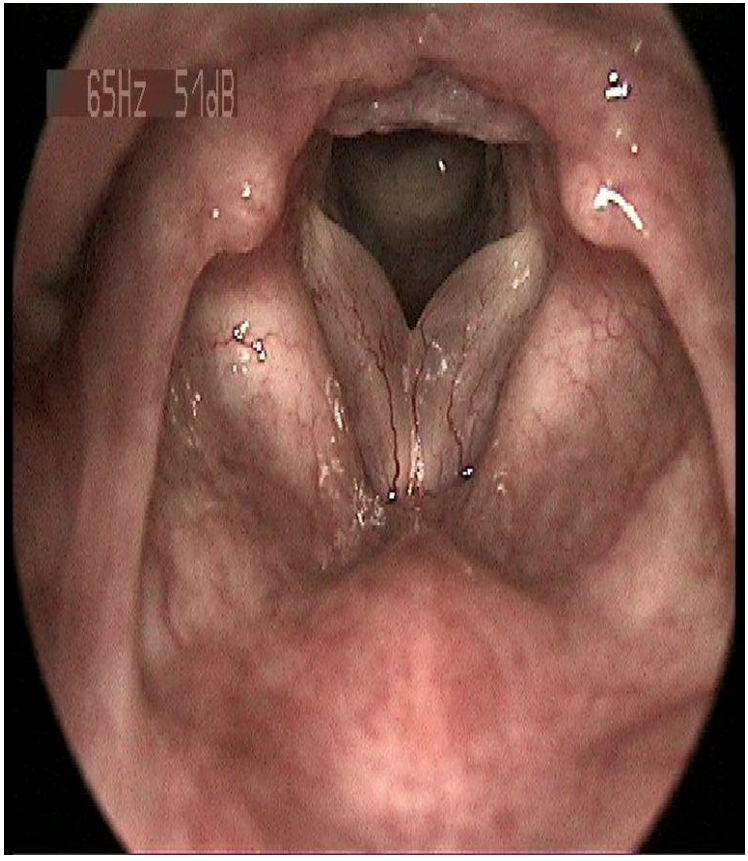
Recurrent respiratory papillomatosis

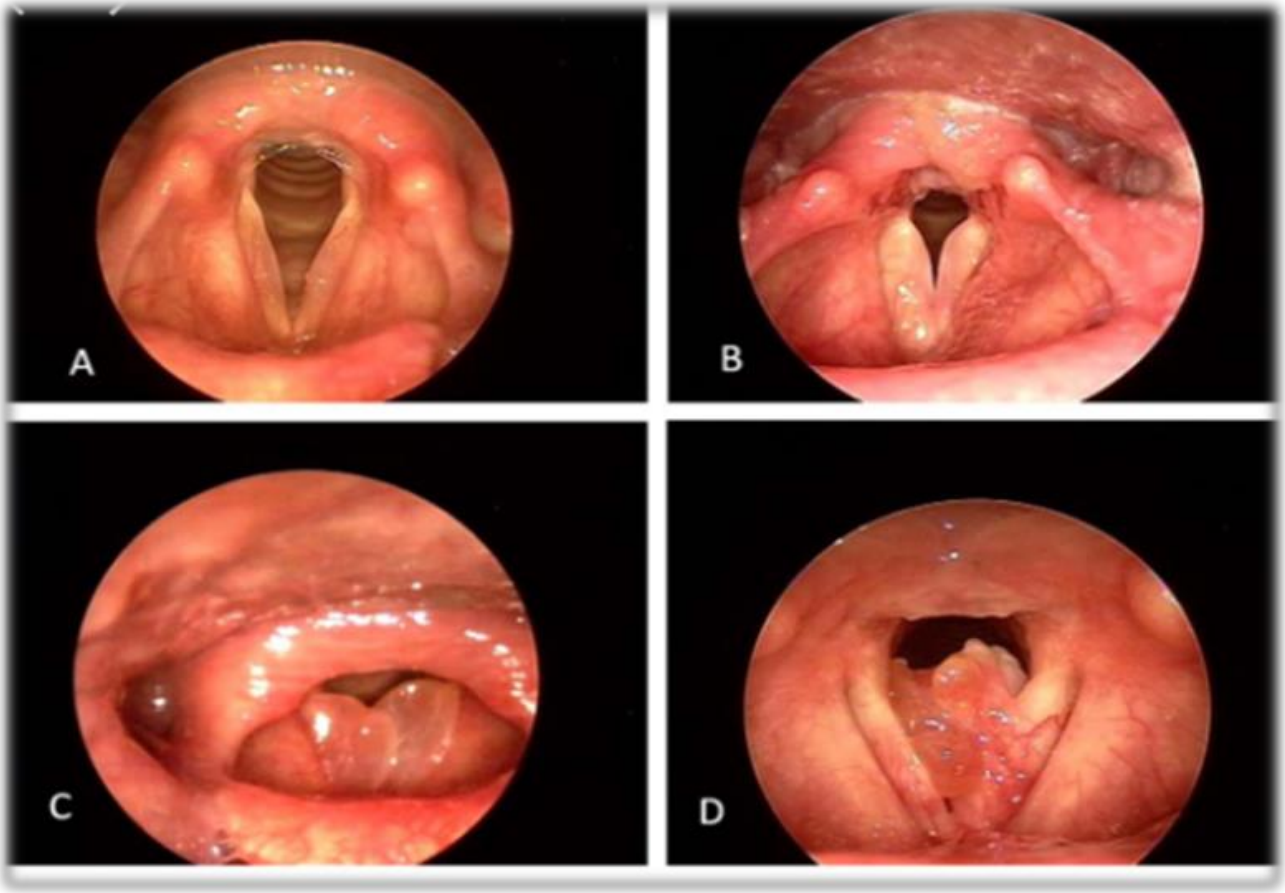
- benign lesions in the larynx, commonly caused by HPV infection. If left untreated, papillomas can grow to cause airway obstruction and hence need surgical excision or debulking. It is common for patients to need repeat procedures as papillomas can recur.



Reinke's edema

- Reinke's edema almost always occurs due to **longstanding smoking.**
- Literature suggests that it may occur secondary to thyroid disease, hormonal change, stomach acid reflux, or voice overuse.
- Reinke's edema does not disappear after smoking cessation, however it may stop growing in size.





The edema develops by degrees, as a nonspecific reaction of the vocal folds to various irritative noxious agents

Treatment : stop smoking, control reflux, microlaryngoscopic surgery

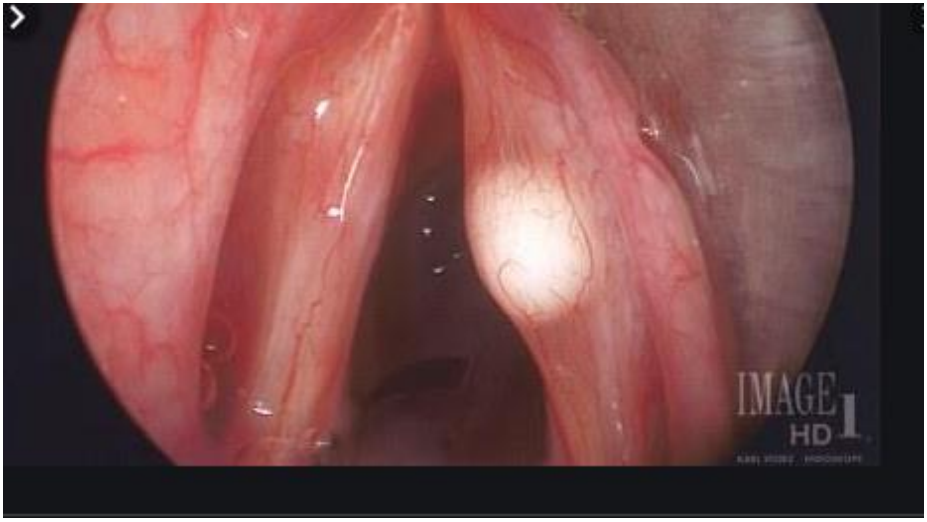
Vocal fold cysts

- benign masses of the membranous vocal folds.
- These cysts are enclosed, sac-like structures that are typically of a yellow or white colour.
- They occur unilaterally on the midpoint of the medial edge of the vocal folds
- Initial treatment of the cysts involves voice therapy to reduce harmful vocal behaviours. If symptoms remain after voice therapy, patients may require surgery to remove the cyst. Surgery is typically followed by vocal rest and further voice therapy to improve voice function. Cysts may also be treated using vocal fold steroid injection.

Vocal cord cysts

- There are several possible causes of vocal fold cysts:

1. They can be congenital.
2. They can result from the blockage of a mucous gland's excretory duct. In this case, they are sometimes referred to as retention cysts.
3. They can be the result of phonotrauma



THANK YOU

"وما النصر إلا صبر ساعة، وما النصر إلا من عند الله، وإنه لجهاد نصر أو استشهاد."