ENT Summary Handbook

Otolaryngology: Head and Neck Surgery
Board-Review Quality . Comprehensive Academic Reference

13 Chapters . Premium Tables . Key Teaching Points
OSCE Pearls . MCQ Traps . High-Yield Facts

2026 Edition

TABLE OF CONTENTS

Chapter 1

Gross Anatomy and Physiology of Hearing

Peripheral Auditory System, Central Pathways, and Sound Transduction Mechanisms

The auditory system transforms acoustic energy from the environment into electrical signals interpreted by the brain. It is divided into peripheral and central components. The peripheral system includes the outer ear, middle ear, inner ear, and the auditory nerve (CN VIII). The central system comprises brainstem nuclei and the auditory cortex in the temporal lobe.

THE OUTER EAR

The outer ear consists of the auricle (pinna) and the external auditory canal (EAC). It collects sound waves and conducts them to the tympanic membrane. The outer ear provides approximately 13 dB of amplification through resonance in the 3 to 6 kHz range.

The Auricle (Pinna)

The auricle is a cartilaginous structure (except the lobule) covered with skin. Its unique shape helps collect and localize sound.

- Helix: the most distal border of the auricle
- Antihelix: anterior ridge that bifurcates superiorly
- Tragus: projects immediately in front of the ear canal
- Concha: divided into cavum (lower) and cymba (upper)
- Lobule: the soft, cartilaginous-free inferior portion

Clinical Importance: The auricle is susceptible to trauma (auricular hematoma leading to cauliflower ear), perichondritis, keloids, and skin cancers (BCC, SCC, melanoma). Congenital anomalies include microtia, anotia, and atresia. Low-set ears may indicate chromosomal abnormalities.

External Auditory Canal (EAC)

The EAC is approximately 2.5 cm long and 0.7 cm in diameter. It follows an S-shaped course. The outer one-third is cartilaginous and contains hair follicles, sebaceous glands, and ceruminous glands. The inner two-thirds passes through the tympanic portion of the temporal bone.

- Two constrictions: osseocartilaginous junction and isthmus (0.5 cm from TM)
- Outer 1/3: cartilaginous with hairs and ceruminous glands
- Inner 2/3: bony, thin skin, very sensitive to instrumentation

- Self-cleaning property: epithelial migration outward, aided by jaw movement

Table 1.1: Outer Ear Structures and Clinical Relevance

Structure	Description	Clinical Relevance
Helix	Outer rim	Frostbite, skin cancer
Tragus	Anterior projection	Tragus sign in otitis externa
Concha	Deep central depression	Cauliflower ear from hematoma
EAC outer 1/3	Cartilaginous, has cerumen glands	Otitis externa, furunculosis
EAC inner 2/3	Bony, thin skin	Very painful to instrumentation
Isthmus	Narrow point 0.5 cm from TM	Site of foreign body impaction

THE MIDDLE EAR

The middle ear is an air-filled cavity within the temporal bone containing the three ossicles (malleus, incus, stapes), two muscles (tensor tympani, stapedius), and connected to the nasopharynx via the Eustachian tube. Its primary function is impedance matching, amplifying sound by approximately 20 to 23 dB.

Tympanic Membrane

The TM is a semi-transparent, concave, oval structure approximately 55 square mm in area. It consists of three layers: outer epithelial, intermediate fibrous, and inner mucosal.

- Pars tensa: the larger, fibrous portion that vibrates
- Pars flaccida: upper portion lacking the fibrous layer
- Umbo: the most depressed point where the malleus handle attaches
- Light reflex: cone-shaped reflection at 5 o'clock (left) or 7 o'clock (right)
- Handle of malleus runs at 11 o'clock in the left ear, 1 o'clock in the right

The Ossicular Chain

The three ossicles transmit and amplify vibrations from the TM to the oval window.

- Malleus: handle embedded in TM, head articulates with incus
- Incus: body articulates with malleus, long process with stapes
- Stapes: smallest bone in the body; footplate in oval window
- Total amplification: approximately 20-23 dB (area ratio 17:1 times lever ratio 1.3:1)

Table 1.2: Middle Ear Structures and Functions

Structure	Function	Innervation
Tympanic membrane	Sound transduction, amplification	CN V, IX, X

Structure	Function	Innervation
Malleus	Transmits vibration from TM	None
Incus	Connects malleus to stapes	None
Stapes	Transmits to oval window	None
Tensor tympani	Tenses TM	CN V3
Stapedius	Dampens loud sounds	CN VII
Eustachian tube	Pressure equalization	CN V3

THE INNER EAR

The inner ear (labyrinth) lies within the petrous part of the temporal bone. It consists of the cochlea (hearing) and the vestibular system (balance). The bony labyrinth contains perilymph, and the membranous labyrinth contains endolymph.

The Cochlea

The cochlea is a spiral-shaped structure making approximately 2.5 turns around the modiolus. It is divided into three chambers: scala vestibuli, scala media, and scala tympani. The organ of Corti within the cochlear duct is the sensory receptor for hearing.

- Scala vestibuli (upper): contains perilymph, begins at oval window
- Scala media (cochlear duct): contains endolymph, houses organ of Corti
- Scala tympani (lower): contains perilymph, ends at round window
- High frequencies at the base; Low frequencies at the apex (tonotopic)

Organ of Corti: Contains inner hair cells (IHCs, about 3,500) and outer hair cells (OHCs, about 12,000). IHCs are the primary sensory receptors (95 percent of afferent innervation). OHCs amplify and fine-tune via electromotility.

Vestibular System

The vestibular system detects head position and movement through three semicircular canals (angular acceleration) and two otolithic organs: utricle and saccule (linear acceleration and gravity).

- Semicircular canals: anterior, posterior, lateral (horizontal)
- Each canal has an ampulla containing the cupula with hair cells
- Utricle: horizontal plane, detects horizontal linear acceleration
- Saccule: vertical plane, detects vertical acceleration and gravity

Table 1.3: Inner Ear Structures and Functions

Structure	Function	Key Feature
Cochlea	Hearing	Tonotopic: high freq at base, low at apex
Organ of Corti	Sound transduction	IHCs sensory, OHCs amplification
Semicircular canals	Angular acceleration	3 canals in 3 planes
Utricle	Horizontal linear acceleration	Macula in horizontal plane
Saccule	Vertical acceleration and gravity	Macula in vertical plane

PHYSIOLOGY OF HEARING

Sound transduction involves energy transformations: acoustic to mechanical to hydraulic to electrochemical to electrical.

- Acoustic: sound waves collected by pinna, through EAC, to TM
- Mechanical: TM vibrates then ossicular chain then stapes footplate
- Hydraulic: stapes pushes oval window creating perilymph waves
- Electromechanical: basilar membrane moves, hair cell stereocilia deflect

TUNING FORK TESTS

The 512 Hz tuning fork is most commonly used. Three essential tests:

- Rinne test: compares air conduction (AC) to bone conduction (BC)
- Positive Rinne: AC greater than BC (normal or SNHL)
- Negative Rinne: BC greater than AC (conductive loss over 25 dB)
- Weber test: fork on vertex; lateralizes to affected ear in conductive loss, to normal ear in SNHL
- Schwabach test: compares patient BC to examiner normal hearing

Table 1.4: Tuning Fork Test Interpretation

Condition	Rinne (affected)	Weber	Schwabach
Normal hearing	Positive	Midline	Normal
Conductive loss	Negative	Lateralizes to affected ear	Prolonged
Sensorineural loss	Positive (both reduced)	Lateralizes to normal ear	Diminished

- Outer ear amplifies sound by about 13 dB through resonance at 3-6 kHz

- Middle ear provides about 20-23 dB amplification via area ratio and lever action
- Cochlea is tonotopically organized: high frequencies at base, low at apex
- Inner hair cells are primary receptors; outer hair cells amplify via electromotility
- Endolymph has high K+; perilymph has high Na+
- Stapedius muscle (CN VII) mediates acoustic reflex; paralysis causes hyperacusis
- Eustachian tube dysfunction is the most common cause of middle ear pathology in children
- Rinne: positive = AC greater than BC (normal or SNHL); negative = conductive loss
- Weber: lateralizes to affected ear in conductive loss, to normal ear in SNHL
- Central auditory pathway has bilateral representation

Chapter 2

Head and Neck Oncology

Carcinogenesis, Epidemiology, Staging, and Management

Head and neck cancer accounts for approximately 4 percent of all cancers worldwide. The vast majority (85 to 90 percent) are squamous cell carcinomas arising from the mucosal epithelium of the upper aerodigestive tract. These cancers are more than twice as common in men and are diagnosed more frequently in people over age 50. Worldwide, approximately 562,328 people were diagnosed in 2020.

EPIDEMIOLOGY AND RISK FACTORS

The two most important risk factors are tobacco and alcohol use. Combined use increases risk by approximately 9-fold (synergistic effect).

- Tobacco (smoking and smokeless): 3-fold increase
- Alcohol: 6-fold increase; combined with tobacco equals 9-fold synergistic increase
- HPV (especially type 16): risk factor for oropharyngeal cancers (tonsil, base of tongue)
- EBV infection: nasopharyngeal cancer and salivary gland cancers
- Betel quid (paan): strongly associated with mouth cancers in Southeast Asia
- Occupational exposure: wood dust, nickel dust, formaldehyde
- Radiation exposure: salivary gland cancers, sarcoma, SCC
- Fanconi anemia: increased risk of precancerous lesions and cancers
- Most common form of cancer in males in India (16.2 percent of all cancers)
- Incidence in young adults under 45 is rising, linked to HPV
- 5-year survival rate remains below 50 percent for oral cavity cancers

Table 2.1: Risk Factors for Head and Neck Cancer

Risk Factor	Associated Cancer	Risk Increase
Tobacco	Oral cavity, hypopharynx, larynx	3x
Alcohol	Oral cavity, hypopharynx, larynx	6x
Tobacco plus Alcohol	Oral cavity, hypopharynx	9x synergistic
HPV (type 16)	Oropharynx	Major risk
EBV	Nasopharynx	Major risk
Betel quid	Oral cavity	Major risk in Asia
Radiation	Salivary glands, sarcoma	Iatrogenic

CARCINOGENESIS

Cancer development follows three steps: (A) initiation, irreversible genetic damage; (B) progression, clonal expansion with additional mutations; (C) metastasis, invasion through basement membrane and colonization of distant sites. Monoclonal growth is the hallmark of malignancy.

Field cancerization: The entire mucosal field of the upper aerodigestive tract is exposed to the same carcinogens, leading to widespread genetic alterations. This explains the high rate of second primary tumors (synchronous or metachronous) in head and neck cancer patients.

LIP AND ORAL CAVITY CANCER

Globally, oral cancer constitutes 2.9 percent of all cancers in males and 1 percent in females. Subsite distribution: Mobile tongue 43 percent, Floor of mouth 14 percent, Alveolar ridge 11 percent, Buccal mucosa 7 percent, Retromolar trigone 4 percent.

- Most common histology: Squamous cell carcinoma (over 90 percent)
- Nodal spread: Levels I-IV; bilateral for midline or mobile tongue
- Distant metastasis at diagnosis is rare; lung is the most common site

Table 2.2: Oral Cavity Cancer Key Features

Feature	Details
Most common site	Mobile tongue (43 percent)
Most common histology	Squamous cell carcinoma (over 90 percent)
Nodal spread	Levels I-IV; bilateral for midline or mobile tongue
Distant metastasis	Rare at diagnosis; lung most common
5-year survival	Below 50 percent overall

OPHARYNGEAL CANCER

Oropharyngeal cancer involves the tonsil, base of tongue, soft palate, and pharyngeal walls. HPV-related OPC has distinct epidemiology and better prognosis compared to HPV-negative OPC.

- HPV type 16 is responsible for over 90 percent of HPV-related OPC
- p16 immunohistochemistry is the routinely used surrogate marker
- E6/E7 HPV-mRNA PCR is the gold standard for confirming active infection

- 5-year OS: 82.4 percent HPV-positive vs 44.0 percent HPV-negative
- Lower risk of disease progression: 7.8 percent vs 21.2 percent
- Fewer second primaries: 16.1 percent vs 49.9 percent

Table 2.3: HPV-positive vs HPV-negative Oropharyngeal Cancer

Feature	HPV-positive (p16+)	HPV-negative (p16-)
Etiology	HPV (type 16)	Tobacco, alcohol
5-year OS	82.4 percent	44.0 percent
Disease progression	7.8 percent	21.2 percent
5-year recurrence	5.6 percent	20.5 percent
Second primaries	16.1 percent	49.9 percent

LARYNGEAL AND HYPOPHARYNGEAL CANCER

New cases in 2020: Larynx 184,615; Hypopharynx 84,254. Both are strongly associated with tobacco and alcohol use.

- Glottis (60 percent): earliest symptom is hoarseness, leading to earlier diagnosis
- Supraglottis (35 percent): dysphagia, odynophagia, referred otalgia
- Subglottis (5 percent): stridor, dyspnea, often late presentation
- Piriform sinus is the most common hypopharyngeal subsite (65-70 percent)
- Hypopharyngeal cancer presents late: 70-85 percent at stage III-IV
- Symptoms include neck mass, throat pain, dysphagia, hoarseness, and weight loss

Table 2.4: Laryngeal Cancer by Subsite

Subsite	Frequency	Early Symptom	Nodal Spread
Glottis	60 percent	Hoarseness	Low (sparse lymphatics)
Supraglottis	35 percent	Dysphagia, otalgia	High (rich lymphatics)
Subglottis	5 percent	Stridor, dyspnea	Moderate (level VI)

- HPV-positive OPC has BETTER prognosis than HPV-negative OPC
- p16 IHC is a SURROGATE marker, not the gold standard (E6/E7 mRNA PCR is gold standard)
- Glottic cancer presents EARLIEST due to hoarseness (not supraglottic)
- Supraglottic cancer has HIGHEST nodal spread due to rich lymphatic drainage

- Tobacco plus alcohol equals SYNERGISTIC (9x), not additive
- Age over 50 with lateral cystic node: rule out metastatic tumor (not just branchial cyst)

- Head and neck cancer: 4 percent of all cancers; 85-90 percent are SCC
- Tobacco plus alcohol equals synergistic 9-fold risk increase
- HPV type 16 causes oropharyngeal cancer with better prognosis
- p16 IHC is surrogate marker; E6/E7 mRNA PCR is gold standard for HPV
- Glottis (60 percent): hoarseness leads to early diagnosis
- Supraglottic cancer has highest nodal spread due to rich lymphatic drainage
- Hypopharyngeal cancer presents late (70-85 percent stage III-IV)
- Mobile tongue is the most common oral cavity site (43 percent)
- Field cancerization explains second primary tumors
- Age over 50 with neck mass: 80 percent of non-thyroid masses are neoplastic, 80 percent of those malignant

Chapter 3

Epistaxis and Nasal Trauma

Anatomy, Classification, Management, and Complications of Nosebleeds

Epistaxis (nosebleed) is one of the most common ENT emergencies, affecting approximately 60 percent of the population at some point in their lives. While most cases are benign and self-limiting, posterior epistaxis can be life-threatening and requires prompt, systematic management.

NASAL VASCULAR ANATOMY

The nasal cavity receives blood supply from both the internal and external carotid arteries. Understanding this dual supply is critical for managing refractory epistaxis.

- Anterior ethmoidal artery: branch of ophthalmic artery (from ICA)
- Posterior ethmoidal artery: branch of ophthalmic artery (from ICA)
- Sphenopalatine artery (SPA): terminal branch of maxillary artery (from ECA); most common source of posterior epistaxis
- Greater palatine artery: inferior septum and anterior hard palate
- Superior labial artery (from facial): anterior septum

Kiesselbach Plexus (Little Area)

An anastomotic arterial network on the anterior nasal septum, approximately 1.5 cm from the anterior naris. Formed by the anterior ethmoidal, sphenopalatine, greater palatine, and superior labial arteries. Source of approximately 90 percent of all epistaxis cases. It is superficial and easily traumatized.

Woodruff Plexus

Located on the posterior lateral nasal wall, just below the posterior end of the inferior turbinate. Formed by anastomoses of the sphenopalatine and pharyngeal artery branches. Most common site of posterior epistaxis.

Table 3.1: Nasal Blood Supply Summary

Artery	Source	Region Supplied
Anterior ethmoidal	ICA (ophthalmic)	Superior septum
Sphenopalatine	ECA (maxillary)	Posterior septum and floor
Greater palatine	ECA (maxillary)	Inferior septum

Artery	Source	Region Supplied
Superior labial	ECA (facial)	Anterior septum

CLASSIFICATION

- Anterior epistaxis (90 percent): from Kiesselbach plexus; usually mild and self-limiting; visible on anterior rhinoscopy
- Posterior epistaxis (10 percent): from Woodruff plexus or SPA; more severe, requires hospital admission; blood flows posteriorly into the pharynx
- Primary (80-90 percent): spontaneous, no identifiable cause
- Secondary (10-20 percent): caused by trauma, surgery, tumors, coagulopathy

ETIOLOGY

- Local trauma: nose picking (most common in children), blunt trauma, foreign body
- Infection: rhinosinusitis, nasal vestibulitis
- Environmental: dry air, altitude, chemical irritants
- Anatomical: septal deviation, septal perforation
- Neoplastic: JNA (adolescent males), SCC, inverted papilloma
- Hypertension: most common systemic association
- Coagulopathy: hemophilia, vWD, thrombocytopenia, anticoagulants
- Hereditary hemorrhagic telangiectasia (Osler-Weber-Rendu syndrome)

Table 3.2: Etiology of Epistaxis

Category	Causes
Local: Trauma	Nose picking, blunt trauma, foreign body, nasal surgery
Local: Infection	Rhinosinusitis, nasal vestibulitis
Local: Neoplastic	JNA (adolescent males), SCC, inverted papilloma
Systemic: Vascular	Hypertension, atherosclerosis
Systemic: Coagulopathy	Hemophilia, vWD, thrombocytopenia, anticoagulants
Systemic: Hereditary	HHT (Osler-Weber-Rendu)

MANAGEMENT

Stepwise approach:

• Step 1: Sit upright, lean forward, apply firm pressure to soft nose for 10-15 minutes
• Step 2: Topical vasoconstrictor (oxymetazoline or cocaine 4 percent)
• Step 3: Chemical cautery (silver nitrate sticks) for visible bleeding point; avoid bilateral simultaneous application
• Step 4: Anterior nasal packing (ribbon gauze, Merocel, or balloon catheters)
• Step 5: Posterior nasal packing (Foley catheter or specialized pack); requires hospital admission and antibiotics
• Step 6: Endoscopic sphenopalatine artery ligation for refractory cases
• Step 7: Embolization by interventional radiology for refractory cases

Table 3.3: Stepwise Management of Epistaxis

Step	Intervention	Indication
1	First aid: pressure, lean forward, ice	All cases
2	Topical vasoconstrictor	Persistent bleeding
3	Chemical cautery (silver nitrate)	Visible bleeding point
4	Anterior nasal packing	Failed cautery or diffuse bleeding
5	Posterior nasal packing	Posterior epistaxis
6	Endoscopic SPA ligation	Failed packing
7	Embolization	Refractory cases

NASAL TRAUMA

• Nasal bone fracture: most common facial fracture
• Diagnosis: clinical (deformity, crepitus, tenderness) plus X-ray lateral view
• Closed reduction within 7-10 days before bone union
• Septal hematoma: SURGICAL EMERGENCY, must drain immediately
• Untreated septal hematoma leads to cartilage necrosis and saddle nose deformity
• Always examine the septum in any nasal trauma patient

• Septal hematoma is a SURGICAL EMERGENCY
• Must be drained immediately to prevent cartilage necrosis
• Untreated leads to septal abscess then saddle nose deformity
• Always examine the septum in any nasal trauma patient

- 90 percent of epistaxis is anterior, from Kiesselbach plexus (Little area)
- Posterior epistaxis: source is usually sphenopalatine artery branches (Woodruff plexus)
- Management follows stepwise approach: pressure to cautery to packing to surgery
- Silver nitrate cautery: avoid bilateral simultaneous application (septal perforation risk)
- Posterior packing requires hospital admission and antibiotics (toxic shock syndrome risk)
- Endoscopic SPA ligation is first-line surgical option for refractory epistaxis
- Septal hematoma is a surgical emergency, drain immediately to prevent saddle nose
- Nasal bone fracture: closed reduction within 7-10 days
- JNA: adolescent male with recurrent epistaxis and nasal obstruction, never biopsy
- HHT (Osler-Weber-Rendu): autosomal dominant with recurrent epistaxis, telangiectasias, and AVMs

Chapter 4

Assessment of Hearing in Children

Screening, Audiologic Tools, and Rehabilitation of Pediatric Hearing Loss

Hearing loss in children is a critical concern because it directly impacts speech and language development, academic performance, and social integration. Early identification and intervention are essential. Universal newborn hearing screening programs aim to identify hearing loss by 1 month, confirm by 3 months, and intervene by 6 months of age.

WHY ASSESS HEARING IN CHILDREN

The most common form of sensorineural hearing loss is due to cochlear pathology. Approximately 50 percent of patients with hearing loss do not have any known risk factor, emphasizing the importance of universal screening.

- 1-3 per 1,000 newborns have significant permanent hearing loss
- 50 percent of hearing loss cases have no identifiable risk factor
- Early intervention before 6 months dramatically improves speech and language outcomes
- Normal hearing in children: up to 15 dB (stricter than adults at 25 dB)

RISK FACTORS FOR HEARING LOSS IN CHILDREN

The Joint Committee on Infant Hearing identifies the following risk indicators:

- Maternal infection: toxoplasmosis, rubella, CMV, herpes simplex, syphilis
- Childhood infectious disease: bacterial meningitis
- Intrauterine exposure to radiation
- Maternal or child ototoxic drug use
- Hypoxia and prolonged mechanical ventilation
- Birth weight under 1500 g
- Prematurity
- Hyperbilirubinemia (kernicterus)
- Family history of hearing loss
- Craniofacial anomalies
- Neurodegenerative disorders
- Parental suspicion of hearing loss or delayed language or speech

Table 4.1: Risk Factors for Pediatric Hearing Loss

Category	Risk Factors
Prenatal	Maternal infections (TORCH), radiation exposure, ototoxic drugs
Perinatal	Prematurity, low birth weight (under 1500g), hypoxia, hyperbiliru
Postnatal	Meningitis, ototoxic drugs, head trauma, family history
Other	Craniofacial anomalies, neurodegenerative disorders, parental co

AUDIOLOGIC TOOLS

Hearing assessment uses objective and behavioral tests. Objective tests (OAE, ABR) do not require cooperation; behavioral tests (PTA, play audiometry, VRA) require participation.

Otoacoustic Emissions (OAE)

Sounds produced by the cochlea from outer hair cell motility. Mechanical energy propagates to the external ear canal and is measured by sensitive microphones.

- 80-90 percent sensitivity and specificity for screening
- Cannot detect auditory neuropathy
- Results affected by external and middle ear conditions (fluid, wax)
- Used in universal newborn hearing screening programs

Auditory Brainstem Response (ABR)

Auditory evoked potentials extracted from brainstem electrical activity after acoustic stimulation, recorded via scalp electrodes. It is a test of synchronous neural function and can estimate hearing sensitivity.

- Detects auditory neuropathy (unlike OAE)
- More specific but more expensive than OAE
- Requires the individual to be sleeping or completely relaxed (sometimes sedation)
- Gold standard for confirming hearing loss in newborns who fail OAE

Pure Tone Audiometry (PTA)

The gold standard for hearing threshold determination. Requires the child to understand and cooperate, mental age above 4 years. The audiogram plots thresholds across frequencies (250-8000 Hz) for air and bone conduction.

- Mental age over 4 years required
- Conductive loss: AC reduced, BC normal (air-bone gap present)
- SNHL: both AC and BC reduced (no air-bone gap)

Play Audiometry

A modification of standard PTA for younger children (2-5 years). The child is conditioned to respond to sound by performing a play activity such as placing a toy in a container.

- Age range: approximately 2-5 years

Visual Reinforcement Audiometry (VRA)

For younger children (6 months to 2-3 years). The child is encouraged to respond to sound by giving a visual stimulus (animated toy) when the child responds correctly.

- Age range: 6 months to 2-3 years
- Uses visual reinforcement to condition head-turn response

Tympanometry

Measures middle ear compliance as a function of air pressure. Essential companion to all audiologic tests to assess middle ear status.

- Type A: normal middle ear function
- Type B: flat curve, suggests effusion (glue ear) or perforation
- Type C: negative pressure, suggests Eustachian tube dysfunction

Table 4.2: Audiologic Tests by Age

Test	Age	Type	Key Feature
OAE	Newborn plus	Objective	Screening; cannot detect auditory ne
ABR	Newborn plus	Objective	Diagnostic; detects auditory neuropat
Tympanometry	All ages	Objective	Middle ear assessment
VRA	6 mo to 3 yr	Behavioral	Visual reinforcement for head-turn
Play audiometry	2-5 yr	Behavioral	Play response to sound
PTA	4 plus yr	Behavioral	Gold standard threshold test

CLASSIFICATION OF HEARING LOSS

- Conductive hearing loss: problem in outer or middle ear; AC reduced, BC normal
- Sensorineural hearing loss: problem in cochlea or CN VIII; both AC and BC reduced
- Mixed hearing loss: both conductive and sensorineural components
- Functional or nonorganic: malingering or psychogenic
- Central hearing loss: damage in brainstem or cortex

Degree of Hearing Loss:

- Normal: up to 25 dB (adults), up to 15 dB (children)
- Mild: 25-40 dB, difficulty with faint speech
- Moderate: 41-55 dB, difficulty with normal conversation
- Moderately severe: 56-70 dB, difficulty with loud speech
- Severe: 71-85 dB, can hear only shouted speech

- Profound: over 85 dB, cannot hear speech

REHABILITATION OPTIONS

- Hearing aids: amplify sound; first-line for most hearing losses
- Cochlear implants: for severe-to-profound SNHL when hearing aids are insufficient
- Bone-anchored hearing aids (BAHA): for conductive or mixed loss
- Hearing rehabilitation: sign language, lip reading, auditory verbal therapy
- FM systems in classroom settings

- Screen by 1 month, confirm by 3 months, intervene by 6 months
- OAE is the primary screening tool; ABR for diagnostic confirmation
- 50 percent of hearing loss cases have NO identifiable risk factor
- Early intervention before 6 months dramatically improves outcomes
- Auditory neuropathy can be missed by OAE, ABR is needed for confirmation

- Universal newborn screening: screen by 1 month, confirm by 3 months, intervene by 6 months
- OAE screening tool cannot detect auditory neuropathy
- ABR diagnostic gold standard detects auditory neuropathy
- Tympanometry must accompany all audiologic testing (Type B equals effusion)
- Normal hearing in children: up to 15 dB (stricter than adults at 25 dB)
- Play audiometry 2-5 years; VRA 6 months to 2-3 years
- Cochlear implants for severe-to-profound SNHL when hearing aids fail
- Risk factors: TORCH infections, meningitis, prematurity, family history
- 50 percent of hearing loss cases have no known risk factor, universal screening is essential

Chapter 5

Tinnitus and Deafness in Adults

Pathophysiology, Classification, Diagnosis, and Management

Tinnitus and hearing loss are among the most common complaints in adult ENT practice. Tinnitus affects approximately 10-15 percent of the adult population, while hearing loss is the most common sensory deficit in adults.

TINNITUS

Tinnitus is the perception of sound in the absence of an external acoustic stimulus. It is a symptom, not a disease. It can be described as hissing, roaring, ringing, clicking, or whooshing. It may be constant, pulsed, or intermittent, and unilateral or bilateral.

Classification

- Subjective tinnitus (95 percent): audible only to the patient
- Objective tinnitus (5 percent): audible to both patient and examiner
- Objective causes: vascular (aneurysms, glomus tumors), muscular (myoclonus), TMJ disorders
- Subjective causes: associated with SNHL in 80 percent of patients; conductive disorders, Meniere disease, presbycusis, noise exposure, ototoxicity
- Non-auditory: systemic diseases (HTN, DM, cervical spondylosis, stress, anemia), drugs

Pathophysiology

The exact mechanism is unknown, but tinnitus is now understood to have a central rather than peripheral origin. It persists after severing the eighth nerve (like phantom limb pain). The current model proposes: acute peripheral insult leads to chronic signal which leads to central modification which leads to psychological enhancement which leads to intractable tinnitus.

Treatment of Tinnitus

The aim is to eliminate the underlying disease. There is no single cure.

- Treat underlying cause: remove ototoxic drugs, treat Meniere disease, vascular lesions
- Sound therapy: tinnitus maskers after matching tests, bedside noise generators
- Cognitive-behavioral therapy: modify maladaptive thoughts and behaviors
- Tinnitus retraining therapy (TRT): habituation through sound therapy plus counseling
- Drugs: limited evidence; treat associated anxiety or depression
- Relaxation techniques: biofeedback, self-hypnosis, stress management

Table 5.1: Tinnitus Management Options

Approach	Details
Treat underlying cause	Remove ototoxic drugs, treat Meniere disease, vascular lesions
Sound therapy	Maskers, hearing aids, white noise generators
CBT	Modify maladaptive thoughts and behaviors
TRT	Habituation through sound therapy plus counseling
Pharmacotherapy	Limited evidence; treat associated anxiety or depression

DEAFNESS IN ADULTS

Hearing loss in adults can be classified by type (conductive, sensorineural, mixed), degree (mild to profound), and etiology.

Outer Ear Causes

- Congenital: microtia, atresia or stenosis of EAC (conductive HL up to 55-65 dB ABG)
- Wax impaction: most common cause of conductive HL in adults
- Tumors: SCC (most common malignant), BCC, melanoma of EAC
- Benign growths: exostosis (bilateral, swimmers), osteoma (unilateral, pedunculated)

Middle Ear Causes

- Congenital: ossicular chain malformation (most common: missing or malaligned stapes crura)
- AOM and OME: common in children, conductive hearing loss
- TM perforation: degree of HL depends on size and location
- Cholesteatoma: squamous epithelium in middle ear, erodes bone
- Otosclerosis: bony growth on stapes footplate; AD inheritance; young females; treat with HA or stapedectomy
- Eustachian tube dysfunction: Type C tympanogram

Inner Ear Causes

- Presbycusis: age-related; most common cause of HL in elderly; progressive symmetric high-frequency loss
- Noise-induced HL: 85+ dB for 8 hours daily; occupational; preventable
- Meniere disease: triad of fluctuating deafness, tinnitus, vertigo; preceded by ear fullness
- Infection: viral cochleitis (sudden SNHL); bacterial meningitis in children
- Ototoxicity: aminoglycosides (permanent), aspirin (reversible), cisplatin (permanent)
- Acoustic neuroma (vestibular schwannoma): unilateral SNHL plus tinnitus; from CN VIII vestibular portion
- Neurogenic: CVA, Chiari malformation, multiple sclerosis

Table 5.2: Causes of Hearing Loss by Location

Location	Causes	Type of HL
Outer ear	Wax, otitis externa, exostosis, atresia	Conductive
Middle ear	AOM, OME, TM perforation, otosclerosis, cholesteatoma	Conductive
Inner ear	Presbycusis, NIHL, Meniere disease, ototoxicity, acoustic neuroma	Sensorineural
Central	CVA, MS, Chiari malformation	Central

Diagnosis

- History: gives 90 percent of correct diagnosis
- Otoscopy and tuning fork tests (Rinne, Weber, Schwabach)
- PTA with air and bone conduction
- Tympanogram: assess middle ear function
- Speech audiometry: speech discrimination scores
- MRI: if asymmetric SNHL (rule out acoustic neuroma)

Treatment

- Treat underlying cause: medical and or surgical
- Sudden SNHL: corticosteroids within 2 weeks of onset
- Hearing aids: most common rehabilitation for SNHL
- Cochlear implants: severe-to-profound SNHL
- BAHA: for conductive or mixed loss

- Aspirin causes REVERSIBLE SNHL (unlike aminoglycosides which cause permanent SNHL)
- Acoustic neuroma: unilateral SNHL plus tinnitus (bilateral equals NF2)
- Otosclerosis: young females, AD inheritance, conductive HL, treat with stapedectomy
- Presbycusis: symmetric high-frequency SNHL; hearing aids are usually successful
- Meniere triad: fluctuating deafness plus tinnitus plus vertigo (NOT fixed deafness)
- Tinnitus is CENTRAL in origin (not peripheral), persists after eighth nerve section
- Sudden SNHL: treat with corticosteroids within 2 weeks
- Weber test: lateralizes to affected ear in conductive loss, to normal ear in SNHL

- Tinnitus: subjective (95 percent) vs objective (5 percent); central origin; treat underlying cause
- Presbycusis: most common cause of HL in elderly; symmetric high-frequency SNHL
- Acoustic neuroma: unilateral SNHL plus tinnitus; MRI to rule out in asymmetric SNHL
- Otosclerosis: young females; conductive HL; stapedectomy is curative
- Meniere disease: triad of fluctuating deafness, tinnitus, vertigo; ear fullness precedes attacks
- Ototoxicity: aminoglycosides and cisplatin cause PERMANENT SNHL; aspirin causes REVERSIBLE SNHL
- Sudden SNHL: emergency, treat with corticosteroids within 2 weeks
- Noise-induced HL: over 85 dB for 8 hours daily; preventable with hearing protection
- History gives 90 percent of correct diagnosis in hearing loss

Chapter 6

Common Adeno-Tonsillar Disease

Pharyngitis, Tonsillitis, Adenoiditis, and Surgical Management

Sore throat is one of the most common reasons for medical consultation. The vast majority of cases are caused by viral upper respiratory tract infections and are self-limiting. However, bacterial tonsillitis, particularly group A streptococcal pharyngitis, requires antibiotic treatment to prevent complications such as rheumatic fever and glomerulonephritis.

ANATOMY

The palatine tonsils are located in the oropharynx between the palatoglossal and palatopharyngeal arches. The adenoids (pharyngeal tonsil) are located in the nasopharynx. Both are part of Waldeyer lymphoid ring.

- Palatine tonsils: between anterior (palatoglossal) and posterior (palatopharyngeal) pillars
- Adenoids: nasopharyngeal tonsil; enlarge up to age 6, atrophy by age 16
- Waldeyer ring: palatine tonsils, adenoids, lingual tonsil, tubal tonsils

PHARYNGITIS VS TONSILLITIS

Sore throat refers to any painful sensation localized to the pharynx or surrounding anatomy. Commonest causes: pharyngitis, tonsillitis, adenoiditis, and laryngitis.

- Pharyngeal infection is the most common cause of sore throat
- Respiratory viruses are the most common cause (rhinovirus, influenza, adenovirus, coronavirus, parainfluenza)
- Streptococcus is the most common bacterial cause (Group A beta-hemolytic streptococcus)

Symptoms of acute pharyngitis and tonsillitis:

- Sore throat, dysphagia, fever, weakness, fatigability
- Poor appetite, dehydration
- Otalgia (referred via tympanic branch of CN IX)

Membranous Tonsillitis Types

- Infectious mononucleosis: EBV; teenagers or young adults; splenomegaly in 50 percent; avoid ampicillin (causes rash); Monospot test positive
- Scarlet fever: Streptococcus pyogenes; rash and strawberry tongue
- Diphtheria: rare in vaccinated populations; thick grey membrane; difficulty breathing

- Vincent angina: fusiform and spirochaete bacteria

Table 6.1: Membranous Tonsillitis Comparison

Condition	Cause	Key Features	Treatment
Infectious mononucleosis	EBV	Toxic, splenomegaly, atypical lymphocytes, risk of airway obstruction	Supportive, steroids if obstruction
Scarlet fever	Strep pyogenes	Rash, strawberry tongue	Penicillin
Diphtheria	Corynebacterium	Grey membrane, airway obstruction	Antitoxin plus antibiotics
Vincent angina	Fusiform and spirochaetes	Pharyngitis and tonsillitis	Antibiotics

COMPLICATIONS OF TONSILLITIS

- 1. Peritonsillar abscess (quinsy): most common
- 2. Airway obstruction
- 3. Otitis media
- 4. Parapharyngeal abscess
- 5. Retropharyngeal abscess
- 6. Rheumatic fever
- 7. Glomerulonephritis

Peritonsillar Abscess (Quinsy)

Accumulation of pus between the tonsillar capsule and the lateral pharyngeal wall. Usually preceded by an attack of tonsillitis. The affected tonsil is pushed medially with bulging above and lateral to it.

- Unilateral swelling and tenderness
- Very high fever, trismus, drooling of saliva
- Hot potato voice (muffled speech)
- Enlarged jugulodigastric lymph node
- Treatment: admission, IV antibiotics, incision and drainage, tonsillectomy after 6 weeks for second attack

Parapharyngeal Abscess

Rare but serious. Diagnosis involves clinical evaluation and CT scan. Treatment: admission, antibiotics, drainage. Complications include airway obstruction, internal jugular vein thrombosis, carotid artery rupture, lower cranial nerve injuries (IX, X, XI, XII), mediastinitis, and septicemia.

MANAGEMENT OF SORE THROAT

- Supportive: painkillers, antipyretics, local soothing agents, good hydration
- Antibiotics for bacterial infections: amoxicillin-clavulanic acid or cephalosporins for 10 days
- Complicated cases: admission, IV fluids, IV antibiotics, swab culture, EBV test, drainage for abscesses

TONSILLECTOMY

Indications: recurrent tonsillitis (over 7 episodes in 1 year, or over 5 per year for 2 years, or over 3 per year for 3 years), obstructive sleep apnea, febrile convulsions, second attack of quinsy, dysphagia and failure to thrive, suspicion of malignancy.

Complications: bleeding (primary under 24 hours; secondary 5-10 days post-op), tonsillar remnants, damage to nearby structures, infection at the tonsillar bed.

ADENOID DISEASE

The adenoid increases in size up to age 6 years, then slowly atrophies and completely disappears by age 16 years. Adenoid disease includes adenoidal hypertrophy (obstructing airflow or Eustachian tube) and adenoidal inflammation or infection.

- Symptoms: nasal congestion, snoring, mouth breathing, sore throat, middle ear infection, sleep apnea, voice changes
- Adenoid face: open mouth, protruded frontal teeth, high-arched hard palate
- Treatment: nasal sprays (saline, steroids, decongestants), antibiotics if infected
- Adenoidectomy: for nasal obstruction, OME, recurrent acute OM, chronic rhinosinusitis, sleep apnea

- Chronic adenoid hypertrophy leads to mouth breathing and adenoid facies
- Features: open mouth, protruded frontal teeth, high-arched hard palate
- Adenoid size peaks at age 6, atrophies by age 16
- Adenoidectomy indicated for OME, recurrent OM, nasal obstruction, sleep apnea

- Most sore throats are viral; Streptococcus is the most common bacterial cause
- Infectious mononucleosis: EBV; avoid ampicillin (causes rash); Monospot test positive
- Peritonsillar abscess (quinsy): most common complication; I and D plus IV antibiotics
- Tonsillectomy: recurrent tonsillitis, OSA, second quinsy, suspected malignancy

- Adenoid hypertrophy: peaks at age 6, atrophies by age 16
- Adenoid face: open mouth, protruded teeth, high-arched palate
- Rheumatic fever and glomerulonephritis are complications of untreated strep tonsillitis
- Referred otalgia in tonsillitis via tympanic branch of CN IX
- Parapharyngeal abscess: rare but life-threatening; can cause carotid rupture, mediastinitis

Chapter 7

Vertigo

Dizziness Types, Peripheral vs Central Vertigo, BPPV, Meniere Disease, and Vestibular Neuritis

Vertigo is the illusion of movement (typically rotational) in the absence of actual movement. It is a symptom, not a diagnosis. Vestibular disorders are frequently encountered in emergency departments and primary care. The male-to-female ratio is 1:2.7, and vertigo is three times more common in the elderly.

DIZZINESS TYPES

- Vertigo: illusion of rotational movement (vestibular)
- Presyncope: feeling of fainting (cardiovascular)
- Disequilibrium: unsteadiness without vertigo (proprioceptive, cerebellar)
- Light-headedness: vague feeling (anxiety, hypoglycemia, anemia)

PERIPHERAL VS CENTRAL VERTIGO

Table 7.1: Peripheral vs Central Vertigo

Feature	Peripheral	Central
Onset	Sudden	Variable
Nystagmus	Horizontal-torsional, unidirectional	Vertical, bidirectional, direction-changing
Visual fixation	Suppresses nystagmus	Does NOT suppress nystagmus
Hearing loss	Common	Rare
Neurologic signs	Absent	Present (diplopia, dysarthria, weakness)
Falling	Toward affected side	Variable direction

PERIPHERAL CAUSES OF VERTIGO

Mnemonic: BEAM MN

- B: Benign Paroxysmal Positional Vertigo (BPPV), most common peripheral cause
- E: Endolymphatic hydrops (Meniere disease)

- A: Associated with hearing loss (acoustic neuroma)
- M: Motion sickness or Migrainous vertigo
- M: Medications (aminoglycosides, cisplatin)
- N: Neuritis (vestibular neuritis)

Benign Paroxysmal Positional Vertigo (BPPV)

The most common cause of vertigo. Caused by canalithiasis (free-floating otoconia) in the posterior semicircular canal. Characterized by brief episodes of rotational vertigo triggered by head position changes.

- Episodes last under 30 seconds
- Latency of a few seconds after head movement
- Fatigues with repeated testing
- Dix-Hallpike test: positive (torsional nystagmus with latency and fatigue)
- Treatment: Epley maneuver (first-line), Semont maneuver, Brandt-Daroff exercises

Meniere Disease

Characterized by the triad of episodic vertigo, fluctuating sensorineural hearing loss, and tinnitus. Attacks are preceded by a sensation of ear fullness. Pathophysiology involves endolymphatic hydrops.

- Episodes last 20 minutes to several hours
- Fluctuating low-frequency SNHL
- Treatment: low-salt diet, diuretics, betahistine, vestibular sedatives
- Refractory: endolymphatic shunt, labyrinthectomy, intratympanic gentamicin

Vestibular Neuritis

Acute, prolonged vertigo without hearing loss. Likely viral in etiology. Differentiated from labyrinthitis by the absence of cochlear symptoms.

- Sudden onset of severe vertigo lasting days to weeks
- No hearing loss (distinguishes from labyrinthitis)
- Treatment: vestibular sedatives (short course), corticosteroids, vestibular rehabilitation

Acoustic Neuroma (Vestibular Schwannoma)

Benign tumor arising from the vestibular portion of CN VIII. Most common complaint is asymmetric or unilateral sensorineural hearing loss and tinnitus.

- Unilateral SNHL plus tinnitus (most common presentation)
- MRI gadolinium: gold standard for diagnosis
- Bilateral acoustic neuromas equals Neurofibromatosis type 2 (NF2)

CENTRAL CAUSES OF VERTIGO

- Migrainous vertigo: most common central cause
- Brainstem ischemia (vertebrobasilar insufficiency)
- Cerebellar infarction or hemorrhage
- Chiari malformation
- Multiple sclerosis
- Episodic ataxia type 2

CLINICAL EXAMINATION

- Otoscopic examination and bedside hearing tests (Weber and Rinne)
- Nystagmus characterization: direction, type, effect of visual fixation
- Dix-Hallpike maneuver for BPPV
- Neurologic exam: gait, Romberg, Unterberger, cranial nerves, cerebellar signs
- Head impulse test (Halmagyi): positive in peripheral vestibular loss
- HINTS exam: Head impulse, Nystagmus, Test of skew, differentiates peripheral from central in acute vertigo

TREATMENT

- Symptomatic (acute): antihistamines (meclizine), benzodiazepines (diazepam), antiemetics (ondansetron)
- Specific: BPPV to Epley maneuver; Meniere to diuretics and betahistine; Vestibular neuritis to steroids
- Vestibular rehabilitation: habituation exercises, VOR adaptation, should begin immediately
- Surgical: for refractory cases, labyrinthectomy, vestibular nerve section, endolymphatic shunt

- BPPV: most common peripheral cause; Epley maneuver is first-line treatment
- Vestibular neuritis: NO hearing loss (distinguishes from labyrinthitis)
- Acoustic neuroma: bilateral equals NF2; unilateral SNHL plus tinnitus
- Meniere triad: vertigo plus fluctuating SNHL plus tinnitus (NOT fixed HL)
- Vertical nystagmus equals always central (never peripheral)
- HINTS exam more accurate than MRI in first 48 hours of acute vertigo
- Visual fixation SUPPRESSES peripheral nystagmus but NOT central nystagmus

- Vertigo equals illusion of movement; NOT a diagnosis, always find the cause
- BPPV: most common cause; brief episodes (under 30 sec); positive Dix-Hallpike; treat with Epley
- Peripheral vertigo: horizontal-torsional nystagmus, suppressed by visual fixation
- Central vertigo: vertical or direction-changing nystagmus, NOT suppressed by fixation
- Meniere disease: triad of vertigo plus fluctuating SNHL plus tinnitus plus aural fullness
- Vestibular neuritis: acute prolonged vertigo WITHOUT hearing loss
- Acoustic neuroma: unilateral SNHL plus tinnitus; MRI with gadolinium is gold standard
- HINTS exam differentiates peripheral from central in acute vertigo
- Migrainous vertigo is the most common central cause of vertigo
- Vestibular rehabilitation should begin immediately after symptom onset

Chapter 8

Rhinosinusitis: Acute and Chronic

Classification, Pathophysiology, Diagnosis, Complications, and Management

Rhinosinusitis is an inflammatory condition involving the paranasal sinuses and nasal passages. Because rhinitis and sinusitis almost always coexist, the correct terminology is Rhinosinusitis. It is one of the most common clinical conditions, with significant impact on quality of life and healthcare costs.

CLASSIFICATION

- Acute rhinosinusitis: symptoms under 12 weeks
- Chronic rhinosinusitis (CRS): symptoms over 12 weeks with objective evidence of inflammation
- Recurrent acute rhinosinusitis: over 4 attacks per year with resolving intervals

ACUTE BACTERIAL RHINOSINUSITIS (ABRS)

Most ABRS follows a viral upper respiratory infection. The sequence is: viral URTI leads to mucosal edema leads to ostial obstruction leads to mucus stasis leads to bacterial overgrowth. The sinus most commonly involved is: Maxillary greater than Ethmoid greater than Frontal greater than Sphenoid.

- Direct healthcare cost: 3.4 billion dollars per year in the US
- 20 percent of antibiotic prescriptions in general practice are for ARS

Microbiology

Viral (95 percent of cases): rhinovirus (most common), adenovirus, RSV, influenza, parainfluenza

Bacterial:

- Streptococcus pneumoniae: 20-40 percent
- Haemophilus influenzae: 20-35 percent
- Moraxella catarrhalis: 2-10 percent
- Streptococcus pyogenes, Staphylococcus aureus (0-9 percent)
- Anaerobes: 10 percent (especially in dental origin sinusitis)

Diagnosis

Cannot be distinguished from viral in the first 3-4 days. The following clinical presentations best identify ABRS:

- Persistent symptoms over 10 days without clinical improvement
- Worsening symptoms after 5-6 days of viral URI (double-sickening)
- Severe symptoms: high fever (over 39C or 102F) plus purulent discharge or facial pain for 3-4 consecutive days
- Endoscopy: best by seeing pus draining from the middle meatus
- CT only if complications suspected, immunocompromised, or deterioration on therapy

Treatment of ABRS

- Antibiotics (10-14 days for children, 5-7 days for adults)
- Topical decongestants (oxymetazoline, phenylephrine)
- Nasal saline irrigation
- Topical corticosteroids
- Antihistamines are AVOIDED (thicken and dry secretions)
- Switch therapy if no improvement in 48-72 hours

Table 8.1: Antibiotic Guidelines for ABRS

Patient	Initial Therapy	Switch Therapy (no improvement i
Adults	Amoxicillin/clavulanate 1g BID	Levofloxacin 500 mg per day
Children	Amoxicillin/clavulanate 90 mg per kg per day BID	Cefixime or cefpodoxime plus clindamycin
Beta-lactam allergic (adults)	Doxycycline 100 mg BID	Levofloxacin

COMPLICATIONS OF ACUTE RHINOSINUSITIS

Orbital Complications (Most Common)

Spread via congenital dehiscence in lamina papyracea, direct extension, or thrombophlebitis of valveless ophthalmic veins. Most common organism: Streptococcus viridans.

Hubert (Chandlers) Classification:

- 1. Pre-septal cellulitis
- 2. Orbital cellulitis (post-septal)
- 3. Subperiosteal abscess
- 4. Orbital abscess
- 5. Cavernous sinus thrombosis

Cavernous Sinus Thrombosis

- 80 percent fatal if untreated

- Most common cause: ethmoiditis; Most common organism: coagulase-positive Staph aureus
- Contralateral eye involvement, spiking fever
- First nerve affected: CN VI (abducens)
- Nerves involved: III, IV, V1, V2
- Treatment: high-dose IV antibiotics crossing BBB (cephalosporin plus metronidazole); consider anticoagulants

Intracranial Complications

- Order of frequency: Subdural abscess greater than Brain abscess greater than Epidural abscess greater than Meningitis
- Frontal sinus: most common source of brain abscess
- Symptoms: headache greater than fever greater than altered mental status greater than purulent rhinorrhea

CHRONIC RHINOSINUSITIS (CRS)

Definition and Subtypes

An inflammatory condition involving the paranasal sinuses and linings of the nasal passages that lasts 12 weeks or longer. Diagnosis requires objective evidence of mucosal inflammation.

- CRS without nasal polyposis (CRSsNP): 60-65 percent
- CRS with nasal polyposis (CRSwNP): 20-33 percent; strongly associated with asthma and aspirin sensitivity

Epidemiology

- Second most common chronic disease in the US
- 33 million Americans affected per year; Mean age of diagnosis: 39 years; Women affected more
- Up to 20 percent of CRS patients have asthma

Risk Factors

Host factors: allergic rhinitis, asthma, aspirin-exacerbated respiratory disease, immunodeficiency, cystic fibrosis, primary ciliary dyskinesia, septal deviation, GERD.

Environmental: microorganisms, cigarette smoke, pollutants, medications (rhinitis medicamentosa)

Pathophysiology

CRS is a proliferative process with remarkable thickening of the mucosa and lamina propria. Eosinophils are the predominant infiltrative cell. IL-4 and IL-5 levels are increased, promoting eosinophil survival. Eosinophil degranulation releases destructive enzymes causing epithelial

damage and disrupting mucociliary clearance.

Key pathogenic mechanisms:

- Osteitis: bony thickening and increased density on CT
- Biofilm formation: present in 45-80 percent of cases
- Staph aureus superantigens: may play a role in nasal polyposis

Diagnosis of CRS

Requires 2 or more cardinal symptoms (nasal obstruction, PND or nasal discharge, facial pain, hyposmia or cough in children) AND documentation of mucosal inflammation (mucopurulent discharge in middle meatus, nasal polyps, or CT findings).

- Nasal endoscopy: gold standard for assessing mucosal inflammation
- CT scan: investigation of choice; indicated after failure of maximum medical therapy, before surgery, suspected complications
- MRI: reserved for tumors, skull base or orbital involvement, fungal sinusitis

Medical Treatment of CRS

Medical treatment of CRS is as effective as endoscopic sinus surgery, combined with topical nasal steroids, both in polypoid and non-polypoidal CRS.

- Saline irrigation: reduces PND, removes secretions, rinses allergens and irritants
- Intranasal corticosteroids: first-line; improve OMC patency, reduce blockage, reduce polyp size
- Systemic corticosteroids: for severe polyposis; contraindicated in DM, PUD, glaucoma, osteoporosis
- Antibiotics: controversial in CRS; macrolides may have anti-inflammatory effects
- Antileukotrienes: 50-72 percent of CRS patients respond to some degree

Surgical Treatment of CRS

Functional Endoscopic Sinus Surgery (FESS) is the standard approach. Extended surgery does not yield better results than limited surgery. Surgery should stop where pathology stops (Stammberger principle).

- Indications: failure of maximum medical therapy, complications, suspected neoplasms

Complications of CRS

- Mucocoeles: chronic expanding lesions causing bony erosion; if infected equals pyocoele
- Orbital complications (same as acute sinusitis)

- Rhinosinusitis (not sinusitis alone), rhinitis and sinusitis coexist
- ABRS: persistent over 10 days, double-sickening, or severe symptoms for 3-4 days

- Most common bacteria: S. pneumoniae (20-40 percent), H. influenzae (20-35 percent), M. catarrhalis (2-10 percent)
- First-line antibiotic: amoxicillin/clavulanate; switch if no improvement in 48-72 hours
- Orbital complications: most common; Chandler classification (1-5)
- Cavernous sinus thrombosis: 80 percent fatal; CN VI first affected
- CRS: over 12 weeks; requires objective evidence of inflammation
- CT scan is investigation of choice for CRS; MRI for tumors or fungal disease
- FESS: surgery stops where pathology stops; medical therapy equally effective with topical steroids

Chapter 9

Neck Masses

Differential Diagnosis, Congenital Anomalies, Neoplastic and Inflammatory Causes

A neck mass is a common clinical finding in patients of all age groups. The overall prevalence ranges from 10-15 percent, increasing with age after 45 years. The rule of 80 states that 80 percent of non-thyroid neck masses in adults are neoplastic, and 80 percent of these masses are malignant. In children, 90 percent of neck masses are benign.

APPROACH TO NECK MASS

Patient Age (Most Important Factor)

- Children: 90 percent benign; congenital greater than inflammatory greater than neoplastic
- Adults: Rule of 80, 80 percent of non-thyroid masses are neoplastic; 80 percent of those are malignant

Location

- Midline cystic: thyroglossal duct cyst (elevates with swallowing) vs dermoid cyst (moves with skin)
- Lateral neck congenital: branchial cleft anomalies, cystic hygroma
- Lateral neck neoplastic: lymphadenopathy, carotid body tumor, salivary gland tumors
- Supraclavicular or posterior triangle: higher malignancy risk

Duration

- Under 6 weeks: likely inflammatory or infectious
- Over 6 weeks after antibiotics: consider neoplasm, biopsy
- Presents since birth: congenital or developmental

CONGENITAL ANOMALIES

Thyroglossal Duct Cyst

Most common congenital anomaly of the midline neck. Caused by a tract of thyroid tissue along the embryologic migration from the base of the tongue to the neck. Intimately related to the hyoid bone and elevates with swallowing or tongue protrusion. May contain the patient only functioning thyroid tissue. Papillary carcinoma has been reported within these cysts.

- Treatment: Sistrunk procedure, excise cyst plus central hyoid bone plus or minus tract to base of tongue
- Thyroid function tests and ultrasound before surgery to confirm normal thyroid

Branchial Cleft Anomalies

Most common congenital masses of the lateral neck. Can present as cysts, sinuses, or fistulae. Typically soft, slow-growing, and painless, located along the sternocleidomastoid muscle. FNA should be performed before excision in the elderly to exclude cystic metastases from SCC.

- 1st branchial cyst: near ear or mandible
- 2nd branchial cyst: most common; along anterior border of SCM at hyoid level
- 3rd and 4th: rare; present as recurrent cervical abscesses
- Treatment: complete surgical excision

Other Congenital Masses

- Dermoid cyst: midline; moves with skin (not like thyroglossal duct cyst)
- Cystic hygroma (lymphangioma): lymphatic malformation; transilluminates

NEOPLASTIC CAUSES

Carotid Body Tumor (Paraganglioma)

Neuroendocrine tumor from glomus cells in carotid body at carotid bifurcation. 90 percent sporadic; 10 percent hereditary (SDH gene mutations). 10 percent of carotid body tumors are bilateral. Malignancy rate: 6-19 percent.

- Clinical: palpable mass at carotid bifurcation; Fontaine sign (horizontal mobility only)
- Functional (catecholamine-secreting): 1-3 percent; test for urinary metanephrines or VMA
- Shamblin classification: Type I (localized), Type II (partially surrounds artery), Type III (encases artery)
- Treatment: surgical excision; preoperative embolization for large tumors
- Rule out pheochromocytoma before surgery if functional

Juvenile Nasopharyngeal Angiofibroma (JNA)

Rare (0.05 percent of H and N tumors). Occurs exclusively in prepubescent or adolescent males. Most common presentation: severe recurrent epistaxis plus nasal obstruction. NEVER biopsy (risk of catastrophic hemorrhage).

- Adolescent male plus recurrent epistaxis plus nasal obstruction equals JNA until proven otherwise
- Preoperative embolization reduces blood loss significantly
- Treatment: endoscopic resection after embolization

Salivary Gland Masses

Parotid gland: 80 percent of salivary gland tumors; 80 percent are benign (pleomorphic adenoma most common). Submandibular gland: 10-15 percent of salivary tumors.

- Facial nerve identification is key during parotid surgery
- FNA for diagnosis; surgical excision for most tumors

Lymphadenopathy

Characteristics suggesting malignancy:

- Hard, irregular, fixed or immobile mass
- Over 2 cm, no response to antibiotics
- Supraclavicular location (especially left, Virchow node)
- Constitutional symptoms: fever, weight loss, night sweats (B symptoms)
- Kawasaki disease: fever plus bilateral conjunctivitis plus strawberry tongue plus lymphadenopathy

Table 9.1: Differential Diagnosis of Neck Mass by Location

Location	Congenital	Inflammatory	Neoplastic
Midline	Thyroglossal duct cyst, dermoid	Thyroiditis	Thyroid cancer, lymphoma
Lateral neck	Branchial cyst, cystic hygroma	Reactive lymphadenopathy	Lymphoma, metastatic SCC
Supraclavicular	None	TB lymphadenopathy	Metastatic (lung, GI, Virchow node)
Carotid bifurcation	None	None	Carotid body tumor

- Rule of 80: 80 percent of non-thyroid adult neck masses are neoplastic; 80 percent of those are malignant
- Children: 90 percent of neck masses are benign (congenital greater than inflammatory)
- Thyroglossal duct cyst: elevates with tongue protrusion or swallowing; Sistrunk procedure
- Dermoid cyst: moves with skin (not like thyroglossal duct cyst)
- 2nd branchial cyst: most common branchial anomaly; along anterior SCM
- FNA before excision in elderly (exclude cystic SCC metastasis)
- Carotid body tumor: Fontaine sign (horizontal mobility only); rule out pheochromocytoma
- JNA: adolescent male plus epistaxis plus nasal obstruction; NEVER biopsy
- Virchow node (left supraclavicular): metastatic (especially gastric)
- Hard, fixed, over 2 cm node not responding to antibiotics equals biopsy

Chapter 10

Chronic Otitis Media

Tubotympanic vs Atticoantral Disease, Cholesteatoma, and Complications

Chronic otitis media (COM) is a long-standing infection of part or whole of the middle ear cleft characterized by ear discharge and a permanent perforation. A perforation becomes permanent when its edges are covered by squamous epithelium and it does not heal spontaneously. COM is classified into tubotympanic (safe or mucosal) and atticoantral (unsafe or squamosal) types.

CLASSIFICATION

- Tubotympanic (safe or mucosal disease): TM perforation or pars tensa retraction; no cholesteatoma
- Atticoantral (unsafe or squamosal disease): cholesteatoma in attic, antrum, posterior tympanum, or mastoid

TUBOTYMPANIC DISEASE

Also known as mucosal disease since there is no invasion of squamous epithelium. Subtypes: active (perforation with inflammation and mucopurulent discharge) and inactive (permanent perforation without inflammation or discharge).

Etiology: sequelae of AOM; ascending infections via Eustachian tube; allergy

Organisms: Pseudomonas aeruginosa, Proteus, E. coli, Staph aureus; anaerobes (Bacteroides, anaerobic Streptococci)

Clinical: ear discharge, hearing loss; otoscopy shows perforation; mucosa may be red or edematous

Treatment: aural toilet, ear drops, systemic antibiotics, treat contributory causes (adenoids, sinusitis, allergy)

Surgery: myringoplasty plus or minus ossicular reconstruction

Pars Tensa Retraction

Related to chronic negative pressure from Eustachian tube dysfunction. Management ranges from observation to cartilage tympanoplasty. Ventilating tubes may be placed for intermediate cases.

ATTICOANTRAL DISEASE (CSOM WITH CHOLESTEATOMA)

Cholesteatoma is the presence of keratinizing squamous epithelium in the middle ear space. It erodes bone, forms granulation tissue, and produces purulent offensive discharge. It is called unsafe because of the risk of serious complications.

Organisms: Pseudomonas (48-98 percent), Staph aureus (15-30 percent), Klebsiella (15-30 percent), Proteus (10-15 percent), anaerobes (20-50 percent)

Theories of Cholesteatoma Formation

- Migration theory: squamous epithelium migrates through TM perforation
- Metaplasia theory: chronic inflammation leads to metaplasia to squamous epithelium
- Retraction pocket theory: negative pressure leads to retraction pocket and keratin accumulation
- Congenital: behind intact TM; epidermoid formation

Classification of Cholesteatoma

- Primary acquired: retraction pocket cholesteatoma (most common)
- Secondary acquired: squamous epithelium migrates through perforation
- Congenital: behind intact TM; no history of OM

Clinical Features

- Ear discharge: persistent, scanty, offensive (foul-smelling)
- Hearing loss: conductive or mixed
- Otoscopy: retraction pocket, attic perforation, white keratin debris
- Tuning fork tests plus audiogram
- Temporal bone CT: extent of disease, ossicular erosion, complications

COMPLICATIONS OF COM

Intratemporal: petrositis (Gradenigo syndrome), facial paralysis, labyrinthitis

Intracranial: lateral sinus thrombosis, meningitis, intracranial abscess

Features indicating complications:

- Pain suggests extradural, perisinus, or brain abscess
- Vertigo suggests erosion of lateral semicircular canal (fistula) and labyrinthitis
- Persistent headache suggests intracranial complication
- Facial weakness suggests erosion of facial canal
- Fever, nausea, vomiting suggest intracranial infection
- Neck rigidity suggests meningitis

- Diplopia suggests petrositis or Gradenigo syndrome

Gradenigo Syndrome

Triad of: (1) Otitis media, (2) Abducens nerve palsy (diplopia), (3) Deep facial or retro-orbital pain (CN V1 involvement). Caused by petrous apex infection (petrositis).

IMAGING

CT scan: excellent bony anatomy review; shows extent of disease, ossicular erosion, lateral semicircular canal fistula

MRI: better for dural inflammation, sigmoid sinus thrombosis, labyrinthitis, abscesses; detects residual or recurrent cholesteatoma

MANAGEMENT

- Medical: aural toilet, ear drops, systemic antibiotics for active infection
- Surgical: definitive treatment for cholesteatoma
- Canal wall up (CWU) mastoidectomy: preserves posterior canal wall; risk of residual disease
- Canal wall down (CWD) mastoidectomy: modified radical or radical; better exposure; creates mastoid cavity
- Tympanoplasty: reconstruction of TM and ossicular chain

- COM: permanent perforation plus chronic ear discharge
- Tubotympanic (safe): no cholesteatoma; mucosal disease only
- Atticoantral (unsafe): cholesteatoma present; erodes bone
- Cholesteatoma: keratinizing squamous epithelium in middle ear; foul-smelling discharge
- Pseudomonas is the most common organism in cholesteatoma (48-98 percent)
- CT: best for bony anatomy; MRI: best for soft tissue complications
- Gradenigo syndrome: OM plus CN VI palsy plus deep facial pain (petrositis)
- Vertigo in COM suggests labyrinthine fistula (lateral SCC)
- Facial weakness in COM suggests erosion of facial canal, urgent surgery
- Treatment of cholesteatoma is always surgical (cannot be cured medically)

Chapter 11

Acute Otitis Media and Otitis Media with Effusion

Pathogenesis, Diagnosis, Management, and Complications

Acute otitis media (AOM) is an acute suppurative infectious process of the middle ear space lasting under or equal to 3 weeks. It is one of the most common childhood infections and the most common reason for antibiotic prescription in children. Otitis media with effusion (OME), also called glue ear, is middle ear fluid without signs of acute infection.

ACUTE OTITIS MEDIA (AOM)

Epidemiology

- Much more common in children (Eustachian tube shorter, narrower, more horizontal)
- Peak incidence: 6-24 months
- 45-60 percent of children have at least one episode before age 5
- Pneumococcal vaccine: 42 percent decline in AOM in children under 5

Microbiology

- Bacterial: S. pneumoniae, H. influenzae, M. catarrhalis, S. aureus, Group A Strep
- Viral: rhinovirus, RSV, parainfluenza, coronavirus, adenovirus
- Rare: diphtheritic otitis, tuberculous otitis, Chlamydia trachomatis

Patient Risk Factors

- Eustachian tube dysfunction or obstruction
- Immune dysfunction
- Ineffective mucociliary clearance (ciliary dyskinesia)
- Adenoid hypertrophy, cleft palate, Down syndrome

Clinical Presentation

- Otalgia and ear rubbing
- Decreased or muffled hearing
- TM rupture with sudden relief of pain plus purulent otorrhea
- Fever (one-third to two-thirds of children)

Otoscopic Findings

- Bulging tympanic membrane
- Reduced mobility on pneumatic otoscopy
- Partial or complete opacification of TM
- Acute perforation with purulent otorrhea
- Erythema of TM

Stages of AOM

- Stage of hyperaemia: mild earache, congested TM
- Stage of exudation: fever, earache, deafness, bulging TM, mastoid tenderness
- Stage of suppuration: purulent discharge, increased deafness, decreased pain and fever
- Stage of resolution: otorrhea stops, hearing normalizes, perforation heals

Treatment of AOM

Observation for viral causes with mild symptoms (48-72 hour observation)

First-line antibiotic: amoxicillin-clavulanate

- Adults: amoxicillin 875 mg plus clavulanate 125 mg BID
- Children: amoxicillin 45-90 mg per kg per day plus clavulanate 6.4 mg per kg per day in 2 doses
- Duration: 10 days for under 2 years or recurrent AOM; 5-7 days for over or equal to 2 years with intact TM

Penicillin allergy: cephalosporins (mild reaction); macrolides (IgE-mediated)

- Second-line: fluoroquinolones
- Analgesics for pain; therapeutic tympanocentesis for refractory cases

Complications of AOM

- Chronic TM perforation (rare)
- Mastoiditis (20 percent)
- Labyrinthitis, facial paralysis (rare)
- Petrositis, meningitis, brain abscess (rare)
- Lateral sinus thrombosis, otitic hydrocephalus (rare)

OTITIS MEDIA WITH EFFUSION (OME)

Also called serous otitis media or glue ear. Defined as middle ear effusion without signs of acute infection. Often occurs after AOM but may occur with Eustachian tube dysfunction without preceding AOM.

Epidemiology

- 90 percent of children have at least one episode by age 4
- Prevalence: 10-17 percent in children 2-4 years; decreases to 3-4 percent at 6-8 years

- More common in winter; declined during COVID-19

Predisposing Factors

- Family history of otitis media
- Bottle feeding (vs breastfeeding)
- Male sex, daycare or school attendance
- Adenoid hypertrophy, exposure to tobacco smoke
- Low socioeconomic status, cleft palate, Down syndrome, obesity

Pathogenesis

Results from inflammation in response to persistent bacterial components after AOM. Eustachian tube dysfunction is a major factor. Bacterial biofilms have a major role (non-typeable H. influenzae most common in middle ear fluid). Poor clearance of biofilms, genetic predisposition, allergies, GERD, and obesity also contribute.

Clinical Features

- Caregiver concern about poor hearing and speech or language delays
- Failed hearing screening
- Hearing loss, feeling of fullness, tinnitus, balance problems
- Mostly asymptomatic apart from hearing loss

Diagnosis

- Pneumatic otoscopy: impaired TM mobility
- Type B tympanogram (flat curve)
- Air-fluid level or bubbles behind TM
- Amber-colored middle ear fluid

Management of OME

Primary interventions:

- Watchful waiting with follow-up for 3 months (if not at risk for speech or language problems)
- Tympanostomy tubes (grommets)

Other interventions: balloon dilation of Eustachian tube, adenoidectomy

Unproven or ineffective: antibiotics, oral or nasal steroids, autoinflation, antihistamines, decongestants, myringotomy without tubes

- AOM: most common childhood infection; peak 6-24 months
- Most common bacteria: S. pneumoniae, H. influenzae, M. catarrhalis
- First-line treatment: amoxicillin-clavulanate

- OME: middle ear fluid without acute infection signs; glue ear
- 90 percent of children have at least one OME episode by age 4
- Type B tympanogram equals flat curve equals effusion
- Watchful waiting for 3 months if not at risk for speech or language problems
- Tympanostomy tubes for persistent OME (over 3 months)
- Antibiotics, steroids, antihistamines are NOT effective for OME
- AOM complications: mastoiditis (20 percent), labyrinthitis, meningitis, brain abscess

Chapter 12

Stridor

Congenital and Acquired Causes, Laryngomalacia, Epiglottitis, and Croup

Stridor is a noisy breathing due to turbulent airflow through a partially obstructed airway at the level of the supraglottis, glottis, subglottis, or trachea. It is NOT a disease, it is a symptom or sign. Stridor may be inspiratory (supraglottic obstruction), expiratory (tracheal or lower obstruction), or biphasic (glottic or subglottic obstruction). Flexible and or rigid endoscopy is required to evaluate the etiology.

PATHOPHYSIOLOGY

When air passes through a narrowed flexible airway in a child, the lateral pressure that holds the airway open drops precipitously (Venturi principle), causing the tube to close. This process obstructs airflow and produces stridor. In children, the airway is narrower and more collapsible than in adults.

CONGENITAL STRIDOR

Laryngomalacia (Most Common)

The most common cause of inspiratory stridor in neonates and infants, accounting for up to 75 percent of all cases. Caused by delayed cartilage development with softening of laryngeal cartilage and omega-shaped epiglottis. Inadequate support causes enfolding of the epiglottis and aryepiglottic folds during inspiration.

- Stridor aggravated by supine position and head flexion
- Relieved by prone position and head extension
- Definite diagnosis: laryngoscopy
- Usually benign and self-limiting; improves by age 1 year (sometimes 2 years)
- Severe cases: tracheostomy until spontaneous resolution
- Surgical correction or supraglottoplasty: if tight mucosal bands or redundant arytenoid mucosa

Vocal Cord Paralysis

Probably the second most common cause of stridor in infants. Unilateral: weak cry, biphasic stridor louder when awake, improves when lying with affected side down. Usually associated with CNS abnormalities. Bilateral: severe stridor, may require tracheostomy.

Subglottic Stenosis

Narrowing below the vocal cords, the narrowest part of the upper airway in children. Can be congenital or acquired. Presents with inspiratory or biphasic stridor.

Acquired causes: prolonged intubation (most common), idiopathic, autoimmune (Wegener), GERD, trauma

Medical treatment: steroids for immature or granular stenosis; antibiotics plus steroids may help

Surgical: endoscopic dilation or balloon or laser for grade 1-2; cricotracheal resection for grade 3-4

Laryngeal Web

Caused by incomplete recanalization of the laryngeal lumen during embryogenesis. 75 percent are in the glottic area. Infants present with weak cry and biphasic stridor.

Other Congenital Causes

- Laryngeal cysts: supraglottic region; stridor, hoarseness, or aphonia
- Hemangiomas and lymphangiomas
- Vascular causes: double aortic arch, vascular ring

ACQUIRED STRIDOR

Acute Epiglottitis

Medical emergency. Caused by Haemophilus influenzae type B (Hib), now rare due to vaccination. Symptoms: severe sore throat (95 percent), odynophagia or dysphagia (95 percent), drooling, muffled hot potato voice (54 percent), high fever over 40C, toxic appearance, severe stridor with air hunger position.

- Lateral neck X-ray: thumb sign (swollen epiglottis)
- DO NOT examine the throat with a tongue depressor (may cause complete obstruction)
- Diagnosis: direct visualization via nasopharyngoscopy or laryngoscopy (preferred over X-ray)
- Treatment: secure airway (most urgent), IV fluids, IV antibiotics (ampicillin plus chloramphenicol), IV steroids, nebulized epinephrine

Laryngotracheobronchitis (Croup)

Most common cause of acute stridor in children aged 6 months to 2 years. Viral infection, most commonly parainfluenza virus. Symptoms: barking cough, stridor, wheezing, low-grade fever. Child looks ill but NOT toxic (unlike epiglottitis).

- X-ray: steeple sign (subglottic narrowing)
- Treatment: humidifiers, secure airway if compromised, IV steroids, nebulized epinephrine

Acute Laryngitis

Most commonly viral URTI. Swelling and inflammation at the vocal cord level. Seen in children under 12 years. Self-limiting. Symptoms: stridor, cough, hoarseness. Treatment: secure airway, cold stream nebulizers with epinephrine, steroids.

Bacterial Tracheitis

Secondary bacterial infection (most commonly Staphylococcus aureus) following a viral process (croup or influenza). More severe than croup.

Foreign Body Aspiration

Sudden onset stridor in a previously well child. Common objects: peanuts, small toys, coins. Diagnosis: history, X-ray (if radiopaque), bronchoscopy. Treatment: rigid bronchoscopy for removal.

Trauma

Larynx is well-protected. Classification by mechanism (thermal, chemical, physical), severity (mild, moderate, severe), and cause (accidental, assault, RTA, sports). Management: secure airway (often tracheostomy), exploration for penetrating injuries, CT scan, rule out cervical spine injuries.

Table 12.1: Epiglottitis vs Croup vs Bronchiolitis

Feature	Epiglottitis	Croup	Bronchiolitis
Age	2-6 years	6 months-2 years	Under 2 years
Cause	Hib (bacterial)	Parainfluenza (viral)	RSV (viral)
Onset	Sudden, rapid	Gradual (1-2 days)	Gradual
Fever	High (over 40C)	Low-grade	Low-grade
Toxic appearance	Yes	No	No
Drooling	Yes	No	No
Cough	Minimal	Barking	Wheezy
X-ray	Thumb sign	Steeple sign	Hyperinflation

- Stridor equals noisy breathing from partial airway obstruction; NOT a diagnosis
- Inspiratory stridor equals supraglottic; Expiratory equals lower airway; Biphasic equals glottic or subglottic

• Laryngomalacia: most common congenital stridor (75 percent); self-limiting by age 1-2 years

• Acute epiglottitis: medical emergency; DO NOT examine throat; thumb sign on X-ray

• Croup: most common cause of acute stridor in children; barking cough; steeple sign

• Epiglottitis: toxic, drooling, high fever; Croup: not toxic, barking cough, low-grade fever

• Subglottic stenosis: narrowest part of pediatric airway; acquired equals prolonged intubation

• Bilateral vocal cord paralysis: severe stridor; may need tracheostomy

• Foreign body: sudden onset in previously well child; rigid bronchoscopy for removal

• Always secure the airway first in acute stridor, then investigate

Chapter 13

Diseases of the External Ear

Congenital Anomalies, Infections, Tumors, and Otitis Externa

The external ear consists of the auricle (pinna) and the external auditory canal (EAC). Diseases range from congenital anomalies (microtia, anotia) to infections (otitis externa, perichondritis), benign conditions (exostosis, keloids), and malignant tumors (SCC, BCC, melanoma). The external ear is supplied by branches of the external carotid artery and innervated by multiple cranial nerves.

ANATOMY REVIEW

- Auricle: cartilage covered by skin; lobule has no cartilage
- EAC: S-shaped, 2.5 cm long, 0.7 cm diameter; outer 1/3 cartilaginous, inner 2/3 bony
- Blood supply: posterior auricular, superficial temporal, occipital, maxillary arteries
- Nerve supply: CN V3 (auriculotemporal), CN VII, CN X (Arnold nerve), Greater auricular, Lesser occipital
- Lymphatic drainage: parotid (anterior), mastoid (posterior), cervical (inferior)

CONGENITAL DISORDERS

Anotia and Microtia

Anotia: complete absence of the pinna. Microtia: small rudimentary pinna. The EAC, middle ear, and ossicles are also commonly malformed. Inner ear development is usually normal (different embryologic origin).

- Isolated finding in 60-80 percent of infants
- Associated syndromes: OAVS (hemifacial microsomia, Goldenhar), Treacher-Collins, trisomy 18
- Teratogenic: retinoic acid embryopathy, thalidomide
- Management: auricular reconstruction (rib cartilage) or prostheses; hearing restoration with BAHA or atresia repair

Congenital Aural Atresia

Spectrum of ear deformities involving failure of EAC development. Also involves TM, middle ear, and ossicles. Inner ear usually normal. Results from abnormal development of 1st and 2nd branchial arches and 1st branchial groove. CT temporal bone is essential for surgical planning.

Pre-Auricular Sinus

Pit or sinus tract in the pre-auricular area, between tragus and crus of helix. Forms due to failure of complete fusion between 1st and 2nd branchial arch elements. May become infected and require excision. Recurs if not completely excised.

Auricular Appendages (Tags)

Accessory auricles or skin tags anterior to the ear. Represent accessory hillocks of His. May be associated with other branchial arch anomalies.

ACQUIRED DISORDERS

Auricular Hematoma (Hematoma Auris)

Collection of blood under the perichondrium, usually from blunt trauma. Common in wrestlers and boxers (boxer ear). If not treated, leads to cartilage necrosis and fibrosis leading to cauliflower ear. Treatment: aspiration (small) or incision and drainage with pressure dressing (large).

Perichondritis

Infection of the auricular cartilage. Causes: trauma, piercing, hematoma, surgery, frostbite, extension of otitis externa. Pathology: abscess between cartilage and perichondrium leads to cartilage losing nutrition and necrosis. Treatment: antibiotics (anti-pseudomonal), debridement of necrotic cartilage, pressure dressing.

Keloids

Overgrowth of scar tissue at the site of healed skin injury. More common in Blacks. Often follows ear piercing. Treatment: intralesional steroids, surgical excision, excision plus serial steroid injections, radiation therapy.

Exostosis and Osteoma

Exostosis: bilateral, multiple, benign bony hypertrophy of the deep EAC. Seen in swimmers (cold water exposure). Usually asymptomatic unless occluding canal.

Osteoma: unilateral, solitary, pedunculated benign tumor. Usually attached to the tympanosquamous suture line near isthmus. Symptomatic if trapping cerumen or blocking canal.

Herpes Zoster Oticus (Ramsay Hunt Syndrome)

Reactivation of varicella-zoster virus in the geniculate ganglion of CN VII. Presents with severe ear pain, vesicles on pinna or EAC or TM, and involvement of CN VII and VIII.

- Triad: ear vesicles plus facial paralysis plus SNHL or vertigo
- Treatment: acyclovir plus oral steroids (10-14 days) plus corneal protection

OTITIS EXTERNA

Classification

- Acute localized otitis externa (furunculosis)
- Acute diffuse otitis externa (swimmer ear)
- Chronic otitis externa
- Otomycosis (fungal)
- Malignant (necrotizing) otitis externa

Acute Diffuse Otitis Externa (Swimmer Ear)

Infection of the EAC skin. Most common organism: Pseudomonas aeruginosa. Predisposing factors: swimming, humid climate, self-inflicted trauma (Q-tips), water retention in EAC.

Symptoms: itching then pain and tenderness then sense of fullness and hearing loss

Signs: tenderness on moving pinna or pressing tragus (pathognomonic); erythematous, edematous EAC; otorrhea; lumen may be obliterated

Treatment: topical antibiotics (ciprofloxacin plus dexamethasone drops), aural toilet, analgesics, wicks for severe edema, systemic antibiotics if extension

Furunculosis (Boil)

Acute localized infection of hair follicles in the outer 1/3 of EAC. Organism: Staphylococcus aureus. Symptoms: intense localized pain, worse with mouth opening. Treatment: local heat, analgesics, oral anti-staph antibiotics, I and D if fluctuant.

Malignant (Necrotizing) Otitis Externa

Life-threatening infection of the EAC and skull base, typically in elderly diabetics or immunocompromised patients. Organism: Pseudomonas aeruginosa. Presents with severe otalgia, purulent otorrhea, granulation tissue in EAC, and cranial nerve palsies (CN VII most common).

- Diagnosis: CT or MRI showing bone erosion; gallium scan for monitoring
- Treatment: long-term IV anti-pseudomonal antibiotics (6-8 weeks); surgical debridement; control diabetes
- Complications: skull base osteomyelitis, cranial nerve palsies, meningitis, death

Otomycosis

Fungal infection of the EAC. Most common organisms: Aspergillus niger (black colonies), Candida albicans. Often secondary to prolonged topical antibiotic use. Symptoms: itching, fullness, hearing loss. Treatment: aural toilet, topical antifungal (clotrimazole, miconazole), stop antibiotics.

MALIGNANT TUMORS OF THE EXTERNAL EAR

Squamous Cell Carcinoma (SCC)

Most common malignant tumor of the external ear. Presents as an indurated ulcer with everted margins. Regional lymph node involvement is common. Treatment: wide excision plus or minus radiotherapy; advanced cases need radical resection including parotidectomy, neck dissection, and mastoidectomy.

Basal Cell Carcinoma (BCC)

Most common malignant tumor of the auricle. Presents as a slightly raised lesion with rolled edges and a penetrating ulcer that bleeds readily. Seen on the tragus, helix border, and meatal entrance. Treatment: wide excision; advanced cases: wide excision plus radiotherapy.

Malignant Melanoma

Nodular pigmented lesion that enlarges rapidly and ulcerates. Regional LN involvement and distant metastasis common. Treatment: radical excision with lymph node dissection.

Table 13.1: Classification of Otitis Externa

Type	Cause	Organism	Key Feature
Furunculosis (localized)	Hair follicle infection	Staph aureus	Painful nodule, outer 1/3 EAC
Acute diffuse OE	Swimmer ear	Pseudomonas	Tender pinna, edema, otorrhea
Chronic OE	Persistent infection	Mixed	Chronic irritation, thickened skin
Otomycosis	Fungal infection	Aspergillus, Candida	Itching, black or white colonies
Malignant OE	Skull base infection	Pseudomonas	Elderly diabetic, CN palsies

- Microtia or anotia: associated with OAVS, Treacher-Collins, trisomy 18
- Auricular hematoma leads to cauliflower ear if untreated; I and D plus pressure dressing
- Perichondritis: anti-pseudomonal antibiotics; debridement if necrosis
- Ramsay Hunt: ear vesicles plus facial palsy plus SNHL or vertigo; treat with acyclovir plus steroids
- Otitis externa: Pseudomonas most common; tenderness on pinna movement is pathognomonic
- Malignant OE: elderly diabetics; Pseudomonas; cranial nerve palsies; 6-8 weeks IV antibiotics

- Exostosis: bilateral, swimmers; Osteoma: unilateral, pedunculated
- SCC: most common malignant tumor of external ear; BCC: most common of auricle
- Pre-auricular sinus: from incomplete fusion of branchial arches; complete excision needed
- Ear piercing can lead to keloids (especially in Blacks), perichondritis, hepatitis transmission