ENT

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Preface

This summary is designed to organize the information presented in lecture slides into a clear, uniform, and structured format. Its goal is to simplify revision and make theoretical content easier to follow.

Is this summary enough for the Mini OSCE?

Short answer: **No (not even a little).**

The Mini-OSCE is designed to test your knowledge from a **clinical and practical angle**. It focuses on:

- Pattern recognition
- Image interpretation
- Short, structured answers

It does **not require deep theoretical detail**. Minimal background knowledge, combined with repeated exposure to cases, is usually sufficient.

Are Dr. Amer Sawalha's videos and Mini-OSCE summaries enough?

Most probably, yes. These resources are specifically tailored for OSCE performance.

Is this summary enough for the Final exam?

Short answer: **Maybe.**

The Final exam is designed to test:

- Deeper understanding
- Memorization of core concepts
- To a lesser extent, clinical reasoning (which is not covered in slides as in Mini-OSCE resources)

This summary is intended to serve as a **strong, slide-based theoretical source**. It focuses on what is presented in lectures and intentionally excludes information that belongs to OSCE-focused resources. This creates a clear distinction between:

- Theory (slides / Final exam)
- Clinical application (Mini OSCE resources)

Are Mini OSCE resources enough for the Final exam?

No.

They are not sufficient for understanding mechanisms, classifications, or detailed concepts required in the Final.

Recommendations

- **PAST PAPERS**
- **If you have enough time: Study both resources in parallel**
  1. Start with this summary to build a solid theoretical foundation
  2. Then use Mini OSCE resources to practice pattern recognition and cover clinically oriented topics not emphasized in slides
- **If you have limited time:**
  - For the Mini OSCE → Dr. Amer's videos + Other mini-OSCE sources
  - For the Final → Dr. Amer's videos at increased speed (×2, ×3 …) + This Summary

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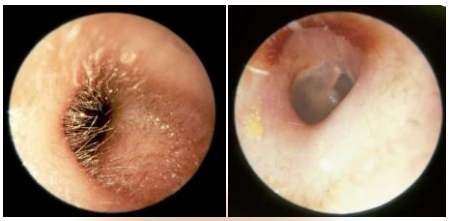
External Ear Conditions

Anatomy of the External Ear

Functions

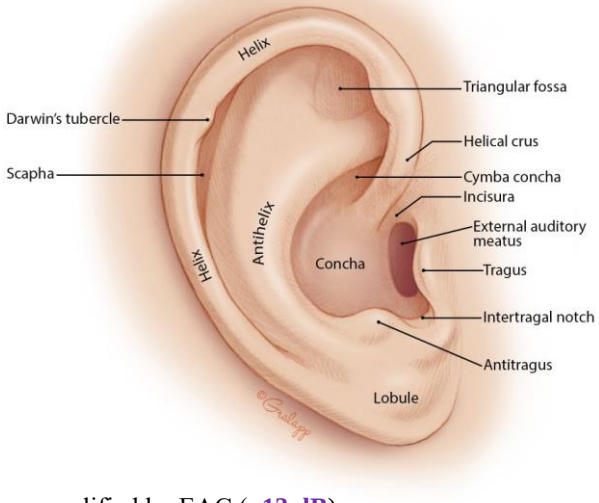
- Sound collection & amplification
- Protection

Components

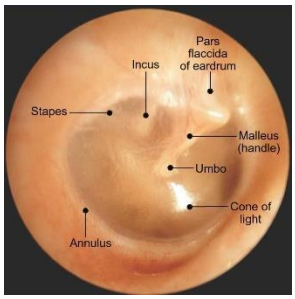


1. Auricle (Pinna)
2. External Auditory Canal (EAC)
    - Lateral 1/3 → cartilaginous
        - Has **ceruminous glands** (wax)
    - Medial 2/3 → bony
        - Thin skin → more sensitive to trauma
3. Tympanic Membrane (lateral surface)

Key Notes



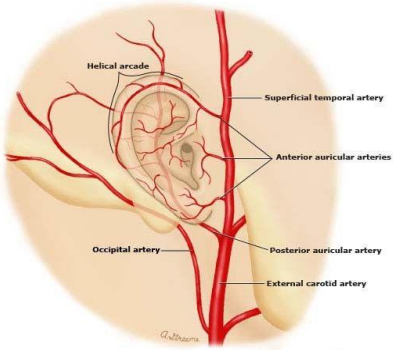
- **Auricle (Pinna)** made of cartilage (except lobule)
    - Functions of Auricle:
        - Collect sound
        - Conduct sound to EAC
- **External Auditory Canal (EAC)**
    - **S-shaped canal**
    - Length: **~2.5 cm**
    - Diameter: **~0.7 cm**
    - Has 2 constrictions (مهم):
        - Osseocartilaginous junction
        - Isthmus (≈0.5 cm from TM)
    - Functions of EAC:
        - Conduct sound to TM
        - **Amplify sound**
            - Frequency that is 3–6 kHz → amplified by EAC (**~13 dB**)
        - Protect TM by:
            - Wax (cerumen)
            - S-shape
    - **Self-Cleaning Mechanism**
        - Epithelial migration outward
        - Jaw movement (chewing, speaking)
- **Tympanic Membrane (TM)**
    - It is the boundary between outer & middle ear
    - Semi-transparent, Concave (from lateral surface), Oval
    - The TM is angled downwards, forwards, and laterally
    - **3 Layers:**
        - Outer (epithelial)
        - Middle (fibrous)
            - Pars flaccida lacks fibrous layer
        - Inner (mucosal)
    - **Orientation rule based on the handle of malleus:**
        - Left ear → 11 o'clock position
        - Right ear → 1 o'clock position
    - **Functions**
        - Converts sound waves → mechanical energy
        - Amplifies sound (**~20–23 dB**) via:
            - Area difference (TM vs stapes footplate)
            - Ossicular lever system
        - Protection via:
            - Acoustic stapedius muscle reflex (**ASMR**)



Blood Supply

From **external carotid artery** branches:

1. Posterior auricular artery
2. Superficial temporal artery → Anterior auricular arteries
3. Occipital artery
4. **Maxillary artery** (deep auricular branch → EAC + TM)



Venous drainage

- Follows arterial supply

Lymphatic Drainage

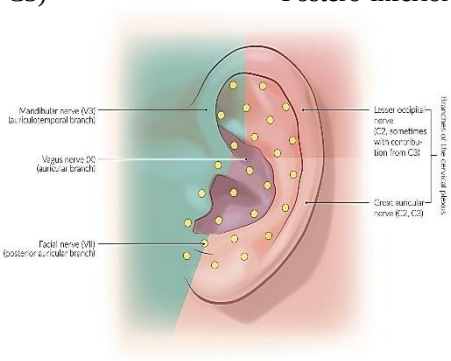
- Anterior → Superficial parotid LNs
- Posterior → Mastoid LNs

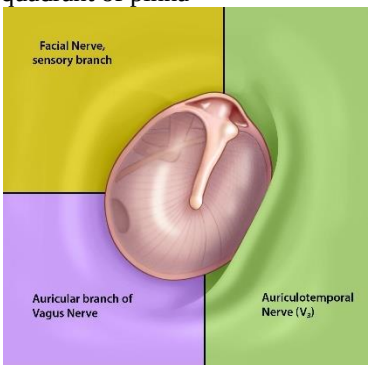
Nerve Supply

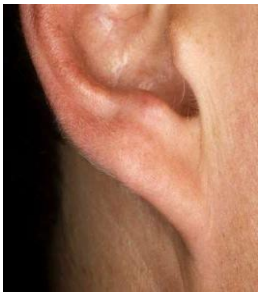
- Facial nerve (VII) → Posterior auricular branch → Postero-superior quadrant of TM
- Vagus nerve (X) → Auricular branch → Postero-inferior quadrant of TM & **EAC**
- Mandibular (V3) → Auriculotemporal branch → Anterior half of TM & Anterior half of pinna
- Lesser occipital nerve (C2) → Postero-superior quadrant of pinna
- Great auricular nerve (C2–C3) → Postero-inferior quadrant of pinna

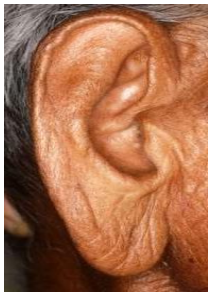
Minor Anatomical Variations

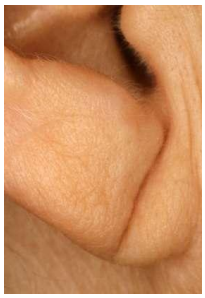
1. Attached lobule
2. Elongated lobule
3. Creased lobule
4. Darwin's tubercle
5. Reverse Darwin's tubercle

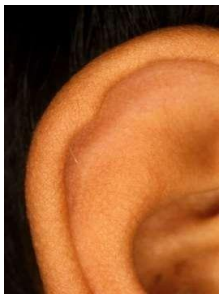


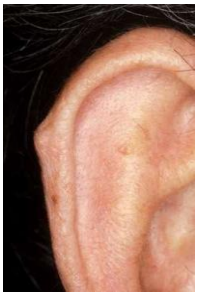








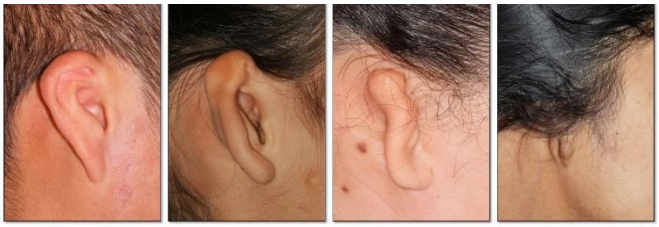


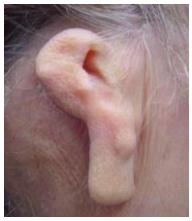


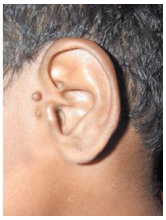
Diseases of the External Ear

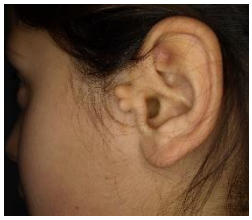
A. Congenital

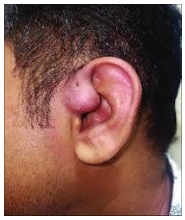
1. Congenital Aural Atresia (Graded into 4 grades)
    - Microtia
    - Anotia
2. Congenital EAC Atresia
3. Bat Ears → **Otoplasty**
4. Auricular Appendages (tags)
5. Preauricular Sinus / cyst
    - Pit/sinus near tragus–helix junction
    - Can get infected

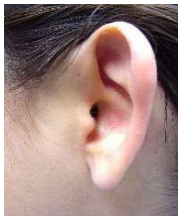












B. Acquired

1. Auricular Hematoma

- Blood collection under perichondrium

Cause:

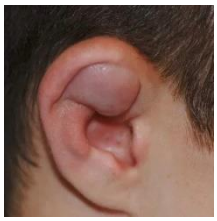
- Blunt trauma

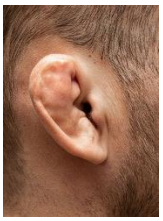
Complication:

- Cartilage necrosis → **Cauliflower Ear (Boxer's Ear)**

Treatment:

- Small → aspiration
- Large → incision & drainage + pressure dressing





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2. Keloid

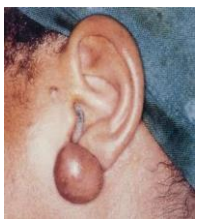
- Excess collagen scar beyond wound margin

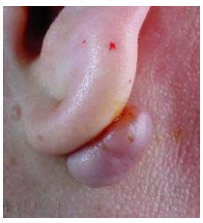
Features:

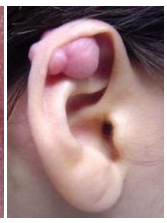
- Common after ear piercing
- More in dark-skinned individuals
- Common at lobule

Treatment:

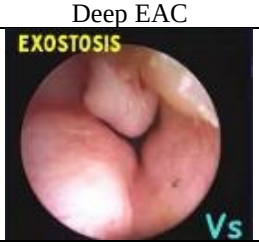
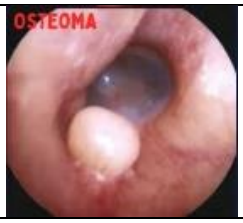
- Intralesional steroids
- Surgical excision
    - Excision + Steroids (↓ recurrence)
- Radiotherapy (recurrent cases)







3. EAC Bony Lesions

Feature	A. Exostosis	B. Osteoma
Nature	Reactive bone growth Associated with cold water exposure e.g., **Swimmers**	True benign tumor
Number	Multiple	Single
Laterality	Bilateral	Unilateral
Shape	Sessile	Pedunculated
Location	Deep EAC	Close to isthmus
Otoscopy		
Management	If caused obstruction → **Surgery**	

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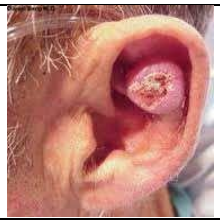
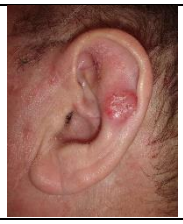
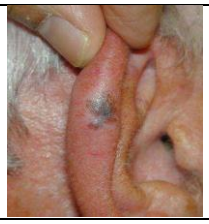
4. Malignant Tumors

Feature	A. SCC	B. BCC	C. Melanoma
Site	Helix/postauricular	Tragus/helix	Sun-exposed
Lesion	Ulcer, everted edges	Pearly, rolled edges	Pigmented nodule
Growth	Moderate–aggressive	Slow	Rapid
LN spread	Common	Rare	Early
Distant spread	Possible	Very Rare	Common
1ry Treatment	Wide excision ± RT	Wide excision	Wide excision
Advanced Treatment	Neck dissection	Rare	Radical + LN
Prognosis	Intermediate	Best	**Worst**
Presentation			

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5. External Ear Infections / External Ear <u>Inflammatory Conditions</u>

A. Auricle Infections

1. Perichondritis

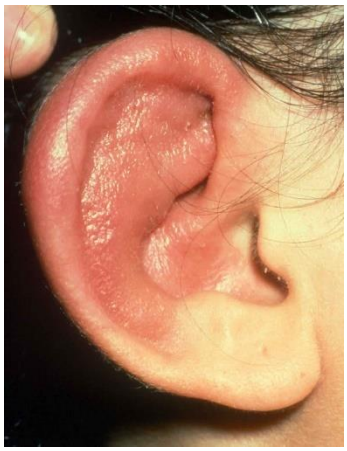
- Infection / Inflammation of auricular cartilage & perichondrium
- Most common organism: **Pseudomonas**

Causes:

- Trauma (especially piercing)
- Infected hematoma / Post-hematoma
- Surgery
- Frostbite
- Extension from otitis externa
- Inflammatory / Autoimmune

Clinical:

- Severe pain
- Red, swollen ear
- **Lobule spared** (important sign)
- ± Abscess



Treatment:

- Systemic anti-pseudomonal antibiotics ± Topical
- ± I&D if abscess
- Debridement
- Pressure dressing

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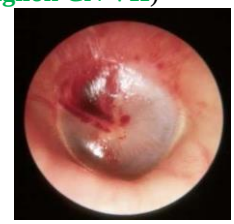
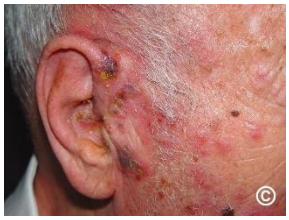
2. Herpes Zoster Oticus (Ramsay Hunt Syndrome)

Cause:

- Reactivation of **VZV** (affecting **Geniculate Ganglion CN VII**)

Clinical:

- Severe, persistent otalgia
- Vesicles on:
  - Pinna
  - EAC
  - Tympanic membrane

Nerve involvement:

- Ramsay Hunt Syndrome Type 2 → Herpes Zoster Oticus + Facial nerve palsy
- **± CN VIII** → hearing loss, vertigo

Treatment:

- Acyclovir + corticosteroids (10–14 days)
- Eye protection (if Type 2)
- Control blood sugar

B. Otitis Externa (EAC Infections)

Definition of Otitis Externa

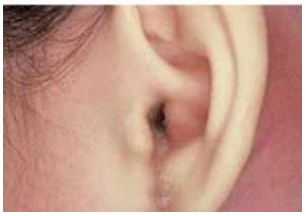
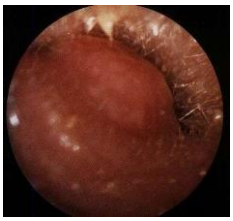
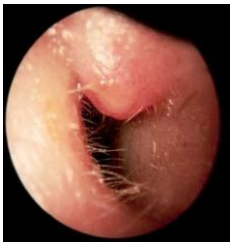
- Infection/inflammation of EAC skin

Epidemiology:

- 10% lifetime incidence
- More common in **summer** (humidity & water exposure)

Types of Otitis Externa

1. Acute Localized Otitis Externa (Furuncle / Boil)

Cause:

- Infection of **hair follicle**
- Usually **Staphylococcus aureus**

Risk factors:

- EAC Trauma
- Cotton buds
- Scratching
- **Diabetes**
- Moisture

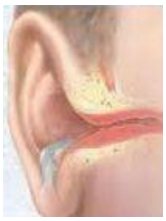
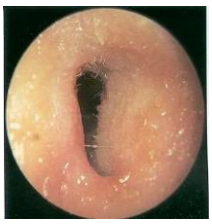
Clinical:

- Severe **throbbing** otalgia
- Tragal tenderness
- Local swelling
- ± Abscess

Treatment:

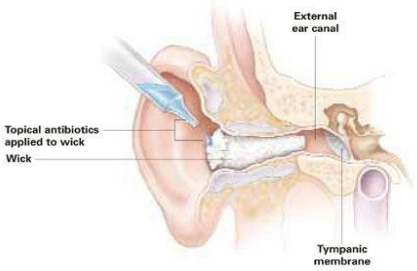
- Analgesics
- Topical anti-staphylococcal antibiotics ± Systemic
- ± I&D if abscess

2. Acute Diffuse Otitis Externa (Swimmer's Ear)

Cause:
- Infection / inflammation of entire EAC
- Usually **Pseudomonas**

Risk factors:
- Water exposure
- Humidity
- EAC Trauma
- Hearing aids
- **Dermatological conditions**…



Clinical:
- Otalgia
- Tragal tenderness
- Canal edema & erythema + purulent discharge
- ± conductive hearing loss

Treatment:
- Aural toilet
  - A medical procedure used to clean the ear canal of wax, discharge, or debris
- Topical antibiotics ± steroids
- Ear wick if swollen
- **Keep ear dry**

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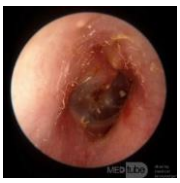
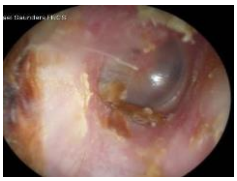
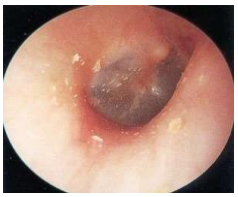
3. Chronic Otitis Externa

Features:
- Symptoms for >2–3 months duration
- **Mixed causes** (bacterial, fungal, dermatologic)

Symptoms:
- Persistent itching
- Mild discomfort



Signs:
- **Dry, flaky skin**
- **Narrow canal**
  - Longstanding infection → thickening of skin → narrowing of lumen

Treatment:
- **Difficult to treat**
- Frequent cleaning
- Long-term topical steroid/antibiotics
- Surgery (last resort)

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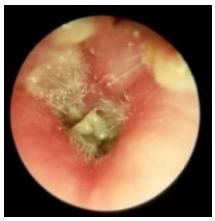
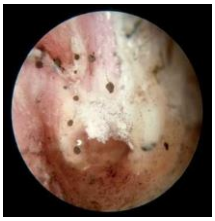
4. Otomycosis (Fungal)

Definition:
- Superficial fungal infection of deep EAC ± TM

Organisms:
- **Aspergillus** (yellow/black/gray) (most common otomycosis)
- **Candida** (white)

Risk factors:
- Dirty water exposure
- Chronic discharge
- Prolonged ear drops
- Diabetes
- Immunosuppression

Clinical:

- **Severe deep pruritis**
- Dull pain
- Hearing loss

Signs:

- Fungal debris:
  - Black / white
  - "Blotting paper" appearance

Treatment:

- Cleaning (dry mopping)
  - Avoid water
- Topical antifungals (Clotrimazole)

---

5. Necrotizing (Malignant) Otitis Externa

Definition

- Severe Otitis Externa → Osteomyelitis of temporal bone
- Lethal infection

Organism:

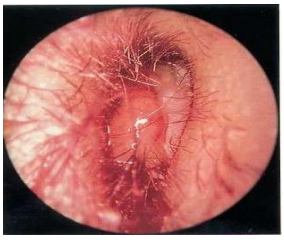
- **Pseudomonas** aeruginosa

Risk group:

- Elderly diabetics
- Immunocompromised

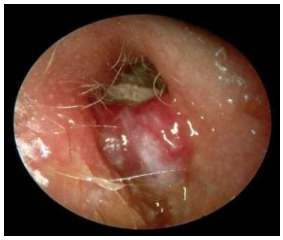
Pathophysiology:

- Starts as otitis externa → spreads outside EAC via:
  - Fissures of Santorini
  - Osseocartilaginous junction



Symptoms:

- Severe deep throbbing pain
- **Chronic discharge**
- Ear fullness



Signs:

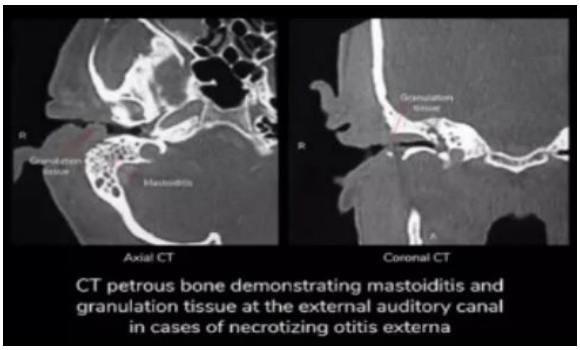
- Granulation tissue
- Purulent discharge
- **Canal obstruction**
- Cranial nerve involvement

Investigations:

- **CT scan** → best initial
- **^{99m}Tc scan** → reveals **osteomyelitis**
- Gallium scan → monitor treatment
- MRI → soft tissue spread

Treatment:

- **Strict glucose control**
- Pain control
- IV anti-pseudomonal **antibiotics** (**4–17 weeks**):
  - 3rd generation Cephalosporins
  - Anti-pseudomonal Penicillins
  - Quinolones
- Hyperbaric oxygen
- Debridement



CT petrous bone demonstrating mastoiditis and granulation tissue at the external auditory canal in cases of necrotizing otitis externa

Anatomy & Physiology of Hearing

Ear Wax (Cerumen)

Importance

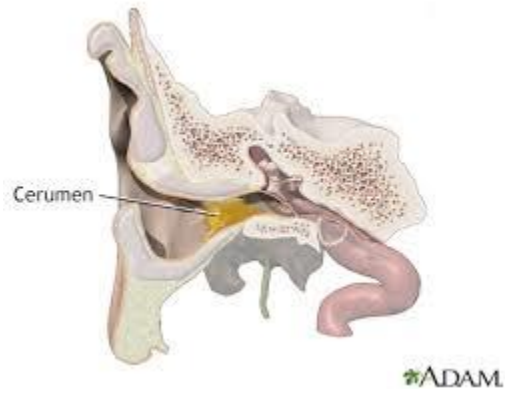
- Ear wax impaction is the most common cause of conductive hearing loss in adults

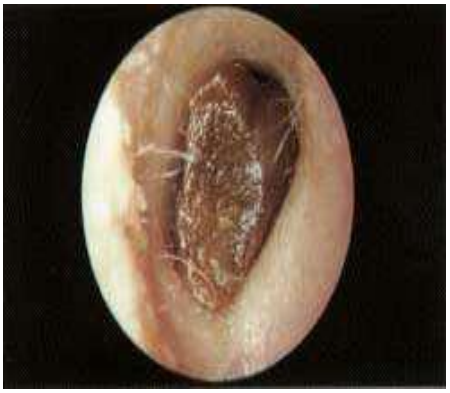
Causes of Impaction

- Excess production
- Improper use of cotton buds (push wax inward)
- Diving (by water pressure)

Symptoms

- Itching
- Otalgia
- Fullness
- Hearing loss, severity depends on:
  - Degree of canal obstruction
  - TM impaction





Acute Otitis Media (AOM)

Definition

- Acute infection of middle ear mucosa
- Rapid onset, **<3 weeks**
- Infective origin
- May extend to:
    - Mastoid air cells
    - Petrous apex air cells
    - Peri-labyrinthine air cells

Epidemiology

- One of the most common childhood infections
- Most common reason for pediatric visits
- Occurs at **all ages** but most prevalent in **childhood** (due to → **short, horizontal eustachian tube**)
- Peak incidence → first year of life (especially 6–12 months)
- There is decrease in incidence with age (except for school entry age 5-6 years)
- 80% of children → at least one episode by age 3
- **Early onset (<9 months) → poor prognostic sign**

Risk Factors

- Age, **male**, race
- Daycare exposure
- Low socioeconomic status
- Lack of breastfeeding (**bottle feeding**)
- Using Pacifier (**لهّاية**)
- Smoke exposure
- Family history
- Medical conditions:
    - **Cleft palate** (↓ after repair)
    - **Down syndrome**
    - **Adenoids**
    - Immunodeficiency
    - Malignancy
    - Procedures: NG, NT tubes
    - Ciliary dysfunction
    - Craniofacial Disorders
        - Treacher-collins syndrome

Routes of Infection

1. Eustachian tube (main route)
2. Hematogenous
3. Through TM perforation or grommet tube

Etiology

→ **Respiratory viruses** detected in ~60-90% of children AOM

1. Mixed AOM (Most Common)

2. Purely Viral AOM

- Respiratory viruses: RSV, Influenza A, Parainfluenza, Rhinovirus, Adenovirus

3. Purely Bacteria AOM

- **Streptococcus pneumoniae** (most common)
- Haemophiles influenzae
- Moraxella catarrhalis
- Streptococcus pyogenes
- Staphylococcus aureus
- **Infants → Gram -ve bacilli**

Clinical Features

A. History

Symptoms:

- **Coryza** (URTI symptoms)
- **Otalgia**, **Otorrhea**
- Feeling of fullness in the ear
- Hearing loss
- Tinnitus

General symptoms:

- Fever
- Irritability
- Poor feeding
- GI: abdominal pain / vomiting / diarrhea / anorexia

B. Physical Examination

- Child ill, febrile, ear rubbing

C. Otoscopy

- **Bulging tympanic membrane** = MOST specific sign
- Redness
- Otorrhea
- Perforated TM (complication)

Stages of AOM (4 stages)

1. Hyperemia
2. Exudation
3. Suppuration
4. Resolution

Differential Diagnosis of AOM

1. OME
2. Chronic OM
3. Otitis externa
4. Bullous myringitis
5. Herpes zoster
6. Referred pain

Treatment

1. Mild cases

- Observation + symptomatic treatment

2. Antibiotics

- First-line: **Amoxicillin–clavulanate**
- Duration:
  - ≥2 years AND Not recurrent → **5–7 days**
  - <2 years OR Severe → **10 days**
- **Alternatives**
  - Penicillin Allergy:
    - Cephalosporins
    - Macrolides
  - **Fluoroquinolones** (2nd line)

3. Procedures

- Therapeutic Tympanocentesis (rare)

Complications

1. Chronic tympanic membrane perforation (rare)
2. Dissemination of infection:
   - **Mastoiditis** (rare) → on imaging: 20% of AOM has mastoid air cell involvement
   - Other sites (rare):
     - Important one to mention →**Meningitis**, **Lateral Sinus Thrombosis** (rare)
3. Facial paralysis (rare)
4. Persistent hearing loss (rare)

Otitis Media with Effusion (OME)

Definition

- Middle ear fluid WITHOUT acute infection
- Also called:
  - Serous Otitis Media
  - **Glue Ear**

Etiology

- Follows AOM
- Eustachian tube dysfunction

Epidemiology

- Very common (asymptomatic):
  - 90% of children by age 4
- Peak: 2–4 years

Risk Factors

- Similar to AOM +
- **Allergies**
- **Obesity**
- **GERD**

Clinical Features

Presentation:

- Caregiver concern about poor hearing or speech delay
- Failed hearing screening

1. History:

- Often asymptomatic
- **NO pain**
- Hearing loss
- Feeling of fullness in the ear
- Tinnitus
- Balance issues (minimal)

2. Otoscopy (Discussed below)

Complications

- Conductive hearing loss
- Myringosclerosis (Sclerosis of TM)
- Retraction pocket (Discussed next lecture)
- Cholesteatoma (Discussed next lecture)

Diagnosis

- Pneumatic Otoscopy
- Tympanometry
  - An objective, painless diagnostic test that measures eardrum mobility and middle ear function by changing air pressure within the ear canal
- Audiometry
  - A non-invasive, subjective test used to evaluate hearing function by measuring an individual's ability to hear sounds at different volumes and pitches (frequencies).

Management

1. First-line

- Watchful waiting (3 months)
- **Tympanostomy** (Grommet tube / Ventilation tube)

2. Other Interventions

- Adenoidectomy
- Balloon dilation of the Eustachian tube
- Hearing aids

3. Unproven OR Ineffective Interventions

- Antibiotics
- Steroids
- Antihistamines
- Decongestants
- Myringotomy without tympanostomy

Otoscopy (most important part of PEx)

OME:

- **Retracted TM**
  - Medialized malleus handle
  - Prominent lateral process of the malleus
  - Disappeared light cone
  - ± Retraction pockets → Cholesteatoma
- **Reduced mobility of TM** → by Pneumatic Otoscopy / Tympanometry
- Opacification of TM
- **Amber** / **Blue** colored middle ear fluid
- Air-fluid level ± bubbles

AOM:

- **Bulging TM**
- **Pus** colored middle ear fluid
- Redness
- Otorrhea
- Perforated TM (complication)

Chronic Suppurative Otitis Media (CSOM)

Definition

- Long-standing infection of middle ear space
- Characterized by:
  - **Persistent otorrhea**
  - **Permanent TM perforation**

Note:

- Perforation is permanent when:
  - Edges are lined by squamous epithelium
  - Does not heal spontaneously

Classification

1. Tubotympanic Disease

Other Names

1. Tubotympanic CSOM
2. Safe Type CSOM
3. Mucosal CSOM
4. CSOM without cholesteatoma

Etiology

- Complication of AOM
- Ascending infection via Eustachian tube from:
  - Adenoids
  - Sinuses
  - Tonsils
- Allergy-related otorrhea (persistent mucoid otorrhea)

Causative Organisms

1. **Pseudomonas aeruginosa**
2. Proteus
3. E. coli
4. Staphylococcus aureus
5. Anaerobes (Bacteroides, anaerobic streptococci)

Types

A. Tympanic membrane perforation
B. Pars tensa retraction

A. Tympanic membrane perforation

Pathology

- Central perforation of pars tensa
- No squamous epithelium invasion

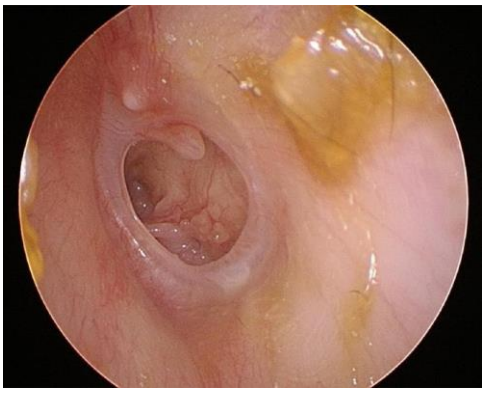
Subtypes

1. Active:

- **Wet Perforation** → Mucosal Inflammation + Mucopurulent otorrhea

2. Inactive:

- **Dry Perforation** → No Mucosal Inflammation + No otorrhea
- No inflammation



Clinical Assessment

A. History

- **Otorrhea**
- Hearing loss

B. Examination

- TM perforation
- Red, edematous mucosa
- Possible **aural polyp**

C. Investigations

- Tuning fork tests
- Audiometry (PTA)
- **Culture** & sensitivity of discharge
- CT temporal bone

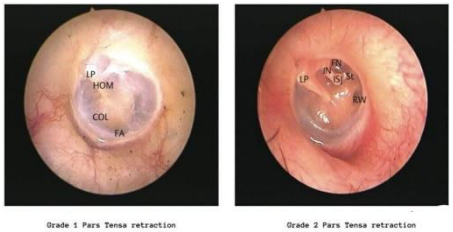
Management

A. Medical:

- Aural toilet
- Ear drops
- Antibiotics
- **Keep ear dry**
- Treat causes:
  - Adenoids
  - Sinusitis
  - Tonsillitis
  - Allergy

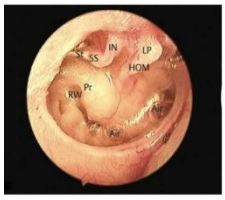
B. Surgical:

- Aural polyp removal
- **Myringoplasty** ± ossicular reconstruction

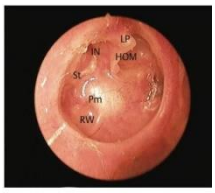


Grade 1 Pars Tensa retraction

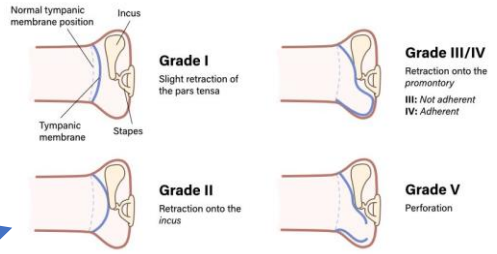
Grade 2 Pars Tensa retraction



Grade 3 Pars Tensa retraction



Grade 4 Pars Tensa Retraction



B. Pars Tensa Retraction

Pars Tensa Retraction → 5 Grades

Cause

- **Eustachian Tube Dysfunction** → chronic negative middle ear pressure → OME → Retraction

Management Options

1. Observation
   - Serial audiometry
2. **Tympanostomy** (Grommet tube)
3. **Tympanoplasty** (cartilage reinforcement)
4. Mastoidectomy (selected cases)

---

2. Atticoantral Disease

Other Names

1. Atticoantral CSOM
2. Unsafe Type CSOM
3. Squamosal CSOM
4. CSOM with cholesteatoma

Definition

- Usually affects the posterosuperior region of middle ear:
  - Attic (Superior middle ear)
  - Posterior middle ear
  - Antrum of mastoid
  - Mastoid air cell system

Key Feature →→→ Inflammation-induced osteoclast activation → Bone destruction

Types

1. Inactive

- Retraction pockets in pars tensa (usually the posterosuperior region) or pars flaccida
- **No discharge initially**

2. Active

- **Cholesteatoma** present of the posterosuperior region of middle ear
- **Foul-smelling otorrhea**
- Bone erosion + granulation tissue

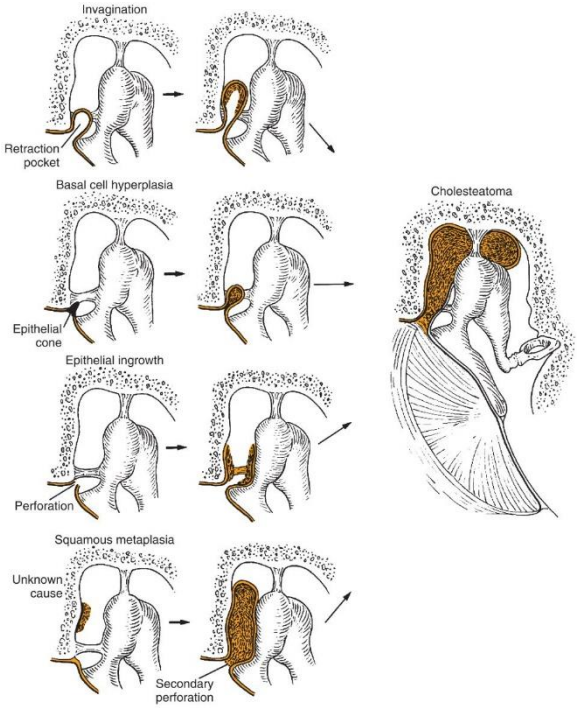
Common Pathogens

- **Pseudomonas** (most common)
- Staphylococcus aureus
- Klebsiella
- Proteus
- Anaerobes
- Polymicrobial
- Fungi

Cholesteatoma

Definition:

- Keratinizing squamous epithelium in middle ear



Types of Cholesteatomas

1. **Primary Acquired Cholesteatoma**
   - From retraction pocket
2. **Secondary Acquired Cholesteatoma**
   - Through TM perforation
3. **Congenital Cholesteatoma**
   - Behind intact TM

Clinical Features

A. History

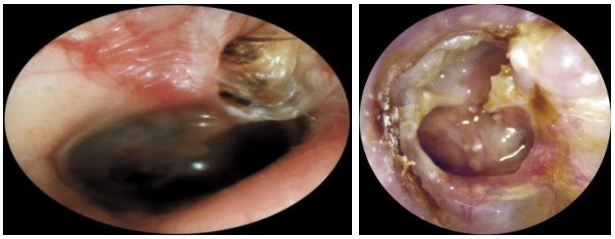
- Persistent foul-smelling otorrhea
- Hearing loss
- Bleeding

B. Otoscopy

- Retraction pocket
- Cholesteatoma debris

C. Investigations

- Tuning fork tests
- Audiometry
- **Culture** & sensitivity of discharge
- **CT temporal bone**
  - Is not needed for diagnosis
  - Indications:
    - Complications are suspected
    - Prior to surgery

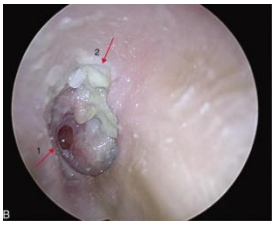


- MRI

Complications

A. Intra-temporal

- **Facial nerve palsy**
- Labyrinthitis
  - Vestibular Ataxia
  - Vertigo
- Petrositis (**Gradenigo syndrome**)
  - Diplopia
- Mastoiditis



B. Intra-cranial

- **Meningitis**
- **Abscesses**
- **Lateral sinus thrombosis**

Management → Surgical (Main Treatment)

Types:

1. Radical mastoidectomy
2. Modified radical mastoidectomy
3. Cortical mastoidectomy
    - Canal Wall Up Procedure
    - Canal Wall Down Procedure

Feature	Canal Wall Up	Canal Wall Down
Appearance	**Normal**	**Open cavity**
Hearing outcome	**Better**	**Poorer**
Regular Cleaning	**Not needed**	**Needed**
Swimming	**Allowed**	**Restricted**
Second surgery	**Needed**	**Not needed**
Recurrence	**High**	**Low**

Note: *Second surgery may be needed to check if there is residual disease*

Hearing Assessment in Children

Types of Hearing Loss

1. Conductive Hearing Loss
2. Sensorineural Hearing Loss
    - Most common due to cochlear pathology
    - Prevalence: 4/1000 children (14/1000 children in Jordan)
    - 50–60% genetic
    - 20% cCMV infection
    - 20-30% environmental
3. Mixed Hearing Loss

Screening for Hearing Loss in Children

- All newborns should be screened (by 1 month)
- High-risk children → repeated screening

Risk Factors

- **50% of hearing loss has NO known risk factors**

A. Prenatal

- Radiation exposure
- TORCH infections:
    - Toxoplasmosis
    - Rubella
    - **cCMV**
    - Herpes

B. Perinatal

- Prematurity
- Low birth weight (<1500 g)
- Hypoxia
- Hyperbilirubinemia

C. Postnatal

- Meningitis
- Head trauma
- Child/ Maternal ototoxic drugs use

D. Other

- Family history
- Syndromes / craniofacial anomalies
- **Parental suspicion of hearing loss**
- **Parental concern of delayed speech & language**

Clinical Evaluation

A. History

B. Physical Examination

C. Audiological Assessment Tools

1. Objective Tests (infants / uncooperative)

1. Otoacoustic Emission (OAE)
2. Auditory Brainstem Response Audiometry (ABR)

2. Behavioral Tests

1. **Pure Tone Audiometry (PTA)**
2. Play Audiometry
3. Visual Reinforcement Audiometry (VRA)

3. All should be combined with → **Tympanometry** (middle ear assessment)

A. Objective Tests

1. Otoacoustic Emissions (OAE)

Principle:

- A probe sends sounds & detects sounds sent back by outer hair cells contraction

Features:

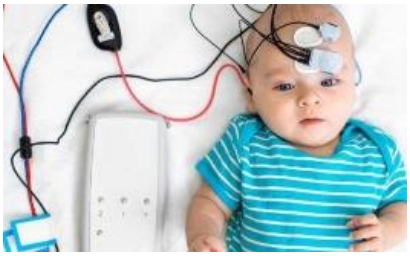
- Objective, **cost-effective**, quick screening tool
- Sensitivity & Specificity: 80–90%
- Cannot detect auditory neuropathy
- Affected by:
  - Middle ear pathology
  - External ear pathology

2. Auditory Brainstem Response (ABR)

Principle:

- Measures brainstem electrical activity after acoustic stimulation

Features:



- Confirmatory tool
- More specific than OAE
- More expensive
- Requires → Sleep / Sedation
- Detects auditory neuropathy
- Can be used to estimate hearing sensitivity

B. Behavioral Tests

1. Pure Tone Audiometry (PTA)

Principle:

- Child wears headphones (Air C.) and sometimes a bone oscillator (Bone C.)
- Pure tones of varying frequency and intensity are presented → lowest intensity heard (threshold) is recorded

Requirements:

- Child cooperation
- Age → >4 years

Interpretation:

- Normal hearing → AC ≈ BC (**≤ 25 dB**)
- Conductive loss → BC normal, AC reduced
- Sensorineural loss → AC = BC, both reduced
- Mixed loss → AC worse than BC, both reduced

2. Play Audiometry

- A modification of standard PTA → makes testing easier

3. Visual Reinforcement Audiometry (VRA)

- A modification of standard PTA
- Used in younger children

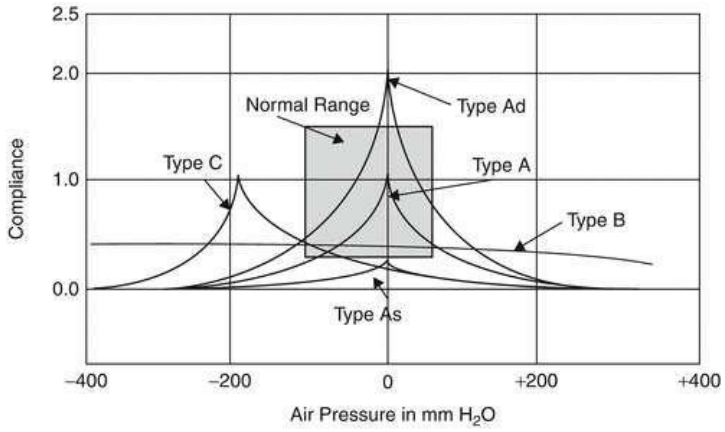
C. Tympanometry

Purpose

- Assess middle ear function
- Device measures compliance (mobility) of the eardrum with changing air pressure

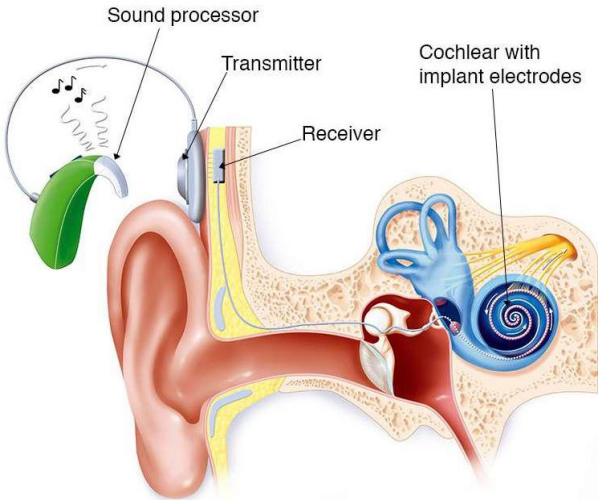
Findings

- **Type A (Normal):**
  - Peak compliance at 0 mmH2O air pressure → Normal middle ear
- **Type As (Shallow)**
  - Peak compliance at 0 mmH2O air pressure
  - BUT reduced compliance → **Stiff middle ear system**
- **Type Ad (Deep)**
  - Peak compliance at 0 mmH2O air pressure
  - BUT excessively high compliance → **Hypermobile middle ear system**
- **Type B (Flat):**
  - No peak → No change in TM motility with changing air pressure → Effusion, Perforation, Wax
  - Wax should be removed before any further assessment (PTA or Tympanometry)
- **Type C (Negative pressure):**
  - Peak shifted left → **Eustachian tube dysfunction**



Rehabilitation Options

1. Hearing aids
2. Cochlear implants →→→
3. Speech therapy
4. Sign language
5. Lip reading



Note: PTA & Tympanometry are one of the most important topics to master in ENT. For these topics, this summary is NOT sufficient for *Mini-OSCE, Final & even the ENT clerkship quiz*

Deafness in Adults

Auditory System Overview

Division

A. Peripheral system

- Outer ear
- Middle ear
- Inner ear

B. Central system

- Brainstem
- Temporal lobe cortex

A. Peripheral system

1. Outer Ear

A. Auricle
B. EAC
C. TM

2. Middle Ear

- An **air-filled** space containing:
- 3 Ossicles:
  - Malleus
  - Incus
  - Stapes
- 2 Tendons:
  - Tensor tympani muscle tendon
  - Stapedius muscle tendon
- Eustachian tube

3. Inner Ear

- Cochlea
  - A **fluid-filled** compartment (perilymph & endolymph)
  - Containing many microscopic components
  - Responsible of → hearing
- Vestibular system:
  - 3 Semicircular canals:
    - Lateral (Horizontal) canal
    - Anterior (Superior) canal
    - Posterior canal
  - 2 Otolithic organs:
    - Utricle
    - Saccule
      - Responsible of → linear acceleration and head position (static equilibrium)
- CN VIII

B. Central system

- **Cochlea → CN VIII** (1st order neuron / **spiral ganglion**)

1. Cochlear nucleus (medulla)
2. Superior olivary complex (pons, first site of bilateral input)
3. Lateral lemniscus
4. Inferior colliculus (midbrain)
5. **Medial geniculate body** (thalamus)
6. Auditory radiations
7. Primary auditory cortex (temporal lobe)

Physiology of Hearing

Requirements of hearing

1. Source (sound)
2. Medium
3. Receptor (ear)

Acoustic Energy Transformation

- Acoustic (outer ear) → mechanical (middle ear) → hydraulic (cochlea) → electromechanical (hair cells) → electrical (CN VIII)

Sound Transmission

1. Air conduction (main pathway)
2. Bone conduction (sound passes directly to inner ear)

Types of Hearing Loss

1. Conductive hearing loss
2. Sensorineural hearing loss
3. Mixed hearing loss
4. Functional (non-organic) hearing loss
5. Central & psychogenic hearing loss

Causes of Hearing Loss

A. Outer Ear Causes

All are conductive hearing loss causes

1. Congenital:

1. Microtia / Anotia
2. EAC Atresia
   - May cause moderate-to-maximal conductive hearing loss
   - Maximum air-bone conduction gap (ABG) **55-65 dBs**

2. Acquired:

1. Infection
2. Tumors (**SCC** most common, BCC, melanoma)
3. Exostosis / Osteoma
4. **Wax impaction** (most common cause of adult conductive hearing loss)
5. Tympanosclerosis

B. Middle Ear Causes

All are conductive hearing loss causes

1. Congenital:

1. Ossicular malformation
   - MC → **Missing / Malalignment of the <u>crura of stapes</u>**

2. Acquired:

A. Infection:
   1. AOM
   2. OME
   3. CSOM → more hearing loss, can become mixed HL if inner ear damaged

B. TM Perforation:
   - Degree of hearing loss depends on size/location
   - **Trauma**:
     - Blast injury
     - Barotrauma (Diving)
     - Self-inflected trauma from Q-tip or other objects
     - Temporal bone fracture

C. Tumors
   - Cholesteatoma (non-neoplastic, destructive, found in CSOM)

D. **Otosclerosis** (stapes fixation)
- AD inheritance, usually unilateral in **young female** (M:F = 1:2)
- Conductive hearing loss (can become mixed HL)
- Treatment → Hearing aids or **Stapedectomy**

E. Eustachian tube dysfunction
- Type C on Tympanogram
- Type B on Tympanogram → if **OME** developed

C. Inner Ear Causes

1. Congenital:
- Hearing loss that from birth or shortly after
- Classification:
  - **Hereditary**
    - 90% AR, 10% AD
  - **Non-hereditary** (TORCH)

2. Presbycusis:
- Age-related hearing loss
- Most common cause in elderly
- **Progressive, symmetrical, bilateral high-frequency SNHL**
- Associated **tinnitus**
- Treatment → Hearing aids (high success rate)

3. Infection:

1. Viral cochleitis
   - Most common infection of inner ear in adults
   - **Sudden SNHL**, **vertigo**, facial paralysis & rarely pain
2. Meningitis
   - Most common infection of inner ear in young children
   - **Profound SNHL**

4. Ménière Disease:
- Triad of **fluctuating symptoms**:
  - Hearing loss
  - Tinnitus
  - Vertigo
- Typically, the attack preceded by a sensation of fullness in the ear

5. Noise Exposure:

1. Noise-induced hearing loss (NIHL)
   - 8hrs./day chronic exposure to noise **>85 dB**
   - Industrial works
2. Acoustic trauma
   - Sudden exposure to high intensity sound briefly

6. Tumor:
- **Acoustic neuroma** (Vestibular schwannoma)
  - Most common benign tumor that causes SNHL
  - Vestibular portion of CN VIII tumor
  - Most common complaint → **unilateral SNHL** + **tinnitus**

7. Neurogenic
- CVA, MS
- Arnold-Chiari malformations

8. Iatrogenic

- Drugs ototoxicity:
  - **Antibiotics** (irreversible SNHL)
    - Aminoglycosides (Gentamycin)
    - Erythromycin
    - Tetracycline
  - **Cisplatin** (irreversible SNHL, among the most common causes of HL)
  - **Diuretics** (Furosemide)
  - **Aspirin** (**reversible** SNHL)
- Radiotherapy
- Surgery:
  - Tympanomastoidectomy
  - Stapedectomy

Degree of Hearing Loss (assessed using pure tone audiometry PTA)

Degree	Hearing level (dB)
Normal	≤25 (adult), ≤15 (child)
Mild	25–40
Moderate	41–55
Moderately severe	56–70
Severe	71–85
Profound	>85

Approach

A. History

B. Examination

- General Exam
- **Otoscopy**
- ENT exam
  - Tuning fork tests

Investigations

- Pure Tone Audiometry
- Tympanogram
- IF needed:
  - Speech audiometry
  - Vestibular tests

Treatment

- Treat cause (medical &/or surgical)
- Hearing aids
- Speech therapy
- Sudden SNHL → Steroids

Tuning Fork Tests

1. Rinne Test

Principle:

- Compare air conduction (AC) vs bone conduction (BC)

Result	Test Result	Interpretation
AC > BC	Positive Rinne	Normal Hearing / SNHL
BC > AC	Negative Rinne	Conductive Hearing Loss

2. Weber Test

Principle:

- Lateralization
- Based on **Stenger principle**
  - States that "when two identical tones of different intensities are presented simultaneously to both ears, only the louder tone is perceived."

Condition	Findings
Normal Hearing / Symmetrical Hearing Loss	Midline (no lateralization)
Conductive Hearing Loss	Lateralizes to affected ear
SNHL	Lateralizes to normal ear

- Very important & high yield:
  - **False Negative Rinne**
    - Occurs when the test incorrectly suggests **conductive hearing loss** in a patient who actually has **profound unilateral sensorineural hearing loss (Dead Ear)**.
    - When the tuning fork is placed on the mastoid bone, the sound is transmitted through the skull and picked up by the opposite (normal) ear. The patient then reports hearing bone conduction better than air conduction, giving a negative Rinne result.
    - This emphasis the importance of doing all fork test to get reliable interpretation.

3. Bing Test

Principle:

- The examiner alternately close and open the ear canal with a finger while the tuning fork is on the mastoid process
- The Bing test is based on the **occlusion effect**:
  - When the EAC is occluded, bone-conducted sound becomes louder in a normal ear
  - This occurs because sound energy is reflected back to the tympanic membrane

Finding	Result	Interpretation
Sound becomes louder when ear is occluded	Positive Bing	Normal hearing or SNHL
No change in sound with occlusion	Negative Bing	Conductive hearing loss

4. Schwabach Test (Outdated test)

Principle:

- Compares the patient's bone conduction (BC) with that of the examiner.

Time Compared to Examiner	Result	Meaning
Equal	Normal Schwabach	Normal
Reduced	Diminished Schwabach	SNHL
Prolonged	Prolonged Schwabach	Conductive Hearing Loss

Tinnitus

Definition

- Aberrant perception of sound without external stimulus
- It is described as ringing, hissing, roaring, clicking, or whooshing

Pathophysiology

- Misinterpretation of signals in the central auditory pathways

Epidemiology

- More common in **males**

Characteristics

Sound Nature

- Tonal:
  - Low- pitched
  - Medium- pitched
  - **High-pitched** → **inner ear**
- Multitonal
- **Noise-like (Crackling)** → **middle ear**

Pattern

- Continuous
- Intermittent
- Pulsatile

Onset

- Sudden
- Gradual

Location

- Ear or head
- Unilateral or bilateral

Types

1. Subjective (most common)

- Not audible to examiner (heard only by patient)

2. Objective (rare)

- Sound is real and externally detectable (heard by examiner)
- Often pulsatile or rhythmic

Causes

A. Objective Tinnitus

1. Vascular (pulsatile)

- Aneurysm
- Glomus tumor
- Carotid body tumor
- AV malformations
- Carotid artery stenosis

2. Muscular

- Palatal myoclonus
- Middle ear muscle spasm (stapedius, tensor tympani) = **Myoclonus**
- Patulous Eustachian tube

3. External canal infestation

4. TMJ disorders (clicking sound)

B. Subjective Tinnitus

1. Idiopathic (most common)

2. Auditory Causes

- Conductive:
  - CHL causes …
- **Sensorineural** (**Tinnitus is common complaint in SNHL patients (80%)**):
  - **Ménière disease**
  - **Acoustic neuroma**
  - **Presbycusis**
  - Noise exposure
  - SNHL causes …

3. Non-auditory Causes

- HTN / Diabetes / Anemia / CVD
- Stress (Psychogenic)
- Allergies
- Obesity
- Drugs

Diagnosis

- History:
- PEx:
  - ENT exam
  - CVS
  - TMJ
  - Neurological exam
- Investigations: (No objective way to measure subjective tinnitus severity)
  - Pure Tone Audiometry
  - Tympanometry
  - Imaging (CT, MRI)
  - Blood tests

Management

1. **Treat (possible) underlying causes**
2. Medications:
   - **Vasodilators** (helps in some patients)
   - Drugs for associated symptoms:
     - Anxiety
     - Depression
3. **Surgery**:
   - **Ménière** → Vestibular nerve section (Limited benefit → ~50%)
   - **The Phantom Limb Concept** explains why surgery often fails to eliminate the tinnitus, because the perception is generated centrally rather than peripherally. An initial ear injury may trigger lasting changes in the brain, so tinnitus persists and can even worsen due to central neural reorganization and psychological reinforcement.
4. Lifestyle modification
5. Sound Therapy (Masking → White noise devices)
6. Advanced:
   - Electrical stimulation
   - Hearing aids / Cochlear implants
7. Psychological interventions:
   - Stress management
   - Relaxation techniques
   - Self-hypnosis
   - Biofeedback
   - Desensitization
   - Cognitive Behavioral Therapy (**CBT**)

Vertigo

Balance System

Balance depends on 3 systems:

1. Vestibular system
2. Visual system
3. Proprioceptive system

These inputs are integrated centrally (brainstem + cerebellum) → to maintain balance

Sensory Conflict & Compensation

- If one system is unreliable → Brain increases reliance on others

Vestibular System

A. Semicircular Canals (Angular Motion Detectors)

Structure:

- 3 canals:
  - Horizontal (lateral)
  - Anterior (superior)
  - Posterior
- Each canal has:
  - Ampulla → contains **crista ampullaris**
  - Hair cells embedded in **cupula** (gel-like structure)

Mechanism (step-by-step):

1. Head rotates
2. Endolymph lags behind (principle of inertia = القصور الذاتي)
   - تخيلها زي الي بصير فيك لما تسرع/ تبطئ فجأة وانت بتسوق
3. This deflects the cupula
4. Hair cells bend which leads to → depolarization or hyperpolarization depending on direction

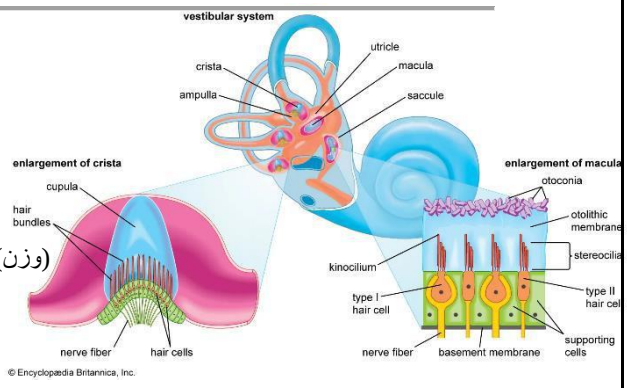
Push–Pull System:

- Canals work in pairs (left vs right):
  - Example: turning head right:
    - Right horizontal canal → excited
    - Left horizontal canal → inhibited
- This bilateral comparison increases sensitivity and accuracy

What they detect:

- **Angular acceleration** ONLY
- Do NOT detect constant velocity well (adaptation occurs)

B. Otolithic Organs (Linear Motion + Gravity)



Structure:

- Utricle → horizontal plane
- Saccule → vertical plane

Contain:

- Macula (sensory epithelium)
- Hair cells embedded in otolithic membrane
- Covered by otoconia ($CaCO_3$ crystals) → add mass (وزن)

Mechanism:

1. Linear acceleration or head tilt occurs
2. Heavy otoconia shift due to gravity/inertia
3. This shears the hair cells
4. Leads to → depolarization or hyperpolarization depending on direction

What they detect:

- **Linear acceleration**:
  - Forward/backward (car movement)
  - Up/down (elevator)
- **Static head position relative to gravity**

Structure & What Detects

Structure	Detects
Semicircular canals	Angular acceleration
Utricle	Horizontal linear acceleration
Saccule	Vertical linear acceleration

Vestibulo-Ocular Reflex (VOR)

Function:

- Keeps vision stable during head movement

Mechanism:

1. Head turns right
2. Eyes move left automatically
3. Image remains stable on retina

Pathway:

- Semicircular canals → vestibular nuclei → cranial nerves:
  - CN III (oculomotor)
  - CN IV (trochlear)
  - CN VI (abducens)

Clinical importance:

- Tested by:
  - Head impulse test
  - Caloric testing

Vestibulospinal Reflexes

Function:

- Maintain posture and balance

Two main tracts:

- Lateral vestibulospinal tract:
  - Facilitates extensor muscles
  - Maintains upright posture
- Medial vestibulospinal tract:
  - Controls head and neck position

Definition of Vertigo

- Vertigo is:
  - A false sensation of movement (usually rotational)
  - A symptom, not a diagnosis
  - A subtype of dizziness
- It has significant clinical importance due to major impact on quality of life

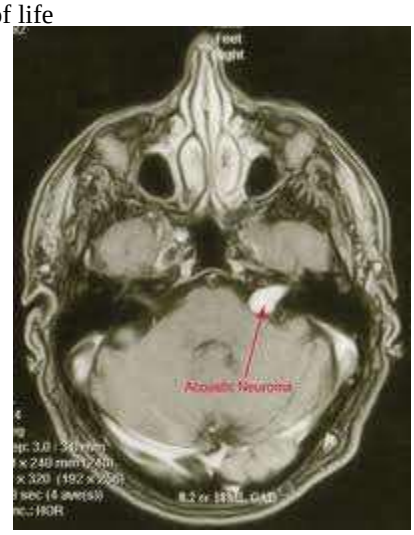
Epidemiology

- Common in emergency and primary care settings
- **Female** predominance (M:F = 1:2.7)
- More frequent in **elderly** (≈3× higher)
- High rate of misdiagnosis (often labeled non-vestibular incorrectly)

Etiology of Vertigo

A. Peripheral (Vestibular) Causes

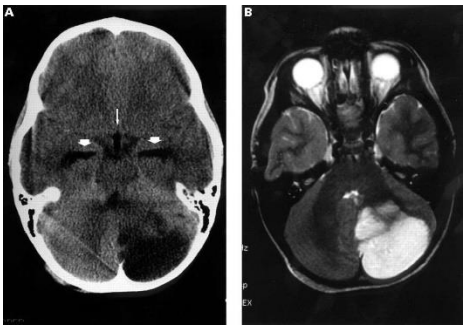
- **Benign paroxysmal positional vertigo (BPPV)**
- **Vestibular neuritis**
- **Ménière disease**
- Labyrinthitis
- Herpes zoster oticus (Ramsay Hunt syndrome)
- Perilymphatic fistula
- Semicircular canal dehiscence
- **Acoustic neuroma**



- Drug-induced (e.g., aminoglycosides)
- Otitis media / cholesteatoma
- Post-surgical causes

B. Central Causes

- **Vestibular migraine**
- **Brainstem ischemia**
- **Cerebellar infarction**/hemorrhage
- **Multiple sclerosis**
- Chiari malformation
- Episodic ataxia type 2



C. Non-Specific Dizziness Causes

- Cardiovascular → arrhythmias, hypotension
- Metabolic → **diabetes**, **anemia**, **hypothyroidism**
- Neuropathy → toxins, infections, **vitamin deficiencies (B1, B6, B12)**
- Proprioceptive disorders
- Psychogenic causes

Clinical Evaluation

A. History (most important)

Key components:

1. **Time Course**
   - **Never permanent** even if lesion persists
   - May present after single or recurrent episodes
   - Duration varies:
     - **Seconds** (**BPPV**)
     - **Minutes–hours** (**Ménière**)
     - **Days** (**Vestibular Neuritis**)
2. **Provoking Factors**
   - Head movements (**positional vertigo**)
   - Coughing/sneezing/exertion/loud noises (**Tullio phenomenon**)
   - Trauma or barotrauma
   - Viral illness
   - Anxiety, visual triggers (e.g., 3D movies)
3. **Associated Symptoms**
   - Nausea, vomiting
   - Hearing loss, tinnitus, ear fullness
   - Neurological deficits (diplopia, weakness)
   - Headache, aura (migraine)
   - Cardiovascular symptoms (palpitations)
4. **Past History**
   - Migraine
   - Stroke risk factors
   - Drug history (ototoxic or cerebellar drugs)
   - Trauma or family history

B. Physical Examination

General Components

- General examination
- Otologic exam → Otoscopy + Hearing Tests
- Neurologic exam
- Cardiovascular exam

Key Elements in PEx

1. **Nystagmus**
2. **Positional Testing (Dix-Hallpike)** → Diagnoses **BPPV**
3. **Balance Tests**:
    - Romberg test
    - Unterberger test
4. **Hearing Tests**

Investigations

- So many (based on H&P)…

Management of Vertigo

A. Pharmacotherapy

1. **Antihistamines**:
    - Meclizine, dimenhydrinate, cinnarizine
2. **Benzodiazepines**:
    - Diazepam, lorazepam, clonazepam
3. **Antiemetics**:
    - Metoclopramide, ondansetron, prochlorperazine
4. Others:
    - **Hydrochlorothiazide** (Ménière disease)
    - **Corticosteroids** (Vestibular neuritis)
    - **Betahistine** (BPPV)
    - Immunosuppressants (selected cases)

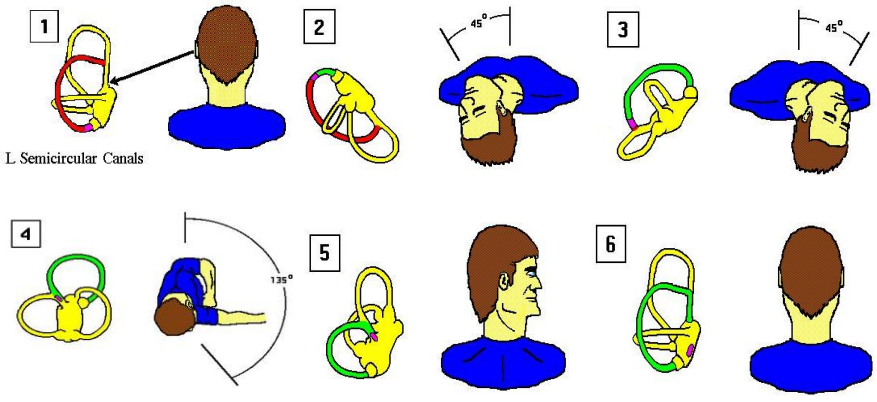
B. Vestibular Rehabilitation

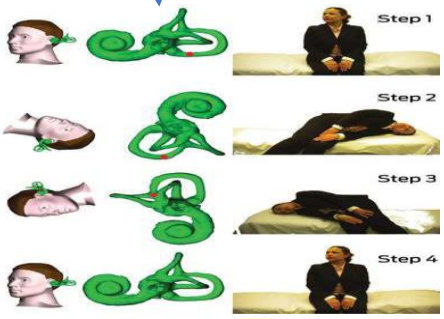
BPPV

- BPPV can trigger **migraine**
- 1st line treatment → particle repositioning maneuvers:
    - **Epley maneuver**
    - Modified Epley maneuver
    - Semont maneuver
    - Modified Semont maneuver
    - Brandt-Daroff exercises

Epley maneuver Contraindications

- Severe neck disease (Rheumatoid Arthritis, high-grade carotid stenosis) → **Semont maneuver**
- Acute neurological conditions **(TIA/Stroke)**
- Severe Orthopnea
- Pregnancy beyond 24th week of gestation





Nystagmus

Definition

Nystagmus is:

- Rhythmic, involuntary oscillation of the eyes
- Consists of:
  - Slow phase → pathological drift
  - **Fast phase** → corrective movement (defines direction)

Physiology (Why it occurs)

Nystagmus results from imbalance in the VOR (VOR discussed earlier):

- Normal: both labyrinths send equal signals → stable gaze
- Peripheral lesion (e.g., right side):
  - Brain perceives head movement toward normal side (left)
  - Eyes drift toward lesion (right)
  - Fast correction toward normal side (left) → left-beating nystagmus

→ Fast phase is away from affected ear (peripheral lesion)

Types of Nystagmus

A. Based on Direction (based on doctor's slides)

1. Purely horizontal
2. Mixed (horizontal + torsional)
3. Mixed (vertical + torsional)

B. Clinical Classification

1. Peripheral Vestibular Nystagmus

- Mixed horizontal–torsional or purely horizontal
- Unidirectional (same direction regardless of gaze)
- Fast phase away from lesion
- Suppressed by visual fixation
- Increased when looking toward fast phase

2. Central Nystagmus

- Mixed vertical–torsional
- May change direction with gaze
- Not suppressed by visual fixation
- Often associated with neurological signs

Behavior of Nystagmus

Alexander's Law

- Nystagmus increases when gaze is toward fast phase

Effect of Visual Fixation

- Peripheral → decreases with fixation
- Central → persists despite fixation
- **Frenzel lenses** used to remove fixation and enhance detection

Nystagmus in Acute Vertigo

- Spontaneous nystagmus is typical
- Indicates acute vestibular imbalance
- Direction helps localize lesion:
    - Fast phase → opposite side of lesion

Positional Nystagmus (Dix-Hallpike Test)

Peripheral (BPPV)

- Latency: 2–20 seconds
- Duration: <1 minute
- Fatigable
- Fixed direction (horizontal/rotatory)
- Severe vertigo

Central

- No latency
- Duration: >1 minute
- Non-fatigable
- Direction-changing
- Less severe vertigo

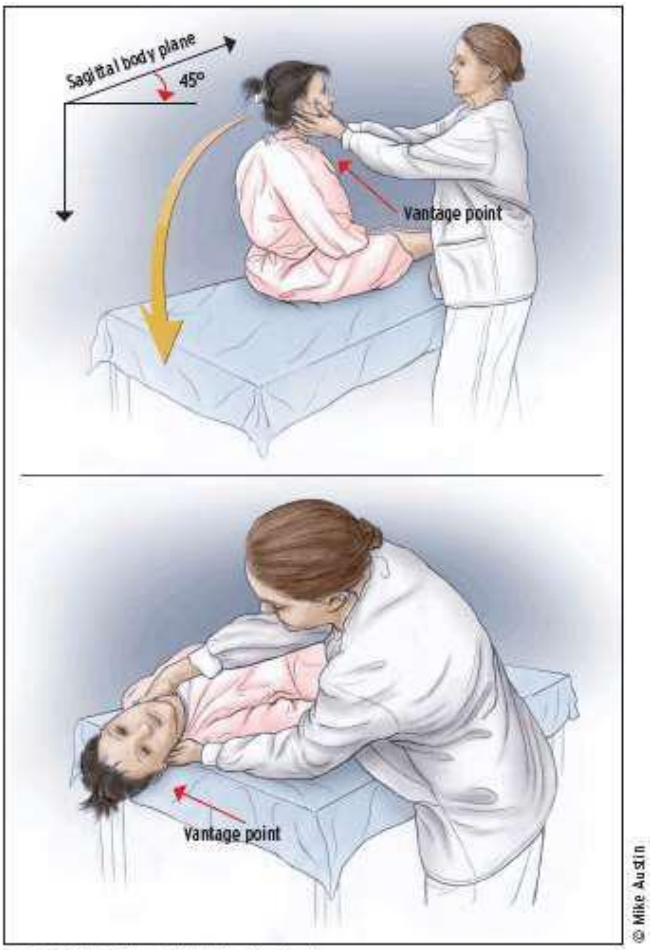


FIGURE 3. The Dix-Hallpike test

Epistaxis & Nasal Trauma

Introduction

Epistaxis (nosebleed) is a common clinical condition encountered across all age groups, particularly in children and elderly individuals. It is usually benign and self-limiting, though in some cases it can be severe or recurrent.

- Lifetime incidence: ~60%
- Only ~10% require medical attention
- More common in **males**

Nasal trauma frequently accompanies epistaxis and ranges from minor soft tissue injury to complex facial fractures.

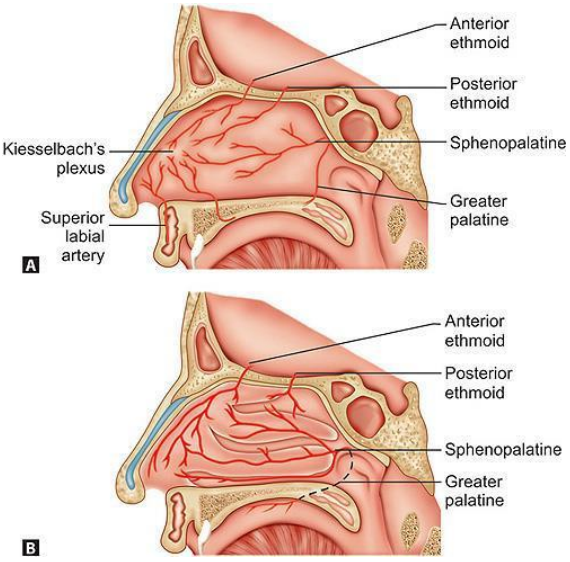
Epistaxis

Anatomy and Blood Supply

The nose has a rich vascular supply derived from both internal and external carotid systems.

Key arterial sources

- Internal carotid A. → **Ophthalmic A.**
  - Anterior ethmoidal artery
  - Posterior ethmoidal artery
- External carotid A.
  - **Maxillary A.** → Sphenopalatine artery → Greater palatine artery
  - **Facial A.** → Superior labial artery



Important vascular plexuses

- **Kiesselbach's plexus (Little's area)**
  - Located on anterior nasal septum
  - Most common site of bleeding
- **Woodruff's plexus**
  - Posterior nasal cavity
  - Associated with posterior epistaxis

Etiology of Epistaxis

A. Local causes

- Trauma (nose picking, injury)
- Anatomical abnormalities
- Inflammation (rhinitis, sinusitis)
- Excessive topical nasal sprays
- Tumors (rare)

B. Systemic causes

- **Hypertension**
- **Cardiovascular disease**
- Bleeding disorders
- Vascular malformations

Classification of Epistaxis

Feature	Anterior Epistaxis	Posterior Epistaxis
Frequency	90%	10%
Age group	Children (<10 yrs.)	Elderly (>50 yrs.)
Site	**Kiesselbach plexus**	**Woodruff plexus**
Presentation	Visible nasal bleeding	Blood in throat, may cause **hematemesis/melena**

Management of Epistaxis

A. Initial (Emergency) Management – ABC Approach

B. Conservative Measures

1. Pinch nose (compression) for 10–20 minutes
2. Head forward ("chin-to-chest")
3. Topical vasoconstrictors
4. Ice packs

C. If Bleeding Source Identified

- Chemical cautery (silver nitrate)
- Electrical cautery

D. If Bleeding Source Not Identified

- Anterior nasal packing
- Posterior nasal packing

E. Surgical Management

Indicated in persistent or severe cases:

1. **Arterial embolization**
2. **Arterial ligation**:
    - Sphenopalatine artery (most common)

Prevention

- Avoid nasal trauma and nose picking
- Maintain nasal moisture
- Use saline sprays or ointments
- Humidification

Nasal Trauma

Types of Nasal Fractures

Type 1

- Single nasal bone fracture
- Caused by anterior trauma

Type 2

- Nasal bone + frontal process of maxilla
- Caused by lateral trauma

Type 3

- Complex fractures (other skull bones)

Clinical Assessment

A. History

B. Physical Examination

- Urgent finding → **Septal hematoma**, **Septal fracture**, **CSF rhinorrhea**

C. Imaging

- Usually not required (**clinical diagnosis**)
- CT scan indicated if:
    1. Type 3 fracture
    2. CSF leak

Management of Nasal Trauma

Primary priorities

- ABC stabilization
- Control epistaxis
- Identify complications

A. Septal Hematoma

- Emergency condition
- Requires **urgent drainage**
- Prevents **septal necrosis** and **saddle nose deformity**

B. Nasal Bone Fracture Reduction

Timing

- **Within 1–2 hours** (before swelling) **OR** **after ~1 week** (once swelling subsides)
- Children: within 4 days
- Avoid after 14 days

Approach

- Local vs general anesthesia
- **Closed reduction** (most common) vs open reduction ± septoplasty

Acute Rhinosinusitis (ARS)

Definition

- Acute rhinosinusitis (ARS) is inflammation of the nasal cavity and paranasal sinuses lasting <12 weeks, with complete resolution of symptoms.
- The most common acute illness of human being
- More seen in **winter**

Rhinosinusitis Classification (by duration):

1. Acute rhinosinusitis (ARS) → < 12 weeks
2. Chronic rhinosinusitis (CRS) → > 12 weeks
3. Recurrent ARS → ≥ 4 attacks/year, with full resolution between attacks

Etiology

A. Viral (Most common ~98%)

- **Rhinovirus** (Most common microorganism)
- **Adenovirus**
  - ARS + **Conjunctivitis**
- RSV
- Influenza virus
- Parainfluenza

B. Bacterial (2%, mainly Gram +ve)

- **Streptococcus pneumoniae** (Most common bacteria)
- **Haemophilus influenzae** (2nd most common bacteria)
- **Moraxella catarrhalis** (3rd most common bacteria)
- Staphylococcus aureus (note: coagulase negative Staph are normal flora in 35% of patients)
- Streptococcus pyogenes

C. Fungal

- Aspergillus
- Candida

Classification

Feature	Viral ARS (Common cold)	Acute bacterial rhinosinusitis (ABRS)
First 3-4 days	First 3-4 days of AVRS cannot be distinguished from ABRS Purulent discharge in first 3-4 days of ARS does not mean ABRS	
Duration	<10 days	>10 days
Course	Improving	Persistent or worsening
Fever	Mild/absent	**High fever >39°C** (for at least 3-4 days)
Discharge	Mucoid	**Purulent** (for at least 3-4 days)
Pattern	-	"**Double worsening**" present
Notes	~ 95-98% are self-limited **< 2%** of colds in adults → ABRS **< 5%** of colds in children → ABRS	More common in **females**

Sinuses Most Commonly Affected in ARS

1) Maxillary sinus (most common)

- Most frequently involved sinus, due to:
  - Drainage against gravity
  - Close relation to teeth (predisposes to odontogenic spread)
    - **Anaerobic** & **Mixed** infections (10% of ABRS) → **Dental Origin**
  - Narrow ostium → easy obstruction

2) Ethmoid sinuses

- Common especially in children
- Located between the orbit and nasal cavity

3) Frontal sinus

- Less commonly involved
- **Develops later** → rare in young children

4) Sphenoid sinus (least common)

- Rare but clinically important
- Close to optic nerve and brain structures

Clinical Features (Conventional Criteria for The Diagnosis of Sinusitis) (مهم)

Major Symptoms:

- Nasal obstruction/congestion
- Purulent anterior nasal discharge
- Purulent/discolored posterior nasal discharge
- Facial pain/pressure (localized to affected sinus)
- Facial congestion/fullness
- Reduced smell
- **Fever**

Minor Symptoms:

- Headache
- Cough
- Ear pain/pressure/fullness
- Toothache (maxillary sinus)
- Halitosis
- Fatigue

Note:

- Fever is major symptom for ARS & minor symptom for CRS
- Clinical Diagnosis → 2 major symptoms

OR

1 major symptom + 2 minor symptoms

Diagnosis → Clinical Diagnosis based on the criteria

Approach

A. Hx & PEx

B. Sinus / Meatal Culture (in worsening despite Abx cases)

C. Endoscopy

D. Imaging

- Not routinely required
- CT only indicated if:
  - Complications suspected (sign & symptoms of complications, immunocompromised pts.)
  - Recurrent or atypical cases
  - Clinical deterioration while on medical therapy

Possible findings:

- Fluid level (CT)
- Frothy or strand secretions (Endoscopy)

Management

A. Viral ARS (Supportive Therapy alone)

B. Acute Bacterial Rhinosinusitis (ABRS)

1. Supportive Therapy:

- Analgesics
- Nasal saline irrigation
- Mucolytics
- Intranasal decongestants
- Intranasal corticosteroids

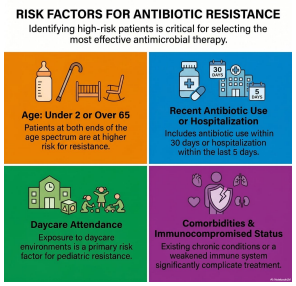
2. Empirical Antibiotics

- 1st Line → **Amoxicillin + Clavulanate**

Alternatives:

1. If beta lactam allergic:
   - Adults → Doxycycline
   - Pregnant women → Macrolides (Azithromycin)
   - Children → Levofloxacin

2. If symptoms worsen after 48-72 hrs. of initial empirical therapy **OR** fail to improve despite 3-5 days of initial empirical therapy → **2nd Line**:
    - Adults → **Levofloxacin**
    - Children → Cefixime or Cefpodoxime plus Clindamycin
3. If fail to improve despite 3-5 days of 2nd line empirical therapy:
    - Suspect complication
    - Refer to ENT specialist



Duration of Antibiotics:

- Adults + Uncomplicated ARS → 5-7 days
- Adults + Complicated/Resistant ARS → 10-14 days
- Children → 10-14 days

Avoid:

- **Avoid Antihistamines** → Anticholinergic S/E → thickens & dries nasal secretions

C. Surgical Treatment → treat complications of ARS

Complications

A. Orbital (MC group of complications, MC microorganism involved → Strep. viridian)

محاضرة عيون كاملة على الموضوع

- Preseptal cellulitis
- Orbital cellulitis
- Orbital abscess
- Subperiosteal abscess
- **Cavernous Sinus Thrombosis (CST)**:
    - MC source → **Ethmoid sinus**, MC microorganism → **Staph. aureus**
    - Infection spreads via valveless facial and ophthalmic veins → cavernous sinus → thrombosis → cranial nerve involvement
    - Within cavernous sinus: (slides says CN II is involved & CN V1, V2 are not?)
        - CN III
        - CN IV
        - CN V1, V2
        - CN VI (abducens)
    - Clinical Features:
        - **Cranial nerve VI palsy** = **earliest sign**
        - Severe headache
        - High fever
        - Periorbital edema and erythema
        - Proptosis
        - Ophthalmoplegia
        - ↓ Vision / papilledema (can occur, but later or if optic nerve gets secondarily affected)
        - **Rapid progression to bilateral (contralateral) signs** → strong clue for CST
    - Diagnosis:
        - **Clinical suspicion** + imaging
        - MRI with MR venography
    - Management (**Emergency**):
        - High-dose IV broad-spectrum **Antibiotics** immediately:
            - Vancomycin + Ceftriaxone ± Metronidazole
        - **Anticoagulants** (controversial but often used)
        - Treat source

B. Intracranial (MC source → Frontal sinus)

Symptoms Frequency: Headache (MC) → Fever → Altered mental status → Purulent rhinorrhea

- **Subdural abscess** (MC intracranial complication)
- Intracerebral abscess (Strep. milleri)
- **Epidural abscess** (Best prognosis)
- Meningitis (usually due to septic thrombophlebitis, more common in **infants**)
- Sinuses thrombosis

C. Local

- Osteomyelitis (e.g., **Pott's puffy tumor**)

Chronic Rhinosinusitis (CRS)

Definition

- Chronic Rhinosinusitis (CRS) is a persistent inflammatory condition of the nose and paranasal sinuses **lasting ≥12 weeks**, characterized by a combination of symptoms and **objective evidence** of mucosal inflammation.
- CRS is a proliferative disease with thickening of mucosa and lamina propria

Epidemiology

- Prevalence: ~10–15% worldwide.
- More common in adults
- **Females** are more affected
- Up to 20% of CRS patients have **Asthma**

Classification (Based on Phenotype)

1. CRS without Nasal Polyps (CRSsNP)
2. CRS with Nasal Polyps (CRSwNP)

Feature	CRS without Nasal Polyps (CRSsNP)	CRS with Nasal Polyps (CRSwNP)
Prevalence	More common (~60–65%)	Less common (~20–33%)
Sex	Slightly **females**	**Males** (2:1)
Dominant cells	**Neutrophils**	**Eosinophils**
Cytokines	IFN-γ, IL-17	IL-4, IL-5, IL-13
Remodeling	Fibrosis	Edema, pseudocysts
Smell	Mild ↓	Marked **anosmia**
Steroid response	Moderate	Good
Recurrence	Lower	Higher

Risk Factors

A. CRSsNP

a. Host Factors

- Genetic predisposition
- Allergy (**Allergic Rhinitis**)
- Immunodeficiency (IgA deficiency, CVID)
- Anatomical defects/variations (**septal deviation**, scarring from previous surgery)
- Trauma
- Foreign bodies
- **Neoplasm → CT scan when suspected**
- GERD

b. Environmental triggers

- Allergens
- Air pollution
- Smoking
- Cilio-static substances
- Medications (**Rhinitis Medicamentosa**)

c. Microbial Factors

- Bacterial biofilms
- Fungal colonization
- Persistent infections

B. CRSwNP

- Strongly associated with:
  - **Asthma**
  - **Aspirin Intolerance**
- Other associations:
  - Eosinophilic mucin rhinosinusitis (EMRS)
  - Allergic fungal rhinosinusitis (AFRS)
  - Cystic fibrosis (**diffuse nasal polyps**)
  - Primary ciliary dyskinesia (e.g., Kartagener syndrome) → can be CRSwNP or CRSsNP

Pathophysiology

Disease Progression Sequence:

- Ostiomeatal complex obstruction → ABRS → fail to resolve → chronic inflammation

Key Processes that involved in pathogenesis:

1. Epithelial barrier dysfunction → impaired mucociliary clearance
2. Chronic inflammatory cell infiltration
3. **Biofilm formation**
4. Tissue remodeling

Bacteriology in CRS:

- Unlike ARS, CRS microbiology is polymicrobial and complex

Most commonly involved:

1. **Coagulase -ve Staphylococcus**
2. **Staphylococcus aureus**
3. Haemophilus influenzae
4. Streptococcus pneumoniae
5. Moraxella catarrhalis
6. Pseudomonas aeruginosa

Role of bacteria in CRS:

- Induction of osteitis
- Biofilm formation
- Superantigen production
  - Staph. **exotoxins** → massive T-cell activation → cytokine storm → promotes nasal polyps

---

CRS initially is a Clinical Diagnosis (no need for imaging & labs)

Approach

A. Hx & PEx

B. Endoscopy

C. Imaging

- **CT scan** (gold standard, limited role in initial assessment)
  - Indications:
    - Failure of maximum medical therapy
    - Before surgical procedures
    - Suspected complications
    - Suspected neoplasm
- Plain Films (less role in CRS than ARS)
- MRI
  - Indications:
    - Neoplasm detected by CT
    - Skull base or orbital complications detected by CT
    - Fungal sinusitis

D. Laboratory Tests

- Indications (based on Hx & PEx):
  - Nasal swab & culture
  - RAST or skin prick test → Allergy suspected
  - Sweat chloride test → CF suspected

Diagnostic Criteria (Discussed in ARS Lecture)

1. **Presence of ≥2 of major symptoms**

   Plus

2. **Objective evidence**:

   A. **Nasal Endoscopy** (polyps, mucopurulent discharge, edema)

   OR

   B. **CT changes in sinuses** (mucosal thickening, sinus opacification, ostiomeatal complex obstruction)

Complications of CRS

- Mucocele
- Pyocele (infected mucocele)

Management

A. Allergen + Irritant Avoidance (most important)

B. Medical Treatment (First-line, very effective)

1. Decongestants

- For **Symptomatic relief**
- No therapeutic role in treatment of sinusitis or polyps

2. Saline Irrigation (Hypertonic saline)

- Improves mucociliary clearance

3. Intranasal Corticosteroids

- Mainstay treatment for **CRSwNP & Allergic Rhinitis**

4. Systemic Corticosteroids

- Short courses in **CRSwNP**
- Used also in ARS, Prophylaxis of recurrent ARS, CRSsNP

5. Antihistamines

- Useful in **Allergic Rhinitis**

6. Leukotriene Modifiers

- Especially useful in **Aspirin Intolerance**

7. Antibiotics

- Routine use of antibiotics is NOT recommended in CRS
- Most CRS exacerbations are viral or inflammatory
- Indication → suspected **Bacterial CRS Exacerbation**

8. Long-term low-dose Clarithromycin

- Improve sinus symptoms in **CRSsNP**

Mechanism of Action

A. Anti-inflammatory Effects

- ↓ Cytokine production
- ↓ Neutrophil activity
- ↓ mucus secretion

B. Immunomodulatory Effects

- Regulate immune response
- Improve mucociliary clearance

C. Anti-biofilm Action

- Disrupt bacterial biofilms
- Reduce bacterial virulence

C. Biologic Therapy (for Refractory CRSwNP)

D. Surgical Management → Functional Endoscopic Sinus Surgery (FESS)

- **Stammberger Principle** about Extent of Surgery: "Surgery stops there, where pathology stops"

Steroid-Specific Safety Data in Chronic Rhinosinusitis

Medical corticosteroid treatment effectively manages CRS, but clinicians must balance therapeutic benefits with specific safety profiles of systemic and topical applications.

Systemic Corticosteroids

Clinical Contraindications

- DM, PUD
- Severe HTN
- Glaucoma
- Advanced Osteoporosis

Major Side Effects

- Osteoporosis
- Pediatric growth retardation
- Cataracts
- Glaucoma
- H.P.A. axis suppression

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Topical (Local) Corticosteroids

Primary Adverse Effects

- Nasal irritation
- Epistaxis (bleeding)
- Crusting
- Nasal septal perforation

Potential Systemic Risks

- Growth monitoring
- Bone density
- H-P-A axis

NotebookLM

Patient Counseling: Proper Use of Nasal Sprays

Proper administration is essential for managing Chronic Rhinosinusitis, ensuring medication reaches the nasal mucosa.

PREPARATION AND APPLICATION TECHNIQUE

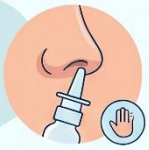
Device Priming and Regular Dosing

Always prime the spray device before use and avoid skipping scheduled doses for consistency.

Correct Head Positioning

Keep the head tilted forward during application; do not tilt the head back.

Incorrect: Tilting Head Back



Gentle Nozzle Placement

Avoid pushing the nozzle too hard or too far into the nasal passage.

POST-APPLICATION CARE AND ROUTINE

Gentle Aftercare Habits

Avoid sniffing hard or blowing your nose immediately after spraying to keep medicine inside.

Follow the Correct Sequence

Always use saline sprays or irrigations before applying corticosteroid sprays, never after.

Medication Destination Matters

Proper technique prevents medicine from depositing in the throat instead of the nose.

NotebookLM

Adeno-tonsillar Diseases

Sore Throat

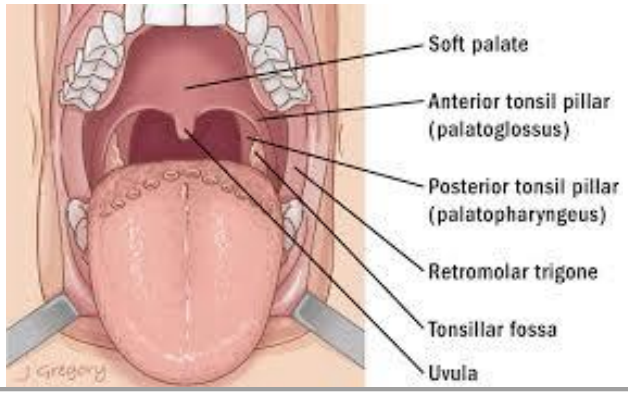
- Painful sensation in the pharynx or surrounding structures
- Can be acute or chronic

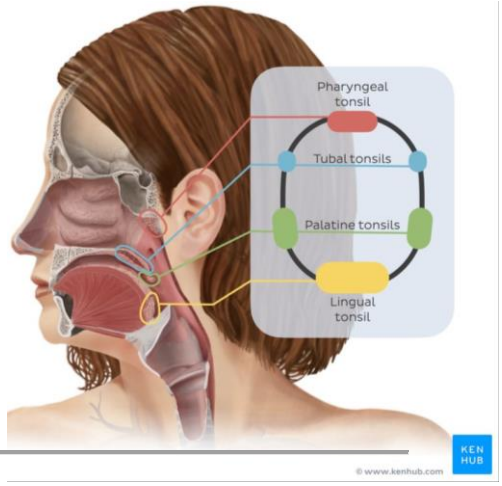
Common causes:

- Pharyngitis
- Tonsillitis
- Adenoiditis
- Laryngitis

Anatomy (Waldeyer's Ring)

- Lymphoid tissue ring at entrance of respiratory & alimentary tracts





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1. Pharyngitis

Etiology:

- Most common cause of sore throat
- **Viral** (most common)
  - **Respiratory Viruses** (rhinovirus, influenza, adenovirus, coronavirus, parainfluenza)
  - **EBV**
  - In infants & young children → **Herpangina (Enterovirus)**
- Bacterial: **Streptococcus** (most common bacterial pharyngitis)

Clinical features:

- Sore throat
- Dysphagia
- Fever, fatigue
- Otalgia (via tympanic branch of CN IX)
- Poor appetite
- Dehydration

---

2. Tonsillitis

Epidemiology:

- Common in children (peak 3–7 years)
- Seen in adults

Etiology:

- **Viral** (most common)
- Bacterial:
  - β-hemolytic streptococcus
  - Staphylococcus
  - H. influenzae
  - Pneumococcus

Feature Comparison:

Feature	Viral Tonsillitis	Bacterial Tonsillitis (GAS)
Onset	Gradual	Sudden
Fever	Low-grade or absent	High fever
Sore throat	Mild–moderate	Severe
Cough	Common	Absent
Rhinorrhea	Common	Absent
Tonsillar exudate	Rare	Common
Lymph nodes	Mild enlargement	Tender anterior cervical lymphadenopathy
Systemic symptoms	Myalgia, fatigue	Headache, nausea, abdominal pain (esp. children)

Clinical features:

- Sore throat
- Dysphagia
- Fever, fatigue
- Otalgia (via tympanic branch of CN IX)
- Poor appetite
- Dehydration

Types of Acute Tonsillitis:

- Parenchymatous
- Follicular
- Membranous

A. Membranous Tonsillitis

1. Infectious Mononucleosis (EBV)

- Most commonly in → Teenagers / Young adults
- Misdiagnosed as streptococcal tonsillitis → No improvement with antibiotics → suggests EBV
- **More systemic manifestations** than simple tonsillitis:
  - Splenomegaly (50%)
  - Hepatomegaly & jaundice (10%)
  - Enlarged jugulodigastric lymph nodes

Laboratory Findings

- Serology:
  - Heterophile antibodies
- Tests:
  - Monospot test
  - Paul–Bunnell test
- Blood picture:
  - Lymphocytosis
  - Atypical lymphocytes / monocytes

Management:

- Avoid ampicillin (rash)
- **Supportive treatment**
- Steroids (oral/systemic) → only in cases of airway obstruction

2. Scarlet Fever

- **Streptococcus pyogenes**
- Features: **strawberry tongue** + **rash**

3. Diphtheria

- Very rare due to vaccination
- Thick gray membrane covering throat & tonsils
- Airway obstruction risk

4. Vincent Angina

- Fusiform + spirochete infection

Complications of Tonsillitis

- **Peritonsillar abscess (Quinsy)** – most common
- Airway obstruction
- Otitis media
- Parapharyngeal abscess
- Retropharyngeal abscess
- For **Scarlet Fever:**
  - Rheumatic fever
  - Glomerulonephritis

A. Peritonsillar Abscess (Quinsy)

Definition:

- Accumulation of pus between the tonsillar capsule & the lateral pharyngeal wall

Features:

- Unilateral swelling
- Tonsil pushed medially
- Very high-grade **fever**
- **Enlarged Jugulo-diagastric LNs**
- **Trismus** (inability to open mouth completely)
- Drooling of saliva
- Airway obstruction
- "**Hot potato voice**"
  - A muffled, thick, "as if speaking with a hot potato in the mouth" voice
  - Speech sounds unclear and indistinct

Management:

- **Admission**
- **IV antibiotics** (1-2 days) → Oral antibiotics (10 days)
- **Incision & drainage** (dramatic improvement immediately)
- Tonsillectomy after recurrent episodes (**after 6 wks. of 2nd attack**)

Other causes of Quinsy:

- Dental Infections
- Foreign body

B. Parapharyngeal Abscess

- Diagnosed involve combination of H&P & **CT scan**

Treatment:

- Admission + Abx + I&D

Complications:

- IJV thrombosis
- Carotid rupture
- Cranial nerve injury (IX–XII)
- Mediastinitis, sepsis

C. Retropharyngeal Abscess

- Potential airway compromise → Requires urgent management

3. Adenoid Disorders

Growth:

- Enlarges until age ~6 → Atrophies → disappears by age ~16

Adenoidal Disease:

1. **Adenoid Hypertrophy** (airway obstruction + Eustachian tube blockage → ear problems)
2. **Adenoiditis**

Symptoms:

- Nasal obstruction
- Mouth breathing
- Snoring & OSA
- Rhinorrhea
- Voice changes
- Sore throat
- **Adenoid Face** (open mouth, protruded frontal teeth)

Management:

Adenoid Hypertrophy:

- **Nasal sprays**- (Saline / Steroids / Decongestant)
- **Antibiotics** (if infected)
- **Adenoidectomy**
  - Indications:
    - Nasal obstruction
    - OSA
    - OME
    - Recurrent AOM
    - CRS

Management of Sore Throat

A. Supportive:

- Analgesics
- Antipyretics
- Hydration
- Local antiseptics

B. Antibiotics (if bacterial, for 10 days):

- **Amoxicillin-clavulanate**
- **Cephalosporins**

C. Severe cases:

- Admission
- IV antibiotics
- Culture + EBV testing
- Drain abscess if present

D. Tonsillectomy

Indications:

- Recurrent tonsillitis (**Paradise criteria: 7 in 1 year / 10 in 2 years / 9 in 3 years**)
- OSA
- Febrile convulsions
- **After 6 wks. of 2nd Quinsy**
- Dysphagia/failure to thrive
- Suspected malignancy

Complications:

- Bleeding (primary/secondary)
- Infection
- Injury to nearby structures
- Tonsillar remnants

Stridor

Overview

Stridor is a noisy breathing sound caused by turbulent airflow through a **partially obstructed airway**.
It is not a disease, but rather a clinical sign or symptom indicating airway compromise.

Classification (based on its timing in the respiratory cycle)

- Inspiratory → Supraglottic obstruction
- Expiratory → Lower airway obstruction
- Biphasic → Glottic obstruction / Subglottic obstruction

Pathophysiology

Stridor results from airflow through a narrowed airway. In children, due to **airway flexibility**, the **Venturi principle** causes reduced lateral pressure during airflow, leading to airway collapse and turbulent flow, producing the characteristic sound.

Clinical Approach

A. History

B. Physical Examination

- Assess respiratory distress (urgent priority)
- Look for cyanosis, retractions, tachypnea
- Perform **laryngoscopy** for definitive evaluation (note: laryngoscopy is a part of PEx)

Stridor Causes:

A. Congenital Causes

1. Laryngomalacia

- **Most common** cause of inspiratory stridor in infants (**~75%** of all cases of stridor)
- Due to **delayed cartilage development** → soft laryngeal structures

Characterized by:

- **Omega-shaped epiglottis**
- Collapse of supraglottic structures during inspiration

Clinical Features

- Stridor worsens in supine position and with head flexion
- Improves in prone position and neck extension

Diagnosis:

- laryngoscopy

Management

- Usually **self-limiting** (resolves by 1–2 years)
- In severe cases:
  - Tracheostomy
  - Supraglottoplasty (removal of redundant tissue or release of mucosal bands)

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2. Vocal Cord Paralysis

- **Second most common** cause in infants
- May be unilateral or bilateral

Features (Unilateral VCP)

- Weak cry
- Biphasic stridor
- Worse when awake, improves when lying on affected side
- Often associated with **CNS abnormalities**

3. Subglottic Stenosis

- Narrowing below vocal cords (narrowest airway region in children)
- Can be congenital or acquired

Causes (Acquired)

1. Idiopathic
2. Autoimmune (e.g., Granulomatosis with polyangiitis GPA)
3. Trauma
4. Prolonged intubation
5. GERD

Clinical Presentation

- Inspiratory or biphasic stridor
- May present anytime in early childhood

Management

A. Medical (early/immature cases):

- Oral/inhaled **Steroids**
- Antibiotics? (Studies on animals)
- **Aggressively treat GERD**

B. Surgical (mature cases):

- Grade 1–2: Endoscopic dilation (laser, balloon, stents)
- Grade 3–4:
  - Tracheostomy
  - Cricotracheal resection (definitive surgery, done at age of 4)

---

4. Laryngeal Web

- Due to incomplete embryologic recanalization
- 75% occur at the glottis

Features

- Weak cry
- Biphasic stridor

---

5. Laryngeal Cysts

- Usually supraglottic (epiglottic folds)

Presentation

- Stridor
- Hoarseness or aphonia
- Airway obstruction if large

---

6. Other Congenital Causes

- Hemangiomas, lymphangiomas
- Vascular anomalies (e.g., double aortic arch)

B. Acquired Causes

I. Inflammatory Causes

1. Acute Epiglottitis

A life-threatening emergency

Symptoms

- Severe sore throat (95%)
- Dysphagia/Odynophagia (95%)
- Drooling of saliva
- "Hot potato" muffled voice
- High fever (>40°C)
- Toxic appearance
- Sitting in air hunger position with severe stridor

Diagnosis

- Diagnostic Shift → **laryngoscopy** (preferred now)
- Lateral Neck X-ray → **Thumb sign** (epiglottic swelling)

Management

- Airway first priority
- IV fluids
- IV antibiotics (ampicillin, chloramphenicol)
- IV steroids
- Nebulized epinephrine

---

2. Acute Laryngotracheobronchitis (Croup)

- Most common cause of acute stridor in children (6 months–2 years)
- Viral (**parainfluenza**)

Symptoms

- **Barking cough**
- Stridor
- Wheeze, crepitations
- Low-grade fever
- Child appears ill but not toxic

Radiology

- **Steeple sign** on X-ray

Management

- Supportive care (**self-limiting**)
- Secure airway if needed
- IV Steroids
- Nebulized epinephrine
- Humidified air

---

3. Acute Laryngitis

- Concurrent viral URTI
- Inflammation at vocal cords
- Seen in children less than 12 years old

Features

- Stridor
- Hoarseness
- Cough

Management

- Supportive care (**self-limiting**)
- Secure airway if needed
- IV Steroids
- Nebulized epinephrine

---

4. Other Inflammatory Causes

- Foreign body inhalation
- Bacterial tracheitis (often after viral infection)
- Peritonsillar abscess
- Spasmodic croup
- Chronic inflammation (e.g., TB)

II. Tumors Causes

- Tumors of the larynx or pharynx causing airway obstruction and stridor

III. Trauma Causes (not common)

Classification

According to Mechanism

- Thermal (burns, hot fumes)
- Chemical (aspiration)
- Physical (blunt or penetrating)

According to Severity

- Mild
- Moderate
- Severe

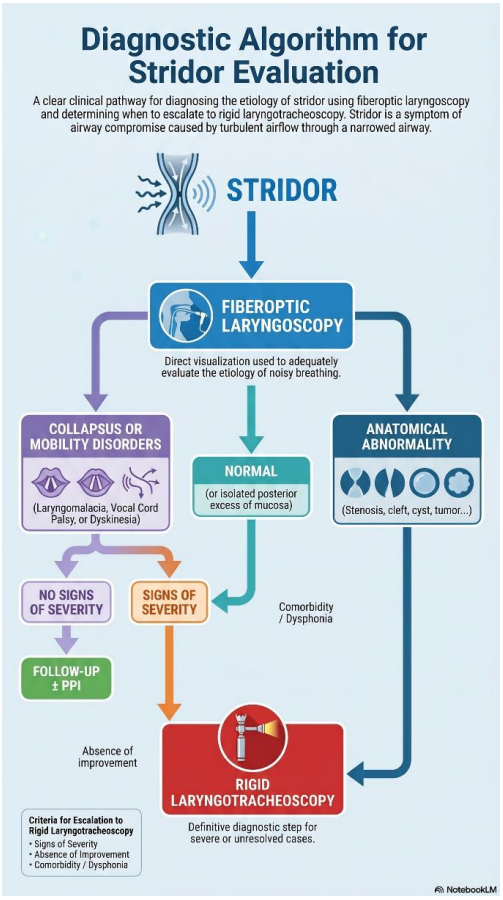
According to Cause (accidental, personal assaults, RTA …)

Management

- Rule out cervical spine injuries before attempting intubation or tracheostomy
- Airway stabilization (often tracheostomy)
- Imaging (CT scan) → detects fracture
- An intact larynx should NOT be opened for exploration; exploration is reserved for obvious penetrating injuries
- Treat fractures and associated injuries
- For cut wounds: Do NOT suture the wound directly → Place a stent
  (to maintain the lumen and prevent future stenosis)

Surgical Airway Options:

Feature	Tracheostomy	Cricothyroidotomy
Timing	Elective	**Emergency**
Site	2nd–3rd tracheal ring	Cricothyroid membrane
Duration	Long-term	Short-term
Technique	Open/percutaneous	Open/needle
Setting	OR/ICU	**ER**/ICU
Equipment	Extensive	Minimal



Neck Masses

Introduction

A neck mass is a common clinical presentation across all age groups.

- **Prevalence:** 10–15%, increasing with age
- **In Adults → Rule of 80:**
  - 80% of non-thyroid neck masses are neoplastic
  - 80% of these are malignant
- **In Children** → ~90% are benign

Differential Diagnosis of Neck Masses

A. Congenital/ Developmental Causes

1. Thyroglossal Duct Cyst

General Info:

- Most common **midline neck** mass

Features:

- Moves with swallowing & tongue protrusion
- **Related to hyoid bone**
  - **24% Suprahyoid**
  - **61% Thyrohyoid**
  - 15% for:
    - Suprasternal
    - Lingual
    - Mediastinal
- May contain the patient's only thyroid tissue → **Pre-operative Assessment**
  - **Neck U/S** / **Nuclear Thyroid Scanning** → to avoid Permanent Hypothyroidism
- Carries risk for **papillary carcinoma** → **Surgery**
- Treatment: **Sistrunk operation**

2. Branchial Cleft Anomalies

Types:

A. Branchial Cleft **Cysts**
B. Branchial Cleft **Sinuses**
C. Branchial Cleft **Fistulas**

General Info:

- Most common **lateral neck** congenital masses
- Location → along the **anterior border of sternocleidomastoid muscle** → **CT scan**
- Treatment → **Surgery** (FNA & Biopsy before treatment)

Embryology: (اقرأهم)

	Bone/Cartilage	Nerve	Artery
I	Incus, malleus, mandible	V2, V3	Maxillary Ext carotid
II	Stapes, styloid, hyoid (lesser cornu/upper body)	VII	Stapedial Hyoid
III	Hyoid (greater cornu/ inferior body	IX	Common carotid Internal carotid
IV	Thyroid & epiglottic cartilages	Vagus Superior laryngeal	Right: aortic arch, subclavian Left: aortic arch
VI	Cricoid cartilage, Arytenoid cartilages, Corniculate cartilage	Vagus Recurrent laryngeal	Right: Pulmonary Left: Pulmonary, ductus arteriosus

Table 1: Derivatives of arches

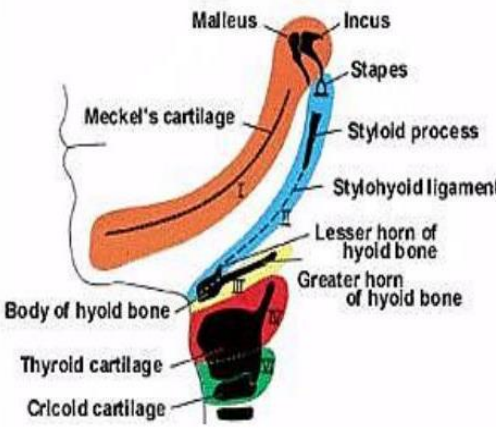


Figure 5: Derivatives of branchial arches

Key relations:

Cleft	Key relations
1st	Located in proximity to the **facial N.** and often associated with the **parotid gland**
2nd	Situated between the internal and external **carotid arteries**
3rd	Lies posterior to the carotid arteries and is related to the **glossopharyngeal N.**
4th	Closely associated with the **recurrent laryngeal N.** and the **thyroid gland**

A. Branchial Cleft Cysts

Features:

- Soft, painless, slow-growing **external swelling**
- **Mass effect**
  - Dysphagia
  - Airway obstruction
- Get infected → **Abscess**

Important Notes: (**مهم جدا**)

- 2nd branchial cleft cyst → rule out **cystic metastasis** from oropharyngeal cancer → ***Biopsy (not FNA)***
- 3rd/4th branchial cleft cyst → may be misdiagnosed as acute suppurative thyroiditis

B. Branchial Cleft Sinuses / Branchial Cleft Fistula

Features:

- Persistent **mucus discharge** from a small opening in the neck
- Recurrent ear/throat/thyroid infection

Other:

3. Dermoid cyst
4. Cystic hygroma (lymphangioma)
5. Vascular Malformation
6. Hemangioma
7. Thymic rests

B. Inflammatory Causes

Most important causes:

1. Reactive lymphadenopathy (most common cause in children <5 years of age)
2. Lymphadenitis
   - **TB**
   - **Cat scratch disease** (Bartonella henselae)
   - Infectious mononucleosis
   - CMV infection
   - Toxoplasmosis
   - HIV infection
3. **Kawasaki disease** (children) → **Neck mass** + **Strawberry tongue** + **Bilateral conjunctivitis**

C. Neoplastic Causes

Most important causes:

1. **Benign:**
   A. Lipoma
   B. Fibroma
   C. Neurofibroma
2. **Malignant:**
   A. Lymphoma
   B. Metastatic squamous cell carcinoma
   C. Thyroid carcinoma
   D. Salivary gland tumors

Special Entities

A. Carotid Body Tumor (Paraganglioma)

Also called:

- Glomus tumor
- Chemodectoma

Key facts:

- **Neuroendocrinal tumors** that originate from glomus cells in paraganglia, these cells are part of SNS and serves as chemoreceptors
- Derived from **neural crest cells**
- **90% Sporadic**
- **10% Hereditary → mutation** in Succinate Dehydrogenase **(SDH) genes**
    - **Features:**
        - **<40 years of age**
        - **Multifocal Paraganglioma + Phaeochromocytoma → chromaffin positive**

Location:

- **Carotid artery bifurcation**

Clinical important notes:

- Typically presents as:
    - Slow-growing lateral neck mass (10% bilateral)
    - May be pulsatile
    - 1–3% secretes Catecholamine (Pheochromocytoma-like symptoms)
        - Palpitations
        - Hypertension
        - Headaches
        - Sweating
        - Complications if left untreated
    - Family Hx is important (**MEN 2A/2B**, **SDH mutations**)
- **Highly vascular** → important for surgery
- Classic imaging sign → **Splaying of carotid vessels (Lyre sign)**
- Important to note that paraganglion may arise in the **Vagus N.** → **Vagal Paraganglion**
    - Vagus N. involvement symptoms
        - Hoarseness
        - Dysphagia
    - On imaging → displace both (ECA & ICA) anteromedially = No Lyre sign

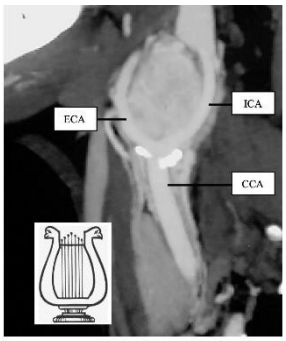


Figure 8a: Splayed carotid bifurcation (Lyre sign)

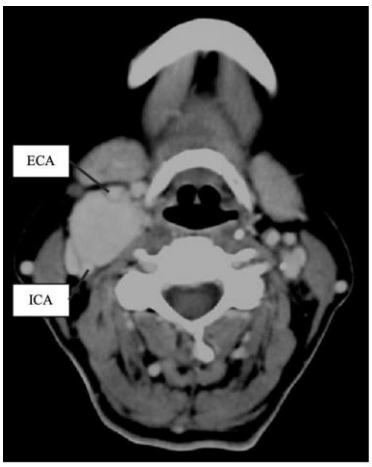
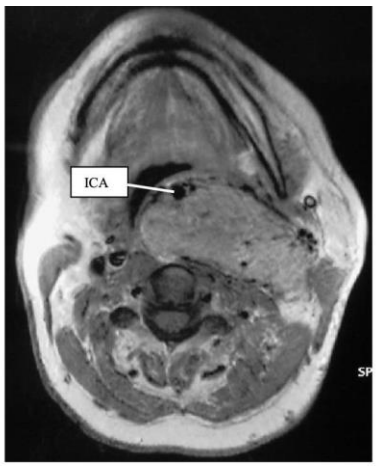


Figure 8b: Carotid body tumours splay the internal and external carotid arteries



Figures 9b: Vagal paraganglioma typically displaces the internal carotid artery anteriorly

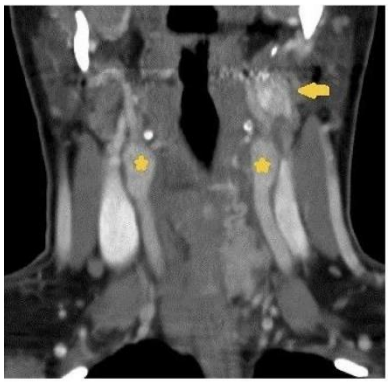


Figure 7: Bilateral carotid body tumours (*) and left vagal paraganglioma (arrow) in a patient with a SDH-D mutation. Following this diagnosis the patient's brother was also diagnosed with an SDH-D mutation and multiple paragangliomas

B. Juvenile Nasopharyngeal Angiofibroma (JNA)

Key facts:

- Rare tumor (~0.05%)
- Occurs almost exclusively in **adolescent males**
- Origin: **highly vascular tumor**
- JNA originates from **Sphenopalatine artery**
  - Sphenopalatine A. is the terminal branch of Maxillary A.

Classic presentation:

- Recurrent severe epistaxis & nasal obstruction
- The epistaxis may even require blood transfusion

Other symptoms:

- Headache
- Facial swelling
- Unilateral rhinorrhea & hyposmia
- Ipsilateral conductive hearing loss (**eustachian tube dysfunction**)

Imaging findings:

- **Mass in nasopharynx** (imaging & examination)
- **Widening of sphenopalatine foramen**
- **Holman-Miller sign**:
  - Anterior bowing of posterior maxillary wall

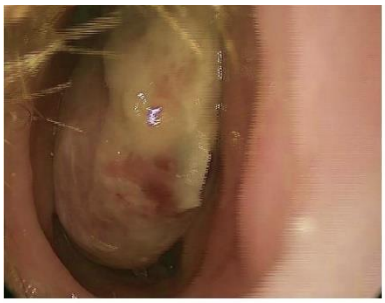


Figure 11: View of right nasal cavity showing large, vascular mass

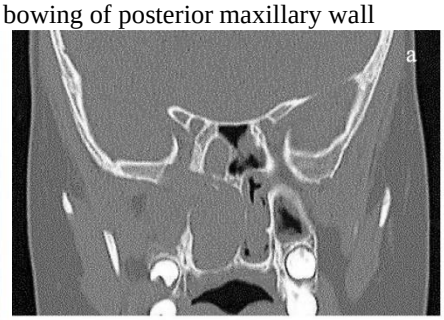


Figure 12a: CT scan: Widening of the sphenopalatine foramen and nasal cavity

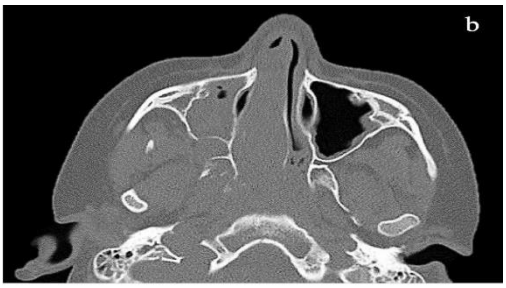


Figure 12b: CT scan: Anterior bowing of posterior wall of maxillary sinus (Holman-Miller sign) and nasal mass

Management:

- **DO NOT BIOPSY** (high bleeding risk)
- **Pre-op embolization** (reduces bleeding)
- Surgical resection

C. Salivary Gland Masses

Anatomy

Parotid Gland

Key points:

- Largest salivary gland
- Located anterior to the ear
- Divided into **superficial and deep lobes**

Submandibular Gland

Key points:

- Located beneath the mandible
- Wraps around the **mylohyoid muscle**
- Closely related to:
  - **Lingual nerve**
  - **Submandibular duct**
  - **Hypoglossal nerve (CN XII)**

Facial Nerve (critical in surgery)

Key points:

- Runs through parotid gland
- Divides into branches (pes anserinus)

Clinical importance:

- Facial nerve palsy → malignancy until proven otherwise
- Must be preserved in surgery

Distribution & Malignancy Risk (Rule: Smaller gland → Higher malignancy risk)

Gland	Frequency	Malignancy risk
Parotid	**80%**	20%
Submandibular	15%	50%
Sublingual/minor	5%	**70%**

Common Tumors

A. Benign:

1. **Pleomorphic Adenoma** (most common overall, 90% of benign)
2. Warthin's tumor

B. Malignant:

1. **Mucoepidermoid** (most common malignant)
2. Adenocarcinoma
   A. Adenocystic Carcinoma (Cylindroma)
   B. Acinic cell Carcinoma
3. Malignant Mixed
4. Lymphoma
5. Metastasis

There is broad DDx (so many in slides)

Red Flags for Malignancy:

- Rapid growth
- Fixation
- Facial nerve palsy
- **Pain**
  - DDx
    - **Sialadenitis** (acute pain + fever)
    - **Sialolithiasis** (acute recurrent pain)
    - **Benign tumor** (**painless**, slowly growing mass)

Investigations

Typical diagnostic approach:

- **Imaging**
  - CT / MRI (tumor extent, nerve relation)
- **FNA**
- **Ultrasound** (stones, cysts)

Management

- **Surgery** (with facial nerve preservation) ± **Radiotherapy**

Clinical Evaluation for Neck Masses

A. History

Key elements include:

- **Age** (most important factor)
  - Malignancy risk increases with age
- **Duration**
  - Acute → Infection
  - Chronic → Tumor
- **Symptoms**
  - Fever, sore throat, tenderness, erythema → Infection
  - Dysphagia, hoarseness, weight loss, night sweats → Malignancy
  - Recent URTI → Reactive lymphadenopathy
- **Risk factors**
  - Smoking, alcohol, radiation exposure → Malignancy
- **Exposure history**
  - Travel, animals, infections → Inflammatory causes
- **Timing**
  - Present at birth → Congenital
  - Rapid growth → Inflammatory
  - Slow growth → Benign Tumor
  - Persistent >6 weeks → Malignancy → **Evaluation & Specialist referral**

B. Physical Examination

Includes:

- **Size**
  - Size > 2 cm → Malignancy
- **Character**
  - Hard, immobile mass → Malignancy
  - Tender, soft mass → Infection
  - **"Shotty" lymph nodes** → Reactive Lymphadenopathy
    - Multiple small LNs that feel like buckshot under the skin
- **Scope**
  - Inspection of skin and mucosa
  - Palpation of:
    - Tongue base
    - Tonsillar area
    - Lymph nodes
  - **Laryngoscopy** for deeper structures
- **Location**
  - High Risk: Masses in → **Supraclavicular area** / **Posterior Triangle**

Location	Common Developmental/Inflammatory	Malignant/High Concern
Midline	**Thyroglossal duct cyst** **Dermoid cyst**	**Thyroid Cancer**
Lateral Neck (SCM)	**Reactive lymphadenopathy** **(2nd – 4th) Branchial cleft cyst** **Lymphadenitis**	**Lymphoma**
Supraclavicular	Vascular malformation	Lymphoma Metastatic lesion
Submandibular	Reactive lymphadenopathy Sialadenitis	Salivary gland tumor
Preauricular	Sialadenitis Hemangioma **1st Branchial cleft cyst**	Salivary gland tumor
Submental	Reactive lymphadenopathy Thyroglossal duct cyst	—
Occipital	Reactive lymphadenopathy	Metastatic lesion

C. Investigations

- **Ultrasound** → Initial imaging modality (especially for children)
- **FNA** → **Most important initial diagnostic test** (any unexplained mass by H&P)
- **Biopsy** → **Definitive Diagnosis** (if persistent >4–6 weeks)

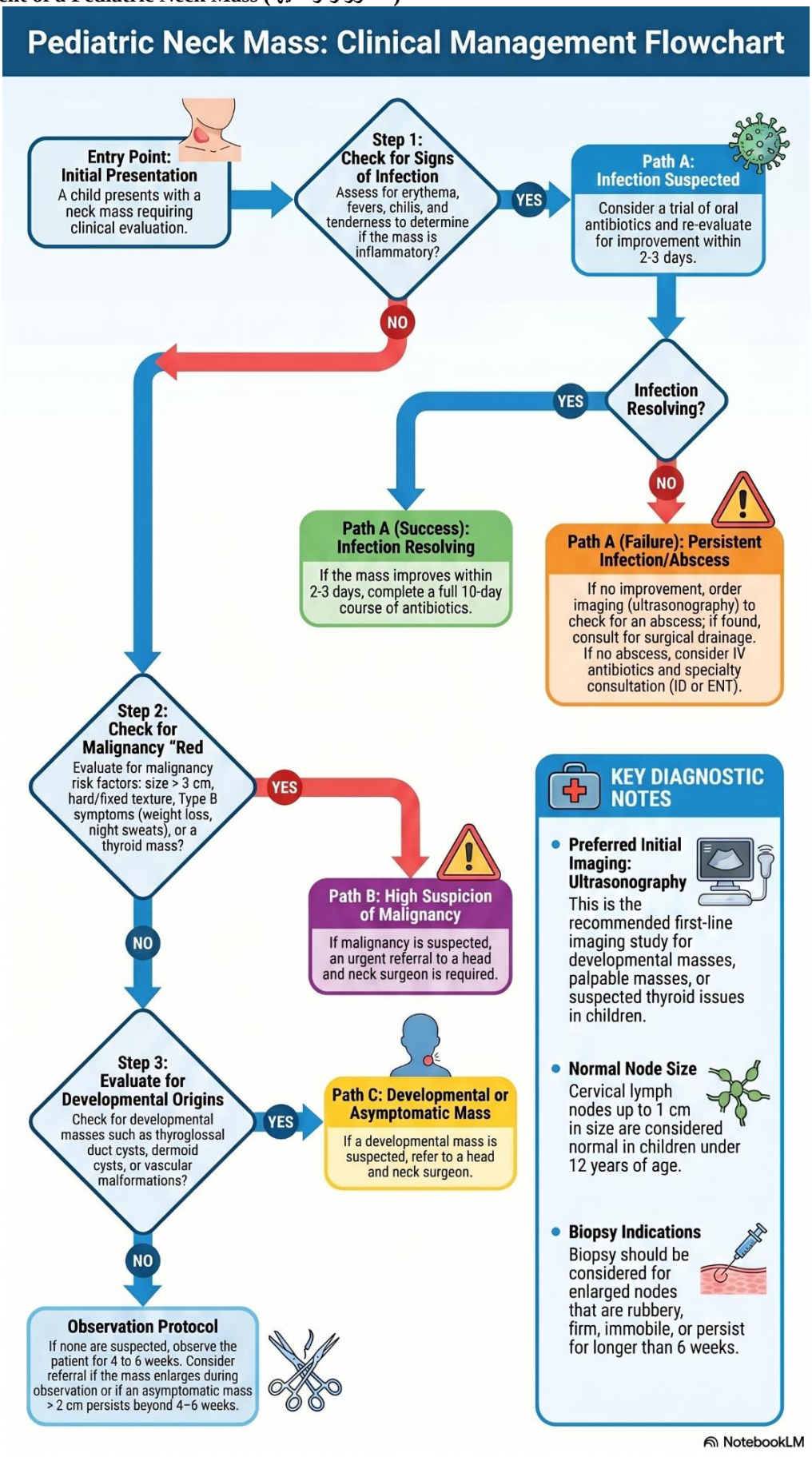
Others:

- **Imaging**
  - **CT** → Deep lesions, suspicious lesions
  - **MRI** → Vascular malformations
- **Labs**
  - **CBC** → Systemic disease
  - **PPD** → TB
  - **Viral titers** (CMV, EBV, HIV)
  - **Bartonella titers**

Red Flags for Malignancy

1. Persistent mass > 6 weeks
2. Size > 2 cm
3. Hard, immobile mass
4. Increasing size despite antibiotics
5. Supraclavicular location
6. Thyroid mass (**Thyroid mass in children → Cancer until proven otherwise**)
7. B-symptoms (**Weight loss**, **Night sweats**)

Management of a Pediatric Neck Mass (الدكتور ركّز عليها)



Head & Neck Oncology

Epidemiology

- Head & neck cancer accounts for ~4% of all cancers
- Strong **male** predominance **(≈2:1)**
- Higher incidence in individuals **over 50 years**
- Increasing cases in younger populations due to viral causes

Primary prevention

- **Smoking and tobacco cessation**
- **Avoidance of oral sex** (HPV-associated cancers)
- **HPV vaccination** **(Gardasil 9)**
- Routine dental and oral screenings

A. Lip & Oral Cavity Cancer

Epidemiology of Lip and Oral Cavity Cancer

- Strong **male** predominance **(≈2:1)**
- Highest prevalence in Asian countries (**China**).
- Most common cancer in **Indian males** (16.2%) → Because of **Betel Quid Use** (**التنباك**)
- Increasing incidence in young adults (<45 years)
- 5-year survival **<50%**

Subsites of Oral Cavity

- **Mobile tongue (anterior 2/3): 43% (most common)**
- Floor of mouth: **14%**
- Buccal mucosa
- Hard palate
- Alveolar ridge/gum
- Retromolar trigone

Risk Factors

- **Tobacco**, **alcohol**, **betel quid use** (Oral Cancer)
- Sun exposure (Lip Cancer)

Clinical Work-Up

A. Hx & PEx

B. Investigations

- **Biopsy** (mandatory)
- Endoscopy
- Imaging:
  - **CT/MRI** (for local spread)
  - **Ultrasound + FNA** (for nodal status)
  - **PET-CT** (for systemic staging)
  - Imaging Focus:
    - **Depth of invasion (DOI)**
    - **Perineural invasion**
    - Nodal status
    - Soft tissue spread
    - Mandibular involvement

Histology

Conventional (≈90%)

- **Squamous Cell Carcinoma** **(SCC)** and its subtypes

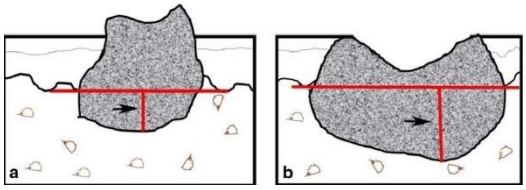
Unconventional (≈10%)

- Oral mucosal melanoma,
- Salivary gland tumors
- Soft tissue and neural tumors
- Hematolymphoid tumors
- Secondary metastases (kidney, lung)

Metastatic Spread in Oral Cancer

Nodal Metastasis

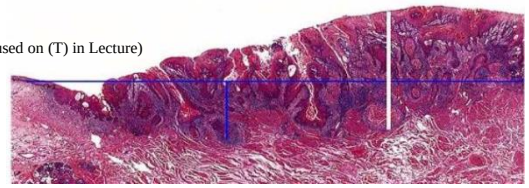
- ↑ Risk if → **High-grade** and **Perineural Invasion (PNI)**
- Poorer prognosis
- Rare "skip metastasis" (spread to distant neck LNs directly)



Distant Metastasis

- Occurs in 15% at time of initial diagnosis

Staging of Oral Cancer (AJCC 8th Edition) → TNM Staging (Doctor Focused on (T) in Lecture)



Tumor (T)

- DOI has replaced **Tumor Thickness** in T staging
- T1–T3 based on → **Size** + **DOI**
- T4 indicates invasion of adjacent structures

B. Oropharyngeal SCC Cancer

Epidemiology

- Strong **male** predominance **(≈2:1)**
- Global incidence: **1.1 per 100,000**
- High rates in **Europe** and **USA**
- **80% HPV-related in the USA**

HPV-Related vs Non-HPV Oropharyngeal Cancer

Feature	HPV+	HPV−
Age	**Younger**	**55–65 years**
Risk factors	**Less smoking**	**Tobacco/alcohol**
Morphology	**Non-keratinizing SCC** (95%)	**Keratinizing SCC** (85%)
Common Staging at Diagnosis	Early T, advanced N	Variable

Subsites of Oropharyngeal Cancer (HPV -ve)

- **Tonsil (44%)**
- **Base of tongue (31%)**
- Soft palate
- Pharyngeal wall

Metastatic Spread in Oropharyngeal Cancer

Distant Metastasis

- Rare at time of initial diagnosis (**~1%**)
- More common during follow-up (7% in HPV+)
  - About HPV+:
    - Characteristic large, cystic LN mets
    - May be mistaken for branchial cleft cysts
      - **Branchial cleft cysts Dx is not acceptable in adults until biopsy done**
- Common distant sites:
  - **Lung** (80%)
  - Bone
  - Liver

Diagnostic Methods (HPV +ve)

- **p16 immunohistochemistry** (Screening)
- DNA ISH (Confirmation, Low Sensitivity)
- PCR (HPV DNA)
  - Alone is insufficient to confirm diagnosis
- **E6/E7 mRNA PCR** **(Gold Standard)**

Prognosis of Oropharyngeal Cancer

- **HPV +ve** → 5 years survival = 82% (**Better Prognosis**)
- HPV −ve → 5 years survival = 44%
- Lower recurrence and progression in HPV+ patients

Staging of Oropharyngeal Cancer → TNM Staging (Doctor skipped this part)

C. Laryngeal Cancer

Epidemiology

- Strong **male** predominance **(≈3:1)**

Subsites of Laryngeal Cancer

- **Glottis** **(60%)**
- **Supraglottis** **(35%)**
- Subglottis **(5%)**

Symptoms (each type can present with any symptom of ↓, but most commonly)

- **Dysphonia** (early sign)
- Glottic → **Hoarseness**
- Supraglottic → **Dysphagia**
- Subglottic → **Dyspnea**

Histology

Conventional (≈90%)

- **Squamous Cell Carcinoma (SCC)** and its subtypes
- **Lymphoepithelial**

Unconventional (≈10%)

- Neuroendocrine tumors
- Salivary gland tumors
- Soft tissue and cartilage tumors
- Hematolymphoid tumors
- Secondary metastases (kidney, lung)

D. Hypopharyngeal Cancer

Epidemiology

- Strong **male** predominance **(≈2:1)**

Subsites of Hypopharyngeal Cancer

- **Pyriform sinus** **(65%)**
- **Posterior wall** **(25%)**
- Retrocricoid **(10%)**

General Features

- ***Aggressive***
  - Early infiltrative
  - Submucosal spread
- ***Late Diagnosis*** **(70–85% stage III–IV)**
  - High nodal and distant metastasis rates
  - Common distant metastasis sites: lung, liver, brain, bone
  - **Field cancerization** (multiple independent primary tumors in the same region)

Clinical Features

- Neck masses
- **Chronic Ear Pain** (referred pain) (The doctor focused on this symptom in lecture)
- Dysphagia, odynophagia
- Hoarseness
- Recent Weight loss

Metastatic Spread Risk at Time of Diagnosis

Site	Nodal Metastasis	Occult Metastasis	Distant Metastasis
Glottic	4%	12%	rare
Supraglottic /Subglottic	50%	23%	2.7%
Hypopharyngeal	**70%**	**20% of Contralateral Neck!!**	**7.2%**

Clinical Use (Why this matters)

- Predict nodal disease → Guide neck management (discussed in last page)

Risk Factors for Laryngeal & Hypopharyngeal Cancer

- **Tobacco**, **alcohol** (most important risk factors)
- **Industrial exposure** (specific for laryngeal cancer)
    - **Asbestoses**
    - **Synthetic fibers**
    - **Dusts**
    - **Fumes**

Approach for Laryngeal & Hypopharyngeal Cancer

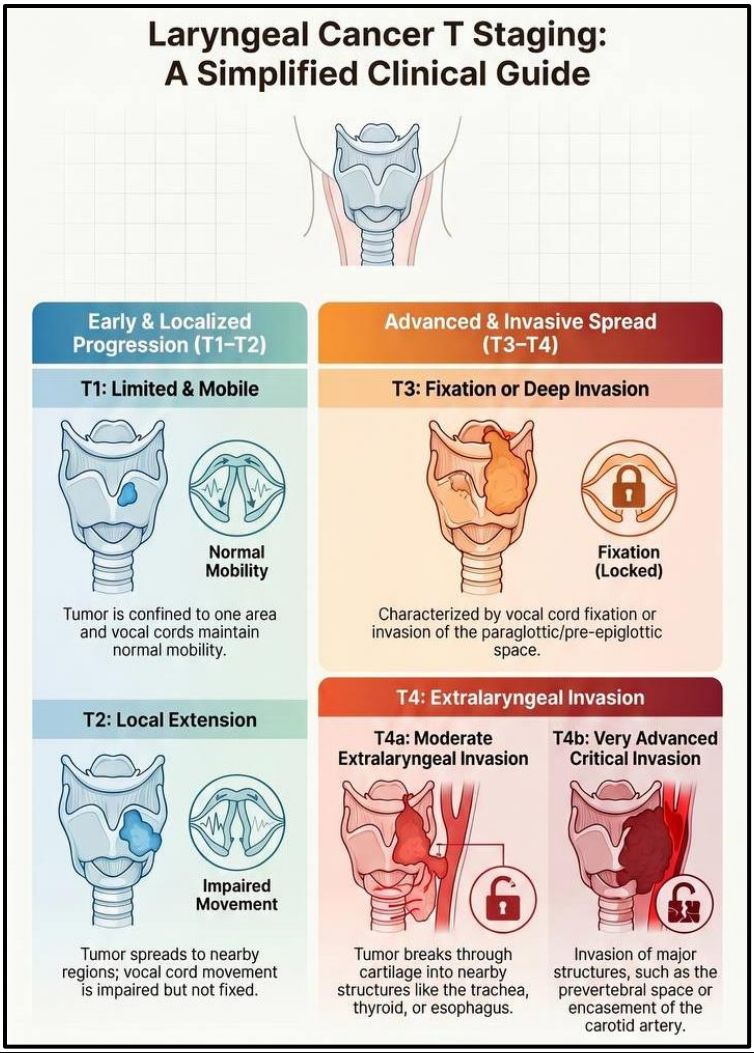
- Hx & PEx
- Endoscopy → **Fiberoptic Examination** (for staging) OR **Video-laryngo-stroboscopy (VLS)**
- **Biopsy**
- Imaging:
    - CT/MRI
    - PET-CT

Imaging and "Magic Plane" Concept

- **CT** is generally accurate
- **Magic Plane**
    - Line tangential to vocal process & perpendicular to the thyroid lamina
- If tumor extends beyond this **magic plane**:
    - CT loses accuracy
    - **MRI** is required
- This indicates **advanced disease with extra-laryngeal spread**

Staging of Hypopharyngeal Cancer → TNM Staging (Doctor skipped this part)

Staging of Laryngeal Cancer → TNM Staging (مهم جدا)



E. Nasopharyngeal Cancer

Epidemiology

- Strong **male** predominance **(≈2:1)**
- Rare in Western countries
- Very high incidence in **China: ~30 per 100,000**
- **Most common head & neck cancer in Jordan (2nd most common in Jordan is laryngeal cancer)**

Histopathology

Main Types

- **Non-keratinizing SCC (most common)**
- Keratinizing SCC
- Basaloid SCC

Other Tumors

- Nasopharyngeal adenocarcinoma
- Salivary gland tumors
- Hematolymphoid tumors
- Notochordal tumors

Local Tumor Spread

NPC spreads to:

- Nasal cavity
- Paranasal sinuses
- Oropharynx
- Parapharyngeal space
- Skull base
- Cavernous sinus
- Cranial cavity and cervical spine

Metastatic Spread

Nodal Metastasis

- **Very common**: **60–75% at diagnosis**

Distant Metastasis

- Common **(~9% at diagnosis)**
- Common sites:
  - **Bone** (50%, other head & neck cancers tend to mets into lungs)
  - Lung / Liver

Clinical Presentation

Neck

- **Neck mass** in **60–75% at diagnosis**

Nasal Symptoms

- Epistaxis
- Chronic nasal obstruction (CRS)
- Mucopurulent discharge
- Loss of smell

Otologic Symptoms

- Otitis media with effusion
  - Note from doctor:
    - Any unilateral OME is **nasopharyngeal cancer** until proven otherwise
    - You must rule out nasopharyngeal cancer
- Hearing loss
- Tinnitus

Neurological Involvement

- III, IV, VI → ophthalmoplegia
- V → facial pain
- IX–XI → jugular foramen syndromes
- XII → tongue deviation
- Sympathetic trunk → Horner syndrome

Risk Factors

- **EBV**
- **Ethnicities**
  - **Chinese**
- **Industrial exposure**
  - **Wood dust**
  - **Nickle dust**
  - **Formaldehydes**

Approach

- Hx & PEx
- Endoscopy
  - Considerations:
    - NPCs may be **submucosal** → endoscopy can appear normal **(10%)**
    - Most NPCs arise in the **Rosenmüller fossa**, a hidden area behind the torus tubarius
- Biopsy (endoscopy-guided)
- **EBV Assessment**
  - Methods:
    - **EBER RNA ISH (must take biopsy)**
    - **Plasma EBV-DNA PCR**
  - Role of EBV plasma levels:
    - Follow-up
    - Screening in endemic regions
    - Prognostic tool → EBV +ve = **Better prognosis**
- Imaging
  - CT / MRI
  - PET-CT / PET-MRI

Principles of Management of Head & Neck Cancer

A. Multidisciplinary Team (MDT) → Patients treated by MDT have **better prognosis**

B. Treatment Goals

- Achieve oncologic control
- Preserve function (speech, swallowing)
- Avoid unnecessary multimodality treatment

C. Disease Factors that determine the modality of treatment

1. Treatment by Site

a. **Nasopharynx**

- **Radiotherapy** (stages I/II)
- ± **Chemotherapy** (stages III/IV)
- ± Surgery (limited role)

b. Oral Cavity

- **Surgery**
- ± Adjuvant RT/CRT

c. Oropharynx

- **Surgery**
- ± Adjuvant RT/CRT

d. **Larynx** & **Hypopharynx**

- **Radiotherapy**
- **Chemoradiotherapy**
- **Surgery** (stage IV)

2. Treatment by Stage

- Stage I → Single modality
- Stage II–III → Dual modality
- Stage IV → Tri-modality

D. Patient Factors that determine the modality of treatment

- Nutritional status
- Comorbidities (DM, IHD, anemia)
- Age >70 → limited chemotherapy benefit

E. Adjuvant Therapy Indications

1. Radiotherapy

Absolute:

- Positive resection margins (<1 mm)
- T4 tumors
- N2/N3 disease
- Extranodal extension (ENE) = **cN3b (TNM)**

Relative:

- N1 disease
- **PNI** (perineural invasion)
- **LVI** (lympho-vascular invasion)
- Close resection margins (1–5 mm)

2. Chemotherapy

- Positive resection margins (<1 mm)
- Extranodal extension (ENE)

F. Neck Dissection

Types

- **Laryngeal & Hypopharyngeal Cancer**
  1. **Therapeutic (TND)** → nodal metastasis
  2. **Elective (END)** → occult metastasis
- **Nasopharyngeal Cancer**
  3. **Salvage (SND)** → previously treated neck by RT/CRT

Indications of Elective Neck Dissection (END):

- When occult metastasis risk > **15–20%**
- DOI > 4 mm is a key indication