Ear, Nose, and Throat Disorders

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Ears

Preauricular Pits/Sinus (PPS)

- Small indentations located anterior to the helix and superior to the tragus
- Can occur unilaterally (~50%) or bilaterally (~50%)
- Prevalence ranges between 1 and 10% depending on ethnicity
- Can occur in isolation with no increased risk of hearing impairment or renal issues
- Can be associated with hearing impairment and organ malformations (i.e., kidney)
  - e.g., Branchio-oto-renal (BOR) syndrome:
    - Most common inherited syndrome causing hearing loss (autosomal dominant)
    - Clinical presentation: preauricular pits, sensorineural hearing loss, branchial cysts, renal anomalies
- PPS do not require surgical excision unless they are frequently draining or infected
- Wang et al. [14] suggest that a renal ultrasound be performed in children with ear anomalies accompanied by any of the following:
  - Other known organ malformation
  - Family history of deafness and auricular and/or renal malformation
  - Maternal history of gestational diabetes mellitus

Otitis Externa

Definition

- Inflammation of the external auditory canal (EAC) due to bacterial (most commonly *P. aeruginosa)*, or fungal infections

Clinical presentation

- Pain and tenderness with tragal pressure/pulling pinna, pruritic, erythematous and edematous EAC, debris in EAC, malodorous otorrhea

Treatment

- Pain control and anti-inflammatories
- Topical ear drops (ensured *pseudomonas* coverage)
- Keep ears dry with water precautions and/or with ear dryer
- Ears drops of solution made of 50:50 white vinegar and rubbing alcohol can provide prophylaxis (if NO tympanic membrane perforation)
- Indications for ENT referral
  - Significant debris in EAC—will require debridement
  - If unable to visualize tympanic membrane due to canal edema—patient will require a temporary ear wick

Foreign Body in the External Ear

- Beads, insects, toys, popcorn, beans, and button batteries are common ear foreign bodies (FB)
- Most foreign bodies do not require emergent removal
- Emergent removal for button batteries
- Indication for referral to ENT
  - FB wedged in canal and cannot be grasped
  - Trauma/bleeding in ear canal
  - Failed attempt at removal

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Hematoma of the Ear Pinna

- Commonly due to trauma
- Can cause avascular necrosis and permanent damage to the underlying cartilage
- Management
  - Urgent aspiration of hematoma to prevent pinna deformity (i.e., Wrestler's ear or cauliflower ear)
  - Pressure dressing applied after evacuation
  - Close follow-up to monitor for reaccumulation

Acute Otitis Media (AOM)

Background

- Signs of an acute infection associated with middle effusion and inflammation (bulging tympanic membrane)
- 80% of children have at least one AOM before 1 year of age; 90% of children have at least two AOM by the age of 3

Risk factors

- Age (6–18 months), positive family history of otitis media, day care attendance, lack of breastfeeding, exposure to tobacco smoke, pacifier use, race/ethnicity (native Americans and Eskimos are at higher risk) [11]

Common pathogen

- Bacterial: *Streptococcus pneumoniae,* nontypeable *Haemophilus influenzae, Moraxella catarrhalis, and S pyogenes (group A Streptococcus)* are the most common causes
- Viral: RSV, picornavirus, coronavirus, influenza, adenovirus

Clinical presentation

- Fever, irritability, apathy, anorexia, vomiting, diarrhea, otalgia, otorrhea, hearing loss
- Frequent night time awakening

Diagnosis

- Pneumatic otoscopy showing decreased tympanic membrane mobility remains the best method for diagnosing the presence of middle ear fluid

Management

- 2013 American Academy of Pediatrics Guidelines [8]
  - Immediate antibiotic treatment for:
    - Children < 6 months of age
    - Children with moderate –severe otalgia
    - Otalgia lasting longer than 48 h
    - Temperature > 39 °C (102.2 °F)
    - Bilateral AOM and less than 24 months of age
  - Immediate antibiotic treatment or observation with pain control
    - 6–24 months of age with unilateral non severe AOM
    - >24 months of age with unilateral or bilateral non-severe AOM

Antimicrobial therapy

- First line: Amoxicillin (90 mg/kg/day divided twice a day) × 10 days
- Second line: amoxicillin-clavulanate
  - Children who failed first line therapy
  - Children with increased risk of beta-lactam resistance
  - Beta-lactam use within past 30 days
  - Concomitant purulent conjunctivitis (likely *H. influenza*)
  - Recurrent AOM unresponsive to amoxicillin
- For patient with hypersensitivity to penicillin
  - Macrolides
  - Cefdinir, cefuroxime, ceftriaxone

Complications of AOM include

- Intratemporal: conductive hearing loss, tympanic membrane perforation, ossicular erosion, labyrinthitis, facial nerve paralysis, mastoiditis, subperiosteal abscess, petrous apicitis, sigmoid sinus thrombosis
- Intracranial: meningitis, epidural/subdural/parenchymal abscess, cavernous sinus thrombosis, otitic hydrocephalus

Suggested follow-up

- <2 years of age: 8–12 weeks after diagnosis/treatment of AOM
- <2 years of age with language or developmental delay: 8–12 weeks after diagnosis/treatment of AOM
- >2 years of age with no comorbidities/language/development delay: next routine visit

Otitis Media with Effusion (OME)

Definition: Middle ear effusion without signs of acute infection

Etiology

- After AOM (typically)
  - In presence of eustachian tube dysfunction in the absence of AOM
- Estimated up to 90% of OME will resolve spontaneously within 3 months
- 30–40% of patients will have recurrent episodes of OME
- Most common cause of pediatric hearing loss

Investigations

- Hearing evaluation
  - Children with OME >3 months

  - Children at risk for speech, language, and learning delay
- Speech language evaluation
  - In children at risk for speech, language, and learning delay

Treatment
- Observation "watchful waiting"
  - In children with OME with low risk of speech, language, learning delay with speech awareness thresholds showing hearing loss less than 20 dBs
  - Monitor every 3–6 months to ensure resolution of effusion
- Myringotomy and tympanostomy tube insertion
  - Refer to Section G for criteria
- Complication of Tympanostomy Tubes
- Refer to Section G for complications

Chronic Suppurative Otitis Media (CSOM)

Definition
- Otorrhea (>6 weeks or recurrent) from a middle ear and/or mastoid infection in the presence of a tympanic membrane perforation (or ventilation tube)

Common pathogen
- Mixed infections
  - Gram-negative bacilli (*pseudomonas, Klebsiella, proteus*, *E.coli*)
  - *Staphylococcus aureus*
  - Anaerobes

Clinical presentation
- Otorrhea, TM perforation, inflamed middle ear mucosa, conductive hearing loss

Treatment
1. Keep the ear clean and dry
   - Water precautions (avoid getting water in ear)
   - Refer to Otolaryngology if debridement required
2. Topical antimicrobial/corticosteroids (must cover *pseudomonas and MRSA*)
3. If failed topical antibiotics, consider systemic antibiotics (broad spectrum covering *pseudomonas and MRSA*)

Acute Mastoiditis (AM)

Background
- Suppurative infection of the middle ear that spreads to mastoid cavity resulting in osteitis of the mastoid bone
- May become purulent and lead to bony breakdown within the mastoid bone (acute coalescent mastoiditis)

Common presentation
- Erythema, tenderness, and edema over the mastoid bone (postauricular region)
- Protuberant ear
- Fever, adenopathy, otitis media

Imaging
- CT temporal bones (look for bony breakdown within mastoid suggestive of coalescence)

Treatment
- Immediate Otolaryngology consultation
- Systemic antibiotics (usually intravenous antibiotics required)
- Possible myringotomy (tympanocentesis/culture) and ventilation tubes (use topical antimicrobial if tube is present)
- Cortical mastoidectomy for coalescent mastoiditis

Cholesteatoma

Definition
- Squamous epithelium in the middle ear and mastoid cavities (misnomer as no cholesterol)
- Risk of leading to recurrent infections, as well as bone and soft tissue erosion

Types
- Congenital
  - Presents as a white mass, most often in the anterior–superior middle ear space with an intact tympanic membrane
- Acquired
  - Squamous epithelium enters middle ear via retraction pocket (invagination), migration through tympanic membrane perforation or iatrogenic implantation

Clinical presentation
- Conductive hearing loss
- Persistent otorrhea
- Tympanic membrane retraction pocket filled with squamous epithelial debris/crusts
- Possible whitish mass behind the TM (not always seen)

Complications
- Erosion/destruction of ossicular chain, chronic otitis media, labyrinthine fistula, intracranial complications, facial nerve paralysis

Treatment
- Otolaryngology consultation is mandatory
- Requires surgery (tympanomastoidectomy, possible ossicular chain reconstruction)
- Long-term follow-up required by Otolaryngology

Labrynthitis

Types
- Extremely rare in children
- Bacterial or viral invasion into cochlear labyrinth associated with permanent hearing loss, vestibular dysfunction, meningitis

Clinical presentation
- Vertigo, hearing loss, tinnitus, possible middle ear infection

Diagnosis
- Clinical presentation
- Obtain an urgent audiogram (sensorineural hearing loss)

Treatment
- Treat underlying infectious process
  - Bacterial: systemic antibiotics;
  - +/− myringotomy/ventilation tube if acute otitis media present

Vertigo

Definition
- Illusion of rotational, linear, or tilting movement (i.e., "spinning," "turning") of the patient or their surroundings

Types of vertigo
- Central/systemic
  - Vascular (i.e., migraines), medications/toxins, neurologic disorders (i.e., seizures), metabolic disorders (i.e., thyroid disease, diabetes)
- Peripheral (related to the ear)
  - Benign paroxysmal positional vertigo (BPPV); Vestibular neuritis due to viral infections; perilymph fistula; trauma to vestibular system; Ménière disease; cerebellopontine angle tumors

Physical exam
- Vital signs
- Head and neck: complete exam, inspect middle ear/TM, pneumatic otoscopy
- Neurologic: complete cranial nerve exam, extraocular movements/nystagmus, coordination (finger-to-nose testing), gait, tandem gait, Romberg's test, gross vision testing
- Special test: Dix–Hallpike maneuver (assesses BPPV)
- Audiometric evaluation

Treatment
- Varies based on etiology of vertigo
- Refer to Otolaryngologist if suspicious of peripheral cause of vertigo

Benign Paroxysmal Positional Vertigo (BPPV)

Definition
- Most common peripheral vestibular disorder; typically self-limiting; can be recurrent

Causes
- Spontaneous, posttraumatic, postviral
- Canalithiasis: loose floating debris in semicircular canals stimulates cupula (vestibular system)

Clinical presentation
- Brief recurrent episodes of vertigo lasting seconds to minutes, triggered by positional head movement (i.e., turning head to one side, rolling in bed to same side, looking up)

Diagnosis
- Clinical history, physical exam
- Positive Dix-Hallpike
  - To test right ear:
  - Patient sits upright with head turned 45° toward the right
  - Patient then lays flat with head extended ~30°—still looking to right
  - Observe eyes for nystagmus
  - Onset delayed ~3 s
  - Rotational
  - Self-limiting (~20 sec.)
  - Associated with subjective sensation of spinning

Treatment
- Usually self-limiting
- Refer to Otolaryngologist for Particle Repositioning Maneuver

Meniere Disease

Background
- Rare in children, but the prevalence ranges from 1.5–4% among children diagnosed with vertigo

Clinical presentation (Triad)
1. Episodic vertigo (minutes to hours)
2. Episodic fluctuating sensorineural (typically unilateral)
3. Tinnitus +/− aural fullness in affected ear

Diagnosis
- Clinical
- Obtain an audiogram at time when patient reports hearing loss

Management
- Refer to an Otolaryngologist if suspicious of Meniere's disease

Congenital Hearing Loss

- Loss of hearing present at or after birth

50% environmental	Cytomegalovirus (CMV)	
	Neonatal icterus	
	Meningitis	
	Rubella	
	Prematurity	
	Ototoxicity	
	Other infections	
50% genetic	30% syndromic	Autosomal recessive: Usher syndrome Pendred syndrome Jervell Lange–Nielsen syndrome Autosomal Dominant: Waardenburg syndrome Stickler syndrome Branchio-oto-renal syndrome Treacher–Collins syndrome
	70% nonsyndromic	Connexin mutations most common

[5]

Genetic Syndromic Hearing Loss

- More than 500 Syndromes are associated with hearing loss, most common are listed below:

Autosomal dominant
- *Waardenburg syndrome*: SNHL, hypertelorism, pigmentary abnormalities
- *Stickler syndrome*: SNHL, ocular abnormalities (myopia, retinal detachment), Marfanoid habitus, Pierre Robin Sequence
- *Branchio-Oto-Renal syndrome*: mixed hearing loss (sensorineural and conductive hearing loss), pinna deformities, preauricular or neck pits/fistulas/tags, kidney abnormalities
- *Treacher-Collins syndrome*: (mandibulofacial dysostosis): CHL (malformed ossicles), aural atresia/stenosis, zygomatic/mandibular hypoplasia
- *Others:* neurofibromatosis type II, Apert syndrome (acrocephalosyndactyly), Crouzon syndrome (craniofacial dysostosis)

Autosomal recessive
- *Usher syndrome*
  - Leading cause of deafness and blindness
  - SNHL, blindness (retinitis pigmentosa), vestibular dysfunction
- *Pendred syndrome*: SNHL, Goiter, enlarged vestibular aqueducts
- *Jervell Lange–Nielsen syndrome*: SNHL, cardiac defects (prolonged QT), syncope, sudden death

X-linked
- *Alport syndrome*: X-linked; hearing loss, progressive nephritis, occasional ocular lesions

Genetic Nonsyndromic Hearing Loss

Connexin mutations
- Most common cause of hereditary nonsyndromic hearing loss
- Connexin 26 mutations (GJB2 gene) accounts for ~80%

Universal Newborn Hearing Screening

- Implemented across all states in the USA and provinces in Canada
- Tests hearing with otoacoustic emission (OAE) screening or with an automated auditory brainstem response (ABR) shortly after birth (usually before neonate leaves the hospital)
- Any infant who fails the initial screen should be referred to an audiologist for a full evaluation no later than 4 months of age
- For all children in whom hearing loss is established by full audiologic evaluation, intervention must begin as soon as possible and no later than 6 months of age

Pediatric Audiometric Testing

Evoked Otoacoustic Emission (OAE)

- OAE detects the sound coming from the cochlea in response to clicks or tones
- OAE affected by external or middle ear debris (high false positive rate)
- Used for all ages
- No infant cooperation is required

Auditory Brainstem Response (ABR)

- ABR measures the electroencephalographic waveform response from the vestibulocochlear nerve to higher central nervous system auditory centers
- ABR minimally affected by external or middle ear debris
- Can be used at any age
- Patient must be asleep, or very still—may require sedation
- Often used to confirm abnormal OAE results

Testing Methods

Behavioral Observation Audiometry (BOA)

- Birth—6 months of age
- Sound presented via speakers. Skilled examiner observes for patient response (i.e., startle or head turning towards sound)
- Grossly assessed auditory thresholds of "better" ear (tests both ears at same time)

Visual Response Audiometry (VRA)

- 6 months—3 years of age
- Toddler encouraged to look for auditory stimulus (i.e., lights, toys, motion for reinforcement)
- Each ear may be tested individually; potential to provide complete audiogram

Play Audiometry

- 3–5 years of age
- Child performs tasks in response to auditory stimulus (e.g., pick up a block and place in the bucket when you hear the beep)
- Each ear tested individually; frequency specific

Conventional Audiometry

- 4–6 years of age and older
- Child instructed to push a button or raise hand when a tone is heard
- Complete audiogram; ear specific; frequency specific

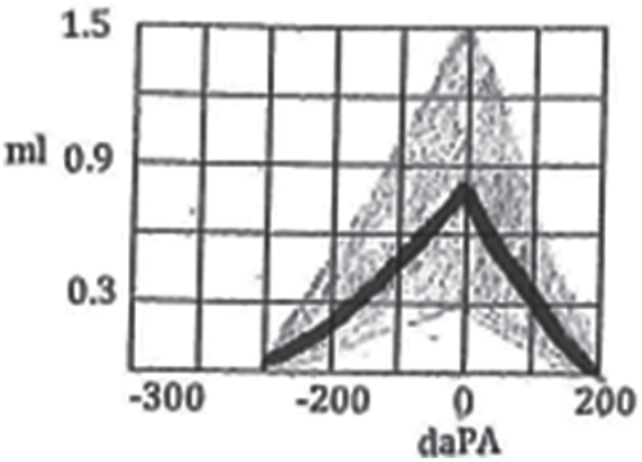
Hearing Loss Classification

- Classified by hearing threshold levels (may vary slightly based on sources)

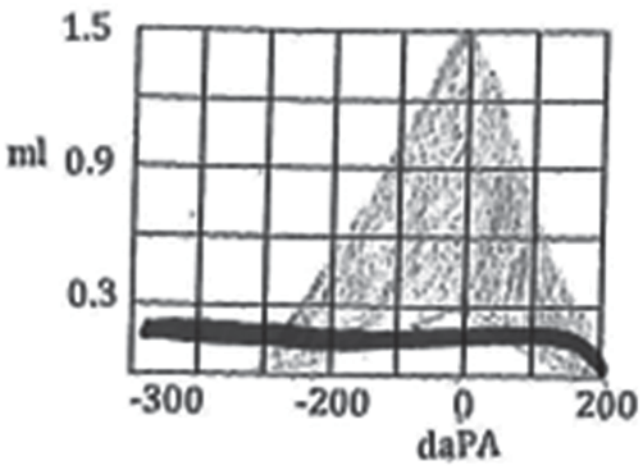
  Normal: <91 dB
  Mild: 20–40 dB
  Moderate: 41–55 dB
  Moderate–severe: 56–70 dB
  Severe: 71–90 dB
  Profound: 91 dB

Tympanometry

- Age: all ages except newborn
- Detects the mobility of TM and external auditory canal volumes
- Normal canal volumes ranges between 0.2 and 1.5 ml
  - Type A
    - Normal peak between −150 and +50 dekapascals

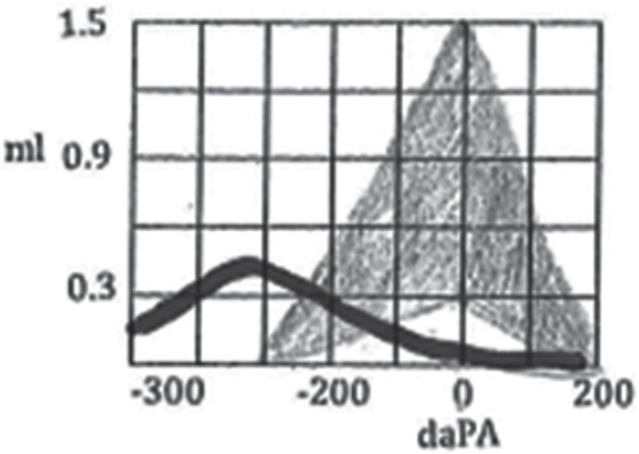


  - Type B
    - Flat, no peak



- Suggestive of:
  - Middle ear effusion (normal to low volumes)
  - Tympanic membrane perforation (high-canal volumes)
  - Patent ventilation tube (high-canal volumes)

– Type C
  - Peak negatively shifted (< −150)
  - Suggestive of a retracted tympanic membrane or Eustachian tube dysfunction



Patterns of Hearing Loss

Interpreting an Audiogram Y-axis = hearing level in decibels (dBs) or the "loudness" of sound
X-axis = frequency of sound presented measured in Hertz (low pitch to high pitch)
"x": Responses from left air conduction line
">": Responses from left bone conduction
ABG: difference between air conduction and bone conduction lines

Three Main Types of Hearing Loss

1. Conductive hearing loss (CHL)
   – Normal bone conduction threshold with abnormal air conduction thresholds
   – Presence of an air-bone gap (ABG)
   – Indicative of a middle ear issue, for example, abnormalities with the tympanic membrane, ossicles, or middle ear space (i.e., effusion; Fig. 1)
2. Sensorineural hearing loss (SNHL)
   – When the air conduction is the same as the bone conduction with both showing abnormal hearing thresholds, this is suggestive of an inner ear issue resulting in sensorineural hearing loss (e.g., damage to cochlear, neural pathways, etc.)
   – No air–bone gap (Fig. 2)

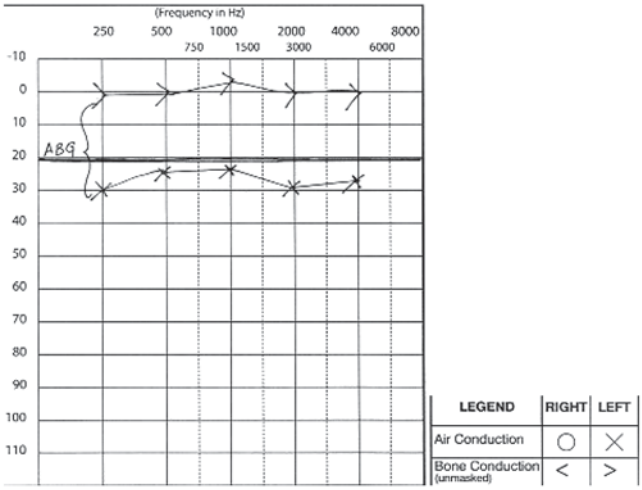


Fig. 1 Mild conductive hearing loss

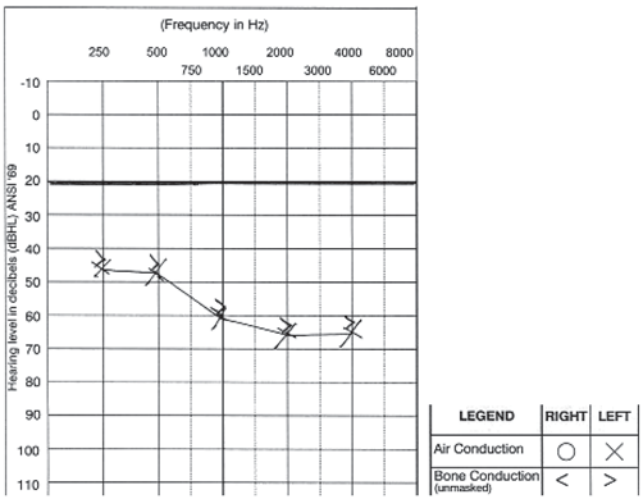


Fig. 2 Moderate to moderate–severe sensorineural hearing loss

3. Mixed hearing loss (CHL + SNHL)
   – Presence of conductive hearing loss and sensorineural hearing loss at same time (Fig. 3)

Common Clinical Scenarios

- Tympanic membrane perforation (Fig. 4)
  – Audiometric findings
    - ABG
    - Flat tympanogram
    - High-canal volumes
    - Mild conductive hearing loss
- Middle ear effusion (Fig. 5)
  – Audiometric findings
    - ABG
    - Flat tympanogram
    - Low or normal canal volumes
    - Mild conductive hearing loss

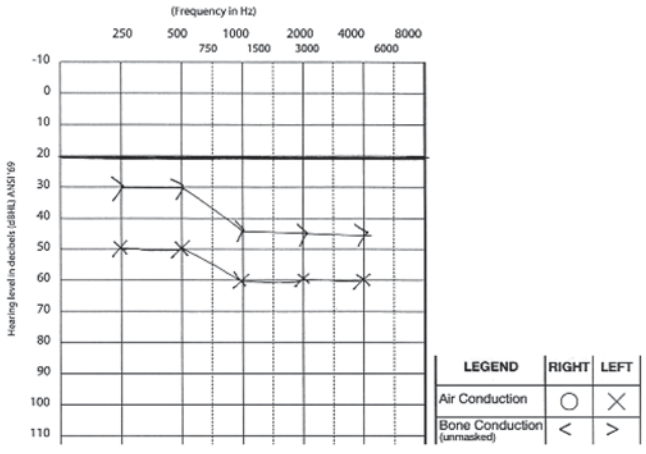


Fig. 3 Mild to moderate sensorineural hearing loss with ~20 dBs conductive hearing loss

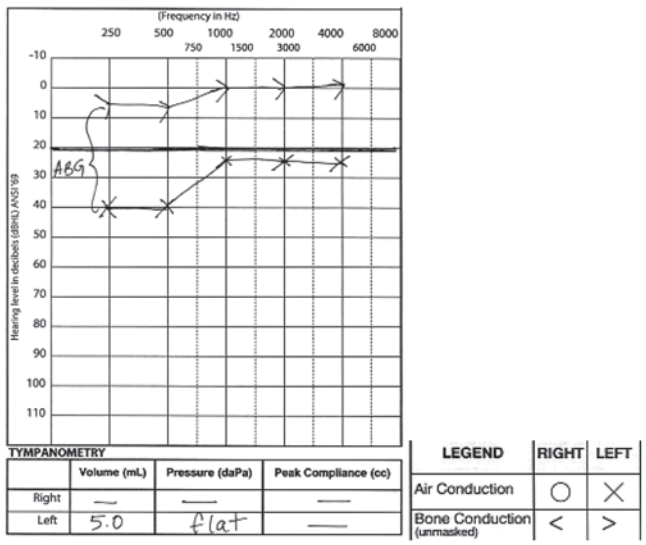


Fig. 4 Tympanic membrane perforation

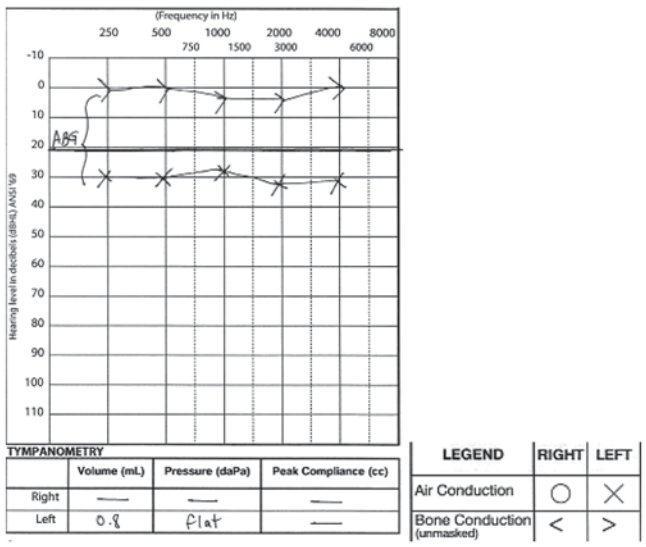


Fig. 5 Audiogram of middle ear effusion

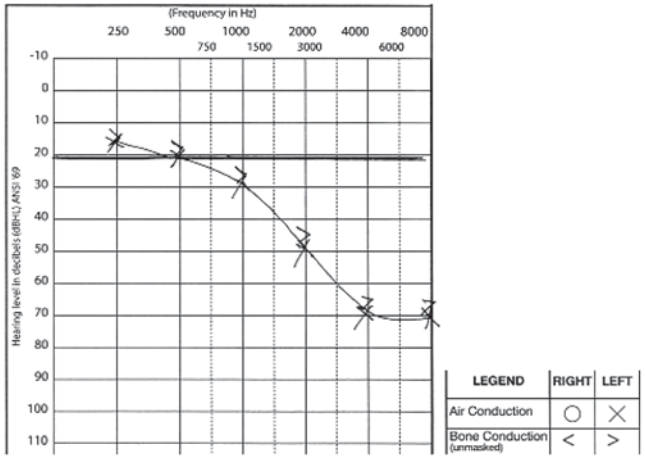


Fig. 6 Audiogram of ototoxicity

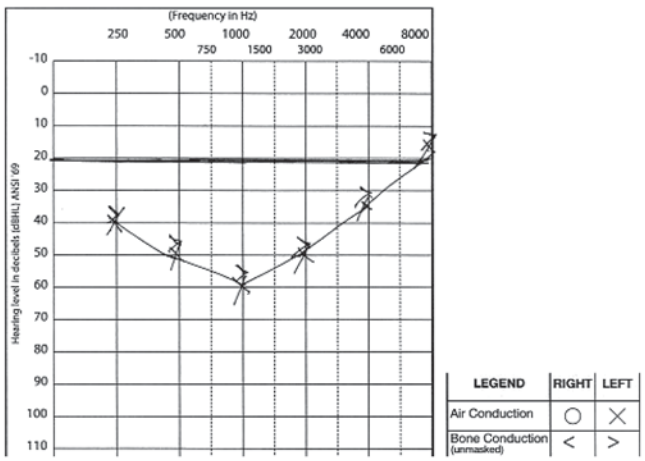


Fig. 7 Audiogram of hereditary hearing loss

- Ototoxicity (Fig. 6)
  - Ototoxic medications cause hearing loss by damaging the hair cells within the cochlea resulting in sensorineural hearing loss, primarily in the high frequencies
  - Most commonly caused by cisplatin/carboplatin
  - Audiometric findings
    - High-frequency sensorineural hearing loss (moderate to moderate-severe)
    - No ABG
    - Normal tympanogram and volumes (typically)
- Hereditary hearing loss (Fig. 7)
  - Cookie bite (U-shape) pattern of sensorineural hearing loss
  - No ABG, normal tympanogram, normal canal volumes

Clues to Hearing Loss in a Child Visit

- Speech delay
- Social and behavioral challenges

- A child asking people to repeat themselves, not hearing instructions
- Listening to loud television or music

Sound Amplification Devices

- Early identification and intervention is required to maximize hearing and speech development, as well as achieving developmental milestones
- Refer to an Otolaryngologist when an abnormal hearing screen is identified
- Hearing interventions are dependent on type of hearing and severity of hearing loss

Hearing Aids

- Non-implantable external hearing device that amplifies frequency specific sounds
- Used for unilateral or bilateral CHL, SNHL, and mixed hearing losses
- Wide variety available depending on hearing needs and preferences
- Fitting and programming process is complex and completed by an audiologist

Bone-Anchored Hearing Aid (Osseointegrated Auditory Implant)

- Titanium implant surgically placed in mastoid bone behind the ear
- Sound processor is placed externally on the implant and conducts sounds via bone contact and vibration
- Primarily used in patients with unilateral or bilateral CHL with congenital ear malformation (i.e., atresia, canal stenosis)

Cochlear Implants

- Generally, it converts sound to electrical signal which stimulates cochlear nerve
- External component captures sounds and converts to electrical signal
- Internal component delivery frequency specific electrical signal to cochlear nerve
- Multiple cochlear implant devices are available depending on hearing loss pattern and patient preferences
- Cochlear implant criteria is very specific and includes a multidisciplinary teams (i.e., otolaryngologist, speech pathologist, audiologist, social worker, psychologist, etc.)
- In general, indicated for children with pre- or postlingual severe to profound bilateral high frequency SNHL
- Fitting and programming is a complex process performed by a specialized cochlear implant audiologist and requires multiple audiology visits

Nose and Nasopharynx

Choanal Atresia

Background
- Congenital obstruction of the choana (posterior nasal aperture—connects nasal cavity to nasopharynx)
- It may be membranous, bony, or mixed (CT scan of the head can help identify type of atresia)
- Unilateral or bilateral 2:1 ratio
- Can be associated with syndromes (e.g., CHARGE)

Clinical presentation
- Bilateral
  - Severe respiratory distress at birth; cyclical cyanosis—pink with crying, cyanotic when not crying
  - Requires immediate oral airway or intubation; refer to Otolaryngology once airway is secured for surgical repair in first few days of life
- Unilateral
  - Identified at birth due to inability to pass 6Fr catheter, or later in childhood with unilateral symptoms of rhinorrhea and decreased nasal patency
  - Surgical repair typically around 4 years of age

Epistaxis

Background
- In children, 90% of epistaxis occurs from anterior septum (Kiesselbach's plexus)
- Posterior epistaxis is rare in children
- Most common causes are: trauma (i.e., nose picking), mucosal irritation and drying, foreign body, and medications (e.g., nasal steroids)

Other causes
- Tumors, e.g., juvenile nasopharyngeal angiofibroma (JNA) (occurs in pubescent males)
- Vascular malformation: Osler–Weber–Rendu syndrome (+ family history)
- Bleeding diathesis: von Willebrand disease, leukemia, or liver disease

Management
- Usually self-limiting with application of constant pressure for 5 min by squeezing sides of the nose shut
- Discourage nose-picking/rubbing
- Avoid mucosal dryness—humidifier in bedroom; apply small amount of nasal lubricant to anterior septum
- If severe, will need IV access, formal nasal packing, +/− airway management, +/− hemodynamic resuscitation
- Refer to an Otolaryngologist if suspicious for FB, tumor, recurrent epistaxis, or severe epistaxis

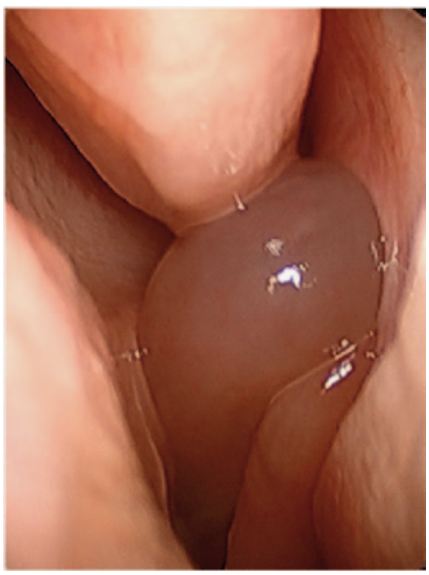
Allergic Rhinitis

- 1/3 of patient with allergic rhinitis have asthma
- Common allergens: pollens, animals (cats, dogs), dust mites, molds, etc

History
- Timing of symptoms: seasonal versus perennial
- Food hypersensitivities, comorbidities (e.g., asthma), fatigue

Symptoms
- Nose: sneezing, itching, congestion, clear rhinorrhea
- Eyes: itchy red watery eyes
- Ears: aural fullness (effusion)
- Face: frontal or periorbital headaches
- Larynx: scratchy throat, cough

Physical examination
- Eyes: dark skin under eyes ("allergic shiners"), periorbital puffiness
- Ears: effusions
- Mouth: mouth breather ("adenoid facies")
- Lungs: wheeze (associated with asthma or reactive airways)
- Nose: clear rhinorrhea, congested nasal mucosa and turbinates, transverse crease on nasal dorsum (suggestive of "allergic salute" from chronic nose rubbing), nasal polyps (rare in children)
  - if nasal polyps identified, consider testing for cystic fibrosis)



← Nasal Polyp

Diagnosis
- Skin allergy testing (e.g., scratch test, prick test, intradermal test)
- In vitro allergy testing indicated when unclear skin test results, risk of anaphylaxis, or presence of a skin disorder (i.e., dermatographia)
  - Radioallergosorbent test (RAST)
  - Enzyme-linked Immunosorbent Assay (ELISA)

Management
- Allergen avoidance
- Intranasal treatments
  - Nasal saline irrigations
  - Nasal corticosteroids (takes up to 3 weeks for maximal benefit)—first line [3]
  - Most effective maintenance therapy for allergic rhinitis
  - Nasal decongestant
  - Approved for patients 6 years of age or older
  - Limit use to 3–5 days to prevent rebound congestion
  - Use only if associated with respiratory distress due to nasal obstruction
- Cromolyn sodium nasal sprays—less effective than nasal steroid
- Antihistamine nasal spray

Systemic therapies
- Oral antihistamines
  - First generation oral antihistamines, for example, diphenhydramine (Benadryl), chlorpheniramine, and hydroxyzine are not recommended in children [12]
  - Second generation oral antihistamines (i.e., loratadine, cetirizine) have been approved for patients over 6 months of age
- Oral decongestants (not recommended in children)
- Oral antileukotrienes

Common Cold

- Self-limiting viral infection of the upper respiratory tract
- Typically, symptoms peak 2–3 days after onset then improve; the associated cough may linger up to 3 weeks

Epidemiology
- Most common in children 6 years of age or younger who on average experience six to eight colds annually
- Adolescents develop 4–5 per year
- Risk factors
  - Lack of previous exposure
  - Explores their environment with concomitant poor hygiene
  - Day care

- Seasonality
  - Cold season occurs between fall and spring
  - Early fall: rhinovirus increase
  - Late fall: parainfluenza viruses increase
  - Winter: RSV and coronavirus
  - Spring/summer: decrease in rhinovirus and enterovirus
- An effective vaccine for the common cold is unlikely

Signs and Symptoms
- Varying degrees of: sneezing, nasal congestion, rhinorrhea, sore throat, cough, low grade fever, headache, and malaise

Virology
- Rhinoviruses
  - Most common
  - Highest in early fall (September) and early spring (March/April)
- Parainfluenza viruses
  - Highest in late fall (October/November)
  - Manifest as croup in younger children and common cold in older children
- Respiratory syncytial virus (RSV)
  - Highest in winter months
  - Causes bronchiolitis in infants and young children
- Influenza viruses
  - Highest in winter months (along with RSV)
  - May manifest as febrile respiratory illness involving the lower respiratory tract, fatigue, muscle aches
- Adenoviruses
  - Present, but to a lesser degree during the fall/winter months
  - May manifests as pharyngoconjunctival fever, injected palpebral conjunctivae, Watery eye discharge, erythema of the oropharynx, fever
- Enteroviruses
  - Present during summer months

Treatment
- Colds self-limiting and treatment is supportive in nature
- Antibiotics have no role in the absence of a bacterial infection

Nasal Trauma

Background
- Nasal fractures are most common facial fracture in children (followed by mandible)
- Most commonly secondary to falls, sporting collisions, motor vehicle accidents

Presentation
- External nasal deformity, nasal obstruction, epistaxis, anosmia, septal deviation, edema, bruising

Assessment
- PALS, r/o injuries to: c-spine, CNS, chest, orbit/vision problems, midface stability, malocclusion, presence of telecanthus, cerebrospinal fluid leak, etc
- Nasal X-rays not useful
- Must evaluate for septal hematoma/abscess
  - Clinical presentation
  - Boggy asymmetrical swelling of the nasal septum not responsive to topical vasoconstriction
  - Management of nasal hematoma
  - Requires urgent drainage by an Otolaryngologist +/− bolster dressing to prevent nasal cartilage necrosis

Management
- If cosmetic deformity +/− functional issues (e.g., decreased nasal patency) refer to Otolaryngology for reduction of nasal fracture
  - Reduction of fracture is performed within 7–10 days of trauma

Sinuses

Acute Rhinosinusitis

Definitions
- Sinusitis: mucosal inflammation of paranasal sinuses typically caused by viral illness
- Acute bacterial rhinosinusitis (ABRS): sinusitis secondary to bacterial infection
- Acute: <90 days

Risk factors
- Upper respiratory tract infection
- Day care
- Allergic rhinitis
- Anatomic anomalies (e.g., septal deviation)

Presentation
- Congestion, purulent rhinorrhea, tenderness over sinuses

Clinical feature	Viral rhinosinusitis	Bacterial rhinosinusitis
Fever	Absent or occurs early (first 24 h)—low grade, resolves 2 days	Present, > 39 C (102 °F) × 3 days, may develop or recur days 6–7 of illness
Nasal discharge	Peaks days 3–6, then improves	Fails to improve or worsens
Cough	Peaks days 3–6, then improves	Fails to improve or worsens
Ill-appearance	Absent	If severe, or complicated
Severe headache	Absent	If severe, or complicated
Clinical course	Peaks days 3–6, then improves	> 10 days, without improvement

- Virology/microbiology
  - Viruses: rhinovirus, parainfluenza, influenza, adenovirus
  - Bacteria: *s. pneumoniae*, *H. influenzae*, *Moraxella catarrhalis*
  - Risk for antimicrobial resistance [4]
    - Age < 2 years, daycare antibiotics in past month, hospitalization within 5 days

Treatment
- Over the counter cold medications or decongestants (either systemic or intranasal) are not recommended for children under under twelve years of age
- Supportive therapies: hydration, saline nasal rinses, acetaminophen/ibuprofen

Treatment of ABRS [13]
- Saline nasal rinses
- Antibiotics
  - First line: amoxicillin/clavulanic acid 45 mg/kg divided BID × 10–14 days
  - If at risk of resistance (see above)—90 mg/kg divided BID × 10–14 days
  - Third generation cephalosporins if penicillin hypersensitivity
- Surgery
  - No role in ABRS, unless evidence of complication (i.e., orbital or intracranial)
- Monitor for complications
  - Orbital
  - Preseptal cellulitis, orbital cellulitis, subperiosteal abscess, orbital abscess, cavernous sinus thrombosis
  - Intracranial
  - Meningitis, epidural abscess, subdural abscess, parenchymal abscess, etc
  - Osteomyelitis (typically of frontal bones)

Imaging
- CT scan of the sinuses is only indicated if:
  - Suspicious for sinusitis complications (e.g., orbital or intracranial)
  - Failure of antibiotic treatment × 48 h
  - Immunocompromised patient
- Findings: Opacification of sinuses, mucosal thickening, air-fluid levels
  - Note: These findings are also present with the common cold

Chronic Sinusitis

Definition
- Persistence of symptoms > 12 weeks
- Symptoms include: nasal congestion, facial pressure, nasal obstruction, rhinorrhea/postnasal drip, altered sense of smell

Risk factors
- Young age (developing immune system), URI, ciliary dysfunction, allergic rhinitis, GERD, immune deficiency, cystic fibrosis

Microbiology
- Aerobes: *S. pneumoniae*, *M. catarrhalis*, *H. influenzae*, *S. aureus*, *pseudomonas*
- Anaerobes: *peptococcus, peptostreptococcus, bacteroides*

Diagnosis
- Clinical diagnosis (imaging not required for diagnosis)
- Plan X-ray films are generally not helpful
- CT scan indicated when:
  - Failed medical management and surgical intervention is being considered

Treatment
- Medical management
  - Saline nasal rinses
  - Antibiotics: amoxicillin-clavulanic acid × 3–4 weeks
  - Topical nasal steroids
  - Consider treatment of GERD if suspicious
- Surgical
  - Only considered if failure of long-term medical management
  - Adenoidectomy is first line of surgery
  - If persistent symptoms following adenoidectomy and continues to fail medical management, may consider functional endoscopic sinus surgery (maxillary antrostomy and ethmoidectomy)
- Ancillary tests
  - If failed medical management consider allergy testing if suspicious for allergies
  - If negative, consider workup for primary immunodeficiency disorder if suspicious

Frontal Sinus Trauma

- Rare in children as frontal sinuses begin forming around 5–6 years of age
- Associated with high impact injury—must rule out c-spine injuries and intracranial injury
- May present with: forehead lacerations or swelling, palpable frontal defect, pain, epistaxis, cerebrospinal fluid leak
- CT scan optimal for identifying fractures; MRI considered in addition to assess intracranial involvement
- Consult Otolaryngology if presence of frontal sinus fracture for further management
- Conservative or surgical depending on fracture pattern
- Consult Neurosurgery if suspicious for intracranial involvement

Throat

Pharyngitis

Etiology
- Infectious (most common), allergy, GERD [6]
- Viral (most common)
  - Rhinovirus, coronavirus, adenovirus, HSV, EBV, coxsackievirus
  - Usually associated with symptoms of cough, sneezing, rhinorrhea, low grade fever
- Bacterial (*streptococci, pneumococci, H. influenzae*)
  - *Group A b-hemolytic streptococcus* (GABHS)—most common bacterial cause
  - Usually associated with symptoms of high-grade fever, tonsillar/palatal petechiae, exudative tonsils, tender lymphadenopathy. Rarely seen with cough or rhinorrhea
  - GABHS pharyngitis should be treated to reduce risk of rheumatic fever, and scarlet fever
  - Other bacterial causes: syphilis, pertussis, gonorrhea, diphtheria

Symptoms
- Sore throat, pain with swallowing, ear pain (referred), malaise, fever, oropharyngeal erythema, cervical lymphadenopathy pharyngeal

Diagnosis
- Based on history and physical exam
- Throat cultures
- GABHS rapid antigen test
- Monospot test (EBV)

Treatment
- Ensure airway safety
- Supportive
  - Hydration, humidity, analgesia
- Antibiotics if bacterial infection suspected (confirm with cultures)

Peritonsillar Abscess

Definition
- Peritonsillar space defined
  - Space between the palatine tonsil, superior constrictors, tonsillar pillars

Etiology
- More common in adolescent
- Spread of infection from tonsil
- Pathogens: Aerobes (*S. pyogenes*, *S. aureus*, *Haemophilus influenzae*, and *Neisseria* species) and anaerobes

Clinical presentation
- Sore throat, painful swallowing, uvular deviation to contralateral side (medialization), trismus, asymmetrical swelling on soft palate, "hot potato" voice, fevers, referred otalgia
- Symptoms are typically present for at least 3 days before abscess is formed

Diagnosis
- History and physical examination
- CT for atypical cases or if concerns for retropharyngeal/parapharyngeal space involvement

Management
- Surgical incision and drainage
- Antibiotic therapy (penicillin or clindamycin)
- Two or more PTA may require a tonsillectomy (bilateral) in the future once infection resolves
- "Quinsy tonsillectomy"—tonsillectomy at time of infection may be considered in younger children

Retropharyngeal Abscess

Definition
- Space between pharyngeal constrictors and alar fascia (skull base to mediastinum)

Etiology
- Infection most common in children
- Spread of infection from tonsils, sinuses, and/or nasopharynx

- Polymicrobial flora (most common: *staphylococcus aureus, Streptococcus species, and anaerobes*)

Clinical presentation
- Fevers, "hot potato voice," painful swallowing, drooling, decreased neck range of motion (typically limited neck extension), possible airway compromise/stridor if severe

Diagnosis
- Lateral neck radiograph: abnormally increased thickness of the prevertebral soft tissue (greater than half thickness of the adjacent vertebral body)
- CT scan with contrast useful for localization, extension, phlegmon or abscess

Treatment
- Airway management if required and/or ongoing airway monitoring
- Hydration and analgesia
- Antibiotics (may consider third generation cephalosporin, clindamycin, or ampicillin/sulbactam for first line)
- Surgical drainage indicated when failed medical management, well-defined rim-enhancing abscess, systemically ill, and/or airway compromise

Retropharyngeal abscess	Peritonsillar abscess
<6 years old	Adolescent
Fever, throat pain, neck stiffness	Fever, throat pain, trismus
Purulence of retropharyngeal lymph node	Purulence of tonsillar fossa
May need imaging studies	Usually diagnosed clinically

Mouth and Oropharynx

Aphthous Ulcers

- Aphthous ulcers are the most common oral ulcer
- Etiology: idiopathic (most common), others causes include immune disorders, infections, hormonal cause, stress, trauma, nutrition
- Painful white ulcers on keratinized gingival surrounded by erythematous border
- Types
  - Minor: most common, <1 cm in diameter, painful, burning/tingling prodrome
  - Major: more painful, 1–3 cm in diameter, 1–10 ulcers at one time, scarring potential
  - Herpetiform: multiple small ulcers (1–3 mm in diameter)
- Sutton's disease: recurrent aphthous ulcers (major type)

Treatment
- Observation (self-limiting course)
- May also consider: analgesia, anti-inflammatories, antibiotics if superinfected, antivirals

Herpangina

- Pathogen: Coxsackie A virus
- Symptoms
  - High-grade fevers, rapid onset of symptoms, fatigue, decrease appetite, possible rash
  - Must have—small (1–2 mm) vesicular or ulcerative lesions surrounded by erythematous halos located on tonsillar pillars, palate, or buccal mucosa
- Diagnosis by clinical history and physical exam

Treatment
- Observation (self-limiting around 5–6 days), oral hygiene, hydration, and analgesics

Hand-Foot-Mouth Disease (HFMD)

- Most common cause is Coxsackievirus A16
- Clinical presentation
  - Low grade fever
  - Vesicles in the anterior and posterior oropharynx and may progress to ulceration
  - Maculopapular, vesicular, or pustular rash on the hand, feet, buttocks and groin
  - Most cases are mild and resolve in 3–5 days

Gingivostomatitis

- Pathogen: HSV-1 (primary infection or reactivation)
  - Primary most common in seronegative children
- Clinical presentation: small painful ulcerative vesicles with erythematous base and gray cover; difficulty swallowing, fever, malaise, cervical lymphadenopathy
- Resolution occurs in 1–2 weeks
- Reactivation is not associated with systemic symptoms
- Diagnosis: history and physical exam; viral cultures, DNA hybridization
- Treatment: supportive, oral acyclovir for infections, consider acyclovir for prophylaxis if immunocompromised

Ankyloglossia

- Abnormally short frenulum limiting effective tongue mobility

- In infants, if severe, may present with suckling difficulties, painful latch (if breastfeeding)
- In older children, may result in speech articulation issues, social mechanical issues (i.e., difficulty licking an ice cream cone, keeping teeth clean, playing wind instruments, "French" kissing)
- Surgical intervention indicated for problematic symptoms

Mucocele

- Painless, bluish submucosal lesion appearing on the lower lip
- Typically, secondary to trauma (i.e., biting lower lip)
- Can slowly grow in size
- Treatment: observation if not bothersome; surgical excision

Parotitis

Etiology
- Salivary stasis, obstruction, retrograde bacterial migration, idiopathic
- Bacteria: *S. aureus* (most common), *streptococcus viridans*, *H. influenzae*, *S. pyogenes*, *E. coli*
- Viruses: HIV, mumps, influenza, coxsackie
- Recurrent parotitis of childhood
  - Unknown etiology
  - Episodes occur every 1–3 months
  - May alternate sides
  - Typically resolves spontaneously
  - No antibiotic therapy needed unless presence of systemic symptoms

Symptoms
- Tender, red, warm parotid gland
- Purulence at Stensen's duct with "milking" of gland

Diagnosis
- History and physical exam
- Cultures of purulent discharge to help guide antibiotic therapy

Treatment
- Conservative: rehydration, warm compresses, parotid massage, sialogogues
- Antibiotics (based on cultures)
- If no improvement with above treatment, consider parotid imaging (CT or US)

Cleft Lip and Palate

Epidemiology
- Second most common malformation (after clubfoot)
- Cleft lip and palate: 1/1000 births
- Cleft palate: 1/2000 births
- Cleft lips (+/− cleft palate) and isolated cleft palate occur in distinct genetic lines
- Higher prevalence in Asians and Native Americans
- Cleft lip: males > females
- Isolated cleft palate: females > males

Risk factors
- Teratogens (ethanol, thalidomide)
- Maternal diabetes
- Amniotic band syndrome

Genetic evaluation
- 8% of isolated cleft palates are associated with a syndrome
- Over 200 syndromes associated with CL/CLP, most common include:
  - Sticklers: CP, retinal detachment, cataracts
  - Treacher Collins Syndrome: CP, midface hypoplasia, eyelid colobomas, ossicular abnormalities
  - Apert syndrome: CP, acrocephaly, fused digits, stapes fixation

Feeding difficulties
- Infants experience difficulty with "seal"—often requires specialized nipple (i.e., Mead–Johnson cross-cut; McGovern's nipples)
- Often requires feeding in more upright position with frequent rests and burping

Otologic disease
- Increased risk of developing eustachian tube dysfunction resulting in OME with CP/CLP
- Often requires myringotomy/ventilation tubes

Timing of surgical intervention
- A cleft lip generally is surgically repaired between the ages of 10 and 12 weeks
- "Rule of tens"—10 pounds, 10 weeks old, and hemoglobin of 10.0 g/dL (100.0 g/L)
- A cleft palate usually is repaired between 9 and 12 months of age

Follow for
- Difficulty in feeding and growth
- Recurrent ear infections/possible hearing loss

- Dysfunctional speech and communication (i.e., velopharyngeal dysfunction)
- Dental problems
- Social struggles because of the child's appearance

Robin Sequence (RS)

- Sequence defined as: micrognathia, cleft palate, and glossoptosis
- Occurs in isolation, or with associated syndrome (i.e., trisomy 18 or Stickler syndrome)
- Infants with RS are at high risk to develop respiratory distress and potentially have "difficult airways" given anatomy. These infants require close airway monitoring in the postnatal period
- Management of respiratory distress in RS
  - Prone positioning
  - Place suture at tip of tongue and pull tongue forward
  - Intubate if needed. If unable to intubate, place a laryngeal mask airway (LMA)
  - If patient fails extubation, patient may require:
    - Mandibular distraction
    - Tracheostomy

Delayed Dental Eruption

- Normal range for dental eruption is between 8 and 18 months
- Delayed dental eruption is considered when teeth fail to erupt within 12 months of "normal range"
- Possible etiologies include: hypothyroidism, hypopituitarism, ectodermal dysplasia, rickets

Odontogenic Infection

Etiology
- Caries are typically primary cause of odontogenic infections
- Polymicrobial
  - *Streptococcus mutans* (most common cause of initial caries infection)
  - Alpha-hemolytic streptococci
  - Anaerobes (*peptostreptococcus*, *bacteroides*, *fusobacterium*)

Clinical presentation
- Localized pain, edema, erythema, purulence
- Sensitivity to temperatures and palpation, loose tooth
- Orofacial swelling
  - Swelling below jaw (mandibular abscess)
  - Periorbital swelling (maxillary abscess)

Imaging
- Evaluate airway compromise, gas-producing organisms, presence of abscess, extent of involvement
- Panorex
- CT scan

Treatment
- Remove source of infection (i.e., tooth)
- Analgesia
- Antibiotics
- I & D if abscess present

Early Childhood Caries

Definition
- Caries affecting the primary dentition especially in the first 3 years of life

Caries formation
- Chronic infectious disease
- Pathogenesis: tooth-adherent bacteria (most commonly *streptococci mutans*) metabolizes sugars to produce acid that leads to demineralization of the tooth structure

Risk factors
- Bottle propping (affects predominantly central incisors)
- Low-income households
- Excessive consumption of sugar
- Genetic factors

Prevention
- A dental visit within the first 6 months of first tooth eruption and no later than one year of age
- Tooth brushing is suggested twice daily with an age appropriate size of fluoridated toothpaste (discourage swallowing toothpaste to prevent fluorosis)
- Avoid high frequency consumption of high sugar liquids/solid foods
- Recommend weaning from bottle between 12 and 18 months and transitioning to a cup

Fluoride supplementation
- Dental fluorosis occurs during the development of the tooth (critical ages between 0 and 6 years of age, with most important being between 15 and 30 months) [7]
- Be aware that access to fluoridated water may be limited in some areas in the USA
- Optimal water fluoridation is 0.7 ppm of fluoride
- If limited access to fluoridated water, supplementation may be considered, especially for patients between 15 and 30 months of age

Dental Trauma and Avulsions

Primary tooth avulsion
- Refer to the dentist for follow-up to rule out any associated problems
- Avoid reimplantation of primary avulsed tooth
- Permanent tooth avulsion (it is a true dental emergency) [1]
  - Reimplantation of tooth
    - If reimplanted within 5 min—tooth survival rate is 85–97%
    - If reimplanted after 1 h of injury—tooth is unlikely to survive
  - Instructions for avulsed permanent tooth:
    - Gently wash the avulsed tooth with no rubbing or brushing
    - Re-implant the tooth into the socket as soon as possible
    - If not possible, preserve the tooth in saliva, milk, or normal saline
    - Goal: to maintain viability of the periodontal ligament fibers
    - Child should be transported to a dentist office or nearest emergency room

Neck

Cervical Lymphadenitis

Pathogens
- Viral: EBV (most common viral), CMV, HSV, adenovirus, enterovirus, roseola, rubella, HIV
- Bacterial: Group A Strep (most common), *Staph aureus*

Clinical presentation
- Fevers (typically low grade for viral), malaise, tender and mobile cervical nodes (Fig. 8)

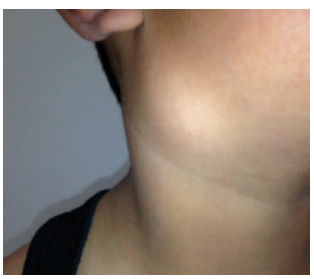


Fig. 8 9-year-old boy presented with high fever 104 °F, malaise, and tender large bacterial cervical lymphadenopathy

Diagnosis
- History and physical examination
- Possible aspiration for culture and sensitivity

Complications:
- Cellulitis, abscess, internal jugular vein thrombosis, mediastinitis, sepsis

Treatment
- Viral: supportive
- Bacterial
  - Antibiotics
  - Incision and drainage if abscess formation

Infectious Mononucleosis

- Caused by Epstein–Barr virus (EBV)

Clinical presentation
- Fever, pharyngitis, and lymphadenopathy
- Symmetric cervical adenopathy (posterior triangle nodes most commonly)
- Axillary and inguinal nodes also may be involved.
- Fatigue, malaise, splenomegaly

Diagnostic tests
- Monospot test ("heterophile antibody")
  - High false negative rate if obtained early on in illness or in children under 4 years of age
- Elevated immunoglobulin M titer to viral capsid antigen (IgM-VCA), indicate acute infection

Cat-Scratch Disease

Pathogen: *Bartonella henselae*

Clinical presentation
- Present ~2 weeks after cat scratch or bite (usually from a kitten)
- Papular lesion at primary scratch site associated with cervical lymphadenopathy (tender initially, then becomes painless)—may ulcerate and form fistula
- Fever (often mild), malaise
- Diagnosis: serology (IgG henselae titers), culture (Warthin–Starry stain), PCR, histopathology

Treatment
- Supportive (typically self-limiting)
- Antibiotic therapy in immunocompromised patients
- Surgical aspiration for culture, but avoid formal incision and drainage to prevent fistula/sinus formation

Atypical Mycobacteria

- Pathogen: *mycobacterium avium complex*, *M. scrofulaceum*, *M. kansasii*
- Risk factors: young children, immunocompromised

Clinical presentation
- Asymptomatic
- Unilateral cervical lymphadenopathy, preauricular adenopathy, commonly located on face over body of mandible
- Adhesive to overlying skin and overlying skin is erythematous in advanced disease

Diagnosis
- Acid-fast stain, culture (requires 2–4 weeks for results)

Treatment
- Watchful waiting (typically takes months to resolve)
- Excision or incision and curettage (avoid incision and drainage)

Other Causes Lymphadenitis

- Tuberculosis: in children, less common than atypical mycobacterium
- Kawasaki disease (mucocutaneous lymph node syndrome)
  - Acute vasculitis affecting multiple organs in children
  - Diagnosis
    - Must have 5 of the following:
    - Fever > 5 days (high)—absolute criteria
    - Erythematous rash
    - Conjunctival injection
    - Oropharyngeal changes
    - Peripheral extremity changes (induration or desquamation)
    - Cervical lymphadenopathy
    - Echocardiogram
    - High risk of developing coronary aneurysm or myocardial infarction

Kikuchi
- Rare disease of unknown etiology
- Presentation: young women, cervical and generalized lymphadenopathy, fever, night sweats, rash, weight loss, nausea and vomiting
- Diagnosis: lymph node biopsy—histiocytic necrotizing lymphadenitis
- Treatment
  - No effective treatment, typically resolves within 1 to 4 months
  - Symptom control with steroids
  - Follow up is necessary as patient with Kikuchi are at higher risk of developing systemic lupus

Tularemia
- Pathogen: *Francisella tularensis*
- Transmission: contact with infected animal (i.e., rabbit or hamster)
- Presentation: febrile illness, ulceroglandular syndrome (painful regional lymphadenopathy and an ulcerated skin lesion)
- Treatment: streptomycin

Castleman's disease
- Lymphoproliferative disorder localized to a single node (unicentric) or systemically (multicentric)
- Unicentric
  - Typically asymptomatic—presents with an enlarged lymph node (20% in neck)
  - CT scan shows a well-circumscribed mass
  - Pathology demonstrates nodal expansion
  - Surgical removal is curative 90% of the time
- Multicentric
  - 50% are associated with Kaposi sarcoma-associated herpes virus and/or human herpesvirus type 8
  - No standard treatment. May include: antivirals, chemotherapy, corticosteroids, monoclonal antibodies
  - Refer to Oncology

Lymphoma

- Most common pediatric malignancy of the head and neck
- Lymphoproliferative disorder
- Hodgkin's and non-Hodgkin's lymphoma may present with cervical lymphadenopathy

Clinical presentation
- Nodal masses—may present with cervical nodes
- Hodgkin: contiguous lymph nodes
- Non-Hodgkin lymphoma: may present with extranodal involvement (i.e., enlarged tonsil, base of tongue, enlarged thyroid, etc.)
- Constitutional symptoms: fevers, night sweats, weight loss

Diagnosis
- History and physical examination
- Evaluation of all nodal sites
- Open biopsy (rather than fine needle biopsy)—fresh tissue is required for immunochemistry

Management
- If positive for lymphoma, refer to oncology

Thyroglossal Cyst

Definition
- Failed obliteration of thyroglossal duct

Clinical presentation
- Midline neck mass (often cystic)—inferior to hyoid bone and superior to thyroid
- Elevates with tongue protrusion (pathognomonic)

Complications
- May become infected
- Rare malignant potential

Treatment
- Treat infection with antibiotics (avoid incision and drainage)
- Surgical removal when not infected (Sistrunk procedure)

Branchial Cleft Cyst

- Alterations of the branchial apparatus resulting in cysts, sinuses, or fistula

Presentation
- Unilateral (most commonly)
- Anterior neck mass (typically anterior to SCM muscle), sinus, or fistula
- May become infected with drainage (associated with URI)

Treatment
- Treat infection with antibiotics (avoid incision and drainage)
- Complete surgical excision of cyst, sinus, and fistula tract once infection resolves

Lymphatic Malformation

- Also known as cystic hygroma and lymphangioma (outdated terms)
- Etiology: abnormal lymphatic development

Presentation
- May occur anywhere in body
- Soft, painless, multiloculated, compressible mass that transilluminates
  - In cervical region, posterior triangle is most common
- Present at birth or shortly thereafter
- Associated symptoms related to mass compression of nearby structures

Imaging: MRI preferred

Management
- Observation if small and no associated complications
- Sclerosing agents
- Surgical excision

Acute Laryngitis

Etiology
- Infectious (most commonly viral, may have secondary bacterial infection)
- Fungal infection (immunocompromised child)
- Vocal strain (secondary to screaming/yelling)

Management
- Generally self-limiting
- Optimize hydration
- Humidification
- Salt-water gargles
- Treat with antibiotics or antifungals if bacterial or fungal infection suspected

Chronic Laryngitis/Hoarseness

- Definition: symptoms of hoarseness, dysphonia, and/or vocal fatigue for > 3 months
- Associated symptoms: chronic cough, frequent throat clearing

Etiology
- Typically noninfectious causes (most common vocal fold "screamers" nodules)
- Environmental irritants
- Environmental allergies
- Postnasal drip
- Medications (e.g., inhaled steroids)
- Gastroesophageal reflux disease
- Rarely, chronic systemic disease (e.g., amyloid, Wegner's, etc.) or malignancy

Diagnosis
- ENT referral for flexible laryngoscopy

Management
- Treat underlying cause

Vocal Fold Paralysis

Background
- One of the most common laryngeal abnormalities in childhood
- Unilateral or bilateral paralysis of the vocal fold
- Congenital or acquired

Etiology
- Iatrogenic (most common): cardiothoracic surgery, tracheoesophageal fistula repair, thyroidectomy)
- Idiopathic
- Viral
- Autoimmune
- Neurologic (e.g., Arnold–Chiari malformation, posterior fossa tumor)
- Pulmonary lesion

Diagnosis
- Refer to ENT for flexible laryngoscope which will assess for vocal fold mobility, mucosal lesions, and laryngeal masses

Workup of vocal fold paralysis
- Observation (if known iatrogenic cause)
- CXR
- Modified barium swallow (to assess for aspiration)
- MRI head
- CT neck and chest

Treatment
- Observation
  - Monitor for signs of aspiration or respiratory distress
  - Monitor for signs for recovery
- Surgery
  - Tracheostomy
  - Vocal fold surgery
  - Hypoglossal to recurrent laryngeal nerve reanastomosis

Surgical Interventions

Indication for Tonsillectomy (+/– Adenoidectomy) [2, 9]

Absolute indications
- Moderate to severe obstructive sleep apnea
- Suspicions of tonsillar malignancy

Relative indications
- Mild obstructive sleep apnea
- Recurrent tonsillitis—must meet criteria
  - Frequency
    - Seven or more episodes in 1 year, or
    - Five or more episodes per year for 2 years, or
    - Three or more episodes per year for 3 years
- Associated with one or more of the following:
  - Temperature > 38.3
  - Cervical lymphadenopathy
  - Tonsillar exudate
  - Positive test for GABHS
- Chronic tonsillitis unresponsive to antimicrobial therapy
- Severe halitosis
- Peritonsillar abscess (greater than one episode)
- PFAPA syndrome (periodic fever, aphthous ulcers, pharyngitis, cervical adenitis)

Indication for Adenoidectomy Alone

- Moderate to severe nasal obstruction with persistent symptoms
- Refractory chronic sinusitis
- Recurrent acute otitis media or otitis media with effusion in a child who had prior tympanostomy tubes which have now extruded (e.g., repeat surgery when indicated would consist of adenoidectomy plus myringotomy ± insertion of ventilation tube)

Postsurgical Complications of Adenotonsillectomy

- Anesthesia related
- Pain: Moderate to severe lasting 7–14 days, requiring analgesia
- Hemorrhage:
  - Bimodal timing
  - < 24 h postoperative: ~<2%
  - 5–7 days postoperative (sloughing of eschar): ~3%
- Dehydration secondary to decrease oral intake
- Halitosis (expected)
- Immediate postoperative airway obstruction (due to anesthesia, analgesia, or sleep apnea)
- Persistence of obstructive sleep apnea
- Velopharyngeal insufficiency (VPI)
  - New onset or worsening of existing VPI
  - High-risk patients: cleft palate, submucous cleft palate, impaired baseline palatal movement (e.g., neurogenic), very large adenoid pad, velocardiofacial syndrome

Indication for Myringotomy and Tympanostomy Tubes for Acute Otitis Media (AOM) and Otitis Media with Effusion (OME) [10]

- Bilateral myringotomy and tympanostomy tubes are indicated when in a patient with:
  - Bilateral OME for 3 months or more and documented hearing difficulties
  - Unilateral or bilateral OME for 3 months and symptoms likely related to OME, for example, vestibular symptoms, poor school performance, behavioral difficulties, ear discomfort, and decreased quality of life
  - Recurrent AOM and unilateral or bilateral middle ear effusion at time of assessment
  - In at risk children, with unilateral or bilateral OME that is unlikely to resolve quickly as reflected by a type B tympanogram (flat) or persistent effusion for 3 months or longer

Complications of Tympanostomy Tubes

- Anesthesia related
- Tube otorrhea (most common)
- Blockage of tube
- Granulation tissue formation
- Displacement of tube in middle ear
- Tympanic membrane changes: myringosclerosis, atrophy, atelectasis, retraction pocket
- Persistent tympanic membrane perforation (may required surgical repair)

Suggested Readings

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