

Lecture Note of otolaryngology For medical Students

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Preface

This book provide the ENT trainee with the information they will need to progress to ENT practice in the field . It give the reader a knowledge base in each of it's topics . This book covers most of the common and important subjects ENT and will give the reader a stepwise account of most of the operations they will need to learn in this field. I think this book will become approachable manner suitable for medical students . We advise readers to look for more comprehensive textbooks of otolaryngology .

Ahmed M. AL Abbasi

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Anatomy of the Ear

The adult human ear divided to 3 subdivisions:

- External ear
- Middle ear
- Inner ear.



- **External ear:**

The external (outer) ear help to collect and introduce sound waves from the environment to the tympanic membrane. It consists from the following:

- Pinna (Auricle): which is composed of cartilage framework that firmly attached to the perichondrium and skin.
- External auditory canal (EAC): which is a 2.4 cm in length tube ends at the tympanic membrane. Lateral 2/3 is cartilage and medial 1/3 is bony.

The cartilaginous part contain hair, ceromucinous gland. The canal is not straight, but by gently moving the auricle upwards and backwards, a straight canal results in adults. In the neonate, no bony external canal presents because the tympanic bone not developed yet, so that the auricle drawn gently downwards and backwards for the best view of the tympanic membrane.

- Blood supply: branches from external carotid artery.
- Nerve supply: cervical branches(C2, C3) + sensory branches from cranial nerves: V, VII, IX, X
- Venous drainage: to external jugular vein and pterygoid plexuses
- Lymphatic drainage: to preauricular, postauricular and upper deep cervical lymph nodes.
- **Middle ear:**

Function to transmit sounds energy from the EAC to the cochlea. The middle ear cleft consists from the following structures:



- Tympanic membrane

- Middle ear cavity
- Eustachian tube
- Mastoid air cells

Tympanic membrane: Consist from three layers:

- Outer: squamous epithelium (skin) without hairs or glands
- Middle: fibrous layer
- Inner: mucosal layer
- Color: pearly grey
- Shape: slightly oval divided into 4 quadrants:
- Light reflex: located in the antroinferior quadrant.

Middle ear cavity:

- Biconcave box like space contain the following structures:
 - Ossicles: malleus, incus, stapes.
 - Nerves: facial nerve and chorda tympani.
 - Muscles: stapedius and tensor tympani.
- Blood supply: branches from both external and internal carotid arteries
- Venous drainage: to pterygoid plexuses
- Lymphatic drainage: to retropharyngeal and upper deep cervical lymph nodes
- Sensory innervation: branches from cranial nerves: V, IX, X
- It has six walls:
 - Lateral: made by tympanic membrane

- Medial: promontory of the cochlea with 2 opening: oval and round windows
- Anterior: contain Eustachian tube orifice allow air to pass freely between the nasopharynx and middle-ear cavities
- Posterior: contain antrum to mastoid air cells
- Superior: bonny roof separates middle ear cavity from the meninges and brain
- Inferior: bonny plate cover the jugular vein bulb

Eustachian tube:

3.6 cm tube connects the middle ear cavity with the nasopharynx lined by stratified squamous epithelium (respiratory epithelium). It run downward and medially from the middle ear to open at the lateral wall of the nasopharynx. Lateral 1/3 is bonny and medial 2/3 is cartilaginous in structure.

- Function: aeration of the middle ear
- Blood supply: ascending pharyngeal and middle meningeal arteries
- Venous drainage: to pharyngeal plexuses
- Lymphatic drainage: to retropharyngeal lymph node

The mastoid air cells:

It is an air-filled, honey comp like space within the petrous part of temporal bone.

The following bound the mastoid air space:

- Roof: middle cranial fossa

- Medially: posterior semicircular canal of the inner ear and posterior cranial fossa
 - Laterally: skin behind auricle
 - Floor: jugular bulb, digastric muscle, sternocleidomastoid muscle
 - Anteriorly: middle ear cavity, external auditory canal and facial nerve
 - Posteriorly: sigmoid sinus

- Inner ear:

The inner ear is in charge of both hearing and balance functions. It is composed from membranous surrounded by bonny labyrinth (which means mazes of tunnels).

The membranous labyrinth filled with an intracellular fluid similar in structure to blood ultra-filtrate called endolymph and surrounded by an extracellular fluid similar in structure to CSF called perilymph.

- Blood supply: labyrinthine artery a branch from anterior inferior cerebellar artery
 - The membranous labyrinth consists of:
 - Cochlea: responsible for hearing
 - Sacule
 - Utricle
 - 3 semicircular canals



Vestibulocochlear nerve:

It is the eighth cranial nerve, which transmits sound and balance information from the inner ear to the brain.

Physiology of hearing

Hearing:

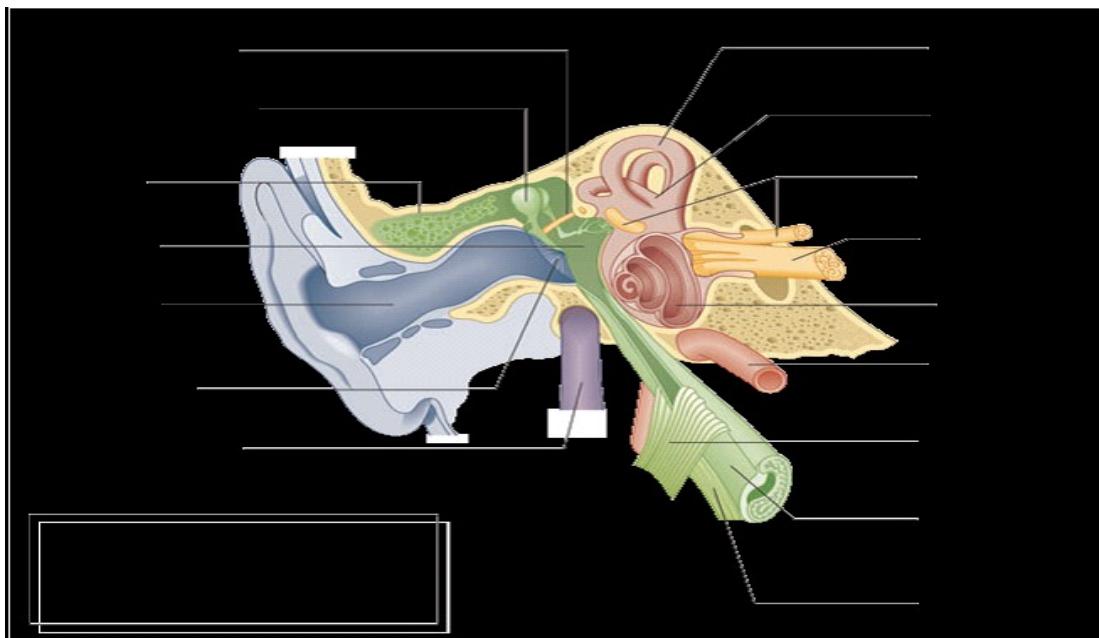
The ability to perceive sound by detecting vibrations through the ear.

- Sound results from vibration of molecules within the air in the form of pressure waves.
- Hearing in humans have very important role in social interaction as well as detection of hazardous situations.
- Speech is the most important manifestation of hearing.

The human hearing apparatus consists of:

- Peripheral auditory system

- Central auditory system



Peripheral auditory system:

The main function is to receive air- pressure sound stimulus and convert them into electrical signals to interpret by the auditory center.

- It consists of:
- Outer ear (pinna and external auditory canal)
- Middle ear (tympanic membrane, Ossicles, auditory muscles)

- Inner ear (cochlea with its hair cells, fluids, and auditory nerve)

In humans, hearing achieved primarily by the auditory system by which mechanical waves, known as vibrations are detected by the ear and converted into nerve impulses, which travel through the auditory nerve and then perceived by the brain.

Central auditory system:

It began at the brainstem, where the auditory nerve ends at auditory nucleus, and then through the Pones and midbrain tell reach the auditory cortex in the temporal lobe of the brain.

- The main function:
- Interpret process and analyses the sound signals.
- Sound localization
- Sound recognition

Mechanism of hearing:

In order to understand the complex mechanism of hearing, the process of sound detection and transfer can divided into 3 part depending on the site being in action:

- **Outer ear:**

It has two main functions:

- The pinna act as an ear trumpet to collect sound waves into the ear canal and then concentrating them toward the eardrum. Because of the asymmetrical character of the outer ear, sounds waves move differently on its way into the ear depending on the vertical location it is coming from. This gives the ability to localize sound.
- Sound localization provided by binaural interactions.

- **Middle ear:**

The middle ear transfer sound energy to the cochlea and providing physical protection for the cochlea.

It include the following:

- The eardrum (tympanic membrane) is an airtight membrane and the sound waves cause it to vibrate following the waveform of the sound.
- The Ossicles: are small bones located in the middle ear cavity arranged in a unique manner (malleus-incus-stapes-oval window) aid in the transmission of the vibrations from the eardrum to the inner ear.
- The purpose of its unique construction is to prevent the impedance mismatch between air and water, by providing impedance matching.
- The impedance matching achieved through the following:
 - The first principle is the area of the vibrating tympanic membrane (55cm^2) is larger than that of the stapes footplate in the cochlea (3.2 cm^2), so this will increase the sound pressure over the oval window.
 - The second principle is the lever action of the middle ear bones.
 - The third is contraction of both middle ear muscles (tensor tympani and stapedius muscles) by increasing the stiffness of the ossicular chain, which will help to protect the cochlea from loud noises.
- The result is amplification of sound by around 18 times in order to bypass the impedance mismatch between air in the middle ear and fluid in the inner ear.

- **Inner ear:**

Cochlea: a spiral-shaped, fluid-filled tube. It contains the organ of Corti, which is the main organ of mechanical to neural transduction through its special hair cells.

The mechanical sound waves after traveling through the middle ear structures reach the stapes footplate and then the oval window of the cochlea. These sound waves cause vibrations in the perilymph fluid of the cochlea, and through complex ion channels active transport and shearing forces of the hair cells of organ of corti, which will cause transduction of energy and conversion of mechanical sound energy into electrical impulses, which will travel through the auditory nerve to the brain.

- **Auditory nerve:**

The sound information from the cochlea travels by the auditory nerve to the cochlear nucleus in the brainstem and then to the primary auditory cortex in the temporal lobe.

Neurotransmitter released from the hair cells causes action potentials in the auditory nerve fibers. The electrical impulses are specific for each sound frequency and precise frequency matching achieved by:

- Phase locking: the neurotransmitter release is synchronous with the sound stimulus and with the action potential generation.
- Auditory nerve fibers code information by means of their frequency selectivity, so each frequency has a specific nerve fiber to transmit.

Physiology of body balance

Balance: A complex interaction between the vestibular, ocular, proprioceptive and central nervous systems (CNS) to maintain head and body position in relation to the environment.

The vestibular apparatus:

The sensory system, which provides the sense of balance and orientation of the body for coordination of movement with balance. It is part of the inner ear labyrinth consisting from specialized sensory cells embedded in inner ear fluids and surrounded by a bonny cover.

It consists of:

- The peripheral vestibular system: which include the semicircular canals, utricle, Sacule and the vestibular nerve.

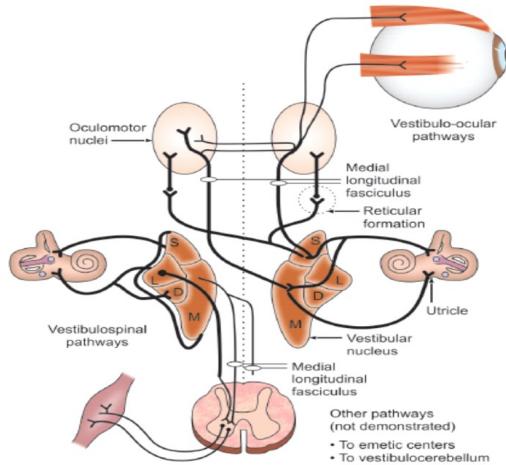


Fig. 4.1: Vestibular pathways

- Central vestibular system: which include the vestibular nuclei and its central connections in the brainstem and cerebellum.

In general, movements is either linear or rotational.

The vestibular system comprises two components:

- Semicircular canals: which detect rotational movements
- Utricle and Sacule: which detect linear accelerations e.g. is gravity.
- The vestibular system sends signals primarily to the neural structures that control eye movements, and to the muscles that keep the body upright. The brain uses information from the vestibular system and from proprioception throughout the body to maintain its position and acceleration from moment to moment.
- The connection between the vestibular system and the eyes provide the anatomical basis of the vestibulo-ocular reflex (VOR) which is required for clear vision.
- The connection with the muscles provide the vestibulo-spinal reflex (VSR) which control posture necessary to keep body upright.

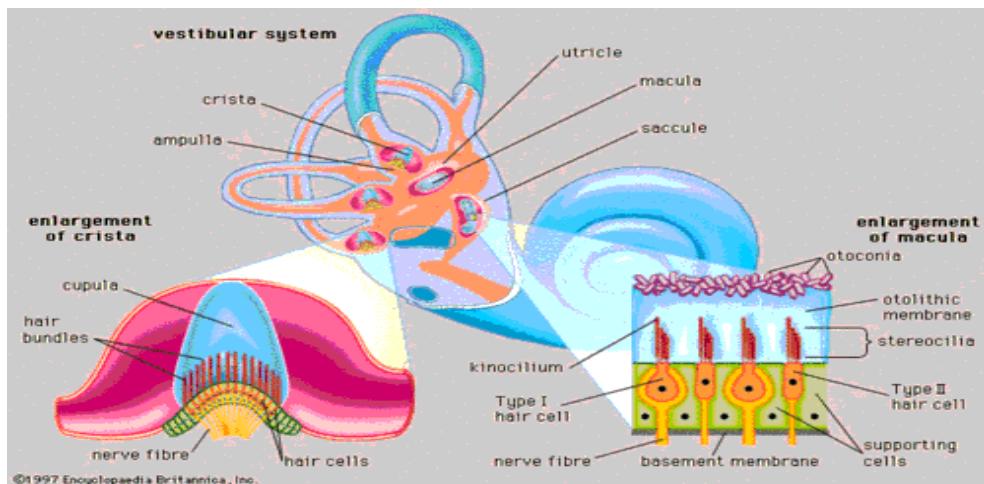
The semicircular canals:

The vestibular system contains three semicircular canals in each labyrinth filled with endolymph and surrounded by perilymph.

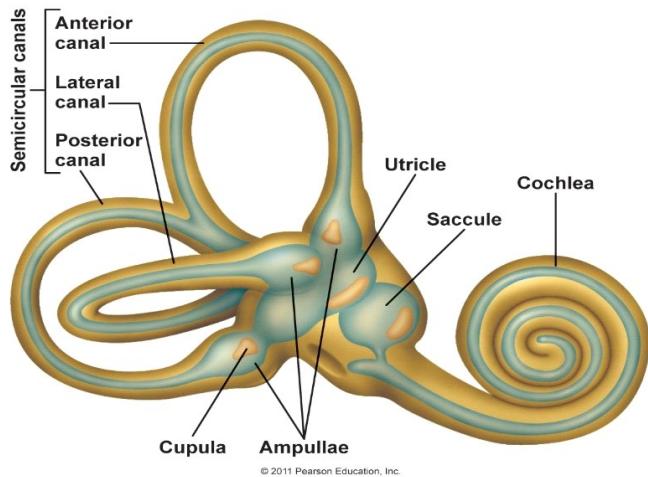
The main sensory organ here is cupula, which contain the sensory hair cells responsible for conversion of mechanical fluid movement's stimuli into electrical signals that transmitted by the vestibular nerve into the brain for analysis.

- The three semicircular canals are approximately right angles to each other:
- The lateral (or horizontal) semicircular canal (LSCC)
- The superior(or anterior) semicircular canal (SCCC)
- The posterior (or inferior) semicircular canal (PSCC).

Movement of fluid within the horizontal semicircular canal corresponds to rotation of the head around a vertical axis (i.e. the neck), as when doing a pirouette (spinning around on single foot like in ballet dance).



The anterior and posterior semicircular canals detect rotations of the head in the sagittal plane (as when nodding), and in the frontal plane, as when spinning. Both anterior and posterior canals are orientated at approximately 45° between frontal and sagittal planes.



Utricle and Sacule:

Those called the otolithic organs as they sense linear accelerations.

The main sensory organ in each utricle and Sacule is the macula, which contain a patch of hair cells.

The brain interprets head orientation by comparing inputs from each Utricle and Sacule with that of other side and to other input from the eyes and stretch receptors in the neck.

The vestibular nerve:

Eighth cranial nerve (Vestibulocochlear nerve) that is responsible for transferring information from the peripheral vestibular system to the brain. It have 2 divisions:

- Superior vestibular nerve (SVN): supply the SSCC, LSCC and Utricle
- Inferior vestibular nerve (IVN): supply the PSCC and Sacule

Central vestibular system

The vestibular nuclei on both sides of the body process and integrate vestibular and nonvestibular stimuli and give impulses that affect the oculomotor and spinal motor systems. Both sides connected with each other, which is important to maintenance of equilibrium.

VOR: is the most important efferent reflex of the vestibular system directed towards the ocular muscles nuclei. The reflex help to maintain visual fixation during movement.

- Disorder in this reflex give raise to Nystagmus

VSR: an efferent reflex from the vestibular system which spinal motor activity including head position, upright gait and postural stability.

- Disorder in this reflex will affect spinal motor function and coordination.
- Disorder in the vestibular system give raise to vertigo

Vertigo: A hallucination of movement that is either rotatory or translational. It is a cardinal symptom of a disorder in the vestibular system either peripheral or central.

Nystagmus: An involuntary, rhythmic movement of the eyes due to a disturbance in vestibulo-ocular reflex (VOR).

Audiology

Simply: the science that study hearing, speech and balance disorders.

Audiologist is a hearing health care professional who can identifies and assesses individuals with hearing and/or balance problems.

Properties of sounds:

- Human ears can detect vibrating sound frequency ranging from 20-20000 Hz.

- These sound waves carried through out a suitable media like air in a speed of 340 m/sec. If no media, like in vacuum, the ears cannot detect sound stimuli.
 - The sound energy measured by Decibel (dB). The human ears detects sounds energy in the range of 0-120 dB as in:
 - Threshold of hearing = 0 dB
 - Whispering = 20 dB
 - Conversational speech = 60 dB
 - Threshold of pain = 120 dB
 - Human ears had a specific field of hearing especially for speech perception in the range of 200-8000 Hz and with sound energy level range from 20-80 dB.
-
- **Humans listen to sounds by 2 means:**
 - Hearing by air conduction: sound waves reach the ear by propagating in the air, entering the external auditory canal, and setting the tympanic membrane in motion; the movement of the tympanic membrane, in turn, moves the malleus, incus, and stapes of the middle ear. The structures of the middle ear serve as an impedance-matching mechanism, improving the efficiency of energy transfer from the air to the fluid-filled inner ear and then these mechanical vibrations transformed into electrical signals transmitted to the auditory center in brain for analysis.
 - Hearing by bone conduction: occurs when the sound source, in contact with the head, vibrates the bones of the skull; this vibration produces a traveling wave in the basilar membrane of the cochlea directly, which in return converted into electrical signals transmitted by the auditory nerve into the brain.

Hearing loss

Hearing loss is very common and has a widespread spectrum ranging from a almost undetectable degree of disability to a profound loss of ability to work in society.

Approximately 10% of the adult population has some hearing loss. Often, this loss presents early in life. However, hearing loss can present at any age. 40% of people over the age of 75 have hearing loss.

Types of Hearing loss:

Hearing loss can result from disorders of the auricle, external auditory canal, middle ear, inner ear, or central auditory pathways.

In general, hearing loss classified as:

- Conductive hearing loss: which results from lesions in the auricle, external auditory canal, or middle ear cause.
- Sensorineural hearing loss: that tends to result from lesions in the inner ear or eighth nerve.
- Mixed hearing loss: had both conductive and sensorineural elements.

Degree of Hearing Loss:

- Normal: 0-15 dB
- Borderline: 16-25 dB
- Mild: 26-40 dB
- Moderate: 41-55 dB
- Moderately severe: 56-70 dB
- Severe: 71-90 dB
- Profound: > 90 dB

Audiometry

A part of audiology, which concern with the means of detection, measuring and assessment of patient's hearing disability and the possible means of correction and curing hearing impairment.

Audiometry done in a quiet room in order to prevent external noises from interfering with the test and to prevent adaption.

The ear canal must cleared before audiometric measurements in order to prevent misleading results.

Tests for Hearing Loss:-

To detect hearing impairment we have a group of test, which classified into:

- Subjective: depends on a clear response from the subject tested on.
- Objective: no need for subject response.

• Subjective tests include the following:

1-Reflex Test

- The test done at infants at age: 0-6 months
- changes in child activity observed in response to a sound stimulus include: eye widening, eye blink, arousal from sleep, startle or definite movement of the arms, legs or body

2-Distraction test

- the test done at age 6-18 months
- It is a subjective test based on the principle that the normal response observed when sound presented to a baby is a head turn to locate the source of sound.

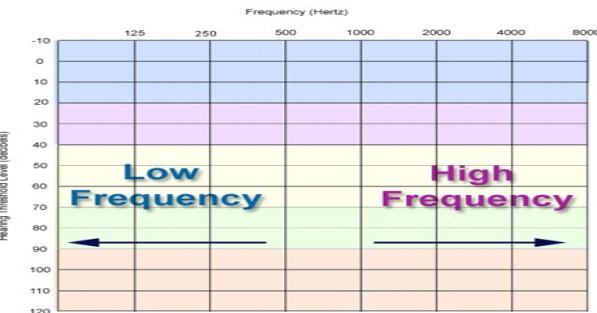
3- Performance test

- The test done at age: 2-5 years
- It is a subjective test tails the principle that the child trained to wait for a sound and then to respond with taking a toy from a table in front of the child.

4- Pure tone audiometry

- Usually this test carried out at age 3 years and above.

- Audiometry is a subjective test of hearing in which measurement performed using an electronic instrument called "audiometer" by delivering specific signals called pure tones at different frequency and intensity.



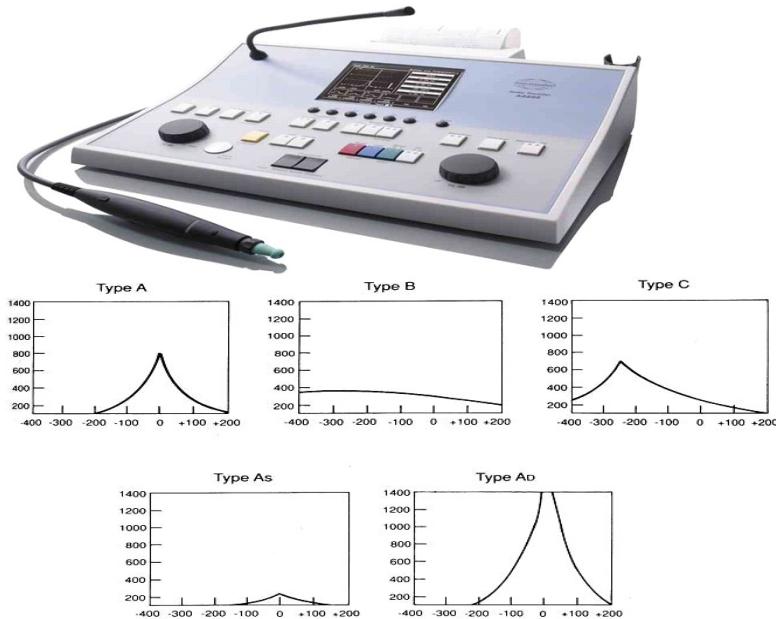
The audiogram is a graph which shows specific frequencies (pure tones) and intensity levels (in decibels, dB) which identify what a person can hear in each ear separately.

5-Speech audiometry:

Uses words present in every day communication instead of pure tone.

- Objective tests include:

1-tympanometry



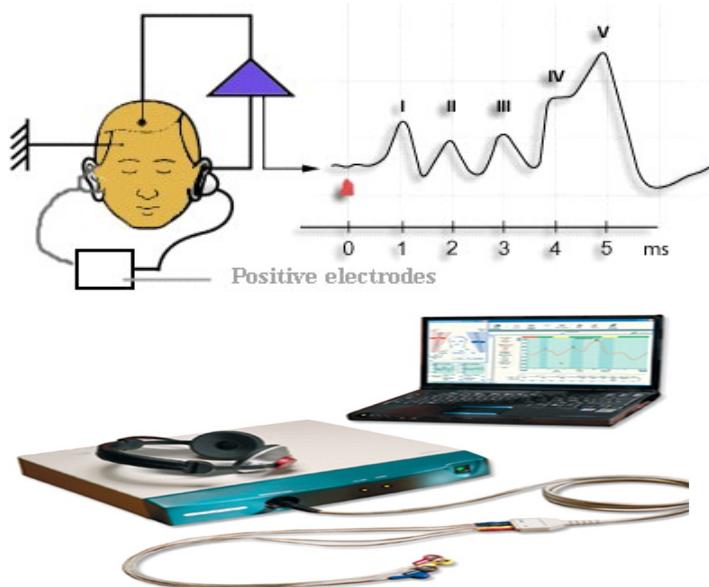
It measures the pressure in the middle ear and movement of the tympanic membrane and is useful in detecting disorders of the middle ear using an electronic instrument called tympanometer and the results recorded in a specific graph called tymanogram.

2-Acoustic Reflex

It refers to the involuntary muscle contraction within the middle ear. It normally occurs in response to high-intensity sounds to protect the ear from acoustic trauma.

3- Auditory Brainstem Response (ABR)

- This test emphasizes on responses in brain waves that are stimulated by a clicking sound to assess the auditory pathways.
- With the assistance of a specialized machine, it is possible to extract the tiny electrical signals by electrodes placed on the patient's head when evoked in the brain by acoustic stimulation throughout the ears.
- Used for infant hearing screening, assessment of central auditory nervous system, monitoring of auditory nervous system function during surgery.



4-Otoacoustic Emissions (OAE) -

- Otoacoustic emissions are low-intensity sounds generated by the cochlea and come into the middle ear and ear canal.

- Useful for infant screening, pediatric hearing assessment and cochlear function monitoring. There are two types of Otoacoustic emissions:-
 - 1-Spontaneous OAEs (SOAEs)
 - 2-Evoked OAEs (EOAEs)

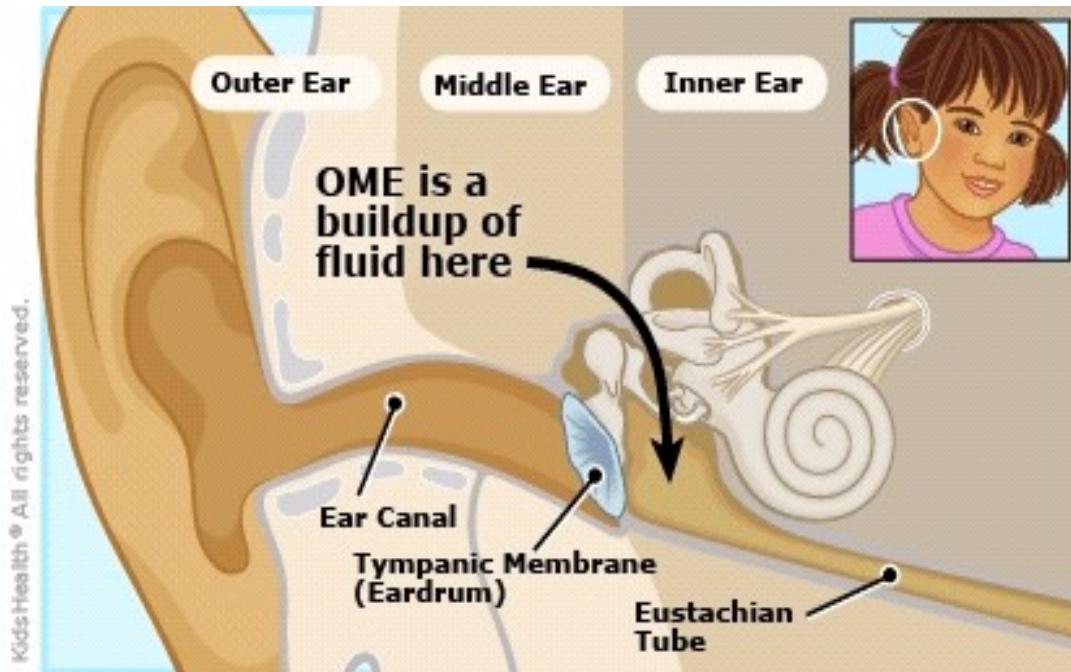
Otitis media with effusion

Also called: secretory otitis media (SOM), non-suppurative otitis media, otitis media with effusion (OME), serous otitis media, chronic non-purulent otitis media and glue ear

Definition:

OME defined as the chronic collection of mucus and secretions within the middle ear and occasionally the mastoid air cell system.

much simply, OME is dense or sticky fluid behind the eardrum.



Epidemiology:

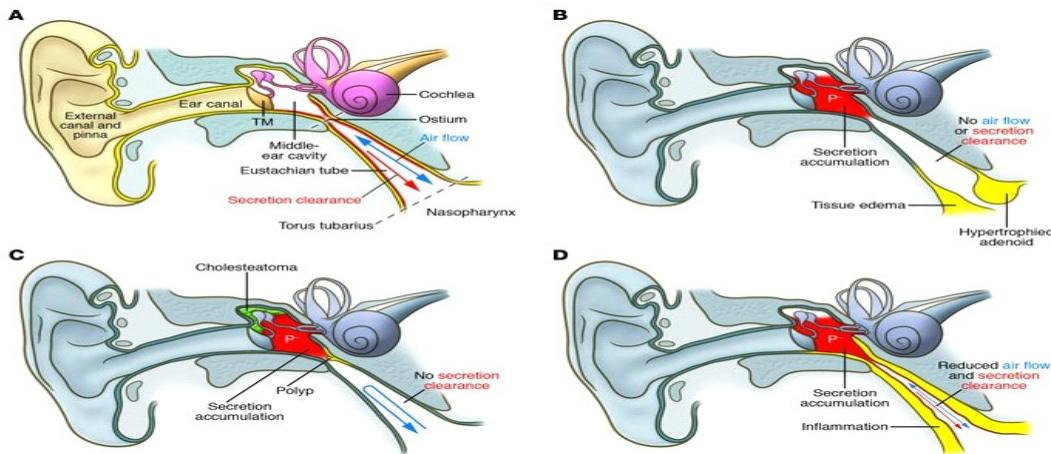
Otitis media with effusion (OME) is one of the most common causes of acquired conductive hearing loss in children.

There is a bimodal distribution with the first peak at two years of age, at which children first attend a playgroup or nursery school. Then, the prevalence drops, until around five years of age when the prevalence increases when most children start attending a primary school.

There is an equal sex distribution. OME is more common during winter months due to high frequency of URTI.

Pathophysiology:

- Eustachian tube dysfunction is the main etiological factor, which affect ventilation to middle ear. It is caused by obstruction to the Eustachian tube due to infection or inflammation secondary to allergies, upper respiratory tract infection (URTI), mass or tumor in the postnasal space area or trauma. A negative pressure develops within the middle ear cavity leading to transudation of fluid from the mucosa with accumulation of a serous, essentially sterile effusion.



- Because of inflammation of the middle ear mucosa caused by a reaction to bacteria already, present in the middle ear.

Risk factors:

- Anatomical factors: Eustachian tube dysfunction, craniofacial anomalies like cleft palate, Down syndrome
- Immunological factors: children with congenital or acquired immunodeficiency, chronic bacterial colonization in middle ear cavity. More recently, analysis of middle ear fluid aspirated in OME found no pathogenic Bacteria in more than 60 %,
- Environmental factors: bottle feeding, daycare attendance, allergy to common environmental entities, lower socioeconomic status, smoking parents.

Clinical features:

- Hearing loss may be the only symptom. Parents usually discover their child poor hearing, which worsen during sleep and lying down.
- Sometimes there is mild pain or fullness.
- Sometimes there is history of repeated episodes of AOM.
- Adults come with unilateral otitis media with effusion and usually present with aural fullness and/or pressure, an ear being plugged, or decreased hearing, pain is rare. In addition, adult may give history of nasal obstruction which, necessitate post nasal space examination looking for mass or tumor in this particular area.

Note: in adults, OME points to diagnosis of nasopharyngeal tumor until prove otherwise.

Examination:

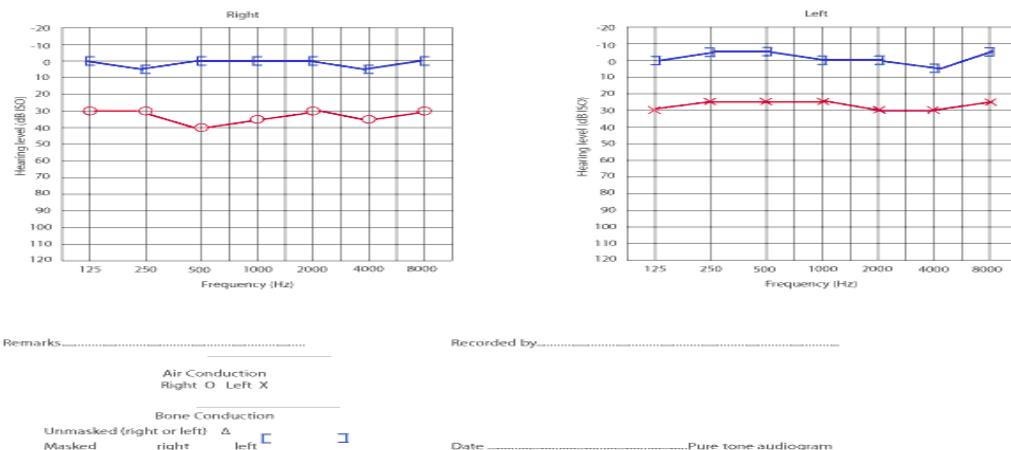
- Pneumatic otoscopy is the most important part of the physical examination for OME.
- The tympanic membrane in OME is often opaque and may appear yellow or blue in color.
- Air-fluid levels or bubbles sometimes seen behind an intact tympanic membrane.
- In addition, there is decreased mobility of the Tympanic membrane.
- In children, tonsillar hypertrophy and adenoid hypertrophy is present.
- Additional findings may include turbinate hypertrophy, postnasal drip and rhinorrhea.
- Postnasal space examination using flexible or rigid naso-endoscopy may show adenoidal hypertrophy, mass or tumor in this area.



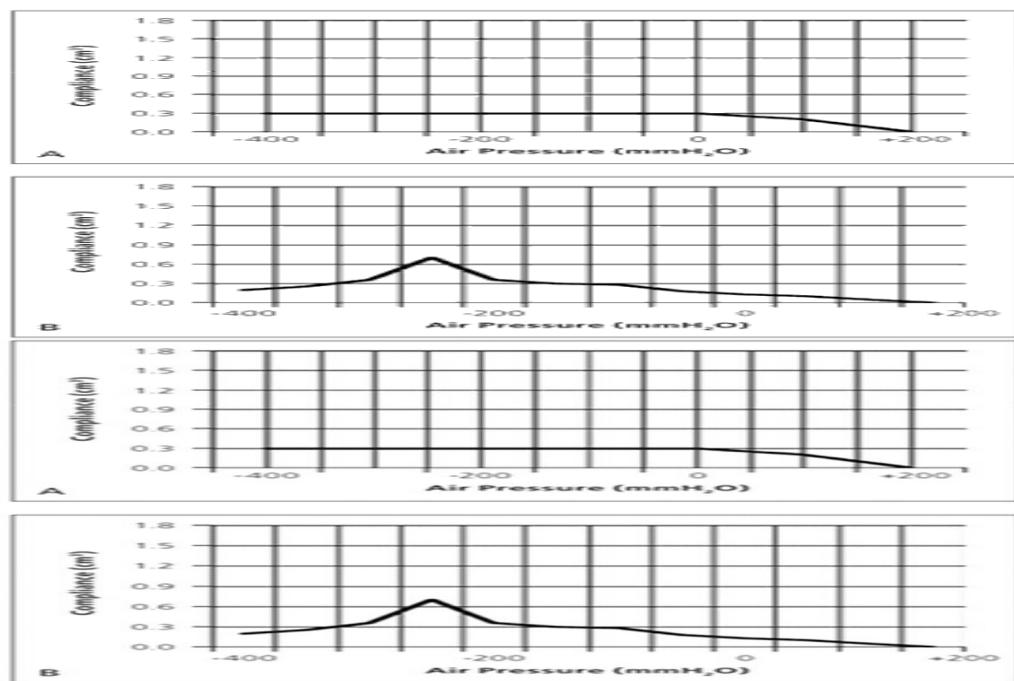
Evaluation of hearing impairment:

OME usually associated with conductive hearing loss.

- Tuning fork test: 1. Rinne test: Air conduction less than Bone conduction
- 2. Weber test: lateralize to ear with disease or more hearing loss
- Pure tone audiometry: conductive hearing loss (Air-Bone gap) up to 40-45 dB



- Tympanometry: reduce middle ear compliance with type B or C tyma

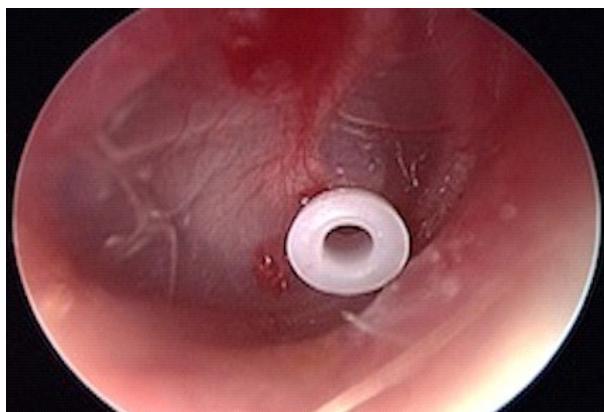


Investigation:

Diagnosis of OME is clinical based on history, clinical examination and supported by audiological investigations in form of pure tone audiometry (PTA) and tympanometry.

Treatment:

- Watchful waiting: In the majority of cases, OME is self-limiting disease with resolution of most of symptoms in 3 months.
- Medical treatments include Antibiotic, Antihistamine, steroids and nasal decongestant.



- Surgical treatments: is reserved to persistent cases of OME in the form of Myringotomy with or without ventilation tube insertion plus adenoidectomy.

Follow up:

The otolaryngologist should observe patients until the condition resolves with medical or surgical intervention. If the patient's hearing is normal, no more active managements needed. If a recognized hearing loss is present, re-evaluation of patient hearing required.

Prognosis:

Otitis media with effusion (OME) is the leading cause of hearing loss in children. This condition is associated with delayed language development in children younger than 10 years, and the loss is usually conductive.

The prognosis for otitis media with effusion is good. Most episodes spontaneously resolve without intervention, and many resolve undiagnosed. Still, 5% of children not treated surgically have persistent otitis media with effusion for 1 year.

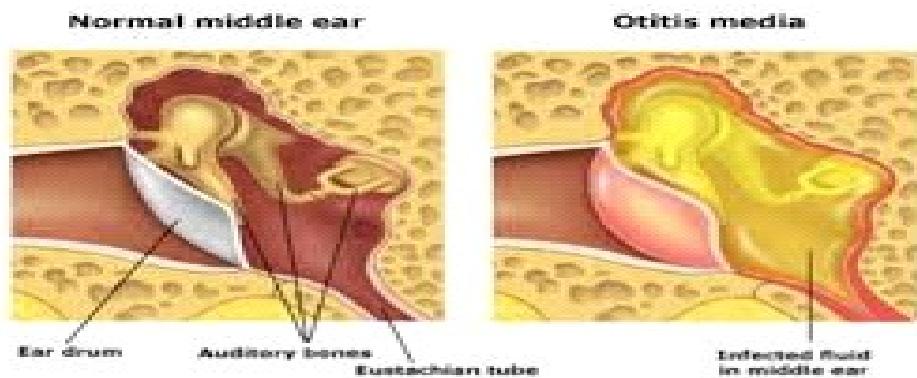
Acute otitis media

Definition:

Acute inflammation of the middle ear cleft: middle ear cavity, Eustachian tube and mastoid air cells.

Acute otitis media (AOM) is the most common disease demanding medical therapy for children younger than 5 years.

Often bilateral especially in children but unilateral cases occur.



Duration of disease:

- The term acute, refer to disease duration less than 3 weeks.
- If disease duration is 3 weeks – 3 months, called sub-acute. More than 3 months called chronic otitis media.

Epidemiology:

It occurs most usually in children and it is important to manage with attention to prevent the following serious complications.

It most commonly follows an acute upper respiratory tract infection and may be viral or bacterial in origin.

Etiology:

AOM can result from both viral and bacterial causes.

Viral: 60-90 % of cases of AOM may be associated with viral infection:

- Respiratory syncytial virus (RSV);
- Influenza A virus
- Parainfluenza viruses
- Human rhinovirus
- Adenoviruses.

Bacterial: either primary bacterial infection from the start or usually bacterial infection complicate viral infection (secondary bacterial infection).The bacteria responsible for acute otitis media are:

- Streptococcus pneumoniae 35%
- Haemophilus influenzae 25%
- Moraxella catarrhalis 15%.

- Group A streptococci and *Staphylococcus aureus*.

Risk factors for AOM:

- Genetic factors like down syndrome and Turner syndrome
- Immunological factors like decreased or defective immune system
- Environmental factors like day-care attendance, parental smoking and poor socioeconomic status, bottle feeding, cold weather,

Pathology:

Eustachian tube dysfunction affect middle ear ventilation with development of negative middle ear pressure and accumulation of fluid in middle ear cavity (effusion), that may infected by different organisms.

Organisms enter the middle ear through:

- Eustachian tube
- Tympanic membrane
- Blood stream

The causative organism causing inflammation, edema, exudate and pus. Edema closes the Eustachian tube, preventing aeration and drainage, pressure from the pus rises, causing the tympanic membrane to swell and bulge, which may cause necrosis of the tympanic membrane and perforation. Finally, the ear continues to drain pus until the infection resolves.



Clinical features:

Symptoms varies according to age:

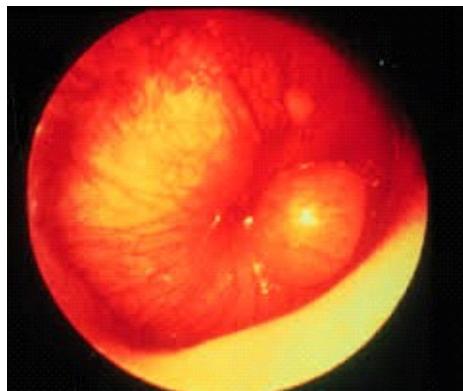
- Neonates: Irritability or feeding problems, high-grade fever and even convulsions.
- Infants: fever, vomiting & diarrhea, otalgia, ear pulling and discharge from the ears.
- Older children and adults: Hearing loss, otalgia, discharge from the ears and fever.

Signs on examination:

- The child appear unwell, and may rub ear.
- Pneumatic otoscopy is the diagnostic tool of choice because it allows assessment of the tympanic membrane inflammation, Bulging,

Perforation and pus alongside with assessment of tympanic membrane mobility.

- The tympanic membrane appears brightly red, bulging with no light reflex.





Investigation:

If diagnosis of AOM made, no need for further investigations.

Treatment:

- **Supportive measures:**
 - Bed rest
 - Antipyretics like paracetamol or ibuprofen.

- **Antibiotic:**

Antibiotic therapy should start immediately and continue for 7-10 days.

Penicillin is the drug of choice, but amoxicillin, Co-amoxiclav is also useful.

Erythromycin used in case of penicillin allergy.

Antibiotic eardrop have no role in AOM without tympanic membrane perforation.

If perforation or tympanostomy tube present, ciprofloxacin eardrop used.

- **Surgical treatment:**

Surgery (Myringotomy) of limited role in AOM, but used in case of complications or severe pain and bulging of the tympanic membrane.

Prognosis:

AOM in 80% of cases is a self-limiting disease.

Lacking antibiotic treatment, suggestive relief from pain and fever occurs in about 60 % of children after 72 hours of diagnosis. With antibiotic, rapid relief of pain and fever occur in almost all patients within 24-48 hours.

Complication of AOM:

AOM can results in serious complication, so carful diagnosis and managements required.

- Extracranial complications:
 - Tympanic membrane: perforations, sclerosis
 - Middle ear effusion
 - Acute mastoiditis
 - Facial nerve paralysis
 - Labyrinthitis (inner ear infection)
 - Pertositis (infection of the petrous bone apex)
- Intracranial complications:

- Meningitis
- Extradural abscess
- Subdural empyema
- Sigmoid sinus thrombosis
- Brain abscess

Paediatric hearing loss

Types and etiology

There are 3 types of hearing loss in children:

A/ Conductive HL

It is caused by problem in the outer or middle ear, in which the sound waves are not sent to the inner ear correctly. Conductive HL is the most common type of hearing loss in children and it is usually acquired, although it can be congenital. Causes of conductive hearing loss include:

- ear infection (otitis media) or fluid behind ear drum
- perforation of the ear drum
- excessive wax
- foreign bodies
- tumours

B/ Sensorineural HL (SNHL)

It is caused by loss of function within the inner ear or with the connection to the brain. It can be congenital or acquired. Causes of congenital SNHL:-

- Genetic cause: About 1 out of 2 cases of hearing loss in babies is due to genetic causes. Some of those babies have family members who also have hearing loss. About 1 out of 3 babies with genetic HL have syndrome e.g Down & Usher's syndrome
- About 1 out of 4 cases of HL in babies is due to maternal infection during pregnancy (e.g CMV), lack of oxygen during birth, complication after birth *e.g (Jaundice, meningitis, head trauma)

C/ Mixed HL

- Children with SNHL also can have middle ear problems e.g fluid in the middle ear. This will make hearing loss worse.

Signs and symptoms of hearing loss

A/ Signs in babies:

- Does not startle at loud noise
- Does not turn to the source of sound after 6 months of age
- Does not say single words e.g (dada or mama) by 1 year of age

B/ Signs in children:

- Speech is delayed
- Speech is not clear
- Does not follow directions
- Turn the TV volume up too high
- Lack of response when his name is called
- Difficulties in school
- Repeated earaches
- Difficulty in understanding what people are saying

Diagnosis

Early diagnosis of hearing loss is vital to allow intervention and appropriate support to avoid development, social and academic delay

- 1- Newborn hearing screening: it measures the child's physical response to quiet sounds. The most common newborn hearing tests are otoacoustic emissions (OAE), auditory brain stem evoked response(ABR)
- 2- Behavioural hearing assessment:- determine the softest sounds that the child can hear (their hearing threshold). Sounds are presented through air conduction (microphones) and bone conduction by device that send vibrations to the skull.

- 3- Tympanometry:- Assess how well the ear drum is functioning. Tympanogram is obtained by placing rubber device in the ear and the ear drum movement is measured in response to air pressure changes.
- 4- Acoustic reflex: it assess the function of a small reflexive muscle in the middle ear (stapedial reflex). Loud noise is presented to the ear through small rubber tip. It provides information about the nerve pathway along the nerve arc.
- 5- otoacoustic emission (OAE): assess the function of the hair cells in the inner ear. These emissions can predict the presence of normal (or no more than mild) hearing loss.
- 6- Auditory brainstem evoked response (ABR):
This test is usually done while the child is sleeping. Electrodes are placed on the forehead and near each ear. This test is often used aid in appropriately fitting a hearing aid for an infant who is not yet capable of behavioral testing. It is also helpful in other populations, who are not developmentally able to have their hearting tested conventionally
- 7- Genetic testing: It provides accurate understanding of the cause of hearing loss and provides guidance regarding treatment and long term medical management

Treatment and management

- medical treatment: e.g treatment of otitis media by antibiotics, decongestants, wax removal
- hearing aids: make sounds louder. They can be worn by people of any age: including infants. Younger children are usually fitted with behind – the – ear style hearing aids
- FM systems: send sounds to the ears to help the child to hear better. They work especially in noisy situations, e.g classrooms
- Surgery: to correct structural deformity. It is an option for some children with chronic ear disease or to restore conductive HL caused by tumours or problems with the eustachian tube.
- Cochlear implant: can be appropriate option for children who are not candidate for other options.
- Speech therapy
- Family support devices

Tumours of nasopharynx

Benign tumours of nasopharynx

Benign tumours of nasopharynx are rare. These include:

- juvenile angiofibroma
- chondroma
- dermoid
- teratoma
- Hamartoma
- Rhabomyoma
- haemangioma
- craniopharyngioma

Juvenile angiofibroma

It is the most common benign tumour of nasopharynx. It is histologically benign but locally aggressive vascular tumour. The most common site of origin is the posterior part

of nasal cavity close to the sphenopalatine foramen. It is seen almost exclusively in adolescent males (testosterone dependant tumour)

Clinical features

The most common symptom is profuse and recurrent epistaxis. Other nasal symptoms may occur which include progressive nasal obstruction, hyponasal speech, anosmia and broadening of nasal bridge. The patient may present with otological symptoms (otalgia, secretory otitis media). Proptosis may occur when there is intraorbital extension. Diplopia occur secondary to erosion of superior orbital fissure and due to 3rd and 6th cranial nerves palsies.

Examination may show pink or purple mass obstructing one or both choana

Diagnosis

- CT – scan with contrast is now investigation of choice. It shows extent of the tumour , bony destruction and anterior bowing of posterior wall of maxillary sinus (Antral sign or Halman muller sign) which is pathognomonic for angiofibroma
- MRI (for soft tissue extention)
- Carotid angiography

Treatment

Surgical excision is the treatment of choice. Preoperative embolization and estrogen therapy or cryotherapy or radiotherapy reduce blood loss during. Surgery-surgical approaches include.

- Transpalatal approach done for tumour confined to nasopharynx
- Lateral rhinotomy for larger tumours involving nasal cavity and paranasal sinuses
- Transpalatine and sublabial approach (Sardana's approach)
- Nasal endoscopic approach

Malignant tumours of nasopharynx

Nasopharyngeal carcinoma (NPC)

It is the most common cancer originating in the nasopharynx. It differs significantly from other head and neck cancers in its occurrence, causes, clinical behaviour and treatment. It is most common in males at (50-70 years of age). It is squamous cell carcinoma or undifferentiated carcinoma. It arises from lateral nasopharyngeal recess or Rosenmuller fossa.

Causes

NPC is caused by combination of factors (viral, environmental and hereditary factors) The Viral influence is associated with infection with EBV. Other etiological factors include genetic susceptibility , consumption of food *(salted fish) containing

carcinogenic volatile nitrosamines, burning of wood (polycystic hydrocarbones), tobacco smoking

Classification

- Type I : Keratinizing sequamous cell carcinoma
- Type II
- Type II a : Non keratinizing sequamous cell carcinoma
- Type II b : Non Keratinizing undifferentiated carcinoma (lymphoepithelioma). It is strongly associated with EBV

Clinical features

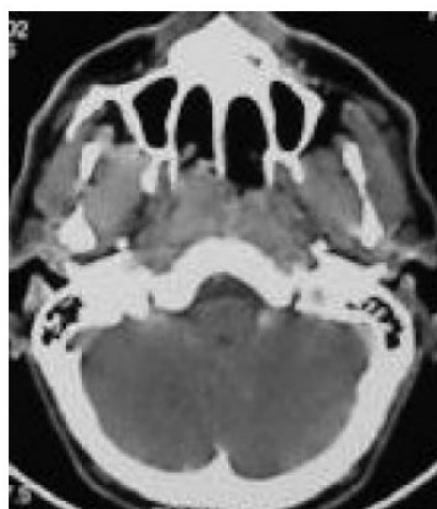
The commonest complain at presentation is cervical LAP (60-90%). Nasal obstruction , post nasal drip, blood stained discharge and epistaxis seen in 30%. Aural symptoms like deafness (otitis media with effusion) and otalgia seen in 20% of patients. Cranial nerve pulsies occur late and involve the last 4 cranial nerves. Trismus may occur in direct infiltration of pterygioid m. Horner's syndrome is rare

Staging

- Stage I: small tumour confined to NP
- Stage II : tumour extending in the local area, or with any evidence of limited nodal involvement
- Stage III: large tumour with or without cervical lymphnode, or tumour with bilateral neck lymphnodes
- Stage IV: large tumour involving intracranial or infratemporal regions , extensive cervical lymphnodes involvement and / or any distant metastasis

Investigation

The most useful investigation is CT-scan of the nasopharynx. The definitive diagnosis is made by biopsy for histopathology



Contrast-enhanced CT scan shows nasal involvement resulting from nasopharyngeal carcinoma.

Treatment

- Radiotherapy is the treatment of choice in NPC. Radical neck dissection is done for recurrent nodal involvement when the primary tumour has been controlled. Cryosurgery can be done in case of recurrence. Chemotherapy with cisplatin or cisplatin and 5 -FU in combination with radiotherapy for stage III and IV.

Hearing loss in adult

Definition of hearing loss

It is decrease ability to perceive sounds. It can be parital or total, sudden or gradual, temporary or permanent

Classification of hearing loss

- 0 – 25 dB : Normal hearing level
- 26 – 40 dB : mild hearing loss
- 41 – 55 dB : moderate hearing loss
- 56 – 70 dB : sever hearing loss
- 71 – 90 dB : very sever hearing loss
- > 90 dB : profound hearing loss

Types and causes of hearing loss

A/ **Conductive HL** : It results from defect in the outer and middle ear that prevent sounds from reaching to the inner ear. Causes of conductive HL include :

- Obstruction of external auditory canal with wax , haematoma and foreign bodies

- Perforated ear drum caused by direct trauma, middle ear infection or explosions
- Otitis media and serous otitis media
- Dislocated ossicles , usually result from ear trauma
- Otitis externa that causes canal swelling
- Retraction of tympanic memberane , which may be associated with cholesteatoma

B/ Sensorineural hearing loss (SNHL)

It result from lesion in the inner ear (cochlea), 8th cranial nerve or central auditory pathway. Causes of SNHL:

- acoustic trauma: chromic exposure to loud noise causes the hair cells on the cochlea to become less sensitive
- head trauma : fracture of temporal bone can disrupt the nerve on the auditory system or the cochlea
- ototoxic drugs : certain drugs can affect the hearing by damaging the nerves involved in hearing. E.g aminoglycosides (gentamycine and vancomycine), diuretics (furosemide), salicylates (Aspirin) , NSAID, antineoplastic drugs
- vascular diseases e.g SCA, D.M
- renal disease
- meniere's disease: that affect hearing and balance. It has gradual onset and may progress to deafness and sever vertigo. It is usually associated with tinnitus. The cause is unknown, but thought to be due to fluid shift in the inner ear
- acoustic neuroma (tumor of auditory nerve in the cerebellopontine angle).
- Aging
- Infections e.g herpes zoster, meningitis.

C/ Mixed hearing loss

In this type, the patient complain from both conductive hearting loss and SNHL e.g CSOM , blast injury

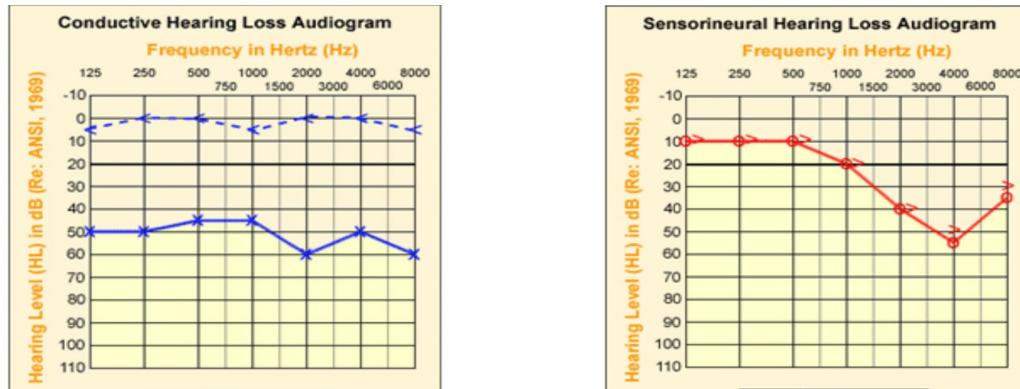
Clinical features

- If hearting loss is sudden, it may be due to trauma , acute inflammation or problem with blood circulation. Gradual onset is suggestive of other causes e.g aging , tumor
- If there are associated symptoms like tinnitus or vertigo, it may indicate problem in the nerve or inner ear or brain
- It may be unilateral or bilateral. Unilateral hearing loss is most often associated with conductive causes, trauma and acoustic neuroma
- Pain in the ear associated with infection, trauma and obstruction in the ear canal. Ear infection may also cause fever and discharge

Examination and tests

- a) The ear canal and tympanic memberane should be inspected with otoscope
- b) The nose, nasopharynx and oropharynx should be examined

- c) General neurological examination which include examination of the nerves that control movement, sensation and reflexes
- d) Tuning fork test: each ear should be tested separately
 - Rinne test: the doctor strikes the tuning fork and place it in front of external ear canal and ask the patient when he no longer hear the sound. Then, the doctor move the fork to the base of mastoid and ask the patient when he no longer hear the sound.
- In normal hearing and SNHL, the patient hear by air conduction longer than bone conduction (positive Rinne), while in conductive HL, the patient hear by bone conduction longer than air conduction (negative Rinne)
- Weber test : the doctor strike the tuning fork and place it on the forehead or on incisive teeth. In normal hearing, the patient hear the sound in both ears. In conductive HL, the sound lateralized to the deaf ear, while in SNHL, the sound lateralized to the normal ear
- e) Pure tone audiometry (PTA) : this test can determine the type of hearing loss
- f) Tympanometry : this test evaluate the ability of tympanic membrane to move
- g) CT-scan and MRI are indicated in certain conditions like acoustic neuroma.



Treatment

This include treatment of underlying causes. Wax and foreign bodies should be removed from ear canal. Infection should be treated with antibiotics. If hearing loss is due to ototoxic drugs, these drugs should be stopped or changed. If associated symptoms are troublesome e.g (tinnitus and vertigo), medications should be prescribed. If meniere's disease is suspected, certain diuretics and antihistamines or nicotinic drugs can be helpful and low salt diet may be suggested. If hearing loss is sudden, oral steroids may be started. If there is tumor e.g acoustic neuroma, referral to neurosurgeon will be made

Prevention

- Wearing protective plugs in case of long term exposure to loud noise e.g workers in factories with high noise
- Know the possible side effects of medications
- Avoid putting foreign bodies in the ear

Facial nerve

Seventh cranial nerve or simply cranial nerve VII

Embryology

The facial nerve developmentally derived from the second [pharyngeal arch](#) during the third week of gestation and fully developed by the eleventh week. In the neonate, the facial nerve anatomy similar to that of an adult, except for its location in the mastoid, which is more superficial.

Anatomy of facial nerve:

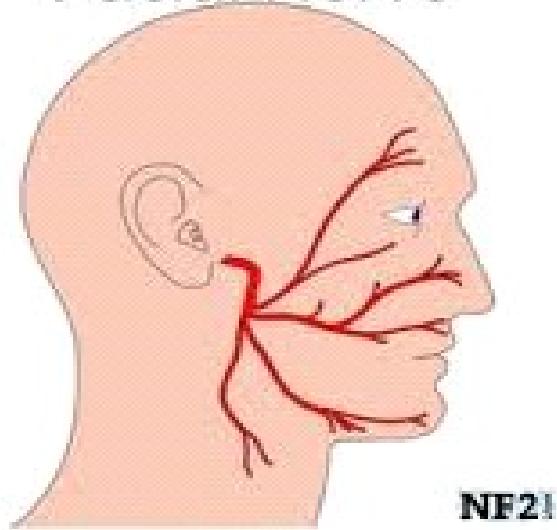
The facial nerve is the nerve supplying the muscles of facial expression. It contains more than 10,000 myelinated fibers, about 7000 are motor and the remaining are sensory and secretomotor. Its nucleus is located in the pons and the nerve begins in the cerebellopontine angle (CPA).

It is associated with the nervus intermedius, which carries secretomotor fibers to the salivary glands of the head and neck (except the parotid gland) from the superior salivary nucleus. This nerve also carries the taste fibers from the anterior two thirds of the tongue.

The facial nerve enters the internal auditory meatus with the VIII nerve and travels through the petrous temporal bone to emerge on the medial surface of the middle ear. Here, the nerve can anatomically be divided into 4 segments:

- Metal segment: 13-15 mm
- Labyrinthine segment: the shortest segment only 3-4 mm
- Tympanic segment: 8-11 mm

Facial Nerve



Mastoid segment: 10-14 mm give rise to stapedius nerve that supply the stapedius muscle.

The nerve then exits the skull at the stylomastoid

foramen then travels between the digastric and stylohyoid muscles to enter the parotid gland where it divides into its final 5 branches:

- Temporal
- Zygomatic
- Buccal
- Marginal mandibular
- Cervical

The five branches supply the facial muscles. The nervus intermedius runs with the facial nerve, giving off the greater petrosal nerve and the chorda tympani, which carry secretomotor fibers to the submandibular and sublingual salivary glands and carries taste fibers from anterior two thirds.

Facial nerve paralysis:

VII nerve can be involved in a variety of pathological conditions due to its long, complex pathway starting from VII nerve nucleus in the brain, throughout its passage in the ear and temporal bone and finally throughout the parotid gland.

The paralysis can be upper or lower motor neuron in nature, unilateral or bilateral, acute or persistent.

Note: UMN facial nerve paralysis may spare the muscles of the upper face due to bilateral cortical innervation.

Causes of facial nerve paralysis:

Classification of facial nerve injuries:

For a pathophysiological point of view, VII nerve paralysis classified as follow:

- Neuropraxia: there is a weakness in the function of VII but without anatomical defect.
- Axonotmesis: there is a defect in the myelin sheath but with intact perineurium and axon fibers.
- Neurotmesis: complete defect with loss of perineural sheath and axon fibers

Diagnosis of facial nerve paralysis:

- **History and clinical examination:**

Complete history taken from the affected patients regarding the cause, onset, duration and progress of facial nerve paralysis together with complete general, neurological, ENT, head & neck examination.

- **Imaging:**

CT scanning and MRI are essential in the diagnosis of damage to intra-temporal and intracranial parts of the facial nerve, like temporal bone fracture patterns (vertical, transversal, mixed) and edema formation.

- **Electrophysiological studies**

Beneficial to decide the degree of nerve disruption, outcome, and treatment options through percutaneous stimulation of the facial nerve.

These studies include:

- maximal stimulation test (MST)
- Electroneuronography (ENog).

Bell's palsy

An acute, unilateral lower motor neuron facial nerve paralysis affecting the entire side of the face. The etiology is unknown, but a viral infection of the facial nerve or an immunological response to viral infection especially represent the most acceptable theory.

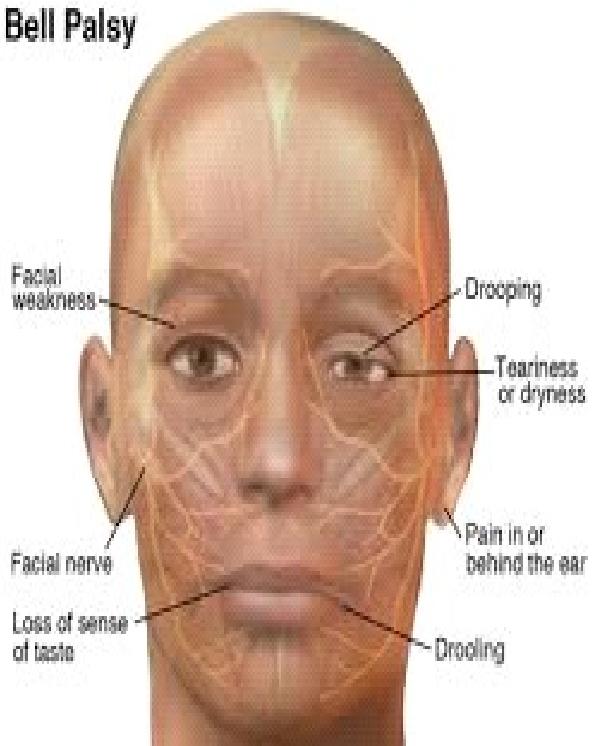
The following viruses have been implicated in the etiology of Bell's palsy:

- herpes simplex virus type 1 (HSV-1) is the major etiology
- herpes simplex virus type 2 (HSV-2)
- human herpes virus
- varicella zoster virus (VZV)
- Influenza B, adenovirus, Coxsackie virus and Epstein-Barr virus (EBV).

Sometimes, a history of upper respiratory tract viral infection precede the paralysis in less than 48 hours. A deep aching pain behind the ear may also precede the paralysis.

Usually affect young and middle age 15-45 yrs. with no sex preference.

Bell Palsy



Presentation:

The patient usually presented with complete paralysis or asymmetry of one side of the face involving the frontal region, loss of taste, dry eye, and pain and increase sensitivity to noise (hyperacusis).

No other systemic manifestation, neurological symptoms or otological symptoms.

Investigation:

Bell's palsy is a diagnosis of exclusion.

No need for other investigations.

Prognosis:

Normal and complete recovery occur in two third of patient within 3 months. Usually no recovery occur after 6 months of initial presentation.

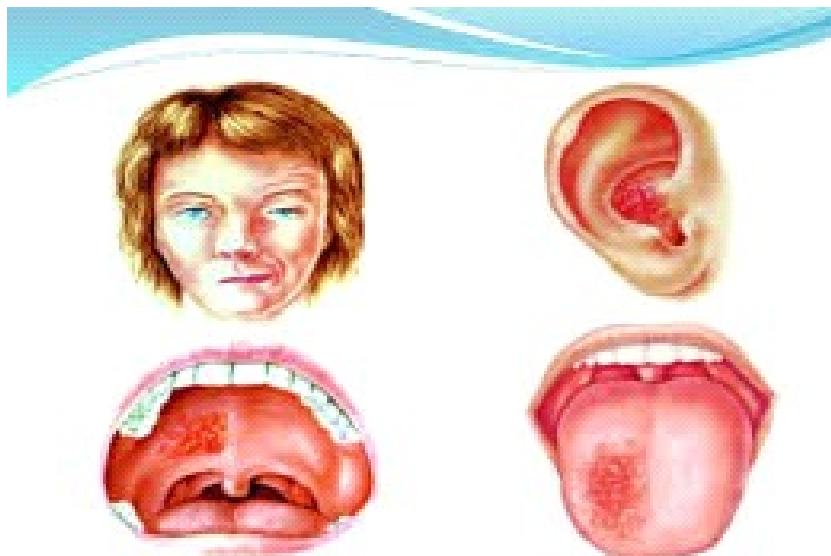
Treatment:

- Steroids (prednisolone) in a dose of 1 mg/kg/day for a 5 days followed by tapering of dose for 10 days.
- Antiviral (acyclovir) in a dose of 200mg five times a day for 10 days.
- Eye protection with wet, sterile napkin and artificial tears.
- Physiotherapy

Herpes Zoster Oticus

Also called Ramsay Hunt syndrome.

It is a pathological disorder triggered by the reactivation of pre-existing Varicella zoster virus (VZV) in the geniculate ganglion or the facial nerve, which cause peripheral facial nerve palsy and erythematous vesicular rash on the same ear or mouth side.



Presentation:

The condition proceeded by severe, lancinating pain in the ear or pharynx associated with sensorineural hearing loss together with facial palsy and skin rash.

Diagnosis:

Based mainly on the clinical picture together with raising VZV antibodies titers by PCR.

Treatment:

A combination of steroids (1 mg/kg prednisolone) for 5 days followed by 10 days taper together with antiviral agent (acyclovir 800 mg five times daily) for 2-3 weeks.

Prognosis:

Worse than Bell's palsy. Persistent facial nerve paralysis occur in 30-50 % of cases and only 10 % who do completely recover.

Cerebellopontine angle tumors

Definition of cerebellopontine angle :

It is a structure at the margin of the cerebellum and pons. It is the one of lateral cistern containing CSF, arachnoid tissue, cranial nerves and vessels. It is a common site for the growth of acoustic neuroma.

Contents of cerebellopontine angle :

It contains the following structures :

- 1 : Facial nerve .
- 2 : Vestibulocochlear nerve .
- 3 : Flocculus of the cerebellum .
- 4 : Lateral recess of the 4 th ventricle .

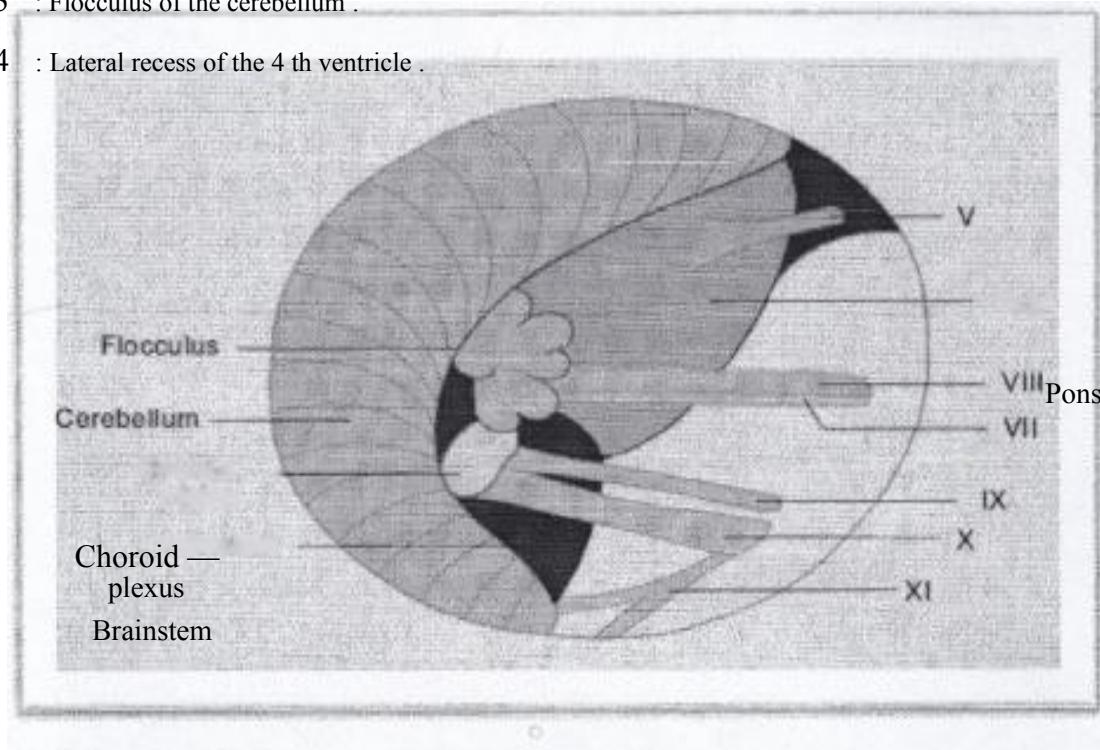


Figure 1: shows anatomy of cerebellopontine angle.

Cerebellopontine angle tumors:

They are the most common tumors in posterior cranial fossa, accounting for 5 - 10 % of intracranial tumors . Most of them are benign with over 85 % being vestibular schwannoma (acoustic neuroma).

Differential diagnosis of cerebellopontine angle tumors:

The most frequent non acoustic cerebellopontine angle tumors are meningioma (313%) , epidermoid (2- 6%) , facial and lower cranial nerves schwannomas(1-2%), arachnoid cyst (1 %), lipoma , primary and secondary malignancies(less than 2%), lipoma, vascular malformations and haemangiomas.

Clinical manifestation of cerebellopontine angle tumors:

Presenting symptoms of cerebellopontine angle (including acoustic neuroma) include the following :

- Hearing loss 95%.
- Tinnitus 80%.
- Vertigo 50 -75 % .
- Headache 25 % .
- Facial hyperesthesia 35 - 50 % .
- Diplopia 10 % .

The presenting symptoms can vary according to the size and location of the lesion Other symptoms

depending on the type of the tumor like in :

meningioma:

- Trigeminal or facial nerve symptoms are likely to occur earlier than hearing loss.
- Patient with large tumor can present with obstructive hydrocephalus and / or symptoms of brainstem compression.

Epidermoid :

- These can become large without symptoms .
- Facial twitching and progressive facial paralysis are more prominent than other cerebellopontine angle tumors .
- Patient may present with cranial nerves or cerebellar dysfunction .

Facial nerve schwannoma:

- Patient can present with conductive hearing loss from the middle ear involvement, parotid mass from extratemporal involvement, sensorineural hearing loss from internal auditory canal or cerebellopontine angle involvement.
- Hemifacial spasm is common .
- cerebellopontine angle lesions do not cause facial weakness until the tumor is very large.

Lower cranial nerves schwannoma:

- Patient may have weakness or hyposthesia of the palate , vocal cord and shoulder (cranial nerves 9 , 10 , 11) or hemiatrophy of the tongue (cranial nerve 12).
- Large tumors may cause deficit of all lower cranial nerves .

Diagnosis :

Examination and tests :

Often, the physical examination is normal when the tumor is diagnosed , Some times, the following signs may present:

- Decrease feeling on one side of the face .
- Drooping of one side of the face .
- Unsteady walk.

Investigation:

The most useful tool to diagnose cerebellopontine angle tumore is MRI of the brain . Other tests include :

- Hearing tests (audiology).
- Electronystagmography .

Figure : MRI of brain showing left cerebellopontine mass (arrow) compressing brain stem .

Treatment:

Treatment depends on the size and location of the tumor , patient age and overall health . Many of cerebellopontine angle tumors are small and grow very slowly, small tumors with few or no symptoms can be followed with regular MRI scanning (especially in elder patients).

A . Surgery :

Removal of cerebellopontine angle tumors is indicated in the following large tumors :

- Tumor causing symptoms .
- Tumor that are growing quickly .
- Tumor that are pressing on the brain .

In these conditions , tumors are removed by surgery .

B . Stereotactic radiosurgery :

- Focuses high powered X - Ray on a small area . It is indicated :
 - 1- To slow down or stop the growth of tumors that are hard to remove with surgery .
 - 2 - To treat patients that are unfit for surgery e.g elderly and very sick patients .

Complications of the surgery :

Removing cerebellopontine angle tumors can damage nerves, causing loss of hearing and weakness of facial muscles . Any hearing that is left is often lost with surgery.

Anatomy and function of nose and paranasal sinuses

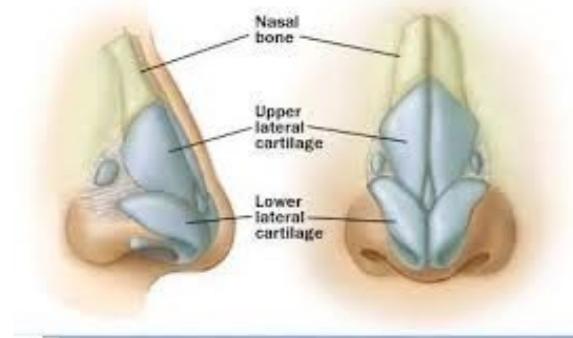
The external nose is triangular bonny cartilaginous pyramid with the apex facing superiorly and the base facing inferiorly. Its internal space is the nasal cavity proper which is opened anteriorly to the exterior by nostril (anterior nares) and posteriorly to nasopharynx by posterior nares (choane)

A. external nose:

The boundary connecting free apex to tip of base is called the dorsum of the nose which is formed superiorly by nasal bridge and inferiorly by ala of the nose. The external nose covered by skin which is firmly adherent at ala and contain numerous sebaceous glands and hair follicle while loosely adherent at nasal bridge.

The bony framework of external nose formed by pair of nasal bones that unit in the midline to form nasal bridge, attached superiorly to nasal spine of frontal bone, laterally to frontal process of maxilla and inferiorly to upper lateral cartilage.

Cartilaginous framework consists of two pairs of fibro elastic cartilages called lateral cartilage (upper and lower), accessory alar cartilage with the contribution of septal cartilage. The upper lateral cartilage is triangular shape attaching superiorly to nasal bones and anteriorly to dorsal part of septal cartilage. The lower lateral cartilage (greater alar cartilage) form major part of ala forming anterior boundary of anterior nares. The medial crus of lower lateral cartilage join that of the other side forming columellar cartilage. Two group of muscles acting on external nose dilators and constrictors all are supplied by facial nerve. The chief muscle action is for the dilators to dilated the nostril during forceful respiration.



Branches of the facial artery supply the alar region while the dorsum and lateral walls of the external nose are supplied by the dorsal branch of the ophthalmic artery and the infraorbital branch of the maxillary. The frontomedian area drains to the facial vein and the orbitopalpebral area to the ophthalmic vein with interconnections to the anterior ethmoidal system and thence cavernous sinus which can be of clinical significance.

The skin of the external nose receives its sensory supply from ophthalmic and maxillary divisions of the trigeminal nerve.

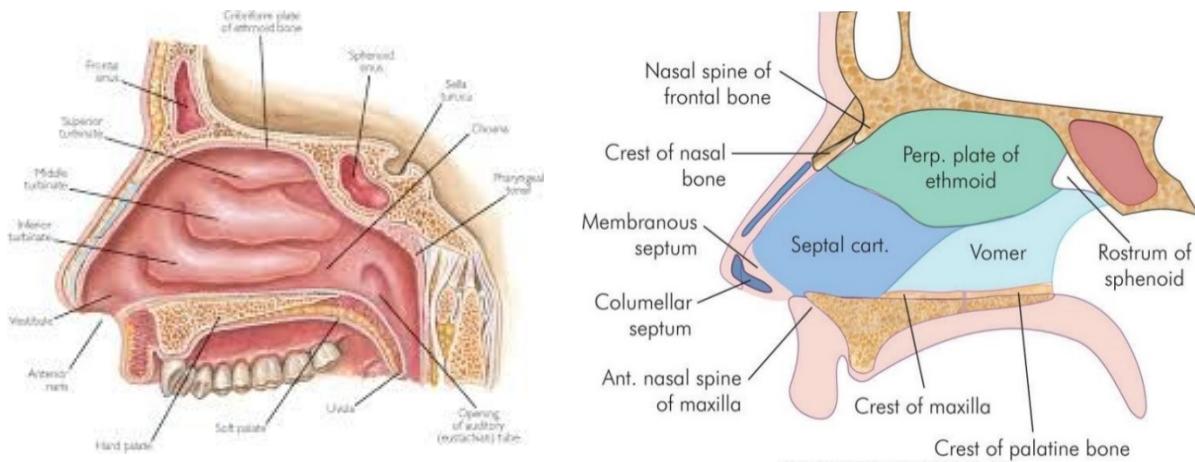
Lymphatic drainage of the external nose with the anterior face is to the submandibular and submental nodes, with buccal nodes adjacent to the facial vein sometimes intervening. There may also be bilateral drainage and flow to the parotid region is possible.

B.Nasal cavity proper

The nasal cavity is divided into two sides by nasal septum, it is opened anteriorly and posteriorly by anterior and posterior nares (choana) respectively. The nasal cavity covered mostly by respiratory epithelium (pseudostratified ciliated columnar epithelium) apart from small area of specialized epithelium responsible for olfaction called olfactory mucosa and other small anterior area called vestibule lined by skin containing hair follicle.

The floor is formed by palatine process of maxilla and horizontal plate of palatine bones. Roof is formed by nasal bones, inferior surface of the nasal spine of the frontal bone, cribriform plate of the ethmoid and inferior surface of the body of sphenoid bone.

The *medial wall* of the nasal cavity is formed by the nasal septum. The antroinferior part is cartilaginous called quadrilateral cartilage, posterosuperiorly formed by perpendicular plate of ethmoid and the rest formed by vomer bone.



The lateral wall of the nose has three bony elevations covered with mucosa called *turbinates*. There are three turbinates —superior, middle and inferior. While the inferior turbinate is a separate bone, the middle and superior turbinates are parts of the ethmoid bone, Below and lateral to each turbinate there is a mucosa covered depression called meatus corresponding to each turbinate.

The superior turbinate drains the posterior ethmoidal air cells. While the complex anatomy of middle meatus contributes to ostemeatal complex that drains anterior group pf paranasal sinuses. The nasolacrimal duct drains to anterior portion of inferior meatus where a mucosal flap cover its opening forming the valve of hanser.

The mucosa covering each turbinate has an erectile function formed by specialized network of thin walled veins called sinusoids that are capable of expansion under

parasympathetic or allergic stimulation leading to turbinate enlargement. The recoiling of mucosal elastic fibers decreases the size of turbinates in response to vasoconstriction caused sympathetic stimulation.

C.Paranasal sinuses:

Paired air filled cavities lined with mucus membrane occupying the bones of skull that are all opened into the nose. They are divided into two groups according to relation to ground lamella of middle turbinate.

The anterior group include maxillary, frontal and anterior ethmoidal air cells that all drain into middle meatus while posterior group include posterior ethmoidal and sphenoid sinus that drain to superior meatus and sphenoethmoidal recess respectively.

The maxillary sinus antrum presents at birth as small cavity lateral to middle turbinate then gradually increase in size during the first 2 years of life during primary dentation. Another phase of development starts at 5-year age with the beginning of secondary dentation till reach to full adult size by age of 18 years where it would be about 15 mm in capacity. Maxillary sinus ostium lies in upper part of its medial wall and drains into middle meatus.

Ethmoidal sinus is a complex structure so it called ethmoidal labyrinth consist of two group of air cell according to relation to ground lamella of middle turbinate. The anterior ethmoidal air cells are larger and fewer in number and drain into middle meatus whereas the posterior cells are smaller and numerous in number and drain into superior meatus.

The frontal sinus is the most variable in size and shape. At birth, the frontal sinuses are small and cannot usually be differentiated from other anterior ethmoidal cells. In the fifth year of life, Pneumatization starts the substance of frontal bone via frontoethmoidal recess and extends superiorly and at twelve years the sinus is largely developed. Pneumatization may continue throughout adolescence till reach to full adult size by age of twenty. the sinus is usually L-shaped composed of a horizontal and a vertical compartment but, in addition, diverticula and incomplete septa are frequently encountered. An intersinus septum is usually present, but may be paramedian and is partially dehiscent in 9 percent.

The sphenoid sinus is recognizable as a small cavity at birth. At the third year of life, Pneumatization of the sphenoid bone progresses and at age seven has frequently reached the floor of the sella. In adults, degrees of pneumatization vary greatly and usually asymmetrical. The sinus cavities are variable in size and shape. It is completely absent in approximately 1 percent of the population.

Function of nose and paranasal sinuses

The nose forms the gateway of the respiratory system and serves the following important functions.

1. *Respiratory passage:* Normally, breathing takes place through the nose. Infants breath obligatory through their nose and
2. *Filtration:* The nose filters the inspired air effectively by:
 - a. Vibrissae (nasal hair) in the nasal vestibule arrest large molecules of the inspired air.
 - b. The fine particles and bacteria are deposited on the mucus blanket which covers the nasal mucosa. The mucus contains various enzymes like lysozymes having antibacterial properties.
3. *Air conditioning and humidification.*
4. *Vocal resonance:* The nose and paranasal sinuses serve as vocal resonators and nasal passages are concerned with production of nasal consonants like M and N. Thus obstructions of the nasopharynx and nose alter the tone of voice (rhinolalia clausa).
5. *Nasal reflex functions:* The receptive fields of various reflexes lie in the nose. These include sneezing, and nasopulmonary, nasobronchial and olfactory reflexes. These protect the mucosa and regulate the vasomotor tone of the blood vessels. Olfactory reflexes influence salivary, gastric and pancreatic glands.
6. The *nasal cavity serves as an outlet for lacrimal and sinus secretions.*
7. *Olfaction.*

Physiology of breathing and olfaction

The nose serves as an air conditioning unit and performs three functions: humidification, heat transfer and filtration.

Humidification and heat transfer:

The nose has good ability to transfer heat and ten percent of the body heat loss occurs through the nose in humans.

The temperature of the inspired air can vary from - 50 to 50°C. Saturation follows the temperature rise rapidly.

During inspiration, Energy is required for warming of inspired air (1/5) and the latent heat of evaporation (4/5). The amount of energy depends on ambient temperature and relative humidity of inspired air. whatever temperature of inspired air is, air in the post-nasal space is approximately 31°C and is 95 percent saturated.

While during expiration, there is drop of temperature of the saturated expired air from slightly below body core temperature at the back of the nose into about 32°C at anterior nares so water condenses onto the mucosa, approximately one-third of the water required to humidify the inspired air is recovered in this way. People who breathe in through the nose and out through the mouth will dry the nasal mucosa.

Water comes from the serous glands, which are extensive throughout the nose. Additional water comes from the expired air, the nasolacrimal duct and the oral cavity. Capillary leakage occurs during inflammation,

AIRFLOW

during inspiration, The air flows upwards and backwards from the nasal valve initially, mainly over the anterior part of the inferior turbinate. It then splits by the middle turbinate, into small part passing into olfactory area and major part passing below middle turbinate into the posterior choana. the velocity at the anterior valve is 12-18 m sec - 1 during quiet respiration.

Expiration lasts longer than inspiration and is more turbulent (Figure). Extrapulmonary airflow is turbulent because the direction changes, marked change in caliber and the walls are not smooth. This turbulence together with nasal resistance create eddy current that aerate the paranasal sinuses.

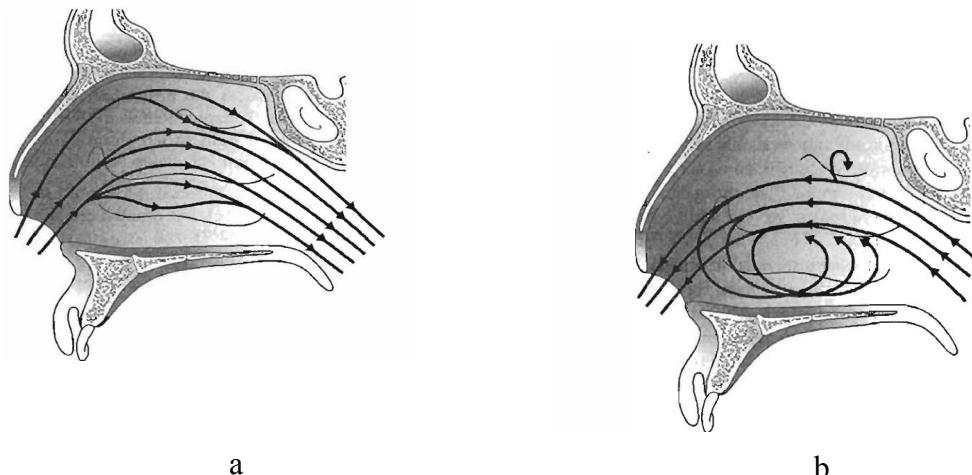


Fig () direction of air flow a. during inspiration b. during expiration

Nasal resistance

The nose accounts for up to half the total airway resistance. The resistance is made up of two elements; one essentially fixed (anatomical) composed by the bone, cartilage and attached muscles, and the other variable, the mucosa where it is variable due to nasal cycle.

The anterior nasal valve is the narrowest part of the nose and is less well defined physiologically than anatomically. It is formed by the lower edge of the upper lateral cartilages, the anterior end of the inferior turbinate and the adjacent nasal septum, together with the surrounding soft tissues.

Nasal cycle

The cycle consists of alternate nasal blockage between passages that occur between four and 12 hours; they are constant for each person. Its physiological significance is uncertain. The changes are produced by vascular activity, particularly the volume of blood on the venous sinoids (capacitance vessels).

In addition to a resistance and flow cycle, nasal secretions are also cyclical with a decrease in secretions in the more obstructed side. Various factors may affect the nasal cycle and include allergy, infection, exercise, hormones, pregnancy, fear and emotions, including sexual activity. The autonomic nervous system controls the changes; vagal overactivity may cause nasal congestion. Drugs, which block the action of noradrenaline, cause nasal congestion. The anticholinergic effects of antihistamines can block the parasympathetic activity and produce an increase of sympathetic tone, hence an improved airway. Times of hormonal changes, such as puberty and pregnancy, affect the nasal mucosa. These are probably mediated directly on the blood vessels.

PROTECTION OF THE LOWER AIRWAY:

A. MECHANICAL AND CHEMICAL

The nose protects the lower airway by removing particles down to approximately 30 Mm, including most pollens from the inspired air. The shape and roughness of smaller particles may cause them to be deposited in the nose. Inspired air velocity drops markedly just after the nasal valve. Turbulence increases deposition of particles.

This protection is done by vibrissae that are numerous in the nasal vestibule (trapping the large molecules) and mucociliary blanket that trap and neutralize smaller particles and evacuate them into nasopharynx to be swallowed and destroyed by acidity of stomach.

Nasal secretions are composed of two elements, mucus and water. Mucus is produced by the mucus glands and the water and ions are produced mainly from the serous glands and indirectly from transudation from the capillary network. Glycoproteins found in mucus are produced in two cell types, the goblet cells within the epithelium and the glandular mucus cells.

The anterior part of the nose contains serous glands only in the vestibular region. These produce a copious watery secretion when stimulated. Sinuses have fewer goblet cells and mixed glands.

The composition of mucus is outlined below:

- water and ions from transudation;
- glycoproteins: sialomucins, fucosmucins, sulphomucins;
- enzymes: lysozymes, lactoferrin;
- circulatory proteins: complement, macroglobulin, C reactive protein;
- immunoglobulins: IgA, IgE, IgG, IgM, IgD;
- cells: surface epithelium, basophils, eosinophils, leukocytes.

Both quality and quantity of the secretion are important and require an intact blood supply and nervous system. Mucins are packaged by the Golgi apparatus in 1-2 mm droplets. Droplets absorb water when secreted, enlarging rapidly over a three second period. Goblet cells respond directly and the exocrine glands secrete through parasympathetic stimulation via muscarinic receptors, M₁ and M₃.

B. IMMUNOLOGICAL:

Mucus contains a number of different compounds able to neutralize antigens, either by innate mechanisms or by learned or adaptive immunological responses.

Lactoferrin, lysozymes, complement, antiproteases and other macromolecules can neutralize a number of bacteria, particularly those without capsules, to give an innate nonspecific immunity. Polymorph leukocytes and macrophages phagocytose and destroy foreign material. Many organisms and viruses are resistant and so specific reactions are required including humoral or cell mediated immunity reactions.

On exposure to an antigen, most immunoglobulins activate complement resulting in cell lysis and phagocytosis. IgA and IgE are mainly present on the surface, and IgM and IgG act if the mucosa is breached.

IgA accounts for up to 70 percent of the total protein in nasal secretions and is divided into two subgroups: IgA 1 and IgA2, IgA1 is a monomer frequently found in the serum and, IgA2 is more common in nasal secretions and is a dimer. IgA dimer diffuses passively into interstitial fluid and is actively taken up by the seromucinous glands and surface epithelium.

In epithelium, a secretory piece is attached to IgA, renders it stable in mucus. When it reacts with an antigen, an insoluble complex is formed, which is swallowed and destroyed by stomach acid. IgA does not activate complement.

IgE is the main immunoglobulin involved in allergic reactions. It is produced mainly in lymphoid aggregates such as the tonsils and adenoids and within the submucosa. IgE is firmly attached to mast cells and basophils (two molecules of allergenic specific reaction). IgE should attach to specific receptor sites on mast cells to cause mast cell degranulation. IgE does not activate complement.

Several bacteria and viruses cannot be neutralized by humeral reaction and require the activation of the cell-mediated immune responses. Antigens are often presented by macrophages or dendritic cells to T lymphocytes. Dendritic cells are important in the allergic response. Two groups of cytokines act on CD4 + T cells and gives rise to two main responses, the Th1 response and the Th2 or allergic response.

OLFACTION

Olfaction initiates and modifies behaviour in any creatures. Olfactory compounds need high water and lipid solubility to be perceived. The solute in mucus is presented to the sensory mucosa. Man discriminates a large number of different smells but the olfactory mucosa and pathway fatigues and subsequently recovers quickly.

Olfaction is mediated by G-protein coupled receptors in the cells which interact with a specific adenyl cyclase within the neuroepithelium. Receptors are coded by between 500 and 1000 genes, but each cell has one or two specific receptors.

Odours react with the lipid bilayer of the receptor cells at specific sites, which causes K⁺ and Cl⁻ efflux and thus depolarize the cells. After a latent period of up to 400 ms, a slow compound action potential, the electroolfactogram (EOG), is recorded from olfactory mucosa. The speed of the rising phase varies with intensity of stimulus. The recovery phase or falling phase is an exponential decay with a time constant of 0.9-1.45 ms.

Olfactory responses show both variations in thresholds and adaptation. The threshold of perception is lower than identification: a smell is sensed before it is recognized. Threshold values vary widely between studies and reflect the nature of smell and different methods of detection. Thresholds depend on levels of inhibitory activity, which are generated by higher centers. Some animals, particularly dogs, have much lower thresholds.

Olfactory responses show marked adaptation and thresholds increase with exposure. Recovery of the EOG is rapid when the stimulus is withdrawn. Adaptation is a peripheral and central phenomenon.

Changes in nasal mucus and its pH will alter olfactory perception. Thresholds decrease with age and are both increased and altered by hormones, particularly the sex hormones. In man, some genetic variations occur which are similar to colour blindness: a familial lack of perception to certain odours is more common in males.

Pathways:

Receptor cells are connected to the olfactory bulb by nonmyelinated nerve fibres prior to the cribriform plate. These fibres synapse on olfactory glomeruli and approximately 25,000 fibres end on each glomerulus. Conduction time between the receptor cells and the glomerulus is 50 ms. Glomeruli fire with an all or none response into mitral or tufted cells whose axons transport the signal through the lateral olfactory tract. Inhibition comes from feedback from high cortical centers.

Higher centers:

The anterior olfactory nucleus sends impulses to the opposite bulb and to the ipsilateral forebrain through the anterior commissure. The primary olfactory cortex lies rostral to the telencephalon and includes the olfactory tubercle, the prepiriform and pre-amgdaloid areas. There are projections to the thalamus where they are integrated with taste fibres, and there are projections to the hypothalamus. Communication from the receptor cell to the brain stem occurs with only two synapses.

Examination of nose

Careful history and clinical examination make diagnosis in nasal disease is often obvious. The essential symptoms are nasal obstruction, sneezing, rhinorrhoea, postnasal drip, headache and facial pains, abnormal sense of smell, epistaxis, snoring and cosmetic deformity. A previous history of trauma, allergy, smoking and occupation may also be relevant. common signs can be elicited during examination include septal deviation, hypertrophied turbinates, septal perforation and nasal polyps. There may be a combination of signs (for example, a deviated septum and nasal polyps) so be thorough with your examination.

Clinical examination:

A strong illumination needed to examine the deep narrow nasal cavity which can be provided by portable head light or bulb electric lamp at level of the patient's left ear and a concave forehead mirror to reflect light into patient's nose.

Inspection of the external nose:

Inspect the dorsum and tip for any redness, bruising, swelling, deformity, scaring, skin thickening, ulceration and the shape of the columella and nares.

Gently lift the tip of the nose with the thumb to obtain a view of the nasal vestibules and look for any skin changes, swelling, bleeding point or dilated vessels.

The patency of each nasal airway is assessed by using a metallic tongue depressor held under the nose where fogging occurs on its shiny surface during expiration.

The nasal cavity:

Anterior rhinoscopy is carried out using a Thudichum speculum that should be gently introduce this into the nose to avoid patient irritation.

Inspection of nasal mucosa for its colour, vascularity, crusting and secretions and assessment for any septal deviation, spur, perforation, size and colour of the inferior turbinate, nasal polyps or mass.

If a better view is needed the nasal mucosa can be shrunk using a local anaesthetic/vasoconstrictor (e.g. 5 or 10% cocaine hydrochloride or phenylephedrine with lignocaine).

In clinical practice a nasendoscope can be used. This will allow an inspection of the lateral nasal wall and the anatomy (and any pathology) of the middle meatus.

Oral examination:

The floor of the maxillary sinus lies over the alveolar process of the maxilla and the roots of the second premolar and first molar teeth. So inspection and percussion of upper teeth is mandatory. Movement of the soft palate should be assessed and if there is a bifid uvula, this raise the suspicion of a hidden submucous cleft.

The nasopharynx:

Examination of postnasal space can be done by small postnasal mirror which will show the posterior end of the septum, the posterior choanae, through which the posterior ends of the inferior turbinates may be visible. In the lateral wall the tubal ridges of the pharyngeal ends of the Eustachian tubes can be seen. The fossae of Rosenmüller lie immediately above the tubal orifices and can be the site of a nasopharyngeal carcinoma. Some patients have too strong a gag reflex so local anesthetics can be used to facilitate examination, if no adequate response anesthesia so flexible nasendoscope can be used to inspect the postnasal space.

The neck:

Inspect and palpate the neck and look for the presence of lymphadenopathy. The lymphatic drainage from the anterior part of the nose is to the submandibular nodes and upper deep cervical nodes. Drainage from the posterior part is to the middle deep cervical nodes.

Summary of examination of the nose:

1. Introduce yourself.
2. Position the patient.
3. Inspect the external nose.
4. Examine the nasal tip and vestibule and assess the nasal airways.
5. Anterior rhinoscopy with a Thudichum speculum.
6. Oral examination.
7. Postnasal space examination.
8. Neck.

Fracture nasal bone

EPIDEMIOLOGY AND AETIOLOGY:

Relatively little force is required to fracture the nasal bones. Isolated fractures of the nasal pyramid account for about 40 percent of all facial fractures. young men are twice as likely to sustain a fractured nose as women. The peak incidence is in the 15-30-year age group when assaults, contact sports and adventurous leisure activities are more common. these are often of a greenstick nature in children especially toddlers. Compound and comminuted fractures are more common in the elderly who are prone to falls.

Pattern of fracture:

Nasal fractures can also be subdivided into three broad categories that characterize the patterns of damage sustained with increasing force. This classification has some practical utility as each category of fracture requires a different method of treatment.

CLASS 1 FRACTURES:

the result of low-moderate degrees of force and hence the extent of deformity is usually not marked. The simplest form of a class 1 fracture is the depressed nasal bone. The nasal septum is generally not involved. In the more severe variant, both nasal bones and the septum are fractured. This fracture was first described by Chevallat and bears his name. Class 1 fractures tend not to cause gross lateral displacement of the nasal bones and may not even be perceptible

CLASS 2 FRACTURES:

Class 2 fractures are the result of greater force and are often associated with significant cosmetic deformity. In addition to fracturing the nasal bones, the frontal process of the maxilla and septal structures are also involved. The ethmoid labyrinth and adjacent orbital structures remain intact.

The pattern of deformity is determined by the direction of the force applied. A frontal impact tends to comminute the nasal bones and cause gross flattening and widening of the dorsum; while a lateral impact produces a high deviation of the nasal skeleton. This pattern of fracture was first described by Jarjavay and bears his name.

CLASS 3 FRACTURES:

Class 3 fractures are the most severe nasal injuries encountered and usually result from high velocity trauma. They are also termed naso-orbito-ethmoid fractures and often have associated fractures of the maxillae.

In severe cases, there is multiple fractures of the posterior frontal sinus wall, roof of the ethmoid and orbit that may extend posteriorly to the sphenoid and parasellar regions. Cerebrospinal fluid leaks, pneumocranum and cerebral herniation may complicate this type of injury.

CLINICAL PRESENTATION

It is important to establish duration of injury, not only for medicolegal purposes but also because there is a limited period of time during which simple reduction is possible.

Fracture nasal bone usually associated with some degree of nasal obstruction, but complete obstruction and persisting pain might indicate the presence of a septal haematoma. Enquiry of Other injuries may also be present involving orbit, palate, maxilla and base of skull that should suspected if patient complaining of watery rhinorrhoea and loss of the sense of smell.

Examination:

Inspect the nose for any external deformity. Gross edema may hide significant deviation so a second inspection a few days later may be necessary. Gently palpate the nasal bones for a step deformity. Inspect the nasal cavities and check for the presence of a septal haematoma or deviation.

INVESTIGATIONS:

1. Plain x-rays of nasal bone.
2. Computerized tomography (CT) scan should be acquired once injury of orbit or base of skull is suspected.
3. B2 transferrin assay may be done to exclude CSF leak in cases of watery rhinorrhea.

TREATMENT:

Majority of patients (80 percent) do not require any active treatment. Many do not have a nasal fracture or the fracture may not be displaced, Reassurance and analgesia are all that these patients require. Topical vasoconstrictor drops are helpful to reduce congestion and obstructive symptoms.

The indications for surgical intervention in the acute phase are significant cosmetic deformity (before onset of edema) and nasal obstruction caused by a septal haematoma.

The optimal time for clinical assessment is around four days, by which time much of the oedema will have subsided and any underlying deformity apparent. In children, healing can take place even more quickly and earlier intervention is indicated.

Reduction of a fractured nose can be performed under local or general anaesthesia. This can be achieved by firm digital pressure. Sometimes instruments are necessary to achieve satisfactory reduction, particularly in those where there has been delay in consultation.

For many class 2 fractures, closed reduction alone rarely achieves a satisfactory result. At least 50 percent of these fractures remain displaced because of overlapping segments of the fractured perpendicular plate of the ethmoid or septal cartilage, which can only be repositioned by open reduction. A splint or plaster applied to the nasal bridge maintains, to some extent, the position of the nasal bones and prevents accidental displacement. Splints are usually kept in place for about seven days.

COMPLICATIONS:

- 1.Poor cosmetic result
- 2.Nasal obstruction due to septal deviation or haematoma.
- 3, Epistaxis usually mild and self-limited, persistent bleeding may be controlled by anterior packing.

4. Septal complications: haematoma, abscess, perforations, deviation or thickening.
5. saddle nose deformity.

Septal haematoma:

submucoperichondrial collection of bleed which could be localized or diffuse involving large area of septum depriving underlying cartilage of its source of nutrition. This can lead to cartilage necrosis and ultimately nasal deformity. Septal haematomas present with intense unilateral or bilateral nasal obstruction and, on inspection, there is a reddish purple, fluctuant swelling of the caudal septum. Untreated, an abscess may develop and the patient becomes very unwell with a fluctuating fever, severe facial and cranial pain. Rarely, cavernous sinus thrombosis or other forms of intracranial sepsis can ensue. The haematoma or abscess must be drained as soon as possible. This can be performed under local or general anaesthetic either by using needle aspiration or, preferably, an incision.

Septal perforations may also develop after nasal fractures, usually as a result of septal haematomas and their surgical treatment. Loss of cartilaginous septal support can also lead to a saddle nose deformity, as well as columellar retraction and a broadened septum.

Epistaxis

Bleeding from *inside* the nose is called epistaxis. It is fairly common seen in all age groups—children, adults and older people. It often presents as an emergency. Epistaxis is a sign and not a disease per se and an attempt should always be made to find any local or constitutional cause.

BLOOD SUPPLY OF NOSE:

Nasal septum and the lateral walls have very rich blood supply from both the external and internal carotid systems.

The internal carotid artery supplies the nose by anterior and posterior ethmoidal artery which are branches of ophthalmic artery while the external carotid supplies it by branches of sphenopalatine artery (branch of maxillary artery), greater palatine artery (branch of maxillary artery), superior labial artery (branch of facial artery), nasal branch of anterior superior alveolar artery (branch of maxillary artery) and branches of facial artery to nasal vestibule.

LITTLE'S AREA

It is situated in the anterior inferior part of nasal septum, just above the vestibule and contain *Kiesselbach's plexus* which is formed by anastomoses of four arteries—anterior ethmoidal, septal branch of superior labial, septal branch of sphenopalatine and the greater palatine, it is a common site for epistaxis in children and young adults due to drying effect of inspired air and finger nail trauma.

Retrocolumellar vein

This vein runs vertically downwards just behind the columella, crosses the floor of nose and joins venous plexus on the lateral nasal wall. This is a common site of venous bleeding in young people.

WOODRUFF'S PLEXUS

It is a plexus of veins situated below posterior end of inferior turbinate. It is a site of posterior epistaxis in adults.

CAUSES OF EPISTAXIS

- A. IDIOPATHIC Many times the cause of epistaxis is not clear (85%).
- B. LOCAL CAUSES
- 1. **Trauma:** Finger nail trauma, injuries of nose and face, intranasal surgery.
- 2. **Infections:** Acute (Viral rhinitis, nasal diphtheria, acute sinusitis) and Chronic (tuberculosis, syphilis septal perforation)
- 3. **Foreign bodies:** Any neglected foreign body especially organic type, rhinolith and Living maggots and leeches.
- 4. **Neoplasms of nose, paranasal sinuses and nasopharynx:** Haemangioma, papilloma, angioma, Carcinoma or sarcoma.
- 5. **Atmospheric changes:** High altitudes, sudden decompression (Caisson disease).
- 6. **nasal septal deviation and perforation.**

C. B. GENERAL CAUSES

1. **haematological diseases:** Aplastic anaemia, leukaemia, thrombocytopenic and vascular purpura, haemophilia, Christmas disease, scurvy, vitamin K deficiency and hereditary haemorrhagic telangiectasia.
3. **Liver disease:** Hepatic cirrhosis (deficiency of factor II, VII, IX and X).
4. **Kidney disease.** Chronic nephritis.
5. **Drugs:** Excessive use of salicylates and other anticoagulant therapy.
6. **Mediastinal compression.** Tumours of mediastinum (raised venous pressure in the nose).
8. **hormonal changes:** menstruation and pregnancy.

Treatment

a. General measures:

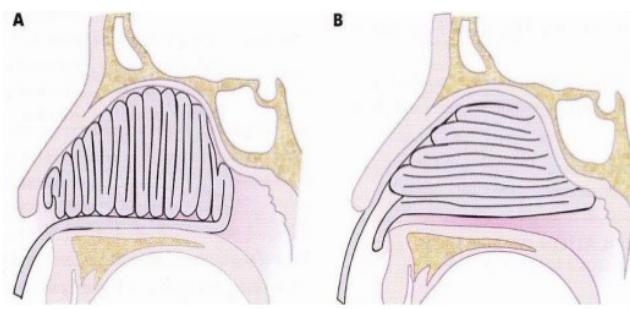
Rapid history should be taken from patient while examining the patient including enquiry about duration and side of epistaxis, number of attacks, history of trauma, infection, drug intake and any chronic disease.

Reassurance of the patient is important and mild sedation may be given. the patient should be nursed in sitting up position with a back rest. Regular checking of pulse, BP and respiration is mandatory to assess haemodynamic state of patient. Blood transfusion may be required in cases of severe bleeding and haemodynamic instability.

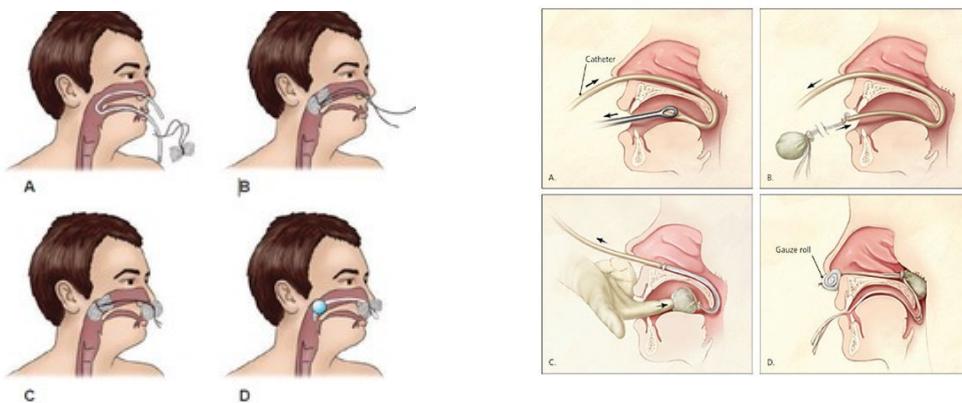
Mild epistaxis can be controlled by simple pressure over the nostrils by finger or ice cold pack for five minutes. If bleeding point detected, then thermal or chemical cautery may be used to arrest bleeding

b. Nasal packing:

In more severe bleeding and/or bleeding point cannot be identified, anterior nasal packing usually done under local anesthesia. One or both cavities may need to be packed. Pack can be removed after 24 h, if bleeding has stopped. Sometimes, it has to be kept for 2–3 days; in that case, systemic antibiotics should be given to prevent sinus infection and toxic shock syndrome. (fig)



Posteriorly situated bleeding cannot be controlled by anterior packing so posterior packing would be performed for 48 – 72 hours using a postnasal pack or Foleys' catheter that closes the postnasal space then anterior nasal packing done bilaterally.



c. ENDOSCOPIC CAUTERIZATION

Under topical or general anesthesia, a rigid endoscope can be used to localize bleeding point It is then cauterized with a malleable unipolar suction cautery or a bipolar cautery.

Elevation of mucopeichondrial flap and submucosal resection.

- d. **ligation of vessels like** External carotid artery, maxillary artery and ethmoidal arteries.
- e. **Transnasal endoscopic ligation of sphenopalatine artery**

f. EMBOLIZATION

It is done by an interventional radiologist through femoral artery catheterization. Internal maxillary artery is localized and the embolization is performed with absorbable gelfoam and/or polyvinyl alcohol or coils. Both ipsilateral or bilateral embolizations may be required for unilateral epistaxis because of cross circulation. Embolization is generally a safe procedure but may have potential risks like cerebral thromboembolism, haematoma at local site. Ethmoidal arteries cannot be embolized.

RHINOSINUSITIS

Rhinosinusitis is a group of disorders characterized by inflammation of the mucosa of the nose and paranasal sinuses.

Major symptoms	Minor symptoms
Facial pain/pressure/ congestion/fullness	Headache
Nasal obstruction/blockage	fever
Nasal discharge/ postnasal drip	Halitosis
Hyposmia/anosmia	Fatigue
Purulence on nasal examination	Dental pain
Fever (acute RS only)	Cough
	Ear pain/pressure/ fullness

- Two or more symptoms needed for diagnosis, one of which should be nasal blockage/obstruction /congestion or nasal discharge (anterior or posterior nasal drip) ± Facial pain or pressure ± Hyposmia or anosmia

Classification of Rhinosinusitis

- Acute rhinosinusitis (ARS)*: symptoms lasting for less than 4 weeks with complete resolution
- Subacute RS*: duration between 4 and 12 weeks
- Chronic RS (CRS) (with or without nasal polyps)*: symptoms lasting for more than 12 weeks without complete resolution of symptoms
- Recurrent ARS*: ≥ 4 episodes per year, each lasting ≥ 7 to 10 days with complete resolution in between episodes
- Acute exacerbation of CRS*: sudden worsening of baseline CRS with return to baseline after treatment.

Acute Rhinosinusitis

It is acute inflammation of nose and paranasal sinuses < 4 weeks duration.

1. ***Acute Viral Rhinosinusitis*** (common cold): most commonly caused by Rhinovirus and influenzae are the agents, self-limited symptoms last for less than 14 days
 2. ***Acute Bacterial Rhinosinusitis***
Haemophilus influenzae, Streptococcus pneumoniae, and Moraxella catarrhalis are the most common agents.
- Three cardinal symptoms for diagnosis.
 - A. Purulent nasal discharge.
 - B. Face pain or pressure.
 - C. Nasal obstruction.
 - Secondary symptoms that further support diagnosis. Anosmia, fever, aural fullness, cough.

Pathophysiology of ARS

- *Anatomic abnormalities may predispose one to ARS:* Septal deviation and spur, turbinate hypertrophy, prominent ethmoidal bulla; pneumatization and inversion of uncinate process.
- Acute viral respiratory infection affects nasal and sinus mucosa leading to obstruction of sinus outflow.
- *Other factors:* Allergies, nasal packing, sinonasal tumors, trauma, and dental infections.

Diagnosis:

1. Anterior rhinoscopy show red and swollen mucosa near sinus opening with purulent discharge
2. X – ray and CT scan may demonstrate air fluid level in the affected sinus.

Treatment of ARS

The effective treatment of ARS requires restoration of drainage of infected sinuses, patency of ostiomeatal complex and radication of bacterial infection to minimize risk of complications or sequelae

- A. Medical treatment of ARS:
 1. Analgesics
 2. mucolytics (saline irrigation)
 3. Nasal corticosteroids shown to be effective
 4. Decongestants should be used for less than 5 days
 5. Oral antihistamines in patients with allergic rhinitis
 6. Antibiotics:

For mild disease (Mild pain and temperature less than 38°C), antibiotics can be postponed for up to 5 days then patient is reevaluated.

For moderate to severe disease (symptoms persistent or worsening after 5 days, temperature > 38°C)

First line: amoxicillin or amoxicillin/clavulanate for 7 to 14 days; in penicillin-allergic patients: TMP/SMX, doxycycline, and macrolide. If no improvement in 72 hours, Switch to respiratory quinolones (levofloxacin, moxifloxacin), high-dose amoxicillin/clavulanate.

B. Surgical treatment of ARS:

Only limited to patients with complications of sinusitis (orbital or intracranial).

Chronic Rhinosinusitis

The signs and symptoms of chronic sinusitis are similar to acute sinusitis, except they last longer than 12 weeks and most commonly affect maxillary sinus. Fever isn't a common sign of chronic sinusitis, as it may be with acute sinusitis.

Diagnosis of CRS

- At least two of the cardinal symptoms + one of the following:
 - A. *Endoscopic evidence of mucosal inflammation:* purulent mucus or edema in middle meatus or ethmoid region
 - B. Polyps in nasal cavity or middle meatus
 - C. Radiologic evidence of mucosal inflammation and thickening.
- Three subtypes of CRS:
 - A. CRS with nasal polyps (20%-33%): Predominantly neutrophilic inflammation.
 - B. CRS without nasal polyps (60%-65%): Predominantly eosinophilic inflammation.

Factors Associated With CRS:

- *Anatomic abnormalities:* Septal deviation and spur, turbinate hypertrophy.
- Blockage of *ostiomeatal complex* by inflammation or infection can lead to obstruction of sinus drainage resulting in sinusitis.
- *Mucociliary impairment:* loss of ciliary function may result from infection, inflammation, or toxin; Kartagener syndrome may be associated with CRS.
- *Asthma:* Up to 50% of CRS patients have asthma.
- *Bacterial infection:* *Staphylococcus aureus*, coagulase-negative *Staphylococcus*, *Pseudomonas aeruginosa*, *Klebsiella pneumoniae*, *Proteus mirabilis*, *Enterobacter*, *Escherichia coli*; with chronicity, anaerobes develop *Fusobacterium*, *Peptostreptococcus*, and *Prevotella*.
- *Fungal infection:* May cause a range of diseases, from noninvasive fungus balls to invasive pathologies.
- *Allergy:* there is increased prevalence of allergic rhinitis in patients with CRS.
- *Samter's triad:* Nasal polyposis, aspirin (ASA) sensitivity, and asthma; mediated by production of proinflammatory mediators, mainly leukotrienes.

- *Granulomatous vasculitis*: Churg-Strauss syndrome: CRSwNP, asthma, peripheral eosinophilia, pulmonary infiltrates, systemic eosinophilic vasculitis, and peripheral neuropathy (p-ANCA may be positive).

Treatment of CRS

Controversial due to the spectrum of disease and underlying etiologies

A. Medical treatment:

1. Long-term oral antibiotics (> 12 weeks), usually macrolide
2. Topical nasal corticosteroids
3. Nasal saline irrigation
4. in cases of chronic rhinosinusitis with nasal polyposis systemic corticosteroids can be used for short period. (1 mg/kg initial dose and taper over 10 days).

B. Surgical treatment of CRS:

- A. Endoscopic sinus surgery is reserved for small percentage of patients with CRS who fail medical management.
- B. Patients with anatomical variants often benefit from surgery to correct the underlying abnormality, reestablishing sinus drainage.
- C. Massive polyposis rarely responds to medical treatment and surgery will relieve symptoms and establish drainage as well as allow for use of topical corticosteroids.
- D. Other indications for surgery include mucocele formation, and suspected fungal rhinosinusitis.

Complications of Rhinosinusitis

A. Ophthalmologic (Chandler's classification)

1. *Preseptal cellulitis*: inflammatory edema; no limitation of extraocular movements (EOM)
2. *Orbital cellulitis*: chemosis, impairment of EOM, proptosis, possible visual impairment.
3. *Subperiosteal abscess*: pus collection between medial periorbita and bone; chemosis, exophthalmos, EOM impaired, visual impairment worsening
4. *Orbital abscess*: pus collection in orbital tissue; complete ophthalmoplegia with severe visual impairment.
5. Superior orbital fissure syndrome (CN III, IV, V1, and VI)
6. Orbital apex syndrome (CN II, III, IV, V1, and VI)
7. Cavernous sinus thrombosis: bilateral ocular symptoms; worsening of all previous symptoms.

B. Neurologic

- *Meningitis*: severe headache, fever, seizures, altered mental status, and meningismus
- *Epidural abscess*: pus collection between dura and bone
- *Subdural abscess*: pus under dura
- *Brain abscess*: pus within brain parenchyma

C. Bony

- *Osteomyelitis*: thrombophlebitic spread via diploic veins

- *Pott's puffy tumor*: subperiosteal abscess of frontal bone including osteomyelitis to erosion of the anterior bony table.

Fungal Rhinosinusitis

- Divided into invasive and noninvasive diseases
- *Invasive*: acute invasive, chronic invasive, and chronic granulomatous
- *Noninvasive*: fungal ball, saprophytic fungal, and allergic fungal rhinosinusitis

Fungal Ball

- Usually single sinus (maxillary sinus most common)
- *Most common fungus*: *Aspergillus fumigatus*
- Immunocompetent patient
- Dense mass of fungal hyphae and secondary debris without mucosal invasion
- Pain over involved sinus
- Treatment is surgical removal

Allergic Fungal Rhinosinusitis (8%-12%)

- Five criteria of Bent and Kuhn:
 - Eosinophilic mucin (Charcot-Leyden crystals)
 - Noninvasive fungal hyphae
 - Nasal polyposis
 - Characteristic radiologic findings:
 1. *CT*: rim of hypointensity with hyperdense central material (allergic mucin)
 2. *MRI*: peripheral hyperintensity with central hypointensity on both T1 and T2
 - D. Type 1 hypersensitivity by history, skin tests, or serology
- Dematiaceous fungi (*Alternaria*, *Bipolaris*, *Curvularia*, *Cladosporium*, and *Dreschlera*)
- Typically unilateral but sometimes bilateral
- Dramatic bony expansion of paranasal sinuses
- High association with asthma

Acute Invasive Fungal Rhinosinusitis

- Also known as acute fulminant fungal rhinosinusitis
- *Symptoms*: nasal painless ulcer or eschar; periorbital or facial swelling, ophthalmoplegia
- Immunocompromised patient (diabetes mellitus [DM], HIV, chemotherapy, or transplant)
- Fungal invasion into mucosa, bone, soft tissues; angioinvasion, thrombosed vessels, necrotic tissue
- Sudden onset with rapid progression
- Organisms
 - Mucorales* (*Rhizopus*, *Rhizomucor*, *Absidia*, *Mucor*, *Cunninghamella*, *Mortierella*, *Saksenaea*, *Apophysomyces*, and *Zygomycosis*): nonseptate, 90° branching, necrotic background, serpiginous (most common in diabetic ketoacidosis patients)
 - Aspergillus*: septate, 45° branching, tissue background, and vermiciform

- *Treatment:* aggressive surgical debridement, systemic antifungals, and correct underlying immunosuppressed states
- Poor prognosis

Chronic Invasive Fungal Rhinosinusitis

- Tissue invasion by fungal elements greater than 4 weeks duration, with minimal inflammatory responses
- Immunocompetent patients
- *Species:* *Aspergillus fumigatus* common, *Mucor*, *Alternaria*, *Curvularia*, *Bipolaris*, *Candida*, or *Drechslera*
- *Treatment:* surgical debridement, systemic antifungals
- Poor prognosis

Chronic Granulomatous Fungal Rhinosinusitis

- Tissue invasion by fungal elements greater than 4 weeks duration, with mucosal inflammatory cell infiltrate
- Immunocompetent patients
- Onset gradual, symptoms caused by sinus expansion
- Multinucleated giant cell granulomas centered on eosinophilic material surrounded by fungus
- *Most common:* *Aspergillus flavus*
- Treatment is surgery for diagnosis and debridement; systemic antifungals

Nasal polyposis

Polyp derived from the Greek word (polypus)

Definition:

Nasal Polyp (NP) are masses or overgrowth bags of edematous mucosa arising mainly from the mucous membranes of the nose and paranasal sinuses that project into the nasal cavity causing a variety of signs and symptoms.

Nasal polyposis are a group of inflammatory reaction involving the mucous membrane of the nose, the paranasal sinuses and often the lower airways consisting of multiple, bilateral nasal polyps and considered as part of the spectrum of chronic rhinosinusitis.

NP most frequently originate from the ethmoid air cells and project into the nose through the middle meatus.

Epidemiology:

The prevalence rate of NP is extremely variable and increases with age, reaching a peak in those aged 50 years and older. The male: female ratio is about 2:1.

Nasal polyposis occurs with a high frequency in people with allergic airway diseases

Etiology:

NP assumed caused by allergy, non-allergic adult asthma. Chronic irritation of the mucosa like that occurring in chronic rhinitis or sinusitis. NP also found in a significant number of patients with cystic fibrosis.

NP also found in association with the following diseases:

- Chronic [rhinosinusitis](#)
- [Asthma](#)
- [Aspirin-induced asthma](#), or aspirin-exacerbated respiratory disease ([AERD](#))

- [Cystic fibrosis](#)
- [Kartagener's syndrome](#)
- Fungal rhinosinusitis

Pathology:

Nasal mucosa, mainly in the region of middle meatus becomes edematous due to collection of extracellular fluid causing polypoidal change with severe eosinophilic inflammation.

NP originate around the area of the osteomeatal complex (openings of the ethmoid sinuses and project from the middle meatus into the nasal cavity)

Macroscopically NP are sessile or pedunculated grape like masses having body and stalk, variable in size, with smooth rounded edges, pale to yellow in color, may be almost translucent project towards the nasal vestibule due to gravity and excessive sneezing.

Microscopically NP covered by respiratory epithelium (ciliated pseudostratified epithelium) but transitional and squamous epithelia are also found, especially in anterior part of the polyps due to effect of inspired dry air. No Sensory nerves are founds in the NP. There is a variable submucosa contain lots of inflammatory cells like eosinophils, mononucleated cells, macrophages and other that secret a variety of inflammatory mediators that initiates and provoke asthma or allergic like reactions.

Clinical features:

Symptoms are nasal airway obstruction, hyposmia or anosmia (due to obstruction of the olfactory groove), nasal quality of speech, headache (due to impaired ventilation and drainage in the paranasal sinuses), snoring, sneezing, frequent nasal discharge and post nasal dripping and frequent throat clearing due to associated postnasal drainage.

Spread to the lower airways can lead to laryngitis with hoarseness and bronchitis.

Signs on examination include congestion of nasal mucosa, frank watery or mucoid or pus draining from the middle meatus along with visualization of the polyp.

Endoscopy with a rigid nasal endoscope is the examination of choice in order to diagnose small polyps in the middle meatus and assessment of the extent of the disease and anatomical abnormalities associated like nasal septum deviation, hypertrophy of the turbinate's.

Investigations:

- Imaging: X-ray is of limited value in diagnosis of NP but C.T scan of the nose and paranasal sinuses is the imaging modality of choice.
- Allergy testing
- Other investigations include evaluation for cystic fibrosis in children.

Treatment:

- Medical treatments include intranasal steroids.
- Surgical treatment of choice is functional endoscopic sinus surgery (FESS).

Endoscopic sinus surgery

The endoscope has revolutionized the diagnosis and treatment of diseases of nose and paranasal sinuses. Now the first line of surgical treatment of rhinosinusitis is FESS. FESS has demonstrated success rates of 76–98%.

The philosophy of FESS is minimal (functional) surgery with mucosal preservation to achieve physiological drainage and ventilation of sinuses and healing. FESS is targeted to diseased sinuses and the normal sinuses are left alone. Aggressive removal of mucosa is avoided as it leads to postoperative healing problems.

Indications:

„ Recurrent and chronic rhinosinusitis, which do not respond to medical therapy.

„ Nasal polyps both ethmoidal and antrochoanal.

„ Foreign body.

„ Septoplasty.

„ Dacryocystorhinostomy.

„ Epistaxis especially uncontrolled posterior bleeding and ligation of sphenopalatine artery.

„ Headache and facial pains: Due to nasal septal deviation and concha bullosa.

„ Complications of rhinosinusitis such as orbital abscess.

„ Cerebrospinal fluid rhinorrhea: Traumatic and iatrogenic.

„ Fungal mycetoma: CT shows heterogeneous and microcalcifications.

„ Juvenile nasopharyngeal angiofibroma.

„ Tumors of nose and paranasal sinuses such as inverted papillomas.

„ Failed previous surgeries such as external maxillary, ethmoidal and frontal procedures.

„ Mucoceles (frontoethmoid and sphenoid)—marsupialization.

,, Encephalocele.

,, Pituitary tumors.

,, Optic nerve decompression.

,, Orbital decompression in Graves' disease.

,, Choanal atresia.

contraindications:

The contraindications include following conditions, which are better tackled by the external approaches:

,, Intracranial complications.

,, Orbital cellulitis with visual field defects.

,, Osteomyelitis.

,, Aggressive fungal infections such as mucormycosis.

Anesthesia:

,, Local anesthesia with Sedation: Endoscopic sinus surgery in adults is usually done under local anesthesia and sedation. It improves safety, as manipulations of orbital perisoteum and dura are painful. The standby anesthesiologist monitors the vital parameters such as blood pressure, pulse, respiration, temperature and oxygen saturation.

,, General anesthesia: It is preferred in pediatric patients, anxious adults, in anticipated long cases and computerassisted navigation systems.

Preparations:

Topical decongestants and anesthetics are administered in nose before the patient comes to operation theater (OT). Local injection with 1% lignocaine with 1:100,000 epinephrine is infiltrated to nasal septum and dorsum, inferior and middle turbinates (infraorbital block) canine fossa, and greater palatine foramen. A small Foley catheter No. 8 or expandable sponges in nasopharynx prevents blood pooling in oropharynx.

Position of patient:

Patient is placed in supine position. A slight reverse Trendelenburg position

with patient rotation towards surgeon helps in reducing blood loss and makes surgeon comfortable.

Techniques:

The endoscopes and microsurgical instruments provide better precision in the removal of tissue and avoid unnecessary stripping of mucosa. ESS instruments are described in chapter Instruments. There are two techniques of ESS: Messerklinger (anterior to posterior) and Wigand (posterior to anterior).

A. Messerklinger Technique: It consists of anterior-to-posterior approach..It includes following steps:

1. Removal of uncinate process and exposure of infundibulum:

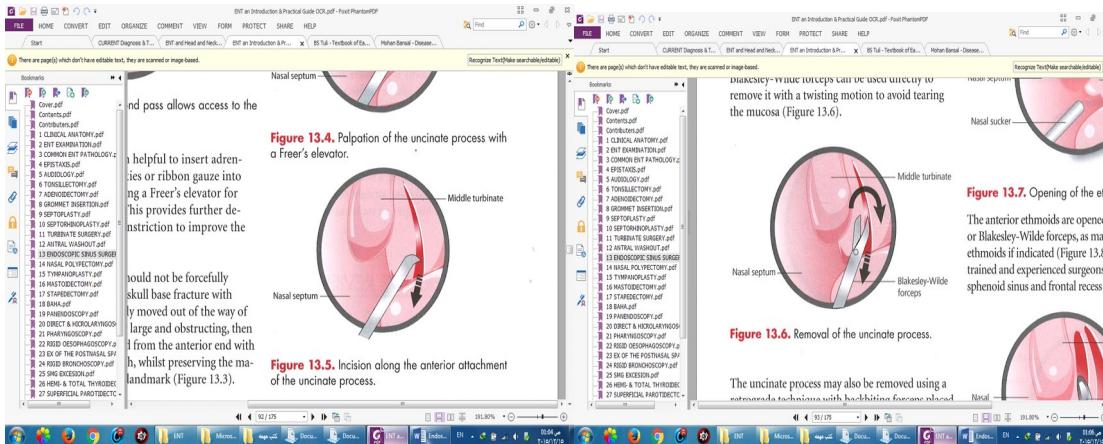
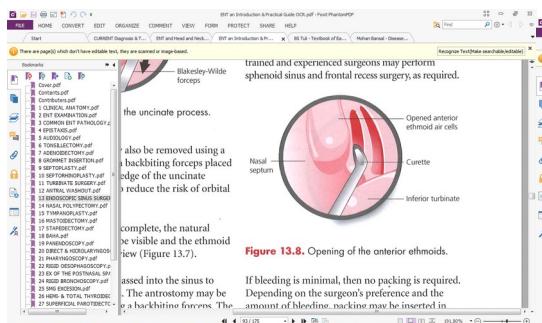
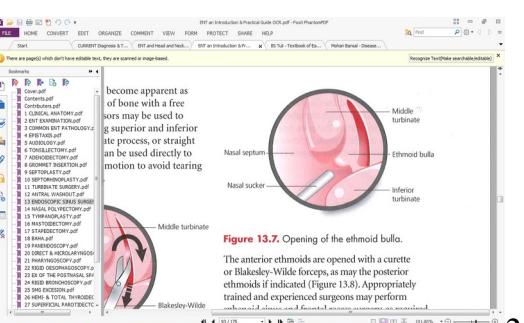


Figure:shows steps of uncinectomy.

2. Identification and widening of maxillary sinus ostium :

**Figure 13.8.** Opening of the anterior ethmoids.**Figure 13.7.** Opening of the ethmoid bulla.

3. Anterior ethmoidectomy:

Figure:shows opening of bulla and anterior ethmoid.

4.Frontal sinusotomy:

5.Identification of roof of ethmoid:

6.Posterior ethmoidectomy:

7.Sphenoid sinusotomy:

B. Wigand Technique: It involves posterior-to-anterior approach and include following steps:

1. Partial resection of middle turbinate.

2. Opening of posterior ethmoidal cells.

3. Removal of anterior wall of sphenoid sinus.

4. Identification of skull base within sphenoid sinus.

5. Removal of anterior ethmoids.

postoperative care:

,, Watch for swelling: Elevation of head and local ice to nose reduce

swelling.

„ Monitoring of visual and mental status.

„ Watch for subcutaneous emphysema: Small fracture of lamina papyracea can cause subcutaneous emphysema, which can increase due to positive pressure ventilation, coughing, vomiting, and blowing of nose.

„ antibiotics: Intraoperative as well as postoperative for 7–10 days.

„ Steroids: Reduces mucosal edema and manage allergy.

„ Analgesics relieve the pain.

„ Other agents: Allergy management, antifungal agents, and leukotriene inhibitors; and irrigations are administrated as per the need of the case.

„ Removal of nasal packing: It is removed at the time of discharge that is usually 24 hours after the surgery.

„ Topical saline and decongestants: Saline nasal spray and a short course of nasal decongestant after the removal of nasal packing.

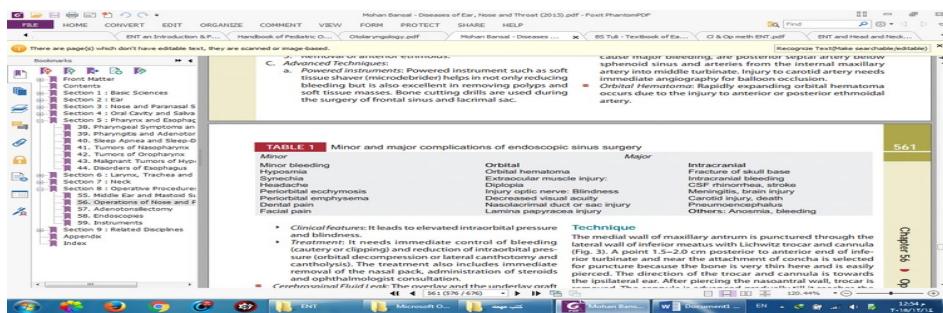
„ Removal of stenting: Plan for the stent removal if that is used.

„ Avoid strenuous activity and nose blowing and medicines that increase risk of bleeding.

„ First postoperative visit: It varies from patient to patient and is usually after 3–6 days. Some patients need frequent cleaning while others may need none. Debridement of old blood and crusts promotes healing and restores mucociliary function. Fixed clots and crusts are not removed as they cause damage to mucosa and bleeding. Middle turbinate should not get lateralized.

Complications:

Complications of ESS are usually divided into two categories: minor and major (orbital and intracranial) ,as shows below:-



The most common minor complication of FESS is the adhesions and major complications are bleeding, blindness and intracranial injury.

,, Subcutaneous Emphysema: Small fracture of lamina papyracea can cause subcutaneous emphysema, which can increase due to positive pressure ventilation, coughing, vomiting and blowing of nose.

,, Bleeding: The common arteries, which can be injured and cause major bleeding, are posterior septal artery below sphenoid sinus and arteries from the internal maxillary artery into middle turbinate. Injury to carotid artery needs immediate angiography for balloon occlusion.

,, Orbital Hematoma: Rapidly expanding orbital hematoma occurs due to the injury to anterior or posterior ethmoidal artery. It leads to elevated intraorbital pressure and blindness. It needs immediate control of bleeding (cautery or clipping) and reduction of intraorbital pressure (orbital decompression or lateral canthotomy and cantholysis). The treatment also includes immediate removal of the nasal pack, administration of steroids and ophthalmologist consultation.

,, Cerebrospinal Fluid Leak: The overlay and the underlay graft materials used in cases of cerebrospinal fluid leak (CSF) due to skull base injury include nasal mucosa, fascia (temporalis), fat, muscle, acellular dermal graft, and bone or cartilage. Fibrin glue adds support and assists healing.

Epiphora

Epiphora means watery eye which may be due to excessive secretion of tears (hyper lacrimation) or obstruction of normal tears flow from eye to nose through lacrimal system.

Hyper lacrimation: due to effects of parasympathomimetic drugs or from stimulation of sensory branches of 5th cranial nerve in the conjunctiva, lids, cornea and sclera.

In adequate drainage: blockage may be at any point of lacrimal drainage system, from puncta, canaliculi, common canaliculus, lacrimal sac and nasolacrimal duct.

The blockage may be congenital, infection, trauma or compression from surrounding structures, or due to functional causes, when pathway is patent, but the drainage system not functioning sufficiently.

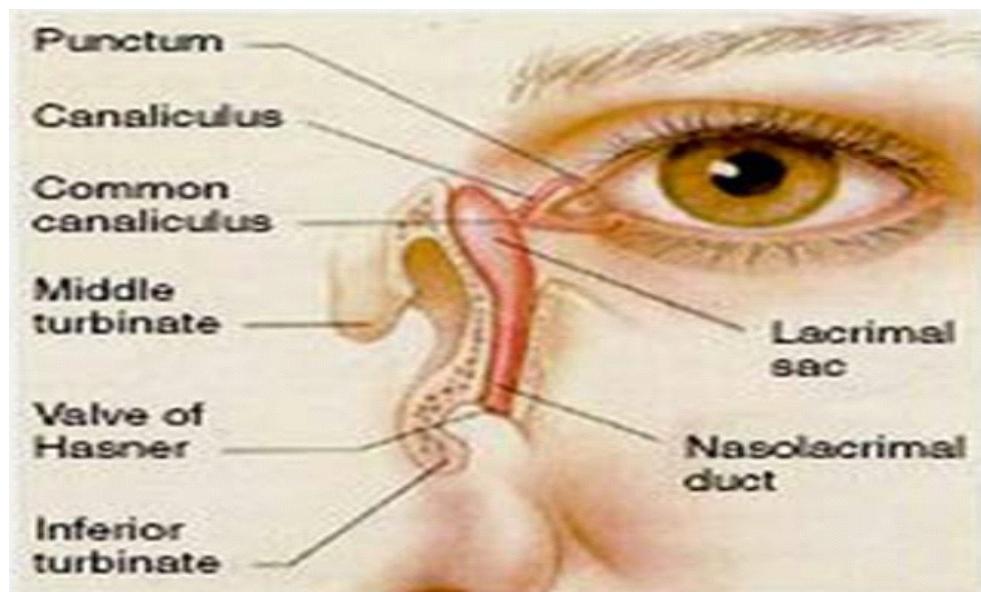
Anatomy of lacrimal system:

Secretory system:

Lacrimal glands: divided into major lacrimal glands which provide major part of tears, located at the superolateral orbital rim, within a shallow depression in the frontal bone and accessory lacrimal glands, which are structurally resemble main lacrimal gland but smaller size, they around 20- 40 in number, situated in the inferior and superior conjunctival fornices.

Excretory system:

Tears produced by lacrimal glands spread over the globe by two mechanisms the gravity and eye lids movement. The tears then collect at medial fornix. Then by capillary mechanism the tear pass through upper and lower **puncta**, then to the upper and lower **canalliculi**, common **canaliculus**, lacrimal sac, **nasolacrimal duct** and then to the nasal cavity at the inferior meatus.



Anatomy of lacrimal system

Pathophysiology:

As obstruction of lacrimal drainage system occurs, normal tears flow to the nose is impaired resulting in epiphora, stagnation of tears in the lacrimal sac leads to infection and purulent discharge through the puncta, also swelling and erythema of skin over the medial canthus.

Investigations:

- Complete ophthalmological examination to exclude ophthalmological causes like entropion, ectropion, trachiasis, blepharitis etc.
- Metal probing and irrigation of lacrimal system through upper punctum which considered therapeutic also.
- Fluresine dye test: by using this dye (yellow dye) in the eye & detecting it in the nasal cavity after two minutes.
- Dacrocystoradiography: by irrigation of the lacrimal system by radio opaque material and radiological assessment of lacrimal system.

- CT scan and MRI are helpful to detect anatomy and pathology of nose and paranasal sinuses.
- Nasal endoscopy to detect nasal pathologies like sepal deviation, turbinate hyper atrophy, rhinolith, foreign body.

Treatment

After diagnosis of acquired nasolacrimal duct obstruction has been established, the main stay of treatment is dacrocystorhinostomy DCR, which is the procedure that creates an artificial opening between lacrimal sac and nasal cavity.

The indications for DCR are:

- Epipora for more than one year, not respond to treatment.
- Recurrent acute dacryocystitis or chronic dacryocystitis.
- Chronic mucoid reflex.
- Painful distention of lacrimal sac.

The DCR is either external or endo nasal.

Types of DCR:

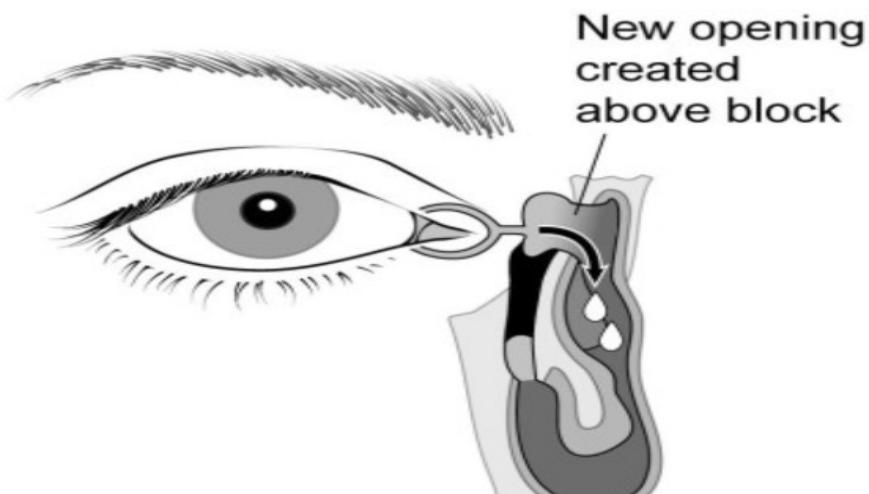
- **External (conventional) DCR:**

Indicated when the obstruction is beyond the medial opening of common canaliculus, it done under general anesthesia with success rate about 90%, the complications of this procedure include: cutaneous scarring, injury to the medial canthus structures, hemorrhage, cellulitis and CSF leak. It considered as gold standard technique.

- **Endoscopic DCR:**

For obstruction beyond medial opening of common canaliculus, it can be done under general or local anesthesia, advantages over conventional DCR are: lack of skin incision, preservation of lacrimal pump function, no CSF leak, shorter operating time, shorter recovery time, minimal blood loss.

- **Endolaser DCR:** performed with Holmium:YAG or KTP laser. It is a quick procedure can be done under local anesthesia, so it suitable for old age patients.
- **Balloon dacrocystoplasty:** involve the passage of an angioplasty balloon catheter and inflation of balloon to dilate the stenosis. It can be done under local anesthesia.



Principle of DCR

Sino-nasal tumor

Tumors of the nose and paranasal sinuses are rare and they represent an overall 3% of tumors of the head and neck region.

Classification:

- Benign tumors like osteoma, inverted papilloma and angiofibroma
- Malignant tumors like squamous cell carcinoma

Osteoma

Definition:

It is a benign, slow-growing osteoblastic lesion affecting the paranasal sinuses.

Etiology:

The most acceptable theory is that the lesions should considered slow-growing osseous hamartoma arising in childhood.

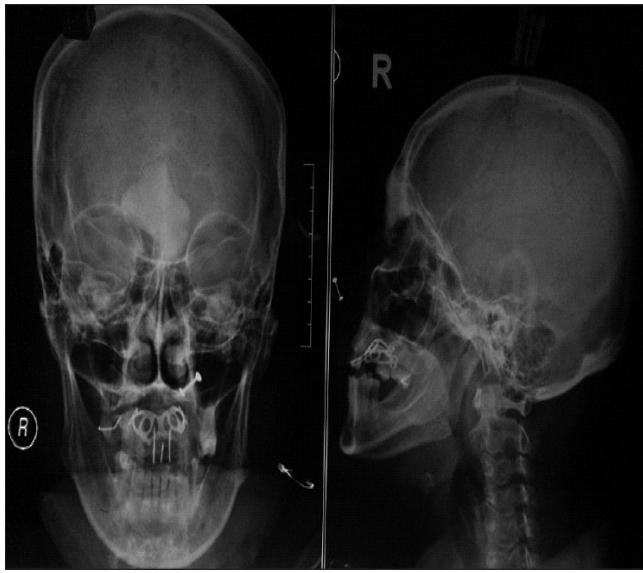
Epidemiology:

Osteoma the commonest benign tumor of the Sino-nasal region. The males slightly affected more than females.

Pathology:

The frontal sinus is the most frequently involved followed by the ethmoid, the maxillary sinus, and, more rarely, the sphenoid sinus.

Macroscopically: osteoma is hard, white, multilobulated masses.



Microscopically: osteoma of the paranasal sinuses mostly of ivory type, lobulated, made of compact, dense bone, containing a minimal amount of fibrous tissue.

Clinical features:

Osteoma usually diagnosed between
The second and fifth decades of life.
Most cases are asymptomatic and
discovered accidentally on x-ray films
of the skull.

When osteoma grows significantly, enough they obstruct drainage to or from the paranasal sinuses, leading secondarily to headaches and recurrent attacks of sinusitis.

Investigation:

C.T scan of the nose and paranasal sinuses is the imaging modality of choice.

Treatment:

Osteoma surgically removed when causing symptoms.

Inverted papilloma

Also called Schneiderian papilloma

Definition:

Benign sinonasal lesions represent an epithelial mass that arise from the mucosal surfaces of the sinonasal tract.

Etiology:

Remains unknown. Suggested causes include allergies, chronic sinusitis, airborne pollutants, and viral infection especially the human papilloma virus (HPV 6, 11, 16 and 18)

Epidemiology:

Inverted papilloma is the second most common benign tumor of the sinonasal tract after osteoma and represents the most common surgical indication for a benign sinonasal tumor. Men affected more commonly than women.

Pathology:

Inverted papilloma usually unilateral.

It is a locally aggressive tumor with the tendency for destruction of adjacent structures and tendency for transformation to squamous cell carcinoma in 2% of cases. Inverted papilloma usually arises from the lateral nasal wall near the middle meatus extending into the ethmoid and maxillary sinuses with the frontal and sphenoid sinuses are rarely involved.

Clinical features:

The lesion commonly observed in the fifth and sixth decades of life.

Symptoms: Patient usually presented with unilateral nasal obstruction with watery rhinorrhea. Epiphora, proptosis, diplopia, and headache may be associated with advanced lesions involving the orbit or the skull base.

Signs: Endoscopy of the nose shows a pale, polypoidal lesion protruding from the middle meatus.

Diagnosis:

A biopsy performed under endoscopic guidance indicated to establish the histologic diagnosis.

Investigation:

Both C.T scan and MRI are useful.

Treatment:

No role for radiotherapy as it can cause malignant transformation

Endoscopic surgical removal of the lesion

Prognosis:

Inverted papilloma is a benign lesion but adequate surgical removal is required because of the risks of malignant transformation (2%) and recurrence (8-10%).

Juvenile Angiofibroma

Also called juvenile nasopharyngeal angiofibroma (JNA)

Definition:

A benign but locally aggressive fibrous and vascular tumor that grows in the back of the nasal cavity.

Etiology:

Unknown but hormonal changes during the period of puberty play role.

Epidemiology:

JNA exceptionally affects adolescent males aging 10-25 yrs.

Pathology:

JNA is a vascular hamartoma, benign lesion originate from the area of the pterygopalatine fossa and then to the skull base. The bonny skull base involved by either resorption by pressure coming or invasion.

In early stage, JNA extends through the sphenopalatine foramen into the nasopharynx and the nasal cavity. Lateral extension leads to invasion of the infratemporal fossa, which in advanced lesions completely filled. When the lesion expands anteriorly, the posterior wall of the maxillary sinus pushed forward. Although benign, juvenile angiofibroma may extend intracranially through the orbit.

Macroscopically: JNA has a lobular surface and is grey or pinkish grey.

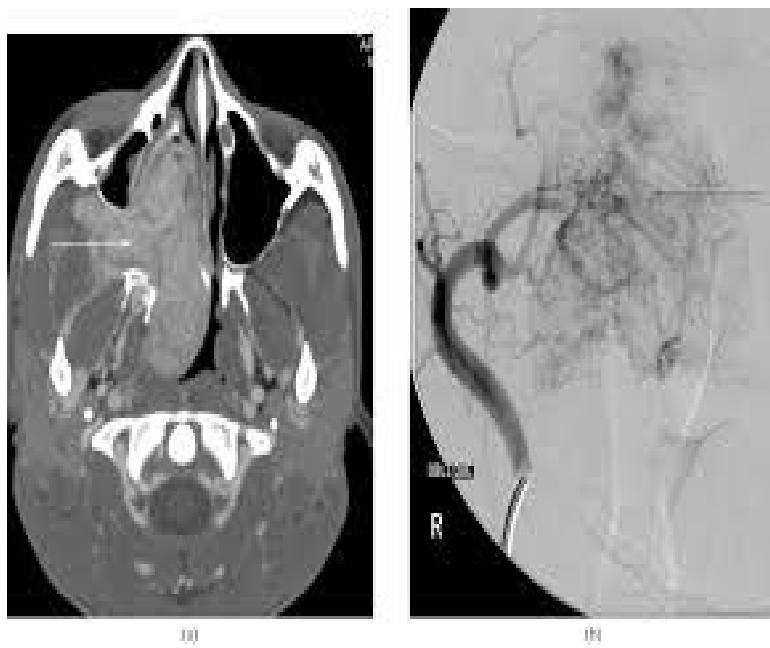
Microscopically: JNA is unencapsulated with lots of blood vessels in the stroma of the tumor with overlying epithelium is either respiratory or stratified squamous in type.

Blood supply to the tumor mainly come from internal carotid artery, the vertebral artery, and branches of the contralateral carotid system.

Clinical features:

Patients with JNA mainly are adolescent males presented with history of unilateral nasal obstruction associated with profuse epistaxis.

In advanced lesions, swelling of the cheek (involvement of the infratemporal fossa), proptosis (involvement of the orbit) or headache (involvement of the cranial fossa) may be present.



Examination by naso-endoscopy shows smooth, hyper-vascularized lesion originating behind the middle turbinate, which usually fill the posterior nasal cavity and project into the nasopharynx or through the mouth from behind.

Diagnosis:

Confirmed by the characteristic clinical picture supported with C.T scan and MRI.

No place for biopsy as it may cause a profuse and life threatening bleeding.

Treatment:

JNA surgically removed either through open approach or endoscopically after embolization of the feeding arterial supply 48 hours prior to surgery in order to minimize the intra-operative bleeding.

Prognosis:

Recurrence of JNA after surgery is common up to 40%. Radiotherapy and chemotherapy reserved for the recurrent lesions.

Malignant tumors of the nose and paranasal sinuses

Squamous cell carcinoma (SCC) of the nose and paranasal sinuses

Epidemiology:

Sinonasal malignant tumors are very rare tumors represent about 3% of tumors in the upper respiratory tract and less than 1% of the head and neck tumors. They occur most commonly in white people and the incidence in males is twice that of females. Most commonly present in the fifth and sixth decades of age.

About 55% of sinonasal tumors originate from the maxillary sinuses, 35% from the nasal cavities and 9% from the ethmoid sinuses, and the remainder from the frontal and sphenoid sinuses. is the most common malignant tumor (approximately 70-80%) followed by adenoid cystic carcinoma and adenocarcinoma (approximately 10% each).

Pathology:

Only a small percent arises at the nasal cavity and due to the proximity of the nasal cavities with the paranasal sinuses, finding the specific site of origin of large sinonasal tumors is often challenging. Their contiguity to vital structures such as the brain, optic nerves, and internal carotid artery carry significant challenges for their treatment and may be the source of significant morbidity and mortality to the patients.

Malignant tumors of the sinonasal tract derived from various histologic elements within the nasal cavity. They include the following:

- Epithelial tumors like Squamous cell carcinoma, Adenocarcinoma and Melanoma
- Nonepithelial tumors like [Rhabdomyosarcoma](#), [Chondrosarcoma](#) and [Osteosarcoma](#)

- Lymphoreticular tumors like Lymphoma, [Giant cell tumor](#).

Etiology

Exposures to environmental factors like industrial fumes, wood dust, nickel refining, and leather tanning have all been associated in the carcinogenesis of various types of sinonasal malignant tumors. There is higher incidence of nasal cancers in cigarette smokers.

Clinical presentation:

Patients with SCC of the sinonasal tract are frequently asymptomatic or commonly present with symptoms similar to those caused by inflammatory sinus disease, such as nasal obstruction, nasal discharge, [epistaxis](#), headache and facial pain, which cause a delay in diagnoses and an advanced stage of disease at the time of diagnosis.

Patients with unilateral symptoms or those that are associated with unilateral facial swelling, diplopia or blurred vision, unilateral proptosis, and cranial neuropathies numbness or hyperesthesia of the infraorbital (V2) branch of the maxillary nerve should raise a high index of suspicion for sinonasal cancer and warrant urgent evaluation.

Regional and distant metastases are uncommon even in the presence of advanced stage tumors. The incidence of cervical lymph nodes metastases on presentation less than 10%.

Distant metastasis on time of presentation is even less common. The presence of regional or distant metastases is a poor prognostic sign.

Diagnosis:

- **Naso-endoscopy:** both for visualization and biopsy taken. Nasal endoscopy may show an ulcerative lesion or polypoidal lesion in the nasal cavity.
- **Imaging:** both C.T scan and MRI are required for evaluation of Sino-nasal tumors.
- **Biopsy** for histopathological examination is mandatory for precise establishment of the diagnosis.

Staging

Staging of nasal cavity and paranasal sinus carcinomas follow the TNM classification.

- TX: Primary tumor cannot be assessed
- T0: No evidence of primary tumor
- Tis: Carcinoma in situ
- T1: Tumor limited to any one site with or without bony invasion
- T2: Tumor invading two sites in a single region or extending to involve an adjacent region within the naso-ethmoidal complex, with or without bony invasion
- T3: Tumor extends to the medial wall or floor of the orbit, maxillary sinus, palate, or cribriform plate
- T4: Tumor invades any of the following: orbital contents, skin of nose or cheek, anterior cranial fossa, dura, brain.

Treatment:

This depend on the staging of the disease.

For small curable tumors (stage 1, 2, early stage 3) a combination of surgery with pre-operative and post-operative radiotherapy and chemotherapy is adequate.

Advanced disease (late stage 3 and 4) the treatment is mainly palliative in form of chemotherapy with or without radiation.

Prognosis:

Depends on the stage, medical status and presence of local and distant metastases but overall 5 years survival is 40%.

Anatomy of the pharynx

Definition:

It is a fibromuscular tube 12 to 14 cm in length, above it is 3.5 cm and below 1.5 cm. It extends from base of skull above to the level of 6th cervical vertebra where it ends into oesophagus.

It is lined by squamous stratified type of epithelium except in naso-pharynx where the lining membrane is columnar ciliated epithelium.

Layers of Pharynx:

It has mucous membrane, submucous lymphoid tissue, pharyngeal poneurosis, muscular coat and buccopharyngeal fascia.

Muscles of the Pharynx:

Extrinsic Muscles:

1. Superior constrictor: Arises from pterygoid hamulus, pterygomandibular ligament and posterior end of myelohyoid line.

2. Middle constrictor: It is a fan-shaped muscle which arises from lesser and greater cornu of hyoid bone.

3. Inferior constrictor muscle: It has two parts:

— upper part, i.e. thyropharyngeus with oblique fibres arising from oblique line of thyroid cartilage.

— lower part, i.e. cricopharyngeus arises from lateral side of cricoid cartilage and transverse fibres form cricopharyngeal sphincter.

Constrictor muscles are supplied through pharyngeal plexus. Inferior constrictor is supplied by recurrent laryngeal nerve. Killian's dehiscence is a gap between oblique and transverse fibres of inferior constrictor .

Intrinsic Muscles:

- Stylopharyngeus.

- Salpingopharyngeus.
- Palatopharyngeus.

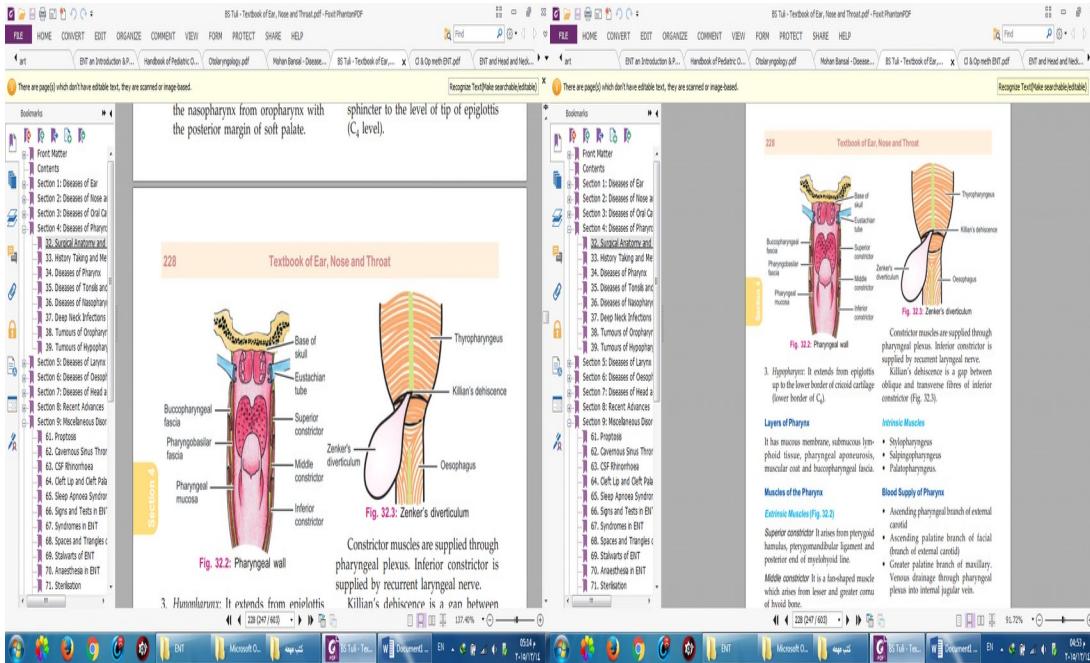


Figure: shows pharyngeal wall.

Figure: shows Killian's dehiscence.

Blood Supply of Pharynx:

- Ascending pharyngeal branch of external carotid.
- Ascending palatine branch of facial (branch of external carotid).
- Greater palatine branch of maxillary.

Venous drainage: through pharyngeal plexus into internal jugular vein.

Nerve Supply:

It is by pharyngeal plexus of nerves which is formed by:

- Branch of vagus (Xth nerve): Motor supply.
- Branches of glossopharyngeal (IXth nerve): Sensory supply.

- Sympathetic plexus.

Lymphatic Drainage:

It is into retropharyngeal and jugulodigastric nodes. Subepithelial collection of lymphoid tissue in the pharynx forms Waldeyer's ring.

It has no afferents and efferents drain into cervical lymph nodes. It consists of nasopharyngeal tonsil, tubal tonsil, faucial tonsil and lingual tonsil.

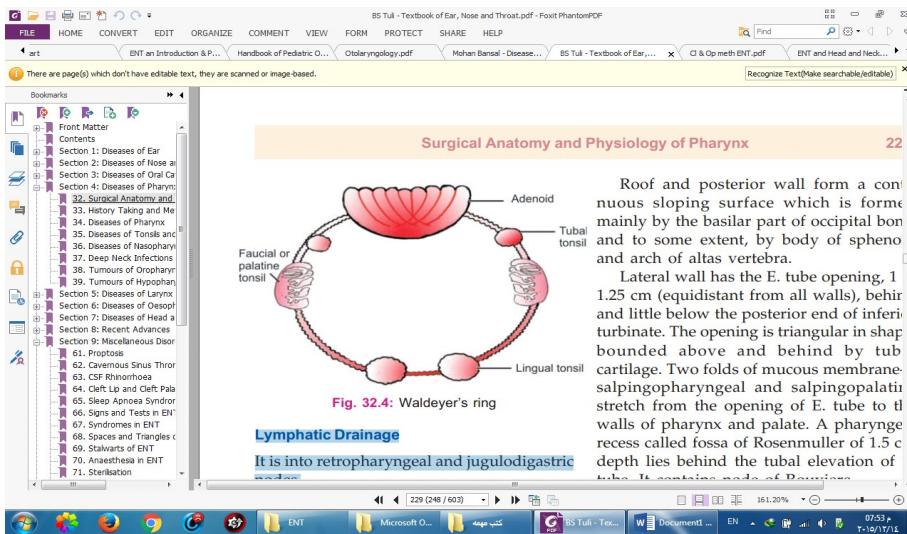


Figure: shows Waldeyer's ring.

Parts of Pharynx :

- Nasopharynx.
- Oropharynx.
- Laryngopharynx or hypopharynx.

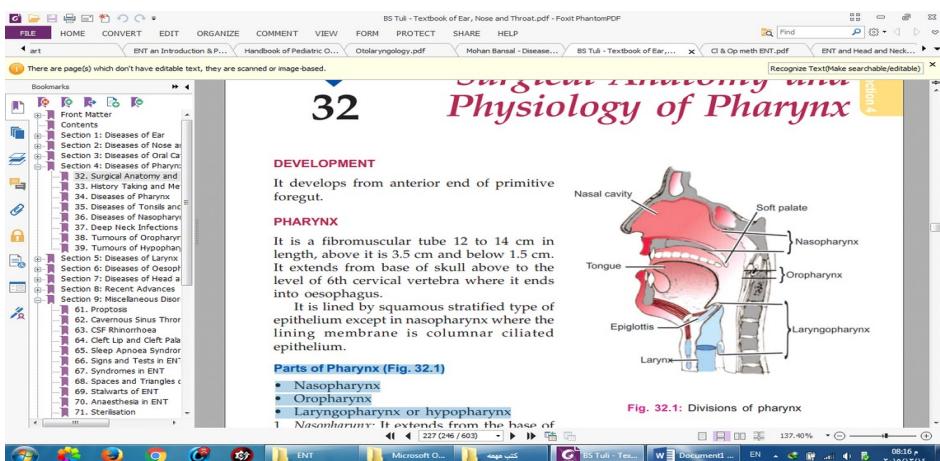


Figure: Shows parts of pharynx.

1. Nasopharynx:

It is also called third chamber of nose which lies behind the nose and above the soft palate. The space communicates anteriorly with the nose through posterior choanae and with the middle ear through eustachian tube opening in the lateral wall of nasopharynx. Dimensions of nasopharynx are:

$$4 \text{ cm (height)} \times 4 \text{ cm (width)} \times 3 \text{ cm (length)}.$$

Roof and posterior wall form a continuous sloping surface which is formed mainly by the basilar part of occipital bone; and to some extent, by body of sphenoid and arch of altas vertebra.

Lateral wall has the eustachian tube opening, 1 to 1.25 cm (equidistant from all walls), behind and little below the posterior end of inferior turbinate. The opening is triangular in shape,

bounded above and behind by tubal cartilage. Two folds of mucous membrane salpingopharyngeal and salpingopalatine stretch from the opening of eustachian tube to the walls of pharynx and palate. A pharyngeal recess called fossa of Rosenmuller of 1.5 cm depth lies behind the tubal elevation of eustachian tube. It contains node of Rouviere.

Soft palate is the anterior wall of nasopharyngeal isthmus. Posterior pharyngeal wall with Passavant's ridge forms the posterior wall. This

isthmus closes during swallowing and speech. Nasopharyngeal tonsil or adenoid mass lies in the nasopharynx. It consists of a number of folds which radiate forward and laterally from a median recess called pharyngeal bursa. It has no capsule and atrophies by 12 to 15 years of age.

2. Oropharynx:

It is the part of pharynx which lies behind the oral cavity. It extends from soft palate to the upper border of epiglottis. It opens anteriorly through nasopharyngeal isthmus into mouth. Its lateral wall presents the palatopharyngeal arch and the faecal tonsil. Posteriorly, it is supported by the body of second cervical and upper part of 3rd cervical vertebra. Valleculeae are situated between base of tongue and lingual surface of epiglottis bounded laterally by lateral glossoepiglottic fold.

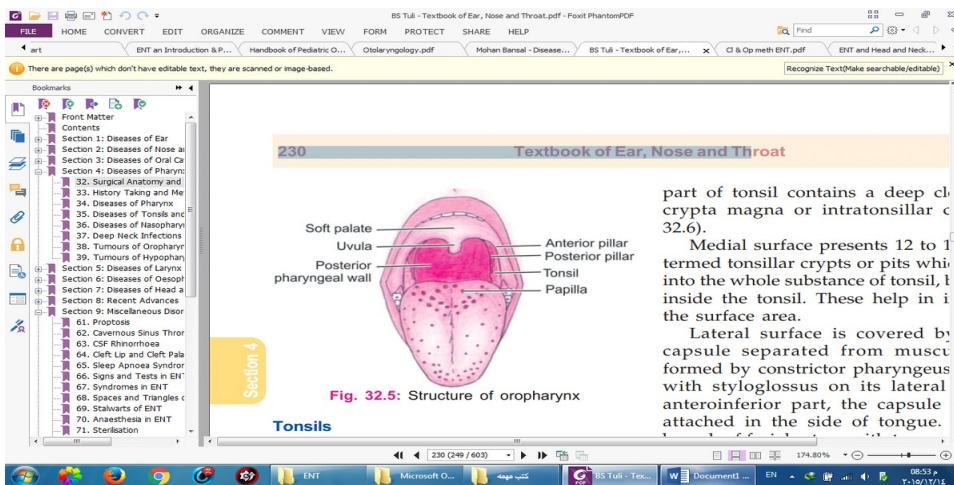


Figure: shows structures of oropharynx.

Tonsils:

These are two masses of lymphoid tissue situated in the lateral wall of oropharynx. Each tonsil is placed between palatoglossal (anterior pillar) and palatopharyngeal fold (posterior pillar). Its medial surface is free and projects into the pharynx. Inferiorly, it extends into dorsum of tongue. Superiorly, it invades the soft palate.

Plica triangularis is a free fold of mucous membrane extending from palatoglossal arch to the anteroinferior part of tonsil. Upper part of tonsil contains a deep cleft called crypta magna or intratonsillar cleft.

Medial surface presents 12 to 15 orifices termed tonsillar crypts or pits which extend into the whole substance of tonsil, branching inside the tonsil. These help in increasing the surface area.

Lateral surface is covered by fibrous capsule separated from muscular wall formed by constrictor pharyngeus superior with styloglossus on its lateral side. At anteroinferior part, the capsule is firmly attached in the side of tongue. Tonsillar branch of facial artery with two veins enters the tonsil at this point. Paratonsillar veins descend from the soft palate onto the lateral aspect of capsule of tonsil and it is this vessel which is responsible for massive bleeding, if injured during operation. Internal carotid artery lies 2.5 cm behind and lateral to the tonsil.

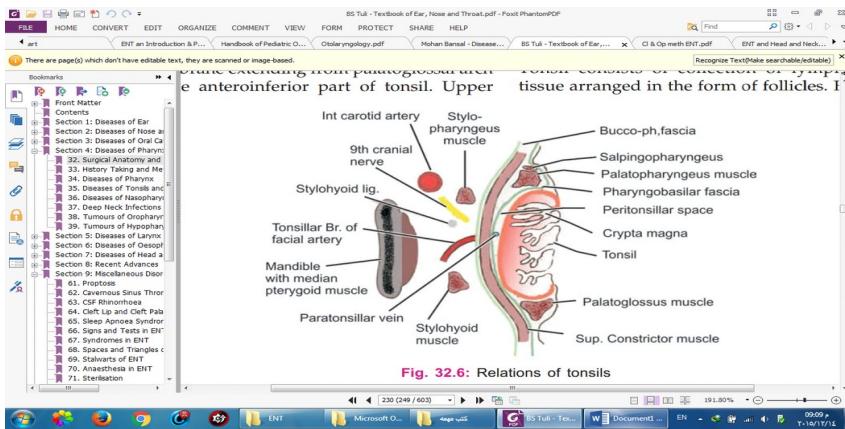


Figure: shows relations of tonsil.

Blood Supply of tonsils:

Main blood supply comes from tonsillar branch of facial artery. Branches of ascending palatine (branch of facial), ascending pharyngeal, dorsalis linguae branch of lingual and greater palatine branch of maxillary artery also supply it.

Veins One or more veins leave the lower part of deep aspect of tonsil; and after piercing the superior constrictor muscle, join the paratonsillar, pharyngeal or common facial vein.

Nerve Supply of tonsils:

It is derived from sphenopalatine ganglion through lesser palatine nerves and glossopharyngeal nerve.

Lymphatic Drainage of tonsils:

These drain into upper deep cervical, i.e. jugulodigastric lymph nodes 1.25 cm below and behind the angle of mandible.

3. Hypopharynx or laryngopharynx:

It extends from the tip of epiglottis to the cricopharyngeus from 3rd to 6th cervical vertebrae. It has three parts, pyriform sinus, posterior pharyngeal wall and cricopharynx.

Anterior wall of hypopharynx presents from above downwards— inlet of larynx, posterior surface of arytenoid cartilage and posterior aspect of cricoid cartilage.

Pyriform sinus lies on each side of laryngeal orifice. It is bounded medially by aryepiglottic fold, and laterally by thyroid cartilage and thyrohyoid membrane. In the floor of sinus lies the internal branch of superior laryngeal nerve after it has pierced the thyrohyoid membrane. Any foreign body in this site may injure the nerve. It is most richly supplied by lymphatics which come out of thyrohyoid membrane to end in upper deep cervical group of lymph nodes.

Posteriorly, the hypopharynx is supported by bodies of 3rd, 4th, 5th and 6th cervical vertebra.

Postcricoid Region:

This part of hypopharynx lies between upper and lower borders of cricoid lamina and is famous for postcricoid growth following Plummer-Vinson syndrome in females. Lymphatics drain into parapharyngeal and paratracheal group of lymph nodes.

Posterior Pharyngeal Wall:

It extends from hyoid bone to the cricoarytenoid joint. Lymphatics drain into parapharyngeal lymph nodes and finally to deep cervical lymph nodes.

Physiology Of Swallowing

Definition:

The taking in of a substance through the mouth, pharynx and oesophagus into the stomach. It is a combination of a voluntary act and a series of reflex actions. Co-ordinated by the swallowing centre in the medulla oblongata and pons.

Introduction:

Coordinated activity of H+N muscles, oral cavity , pharynx , larynx and oesophagus needed to pass food bolus and flowids from oral cavity to stomach. About 600 swallows occur every 24 hours, 150 concerned with food and drink; the rest simply clear saliva from the mouth.

Structures involved in swallowing:

- 1.Oral cavity .
- 2.Pharynx.
- 3.Oesophagus.
- 4.Neural structures within the brain.

Function of swallowing:

1. Transmission of food + drinks from oral cavity to stomach.
2. Opening of eustachian tube.
3. Continuous flow of saliva in upper GIT.

Phases of swallowing:

There are three phases of swallowing:

1.Oral phase:

It can be divided into:

A.Oral preparatory phase:

The food is processed by mastication, combined with the movement of the tongue form a bolus to an appropriate size to pass through the pharynx and oesophagus.

B.Oral propulsive phase:

The bolus is moved to the back of the tongue. Next, the anterior tongue lifts to the hard palate and retracts in a posterior direction to force the bolus to the oropharynx.

In oral phase with single swallows of liquid, the entire sequence lasts about 1 second. For swallows of solid foods, a delay of 5-10 seconds may elapse while the bolus accumulates in the oropharynx.

2.Pharyngeal phase:

The bolus is advanced from the pharynx to the oesophagus through peristalsis, The tongue pushes backward into the pharynx, the soft palate is elevated to the posterior nasopharyngeal wall, through the action of the

levator veli palatini. The palatopharyngeal folds are brought close together through the superior constrictor muscle, so that only a small bolus can pass.

The larynx and hyoid are elevated and pulled forward to the epiglottis to relax cricopharyngeous muscle. This passively shuts off its entrance and the vocal cords are pulled close together, narrowing the passageway between them. This phase is passively controlled reflexively and involves V,X,XI,XII cranial nerves. The respiratory centre of the medulla is directly inhibited by the swallowing centre for the very brief time that it takes to swallow. This is known as deglutition apnoea . This swallowing reflex lasts approximately for 1 second.

3.Esophageal phase:

The upper oesophageal sphincter relaxes to let food pass, after which various striated constrictor muscles of the pharynx as well as peristalsis and relaxation of the lower oesophageal sphincter sequentially push the bolus of food through the oesophagus into the stomach. An interval of **8-20 seconds** may be required for contractions to drive the bolus into the stomach.

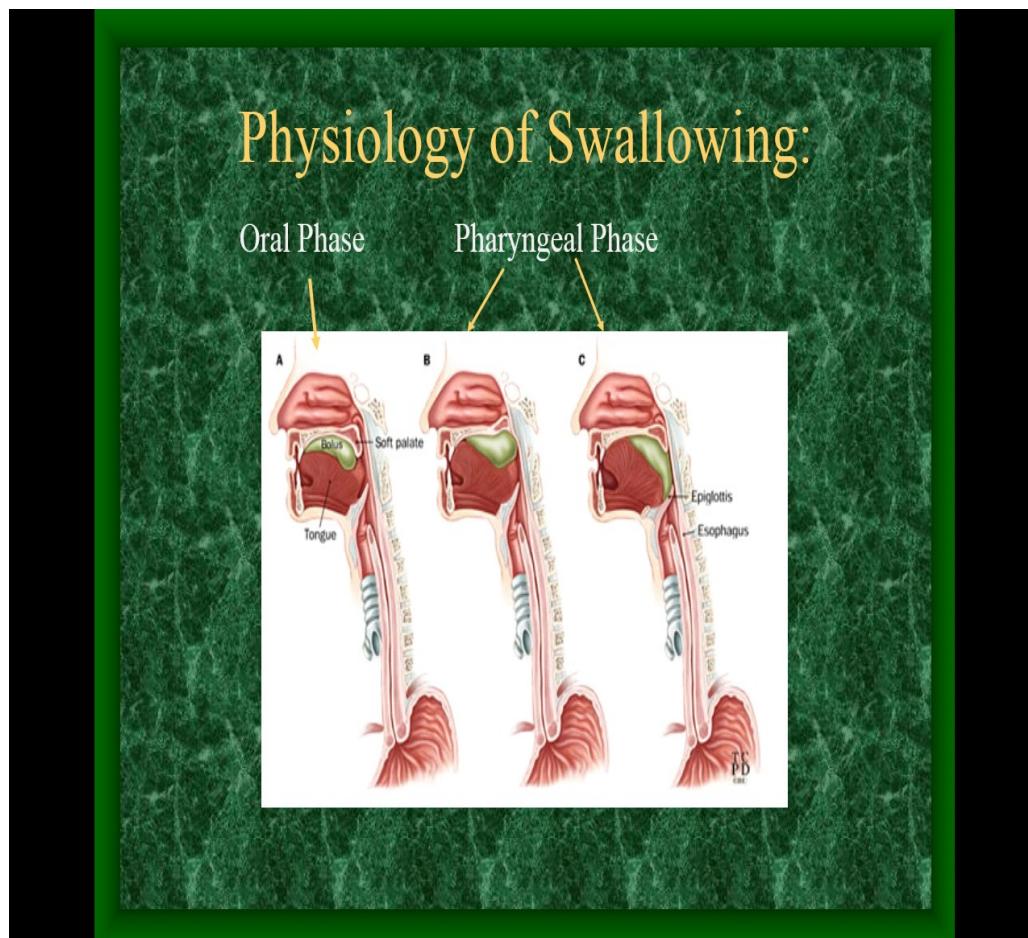


Figure:(A) Showing oral phase of swallowing.

(B) and (C) Showing pharyngeal phase.

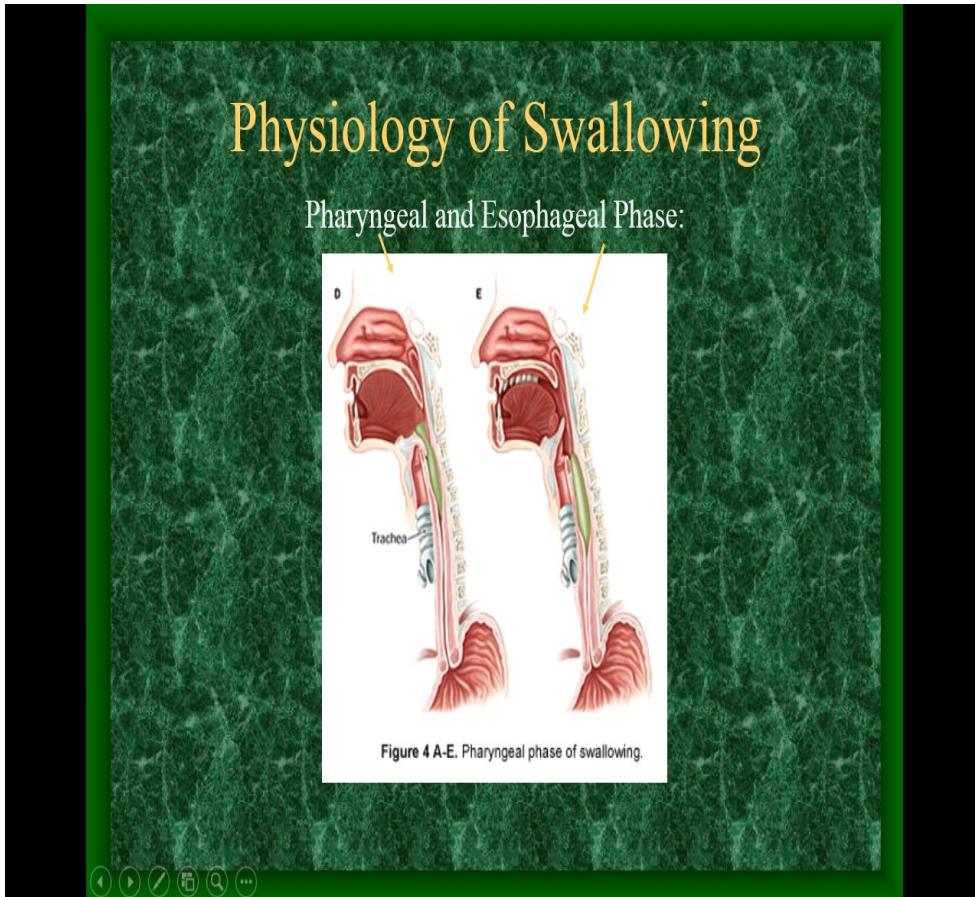


Figure: Showing (D) pharyngeal phase.

(E) Esophageal phase.

Valve action for Deglutition:

There are **six valves** that operate during swallow:

- 1. Lips (orbicularis oris muscle):** closing around a spoon, fork, cup, etc... to capture food and liquid in the oral cavity and preventing drooling.
- 2. Glosso- palatal valve:** Action is holding a bolus in the mouth during preparatory stage of swallow.

3.Velo- pharyngeal valve:It closes during the pharyngeal phase of swallow to prevent the entry of food or liquid into the nasal cavity.

4.Larynx: Closes at different levels to prevent the entry of food or liquid into the airway.

5.Crico-pharyngeal valve= Upper esophageal sphincter (UES): It mainly serves to prevent the entry of air into the esophagus during respiration, in addition it prevents acid reflux into pharynx.

Measures to protect the airway during swallowing:

- 1.Interruption of breathing.(Swallowing apnoea).
- 2.Closure of the glottis.
- 3.Tipping the larynx forward so the back of the tongue bulges over it.
- 4.Bending of the epiglottis back and down over the laryngeal opening.

Neural Control Of Swallowing:

Control of swallowing is divided between cortex and brainstem.Cortical control is bilateral with one dominant hemisphere.

Sensory input from the back of the mouth activates a set of neural circuits within the brain stem that collectively produce the pattern of motor activity constituting a swallow The relevant network of brain stem neurons also receives excitatory fibres descending from the cerebral cortex.

Pharyngitis

Defintion:

An infection or irritation of the pharynx and/or tonsils . The etiology is usually infectious with 40-60% of cases being of viral origin and 5-40% of

cases being of bacterial origin. Other causes include allergy, trauma, toxins, and neoplasia.

Classification of pharyngitis:

A. Infectious Pharyngitis:

1. Bacterial pharyngitis:

1. Streptococcal infection: Most frequent group A beta-hemolytic streptococci (GABH).

Diagnosis:

1-Enzyme immunoassay rapid antigen test or optical immunoassay rapid antigen test.

2-If negative, then many recommend culture and sensitivity, however culture still remains the safest and most cost-effective treatment strategy

Treatment:

1-penicillin.

2-erythromycin in penicillin allergic patient.

3-first-generation cephalosporins recommended for treatment failures.

Complications:

1-rheumatic fever, rheumatic heart disease.

2-acute, poststreptococcal glomerulonephritis(risk not reduced by antibiotics).

3-Grisel syndrome (subluxations of atlantoaxial joint due to infectious inflammatory process .

4-Scarlet fever: acute streptococcal pharyngotonsillitis accompanied by rash and production of erythrogenic toxin (face, palms, soles spared by rash).

2. Staphylococcal infection: mucopurulent drainage, mucosal erythema and localized pustules treated by anti-staphylococcal penicillins, or erythromycin or cephalosporins.

3. Diphtheroid Infection: caused by *Clostridium diphtheriae* (gram positive, nonfilamentous rods produces exotoxins that cause localized tissue necrosis and inflammation), rarely seen due to vaccinations.

Antitoxin remains only specific method of treatment, antibiotics useful as adjuvant therapy.

4. Pertussis: caused by *Bordetella pertussis* (nonmotile, pleomorphic, gram-negative coccobacillus), highly communicable infection causes violent paroxysms of coughing accompanied by loud inspiratory sound (whooping cough), usually self-limiting; death rarely occurs, antibiotics do not alter the course of disease. It can be eradicated by immunization.

5. Gonorrhea: caused by *Neisseria gonorrhoea* (gram-negative diplococcus must be cultured on chocolate agar), generally asymptomatic, but may present with sore throat, tonsillar hypertrophy or cervical adenopathy. Treated by penicillin, tetracycline, cephalosporins or quinolones.

6. Syphilis: caused by *Treponema pallidum*, primary stage: chancre: may affect tonsils; secondary stage: skin lesions, lymphadenopathy and pharyngotonsillitis; tertiary syphilis: CNS involvement and gammas: granulomatous nonprogressive lesions.

Investigations:

- VDRL (screening).
- FTA-ABS (confirmation).

Treatment: penicillin.

2. Viral pharyngitis:

1. Herpes Simplex Virus:

Primary infection most frequently presents as gingivostomatitis, may present as acute pharyngitis in form of vesicular lesions that bleed easily and are covered with black crust or shallow tonsillar ulcers covered with a gray exudate. Management essentially symptomatic, anti-virals best reserved for immunocompromised patient.

2. Measles:

Coryza and conjunctivitis followed by typical spotty exanthematous lesions on buccal mucosa (Koplik spots) and generalized cutaneous rash.

3. Epstein-Barr Virus (infectious mononucleosis):

lymphadenopathy and splenomegaly (50%).

Diagnosis:

- absolute lymphocytosis with atypical lymphocytes.
- mono-spot test.

Treatment: is supportive, including rest and fluids.

4.Cytomegalovirus:

Most infections are asymptomatic, except in immunocompromised patient ,may present as an infectious mononucleosis-like picture.

3.Fungal pharyngitis:

Candida albicans:

If immune system is compromised then invasion of mucosal surfaces can occur causing dysphagia, treated with topical nystatin or oral ketoconazole or fluconazole or amphotericin B for systemic involvement.

B.Inflammatory and autoimmune pharyngitis:

1.Granulomatous Pharyngitis:

If confronted with presumed infectious process unresponsive to empirical antibiotic therapy, appropriate cultures and biopsy sampling of affected tissues are necessary.it is caused by bacteria, mycobacteria, fungi, syphilis, parasites, sarcoid, Wegener granulomatosis, Crohn disease and neoplasms.

2.Stevens-Johnson Syndrome:

Onset may follow URTI or use of certain drugs, esp. sulfonamides, anticonvulsants and barbiturates. Angiitis producing erythematous vesicular skin lesion which may become bullous.

Treatment: symptomatic with maintenance of fluid and electrolyte balance during acute phase.

3.Pemphigus vulgaris:

Rapid appearance of various-sized vesicles and bullae, often involving large surface areas.

Treatment: steroids, immunosuppressive agents and antibiotics for secondary infections.

4.Cicatricial Pemphigoid:

Vesiculobullous, probably autoimmune disease,lesions typically occur on conjunctiva and upper airway mucosa.

5.Bullous Pemphigoid:

Disease of the elderly,generalized non-specific rash that persists for varying periods of time before vesiculobullous lesions appear.

6.Epidermolysis Bullosa:

Bullae or vesicles affect skin and mucous membranes with various degrees of severity, ruptures of bullae leave raw, painful surfaces that heal by scarring.

C.Iatrogenic pharyngitis:

Radiation pharyngitis:

Inevitable side effect of head and neck radiotherapy, need symptomatic and treatment of superinfections include topical antifungals or systemic antibiotics.

D.Irritative pharyngitis:

Reflux pharyngitis:

Associated with hoarseness, sore throat, chronic cough, globus pharyngeus, halitosis, cervical dysphagia, esophageal and laryngeal carcinoma. Most accurate diagnostic test is 24h pH monitoring with both proximal and distal sensors.

Treatment: empiric therapy with PPI or H2-blockers acceptable.

E.Idiopathic pharyngitis:

a-dietary and personal habits may play a role.

b-medications and commercial mouth washes can irritate throat.

c-thermal injury.

Adenotonsillar disease

Introduction:

Over the last 20 years there has been a shift in the way adenotonsillar disease in children presents to the otolaryngologist. Prior to the last two decades recurrent throat infections were the predominant reason for an adenotonsillectomy. Indications for adenotonsilectomy have evolved due to increasing recognition of the role of adenotonsillar hypertrophy in sleep disordered breathing in children.

Epidemiology of adenotonsillar disease:

Adenotonsillectomy is the most common major surgical procedures performed in children, the overall incidence rates have nearly doubled in the past 35 years. The surge in the incidence of adenotonsillectomy in very young children accompanied by a slight increase in the incidence of adenotonsillectomy in older children and young adults is due to the change in indications for the procedures. There has been a dramatic trend toward adenotonsillectomy for sleep disordered breathing rather than for infectious reasons. The incidence of sleep disordered breathing is known to peak during early childhood and to affect 1%-4% of children.⁶ In addition, older children and young adults are more frequently being treated for sleep disordered breathing with adenotonsillectomy.

At least 400,000 children undergo an adenoidectomy each year in the United States. This number, however, likely underestimates the true incidence by at least 25% since it does not include those performed at free standing ambulatory surgery centers.

Anatomy and physiology:

The palatine tonsils, adenoids (pharyngeal tonsils) and lingual tonsils, comprise a collection of lymphoid tissue named Waldeyer's ring. Waldeyer's ring is located at the cranial end of the pharynx. This collection of lymphatic tissue is thought to be involved in helping fight off pharyngeal and upper respiratory tract infections as part of the immune system. The immune function of these tissues is most active during childhood and declines with age.

The palatine tonsils lie at the entrance to the oropharynx between the palatoglossus (anterior tonsillar pillar) and palatopharyngeus (posterior tonsillar pillar). The palatine tonsils are lined by non-keratinized stratified squamous epithelium. The adenoid tissue lies at the superior aspect of the nasopharynx, extending laterally to the eustachian tubes and the walls of the pharyngeal recess. Pseudostratified ciliated columnar epithelium (respiratory epithelium) lines the surface of the adenoids.

Clinical presentation:

Adenotonsillar disease is broadly caused by either infectious disease or hypertrophic tissue leading to airway obstruction. The disease processes can affect the adenoid tissue, tonsils, or both.

Adenoid:

Infectious problems include acute and chronic adenoiditis, and otitis media. Adenoid hypertrophy has been associated with allergic rhinitis, otitis media, higher prevalence of sleep disordered breathing, and increased incidence of lower respiratory infections.

Acute adenoiditis often presents as a sudden onset of symptoms which are difficult to distinguish from those of generalized upper respiratory tract infections and rhinosinusitis. Symptoms can include nasal obstruction, rhinorrhea (purulent or clear), elevated temperatures, postnasal drainage, cough, mouth breathing, and possible concomitant otitis media. Chronic adenoiditis has a longer duration (greater than three months) of symptoms, and can result in the adenoid harboring bacteria, serving as a reservoir of infection for sinusitis.

Adenoid tissue plays an important role in the development of otitis media. The anatomical relationship of the middle ear, eustachian tube, and the adenoid tissue in the nasopharynx predisposes the child to otitis media via eustachian tube obstruction. The presentation of the classic complaints of otitis media, with or without effusion, in a child with acute or chronic nasal obstruction, rhinorrhea, mouth breathing, and postnasal drainage should prompt evaluation of the nasopharynx in addition to the middle ears.

Adenoid hypertrophy may occur in the presence or absence of infection. Hypertrophy of the adenoid tissue leads to upper airway obstruction that is more likely to be partial than complete. The characteristic complaints associated with sleep apnea may present with adenoid hypertrophy and include daytime somnolence, hypopneic breathing, snoring, arousals, and enuresis.

Complications of adenoid hypertrophy:

- 1- eustachian tube obstruction leading to serous otitis media.
- 2- interference with nasal breathing, necessitating mouth-breathing.
- 3- malocclusion.
- 4- sleep apnea/respiratory disturbance.
- 5- orofacial developmental abnormalities.

Adenoid facies:

- „ An elongated face.
- „ Retrognathic mandible.
- „ Dull expression.

- „ Dark circles under the eyes.

- „ Open mouth.

- „ Pinched nose due to disuse atrophy of alae nasi.

- „ Hitched up upper lip.

- „ Open bite, protrusive maxilla and buccal posterior crossbite.

- „ Prominent and crowded upper teeth.



Figure:shows adenoid face.

Diagnosis:

- History and clinical examination(nasal obstruction,mouth breathing,sleep apnea,adenoid facies).
- enlarged adenoids on direct/indirect nasopharyngeal exam.
- enlarged adenoid shadow on lateral soft tissue x-ray.

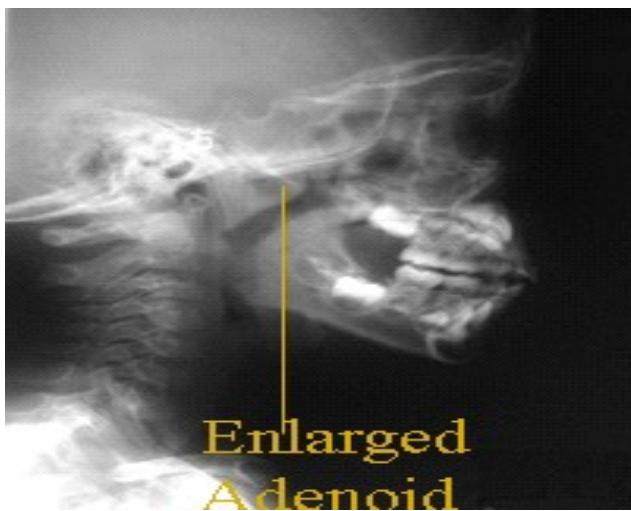


Figure:shows enlarged adenoid on x-ray of postnasal space.

Treatment:

Medical: Following measures may cure early disease without resort to surgery:

- Breathing exercises.
 - Decongested, saline or steroid nasal drops.
 - Antihistaminics.
 - Antibiotics.
- Management of any associated nasal allergy.
-

Surgical: Adenoidectomy.

Tonsils:

Infectious problems include acute tonsillitis, chronic tonsillitis, and peritonsillar abscess. Hypertrophic tonsils with or without hypertrophic adenoids can lead to sleep apnea. Asymmetric tonsils or sudden growth of the tonsils should include a differential diagnosis of malignancy.

Acute tonsillitis is one of the most common infections in children. Symptoms can include sore throat, fever, dysphagia, tender cervical adenopathy, erythema of the tonsils and pharyngeal walls, and possibly tonsillar exudate or enlargement. The duration of symptoms depends on the etiology of the infection, which can be bacterial or viral. The commonly agreed definition of recurrent acute tonsillitis is four to seven episodes in one year, five episodes in each of two consecutive years, or three episodes per year for three consecutive years.

Viral causes of tonsillitis include rhinovirus, influenza virus, parainfluenza, adenovirus and Epstein-Barr virus (EBV). EBV causes infectious mononucleosis, a distinct constellation of symptoms including enlarged tonsils covered with a gray exudate, high fevers, and malaise. Other features of infectious mononucleosis are posterior cervical lymphadenopathy, enlarged spleen, petechiae along the line between the hard and soft palate and maculopapular rash on administration of amoxicillin. Bacterial tonsillitis is most commonly caused by group A beta hemolytic

Streptococcus pneumoniae, the pathogen associated with “strep throat.” Haemophilus influenza and Moraxella catarrhalis are the next most common bacterial causes of the classic tonsillitispharyngitis. Less common causes of tonsillitis include Neisseria gonorrhoeae, and Corynebacterium diphtheria. Diphtheria can lead to edema of the airway with a sloughing exudate causing airway obstruction, requiring emergent airway management.

Tonsillar infection with Candida species, usually presents as adherent white plaques at the oral and pharyngeal mucosa, which can be scraped off to reveal a raw, bleeding mucosal base. Patients are often immunocompromised or recently treated with antibiotics.



Figure:shows kissing tonsils.

Complications:

- deep neck space infection.
- abscess: peritonsillar, intratonsillar.

- sepsis.
- glomerulonephritis, arthritis, scarlet fever, rheumatic heart disease.

Diagnosis:

History and physical examination:

Symptoms:

- sore throat.
- dysphagia, odynophagia, trismus.
- malaise, fever.
- otalgia (referred).

Signs:

- tender cervical lymphadenopathy especially submandibular and jugulodigastric.
- tonsils enlarged, inflammation ± exudates/white follicles.
- strawberry tongue, scarletiform rash (scarlet fever).
- palatal petechiae (infectious mononucleosis).

Investigation:

- CBC.
- swab for C&S.
- latex agglutination tests.
- Monospot test.

Treatment:

Medical:

- bed rest, soft diet, ample fluid intake.
- gargle with warm saline solution.
- analgesics and antipyretics.
- antibiotics (only after appropriate swab for C&S, 1st line penicillin or amoxicillin x 10 days).

Surgical: Tonsillectomy.

Adenoidectomy and tonsillectomy

Preoperative assessment:

1. Family history of coagulation disorders.
2. History of easy bruising.
3. Coagulation screening: Prothrombin time, partial thromboplastin time, bleeding time and platelet count.
4. If warranted, evaluation of coagulation studies.
5. In cases of adenotonsillar hypertrophy associated with severe airway obstruction (severe snoring, obstructive sleep apnea, cor pulmonale):
 - a. Polysomnography or sleep sonography.
 - b. X-ray chest.
 - c. ECG.
 - d. Cardiology consultation.
6. Further testing for any present systemic medical conditions such as history of bronchospasm.
7. Screening for sickle cell disease.
8. Beta-human chorionic gonadotropin to rule out pregnancy.
9. Angiography to rule out medially placed carotid artery in velocardio-facial syndrome.

Adenoidectomy:

Indications:

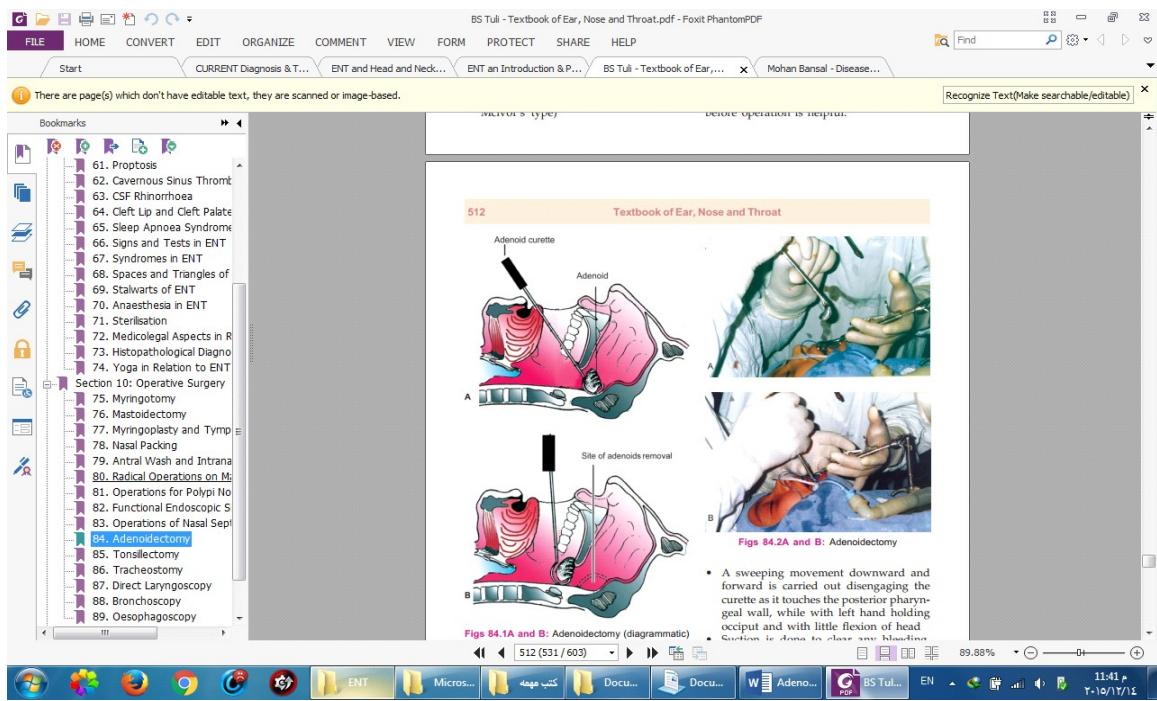
1. Adenoid hypertrophy causing:
 - Excessive snoring.

- Mouth breathing.
 - OSA syndrome or sleep disturbances.
 - Cor pulmonale.
 - Failure to thrive.
 - Enuresis.
 - Dysphagia.
 - Speech abnormalities such as rhinolalia clausa.
 - Craniofacial growth abnormalities.
2. Purulent adenoiditis.
3. Recurrent rhinosinusitis.
4. Middle ear infections:
- Chronic secretory otitis media.
 - Chronic recurrent acute otitis media.
 - Benign CSOM with recurrent ear discharge.
5. Dental malocclusion.

Contraindications:

1. Anemia: Hemoglobin less than 10 g%.
2. Acute upper respiratory tract infection or acute tonsillitis.
3. Overt or submucous cleft palate: Conservative adenoidectomy leaving the lower portion of adenoidal pad intact prevents velopharyngeal insufficiency.
4. Bleeding disorders such as leukemia, purpura, aplastic anemia and hemophilia.
5. Uncontrolled systemic disease such as diabetes, cardiac disease, hypertension or asthma.
6. During the period of menses in females.

Operative technique:



Two techniques are commonly used for adenoidectomy:

1- Adenoidal curettage:

Figure:shows adenoidectomy.

2. Suction diathermy:

This technique has recently gained popularity . Evidence suggests that suction diathermy adenoidectomy results in less intra-operative blood loss, less remnant adenoidal tissue and less postoperative nasal regurgitation of food.

Postoperative Care:

Antibiotics, hygiene of oral cavity, pain killers and normal diet.

Complications:

1. Haemorrhage.
 - a. Primary.
 - b. Reactionary—It is treated in the same way as primary haemorrhage and is usually due to incomplete removal of adenoid tissue or excessive curetting of nasopharynx.
 - c. Secondary—conservative treatment with injectable antibiotics usually arrests bleeding. Postnasal pack is rarely required.
2. Trauma: To the uvula, soft palate, eustachian tubes and subluxation of atlantoaxial joint may occur.
3. Otitis media: May occur in badly performed adenoidectomy leading to fibrosis of eustachian tube opening
4. Nasal twang: May occur if large adenoids are removed or the surgery is performed in submucous cleft palate due to velopharyngeal insufficiency.
5. Torticollis: Due to spasm of neck muscles.
6. Chronic nasopharyngitis.
7. Incomplete removal: As it is a blind procedure.

Tonsillectomy:

Indications:

Absolute:

1. Chronic or recurrent tonsillitis.

Seven episodes in 1 year, or

Five episodes per year for 2 years, or

Three episodes per year for 3 years, or

Two weeks or more of lost school or work in a year.

2. Peritonsillar abscess: In children after 4–6 weeks; in adults after second attack.
3. Tonsillitis causing febrile convulsions.
4. Cardiac valvular disease associated with recurrent streptococcal tonsillitis.
5. Hypertrophy of tonsils causing:

Excessive snoring or sleep disturbances.

Obstructive sleep apnea.

Cor pulmonale.

Dysphagia.

Interfere with speech.

6. Suspicion of malignancy—Asymmetric tonsillar hypertrophy.

Relative:

1. Diphtheria carriers, who do not respond to antibiotics.
2. Streptococcal carriers, who may be the source of infection to others.
3. Chronic tonsillitis with bad taste or halitosis.
4. Recurrent streptococcal tonsillitis in a patient with valvular heart disease.
5. Tender cervical adenitis.
6. Diffulty in eating.
7. Tonsillolithiasis.
8. Orofacial and dental abnormalities.
9. Failure to thrive.
10. Enuresis.
11. Obstructive tonsils in infectious mononucleosis not responding to medical therapy.

Contraindications:

1. Anemia: Hemoglobin less than 10 g%.

2. Acute upper respiratory tract infection or acute tonsillitis.
3. Bleeding disorders such as leukemia, purpura, aplastic anemia, hemophilia.
4. Epidemic of polio.
5. Uncontrolled systemic disease such as diabetes, cardiac disease, hypertension or asthma.
6. During the period of menses in females.

Surgical Methods:

- a) Guillotine method.
- b) Dissection and snare method (with sharp instrumentation).
- c) Electrocautery (Bovie and bipolar).
- d) Laser.
- e) Cryosurgery.
- f) Coblation.

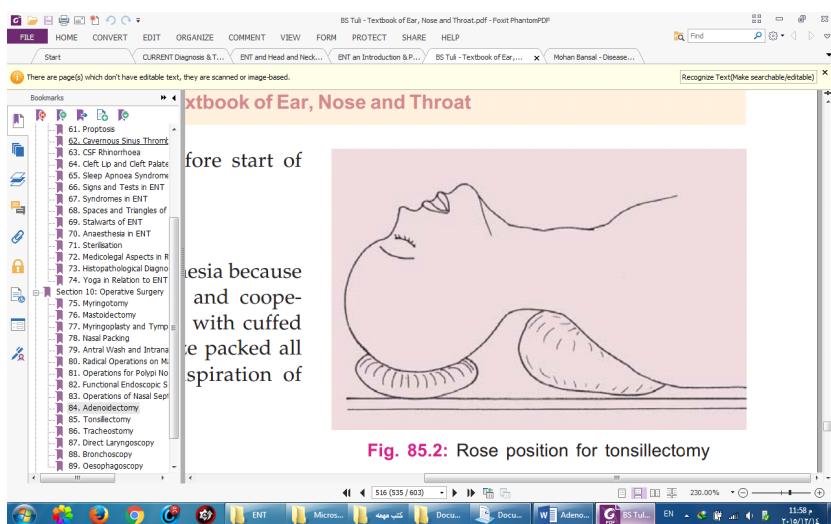


Figure: shows Rose position for tonsillectomy.

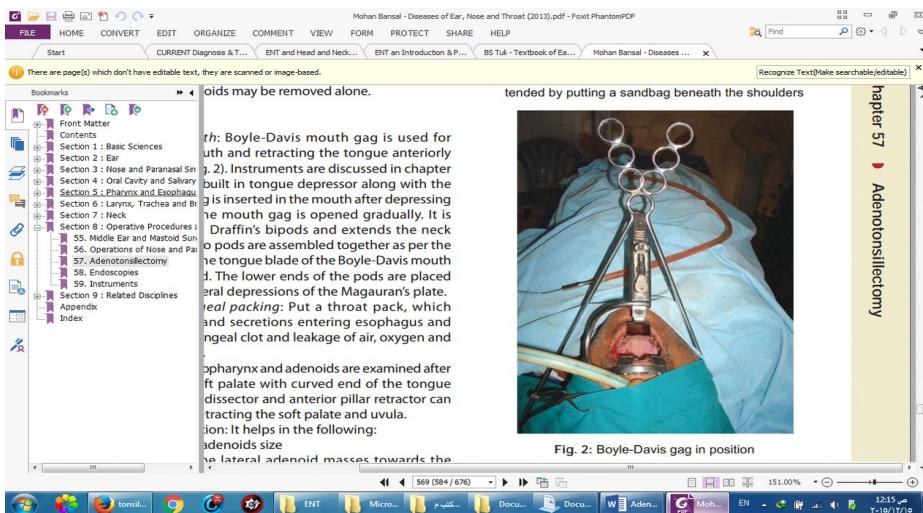


Figure: shows Boyle-Davis mouth gag in position.

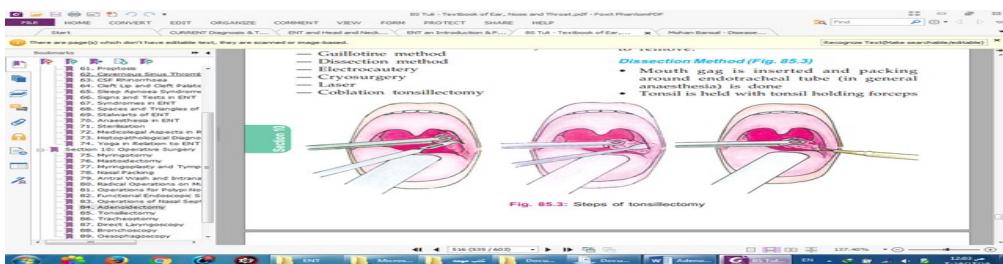


Figure: shows steps of tonsillectomy.

Postoperative care:

- Diet: Plenty of cold fluids such as cold milk or ice cream. Sucking of ice cubes helps in relieving pain. The child may take soft diet on the second day such as custard, jelly, boiled eggs, porridge or slice of bread soaked in milk. Solid foods subsequently are allowed as tolerated.
- Oral hygiene: Saline gargles 3–4 times a day. Mouth should be washed with plain water after every feed.
- Analgesics: Paracetamol with or without codeine.

d) Antibiotics: For a week.

e) Instruction: Report immediately if there is any bright red colored bleeding from nose or mouth.

Complications:

Immediate:

a) Primary hemorrhage: Bleeding during the operation is usually controlled by pressure, ligation or electrocoagulation. Coagulopathy must be ruled out. Residual remnants should be completely removed.

b) Reactionary hemorrhage: Bleeding after the recovery from anesthesia on the day of surgery is usually controlled by removing the clot, applying pressure or vasoconstrictor.

In cases of refractory bleeding, patient is taken back to operation room and ligation or electrocoagulation of the bleeding vessels is done under general anesthesia.

c) Injury: Oral cavity and oropharyngeal structures such as tonsillar pillars, uvula, soft palate, tongue, superior constrictor muscle, or teeth can be injured during tonsillectomy.

d) Aspiration and foreign bodies: Such as blood, tissue of tonsil or adenoids or tooth.

e) Pulmonary edema: It can occur in cases of OSA and cor pulmonale. Edema of tongue and palate: Need replacement of nasal trumpet and intravenous steroids.

f) Edema: Face or eyelids.

g) Surgical emphysema: Due to superior constrictor muscle injury.

Delayed:

a) Secondary hemorrhage: Bleeding seen between 5th-10th postoperative days is the result of sepsis and premature separation of the membrane. Systemic antibiotics control the infection. If bleeding is not controlled after removal of clot and topical application of dilute adrenaline, hydrogen

peroxide and with pressure, then patient is taken to operation room. Under general anesthesia, bleeding vessel is electrocoagulated or ligated.

- b) Infection: Infection may cause parapharyngeal abscess or otitis media.
- c) Pulmonary complications: Aspiration of blood, mucus or tissue fragments may lead to atelectasis or lung abscess.
- d) Scarring: Soft palate and pillars.
- e) Tonsillar remnants: The remaining tonsil tags or tissue may cause repeated infection.
- f) Hypertrophy of lingual tonsil: It is compensatory to loss of palatine tonsils.
- g) Velopharyngeal insufficiency: Hypernasality is a complication of adenoidectomy. It is usually managed by speech pathologist. But refractory cases may need reconstructive surgery (pharyngeal flap, sphincteroplasty or posterior pharyngeal wall augmentation).
- i) Nasopharyngeal stenosis: It occurs due to scarring after excessive damage to nasopharyngeal mucosa (roof, posterior and lateral walls) and resection of posterior tonsillar pillar. These children are difficult to manage.
- j) Recurrence: Remaining adenoids and tonsil tissue may grow again. If plica triangularis near the lower pole of tonsil is not removed along with tonsil, it may get hypertrophied.
- h) Atlantoaxial subluxation (Grisel's syndrome): It leads to stiff neck and spasm of sternocleidomastoid and deep cervical muscles. Treatment includes intravenous antibiotics and cervical traction.

Anatomy of larynx

Embryology & development

Formation of larynx begins during the fourth week of embryological development. Larynx develops from the tracheobronchial groove, a midline diverticulum of foregut. The laryngeal cartilages develop from fourth and sixth branchial arches.

The cricothyroid muscle is derived from the fourth arch and supplied by superior laryngeal nerve. All other intrinsic muscles are derived from sixth arch and supplied by the recurrent laryngeal nerve.

The level of larynx is high at birth and the descent continues through the life.

Laryngeal cartilages

They are nine in number 3 paired (arytenoid, corniculate, epiglottis) and 3 un paired (thyroid, cricoid, cuniform)

- **Thyroid cartilage:** the largest of laryngeal skeleton, it is V shaped open posteriorly and provides protection to the larynx, it composed

of two lamina which meet anteriorly to form the protuberance known as Adam's apple. Posteriorly each lamina has superior cornu extending upward and inferior cornu extending downward.

Angle of fusion of lamina male: 90 degree

Female: 120 degree

- **Cricoid cartilage:** the strongest of the laryngeal skeleton located directly below the thyroid cartilage, it is articulates with the inferior cornu of thyroid cartilage. It shaped like signet ring.
- **Epiglottis:** leaf shaped fibro elastic cartilage attached to the inside of the thyroid cartilage and extends up and backward above the laryngeal opening.
- **Arytenoid cartilage:** they are the chief moving part of the larynx, they are pearl shaped small cartilages articulate with cricoid lamina. The anterior projection of each cartilage is known as vocal process and gives attachment to the thyroarytenoid muscle. The lateral prominence is known as muscular process and give attachment to the posterior cricoarytenoid m. the inter arytenoid muscle connects the medial surfaces of these cartilages.
- **Sesamoid cartilages:** the corniculate cartilages (Santorini) in the aryepiglottic folds, and the cuneiform cartilages (Wisberg) .
- **Hyoid bone:** it is U shaped bone that suspended from the mandible by ligaments, it provides the stability to the larynx and pharynx and gives attachment to the muscles of the neck.

Ligaments & membranes of the larynx

- **Thyrohyoid membrane:** connects the thyroid cartilage to the hyoid bone. Its median and lateral parts thickened to form

median & lateral thyroarytenoid ligament respectively. It pierced on each side by superior laryngeal vessel and internal laryngeal nerve. It is relatively avascular and can be pierced by emergency tracheostomy.

- **Quadrangular membrane:** extends from lateral border of epiglottis to the arytenoid cartilage and inferiorly attached to the false vocal cords.
- **Conus elasticus** (triangular membrane) attached to the cricoid cartilage inferiorly and vocal process of arytenoid cartilage superioposteriorly.
- **Vocal ligament:** the free upper edge of conus elasticus.

Interior of the larynx

The inlet of the larynx consist of free edge of epiglottis anteriorly, aryepiglottic folds laterally and mucous membrane over inter arytenoid muscle posteriorly. The cavity of the larynx extends from the inlet of the larynx to the lower border of cricoid cartilage. Within the cavity of the larynx there are two folds of mucous membrane on each side. The upper fold is called vestibular folds or (false vocal cords) and the lower fold is called the true vocal folds. The space between the right & left vestibular folds is called as **Rima vestibule** and the space between vocal folds is **Rima glottides** which is the narrowest part of larynx.

The vestibular and vocal folds divide the laryngeal cavity into:

- The part above vestibular fold called vestibule of the larynx
- The part between vestibular and vocal folds is called sinus of Morgagni or ventricle of the larynx.
- The part below the vocal folds is subglottic part.

There is an extension in the anterior part of the laryngeal ventricle called the saccule of the larynx. Numerous minor salivary glands open into the surface of overlying mucosa for lubrication of the vocal cords.

Compartment of larynx

- **Supraglottis:** from the tip of the epiglottis to the true vocal cords
- **Glottis:** the true vocal cords and posterior commissure.
- **Subglottis:** from the inferior edge of true vocal cords to the inferior edge of cricoid cartilage.

Spaces of the neck:

- **Paraglottic space:** bounded by thyroid cartilage lamina, conus elasticus & quadrangular membrane.
- **Preepiglottic space:** bounded by thyroid cartilage, thyrohyoid membrane and epiglottis.

The vocal cords:

Two like folds structures extend from middle of the angle of the thyroid cartilage anteriorly to the vocal process of the arytenoid cartilage posteriorly.

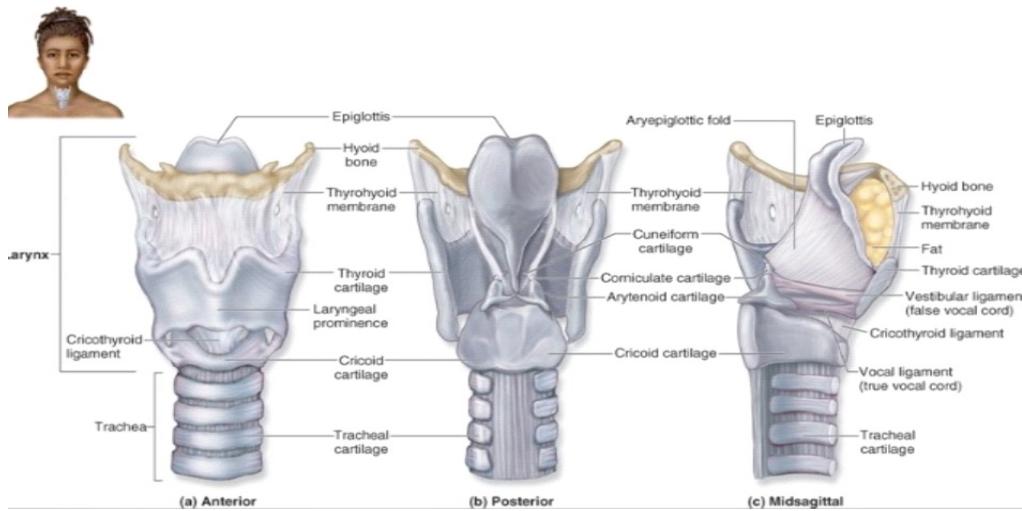
Layers of the vocal cords:

Mucosa: stratified squamous epithelium.

Lamina properia (vocal ligament) consist of three layers:

- Superficial layer: or Reinke's space (loose fibrous tissue with soft gelatin)
- Middle layer: more dense with elastic fibers.
- Deep layer: collagen fibers

Note: mucous glands are absent over vocal cords, they are lubricated by mucous glands present in the laryngeal saccule.



Muscles of the Larynx

Extrinsic Muscles:

- Muscles that elevate the larynx: (thyrohyoid, stylohyoid, digastric, geniohyoid, mylohyoid, and stylopharyngeus). These muscles suspend larynx, and are important in the elevation and anterior displacement of the larynx during swallowing.
- Depressors of the larynx: (omohyoid, sternothyroid, and sternohyoid). These muscles displace the larynx downward during inspiration.
- Constrictors of pharynx: help to organize laryngeal reflexes.

Intrinsic Muscles:

- With the exception of the inter arytenoid, the intrinsic muscles are paired and these paired muscles appear act synchronously.

- All are supplied by recurrent laryngeal nerve via inferior laryngeal nerve, except Cricothyroid which is supplied by external branch of superior laryngeal nerve.
- Cricothyroid is the only abductor of larynx.

Action	Insertion	Origin	Muscle
brings anterior arch of the cricoid superiorly while displacing posterior cricoid lamina inferiorly. This tense, vocal folds	Pars recta: inferior border of the thyroid cartilage Pars oblique: to inferior horn of thyroid cartilage	arch of the cricoid cartilage with 2 bellies: Straight (Pars recta) Oblique (Pars oblique)	Cricothyroid
abducts the vocal folds	muscular process of the arytenoid cartilage	posterior surface of the lamina of the cricoid cartilage	Posterior cricoarytenoid
adducting the vocal folds	<ul style="list-style-type: none"> • posterior surface and apex of the contralateral arytenoid cartilage • Some fibers pass along Quad. membrane forming (Aryeiglottic muscle) 	posterior surface of the arytenoid cartilage	Inter-arytenoid <ul style="list-style-type: none"> • Transverse • Oblique
adducts the vocal folds	muscular process of the arytenoid cartilage	Superior arch of the cricoid cartilage	Lateral cricoarytenoid
Shortening (relaxing) and adducting the vocal folds	<ul style="list-style-type: none"> • lateral surface of arytenoids • externus sends fibers onto Quad.membrane forming (Thyroepiglottic muscle) 	Anterior commissure	Thyroarytenoid <ul style="list-style-type: none"> • External • Internal(Vocalis)

Nerve supply of the larynx:

The larynx is supplied by two branches of vagus nerve

- **superior laryngeal nerve:** arise from inferior ganglion of vagus and divides at level of greater cornu of hyoid into:

Internal laryngeal N: sensory (above the level of vocal cords)

secretomotor

External laryngeal N: supplies cricothyroid muscles.

- **Recurrent laryngeal nerve:** supplies motor innervation to all intrinsic muscles of larynx except cricothyroid. Also supply sensory innervation below the level of the vocal cords.

Blood supply of larynx:

Arterial

- Superior laryngeal artery: arise from superior thyroid artery. A branch of external carotid artery.
- Inferior laryngeal artery: from inferior thyroid artery, a branch from thyrocervical trunk.

Venous

- Superior laryngeal vein: drains into superior thyroid vein and then into the internal jugular vein.

- Inferior laryngeal vein: drain into inferior thyroid vein and then into innominate vein.

Lymphatics of the Larynx:

- Supraglottic structures follows the superior laryngeal and superior thyroid vessels. Thus, the lymphatics flow from the piriform sinus through the thyrohyoid membrane to end primarily in the deep jugular chain, the epiglottis is a midline structure; thus, its lymphatic drainage is bilateral.
- True vocal folds are devoid of lymphatics, accounting for the high curability of cancer localized to this structure.
- Subglottic larynx has 2 lymphatic drainage systems.
 - One system follows the inferior thyroid vessels to end in the lower portion of the deep jugular chain as well as the subclavian, paratracheal chains.
 - The other system pierces the cricothyroid membrane, disseminate bilaterally to the middle deep cervical nodes as well as the prelaryngeal (Delphian) nodes.

Physiology of larynx

The main function of the larynx is the protection of the air way, phonation & speech are secondary functions.

- **Protective function:** by glottis closure reflex, cough reflex & swallowing reflex.

Glottic closure: this reflex protect the airway from inhalation & aspiration, whoever when exaggerated this will lead to laryngospasm.

Levels of protection:

- Epiglottis: moves down & back to divert bolus away from midline.
- Aryepiglottic folds: contract to constrict the middle laryngeal inlet.
- False vocal cords: tight closure.
- True vocal cords: tight closure.

Laryngospasm: is aggressive response of vocal cords (adduction) after stimulus has been removed occurs mostly in endotracheal intubation or manipulation of the air way.

Cough:

Cough ejects mucus and foreign matter from the lungs and helps to maintain patency of the pulmonary alveoli.

Cough may be voluntary but more often involuntary in response to stimulation of receptors in the larynx or lower respiratory tract.

Phases:

- **Inspiratory phase:** maximal laryngeal opening to permit rapid and deep inspiration. If the cough is voluntary, the degree of glottal abduction and inspiratory effort is proportional to the intended strength of the cough.
- **Compressive phase:** tight closure of the glottis and strong activation of expiratory muscles, thus extremely raising intrathoracic and subglottic pressure.
- **Expulsive phase:** glottis suddenly opens widely, with a sudden outflow of air.

Reflex swallowing:

Although not classically considered part of the protective reflex, it may have protective functions.

- **Respiratory Function:**

Maintains synchronous opening of glottis and descent of diaphragm during inspiration.

Role of Crico-thyroid and cricoarytenoid muscles:

Glottic opening is primarily through the action of the posterior cricoarytenoid. However, in hyperpneic conditions, the cricothyroid contracts rhythmically with the posterior cricoarytenoid. The effect is to lengthen the open glottis, thus increasing the cross-sectional area for airflow (maximal glottic opening).

- Rhythmicity of the phrenic nerve and the posterior cricoarytenoid can be increased by hypercapnia and ventilatory obstruction. It is lessened by hypocapnia.
- The longer abductor activity of posterior cricoarytenoid is lost, the more difficult it is to re-establish. This is the rationale for down-sizing tracheotomy tubes (thus gradually increasing ventilatory resistance) prior to de cannulation.

- **Phonation:**

Vocal cords vibrate passively powered by exhaled air. The requirements for normal phonation are normal vocal cords, adequate expiratory force, normal layers of lamina propria. The true vocal cords are adducted & tensed & subglottic pressure increase and induce vocal cords vibration, & opening of glottis from an inferior to superior (**myoelastic_aerodynamic theory**). Air flow through glottis creates a negative pressure which pull vocal cords back together (**Bernouli's effect**).

Normal vocal cords vibration occurs vertically from inferior to superior.

Components of speech:

Phonation: production of voice, determined by vocal cords position, expiratory force, vibratory capacity of vocal cords & vocal cords length & tension.

Resonance: oral & nasal speech balance determined by velopharyngeal musculature & by structure of chest , nasopharynx, nasal cavity & oral cavity.

Articulation: production of voice sounds , determined by action of lips ,tongue & jaw musculature action.

Voice parameters:

- **Pitch:** related to the frequency of vocal cords vibration, determined by length, tension & speed of movement of vocal cords.
- **Loudness:** or intensity of voice, determined by subglottic pressure, glottis resistance, air flow rate, vocal cords vibration &force of vocal cords contact.
- **Quality (timbre):** determined by synchronicity of vocal fold vibration & glottis competence.
- **Circulatory Reflexes:**

Stimulation of the larynx can produce changes in heart rate and blood pressure.

This effect is most noticeable during:

- Endotracheal intubation.
- Obstructive sleep apnea (OSA). When upper airway patency is not maintained during sleep, the resulting increase in negative airway

pressure can stimulate receptors in the larynx so strongly that cardiac arrhythmias & hypertension.

The afferent limb is the superior laryngeal nerve. The efferent limb for arrhythmia is vagus nerve, but the efferent limb for blood pressure elevation is not known.

- **Valsalva Maneuver:**

Refers to forced expiration against a tightly closed glottis.

It is important in defecation because the pressure is transmitted to the abdominal cavity.

Valsalva also serves to stabilize the thorax during heavy lifting by the arms.

Laryngitis

Acute & chronic inflammation of the larynx

Acute viral laryngitis

Pathogen: rhinovirus (most common), influenza, parainfluenza, respiratory syncytial virus, adenovirus.

Signs & symptoms: hoarseness of voice, low grade fever, cough & rhinitis.

Diagnosis: history & clinical examination.

Treatment: symptomatic treatment (hydration, humidification, voice rest, decongestant, stop smoking) antibiotic not recommended unless there is suspicion of secondary bacterial infection.

Epiglottitis (acute supraglottitis)

Acute inflammatory condition of supraglottic structures (epiglottis, aryepiglottic folds & arytenoids)

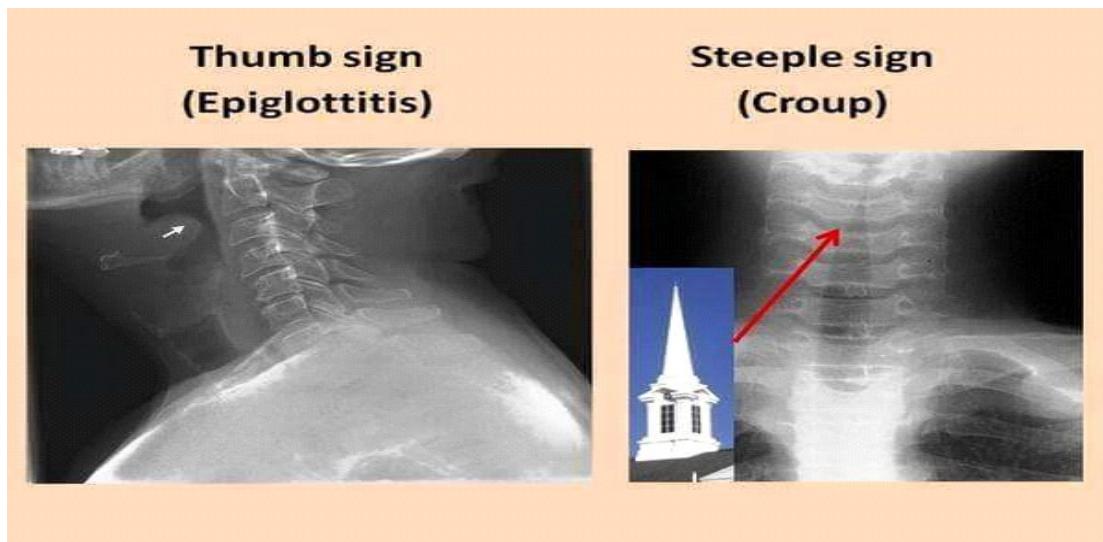
Pathogen: homophiles influenza B bacteria.

Clinical features: there is usually short history with rapid progression,

most commonly in children 3 – 6 years old , high fever , dysphagia, drooling of saliva, dyspnea & stridor & the child prefer sitting position (tripod position) .normal voice & no cough.

Diagnosis: care should take when we examine the mouth by depressing the tongue because it may lead to glottic spasm. Lateral soft tissue x-ray of neck shows swollen epiglottis (**thumb print sign**)

Treatment: hospitalization, I.V antibiotic, steroid, O₂ inhalation, humidification, hydration, prepare for emergency endotracheal intubation or tracheostomy if there is respiratory depression.



Acute laryngotracheobronchitis (CROUP)

Most common cause of stridor in children.Involves the sub glottis region.

Pathogen: parainfluenza 1 (most common), parainfluenza 3
Children 1 -5 years.

Clinical features: stridor, gradual onset (3-7 days) , low grade fever, hoarseness, brassy cough, no dysphagia, no drooling of saliva, child prefers recumbent position.

DX: history & clinical examination. Plain X -ray of neck (steeple sign) **Treatment:** humidified oxygen, hydration, nebulized racemic epinephrine , steroid (controversial) , antibiotic not

needed unless there is secondary bacterial infection, intubation & tracheostomy is rarely needed .

Chronic laryngitis:

Pathogens: smoking, pollution, vocal abuse ,chronic rhinosinusitis

Clinical features: hoarseness, throat pain, dysphagia, cough.

Diagnosis: flexible nasopharyngoscopy, video stroboscopy.

Treatment according to etiology (stop smoking, voice rest, treatment of rhinosinusitis, treatment of gastric reflex) , humidification, mucolytic & sometimes short course of steroid.

Tubercular laryngitis

Associated with pulmonary **TB** ,Shallow ulcer with undermined edge (mouse nibbled appearance) effect **posterior glottis**. Pseudo edema of epiglottis (turban epiglottis) **Diagnosis:** by chest X- ray , sputum for **AFB**
Treatment : anti TB

Syphilitic laryngitis

All stages of disease can be manifested , effect anterior part of larynx .

Primary stage: primary chancre.

Secondary stage: multiple vesicles & popular rash.

Tertiary stage: gummatous lesion.

Treatment: penicillin.

Leprosy (Hansen's disease)

Pathogen: mycobacterium leprae.Ulcerative lesion in the supraglottis.

Diagnosis: biopsy.

treatment: dapsone, corticosteroid.

Fungal laryngitis:

Occur in immunocompromised patients (AIDS, uncontrolled diabetes, steroid abuse, radiotherapy, long term antibiotics)

Pathogens: candida, aspergillosis, blastomycosis, histoplasmosis.

Clinical features: odynophagia, mucositis, dysphonia, cough, aspiration.

DX: endoscopy & biopsy.

RX: anti fungal regimen.

Hoarseness & stridor

Hoarseness of voice: abnormal changes of voice, in which the voice become rough, breathy, raspy and strained, or show changes in volume or pitch. The changes are related to sound producing organs (vocal cords)

Causes of hoarseness:

Acute laryngitis: the most common cause which leads to edema and swelling of vocal cords due to viral infection, common cold or voice strain.

Voice abuse: which means excessive use of voice or using inappropriate pitches (high or too low) also speech in noisy surrounding.

Benign vocal cords lesions: too much use of voice or too loudly may lead to formation of vocal cords nodules (singer nodules) or vocal cords polyp or cyst.

Vocal cords hemorrhage: occurs after strenuous use of voice, which lead to rupture of blood vessel on the surface of vocal cord and the lamina properia is filled with blood. it treated as surgical emergency and complete voice rest.

Gastroesophageal reflux disease GERD: when the acid content of stomach comes up and irritates the vocal cords. The patient has hoarseness worse at morning and improves during the day. The patient also has heart burn and excessive throat clearing.

Reinke's edema: accumulation of fluid in the lamina propria due to vocal abuse, smoking or allergy.

Benign and malignant tumors of larynx: by their effect on the mobility of vocal cords.

Smoking: by causing Reinke's edema or carcinoma of larynx.

Neurological causes: such as Parkinson disease, stroke, spasmodic dysphonia or vocal cords paralysis. Other causes of hoarseness are

Allergy, thyroid problems, trauma to the larynx and menstruation.

Indication of consultation of otolaryngologist:

- Hoarseness more than three weeks with history of smoking.
- There is no flu.
- Patient has lump in the neck.
- If there is dysphagia or odynophagia.
- If there is cough with blood.
- If the patient is voice performer and hoarseness interfere with his job.

Investigation:

By indirect mirror examination, video stroboscopy or by video laryngoscopy.

The **treatment** is according to the cause.

Stridor

Noisy breathing that occurs due to the partial obstruction of air way in the larynx or lower in the bronchial tree. It should be differentiated from stridor which is a lower pitch, snoring type sound generated at the level of nasopharynx or oropharynx.

Stridor is a symptom, not diagnosis or disease. It may be inspiratory in laryngeal obstruction, expiratory in tracheobronchial obstruction or biphasic in sub glottis or glottis obstruction.

Causes of stridor:

Congenital:

Laryngomalacia: usually benign and self-limiting condition & improves as the child reaches one year of age. It is due to under developed cartilages of epiglottis and rarely need surgical intervention.

Sub glottis stenosis: usually biphasic stridor, the symptoms usually not found at neonatal period and become evident at first years of life.

Laryngeal web: caused by incomplete recanalization of laryngeal lumen during embryonic life, infant has biphasic stridor and weak cry. Intervention is recommended in case of significant obstruction.

Laryngeal cyst: usually in supraglottic region.

Laryngeal hemangioma: glottis or sub glottis , usually rare.

Congenital vocal cords paralysis.

Tracheomalacia.

Other extra laryngeal causes: vascular ring compression, cystic hygroma, thymus gland and enlarged lymph node.

Inflammatory causes:

Croup (laryngotracheobronchitis): most common cause, especially in children 6 months – 2 years.

Acute laryngitis.

Diphtheria.

Retropharyngeal abscess: usually in children below 6 years.

Ludwig angina: sub mandibular space infection, which cause swelling and compression of airway.

Per tonsillar abscess (Quinzy) : pus collected between tonsillar capsule and superior constrictor muscle causing narrowing of airway.

Bacterial tracheitis: rare, caused by staphylococcus aureus, effect children younger than 3 years.

Allergic reaction: (anaphylaxis) occurs 30 minutes after adverse exposure, accompanied by sneezing, itching, nasal congestion, wheezy chest and stridor.

Edema: may be due to GERD

Foreign body: Which may be laryngeal, tracheal, bronchial or esophageal and cause external compression to the airway.

Trauma: by sharp or blunt trauma, thermal or chemical or iatrogenic.

Neoplasms: benign like cyst or papilloma, or malignant tumor of larynx.

Neurological causes: like vocal cords paralysis.

Investigations:

- Video laryngoscope or bronchoscope is the best methods of examination.
- Anteroposterior and lateral plain radiograph of neck and chest.
- Barium esophagography: detects vascular compression, tracheoesophageal fistula or GERD.
- CT scan: detect mediastinal masses or aberrant blood vessels. Also MRI is useful in detecting vascular abnormalities.
- If GERD is suspected PH probe is used or barium swallows.

Treatment: the priority is to secure the air way and then treat the underlying cause.

Dysphagia

Definition:

Dysphagia is difficulty in swallowing, The term odynophagia is used when Swallowing causes pain. The later is more marked in ulcerative and inflammatory lesions of food passages.

Aetiology:

The cause of dysphagia may be preoesophageal (i.e due to disturbance in the oral or pharyngeal phase of deglutition) , or oesophageal (when disturbance in oesophageal phase). This classification is clinically useful as most of the preoesophageal causes can be easily excluded by physical examination while oesophageal once need investigation.

Preoesophageal causes:

1.Oral phase: Normally, food must be masticated lubricated with saliva,converted into a bolus by movements of tongue and then pushed into the pharynx by elevation of the tongue against hard palate. Any disturbance in these events will cause dysphagia.Thus cause may be:

- a) Disturbance in mastication . e. g trismus, fractures of mandible,tumors of upper or lower jaw and disorders of temporomandibular joint.
- b) Disturbance in lubrication. e.g xerostomia following radiotherapy, Mikulicz's disease.
- c) Disturbance in mobility of tongue.e. g paralysis of tongue,painful ulcers,tumors of tongue,lingual abcess,total glossectomy.
- d) Defects of palate.e. g cleft palate,oronasal fistula.
- e) Lesions of buccal cavity and floor of mouth. e.g stomatitis,ulcerative lesions, Ludwig's angina.

2.Pharyngeal phase: For a normal swallow ,food should enter the pharynx and then be directed towards oesophageal opening.All unwanted communications into the nasopharynx, larynx,oral cavity should be closed.Disturbances in this phase can arise from:

- a) Obstructive lesions of pharynx , e.g tumors of tonsil,soft palate,pharynx,base of tongue,supraglottic larynx,or even obstructive hypertrophic tonsils.
- b) Inflammatory conditions , e.g acute tonsillitis, peritonsillar abscess ,retro or parapharyngeal abscess,acute epiglottitis,edema of larynx.
- c) Spasmodic conditions, e.g tetanus,rabies.
- d) Paralytic conditions , e.g paralysis of soft palate due to diphtheria, bulbar palsies,cardiovascular accidents. They cause regurgitation into the nose,while paralysis of larynx, lesions of vagus and bilateral superior laryngeal nerves cause aspiration of food into the larynx.

Oesophageal causes:

The lesions may lie in the lumen,in the wall or outside the wall of oesophagus:

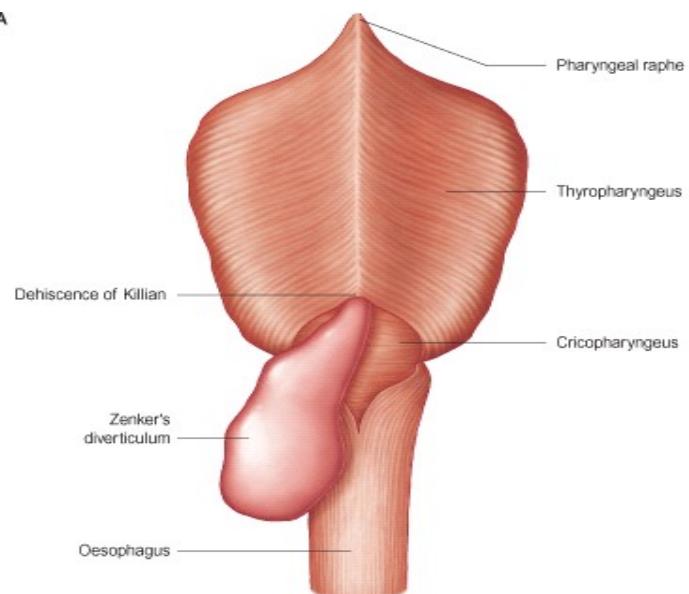
- 1.Lumen:Obstruction to lumen can occur in atresia,foreign body,strictures,benign or malignant tumors.
- 2.Wall:It can be acute or chronic oesophagitis,or motility disorders.The latter are:
 - a) hypomotility disorders,e.g Achalasia,scleroderma,amyotrophic lateral

sclerosis.

b) Hypermotility disorders, e. g cricopharyngeal spasm, diffuse oesophageal spasm.

3. Outside the wall: The lesions cause obstruction by pressing on the oesophagus from outside:

a) Hypopharyngeal diverticulum (Zenker's diverticulum): It is a diverticulum of the mucosa of the pharynx, just above the upper oesophageal sphincter in Killian's dehiscence (area of weakness).



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Figure: Showing Zenker's diverticulum.

b) Hiatus hernia(the stomach bulges up into the chest).

c) Cervical osteophytes.

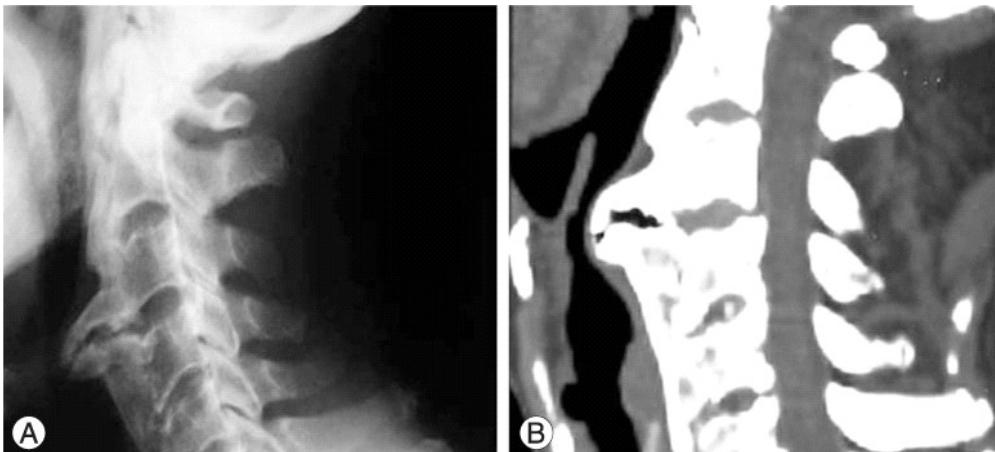


Figure 2: Cervical osteophytes causing dysphagia.

- d) Thyroid lesions,e, g enlargement,tumors,Hashimoto's thyroiditis.
- e) Mediastinal lesions , e.g tumors of mediastinum,lymph nodes enlargement, aortic aneurysm, cardiac enlargement.
- f) Vascular ring(dysphagia lusoria): Most commonly by anomalous right subclavian artery.

Diagnosis:

- 1.History: A detailed history is of paramount importance.Ascertain,if dysphagia of:
 - a) Sudden onset : Foreign body or impaction of food on a preexisting stricture or malignancy,neurological disorders.
 - b) Progressive : e. g malignancy.
 - c) Intermittent: spasm or spasmodic episodes over an organic lesion.
 - d) More to liquids: Paralytic lesions.
 - e) More to solids and progressing even to liquid:Malignancy or stricture. f) Intolerance to acid food or fruit juices:Ulcerative lesions.
 - g) Any associated symptoms, e.g regurgitation and heart burn (hiatus hernia),regurgitation of undigested food while lying down,with cough at night(hypopharyngeal diverticulum);aspiration into the lungs(laryngeal paralysis);aspiration into the nose(palatal paralysis).

2.Clinical examination:

Examination of oral cavity,oropharynx,larynx and hypopharynx can exclude most of the preoesophageal causes of dysphagia Examination of the neck,chest and nervous system including cranial nerves should be done.

3.Blood examination:

Haemogram is important in the diagnosis and treatment of Pulmmer - vinson syndrome and to know the nutritional status of the patient.

4.Radiography:

- a)X-ray chest: To exclude cardiovascular,pulmonary and mediastinal diseases.
- b) Lateral view neck : To exclude cervical osteophytes and any soft tissue lesions of postcricoid or retropharyngeal space.
- c) Barium swallow: It is useful in the diagnosis of malignancy,cardiac aschlasia , strictures ,diverticula , hiatus hernia or oesophageal spasm.Combined with fluoroscopic control or cineradiography, it can help in the diagnosis of motility disorders of oesophageal wall or sphincters.

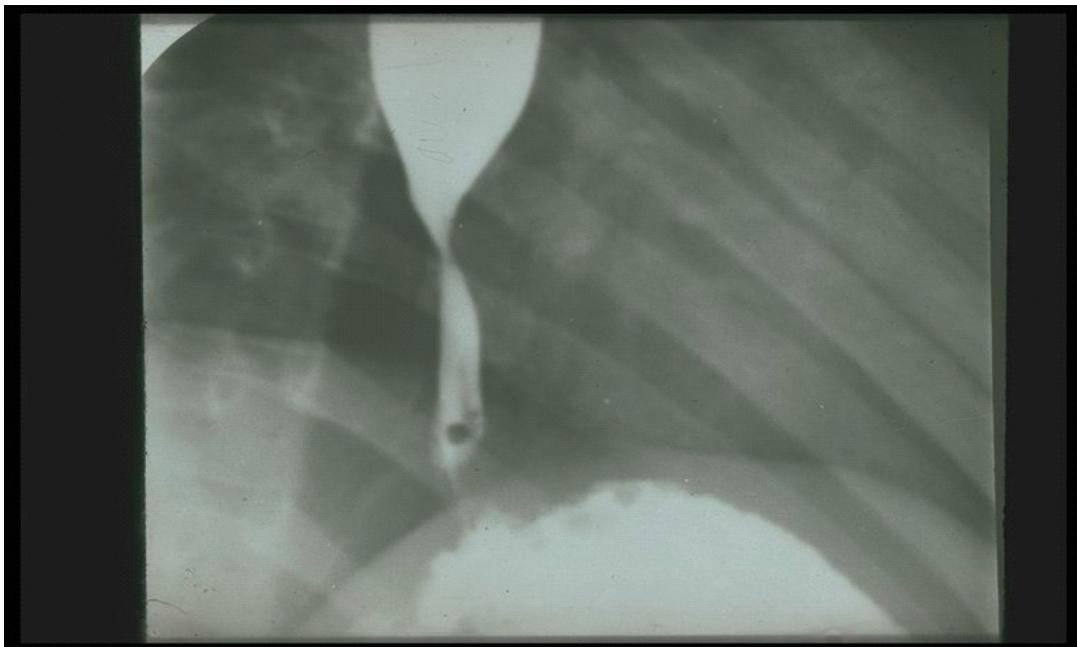


Figure:Barium swallow showing aschlasia cardia.

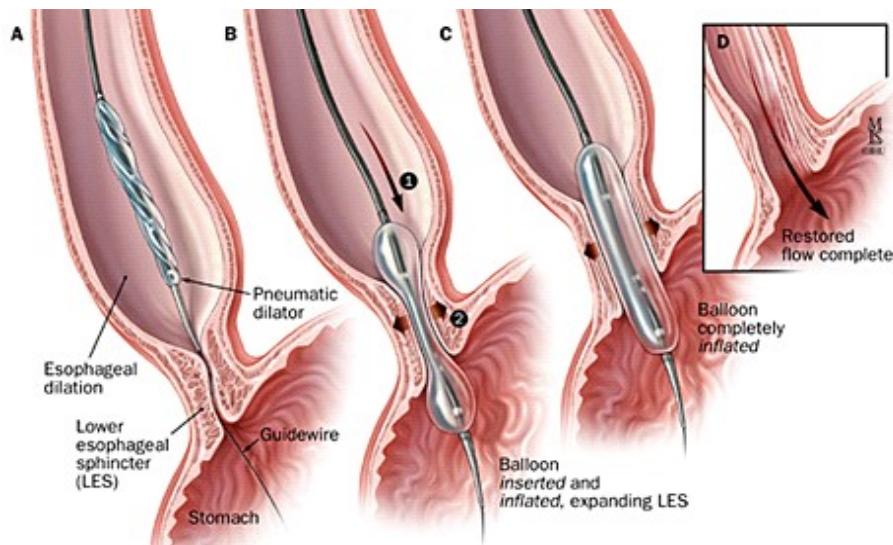
5.Manometric and pH studies: A pressure transducer along with a pH electrode and an open-tipped catheter is introduced into the oesophagus to measure the pressures in the oesophagus and its sphincters . Acid reflux is measured by pH electrode . These studies help to diagnose motility disorders,gasrg-Oesoivihagfial reflux and to find whether oesophageal spasms are spontaneous or acid induced.

6.Oesophagoscopy: It gives direct examination of oesophageal mucosa and permits biopsy.Flexible fibroptic or rigid scopes can be used.

7.Other investigation:

Bronchoscopy(for bronchial carcinoma),cardiac catheterization(for vascular anomalies),thyroid scan(for malignant thyroid lesion) may be needed.

Treatment: Depending on the type and cause of dysphagia:



a) Swallowing rehabilitation: Certain exercise may help to coordinate swallowing muscles or restimulate the nerve that trigger the swallowing.Change food consistencies (purred diet easier to tolerate initially , liquids are more difficult to manage). Learn some ways for chewing or positioning the body or head to help swallowing.

b) Medications: Dysphagia associated with gastro-oesophageal reflux

disease can be treated with oral medications to decrease stomach acid , in eosinophilic esophagitis,the patient may need corticosteroid.

c)**Oesophageal dilatation:** For aschlasia cardia or stricture ,the doctor may use endoacope with special balloon.

(Figure:Showing balloon dilatation.)

d) **Surgery:** For tumors,pharyngeal diverticulae or cricopharyngeal spasm(cricopharyngeal myotomy).Sever dysphagia may need gastric or jejunal feeding tube.

Laryngeal tumors

Benign laryngeal tumors

Squamous papilloma: the most common benign tumor of larynx & it is of two types

- Juvenile papilloma: usually caused by viral infection, HPV 6 & 11, multiple sessile, friable, bleed on touch. Occurs in infants & young children, most common site is vocal folds. Patient presented with hoarseness of voice, later become worse with dyspnea & stridor. The treatment usually by micro endoscopic co2 laser excision, also treated by interferon alfa which used as adjuvant therapy in severs disease. The recurrence rate is high.
- Adult onset papilloma: single, smaller, less aggressive course. No recurrence after removal. Usually effects 30- 40 years old & mostly in male patient .patient usually presented with hoarseness, treatment is similar to juvenile type.

Chondroma: arise from cricoid cartilage & causing dyspnea & feeling of lump in throat. Usually effect male 40-60 years.

Hemangioma: juvenile type mainly involves sub glottis area & presented with stridor which tends to resolve spontaneously, sometimes patient need tracheostomy in case of respiratory obstruction.

Treated by co2 laser.

Laryngeal cancer:

Epidemiology:

- 2% of all cancer worldwide.
- Male is more affected than female.
- Age group mostly affected is 60- 70 years.

Histopathology:

90%- 95% is squamous cell carcinoma; the other 5% include adenoid cystic carcinoma, neuroendocrine carcinoma & chondrosarcoma.

Etiology:

- Smoking & alcohol are the most important factors, & combination of them increases the risk to 15 times.

- Environmental exposure to wood dust, asbestos, nitrogen mustard & petroleum products.
- Immune deficiency.
- Genetic predisposition.
- Viral infection HPV 16

Classification

Laryngeal cancer classified according to the site of involvement into supraglottic, glottis & subglottic.

- **Supraglottic cancer:** usually affects the epiglottis, false cords or aryepiglottic folds. Symptoms: initial symptom is throat pain or sometimes neck mass due to lymphatic spread which is common in this site, hoarseness is late symptom. Spread occurs to vallecular, base of tongue & pyriform fossa.
- **Glottic cancer:** most common type, usually effect true vocal cords. hoarseness is early symptoms so it can be detected early, late symptoms are stridor & throat pain. Spread usually is by local extension because true vocal cords are devoid of lymphatics.
- **Sub glottic:** the less common type. Early symptom is stridor which appears late, so it cannot be diagnosed early, also hoarseness is a late feature. Spread usually down ward to trachea or upward to vocal cords.

Diagnosis:

Risk factors, duration of symptoms, associated medical conditions, symptoms of metastatic disease	History
Status of airway, extent of primary tumor, appearance of tumor (fungating, ulcerating, nodular), vocal cord fixation, neck disease, laryngeal click, neck extension (Talaat sign)	Physical examination
By indirect & direct laryngoscopy, biopsy taking	Endoscopy
Lab. Tests	
Assess nutritional status	Albumin, transferrin
Hypercalcemia may signify metastatic disease	Calcium
Elevated with bony metastasis, liver disease, and other	Alkaline

conditions	phosphatase
Routine preoperative screening	Electrolytes, creatinine, blood count
Radiology	
Useful in detecting subglottic extension, cartilaginous involvement, extralaryngeal spread, and nodal disease	CT scan of neck with contrast
Helps detect metastatic disease as a separate primary in the chest	CT scan of chest/chest radiograph
Most useful in detecting occult nodal disease	MRI

Staging

Primary tumor	
primary tumor cannot be assessed	Tx
no evidence of primary tumor	T0
Supraglottic	
one subsite, normal mobility	T1
involving mucosa of adjacent subsite (suprahyoid epiglottis, infrahyoid epiglottis, arytenoids,	T2

aryepiglottic fold, false vocal fold)	
Tumor limited to larynx with vocal cords fixation	T3
Tumor extend beyond larynx	T4
Glottic	
limited to one vocal fold	T1a
both vocal folds (or involved anterior commissure)	T1b
extends to supraglottis or subglottis or with limited vocal fold mobility (transglottic)	T2
Limited to larynx with vocal cords fixation	T3
Same as supraglottis	T4
Subglottic	
within subglottis	T1
Extends to true vocal folds, normal mobility	T2
limited to larynx with <i>vocal cord fixation</i>	T3
invades through <i>thyroid cartilage</i> , and/or extends into <i>tissues beyond</i>	T4a
invades <i>prevertebral space</i> , encases <i>carotid artery</i> , or invades <i>mediastinal structures</i>	T4b
Lymph nodes	
regional nodes cannot be assessed	Nx
no regional node metastasis	N0
single ipsilateral node, < 3 cm	N1
single ipsilateral node, > 3 cm and < 6 cm	N2a
multiple ipsilateral nodes, < 6 cm	N2b
contralateral or bilateral nodes, < 6 cm	N2c

node > 6 cm	N3
Distal metastasis	
Distant metastasis cannot be assessed	Mx
No distant metastasis	M0
Distant metastasis	M1

Management

Supraglottic Cancer:

T1 radiotherapy

T2 supraglottic laryngectomy with or without neck dissection.

T3, T4 total laryngectomy with neck dissection & postoperative radiotherapy.

Glottic Cancer:

Carcinoma **in situ** endoscopic removal by CO₂ laser

T1, T2 radiotherapy

T3m T4 total laryngectomy with radical neck dissection for clinically positive lymph nodes, with or without postoperative radiation

Subglottic cancer:

T1, T2	radiotherapy
T3, T4	total laryngectomy with postoperative radiotherapy

Thyroid Gland

Anatomy:

Situated anteriorly in the visceral compartment of the neck at the level of the fifth, sixth and seventh cervical and first thoracic vertebrae. It consists of right and left pear-shaped lobes 5 cm in length, connected by a narrow region of gland (isthmus).

The pyramidal lobe is a variable tail of thyroid tissue, a remnant of descent of thyroid, present in approximately 40% of people. The thyroid gland is suspended in the neck from the cricoid and thyroid cartilage by two ligaments:

- a. Anterior suspensory ligament: attaches the superior border of the isthmus and superomedial aspect of each lobe to the cricoid cartilage.
- b. Posterior suspensory (Berry's) ligament: thickening of fascia extends from deep portions of each lateral lobe to the lateral surface of the cricoid cartilage and first two tracheal rings.

Arterial supply:

1. **Inferior thyroid artery:** The dominant artery to the thyroid, arises from the thyrocervical trunk of subclavian artery.
2. **Superior thyroid artery:** The first branch of the external carotid artery, closely related to the course of the external branch of the superior laryngeal nerve.
3. **Thyroidea Ima artery:** Arises from the innominate (brachiocephalic) artery, courses to the isthmus in midline

Venous drainage:

1. **Superior thyroid vein:** Arises from upper pole, accompanies the superior thyroid artery, empty into the internal jugular vein.
2. **Middle thyroid vein:** Highly variable, present in roughly 50% of patients.
3. **Inferior thyroid vein:** Drains the lower portion of the gland to the brachio cephalic vein of the same side. Inferior thyroid veins form thyroid impar plexus, a rich anastomosis in front of the trachea, which can produce troublesome bleeding in a tracheostomy.

Nerve supply:

From the superior cervical ganglion of the sympathetic trunk and the superior laryngeal branch of the vagus.

These nerves are related functionally to blood vessels only and not directly to the function of glandular tissue.

Lymphatics:

Isthmus and median lateral lobes drain to the delphian (prelaryngeal) and diagastric nodes. Inferior lateral lobes drain to pretracheal and cervical nodes.

Physiology:

- Thyroid gland contains two types of functioning endocrine cells:
 1. **Follicular cells:** secrete L-thyroxine (T4) and Triiodo-L-thyronine (T3).
 2. **Parafollicular cells (C cells):** secrete Calcitonin which influence calcium metabolism.

Production & liberation of active thyroid hormone:

- a. Energy-dependent uptake of extracellular iodide across the follicular cell membrane.
- b. Oxidation of iodide.
- c. Iodination of tyrosine on newly synthesized thyroglobulin produces T3 and T4, which are stored in the follicular lumen bound to thyroglobulin.
- d. Previous reactions are catalyzed by thyroid peroxidase enzyme.
- e. Liberation of active thyroid hormone requires uptake of colloid through the follicular cell with proteolysis of thyroglobulin and release of free T3 and T4.
- f. Follicles secrete only 20% of serum T3; de iodination of circulating T4 produces the remainder. Thus, T4 exerts most of its metabolic effects through its conversion to T3.

- Plasma proteins reversibly bind almost all of the serum T3 and T4, leaving only 0.3% of T3 and 0.03% of T4 free to act on receptor sites. (ie. concentration of free T3 is about 10 times greater than free T4; due to weaker binding of T3 to plasma protein).
- Thyrotropin = Thyroid-stimulating hormone [TSH]:
 - Secreted by the anterior pituitary gland.
 - It acts on the follicular cell receptor to increase all aspects of follicular cell metabolism, including synthesis of hormone and thyroglobulin and hormone secretion.
 - Hypothalamic secretion of Thyrotropin-releasing hormone (TRH) controls TSH secretion.
 - Free thyroid hormones in serum exert negative feedback at the level of the pituitary by inhibiting TSH secretion and antagonizing TRH.
 - At the level of the thyroid gland, iodine depletion enhances the responsiveness to TSH, whereas iodine enrichment inhibits the TSH response.

Calcitonin:

- The main endocrine effect is to decrease the number and activity of osteoclasts, thereby reducing bone resorption.
- Extra-thyroid calcitonin synthesized in the pancreas, gastrointestinal tract, pituitary, and brain probably acts as a locally inhibitory neurotransmitter.

Thyroid hormone functions:

- Increase metabolic rate
- Essential for normal skeletal and neural development.

- Increase sympathetic activity.
- Releases steroid hormones.
- Stimulates erythropoiesis.

Disorders of the Thyroid Gland

Goitre

1. Non-toxic Goiter:

a) Physiologic goiter: During puberty and pregnancy, the thyroid gland normally undergoes a diffuse enlargement. This is related in part to increased estrogens and subsequent increase in thyroid binding globulin (TBG). This condition is usually self-limited and rarely requires treatment.

b) Endemic goiter: When 10% or more of the population has thyroid enlargement.

It generally reflects a dietary deficiency of iodide particular to a geographic region, causing insufficient thyroid hormone secretion. Histologically is colloid goiter.

c) Non-toxic multi-nodular goiter: Cause of nodular goiter is poorly understood but may be related to varying levels of TSH over a lifetime, female to-male ratio 6 to 1.

Histologically normal parenchyma is scattered between nodules of varying size and consistency. Nodules smaller than 2 cm are rarely noticed by patients.

Large multinodular goiters may be asymptomatic, or they can cause compression of neck structures, resulting in dysphagia, cough, or respiratory distress or a feeling of constriction in the throat. Treatment is indicated if a goiter is a cosmetic problem or if it becomes symptomatic, medical treatment including exogenous thyroid hormone to suppress TSH secretion or radioiodine for more extensive cases.

d) Solitary thyroid nodule: isolated mass, greater than or equal to 1 cm in diameter, discovered by palpation of a thyroid gland otherwise of normal size and consistency.

Occur in up to 4% of the population. With ultrasonography, nodules occur in up to 50% of individuals over 50 years of age.

The most common cause of solitary thyroid nodules is follicular adenoma.

Spontaneous disappearance was found to be the most common outcome for an untreated, long-standing nodule (40 %), 45% were of stable size, 15% of nodules were enlarging. Needle biopsy demonstrated malignancy in 25% of enlarging nodules, compared with 5% of nodules that were of stable size. The overall incidence of carcinoma in solitary nodules ranges from 1 to 20%.

2. Toxic Goiter:

a) Graves' Disease: [The most common cause of thyrotoxicosis.]

Triad of:

1. Hyperthyroidism with diffuse goiter.
2. Ophthalmopathy.
3. Dermopathy (localized myxedema).

Etiology:

Autoimmune mechanism with production of long-acting thyroid stimulating antibody, IgG type, that stimulates thyroid gland TSH receptors.

Symptoms: Sympathetic overdrive symptoms: nervousness and tremors, tachycardia, hypertension, diarrhea, insomnia, and heat intolerance, diffuse goiter.

Ocular signs: Stare look, with infrequent blinking, and lid lag owing to sympathetic overstimulation, proptosis, ophthalmoplegia. More common in female

Dermopathy: thickened skin.

Thyroid crisis (storm):

Occurs in a thyrotoxic patient who has an acute infection or other medical illness, an injury or a major operation. It may also occur after ^{131}I therapy, discontinuation of an ant thyroid medication, or spontaneously.

Clinical findings: fever, tachycardia, anxiety, agitation and delirium, seizures, and as a terminal event, coma. Severe cardiovascular effects such as congestive heart failure or atrial fibrillation may also be present. Serum T4 and T3 concentrations may be high but no more so than in ordinary thyrotoxicosis.

Treatment:

- a. Propranolol given orally or intravenously is the most immediately effective treatment.
- b. Antithyroid medications.
- c. Corticosteroids in large doses.
- d. Inorganic iodine given orally.
- e. Additional supportive therapy to reduce hyperpyrexia and appropriate parenteral fluid and electrolyte support.

Diagnosis (Graves): mainly clinical:

Increased total T4 RIA, free T4 and T3 resin uptake. Histopathologic examination: hypertrophy and hyperplasia of the thyroid follicular cells.

Medical Treatment:

1. Antithyroid therapy: Propylthiouracil or Metimazole: Inhibit thyroid hormone biosynthesis by inhibiting the oxidation and organification of iodine and the coupling of iodotyrosines, reactions that are catalyzed by thyroid peroxidase.
2. Glucocorticoids: Help to reduce the production of long acting thyroid stimulator and improves ophthalmopathy.
3. Inorganic (Lugol's) iodine: Inhibits thyroid hormone secretion, primarily by inhibiting thyroglobulin proteolysis, and also inhibits iodine transport, oxidation, and organification.
4. B-blockers: Minimize many of the sympathetic overdrive symptoms, used for preparation before surgery, used to control thyroid storm.

Contraindicated in patients with severe thyrotoxic cardiomyopathy and heart failure.

Radioiodide (I^{131}):

A major advantage is that only a single usually reduces thyroid size to normal, and it is safe.

Subtotal (Near-total) thyroidectomy:

Used in young patients or in those for whom radioiodide is contraindicated.

b) Toxic Nodule (Adenoma):

Caused by thyroid hormone secreted by follicular adenoma.

This can follow a long period of euthyroid function in which negative feedback suppression of normal gland balances the hyper functioning adenoma.

Radioiodide is an effective treatment for toxic adenoma, thyroid lobectomy is appropriate for patients at risk.

c) Toxic Multinodular Goiter: (Plummer's disease)

Occurs late in the natural history of long standing simple (non-toxic) multinodular goiter, usually in women 50 years old or older.

Characteristic history of thyroid enlargement with insidious development of thyrotoxicosis. These patients generally do not demonstrate ophthalmopathy or localized myxedema and do not undergo spontaneous remissions.

I¹³¹ is the treatment of choice because surgical resection generally requires removal of most of the thyroid gland and patients who are not candidates for radioiodine therapy may undergo surgery.

Hypothyroidism:

Women-to-man ratio = 6:1

Causes:

- 1. Thyroid agenesis**
- 2. Iatrogenic:** surgical removal, radioiodine, irradiation, antithyroid medications.
- 3. Hashimoto's thyroiditis** (the most common cause)
- 4. Replacement by cancer and infiltrative diseases** (amyloidosis, scleroderma)
- 5. Iodine deficiency**

Clinical Manifestations:

General:	GIT:	Skin:	Goiter
Fatigue, lethargy	Constipation	Dry, rough, thick skin	CNS:
Weight gain	Anorexia, vomiting	Coarse hair	Slowed speech, movement
Cold intolerance	Cardiovascular	Non pitting edema (myxedema)	Genitourinary
ENT:	:	Periorbital edema	:
Macroglossia	Bradycardia	Loss of lateral eyebrows	Menstrual irregularity
Hearing loss, tinnitus	Pericardial effusion	Musculoskeletal:	(menorrhagia)
Middle ear effusion	Pulmonary: Pleural effusion	Arthralgia	
Vertigo, tinnitus		Carpel tunnel syndrome	
Hoarseness of voice			
Dysphagia			

Myxedema Coma:

Myxedema coma/crisis occurs most commonly in older women with long-standing undiagnosed or undertreated hypothyroidism who experience an

additional significant stress, including cold environment, infection, other systemic disease, or certain medications.

Reduced metabolic rate and decreased oxygen consumption result in peripheral vasoconstriction, which maintains core temperature.

Myxedema coma/crisis is a form of decompensated hypothyroidism in which adaptations are no longer sufficient. Essentially, all organ systems are affected.

Characterized by Coma or pre coma with sever clinical manifestations of myxedema including extreme hypothermia, bradycardia, pleural and pericardial effusions, hypoventilation and hypoxia. Focal or generalized seizures typically precede the coma.

Management is with large doses of intravenous T4 and hydrocortisone.

Supportive care includes intubation and assisted ventilation, cautious warming, support of blood pressure, and management of infection.

Chronic Lymphocytic Thyroiditis (Hashimoto Thyroiditis):

Immunologically mediated thyroid cell damage caused by antibodies against thyroid peroxidase and thyroglobulin (TG), with lymphocytic infiltration of the thyroid gland, results in thyroid gland failure and variable degrees of enlargement. It is the most common etiology for goiterous hypothyroidism.

Pathology:

There is lymphocytic infiltration of the thyroid tissue.

There is progressive fibrosis, which may be extensive.

Clinical Manifestations:

Most commonly presents as an asymptomatic goiter in a middle-aged woman, but can occur at any age. Goiter is symmetrical, non-tender, and rubbery.

Occasional regional lymph node enlargement.

Diagnosis:

- Antibodies to thyroid peroxidase (90% of cases) or to TG (50%).
- Thyroid isotope scan: patchy appearance.
- Ultrasound: hypoechogenicity and heterogeneity.
- FNA biopsy to rule out malignancy.

Treatment:

- Thyroid hormone replacement for hypothyroidism.
- Throidectomy.

Malignant tumors of the thyroid gland:

1. Papillary Adenocarcinoma: 70%

The most common type, it accounts for 90% of radiation-induced thyroid carcinomas. the women-to-man ratio = 3:1. And the peak incidence is in the third and fourth decades.

25% of papillary carcinomas are occult, discovered incidentally at surgery.

Histologically: un encapsulated tumor, with well differentiated cells arranged in papillae. This lesion generally has a prolonged course, and the mortality is estimated at 10% or less. The patient's age at diagnosis is the most important prognostic factor. The prognosis is excellent in children and young adults, even in the presence of advanced primary disease or lymph node metastases.

Variants:

1. Warthin-like tumor: characterized by prominent T- and B-cell lymphocytic infiltration in stalks of papillae similar to Warthin salivary tumor.
2. Tall cell variant: it is usually presented in older age group, tends to present at an advanced stage associated with a more aggressive clinical course.

Treatment:

- For lesions confined to thyroid: total thyroidectomy is the modality of choice.
- For more extensive well-differentiated disease, total thyroidectomy + neck dissection (if lymph nodes) + I ¹³¹ administration after 6 weeks for ablation of the remnant.

2. Follicular Adenocarcinoma: 20%

Peaking in the fifth decade of life, and female to male ratio is 3-1

Histologically: tumors can be encapsulated, with well differentiated cells arranged in follicles. Shows worse prognosis. Distant metastases, especially to lung and bone, are found commonly.

Hürthle Cell Carcinoma:

- A variant of follicular carcinoma formed of Hürthle cells (which observed in Hashimoto's disease as a degenerative transformation of the follicular cells).

- This disease behaves clinically as an intermediate between low-grade and angioinvasive follicular carcinoma.

Treatment: Total thyroidectomy + neck dissection + ablative radioiodine.

3. Medullary Carcinoma: 5%

They are derived from C cells (parafollicular cells) of the thyroid gland and are capable of producing calcitonin. Lesions are capable of local invasion, spread to regional lymphatic vessels, or distant metastases.

Calcitonin is an important tumor marker.

Associated autosomal dominant Multible Endocrine Neoplasia Syndromes

Treatment: total thyroidectomy + neck dissection ((Medullary carcinoma does not take up radioiodide, so this treatment is not recommended)).

4. Anaplastic Carcinoma: 5%

A disease of the elderly which often rapidly fatal, aggressive, enlarging bulky mass that distorts the anterior neck and often obstructs the aerodigestive tract.

Tumor is usually inoperable, palliative radiation therapy and chemotherapy may prolong life.

Complications of thyroid surgery:

1. Recurrent laryngeal nerve injury:
2. Superior laryngeal nerve injury
3. Injury to sympathetic trunk (with a resultant Horner's syndrome)

4. Hemorrhage:

5. Seroma: if large enough, should be aspirated.

5. Injury to thoracic duct: with resulting chyle fistula.

6. Infection:

7. Respiratory obstruction secondary to tracheal malacia.

8. Pneumothorax.

9. Injury to parathyroid glands and their blood supply,

- Transient mild decrease in serum calcium, which is asymptomatic occurs in 25% of cases after total thyroidectomy.

- Manifestations of hypocalcemia:

- Neuromuscular irritability, Tetany.
- Numbness, paresthesias.
- Seizures can be seen.
- Laryngeal tetany.
- Cardiac arrhythmias.

10. Myxedema: Can occur in 4 to 6 weeks after a thyroidectomy.

11. Thyroid storm.

