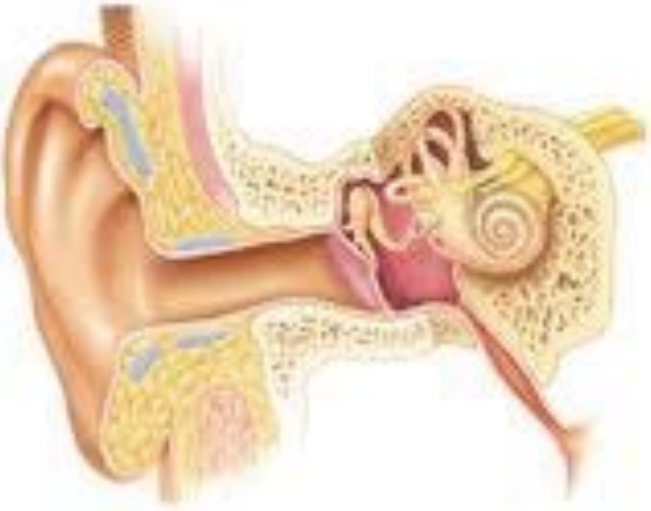
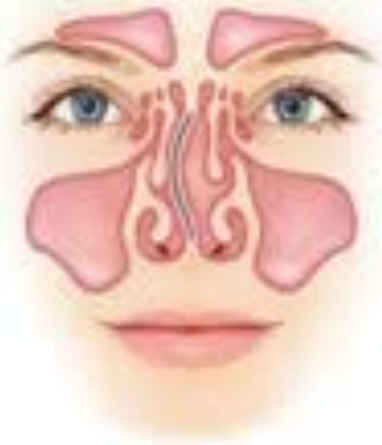
ENT Teams 430 & 429

430 Teams

Diseases of the Ear,Nose and Throat





ear 1(anatomy and physiology)

Done by: Lama Aleisa

Revised by: Hadeel S. Al-Madany

The slides were provided by Dr. Abdulrahman Hagr Al-Ghamdi
Additional information from: -Clinical Anatomy by Richard S.Snell
-429 team

Important Notes in red
Copied slides in black
additional notes in green/ blue

2 Ear anatomy and physiology (ear 1)

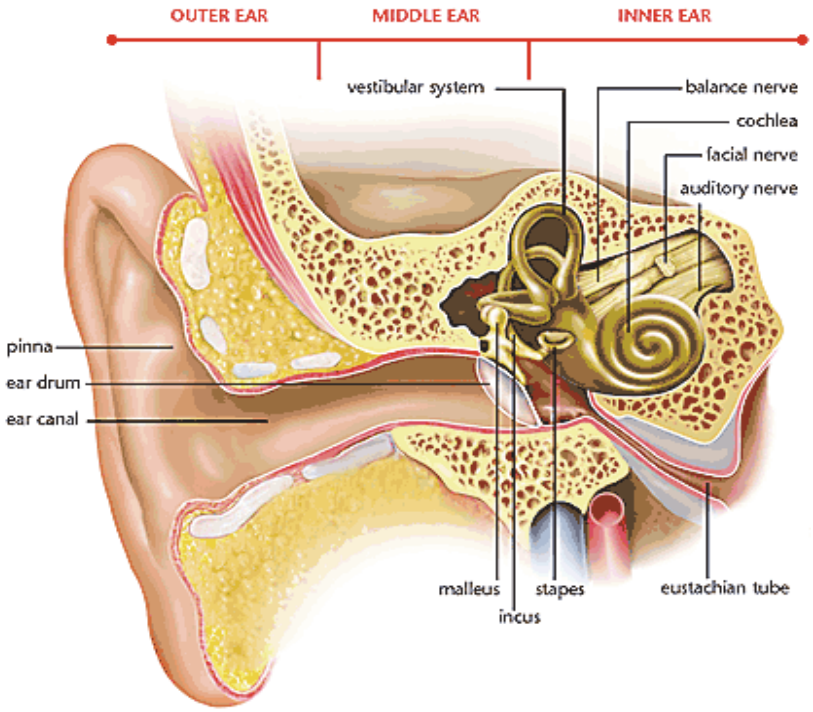
Objectives of the lecture:

- Anatomy of: external, middle and internal ear. Including structures, relations, and nerve supply.
- Brief embryology of the ear.
- Physiology of the ear. Including hearing and balance.
- Earache

Anatomy of the ear:

The ear consists of

1) External ear: from the outer part till the eardrum (tympanic membrane)
2) Middle ear (tympanic cavity): from the eardrum till the stapes footplate
3) Internal ear: cochlea and semicircular canals.

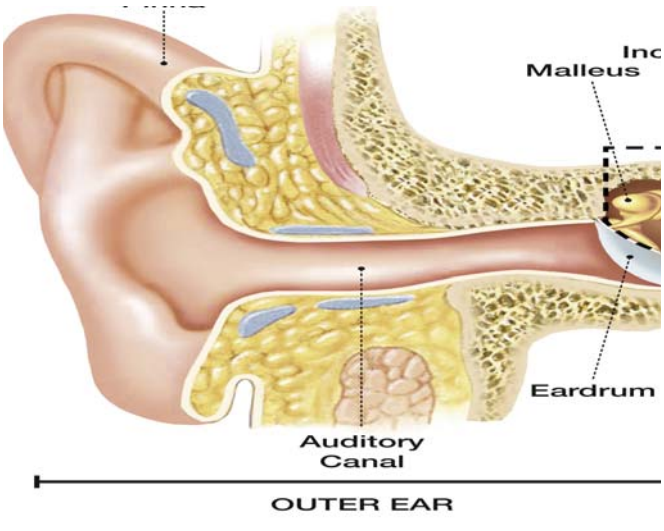


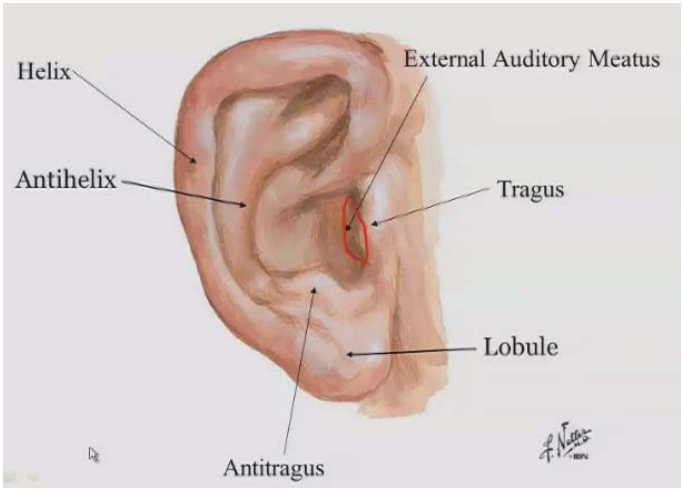
1) External ear: (outer ear)

External ear is formed by:

1) Auricle
2) External auditory meatus (auditory canal).

Both of them are lined by skin.





The external auditory meatus (2.5 cm) is an S shape canal (to protect the ear drum and middle ear).
And it consists of:

1) Cartilaginous part (outer 1/3): formed by elastic cartilage and contains hair follicles, sebaceous and ceruminous glands (secrete wax).

2) Bony part (inner 2/3):

The narrowest portion is at the bony-cartilaginous junction.

-Tympanic membrane (ear drum) considered part of the external ear.

> The doctor said that tympanic membrane is part of the external ear, but in other resources it considered as part of the middle ear.

2) Middle ear: (tympanic cavity)

The middle ear is a sterile air-containing cavity communicates with the nasopharynx through the Eustachian tube (Auditory tube).

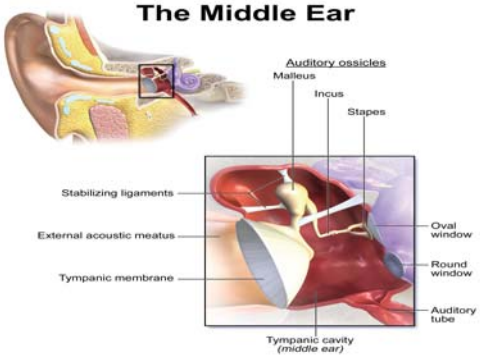
Tympanic membrane:

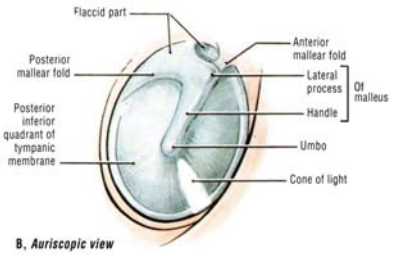
Is a thin fibrous membrane that is pearly gray in color.

1) Umbo: Tip of handle of Malleus
2) Cone of light (reflex from otoscope): anteriorly and inferiorly from the Umbo.
3) Pars Flaccida: slack part bounded by posterior and anterior mallear folds.
4) Pars tensa: the reminder tense part of the membrane.

Tympanic membrane is formed of 3 parts:

1. Outer layer → stratified squamous epithelium (skin). "ectoderm"
2. Middle layer →fibrous layer. "mesoderm"
3. Inner layer → mucous membrane. "endoderm"





Auditory ossicles:

1) Malleus مطرقة 2) Incus سندان 3) Stapes ركاب

The stapes ends up with footplate which then communicates with the inner ear through the oval window to vibrate the fluid in the cochlea.

-The Stapes receives the insertion of stapedius muscle.

-Handle of Malleus receives the insertion of Tensor tympani muscle.

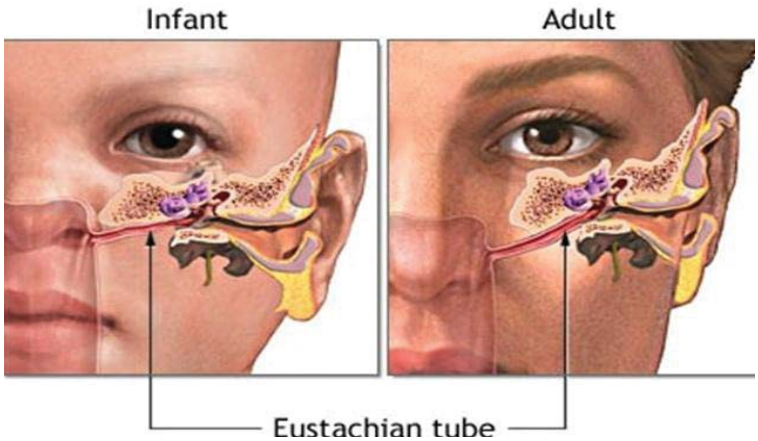
> Contraction of the stapedius muscle restrict the movement of the stapes (this consider a physiologic reflex that protects the inner ear from very loud sounds (Attenuation reflex)).

Eustachian tube (Auditory tube):

Eustachian tube connects the anterior wall of the tympanic cavity to the nasopharynx. It is lined by ciliated mucosa which helps in clearing the middle ear secretions. It also serves to equalize the pressure in the tympanic cavity with the nasopharynx. It is normally closed at rest, but it opens during swallowing, sniffing and yawning by the action of Tensor veli palatini muscle.

Clinical notes:

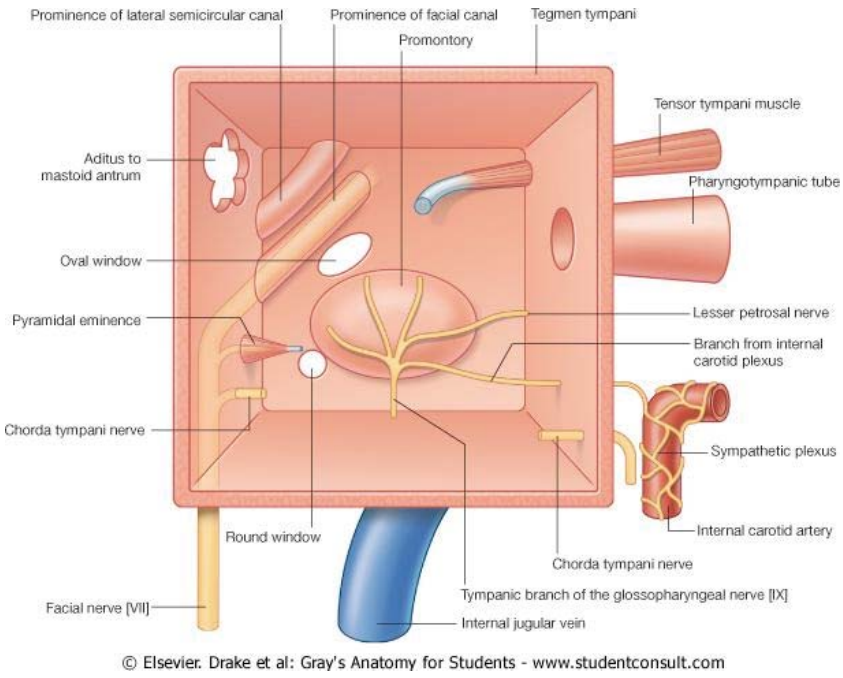
- Folliculitis can develop in the cartilaginous part of the auditory canal.
- Otitis externa (OE) causes very severe pain in the bony part because it is very thin and sensitive.
- To examine the ear in adult pull the auricle upward, backward and outward. In children pull the auricle downward, backward and outward.
- The cartilaginous part of the auricle is avascular (no blood vessels), it gets its blood supply from skin's blood vessels. Hence separation of skin from the cartilage will lead to deprivation of cartilage form blood supply, which will cause necrosis very easily.



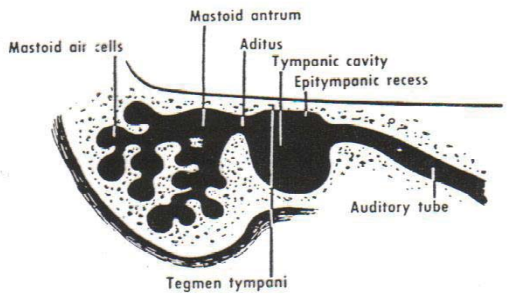
There are differences between the Eustachian tube in kids and adults:
In Children:
- Longer bony portion
- 10 degree angle (Horizontal)
- Larger isthmus
- Nasopharyngeal orifice
  – Relatively large
  – Obstructed by adenoid
  – Supine (opens)
  – Crying & Sniffing (opens → secretions get in)

Clinical notes:
-The more horizontal the tube is the easier the fluid moves from the nasopharynx to the middle ear (increase incidence of OM in children).
-Adenoid hypertrophy can block the Eustachian tube.

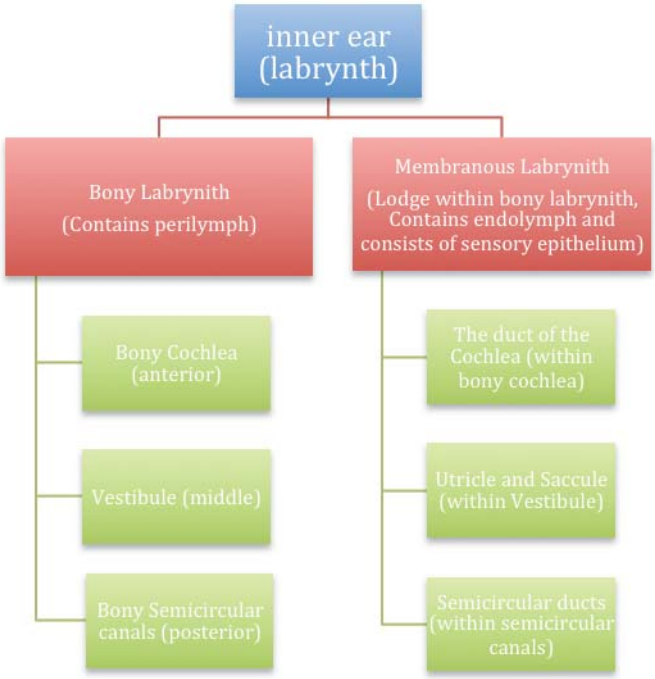
Relations of the middle ear:
-lateral wall: Tympanic membrane
-Posterior wall: aditus to the mastoid antrum (which communicates with mastoid air cells), tendon of stapedius muscle.
-Medial wall: lateral wall of inner ear, which shows the promontory from the first turn of the cochlea. It also contains the oval window (fenestra vestibule) closed by the footplate of stapes, and the round window (fenestra cochleae) closed by secondary tympanic membrane. Above the promontory there is the prominence of facial nerve canal.
-Anterior wall: thin plate of bone that separates the tympanic cavity from internal carotid artery. It has two opening; the Eustachian tube and canal for the tensor tympani muscle.
-Roof: temporal bone (tegmen tympani), which separates the cavity from the temporal lobe.
-Floor: thin plate of bone which separates the cavity from internal jugular vein.

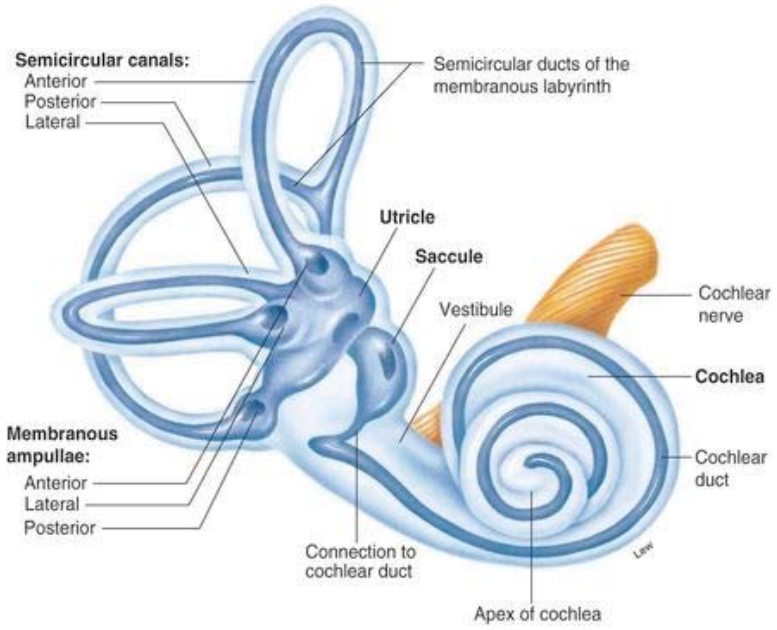


Mastoid Antrum and Mastoid Air cells:
The mastoid air cells are a series of communicating cavities within mastoid process that continues above with the antrum and the middle ear. They are lined with mucous membrane.



Inner ear: (Labyrinth)





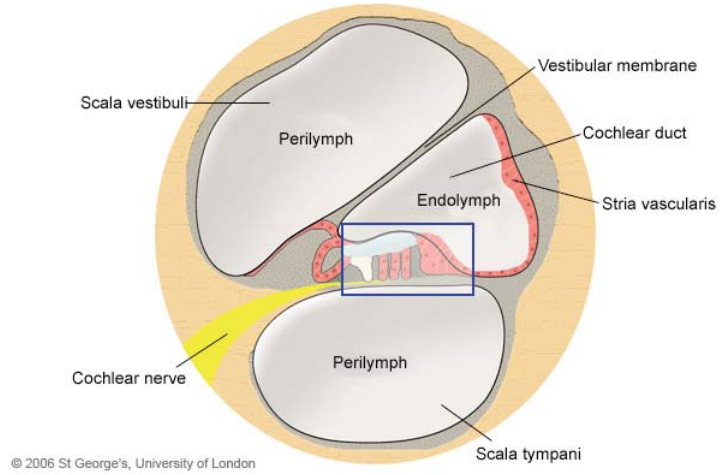
The cochlea lies within the petrous bone which is the strongest bone in our body

Cochlea:

The Cochlea consists of cochlear canals and one cochlear duct (scala media).

There are partitions inside the cochlea: (basilar membrane) that separate it into two parts; upper (Scala Vestibuli) and lower (Scala Tympani).

The perilymph of scala vestibuli is separated from the middle ear by the base of stapes at the fenestra vestibule. The perilymph of scala tympani is separated from the middle ear by the secondary tympanic membrane at the fenestra cochleae.



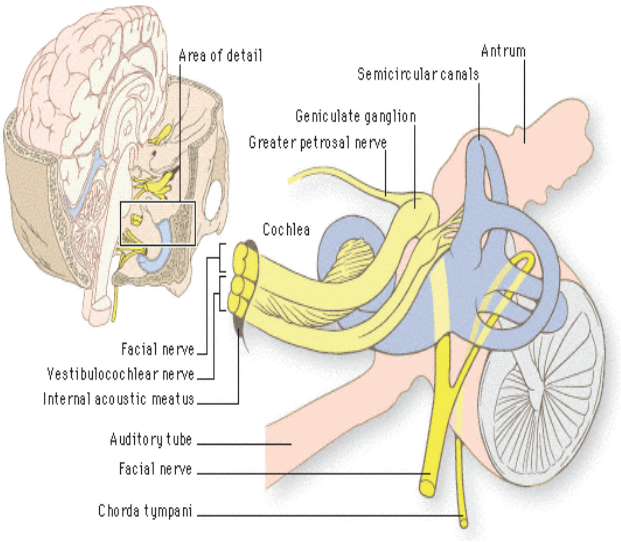
Semicircular canals and duct:

The three semicircular canals (superior, posterior and lateral) are perpendicular to each other and parallel to the other ear. They contain the ducts which open in the posterior part of the vestibule and each one ends with a swelling called the ampulla. Whenever there is a movement of the head (accelerate or decelerate), the endolymph in the semicircular ducts changes its speed of movement relative to that of the walls of semicircular ducts this change is detected in the sensory receptors in the ampulla.

Vestibule, utricle and saccule:

Utricle and saccule are lodged within the vestibule. They are directly connected to each other. They contain specialized sensory receptors which are sensitive to the orientation of the head to gravity or other acceleration forces.

Facial nerve is very close to the inner ear. There is a high risk of facial nerve paralysis in some surgeries such as cochlear implant. It comes out from the pons (cerebellopontine angle) then it enters the external auditory canal after that it enters the middle ear and runs through the parotid.



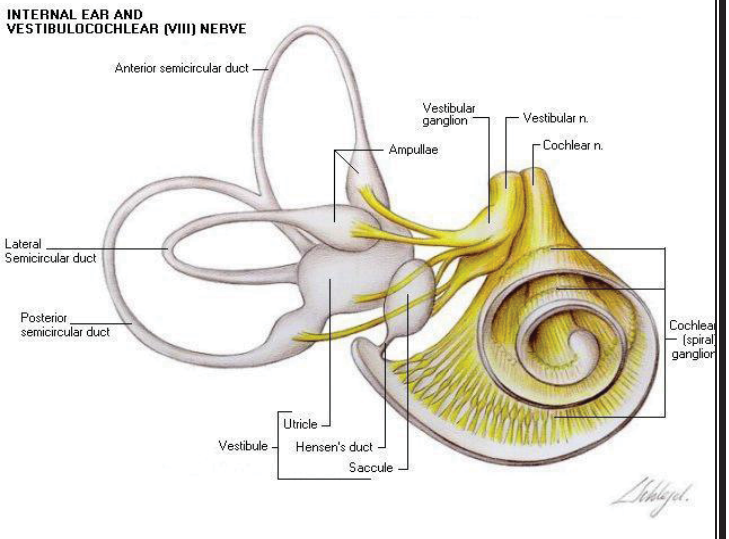
vestibulocochlear nerve (CN VIII):

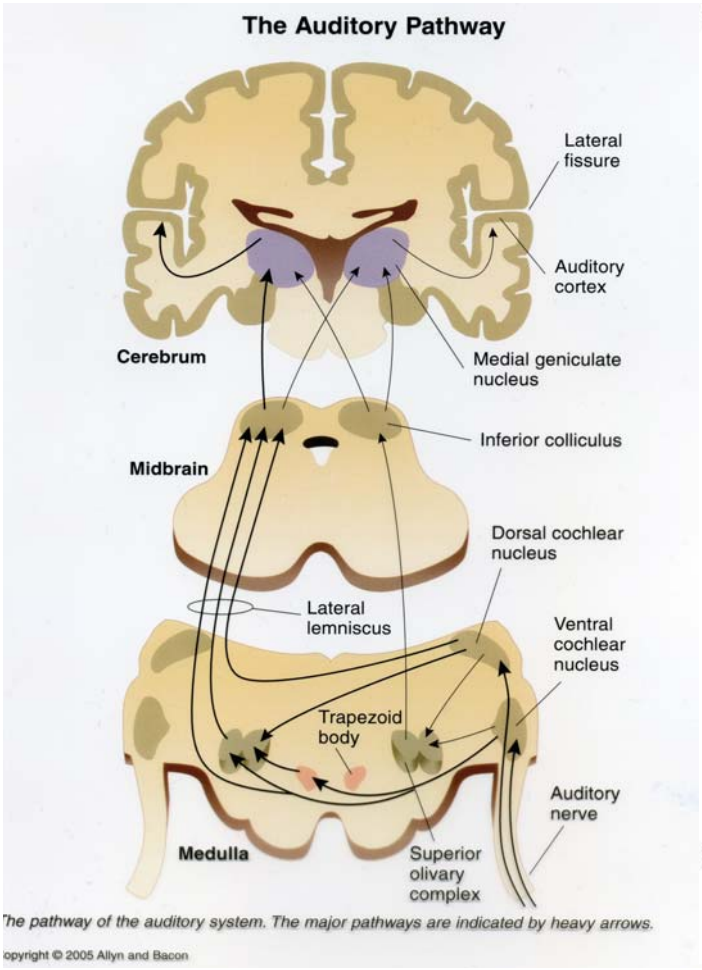
Clinical notes:

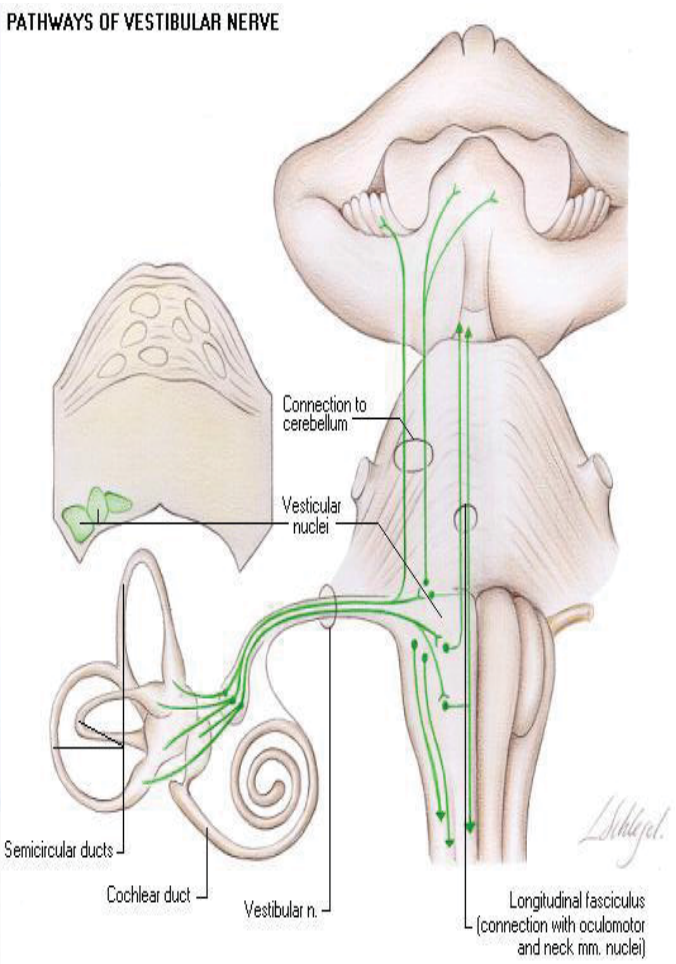
-Itching in the nose from allergy can refer to the distribution of the trigeminal nerve involving the ear

- While examining the ear the patient may cough because of the stimulation of CN IX. Or he may have Vaso-Vagal attack because of the stimulation of CN X, that's why the patient usually has to lie down during cleaning or dewaxing.

- Great auricular nerve passes through the parotid gland which may be injured during parotid surgery (parotidectomy) resulting in loss of sensation of the lobule of the auricle which can lead to frostbite in cold weather.







Cochlear nucleus→ midbrain →cochlear cortex (bilaterally)

Clinical notes:
When someone has brain tumor in one side of the auditory cortex the other side will still work and hearing will not be affected.

Nerve supply:
Motor:
-Tensor tympani muscle →trigeminal nerve (CN V)
-Stapedius muscle →facial nerve (CN VII)
Sensory:
External ear:
- Auriculotemporal nerve → trigeminal nerve CN V
-Auricular→ vagus CN X
- Great auricular and lessor occipital →cervical II & III
Middle ear:
-Tympanic nerve → glossopharyngeal nerve CN IX

Arterial supply:
– Superficial temporal
– Posterior auricular

Venous drainage:
– Superficial temporal
– Posterior auricular veins

Lymphatic drainage:
– External ear: Parotid, deep cervical lymph nodes.
– Middle ear: Retropharyngeal lymph nodes.

8 Ear anatomy and physiology (ear 1)

Physiology of the ear:

Functions of the external ear:

1- Auditory functions→Sound collection & conduction

2- Protection of the middle ear:

- Curvature
- Cerumen (Wax)
- Hairs

Functions of the middle ear:

1. Conduction and amplification of sound
2. Protection of the inner ear (Stapedius reflex)

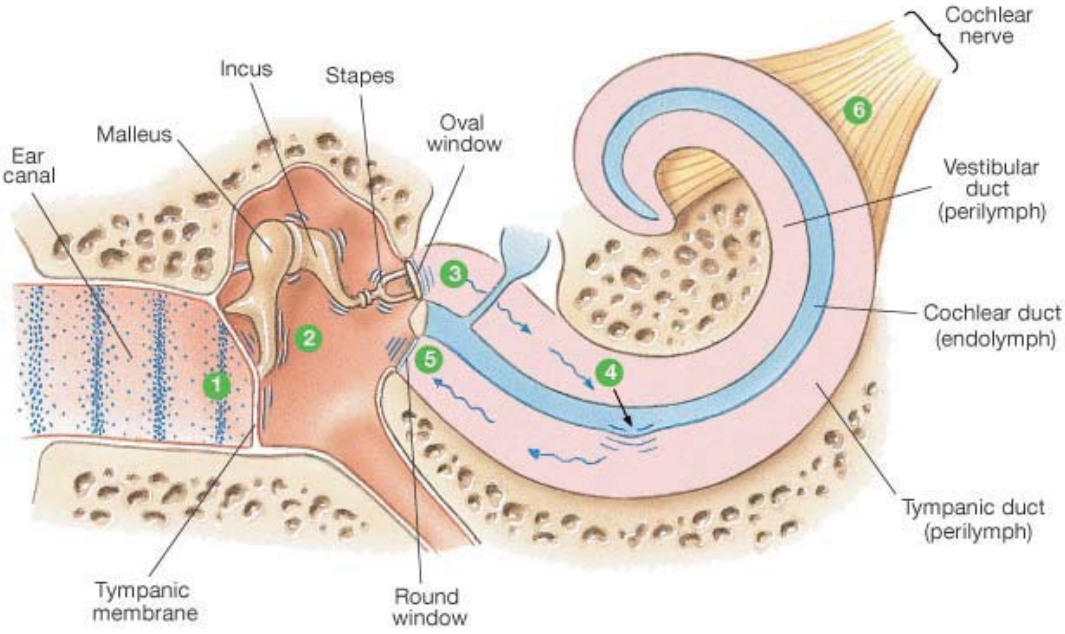
Functions of the inner ear:

1- Hearing Function (Cochlea): Transduction of sound to action potentials.
2- Vestibular Function (semicircular canals, Utricle and saccule): Participate in maintaining body balance

Types of Spatial Movement:

Rotational -3 degrees of freedom→Semicircular Canals
Translational -3 degrees of freedom→ Otolith Organs (utricle+ saccule)
The vestibular nerve is always in a firing level but it goes up with acceleration and down with deceleration.

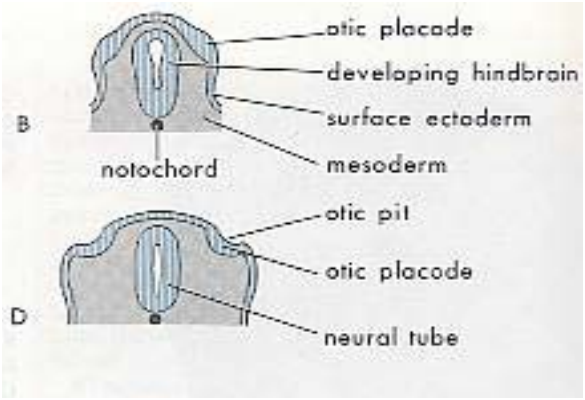
1. Sound waves strike the tympanic membrane and become vibrations.
2. The sound wave energy is transferred to the three bones of the middle ear, which vibrate.
3. The stapes is attached to the membrane of the oval window. Vibrations of the oval window create fluid waves within the cochlea.
4. The fluid waves push on the flexible membranes of the cochlear duct.
5. Energy from the waves transfers across the cochlear duct into the tympanic duct and is dissipated back into the middle ear at the round window.
6. Hair cells within the cochlear duct create action potentials in the sensory neurons of the cochlear nerve.

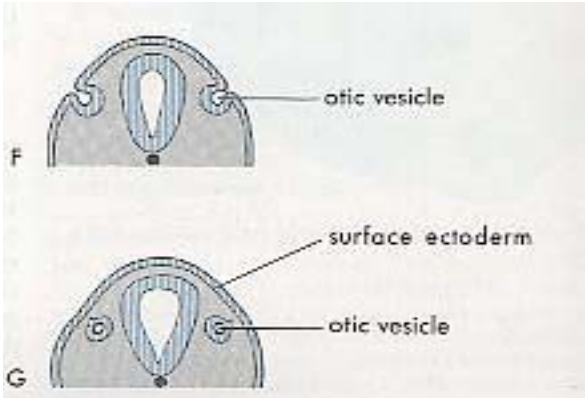


Embryology of the ear:

The ear starts to develop in the 4th weak.

Clinical notes:
-ingestion of Drugs such as Antibiotics after the 9th week will not affect the development of the ear because it will be





External ear development:

6 auricular hillocks
• The pinna
– Initially develops in the neck.
–The growth of the Mandible ascends the ear to the level of the eyes.
• Part of the auricle originating from
– 1st branchial arch (innervated by CN V)
– 2nd branchial arch (innervated by CN VII)

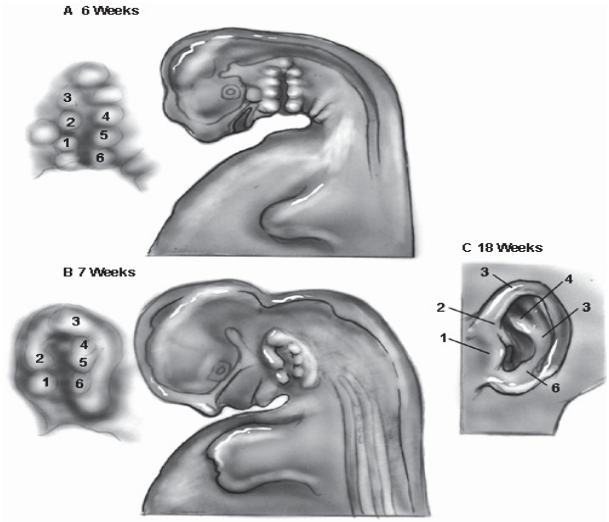
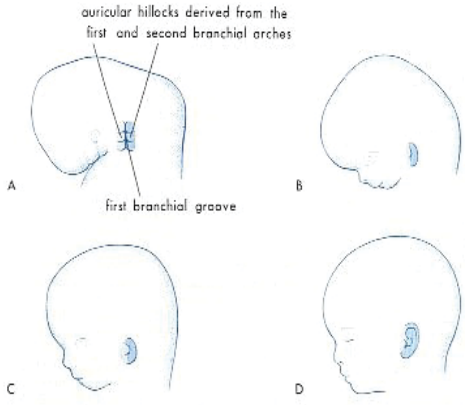
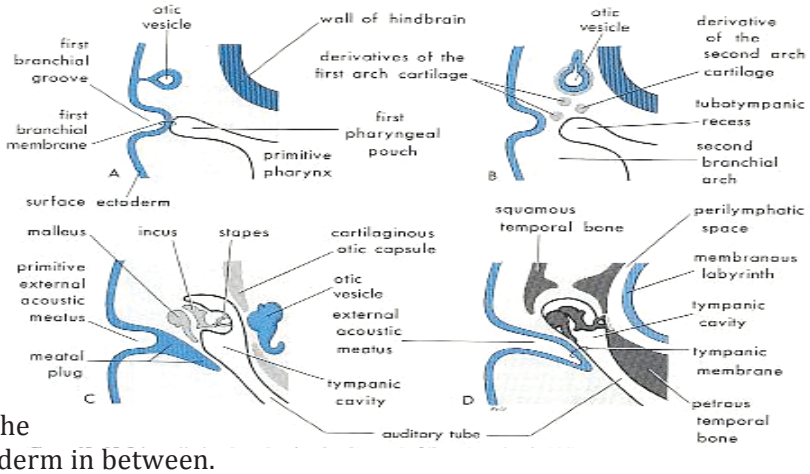


FIG. 5-1. Development of the auricle. A: Six hillocks form on the first and second branchial arches; all are identifiable at 6 weeks. B: Seven-week stage. C: By 18 weeks, the adult form is recognizable.

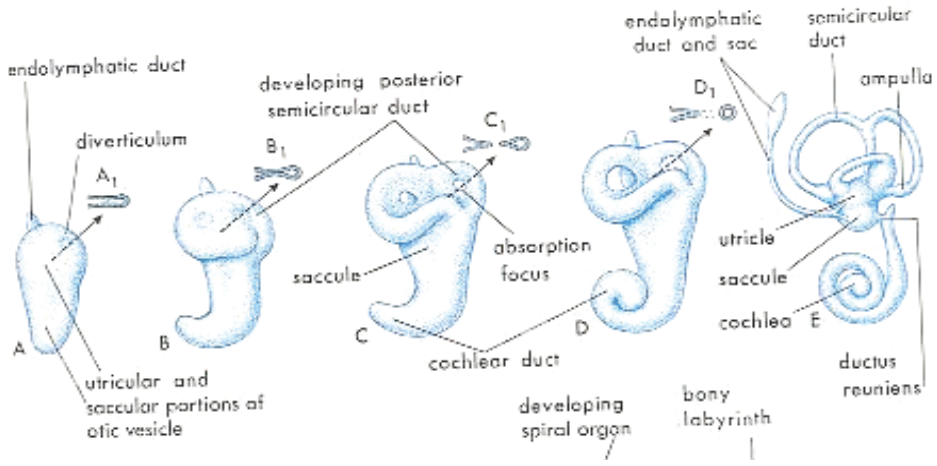


Middle ear development:

1st pharyngeal pouch elongates into the tubotympanic recess. The tubotympanic recess becomes the tympanic cavity and mastoid antrum.
• Distally contacts the 1st pharyngeal cleft → TM
• Proximally connects the pharynx→Eustachian tube
-The tympanic membrane is ectoderm from the outside, endoderm from the inside and mesoderm in between.



Inner ear development:

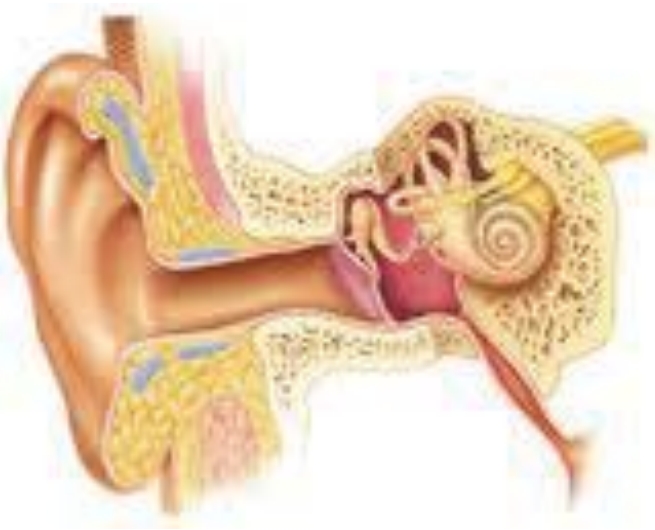


It is an ectoderm which is thickened and migrates. First it starts as a sac that develops until it forms the cochlea and semicircular canals, this development can stop at any stage and can be detected by CT scan.
Otic vesicle:
– Invaginates of mesenchyme – Detaches from ectoderm
– Divides into 2 regions:
1) Utricular portion
– Utricle – Semicircular canals
2) Saccular portion
– Saccule – Cochlea

Good luck =)

430 Teams

Diseases of the Ear, Nose and Throat



15sth Lecture:

Ear II

Done by: Shahad Almohanna

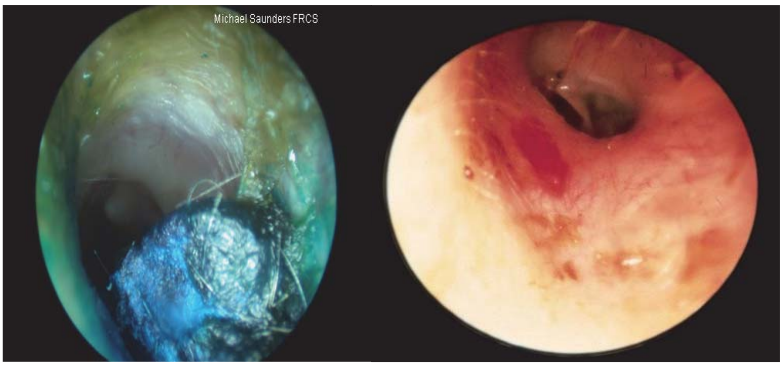
Revised by: Hadeel S. Al-Madany

The slides were provided by doctor (Dr. Abdulrahman Hagr Al-Ghamdi)
Important Notes in red
Copied slides in black
Your notes in green/ blue

Disease of external ear

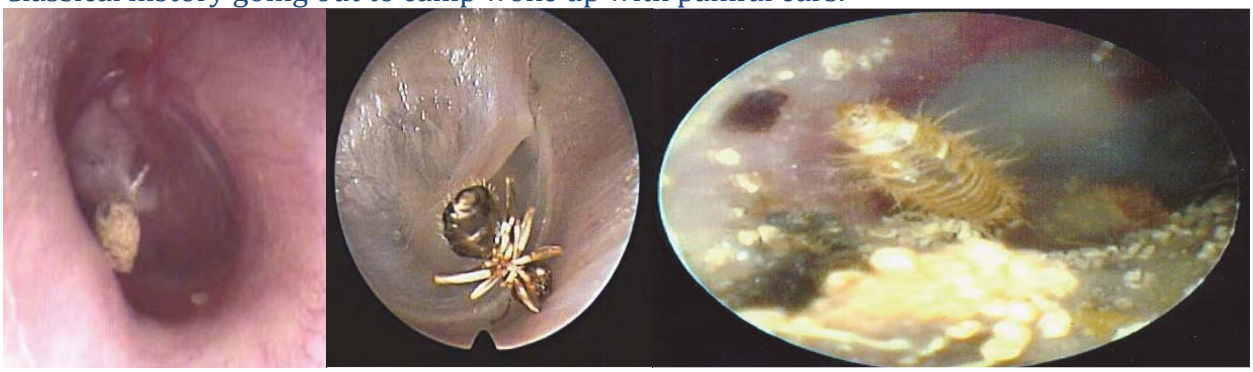
Wax: (Cerumen Impaction)

Definition:
Accumulation of the secretion of sebaceous and cermunous glands situated in the outer cartilaginous part of external canal.
Normally, the glands secrete a little amount of wax and migrate by movement of jaw.
It's the commonest cause of conductive hearing loss caused by excessive use of Q-tips.
The ear is the only organ that cleans itself.
Symptoms:
1. Deafness, which is Sudden. (Conductive Hearing Loss)
2. Earache.
3. Pain
4. Tinnitus
Treatment:
Removal of the accumulated wax by suction.



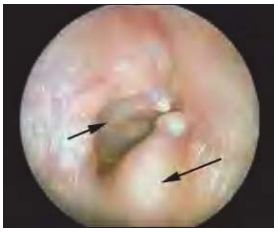
Foreign body:

Classical history going out to camp woke up with painful ears.



Painful ears from an insect treated with Jonson oil because it will drown the insect till it dies.

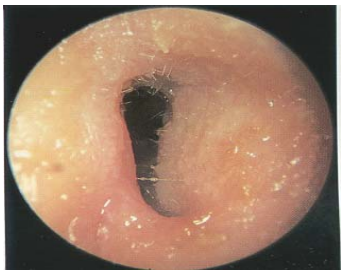
Tumors:



Benign: Exostosis, Osteoma (periosteal tumors)
Malignant: Rare, Metastasis

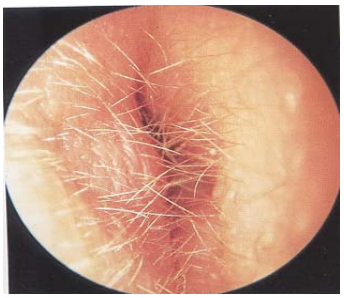
Infection (otitis externa):

Clinical Course:
Itching is the classical finding, progresses to: pain, decreased hearing, discharge (usually from bacterial infection)



AOE: Mild to Moderate Stage
• Symptoms: pain, increased pruritus.
• Signs: Erythema, increasing edema, canal debris, discharge

AOE: Severe Stage
• Symptoms: severe pain, worse with ear movement, chewing.
• Signs: lumen obliteration, purulent otorrhea, and involvement of periauricular soft tissue.



Otorrhea: little scanty, watery or purulent discharge.

Microbiology: skin organisms most common

• Bacteria 50% of cases
  - Staph aureus
  - Pseudomonas
  - Proteus
• Fungi
  - Aspergillus – tropical
  - Candida albicans – temperate

Epidemiology:
•Warm, humid climate
  - Swimmer's ear (wax is removed from the increase amount of chloride or shampoo)
• Poor hygiene (removing wax with a pen)
• Closed canal
  - Hearing aid (headphones)
  - Turbans in India
• Composition of cerumen
  - pH changes from acid to alkaline (D.M)
  - Softer – washed out
  - Hard block the canal
• Instrumentation of ear canal (non expert examination or cleaning which lead to injury)

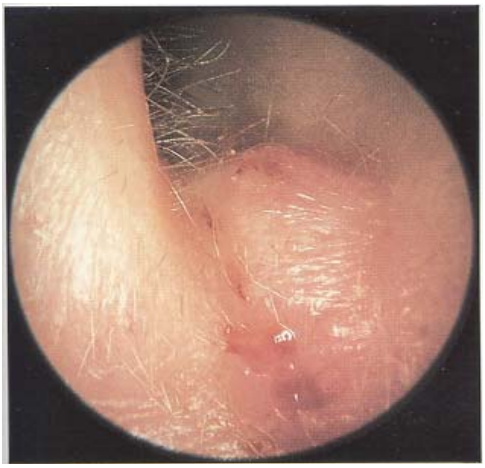
Diagnosis:
•Persistent disease could be:
  - Resistant
  - Fungal
  - Dermatological etiologies (always ask history and examine carefully)

Cultures will be helpful to identify the cause.

Treatment:

- meticulous cleaning every 2-3 days then weekly
- Topical antibiotic
- Water precautions

Furunculosis:



- Acute localized infection
- Lateral 1/3 of posterosuperior canal
- Obstructed apopilosebaceous unit (hair follicle unit)
- Pathogen: S. aureus

Symptoms:

- Localized pain
- Pruritus
- Hearing loss (if lesion occludes canal)

Signs:

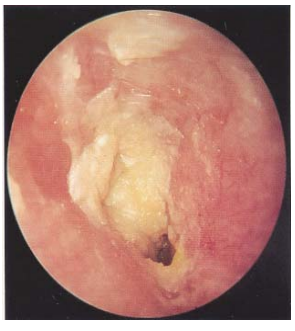
- Edema
- Erythema
- Tenderness
- Occasional fluctuance

Treatment:

- Local heat
- Analgesics
- Oral anti-staphylococcal antibiotics
- Incision and drainage reserved for localized abscess
- IV antibiotics for soft tissue extension

Otomycosis: (oto = ear mycosis = fungal)

- Fungal infection of outer ear canal skin
- Primary or secondary (AB) (secondary due to middle ear infection with fluid; fluids that go to outer ear makes it wet and this will lead to fungus accumulation so the key is to make the ear dry)
- Most common organisms: Aspergillus and Candida

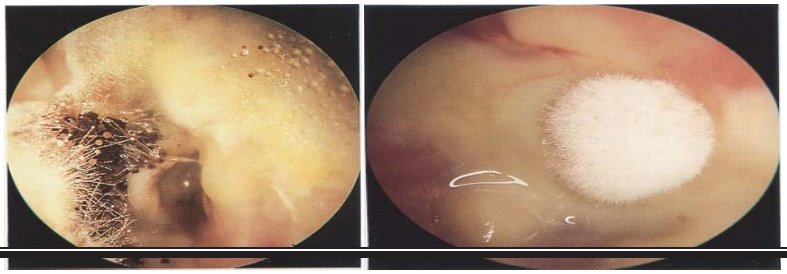


Symptoms:

- Often indistinguishable from bacterial OE
- Pruritus deep within the ear
- Dull pain
- Hearing loss (obstructive)
- Tinnitus

Physical Exam:

- Early
  - –Normal
  - – Canal erythema
  - – Mild edema
- Later
  - – "Wet newspaper"
  - – Red, tender skin
  - –Fungal hyphae



Otomycosis Fungal hyphae:

Treatment:

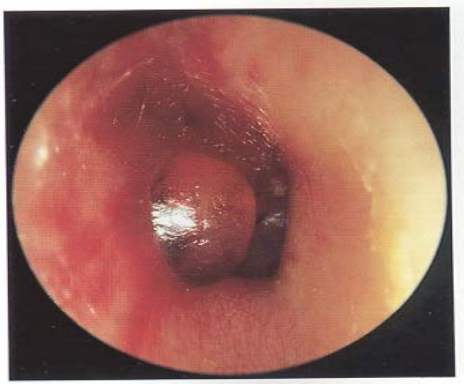
- Thorough cleaning
- Drying of canal
- Topical antifungals

Bullous Myringitis:

- Viral infection
- Bacteria of OM
- Confined to tympanic membrane
- Children

Symptoms:

- Sudden onset of severe pain
- No fever
- No hearing impairment
- Bloody otorrhea (significant) if rupture



Signs:

- Inflammation limited to TM & nearby canal
- Multiple reddened, inflamed blebs
- Hemorrhagic vesicles

Treatment:

- Self-limiting
- Analgesics
- Topical antibiotics to prevent secondary infection
- Incision of blebs is unnecessary

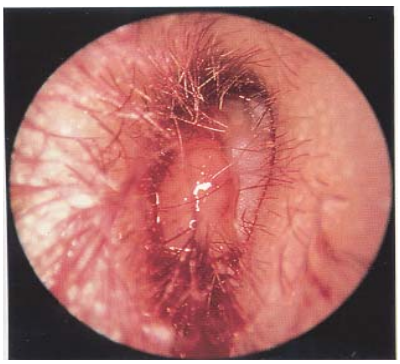
Necrotizing External Otitis (NEO)

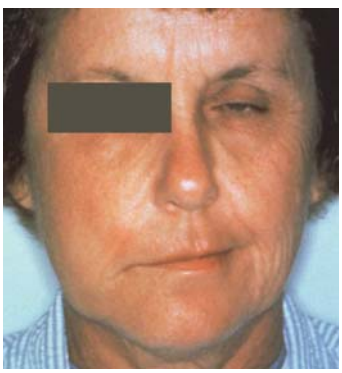
- Potentially lethal infection
- More in DM and immunocompromised patients
- Pseudomonas aeruginosa

Called also Malignant Otitis Externa

4 Ds

- Diabetes mellitus
- Discharge (Purulent)
- Discomfort
- Dysfunction Cranial nerve





Granulation obscured TM >>>

Imaging:

- Plain films
- Computerized tomography – most used

- Technetium-99 – reveals osteomyelitis
- Gallium scan – useful for evaluating Rx
- Magnetic Resonance Imaging

Treatment:

- Antibiotics: Intravenous at least 4 weeks.
- Local canal debridement
- DM control
- Pain control
- Hyperbaric oxygen experimental
- Serial gallium scans monthly

Mortality:

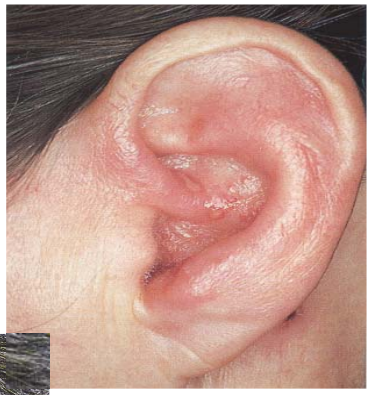
- 25 % Death rate
- 60% with multiple cranial neuropathies
- 25 % Recurrence
- May recur up to 12 months after treatment

Herpes Zoster Oticus:

- J. Ramsay Hunt
- Varicella zoster
- Shingles: Infection along one or more cranial nerve dermatomes (v hsgÅe` bh` kÅmdqudÅ chr sqhat shnm)Å

Ramsey Hunt syndrome:

- Herpes zoster of the pinna
- Otalgia
- Facial paralysis

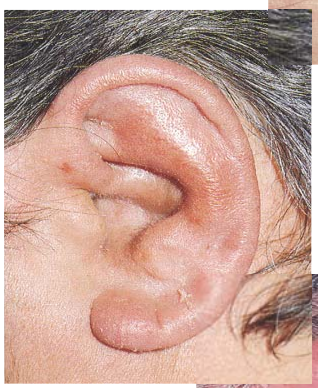


Symptoms:

Early: burning pain in one ear, headache, malaise and fever
Late (3 to 7 days): vesicles, facial paralysis

Treatment:

- corneal protection most important.
- Oral steroid then taper it (10 to 14 days)
- Antivirals



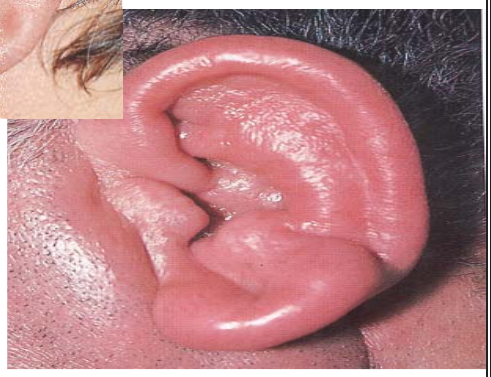
Perichondritis: infection of auricle cartilage

Signs:

- Tender auricle
- Induration
- Edema

Advanced cases:

- Crusting
- Involvement of soft tissues



Erysipelas:

Acute superficial cellulitis

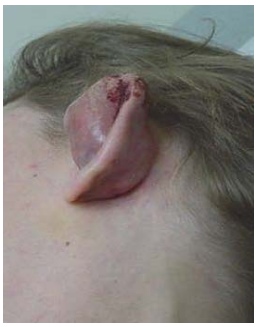
- Group A beta hemolytic streptococci.

- Skin: bright red, well demarcated.

Rapid treatment with oral or IV antibiotics if insufficient response

Ear Trauma

Auricle injuries:



Hematomas separate the perichondrium (blood supply) from the cartilage.

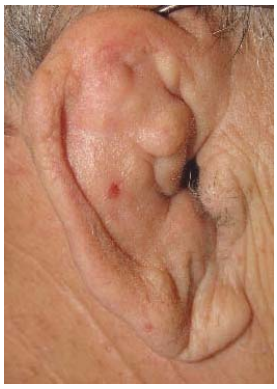
Treatment:

- Excise fibrous tissue
- Apply pressure-dressing, drain

Avulsion:

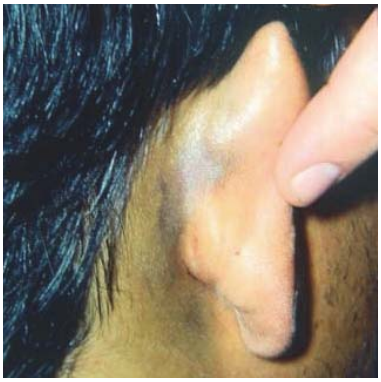
- Reimplantation.
- Microvascular anastomosis.

Cauliflower Ear a deformity as a complication for untreated auricle infection.

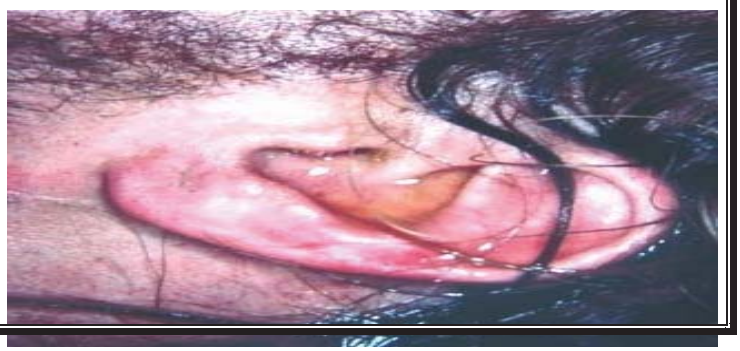


Racoon eyes sign

Battle's sign



Blood or CSF leakage:



Fractures:

Longitudinal:

– 80% of Temporal Bone Fractures
– 15-20% Facial Nerve involvement

Transverse:

– 20% of Temporal Bone Fractures
– 50% Facial Nerve Involvement

Acute Otitis media

Inflammation of the middle ear, may also involve inflammation of mastoid, petrous apex, and perilabyrinthine air cells.

- Most common reason for visit to pediatrician.
- Tympanostomy tube placement is 2nd most common surgical procedure in children.
- Development of multidrug-resistant bacteria.

Classification:

- Acute OM < 3 weeks
- Subacute OM 3 weeks to 3 months
- Chronic OM > 3 months

Epidemiology:

- Age (common in children)
- Sex
- Day care
- Seasons (more in winter)
- Genetics (runs in families)
- Breast-feeding (protective factor)
- Smoke exposure (increase risk)
- Medical conditions (examples bellow)

Medical Conditions:

- Cleft palate (effect Eustachian tube)
  - Decreases after repair
- Craniofacial disorders
  - Treacher-Collins
- Down's syndrome
- Ciliary dysfunction
- Immune dysfunction
  - AIDS
  - Steroids, chemo
  - IgG deficiency
- Obstruction
  - Adenoids (obstruct Eustachian tube)
  - NG tubes

- NT intubation
- Malignancy (nsopharyngeal cancer)

OM increases after newborn period:

- 2/3 with AOM by one year of age
- 1/2 with >3 episodes by three years
- Most common in 6 - 11 months

A lot of people mistake OM with teeth problem in children.

Day Care:

- Greater risk of AOM in children < 3 years
- Home care is best

Day care side effects:

- Large group
- Exposures with wider range of flora
- Increased URI's

Breast-feeding

- Decreases incidence of URI and GI disease
- Decreases duration of OM
  - Protective factor in breast-milk? (Antibodies from mother) Position for bottle-feeding is more transfers than breast- feeding position → Increase probability of regurgitation of milk to middle ear via Eustachian Tube.

Smoke exposure:

- Induces changes in respiratory tract
- Increased AOM and persistent effusion
- Increased chronic and recurrent AOM

Eustachian Tube:

Connects middle ear and nasopharynx, lumen shaped like two cones
Mucosa: Mucous producing cells, ciliated cells
Usually closed
Tensor veli palatini > active opening
Opens during:

- Swallowing
- Yawning
- Sneezing

Opening involves cartilaginous portion
Functions:

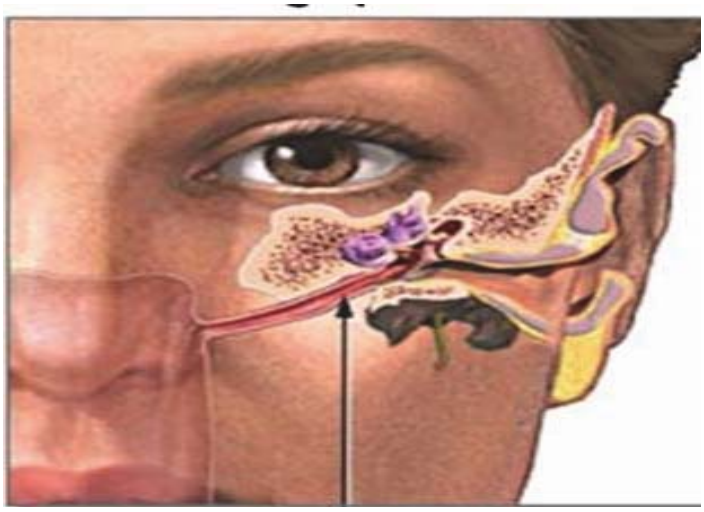
1. Protection from nasopharyngeal sound, secretions.
2. Clearance of middle ear secretions.
3. ME Ventilation (pressure regulation).

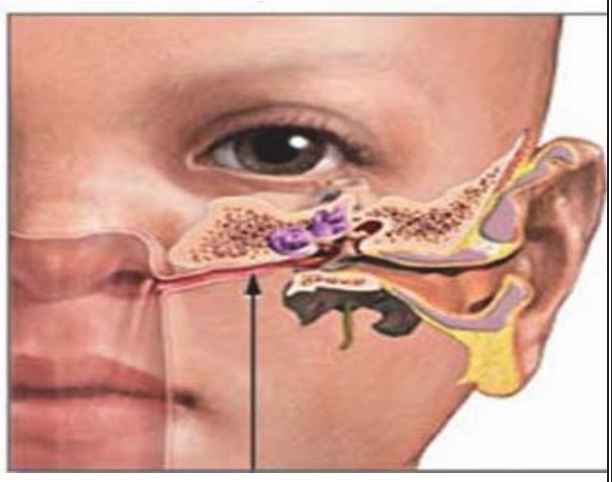
Children Eustachian tube:

- Longer bony portion
- 10 degree angle (Horizontal)
- Larger isthmus

Nasopharyngeal orifice in children:

- Relatively large
- Obstructed by adenoid, supine, crying and sniffing





Middle ear Pathology:

- Inflammation Edema
- PMN infiltration
- Epithelial ulceration
- Granulation tissue
- Fibrosis
- Influx of chronic inflammatory cells
- Increased columnar and goblet cells
- Osteitis

Microbiology:

- S. pneumoniae - 30-35%
- H. influenzae - 20-25%
- M. catarrhalis - 10-15%
- Group A strep - 2-4%
- Infants with higher incidence of gram negative bacilli

Virology:

- RSV - 74% of middle ear isolates
- Rhinovirus
- Parainfluenza virus
- Influenza virus

Treatment – AOM:

- Adults and older children - observation
- Antibiotics - consider drug resistance patterns

Antibiotics:

- First line:
  - Amoxil
  - Ceftin - B lactam stable
  - Bactrim
- Second line:

– Augmentin
– Ceftin
– Rocephin
- Macrolides - Zithromax, Biaxin

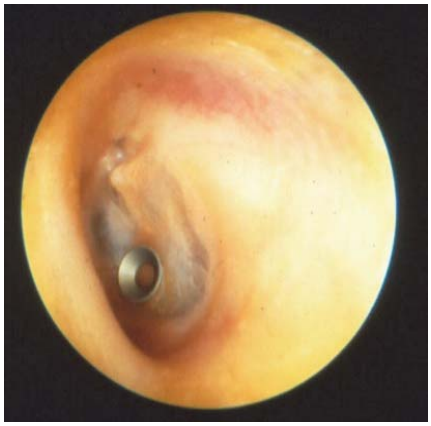
Treatment - Recurrent AOM: (if more than 3 episode in 6 monthes)

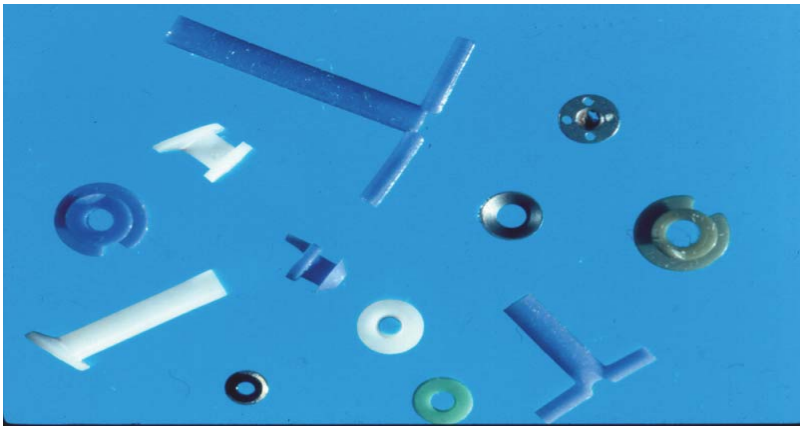
• Chemoprophylaxis (not that good! Long term drugs causes resistance)
– Sulfisoxazole, amoxicillin, ampicillin, Penicillin
– Less efficacy for intermittent propylaxis
• Myringotomy and tube insertion (better solution)
– Decreased # and severity of AOM
– otorrhea and other complications
• Adenoidectomy

Tympanostomy tube insertion:

• Unresponsive OME > 3 months
• Recurrent MEE (middle ear effusion)(if he stops antibiotics the infection comes again)
• Suppurative complication

Ventilating Tubes: different sizes (big ones for the recurrent)
T-tube is used for long durations but it leaves a hole in the tympanic membrane.

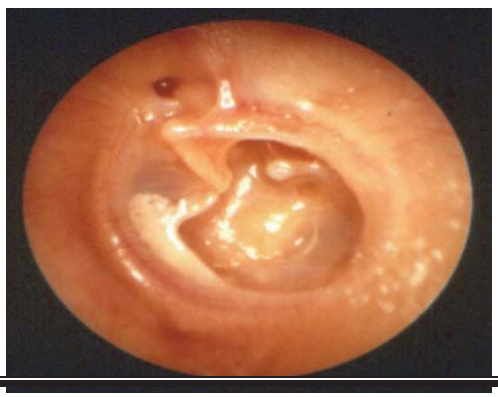




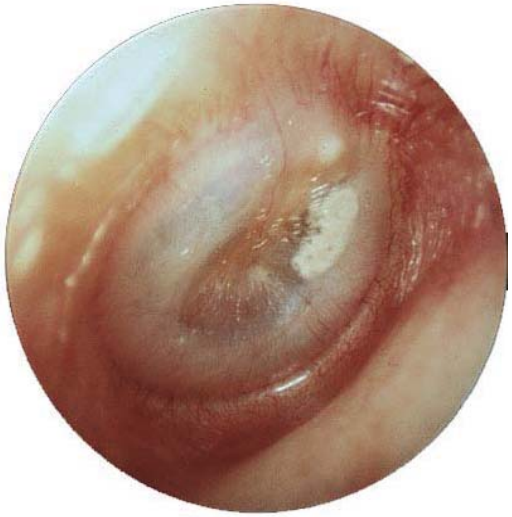
Complications of ventilation tube:

• Intratemporal:

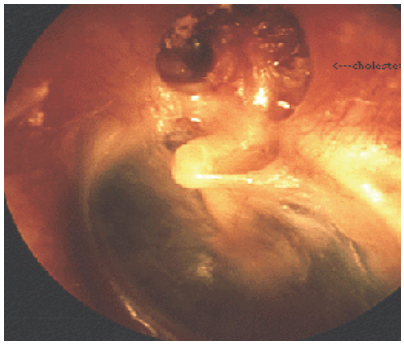
– hearing loss
– TM perforation
– CSOM
– retraction pockets
– cholesteatoma (3rd picture bellow)
– mastoiditis
– petrositis
– labyrinthitis
– adhesive OM (1st picture bellow)



– tympanosclerosis (2nd picture bellow)
– ossicular dyscontinuity and fixation
– facial paralysis

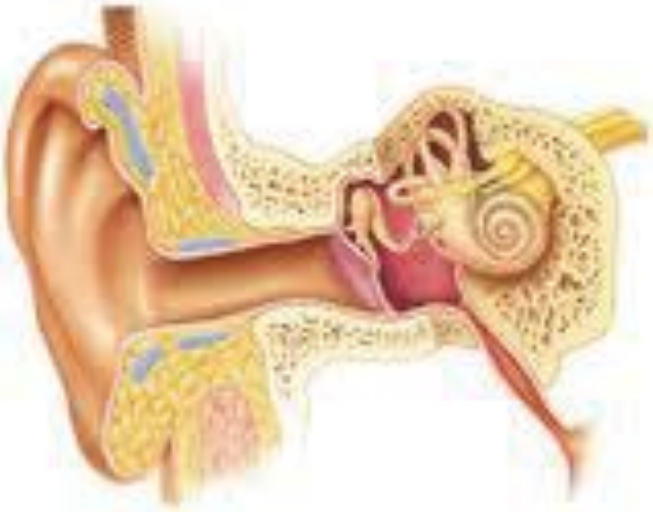


• Intracranial:
– meningitis
– extradural abscess
– subdural empyema
– focal encephalitis
– brain abscess
– lateral sinus thrombosis
– otitic hydrocephalus



430 Teams

Diseases of the Ear, Nose and Throat



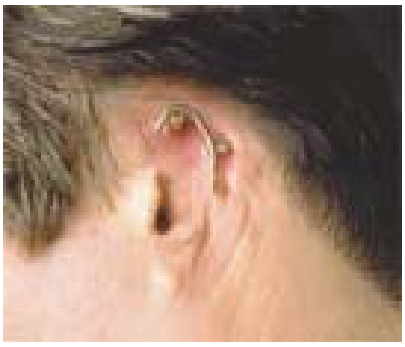
Lecture:

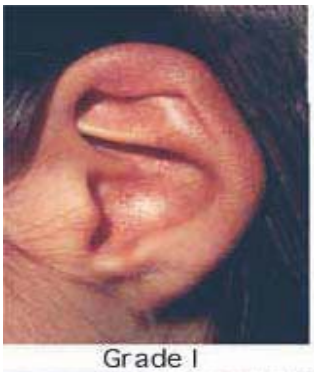
Ear I & II (Cases)

Done by: Hadeel S. Al-Madany

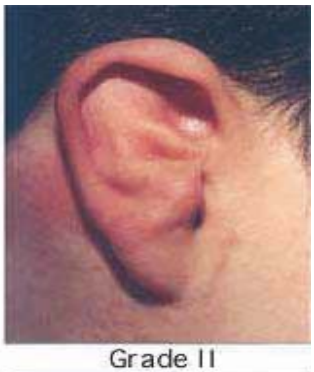
The slides were provided/ not provided by doctor (Abdulrahman Hagr Al-Ghamdi)
Important Notes in red
Copied slides in black
Your notes in green/ blue
Titles and subtitles in this color
Highlight possible MCQs mentioned or pointed by the doctor

Microtia:

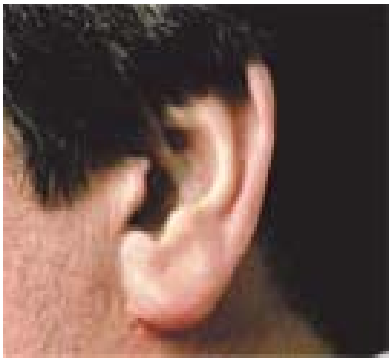


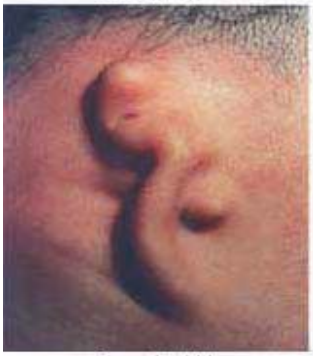


Grade I

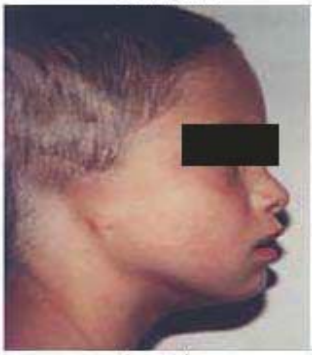


Grade II

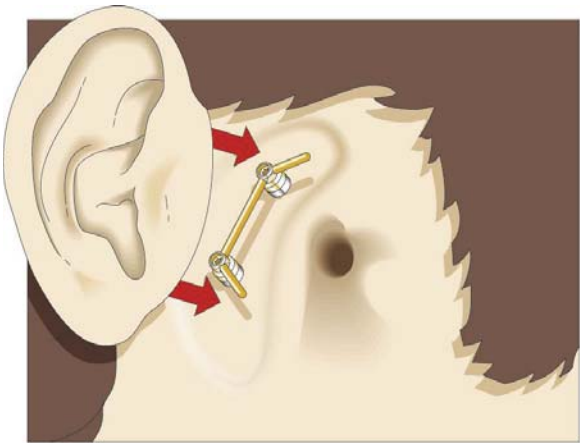




Grade III



Anotia



Microtia is a congenital deformity where the pinna (external ear) is underdeveloped. A completely undeveloped pinna is referred to as anotia.

Microtia can be unilateral (one side only) or bilateral (affecting both sides).

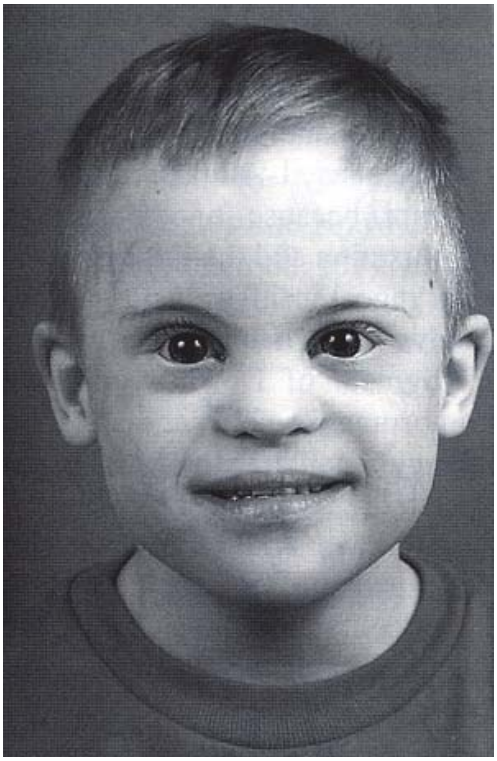
In unilateral microtia, the right ear is most commonly affected.

- Grade I: A less than complete development of the external ear with identifiable structures and a small but present external ear canal
- Grade II: A partially developed ear (usually the top portion is underdeveloped) with a closed [stenotic] external ear canal producing a conductive hearing loss.
- Grade III: Absence of the external ear with a small peanut-like vestige structure and an absence of the external ear canal and ear drum. Grade III microtia is the most common form of microtia.
- Grade IV: Absence of the total ear or anotia

Bat ear:

This is an abnormally protruding ear. The concha is large with poorly developed antihelix and scapha. The deformity can be corrected surgically any time after the age of 6 years, if cosmetic appearance so demands.

Down Syndrome:



- Trisomy 21
- 1 in 700 births
- Maternal age >35

Hearing Concerns

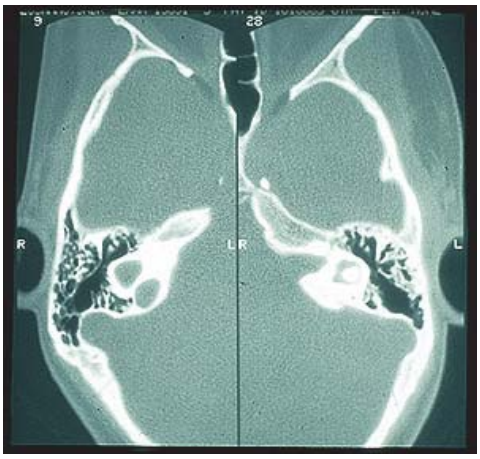
- Conductive hearing loss
  - more common
  - small pinna
  - stenotic EAC
  - eustachian tube dysfunction
  - ossicular fixation
- Sensorineural hearing loss
  - less common

Michel Aplasia

- 9 weeks gestation Cochlea fully formed
- Complete agenesis of IE
- Normal External and middle ear
- Affected ears are anacusic

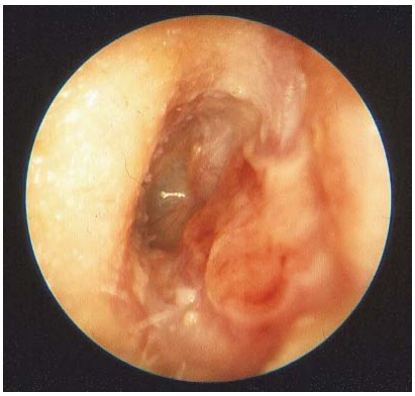
Mondini Aplasia

- Only the basal coil can be identified
- Interscalar septum is absent
- enlarged endolymphatic duct



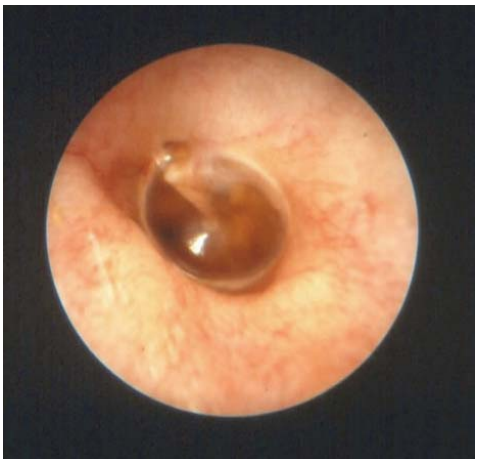
Cases:

Malignant (Necrotizing) Otitis Externa:



- 55 Y
- Left ear:
– Pain
– Discharge
- Left VII paralysis

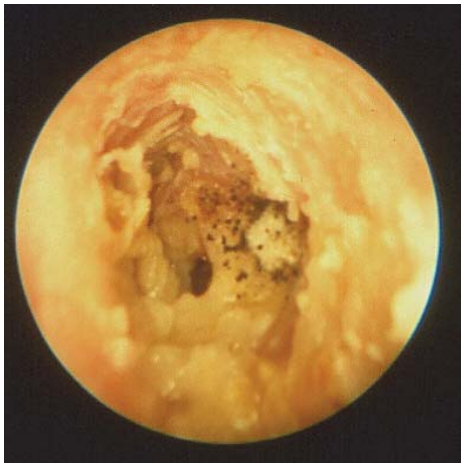
Secretory Otitis Media (Glue Ear):



- 3 Y
- Recurrent OM
- Hearing Loss

* pus behind the eardrum (bulging)

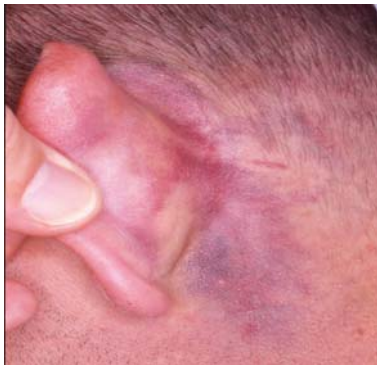
Otomycosis (Fungal infection):



- 45 Y
- Severe itching
- Pain
- Hearing loss

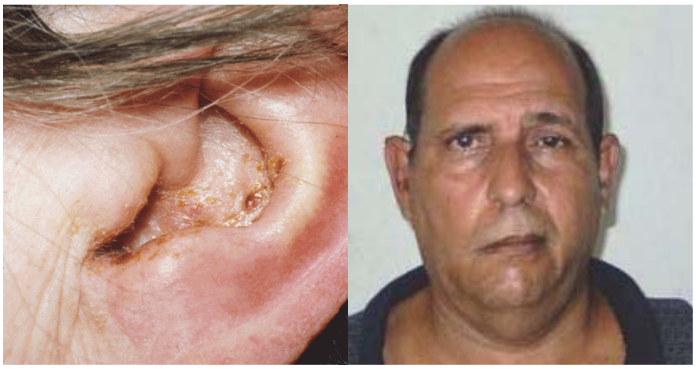
Fracture Base of Skull:

- MVA
- Left earache
- Hearing loss



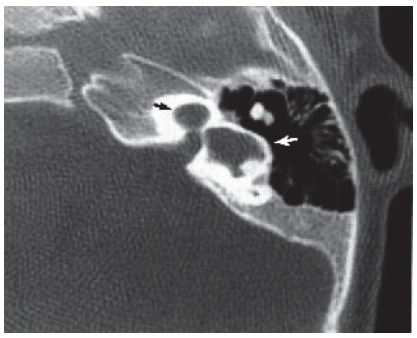
Ramsay Hunt Syndrome

- 55 Y
- Bilateral Earache
- Facial weakness



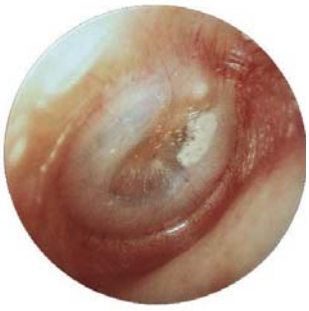
Mondini

- 4 Y
- Normal exam
- Rt moderate SNHL



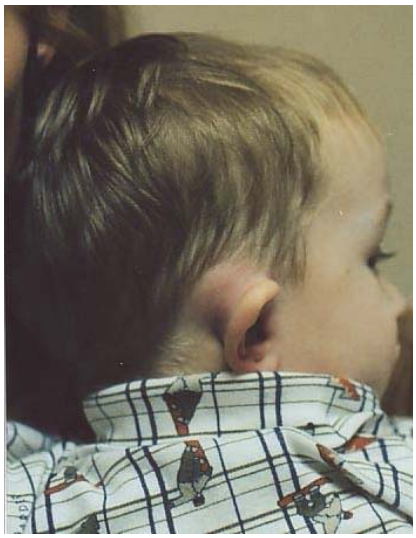
Otosclerosis vs Tympanosclerosis

- 33 y
- No hearing loss
- Ear exam→



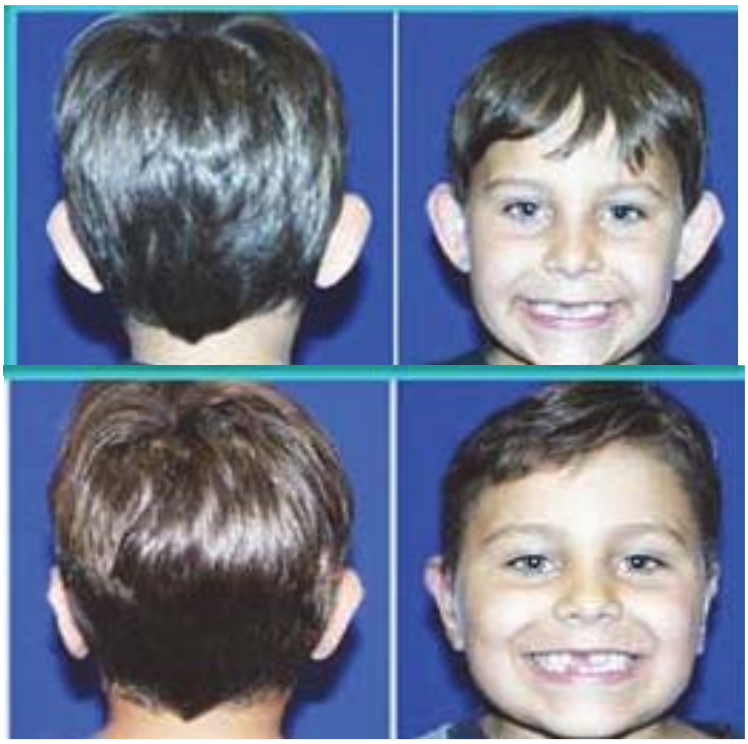
Mastoiditis

- 3 Y
- Fever
- Earache
- Irritability

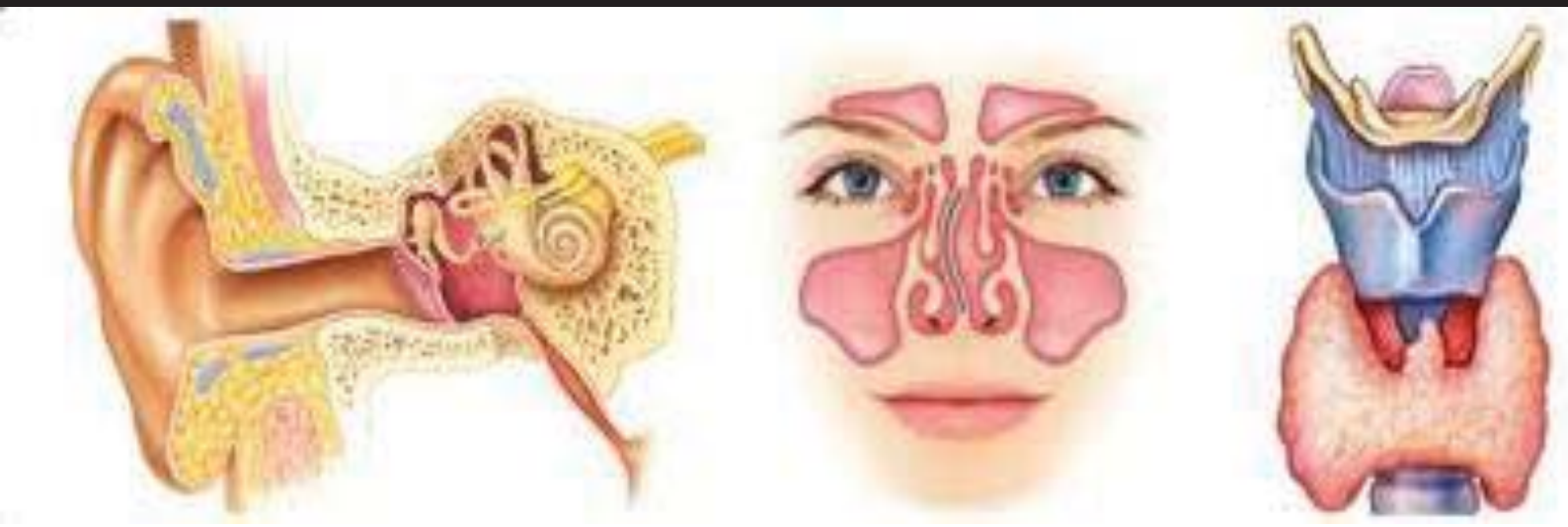


Bat ear

- 4 Y
- Era deformity



Diseases of the Ear, Nose and Throat



Lecture:

CHRONIC OTITIS MEDIA and MIDDLE EAR EFFUSION

Done by: Layan Akkielah

The slides were provided by Doctor Al- Essa
Source: recording, the slides , Toronto notes, ENT lecture notes, CURRENT diagnosis and treatment LANGE, Diseases of EAR, NOSE & THROAT PL Dhingra, Team 429
Important Notes in red
Slides and doctor notes are in **black**
Our notes in **green**

GROSS ANATOMY OF THE EAR

The ear is divided into three parts:

- **The External ear**
  It is formed by the Auricle or the Pinna, the external acoustic meatus and the tympanic membrane which acts as a partition between the external and the middle ear.

- **The Middle ear cleft**
  It is composed of:

➔ The Eustachian Tube:
It's a tube that connects the middle ear cavity to the nasopharynx.
The normal physiologic functions of the tube include:

1- Equalization of the middle ear pressure and the atmospheric pr ssure.
2- Prevent reflux of contents of the nasopharynx into the middle ear.
3- Clear secretions of the middle ear by both mucociliary transport and a "pump action" of the Eustachian tube.
Failure of any of these functions can result in Otitis Media.

➔ The middle ear cavity:

· The main content of the middle ear is ***air***.
· Three little bones known as the auditory ossicles: Mallues, Incus and Stapes.
· Muscles: tensor tympani muscle which is attached to the mallues, and the stapedius muscle which is attached to the stapes.
· Nerves

➔ The mastoid antrum and air cells.

The normal tympanic membrane is a pale gray semitransparent ovoid cone shaped disc located at the end of external canal. Cone-shaped reflection of light appears in the anterior inferior quadrant of the membrane

The tympanic membrane has three layers:
1- Outer epithelial layer which is continuous with the skin lining the meatus.
2- Middle fibrous layer which encloses the handles of malleus.
3- Inner mucosal layer which is continuous with the mucosa of the middle ear.

- **The Inner ear**
  It is composed of the Cochlea, which transforms sound into nerve impulses that travel to the brain, and fluid-filled semicircular canals (labyrinth), which send information on balance and head position to the brain.

MECHANISM OF HEARING

- The auditory ossicles conduct sound from the tympanic membrane to the inner ear. Sound waves picked up by the external ear vibrate the tympanic membrane, which in turn mobilizes the ossicular chain of the middle ear. The footplate of the stapes applies a mechanic pressure on the oval window and the vibration reaches the perylymphatic fluid of the cochlea. Inside the inner ear these vibrations are converted to nerve signals that are carried by the auditory nerve to the brain.

CHRONIC OTITIS MEDIA

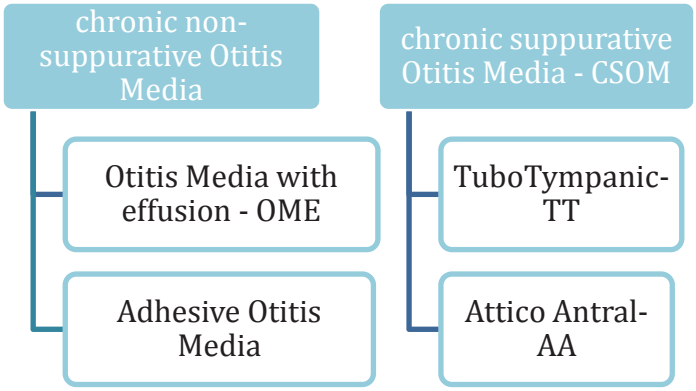
Definition:

- **Chronic Otitis Media** is an infection involving a part of the ***middle ear cleft*** or all its components *that is persistent for* ***more than 3 months***.

Route of infection:

1- Local: From one part to another.
2- Via the Eustachian Tube: An Upper respiratory tract infection can spread to the ear through the Eustachian tube.
3- Blood – brone.
4- Lymphatic spread

Classification of chronic otitis media:



Note:
There are many different classifications. This classification is classified according to the cause as the doctor said.

Suppurative = Puss formation.

- The tympanic membrane is ***intact (not perforated)*** in ***Chronic non-suppurative otitis media***, while in ***chronic suppurative otitis media*** it is ***not intact (perforated).***
- Chronic suppurative otitis media is divided ***clinically*** into two types:

1- TuboTympanic, which is also known as the ***Safe*** type, has no risks of serious complications.
2- AtticoAntral, which is also known as the ***Unsafe*** type, has a high risk of developing complications.

- To have a discharge coming through the external canal the membrane has to be perforated.

1. CHRONIC NON-SUPPURATIVE OTITIS MEDIA

1.1 OTITIS MEDIA WITH EFFUSION

Other terms:

- Secretary middle ear, Syn. Serous Otitis media, Mucoid otitis media , Glue ear, Catarhal otitis media, Sero-mucinous otits media.

Definition

- It is a condition characterized by the presence of non-purulent fluid (sterile) within the middle ear cleft that persists for more than 3 months with an intact tympanic membrane.

Prevalence:

- It is a very common disease especially in children at age of 5 years and below.
- Almost everyone during one time or another will have one attack of OME, even though it's self limiting and can't be recognized.
- Between 20% and 50% of children do have OME at some time between 3 and 10 years of age.
- Two peaks at 2 and 5 years of age.

Pathogenesis :

- Normally, the middle ear is aerated all the time by the eustachian tube. If the eustachian tube was obstructed for any reason, the normal mucosa of the middle ear will start absorbing the air, which is one of its normal functions, and will create negative pressure in the middle ear. Due to that, the tympanic membrane is retracted and therefore its movement is hindered causing conductive hearing loss. Also, it becomes unable to drain the fluid that started to accumulate due to the increased secretory activity of the middle ear mucosa

Histopathology:

- Changes in the mucosa:
  - Vasodilatation and mono nuclear cell infiltration.
  - Metaplasia of the epithelium to ciliated columnar epithelium
  - Mucus secreting gland formation.
- Formation of fluid in the middle ear:
  - Transudate.
  - Exudates.
  - Secretions.

Risk factors:

Environmental factors:

- It is more common in ***winter*** months.
- It's more common in ***developing countries*** with low socio-economic status.
- ***Exposure to cigarette smoke increases*** the risk of developing the infection. Passive smoking results in inflammation of the mucosa of the middle ear cleft as well as impaired mucociliary clearance, which will potentially facilitate bacterial colonization and congestion.

Genetic factors:

- Both females and males are at ***the same risk*** of getting OME.
- It's more common in people with ***fair skin.***
- Considering the variations in anatomy of the Eustachian tube between adults and children, Otitis media with effusion is ***more common in children*** where the Eustachian tube is shorter, wider and more horizontal. This will make it easier for organisms from URTI to access the middle ear.
- Otitis media is almost universally seen in children with ***Cleft palate***. Because the tensor veli palatine muscle lacks its normal insertion into the soft palate, it is unable to open the Eustachian tube properly on swallowing or chewing.

Normally, the Eustachian tube is closed at rest. It opens either actively by contraction of muscles as in swallowing or by Valsalva maneuver, which is increase in the pressure of the Eustachian tube and middle ear by forcible exhalation effort against occluded nostrils and closed mouth, or passively.

- ***Nasopharyngeal anatomical abnormalities:*** Eustachian tube obstructions.
- Allergy. Seasonal or perennial allergy to inhalants or foodstuff is common in children. This not only obstructs Eustachian tube by oedema but may lead to increase secretory activity as middle ear mucosa acts as a shock organ in such cases.

Important note:

- Case:
  A healthy adult came to your clinic complaining of sudden gradual **unilateral** hearing loss. He has no history of previous infections (whether URTI, tonsillitis, adenoiditis..etc).
  On examination you find fluid in the middle ear cleft.
  Diagnosis: otitis media with effusion due to an obstruction in the Eustachian tube caused by ***Nasopharyngeal carcinoma.***
- Always think of nasopharyngeal carcinoma in adults with an explained otitis media with effusion.
- Glue ears and unilateral cervical lymphoadenopathy can be the first clinical presentation of that carcinoma in some patients.
  (the first sign will be streaks of blood in the throat, but no one would see that)
- Patients with nasopharyngeal carcinoma will present with conductive hearing loss and a lump on the neck.

Etiology

- Unresolved AOM acute otitis media: Inadequate Antibiotic therapy will not resolve the infection completely. There will be fluid remained and remnants of bacteria.
- Mainly Eustachian tube dysfunction:
  Causes that will result in an obstruction in the Eustachian tube and accumulation of fluid:
  - The chronic inflammation itself can cause obstruction of the tube by formation of oedeam around the esustachian tube.
  - Nasopharyngeal carcinoma in adults but it is rare.
  - In children:
    URTIs including sinusitis
    Tonsillitis: enlarged tonsils will obstruct the movement of the cleft palate.
    Adenoiditis: either by inflammation by hypertrophy of Lymphoid tissue or enlarged adenoids which can cause obstruction of the tube.
  - Cleft palate
  - Poor muscular function: Diseases that causes hypotonia such as Down syndrome.
  - Baro-trauma: It means air pressure changes, such in rapid descent during flights or during diving. If the atmospheric pressure reaching is ***higher than 90 mm of Hg*** (Doctor mentioned if around 80 mm of Hg but he said he wasn't sure), Eustachian tube gets blocked. It will be associated with severe pain and retracted tympanic membrane due to the negative pressure.

Symptoms:

- ***Hearing impairment:***
  - It is the most common symptom in children with OME.
  - How will the parent notice their child's hearing loss? The child will not respond to calls and will only listen to high volumes.
  - It happens due to the restriction of movement of the tympanic membrane and ossicles because of the retraction of the membrane and fluid accumulation.
  - In OME the patient suffers of *conductive hearing loss.*
  - *It might cause speech impairment after a long time, but this is rare.*

There are three types of hearing loss, depending on the area affected in the ear:
1- Conductive hearing loss: It is caused by any disease affecting the external ear or the middle ear cleft.
2- Sensorineural hearing loss: results from lesions of the cochlea (sensory type) or VIIIth nerve (neural type). It is associated with speech impairment.
3- Mixed hearing loss: elements of both conductive and sensorineural are present in the same ear.

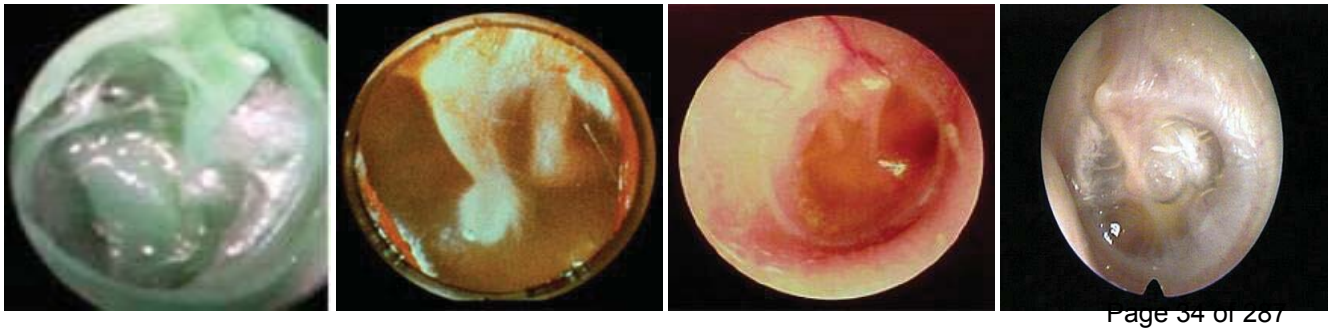
- **Otalgia** is sometimes present. Such as OME caused by Baro-trauma.
- **Fluid sensation** with the movement of the head.

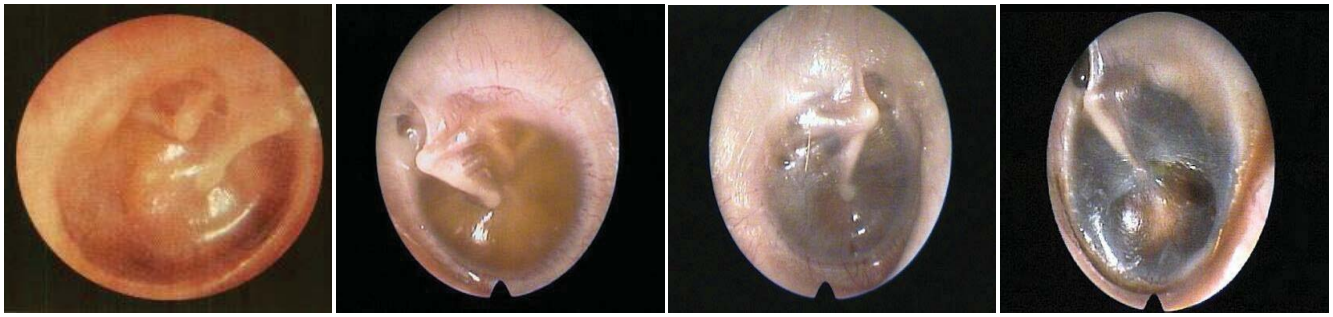
Diagnosis:

➔ **Otoscopic findings:**

The tympanic membrane is often dull, opaque, might vary in the degree retraction, and appears yellow in early stages, grey or bluish in late stages. There will be loss of light reflex.
The handle of mellues will seem horizontal.

Causes of blue tympanic membrane:
- Long standing OME.
- Blood in the middle ear - hemotympanum





➔ **Tuning fork tests**: shows conductive hearing loss.

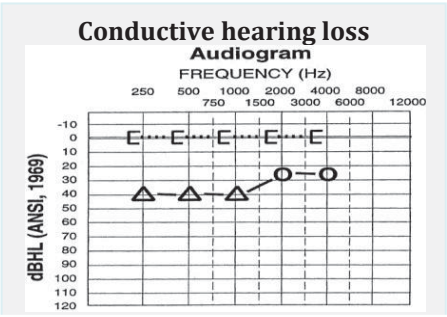
- ***Weber's test:***
  The base of a vibrating tuning fork is held on the middle of the skull and the patient is asked whether the sound is heard centrally or is referred to one or other ear.
  Interpretation:
  In conductive hearing loss the sound is heard in the deafer ear but in sensorineural deafness the sound is heard is the better hearing ear.
  If it is heard in both ears equally, this is normal.

In webber's test the most sensitive areas are the upper incisors, lower incisors or the mandible.

- **Rinne's test:**
  This test compares the relative effectiveness of sound transmission through the middle ear by air conduction, AC, and bypassing the middle ear by bone conduction, BC. A tuning fork of 512 Hz is struck and held close to the patient's ear (AC), the base is then placed firmly on the mastoid process behind the ear (BC), the patient is then asked whether it is heard better by BC or AC.

Ideally, in the exam while student is performing renne's test he/she must say:
I will put the vibrating fork next to the patient's ear and wait until he stops hearing anything, then place it on the bone.

Interpretation of Rinne's test:
If AC > BC it is considered a positive test, means that middle and outer ear are functioning normally.
If BC>AC it is considered a negative test, means that there is a defective function of the outer or the middle ear (conductive hearing loss).

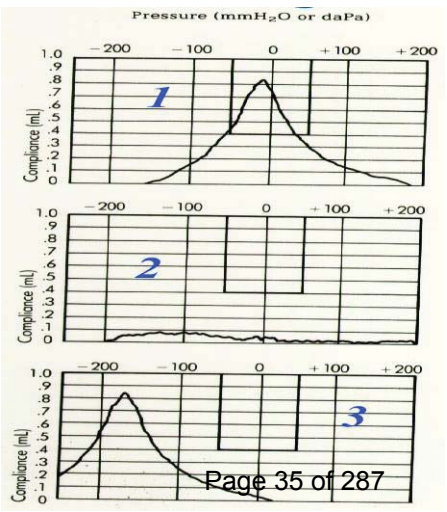


➔ **Pure tone audiogram:**

- Can be done for children older than 4-5 years.
- -10 – 25 dB HL = Normal range
- If there is an air- bone gap it's a conductive hearing loss.

➔ **Myringotomy**

➔ **Tympanometry:**
The picture aside describes the different types of curves seen in a tympanometry:



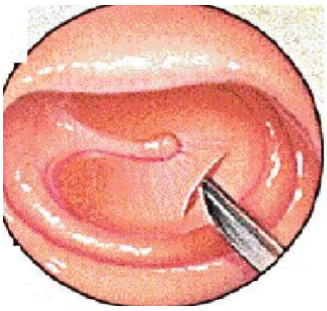
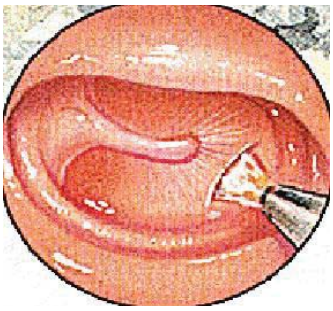
- The first curve -1-: Type A, it defines a normal curve which will be at zero level.

- The second curve -2-: Type B, a flat or dome-shaped graph Seen in middle ear fluid or thick tympanic membrane.

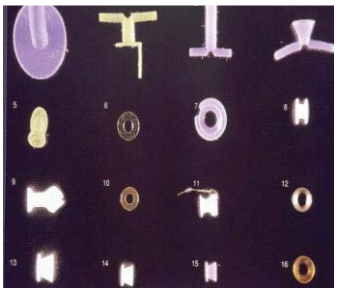
- The third curve -3-: Type C, is in the negative side. Seen in the eustachian tube dysfunction, retracted tympanic membrane and may show some fluid in the ear.

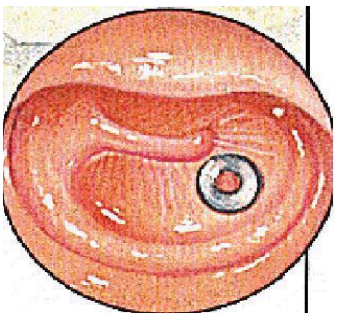
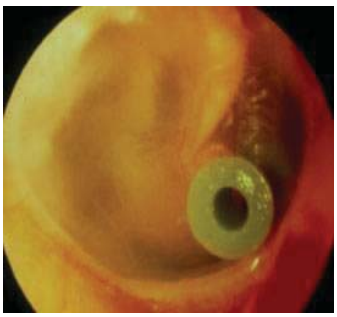
Treatment

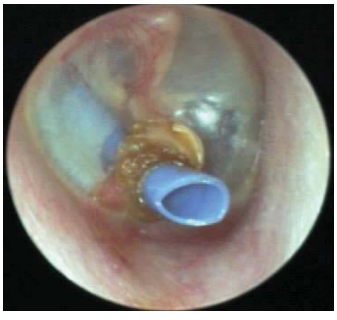
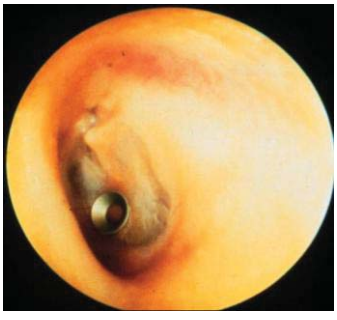
- Treatment of the cause if feasible
- *Observation:* OME can be a self-limiting disease.
  Spontaneous resolution occurs in some cases. Observing the patient for three months from the beginning of the onset or the diagnosis is advisable before interfering.
- *Medical treatment*
  - Antibiotics: useful in cases of unresolved acute suppurative otitis media (fever, discharge)
  - Decongestants (local or systemic): it is given to help relieve the oedema of the eustachian tube.
  - Antihistamine for allergy.
  - Steroids, it is controversial, nobody will give steroid.
- *Surgical interference* is done when there is no improvement by medications:
  - Surgical treatment of causative factors (adenoidectomy, tonsilictomy)
  - Myringotomy and aspiration are done to repair the tympanic membrane and equalize the pressure.
  - Ventilation tubes/ myringotomy tubes/grommets:
    ➔ When the tube is inserted, it is left in place until it comes out by itself. It might take from six months up to two/three years until it falls off by the shedding process of the tympanic skin leaving the tympanic membrane intact.

This is not the right incision, the incision should be anterior inferior and it should be radial not transitional like showed in the pictures. Suction should be done, if suction is not done and it is clear you put the ventilation tube.



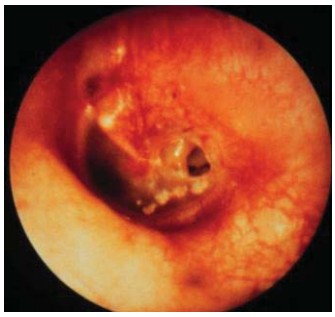




There are different types of tubes depending on the case. We have two main types:

1. Short-term tubes (eg, grommets), which remain in the tympanic membrane for an average of 12 months.
2. Long-term tubes(eg, T-tubes),which can remain for several years, and in cases such as cleft palate.

- Complications of T-tubes: It may cause perforation after removing it. So we should consider using the simple grommets tubes before jumping into the decision of using the T-tubes.



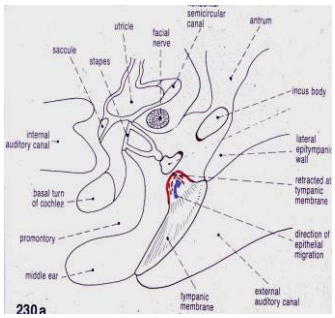
➔ complications of ventilation tube insertion:
- Infection: the ventilation tube will act as a foreign body. If there was no improvement with the Antibiotics given, removal of the tube is required.
- Blockage: It could be blocked by blood, mucus or wax. It has to be cleared because if it got obstructed it won't function.
- Extrusion: Bulging out.
- Scarring of the tympanic membrane in late stage and formation of tympano scarring patch.
- Tympanosclerosis: hyalinization of the fibrous tissue followed by calcium deposits in the tympanic membrane and around the ossicles and their joints, which causes their fixation.
- Persistent perforation in a late stage.

Factors affecting treatment:

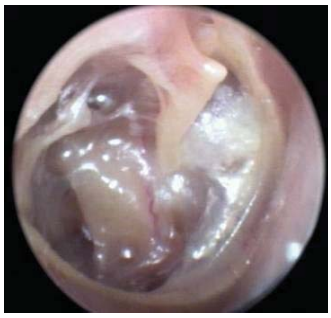
- Age: In young children, observation is required before intervention because the disease might resolve by itself.
- Duration: In a long standing disease, intervention is required. Also, if the child is in a preschool age or if it is causing speech impairment intervention is required.
- Unilateral or bilateral.
- Degree of hearing impairment
- Previous treatment: Having a history of previous tube surger. Surgery is then required.
- Associated conditions such as cleft palate and myotonia in Down syndrome.
- Intervention is required when there are severe changes in the tympanic membrane such as severe retraction of tympanic membrane.

SEQUELAE

- Spontaneous resolution:
  50% of the cases resolve within 3 months.
  Only 5% persists for more than 12 months.
- Tympanosclerosis.
- Scarring, retraction, atelectasis of the middle ear and ossicular necrosis.
- Cholesteatoma and cholesterol granuloma.



1.2 CHRONIC ADHESIVE OTITIS MEDIA



Definition:

Formation of adhesions in the middle ear (between the medial wall of the middle ear and tympanic membrane):1. After reactivation and subsequent healing of either CSOM or OME. 2. Due to severe negative pressure due to ome or csom with a perforated drum which is healed by a thin membrane, and eustachian dysfunction that will cause severe retraction of the membrane.

Clinical features:

1- Previous history of CSOM or OME.
2- Deafness/Conductive hearing loss is usually the main symptom.

3- Tympanic membrane shows various structural changes such as severe retraction and very prominent ossicles.

Treatment

- Observation, every 6 months if there were any complications.
- Surgical treatment: Ventilation tube insertion.
- Hearing aid will help a lot

2. CHRONIC SUPPURATIVE OTITIS MEDIA (CSOM)

Definition:

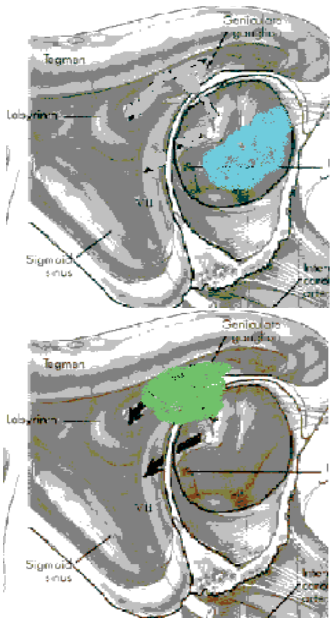
Chronic suppurative otitis media is a long standing infection of a part or whole of the middle ear cleft characterized by ***ear discharge (Otorrhea)*** and ***permanent perforation*** of tympanic membrane.

Etiology

- Environmental: It is common in humid and hot areas.
- Genetic, It may be related.
- Previous history of persistent AOM
- Upper respiratory tract infections.
- Eustachian tube dysfunction

Clinical- pathological types of CSOM:

Chronic suppurative otitis media is divided ***clinically*** into two types:



1- TuboTympanic (TT):
   - Also known as the ***Safe*** type.
   - It involves the anterior inferior part of middle ear cleft.
   - Perforation is central.
   - It has no risks of serious complications.

2- AtticoAntral(AA):
   - Also known as the ***Unsafe*** type.
   - It involves the posterosuperior part of the cleft such as the attic, antrum and mastoid.
   - Perforation could be attic or marginal.
   - It has a high risk of developing complications.
   - It is associated with Cholesteatoma.

Pathology:

- Signs of suppurative infection
  - Discharge & perforation
  - Chronic inflammatory reaction in the mucosa and the bone (ostietis)
- Signs of healing attempts:
  - Granulation tissue & polyps
  - Fibrosis & tympanosclerosis
- Cholesteatoma (attico-antral type)

Clinical Features::

Common presentation of uncomplicated chronic otitis media:

- *Otorrhea*: - Intermittent, profuse, mucoid & odorless in TT type
  Persistent, scanty, purulent & foul smelling in AA type.

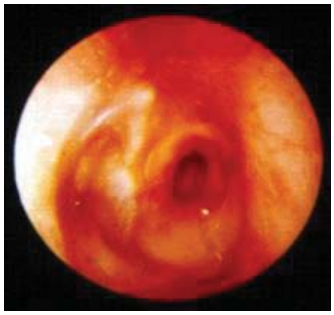
- *Deafness*: conductive hearing loss.
- Tinnitus
- Any other symptom other than these three is considered a sign of complication.
  Ex: Headaches (could indicate intracranial complications),
  Vertigo (indicates involvement of the inner ear),
  Facial movement abnormalities (indicates involvement of facial nerve),
  Vomiting and fever (indicates involvement of the balance system and increased intracranial pressure).

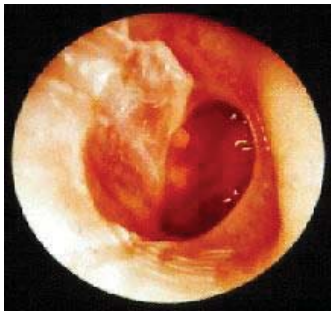
Otorrhea is the medical term for ear discharge.
- It could be blood, ear wax or fluid coming through the external canal.
- Characteristics of the discharge according to the cause:
  1- Purulent discharge seen in acute/chronic otitis externa.
  2- Mucopurulent discharge is seen in middle ear diseases.
  3- Clear fluid (CSF leakage) is seen with skull traumas (especially in RTA).
  4- Bloody discharge is seen with base of skull traumas, acute otitis media, or malignancy in middle ear.

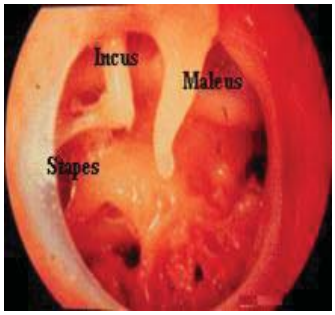
Otoscopic examination

- Discharge:
  - Present in TT type if active but may be absent
  - Usually it is persists in AA type.
- Perforation:
  - Central perforation : in TT type.
  - Marginal or attic in AA type with cholesteatoma.
- Polyps, granulation tissue, tympanosclerosis

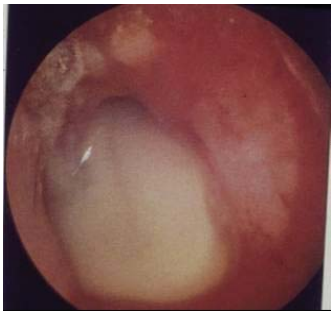
Perforation in TT CSOM:


Central perforation

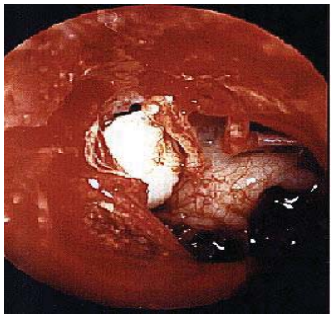

Subtotal perforation

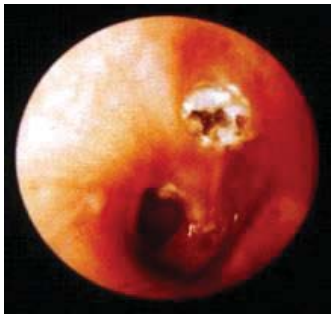


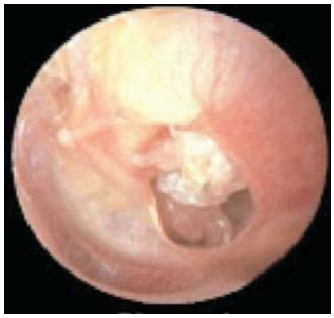
Almost total perforation

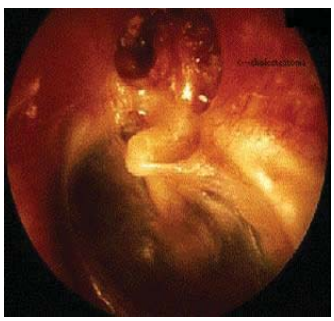

Active CSOM with pure purulent discharge

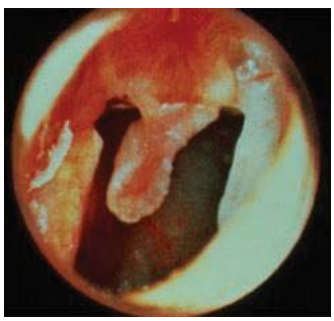
Perforation in AA CSOM:



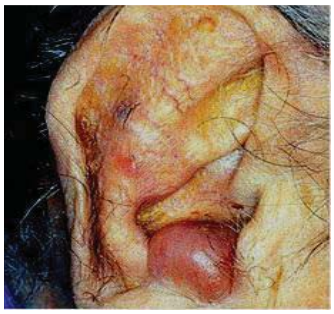


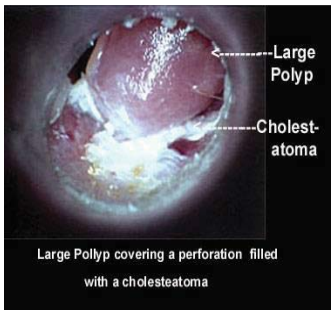


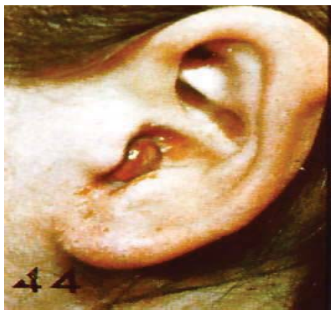




Polyps protruding from the auricle (it's extreme)







Bacteriology

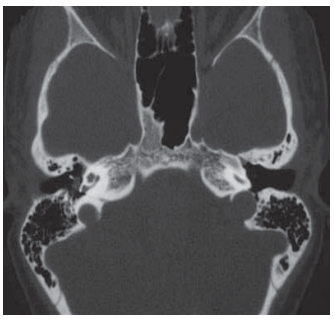
- Aerobes:
  Pseudomonas aergunosa (it is the most common organism causing the infection)
  Staphylococcus (gram positive)
  Proteus (gram negative)
  Escheria coli (gram negative)
  Klebsiella (gram negative)
- Anaerobes:
  Bacteroides fragillis
  Peptostreptococcus

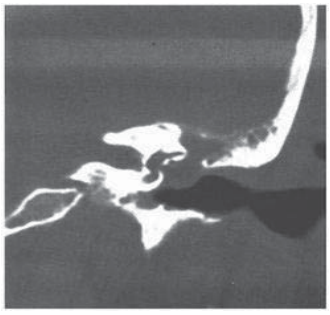
Infections from bacteroids and anaerobes with osteitis are usually associated with a bad odor in case of the unsafe type.

Investigations.

- Audiometry and tunning fork test (it is done to measure the level of hearing loss and type of hearing loss)
- Bacteriology
- Imaging Ex: mastoid x-ray, CT done usually in the Atticoantral type to check for complications such as cholesteatoma.

Congenital cholestetoma Cloudy middle ear in CSOM Cholestetoma with attic erosion





Treatment:

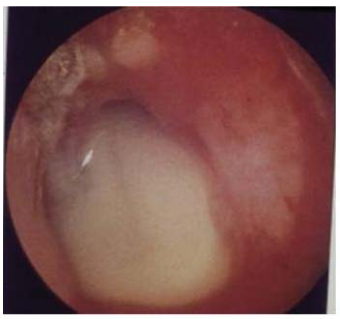
- Depends on the type and presentation.

1- Management of Tubotympanic CSOM:

The aim of treatment is to control infection and eliminate ear discharge, and at later stage, to correct hearing loss by surgical means:

- Conservative management (in active infection until it becomes inactive - the ear dries out):
  - The mainstay of treatment is regular ear toilet. It is removal of ear discharge and debris from the ear.
  - Ear drops

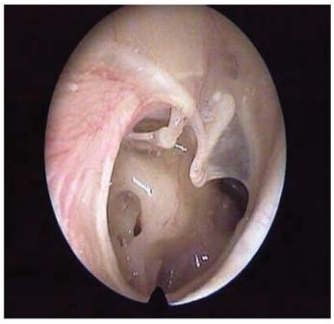
- Precautions: patients are instructed to keep water out of the ear during bathing and swimming. Rubbers inserts can be used. Hard nose blowing should be avoided.
- Treatment of contributory causes.
- Surgical treatment: removal or aural polyp or granulation, if present. It will facilitate ear toilet and permit ear drops to be used effectively.
- Antiobiotics should be given according to culture results (e.g. clindamycin or second generation cephalosporins).



Active TT type

Conservative treatment

→



Inactive TT type

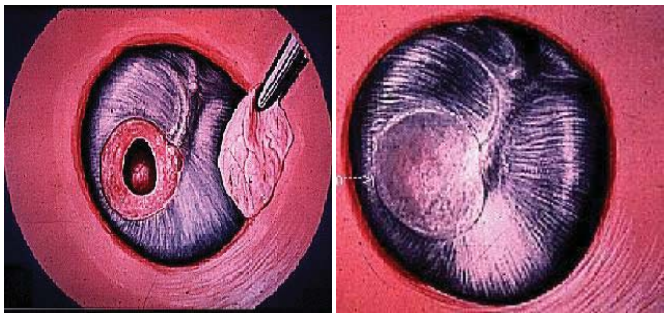
- Reconstructive surgery:
  Myringoplasty: - it's an operation performed to repair the tympanic membrane.
  Once ear is dry, myringoplasty with or without ossicular reconstruction can be done to restore hearing loss. Closure of perforation will also prevent repeated infection from the external ear.

Tympanoplasty

It is an operation performed to eradicate disease in the middle ear cavity and to reconstruct the hearing mechanism. It is done by closing the perforation with a graft (most commonly from temporalis fascia) and repairing the ossicles.

Aims of tympanoplasty

- To close the perforation,
- To prevent re-infection.
- To improve hearing.



2- Treatment of attico antral CSOM:
Surgery in this type is the mainstay of management.
Removal of cholesteatoma by mastoid operation
Mastoidectomy:
It is the removal of the mastoid contents

- Cortical mastoidectomy → converting the mastoid antrum and air cells into one cavity without disturbing the existing middle ear contents.
- Radical mastoidectomy → An operation in which the mastoid antrum and air cells, attic and middle ear are converted into common cavity, exteriorized to the external canal.

The tympanic membrane, malleus and incus are removed leaving only the stapes in situ (keep stapes in situ to avoid causing sensorineural hearing loss)

- Modified radical mastoidectomy:
  An operation in which the mastoid antrum and air cells, attic and middle ear are converted into common cavity, exteriorized to the external canal. The tympanic membrane and ossicles remnants are retained.

Types of mastoidectomy:

- Canal wall up: they leave the mastoid cavity open into the external canal so that the diseased area is fully exteriorized.
- Canal wall down: here disease is removed by combined approach through the meatus and mastoid but retaining the posterior meatal wall intact, thereby avoiding an open mastoid cavity.

Aims of radical & modified radical mastoidectomy:

- Safety
- Dry ear
- Preserve hearing

Types of incisions:
1- Behind the auricle
2- Endo- oral
3- Transcanal (not used anymore)

Complications of mastoidectomy:

- Facial paralysis (facial nerve injury during surgery)
- Hearing impairment.

CHOLESTEATOMA

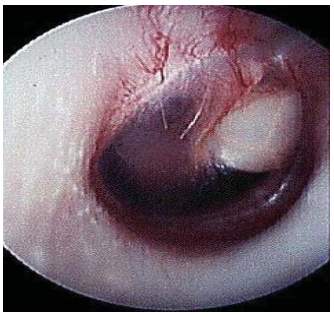
Definition:

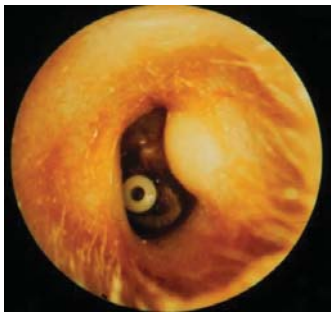
- It's the presence of a desquamating stratified squamous epithelium (skin) in the middle ear.
- It has tendency of multiplying, forming granulation tissue underneath and producing letic enzymes which causes local destruction.

Pathogenesis:

It could be congenital or acquired

- Congenital cell rests: skin trapped in the middle ear cleft since birth.
- Pocket formation: Invagination of the tympanic membrane from the attic or posterosuperior part of pars tensa in the form of retraction pockets. The outer surface of the tympanic membrane is lined by stratified squamous epithelium which after invagination forms the matrix of cholesteatoma and lays down keratin in the pocket.
- Metaplasia of the middle ear mucosa due to repeated infections and transforms into squamous epithelium.
- Epithelial invasion: the epithelium from the meatus or outer drum surface grows into the middle ear through a pre existing perforation. Ex: Iotrogenic External canal cholesteatoma (injury of the external canal in myringotomy then skin gets trapped behind the tympanic membrane).
- Basal cell hyperplasia. The basal cell of germinal layer of skin proliferate under influence of infection, and lay down keratinising squamous epithelium.



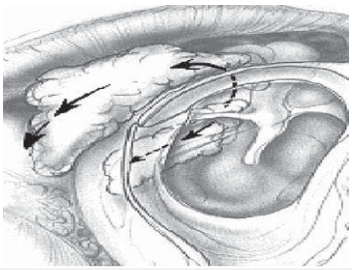


Classification of cholesteatma:

- Congenital cholesteatoma arises from the embryonic epidermal cell and rests in the middle ear cleft or temporal bone.
- Acquired:
  - Primary called primary as there is no history of previous otitis media or perforation.
  - Secondary in these cases there is a pre existing perforation and it's often associated with posterosuperior marginal perforation or sometimes large central perforation.

Effect of Cholesteatoma

- Keratin encourages persistence of the infection because the cholestatoma is there all the time and could cause discharge most
- Matrix causes bone erosion and ostitis associated



conclusion

Otitis media with Effusion:

- OME is very common in children.
- Etiology is associated with Eustachian tube dysfunction and/or chronic infections (URTI).
- In adults: Nasopharyngeal pathology should be considered.
- Most cases resolve spontaneously.
- Conservative treatment is of doubtful value.
- Ventilation tube insertion restore hearing in selected cases. It is the main stay of treatment in chronic cases of OME.

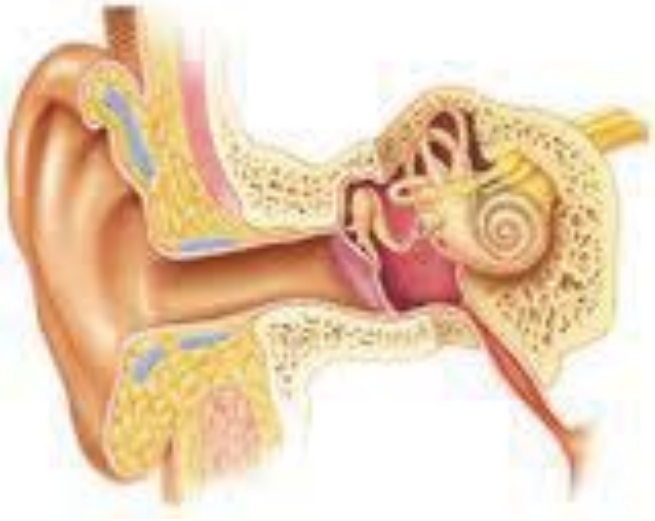
Chronic Suppurative otitis media

- In Tubotympanic type (safe type) the discharge is usually copious, intermittent and odorless. The perforation is central. Treatment is conservative (if there is an active infection) followed by tympanoplasty to prevent re-infection and improve hearing.
- In the AtticoAntral type (the unsafe type) the discharge is usually scanty, persistent and of bad odor. The perforation is attic or marginal with cholesteatoma. Treatment is by mastoidectomy to provide safety and dry ear.

- Middle ear effusion → myringotomy and ventilation tube insertion.
- Safe type CSOM → tympanoplasty
- Unsafe type with cholestetoma → mastoidectomy

430 Teams

Diseases of the Ear, Nose and Throat



1st Lecture:

COMPLICATIONS OF SUPPURATIVE OTITIS MEDIA

Done by: Ahlam Al-Sulaiman

The slides were provided
(the Dr Alaisa slides / dr notes during the lec + Dr Alsnosi notes durin the lec / diseases of nose, ear and throat, Lecture note books)
Important Notes in red / the doctor notes during the lecure in dark blue and Dr alsnosi notes the purple colure
Copied slides in black

2 Complication of suppurative otitis media

ROUTES OF SPREAD:

- Direct extension e.g. facial nerve paralysis
- Thrombophlebitis by blood
- Normal anatomical pathways (Eustachian tube (ET) → nasophrynx, Perforated TM→External Auditory Canal)
- Non anatomical bony defects (tegmen tympani →intracranial structures, mastoid tip→ Neck)

COMPLICATIONS OF SUPPURATIVE OTITIS MEDIA:

A. Extracranial complications
B. Cranial (intra-temporal) complications ((within the middle ear cleft+ Temporal bone))
C. Intracranial complications

*Note : Don't confuse between Cranial and Intracranial they are different

A. EXTRACRANIAL COMPLICATIONS:

- **Otitis externa**: by the perforated TM the pus extend to the external canal, Sx: painful ear by touch Tx: by Abx.
- **Retropharyngeal abscess:** ET → nasopharynx → (sub mucosal) of posterior wall of the pharynx.
  Diagnosis: x- ray , Treatment: **Drainage** according to the type : Acute (the drainage from **inside** or outside) Chronic mostly related to TB infection (drainage from **out side** to prevent the spread to the lungs)
- **Septicemia:** Emergency case but less incidence due the Antibiotics.

Bactermia: the presence of bacteria in the boold / ***Septicemia***: the infection resulted from the bacteria toxins

B. CRANIAL (INTRATEMPORAL) COMPLICATIONS:

- Acute mastoiditis
- Petrositis
- Facial nerve paralysis
- Labyrinthine fistula and labyrinthitis

*ACUTE MASTOIDITIS:

Involvement of the bone of the mastoid air cells by **acute suppurative inflammation**

Pathway: Middle ear → attic → Mastoid

Acute Mastoiditis Pathology:

1. **Production of pus under tension:**
   Swollen mucosa of the antrum and attic also impede the drainage system resulting in accumulation of pus under tension
2. **Hyperaemic decalcification:**
   Hyperaemia and engorgement of mucosa causes dissolution of calcium from the bony walls of the mastoid air cells (hyperaemic decalcification).

*Both these processes combine to cause destruction and coalescence of mastoid air cells, converting them into a single irregular cavity filled with pus (Empyema of mastoid).

Symptoms:

They are similar to that of acute suppurative otitis media.
it is the **change in the character of these symptoms** which is significant and a pointer to the development of acute mastoiditis.

- *Pain behind the ear:*
  Pain is seen in acute otitis media but it subsides with establishment of perforation or treatment with antibiotics. It is the persistence of pain, increase in its **intensity** or **recurrence of pain**, once it had subsided. These are significant pointers of pain.

- *Fever:*
  It is the persistence or recurrence of fever in a case of acute otitis media, in spite of adequate antibiotic treatment that points to the development of mastoiditis.

- *Ear discharge:*
  In mastoiditis, discharge becomes **profuse** and increases in **purulence**.

*__Note__: Any persistence of discharge beyond three weeks, in a case of acute otitis media, points to mastoiditis
((It happens more as a complication ***after acute OM*** than the chronic))

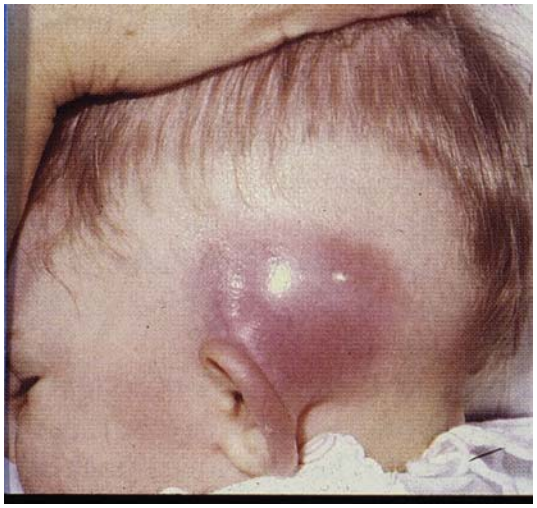
Signs:

- **General constitutional manifestations:**
  Patient appears ill and toxic with low-grade fever. In children, fever is high with a rise in pulse rate.
- **Tympanic membrane changes:**
  - Usually, a small perforation is seen *in pars tensa with congestion of the rest* of tympanic membrane.
  - Perforation may sometimes appear as a *nipple-like protrusion.*
  - Sometimes, tympanic membrane is *intact but dull and opaque* especially in those who have received inadequate antibiotics.
- **Sagging of posterosuperior meatal wall (seen by the otoscope)**
- **Otorrhea and reservoir sign.**
- **Retroauricular tender red swelling.**
- **Abscess: (These Abscess considered *extracrinal complication*)**
  1. **Subperiosteal abscess (postauricular abscess):** is the ***commonest abscess that forms over the mastoid.*** Pinna is displaced forwards, outwards and downwards.

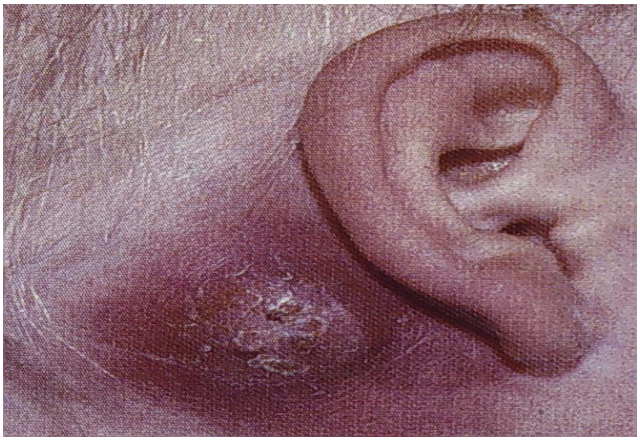
Positive reservoir sign i.e. rapid re-accumulation of discharge after cleaning of the ear.

The extracrinal complications are rare.
If the patient present with acute mastoiditis with postauricular abscess ,it is an Emergency case and you have to admit the pt and do CT to know the extent of the bone destruction ,and if you suspect intracrinal involvement do MRI .

2.Bezold's abscess: pus breaks through the mastoid tip and forms an abscess in the *neck*. Just lateral to the sternocleidomastoid



3.Zygomatic mastoiditis: result on swelling over the zygoma .

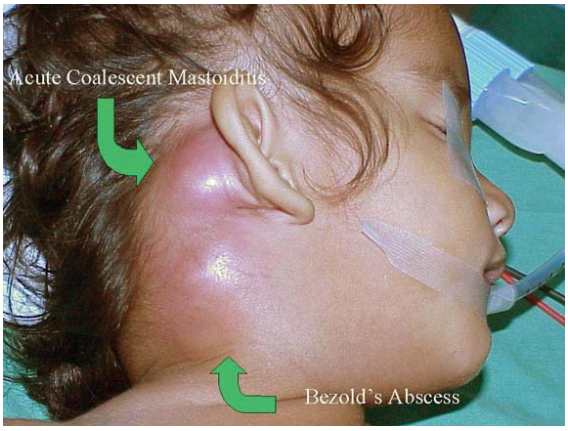


Postauricular Abscess **Lymphadenitis**

Subperiosteal abscess ((IMP SAQ))

And they asked about the management ((Look at it below))

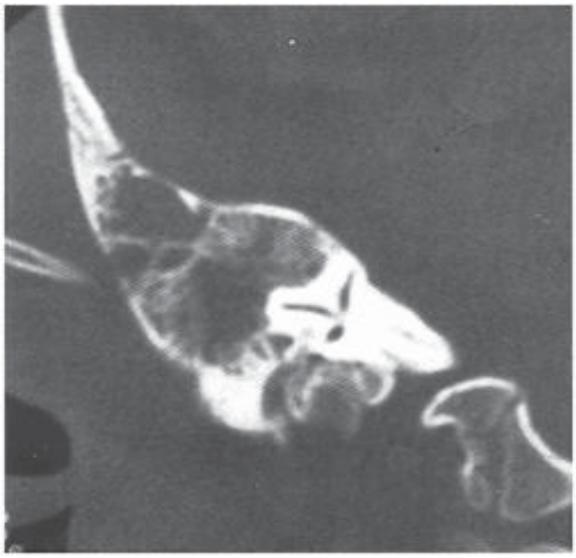
***Note:** To differentiate between them in Lymphadenitis there is no symptoms from ear.

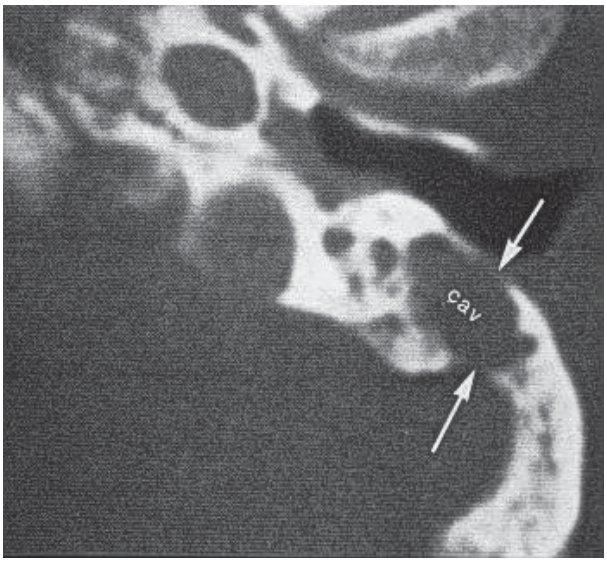


Bezold's abscess

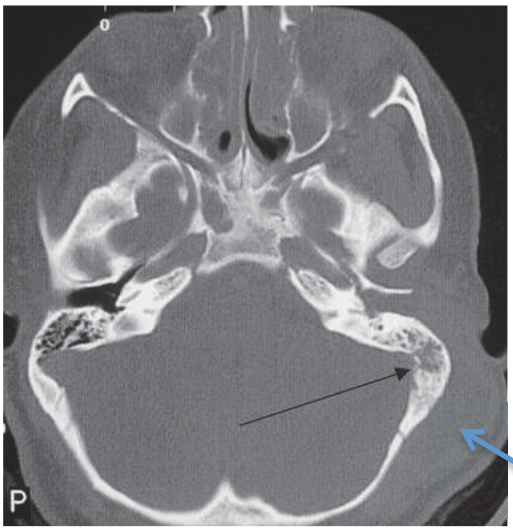
Investigations:

CT scan





CT , there is no air in the mastoid , the opacity mean that there is a fluid in the mastoid antrum .



Opaque defect in the bone that may lead to intracranial Complication.

Subperiosteal Abscess

TREATMENT OF ACUTE MASTOIDITIS: ((SAQ))

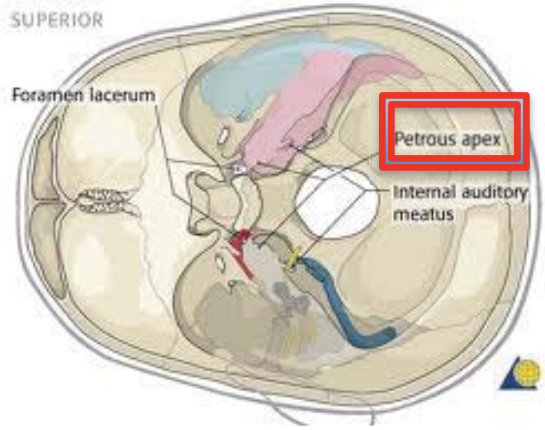
1. Admit the patient.
2. **IV antibiotics** (Cephalosporin 2G ± Metronidazole or clindamycin) to cover the aerobes & anaerobes
3. Cortical **mastoidectomy** if:
   - Medical treatment fails.
   - Signs of abscess formation.

- An operation in which the mastoid antrum and air cells are converted into one cavity without disturbing the middle or external ears *(it wont affect the hearing as a complication)* .
- It may be combined with myringotomy (TM opening). Which is indicated in:
  - Middle effusion
  - Mastoiditis if TM is intact
  - Recurrent attack of OM

- Observe for other complication (new symptoms)

Drain it until completely dry, this operation wont affects the hearing.

PETROSITIS (PETROUS APICITIS):



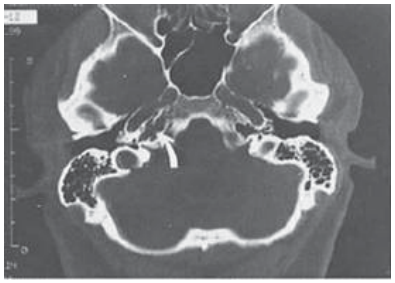
Dr.Alsnosi didn't talk about it ☺

An extension of infection from the middle ear into a pneumatized petrous apex.

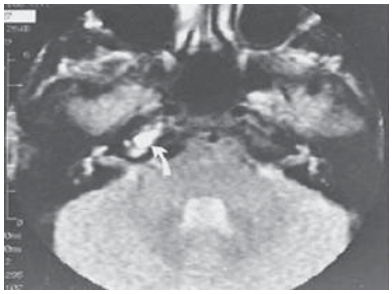
- Gradenigo's syndrome
  - Otitis media (otorrhea)
  - Retro-orbital pain
  - Squint (VI cranial nerve palsy) abducent.

* ***Note:*** Its not nessccary to have all of those 3 symptoms together.

Diagnosis:



CT



MRI T2

Show Abscess near to the cerebellum
Diagnosis of petrous apicitis requires both CT scan and MRI.

Treatment:

1. Admit the patient.
2. Antibiotics and myringotomy (If TM intact) and drain the abscesses by mastectomy
3. Surgical drainage if medical treatment fails

FACIAL PARALYSIS:

- The temporal & the mastoid part of the nerve are our concern in OM . **Dr alsnosis said the tympanic segment the commonest dehiscent area and dr Sami said the labrynith segment ☹ anyway u gonna to take it in sprerate lec in details (facial nerve)**
- 30% has dehiscent facial nerve ((not covered with bone)) so in middle ear infection affect the facial nerve . IN chronic OM the chloestotoma eat the bone that cover the nerve.
- Fallopian Canal: is the facial nerve canal

	AOM	CSOM
The pathology	Mostly due to pressure on a dehiscent nerve by inflammatory products	Usually is due to pressure by cholesteatoma or granulation tissue
The onset	Sudden in onset	Insidious in onset
The clinical course	Usually is partial facial palsy	May be partial or complete (start as partial and continue to become complete)
Treatment	Treatment is by systemic antibiotics IV and myringotomy (To relive the pressure on the nerve) *Prognosis : is Good usually the symptoms disappear after three days of Tx.	Treatment is by immediate surgical exploration by masodectomy and "proceed" (look for any cause for the obstruction e.g. granulation tissue and remove it)

Dr. Alsnosi treatment : Acute OM → cortical mastectomy +ventilation tube
Chronic OM→ mastectomy + facial nerve paralysis Tx

Both Drs shared the same Tx in chronic but not in the acute cortical mastectomy wasn't mentioned in dr.alisa lec.

Labyrinthine fistula and labyrinthitis (medial extension):

- **Labyrinthine fistula**
- Labyrinthitis has three types: **These types Dr.alsnosi did not talk about ☺**

➢ Circumscribed labyrinthitis
➢ Acute diffuse serous labyrinthitis
➢ Acute diffuse suppurative labyrinthitis

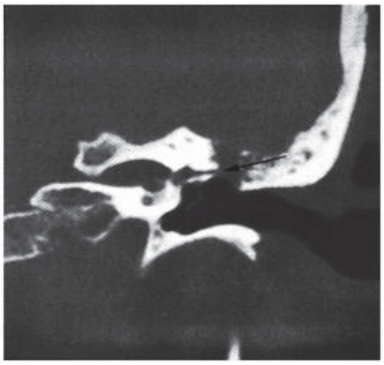
- Chronic labyrinthitis

LABYRINTHINE FISTULA: (MCQ)

common occur as a complication after chronic OM not the acute.

Loss of the bony labyrinthine wall exposing the endosteum (part of periostum) .
Fistula is an opening btw 2 epithelium, common with unsafe type due to the presence cholesteatoma which help spreading the infection .

Note: the horizontal or the **lateral labyrinth** is The **Most Commonly** affected labyrinth



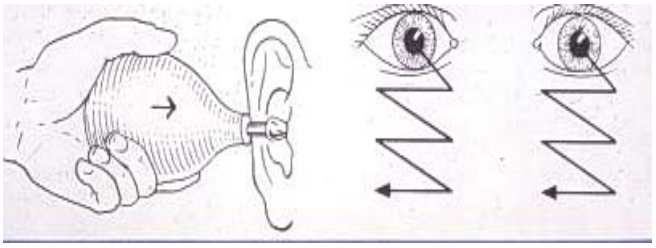
Diagnosis:

- No symptoms
- Vertigo if present indicate inner ear involvement
  SNHL (indicate cochlea involvement)
- **Fistula test** (+) **MCQ**

Applying Pressure to the ear by your finger will lead to vertigo or pump air inside the ear, which will induce **vertigo** and **Nystagmus** (Look below For more Explanation)

- CT scan diagnostic !

CT Fistula sometime missed because it is very small



Treatment:
Mastoid exploration is often required to eliminate the cause e.g. cholesteatoma.
Systematic antibiotics should be instituted before and after the surgery.
((Mastoidectomy look for the defect and close it))

INTRACRANIAL COMPLICATIONS:

A. Extradural abscess
B. Lateral sinus thrombophlebitis
C. Subdural empyema
D. Meningitis
E. Brain abscess
F. Otitic hydrocephalus

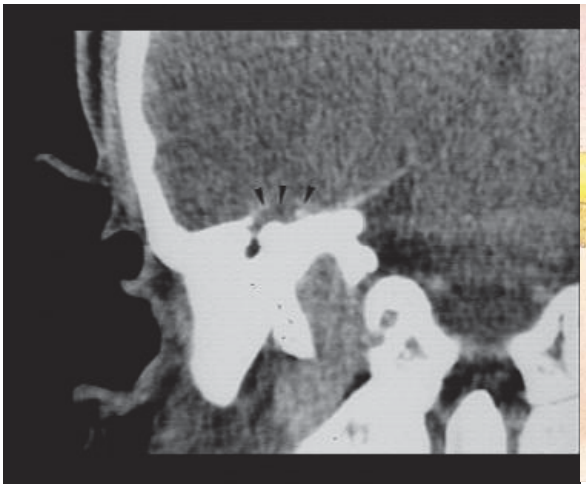
Note: Any pt with chronic disease think about intracranial extension

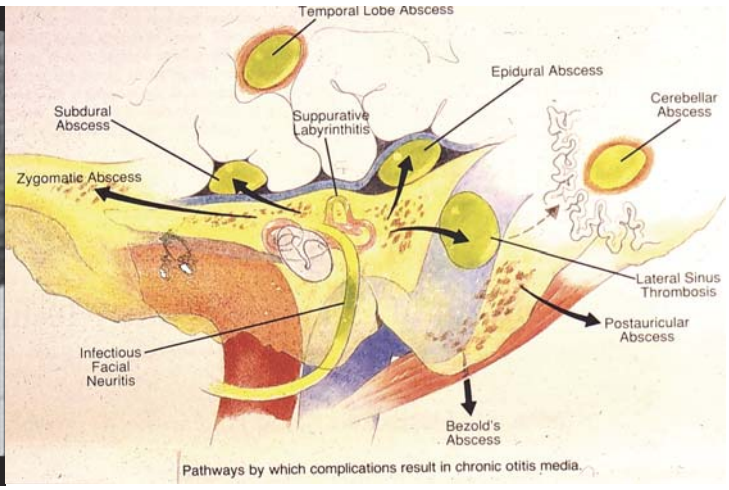
A. Extradural abscess: MCQ the commonest abscess

- Accumulation of pus between dura and bone (Defect in the bone that has been eaten up due to the infection , the bones involved are the tegmen tempani & tegmen antri)
- In the middle or posterior fossa (perisinus)

Outside the dura of lateral venous sinus its called (perisinus abscess)

- Causes headache (if there is increase in ICP ,persistence headache on the side of OM) but may be **silent.**
  Most of the time, extradural or perisinus abscesses are asymptomatic and Silent, and are discovered accidently during cortical or modified radical mastoidectomy. The Dr said that they discovered accidently by CT when looking for What is going on in middle & posterior fossa.
- Diagnosis is confirmed by CT or MRI
- Treatment is by **drainage** and **mesdectomy + IV Abx to prevent Recurrence.**





SUBDURAL ABSCESS (EMPYEMA): Rare

- Suppuration of the subdural space.
- May be localized, multiple or diffuse.
- Sever headache due to high ICP, fever, irritative and paralytic focal neurological symptoms
- CT and MRI
- Treatment is by neurosurgical drainage (combination btw neurosurgery and ENT by mastoidectomy for drainage)

Lumbar puncture should not be done as it can cause herniation of the cerebellar tonsils. It is a neurological emergency. A series of burr holes or a craniotomy is done to drain subdural empyema. Intravenous antibiotics are administered to control infection. Once infection is under control, attention is paid to causative ear disease which may require mastoidectomy.

LATERAL SINUS THROMBOPHLEBITIS:

Its either directly from mastoid or by venous channels

The pathological process can be divided into the following stages:

a. **Perisinusitis:**
Abscess forms in relation to outer dural wall of the sinus

b. **Mural thrombus:**
Inflammation spreads to inner wall of the venous sinus with deposition of fibrin, platelets, and blood cells leading to thrombus formation within the lumen of sinus.

c. **Occluding thrombus:**
Mural thrombus enlarges to occlude the sinus lumen completely.

d. **Suppuration:**
Organisms may invade the thrombus causing intrasinus abscess

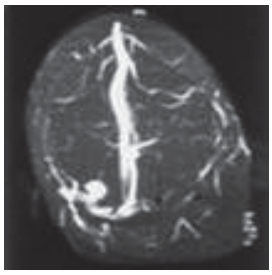
e. **Embolization:**
Though central part of thrombus breaks down due to intrasinus abscess, thrombotic process continues both proximally and distally. Proximally, it may spread to confluence of sinuses and to superior sagittal sinus or cavernous sinus, and distally, into mastoid emissary vein, to jugular bulb or jugular vein.

Diagnosis:

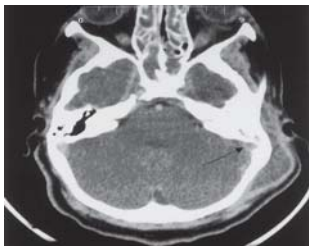
- Fever, **rigor**, and sweating
- Headache and neck pain
- Positive **Greissinger's sign, which is edema and** tenderness over the area of the mastoid emissary vein.
- **Tenderness** and edema in the neck (at the intrnal jugular vien area) **When the clot extends to the jugular vein, the vein will be felt in the neck as** a **tender cord** and there will be a manifestation of increased IC pressure (headache, vomiting, and papilledema).
- Propagation and embolic manifestations
- **Blood culture is positive during the febrile phase,** CSF manometry
- **CT with contrast** , **MRI** to show the complication

The problem with the lateral sinus thrombosis that it may send a distal emboli not to the brain only but to the lungs, abdomen and anywhere else. Its go to the heart as a complication.

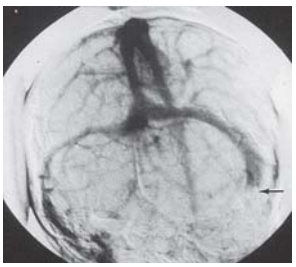
MRI Angiogram



CT Abcsess



Subtraction Angiogram



Imaging Study: Reading

Contrast-enhanced CT scan can show sinus thrombosis by typical delta sign. It is a triangular area with rim enhancement, and central low density area is seen in posterior cranial fossa on axial cuts. MR imaging better delineates thrombus. **"Delta sign"** may also be seen on contrast-enhanced MRI. MR venography is useful to assess progression or resolution of thrombus.

TREATMENT:

- Admit
- IV antibiotics
- Surgery should follow within 48 hours unless there is dramatic clinical and radiological improvement (expose the sinus and clean it)

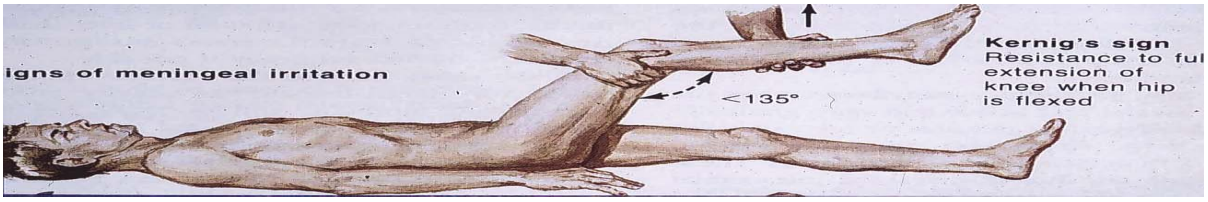
*IN Cavernous sinus syndrome: the abducent nerve the commonest affected .

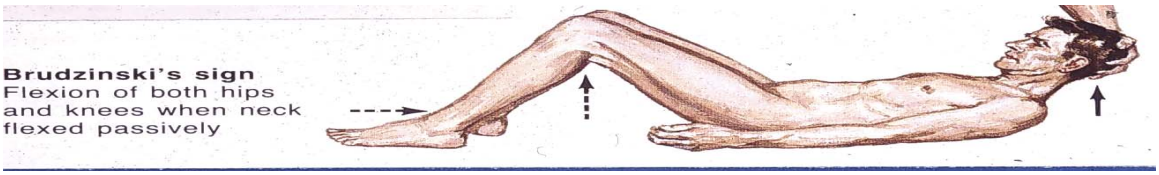
SURGICAL TREATMENT OF SINUS THROMBOPHLEBITIS: the doctor said it **is not imp** just if you want to read about it + Dr ALSNOSI did not mention ☺:

- Exposure of healthy dura proximal and distal
- Verify the sinus content
  - Blood clot: leave alone
  - Pus:incise to drain
- Ligate only if there is repeated embolisms or uncontrolled extension

OTOGENIC MENINGITIS:

- Infection of the subarachnoid space
- **The most common intracranial complication (middle ear infection)**
- Fever, headache, neck stiffness, phonophobia, restlessness etc
- Kernig's & Brudziniski signs
- Lumber puncture to confirm the Dx and culture it





Treatment: refral to neurosurgery the ENT role in this if there is mastoid pus accumulation they drain it

OTOGENIC BRAIN ABSCESS: the brain abscess the most lethal

- 25% of children's and 50% of adult's brain abscesses are otogenic.
- Mostly in temporal lobe or cerebellum (2:1) More in the temporal and the manifestations are according to the site.

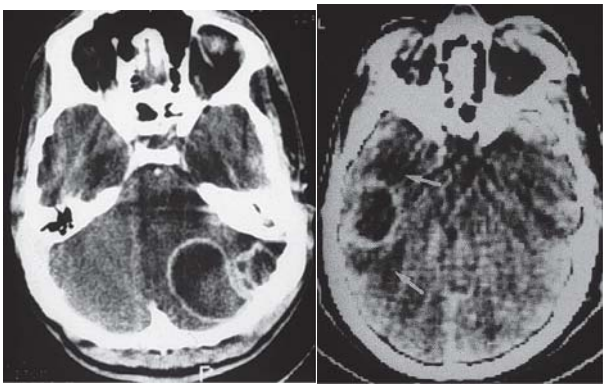
Clinical manifestations

- General manifestations:fever, lethargy, headache sever generalized worse in the morning .
- Manifestation of raised IC pressure (headach , N&V) the latter usually projectile seen more often in cerebellar lesions.
- Focal manifestations
  - Temporal: Aphasia, hemianopia, paralysis
  - Cerebellar: ataxia, vertigo, nystagmus, muscle incoordination

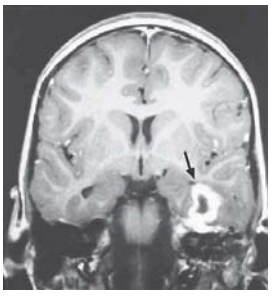
Diagnosis

- CT
- **MRI (tissue extension)**
- LP (lumbar puncture)
- Burr hole needling

The manifestation gives us a hint about the site .



CT: Abscess with well formed wall, they should be referred to the neurosurgery (temporal lobe , cerebellum)



MRI

Treatment:

- Repeated aspiration
- Excision (the neurosurgeon will drain it)

OTITIC HYDROCEPHALUS dr.alsnosi didn't mention ☺

- Very rare
- An idiopathic benign intracranial hypertension associated with ear disease. **It most often follows lateral sinus (sigmoid) thrombophlebitis**
- Clinically: Manifestations of increased IC pressure
- Treatment:steroids, diuretics, hyperosmolar dehydrating agents, repeated LP

They are referred to neurosurgery

GENERAL PRINCIPLES OF TREATMENT OF THE COMPLICATIONS:

- Parental antibiotics IV
- Surgery for the complication if applicable
- Treatment of the ear lesion
  - Myringotomy in AOM
  - Mastoidectomy in CSOM

AT The End

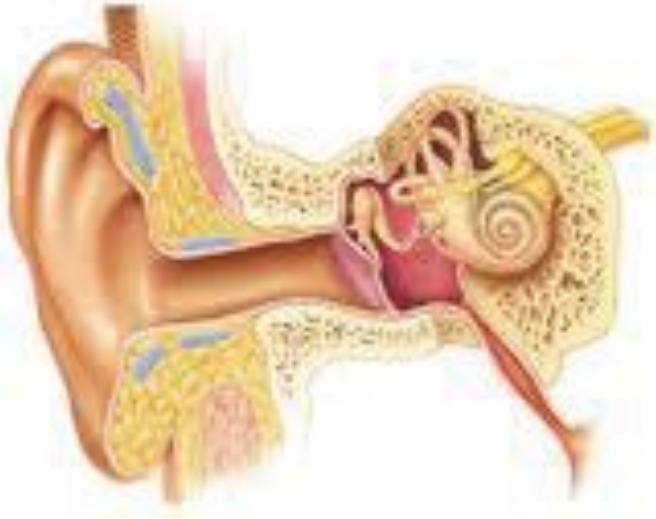
This lec was given by Dr. Alaisa for F2 and by Dr. Alsanosi for F1
Regarding the differences both of our doctors emphasized on the IMP points equally.
Whoever, both drs notes have been covered

وإن أصبت فمن الله وإن أخطأت فمن نفسي والشيطان

GOOD LUCK
☺

430 Teams

Diseases of the Ear, Nose and Throat



1st Lecture:

Audiological Evaluation

Done by: Anfal Alshaya

The slides were provided by doctor (Name)
Important Notes in red
Copied slides in black
Your notes in green/ blue
Titles and subtitles in this color
Highlight possible MCQs mentioned or pointed by the doctor

Physically to hear we need a sound.
Sound is a physical stimulus that evokes the sensation of hearing. And it is a pressure changes around the …. It transmitted through any medium (solid or air) to reach a sensor and stimulate it.
So to hear we need a sound source, a vibrating source like vocal cord as such, which will produce a wave of condensations and refractions transmitted through a medium that is the air in this case to reach the sensor of the ear which will be activated by this sound.

The sensor/ auditory system made of two parts mechanical and electrical.
The mechanical part is the external ear and the middle ear: the sound wave will pass through the external ear to the middle ear when the stapes moved over the oval window the energy will be conducted to the fluid of the inner ear. The movement of the fluid will take the basilar membrane up and down and then this will move the cilia and hair cells. The hair cells will go to biochemical activity result in sharing action between cilia of hair cell and tectorial membrane above it. Then it will be conducted into electric signal as firing in auditory nerve which is the sensory part.

And according to this hearing loss could be conductive when the problem is either in the external or middle ear, sensory if the problem in the inner ear and the ascending neural connections , or mixed.

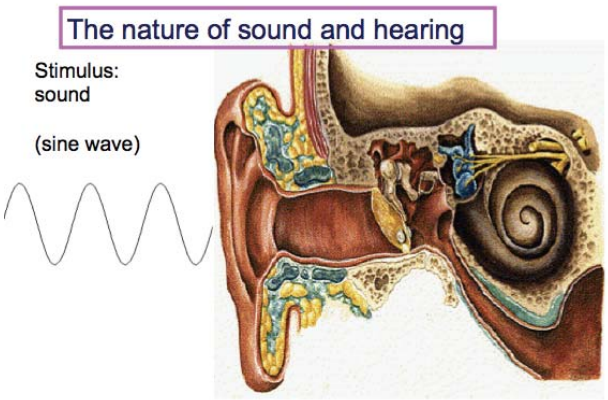
Audiology:

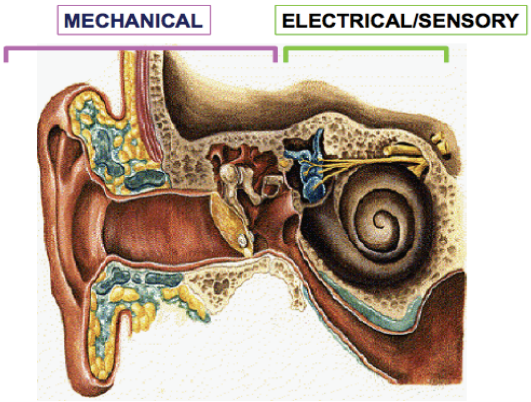
The study of sound and hearing

Sound=physical stimulus that evoke sensation of hearing.
Audiometry=the measurement of hearing sensitivity.

Sound:

Sound is a form of vibration
Vibration is the to-and-fro motion of an object (guitar string, vocal folds, diaphragm on an earphone or loudspeaker, tuning fork)

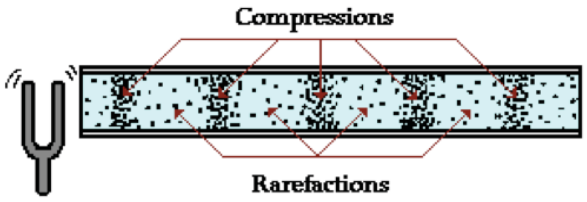




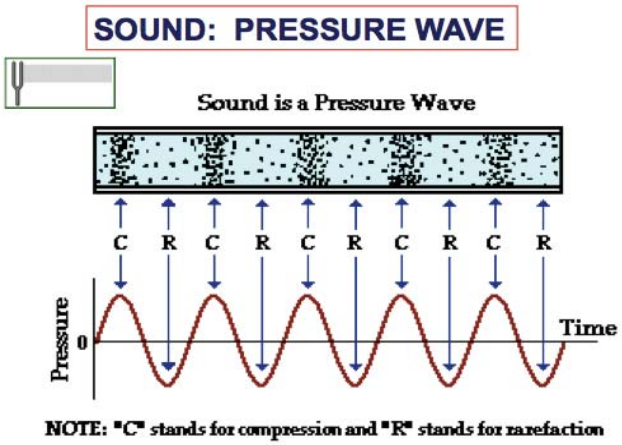
For sound to occur, must have a:

SOURCE: Something has to be disturbed.
FORCE: Something has to disturb it.
MEDIUM (e.g. air): Something has to carry the disturbances.

When air molecules are displaced, pressure waves occur



http://www.glenbrook.k12.il.us/gbssci/phys/Class/sound/u11l1c.html



NOTE: "C" stands for compression and "R" stands for rarefaction

The particles after the sound wave started will go into motion and in order to stay longer it goes into rarefaction and compressions. This wave drown in a graph called sine wave. Where it goes from resting point (0) to maximum condensation (C), resting again, and then to rarefaction (R).

Characteristics of the waveform (amplitude x time):

CYCLE: One complete period of compression and rarefaction of a sound wave
PERIOD: The amount of time that it takes to complete one vibratory cycle.

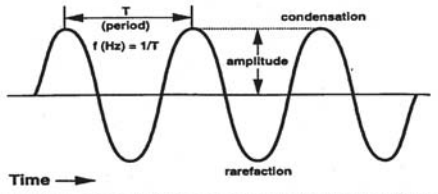
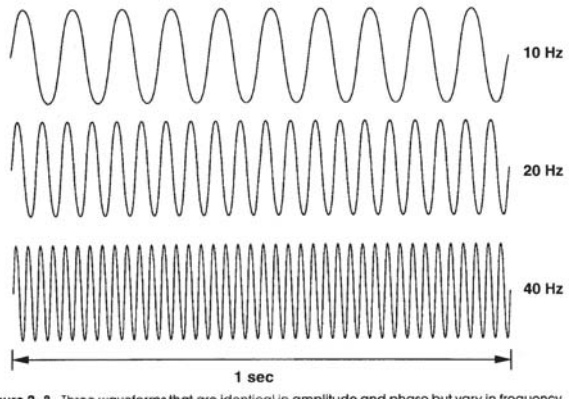


Figure 2–5. A sinusoidal waveform, describing the various properties of sound, including amplitude and frequency (f).

B. A. Stach (1998). Clinical Audiology: An Introduction. San Diego: Singular Publishing



FREQUENCY: The number of cycles that occur in one second.

Hertz (Hz): Unit of measurement of frequency
100 cycles per second = 100 Hz

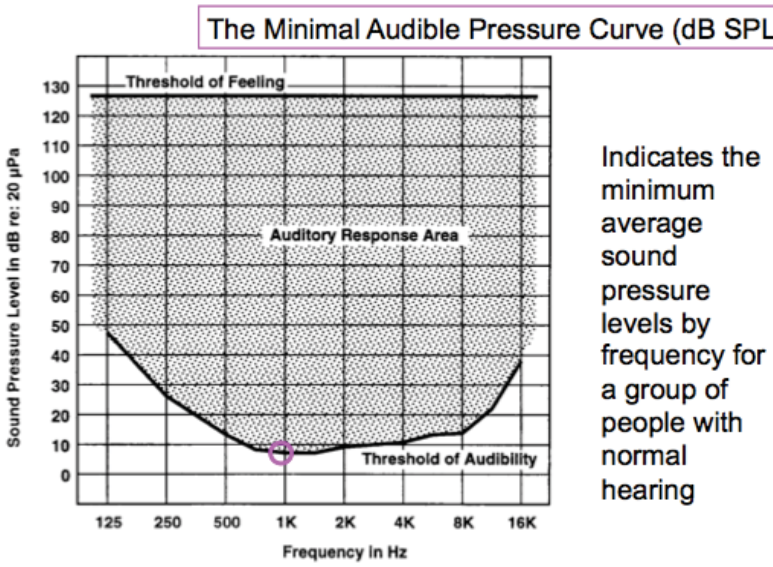
Pitch: Psychological percept of frequency.
e.g., low frequency sounds = low pitch

Frequency Range of Hearing Sensitivity:
Humans: 20 Hz to 20 kHz.

- Below 20 Hz, we feel a vibration rather than hear a sound.
- Most people have very diminished sensitivity for frequencies > 8000-10, 000 Hz.

Bats (auditory specialists) : 2 kHz-100 kHz.

Practically human ear hear from 250 kHz to 8000 kHz .



Indicates the minimum average sound pressure levels by frequency for a group of people with normal hearing

Frequency axes:
In order to hear sounds out of our range from 250 to 8000 we need sounds of high pressure/intensity.

Intensity axes:
Hearing threshold: the lowest intensity, which can be detected by ...in 50% of trails. Just awareness of sound (no discrimination).

Comfortable level: discrimination 100%

Uncomfortable level: above the comfortable level by 40 dB

Hazardous: above 100 dB.

If we increase the intensity we will reach the sensation area and there will be tactile sensation.

Amplitude/ Intensity: (loudness)
The quantity or magnitude of sound.
Decibel (dB): Unit of amplitude used most frequently in clinical audiology.

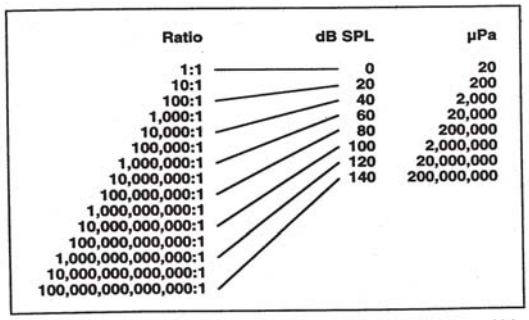
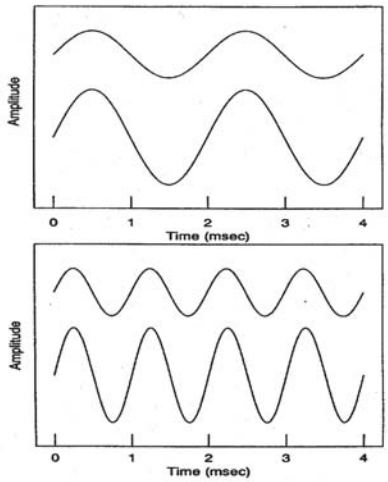


Figure 2–7. The relationship of the ratio of sound magnitude to the range of sound intensity expressed in sound pressure level. Sound ranges from barely audible at 20 µPa to painful at 200,000,000 µPa.

B. A. Stach (1998). Clinical Audiology: An Introduction. San Diego: Singular Publishing Group, Inc.



S. A. Gelfand (1997). Essentials of Audiology. New York: Thieme

Hearing loss prevention:

Noise controls, hearing protectors

- Primary prevention ➔ reduction or elimination of HL

Screening neonates, school age, elderly, industrial

- Secondary prevention ➔ early identification to reduce negative effect of HL

Audiology services (hearing aids, rehab)

- Tertiary prevention ➔ services to deal with adverse effects of HL

Hearing assessment

Types of Tests:

BEHAVIOURAL / subjective: patient listens to different sound at different frequency and at each frequency we raise and decrease the sound till it reaches the threshold.

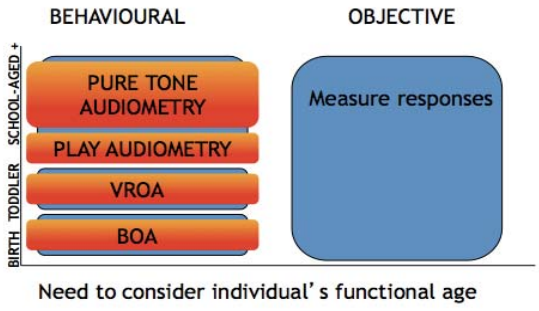
- Reliable & consistent response to sound
- Developmental age
- Not used in newborn screening
- Not critically ill

In children below age of school we can assess hearing either by play audiometry or behavioral observation audiometry.

OBJECTIVE: record the biochemical/firing activity from the inner ear (cochlea or nerve)

- No voluntary response
- Infants and young children
- Non compliant subjects
- People with developmental level that doesn't allow other testing.

Age based hearing assessment:



Overview

- Behavioral audiometry
- Tympanometry
- Acoustic reflex measurements
- ECochG
- Auditory Brainstem Response (ABR)
- Otoacoustic Emissions

Behavioural Observation Audiometry (BOA):
Observing changes in behaviour in response to sounds
Who?
Very young babies (under 6mths corrected) or with similar functional age.
Test sounds & materials:

- Calibrated (known frequency and intensity) noisemakers
- Audiologist records sound level (from sound level meter), sound type & observed response- observer determines whether response is present/absent

Infants 7 months-3 years:

Aim: to detect hearing impairment greater than 20-30 dB HL
Typically use behavioural techiques
Visual Reinforcement Orientation Audiometry (VROA) for 6-18 months
Play audiometry
May incorporate objective testing if non-compliant or very difficult to test

Visual Reinforcement Oreintation Audiometry (VROA):
Uses operant conditioned response and visual reinforcement

- Response typically head turn. Eye turn also possible
- Complex visual reinforcement usually lighted puppet theatre- colour movement and light are important

Play audiometry 3-9 years:
Before testing

- Subjective check of audiometer
- Check test environment, audibility of tones
- Avoid visual clues
- Instruct client, demonstrate procedure
- Position headphones
- Present orienting tone (40dBHL) and check client's response. Re-instruct if necessary

Screening with Play Audiometry

- use peg board, blocks etc.
- if very young get parents to train child at home
- headphones on desk present 100dB tone
- train child without headphones- Stimulus -Response
- introduce headphones
- present 40dB HL tone with headphones on. Repeat
- decrease tone to 20dB HL for screen

Pure Tone Audiometry:

- Most common test
- Threshold of audibility
- Activation of auditory system
- Energy formatted into neural code
- Air conduction assesses entire system
- Bone conduction assesses cochlea onwards

Pure Tones: important

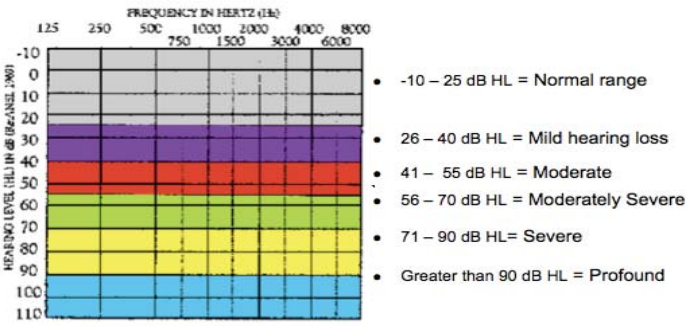
- Auditory acuity

- Spectrally specific
- High frequency tones stimulate basal turn of the cochlea
- Low frequency tones stimulate apical turn of the cochlea

Pure tone audiometry:

Assessment of thresholds

- Octave frequencies tested
- Bone conduction thresholds
- Mastoid or forehead used
- Mastoid preferred because less intensity required
- Occlusion effect
- Ascending series of tone presentations



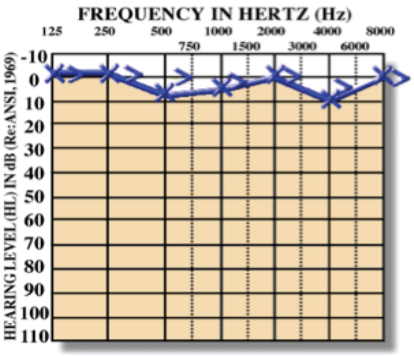
Air: medium between the sound source and ear is air we measure it using headphones or speakers
Bone: the medium is bone and measured by placing the vibrator over the skull.

Types of hearing loss:
Conductive: air hearing loss: the problem either in the external or middle ear
Sensorineural: bone hearing loss: there is problem in the inner ear
Mixed: both conductive and sensorineural hearing loss

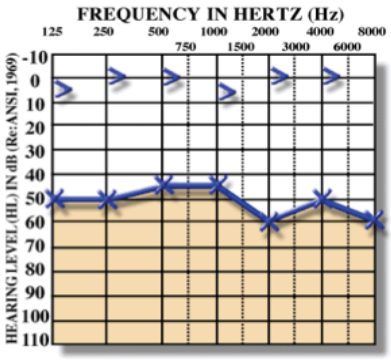
Ranges of Hearing Loss:

If the air line goes down that means it is a conductive hearing loss and it could be mild moderate, or sever according to the graph above. If both lines go down it is a sensorineural hearing loss, In mixed type both lines will go down however there is a gap between them and the bone is better.

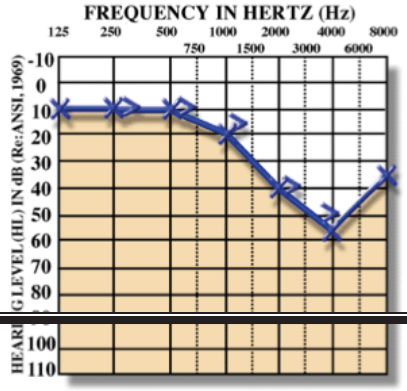
Normal Hearing



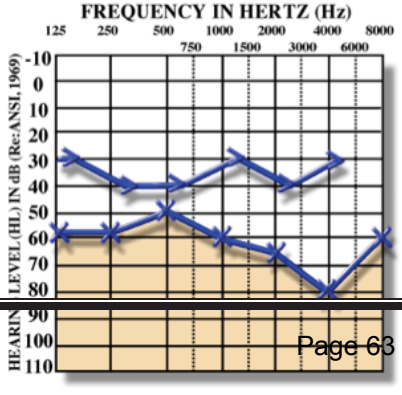
Conductive Hearing Loss



Sensorineural Hearing Loss



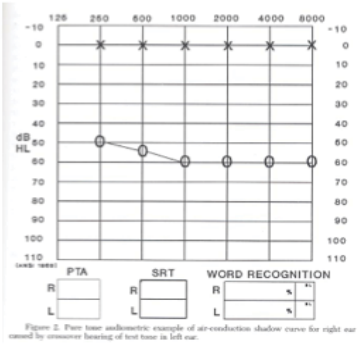
Mixed Hearing Loss



Speech Audiometry:

- Speech Reception Threshold using spondaic words
- Standardized word lists
- Familiarization with spondees
- Ascending series of presentation
- Excellent speech discrimination in conductive hearing loss patients
- Poor speech discrimination in cochlear hearing loss patients
- Poorest speech discrimination in retrocochlear hearing loss patients

Shadow Curve



Clinical Masking:

- Nontest ear can influence thresholds of test ear
- Shadow curve apparent without masking / false hearing threshold taking from the nontest ear
- Interaural attenuation varies from 40 to 80 dB with air conduction
- Interaural attenuation is about 0 dB with bone conduction
- Compare bone conduction threshold of nontest ear with air conduction threshold of test ear to determine whether masking is necessary
-

Plateau method:

- Mask nontest ear with progressively greater amounts of sound until threshold does not rise.
- Masking Dilemma
-

Objective Audiological Tests:

In short: for the external ear: otoscope
For the middle ear: immittance/tympanometry
For the inner ear: cochlea: otoacoustic emissions
Nerve: EBR (audiology brainstem response)

Electrophysiological Tests:

- Immittance
- Evoked Potential
- Otoacoustic Emissions

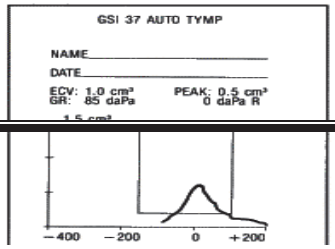
Immittance:

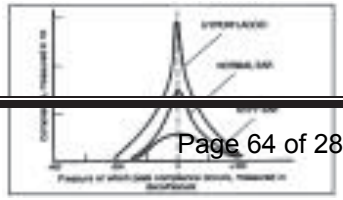
- Ear Canal Volume
- Tympanometry
- Static Compliance
- Acoustic Reflex, Decay, & Latency

Ear Canal Volume:

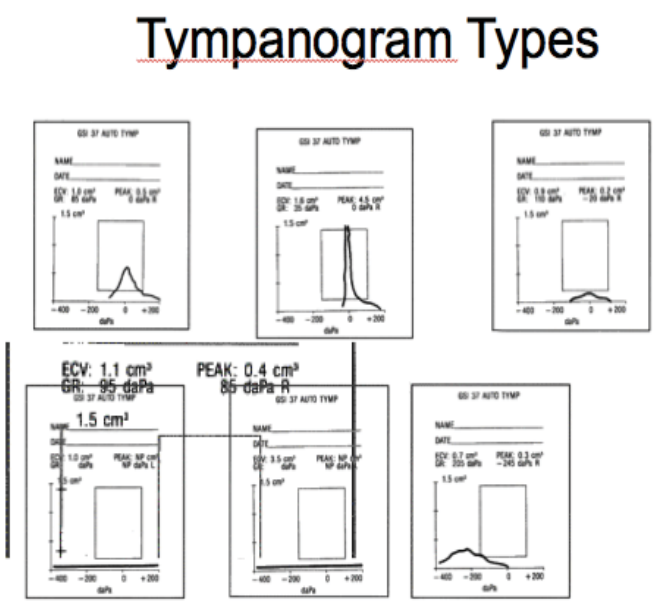
- Measure at +200 mmH20
- Provides measure of volume of external ear canal
- Volumes based on age
- Volumes greater than 2.5 suggest:
  - Perforation or
  - Patent V. tube

Tympanometry:



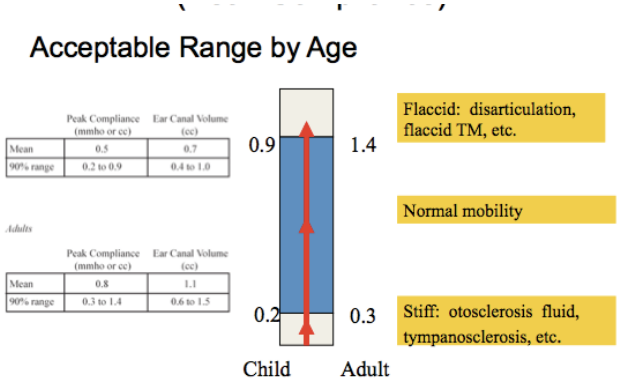


- Objective measure of the function of the TM and middle ear
- 5 or 6 basic shapes
- Tympanogram Types:



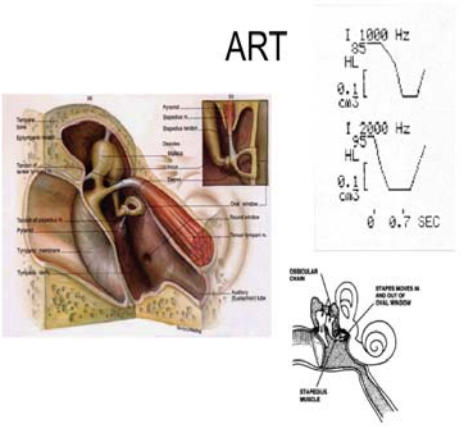
Type A/ normal curve:The peak more or less around zero (atmospheric pressure), and the amplitude from 0.3 to 1.7 and indicates normal middle ear function.
Type AD: very long curve indicates disarticulation
Type AS: very short curve indicates otosclerosis.
Type B: flat curve in case of middle ear effusion. If it's high that means there is perforation.
Type C: curve shifted to the negative and indicates eustachian tube dysfunction.

Static Compliance (Peak Compliance):



For the inner ear:
Otoacoustic emission: for the biochemical function of the outer hair cells of the cochlea
If I can record it that's mean we have normal cochlea
ABR: we test the nerve at the brainstem. We place 3 surface electrodes (forehead, right ear, and left ear) over the skull and deliver a sound through headphone to record the electric activity/firing of nerve. This is an evoked process meaning that we have to drive the auditory system into action by delivering a sound in order to be able to record the nerve activity. Normally there will be five waves. The most important one is wave 5 at 5 m. second which is time of onset of stimulus.

ART:



Acoustic Reflex Threshold:

- Stapedial muscle contraction
- Temporary increase in middle impedance
- Bilateral Stimulation
- Adaptation
- Neural network in lower brainstem

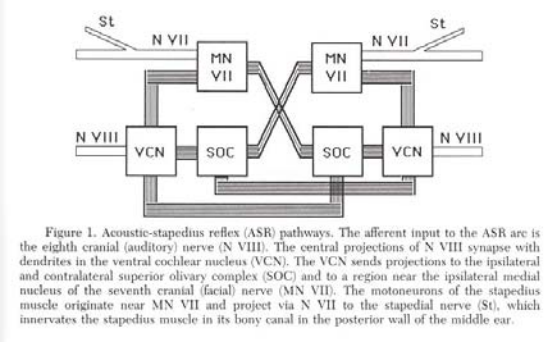
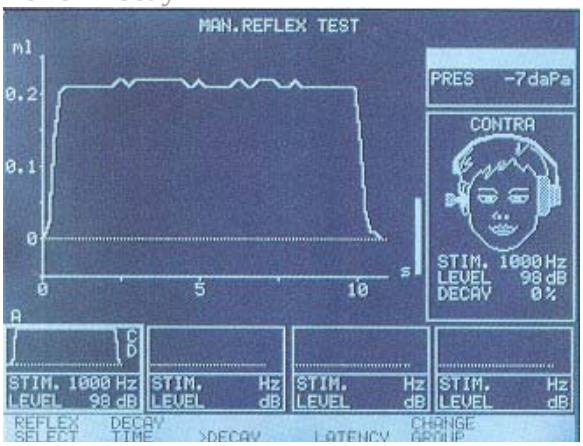


Figure 1. Acoustic-stapedius reflex (ASR) pathways. The afferent input to the ASR arc is the eighth cranial (auditory) nerve (N VIII). The central projections of N VIII synapse with dendrites in the ventral cochlear nucleus (VCN). The VCN sends projections to the ipsilateral and contralateral superior olivary complex (SOC) and to a region near the ipsilateral medial nucleus of the seventh cranial (facial) nerve (MN VII). The motoneurons of the stapedius muscle originate near MN VII and project via N VII to the stapedial nerve (St), which innervates the stapedius muscle in its bony canal in the posterior wall of the middle ear.

Clinical application of ASR

- Middle Ear Disease
- Otosclerosis
- Cochlear hearing loss and loudness recruitment
- Retrocochlear lesions may abolish the ASR
- Brainstem lesions may abolish the contralateral reflexes
- Determination of site of a seventh nerve lesion
- Acoustic Reflex Decay

Reflex Decay:



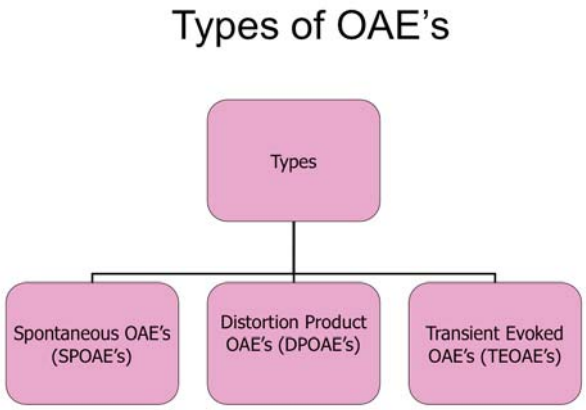
otoacoustic emissions:

Background:
The presence of cochlear emissions was hypothesized in the 1940's on the basis of mathematical models of cochlear nonlinearity. However, OAEs could not be measured until the late 1970s, when technology created the extremely sensitive low-noise microphones needed to record these responses.
David Kemp first discovered Otoacoustic emissions in 1978.

Otoacoustic Emissions:
- Otoacoustic emissions are sounds that are produced by healthy ears in response to acoustic stimulation.
- OAE's arise because our ears have evolved a special mechanism to give us extra hearing sensitivity and frequency responsiveness. The mechanism is known as the cochlear amplifier and it depends on a specialized type of cell called "outer hair cells."
- It's the job of the cochlea to receive the sound energy collected by the outer and middle ear and to prepare it for neural transmission.

Purpose of OAE's:
- The primary purpose of otoacoustic emission (OAE) tests is to determine cochlear status, specifically hair cell function. This information can be used to
- (1) screen hearing
- (2) partially estimate hearing sensitivity within a limited range
- (3) differentiate between the sensory and neural components of sensorineural hearing loss
- (4) test for functional hearing loss.

Types of OAE's:



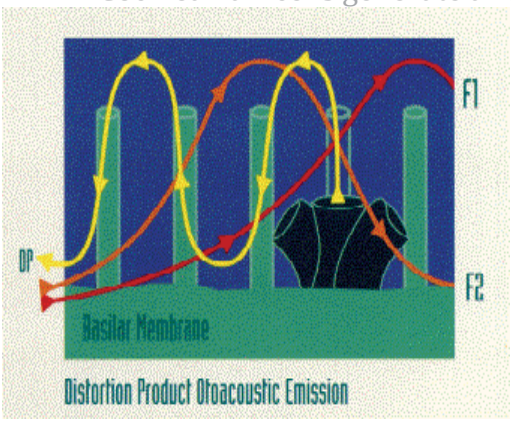
Spontaneous OAE's:
- Occurs in the absence of any intentional stimulation of the ear.
- Prevalence is in about 40-60% of normal hearing people.
- When you record SOAE's, you average the number of samples of sounds in the ear and perform a spectral analysis.
- The presence of SOAE's is usually considered to be a sign of cochlear health, but the absence of SOAE's is not necessarily a sign of abnormality.

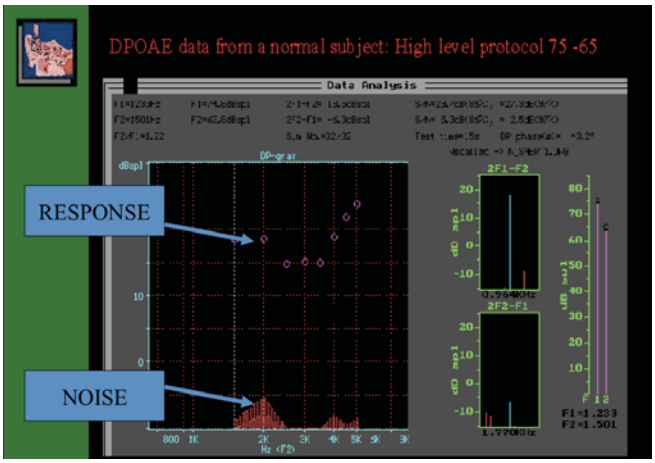
Distortion Product OAE's:
- Result from the interaction of two simultaneously presented pure tones.
- Stimuli consist of 2 pure tones at 2 frequencies (ie, f1, f2 [f2>f1]) and 2 intensity levels (ie, L1, L2). The relationship between L1-L2 and f1-f2 dictates the frequency response.

- DPOAEs allow for a greater frequency specificity and can be used to record at higher frequencies than TOAE's. Therefore, DPOAE's may be useful for early detection of cochlear damage as they are for ototoxicity and noise-induced damage.
- DPOAEs often can be recorded in individuals with mild-to-moderate hearing losses for whom TOAE's are absent.
- DPOAE's do not occur in the frequency regions with more than 50-55dB Hearing loss.
- DPOAE's can be elicited from ears that have a greater hearing loss than TEOAE's.

DPOAEs:

- 2 tone stimuli (F1 and F2)
- Cochlea hair cells generate a resonance

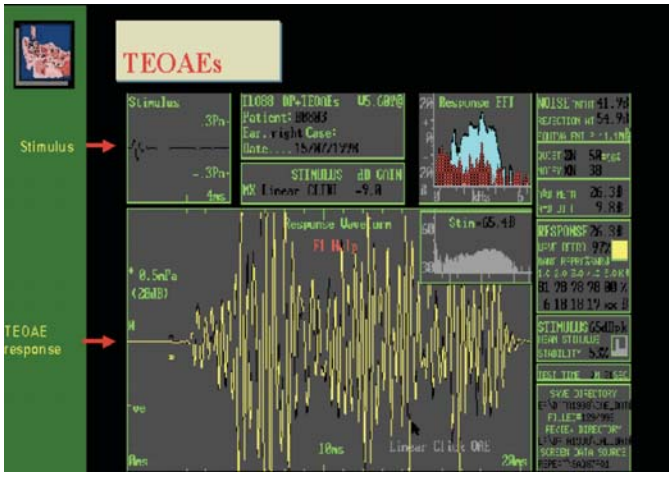


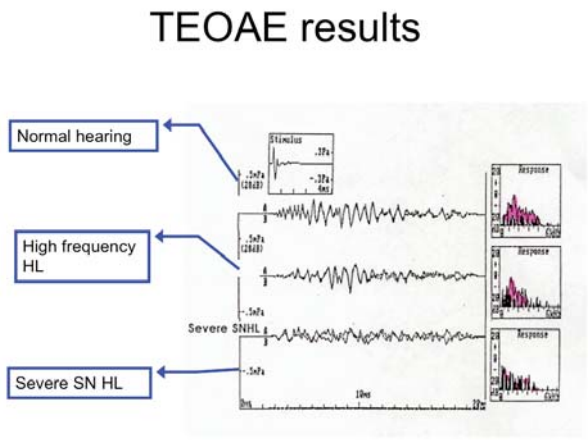


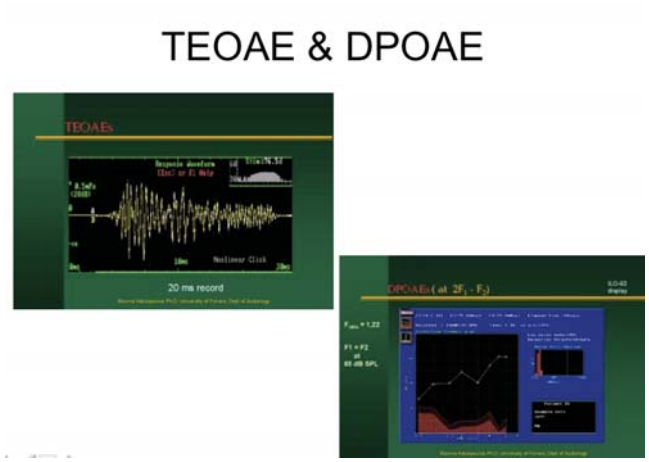
Transient Evoked OAE:

- TEOAE's are frequency responses that follow a brief acoustic stimulus, such as a click or tone burst.
- The evoked response from this type of stimulus covers the frequency range up to around 4 kHz.
- In normal adult ears, the click-elicited TEOAE typically falls off for frequencies more than 2 kHz, and is rarely present over 4 kHz, because of both technical limitations in the ear-speaker at higher frequencies and the physical features of adult ear canals so that is why DPOAE's would be more efficacious.
- For newborns and older infants, the TEOAE is much more robust by about 10 dB and typically can be measured out to about 6 kHz indicating that smaller ear canals influence the acoustic characteristics of standard click stimuli much differently than do adult ears.
- TEOAE's do not occur in people with a hearing loss greater than 30dB.

TEOAE results

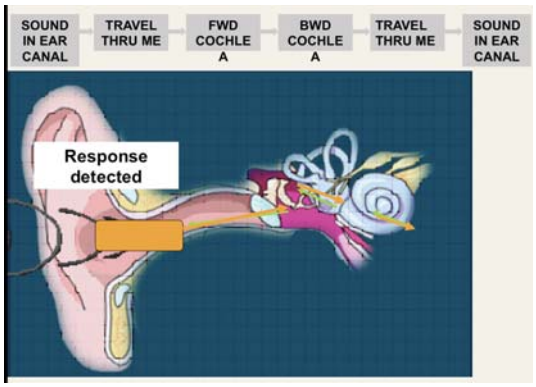






Recording OAE's:

- OAEs are measured by presenting a series of very brief acoustic stimuli, clicks, to the ear through a probe that is inserted in the outer third of the ear canal. The probe contains a loudspeaker that generates clicks and a microphone that measures the resulting OAE's that are produced in the cochlea and are then reflected back through the middle ear into the outer ear canal.
- The resulting sound that is picked up by the microphone is digitized and processed by specially designed hardware and software. The very low-level OAEs are separated by the software from both the background noise and from the contamination of the evoking clicks.



OAEs:

- Otoacoustic emissions
- "Echo"-like response of outer hair cells of the cochlea
- Can only indicate functioning outer hair cells and good middle ear function.

Types of OAEs:

Spontaneous:

- 20-60% of population, related to age
- Not clinically useful
- Not related to tinnitus

Evoked:

- Present in normal ears
- Not present in ears with SNHL greater than 25-30 dB
- Absent in presence of conductive hearing loss. WHY?

Evoked OAEs:

Types:

- Click (transient) evoked OAE- TEOAE : Absent for sensori neural loss greater than 20-30dB HL
- Distortion product OAE (DPOAE) : Absent in sensori neural losses greater than 45-55 dB HL

Acquisition:

- Not affected by sleep but needs test subject to be still and compliant
- Very quick

clinical applications:

- Quick screening tool
- Good indicator of cochlear reserve- correlated with hearing
- Monitoring
- TEOAE present with hearing loss up to 30dBHL
- DPOAE present with hearing loss up to 50dB HL
- Monitoring of drug ototoxicity (can affect OAE before HL present)
- Sensory vs. neural HL

clinical limitations:

- Problems because of middle ear disease
- Not sensitive for neonates within 24 hours of birth
- Results affected by test conditions
  - Noise
  - Electrical interference
- Not a test of hearing- limited application

Electrocochleography:

History:

- Little confusion in the literature, apart from what letters of the original appear in the abbreviation
- Animal models first discovered in 1930s
- Clinical applications started in 1960s

Components:

- Cochlear microphonic: outer hair cell response
- Summating potential: cochlear activity
- Action potential: Firing of auditory nerve (same as ABR wave 1)
- All occur within the first 1.5-2 ms after an acoustic stimulus

stimulus & acquisition:

- Recording electrode must be as close to response as possible (transtympanic)
- Children: general anaesthetic
- Adults: may be done without anaesthetic
- resistant to effects of drugs and subject state of arousal
- Can be used in pre-implant assessment to test cochlear function

clinical applications:

- Diagnosis of Meniere's disease
- Diagnosis of cochlear hearing loss/auditory dysynchrony, sensory vs neural.
- Assessment of hearing status for difficult to test subjects

clinical limitations:

- Auditory information only provided to cochlea
- Very invasive
- Results can vary up to 20dB from actual hearing
- Limited frequency specificity
- Expensive

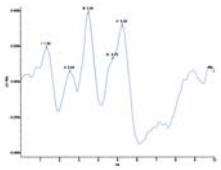
auditory brainstem response:

history:

- First complete description in 1970s
- Response found between 1-15ms after stimulation.
- Recording has 7 peaks, peak five being the most prominent.
  - The amplitudes, latencies and relationship of those peaks can be used to diagnose certain pathological conditions.

What is an ABR?

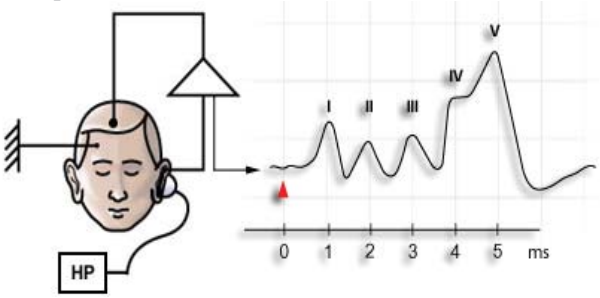
- The Auditory Brainstem Response is the representation of electrical activity generated by the eighth cranial nerve and brainstem in response to auditory stimulation

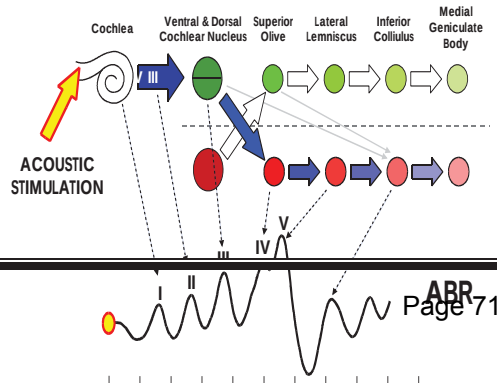


How is an ABR recorded?

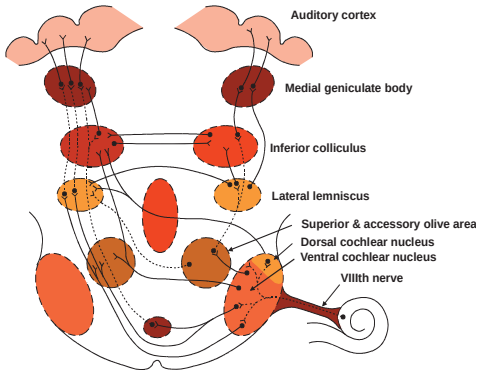
- Electrodes are placed on the scalp and coupled via leads to an amplifier and signal averager. EEG activity from the scalp is recorded while the ear(s) are stimulated via earphones with brief clicks or tones.
- A series of waveforms unique to the auditory neural structures is viewed after time-locking the EEG recording to each auditory stimulus and averaging several thousand recordings.

Components:

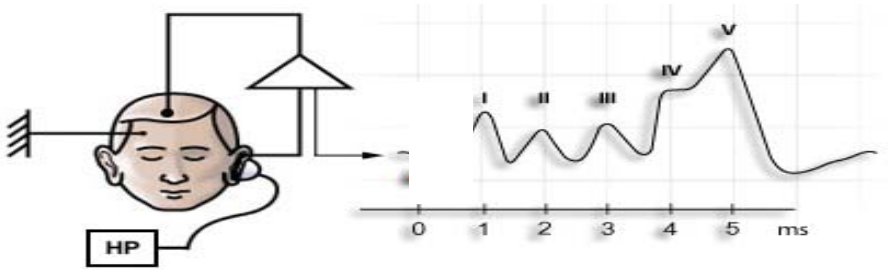




Generators of the ABR:

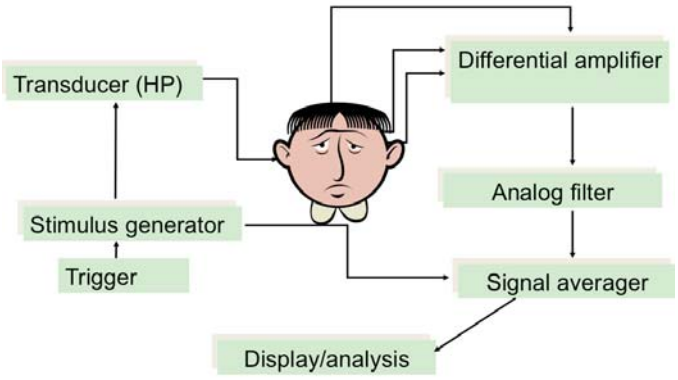


Anatomy:

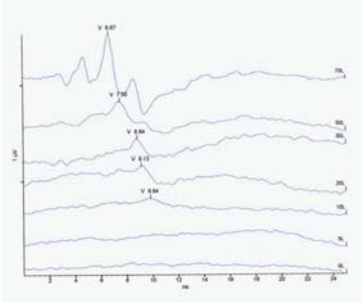


stimulus & acquisition:

- Short clicks or tone bursts used
- Rate of around 20/sec or faster
- Responses can be + or – 20dB on true thresholds, mixed in with EEG
- Electrodes on head (surface electrodes)
- Can be influenced by subject characteristics (age, gender, body temperature)
- Not affected by arousal state or most drugs



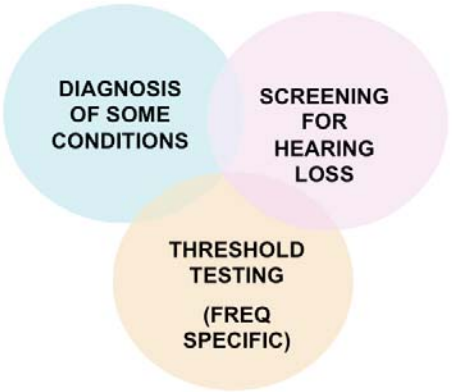
Example Normal Hearing:



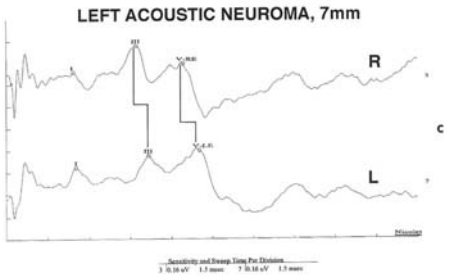
clinical applications:

- Basis of Newborn screening tests: non-invasive, high success rate
- Estimation of thresholds for difficult to test people
- Neurodiagnosis of VIIIth nerve/ brainstem problems
- Intraoperative monitoring
- Cochlear implant evoked responses
- Test-retest reliability

Why use ABR testing?

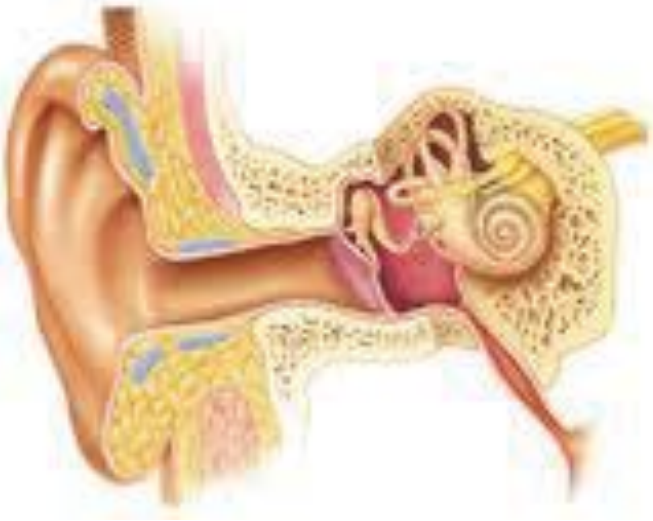


Retrocochlear lesion:



430 Teams

Diseases of the Ear, Nose and Throat



19th Lecture:

Vertigo

Done by: Khawlah Ateeq

Revised by: Hadeel Al-Madany

Sources: Dr. Hagr's lecture, 429 ENT Team Notes (Vertigo).
Important Notes in red
Copied slides in black
Your notes in green/ blue
Titles and subtitles in this color
Highlight possible MCQs mentioned or pointed by the doctor

2 [VERTIGO

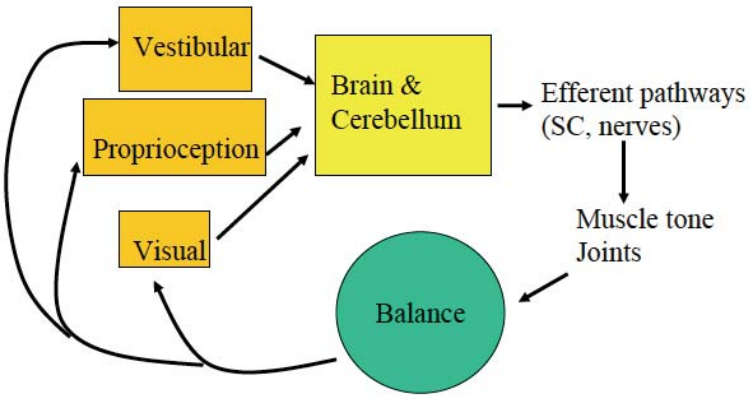
INRODUCTION:

What are the components of balance system?

1. Inner ear (5)
- **a.** 3 semicircular canals: one lateral one vertical one posterior (Why? Any movement can be detected through them) Rotational
- **b**. Otolith organs: control back and forth and side-to-side movement (Translational)

2. Cerebellum
3. Vision
4. Proprioception

Basic Mechanism of Detection of Rotation
• INERTIA: Would resists any movement and this resistance picked up by the nerve delivered to the brain. This always happen at the firing level. Like any muscle that has a tone, there is a firing level in both sides of vestibular organ that balance themselves; if tone is firing the other one is lowering. i.e. they are balancing each other. This is always in the acceleration phase. Therefore,balance problems can be detected through acceleration and deceleration as movements in the car (Ups & Downs like الهدا)
• Detects head acceleration – but encodes head velocity (i.e. integrator)



How does the balance system work?

1. Transformation of the forces associate with head acceleration and gravity into biological signals that the brain can use to develop subjective awareness of head position in space (orientation)
2. Production of **motor reflexes** that will maintain posture and ocular stability
3. Any pathology will cause: imbalance, posture and gait imbalance & visual distortion (oscillopsia).

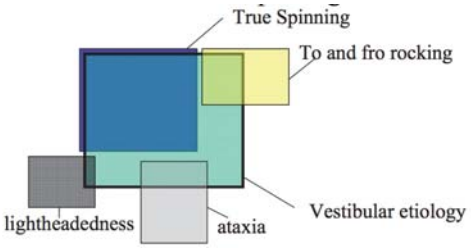
Why do we have a vestibulo ocular reflex?

1. Stabilizes retina in space – fast! On head movement
2. Posture Control (Do finger test)

What is dizziness? In general; ear causes vertigo NOT dizziness.

It is the illusion of movement of self or environment. It is a very vague term that could mean:
- True spinning (vertigo)
- Lightheadedness
- Unsteadiness
- Fainting/passing out

Due to cardiovascular, neurological, or inner ear disease



What is vertigo?

• It is the illusion of rotational, linear, or tilting movement of self or environment.
• Rule: "All true spinning is vestibular – not all vestibular is spinning"
*You have to think if it's central or peripheral; it is central in patients with neurological symptoms (headache, nystagmus, loss of consciousness, risk factors, atrial fibrillation…etc) → go to neurologist.
*Peripheral from the ear (more severe); nausea, vomiting, inability to walk, ataxic gate.

Prime clue #1: Is it vertigo (true spinning)? *(Usually spinning worse w/movement + ataxia + nystagmus + nausea & vomiting)*

• History: (most importantly to ask about first and last episode)
o Onset
o Character: establish that it is true spinning not just dizziness from other cause
o Duration

o Frequency
o Aggravating/relieving factors
o Associated auditory symptoms
o Hx of ear disease or ear surgery
o Hx of trauma
o Hx of migraine
o Hx of ototoxic drug intake

• Peripheral vs. central history:
o Neurologic symptoms e.g. new severe headache, loss of consciousness
o Type of nystagmus
o Risk factors (HTN, DM, Atrial fibrillation)
o No improvement with 48 hours

If Central → Neurologist
Symptoms usually are severe not like other disease regarding the nausea and vomiting, imbalance.

Symptoms	Peripheral	Central
Imbalance	Mild- Moderate	Severe
Nausea and Vomiting	Severe	Variable
Auditory Symptoms	Common	Rare
Neurological Symptoms	Rare	Common
Compensation	Rapid	Slow
Nystagmus	UNI directional (Horizontal or Rotatory)	Bi directional (Horizontal or Vertical)

Prime clue #2: What is the duration of the attack? Is it seconds to minutes, minutes to hours, days or constant?

Vertigo	*With Hearing Loss*	*Without Hearing Loss*
Seconds-Minutes		BPPV
Minutes-Hours	Meniere's Disease	RV, MAV
Hours-Days	Labyrinthinitis	Vestibular Neuritis

• Can it be more than one type? Yes
o Example: vestibular neuritis followed by BPPV (benign paroxysmal positional vertigo).
o Distinguish: 1st episode vs. most recent episode
o How **often**? How **long**? How is it **changing**?

• Worrisome features:
o **D**iplopia, **D**ysarthria, **D**ysphagia, **D**ifficulty moving 1 side/limb, paraesthesia 1 side/limb
o **Bowel or bladder disturbance**
o True **loss of consciousness**
o Prominent **arrhythmia**
if one of the above is present, go to a Neurologist. Not related to ENT.
Prime clue #2: What is the duration of the attack? Is it seconds to minutes, minutes to hours, days or constant?

PERIPHERAL CAUSES OF VERTIGO
1] BENIGN PAROXYSMAL POSITIONAL VERTIGO (BPPV)

• Definition: Acute attacks of severe **transient** vertigo lasting seconds to minutes initiated by certain head positions accompanied by rotatory nystagmus (geotropic = fast phase towards the floor).
• The most common cause of episodic vertigo, and vertigo in patients > 40 years
• Multiple attacks per day with no hearing loss.
• Comes with nausea and vomiting

• Etiology:
o **Not identifiable**
o Closed head injury followed (surgery)

o Infections (15% vestibular neuritis)
o Prolonged bed rest
o Ménière's disease
o Recurrent vestibulopathy
o Migraine

• Pathophysiology:

o Canalithiasis: degenerative debris from utricle (otoconia) floating freely in the endolymph
o Posterior canal hangs down like the water trap in a drainpipe, allowing the crystals to settle in the bottom of the canal.

• History:

1. Onset: sudden
2. Duration: seconds-minutes
3. Frequency: bouts of vertigo then remissions
4. Severe vertigo

Accompanied by nausea and vomiting, Disease affecting the inner ear, multiple attack per day, short duration, disease of the young.

5. Associations with changes in head position

• Rolling over or getting into bed
• Assuming a supine position
• Arising from a bending position
• Looking up to take an object off a shelf
• Tilting the head back to shave

6. Worse on awakening in the morning
7. Chronic balance problems

*Pathognomonic: distinctively characteristic of a particular disease or condition

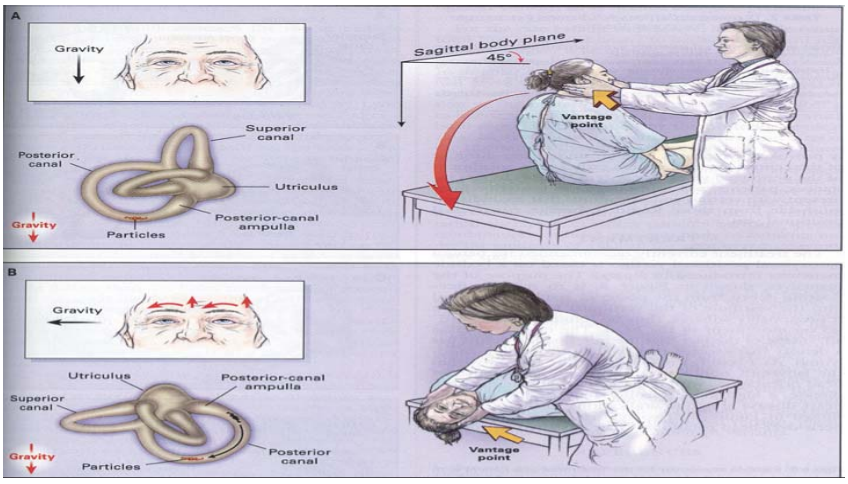
• Clinical approach (diagnosis):

History is virtually pathognomonic. It is the only type of vertigo:

- Multiple times per day
- Brief episodes
- Unaccompanied by auditory complaints

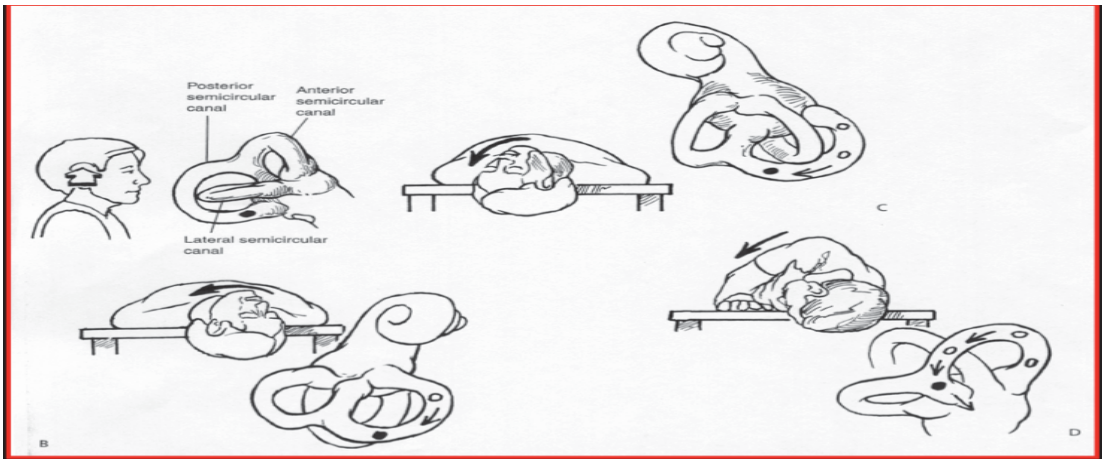
o Dix-Hallpike maneuver: diagnostic test to identify BPPV

- Maneuver: patient in sitting position w/legs extended → head is rotated 45° → lie down backwards quickly with head in 20° of extension → return to sitting position
- Positive test: nystagmus MUST be present = “Hagr’s 6 D’s”
  - Delay of ~20 seconds
  - Downward (geotropic) rotatory nystagmus
  - Duration <1 minute
  - Directional change (upon sitting up – direction reverses)
  - Dizziness (subjective)
  - Disappearance (fatigable: less vertigo each time test is repeated)



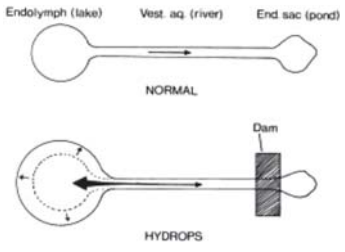
• Differential diagnosis
o Postural hypotension (Hx of antihypertensive drugs, cardiovascular disease…etc)
o Perilymph fistula

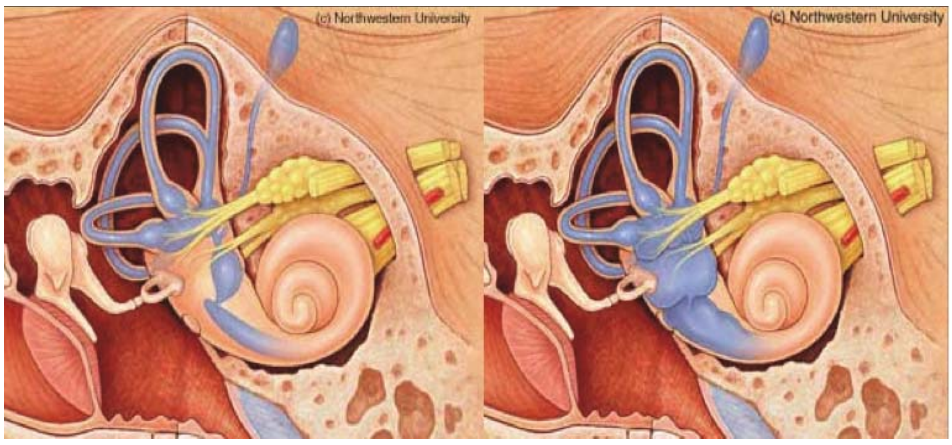
• Treatment:
o Reassure patient that process resolves spontaneously
o Particle repositioning maneuvers Epley's maneuver (performed by MD)

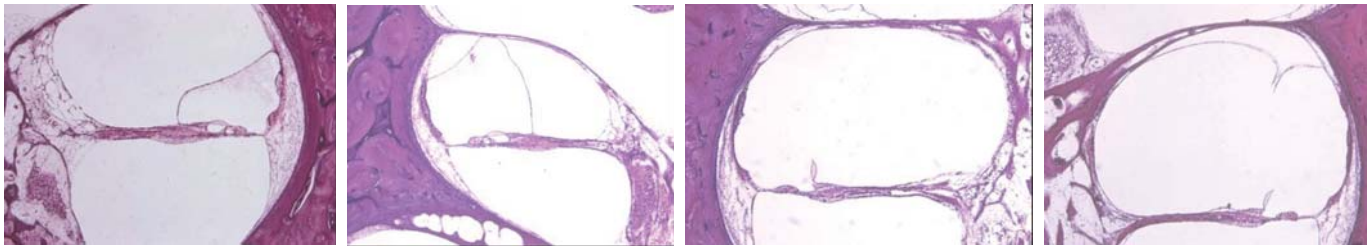


- Brandt-Daroff exercises (performed by patient)
o Surgery for refractory cases
o Anti-emetics for nausea/vomiting
o Drugs to suppress the vestibular system delay eventual recovery and are not used

2] MENIERE'S DISEASE The worst disease patients could have in their life.
• Definition: episodic attacks of vertigo, tinnitus, hearing loss, and aural fullness lasting minutes to Hours
Sever vertigo lasting for hours with nausea and vomiting. The patient is usually very depressed. The doctor said the attacks are like epileptic attacks but the patients are awake which affect their psychological status.
4-5 attack for half hour to an hour.
• Pathology: Unknown, this what they think might be due to:
1. **Decreased** endolymphatic **reabsorption**
2. Progressive **hydrops**
3. Membranous **ruptures**
4. Spillage of large amounts of **neurotoxic endolymph** into the perilymphatic compartment
5. **Healing** of the membranes
6. **Distortion and atrophy** of sensory and neural structures







• Causes: Overproduction or retention of endolymph (Pressure building up in the inner ear) Stress, fasting, could precipitate an attack. Relaxing could prevent or at least help.

1. Unknown
2. Autoimmune
3. Ischemia
4. Mumps
5. Syphilis
6. Hypothyroidism
7. Head trauma
8. Previous infection
9. Hormonal (Pregnant females are more prone)

• Course:

1. Early
   a. Predominant Vertigo
   b. Deafness
   c. Normal hearing between the attacks
2. Later
   a. Hearing loss stops fluctuating
   b. Progressively worse (50db)

• Diagnosis:

1. History & physical (unilateral)
2. Pure Tone Audiometry = **low frequency** SNHL
3. By exclusion: R/O other DDx

GOALS
1. Education
2. To **treat** the acute attacks
3. To **prevent** further attacks
4. To **improve hearing**
5. Vestibular rehabilitation

• Treatment:

1. Acute attacks:
   a. Prevent falls (bed rest)
   b. Head should be restricted
   c. Anticholinergics
   d. Antihistamines (Serc®)
   e. Phenothiazine
   f. Benzodiazepines
2. Long-term
   a. Medical
      • Low salt diet, diuretics (e.g. hydrochlorothiazide, triamterene, amiloride)
      • Local application of gentamicin (poisonous) to destroy vestibular end-organ, results in complete SNHL
      • Serc® (betahistine; anti-H_1) prophylactically to ↓ intensity of attacks
   b. Surgical: elective vestibular neurectomy or transtympanic labyrinthectomy
   c. Follow-up: must monitor opposite ear. Disease is bilateral in 35% of cases.

3] VESTIBULAR NEURONITIS

• Definition: acute onset of disabling vertigo often accompanied by nausea, vomiting and imbalance without hearing loss, that resolves **over days, leaving a residual imbalance** that lasts days to weeks.

• Etiology:

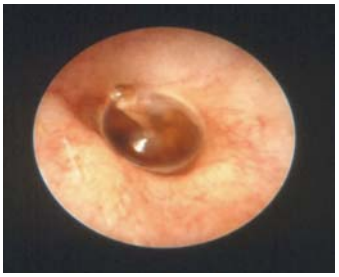
o Viral infection of vestibular organ (e.g. Measles, Mumps, Herpes Zoster)
o In 50%, infectious
Ototoxic medication (gentamycin injections)
*the doctor didn't mention this part.

• Abrupt onset
• Single, severe and prolonged episode
• No hearing loss
• No neurologic signs or symptoms
• Nystagmus

- Features:
    1. Acute Phase:
        a. Severe vertigo with nausea, vomiting and imbalance lasting 1 to 5 days
        b. Irritaitive nystagmus (fast component towards the offending ear)
        c. Patient tends to veer towards affected side
    2. Convalescent Phase:
        a. Imbalance and motion sickness lasting days
        b. Spontaneous nystagmus away from affected side
    3. Recovery (within 3 weeks). Incomplete recovery likely with specific risk factors: e.g. elderly.
    4. Repeated attacks can occur.

- Treatment: requires symptomatic treatment **ONLY**
    - **o Acute phase:** bed-rest, vestibular sedatives (Gravol: antihistamine; anticholinergic, antiemetic, sedative), diazepam (benzodiazepine)
    - **o Convalescent:** progressive ambulation, vestibular exercises (involves head & eye movement)

OTOTOXICITY



- Usually due to aminoglycosides e.g. gentamicin
- Patients complain of oscillopsia (visual distortion)

Investigations: For patient with vertigo

Audiology (PTA pure tone audiometry, ENG Electronystagmography , posturography, rotation chair), radiology (CT/MRI), blood tests (CBC, thyroid function tests (TFT), FT-Abs)

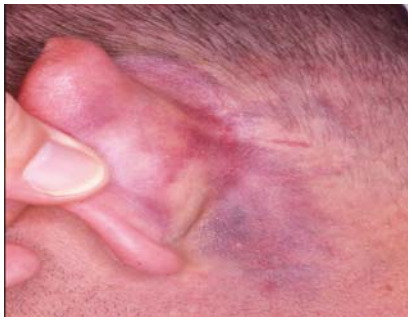
Cases:

Secretory Otitis Media (Glue Ear)

Fluid behind the interim

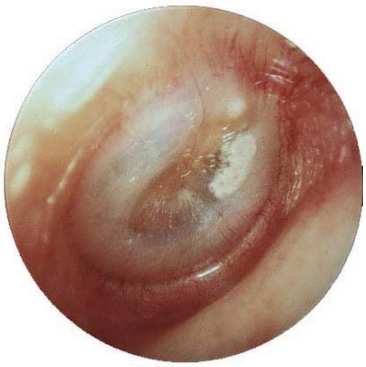
- 3 Y
- Recurrent OM
- Hearing Loss

Fracture Base of Skull



- Motor Vehicle Accident
- Left earache
- Hearing loss

Otosclerosis vs Tympanosclerosis?

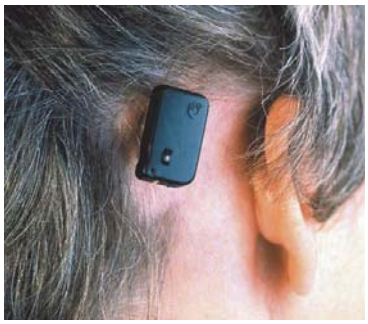


Tympanosclerosis

- 33 y
- No hearing loss
- Ear exam □

Cochlear implant
• What is this?
Hearing aid (Cochlear implant)
• which type of hearing loss?
Sensory neural hearing loss.

15 years old girl
• What is this?
B.A.H.A
• Which type of hearing loss?
For conductive hearing loss.



429 notes doctor didn't mention

CINICAL SCENARIOS

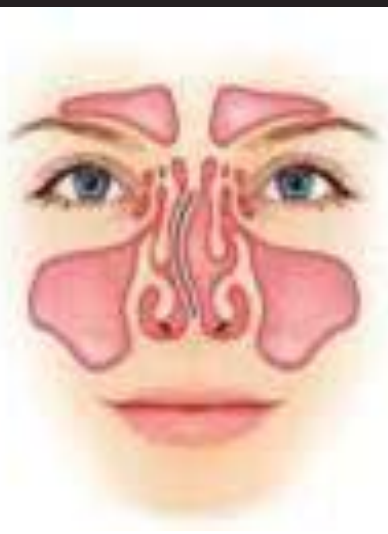
1. The patient who is having his first attack ever of acute spontaneous vertigo:
    a. DDx: Acute vestibular neuritis or cerebellar infarction
    b. How to differentiate?
        i. Clinically (General appearance of patient /nystagmus/head impulse test)
        ii. Radiology
    c. Note that: chronic and recurrent (BPPV)

2. The patient who has repeated attacks of vertigo, but is seen while well
    a. Recurrent spontaneous vertigo
        i. Menière's disease
        ii. Migraine induced vertigo
        iii. Perilymph fistula
    b. Recurrent positional vertigo
        i. BPPV

3. The patient who is off-balance
    a. Bilateral vestibulopathy
    b. Normal pressure hydrocephalus (triad of ataxic gait, urinary incontinence and dementia)
    c. Posterior fossa tumor

• Differential diagnosis (according to Hx)

Condition	Duration	Hearing Loss	Tinnitus	Aural Fullness	Other Features
BPPV	Seconds	-	-	-	-
Meniere's Disease	Minutes to Hours	Uni/Bi lateral Fluctuating	+	Pressure/ Warmth	-
Vestibular Neuronitis	Hours to Days	Unilateral	-	-	-
Labyrinthitis	Days	Unilateral	Whistling	-	Recent AOM
Acoustic Neuroma	Chronic	Progressive	+	-	Ataxia CN VII Palsy


1st Lecture:

DEAFNESS

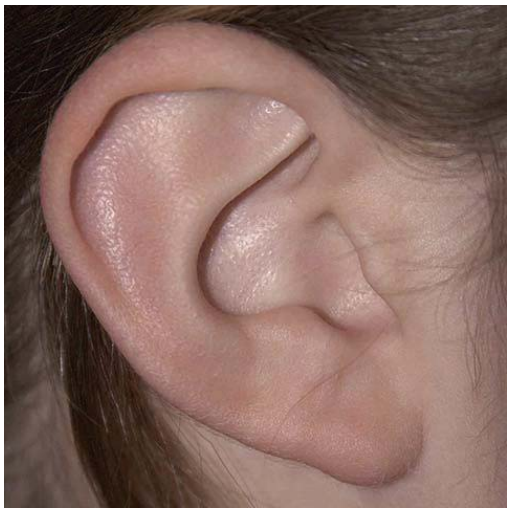
Done by: Reem Aldhahi
Revised by Yusra Al-Kayyali

The slides were provided by: Dr. Abdulrahman Hagr
Important Notes in red
Copied slides in black
Doctors notes in green
Our extra notes in blue
Possible MCQs mentioned or pointed by the doctor

HEARING

- Introduction
- CHL otosclerosis
- SNHL

– congenital, trauma, infection, noise, ototoxic, presbycusis, acoustic neuroma



H_2O O2

-Vision is like water (H_2O), hearing is like O_2. So hearing is more important as O2 is more important.
- (بن باز- ال الشيخ) are blind and it didn't stop them from being scholars and famous people; however no one deaf could do it.
-القران معجزه سمعيه. السمع ذكر في القران ويقدم السمع على العمى لاهمية السمع
-Down syndrome, cleft lip, spina bifida, sickle cell anemia, phenylketonuria (which we have screening program for it in KSA) all are common congenital problems but the COMMONEST congenital problem is hearing loss.
-The first sense in our body start to develop is hearing. At the age of 3 months the fetus starts to hear, full development of hearing is in the 5th month. Vision is weeks and weeks after.
-Hearing is the strongest sense in our body. It is the first sense developed and the last one lost in sleeping, sedation and after death.
It has the widest range of 360 degrees, it is not limited by the room you are in or corners, of course it has a range of space but vision for example is only 180 degrees horizontally and 145 degrees vertically.
-The best protected organ in the body is the ear: it is inside the Petrous Bone (العظم الصخري which is the strongest bone in the body, it protects the cochlea.
-Hearing loss has a major impact on life especially communication and learning.

How common is hearing loss?

• Overall about 1 in 10 (people usually don't recognize it but it increasing)
• 1 in 3 adults 65 - 75
• 1 in 2 older than 75
• 1-2% school age children
• 4% children under 5 (high incidence in KSA parent complain that their child can't understand غبي ومايفهم!)

Common and Important

Signs of Hearing Loss

• Talking louder than necessary (people misunderstand them as arrogant because they talk loudly)
• Turning up volume on the TV or radio (like grandfathers turning up the radio volume)
• Complaints that other people "mumble" (they think other people talk badly about them)
• Confusion of similar sounding words نحلة/ نخلة (people will make jokes about them)
• Inappropriate responses in conversation (as they try to communicate but they can't)
• Ringing or buzzing in the ears (can't hear normal talking but continuously hear annoying noises)
• Lip Reading (they develop it in order to communicate and understand what people are saying; however here in KSA it is considered inappropriate to stare at peoples' lips) اكثر شي في الاطفال عشان كذا الاهل ماينتبهون انه طفلهم مايسمع
– Watching a speaker's face intently
– Difficulty "hearing" someone behind (so they think he is ignoring them on purpose)
– Having difficulty on the telephone

Effects

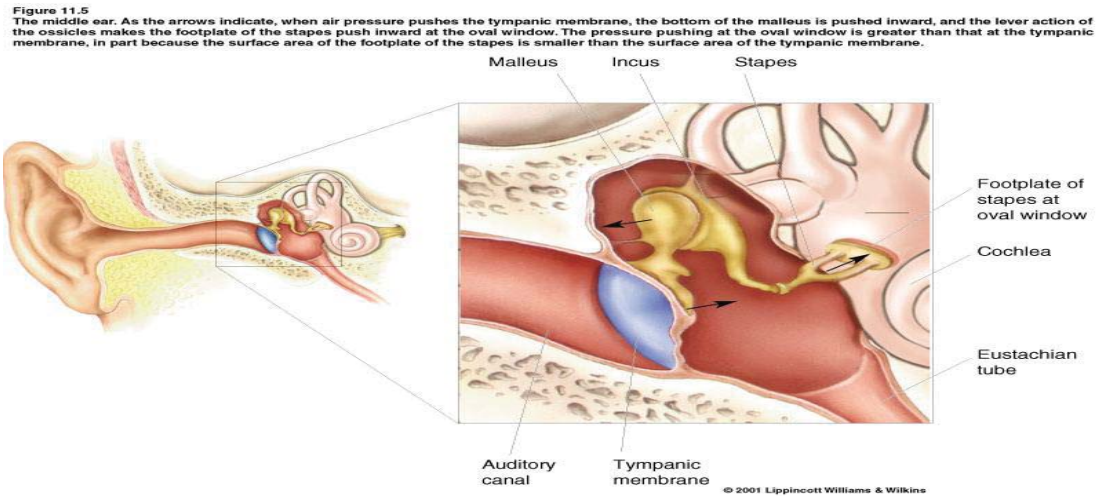
• Don't enjoy conversations – too much work (which is important to build relationships that's why they have their own language where they can build strong relationships with each other(deaf people among each other) sometimes its stronger than their relationship with their own mothers).
• People think you are an idiot
• Scared to try new contacts (don't want to meet new people)
• Scared to take new jobs
• Limits your world (جدي حبيب من المسجد للبيت it's not true but he's trying to protect himself from jokes)

Hearing Loss

• limit activities
• Isolation
• Depression
• Anxiety
• Insecurity (they think that people around them are talking about him/her)
" إذا كانوا ثلاثة فلا يتناجى اثنان دون الثالث "
• strain relationships (insecurity may progress to more serious problem where the patient may think people around him are plotting against him due to the constant whispering which he thinks is about him it may lead him to thinking that his mother wants to kill him).
• Increases psychosocial difficulties (a lot of psychiatric impact)

Deafness & Recruitment

Recruitment: The cochlea normally acts as a filter; it decreases loud voices and amplifies the low sounds. However here the cochlea filter sounds and therefore they can't protect themselves from loud sounds or hear low sounds. (That's why even though they can't hear well, loud sounds like that of a crying baby can bother them because they hear it louder than we do).

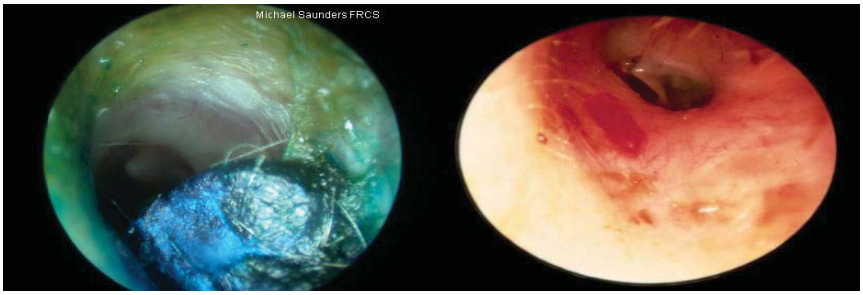


- Anything from the stapes footplate to the outside gives conductive hearing loss.
- Anything from the cochlea gives sensory hearing loss.
- Anything from the nerve give neural hearing loss; however the hearing test combines both of them together that is why we call them sensorineural hearing loss.
- All of them together called mixed.

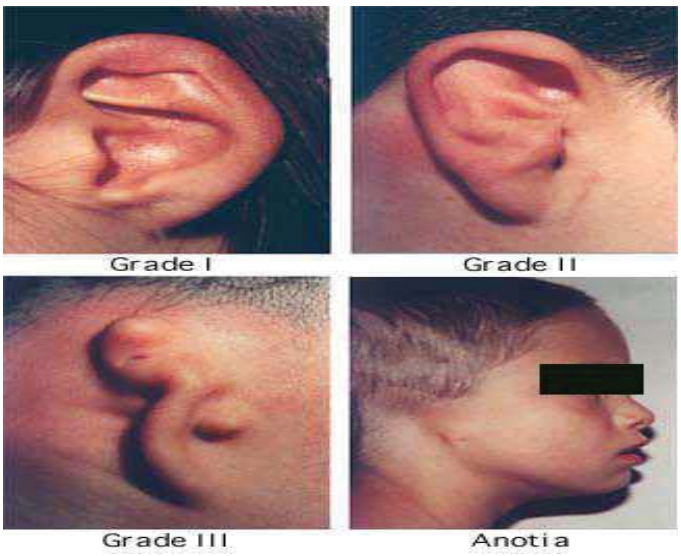
❖CONDUCTIVE DEFECTS

• Wax & foreign bodies (most common cause because of Q-tips we should not clean our ears. the ear cleans itself! (Unless there is a problem))
• Otitis externa
• Ear drum Scarring; perforation
• Otitis media (ASOM)
– Acute suppurative
– Otitis media with effusion (OME)
– Chronic otitis media (CSOM)
• Otosclerosis
• Ossicular chain disruption

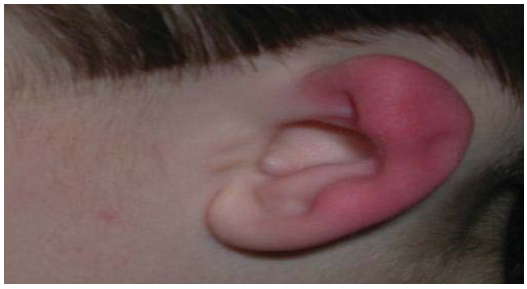
Wax



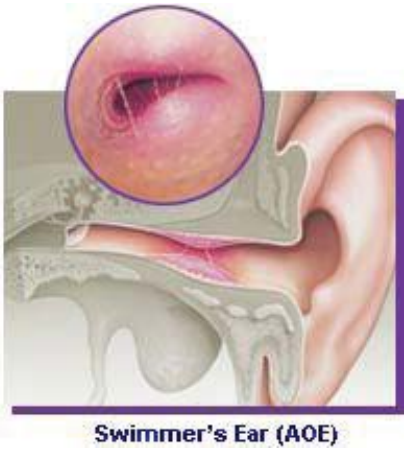
Microtia=صمعاء
الاذن الصغيره

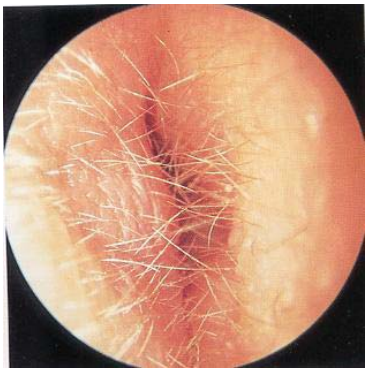


Atresia = رتق (atresia of the canal)

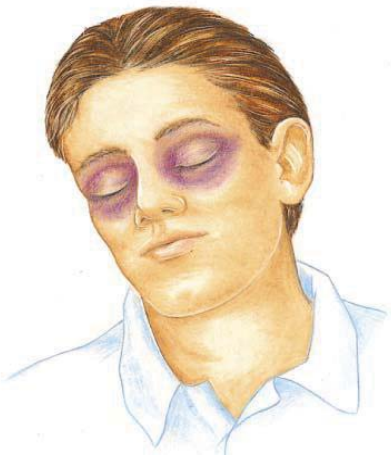


AOE (Otitis Media Externa)

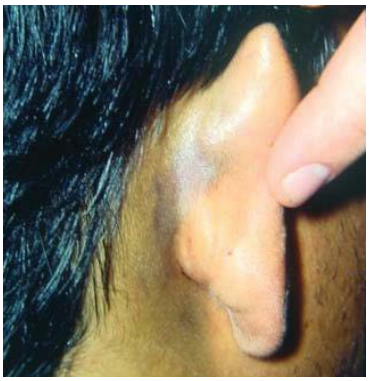




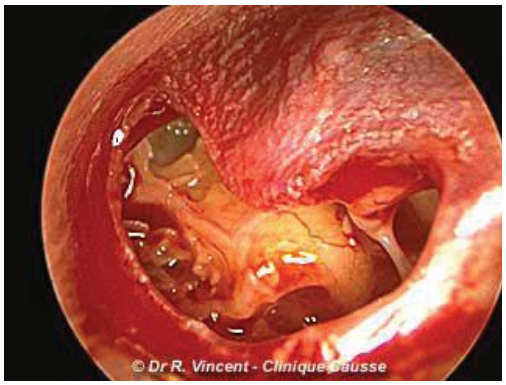
Racoon eyes sign (skull base fracture blood goes to the external auditory canal, tympanic membrane perforation and blood in the middle ear)



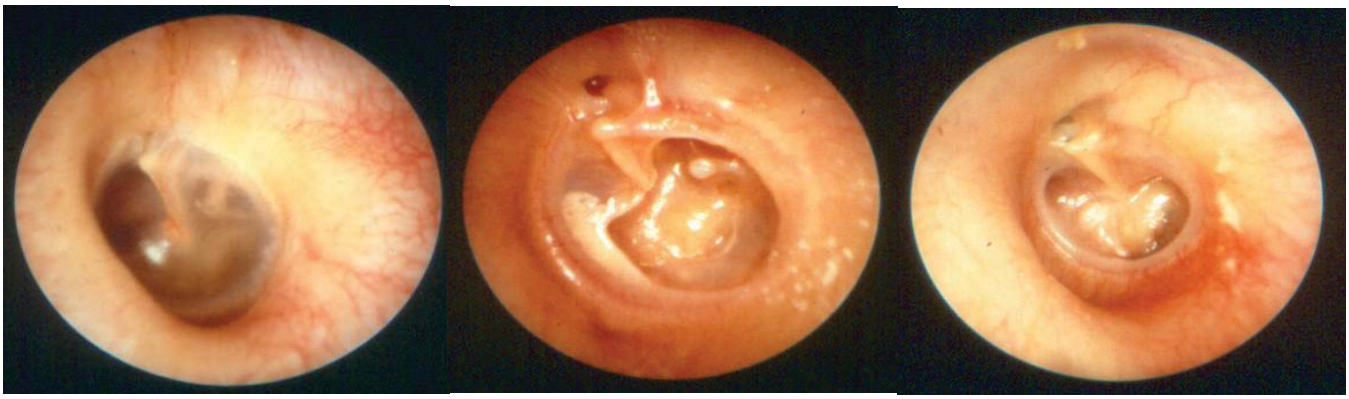
Battle's sign



Tympanic membrane perforation (this is a new trauma because there is bleeding so acute situation)



Drum Retraction (Adhesive OM)

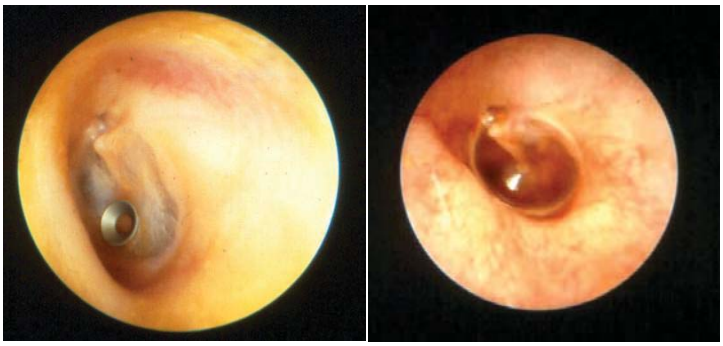


Normal

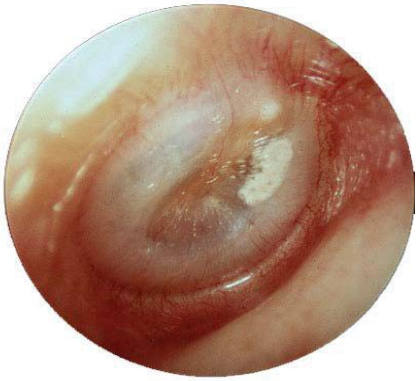
Ear drum sucked inside. It's called Atresia, Atelectatic ear
Adhesive OM

- The tympanic membrane gets sucked in because of Eustachian Tube Dysfunction and negative pressure which will suck the ear drum inside. We treat it by ventilation tube which prevents the ear from getting sucked inside by preventing the negative pressure.
So perforation and retraction both of them are causes conductive hearing loss

MEE

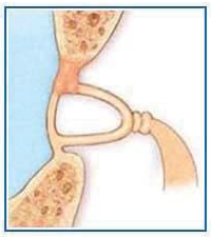


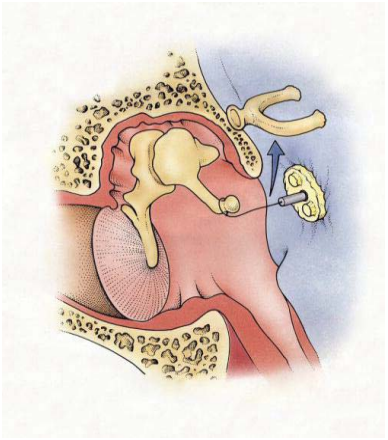
Tympanosclerosis (white tissue patches on ear drum) tymp=طبلي sclerosis=التحجر
It's very common. Come from recurrent infection and usually asymptomatic but it can cause conductive hearing loss.



Otosclerosis:

(Not common in KSA but common in west, oto= ear, sclerosis=stiffness)
• 10% otosclerotic lesions (10% symptomatic) (10% of Caucasian)
• Females: Male 2: 1 (because of hormonal changes)
• Middle-age
• Worse during pregnancy
• Stapedectomy (remove the stapes footplate and put prosthetic one = the patient starts to hear in the recovery room, however it may cause vertigo)





❖SENSORINEURAL HEARING LOSS (SNHL)

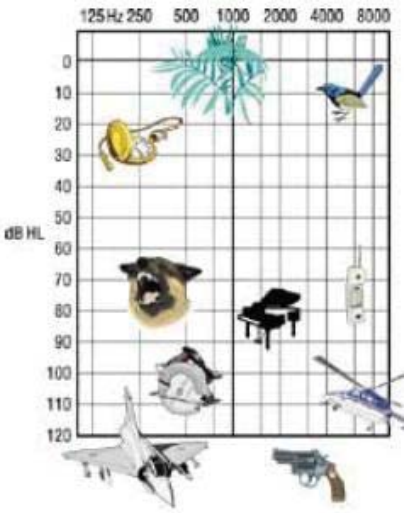
• Congenital
• Trauma
• **Infection**
• Noise
• Ototoxic
• Presbycusis
• Acoustic neuroma

Congenital hearing loss

• Deafness affects 0.2% (the commonest congenital anomaly in humans)
• SNHL attributed to
– 50% genetic factors
– 20-25% environmental
– 25-30% sporadic
• Genetic (we have the biggest center for cochlear implant)
– 75% AR (autosomal recessive common in KSA زواج الاقارب)
– 20% to AD (autosomal dominant)
– 5 % X-linked
• Over 400 syndromes

Noise induce SNHL

• Boilermaker's deafness (because people who work at boilermaker's usually develop deafness with time)
• One of the most common occupationally induced disabilities (example: Soldiers)
• Tinnitus (can drive people crazy it's most common in quite areas such as before sleep so they will develop insomnia, and depression! Some suicide)
– Commonly accompanied NISNHL
– Warning sign (wearing hearing aid increases the normal sounds and decrease the tinnitus)



98 *(No need to learn this)*

- 90 db for 8 hours (normal speaking 60-65 db)
- 95 db for 4 hours (should not stay in the place more than 4h)
- 100 db for 2 hours
- 105 db for 1 hour (when increase can cause death because it is a form of torture)

Ototoxicity

Medications

- Antibiotics
- Diuretics
- Antineoplastics
- Antiinflammatories
- Antimalarial agents
- Ototopic agents
- Others

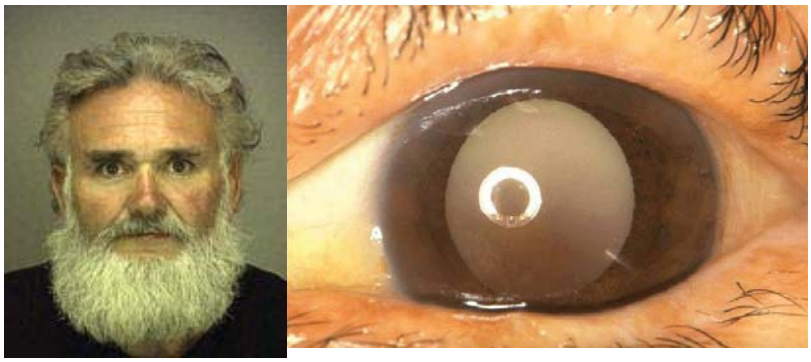
Higher risk

1. Renal failure (Elevated peak and trough levels)
2. Liver failure
3. Immunocompromise
4. Collagen-vascular disorders
5. Advanced age (> 65 years)
6. Prior ototoxicity
7. Concurrent use of known ototoxic agents
8. Preexisting HL or Vestibular
9. Bacteremia (fever)
10. Treatment course longer than 14 days
11. + ve FHx of AG ototoxicity

Presbycusis

It means hearing loss with aging. Most people at age of 40 start to have degenerations in body including hearing, usually they will accept having grey hair and decreased vision but not hearing loss. It's difficult for them to admit that there is a problem.

Presbycusis = Deafness + Tinnitus + Recruitment



Overview of Hearing Loss

- #1 Handicapping disorder
- 60% of Americans > 65 HL
- 90% of > 75 Y have HL
- HL + degenerative processes of aging.
- ½ Vestibular symptoms

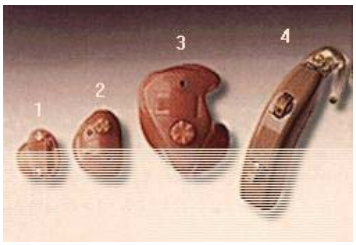
Problems with Diagnosis

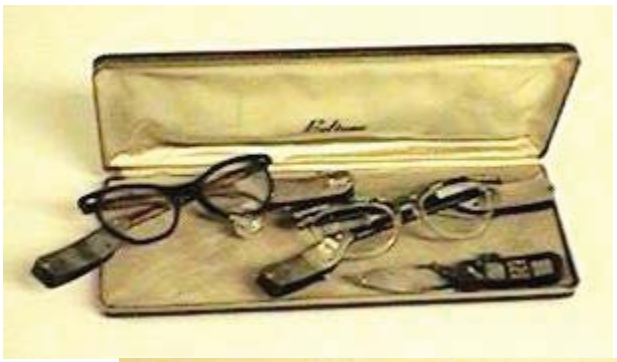
- Shame or embarrassment. (Especially from wearing hearing aids)
- HA social stigma
- Embarrassment prevents 15 million elderly people from getting help.

❖ Hearing Aids

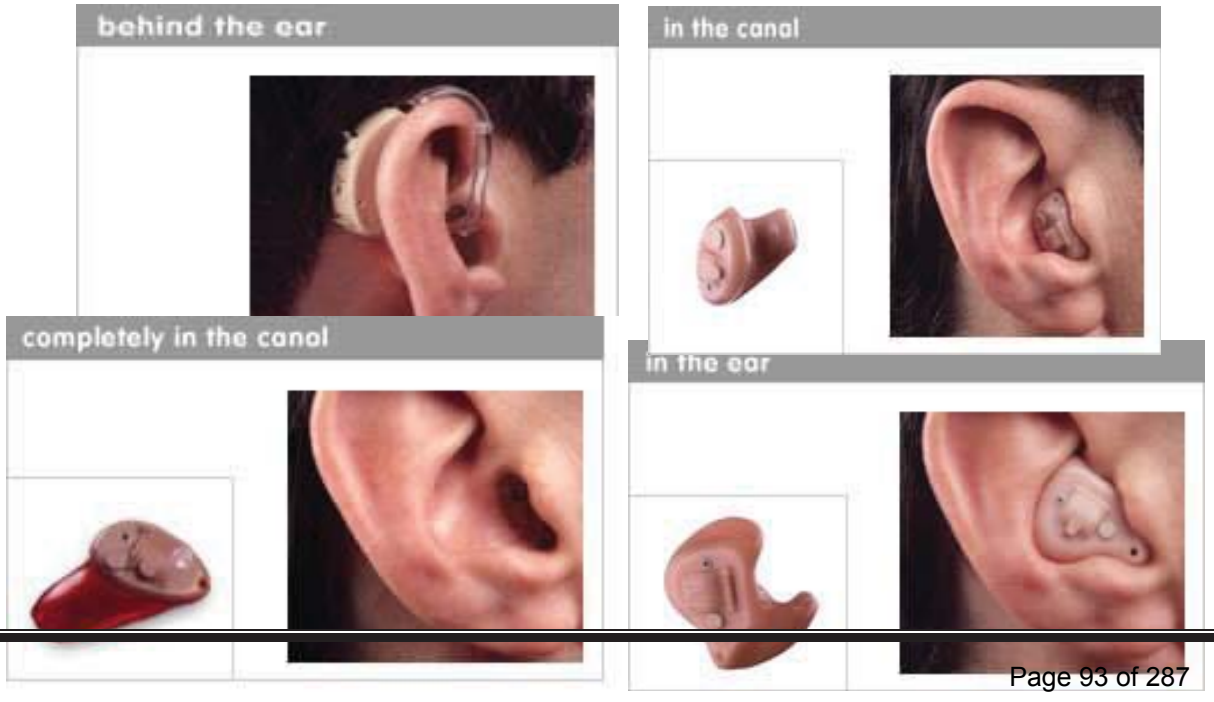
History

1550 by Girolamo Cardano when he saw that sound could be transmitted through the teeth.





Hearing Aids

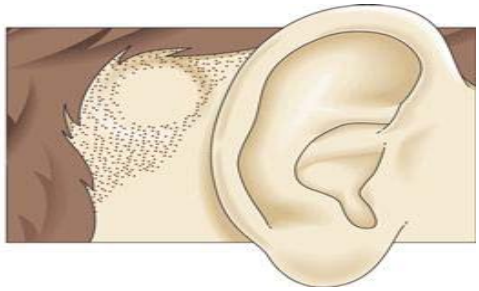


❖ Cochlear implant: (1827-1745) فولتا الكساندرو

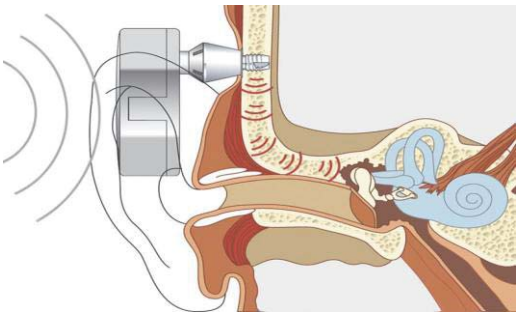
It's started when Volta put a battery in his head and turned it on, he passed out. When he woke up he noted down that he notice that the battery produced electricity (voice).

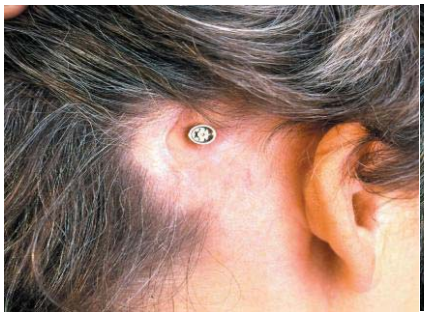
Bone Anchored Hearing Aids B.A.H.A

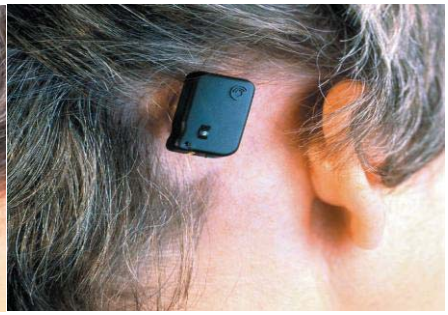
Fixing the implant on bone using titanium; it doesn't react with body. Used for conductive hearing loss.



Direct bone Conduction





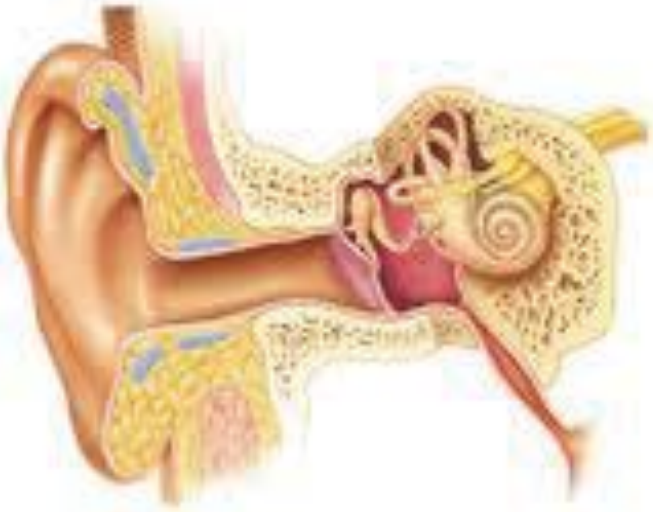


Auditory brainstem implant

This is used in a patient who does not have a cochlea(congenitally or due to fracture) or a nerve. We go to the brain stem and the cochlea nucleus and stimulate the nerve. They may develop arrhythmia or respiratory arrest from the electrical stimulation.

430 Teams

Diseases of the Ear, Nose and Throat



8th Lecture:

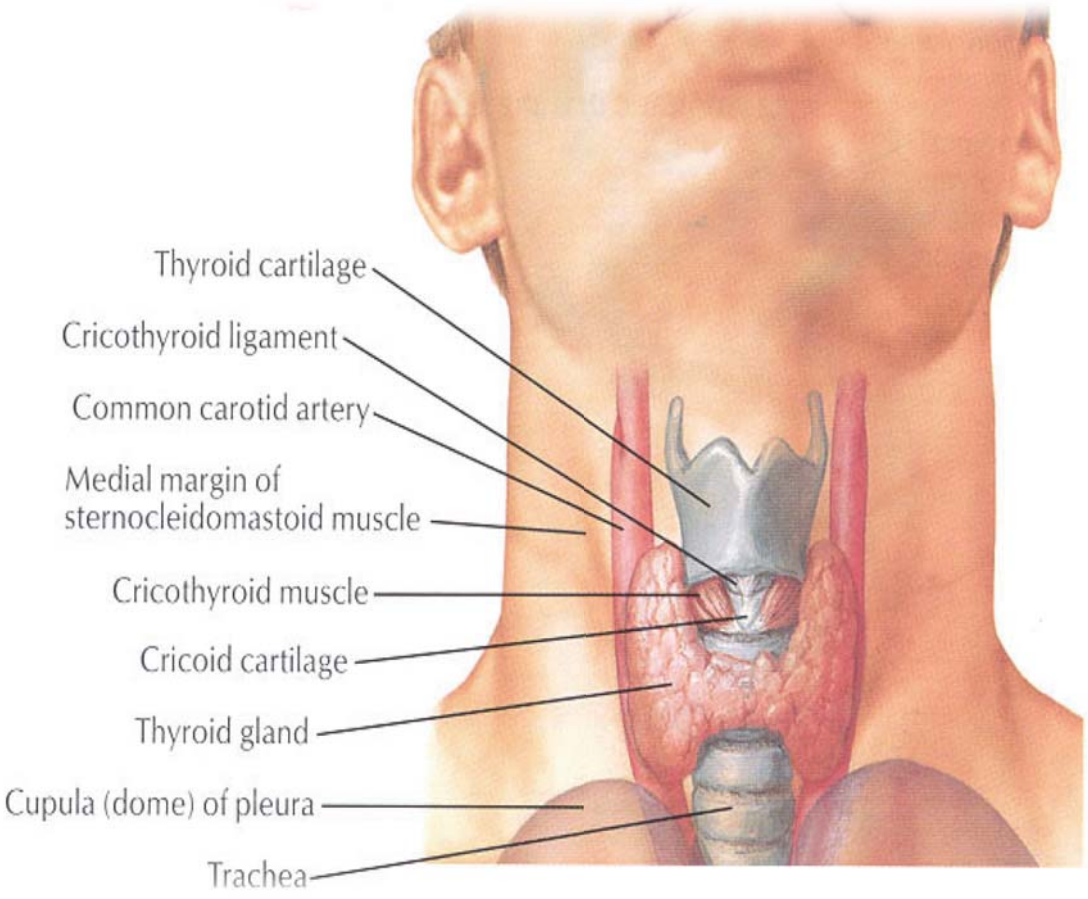
Evaluation and Management of the Patient with a Neck Mass

Done by: Shatha Al Harbi

The slides were provided by dr. aldhahri
Important Notes in red
Copied slides in black
Your notes in green/ blue

Anatomy of the neck:

1. Anatomical landmarks:

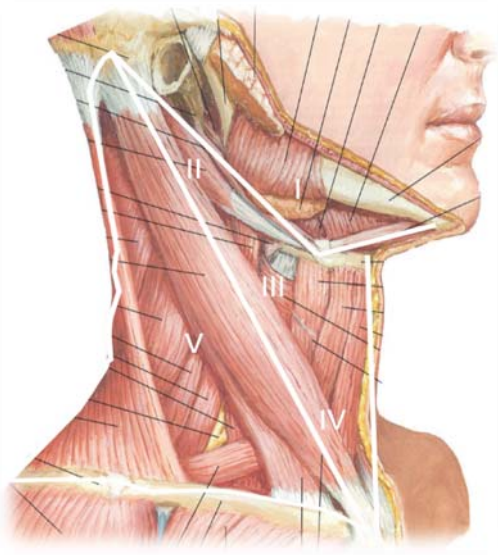


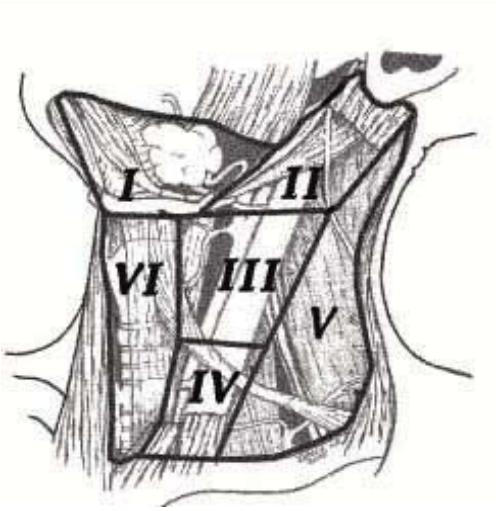
*the clavicle, sternal notch and sternocleidomastoid can be assessed in all patients.
*While thyroid cartilage, cricoid cartilage and trachea maybe difficult to assess in some.

2. Triangles of the neck (lymphatic drainage levels):

Level 1	Submental and submandibular region.
Level 2	Upper jugular & digastric lymph nodes.
Level 3	Mid-jugular lymph nodes.
Level 4	Low jugular lymph nodes.
Level 5	Behind the sternocleidomastoid.
Level 6	Anterior compartment. (mid-line)

*levels 1-4 are anterior to sternocleidomastoid. (Anterior triangle).
*level 5 is posterior to sternocleidomastoid. (Posterior triangles).





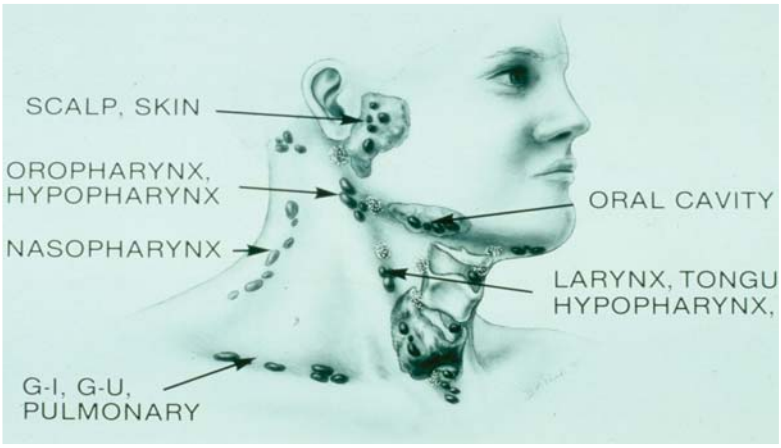
Lymphatic drainage is from up to down and medial to lateral so metastatic lymphatic pathway is in that direction too. For example, a lesion under the tongue may metastasis to level one (submental area).

3. Carotid bulb:
*Dilated area located at the bifurcations of the carotid arteries and containing numerous baroreceptors that function in the control of blood pressure by mediating changes in the heart rate.
*Located At the level of hyoid bone, helps in detecting neurovascular tumors in patients complaining of neck masses.

General considerations:

AGE	LOCATION
Pediatrics (0 – 15 years): mostly benign Young adults (16 – 40 years): similar to pediatric Old adults (>40 years): high risk of malignancy	Congenital masses: consistent in location. Metastatic masses: location maybe variable, follow the track of spread to primary tumor.
In elderly it <u>almost</u> always a malignancy,	

Metastasis Location according to Various Primary Lesions



Important points:

- Commonest congenital neck masses: 1. Thyrogolossalduct cyst. (Located at mid-line below hyoid bone)
  2. Branchial cleft cyst. (At level II, lateral part of the neck).
- Nasopharyngeal carcinoma metastasis to level II most commonly.
- Most common route of metastasis of head and neck tumors is lymphatic spread.
- When a patient is found to have enlarged lymph nodes at level II, you should examine the tonsils.

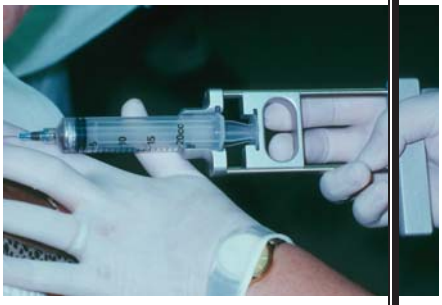
Diagnostic Steps:

History	Physical examination
1. Developmental time course: • Very quickly: infectious (e.g. over 3 days). • Quickly: malignant (e.g. over a month). • Very slowly: benign (e.g. over 2 years). 2.Associated symptoms: • Otalgia: referred pain from larynx, nasopharynx, base of tongue by 9th cranial nerve. • Dysphagia. • Horsiness of voice: a change in the voice, alarming with history of cancer, smoking or otalgia. • Diplopia: tumor invading cavernous sinus. • Facial numbness: tumor invading trigeminal nerve ganglion. Distinguish from Bell's palsy where the symptoms are sudden. 3. Personal habits (tobacco, alcohol, chewing shamah). 4. Previous irradiation (thyroid cancer).	Under good illumination assess: 1. Oral cavity: buccal & sulcus areas, under tongue, palate, tonsils, area between buccal and maxilla). 2. Neck lymphatic levels & other head lymph nodes. 3. Nose: using fiber-optic, examine the nasopharynx and larynx. 4. Ear.

Investigations:

1. Fine needle aspiration biopsy (FNAB): most important tool in evaluating neck masses & gold standard for diagnosis

Indications	contraindications
*a neck mass persistent after an empirical antibiotic course. (When you suspect an infectious process in a patient presented with a neck mass, give antibiotics and follow up after two weeks). *small gauge needle.	*if vascular lesions are suspected: e.g. hemiangioma, carotid body tumor (it may cause hypotension for patient).



Open biopsy is indicated when:
1. 4 collections of FNA are inconclusive.
2. Suspicion of lymphoma (history of low-grade fever).
3. Suspicion of tuberculosis.

*Nasopharynx is the 2nd most common cancer location after thyroid in head and neck.

*incidence of nasopharyngeal carcinoma is linked to Epstein Barr virus & genetic alterations in Saudi population.

2. Computed tomography: 1st radiological imaging to order. Helps in assessing site, size (more than 1.5 cm in considered pathological), extent, primary lesion and staging.
* Avoid contrast in thyroid lesions

3. MRI: for soft tissue visualization (detection of deep enlarged lymph nodes) and vascular lesions.

4. Ultrasonography: important tool in diagnosing thyroid masses, cystic lesions, pediatric patients (no need for general anesthesia), guided biopsy. (Can detect masses as small as 2mm).

5. Radionuclear imaging: for salivary tumors, thyroid masses, bone scan.
* Not indicated in thyroid masses unless there is a suspension of thyrotoxicosis.
*PET scan is indicated for functional assessment of unknown primary tumors. There will high up take of glucose in active metabolic areas of malignancy.

General rules of nodal mass workup in adults:
1. Any solid asymmetric mass **MUST** be considered a metastatic neoplastic lesion until proven otherwise.
2. Asymptomatic masses: 12% are cancers. (80% of squamous cell carcinoma patients are presented with sole complain of neck mass).
3. Ipsilateral otalgia with normal otoscopy: direct attention to tonsil, tongue base, supraglottis and hypopharynx.
4. Unilateral serous otitis: direct examination of nasopharynx (obstruction of Eustachian tube).
5. Open biopsy is contraindicated in squamous cell carcinoma, because it changes the lymphatic drainage pathway, introducing metastasis in new locations.
6. When the primary lesion is not located after FNA of base of tongue, nasopharynx, larynx and after tonsillectomy:
    *if the histological subtype is established by FNA, start treatment.
    *if the histological subtype is not known after FNA, do open biopsy.

Benign tumors of parotid gland:
Most common is benign pleomorphic adenoma (it is the most common in submental, sublingual, minor salivary glands).
*2nd most common is warfin tumor (papillary cyst adenoma).
Malignancies: (the larger the gland the lesser the chances of malignancy development**)**

Parotid gland	Submandibular glands	Sublingual glands	Minor salivary

1. Mucoepidermoid tumor. 2. Adenocystic carcinoma.	1. Adenocystic carcinoma. 2. Mucoepidermoid tumor. 40% chance of malignancy.	1. Adenocystic carcinoma. 2. Mucoepidermoid tumor. 60% chance of malignancy.	1. Adenocystic carcinoma. 2. Mucoepidermoid tumor. 80% chance of malignancy.

Case 1

A 2 years old child presented to your clinic with fever, decreased oral intake, runny nose and crying for the past 5 days. On examination, the child is irritable; there is a large left neck mass, red and tender.

Infection (lymphadenitis) developed an abscess. Treatment: drainage and antibiotics.

Case 2

65 years old man injured his neck while shaving, 4 days later he developed neck swelling and low-grade fever.

Cellulitis, treat by antibiotics.

Case 3

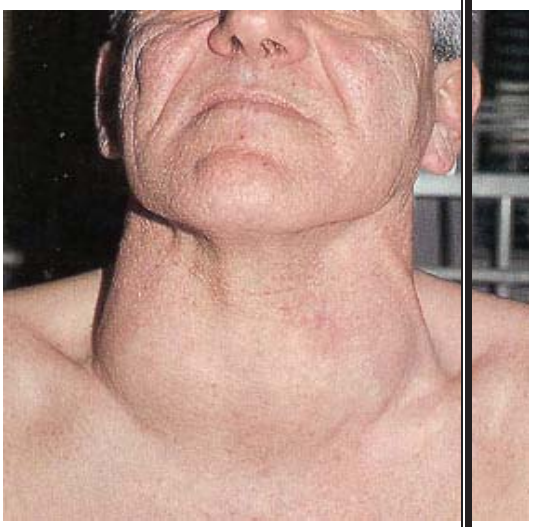
28 years old woman has a neck mass for the past 8 months; midline mass of both sides of the neck, started snoring, choking, no weight loss, no pain, and no voice changes.

BaGoiter (thyroid enlargement). In history: symptoms of hypo-/ hyperthyroidism, family history of thyroid cancer, radiation exposure.

*Most important investigations are FNA + ultrasound.

*follicular thyroid lesions are benign, treated surgically: thyroidectomy.

*thyroid carcinoma: patient will complain of horsiness, swelling is more towards one side of the neck and weight loss.



Case 4

An elderly woman presented with left temporal skin lesion, growing for the past 8 months, no response to creams.

Basal cell carcinoma.

*examine the eye (frozen eye if orbit is invaded), facial nerve, parotid gland, and other skin lesions.

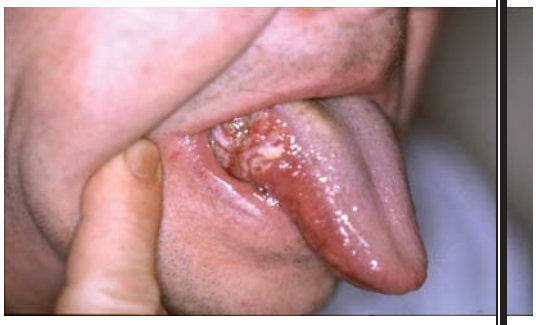
*confirm by biopsy from junctional area, containing both normal and neoplastic tissue. (At the center, there will be only necrosis)

*CT scan to assess extension.

*Basal cell carcinoma treated by excision/ Squamous cell carcinoma by excision and radiotherapy.

Case 5

40 years old male presented with right tongue lesion for the past 5 months; slowly growing, causing difficulty in eating, he is a smoker.



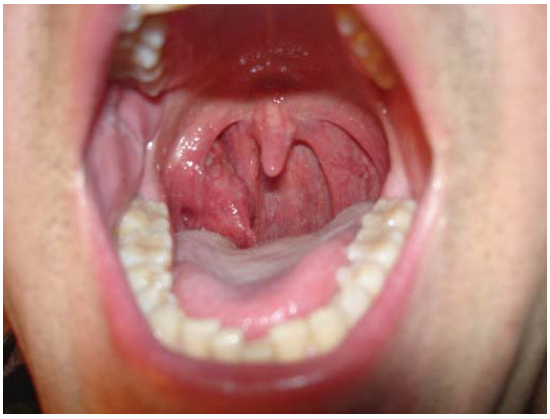
Oral cancer (squamous cell carcinoma most common histological type).

*take a scrap tissue + CT (neck, chest, and abdomen).

*T1 tongue is more aggressive than T4 pharynx.

Case 6

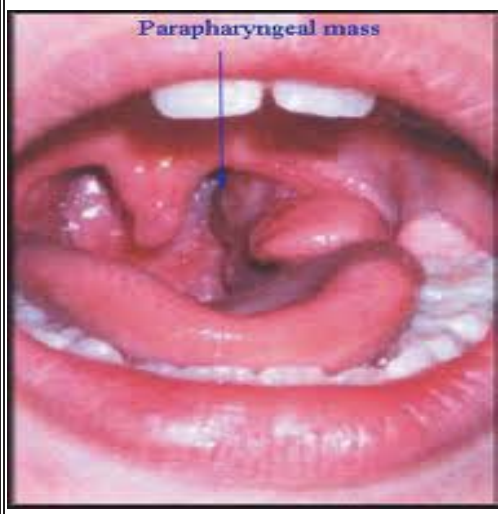
Previously healthy young man presented with recurrent tonsillitis, constant throat pain, he was on four antibiotic courses with no improvement, his throat pain is worse upon swallowing, he has right ear pain and decreased weight over the past 6 weeks. On examination, he has enlarged right tonsil.



Tonsillar carcinoma, the uvula is in med-line.

If on examination, he had multiple cervical lymph nodes enlargement, low-grade fever, hepatosplenomegaly.

Lymphoma



Case 7

Patient is completely asymptomatic; dentist referred her for a mass behind the right tonsil.

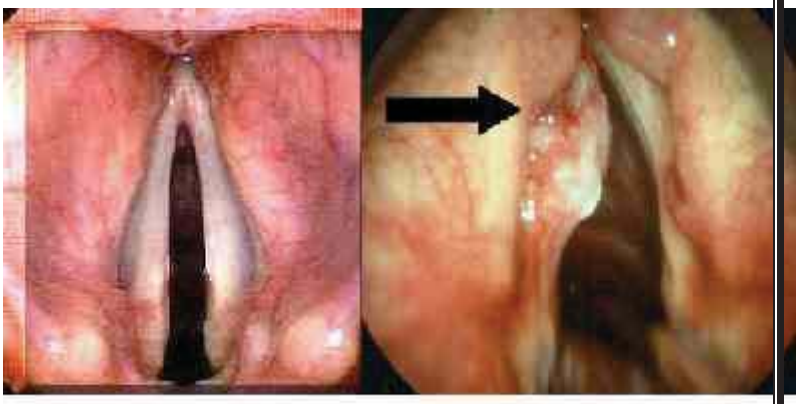
Para-pharyngeal space tumor.

Case 8

65 years old man complains of horsiness for the past 10 months, heavy smoker (2packs/day for the past 14 years), mild stridor, decreased weight, cannot walk because of stridor. On examination, he has a neck mass 2x2 cm, vocal cord paralysis, mass on right vocal cord.

Squamous cell carcinoma, to confirm it do open direct biopsy.

*treat by total laryngectomy.



Normal Cords Cancer (arrow)

Case 9

Patient asymptomatic has a left parotid mass for the past 6 months.

Diagnose by FNA, treatment is surgical excision.

Case 10

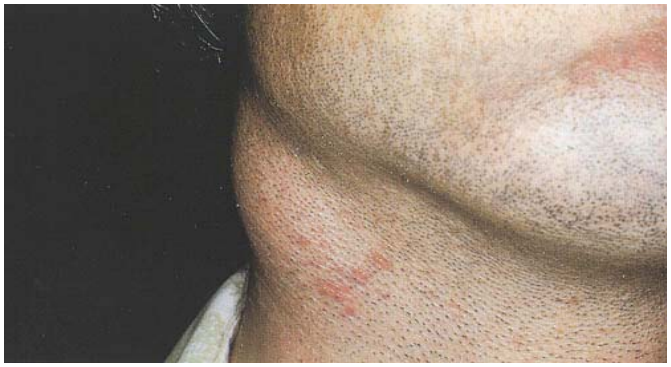
Elderly woman using denture complains of pain and gum bleeding.

Squamous cell carcinoma of buccal mucosa, confirm by biopsy.

Case 11

Elderly man with a left neck mass that does not change in size, has been there for at least 14 years, disappears sometimes.

Branchial cleft cyst



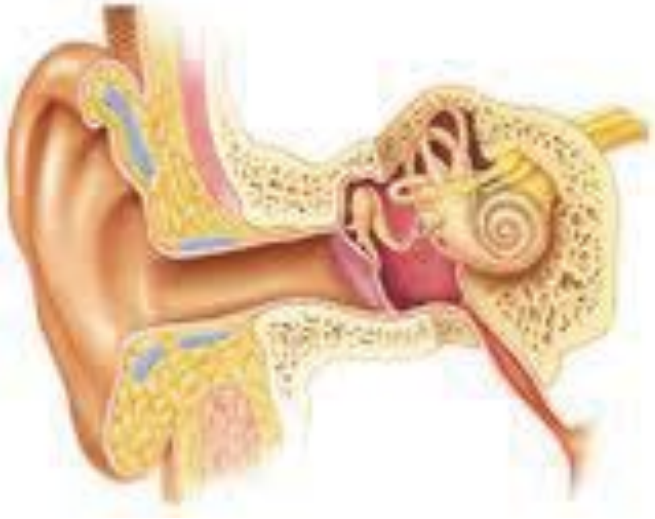
Thyroglossalduct cyst: disappears with tongue protrusion. Treat by cyst trunk procedure (resect cyst, track, body of hyoid bone).

Pyogenic granuloma: repeated irritation of the lip, usually with history of smoking and old age.

Anaplastic thyroid carcinoma has the worst prognosis of thyroid tumors.

430 Teams

Diseases of the Ear, Nose and Throat



1st Lecture:

Salivary Gland Tumors

Done by: Bushra Alhela

The slides were provided (Dr. Saleh Aldhahri)
Important Notes in red
Copied slides in black
Your notes in green/ blue
Titles and subtitles in this color
Highlight possible MCQs mentioned or pointed by the doctor

What is the most common tumor of the parotid?

- 90% of parotid tumors are benign tumor
- 90% of parotid tumors are Pleomorphic adenoma (other name :benign mixed tumor)
- Second most common benign tumor Warthin tumor

If you go to submandibular gland , what's the chance of patient with submandibular tumor having malignant tumor ?
40%

What are the benign tumors of submandibular gland exactly the same ; Pleomorphic adenoma and Warthin tumor.

How about sublingual? What is the chance that they have malignant tumor ?
60%
What are most benign tumors? The same.

How about minor salivary gland (found in palatal buccal, , and tongue) ?
80-90% malignant.
Mast common benign ?the same

The key point here is the larger the gland the less likely the malignant behavior, and the smaller the gland the most likely that the patient have malignancy.

When I see a minor salivary gland in the palat →immediately this most likely malignant tumor.

While the parotid the opposite → most likely benign

Of Corse, this is if we are talking about general behavior ;general principle. However symptoms could be variable.

I could present you a patient with acute 6 or 7 weeks rapidly growing mass in the parotid →this mast likely malignant.

We have 4 tumors we have to remember
2 benign : -Pleomorphic adenoma
-Warthin tumor

2 malignant: -mucoepidermoid carcinoma
-Adenocystic carcinoma

The parotid the most common is mucoepidermoid carcinoma
The second most common is Adenocystic carcinoma
The rest of salivary gland is revers

Diagnosis : we have to do FNA.

investigation :CT scan ,MRI as needed.

Treatment of salivary gland tumors usually surgical

If I tell you this is pleomorphic adenoma, what is the classical treatment?
Superficial parotidectomy ;you remove all the tumor above the facial nerve.
If it is malignant we be more aggressive in the surgery whether we do radical were we remove facial nerve and everything or we just save the facial nerve it's quite different based on different pateints.

Alarming sign in parotid mass

- Facial nerve paralysis
- Pain
- Skin fixation
- Presence of lymph nodes

So, if you have a parotid mass with lymph node and rapidly growing this is very bad sign ,this is most likely malignant.

If you have pain in the parotid tumor ,this is very bad sign.

If you have patient with facial nerve paralysis this is not benign , this is most likely malignant.

If you have skin invasion this is most likely malignant.

Patient presented with left neck mass for almost 3 years , CT scan shows large cystic lesion with lymphoid component in it , what is the treatment ?
Superficial parotidectomy

Patient did well and alright ,for about 4-5 weeks he started to have rapidly growing mass in the left side of her neck with low grade fever ,weight loss ,multiple cervical lymphadenopathy and hepatosplenomegaly. What is the most likely diagnosis ?
I couldn't hear it

What is the cystic lesion that stays for years without causing any problem ,get infected sometime and disappear ,located exactly mid side of the neck?
Branchial Cleft Cysts

What is the treatment of Thyroglossal Cyst?
Thyroglossal Cyst most of the time they have tract that go inside ,behind the hyoid bone ,and coming through the base of the tongue.
So, if just excise the cyst it will come back again, so we do cyst tract procedure and this is a very common question by the way(if you forget the name it's not a big deal for me),the treatment is the following :dissecting the cyst with the tract and the body of hyoid bone .

Doctor's slides

Salivary Gland Tumors

- Enlarging mass anterior/inferior to ear or at the mandible angle is suspect
- Benign
  - Asymptomatic except for mass
- Malignant
  - Rapid growth, skin fixation, cranial nerve palsies, pain

- Diagnostic tests
  - Open excisional biopsy (submandibulectomy or parotidectomy) preferred
  - FNAB
    - Shown to reduce surgery by 1/3 in some studies
    - Delineates intra-glandular lymph node, localized sialadenitis or benign lymphoepithelial cysts
    - May facilitate surgical planning and patient counseling
    - Accuracy >90% (sensitivity: ~90%; specificity: ~80%)
  - CT/MRI – deep lobe tumors, intra vs. extra-parotid
- Be prepared for total parotidectomy with possible facial nerve sacrifice

Branchial Cleft Cysts

- Branchial cleft anomalies
- 2nd cleft most common (95%) – tract medial to XII nerve between internal and external carotids
- 1st cleft less common – close association with facial nerve possible
- 3rd and 4th clefts rarely reported
- Present in older children or young adults often following URI
- Most common as smooth, fluctuant mass underlying the SCM
- Skin erythema and tenderness if infected
- Treatment
  - Initial control of infection
  - Surgical excision, including tract
- May necessitate a total parotidectomy (1st cleft)

Thyroglossal Duct Cyst

- Most common congenital neck mass (70%)
- 50% present before age 20
- Midline (75%) or near midline (25%)
- Usually just inferior to hyoid bone (65%)
- Elevates on swallowing/protrusion of tongue
- Treatment is surgical removal (Sis trunk) after resolution of any infection

Head and Neck I, II, III

I: How to evaluate neck and thyroid masses.

II: Salivary gland diseases and tumors.

III: Mucosal tumors.

H & N(I):

***Anatomy of the neck:**

anterior triangle

posterior triangle

midline

- anterior and posterior triangles are separated by sternocleidomastoid muscle.

- 5 levels of the neck:

(: تلقوها في الملزمة حقت 429 ص 441 مع رسمة

-carotid sheath contents:

تلقوها في الملزمة ص 442

***How to approach a patient with neck mass ?**

A-Hx: 8 Qs

1-Age and gender

2-C.C

3-H.P.I: onset and site

4-past medical and surgical

5-Family Hx

6-social: smoking, alcohol

7-medication

8-allergy

*Risk Factors:

1-Smoking(very important)

2-Alcohol consumption

3-Family Hx: very important in thyroid cancer

4-Hx of other cancer

5-Exposure to radiation: very important in thyroid cancer

B-Ex: 8 areas

1-neck: examine the mass and examine the other levels of the neck(5levels)+thyroid

2-face and parotid area

3-mouth and oral cavity

By the scope, examine:

4-nose

5-nasopharynx

6-oropharynx

7-hypopharynx

8-larynx

C-D.D:

1- inflammatory:

1- age less than 40

2-duration less than 1 week

3-patient has fever, tenderness, redness

4-CBC: high WBC الدكتور ما ركز عليها مرة

treatment: give antibiotic for 2 weeks and ask the patient to come back. if no improvement, deal with it as non inflammatory mass.

2-non-inflammatory:

1-CT **except** in thyroid mass do US 2-FNA

Age:

≤15 benign

16-40 mostly benign

>40 high risk of malignancy

Duration:

less than 1 week infection

weeks-months malignant

years benign

***Congenital Neck Masses**

موجودة في الملزمة ص444

حأذكر بس النقاط اللي ركز عليها الدكتور في كل نوع

1-Epidermal&Sebaceous Cyst:

ذكر كل النقاط اللي مكتوبة في الملزمة عن هذا النوع

2-Branchial Cleft Cyst(the doctor said it is very important for the exam**):** common in pediatric, there are 4 types, type 2 most common, usually in level 2, usually after URTI, treatment: antibiotic for 2 weeks, if no improvement do CT and FNA

3-Thyroglossal Duct Cyst:

ذكر كل النقاط اللي مكتوبة في الملزمة عن هذا النوع

4-Vascular tumor:

ما شرحها الدكتور قال مو مهمة

***Thyroid Masses**

-Anatomy, Arterial supply, venous drainage:

اقرؤوها من الملزمة ص445

-how to approach a patient with thyroid mass?

هذا هو الكلام اللي ذكره الدكتور في المحاضرة

1-Hx: as mentioned before

2-Ex: as mentioned before

3-Investigations: US+FNA

-The result of FNA:

1-Benign:

follow up every 6 months

2- Inconclusive:

repeat FNA with US guidance

3-Intermediate:

do hemithyroidectomy:

a-if benign: follow up

b-if malignant: treat

4-Malignant: treat

-what is the most common type of thyroid cancer?

papillary thyroid cancer

-Treatment of Thyroid cancer:

1- If well differentiated: do total thyroidectomy+ Iodine 131

2-If poorly differentiated(most common type is medullary):

do total thyroidectomy+ neck dissection (removing some lymph nodes with the thyroid)

-complication of thyroid surgery:

1-Recurrent laryngeal nerve injury(most important) 12%

2-Hypocalcemia 10%

3-Hematoma 4%

-Indication of thyroid surgery:

ما ذكرها الدكتور بس اقروٗوها احتياط ص447

H & N (II):

***salivary glands**

-Anatomy and Physiology:

الدكتور ذكر كل شيء مذكور في الملزمة ص 449

the amount of secreted saliva: 1-1.5 L/ day

-Infections & Benign Lesions:

الدكتور تقريبا ذكر كل شي في الملزمة ص450و451

-sialolithiasis:

الدكتور ذكر كل شي موجود في الملزمة وهذي ملاحظات اضافها

-most important symptoms is recurrent swelling and pain with eating specially sour things

Treatment: treat the infection if the patient presented with infection, if not do CT to see the stones then the treatment is to remove the stone with or without the gland. this depends on the site of the stone:

a- distal in the duct: remove the stone only

b-deep in the duct: remove the stone and the gland

-Tumors:

ذكر كل شي في ص451

ماقال شي في ص452 بس ذكر لمحة عن

pleomorphic adenoma and Warthin's tumors

ملاحظات اضافها

-treatment of malignant tumors:

1-surgery
2-neck dissection } together

3-radiation } 6 weeks after surgery

-the large the gland the benign the tumor, the small the gland the malignant the tumor

-parotid tumors:80% overall, 80% benign, 80% pleomorphic adenoma

-Treatment of pleomorphic adenoma and Warthin's tumors :

you should do surgery for 3 reasons:

1-there is chance of transformation of malignancy 5-10%

2-FNA is the least accurate in salivary gland tumor

3-for cosmetic reason

-complication of post-op

ذكر اول 6 اسطر في ص453

نقطة ذكرها في

Frey's syndrome

how to prevent it?

Facial Flap should be thick

the doctor said Frey's syndrome is very important for the exam: what is it, how to prevent, treatment

---

H & N (III):

هذا فقط الكلام اللي ذكره الدكتور بخصوص هذي الجزئية

*Types of mucosa:

1-nasopharynx ┐ group A (non-surgical)

2-orogharynx
3-hypopharynx } group B (surgical)
4-larynx
5-oral cavity

*you have to know about each type:

1-most common tumor

2-most common presentation

3-risk factors

4-treatment

*nasopharynx(group A):

1-most common tumor: squamous cell carcinoma

2-most common presentation:

a- neck mass

b- unilateral: epistaxis, hearing loss, nasal obstruction

3-risk factors: genetics, virus(EBV), types of food

4-treatment: non-surgical

early: radiation alone

late: radiation + chemotherapy

*Group B:

1- most common tumor: squamous cell carcinoma

2- most common presentation:

a-neck swelling,

b-other symptoms depend on the site:

-oral cavity: pain and ulcers

-oro/hypopharynx: dysphasia

-larynx: hoarseness

3-risk factors: smoking, alcohol, HPV

-oral cavity: trauma

-larynx + pharynx: reflux GERD or LPR(laryngeo/pharyngel reflux)

4-treatment:

early: radiation or surgery

late: chemotherapy + radiation or surgery + radiation

*we don't use chemotherapy with surgery in treatment of head and neck tumors.

*2 important messages you have to know from this lecture:

*neck mass in adult >40 it is tumor until proven otherwise

*unilateral ENT sign or symptoms it is tumor until proven otherwise

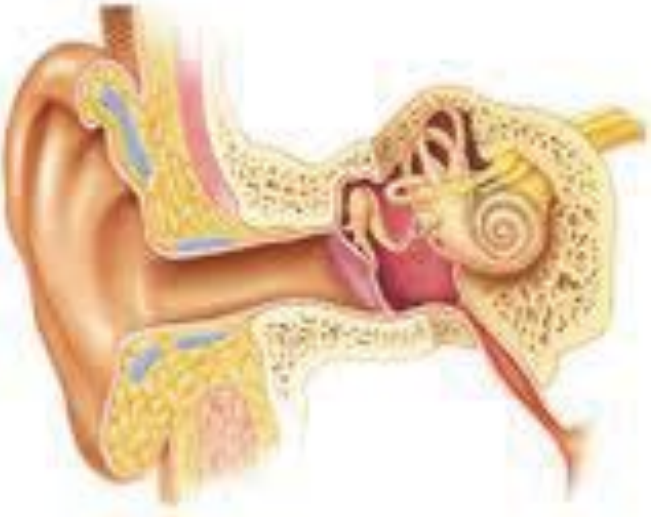
Done :)

Najla Al-Ghabban

كنت اتمنى اسويها بشكل كامل بدال ما اخليكم ترجعوا للملزمة في بعض النقاط لكن مافي وقت للأسف

واعتذر اذا فيها اي غلط.

Diseases of the Ear, Nose and Throat



1st Lecture:

Nose 1

Done by:
Hessah Al-ammar
Ahlam Al-suliman

Source (dr. Sami alharethy & bafqeeh slids ,429 team , anatomy books and records) then dr.Fatima Al-enzi slides were incorporated

Important Notes in red
Extra-notes in green/green

Objectives:

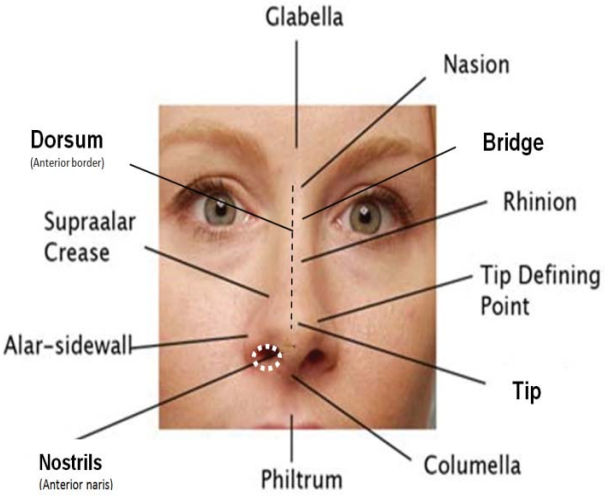
1- Anatomy and physiology of nose and paranasal sinuses(PNS)
   A. Anatomy: external nose and nasal cavity, PNS , blood and nerve supply {in brief}
   B. Function: nose and PNS
2- Congenital anomalies {in brief} (choanal atresia)

First: Anatomy and physiology:

A Nose:

Nose is divided into:
1- External nose
2-nasal cavity “fossa”

1. External nose:



* The external nose is pyramidal in shape and has:
- Anterior border called "**dorsum**" and two lateral sides.
- Root called “**nasion**” lies at the naso-facial suture.
- **Bridge** overlies the nasal bone
- **Rhinion** is osseocartilaginous junction at the lower end of the suture between the nasal bones
- Apex called the **tip** of the nose.
- Two elliptical orifices called the **anterior naris “nostrils”**, which are separated from each other by the "**columella**".

∞ *Columella is the fleshy external end of the nasal septum.*

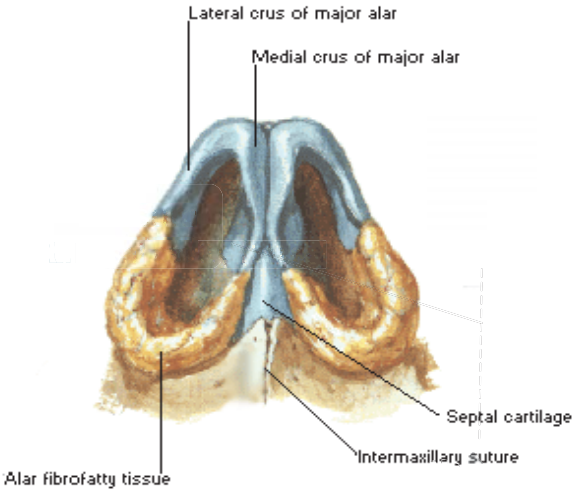
- The lateral margin of nostrils called the “**ala nasi”** is rounded and mobile.

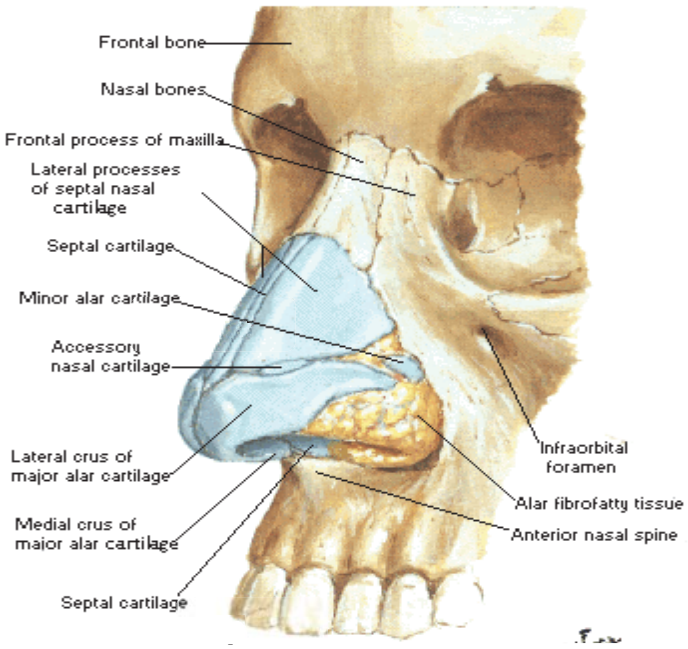
* Its shape is maintained by skeletal frame work.

1- Upper1/3 bony part:-
- Nasal bones
- Nasal process of frontal bone.
- Nasal (ascending) process of maxillary bone.

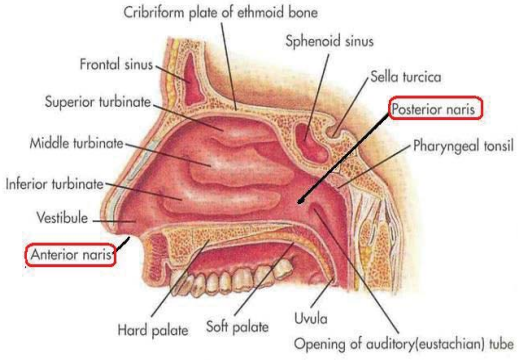
2- lower2/3 cartilagenous part:-
- Quadrangular septal cartilage.
- Upper lateral cartilage.
- Lower lateral cartilage(Major alar cartilage). it is composed of two crura lateral and medial.
- Minor alar cartilage.

∞ *A strip of skin, connective tissue and medial crura of the lower lateral cartilage form the"columella".*
∞ *The cartilages are connected with each other and with the bones by continuous perichondrium and periosteum .*
∞ *The cartilages don’t have blood vessels and they get their blood supply by diffusion from the overlying perichondrium. So any lesion that will separate the cartilage from the perichondrium (hematoma, abscess...) will lead to ischemia and necrosis of the cartilage (emergency)*



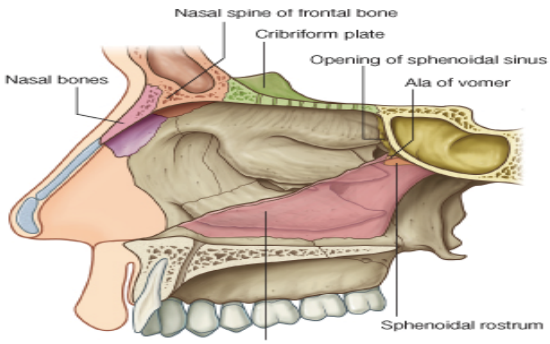


2. Nasal cavity (fossa):



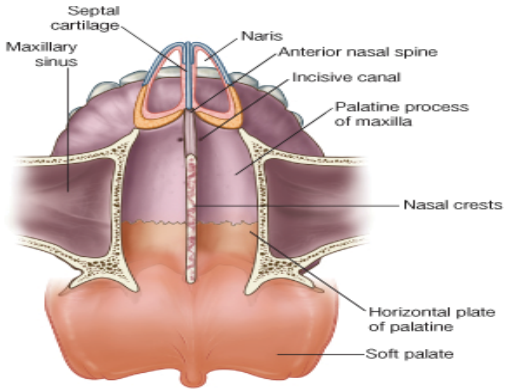
* Extends from the external (anterior) nares to the posterior nares (choanae).
* The most anterior part of the nasal cavity called nasal vestibule
* Divided into right & left halves by the nasal septum.
* Each half has a:
  - Roof
  - Lateral wall
  - Medial wall (septum)
  - Floor

ROOF:



* Narrow & formed (from behind forward) by the:

1. Body of sphenoid.
2. Cribriform plate of ethmoid bone.
3. Frontal bone.
4. Nasal bone .



FOOR:

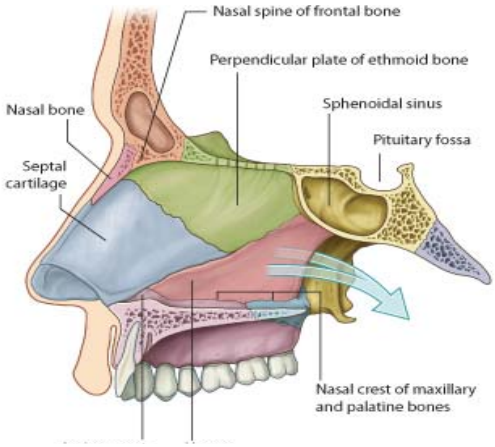
* Separates it from the oral cavity and Formed by:
* Nasal (upper)surface of the hard (bony) palate:

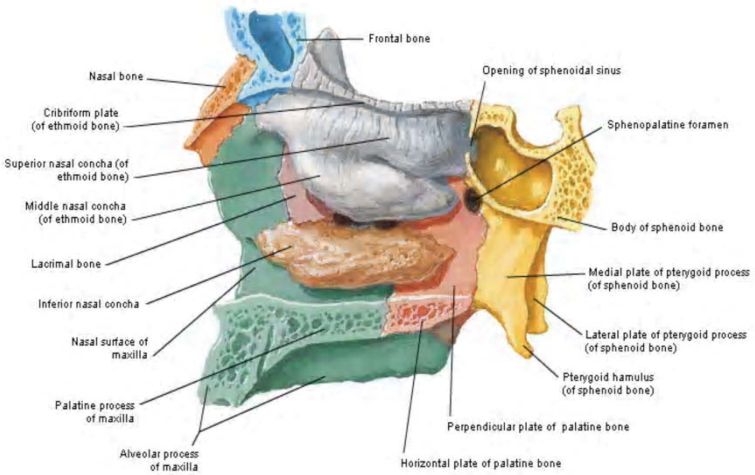
1. Palatine process of maxilla, anteriorly.
2. Horizontal plate of the palatine bone, posteriorly.

MEDIAL WALL (NASAL SEPTUM):

* Osteocartilaginous partition, covered by the mucoperiosteum.
* Formed:

1. Superiorly by the vertical (perpendicular) plate of ethmoid bone.
2. Posterior and inferior by the vomer bone.
3. Anterior and inferior by the septal (quadrangular) cartilage.

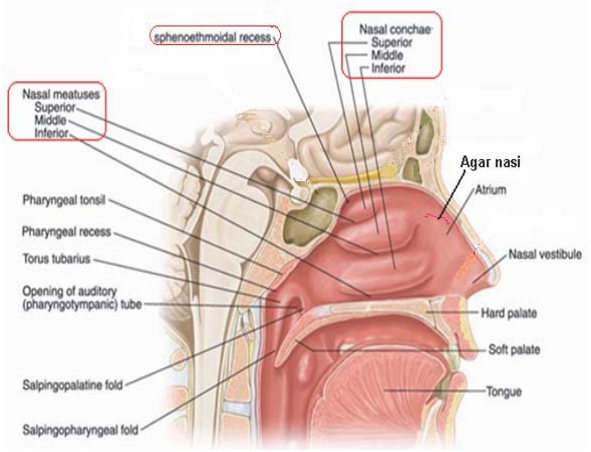




LATRAL WALL:

* formed by:

1-Nasal surface of ethmoid labyrinth, superiorly.
2-Perpendicular plate of palatine bone, posteriorly
3-Nasal surface of maxilla, lower and anterior part
4- inferior concha bone and lacrimal bone, in the middle.



Features of the lateral wall of the nose:

1-Nasal conchae (turbinates):

Three bony elevations on the lateral wall covered by mucous membrane (superior, middle and inferior conchae)

During examination we can see the inferior turbinate and sometimes the middle as well.

- A **highest** nasal concha is sometimes present
- The superior and middle conchae are parts of the **ethmoid labyrinth** whereas the inferior concha is a separate bone.

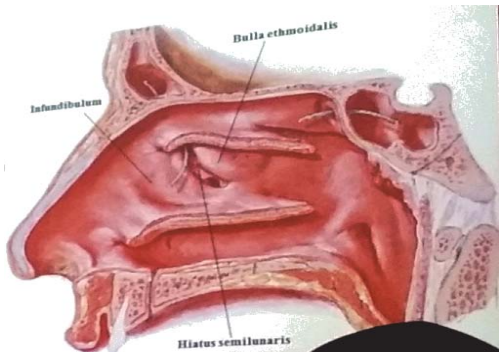
2-Nasal meatues:

- The depressions(cavities) below the conchae are called **meatuses** (superior, middle and inferior meatuses)
- The depression between the roof and superior concha is called the **sphenoethmoidal recess.**
- **Features of middle meatus:**

» **Bulla ethmoidalis:** a bulging in the lateral wall of middle meatus.

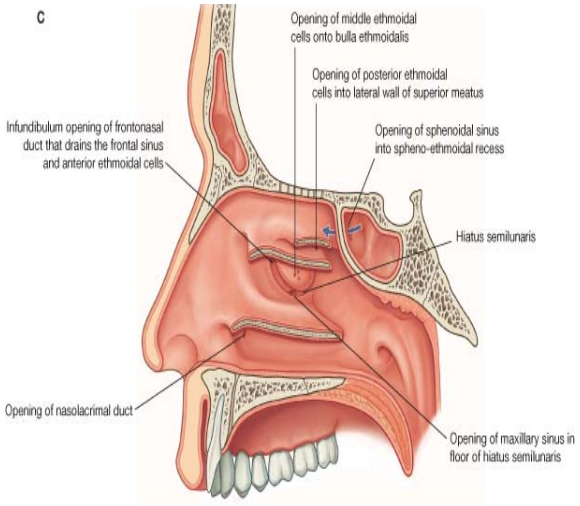
» **Hiatus semilunaris:** a crescentic groove below the bulla ethmoidalis

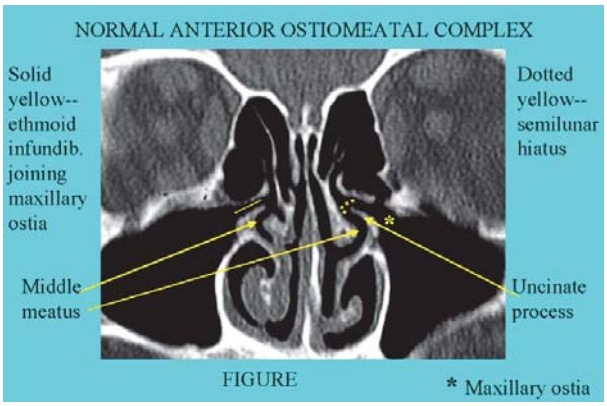
» **Infundibulum:** a recess at the anterior end of hiatus semilunaris



openings into the lateral wall of nasal cavity:

Sphenoethmoidal recess	sphenoidal sinus
Superior meatus	posterior ethmoidal sinus
Middle meatus	*anterior ethmoidal→ open into the anterior part of hiatus semilunaris *middle ethmoidal →open above the bulla ethmoid * maxillary→ opens into the hiatus semilunaris *frontal sinuses→opens into the infundibulum (through the frontonasal duct)
Inferior meatus	nasolacrimal duct→ opens into the anteriorpart





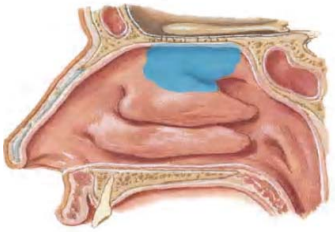
The ostiomeatal complex(OMC): is the common drainage pathway of the anterior paranasal sinuses (frontal, anterior ethmoid and maxillary sinus) into the middle meatus.

This is composed of the following structures: uncinate process(UP), ethmoid bulla(EB), middle turbinate(MT), and the spaces between these structures (ethmoidal infundibulum (EI), middle meatus (MM) and hiatus semilunaris (HS))

Lining of the Nasal Cavity:

1. Modified skin:

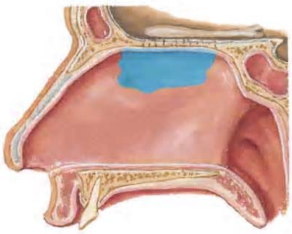
- covering the Vestibule.
- Keratinized stratified squamous epithelium containing sebaceous glands, sweat glands and short, curved hair called vibrissae.



2. olfactory mucosa

- covering the roof, upper part of the septum, upper surface of the superior concha, and the sphenoethmoidal recess.
- contains the olfactory receptor nerve cells (their axons from the olfactory nerves)

3. respiratory mucosa



-covering the rest of the cavity

-Pseudostratified ciliated columnar epithelium with goblet cells.

* *Rests on thick network of veins that **warms the air** as it flows through the cavity.*
* *Glands produce 'mucus', which:*
  - *moisten the air.*
  - *cleans the air by trapping the incoming bacteria and foreign debris.*
* *Cilia help in moving the contaminated mucus posteriorly towards the throat, where it is swallowed and digested by the stomach juices.*

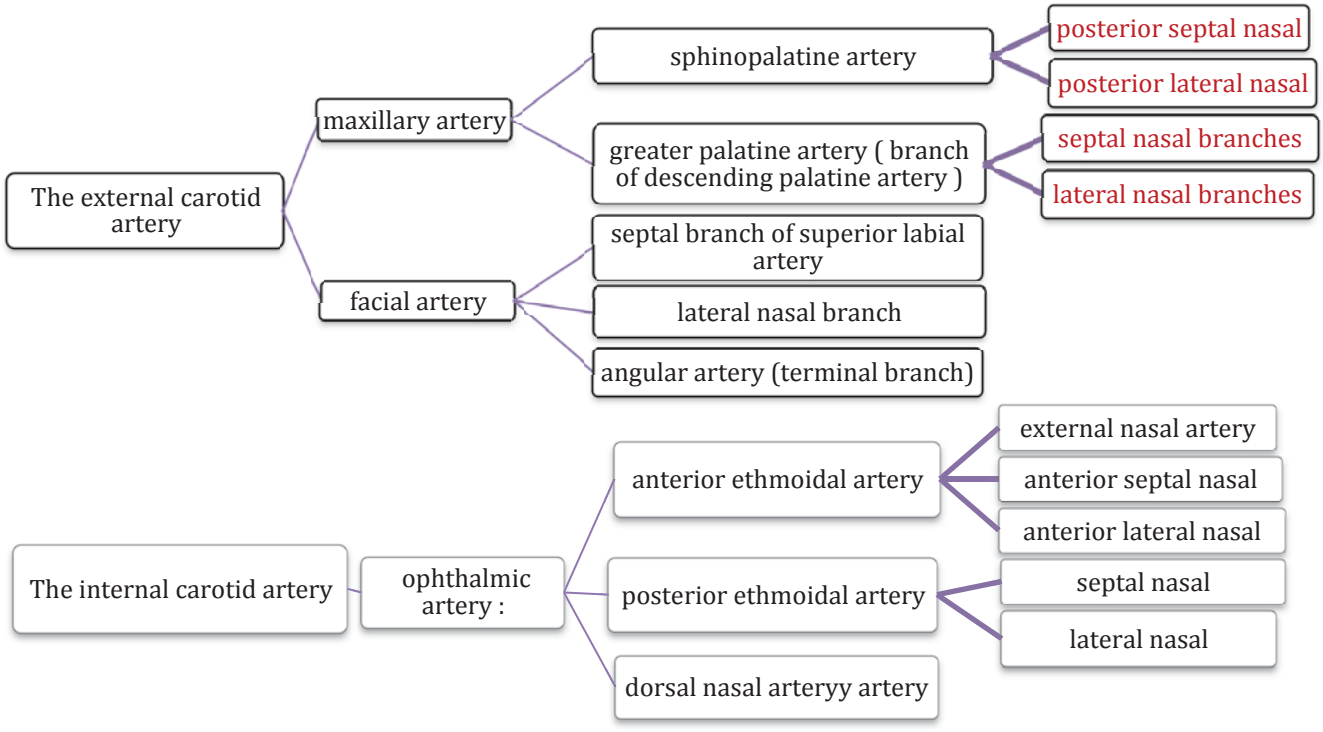
The mucosal lining of the nasal cavity is **continuous with**:

- mucosa of **nasopharynx** through the posterior nasal apertures.
- Mucosa of the **paranasal sinusses** through their openings
- **Conjunctiva**: through the nasolacrimal duct

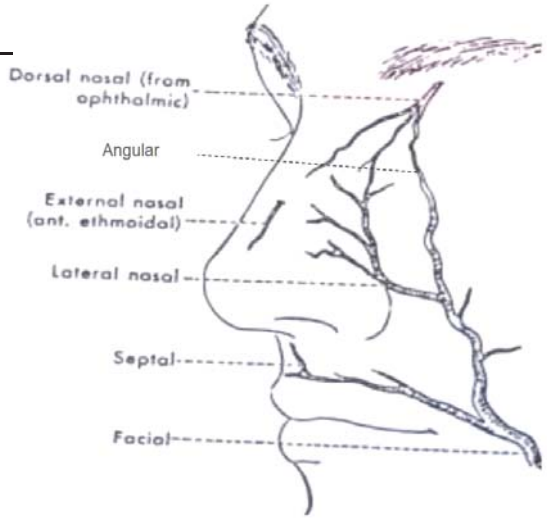
Arterial blood supply of the nose:

* **In general:**

  The nose has dual blood supply from internal and external carotid arteries.



6 [NOSE 1



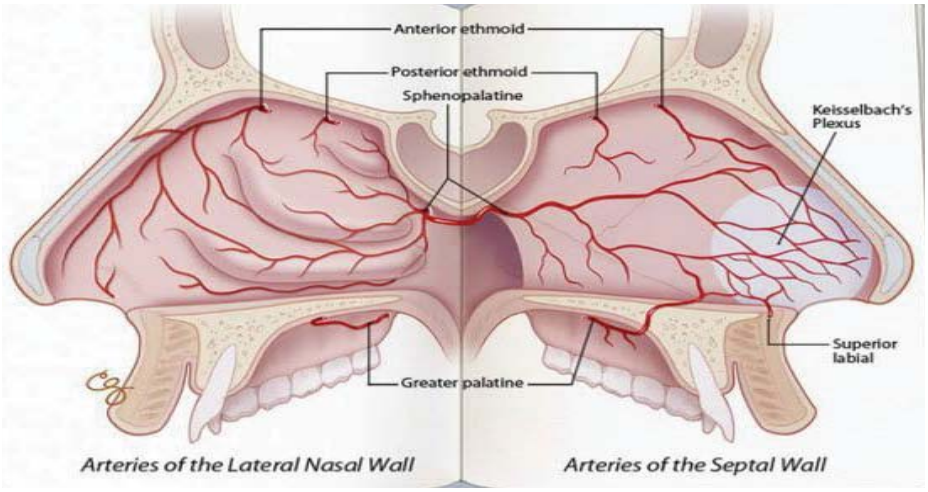
A] External nose:

1) External carotid artery
- *Facial artery :*
  - *septal branch of superior labial artery*
  - *lateral nasal branch*
  - *angular artery (terminal branch)*

2) Internal carotid artery:
- *Ophthalmic artery :*
  - *Dorsal nasal br.*
  - *anterior ethmoidal artery →external nasal artery*

B] Nasal cavity:

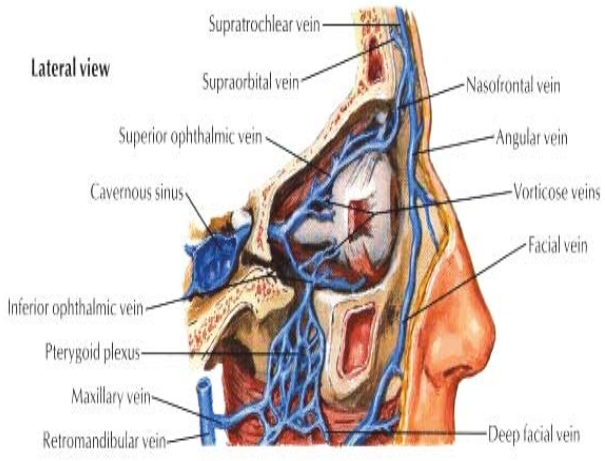
		Lateral wall	Septal wall
external carotid	maxillary	1-posterior lateral branch of Sphinopalatine artery (main supply) 2-greater palatine artery *-supply the middle turbinate and area below*	1-posterior septal branch of sphinopalatine artery (main supply) 2-greater palatine artery
	facial		1-septal branch of superior labial artery
internal carotid	ophthalmic	1-anterior lateral branch of anterior ethmoidal artery 2-lateral branch of posterior ethmoidal artery *-supply the area above the middle turbinate*	1-anterior septal branch of anterior ethmoidal artery 2-septal branch of posterior ethmoidal artery



sphinopalatine artery is behind the middle turbinate , when it is injured during surgery(FESS or turbinectomy) it will be retracted to the olfactory fossa and it is difficult to pull it

*kiesselbach plexus (little's area):
- Located on the anterior-inferior part of the cartilaginous septum.
- Formed by the anastomose of four arteries:
  Anterior ethmoidal a. – Sphenopalatine a.- Greater palatine a. - Septal branch of the superior labial a.
- the posterior ethmoidal artery don`t participate in anastomosis
- The most common site of anterior epistaxis (MCQ) *especially in young people and children due to dryness or picking of nose.*
- *In old people with hypertension the epistaxis is posterior from Woodruff's plexus, Vascular area situated over the posterior end of inferior turbinate result from anastomoses of post.nasal artery of Sphenopalatine with posterior pharyngeal artery New study side it is a venous plexus ??*

Venous drainage of the nose:



* Accompany the arteries

1. *Anterior and posterior etmoidal veins drain to →* ophthalmic vein→ cavernous sinus.
2. sphenopalatine *& greater palatine veins drain to →pterygoid plexus →*maxillary vine *→external jugular vine.*
3. *Angular vein, lateral nasal and superior labial veins drain to →* facial vein → internal jugular vein.

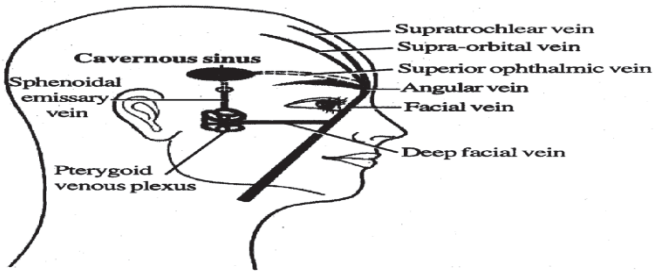
* *The pterygoid venous plexus and facial vein also communicate with the cavernous sinus.*

- *pterygoid venous plexus → emissary vein → cavernous sinus*
- *The facial vein communicates with the cavernous sinus by 2 ways:*
  1. *angular vine →nasofrontal vine →superior ophthalmic vein → cavernous sinus*
  2. *deep facial vein →pterygoid venous plexus → emissary vein → cavernous sinus*

* Dangerous area of the face(Bermuda triangle)

- *It is the area between the root of the nose and 2 angles of the mouth.*
- *The veins drian this region (mostly facial v.) are:*
  1. *connected to cavernous sinus (as we mentioned)*
  2. *valveless which facilitate retrograde flow of blood from the face to the cavernous sinus.*
- *So any infection in this area may lead to cavernous sinus thrombosis and intracranial complication.*

Dangerous area of the face



Lymphatic Drainage:

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

Nerve Supply: *(Dr. said know it in brief)*

A) External nose (skin):

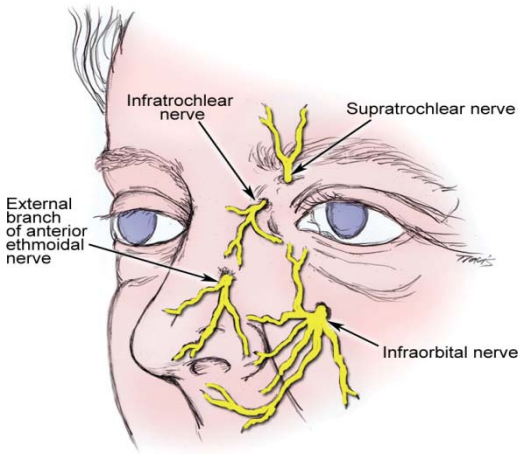
-It is innervated by the ophthalmic (V1) and maxillary (V2) divisions of the trigeminal nerve (CN V).

1-The superior aspect of the nose, including the tip,
Is supplied by the ophthalmic (V1):

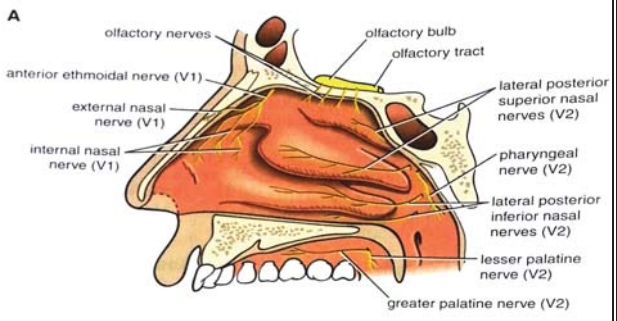
- *the infratrochlear nerve*
- *the supratrochlear nerve*
- *The external nasal branch of the anterior ethmoidal nerve.*

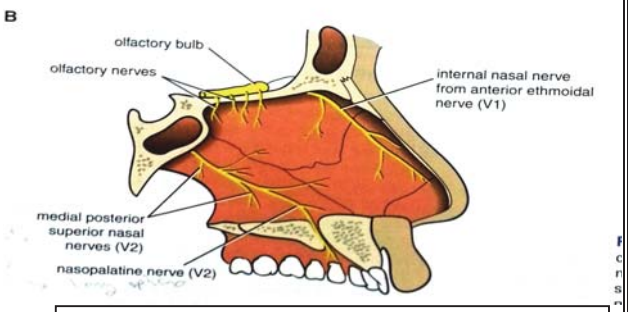
2-The inferior and lateral aspects of the nose,
Is supplied by by the maxillary (V2):

- *The infraorbital nerve.*



B) Nasal cavity:





1-alfactory sensation:

Carried by olfactory nerve from:

- The roof
- The uppermost parts of the medial and lateral walls.

2-General sensation:

Carried by ophthalmic & maxillary (via sphenopalatine ganglion) divisions of the trigeminal nerve CN V.

***lateral wall**

1- Lateral posterior superior nasal(*short sphenopalatine nerve*) branch of sphenopalatine ganglion(V2)

2- Lateral posterior inferior nasal branch of greater palatine nerve (V2)

3- Lateral internal nasal branch of the anterior ethmoidal nerve (V1).

***nasal septum**

1- Nasopalatine (*long sphenopalatine nerve*) nerve branch of sphenopalatine ganglion(V2)

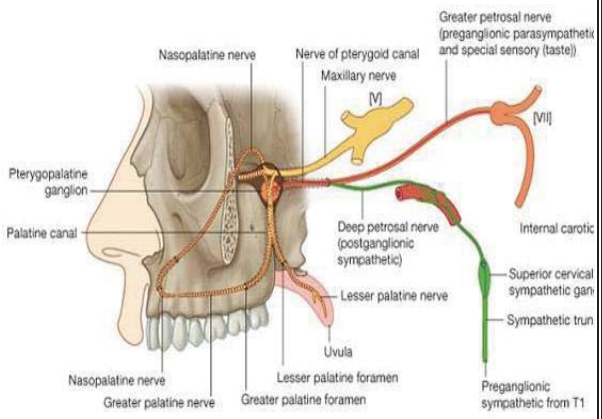
2- medial posterior superior branch of sphenopalatine ganglion(V2)

3- Medial internal nasal branch of the anterior ethmoidal nerve (V1).

**Sphenopalatine ganglion is important in the nose*
**Some diseases like vasomotor-rhinitis treated by cauterization of this ganglion (not used now)*

3-Autonomic fibers:

- Sensory branches of the sphenopalatine ganglion supplying the nasal mucosa carry postganglionic secretomotor fibers from the sphenopalatine ganglion to the nasal glands.
- Control vascular tone and secretion of the nasal mucous glands.
  *Sympathetic : vasoconstriction
  *Parasympathetic : vasodilatation and increase nasal secretion



-Pathway:

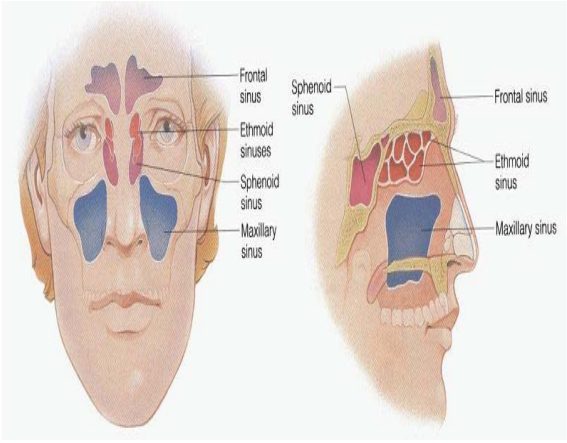
- Postganglionic sympathetic fibers pass from the superior cervical ganglion →deep petrosal nerve.
- Preganglionic parasympathetic fibers pass via the sensory root of the facial nerve → the greater petrosal branch.
- The deep petrosal and greater petrosal nerves merge to form the vidian nerve(nerve of the pterygoid Canal) → pterygopalatine ganglion where the Parasympathetic fibers synapse with the postganglionic secreto-motor fibers.
- pterygopalatine ganglion gives terminal branches carrying the post gangelionic sympathetic and parasymoathatic fibers to their targets in the nasal cavity (blood vessels and nasal glands)

Function of the nose:

1-Nasal respiration
2- Smell
3-protective function of the nose (purification) :
Vibrissae – cilia (beating back to throat)
Lysozomes – reflex sneezing - warming (inspired air 37°)
Moistening:
- Transudation of fluid through the epithelium
-secretion from the mucosal glands.

The amount of postnasal mucus drip (through cilia) :0.5 L/day (like a small bottle of water)usually we do not feel it except if it is large or thick

B Para nasal sinuses (PNS):



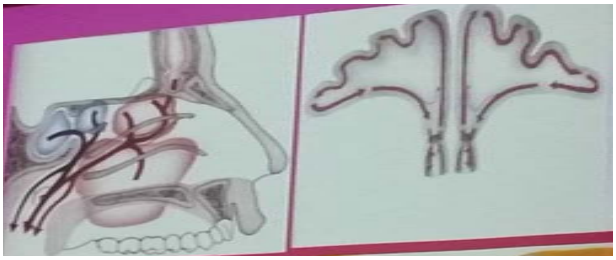
- PNS are air-filled cavities into the skull bone which are connected to the nasal cavity by a small opening (ostium)
- There are four paired of paranasal sinus :
1. Frontal →*located in the forehead region*
2. Maxillary →*in the cheek area*
3. Ethmoidal →*between the eyes*
4. Sphenoid→*deep in the center of the skull*

They are divided as:
Anterior group → frontal, anterior ethmoidal and maxillary sinuses
Posterior group→ posterior ethmoidal and sphenoid sinuses

Lining of the paranasal sinuses:

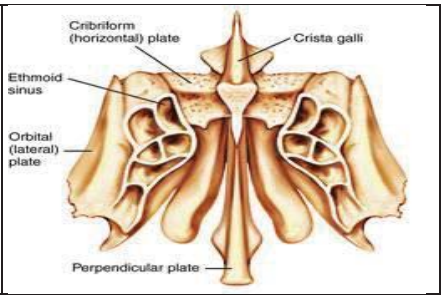
- lined by Pseudostratified ciliated columnar epithelium with goblet cells (respiratory epithelium) which is continuous with the nasal epithelium.

- The mucosa secrets mucus which traps bacteria, and then it is naturally extruded through sinus ostia to be expectorated or swallowed.
- Each person produces about one quart or one liter of mucus per day.
- The drainage of the maxillary and frontal sinuses follows a circular pattern through the natural ostia.

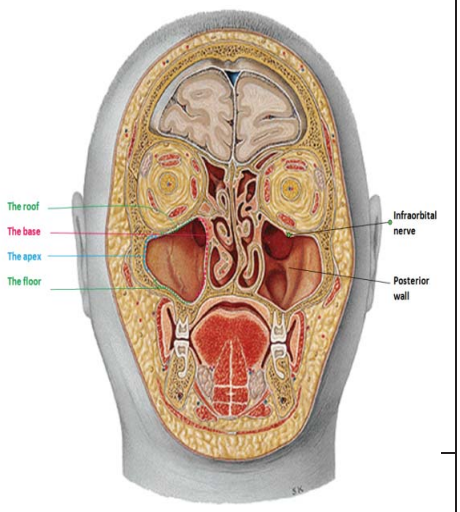


1) The Ethmoid sinus:

Presence at birth	*Present at birth, small, adult size at age 12.*	
Embryology	*Appears as evagination of the lateral nasal wall around the third month of fetal gestation.*	
Anatomical characteristic	**Consist of a number (approximately 7-15) cavities* **lies within the lateral masses (labyrinth) of the ethmoidal bones* **very thin walls contributing to easy injury (during FESS) or spread of infections and tumors to adjacent structures (orbit, anterior cranial fossa),* **separated by the ground (base) lamella of middle turbinate. into anterior and posterior ethmoidal sinus*	
Ostia opening	**anterior and middle sinuses →the middle meatus* ** posterior sinus →superior meatus*	
relations	*-the orbit (lamina papyracea)* *-the anterior cranial fossa (cribriform plate)*	
Blood supply	1-external carotid (through the sphenopalatine) 2-internal carotid (through the anterior and posterior ethmoidal)	
Nerve supply	*Both V2 and V3 (in the book it is different?) **Both V1 and V2 innervate this region:* *1-Anterior and posterior ethmoidal nerves a branch of nasocilliary (V1)* *2- short sphenopalatine nerve a branch of pterygopalatin ganglion (V2)* **Parasympathetic and Sympathetic innervation is via the vidian nerve.*	

Other information	The ethmoid bone Consist of the vertical and horizontal palates. - The vertical plate is divided into two portions, the perpendicular plate of the ethmoid and the crista galli. - The horizontal plate is known laterally as the fovea ethmoidalis(part of frontal bone) and medially as the cribriform plate. Medial wall is the lamina papyracea. -the comments site of sinusitis	

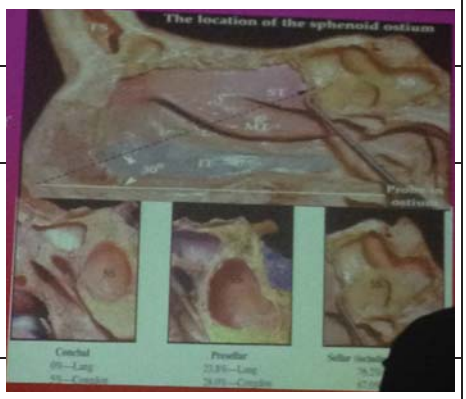
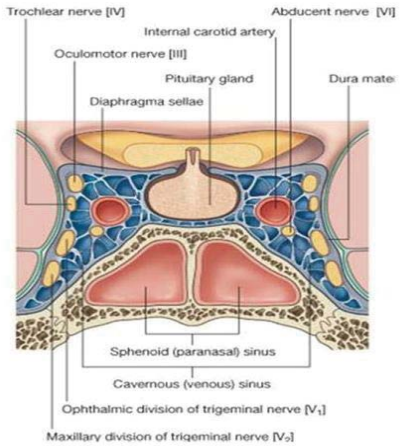
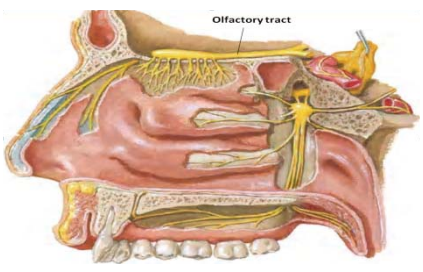
2) 2) The Maxillary sinuses:

Presence at birth	*Present at birth and continues to grow until the 3rd decade.*	
embryology	*First sinus to develop*	
Anatomical features	*- It is also called antrum of Highmore* *- lies in the body of the maxilla.* -The largest sinus. - Pyramidal in shaped With: *the apex lies laterally in zygomatic portion of the maxilla. *The base (Medial wall) between the sinus and the Nasal fossa. *the floor near nasal floor in children, 1cm below nasal floor In adult. *the roof is formed by the floor of the orbit *the anterior and posterior wall.	
Ostia opening	- natural ostia located anteriorly in the middle meatus. - Accessory ostia are usually more posterior and are a sign of chronic disease.	
relations	*superiorly (roof): *1-Orbit →any disease process involving the maxilla is likely to affect the orbit through this wall* 2-The infra-orbital nerve: runs along roof, and is often dehiscent (uncovered with bone). At risk during antral procedures. *Inferiorly (floor): 1-Upper teeth: the floor is often thin and dehiscent over tooth roots. Usually the 2nd premolar and 1st molar are related. *-Dental infections may involve the maxillary sinus.* *Medially (base): *nasal cavity* *Nasolacrimal duct* *posteriorly: *maxillary artery and nerve: through pterygopalatine fossa.*	
Blood supply	*Divisions of the maxillary artery (external carotid): Superior alveolar and Infraorbital arteries	
Nerve supply	**from V2 (via Superior alveolar and infraorbital nerves).* **Parasympathetic and Sympathetic innervation is via the vidian nerve.*	

3) Frontal Sinus:

Presence at birth	Rarely present at birth; usually not visible until age 2.	
Embryology	begins as evagination of the anterior nasal capsule around the fourth month of development	
Anatomical characteristic	- Great variability in size -congenitally absent in 5%. *-extensively pneumatized when fully developed*	
Ostia opening	*Drain into frontal recess in the middle meatus near the upper portion of the infundibulum .*	
relations	*Orbit and anterior cranial fossa.*	
Blood supply	*from supraorbital and supratrochlear arteries of the ophthalmic artery (internal carotied)*	
Nerve supply	*innervations from nerves of the same name.(V1)* **Parasympathetic and Sympathetic innervation is via the vidian nerve.*	
Other information	*Like the maxillary sinus have a circular muco-ciliary clearance.*	

4) Sphenoid sinus:

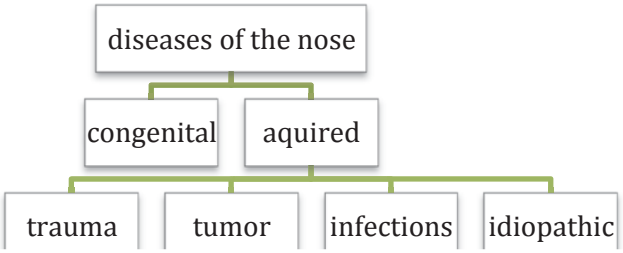
Presence at birth	- Rarely present at birth usually seen around age 4 -development at puberty.	
Embryology	Began as outpunching of the superior nasal vault around the fourth month of gestation	
Anatomical characteristic	*-It occupies the body of the sphenoid bone.*	
Ostia opening	- drain into the sphenoethmoidal recess , ostia of variable size	
relations	*superiorly: *1-Pituitary gland (superior-posterior): The pituitary gland may be approached surgically through the sinus.* 2-optic nerve/chiasm (superior-anterior) *3-frontal lobe of brain* *4-olfactory tract* *laterally: 1-Cavernous sinus containing the: III, IV, V(ophthalmic V1 and maxillary V2 divisions) and VI cranial nerves and internal carotid artery -the carotid artery is dehiscent in 50% of specimens *posteriorly: 1-the pons *and basilar artery* *-Very dangerous because it is near important structures*	 
Blood supply	*1-internal carotid arteries via the posterior ethmoidal arteries (roof)* *2-external carotid arteries via the sphenpalatine (floor)*	

Nerve supply	*Innervations from V1 and V2:* *1-posterior ethmoidal nerves a branch of The nasociliary nerve (V1) supplies the roof.* *2-The branches of the sphenopalatine nerve(V2)supply the floor.* **Parasympathetic and Sympathetic innervation is via the vidian nerve.*	
Other information	*- Patient with sphenoid sinus disease comes with headache.*	

Functions of paranasal sinus:

1. Provide mucous to upper airway. Vehicle for trapping viruses,bacteria, foregin material for removal
2. Humidifing and warming inspired air
3. Lubrication
4. Give charactaristics voice reonance.
5. Reduce skull weight
6. regulation of intranasal pressure
7. Increasing surface area for olfaction.
8. absorbing shock in trauma.
9. Contribute to facial growth

Second: Disease of the nose:



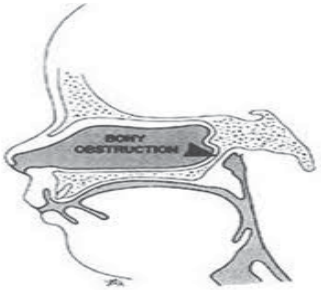
***congenital anomalies of the nose:**

A) Congenital nasal atresia:

1) Atresia and stenosis of anterior nares:

* caused by non canalization of an epithelial blug between the median and lateral nasal process
* *if it is bilateral → incompatible with life and died in utero (stillbirth)*
* treated by excision

2) Atresia of posterior nares (congenital choanal atresia) **(IMP):**



Definition:

**A congenital condition where the back of the nasal passage (choana or posterior nares) is blocked.*

**Females are more commonly affected than males.*

**rare congenital anomalies*

Causes:

Failure of canalization of the primitive bucconasal membrane

Types:

- *Unilateral or bilateral*
- Bony (most common), membranous or mixed

Degree:

1- complete unilateral : is the most common usually asymptomatic Diagnosed by history of rhinorrhea from one side.

2- complete bilateral

3- incomplete unilateral

4- incomplete bilateral

- Unilateral choanal atresia:

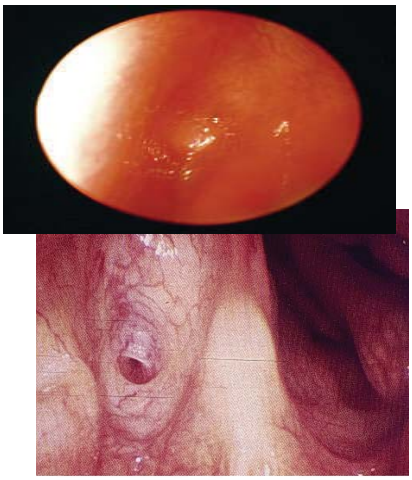
- *Usually diagnosed late in life*
- Presents by unilateral nasal obstruction and unilateral excessive watery nasal discharge
- *Treatment is by elective surgical repair*

- Bilateral choanal atresia:

- *Isolated anomaly in 60-70%*
- *May be linked to* CHARGE *association*
  - » **C**oloboma : is a hole in one of the structures of the eye, such as the iris, retina, choroid or optic disc.
  - » **H**eart disease
  - » **A**tresia
  - » **R**etarded growth
  - » **G**enital hypoplasia
  - » **E**ar deformity
- Usually presents at birth by attacks of cyclic cyanosis and respiratory obstruction (Asphyxia)→ blue reviled by crying
- Nasal discharge
- it is an emergency because new born are obligatory nasal breather and don`t know how to breath from mouth(MCQ)

Diagnosis:

- *Clinical examination: "mirror test" by placing a mirror in front of nose Watch for the fog →the fog indicating air flow.*
- Total absence of the nasal air flow.
- Inability to pass a catheter into the nasopharynx (suction at birth)
- Endoscopy (Post-rhinoscopy)
- Radiographs



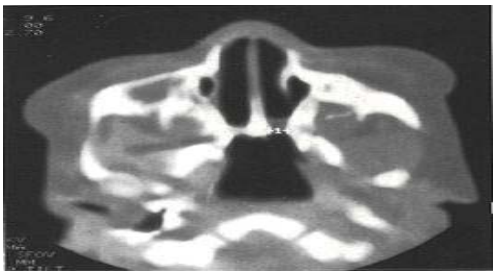
Management:

- Emergency

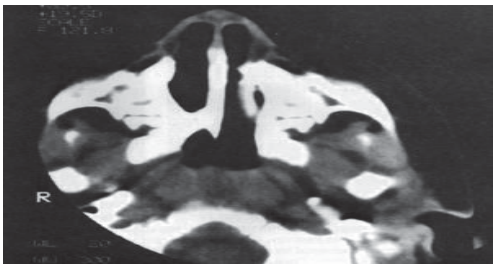
-Immediate airway support with oral airway, McGovern nipple, or intubation

- Definitive surgery

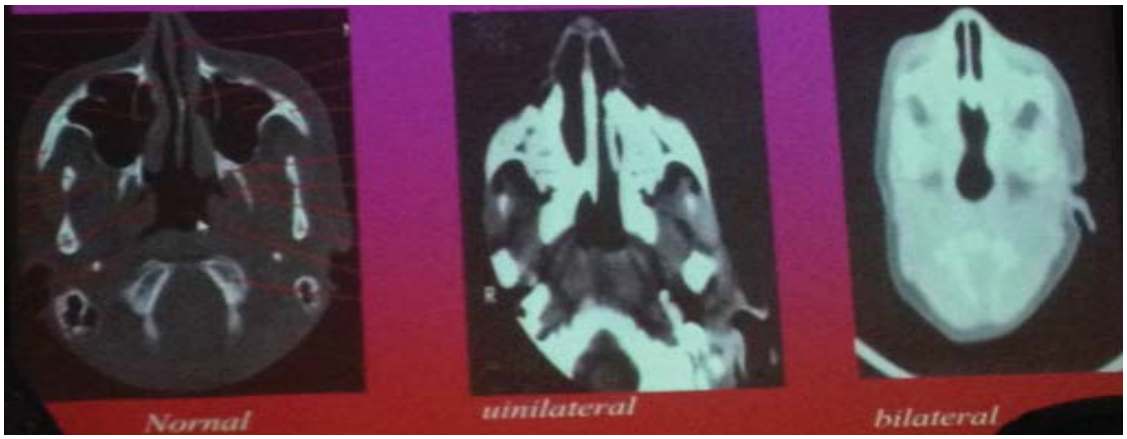
-transnasal perforation or transpalatal excision *with indwelling tubes to prevent* reclosure.

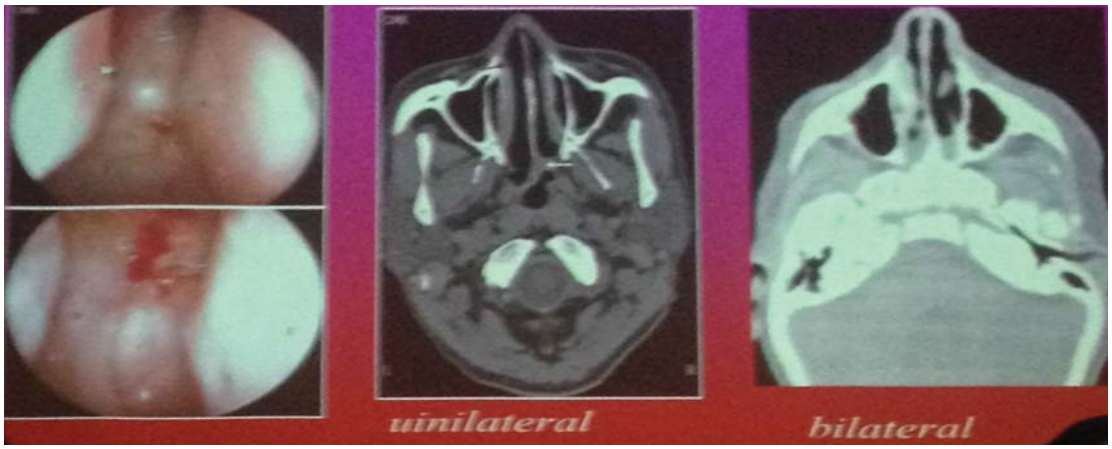


CT – bilateral choanal atresia



CT – unilateral choanal atresia



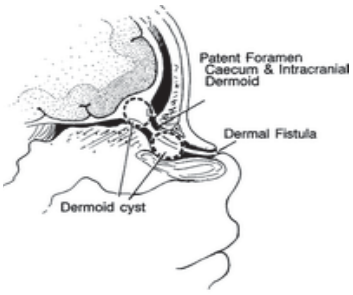


If a less than 6 months baby present with snoring and breathing from the mouth it's not adenoid because adenoid is lymph and the immunity until 6 months is from the mother so you need to rule out congenital cause.

B) Congenital nasal masses:

- *Include nasal dermoids, nasal gliomas, and encephaloceles.*
- *Rare congenital anomalies.*
- *May present as extranasal mass or as intranasal mass.*
- *Most present in infants and children.*
- *Any nasal mass in a child should be evaluated for a congenital midline mass (imp).*
- *Have potential for connection to the central nervous system.*
- *Biopsy of a lesion with an intracranial connection can lead to meningitis or cerebrospinal fluid leak. So biopsy of extra or intranasal mass in a child is contraindicated before imaging.*
- *The treatment is surgical excision.*

1- **Nasal dermoid (Sinus or cyst):**



Definition:

Nasal dermoids are epithelial-lined cavities (cysts) or sinus *tracts consist of both ectodermal and mesodermal elements, including hair follicles, sebaceous glands, and sweat glands.*

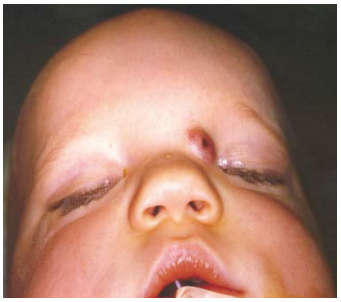
*They constitute the most common congenital nasal anomaly.

*The nasal lesions account for 3% of dermoids in the head and neck and 1% of all body dermoids.

**Site: 1- intra nasally 2- extranasally →anywhere in the midline from the columella base to the glabella with the most common site being the lower third of the nasal bridge.*

Clinical features:

Symptoms:



1. *Nasal dermoid present as masses (intra or extranasal), sinus tracts (pit) with opening on the skin, or as a combination of the two.*
2. *They may present with intermittent discharge of sebaceous material or pus from the opening.*
3. *hair protruding from the site is pathognomonic*
4. *They can cause broadening of the nasal dorsum*

Sings

1- Firm 2- uncompressible 3- slowly growing mass 4- Do not transilluminate.

5- No expansion of these lesions with crying, Valsalva maneuver, or bilateral compression of the jugular veins (-ve Furstenberg test).

Diagnose

- *CT scan: is useful for visualizing bony defects of the skull base.*
- *MR imaging: it is superior for visualizing soft tissues and diagnosing intracranial extension and is thus the preferred imaging study.*
- *Biopsy is contraindicated.*

Treatment:

Surgical excision

2- Nasal glioma:

Definition:

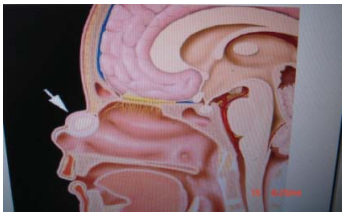
Glioma is uncapsulated collections of glial cells situated outside the CNS.

**15% of gliomas connect with the dura.*

**Site:*

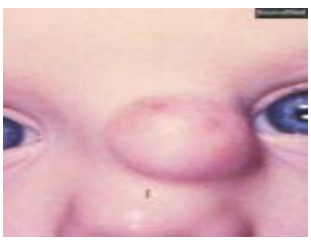
1-intranasally →usually arise from the lateral wall unlike nasal encephalocele which arise medially

2-Extranasally →Unlike dermoids, they do not necessarily occur in midline usually located at glabella level or nasomaxillary suture.



Clinical features:

Symptoms:



1- present as mass

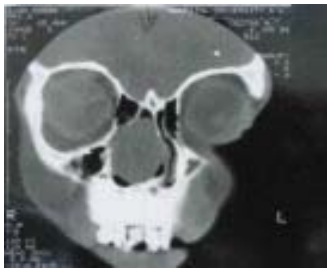
- *extranasal → Red or bluish lump*
- intranasal → mimic polyp ,unilateral nasal mass, unilateral nasal obstruction, snoring, epistaxis or cerebrospinal rhinorrhea

Sings

1- Firm 2- uncompressible 3- nonpulsatile mass 4- Do not transilluminate.

5- No expansion of these lesions with *crying, Valsalva maneuver,* or bilateral compression of the jugular veins
(-ve Furstenberg test).

6-May have telangiectasias of the overlying skin.



Diagnose

Same as dermoid

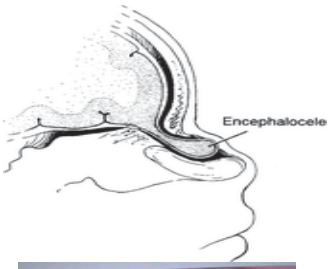
Treatment:

Surgical excision of the mass

3-Nasal Encephalocele:

Definition:

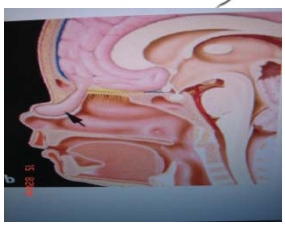
herniation of neural tissue (meninges and/or brain) through defects in the skull *with Subarachnoid connection*



*They may contain meninges (meningiocele), brain matter and meninges (encephalomeningiocele) or they may communicate with a ventricle (encephalomeningocystocele).

*20% of all encephaloceles occur in the cranium, 15% nasal

** Always have intracranial connection.*



Clinical features:

Symptoms:

1- *Bluish*, masses over the glabella orinside the nose.

2- Enlarge with crying

3- May have associated CSF leak

Sings

1- Soft 2-compressible *3- pulsatile masses 4-Do transilluminate.*

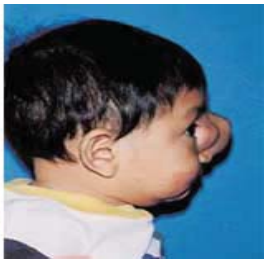
5- expansion of these lesions with crying, Valsalva maneuver, or bilateral compression of the jugular veins (+veFurstenberg test).because of the ↑ICP

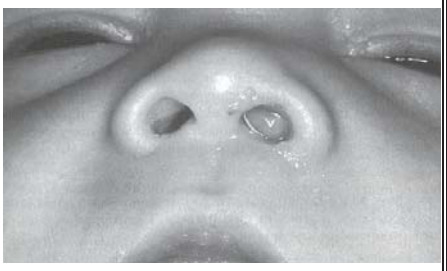
Diagnose

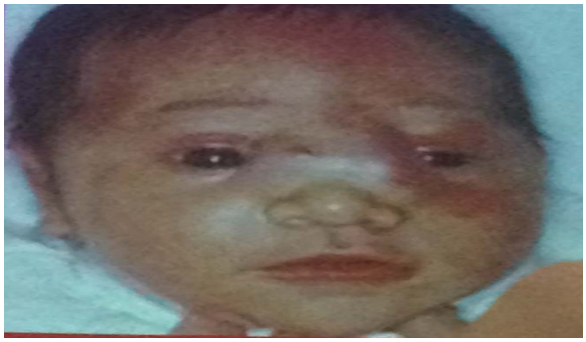
Same as dermoid

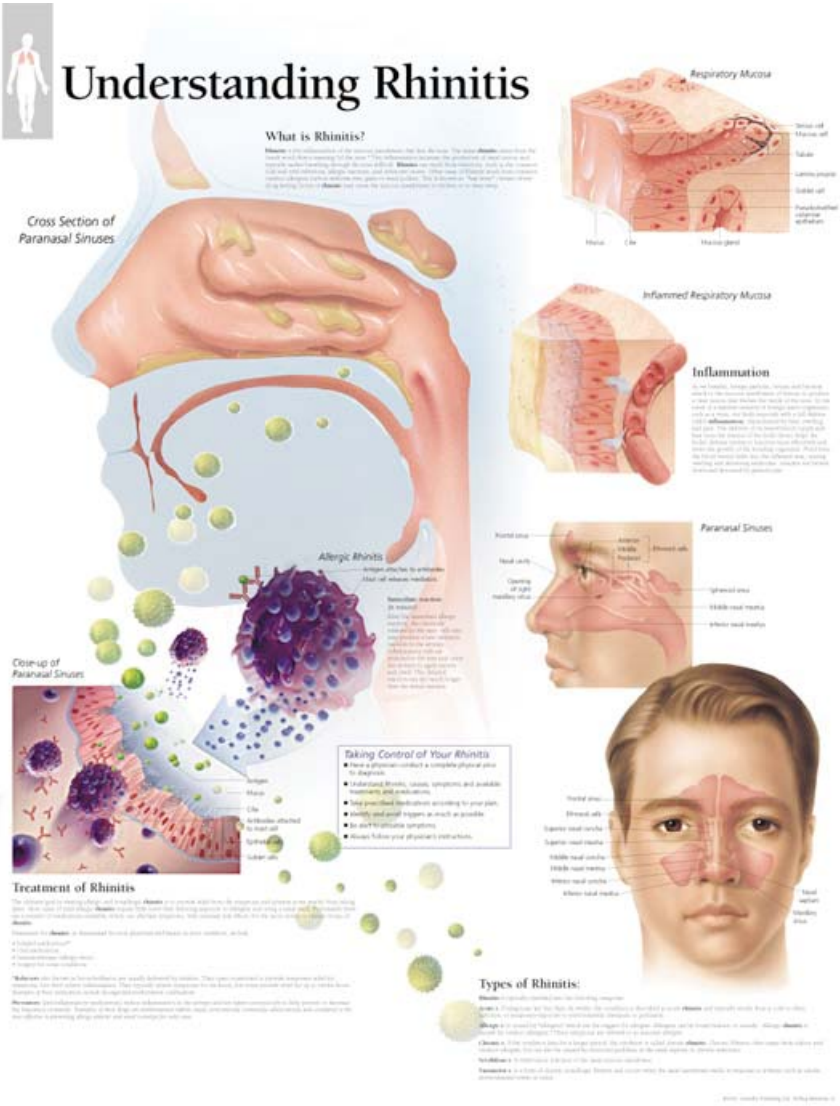
Treatment:

Surgical excision









Nose 2 (Rhinitis)

429 ENT Team (F2)

Sources: - Dr. Sameer Bafaqeeh's lecture. - ENT Team Notes (Nose 2).
- LECTURE NOTES ON Diseases of the Ear, Nose and Throat by P.D. BULL, Ninth Edition

Objective:

- Acute and chronic rhinitis
- Allergic and non-allergic rhinitis
- Vestibular and furunculosis
- Nasal polyps (allergic and antrochoanal) etc
- Radiology illustration (e.g. CT scan)

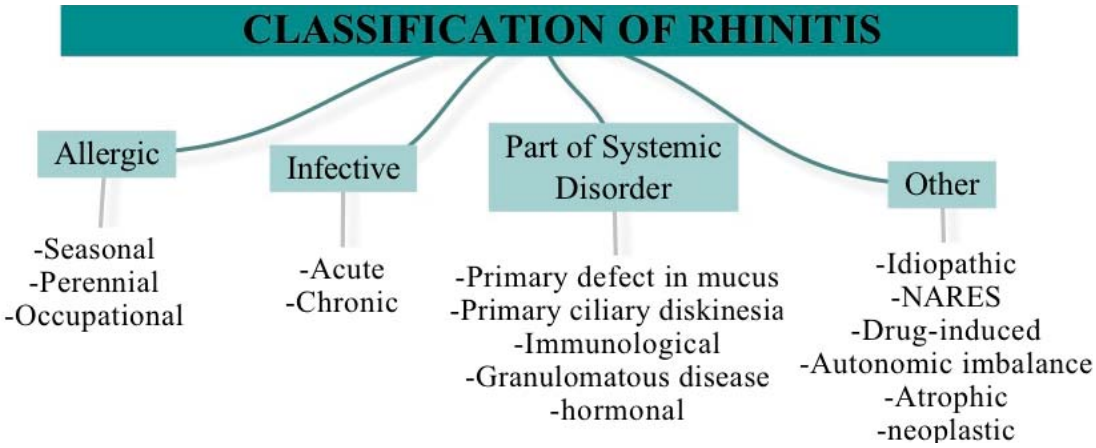
Done by: Nourah AlSyefi

Introduction:

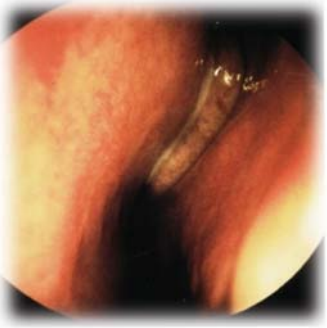
- Rhinosinusitis: Disease of the nose that has the similar findings in the sinuses because they are all lined by the same columnar ciliated epithelium.
- All sinusitis causes must be coming from the nose
- As well as causing typical symptoms, rhinitis can significantly impair quality of life.
- While seasonal allergic rhinitis (hay fever) is confined to the pollen season, allergic rhinitis is often perennial.
- Allergic factors can be identified easily & specific avoidance or therapy prescribed.
- AR in the last years due to pollution >> increased allergen >> sensitizations.
- AR & asthma usually coexist and proper treatment of the nose will lead to pulmonary improvement.
- Any obstruction around the osteomeatal complex will lead to sinusitis and rhinosinusitis

Incidence:

It affects 20-30% of the population (1 person in 6)



1- Acute Rhinitis (common cold):



Clinical stages :

1. Dry prodromal stage (few hours)
2. The Catarrhal stage (few days)
3. Mucous stage (3-5 days)
4. Resolution stage (5 days- 1 week)
5. Secondary bacterial infection

prodromal stage	Catarrhal stage	Mucous stage	Resolution stage	Secondary bacterial infection
Generalized Symptoms: •Chills, cold &heat . •Headache, fatigue. •Loss of Appetite . •Subfebrile temperature. •(Itching, burning, dryness, irritation) of the nose and throat.	•Watery profuse Secretions. •Nasal Obstruction. •Loss of smell. •Lacrimation . •Rhinolalia clausa (hyponasality of the voice). •Constitutional symptoms worsen.	•All generalized symptoms become improved. •Thick secretions. •Local symptoms regress.	•usually in one week.	•Greenish yellow secretions. •Resolve slowly.
Examination: •Nothing but mucosa pale & dry	Examination: •The mucosa deep red +swollen around the turbinate . •Secret profusely .			Most common organism: •Haemophilus Infl. •Streptococc Pneumoniae.
✣ If the initial stage was catarrhal: •It will be due to: **Influenza**,Parainfluenza , adenovirus ,Rheovirus, Coronavirus , Enterovirus, myxovirus& Respiratory syncytial virus. •Symptoms will be as before in addition to: Entire respiratory tract, G.I.T,Meninges,Pericardium , Kidneys & Muscles.				

Pathogenesis:

- Usually due to rhinovirus
- more than100 types that are related to Picorna group
- I.P: 1-3 days
- Spread by :Droplet
- Initiated by cooling the body

Diagnosis:

Usually difficult to dingoes in the beginning and must be differentiated from AR & vasomotor rhinitis

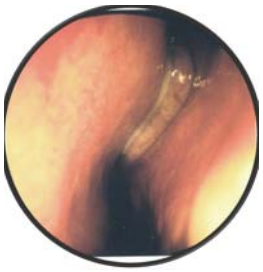
D.D :

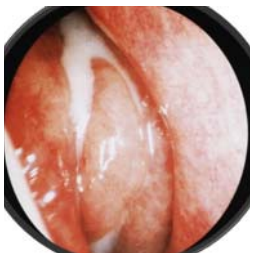
- Acute Exanthema
- Allergic
- VMR (vasomotor rhinitis)
- Cog. Syphilis
- N. Diphtheria

Treatment:

Symptomatic treatment	Prophylaxis
•Nasal decongestant. •Oral decongestant. •Antibiotics(in 2dry bacterial infection). •Steam inhalations. •Infrared lamps. •Analgesics. •Bed rest.	•Sauna baths •Therapeutic regimens •Hydrotherapy •Sports •Vitamin C •Careful hygiene •Adenoidectomy(in children) •Immunization [not available in Coryza virus] •Vaccines [available for Influenza]

Infective rhinosinsitis:

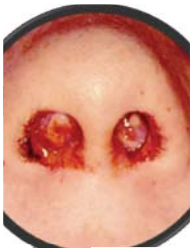




Same as acute rhinitis:

- Common Cold
- Large number of viruses
- Droplet spread
- Resolves or 2nd bacterial:
- Haemophilus influenza& Streptococcus pneumonia
- Hypersecreting & hypertrophic
- Muco-purulent >> rhinosinusitis

2- Chronic Rhinitis:



Due to chronic irritation Or Inflammation>>Hypertrophy of Nasal Mucosa>>Hyperemia edema(True tissue hypertrophy)

Symptoms:

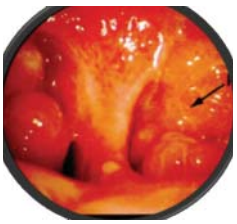
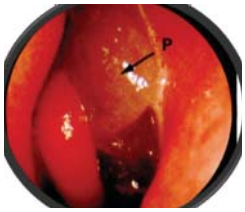
- **Nasal obstruction**
- **Tough, Stringy, Colorless secretions (rarely purulent)**
- Post nasal catarrh
- Rhinolalia clausa(hyponasality)
- **2nd dacryocystitis** (an inflammation of the nasolacrimal sac)
- **2nd pharyngitis**
- Fatigue ,sleeplessness
- Unsteady or woozy feeling
- Headache
- Feeling of pressure in the head
- Psychological & physical well-being loss
- **Epiphora(tearing)**

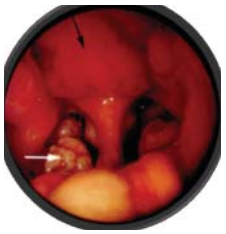
Pathogenesis (Causes):

- Recurrent inflammation
- Sinusitis
- masses in nasopharynx (Enlarged adenoid,tumor)
- VMR & Side effects of drugs
- Pregnancy, menstruation, menopause
- Endocrine disturbances
- Heart &circulatory diseases
- Infective allergy "late-type allergy"
- Tobacco, smoke, dust, chemicals, acquired toxins, temperature extremes , humidity

Diagnosis (imp):

From Hx:	On examination:
Long-standing disease	Dark red &bluish-violet swelling turbinate
History of toxins	Narrowed or obstructed nasal cavity
NOT improved with nasal decongested	Mucosa:Granular then nodular surface demonstrating Micro-polyps then nasal polypi
	Mulberry-like masses



Differential diagnosis:

- Sinusitis
- Adenoidal hypertrophy
- Tumors(biopsy)
- Foreign bodies
- wegener's granuloma
- Specific infections
- allergy

Treatment

Conservative	Surgical (the only way to improve Sx)
Treatment of etiological agents	Reduction of the inferior turbinate by sclerosing agents, cryoprobe, or the laser. -Electrocoagulation>> multiple, localized scars in N.M(turbinate) -Cryosurgery>>partial obliteration -CO2 or argon laser>>mucosal scars (evaporation or coagulation)
Drug overuse controlled	
Endocrinologic investigation	
Environment & occupation	Turbinectomy or mucotomy
Symptomatic treatment by decongestant nose drops (Not in long term)	**Turbinoplasty** (the best approach)

The classification "seasonal" and "perennial" allergic rhinitis has been changed to"intermittent" and "persistent" allergic rhinitis

3- Allergic rhinitis :

- All rhinitis has allergic factors except 20-30% are not and called non allergic rhinitis or vasomotor rhinitis.
- Due to inhalation of the allergen.
- The disease is hereditary
- AR Antigens are usually wind-borne [eg ; grass & tree pollens],HDM ,fungi,dog &cat dander
- Types: Seasonal, Perennial, Occupational.

a- Seasonal Allergic rhinitis :

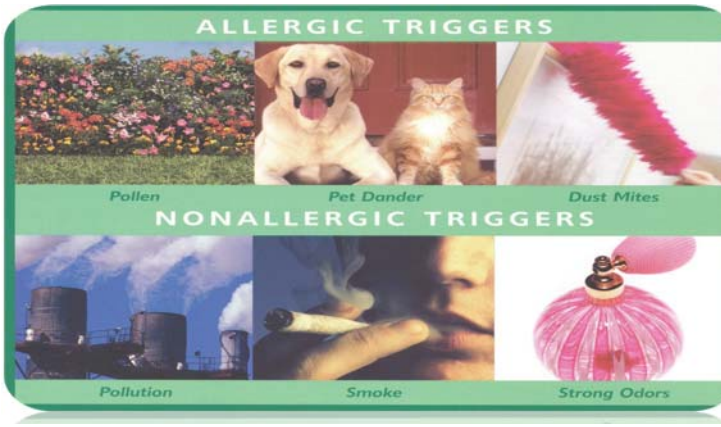
•Start ate Early Spring by[tree pollen]ترابthen in Midsummer by [grass pollen] and end in Autumn by [molds]لقاح

KEY POINTS :
•Seasonal allergic rhinitis occurs only when pollens in the air (hay fever).
•Perennial rhinitis occurs all year round & can be a myriad of substances, although house dust mite is high on the list.

b- Perennial Allergic rhinitis :

Causes

•The **house dust mite** is the most common cause.
•A mountain hut in Sweden. (go there to escape AR!)
•Fungi, animal hair, house dust& mites.
•Houseplants: [primulas & rose].
•Food: [fish, strawberries, nuts, eggs ,milk, &flour].

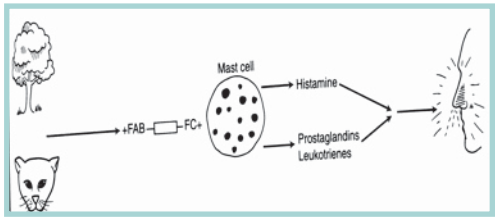


c- Occupational allergies:

[bakers, hairdressers &painters]
(e.g.; latex, powders, paint vapors)

The Classic Reaction

•Type1 IgE mediated reaction, produced in Plasma. cells and regulated in T-Lymphocytes.
•IgE has a crystallized fraction that bind to MC (mast cell) then release protein(Fab).
•Fab +Antigen lead to triggering of mast cell degranulation and release of material like (Histamine, Leukotrine, Prostaglandin).
•These substances causes :Mucosal edema &nasal secretion.



Note:

•Nonorganic substances can induces inflammatory reaction and nonspecific irritants like: smoke and dust >> Vasoactive substances (the reaction is not IgE mediated but it's IgG).
•Physical factors can affect the mast cell like: Temperature change& alcohol.

Diagnosis of AR:

•Detailed medical history.
•ENT examination.
•Other tests as appropriate: allergy tests, endoscopy, nasal smear, nasal swab, radiology, nasal airway assessment, olfaction, blood tests.

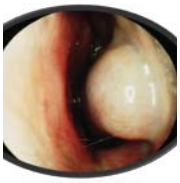
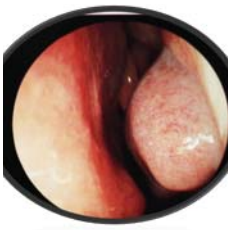
AR Symptoms:

•"SNEEZERS AND RUNNERS": Itchy nose, sneezing, watery rhinorrhoea, nasal congestion (variable), diurnal rhythm (worse during day), often associated conjunctivitis.

•"BLOCKERS":Little or no sneezing, thick catarrh (with post nasal drip), no itch, constant symptoms - possibly worse at night.

Examination:

•allergic crease
•Nasal mucosa : livid & pale
•The turbinates : swollen
•Clear secretion : +++

Investigation:

•**Skin prick testing**	•Nasal Cytology	•Intracutaneous tests
•**RAST (radioallergosorbent test)**	•IgE	•Mucociliary clearance
•Typical history	•Rhinomanometry	•Rhinoscopy tests
•Patch tests	•Nasal provocation test	•P.N.S. CT

D.D of AR:

•**Vasomotor rhinitis**	•**Common cold**	•Cerebrospinal rhinorrhoea
•Polyps.	•Granulomas e.g. Wegener's Granulomatosis.	•Mechanical factors: deviated septum, hypertrophic turbinates, foreign bodies.
•Coryza		•Tumors.

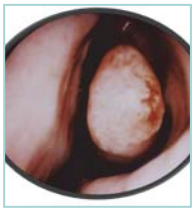
Treatment:

Causal Treatment	Symptomatic Treatment (imp)
•Immunotherapy or Desensitization. •Allergen Avoidance. •Local or systemic inhibitions of H substance.	•Antihistamines. •Topical Steroids & Cromoglycate. •Systemic Steroids. •Nasal Decongestant. •Anticholinergic. •Antibiotics. (after culture & sensitivity) •Polypectomy & turbinoplasty.

Prognosis & Complications:

Prognosis :
- Good prognosis
- Regresses with time
- Some times →bronchial asthma

Complications:
- Nasal sinuses
- L.R.T.
- **Nasal or sinuses polyps**



Indications for surgery:
- Anatomical abnormalities
- Excessive mucosal swelling
- Presence of irreversibly diseased tissue

Surgical procedure:
- Polyp removal
- Turbinate surgery
- Removal of adenoids
- Correction of septal deformities
- Sinus drainage

4- Vasomotor Rhinitis

Symptoms : as Perennial Allergic Rhinitis
Has a paroxysmal course

Examination:
- Livid, pale nasal mucosa.
- Profuse watery secretion
- Nasal turbinates swollen

Pathogenesis
- Neurovascular disorder of blood supply of nasal cavity but No specific antibodies and No specific reflex hypersensitivity.
- Caused by various influences e.g:Change of temperature or humidity

Alcohol , dust, smoke, mechanical irritation, stress, anxiety neurosis, endocrine disorders, rhinitis of pregnancy.
- Drugs: (e.g., antihypertensive agents as reserpine or beta-blockers, oral contraceptives)
- Drug abuse: (imidazoline & catechol derivatives, clomethiazole, etc.)

Diagnosis
By exclusion (Typical history, Negative allergen tests, No elevated IgE in the secretion)

Differential diagnosis
(Allergic Rhinitis, Foreign body in the Nose, Common Cold)

Treatment: (imp)

Conservative:	Surgical :
•Elimination of irritant factors. •Antihistamines. •Nasal decongestant drops. •Oral decongestant drugs. •Steroids (e.g., beclomethasone). •Metabolic & endocrine systems. •Sedatives. •Imidazoline preparations (Potential for habituation).	•Turbinate surgery Electrocautery,cryosurgery, laser. (**Turbinoplasty is the best**) •Correction of anatomical deformity. •Conchotomy. •Parasympathetic nasal fibers divisions (Pterygoid canal n., vidian n., greater petrosal n.) M.C.F.

Prognosis

It maybe Uncertain or Pt. may suddenly improves or become resistant to treatment

5- Rhinitis Sicca Anterior:

Pathogenesis:

Anterior part of the septum usually exposed to hot, dry weather. so trauma or dryness >> irritation to this part >> Crusts formation and attempt to remove it >> Nasal bleeding>> septal perforation.

On examination:

- Nasal septum is dry
- Mucosal surface is: Raw, roughened, & granular.
- Crustation >>ulceration>> Septal perforation

Differential Diagnosis

- Chemical injury (Chromium workers)
- Trauma
- Leprosy
- Iatrogenic septal perforation
- Lupus
- syphilis

Treatment:

- **Nasal ointments**
- Septal perforation closure

- Main causes of septal perforation in KSA are: surgery then nose picking.
- But in foreign country is : sniffing drugs.

6- Pregnancy Rhinitis:

- Nasal swelling & obstruction
- Start at the 2nd half of pregnancy
- Resolve after delivery

7- Rhinitis Medicamentosa (imp):

Reversible or irreversible damaged mucosa caused by topically or systemically applied drugs:-

- Hyperplastic Rhinitis
- Dryness of the nasal mucosa
- Toxic Rhinopathy(Vasoactive substances)

“Acute intoxication in infants & small children”

8- Atrophic Rhinitis& Ozena (imp):

Atrophic rhinitis+ foul smell = Ozena

Types:

- Primary: rare
- Secondary:common, and it's due to massive cutting of the turbinate in surgery,trauma or Occupational exposure to:Glass, wood, asbestos, etc.
- Mainly in women, at puberty, Flattened nose& broad Face (due to fullness of crustation)

Examination:

- Greenish–yellow or brownish-black crusts
- Atrophic mucosa & dry: Subepithelial layer fibrosis
- Anosmia & social problem
- Wide nasal cavity
- Fetid secretion &crusts (Ozena)
- Nasal obstruction

Pathogenesis

- Unknown cause but is multifactorial
- Common in orientals than in whites than in blacks
- Respiratory epith. >> sq. metaplasia
- Destroyed mucociliary cleaning system
- Bacterial proteolysis decomposed the thick & gluey secretions

Differential Diagnosis

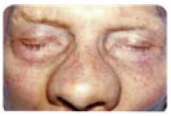
- Atrophic rhinitis with fetor (ozena)
- Purulent Rhinitis & Sinusitis
- Gumma due to stage III Syphilis
- glanders
- Tumors of the Nose & Sinuses
- Rhinolith & foreign body
- Nasal diphtheria &Nasal Tuberculosis

Treatment:

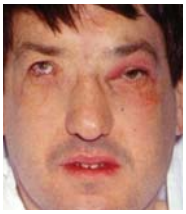
Conservative(better than surgery)	Operative
•Nasal douching. •Alkaline nasal lotion. •Greasy ointments. •Oily nasal drops, emulsions , or ointments. •Steam inhalations. •Osmotic Powders :Dextrose.	•Bolstering of the Nasal Mucosa(Cartilage or Bone chips). •Median Displacement of the lateral nasal wall by internal rotation of the mobilized lateral nasal wall.

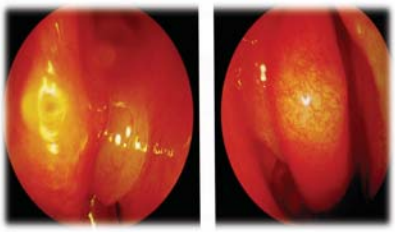
9- Nasal polyps:

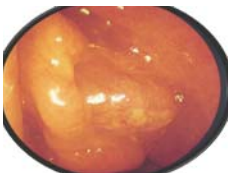
•Benign pedicle or sessile pale gray sacks of mucosa.
•Usually ethmoid sinus is affected .
•Etiology : infection, allergic, 90% eosin.
•Any age is possible but uncommon in children(exclude encephalocele) .
•Male affected more than female but if the pt. is having asthma the ratio is equal.
•Mostly bilateral(if unilateral think of inverted papilloma, encephaolce or carcinoma).
•100 to 1000 times histamine / serum (histamine is 100 times more than serum indicating mast cell degranulation in sinuses). "**causes of mast cell degranulation extreme temp. ,drugs, &complement factors."**
•If the eosinophil >90% it suggests association with AR.
•50% nasal polyps will develop asthma (proper treatment of the polyp will improve asthma).
•80% have ASA or Sempter's triad (polyp, aspirin sensitivity and asthma).
•Nasal polyposis is chronic & recurrent disease.
•Patient have Miserable time and they respond well to Salicylate-free diet.

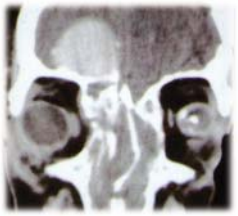
Clinical Feature:	**examination:**	**Investigation**
•Nasal obstruction, decrease sense of smell, sneezing, rhinorrhea and Postnasal drip: clear, yellow or green (depend on degree of eosinophilia or infection). •Hyponasal voice [permanent cold]. •No pain unless secondary infection. •No bleeding no discharge but if there is bleeding or serosanguinous discharge think of carcinoma	• Bilateral pale, glistening gray sacks of polyp hanging from the sinuses. • Frog face (in a massive polyp)  •differentiate between polyp and turbinate by pushing it with a probe if it's movable and painless >> polyp if not >> turbinate	•CT scan (because FESS is usually indicated) •Skin tests [H. dust or pollen] •In Child do : •CT scanning to exclude (Encephalocele) •Sweat test to exclude (Cystic Fibrosis)

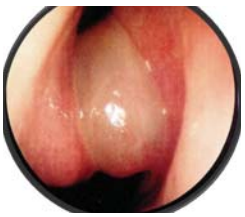
Treatment:

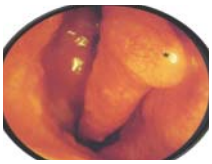


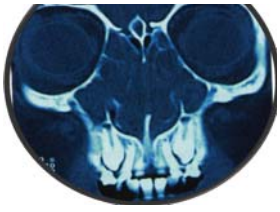
Medical treatment	Surgical Treatment
Topical steroids : good in 50% First line of management 1- month course & review Head down position [drops] Aqueous spray :daily basis Patient must be aware of unwanted effects of steroid sprays	Nasal polypectomy(Partial or total ethmoidectomy) Postoperatively Topical steroids :- -Minimal time is 3 months 
Systemic steroids :Short reducing dose (it may lead to Avascular necrosis of the head of femur)	

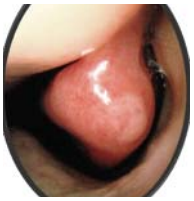




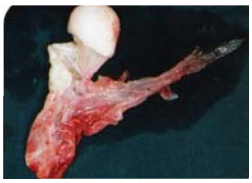




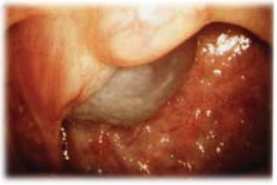




Antrochoanal polyp:

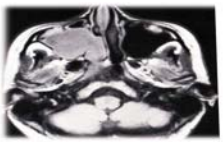


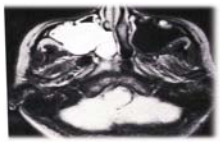
- Long pedicle unilateral solitary benign polypoidal lesions
- Unknown etiology
- Dumbell shape
- Arises in the intramural maxillary sinus cysts in the post. aspect of the sinus
- Can recur after treatment.
- Unilateral nasal blockage



Surgical treatment:

- Endoscopic nasal removal (by FESS)
- Caldwell-Luc procedure
- Snare Simple polypectomy :20%recurrence

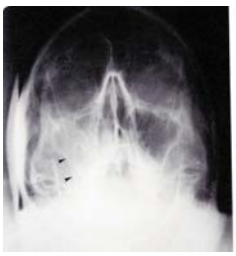


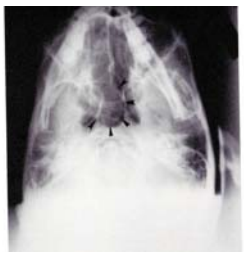


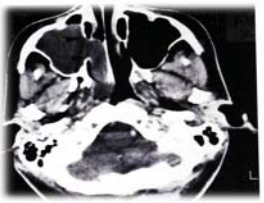
D.D :

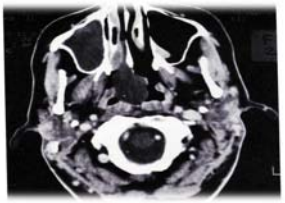
Inf. Turbinate enlargement, polypoid rhinosinusitis, juvenile N angiofibroma, mucus R cyst, mucocele, benign or malignant nasopharyngeal tumors

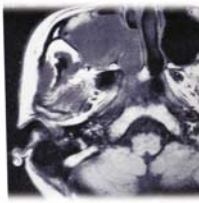
Preop. Components identification by it's characteristic radiographic appearance





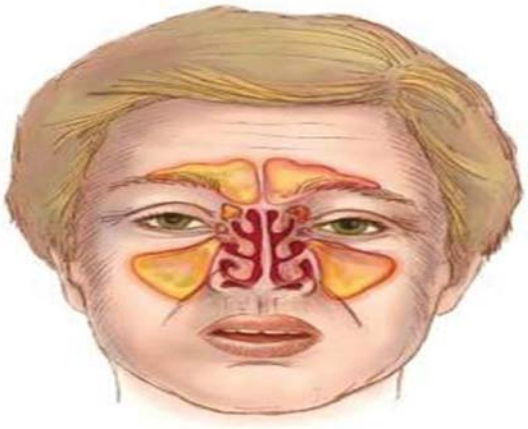






Nasal vestibulitis	Furunculosis
•infection of the skin of the nasal vestibule. •It affects both children and adults •caused by pyogenic staphylococci •The site becomes sore and fissured and crusting will occur. •Diagnosis: a swab for culture and sensitivity. •Treatment, which needs to be prolonged, consists of topical antibiotic/antiseptic ointment and systemic flucloxacillin.	•Abscess in a hair follicle is rare but must be treated seriously as it can lead to cavernous sinus thrombosis. •The tip of the nose becomes red, tense and painful. •Systemic antibiotics should be given without delay, preferably by in- jection. + Drainage may be necessary but should be deferred until the patient has had adequate antibiotic treatment for 24 h. In recurrent cases, diabetes must be excluded.

Sinusitis and Its Complications



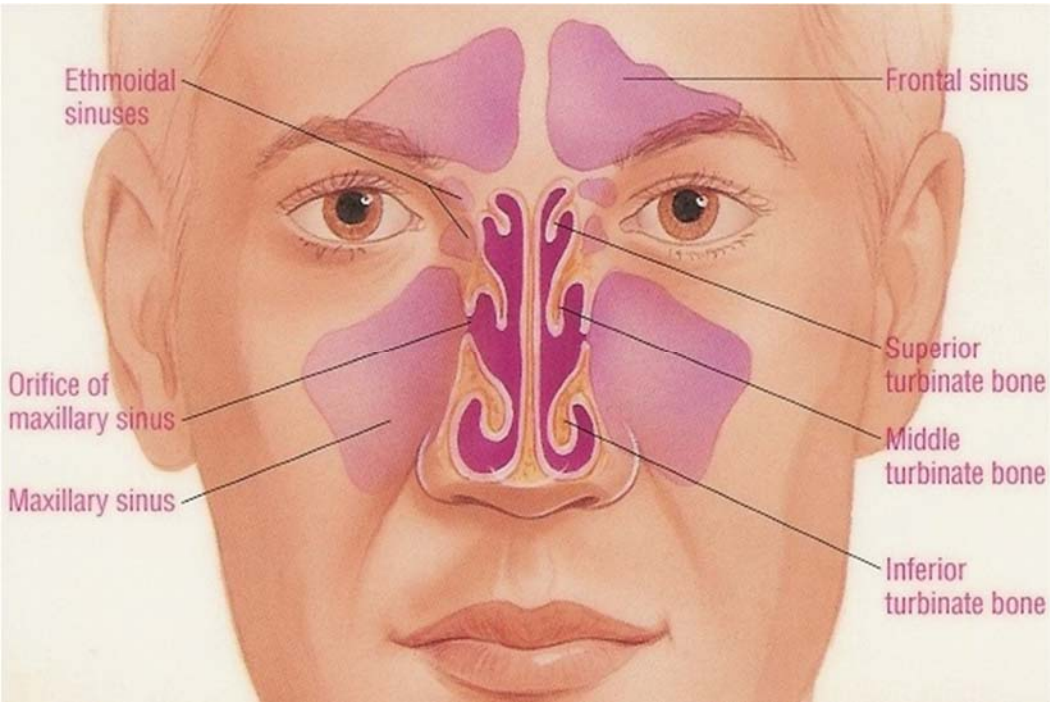
Sami Alharethy
Consultant ORL-H&NS
facialplastic surgeon

- This is dr. Sami's slides . I only listened to the record and put notes on them.
- the objectives are covered but I don't know whether it's enough to study from or not.
- The blue notes were added from the record.
- The purple ones dr. sami didn't mention during the lecture.

Good Luck

Maram Alhamad
Asma Almadhi

Sinusitis and Its Complications



Anatomy

• Maxillary. • Anterior ethmoid. • Frontal.	Open in	Middle meatus (under the middle turbinate).
• Posterior ethmoid. • Sphenoid.	Open in	Superior meatus (under the superior turbinate).
• Lacrimal ducts.	Open in	Inferior meatus (under the superior turbinate).

Development

- Maxillary and ethmoid sinuses develops during 3rd & 4th gestaı onal month and grow in size until late adolescence (so, at birth only maxillary and ethmoid sinuses are present).
- Sinusitis in general more common in children and they usually arise from maxillary and ethmoid sinuses .
- Sphenoid sinus presents by 2 years of age
- Frontal sinus develops during 5 and 6 yrs.

Definitions

- ***Rhinosinusitis*** - broadly defined as an inflammation and/or infection involving the nasal mucosa and at least one of the adjacent sinus cavities
- ***Acute rhinosinusitis (AS)*** – the persistence and worsening of upper respiratory symptoms for greater than a 7-day course but lasts less than 4 weeks.
- ***Subacute rhinosinusitis (SAS)*** - is defined as nasal symptoms lasting 4 weeks to 12 weeks.
- ***Chronic Rhinosinusitis (CRS***) – persistence mucosal inflammation for > 12 consecuı ve weeks despite medical therapy **or** occurrence of more than four episodes of symptoms a year with persistent radiographic changes.

Rhinosinusitis

Acute

- Less than 3 months
- ***S. Pneumo, H. Flu, M. Catarrhalis***
- More severe symptoms
- General stems from acute viral infection

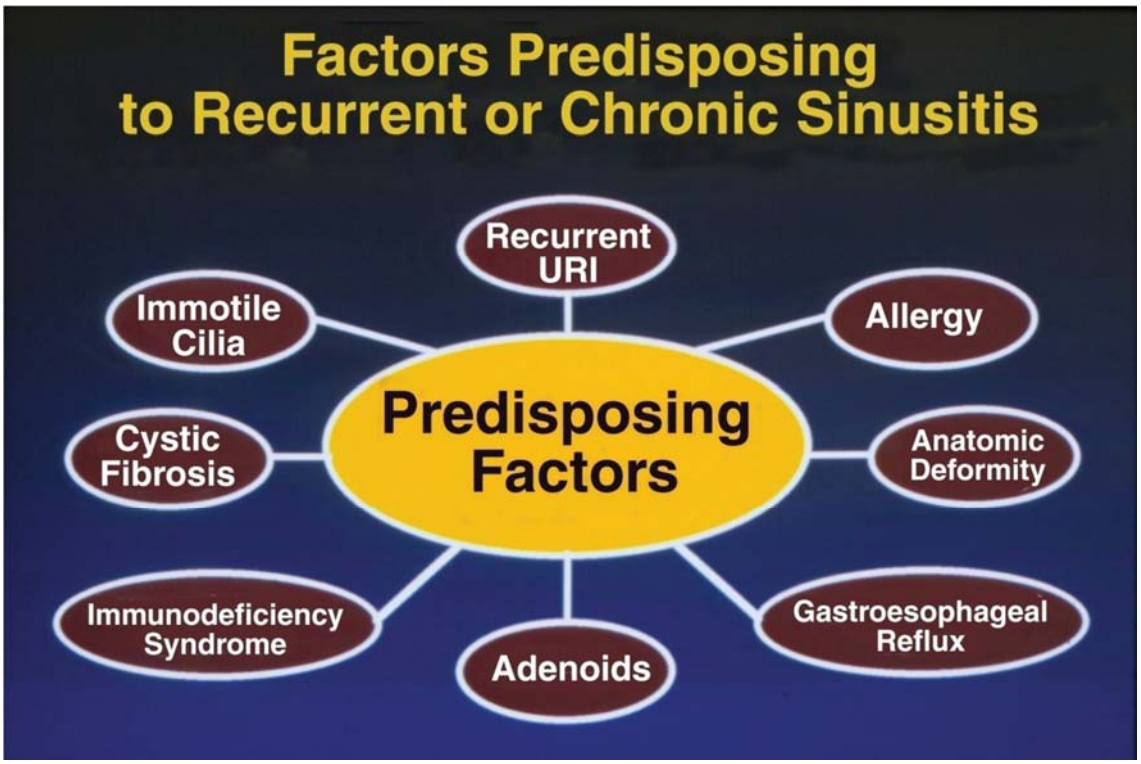
Chronic

- Greater than 3 months
- *S. Aureus,Anerobes , α-hemolytic strep, m. catarrhalis .*
- Milder symptoms
- Additional symptoms present:
  - **chronic cough (continues cough compared to bronchial asthma patient who has cough at night) , bronchitis, fatigue, malaise, and depression**.

Physiology

THREE KEY ELEMENTS

- patency of the ostia
- function of the ciliary apparatus
- quality of secretions



Predisposing factors

- Immotile cilia → the secreı ons will stay in the sinuses.
- Cystic fibrosis → thick secreı ons .
- Immunodeficiency→ more infections
- Adenoids→ mechanical obstrucı on also could be reservoir for the infecı ons.
- GERD → impaired ciliary funcı on.
- Anatomical deformities such as choanal atresia, deviated septum → obstruct the sinuses.
- Allergy.
- Recurrent URI –specially children.

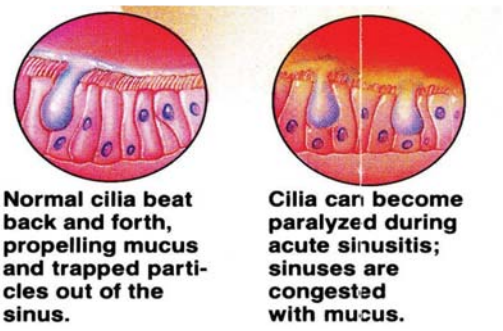
Pathophysiology

- Most important pathologic process in disease is obstruction of natural ostia.
- Obstruction leads to hypo-oxygenation .
- Hypo-oxygenation leads to ciliary dysfunction and poor mucous quality.
- Ciliary dysfunction leads to retention of secretions.

Mucociliary clearance

- Ciliary function very important
- Ostia are small and located in locations not conducive to spontaneous-drainage (ostium of the maxillary sinus and sphenoid also they are not with the gravity which means some mucociliary movement pushing the secretions up then through the ostium).

- Important factors:
  - Number of cilia
  - Structure
  - Activity
  - Coordinated Activity



Normal cilia beat back and forth, propelling mucus and trapped particles out of the sinus.

Cilia can become paralyzed during acute sinusitis; sinuses are congested with mucus.

Decreased MCC

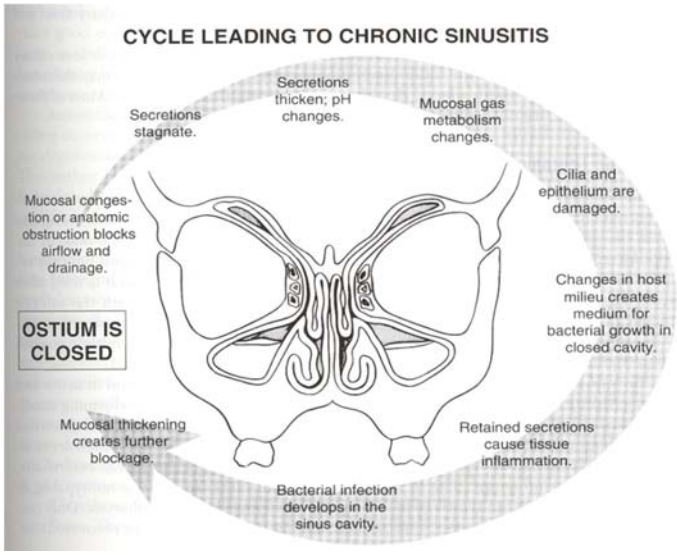
- Kartagener syndrome (Primary ciliary dyskinesia)
- Cystic fibrosis
- Radiotherapy
- GERD
- Rhinosinusitis

Primary ciliary dyskinesia

- Autosomal recessive.
- Dynein arm defects (total, partial, inner, outer or both arms)
- No sex or racial predilection
- Associated with dextrocardia, sinusitis, rhinitis, pneumonia, and otitis media
- Male infertility is common.

Cystic Fibrosis

- Autosomal recessive.
- Decreased chloride secretion with resultant water retention within cell.
- Thicker/stickier mucus adherent to bacteria.
- Leads to infection and inflammation.
- Viscosity leads to dysfunction:
  - Respiratory tract Sweat glands
  - Pancreas Other exocrine glands
  - GI tract



Signs and Symptoms

- Day and night cough
- Purulent nasal discharge (v. imp) (diagnostic) if there is pus coming from the sinus it means sinusitis for sure. We see that then treat it with broad spectrum antibiotic .
- Nasal airway obstruction
- Headache, irritability, or facial pain
- Fever
- Postnasal drip.

Diagnosis- Sinus Aspiration

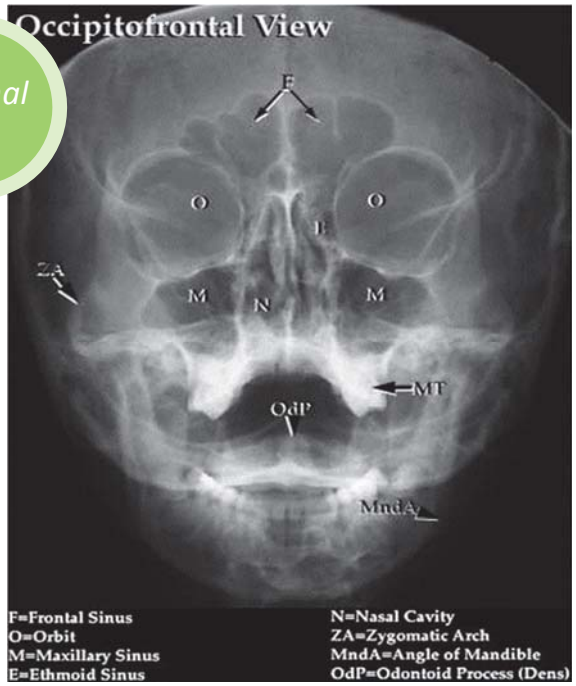
Indications

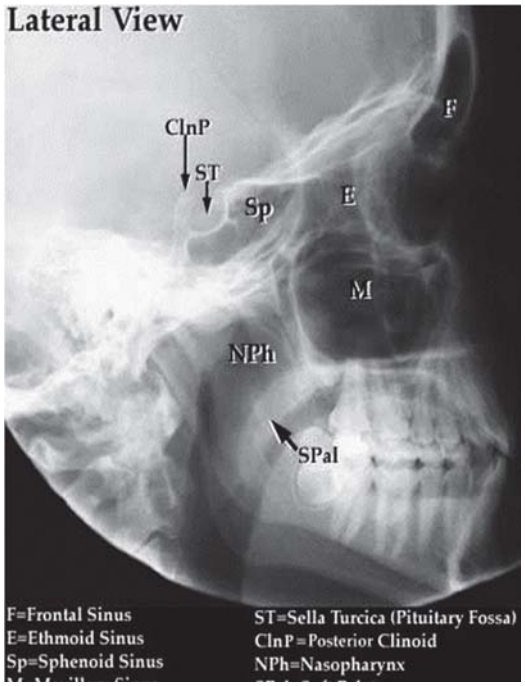
- failure to respond to multiple antibiotics
- severe facial pain
- orbital or intracranial complications
- evaluation of an immunoincompetent host (see which organism causing sinusitis)
- Diagnosis is either clinical or sometimes we need x-ray.

Diagnosis-Imaging

- Standard views
  - Anterioposterior
  - Lateral (we can see adenoid)
  - Occipitomental
- Findings
  - acute-diffuse opacification,mucosal thickening of at least 4 mm, or an air-fluid level

Normal





Diagnosis- CT Scans

- Frequent abnormalities are found in patients with a “fresh common cold”

Indications

  - complicated sinus disease(either orbital or CNS complications)
  - numerous recurrences
  - protracted or nonresponsive symptoms(surgery is being contemplated)
    (the gold standard for investigating chronic sinusitis is CT)

Microbiology

- *Streptococcus pneumoniae* 30-40%
- *Haemophilus influenzae* 20%
- *Moraxella catarrhalis* 20%
- Strep pyogenes 4%
- Respiratory viral isolates 10%
  - adenovirus
  - parainfluenzae
  - influenzae
  - rhinovirus
- Other rarer isolates- group A strep, group C strep, viridians strep, peptostrep, Moraxella species, *Eikenella corrodens*

Treatment-Most Comprehensive Coverage (broad spectrum AB)

- Amoxicillin/potassium clavunate (Augmentin)
- Erythromycin-sulfisoxazole
- Cefuroxime axetil
- Cefpodoxime
- Proxetil
- Azithromycin

Other Medications

- Medications to facilitate drinage:
- Antihistamines if there is allergy
- Decongestants
- Anti-inflammatory agents ex. Steroids which will decrease the edema. So patient who develop sinusitis needs antibiotic + these other medications.

Surgery

- Rarely required
- Consider if orbital or central nervous system complications or
- Failure of maximal medical therapy
- Functional endoscopic sinus surgery (FESS):

- Removal of uncinate process, ethmoid bulla, and variable number of anterior ethmoidal cells, maxillary sinus ostium enlarged and frontal recess diseased tissue is removed if present.

Absolute Indications for Surgery

- Causing brain abscess or meningitis, subperiosteal/orbital abscess, cavernous sinus thrombosis, another contiguous infection, or an impending complication (Pott's tumor)
- Sinus mucocele or pyocele
- Fungal sinusitis
- Nasal polyps (massive)
- Neoplasm or suspected neoplasm

Recurrent Sinusitis

- Most common cause is recurrent viral URIs
  - day care attendance
  - presence of other school age siblings in house
- Other predisposing conditions (we have to rule out these serious conditions):
  - allergic and nonallergic rhinitis
  - CF
  - immunodeficiency disorder
  - ciliary dyskinesia
  - anatomical problem

Acute Fungal Sinusitis

- Uncommon
- Aspergillosis, mucormycosis, candidiasis, histoplasmosis and coccidiomycosis seen
- Aspergillosis most common
- Requires high index of suspscion
- Diagnosed by biopsy and culture

Introduction

- Fungi are ubiquitous (everywhere)
- Immune system keeps organisms suppressed
- Most infections are benign, non-invasive
- Immunocompromised – higher risk of invasive disease

Classification of Infection

- Non-invasive
  - Sinus fungal ball (mycetoma)
  - Allergic fungal sinusitis

- Invasive
  - Acute fulminant invasive fungal sinusitis (very bad)
  - Chronic invasive fungal sinusitis (milder than the acute)

*invasive : invading the mucosa & the basement membrane.

- Aspergillosis a common pathogen of soil, fruits, vegetables, grains, birds and mammals
- Suspect if dark, greasy material seen
- Cultures of nose usually not diagnostic
- Antrostomy (get to the maxillary sinus) with biopsy and fungal stain required

Sinus Fungal Ball (Mycetoma)

- Sequestration of fungal elements within a sinus without invasion or granulomatous changes
- Inhaled spores → grow while evading host immune system (no invasion)
- **Aspergillus most common species**
- Maxillary sinus most oı en involved (70-80% of cases)

Clinically

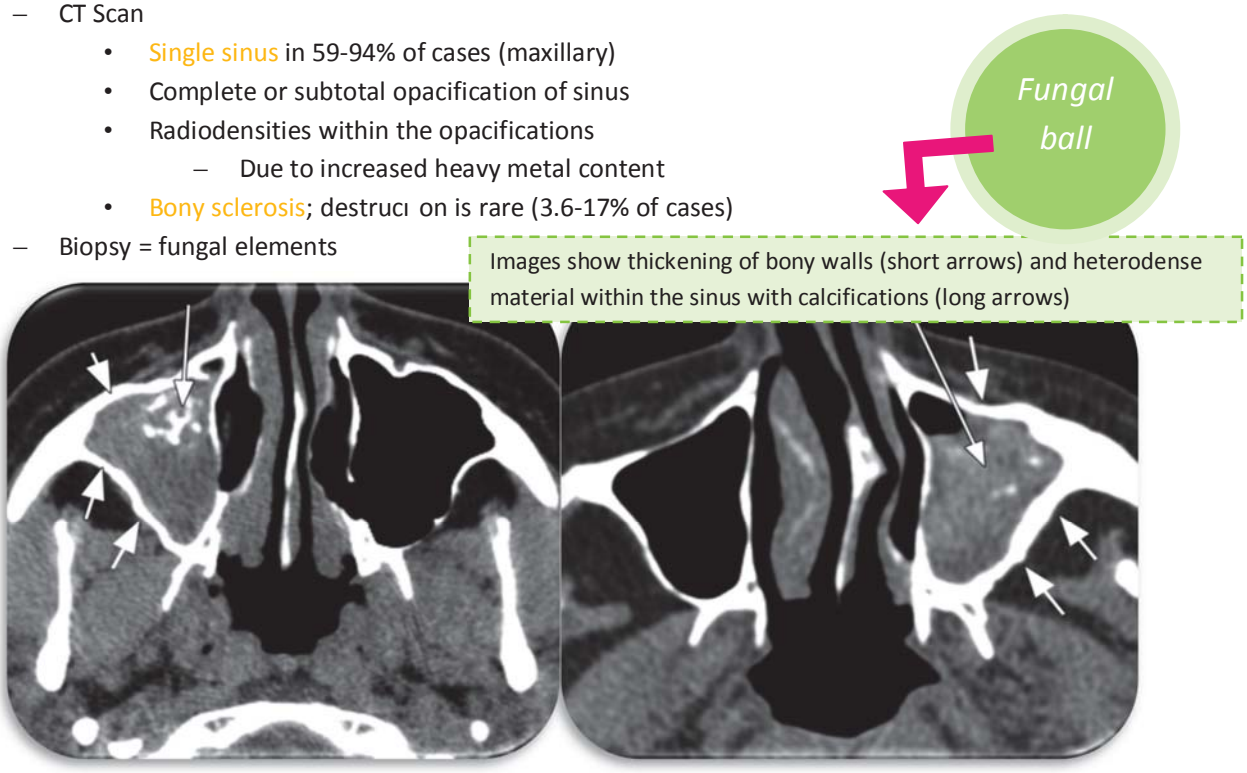
- Symptoms due to mass effect and sinus obstruction
- Presents similar to rhinosinusitis
- Congestion, facial pain, headache, rhinorrhea

Physical examination

- Mild to minimal mucosal inflammation
- Polyps in 10% of cases

Diagnosis

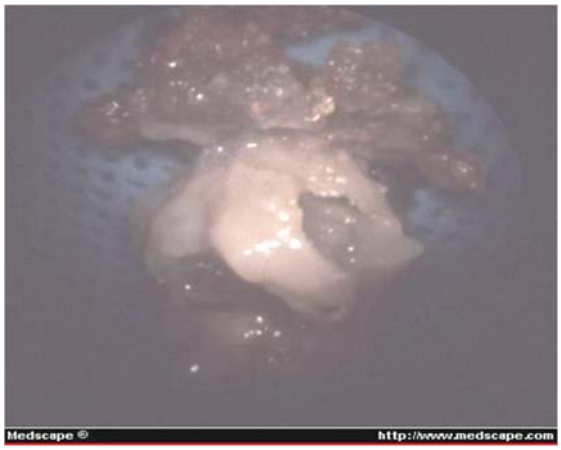
- CT Scan
  - Single sinus in 59-94% of cases (maxillary)
  - Complete or subtotal opacification of sinus
  - Radiodensities within the opacifications
    - Due to increased heavy metal content
  - Bony sclerosis; destrucı on is rare (3.6-17% of cases)
- Biopsy = fungal elements



Images show thickening of bony walls (short arrows) and heterodense material within the sinus with calcifications (long arrows)

Treatment

- Complete surgical removal of fungal ball
- Irrigation of involved sinuses
- Antifungal therapy
  - Only if patient is high risk for invasive disease (very rare)
    - Severely immunocompromised
    - Continued recurrence of disease despite proper medical/surgical management
  - Consider topical antifungal irrigation first and then systemic therapy if no improvement

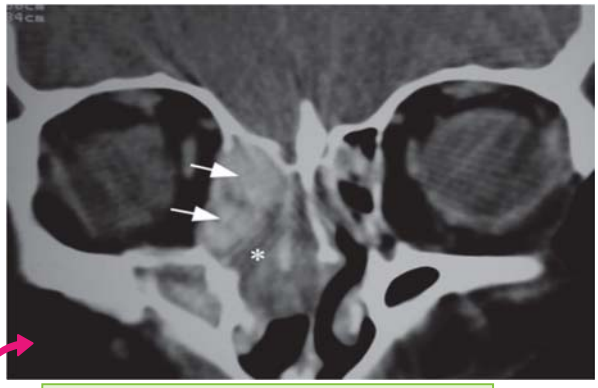


Allergic Fungal Sinusitis

- Fungal colonization resulting in allergic inflammation without invasion
- IgE mediated response to fungal protein
- Symptoms:
  - Nasal obstruction (gradual)
  - Rhinorrhea
  - Facial pressure/pain
  - Sneezing, watery/itchy eyes
  - Periorbital edema

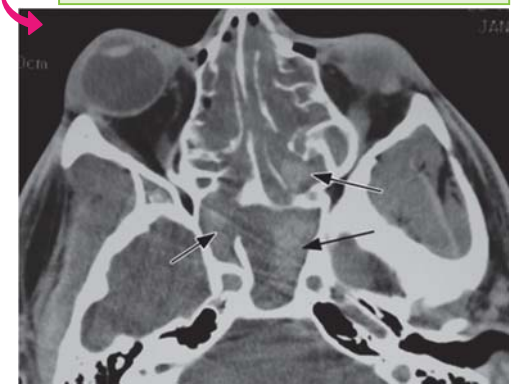
Diagnostic Criteria

1. Eosinophlic mucin
2. Nasal polyposis
3. Radiographic findings (heterogeneity in the sinus)
4. Immunocompetance
5. Allergy to fungi . History of allergy



Double densities (arrows). Expansion of sinus with extension of disease into the nasal cavity (star)

Arrows show double densities. Note sinus expansion but no erosion that's why it is not invasive



Acute Fulminant Invasive Fungal Sinusitis (the worst type)

Patient population

Most often compromised immune system

DM, AIDS, hematologic malignancies, organ transplant, iatrogenic (chemotherapy and steroids).

Most common fungi

- Aspergillus
- Mucormycosis (it is usual here and it is fetal disease)
- Mucor, Rhizopus, Absidia

Less common fungi

- Candida
- Bipolaris
- Fusarium

*not mentioned by the doctor

Pathogenesis

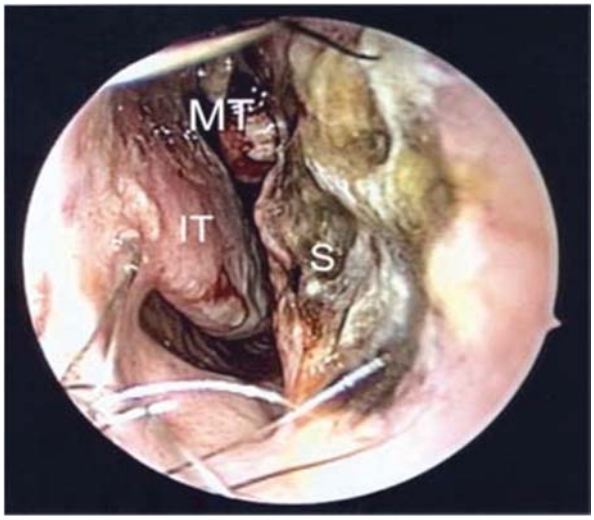
- Spores inhaled → fungus grows in warm, humid sinonasal cavity
- Fungi invade neural and vascular structures with thrombosis of feeding vessels
- Necrosis and loss of sensation → acidic environment → further fungal growth
- **Extrasinus extension occurs via bony destruction, perineural and perivascular invasion**
  - Nasal and palate mucosa destroyed
  - Facial anesthesia
  - Proptosis
  - Cranial nerve deficits
  - Mental status changes

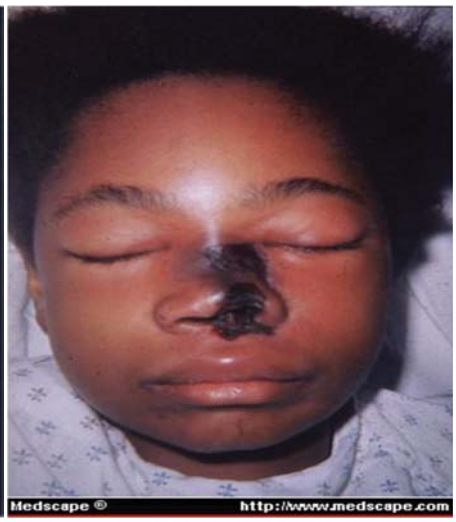
Other signs/symptoms

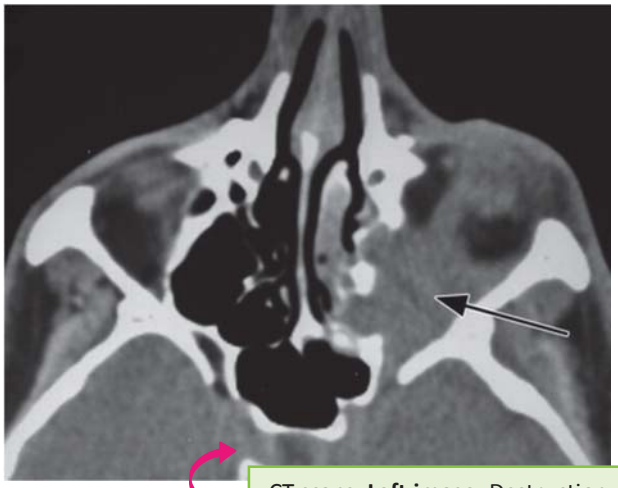
- Fever (most common – 90% of cases)
- **Loss of sensation over face or oral cavity (maxillary nerve)**
- **Ulceration of face and sinonasal/palatal mucosa**
- Rhinorrhea, facial pain/anesthesia, headaches
- Seizures, CN deficits
- Fast progressing symptoms
  - In some cases, hours to days till death!

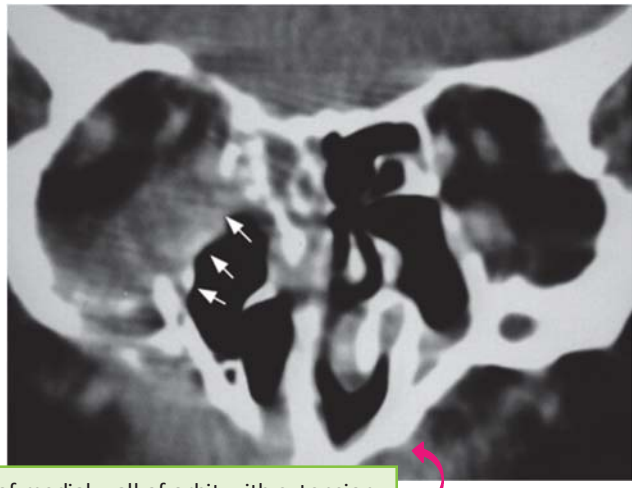
Endoscopic findings

- Loss of sensation and change in appearance of mucosa (pale or black)
  - Most consistent finding
- Ulcerations and black mucosa are late findings
- Serial examinations are required

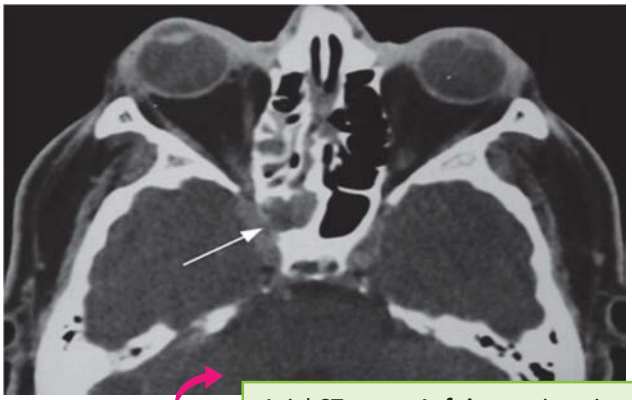


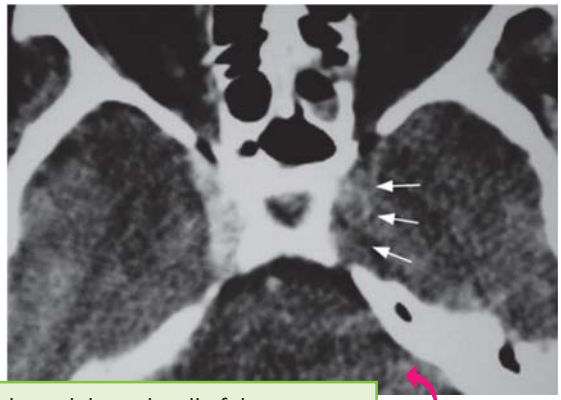






CT scans; **Left image**: Destruction of medial wall of orbit with extension of disease into the orbit. **Right image**: Destruction of medial and inferior walls of the orbit with extension of disease into the orbit





Axial CT scans. **Left image:** invasion through lateral wall of the sphenoid sinus and into the cavernous sinus. **Right image:** lack of enhancement of the cavernous sinus due to fungal thrombosis

- Noninvasive Aspergillosis seen as fungal ball, usually in maxillary sinus
- Invasive aspergillosis can invade bone.
- Fulminant aspergillosis occurs in immunocompromised and invades adjacent structures
- Therapy for noninvasive forms is surgical excision followed usually by PO antifungals

- Therapy for invasive forms requires wide local debridement and intravenous ampo B
- Mucormycosis is encountered in dust and soil and enters through the respiratory tract
- The fungus invades vascular channels and causes hemorrhagic ischemia and necrosis
- Frequently fatal. 90% mortality in immunocompromised .

- Ketoacidosis predisposes to mucormycosis, as the fungus thrives in acidic environments
- Initially seen as engorgement of turbinates, followed by ischemia and necrosis of the turbinates and adjacent nose
- Treated with radical surgical debridement, amphotericin B and correction of underlying immunosuppression

Chronic Invasive Fungal Sinusitis

- Slower disease process than acute
- Rare
- Biggest difference:
  - Most patients are **immunocompetent**
- Common fungi
  - Aspergillus (most common - >80% of cases)
  - Bipolaris
  - Candida
  - Mucormycosis

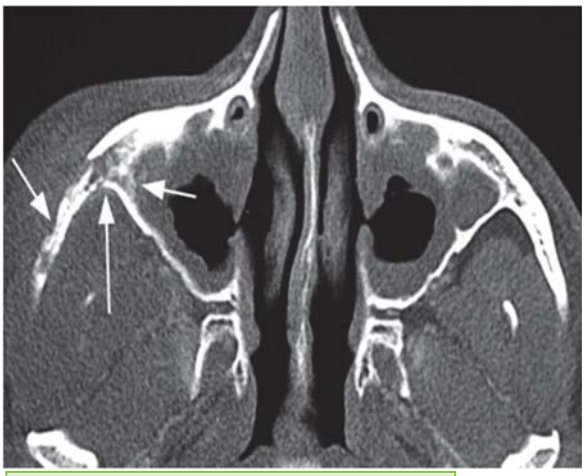
Signs/Symptoms

- Similar to symptoms of chronic rhinosinusitis
  - Nasal congestion, rhinorrhea, facial pressure, headaches, polyposis
- Proptosis, visual changes, anesthesia of skin, epistaxis
  - More concerning
- Does not respond to antibiotics
- Worsens with steroids

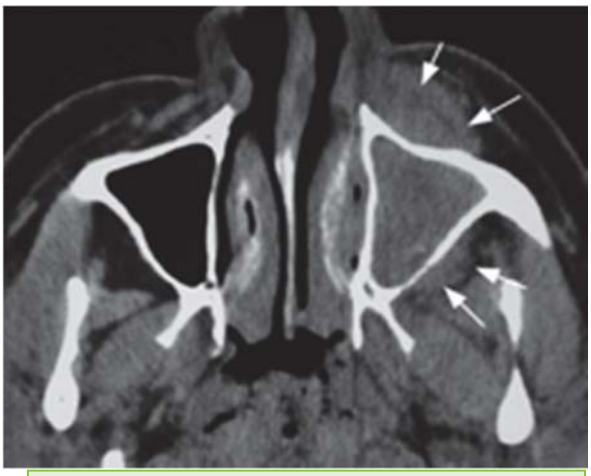
→ Which give you a hint that it is fungal not bacterial

Diagnosis

- Full H&N "head and neck "examination with nasal endoscopy
  - Nasal polyps, thick mucus
  - Rarely find ulcerations
  - Biopsy if suspect fungal disease or note any changes
- CT & MRI
  - Similar findings to AFIFS (Acute Fulminant Invasive Fungal Sinusitis)– bony destruction, extrasinus extension, unilateral. But the patient here is imunocompetent.



CT showing destruction of right lateral maxillary sinus and zygomatic arch



CT showing opacification of left maxillary sinus with extrasinus extension of disease into the periantral tissues (arrows)

Complications of Sinusitis

Orbital

- Most commonly involved complication site
  - Proximity to ethmoid sinuses
  - Periorbita/orbital septum is the only soft-tissue barrier
  - Valveless superior and inferior ophthalmic veins
- Children more susceptible
  - < 7 years – isolated orbital (subperiosteal abscess)
  - ≥ 7 years – orbital and intracranial complications why??
  - Remember intracranial complications usually are either from frontal or sephnoidal sinuses which develop aı er 7 years.
  - Where orbital complications alone caused by ethmoid then maxillary which develop early in age.
  - Children get infections more than adults and so orbital complications.

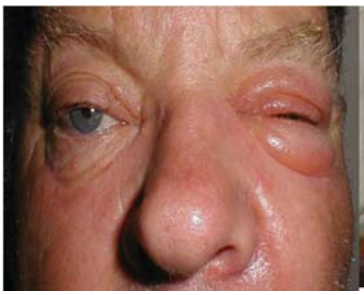
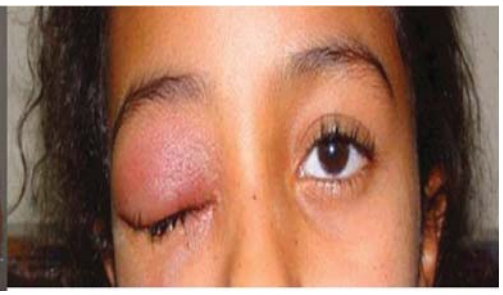
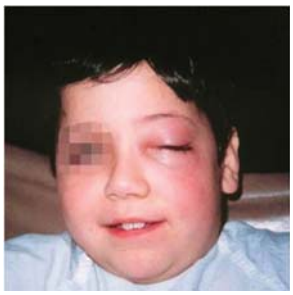
Chandler Criteria

- **FIVE CLASSIFICATIONS**
  - Preseptal cellulitis
  - Orbital cellulitis
  - Subperiosteal abscess
  - Orbital abscess
  - Cavernous sinus thrombosis

In the complications, dr.sami mentioned mainly the treatment of each complication .

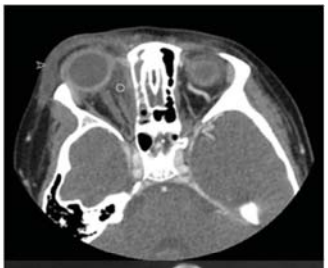
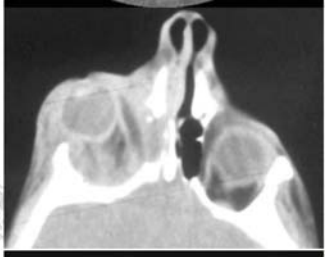
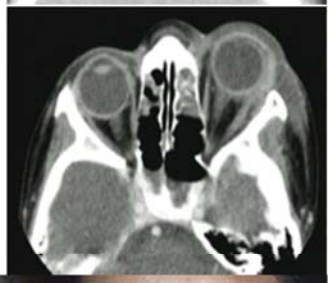
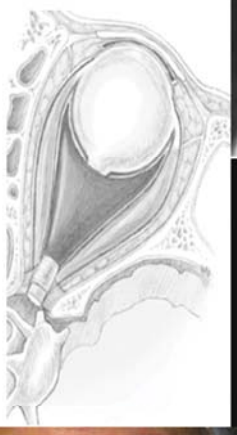
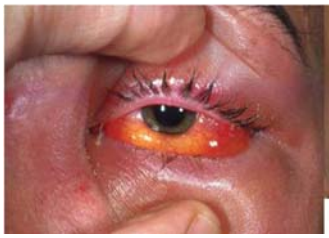
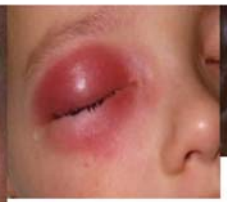
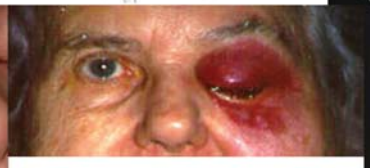
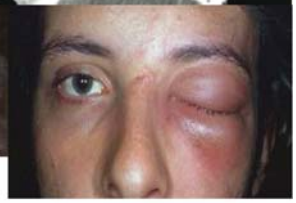
PRESEPTAL CELLULITIS

- Medical therapy typically sufficient
  - Intravenous antibiotics
  - Head of bed elevation
  - Warm compresses
- Facilitate sinus drainage
  - Nasal decongestants
  - Mucolytics
  - Saline irrigations

ORBITAL CELLULITIS

- Symptomatology
  - Eyelid edema and erythema
  - Proptosis and chemosis
  - Limited or no extraocular movement limitation
  - No visual impairment
  - No discrete abscess
- Low-attenuation adjacent to lamina papyracea on CT
- Facilitate sinus drainage
  - Nasal decongestants
  - Mucolytics
  - Saline irrigations
- Medical therapy typically sufficient
  - Intravenous antibiotics
  - Head of bed elevation
  - Warm compresses
- May need surgical drainage
  - Visual acuity 20/60 or worse
  - No improvement or progression within 48 hours

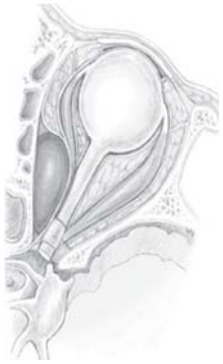
Subperiosteal Abscess

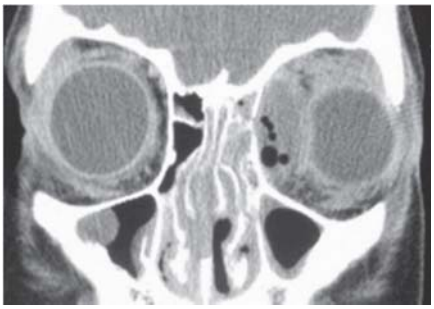
Symptomatology

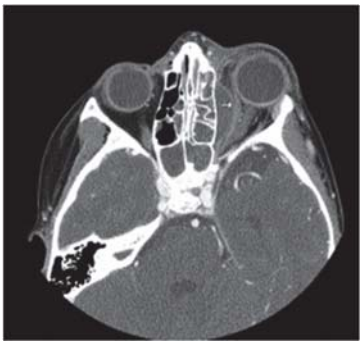
- Pus formation between periorbita and lamina papyracea
- Displace orbital contents downward and laterally
- Proptosis, chemosis, ophthalmoplegia
- Risk for residual visual sequelae
- May rupture through septum and present in eyelids

- Rim-enhancing hypodensity with mass effect

NOTES: Patients will complain of diplopia, ophthalmoplegia, exophthalmos, or reduced visual acuity. The patient has limited ocular motility or pain on globe movement toward the abscess.; may have normal movement early on. Orbital signs include proptosis, chemosis, and visual impairment.





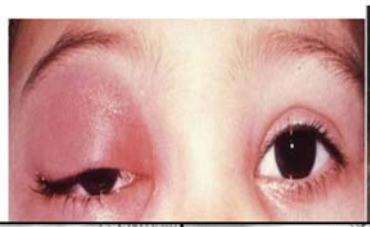


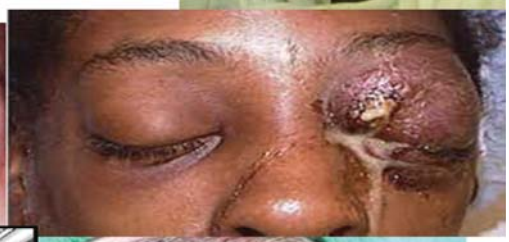
- Surgical drainage
  - Worsening visual acuity or extraocular movement
  - Lack of improvement after 48 hours
- May be treated medically in 50-67%

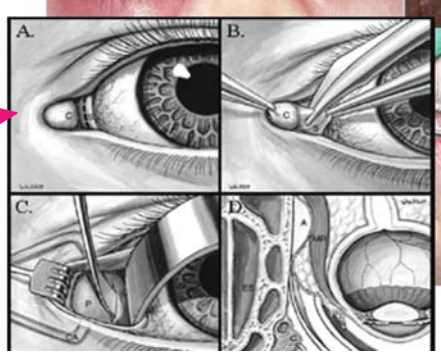
Approaches

- External ethmoidectomy (Lynch incision) is most preferred
- Endoscopic ideal for medial abscesses
- Transcaruncular approach
- Transconjunctival incision

- Extend medially around lacrimal caruncle







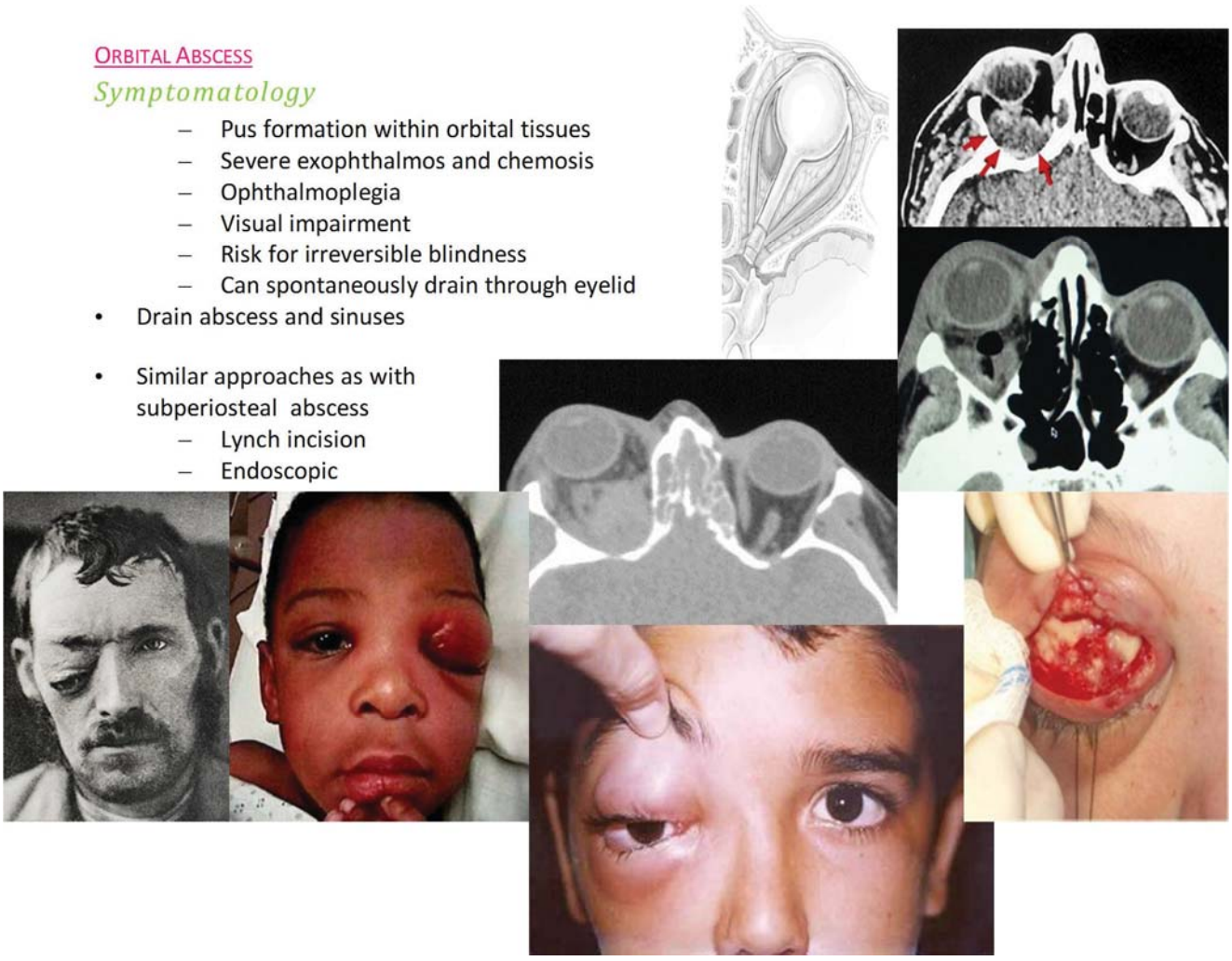
Orbital Abscess

Symptomatology

- Pus formation within orbital tissues
- Severe exophthalmos and chemosis
- Ophthalmoplegia
- Visual impairment
- Risk for irreversible blindness
- Can spontaneously drain through eyelid

- Drain abscess and sinuses

- Similar approaches as with subperiosteal abscess
  - Lynch incision
  - Endoscopic



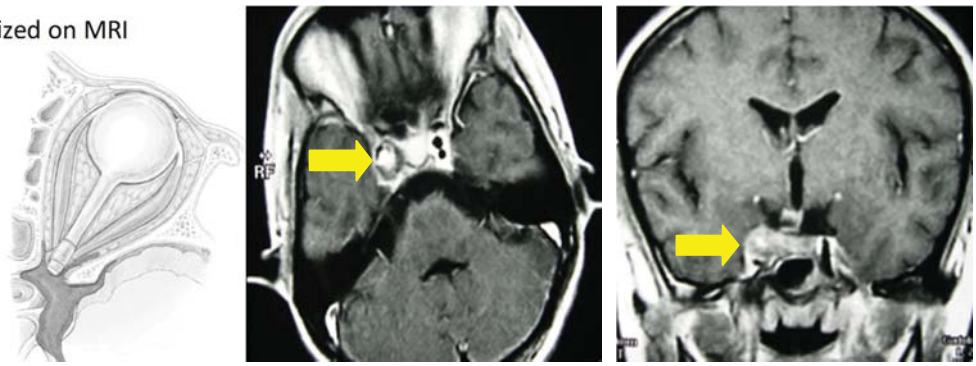
Cavernous Sinus Thrombosis

Symptomatology

- Orbital pain
- Proptosis and chemosis
- Ophthalmoplegia
- **Symptoms in contralateral eye (both eyes will be involved)**
- Associated with sepsis and meningismus

Radiology

Better visualized on MRI



- Mortality rate up to 30%
- Surgical drainage
- Intravenous antibiotics
  - High-dose
  - Cross blood-brain barrier
- Anticoagulant use is controversial

- Mortality rate up to 30%
- Surgical drainage
- Intravenous antibiotics
  - High-dose
  - Cross blood-brain barrier
- Anticoagulant use is controversial

Intracranial complications of sinusitis

- Male teenagers affected more than children
- Direct extension
  - Sinus wall erosion
  - Traumatic fracture lines
  - Neurovascular foramina (optic and olfactory nerves)
- Hematogenous spread
  - Diploic skull veins
  - Ethmoid bone

Types

- **FIVE TYPES**
  1. Meningitis (the most imp)
  2. Epidural abscess
  3. Subdural abscess
  4. Intracerebral abscess
  5. Cavernous sinus, venous sinus thrombosis

Common signs and symptoms

- Fever (92%)
- Headache (85%)
- Nausea, vomiı ng (62%)
- Altered consciousness (31%)
- Seizure (31%)
- Hemiparesis (23%)
- Visual disturbance (23%)
- Meningismus (23%)

MENINGITIS

- Most common intracranial complication of sinusitis

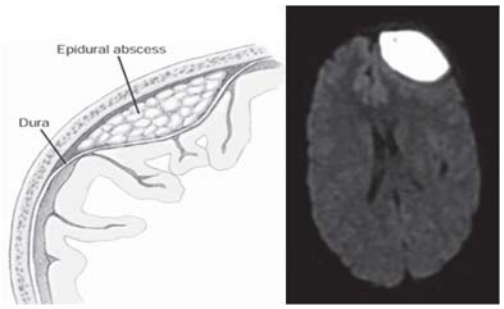
Symptomatology

- Headache
- Meningismus
- Fever, septic

  - Cranial nerve palsies
- Usually amenable with medical treatment
- Drain sinuses if no improvement after 48 hours
- Hearing loss and seizure sequelae

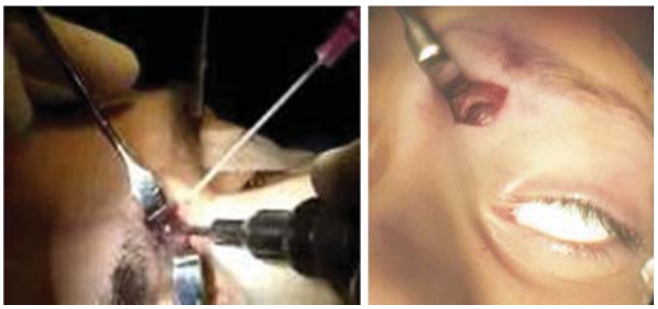
Epidural Abscess

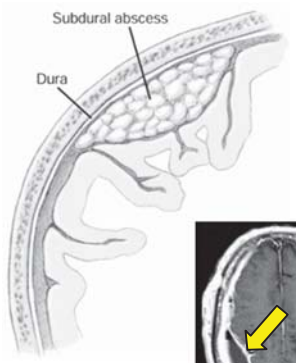
- Second-most common intracranial complication
- Crescent-shaped hypodensity on CT
- Lumbar puncture contraindicated (not to cause herniation)
- Antibiotics
  - Good intracerebral penetration
  - Typically for 4-8 weeks
- Drain sinuses and abscess

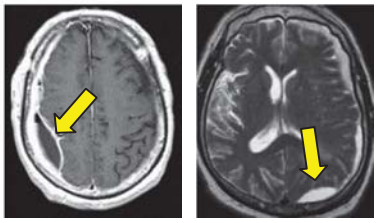


Subdural Abscess

- Generally from frontal or ethmoid sinusitis
- Mortality in 25-35%

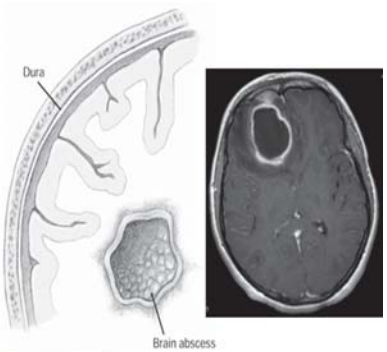






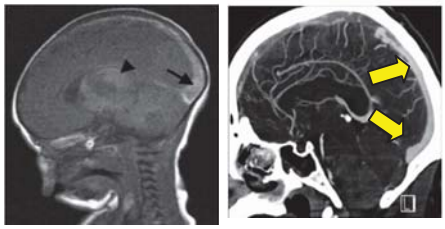
Intracerebral Abscess

- Uncommon, frontal and frontoparietal lobes
- Mortality 20-30%



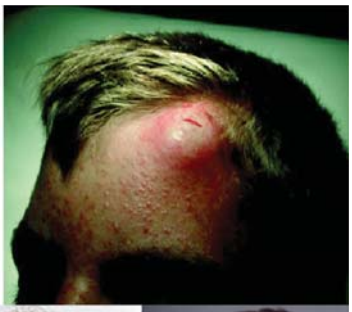
Venous Sinus Thrombosis

- Sagittal sinus most common
- Retrograde thrombophlebitis from frontal sinusitis
- Extremely ill
- High mortality rate



Bony – Complications of sinusitis

- Pott's puffy tumor (osteomyelitis of the frontal bone)
- Frontal sinusitis with acute osteomyelitis
- Subperiosteal pus collection leads to "puffy" fluctuance

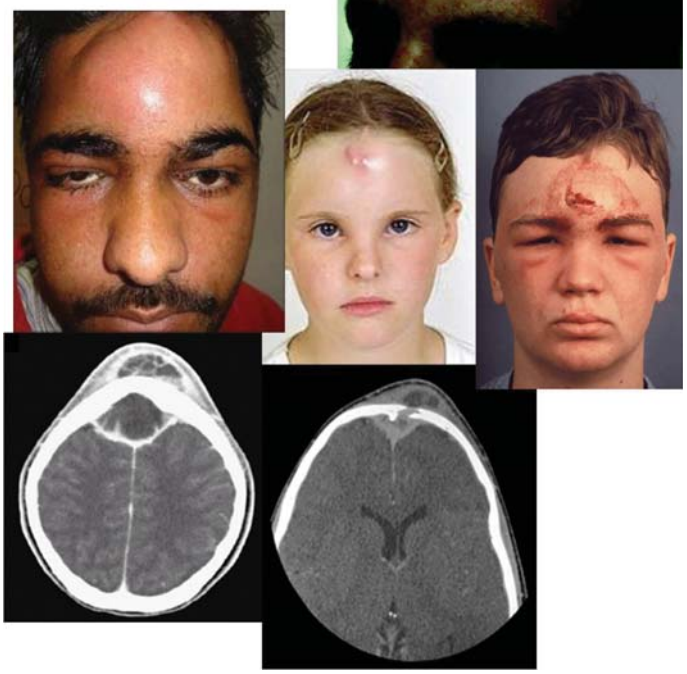


Rare complication

- Only 20-25 cases reported in post-antibiotic era (Raja 2007)

Symptomatology

- Headache
- Fever
- Neurologic findings
- Periorbital or frontal swelling
- Nasal congestion, rhinorrhea



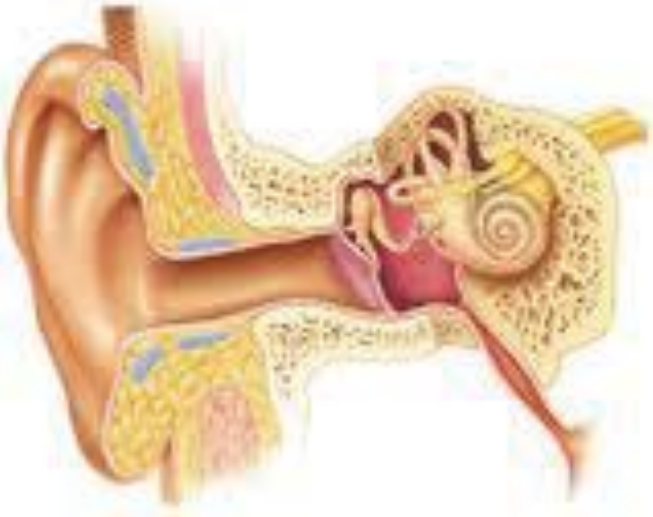
- Associated with other abscesses in 60%
- Cooperative effort
  - Otolaryngology
  - Neurosurgery
  - Infectious disease
- Surgical and medical therapy

Conclusions

- Complications are less common with antibiotics
  - Orbital
  - Intracranial
  - Bony
- Can result in fatal sequelae
- Drain abscess and open involved sinuses
- Surgical involvement
  - Ophthalmology
  - Neurosurgery

430 Teams

Diseases of the Ear, Nose and Throat

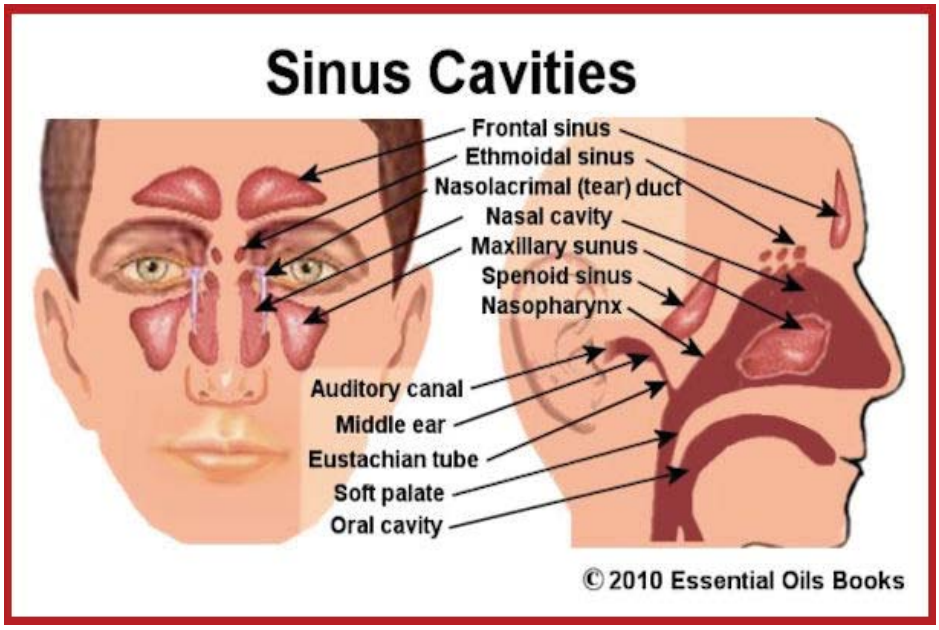


1st Lecture:

Nose3 Sinusitis

Done by: Farhoud

The slides were not provided by doctor (DR.DOUSARY). They were photographed some are clear some aren't. These are my notes and 429team notes. Please do not rely on this as a source of studying.
If not provided mention your source (e.g. 429 slides / females)
Important Notes in red
Copied slides in black



Anatomy:
there are four sinuses: the frontal sinus, the ethmoidal sinus, the maxillary sinus, and the sphenoid sinus.
All sinuses drain into the Osteomeatal Complex except for the Posterior Ethmoid and Sphenoid.
OMC: It is a functional entity where the frontal and maxillary sinuses normally open into the nasal cavity, obstruction predisposes infection in the sinus cavities.

Maxillary, anterior ethmoid, frontal -→ Middle meatus underneath the middle turbinate
Posterior ethmoid, Sphenoid → Superior meatus underneath the superior urbunate
Lacrimal ducts → inferior meatus

Histology:
(Nasal Mucosa) Pseudostratified Columnar Epitheliem
*Cilia specifically arranged (similar mucosa to remainder of tracheobronchial tree)
*Squamous Epitheliem

Normal Function of PNS:
!) patent Ostia 2) Normal ciliary function 3) Normal quality of mucous
disturbance of these functions will lead to sinusitis

Pathophysiology of Rhinosinusitis:
The most important pathologic process in the disease is obstruction of the natural ostia
Obstruction will then leas to hypooxygenation causing ciliary disfuction and poor mucous quality
Ciliary dysfunction leads to retention of secretions.

Acute and Chronic Rhinosinusitis:
Acute: is defined as a disease lasting less than three weeks
Subacute: sinusitis lasting from 1 to 3 months
Chronic: is defined as disease lasting more than 3 months

Etiology:
Inflammatoy: URTI (95% Viral-5% bacterial), Allergic
Mechanical: Naso/septal deformity, OMC obstruction, turbinate hypertorophy, polyps, tumours, large adenoid, foreign body, cleft palate, chonal atresia
Systemic disease: Cystic fibrosis, immobile cilia syndrome, kartegener's syndrome
Miscellaneous: Swimming, flying, diving.

Viral URI:
Clinical presentation:
*unable to differentiate within 10 days
*serous rhinorrhea (maybe mucopurulent)
*nasal congestion and cough prominent
*low grade fevers, malaise, headaches
*nighttime cough may linger

Acute Rhinosinusitis:
*Persistent cold symptoms over 10 days *rhinorrhea *cough (dry or wet) worse at night
*low grade fevers *fetid breath *painless periorbital swelling in the morning *facial pain/ dental pain
*headaches (periodicity) *anosmia *naal and postnasal discharge

Chronic Rhinosinusitis diagnosis: Major and Minor factors
Major: Facial pain/pressure, Facial congestion, Nasal Obstruction/blockage, Nasal discharge/purulence/discolored, PND Postnasal drainage, Hyposmia/anosmia, Purulence in nasal cavity on examination, fever

Minor: headache, fatigue, halitosis, dental pain, cough, ear pain/ pressure/ fullness.

Diagnosis:
*Symptoms present longer than 12weeks
*two or more symptoms one of should be either nasal blockage/obstruction/congestion or nasal discharge (anterior/posterior nasal drip)
*facial pain/pressure
*reduction or loss of smell

Signs: ENT Examination, endoscopy *review primary health care physician's diagnosis and treatment *questionnaire for allergy and if positive. Allergy testing if it has not already been done.

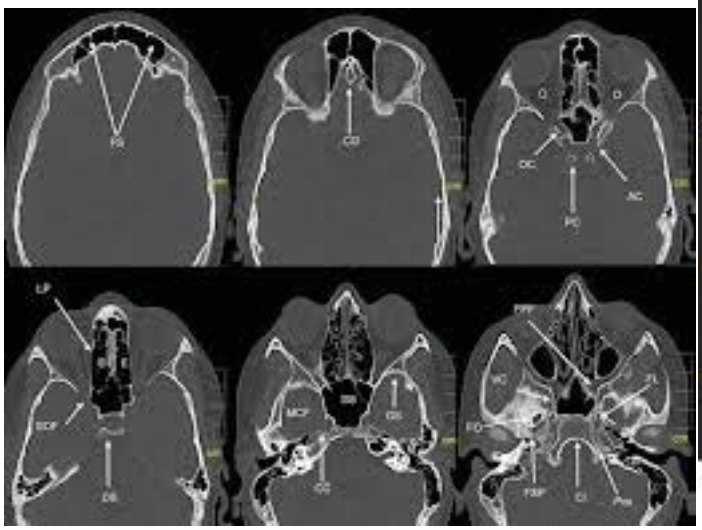
(One of the following situations DX): two major factors, one major factor and two minor, pus in the nose on examination

Physical Examination:
Oropharynx, tenderness over the sinuses, periorbital edema and discoloration
Most specific finding: Mucopurulence, periorbital swelling, facial enderness.

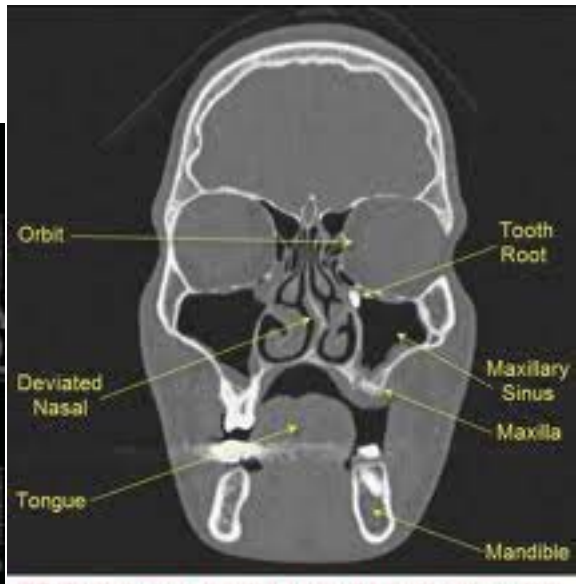
Nasal exam:
Nasal endoscopy: Flexible, Rigide
Plain Radiography lateral, submentovertex

Computed Tomography Coronal (perpendicular to the hard palate), Axial (Parallel to the Hard palate), reformatted sagittal, Multiplannr CT Scan.

AXIAL CT:



CORONAL CT:



CT (Coronal view) showing a root of a tooth lodged within the Ostium of the L *Maxillary Sinus*

*most polyps are from the middle meatus *CT is Gold standard

Sinus Swab and aspirate:
*Nasal, oral, nasopharyngeal cultures give poor results
*needs co-operative patient (usually need middle meatal cultures)
*staining *culture

Microbiology in Acute sinusitis:
-Steptococcus pneumonia 20-30%
-Moraxella catarralis 15-20%
-H.Influenze 16-20%
-Streptococcus Pyogens 2-5%
-Sterile 20-35%
-Anearobs 2-5%
-Rare viruses, anaerobes, staphylococcus
-Normal flora in the sinus (controversial)

Microbiology in Chronic sinusitis:
Gram negative: Bacteroid, Klebcilla
Anearobs
Usually Polymicrobial

Medical Management:
*antibiotic for 10-14days (penicillin, Cephalosporin)
*decongestant (topical, systemic)
*steroid topical spray
*symptomatic treatment
*treat the underlying disease

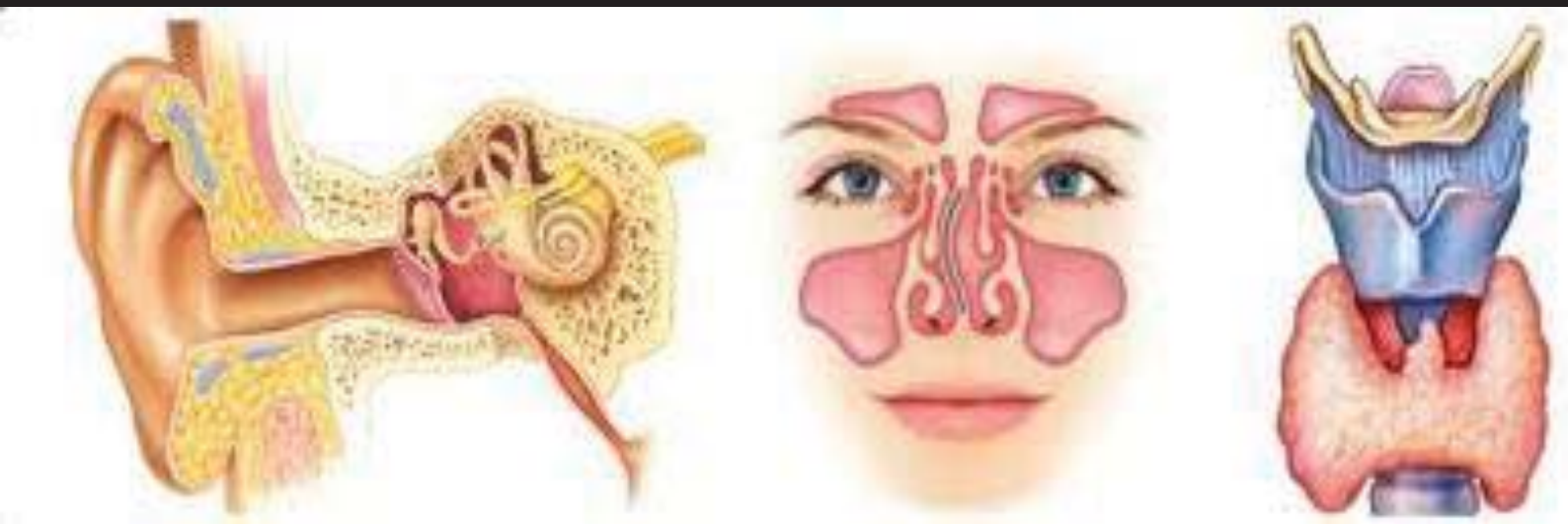
Recalcitrant Rhinosinusitis
*****allergy *Immunodeficiency *Cystic fibrosis *ciliary dismotility disorders *GERD
(Repeate treatment two to three times over 2-3months) + obtain a CT scan

If complication or severe illness: IV Cefotaxime,Ceftriaxone/Clindamycin

Surgery:
Rarely required, concider if orbital or CNS complications or failure of maximal medical RX.
(FESS) Functional Endoscopic Sinus Surgery
removal of uncinate process, ethoid bulla, and variable number of anterior ethmoid cells. Maxillary sinus ostium is enlarged and frontal recess diseased tissue is removed if present

absolute indications of surgery;
Brain abscess or nebibgitis, subperiosteal orbital abscess, cavernous sinus thrombosis, another contagious infection, impending complication (Pott's tumor) Sinus mucocele or pyocele, Fungal sinusitis, nasal polyps (massive), Neoplasm or suspected neoplasm.

Allergic fungal sinusitis:
Fungal colonization resultin in allergic inflammation without invasion. IgE mediated response to fungal protein.
Symptoms: Nasal Obstruction, Rhinorrhea, Facial pressure/pain
Sneezing, watery itchy eyes, periorbital edema.
Diagnostic Criteria:
Eosinophilic mucin, Nasal polyposis, radiographic findings, Immunocompetence, Allergy to fungi.



DISEASES OF THE NASAL SEPTUM, EPISTAXIS, TURBINATE HYPERTROPHY

Done by: Raghad Bokhari
Assisted in the work : Ayan Saeed and May Al-Abdulaaly
Edited by: Yusra Al-Kayyali

Source: slides of the doctor and the recording
Important Notes in red
Copied slides in black
Doctors words in blue
Our notes in green
Titles and subtitles in this color
Highlight possible MCQs mentioned or pointed by the doctor

Causes of epistaxis
Nose blood supply
History, examination, and investigations
Management
Blood loss management
avoidance

Causes Of Epistaxis

Local Causes:

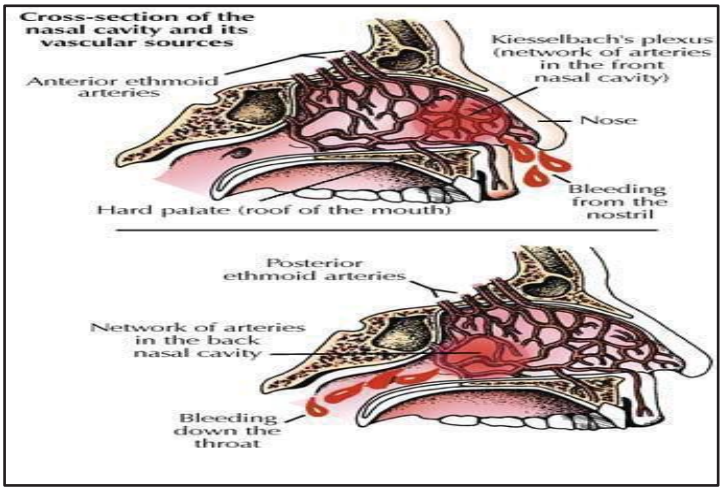
External	**Trauma**	**nose picking** **foreign body** **forceful nose blowing**
	Infection	Chronic Rhinitis
	Irritant \ Allergy	**Chemical Irritant (Nasal Spray),** allergic rhinitis
Internal	Septum	Deviated or Perforated
	Mucosa	**Drying due to low humidity** especially in summer
	Vascular	Vascular malformation
	Mass	Polyp of the septum or lateral nasal wall (inverted papilloma) Neoplasm of the nose or sinus **Nasophargeal Angiofibroma** or Nasopharyngeal Carcinoma , so when adult male presented with epistaxis examine the nasopharynx to exclude carcinoma

Systemic

CVS	Systemic Arterial Hypertension
Endocrine	Pregnancy Pheochromocytoma
Hematological	**Hereditary Hemorrhagic Telangiectasia**(common, runs in families) Thrombocytopenia Idiopathic Thrombocytopenic Purpura (ITP) Leukemia Hemophilia Anticoagulants (ASA , NSAIDs)
GI	Hepatic Disease

Blood Supply:

- Internal and External Carotid Arteries
- Many Arterial and Venous Anastomosis
- Kiesselbach's Plexuses (Little Area) in the Anterior
- Woodruff's Plexuses In Posterior Septum

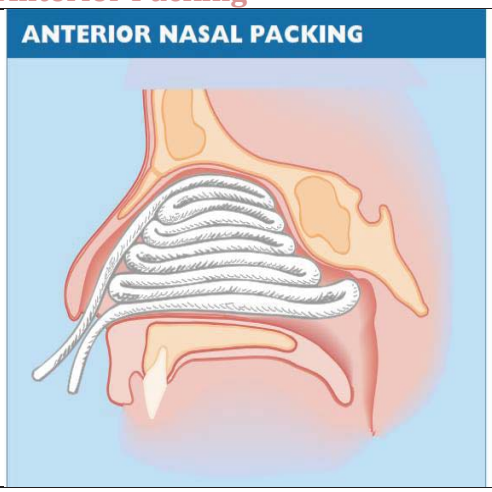
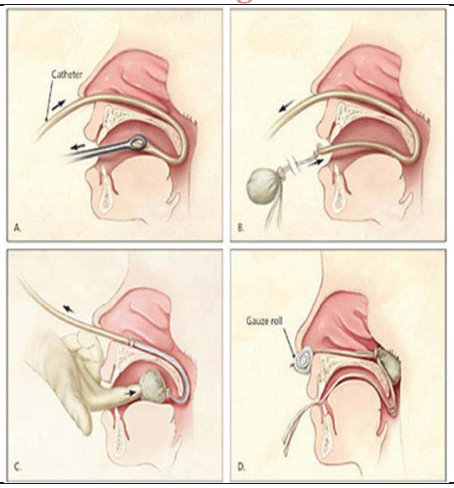


History	Examination
• Previous bleeding episodes • Nasal trauma • Family history • Hypertension –current medication and its control • Hepatic disease • Medical condition, DM, CAD • Use of anticoagulant	• Measure the blood pressure and take the vital signs The 1st change will be increase in the heart rate, BP drops after 25% blood volume loss. • Apply direct pressure to external nose to decrease bleeding • Use vasoconstricting spray mixed with Tetracaine in a 1:1 ratio for topical anesthesia • identify the bleeding source

Assessing Blood Loss	Blood Loss Management
1. Clinical Assessment	1. Blood Loss Control
2. Laboratory Assessment The blood loss assessment in acute phase is by hematocrit level.	2. Blood Loss Replacement if a lot of blood is lost

Types of Nose 3Bleed

TYPE	ANTERIOR	POSTERIOR
AGE	Young population	Older population
PATHOPHYSIOLOGY	Nasal mucosa dryness. Most of the times recurrent	Hypertension Systemic disease Nasal septum deviation
PATTERN	Alternating (but generally less severe)	Significant bleeding in the posterior pharynx
CONTROL	Conservative management	Challenging to control
TREATMENT	• Localized digital pressure **on the tip of the nose** for minimum of 5-10 minutes perhaps up to 20 minutes • Silver nitrate cautery • Topical coagulant : collagen absorbable hemostat or other topical coagulant	• IV pain medication and antiemetics • Topical anesthetic and vasoconstrictive spray for improved visualization and patient comfort • Balloon type epistaxis device (easiest) we don't like to use it because it may damage and cause mucosal laceration. • Folly catheter or posterior packs
Treatment For Refractory Cases	Anterior nasal packing use : • Expandable sponge packing or gauze packing Criteria: • Many shapes and sizes • Impregnated with antibacterial agents	

Type Of Packing	Anterior Packing	Posterior Packing
Image		
Packing Duration	Actual duration will vary according to the patient's particular need	
	At least 24-48 Hours (until there is no bleeding)	At least 48 -72 hours (until there is no bleeding)
Specific Consideration		If balloon is used , advised tapering deflation of the balloons (most successful when volume is documented)
General Consideration	• Best to place the patient on P.O. Antibiotic to decrease risk of sinusitis and toxic shock syndrome • Advice patient to avoid straining, bending forward or removing packing early. • Most patients are treated as outpatients. • Some of the packing is absorbable while other types are not (we use absorbable types in hemophilic patients) • Strongly consider admission in case of : 1. Posterior packing, as SAO_2 should be monitored. keep patient in ICU • admission in case of : 1. Patient has: severe HTN, CAD, significant anemia, and give them supplemental oxygen via humidified face tent.	

Other treatment for Refractory Epistaxis:

- Greater Palatine Foramen Block
- Septoplasty
- Endoscopic Cauterization
- Selective Embolization by interventional radiologist
- Intraoral Maxillary Artery Ligation
- Anterior and Posterior Ethmoid Artery Ligation
- External Carotid Artery Ligation

Preventive Measures

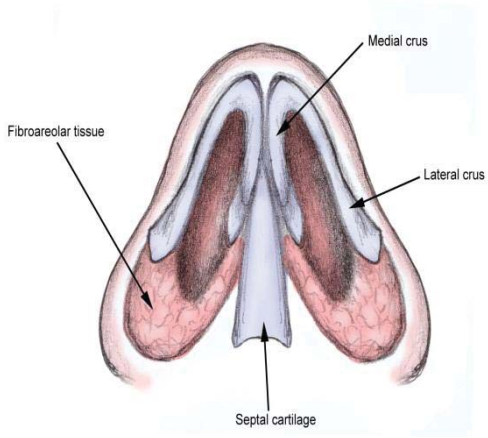
- Keep allergic rhinitis under control
- Use saline spray frequently to clean and moisture the nose
- Avoid forceful nose blowing
- Avoid digital manipulation of the nose with fingers or other object
- Use saline based gel intranasal for mucosal dryness
- Consider using a humidifier in the bedroom.
- Keep vasoconstricting spray at home to use **only during epistaxis.**

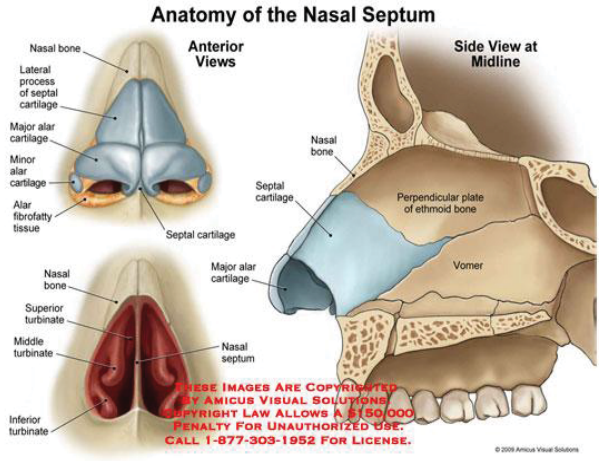
The Nasal Septum:

- Cartilaginous Vault
- Bony Vault
- The Membranous Septum(Mobile Septum)

Cartilaginous Septum

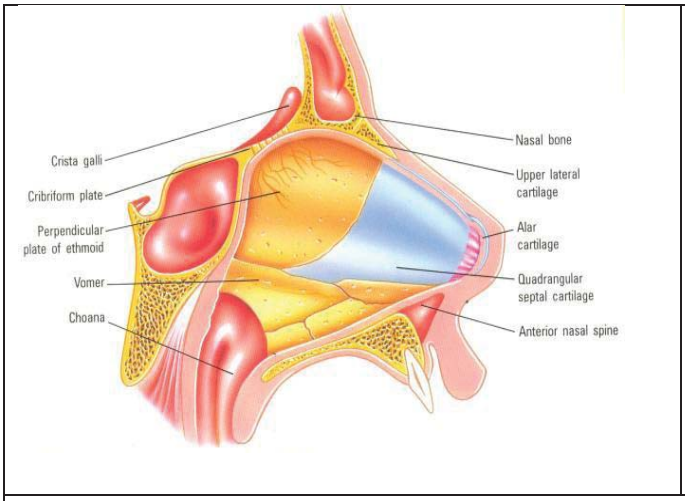
1- Septal (Quadrilateral) Cartilage
2- The Vomeronasal Cartilage (small one)
3- Medial Crura Of The Alar (Lower lateral) Cartilages (anterior part)

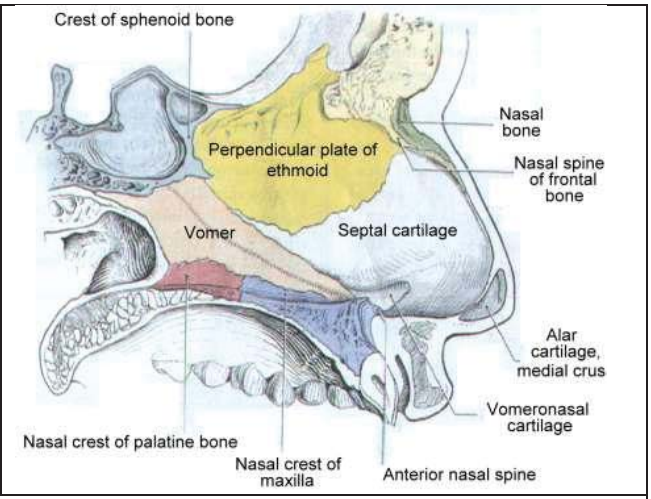




Bony Vault

1- The Vomer
2- The Perpendicular Plate of The Ethemoid





The Vomer

- Develops from connective tissue membrane on each side of the septal cartilage
- For the opposing lamellae of the vomer to fuse the intervening cartilage must be absorbed completely by mid adulthood.

The Perpendicular Plate of the Ethmoid (Mesoethmoid)

- The ossified upper to midline portion of the primitive nasal capsule
- Ossification completely by 17th year age (due to this in case of a nasal surgery we wait until after the age 17 (after the growth of the nose become mature)
- Replacement of the cartilaginous septum with thin bone
- At the nasal roof it articulate with cribriform plate and extend at Crista Gallia

Cribriform Plate

- Fibrous structure until it become ossified in the third year
- Firm union between the lateral and medial Ethmoidal elements.

Also The Nasal Septum Composed Of :

The Membranous Septum (Mobile Septum)

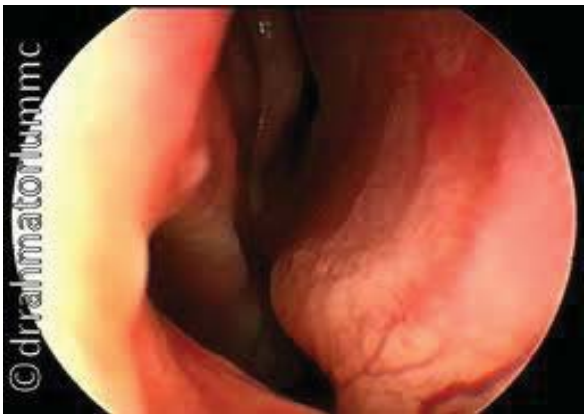
- Anterior to the end of the septal cartilage
- It is formed by skin and subcutaneous tissue of the nasal columella

Septal Articulating Points (fixing points)

- Nasal spine of the frontal bone
- Rostrum of the sphenoid
- Crests of the nasal , maxillary , and palate bone

Inequality of Growth

- Buckle laterally, creating the posterior **septal spur** (excessive bony projection)
- Even in the normal, fully matured septum, elevation and ridge like **protuberance** interrupt the smooth surface.



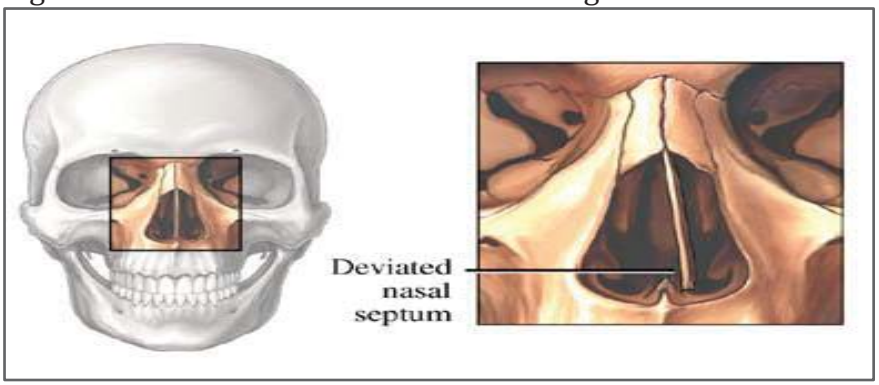
Septal Spur

Most times the septum doesn't grow equally on both sides.
When there is a nasal spur on examination there will be only convex part in one side of the nose, where as in the deviation there is concavity in one side and the other side will be convex.
A spur can occur if a nose is roomy (big) and it wants to narrow it to create turbulence.

There is a difference between dislocation and deviation: dislocation is means it is out of its place and is usually anteriorly.
Most of the time septum deviated anteriorly.

Septal Positions

- Septum **bows** entirely into one nasal cavity
- Double bucking occurs with an **s-shaped** deformity affecting both cavities
- The septal cartilage is often **dislocated** out of the midline groove of the maxillary crest.



Asymmetry of the Nasal Septum

- Approximately 80% of humans have some deformity of the nasal septum.
- Any or all parts of the septum except for **the posterior free border at the choanae**, where it is always midline.
- A common are of the deflection is along the **articulation** between the vomer and the perpendicular plate of the ethmoid, especially when these two bones are separated for considerable distance by the sphenoidal process of the septal cartilage.

Septal Deviations:

- **Types:** traumatic and congenital
- **Common defect:** spur , crest , dislocation of the quadrangular septal cartilage, buckling
- **Signs and symptoms:** **unilateral nasal obstruction** (may be bilateral), hyposmia, **epistaxis,** recurrent sinusitis.
- **Diagnosis:** Anterior Rhinoscopy, or endoscopy.
- **Spurs:** Ridge like deflections and spurs may occur there, even if the rest of the septum is straight.
- **Surgical Management Of Septal Deviation:**
  - **Submucosal Resection:** Obstructing cartilaginous and bony portion of the nasal septum is removed.
  - **Septoplasty**: Removal of the deviated cartilaginous and bony septum with reinsertion after remodeling and repositioning (preserves support system, less risk for perforation).

Septoplasty

Indications:

- Nasal Obstruction (Deviated Nasal Septum)
- Epistaxis
- Chronic Sinusitis (when the septum is obstructing)
- Access for transeptal sphenoidectomy ,
- headache from the impacted spur
- septal neoplasia (rare)

Complications:

- Bleeding, perforation, saddle nose deformity, cribriform plate fracture **(CSF leak)**, septal hematoma, anosmia, septal abscess.

Surgical correction of nasal valve deformities

1- Widening The Valve Apex :
   -Spreader Graft
   -Osteotomies
2- Widening The Valve Angle
   -Flaring Suture
   -Suspension Sutures
   -Butterfly Graft
3- Stiffening The Lateral Cura
   -Alar Batten Graft
   -Lateral Crural J-Flap

Synechia:

adhesion between septum and lateral nasal wall, because there are 2 raw surface areas between the turbinate and the septum sometimes if there are 2 raw surface areas we need to put a sheath to prevent their adhesion

Causes

Manifestation

Treatment: remove the synechia and put a sheath in there.

Septal Perforation:

Cause:

- Septoplasties (most common cause>50%)
- Infections
- Tertiary Syphilis
- Trauma (nose picking)
- Granulomatous
- Vacsulitis
- Cocaine Abuse
- Corticosteroid Nasal Spray

Manifestation:

- Obstruction sensation from turbulent flow , may be asymptomatic
- Crusting
- Epistaxis
- Whistling (if small size)

All these symptoms are not found in the posterior perforation

Diagnosis:

- Anterior rhinoscopy
- Biopsy of granulation tissue or abnormal mucosa

Treatment:

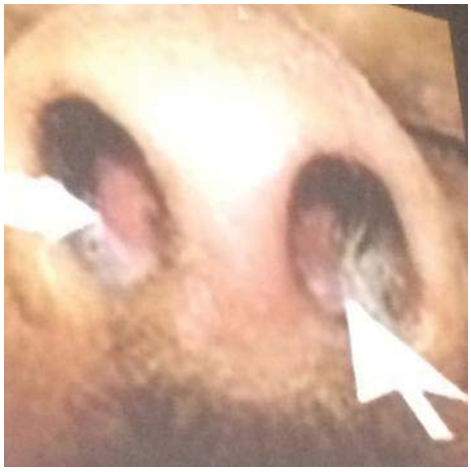
1- Saline irrigation , emollients
2- Consider sliding or rotating mucoperichonderial flaps with or without a fascial graft; contraindicated for large perforation (approximately >2cm of vertical height) cocaine abusers, malignancy, granulomatous or vascular diseases
3- Silastic button

Saddle Nasal Deformity:

Cause
Manifestation
Treatment: rhinoplasty

Septal Hematoma:

- **Pathophysiology:** Hemorrhage (usually from trauma) which collects beneath mucoperichondeium and mucoperiostum resulting in elevation of the mucosa off the cartilaginous septum (loss of vascular supply).



Symptoms and signs:

- Unilateral obstruction (it may be bilateral)
- Septal swelling

Complications:

- Septal perforation.
- Cavernous sinus thrombosis.
- Saddle nose deformity.

- **Abscess formation.**

Treatment:

- Immediate evacuation of the trauma. (both sides)
- Nasal packing
- Antibiotic prophylaxis

emergencies nasal obstruction

DIAGNOSIS	EMERGENCY	COMPLICATIONS
-Septal Hematoma	Elevation of mucosal periconderum with cartilage of a saddle nose deformity devascularization .	**Septal cartilage necrosis development**
-Septal Abscess	Intracranial extension of the infection..	**Septal cartilage necrosis , development of saddle nose deformity,cavernous sinus thrombosis and intracranial infection ..**
-Mucormycosis **FATAL**	Tissue destruction	**Extension to the brain or orbit**

Functional Endoscopic Sinus Surgery:

- Medialization of the middle turbinate
- Excise uncinate process
- Anterior then posterior ethemoidectomy
- Sphenoidectomy
- Frontal recess sinusectomy
- Create maxillary antrostomy

Indications for endoscopic sinus surgery:

Sinusitis	Para nasal sinus	Nose	Ophtha
• Chronic sinusitis, complicated sinusitis, • Recureent acute sinusitis • Failured medical mangment of acute sinusitis • Obstructive nasal polypsosis • Sinus mucoceles • Fungal sinusitis	• Transsphenoidal hypophysectomy • CSF leak repair	• Remove foreign bodies • Tumore excision • Chonal atresia repair • Control epistaxis, • Seproplasty, • Turbinectomy	• Orbital decompression, • Dacryocystorhinotomy, • Orbital nerve decompression, • Grave's ophthalomolgy

Surgical treatment guide:

- Complete extirpation of all the diseases
- Permenant drainage and ventation of the affected sinuses
- Postoperative access to previously diseases areas

Extended FESS:

- CT guided FESS
- Power instrument
- Min FESS

Post operative care:

•sinus packing
•oral antibiotic for a minimum of 2 weeks
•aggressive nasal hygiene to prevent adhesions (saline irrigation)
•nasal steroids
•nasal debridement at 1, 3, and 6 weeks

Excellent results:

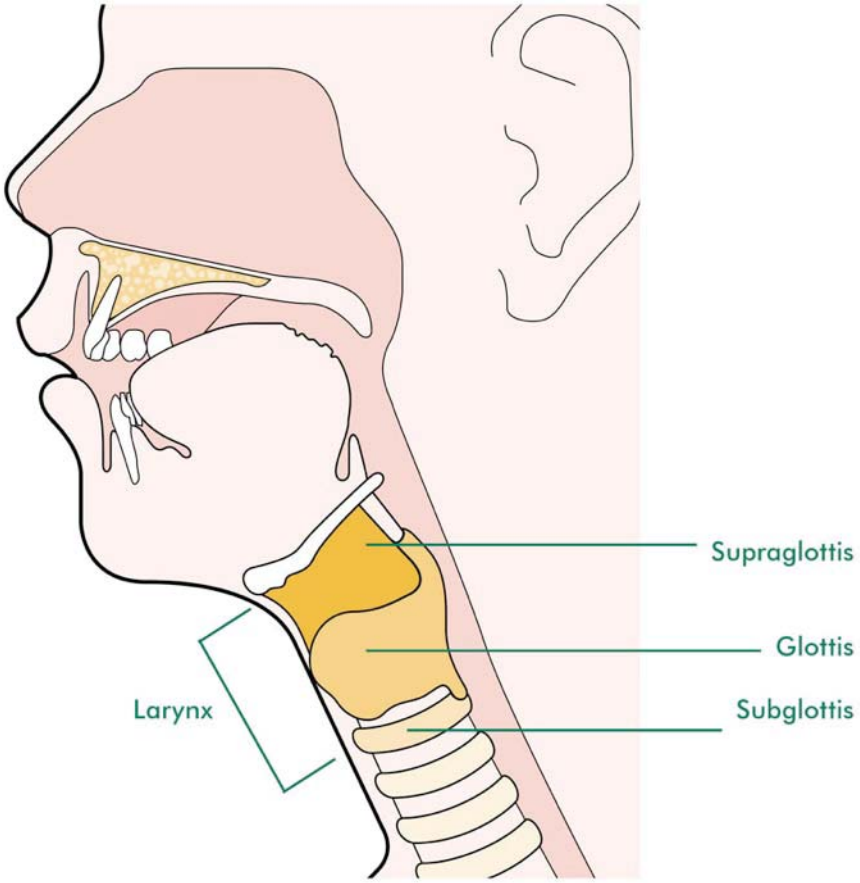
•71% normal at one year
•Meta analysis 89% success with high recurrence
•with 0.6% complications

FESS orbital complications:

•blindness
-indirect injury (retrobulbar hematoma)
-direct injury to the optic nerve
•orbital fat penetration
-increase risk of retrobulbar hematoma
-treatment: recognize orbital fat (orbital fat floats), avoid further trauma, may complicate FESS, avoid tight packing
-observe for vision changes, proptosis, or restricted ocular gaze.

Turbinate hypertrophy

Causes: infection, compensation, dysfunctional, allergies
Manifestations: nasal obstruction, mouth breathing, cause manifestations



LARYNX I&II

429 ENT Team (F2)

Resources: Doctor's lecture, ENT team note, LECTURE NOTES ON Diseases of the Ear, Nose and Throat by P.D. BULL, Ninth Edition

Objectives:

Larynx I

•anatomy and physiology of the larynx.
•gross anatomy , blood and nerve supply.
•congenital diseases of the larynx (in brief) (laryngomalacia, web, subglottic stenosis, and hemangioma).
benign swelling of larynx (Singer's nodule, polyps, granuloma, J. L. papillomatosis).

Larynx II

•acute and chronic laryngitis.
•non-specific laryngitis.
•specific laryngitis (acute epigllotitis, croup).
•laryngeal paralysis (unilateral and bilateral).

Done by: Sarah Al-Muneef

Larynx

Anatomy:

-**Histology:** ciliated columnar epithelium with goblet cells, except over vocal folds; squamous epithelium.

-**Laryngeal Neuromuscular Anatomy:**

1]Extrinsic Muscles: move muscles up & down during swallowing:

-Elevation: digastrics, stylohyoid, mylohyoid, geniohyoid, stylopharyngeus, salpingopharyngeus, palatopharyngeus.

- Depression: sternohyoid, sternothyroid, omohyoid.

2]Adductors 1- lateral cricoarytenoid

2- thyroarytenoid(vocalis) (relaxing the vocal cords)

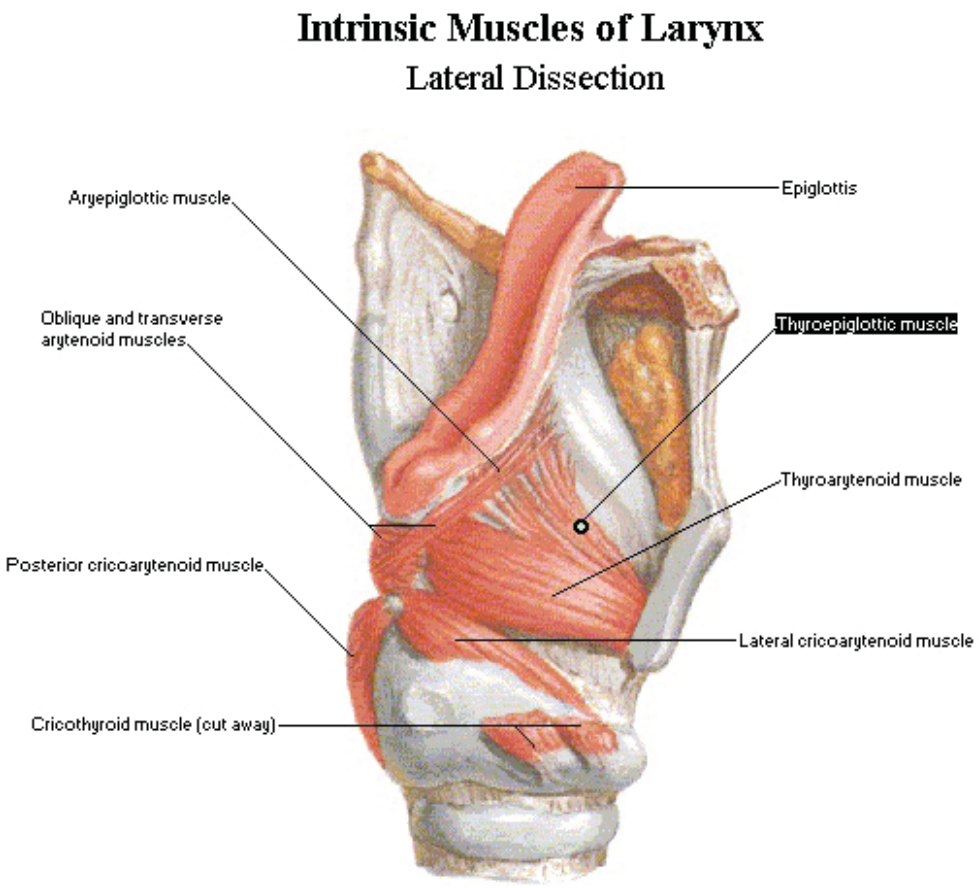
3- interarytenoid (only single muscle)

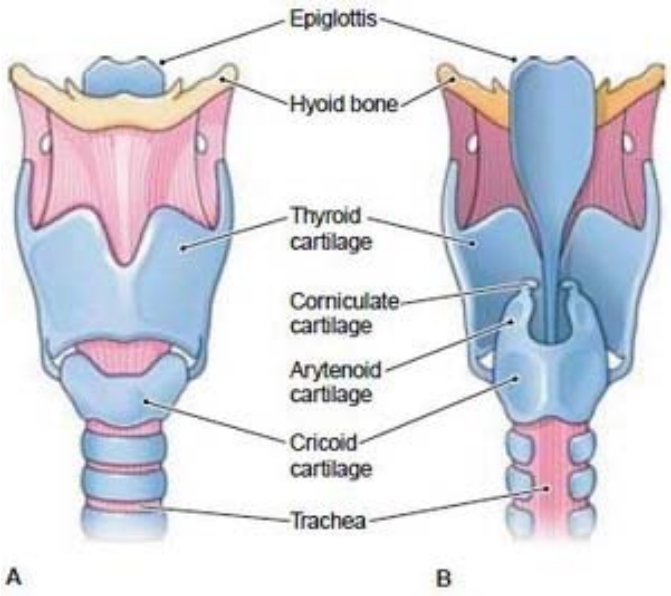
3]Abductors: 1- posterior cricoarytenoid (paralysis will cause asphyxia) **MCQ**

4]Tensors: 1-thyroarytenoid (vocalis) (relaxing the vocal cords),

2-cricothyroid (supplied by superior laryngeal nerve)

Supplied by recurrent laryngeal nerve





-**Laryngeal Cartillages:**-

Single: 1-cricoid: complete signet ring just above the trachea

2-thyroid: largest cartilage of the larynx , felt in midline as "Adam's Apple"

3-epiglottis.→Elastic cartilage

→ Hyaline cartilages

Paired: 1-cuneiform: strengthen aryepiglottic folds

2-arytenoids

3- corniculate: give attachment to aryepiglottic folds

4-trieceous

→ Hyaline cartilages

-Laryngeal Joints: 1-cricothyroid joints→synovial rocks (hinge)

2- cricoarytenoid joints→synovial rocking motion

-Vocal Fold Layers: 1- squamous epitheilium

2-superficial lamina propria (rinke's space)

3-intermediate lamina propria

4- deep lamina propria

5-thyroarytenoid muscle complex

• **Pediatric Airway Anatomy:**
- > 90% of neonates are obligate nasal breathers until 2 months.
-1mm of laryngeal edema in the neonate can be reduce airway by 60%.

-Sensory Innervation:

1- internal branch of superior laryngeal nerve: above vocal cords

2-recurrent laryngeal nerve: below level of vocal cords

Physiology:

1-Phonation:voice (air passes through vocal folds→ vibration→ expiration→ voice)

2-Resonation: most common pathologies

3-Articulation

4-Respiration

Evaluation of the Dysphonic Patient:

-History

- Character of Dysphonia
- Associated Symptoms
- "KITTENS" for differential diagnosis (K:congenital, I:inflammatory,T:trauma, T:tumor, N:neurogenic, E:endocrine, S:systemic)

-Physical Exam

- Quality of Voice
- Indirect and Direct Laryngosocopy (Mirror, Flexible Nasopharyngoscopy, Videostroboscopy)
- Head & Neck Exam

VOICE PARAMETERS :
Pitch. (300-500 Hz) normal speech frequency. Fundamental frequency.(men—>lower frq , women) Loudness (decibels). Quality (Timbre).

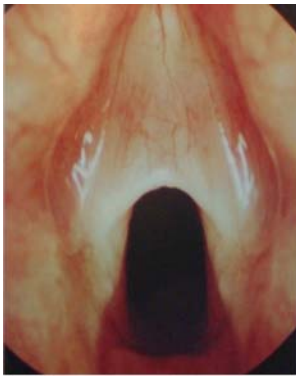
ANCILLARY TESTS
▪Videostroboscopy ▪Laryngeal EMG

To Examine Hypernasality ask the Patient to say S. To Examine Hyponasality ask the Patient to say N (or) M , while the nose is closed

DDx of Dysphonia:

Congenital	Inflammatory	Trauma	Tumor	Endocrine	Neurologic	Systemic
Congenital Under -Developed Larynx	Laryngitis (Viral, Bacterial, Fungal) Vocal Cord Paralysis Adductor Spasmodic Dysphonia Muscle-Tension Disorders	Voice Abuse Reinker's Edema Arytenoid Dislocation Caustic Inhalation Injury	Laryngeal Cysts, Nodules & Ulcers Laryngeal Cancer Benign Laryngeal Neoplasms (Hemangiomas, cystic Hydromas) Vocal Fold Granulomas	Hypothyroidism (Laryngeal Myxedema) Adrenal, Pituitary, Gonadic Disorders Pubescence	Cerebral Palsy Extra Pyramidal Lesions (Parkinsons) Stroke	GERD Connective Tissue Disorders (Rheumatoid Arthritis, SLE) Psychogenic Gullian Barre Myasthenia Gravis Other Neurological Disorders

- **Benign Laryngeal Pathology:**



1)Congenital Laryngeal Defects:

1)Congenital Webs: bands between vocal cords, anteriorly (most commonly)

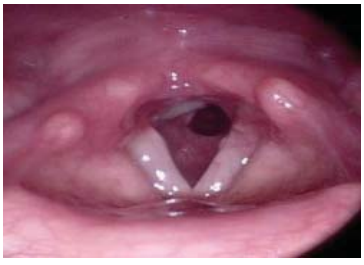
Pathophysiology: results from incomplete recanalization.

Types: supraglottic (2%) glottic (75%), subglottic (7%)

Symptoms: aphonia, stridor

Management: surgical excision by endoscopy or external approach if large.

2)Congenital Subglottic Stenosis: <4mm in newborn, most common disease



Pathophysiology: results from incomplete recanalization

Types (based on stiffness):

1. Membranous
2. Cartilaginous
3. Mixed

Grades:

I.<50% obstruction. } Endoscopic treatment

II.50-70%.

III. 70-90%

IV.> 90-Complete obstruction

Symptoms: stridor

Open Surgery:

- Anterior Cricoid Split
- Posterior Cricoid Split
- Laryngofissure
- **Segmental Resection with End to End Anastomosis**

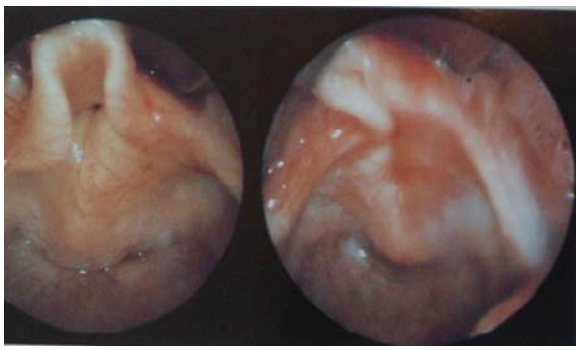
3)Laryngomalacia:

Most common laryngeal anomaly , Most common cause of stridor in neonate and chronic pediatric stridor

Pathophysiology : immature cartilage, omega shaped epiglottis

Symptoms: inspiratory stridor

Management: observation , epiglottoplasty , correct GERD if present.



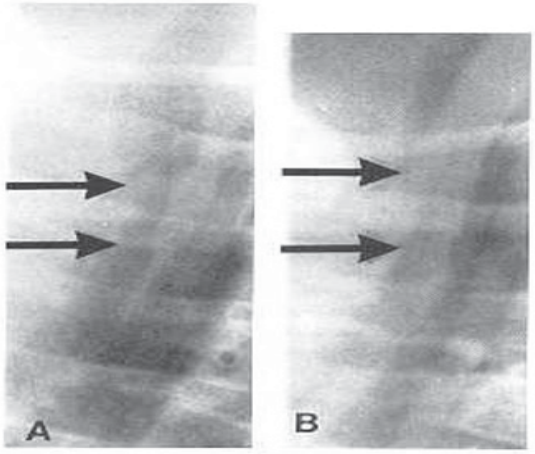
4)Tracheomalacia: Less common

Pathophysiology : immature laryngeal cartillage

Symptoms: expiratory stridor

Diagnosis: bronchoscopy (acolapced area)

Management: observation.



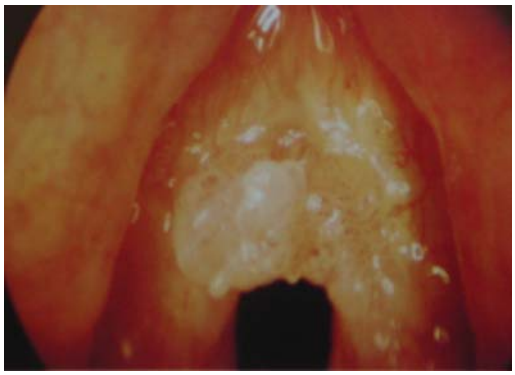
	Pathophysiology	**Symptoms**	**Management**
1-Webs	Bands between cords Results from incomplete recanalization	Aphonia Stridor	surgical excision by endoscopy or external approach if large.
2-Subglottic Stenosis	results from incomplete recanalization	Biphasic Stridor: inspiratory & expiratory	*Grade I-II: Endoscopic management *Grade III-IV: Open Procedures: -Anterior Cricoid Split -Posterior Cricoid Split -Laryngofissure -Segmental Resection with End to End Anastomosis
3-Laryngomalacia	Immature cartilage Omega shaped epiglottis	Inspiratory stridor	-observation -epiglottoplasty -correct GERD if present.
4-Tracheomalacia	Immature laryngeal cartilage	Expiratory stridor	observation

COMMON BENIGN LARYNGEAL NEOPLASMS

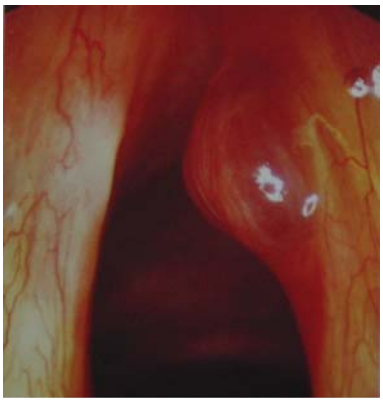
	Pathophysiology	Appearance	Symptoms	Treatment
1]Recurrent Respiratory Papillomatosis	-HPV (6,11) -Hormonal influence -Acquired during delivery *viral infection that affects transit zone of larynx	Wart like lesion. Irregular exophytic.	Hoarseness.(2nd most common cause of hoarseness in children) Stridor.	-Microlaryngoscopy with laser excision. -Avoid tracheostomy. -Adjunctive therapy (a-INF)
2]Systemic Diseases	Sarcoidosis, wegener's granulomatosis, amyloidosis, arthritis of cricoarytenoid joint			
3]Common Laryngeal Lesions:	-Laryngeal -Subglottic			
1-Acquired Stenosis 2-Edema	-Angioedema -Reinke's edema		Common in smokers, reflux & voice abuse. Harsh voice	
3-Cysts	-Laryngeal cysts -Laryngocele	Usually unilateral , on surface		
4-Ulcer	-Contact ulcer (with reflux disease)			
5-Nodules	1)Singers 2)Vocal Cord Callus	Midzone, always bilateral (pointing towards each other) Red, soft, bilateral nodules	Hoarseness when bilateral.	-Speech therapy -Drink water to hydrate vocal cords to decrease friction.
6-Polyp	Most common benign tumor of vocal cord Subepithelial capillary breakage	Mucoid Soft, smooth, fusiform, pedunculated mass Unilateral, asymetric	Structural manifestation of vocal cord irritants Angiomata	-observation
4]Hemangioma	Most common head & neck neoplasm in children -presents by 6 months and involutes by 2 years of age.	Abnormal blood vessel growth. Polypoid or sessile lesion -most common laryngeal site is subglottis.	Biphasic stridor, worse with crying Dysphonia Dysphagia Bleeding in larynx	-Endoscopy (avoid biopsy) -Observe -Embolization -Corticosteriods or interferon -YAG lasers -Radiation therapy

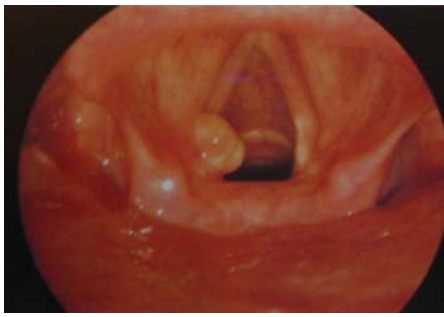
*most common tumors of vocal cords are squamous cell tumors

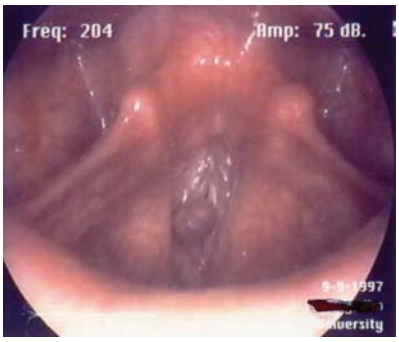
*tumor of vocal cords have good prognosis because there is no lymphatic drainage, unless extended to supraglottic or subglottic regions.

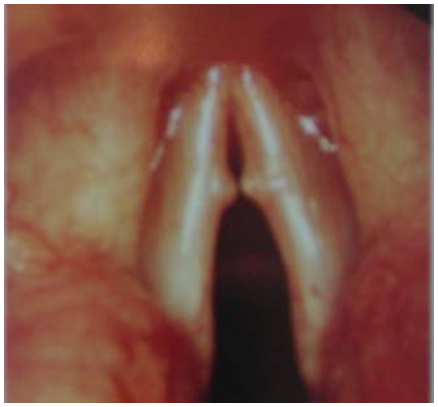


Recurrent Respiratory Papillomatosis







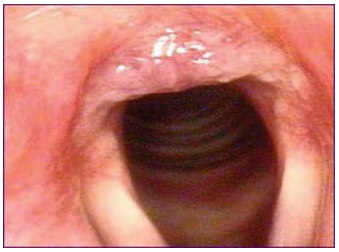


Vocal Cord Nodule

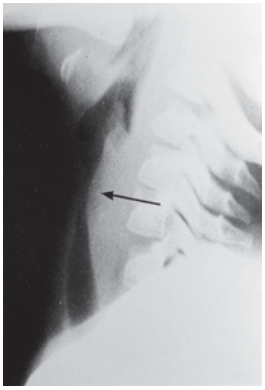
Laryngitis:

	Pathogen/ Pathology	**Symptoms**	**Diagnosis**	**Treatment**
1]Acute Viral Laryngitis (Adults)	Rhinovirus.	-Change in voice (aphonia, dysphonia) -Cough –painful sometimes -Sputum -Malaise -Rigor -Fever	Indirect laryngoscopy: red swollen larynx, sometimes with stringy mucus between cords.	-Conservative management: total voice rest, inhalations with steam, avoid smoking.
2]Adult Supraglottitis	H.influenza. S.pneumonia. S.aureus. B hemolytic streptococcus.	-Stridor change in voice airway collapse -Severe pain (worsened on swallowing)		-Evaluate airway -Humidification -Pareneral antibiotics
3]Reflux-Induced Laryngitis	Increase in saliva production in an attempt to overcome acid chronic inflammation.	-Dysphonia -Cough		-Avoid aggravating factors. -Elevation of head during sleep -H-1 blockers, Proton Pump Inhibitors.
4]CROUP (Acute Laryngotracheobronchitis (LTB))	Para influenza 1.	-Inspiratory or Biphasic stridor. -Gradual onset. -Low grade fever. -Cough (at night)	Plain neck films (steeple) Mucosa becomes swollen & edematous.	-Assess airway. -Medical management: oral steroids, nebulized ventolin, paracetamol. -Endoscopy. -Humidification.
5]Epiglottitis (Children)	Bacterial H.influenza type B (uncommon due to HiB vaccine)	-Drooling saliva. -Dysphagia. -High fever. -Sniff position. -Stridor.	Plain neck films (thumbprint sign) Never examine child in ER.	-Establish emergent airway (endotracheal intubation tracheostomy). -Post op care: pareneral asntibiotics & corticosteroids.

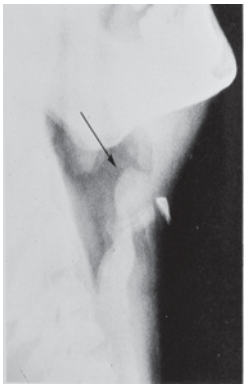
	Pathogen/ Pathology	Symptoms	Diagnosis	Treatment
6]Chronic Laryngitis		-Hoarseness. -Pain. -Chronic cough.	Rule out internal causes and malignancies. Laryngoscopy: cords erythmatous, thickened with ulceration & granuloma formation & normal mobility	-Rest voice. -Treat upper airway sepsis. -Steam inhalations. -Voice therapy.
1-TB Laryngitis	Pulmonary Tuberculosis.			-Antituberculosis drugs.
2-Laryngeal Diphtheria		-Ill -Stridor(spread to larynx & trachea).	Membrane on phaynx.	-Hospital admission. -Antitoxin & general supportive measures. -Tracheotomy.
3-Fungal Laryngitis		-No fever	White membranes.	

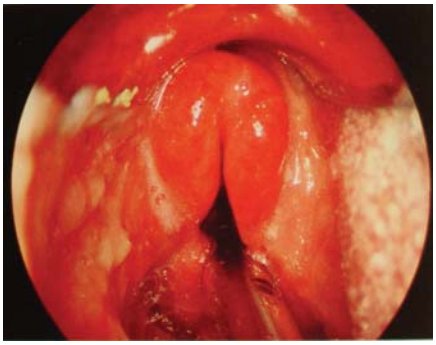


Reflux Induced Laryngitis



CROUP



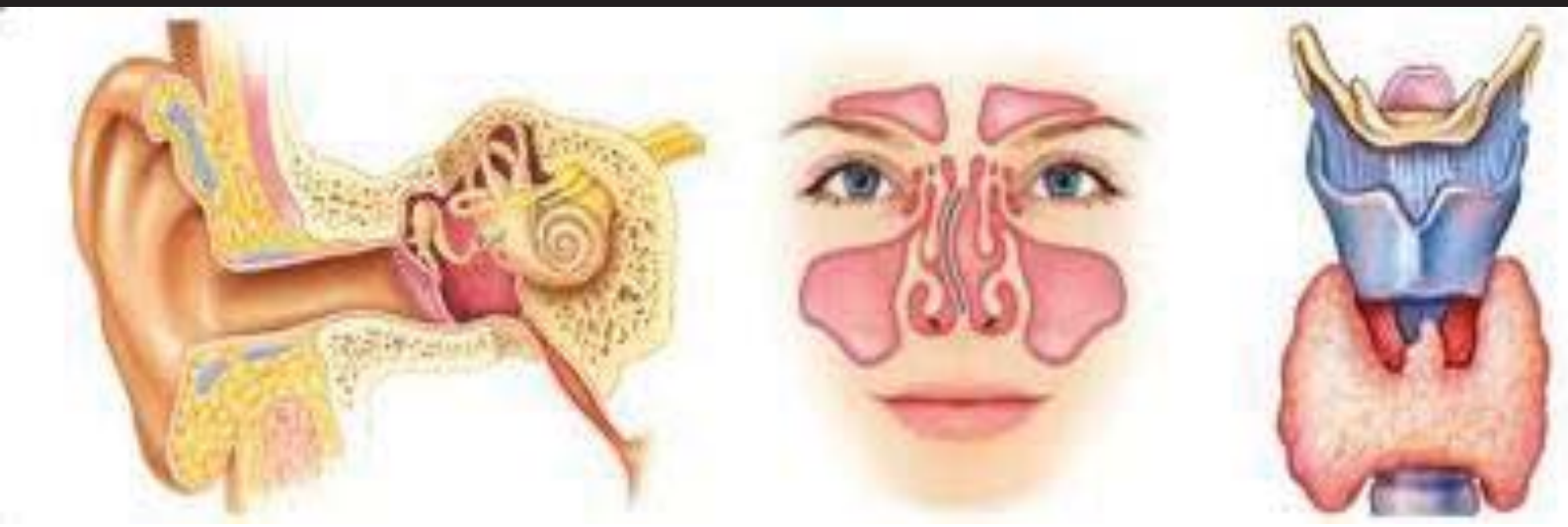


Epiglottitis

	Acute LTB	Acute Epiglottitis
Pathogen	Parainfluenzae virus1	Haemophilis influenzae-B
Age	<5 years old	2.-6 years old
location	Subglottic	Supraglottic
Onset	Gradual (over days)	Sudden onset (hours)
Cough	Barky	Normal
Posture	Supine	Upright
Drooling	No	Yes
Fever	Low grade	High fevers
Radiographs	Steeple sign	Thumb printing
Treatment	Supportive	Airway management

Vocal Cord Paralysis:

Evaluation:	**Causes of Vocal Fold Paralysis in Adults:**	**Causes of Vocal Fold Paralysis in Pediatrics:**	**Management:**	
- History and Physical - Ancillary test - Vocal Fold Positioning: -Recurrent Laryngeal Nerve Paralysis: paramedian vocal folds -Superior Laryngeal Nerve Paralysis: bowing deformity -RLN & SLN Paralysis: cadaveric, intermediate vocal folds -Bilateral Vocal Fold Paralysis: typically near midline	- Neoplastic - Iatrogenic Injury - Idiopathic - Trauma - Neurological - Infectious - Systemic Diseases - Toxins	- Idiopathic - BirthTrauma - Iatrogenic Injury - Infection - Vascular Abnormalities	- <u>Unilateral vocal cord paralysis:</u> •Must determine if self limiting or permanent paralysis. • May not require a surgical management. •The goal is to medialize vocal folds Surgical Management: **1- Vocal Fold Injections.** **2- Thyroplasty** 3-Arytenoid Adduction 5-Tracheotomy	-<u>Bilateral Vocal Cord Paralysis</u>: •The goal is to lateralize vocal folds 1- Tracheotomy: gold standard treatment 2-Cordotomy (Laser) 3Arytenoidectomy 4-reinnervation procedure



Anatomy of Larynx

Done by: Alia K. Habash
Revised by: Yusra Al-Kayyali

Objectives:

- To know the basic larynx anatomy and physiology.
- To recognize assessment and management of common laryngeal diseases, include ability to obtain patients' history, perform comprehensive physical and mental status assessment, interprets findings
- To know how to handle common laryngeal emergencies.
- To be aware of common laryngeal operations.

Larynx I

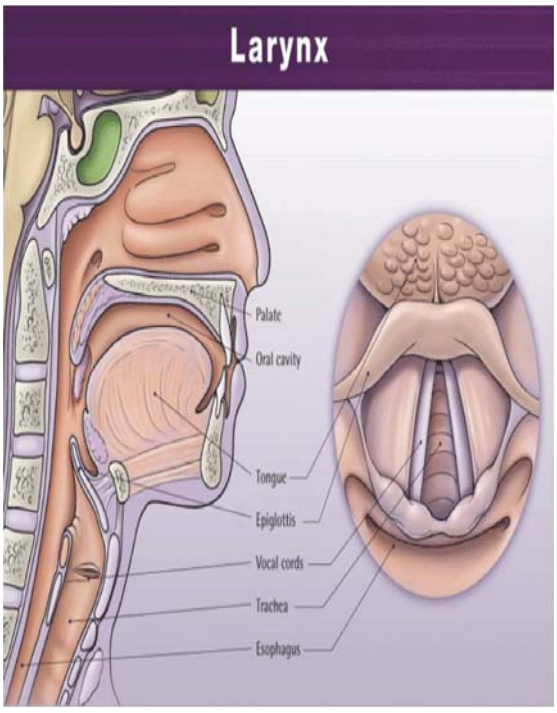
- anatomy and physiology of the larynx
- gross anatomy , blood and nerve supply
- congenital diseases of the larynx (in brief)
- (laryngomalacia, web, subglottic stenosis, and hemangioma)
- benign swelling of larynx (Singer's nodule, polyps, granuloma, J. L. papillomatosis)

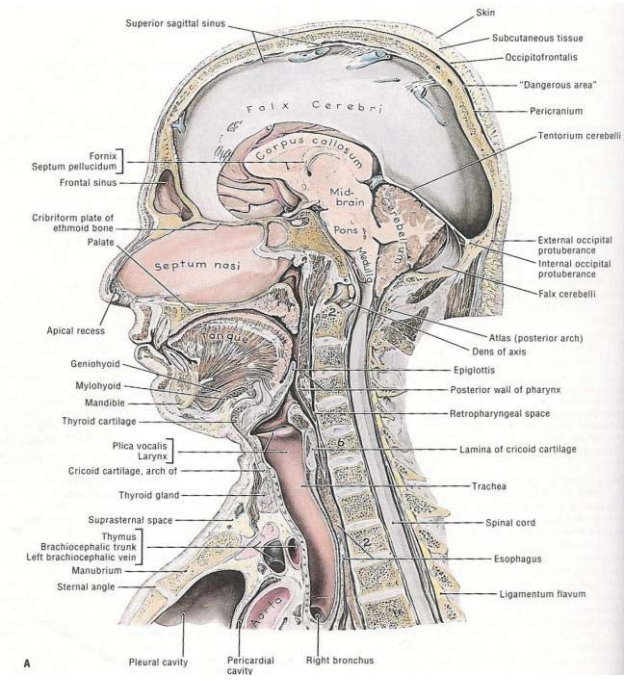
-Dysphonia: is a **descriptive** medical term meaning disorder of voice.
-Hoarseness: is a **subjective** term, and usually refers to a weak or altered voice.
-voice changes are: breathy, harsh, tremulous, weak, reduced to a whisper, or vocal fatigue (voice deteriorates with use).

Dysphonia = Hoarseness
In OSCE ask about: onset, worsening, relating to any factor, URTI and previous surgery…
Breathy : defect in closure of the airway , vocal cord paralysis .

Normally person talks at end of respiration(you breath then talk and never breath in while talking without stopping, therefore while talking you are building up pressure causing the vocal cords to move and close during phonation.. (In breathy voice 1 of the vocal cords is not moving, so instead of saying 10 words in 1 sentence someone with a breathy voice will say around 3 words and stop for a breath)

- Spasmodic dysphonia: hyperadduction of vocal cords
- teachers have voice fatigue and pain at the end of the day.





SKELETOMEMBRANOUS FRAMEWORK OF LARYNX:

1- Thyroid cartilage
2- Cricoid cartilage
3- Epiglottis

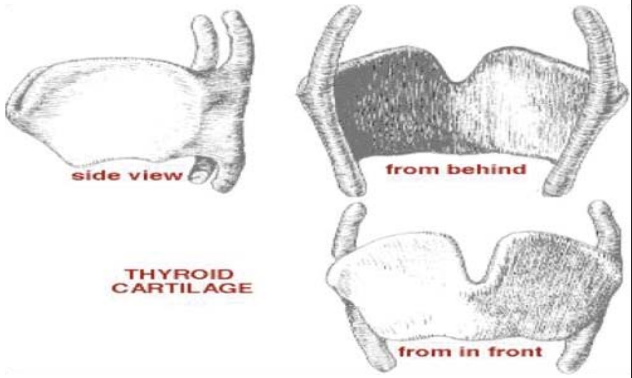
4- Paired arytenoids cartilage

5- Hyoid bone.

1, 2, and 3 are single cartilages while 4 is paired.

1. Thyroid Cartilage: Shield like.

Thyroid cartilage is opened posteriorly.
In men it is noted as Adam's apple.
It is attached to the cricoids.

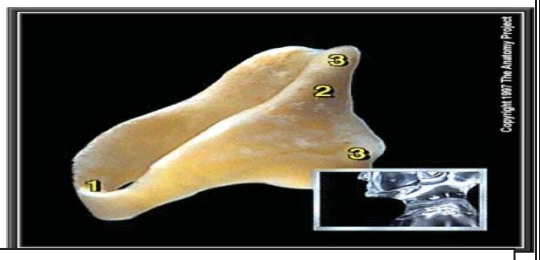


2. Cricoid Cartilage:

- Signet ring shaped.
- The only complete skeletal ring in the air way.

♦Both thyroid and cricoid cartilage ► hyaline ► calcification (seen on X-Ray)

- Cricothyroid joint : Synovial joint ► hinge motion

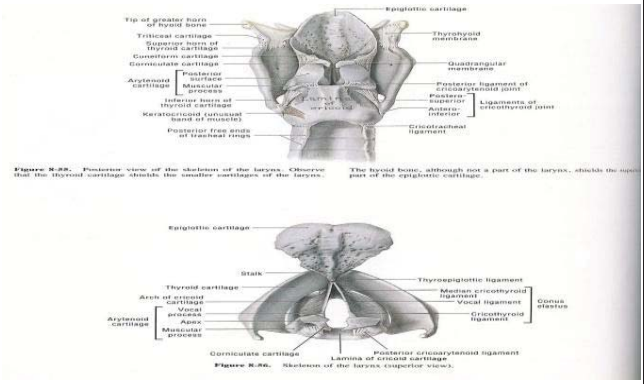


Cricoid Cartilage is the narrowest area where the airway obstruction usually happens because it is a complete ring.
Thyroid and cricoid might be seen like bones (calcified) in an X-Ray of a 40 year old patient.

3. Epiglottis cartilage:

Attached to the inner margin of the thyroid cartilage

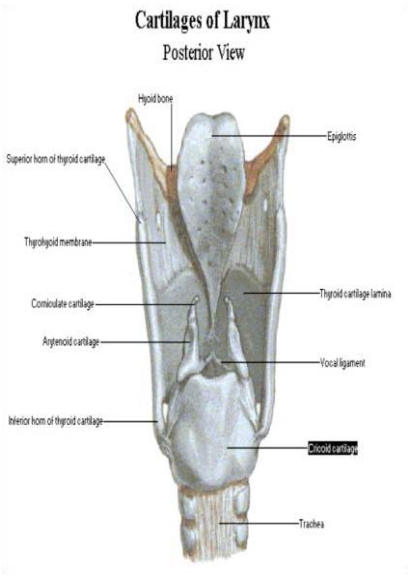
- Leaf like structure
- Elastic cartilage :
  - Thyroepiglottic ligament. to thyroid
  - Hyoepiglottic ligament attached to hyoid
  - Glossoepiglottic fold →Valleculae is the base of the tongue where the tongue is attached to the epiglottis.



4. Arytenoid Cartilage:

- Pyramidal shaped
- Apex, vocal & muscular process.
- Cricoarytenoid joint
  - Synovial
  - rocking motion

Arytenoid :
Vocal process (anteriorly)
Muscular (posteriorly)



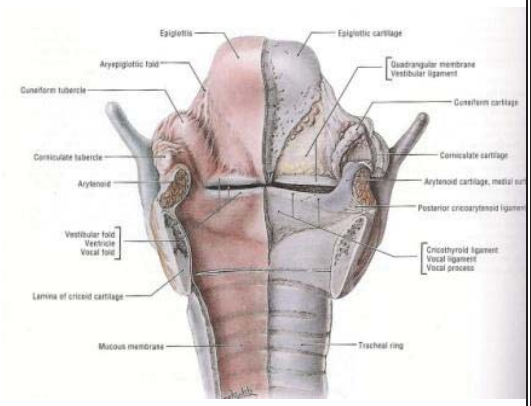
5. Corniculate and cuneiform cartilage.

LARYNGEAL MEMBRANES:

The cartilages are covered by membrane

1. Quadrangular Membrane.

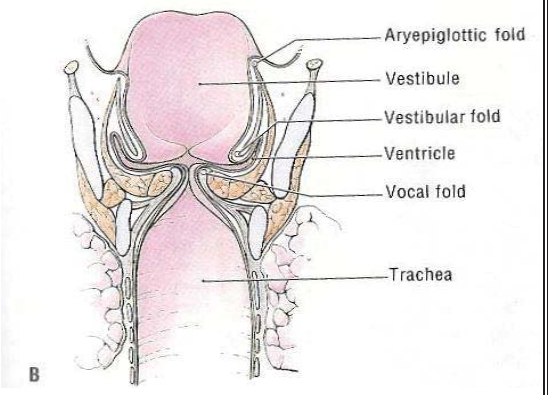
- Upper and lower border ►thickened
- **aryepiglottic fold**
- **Vestibular fold**



2. Triangular Membrane (conus elasticus).

- Medial and lateral border is free► thickened ►**vocal ligament**

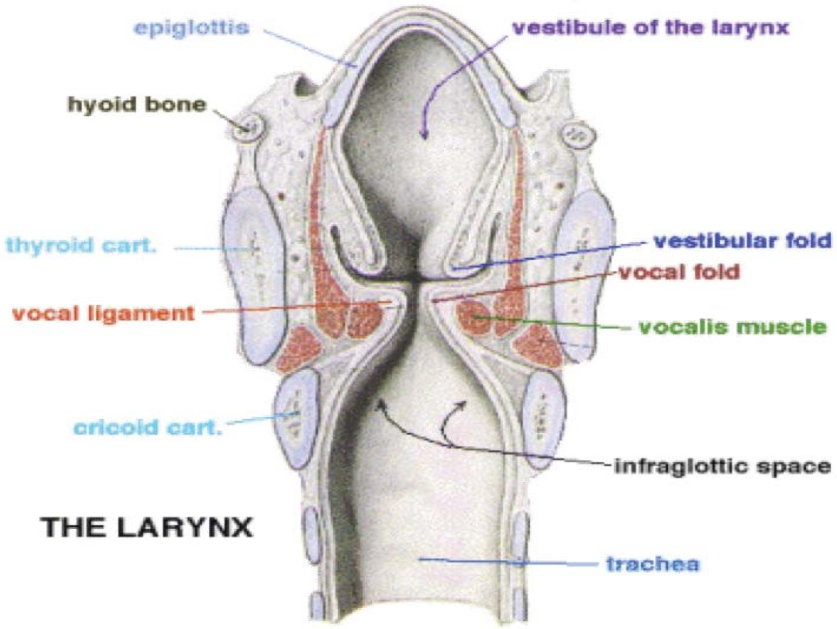
Between the upper membrane (quadrangular m) and the lower membrane (triangular m.) there is a very weak area which is not covered by any membrane we call it the ***ventricle*** .



LARYNGEAL MUCOSA :

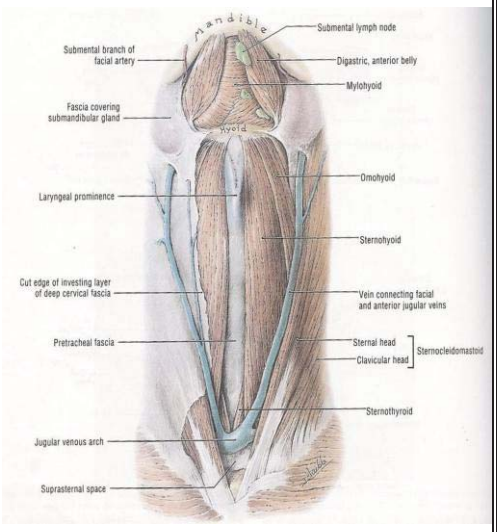
- All mucosa from trachea to aryepiglottic fold ►**ciliated columnar epithelium**.
- Except vocal cord and aryepiglottic fold ►**squamous epithelium.**
- **Commonest tumor in larynx: Squamous Cell Epithelium**

Cavity of Larynx:



The area between the Vocal Cords is Glottis
The area above the Vocal Cords is Supraglottis or Epiglottis
The area below the vocal Cords is Subglottis

LARYNGEAL MUSCULATURE:



- **Extrinsic depressors: (C1-C3)** attachment to sternum

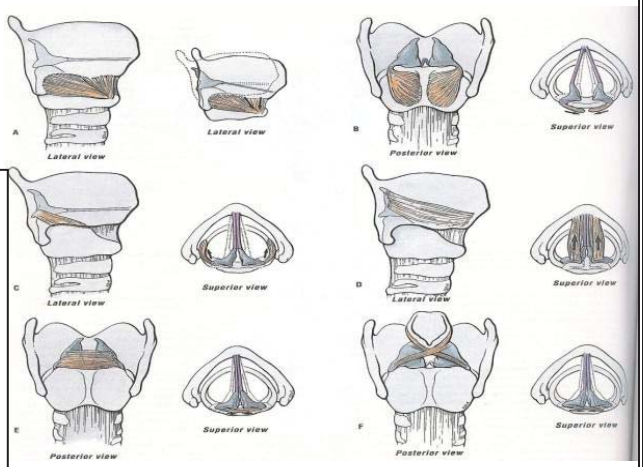
  Sternohyoid sternothyroid thyrohyoid, omhyoid.

- **Extrinsic elevators:** attachment is above

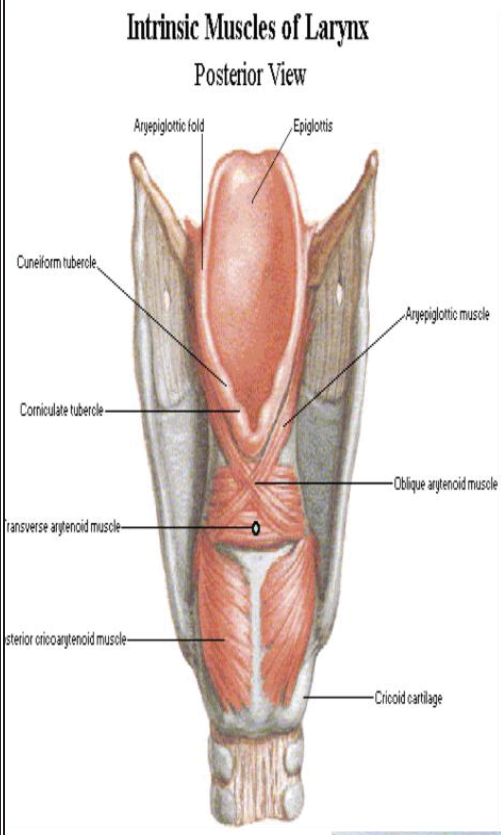
  Genohyoid (C1), diagastric (CNV-CNVII) mylohyoid (v) stylohyoid (VII).
  Used in swallowing.

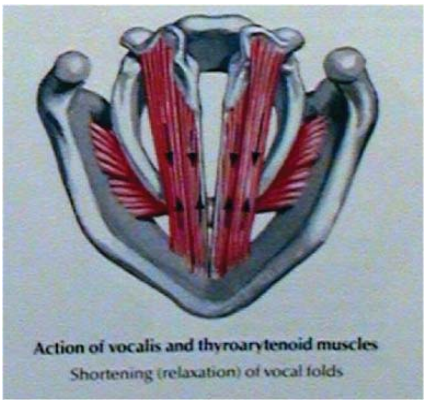
3. Intrinsic musculature:

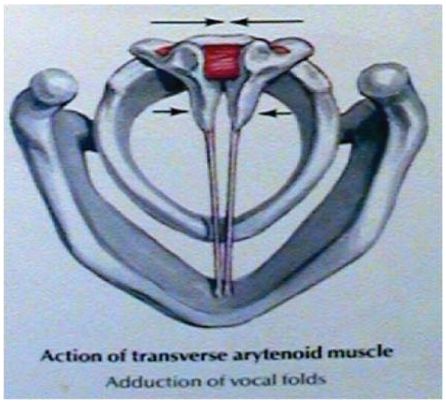
Abductors: (breathing)
posterior cricoarytenoid (PCA)
Adductors: (talking)
thyroarytenoid (TA) ,lateral cricoarytenoid (LCA) ,cricothyroid, interarytenoid .

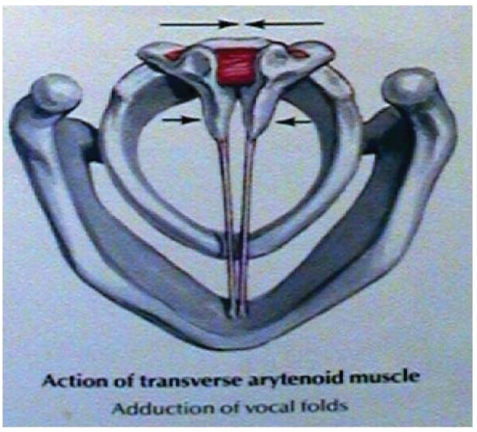


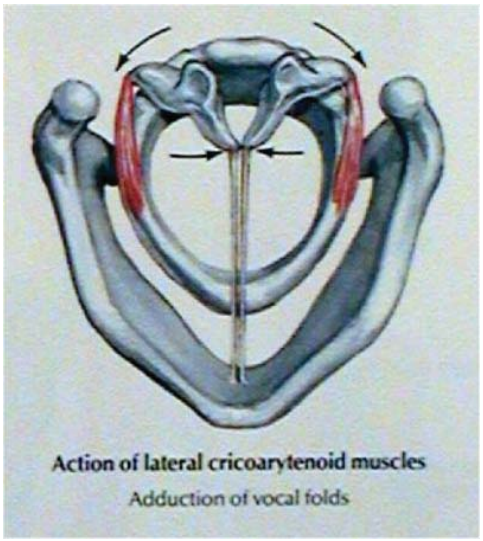
Vocal cords have 2 movements:
Adductors (4muscles) are used for speaking. **MCQ**
Abductor (1muscle) used for breathing . Located Posteriorly **MCQ** .
Cricoidthyroid is an adductor ms. but mainly it is responsible for the Vocal Cord tension **MCQ** of the vocal cords and supplied by the Superior Laryngeal Nerve (SLN).

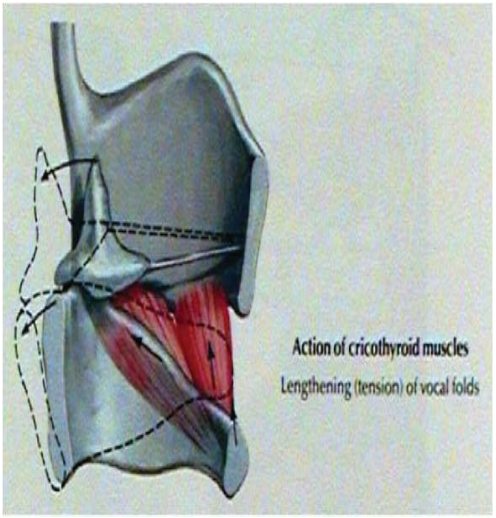

Intrinsic Muscles of Larynx
Posterior View
Aryepiglottic fold
Epiglottis
Cuneiform tubercle
Aryepiglottic muscle
Corniculate tubercle
Oblique arytenoid muscle
Transverse arytenoid muscle
Posterior cricoarytenoid muscle
Cricoid cartilage

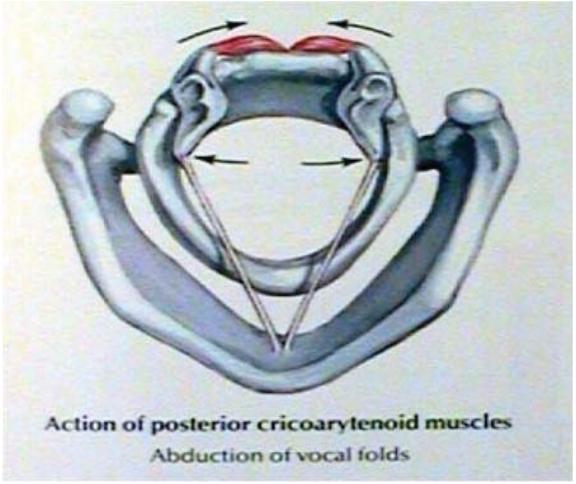

Action of vocalis and thyroarytenoid muscles
Shortening (relaxation) of vocal folds


Action of transverse arytenoid muscle
Adduction of vocal folds


Action of transverse arytenoid muscle
Adduction of vocal folds

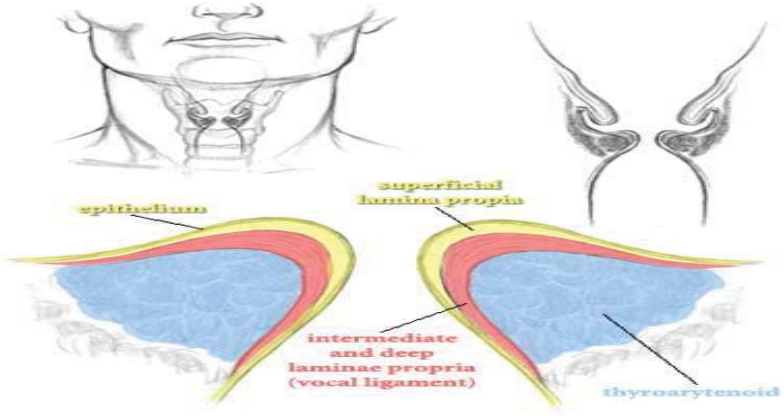

Action of lateral cricoarytenoid muscles
Adduction of vocal folds


Action of cricothyroid muscles
Lengthening (tension) of vocal folds


Action of posterior cricoarytenoid muscles
Abduction of vocal folds

HISTOPATHOLOGY:

Vocal cord layers



Histology:

1- Squamous epithelium

2- Lamina propria

1. **superficial layer Reink's space:** little collagen, fibrous tissue and elastin
2. **Intermediate layer:** more fibers and elastin it gives the whitish color
3. **Deep layer.**

– **Intermediate + deep layers =vocal ligament** (the 2 layers are attached to each other)

3- Vocalis (thyroarytenoid muscle)

Blood Supply:

– Superior and inferior laryngeal arteries and veins.

Lymphatic Drainage:

– Above vocal cord ► Upper deep cervical lymph node.

– Below vocal cord lower ►lower deep cervical node

Vocal Cords have no lymphatic drainage, So when the patient has vocal cord carcinoma he won't have metastasis unless it goes supraglottic or subglottic he can start to have metastasis from there.

Any smoker should undergo vocal cord carcinoma screening.

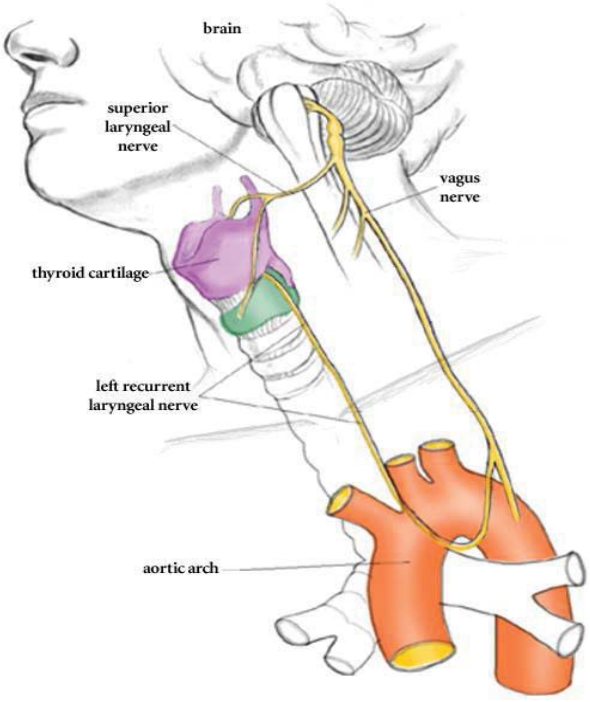
Lymph nodes drain into cervical lymph nodes so any patient that comes with neck mass especially painless you should consider lymphoma.
History: onset and duration, URTI, complete head and neck examination

Nerve supply:

Vagus gives 2 branches:

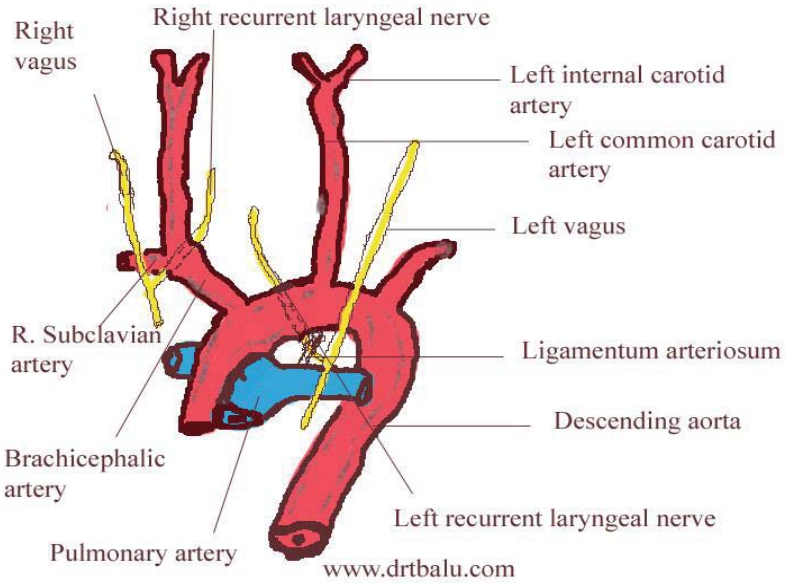
1. **Superior laryngeal nerve**
   - Internal branch (sensory) +superior laryngeal artery.
     - External branch (motor) ►**cricothyroid muscle only**
2. **Recurrent laryngeal nerve**
   - RT side: crosses the subclavian artery
   - LT side: arises on the arch of the aorta deep to ligamentum arteriosum (left is longer)

   It is divided behind the cricothyroid joint
   - Motor ►**all** the intrinsic muscles except the cricothyroid
   - Sensory



SLN: sensation above the vocal cords, chocking means (they are working well, good sensation).
RLN: sensation below the vocal cords. Left course is longer than the right course.

However, vocal cord paralysis might be caused by:
(thyroidectomy is the most common), brain tumor, vocal cord tumor, thyroid tumor, esophageal, mediastinal.. tumors compressing the nerve, Iatrogenic causes: cardiac thoracic surgery.. or idiopathic: waking up in the morning sounding weird.)
Most common is to have left vocal cord paralysis due to the long course of the left recurrent laryngeal nerve.



Pediatric Airway Anatomy:

- The neonates are obligate nasal breathers until 2 months. (they can't breathe from their mouth)
- The epiglottis at birth is omega Ω shaped
- The infants have high larynx C1-C4

APPLIED PHYSIOLOGY OF THE LARYNX:

1. Protection of the lower air passages
2. Respiration
3. Phonation

1. Protection of the lower air passages:

- Closure of the laryngeal inlet
- Closure of the glottis
- Cessation of respiration
- Cough reflex (forced expiration is made against a closed larynx

Closure of the airways during swallowing the bolus.

2. Phonation:

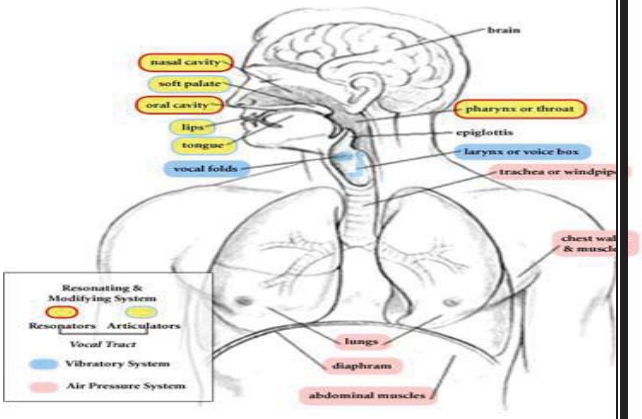
- Voice is produced by vibration of the vocal cord
- Source of energy is the airflow (good lung --> good voice)
- Normal vocal fold vibration occurs vertically from inferior to superior
- The mouth ,pharynx ,nose ,chest (are resonating chambers)

COPD , Smoker → low air amount → low vocal vibration → dysphonia

We speak at the end of expiration so that air comes out of the lungs, through the trachea, and into the larynx. The air makes the vocal folds vibrate. So we need normal air and mucosa not thick secretions or masses or infections or allergic rhinitis closing the resonating chambers.
When the vocal folds vibrate, they alternately trap air and release it.
Each release sends a little puff of air into the pharynx; each puff of air is the beginning of a sound wave
The sound wave is enhanced as it travels through the pharynx; by the time it leaves the mouth, it sounds like a voice.
The mouth, pharynx, nose, and chest which all should also be normal.
Tongue is important for articulation of the voice.

3. Respiration:

Vocal cord in abduction position



VOICE MECHANISM:

Speaking involves a voice mechanism that is composed of three subsystems:

- Air pressure system
- Vibratory system
- Resonating system

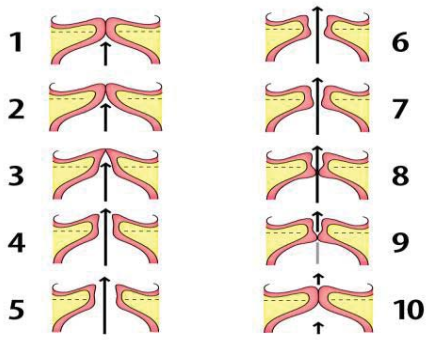
The "spoken word" result from three components of voice production:

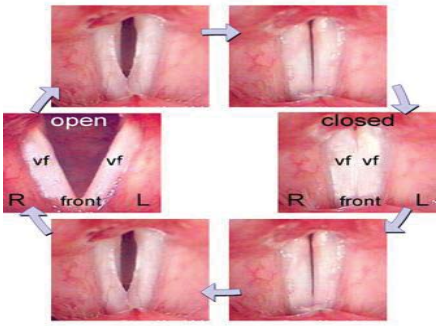
❖ Voiced sound, resonance, and articulation

- **Voiced sound:** the basic sound produced by vocal fold vibration "buzzy sound"

- **Resonance:** voiced sound is amplified and modified by the vocal tract resonators (throat, mouth cavity, and nasal passages)
- **Articulation**: the vocal tract articulators (the tongue, soft palate, and lip) modify the voiced sound.
    - Vocal fold vibrate rapidly in sequence of vibratory cycles with a speed of about:
    - 110 cycles per second (men)= lower pitch
    - 180 to 220 cycles per second (women)=medium pitch
    - 300 cycles per second (children)= higher pitch
    - Louder voice : increase in amplitude of vocal fold vibration

Vocal cord vibration: Bernoulli Effect





Air comes from the lung opens the lower lip then the middle then the upper lip. Cannot be seen by direct visualization (very fast -300 cycle). Can be seen by stroboscope.

Laryngeal sphincters:

- True vocal cord
- false vocal cord
- Aryepiglottic sphincter

Spasm during swallowing

Aryepiglottic fold is between the epiglottis and arytenoid. If it is short then the epiglottis will always be covering the airway.

EVALUATION OF THE DYSPHONIC PATIENT:

HISTORY

Dysphonia (hoarseness)

Onset , duration ,severity , URTI ,fever ,cough ,(voice abuse (job) ,tobacco or alcohol), dysphagia ,aspiration , breathing difficulty(stridor) ,weight lost ,GERD ,trauma , previous surgery neck mass. laryngopharyngeal reflux (clearing your throat-it is very bad) problem is posteriorly
(Occupation and medication are important)

EXAMINATION:

Complete ENT examination

Laryngeal examination and voice assessments :

4. Indirect laryngoscope (mirror)
5. Direct laryngoscope
6. Fibreoptic flexible scope
7. Stroboscopy
8. Acoustic analysis
9. Cranial nerves (tumors might be compressing the involved nerves)
10. neck examination

Always say in the OSCE that you want to examine the cranial nerves.

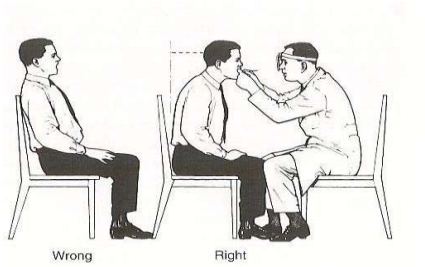
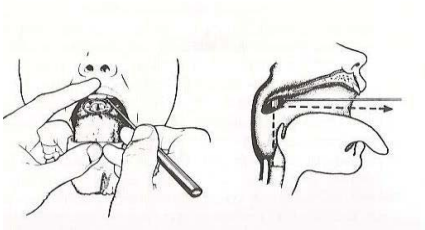
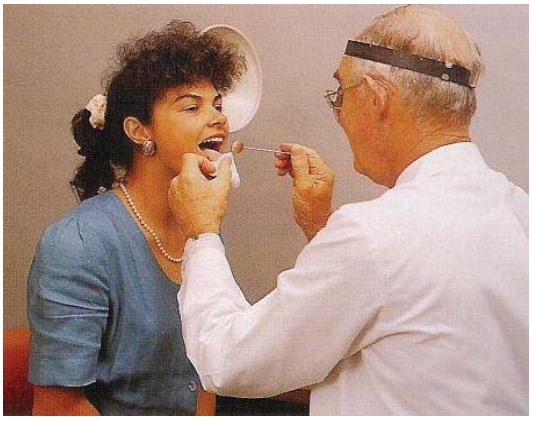


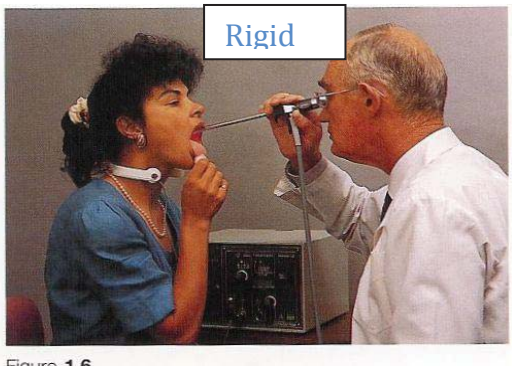
Figure 1.3
Position of the patient's head and neck for indirect laryngoscopy to create the best angle for a comprehensive view of the laryngeal structures.

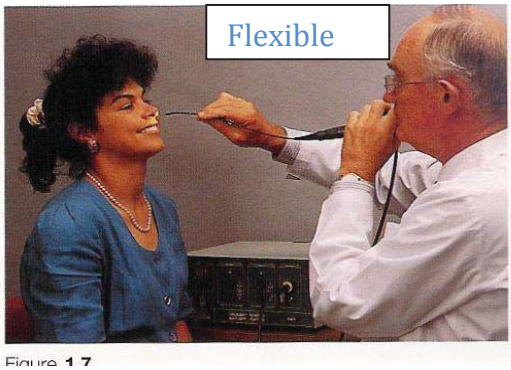




Indirect laryngoscope

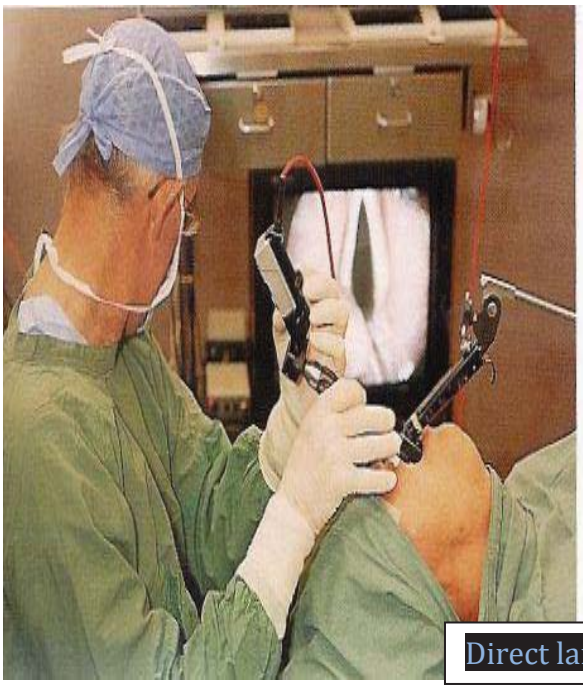
Nasal polyp surgery → FESS
Vocal Cord polyp nodule → microlaryngoscopy
Deviated septum → septoplasty

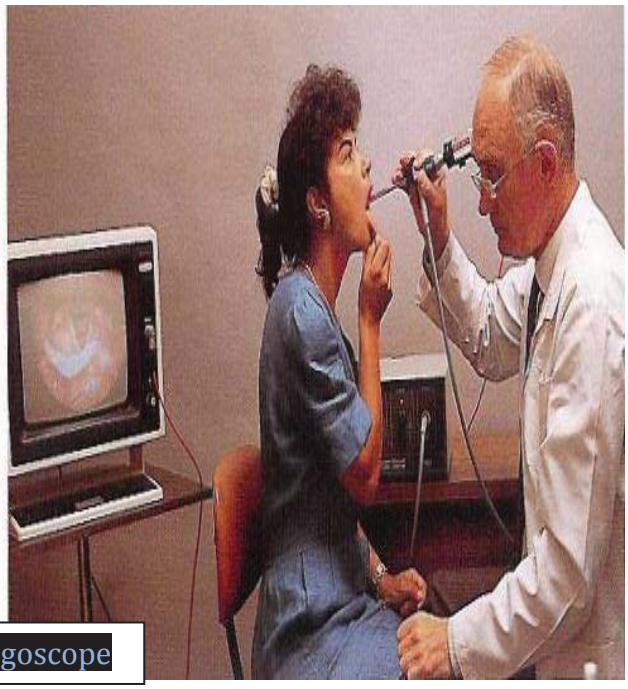

Rigid

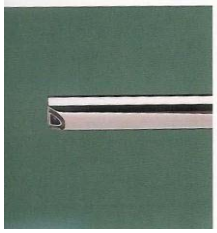

Flexible

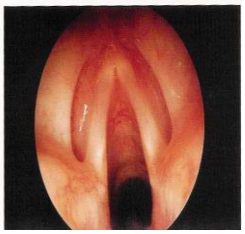
In Exam : mention 2 indications of the flexible fibre optic always:
Children and Gag reflex (GERD)

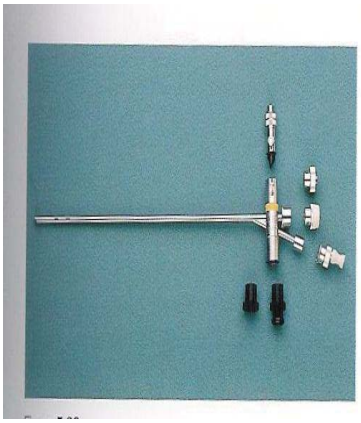
In the rigid nasopharyngoscope :
Nose use the angle of (0-30) degrees.
Looking down use (70-90) degrees.




Direct laryngoscope

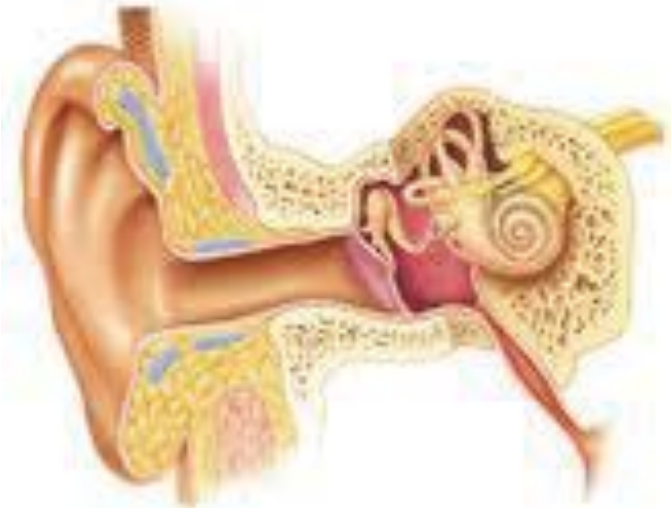






Long- horn is called broncoscopy
Indications: foreign body removal, biopsy, washing trachea and visualization.

Diseases of the Ear, Nose and Throat



Hadeel AlMadany

1st Lecture:

Airway Obstruction

Done by: Seham Alarfaj

The slides were provided by doctor (Manal Bukari)
Important Notes in red
Copied slides in black
Your notes in green/ blue
Titles and subtitles in this color
Highlight possible MCQs mentioned or pointed by the doctor

Objectives:

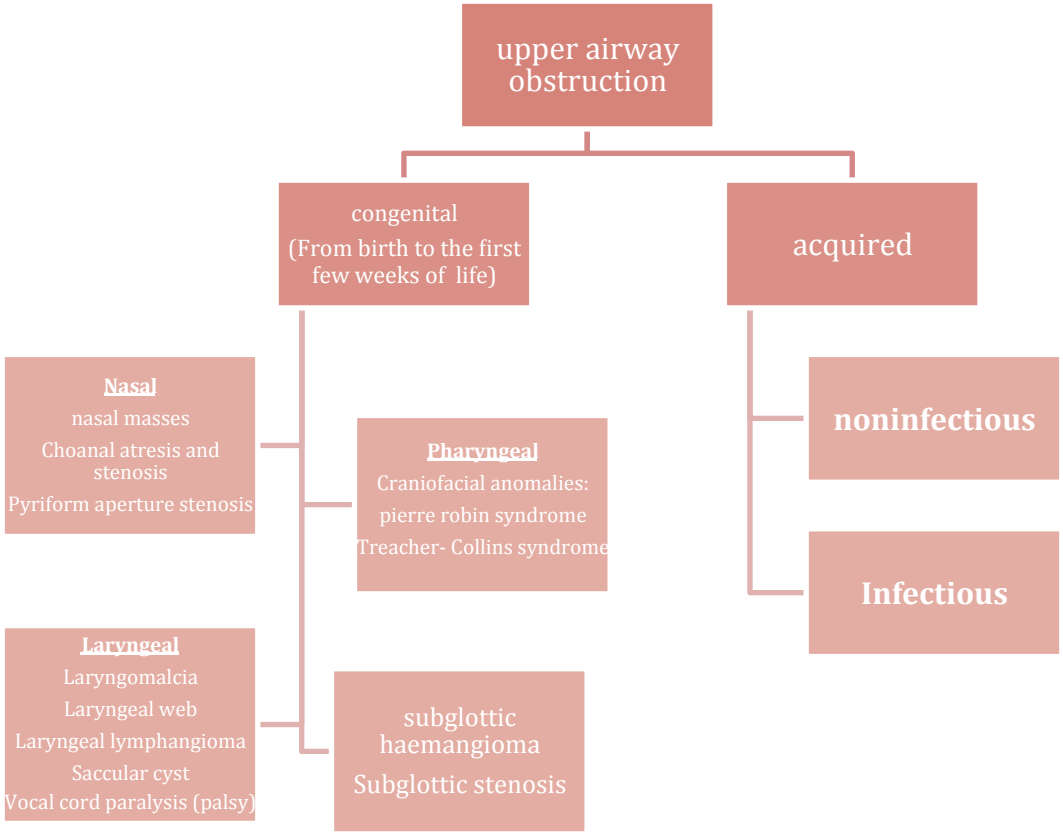
Airway Obstruction I:

- Causes of airway obstruction (congenital and acquired)
- Signs and symptoms

Airway Obstruction II:

- Investigation of airway obstruction
- Radiology illustration
- Medical and surgical treatment
- Operations (indication, procedure and complication) tracheostomy, cricothyroidectormy, intubation, choanal atresia repair etc..

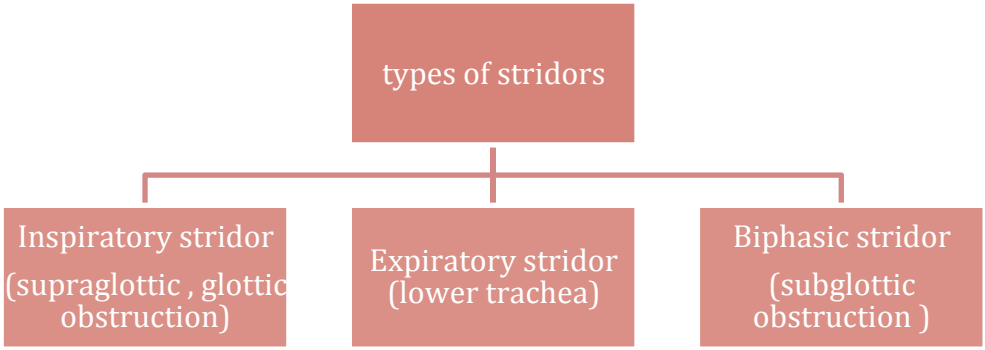
The Upper airway extended from the nares and lip to the subglottic area



Q: 3 years old child who has stridor for 3 weeks, what is stridor? And what's the difference between it and snoring?

Stridor is a high-pitched wheezing sound resulting from turbulent airflow in the upper airway. Stridor is a physical sign, which is produced by narrowed or obstructed airway path

Type of stridor:



e.g: inspiratory stridor in started after birth think about laryngomylacia
Biphasic stridor in started after birth: subglottic stenosis, subglottic hemangioma

Snoring: is low pitch sound caused by tissue vibration of the nasopharynx pharynx and soft plate due to obstruction above the larynx.

Nasal obstruction:

Neonates are obligatory nasal breather
In neonate Cyanosis improved with crying but worsens on feeding (cyclical cyanosis)

1. Nasal masses:

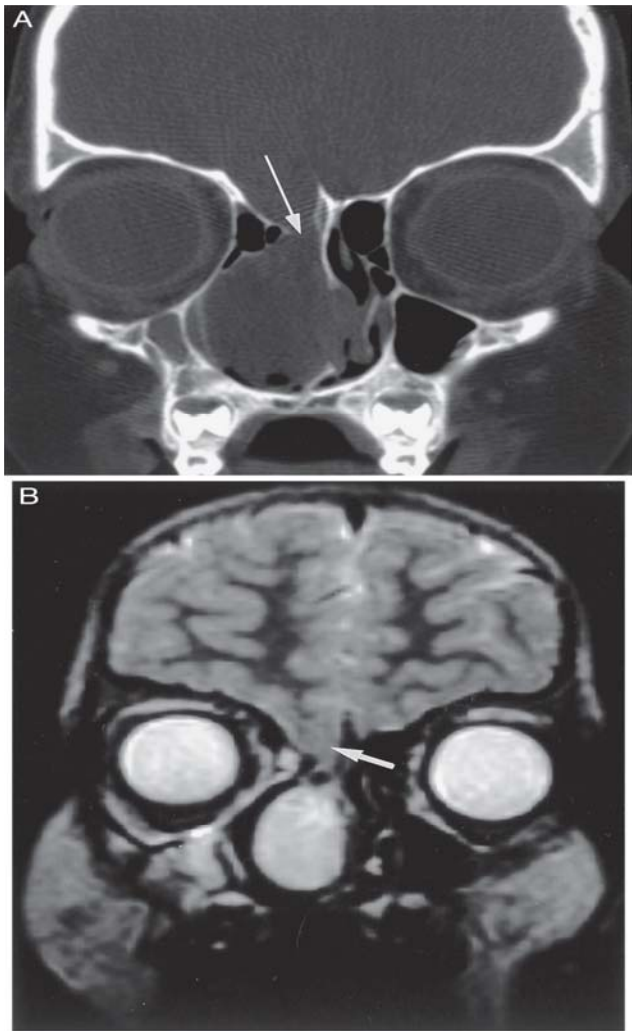
Types:

- Cystic (meningoencephalocoele,)
- Solid (glioma, hamartoma ,teratoma ,and lymphangioma)

Diagnosis: CT scan and MRI (better in visualizing soft tissue)
Don't take biopsy before imaging studies

Cystic	Solid
Meningoencephalocele	Haemangioma
Meningocele	Neurofibroma
Dermoid cyst	Glioma
Epidermoid cyst	Lymphangioma
	Neuroblastoma
	Craniopharyngioma
	Rhabdomyosarcoma
	Chordoma

Differential diagnosis: dermoid, meningoenchephalocele



A: coronal CT scan showing homogenous mass in the right nostrils (arrow)
B: MRI shows communication
(Homogenous = all the same color)
Every exam contain CT scan sinus (homogenous opacification of sinus = fluid, polyp)
(Heterogenous with spiking = fungal or malignancy)

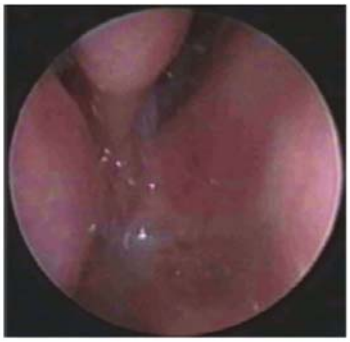
2. Choanal atresia:

- Lack of patency of posterior nasal aperture
- Bilateral atrasia presents soon after birth with sever respiratory distress
- Unilateral atrasia may undiagnosed until later in childhood (rhinorrheoa)

Q: What is the commonest cause of unilateral nasal obstruction and nasal discharge in pediatric?
A: Foreign body (purulent, foul smelling discharge)

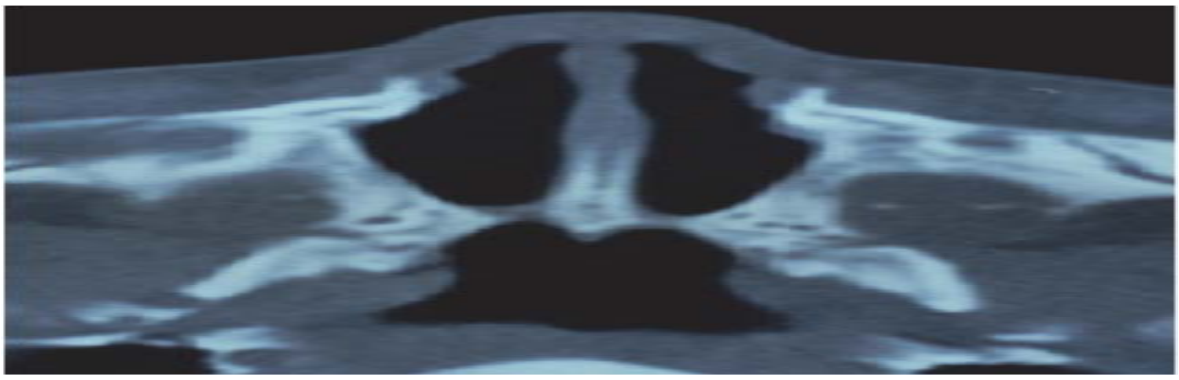
Types:

1. Membranous 10%
2. Bony
3. Mixed

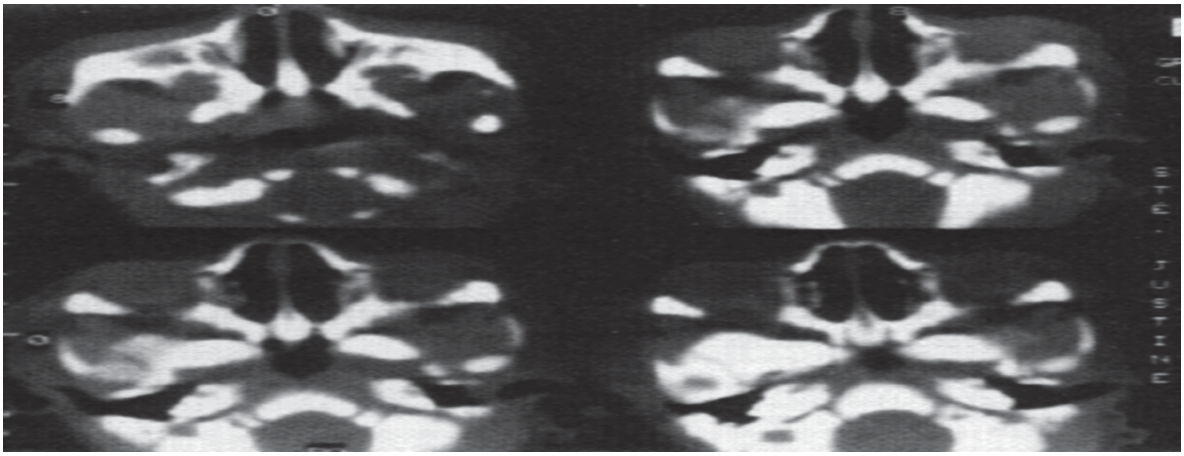


Diagnosis:

- Cyanosis improved with crying
- Inability to pass size 6 French catheter
- Do CT scan to differentiate between the subtypes



A 3 wks old baby presented with cyanosis and difficulty breathing since birth
An axial CT scan showing bilateral membranous choanal atresia (membranous=grey, white=bone)



An axial CT scan showing bilateral mixed choanal atresia (not important to pick the subtype from Ct scan as long as you can identify the choanal atrsia)

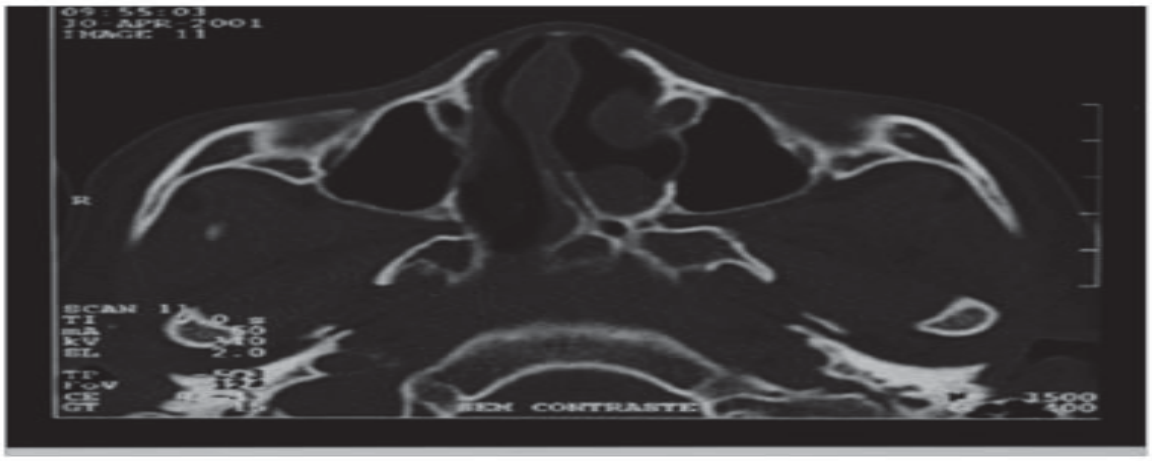
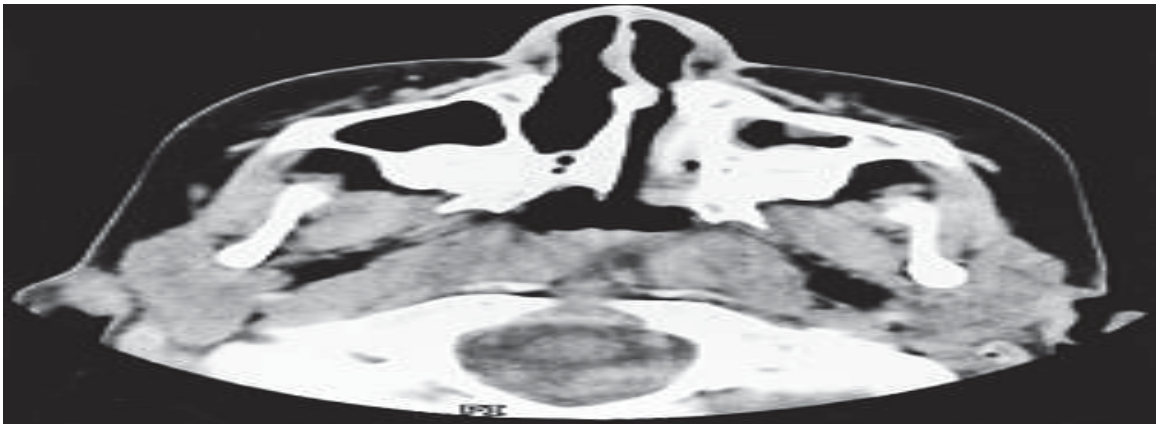


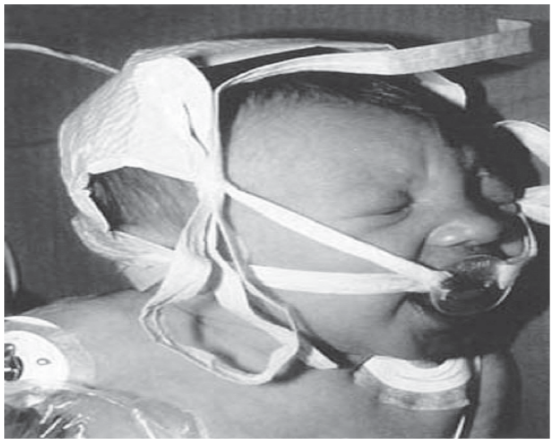
Figure 1. A case of left side choanal atresia and symmetrical maxillary sinuses and no sinus disease.



An axial CT scan showing unilateral bony choanal atresia

70% of choanal atrasia associated with CHARGE:

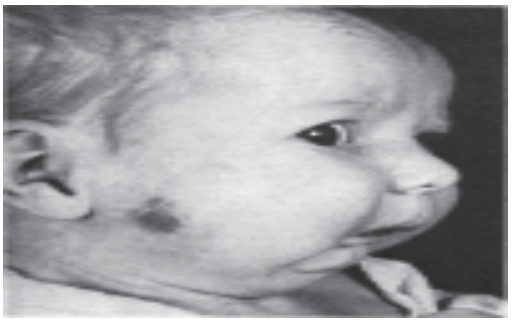
- C: coloboma (is a hole in one of the structures of the eye, such as the iris, retina, choroid or optic disc)
- H: heart disease
- A: atrasia
- R: retarded growth
- G: genital hypoplasia
- E: ear deformity



Treatment:

- Emergency treatment is by insertion of oral tube
- Surgical treatment is by either transnasal or transpalatal choanal atrasia repair (different techniques but now a days it is done endoscopicaly)

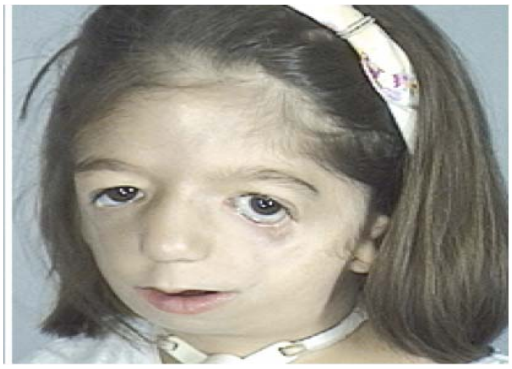
Pharyngeal obstruction:



Craniofacial anomalies:

1. Pierre –Robin syndrome:

Glossoptosis (airway obstruction caused by backwards displacement of the tongue base), micrognatheia (small lower mandible) (causes narrowing of the airway), cleft palate



2. Treacher- Collins syndromes:

(Mandibulofacial Dysostosis is a disorder of the development of bone, in particular affecting ossification.)
Narrow nose high arched palate

If the baby was born with a syndrome sometimes he needs intubation or even tracheostomy

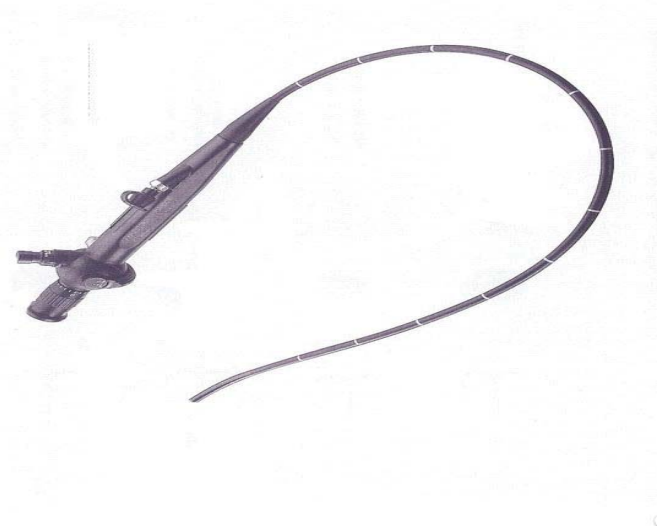
Laryngeal:

1. Laryngomalcia:

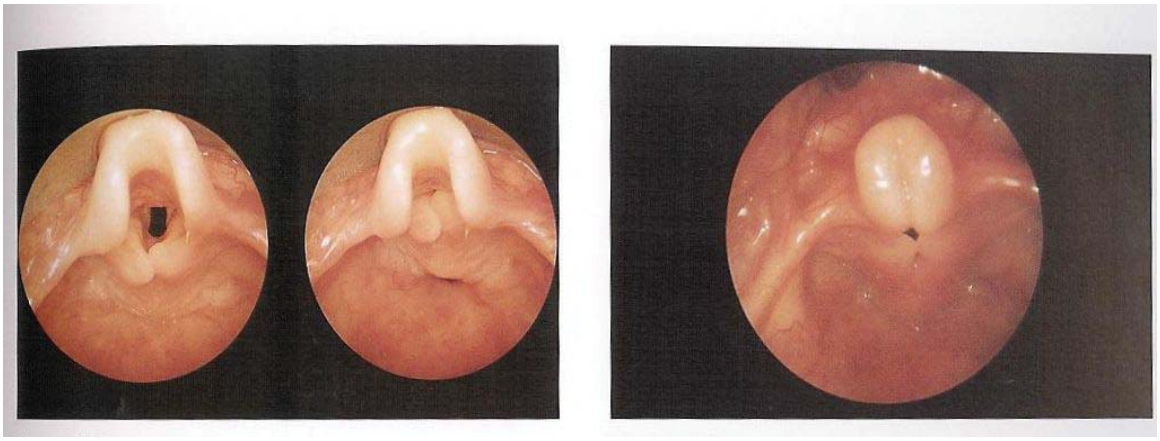
The most common cause of congenital airway obstruction
The most common cause of stridor in infancy
Cause: immaturity of the cartilage, associated with inward and forward arytenoid when child breaths with short aryepiglottic folds.
Symptoms:

- Stridor in the first weeks of life in the inspiratory phase
- Worse with crying, feeding, and respiratory tract infection (leading to failure to thrive)
- Improved in prone position

Diagnosis: flexible fibrotic endoscopy while the child is awake to see the pattern of breathing



Name this instrument and its indications?
Flexible fibroptic
Examination of nose, nasopharynx, oropharynx, hypopharynx, biopsy



Endoscopic finding:

- ❑ Tall ,omega shape epiglottis
- ❑ Inward forward movement of arytenoid mucosa (sucked)
- ❑ Short aryepiglottis fold

Treatment:

Mild cases: (no cyanosis and not effecting the child growth):

Observation spontaneously subside by 12 to 18 months in 90% of cases

Sever cases:

1. supraglottoplasty ,
2. Tracheostomy

2. Vocal cord paralysis:

Nerves that supply the vocal cords are superior and recurrent laryngeal nerves

Can be unilateral or bilateral ,congenital or acquired

Congenital form may associated with abnormality of the central nervous system (Arnold Chiari syndrome) or cardiovascular anomalies

Symptoms:

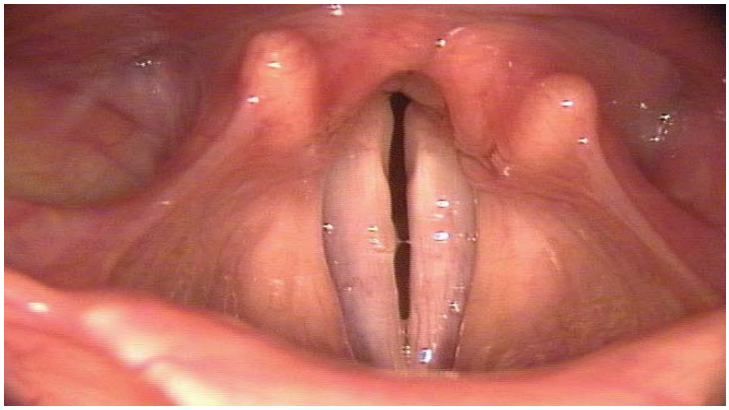
High pitch inspiratory stridor (level of glottis)

Treatment:

Tracheostomy in sever cases

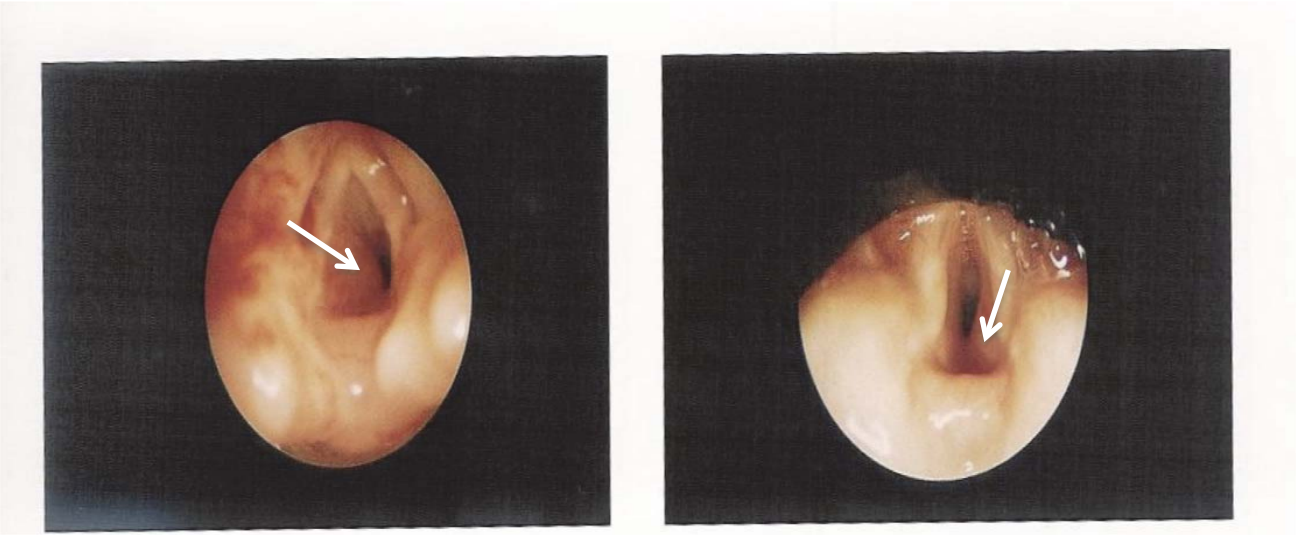
Spontaneous recovery occurs in half patients

Surgical intervention postponed until the patient become old **Vocal cord lateralization,** arytoidectomy and laser cordotomy



False vocal cord (vestibular fold, ventricular fold) between the true and false vocal cords we have a groove called (vestibule or saccule)

3. Subglottic haemangioma:



Congenital vascular lesion

Not present at birth but grow rapidly over the first few months of life

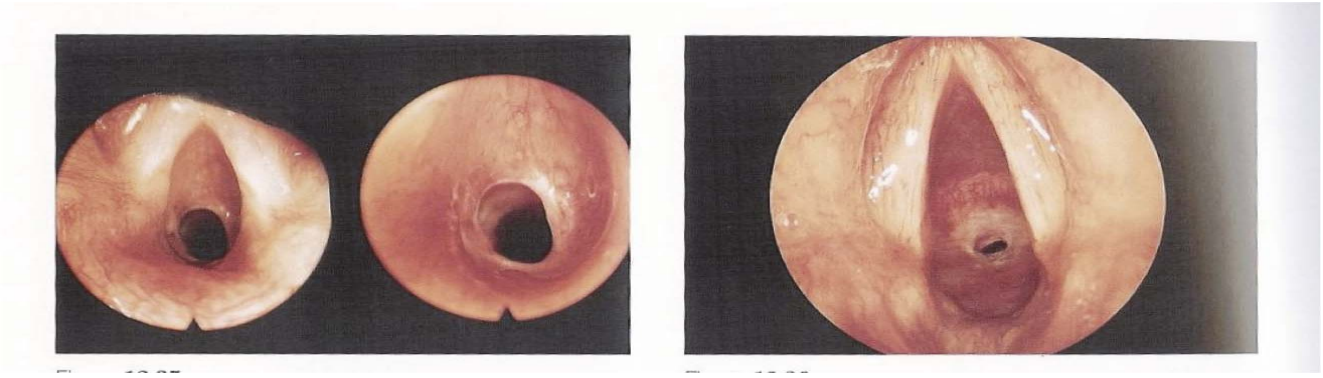
Symptoms:

- Biphasic stridor
- Tend to involute slowly after one year
- 50% of the patients have cutaneous haemangioma in the head and neck

Treatment:

Systemic steroid, interlesional steroid, **Propranolol (new treatment),** laser ablation tracheostomy if not improving

4. Congenital subglottic stenosis:



Subglottic area is the narowest area in the airway (cause it is at the level of cricoid cartilage which is a complete ring), stenosis if the diameter less than 4 mm in term infant

Symptoms: depend on the degree of stenosis

- Biphasic stridor
- Respiratory distress
- Recurrent/prolonged croup

Diagnosis:

- Bronchoscopy: to grade the stenosis
- plain xray HKV (high kilo voltage x-ray: start with lateral x-ray to see narrowing of the air column)

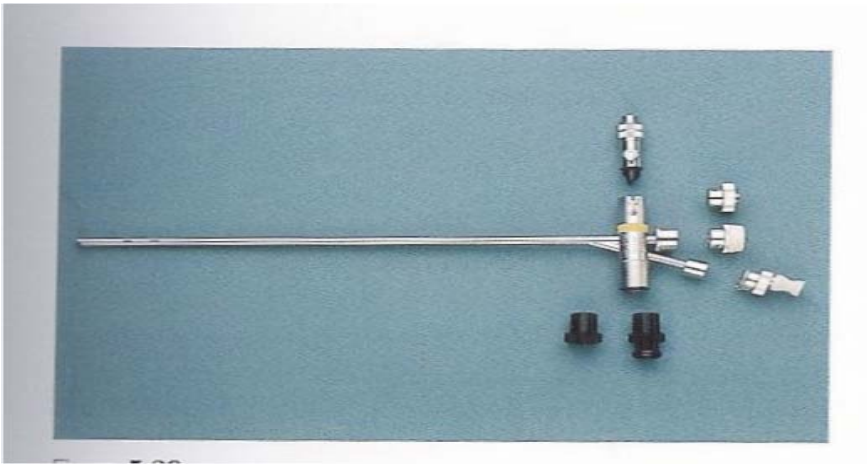
New grading system starts from 70%:
Grade I: 70%
Grade II: 70-90%
Grade III: 91-99%
Grade IV: no detectable lumen

- The maximum percentage of airway obstruction is determined and assigned a grade:
- Grade I <50% obstruction
- Grade II 51-70% obstruction
- Grade III 71-99% obstruction
- Grade IV no detectable lumen

Treatment:
Depend on the degree of stenosis:
Grade I&II: endoscopic balloon dilatation

- Tracheostomy
- Laser excision (if soft tissue)

Grade III&IV: Laryngotracheal reconstruction (LTR) (if cartilage)
Criocotracheal resection (CTR)



Instrument's name: bronchoscope
Indications: foreign body removal, biopsy

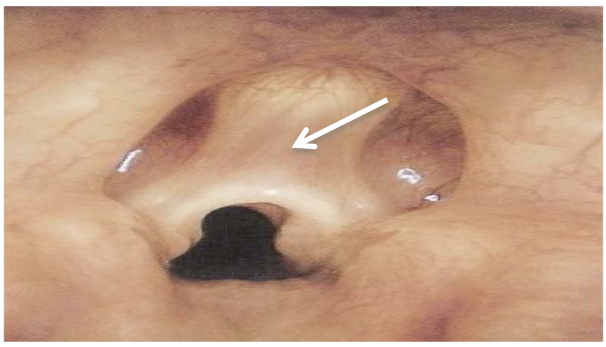
5. Laryngeal web:

Symptoms depend on the grade of stenosis:

- ❑ Week cry
- ❑ Stridor

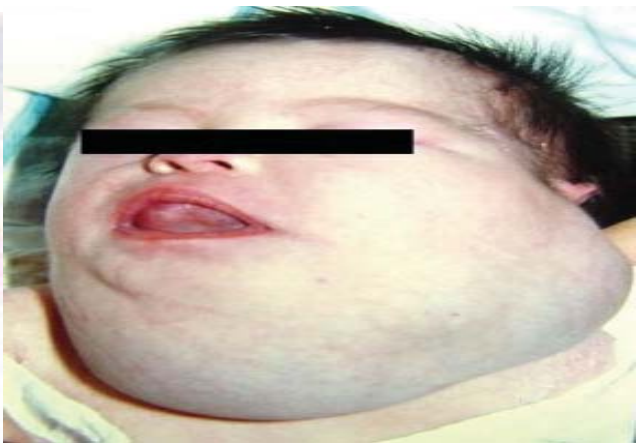
Treatment: **endoscopically**

- ❑ Laser excisions
- ❑ Trachestomy



Extratracheal compression:

1. Cystic hygroma (lymphangioma)



Definition: lymphatic malformation arising from vestigial lymph channels of neck
Clinical features:

- Usually present by age 2
- Thin walled cyst extending from floor of mouth to mediastinum, in posterior triangle or supraclavicular area
- Painless, soft, compressible
- Infection causes a sudden increase in size

Diagnoses: intranatally by ultrasound
Treatment: surgical excision (debulking) if it fails to regress- difficult dissection duo to numerous cyst extensions cystic hygroma is consisting of lobulated masses when they open one another one appear

Acquired upper airway obstruction

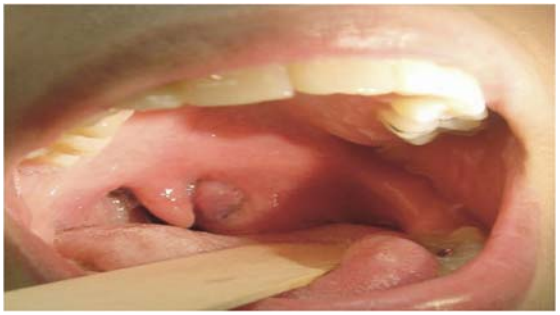
acquired upper airway obstruction are more common than congenital type
Subglottic area is the narrowest area
Causes:
Infectious

1. Peritonsillar abscess
2. Retropharyngeal abscess
3. Epiglottis
4. Croup
5. Bacterial tracheitis

noninfectious

1. FB aspiration
2. acquired vocal cord paralysis
3. Acquired subglottic stenosis
4. adenotonsillar enlargement
5. respiratory papillomatosis
6. malignancy
7. Angiodema
8. cautsic ingestion
9. trauma
10. laryngospasm

Q: patient have sore throat, fever, took antibiotics but didn't improve and it is getting worse with mass inside his throat, trismus
A: Peritonsillar abscess (quinsy)



1. Peritonsillar abscess (Quinsy):

Common deep infection in late childhood
Definition:
Cellulitis of space behind tonsillar capsule extending onto soft palate leading to abscess
Symptoms: low grade fever, sever sore throat, muffled voice (hot potato voice), drooling, trismus

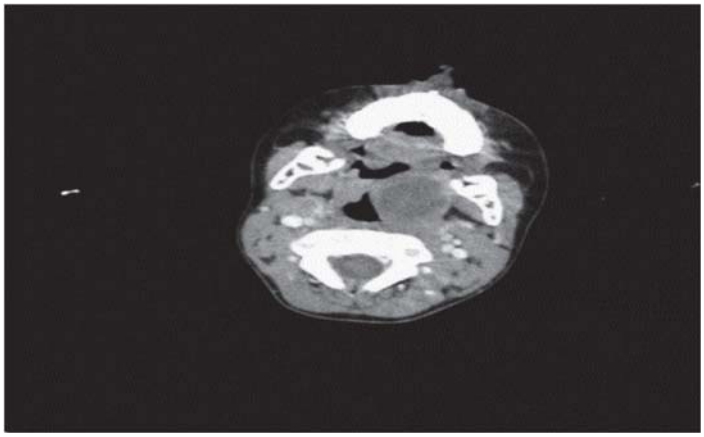
Diagnosis:

- Clinical diagnosis
- CT scan

Treatment:

- Aspiration
- Excision and drainge
- Later tonsillectomy
- IV ABX

The difference between tonsillitis and quinsy: in quinsy the soft palate bulge while in tonsillitis the tonsils themselves are enlarged



Axial CT scan the arrow shows a mass compressing the airway

Complication:

Retropharyngeal and parapharyngeal abscess

Parapharyngeal space is divided by styloid into two parts pre and post styloid. The post styloid contains cranial nerves and carotid artery.
Paraparyngeal abscess = neck mass
Retropharyngeal = intraoral mass

2. Retropharyngeal abscess:

Symptoms:

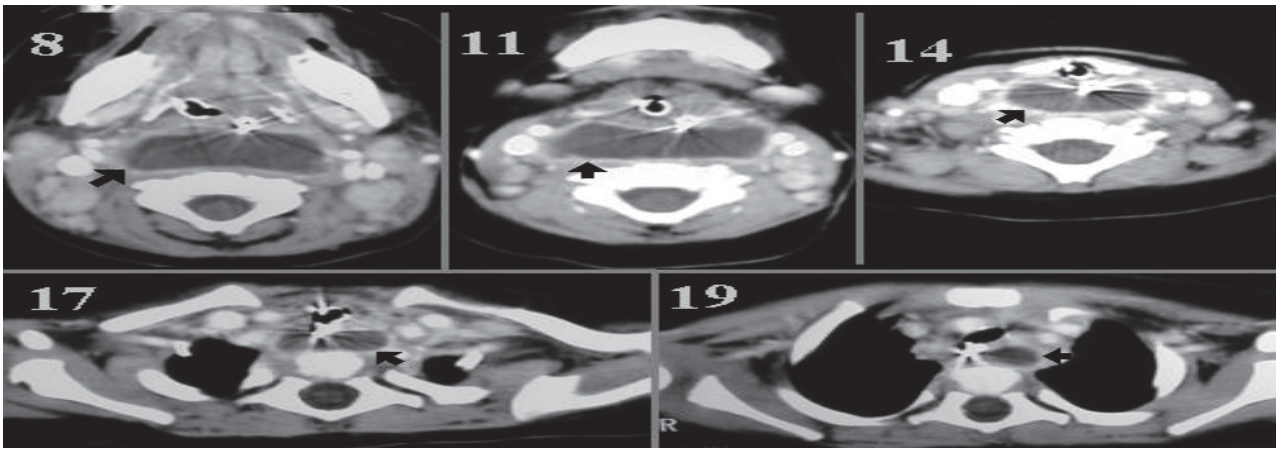
Fever, cervical adenopathy, stridor torticollis (cannot move his neck), drooling

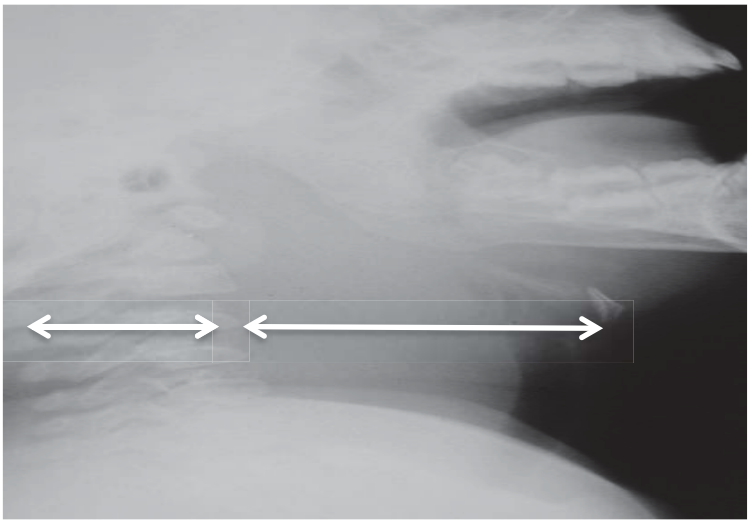
Causes:

Progressive pharyngitis S.aureus ,Haemophilus , group A beta haemolytic sterptococcus , bacteroides

Treatment:

- Transoral excision and drainge
- IV ABX
- INTUBATION
- Tracheotomy





lateral x-ray shows the diameter of the soft tissue is more than the diameter of the body of vertebra

Q: a child comes to the emergency with fever drooling of the saliva sitting in sniffing position
A: Epiglottitis:

3. Epiglottitis:

It is life threatening rapidly progressive condition

Cause: H influenzae typeB

Clinical features:

Any age most commonly: 2-7 years

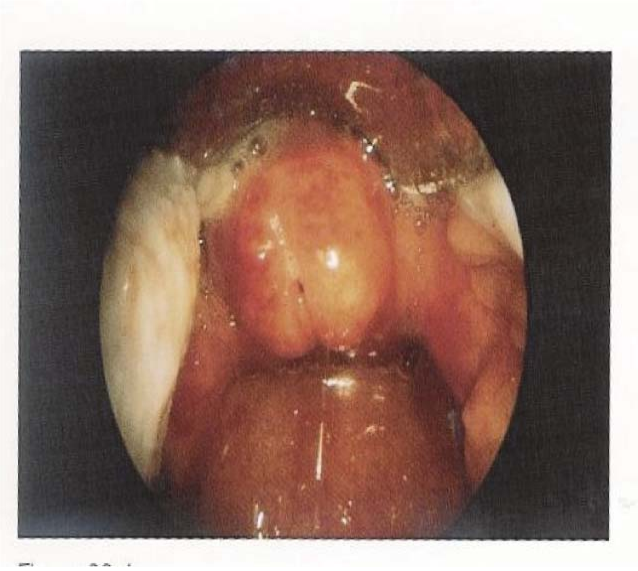
Rapid onset

High fever dysphagia, drooling, inspiratory stridor, toxic looking child (sit upright with head extended)

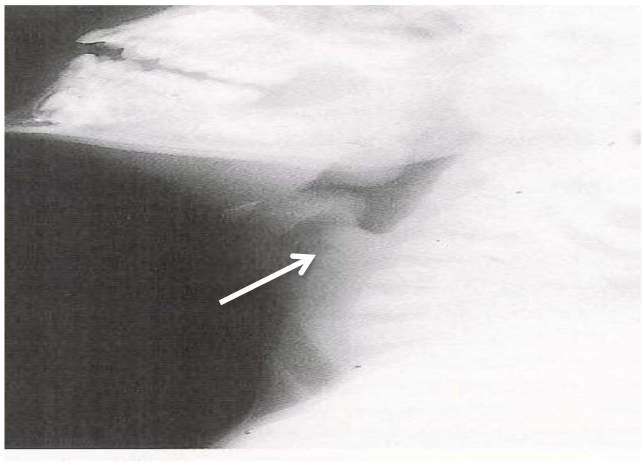
Thumb sign in lateral xray

Treatment: **NO EXAMINATION SHOULD BE DONE IN ER** take the patient to the OR maintain the airway:

- Intubation
- tracheostomy

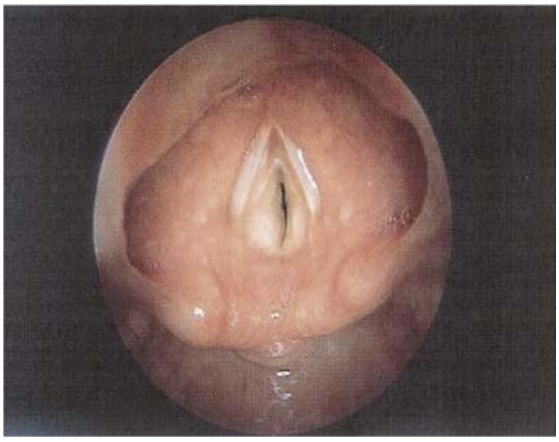


- Start IV ABX cause it is caused by bacteria unlike croup which is caused by virus

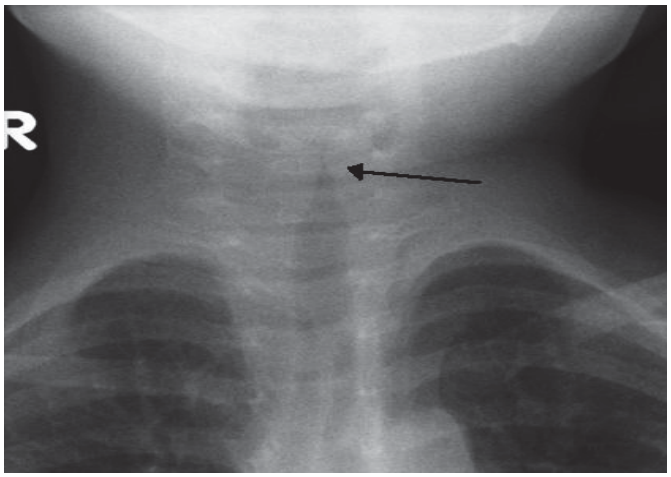


Lateral x-ray of head and neck shows thumb sign

4. Croup (laryngotracheobronchitis):



Inflammation primarily involves tissue in the subglottic space +/- tracheobronchial tree
Cause: Parainfluenza 1 (most common), 2, 3
Clinical features: age group: 6 months-3 years
Preceded by URTI symptoms
Generally occurs at night
biphasic stridor, fever , brasssy cough (loud, sea-lion bark) hoarseness , no dysphagia (3 S's: stridor, subglottic swelling, seal bark cough)
supraglottic area normal
if recurrent croup, think subglottic stenosis
Diagnosis:
steeple sign on AP x-ray of neck
Treatment:
Humidified oxygen, racmic epinephrine ,steroid



The **steeple sign** is a radiologic sign found on a frontal neck radiograph where subglottic tracheal narrowing produces an inverted "V" shape within the trachea itself.

Non infectious causes:

1. FB aspiration

Foreign body aspiration is a common accident in children and represents an important cause of morbidity and mortality.

500 children die of FBA each year in the USA and 40% of lethal accident among children under 1year of age were caused by FBA

FBA is common among infants and preschool children

Clinical presentations:

Acute episode: period of choking , gagging, wheezing ,hoarseness

Inhalation or aspiration: goes to airways while ingestion means it goes to esophagus

Ingested usually stuck at cricopharyngous while aspirated stuck at right mainstem bronchus

Asymptomatic (mis-diagnosis?)

Cough , wheezing …

Complications:

Pneumonia, obstructive emphysema and bronchiectasis….

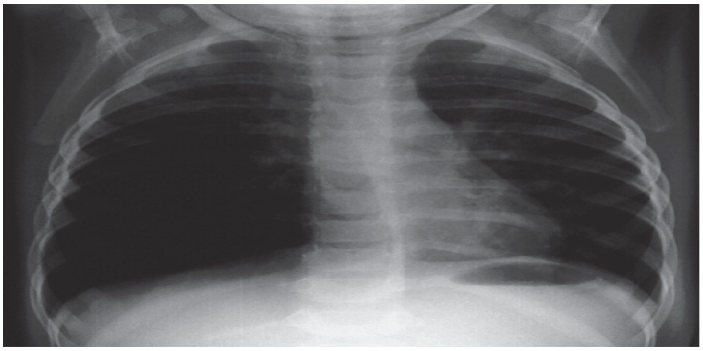
Medical history is the key to diagnosis FBA:

- Physical examination finding were abnormal in 80% of children with FBA , these were abnormal also in 40% of children without FBs (don't depend on examination).
- The sensitivity and specificity of physical examination were 80.4% and 59% respectively.

Radiological examination:

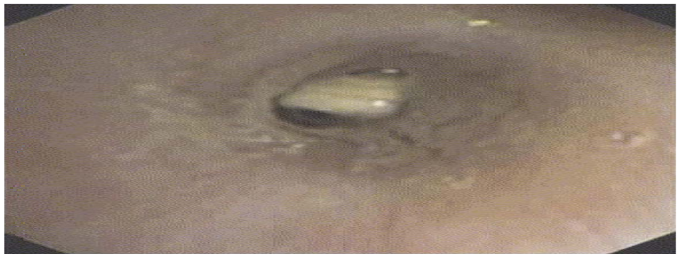
Many FB are not radiopaque and small FB may cause symptoms but not radiographic changes:

- Plain film (inspiratory expiratory)
- Air trapping,
- Obstructive emphysema, mediastinal shift
- Rt and LT lateral decubitus film



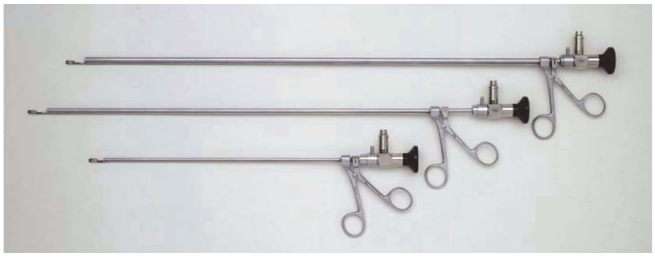
chest x-ray film shows air trapping

- The most common objects aspirated by young children are food products (peanuts ,seeds)
- Beans and seeds absorb water over time (organic things are more dangerous)
- Inert FB (Pieces of toys casuse less reaction)
- Batteries MUST be ruled out as foreign body as they are lethal and can erode into aorta

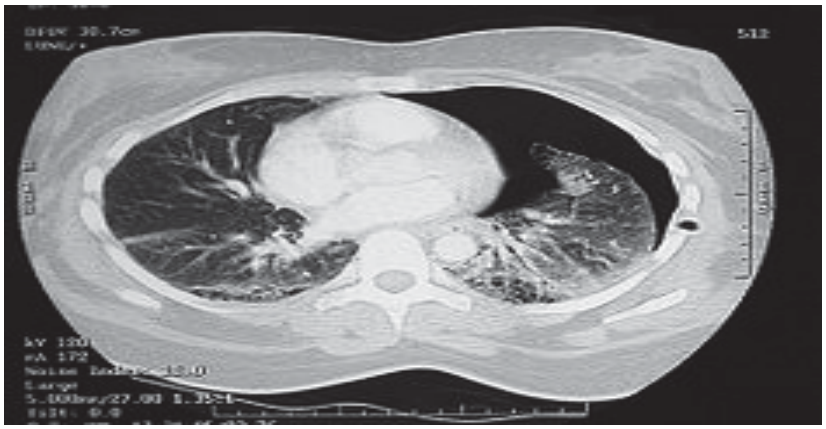


Bronchoscopy shows foreign body inside bronchus

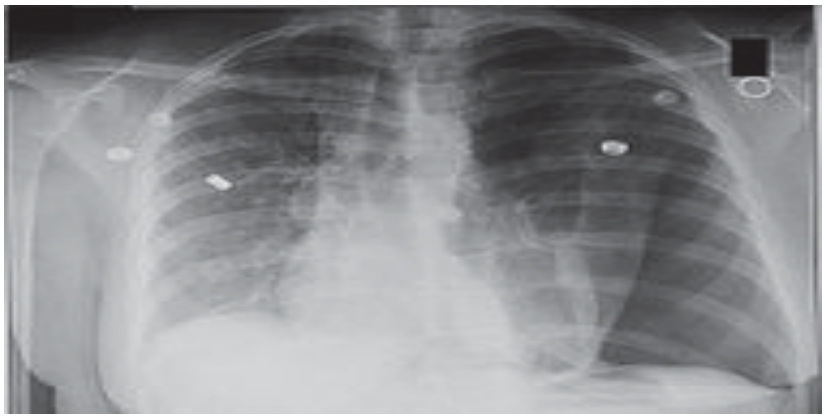
- Negative imaging studies do not exclude the presence of an FB
- **A High degree of clinical suspicion is the most important element in the diagnostic work –up**
- Airway foreign bodies are removed most safely under general anesthesia using the ventilating rigid bronchoscope



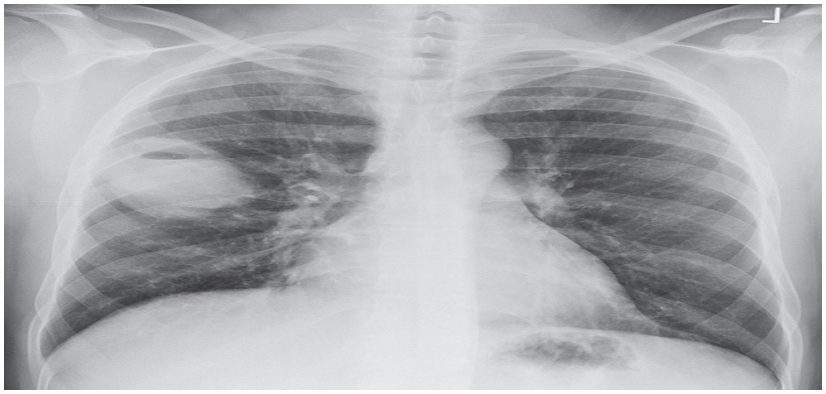
Bronchoscope with forceps: when removing the foreign body check the presence of other objects plus suction the secretion in that area.



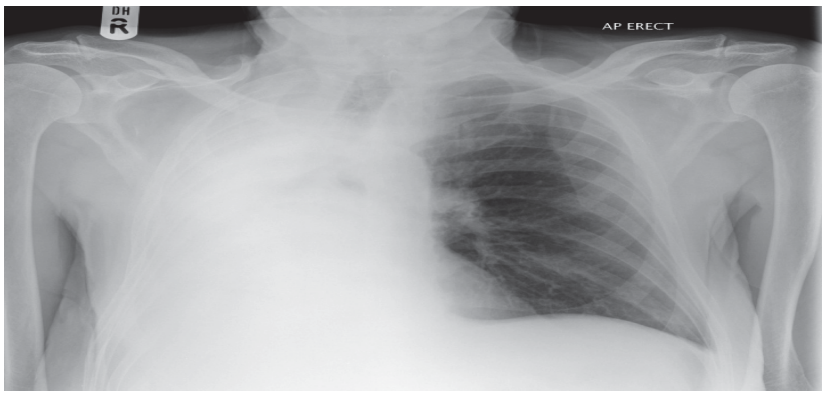
An axial chest CT scan shows pneumothorax



Chest x-ray of patient complaining of foreign body aspiration showing pneumothorax in the left lung



Chest X-ray shows abscess in the right lung



Chest x-ray shows atelectasis of right lung

2. Acquired vocal cord paralysis

- Unilateral
- Bilateral

Causes:

- Birth trauma
- (forceps delivery)
- Cardiac surgery(PDA repair)
- Mediastinal or neck surgery
- Tracheo-esophageal fistula repair
- Bilateral vocal cord paralysis (abduction type)

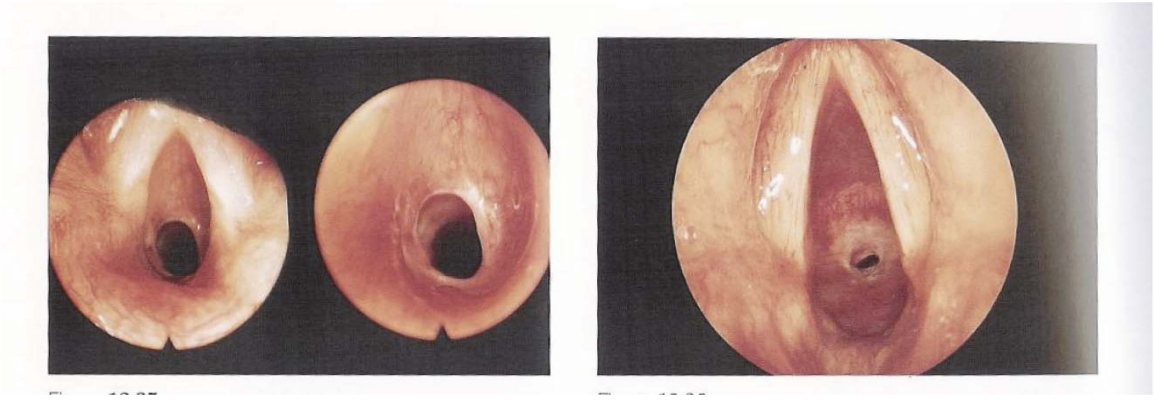
Symptoms:

Stridor

Treatment:

- Need temporary tracheotomy
- Vocal cord lateralization
- Cordectomy arytenoidectomy

3. Acquired subglottic stenosis:



Risk factors:

- Prolong intubation
- Size of the tube, material
- Care of intubated patient
- High pressure cuffs tube
- Difficult intubations
- Multiple intubation

- GERD
- Tracheobronchial infection

  Laryngeal → granulation tissue → ulceration → perichondratis → subglottic stenosis

Treatment:

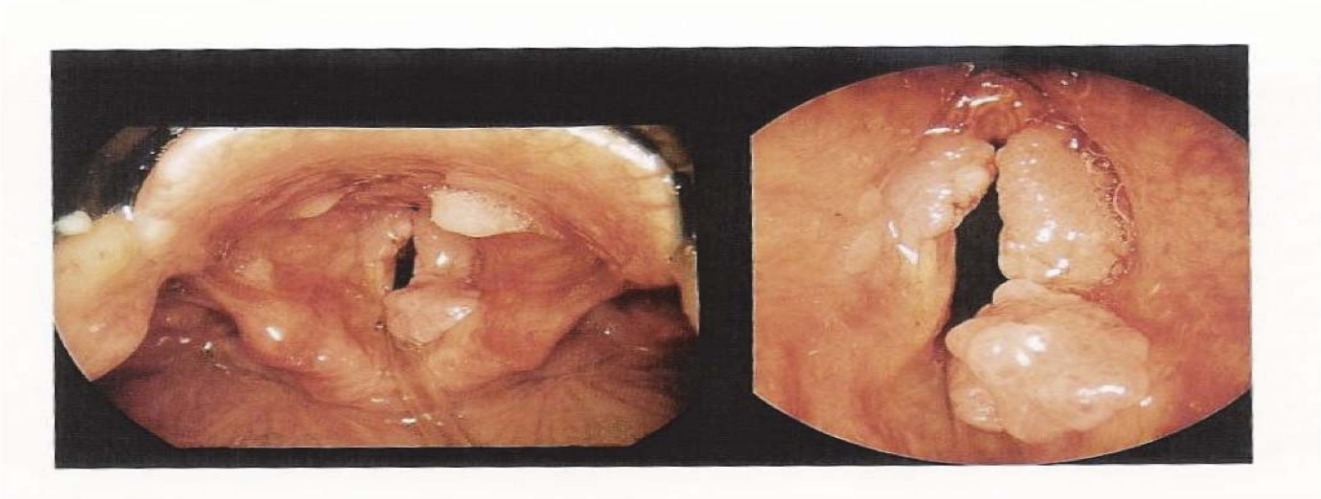
Grade I&II

- Observation
- Laser excision

Grade III&IV

- Trachestomy
- Laryngotracheal reconstruction
- Cricotracheal resection

4. Respiratory papillomatosis:



2/3 before age 15

rarely malignant change

Cause: HPV 6-11 (benign changes)

16-18 associated with malignancy

Risks: younger first time mother (condyloma acuminata)

Lesions: wart like (cluster of grapes)

Types:

- Juvenile (worst)
- Senile

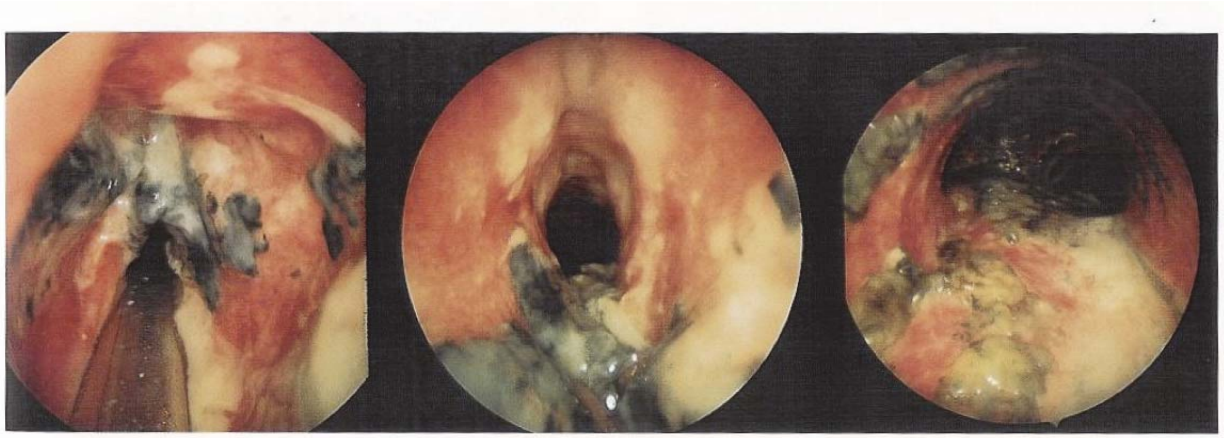
Signs and symptoms:

Hoarseness, stridor

Treatment

- laser excision ,microdebrider
- Adjunctive therapy:
- **Cidofovir,** acyclovir , interferon (very toxic drug given IV only in refractory cases which are aggressive reoccurring every one month and involving the trachea and lung)

5. Thermal injury:



Aspiration hot liquid, caustic fluid

Treatment:

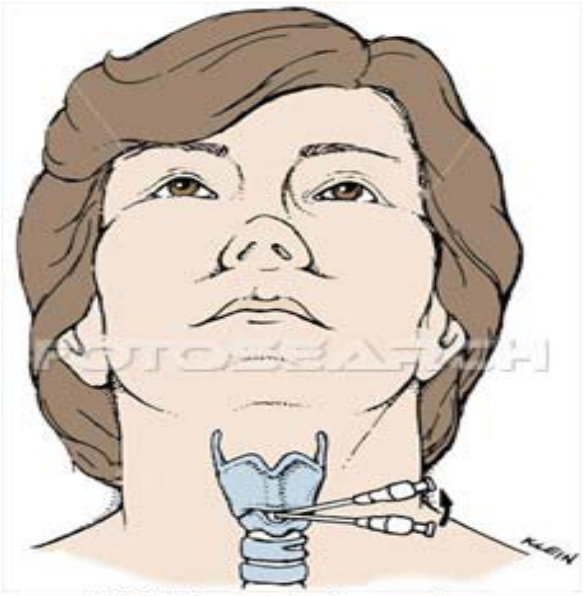
- Intubation
- Tracheostomy
- IV ABX and antireflux

Cricothyroidectomy:

- A technique for obtaining an emergency airway through the cricothyroid membrane(between thyroid and cricoid cartilages) when standard airway techniques have failed.
- In which a large-bore intravenous cannula is inserted directly through the membrane.
- Surgical cricothyroidotomy in which a surgical hole is made in the membrane and a cuffed tube, similar to a short endotracheal tube is inserted directly.

Indications:

- Need for an emergency airway where:
- Intubation is not possible
- Need to avoid neck manipulation
- Severe maxillofacial trauma
- Oedema of throat
- Severe oropharyngeal/tracheobronchial haemorrhage
- Foreign body in upper airway
- Lack of equipment for endotracheal intubation
- Technical failure of intubation Severe



Complications:

- Thyrohyoid membrane incision
- Intra/postoperative bleeding
- Subglottic stenosis
- Dysphonia/hoarseness
- Laryngeal damage
- Tube misplaced in bronchus
- Pulmonary aspiration
- Tracheal stenosis.

- Recurrent laryngeal nerve injury
- Esophageal perforation or tracheo-esophageal fistula.
- Tracheo-left brachiocephalic vein fistula
- Fracture of thyroid cartilage

Tracheostomy:

(Takes longer time to perform than cricothyroidectomy):

Performed between 2nd and 3rd or 3rd and 4th tracheal ring

Indications:

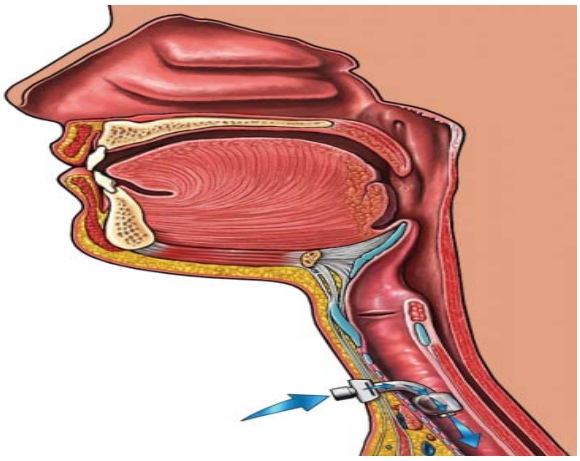
- Obstruction of the upper airway, e.g. foreign body, trauma, infection, laryngeal tumour, facial fractures
- Impaired respiratory function, e.g. head trauma leading to unconsciousness, bulbar poliomyelitis
- To assist weaning from ventilatory support in patients on intensive care longer than 3 weeks (more safer)
- To help clear secretions in the upper airway (lung lavage in COPD)

When removing the tracheostomy; cover the area with gauze, within 48hrs it will collapse and close full complete healing it takes from 1-3 weeks

Complications:

Immediate:

- Haemorrhage, e.g. from thyroid isthmus
- Hypoxia
- Trauma to recurrent laryngeal nerve
- Damage to oesophagus
- Pneumothorax
- Subcutaneous emphysema (3-4cm skin incision but when opening the trachea air will pass to the adjacent skin leading to crepitus when touched)

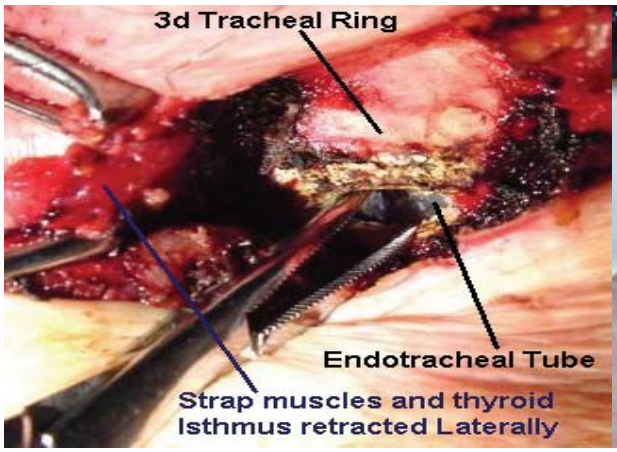


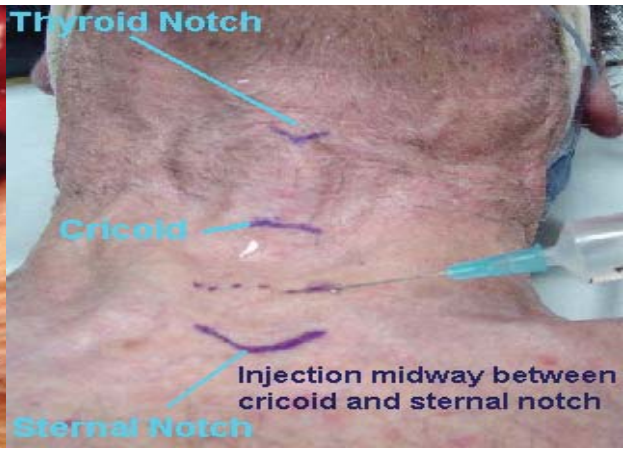
Early:

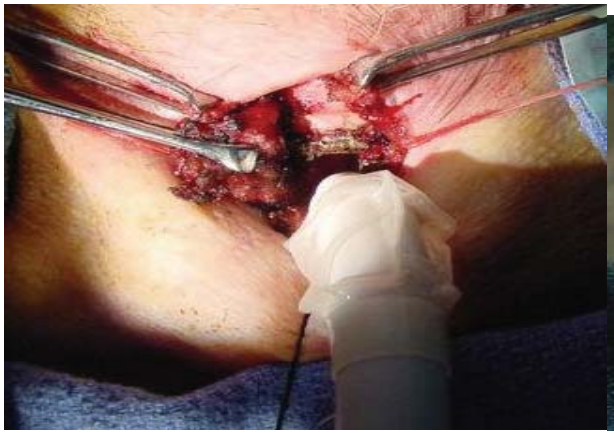
- Tube obstruction (dislodging) or displacement (stitch it to the skin to prevent this and remove after 5 days to one week)
- Aspiration
- Bleeding from tracheostomy site
- Infection

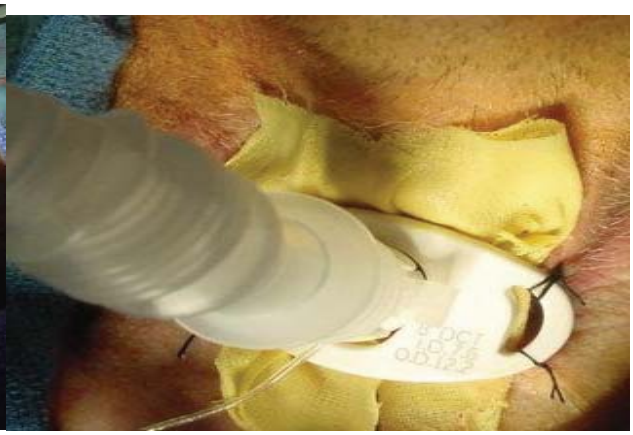
Late:

- Airway obstruction with aspiration
- Tracheomalacia
- Aspiration and pneumonia
- Fistula formation: e.g. tracheo-cutaneous (when the patient comes complaining of fistula that didn't close after a month of removing the tracgeostomy. This is commonly seen in patiants that had radiation for skin or neck tumors). or tracheo-oesophageal complain of regrugtation or trachea-pnemonitic artery complaining of bleeding from tracheal incision.
- Damage to larynx, e.g. stenosisTracheal stenosis









Recurrent laryngeal passes laterally but more laterally is the esophagus.
In kids innominate artery (brachiocephalic artery or trunk) is higher in position than in adults and it resembles the trachea in shape

In summary:

Clinical assessment of child with upper airway obstruction:

History:

- Age (congenital vs acquired)
- Speed of onset and precipitating event (crying, feeding..etc)
- Associated symptoms (fever, drooling. Hoarseness..)
- Feeding difficulty
- Past medical history (birth trauma, intubation…)

Examination :

Children examination should be from head to toe to check for syndromes

- Craniofacial anomalies
- Cutaneous haemangiomas
- Respiratory distress sign (using flaring of ala, suprasternal retraction, accessory muscle. Take immediately to OR)
- Neck mass
- Growth chart
- COMPLETE ENT EXAMINATION
- Flexible fibroptic examination

Investigation :

Physiological studies:

- ABG
- Spirometry

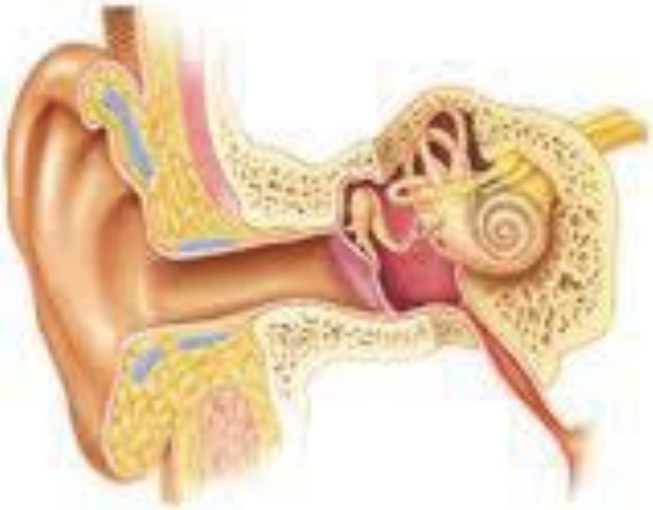
Imaging :

- Chest Xray (FB, complications such as pneumonia and atelectasis)
- HKV (subglottic stenosis)
- CT scan (choanal atrasia ,retropharyngeal abscess ,tumor)
- MRI (intracranial extension)
- Barium swallow (vascular ring trachea is compressed by big vessels)

Endoscopy: is the tool of examination

430 Teams

Diseases of the Ear, Nose and Throat



7th-8th Lecture:

Trauma and Foreign Body Part 1

Done by: May Alabdulaaly

Revised by: Yusra Al- Kayyali

The slides were not provided by doctor (Dr.Fatimah Al-Anezi)
Source: recording & some pictures of the slides
Important Notes in red
Copied talk in black/purple
Copies slides as they are gray
Our external notes in grey/ blue

2 TRAUMA & FOREIGN BODY PART 1

Team part 1 will talk about:

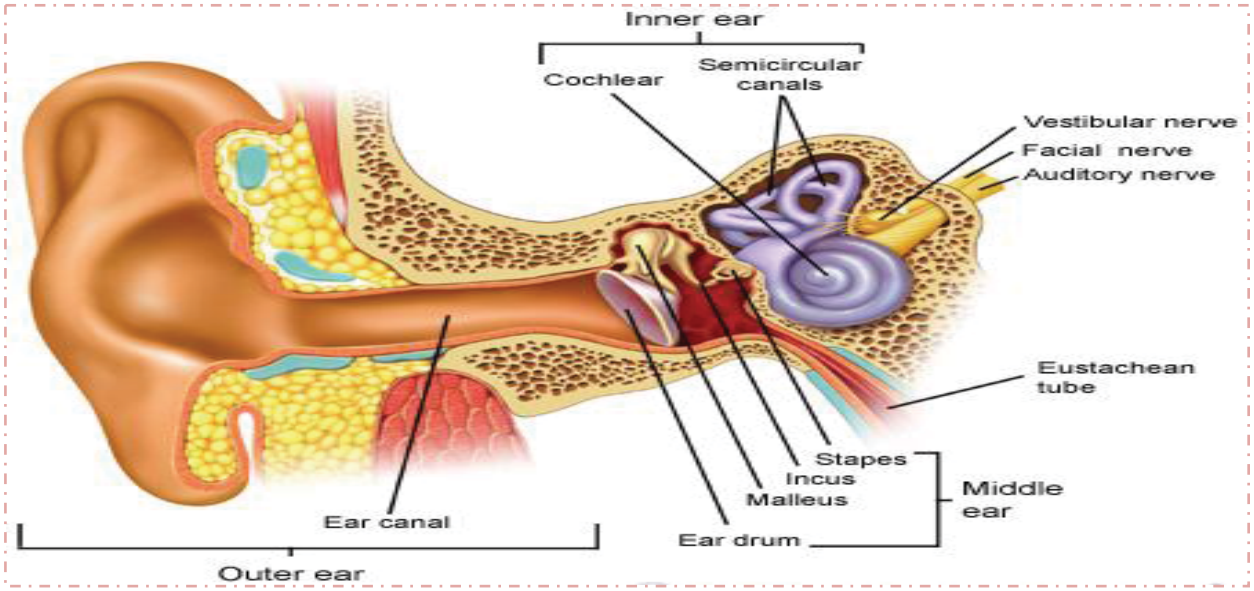
-Ear Trauma: Inner, Middle & External.
-Temporal Bone Fracture
-Facial Trauma

Team part 2 will talk about:

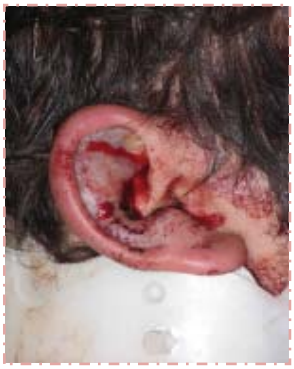
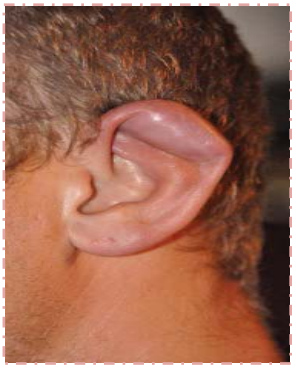
-Facial Trauma
-Nasal Trauma
-Laryngeal Trauma
-Foreign Bodies
-Esophageal Perforation

**EAR TRAUMA

Ear trauma can be divided to external(auricular), middle, or inner ear trauma.
It could be a laceration, or avulsion (completely cut off). It could also be a burn, or radiation injury, or hematoma.



**EXTERNAL EAR:
(Auricle injuries)

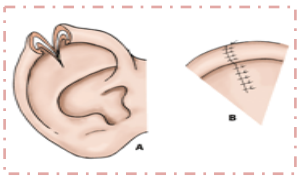



1- Hematoma
Cartilage injuries in general cause hematoma.
Very common, we see 2-3 cases per week.
The child fell, adults were in a fight, ear got hit.
Why hematoma is different in ear than in thigh?
Cartilage.
Cartilage in general does not have blood vessels; therefore it takes its nutrition either from periosteum (Bone) or perichondrium (CT). If there hematoma then the cartilage will get separated from these structure and will not get nutrition.
If left untreated → Necrosis of the cartilage → permanent deformity (Cauliflower deformity)
Therefore it needs to be diagnosed early.

Treatment: Drain it & apply pressure dressing (To reattach it together with the periosteum). Excise fibrous tissue.

2-Avulsion:
Ear or part of ear is cut off.
If patient presents within 3-4 hours to the ER we can re-implant it and re-vascularize it.
If the patient presents late the surgeon can install a plastic ear.

3-Laceration:
From glass, knives, bite injuries
Treatment: Sutures



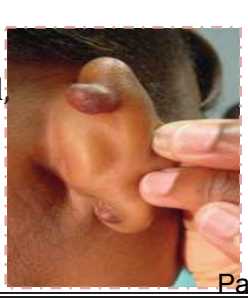
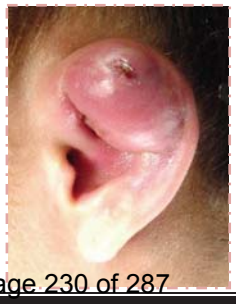
4-Cancer:
Ear is affected by cancer SCC (Squamous cell carcinoma) or BCC (Bassal cell carcinoma)

5-Frostbite:
In cold countries the cartilage gets necrosis.

6-Burns

7-Split or Cauliflower Injuries From Ear Piercings or Earrings.
-The lobule (where our ear pierced by our parents) is made of soft tissue and has no cartilage. It can get split in half.(if earring was pulled)
Treatment: Suture it in the clinic.

-If the piercing is higher in the ear, in the cartilage it can get infected,
Cause a deformity, hematoma, abscess and even keloid in dark skinned people.
Treatment: Drain if abscess or hematoma.
Local steroids is the treatment for Keloid as it returns if removed

**MIDDLE EAR:

. Trauma can happen to the tympanic membrane, ossicles, inner ear bone & structure (labyrinth), Incudostapedial joint…

1- Can Get Traumatic Perforation:

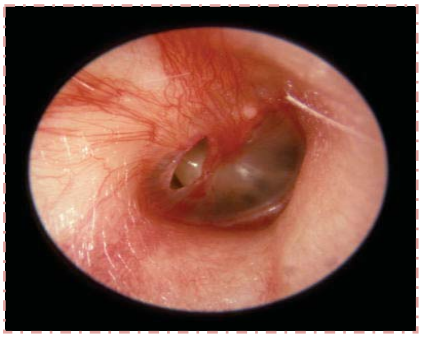
- Blast injuries. From pressure or small body inside the ear. Can also affect inner ear.
- Insertion of foreign objects in the ear. By cleaning the ear and accidentally perforating it. Example ear cotton buds.
- Slap
- Skull base fracture (also affects facial nerve).

How to differentiate traumatic perforation of tympanic membrane from chronic infection perforation?
Onset from history, pain, hearing loss, and vertigo (all those are present in acute, possibly discharge in chronic).
Important for medico-legal cases.

Acute → the membrane will be irregular, and red (bleeding), and there will be blood collected in external canal.
Chronic → the membrane will be epithilialized (epithelium re-grow) with more rounded edges.

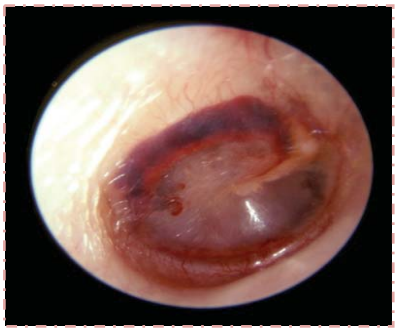
**Incudostapedial joint is the most commonly affected area by trauma, as it is a small joint with very small blood vessels. Most affected by trauma patient comes with hearing loss.
Ossicular trauma alone has more hearing loss than tympanic membrane trauma alone. If combined more hearing loss.

Treatment:
If small perforation → it can heal by itself in 6-8 months. But the patient must avoid infection because it prevents re-growth.
If large → requires Graft Tympanoplasty. If the ossicles are involved do an ossiculoplasty.
How to confirm if the ossicles are involved? Must do an audiogram
Displacement of the ossicles will cause conductive hearing loss. Forcible displacement of the stapes into the inner ear can cause sensory neural hearing loss that can't be corrected even if the ossicles are put back into their proper position.



2-Hemotympanum:

When a blow, fracture, or blast is not severe enough to cause perforation but causes hematoma. A child inserted a pencil in his ear.
Symptoms: Pain. Hearing is affected (decreased).
Treatment: Do nothing it will resolve by itself in 1-2 weeks.



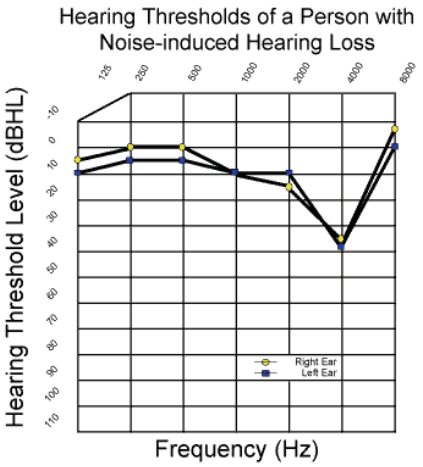
**INNER EAR:

NIHL, Acoustic trauma, and Barotrauma.

1-NIHL(Noise Induced Hearing Loss): 1 of the commonest occupational induced disabilities

Noise induced Sensory neural hearing loss NISNHL

When hearing loss has occurred because of exposure to noise, the audiogram will show that the individual has lost the most hearing at the 4,000-Hertz frequency. This hearing loss will appear as a notch, or dip, in the audiogram at 4,000 Hertz frequency, as shown on the graph below. In addition to the occupational and personal history, this is how the doctor can determine from the audiogram that your hearing loss is related to noise.



People who work in airports or in ambulances are most affected if not wearing protective gear; however they do not notice it at first. *Tinnitus* is the first warning sign.

Not all people that work in areas with high frequencies suffer from NIHL; different people have different susceptibilities.

Some people are affected from the first encounter with high frequency Noise while others need recurrent exposure for longer periods.

It is not really dependent on age, race, or gender.

frequency	exposure
90 db	8 hours
95 db	4 hours
100 db	2 hours
105 db	1 hour

NIHL happens when a person is exposed to more than 90 db for an average of 8 hours.

Prevention: People who work at noisy places such as airports or ambulances should wear ear protective gear. Those who wear headphones with loud voices can also get it, as well as those who frequently attend bars, discos and weddings playing loud music.

Important: To have early identification either so that the company will compensate the worker or to change his place of work so he wouldn't be harmed if he couldn't hear the high frequencies.

2-Barotrauma is a physical damage to the body tissues caused by a significant ambient pressure change. Examples: such as when a scuba diver, a free-diver or in passengers of an old non pressurized airplane as well as in military pilots and pilots or passengers with T QS HÅT oodqÅqdr ohq` snqxÅsq` bsÅ hmedbshmm).

Causes: Injury to the tympanic membrane TM and middle ear ME, flying or underwater diving, ETD (Eustachian tube dysfunction) may predispose.

Signs & Symptoms: Ear pain, hearing loss, hyperemia and possible TM perforation, edema and ecchymosis of the ME membrane, conductive hearing loss and/or transudative middle ear perfusion.

The Eustachian Tube together with the external canal equalize the pressure in the 2 sides of tympanic membrane (middle & external ear pressure).

If someone's eustachian tube is not working he/she will have -ve pressure in the middle ear → sucking of blood from blood vessels to tympanic membrane or accumulation (Mostly) of blood or fluid in the middle ear. (hemotypanum)

Seen always in people with cold who get in airplane after it lands they have hearing loss, pain.

What other Symptoms? Vertigo and conductive hearing loss when doing audiogram (due to fluid).

In extreme cases diving can get hemotympanum (accumulation of blood in the middle ear).

Treatment: Important for airplane pilots with URTI to have it treated prior to flying the airplane. Give them Nasal decongestant before they fly so their ET will open. (Aviation medicine)

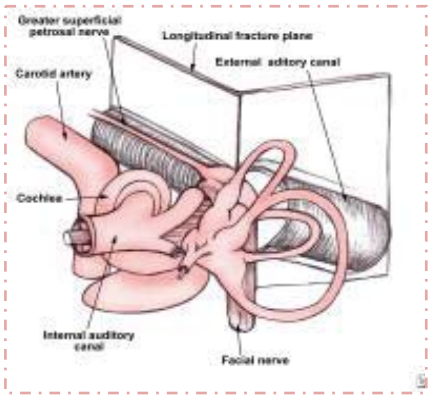
If come afterward with the hearing loss and pain? Give decongestant nasal spray; most usually recover in 2 weeks.

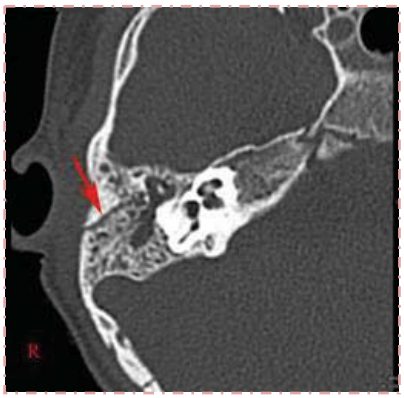
**TEMPORAL BONE FRACTURE

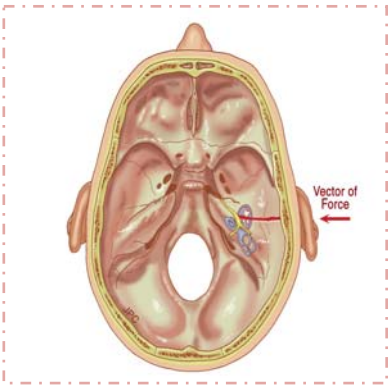
Temporal bone area contains the Middle and Inner Ear.
Fractures can happen either from a hit to frontal, occipital or from a side injury.
Etiology:
-Blunt closed head trauma: Most are from RTA(road traffic accident) Can be due to a child falling, from a Motorcycle accident, etc... (blunt is the most Common)
-Penetrating injuries. From glass, knives, blast, fights, laceration

Can affect facial nerve, semicircular canal (vestibule), ossicles.
Temporal bone axis is not straight
Blunt trauma: 5% of people who have head trauma will have temporal bone fracture. These are classified with respect to the axis of the Petrous Ridge and include:

- Longitudinal 70-80% of breaks but causes less damage
- Transverse 10-20% worse diagnosis. More facial nerve injury, as well as semicircular canal injury. However Less severe than mixed.
- Mixed 10% worst presentation and prognosis.







Facial paralysis commonly occurs after transverse fractures of the temporal bone (50%). However it can also occur after longitudinal fractures (25%).

Inner Ear: the Vestibulocochlear Nerve (CN 8) & Vestibules (Semicircular canal) pass through it. Those are less involved in longitudinal fracture, and more with transverse.
(Therefore vertigo happens more with transverse)
Middle Ear has the facial nerve passing through it. It is more involved in transverse fractures, and less in longitudinal.

How to diagnose them?
If the patient comes with multisystemic trauma:

History: Do thorough evaluation. The mechanism and details of the traumatic forces involved.
Physical examination: ABCDE. Then after the patient is hemodynamically stable and the acute injuries are recognized and dealt with he/she should be re-evaluated again for other less significant injuries.
CT Scanning: high resolution CT scan of the temporal bone. The integrity of the ossicular chain may also be revaluated with an optimal CT scan.
Usually in these cases the diagnosis of temporal bone trauma, facial nerve palsy and other internal injuries can be missed or diagnosed late especially in severe RTAs.

GOLD STANDARD for temporal bone fracture: high resolution CT scan.

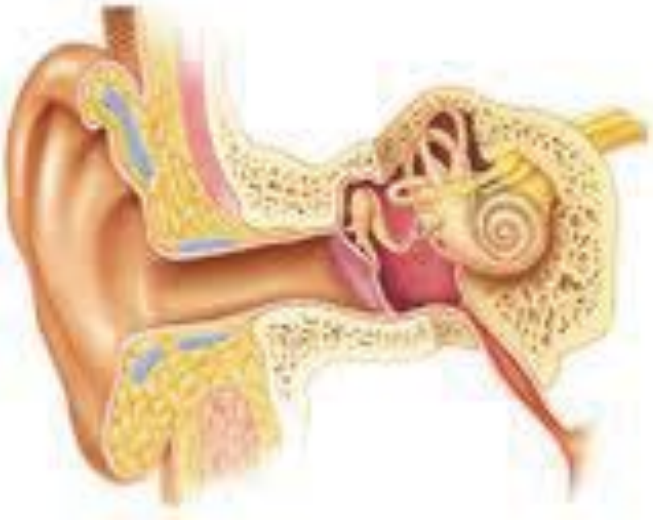
Skull base fracture = temporal bone fracture.

Complications of temporal bone fracture:

- Facial paralysis.
- Hearing loss (check if Tympanic membrane is punctured or cut.)
- CSF leakage from ear as discharge
- Tinniuitis
- Carotid injury
- Vertigo

How to treat it? We treat it based on whether it is simple or complex.
Complex needs surgery with Neurosurgeon. If facial nerve is trapped release it.

Diseases of the Ear,Nose and Throat



7-8st Lectures:

Trauma and Foreign Body –Part2

Done by: Ayan Hussain
Revised by: Yusra Al-Kayyali

The slides were not provided by (Fatimah Al-Anezi)
If not provided mention your source (e.g. 428 team slides / females)
Important Notes in red
Copied slides in black
Notes in green/ blue

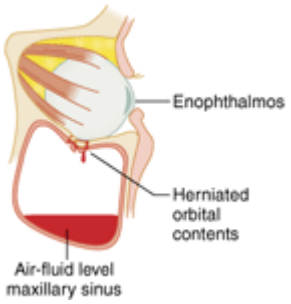
-The external features are NOT always indicative of the severity!
-First thing is to make sure the airway is patent.

**FACIAL TRAUMA :

- Orbital Floor Fractures (blowout):

Trauma will affect the weak part of the orbit: Floor of the orbit (roof of maxillary sinus).

- ✓ It can occur as an isolated injury.
- ✓ Or in combination with zygomatic arch fractures, Le Fort type II or III mid-face fractures, medial wall or orbital rim fractures.
- ✓ When it is an isolated injury the object is usually intermediate in size. not small enough to perforate the eye but not large enough to reach or affect the eyebrow or other areas of the face. The commonest example is a tennis ball.
- ✓ The force may lead to inferior rectus entrapment and fat herniation inferio-medially leading to enophthalmos due to the break in the floor of the orbit.
  Patient should be referred to ophthalmologist for vision examination.



-Etiology:

- ➢ Pure Orbital floor fractures results from an impact injury to the globe and upper eyelid.
- ➢ The object is usually small enough to not fracture the orbital rim, but large enough not to perforate the globe.

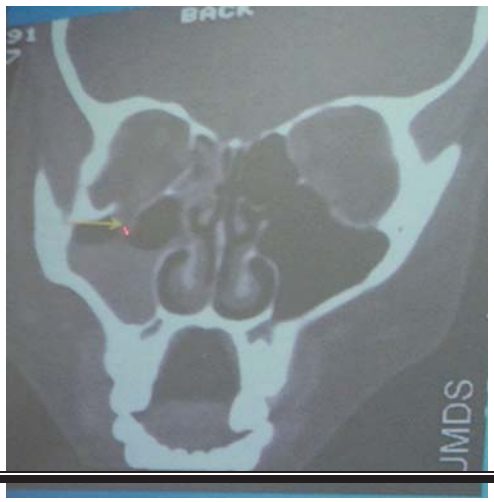
-Presentation:

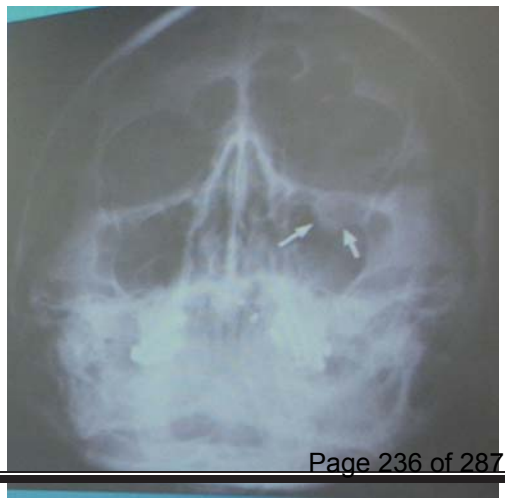
1. Limitation of movement.
2. Diplopia (especially in up-gaze)
3. Decreased visual acuity.
4. Blepharoptosis: drooping or abnormal relaxation of the upper eyelid.
5. Patients may complain of epistaxis and eyelid swelling following nose blowing.
6. The globe can be ruptured
7. Enophthalmos (posterior displacement of the eyeball within the orbit)

-Imaging studies:

- ✓ AP X-ray views of the orbit.
- ✓ The most common views are the Caldwell and Water's Projections (also known as occipitofrontal and occipitomental views relatively)
- ✓ CT scanning (**The Best**): obtains both axial and direct coronal to properly evaluate the orbit and its floor.

_**Teardrop** fracture sign on x-ray

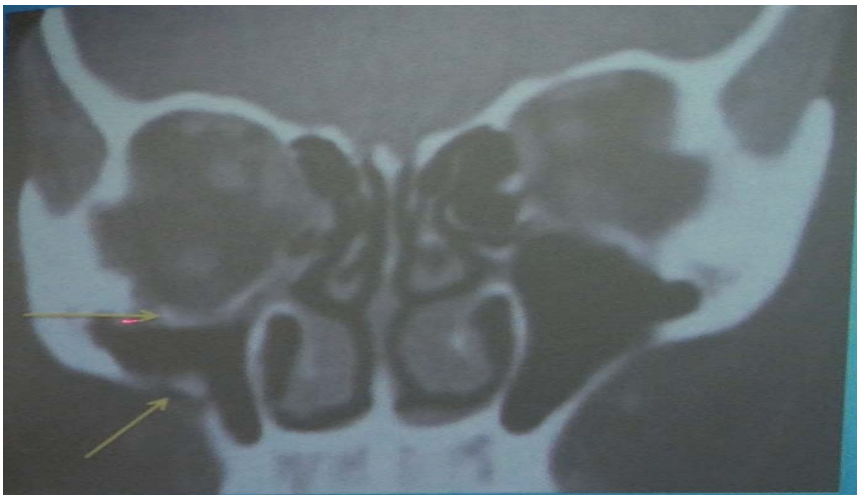




Blowout Fracture on CT Scan:

-Facial trauma orbital floor fracture (blowout).

-Coronal CT scan image is showing an orbital floor fracture posterior to the globe; a fracture of the lateral maxillary sinus wall is also present.



-Treatment: cosmetic as well as to explore and release the displaced soft tissue, and to repair the bony deficit by removing or repositioning the bony fragment.

***Surgical Treatment** for the orbital floor to be carried out through:
- A conjuctival approach: transconjunctival
- Cutaneouse exposure
- Transmaxillary approach
- Endoscopically: enter through maxillary sinus and push up the roof

Keep in mind that it is rare to have an isolated injury so always look for other fractures and injuries.

**NASAL TRAUMA:

It is important to ask about the DURATION of trauma. (onset)

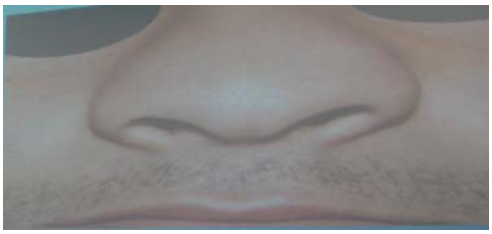
- Causes:

1. Traumatic most commonly.
2. Iatrogenic (surgical).
3. Foreign bodies: If stayed for long time will lead to necrosis of cartilage.

Repair time is limited:
Nasal bone reduction:
Pediatrics: within 10 days
Adults: Up to Two Weeks
If more than that will need to do a septorhinoplasty.

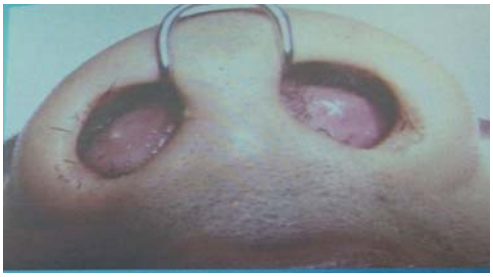
- ✓ The bones of the nose are the most frequently broken bones in the face as they are the most prominent. A nose break will affect the patient's appearance. There will probably be deviation, distortion and swelling over the nasal bridge.
- ✓ Immediate evaluation is necessary to make sure there is no septal hematoma (blood between the septum and cartilage).
- ✓ If septal hematoma develops (it should be drained), it might be complicated by an infection, and 5 days later it might progress to an abscess. This may lead to cartilage necrosis and the patient might end up with a saddle nose deformity because of supportive cartilage loss.
- ✓ Usually swelling and edema may interfere with a proper evaluation. Therefore, we re-examine for any deviation or fracture after 3-4 days if a child or a week if an adult (children heal faster than adults).
- ü Do a nasal bone reduction if patient presents early: pediatrics within 10 days and adults up to two weeks. However If the bone has healed alone, or its a complicated fracture, or there is a hematoma: septorhinoplanty needs to be done. For children wait until the age of 18.

Case Scenario:

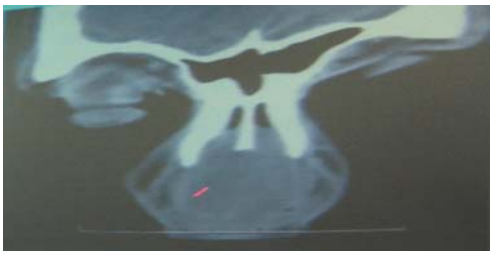


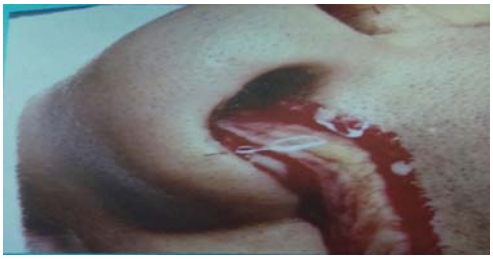
A 30 years old male presented with progressive painless nasal obstruction and widening of the nose for 2 weeks. Upon questioning he denied having any surgeries but gave a vague history of nasal trauma while playing soccer.

On Physical Examination , the septum was Ballooned, fluctuant and filled both nasal cavities.



Coronal CT scan imaging showed , Expansile swelling of the nasal septum .Notice the complete bilateral nasal airway obstruction..





On incision and Drainage: Thick yellow pus was recovered

A Penrose Drain was left inside the abscess cavity and Doyle nasal splint was placed bilaterally to compress the nasal septum.

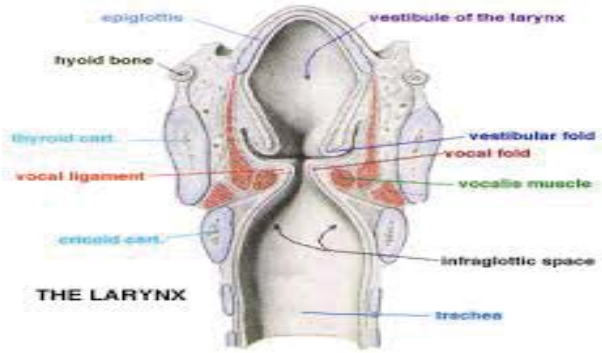
**LARYNGEAL TRAUMA:

Very important because it is underestimated but it is life threatening.

-Laryngeal Anatomy:

Function:

1-Airway
2-voice
3-Swalloing
well protected by Mandible and sternum
supports hyoid , thyroid and cricoids
Outcomes are determined by initial management ..

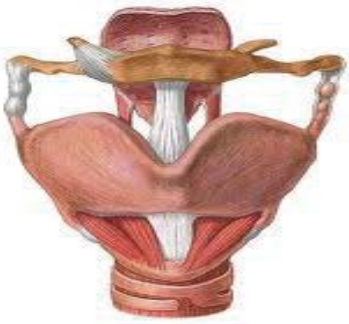


-Mechanism of injury:

1.Blunt:

Motor Vehicle Accident, road traffic accident, strangulation (suicide), clothesline, sports related.

Significant internal damage with minimal signs.



2.Penetrating: its common
Gunshot wounds: damage related to velocity.
Knife: easy to underestimate damage.

-History is very important! Ask about the following:

1. Change in voice – most reliable and very alarming. Indicates vocal cord involvement. Can be the only presentation
2. Dysphagia or Odynophgia following trauma.
3. Difficulty breathing (dyspnea)– more severe injury.
4. Hemoptysis
5. Anterior neck pain

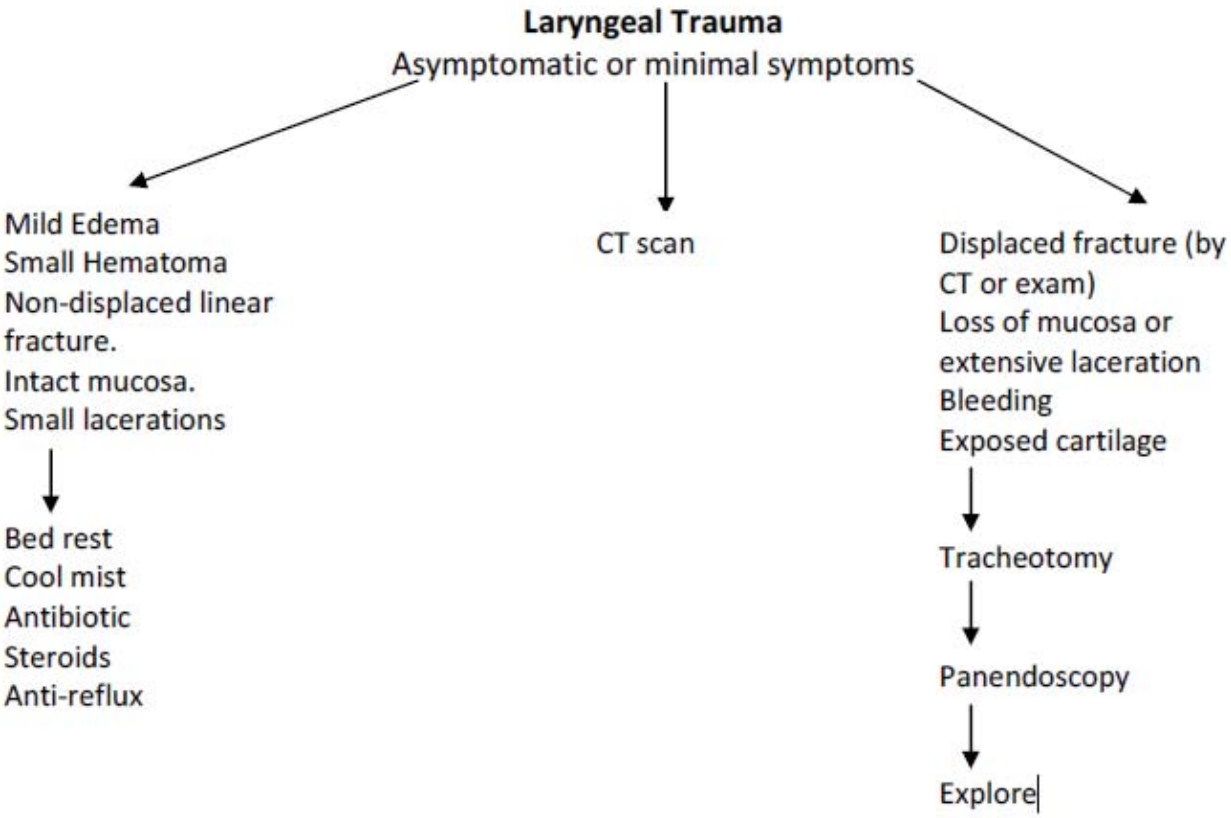
-Physical exam: Look for:

1. Stridor: Noisy breathing
2. Hoarseness
3. Subcutaneous emphysema: Cut in the larynx.
4. Laryngeal tendeness, ecchymosis, edema.
5. Loss of thyroid cartilage prominence.
6. Major blood vessel or nerve injury. Especially in gunshot wounds.
7. Associated injuries – vascular, cervical spine, esophageal.

-Investigations:

1. **Flexible Fiberoptic Laryngoscope**: Perform in emergency room and its findings dictate next step.
2. CT scan.
3. Tracheotomy.
4. Endoscopic or external surgical exploration.
5. Other studies include angiography, cervical spine radiograph and barium esophagram swallow depend on the case itself.

-Management: you have to always secure the airway as an initial step.
Airway management: Laryngo-tracheal tube or tracheostomy. If inhalational injury, ventilate the patient.
Medical management: We might give epinephrine, steam inhalation to decrease congestion, and steroids to decrease the inflammation.
Surgical management



**FOREIGN BODIES:

-It is very common in children and mentally retarded patients.

1-FOREIGN BODIES IN THE EAR

- ✓ It's a common problem especially in toddlers.
- ✓ The vast majority of the items are lodged in the ear canal.
- ✓ Most cases of the foreign bodies in the ear are not serious.
- ✓ Common objects found in the ears include: Food material, beads, toys and insects.

-Signs and symptoms:

- ✓ If the foreign body in the ear goes **undetected** it can cause an **infection** in the ear, the patient will comes with discharge.
- ✓ **Pain.**
- ✓ Decrease in **hearing.**
- ✓ **Bleeding** is also common but is not urgent: does not require immediate intervention
- ✓ A live insect in the ear. The insect's movement can cause a **buzzing** the ear.

-Treatment:.

Removal of the foreign body is done in the clinic, if uncooperative child we remove it microscopically under minor sedation; it is usually not urgent.
Urgent removal is indicated if the object is causing **significant pain** or discomfort. Also if it was a food or a plant material such as beans because they will swell when they are moistened and if swollen will affect the external canal and might lead to otitis externa. If it enlarges the physician will no longer be able to remove it. Therefore, we remove it under GA in children and give antibiotics.
Remove BUTTON BATTERIES immediately as they can decompose within 25 hours in the body, allowing the chemicals to leak out and cause chemical burns. **Urgent** removal is required.
Small insects such as ants are removed by simply putting baby oil or water (contraindicated in tympanic membrane is perforated).
Ticks: put some local anesthetic, they will release themselves and be easily removed.

2-FOREIGN BODIES IN THE NOSE:

- ✓ The most common site is between the inferior turbinate and the nasal septum.
- ✓ It differs from the ear in that the nose is part of the airway tract.
- ✓ Painful
- ✓ If the foreign body stays in the nose for a long time it will cause perforation.

- Treatment:

1. The most important thing is to secure the airway.
2. If the foreign body is located anteriorly and the child is cooperative we can remove it by forceps in the clinic.
3. If it is positioned posteriorly, at the level of the nasopharynx; or if the child is struggling or uncooperative the foreign body could be pushed further back when attempting to remove it and might lead to further complications such as: the foreign body's inhalation or reaching the lungs. In these cases, take the patient to the O.R and remove it under G.A.

3-FOREIGN BODY IN THE PHARYNX AND OROPHARYNX : IMP

- ✓ MAINTAIN PATENT AIRWAY!!
- ✓ Foreign body ingestion.
- ✓ Coins: important, if stayed in any place for a long time it will melt and its components will lead to necrosis and if in the nose it will lead to septal perforation, if in the esophagus, **esophageal perforation.**
- ✓ Meat (Fish bone)
- ✓ Vegetable matter
- ✓ Dentures
- ✓ Less than 24 hour, painful

Its size depends on whether it'll be removed in the clinic or in the OR, or if physician was not able to remove it in clinic then go to OR.

All pharyngeal foreign bodies are medical emergencies that require airway protection.

- complete airway obstruction usually occurs at the time of aspiration and results in immediate respiratory distress, emergency intervention is essential. Common obstructing foreign bodies in children include balloons, pieces of soft deformable plastic, and food boluses.
- Patients with nonobstructing or partially obstructing foreign bodies in the throat often present with a history of choking, dysphagia, odynophagia, or dysphonia.Pharyngeal foreign bodies should also be suspected in patients with undiagnosed coughing, stridor, or hoarseness.

Parents and caregivers of children with symptoms of partial airway obstruction should be asked whether choking and aspiration have occurred. Diagnosis is often complicated by delayed presentation. Case reports describe foreign bodies in the throat that were misdiagnosed and treated as croup. Thus, physicians must have a high degree of suspicion in patients with unexplained upper airway symptoms, especially in children who have a history of choking.

4-Foreign body aspiration: (lung)

- It is more serious than ingestion.
- Sometimes parents do not notice the child eating something that caused him/her to chock, or the patients were not around when it happened. Example popcorn.

-History:

- ✓ Parental suspicion in pediatrics
- ✓ Chocking
- ✓ Gagging
- ✓ Wheezing if prolonged in the chest, might be mistaken with bronchial asthma.
- ✓ Hoarseness
- ✓ Dysphonia.
- ✓ Pneumonia, foreign body can lead to infection.
- ✓ A positive history must never be ignored, while a negative history may be misleading.
- ❖ **Note:** The commonest site of ingestion injury is in the cricopharyngeal fossa because the cricopharyngeal sphincter has a protective role. Ingestion injury is common among neurological disease affecting swallowing. It is not serious unless very large object.

-Physical exam and investigations:

- ➢ Larynx/cervical trachea.
- ➢ Inspiratory or biphasic stridor.
- ➢ Intrathoracic treachea.
- ➢ Prolonged expiratory wheeze.
- ➢ Bronchi
- ➢ Unequal breath sounds.
- ➢ Diagnostic triad - <50%

1. Unilateral wheeze
2. Cough
3. Ipsilaterally diminished breath sounds.

- Assess nares/choanae.
- Assess adnoid and lingual tonsil.
- Assess TVC mobility.
- Assess laryngeal structures.

-Investigations:

- **Fiberoptic laryngoscopy: golden standard**
- Bronchoscopy if laryngoscopy is not available and is also really good.
- Proper equipment.
- Plain films: not all foreign bodies are radio-opaque therefore will not be visualized in these cases we go by the history even in the absence of +ve radiographs. Radiolucent bodies such as food like popcorn or vegetables.
  - Chest and airway AP and lat.
  - Expiratory films.
- Fluoroscopy if foreign body stayed for long and you are suspecting an injury.
- Barium swallows.
- CT, MRI, Angiopraphy.
- **Note:** inhalation injury is more serious than ingestion, but ingestion is more common.

5-Foreign body in the Tonsils

6-Foreign Body in the Larynx: dangerous if the foreign body is big

7-Foreign body in the esophagus

-Most of the foreign bodies are found at the level of the cricopharyngus muscle.

-ESOPHAGEAL PERFORATION: 50% mortality rate

- ✓ **The most common cause of an esophageal perforation is injury during placement of a naso-gastric tube or a medical procedure such as esophagoscopy.**
- ✓ A tumor, gastric reflux with ulceration, violent vomiting or swallowing a foreign object or caustic chemicals or dentures.
- ✓ Injuries that hit the esophagus area (blunt trauma) and injury to the esophagus during an operation on another organ near the esophagus. Rare cases have also been associated with childbirth defecation, seizures, heavy lifting, and forceful swallowing.

-Symptoms and signs:

- ✓ The main symptom is **pain**, but the condition can progress to shock – even death – if untreated.
- ✓ Signs include **fast breathing, rapid heart rate, low blood pressure, and fever**.
- ✓ Patient with **a perforation** in the uppermost portion of the esophagus (cervical part) may complain of neck pain or stiffness and air bubbles underneath the skin.
- ✓ Patients with a perforation in the middle portion or lowermost portion of the esophagus may have difficulty swallowing, chest pain, and difficulty in breathing.

-Exams and tests:

- ✓ **A chest x-ray** may reveal that there is air in the soft tissues of the chest, fluid that has leaked from the space around the lungs, or a lung collapse. Do before CT

- ✓ **A chest CT scan** may show an abscess in the chest or esophageal cancer. X-rays taken after you drink a non-harmful dye can help pinpoint the location of the perforation. Definitive

-Treatment could be either:

A. Initial
B. Definitive

A. Initial Phase:

It includes diagnostic studies to determine the location and the cause of the hole. Administer IV fluids and IV Antibiotics to prevent or treat the infection. Fluids that have collected around the lungs may be treated by a chest tube to drain it away.

B. Definitive Phase:

It is to repair perforation. Early surgery is appropriate for almost all patients. Every effort should be done to have surgery within 24 hours of perforation.

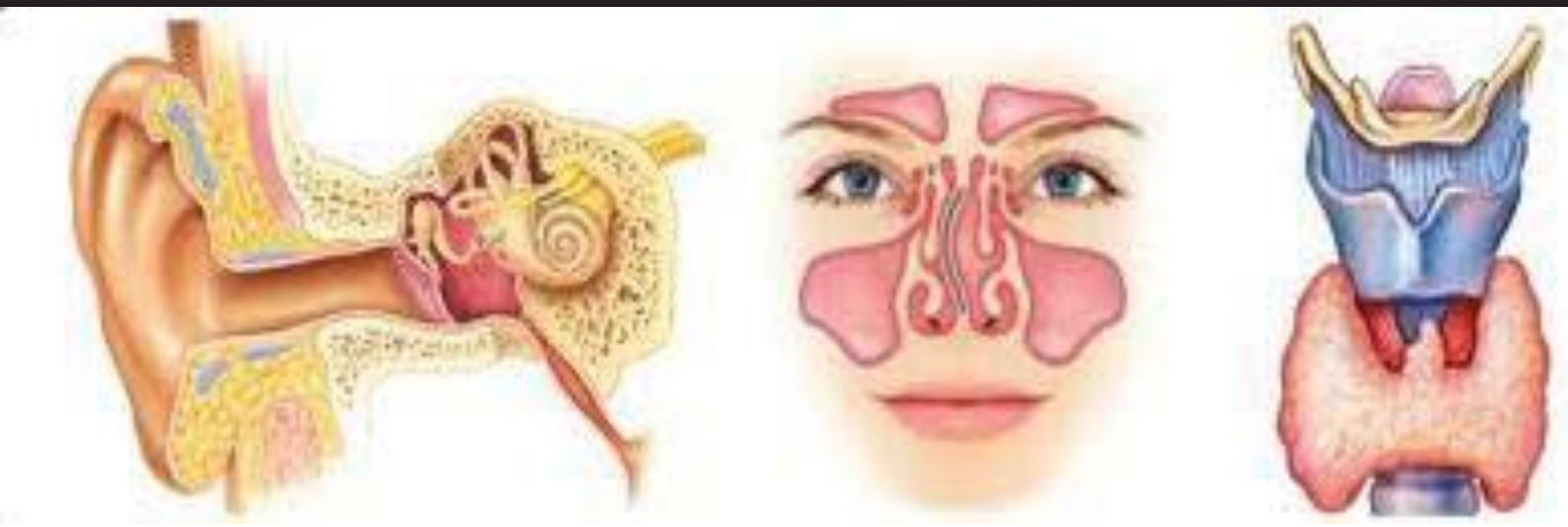
- ➢ Is to repair the perforation, for some patients with perforation in the uppermost (neck region) of the esophagus, the perforation may heal by itself if the patient does not eat or drink for a period of time. In this case nutrition must be provided by another source, such as a stomach feeding tube.
- ➢ For perforation in the mid-portion and lower-most portions of the esophagus, an operation is usually required for repair. Depending on the size and location of the perforation, the leak may be treated by simple repair or by removal of the esophagus.

-Complications: 50% of the patients deteriorate.

-Possible complications include:

- ✓ Permanent damage to the esophagus (narrowing or stricture).
- ✓ Abscess formation in and around the esophagus, lungs and abdomen.
- ✓ Infection of the lungs.

Diseases of the Ear, Nose and Throat



1st Lecture:

Pharynx

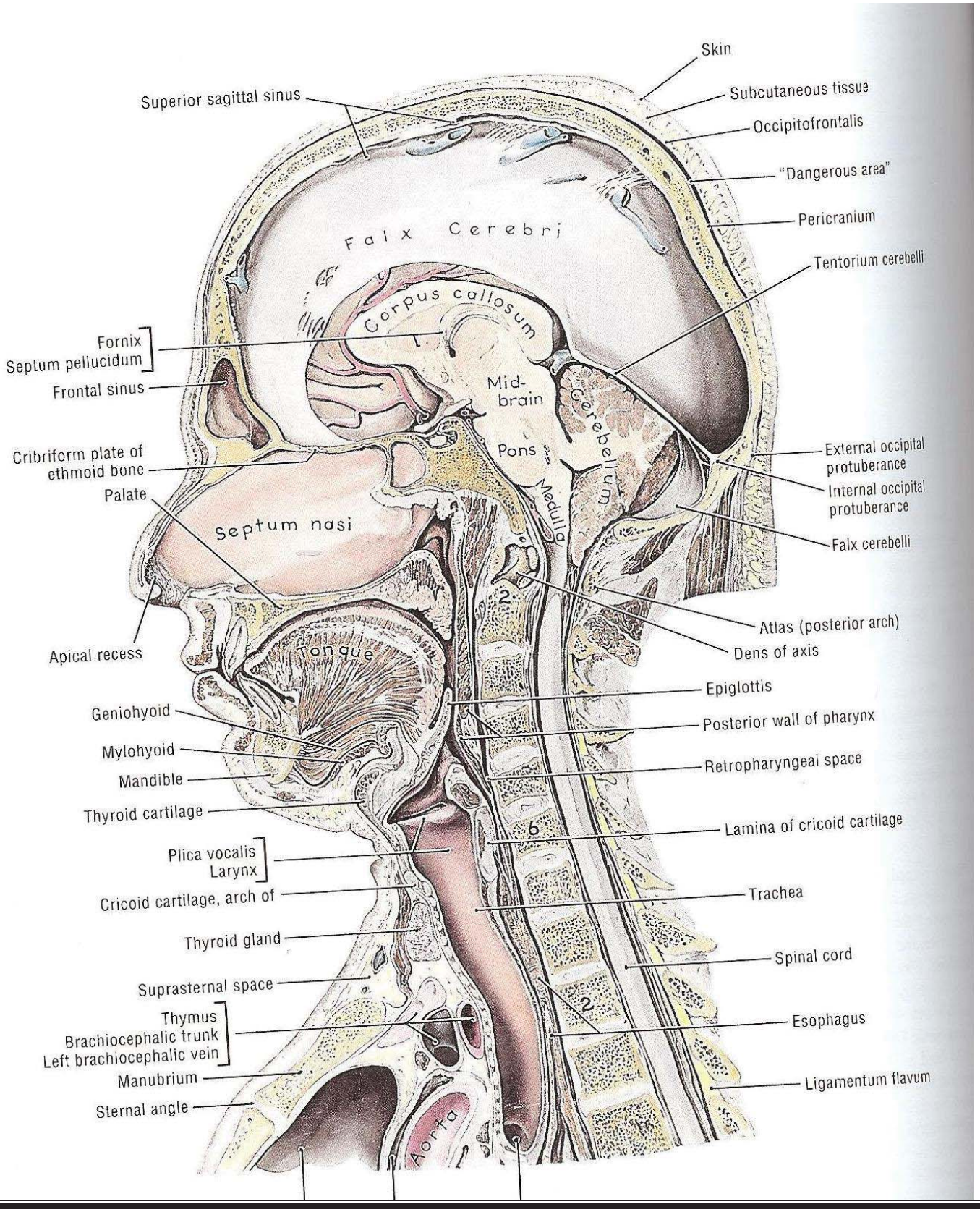
Done by: Alaa Al-Humaid and Yusra Al-Kayyali

The slides were provided by doctor (**Dr. Manal Bukhari**)
Important Notes in red
Copied slides in black
Doctor's notes in blue
Alaa's Notes in green
Highlight possible MCQ or OSCE questions mentioned or pointed out by the doctor

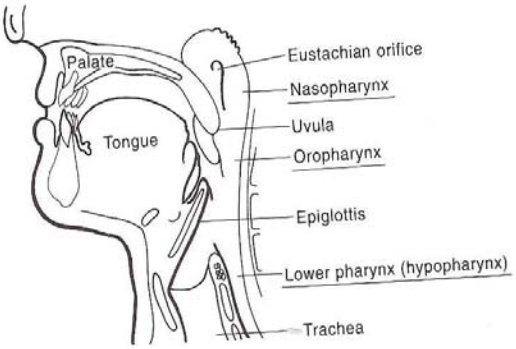
2 PHARYNX

PHARYNX:

It extends from the base of the skull to the level of the 6th cervical vertebra at the lower border of the Cricoid Cartilage.
It is funnel shaped that is 10 cms in length.



❖ ***Parts of the pharynx:*** 3 parts



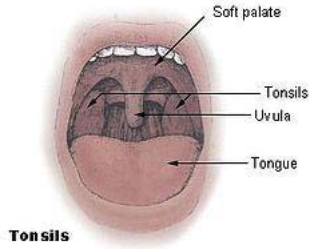
1- NASOPHARYNX:

Opens Anterior to the nose,
Above: the base of skull
Below: soft palate (when u open your mouth u can't see it because it's opposite to the nose)
Laterally: opening of the Eustachian tube
Torus tuberous
Pharyngeal recess (Fossa of Rosenmuller): the area where the nasopharyngeal cancer most commonly occurs.
Adenoid (lymph tissue): if the child has a big adenoid that causes him not to hear well, why? Because it block the Eustachian Tube the middle ear will not get good ventilation; the inside pressure and the outside pressure= negative pressure causing accumulation of fluid). fluid= glue ear.
Adenoids can reoccur. (Tonsils cannot)

Adult 45 years old smoker has nasal obstruction from time to time with ear blockage went to the doctor and was diagnosed with allergy?
Any adult + nose problem+ smoker = Have to examine the nasophaynx because of the risk of nasopharyngeal cancer (from pharyngeal recess).

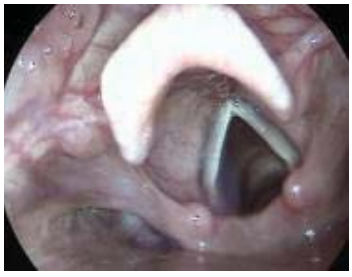
Nasopharyngeal isthmus
Scenario: Adult patient and a smoker and nasal obstruction secretory otitis media . Coughing blood so must think of nasopharyngeal cancer and not adenoid because it grows in children

2- OROPHARYNX:



Open Anterior to mouth (opposite to the throat)
Above: soft palate.
Below: the upper border of epiglottis.
Palatine tonsils (these are the tonsils that mostly get affected in tonsillitis) the tonsils are between the anterior pillars and the posterior pillars.
Tonsil size grading: imaginary line from uvula and the 2 pillars are considered the house of the tonsils. If the tonsils are 25% out of the pillars grade 1, 50% grade 2. Grade 4 "kissing tonsils." Grades 3 and 4 cause difficult breathing

3- Laryngopharynx (hyopoharynx):



Open Anterior to the larynx
Above: the upper border of the epiglottis
Below: lower border of cricoid
Pyriform fossa
valleculae: between tongue and epiglottis contains the pyriform fossa

4 PHARYNX

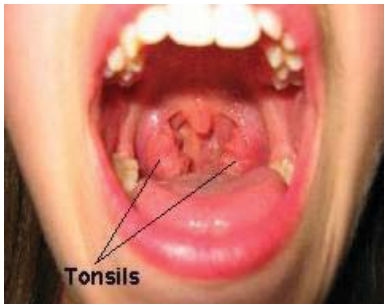
STRUCTURE OF THE PHARYNX: Fibromuscular tube, four layers:

1-MUCOUS MEMBRANE:

- Ciliated epithelium
- Stratified squamous epithelium
- Transitional epithelium
- Subepithelial lymphoid tissue of the pharynx (waldeyer's ring) : scattered rings of lymphoid tissue in lateral pharyngeal wall, in nasopharynx, in posterior wall

Palatine tonsils:

Some patients come complaining of small white tissue coming from their mouth and bad smell from the mouth (halitosis), without fever, sore throat or pain it is not tonsillitis --- it's the secretion of the crypt. Some patients will need tonsillectomy.
tonsils cannot reoccur.



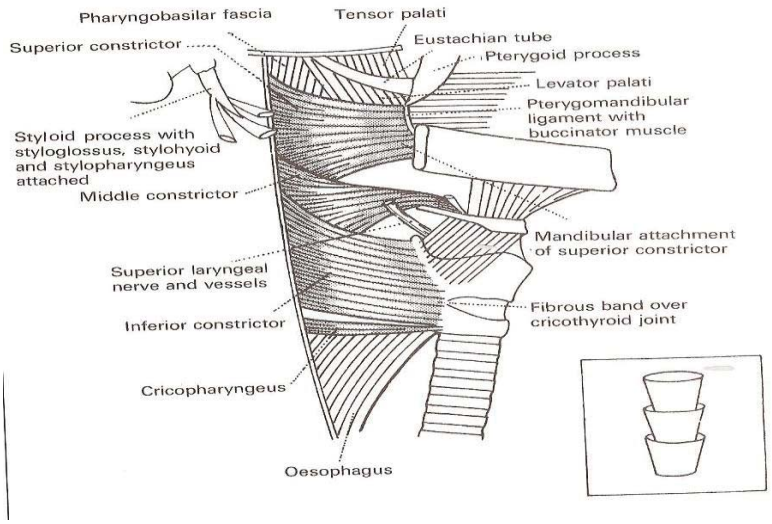
(12-----15 crypt)
The deep surface is separated from the constrictor muscles of the pharynx by connective tissue' capsule'
So when we do tonsillectomy: we dissect the capsule and remove the tonsil (be in the capsule away from the muscles)
Adenoid - No capsule
Lingual tonsils
Tubal tonsils
Lateral pharyngeal bands
Discrete nodules

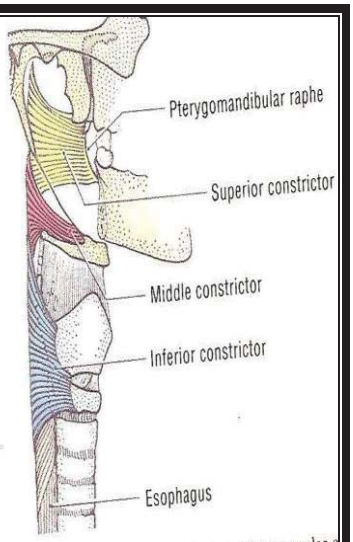
Halitosis is a common complaint:
Differential Diagnosis:

1- Teeth cavity
2- Diverticulum
3- Resent tonsillitis
4- Tongue (brush it)
5- Heart burn
6- Post nasal drip/discharge

2-Pharyngeal Aponeurosis

- Incomplete connective tissue coat in the lateral and posterior walls of the pharynx between the muscular layers (to strengthen the tube)
- Pharyngobasilar fascia: fascia that comes from the base of the skull covering the gap and the muscles giving them more strength.

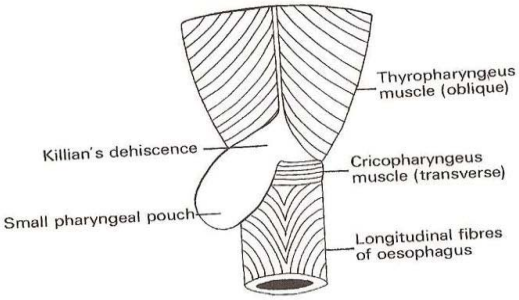




3-Muscular Coat:

A. **External :** Three constrictor muscles:
   1. **Superior constrictor:** arise from pterygoid, ptergomandibular ligament post end of mylohyoid fibers
   2. **Middle constrictor:** Arise from the hyoid bone and stylohyod ligament
   3. **Inferior constrictor:** 2 parts
      i. **Thyropharyngeus** : from thyroid
      ii. **Cricopharyngus :** from cricoid

Killian's Dehiscence: Potential gap between the thyropharyngus and cricopharyngus where the mucosa may herniate forming a small pharyngeal pouch, some food may get into it and later regurge. The pouch needs to be removed by diverticulectomy.

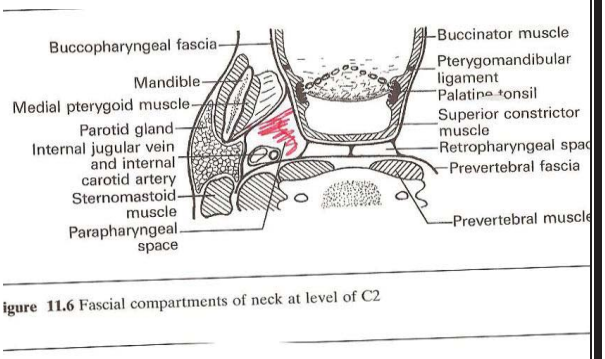


B. **Internal:** Three muscles:
   1. **Stylopharyngus**
   2. **Salpingopharyngus**
   3. **palatopharyngus**

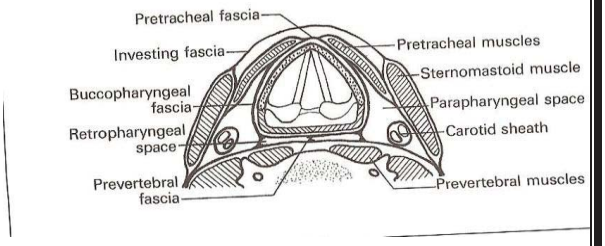
Buccopharyngeal Fascia: covering the muscles from the outside giving more strength to the pharynx

Relations of the Pharynx:

- **Posteriorly : prevertebral fascia**
- **Anteriorly: Parapharyngeal space:** Potential space lies outside the pharynx, Triangular in cross section , it extend from the base of the skull above to the sup mediastinum and apex of hyoid bone
  - Anteromedial wall: buccopharyngeal fascia
  - Posteromedial wall : cervical vertebrae, prevertebral muscle and fascia
  - Lateral wall: (up) the mandible ,tergoid muscle, parotid gland
    (Lower) sternomastoid muscle
- Compartment: divided into two parts by styloid process:
  - prestyloid: internal maxillary artery, fat, inferior alveolar, lingual, and auricultemporal nerves.
  - Poststyloid: neurovascular bundle (carotid artery, internal jugular vein, sympathatic chain ,CN IX,X and,XI



igure 11.6 Fascial compartments of neck at level of C2



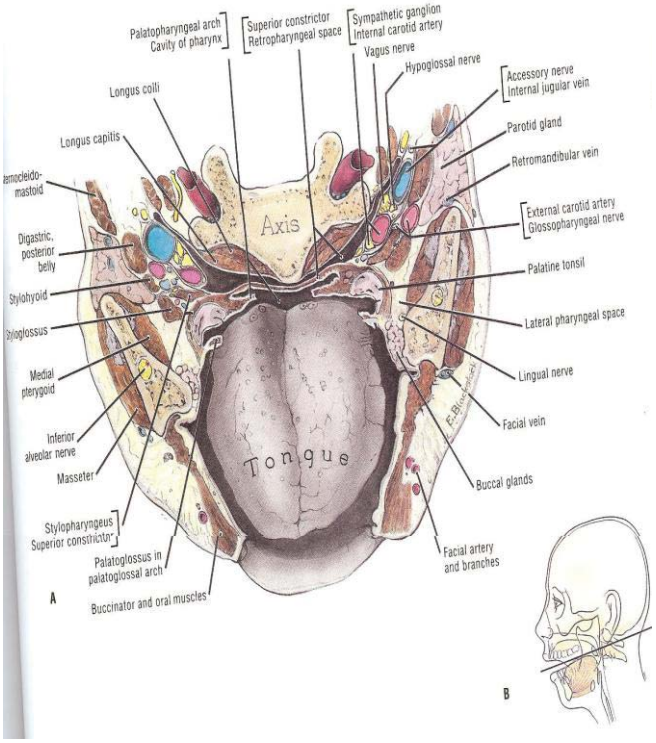
** If tonsillitis and not treated well they might get complicated by peritonsillar abscess in the Parapharyngeal Space leading to rupture of carotid or cranial nerve injury …. So it is dangerous if you don't treat it. MUST BE DRAINED

6 PHARYNX

Retropharyngeal space: It extends from the base of skull to super mediastinum
Lies behind the pharynx
Contains only lymph nodes

- Ant: posterior pharyngeal wall and its covering buccopharyngeal fascia
- Post: cervical vertebrae and muscles and fascia

Contents: Reteropharyngeal lymph nodes
Scenario: A 50 year old patient with torticollis and bulging in posterior pharyngeal wall (seen when opening mouth) should take it seriously because it is a site of lymph nodes so think of TB IF adult; in child think of tonsillitis that spread to this space.



Physiology:
Functions of the sub epithelial lymphoid tissue:

→ **Protective functions:**

- Formation of lymphocytes
- Formation of antibodies
- Acquisition of immunity
- Localization of infection

→ **Salivation:**

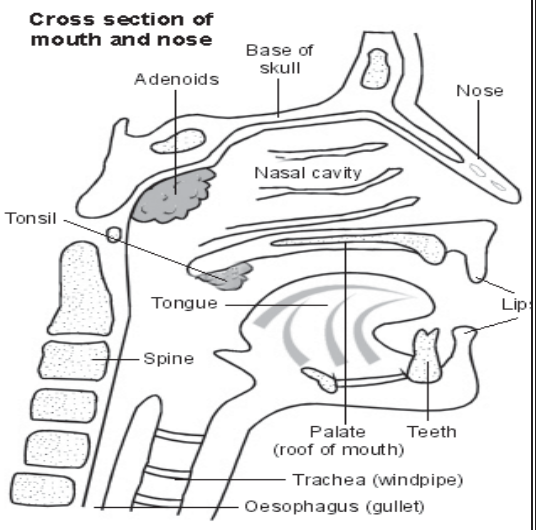
→ **Deglutition:** Three stages

1. ***Oral stage*:** voluntary, closure of mouth, cessation of respiration ,rising of larynx ,sudden elevation of the tongue, press the tongue against the palate, and pushes it backwards towards the oropharynx
2. ***Pharyngeal Stage*:** nonvoluntary reflux, contraction of nasopharynx sphincter, larynx rises more, laryngeal inlet closure, epiglottis diverts the food into cricopharyngeal sphincter, contraction of constrictor muscles, relaxed cricopharyngeal sphincter.
   If the soft palate is not closed by the nasopharynx the food will get regurgitated through the nose so you must check for nasopharyngeal imcompetence and the patient will complain of hypernasality.
   -if sphincter is not functioning properly food will remain in the airway

3. ***Esophageal Phase***

-

→ **Respiration**
→ **Speech**
→ **Resonating cavity** any mass in the mouth will affect the resonation of your voice
→ **Articulation**
→ **Taste:** taste buds in the oral cavity



Adenoid: A hypertrophy of the nasopharyngeal tonsil to produce symptoms, most commonly between the ages of 3 to 7 years, most common cause of nasal obstruction in children. Very common in pediatrics but may regress after the age of 7. We only remove it if it causes sleep apnea or secretory otitis media.

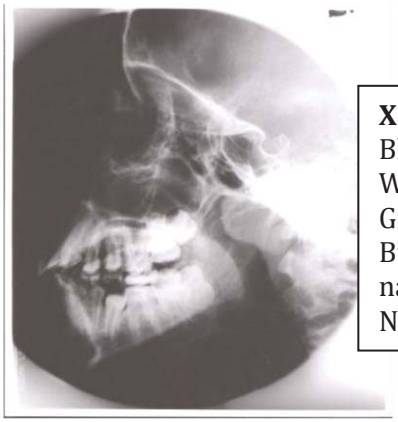
Pathological types:
1- simple inflammatory
2- tuberculosis

Clinical features: Mouth breathing, snoring, sleep disturbance, toneless voice, adenoid face (mickey mouse face don't say it especially in rounds!!), nasal discharge, Eustchain tube obstruction if big enough because it opens in the nasopharynx, so interferes with the aeration of middle ear and it is not equalizing the outside and inside pressure secretory otitis media (glue ear) affecting hearing- adenoid face.
How do you see the adenoid: introduce the scope through the nose.

- Grade the obstruction by adenoid grade 1 : 25%
- grade 2 : 50%
- grade3 : 75%
- grade 4: 100%

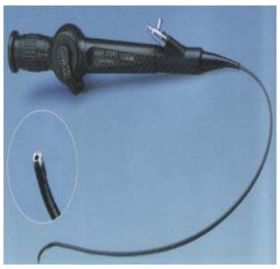
Diagnosis:

- X ray: if no endoscope, you follow air column if there is a cut in it then adenoid, to comment on xray neck must be extended and at the end of inspiration so difficult but sometimes for medico-legal reasons its necessary.



X-Ray:
Black air
White bone
Grey soft tissue
Bulge at nasopharynx
Narrowing of

- Flexible Fiberoptic: better than the X-Ray because there is no radiation and could see the exact grade of the adenoid.



Treatment:

- if small adenoid: conservative steroidal nasal spray
- Surgical: adenoidectomy if complete obstruction

8 PHARYNX

SLEEP APNEA AND SNORING:

Snoring is a sign of partial obstruction of the upper airway during sleep
Snoring is always present during type of sleep apnea
Sleep apnea: Cessation of airflow at the mouth and nostrils lasting 10 seconds for at least 30 apnoeioc episodes (OSCE)
Types:

- **Central sleep apnea:** Failure of respiratory drive from the brain, no movement of the chest , should be treated by neurologist
- **Obstructive sleep apnea (OSA):** Due to anatomical narrowing of the upper airway due to adenoid , large tonsils, his chest is moving
- **Mixed**

If left untreated will develop Cor Pulmonale, pulmonary edema, heart failure, and or HTN; they do not go into deep sleep so hormonal changes

Sleep Apnea is diagnosed by sleep study.
Stage of sleep:

- Slow wave sleep : Brain waves are slow deep restful sleep decrease in vascular tone and respiratory rate and basal metabolic rate
- Rapid eye movement : Brain quite active active dream

Pathophysiology of OSA:

- During REM or deep sleep ,obstructive occurs resulting in decrease arterial oxygen and increased arterial carbon dioxide pressure
- Nocturnal desaturation(decreased oxygen) arouses patient and causes increase pulmonary artery, systemic arterial pressure
- lead to hypersomnolence

Investigation:
Sleep study: EEG, EKG, EOG, pulse oximeter, respiration rate, and nasal and oral air flow for 7 hours. The number of times apnea hypopnea happens, they calculate the apnea hypopnea index and decide whether the case is mild, moderate or severe obstructive apnea, or if it is not apnea at all.

Treatment:
Nonsurgical :

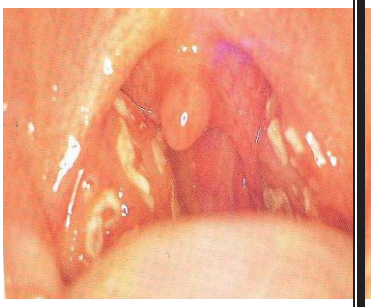
- behavior modification : if excess weight must lose weight, don't eat before sleeping
- medical treatment : nasal spray
- CPAP: continuous positive air pressure (causes driness)

Surgical :
UPPP : uvulopalatopharyngioplasty, it is very painful.

Diseases of Oropharynx

A. ACUTE INFECTION OF OROPHARYNX

1. ACUTE TONSILLITIS:

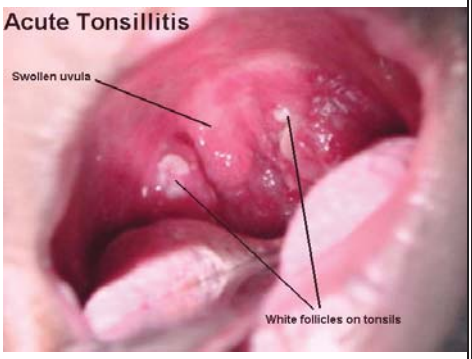


Causes:

- viral infection mostly, followed by bacterial
- bacterial group AB-hemolytic streptococcus, Moraxella, H. influenza, Bacteroides

Signs and symptoms: fever, sore throat, odynophagia, trismus, halitosis, and dysphagia

Phases: erythema (tonsils enlarge), exudative, follicular tonsillitis
Serious and needs to be treated. Give fluids

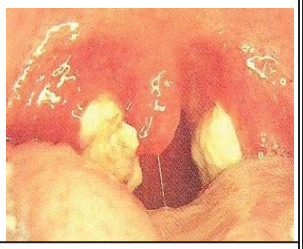


Complication:

- peritonsillar abscess,
- parapharyngeal abscess,
- retropharyngeal abscess,
- rheumatic fever (palpitations)
- glomerulonephritis (comes with flank pain)

Treatment of Acute Tonsillitis: oral antibiotics, bed rest, hydration, analgesia
If patient does not respond well to oral antibiotic we admit and give IV antibiotic and fluids.

2. INFECTIOUS MONONUCLEOSIS (VIRAL)



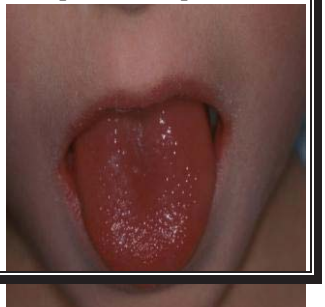
Pathogen: Epstein barr virus
Signs and Symptoms: fever, sore throat, lymphadenopath malaise, exudative tonsilitis, hepatosplenomegaly,white membreane of tonsils
Diagnosis:

- monosopt test
- paul bunnel test (heterophil antibodies in serum)
- white membrane (yellowish white) over the tonsils
- bilateral lymphadenopathy in the neck
- in children: hepatosplenomegaly
- 80% mononuclear and 10% atypical lymphocytes on smear
- CBC: mainly lymphocytes

Yellowish – white membrane over the tonsils

**Differs from tonsillitis by the white membrane over the tonsils and the systemic involvement (hepatosplenomegaly and lymphadenopathy).

Complications: cranial nerve involvement, meningitis, autoimmune Hemolytic A., splenic rupture
Treatment: hydration, analgesia oral hygiene Child should be kept at home for supervision; antibiotic is not needed as this is viral.



3. SCARLET FEVER: (OSCE: fever + red tongue = Scarlet fever)

Endotoxin produced by type A beta-hemolytic streptococcus

10 PHARYNX

SSX: red pharynx, *strawberry tongue*, perioral skin erythema and desquamation, dysphagaia ,malaise, severe cervical lymphodenopathy, fever, sore throat
Differential Diagnosis: Dick test
Treatment: Antibiotic

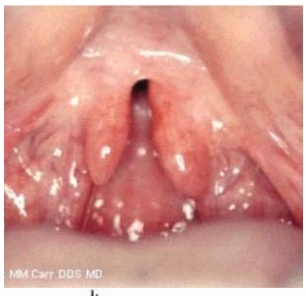
4. DIPHTHERIA:

it is a differential diagnosis of Infectious Mononucleosis
Organism: Corynbeactrium diphtheria
Signs and Symptoms: sore throat, fever, green plaques friable membrane, white membrane on the tonsils
Diagnosis: culture
Complications: nephritis, airway obstruction, death
Treatment: Antibiotic, antitoxin
No cases of diphtheria due to vaccinations.

5. VINCENT'S ANGINA:

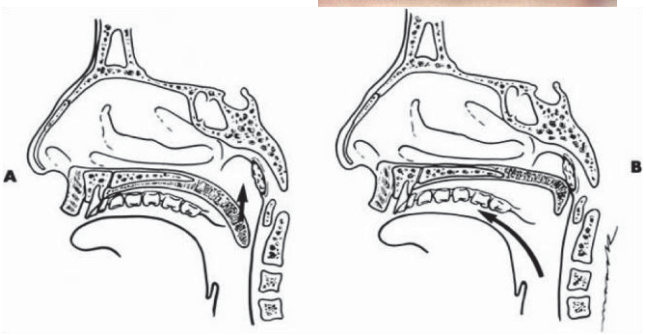
Acute ulcerative lesion (ulcer on tonsils causing severe pain)
Gram negative fusiform bacillus and a spirillum with anaerobic
Signs and Symptoms: Sudden in onset, severe pain, fever, cervical adenitis, the base of the deep ulcers bleeds when the membranous slough is removed; the symptoms subside in 4—7 days.
Treatment: Metronidazole (flagyl), antiseptic, and mouthwash

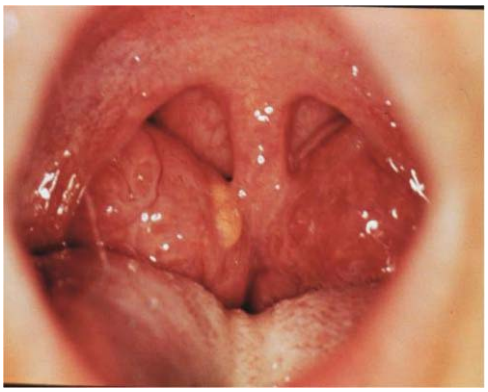
6. BIFID UVULA:



Signs and symptoms: snoring and mouth breathing
Sometimes the adenoid helps close the soft palate. Before deciding on doing an adenoidectomy the doctor has to make sure the uvula is not short, there is no submucoidal cleft, and no bifid uvula: any of those 3 is a reason to not do the adenoidectomy. If the adenoidectomy is done it will cause hypernasality and regurgitation from the nose.

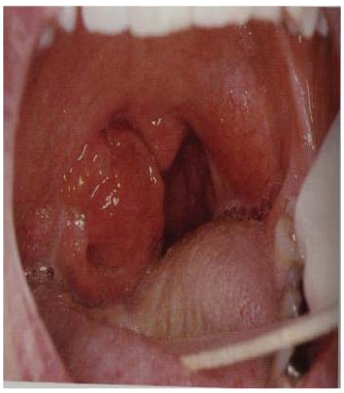
Therefore it is contraindicated. However if necessary do partial adenoidectomy: remove the part against the nose but keep the oral part because it supports the defect in the soft palate.

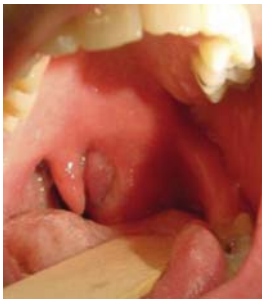




Left picture: Kissing tonsils grade 4 tonsils.

Right picture: unilateral tonsilar enlargement.





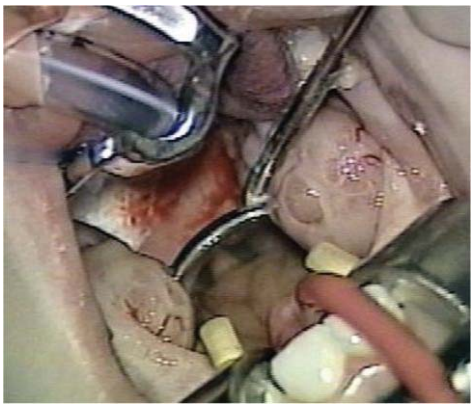
TONSILLECTOMY: under GA

In Down Syndrome patients they have neck sublaxation so must be gentle and keep that in mind during the surgery to prevent injuring them

Adenoid is approached from the oral cavity.

Indications for tonsillectomy:

1- If recurrent: 6-7 attacks per year, 4 attacks per year for 2 years, or if 3attacks per year for 3 years.
2- snoring, mouth breathing, causing obstruction (hypertrophied tonsils)
3- asymmetrical tonsilar enlargement suspecting malignancy (smoker or not)
4- Peritonsillar abscess (quinsy) (mostly unilateral) fever, and dysphasia. Has hot potato voice due to the big mass. Abscess collection between lateral wall and capsule, so the tonsil is pushed to the midline, pushing the uvula also. Treat it by incision and drainage, but if recurrent tonsillitis and peritonsillar abscess you treat the abscess, wait for 6 weeks then do the tonsillectomy.



Procedure:

- **Cold technique:** knife and scissors, adenoid is one of its indications.
- **Hot technique:** laser or lasek, suction diaythermy

Adenoids can reoccur because they do not have capsules but not commonly

Complications of Tonsillectomy:

1. Hemorrhage (most common)

- ***Primary hemorrhage:*** Bleeding occurring during the surgery
  - **Causes**
    1. Bleeding tendency
    2. Acute infections
    3. Bad technique
  - **Management**
    1. General supportive measures
    2. Diathermy, ligature or stitches

3. Packing

- ***Reactionary hemorrhage:*** within the first 24 hours postoperative period

➢ **Causes**

1. Bleeding tendency
2. Slipped ligature

➢ **Diagnosis**

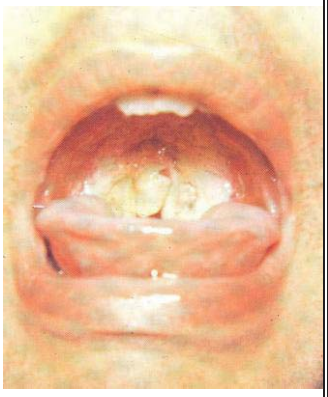
1. Rising pulse & dropping blood pressure
2. Rattle breathing
3. Blood trickling from the mouth
4. Frequent swallowing
5. Examination

➢ **Treatment**

1. General supportive measures
2. Take patient back to OR
3. Control like reactionary hemorrhage

- ***Secondary hemorrhage:*** very important

Occur 5-10 days postoperatively
Due to infection
Treated admit and give 2 IV antibiotics
May need diathermy or packing
Slough tissue which is white forms after few hours after the surgery and may stay for 2-3 weeks so the doctor must inform the mother or any other caregiver about it or else they may think its abscess. We need to instruct them about the food habits needed postop to help with the healing process. The caregiver needs to be well informed about the food, the antibiotics and the analgesia (they need regular analgesia). They also need to be well informed about the slough tissue so that they would not think it's a complication and change the patient's medications or worry.



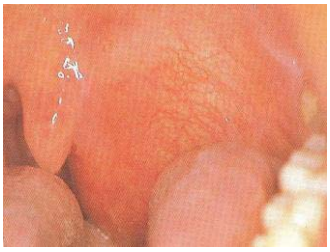
2. Respiratory obstruction
3. Injury to near-by structures
4. Pulmonary and distant infections

7. MONOLIASIS:

White patches (thrush) caused by candida albicans fungus. in bronchial asthma patients(using inhaled steroids), or immunocompromised patients. Such as patients on renal dialysis.

Treatment: nystatin gel can gurgle with it then swallow it.

8. PERITONSILLAR ABSCESS (QUINSY):



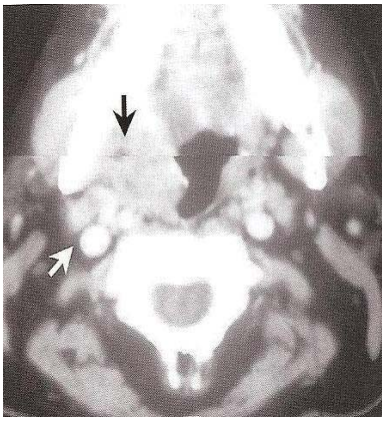
It is an abscess between the tonsil capsule and the adjacent lateral pharyngeal wall.

Signs and Symptoms: fever, otalgia, odynophagia, uvular deviation, trismus, and drooling of saliva, the patient looks sick and dehydrated.

Complication: Para and retropharyngeal abscess, aspiration pneumonia

Treatment: Incision and drainage done in the ER, aspiration, and are given IV antibiotic.

9. PARAPHARYNGEAL ABSCESS:



Source of the infection: odontogenic ,tonsils, , parotid , teeth

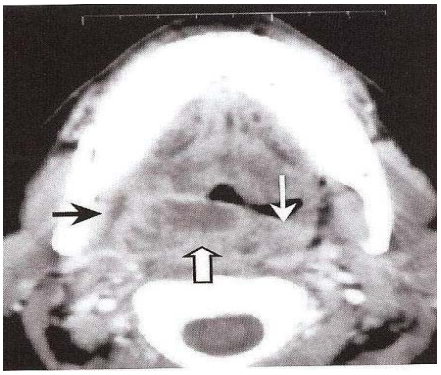
Signs and Symptoms: trismus, fever, muffled voices, intraoral bulge

Complication: aspiration, cranial nerve palsy 9, 10, 11 - airway compromise, septic thrombophlebitis, carotid blowout, endocarditis

Treatment: external drainage, IV antibiotic, airway management is very important,

In picture: axial CT scan, with contrast, white blood vessels, patient has history of tonsillitis, fever, trismus, can't open his mouth, and neck swelling. You can see abscess in the paraphyaryngeal space (dangerous area). The abscess may rupture causing aspiration inside the mouth; the puss will go into the airway causing obstruction and the other complications.

10. RETROPHARYNGEAL ABSCESS:



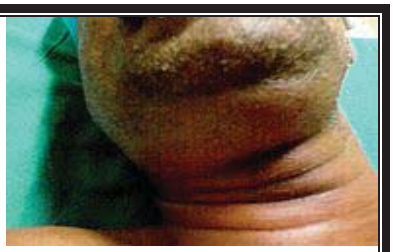
More common in children

Signs and Symptoms: odynophagia, hot potato voice, drooling stiff neck, fever, and stridor.

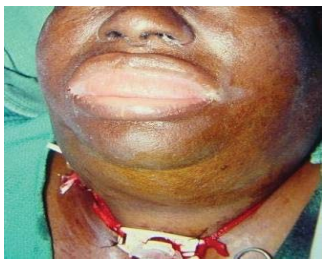
Complications: mediastinitis, respiratory distress, and ruptured abscess

Treatment: drainage, IV antibiotic, airway management is very important

Picture shows both retro- and para- pharyngeal abscesses. The black is the airway. Patient is barely breathing and might need a trachestomy



11. LUDWIG'S ANGINA:



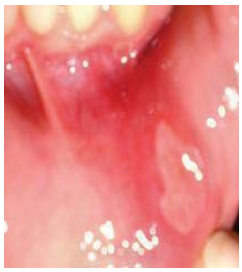
Bilateral cellulitis of submandibular and sublingual spaces
Signs and Symptoms: wooden floor of the mouth due to cellulitis, neck swelling and indurations, can't open their mouth, drooling, respiratory distress ,swollen tongue ,dysphagia and trismus.
Complications airway distress, sepsis
Treatment: tracheotomy, external drainage, IV antibiotic
Top emergency

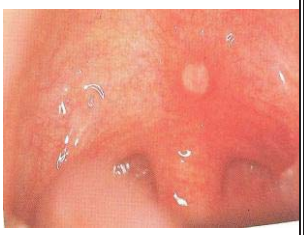
12. CHRONIC PHARYNGITIS:

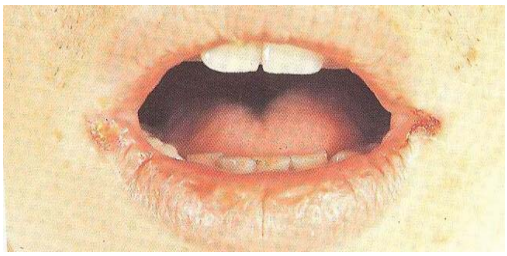
Pathogenesis: postnasal drip, irritant (dust. Dry heat, smoking, alcohol), reflux, esophagitis chronic mouth breathing ,allergy granulomatoues disease connective tissue disease , malignancy
Signs and Symptoms: Constant mouth clearing, dry throat pharyngeal crusting, thick granular wall
Treatment: address underlying etiology

13. APHTHOUS ULCER: caused by stress, dehydration…

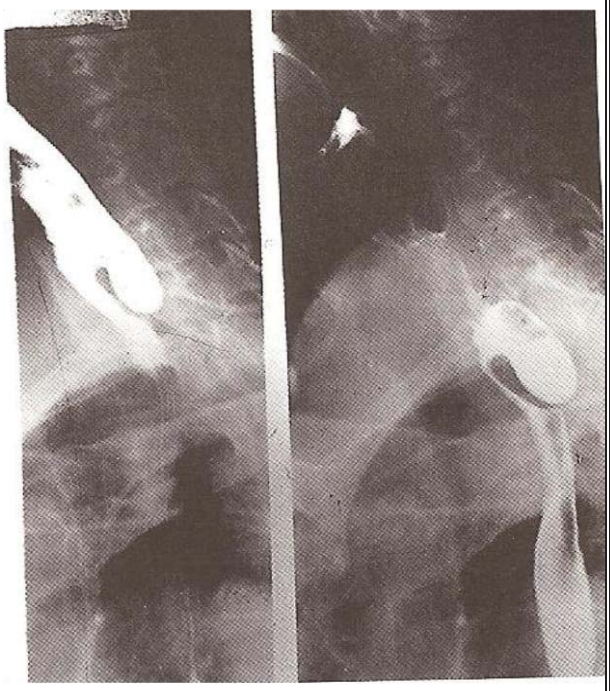
Probably has family history …
Tx: goes spontaneously.







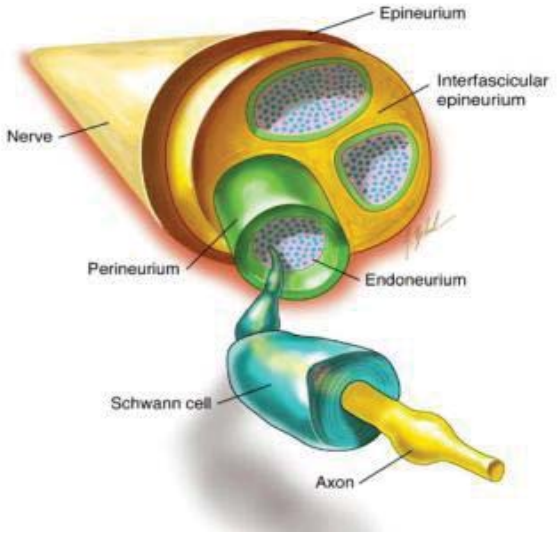
14. ZANKER'S DIVERTICULUM:



Herniation of the mucosa at killian's triangle due to increase intraluminal pressure
Signs and Symptoms: dysphagia, regurgitation of
Diagnosis: Undigested food aspiration do barium swallow
Treatment:

- excision by endoscope or surgery
- Cricopharyngeal myotomy.
- Diverticulectomy

FACIAL NERVE VII



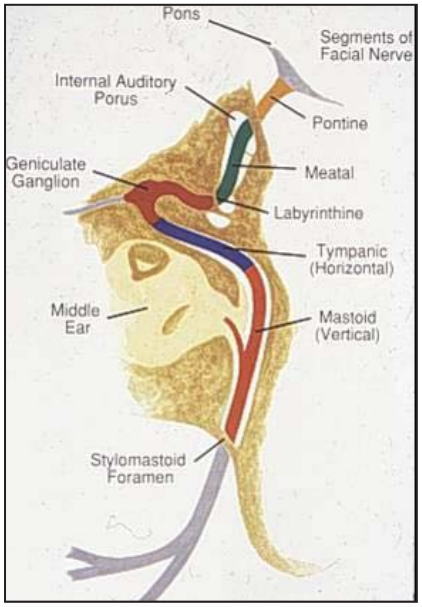
Objectives: *Anatomy (course + branches) + *Causes of facial palsy (congenital, traumatic, middle ear complication , Ramsy Hunt's syndrome and bell's palsy)

Sources: the same as 429 lecture notes with the addition of some pictures from the doctor's slides.

GOOD LUCK..

ASMA ALHEDAITHY

The Facial Nerve VII



FACIAL NERVE FIBERS:

- **Motor** > to the stapedius and facial muscles.
- **Secreto-motor** > to the submandibular, sublingual salivary glands and to the lacrimal glands.
- **Taste** > from the anterior two thirds of tongue and palate (Chorda Tympani).
- **Sensory** > from the external auditory meatus.

ANATOMICAL DIVISIONS:

- **Intracranial :** includes Nuclei+ Cerebellopontine angle
- **Cranial** (intratemporal)
- **Extracranial** (extratemporal) : after leaving stylomastoid foramen.

(Course of Motor fibers)

1. Intracrainal part:

It has the nuclei WHICH ARE:

- Superior salivary nucleus (**Parasympathetic)**
- Motor Nucleus (most part of the facial nerve)
- Nucleus tractus solitarii (Solitary Nucleus) > (**taste**)

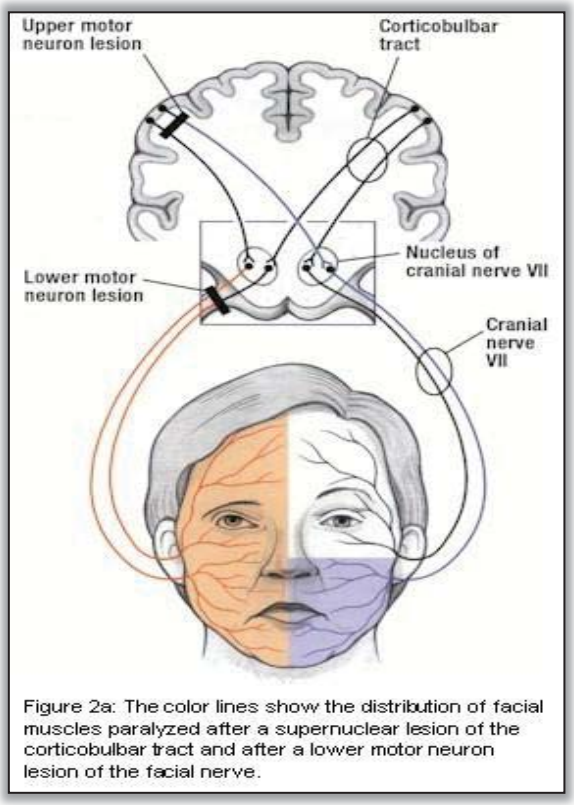
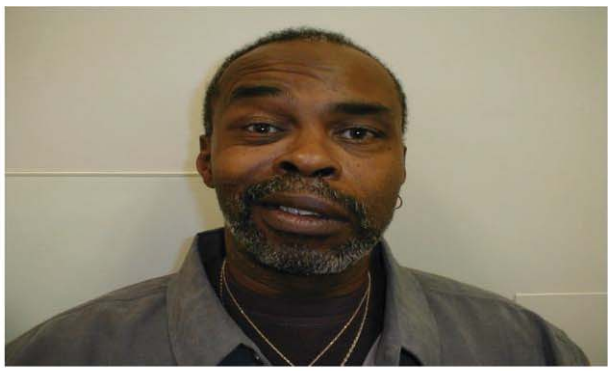


Figure 2a: The color lines show the distribution of facial muscles paralyzed after a supernuclear lesion of the corticobulbar tract and after a lower motor neuron lesion of the facial nerve.

---Upper part of the motor nucleus receives fibers from both cerebral cortexes.
--- Lower part of the motor nucleus receives fiber from the contralateral cerebral cortex.

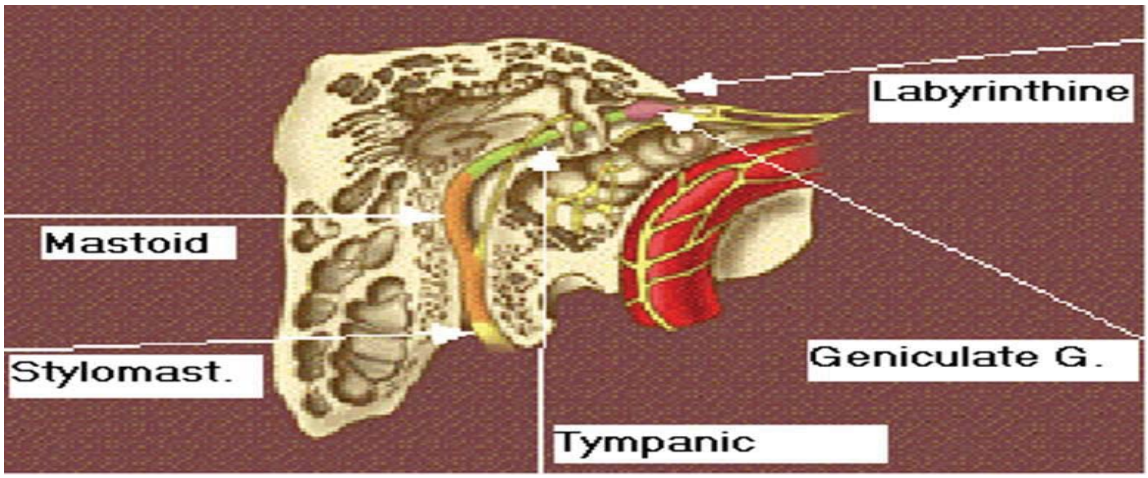
- **Upper motor lesions** spare the upper facial muscles (frontalis M. and Orbicularis Occuli M.) and affect the contralateral lower face.
- **Lower motor lesions** affect all the ipsilateral (Whole half) facial muscles.

UPPER MOTOR

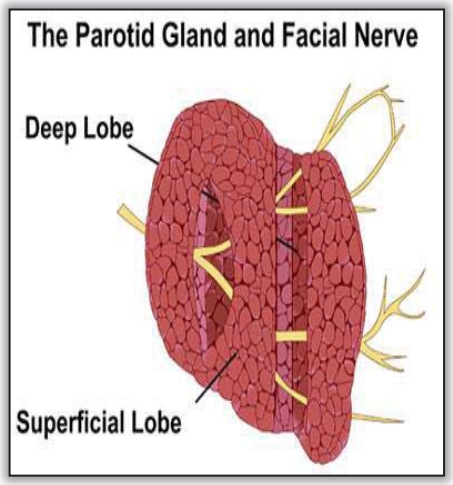


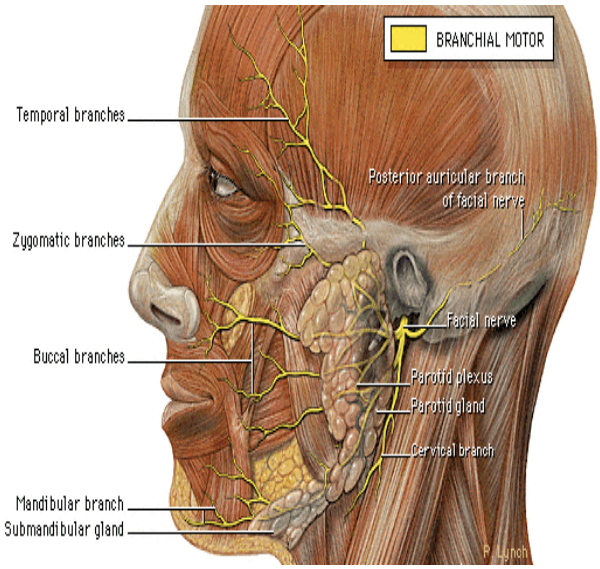
LOWER MOTOR

2. Intratemporal part:
From the Internal Auditory Meatus (association with **vestibulocochlear nerve**). Then goes to Fallopian canal.
Fallopian Canal has a horizontal part and a vertical part. AND its divided to:
- **Labyrinthine:** Related to the inner ear.
- **Tympanic:** Related to the middle ear.
- **Vertical part (mastoid):** Related to the external ear (Branches: 1-Supplies Stapedius Muscle 2-Chorda Tympani).



3. Extracranial part:
After it leaves the temporal bone through the **Stylomastiod foramen**, it goes through the parotid gland.
* In the parotid gland it divides into its **FIVE** branches which are:
- **Temporal:** Supplies frontalis
- **Zygomatic:** orbicularis oculi (most important branch) responsible for closure of the eye.
- **Buccal:** buccinator
- **Mandibular:** orbicularis oris
- **Cervical:** Platysma





Branches of the Facial Nerve

(Course of Secreto-motor [parasympathetic] + taste)

Parasympathetic fibers: (Superior Salivary Nucleus) Some fibers go with motor fibers and some fibers leave the facial nerve at the Geniculate ganglion and goes to the lacrimal gland and some other glands.

The other secreto-motor fibers leave the vertical part of the facial nerve through the Chorda Tympani, which crosses the middle ear.

The Chorda Tympani then joins the **Lingual** nerve and innervates the two salivary glands: the submandibular gland and sublingual gland.

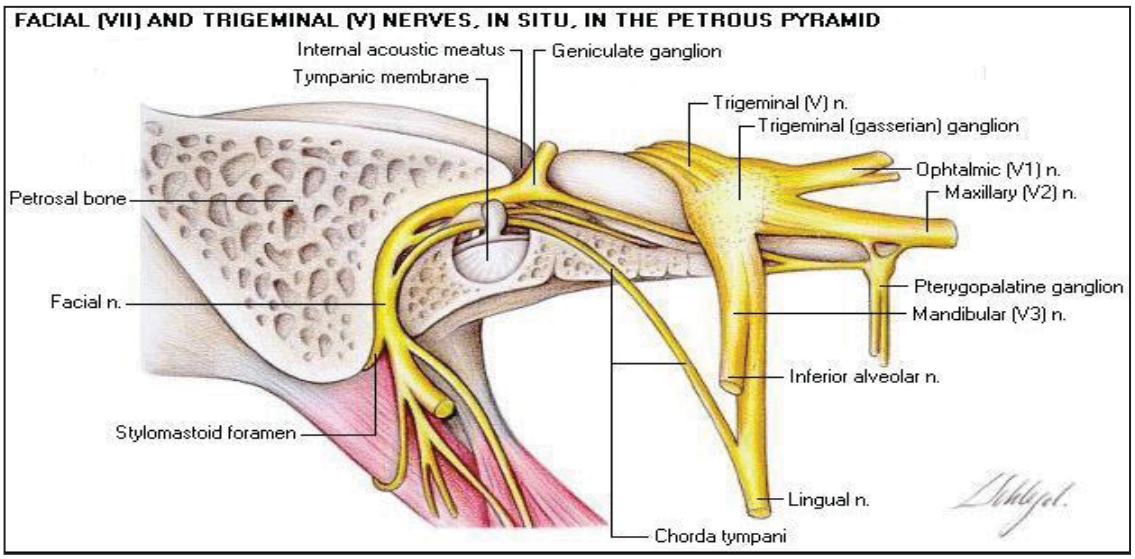
Taste: takes the same course of the Parasympathetic but in the opposite way (from peripheral to central).

* Fibers from the palate goes to the Geniculate ganglion> nucleus solitarii.
* Fibers from the anterior 2/3 of the tongue> chorda tympani> nucleus solitarii.

NOTE: This is important to know the level of the lesion of the facial nerve.

For Example:

- If the lesion is above the level of the chorda tympani> patient will loose the function of chorda Tympani he will have dryness and loose of taste sensation.
- If the lesion is below the level of chorda tympani> patient will have only facial paralysis with normal taste sensation.



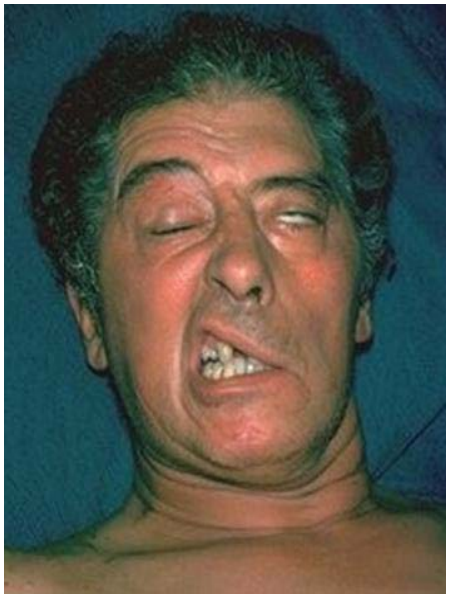
VARIATIONS AND ANOMALIES :

Only one normal variation the doctor talked about, which is:

- At the junction of the Tympanic vertical part there is: (dehiscent) no bony coverage of the nerve (no protection). It's a common anomaly.

This subjects the facial nerve to infection and trauma in the middle ear.

CLINICAL MANIFESTATIONS



- **Paralysis of facial muscles**
  - Asymmetry of the face
  - Inability to close the eye
  - Accumulation of food in the cheek
- **Phonophobia>** Stapedius muscle (LMNL).
- **Dryness of the eyes>** impaired drainage.
- **Loss of taste >** anterior 2/3 of the tongue.

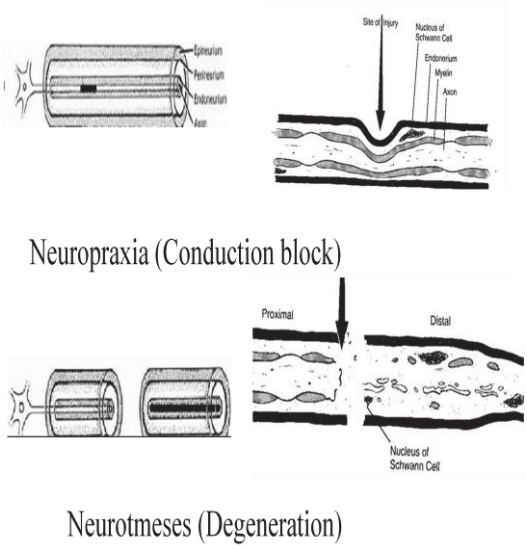
Pathologies of facial nerve injury:

Facial nerve paralysis could be:

1- Conduction block (Neuropraxia) : due to mild trauma, nerve is affected functionally only and there is no degeneration anatomically. this one you expect complete recovery if the cause was removed.

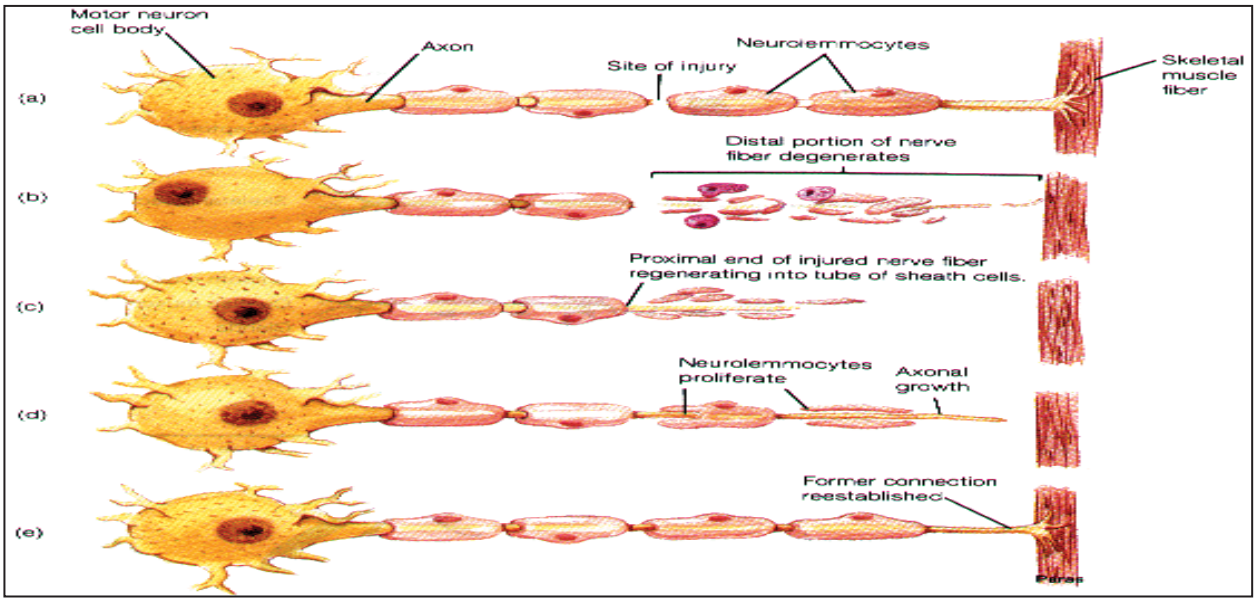
2- Degenerative (Neurotomesis) : you have to bring the nerve together (bring distal part to proximal part), recovery is much slower. The rate of regeneration is 1mm/per day. (Distal part will have degeneration)

3- Mixed



Neuropraxia (Conduction block)

Neurotmeses (Degeneration)



Principles of management of facial nerve paralysis:

• Care of the eye.
• Treatment of the cause if applicable.
• Treatment of the nerve varies according if the paralysis is partial or complete.

A) Partial facial paralysis:

Being partial means that the nerve fibers are in continuity and recovery is expected by **conservative** treatment (e.g. removal of pressure, steroid etc) Remove the cause only, NO SURGERY.

B) Complete facial paralysis:

- If it is due to **neuropraxia**, recovery is expected by conservative treatment.
- If it is due to **degeneration (Neurotmeses)** , surgical treatment is required.
- To differentiate between degeneration and neuropraxia **electrophysiological tests** are required

Electrophysiological Tests:

- **Nerve Excitability Test (NET)**
- **Electroneurography (ENoG)**

- It detect degeneration of the nerve fibres .
- Useful only 48-72 hours following the onset of the paralysis.

Nerve excitability test (NET): The current's thresholds required to elicit **just-visible muscle contraction** on the normal side of the face are compared with those values required over corresponding sites on the side of the paralysis.

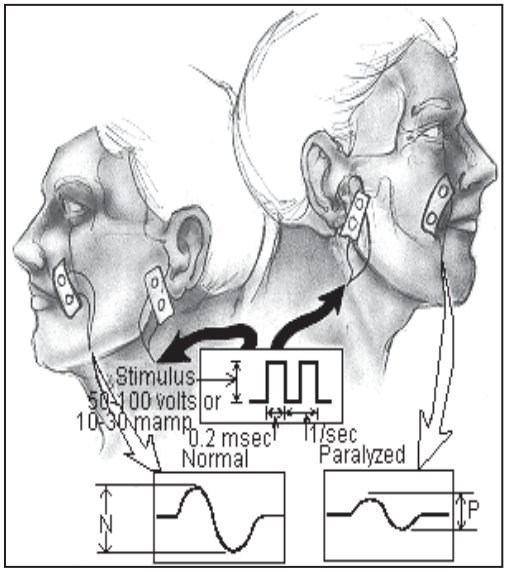
- **Advantage:** Simple, available everywhere
- **Disadvantage:** Subjective only (rough estimation either you see it move or not)

Electroneurography (ENoG):

***Compares compound action potential of both sides**
-Stimulate nerve at stylomatoid foramen.
-Measure muscular response near nasolabial groove

*Less intact motor axons with Wallerian degeneration.
*Worse prognosis with rapid degeneration.
*Inaccurate within first 3 days of Bell's palsy onset.
*Quantitative analysis, observer independent

Advantage: More accurate measurements, Objective
Disadvantage: Not available as common as NET



Interpretation of the tests:

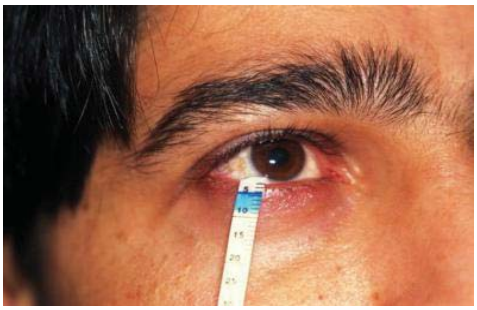
*Not useful in the first 48 – 72 hours
*After 48-72 hours (the time required for degeneration to take place)
-**Normal results** means that there is no degeneration **(Neuropraxia)**
-**Abnormal** results means **degeneration** .

*So, If the patient is presenting already with partial paralysis this test will be waste of time because you already know that there is some fibers acting and some are not!

For partial you do:

Topognostic tests : Indicated in some cases to locate the site of injury:

- **Schirmer's test >** Test the lacrimation function
- **Stapedial reflex**
- **Taste sensation**



Causes of Facial nerve paralysis:

we can distribute it as Anatomical or Pathological

Anatomical:

-Intracranial causes
-Cranial (intratemporal) causes
-Extracranial causes

Pathological:

- Congenital: Birth trauma
- Traumatic: Head and neck injuries & surgery
- Inflammatory: O.M, Necrotizing O.E., Herpes
- Neoplastic: Meningioma, malignancy ear or parotid
- Neurological: Guillain-Barre syndrome, multiple sclerosis
- Idiopathic: **Bell's palsy** (most common)

Congenital Facial Palsy:

- 80-90% are associated with birth trauma
- 10 -20 % are associated with developmental lesions
- at the level of Stylomastoid Foramen; Because it is not well developed yet and the nerve is superficial there.

Inflammatory causes of facial paralysis:

A- Facial Paralysis in AOM (Acute Otitis Media)

- Mostly due to pressure on a dehiscent nerve by inflammatory products
- Usually is **partial** and **sudden** in onset
- Treatment is by antibiotics (treat otitis media) and myringotomy (drainage/relieve pressure)

B- Facial Paralysis in CSOM (Chronic supportive Otitis Media)

- Usually is due to pressure by cholesteatoma or granulation tissue
- Insidious in onset
- May be partial or complete
- Treatment is by immediate surgical exploration and "proceed"

C- Herpes zoster oticus (RAMSAY HUNT SYNDROME)

- Herpes zoster affection of cranial nerves VII, VIII, and other nerves
- Facial palsy, pain, skin rash, SNHL and vertigo
- Vertigo improves due to compensation
- SNHL is usually irreversible
- Facial nerve recovers in about 60% (Means that 60% of patients is having Neuropraxia and the other 40% is having Neurotomesis)
- Treatment by: Acyclovir, steroid and symptomatic

Traumatic facial injury:

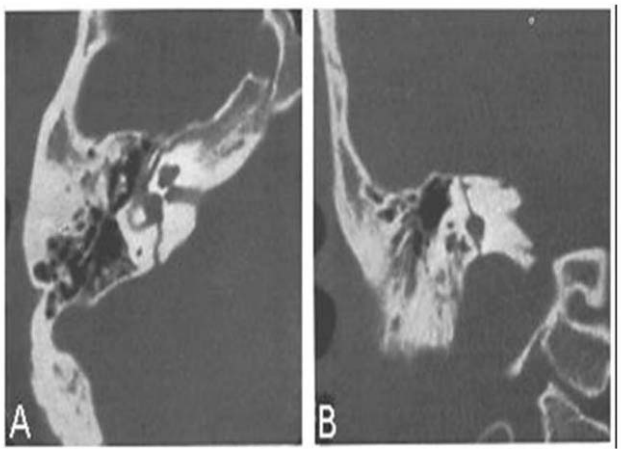
A-Iatrogenic (surgical) :
Operations at the CP angle, ear and the parotid Glands.

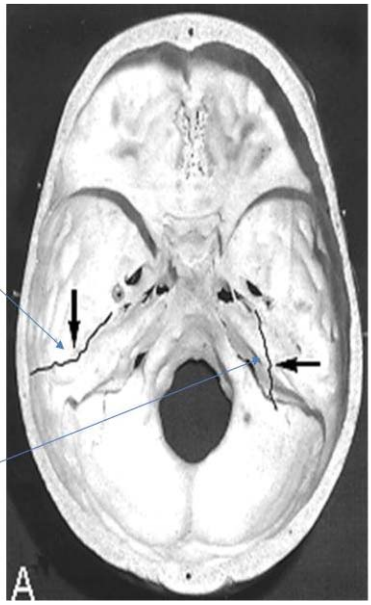
B-Temporal bone fracture:

1-Longitudinal: More common, Less severe, might cause conductive hearing loss, usually does not affect facial nerve.
2-Transverse: Less common, More severe, More likely to affect facial nerve.

*Pathology:
(Why the Facial Nerve get effected)?
Edema: will lead to partial paralysis
Transaction of the nerve: will lead to complete paralysis.

Transverse Fracture





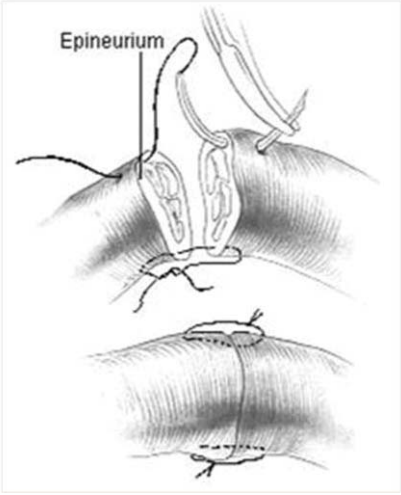
Management of Traumatic Facial Nerve Injury:

- If it is *delayed* in onset, it is usually <u>incomplete</u> and is due to edema
  - **Conservative**
- If of *immediate* onset, it is usually <u>complete</u> and due to transection of the nerve
  - **Surgical repair**

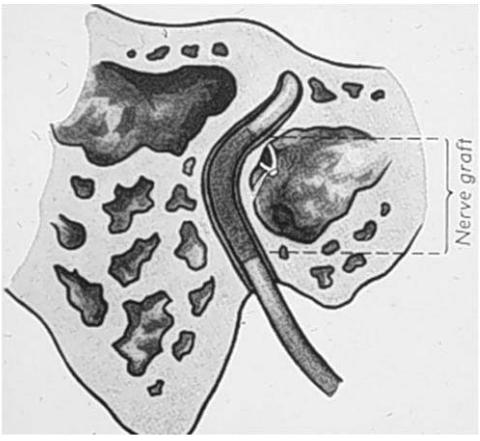
SURGICAL REPAIR:

1) **Direct Anastomosis** > No gap in nerve
2) **Nerve Graft** > Gap (From the great ocular Nerve)
3) **Nerve Transfer** > Distal part is there, but NO proximal (From the Hypoglossal Nerve; it is supplying the tong Motor but paralysis to ipsilateral to the tong is not going to effect it function that is why we use it)
4) **Muscle Flap** > NO distal and NO proximal (either Buccinators or temporalis)

1) Direct Anastomosis



2) Nerve Graft



3) Nerve transfer (anastomosis)

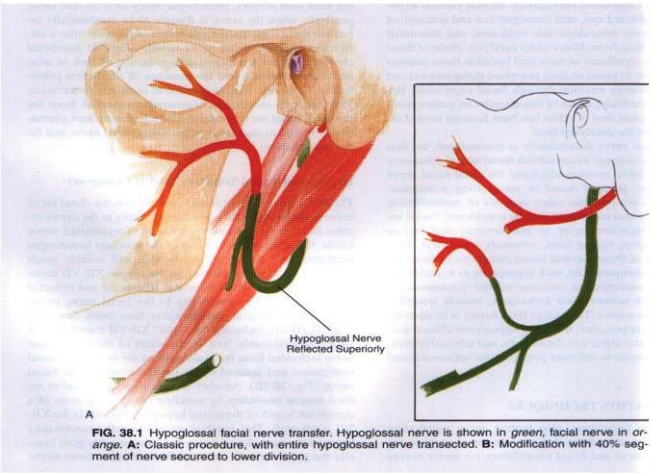
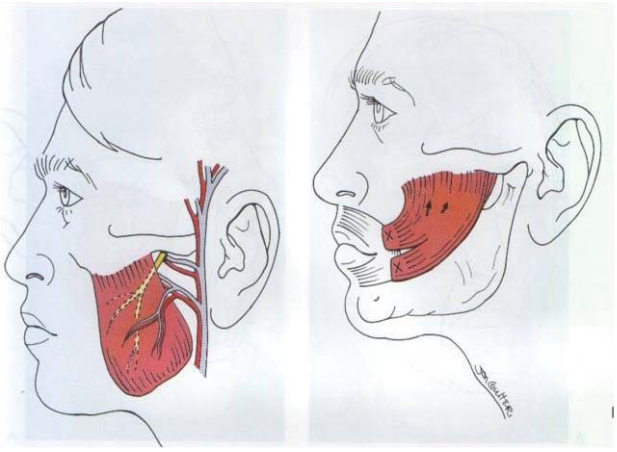


FIG. 38.1 Hypoglossal facial nerve transfer. Hypoglossal nerve is shown in *green*, facial nerve in *orange*. **A:** Classic procedure, with entire hypoglossal nerve transected. **B:** Modification with 40% segment of nerve secured to lower division.

4) Muscle flap



BELL'S PALSY

• Most common diagnosis of acute facial paralysis
• Diagnosis is **by exclusion** (exclude all other causes of face paralysis)

***Pathology:** Edema of the facial nerve sheath along its entire intratemporal course (Fallopian canal)

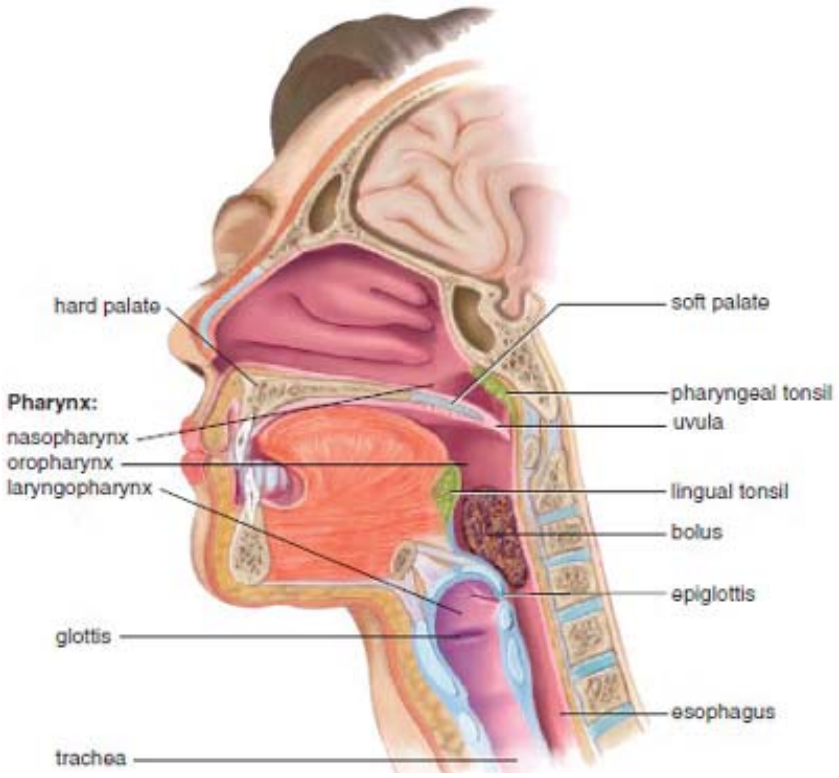
***Etiology:** Two theories: Viral vs Vascular

***Clinical features:** (Don't diagnose it at least it's typical)
• Sudden onset unilateral FP
• Partial or complete
• No other manifestations apart from occasional mild pain
• May recur in 6 – 12%

***Prognosis:**
• 80% complete recovery
• 70%satisfactory recovery
• 10% no recovery

***Treatment:**
• Reassurance
• Eye protection
• Physiotherapy
• Medications (steroids, antivirals,vasodilators)
• Surgical decompression in selected cases.

***Surgical Management:**
• Debate over years
• Patients with 90% degeneration
• Within 14 days of onset



Communication and Swallowing I & II

429 ENT Team (F2)

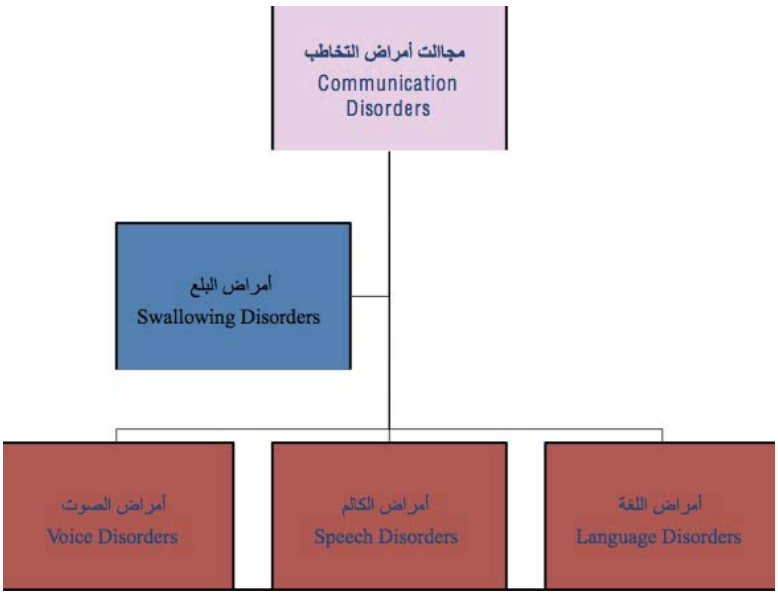
Resources: Dr.Tamer Mesallam's lecure, student notes.

Objectives:

Communication and Swallowing I:
- Physiology of swallowing.
- Swallowing disorders.
- GERD.

Communication and Swallowing II:
- Voice disorder.
- Language disorder.
- Speech disorder.

Done by: Bodoor Tayeb



Definitions:

Language:
A symbolic arbitrary system relating sounds to meaning.

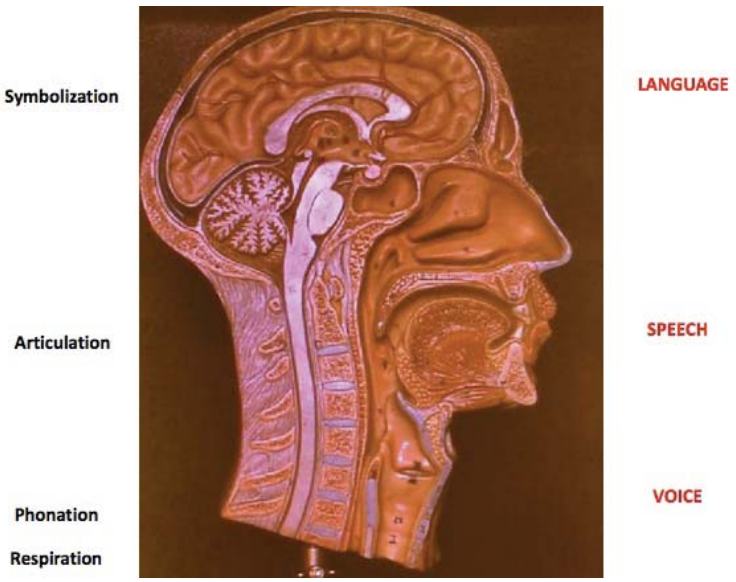
Speech:
A neuro-muscular process whereby language is uttered. It includes the coordination of respiration, phonation, articulation, resonation and prosody.

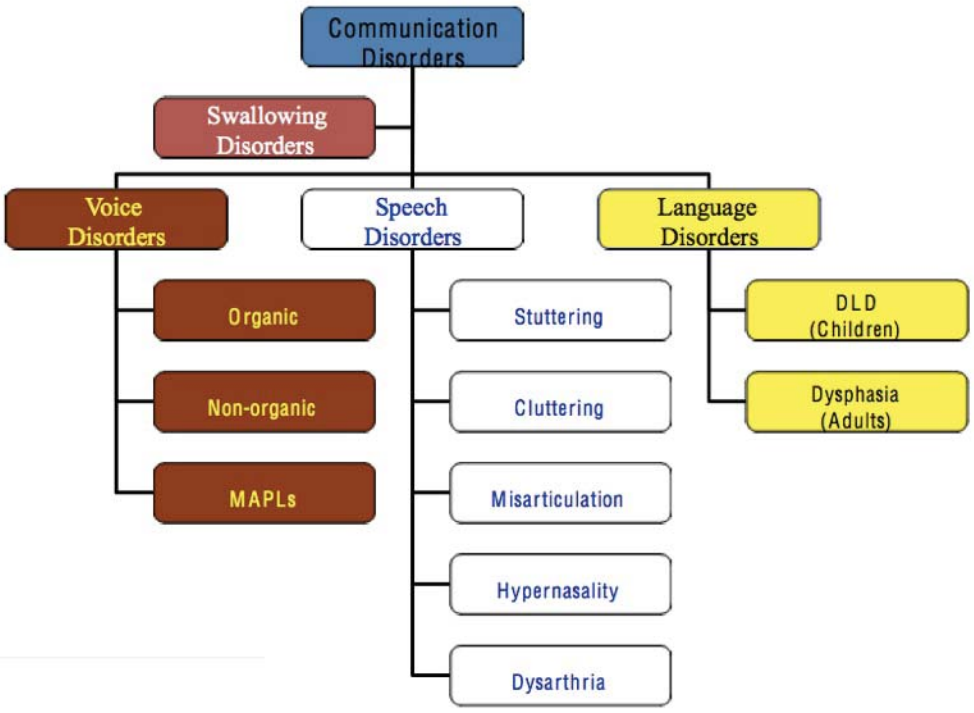
Voice:
The result of vibration of the true vocal folds using the expired air.

Swallowing:
The process of successful passage of food and drinks from the mouth through pharynx and esophagus into the stomach.

☆ In order to speak we need:

1- respiration (exhalation) →a-voice
2- phonation (vocal cord) → ″ ″ ″ ″
3- articulation → b-speech
4- symbolization → c- language





Language Disorders

[1] Delayed Language Development (DLD)
[2] Dysphasia

[1] Delayed Language Development (DLD)

Definition:
Delay or failure to acquire language matched with age.

Central language control:

-The left hemisphere is the processor of language functions in almost all people regardless handedness. It is the dominant hemisphere.
- Language areas are distributed along the rolandic fissure
-Anterior language area mainly in the temporal region concerned with expressive aspect.
-Posterior language area mainly in the parietal region concerned with receptive aspect.

Structural domains of language;

-Semantics; meaning.
-Phonology; articulation
-Syntax; grammar

Stages of normal language development

•2-4 months;	Babbling
•6 months;	Vocal play
•9 mo-1 year;	1st word
•1-1/2 years;	20 words
•2 years;	200 words, 2 word sentence
•3 years;	2000 words, 3 word sentence
•4 years;	4 word sentence
•5-7 years;	Full maturation of all language modalities.

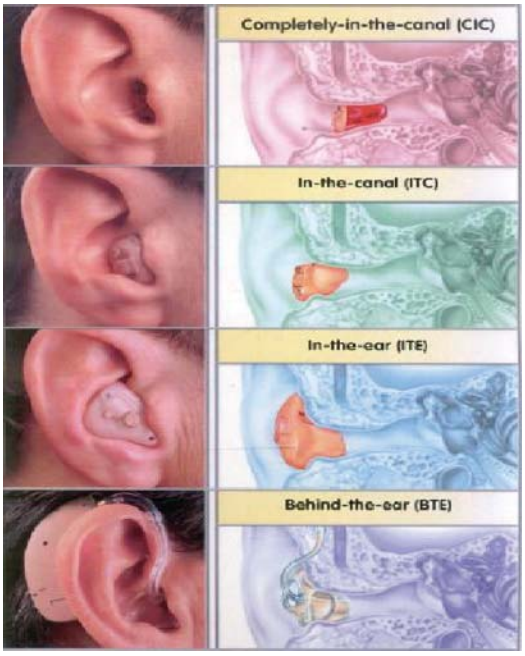
Pre-requisites of normal language development

-Intact brain functions (conceptual, motoric and cognitive abilities).
-Intact sensory channels; (Auditory, Visual, Tactile, Kinesthetic).
-Intact psyche.
-Stimulating environment.

Etiology of delayed language development:

A- Brain damage:

- Diffuse subcortical lesion (M.R.).
- Localized brain damage with motor handicap (BDMH).
- Minimal brain damage (ADHD).



B- Sensory deprivation:

- Hearing impairment
(Conductive, Sensorineural, Mixed or Central auditory processing disorder)
- Visual impairment

C- Psychiatric illness:

- Autism.
- Autism Spectrum Disorder (ASD).

D- Environmental deprivation

E- Idiopathic (Specific Language Impairment).

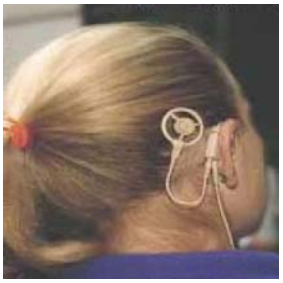
Assessment of language development:

I. History taking.
II. Physical examination.
III. Investigations:

–Psychometry (IQ).
–Brian Imaging
–Ophthalmological consultation
–Audiometry.
–EEG

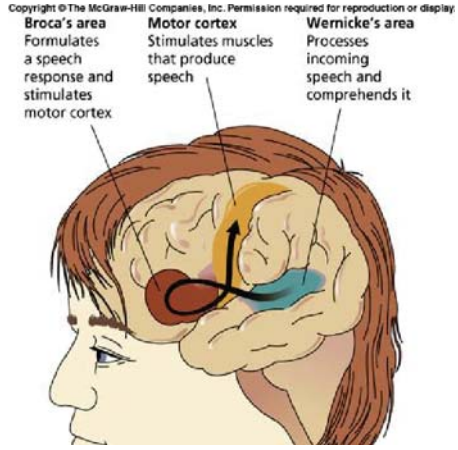
Management of DLD:

- Early detection.
- Providing the suitable aid:
a.Hearing (HA or CI).
b.Physiotherapy Family counseling.
c.Visual Aid.
- Direct language therapy (Individual- group).
- Medications (Autism and ADHD).



[2] Dysphasia:

Definition:
Language deterioration after its full development due to brain insult: infarction, hemorrhage, atrophy, etc



ex:
if a 12 y/o boy lost his language after it already developed , we call it dysphasia because language is fully developed

Etiology:
CVA, Neoplastic, Traumatic, Inflammatory, Degenerative, Metabolic, Poisoning

Types of dysphasia:
1. Expressive. 2. Receptive. 3. Mixed predominantly expressive.
4. Mixed predominantly receptive. 5. Global.

Assessment of Dysphasia:
I. History taking.
II. Physical examination: ... , neurological exam.
III. Investigations:
- CT / MRI brain.
- Psychometry (IQ).
- Dysphasia test.
- Audiometry.

Management of Dysphasia:
-Management of the cause.
-Physical rehabilitation (Physiotherapy).
-Family counseling.
-Language therapy.
-Alternative and augmentative communication.

Speech Disorders:

1. Dyslalia (Misarticulation):

Definition:
Faulty articulation of one or more of speech sounds not appropriate for age.

Types of dyslalia:

A) Sigmatism (/s/ defect):-
- Interdental sigmatism.
- Lateral sigmatism.
- Pharyngeal sigmatism.

B) Back-to-front dyslalia:-
/k/ /t/
/g/ /d/

C) Rotacism (/r/ defect).

D) Voiced-to-nonvoiced dyslalia:-
/g/ /k/
/d/ /t/
/z/ /s/ etc…

Assessment of dyslalia:

I. History taking.
II. Physical examination: ... , tongue, ...
III. Investigations:
- Audio recording. - Articulation test.
- Psychometry (IQ). - Audiometry.

Management of dyslalia:

a. Treatment of the cause:
-Tongue tie.
-Dental anomalies.
-Hearing aids
b. Speech therapy.

2. Stuttering:

Definition:

The intraphonemic disruptions resulting in sound and syllable repetitions, sound prolongations, and blocks.

Normal dysfluency:

- Less than 6 years.
- Only repetitions.
- No associated muscular activity.
- Not aware.

Incidence of stuttering: 1%.

Onset:

- Earliest = 18 months. - Latest = 13 years.

Epidemiology:

- more in families with history of stuttering.
- can occur in mentally retarded.
- very rare in the hearing impaired.

Gender ratio: 4 : 1 (male : female)

Theories of Stuttering:

The exact cause is unknown.
- Organic theory.
- Neurosis theory.
- Learning theory.

Assessment of stuttering:

I. History taking.
II. Physical examination: APA, VPA, ...
III. Investigations:
- Audio and video recording.(explained next)
- Stuttering severity (eg SSI).
- Articulation test.
- Psychometry (IQ).

Auditory Perceptual Analysis (APA):

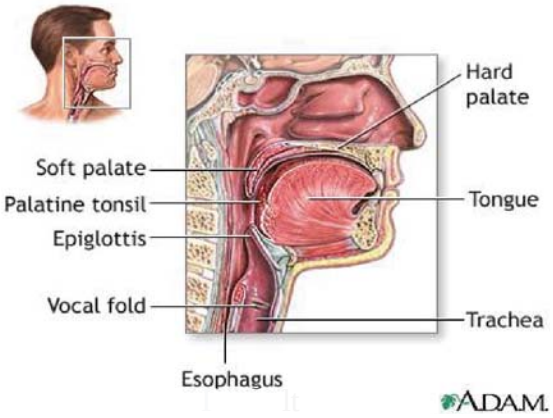
A. Core behaviors:
- Intraphonemic disruption. - Repetitions. - Prolongations. - Blocks.

B. Secondary reactions:
- Muscular activity and struggle. - Interjection. - Word substitutions and circumlocution.

C. Concomitant reactions:
- Fear.
- Breathing (antagonism, interruption, prolongation, cessation, ...).
- Eye contact.
- Skin pallor/flushing.

Management of stuttering:

I.Family and patient counseling.
II.Speech therapy:
a. Indirect therapy: if not aware.
b. Direct therapy: if aware.



3. Hypernasality:

Definition:

Faulty contamination of the speech signal by the additior
velopharyngeal insufficiency (VPI).

Velum: At rest and during speech

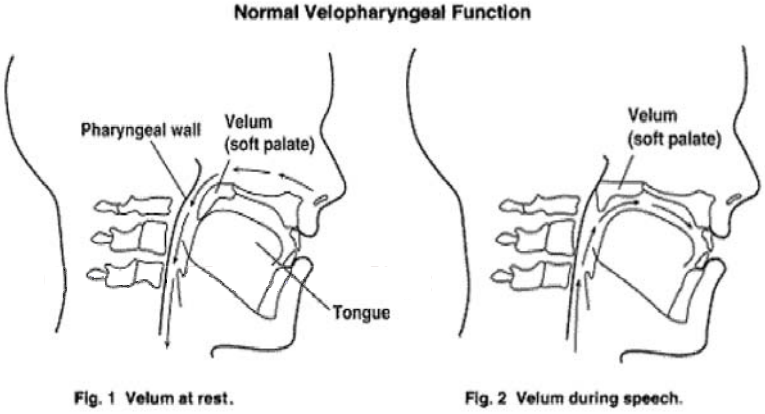


Fig. 1 Velum at rest. Fig. 2 Velum during speech.

Causes of hypernasality:

I. Organic:

1.Structural: (VP Insufficiency)		2. Neurogenic: (VP Incompetence)
a) Congenital:	b) Acquired:	- Palatal upper motor neuron lesion. - Palatal lower motor neuron lesion.
- Overt cleft palate. - Submucous cleft palate. - Non-cleft causes: a.Congenital short palate. b.Congenital deep pharynx.	- Adenotonsillectomy - Palatal trauma. - Tumors of the palate & pharynx.	

II. Non-organic (Functional) VP Mis-learning:

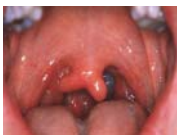
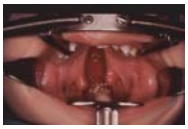
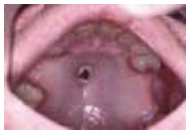
- Faulty speech habits. - Mental retardation. - Neurosis or hysteria.
- Hearing impairment. - Post-tonsillectomy pain.

Effects of VPD:

- Feeding problems: nasal regurgitation.
- Psychosocial problems.
- Communicative problems:

(Speech: hypernasality. / Language: DLD. / Voice: hyper or hypofunction)

Assessment of hypernasality (VPD)

1-Parent interview

2-Perceptual.

a. Simple tests:
   - Gutzman's (a/i) test
   - Czermak's (cold mirror) test.

b. Resonance.

c. Articulation.

d. Nasal air emission.

e. Voice

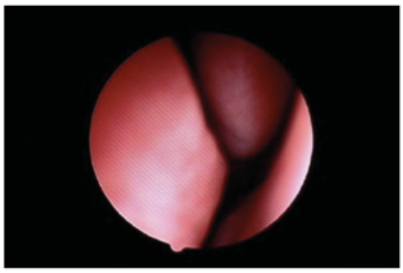
3- Intra-oral evaluation

4- Instrumental:
   - Nasopharyngoscopy
   - Nasometry

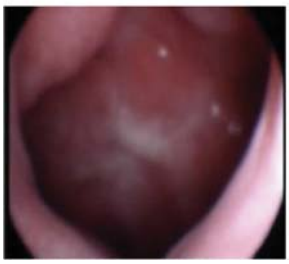
Gutzman's (a/i) test: tell the patient to say a/i while the nose is open then again while the nose is closed (normally there is no difference)

Submucus Cleft

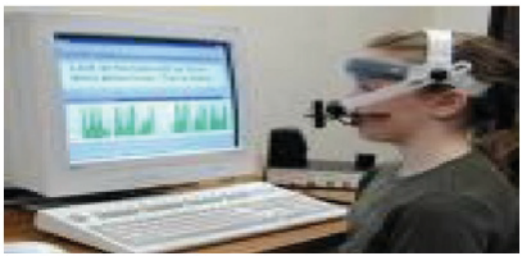
Normal closure



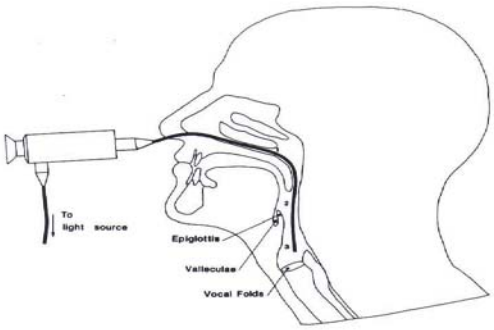
Severe VPD



Nasometry



Flexible nasopharyngoscopy



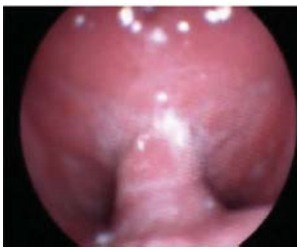
Management of VPD:

-Multidisciplinary team.
-Family counseling .
-Management of feeding problem.
-Management of otological and audiological problems.
-Surgical intervention.
-Orthodontic intervention.
-Phoniatric intervention (language, speech, voice).

Treatment Decision:

-Velopharyngeal insufficiency
surgery (speech therapy post-op)
-Velopharyngeal incompetence
surgery (speech therapy post-op) - prosthetic devices - speech therapy.
-Velopharyngeal mislearning
speech therapy.

Pharyngeal flap

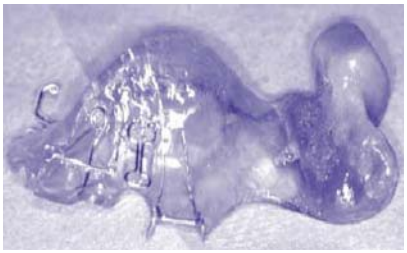


Surgery:

-Pharyngeal flap.
-Sphincter-platoplasty
-Post-pharyngeal wall augmentation.

Prosthetic Devices:

-Palatal lift: to raise the velum when there is poor velar movement (i.e. dysartheria)
-Platal obturator: to occlude an open cleft or fistula
-speech bulb: to occlude nasopharynx



4. Dysarthria:

Definition:

Any combination of disorders of respiration, phonation, articulation, resonance, and prosody, that may result from a neuromuscular disorder.

Types of dysarthria:

1. Flaccid dysarthria:	2. Spastic dysarthria:	3. Ataxic dysarthria:	4. Dyskinetic dysarthria:	5. Mixed dysarthria:
- Lesion: lower motor neuron level. - Communication: *breathy phonation. *hypernasality.	- Lesion: upper motor neuron level. - Communication: *strained strangled phonation. *labored breathing.	- Lesion: cerebellum level. - Communication: *increased equal stresses. *irregular articulatory breakdown.	- Lesion: basal ganglia level. a.Hypokinetic type (Parkinsonism): * breathy phonation. * rapid rate. * short rushes of speech with final decay. b.Hyperkinetic type: i. Quick hyperkinetic (Chorea):variable rate and loudness. ii. Slow hyperkinetic (Athetosis): slow rate.	- may the most common. - Examples: *Motor neuron disease ➪Flaccid + Spastic. *Multiple sclerosis➪ Ataxic + Spastic. *Wilson's disease➪ Ataxic + Spastic + Hypokinetic.

Assessment of dysarthria:

I.History taking.

II.Physical examination: ... , mouth, palate, ... , neurological exam, ..

III. Investigations:

- Audio recording.
- CT/MRI brain .
- Nasometry.
- Dysphasia test.
- Fiberoptic nasopharyngolaryngoscopy.
- Psychometry (IQ).
- MDVP.
- Articulation test.
- Audiometry.
- Aerodynamics(AerophoneII).

Management of dysarthria:

Individualized:

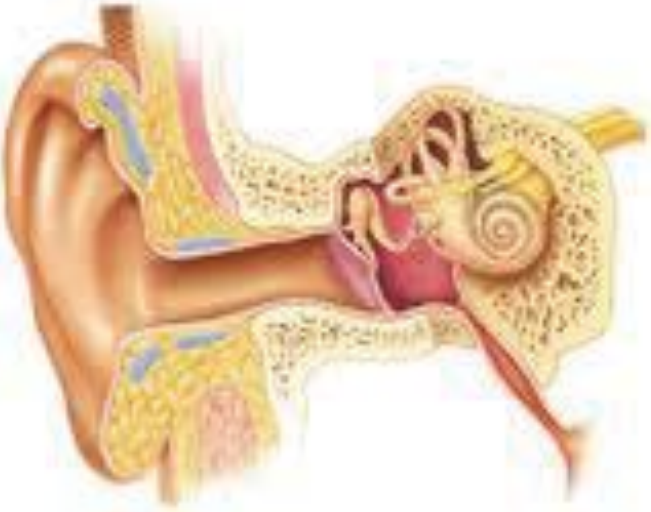
•Management of the cause.

•Patient counseling.

•Communicative therapy: * Articulation. * Phonation. * Resonance. * Respiration. * Prosody.

•Alternative and augmentative communication.

Diseases of the Ear, Nose and Throat



22st Lecture:

Communication and Swallowing Disorders (part 2)

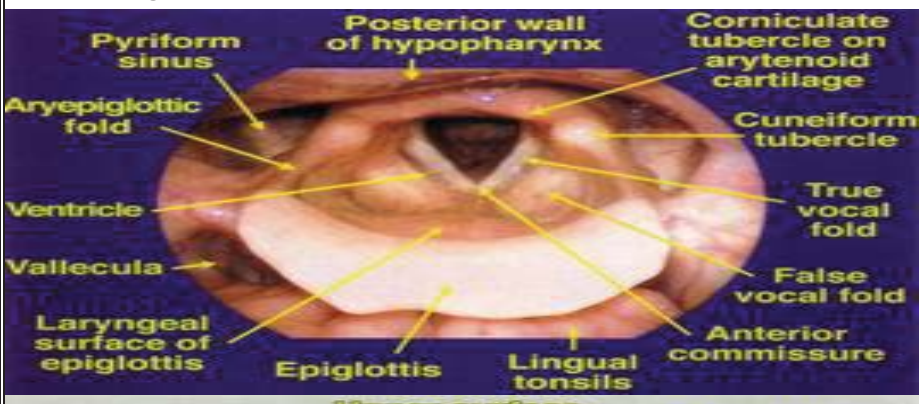
Done by: ayshah al-mahboob

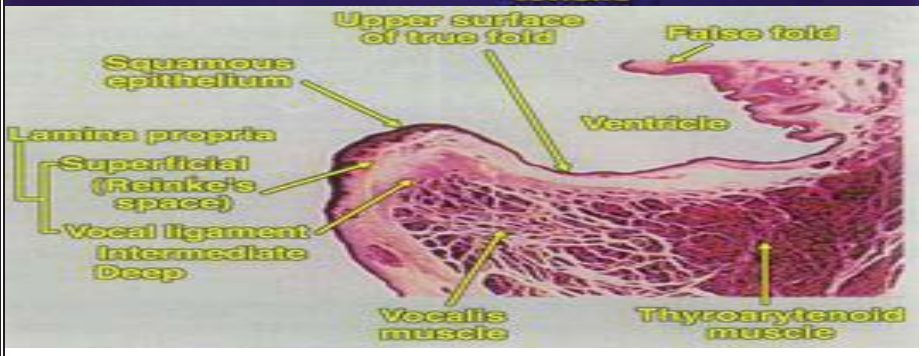
The slides were provided by doctor (**Tamer Mesallam**)
my source (the lecture slides and records only - NOTE: for better understanding , study larynx lectures first)
Important Notes in red
Copied slides in black
Your notes in blue
Doctor notes in green
Highlight possible MCQs mentioned or pointed by the doctor

2 Communication and Swallowing Disorders

In part 2 we will study :

-voice disorders
-swallowing disorders





The doctor read every single word in this picture
–note:
to decide the side of any part of the larynx lesion (eg: vocal cord lesions) whether it's right or left side lesion
(**consider the land mark anteriorly is** : anterior commissure and epiglottis and **your posterior land mark is** : arytenoid cartilage and hypopharynx)
because in the EXAM some picture will be inverted , so it's very important to know the correct site of the lesion.

Cross section of the vocal cords which contains :
Mucosa :
1-Squamous epithelium
2-lamina propria which contains:
- the superficial layer
- the intermediate and deep layers which form the vocal ligament

Prerequisites of "normal" voice production:

The doctor did not read the prerequisites

1. Normal range of movement of vocal folds.
2. Normal mobility of mucosa on deep layers.
3. Optimal coaptation of vocal folds' edges.
4. Optimal motor force.
5. Optimal pulmonary support.
6. Optimal timing between vocal fold closure and pulmonary exhalation.
7. Optimal tuning of vocal fold musculature (int. & ext.).

Usually the presenting symptoms in voice disorders are:

- **Dysphonia:** Any change of the patient's voice from his habitual one.
- **Aphonia:** Loss of the patient's voice (functional or organic).
- **Phonasthenia**: a subjective complaint of dryness, tightness, globus feeling ,foreign body sensation and voice fatigue, while the patient's voice and larynx is normal.
- **Dysodia:** Change of the singing voice while the speaking voice is normal. This complain we see it usually in professional voice users like : the singers , quran readers, teachers .
  (يعني يشتكي من تغير صوته بالغناء فقط لكن صوته المعتاد لم يتغير)

Definition of dysphonia and Hoarseness:

We like to say dysphonia in any voice changes rather than hoarseness because **dysphonia means :** Difficulty in phonation and the Changing of voice from his /her habitual and it includes any kind of voice character changes however **Hoarseness means only** roughness & harshness of voice

Etiological classification of dysphonia:

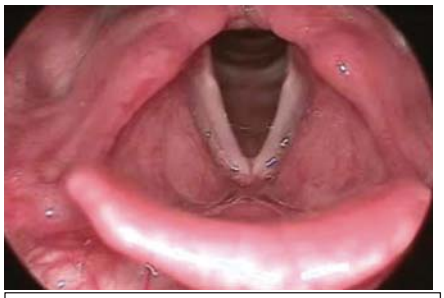
I. Organic Causes
II. Non-Organic Causes: Habitual , Psychogenic
III. Benign vocal fold lesions =Minimal Associated Pathological Lesions (MAPLs) includes cyst , nodules and follicles …etc which we will mention later
IV. Accompaniment of Neuro-psychiatric Ailments

III.Voice disorders:

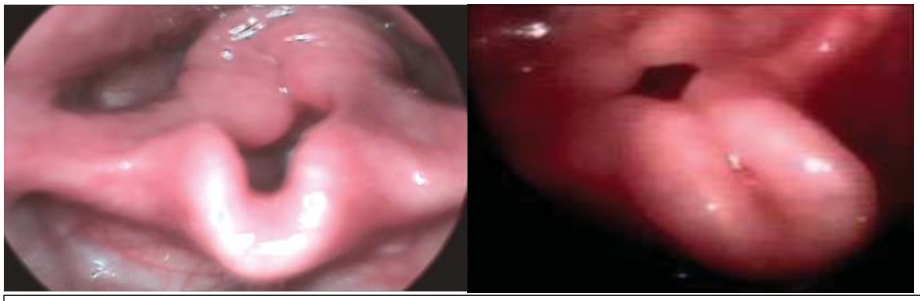
A) Organic voice disorders:

. Congenital.
. Inflammatory.
. Traumatic.
. Neurological.
. Neoplastic.
. Hormonal.
. Status post-laryngectomy.

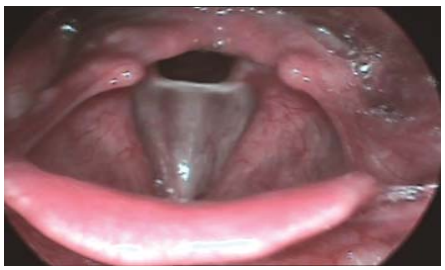
All of the following pictures regarding organic voice disorders are possible exam questions IMP :



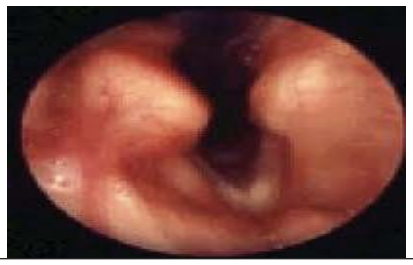
Normal larynx



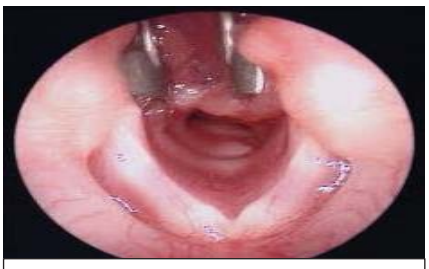
Description: Omega shape epiglottis whenever you see **it's most likely** Laryngomalacia
It's common in children 1-2 years old
The common presentation: it is not a dysphonia , it's breathing problem , especially when he is laughing or playing , he can get a noisy breathing (stridor) due to collapse epiglottis over the arytenoid cartilage
Treatment:
Depends on the degree
Mild case: observation and tell the family that this condition it's self-limiting
But in severe cases :where it's interfere with respiration we do surgery



Description: Congenital vocal folds web
The history is about 18 years old patient , he diagnosed late because he did not complain from breathing problem
Common presentation : The symptoms depends on the stages of the web , eg :when the web extend posteriorly , it will obstruct the air way and the patient will suffer from breathing related problems and he will be presented early .
Treatment:
Surgical excision but I have to be aware from post surgical atresia (eg: After the incision in approximately two weeks , there will be adhesion or synchea between the vocal cords when they are closed to each other , so I have to put some thing between them to prevent the adhesion. (I could not hear what is the name of the thing that they put it between the vocal cords ?)

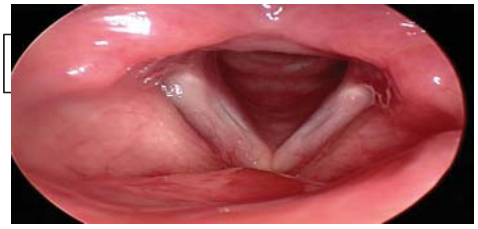


This is Severe type when the cleft extend deep to the trachea

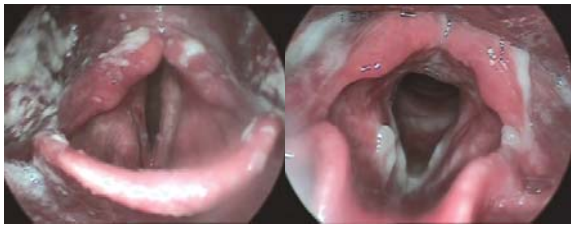


Type 1 in the arytenoid clefting

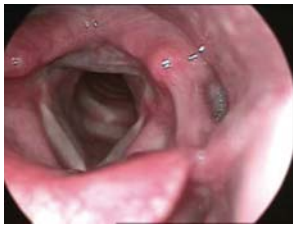
Description :laryngeal cleft or laryngotracheoesophageal cleft is a rare congenital abnormality in the posterior laryngo-tracheal wall . It means there is a gap between the oesophagus and trachea, which allows food or fluid to pass into the airway.
Types: **Type I** extends no further down than the vocal cords, **type II** extends below the vocal cords and into the cricoid cartilage, **type III** extends into the cervical section of the trachea and **type IV** extends the furthest—into the thoracic section of the trachea
Management :Surgery



Description: Sulcus Vocalis (Congenital) , Very difficult condition to diagnose , it's rare but common here in KSA ,
Presentation: severe dysphonia
Treatment: Surgery (vocal cord augmentation to decrease the gabs)

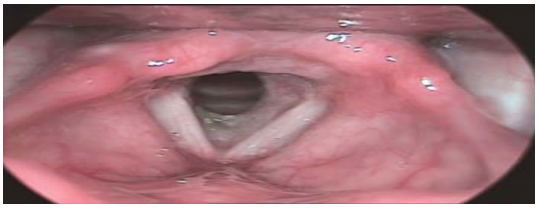


Description: Fungal infection (Inflammatory)
Presentation: Imp finding is a history of immune compromised patient(diabetic patient or patient in immune suppressive drugs)
Treatment: Antifungal drugs



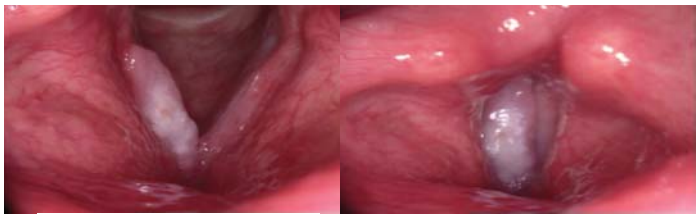
Laryngopharyngeal Reflux (Inflammatory) : Notice that You will see congestion

This early stage of this condition (crustation)

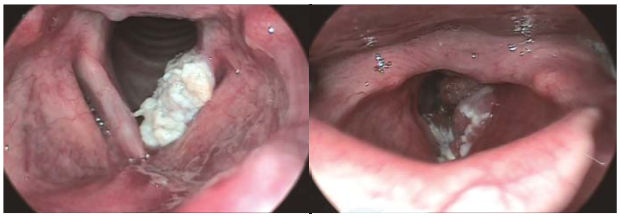


This is late stage when we see granulation tissue and subglottic stenosis

Description: The name of this chronic specific infection is Laryngoscleroma (Inflammatory)
Rare here but common in Egypt
Treatment :
In early stage: Selective antibiotic :ciprofloxacin , the response is good
If left untreated : the patient can develop granulation tissue and end in subglottic stenosis

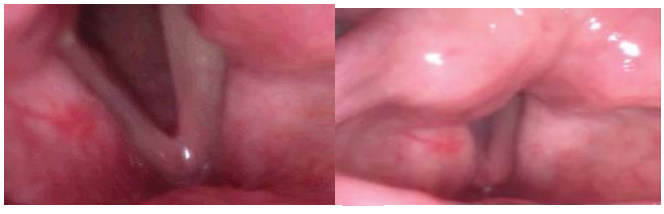


Respiration Phonation

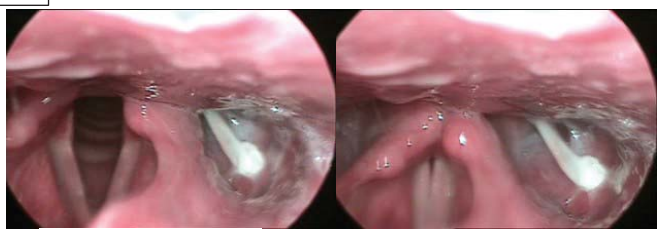


Description: Laryngeal carcinoma (Neoplastic)
-right vocal cords shows squamous cell carcinoma
Common Presentation : dysphonia
Management: surgical
Notice The land mark to decide the direction of the lesion

Advanced malignancy , Cancer (Neoplastic)



Respiration Phonation



Respiration Phonation

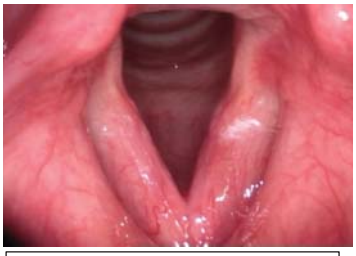
Description: Unilateral Left vocal fold paralysis (Neurological)
Notice during the abduction the left vocal cords is slightly abducted than the other one
Common Presentation : Aspiration , dysphonia
Treatment is : Augmentation of the vocal cord by medialization laryngoplasty to reduce the gab
The dr said that I guess you will not know it because Moving video is the best way to judge the paralysis

- The History will help you to know the finding .
- In this case there is history of Swelling of the left side of the nick after 3 days from aspiration and chocking during eating.
- **Finding:** There is Bone of chicken in pyriform sinus (notice that this is the first area that you have to look for in case of suspicion of foreign body) !!

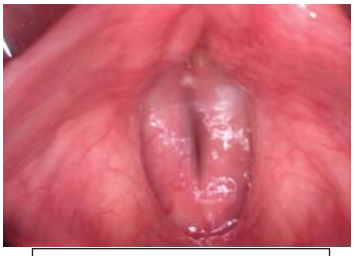
B) Non-organic voice disorders:

i. Habitual:

1. Hyperfunctional childhood dysphonia (a child who always talk and cry).
2. Incomplete mutation (also called tebophonea? (I could not hear it)? . normally in male puberty there will be changes from high pitch voice to low pitch , in patient who has incomplete mutation this changes will not occur).
3. Phonasthenia (Voice fatigue).
4. Hyperfunctional dysphonia (mis-using of the voice).
5. Hypofunctional dysphonia (severe stage of Hyperfunctional dysphonia may lead to this) .
6. Ventricular dysphonia (this is when vocal cords and false ventricular bands shared in phonation)

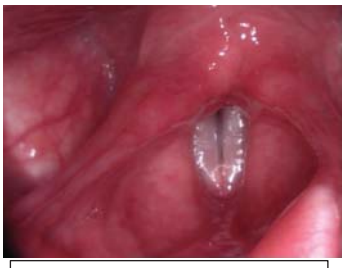


Respiration

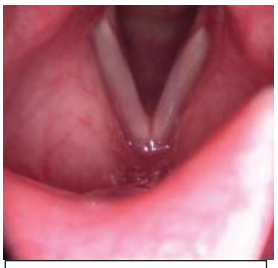


Phonation

Hyperfunctional dysphonia
Larynx shows No organic lesion but there is **incomplete adduction in phonation** called (phonatory gap) , this is a sign of exhausted muscle



Phonation



Respiration

Phonasthenia
Also there is No organic lesion , just there is there is (phonatory gab)

B) Non-organic voice disorders (cont.):

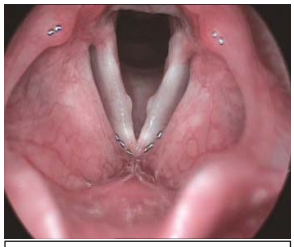
ii. Psychogenic: it's more common among female , it is also called physco-somatic conversion (the patient converts his psychogenic problem to somatic (voice) problem), so there is no real voice disorder , the patient is just acting .

1- Psychogenic dysphonia. 2- Psychogenic aphonia

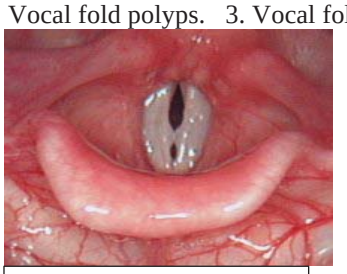
C) Benign vocal folds lesions:

They included in one category because they shares same etiology which is mis-use of voice imp OSCE :

1. Vocal fold nodules. 2. Vocal fold polyps. 3. Vocal fold cysts. 4. Reinke's edema. 5. Contact granuloma.

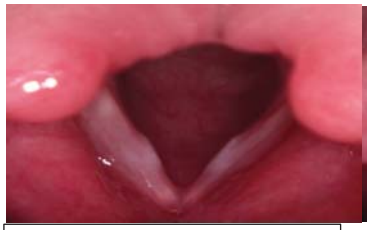


Respiration

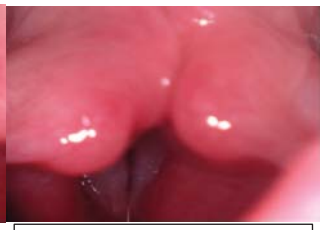


Phonation

Description: Vocal Fold Nodules: Adult Type
Presentation : Dysphonia
Management: Voice rest and voice thereby , do not say surgery
Note: vocal cord nodule always Bilateral symmetrical nodules In (adult and juvenile type) but the nodules look more softer in juvenile type.

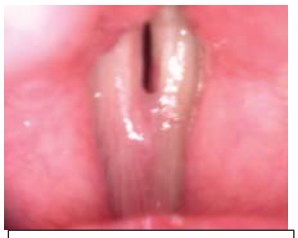
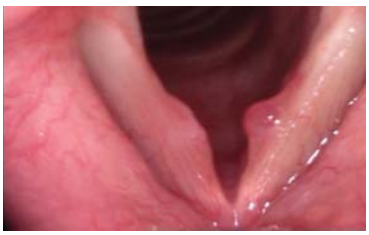


Respiration



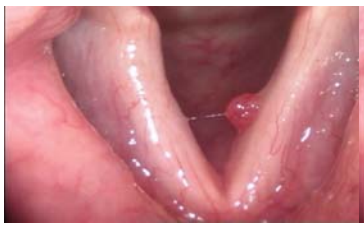
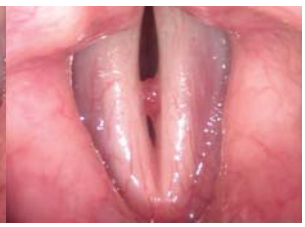
Phonation

Description : Vocal Fold Nodules: Juvenile Type
Notice that the juvenile type nodules are softer than the adult type
Common presentation : It comes with children who has hyper functional disorder

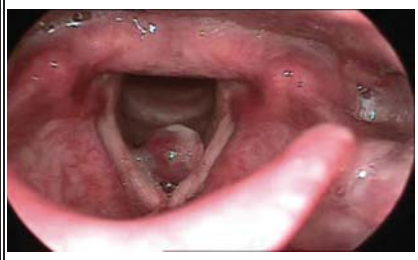
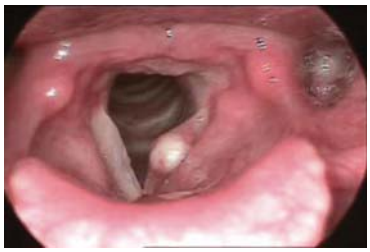
Phonation | Respiration

Description: Left Vocal Fold Polyp with a Reaction(notice the reaction in the other side)
Treatment : Excision just for the polyp not the reaction

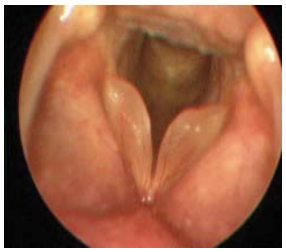
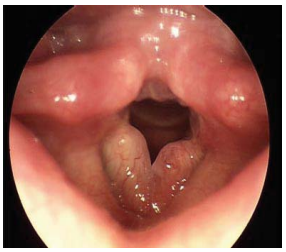
Respiration | Phonation

Discreption: Left Vocal Fold Polyp
-The same finding as previous one just that there is sessile hemorrhagic polyp without reaction

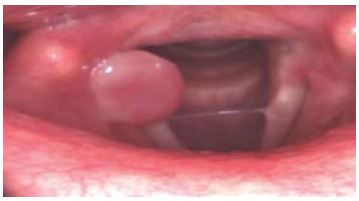
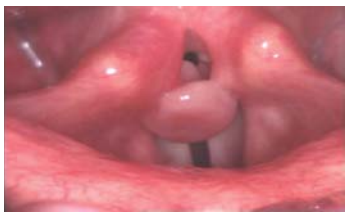



Description: Right Vocal Fold Polyp
-it's Pedunculated polyp , this is easier in excision than the previous one
-always the polyp has well defined edge
- so it's Not a cyst , the cyst has ill defined edge

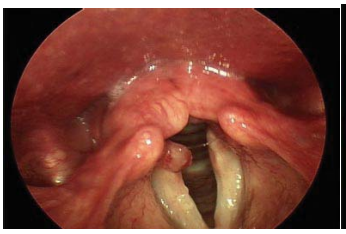
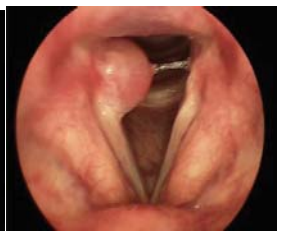
Description: Left Vocal Fold cyst
Treatment:Surgical excision

Description: Bilateral Reinke's edema
Common presentation :common with female smoker Because this condition cause deepening of the voice so the male will not complain because they do not notice this change .

Description: Right-sided Intubation Granuloma
Common presentation : History of prolonged intubation after for example : cardiac surgery and the Lesion must be in posterior part of the vocal cord
Treatment : conservative treatment and you can discharge the patient

Description: Right-sided Contact Granuloma
Etology : reflux
Treatment : anti reflex thereby by conservative thereby for long time
Note that the granuloma is posterior lesion in opposite to the polyp which is Anterior lesion

Assessment of dysphonia:

I. History taking.
II. Physical examination: APA , … , neck , …
III. Investigations:

- Audio recording.
- Digital High speed imagin
- GERD (LPR) work-up.
- Digital laryngostroboscopy
- Acoustic analysis (MDVP)
- CT neck.
- Digital laryngokymography.
- Aerodynamic analysis (Aerophone II).

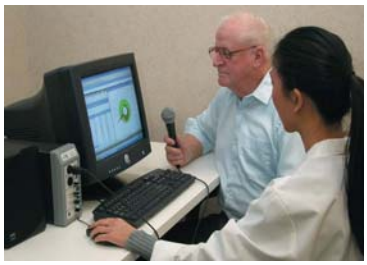
Dr did not read the investigations just said that they have many of them to assess the voice




Video sterboscopy with Rigid or fixable Laryngoscopy

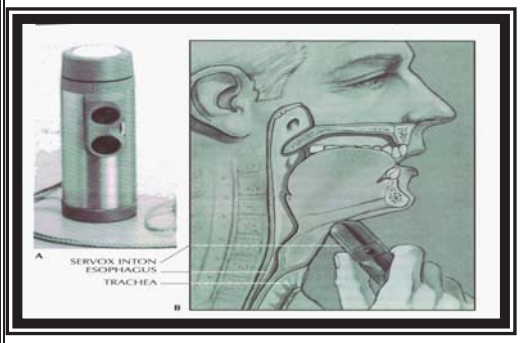
High speed laryngeal imaging

Pharyngeal pH Monitoring For GERD , A Device fixed in the oropharynx and sense the PH of reflex

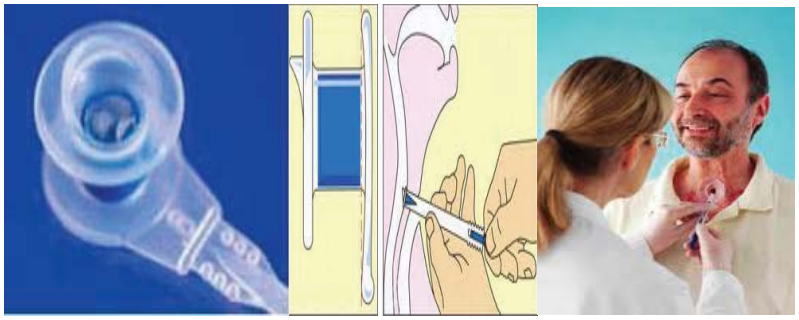


Computerized speech lab. (CSL) For Voice analysis

Phonatory Aerodynamic System (PAS) Assess the phonatory air flow During (phonation and vibration)



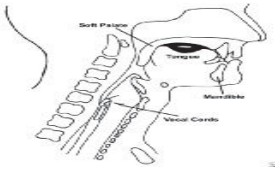
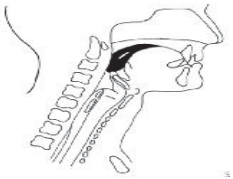
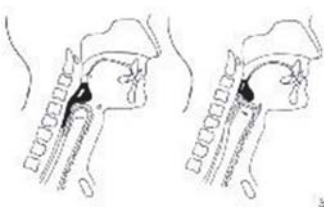
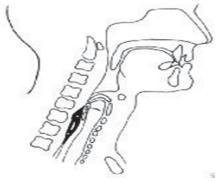
Artificial larynx in patient who got total laryngioctomy , can induce mechanical voice But most of patient do not like it



Tracheo-esophageal puncture , it is The most common procedure done for the patient who undergoes laryngectomy , an instrument is introduced in trachea and through the air entry into trachea , the phonation will occur

Swallowing Disorders

Phases of normal swallowing: We are interested mainly by oral and pharyngeal swallowing not the esophageal one

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1. Oral preparatory phase
2. Oral propulsive phase
3. Pharyngeal phase
4. Esophageal phase

- Definition of dysphagia: Difficulty in moving food from the mouth to the stomach.
- Odynophagia : painful swallowing due to a disorder of the esophagus

Consequences of dysphagia:

- Dehydration. - Weight loss.
- Aspiration pneumonia. - Airway obstruction. - Loss of joy of eating

Causes of dysphagia:

Oropharyngeal

- **Structural:**
  Head & Neck Surgery
- **Neuromuscular**:
  CVA

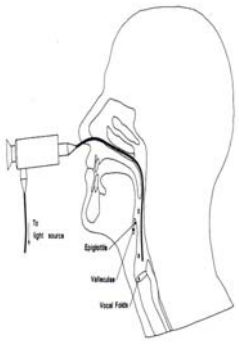
Esophageal:

- **Mechanical [Solids]**
  Tumors
- **Neuromuscular**
  (Esophageal Dismotility) [Solids & Liquids]
  Achalasia

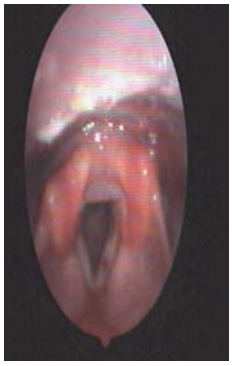
Assessment of dysphagia:

I. History taking
II. II. Physical examination:
III. - General examination.
- Language and Speech assessment.
- Vocal tract examination.
- Neck examination.
- Trail feeding (Bed-side assessment). Most imp , seeing the patient behavior during eating.

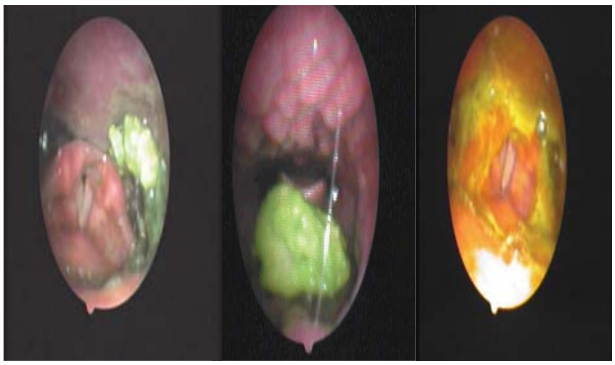
III. Investigations:
- FEES.
- VFES (MBS).
- GERD (LPR) work-up.



FEES



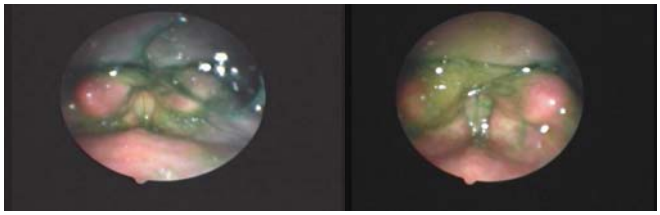
Normal FEES



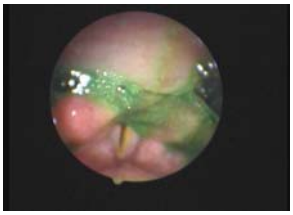
Residue in left pyriform sinus

Residue

Residue



Penetration

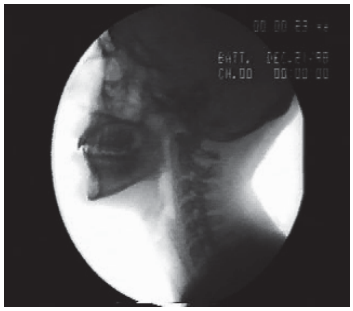


Aspiration
When I can not see the subglottic space clearly ,Means this materials enter air way (below the vocal cord) and coughed out

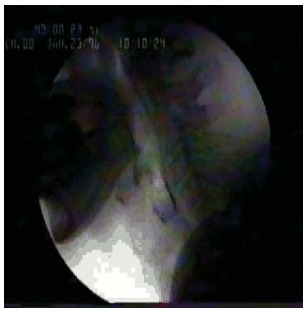
In FEES: We look for residue after swallowing
If the Bolus is found
Above the vocal cords it is penetration
Below the vocal cord it is aspiration

I. FEES protocol of evaluation (Langmore, 2003):

- Anatomic and physiologic assessment.
- Assessment of food and liquid swallowing.
- Assessment of theraputic interventions.



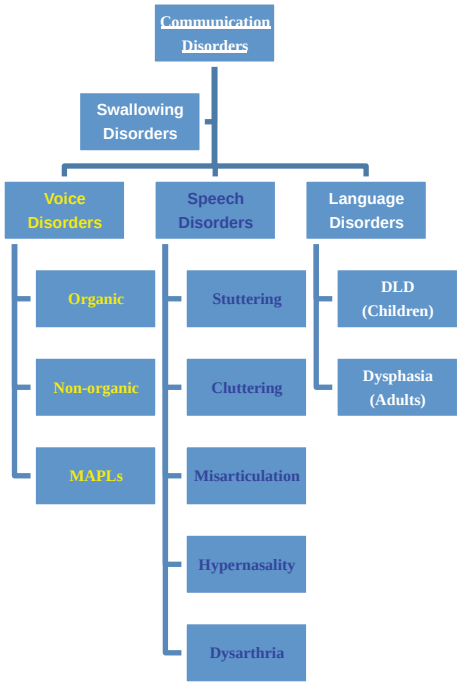
Normal (MBS)modified Barium swallowing

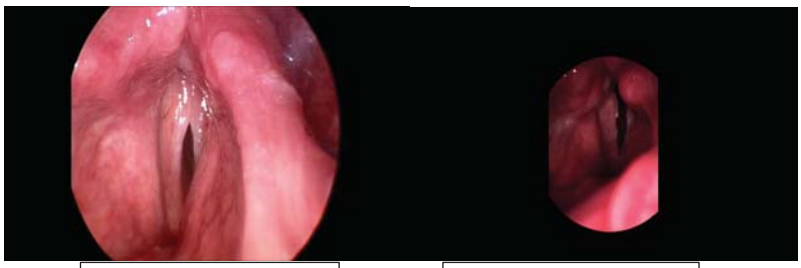


Aspiration (MBS) Bolus is aspirated and enter trachea

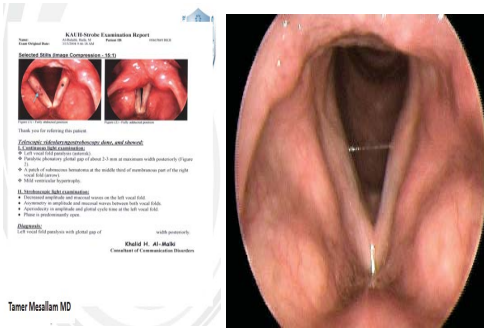
Management of dysphagia:

- **Swallowing therapy**:
  - Diet modification. - Postural techniques. - Swallowing maneuvers.
  - Sensory enhancement techniques. - Motor exercises.
- **Surgical treatment, eg** medialization laryngoplasty in vocal cord paralysis.
- **Medical (Drug) treatment,** eg anti-parkinsonism drugs.
- **Intraoral prosthesis.**
- **Alternative routes of feeding**, eg NG tube feeding. If the patient failed to compline the previous treatment ways





Strobe :



High speed laryngeal

The doctor show us a video to compare the difference between this two methods , I can not write all what he said but the conclusion is :
Strobe :Giving flashing of light in slow motion
High speed laryngeal imaging :Shows the vocal cords in very very slow motion , more detailed

Dr did not mention any thing about these two picture