Essentials of
Otorhinolaryngology

By
Staff Members of Otorhinolaryngology Department
Faculty of Medicine
Mansoura University - Egypt
2007
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First Edition 2007
A Gift

Any revenue generated from the sales of this book will be considered as a donation and contribution from all the staff members of the Otorhinolaryngology department to enhance work performance and improve all the services provided by the beloved department.

Staff Members of
Otorhinolaryngology Dept.
Faculty of Medicine
Mansoura University
Mansoura, Egypt
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Introduction to the first edition

Dear medical students

On behalf of the staff members of Otorhinolaryngology department of Mansoura University medical school, we would like to introduce this book of Otorhinolaryngology for undergraduate teaching. The book provides knowledge that covers the basic principles of Otorhinolaryngology to support general practitioners in their professional life.

Skills required by general practitioners and family medicine physicians will be added to this knowledge base. They will be provided and stress upon in the clinical rounds and facilitated by the audiovisual aids in the new teaching rooms in our department.

Attitudes and medical ethics will be a supreme goal in our teaching curriculum, despite it may seem to be a hidden one.

We are sufficiently optimistic that this book will be a real help in your scientific life.

Staff Members of
Otorhinolaryngology Dept.
Faculty of Medicine
Mansoura University
Mansoura, Egypt
First Edition
2007
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The ear consists of three parts: - the external, middle and inner ear. Fig. (1).

![Diagram of ear parts](image1)

Fig. (1) The right ear.

I- Anatomy of the External Ear

Parts: Fig. (1)
1. The auricle (Ear pinna).
2. The external auditory canal.
3. The tympanic membrane.

1. The Auricle (Ear Pinna) Fig. (2)

It is the comma shaped structure attached to the side of the head by ligaments and muscles. It is composed of a sheet of fibro-elastic cartilage. The covering skin is closely adherent to the perichondrium on the lateral surface.

![Diagram of auricle parts](image2)

Fig. (2): The Auricle.
Blood supply:
The external carotid artery branches: superficial temporal artery, occipital artery, and posterior auricular artery.

Venous drainage:
Corresponds to the feeding arteries internal and external jugular veins.

Nerve supply:
- Motor: The facial nerve to the vestigial auricular muscles
- Sensory:
  1. The auriculotemporal nerve from V cranial nerve to supply the upper 2/3 of the lateral surface.
  2. The lesser occipital nerve (C2) to supply the upper 1/3 of the medial surface.
  3. The greater auricular nerve (C2, 3) to supply the lower 1/3 of the lateral and 2/3 of the medial surfaces.
  4. The facial nerve to the area around the concha at the external meatus.

Lymphatic drainage:
The pre and post auricular lymph nodes.

2. **The External Auditory Canal (Meatus)**
- It extends from base of the concha of the auricle to the tympanic membrane (24mm length in adults).
- The lateral third is cartilaginous, directed medially, upwards, and backwards, continuous with the auricular cartilage. Its skin contains hairs, sebaceous and ceruminous glands (secrete wax).
- The medial two thirds are bony, directed medially, downwards and forwards, continuous with the bony walls of the middle ear. Its skin is much thinner than the outer third and contains no hairs or glands.

Nerve supply:
Sensory only anterior half is the auriculotemporal nerve and posterior half is Arnold's branch of the vagus nerve.

3. **The Tympanic Membrane (The Ear Drum):** Fig. (3)
Site: It is located at the medial end of the external auditory canal, separating the external from the middle ear. It lies at an angle of 55° with the horizontal because the anterior and inferior walls of the external canal are longer than its posterior and superior walls.

Shape: The drum is oval (about 8x10 mm), about 0.1 mm thickness, pearly grey, semi-translucent and concave laterally (towards the external auditory canal) showing the reflected cone of light on examination.

Structure: Outer layer is the skin, the middle layer is fibrous and the inner layer is mucosa.
It is divided into two unequal parts:

1. The larger part: called pars tensa which has a thick rolled periphery called annulus.
2. The smaller uppermost part: called pars flaccida or Shrapnell's membrane.

The two parts are separated by two folds: the anterior and posterior malleolar folds.

Blood supply
Branches from the external carotid artery.

Nerve supply: (Sensory only)
- The inner surface: Jacobson's branch of the glossopharyngeal nerve.
- The anterior ½ of the lateral surface: The auriculo-temporal nerve.
- The posterior ½ of the lateral surface: Arnold's branch of the vagus nerve.

II- Anatomy of the Middle Ear Cleft: - Fig. (4)
It consists of tympanic cavity, Eustachian tube and mastoid air cells.

A. The Middle Ear Cavity (Tympanic Cavity):- Fig. (5)
It is located in the temporal bone between the external ear and the inner ear. It is a six-walled cavity about 15 mm in height, 15 mm antro-posteriorly and 2-6 mm from side to side. All the walls are bony, except the lateral wall (tympanic membrane) and are lined with mucous membrane, which is ciliated columnar epithelium except its postero-superior part that has flattened squamous epithelium.
Tympanic Cavity is divided into three regions:
1. Meso-tympanum: lies opposite to the tympanic membrane.
2. Epi-tympanum (attic): lies above the tympanic membrane level.
3. Hypo-tympanum: lies below the tympanic membrane level.

Contents: It is filled with air and contains: Fig. (5)
1. Three bones (auditory ossicles): malleus, incus and stapes. Fig. (6)
2. Two nerves: The chorda tympani nerve and the tympanic plexus.
3. Two muscles: The tensor tympani muscle and the stapedius muscle.

![Fig. (5): The middle ear cavity](image)

**Walls of the Middle Ear cavity**

**Lateral wall**
It separates the middle ear from the external ear.
- Formed by:
  1. The tympanic membrane with bones above and below.
  2. The outer attic mass (part of the squamous temporal bone) forms the upper part of the lateral wall of the attic (epi-tympanum).

**Medial wall**
- It separates the middle ear from the inner ear.
- **Formed mainly by:**
  1. The promontory is the first turn (basal) of the bony cochlea of the inner ear.
  2. Two openings connect the middle ear with the inner ear:
     - *The oval window:* lies above and behind the promontory. It is closed by the footplate of stapes.
     - *The round window:* lies below and behind the promontory. It is closed by the secondary tympanic membrane.
  3. The horizontal part of the facial nerve: runs in a bony canal above the oval window.
Superior wall
- It separates the middle ear from the middle cranial fossa and temporal lobe of the brain. It is a thin bony plate called the tegmen tympani.

Inferior wall
- A thin bony plate that separates the middle ear from the bulb of internal jugular vein.

Anterior wall
- It separates the middle ear from the internal carotid artery. It has an opening for the Eustachian tube. The tensor tympani muscle enters the middle ear through this wall.

Posterior wall
- It separates the middle ear from the mastoid process.
- It has an opening (the aditus ad antrum) which connects the epitympanum (attic) with the mastoid antrum.
- The stapedius muscle enters the middle ear through this wall through a bony ridge (the pyramid).
- The vertical part of the facial nerve runs in a bony canal in this wall.

B. The Eustachian Tube (E.T.):-

The Eustachian tube communicates the middle ear cavity with the nasopharynx. Its length is about 36 mm and is directed medially, forwards and downwards. Its lateral third is bony while its medial two thirds are cartilaginous. It is lined with mucous membrane with ciliated columnar epithelium. The tube is closed at rest. It opens by contraction of the tensor veli palatini muscle during swallowing and yawning to ventilate the middle ear. The tube is shorter, wider, and more horizontal in children than in adults.

C. The Mastoid Air Cells: - Fig. (7)

According to the degree of cellularity the mastoid process may be: cellular, diploic or acellular (sclerotic) mastoid. They are located within the mastoid process of the temporal bone which is a pyramidal bony projection directed inferiorly behind the auricle. They are small air-filled bony cavities that communicate with each other and lined with flattened squamous epithelium. They communicate anteriorly with the middle ear cavity through the aditus ad antrum. The largest air cell is the mastoid antrum, which lies behind the attic.
The number and size of the other cells vary and are usually arranged in groups as in Fig. (7). The mastoid process is not present in the newborn. It forms from traction on the squamous and petrosal parts of the temporal bone by cervical muscles, since the child starts to support his head.

![Fig. (7): The mastoid air cells.](image)

The functions of the mastoid air cells are still unknown but they are thought to share in pressure-regulating mechanism of the middle ear cavity and in decreasing the weight of the skull bones.

### III- Anatomy of the Inner Ear (Labyrinth)

It is located within the petrous part of the temporal bone between the middle ear and the internal auditory canal. It consists of a membranous labyrinth surrounded by a bony labyrinth:

- The bony labyrinth is a hollow bony capsule, which is a part of the petrous portion of the temporal bone.
- The membranous labyrinth is a group of delicate ducts and sacs.

The membranous labyrinth is filled with a fluid called endolymph and is surrounded by a fluid called perilymph, the membranous labyrinth contains the labyrinthine sensory end organs.

The Bony Labyrinth consists of: Fig.(8)

a) Bony cochlea: A coiled canal that resembles a snail's shell having 2.5 turns.

b) Three bony semi-circular canals (lateral, superior and posterior), each canal forms 2/3 of a circle and they are perpendicular to each other.

c) Vestibule: Between the cochlea and the semi-circular canals.

![Fig. (8): The bony labyrinth.](image)
The Membranous Labyrinth consists of: Fig.(9)

a) Membranous cochlear duct: Within the bony cochlea contains the organ of Corti to Cochlear division of the 8\textsuperscript{th} Cranial nerve.

b) Membranous semi-circular ducts: Within the bony semi-circular canals to vestibular division of the 8\textsuperscript{th} Cranial nerve.

c) Utricle and Saccule: Within the vestibule to vestibular division of the 8\textsuperscript{th} Cranial nerve.

d) Endolymphatic duct and sac.

The Cochlea: Fig. (10)

1. The cochlea forms 2.5 turns around an axis called the modiolus.
2. Cochlear nerve fibers leave the cochlea through the modiolus.
3. The cavity of the bony cochlea is divided into three compartments:
   - A middle compartment called scala media (cochlear duct), filled with endolymph. The organ of Corti (the sensory end-organ of the cochlea) lies in this compartment. It rests on a membrane called basilar membrane.
   - An upper compartment called scala vestibuli, filled with perilymph.
   - A lower compartment called scala tympani, filled with perilymph.

The Labyrinthine Sensory End Organs:

They are the sensory end organs of hearing and equilibrium, located within the membranous labyrinth

- The sensory end organ of the cochlea is called: the organ of Corti.
- The sensory end organ of the semi-circular duct is called: the crista.
• The sensory end organ of the utricle and saccule is called: the macula.
  Each sensory end organ contains special sensory hair cells.

**Physiology of Ear**

β The function of the ear is hearing and equilibrium.

**Physiology of Hearing**

β Hearing is the sensation of sound.

The hearing system of the ear is divided into two parts: -
Conductive part and sensory-neural part.

1- **The conductive part**

It transmits sound as mechanical vibrations and it consists of:

a) The External Ear

β The auricle collects the air-born sound vibrations.

β The external auditory canal transmits the air-born sound vibrations to the tympanic membrane.

β When the air-born sound vibrations reach the tympanic membrane, it vibrates and converts the air-born vibrations to mechanical vibrations.

b) The Middle Ear

β The ossicles have two functions: transmition of the sound waves from the tympanic membrane to the oval window, and amplification of the sound waves by two mechanisms:-

a) The area ratio between the vibrating area of the tympanic membrane and the area of the oval window, is 17 : 1

b) The lever ratio between the arm of malleus and the arm of incus, which is 1.3:1

So, the overall amplification is 17 x 1.3 = 22 - i.e. the intensity of sound at the oval window is as 22 times as that at the tympanic membrane. Fig. (11)

The Eustachian tube ventilates the middle ear to equalize the middle ear and atmospheric pressure. This is important for free movements of the tympanic membrane and ossicles.

Fig. (11): Mechanism of sound amplification.
2- The Sensory-neural part.
It consists of the Cochlea and Cochlear nerve. It transmits sound as electrical impulses.

- The Cochlea
  Converts the mechanical sound vibrations to electrical impulses by vibration of the footplate of stapes in the oval window leading to vibration of the cochlear fluids causing vibration of the basilar membrane leading to stimulation of the cochlear hair cells which lie in the organ of Corti leading to conversion of the mechanical sound vibrations to electrical impulses.

- The Cochlear Nerve
  It transmits the electrical impulses to the cochlear nuclei in the brainstem to the higher hearing centers in the brain (auditory area in the temporal lobe), which perceives them as meaningful sounds.

Physiology of Equilibrium
  Maintenance of equilibrium occurs in three steps:
  1. Afferents (Sensory inputs): The brain receives sensory information about position of the head and body in the space from:
     a. Vestibular labyrinth: Semicircular canals, utricle and saccule.
     b. Visual system.
     c. Proprioceptive receptors of the muscles and joints.
  2. Brain: integrates this sensory information.
  3. Efferents: The brain sends motor orders to the effectors:
     a. Extra-ocular muscles to keep the eyes stable (ocular equilibrium).
     b. Spinal muscles to keep trunk stable (postural equilibrium).

β Normally, there is balance between the two sides through the effect of the resting activity in the two vestibular labyrinths on the cerebellum.

Examination of the Ear

1. The auricle is examined first for congenital anomalies or any abnormality such as position, size, shape, color, consistency and tenderness.
2. The mastoid process is examined for signs of inflammation.
3. The External auditory canal: for discharge, pain sensation on moving.
   Then an ear speculum is introduced in the external auditory canal after its straightening by pulling the auricle upwards and backwards in adults downwards and backwards in children.

The structures seen are:
1- The external auditory canal.
2- The tympanic membrane: The prominent landmarks on the membrane are:
   a) The handle of malleus, which normally runs downward and backwards towards the centre of the membrane.
   b) The cone of light, which appears in the antero-inferior part of the membrane due to reflection of the examination light.
Clinical Examination of Hearing

Tuning Fork Tests

They are simple tests to assess the type of hearing loss (HL) i.e. whether conductive or sensory-neural by using the tuning fork.

The Tuning Fork Fig. (12)

- Made of stainless steel, having a base, stem, and two prongs.
- Frequency used: Commonest 256 and 512 Hz.
- Activation: The prongs are struck on a firm surface as the elbow or patella.

![Tuning fork](image)

Fig. (12): Tuning fork

I- Rinne's Test Fig. (13):

![Rinne's Test](image)

Fig. (13): Rinne's Test

Principle:

It compares hearing by air conduction (AC) with hearing by bone conduction (BC) in the same ear by the tuning fork.

- During hearing by air conduction: Sound passes through the external ear to middle ear to the inner ear.
- During hearing by bone conduction: Sound passes directly to the inner ear without passing through the external or middle ear by bone conduction.

Method:

- To test hearing by AC, the prongs of the vibrating tuning fork are placed near the external canal.
- To test hearing by BC, the base of the vibrating tuning fork is placed on the mastoid process.

Results:

- Normal hearing: AC better than BC = Rinne positive.
- Conductive HL: BC better than AC = Rinne negative.
- Sensory-neural HL: AC better than BC, but both are reduced = reduced Rinne positive
N.B. Unilateral severe Sensory-neural HL: BC appears better than AC = false Rinne negative, because the patient does not hear AC and BC sounds by the diseased ear, but during the BC testing on this diseased ear, sound is transmitted across the skull to be heard by the healthy ear.

II- Weber's Test Fig. (14):

**Fig. (14): Weber's Test**

Principle:
It compares hearing by BC of the patients in the both ears simultaneously. It is useful in case of unilateral hearing loss.

Method:
The base of the vibrating tuning fork is placed on the midline of the forehead or on the upper incisor teeth.

Results:
- Normal hearing: sound is heard in midline or equal in both ears.
- Conductive HL: Sound is heard better in (lateralized to) the diseased ear because the background noise masks hearing in the healthy or better other ear.
- Sensory-neural HL: Sound is heard better in (lateralized to) the healthy or better ear because it has a better inner ear function.

III- Schwabach's Test

Principle
It compares hearing by bone conduction of the patient with that of the examiner, provided that the examiner has normal hearing.

Method
The base of the vibrating tuning fork is placed on the mastoid process of the patient. When the patient no longer hears the tone the tuning fork is immediately transferred to the mastoid process of the examiner.

Results
- Normal hearing: The hearing duration of the patient and examiner are equal = Schwabach normal.
- Conductive HL: The tone is heard longer by the patient = Schwabach prolonged.
- Sensory-neural HL: The tone is heard longer by the examiner = Schwabach shortened.
Eustachian Tube Function Tests

Valsalva's Maneuver:
Forced expiration with closed mouth and nose leads to increased air pressure in nasopharynx leads to opening of the Eustachian tube. When the Eustachian tube is patent causes bulging of the tympanic membrane laterally.

Audiological Evaluation

Audiology
Is the branch of science dealing with normal and abnormal aspects of hearing. The scope of audiology includes clinical audiology, rehabilitative audiology, which includes also prescription, trial and fitting of hearing aids.

Sound
Is the form of physical energy dealt with by auditory system. Sound in nature is found in different forms. It may be a simple sound as pure tones, or it may be a complex sound as noise and speech.

Physical characteristics of sound:
1. Frequency: sounds may be harsh in pitch as the sound of a drum, these are low frequency sounds. High frequency sounds are like violin. The unit of measurement of frequency is called Hertz (Hz).
2. Intensity: is the amount of loudness of sound. Low intensity sounds, are soft while high intensity sounds are loud. The unit of measurement is called decibel (dB).

Audiological Assessment

1. Subjective tests: needs active cooperation of the patient.
   - Pure tone audiometry (PTA).
   - Speech audiometry.
2. Objective tests: do not need active cooperation of the patient.
   - Impedance audiometry.
   - Auditory brain stem response audiometry (ABR).

Objective tests are the only measures to test unable (children and mentally retarded) or unwilling (malingering) patients.

1. Pure Tone Audiometry (PTA)
Measuring the hearing threshold by using pure tones. Pure tones are delivered to the ear through headphones (AC), and by bone vibrator applied to the mastoid (BC). The frequencies tested range from 125 to 8000 Hz, at intensities from -10 to 120 dB.

Technique:
The patient is seated in a sound-treated room wearing headphones connected to an audiometer and responds by pushing a button or raising his hand. In children below three years of age, the free-field technique is used in which the examiner observes the child's responses to sound presented through loudspeakers in a sound-treated room. Above the age of three years, the child is conditioned to respond by performing a certain task as a play whenever he hears a sound (play audiometry).
1. Measurement of the hearing threshold level:
   Measuring the minimal sound intensity which can be heard at each frequency. Air conduction (AC) and bone conduction (BC) hearing thresholds at different frequencies are recorded as a pure tone audiogram.

2. Assessment of the Type and Degree of hearing loss: Fig. (15)
   Type of hearing loss is based on air and bone conduction thresholds.
   CHL: AC threshold is elevated, while BC threshold is normal. The difference between them is called air-bone gap. BC threshold measures the cochlear reserve.
   SNHL: Both AC and BC thresholds are elevated with no air bone gap.
   Mixed HL: Both AC and BC thresholds are elevated but with air-bone gap.

Fig. (15): Types of audiogram

Degree of hearing loss is based on air conduction threshold.
   The threshold of normal hearing ears is 0 dB up to 20 dB. Elevation of AC threshold beyond this limit denotes the presence of hearing loss.
   Hearing loss is categorized according to the degree into: mild, moderate, moderately severe, severe, profound and total hearing loss.

2. Speech Audiometry
   Measuring the ability to hear and understand speech.

3. Impedance audiometry
   a) Tympanometry:
      Measuring the mobility (compliance) of TM and ossicular chain, on changing the air pressure in sealed external auditory canal from -400 to +200 mm H₂O. The results are recorded in a graph called tympanogram
      Fig. (16).
Value of tymanogram in the diagnosis of middle ear diseases:-
1. Normal middle ear: Type A tympanogram (normal compliance 0.3 - 1.5 ml with normal middle ear pressure -100 to +100 mm H2O).
2. Eustachian tube obstruction: Type C tympanogram (normal compliance with negative ME pressure).
3. Ossicular disruption: Type Ad tympanogram (increased compliance)
4. Otosclerosis: Type As tympanogram (reduced compliance)
5. Secretory otitis media: Type B tympanogram (flat curve).

![Fig. (16): Types of tympanogram](image)

b) Measurement of Stapedial Reflex:
High intensity sound is introduced into the ear and the change in the impedance caused by contraction of stapedius muscle, is measured.
β CHL: Absent stapedial reflex.
β Retro-cochlear SNHL: Absent or elevated reflex threshold 120 dB.
β Cochlear SNHL: Stapedial reflex is elicited at lower stimulation thresholds.

Value:
1. Determination of hearing threshold in children.
2. Differentiation between cochlear and retro-cochlear SNHL.
3. Localisation of the facial nerve paralysis.

4. Auditory Brain Stem Response (ABR)
Measuring the electrical potentials in the auditory pathway, which arise from the brain stem in response to sound stimuli.
Value:
1. Measuring hearing threshold level in infants, young children and malingers.
2. Determining the site of lesion in SNHL (cochlear, retro-cochlear or brain stem) by changes in the latency of waves I to V compared to the other ear.

**Radiographic Evaluation of the Ear Disease**
1. Plain x-ray films are of limited use nowadays.
2. Computed tomography (CT), usually gives the best bony definition of the temporal bone. CT scanning does the best assessment of congenital abnormalities and bone destruction by tumour or infection.
3. Magnetic resonance imaging (MRI) with gadolinium gives excellent soft tissue definition of structures in and around the temporal bone. Therefore, inflammatory lesions and tumours are well defined but not the bony lesions.
4. Angiography of temporal bone lesions, especially glomus tumours, may help in defining their extent and blood supply.
Diseases of the External Ear

The Auricle

Congenital anomalies:

- Anotia: Absent auricle. Fig. (17)
- Microtia: Small deformed auricle.
- Macrotia: Abnormally enlarged auricle.
- Accessory auricle: Small-elevation formed of skin and cartilage in front of the auricle.

Treatment: Surgical excision.
- Protruding (Bat) ears: (The commonest anomaly). The ears are protruding and prominent because the antihelix is under developed. Treatment: Otoplasty at the age of 5 years before school admission.
- Pre-auricular sinus: A pin point depression in the skin just in front of the auricle due to lesion defect between auricular tubercles of the first and second branchial arches.

Treatment: Excision if recurrent infections occur.

![Fig. 17: Anotia.](image)

Haematoma of the auricle: - Accumulation of blood between the auricular cartilage and its perichondrium.

Causes:- Blunt trauma, commonly in boxers (boxer's ear). Spontaneous as in elderly and haemorrhagic blood diseases.

Clinical picture: The auricle is painful, swollen, bluish and cystic.

Complications: perichondritis.

Treatment: Incision and evacuation under complete aseptic condition and followed by firm dressing to prevent recollection of blood.

Perichondritis:

Inflammation of the auricular perichondrium mainly due to pyogenic bacteria.

Causes: Infected haematoma, septic surgical trauma, furunculosis of the external auditory canal, post-operative infection, or animal bite or postirradiation.
Clinical picture: The auricle is painful, swollen, hot, reddish soft and tender. The lobule is free, because it has no cartilage. Later on deformity of the auricle occurs.
Complications: Cartilage necrosis leads to fibrosis and auricular deformity called "cauliflower ear".
Treatment: Intravenous antibiotics, incision and drainage with removal of necrosed cartilage

**Foreign Body (F.B.):**
In incidence: Most commonly in children and mentally retarded persons.
Types:
1. Inanimate foreign bodies:
   - Non-vegetable foreign body as pieces of papers, beads or buttons.
   - Vegetable foreign body as beans and peas, they swell with water.
2. Animate foreign bodies as flies, mosquitoes, myasis (larvae) or fleas.
Clinical picture:
1. May be asymptomatic if small F.B..
2. Hearing loss, when the foreign body occludes the external canal.
3. Animate foreign body causes severe irritation, and noise in the ear.
Complications:
Produced by the foreign body or during unskilled attempts of its removal:
1. Injury of the external canal, tympanic membrane or ossicles.
2. Otitis externa or otitis media.
Treatment:
- Hooks can remove most foreign bodies and should be used to remove impacted or large vegetable foreign body. Fig. (18)
- Ear washes: Can remove most foreign bodies except impacted or large vegetable foreign bodies.
   - Insects should be killed by alcohol or oil before wash.
- Under general anaesthesia:
   - It is necessary in cases of impacted foreign body and uncooperative patients as children.

Fig. (18): Removal of a foreign body with a hook.
Otitis Externa:
Inflammation of the skin of the external auditory canal and its types are:-

1- Diffuse otitis externa:
Bacterial infection of the whole skin of the external auditory canal.
Causes:-
Streptococci, Staphylococci, Pseudomonas aeruginosa, B. proteus.
Predisposing factors:-
1. Skin laceration:
2. Skin maceration: due to hot humid atmosphere with excessive sweating and frequent bathing or discharge of chronic suppurative otitis media.
3. Diabetic or allergic patients.
Clinical picture:
1. Pain in the ear increases on moving the jaw as during mastication with scanty and purulent ear discharge.
2. Hearing loss when oedema is severe and occludes the external canal.
3. Skin of the canal is red, oozing, tender, and swollen with narrow lumen.
4. Tenderness on moving the auricle and pressure on the tragus.
5. Pre and post-auricular lymphadenitis.
6. Normal tympanic membrane if it can be seen.
Treatment:
- Prevention of the predisposing factors.
- Systemic treatment: antibiotics and analgesics.
- Local treatment: for the ear discharge by suction or dry mopping.
- Packing the canal with a gauze strip soaked with antibiotic, corticosteroid.

2- Furunclosis: Fig. (19)
- Localized bacterial infection of a hair follicle or glands of the skin of the outer cartilaginous part of the external auditory canal.
- Causative Organism: Staphylococcus aureus.
- Predisposing Factors:
  1. Trauma or scratching the canal skin.
  2. Diabetes mellitus leads to recurrent attacks.
Clinical picture:
- Pain on mastication, touching or moving the auricle. Deafness only if the furuncle is large enough to occlude the canal. Tenderness on moving the auricle or pressing the tragus. Post auricular groove is obliterated. Discharge is purulent & scanty. Tympanic membrane is normal if visible.
Investigations:
- Blood sugar level, especially in bilateral and recurrent attacks.
Fig. (19): A furuncle in the external auditory canal.

Treatment:
1. Systemic treatment: antibiotics (anti-staphylococcal) and analgesics.
2. Local treatment:
   a. Aural toilet i.e. repeated removal of the ear discharge by suction or dry mopping.
   b. Packing the canal with a gauze strip soaked with glycerin ichthyol 10% (glycerin is hygroscopic i.e.decreases oedema) or antibiotic drops.
   c. Surgical incision: only when pointing occurs (very rare).

3- Necrotizing otitis externa (Malignant otitis externa):
A destructive and potentially fatal form of otitis externa, which extends beyond the external auditory canal to the base of the skull.
Incidence: Rare and most commonly in elderly uncontrolled diabetics.
Causative Organisms: Pseudomonas aeruginosa.
Pathogenesis:
The organism produces enzymes, which cause necrotizing vasculitis leading to necrosis. Diabetic micro-angiopathy of the blood vessels also causes necrosis.
Clinical picture:
1. Severe stabbing ear pain.
2. Scanty, sero-sanginous and purulent ear discharge.
3. Granulation tissue in the floor of the external canal, at the junction of cartilaginous part with bony part.
Investigations:
1. Culture and sensitivity tests of the ear discharge.
2. CT scans of the temporal bone reveals skull base destruction.
3. Biopsy: to exclude malignancy.
Complications:
1. Spread of infection leads to osteomyelitis of the temporal bone and skull base.
2. Cranial nerves palsy: Facial nerve, at stylo-mastoid foramen, glossopharyngeal, vagus and accessory nerves, at jugular foramen.
Treatment:
1. Control of diabetes.
2. Massive antibiotic therapy (anti-pseudomonas) e.g. quinolones and aminoglycosidcs, continued until complete cure in at least 6 weeks.
3. Local treatment:
   - Aural toilet i.e. repeated removal of the ear discharge by suction
   - Antibiotic ear drops.

4- **Otomycosis:**
Fungal infection of the skin of the external auditory canal.
Causative organisms: Fungi, Aspergillus niger (black) and Candida albicans (white).
Predisposing factors:
   - Excessive sweating, seborrhea, or hot humid and dusty weather or prolonged use of local antibiotic ear drops.
Clinical picture:
   1. Itching with greyish white ear discharge.
   2. Ear pain due to secondary infection.
   3. Hearing loss, when the fungus mass occludes the external auditory canal.
   4. The lumen of the canal contains a whitish mass with black spots appears as wet newspaper.
Treatment:
   1. Removal of the fungus mass by suction or ear wash.
   2. Anti-fungal ear drops and creams e.g. clotrimazole, nystatin or 2% salicylic acid (keratolytic) in alcohol (fungicidal) for 3 weeks.

5- **Herpes zoster oticus (Ramsey- Hunt syndrome):**
Causative Agent: herpes zoster virus infection of the geniculate ganglion of the facial nerve.
Clinical picture:
   Pain in and around the ear with vesicles on the auricle and in the external auditory canal. Lower motor neuron facial nerve paralysis due to affection of the geniculate ganglion of the facial nerve. May be associated with sensory-neural hearing loss and vertigo due to affection of the cochleo-vestibular nerve (Ramsey-Hunt syndrome).
Treatment:
   1. Anti-viral therapy as acyclovir (oral and local).
   2. Corticosteroids (oral and local) in severe cases with affection of the facial and or cochleo-vestibular nerves.

**Tumours of the External Ear:**

1-Tumours of the External auditory canal:
   **Benign tumours:**
   - Exostosis of the external canal with bone growth due to irritation of the periosteum e.g. cold water in swimmer's ear. If large, it leads to occlusion of the canal causing conductive deafness.
Treatment is by drilling the exostosis and widening the bony canal.

- Osteoma differs from exostosis in being solitary and pedunculated. Treated by excision.
- Fibrous dysplasia: the external canal is involved as a part of the affected temporal bone. Ground glass appearance on X-rays or CT is diagnostic. Treatment is by drilling and recontouring of the affected bones.

**Malignant tumours:-**
- Squamous cell carcinoma. It originates deep in the canal. It presents early with bloody ear discharge, and deafness. It commonly complicates chronic otitis media. Biopsy is diagnostic. Treatment:-
  - Temporal bone resection is performed with postoperative radiotherapy.

**Wax (Cerumen)**
- It is a mixture of secretions of the ceruminous (wax) and sebaceous glands with desquamated skin cells of external canal.
- Wax is expelled outside the canal, in the form of flakes, by epithelial migration.
- Abnormal accumulation of ear wax in the external auditory canal leads to hearing loss.

**Functions of the wax:-**
1. Lubrication of the skin of the external canal.
2. Contains fungicidal and bactericidal enzymes.
3. Prevent dust and sand to enter the ear as they stick to it.

**Causes of wax accumulation:**
1. Attempt of the patient to clean his ear with cotton buds leads to pushing the wax deeper into the canal.
2. Increased wax production.
3. Narrow external canal.

**Clinical picture:**
1. Wax occlusion of the external auditory canal is the commonest cause of conductive hearing loss in adults.
2. Unilateral or bilateral hearing loss, fullness of the ears and/or tinnitus especially after bathing.
3. On examination the wax is brownish mass in the external canal.

**Treatment:**
1. The hard wax must be softened by a wax solvent as glycerin Na. bicarbonate ear drops before ear wash.
2. Removal the wax by ear wash or instruments.

**Ear Wash**

**Indications:**
1. Wax with complete occlusion of external auditory canal with hearing loss.
2. Foreign body in the external auditory canal.
3. Otomycosis.
Contraindications:
1. Tympanic membrane perforation.
2. Otitis externa.
3. Impacted or large vegetable foreign body in the external auditory canal.
4. A fistula between the middle and inner ears.

Technique: - Fig. (20)
1. Use syringe of ear wash by warm water at body temperature (37° C).
2. Pull the auricle upwards and backwards to straighten the meatus.
3. Direct the nozzle of the syringe of ear wash upward and backward in the external auditory canal and be gentle and careful during wash.

Complications:
1. Reflex: Cough or syncope due to stimulation of the vagus nerve.
2. Vertigo and nystagmus: due to caloric (thermal) stimulation of the inner ear by too cold or hot water.
3. Trauma: Rupture of the tympanic membrane due to forcible jet of water directed toward the tympanic membrane. The patient feels sudden pain, hearing loss, tinnitus, slight bleeding from ear, and water in his throat or injury of the canal skin by very hot water or the nozzle of syringe due to sudden patient movement.
4. Infective:
   § Otitis externa, otomycosis due to use of non-sterile water or syringe.
   § Otitis media due to traumatic rupture of tympanic membrane.

Diseases of the Tympanic Membrane

I- Rupture Tympanic Membrane:
Causes:
1. Direct trauma:
   § Foreign body or during its removal.
   § Self-inflicted trauma.
   § Improper ear wash or instrumentation.
   § Longitudinal fracture of temporal bone.
2. Indirect trauma: Due to rapid pressure changes:
   § Hand slap on the ear (commonest).
Otitic barotrauma.
Blast injury (explosion).
Sudden fluid compression in jumping in swimming pool.

Clinical picture:
1. Ear pain: transient at the time of rupture.
2. Bloody ear discharge: mild and transient at the time of rupture.
3. Hearing loss and Tinnitus.
4. Air escapes out of the ear on blowing of the nose producing whistle.
5. Perforation occurs commonly in pars tensa. It usually locates in the antero-inferior quadrant (in indirect trauma as slap), irregular in shape, has hyperaemic edges and is surrounded by blood clots. In self-inflicted cases, the perforation is posteroinferior.
6. Tuning fork tests: conductive hearing loss.

Differential diagnosis: Fig. (21) and Table (1)

Table (1): Differential diagnosis of traumatic perforation of TM.

<table>
<thead>
<tr>
<th></th>
<th>Traumatic Perforation</th>
<th>Pathological Perforation</th>
</tr>
</thead>
<tbody>
<tr>
<td>History</td>
<td>Trauma.</td>
<td>Suppurative Otitis media.</td>
</tr>
<tr>
<td>Ear discharge</td>
<td>Bloody (scanty).</td>
<td>Mucopurulent.</td>
</tr>
<tr>
<td>Perforation</td>
<td>- Pars tensa.</td>
<td>- Pars tensa</td>
</tr>
<tr>
<td></td>
<td>- Central, usually antroinferior.</td>
<td>- Central, marginal or attic.</td>
</tr>
<tr>
<td>Site</td>
<td>Irregular. Thin, sharp, hyperaemic and surrounded by blood clots at the edge.</td>
<td>Rounded, oval or kidney-shaped. Thick, smooth, regular edge and surrounded by pus.</td>
</tr>
<tr>
<td>Shape and Edge</td>
<td>Usually small.</td>
<td>Any size.</td>
</tr>
<tr>
<td>Size</td>
<td>Normal.</td>
<td>May be congested.</td>
</tr>
<tr>
<td>Rest of drum</td>
<td>Normal.</td>
<td>May be congested.</td>
</tr>
<tr>
<td>Mucosa of middle ear</td>
<td>Normal.</td>
<td>May be congested.</td>
</tr>
</tbody>
</table>

Complications:
1. Otitis media.
2. Permanent drum perforation.
3. Ossicular dislocation.
4. Implantation cholesteatoma.

Treatment:
1. Conservative treatment:
   Prophylactic systemic antibiotic therapy, decongestant nasal drops, avoid ear contamination, do not wash the ear, do not use eardrops and do not blow the nose forcibly.
2. Surgical treatment: Myringoplasty when the perforation fails to heal after three months.

II- **myringitis Bullosa:**
Aetiology: mostly viral (Influenza virus).
Clinical picture:
- Severe pain and fullness in the ear are common. The drum and sometimes the deep meatus show hyperemic bullae. The bullae can rupture spontaneously, causing a small amount of serous or serosanguineous drainage.

Treatment:
- Symptomatic treatment, an audiogram should be performed.

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**Diseases of the Middle Ear**

**I- Congenital Anomalies**
1. Aplasia or hypoplasia: very rare; cause congenital conductive deafness.
2. Absence or fusion of ossicles: cause congenital conductive deafness.
3. Dehiscence of the floor of tympanic cavity: expose the jugular bulb.
4. Dehiscence of the facial canal: expose the facial nerve.

**II- Traumatic lesions**
1. **Otitic Barotrauma:**
   It is trauma to the middle ear caused by acute atmospheric pressure changes that may cause rupture of tympanic membrane.
   Mechanism Fig. (22)
   1. During descent of the airplane or diving, in presence of Eustachian tube obstruction.
   2. E.T fails to open so the air fails to enter the middle ear to equalize the pressure on both sides of the tympanic membrane leads to lowering of the air pressure in the middle ear cavity than that of the atmosphere causing retraction of the tympanic membrane with middle ear effusion.
   3. In rapid descent, the relatively high atmospheric pressure pushes the tympanic membrane medially leads to rupture of the tympanic membrane.

Fig. (22): Mechanism of otitic barotrauma
Predisposing factors leads to Eustachian tube obstruction:
1. Inflammation (rhinitis or sinusitis)
2. Allergic rhinosinusitis
3. Adenoid hypertrophy.

Clinical picture:-
1. Severe ear pain and sense of ear fullness.
2. Deafness, tinnitus.
3. Congested retracted drum.
4. Fluid or hemorrhage in middle ear in severe cases.
5. Tympanic membrane may rupture, bleeding per ear in severe cases

Treatment:-
1. Prophylactic:
   a. Avoid flying with nasal obstruction.
   b. Avoid sleeping during descent of the airplane (the swallowing action or chewing gum opens the Eustachian tube).
   c. Nasal decongestant drops
2. Therapeutic:
   a. Middle ear inflation by Valsalva's method or chewing gum.
   b. Myringotomy and aspiration if middle ear effusion occurs.
   c. Treatment of tympanic membrane rupture.

2. **Traumatic Ossicular Disruption**

Aetiology:
1. Head trauma. 2. Foreign body. 3. Ear surgery.

Pathology:
- Disruption of incudo-stapedial joint or incudo-malleolar joint.
- Dislocation of stapes (causing perilymph fistula).

Symptoms:
1. Deafness and tinnitus.
2. Vertigo due to perilymph fistula.

Signs:
1. Intact or ruptured tympanic membrane.
2. Conductive deafness.
3. +ve Fistula test: in case of perilymph fistula.

Investigations:
- Pure tone audiometry: Conductive deafness may be mixed with SNHL (due to inner ear injury).
- Tympanometry: Type Ad tympanogram.

Treatment: Middle ear exploration and ossiculoplasty.

3. **Temporal Bone Fractures (Fracture base of skull)**

Aetiology:
Head injury due to car accidents or fall from height.
Types:
a. Longitudinal fractures (80%): Fracture line passes along the longitudinal axis of petrous bone. It involves the middle ear, TM and EAC.
b. Transverse fractures (20%): Fracture line passes at right angle to the longitudinal axis of petrous bone. It involves the inner ear, 7th and 8th nerves.

Clinical picture:
a. Longitudinal fractures: Fig. (23)
   1. Bleeding from ear and may be cerebrospinal otorrhea: if TM is ruptured.
   2. Lacerations in the skin of EAC.
   3. Ruptured TM or intact TM with haemotympanum.
   4. Ecchymosis over the mastoid (Battle sign).
   5. Conductive hearing loss: due to rupture TM, haemotympanum and/or ossicular disruption.
b. Transverse fractures: Fig. (23)
   1. Haemotympanum with intact TM.
   3. Vertigo and Nystagmus (due to unilateral labyrinthine failure).
   4. L.M.N. Facial paralysis: Common (50%); usually immediate and complete.

Investigations:
1. CT scan temporal bone.
3. Vestibular assessment: ENG.
4. Electroneuronography test (ENoG): to detect facial nerve degeneration.

Treatment:
Conservative:
1. Same conservative lines of treatment for rupture tympanic membrane, CSF otorrhea and facial paralysis.
2. Vestibular sedatives: for vertigo.
Surgical:
1. Myringoplasty and/or ossiculoplasty: for persistent TM perforation and/or ossicular disruption.
3. Facial nerve decompression, anastomosis or grafting: for immediate complete facial paralysis, or if ENoG showing 90% nerve degeneration.
Otitis Media

Acute Suppurative Otitis Media
Definition:
- Acute inflammation of the mucosal lining of the middle ear cleft.
Incidence:
- Occurs more commonly in infants and children than in adults.
Causative Organisms:
- Beta hemolytic streptococci and streptococcus pneumonia.
- Haemophilus influenzae.
- Staphylococcus aureus.
- Moraxilla catarrhalis
- Viral infection payes the way for 2ry bacterial invasion.
Routes of Infection:
- Through the Eustachian tube (E.T.) from: nasopharynx; from adenoids, and post nasal discharge, vomitus and milk regurgitation. Nose and sinuses; during rhinitis and sinusitis.
- Through drum perforation: during bathing, swimming, and ear wash.
- Through blood stream (very rare).
Pathology:
1. Stage of E.T. obstruction: - It occurs due to oedema and congestion of the mucosal lining of the E.T. leading to negative pressure inside the middle ear leading to mild retraction of the tympanic membrane.
2. Stage of catarrhal otitis media: - Means oedematous and congested middle ear mucosa causes serous exudation inside the middle ear cavity.
3. Stage of suppuration: - Accumulation of purulent and mucopurulent secretions inside the middle ear causes bulging tympanic membrane which may necrose and rupture leading to perforation of the drum.
4. Stage of resolution: - The condition is self limiting in most of the cases depending on the virulence of the organism and immunity of the patient.
Clinical Picture:
According to the pathological staging, the manifestations will be classified into:-
1. Stage of E.T. obstruction:
   Complaint: Mild deafness (autophony).
   Examination: Mild drum retraction and mild conducive hearing loss
2. Stage of catarrhal otitis media:-
   Complaint: Pain, deafness, tinnitus and mild fever.
   Examination: Congested tympanic membrane with mild to moderate conducive hearing loss.
3. Stage of suppuration:-
   a. Before perforation:
      Complaint: Severe ear pain, fever, deafness and tinnitus.
      Examination: Bulging tympanic membrane which is severely congested with a pale center. Moderate conducive hearing loss.
b. After perforation:
Complaint: Pain disappears and fever drops down, mucopurulent and purulant discharge. Mild deafness and tinnitus.
Examination: Discharge in the external ear canal, mucoid or mucopurulent and excessive and drum perforation. Mild conductive deafness.

4. Stage of resolution:
Resolution gradually occurs with subsidence of symptoms and signs in any of the above stages. Resolution may be complete or incomplete.

Fate of acute suppurative otitis media:
1. Complete resolution: perforation healed; drum intact with normal position and normal hearing.
2. Incomplete resolution with: -
   a. Residual discharge: due to high or small perforation.
   b. Residual deafness: i.e. Secretory otitis media, or residual perforation.

Treatment:
1. General Measures: systemic antibiotic, analgesics, rest, and fluids.
2. Local treatment:
   a. Before perforation:
      - Decongestant nasal drops to open the E.T. to ventilate the middle ear cavity.
      - Anesthetic ear drops to relive the pain
      - Myringotomy (incision of the drum to drain the pus)
   b. After Perforation:
      - Suction of the discharge
      - Antibiotic ear drops.
      - Decongestant nasal drops.
      - Myringotomy is only indicated if the perforation is high, small, or inadequate to drain the secretions.

Acute Suppurative Otitis Media in Infants and Children

Incidence:
- More common in this young age due to:
  - The E.T. is wider, shorter, and more horizontal than in adults leading to easy transmission of infection.
  - The adenoids enlargement in infants.
  - The frequency of upper respiratory infection is more in children.
  - The low resistance in such age due to teething and artificial feeding.

Clinical Picture:
- Severe general symptoms:- fever, vomiting and diarrhea (may be mistaken as gastroenteritis)
- Crying, restlessness, no sleeping and pulling ear.
The tympanic membrane is diffusely congested but bulging occurs later than in adults (thick drum).

Treatment:
- The same as in adults except:
  - Myringotomy is indicated if pain and fever persists for more than 48 hours despite medical therapy as bulging of the drum is late.

Acute necrotizing otitis media of children.
- It is severe acute inflammation in patients having low resistance as during exanthemata. This inflammation will lead to necrosis of tympanic membrane which may be totally lost.

Chronic Otitis Media
A- Chronic Non-Suppurative Otitis Media

1. Secretory Otitis Media (Otitis Media with Effusion)
Definition: Accumulation of non-suppurative effusion (serous or mucoid) in the middle ear cleft behind an intact tympanic membrane.

Incidence:
- More in children and is commonly bilateral.

Aetiology:
1. Prolonged eustachian tube obstruction: due to adenoids, nasopharyngeal tumour, rhinitis, sinusitis, radiotherapy or cleft palate.
2. Unresolved ASOM: due to inadequate antibiotics or inadequate tubal drainage.
3. Otitic barotrauma.
4. Viral infection.
5. Allergy.
6. Hypogamma-globulinaemia.

Types: Effusion could be: Serous, Mucoid (glue ear) or Sero-mucinous.

Symptoms:
1. Deafness, tinnitus and ear discomfort.
2. May be bubbling sound in the ear.
3. May be asymptomatic.

Signs:
1. Tympanic membrane: retracted, colour is amber yellow or dull grey, mobility is restricted, and there may be a fluid level (biconcave hairline) and air bubbles.
2. Tuning fork tests: Conductive deafness.
   N.B. In case of secretory otitis media of adults, the nasopharynx must be examined very well to exclude malignant tumour of the nasopharynx.

Investigations:
1. Pure tone audiometry: CHL.
2. Tympanometry: type B flat tympanogram.
Treatment:
Medical: should be tried initially for 3 months.
1. Treatment of predisposing factors.
2. Antibiotics, corticosteroids, mucolytics and nasal decongestants.
3. Auto-inflation of the Eustachian tube by Valsalva’s manoeuvre.

Surgical: After failed medical treatment myringotomy with ventilation tube insertion. Fig.(24)
Aim: to drain by myringotomy and suction of the fluid and ventilate the middle ear by the tube.

Fig. (24): Insertion of grommet tube.

Sequelae:
1. Resolution with near normal hearing.
2. Atelectasis, Adhesive otitis media or Tympanosclerosis.
3. Cholesteatoma formation.

2. Atelectatic middle ear:
   • Displacement of the drum medially towards the ossicles and the promontory. The drum becomes very thin due to loss of its middle fibrous layer. This condition may lead to retraction pocket and cholesteatoma.
   • Complaint: Deafness & tinnitus.
   • Examination: Severely retracted and very thin drum.
   • Conductive deafness.

Treatment:
   • Trials of repeated inflation of E.T e.g by Valsalva Manoeuvre and insertion of ventilation tube. In resistant cases cartilage tympanoplasty.

3. Adhesive otitis media
   • Long standing untreated secretory otitis media may lead to adhesions of the drum to the bony structures inside the middle ear, the patient will have deafness, and the drum is fixed.

Treatment: Hearing aid.
4. Tympanosclerosis
Definition: Whitish patches (chalky patches) seen on the tympanic membrane, due to calcium carbonate deposition in the tympanic membrane
Site: Commonly in the drum but may invade the ossicles.
Treatment: If no symptoms no treatment. Tympanoplasty in severe conductive deafness.

Myringotomy Operation
Definition: Surgical incision of the tympanic membrane.
Indications:
1. In acute suppurative otitis media with:
   - Bulging drum about to perforate.
   - Small or too highly seated perforation.
   - A.O.M. with complications.
   - A.O.M. with failed medical treatment for 48 hours.
   - A.O.M. in infants.
2. In chronic secretory otitis media.
3. In otitic barotrauma.
4. As a step in cortical mastoiditis.

Fig. (25): Myringotomy for ASOM.

Technique: Fig. (25)
1. Under general anaesthesia (infant and children) or local anaesthesia (adults).
2. Sterilization of the ear.
3. Through ear speculum, use the myringotomy knife a crescentic incision form below upwards in postroinferior part of the drum to avoid injury of the ossicles and jugular pulp
4. Suction of the discharge.
5. In case of chronic secretory otitis media radial incision of anteroinferior part and put grommet tubes (ventilation tube).
Complications:
1. Injury of the incudostapedial joint.
2. Injury of high jugular bulb (in congenital dehiscence of the middle ear floor).
3. Injury of the external canal skin.

B- **Chronic Suppurative Otitis Media (CSOM)**

Definition: Chronic suppurative inflammation of the mucoperiostium lining the middle ear cleft with drum perforation and purulent discharge.

![Fig. (26): A- Tubo-tympanic CSOM B- Attico-antral CSOM](image)

**Clinical types of CSOM:**

**A. Tubo-tympanic CSOM (safe type):** Fig. (26, A)

Infection lies mainly in the E.T. and tympanic cavity. It is safe type as it affects the mucosa only and less liable to cause complications.

Aetiology:-

- Incomplete resolution of ASOM with perforation due to:-
  - Inadequate medical treatment of acute suppurative otitis media.
  - Inadequate drainage through small or high perforation
  - Recurrent attacks of infection.
  - Highly virulent organism (exanthemata).
  - Low patient's resistance.

Histopathology:

- Area of infection is lined by ciliated columnar epithelium that responds to infection by swollen congested mucosa and exudation of mucopurulent.

Symptoms:

- Discharge: recurrent attacks of profuse, mucopurulent odourless discharge.
- Deafness and tinnitus.

Examination:

- Mucopurulent non fetid discharge in the external meatus.
- Perforation of membrane tensa of the ear drum is central.

Central perforation in the drum means a perforation at any site in the membrana tensa of the drum with any size but it is surrounded by drum remenant all around the perforation.

Site: according to the sectors of the drum: Anterosuperior, Anteroinferior, Posterosuperior or Posteroinferior of membrane tensa.

Size: Small, large, or subtotal.
Shape: Round or kidney shaped.
Middle ear mucosa: red and oedematous in active stage, pale and dry in inactive stage.
Aural polyp: is a pale oedematous mucosa coming through the perforation.
Tuning fork test: conductive i.e. negative Rinne's test.

Investigations:
1. Hearing tests: by pure tone audiometry mild to moderate conductive deafness.
2. Culture sensitivity test for ear discharge.

Differential Diagnosis:-
1. Between traumatic and pathological perforations of the drum. Table (1)
2. Attico-antral CSOM .Table (2)

Treatment:
1. Medical therapy to convert active stage into an inactive stage.
   - Treatment of predisposing factors (adenoid and chronic sinusitis).
   - Proper antibiotic (systemic and local).
   - Aural toilet (repeated suction).
   - Follow up to assess complete healing to get the inactive stage (dry central perforation).
2. Surgical treatment: For inactive CSOM i.e. dry ear for 3 months
   - Tympanoplasty without or with ossiculoplasty
   - Tympanoplasty with mastoidectomy for persistent discharge.

B. Attico-antral CSOM (Cholesteatomatous, or unsafe type) Fig.(26,B)
   - The infection is in the attic and antral areas of the middle ear cleft with affection of the mucosa and bone and almost is associated with cholesteatoma.
   - This area of infection is lined by flat epithelium that responds to infection by granulations and exudation of scanty pus.
   - It is unsafe type as it is more liable to cause complications.
**Cholesteatoma:**

**Definition:**
- Presence of keratinized stratified squamous epithelium in the middle ear cleft. It is a skin in the wrong place.

**Pathology**
- It is a sac present inside the middle ear cleft, lined by keratinized stratified squamous epithelium, filled with exfoliated squamous epithelium that accumulates in concentric layers (keratin), containing cholesterol crystals and proteolytic enzymes.

**Behavior of cholesteatoma:** Progressive expansion with bone erosion by pressure necrosis and osteolytic enzymes secreted on secondary bacterial infection. This causes:
1. Osteitis with granulation tissue and polyp formation.
2. Erosion of ossicles.
3. Spread of infection causing otogenic complications.

**Types of cholesteatoma:**
1. **Congenital cholesteatoma:**
   - Embryonic epithelial cell rests in the temporal bone as inner ear is ectodermal in origin. It is more and usually with normal drum. Occupy any site in the temporal bone (CPA, petrous bone, middle ear).
2. **Primary acquired cholesteatoma:** (no history of previous ear disease)
   - Long time of E.T. obstruction leads to negative middle ear pressure cause postero-superior retraction pocket of the drum leads to formation of skin sac inside the attic leads to attic cholesteatoma (invagination theory).

**Theories of pathogenesis:** Fig. (28)
- **Invagination theory:** Prolonged Eustachian tube obstruction → negative intra-tympanic pressure → invagination of pars flaccida → attic retraction pocket → accumulation of keratin → cholesteatoma.
- **Invasion theory:** Invasion of the attic by hyperplasia of the basal epithelial layer of pars flaccida of TM → cholesteatoma
3. **Secondary acquired cholesteatoma:** Occurs on top of chronic Suppurative otitis media.

**Theories of pathogenesis:** Fig. (28)
- **Migration theory:** The stratified squamous epithelium migrates from the outer surface of the drum through the edge of the perforation to inside of the middle ear.
- **Metaplasia theory:** Long standing infection in the middle ear mucosa causes metaplasia of the epithelial lining so simple columnar change to keratinized stratified squamous epithelium (cholesteatoma).
Clinical Picture:

Symptoms:-
- Discharge is purulent, scanty, and offensive (characteristic).
- Deafness and tinnitus.

Signs:
- Discharge is purulent, scanty, and offensive
- Attic perforation (in pars flaccida) or marginal perforation and cholesteatoma which appear as pearly whitish mass in the attic or the posterosuperior part of the drum. Granulation tissue or polyp may be present.
- Tuning fork tests:- Conductive deafness.(Negative Rinne’s Test)

Investigations:
- Culture and sensitivity of ear discharge
- Hearing tested by pure tone audiometry: - Pure tone audiometry: Moderate to severe conductive hearing loss due to T.M. perforation and ossicular necrosis. Sensori-neural hearing loss may occur due to spread of infection to the inner ear.
- Plain x-ray both mastoids
- C.T. scan: Cholesteatoma appears as radiolucent area surrounded by sclerosed margin.

Differential diagnosis:

<table>
<thead>
<tr>
<th>Table (2): Differential diagnosis of types of CSOM.</th>
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<tbody>
<tr>
<td><strong>Ear discharge</strong></td>
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<tr>
<td>Central in pars tensa.</td>
</tr>
<tr>
<td>Perforation</td>
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<tr>
<td>Cholesteatoma</td>
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<tr>
<td>Granulations</td>
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<td>Polyp</td>
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</tbody>
</table>

Treatment: Is surgery
A- Open mastoidectomy.
   - Removal of all or part of the posterior bony canal wall.
   Aim: creating an exteriorized cavity to make the ear safe and dry.
   1. Radical mastoidectomy: Fig. (29)
      Indication: Extensive cholesteatoma with sensori-neural hearing loss.
   2. Modified radical mastoidectomy:
      *Indication*: Localized cholesteatoma with serviceable hearing.
B- Closed mastoidectomy:
   - The posterior bony canal wall is not removed.
   Aim: to preserve normal anatomical contours with better hearing results.
   *Indication*: Localised cholesteatoma with serviceable hearing.
C- Tympanoplasty with mastoidectomy: either in one or two stages:
   1. Eradication of pathology in the middle ear and mastoid through
      atticotomy, modified radical mastoidectomy or canal wall up
      mastoidectomy.
   2. Reconstruction of sound conductive hearing mechanism.
D- Atticotomy:
   *Indication*: Localised attic cholesteatoma.

**Radical mastoidectomy** Fig. (29):
Definition:
   - Removal of all the mastoid air cells and the middle ear contents except
     the stapes.
   - Convert the mastoid cavity and middle ear cavity into one cavity
     after removal of the posterior meatal wall.

![Fig. (29): Radical mastoidectomy](image)

Indications:
   - Attico-antral CSOM with extensive cholesteatoma, SNHL or
     complications.
   - Chronic specific otitis media (T.B. or syphilis).
   - Tumours of the middle ear glomus or carcinoma.

Technique:
   1. postauricular incision.
   2. Cortical mastoidectomy.
   3. Removal of the "bridge" formed by the outer attic wall (the posterior
      meatal wall overlying the ossicles).
   4. Lowering of the facial ridge by removal of the posterior meatal wall to
      the level of facial nerve.
5. Removal of all middle ear contents except the stapes to avoid spread of infection to the inner ear.
6. Obliteration of Eustachian tube orifice by muscle graft.
7. Meatoplasty: to widen the EAC to facilitate aeration, drainage, regular inspection and cleaning of the mastoid cavity.

Complications of radical Mastoidectomy:

- Injury of facial nerve.
- Injury of lateral sinus leads to bleeding.
- Injury of Inner ear.
- Injury of the dura.

**Tympanoplasty Operation**

Aim:

1. Eradication of pathology in the middle ear cleft (granulations, polyps, and diseased cells)
2. Reconstruction of conductive mechanisms to improve the hearing by closure of the perforation ± ossicular reconstruction.

**Complications of Suppurative Otitis Media**

It occurs due to spread of infection beyond the mucoperiosteal lining of the middle ear cleft. Despite the advances in the antibiotics, it still frequently occurs.

The spread of infection occurs due to:

1. Bone erosion by:
   a. Hyperaemic decalcification in acute suppurative otitis media
   b. Granulations or cholesteatoma in chronic suppurative otitis media.
2. Preformed pathways in congenital dehiscence, non-closed sutures, fractures, and after surgery.
3. Retrograde thrombophlebitis, along the venous pathway and the emissary veins, is especially important in acute suppurative otitis media.

Complications can occur with all forms of suppurative otitis media. So the concept of safe otitis media is not true, since no infection is safe. The complaint of pain, vertigo, localized headache in the ear, and increasing tinnitus of patients with chronic otitis media, may give as sign of complication. Depending on the pattern of spread of middle ear infection, the complications are usually classified as cranial, intra-cranial, and extra-cranial. Fig. (30).
Complications of suppurative otitis media are: Fig. (31)
1. Cranial complications (bony skull):
   a. Mastoiditis.
   b. Petrositis.
   c. Otitic labyrinthitis.
   d. Otitic facial paralysis.
2. Intra-cranial complications (inside the intra cranial cavity):
   a. Extra-dural abscess.
   b. Sub-dural empyema.
   c. Lateral sinus thrombosis or thrombo-phlebitis
   d. Meningitis.
   e. Brain abscess (Temporal lobe or Cerebellum).
   f. Otitic hydrocephalus
3. Extra-cranial complications (in the soft tissues of the head and neck):
   a. Otitis externa.
   b. Retropharyngeal abscess.

1-Cranial complications (bony skull)
   a-Mastoiditis:-Fig. (32)
      Extension of infection from the mucoperiosteal lining to the bony septa and cortex of the mastoid process in the course of suppurative otitis media.
Acute Mastoiditis:

- The commonest complication of suppurative otitis media. It commonly occurs in the course of acute suppurative otitis media in the well-pneumatized mastoids, more in children.

Stages:

1. Acute coalescent Mastoiditis: with necrosis of the bony septa of the mastoid air cells forming an abscess (Intra-mastoid abscess with intact cortex).
2. Subperiosteal or mastoid abscess: when pus erodes the mastoid cortex it escapes under the periosteum causing a fluctuant swelling, usually behind the auricle (post-auricular abscess). If the root of the zygoma is pneumatized, the abscess may form in front and above the auricle (Zygomatic abscess). Pus may escape under the periosteum to reach the fascial planes of the muscles attached to the mastoid tip as the sternocleidomastoid (Bezold's abscess) or the posterior belly of the digastric (Citelli's abscess).
3. Mastoid fistula: forms if the abscess is bursts through the scalp or incised.

Fig. (32): Right post-auricular abscess.

Symptoms:

- The condition commonly occurs in the inadequately treated acute suppurative otitis media and manifests as:
  1. Increasing pain in the ear.
  2. Increasing or recurring fever.
  3. Increasing and persistent discharge.
  4. A post-auricular swelling appears.

Signs:

1. Discharge is muco-purulent, profuse, and reaccumulates rapidly after removal (positive reservoir sign).
2. Tenderness on the mastoid antrum (very characteristic).
3. Sagging of the posterosuperior skin of the deep part of the external auditory meatus due to oedema from the underlying periostitis.
4. Tympanic membrane is congested and usually perforated or less commonly intact and bulging.
5. A fluctuant abscess may form at the possible sites as mentioned before.
6. Conductive deafness.
Differential diagnosis:
1. Furunculosis of the external auditory canal.
2. Histiocytosis-X (e.g. eosinophilic granuloma), can present as acute mastoiditis especially in infants.
3. Post-auricular lymphadenitis, due to scalp infection.

Investigations
- CT: is more accurate, and can show other co-existing complications.
- Audiological evaluation: though difficult but sometimes necessary in mastoiditis.
- Culture and sensitivity test of the ear discharge.

Treatment:
1. Medical treatment, includes:
   a. Antibiotics use broad-spectrum antibiotics that have the ability to bypass brain barrier.
   b. Analgesic, antipyretics and anti-inflammatory agents.
   c. Nasal decongestants, local & systemic to improve the function of the Eustachian tube.

Cortical Mastoidectomy (Schwartz Operation):- Fig. (33)
- It is an operation used to drain the mastoid infection by removing the mastoid air cells and cortex, without interrupting the middle ear or its ossicles. Myringotomy is usually combined to drain the middle ear.

![Cortical mastoidectomy](image)

Fig. (33): Cortical mastoidectomy.

Indications:
1. Acute mastoiditis with:
   - Failure of medical treatment after 48 hours.
   - Mastoid abscess.
   - Mastoid fistula.
2. As a step in other more extensive surgery as:
   - In chronic suppurative otitis media safe type.
   - Posterior tympanotomy, for cochlear implantation.
   - Glomus tympanicum surgery.
   - In translabyrinthine approaches for internal auditory canal and CPA tumours.
   - In endolymphatic sac decompression and shunt in Meniere's disease.
   - In facial nerve decompression.
Chronic Mastoiditis:
It occurs in the course of chronic otitis media, with granulation tissue involving the mastoid, eroding the bone, and causing more complications. It is more common in the sclerotic mastoids.
Treatment: Mastoidectomy with or without tympanoplasty.

b. Petrositis
Inflammation of the air cells in the petrous apex. It is one of the causes of persistent discharge after previous inadequate cortical or radical mastoidectomy.
Clinical picture:
1. Persistent otorrhea.
2. Ipsilateral retro-orbital pain due to irritation of the nearby trigeminal ganglion.
3. Ipsilateral squint and diplopia due to paralysis of the 6th nerve.
Investigation:
CT is diagnostic.
Treatment:
1. Antibiotics.
2. Surgical drainage, through following the infected cell to the petrous apex.

c. Otitic Labyrinthitis
Labyrinthitis due to spread of the infection through the following stages:
1. Circumscribed labyrinthitis due to fistula between the middle and inner ears. The patient will have transient vertigo and nystagmus and with positive fistula test.
N.B. Fistula test: a test to detect the presence of fistula between the middle and inner ears. With the use of a Siegle's pneumatoscope, or pressure on the tragus.
2. Diffuse labyrinthitis
Clinically, the patient will have severe incapacitating vertigo that is continuous for days or weeks. Other manifestations include tinnitus, profound deafness (perceptive), nausea, and vomiting. The patient is usually bed ridden.
Complications:
1. Meningitis.
2. Labyrinthitis ossificans and dead ear.
Treatment:
1. Medical: Antibiotics, anti vertiginous, nutritional support.
2. Mastoidectomy to eradicate the ear pathology.
3. Labyrinthectomy: to drain the infected inner ear.
4. Rehabilitation for the resulting hearing loss by hearing aids, or cochlear implantation.
d. Otitic Facial Paralysis

It can occur in the course of otitis media in the following situations:

1. Acute suppurative otitis media if the fallopian canal is dehiscent.
2. Acute mastoiditis if osteitis affects the retrofacial cells.
3. Chronic suppurative otitis media with bone eroding conditions as granulations and cholesteatoma.
4. As a complication of surgery. (see facial nerve paralysis)

2-Intra-Cranial Complications

a. Extradural Abscess:

- Collection of pus between the dura, and the eroded temporal bone. It commonly occurs in the areas of the tegmen tympani, sinodural angle, or over the lateral sinus. The abscess is usually small and localized.

Symptoms:

- Usually symptomless, and is an incidental finding during surgery for chronic suppurative otitis media.
- Low-grade fever. Persistant earache and headache with persistant discharge.

Signs:

- Chronic suppurative otitis media with pulsating discharge (Transmitted pulsations from the exposed dura).

Investigations:

- CT is diagnostic, locates the abscess & detects other complications.

Treatment:

- Surgical drainage, usually through radical mastoidectomy.

b. Subdural Empyema

It is the least common form of complicated otitis media. It results from collection of big amounts of pus (empyema) in the capacious subdural space, causing rapid mass effect and increase in the intracranial tension. The patient presents with a downhill course with convulsions, stupor, coma and death.

Investigations:

- CT is urgent, and can define the size of the collection.

Treatment:

- Is urgent, with trephine and drainage of the pus (by the neurosurgeon) and radical mastoidectomy.
- Large doses of effective antibiotics are required.
c. Lateral Sinus Thrombosis or Thrombophlebitis

Pathogenesis:

The infection spreads through the wall of the sinus causing a mural thrombus, which enlarges to occlude the sinus (thrombosis) and gets infected (thrombophlebitis).

Clinical picture:

Clinical manifestations will be the result of the propagating thrombus or the septic emboli of the disintegrated infected thrombus.

1. Manifestations of the occlusion by the propagating thrombus:
   a. Down spread to the internal jugular vein, with tender cord like vein, palpated in the carotid triangle in the neck.
   b. Upward spread to the superior sagittal sinus interfering with drainage of the CSF leads to increased intracranial tension (Otitic hydrocephalus) causes severe headache, projectile vomiting (without nausea and not related to meals) and blurred vision. Papilloedema (oedema of the optic disc) occurs in neglected cases (after 1-3 weeks).
   c. Forward spread via the superior petrosal sinus to the cavernous sinus leads to unilateral chemosis, ptosis, proptosis, papilloedema, and ophthalmoplegia.
   d. Lateral spread to the mastoid emissary vein causes tender postauricular oedema (Griesinger's sign).

2. Manifestations of thrombophlebitis disintegration:
   a. Fever, usually hectic or intermittent with irregular intervals, between which the patient is relatively well. Rarely the fever is continuous or masked by the use of antibiotics.
   b. Rigors, due to septic emboli.
   c. Signs of meningism with headache, neck rigidity, and nystagmus may be present

Investigations:

1. Blood picture shows leucocytosis.
2. Positive culture for bacteria if samples taken during rigors.
3. CT: failure to visualize the contrast in the lumen of the lateral sinus is diagnostic.
4. Tests of historical interest, are: -
   a. Queckenstedt's test or Tobey- Ayer's test: pressure on the contra lateral internal jugular vein will cause rise of CSF pressure in lumbar puncture manometer. Pressure on the affected internal jugular vein will not affect the pressure in the manometer.
   b. Crowe test: - pressure on the healthy internal jugular vein → dilatation of the retinal veins (detected by ophthalmoscope).
Differential diagnosis:
1. Malaria, though fever is hectic or intermittent, the intervals are usually regular. There is leucoponia and the patient is ill between the attacks. Malaria parasites can be seen in nocturnal blood samples.
2. Typhoid: - the fever is continuous, it shows a stepladder pattern. No rigors but severe frontal headache with constipation. Spleen is usually enlarged. Blood tests reveal leucopoenia and positive Widal test.

Treatment:
1. Antibiotics, have largely changed the course of the disease, and made the surgery less radical.
2. Radical mastoidectomy, and exploration of the lateral sinus: The diseased sinus is dull, covered with granulations, noncompressible, with transmitted pulsations, and no fresh blood could be aspirated. The thrombosed sinus is incised and the thrombus is removed until free blood flow is seen proximally and distally.
3. Proper heparinization and postoperative follow up.

d. Otogenic Meningitis

Meningitis is an inflammation of the pia-arachinoid with infection of the CSF.

Clinical manifestations:
1. Systemic manifestations of infection: - severe fever and toxemia (toxic earthy look, anerxia and rapid pulse).
2. Manifestations of meningeal irritation: -
   a. Irritability, photophobia, neck rigidity, retracted head and opisotonus position.
   c. Brudzinski's sign positive: - neck flexion causes flexion of the hips and knees.
3. Manifestations of increased intracranial tension: -
   a. Severe generalized headache.
   b. Projectile vomiting which is not related to meals, not preceded by nausea.
   c. Blurred vision with papilloedema.
   d. Drowsiness, stupor, and coma in terminal cases.

Investigations:
1. CT scan: - outlines the ear pathology and detects other complications.
2. Lumbar puncture for CSF analysis and culture.

Complications: -
1. Sensory- neural hearing loss: - is due to labyrinthitis complicating meningitis.
2. Persistent squint, due to affection of the 6th nerve by the basal meningitis.
3. Persistent nystagmus, due to affection of the vestibulo-ocular pathways.
4. Benign inracranial hypertension (Otitic hydrocephalus).
Treatment:
Should be urgent as the condition is fatal.

1. Medical:-
   a. Antibiotics, parenteral, massive, broad-spectrum, and cross the blood brain barrier, as 3rd generation cephalosporins, ciprofloxacins, metronidazoles, sulfatrimethoprim.
   b. Reduce the high intracranial pressure by i.v. Mannitol, glucose 25%, lasix, or repeated lumbar puncture. Intra-thecal penicillin may be given if puncture is performed.

2. Surgery: mastoidectomy after control of the general condition.

3. Rehabilitation of the sequelae of meningitis e.g. deafness treated by hearing aid or cochlear implantation

e. Otogenic Brain Abscess Fig. (45):
It is the result of encephalitis with collection of pus in the brain parenchyma. It either collects in the temporal lobe or the cerebellum.

Fig.(35) Otogenic Brain Abscess

Clinico- pathological stages:
1. Stage of encephalitis with localized oedema and congestion in the particular site. Clinically the patient will have fever, rigors, generalized headache, nausea, and vomiting. He is irritable, and soon becomes drowsy and comatose.

2. Stage of localization and abscess formation:
   a. acute abscess: due to focal suppuration and necrosis in the area of encephalitis
   b. Chronic abscess: if not drained, gliosis will form the wall of the cavity. Clinically the patient general symptoms improve (latent stage) for a variable period.

3. Stage of enlargement: - (Manifest stage): the abscess enlarges destroying nearby areas of the brain parenchyma adding more dysfunction and increasing the intracranial tension. Clinically the patient will show the localizing according to the site of abscess.
Temporal lobe abscess:
- Nominal aphasia: inability to name objects and persons due to affection of speech areas, especially in the dominant hemisphere.
- Homonymous hemianopia: due to affection of the optic radiation.
- Uncinate fits: hallucination of smell and taste due to pressure on the uncinate gyrus.
- Contralateral hemiplegia and hemianaesthesia: according to the size of the abscess, due to affection of pre and post-central gyri respectively.

Cerebellar lobe abscess:
- Ipsilateral hypotonia and muscle weakness.
- Intension tremors.
- Incordination: dysmetria on finger to nose test.

4. Terminal stage with the enlarging abscess approaching one of the ventricles rupturing in it causes fatal meningioencephalitis. Leads to coma and death.

Investigations:
CT & MRI are diagnostic for otogenic brain abscess.

Treatment:
The team will include beside ear surgeons, neurologists and neurosurgeons. The patient is managed on intensive care basis.
1. Neurosurgeons: - drain the acute abscess or excise the chronic abscess.
2. Ear surgeons: - treat source of infection through the mastoidectomy few days after the neuro-surgical procedure to avoid recollection.
3. Rehabilitation for the resultant deficits.

Tumours of the Middle Ear

1-Benign tumours:
- Glomus tumours of the ear are the commonest. They are locally destructive and occur in two sites in the middle ear:
  a. Glomus tympanicum.
  b. Glomus jugulare.
Treatment: - is wide local excision by a team including beside the ear surgeon, the neurosurgeons, vascular surgeons, and neck surgeons.
  Radiation is palliative alternative.
2- Malignant tumours: -
- Squamous cell carcinoma.
Treatment is wide local excision and chemotherapy.
Some diseases of the Inner Ear and 8th Nerve

1) Presbyacusis:
Definition:
- Bilateral sensorineural hearing loss associated with tinnitus and decreased discrimination at the age of 60 years. It is the commonest cause of sensori-neural hearing loss, since it would affect every person. Most people will suffer the condition by the age of 60 years; however, it may start in 40's in some people. The patients complain of hearing loss; however tinnitus may be the troubling symptom.

2) Benign Paroxysmal Positional Vertigo (BPPV):
- It is caused by cupulolithiasis (calcifications in the cupula) of the posterior semi-circular canal. The patient is usually above 40 years old. History of old head trauma or recent viral infection is usually present. The patient experiences sudden attacks of rotational vertigo on moving the head, usually posteriorly. The attack lasts for seconds, and is fatigable. In some patients the attacks incapacitate the patient and interfere with his daily activities. Mild cases, may improve with the use of labyrinthine sedatives, as Dramamine. Severe cases might require positional therapy. Singular neurectomy is an alternative.

3) Meniere's disease (endolymphatic hydrops) Fig. (36):
Definition:
- Paroxysmal attacks of vertigo, tinnitus, perceptive deafness, and aural fullness, due to increased volume of the endolymph (Endolymphatic hydrops).

Fig.(36): Endolymphatic hydrops

Causes:
- The aetiology is unknown. Many theories to explain the hydrops have been provided. It may be due to two mechanisms:
  - Hypoabsorption of the endolymph due to obstructed drainage, in the endolymphatic duct and sac. This can occur due to infection or trauma.
  - Hypersecretion of the endolymph from the stria vascularis. The following theories have been proposed:
    • Vascular ischemia, due to affection of the microcirculation of the stria vascularis.
• Local disturbance of salt and water mechanisms.
• Local allergy in the inner ear.
• Autonomic imbalance in the inner ear.
• Autoimmune mechanisms.
• Genetic factors.

Symptoms:
1. Vertigo: is sudden, severe, and recurrent. It takes the form of true rotation that persists for few to several minutes or hours. It may be associated with nausea and vomiting.
2. Tinnitus: may precede or accompany the vertigo.
3. Hearing loss: is of the sensory type. It is fluctuant, reversible and affects low frequencies early in the disease. In late stages, all frequencies are permanently affected. Patients do not tolerate loud sounds and may report diplacusis (different pitches in the two ears).
4. Aural fullness: is a sense of pressure in both ears that usually precedes the vertigo.

Diagnosis:
1. History is highly suggestive for diagnosis in most cases.
2. Ear drum examination is normal.
3. Audiological evaluation shows unilateral mild low tone loss in early stages. In late stages, all tones are affected.
4. Electrocochleography is diagnostic.

Treatment:
I- Medical treatment:
1. Diet: salt restriction
2. Anti-vertigous and labyrinthine sedatives as dramamine and cinnerzine.
3. Vasodilators (ischemia theory) as betahistine (oral histamine).
4. Diuretics (salt and water retention theory).
5. Corticosteroids (autoimmunity & allergy).
6. Minor tranquilizers.

II- Intra tympanic injection of ototoxic drugs.

III- Surgery: is indicated when medical treatment failed. The type of surgery will depend on the level of hearing as follows:
1. When hearing is serviceable.
   a. Decompression of the Endolymphatic sac or subarachnoid shunt.
   b. Unilateral vestibular neurectomy.
2. If hearing is not serviceable:
   a. Labyrinthectomy and destruction of the inner ear.
   b. Chemical destruction of the inner ear by local application of gentamicin in unilateral cases or systemic use of streptomycin in bilateral cases.
Facial Nerve Paralysis

Anatomy: Fig. (37)
The facial nerve possesses a motor and a sensory root (nervus intermedius). The motor root carries fibers to the innervated muscles and secretomotor fibers to the innervated glands. The sensory root conveys taste sensation.

![Facial Nerve Diagram](image)

**Course of the facial nerve:**

1. Intracranial course:
The facial nerve nucleus lies in the pons which is supplied by Pyramidal fibers from motor area 4 for voluntary movements. The upper part of the nucleus is supplied from both pyramidal tracts of the brain i.e. bilaterally represented while the lower part is supplied only from the opposite pyramidal tract. Extrapyramidal fibers from the hypothalamus for involuntary movements of the face. The nerve emerges from the lower border of the pons. Then the nerve passes with the 8th cranial nerve into the internal auditory canal for a distance of 1 cm to enter its bony fallopian canal.

2. Cranial course:
   Divided into four segments:
   a. Meatal segment: The nerve passes laterally with the 8th nerve in IAM. Then it enters the Facial canal.
   b. Labyrinthine segment: The nerve passes laterally above the labyrinth to the geniculate ganglion, where it gives the greater superficial petrosal nerve.
   c. Tympanic (horizontal) segment: At the geniculate ganglion, the nerve...
turns (1st genu) and runs horizontally backwards along the medial wall of tympanic cavity till the posterior wall of tympanic cavity.

d. Mastoid segment: It turns as 2nd genu and runs vertically downwards behind the tympanic cavity, giving nerve to stapedius and chorda tympani nerve. Then it emerges from the skull through the stylomastoid foramen.

3. Extracranial course:
The nerve leaves the skull through the stylomastoid foramen, passes between the superficial and deep lobes of the parotid gland and divides into its terminal branches that innervate the face muscles.

**Branches of the Facial Nerve: - Fig. (37)**

- a. The greater superficial petrosal nerve: it contains secretomotor fibers to the lacrimal glands and nasal mucosa.
- b. The nerve to stapedius: motor nerve to stapedius muscle.
- c. Chorda tympani nerve: arises in the vertical part, it contains: Secretomotor fibers to the submandibular and sublingual salivary glands and afferent taste fibers from the anterior 2/3 of the tongue.
- d. Muscular branches: After the nerve comes out from the stylomastoid foramen, it gives nerves to muscles of expression, muscles of the auricle, occipitofrontalis, stylohyoid and posterior belly of digastric.
- e. Small sensory twigs: to the skin of the external canal and concha.

**Aetiology of Facial Nerve Paralysis:**

A. Upper motor neurone lesions (UMNL): *Central lesions.*

2. Encephalitis, brain abscess.

B. Lower motor neurone lesions (LMNL): *Peripheral lesions.*

1. Intracranial:
   - Pons: haemorrhage, poliomyelitis, multiple sclerosis and tumours.
   - Cerebello-pontine angle CPA: acoustic neuroma, meningioma, arachnoid cyst and congenital cholesteatoma.
2. Cranial (intra-temporal):
   - Idiopathic as Bell's palsy (the commonest cause).
   - Traumatic as skull base surgery and fracture temporal bone.
   - Inflammatory as otitic facial paralysis due to acute and chronic suppurative otitis media and viral as herps zoster oticus (Ramsy Hunt syndrome).
   - Neoplastic as middle ear cancer or glomus jugular tumour and facial nerve fibroma.
3. Extracranial:
   - Parotid gland tumour and its surgery.
   - Cut wounds of the face at the parotid region.
   - Forceps of delivery and birth injuries.
- Postauricular incision in infants due to under developed mastoid process and superficial nerve.

The commonest causes of facial nerve paralysis:
1. Bell's palsy (Idiopathic facial paralysis).
2. Traumatic facial paralysis.
3. Herpes zoster oticus.
4. Otitic facial paralysis as a complication of suppurative otitis media.

Clinical Picture: - Fig. (38):
- Complete paralysis of the facial nerve causes face asymmetry at rest.
- There is inability to raise the eye brow and loss of frontal corrugations.
- Inability to close the eye.
- Inability to whistle and collection of food between cheek and teeth.
- The angle of the mouth sags on the affected side with loss of nasolabial fold.
- The mouth is pulled to the active side on smiling or showing the teeth.
- Tears overflow and saliva may dribble out on the paralyzed side.

Fig. (38): Right LMN facial nerve paralysis.

Fig. (39): Topography of the facial nerve paralysis
Diagnosis and evaluation:
Careful history taking, through examination and proper investigations for proper diagnosis.

I- Level of the lesion (Topographic diagnosis) Fig. (39):
β Lower motor neurone lesion (LMNL) or upper motor neurone lesion (UMNL):

<table>
<thead>
<tr>
<th></th>
<th>LMNL</th>
<th>UMNL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Level of lesion:</td>
<td>Nuclear or infranuclear.</td>
<td>Supranuclear.</td>
</tr>
<tr>
<td>Upper and lower halves of the face are paralysed.</td>
<td>Only the lower 1/2 of the face is paralysed (upper half being supplied from both pyramidal tracts).</td>
<td></td>
</tr>
<tr>
<td>Loss of both voluntary and involuntary (emotional) movements.</td>
<td>Loss of only voluntary with intact involuntary movements (e.g. smiling) (controlled by extrapyramidal tracts).</td>
<td></td>
</tr>
<tr>
<td>Flaccid paralysis (hypotonia). Muscle atrophy and twiches.</td>
<td>Spastic paralysis (hypertonia). No muscle atrophy or twiches.</td>
<td></td>
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</tbody>
</table>

II- Topographical diagnosis: of LMNL (Localization of the level). Fig.(40)
Total facial paralysis (upper and lower halves of the face) is associated with:
1. At the geniculate ganglion:
   β Loss of lacrimation.
   β Loss of stapedial reflex and Hyperacusis.
   β Loss of taste on the anterior 2/3 of the tongue.
   β Loss of salivation from the submandibular salivary gland.
2. Tympanic segment above the nerve to stapedius:
   β Loss of stapedial reflex and Hyperacusis.
   β Loss of taste on the anterior 2/3 of the tongue.
   β Loss of salivation from the submandibular salivary gland.
3. Mastoid segment above the chorda tympani:
   β Loss of taste on the anterior 2/3 of the tongue.
   β Loss of salivation from the submandibular salivary gland.
4. Mastoid segment below the chorda tympani (at the stylomastoid foramen):
   β Lacrimation, stapedial reflex, taste and salivation are intact.
5. Extra cranial part:
   β Lacrimation, stapedial reflex, taste and salivation are intact.

III-Aetiological diagnosis:
a. Examination of the ear: may show manifestations of acute or chronic suppurative otitis media, Herpes zoster oticus, malignant otitis externa, glomus tumour or carcinoma.
b. Examination of the head and neck: may show parotid swelling.
Investigations:
A. For localization of the level of LMNL:
   1. Schirmer's test (for lacrimation): Fig. (40). Compare the lacrimal flow on both sides. A strip of absorbent paper is inserted on both lower eye lids for 5 minutes. Then the length of the moistened segment of paper is measured on the paralysed and normal sides. It is significant when the difference between the lacrimal flow on both sides exceeds 30% of the total bilateral lacrimation, indicating a lesion at or proximal to the geniculate ganglion.

   ![Fig. (40): Schirmer's test](image)

   2. Stapedial reflex (for stapedius muscle): Lost in any lesion proximal to the nerve to stapedius.
   3. Taste sensation tests:
      a. Qualitative: compare taste of salt, sugar, sour and bitter applied to lateral edge of the anterior 2/3 of the tongue
      b. Quantitative: electrogustometry
   4. Salivary flow rate test: compares the amount of saliva by cannulation of submandibular ducts in both sides.

B. To detect the cause:
   1. Pure tone audiometry for hearing test.
   2. CT scan and MRI of the temporal bone and brain.

C. Prognostic tests: Electrodiagnostic tests:
Types:
   1. Nerve excitability test: valid after 3 days.
      Determine the minimal electric current in milliamperes required to produce just visible contraction and compare both sides.
   2. Electroneuronography (ENOG): valid after 3 days.
      Record the evoked action potential of the muscles when stimulated by maximal stimulation of the nerve and compare both sides.
   3. Electromyography (EMG):
      Record the spontaneous non-evoked electric potential of the tested muscles (at rest and during active movement) without stimulation of the facial nerve.

Treatment of facial paralysis:
1. General conservative measures:-
   a. Care of the eye: The eye is at risk due to lack of blinking, incomplete closure, decreased lacrimation and corneal exposure. Eye drops and eye glasses by the day, eye ointment by night. Tarsorrhaphy (surgical closure of the eye lids).
b. Care of the muscles: The muscles are at risk of atrophy and fibrosis; so will need massage, active muscle exercise and physiotherapy.

2. Treatment of the cause, e.g.
   - Myringotomy and antibiotics in ASOM
   - Steroids and antiviral drugs in Bell's palsy and herpes zoster.
   - Exploration and decompression end to end anastomosis, or grafting of the nerve in traumatic facial paralysis.

3. Facial rehabilitation (for irreversible cases): Plastic surgery; to improve the appearance.

**Bell's palsy (Idiopathic facial paralysis)**

It is the commonest cause of facial nerve paralysis. It is an idiopathic sudden paralysis of the facial nerve (LMNL).

**Aetiology:**
- Unknown.
- Viral infection in the geniculate ganglion (the most accepted).
- Vascular spasm of vasa nervosa of the 7th nerve after exposure to cold air current.
- Autoimmune disease.

**Pathology:**
The paralysis is due to oedema of the facial nerve inside the bony canal, causing nerve compression and ischaemia.

**Clinical picture:**
1. Acute onset usually after exposure to cold air current or viral prodorma.
2. Usually unilateral
3. Hyperacusis, may be present
4. Pain around the ear, may be present.

**Prognosis:**
Good with spontaneous recovery in most cases.

**Treatment:**
1. Medical: Steroids, antiviral therapy (acyclovir), vasodilators.
2. General conservative measures: mentioned.
3. Surgical: Facial nerve decompression by splitting the nerve sheath. It is performed by trans-mastoid or middle cranial fossa approach.
   - Indication: If ENoG showing 90% nerve degeneration within 2 weeks.

**Otosclerosis**

It is a disease of the bony labyrinth (otic capsule). It is characterized by formation of a new vascularised spongy bone in a localized otosclerotic focus replacing the normal compact bone of the bony labyrinth resulting in hearing loss which is commonly bilateral. It is progressive fixation of the foot plate of stapes and it may invade the cochlea.
Aetiology:
1. Unknown.
2. Heredofamilial (Genetic).
3. Developmental Theory: otosclerotic focus replacing the cartilaginous cell rests of the otic capsule.
4. Endocrinal as it is more common in family and it is aggrevated by pregnancy.
5. Enzymatic Theory: the focus is formed by the action of lysosomal hydrolase released from histocytes and osteoclasts.

Types of otosclerosis:
1. Stapedial otosclerosis (commonest): causes CHL.
2. Cochlear otosclerosis (very rare): causes pure SNHL.
3. Mixed or combined otosclerosis: cause mixed HL

Incidence:
- Otosclerosis is more common in females.
- The age of onset is usually between 20-40 years.
- Family history is positive in 50% of cases.
- The commonest cause of bilateral CHL in adults.

Symptoms:
1. Hearing loss, usually bilateral and, progressive.
2. Tinnitus.
3. Paracusis Willshii (i.e. hearing is better in noisy areas than quite due to the loud voices used against the background noise).

Signs:
1. The tympanic membrane is normal in shape and mobility.
2. Schwartz sign (Rare): flamingo red tinge seen behind the tympanic membrane due to active vascular bone on the promontory.
3. Bilateral C.H.L. usually, but rarely it may be pure S.N.H.L. or mixed.

Differential Diagnosis:
From other causes of CHL with intact drum
- Ossicular fixation due to congenital or inflammatory causes.
- Otitis media with effusion
- Middle ear atelectasis
- Adhesive otitis media
- Tympanosclerosis
- Ossicular disconnection

Investigations:
1. Pure tone audiometry differ according to the type of otosclerosis: - Stapedial (CHL), cochlear (SNHL) or combined (Mixed HL).
2. Tympanometry (type As) in Stapedial otosclerosis.
Prosthesis connects long process of incus to the hole made in footplate of stapes

Fig. (41): Otosclerosis and stapedectomy.

Treatment:
1. Stapedectomy Fig. (41): Surgical removal of the stapes and then replaced by an artificial prosthesis as teflon piston or fat and wire. This operation is indicated for stapedial and combined otosclerosis.
2. Hearing aids: if surgery is refused or contraindicated and in cochlear otosclerosis.
3. Medical Treatment: as sodium fluoride for active and cochlear otosclerosis.

Symptomatology of Ear Diseases

Diseases of the ear express themselves in the following symptoms:-
1. Hearing loss (Deafness).
2. Tinnitus.
3. Otorrhoea (Ear discharge).
4. Vertigo.
5. Earache (Otalgia).

1. Deafness (hearing loss)

Definition:
It is diminution of hearing and there are different types of hearing loss: -
- Conductive hearing loss: It results from fault in conduction apparatus (the external and middle ears) while the inner ear is healthy. It is frequently correctable by medical or surgical treatment.
- Sensorineural hearing loss: It results from abnormalities in the cochlea or the 8th nerve. It is permanent and is rarely correctable.
- Mixed hearing loss: It usually starts as conductive deafness and ends by a mixture of conductive and sensorineural deafness.
- Functional "psychogenic or malingering".

A. Causes of conductive hearing loss:
   a) External canal
      - Wax accumulation in the external auditory canal with complete obstruction.
      - Congenital atresia.
      - FB impaction.
      - Otitis externa, large furuncle, or fungus mass causes obstruction.
b) Tumours e.g. exostosis and cancer.

b) Middle ear
   - Congenital anomalies.
   - Drum: perforations, adhesion, bullous myringitis, or tympanosclerosis.
   - Tympanic cavity:
     - Inflammation:- Acute and chronic SOM, Non-SOM (secretory & adhesive OM and atelactatic ME)
     - Trauma:- Hemotympanum (Fracture base), and Otitic barotraumas.
     - Tumours:- Glomus and carcinoma.

c) Congenital anomalies.
   - Drum: perforations, adhesion, bullous myringitis, or tympanosclerosis.
   - Tympanic cavity:
     - Congenital absence or fixation.
     - Traumatic dislocation.
     - Inflammatory necrosis or adhesion.

d) Ossicles:
   - Otosclerosis.
   - Congenital absence or fixation.
   - Traumatic dislocation.
   - Inflammatory necrosis or adhesion.

c) Eustachian tube
   - Mechanical obstruction by adenoids or nasopharyngeal tumours.
   - Inflammation e.g. Eustachian catarrh causing oedema.
   - Cleft palate.

B. Causes of sensorineural hearing loss

I. Cochlear lesions
   a) Congenital
      - Heridity: Consanguinity.
      - Prenatal:
        - TORCHES (Toxoplasma, Rubella, CMV, Herpes, Syphilis) affecting the mother during the first 3 months of pregnancy, or intake of ototoxic drugs by mother.
      - Natal: Birth injury, or Neonatal anoxia
      - Postnatal: Kernicterus (Neonatal jaundice) due to RH incompatibility.
   b) Trauma
      - Fracture base of skull, Acoustic trauma acute or chronic.
   c) Inflammation:
      - Viral measles, mumps and influenza, bacterial meningitis, typhoid, T.B., syphilis or complications of C.S.O.M.
   d) Toxic:
      - Endogenous: Diabetes mellitus or Hypothyroidism.
      - Exogenous: Ototoxic drugs.
   e) Vascular spasm, haemorrhage or thrombosis of the internal auditory artery.
   f) Miscellaneous:
      - Meniere's disease.
      - Senile deafness (presbyacusis).
      - Cochlear otosclerosis.
      - Bone disease

II. Cochlear nerve lesions: Tumours as acoustic neuroma.
III. Brain lesions:
- Vascular vertebrobasilar insufficiency, Tumours, or Multiple scleroses.

C. Mixed Hearing Loss
Aetiology:
- Congenital meatal atresia with inner ear anomaly.
- Combined longitudinal and transverse temporal bone fracture.
- CSOM with labyrinthitis.
- Combined stapedial and cochlear otosclerosis.

N.B.
- The most common cause of CHL is wax accumulation.
- The most common cause of progressive CHL in adults age is otosclerosis.
- The most common cause of deafness in children is secretory otitis media.
- The most common cause of deafness in elderly people is presbyacusis.

Deaf Mutism:
Definition: Loss of both hearing and speech
Aetiology: due to severe deafness in the first 2 years of life before the infant acquires the speech.
1) Congenital causes: The most important are:
   - Congenital anomalies of the inner ear.
   - TORCHES (Toxoplasma, Rubella, CMV, Herpes, Syphilis).
   - Kernicterus (Neonatal Jundice).
2) Acquired causes:
   - Meningitis with secondary labyrinthitis, measles or trauma during birth.
Treatment:
   a. Auditory rehabilitation e.g. lip reading, hearing aids.
   b. Cochlear implantation.

2. Tinnitus
Definition: Sensation of noise in the ear or head. It may be continuous or intermittent noise e.g. ringing, hissing or pulsating sound without external sound stimuli.
Types and causes:
A. Subjective tinnitus:
   - The patient hears the tinnitus which cannot be heared by the examining physician.
   a) Tinnitus with hearing loss:
      1. With CHL: It is low pitched and intermittent and is due to obstruction in the external or lesion of the middle ear.
      2. With SNHL: It is high pitched, continous and persistent and is due to cochlear or retrocochlear causes of deafness.
   b) Tinnitus without hearing loss:
      - Transmitted sounds from the carotid artery or jugular vein to the ear
due to bone deficiency between them e.g.
1. Aberrant carotid artery or persistent stapedial artery.
2. Blood changes: Low blood pressure, high blood pressure with atherosclerosis or high cardiac output in anemia, fever, thyrotoxicosis, pregnancy, or migraine.

Patulous E.T.

B. Objective tinnitus:

The sound can be heard by the patient and the examiner as well due to:
1) Muscular: Palatal myoclonus or tensor tympani syndrome "spasm"
2) Vascular:
   a. Aneurysms of the internal carotid artery or vascular intracranial tumours.
   b. Arteriovenous malformation between the occipital artery and transverse sinus.

Treatment:
1. Treatment of the cause.
2. Medications e.g car bamazine
3. Rehabilitative:
   - Tinnitus maskers.
   - Biofeedback training to modify patient's reaction to tinnitus.
   - Hearing aids if there is significant hearing loss.

3. Otorrhoea (Ear discharge)
The causes differ according to the type of discharge:
a) Purulent discharge:
   - Furunculosis and acute otitis externa.
   - Cholesteatoma (offensive discharge due to A.A.CSOM type).
b) Mucoid or mucopurulent:
   - Acute and chronic suppurative otitis media (T.T.CSOM type).
c) Bloody:
   - Trauma to: External ear, drum, fracture temporal bone.
   - Inflammation:
     - Myringitis bollosa heamorrhagica.
     - Acute suppurative otitis media at time of T.M. perforation.
     - Chronic suppurative otitis media with granulations.
     - Otitis externa with granulations.
   - Tumours
     - Glomus tumours or carcinoma of the middle ear or carcinoma of the external ear.
d) Watery (CSF) otorrhoea:
   - Fracture temporal bone.
   - Intraoperatively if the dura is injured.
   - Spontaneous CSF otorrhoea.
4. **Vertigo**

Definition: subjective sensation of disturbed relationship between the individual and his environment in which either the patient or his environment is moving. It is commonly rotatory, but sensations such as swaying or rocking may be considered as vertigo. It is usually of vestibular origin.

Aetiology:

Vertigo may result from peripheral lesions involving the vestibular sense organ or from central lesions involving the vestibular pathways and centres.

1) peripheral (vestibular labyrinth) causes:
   a) Idiopathic:
      - Meniere's disease.
      - Benign paroxysmal positional vertigo (BPPV).
      - Motion sickness
   b) Traumatic:
      - Temporal bone fracture.
      - Operative trauma e.g. stapedectomy.
      - Labyrinthine membrane rupture.
      - Concussion of brain.
      - Whiplash injury of the neck.
   c) Vascular: Vascular insufficiency, hemorrhage or thrombosis.
   d) Toxic: Streptomycin (ototoxic) or ureamia.
   e) Inflammatory: Labyrinthitis.
   f) Neoplastic: Acoustic neuroma.

2) Vestibular nerve:-
   - Vestibular neuritis.
   - Cerebello-pontine angle lesions i.e acoustic neuroma.

3) Central (brain stem and brain) causes:
   a) Vascular: Vertebrobasilar insufficiency, basilar artery migraine.
   b) Traumatic: Brain concussion.
   c) Inflammatory: Brain abscess, or encephalitis.
   d) Idiopathic: Syringiobulbia or multiple sclerosis.

Vertigo without Hearing Loss:

- Benign paroxysmal positional vertigo.
- Vestibular neuritis.
- Vertibro-basilar insufficiency.
- Central causes of vertigo.
- General causes e.g. hypotension.

Differentiate vertigo from:

- Syncope which means feeling of going to faint. It is usually due to hypotension.
- Dizziness or unsteadiness which means subjective sensation of lost equilibrium in relation to environment. The aetiology could be cerebral, cerebellar, posterior column or vestibular.
Treatment:
- Treatment of the cause.
- Anti-vertiginous drugs as cinnerazine.
- Rehabilitation vestibular exercises which build up the compensation mechanisms of the brain.

5. Earache "Otalgia"

Definition: Pain in the ear.

Causes:
A. Local causes
   i) External ear diseases:
      - Perichondritis, impacted F.B., diffuse otitis externa, furunculosis, myringitis bullosa, malignant otitis externa, herpes zoster oticus and neoplasm (carcinoma).
   ii) Middle ear disease:
      - Acute otitis media.
      - Complicated suppurative otitis media e.g. mastoiditis, petrositis and intracranial complications.
      - Malignant disease.
B. Referred otalgia: from diseased organs sharing the same nerve supply of the ear as following.
   i) The 5th nerve "the trigeminal":
      a. Lesions in the nose and sinuses
         - Sinusitis "especially sphenoidal and maxillary"
         - Pressure on the middle turbinate from septal spur or high septal deviation.
      b. Lesions in the teeth and jaw
         - Carious tooth
         - Impacted wisdom tooth
         - Osteoarthritis of the temporomandibular joint
      c. Lesions in the nasopharynx
         - Post adenoidectomy
         - Neoplasm of the nasopharynx.
      d. Lesions in the salivary glands or ducts
         - Acute sialadenitis and calculus
      e. Trigeminal neuralgia.
   ii) The 2nd and 3rd cervical nerves:
      - Cervical disc lesions.
      - Arthritis of the cervical spine.
   iii) The 9th nerve "Jacobson's nerve of the glossopharyngeal":
      a. Lesions in the tongue:
         - Ulceration especially herpes.
         - Neoplasm.
      b. Lesions in the oropharynx:
         - Acute pharyngitis and tonsillitis.
         - Post tonsillectomy.
Peritonsillar abscess.
Parapharyngeal and retropharyngeal abscess.
Tuberculous ulceration.
Neoplasms.
c. Elongated styloid process stretching the glosopharyngeal nerve.
d. Glossopharyngeal neuralgia.
iv) The 10th nerve "Arnold's nerve of the vagus":
Lesions in the larynx or hypopharynx:
Neoplasms especially of the pyriform fossa and post cricoid region.
Cancer larynx.
Tuberculosis of the larynx.
Anatomy of the Nose

- The nose is divided into external nose and two internal nasal cavities.

**External nose:** Fig. (1) is pyramidal in shape and is formed of:
- The upper third of the external nose is bony and consists of:
  - The nasal bones which unite with each other in the midline.
  - The nasal process of the frontal bone.
  - The frontal process of the maxilla.
- The inferior two thirds are cartilaginous and consist of the upper lateral cartilage, lower lateral cartilages and alar cartilages. The skin over the cartilaginous part is closely adherent and contains multiple sebaceous glands.

![Fig. (1) The external nasal skeleton.](image)

**Nasal cavities:** Each nasal cavity Fig. (2) starts from the nostrils (anterior nares) to the posterior nares (choanae), which lead to the nasopharynx. The two internal cavities, right and left, are separated by the basal septum.

![Fig. (2) The nasal cavity and nasopharynx](image)
• The roof of the nose is formed by parts of frontal bone, ethmoid roof & sphenoid bone separating it from the anterior cranial fossa.
• The floor constitutes the hard palate, and is formed by the horizontal plates of the maxillary and palatine bones.
• The lateral wall of the nose supports three main ridges called the superior, middle and inferior turbinates (conchae). Beneath each concha there is a space, called superior, middle and inferior meatus respectively. The space between the superior concha and the nasal septum is called the sphenethmoidal recess. The sphenoid sinus opens into this recess and the posterior ethmoidal air cells open into the superior meatus. The nasolacrimal duct opens into the inferior meatus. The remaining paranasal sinuses drain into the middle meatus.
• The medial wall (nasal septum) Fig. (3) Consists of a bony part (perpendicular plate of ethmoid and vomer) and a cartilaginous part (septal or quadrilateral cartilage). Inferiorly it is inserted into a groove in the maxillary and palatine crest. The septum is covered with mucoperichondrium and mucoperiostium over the cartilage and bone, respectively.

The nasal cavity is divided into three areas, vestibular, olfactory and respiratory. The vestibular area is the region just inside the nostril, lined by skin. The olfactory area contains the olfactory epithelium and occupies the roof and the uppermost parts of the septum and the lateral wall, above the superior concha. The respiratory area constitutes the rest of the nasal cavity and is lined by respiratory pseudostratified columnar ciliated epithelium.

**Blood supply:** Fig. (4)
Both the internal and external carotid arteries supply the nose via their terminal branches. The region above the root of the middle turbinate is supplied by the anterior and posterior ethmoidal arteries, these are branches of the ophthalmic artery (internal carotid), with the remaining areas being supplied by
the sphenopalatine, greater palatine and superior labial branch of facial artery (external carotid). The sphenopalatine artery is considered to be the main arterial supply of the nasal cavity and originates from the maxillary artery. The nasal arteries anastomose at the antero-inferior region of the septum which is called Little's area or (Kiesselbach's plexus). This anastomosis includes the anterior ethmoidal, sphenopalatine, greater palatine and superior labial branch of facial artery.

![Arterial supply of the nose](image)

Fig. (4) Arterial supply of the nose.

• Venous drainage of the nose is of importance, as blood is drained via the pterygoid plexus, facial and ophthalmic veins to the cavernous sinus.

• Surgical importance: These veins are connected to the cavernous sinus. Therefore, infection in the nose may lead to cavernous sinus thrombosis. Therefore the area bounded by the root of nose and the angles of mouth is called the dangerous area of the face.

Nerve supply:
The olfactory area is supplied by the 1st cranial nerve (olfactory nerve). The main sensory supply is via the maxillary division of the trigeminal nerve. Secretory glands are under control of the autonomic nervous system by the vidian nerve. The nasal vascular supply is constricted by sympathetic nerve stimulation and dilated by parasympathetic one.

Lymphatic drainage:
The anterior part of the nose drains with the external nose to the submandibular nodes, while the posterior drainage is to the retropharyngeal and upper deep cervical lymph nodes.

Anatomy of the paranasal Sinuses

The paranasal sinuses Fig. (5) are extensions of the nasal cavity as air-filled spaces lined with pseudostratified columnar ciliated epithelium continuous with that of the nasal cavity. They are grouped as anterior (the frontal, anterior ethmoid and maxillary sinuses) and posterior (the posterior ethmoid and sphenoid sinuses). The anterior group drains into the middle meatus.
The posterior ethmoid and sphenoid sinuses drain into the superior meatus and sphenoethmoidal recess respectively. The drainage area of the anterior group of paranasal sinuses is called the osteomeatal complex. Fig. (5).

**Maxillary sinus:**
The maxillary sinus is pyramidal in shape and present in the body of the maxilla. It is related to the orbit superiorly, alveolus inferiorly, and pterygopalatine fossa posteriorly. Medially, it is related to the inferior and middle meati in the lateral nasal wall. Its ostium opens high up in its medial wall, to drain in the middle meatus. The second premolar and first molar teeth are closely related to the floor of the sinus.

**Ethmoid sinus:**
The ethmoid sinus described as a labyrinth of air-filled cavities located in the superior part of the lateral nasal wall. It is separated into anterior and posterior by the basal lamella of the middle turbinate. Important adjacent structures include the orbit laterally and anterior cranial fossa superiorly.

**Frontal sinus:**
The frontal sinus lies between the outer and inner tables of the frontal bone. It is not present at birth. Its boundaries are the orbit inferiorly, the anterior cranial fossa posteriorly, the forehead anteriorly and separated from the other frontal sinus medially by the septum. Its ostium locates at the medial part of its floor to drain in the middle meatus.

**Sphenoid sinus:**
The sphenoid sinus is situated in the body of the sphenoid bone. It has a close lateral relationship to cavernous sinus, the internal carotid artery and the optic nerve. The pituitary fossa lies posterosuperiorly.

Fig. (5) The Nasal Sinuses
Physiology of the Nose and Sinuses

Functions:
1. Respiratory airway.
2. Air conditioning of the inspired air.
3. Olfaction.
4. Purification of the inspired air.
5. Resonance of voice.
6. Tears drainage.

1. Respiratory airway:
The nose is the first station of breathing, which is the main function of the nose. The nose is the natural respiratory airway.

2. Air Conditioning:
The inspired air must reach the lower respiratory system in a suitable temperature and humidity condition. The rich vascular nasal mucosa gives the suitable temperature to the air current; the mucous secretion of the nasal mucosa gives it humidity. The turbinal projection increases the surface area which helps the above functions.

3. Olfaction:
Olfaction is an important sense. The olfactory mucosa is located in the roof of the nasal cavity. It contains bipolar neurons with their dendrites and cilia. The axons of these neurons synapse with the olfactory bulb. This sense depends on the integrity of the olfactory pathway from the olfactory mucosa to the olfactory center.

4. Purification:
This is done through the vestibular hair mesh for the large dust particles. Mucociliary clearance: the smaller particles stick to the mucus blanket and pass backwards by the ciliary movement of the mucous membrane to the nasopharynx to be expelled outside or go through the upper digestive system.
5. **Resonance of Voice:**
   The nasal cavity and the paranasal sinuses share an important role in this function.

6. **Tear Drainage:**
   Tears from the lacrimal glands are drained through the nasolacrimal duct to the inferior meatus.

### Symptoms of the Nasal Diseases

#### I. Nasal Obstruction:
May be unilateral or bilateral, partial, or complete obstruction.

**Causes:**

2. Developmental: Deviated septum.
3. Trauma:
   - Foreign body.
   - Accidental trauma:
     - Fracture nasal bones and associated fracture of septum.
     - Septal haematoma.
   - Operative trauma:
     - Haematoma after septal operation.
4. Inflammatory:
   - All inflammations (acute or chronic, specific or non specific) rhinosinusitis.
5. Allergy:
   - Allergic rhinitis and vasomotor rhinitis
6. Polyps
   - sinonasal polypi and antrochoanal polyp.
7. Tumours:
   - Benign and malignant tumours.
8. Causes in the nasopharynx:
   - Adenoids enlargement in children.
   - Nasopharyngeal angiofibroma (Benign tumours).
   - Malignant tumours.

**Causes of unilateral nasal obstruction:**

2. Trauma: Foreign body.
3. Inflammatory:
   - Antrochoanal polyp.
   - Unilateral Rhino-sinusitis.
   - Nasal diphtheria.
4. Deviated septum: to one side.
5. Tumours: benign and malignant.
II. Nasal Discharge:
Types of nasal discharge:
1. Mucopurulent and purulent discharge:
   - Acute or chronic rhino sinusitis.
2. Blood:
   - Epistaxis.
3. Watery:
   - Allergic rhinitis.
   - Vasomotor rhinitis
   - Early acute rhinitis.
   - Excessive secretions of lacrimal glands.
   - CSF rhinorrhoea.
4. Fluid and food: (Nasal Regurgitations)
   - Congenital: cleft palate and short palate.
   - Traumatic: Oroantral and oronasal fistulae.
   - Inflammatory: Syphilitic perforation of the hard palate and osteomyelitis.
   - Neurogenic: Post-diphtheritic, bulbar and pseudobulbar palsy.
   - Neuromuscular: Myasthenia gravis.
   - Malignant: Erosion of the palate.

Causes of unilateral nasal discharge:
1. Unilateral purulent or mucopurulent discharge: Unilateral rhinosinusitis.
2. Unilateral watery discharge: CSF rhinorrhoea.
3. Unilateral regurgitation of water and food: Oroantral and oronasal fistulae.

N.B. Unilateral fetid nasal discharge
1. Foreign body of the nose.
3. Unilateral sinusitis of dental origin.
4. Malignant tumours of nose and sinuses.

III. Olfactory Disorders:
1. Anosmia (complete loss of the sense of smell):
   - It must be bilateral.
   - It is often described with loss of taste because flavour is largely perceived by olfaction.

Causes:
1. In the nose:
   - Collapsed alae nasi.
   - Bilateral nasal polypi and hypertrophy of the turbinates.
   - Atrophic rhinitis.
   - Peripheral neuritis after influenza or exposure to gases.
b. Cranial causes:
   • Fracture skull base involving cribriform plate.

c. Intracranial:
   • Compression of the nerve tract by brain tumours and abscess.
   • Basal meningitis

2. **Hyposmia** (diminished sense of smell) causes those like anosmia.

3. **Cacosmia** (Patient himself perceives bad smell):
   Causes:
   a. Maxillary sinusitis of dental origin.
   b. Foreign body in the nose with fetid discharge.

4. **Parosmia**: (Hallucination of smell)
   It is a perversion of the sense of smell (i.e. patient smells non-existing odours).
   Causes:
   b. Organic:
      • Influenzal neuritis.
      • Epileptic aura.
      • Uncinate fits.
      • Hysteria.

IV. **Facial Pain and Headache**:
   Causes according to site:-
   A. Extracranial:
      i. Nose and sinuses:
         • Sinus headache: acute and chronic sinusitis. Dull aching pain over the affected sinus, increases on bending forwards and shows periodicity.
         • Contact headache: deviated septum, concha bullosa.
         • Inflammations: acute rhinitis, vestibulitis.
         • Malignant tumours of the nose, sinuses and nasopharynx.
      ii. Ear:
         • Inflammations: Otitis externa, ASOM, complications of CSOM.
         • Tumours: Glomus tumour, carcinoma.
      iii. Eye:
         • Acute glaucoma, orbital infections and tumours: Orbital or retro-orbital pain.
         • Astigmatism: Headache at the end of the day.
      iv. Temporo-mandibular joint: Arthritis, dislocation and malocclusion.
      v. Teeth:
         • Dental caries, periapical abscess, periodontitis, and post-extraction neuralgia: Pain is localized or referred along maxillary or mandibular nerves. Teeth are sensitive to thermal stimulation and percussion.
vi. Cervical:
  • Cervical spondylosis, disc prolapse: Occipital headache, frontal region or neck and shoulders.

vii. Neuralgia:
  • Trigeminal neuralgia: Facial pain along the maxillary or mandibular divisions of trigeminal nerve, precipitated by washing the face, shaving or tooth brushing.
  • Glossopharyngeal neuralgia: Pain in the tonsillar region and referred to the ear.

viii. Vascular headache:
  • Migraine: Paroxysmal attacks of throbbing unilateral cranial headache and facial pain, lasting for several hours and associated with nausea and photophobia. It is due to spasm followed by dilatation of intra- and extra-cranial arteries. Classic migraine is preceded by prodroma of blurring of vision and flashes of light.
  • Temporal arteritis: Persistent throbbing pain with tender cord like superficial temporal arteries.

ix. General causes:
  • Systemic infections.
  • Toxaemia: Renal and hepatic diseases.
  • Constipation.
  • Hypertension and hypotension.

x. Psychogenic or Tension headache: Occipital headache, may refer to frontal region, and associated with spasm of neck muscles.

B. Cranial: Osteomyelitis of cranial bones e.g. Frontal bone.

C. Intracranial:
  • Space occupying lesions e.g. tumours, cyst, abscess and aneurysm.
  • Inflammations: meningitis, encephalitis.
  • Increased intracranial pressure (empty sella symptoms).
  • Hydrocephalus.

V. Nasal Deformity:
  • Deformities of the external nasal shape include soft, bony and cartilaginous components of the nasal framework. The aetiology may be traumatic, developmental or congenital.

VI. Sneezing:
  • It is due to irritation of the mucous membrane and nerve endings. Sneezing is one of the major symptoms of allergic rhinitis; it may be also due to exposure to irritant gases, fumes, dust and sudden change of temperature.
Nasal Examination

- Nasal examination must include external and internal examination of the nose.

I. External nasal examination:
   A. Inspection: Of the shape and position of the nose whether central or twisted, the nasal dorsum for hump, or saddle deformity and skin inspection for redness and scars or tumours.
   B. Palpation of the nose: for tenderness as in vestibular furunculosis and consistency of nasal masses and cysts.

II. Internal nasal examination:
   A. Anterior rhinoscopy:
      By head mirror, head light or endoscopes to examine:
      - Patency of the nasal cavities: whether normal, or obstructed and the presence of polyps or masses.
      - Mucous membrane: for coloration, dryness, polyps, ulceration and tumours.
      - Nasal discharge: for mucoid, mucopurulent, watery, bloody discharge, odour and crustation as in atrophic rhinitis.
      - Nasal septum: for spurs, type of deviation and the presence of septal perforation.
      - Little's area: for apparent congested vessels.
      - The turbinates: for size (hypertrophic or atrophic) and the covering mucous membrane.
      - The meati: for discharge, polyps and tumours.
   B. Posterior rhinoscopy:
      - Using postnasal mirror.
   C. Oral examination:
      - The hard palate for swelling in relation to the floor of the nose and maxillary sinuses, postnasal discharge and for cleft palate and movement of the soft palate.
   D. Transnasal endoscopy:
      - This is direct vision with excellent illumination for all the internal nasal compartments whether anterior or posterior. It is done by the nasal endoscopes with different angles of vision. It allows proper examination of the sinus ostia, presence of the polyps or tumours in the middle meatus. It also visualizes the posterior nasal compartments, superior turbinate and meatus, patency of the posterior choana and examination of the nasopharynx and Eustachian tubes.
Investigations

I. Radiological:
   1. Plain X-ray:
      - Is now widely replaced by CT scan.
   2. Computerized Tomography Scanning (CT):
      - It gives proper imaging of the nasal cavities. It shows details of the fine structures as sinus ostia, masses, and the state of the paranasal sinuses. Also it gives details of the important structures neighbouring the nasal cavity as orbit, skull base, and optic nerves.
   3. Magnetic Resonance Image (MRI):
      - It gives good imaging of the soft tissue lesion as malignant tumours, cyst and swellings showing its dimensions and relations to the nearby structures.

II. Laboratory:
   1. Blood tests: as complete blood picture, B.T., C.T., renal and liver function tests in cases of epistaxis.
   2. Histopathological examination: For biopsies taken from nasal tumours or granulomas.

III. Rhinometry:
   - This apparatus measures the nasal resistance in relation to the cross sectional area of the nasal cavities.
Diseases of the Nose and Paranasal Sinuses

Congenital Disease of the Nose

**Choanal Atresia** Fig. (8)

Choanal atresia is a rare congenital closure of posterior nasal opening due to failure of canalization of the primitive bucconasal membrane. This results in the persistence of a bony plate (most commonly), membrane or both, obstructing the posterior nares. The condition may be unilateral (75%) or bilateral (25%).

![Fig. (8) Choanal Atresia](image)

Clinical picture:

- Bilateral presents as an emergency at birth because neonates are obligate nasal breathers. The neonate suffers severe respiratory difficulties and cyanosis until he cries and the mouth is opened. After a few quick breathes, the lips close again and this sequence of events continues. If not properly managed, the child may die from respiratory obstruction.

- Unilateral cases are usually asymptomatic at birth. It usually presents later in life with unilateral nasal obstruction and persistent thick mucoid discharge.

Investigations:

- Inability to pass a catheter or coloured drops from the nose to the nasopharynx.
- X-ray after instillation of a radio-opaque dye will show arrest of the dye in the nose. i.e. not posses to nasopharynx.
- CT is the method of choice for detection of the atresia.
- Diagnostic endoscopy in older children and adults shows atresia.

Treatment:

First aid: In bilateral cases the first priority is to insert and maintain an oral airway. An emergent perforation of the occluding plate by a probe or a wide bore trocar may be tried.

Definitive treatment:

- Transnasal: The transnasal route entitles the use of burrs or laser to perforate and widen the occluding plate under microscopic or endoscopic visualization. A stent may be inserted for 6 weeks.
Transpalatal: After elevation of a mucoperiosteal flap, the atretic plate and the posterior part of the nasal septum are resected followed by insertion of a stent.

Inflammations of the Nose

Furunculosis of the vestibule
It is an infection of a hair follicle in the nasal vestibule caused mainly by staphylococcus aureus.

Clinical picture:
The nose shows a red, hot, very painful swelling. The extreme tenderness is due to the tight attachment of the skin to the underlying cartilage.

Management:
1. Systemic and topical antibiotics.
2. The patient is advised not to squeeze the furuncle as there is a potential risk of spreading infection to the cavernous sinus via the facial and ophthalmic veins (dangerous area of the face).
3. In recurrent cases,
   a. A swab should be taken.
   b. Blood glucose level should be tested to exclude diabetes mellitus.
   c. Exclude the possibility of a nasal carrier.
4. Incision is delayed unless the furuncle is pointing if needed.

Rhinitis

The term rhinitis implies an inflammation of the lining membrane of the nose.
Actually the nasal mucous membrane is continuous anatomically with the paranasal sinuses mucous membrane. So every case of rhinitis is accompanied by a degree of sinusitis, also every case of sinusitis is associated with a variable degree of rhinitis. So the term (rhinosinusitis) is commonly used for description of inflammations of the nose and paranasal sinuses. However for simplification of the subject we use the term (rhinitis) when the main lesion is in the nose while the term (sinusitis) is used when the main lesion is in the sinuses.

Acute rhinitis:
   a. Acute non-specific rhinitis e.g. acute coryza (common cold) and influenza rhinitis.
   b. Acute specific rhinitis e.g. Nasal diphtheria.

Common cold (coryza)
In the common cold, nasal mucosa is infected by a virus. Those particularly implicated are, adenovirus, rhinovirus, respiratory syncytial virus and para
influenzae virus. A secondary bacterial infection usually supervenes.

Predisposing factors:
General factors:
- Bad ventilation.
- Fatigue.
- Malnutrition and vitamin deficiency.
- Low general resistance e.g. renal, hepatic, diabetic and immunodeficient patients.

Local factors:
- Nasal obstruction.
- Foci of chronic infection in the sinuses and nasopharynx.

Clinical picture:
The course of a common cold passes in four stages:
1. Prodromal stage: nasal dryness, irritation and sneezing.
2. Hyperaemic stage: nasal obstruction, watery discharge and general symptoms of mild toxaemia and fever. The mucous membrane appears red and swollen.
3. Stage of secondary infection: the discharge thickens, diminishes and becomes mucopurulent. Nasal obstruction and toxaemia are at their maximum.
4. Resolution stage: the symptoms and signs gradually diminish and recovery takes place after 5-10 days.

Complications:
- Sinusitis.
- Otitis media.
- Tonsillitis and pharyngitis.
- Laryngitis, tracheitis, bronchitis, pneumonia and asthma exacerbation.

Treatment:
- Treatment is symptomatic as the disease is self-limiting.
- The constitutional symptoms of pyrexia and muscular pain are controlled by an analgesic antipyretic such as aspirin or paracetamol.
- Steam inhalation and topical nasal decongestants may provide some relief from nasal obstruction.
- Antibiotics may be required for control of secondary infection.

Nasal Diphtheria
Diphtheria now is extremely rare. Nasal diphtheria is an inflammation of the nasal mucous membrane caused by Corynebacterium diphtheriae. It is usually secondary to faucial diphtheria, but very rare may be primary.

The nasal symptoms are obstruction and fetid discharge which is watery at first and later becomes blood stained and mucopurulent. The inferior turbinate, the floor of the nose and sometimes the septum are covered with a greyish adherent membrane. Removing this membrane leaves a raw bleeding surface.
Nasal swabs are essential for diagnosis.
Treatment:-
- Systemic antibiotics, usually parenteral penicillin and nasal toilet.
- Systemic antitoxins are also indicated.
- Patients should be isolated until negative 3 successive nasal swabs.

**Chronic Rhinitis:**

1. **Chronic Non-specific Rhinitis**
   a. **Chronic Hypertrophic Rhinitis**

Aetiology:
1. Recurrent acute rhinitis or sinusitis.
2. Allergic rhinitis.
3. Vasomotor rhinitis.

Pathology: Hypertrophy of the nasal mucosa and submucosa.

Symptoms:
1. Bilateral Nasal obstruction.
2. Bilateral Mucoid nasal and postnasal discharge.

Signs:
1. Congested hypertrophied nasal mucosa.
2. Congested hypertrophied inferior turbinate. It does not shrink with decongestive drops indicating irreversible changes.

Treatment:
1. Treatment of the cause.
2. Topical steroids.
3. Reduction of the inferior turbinate by:
   a. Surgical partial inferior turbinectomy.
   b. Submucous diathermy.
   c. Laser turbinate reduction.

b. **Atrophic rhinitis (Ozaena):**

Atrophic rhinitis is a chronic non-specific rhinitis characterized by progressive atrophy of the nasal mucosa and underlying bony turbinates. It usually commences at puberty.

Aetiology:

Primary atrophic rhinitis more common in females

The aetiology of atrophic rhinitis is still unknown but may be due to:
- Infection: cocobacillus ozaenae, klebiella ozaenae and other gram negative organisms have been isolated from cultures.
- Endocrine imbalance: oestrogen deficiency has been suspected.
- Malnutrition: iron and vitamin A deficiency have been claimed.
- Autoimmune disease.
- Autonomic imbalance.
- Hereditary factors.
Secondary atrophic rhinitis

- Long standing purulent rhinitis or sinusitis during childhood.
- Excessive surgical destruction of the nasal mucous membranes e.g. radical turbinectomy and repeated cauterization.
- Chronic specific rhinitis e.g. scleroma and syphilis.
- Severely deviated nasal septum (in the wider side).
- Post-irradiation.

Pathology:
- Atrophy of the mucosa, submucosa and bony turbinates.
- Atrophy of the sero-mucinous glands.
- Endarteritis of the arterioles.

Clinical picture:
- Crusty nasal discharge, foul odour. (Usually not smelled by the patient, as he has anosmia due to atrophy of the olfactory mucosa). Sense of nasal obstruction; in spite of roomy nose due to dullness of sensation of air on the atrophic mucosa, epistaxis on removing the crusts and sore throat, due to associated atrophic pharyngitis.

- Clinical examination confirms the presence of fetor and green or black crusts in roomy nasal cavities. Nasal mucosa is thin, pale, dry (atrophic) with atrophic inferior turbinates.

Treatment:
- Conservative:
  - Regular nasal douching with an alkaline solution should be considered twice daily. Other measures include 25% glucose in glycerine pack, topical oestrogen, oral potassium iodide and human placental extracts.
  - Antibiotics after culture and sensitivity tests can be used as well.
  - Treatment of the cause in secondary atrophic rhinitis.

- Surgical:
  - Different surgical procedures have been tried aiming at narrowing the nasal cavities or temporary closing the nostrils for 6-12 months.

2. Chronic Specific Rhinitis

Rhinoscleroma (Nasal Granuloma)

An endemic chronic specific infection of the nose in Egypt. More common in young adult females.

Aetiology: Klebsiella rhinoscleromatis: Gram -ve Frisch diplobacillus.

Pathology:
Submucosal chronic inflammatory cellular infiltration characterized by: Mikulicz cells: (large foamy cells containing the Frisch bacilli within its vacuoles) Russel bodies: (red-stained degenerated plasma cells), Plasma cells, lymphocytes and fibroblasts.
Clinical picture:
1. Catarrhal stage: resemble acute rhinitis.
2. Atrophic stage: Clinically similar to atrophic rhinitis.
3. Hypertrophic (granulation) stage: Bilateral hard, non-ulcerating submucous granulomatous nodules appear at the muco-cutaneous junction, then spread and coalesce to fill the nasal cavities and broaden the nose. Nodules may spread to the subcutaneous tissues of the nasal tip, upper lip and dorsum of the nose.
4. Fibrotic (Cicatrizing) stage: Dense fibrosis leading to nasal stenosis and external nasal deformity.

Investigations:
1. Biopsy and pathological examination.
2. Culture and antibiotic sensitivity test.

Sequelae: It may spread to: The pharynx (pharyngoscleroma), larynx (laryngoscleroma), rarely middle ear (tymanoscleroma) or lacrimal sac (dacryoscleroma).

Treatment:
1. Medical: long course of antibiotics for a minimum 4-6 weeks as the Frisch bacilli are intracellular and the antibiotics do not reach it easily.
   a. Streptomycin: 1gm I.M. daily, for 40 days (ototoxic and nephrotoxic drug).
   b. Rifampicin: 300 mg orally twice daily, for 40 days (hepatotoxic).
   c. According to the culture and sensitivity test.
2. Surgical:
   a. Re-establishment of the nasal or laryngeal air way by removal of the granulomatous masses or fibrous tissue by surgery or CO2 laser.
   b. Tracheostomy in case of severe laryngoscleroma with upper respiratory obstruction.

Nasal Lupus Vulgaris
Aetiology: Attenuated form of T.B. bacilli.
Clinical picture: Nasal obstruction and nasal discharge with apple-jelly nodules at the muco-cutaneous junction of the vestibule and nasal septum. They appear on blanching them by pressing it with glass slide or decongestive drops. Later on it gives, nodular ulceration with perforation of the cartilaginous septum and atrophic rhinitis.

Investigations:
2. Biopsy reveals T.B granuloma.

Treatment:
- Anti-tuberculous drugs and calciferol (vitamin D2).
**Sinusitis**

Inflammation of the mucoperiosteum lining the nasal sinuses.

**Classification:**

Sinusitis is classified according to the duration of symptoms into acute (less than 4 weeks), subacute (between 4 -12 weeks) and chronic (more than 12 weeks).

**Acute Sinusitis**

Acute inflammation of the mucoperiosteum lining the nasal sinuses.

**Aetiology:**

Causative organisms:
- Anaerobes in maxillary sinusitis of dental origin.

Source of infection:

I. Nasal:
   1. Acute rhinitis is the commonest.
   2. Diving and swimming during acute rhinitis.
   3. Nasal packing for long time.

II. Dental:
   - Unilateral maxillary sinusitis mostly is an anerobic infection through:-
     1. Dental infection: of upper second premolar or first molar tooth.
     2. Oro-antral fistula after extraction of the upper second premolar or first molar teeth.

III. Traumatic:
   1. Sinus foreign body.
   2. Fracture of the sinus.
   3. Sinus barotrauma.

![Fig. (8) Maxillary sinusitis secondary to dental infection](image)

Pathology: Acute catarrhal followed by suppurative sinusitis: There is congestion and oedema of the sinus mucosa with inflammatory exudates. Oedema leads to occlusion of the ostium and retention of exudates inside the sinuses.
Clinical picture:
- Fever, malaise and anorexia associated with nasal obstruction, anterior nasal discharge: mucopurulent or purulent and may be fetid in dental maxillary sinusitis and postnasal mucopurulent discharge with irritative cough.
- Facial pain and headache, pain is usually over the affected sinus as following: Maxillary sinusitis: over the cheek and referred to teeth, frontal sinusitis: over the forehead above the eye and periodic vacuum headache it starts in the morning and subsides at noon when the sinus is drained by gravity, ethmoiditis: in-between eyes, retro-orbital and occipital.

Signs: Facial examination reveals:-
- Oedema and redness of the skin over the affected sinus, tenderness over the cheek in maxillary sinusitis, floor of the frontal sinus in frontal sinusitis or inner canthus in ethmoiditis.

Nasal cavity examination:-
1. Congested oedematous nasal mucosa over the turbinates.
2. Mucopurulent or purulent discharge in middle meatus: in maxillary, frontal and anterior ethmoidal sinusitis, superior meatus in posterior ethmoiditis, sphenoethmoidal recess in sphenoiditis.
3. Postnasal mucopurulent discharge.

Differential diagnosis;
- Other causes of facial pain: dental pain, trigeminal neuralgia, migraine, or tumours of the sinuses.

Complications:
1. Chronic sinusitis.
2. Complications of sinusitis: more common in children. (see later)

Investigations:
1. Culture and sensitivity test of the sinus discharge.
2. Plain X-ray and CT scan: Opacity, fluid level or thickened mucosa of the affected sinus, obstruction of the osteomeatal complex, complications if present.

Treatment:
Medical:
- Rest, antibiotics (for 10-14 days), analgesics, antipyretics, decongestants, mucolytics and antihistamines. Decongestive nasal drops and steam inhalation to decrease oedema around the ostium and help sinus drainage and ventilation.

Surgical:
- Indications: to drain infected sinus in case of:-
  2. Threatened complications of sinusitis.
Chronic Sinusitis

Chronic inflammation of the mucoperiosteum lining the paranasal sinuses.

Aetiology:

Cause:

- Prolonged obstruction of the natural ostium of one or more of the paranasal sinuses leads to:-
  - Inadequate ventilation & drainage of the sinus
  - Overgrowth of organisms & infection of the mucous membrane.
  - Oedema and damage of the cilia causes more defective ventilation & damage leading to a cycle of chronic sinusitis.

Predisposing factors to chronic sinusitis

1. Inadequate treatment of acute sinusitis:
   - Virulent or atypical organisms.
   - Inappropriate selection or short course of antibiotics.

2. Local predisposing factors:
   - Anatomic variations that narrow the ostium area e.g. deviated septum or large middle turbinate.
   - Mucosal disease e.g. allergy, polyposis or mucosal transport disease.
   - Adenoiditis or dental infection leads to chronic maxillary sinusitis.
   - Disturbed mucociliary clearance: Cystic fibrosis, immotile cilia (Kartaggar's) syndrome: sinusitis, bronchiectasis, sterility.

3. Systemic:
   - Poor immunity e.g. DM or prolonged corticosteroid therapy.
   - Environmental factors e.g. smoking or pollution.

Causative organisms: Mixed aerobic and anaerobic organisms.

Clinical picture:

Symptoms:

1. Nasal obstruction, anterior nasal discharge: mucopurulent or purulent and may be fetid in dental maxillary sinusitis, postnasal mucopurulent discharge with irritative cough.

2. Facial pain is a localized dull aching pain over the affected sinus.

Site of pain: below the eye in maxillary sinusitis in-between eyes in ethmoiditis, above the eye in frontal sinusitis or behind the eye in sphenoiditis.

N.B. Vacuum headache: is characteristic for frontal sinusitis. This pain is maximal in the morning and decreases gradually over the day. The cause may be due to closure of the sinus ostium helped by congestion of the head due to lying position with absorption of the air from within the sinus cavity. Erect postion during the daytime gradually relieves the ostial obstruction leading to headache release.

3. Symptoms of descending infection: otitis media, pharyngitis, laryngitis and bronchitis.

4. Symptoms of septic focus: low grade fever, headache, rapid fatigue and arthritis.
Signs:
Diagnostic transnasal endoscopy: This should be performed routinely for examination of all patients with symptoms suspecting chronic rhinosinusitis. The aim is to detect signs of sinusitis e.g. purulent discharge from the sinus ostium, oedematous mucosa and polyps and also to identify anatomical or pathological abnormalities in the middle meatus that may contribute in sinusitis, so you can see:-
1. Congested oedematous nasal mucosa over the turbinates.
2. Mucopurulent or purulent discharge: In middle meatus in anterior group sinusitis, superior meatus in posterior ethmoiditis or sphenethmoidal recess in sphenoiditis.
3. Nasal polyps may be present in middle meatus: in chronic ethmoiditis.

Differential diagnosis:
1. Fungal sinusitis.
2. Tumours of the sinuses.

Investigations:
1. Radiological examination:
   a. Plain x-ray is no longer performed for diagnosis of chronic sinusitis as it poorly demonstrates the ethmoid, upper two thirds of the nasal cavity and frontal recess.
   b. Computed tomography (CT) scanning is the gold standard for diagnosis of chronic sinusitis. The aim is to determine the extent of pathology and to delineate the anatomy in patients undergoing surgery.
2. Culture and sensitivity test of the discharge.

Treatment:
Medical:
- Antibiotics should be given for at least 2 weeks, analgesics, decongestants, mucolytics.
- Decongestive nasal drops, steam inhalation and alkaline nasal wash.

Treatment of the predisposing factors: Septoplasty or adenoidectomy.

Surgical:
- Indication: Failed proper medical treatment or if complications occurred as indicated by endoscopic examination and CT scan.
- Functional Endoscopic Sinus Surgery (FESS): Fig. (9) The aim of this type of surgery is to restore function and patency of the natural ostium of the sinus to provide normal ventilation and drainage. This will allow diseased intra-sinus mucosa to return to its normal functioning state. This is achieved by endoscopic removal of the predisposing cause of ostium obstruction e.g. septoplasty, polyp removal and widening of the natural ostium of the involved sinus.
**N.B.** Indications of Endonasal Endoscopic Surgery of the sinuses:
1. Chronic rhinosinusitis.
2. Sinonasal polyposis.
3. Acute or chronic sinusitis with certain complications.
4. Mucoceles of the paranasal sinuses
5. Choanal atresia.
6. Endoscopic resection of benign nasal tumours when feasible (e.g. inverted papilloma and angiofibroma).
7. It can be used in epistaxis for identification and cautery of the bleeding vessel or ligation of the sphenopalatine artery transnasally when indicated.
8. Endoscopic closure of CSF rhinorrhea.
9. Endoscopic DCR (dacryocystorhinostomy)

Fig. (9) The sinuses after FESS.
Conventional sinus surgery: which are rarely needed now after FESS.

- Repeated antral puncture and lavage, intranasal inferior meatal antrostomy or radical antrum (Caldwell-Luc) operation for Maxillary sinusitis.
- External ethmoidectomy for Ethmoiditis.
- External frontal operation frontal sinusitis if ESS fails.
- External sphenoidal operation for Sphenoiditis.

Disadvantages:
1. The external facial scar and disturbed bony skeleton of nose and sinuses after external sinus operations.
2. The removed sinus mucosa is replaced by fibrosis lacking mucociliary activity and leading to recurrent symptoms.
3. The mucociliary clearance of the maxillary sinus is always towards the natural ostium and bypass the inferior meatal antrostomy.

**Fungal Sinusitis**

Types:
1. Allergic fungal sinusitis
2. Non-invasive fungal sinusitis
3. Invasive fungal sinusitis

1. **Allergic Fungal Sinusitis**
   This is not true infection but represents an allergic response to fungal growth within the sinuses leading to accumulation of fungal hyphae and allergic mucin within the sinus.

   Aetiology: Aspergillus Fungi.
   Clinical picture: Usually unilateral sinusitis.
   - Similar to chronic sinusitis with brownish tenacious mucoid nasal discharge, allergic mucin with multiple allergic nasal polyps arising from the middle meatus.

   Investigations:
   - CT scan: heterogeneous sinus opacity with focal hyperdense spots.
   - Biopsy and culture of discharge with special stains for fungus.

   Treatment:
   - Endoscopic sinus surgery to drain and ventilate the sinuses.
   - Steroids and immunotherapy.

2. **Non-Invasive Fungal Sinusitis (Mycetoma or Fungal ball)**
   Accumulation of fungal hyphae (fungal ball) within the sinus.

   Aetiology: Aspergillus, in immuno-competent patients.
   Clinical picture: Similar to chronic sinusitis, or asymptomatic and may be discovered accidentally.

   Treatment: Endoscopic sinus surgery to remove the fungal ball to ventilate the sinuses.
3. Invasive Fungal Sinusitis

A. Acute Invasive (Fulminate) Fungal Sinusitis

Aetiology:
- In immuno-compromised patients as leukaemia, AIDS, organ transplant, chemotherapy and uncontrolled DM.
- Mucormycosis cause intravascular thrombosis and gangrene of the sinuses.

Progress: Acute onset, rapid progressive course and lethal in short time.

Clinical picture: Unilateral sinusitis.
1. Similar to acute sinusitis with rapid deterioration of the general condition.
2. Necrosis of the nasal mucosa with gangrenous blackish turbinates.

Complications:
- Extension to orbit leads to proptosis, blindness and intracranial invasion or to the palate cause necrosis of palate, leads to coma and death.

Investigations:
1. CT scan: unilateral sinus opacity extending to the orbit or cranial cavity.
2. Culture of discharge with special stains for fungus: biopsy showed invasion of tissue with fungus.

Treatment:
1. Urgent endoscopic drainage of the sinus and debridement of gangrenous tissues at the same time with systemic antifungal: Amphotericin B.
2. Control of predisposing factor.

B. Chronic (Indolent) invasive fungal sinusitis

Aetiology:
- Aspergillus, in immuno-competent patients.

Progress:
- Insidious onset and slowly progressive course for months or years.

Clinical picture:
- Similar to chronic sinusitis but the infection is invasive to orbit or intracranial.

Investigations:
- Similar to acute invasive fungal sinusitis.

Treatment:
1. Endoscopic endonasal complete surgical debridement of gangrenous tissues.
2. Systemic antifungal, Amphotericin B.
Complications of Sinusitis

Spread of infection into or beyond the bony walls of the sinuses.

Aetiology:
1. Acute sinusitis.
2. Acute on top of chronic sinusitis.
3. Invasive fungal sinusitis.

I. Extracranial Complications

Orbital Complications: - Fig. (11)

The commonest complications of sinusitis and usually occur in children. They are common in ethmoid sinusitis. The extension of infection can occur either by direct extension from the ethmoid through the medial orbital wall (lamina papyracea) or by retrograde thrombophlebitis via valveless veins.

1. Eyelid oedema (preseptal cellulitis)
2. Orbital cellulitis (postseptal cellulitis) is inflammation of the orbital contents. It is manifested by axial proptosis, chemosis and ophthalmoplegia. It may progress to orbital abscess and blindness
3. Subperiosteal abscess: collection of pus between periorbita (periostium of orbit) and the bony orbital wall. The clinical picture shows pain, lateral proptosis and limitation of eye movements.
4. Orbital abscess: collection of pus in the orbit. It has the same clinical presentation of orbital cellulitis. CT scan with contrast on paranasal sinuses and orbit can distinguish between orbital cellulitis, subperiosteal and orbital abscess.
5. Cavernous sinus thrombosis: proptosis, ophthalmoplegia, chemosis, lid oedema and visual loss with development of contralateral ocular signs if the case is neglected.

Fig. (11) Orbital complications of sinusitis: (A) Preseptal cellulitis(B) Orbital cellulitis (C) Subperiosteal abscess (D) Orbital abscess (E) Cavernous sinus thrombosis.
Treatment:
Medical: Extensive broad spectrum antibiotics should be given.
Surgical: surgery in the form of FESS or external frontoethmoidectomy.

II. Cranial Complications
Osteomyelitis of the Frontal Bone
Symptoms:
1. Fever, headache, malaise, anorexia and toxaemia.
2. Severe pain and swelling over the forehead.
Signs:
1. Oedema and tenderness of the forehead.
2. Fluctuant swelling over the forehead (Pott’s puffy tumour) due to subperiosteal abscess. It may rupture leading to fistula.
Investigations:
• Plain X-ray and CT scan: Moth-eaten appearance and sequestration of frontal bone.
• Culture and sensitivity test for discharge.
Treatment:
1. I.V. Broad-spectrum antibiotics.
2. Endoscopic sinus surgery to drain and ventilate the sinus.
3. External frontal drainage operation and sequestrectomy.

Osteomyelitis of the Maxilla
Age: Common in infants and children.
Symptoms:
1. Fever, headache, malaise, anorexia and toxaemia.
2. Pain, redness and swelling over the cheek.
Signs:
1. Oedema and tenderness over the cheek.
2. Fluctuant swelling of the maxilla due to subperiosteal abscess.
3. It may rupture leading to oro-antral fistula or cutaneous fistula.
Investigations:
• Plain X-ray and CT: is diagnostic.
• Culture and sensitivity test for discharge.
Treatment:
1. I.V. Broad-spectrum antibiotics
2. Surgical drainage and sequestrectomy.

III. Intracranial Complications
Extradural abscess, subdural abscess, meningitis and frontal lobe abscess: often complicate frontal sinusitis.
Treatment requires external or endoscopic drainage of the paranasal sinus, drainage of intracranial abscess and aggressive broad-spectrum antibiotics.
**Cavernous Sinus Thrombophlebitis: - Fig. (12)**

**Aetiology:**

3. Skin infections of dangerous area of the face (the external nose and upper lip) through the anterior facial vein.
4. Suppurative otitis media complicated by lateral sinus thrombophlebitis.

![Diagram](image)

**Clinical picture:**

1. High fever, rigors, severe headache and toxæmia.
2. Ipsilateral orbital manifestations:
   a. Lid oedema, conjunctival chemosis and Proptosis.
   b. Ptosis, dilated pupil and ophthalmoplegia (because 3<sup>rd</sup>, 4<sup>th</sup>, 6<sup>th</sup> cranial nerves pass inside the cavernous sinus).
   c. Fundus examination:- papilloedema and engorged retinal veins.
3. The other side is commonly involved.

**Prognosis:** Usually fatal if untreated properly as it leads to coma and death from meningitis and cerebral thrombophlebitis.

**Investigations:**

1. CT scan with contrast.
2. MRI with angiography.

**Treatment:**

1. Aggressive broad-spectrum antibiotics, anticoagulants and analgesics.
2. Endoscopic drainage and ventilation of the infected nasal sinus.

**Allergic rhinitis**

It is an Ig-E mediated hypersensitivity of the mucous membrane of the nose to specific allergens.

**Aetiology:** Genetic predisposition is the basic factor. The relatives of the patient may have any atopic manifestation (nasal allergy, bronchial asthma or allergic dermatitis).
Types:
- Seasonal allergic rhinitis: Occurs in seasons e.g. hay fever.
- Perennial allergic rhinitis: Persistent all over the year.

Allergens in the form of:
1. Inhalants are the commonest:
   - Seasonal: Pollens of trees and grasses.
   - Perennial: House dust mites, fungus spores animal dander, feather and occupational inhalants e.g. flour, wood and latex.
2. Ingestants (rare): Egg, strawberry, bananas, fish, and milk.
3. Drugs: Aspirin, penicillin and plasma.

Pathology: (type I hypersensitivity reaction).
1. Initial exposure of atopic individual to allergen: = Sensitization
   - The inhaled allergens stimulate specific IgE production by plasma cells. These immunoglobulins fix to the cell membrane of the mast cells. Such cells become sensitized to the allergen.
2. Further exposures to the specific allergen: = Hypersensitivity
   - The allergens bind to the fixed IgE on the sensitized mast cells causing degranulation of the mast cells leading to release of chemical mediators: e.g. histamine, leukotrienes, prostaglandins and chemotactic factors manifested by:
     - Stimulation of nerve endings causes sneezing and nasal itching.
     - Stimulation of seromucinous glands causes rhinorrhea.
     - Vasodilatation congestion increased capillary permeability causes oedema leads to nasal obstruction.
     - Cellular infiltration of eosinophils and basophils attracted by chemotactic factors. Basophils release vasoactive mediators which cause nasal obstruction and rhinorrhea. The nasal mucosa is continuous with the sinus mucosa, so sinuses are usually involved with thickening of the lining mucosa and fluid effusion.

Clinical picture:
Symptoms:
- Repeated and paroxysmal attacks of sneezing.
- Itching in the nose and palate.
- Bilateral watery nasal discharge (Rhinorrhea).
- Bilateral and intermittent nasal obstruction
- Associated conjunctival allergy and bronchial asthma.

Signs:
- Hypertrophy of the turbinates.
- Oedematous pale or violet coloured mucosa of the nose.
- Watery or mucoid nasal secretion.

Sequelae:
- Chronic hypertrophic rhinitis.
- Allergic nasal polypi.
Investigations if needed:-

1. Skin sensitivity tests: with several allergens to detect the causative allergen.
2. Nasal provocation tests: A spray of the suspected allergen applied into the nose produces sneezing and rhinorrhea.
3. Blood tests:
   - Radio-immuno-sorbent test: Increased total plasma Ig E level.
   - Radio-allergo-sorbent test: Increased plasma Ig E level to specific allergen.

Treatment:

1. Avoidence: avoid exposure or intake of the allergen if possible.
2. Oral antihistamines: the recent non-sedating antihistamines are tolerable by the patient.
3. Local antihistamines: levocabastine nasal spray.
4. Local and oral chromoglycates: which are mast cell stabilizers.
5. Local corticosteroids (sprays): very effective in most of the cases.
6. Systemic corticosteroids: may be given in selected cases.
7. Immunotherapy: if medical treatment fails.

Vasomotor Rhinitis

Abnormal reaction of the nasal mucosa to non-allergic factor due to over activity of the nasal parasympathetic supply (cholinergic hyperactivity) causing vasodilatation and watery nasal secretion.

Aetiology:

1. Unknown.
2. Environmental triggers: dust, tobacco, and changes in humidity.
3. Endocrinal factors: pregnancy, menopause, menses, and hypothyroidism.
4. Drugs: antihypertensive drugs, contraceptive pills, and abuse of decongestive nasal drops (rhinitis medicamentosa due to rebound congestion).
5. Emotional upset.

Clinical picture.

Similar to allergic rhinitis with negative allergic tests.

Investigations: - Negative of all tests for allergy

Treatment:-

Medical: Ipratropium (anticholinergic) nasal spray.
Surgical: vidian nerve neurectomy if no response to medical treatment.

Nasal Polyps

A term sinonasal polypi is commonly used nowadays as they originate from the sinuses. A polyp is a pedunculated oedematous mucosal swelling, soft in consistency and smooth in surface. There are two main types: Ethmoidal polypi and Antrochoanal polyp.
Pathology:
Polyps are covered by ciliated columnar epithelium with areas of squamous metaplasia. Submucosa is oedematous with eosinophil cellular infiltration.

**Ethmoidal Polyps** Fig. (13)
Arise from the ethmoid sinuses and middle meatus.

Age: commonly in adults.

Aetiology:
1. unknown
2. Allergy: Allergic rhinitis (the commonest 90%).
3. Inflammatory: Chronic ethmoiditis.
5. Drugs: idiosyncrasy to aspirin and other anti-inflammatory drugs, this is usually associated with bronchial asthma.

![Fig. (13) Multiple polyps arising from the ethmoid sinuses and middle meatus.](image)

Clinical picture:

Symptoms:
1. Bilateral persistent nasal obstruction and watery or mucoid discharge. This obstruction is partially at the beginning and complete when polyps fill the nose.
2. Anosmia.
3. Symptoms of allergic rhinitis.

Signs:
- Bilateral multiple pale glistening smooth soft pedunculated polyps (grape like) arising mainly from the middle meatus.

Investigations:
1. CT scan: Opacity of the nose and sinuses.
2. Biopsy (if tumour is suspected): revealed eosinophilic cellular infiltration of the polypi.

- **N.B.** The sinonasal polypi must be differentiated from other polypoid lesions in the nose as:
  1. Neoplastic polypoid masses, bleeding polypus of the septum, inverted papilloma and malignant tumours.
  2. Meningocele and meningoencephalocele of the nose.
Treatment:
   Medical:
   – Treatment of allergic rhinitis to avoid recurrence.
   – Antibiotics: for ethmoiditis.
   – Steroids (oral and topical): for small polyps and preoperatively.

   Surgical:
   – Functional endoscopic sinus surgery followed by long term local corticosteroid therapy. Short courses of systemic corticosteroids may be required in some cases.
   – The old trend of polypectomy alone is followed by recurrence in 100% of cases.

Behaviour: Recurrence after surgery is common.

Special types of nasal polypi:
   1. Cystic fibrosis: extensive sinonasal polyps in children with thick tenacious nasal discharge and broadening of the nasal bridge. Sweat test (low sodium level in sweat) is diagnostic.
   2. Triad of aspirin sensitivity, bronchial asthma (ASA) with extensive sino-nasal polyps. It is due to alteration of prostaglandins synthesis.
   3. Allergic fungal sinusitis with polyposis.

Antro-choanal Polyp Fig. (14)
   It is a single unilateral polyp arises from the maxillary sinus (so the term antro) and passes the middle meatus then backwards to the choana (so the term choanal).
   Age: Children and young adults.

Aetiology:
   Unknown. It is not related to infection or allergy.

Clinical picture:
Symptoms:
   Unilateral persistent nasal obstruction and may become bilateral when the polyp markedly enlarged to fill the nasopharynx.
   Unilateral mucoid, or mucopurulent nasal discharge.

Fig. (14) Antro-choanal Polyp
Signs:
- Unilateral single pale glistening smooth soft polyp arising from the middle meatus, passing to the choana and projecting into the nasopharynx.
- Posterior rhinoscopy will show the polyp in the nasopharynx.

Investigation:-
- CT scan: Unilateral opacity of the maxillary sinus, nose and nasopharynx.

Behaviour: High rate of recurrence after surgery.

Differential Diagnosis of nasal mass:
1. Benign tumours.

Treatment:
- Endoscopic polypectomy with wide middle meatal antrostomy.
- Polypectomy by avulsion with a high recurrence rate.
- Caldwell-Luc operation: for recurrent antro-choanal polyp.

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**Nasal Trauma**

**Fracture Nasal Bones**
Aetiology: Direct trauma to nose as fall, road traffic accidents, sport accidents or personal assault.

Incidence:
- Males are more common than females because they are more liable to trauma and the nose is more prominent.
- Adults are more common than children.

Symptoms:
- Epistaxis: due to mucosal tears. It is usually mild and self-limited.
- Pain: at the time of trauma.
- Swelling of the nose.
- Skin: cut wounds of nasal skin.
- Nasal obstruction.

Signs:
- External nasal deformity.
- External swelling: due to odema and ecchymosis.
- Tenderness and crepitus (click sound).
- Black eye: due periorbital ecchymosis.
- Associated septal haematoma, fracture or dislocation → nasal obstruction.

Investigations:
- Plain X-ray of nasal bones: for medico-legal aspect.
Treatment:

- Control of epistaxis, analgesics and cold compresses for 24 hours to reduce the swelling.
- Reduction and fixation of the fracture.
  a. Fractures (within 6 hours) with no oedema need:
     - Immediate reduction of the nasal bones by Walsham's forceps Fig. (15) and septum by Ash's forceps.
     - External fixation by nasal splint or plaster of Paris.
     - Internal fixation by anterior nasal pack (for 48 hours).
  b. Fractures with marked oedema:
     - Delayed reduction and fixation after 7 days till oedema subsides.
     a. Malunited fractures (after 2 weeks): Rhinoplasty or Septorhinoplasty.

![Fig. (15) Reduction of fracture nasal bone with Walsham's forceps](image)

**Foreign Body in the Nose**

Usually in children and mentally retarded adults.

**Types of F.B.:**

1. Inanimate: either metallic, button, beads, vegetable or non-vegetable F.B.

**Symptoms:**

- Unilateral fetid purulent, may be blood stained nasal discharge if undetected early.
- Unilateral nasal obstruction.

**Signs:** Foreign body is seen and felt by probing in the nasal cavity.

**Complications:** Undetected foreign bodies lead to:

1. Rhinitis and sinusitis.
2. Rhinolith formation (Nasal stone).

**Treatment:**

**Extraction without anaesthesia:**

1. Under vision with good illumination,
2. Steady head.
3. Using a hook and never use forceps to avoid backward displacement of the F.B.
General anaesthesia is indicated in:
1. Posteriorly located F.B.
2. Uncooperative uncontrollable patient.
3. Neglected F.B. with possibility of bleeding.

**Rhinolith (Nasal stone)**
Calcium and magnesium salts precipitated from the nasal secretions on a nasal foreign body. It is usually unilateral, single, hard or friable, greyish brown with rough mulberry-like surface which bleeds during removal.

**Cerebrospinal Rhinorrhea**
Leakage of cerebrospinal fluid from the nose.

Aetiology:
I. Traumatic: (80%)
   1. Casual trauma: car accident, personal assault..etc.
   2. Surgical trauma (iatrogenic): after neurosurgical operations, hypophysectomy or endoscopic endonasal sinus surgery.

II. Non-traumatic (spontaneous): (20%)
   1. Hydrocephalus.
   2. Brain tumours particularly pituitary tumours.
   3. Syphilis.
   4. Meningocele or meningoencephalocele.

Clinical picture:
- Unilateral dripping of clear watery nasal discharge. It increases by leaning forwards and straining, and it doesn't stiffen handkerchief (no mucus).
- In fresh traumatic cases, it is usually mixed with blood and so passed unnoticed but it can be "halo test".
Site of leak: Defect in:
1. Cribriform plate.
2. Cranial wall of the ethmoid, frontal or sphenoid sinuses.

Investigations:
Complete diagnosis of a case of CSF rhinorrhea should include:
1. Fluid analysis: presence of glucose or reducing substances in the fluid
2. is not diagnostic, but detection of 30 mg/dl glucose or more in the leaking fluid is diagnostic.
3. CT scan: with intrathecal contrast may demonstrate the dye leaking to the nose, defining the site and detect any underlying cause.
4. MRI: is more sensitive than CT with contrast.
5. Intrathecal flourescein injection: followed by endoscopic examination to detect the yellowish green colour of the dye inside the nose. This will ensure the diagnosis and define the site of leak.
6. Beta transferrin detection in the fluid is diagnostic as this substance is present only in CSF.

Complications: Meningitis, intracranial aerocele.

Treatment:
Conservative: as most of tears of dura heal spontaneously.
- Rest in semi-sitting position.
- Avoid nose blowing, straining, coughing and nasal packing.
- Antibiotics crossing brain barrier.

Surgical: Failed conservative treatment for 2-3 weeks repair of the defect by tissue graft; (fat, fascia or muscle) by: transnasal endoscopic or transcranial approach.

Differential diagnosis:
1. Allergic rhinitis
2. Common cold
3. Vasomotor rhinitis.

**Oro-antral Fistula**
It is abnormal communication between the oral cavity and the maxillary sinus.

Aetiology:
- Traumatic:
  - Dental extraction of upper second premolar or first molar tooth (commonest cause).
  - Enucleation of dental cyst.
  - Caldwell-Luc operation.
- Inflammatory necrosis: Osteomyelitis of the maxilla, or syphilis (gumma).
- Cancer maxilla (neoplastic erosion).
Clinical pictures:
- It may be discovered immediately by the dentist during the dental extraction.
- If not discovered immediately:
  - The patient may feel air escaping from the fistula on blowing of the nose.
  - Nasal regurgitation of fluid or sometimes food.
  - Manifestation of sinusitis if infection starts.
  - The fistula site can be seen in the site of tooth socket and can be assured by probing (This maneuver is contraindicated in recent cases as it will disturb healing).

Symptoms:
- Unilateral nasal regurgitation of fluid and food.
- Escape of air from the fistula on nose blowing.
- Unilateral fetid purulent nasal discharge (maxillary sinusitis).
- Purulent discharge from the fistula.

Signs:
- The fistula can be seen through the oral cavity.
- A probe can be passed through the fistula to the sinus.

Complication: Maxillary sinusitis.

Investigations:
- CT scan of paranasal sinuses: shows the fistula and maxillary sinusitis.

Treatment:
Recent fistula (within 24 hours):
- Antibiotics, decongestive nasal drops and avoid nose blowing.
- Surgical repair by direct suturing of the mucosal edges.

Late fistula:
1. Surgical repair by buccal or palatal flap.
2. Radical antrum operation or endoscopic sinus surgery for treatment of sinusitis.

**Epistaxis**

Definition: Bleeding from the nose. It is a common ENT emergency.

Aetiology:

I. Local Causes:
1. Idiopathic: the commonest cause in children and adolescents. (90% of cases mostly from Little's area).
2. Traumatic: Nose picking, blow to nose, fracture nose and skull base, foreign body and after nasal operations.
4. Septal: Deviated nasal septum, septal perforation.
5. Inflammatory: Acute and chronic rhinitis and sinusitis.
6. Tumours:
   b. Malignant tumours of nose, sinuses and nasopharynx.

II. General Causes;
   1. Hypertension usually from sphenopalatine artery (posterior epistaxis).
   2. High venous pressure: Mitral stenosis, heart failure and mediastinal tumours.
   5. Fevers: Rheumatic fever, exanthemata and typhoid.
   6. Drugs: Anticoagulants as heparin, antiplatelets (as salicylates).

Management of Epistaxis
I. First Aid Measures:
   1. Pinch the nose by thumb and index fingers to compress the little's area.
   2. Place the patient in sitting and leaning forwards position unless shocked.
   3. Apply cold compresses to the nose to induce reflex vasoconstriction.
   4. Insert piece of cotton soaked with adrenaline (1/100,000) or vasoconstrictor nasal drops. Instead ephedrine is used in hypertensive patients.

II. Control of Shock: if present
   1. Supine position with legs elevated, sedatives, IV fluids, coagulants and blood transfusion if it is needed.
   2. Patient is monitored for: vital signs, Hb, haematcrite, and urine output.

III. Assessment: during preparations for control of the epistaxis
   a. History: Unilateral or bilateral epistaxis, duration and frequency, Previous attacks.
   b. General examination to detect:
      1. Signs of hypovolaemic shock.
      2. General cause of epistaxis e.g. fever, hypertension …..
   c. Nasal examination by anterior rhinoscopy and endonasal endoscopy to detect:
      1. Site of bleeding: Fig.(17) - Anterior from Little's area, Superior above middle turbinate 'from ethmoidal arteries' or Posterior posterior to middle turbinate from sphenopalatine artery
      2. Local cause of epistaxis e.g tumours.
IV. Control of epistaxis by:

1. Cauterization of bleeding point under local anaesthesia by :-
   a. Chemical cautery (silver nitrate or chromic acid crystals).
   b. Electrocautery or diathermy.

2. Anterior nasal pack: Formed of ribbon gauze soaked with antibiotic ointment Fig. (17).

3. Inflatable balloon Fig (18).
   Indication: Failed cauterization. It is removed after 24-48 hours.

4. Posterior nasal pack: Fig. (19) Gauze pack in nasopharynx associated with anterior nasal pack, it is removed after 24-48 hours.
   $\S$ Indication: in posterior epistaxis with failed anterior nasal pack.

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Fig. (16) Site of bleeding in epistaxis.

Fig. (17) Anterior nasal pack.

Fig. (18) Double balloon nasal pack.
Fig. (19) Steps of posterior nasal pack
5. Endoscopic diathermy of sphenopalatine artery for uncontrolled posterior nasal bleeding.
6. Arterial embolization.
7. Arterial ligation:
   a. Indication: Severe epistaxis with failed diathermy and nasal packing.
      a. If bleeding from superior part above middle turbinate:
         b. Ethmoidal arteries ligation, via external ethmoidectomy approach.
   b. If bleeding from posterior part either.
      b. Maxillary artery ligation, via trans-maxillary sinus approach.
   b. External carotid artery ligation, via cervical approach.

V. General Treatment:
   Rest, sedatives, coagulants and vitamin K, antibiotics with nasal packing.

Diseases of the Nasal Septum

Deviated Nasal Septum (D.S.)

The nasal septum is the bony-cartilagenous wall separating the two nasal cavities. The anterior part is cartilaginous and formed by quadrangular septal cartilage. Posterior parts are the vomer bone and perpendicular plate of ethmoid bone which form the bony component of the nasal septum. The nasal septum is usually deviated to one or the other side and this is of no clinical value if there is no complaint by the patient. Severe septal deviation presenting itself clinically and needs surgical interference for reposition of the septum to more or less middle position.

Aetiology:
- Traumatic: either intrauterine, birth, or accidental trauma to the nose.
- Developmental: High arched palate faster growth of septum than cranial bones or excessive ossification of one plate of vomer.

Types of D.S.: Fig. (20)
- C shaped: to one side.
- S shaped: i.e. bilateral deviation.
- Spure or sharp angulation: usually at the inferior part of the septum.

Symptoms:
- Nasal obstruction: Persistent unilateral or bilateral.
- Facial pain: contact headache due to contact of septum with turbinates, or vacuum headache due to impaired ventilation of frontal sinus → sinusitis.
- Epistaxis: due to angulation of vessels over spur or drying of
mucosa.

- Hyposmia due to misdirection of air.

Signs:

- Deviation: of the nasal septum in form of C- or S- shape or spur.
- Anterior dislocation of caudal edge of nasal septum.

Complications:

- Sinusitis: due to impaired ventilation and drainage of sinuses.
- External nasal deformity.
- Otitis media due to impaired ventilation.

Treatment: Surgical correction by submucous resection or septoplasty when deviated septum causes symptoms or complications.

Complications of septal surgery:

1. Septal perforation: due to bilateral trauma of the mucoperchondrial flaps opposite each other.
2. Septal haematoma and septal abscess.
3. Adhesions and synachiae: between septal mucosa and lateral nasal wall.
4. Saddle nose: due to over resection of the dorsal border of the septal cartilage.
5. Dropped nasal tip: due to resection of the caudal margin.

![Fig. (20) Types of D.S](image)

**Septal Haematoma** Fig. (21)

It is collection of blood between the mucoperichondrial layer and the septal cartilage. It may be unilateral or bilateral

Aetiology:

- Nasal trauma, septal operation e.g. after SMR or spontaneous in blood diseases.

Clinical picture:

Symptom: Bilateral persistent nasal obstruction.

Signs: Bilateral smooth red soft fluctuant swelling of the nasal septum.
Fig. (21) Septal Haematoma

Complications:
- Secondary infection leading to septal abscess (danger area of face).
- Organization of haematoma leading to permanent thickening of the septum.

Treatment:
- Septal incision and drainage of haematoma and anterior nasal pack to avoid recollection.
- Antibiotics and anti-inflammatory drugs.

**Septal Abscess:**
Collection of pus between mucoperichondrium and septal cartilage.

**Aetiology:** Secondary infection of septal haematoma.

**Clinical picture:**

**Symptoms:**
- Fever, general malaise and nasal pain.
- Bilateral persistent nasal obstruction.

**Signs:**
- Tenderness over the nasal dorsum.
- Bilateral smooth red pale soft fluctuant tender swelling of the septum.

**Complications:**
- Cavernous sinus thrombophlebitis.
- Septal perforation due to cartilage and mucosal necrosis.
- Saddle deformity due to cartilage necrosis.

**Treatment:**
- Aggressive broad spectrum antibiotics.
- Incision and drainage (on one side).

**Septal Perforation** Fig. (22)

Septal perforation is a hole in the nasal septum connecting the two nasal cavities. It may be anterior or posterior. Its size varies from small to big perforation.
Aetiology:

I. Anterior (cartilaginous) septal perforation:
   1. Traumatic: nasal picking, cautery to stop epistaxis, septal surgery (SMR), neglected rhinolith.
   2. Inflammatory: septal abscess, lupus, leprosy.
   3. Poisons: Cocaine, chrome fumes.

II. Posterior (bony) septal perforation: Syphilis (gumma).

Clinical picture:
- Whistling sound with small anterior perforation.
- Crust formation and epistaxis.
- Asymptomatic. Usually with posterior perforation.

Treatment:
- If symptomless no interference.
- Nasal douching with alkaline nasal wash to separate crusts.
- Closure by silastic button obturator.
- Surgical closure by mucosal flaps or grafts for symptomatic small perforation.

Cysts of the Sinuses

Mucocele of the Sinuses: Fig. (23)
Definition:
Cystic expansion of a sinus due to accumulation of mucus. Most commonly occurs in the frontal and ethmoid sinuses.

Aetiology:
1. Obstruction of the sinus ostium (due to chronic sinusitis, polypi or benign tumours) with accumulation of mucus within the sinus.
2. Obstruction of a mucus gland in the mucosal lining of the sinus.
Clinical picture:
Painless slowly growing bony swelling. When the bone is thinned out, it gives an egg shell crackling sensation. Frontal mucocele in the medial part of the roof of the orbit. Ethmoidal mucocele at the medial canthus. Large mucocele produces proptosis and diplopia.

Investigations:
CT scan: shows the opacification and expansion of the sinus.

Treatment:
Endoscopic sinus surgery for drainage and marsipulization of the mucocele.

**Pyocele of the sinuses:**
Aetiology: Secondary infection of mucocele with accumulation of pus.
Clinical picture: Similar to mucocele but the swelling is painful and tender, and the overlying skin is red and oedematous with purulent nasal discharge.
Investigations: Similar to mucocele.
Complications:
- Sinus: if it ruptures through the skin.
- Like complications of sinusitis.

Treatment:
1. Aggressive broad spectrum antibiotics.
2. Endoscopic sinus surgery for drainage and marsipulization of the pyocele.
3. Treatment of the cause.

**Odontogenic Cysts (Cysts of Dental Origin)**

**Dentigerous Cyst** Fig. (24)
Age: Young adults.
Aetiology: Cystic degeneration of the tooth follicle of unerupted or supernumerary tooth.
Clinical picture:
  Painless slowly growing swelling of the maxilla with egg-shell crackling sensation.
  Deficient tooth count with missing tooth in relation to the swelling, or normal tooth count in case of supernumerary tooth.

Investigations: Plain X-ray: Unerupted or supernumerary tooth appears inside the cyst.

Treatment: Enucleation of the cyst and associated tooth by sublabial incision.

Dental Cyst Fig. (25)
Age: Adults.
Aetiology: Cystic degeneration of chronic periapical dental infection of infected or carious tooth.

Clinical picture:
  1. Painful slowly growing swelling of the maxilla with egg-shell crackling sensation.
  2. Normal tooth count with infected tooth in relation to the swelling.

Investigations: Plain X-ray: Infected tooth appears outside the cyst or in its wall.
Treatment: Enucleation of the cyst and remove the infected tooth by sublabial incision.
Neoplasms of the Nose and Sinuses
Benign Tumours

Osteoma
The most common benign tumour of the nose and sinuses commonly in frontal sinus and followed by ethmoid.
Types:
- Compact (ivory) osteoma: common in the frontal sinus.
- Cancellous osteoma: common in the ethmoid sinuses.
Clinical picture:
- Young adults (15-30 years)
- Commonly asymptomatic and discovered accidentally by X-ray.
- Slowly growing painless bony swelling and may be presented by swelling of frontal sinus in the medial part of orbital roof and in ethmoidal sinus at the inner canthus.
- Facial pain and headache.
Complications:
- Secondary mucocele or pyocele due to obstruction of ostia.
- Proptosis.
Investigations:
- Plain X-ray shows a well defined bony shadow.
- CT scan define the extent of osteoma.
Treatment: If symptomatic excision by:
- External ethmoidectomy for ethmoid osteoma.
- Frontal osteoplastic flap for frontal osteoma.

Haemangioma
Types:
1. Capillary haemangioma: Bleeding polypus of the nasal septum. Soft reddish polyp on the anterior part of septum that bleeds easily on touch. 
   Treatment: Excision and cauterization of the base of polyp.
2. Cavernous haemangioma: on the lateral nasal wall.
   Treatment: Laser photocoagulation.

Inverted Papilloma
A locally destructive tumour which arises from the epithelial lining of the nose. Males more than females (5:1).
Pathology:
- The proliferating epithelial cells grow into the underlying stroma, (inverting). The surface epithelium consists of alternating layers of ciliated columnar or stratified squamous but with inact basement membrane.
- It arises from the lateral nasal wall.
- Malignant transformation: 5%
- Recurrence: is common after surgery.
Clinical picture:
Symptoms: Unilateral nasal obstruction, discharge and epistaxis.
Signs: Unilateral polypoidal fleshy reddish grey nasal mass.
Investigations:
1. CT scan and MRI: to assess tumour extension to sinuses.
2. Biopsy: to confirm the diagnosis.
Treatment:
- Wide surgical excision by using endonasal endoscopic approach lateral rhinotomy or mid-facial degloving.

Malignant Tumours

Squamous Cell Carcinoma
The commonest malignant tumour of the sinuses.
Sex and age: most commonly males above 40 years.
Site: most commonly the lateral nasal wall, the maxillary and the ethmoidal sinuses.
Predisposing factors:
- Exposure to formaldehyde, nickel and wood dust

Clinical picture: Fig. (26)
The manifestations are due to spread of the tumour beyond the sinus.
They are usually unilateral.
1. Nasal manifestations:
   - Nasal obstruction.
   - Epistaxis.
   - Offensive nasal discharge.
   - Ulcerating friable nasal mass.
2. Oral manifestations:
   - Persistent dental pain in the upper premolar and molar teeth.
   - Loose teeth.
   - Platatal swelling and ulceration.
   - Oro-antral fistula.
3. Orbital manifestations:
   - Proptosis.
   - Diplopia or vision loss.
   - Epiphora (invasion of the naso-lacrimal duct).
4. Facial manifestations:
   - Facial paraesthesia (invasion of the infra-orbital nerve).
   - Cheek swelling.
5. Neck mass:
   - Enlarged upper deep cervical lymph nodes.
Fig. (26) Spread of carcinoma of maxilla

Investigations:
1. CT scan and MRI: assess tumour extension and lymph node involvement.
2. Biopsy: from nasal mass or endoscopically from maxillary sinus.

Treatment: Combined surgery and postoperative radiotherapy.
- Surgical excision:
  Through the following surgical options:
  - Cancer maxillary sinus: - partial maxillectomy via lateral rhinotomy or mid-facial degloving or total maxillectomy through Weber-ferguson's incision. Total maxillectomy entails total removal of the maxilla, including the hard palate. The palate is reconstructed by dental prosthesis.
  - Cancer ethmoid sinus → Cranio-facial resection for tumours extending to ethmoid cavity.
  - Orbit exenteration: if the tumour invades the orbit.
  - Radical neck dissection: in presence of lymph nodes involvement.
Anatomy of the Pharynx

- It is the upper part of the respiratory and digestive airways. It is about 12-15 cm in length in adults. It extends from the base of the skull superiorly to the level of the sixth cervical vertebra, at the lower border of the cricoid cartilage. It is continuous with oesophagus inferiorly. The pharynx opens anteriorly into the nose, mouth and larynx from above downwards. So it is divided into three parts: (1) nasopharynx, (2) oropharynx and (3) laryngopharynx Fig. (1).

Fig. (1) Anatomy of the pharynx

1. **Nasopharynx:** (Post-nasal space)
   - Lies behind the nasal cavities in front of 1st cervical vertebra. Extends from the basi-sphenoid to the level of lower border of soft palate. Communicates anteriorly with the nasal cavities through the choanae, and below with the oropharynx through the velo-pharyngeal sphincter. The adenoid (Nasopharyngeal tonsil) lies at the junction of the roof and posterior wall. The nasopharyngeal opening of the Eustachian tube lies in the lateral wall 1 cm behind the posterior end of inferior turbinate. Rosenmuller fossa is a small recess behind the opening of Eustachian tube and tubal tonsil lies behind fossa of Rosenmuller.

2. **Oropharynx:**
   - Lies behind the oral cavity in front of 2nd and 3rd cervical vertebrae and is bounded above by the soft palate, below by the upper border of the epiglottis. The palatine tonsils lie in the lateral walls, between the anterior and posterior pillars.

3. **Laryngopharynx (Hypopharynx):**
   - Lies behind the larynx in front of 4th - 6th cervical vertebrae and extends from the tip of epiglottis to the upper end of oesophagus. It consists of the following regions: Postcricoid region behind the cricoid cartilage, posterior pharyngeal wall and two pyriform fossae on either side of the larynx. The pharyngeal wall consists of layers; from inwards to outwards:
Mucous membrane: Consists of stratified squamous epithelium, except the nasopharynx, which is columnar ciliated epithelium.

Subepithelial lymphoid tissue: The pharynx has a large amount of subepithelial lymphoid tissue forming Waldeyer's ring Fig. (2) consists of large aggregations of this tissue and smaller ones. The largest are the nasopharyngeal tonsil on the roof of the nasopharynx (Adenoids) and the two palatine tonsils in the oropharynx. The smaller aggregations are the tubal tonsils above and behind the pharyngeal opening of the Eustachian tube and the lingual tonsils.

Pharyngeal aponeurosis: completed above by the pharyngobasilar fascia.

Muscular layer: consists of the superior, middle and inferior constrictor muscles which overlap each other from below upwards and are inserted into the median raphe.

Buccopharyngeal fascia: attached to the prevertebral fascia by a median raphe.

Palatine (faucial) tonsils: two ovoid masses of lymphoid tissue and each tonsil has upper and lower poles. The medial surface is free and lined with stratified squamous epithelium that invaginates into the tonsil forming 12-15 crypts. The largest crypt (crypta magna) is present near the upper pole of the tonsil. The lateral surface is covered by the tonsillar capsule and is related to the superior constrictor, buccopharyngeal fascia and medial pterygoid muscle. Lingual tonsil lies between the lower pole of palatine tonsil and the tongue.

Arterial supply of the palatine tonsil: Fig. (3)

- All arteries are branches from the external carotid artery.
  - Tonsillar of the facial artery (main artery of the tonsil).
  - Ascending palatine of the facial artery.
  - Ascending pharyngeal of the external carotid artery.
  - Dorsalis linguae of the lingual artery.
  - Descending palatine of the maxillary artery.

Venous drainage:

- This forms pharyngeal plexus of veins, which communicates above with the pterygoid plexus and drains into the common facial and internal jugular veins. Paratonsillar vein which runs on the lateral surface of palatine tonsil.
Fig. (3) Arterial supply

Lymphatic drainage:
- Nasopharynx: Retropharyngeal LN to upper deep cervical LN.
- Oropharynx: Upper deep cervical LN.
- Laryngopharynx: Upper and lower deep cervical LN.

Nerve supply:
Sensory nerve supply:
- Nasopharynx by 5th cranial nerve.
- Oropharynx and hypopharynx by 9th and 10th cranial nerves.

Motor nerve supply:
- From the cranial part of the accessory nerve which is distributed through the pharyngeal branches of the vagus nerve. It supplies all the muscles of the pharynx except: Stylopharyngeus muscle and tensor palati muscle.

Functions of the Pharynx

1. Respiration:
   - The pharynx forms part of the upper respiratory tract between the nasal cavities and the larynx.

2. Deglutition:
   - Food is transferred from the mouth to the stomach by an orderly sequence of co-ordinated movements of the muscles of the mouth, pharynx and oesophagus.

3. Phonation:
   - The pharynx, mouth and nose act as a resonating cavities which modify the basic laryngeal sounds. The pharynx, soft palate, tongue and lips, all play a part in the articulation of the various sounds which make up speech.

4. Protective function:
   - The subepithelial lymphoid tissue (Waldeyer's ring) acts as a defense line at the entrance of the air and food passages. This is evidenced by: formation of lymphocytes, formation of antibodies, thus reinforcing the mucosal immunity of the aero-digestive tract, localization of infection and they act as filters to the upper respiratory passages.
Examination of the Pharynx

Inspection: of lips and buccal mucosa, palate, tongue and floor of the mouth, and oropharynx.
Palpation: The floor of the mouth and cheek must be examined by bimanual specially submental and submandibular swellings.
Nasal Examination first by post-nasal mirror (posterior rhinoscopy), flexible fiberoptic endoscopy (under local anaesthesia) and rigid endoscope introduced through the nose or mouth (under L.A. or G.A.) using different angles (70° or 90°).
Examination of the oropharynx: examination is done through an open mouth with tongue depressor, and digital palpation whenever malignancy is suspected.
Examination of the laryngopharynx (hypopharynx) by indirect laryngoscopy using laryngeal mirror, flexible nasolaryngoscope, rigid endoscope (90° or 70°) and direct laryngoscopy under general anaesthesia by microscope.

Investigations of the Pharynx

Radiological investigations:
- Plain X-ray: demonstrates the outlines of the upper aerodigestive tract, foreign bodies, occasionally cysts and tumours.
- Barium swallow: demonstrates the lumen of the hypopharynx, diagnosis of pharyngeal pouches and achalasia of the cardia.
- CT scan and MRI: demonstrate the pharynx and surrounding spaces.
- Angiography: (alone or with MRI) shows the vascularity of some vascular lesions, e.g. angiofibromas and glomus jugulare tumours.

Diseases of the Nasopharynx

Adenoids:
- Enlargement of the nasopharyngeal tonsil (adenoids) sufficient to produce symptoms. Physiological enlargement occurs at the age of 3 to 7 years. Pathological enlargement is due to simple inflammation during recurrent attacks of upper respiratory tract infection. The size of the mass relative to that of the nasopharyngeal space is the important factor in producing the symptoms.
Clinical picture:
- Nasal obstruction: this leads to mouth breathing, snoring, difficulty in feeding specially in infants, hyponasal voice and obstructive sleep apnea syndrome (OSAS).
- Nasal discharge:-
  - Mucoid or mucopurulent (egg-white plug of mucus seen behind the uvula on gagging), the post-nasal discharge leads to recurrent nocturnal cough and excoriation of the nasal vestibule.
• Eustachian tube obstruction: predisposes to secretory otitis media, recurrent acute suppurative otitis media and later on chronic otitis media may occur.
• Adenoid facies: Fig. (4): May develop with pinched nostrils, prominent incisors, open mouth leading to spongy gums and dental caries, high arched palate and flat or pigeon chest.
  † Mental dullness and apathetic looking due to hearing loss, chronic hypoxia and look of adenoid face
  † Nocturnal enuresis.

Fig. (4) Adenoid facies.

Diagnosis:
  • Posterior rhinoscopy: can show the adenoids but difficult.
  • Digital palpation: to feel the adenoids but should be avoided.
  • Endoscopic examination is the best method if possible.
  • Lateral X-ray nasopharynx for soft tissue is good.

Complications:
  • Descending infection: secretory otitis media, suppurative otitis media, sinusitis, rhinitis, laryngitis and bronchitis.
  • Obstructive sleep apnea syndrome (OSAS).
  • Impaired concentration and excessive day time sleepiness.

Treatment:
  • Adenoidectomy.

Adenoidectomy Operation
Indications:
  • Enlargement of the adenoids sufficient to produce symptoms.
Contraindications:
  • Systemic: Bleeding tendency as haemophilia and purpura.
  • Local: Cleft palate.

Technique:
  • General anaesthesia.
  • Position is supine with extended neck by pillow under shoulders
  • The adenoid curette is passed behind the soft palate into the nasopharynx to shave the adenoids mass Fig. (5). A pack is put for few minutes to stop bleeding then removed.
Complications: Similar to tonsillectomy.
  • Other complications: include trauma to the uvula, soft palate, and Eustachian cushions.

**Tumours of the Nasopharynx**

**Nasopharyngeal Angiofibroma**

• The commonest benign tumour of the nasopharynx.
• Age: 10-20 years.
• Sex: Only males.
• Site of origin: Fig. (6) Periosteum of the roof of nasopharynx or margin of the sphenopalatine foramen in the posterior part of lateral nasal wall.
• Histopathology: Large sinusoidal vascular spaces with no muscular coat, separated by fibrous tissue stroma.
• Behaviour: Although histologically benign, but it is clinically aggressive in behaviour because of its extension by pressure necrosis and displacement of surrounding structures, into nasal cavity, orbit, pterygopalatine fossa, infratemporal fossa, cheek and intracranial cavity.
• Spontaneous regression: may occur at the age of 25 years.

Symptoms:
• Nasal obstruction with recurrent severe attacks of epistaxis and hyponasality of speech.
• Aural: Hearing loss due to Eustachian tube obstruction leads to secretory otitis media.
• Proptosis and Swelling of cheek due to extension.

Signs: by endoscopic examination as following: unilateral nasal pinkish mass, which bleeds on touch with nasopharynx geal: smooth pinkish lobulated mass covered by intact mucosa. Secretory otitis media and conductive deafness may present.

N.B. digital palpation should be avoided as it causes profuse bleeding.

Signs of extension of the tumour:
• Widening of the nasal bones and broadening of the nose (frog face deformity), unilateral swelling of the cheek, unilateral proptosis and manifestations of intracranial extension.
Fig. (6) Angiofibroma

Investigations:
- CT scan and MRI: To confirm diagnosis and to assess tumour extension.
- Carotid angiography: To identify the feeding vessels.
- Biopsy: usually not necessary to avoid severe bleeding. When indicated, should be done in the operating room with facilities to control bleeding.

Differential diagnosis:
- Nasopharyngeal carcinoma: CT scan is diagnostic.
- Antro-choanal polyp: never bleeds and CT scan is diagnostic.

Treatment:
- Better preoperative embolization of the feeding vessels to minimize intraoperative bleeding.

Surgical excision: Transnasal endoscopic excision, transpalatal, lateral rhinotomy or mid-facial degloving approach.

Nasopharyngeal Carcinoma
- The commonest malignant tumour of the nasopharynx.
Age: 40-60 years. Sex: Males more than females. (3:1)
Race: commonest in Far East countries especially China, due to genetic and enviromental factors.

Pathogenesis:
- Epstein-Barr virus as evidenced by raised antibody titre, genetic predisposition and enviromental factors: as formaldehyde and wood dust.

Pathology:
- Site of origin:
  - Fossa of Rosenmuller (the commonest site), lateral, superolateral or posterior wall of the nasopharynx.
- Histopathology:
  - Squamous cell carcinoma (the commonest), anaplastic carcinoma with infiltration of lymphocytes (Lympho-epithelioma) or adenocarcinoma and mixed salivary tumour (from minor salivary glands).

Spread:
- Direct spread:
  - Forwards to nasal cavity.
  - Forwards and laterally to the orbit.
  - Upwards to intra-cranial cavity.
  - Laterally to Eustachian tube and para-pharyngeal space.
• Lymphatic spread:
  ß Early, common and usually bilateral to the retro-pharyngeal lymph
  nodes (cannot be palpated clinically) and upper deep cervical lymph
  nodes.
• Distant metastasis: in advanced cases to the liver, lung, bone and
  brain.
Clinical Picture:
• Nasal obstruction and mild epistaxis with or without unilateral or
  bilateral upper deep cervical lymph node metastasis. This may be the
  earliest and only manifestation before the primary in some of cases as the
  nasopharynx is a silent area. Unilateral conductive deafness due to
  secretory otitis media.
• The neurological manifestations are: 3\textsuperscript{rd}, 4\textsuperscript{th} and 6\textsuperscript{th} cranial nerve
  affection: causing squint, ptosis and ophthalmoplegia. 5th cranial nerve
  affection: the earliest, causing facial pain. 9\textsuperscript{th}, 10\textsuperscript{th} and 11\textsuperscript{th} cranial nerve
  affection (jugular foramen syndrome): causing palatal, pharyngeal and
  laryngeal paralysis. 12\textsuperscript{th} cranial nerve affection: causing tongue paralysis.
• Trotter's triad: is diagnostic for nasopharyngeal carcinoma. It consists of:
  1-unilateral conductive hearing loss (secretory otitis media.
  2- Ipsilateral earache and facial pain (trigeminal nerve).
  3- Ipsilateral paralysis of the soft palate.
Investigations:
• CT scan and MRI: to assess tumour extension and nodal metastasis.
• Biopsy by transnasal endoscope: to confirm diagnosis.
• Chest X-ray, abdominal ultrasound and bone scan for distant metastasis.
Treatment:
• Radiotherapy for the nasopharynx and neck.
• Combined chemo-radiotherapy.

Diseases of the Oropharynx

Inflammation of Pharynx

Pharyngitis
• Inflammation of the mucosal lining of the pharynx.

Acute Nonspecific Pharyngitis:
• Is viral infection in association with common cold and influenza,
  followed by secondary bacterial infection.
Clinical picture
Symptoms: Fever, headache, malaise, sore throat, dysphagia and otalgia.
Signs: diffuse congestion of the pharyngeal mucosa and tonsils with enlarged
  tender cervical lymph nodes.
Treatment:
• Rest, fluids, analgesics, antipyretics, antibiotics, and antiseptic gurgle.
Diphtheria (Faucial Diphtheria)

- Acute specific infectious disease caused by Corynebacterium diphtheria. It is a serious condition; fortunately now it is very rare disease due to obligatory immunization.

Age: Children 2-5 years.

Aetiology:
- Causative organism: Gram positive bacilli Corynebacterium diphtheria.
- Transmission: Droplet infection from patient or carrier with incubation period: 2-5 days.

Pathology: It is an acute membranous inflammation of the mucous membrane. There is a pseudomembrane at the site of infection. The organism causes necrosis of the superficial layer of epithelium. An adherent false membrane is formed. It is composed of necrotic mucosa, fibrin, bacteria, RBCs and inflammatory cells. Systemic: Toxemia due to absorbed exotoxin. It has high affinity to cardiac and neural tissues and may affect kidney and liver.

Clinical picture:

Symptoms:
- Insidious onset of low grade fever, sore throat, severe toxemia and malaise.

Signs:
- Moderate fever (38-39°C) with rapid weak pulse not synchronous with the temperature.
- False membrane (pseudomembrane) is formed on the tonsils, pharynx and may extend to the larynx. The membrane is greyish-yellow in colour and usually unilateral. It is adherent to the tissues and recurs rapidly when removed.
- Bilateral enlarged cervical glands (bull's neck).

Investigations:
- Throat swab from the membrane is examined for diphtheria bacilli by direct smear stained with gram stain and culture on Loffler's serum or blood agar.

Differential diagnosis: From other causes of membrane on the tonsil and pharynx: Acute follicular tonsillitis, Vincent's angina, Moniliasis or Blood diseases (Agranulocytosis, acute leukaemia, infectious mononucleosis)

Fig. (7) Acute tonsillitis and diphtheria
Complications:
- Diphtheria exotoxins have a special affinity to nervous, cardiac and renal tissue.
- Paralysis may occur after 2-3 weeks.
  - **Head and neck:**
    - Paralysis of the soft palate leads to nasal regurge, nasal tone, and uvula deviation to the normal side.
    - Paralysis of the eye muscles cause lack of accomodation, diplopia and squint.
    - Paralysis of the pharynx causes dysphagia.
    - Paralysis of the larynx leads to stridor.
  - **Chest muscles:**
    - Including the diaphragm and intercostal muscles cause respiratory failure.
  - Heart failure may occur early due to toxic myocarditis or late due to vagal neuritis.
  - Respiratory obstruction by the membrane of diphtheria.
  - Acute nephritis (Albuminuria).

Treatment:
- Antitoxic serum: 40000 - 100000 units according to the severity and extension of the diphtheric membrane and the weight of the child is given immediately if diphtheria is suspected (S.C., I.M., or I.V.).
- Antibiotics: penicillin acts against the organism.
- Rest in bed.
- Isolation in a fever hospital until 3 successive swabs are -ve.
- Treatment of complications e.g tracheostomy for laryngeal obstruction.

Prophylaxis:
- Passive immunization: 3000-10000 I.U. of antitoxic serum given for contacts.
- Active immunization is usually given in the triple vaccine D.P.T. (diphtheria, pertussis and tetanus) to infant. It is given in 3 doses one every 2 months, starting at the age of 2 months with a booster dose at 1.5 and 6 years old.
- Tonsillectomy in diphtheric carrier.

**Vincent's angina:**
Aetiology: The disease is usually associated with two organisms: a fusiform bacillus and spirochate (borrelia vincenti).
Clinical pictures:
Symptoms: severe sore throat, mild fever, dysphagia, foetor oris and malaise.
Signs:
- Unilateral greyish yellow membrane of the tonsil, which rarely extend to the pillars and soft palate, and ulcer with deep and irregular edge.
- Enlarged tender cervical lymph nodes.
Differential diagnosis: Acute follicular tonsillitis
Investigations: Throat swab is cultured and examined for the organisms.
Treatment:
- Hydrogen peroxide mouthwash and gurgle.
- Penicillin and Metronidazole (Flagyl)

**Infectious Mononucleosis** (Glandular fever)
- A viral infection (Epstein Barr virus) which leads to marked increase in the mononuclear cells (lymphocytes and monocytes).

Clinical picture: There are three types of the disease:
- Glandular type: Pyrexia and malaise for few days followed by generalized lymphadenopathy. The tonsils may be red but there is no sore throat.
- Anginose type: Sore throat and dysphagia for 2 to 3 weeks, followed by a membranous affection of the throat resembling Vincent's angina followed by generalized lymphadenopathy.
- Febrile type: Pyrexia, headache and general malaise, a week later a maculo-papular rash appears, and after other two weeks followed by generalized lymphadenopathy.

Investigations:
- Blood picture: Leucocytosis with relative monocytosis and lymphocytosis.
- Serological tests: Positive monospot test and positive Paul-Bunnell test.

Treatment:
- Rest, fluids, analgesics, antipyretics, broad spectrum antibiotics and avoid ampicillin because it may cause skin rashes.

**Moniliasis:**
- This is the commonest fungal affection of the throat. It occurs in debilitated patients. It is caused by Candida albicans

Clinical picture:
Symptoms: There is sore throat and slight dysphagia but no fever.
Signs: Milky white patches are scattered on the inner side of the cheek, lateral margin of the tongue, palate and faucial pillars.

Treatment:
- Local antifungal: Nystatin and systemic antifungal in severe cases.
- Gentian violet 1% solution may be used as paint.
Acute Pharyngitis associated with Blood Diseases

1. **Agranulocytosis**: Marked reduction of the neutrophils in the blood due to depression of the bone marrow by cytotoxic drugs, chloramphenicol or following irradiation and may be idiopathic.

   Clinical picture:
   - Extensive ulcerations and false membrane formation on the tonsils, pharyngeal and buccal mucosa surrounded by little inflammatory reaction and the condition may be fatal.

   Investigations:
   - Blood picture: Leucopenia (agranulocytosis with relative lymphocytosis).
   - Bone marrow aspiration: is diagnostic.

   Treatment:
   - Stop the drug and causative factor, isolation to avoid infection, antibiotics and fresh blood transfusion.

2. **Acute Leukaemia**: Increase in the number of white cells with the appearance of immature cells in the peripheral blood

   Clinical picture:

   Investigations:
   - Blood picture: marked leucocytosis with increased blast cells (immature cells), anaemia, and thrombocytopenia.
   - Bone marrow aspiration: is diagnostic.

   Treatment:
   - Isolation of the patient, fresh blood transfusion, antibiotics and cytotoxic drugs.
   - Bone marrow transplantation.

3. **Acquired Immuno-Deficiency Syndrome (AIDS)**

   Aetiology: Human immuno-deficiency virus (HIV) that invades T lymphocytes causing impaired immunity.

   - Mode of transmission: Sexual contact and blood transfusion.

   - Otolaryngological manifestations:
     - **Pharynx**: Oral candidiasis, herpetic ulcers and oropharyngeal ulcers.
     - **Nose**: Chronic rhino-sinusitis and invasive fungal sinusitis.
     - **Larynx**: Viral and fungal laryngitis.
     - **Ear**: Acute otitis externa, media & SNHL.

   Investigation:
   - ELISA (Enzyme-linked immunosorbent assay): detects only antibodies to HIV.
Chronic Pharyngitis

Chronic non-specific Pharyngitis

Aetiology:
- Recurrent acute pharyngitis, chronic sinusitis, chronic tonsillitis, gastro-oesophageal reflux, smoking and alcohol.

Clinical picture:
Symptoms:
- Sore throat with sensation of throat dryness and irritation, frequent hawking and hemming.

Signs:
- Catarrhal pharyngitis: Congestion of pharyngeal mucosa.
- Hypertrophic pharyngitis: Hypertrophied lateral pharyngeal bands and submucosal lymphoid follicles on posterior pharyngeal wall.
- Atrophic pharyngitis: Pharyngeal mucosa is dry and glazed with crusts.

Treatment: Treatment of predisposing factors.

Chronic Specific Pharyngitis

1. Tuberculosis:
Aetiology: usually secondary to pulmonary TB.
Clinical picture: Pharyngeal mucosa is pale with painful shallow ulcer with undermined edges and caseous yellow floor.
Treatment: Anti-tuberculous therapy.

2. Scleroma: (pharyngoscleroma)
Aetiology: usually secondary to rhinoscleroma.
Clinical picture:
- May be an atrophic stage with dry glazed mucosa or nodular stage with painless non ulcerating pinkish nodules or fibrotic stage with fibrosis and pharyngeal stenosis and soft palate is fibrotic with short uvula.
Treatment: similar to rhinoscleroma.

Pharyngeal Ulcers

- Ulcer means loss of continuity of pharyngeal mucosa.
Aetiology:
- Traumatic ulcers:
  - Mechanical: Mucosal laceration by F.B.
  - Chemical: Caustic ingestion.
- Inflammatory ulcers:
  - Acute specific pharyngitis:
    - Viral: Infectious mononucleosis, acquired immune deficiency disease (AIDS), herpes simplex, zoster and measles (Koplik’s ulcers)
- Bacterial: Diphtheria & Vincent's angina.
- Fungal: Moniliasis.

Chronic specific pharyngitis:
- Tuberculosis (bluish undermined edge, yellow caseous floor).
- Syphilis (deep, punched out edge and dirty yellow brown necrotic floor).

• Ulcers of systemic diseases:
  - Blood diseases:
    - Agranulocytosis.
    - Leukaemia.
  - Diseases of unknown aetiology:
    - Recurrent aphthous ulcers (commonest).
    - Behcet's ulcer.
  - Skin diseases:
    - Pemphigus and lichen planus.
  - Drugs and toxins:
    - Lead, mercury poisoning and antidepressant drugs.

• Neoplastic ulcers:
  - Malignant tumours:
    - Squamous cell carcinoma (everted edge, indurated base and necrotic floor).

Tonsillitis

Acute Tonsillitis

• Acute non-specific inflammation of the palatine tonsils.

Aetiology:
• Causative organism: Group A β-haemolytic Strept. (the commonest), Strept. pneumoniae, Staph. aureus and Haemophilus influenzae.

Symptoms:
• Sore throat, dysphagia and foetor oris associated with ear ache.
• High fever, malaise and arthralgia.

Signs:
• High fever with tachycardia proportionate to the rise of temperature.
• Local: manifestations are according to the types of tonsillitis, if acute catarrhal tonsillitis: shows congested tonsils. If acute follicular tonsillitis: shows congested tonsils with yellowish spots on the surface, spots may coalesce to form yellowish membrane which can be easily removed. If acute parenchymatous tonsillitis shows markedly swollen congested oedematous tonsils and enlarged tender cervical lymph nodes.

Complications: The most important are:
• Chronic tonsillitis.
• Peritonsillar abscess.
• Parapharyngeal and retropharyngeal abscess.
• Acute glomerulo-nephritis.
• Acute rheumatic fever, often after a latent period of about 6 weeks.
Differential diagnosis: From many conditions causing acute pharyngitis, the most important are:

- Scarlet fever.
- Diphtheria.
- Vincent's angina.
- A granulocytosis.
- Glandular fever (Infectious mononucleosis).

Treatment:
- Rest, fluids, soft diet, analgesics, antipyretics and mouth wash.
- Systemic antibiotics: penicillin or amoxicillin for 10 days.

**Chronic Tonsillitis**

- Chronic inflammation of the palatine tonsils due to recurrent acute tonsillitis with inadequate antibiotic treatment.

Pathological types: Follicular, hypertrophic or atrophic tonsillitis.

Clinical picture:

Symptoms:
- Local:
  - Recurrent acute tonsillitis and persistent sore throat with frequent hawking and hemming, bad mouth odour due to accumulation of purulent material in the crypts, or obstructive sleep apnea in hypertrophic tonsillitis.
- General: Symptoms of septic focus: General ill health, fatigue, anorexia, low-grade fever, headache and arthralgia.

Signs of chronic tonsils are:
- Irregularity of the size and shape of tonsils may be unequal, hypertrophic or atrophic.
- Congestion of the anterior pillars of tonsils in absence of acute infection.
- The crypts ooze pus on pressure.
- Persistent enlargement of jugulodigastric lymph nodes.

Complications:
- Rheumatic fever and acute glomerulonephritis.

Treatment: Tonsillectomy.

**Tonsillectomy Operation**

Definition:
- Excision of the palatine tonsils.

Indications:
- Local indications:
  - Repeated attacks of acute tonsillitis (more than 5 attacks per year).
  - Marked enlargement of the tonsils interfering with deglutation and respiration (obstructive sleep apnea).
  - One attack of peritonsillar abscess (Quinsy).
  - Signs of chronic tonsillitis.
  - Suspected tumour of the tonsil.
  - Carrier state particularly in streptococci and diphtheria carriers.
• Focal indications:
  † Persistent cervical lymphadenitis when T.B. infection is suspected.
  † Recurrent attacks of rheumatic fever, if they are associated with attacks of tonsillitis.
  † Attacks of acute glomerulonephritis, if they are associated with attacks of tonsillitis.
• As a part of other operations:
  † In some cases of uvulopalatopharyngoplasty (UPPP) for treatment of obstructive sleep apnea syndrome.
  † Excision of long styloid process or glossopharyngeal neurectomy through the tonsillar bed (9th neuralgia).

Contraindications:
• Absolute:
  † Blood diseases as haemophilia, purpura, leukaemia and agranulocytosis.
  † When enlargement is a part of a systemic disease as lymphoma and leukaemia.
• Relative:
  † During acute attacks on top of chronic.
  † During upper respiratory tract infections.
  † During active rheumatic fever.
  † During epidemics of poliomyelitis.
  † During menses.
  † Cleft palate.
  † Age: less than 3 years.

Investigations:
  † Complete blood picture.
  † Bleeding time
  † Clotting time
  † Prothrombin time.
  † ESR.

Premedications:
  † Sedation in the night before operation.
  † Fasting for 6 hours before the operation.

Anaesthesia:
• General with endotracheal tube.

Position:
  † The patient lies in the supine position with a pillow under his shoulders for neck extension.
  † The surgeon sits on the head of the table.
Technique: Fig. (8)

- **Tonsillectomy by dissection:** This is the most common technique.
  - The tonsil is grasped by a tonsil holding forceps.
  - Incision along the anterior pillar.
  - Dissector is used to dissect the tonsil out of its bed with its capsule.
  - A straight artery forceps is used to control the bleeding points.
  - Control the bleeding by bipolar diathermy forceps or stitches.
- **Tonsillectomy by other techniques:**
  - By CO2 laser, radiofrequency or coblation technique.

![Fig. (8) Steps of dissection tonsillectomy.](image)

Post-operative care:
- **Extubation:** The endo-tracheal tube is removed only after the return of the cough and swallowing reflexes to avoid aspiration.
- **Position:** Fig. (9) "Post-tonsillectomy position" the patient lies on his side with the lower knee is flexed and the other one is extended. The upper arm is flexed and the hand is placed under his cheek and a small pillow is placed in front of his chest.

![Fig. (9) Post-tonsillectomy position.](image)

- **Observation for:**
  - Signs of respiratory obstruction: noisy breathing and cyanosis.
  - Signs of bleeding: Spitting of fresh blood, frequent swallowing, vomiting of dark blood, rising pulse and falling blood pressure.
  - Feeding: Starts after few hours. In the first day, the diet consists of cold fluids and semi-solids and from the 2nd to the 7th day, avoid hard, spicy and hot food.
  - Systemic antibiotics and analgesics.
Complications:

- General anaesthesia: e.g. hypersensitivity to anaesthetic drugs or succinyl choline apnea.

- Respiratory obstruction: This is the most serious complication, it may be due to early extubation, laryngeal spasm, inhalation of blood clots, vomitus or cotton piece or falling back of the tongue during recovery from anaesthesia.

- Haemorrhage (bleeding):
  - Primary: It is intra-operative blood loss. It is usually due to bad selection of the patient, bad dissection or injury of blood vessels. It can be controlled by stitches, ligation, diathermy or laser.
  - Reactionary: Occurs within the first 24 hours after return from the operating theatre, it is usually due to slipping of a ligature, oozing from the vessels after relaxation of the stretched faucial tissues on removal of the mouth gag or rise of blood pressure during recovery from anaesthesia.

Treatment: Removal of blood clots from the tonsillar pillars. If bleeding continue re-anaesthize the patient and ligate the bleeding vessel.

- Secondary: usually occurs between the 3rd and the 10th post operative days due to infection in the tonsillar fossa which causes secondary haemorrhage.

Treatment: Systemic antibiotics. Rest in bed and sedation will control bleeding in most cases. If this fails re-anaesthize the patient and suture the tonsillar pillars and pack the tonsillar fossa. Blood transfusion may be indicated.

- Pneumonia or lung abscess: may result from inhalation of blood, a tooth, or infected tissue.
Suppurations Related to the Pharynx

Fig. (10) Suppurations related to the pharynx.

Peritonsillar Abscess (Quinsy)
- It is suppuration of the peritonsillar space between the capsule of the tonsil and the superior constrictor muscle. It starts as peritonsillitis and later on suppuration occurs. Fig. (10)

Aetiology
- Acute follicular tonsillitis, when suppuration involves the crypts of the supratonsillar fossa that are closely related to the capsule of the tonsil.

Clinical picture:
- The onset is usually preceded by acute follicular tonsillitis,

Symptoms:
- Fever, headache, malaise and aneroxia.
- Local: The same symptoms of acute tonsillitis but become more severe.
  - The pain is severe progressive and throbbing and may be referred to the ear.
  - Painful dysphagia: This causes accumulation of saliva and leads to drooling.
  - Trismus: due to spasm of the muscles of mastication.
  - Torticollis: towards the affected side. It is due to spasm of the sternomastoid.
  - Change of voice (hot potato voice): due to accumulation of saliva, trismus, oedema and painful mobility of the palate.

Signs:
- Proportional fever and tachycardia.
- Local:
  - Unilateral swelling of the soft palate and medial displacement of the soft palate and oedematous uvula with limited mobility and downward and medial displacement of the tonsil. Enlarged tender cervical lymph nodes.

Differential Diagnosis:
- Tumours of the tonsil (biopsy) and carotid aneurysm (blood on aspiration).
Complications:
- Rupture: inhalation of discharge.
- Spread of infection: Parapharyngeal abscess.
- Laryngeal oedema and suffocation.

Treatment:
- Conservative: Systemic antibiotic (I.V.), anti-inflammatory analgesic, throat irrigation with warm saline in the stage of peritonsillitis before suppuration.
- Surgical: Fig. (11) Incision and drainage of the pus under local or general anaesthesia. It is opened with quinsy knife. The incision is done at either:
  - The most pointing point (the best).
  - Through the crypta magna.
  - Half cm lateral to the meeting point between a horizontal line at the base of the uvula and a vertical line at the attachment of the anterior pillar with the tongue.
- Tonsillectomy: Some surgeons do quinsy tonsillectomy or after one month to prevent abscess recurrence.

![Incision and Drainage](image)

**Fig. (11) Drainage of Peritonsillar abscess.**

**Parapharyngeal Abscess**
- It is suppuration of the parapharyngeal space Fig. (10).
- Parapharyngeal space: Potential fascial space around the pharynx. It extends from the skull base above to the superior mediastinum below. Its boundaries are: - Anteromedial is buccopharyngeal fascia of lateral pharyngeal wall. Posteromedial: is prevertebral fascia. Lateral: is ramus of mandible, parotid gland, and sternomastoid muscle.
- Its contents are: - Carotid artery, internal jugular vein, last four cranial nerves, cervical sympathetic chain and upper deep cervical lymph nodes.

Source of infection:
- Acute tonsillitis, quinsy, extension from masticator space (dental infections) and infection after tonsillectomy.

Clinical picture:
- General: Fever, aneroxia, malaise and severe pain.
• Local: Beck's triad
  ♂ External cervical swelling: at the submandibular region which is tender.
  ♂ Trismus due to spasm of the medial pterygoid.
  ♂ Internal swelling: of the lateral pharyngeal wall, pushing the tonsil medially.

Investigations:
• CT scan and MRI of neck are diagnostic.

Complications:
• Rupture of the abscess internally leading to inhalation pneumonia.
• Spread to the mediastinum leads to mediastinitis which is fatal.
• Thrombosis of the internal jugular vein with septicaemia.
• Erosion of the carotid sheath causes fatal haemorrhage.

Treatment:
• Should be hospitalized and start therapy in the early stages with aggressive parenteral broad spectrum antibiotics and analgesics.
• The abscess is drained externally through a transverse cervical incision in the neck.

Retropharyngeal Abscess
• Retropharyngeal space: Lies between the bucco pharyngeal fascia of posterior pharyngeal wall anteriorly and prevertebral fascia posteriorly.

Acute Retropharyngeal Abscess Fig. (10)

Aetiology:
• It is due to suppuration of the retropharyngeal L.N., which present on each side of the median raphe. The source of infection is usually from adenoiditis or tonsillitis.
• Age: Classically, retropharyngeal abscess is a disease of infancy and early childhood, because the glands of Henle usually become atrophic at the age of five years.

Clinical picture:

Symptoms:
• There is usually a preceeding acute infection in the throat.
• The patient then complains of fever, malaise, aneroxia, pain in the throat, painful dysphagia and difficulty in suckling, obstructive symptoms snoring, noisy breathing, nasal obstruction and mouth breathing.

Signs:
• Fever and irritability
• Local:
  ♂ Swelling of the posterior pharyngeal wall limited to one side. It is cystic swelling and pus on aspiration.
  ♂ Enlarged cervical L.N.

Differential diagnosis:
• Carotid aneurysm (pulsating and blood on aspiration) and TB spine (swelling is midline).
Complications:
• Laryngeal obstruction: leading to stridor and suffocation. It may be due to extension of the oedema to the larynx or direct compression on the larynx.
• Rupture of the abscess leads to inhalation pneumonia.
• Spread of the infection to parapharyngeal abscess.

Treatment:
• Medical treatment: Parenteral antibiotics, analgesics and hydration.
• Trans oral incision and drainage:
  § Under general anaesthesia with a cuffed endotracheal tube.
  § From inside the pharynx.
  § The head should be kept downward to avoid inhalation of pus.
• Suction of the pus.

Chronic Retropharyngeal Abscess:
• It is a cold abscess (T.B. infection) of the cervical vertebrae (Pott's disease) with extension of infection and accumulation of the caseating material behind the prevertebral fascia (prevertebral space) Fig. (10).

Age:
• It is usually in adult with pulmonary T.B.

Clinical picture:
Symptoms:
• General: T.B. toxaemia (loss of weight, night sweating, night low grade fever and loss of appetite).
• Local:
  § Severe pain in the throat and the back of the neck.
  § Mild dysphagia.
  § Cough and expectoration of bloody sputum with open pulmonary T.B.

Signs:
  § Tenderness and rigidity of the cervical vertebrae, which are kyphotic.
  § Swelling of the posterior pharyngeal wall, which is central. It is fluctuating and cystic getting out caseating material on aspiration.

Investigations:
• Chest X-ray for T.B.
• Lateral neck X-ray: destruction of the vertebrae- widening of the prevertebral space and kyphosis.
• Aspiration with direct film shows T.B. Bacilli.

Treatment:
• Local drainage: The incision is from outside parallel to the posterior border of the sternomastoid.
N.B. Trans oral drainage should be avoided as it causes secondary bacterial infection and T.B. sinus.
Ludwig`s Angina
• Cellulitis of the floor of the mouth involving the submandibular space of the neck. Suppuration is rare and occurs late.

Cause:
• Dental infection, acute pharyngitis or submandibular sialadenitis.

Clinical picture:
• General: Severe pain, fever and malaise.
• Local:
  ß External swelling: at submandibular region with signs of acute inflammation.
  ß Internal swelling: oedema of the mucosa and elevation of the floor of the mouth with protrusion of the tongue.
  ß Trismus, dysphagia, salivation and muffled voice.

Complications:
• Laryngeal oedema leads to respiratory obstruction.
• Spread to other spaces.

Treatment:
• All cases should be hospitalized. Initial therapy in the early stages is tried with parenteral broad spectrum antibiotics, hydration and analgesics.
  ß Surgical drainage is required in most of the cases. This is done through an external submandibular incision below and parallel to the body of the mandible.
  ß Tracheostomy in case of laryngeal obstruction.

Tumours of the Oropharynx

Benign:
• Papilloma: usually occurs over the pillars and uvula.
• Mixed salivary tumours: Arises in the minor salivary glands of the mucous membrane.

Malignant:
• Squamous cell carcinoma, lymphoma and sarcomas of the tonsil, palate or posterior third of tongue.

Diseases of the Hypopharynx

Foreign Body Impaction
Incidence: usually in children and elderly.
Types: Coins, fish bone, dentures or piece of meat.
Site: At the cricopharyngeus sphincter.
Symptoms: Dysphagia.
Investigation: Plain X-ray confirms the diagnosis.
Treatment: Removal by hypopharyngoscopy under general anaesthesia.
**Chronic Pharyngo-oesophagitis**

**Synonyms:** Plummer-Vinson Syndrome or Patterson-Brown-Kelly's disease.

**Definition:** Chronic inflammation of the mucous membrane of the hypopharynx and upper oesophagus.

**Aetiology:** Unknown, may be iron deficiency anaemia, vitamin deficiency or autoimmune.

**Incidence:** Commonly in females above 40 years.

**Pathology:** Atrophic mucosa and submucosal fibrosis leading to formation of webs.

**Clinical picture:**

**Symptoms:** gradual progressive dysphagia.

**Signs:**
- Angular stomatitis with fissured angles of the mouth, glossitis: (smooth glazed dry tongue), glazed atrophic mucosa of the hypopharynx and upper oesophagus with mucosal webs, splenomegaly and spooning of the nails (Koilonychia).

**Investigations:**
- Blood picture shows hypochromic microcytic anaemia.
- Gastric secretions: achlorohydria due to atrophic gastritis may be caused by vit. B12 deficiency.
- Hypopharyngoscopy: web formation and stenosis.

**Complications:**
- Submucosal fibrosis leading to web formation and stenosis.
- Pre-cancerous leads to Post-cricoid carcinoma.

**Treatment:**
- Iron and vitamin B complex by injection.
- Repeated endoscopic dilatation.
- Regular follow up to detect early post-cricoid carcinoma.

**Pharyngeal Pouch** Fig. (12)

**Definition:** a herniation of the pharyngeal mucosa between the upper oblique and lower circular muscle fibers of the inferior constrictor muscle (Killian's dehiscence).

**Incidence:** commonly in elderly males.

**Aetiology:** neuromuscular incoordination causing premature closure of the cricopharyngeal sphincter during the act of swallowing.

**Pathology:**
- The pouch is composed of mucosa and fibrous tissue only.
- The pouch sags downwards behind the oesophagus and exerts pressure on it when the pouch is filled with food.

**Clinical picture:**

**Symptoms:** (Small pouches are usually asymptomatic).
- Dysphagia: progressive and long standing.
- Regurgitation of undigested food with bad breath (Halitosis).
- Spasmodic cough on swallowing: caused by spill over of fluids to larynx.
Signs:
- Cystic swelling: usually in the left side of the neck. It may gurgle and empty on external pressure.
- Emaciation from oesophageal obstruction or malignant transformation.

Diagnosis:
- Barium swallow shows characteristic pear-shaped appearance.
- Hypopharyngoscopy shows the mouth of the pouch.

Treatment:
- Small pouch: Repeated endoscopic dilatation of cricopharyngeal sphincter or cricopharyngeal myotomy (cutting the circular muscle fibres).
- Large pouch: Endoscopic Laser division of the septum between the pouch and pharynx or external excision: Diverticulectomy with cricopharyngeal myotomy.

![Fig. (12) Pharyngeal pouch](image)

Malignant Tumours of the Hypopharynx

Surgical anatomy:
- The hypopharynx (laryngopharynx) lies below and behind the base of the tongue; and behind and on each side of the larynx.
- It extends from the level of the hyoid bone to the lower border of the cricoid cartilage.
- The hypopharynx is divided into 3 regions:
  1. Post cricoid area.
  2. Pyriform fossae.
  3. Posterior pharyngeal wall.

Types:
- Benign:
  - Very rare. The most common: fibroma, leiomyoma and adenoma.
- Malignant:
  - The commonest is squamous cell carcinoma.

Site: Post – cricoid carcinoma (PCC) is the most common in Egypt.
Sex: Males are more affected than females.
Age: Over 40 years. However, PCC may affect younger patients in late twenties and thirties.
Predisposing factors: Excessive tobacco smoking, alcohol and Plummer-Vinson syndrome in PCC.

Prognosis: is poor because of the following:
- Late diagnosis.
- Aggressive nature of the tumour.
- Rich lymphatic network which enhances rapid regional metastasis.

The Oesophagus

Anatomy:
- The oesophagus is a fibromuscular tubular structure extending from the 6th cervical vertebra where the pharynx ends in the neck down to the superior mediastinum and then to the posterior mediastinum and then piercing the diaphragm at the level of the 10th thoracic vertebra finally ending in the stomach at the level of the 11th thoracic vertebra. Length of the oesophagus is 25 cm.

Anatomical constrictions of the oesophagus
1. At the inlet of the oesophagus (the strong circular muscular fibers which act physiologically with cricopharyngeus as one unite) the most narrow part in the alimentary tract.
2. The arch of the aorta crossing and compressing the oesophagus in the superior mediastinum.
3. The left main bronchus crossing and compressing it in the superior mediastinum.
4. At the level of the diaphragm.

Diseases of the oesophagus

Congenital Anomalies
- Congenital atresia with or without tracheo-oesophageal fistula:
  - It is an emergency.
  - Suspected when regurge of every feeding after birth.
  - Chocking, cough, aspiration pneumonia in some cases.

Traumatic Lesions

Foreign Body Impaction
- The variety of foreign bodies in the oesophagus are coins, bones, safety pins, dentures and lumps of meat are by far the most common to be encountered. It is commonly seen in children, in mentally retarded and in elderly over 50 years of age who get obstruction with meat or bones impacting due to lack of teeth.
• The sites of the F.B. are at the normal anatomical constrictions, post-corrosive stricture, tumour, achalasia. The narrowest part of the oesophagus is the upper oesophageal sphincter (cricopharyngeus muscle) and two-thirds of the F.Bs. are found at this level.

Diagnosis:
Clinical picture:
History of swallowing F.B.
• Dysphagia and retrosternal dull pain of sudden onset. Discomfort and excessive salivation. Associated coughing or choking if the foreign body is impacted at or just below the laryngeal inlet. The patient is often able to point at the exact site particularly if it is lodged in the upper part of the oesophagus.

Investigation:
• Plain x-ray of the neck and chest may demonstrate the F.B. particularly if it is radio-opaque.
• Oesophagoscopy for diagnosis.

Treatment:
• Removal of the F.B. as soon as possible through rigid oesophagoscopy under general anaesthesia.

**Corrosive Oesophagitis**
• Swallowing of corrosives: the commonest is caustic potash and less common strong acids. This swallowing may be accidental: as in children or suicidal: as in adults.

Clinical Stages are: acute, quiescent and chronic stages.
1. Acute Stage:
   Symptoms:
   ß Severe pain and burning sensation in the mouth and pharynx, painful dysphagia, salivation and regurgitation, stridor and hoarseness may occur due to laryngeal oedema. Shock and dehydration.

   Signs: Burns and ulcers of the mouth tongue and pharynx.

   Complications:
   • Laryngeal oedema, dehydration, electrolyte imbalance and bronchopneumonia.

   Treatment:
   • Antidote: Vinegar for alkalis and aluminium hydroxide gel for acids.
   • Emollients: milk, egg white or olive oil.
   • Steroids to decrease oedema and subsequent fibrosis and stricture.
   • Anti-shock measures and correction of electrolyte imbalance.
   • Systemic Antibiotics.
   • Passage of a nasogastric tube for feeding and to prevent stricture.
   • Tracheostomy in respiratory obstruction.

2. Quiescent Stage: (2-3 weeks)
   • Symptomless interval.

3. Chronic Stage (Post Corrosive Oesophageal Stricture)
   Symptoms:
• Progressive Dysphagia: first to solids then to fluids, regurgitation with recurrent chest infection due to aspiration, dehydration and loss of weight due to oesophageal stricture.

Investigations:
• Barium swallow: To assess the degree, site and length of stricture.
• Oesophagoscopy for diagnosis.

Treatment:
• Permeable stricture:
  Ø Repeated gradual and careful dilatation through rigid oesophagoscopy using dilators and take care to avoid oesophageal perforation by dilator.
• Impermeable stricture:
  Ø Temporary gastrostomy to maintain nourishment.
  Ø By-pass the oesophageal obstruction by colon segment, jejunal loop or gastric pullup.

Achalasia of the Cardia
• Dysphagia with marked dilatation of the lower 2/3 of the oesophagus and more commonly in middle-aged neurotic females.

Aetiology: Unknown but may be neuromuscular incoordination due to degeneration of Auerbach's plexus, leading to failure of relaxation (achalasia) or spasm of the cardiac sphincter.

Clinical picture:
Symptoms:
• Dysphagia: long standing, intermittent and more to fluids than solids. Regurgitation of undigested food. No loss of weight despite the long standing dysphagia; due to intermittent course.

Complications:
• Aspiration pneumonia and malignant change at lower end of oesophagus very rare.

Investigations:
1. Barium swallow: Marked dilatation of the lower 2/3 of the oesophagus with smooth tapering lower end (parrot-peak appearance).
2. Oesophagoscopy: To confirm diagnosis and to exclude coexisting carcinoma.

Treatment:
• Medical:
  Ø Antispasmodics e.g. amyl nitrite inhalation before meals.
• Surgical:
  Ø Repeated dilatation of the gastro-oesophageal sphincter.
  Ø Heller's cardiomyotomy: Cutting the circular muscle fibres of the cardia down to the mucosa.
  Ø Cardioplasty: A longitudinal incision which is then sutured transversely.
  Ø Oesophago-gastrostomy: in advanced kinked cases.

Gastro-Oesophageal Reflux Disease (GERD)
• Retrograde flow of gastric juice into the oesophagus, pharynx and larynx, may be due to decreased lower oesophageal segment pressure or sliding hiatus hernia.

GERD causes: Oesophagitis with ulceration and stricture and chronic pharyngitis and laryngitis.

Clinical picture:
Symptoms:
- Heartburn, but may be absent, sore throat, sensation of lump in the throat and choking, hoarseness or chronic irritative cough.

Signs:
- Congestion of the interarytenoid region and posterior parts of vocal cords.
- Oesophagoscopy: may be oesophageal ulcers and stricture.

Diagnosis: 24-hours oesophageal pH monitoring.

Treatment:
- Modification of life style: Weight reduction; avoid smoking, caffeine and fatty meals, and eating 2 hours before bed time.
- H₂ blockers to decrease gastric acid production.

Carcinoma of the Oesophagus
• Carcinoma of the oesophagus is usually squamous cell carcinoma especially in the upper and middle third. Incidence of adenocarcinoma increases in the lower third.

Clinical manifestations:
• The patient is usually an elderly male complaining of rapidly progressive dysphagia at first for solids more than fluids and then absolute with progressive loss of weight.
• Haematemesis in some cases.
• Retrosternal pain.

Investigations:
• Barium swallow is diagnostic by showing the lesion as an irregular filling defect and/or shouldering (rat tail appearance).
• Oesophagoscopy and biopsy are essential.
• CT neck and chest.

Treatment:
• Surgical resection followed by reconstruction, LN dissection and radiotherapy.
• Prognosis is very bad.
Dysphagia

• Dysphagia is defined as difficulty and/or painful swallowing.

Causes:

• As swallowing is divided into oral, pharyngeal and oesophageal phases, so the causes may be:
  
  • Oropharyngeal:
    - Congenital: Short or cleft palate.
    - Trauma: e.g. corrosives.
    - Inflammations: e.g. as aphthous ulcers, glossitis, stomatitis and pharyngotonsillitis.
    - Neurological diseases: Cerebrovascular accidents, cerebral palsy, multiple sclerosis, cricopharyngeal achalasia and pharyngeal pouch.
    - Neoplasms: carcinoma of the tongue, nasopharynx and oropharynx.
  
  • Oesophageal:
    - Intra-mural: F.B.
    - Mural:
      - Congenital: e.g. stricture and tracheo-oesophageal fistula.
      - Trauma: following endoscopy and in cases of F.B.
      - Inflammations: e.g. reflux oesophagitis and fungal infection.
      - Tumours: e.g. leiomyoma and carcinoma.
      - Neuromuscular: achalasia, hiatus hernia, scleroderma, myasthenia gravis and Parkinson’s disease.
    - Extra-mural:
      - In the neck as malignant thyroid, pharyngeal pouch or mediastinal masses as lymphadenopathy, carcinoma, aortic aneurysm and dilated left atrium.
Anatomy of the Larynx

• The larynx projects forwards in the median part of the front of the neck. It extends from the root of the tongue to the trachea, lying opposite the third, fourth, fifth and sixth cervical vertebrae. Its upper end opens into the laryngopharynx by the laryngeal inlet, while its lower part is continuous with the trachea at the level of the sixth cervical vertebra Fig. (1).
• It consists of a framework of cartilages, connected by ligaments and membranes, lined by mucous membrane and moved by muscles.

Fig. (1): Position of the larynx

Laryngeal cartilages: Fig. (2)

• Single cartilages:
  ▶️ Thyroid cartilage: It consists of two quadrangular laminae that meet anteriorly in the midline forming the thyroid angle. The posterior border of each lamina is elongated to form the superior and inferior cornu.
  ▶️ Cricoid cartilage: It resembles a signet ring. It consists of a quadrangular broad posterior lamina and a narrow anterior arch.
  ▶️ Epiglottis: It is a thin leaf-like cartilage, with an upper free broad part that projects upwards behind the tongue and hyoid bone. Its narrow lower part is attached to the middle of the inner surface of thyroid angle. It has two surfaces: anterior and posterior.

• Paired cartilages:
  ▶️ Arytenoid cartilages: Each resembles a 3-sided pyramid. Its base articulates with the cricoid lamina. It has posterior, medial and anterolateral surfaces. It has an anterior process (vocal) and lateral process (muscular).
  ▶️ Corniculate cartilages: Small cartilages lying on the arytenoid cartilages.
  ▶️ Cuneiform cartilages: Very small cartilages lying in the aryepiglottic folds.
Joints:
- Cricothyroid joints.
- Cricoarytenoid joints.

The folds of the larynx from inside: Fig. (3) and (4)
- Vocal folds (true vocal cords): Ligamentous folds between the vocal processes of arytenoids and the middle of the inner surface of thyroid angle. The two vocal folds meet anteriorly at the anterior commissure.
- Ventricular folds (false vocal cords): Mucosal folds above the vocal folds.
- Aryepiglottic folds: between the arytenoids and lateral borders of epiglottis.
- The ventricle: a cavity between the false vocal cord and the true vocal cord.

Laryngeal mucosa:
- The larynx is lined by respiratory epithelium (pseudostratified ciliated columnar epithelium), except the vocal folds which are lined by non-keratinized stratified squamous epithelium.
Laryngeal muscles and its action: Fig. (5) and (6)

- Intrinsic muscles:
  - Abductors: open the glottis during inspiration (posterior cricoarytenoid muscle).
  - Adductors: close the glottis during phonation, swallowing and chest fixation. These muscles are: Lateral cricoarytenoid, transverse interarytenoid and thyroarytenoid muscles.
  - Tensors: increase vocal fold tension (cricothyroid and vocalis muscles).
  - Opening of laryngeal inlet: Thyroepiglottic muscle.
  - Closure of laryngeal inlet: Oblique interarytenoid and aryepiglottic muscles.

- Extrinsic muscles:
  - Depressors of the larynx: Infrahypoid group of muscles.
  - Elevators of the larynx: Suprahypoid group of muscles.
Nerve supply: Branches of the vagus nerve: Fig. (7)
- Superior laryngeal nerve: Motor to the cricothyroid muscle and sensory to the mucosa above the vocal cords.
- Recurrent laryngeal nerve:
  1. Motor to all muscles of the larynx except the cricothyroid muscle and sensory to the mucosa below the vocal cords.
  2. The left recurrent nerve turns around the arch of aorta in the chest, while the right one turns around the subclavian artery in the neck.

Regions of the larynx: fig. (3)
1. Supraglottis: from the tip of the epiglottis to the floor of the ventricle.
2. Glottis: Area bounded by both vocal cords from side to side and anterior and posterior commissures anteroposteriorly.
3. Subglottis: 10 mm below the free margin of vocal folds to the inferior edge of the cricoid cartilage.
4. Blood supply:
   1. Superior laryngeal artery: from the superior thyroid artery.
   2. Inferior laryngeal artery: from the inferior thyroid artery.

Lymph drainage:
- Supraglottic region: Upper deep cervical and pre-epiglottic lymph nodes.
- Glottic region: has minimal or no lymph drainage.
- Subglottic region: Lower deep cervical, prelaryngeal and paratracheal lymph nodes.
Functions of the Larynx

• The principal function of the larynx is not speech as is commonly believed, but protection of the airways.
1. Protection of the lower air way: against aspiration during swallowing. This is achieved by: closure of the laryngeal inlet and glottis, cessation of respiration, elevation of the larynx under the tongue, epiglottis closes laryngeal inlet and reflex coughing and closure if foreign body enters the larynx.
2. Phonation: Voice production by vibration of the vocal cords (vocal folds).
3. Respiratory: The larynx serves as an air channel and control of the width of the glottis regulates gase exchange and carbon dioxide level in the blood.
4. Circulatory: The alternating positive and negative intra-thoracic pressure help the blood circulation.

Examination of the Larynx

• Inspection: for position i.e. shifted to one side, movement with deglutition, dilated veins or skin scar.
• Palpation: for tenderness and laryngeal click which is normally elicited due to moving of the cricoid cartilage on the vertebral column from side to side (Moure’s sign).
• Palpation of the neck: Cervical lymph nodes, thyroid gland and trachea.
• Indirect laryngoscopy: Fig. (8) This examination is conducted by a warm laryngeal mirror.
• Direct laryngoscopy (D.L.):
  ❍ Rigid telescope (70º or 90º) Fig. (9) or flexible fiberoptic nasopharyngoscope Fig.(10), under local anaesthesia for difficult cases i.e. overhanging epiglottis or inability of the patient to tolerate indirect laryngoscopy
  ❍ Rigid Laryngoscopy: Fig. (11) Under general anaesthesia for examination by microscope for magnification
Fig. (8): Indirect laryngoscopy by laryngeal mirror.

Fig. (9): Rigid telescope of larynx

Fig. (10): Fiber-optic nasolaryngoscopy.  

Fig. (11): Rigid Laryngoscopy
Symptomatology

**Stridor**
Definition: Difficult noisy breathing due to partial laryngeal obstruction.

Types of stridor:
1. Inspiratory: due to laryngeal obstruction.
2. Expiratory: due to lower respiratory obstruction e.g. asthma.
3. Bi-phasic stridor (during inspiration and expiration) due to subglottic or tracheal obstruction.

Causes of stridor (laryngeal obstruction) leading to inspiratory stridor:
- **Congenital:**
  - Congenital laryngomalacia, laryngeal web, subglottic stenosis and bilateral vocal cord paralysis.
- **Traumatic:**
  - Inhaled foreign body.
  - Accidental trauma:
    - External trauma e.g. cut throat or strangulation
    - Internal trauma:
      - Chemical e.g. corrosives.
      - Physical e.g. hot water or burns.
  - Surgical trauma (Iatrogenic):
- **Inflammatory:**
  - Acute non specific: in children.
  - Acute specific: diphtheria.
  - Chronic specific: scleroma, syphilis, T.B.
- **Allergic oedema:** e.g. angioneurotic oedema of larynx or drug sensitivity.
- **Neoplastic:**
  - Benign: multiple papillomata in children.
  - Malignant: carcinoma.
- **Neurogenic:**
  - Bilateral abductor paralysis: most commonly after thyroidectomy.
- **Functional:** Laryngismus stridulus.

**Hoarseness of voice**
Definition: Change of voice, so it becomes rough low-pitched voice, due to incomplete coaptation, tension or vibration of the vocal cords during phonation.

Aetiology:
- **Congenital:** Congenital laryngeal web and unilateral vocal cord paralysis.
- **Traumatic:** External trauma, internal burns, iatrogenic trauma, inhaled foreign bodies and voice trauma.
- **Inflammatory:** Acute laryngitis, chronic non-specific laryngitis, vocal nodules, polyp and leukoplakia, chronic specific laryngitis except laryngoscleroma.
- **Laryngeal oedema:** Angioneurotic oedema.
- Tumours: Benign and malignant tumours.
- Pseudotumours: Cysts and laryngocele.
- Neurological: Unilateral vocal cord paralysis.
- Cricoarytenoid joint: arthritis and fixation.
- Myasthenia gravis.
- Functional: Hysterical aphonia.

N.B.: Persistent hoarseness for more than two weeks in middle or old age patient should be investigated to exclude cancer larynx.

**Cough:**
- This is a common complaint in laryngeal diseases. Cough may be dry or productive. In cases of foreign bodies, cough is a protective reflex and paroxysmal.
- Expectoration:
  - Expectoration may be mucous (catarrhal laryngitis), mucopurulent, purulent (laryngeal abscess) or it may be blood stained (in malignancies).

**Pain:**
- Local pain: felt as a sense of soreness or actual pain of larynx.
- Referred pain: to the ear via the auricular branch of the vagus.

**Dysphagia:**
- Dysphagia is present in laryngeal disease when there is affection of the arytenoid region and the outer aspect of the ary-epiglottic fold.
- Dysphagia may also be due to extention of laryngeal tumour to the hypopharynx.
Diseases of the Larynx

Congenital Anomalies

**Laryngomalacia** Fig. (12)
- The commonest cause of congenital stridor.

**Aetiology:**
- Exaggerated infantile larynx.
- Abnormal softening and flabbiness of supraglottic laryngeal tissues, which collapse during inspiration.
- Laryngeal arrhythmia due to incoordination of respiration.

**Clinical picture**

**Symptoms:**
- It starts shortly after birth with inspiratory stridor.
- Stridor increases on agitation and decreases in the prone position or when the neck is hyperextended. Severe respiratory distress is very uncommon.
- Feeding is usually normal and the child is of good health, with no fever neither nor hoarseness of voice (normal cry).

**Signs:** by direct laryngoscopy
- Exaggerated infantile larynx: Epiglottis is long, narrow and folded backwards (omega shaped). Aryepiglottic folds are short and approximated. Laryngeal inlet is reduced to a cruciate slit.
- Edges of laryngeal inlet are sucked in with each inspiration.

**Treatment:**
- Only reassurance as stridor disappears by the age of two years (no treatment).
- Surgical treatment is needed (rarely) in severe stridor: wedge resection of the aryepiglottic folds by laser. Tracheostomy as an emergency.
**Congenital Laryngeal Web**
- A fibrous web between the anterior part of the vocal cords due to arrest of canalization of the larynx.

Clinical picture
Symptoms:
- In mild cases: no symptoms.
- In moderate cases: there is a weak hoarse cry, dyspnea and inspiratory stridor.
- In severe cases: immediate death after birth and diagnosed at autopsy.

Signs: By direct laryngoscopy, the web is seen extending between the vocal cords. It is white or pink in colour with sharp curved posterior border.

Treatment:
- In mild cases: No treatment.
- In severe cases: Surgical or laser excision of the web.
- Tracheostomy: Indicated only if stridor is severe.

**Congenital Subglottic Stenosis**
Aetiology: Incomplete canalization of the subglottic space.
Symptoms: Bi-phasic stridor (during inspiration and expiration) without hoarseness of voice. It is unaffected by posture.
Signs: Narrowing of subglottic region.
Differential Diagnosis: Acquired subglottic stenosis.

Treatment:
- Mild stridor: No treatment.
- Severe stridor: - Tracheostomy and microlaryngoscopic laser vaporization or laryngoplasty.

**Trauma of the Larynx**

External trauma:
- Closed injuries: Motor car accidents, blows and strangulation.
- Open injuries: Stabs, cut throat and gunshots.

Internal trauma:
- Inhalation: of steam, irritant fumes or gases.
- Swallowing of corrosives.
- Surgical:
  - Rough laryngoscopy.
  - High tracheostomy.
- Intubation during anaesthesia: Due to rough intubation, prolonged intubation or large tube.
- Radiotherapy of head and neck: laryngeal oedema and perichondritis.

Inhaled foreign bodies:

Voice trauma:
- Leads to: - submucosal haemorrhages on vocal cords or vocal cord nodules (Singer's nodule).
**Foreign Body Inhalation**

**Aetiology:**

**Types of patients and F.B.**
- Usually a child: F.B. inhalation usually occurs during eating while the child is laughing or frightened. The commonest foreign bodies are watermelon seeds, peanuts and fish bones.
- Sometimes the patient is a young female inhaling F.B. as pins.
- Mentally-ill adults.

**Fate of F.B. inhalation:**
- F.B. is coughed outside.
- F.B. becomes impacted in the larynx (Laryngeal F.B.) which is rare.
- F.B. becomes inhaled inside the tracheobronchial tree (bronchial F.B.) it usually enters the right bronchus because it is wider than the left and more in line with the trachea.
- Vegetable F.B. causes diffuse violent irritation of the mucosa.
- Non-vegetable F.B. causes no symptoms unless it is obstructing a bronchus partially or completely.

**Clinical picture:**
- **Laryngeal F.B.**:
  - Laryngeal spasm, choking, cough, stridor and cyanosis. Large F.B. may cause complete obstruction of the glottis and death.
- **Bronchial F.B.**:
  - Initial stage: As the foreign body passes through the larynx, it causes a violent attack of cough, choking and dyspnea.
  - Latent stage: A symptomless interval, which is shorter with vegetable F.B.
  - Manifest stage: Fig. (13)
    - Partial obstruction leads to emphysema of the lung as the F.B. allows air in but not out, mediastinum becomes shifted to the other side.
    - Complete obstruction causes collapse of the lung (atelectasis) with mediastinal shift to the same side.

Fig. (13): F.B. in bronchus
Diagnosis:
- History: Typical history of sudden choking while eating is an essential step in diagnosis.
- Clinical picture: of unresolved or repeated chest infection of children inspite of good medical treatment.
- Chest X-ray: to show radio-opaque F.B. (very rare) or complications as atelectasis or emphysema or lung abscess if the F.B. is not radio-opaque.

Treatment:
1. Laryngeal F.B.:
   a. First aid measures:
      a. Back blows by slapping the back of the child sharply with the hand with the head down.
      b. The Heimlich maneuver: 1- Standing behind patient and apply sudden pressure in the epigastrium with clinched fists. 2- Kneel on the side of the supine patient and apply epigastric pressure with the palm of the hand.
   b. Laryngoscopic removal of F.B. under general anaesthesia.
   c. Rarely laryngofissure in large impacted F.B.

Inflammation of the Larynx

Laryngitis

Acute Non-specific Laryngitis

Acute non-specific laryngitis in adults:

Aetiology:
- Usually viral infection followed by secondary bacterial infection. Predisposing factors: Rhinitis, sinusitis, pharyngitis, smoking.

Clinical picture:

Symptoms:
- Hoarseness of voice is the main symptom.
- Dry cough and feeling of discomfort in the throat especially on talking.

Signs:
- Congestion and oedema of the laryngeal mucosa mainly the vocal folds.

Treatment:
- Local treatment:
  - Voice rest, avoid laryngeal irritants as smoking and steam inhalation as Tincture Benzoin co.
- General treatment:
  - Antibiotics, cough suppressants.
Acute non-specific laryngitis in children:
Aetiology:
• Usually viral infection followed by secondary bacterial infection.
Clinical picture:
Symptoms:
• Acute laryngitis in children is a serious condition, because:
  β Stridor is the main symptom due to:
    – Laryngeal submucosa is loose, so oedema easily forms.
    – Laryngeal cartilages are soft, so collapse easily.
    – Larynx is relatively small, so obstruction occurs easily.
    – Laryngeal reflexes are immature, so spasm occurs easily.
  β Dry cough may be accompanied with laryngeal spasm.
Signs: difficult examination
• Congestion and oedema of the laryngeal mucosa, mainly in the subglottic region.
Treatment: Hospitalization and close observation are mandatory.
• General treatment:
  β Antibiotics, corticosteroids, cough suppressants and mucolytics.
  β Rest in bed in the sitting or semisitting position.
  β Room humidification by an atomizer or steam.
• Local treatment:
  β Oxygen inhalation.
  β Tracheostomy or intubation in severe laryngeal obstruction.

Acute epiglottitis (supra-glottitis):
Aetiology:
• The most common pathogen is Haemophilus influenza type B.
Clinical picture:
Age: infants and children are more common than adults.
Symptoms:
• Rapidly progressive fever, anorexia and malaise.
• Inspiratory stridor, rapidly progressive, and potentially fatal.
• Painful swallowing with drooling and hot potato muffled voice.
Signs:
• Examination by tongue depressor may induce laryngeal spasm, which may be fatal
• Lateral view X-ray neck shows marked thickened epiglottis.
Treatment:
• It is an emergency condition as it is life threatening.
  β Great care of the airway is mandatory. So endotracheal intubation is usually needed and rarely tracheostomy.
  β Medical treatment as acute non specific laryngitis in children
Acute laryngo-tracheo-bronchitis:
- It is more common in infants, especially below 2 years of age.

Aetiology:
- Viral infection of the mucosal lining of the whole respiratory tract. Usually starts as simple acute rhino-pharyngitis.

Clinical picture:
Symptoms:
- Progressive biphasic stridor and respiratory distress.
- Fever, anorexia and malaise. Croupy cough, with expectoration of very thick tenacious sputum.

Signs: difficult examination.
- Hyperemia and oedema of the mucosa of the subglottic region, trachea and bronchi. The mucosa is covered with very viscid muco-purulent exudate.

Treatment:
- Similar to acute non specific laryngitis in children.
- Tracheostomy may be needed: to bypass obstruction and aspirate thick secretions.

Acute Specific Laryngitis:
Diphtheritic laryngitis:
- It is very rare nowadays because of the mandatory DPT vaccination.

Aetiology: secondary to faucial diphtheria (caused by corynbacterium diphtheria)

Clinical picture:
Symptoms:
- Manifestations of diphtheritic toxemia (low grade fever, malaise, disproportionate tachycardia).
- Stridor, due to laryngeal obstruction by the diphtheritic membrane.
- Hoarseness of voice and cough.

Signs:
- The laryngeal mucosa is covered with a grayish yellow pseudomembrane.

Treatment: As faucial diphtheria.
- Rest, isolation, antitoxic serum and penicillin.
- Tracheotomy, if needed.

Chronic laryngitis

Chronic Non specific Laryngitis
I. Chronic Diffuse Hypertrophic Laryngitis.
Aetiology:
- Repeated attacks of acute laryngitis, repeated attacks of upper respiratory infections as sinusitis and pharyngitis, voice abuse, prolonged exposure to laryngeal irritants as tobacco and dust and gastro-oesophageal reflux.
Clinical picture:
Symptoms:
• Hoarseness of voice with dry irritating cough with frequent hawking, hemming and sore throat.
Signs:
• Bilateral symmetrical thickening of both vocal folds, which appear whitish, reddish or pale and oedematous.
Treatment:
• Speech therapy.
• Avoid and /or treat the aetiologies.
• Micro-laryngeal stripping of the mucosa of the vocal cords is rarely needed.

II. Chronic Localized Hypertrophic Laryngitis.
1. Vocal nodules (Singer's):
Aetiology:
• Prolonged abuse of voice in untrained voice users as singers and teachers (professional voice users).
Pathology:
• Localized epithelial hyperplasia and/or sub-epithelial organized hematoma of the vocal fold.
Clinical picture:
Symptoms: Hoarseness and voice fatigue.
Signs:
• Bilateral small sessile smooth nodules at the junction of the anterior and middle thirds of the vocal folds because it is the site of maximum contact of the vocal folds during phonation.
Treatment:
• Speech therapy may lead to regression and help to avoid recurrence.
• Micro-laryngeal excision by surgical instruments may be needed and followed by speech therapy to avoid recurrence.

2. Vocal cord polyps: Fig. (15)
Aetiology: usually due to an acute violent voice trauma e.g. shouting.
Pathology: Localized sub-epithelial oedema (oedematous polyp), vascular engorgement (vascular polyp) or fibrosis (fibrotic polyp) of the vocal fold.
Symptoms: Hoarseness of voice
Signs:
  • Unilateral single, variable-sized, sessile or pedunculated, smooth swelling of anterior commissure or the vocal fold.
Treatment:
  • Micro-laryngeal excision by surgery or laser.
  • Speech therapy, to avoid recurrence.

3. Leukoplakia:
Definition:
  • Circumscribed white raised areas of the mucous membrane of the vocal fold (pre-cancerous lesion).
Aetiology:
  • Unknown and predisposing factors may be chronic irritation by excessive smoking, alcohol or gastric reflux.
Pathology:
  • Localized epithelial hyperplasia and hyperkeratosis of the vocal fold. The basement membrane of the epithelium remains intact.
Clinical picture:
Symptoms: Hoarseness of voice with dry irritative cough with hemming.
Signs:
  • Irregular white raised patch or patches on the vocal cords.
Treatment:
  • Avoid predisposing factors.
  • Excision by micro-laryngeal surgery or laser with proper histopathological examination, to exclude malignant changes.
  • Regular follow up, as it is a pre-cancerous lesion.

III. Chronic Atrophic Laryngitis
Aetiology: Associated with atrophic rhinitis.
Symptoms: Hoarseness of voice, irritation in the throat, foul odour and dyspnea.
Signs: Laryngeal mucosa is pale, glazed, dry and covered with crusts.
Treatment:
  • Mucolytics, potassium iodide, menthol inhalation and laryngeal spray with sodium bicarbonate solution.
  • Repeated endoscopic removal of crusts.

Chronic Specific laryngitis

Laryngoscleroma: Fig. (16)
  • It is the commonest laryngeal granuloma in Egypt, its site is in subglottic region.
Aetiology:
  • Chronic specific granuloma caused by Frisch bacillus, most commonly secondary to rhinoscleroma.
Clinical picture:
Symptoms:
• Stridor (which is bi-phasic as it is a subglottic lesion) and dyspnea are the main symptoms.
• Cough with expectoration of greenish crusts.
Signs: Fig. (16)
• Symmetrical masses or webs covered with greenish crusts in the subglottic region.
• Healing may occur by dense subglottic fibrous tissue leading to subglottic stenosis.

Fig. (16): Laryngo-scleroma in subglottis.

Treatment:
• Medical treatment: as rhinoscleroma.
• Surgical treatment:
  ß Treatment of subglottic stenosis by micro-laryngeal laser therapy or repeated dilatation.
  ß Tracheostomy in severe stridor.

Tuberculosis of the larynx: Fig. (17)
• Always secondary to pulmonary tuberculosis and its site is the posterior part of larynx.
Clinical picture:
Symptoms:
• T.B. toxemia: night fever, night sweating and loss of weight.
• Hoarseness of voice: the voice is weak and soft.
• Pain in the throat on speech and swallowing, may be referred to the ear.
• Cough and hemoptysis.
• Stridor and dyspnea (late).
Signs: Fig. (17)
• Pale granulations, ulcers, perichondritis and fibrosis. It affects mainly the posterior part of the larynx in the inter-arytenoid region and posterior parts of the vocal folds.
Complications:
• Perichondritis.
• Healing by dense fibrous tissue leading to laryngeal stenosis.
Investigations:
• X-ray chest.
• Sputum examination for TB bacillus.
• Tuberculin test.
• Direct laryngoscopy and biopsy.
Fig. (17): Tuberculous laryngitis.  

Fig. (18): Syphilitic laryngitis.

Treatment:
• Medical treatment: Anti-tuberculous therapy.
• Surgical treatment:
  § Tracheostomy when necessary.
  § Treatment of laryngeal stenosis

**Syphilis of the larynx:** Fig. (18)
In the anterior part of the larynx and very rare or even absent nowadays.

**Cysts of the Larynx**

Types:
• Congenital cysts: on aryepiglottic folds and ventricular bands.
• Mucous retention cysts: on aryepiglottic folds, ventricular bands and epiglottis.

Symptoms: Hoarseness of voice and stridor.
Treatment: Microlaryngoscopic excision or marsupialization of the cyst.

**Laryngocoele** Fig. (19)
• Pneumatic cystic expansion of the laryngeal ventricle and saccule, so it contains air usually in adult males.

Aetiology:
• Expiration against resistance as in glass blowers and trumpeters.
• Carcinoma of laryngeal ventricle acting as one way valve; so the air goes into the ventricle but not coming out.

Fig. (19): Laryngoceles
Types of laryngoceles: Fig. (19)
- Internal (20%): within the larynx. It passes behind the thyrohyoid membrane and bulges into the vallecula.
- External (30%): It passes through the thyrohyoid membrane to outside into the neck.
- Mixed internal and external (50%).

Symptoms:
- External laryngocele: Neck swelling.
- Internal laryngocele: Hoarseness of voice, stridor and dysphagia.
- Mixed laryngocele: Symptoms of both internal and external.

Signs:
- External laryngocele: Cystic neck swelling in thyrohyoid region, which expands on straining by Valsalva's manoeuvre and empties on digital pressure.
- Internal laryngocele: Submucosal swelling of the ventricle, false cord and vallecula.

Investigations:
- Plain X-ray neck: sac filled with air.
- Direct laryngoscopy: to exclude underlying hidden carcinoma of the ventricle.

Treatment:
- External laryngocele: Excision through external cervical approach.
- Internal laryngocele: Microlaryngoscopic marsupialization or excision.

**Vocal Cord Paralysis**

Aetiology:
A. Congenital:
- Birth trauma, meningocele and congenital mediastinal masses.

B. Acquired:
- Idiopathic (25%): may be viral neuritis.
- Supra-nuclear lesions: Rare bilateral extensive cortical lesions.
- Nuclear (Bulbar) lesions:
  - Brain stem tumours.
  - Brain stem vascular accidents hemorrhage or thrombosis.
  - Encephalitis, poliomyelitis and diphtheria.
- Vagus nerve.
  - Skull base at jugular foramen:
    - Fracture base.
    - Basal meningitis.
    - Nasopharyngeal carcinoma and glomus jugular tumour.
- Recurrent laryngeal nerve lesions:
  - In the neck:
    - Thyroidectomy and radical neck dissection.
    - Malignant tumours of thyroid, lymph nodes, hypopharynx, and oesophagus.
• In the thorax: (left recurrent laryngeal nerve).
   Surgery of lung, heart and oesophagus.
   Malignant tumours of mediastinal lymph nodes, oesophagus and bronchi.
   Aortic aneurysm and dilated left atrium.

VI. Peripheral neuritis: Influenza, diphtheria and diabetes.

N.B. Left vocal cord paralysis is more common than right vocal cord paralysis because the left recurrent laryngeal nerve has longer course to the chest.
• Unilateral paralysis is more common than bilateral paralysis.
• Abductor paralysis is more common than adductor paralysis.

Positions of the vocal cord: Fig. (20)
• Median: in the midline (during phonation).
• Paramedian: 1.5 mm from the midline.
• Intermediate (cadaveric) position: 3.5 mm from the midline.
• Gentle abduction: 14 mm glottic chink (during quiet respiration).
• Full abduction: 18 mm glottic chink (during deep inspiration).

Types of paralysis:
- Abductor paralysis:
  • Due to recurrent laryngeal nerve lesions leading to vocal cord paralysis in median or paramedian positions.
- Adductor paralysis:
  • Due to vagus nerve lesions (combined superior and recurrent laryngeal) leading to vocal cord paralysis in intermediate (cadaveric) position.

Differential Diagnosis:
• Arytenoid fixation due to cricoarytenoid joint arthritis as with rheumatoid arthritis.

Investigations:
• Clinical: Full head, neck and chest examination.
• Radiological: (according to the suspected site): X-ray chest, C.T. scan for skull base, thorax, thyroid scan and barium swallow.
• Panendoscopy includes: laryngoscopy, nasopharyngoscopy, hypopharyngoscopy, oesophagoscopy and bronchoscopy.
Unilateral Vocal Cord Paralysis
Symptoms:
  a. Unilateral abductor paralysis: Hoarseness of voice.
  b. Unilateral adductor paralysis: Hoarseness of voice and aspiration (choking).

• Symptoms usually improve within 6 months due to compensation of the other mobile vocal cord, which crosses the midline during phonation to meet the paralysed cord.

Signs:
• Vocal cord is paralysed in median or paramedian position (abductor paralysis) or in intermediate position (adductor paralysis) with glottic gap.

Treatment:
• Treatment of the cause.
• Speech therapy and observation till compensation.
• Surgical treatment: by medialization of the paralysed vocal cord, if compensation is not satisfactory with persistence of symptoms after 6 months.

Bilateral Vocal Cord Paralysis

Bilateral Abductor Paralysis
Symptoms:
• Stridor and no hoarseness but voice tires easily.

Signs:
• Both vocal cords are paralysed in the median or paramedian position with narrow glottic space.

Treatment:
• Treatment of the cause.
• Surgical widening of glottis: improves the airway on the expense of voice.
• Permanent tracheostomy with speaking valve.

Bilateral adductor paralysis
Symptoms:
• Aphonia, aspiration with repeated coughing due to incompetent larynx and insensitive supraglottis. Aspiration results in chest infection, which may be fatal.

Signs:
• Both vocal cords are paralysed in intermediate cadaveric position.

Treatment of aspiration by:
• Treatment of the cause.
• Tracheostomy with cuffed tube.
• Laryngeal closure by surgery with tracheostomy.
• Total laryngectomy in case of failure of the above treatments.
Functional Disorders

Hysterical Aphonia (Functional bilateral adductor paralysis)
Aetiology: Emotional disturbance, commonly in young adult females.
Symptoms:
• Sudden aphonia, with whisper voice, normal coughing and no aspiration.
Signs:
• Incomplete adduction of the vocal cords on phonation.
Treatment:
• Psychotherapy and voice therapy.

Laryngismus stridulus.
Aetiology:
• It is due to active spasm of the glottis due to calcium deficiency, or gastroesophageal reflux.
Clinical picture:
• Sudden attacks of stridor in children at night, which may be associated with carpopedal spasm. After a while, the child takes a deep breath followed by deep sleeping. The attack may be repeated at the same or every night.
Treatment:
• During the attack: By measures helping to stimulate active opening of the cords as: Splashing with cold water and pulling the tongue forwards.
• After the attack: Give vitamins, tonics, treatment of calcium deficiency, anti-reflux measures and removal of the enlarged adenoid.

Tumours of the Larynx

Benign Tumours of the Larynx

Squamous Cell Papilloma
• The commonest benign laryngeal tumour.
• It is single in adults and multiple in children.
Microscopically: Vascular connective tissue core covered by hyperplastic stratified squamous epithelium.

1. Multiple Papillomata Fig. (21)
Age: Children.
Aetiology: Human papilloma virus.
Pathology: Multiple and sessile wart-like small masses.
Site: Anywhere in the larynx and it may extend to trachea.
Symptoms:
• Hoarseness of voice and stridor.
Signs: Multiple, sessile, pinkish, wart-like small masses.
Recurrence: Common with spontaneous regression at puberty and never turn malignant.
Treatment:
- Repeated excision by microlaryngeal surgery or laser, followed by Interferon therapy.
- Tracheostomy: almost indicated, must be low and cork it after each excision to allow normal growth of larynx.

2. Single Papilloma Fig. (22)
Age: Adults.
Sex: Males: Females 2:1
Aetiology: True benign neoplasm.
Pathology: Single, sessile or pedunculated.
Site: Common in the anterior 1/2 of vocal cord or anterior commissure.
Symptoms: Hoarseness of voice and rarely stridor.
Signs: Single, sessile or pedunculated, pinkish, wart-like mass.
Recurrence: Uncommon; if excised completely.
Malignant transformation: May turn malignant as it is a pre-cancerous lesion.
Treatment:
- Excision by microlaryngeal surgery or laser.
- Excision through laryngofissure, if large.

Cancer of the Larynx

Commonest is squamous cell carcinoma.
Incidence:
- Age: commonly over 40 years
- Sex: more in males than females (10:1).
- Predisposing factors: Tobacco, alcohol, radiation, genetic susceptibility.
- Pre-cancerous lesions: leukoplakia, adult papilloma.

Classification of tumours according to region: Fig. (23 and 24)
- The larynx is divided into 3 regions and each region is subdivided into sites as follows:
  - Supraglottic region consists of the following sites:
    - Epiglottis, ventricular folds, aryepiglottic folds, ventricles and arytenoids.
Glottic region consists of the following sites:
- Vocal cords to 10 mm below the free margin.
- Anterior commissure.
- Posterior commissure.

Subglottic region consists of the following sites:
- 10 mm below the free margin of vocal folds to the inferior edge of the cricoid cartilage.

Anatomical incidence: glottic (60%), supraglottic (35%), subglottic (5%)

Fig. (23): Glottic carcinoma of vocal cord

Fig. (24): Regions of laryngeal carcinoma

Pathology:
Gross pathology: ulcerative (commonly glottic and subglottic) or exophytic (supraglottic).
Microscopic appearance: sq. cell carcinoma (90-95%), other epithelial and mesenchymal malignant tumours (5-10%).

Spread:
• Direct to the adjacent region of the larynx and extra laryngeal spread.
• Lymphatic: according to the region:-
  - Supraglottic to upper deep cervical LNs (almost bilateral)
  - Glottic has no lymphatic drainage (early glottic cancer doesn't spread to LNs) except after spread to the adjacent areas.
  - Subglottic to the lower cervical LNs.
• Distant metastasis (rare).
Clinical Picture:

Symptoms:
• Hoarseness: the earliest symptom in glottic carcinoma.
• Discomfort in the throat; the earliest symptom in supraglottic carcinoma.
• Dyspnea and stridor; the earliest symptoms in subglottic carcinoma.
• Local pain due to deep invasion and perichondritis.
• Referred pain to the ear via vagus.
• Dysphagia indicates infiltration of the pharynx.
• Cough and choking; late and rare.
• Late symptoms as neck swelling, hemoptysis, and loss of weight.

Signs:

Neck Examination:
• Neck mass either cervical L.N. or laryngeal infiltration.
• Broadening of the larynx.
• Tenderness of the larynx due to perichondritis.

Larynx Examination: by indirect or direct laryngoscopy.
• Supraglottic tumours: usually fungating and tend to be bilateral.
• Glottic tumours: tend to be unilateral as irregular thickening and roughness of the cord, mass or ulcer of vocal cord and later on as a cauliflower mass.
• Subglottic tumours: commonly bilateral ulcer or fungating mass.

General Examination: looking for distant metastasis.

Prognosis: Glottic carcinoma has better prognosis than other types due to:
• Early diagnosis as it presents early with symptom of hoarseness of voice and late lymphatic spread.

Investigations:

Laryngoscopy by rigid telescope or flexible nasopharyngoscope to:
• Assess the vocal cords mobility as vocal cord fixation indicates infiltration of intrinsic muscles of the larynx, cricoarytenoid joint or recurrent laryngeal nerve.
• Direct laryngoscopy by microscope under general anaesthesia to map the tumour and to take biopsy for histopathological examination.

Radiological:
• C.T. scan and MRI of the neck are the best to evaluate the tumour extension, cartilage invasion, extralaryngeal spread, and lymph node involvement of the neck.
• Plain X ray of chest to exclude metastasis.

Staging of the tumours:

TNM classification: to standardize treatment method and to report the treatment results.

• Primary Tumours (T):
  ⚫ T\textsubscript{x}: Primary tumour can't be assessed.
  ⚫ T\textsubscript{0}: No evidence of primary tumour.
  ⚫ T\textsubscript{IS}: Carcinoma in situ.
T1: Tumour limited to one site with normal vocal cord mobility.
T2: Tumour extending to more than one site with normal vocal cord mobility.
T3: Tumour limited to larynx with vocal cord fixation.
T4: Tumour extending beyond the larynx.

• Lymph Nodes (N):
  N0: No clinically positive nodes.
  N1: Single clinically positive ipsilateral node 3 cm or less in diameter.
  N2: Single clinically positive ipsilateral node more than 3 cm but less than 6 cm in diameter.
  N3: Ipsilateral nodes ≥6 cm in diameter or bilateral or contralateral nodes. Of any size.

• Distant Metastasis (M):
  M0: No evidence of distant metastasis.
  M1: Tumour with distant metastasis.

Treatment of Cancer Larynx
I. Curative:
1. Surgery:
   • Resection of the primary tumour:-
     A. Conservative laryngeal surgery with preservation of laryngeal functions.
        Endoscopic resection for Tis and early T1 lesions.
        Partial laryngectomy for T1 and T2 lesions.
     B. Total laryngectomy for T3 and T4 lesion.
        Radical neck dissection for palpable cervical lymph nodes.
        In total laryngectomy:
          – All larynx is removed.
          – There is a permanent tracheostomy.
          – Patient can not phonate unless voice restoration is done.
   2. Radiotherapy: This is curative in T1 and T2 Tumours with preservation of laryngeal functions.
   3. Combined therapy (surgery + radiotherapy)

Postoperative radiotherapy:
Indicated in case of:-
• No free safety margin after laryngectomy.
• Occult cervical lymph nodes metastasis.
• Preoperative tracheostomy due to high incidence of stomal recurrence.
Voice restoration after total laryngectomy:
• Physiological methods: oesophageal speech.
• Surgical methods: tracheo-oesophageal fistula.
• Artificial methods: electronic larynx.
II. Palliative:
Indications: Inoperable tumours due to:
• Distant metastasis.
• Involvement of unresectable structures e.g. carotid artery.
• Poor general condition.
Palliative treatment:
• Palliative radiotherapy and chemotherapy.
• Tracheostomy: for airway obstruction.
• Ryle tube feeding or gastrostomy: for dysphagia.
• Palliative laser excision of a fungating mass.
• Pain killers.

Operations of the larynx

1. Micro-laryngeal surgery
   • To examine and operate the laryngeal lesions by microscope with
     magnification.
Indications:
• Biopsy from laryngeal tumours.
• Excision of benign tumours, laryngeal polyp, laryngeal cyst and singer’s
   nodules.
• Excision of T1 or T2 cancer larynx.
• Surgical treatment of vocal cord paralysis.
• Excision of laryngeal web.

2. Laryngofissure Fig. (25)
• Operation in which the thyroid cartilage is split in the mid-line like an
  open book.
Indications:
• Excision of benign tumours or early malignant tumours of the larynx.
• Replacement of fractured laryngeal cartilages.
• Correction of laryngeal stenosis after trauma or inflammations.
• Removal of impacted foreign body in the larynx

Fig. (25) Laryngo fissure
3. Tracheostomy
Definition:
• This is a surgical artificial opening in the anterior wall of the cervical trachea to keep patent airway.
Indications:
1. Upper respiratory tract obstruction (mechanical obstruction):
• Laryngeal obstruction: causes of stridor.
• Supralaryngeal obstruction:
  ø Retropharyngeal abscess.
  ø Ludwig's angina.
  ø Tumours of the tongue base.
• Infra laryngeal obstruction:
  ø Malignant thyroid.
  ø Cellulitis in the neck.
2. Lower respiratory tract obstruction (secretory obstruction):
• Any condition causing abolished or weak cough reflex leads to accumulation of secretions inside the alveoli. This prevents gas exchange leading to hypoxia and acidosis. The patient is said to be drowned in his own secretions.

Causes of secretory obstruction:
• Coma: as in
  ø Cerebro-vascular accidents: e.g thrombosis and hemorrhage.
  ø Trauma: head injuries and fracture base.
  ø Toxins:
    – Exogenous e.g barbiturate poisoning.
    – Endogenous e.g uraemia and diabetic coma.
  ø Tumours: of the brain.
• Failure of chest muscles: as in
  ø Paralysis of chest muscles: diphtheria and poliomyelitis.
  ø Trauma: multiple fracture ribs.
  ø Myopathies: Myasthenia gravis.
3. Preoperative indications:-
  ø Before bloody operations in the mouth and pharynx to prevent aspiration of blood. Examples are nasopharyngeal fibroma, glossectomy and maxillectomy
  ø As a step in total laryngectomy and laryngofissure.

Aim of tracheostomy:
1. In mechanical obstruction:
• To by-pass upper respiratory obstruction.
• To overcome tracheal obstruction below the level of tracheostomy by passing a hard tube (Koenig's tube).
2. In secretory obstruction:
• Aspiration of accumulated secretions in the lung.
• To maintain artificial respiration in central respiratory failure.
• To reduce the dead space of upper respiratory air way.
3. In preoperative indications:
   • Tracheostomy is done and pharynx is packed well to prevent inhalation or swallowing of blood.

**Types of tracheostomy according to site in trachea:**
1. High tracheostomy:
   • Done in the 1st and 2nd tracheal rings, above the thyroid isthmus.
   • Indicated in malignant thyroid and in urgent cases.
   • Disadvantages: it causes perichondritis of cricoid cartilage and permanent stenosis.
2. Mid tracheostomy:
   • Done in the 3rd and 4th tracheal rings, behind the thyroid isthmus and it is the operation of choice.
3. Low Trachesotomy:
   • Done in the 5th and 6th tracheal rings, below the thyroid isthmus.
   • Indicated in subglottic extension of cancer larynx.
   • Disadvantages:
      § The tube is easily slipped because the trachea is deep.
      § It may cause injury of the pleura and neck vessels specially in children.

**Types of tracheostomy tubes according to its materials:** Fig. (26)
• May be metallic or plastic.
• Composed of inner and outer tubes. The inner tube is longer than the outer so that it is easily removed for cleaning if obstructed while the outer tube is left in place.
• Plastic tube with cuff.

![Fig. (26): Tracheostomy tube](image1)
![Fig. (27): Patient with tracheostomy tube after total laryngectomy](image2)
Steps of tracheostomy. Fig. (28)

1. Anaesthesia:
   - No anaesthesia: in unconscious patient.
   - Local anaesthesia: in emergency cases. Usually xylocaine solution 2 % is used with adrenaline 1 / 1000.
   - General anaesthesia: in elective cases.

2. Position:
   - Supine with a pillow under the shoulders to allow extension of the neck.

3. Incision:
   - Vertical incision: from the upper border of cricoid cartilage to suprasternal notch. It is usually done in emergency cases and gives rapid exposure of the trachea but it is not cosmetic.
   - Transverse incision: midway between cricoid cartilage and suprasternal notch. It is usually done in elective cases and is more cosmetic but takes more time than the vertical one.

4. Steps:
   - Incision of the skin and fascia down to the strap muscles which are retracted laterally.
   - The isthmus is divided and transfixed to avoid bleeding and leakage of thyroxin.
   - The pretracheal fascia is cut and cleared from the trachea.
   - A cricoid hook is applied to fix and elevate the trachea.
   - An incision is done in the 3rd and 4th tracheal rings; a tracheal dilator is inserted to widen the opening and a suitable tracheostomy tube is inserted.
   - The tube is fixed by ribbons around the neck and the wound is closed without tension.

Post operative care:
- Position: semi-sitting to help easy breathing and effective coughing.
- Room atmosphere should be humidified with steam to compensate for the moisture lost.
- Observation of respiration with the tracheostomy tube by:
  - Movement of a piece of cotton or condensation of water vapour over a mirror placed in front of the tube.
  - The patient cannot speak.
- Broad spectrum antibiotics.
- Care of the tube:
  - Repeated suction through the tracheostomy to avoid tube blocking.
  - Repeated removal of the inner tube to clean it.
- Decannulation: i.e removal of the tube after treatment of the cause.
- The tube is closed with a cork as a test and the patient is observed for 2 days.
Complications of tracheostomy:

1. Anaesthetic complications:
   - Shock from local anaesthesia (idiosyncrasy or overdosage).

2. Haemorrhage:
   - Primary hemorrhage: Occurs during operation. Treated by ligation or coagulation.
   - Reactionary hemorrhage: Occurs within the first 24 hours due to slipped ligature. Treated by ligation.
   - Secondary hemorrhage: Occurs between the 5th and 10th day due to sepsis. Treated by antibiotics and ligation.
3. Pulmonary complications:
   - Apnea: arrest of respiration due to sudden wash of CO₂ after tracheostomy. The respiratory centre fails because it has been accommodated on a high level of CO₂.
     Treatment:
     β Close the tube for a while or give a mixture of O₂ and CO₂ 10%.

   - Pulmonary oedema: Intra-alveolar exudation causing noisy respiration, froth and cyanosis. It occurs due to sudden drop of intra-alveolar pressure after opening the trachea leading to increased capillary permeability and intra-alveolar exudation.
     Treatment:
     β Respiratory and cardiac stimulants and connect the tube to underwater seal system making expiration under pressure.

   - Pneumothorax: Due to injury of the pleura leading to entry of the air in the pleural cavity. The right pleura apex is high, specially in children and females, and may be injured during operation.
     Treatment:
     β Aspiration of the air through an intercostal needle and connected to underwater seal.

   - Surgical emphysema: -
     In the neck:
     β It is air spreading under the skin. It occurs due to leakage of air from around the tube if it is small in relation to a wide tracheal opening or tight closure of the wound.
     Treatment:
     β Remove the sutures of the wound and insert a suitable tracheostomy tube. Very rarely, multiple skin incision may be needed.

     In the mediastinum:
     β Air extending down to the mediastinum causing cardiac embarrassment. It occurs due to forgetting to incise the pretracheal fascia before opening the trachea.

4. Injury of important structures:
   - Injury of blood vessels causes haemorrhage.
   - Injury of pleura causes pneumothorax.
   - Injury of the cricoid cartilage in high tracheostomy leads to perichondritis and permanent subglottic stenosis.
   - Injury of oesophagus posteriorly by deep incision of trachea or by long tube causes tracheo-oesophageal fistula.
5. Complications of tracheostomy tube: Fig. (29)
   • Small tube leads to surgical emphysema.
   • Long tube causes tracheo-oesophageal fistula.
   • Obstructed tube by mucous or crustations causes stridor.
   • Slipped tube specially in low tracheostomy causes stridor.
   • Epithelialization of the site of the tube → tracheocutaneous fistula.
   • Failure of decannulation due to laryngeal or tracheal stenosis.

Fig. (29): Complications of tracheostomy tube
Frunculosis of EAC
Otomyecosis
Acute suppurative otitis media

ASOM bulging T. M.
Tubo-Tympanic CSOM
Attico-Antral CSOM (Attic cholesteatoma)

Marginal perforation of T. M.
Adhesive otitis media
Aural polyp

Glomus tumour (sunrise appearance)
Glomus jugulare tumour
Mastoiditis
Dilatated nasal polyps  Frunculosis  Orbital abscess

Posterior rhinoscopy in sinusitis  Rhinoscleroma  Frontal mucosa

Oromaxillary fistula  Haemangiomata of the nose  Perforation of nasal septum

Nasal polyp  Antrochoanal polyp  Antrochoanal polyp in nasopharynx