**GENERAL AND PAEDIATRIC ORL**

**Introduction to Otolaryngology**

Otolaryngology deals with all of the problems arising in the head and neck, with the exclusion of the intracranial and intraorbital contents (and even then there is usually discussion). It is divided into a number of subspecialties which each deal with their individual subset. Note that ORL problems are extremely common in the community, and a GP can expect to spend 30% of their time on ORL issues.

**Common ORL symptoms:**

1. **Ear** – hearing loss, otalgia, fullness, pressure, vertigo, tinnitus, discharge, facial n. paralysis
2. **Nose** – nasal obstruction, rhinorrhoea, anosmia/hyperosmia, postnasal drip, facial pain, halitosis, sneezing, itchiness, bleeding, headaches
3. **Throat** – sore throat, dysphagia, odynophagia, dysphonia, haemoptysis, 'lump in the throat', regurgitation, chronic cough, throat clearing
4. **Neck** – lump, swelling, pain/discomfort, shortness of breath, tightness, ache

**Neck Masses in Adults and Children**

**Paediatric neck masses** are generally either congenital (e.g. branchial cysts) or infective (most commonly lymphadenopathy 2° to URTI). Malignancies are extremely rare (usually lymphoma or rhabdomyosarcoma), but should be considered in the differential.

1. **Cervical lymphadenopathy and abscesses**
   a. Usually due to viral (EBV) or bacterial (*Staph.*, *Strep.* – 53-89%) infection
      i. Adenopathy of anterior cervical chain almost always infectious
      ii. Adenopathy of posterior cervical chain malignant in ~50% of cases
   b. Clinical features:
      i. History – age, systemic manifestations, associated infections, travel, tuberculosis exposure, ingestion of undercooked foods or unpasteurized milk
      ii. Examination – adenopathy, erythema, heat, tenderness, lymphangitis, suppuration; fever, rash, generalised adenopathy, hepatosplenomegaly
      iii. Investigations – FBC, ESR, EBV test, swab/biopsy and culture, USS/CT
   c. Management:
      i. Parenteral therapy if age <1/12, moderate to severe systemic manifestations, suppurative nodes or overlying cellulitis
      ii. Treat with appropriate antibiotics for 10-14 days (5 days after symptoms resolve); may need incisions and drainage if no response after 2-4/52

2. **Non-tuberculous mycobacterial lymphadenitis:**
   a. Usually due to atypical mycobacteria e.g. *Mycobacteria avium*
   b. Presents with a slowly enlarging mass in the submandibular or parotid nodes
      i. Patient usually well despite enlarging mass
      ii. May be associated with overlying skin erythema, fistula → chronic drainage
   c. Management:
      i. Excision (not incision and drainage)
      ii. Standard anti-TB therapy is ineffectual, but need to exclude pulmonary TB

3. **Congenital neck masses**
   a. Midline:
      i. Thyroglossal duct cyst – may enlarge with infection; treatment by excision (must remove body of the hyoid to prevent relapse – Sistrunk’s procedure)
      ii. Midline dermoid cyst – less common, not attached to deeper neck structures
   b. Lateral – branchial sinuses, cysts, fistulae
      i. Abnormalities of the 1st branchial apparatus occur in the region of the parotid gland, while all others present in the anterior triangle of the neck
      ii. Usually infected (rarely malignant), treatment is by surgical excision
   c. Others (hamartomatous conditions):
      i. Haemangiomas – vascular malformations, capillary/small vessel proliferation
         1. Normal history is proliferation, involution, fibrosis over a few years
         2. Treatment – steroids, sclerosants, laser, excision, embolisation
      ii. Lymphangiomas – vascular malformations similar to haemangiomas
         1. Commonly seen as a cystic hygroma in the submandibular region or floor of the mouth
         2. Treatment is surgical, but high recurrence due to infiltrating nature

4. **Congenital syndromes:**
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a. Down syndrome (trisomy 21) – otitis media, narrow ear canals, glossophtosis, upper airway obstruction, cholesteatomas, cardiac problems (VSD), unstable cervical spine
b. Cruzan's syndrome – craniofacial abnormalities, upper airway obstruction, Eustachian tube dysfunction, otitis media with effusion
c. Cleft lip and palate (1:1000 live births) – otitis media (100%), feeding difficulties, septal deviation, external nasal deformity. Lip repair at age 3/12, palate at 9-12/12
d. Treacher-Collins syndrome – auriculomandibulo facial dysplasia, hypoplastic mandible, upper airway obstruction, tracheotomy, ear anomalies, mixed hearing loss

Adult neck masses can be roughly divided into acute or chronic onset:
1. Acute:
   a. Differentiate between glandular (thyroid, submandibular, parotid) or nodal
   b. Associated symptoms – sore throat, dental problems, trauma, localised tenderness
   c. Common presentations:
      i. Generalised lymphadenopathy – infection
      ii. Saliva outflow obstruction – infection, ductal calculus
      iii. Thyroid enlargement – thyroiditis, bleeding into a thyroid cyst
2. Chronic – usually painless, non-fluctuant and persistent
   a. Differentiate between glandular (thyroid, submandibular, parotid) or nodal
   b. Multiple lymph nodes (unilateral or bilateral) usually lymphoma or chronic infection
   c. Solitary, painless and firm nodal mass is a malignancy until proven otherwise:
      i. Associated symptoms – referred otalgia, dysphagia, URT bleeding, nasal obstruction, middle ear effusion, stridor, hoarseness
      ii. Nodes in supraclavicular fossa may be metastases from lung/kidney/breast (examine, CXR, USS) but 1st head and neck tumours are more common
      iii. Refer to an ENT surgeon for assessment, biopsy and treatment

General management of neck masses:
1. FNA (histology), USS (glandular masses), CT neck (looking at the actual mass and relations, may show other lymph nodes or primary lesion), MRI (help differentiate soft tissue masses)
2. Suspicious nodal masses (even if FNA negative) are an indication for endoscopy – involving examination under GA and biopsy of suspicious lesions and sentinel regions (nasopharynx, base of tongue, tonsil and pyriform fossa)

• Head and Neck Cancer (the lecture was fucking useless)

General principles
1. Classification – by tissue that the tumour arises from
   a. Mucous membranes – squamous cell carcinoma (oral cavity, larynx, pharynx)
   b. Salivary glands (usually parotid) – adenomas, adenocarcinomas, mucoepidermoid
   c. Skin cancers – squamous cell carcinoma and basal cell carcinoma
   d. Others – nasopharyngeal carcinomas, papillary/follicular carcinomas, lymphomas
2. Common head and neck cancers in adults:
   a. Tongue cancers most common, usually lateral (~25%)
   b. Lip cancers (good prognosis) and floor of mouth cancers (~14% each)
   c. Tonsillar cancers (~12%)
   d. Other parts of mouth, total palate, pyriform sinus (~8% each)
   e. Nasopharyngeal cancer (~5%)
   f. Hypopharynx and oropharynx cancers least common (~4% each)
3. Risk factors include tobacco, increasing age, alcohol (>15x risk if combined with smoking), HPV type 16 infection (2x risk), occupational exposures (e.g. textiles – sinonasal carcinoma)
4. Screening is debatable:
   a. American Cancer Society and National Cancer Institute recommend very careful screening exam by a general internist and dentist
   b. US Preventative Task Force says there is insufficient evidence for general screening, but recommends screening for high risk patients
   c. Assess for leukoplakia, erythroplakia, frank neoplasms, lymphadenopathy and evaluate salivary glands carefully

Clinical features:
1. History:
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1. Symptoms
   a. Oral cavity lesions that do not heal or that bleed easily
   b. Chronic hoarseness or cough or change in voice (laryngeal carcinoma)
   c. Nasal obstruction and/or recurrent epistaxis (nasopharyngeal or sinus carcinoma)
   d. Persistent lymphadenopathy or neck mass
   e. Odynophagia, dysphagia, sore throat (hypopharyngeal or oesophageal lesions)

2. Examination
   - complete head and neck exam essential. Important issues include:
     a. Careful examination of mucous membranes – examination and palpation
     b. Indirect and fiberoptic (direct) laryngoscopy
     c. Serous otitis media → nasopharyngeal carcinoma (obstructed Eustachian tube)

3. Investigations:
   a. FNA for neck nodes or masses has >90% accuracy
   b. CT with contrast or MRI
   c. Staging by TNM and clinical systems

4. Treatment
   - multiple modalities are required for all but the most localised lesions
     a. Surgery – conservation of organ function with removal of tumour, often disfiguring
     b. Radiation therapy – substantial side-effects (mucositis, xerostomia)
     c. Chemotherapy as adjuvant (useless – 10% effect) or for metastatic/advanced disease

• Facial Plastics

Otoplasty
   - note correction of prominent ears is psychologically very rewarding for child and parents
   1. Unfolded helix (most common) → smooth contour and prominent pinna
   2. Posterior and anterior techniques modifying cartilage to recreate an antihelical fold
   3. Overcorrection → telephone deformity (upper antihelix/lobule protrude over middle antihelix)

Rhinoplasty may be functional, cometic or reconstructive. A good understanding of the nature of tissues and healing – and patience – is required. Preoperative planning and photographic documentation is also crucial. It should restore a normal form to the nose so it harmonises with the rest of the face, and the function of the nose and nasal sinuses should be maintained, improved or restored.
   1. Approach – endonasal (minimally invasive) or external (incisions made for better exposure)
   2. Elevation of soft tissue covering the nasal skeleton
   3. Isolation, mobilisation and correction of all skeletal elements
   4. Union of the mobilised and corrected skeletal parts → pleasing/functional nasal framework
   5. Fixation of the mobilised, corrected skeletal elements in the required position

Septoplasty (note that rhinoplasty usually needs septoplasty, thought the reverse is not always true)
   1. Indications – improve function, access, graft material, treatment of associated conditions
   2. Complications – failure, infection/bleeding, septal perforation, altered tip sensation, upper teeth numbness, cosmetic (saddle deformity, columella retraction), others

Basic plastic-reconstructive procedures of the head and neck:
   1. Free skin grafts – various thicknesses, used to cover superficial defects of the skin/mucosa
   2. Local flaps – derived from tissue adjacent to the defect with intact circulation, positioned by advancement or rotation. Note that relaxed skin tension lines are crucial for minimal scarring

Ear
• Disorders of the External Ear

Congenital disorders:
   1. 1st-2nd brachial arches – abnormal fusion → external ear and canal abnormalities
   2. Atresia
   3. Pre-auricular pits – treat symptoms with antibiotics, can be excised

Infection:
   1. Otitis externa:
      a. Acute localised (furunculosis) – Staph. infection of a hair follicle in the ear canal
         i. Pain exacerbated by movement of the auricle or pressure on the tragus, mild hearing deficit, swelling, lymphadenopathy
         ii. Management – insertion of an antiseptic wick or hygroscopic agents to reduce oedema, systemic antibiotics, incision and drainage if not settling
      b. Acute diffuse (‘swimmer’s ear’) – usually bacterial (Pseudomonas) or fungal

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i. Pain, itching, swelling and oedema; usually in hot, humid weather or following trauma. May spread to the pinna → perichondritis

ii. Management – swab and culture, local ear toilet, topical antibacterial/steroid or antifungal drops, systemic antibiotics if resistant

c. Chronic – irritation of drainage from the middle ear in patients with suppurative OME

i. Associated with reactive inflammation (eczema) – local itching → scratching

ii. Avoid local trauma, local ear toilet, steroids, antibacterial in active phase

d. Malignant – severe necrotizing Pseudomonas infection with local tissue invasion

i. Diabetics, elderly and debilitated patients at increased risk

ii. Severe, unrelenting pain and tenderness, otorrhoea, hearing loss, fever; may spread to the skull base → cranial nerve damage, meninges, brain → death

iii. CT scan, technetium scintigram, gallium scintigram to monitor treatment

iv. Treatment – repeated debridement, topical anti-pseudomonal or acetic acid drops, systemic antibiotics with anti-pseudomonal cover (e.g. ciprofloxacin)

2. Otomycosis – fungal infection (Aspergillus, Actinomyces, Candida) on cerumen and detritus

a. Associated with humid conditions, immunosuppressed individuals

b. Scaling, itching, pain; may become chronic; treat with topical antifungal drops

3. Herpes Zoster – reactivation of virus in the geniculate ganglion → Ramsay-Hunt syndrome

a. Facial nerve involvement – ear, palate, pharynx or neck

b. Pain and vesicles in external auditory canal with hyperacusia

c. Loss of sense of taste in the anterior 2/3 of tongue

d. Ipsilateral facial palsy

4. Chondritis – infection of cartilage, often 2° to trauma → abscess, necrosis → ‘cauliflower’ ear

Miscellaneous:

1. Cerumen – waxy substance (modified sebum) excreted from ceruminous glands

a. Beneficial (waterproof, low pH, IgA antibodies, lysozyme), removal may → infection

b. Hearing loss if accumulates → obstruction, or if pushed against tympanic membrane

c. Remove by gentle probing under direct vision or softening ear drops followed by suction or gentle syringing (do not syringe if tympanic membrane perforated)

2. Contact dermatitis -

3. Exostosis – benign bony swellings of the ear canal (hyperplasia rather than neoplasia)

a. Seen in swimmers, surfers and divers – response of periosteum to repeated cooling

b. Large exostoses can cause wax accumulation and otitis externa → surgical excision

4. Keloids – hyperplastic scar tissue, higher risk with highly pigmented people

5. Keratosis obliterans – ‘canal cholesteatoma’, recurrent keratin accumulation → obstruction

Trauma:

1. Foreign bodies – few cause trouble unless the foreign body is inadvertently forced further into the ear canal where it may damage the drum and middle ear; if in doubt, refer

2. Haematoma – extravasation of blood between the cartilage and perichondrium

a. If untreated, the blood becomes organised → cauliflower deformity (fibrous tissue)

b. Other complications include perichondritis, abscess, necrosis → auricular deformity

c. Treatment – prevention, evacuate with firm dressing or mattress suture to prevent reaccumulation, broad-spectrum antibiotics to prevent perichondritis

3. Lacerations – meticulous suturing

4. Perforation of the tympanic membrane

a. Observe if small and edges not everted; keep water out of ear

b. May need referral for eversion of edges and paper patch

c. Antibiotics if infected or water involved – most heal spontaneously, may need surgery

Tumours:

1. Benign:

a. Ceruminoma – tumour of ceruminous glands, rarely malignant change. Treated by wide local excision, radiotherapy if histology is malignant.

b. Osteoma – slow-growing mass of mature, predominantly lamellar bone, usually unilateral and arising from the skull or mandible inferior to the canal

2. Malignant – squamous cell carcinoma, basal cell carcinoma, melanoma

a. Prognosis depends on location, extent and type – SCC → 50% 5yr survival, BCC 65% 

b. Medial part of ear canal and middle ear involvement is a poor prognostic sign
Disorders of the Middle Ear

Otitis media:

1. **Acute otitis media**
   a. Aetiology – *Pneumococcus*, other *Strep.*, *H. influenzae*, *M. catarrhalis*, *S. aureus*
      i. Poor Eustachian tube function, particularly in children and with URTIs
      ii. Lower immunity in children
      iii. Nasopharyngeal sepsis (adenoiditis, acute sinusitis with purulent discharge)
      iv. Incidence higher in males, kids in day-care, family history, immunodeficient
   b. Symptoms – earache, irritability, pulling at ears, fever, malaise, hearing loss
   c. Signs – bulging red tympanic membrane → may rupture and discharge
   d. Treatment – analgesics ± oral antibiotics, assess resolution; recurrent → grommets, treat nasopharyngeal sepsis (adenoidectomy), low-dose prophylactic antibiotics
   e. Complications – incomplete resolution (→ OME), recurrent (→ tympanosclerosis, permanent perforation), acute mastoiditis (red swelling, systemic disturbance)

2. **Otitis media with effusion** (secretory otitis media, ‘glue ear’)
   a. Aetiology – multifactorial including poor Eustachian tube function, URTI, recent AOM, palatal abnormality (e.g. cleft), nasopharyngeal abnormality (adenoids, malignancy)
   b. Symptoms – relatively silent, may have conductive hearing loss, behavioural change, poor school performance, balance disturbance. Often picked up during screening
   c. Signs – dull, hypervascular, immobile drums ± retraction
   d. Treatment – most resolve spontaneously, may try antibiotics after 4/52 (e.g. 4/52 o cotrimoxazole), treat any nasal pathology, consider grommets if continues 3/12
   e. Sequelae – language/educational delay, middle ear changes (atelectasis, adhesive otitis media, cholesteatoma)

3. **Chronic suppurative otitis media**
   a. Mucosal CSOM (‘tubo-tympanic’ disease, ‘safe ears’)
      i. Repeated or unresolved infection → chronic tympanic perforation → hearing loss, otorrhoea, fibrosis of middle ear, tympanosclerosis, ossicle defects
      ii. Signs – central perforation ± purulent discharge, white plaques of tympanosclerosis on the tympanic membrane, conductive hearing loss
      iii. Treatment – swab and culture, local ear toilet, topical antibiotics/steroid drops ± oral antibiotics, repair membrane/ossicle (myringoplasty, ossiculoplasty)
      iv. Generally benign, but occasionally leads to intracranial complications
   b. Cholesteatoma (‘attico-antral’ disease, ‘dangerous ears’)
      i. Combination of chronic negative middle ear pressure and chronic inflammation → expanding cyst-like lesion in the middle ear, attic or mastoid
         1. Lined with keratinising stratified squamous epithelium, full of keratin, surrounded by granulation tissue
         2. Erosion of surrounding structures:
            a. Ossicular chain → conductive hearing loss
            b. Labyrinth → sensorineural hearing loss, vertigo
            c. Facial nerve canal → facial palsy
            d. Intracranial → meningitis, abscess, lateral sinus thrombosis
      ii. Symptoms – chronic, offensive-smelling discharge, hearing loss
      iii. Signs – attic or postero-superior marginal perforations
      iv. Treatment – local treatment as per mucosal CSOM, surgery to remove the disease and restore hearing where possible (mastoidectomy, tympanoplasty)

Otosclerosis is a disorder of the otic capsule in which new spongy bone formation causes fixation of the anterior end of the stapes footplate and a conductive hearing loss, occasionally with cochlea invasion leading to sensorineural hearing loss.

1. **Aetiology**
   a. Hereditary in 50%, autosomal dominant with 30% penetrance
   b. Exacerbated by high oestrogen levels in pregnancy
2. **Pathogenesis** – immature spongy bone laid down → replaced with hard, mineralized bone
3. **Presentation** – hearing loss and tinnitus, often bilateral
4. **Management:**
   a. Sodium fluoride, calcium and vitamin D to stabilise active disease
   b. Surgery – stapedectomy, stapedotomy → corrects conductive hearing loss
   c. Hearing aids – if surgery unacceptable and to correct sensorineural hearing loss
Tumours of the middle ear:

1. **Squamous cell carcinoma**
   a. Increased incidence with CSOM, often presents with exuberant granulation tissue difficult to distinguish from that seen in SOM
   b. Presentation – pain, bleeding, facial palsy, vertigo, signs of intracranial spread
   c. Investigations – biopsy (diagnosis), CT (extent)
   d. Treatment – radical surgery (petrosectomy) and radiotherapy, very poor prognosis

2. **Glomus tumours** – slow growing, usually benign (96%) but locally invasive vascular tumour arising from the chemoreceptors of the jugular bulb (glomus jugulare) or Jacobson’s nerve on the promontory of the middle ear (glomus tympanicum)
   a. Incidence – peak age incidence in 30s, F3:M1, 10% have multiple tumours
   b. Pathogenesis- can spread intracranially or to the neck, profuse blood supply (as it’s a vascular tumour, silly) → bleeds profusely if probed or biopsied
   c. Presentation – pulsatile tinnitus, conductive hearing loss, CN palsies (jugular foramen syndrome), visible tumour behind tympanic membrane, blanches on pressure
   d. Investigations – CT scan, 4-vessel angiography (don’t biopsy!)
   e. Treatment – surgical excision (extensive skull-base approach with preoperative embolisation of feeding vessels), radiotherapy if surgery not possible (palliative)

Selected aspects of middle ear surgery:

1. **Grommets**
   a. Indications – unresolving OME, recurrent AOM, chronic Eustachian dysfunction
   b. Inserted under GA in children (LA in adults), usually stay in for 1-2 years
   c. Minor swimming restrictions – no diving, deep swimming or hot pools
   d. Complications:
      i. Chronic otorrhoea – note that acute otitis media also leads to a discharge
      ii. Perforation after tube extrudes (1-2%)
      iii. Atrophic or sclerotic area in old grommet site
      iv. Implantation cholesteatoma (very low incidence)

2. **Mastoid surgery** – mainly used for removal of cholesteatoma (no longer used for mastoiditis)
   a. Canal-wall-up procedure preserves the ear canal and requires less postoperative care, but has a 40% change of recurrence/persistence
   b. Canal-wall down procedure is 95% effective in removing disease but leaves a larger canal opening and large cavity (mastoid bowl) that needs cleaning every 6-12 months
   c. Complications – numbness of lateral tongue (chorda tympani nerve), dizziness or infection (2° to water in ear), hearing loss, dizziness, dura injury, facial nerve palsy

3. **Myringoplasty** – repair of tympanic membrane
   a. Margins of the hole are rimmed (removal of skin and tissue) and a small piece of fat placed into the hole, can be done quickly under local anaesthetic in a GP setting
   b. Complications – failure to close the hole or to improve hearing and numbness of the lateral tongue; rarely vertigo and injury to the facial nerve → facial paralysis

4. **Stapedectomy** – treatment of otosclerosis by removal and replacement of stapes
   a. Stapes and all or part of its footplate is removed and a prosthesis placed between the incus and the inner ear to restore hearing
   b. Distance between stapes footplate and saccule is critical – in patients with Meniere’s disease the endolymph bulges towards the footplate (risk of post-operative deafness)
   c. Complications – lateral tongue numbness, dizziness, hearing loss, deafness, eardrum perforation; rarely facial paralysis, meningitis

5. **Tympanoplasty** – repair of middle ear (typically tympanic membrane ± middle ear bones)
   a. Formal tympanoplasty – middle ear entered via a canal skin flap and a piece of muscle tendon or fascia is placed beneath the perforation
   b. Tympanoplasty plus ossiculoplasty (usually with an ossicular prosthesis)
   c. Complications as for myringoplasty

• Disorders of the Inner Ear

**Deafness in early childhood**

1. **Genetic issues** – 60% of all deafness in early childhood is inherited
   a. 80% is autosomal recessive, usually stable but can be progressive
   b. 20% is dominant, sex-linked or mitochondrial

2. Pathogenesis:
   a. Malformation of organ of Corti with loss of hair cells (deaf white cats, Dalmatians)
   b. Most auditory fibres are preserved (initially)
   c. Surrounding bone may also be malformed
   d. Conductive hearing loss – atresia of ear canal with microtia

3. Aetiology:
   a. Inherited causes
   b. Birth/post-natal problems – low birth weight, prematurity, hypoxia, sepsis, jaundice
   c. Meningitis – ossification of scala tympani, CNS problems → urgent cochlear implant
   d. Other infections (TORCHS – toxoplasmosis, other (PID, bacterial vaginitis), rubella, CMV, HSV and HIV, syphilis) – CMV is the commonest infectious cause:
      i. 1-2% of live births, 90% have no overt symptoms as neonates, 15% → loss
      ii. 15 per year in NZ have CMV inclusion disease

4. Detection – if a parent suspects a hearing problem in their child, there is one until proven otherwise by formal audiometric testing
   a. Tests:
      i. Auditory brainstem response (electrophysiological testing) may be useful
      ii. Behavioural testing – operant conditioning for 8-9 months performed in a sound filed, play audiometry from 2½ to 3 years of age
      iii. Otoacoustic emissions
   b. Screening should ideally be done on all newborns, but the current median age of diagnosis is 39 months.
      i. Automated auditory brainstem response ± otoacoustic emissions
      ii. Immediate referral for confirmation – avoid the “middle man” stage
      iii. Screening (audiogram) is also performed at school entrance and variable tympanometry is performed in preschools

5. Management – note that diagnosis often leads to grief as the majority of parents have normal hearing. It is important to explain carefully, keep the parents talking and communicating with their child, and arrange for them to meet other parents with similar experiences
   a. Genetic testing may be useful if the parents wish to have other children
   b. A hearing aid should be fitted immediately with regular mould changes, consider cochlear implantation for congenital profound deafness
   c. Consider oral or signing (whole body communication) or both
   d. Ongoing rehabilitation to reinforce development of nerve connections in the brain via sound stimulation (teaches child to attach meaning to sound)

Progressive hearing loss is one of the commonest causes of disability, though the exact economic effects are unknown. Most cases are sensorineural, and the majority of losses are idiopathic.

1. It may be difficult to estimate the effects on the individual, and a good rule of thumb is the hearing in the better ear:
   a. None (<30dB)
   b. Mild (30-50db) – some mistakes in conversation, some environmental sounds lost, tend to present around 45db
   c. Moderate (50-70db) – approximately 50% of conversation lost, many environmental sounds lost, requires hearing aids
   d. Severe (70-90db) – no conversation, most environmental sounds lost, dependant on hearing aids – may be a candidate for cochlear implant
   e. Profound (>90db). – no conversation, almost all environmental sounds loss, hearing aids ineffective – often a candidate for cochlear implant

2. Aetiology – extremely difficult to study, many factors involved over a lifetime
   a. Certain – noise, trauma, drugs
   b. Probably – genetics, atherosclerosis, diabetes, diet, stress

3. Pathology
   a. Hair cell loss – noise, drugs, presbycusis
   b. Neuronal loss – vestibular schwannoma, trauma, meningitis
   c. Central auditory pathway – lack of stimulation
4. Hearing aids:
   a. New hearing aids – implanted hearing aids and bone anchored hearing aid
   b. Cochlear implants – always a restricted subset of patients

Common causes of acquired hearing loss:
1. **Presbycusis** – hearing loss in the elderly ?2° to normal ageing or lifetime noise exposure
   a. Loss of hair cells, neural atrophy in spinal ganglion, atrophy of stria vascularis
   b. May be related to reduced elasticity of basilar membrane, central pathways
   c. Symptoms – gradual, high frequencies first (“everyone mumbles”), tinnitus
   d. Treatment – hearing aids, counselling on how to maximise remaining hearing
2. **Noise-induced hearing loss** – prolonged exposure to >85db (intensity, duration, susceptibility)
   a. Damage and loss of cilia on hair cells → neural degeneration
   b. Symptoms – tinnitus, 4-6Hz dip on hearing thresholds (may also → other frequencies)
   c. Treatment – prevention, hearing aids, ACC for rehabilitation
3. **Ototoxicity** – aminoglycosides, cytotoxic drugs, ?frusemide, quinine/salicylates (reversible)
   a. Aminoglycosides concentrate in inner ear fluids and kidney
   b. Damage to stria vascularis, hair cells → sensorineural loss (also vestibular problems)
   c. Management – monitor blood levels (especially in renal failure), serial audiometry
4. **Sudden sensorineural deafness**
   a. Hair cells – ototoxicity, trauma (including noise), viral infection, meningitis, labyrinthitis
   b. Round window – perilymph fistula (rupture)
   c. Hydrops – Meniere’s syndrome, syphilis
   d. Neural lesions – acoustic neuroma, MS, brainstem tumours, CVA → brainstem nuclei
   e. Others – vascular occlusion, viral infection, membrane rupture, cellular problem
5. **Acoustic neuroma** – slowly growing benign tumour derived from the neurilemmal sheath
   a. Most common intracranial tumour – asymmetrical sensorineural hearing loss must be investigated for acoustic neuroma with MRI (CTI and ABR if MRI not available)
   b. Symptoms – slowly progressive sensorineural hearing loss, tinnitus; onset usually insidious (vertigo is unusual). Bleed into tumour may → sudden onset symptoms
   c. Untreated → facial/trigeminal nerve symptoms, ↑intracranial pressure, death
   d. Treatment by excision, though some risk of facial nerve damage. Most lose hearing unless diagnosed early, at which point there is some possibility of preservation

**Cochlear implants** provide auditory sensations to severely or profoundly deaf individuals by direct stimulation of the nerve endings in the inner ear. It consists of an external component (microphone, transmitter and speech processor) and an internal component (receiver and electrode array).
1. **Benefits** include environmental sound, improved lip-reading ability, better speech monitoring and production – up to 70% of patients are able to understand speech
   a. Depend on age of onset of deafness, previous hearing experience, length of profound deafness, personal motivation and ongoing habilitation
   b. Misconceptions – normality of speech sounds, effects of background noise, invisibility, no need for ongoing habilitation
2. **Candidates:**
   a. Severe to profound hearing loss in adults
   b. Children <2yrs (must be <4yrs for full benefit) – note surgery vs vibrotactile aid
   c. Marginal benefit for hearing aid users, pre-lingual adults
   d. No benefit for acquired pre-lingually deaf adults, congenitally deaf adults
3. **Follow-up:**
   a. Habilitation – mapping changes every 3-6 months for the first year, 6 monthly in the second year and annually thereafter
   b. Children need a far more intensive program (particularly the congenitally deaf) and a 4-5 year structured course is necessary to maximise the use of the implant

**Balance and peripheral vestibular disorders** – note that 20% of people over 65 years complain of dizziness (sensation of losing contact with the environment), while vertigo (hallucination of movement) is generally more serious and implies a vestibular disorder. As a general rule vertigo lasting >2/52 is central until proven otherwise (indicated by abnormal nystagmus – MLF → vestibulo-ocular reflex)
1. **Anatomy and physiology:**
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a. Maintenance of equilibrium with the environment is performed by the vestibular end organs, eyes and limb proprioceptors. The input is modulated by the cerebellum, and the output from the vestibular spinal tract acts on postural and eye (fixation) muscles

b. Disorder of input:
   i. False sense of movement (vertigo)
   ii. Oculomotor pathways attempt to fixate on “moving environment” (nystagmus)
   iii. Movement of postural muscles 2° “moving environment” (staggering, ataxia)
   iv. Involvement of other brainstem centres (e.g. vomit centre)

c. Resolution:
   i. Recovery of normal inputs (e.g. after bout of Meniere’s disease)
   ii. Central adaptation to new input (labyrinthitis, labyrinthectomy, temporal #)
   iii. Note that the ability to adapt deteriorates in old age

2. Meniere’s disease – distension of endolymphatic compartment of the inner ear
   a. Symptoms – fluctuating sensorineural hearing loss, tinnitus, severe vertigo
   b. Triggers – stress, local sepsis, nicotine, hypothyroidism, diabetes
   c. Treatment – avoid triggers, diuretics, vasodilators, vestibular suppressants, bed rest
   d. Surgery – endolymphatic decompression, vestibular nerve section, labyrinthectomy

3. Vestibular neuritis – damage of vestibular nerves 2° to viral illness
   a. Symptoms – acute vertigo, nausea, vomiting, nystagmus (no hearing loss)
   b. Acute symptoms generally last several days, and balance disturbance settles over weeks to months with central adaptation
   c. May provide symptomatic relief with vestibular suppressants, sedation

4. Acute suppurative labyrinthitis – rapid, permanent damage to cochlea and vestibular apparatus leading to fibrosis and ossification, usually 2° to middle ear sepsis, meningitis
   a. Symptoms – severe vertigo, acute hearing loss
   b. Parenteral antibiotics – limit damage, prevent spread through cochlear aqueduct

5. Benign paroxysmal positional vertigo – utricular otoconia settle in posterior semicircular canal
   a. Symptoms – sudden brief vertigo on certain head movements, ↓ on repetition
   b. Diagnosed on history and Dix-Hallpike positioning test:
      i. Sit patient up, then lie down with head to one side ➔ strong stimulus
      ii. Repeat – should be reduced impact
      iii. Repeat other side – should have no effect
   c. Self-limiting condition, may try particle repositioning manoeuvres putting the otoliths back into the utricle (instant success in ~80% of patients)

6. Non-labyrinthine causes of dizziness:
   a. Vestibular – postural hypotension, vasovagal, TIV/CVA, vertebrobasilar ischaemia
   b. Neurological – atypical migraine, MS, brainstem or cerebellar tumours
   c. Hyperventilation (respiratory physiotherapy may be useful)

Selected aspects of inner ear surgery

1. Vestibular nerve section
   a. Following inner ear disease, the vestibular nuclei may re-learn a new baseline level of function – central processes are unable to do this if the baseline is fluctuating
   b. Vestibular predictability is the key – however, while a destructive inner ear procedure gives a constant level of peripheral vestibular dysfunction, it will also lose hearing
   c. To circumvent this loss the inner ear is bypassed and the vestibular nerve interrupted by retrosigmoid craniotomy, leaving the cochlea fibres (and hence hearing) intact

2. Temporal bone surgery – Important relations include the facial nerve, internal carotid artery
   a. Middle ear – approach via the external canal (canaloplasty)
   b. Mastoid antrum – approach via cortical mastoidectomy
   c. Both plus posterior tympanotomy ➔ combined approach tympanoplasty
   d. Removing bridge of nose between canal/mastoid ➔ modified radical mastoidectomy

3. Temporal bone fracture
   a. Longitudinal fractures (most common) run in font of the otic capsule, often with an external canal manifestation and rarely with any inner ear damage
   b. Transverse fractures are associated with significant frontal/occipital impact – inner ear function is often lost and the facial nerve may be transected

4. Facial nerve
   a. Idiopathic Bell’s palsy – probably due to HSV affecting the nerve as it traverses the internal meatus, compromising function via oedema within a closed space

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i. Usually evolves rapidly → variable level of compromise and recovery
ii. Management – acyclovir, prednisone, early eye cares, regional imaging
b. Ramsay Hunt syndrome – similar viral entity but due to Herpes Zoster
   i. Paresis of facial nerve with cutaneous vesicles in the cavum concha along
   with a neurosensory hearing loss
   ii. Treatment – acyclovir, prednisone

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C. Traumatic facial nerve injuries – many causes, often vestibular schwannoma removal

i. Usually recover spontaneously but various levels of assistance may be
necessary including nerve decompression, neural repair or bypass
ii. Until function recovers care is required to protect the eye as blink reflexes are
compromised, eye closure is poor and lacrimal function is reduced (due to
close relationship of the 7th nerve to the nervus intermedius)

Audiometry:

1. Pure tone audiometry – identifies thresholds of hearing at selected frequencies (250-8000Hz)
   a. Air conduction testing uses headphones, bone conduction uses vibrator on mastoid
   b. Air-bone gap → conductive hearing loss; both reduced → sensorineural hearing loss
   c. Note that the other ear needs to be masked with white noise when bone conduction is
   being tested as it receives noise at the same time as the other ear

2. Speech audiometry – uses a standard word list heard at specific levels from a tape recorder
   a. Tests the processing of more complex auditory information
   b. Helps to determine the site of the lesion (cochlear good discrimination, neural poor)
   c. Can predict the likely help of a hearing aid

3. Acoustic emittance audiometry – assesses stiffness of conducting mechanism in middle ear
   a. Middle ear pressure ( tympanometry), function of Eustachian tubes, measurement of
   stapedial reflexes
   b. Useful in middle ear disorders and those affecting the stapedial reflex arc

4. Auditory evoked potential – identifies electrical signals in the auditory pathways
   a. Electrocochleography – objective measure of hearing thresholds; needle electrode
   b. Auditory brainstem response – threshold testing in babies, sensorineural hearing loss
   c. Middle latency response – evaluates higher auditory function
   d. Otoacoustic emission testing – measure acoustic output of hair cells on sound stimuli

Nose

• Nasal and Sinus Infections

Functional anatomy of nose and sinuses:

1. Air conditioning (warming, humidification, filtration, absorption/trapping)
   a. Increased surface area – turbinates with mucus glands and rich blood supply
   b. Control of mucus production and blood flow – sympathetic/parasympathetic
   c. Laminar air flow – slit-like passage with even cross-section

2. Smell – slight turbulence required to get air into the olfactory area (specialised sensory cells)

3. Sinuses (bacteriologic control, lighten the face, vocal resonance)
   a. Drainage and ventilation are the most important factors
   b. Depend on amount of mucus, composition, cilia beating, reabsorption, ostia and
   ethmoidal clefts; drainage via ethmoidal infundibulum and frontonasal recess

Rhinosinusitis – acute (<3/52), subacute (3/52-3/12), chronic (>3/12) or recurrent (>3x or >3/12/yr)

1. Aetiology and pathogenesis:
   a. Usually due to S. pneumoniae, H. influenzae or Moraxella catarrhalis
   b. Mucosal swelling and osteomeatal unit obstruction, decreased O2 tension,
   transudation of fluid, impaired mucociliary function, stasis, 2° infection
   c. Risk factors include:
      i. Allergic rhinitis (20%)
      ii. Mucociliary problems (5%) – CF, Kartagener’s syndrome, Young’s syndrome
      iii. Immunocompromised (<1%) – inherited or acquired
      iv. Nasal polyposis – Samter’s triad (asthma, nasal polyps, aspirin sensitivity)

2. Clinical features – note that signs are minimal, and diagnosis is usually based on history
   a. Acute – fever, purulent secretions, nasal obstruction, headache and facial
tenderness, anosmia, cough, sore throat, otitis media with effusion (children)
b. Chronic – fever less common, cough more prevalent (particularly children), rhinorrhoea and post-nasal drip, headache and facial tenderness

c. Complications:
  i. Pott’s puffy tumour – extension of frontal sinusitis anteriorly into frontal bone (maxilla in infants) causing a distinct swelling → debridement, IV antibiotics
  ii. Orbital – periorbital/orbital cellulitis, inflammatory oedema, subperiosteal and orbital abscess, optical neuritis, osteomyelitis
  iii. Cranial – cavernous sinus thrombosis, meningitis, encephalitis, epidural/subdural/parenchymal abscess
  iv. Mucocele – more common in frontal sinus than ethmoid, may extend to orbit and anterior fossa gradually; treatment by permanent drainage to nose

3. Investigations:
   a. Plain X-ray may detect maxillary, frontal disease (not sphenoid or ethmoid)
   b. CT scan – not needed acutely but useful in chronic sinusitis (detects everything)
   c. Ancillary tests e.g. skin testing for Ig levels

4. Management – note that 40% resolve spontaneously
   a. Medical:
      i. Topical decongestants for 3/7 (note rebound congestion) to improve drainage
      ii. Antibiotics if severe – amoxicillin ± clavulanate, doxycycline, co-trimoxazole, or roxithromycin for 14/7 and/or 7/7 post symptoms (at least 3-4/52 if chronic)
      iii. Adjunctive therapy:
         1. Nasal glucocorticoid inhaler improves recurrent sinusitis
         2. Oral steroids (prednisone) for 10/7 useful for chronic sinusitis
         3. Antihistamines and oral decongestants of limited benefit, as these thicken secretions and decrease ciliary activity
   b. Surgical – endoscopic sinus surgery (cure rates up to 60% for chronic sinusitis)

Other causes of rhinitis:
1. Allergic rhinitis
   a. Common (10% of population condition presenting in childhood or adolescence with seasonal sneezing, rhinorrhoea, nasal congestion, and upper airway irritation
   b. Type I (IgE mediated) hypersensitivity reaction to aeroallergens inhaled via the nose
      i. Allergens processed by macrophages, dendritic and antigen-presenting cells
      ii. Antigens expressed with class II MHC molecules on APC surface
      iii. APC activate T-lymphocytes → cytokines → B lymphocytes produce IgE
      iv. Mast cell degranulation → histamine, prostaglandins, leukotrienes
   c. Examination:
      i. Nasal mucosa boggy, pale blue, clear discharge (± bacterial superinfection)
      ii. Sinus tenderness, conjunctivitis, post-nasal drip (simulates chronic cough)
   d. Management:
      i. Investigations – allergen identification with skin or radioallergosorbent testing
      ii. Conservative – allergen avoidance
      iii. Medical – topical steroids, oral or topical antihistamines, Na+ cromoglycate
      iv. Surgical – help nasal obstruction, allows access for steroid nasal sprays

2. Vasomotor rhinitis
   a. Symptom complex resembles allergic rhinitis but with non-specific stimuli and in the absence of any established allergic basis
   b. Mucosa tends to be red (c.f. pale mucosa in allergic rhinitis)
   c. Management may be difficult:
      i. Medical – topical anticholinergics for nasal drip
      ii. Surgical – may help symptoms of nasal obstruction

• Nasal Tumours

Benign nasal tumours:
1. Papillomas – most commonly inverted papilloma 2° to HPV
   a. Usually unilateral, arising from within the nose, ethmoid and maxillary sinus
   b. Present with epistaxis, nasal obstruction, local destruction – malignant potential
   c. CT scan required, treatment by surgical removal
2. Osteomas
   a. Bony tumours, usually in the sinuses
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3. Angiofibromas – only adolescent males
   a. Arise from posterior nose, grow rapidly and expansively
   b. Present with nasal obstruction, purulent nasal discharge and frequent epistaxis, obstruction of Eustachian tube, cranial nerve involvement
   c. CT, MRI, angiography (embolisation of feeding vessels), surgery/radiotherapy if large

4. Granulomas
   a. Present with obstruction, tissue destruction, crusting and bleeding
   b. Histology useful; treatment by chemotherapy or radiotherapy

Malignant nasal tumours (don’t forget the external nose) are rare with poor prognosis – sinuses may harbour large tumours asymptptomatically for a great deal of time. Most commonly these are squamous cell carcinomas, adenocarcinoma (wood dust exposure) and adenoid cystic carcinoma.

1. Clinical features:
   a. General – epiphora (tear overflow), lymphadenopathy (10%)
   b. Nasal – epistaxis, obstruction
   c. Ethmoidal – orbital signs and nasal symptoms
   d. Maxillary – facial swelling, loosing of teeth, proptosis, diploplia

2. Warning signs
   a. Facial swelling
   b. Diplopia or eye displacement
   c. Gum swelling or tooth loosening
   d. Unilateral bloody discharge
   e. Unexplained facial pain

Epistaxis and Nasal Trauma

Epistaxis is a haemorrhage from the nostril, nasal cavity or nasopharynx. It may be from the anterior nasal cavity (usually Little’s area on the septum above the posterior end of the nasal vestibule, or the anterior end of the inferior turbinate) or from the posterior nasal cavity or nasopharynx (usually under the posterior half of the inferior turbinate, or the roof of the nasal cavity).

1. Aetiology:
   a. Nasal:
      i. Trauma – nose blowing, sneezing, fracture, intubation, nose picking, surgery
      ii. Desiccation – cold air, dry heat, nasal sprays, septal deviation/perforation
      iii. Inflammation – allergic (rhinorrhea) or infective (sinusitis, URTI)
      iv. Septal disease – AIDS, cocaine, granulomatous disease, polychondritis
      v. Neoplasia – benign (angiofibroma or papilloma) or malignant
   b. Systemic:
      i. Coagulopathy – EtOH, liver disease, CRF, haemophilia and VWD, DM, DIC
      ii. Blood dyscrasia – leukaemia, multiple myeloma, thrombocytopaenia
      iii. Drugs – NSAIDs, aspirin, warfarin, heparin

2. Evaluation:
   a. Use topical anaesthetic (lidocaine with vasoconstrictor, topical cocaine)
   b. Visible bleed → anterior, not visible → posterior, successful pinch → septal

3. Management:
   a. General – head back with nasal pinching, ice on nasal area, topical vasoconstrictor
   b. Anterior epistaxis (most common, usually unilateral)
      i. Usually from Kiesselbach’s plexus, derived from anterior ethmoidal artery
      ii. Treat with cautersation, typically with silver nitrate sticks
   c. Posterior epistaxis (usually bilateral and/or nasopharyngeal bleeding)
      i. More difficult to control, may need posterior packing
      ii. Almost always associated with hypertension or coagulopathy

4. Severe intractable epistaxis
   a. Coagulopathy must be ruled out
   b. Otolaryngologist referral for nasal endoscopy to rule out tumour, Osler-Weber-Rendu
   c. Treatment – operative endoscopic cautery, operative ligation of internal maxillary
      and/or anterior ethmoid arteries, endovascular embolisation of vessels
Nasal foreign bodies are a common presenting complaint in children, possibly with unilateral purulent malodorous discharge if presenting subacutely. The foreign body should be removed if good light, anaesthesia, instruments and cooperation are available – otherwise general anaesthetic needed.

Nasal fracture:
1. **History** – force/direction of blow, premorbid shape, concomitant fractures (zygoma, facial sinuses, hypertelorism, telecanthus), neck pain or ↓ movement, domestic violence/abuse
2. **Assessment**
   a. Look for nasal deviation and septal haematoma (before swelling sets in)
   b. Oedema suggest underlying nasal fracture, review when swelling reduced
   c. Pain on palpation may indicate site of fracture – infraorbital rim should be palpated
3. **Radiology and classification:**
   a. Grade 1 (undeviated) – lateral glancing blow of moderate force, apparent deviation due to depression of bone fragment
   b. Grade 2 – pyramidal fracture → lateral displacement (horny dorsum remains intact), fracture lines parallel to dorsum, connected across the midline
   c. Grade 2 with greater frontal force → depresses the whole frontal segment (dorsum intact), associated ethmoid fracture (perpendicular plate)
   d. Grade 3 – pyramidal fracture with lateral displacement of fragments (dorsum intact), fracture lines parallel to dorsum, C-shaped fracture including ethmoid, vomer and posteroinferior portion of cartilaginous septum
4. **Management** – simple manipulation is the most cost-effective option as although open reduction improves outcomes, the nose is weakened in case of further injury
   a. Grade 1 – local anaesthetic, depressed fracture elevated
   b. Grade 2 – local anaesthetic, septoplasty may be required
   c. Grade 2 with greater frontal force – reduction difficult, rhinoplasty at a later date
   d. Grade 3 – correction and reconstruction of nasal septum
   e. Cartilaginous injuries – difficult to repair initially, surgery at a later date

### THROAT

#### Pharyngitis, Tonsillitis and Adenoiditis

**Waldeyer’s ring** of lymphoid tissue surrounds the entry points to the upper aerodigestive tract and traps and presents inhaled/ingested pathogens to the immune system. Unlike all other lymphoid tissue in the body, they do not have any afferent lymphoid connection. The ring is comprised of:
1. Nasopharyngeal tonsils (adenoids)
2. Palatine tonsils
3. Lingual tonsils
4. Lateral pharyngeal bands
5. Pharyngeal lymphoid islands

**Pharyngitis** is an infection (most commonly viral) of the pharynx and faucial pillars without evidence of involvement of the tonsils. Pharyngeal bands and the islands of lymphoid tissue on the posterior pharyngeal wall are most commonly affected.

**Tonsillitis:**
1. **Description** – common infection of the palatine tonsils, which are typically enlarged, reddened and covered with white spots or a membrane (especially in infectious mononucleosis).
   a. May be viral or bacterial (group A Streptococci, H. influenzae and Staphylococci)
   b. Lingual tonsillitis – lingual tonsils are primarily affected
2. **Pathogenesis:**
   a. Tonsils form in the 7th month of foetal life and have a folded surface and plentiful B cells. Note that their proximity to the Eustachian tube can → associated otitis media
   b. Recurrent URTI may lead to proliferation of immunocompetent cells → obstruction
   c. Chronic infection may lead to depletion of immunocompetent cells → colonisation
3. **Clinical features:**
   a. Acute tonsillitis – odynophagia, sore throat, earache, abdominal pain, post-URTI
      i. Fever, erythema of tonsils and/or pharynx, tender cervical lymph nodes
      ii. Tonsils – variable, often ‘prominent’ rather than enlarged, bulge with gagging

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b. Chronic tonsillitis – recurrent low grade sore throats, scarred tonsils, debris exuding from tonsil crypts, halitosis, tonsilloliths and general malaise

4. Management:
   a. Investigations – FBC looking for elevated WBC, infectious mononucleosis test
   b. Take a throat swab, then start on antibiotics (discontinue if swab is negative):
      i. Penicillin 7-10 days, erythromycin if allergic
      ii. Resistant disease can be treated with amoxicillin and clavulanic acid
   c. Adenoidectomy + tonsillectomy:
      i. Indications:
         1. Obstruction – stridor, cor pulmonale, OSA, dysphagia
         2. Recurrent acute tonsillitis or chronic tonsillitis (debris, halitosis)
         3. Quinsy or recurrent peritonsillar abscess (2 times)
         4. Tonsillar malignancy
         5. Recurrent otitis media with effusion (benefits unclear)
      ii. Complications – haemorrhage (early or late), airway obstruction (oedema, blood clot, dislodged ET tube), pulmonary oedema, nasopharyngeal stenosis

5. Complications:
   a. Peritonsillar abscess (Quinsy) – between tonsil capsule and constrictor muscles, presenting with pain, trismus, fetor oris, fever, dysphagia, drooling, abnormal speech
      i. Classic triad – muffled ‘hot potato’ voice, trismus and uvular shift
      ii. Complications – airway obstruction, rupture with aspiration, abscess with extension to deep neck spaces, haemorrhage (carotid artery rupture)
      iii. Treatment – transmucosal needle aspiration, incision and drainage, immediate tonsillectomy (debatable – severe cases), antibiotic coverage
   b. Recurrent tonsillitis in 20% of patients

Adenoiditis – difficult to diagnose as they are not readily visualised (involute at age 5)

1. Clinical features:
   a. Evidence of infection – pus, post-nasal drip, recurrent nasal infection/discharge
   b. Obstruction of posterior choanae of nose

2. Management:
   a. Investigations – endoscopy, lateral x-ray
   b. Indications for adenoidectomy:
      i. Airway obstruction
      ii. Recurrent infection with evidence of adenoid hypertrophy
      iii. Recurrent acute otitis media with associated nasal symptoms
      iv. Persistent otitis media with effusion (particularly with nasal symptoms

Other pharyngeal infections:

1. Retropharyngeal abscess – infection in the retropharyngeal space precipitated by an episode of pharyngitis or foreign body injury. Often difficult to diagnose.
   a. Lateral nasopharyngeal X-ray may show widening of the retropharyngeal space
   b. Treatment – incision and drainage (through the mouth)

2. Parapharyngeal abscess – infection that has tracked to the parapharyngeal space
   a. Easily delineated on CT examination
   b. Treatment – incision and drainage (through the neck)

3. Cervical abscess (differential diagnosis) – abscess due to suppuration in a lymph node, usually secondary to an episode of pharyngitis
   a. Ultrasound examination is the best means of diagnosis
   b. Treatment – incision and drainage through the neck

4. Oedematous uvula – 2° to trauma or infection, symptoms out of all proportions

Note that recurrent rhinitis is a very common presenting complaint. Causes include URTIs (very common in preschoolers), recurrent viral infection, allergic rhinitis, sinusitis (may be associated with CF), and gastroesophageal reflux.

• Voice Disorders

General principles – note that there are three primary laryngeal functions (airway, airway protection and voice), and disease may cause problems through destruction, obstruction or restriction

1. Laryngeal symptoms and signs

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1. Symptoms:
   a. Stridor, dyspnoea, dysphagia, neck swelling, pain
   b. Hoarseness, dysphonia
   c. Cough, haemoptysis

2. Common conditions:
   a. Papilloma (may recur), adenoma, chondroma, haemangioma, lipoma, neurofibroma
   b. Non-neoplastic disease – vocal fold polyp, retention cyst, contact ulcer, vocal fold nodules, chronic laryngitis, vocal fold paralysis, infections (TB, syphilis), functional dysphonia, muscle tension dysphonia, post-radiotherapy

3. Treatment overview:
   a. Benign tumours are usually removed at the time of biopsy (juvenile papillomas recur)
   b. Other conditions may be able to be treated conservatively with speech therapy, antibiotics, avoidance of aggravating factors

**Hoarseness** and alteration in voice are the common symptoms of laryngeal pathology (note that respiratory symptoms are more common in children). It is useful to consider diseases of as abnormalities of structure (inflammation, neoplasia, degenerative change) or abnormalities of function (neurological (central/peripheral), muscle tension, spasmodic).

1. Acute hoarseness (arising in minutes/hours/days)
   a. Causes – infection, trauma (voice abuse, vomiting, irritants, external trauma, allergy)
   b. Associated symptoms and signs – sore throat, dysphagia, general signs of infection
   c. Treatment is symptomatic – voice rest, inhaled bronchodilators, antibiotics, antihistamines, steroids

2. Chronic hoarseness (lasting >2 weeks)
   a. Aetiology – chronic laryngitis, contact ulcers, vocal cord polyps, laryngeal papillomata, infections (TB, syphilis), vocal cord paralysis, vocal fold nodules
      i. Early carcinoma of the vocal folds needs to be excluded
      ii. Vocal fold nodules are a local form of chronic laryngitis (2° to voice abuse) occurring at the junction of the anterior 1/3 and posterior 2/3 of the vocal folds
   b. Treatment – treat cause, retrain voice with speech therapy, remove nodules surgically

**Vocal fold paralysis** occurs with palsy of the motor nerves of the larynx – recall that these are the recurrent laryngeal nerve (left is more vulnerable) and the external branch of the superior laryngeal nerve. Damage ranges from neurapraxia → axonotmesis → neurotmesis.

1. Aetiology
   a. Malignant disease – lung, oesophagus, thyroid
   b. Surgical trauma – particularly post thyroidectomy
   c. Inflammation, trauma, carotid aneurysms, RA of the cricoarytenoid joints, neurological

2. History:
   a. Dyspnoea, stridor, poor breath control
   b. Cough, aspiration, reduced power
   c. Altered pitch, reduced clarity of voice

3. Examination:
   a. Cords may have characteristic positions but often do not fall into a specific category
   b. Laryngeal examination is important and may indicate the site of the lesion
   c. Vagal lesions leave the paralysed vocal cord more laterally than recurrent laryngeal

4. Investigations
   a. CXR, respiratory function tests, thyroid ultrasound and/or scan
   b. CT/MRI from base of skull to lower neck – nasopharynx, vagus nerve, carotid sheath, thyroid gland, lymph nodes
   c. Speech recordings, video laryngoscopy, endoscopy under GA occasionally

5. Treatment involves speech therapy, vocal fold injections, laryngeal framework surgery, arytenoidectomy, surgery to lateralise or medialise the vocal fold, cricopharyngeal myotomy, tracheostomy, partial or total laryngectomy

**Hoarseness persisting for more than four weeks need specialist otolaryngological referral for direct or indirect laryngeal examination to exclude malignancy.**

Note that a whole pile of different techniques are available for examination and evaluation of the upper airways (including flexible fiberoptic laryngoscopy, videostroboscopy, rigid laryngoscopy,
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computer voice analysis, electroglottography, electromyography, spectography, airflow analysis) and a team approach may be necessary, especially when dealing with professional voice users.

- **Airway Disorders**

**General principles:**

1. **Anatomy** – important differences compared to adult airways are:
   a. Neonates are obligate nasal breathers
   b. The larynx is higher and smaller than adults
      i. Epiglottis is curled in an omega shape → easier viewing of the vocal cords
      ii. Elevating the glottis to allows young children to feed and eat at the same time
   c. The narrowest site of the paediatric airway is at the level of the cricoid cartilage (glottis and vocal cords in adults)

2. **Symptoms:**
   a. Stridor – a musical sound occurring on respiration produced by airflow through a narrowed segment at or below the level of the larynx (e.g. croup)
   b. Stertor – a snoring, snuffly noise sound occurring on respiration produced by airflow through a narrowed airway above the level of the larynx (e.g. snoring)

3. **Signs of airway obstruction** – note that SpO2 drops are a late sign
   a. Stridor – inspiratory (tracheal), expiratory (LRT) or biphasic (tracheal)
   b. Increased respiratory effort, tachypnoea
   c. Chest retraction, epigastric indrawing (Harrison’s sulcus)
   d. Tracheal tug, intercostal recession

**Infections:**

1. **Epiglottitis** – rare, affects children aged 3-5 years but also occasionally adults
   a. Caused by *Haemophilus influenza B* infection (lowering incidence 2° to immunisation)
   b. Rapidly progressive (6-12 hours) with
      i. Supraglottic obstruction → stridor and severe obstruction
      ii. Painful sore throat and drooling
   c. Lateral neck X-ray will show a swollen epiglottis (‘thumb print sign’)
   d. Treatment by ET intubation, IV amoxycillin plus clavulanic acid

2. **Croup** – common, affects 1-3 year olds almost exclusively
   a. Usually viral (adenovirus, RSV and parainfluenzae)
   b. Slow onset (6-12 hours) with:
      i. Subglottic obstruction → inspiratory stridor
      ii. Seal-like barking cough
   c. AP airway x-ray will show subglottal narrowing (‘Steeple sign’), also on lateral
   d. Treatment – steam inhalation, supportive treatment, intubation rarely required

3. **Respiratory papillomatosis** – laryngeal, pharyngeal and oesophageal (HPV 11, 6)
   a. May present with airways obstruction or hoarseness
   b. Treatment with CO2 laser, micro shaver, cidoforir

**Congenital anomalies:**

1. **Choanal atresia** – failure of the posterior choanae to canalise in birth (1:10,000)
   a. CHARGE association (coloboma, heart malformation, atresia choanae, retarded growth and development, genital hypoplasia, ear anomalies)
   b. May be unilateral or bilateral, bony or membranous
   c. Diagnosis by mist test, passing a size 14F catheter through nose or CT scan

2. **Laryngomalacia** – immature floppy cartilage/soft tissue, commonest cause of stridor (75%)
   a. Presents a few days to a week after a birth with rhythmic/musical inspiratory stridor (expiratory if below vocal cords) worse when awake, feeding or crying
   b. Signs – stridor, tachypnoea, costal indrawing, tracheal tug
   c. Generally resolves by 14-18 months or → endoscopic laser wedge resection
   d. Apnoeas, failure to thrive, poor feeding, aspiration are indications for surgery

3. **Tracheomalacia** – floppy or weak tracheal cartilage → reduced cartilage : trachealis muscle ratio (normally 4:1)
   a. May be primary or secondary (e.g. mediastinal mass effect), presenting with stridor, wheeze, apnoeas and chest infections – diagnosis by endoscopy
   b. Treatment is required (patients do not grow out of the diseases) – treat other associated disease, CPAP, tracheostomy, stents, resection

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4. **Others** — midline vocal cord paralysis, webs, clefts

**Airway trauma:**

1. **Subglottic stenosis** — caused by intubation trauma at the level of the cricoid (rarely due to a congenitally narrowed cricoid)
   a. Incidence is reducing due to better neonatal care and CPAP
   b. Presents with stridor, dyspnea, tachypnea, exercise intolerance and failure to thrive
   c. Diagnosis by endoscopy — laryngoscopy and bronchoscopy

2. **Airway foreign bodies** — common in 2-5 year olds (history essential /+/- witness)
   a. Commonest object is a peanut, usually lodged in the right main bronchus
   b. Presents acutely with cough, wheeze and stridor or more chronically with asthma or pneumonia unresponsive to treatment
   c. Inspiratory/expiratory PA CXR should be done (not all foreign bodies are opaque!)
      i. Inspiratory film — usually normal
      ii. Expiratory film — mediastinal shift away from the side of the foreign body due to air trapping (ball-valve effect)
   d. Bronchoscopy is diagnostic and therapeutic; if removal is impossible → lobectomy

3. **Laryngeal trauma**
   a. Rare, as the mandible protects the larynx
   b. Presents with neck injury, bruising, deformity of laryngeal contour, hoarseness, stridor, pain, dysphagia
   c. Should be taken seriously — admit, observe and refer

**Airway-related procedures:**

1. **Tracheotomy (-ostomy)**
   a. Connection from trachea to the skin of the neck, kept open with a tracheotomy tube
   b. Usually a temporary measure — except in post-laryngectomy a end-opening tracheotomy is performed as a permanent measure
   c. Indications include prolonged ventilation, tracheal/laryngeal stenosis, compressive neck masses, OSA, chronic aspiration, facilitating pulmonary management
   d. Complications — airway obstruction (dislodgement of tube, crusting, mucosal swelling, granulations), bleeding (carotid, thyroid), perforation (oesophageal), stenosis and tracheomalacia, dysphagia and aspiration, infection

2. **Cricothyrotomy** — emergency tracheotomy
   a. Feel for cricothyroid membrane below the Adam’s apple
   b. Finger on the spot → knife into trachea and insert a hollow object
   c. Useful to use a scalpel blade — dive straight down, then twist 90° to open up

3. **Heimlich manoeuvre**
   a. Stand behind the person with your clasped hands around them and pull sharply up into the upper abdomen, immediately below the sternum
   b. Theory is forcing air up the trachea, dislodging the obstruction from below

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**Snoring and Obstructive Sleep Apnoea**

**Snoring** and **obstructive sleep apnoea** are two ends of a pathological spectrum. Uncomplicated habitual snoring is common (40% of males), while 2% of the adult male population have OSA.

1. **Definitions:**
   a. Snoring is an audible inspiratory pharyngeal vibration produced during sleep
   b. Apnoea is cessation of airflow at the nostrils and mouth for at least 10 seconds
   c. Hypopnoea is a fall in average tidal volume by more than 50%
   d. OSA — frequent apnoeas and hypopnoeas (>10s or more than 15 events per hour) during sleep with or without PO₂ desaturations

2. **Pathophysiology**
   a. Narrow oropharyngeal airway
      i. Elongated palate and uvula
      ii. Large tonsillar pillars with redundant lateral pharyngeal wall mucosa
      iii. Retrognathism and macroglossia may be present
      iv. Nasal abnormalities are common
   b. Hypotonia of the airway → collapse of musculature
      i. Pharyngeal muscles relax and airway loses tone with sleep
      ii. May → turbulent flow → vibration of uvula, palate, posterior faucial pillars
c. OSA if airway completely collapses → hypoventilation, pulmonary and systemic hypertension, ECG abnormalities, sudden death.

3. OSA risk factors – male, post-menopause, obesity, adenotonsillar hypertrophy, retrognathism, nasal obstruction, alcohol ingestion, macroglossia, hypothyroidism, acromegaly

Clinical approach:
1. History – best taken with the sleeping partner in attendance, tape recording may be useful
   a. Consistency of snoring, every night in every position, periods of apnoea, sleep quality
   b. Ask about restlessness, inappropriate daytime snoring and somnolence
   c. May also present with headaches, impotence, nasal obstruction, weight gain
   d. Aggravating factors – EtOH, hypnotics, tranquilisers, narcotics, antihistamines
   e. Exclude – myxoedema, acromegaly, adrenocortical tumours
2. Examination:
   a. Oedematous uvula, elongated soft palate, redundant pharyngeal mucosa
   b. Particular attention to the nasal airway
   c. Polycythaemia and heart failure are rare
   d. Investigations – FBC, glucose, TFTs, ECG, CXR
3. Management:
   a. General – weight loss, dietary advice, stop smoking, avoid aggravating factors
   b. Refer if medical management fails, significant nasal obstruction or OSA
   c. Polysomnography may be used to grade severity and measure benefits of therapy
   d. Treatment – nasal surgery, CPAP, uvulopalatopharyngoplasty, tracheostomy

Swallowing Disorders

Physiology of swallowing:
1. Oral preparatory – voluntary (XII, VII, V)
   a. Lips close
   b. Labial and buccal muscles tense
   c. Grinding of teeth
   d. Movement of tongue
   e. Bulging forward of soft palate
2. Oral stage – voluntary (XII, IX)
   a. Tongue moves up → squeezes food against hard palate
   b. Food comes into contact with anterior faucial pillars
3. Pharyngeal stage – involuntary (IX, X)
   a. Soft palate closes, blocking nose and nasopharynx
   b. Elevation and closure of the larynx
   c. Peristalsis of pharynx
   d. Cricopharyngeus relaxes
4. Oesophageal stage
   a. Peristalsis

Gastroesophageal reflux is abnormal propulsion of stomach contents in a retrograde fashion into the oesophagus.
1. Pathogenesis – direct and indirect factors:
   a. Direct – loss of LOS tone, hiatal hernia, diet, alcohol, tobacco, medication
   b. Indirect – abnormal motility (neuromuscular, alcohol), delayed gastric emptying, high abdominal pressure, gastric hypersecretion (stress, tobacco, alcohol, medication)
2. Presentation – heartburn, pain, dysphagia, odynophagia, regurgitation, burping, globus, chronic sore throat, hoarseness, indigestion
   a. 50-60% don’t have classic symptoms (heartburn, sore throat, dysphagia)
   b. Others include laryngitis, chronic pharyngitis, nodules, polyps, globus, sinusitis, OME
3. Investigation – dual probe 24hr pH monitor, barium swallow with videofluoroscopy, oesophageal manometry, pharyngo-oesophagoscopy (observe, biopsy, dilate stricture)
4. Treatment – diet, coffee, alcohol, smoking, weight loss, medication (antacids, H2 receptor antagonists, proton pump inhibitors, gastrokinetic agents), surgery (fundoplication)

Oesophageal foreign body – common sites are cricopharyngeus and cardiac border (may be difficult to assess if it is still impacted). Corrosive foreign bodies need urgent attention.
1. Symptoms – often during or following a meal, dysphagia, odynophagia, drooling
a. Often able to localise the site with localised tenderness to palpation
   b. Inhaled foreign bodies may have stridor and respiratory distress
2. Investigations – CXR, soft tissue lateral X-ray of neck, flexible nasopharyngoscopy
3. Treatment – if suspicious or unsure → endoscopy (rigid or flexible)

Other swallowing-related problems include cricopharyngeal spasm, dribbling/drooling, velopharyngeal insufficiency, incoordinate swallowing and globus pharyngeus. Hands up who cares?