OTOLARYNGOLOGY

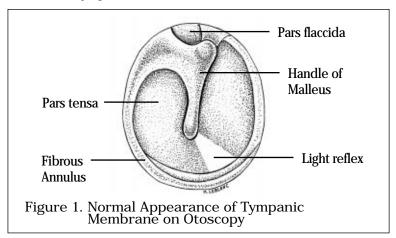
Dr. J. Irish and Dr. B. Papsin Avik Banerjee and Francis Ling, editors T.J. Lou, associate editor

PHYSICAL EXAMINATION 2 Head and Neck Ear	SINUSITIS
Nose Oropharynx Nasopharynx Hypopharynx and Larynx Other Areas of Examination	PEDIATRIC OTOLARYNGOLOGY
ANATOMY OF THE EAR	Adenoid Hypertrophy Acute Tonsillitis Tonsillectomy Airway Problems in Children Signs of Airway Obstruction Acute Laryngotracheobroncheitis - (Croup) Acute Epiglottitis Subglottic Stenosis Laryngomalacia Foreign Body Surgical Airway Management
HEARING LOSS	DYSPHAGIA
Acute Otitis Media and Otitis Media with Effusion Cholesteatoma Mastoiditis Otosclerosis Congenital Sensorineural Hearing Loss Presbycusis	DEEP NECK SPACE INFECTIONS
Sudden Sensorineural Hearing Loss Drug Ototoxicity Noise-Induced Sensorineural Hearing Loss Acoustic Neuroma Temporal Bone Fractures Aural Rehabilitation VERTIGO	HOARSENESS
Differential Diagnosis Otoneurological Examination TINNITUS	SALIVARY GLANDS
OTALGIA	Salivary Gland Manifestations of Systemic Disease NECK MASSES
FACIAL NERVE PARALYSIS	Evaluation Congenital Neck Masses
NASAL OBSTRUCTION	NEOPLASMS OF THE HEAD AND NECK 44 Introduction Etiological Factors in Head and Neck Neoplasia Principles of Management Carcinoma of the Nose and Paranasal Sinuses Carcinoma of the Nasopharynx Carcinoma of the Lip Salivary Gland Neoplasms Carcinoma of the Oral Cavity Carcinoma of the Orapharynx Carcinoma of the Hypopharynx Carcinoma of the Larynx Thyroid Neoplasms Thyroid Carcinoma

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 • observe size, mobility, consistency, tenderness, warmth, regular/irregular border • occipital, posterior auricular, superficial posterior cervical, deep cervical, preauricular, 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
External Examination of Ear inspect external ear structures note position of ear look for deformities, nodules, inflammation, or lesions potential findings discharge: note colour and consistency remnant of first branchial arch: small dimple in front of tragus tophi: sign of gout microtia or macrotia: congenitally small or large auricles "cauliflower ear": gnarled pinna due to repeated trauma 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
Auditory Acuity
Otoscopic Examination select largest speculum that will fit into external canal inspect external canal look for evidence of inflammation, foreign bodies, or discharge inspect tympanic membrane normal membrane: intact, translucent, gray note landmarks handle of the malleus "light reflex": directed anteroinferiorly may see chorda tympani nerve behind short process of malleus possible abnormal findings diseased dull, red or yellow membrane injection of blood vessels tympanosclerosis: dense white plaques fluid or pus in middle ear membrane perforation

- ☐ mobility of tympanic membrane
 - only if there is a question of middle ear infection
 - pneumatic otoscopy to demonstrate decreased movement of tympanic membrañe



Drawing by Monique LeBlanc

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

- - size, colour and mucosa of inferior and middle turbinates
 possible abnormal findings
 - - septal deviation or perforation
 - exudate, swelling, epistaxis
 - nasal polyps

Other Tests

- ☐ transillumination of the sinuses
 - maxillary
 - direct light downward from under the medial aspect of the eye
 - observe transilluminated hard palate
 - frontal
 - direct light upward from under medial aspect of eyebrow

OROPHARYNX

Examination of Oral Cavity

- □ lips
- □ buccal mucosa

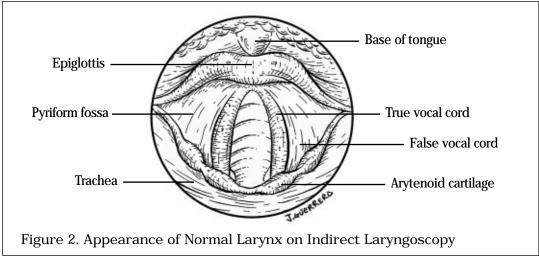
 - pull cheek away from gums and inspect for lesions
 identify Stensen's duct (parotid gland duct)opposite upper first or second molar
- gingivae and dentition
 - 32 teeth in full dentition
 - · look for malocclusion
- □ hard and soft palates

 - ask patient to remove dentures inspect for ulceration or masses

□ floor of mouth • palpate for any masses • identify Wharton's duct (submandibular gland duct) on either side of the frenulum of the tongue □ tongue • inspect for colour, mobility, and masses • palpate tongue for any masses • test cranial nerve XII
Examination of Pharynx ☐ anterior faucial pillars, tonsils, tonsillolingual sulcus • depress middle third of tongue with tongue depressor and scoop tongue forward in order to visualize tonsils • note size and inspect for any exudates from tonsils ☐ posterior pharyngeal wall
NASOPHARYNX
Postnasal Mirror (Indirect) lead ensure good position of the patient • must sit erect with chin drawn forward with adequate tongue depression, the mirror is placed to 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)
Nasopharyngoscope (Direct) ☐ detailed view of nasal cavities and nasopharynx
HYPOPHARYNX AND LARYNX
 Indirect Laryngoscopy □ ensure good position of the patient □ while holding tongue with some gauze, introduce a slightly warmed laryngeal mirror into mouth and position mirror in the 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 sometimes be suppressed if patients are told to
pant in and out image seen in mirror will be reversed • anterior structures are seen at the top while posterior structures are seen at the bottom of the mirror (see Figure 2) inspect the following structures
 circumvallate papillae and base of tongue lingual tonsils valleculae
• epiglottis
 aryepiglottic folds and pyriform fossae should be smooth, pink, and symmetrical false vocal cords
 should be dull pink, symmetrical and thicker than true cords abnormal if they overhang and conceal the true cords true vocal cords
 white and sharp-edged attached anteriorly to the thyroid cartilage (fixed) attached posteriorly to the vocal processes of the arytenoid cartilages (mobile)
 note any irregularity of the edges, nodules or ulcerations

- 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



Drawing by Jason Guerrero

Direct Laryngoscopy with Fibreoptic Nasopharyngoscope

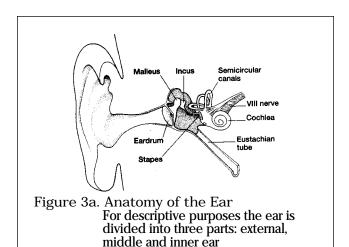
prepare patient with topical anesthetic administered by nasal spray
lexible scope passed via nasal cavity to view structures in the larynx as mentioned above

OTHER AREAS OF EXAMINATION

Cranial Nerves (see Neurology Notes)

Vestibular Function (see Otoneurological Examination Section)

ANATOMY OF THE EAR



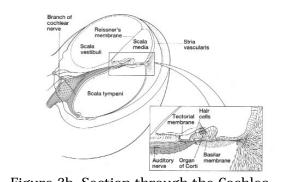
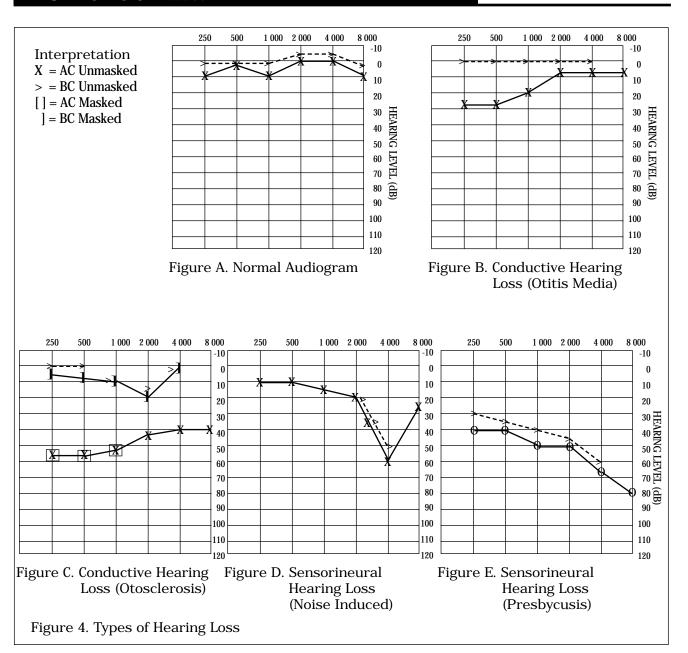


Figure 3b. Section through the Cochlea Hair cells in the organ of Corti transform mechanical energy (vibration) into electrical impulses

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.

AUDIOLOGY Notes

PURE TONE AUDIOMETRY
☐ threshold is the faintest intensity level at which a patient can hear
the tone 50% of the time
☐ 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
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
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
16-25 dB slight
26-40 dB mild 41-55 dB moderate
56-70 dB moderate-severe
71-90 dR severe
91 + dB profound
☐ types of hearing loss (see Figure 4)
91 + dB profound 1 types of hearing loss (see Figure 4) 1 conductive (something is impairing the conduction of sound to the cochlea - i.e. fused or broken ossicular chain)
cochlea – i.e. fused or broken ossicular chain)
• bone thresholds in normal range
 bone thresholds in normal range air conduction thresholds increased by 15-20 dB or more above bone conduction thresholds
above done conduction thresholds
 sensorineural (the sensory component of the inner ear, brainstem or cortex is damaged)
air and bone conduction thresholds below normal and similar (if
the loss is unilateral it should be investigated further to rule out
acoustic neuroma, noise-induced hearing loss, etc)
□ mixed
 air and bone conduction thresholds below normal, as well as
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 500-2000 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

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

AUDIOLOGY ... CONT.

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)

IMPEDANCE AUDIOMETRY

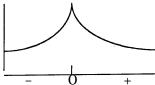
Tympanogram ustachian tub

eustachian tube equalizes the pressure between outer 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 H₂O peak of tympanogram occurs at the pressure of the pressure o

where the pressure in the external canal is equivalent to the pressure in the middle ear

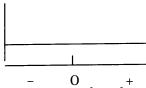
 \square normal range: -100 to +50 mm H20

Type A Tympanogram



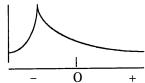
normal middle ear pressure peak at 0 mm H₂O, note that with otosclerosis the peak is still at 0mm H₂O but has a lower amplitude (called an As tympanogram)

Type B Tympanogram



no pressure peak, and 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 exposed to loud sound and results in increased stiffness or impedance of middle ear system (TM and ossicles)

- (I'M and ossicles)
 stimulating either ear causes reflex to occur bilaterally and symmetrically reflex measured ipsilaterally by stimulating and measuring from same ear, or contralaterally by stimulating one ear and measuring impedance in the contralateral ear
 □ 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)
 □ 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

 □ for reflex to be present, CN VII must be intact and there must be no conductive hearing loss in the monitored ear if reflex 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 MS)
TUNING FORK TESTS
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
Weber's Test ☐ vibrating fork is held on vertex of head and patient states whether it is heard centrally or is lateralized to one side ☐ lateralization indicates ipsilateral conductive hearing loss or contralateral sensorineural hearing loss
Table 1. The Interpretation of Tuning Fork Tests

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 cochlea, 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) $\,$

Notes

DIFFERENTIAL DIAGNOSIS Conductive external ear canal cerumen otitis externa foreign body congenital ătresia keratosis obturans tumour of canal: squamous cell carcinoma (rare) ☐ middle ear acute otitis media · serous otitis media • tympanic membrane perforation otosclerosis • congenital: ossicular fixation trauma, i.e. hemotympanum • tumour, i.e. cholesteatoma Sensorineural congenital □ acquired presbycusis (very common in elderly) Menière's disease noise-induced (dip at 4000 Hz on audiogram) ototoxic drug (high frequency loss) head injury sudden sensorineural hearing loss labyrinthitis (viral or bacterial) meningitis demyelinating disease (e.g. MS) trauma (e.g. temporal bone fracture) tumour (e.g. acoustic neuroma) OTITIS EXTERNA 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 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

☐ clean ear under magnification with irrigation, suction, and dry swabbing

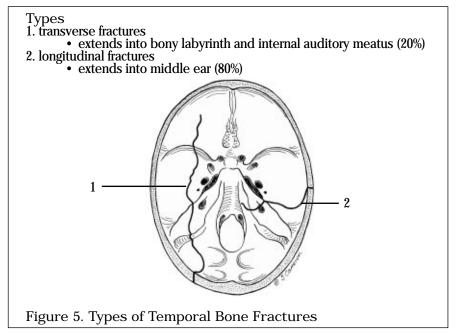
 bacterial etiology topical aminoglycoside antibiotics +/- corticosteroids (e.g. Garamycin, Neosporin, Corticosporin) introduction of fine gauze wick (pope wick) if external canal edematous +/- 3% acetic acid solution to acidify ear canal systemic antibiotics when cervical lymphadenopathy or cellulitis present - obtain cultures from external canal 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 □ due to <i>Pseudomonas</i> osteomyelitis of temporal bone □ associated with diabetics, elderly, perichondritis, cellulitis, parotitis, +/- chronic symptoms □ requires hospital admission, debridement, IV antibiotics and emergent CT scan
ACUTE OTITIS MEDIA AND OTITIS MEDIA WITH EFFUSION (see Pediatric ENT Section)
CHOLESTEATOMA (see Colour Atlas 17) □ keratinized squamous epithelium in middle-ear or mastoid
Congenital behind an intact tympanic membrane "small white pearl", not associated with otitis media usually presents with conductive hearing loss
Acquired ☐ frequently associated with 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 • failure of medical treatment after 48 hours
 symptoms of intracranial complications aural discharge persisting for 4 weeks and resistant to antibiotics

OTOSCLEROSIS □ 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 2000 Hz (Carhart's Notch) on audiogram (Figure 4)
Treatment ☐ stapedectomy with prosthesis is definitive treatment ☐ hearing aid may be used, however usually not a good long term solution
CONGENITAL SENSORINEURAL HEARING LOSS
$egin{array}{cccccccccccccccccccccccccccccccccccc$
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 Toxoplasmosis Others e.g. HIV Rubella Cytomegalovirus (CMV) Herpes simplex
Perinatal Rh incompatibility anoxia kernicterus 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) • kemicterus - 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 ☐ 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 and 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 □ refer to audiogram in Figure 4	
Treatment ☐ hearing aid if hearing loss > 30-35 dB ☐ +/- lip reading and auditory training	
SUDDEN SENSORINEURAL HEARING LOSS (UNILATERAI presents as a sudden onset of significant hearing loss (usually unilateral) +/- tinnitus unexplained etiology	(بـ
 20% experience partial resolution 10% experience permanent hearing loss DRUG OTOTOXICITY	
Aminoglycosides increased toxicity with oral administration 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), gentamicin (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	

Cisplatinum
Quinine and Antimalarials i tinnitus □ reversible if discontinued but can lead to permanent loss □ treat drug ototoxicity with IV low molecular weight dextrans
NOISE-INDUCED SENSORINEURAL HEARING LOSS □ may be occupational, often associated with tinnitus □ 85-90 dB over months or years causes cochlear damage □ early-stage hearing loss at 4000 Hz, extending 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 □ refer to audiogram in Figure 4
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
Limits of Noise Causing Damage □ continuous sound pressure of 85-90 dB and higher □ single sound impulses > 135 dB
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 ☐ Schwannoma of the vestibular portion of CN VIII ☐ most common intracranial tumour causing hearing loss ☐ starts in the internal auditory canal and expands into CPA, compressing cerebellum and brainstem ☐ may be associated with Type 2 neurofibromatosis (bilateral tumours of CN VIII in internal auditory canal, cafe-au-lait lesions, multiple intracranial lesions)
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
Clinical Pearl Any unilateral sensorineural hearing loss is an acoustic neuroma until proven otherwise
Diagnosis ☐ enhanced CT/MRI ☐ audiogram - puretone threshold elevated ☐ poor speech discrimination and stapedial reflex ☐ absent or significant reflex decay ☐ Acoustic Brainstem Reflexes - increase in latency of the 5th wave ☐ electronystagmography (ENG)

TEMPORAL BONE FRACTURES



Drawing by Teddy Cameron

Table 2. Features of Temporal Bone Fractures		
	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

- Diagnosis

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

☐ hemotympanum signifies significant force sustained by the temporal bone, 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 early meningitis (mastoidectomy) bleeding from sinus CSF otorrhea CN VII palsy gunshot wound depressed fracture of external auditory meatus
Complications acute otitis media +/- mastoiditis meningitis/labyrinthitis 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
 patients can present with a wide range of subjective descriptions of their symptoms: dizziness, spinning, lightheadedness, giddiness, unsteadiness true vertigo 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)
DIFFERENTIAL DIAGNOSIS □ peripheral vestibular (labyrinthine) disorders • Meniere's disease • benign positional vertigo (BPV) • vestibular neuronitis • recurrent vestibulopathy - post traumatic or post viral • tumour - acoustic neuroma • trauma - skull fractures, barotrauma, surgery • suppurative labyrinthitis

- $\ \square$ central vestibular disorders brainstem
 - tumour
 - multiple sclerosis

- systemic
 hypoglycemia
 hyperventilation
 anemia

 - ototoxic drugs vascular lesions vertebrobasilar insufficiency

Table 3. Differential Diag	gnosis of Vertig	О			
Condition	Duration	Hearing Loss	Tinnitus	Aural Fullness	Other Features
BPV	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	chronic	progressive	none	none	ataxia CN VII palsy

Clinical Pearl \Box 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 Positional Vertigo (BPV)
☐ most common cause for episodic vertigo
acute attacks of transient vertigo initiated by certain head positions lasting second
to minutes, accompanied by nystagmus that fatigues on repeated testing
ue 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 receive 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 exercise to continually stimulate
vestibular system to allow it to compensate
 drugs to suppress the vestibular system delay eventual recovery
and are therefore not used
Maniara's Disassa (andalymphatic hydrons)
Meniere's Disease (endolymphatic hydrops)
 affects the young to middle-aged characterized by the quadrad of vertigo, hearing loss,
tinnitus, and aural fullness
early in the disease, hearing returns to normal in the attack-free states
☐ later stages are characterized by a unilateral, fluctuating
low-tone deafness and a persistance of tinnitus
□ attacks come in clusters and may be very debilitating to the patient,
may be triggered by stress
may be triggered by stress most hearing loss becomes bilateral with time
vertigo disappears with time and patient is left only with hearing loss
□ pathogenesis: abnormal buildup of K+
 increase in osmotic pressure causes a rupture of Reissner's
membrane which divides the endo- and perilymphatic spaces
membrane which divides the endo- and perilymphatic spaces disruption allows for mixing of endo and perilymph resulting in a
net rise of K ⁺ in the intracellular space
 as a result afferent neurons of the acoustic and vestibular nerves

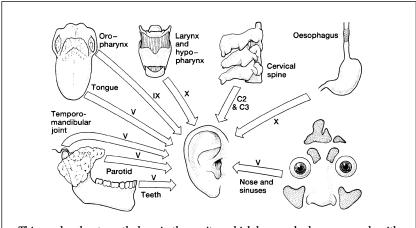
are depolarized (causing symptoms)

 periodic resolution occurs when the membrane reseals and chemical composition of endolymph and perilymph normalizes
 treatment acute management may consist of bedrest, IV antiemetics, antivertiginous drugs, 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 episodic vertigo of similar duration as Meniere's etiology unknown transient deafferentation of vestibular nerve post-traumatic post-viral no hearing loss, tinnitus, or focal neurological deficit
peak age 30-50 years old, M = F treatment: symptomatic, most eventually go into remission
Vestibular Neuronitis □ unknown etiology (microvascular upset due to infection, autoimmune process, or a metabolic disorder) □ 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
Labyrinthitis □ sudden onset of dizziness, nausea, vomiting, whistling noises, and deafness, with no associated fever or pain □ may occur through spread from a cholesteatomic fistula or through direct 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
OTONEUROLOGICAL EXAMINATION □ 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)
Dix-Hallpike Positional Testing with Frenzel's (Magnifying) Eyeglasses □ 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, this position is held for 20 seconds □ onset of vertigo is noted and the eyes are observed for nystagmus □ the patient sits up and the maneouvre is then repeated with the head turned to the opposite side □ rotatory nystagmus beating towards floor (geotropic), which is accompanied by vertigo, is reproducible and fatigues, is associated with benign positional vertigo (BPV)

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 30 degrees to bring the horizontal semicircular canal into a vertical position. The volume of endolymph is changed by irrigating the labyrinthine capsule with water at 30°C or 44°C for 35 seconds □ the change in volume causes deflection of the cupula and subsequent nystagmus through the vestibuloocular reflex □ 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 (COWS - cold opposite, warm same)
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 • MS □ 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 palate tetany

OTALGIA

Local furuncle foreign body in external auditory canal/impacted cerumen otitis externa trauma to tympanic membrane and canal acute otitis media and its complications acute mastoiditis and its complications barotrauma
Referred (10 T's + 2) - see Figure 6 □ pain referred to ear from sites in pharynx or oral cavity □ CN V and CN X refer to external canal and CN IX to middle ea 1) teeth - impacted 2) throat - cancer of larynx, vallecula, pyriform fossa 3) thyroiditis 4) tic (CN IX) - glossopharyngeal neuralgia
5) TMJ syndrome 6) tongue 7) tonsillitis, tonsillar cancer, post tonsillectomy 8) trachea - FB, tracheitis 9) trismus (i.e. pterygoids, quinsy) 10) eustachian tube 11) geniculate herpes and Ramsey Hunt Syndrome 12) +/- CN VII palsy



This can be due to pathology in those sites which have a dual nerve supply with the ear. All these potential penpheral areas must be examined in all cases of referred otalgia ${\sf case}$

Figure 6. Referred or Non-Otological Otalgia

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Etiology supranuclear and nuclear
Diagnosis ☐ supranuclear lesions: movement of upper part of face is likely to be unaffected as the frontalis muscle receives bilateral corticobulbar innervation ☐ nerve conduction tests and EMG ☐ site of lesion testing (e.g. stapedial reflexes, Schirmer's, taste to anterior 2/3 of tongue)

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

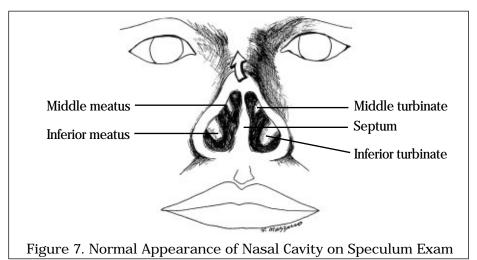
- BELL'S PALSY (see Colour Atlas I1) a diagnosis of exclusion, therefore must rule out other causes of facial paralysis (e.g. ear infection)
 idiopathic, may be a disturbance of microcirculation +/- viral etiology
 80% recover
 sequelae: "crocodile tears", facial asymmetry and ectropion, corneal

- abrasions
- ☐ treat with steroids (e.g. oral prednisone), stellate ganglion block or low molecular weight dextrans with decompression of nerve reserved for progressive denervation

NASAL OBSTRUCTION

Table 4. Differential Diagnosis of	Nasal Obstruction
Acquired	Congenital
nasal cavity rhinitis - acute/chronic - vasomotor - allergic polyps foreign bodies trauma enlarged turbinates tumour - benign- inverting papilloma - malignant- squamous cell carcinoma esthesioneuroblastoma adenocarcinoma	nasal dermoid encephalocele glioma
nasal septum septal deviation septal hematoma/abscess	dislocated septum
nasopharynx adenoid hypertrophy tumour - nasopharyngeal carcinoma - benign - juvenile nasopharyngeal angiofibroma - malignant: nasopharyngeal carcinoma	choanal atresia

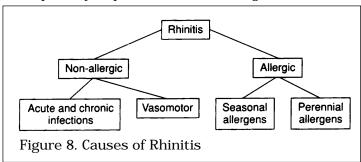
Table 5. Nasal D Condition	ischarge: Character and Associated ns
Character	Associated Conditions
watery/mucoid	allergic, viral, vasomotor, CSF leak
mucopurulent	bacterial, foreign body
serosanguineous	neoplasia
bloody	trauma, neoplasia, bleeding disorder, hypertension/vascular disease



Drawing by Vince Mazzurco

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



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Etiology $\hfill \Box$ 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

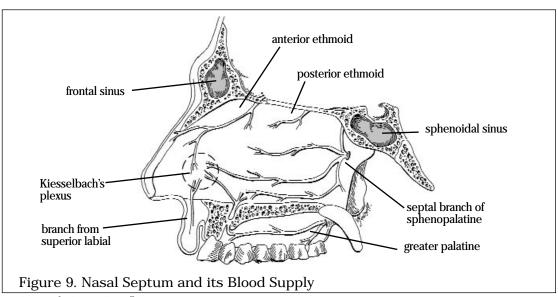
- ☐ symptoms subside in 4-5 days

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 obstruction sneezing clear, recurrent rhinorrhea (containing increased eosinophils) itching of nose and eyes 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 □ serous otitis media □ nasal polyps
Diagnosis □ history □ skin/allergy testing: scratch test, intradermal injections □ nasal speculum exam
Treatment ☐ identification and avoidance of allergen ☐ oral decongestants ☐ antihistamines ☐ injection of long-lasting steroid if severe ☐ topical steroid sprays, e.g. fluticasone (Flonase) - effective for seasonal rhinitis ☐ 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
 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) chronic rhinitis/sinusitis (ethmoids) idiopathic note: triad of polyps, aspirin sensitivity, asthma 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)
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 □ recurrent ear infections, recurrent sinus infections □ 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 may lead to infection —> abscess —> cavernous sinus thrombosis ☐ septal perforation ☐ ischemic necrosis of septum and saddle deformity
Presentation In an
Treatment ☐ incision and drainage with nasal packing ☐ antibiotics
SEPTAL PERFORATION
Etiology trauma: surgery, physical, digital infection: syphilis, tuberculosis inflammatory: 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

EPISTAXIS



Drawing by Victoria Rowsell

blood supply to the nasal septum
 superior posterior septum: internal carotid --> ophthalmic artery
 --> ant and post ethmoidal

posterior septum: external carotid --> internal maxillary --> sphenopalatine artery

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. Et	ble 6. Etiology of Epistaxis	
Туре	Causes	
local	idiopathic (most) injection (vestibulitis) trauma (digital, dry air) foreign body tumours benign - junvenile 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
- ☐ 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!)
 assess blood loss (it can be a potentially fatal hemorrhage)
- pulse and BP
 sign of shock
 IV NS, cross match for 2 units packed RBCs if significant
 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
- a differior/posterior hemorinage defined by location in relationship to bony septum
 □ control the bleeding
 first line topical vasoconstrictors
 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
- ☐ anterior hemorrhage treatment
- □ 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
 can also attempt packing with Merocel or nasal tampons of different shapes
 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
- - complications such as hypoxemia (naso-pulmonic reflex) and toxic shock syndrome (if present remove packs immediately)

- 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

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

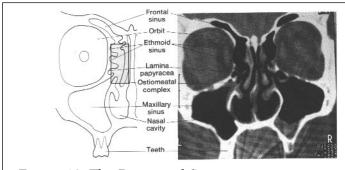


Figure 10. The Paranasal Sinuses

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ACUTE SUPPURATIVE SINUSITIS

- associated with
 - common cold

 - swimming/diving diseased tooth roots
- organisms

 - S. pneumonia
 H. influenza
 S. aureus diabetic
 Klebsiella, Pseudomonas, 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
 esphenoid vertex

 mucosa hyperemic and edematous with enlarged to
- ☐ mucosa hyperemic and edematous with enlarged turbinates
- x-ray involved sinus opaque +/- fluid level (see Colour Atlas I9)

□ 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	
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) □ 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 acu ■ periorbital or orbital edema, cellulitis, abscess ■ periostitis ■ phlegmon □ Frontal	ity
 trephination frontoethmoidectomy - removal of mucosa and floor of sinus together with an ethmoidectomy complications of frontal sinusitis (see Figure 11) mucocele Pott's puffy tumour (osteomyelitis of frontal bone often with fistula for 	rmation)

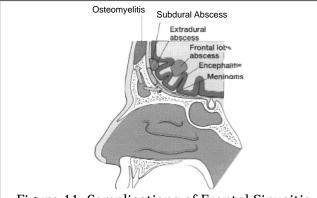


Figure 11. Complications of Frontal Sinusitis

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- □ 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

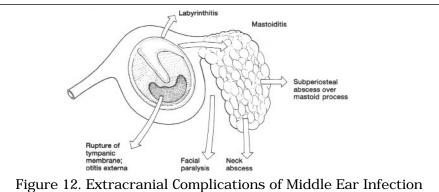
 mucocele
 oroantral fistula
 facial cellulitis

 - - tooth-loosening
 osteomyelitis of skull vault bones or upper jaw

PEDIATRIC OTOLARYNGOLOGY

ACUTE OTITIS MEDIA ☐ 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 \(\beta\)-lactamase producing) anaerobes (newborns) viral	
Predisposing Factors eustachian tube dysfunction/obstruction * swelling of tubal mucosa * URTI * allergies/allergic rhinitis * chronic sinusitis * obstruction/infiltration of eustachian tube ostium * tumour - nasopharyngeal CA (adults) * adenoid hypertrophy * barotrauma (sudden changes in air pressure) * inadequate tensor palati function - cleft palate * abnormal spatial orientation of eustachian tube * Down's Syndrome (horizontal position of eustachian tube),	,
Risk Factors bottle feeding passive smoke day care/group child care facilities	
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	

Procentation
Presentation triad of otalgia, fever, and conductive hearing loss fullness of ear
 otorrhea if tympanic membrane perforated (see Colour Atlas I8) pain over mastoid infants/toddlers
ear-tuggingirritable, poor sleepingvomiting and diarrhea
• anorexia
otoscopy of tympanic membrane (see Colour Atlas I5)hyperemia
bulgingcontour of handle of malleus and short process disappear
Treatment ☐ antibiotic treatment hastens resolution - 10 day course
• amoxicillin - 1st line
 trimethoprim-sulphamethoxazole (Bactrim) - if penicillin-allergic AOM deemed "unresponsive" if clinical signs and symptoms and otoscopic findings persist beyond 48 hours of antibiotic treatmen change to broad spectrum: cefaclor (Ceclor), erythromycin sulfisoxizole (Pediazole), cefixime (Suprax)
• clarithromycin (Biaxin) for recurrent AOM
 antipyretics (e.g. acetaminophen) no role for decongestants in AOM
☐ myringotomy with tubes - indications
 complications of AOM suspected
 recurrent AOM (> 5 in a year) immunologically compromised child
• failure of 3 different antibiotics - fever and bulging of drum
Complications of AOM
□ extracranial (see Figure 12)
 acute mastoiditis, chronic suppurative otitis media
• facial nerve paralysis
febrile seizuresnystagmus - bacterial labyrinthitis
TM perforation
intracranial
meningitisextradural, subdural, cerebral abscess
• petrositis
 lateral sinus thrombosis
OTITIS MEDIA WITH EFFUSION
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
can be
mucoid - follows AOM
resolving mucoid
serousbarotrauma in adults
- Daiouauna in addis



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Presentation fullness - blocked ear hearing loss +/- tinnitus confirm with audiogram (see Figure 4) and tympanogram (flat) minimal pain, possibly low grade fever, no discharge otoscopy of tympanic membrane (see Colour Atlas I6) 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 antibiotics thought to decrease viscosity of effusion by killing residual organisms no statistical proof that antihistamines, decongestants, antibiotics clear faster than no treatment surgery: myringotomy +/- ventilating tubes +/- adenoidectomy (if enlarged) (see Colour Atlas 110) ventilating tubes indications persisting effusion > 3 months hearing loss > 30 dB speech delay atleectasis 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 chronic inflammation hearing loss, speech delay, learning problems in young children can lead to chronic suppurative otitis media 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 6 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 ☐ enlarged adenoid shadow on lateral soft tissue x-ray ☐ lateral view of the nasopharynx may show a large pad of adenoidal tissue

PEDIATRIC OTOLARYNGOLOGY \dots cont.

Complications used 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 Strep and Group G Strep Pneumococci S. aureus H. influenza EBV
Presentation □ symptoms • sore throat • dysphagia, odynophagia • malaise, fever • otalgia (referred) □ signs • cervical lymphadenopathy especially submandibular, jugulodigastric • tonsil enlarged, inflamed +/- spots (see Colour Atlas 12) • 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 (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 (see Pediatrics Notes) uncommon since the use of antibiotics • rheumatic heart disease • nephritis • arthritis • scarlet fever deep neck space infection

PEDIATRIC OTOLARYNGOLOGY ... CONT.

□ abscess: peritonsillar (quinsy), intratonsillar□ sepsis
TONSILLECTOMY
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, Down's, lymphangioma, hemangioma □ laryngeal • laryngomalacia - most common • laryngocele • vocal cord palsy (Arnold-Chiari) • glottic web • subglottic stenosis • laryngeal cleft □ tracheal • tracheoesophageal fistula • tracheomalacia
2-3 Months □ congenital • laryngomalacia • vascular: innominate artery compression, double aortic arch, subglottic hemangioma • laryngeal papilloma □ acquired • subglottic stenosis - post intubation • tracheal granulation - post intubation • tracheomalacia - post tracheotomy and TEF repair
Infants - Sudden Onset ☐ foreign body aspiration ☐ croup ☐ caustic ingestion ☐ epiglottitis
Children and Adults congenital lingual thyroid/tonsil infection Ludwig's angina peritonsillar-parapharyngeal abscess retropharyngeal abscess neoplastic squamous cell carcinoma (adults): larynx, hypopharynx retropharyngeal: lymphoma, neuroblastoma nasopharyngeal: rhabdomyosarcoma

	allergic
	trauma • laryngeal fracture, facial fracture • burns and lacerations • post-intubation • caustic ingestion
	IGNS OF AIRWAY OBSTRUCTION symptoms and signs of airway obstruction require a full assessment to diagnose potentially serious causes
	ridor note quality, timing body position important • lying prone: subglottic hemangioma, double aortic arch • lying supine: laryngomalacia, inominate artery compression, glossoptosis site of stenosis • larynx or above: inspiratory stridor • trachea: biphasic stridor • bronchi/bronchioles: expiratory stridor
	espiratory Distress nasal flaring supraclavicular and intercostal indrawing sternal retractions tachypnea cyanosis
	eeding Difficulty and Aspiration supraglottic lesion laryngomalacia vocal cord paralysis post laryngeal cleft —> aspiration pneumonia tracheoesophageal fistula
	CUTE 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
Et	ciology viral: parainfluenzae I (most common), II, III, influenza A and B, RSV
00000000	resentation 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 if recurrent croup, think subglottic stenosis
0000000	reatment 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)
A	CUTE EPIGLOTTITIS acute inflammation causing swelling of supraglottic structures of the larynx without involvement of vocal cords

PEDIATRIC OTOLARYNGOLOGY ... CONT.

 2-5% of laryngeal inflammatory disease occurs in northern USA, Europe, and Canada highest in December
Etiology H. <i>influenza</i> 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 rule out severe tonsillitis, peritonsillar abscess, retropharyngeal abscess
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 ☐ bag-mask ventilation with Ambu bag prior to endoscopy ☐ intubate prior to any other treatment ☐ endoscopy in operating room suite ☐ 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 ibiphasic stridor ir respiratory distress ir recurrent/prolonged croup
Diagnosis □ laryngoscopy □ CT
Treatment if soft tissue - laser and steroids if cartilage - wait, do tracheostomy, and laryngotracheoplasty when older
LARYNGOMALACIA □ most common laryngeal anomaly □ elongated omega-shaped epiglottis, short aryepiglottic fold, pendulous mucosa
Presentation ☐ high-pitched crowing inspiratory stridor at 6 weeks being constant or intermittent and more pronounced supine

PEDIATRIC OTOLARYNGOLOGY ... CONT.

 associated with feeding difficulties symptoms gradually subside at 18-24 months as larynx grows and thus requires no treatment
FOREIGN BODY
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 ☐ bronchoscopy and esophagoscopy with removal
SURGICAL AIRWAY MANAGEMENT surgical creation of secondary airway laryngotomy also known as cricothyroidotomy 14 gauge needle or IV cannula inserted through cricothyroid membrane tracheostomy incision made at the level of 2nd tracheal ring division of strap muscles division +/- ligation of thyroid isthmus removal of circular window of cartilage placement of double lumen tracheostomy tube indications to bypass obstruction bronchial toilet long-term ventilation > 1-2 weeks, to prevent endotracheal tube-induced glottic/subglottic stenosis complications hemorrhage: innominate artery midline scar subglottic stenosis
DYSPHAGIA
(see Gastroenterology Notes) DIFFERENTIAL DIAGNOSIS Oral Cavity □ viral ulcers (gingivitis): Coxsackie, aphthous, Herpes □ trauma (including caustic ingestion) □ tumour □ Ludwig's angina
Oropharynx tonsillar carcinoma/tonsillitis/tonsillar hypertrophy/pharyngitis retropharyngeal abscess, pharyngeal/palatal paralysis/VPI
Hypopharynx/Larynx tumour: intrinsic or extrinsic (thyroid mass and other neck masses) trauma (including caustic ingestion) foreign body neuromuscular disturbance • pharyngeal/laryngeal paralysis • cricopharyngeal spasm Plummer-Vinson syndrome inflammatory

Esophagus obstructive (sudden onset with progression) intrinsic hiatus hemia tumour corrosive esophagitis and stricture esophageal web foreign body esophageal diverticulum (Zenker's) extrinsic mediastinal abnormalities vascular compression motility (gradual onset) achalasia diffuse esophageal spasm scleroderma diabetic neuropathy trauma/perforation
History ☐ chronology of the symptoms: acute vs. chronic, static vs. progressive ☐ liquids, solids or both ☐ associated symptoms that may give insight to the etiology of the dysphagia such as cough, odynophagia, hoarseness, reflux
Physical assess labial competence for control of oral secretions, foods and fluids assess the tongue: sensory and motor functions agag reflex examine the oro- and hypopharynx, pooling of secretions in the vallecula, pyriform sinuses or oral cavity often indicate swallowing dysfunction, with the amount of pooled secretions signifying the severity of the problem examine the larynx and neck
Investigations ☐ soft tissue x-rays of the neck looking for swelling, displacement of airway, presence of foreign body ☐ chest x-ray: displacement of airway, esophageal air-fluid levels ☐ dynamic imaging studies: barium pharyngoesophagogram ☐ assessment of reflux: pH probe manometer, endoscopy ☐ CT/MRI or U/S
Treatment (see Gastroenterology Notes)
DEEP NECK SPACE INFECTIONS
 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 line fever, pain, swelling line +/- trismus, fluctuance, dysphagia, and dental abnormalities line r/o 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

DEEP NECK SPACE INFECTIONS ... CONT.

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 □ can develop from acute tonsillitis □ unilateral, most common in 10-30 year old age group
Etiology Dacterial: Group A Strep, <i>S. pyogenes, S. aureus, H. influenzae</i> and anaerobes
Presentation dysphagia and sore throat into potato" voice increased salivation and trismus referred otalgia cervical adenopathy and fever extensive peritonsillar swelling but tonsil may appear normal uvula deviated across midline edema of soft palate
Treatment □ surgical drainage (incision or needle aspiration) - do C&S □ tonsillectomy at presentation or 6 weeks later □ IV antibiotics (clindamycin) □ warm saline irrigation
RETROPHARYNGEAL ABSCESS in adults secondary to spread from parapharyngeal space due to an abscess or trauma of posterior pharyngeal wall 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. ETT or suction in neonate, popsicle stick abrasion in child)
Presentation □ child • infant/child with unexplained fever post URTI with loss of appetite, speech change, or difficulty swallowing • 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
Treatment ☐ IV antibiotics ☐ surgery: incision and drainage with airway secured
LUDWIG'S ANGINA □ cellulitis/inflammation of superior compartment of suprahyoid space between geniohyoid and mylohyoid muscles □ causes firmness of floor of mouth, tongue protrudes upward and back causing airway obstruction

DEEP NECK SPACE INFECTIONS CONT.
Etiology dental infection (anaerobic bacilli + Vincents spirochete) suppurative lymph node in submaxillary space
Treatment ☐ incision through midline and prepare for possible tracheostomy because of the difficulty in intubation ☐ IV penicillin + metronidazole
ACUTE TONSILLITIS and TONSILLECTOMY (see Pediatric ENT Section)
HOARSENESS
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
DIFFERENTIAL DIAGNOSIS
Infectious □ acute viral laryngitis □ bacterial tracheitis/laryngitis □ laryngotracheobronchitis (croup)
Inflammatory ☐ from gastro-esophageal reflux, smoke irritation, or chronic cough • vocal cord polyps • Reinke's edema • contact ulcers or granulomas • vocal cord nodules
Trauma ☐ external laryngeal trauma ☐ endoscopy and endotracheal tube

- Neoplasia

 □ benign tumours

 vocal cord polyps

 papillomas

 chondromas, lipomas, hemangiomas

 □ malignant tumours

 squamous cell carcinoma

 Kaposi's sarcoma

- Cysts
 ☐ retention cysts
 ☐ laryngoceles

- - rheumatoid arthritis,
 SLE
- □ angioneurotic edema

Neurologic (vocal cord paralysis) □ central lesions • CVA • head injury • MS • Areald Chievi

- - Arnold-Chiari
 - neural tumours

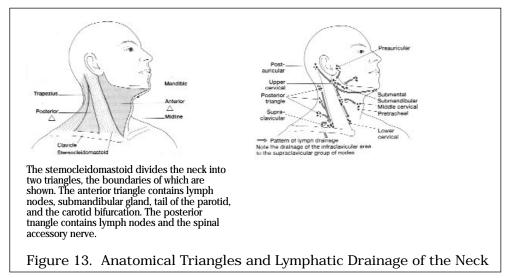
 peripheral lesions tumours: glomus jugulare, thyroid, bronchogenic, esophageal, neural surgery: thyroid surgery, cardiovascular or thoracic/esophageal surgery cardiac: left atrial enlargement, aneurysm of aortic arch neuromuscular myasthenia gravis presbylaryngeus spastic dysphonia
Functional psychogenic aphonia (hysterical aphonia) habitual aphonia ventricular dysphonias
ACUTE LARYNGITIS
Etiology □ viral +/- URTI - influenza, adenovirus, <i>GAS</i> □ 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 □ agents causing laryngeal inflammation
Presentation hoarseness, aphonia, cough attacks +/- dyspnea laryngoscopy shows polyp on free edge of vocal cord on a pedicle or sessile
Treatment

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 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 CO2 laser and microsurgery
LARYNGEAL CARCINOMA (see Neoplasms of the Head and Neck Section)
SALIVARY GLANDS
SIALOADENITIS
Etiology ☐ obstructive vs. non-obstructive ☐ bacterial: (commonly <i>S. aureus</i>) 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
ϵ

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Diagnosis
☐ imaging with U/S employed to differentiate obstructive vs. non-obstructive sialadenitis

Treatment bacterial: treat with cloxacillin +/- abscess drainage viral: no treatment
SIALOLITHIASIS ☐ ductal stone with chronic sialadenitis ☐ predisposing factors are any conditions 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 excise
SALIVARY GLAND MANIFESTATIONS OF SYSTEMIC DISEASE Sjögrens syndrome: diffuse non-tender, asymptomatic enlargement of the parotid glands and occasionally other salivary glands bulimia nervosa: bilateral swelling of parotid glands, approximately 30% of patients will have resolution with control of bulimia



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DIFFERENTIAL DIAGNOSIS

duration

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

Congenital ☐ midline

- thyroglossal duct cyst
 thyroid tumour/goitre
 pyramidal lobe of thyroid gland
 midline dermoid cyst
- thymus cyst

- □ lateral
 branchial cleft cyst
 cystic hygroma

Acquired

Table 7. Ac	equired Causes of Neck Lumps
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	1º or 2º malignant disease

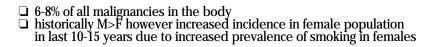
EVALUATION

Investigation
□ history and physical □ indirect tests - supply information about physical characteristics of mass • WBC infection vs. hymphoma
 WBC - infection vs. lymphoma Mantoux TB test
thyroid function tests and scan
• neck U/S
CT scan angiography wascularity and blood supply to mass.
 angiography - vascularity and blood supply to mass direct test - for histologic examination
• fine needle aspiration - less invasive
• needle biopsy
open biopsy-for lymphoma search for the primary tumour fill atalogue golden areas including pages horney and lawrey.
 full otolaryngologic exam - including nasopharynx and larynx radiologic exam of stomach, bowel and sinuses
• panendoscopy
• nasopharyngoscopy
• laryngoscopy
bronchoscopy with brushings combagascopy
 esophagoscopy biopsy of normal tissue of nasopharynx, tonsils, base of
tongue and hypopharynx
if primary still occult (5%) - excisional biopsy of node for diagnosis,
manage with radiotherapy or neck dissection (squamous cell carcinoma) if primary found, stage and treat
CONGENITAL NECK MASSES
Branchial Cleft Cysts/Fistulae (see Colour Atlas I4)
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☐ 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 surgical removal of cyst or fistula tract
if infected - allow infection to settle before removal
Thyroglossal Duct Cysts (see Colour Atlas I3)
☐ 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

0	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)
Cy U	stic Hygroma lymphangioma arising from vestigial lymph channels of neck 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 surgical excision if it fails to regress - difficult dissection due to

NEOPLASMS OF THE HEAD AND NECK

Location	Presentation	Risk/Etiological Factors	Diagnosis	Treatment
Nose/Paranasal Sinus	depends on where tumour has invaded through bone	hardwood dust nickel chromium	clinical suspicion on CT biopsy	surgery + radiation
Nasopharynx	nasal obstruction neck mass epistaxis unilat. SOM	EBV salted fish nickel exposure poor hygiene	flexible scope biopsy CT/MRI	1º radiation surgery 2nd line
Lip	white patch on lip lip Ulcer	UV light poor hygiene smoking/EtOH	biopsy	1º 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	1º surgery radiation 2nd line
Oropharynx	odynophagia otalgia enlarged tonsil fixed tongue with trismus	smoking and EtOH	biopsy	1º radiation surgery 2nd line
Hypopharynx	pain and dysphagia otalgia cervical node hoarseness	smoking and EtOH	rigid scope CXR CT	1º radiation surgery 2nd line
Larynx	dysphagia, otalgia odynophagia hoarseness foreign body feeling dyspnea/stridor cough/hemoptysis	smoking and EtOH	indirect and direct laryngoscopy CT	1º radiation surgery 2nd line
Thyroid	thyroid mass vocal cord paralysis cervical nodes hyper/hypo thyroid	radiation exposure family hx	see figure 16	1º surgery I ¹³¹ for metastatic deposits



PRINCIPLES OF MANAGEMENT ☐ initial metastatic screen includes chest x-ray and LFT's; ☐ scans of liver, brain and bone only if clinically indicated ☐ 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 no role for chemotherapy in tumours of the head and neck 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
CARCINOMA OF THE NOSE AND PARANASAL SINUSE □ rare tumours with decreased incidence over the last 5-10 years □ increased incidence in Africans, Japanese, and Arabs □ 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 NASOPHARYNX ☐ 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 South Chinese origin 50-59 year old age group, M:F = 2.4:1

Notes

	etiological factors include EBV, salted fish consumption, nickel exposure, poor hygiene squamous cell carcinoma most common (approximately 90%) lymphoma (approximately 10%)
	resentation 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)
	lagnosis 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
	reatment 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
	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%
	ARCINOMA OF THE LIP 50-70 year age group whites > blacks M:F = 30:1 95% squamous cell carcinoma
	ciology UV light - to lower lip poor oral hygiene smoking and alcohol contribute but are less significant than in other head and neck SCC
	resentation 85% lower lip dyskeratosis manifests as white patch on lip (actinic chelitis) ulcer formation may indicate carcinoma
Di	agnosis biopsy
	reatment 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
	rognosis 85% 5-year survival following surgery 80% 5-year survival following radiation therapy

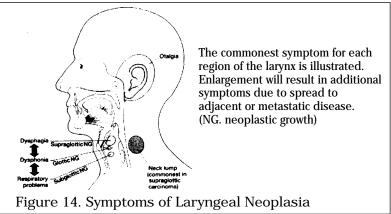
	ALIVARY GLAND NEOPLASMS
	M=F 80% of salivary gland tumours are parotid submandibular tumours uncommon (10%), sublingual rare (1%) only 20% of parotid swellings are malignant, whereas 75% of submandibular gland swellings are malignant, generally the smaller the gland the greater chance of malignancy
	athology 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%
	arotid Gland painless slow-growing mass if bilateral, suggests benign process (Warthin's tumour, Sjögren's, 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
	agnosis fine needle biopsy CT scan to determine depth of tumour
	reatment 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 with
	sural nerve, or hypoglossal attached to remaining stump rognosis benign: excellent, although pleomorphic adenomas may recur 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
	ARCINOMA OF THE ORAL CAVITY oral cavity consists of the anterior/oral tongue, the floor of mouth, the alveolus, the retromolar trigone, and the hard palate above 1.5-3% of all cancers occurring in North America 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 (younger trend in recent years) 95% squamous cell (others include salivary gland: mucoepidermoid, adenoid cystic, acinic cell, also sarcoma and melanoma)

Notes

	ciology 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
0000000	resentation 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 mets 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
	iagnosis adequate visualization is key small local biopsy of lesion imaging studies generally not required unless mandibular involvement is suspected or planning extensive resection
	reatment 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
00	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
	ARCINOMA 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 alcohol abuse and smoking 90% squamous cell carcinoma - poorly differentiated
	resentation 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 dependant ☐ 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 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) piriform 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 alcohol, tobacco □ associated with Plummer-Vinson syndrome (post-cricoid region)
Presentation often presents late pain dysphagia otalgia cervical node +/- hoarseness
Diagnosis □ clinical suspicion - definitive diagnosis often by rigid endoscopy □ chest x-ray to rule out pulmonary mets □ 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 LARYNX



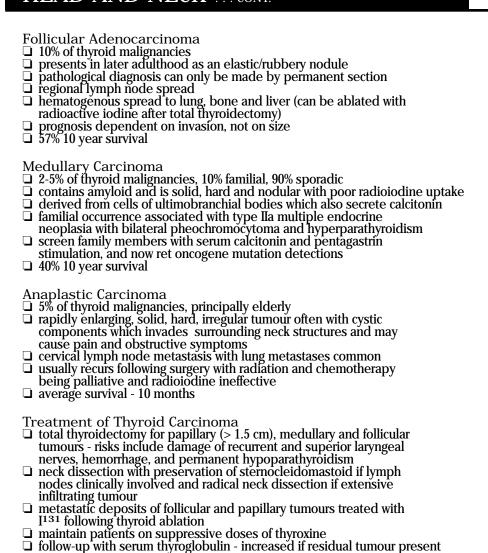
Reproduced with permission from Churchill Livingston, Dhillon, R.S, East, C.A. Ear, Nose and Throat and Head and Neck Surgery. Churchill Livingston, UK, 1994. □ squamous cell most common ☐ 45% of head and neck carcinoma ☐ common between 45-75 years of age ☐ M:F = 10:1 etiologic agents include heavy smoking 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 glottic (60-65%) few lymphatic channels nodál metastasis rare · tumour remains local for a long period produces hoarseness early giving a better prognosis subglottic (1%) abundant lymphatics, lateral neck and paratracheal nodes are involved at presentation in 20% symptoms occur late
may be difficult to distinguish if primary tumour arises in subglottis or in trachea

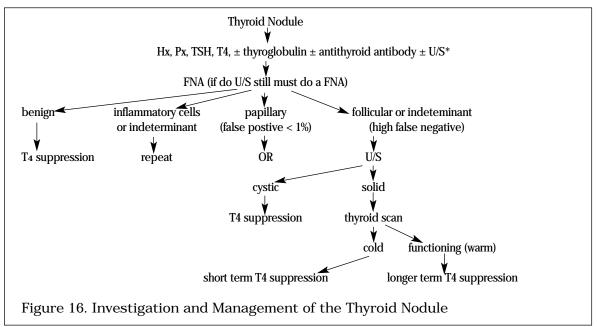
dysphagia, odynophagia or referred otalgia (suggest supraglottic lesion)
hoarseness (suggests glottic involvement)
clearing throat/foreign body feeling
dyspnea/stridor
cough/hemoptysis regional lymphadenopathy Diagnosis \Box direct and indirect laryngoscopy to assess site and extent of tumour and cord 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 tracheostomy +/- 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		
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 MEN II (medullary ca.) ☐ 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 of benign indirect laryngoscopy - vocal cord paralysis increases suspicion of malignancy ☐ cervical lymphadenopathy - deep cervical chain suggestive of	ţ	
metastatic disease signs of hypo/hyperthyroidism		
THYROID CARCINOMA		
Papillary Adenocarcinoma accounts for 60-70% of thyroid cancers can be multifocal lymphatic spread presents in early adulthood as a solitary nodule with 20% having palpable lymph nodes late metastases to lungs or bone rate of growth may be stimulated by TSH microscopically - papillary projections of columnar epithelium with nuclear notching, cytoplasmic inclusions, and 60% having Psammoma bodies (a mixed papillary-follicular or follicular variant also found)		





^{*} 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