OTOLARYNGOLOGY - HEAD & NECK SURGERY

Dr. J. Irish and Dr. B. Papsin
Yvonne Chan, Neety Panu and Evan Propst, chapter editors

PHYSICAL EXAMINATION

HEAD AND NECK

Inspection of Head and Neck

❑ position of head
❑ symmetry of facial structure
❑ look for neck scars, asymmetry, masses, enlarged thyroid

Palpation of Head and Neck

❑ lymph node examination (see Figure 12)
- observe size, mobility, consistency, tenderness, warmth, regular/irregular border
  - occipital, posterior auricular, preauricular, superficial posterior cervical, deep cervical, tonsillar, submandibular, submental, supraclavicular

❑ salivary gland examination
- palpate parotid and submandibular glands for tenderness, swelling, masses, or nodules

Thyroid Gland

❑ inspection of gland symmetry and mobility
❑ palpation via anterior or posterior approach
- note size, shape, and consistency of gland
- identify any nodules or areas of tenderness

❑ if gland is enlarged, auscultate with bell
- listen for thyroid bruit suggestive of a toxic goiter

EARS

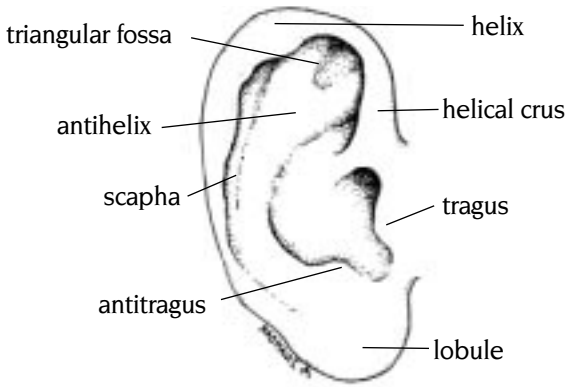


Figure 1. Surface Anatomy of the Ear

Illustration by Aarti Inamdar

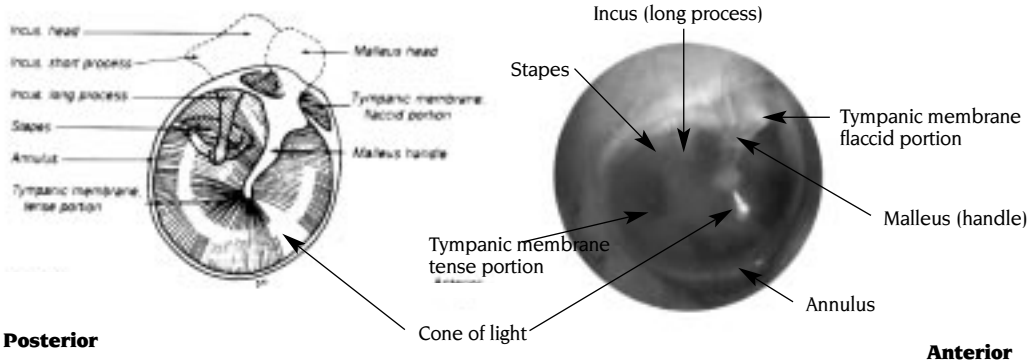
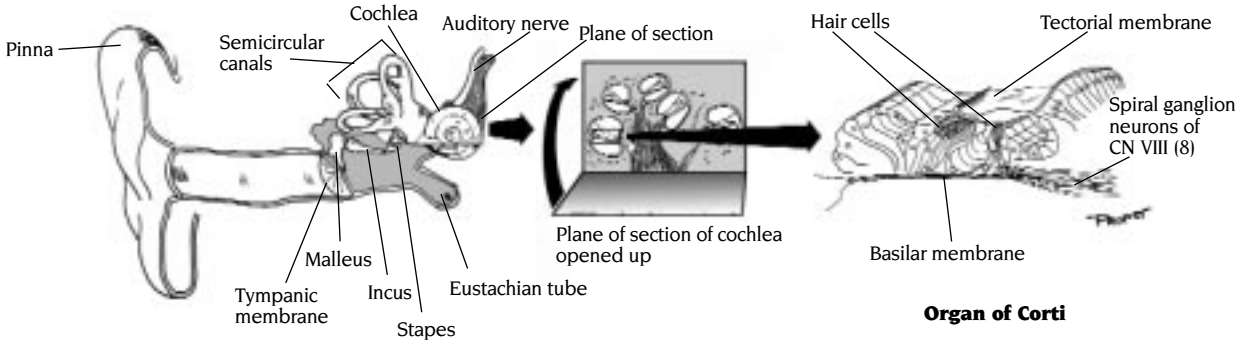


Figure 2. Normal Appearanc of Right Tympanic Membrane on Otoscopy

Diagram from Lucente FR, Sobol SM. *Essentials of Otolaryngology 3rd Edition*. Raven Press, 1993, New York, New York. Page 5.

Physiology of the Ear



- in normally hearing individuals, sound travels down the auditory canal and vibrates the tympanic membrane
- these vibrations are amplified by the middle ear ossicles (malleus, incus, stapes) and are transmitted to the oval window of the cochlea
- varying pressure on the fluid in the cochlea causes movement of the basilar membrane
- hair cells housed within the organ of Corti (which rests on the basilar membrane) are stimulated by this movement
- the auditory signal is transduced to a neural code, which is passed along spiral ganglion neurons of the auditory nerve (CN VIII) and up to the inferior colliculi (centers for auditory reflex) and via the medial geniculate body in the thalamus to the auditory cortex (Brodmann's area 41).

Figure 3. Physiology of Normal Hearing

Illustration by Evan Propst

External Examination of Ear (see Figure 1)

❑ inspect external ear structures
- note position of ear
- look for deformities, nodules, inflammation, or lesions
- potential findings
  - microtia or macrotia: congenitally small or large auricles
  - cauliflower ear: deformity of pinna due to subperichondrial hematomas resulting from repeated mechanical trauma
  - small sinus in front of tragus : remnant of first branchial arch
  - tophi: sign of gout
  - discharge: note colour and consistency

❑ palpate external ear structure
- examine for infection of external ear
  - pain elicited by pulling pinna up or down, or pressing on tragus
- apply pressure on mastoid tip
  - tenderness may indicate infective process of the mastoid bone

Otoscopic Examination (see Figure 2)

❑ select largest speculum that will fit into external canal with minimal discomfort

❑ inspect external canal
- look for evidence of inflammation, foreign bodies, or discharge

❑ inspect tympanic membrane (TM)
- normal membrane: intact, translucent, gray
- Use E.M.I.L.Y. method
  - Eustachian tube: consider how tube affects mobility and appearance of TM
    Erythema: normal at the following areas:
    - junction where cartilage becomes bone
    - vessels on long process of malleus
    - veins on tympanic membrane
    - tympanic membrane when child is crying
  - Malleus: assess the short process, long process, umbo
  - Insufflation
    - only if there is a question of middle ear infection
    - pneumatic otoscopy to demonstrate decreased movement of tympanic membrane
  - Light Reflex: directed anteroinferiorly
  - Yellow: look for colour of fluid behind tympanic membrane
    - gray: hemorrhage
    - yellow: infection
    - clear yellow: serous otitis media
- possible abnormal findings
  - acute otitis media: erythema of pars flaccida and tensa, malleus not visualized due to inflammation, lack of motion of tympanic membrane, absence of light reflex
  - otitis media with effusion: erythema of malleus, pars tensa injected, prominent short process of malleus, limited motion, decreased light reflex, yellow serous fluid behind tympanic membrane
  - tympanosclerosis: dense white plaques
  - membrane perforation

Auditory Acuity

❑ mask one ear and whisper into the other
❑ tuning fork tests - see Table 1
- Rinne's Test
  - 512 Hz tuning fork is struck and held firmly on mastoid process to test bone conduction (BC). When it can no longer be heard it is placed close to ear to test air conduction (AC)
  - if it can then be heard then AC > BC or Rinne positive
  - a loss of approximately 15 dB is required to reverse the Rinne (BC > AC)

- Weber's Test
  - vibrating fork is held on vertex of head and patient states whether it is heard centrally (Weber negative) or is lateralized to one side (Weber right, Weber left)
  - lateralization indicates ipsilateral conductive hearing loss or contralateral sensorineural hearing loss
  - place vibrating fork on patient's chin while they clench their teeth, or directly on teeth to elicit more reliable response
  - a difference of approximately 5 dB is required for the Weber to lateralize

Table 1. The Interpretation of Tuning Fork Tests

Examples	Weber	Rinne
Normal or Bilateral Sensorineural Hearing Loss	Central	AC>BC (+) bilaterally
Right Sided Conductive Hearing Loss, Normal Left Ear	Lateralizes to Right	BC>AC (–) right
Right Sided Sensorineural Hearing Loss, Normal Left Ear	Lateralizes to Left	AC>BC (+) bilaterally
Right Sided Severe Sensorineural Hearing Loss or Dead Right Ear, Normal Left Ear	Lateralizes to Left	BC>AC (–) right *

* a vibrating fork on the mastoid stimulates both cochleae, therefore in this case, the left cochlea is stimulated by the Rinne test on the right, i.e. a false negative test

These tests are not valid if the ear canals are not free of cerumen (i.e. will create conductive loss)

NOSE

External Examination of Nose

❑ inspect nose
- look for swelling, trauma, congenital anomalies, deviation
- test patency of each nostril if deviation is suspected

❑ palpate sinuses
- tenderness over frontal and maxillary sinuses may indicate sinusitis

Internal Examination of Nose

❑ inspect with nasal speculum
- position of septum
- colour of nasal mucosa
  - normally pink and moist with a smooth clean surface, blue/grey secondary to allergies, and red secondary to inflammation
- size, colour and mucosa of inferior and middle turbinates
- possible abnormal findings
  - septal deviation or perforation
  - exudate, swelling, epistaxis
  - nasal polyps

ORAL CAVITY

- ❑ ask patient to remove dentures
- ❑ lips
  - colour of skin and mucosal surface, presence of lesions
- ❑ buccal mucosa
  - use two tongue blades and slowly move around the mouth
  - identify Stensen's duct (parotid gland duct orifice) opposite upper first or second molar
- ❑ gingivae and dentition
  - 32 teeth in full dentition; colour and condition of gingiva
  - look for malocclusion
- ❑ hard and soft palates
  - examine for symmetry
  - inspect for ulceration or masses
- ❑ tongue
  - inspect for colour, mobility, masses, tremor, and atrophy
  - use tongue depressor to manipulate tongue to examine undersurface and sides
  - palpate tongue for any masses
  - test cranial nerve XII
- ❑ floor of mouth
  - palpate for any masses
  - identify Wharton's ducts (submandibular gland ducts) on either side just lateral to frenulum of tongue
  - bimanually palpate submandibular glands

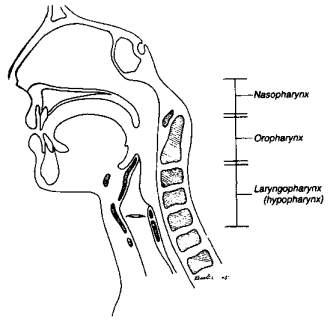


Figure 4. Sagittal Section with Divisions of Nasopharynx, Oropharynx, Hypopharynx

Figure from Essentials of Otolaryngology. 4th ed. Lucente FE and Har-EIG. (eds)

Oropharynx

- ❑ anterior faucial pillars, tonsils, tonsillolingual sulcus
  - depress middle third of tongue with tongue depressor and scoop tongue forward to visualize tonsils
  - note size and inspect for tonsillar exudate or lesions
- ❑ posterior pharyngeal wall

NASOPHARYNX

Postnasal Mirror (Indirect)

- ❑ ensure good position of patient
  - must sit erect with chin drawn forward ("Sniffing Position")
  - instruct patient to breathe through nose, allowing palate to depress and nasopharynx to open
- ❑ with adequate tongue depression, the warmed mirror is placed next to uvula and almost touches the posterior pharyngeal wall
- ❑ rotate mirror to inspect the following areas
  - choana
  - posterior end of the vomer: should be in midline
  - inferior, middle, and superior meatus
    - may see pus dripping over posterior end of inferior meatus (sign of maxillary sinusitis)
  - eustachian tubes
  - adenoids (mostly in children)

Nasopharyngolaryngoscope (Direct)

- ❑ detailed view of nasal cavities and nasopharynx

HYPOPHARYNX AND LARYNX

Indirect Laryngoscopy

- ❑ ensure good position of patient
- ❑ while holding tongue with gauze, introduce slightly warmed mirror into mouth and position mirror in oropharynx
- ❑ ask patient to breathe normally through mouth while mirror is pushed upward against the uvula
  - touching the uvula and soft palate usually does not elicit a gag reflex, unlike touching the back of the tongue
  - the gag reflex can be suppressed if patients are told to pant in and out
- ❑ image seen in mirror will be reversed (see Figure 5)
- ❑ inspect the following, noting any irregularity of the edges, nodules or ulcerations
  - circumvallate papillae and base of tongue, lingual tonsils, valleculae epiglottis, aryepiglottic folds and pyriform fossae, false vocal cords, true vocal cords
  - note position and movement of cords
    - quiet respiration
      - cords are moderately separated
    - inspiration
      - cords abduct slightly
  - ask patient to say "eeee"
    - cords adduct to midline
    - look for signs of paralysis or fixation

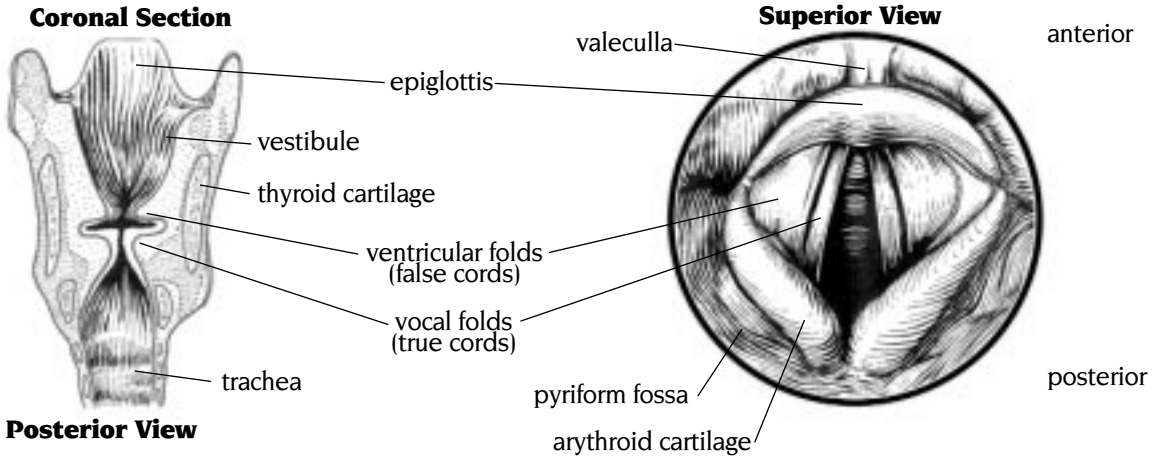


Figure 5. Anatomy of Normal Larynx

Illustration by Glen Oomen

Direct Laryngoscopy with Fibreoptic Nasopharyngolaryngoscope
- ❑ prepare patient with topical anesthetic administered by nasal anaesthetic/decongestant spray
- ❑ flexible scope passed via nasal cavity to view structures in the larynx as mentioned above

OTONEUROLOGICAL EXAMINATION (see Neurology Chapter)
- ❑ otoscopy
- ❑ cranial nerve testing (II-XII inclusive)
- ❑ cerebellar testing

Nystagmus
- ❑ assess nystagmus - describe quick phase, avoid examining in extremes of lateral gaze
- ❑ horizontal nystagmus that beats in the same direction = peripheral vestibular disorder
  - the lesion is usually on side of the slow beat, with the fast phase beating away from the side of the lesion
- ❑ horizontal nystagmus that changes direction with gaze deviation = central vestibular disorder
- ❑ vertical upbeating nystagmus = brainstem disease
- ❑ vertical downbeating nystagmus, usually = medullocervical localization (e.g. Arnold-Chiari)

Assess Brain Perfusion
- ❑ carotid bruits, subclavian stenosis
- ❑ positional blood pressure measurements

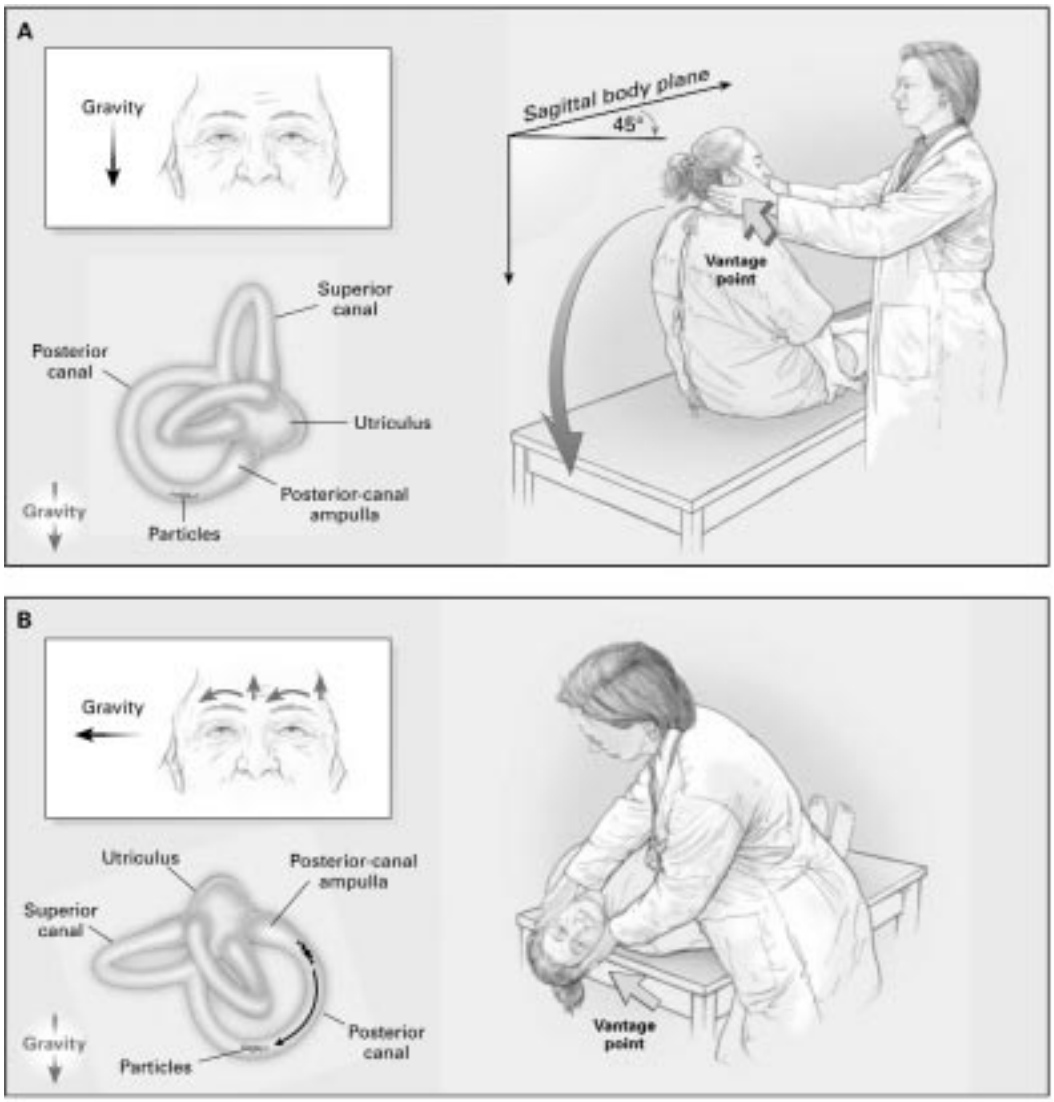
Balance Testing
- ❑ Romberg's test: patient stands upright with feet together, eyes closed, and arms folded in front of chest
  - sway is associated with loss of either joint proprioception or a peripheral vestibular disturbance
  - the patient leans or tends to fall toward the side of the diseased labyrinth
- ❑ Unterberger's test: marching on the spot with the eyes closed
  - peripheral disorders: rotation of body to the side of the labyrinthine lesion
  - central disorders: deviation is irregular

Electronystagmography (ENG)
- ❑ electrodes placed around eyes
- ❑ eye is a dipole, cornea (+), retina (–)
- ❑ used to measure rate, amplitude, and frequency of nystagmus elicited by different stimuli

Caloric Stimulation Test
- ❑ with the patient supine, the neck is flexed 30º to bring the horizontal semicircular canal into a vertical position
- ❑ the volume of endolymph is changed by irrigating the labyrinthine capsule with water at 30ºC or 44ºC for 35 seconds
- ❑ the change in volume causes deflection of the cupula and subsequent nystagmus through the vestibuloocular reflex (VOR)
- ❑ the extent of response indicates the function of the stimulated labyrinth
- ❑ cold water will result in nystagmus to the opposite side of irrigation and warm to the same side irrigation (COWS - Cold Opposite, Warm Same)

Dix-Hallpike Positional Testing with Frenzel's (Magnifying) Eyeglasses (See Figure 6)

- ❑ the patient is rapidly moved from a sitting position to a supine position with the head hanging over the end of the table, turned to one side at 45°. This position is held for 20 seconds
- ❑ onset of vertigo is noted and the eyes are observed for nystagmus



In Panel A, the examiner stands at the patient's right side and rotates the patient's head 45 degrees to the right to align the right posterior semicircular canal with the sagittal plane of the body. In Panel B, the examiner moves the patient, whose eyes are open, from the seated to the supine right-ear-down position and then extends the patient's neck slightly so that the chin is pointed slightly upward. The latency, duration, and direction of nystagmus, if present, and the latency and duration of vertigo, if present, should be noted. The red arrows in the inset depict the direction of nystagmus in patients with typical benign paroxysmal positional vertigo. The presumed location in the labyrinth of the free-floating debris thought to cause the disorder is also shown.

Figure 6. The Dix-Hallpike Test of a Patient with Benign Positional Vertigo Affecting the Right Ear

Source: Furman JM *nad* Cass SP. *Benign Paroxysmal Positional Vertigo. The New England Journal of Medicine. Vol.* 341 (21): 1590-1596. 1999.

AUDIOLOGY

PURE TONE AUDIOMETRY

- ❑ threshold is the faintest intensity level at which a patient can hear the tone 50% of the time
- ❑ the lower the threshold, the better the hearing
- ❑ typical conversation is at 45 dB
- ❑ thresholds are obtained for each ear for frequencies 250 to 8000 Hz
- ❑ air conduction thresholds are obtained with headphones and measure outer, middle, inner ear, and auditory nerve function
- ❑ bone conduction thresholds are obtained with bone conduction oscillators which effectively bypass outer and middle ear function

Clinical Pearl

❑ **Air conduction thresholds can only be equal to or greater than bone conduction thresholds.**

Degree of Hearing Loss

❑ determined on basis of the Pure Tone Average (PTA) at 500, 1000, 2000 Hz

0-15 dB normal	56-70 dB moderate-severe
16-25 dB slight	71-90 dB severe
26-40 dB mild	91 + dB profound
41-55 dB moderate	

Types of Hearing Loss

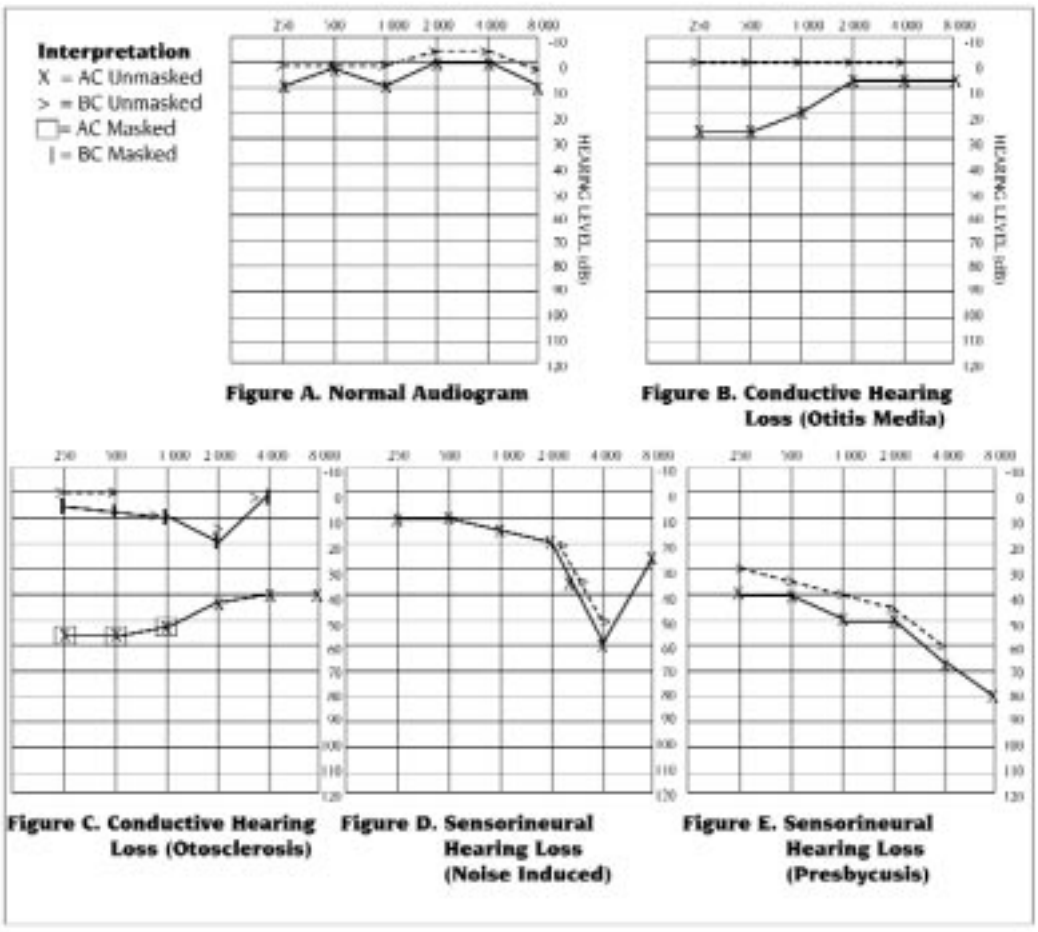


Figure A. Normal Audiogram

Figure B. Conductive Hearing Loss (Otitis Media)

Figure C. Conductive Hearing Loss (Otosclerosis)

Figure D. Sensorineural Hearing Loss (Noise Induced)

Figure E. Sensorineural Hearing Loss (Presbycusis)

Figure 7. Types of Hearing Loss and Associated Audiograms

1. Conductive Hearing Loss (CHL)
- ❑ the conduction of sound through the entire ear to the cochlea is impaired
- ❑ can be caused in external and middle ear disease
- ❑ features
    1. bone conduction in normal range
    2. air conduction outside of normal limits
    3. gap between AC and BC thresholds >10 dB ("an air-bone gap")

2. Sensorineural Hearing Loss (SNHL)
- ❑ the sensory component of the cochlea acoustic nerve (CN VIII), brainstem or cortex is damaged
- ❑ unilateral SNHL should be investigated to rule out acoustic neuroma
- ❑ can be caused by inner ear disease
- ❑ features
    1. both air and bone conduction thresholds below normal
    2. gap between AC and BC < 10 dB ("no air-bone gap")
- ❑ otosclerosis shows a typical dip in the audiogram at 2,000 Hz (Carhart's notch)
- ❑ noise induced hearing loss shows a dip at 4,000 Hz because the temporal bone resonates at this frequency when exposed to prolonged noise (i.e. machinery)

3. Mixed
- ❑ the conduction of sound to the cochlea is impaired, as is the transmission through the cochlea to the cortex
- ❑ features
    1. both air and bone conduction thresholds below normal
    2. gap between AC and BC thresholds > 10 dB ("an air-bone gap")

SPEECH AUDIOMETRY

Speech Reception Threshold (SRT)
- ❑ lowest hearing level at which patient is able to repeat 50% of two syllable words ("spondees", e.g. "hotdog", "baseball")
- ❑ SRT and best pure tone threshold in the 500-2,000 Hz range (frequency range of human speech) usually agree within 5 dB. If not, suspect a retrocochlear lesion or functional hearing loss

Speech Discrimination Test
- ❑ percentage of words the patient correctly repeats from a list of 50 monosyllabic words (e.g. boy, aim, go)
- ❑ tested at a level 35-50 dB > SRT, so degree of hearing loss is taken into account
- ❑ classification of speech discrimination testing

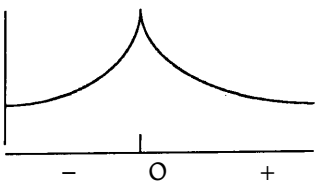
90-100%	excellent	40-60%	poor
80-90%	good	< 40%	very poor
60-80%	fair		

- ❑ patients with normal hearing or conductive hearing loss score > 90%
- ❑ score depends on amount of sensorineural hearing loss present
- ❑ a decrease in discrimination as sound intensity increases is typical of a retrocochlear lesion (rollover effect)
- ❑ investigate further if scores differ across ears by > 20%

IMPEDANCE AUDIOMETRY

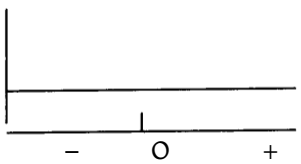
Tympanogram
- ❑ the eustachian tube equalizes the pressure between external and middle ear
- ❑ tympanogram is a graph of the compliance of the middle ear system over a pressure gradient ranging from +200 to –400 mm H_2O
- ❑ peak of tympanogram occurs at the point of maximum compliance where the pressure in the external canal is equivalent to the pressure in the middle ear
- ❑ normal range: -100 to +50 mm H_2O

Type A Tympanogram



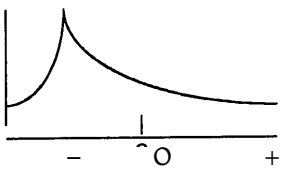
normal pressure peak at 0
note that with otosclerosis the peak is still at 0mm H_2O but has a lower amplitude (an As Tympanogram)

Type B Tympanogram



no pressure peak
poor TM mobility indicative of middle ear effusion (e.g. otitis media with effusion) or perforated TM

Type C Tympanogram



negative pressure peak
indicative of chronic eustachian tube insufficiency (e.g. serous or secretory otitis media)

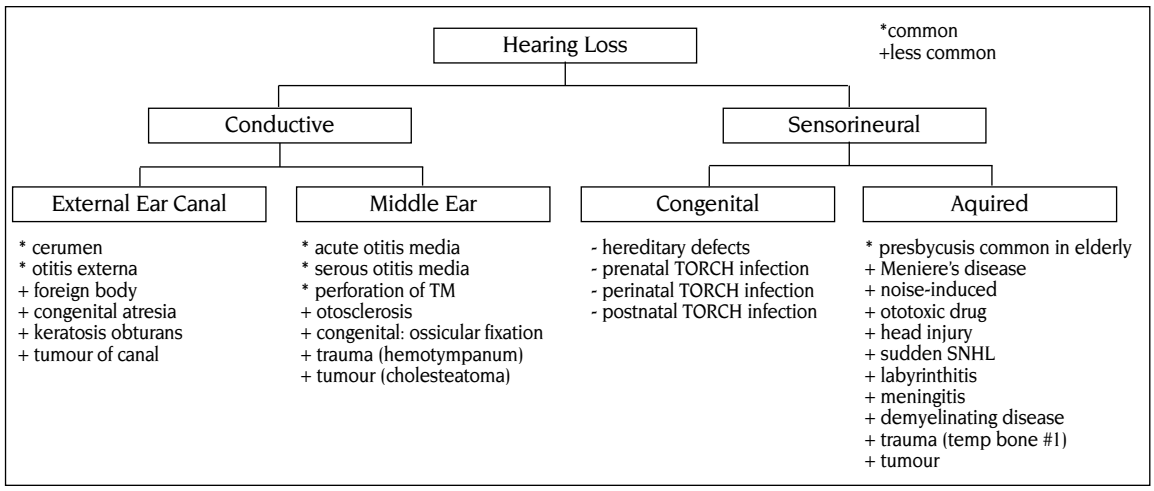
Static Compliance
- ❑ volume measurement reflecting overall stiffness of the middle ear system
- ❑ normal range: 0.3 to 1.6 cc
- ❑ negative middle ear pressure and abnormal compliance indicate middle ear pathology

Acoustic Stapedial Reflexes
- ❑ stapedius muscle contracts when ear is exposed to loud sound and results in increased stiffness or impedance of middle ear system (TM and ossicles)
- ❑ acoustic reflex thresholds occur at 70-100 dB above hearing threshold
- ❑ if hearing threshold is greater than 85 dB, the reflex is likely to be absent
- ❑ stimulating either ear causes reflex to occur bilaterally and symmetrically
- ❑ reflex pathway involving vestibulocochlear cranial nerve, cochlear nucleus, trapezoid body, superior olivary nucleus, facial nucleus, and facial nerve (i.e. a measure of central neural function)
- ❑ for reflex to be present, CN VII must be intact and there must be no conductive hearing loss in the monitored ear. If reflex is absent without conductive loss or severe sensorineural loss, suspect CN VIII lesion
- ❑ acoustic reflex decay test: tests the ability of the stapedius muscle to sustain contraction for 10 s at 10 dB stimulation
- ❑ normally, little reflex decay occurs at 500 and 1000 Hz
- ❑ with cochlear hearing loss the acoustic reflex thresholds are typically 25-60 dB
- ❑ with retrocochlear hearing loss (e.g. acoustic neuroma) may find absent acoustic reflexes or significant reflex decay (> 50%) within 5 second interval

AUDITORY BRAINSTEM RESPONSE (ABR)
- ❑ the patient is exposed to an acoustic stimulus while an electroencephalogram is recorded to assess any changes in brain activity
- ❑ delay in brainstem response is suggestive of cochlear or retrocochlear abnormalities (for the latter think tumour or multiple sclerosis (MS))

Clinical Pearl
- ❑ **This objective test can be used in screening newborns or much more rarely to uncover normal hearing in malingering patients.**

HEARING LOSS



OTITIS EXTERNA (OE)

Clinical Pearl
❑ **Otitis externa has two forms: a benign painful infection of the outer canal that could occur in anybody and a potentially lethal less painful (damaged sensory nerves) disease which usually occurs in old, immunosuppressed or diabetic patients.**

Etiology
❑ caused by
- bacteria: P. *aeruginosa*, P. *vulgaris*, E. *coli*, S. *aureus*
- fungi: *Candida albicans*, *Aspergillus niger*

❑ more common in summer
❑ associated with swimming ("swimmer's ear"), mechanical cleaning (Q-tips, skin dermatitides)

Presentation
❑ acute
- pain aggravated by movement of auricle (traction of pinna or pressure over tragus)
- +/– unilateral headache, +/– low grade fever
- otorrhea - sticky yellow purulent discharge
- conductive hearing loss - due to obstruction of external canal with purulent debris
- post-auricular lymphadenopathy

❑ chronic
- pruritus of external ear +/– excoriation of ear canal
- atrophic and scaling epidermal lining
- +/– otorrhea, +/– hearing loss
- wide meatus but no pain with movement of auricle
- tympanic membrane appears normal

Treatment
❑ clean ear under magnification with irrigation, suction, dry swabbing, and C+S
❑ bacterial etiology
- if membrane intact, give topical aminoglycoside antibiotics +/- corticosteroids (e.g. Garamycin, Neosporin, Corticosporin)
- if perforated membrane, give ciprofloxacin otic drops, because aminoglycosides can be ototoxic
- introduction of fine gauze wick (pope wick) if external canal edematous
- +/– 3% acetic acid solution to acidify ear canal
- systemic antibiotics if either:
  1. cervical lymphadenopathy
  2. cellulitis

❑ fungal etiology
- alcohol/acetic acid instillation, clotrimazole, locacortin (Vioforme)

❑ +/– analgesics
❑ chronic otitis externa pruritus without obvious infection - corticosteroid alone e.g. diprosalic acid

Malignant Otitis Externa (rare)
❑ osteomyelitis of temporal bone, 99% of which are due to P*seudomonas*
❑ associated with diabetics, elderly, perichondritis, cellulitis, parotitis, +/– chronic symptoms
❑ requires hospital admission, debridement, IV antibiotics and emergent CT scan

ACUTE OTITIS MEDIA (AOM) AND OTITIS MEDIA WITH EFFUSION (OME)
(see Pediatric ENT section)

CHOLESTEATOMA (see Colour Atlas OT5)
❑ in growth of keratinized squamous epithelium in middle-ear or mastoid as a result of retraction of the TM

Congenital
❑ behind an intact tympanic membrane “small white pearl", not associated with otitis media
❑ usually presents with conductive hearing loss

Acquired
❑ frequently associated with retraction pocket in pars flaccida and marginal perforations of the tympanic membrane
❑ erodes mastoid bone, then ossicles
❑ associated with chronic otitis media with painless otorrhea

Complications
❑ chronic otitis media
❑ CNS dysfunction/infection
❑ late complications: hearing loss, vertigo, facial palsy

Treatment
❑ excision via cortical, modified radical, or radical mastoidectomy depending on the extent of disease +/– tympanoplasty
❑ tympanic membrane repair and ossicle reconstruction if no sign of recurrence

MASTOIDITIS
- ❑ osteomyelitis (usually subperiosteal) of mastoid air cells, most commonly seen approximately two weeks after onset of untreated (or inadequately treated) acute suppurative otitis media
- ❑ previously common but is now rare due to rapid and effective treatment of acute otitis media with antibiotics

Presentation
- ❑ pinna displaced laterally and inferiorly
- ❑ persistent throbbing pain and tenderness over mastoid process
- ❑ development of subperiosteal abscess, post-auricular swelling
- ❑ spiking fever
- ❑ hearing loss
- ❑ otorrhea with tympanic membrane perforation (late)
- ❑ radiologic findings: opacification of mastoid air cells by fluid and interruption of normal trabeculations of cells

Treatment
- ❑ IV antibiotics with myringotomy and ventilating tubes
- ❑ cortical mastoidectomy
  - debridement of infected tissue allowing aeration and drainage
  - requires lifelong follow-up with otolaryngologist
- ❑ indications for surgery
  1. failure of medical treatment after 48 hours
  2. symptoms of intracranial complications
  3. aural discharge persisting for 4 weeks and resistant to antibiotics

OTOSCLEROSIS (see Figure 7)
- ❑ commonest cause of conductive hearing loss between 15 and 50 years of age
- ❑ autosomal dominant, variable penetrance approximately 40%
- ❑ female > male - progresses during pregnancy (hormone responsive)
- ❑ 50% bilateral

Presentation
- ❑ progressive conductive hearing loss first noticed in teens and 20s (may progress to sensorineural hearing loss if cochlea involved)
- ❑ +/– pulsatile tinnitus
- ❑ tympanic membrane normal +/– pink blush (Schwartz's sign) associated with the neovascularization of otosclerotic bone
- ❑ characteristic dip at 2,000 Hz (Carhart's Notch) on audiogram (Figure 7)

Treatment
- ❑ stapedectomy with prosthesis is definitive treatment
- ❑ hearing aid may be used, however usually not a good long term solution

CONGENITAL SENSORINEURAL HEARING LOSS
- ❑ genetic factors are being identified increasingly among the causes of hearing loss

Hereditary Defects
- ❑ non-syndrome associated (70%)
  - often idiopathic
  - autosomal recessive
- ❑ syndrome associated (30%)
  - Waardenburg's - white forelock, heterochromia iridis, wide nasal bridge and increased distance between medial canthi
  - Pendred's - goiter
  - Treacher-Collins - first and second branchial cleft anomalies
  - Alport's - hereditary nephritis

Prenatal TORCH Infections
- ❑ **T**oxoplasmosis, **O**thers (e.g. HIV), **R**ubella, **C**ytomegalovirus (CMV), **H**erpes simplex

Perinatal
- ❑ Rh incompatibility
- ❑ anoxia
- ❑ hyperbilirubinemia
- ❑ birth trauma (hemorrhage into inner ear)

Postnatal
- ❑ meningitis
- ❑ mumps
- ❑ measles

High Risk Registry (For Hearing Loss in Newborns)

- ❑ risk factors
  - low birth weight/prematurity
  - perinatal anoxia (low APGARs)
  - kernicterus - bilirubin > 25 mg/dL
  - craniofacial abnormality
  - family history of deafness in childhood
  - 1st trimester illness - CMV, rubella
  - neonatal sepsis
  - ototoxic drugs
  - perinatal infection, including post-natal meningitis
  - consanguinity
- ❑ 50-75% of newborns with sensorineural hearing loss have at least one of the above risk factors, and 90% of these have spent time in the NICU
- ❑ presence of any risk factor: Auditory Brainstem Response (ABR) study done before leaving NICU and at 3 months adjusted age
- ❑ refer for hearing assessment
- ❑ if not identified and rehabilitated within six months, intellectual deterioration in deaf children occurs
- ❑ must detect and rehabilitate hearing loss near birth in every case so that the child can reach his/her potential

PRESBYCUSIS (very common) (see Figure 7)

- ❑ hearing loss associated with aging - 5th and 6th decades
- ❑ most common cause of sensorineural hearing loss

Etiology

- ❑ hair cell degeneration
- ❑ age related degeneration of basilar membrane
- ❑ cochlear neuron damage
- ❑ ischemia of inner ear

Presentation

- ❑ progressive, gradual bilateral hearing loss initially at high frequencies, then middle frequencies
- ❑ loss of discrimination of speech especially with background noise present - patients describe people as mumbling
- ❑ recruitment phenomenon: inability to tolerate loud sounds
- ❑ tinnitus

Treatment

- ❑ hearing aid if hearing loss > 30-35 dB
- ❑ +/– lip reading and auditory training

SUDDEN SENSORINEURAL HEARING LOSS (UNILATERAL)

- ❑ presents as a sudden onset of significant hearing loss (usually unilateral) +/– tinnitus
- ❑ unexplained etiology (?autoimmune, viral, microcirculation, trauma)
- ❑ rule out transient ischemic attack (TIA) and systemic lupus erythematosus (SLE)
- ❑ CT to rule out tumour or cerebrovascular attack (CVA) if associated with any other focal neurological signs (e.g. vertigo, ataxia, abnormality of CN V or VII, weakness)
- ❑ treat with
  - low molecular weight dextran
  - corticosteroids (systemic or intratympanic)
  - bedrest
- ❑ prognosis
  - 70% resolve spontaneously within 10-14 days
  - 20% experience partial resolution
  - 10% experience permanent hearing loss

DRUG OTOTOXICITY

Aminoglycosides

- ❑ increased toxicity with oral administration
- ❑ can occur with topical preparations in patients with perforated tympanic membranes
- ❑ destroys sensory hair cells
- ❑ high frequency hearing loss develops earliest
- ❑ ototoxicity occurs days to weeks post-treatment
- ❑ streptomycin (vestibulotoxic), kanamycin and tobramycin (toxic to cochlea), gentamycin (vestibulotoxic and cochlear toxic)
- ❑ must monitor levels with peak and trough levels when prescribed, especially if patient has neutropenia, history of ear or renal problems
- ❑ q24H dosing, with amount determined by creatinine clearance not serum creatinine alone

Salicylates

- ❑ hearing loss with tinnitus, reversible if discontinued

Quinine and Antimalarials
- ❑ tinnitus
- ❑ reversible if discontinued but can lead to permanent loss
- ❑ treat drug ototoxicity with IV low molecular weight dextrans

NOISE-INDUCED SENSORINEURAL HEARING LOSS (see Figure 7)
- ❑ may be occupational, often associated with tinnitus
- ❑ 85-90 dB over months or years causes cochlear damage
- ❑ early-stage hearing loss at 4000 Hz (because this is the resonant frequency of the temporal bone), extends to higher and lower frequencies with time
- ❑ speech reception not altered until hearing loss > 30 dB at speech frequency, therefore considerable damage may occur before patient complains of hearing loss
- ❑ difficulty in discriminating, especially in situations with competing noise

Phases of Hearing Loss
- ❑ dependent on intensity level and duration of exposure
- ❑ temporary threshold shift
  - when exposed to loud sound, decreased sensitivity or increased threshold for sound
  - with removal of noise, hearing returns to normal
- ❑ permanent threshold shift
  - hearing does not return to previous state

Treatment
- ❑ hearing aid
- ❑ prevention
  - ear protectors: muffs, plugs
  - machinery which produces less noise
  - limit exposure to noise with frequent rest periods
  - regular audiologic follow-up

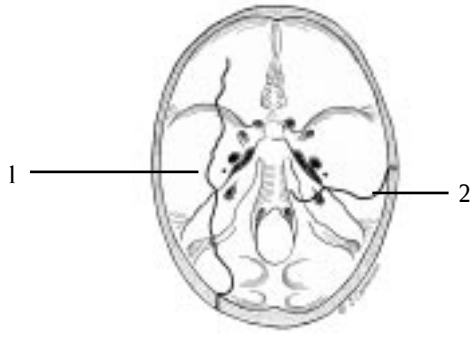
ACOUSTIC NEUROMA (AN) (see Neurosurgery Chapter)
- ❑ Schwannoma of the vestibular portion of CN VIII
- ❑ most common intracranial tumour causing hearing loss
- ❑ starts in the internal auditory canal and expands into cerebellopontine angle (CPA), compressing cerebellum and brainstem
- ❑ may be associated with Type 2 neurofibromatosis (NF2) (bilateral tumours of CN VIII in internal auditory canal, cafe-au-lait lesions, multiple intracranial lesions) (see Neurology Chapter)

Presentation
- ❑ usually presents with unilateral sensorineural hearing loss
- ❑ dizziness and unsteadiness may be present, but no true vertigo
- ❑ facial nerve palsy and trigeminal (V1) sensory deficit (corneal reflex) late complications

Diagnosis
- ❑ enhanced CT/MRI
- ❑ audiogram - puretone threshold elevated
- ❑ poor speech discrimination and stapedial reflex
- ❑ absent or significant reflex decay
- ❑ Acoustic Brainstem Reflexes (ABR) - increase in latency of the 5th wave
- ❑ electronystagmography (ENG)

Treatment
- ❑ conservative "wait and see"
- ❑ definitive management is surgical excision
- ❑ other options, such as gamma knife

TEMPORAL BONE FRACTURES



Types
1. transverse fractures
   - extends into bony labyrinth and internal auditory meatus (20%)
2. longitudinal fractures
   - extends into middle ear (80%)

- in reality, the fractures rarely adhere to either of these patterns

Figure 8. Types of Temporal Bone Fractures

Illustration by Teddy Cameron

Table 2. Features of Temporal Bone Fractures (see Figure 8)

	Transverse	Longitudinal
Incidence	10-20%	70-90%
Etiology	frontal/occiptal	lateral skull trauma
CN pathology	CN VII palsy	CN VII palsy (10-20%)
Hearing loss	sensorineural loss due to direct cochlear injury	conductive hearing loss secondary to ossicular injury
Vestibular symptoms	sudden onset vestibular symptoms due to direct semicircular canal injury (vertigo, spontaneous nystagmus)	rare
Other features	• intact external auditory meatus, tympanic membrane +/– hemotympanum • spontaneous nystagmus • CSF leak in eustachian tube to nasopharynx +/or rhinorrhea (risk of meningitis)	• torn tympanic membrane with hemotympanum • bleeding from external auditory canal • step formation in external auditory canal • CSF otorrhea • Battle's sign = mastoid ecchymoses • Raccoon eyes = periorbital ecchymoses

Diagnosis
- ❑ otoscopy
- ❑ do not syringe or manipulate external auditory meatus due to risk of inducing meningitis via TM perforation
- ❑ radiology
  - CT
- ❑ facial nerve tests (for transverse fractures), EMG, Schirmer's test, gustometry, stapedial reflexes, ENG

Treatment
- ❑ hemotympanum signifies significant force sustained, therefore monitor hearing until it returns to normal
- ❑ medical - expectant, prevent otogenic meningitis
  - IV antibiotics if suspect CSF leak (penicillin G for 7-10 days)
- ❑ surgical - explore temporal bone, indications are
  1. early meningitis (mastoidectomy)
  2. bleeding from sinus
  3. CSF otorrhea
  4. CN VII palsy (complete)
  5. gunshot wound
  6. depressed fracture of external auditory meatus

Complications
- ❑ acute otitis media +/– labyrinthitis +/– mastoiditis
- ❑ meningitis / epidural abscess / brain abscess
- ❑ post-traumatic cholesteatoma

AURAL REHABILITATION
- ❑ dependent on degree of hearing loss, communicative requirements and difficulties, motivation and expectations, age, and physical and mental abilities
- ❑ factors affecting prognosis with hearing aid/device
  - poor speech discrimination
  - narrow dynamic range (recruitment)
  - unrealistic expectations
  - cosmetic
- ❑ types of hearing aids
  - behind the ear - BTE
  - all in the ear - ITE
  - bone conduction
  - contralateral routing of signals (CROS)
- ❑ assistive listening devices
  - direct/indirect audio output
  - infrared, FM, or induction loop systems
  - telephone, television, or alerting devices
- ❑ cochlear implant
  - electrode is inserted into the cochlea to allow direct stimulation of the auditory nerve
  - for profound bilateral sensorineural hearing loss not rehabilitated with conventional hearing aids
  - established indication: post-lingually deafened adults and children

VERTIGO

EVALUATION OF THE DIZZY PATIENT

- ❑ patients can present with a wide range of subjective descriptions of their symptoms: dizziness, spinning, lightheadedness, giddiness, unsteadiness
- ❑ true vertigo is defined as an illusion of rotary movement of self or environment, made worse in the absence of visual stimuli
- ❑ it is important to distinguish vertigo from other disease entities that may present with similar complaints (e.g. cardiovascular, psychiatric, neurological, aging)
- ❑ diagnosis is heavily dependent upon an accurate history
    - description of rotary movement
    - onset and duration
    - hearing and tinnitus
    - effect of dark/eye closing
    - relation to body position
    - alcohol and drug history (antihypertensives, aminoglycosides)
    - medical history (vascular disease, anxiety disorder)

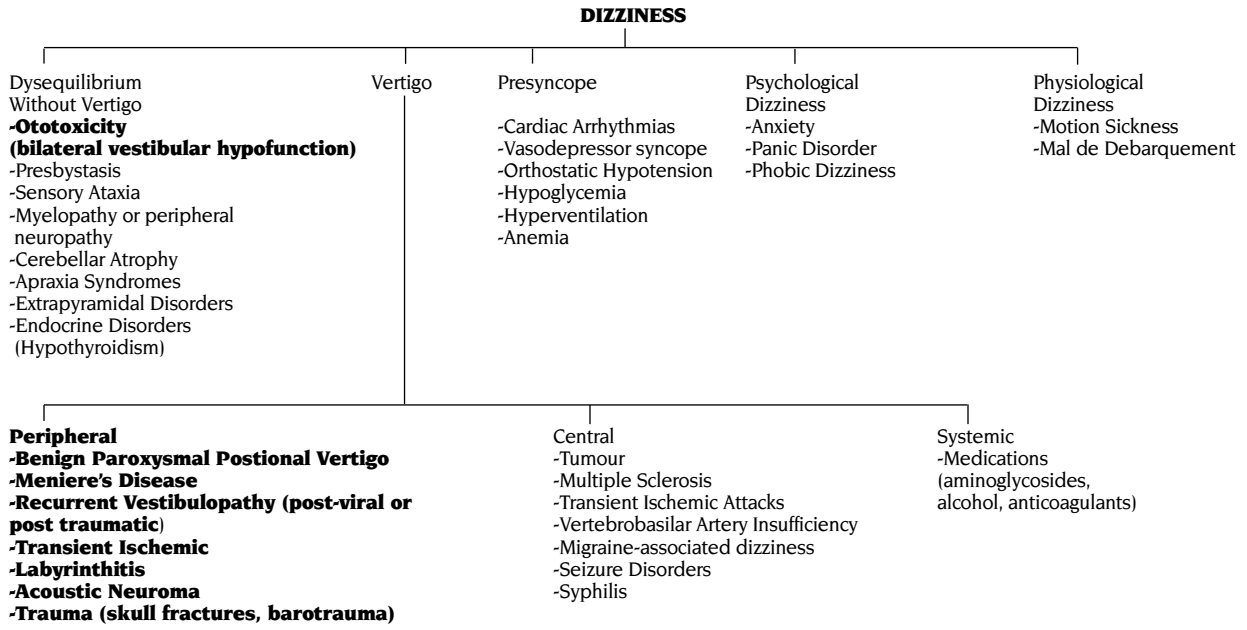


Figure 9. Differential Diagnosis of Dizziness (all those in bold = Otolaryngologist's domain)

Table 3. Differential Diagnosis of Vertigo

Condition	Duration	Hearing Loss	Tinnitus	Aural Fullness	Other Features
Benign Paroxysmal Positional Vertigo (BPPV)	seconds	none	none	none	
Meniere's Disease	minutes-hours precedes attack	uni/bilateral	+	pressure/warmth	
Recurrent Vestibulopathy	minutes to hours	none	none	none	
Vestibular Neuronitis	hours-days	unilateral	none	none	
Labyrinthitis	days	unilateral	whistling	none	recent AOM
Acoustic Neuroma (see OT14)	chronic	progressive	none	none	ataxia CN Vll palsy

Clinical Pearl

❑ **True nystagmus and vertigo will never last longer than a couple of weeks if caused by a peripheral lesion because compensation occurs; such is not true for a central lesion.**

Benign Paroxysmal Positional Vertigo (BPPV)
- ❑ most common cause for episodic vertigo
- ❑ acute attacks of transient vertigo initiated by certain head positions lasting seconds to minutes, accompanied by nystagmus that fatigues on repeated testing
- ❑ due to migration of a small flake of bone or detached mineral crystals from utricular otolith organ (cupulolithiasis) into posterior semicircular canal —> floats to rest on one of the sensitive balance organs
    - may occur following a head injury, viral infection (URTI), degenerative disease or idiopathic
    - results in slightly different signals being received by the two balance organs resulting in sensation of movement
- ❑ diagnosed by history and positive Dix-Hallpike manoeuvre (see Otoneurological Examination section)
- ❑ treat symptomatically and reassure patient that process resolves spontaneously
    - commonly treated with particle repositioning maneuvers (Epley's manoeuvre or Brandt-Daroff exercises)
    - drugs to suppress the vestibular system delay eventual recovery and are therefore not used

Meniere's Disease (endolymphatic hydrops)
- ❑ peak incidence (40-60 years)
- ❑ characterized by vertigo, fluctuations in hearing loss, tinnitus, and aural fullness, +/– drop attacks (N/V)
- ❑ vertigo (lasting minutes to hours) disappears with time and patient is left only with hearing loss
- ❑ early in the disease, hearing returns to normal in the attack-free states
- ❑ later stages are characterized by a unilateral, fluctuating low-frequency hearing loss and a persistance of tinnitus (most hearing loss becomes bilateral with time)
- ❑ attacks come in clusters and may be very debilitating to the patient, may be triggered by stress
- ❑ pathogenesis: inadequate absorption of endolymph leads to endolymphatic hydrops (over accumulated) that distorts membranous labyrinth
- ❑ treatment
    - acute management may consist of bedrest, IV antiemetics, antivertiginous drugs (Serc), and low molecular weight dextrans
    - longterm management may be
        - medical
            - low salt diet, K+ sparing diuretics (e.g. triamterene, amiloride)
            - local application of gentamicin to destroy vestibular end-organ
        - surgical - selective vestibular neurectomy or transtympanic labyrinthectomy
    - may recur in opposite ear after treatment

Recurrent Vestibulopathy
- ❑ peak age 30-50 years old, M = F
- ❑ episodic vertigo lasting hours to minutes
- ❑ no hearing loss, tinnitus, or focal neurological deficit
- ❑ etiology unknown (?post-traumatic, ?post-viral, ?deafferentation of CN VIII)
- ❑ treatment: symptomatic, most eventually go into remission

Vestibular Neuronitis
- ❑ severe vertigo with nausea, vomiting, and inability to stand or walk
- ❑ symptoms can last for 3 to 4 days (risk of dehydration from vomiting)
- ❑ attacks leave patient with unsteadiness and imbalance for months
- ❑ repeated attacks can occur
- ❑ unknown etiology (microvascular upset due to infection, autoimmune process, or a metabolic disorder)

Labyrinthitis
- ❑ sudden onset of vertigo, nausea, vomiting, whistling tinnitus, and unilateral hearing loss, with no associated fever or pain
- ❑ lasts for days
- ❑ may occur through spread from a cholesteatomic fistula or throughdirect infection after a transverse fracture of the temporal bone or post-operative infection
- ❑ treat with IV antibiotics, drainage of middle ear +/– mastoidectomy
- ❑ beware of meningeal extension and labyrinth destruction

TINNITUS

- ❑ an auditory perception in the absence of stimulation, often very annoying to the patient
- ❑ etiology
  - presbycusis (most common cause in elderly)
  - serous otitis media (most common cause in young)
  - Meniere's Disease
  - acoustic trauma
  - labyrinthitis = acoustic neuronitis
  - Acoustic Neuroma
  - multiple sclerosis (MS)
  - drugs (NSAIDs, salicylates, aminoglycosides, antimalarials, caffeine, alcohol)
- ❑ pulsatile (objective) tinnitus (rare)
  - bruits due to vascular lesions (e.g. glomus jugulare, hemangiomas, carotid body tumours, AVM, internal carotid artery bruits)
  - patulous eustachian tube
- ❑ clicking tinnitus
  - myoclonus of muscles - stapedius, tensor tympani, levator and tensor palati
  - tetany

Treatment

- ❑ avoid loud noise to prevent worsening of symptoms
- ❑ mask tinnitus
  - white noise masking devices
  - hearing aid
  - music earphones
- ❑ tinnitus workshops
- ❑ psychotherapy
- ❑ trial of tocainamide

OTALGIA

Local Causes

- ❑ furuncle (boil), usually as a result of a *Staph Aureus* infection
- ❑ foreign body in external auditory canal/impacted cerumen
- ❑ infection
  - otitis externa
  - acute otitis media and its complications
  - acute mastoiditis and its complications
- ❑ trauma to tympanic membrane and canal
- ❑ barotrauma

Referred (10 T's + 2)

- ❑ CN V and CN X refer to external canal and CN IX to middle ear

1) eustachian **T**ube
2) **T**MJ (tempromandibular joint) syndrome
3) **T**rismus (i.e. pterygoids, quinsy)
4) **T**eeth - impacted
5) **T**ongue
6) **T**onsillitis, tonsillar cancer, post tonsillectomy
7) **T**ic (CN IX) - glossopharyngeal neuralgia
8) **T**hroat - cancer of larynx, vallecula, pyriform fossa
9) **T**rachea - foreign body, tracheitis
10) **T**hyroiditis
11) Geniculate herpes and Ramsey Hunt Syndrome
12) +/– CN VII palsy

FACIAL NERVE PARALYSIS

Etiology

❑ supranuclear and nuclear (MS, poliomyelitis, cerebral tumours)

❑ infranuclear

- Bell's palsy **(see Colour Atlas OT9)**
  - diagnosis of exclusion, so must rule out other causes
  - acute onset of unilaeral, lower motor neuron (LMN) paralysis or paresis
  - idiopathic (viral, herpes zoster oticus)
  - 84% recover, 13% recurrrence
  - sequelae: corneal abrasions, "crocodile tears"
  - treat with steroids (oral prednisone), stellate ganglion block or low molecular weight dextrans
  - possible nerve decompression
- trauma: blunt, penetrating or surgical
  - acute onset
  - if paresis - function usually returns
  - surgical repair as soon as possible
- Ramsay Hunt syndrome
  - Herpes zoster infection of external auditory meatus and auricle,
  - may affect CN VII
- tumour invasion
  - in parotid gland or cerebellar pontine angle (CPA)
  - slow progression to complete paralysis
- infection: otitis media mastoiditis, direct CN VII infection, lyme disease, HIV
- birth: congenital, birth trauma (forceps delivery)
- other: MS, Guillian-Barré syndrome

Treatment

❑ treat according to etiology plus provide corneal protection with artificial tears, nocturnal lid taping, tarsorrhaphy, gold weighting of upper lid

NASAL OBSTRUCTION

Table 4. Differentia1 Diagnosis of Nasal Obstruction

Acquired	Congenital
Nasal Cavity	
Rhinitis - acute/chronic - vasomotor - allergic	Nasal dermoid Encephalocele Glioma
Polyps	
Foreign bodies	
Trauma	
Enlarged turbinates	
Tumour	
- benign - inverting papilloma	
- malignant - squamous cell carcinoma (SCC) esthesioneuroblastoma adenocarcinoma	
nasal septum	
Septal deviation	Dislocated septum
Septal hematoma/abscess	
nasopharynx	
Adenoid hypertrophy	Choanal atresia
Tumour - nasopharyngeal carcinoma	
- benign - juvenile nasopharyngeal angiofibroma	
- malignant - nasopharyngeal carcinoma	

Table 5. Nasal Discharge: Character and Associated Conditions

Character	Associated Conditions
watery/mucoid	allergic, viral, vasomotor, CSF leak
mucopurulent	bacterial, foreign body
serosanguineous	neoplasia
bloody	trauma, neoplasia, bleeding disorder, hypertension/vascular disease

ACUTE RHINITIS

- ❑ irritation of nasal mucosa due to any cause
- ❑ most common cause is common cold
- ❑ children < 5 years most susceptible
- ❑ spread by droplet contact from sneezing

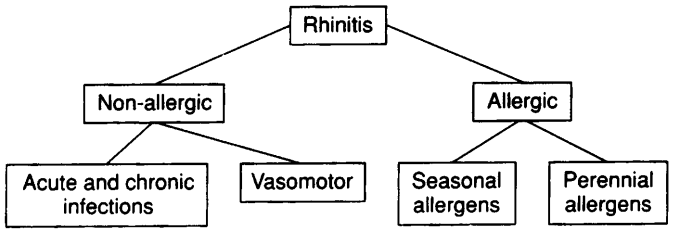


Figure 9. Causes of Rhinitis

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Etiology

- ❑ viral (ususally rhinovirus), may have secondary bacterial infection

Presentation

- ❑ irritation/burning sensation in nasopharynx; sneezing
- ❑ serous nasal discharge, may be purulent if secondary bacterial infection
- ❑ nasal obstruction, mucosal swelling and erythema
- ❑ +/– fever and malaise
- ❑ symptoms subside in 4-5 days

Complications

- ❑ sinusitis
- ❑ otitis media
- ❑ bronchitis
- ❑ tonsillitis
- ❑ pneumonia

Treatment

- ❑ rest, fluids, normal diet
- ❑ oral decongestants for symptomatic relief
- ❑ +/– analgesics, antihistamine, corticosteroid spray (e.g. triamcinolone, fluticasone, betamethasone)
- ❑ no indication for antibiotics, unless secondary bacterial infection present

Clinical Pearl

- ❑ **Congestion reduces nasal airflow and allows the nose to repair itself. Treatment should focus on the initial insult rather than at this defense mechanism.**

ALLERGIC RHINITIS (HAY FEVER)

- ❑ acute and seasonal or chronic and perennial
- ❑ perennial allergic rhinitis often confused with recurrent colds

Presentation

- ❑ early onset (< 20 years)
- ❑ past history or family history of allergies/atopy
- ❑ nasal: obstruction with pruritus, sneezing
- ❑ clear, recurrent rhinorrhea (containing increased eosinophils)
- ❑ eyes: itching of eyes with tearing
- ❑ frontal headache and pressure
- ❑ mucosa - swollen, pale, lavender colour, and “boggy"
- ❑ seasonal (summer, spring, early autumn)
    - pollens from trees
    - lasts several weeks, disappears and recurs following year at same time
- ❑ perennial
    - inhaled: house dust, wool, feather, foods, tobacco, hair, mould
    - ingested: wheat, eggs, milk, nuts
    - occurs intermittently for years with no pattern or may be constantly present

Complications

- ❑ chronic sinusitis/polups
- ❑ serous otitis media

Diagnosis

- ❑ history
- ❑ direct exam
- ❑ nasal airflow test

Treatment
- ❑ identification and avoidance of allergen
- ❑ topical steroid sprays, e.g. fluticasone (Flonase) - effective for seasonal rhinitis
- ❑ nasal irrigation with saline
- ❑ oral decongestants
- ❑ antihistamines
- ❑ injection of long-lasting steroid if severe
- ❑ desensitization by allergen immunotherapy

VASOMOTOR RHINITIS
- ❑ neurovascular disorder of nasal parasympathetic system (vidian nerve) affecting mucosal blood vessels
- ❑ nonspecific reflex hypersensitivity of nasal mucosa
- ❑ caused by
    - temperature change
    - alcohol, dust, smoke
    - stress, anxiety, neurosis
    - endocrine - hypothyroidism, pregnancy, menopause
    - parasympathomimetic drugs
    - beware of rhinitis medicamentosa: reactive vasodilation due to prolonged use (> 2 days) of nasal drops and sprays (Dristan, Otravin)

Presentation
- ❑ chronic intermittent nasal obstruction, varies from side to side
- ❑ rhinorrhea: thin, watery, worse with temperature changes, stress, exercise, EtOH
- ❑ nasal allergy must be ruled out
- ❑ mucosa and turbinates: swollen, pale between exposure
- ❑ symptoms are often more severe than clinical presentation suggests

Treatment
- ❑ elimination of irritant factors
- ❑ parasympathetic blocker (Atrovent nasal spray)
- ❑ decongestants (nose drops/oral)
- ❑ steroids (e.g. Beclomethasone)
- ❑ surgery: electrocautery, cryosurgery, laser treatment or removal of inferior or middle turbinates
- ❑ vidian neurectomy (rarely done)
- ❑ symptomatic relief with exercise (increased sympathetic tone)

ADENOID HYPERTROPHY (see Pediatric ENT section)

NASAL POLYPS (see Colour Atlas OT6)
- ❑ benign pedunculated/sessile masses of hyperplastic ethmoidal mucosa caused by inflammation
- ❑ antrochoanal polyps - (uncommon) arise from maxillary sinus and extend beyond the soft palate into the nasopharynx
- ❑ may obstruct airway

Etiology
- ❑ mucosal allergy (majority)
- ❑ sinonasal rhinosinusitis
- ❑ note: triad of polyps, aspirin sensitivity, asthma (Samter's triad)
- ❑ cystic fibrosis/bronchiectasis (child with polyps - cystic fibrosis until proven otherwise)

Presentation
- ❑ progressive nasal obstruction, hyposmia, snoring
- ❑ post-nasal drip, stringy colourless/purulent rhinorrhea
- ❑ solitary/multiple glazed, smooth, transparent mobile masses (often bilateral)

Treatment
- ❑ eliminate allergen
- ❑ steroids (preoperative prednisone) to shrink polyp
- ❑ polypectomy - treatment of choice, however, polyps have marked tendency to recur

Complications
- ❑ sinusitis
- ❑ mucocele
- ❑ nasal widening (pseudohypertelorism)

Clinical Pearl
- ❑ **Bilateral nasal polyps virtually never occur in children in the absence of Cystic Fibrosis.**

SEPTAL DEVIATION

Etiology
- ❑ developmental - unequal growth of cartilage and/or bone of nasal septum
- ❑ traumatic - facial and nasal fracture or birth injury

Presentation
- ❑ unilateral nasal obstruction (may be intermittent)
- ❑ anosmia, crusting, facial pain
- ❑ septum: S-shaped, angular deviation, spur
- ❑ compensatory middle/inferior turbinate hypertrophy on nasal space

Treatment
- ❑ if asymptomatic - expectant management
- ❑ if symptomatic - submucous resection (SMR) or septoplasty

Complications of Surgery
- ❑ post-op hemorrhage (can be severe)
- ❑ septal hematoma, septal perforation
- ❑ external deformity (saddle-nose)
- ❑ anosmia (rare but untreatable)

SEPTAL HEMATOMA
- ❑ most common in children - secondary to trauma, even mild trauma

Complications
- ❑ may lead to infection —> abscess —> cavernous sinus thrombosis
- ❑ septal perforation
- ❑ ischemic necrosis of septum and saddle deformity

Presentation
- ❑ nasal obstruction
- ❑ pain/tenderness
- ❑ occurs in anterior part of septum
- ❑ swollen nose

Treatment
- ❑ incision and drainage with nasal packing
- ❑ antibiotics

SEPTAL PERFORATION

Etiology
- ❑ trauma: surgery, physical, digital
- ❑ infection: syphilis, tuberculosis
- ❑ inflammatory: systemic lupus erythematosus (SLE)
- ❑ neoplasia: squamous/basal cell, malignant granuloma infection
- ❑ miscellaneous: cocaine sniffing, chromic gases

Presentation
- ❑ perforation seen on exam
- ❑ crusting
- ❑ recurrent epistaxis
- ❑ whistling on inspiration/expiration

Treatment
- ❑ refer suspected neoplasia for biopsy
- ❑ surgical closure for small perforations, occlusion with Silastic buttons, free fascial graft, mucosal flap

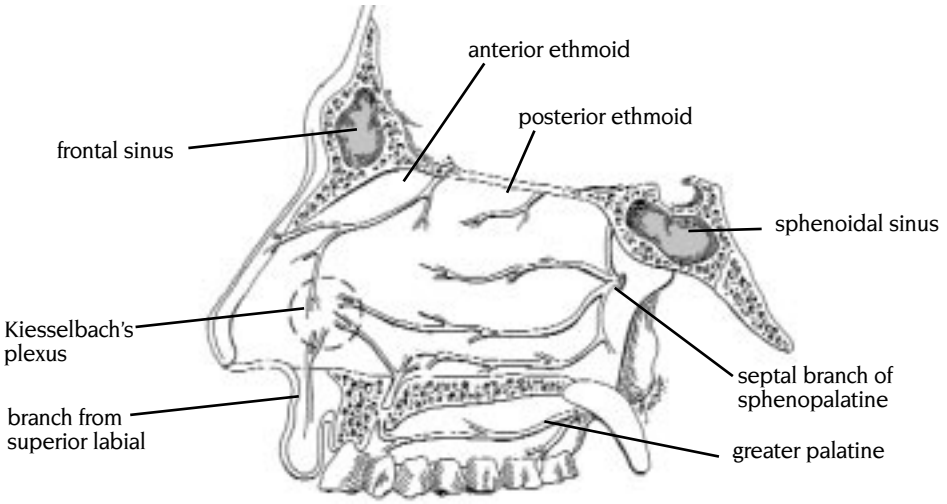

Figure 10. Nasal Septum and its Blood Supply

Illustration by Victoria Rowsell

Blood Supply to the Nasal Septum

1. Superior Posterior Septum
- internal carotid —> ophthalmic —> anterior/posterior ethmoidal

2. Posterior Septum
- external carotid —> internal maxillary —> sphenopalatine artery

3. Lower Anterior Septum
- external carotid —> facial artery —> superior labial artery

❑ these arteries all anastomose to form Kiesselbach's plexus, located at Little's area (anterior portion of the cartilaginous septum), this area is responsible for approximately 90% of nosebleeds
❑ bleeding from above middle turbinate is internal carotid, from below, external carotid

Table 6. Etiology of Epistaxis

Type	Causes
Local	Idiopathic Injection (vestibulitis) Trauma (digital, dry air) Foreign body Tumours Benign - juvenile angiofibroma (occurs in adolescent males) - polyps Maligant - squamous cell carcinoma
Systemic	Hypertension Arteriosclerosis Drugs (anticoagulants, e.g. aspirin and coumadin) Bleeding disorders Hereditary hemorrhagic telangiectasia (Osler-Weber-Rendu disease)

Treatment
❑ aim is to localize bleeding and achieve hemostasis

1. First-aid
❑ patient sits upright with mouth open (to prevent swallowing)
❑ firm pressure is applied for 5 minutes superior to nasal alar cartilages (not bony pyramid!)

2. Assess Blood Loss (it can be a potentially fatal hemorrhage)
❑ pulse, blood pressure (BP), and other signs of shock
❑ IV NS, cross match for 2 units packed RBCs if significant

3. Determine site of bleeding
- ❑ if suspicion, coagulation studies
- ❑ insert cotton pledget of 4% cocaine, visualize nasal cavity with speculum and aspirate excess blood and clots
- ❑ anterior/posterior hemorrhage defined by location in relationship to bony septum

4. Control the bleeding
- ❑ first line topical vasoconstrictors (Otrivin, cocaine)
- ❑ if first line fails and can adequately visualize bleeding source can try and cauterize with silver nitrate
- ❑ do not attempt to cauterize both sides of the septum because of the risk of septal perforation

A. Anterior hemorrhage treatment
- ❑ if fail to achieve hemostasis with cauterization
- ❑ anterior pack with half inch vaseline and bismuth-coated gauze strips or absorbable packing (i.e. Gelfoam) layered from nasal floor toward nasal roof extending to posterior choanae for 2-3 days (see Surgical Procedures section)
- ❑ can also attempt packing with Merocel or nasal tampons of different shapes

B. Posterior hemorrhage treatment
- ❑ if unable to visualize bleeding source, then usually posterior source
- ❑ insert cotton pledget with 4% cocaine
- ❑ different ways of placing a posterior pack with a Foley catheter, gauze pack or a Nasostat balloon
- ❑ bilateral anterior pack is layered into position
- ❑ antibiotics for any posterior pack or any pack in longer than 48 hours
- ❑ admit to hospital with packs in for 3 to 5 days watch for complications such as hypoxemia (naso-pulmonic reflex) and toxic shock syndrome (if present remove packs immediately)

C. If anterior/posterior packs fail to control epistaxis
- ❑ selective catheterization and embolization of branches of external carotid artery
- ❑ vessel ligation of
- ❑ anterior/posterior ethmoid artery
- ❑ internal maxillary
- ❑ external carotid

5. Prevention
- ❑ prevent drying of nasal mucosa with humidifiers, saline spray, or topical ointments
- ❑ avoidance of irritants
- ❑ medical management of hypertension

SINUSITIS

Development of Sinuses
- ❑ birth - ethmoid and small maxillary buds present
- ❑ age 9 - maxillary full grown, frontal and sphenoid cell starting
- ❑ age 18 - frontal and sphenoid cell full grown

Drainage of Sinuses
- ❑ frontal, maxillary, anterior ethmoids: middle meatus
- ❑ posterior ethmoid: superior meatus
- ❑ sphenoid: sphenoid ostium (at level of superior meatus)

Pathogenesis of Sinusitis
- ❑ inflammation of the mucosal lining of the paranasal sinuses
- ❑ anything that blocks air entry into the sinuses predisposes them to inflammation

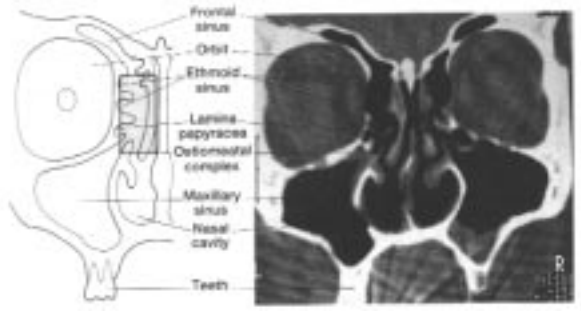

Figure 11. The Paranasal Sinuses

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ACUTE SUPPURATIVE SINUSITIS

❑ associated with
- common cold
- swimming/diving
- diseased tooth roots

❑ organisms
- S. *pneumonia* (with H. I*nfluenza*, accounts for 50%)
- H. *influenza*
- S. *aureus* - diabetic
- K*lebsiella*, P*seudomonas*, anaerobes
- in immunocompromised patients beware of fungal sinusitis —> mucormycoses 50% fatal

Presentation

❑ stuffy nose, purulent rhinorrhea
❑ malaise, fever, headache exacerbated by bleeding
❑ pressure/pain over involved sinus
- maxillary - over cheek and upper teeth
- ethmoids - medial and deep to eye
- frontal - forehead
- sphenoid - vertex

❑ mucosa hyperemic and edematous with enlarged turbinates
❑ x-ray - involved sinus opaque +/– fluid level **(see Colour Atlas OT12 and OT13)**

Treatment

❑ analgesics and decongestants - systemic and nose drops
❑ hot compresses
❑ antibiotics - oral with maxillary, and IV with frontal or ethmoid sinus involvement or orbital complications
❑ first line: amoxicillin, if failure can go to amoxicillin + clavulanic acid (Clavulin) or cefaclor
❑ surgery
- maxillary - antral puncture and lavage
- frontal/ethmoid - trephine of superior medial orbital canthus, irrigate, and drain
- sphenoid - drain via posterior ethmoids

CHRONIC SINUSITIS

❑ irreversible changes in lining membrane of one or more sinuses due to
- neglect of acute and subacute phase
- recurrent attacks or obstruction of osteomeatal complex (by polyp, deviated septum, FB, allergic rhinitis, or anatomic narrowing)

Presentation

❑ chronic nasal obstruction
❑ pain over sinus or headache
❑ halitosis
❑ yellow-brown post-nasal discharge

Treatment

❑ dependent upon involved sinus, as confirmed by coronal CT of head
❑ decongestants, antibiotics, steroids; if fails, then surgery

Surgical Treatment

❑ removal of all diseased soft tissue and bone, post-op drainage and obliteration of pre-existing sinus cavity
❑ Functional Endoscopic Sinus Surgery (FESS)

PEDIATRIC OTOLARYNGOLOGY

ACUTE OTITIS MEDIA (AOM)

❑ inflammation of middle ear associated with pain, fever, irritability, anorexia, or vomiting
❑ 60-70% of children have at least 1 episode of AOM before 3 years of age
❑ 18 months to 6 years most common age group
❑ peak incidence January to April
❑ one third of children have had 3 or more episodes by age 3

Etiology

❑ S. *pneumoniae* - 35% of cases
❑ H. *influenzae* - 25% of cases
❑ M. *catarrhalis*
❑ S. *aureus* and S. *pyogenes* (all β-lactamase producing)
❑ anaerobes (newborns)
❑ viral

Predisposing Factors
- ❑ eustachian tube dysfunction/obstruction
  - swelling of tubal mucosa
    - upper respiratory tract infection (URTI)
    - allergies/allergic rhinitis
    - chronic sinusitis
  - obstruction/infiltration of eustachian tube ostium
    - tumour - nasopharyngeal CA (adults)
    - adenoid hypertrophy (not due to obstruction but by maintaining a source of infection)
    - barotrauma (sudden changes in air pressure)
  - inadequate tensor palati function - cleft palate (even after repair)
  - abnormal spatial orientation of eustachian tube
    - Down's Syndrome (horizontal position of eustachian tube), Crouzon's, and Apert's syndrome
- ❑ disruption of action of
  - cilia of eustachian tube - ?Kartagener's syndrome
  - mucus secreting cells
  - capillary network that provides humoral factors, PMNs, phagocytic cells
- ❑ immunosupression/deficiency due to
  - chemotherapy
  - steroids
  - diabetes mellitus
  - hypogammaglobulinemia
  - cystic fibrosis

Risk Factors
- ❑ bottle feeding
- ❑ passive smoke
- ❑ crowded living conditions (day care/group child care facilities) or sick contact
- ❑ male
- ❑ family history

Pathogenesis
- ❑ obstruction of eustachian tube —> air absorbed in middle ear —> negative pressure (an irritant to middle ear mucosa) —> edema of mucosa with exudate —> infection of exudate

Presentation
- ❑ triad of otalgia, fever (especially in younger children), and conductive hearing loss
- ❑ otorrhea if tympanic membrane perforated **(see Colour Atlas OT4)**
- ❑ pain over mastoid
- ❑ infants/toddlers
  - ear-tugging
  - irritable, poor sleeping
  - vomiting and diarrhea
  - anorexia
- ❑ otoscopy of tympanic membrane **(see Colour Atlas OT1)**
  - hyperemia
  - bulging
  - contour of handle of malleus and short process disappear (middle ear effusion)

Treatment
- ❑ antibiotic treatment hastens resolution - 10 day course
  - amoxicillin - 1st line
  - trimethoprim-sulphamethoxazole (Bactrim) - if penicillin-allergic
  - AOM deemed "unresponsive" if clinical signs/symptoms and otoscopic findings persist beyond 48 hours of antibiotic treatment
  - change to broad spectrum: cefaclor (Ceclor), Clavulin, erythromycin + sulfisoxizole (Pediazole), cefixime (Suprax)
  - clarithromycin (Biaxin) for recurrent AOM
- ❑ antipyretics (e.g. acetaminophen)
- ❑ no role for decongestants in AOM
- ❑ myringotomy with tubes - indications (see Surgical Procedures section)
  1. complications of AOM suspected
  2. recurrent AOM (> 5 in a year)
  3. immunologically compromised child
  4. failure of 3 different antibiotics - fever and bulging of drum
- ❑ adenoidectomy

Complications of AOM
- ❑ extracranial
  - chronic suppurative otitis media
  - acute mastoiditis
  - facial nerve paralysis
  - febrile seizures
  - nystagmus - bacterial labyrinthitis
  - TM perforation
- ❑ intracranial
  - meningitis
  - extradural, subdural, cerebral abscess
  - petrositis
  - lateral sinus thrombosis

OTITIS MEDIA WITH EFFUSION (OME) (see Figure 7)

❑ not exclusively a pediatric disease
❑ chronic middle ear effusions (may or may not be associated with initial episode of pain and fever)
❑ follows AOM frequently in children

Presentation

❑ fullness - blocked ear
❑ hearing loss +/– tinnitus
- confirm with audiogram and tympanogram (flat)

❑ minimal pain, possibly low grade fever, no discharge
❑ otoscopy of tympanic membrane **(see Colour Atlas OT2)**
- discolouration - amber or dull grey with "glue" ear
- meniscus fluid level
- air bubbles
- retraction pockets/TM atelectasis
- foreshortening of malleus
- prominent short process
- tenting of tympanic membrane over short process and promontory of malleus
- most reliable finding with pneumotoscopy is immobility

Treatment

❑ expectant - 90% resolve by 3 months
❑ no statistical proof that antihistamines, decongestants, antibiotics clear faster than no treatment
❑ surgery: myringotomy +/- ventilating tubes +/– adenoidectomy (if enlarged) **(see Colour Atlas OT3)**
❑ ventilating tubes
- indications
  - persisting effusion > 3 months
  - hearing loss > 30 dB
  - speech delay
  - atelectasis of tympanic membrane
- function
  - equalization of middle ear pressure
  - aeration and drainage of middle ear
  - restoration of hearing and balance
  - duration - 9 to 18 months
- complications
  - tympanosclerosis
  - persistent TM perforation
  - persistent otorrhea

Complications of Otitis Media with Effusion (OME)

❑ hearing loss, speech delay, learning problems in young children
❑ chronic mastoiditis
❑ ossicular erosion
❑ cholesteatoma especially when retraction pockets involve pars flaccida or postero-superior TM
❑ retraction of tympanic membrane, atelectasis, ossicular fixation

ADENOID HYPERTROPHY

❑ size peaks at age 5 and resolves by 12 to 18 years of age
❑ increase in size with repeated URTI and allergies

Presentation

❑ nasal obstruction
- adenoid facies (open mouth, dull facial expression)
- hypernasal voice
- history of snoring
- long term mouth breather; minimal air escape through nose

❑ choanal obstruction
- chronic sinusitis/rhinitis
- obstructive sleep apnea

❑ chronic inflammation
- nasal discharge, post-nasal drip and cough
- cervical lymphadenopathy

Diagnosis

❑ enlarged adenoids on mirror nasopharyngeal exam or nasopharyngoscopic (direct) exam
❑ enlarged adenoid shadow on lateral soft tissue x-ray
❑ lateral view of the nasopharynx may show a large pad of adenoidal tissue

Complications

❑ eustachian tube obstruction leading to serous otitis media
❑ interference with nasal breathing, necessitating mouth-breathing
❑ malocclusion
❑ sleep apnea/respiratory disturbance

Indications for Adenoidectomy
- ❑ chronic upper airway obstruction with sleep disturbance/apnea +/– cor pulmonale
- ❑ chronic nasopharyngitis resistant to medical treatment
- ❑ chronic serous otitis media and chronic suppurative otitis media
- ❑ recurrent acute otitis media resistant to antibiotics
- ❑ suspicion of nasopharyngeal malignancy
- ❑ chronic sinusitis

Contraindications for Adenoidectomy
- ❑ bleeding disorders
- ❑ recent pharyngeal infection
- ❑ short or abnormal palate

ACUTE TONSILLITIS

Etiology
- ❑ Group A β-hemolytic S*treptococcus* and Group G S*treptococcus*
- ❑ P*neumococci*
- ❑ S. *aureus*
- ❑ H. *influenza*
- ❑ M. *catarrhalis*
- ❑ Epstein Barr virus (EBV)

Presentation
- ❑ symptoms
  - sore throat
  - dysphagia, odynophagia, trismus
  - malaise, fever
  - otalgia (referred)
- ❑ signs
  - cervical lymphadenopathy especially submandibular, jugulodigastric
  - tonsil enlarged, inflamed +/– spots **(see Colour Atlas OT7)**
  - strawberry tongue, scarlatiniform rash (scarlet fever)
  - palatal petechia (infectious mononucleosis)

Investigations
- ❑ CBC
- ❑ swab for C&S
- ❑ latex agglutination tests
- ❑ Monospot - less reliable children < 2 years old

Treatment
- ❑ bedrest, soft diet, ample fluid intake
- ❑ gargle with warm saline solution
- ❑ analgesics and antipyretics
- ❑ antibiotics
  - only after appropriate swab for C&S
  - start with penicillin or amoxicillin (erythromycin if allergic to penicillin) x 10 days
  - rheumatic fever risk emerges approximately 9 days after the onset of symptoms: antibiotics are utilized mainly to avoid this serious sequela and to provide earlier symptomatic relief
  - no evidence for the role of antibiotics in the avoidance of post-streptococcal glomerulonephritis

Complications
- ❑ uncommon since the use of antibiotics
  - rheumatic heart disease
  - poststreptococcal glomerulonephritis
  - arthritis
  - scarlet fever
- ❑ deep neck space infection
- ❑ abscess: peritonsillar (quinsy), intratonsillar
- ❑ sepsis

TONSILLECTOMY (see Surgical Procedures section)

Absolute Indications
- ❑ acute airway obstruction +/– cor pulmonale
- ❑ excisional biopsy for suspected malignancy (lymphoma/squamous cell carcinoma)

Relative Indications
- ❑ age 1-4 years: tonsillar hypertrophy leading to
  - sleep apnea —> cor pulmonale
  - mouth breathing —> malocclusion
  - difficulty swallowing —> FTT
- ❑ school age: chronic recurrent tonsillitis if > 5 episodes
- ❑ any complication of tonsillitis
  - quinsy, parapharyngeal abscess, retropharyngeal abscess
  - Strep bacteremia: rheumatic heart disease, nephritis, arthritis
  - Strep carrier: infective or has halitosis

AIRWAY PROBLEMS IN CHILDREN

Differential Diagnosis

Neonates
- ❑ extralaryngeal
  - choanal atresia
  - nasopharyngeal dermoid, glioma, encephalocele
  - glossoptosis - Pierre-Robin sequence, Down's syndrome, lymphangioma, hemangioma
- ❑ laryngeal
  - laryngomalacia - most common cause of stridor in children
  - laryngocele
  - vocal cord palsy (Arnold-Chiari)
  - glottic web
  - subglottic stenosis
  - laryngeal cleft
- ❑ tracheal
  - tracheoesophageal fistula
  - tracheomalacia

2-3 Months
- ❑ congenital
  - laryngomalacia
  - vascular: subglottic hemangioma (more common), innominate artery compression, double aortic arch
  - laryngeal papilloma
- ❑ acquired
  - subglottic stenosis - post intubation
  - tracheal granulation - post intubation
  - tracheomalacia - post tracheotomy and tracheoesophageal fistula (TEF) repair

Infants - Sudden Onset
- ❑ foreign body aspiration
- ❑ croup
- ❑ bacterial tracheitis
- ❑ caustic ingestion
- ❑ epiglottitis

Children and Adults
- ❑ congenital
  - lingual thyroid/tonsil
- ❑ infection
  - Ludwig's angina
  - peritonsillar-parapharyngeal abscess
  - retropharyngeal abscess
- ❑ neoplastic
  - squamous cell carcinoma (SCC) (adults): larynx, hypopharynx
  - retropharyngeal: lymphoma, neuroblastoma
  - nasopharyngeal: carcinoma, rhabdomyosarcoma
- ❑ allergic
  - angioneurotic edema
  - polyps (suspect cystic fibrosis in children)
- ❑ trauma
  - laryngeal fracture, facial fracture
  - burns and lacerations
  - post-intubation
  - caustic ingestion

SIGNS OF AIRWAY OBSTRUCTION

Clinical Pearl
❑ **Symptoms and signs of airway obstruction require a full assessment to diagnose potentially serious causes.**

Stridor
❑ note quality, timing
❑ body position important
- lying prone: subglottic hemangioma, double aortic arch
- lying supine: laryngomalacia, glossoptosis

❑ site of stenosis
- larynx or above: inspiratory stridor
- trachea: biphasic stridor
- bronchi/bronchioles: expiratory stridor

Respiratory Distress
❑ nasal flaring
❑ supraclavicular and intercostal indrawing
❑ sternal retractions
❑ use of accessory muscles of respiration
❑ tachypnea
❑ cyanosis

Feeding Difficulty and Aspiration
❑ supraglottic lesion
❑ laryngomalacia
❑ vocal cord paralysis
❑ post laryngeal cleft —> aspiration pneumonia
❑ tracheoesophageal fistula

ACUTE LARYNGOTRACHEOBRONCHITIS (CROUP)
❑ inflammation of tissues in subglottic space +/-tracheobronchial tree
❑ swelling of mucosal lining and associated with thick viscous, mucopurulent exudate which compromises upper airway (subglottic space narrowest portion of upper airway)
❑ normal function of ciliated mucous membrane impaired

Etiology
❑ viral: parainfluenzae I (most common), II, III, influenza A and B, RSV

Presentation
❑ age 4 months - 5 years
❑ preceded by URTI symptoms
❑ generally occurs at night
❑ biphasic stridor and croupy cough (loud, sea-lion bark)
❑ appear less toxic than with epiglottitis
❑ supraglottic area normal
❑ rule out foreign body and subglottic stenosis
❑ "steeple-sign" on AP of neck (x-ray) **(see Colour Atlas P3)**
❑ if recurrent croup, think subglottic stenosis

Treatment
❑ humidified O_2
❑ racemic epinephrine via nebulizer q1-2h, prn
❑ systemic corticosteroids (e.g. dexamethasone, prednisone)
❑ adequate hydration
❑ close observation for 3-4 hours
❑ intubation if severe
❑ hospitalize if poor response to steroids after 4 hours and persistent stridor at rest
❑ consider alternate diagnosis if poor response to therapy (e.g. bacterial tracheitis)

ACUTE EPIGLOTTITIS
❑ acute inflammation causing swelling of supraglottic structures of the larynx without involvement of vocal cords
❑ virtually unknown disease since HIB immunization

Etiology
❑ H. *influenza* type B
❑ relatively uncommon condition due to HiB vaccine

Presentation
❑ any age, most commonly 1-4 years
❑ rapid onset
❑ toxic-looking, fever, anorexia, restless
❑ cyanotic/pale, inspiratory stridor, slow breathing, lungs clear with decreased air entry
❑ prefers sitting up, open mouth, drooling, tongue protruding, sore throat, dysphagia

Investigations and Management
- ❑ investigations and physical examination may lead to complete obstruction, thus preparations for intubation or tracheotomy must be made prior to any manipulation
- ❑ ENT/Anesthesia emergency consult(s)
- ❑ lateral neck radiograph - cherry-shaped epiglottic swelling ("Thumb Sign" - if stable) **(see Colour Atlas P4)**
- ❑ WBC (elevated), blood and pharyngeal cultures after intubation

Treatment
- ❑ IV access with hydration
- ❑ antibiotics - IV cefuroxime, cefotaxime, or ceftriaxone
- ❑ moist air
- ❑ extubate when leak around tube occurs and afebrile
- ❑ watch for meningitis

SUBGLOTTIC STENOSIS

Congenital
- ❑ diameter of subglottis < 4 mm in neonate (due to thickening of soft tissue of subglottic space or maldevelopment of cricoid cartilage)

Acquired
- ❑ following nasotracheal intubation due to
  - long duration
  - trauma of intubation
  - large tube size
  - infection

Presentation
- ❑ biphasic stridor
- ❑ respiratory distress
- ❑ recurrent/prolonged croup

Diagnosis
- ❑ laryngoscopy
- ❑ CT

Treatment
- ❑ if soft tissue - laser and steroids
- ❑ if cartilage - do laryngotracheoplasty (LTP) single stage now done first - rarely do tracheostomy first

LARYNGOMALACIA
- ❑ most common laryngeal anomaly
- ❑ elongated omega-shaped epiglottis, short aryepiglottic fold, pendulous mucosa

Presentation
- ❑ high-pitched crowing inspiratory stridor at 1-2 weeks being constant or intermittent and more pronounced supine
- ❑ associated with feeding difficulties
- ❑ symptoms gradually subside at 18-24 months as larynx grows and thus requires no treatment

FOREIGN BODY (FB) **(see Colour Atlas P6)**

Ingested
- ❑ usually stuck at cricopharyngeus
- ❑ coins, toys
- ❑ presents with drooling, dysphagia, stridor if very big

Aspirated
- ❑ usually stuck at right mainstem bronchus
- ❑ peanuts, carrot, apple core, popcorn, balloons
- ❑ presentation
  - stridor if in trachea
  - unilateral "asthma" if bronchial, and therefore is often misdiagnosed as asthma
  - if impacts to totally occlude airway: cough, lobar pneumonia, atelectasis, mediastinal shift, pneumothorax

Diagnosis and Treatment
- ❑ inspiration - expiration chest x-ray (if patient is stable enough)
- ❑ bronchoscopy and esophagoscopy with removal
- ❑ rapid onset, not necessarily febrile or elevated WBC

DYSPHAGIA

❑ there are both oropharyngeal and esophageal causes of dysphagia
(see Gastoenterology Chapter for esophageal causes)

OROPHARYNGEAL CAUSES OF DYSPHAGIA

Oral Cavity
❑ inflammatory/infectious
❑ viral ulcers (gingivitis): Coxsackie, aphthous, Herpes
❑ Ludwig's angina
❑ trauma (including caustic ingestion)
❑ tumour
❑ structural (cleft palate)

Oropharynx
❑ inflammatory
❑ tonsillitis/tonsillar hypertrophy/pharyngitis
❑ retropharyngeal abscess
❑ tumour: tonsillar carcinoma
❑ neuromuscular disturbance: pharyngeal/palatal paralysis

Hypopharynx/Larynx
❑ tumour: intrinsic or extrinsic (thyroid mass and other neck masses)
❑ trauma (including caustic ingestion)
❑ foreign body (FB)
❑ neuromuscular disturbance
- pharyngeal/laryngeal paralysis
- cricopharyngeal spasm

❑ inflammatory: Plummer-Vinson syndrome (iron deficiency anemia anddysphagia associated with a post-cricoid web)

DEEP NECK SPACE INFECTIONS (DNSI)

❑ most deep neck space infections (DNSI) contain mixed flora
❑ most common cause is odontogenic which will have anaerobes
❑ salivary gland infections in adults
❑ pharyngeal and tonsillar infections in children
❑ note: infections of the retropharyngeal space can spread to the superior mediastinum

Presentation
❑ fever, pain, swelling
❑ +/– trismus, fluctuance, dysphagia, and dental abnormalities
❑ rule out mediastinitis if associated with dyspnea, chest pain and fever

Diagnosis
❑ CT or MRI
❑ ultrasound
❑ chest x-ray may show mediastinal widening if mediastinitis present
❑ soft tissue lateral x-rays of the neck can be diagnostic for retropharyngeal abscess

Treatment
❑ with all DNSI assess and secure airway
❑ identify and drain space, either by incision and drainage or by needle aspiration +/– U/S guidance
❑ IV antibiotics

PERITONSILLAR ABSCESS (QUINSY)
❑ cellulitis of space behind tonsillar capsule extending onto soft palate leading to abscess
❑ once thought to be due to inadequately treated chronic tonsillitis
❑ now thought to be secondary to infection of peritonsillar salivary gland
❑ can develop from acute tonsillitis
❑ unilateral, most common in 10-30 year old age group

Etiology
❑ bacterial: Group A Strep (GAS) (accounts for half of cases), S. *pyogenes*, S. *aureus*, H. *influenzae* and anaerobes

Presentation
- ❑ fever and dehydration
- ❑ sore throat and dysphagia
- ❑ extensive peritonsillar swelling but tonsil may appear normal
- ❑ edema of soft palate
- ❑ uvula deviated across midline
- ❑ "hot potato" voice (edema->failure to elevate palate->hot potatoe voice)
- ❑ increased salivation and trismus (trigeminal nerve disturbance with spasm of masticatory muscles and lockjaw)
- ❑ unilateral referred otalgia
- ❑ cervical lymphadenopathy

Complications
- ❑ may dissect inferiorly and rupture into airway or penetrate mediastinum
- ❑ may extend laterally and weaken wall of carotid artery
- ❑ may ascend and penetrate skull base

Treatment
- ❑ surgical drainage (incision or needle aspiration) - do C&S
- ❑ possible tonsillectomy at presentation or 6 weeks later
- ❑ IV antibiotics (clindamycin)
- ❑ warm saline irrigation

RETROPHARYNGEAL ABSCESS
- ❑ most commonly in young infants/children < 2 years old
- ❑ in children
  - due to accumulation of pus between posterior pharyngeal wall and prevertebral fascia
  - pus is from breakdown of lymph node in retropharyngeal tissue
  - often secondary to posterior pharyngeal trauma (e.g. endotracheal tube (ETT) or suction in neonate, popsicle stick abrasion in child)
- ❑ in adults
  - secondary to spread from parapharyngeal space due to an abscess or trauma of posterior pharyngeal wall

Presentation
- ❑ child
  - acute throat pain, difficulty swallowing, loss of appetite, speech change
  - unexplained high fever post URTI
  - stiff neck, stridor
- ❑ adult
  - dysphagia
  - odynophagia
  - symptoms of airway obstruction
  - pain and swelling in neck

Diagnosis
- ❑ lateral soft tissue radiograph showing increased soft tissue between pharyngeal airway and cervical vertebral bodies +/– trapped air
- ❑ barium swallow (rarely necessary)

Treatment
- ❑ IV antibiotics
- ❑ surgery: incision and drainage with airway secured

LUDWIG'S ANGINA
- ❑ cellulitis/inflammation of submandibular, sublingual, and submental spaces
- ❑ causes firmness of floor of mouth, tongue protrudes upward and back causing airway obstruction

Etiology
- ❑ most commonly secondary to a dental infection (multiple organisms involved usually - including anaerobic bacilli)
- ❑ suppurative lymph node in submaxillary space

Treatment
- ❑ incision through midline and prepare for possible tracheostomy because of the difficulty in intubation
- ❑ IV penicillin + metronidazole/clindamycin

ACUTE TONSILLITIS and TONSILLECTOMY
(see Pediatric ENT and Surgical Procedures section)

HOARSENESS

- ❑ hoarseness: change in voice quality, ranging from voice harshness to voice weakness
- ❑ reflects abnormalities anywhere along the vocal tract from oral cavity to lungs
- ❑ dysphonia: describes a general alteration in voice quality
- ❑ aphonia: no sound is emanated from vocal folds

Clinical Pearl
- ❑ **If hoarseness present for > 2 weeks in a smoker, laryngoscopy must be done to rule out cancer.**
- ❑ **Acute < than 2 weeks, chronic > 2 weeks.**

Table 7. Differential Diagnosis of Hoarseness

1. Infectious	❑ acute viral laryngitis ❑ bacterial tracheitis/laryngitis ❑ laryngotracheobronchitis (croup)
2. Inflammatory	❑ from gastro-esophageal reflux (GERD), smoke irritation, or chronic cough • vocal cord polyps • Reinke's edema • contact ulcers or granulomas • vocal cord nodules
3. Trauma	❑ external laryngeal trauma ❑ endoscopy and endotracheal tube
4. Neoplasia	❑ benign tumours • vocal cord polyps • papillomas • chondromas, lipomas, hemangiomas ❑ malignant tumours • squamous cell carcinoma (SCC) • Kaposi's sarcoma
5. Cysts	❑ retention cysts ❑ laryngoceles
6. Systemic	❑ endocrine • hypothyroidism • virilization ❑ connective tissue disease • rheumatoid arthritis (RA) • SLE ❑ angioneurotic edema
7. Neurologic (vocal cord paralysis)	❑ central lesions • cerebrovascular accident (CVA) • head injury • multiple sclerosis (MS) • Arnold-Chiari • neural tumours ❑ peripheral lesions • tumours: glomus jugulare, thyroid, bronchogenic, esophageal, neural • surgery: thyroid surgery, cardiovascular or thoracic/esophageal surgery • cardiac: left atrial enlargement, aneurysm of aortic arch ❑ neuromuscular • myasthenia gravis • presbylaryngeus • spasmodic dysphonia
8. Functional	❑ psychogenic aphonia (hysterical aphonia) ❑ habitual aphonia ❑ ventricular dysphonias
9. Congenital	❑ webs, atresia ❑ laryngomalacia

ACUTE LARYNGITIS

Etiology
- ❑ viral +/– URTI - influenza, adenovirus, GAS
- ❑ voice abuse
- ❑ toxic fume inhalation

Presentation
- ❑ URTI symptoms and hoarseness, aphonia, cough attacks, +/– dyspnea
- ❑ indirect laryngoscopy shows true vocal cords erythematous and edematous with vascular injection and normal cord mobility

Treatment
- ❑ self-limited
- ❑ voice rest with humidification to prevent further irritation of inflamed cords
- ❑ removal of irritants (e.g. smoking)
- ❑ if bacterial - treat with antibiotics

CHRONIC LARYNGITIS
- ❑ long standing inflammatory changes in laryngeal mucosa

Etiology
- ❑ repeated attacks of acute laryngitis
- ❑ exposure to irritating dust/smoke
- ❑ voice abuse
- ❑ esophageal disorders: Zenker's diverticulum/hiatus hernia/GERD
- ❑ systemic: allergy, hypothyroidism, Addison's

Presentation
- ❑ longstanding hoarseness and vocal weakness - rule out malignancy
- ❑ indirect laryngoscopy - cords erythematous, thickened with normal mobility

Treatment
- ❑ remove offending cause
- ❑ treat related disorders
- ❑ speech therapy with voice rest
- ❑ +/– antibiotics, +/– steroids to decrease inflammation

VOCAL CORD POLYPS
- ❑ commonest benign tumour of vocal cords
- ❑ usually in men between 30 and 50 years of age

Etiology
- ❑ vocal abuse or misuse
- ❑ agents causing laryngeal inflammation (reflux, allergies, tobacco)

Presentation
- ❑ hoarseness, aphonia, cough attacks +/– dyspnea
- ❑ laryngoscopy shows polyp on free edge of vocal cord on a pedicle or sessile

Treatment
- ❑ avoid causative factors
- ❑ remove with endoscopic laryngeal microsurgery

VOCAL CORD NODULES
- ❑ called screamer's or singer's nodules
- ❑ more frequently occur in females, singers and children

Etiology
- ❑ chronic voice abuse
- ❑ URTI, smoke, alcohol

Presentation
- ❑ laryngoscopy shows red, soft looking nodules, often bilateral at the junction of the anterior and middle 1/3 of vocal cords
- ❑ chronic nodules may become fibrotic, hard and white

Treatment
- ❑ voice rest
- ❑ speech therapy
- ❑ avoidance of aggravating factors
- ❑ surgery is rarely indicated

BENIGN LARYNGEAL PAPILLOMAS
❑ biphasic distribution - birth to puberty (most common laryngeal tumour) and adulthood

Etiology
❑ human papilloma virus (HPV) types 6, 11
❑ ?hormonal influence

Presentation
❑ hoarseness and airway obstruction
❑ can seed into tracheobronchial tree
❑ recurs after treatment
❑ some juvenile papillomas resolve spontaneously at puberty
❑ papillomas in adults may undergo malignant degeneration
❑ laryngoscopy shows wart-like lesions in supraglottic larynx and trachea

Treatment
❑ CO_2 laser and microsurgery
❑ +/– interferon if pulmonary involvement

LARYNGEAL CARCINOMA (see Neoplasms of the Head and Neck section)

SALIVARY GLANDS

❑ major salivary glands: paired parotid, submandibular, and sublingual glands

SIALOADENITIS
❑ sialoadenitis: inflammation of salivary glands

Etiology
❑ obstructive vs. non-obstructive
❑ bacterial: (commonly S. *aureus*) patient prone to bacterial infection when salivary flow is decreased or obstructed
❑ viral: most common infectious cause

Presentation
❑ acute onset of pain and edema of parotid or submandibular gland that may lead to marked swelling
❑ +/– fever
❑ +/– leukocytosis
❑ +/– suppurative drainage from punctum of the gland
❑ mumps usually presents with bilateral parotid enlargement, +/– sensorineural hearing loss, +/– orchitis

Diagnosis
❑ imaging with U/S employed to differentiate obstructive vs. non-obstructive sialoadenitis

Treatment
❑ bacterial: treat with cloxacillin +/- abscess drainage
❑ viral: no treatment

SIALOLITHIASIS
❑ ductal stone with chronic sialadenitis
❑ 80% occurs in the submandibular gland, <20% in parotid gland, ~1% in sublingual gland
❑ predisposing factors:
  - any condition causing duct stenosis or a change in salivary secretions (e.g. dehydration, diabetes, EtOH,hypercalcemia)

Presentation
❑ pain and tenderness over involved gland
❑ intermittent swelling related to meals

Diagnosis
❑ by digital palpation of calculi
❑ sialogram

Treatment
❑ remove calculi by dilating duct and orifice or excision through floor of mouth
❑ if calculus is within the gland parenchyma then the whole gland must be excised

SALIVARY GLAND MANIFESTATIONS OF SYSTEMIC DISEASE
❑ Sjögrens syndrome: chronic disorder characterized by immune-mediated destruction of exocrine glands (triad of conjunctivitis sicca, xerostomia, parotidenlargement) diffuse non-tender, asymptomatic enlargement of the parotid glands and occasionally other salivary glands
❑ bulemia nervosa: bilateral swelling of parotid glands, approximately 30% of patients will have resolution with control of bulemia

NECK MASSES

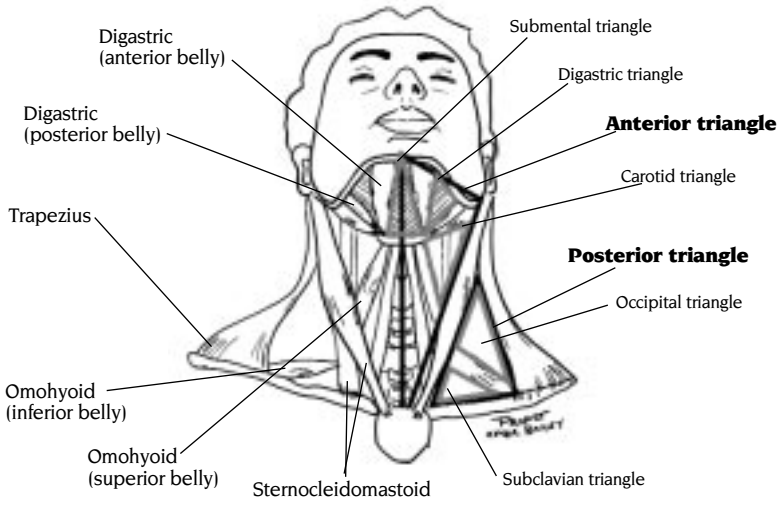


Figure 11. Anatomical Triangles of the Neck

Illustration by Evan Propst

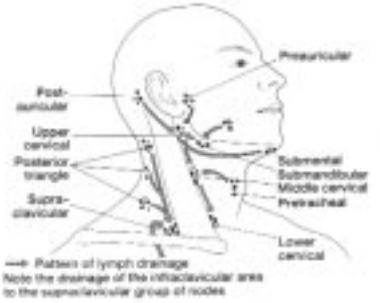


Figure 12. Lymphatic Drainage of the Neck

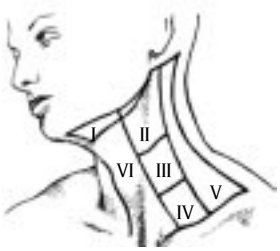


Figure 13. Zones of the Neck

Reproduced with permission from Churchill Livingston, Dhillon, R.S, East, C.A. *Ear, Nose and Throat and Head and Neck Surgery*. Churchill Livingston, UK, 1994.

The stemocleidomastoid divides the neck into two triangles (see Figure 11)

❑ Anterior triangle
- bounded by anterior border of sternocleidomastoid, midline of neck and lower border of mandible
- divided into
  - submental triangle: bounded by both anterior bellies of digastrics and hyoid bone
  - digastric triangle: bounded by anterior and posterior bellies of digastric, and inferior border of mandible
  - carotid triangle: bounded by sternocleidomastoid, anterior belly of omohyoid, and posterior belly of digastric
- contains tail of parotid, submandibular gland, hypoglossal nerve, carotid bifurcation and lymph nodes

❑ Posterior triangle
- bounded by posterior border of sternocleidomastoid, anterior border of trapezius, and middle third of clavicle
- divided into
  - occipital triangle: superior to posterior belly of the omohyoid
  - subclavian triangle: inferior to posterior belly of omohyoid
- contains spinal accessory nerve and lymph nodes

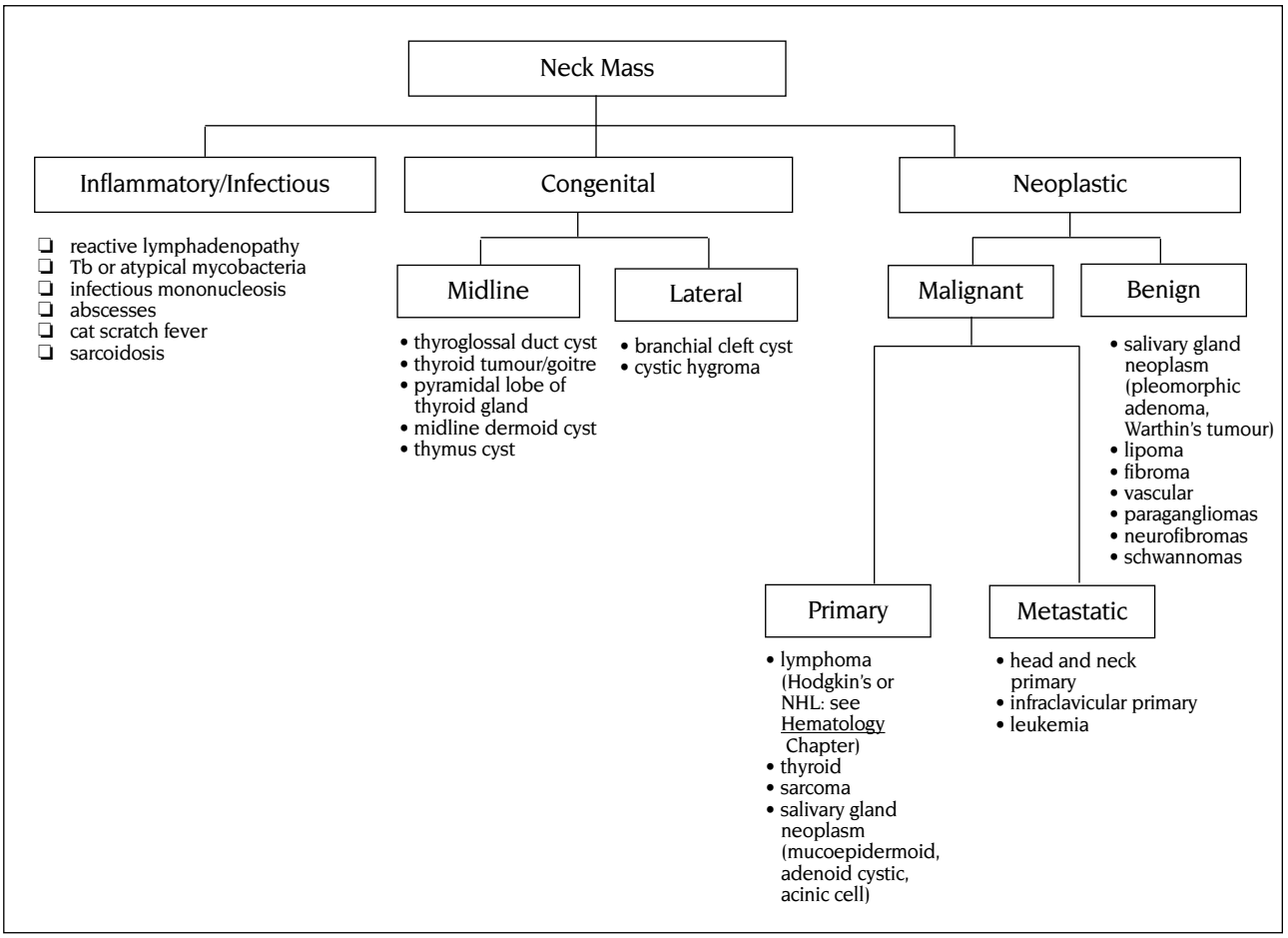
APPROACH TO A NECK MASS

❑ ensure that the neck mass is not a normal neck structure (hyoid, transverse process of C1 vertebra)

❑ duration
- if 7 days: inflammatory
- if 7 months: neoplastic
- if 7 years: congenital

Table 8. Acquired Causes of Neck Lumps According to Age	
Age(years)	**Possible Causes of Neck Lump**
< 20	inflammatory neck nodes (e.g. tonsillitis, infectious mononucleosis) lymphoma
20-40	salivary gland pathology (e.g calculi, infection, tumour) thyroid pathology (e.g. goitre, infection, tumour) granulomatous disease (e.g. TB, sarcoidosis) HIV
> 40	primary or secondary malignant disease

DIFFERENTIAL DIAGNOSIS OF A NECK MASS



EVALUATION

Investigation

- ❑ history and physical (including nasopharynx and larynx)
- ❑ indirect tests - supply information about physical characteristics of mass
  - WBC - infection vs. lymphoma
  - Mantoux TB test
  - thyroid function tests and scan
  - neck U/S
  - CT scan
  - angiography - vascularity and blood supply to mass
- ❑ direct test - for histologic examination
  - fine needle aspiration (FNA) - less invasive
  - needle biopsy
  - open biopsy-for lymphoma
  - radiologic exam of stomach, bowel and sinuses
  - search for the primary tumour (panendoscopy: nasopharyngoscopy, laryngoscopy, bronchoscopy with brushings, esophagoscopy)
  - biopsy of normal tissue of nasopharynx, tonsils, base of tongue and hypopharynx
- ❑ if primary found (95%), stage and treat
- ❑ if primary still occult (5%) - excisional biopsy of node for diagnosis, manage with radiotherapy or neck dissection (squamous cell carcinoma)

CONGENITAL NECK MASSES IN DETAIL

Branchial Cleft Cysts/Fistulae (see Colour Atlas OT11)

- ❑ at 6th week of development, the second branchial arch grows over the third and fourth arches and fuses with the neighbouring caudal pre-cardial swelling forming the cervical sinus
- ❑ branchial fistula formed by persistence of external opening of sinus while persistent parts of the cervical sinus without an external opening cause branchial cysts
- ❑ 2nd branchial cleft cysts most common
- ❑ clinical presentation
  - fistulas with an internal or external communication usually manifest during infancy as a small opening anterior to the sternocleidomastoid muscle
- ❑ branchial cysts that do not have an external or internal opening present in teens and twenties as a smooth painless slowly enlarging lateral neck mass, often following an acute URTI infection
- ❑ treatment
  - surgical removal of cyst or fistula tract
- ❑ if infected - allow infection to settle before removal

Thyroglossal Duct Cysts (see Colour Atlas OT10)

- ❑ thyroid originates as ventral midline diverticulum of floor of pharynx caudal to junction of 1st and 2nd branchial arches (foramen cecum)
- ❑ thyroid migrates caudally along a tract ventral to hyoid then curves underneath and down to cricoid with thyroglossal duct cysts being vestigial remnants of tract
- ❑ clinical presentation
  - usually presents in the second to fourth decades as a midline cyst that elevates with swallowing and tongue protrusion
- ❑ treatment
  - consists of pre-operative antibiotics to reduce inflammation followed by complete excision of cyst and tract up to foramen cecum at base of tongue with removal of central portion of thyroid (Sistrunk procedure)

Cystic Hygroma

- ❑ lymphangioma arising from vestigial lymph channels of neck
- ❑ clinical presentation
  - usually presents by age 2 as thin-walled cyst in tissues from floor of mouth down to mediastinum, usually in posterior triangle or supraclavicular area
  - infection causes a sudden increase in size
- ❑ treatment
  - surgical excision if it fails to regress - difficult dissection due to numerous cyst extensions

NEOPLASMS OF THE HEAD AND NECK

Table 9. Summary of Head and Neck Neoplasia

Location	Presentation	Risk/Etiological Factors	Diagnosis	Treatment
Lip	white patch on lip lip ulcer	UV light poor hygiene smoking/EtOH	biopsy	1° surgery radiation 2nd line
Salivary Gland	painless mass	radiation nickel exposure smoking/EtOH	fine needle biopsy CT	surgery
Oral Cavity	neck mass ulcer +/– bleeding dysphagia/sialorrhea dysphonia	smoking/EtOH poor hygiene	biopsy	1° surgery radiation 2nd line
Oropharynx	odynophagia otalgia enlarged tonsil fixed tongue with trismus	smoking and EtOH	biopsy	1° radiation surgery 2nd line
Nose/Paranasal Sinus	nasal obstruction epistaxis dental pain/numbness	hardwood dust nickel chromium	clinical suspicion on CT biopsy	surgery + radiation
Nasopharynx	nasal obstruction neck mass epistaxis unilateral SOM	EBV salted fish nickel exposure poor hygiene Southern Chinese	flexible scope biopsy CT/MRI	1° radiation surgery 2nd line
Hypopharynx	pain and dysphagia otalgia cervical node hoarseness	smoking and EtOH	rigid scope CXR CT	1° radiation surgery 2nd line
Larynx	dysphagia, otalgia odynophagia hoarseness foreign body feeling dyspnea/stridor cough/hemoptysis	smoking and EtOH	indirect and direct laryngoscopy CT	1° radiation surgery 2nd line
Thyroid	thyroid mass vocal cord paralysis cervical nodes hyper/hypo thyroid	radiation exposure family history	see figure 14	1° surgery I^{131} for metastatic deposits

- ❑ 6-8% of all malignancies in the body
- ❑ most lesions are squamous cell carcinomas
- ❑ historically M>F however increased incidence in female population in last 10-15 years due to increased prevalence of smoking in females

PRINCIPLES OF MANAGEMENT

- ❑ initial metastatic screen includes chest x-ray
- ❑ scans of liver, brain, and bone only if clinically indicated
- ❑ TMN (tumour, metastases, nodes) classification varies slightly depending on the specific type of head and neck tumour
- ❑ TNM classification widely used for staging in order to:
  - guide treatment planning
  - indicate prognosis
  - assist in evaluating results of treatment
  - facilitate accurate exchange of information
- ❑ treatment depends on
  - histologic grade of tumour
  - stage
  - physical and emotional situation of patient
  - facilities available
  - skill and experience of the oncologist and team
- ❑ in general
  - primary surgery for malignant tumours of the oral cavity with radiotherapy reserved for salvage or for poor prognostic indicators
  - primary radiotherapy for malignancies of the nasopharynx, oropharynx, hypopharynx, and larynx with surgery reserved for salvage
  - very minor role for chemotherapy in tumours of the head and neck

CARCINOMA OF THE LIP

- ❑ 50-70 year age group
- ❑ whites > blacks
- ❑ M:F = 30:1
- ❑ 95% squamous cell carcinoma (SCC)

Etiology

- ❑ UV light - to lower lip
- ❑ poor oral hygiene
- ❑ smoking and alcohol contribute but are less significant than in other head and neck SCC

Presentation

- ❑ 85% lower lip
- ❑ dyskeratosis manifests as white patch on lip (actinic chelitis)
- ❑ ulcer formation may indicate carcinoma

Diagnosis

- ❑ incisional biopsy

Treatment

- ❑ primary surgery including wedge excision with primary closure and careful approximation of vermilion border
- ❑ local flap may be required to repair an extensive surgical defect
- ❑ radiotherapy second line - for salvage or extensive disease

Prognosis

- ❑ 85% 5-year survival following surgery
- ❑ 80% 5-year survival following radiation therapy

SALIVARY GLAND NEOPLASMS

- ❑ M=F
- ❑ 80% of salivary gland tumours are parotid
- ❑ submandibular tumours uncommon (10%), sublingual rare (1%)
- ❑ only 20% of parotid swellings are malignant, whereas 75% of submandibular gland swellings are malignant
- ❑ generally the smaller the gland the greater chance of malignancy

Pathology

- ❑ malignant
  - mucoepidermoid (low vs. high grade) 40%
  - adenoid cystic 30%
  - acinic cell 5%
  - malignant mixed 5%
  - lymphomas 5%
  - adenocarcinoma
- ❑ benign
  - benign mixed (pleomorphic adenoma) 80%
  - Warthin's tumour (5-10% bilateral M>F) 10%
  - cysts, lymph nodes and adenomas 10%
  - oncoctyoma <1%

Parotid Gland Tumour

- ❑ painless slow-growing mass
- ❑ if bilateral, suggests benign process (Warthin's tumour, Sjögren's, bulemia, mumps) or possible lymphoma
- ❑ signs suggestive of malignancy
  - pain or CN VII involvement
  - rapid growth
  - involvement or invasion of overlying skin
  - facial nerve dysfunction
  - cervical lymphadenopathy

Diagnosis

- ❑ fine needle biopsy
- ❑ CT scan to determine depth of tumour

Treatment

- ❑ surgery is the treatment of choice for salivary gland neoplasms
- ❑ benign tumours are also excised due to small but potential risk of malignant transformation of pleomorphic adenoma
- ❑ superficial lesion
  - superficial parotidectomy above plane of CN VII, +/– radiation
  - incisional biopsy contraindicated
- ❑ deep lesion
  - near-total parotidectomy sparing as much of CN VII as possible
  - if CN VII involved then it is removed and cable grafted

Prognosis
- ❑ benign: excellent, although pleomorphic adenomas may recur (< 5%)
- ❑ mucoepidermoid: good if low grade - 80% 5-year survival
- ❑ others: fair, but tend to recur - 40% 5-year survival
- ❑ if neck nodes involved: 20% 5-year survival

CARCINOMA OF THE ORAL CAVITY
- ❑ oral cavity consists of the lip (described above) anterior/oral tongue, the floor of mouth, the alveolus, the retromolar trigone, the buccal mucosa, and the hard palate above
    - 1.5-3% of all cancers occurring in North America
    - most common site of head and neck cancers
- ❑ 50% of oral cavity cancer occurs on the anterior 2/3 of the tongue
- ❑ historically, far more prevalent among males, but recent increase in female smokers has changed this
- ❑ 50 to 60 year old age group
- ❑ 95% squamous cell (others include salivary gland: mucoepidermoid, adenoid cystic, acinic cell, also sarcoma and melanoma)

Etiology
- ❑ heavy smoking (note smokeless tobacco)
- ❑ alcohol (synergistic with tobacco)
- ❑ association with poor oral hygiene, chronic dental irritation, oral lichen planus, mucosal atrophy
- ❑ leukoplakia or erythroplakia may signify pre-malignant lesion or carcinoma in situ

Presentation
- ❑ 30% present as an asymptomatic mass in the neck
- ❑ ulcer with raised edges +/– bleeding
- ❑ pain with radiation to ear and neck
- ❑ dysphagia or dysphonia may occur
- ❑ oral fetor
- ❑ sialorrhea
- ❑ 10-15% of oral cavity tumours have cervical metastases at time of presentation
- ❑ lymph node metastasis in tumours of tongue and anterior floor of mouth tend to involve the submental and upper deep jugular chains
- ❑ purplish brown lesions on palate or buccal mucosa suggest Kaposi's sarcoma in HIV patients

Diagnosis
- ❑ adequate visualization is key
- ❑ small local biopsy of lesion
- ❑ imaging studies generally not required unless mandibular involvement is suspected or planning extensive resection

Treatment
- ❑ carcinoma of the oral cavity is primarily a surgical problem with post-operative radiotherapy reserved for patients with poor prognostic indicators (see below)
- ❑ primary radiotherapy occasionally employed in older or infirm patients
- ❑ surgery consists of:
    - partial/total glossectomy +/– mandibular resection
    - neck dissection if > 2 cm lesion or palpable nodes
    - reconstruction: none (if small defect), skin grafts, fascio/osseocutaneous vascularized free flaps, dental plates

Prognosis
- ❑ poor prognostic indicators include:
    - site of tumour (tongue worse than floor of mouth) and deep invasion
    - multiple positive cervical nodes
    - extra-capsular spread
    - peri-neural or peri-vascular involvement
    - close (< 5 mm) surgical margins
- ❑ early stage (T1 and T2) 75% disease free survival at 5 years
- ❑ late stage (T3 and T4) 30-35% disease free survival at 5 years
- ❑ no change in mortality in last 15-20 years but significant decrease in morbidity due to new reconstructive and rehabilitative techniques

CARCINOMA OF THE OROPHARYNX

- ❑ oropharynx consists of the tongue base (area behind the circumvallate papillae) to the back of the pharynx including the tonsillar fossae and pillars, and the soft palate down to the superior aspect of the supraglottis
- ❑ M:F = 4:1
- ❑ 50 to 70 year old age group
- ❑ etiologic agents include smoking and alcohol abuse
- ❑ 90% squamous cell carcinoma (SCC) - poorly differentiated

Presentation

- ❑ tend to present late (especially tongue base)
- ❑ odynophagia
- ❑ otalgia
- ❑ indistinct speech – "hot potato" voice
- ❑ ulcerated/enlarged tonsil
- ❑ oral fetor
- ❑ bleeding with blood-stained sputum
- ❑ tongue fixed with trismus
- ❑ induration of tonsil or tongue base
- ❑ 60% have nodal metastases at presentation (15% bilateral) - including small lesions
- ❑ parapharyngeal and retropharyngeal nodes at risk
- ❑ 7% distant metastases to lung, bone and liver

Diagnosis

- ❑ clinical suspicion
- ❑ confirmatory biopsy

Treatment

- ❑ radiotherapy is primary modality with surgery reserved for salvage
- ❑ surgery depends on extent of disease and may employ composite resection, +/– neck dissection and flap reconstruction
- ❑ radiotherapy preferred modality due to high morbidity associated with surgery and inaccessibility of at-risk nodal groups

Prognosis

- ❑ site dependent
- ❑ base of tongue: control rates for T1 lesions reported at > 90%, however poor control rates (13-52%) reported for T4 lesions
- ❑ tonsils: cure rates of 90-100% reported for T1 and T2 lesions using external beam radiation; control rates for advanced lesions are very poor - 15-33% reported for T4 lesions

CARCINOMA OF THE NOSE AND PARANASAL SINUSES

- ❑ rare tumours with decreased incidence over the last 5-10 years
- ❑ most frequently presents during the 5th to 7th decades
- ❑ risk factors - dust from hard woods (ethmoid sinus and nose), nickel (maxillary sinus cancer), chromium
- ❑ 99% occur in maxillary and ethmoid sinuses
- ❑ 75-80% squamous cell carcinoma
- ❑ 10% arise from minor salivary glands (i.e. adenoid cystic + mucoepidermoid)
- ❑ 10% sarcomas

Presentation

- ❑ symptoms begin to occur after tumour has invaded through the bony confines of the sinus
- ❑ depends where the erosion through bone has occurred
    - nose - nasal obstruction, epistaxis, pain
    - orbit - proptosis, diplopia, ophthalmoplegia, pain, epiphora due to nasolacrimal duct obstruction
    - nerves - numbness, palatal palsy, CN VII palsy, facial pain
    - dental - tooth/oral pain, loosening of teeth
    - skin - occurs late
    - intracranial or skull base extension - headache

Diagnosis

- ❑ based on clinical suspicion
- ❑ confirmed with CT or MRI (CT used routinely)
- ❑ biopsy for histopathology

Treatment

- ❑ almost all sinus cancers are treated with a combined approach involving surgery and post-operative radiotherapy

Prognosis

- ❑ overall 5-year survival = 25% (poor due to late presentation)
    - 55% if inferior antral involvement only

CARCINOMA OF THE HYPOPHARYNX

- ❑ continuous with the oropharynx above and extending inferiorly to the esophagus, the hypopharynx includes the area from the tip of the epiglottis to the lower surface of the cricoid cartilage
- ❑ 3 areas: 1) posterior pharyngeal wall (10% of tumours); 2) pyriform sinus (60%); 3) post-cricoid space (30%)
- ❑ 8-10% of all head and neck malignancies
- ❑ 95% squamous cell carcinoma
- ❑ 50-60 year old age group; M>F
- ❑ etiological factors include tobacco, alcohol
- ❑ associated with Plummer-Vinson syndrome (post-cricoid region)

Presentation

- ❑ often presents late
- ❑ odynophagia
- ❑ dysphagia
- ❑ referred otalgia
- ❑ cervical node
- ❑ +/– hoarseness

Diagnosis

- ❑ clinical suspicion - definitive diagnosis often by rigid endoscopy
- ❑ chest x-ray to rule out pulmonary metastases
- ❑ CT to evaluate deep extension

Treatment

- ❑ radiation employed as primary modality
- ❑ rigid endoscopy to determine 8-10 week post-treatment response
- ❑ favorable response to radiotherapy can be followed for 5-10 years
- ❑ if radiation fails: surgical resection of larynx and hypopharynx +neck dissection
- ❑ reconstructive options include closure of the pharynx, pedicle flap reconstruction (e.g. pectoralis major) free jejunal interposition, and gastric pull-up

Prognosis

- ❑ generally poor: 60% cure rates have been reported for T2-T3, 25-40% five year survival with T4 lesions
- ❑ post-operative morbidity with fistula formation in 20-25% of previously irradiated patients
- ❑ gastric pull-up associated with 14% peri-operative mortality

CARCINOMA OF THE NASOPHARYNX (NP)

- ❑ the nasopharynx is the cuboidal space bounded anteriorly by the posterior choanae of the nose, posteriorly by the clivus, C1 and C2 vertebrae, superiorly by the body of the sphenoid and inferiorly by the soft palate
- ❑ the eustachian tubes open onto the lateral walls of the NP which are comprised of pharyngeal fascia
- ❑ incidence 0.8/100 000; markedly increased among those of Southern Chinese origin (100 fold increased incidence)
- ❑ 50-59 year old age group, M:F = 2.4:1
- ❑ etiological factors include EBV, salted fish consumption, nickel exposure, poor hygiene
- ❑ squamous cell carcinoma most common (approximately 90%)
- ❑ lymphoma (approximately 10%)

Presentation

- ❑ neck mass at presentation in 60-90% (note: deep posterior cervical node at mastoid tip)
- ❑ nasal obstruction/discharge, epistaxis
- ❑ voice change, mandibular neuralgia, decrease in soft palate mobility, dysphagia
- ❑ unilateral serous otitis media and/or hearing loss
- ❑ proptosis (secondary to tumour extension into orbit)
- ❑ cranial nerve involvement in approximately 25% (CN III-VI can be involved by cavernous sinus extension; CN IX-XII can be involved by retropharyngeal space encroachment or lymphadenopathy)

Diagnosis

- ❑ clinical findings (include digital palpation)
- ❑ flexible nasopharyngoscopy for direct visualization
- ❑ biopsy with topical anesthetic
- ❑ CT/MRI for assessment of extent of tumour invasion and involvement of adjacent structures

Treatment

- ❑ primary radiotherapy of nasopharynx and adjacent parapharyngeal and cervical lymphatics is the treatment of choice
- ❑ +/– radical neck dissection for salvage and recurrence
- ❑ use of chemotherapy controversial

Prognosis

- ❑ excellent local control possible for T1 lesions (90-95% control rates reported)
- ❑ 5 year survival rates vary according to stage:
    - I: 78%; II: 72%; III: 50-60%; IV: 36-42%

CARCINOMA OF THE LARYNX

- ❑ squamous cell carcinoma most common
- ❑ 45% of head and neck carcinoma
- ❑ common between 45-75 years of age
- ❑ M:F = 10:1
- ❑ etiologic agents include heavy smoking and heavy alcohol consumption

Classification

- ❑ classified according to site within larynx:
  - supraglottic (30-35%)
    - rich in lymphatics
    - early nodal spread with 30-40% having occult or palpable neck disease at presentation
    - primary tumour enlarges substantially before causing symptoms (usually presents with dysphagia)
  - glottic (60-65%)
    - few lymphatic channels
    - nodal metastasis rare
    - tumour remains local for a long period
    - produces hoarseness early giving a better prognosis (usually presents with dysphonia)
  - subglottic (1%)
    - abundant lymphatics, lateral neck and paratracheal nodes are involved at presentation in 20%
    - symptoms occur late (usually airway compromise or respiratory problems)
    - may be difficult to distinguish if primary tumour arises in subglottis or in trachea

Presentation

- ❑ dysphagia, odynophagia, or referred otalgia (suggest supraglottic lesion)
- ❑ hoarseness (suggests glottic involvement)
- ❑ clearing throat/foreign body feeling
- ❑ dyspnea/stridor
- ❑ cough/hemoptysis
- ❑ regional lymphadenopathy

Diagnosis

- ❑ direct and indirect laryngoscopy to assess site and extent of tumour and cord mobility
- ❑ bilateral nodal metastasis more common if carcinoma crosses midline
- ❑ CT/MRI imaging: to assess depth of spread and involvement of underlying cartilage

Treatment

- ❑ organ preservation is goal of therapy
- ❑ primary radiotherapy for all laryngeal carcinomas except for bulky T4 lesions with radiographic evidence of cartilaginous involvement
- ❑ surgery reserved for salvage or for late stage lesions
  - microsurgical decortication of vocal cords
  - cordectomy
  - partial to total laryngectomy with trachestomy +/– neck dissection
- ❑ voice and speech rehabilitation options
  - tracheo-esophageal puncture to allow phonation
  - esophageal voice
  - electrolaryngeal devices

Prognosis

- ❑ 10-12% of small lesions will fail radiotherapy and can be treated with partial laryngectomy and muscle flap rehabilitation
- ❑ glottic lesions:> 90% of early lesions (mobile cords) controlled with primary radiation; this drops to 30-60% with cord fixation
- ❑ 70% of T3 supraglottic lesions controlled by radiation alone
- ❑ 5 year survival of > 40% has been reported for T4 lesions following laryngectomy and post-operative radiation

THYROID NEOPLASMS (see Endocrinology Chapter)

Differential Diagnosis

- ❑ benign
  - colloid nodule
  - multinodular goitre (hyperplastic or regenerative nodule)
  - thyroid cyst
  - follicular adenoma
  - thyroiditis
- ❑ malignant (16% of thyroid nodules)
  - papillary carcinoma 60-70%
  - follicular carcinoma 15-20%
  - medullary carcinoma 2-5%
  - anaplastic 1-5%
  - Hürthle cell 1-5%
  - lymphoma 3%
  - metastatic 1-2%

History
- ❑ F > M for nodules but in males a nodule is more likely to be malignant
- ❑ history of head and neck irradiation
- ❑ occupational/environmental radiation exposure associated with papillary carcinoma
- ❑ local compressive neck symptoms - hoarseness, dysphagia, dyspnea, and aspiration
- ❑ family history of multiple endocrine neoplasia type II (MEN II) (medullary cancer)
- ❑ nodule in patient with a history of Hashimoto's - at risk for lymphoma
- ❑ rapid increase in size of nodule - may indicate malignancy

Physical Findings
- ❑ palpation of thyroid - solitary, hard, irregular nodule is suggestive of malignancy, multinodular suggestive
- ❑ benign indirect laryngoscopy - vocal cord paralysis increases suspicion of malignancy
- ❑ cervical lymphadenopathy - deep cervical chain suggestive of metastatic disease
- ❑ signs of hypo/hyperthyroidism

THYROID CARCINOMA

	Papillary Adenocarcinoma	**Follicular Adenocarcinoma**	**Medullary Carcinoma**	**Anaplastic**
Incidence (% of all thyroid cancers)	60-70 %	10%	2-5% (10% familial, 90% sporadic)	5%
Route of Spread	Lymphatic Orpan Annie nuclei	Hematogenous	Lymphatic and hematogenous	
Histologic Findings	Psammoma bodies	Capsular or blood vessel invasion delign malignancy	Amyloid secrete calcitonin	Giant cells, spindle cells
Other	P's of papillary adenocarcinoma - Papillary cancer - Popular (most common) - Palpable lymph nodes - Positive ^{131}I uptake - Positive prognosis - Post-op ^{131}I scan to diagnose treatments	**F**'s of follicular cancer - **F**araway mets - **F**emale (3:1) - **F**NA, NOT (can't be diagnosed by FNA) - **F**avourable prognosis	**M**'s of medullary cancer - **M**EN (associated with **M**EN II) - **M**ultiple endocrine neoplasia	
10 yr survival rate	95%	90%	- 50% - 20% if detected when clinically palpable	- Survival past 2 years is rare
Treatment (see Surgical Procedures section)	- Small tumours: - Near total thyroidectomy or/thyroid lobectomy - Diffuse/bilateral - Total thyroidectomy	- Small tumours: - Near total thyroidectomy/lobectomty /isthemectomy - Large/diffuse tumours total thyroidectomy	- Total thyroidectomy and median lymph node dissection - Modified neck dissection, if lateral cervical nodes are positive	- Small tumours: - Total thyroidectomy ± external beam - Airway compromise: debulking surgery and tracheostomy

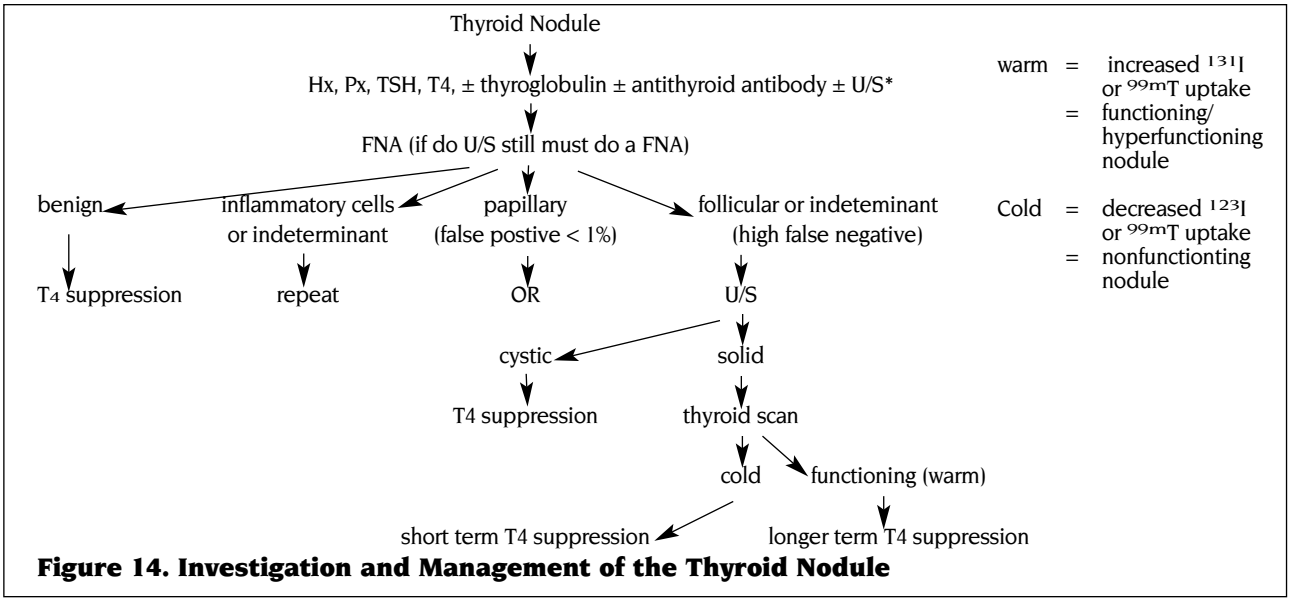


Figure 14. Investigation and Management of the Thyroid Nodule

*U/S findings: cystic: risk of malignancy < 1%, solid: risk of malignancy approx. 10%, solid with cystic components: risk of malignancy same as if solid

SURGICAL PROCEDURES

SURGICAL AIRWAY MANAGEMENT

- ❑ surgical airway: the surgical creation of secondary airway in the neck
- ❑ indications
  - to bypass obstruction
  - airway protection
  - prolonged ventilation > 1-2 weeks, to prevent endotracheal tube-induced glottic/subglottic stenosis
  - bronchial toilet
  - treatment of obstructive sleep apnea
  - elimination of dead space ventilation
- ❑ in adults, most causes of obstruction are inflammatory or neoplastic
- ❑ in children, most causes of obstruction are inflammatory or congenital
- ❑ complications
  - hemorrhage: innominate artery
  - midline scar
  - subglottic stenosis
  - esophageal perforation
  - infection

CRICOTHYROTOMY

- ❑ indications
  - in emergency situations, it is the preferable method to secure an airway since the cricothyroid membrane is easily palpable at the skin surface, little dissection is involved
  - palliative treatment
  - in the presence of anatomic variations that prevent standard tracheotomy
- ❑ contraindications
  - pediatric patients
  - presence of laryngeal infection or inflammation
  - endotracheal tube (ETT) already in place for > 1 week
- ❑ brief procedure description
  - right-handed surgeon stands on patient's right side
  - thyroid cartilage secured with right hand, as left index finger locates the cricothyroid membrane ~2-3 cm below thyroid notch (1.5-2 cm below vocal cords)
  - membrane is ~ 10 mm in height
  - quick stab is placed through overlying skin and directly through cricothyroid membrane with scalpel in right hand
  - knife handle is then inserted into subglottic space and twisted vertically and enlarging access for tube placement

Ref: Weissler MC. Tracheotomy and Intubation. Chapter 57. In Bailey BJ (ed). Head & Neck Surgery - Otolaryngology 2nd ed. Loppincott-Raven Publishers. Philadelphia. 1998.

Causes of Upper Airway Obstruction

Site	Adult	Pediatric
Nasal cavity	Neoplasm, Trauma	Bilateral choanal atresia Enlarged adenoids Congenital stenosis of pyriform aperture Tumour (glioma, dermoid, encephalocele)
Oral cavity	Neoplasm, Trauma Iatrogenic (surgery at base of tongue) Angioedema Infection (Ludwig's angina, retropharyngeal abscess)	Macroglossia Micrognathia (Pierre-Robin sequence) Down's syndrome Hemangioma or lymphangioma of tongue Dermoid cyst or cystic hygroma of mouth Ludwig's angina Angioedema
Pharynx	Neoplasm, Trauma Infections (abscess)	Lingual thyroid Large thyroglossal duct cyst Large branchial cleft cyst Dermoid cyst Enlarged tonsils and adenoids, Angioedema Infections (abscess: peritonsillar, retropharyngeal, parapharyngeal)
Larynx		
Any site	Neoplasm, Trauma Iatrogenic (surgery or intubation) Foreign Body Angioedema	
Supraglottic	Bilateral vocal cord paralysis Angioedema	Laryngomalacia Supraglottic cyst Epiglottitis
Glottic	Bilateral vocal cord paralysis Laryngospasm Stenosis (d/t prolonged intubation)	Bilateral vocal cord paralysis Vocal cord web Stenosis Papillomatosis
Subglottic	Stenosis (d/t prolonged intubation)	Congenital stenosis Hemangioma Acquired stenosis d/t prolonged intubation Croup Subglottic cyst
Trachea		
Intrinsic	Neoplasm, Trauma Foreign body	Foreign body (FB) Tracheobronchial malacia Web Stenosis
Extrinsic	Neoplasm Vascular anomalies	Vascular anomaly Tracheobronchogenic cyst Tumour (thyroid, thymus)

TRACHEOSTOMY

- ❑ tracheotomy is a temporary alternative airway, whereas tracheostomy is a permanent or semi-permanent tracheocutaneous fistula. In practice, the two are often used interchangeably
- ❑ indications
  - airway obstruction
  - long-term ventilatory support
  - prevention of aspiration by allowing suctioning of excessive airway secretions
- ❑ tracheostomy
  - open surgical tracheostomy
    - first incision is horizontal, midway between the sternal notch and cricoid cartilage (~2 fingerbreadths above the sternal notch)
    - this incision is continued down through skin, subcutaneous tissue, and platysma
    - separate the sternohyoid and sternothyroid muscle pairs with a midline vertical dissection
    - these muscles are pulled away from midline with retractors, revealing the thyroid isthmus
    - isthmus is transected vertically and each side is suture-ligated
    - a cricoid hook (placed between the cricoid cartilage and the 1st tracheal ring) is used to pull the trachea superiorly while the tracheal incision is placed at the level of 2nd tracheal ring
    - removal of circular window of cartilage
    - placement of double lumen tracheostomy tube
  - percutaneous dilatational tracheostomy
    - frequently used within many ICU settings
  - translaryngeal tracheostomy
    - the most recent technique
- ❑ complications
  - hemorrhage
  - tube obstruction
  - mortality rate is < 2%

Reference

MacCallum PL *et. al. Comparison of open, percutaneous,and translaryngeal tracheostomies.* Otolaryngol Head Neck Surg 2000;122:686-90.

Weissler MC. *Tracheotomy and Intubation. Chapter* 57. *In Bailey* BJ (*ed*). *Head & Neck Surgery - Otolaryngology 2nd ed. Loppincott-Raven Publishers. Philadelphia.* 1998.

FUNCTIONAL ENDOSCOPIC SINUS SURGERY (FESS)

- ❑ endoscopic techniques aimed at restoring the functional physiology of sinus aeration and drainage via the expanded osteomeatal complex while minimizing changes to the normal anatomic pathways
- ❑ reduced length of hospitalization and discomfort
- ❑ indications
  - refractory sinusitis
  - complicated cases of sinusitis
  - recurrent polyps
  - severe allergies
  - history of previous external procedures
  - expansive mucoceles
  - invasive fungal sinusitis
  - suspected or actual neoplasms
  - symptoms of pressure, discharge, and obstruction most likely will respond
  - pain is less responsive to surgery
- ❑ different FESS techniques are used to gain access to the various sinuses
- ❑ complications
  - cerebrospinal fluid (CSF) leak
  - orbital hematoma
  - hemorrhage
  - symptomatic lacrimal duct obstruction requiring surgery
  - middle turbinate adhesions
  - lamina papyracea penetration
  - sinus infection
  - epistaxis
  - bronchospasm
  - loss of smell

Open Surgical Approach for Extensive Disease

❑ Ethmoid
- intranasal ethmoidectomy via endoscopy - fenestration made into the maxillary sinus which usually restores mucociliary clearance
- complications of unresolved ethmoid sinusitis
- first signs - proptosis, diplopia, chemosis, ophthalmoplegia, poor acuity
- periorbital or orbital edema, cellulitis, abscess
- periostitis
- phlegmon

❑ Frontal
- trephination
- frontoethmoidectomy - removal of mucosa and floor of sinus together with an ethmoidectomy
- complications of frontal sinusitis
- mucocele
- Pott's puffy tumour (osteomyelitis of frontal bone often with fistula formation)

❑ Maxillary
- antrostomy by either enlarging the natural ostium or removing a segment of the sinus floor
- Caldwell-Luc operation - a sublabial approach to removal of diseased lining of the sinus
- complications
  - mucocele
  - oroantral fistula
  - facial cellulitis
  - tooth-loosening
  - osteomyelitis of skull vault bones or upper jaw

NASAL PACKING

Nasal Packing – Anterior (see Figure 15)

❑ there are several types of anterior nasal packing (traditional ribbon gauze pack, prefabricated expandable packs, intranasal balloons).

❑ packing works by direct pressure and by creating inflammation and edema.

❑ the traditional anterior pack
- spray cocaine and lidocaine mixture into nose
  - local anaesthesia decreases discomfort, and blocks nasal-vagal reflex thus decreasing risk of apnea, bradycardia, hypotension.
- if possible, place single layer of absorbable material such as Surgicel, Oxycel, or Gelfoam over bleeding site to prevent rebleeding after pack removal
- prepare vaseline impregnated gauze packing strips 0.5 x 72-inch
- squeeze antibiotic ointment into the gauze to prevent growth of S.*aureus* and toxic shock
- may use prophylactic antibiotics to prevent sinusitis caused by sinus drainage obstruction (amoxil and clavulanic acid)
- use nasal speculum to open nostril
- grasp gauze with bayonet approximately 4 inches from end of strip, and lay packing in nose in a pleated fashion, starting inferiorly along the floor and paking superiorly. (only closed loops of of gauze are placed posteriorly to prevent single strands from hanging into the nasopharynx)
- leave pack in for 2 to 5 days

Reference: *Santos* PM, *Lepore* ML. *Epsitaxis, Chapter* 38. *In*: H*ead and* N*eck Surgery – Otolaryngology, Second Edition, edited by* J. B*ailey*. *Lippincott-Raven Publishers, Philadelphia*, 1998.

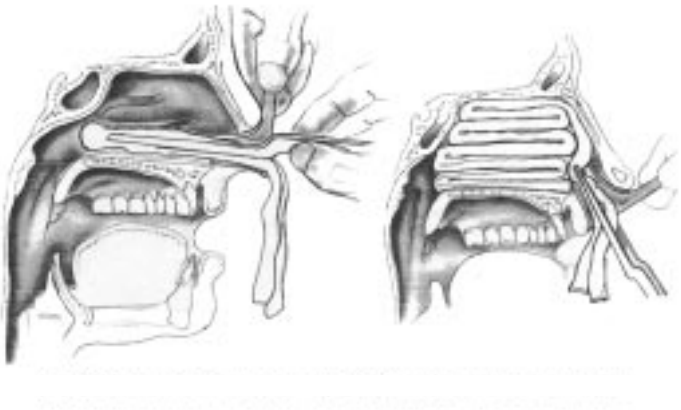


Figure 15. Placement of Traditional Anterior Nasal Pack

Reproduced with permission from Byron J. Bailey, H*ead and N*eck *Surgery-Otolaryngology, Second Edition*. Lippincott-Raven Publishers, Philadelphia, 1998.

TONSILLECTOMY

- ❑ excision of tonsils
- ❑ indications
  - most common reason: obstructive tonsillar hyperplasia
  - absolute
    - obstructive sleep apnea or cor pulmonale
    - malignancy or suspected malignancy
    - tonsillitis resulting in febrile convulsions
    - persistent or recurrent tonsillar hemorrhage
  - relative
    - recurrent acute or chronic tonsillitis
    - peritonsillar abcess
    - eating or swallowing disorders
    - tonisllolithiasis
    - haletosis
    - orofacial or dental abnormalities
  - contraindications
    - blood dyscrasias
    - uncontrolled systemic diseases (diabetes, etc.)
    - cleft palate
    - acute infections
- ❑ technique: electrocautery (hot) vs. "cold steel" (uncommon)
- ❑ complications
  - bleeding
  - airway obstruction
  - velopharyngeal insufficiency (VPI)
  - pulmonary edema
  - dehydration and weight loss

MYRINGOTOMY (EAR) TUBES

- ❑ incision into tympanic membrane that allows drainage of middle ear secretions
- ❑ most tubes stay in place ~3-18 months (average 9 months)
- ❑ indications

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  - severe acute otitis media (AOM)
  - hearing loss > 30 dB in patients with otitis media with effusion
  - poor response to antibiotics for otitis media
  - impending mastoiditis or intracranial complications due to otitis media
  - otitis media with effusion for greater than 3 months
  - recurrent episodes of acute otitis media (more than 3 episodes in 6 months or more than 4 episodes in 12 months)
  - chronic retraction of tympanic membrane or pars flaccida
  - barotrauma
  - autophony (hear body sounds such as breathing) due to patulous (wide open) eustachian tube
  - craniofacial anomalies that predispose to middle ear dysfunction (e.g. cleft palate)
  - middle ear dysfunction due to head and neck radiation and skull base surgery
- ❑ complications (generally uncommon)
  - external auditory canal laceration
  - persistent otorrhea
  - granuloma formation
  - cholesteatoma
  - chronic tympanic membrane perforation
  - structural changes: tympanic membrane retraction, faccidity, myringosclerosis
- ❑ avoid insertion into posterior superior quadrant
  - most compliant part of pars tensa
  - chronic perforation, atrophic scarring, or retraction
  - possible injury to ossicles

THYROIDECTOMY

❑ excision of the thyroid gland
- can be total, subtotal, lobectomy and isthmusectomy
- neck dissection with preservation of sternodedomastoid if lymph nodes clinically involved
- radical neck dissection of extensive infiltrating tumour

❑ indications
- any thyroid abnormality in a patient with previous irradiation of neck
- a solitary nodule in patient less than 20 years old
- a solitary nodule in male patient
- a nodule associated with signs suggestive of malignancy, inducing recurrent nerve paralysis, palpable nodes in the neck, extreme hardness, or extension into adjacent tissues
- a solitary thyroid nodule in a patient over 60 years of age
- most (solitary) nodules
- a normal thyroid gland in a patient with proven metastatic thyroid cancer
- a patient with an abnormal calcitonin stimulation test

❑ complications
- damage to recurrent laryngeal nerves (hoarseness if unilateral, airway obstruction if bilateral)
- damage to superior laryngeal nerve (deeper and quieter voice)
- damage to parathyroid glands = hypoparathyroidism (signs of hypocalcemia)
- excessive bleeding

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