Deafness and Tinnitus

Ear symptoms
Hearing loss
Otalgia
Tinnitus
Vertigo
Otorrhoea

Deafness
Causes:
- Conductive:
  - External ear - atresia, meatal obstruction e.g. wax, foreign body, infection.
  - Middle ear – congenital anomaly, otitis media, cholesteatoma, otosclerosis, neoplasia.
- Sensory neural:
  - Cochlear – congenital, presbycusis, labyrinthitis, otosclerosis, Meniere’s disease.
  - Retro-cochlear – psychogenic, meningitis, MS, acoustic neuroma.
Conductive deafness – sound doesn’t reach cochlea, problems with middle or outer ear.

Pure tone audiograms
Subjective test of hearing.
Documents level at which patient can hear sounds of different frequencies – normal is levels better than 25dB for 4 frequencies.
Can test air or bone conduction (mask other ear).
Symbols:
- Right ear 0
- Left ear X
- Bone conduction right ear [
- Bone conduction left ear ]

Tympanometry
Measure compliance of ear drum.
Calculate middle ear pressure (sound is transmitted best when pressures are equal).
Normal graph has a peak.
Fluid in middle ear gives flat line.
Negative middle ear pressure shifts peak to left.

Can show stapedius reflex – dampens tympanic membrane with large sounds. This helps diagnose facial nerve lesions.

Objective tests
Electric response audiometry – electrodes pick up response in CNS pathway.
Otoacoustic emissions – hair cells in cochlea produce sounds as part of transducing sound energy. These can be measured using a small microphone and averaging computer. This gives an indication of cochlear function to screen hearing in young children.
Startle response – babies.
Tuning fork tests
Use 512 Hz fork.
Rinne’s test:
− Compares bone and air conduction.
− Hold tuning fork in front of air then on mastoid process.
− Air conduction should be better (Rinne positive).
− Better bone conduction implies conductive deafness.
− False negative is when sound is heard by bone conduction in other ear (sensorineural deafness).
Weber’s test:
− Hold tuning fork on forehead.
− Normal is sound hear equally on both ears.
− With conductive deafness localises to affected ear.
− With sensorineural deafness localises to other ear.

Tinnitus
Related to hearing loss of any cause but usually sensorineural.
Can be extrinsic (e.g. insects, vascular causes) or intrinsic (drugs, labyrinthitis, presycusis, Menieres disease, noise induced, otosclerosis, acoustic neuroma, epilepsy).
If pulsatile suspect a vascular tumour close to ear (glomus tumour of paraganglionic cells of nerves around jugular bulb which may extend into middle ear).
If unilateral must be investigated as may be acoustic neuroma.

Generally manage by reassurance. Can also use masking device, avoid aspirin and alcohol.

Choleastoma
Defect in migration of epithelium leads to sac of keratinising squamous epithelium.
Usually in attic or epitympanic part of middle ear. Ear drum is retracted and migrating epithelium accumulates and becomes infected. Can erode bone and therefore damage nearby structures.
Causes chronic, foul smelling discharge, conductive hearing loss, attic retraction, attic aural polyp and complications (facial palsy, vertigo, intracranial sepsis).
Rarely congenital, mostly acquired.
Treat by surgical removal.

Glue ear
Sterile effusion in middle ear (also secretory otitis media).
Poor ventilation of middle ear leads to accumulation of thick sticky effusion, which causes hearing loss and can predispose to infection.
Can be due to – acute otitis media, Eustachian tube dysfunction, nasopharyngeal tumour.
Very common in children but normally resolves spontaneously. If not treat by grommet insertion – allow middle ear ventilation.
Appearance on otoscopy – dullness, radial blood vessels, more horizontal malleus handle, absent light reflex.
Otosclerosis
Disease of bony labyrinth – hard compact bone is replaced by spongy bone. Usually have a family history.
Causes hearing loss due to:
- Toxin production affecting cochlear.
- Fixation of stapes foot plate and conductive hearing loss.
Consider if progressive conductive hearing loss with normal ear drum, often starts in pregnancy.
Can replace fixed stapes with piston if severe.

Sensorineural deafness
Inner ear damage due to:
- Congenital damage due to anoxia.
- Degenerative – presbycusis (hearing loss at higher frequencies), Menieres disease (episodic hearing loss especially low frequencies, tinnitus and vertigo).
- Labyrinthitis – vertigo.
- Acoustic trauma – acute or chronic (noise induced hearing loss is generally in a narrow frequency range).
- Drug ototoxicity e.g. aminoglycosides, frusemide.

Ear wax
Can block EAM causing conductive deafness.
Wax softening agents e.g. oil or sodium bicarbonate may help.
Syringing can remove wax if no perforations, grommets or ear infection. Involves washing out wax with warm water – squirt back along top of ear canal.

Otoscopy
Handle of malleus, pars flaccida, pars tensa, light reflex in anteroinferior quadrant (as concave shaped).
Membrane should normally be pearly grey and slightly translucent.
Otitis media – hyperaemia of eardrum, then loss dull and may bulge.
Glue ear – concave dull drum, superficial radial vessels
Ear Infections

Anatomy of the ear

External ear:
- Pinna or auricle and external auditory meatus.
- Transmits sound to tympanic membrane.
- Auricle develops from 6 nodules, consists of elastic cartilage covered in skin.
- Auricle has named folds = Darwin’s tubercle, antihelical fold, helical fold, tragus, antitragus, lobule.
- External auditory meatus – outer third is cartilaginous with sebaceous and wax (not mucus) glands and hairs, inner two thirds are bony.
- Complex nerve supply – auricolotemporal branch of trigeminal (anterior), greater auricular (posterior), also glossopharyngeal and vagus (so examination of ear can cause coughing). This means that otalgia may be due to referred pain from many structures (TMJ, parotid, sinuses, teeth, tongue, tonsil, C spine, pharynx, larynx), especially carcinoma of pyriform fossa.
- Skin is migratory and travels outwards so ears are self cleaning.
- Wax is bacteriostatic.

Middle ear:
- Air filled space in petrous temporal bone.
- Transmit and amplify (tympanic membrane is larger than oval window, ossicles act as levers) sound to fluid in inner air.
- Connected with mastoid air cells (via antrum).
- Eustachian tube communicates with nasopharynx (part bony, part cartilaginous). Needed to allow oxygen to mucosa, equalise air pressures so tympanic membrane can vibrate and drain secretions.
- Tympanic membrane forms lateral border – external squamous epithelium, middle fibrous, inner layer continuous with middle ear mucosa.
- Epitympanum above and hypotypanum below.
- 3 ossicles – malleus, incus, stapes (footplate on oval window so sets up fluid pressure wave to stimulate cochlea).
- Medial wall opens to inner ear via round and oval windows, basal turn of cochlear forms a bulge (promontory).
- Facial nerve runs across medial wall in bony channel.

Inner ear:
- Membranous and bony labyrinth in petrous part of temporal bone.
- Membranous labyrinth, filled with endolymph – cochlea, vestibular system (saccule, utricle, semicircular canals).
- Surrounded by perilymph (like CSF).
- Hear by organ of Corti on basial membrane or cochlea. Hair cells are linked to tectorial membrane.
Otitis externa
Common. Generalised inflammation of skin of EAM – tender, swollen, narrowed EAM.
Causes –
  - eczema, psoriasis, trauma, local infection (pseudomona, staphylococcus, candida, viral).
  - Generally a combination e.g. water causes eczematous response, itching leads to scratching causing local trauma, infection can enter etc.
Symptoms –
  - otorrhoea, hearing loss, pain, itching.
External ear canal does not have mucous glands so if mucinous discharge must have perforation and underlying middle ear problem.
Treatment –
  - Aural toilet (suction and microscope or dry mopping)
  - Local medication (antibiotic, steroid, antifuncal, glycerin and ichthammol) as drops or wick.
  - May lead to perichondritis or facial cellulitis.
Malignant otitis externa - severe pseudomonas infection which spreads to bone causing osteomyelitis of skull base. Causes severe pain and cranial nerve palsies.

Otitis media
Inflammation of the middle ear leads to formation of an effusion – either sterile (glue ear) or suppurative (infection).
Pressure build up stretches eardrum and causes pain. The drum then perforates causing otorrhoea.

Acute otitis media:
  - Usually children following URTI, which spreads via Eustachian tube.
  - Symptoms – hearing loss, pain, otorrhoea, pyrexia, systemic upset.
  - Can become a recurring problem if glue becomes reinfected. Grommets also drainage.
  - Typically H inf and Strep pneumoniae.
  - Treat with antibiotics e.g. amoxicillin and analgesia. If perforation keep ear dry. Nasal decongestants can speed recovery. Antibiotic and steroid ear drops may help.
  - Usually resolve but can lead to residual perforation, tympanosclerosis, ossicular adhesion, spread elsewhere (mastoiditis, facial nerve palsy, labyrinthitis, petrositis, intracranial abscess or thrombosis).

Chronic suppurative otitis media:
  - Repeated acute otitis media can damage tympanic membrane causing non-healing perforation.
  - This can infect mastoid system producing mucopus which leaks through perforation.
  - Leads to hearing loss, otorrhoea (typically intermittent).
  - Can progress to cholesteatoma – especially if marginal pars tensa or attic perforation.
**Dizziness**

**Vertigo**

Need balance and vestibular system to keep upright and maintain visual fixation despite movement of head.

Maintain balance by proprioception, vision, labyrinth (need at least 2 of these). Conflicting information from these (i.e. if one is not working) leads to dizziness. If there is a problem with the inner ear then turning the head causes dizziness – normally should get equal and opposite response in each ear, this does not occur if one ear is not functioning.

Vertigo is the abnormal sensation of movement. When due to vestibular disease is usually rotatory. It is often accompanied by nausea, vomiting or pallor.

Need to distinguish from unsteadiness and fainted, which are not true vertigo. These should be suspected if there is faintness, loss of consciousness, associated breathlessness, headache or if occur on exercise.

Causes:

- Peripheral – labyrinthitis, BPPV, Menieres, middle ear disease, drugs
- Central – vestibular neuronitis, tumours, MS, head injury, vascular occlusion, migraine (recurrent episodes with headaches), alcohol.
- Other causes of balance disturbance that are not true vertigo – cardiac insufficiency, C spine disorders, neurological disorders, anaemia, epilepsy, presbystasis (loss of balance when walking, in old age, probably ischaemic in origin).

Typically peripheral causes give more severe vertigo which is sudden onset with hearing loss, tinnitus, nausea, vomiting and nystagmus. These are less common in central vertigo.

Examination – CN function, cerebellar function, Romberg test, (if positive worse when eyes shut, implies impaired vestibular or joint position function), assess nystagmus.

Manage by treating symptoms:

- Anti-emetic e.g. prochlorperizine
- Vestibular sedative e.g. cinnarizine

**Nystagmus**

Involuntary rhythmic to and fro movement of eyes (clinical sign of vestibular problem).

Also get reduced caloric response (induce nystagmus by irrigating ear canal with warm or cool water).

Direction:

- Vestibular disease is horizontal or rotatory.
- Vertical nystagmus is due to central lesion.
- Destroyed labyrinth function – away from affected ear.
- Irritated labyrinth – towards affected ear.

Degrees:

- Indicate acuteness.
- 3rd – when looking in all directions, sudden total vestibular failure.
- 2nd – when looking straight and in direction of nystagmus, after a few days.
- 1st – only when looking in direction of nystagmus, after few weeks.
- Disappears over months due to CNS compensation.
- Worsen by removing optic input.
Vestibular neuronitis
8th cranial nerve connects in brainstem with auditory and vestibular nuclei. Inflammation of vestibular portion causes vertigo. Usually due to a viral infection or drug toxicity.

Labyrinthitis
Acute inflammation of the inner ear, usually following a simple URTI. Disabling vertigo lasting days or weeks, with residual vertigo for months afterwards. If severe can cause hearing loss and vestibular destruction. Treat with antibiotics, vestibular sedatives and rest. Most will be better within 6 months due to compensation in the brain (central plasticity). Hair cells do not recover.

Menieres disease
Hearing loss, tinnitus and vertigo. Very acute onset disabling vertigo, with nystagmus and sometimes nausea and vomiting, often preceded by feeling of fullness. Probably due to distension of membranous labyrinth and excessive accumulation of endolymph. Treatment – symptomatic treatment of vertigo (bed rest, vestibular sedatives, anti-emetics), operative decompression of endolymph, labyrinthectomy, installation of otoxic drugs.

Benign paroxysmal positional vertigo
Episodic vertigo which occurs when the head is moved in certain positions, classically when turning in bed or looking up. Attacks are sudden onset and usually last for a few minutes. No otological symptoms. Thought to be due to otoconia, which trigger hair cells. Diagnostic test is Hallpike manoeuvre – patient quickly lies flat with head turned. If positive get symptoms and geotropic fatigueable nystagmus which disappears in about 30secs (rotatory towards affected ear). Treat by reassurance and positioning manoeuvres.

Presbycusis
Degenerative disorder in old age. Mostly hearing loss in both ears, atrophy of labyrinth. Associate with presbystasis, which presents as unsteadiness in the elderly, worse on movement. This does not need treatment.

Acoustic neuroma
Schwannomas of the vestibular division of the 8th cranial nerve. Slow expansion compresses nerve giving unilateral otological symptoms. Diagnose by MRI. Treat by surgical excision or watch and wait.

Acute labyrinthine failure
Cause – idiopathic, local occlusion, autoimmune. Present with sudden deafness and severe vertigo. Treat vertigo symptomatically, plasma expanders and carbogen to improve labyrinth blood supply, steroids and cyclophosphamide if possible autoimmune. Generally vertigo gradually improves but hearing loss often does not.
Facial Nerve Dysfunction

Course and function of facial nerve

Function:
- Motor nerve to face – muscles of facial expression.
- Associated with nervus intermedius – secretomotor fibres to salivary glands (except parotid).
- Branch to stapedius – loss causes hyperacusis.
- Carries taste fibres to anterior two thirds of tongue in chorda tympani.

Course:
- Leaves nucleus between pons and medulla.
- Goes through internal auditory meatus (with 8th nerve) and travels through petrous temporal bone into medial surface of middle ear.
- Forms geniculate ganglion and gives of greater petrosal nerve (lacrimal gland).
- Gives of branch to stapedius.
- 2 turns (genu). Gives of chorda tympani which passes on medial surface of tympanic membrane.
- Travels through mastoid to stylomastoid foramen and into parotid.

Looking at which functions are lost can show site of lesion.

Symptoms of a facial nerve palsy

Obvious deformity.
Loss of facial movements.
Dribbling.
Loss of stapedius reflex.
Decreased submandibular salivary production.
Loss of lacrimation (Schirmer’s test).

Examination of facial nerve and associated structures

Document degree of weakness.

Forehead sparing? – upper or lower motor neurone.
Other neurological deficits – especially 8th to exclude systemic neurological disorder or intracranial neoplasm.
Examine ear – middle ear disease.
Vesicles in ear – Ramsay Hunt syndrome.
Parotid – tumours
Can patient close eye? – if not at risk of corneal ulceration.
Management of facial nerve palsy

Protect eye - artificial tears, dark glasses, tape during sleep, tarsorrhaphy. Hooks and cheek plumpers to improve appearance.

Bell’s palsy:
- Viral infection, usually sudden onset.
- Swelling causes pressure and dysfunction.
- High dose oral steroids if present early.
- Generally resolves completely.

Due to middle ear infection:
- Treat infection – antibiotics, decongestants, sometimes myringotomy.
- Exclude cholesteatoma.

Ramsay Hunt syndrome:
- Herpes zoster infection.
- Palsy associated with pain and vesicles in the ear.
- May be associated with vertigo and deafness.
- Treat with acyclovir.
- Facial weakness often does not recover.
Nasal and Paranasal Sinus Problems and Snoring

Anatomy
The nose is needed to warm and humidify inspired air and collect water from expired air. Hairs remove particles from the air.

External nose:
- Upper one third is bone, attached to frontal bone and maxilla.
- Lower two thirds are cartilage – upper and lower (alar) lateral cartilages.
- The columnella is the skin connecting the upper lip to the nasal tip, normally connected to the nasal septum.

Nasal septum:
- Thin flat bony sheets posteriorly – vomer, perpendicular plate of ethmoid.
- Cartilage anteriorly.
- May be deviated and cause obstruction.
- Rich blood supply – 4 arteries anastamose in Little’s area – common site for nose bleeds.

Lateral nasal wall:
- Lateral to the nose are the orbit, maxillary and ethmoid sinuses.
- Lateral nasal wall has 3 ridges called the turbinates – bone covered with vascular mucoperiostium.
- Space under the turbinates is called a meatus.
- Frontonasal duct and maxillary sinus open under middle turbinate. Nasolacrimal duct opens under inferior turbinate. Sphenoid sinus opens on posterior wall between superior and middle turbinate.
- Inferior turbinates can well and shrink to regulate air flow.

Post nasal space:
- Posterior end of nasal cavity has 2 oval spaces – choanae – leading to post nasal space.
- Eustachian tube opens into this.

Sinuses:
- Frontal, maxillary, ethmoid, sphenoid.
  - Anterior group drain to middle meatus – maxillary, frontal, anterior ethmoidal.
  - Posterior group drain to superior meatus – posterior ethmoids, sphenoids (spheno-ethmoidal recess).
- Air-filled outpouchings of the nasal cavity which invaginate bones of the skull. Lined by ciliated epithelium.
- Needed to reduce weight of skull, voice formation, protect eye and brain, separate nose and brain.

History
Most nasal conditions (blocked and runny nose) are due to either structural abnormality or inflammation.
Structural – long history, constant, asymmetrical, previous trauma, snoring.
Inflammation of lining – sneezing, itch, hay fever, asthma, bilateral, rhinorrhea, postnasal drip.
In a child unilateral discharge may be due to a foreign body.
Examination of nose
Shape and size.
Test airway – occlude each nostril in turn and ask patient to sniff, looking for collapse of soft tissues.
Elevate nasal tip and examine vestibule.
Using thudicums speculum – examine nasal cavity – septum, floor, lateral wall.
Nasal mucosa – colour, surface, hydration.
Postnasal space – mirror or fibreoptic endoscope.
Examine ears.

Rhinitis
Inflammation of the nasal lining.
Symptoms – nasal congestion, rhinorrhoea, postnasal drip, sneezing, irritation, facial pain, loss of sense of smell.
Long standing rhinitis can cause turbinate hypertrophy, which may need surgery.
Often multifactorial – rhinitis leads to mucosal swelling with obstructs sinuses and predisposes to infection.

Acute infective rhinitis:
− Common cold – rhinovirus, coronavirus, adenovirus, RSV. Can get Strep of H. inf infection after giving cheek pain and pyrexia.
− Can lead to middle ear effusion, sinusitis, peri-orbital cellulitis, osteomyelitis, intracranial infection.

Allergic rhinitis:
− Nasal lining becomes sensitive to particles and has hypersensitivity IgE mediated reaction.
− Allergens – pollen, house dust, animal dander, feathers, eggs, milk.
− Seasonal allergic rhinitis is hayfever.
− Avoid allergen, drug therapy e.g. steroids, antihistamine, sodium cromoglycate (mast cell stabilisers), desensitising immunotherapy.

Vasomotor rhinitis:
− Similar to allergic rhinitis but no allergen found.

Rhinitis medicamentosa – use decongestant for too long, when stop get obstruction.
Sinusitis
Symptoms:
- Facial pain (only if acute exacerbation), nasal obstruction, anosmia, cachosmia, halitosis.
- Acute – systemic upset, rhinorrhoea with pus.
- Chronic – postnasal drip, muzzy head.

Predisposed by structural nasal defects which block ostia of sinuses and mucopus which depresses cilia activity.

Acute sinusitis:
- Inflammation of lining of sinuses.
- Usually viral – reduce ciliary function, stagnant secretions are secondarily infected.
- Treatment – reduce inflammation (decongestants, antibiotics), analgesia, aspiration and washout.
- Complications – chronic sinusitis, cellulitis, osteomyelitis, meningitis, abscess, mucocoele (collection of sterile mucus in obstructed sinus).
- Frontal sinusitis – severe frontal headache worse on bending, can easily to orbit and cause blindness or abscess.
- Treat with amoxicillin and topical decongestant.

Chronic sinusitis:
- Longstanding inflammation induces changes in the nose which further narrows sinus openings.
- Chronic production of green sputum, nasal obstruction.
- Medical treatment to treat cause, surgery to improve drainage – flushing, enlarge opening, new opening. Needs longer course of co-amoxiclav.

Structural nasal defects
Foreign body – especially children. Causes inflammatory response and foul discharge, with unilateral symptoms. If nasal secretions solidify around object forms rhinolith (obstruction and epistaxis).


Septal perforation – trauma, avascular necrosis, inflammation, tumours. Turbulent flow can lead to sense of obstruction, whistling, crusting and epistaxis.

Septal haematoma – septum swells and causes obstruction. Leads to avascular necrosis and collapse of the nose.

In children can get obstruction due to adenoidal hypertrophy. This spontaneously improves at about 7 years.

Systemic disorders affecting the nose
Primary defect in mucus – e.g. CF
Primary ciliary dyskinesia
Immunological
AIDS
Granulomatous disease – Wegner’s, sarcoidosis
Obstructive sleep apnoea
The adenoid is a collection of loose, non-encapsulated lymphoid tissue at the back of the postnasal space attached to the posterior wall of the nasopharynx. Enlarged adenoid – nasal obstruction (nasal voice, mouth breathing, runny nose), infection, snoring, Eustachian tube obstruction and glue ear, ascending infection to middle ear.

Snoring – the noise produced in sleep by vibration of soft tissues of the pharynx. Sleep apnoea:
- 30 or more episodes of cessation of breathing for at least 10 seconds over a nights sleep.
- Obstructive sleep apnoea is when this is due to upper airway collapse.
- Blood oxygen levels fall and waken patient.
- In long term causes pulmonary hypertension and RV strain leading to cor pulmonale.

Signs and symptoms:
- Typically overweight, large neck, overindulge in alcohol.
- Adenotonsillar hypertrophy in children.
- Snoring.
- Daytime sleepiness, poor concentration.

Investigation – sleep study, sleep nasoendoscopy.
Potential causes – adenoids, tonsils, nasal polyps, long plate, big tongue, obstruction in larynx.

Management:
- Lifestyle – lose weight, reduce alcohol intake, stop sedatives.
- Medical – reduce REM sleep, respiratory stimulants, CPAP.
- Surgery – adenotonsillectomy, remove obstruction e.g. polyps.

Nasal polyps
Can be benign or malignant.
Can be confused with turbinates but move on probing where turbinates do not.
Cause obstruction, anosmia and rhinorrhoea.
Treat by decongestants, antihistamines and steroids or surgery.

Tumours of the nose
Many different tumours. Mostly squamous cell, sometimes adenocarcinoma.
Common symptoms – unilateral obstruction, blood-stained rhinorrhoea, lump, epistaxis, chronic progressive pain, bone invasion giving cheek swelling, numbness, loose teeth, exopthalmus, CN palsies.
Treat by surgery and radiotherapy.
Voice Disorders

The larynx
Superiorly connects with pharynx, inferiorly with trachea.

Laryngeal cartilage skeleton –
- Thyroid
- Cricoid – only complete ring.
- Epiglottis
- Arytenoids – sit on cricoid, can move to change position and tension of folds.
- Cornicular

Hyoid bone
Membranes – thyrohyoid, cricothyroid.

Piriform fossa – in the hypopharynx, separated from the larynx by aryepiglottic folds, food passes through these in swallowing.
Follicular fossa – between the base of the tongue and epiglottis.

The larynx is divided into the:
- Supraglottis - up to hyoid bone, innervated by internal branch of sup laryngeal n, drains to cervical nodes
- Glottis - level of vocal cords, most common site for tumours, very little lymph drainage so good prognosis.
- Subglottis - down to cricoid cartilage, recurrent laryngeal n, drains to paratracheal and cervical nodes.

False cords – lie above true cords.
Vestibule – contains mucoous producing glands
True cords – attached to thyroid and arytenoids cartilages.
Vocalis muscle – under the vocal cord, part of thyroarytenoid muscle, supplied by recurrent laryngeal n.
Cricothyroid muscle – adjust tension of folds, only muscle on the outside of the larynx, supplied by sup laryngeal n external branch.
Posterior crico-arytenoid muscle – only muscle that abducts cords, thus allows airflow.
Reinke’s space – a ‘bag of thin fluid’ in the vocal cords which can move creating oscillations.

Functions of the larynx –
- protection of the lower airway - vocal cords, false cords, aryepiglottic folds
- cough generation
- voice production

Functions of the hypopharynx (piriform fossa and post-cricoid) –
- channel for food
- voice sounds
Voice production
Vocal cords produce a vibrating noise:
- Males – about 100 Hz.
- Females – about 200 Hz.
- Max range is 1000 Hz.

Production of voice (a complex sound wave) needs:
- Power - lungs
- Oscillator – vocal folds
- Resonator – vocal tract (pharynx, oral cavity, tongue, lips, teeth)

Voice problems
Diseases of the larynx present with either voice or airway problems. History – smoking, spirits, reflux, swallowing, breathing, constant/episodic/return to normality, otalgia.

Dysphonia – problem with vocal cords:
- Harsh voice – aperiodic vibration e.g. vocal cord tumour so don’t vibrate smoothly
- Breathy voice – turbulence, not using all the air passing through e.g. if vocal cords don’t come together in recurrent laryngeal n palsy.

Dysarthria – failure to control resonating chambers.
Problems with breathing e.g. COPD so can’t power vocal cords.
Dysphasia – linguistic problems, can be receptive or expressive.

A hoarse voice may be due to anything that impairs vocal cord motility:
- Neoplasia
- Inflammation (either infective or reflux)
- Neuromuscular (e.g. MS, Parkinson’s, nerve palsy, stroke – also affects swallowing causing aspiration)
- Behavioural (e.g. stress, hydration)

Patients with a hoarse voice for more than 3 wks should be referred.
Take history and visualise cord. Assume malignancy until proven otherwise – CXR and CT.

Inflammation
Causes a hoarse voice or aphonia. Cord movement is restricted but symmetrical.

Infective laryngitis is suggested by pain and URTI.
Inflammation may also be due to reflux.
If just larynx may also be – voice strain, irritants e.g. cigarette smoke.

Epiglottitis is acute and life-threatening as can rapidly cause airway obstruction especially in children. Present with sore throat, URTI, difficulty swallowing. Usually due to H. inf.

Croup is diffuse inflammation of the airways, usually viral. Can be life threatening like epiglottitis but has longer history.

Chronic laryngitis – vocal abuse, reflux, pollutants, smoking, spirits, post-nasal drip, chronic bronchitis. Hoarse voice with thickened erythematous vocal cords. As vocal cords have poor lymph drainage oedema is slow to resolve. Predisposes to carcinoma.
Muscle tension dysphonia:
- Commonest cause of voice problems.
- Due to imbalance of muscles.
- Diagnosed by looking at larynx.
- Treat by voice therapy.

Recurrent laryngeal nerve palsy:
- Can’t bring cords together, so get a weak voice, choking and bovine cough.
- Due to thyroidectomy, malignant disease (bronchus, thyroid, oesophagus).
- Most common on left due to long course under arch of aorta.
- Can sometimes compensate by encouraging other vocal cord to move over and meet lateralised cord. Can treat by cord medialisation e.g. by injecting Teflon or inserting silastic implant.
- If cord is fixed medially, airway is impaired so need to reposition cord laterally or excise cord.

Malignant disease:
Risk factors for neoplasia include:
- Smoking, aged over 40, stridor, neck lump, change in voice, otalgia, dysphagia.
This is seen as a thickened, irregular cord with leukoplakia.
Most common is squamous cell, almost always in smokers.
If on vocal cords, has good prognosis as generally presents early with hoarseness and poor lymph drainage.

Treatment – endoscopic removal, radiotherapy, radical surgical excision.
For small tumours radiotherapy is better to preserve voice.
Voice restoration after laryngectomy – oesophageal speech (either air swallowing or tracheo-oesophageal fistula at surgery), external vibrating device.

Benign disease:
- Reinke’s oedema – due to smoking, leads to thickened cords and a deeper voice.
- Nodules – give a husky voice e.g. if shout a lot, sing. Generally improved by speech therapy, otherwise need surgery. Form at area of maximal forceful glottic closure – junction of ant third and posterior 2 thirds.
- Polyp – appears after a cough or cold, must be removed by surgery.
- Papillomata – rare, due to HPV, surgical treatment but can recur and have malignant transformation.

Treatment of voice problems
- Vocal hygiene advice
- Voice therapy
- Phonosurgery
Upper Airway Obstruction and Tracheostomy

Symptoms and signs of upper airway obstruction
Noisy breathing.
Increased shortness of breath.
Change in voice.

Stertor = noise from orophraynx (snoring).
Stridor = noisy breathing, occurs with airway obstruction. Noise depends on which part of airway is narrowed causing noise production:
  - Laryngeal stridor is high pitched sound on inspiration.
  - Bronchi and bronchioles is expiratory wheeze e.g. asthma.

Stridor is more common in children due to:
  - Relatively and absolutely narrower airway
  - Less firm cartilage can collapse
  - Mucosa can swell
  - Upper airway obstruction increases the effort needed to breathe considerably.

Causes of upper airway obstruction
Infective – epiglottitis, croup
Neurological – vocal cord palsy
Congenital – laryngomalacia (excessively floppy), subglottic stenosis
Angioedema
Foreign body
Tumour
Trauma – bruising, surgical emphysema.
Smoke inhalation – delayed, smoke particles around nose.

Emergency management of acute upper airway obstruction
Estimate severity:
  - Look at patient – colour, intercostals recession, respiratory rate.
  - Can patient talk in full sentences, stridor or wheeze.
  - Respiratory rate, pyrexia, air entry, oxygen sats.

Endotracheal intubation – prevent aspiration. Generally better than tracheostomy as quicker, more readily available, makes subsequent tracheostomy easier, fewer complications.
Laryngotomy – hollow tube inserted into lumen of larynx.
Crico-thyroidotomy
Tracheostomy
Heliox – flows better so easier to breathe.
Nebulised racemic adrenaline
Systemic steroids
Tracheostomy

Indications:
- URT obstruction – either life-threatening or worsening.
- Protect tracheo-bronchial tree if aspiration of secretions e.g. MND, mini-tracheostomy for sputum retention.
- Respiratory failure needing prolonged ventilation.
- Laryngectomy (end tracheostomy).

Management – humidification, warming, filters, cleaning tubes, clear tracheal secretions, physiotherapy.

Early complications – tube displacement, blocked tube due to secretions or blood, pneumothorax, surgical emphysema, haemorrhage, air embolism.

Late complications – atelectasis, dysphagia, tracheomalacia, tracheo-cutaneous fistula, tracheo-oesophageal fistula, stenosis, scarring.

Croup

Common infective condition in children.
Oedema and vascular engorgement of airways especially subglottis. RTI leads to inspiratory stridor, general deterioration, cough.

Epiglottitis

Affects children and adults. More rapid onset.
Typically caused by Hib.
Erythema and oedema of epiglottis. Painful to swallow so drool. Better sitting up.
In a child, it is important to avoid precipitating airway obstruction. Don’t upset child e.g. by examining throat, inserting cannulae and causing them to cry. Need to get anaesthetist and ENT surgeon first.
Neck Masses and Head and Neck Cancer

Anatomy

Divided anatomically into anterior and posterior triangles by SCM.

Anterior triangle:
- Lower border of mandible, midline of neck, posterior border of SCM.
- Contains digastric, carotid, submental and muscular triangle.
- Hyoid bone, carotid bifurcation, thyroid, cricoid cartilage, trachea, submandibular gland.

Posterior triangle:
- Posterior border of SCM, anterior border of trapezius, middle part of clavicle.
- Contains nerves – accessory (leaves SCM halfway down d passes to trapezius), transverse cervical, supraclavicular, greater auricular, lesser occipital.
- Occipital and subclavian triangles, separated by omohyoid.

Simply, neck contains:
- Posterior muscular compartment with cervical spine and musculature. Contained by prevertebral fascia.
- Anterior visceral compartment, contains bundles of structures enclosed by fascia.
- Pretracheal fascia enclosing trachea and thyroid gland (so move together on swallowing).
- Carotid sheath containing carotid, internal jugular and vagus.
- Investing fascia around SCM and trapezius.
- Retropharyngeal space with oesophagus.

History

Duration – if less than 3 wks probably from infection.
Intercurrent illness
Pain or swelling – generally inflammation.
Relation to eating – sialadenitis/sialolithiasis.

Examination and investigations

Site, size, shape, surface, symmetrical, solid/cystic, tenderness, one lump or many, attachments, pulsatility.
Blood tests – FBC, monospot test.
Radiology – CXR, USS (solid v cystic), CT, MRI.
Cytology – FNAC
Endoscopy – may be metastasis from head or neck carcinoma.
Biopsy – generally excisional.

Staging:
N1 – single ipsilateral node less than 3cm.
N2 – single ipsilateral node less than 6cm, multiple ipsilateral nodes, contralateral nodes.
N3 – larger than 6cm.

Branchial cysts

Congenital and generally occur before 30 (present later than thyroglossal duct cysts).
Typically present with lump in neck in the middle third of SCM. May be painful if infected.
FNAC gives pus-like aspirate with cholesterol crystals.
Thyroglossal duct cysts
Congenital but generally presents in childhood or early adulthood.
Lie in midline between tongue base (foramen caecum) and thyroid.
Move when patient sticks out tongue.
Treat by surgical excision of whole tract including body of hyoid bone.

Thyroid masses
Goitre = thyroid swelling.
Thyroid masses move on swallowing.
Diffuse enlargement – iodine deficiency, pregnancy, Graves’.
Nodular enlargement – malignancy, TSH hypersecretion.

Thyroid masses can damage recurrent laryngeal nerves causing hoarse voice and poor breathy cough.
They can also invade causing dysphagia, hoarseness and shortness of breath.
Can extend retrosternally and compress mediastinum especially great veins.
Hot nodules (secreting thyroxine) are nearly always benign.

Thyroid tumours:
- Papillary – 50%, 40-50 yrs, multifocal, good prognosis, total thyroidectomy, neck dissection and radioactive iodine.
- Follicular – 25%, older, capsular, haematogenous spread, similar treatment.
- Medullary – 5%, MEN syndrome, CEA marker.
- Anaplastic – 20%, poor prognosis, pain, invasion symptoms.
- Benign adenoma – may cause thyrotoxicosis,
Salivary Gland Disorders

Anatomy
3 paired salivary glands – parotid, submandibular, sublingual - and a large number of tiny glands. Produce saliva to lubricate oral mucosa, begin food digestion and antibacterial action. Produce up to 1l per day.

Parotid gland:
- Largest
- Serous gland – watery saliva
- Between mastoid process and mandible, covered by thick parotid fascia which is painful when stretched.
- Drain via parotid duct (Stenson's) which opens opposite second upper molar tooth.
- Facial nerve passes through dividing into 5 branches, divides parotid into deep (20%) and superficial lobes. External carotid in deep lobe. Lymph nodes

Submandibular:
- Mixed serous and mucous.
- Bounded by mylohyoid and mandible.
- Large superficial lobe and deep lobe in floor of mouth.
- Duct (Wharton's) opens as papilla next to tongue frenulum.
- Hypoglossal (tongue movement) and lingual (taste) nerves associated with deep lobe, mandibular branch of facial nerve runs over gland. Facial artery passes posterosuperior aspect.

Sublingual:
- Smallest
- Mucous gland.
- Lies in floor of mouth in course of submandibular duct.
- Drains by many small ducts into floor of mouth or submandibular duct.

Minor glands:
- Beneath mucosa of upper aero-digestive tract.
- Named according to region.

Innervation:
- Reflex stimulation by smell, taste (especially acid and sugar) and psychic stimuli (thought of food, chewing).
- Both nerves pass through middle ear whilst going between cranial nerves.
- Parotid – inferior salivary nucleus with IX CN, then V3 CN.
- Submandibular – superior salivary nucleus with facial, then lingual.
**Presentation**
Main symptoms are swelling and pain, if widespread disease then dry mouth. May be diffuse or a single gland. Lacrimal gland may also be involved – dry itchy eyes.

Questions –
Swelling after meals, pain, facial palsy, unilateral or bilateral, foul taste in mouth, associated arthritis, trismus.

A dry mouth can be due to –
Anxiety, drugs (antimuscarinic or sympathomimetic), Sjogren’s syndrome.

**Examination**
Inspection, palpation (external and intra-oral). Examine oral cavity and oropharynx (can see deep lobe parotid tumours) or could be primary tumour. Check facial nerve. Palpate neck.

**Investigations**

**Systemic viral infections**

**Sialadenitis**
Acute infection of gland – pain and swelling. Generally older patients (dehydrated, poor oral hygiene) or stasis of saliva (stones, dehydration, medication). Treatment - high dose antibiotics, rehydration, oral hygiene, citrus mouthwashes to improve saliva flow, surgical drainage.

**Sialolithiasis**
Calculi form in glands often with chronic sialadenitis. Generally occur in submandibular gland- can feel stone in floor of mouth. Post prandial swelling and pain. Conservative treatment – oral fluids, sialogogues to pass stones spontaneously. Surgical excision of stone or gland.
**Tumours**
Most arise in parotid – 80% benign. Tumours in submandibular and small gland are more likely to be malignant. Tumours are thought to come from reserves cells (can differentiate into different duct cell types).
Benign – generally pleomorphic adenoma, sometimes adenolymphoma.
Malignant – involve other structures e.g. nerves, rapid growing, neck metastases. Common types are adenoid cystic carcinoma (propensity for perineural infiltration), carcinoma ex pleomorphic adenoma, adenocarcinoma, lymphoma and muco-epidermoid (variable malignancy).
Treat by wide excision biopsy, with maybe neck dissection and maybe radiotherapy.
Non-salivary swellings can also present e.g. hypertrophy of masseter, ageing (absorption of adipose tissue makes glands more prominent), neuromas, lymphadenopathy, mandibular tumour, abscesses etc.

**Inflammatory conditions and other syndromes**
Sjogrens syndrome:
- Chronic autoimmune.
- Dry mouth and eyes, joint pain (if secondary to connective tissue disease e.g. RA, SLE.
- Risk of developing lymphoma.
Heeford’s syndrome – varicose veins and facial paralysis associated with sarcoidosis.
Wegener’s syndrome – respiratory tract and kidney granulomatosis.
Frey’s syndrome – after parotidectomy – skin sweating over parotid gland on eating.
Pharynx and Throat

Pharynx
Chamber with bony and fibromuscular walls – tract to respiratory and alimentary systems. Communicates with various chambers guarded by muscular mechanisms:
- Nasal chambers – soft palate and sphincter muscle
- Eustachian tubes – palatal and salpingopharyngeal muscles
- Oral cavity – base of tongue
- Larynx – aryepiglottic folds
- Oesophagus – cricopharyngeal sphincter
Functions include channel for air and food, pharyngeal phase of swallowing, sneezing, formation of speech sounds, sensation.
Divided into:
- Nasopharynx – limited inferiorly by hard palate, includes adenoids and Eustachian tube openings.
- Oropharynx – between hard palate and hyoid bone, separated from oral cavity by palatine tonsil and circuvallate papillae line. Contains soft palate, uvula, tonsils and posterior third of tongue.
- Hypopharynx – between hyoid bone and cricoid. Contains piriform fossae, posterior pharyngeal wall and postcricoide space.

Catarrh
No set definition, generally implies excessive mucus secretion. May be chronic infection, allergy or autonomic dysfunction due to emotion, endocrine changes, cold etc.

Sore throat
Tonsillitis – bilateral throat pain with odynophagia, systemic upset and pyrexia which is acute onset due to a bacterial infection.
Quinsy – develops from tonsillitis. Systemic upset, odynophagia, trismus (can’t open mouth as pterygoid muscles in spasm).
Chronic pharyngitis – long standing discomfort in throat, pain on swallowing and earache. May be related to chronic sinusitis, bronchitis or reflux or local irritants e.g. fumes, smoking. Need to exclude carcinoma.
Acute pharyngitis – usually viral e.g. rhinoviruses, influenza, also diphtheria, infectious mononucleosis.

Ear ache
Can be associated with throat irritation due to referred pain via CN IX and X. Most common causes – tonsillitis, dental lesions, ulcerations, neoplasms.

Swallowing
Complex co-ordinated reflex usually initiated voluntarily:
- Oral stage – voluntary
- Pharyngeal stage – involuntary
- Oesophageal stage – involuntary
Dysphagia – difficulty swallowing.
Globus pharyngeus – sensation of pressure in throat, which is relieved by swallowing. Investigate by contrast swallow (structural abnormalities) or dynamic contrast swallow (physiological abnormalities).
Cough
Generated from larynx by local irritation.
Tends to be associated with respiratory inflammatory diseases or lower digestive inflammatory conditions.
May be due to aspiration – salivary, prandial or reflux.

ENT Emergencies

Trauma to ear
External ear:
- Vulnerable as exposed and soft.
- Blunt trauma – bruising, haematoma causing scarring or necrosis of cartilage and deformity (cauliflower ear) if infection occurs. Treat by drainage, pressure and antibiotics.
- Sharp trauma – lacerations to avulsions. Good blood supply so heal well.
- Thermal trauma – frostbite, needs rewarming and maybe debridement.
- Foreign bodies – pain, discharge, deafness.

Middle ear:
- Trauma causes tympanic membrane perforation and ossicular dislocation leading to hearing loss. Perforation causes hearing loss of about 20dB, ossicular dislocation about 60dB.
- Perforations usually heal if ear is kept clean and dry. Dislocation needs surgical intervention.
- Foreign body - typically cotton buds.
- Air pressure – loud noise or slap can cause perforation or ossicular damage.
- Head injury – temporal bone fracture, disrupt EAM etc.
- Barotrauma – especially if diving or flying with poor Eustachian tube function. Middle ear effusion, deafness, pain, vertigo. Treat with nasal decongestants, myringotomy or grommets.

Inner ear:
- Acoustic trauma – sensorineural deafness, if chronic usually dip at 4kHz and tinnitus.
- Labyrinthine concussion – head injury, high freq hearing loss, tinnitus, vertigo.
- Temporal bone fracture – sensorineural hearing loss and profound vertigo, facial nerve palsy, CSF otorrhoea, bleeding from ear.
- Round or oval window rupture – rapid pressure changes – fluctuating hearing loss and vertigo.

Trauma to nose
Trauma is common – generally fractured nose.
Septal haematomas must be drained.
If no cosmetic deformity leave to heal. If problem, manipulate under anaesthetic within 2 weeks.

Epistaxis
Usually mild and self-limiting but can be life threatening.
Causes –
- Local – idiopathic, trauma, infection, tumour.
- Systemic – hypertension, anticoagulants, coagulopathy, hereditary haemorrhagic telangiectasia.
Generally from anterior septum (Little’s area).
**Tonsillitis**
Symptoms – sore throat, difficulty swallowing, pyrexia, malaise, halitosis, lymphadenopathy, inflammation, enlargement of tonsils.
Complications – febrile convulsions, abscesses (peritonsillar, parapharyngeal and retropharyngeal).
Tonsillectomy – suspected malignancy, sleep apnoea, recurrent tonsillitis (5 attacks in one year).
Main risk is bleeding – reactionary (few hours) or secondary (few days due to infection).

**Peri-tonsillar abscess (quinsy)**
Most common infective complication of tonsillitis.
Tonsil is pushed medially and uvula displaced. Patient is more unwell with drooling, fetor and trismus.
Treat by decompression (aspiration or incision) and antibiotics.

**Important Note**
These notes were written by Liz Tatman, as a fourth year medical student in 2006. They are presented in good faith and every effort has been taken to ensure their accuracy. Nevertheless, medical practice changes over time and it is always important to check the information with your clinical teachers and with other reliable sources.
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