

COMMON EXTERNAL, MIDDLE AND INNER EAR DISORDERS

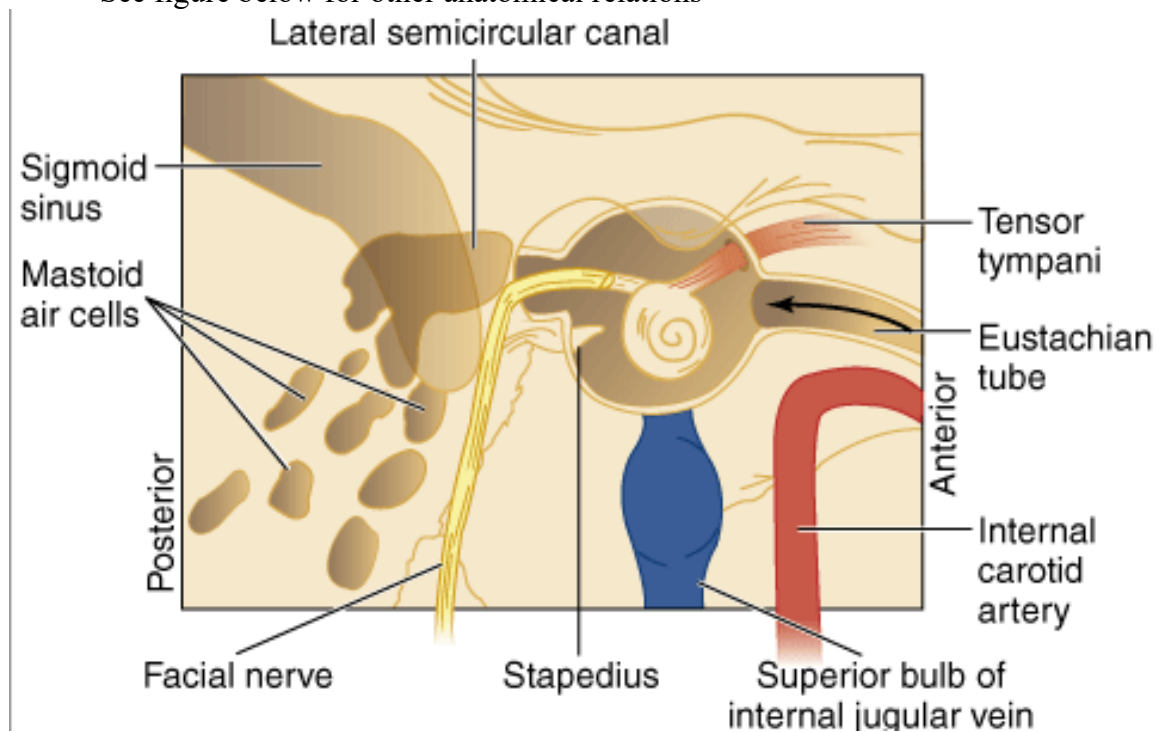
NORMAL ANATOMY:

EXTERNAL EAR:

- The AURICLE (or PINNA), is the visible portion of the ear
- The outer one third of the external auditory canal is composed of an incomplete cartilaginous tube
- The inner two thirds is composed of bone covered by a thin layer of tightly adherent skin
- The posterior auricular vein frequently connects to the sigmoid sinus, providing a route for extension of infected material into the intracranial cavity

MIDDLE EAR:

- An air-containing cavity in the petrous temporal bone
- Contains the auditory ossicles, which transmit vibration so the TM to the perilymph of the inner ear
- Communicates with the nasopharynx by the Eustachian tube and with mastoid air cells by the aditus ad antrum
- See figure below for other anatomical relations



INNER EAR:

- Consists of the cochlea (which contains the auditory sensory receptors) and the vestibular labyrinth (balance receptors)
- Blood supply is from the vertebrobasilar system

OTALGIA:

- PRIMARY OTALGIA is caused by auricular and periauricular disease
 - Referred otalgia is caused by disease originating from remote structures
 - Referred otalgia is common because of the multiple cranial nerves and branches of cervical plexus that supply this region

Table 237-1 Causes of Otalgia		
Primary	Referred	Neuralgias
Trauma	Dental	Trigeminal (tic douloureux)
Infection	Temporomandibular joint disease	Herpetic geniculate (Ramsay Hunt syndrome)
Otitis externa		
Otitis media	Abscessed teeth/dental carries	Foramina narrowing
Mastoiditis		
Bullous myringitis	Malocclusion	
Foreign bodies	Bruxism	
Cerumen impaction	Trauma	
Cholesteatoma	Retro- and oropharyngeal	
Neoplasms	Tonsillitis	
Pinna cellulitis	Abscess	
	Neoplasm	
	Nasal cavity	
	Sinusitis	
	Deviated septum	
	Throat and neck	
	Foreign body	
	Thyroid disease	
	Cervical strain	
	Neoplasm	
	Foreign bodies	

PRIMARY OTALGIA:

- The mandibular division of the trigeminal nerve mediates ear sensation for the anterior outer ear and the facial nerve carries sensory innervation from the external auditory canal and the skin behind the auricle
- Disease from any portion of the ear or its surrounding skin and structures may result in primary otalgia

REFERRED OTALGIA:

- The maxillary and mandibular divisions of the trigeminal nerve receive sensory input from the nasopharynx, paranasal sinuses, teeth, parotid gland and muscles of mastication

- Referred pain is common and has multiple causes (see above)

TINNITUS:

- The perception of sound without external stimulation
- Most prevalent between the ages of 40-70
- TWO TYPES → OBJECTIVE (that which can be heard by the examiner) and SUBJECTIVE (more common and its exact origin is unknown, thought to be due to damage to cochlear hair cells).

Table 237-2 Common Causes of Tinnitus	
Objective	Subjective
Vascular	Sensorineural hearing loss
Arteriovenous malformations	Hypertension
Arterial bruits	Conductive hearing loss
Mechanical	Head trauma
Enlarged eustachian tube	Medication side effects
Palatomyoclonus	Temporomandibular joint disorders
Stapedial muscle spasm	Depression, stress
	Neurologic
	Acoustic neuroma
	Multiple sclerosis
	Benign intracranial hypertension
	Ménière disease
	Cogan syndrome

- Pharmacologic causes account for at least 10%

Table 237-3 Common Ototoxic Agents Causing Tinnitus	
Loop diuretics	Chemotherapeutic agents
Ethacrynic acid	Cisplatin
Furosemide	Carboplatin
Bumetanide	Vinblastine
Salicylates	Vincristine
NSAIDs	Topical agents
Quinine	Solvents
Antibiotics	Propylene glycol
Aminoglycosides	Antiseptics
Erythromycin	Ethanol
Vancomycin	Antibiotics
	Polymyxin B
	Neomycin

- The most commonly implicated agents are aspirin (and aspirin-containing drugs), as well as antibiotics (particularly aminoglycosides)
- Refer to ENT
- Antidepressants are currently the only class of drug found to be useful in alleviating tinnitus for which no correctable cause can be found

SUDDEN HEARING LOSS:

- Defined as hearing loss that occurs over three days or less
- Indicators of poor prognosis include more severe hearing loss on presentation and the presence of vertigo

Table 237-4 Causes of Sudden Hearing Loss	
Infection	Rheumatologic
Mumps	Temporal arteritis
Epstein-Barr virus	Polyarteritis nodosa
Herpes	Wegener granulomatosis
Cytomegalovirus	Other
Syphilis	Ménière disease
Labyrinthitis	Cogan syndrome
Hematologic and vascular	Acoustic neuroma
Leukemia	Pharmacologic (Table 237-3)
Sickle cell anemia	Cochlear rupture
Polycythemia	Conductive
Berger disease	Otitis externa
Cerebral aneurysm	Otitis media
Metabolic	Ruptured tympanic membrane
Diabetes mellitus	Neoplasms
Hyperlipidemia	Otosclerosis

- Viral infections, most typically mumps have been associated with sudden hearing loss
- COGAN SYNDROME → autoimmune disorder that presents with bilateral hearing loss classically associated with vertigo
- Coexistent tinnitus and vertigo implicates Meniere’s disease
- Popping sound prior suggests perforation of TM or dislocation of the ossicles

INFECTIONS:

OTITIS EXTERNA:

- Includes infection and inflammation of the external auditory canal and auricle
- Divided into ACUTE DIFFUSE AND MALIGNANT TYPES
- **ACUTE DIFFUSE OTITIS EXTERNA:**
 - Known simply as OE or “Swimmer’s ear”
 - Characterised by pruritus, pain and tenderness to the external ear

- Signs → erythema and oedema of the external auditory canal, which may spread to the tragus and auricle
- Other signs are clear or purulent otorrhoea with crusting of the external canal
- There may be lateral protrusion of the aurical secondary to inflammation
- PATHOPHYSIOLOGY:
 - Predisposing factors → elevation of local pH, trauma to the skin. pH can be raised by freshwater bathing frequently
 - Trauma can be due to overzealous disimpaction of cerumen (this normally forms a physical barrier that protects the skin of the external auditory canal and it has an acidic pH that protects the skin)
- MICROBIOLOGY:
 - Most common organisms → PSEUDOMONAS AERUGINOSA, enterobacter, Proteus and Staph
 - OTOMYCOSIS (fungal OE) is found in tropical climates or in the immunocompromised, caused by Aspergillus or Candida
- TREATMENT:
 - Analgesia, cleansing of the external canal, acidifying agents and topical antimicrobials
 - Although there are few established cases of ototoxicity, there is a theoretical risk of both auditory and vestibular toxicity with use of aminoglycosides, polymyxin and acetic acid preparations
 - Instill the medication in to the cleansed ear with the ear facing up, with this position held for 3 minutes

1 dexamethasone 0.05% + framycetin 0.5% + gramicidin 0.005% ear drops 3 drops instilled into the ear, 3 times daily for 3 to 7 days [Note 1]

OR

1 flumethasone 0.02% + clioquinol 1% ear drops 3 drops instilled into the ear, twice daily for 3 to 7 days [Note 1].

- If oedema of the external canal obstructs the lumen, insert a commercial wick or a piece of gauze into the canal and keep it moist with otic drops
- Oral antibiotics are reserved for febrile patients and those with periauricular extension
- Need to keep the ear dry
- OTOMYCOSIS should be treated with systemic FLUCONAZOLE
- **MALIGNANT OTITIS EXTERNA:**
 - A POTENTIALLY LIFE-THREATENING INFECTION of the external auditory canal involving the pinna and soft tissues with variable extension to the skull base
 - In >90% cases, it is caused by P. aeruginosa
 - When infection is limited to the soft tissues and cartilage, it is called NECROTISING OTITIS EXTERNA, when there is involvement of the temporal bone or skull base it is called SKULL-BASE OSTEOMYELITIS
 - PATHOPHYSIOLOGY:
 - Begins as a simple otitis externa and then spreads to the deeper tissues

- DIABETES AND IMMUNOSUPPRESSION predispose to the onset of pseudomonal infection
 - Cerumen of diabetic patients has a higher pH
- CLINICAL FEATURES AND DIAGNOSIS:
 - An individual with persistent otitis externa despite 2-3 weeks of topical antimicrobial therapy and those with otalgia and oedema of the external auditory canal with or without otorrhoea
 - OTALGIA OUT OF PROPORTION FOR ROUTINE OTITIS EXTERNA
 - The infected ear will be erythematous, oedematous and more prominent
 - Parotitis may be present and trismus indicates involvement of the masseter muscle or TMJ
 - Cranial nerve involvement is a SERIOUS SIGN → skull base osteomyelitis first involves CN VII, then IX, X and XI (implies even more extensive disease)
 - Lateral or sigmoid sinus thrombosis and meningitis are more serious possible complications
 - CT IS USED FOR DIAGNOSIS
 - In kids, this condition tends to be rapidly progressive and they may be ill-appearing on presentation
- TREATMENT:
 - Treat for pseudomonas aeruginosa with systemic agents

If sepsis is due to *Pseudomonas aeruginosa*, until susceptibility results are available, use:

gentamicin 7 mg/kg (child less than 10 years: 7.5 mg/kg; 10 years or more: 7 mg/kg) IV, for 1 dose, then determine dosing interval for a maximum of either 1 or 2 further doses based on renal function (see [Table 2.25](#))

PLUS EITHER

1 ceftazidime 2 g (child: 50 mg/kg up to 2 g) IV, 8-hourly

OR

1 piperacillin+tazobactam 4+0.5 g (child: 100+12.5 mg/kg up to 4+0.5 g) IV, 6-hourly [[Note 1](#)].

For patients with immediate hypersensitivity to penicillin (see [Table 2.2](#)), until susceptibility results are available, use:

ciprofloxacin 400 mg (child: 10 mg/kg up to 400 mg) IV, 8-hourly

PLUS

gentamicin 7 mg/kg (child less than 10 years: 7.5 mg/kg; 10 years or more: 7 mg/kg) IV, for 1 dose, then determine dosing interval for a maximum of either 1 or 2 further doses based on renal function (see [Table 2.25](#)).

OTITIS MEDIA:

- See discussion in paediatric chapter for detail
- There are no treatment guidelines specifically for adults
- Preferred treatment is still amoxicillin and analgesia
- **COMPLICATIONS OF OTITIS MEDIA:**
 - **PERFORATION:**
 - Most often occurs in the pars tens with resultant otorrhoea
 - Healing usually occurs in 1 week, although a chronic perforation may result
 - **ACUTE MASTOIDITIS:**

- Results from spread of infection from the middle ear to the mastoid cells by the aditus ad antrum and can then spread to the overlying periosteum by the venous channels → acute mastoiditis with periostitis
- In addition to otalgia and fever, patients will have postauricular erythema, swelling and tenderness with protrusion of the auricle and obliteration of the post-auricular crease
- CT delineates the extent of bony involvement
- REQUIRES ADMISSION FOR IV ANTIBIOTICS, TYMPANOCENTESIS AND MYRINGOTOMY
- INCISION AND DRAINAGE OF SUBPERIOSTEAL ABSCESS OR MASTOIDECTOMY MAY ULTIMATELY BE REQUIRED
- **INTRACRANIAL COMPLICATIONS:**
 - More likely with chronic than with acute OM and are decreasing with widespread use of antibiotics
 - Suppurative intracranial extension is a severe complication → meningitis and brain abscess are the most common complications
 - Extradural abscess and subdural empyema are also potential complications
- **LATERAL (SIGMOID) SINUS THROMBOSIS:**
 - Arises from extension of infection and inflammation of the mastoid with eventual inflammation of the adjacent sigmoid sinus
 - HEADACHE is most common symptom with papilloedema, sixth-nerve palsy and vertigo being less frequently found

BULLOUS MYRINGITIS:

- A painful condition of the ear characterised by bulla formation on the TM and deep external auditory canal
- Blisters occur on the outer epithelium and inner fibrous layer of the TM → hence severe otalgia
- Treatment is pain control but antibiotics are an option

FROSTBITE:

- Extremely susceptible to extremes in temperature
- Quickly rewarm with saline-soaked gauze

BURNS:

- Even with lesser injury, disruption of the auricular skin can lead to damage of the underlying cartilage, which is particularly susceptible to infection once damaged
- Should be seen in a burn unit
- Osteochondritis is a potentially disfiguring complication of otic burns → surgical debridement plus systemic antibiotics

HAEMATOMA:

- An auricular haematoma can develop from almost any type of trauma to the ear

- Blunt force tends to shear the perichondrium from the underlying cartilage and tear the adjoining blood vessels
 - The cartilage depends on the perichondrial blood vessels for viability and any interruption can result in necrosis → which can result in an asymmetric formation of new cartilage growth → CAULIFLOWER EAR
- THE GOAL OF TREATMENT IS TO REMOVE THE FLUID COLLECTION BY INCISION OR ASPIRATION (aspiration alone does not completely evacuate the clot) → use a semicircular incision through the skin with caution not to violate the underlying perichondrium and remove the clot by gentle suction curettage.
- Prophylactic antibiotics after I&D warranted

FOREIGN BODIES:

- Live objects can be drowned with 2% lignocaine or viscous lignocaine
- Irrigation with room-temperature is adequate for small particles, but should not be used unless the TM is completely visualized and intact



These objects can be used to remove FB or cerumen

CERUMEN IMPACTION:

- Most impacted individuals will report decreased hearing, a sensation of pressure or fullness in the ear, with dizziness or otalgia
- May need to use softener first (waxsol or similar agent)
- Can use cerumen loops or scoops can be used in most cases
- Syringing can be used, but is associated with traumatic TM perforation → safer to defer to ENT
 - Important to rely on symptoms (sudden hearing loss, severe otalgia or vertigo) rather than signs as the TM may be obscured by irrigation fluid

TYMPANIC MEMBRANE PERFORATION:

- The pars tensa is the largest area of the TM and is only a few cell layers thick and thus is easily torn/perforated
- Patient usually complains of an acute onset of pain and hearing loss, with or without bloody otorrhoea
- They may be associated vertigo or tinnitus, but this is usually transient
- TM perforations heal spontaneously and can be safely discharged in most cases
 - Should be instructed not to allow water to enter the canal
 - Perforations in the posterosuperior quadrant or those secondary to penetrating trauma have a greater likelihood of ossicular chain damage and should be referred to ENT within 24 hours