HEARING LOSS:
AN OVERVIEW OF THE CAUSES, INVESTIGATIONS AND MANAGEMENT

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ANATOMY REMINDER

- OUTER EAR
- MIDDLE EAR
- INNER EAR

Requisites for hearing:
- unimpeded access to a mobile drum & ossicles,
- an aerated middle ear,
- functioning cochlea, nerve, and central auditory pathways
EXTERNAL EAR

- WAX, SKIN DEBRIS, EXOSTOSES, OSTEOMATA, GRANULOMAS, POLYPS, OBLITERATIVE OTITIS EXTERNA

- (Remember necrotising otitis externa – blocked ear, pain, polyp, blood, diabetic patients)
MIDDLE EAR (mostly conductive)

- Eustachian insufficiency
- **Effusion**
- Chronic tympanic retraction
- Tympanosclerosis
- Atelectasis
- Chr. suppurative otitis media +/- cholesteatoma
- Ossicular disarticulation
- Perforation
- **Otosclerosis**
INNER EAR (sensorineural)

- Congenital s.n. deafness
- Acquired s.n. loss – viruses, drugs, diabetes
- noise-induced
- Presbyacusis
- Compressive lesions e.g. acoustic neuroma, meningioma
- Demyelination
- (Meniere’s)
CONDUCTIVE DEAFNESS
- external / middle ear

- **Congenital** cond. deafness appearing *at birth*;
  usually *syndromic*, e.g. Marfan/Crouzon/Down;
  cong. cond. deafness *in childhood / adults*,
  e.g. *otosclerosis*;
  congenital disorders leading to middle ear effusion,
  e.g. cystic fibrosis, cleft palate

- **Acquired** cond. deafness: inflammation, including
  otitis media with effusion *(OME)* and chronic
  infection; occluded external meatus
SENSORINEURAL DEAFNESS
inner ear and central auditory pathways

- **Congenital genetic** – at birth / in childhood; may be syndromic or deafness alone

- **Congenital non genetic** – infections in utero e.g. rubella, cmg, drugs, metabolic disorders

- **Perinatal disorders** – e.g. hypoxia

- **Acquired conditions** – **noise**, infection, immunisation (tetanus), meningitis, drugs, trauma including barotrauma, autoimmune deafness, MS, neoplasm (acoustic neuroma)
NORMAL EARS?
OTITIS MEDIA – ACUTE /CHRONIC
CHOLESTEATOMA
NEONATES with s.n. hypoacusis

- 1 in 1000 children suffer from sensorineural deafness;
  - 50 % hereditary
  - 50 % acquired

- National Screening; presently only at risk screening
  - OTOACOUSTIC EMISSIONS testing:
    - Pass: follow-up as necessary
    - Fail: investigate further: D.OAE, ABR

- Confirmed s.n. deafness – identify possible ante/peri-natal cause, consider genetic testing/counseling;

- Longterm assessment of severity; consider aiding if severe;
  - if profound consider COCHLEAR IMPLANT
CHILDREN with hypoacusis

- Most have a reversible conductive loss secondary to otitis media with effusion.
- Truly deaf children are usually picked up during testing for speech delay.
- Any child with suspicion of deafness should be adequately tested.
- Moderate to severe irreversible deafness requires a hearing aid.
- Profound deafness may require an IMPLANT.
TESTING children

- All children with aerated middle ears: Otoacoustic emissions
- 4–30 months: distraction testing
- 2 years: conditioning (play) audiometry
- 2-3 years: pure tone audiometry
- *Tympanometry is not an audiometric test*
- Any age: auditory brainstem responses for difficult diagnoses; may require sedation
OTITIS MEDIA WITH EFFUSION
(old term ‘glue ear’)

- Serous/mucoid effusion secondary to Eustachian malfunction or an altered mucociliary system
- Causes – adenoids, hyperreactivity, viruses (surfactant deficiency), hormones (thyroid), cystic fibrosis, fungal allergy, (parental)smoking, craniofacial abnormality (Down’s, Hunter’s), nasopharyngeal tumours (one ear)
OTITIS MEDIA WITH EFFUSION  
(old term ‘glue ear’) cont.d

- nearly always resolves spontaneously; never progresses to s.n. deafness typically variable, recurrent

- 20 – 30 dB conductive hearing loss

- No other symptoms

- Flat tympanogram

- Typical otoscopic changes
OTITIS MEDIA WITH EFFUSION

- management

most cases resolve spontaneously

IN ADULTS ALWAYS EXCLUDE NASOPHARYNGEAL CARCINOMA

- Medical treatment – topical steroids, A.H.

- Surgery if hearing loss significant (+30 dB) for more than 3 months – still being debated:
  adenoidectomy / myringotomy / laser / grommets / T-tubes / ? Aid
Ventilation tubes
COCHLEAR IMPLANTS

- *Not hi-tech hearing aids – no amplification*

- Used for profound s.n./cond./mixed loss

- **Components:**
  - MICROPHONE – picks up sound
  - SPEECH PROCESSOR – prioritises speech over background noise
  - TRANSMITTER – delivers transcutaneous signal to an implanted subcutaneous internal receiver
  - RECEIVER – elaborates signal and sends it to an implanted electrode in cochlea
COCHLEAR IMPLANTS
HEARING AIDS - amplifiers

- Microphone, processor with volume control, tubing, ear mould
- Behind-the-ear, in-the-canal, in-the-ear, bone-anchored, implantable
- Analogue / digital
Otosclerosis

- Commonest cause of non-inflammatory conductive hearing loss
- Genetic, autosomal dominant
- 2% of the population
- Measles
- Hormonal challenge
- New bone deposition at the ovalo-stapedial joint resulting in fixation of stapes
Otosclerosis - management

- **Option 1**
  Hearing aid – no risks but disease progresses unchecked

- **Option 2** - Surgery – high success rates; disease arrested; 1 – 2 % risk of damage (irreversible) to the operated ear; enables use of a hearing aid in profound mixed loss
Stapedotomy surgical technique

- Permeatal approach, elevation of drum, division of stapedius tendon, occasionally also of corda tympani, excision of stapes superstructure, teflon piston interposed between incus and surgical finestra in oval window.

- Using laser – no touch technique allows for a minimum manipulation, reducing complications
Stapedotomy - complications

- Failure – resulting from displacement of piston
- 1 % dead ear – exposed labyrinth
- 1% persistent vertigo – fistula
- Remote risk of VII palsy
- Remote risk of implantation cholesteatoma
Stapedotomy is a highly successful intervention requiring superior technical skills which should only be carried out by otologists experienced in this procedure.
Stapedotomy - techniques