CHAPTER 11.

EAR TRAUMA

Trauma of various types may affect the ear. The trauma may be direct, e.g. penetrating canal injuries, or indirect, such as a concussive head blow. The injury can affect one or several of the ear component structures.

1. PINNA

The protrusion of the pinna from the side of the head places it at risk from lacerations or crushing injury. Developmentally, the pinna is highly vascular, which aids recovery from ragged injury, and helps to avoid severe infection from such insults.

a) Lacerations

These may be accidental, inflicted, or surgical. Soiled wounds should be carefully cleaned, then sutured accurately, apposing cartilage fragments and immobilising same if necessary, for optimal cosmetic outcomes.

b) Haematoma auris

Crushing injuries may produce pinna haematomas. The problem is especially likely if a rolling compressing force is applied that shears the perichondrium off the underlying elastic cartilage. This may produce a substantial clot between the two, possibly on both sides of the cartilage, which may be torn in the process. Resultant avascular necrosis may occur if the cartilage is left ischaemic from separation off its vascular supply. Left untended, gradual fibrosis occurs as the clot organises, and the cartilage necroses. Permanent and unsightly disfigurement follows, characterised by a thickened, stiff pinna with distorted contours – “cauliflower ear”.

Figure 1: Pinna defect secondary to a dog bite. Loss of the upper helix. Difficult to repair cosmetically.

Surgery, especially for malignancy, commonly involves significant resections. These should be planned for best outcomes, possibly employing local flaps to restore the defects.
Prevention includes prompt sterile evacuation of the clot, correction of the structural alignment, and appropriate antibiotic care to avoid perichondritis. Once cleared of clot, the pinna is dressed carefully to maintain contour alignment during the healing phase.

Other frequent problems occur from syringing (without steadying the syringe hand against the head), removal of insects, or by blind cleaning attempts.

Lacerations commonly result in blood clot accumulation in the canal, causing deafness and obscuring drum details. Infection may follow.

Figure 2: Haematoma auris, rugby injury. The subperichondrial clot should be evacuated under sterile conditions, to minimise any cosmetic sequelae.

2. EXTERNAL CANAL TRAUMA

Direct trauma to the canal skin is regrettably common. The problem is frequently self-inflicted during cleaning episodes with cotton buds, hair/paper clips or other such devices that readily gouge the delicate canal lining.

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Figure 4: Infected granuloma, following EAC trauma. Clean and manage with topical antibiotics.

Less commonly, canal trauma may follow skull fractures, and sometimes compression of the temporo-mandibular joint into the canal by direct trauma to the chin.

The canal skin trauma is managed by cleaning, preferably with suction toilet, followed by topical antibiotics to avoid infection. Rapid healing is the norm. If the status of the drum or middle ear is in doubt, a specialist's opinion is advisable.

Occasionally, more extensive gouging, post-surgical skin loss, or pressure effects by hearing aids may cause extensive skin loss that may require surgical correction by grafting with fascia or perichondrium to facilitate re-epithelialisation, that may be otherwise prolonged, perhaps over several months.

Figure 3: Minor bruising of the pars tensa. Cotton bud ear cleaning injury.
3. TYMPANIC MEMBRANE TRAUMA

The eardrum may be damaged by a variety of insults. As well as the penetrating injuries also suffered by the EAC, the drum is also prone to slapping or blast compressive injuries, hot particles and inappropriate cleaning techniques, including syringing.

The drum may also be ruptured as a result of a temporal bone fracture.

Concussive injuries may produce a rent in the pars tensa, whereas a spot of welding slag may produce a rounded hole. Penetrating injuries are typically bloody, often with concurrent canal skin tears. Syringing is a particular risk when prior tubal insufficiency has caused drum atrophy.

The causative agent may affect the drum’s healing pattern. Dry concussive events may heal rapidly, as may clean penetrative patterns. Burns however, are notorious for failed healing, and water-caused concussive events are often due to soiled water; healing may be slow or halted by resultant infection.

More severe concussive events (blast) may implode the drum, perhaps impacting squamous epithelium that later causes cholesteatoma.

When a traumatic perforation is present, clean the canal by suction, and use a systemic broad spectrum antibiotic if there is probability of impending infection, but not otherwise. Add antibiotic drops if infection appears present. Once the acute phase of the injury has settled, arrange audiology to check for evidence of ossicular damage. Up to 80+% of non-infected (non-burn) defects will close over 1-2 months, less so if infected.

Failure to heal is managed by myringoplasty.
4. OSSICULAR CHAIN TRAUMA

As with the drum, the chain may be damaged by direct, concussive or head injury trauma. Healing of a drum injury may leave persisting deafness, with the Weber referred to the deaf side and a negative Rinne test, indicating a conductive pattern. Attic fixation, incudal dislocation and stapes superstructure damage are common. A fracture line may be discerned, usually in the postero-superior canal. Close inspection under microscopy may suggest wrinkling of the pars tensa, or the incus may be seen to be malpositioned.

Figure 7: Trauma to the drum and ossicular chain resulting from a longitudinal temporal bone fracture. The malleus handle has snapped.

Audiology may confirm a significant conductive loss. If so an ossiculoplasty or hearing aid use is appropriate. The former generally returns good results, as there is usually no other active ear pathology.

5. TEMPORAL BONE FRACTURES

a) Longitudinal Fractures

Longitudinal defects result from blows to the side of the head. The fracture line descends across the squamous temporal, sometimes as a barely discernable hairline on plain film studies. The fracture then passes antero-medially through the roof of the EAC, then possibly through the attic of the middle ear, but skirting lateral to the hard bone of the otic capsule. The facial nerve may be compromised at this point.

Figure 8. Ossicular chain disruption in the attic. The incudo-malleolar joint has separated, causing a major conductive loss.
If CSF otorrhoea is present, treat with topical and systemic broad spectrum antibiotic cover after cleaning the EAC. Reassess the hearing once the acute phase has settled.

If facial palsy is noted, early exploration and decompression may overcome compression by the fracture and avoid long term paralysis.

b) Transverse Fractures

Transverse fractures result from more severe trauma delivered antero-posteriorly. Perhaps 20% of temporal fractures, these are frequently accompanied by CNS damage or death.

The fracture line passes across the petrous temporal, from the posterior fossa, through the dense bone of the otic capsule, rending and destroying the membranous labyrinth. The facial nerve, fixed to the capsule in its horizontal course is also transfixed.

The patient’s general condition is critical, such that the ear trauma may not be immediately apparent, or may be triaged as less pressing.

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Figure 9: Temporal bone fractures. Blows to the side of the head cause longitudinal fractures (L) that pass through the upper EAC and the attic. Frontal blows cause transverse patterns (T), disrupting the chain and drum, and may cause facial paralysis. They transect the otic capsule, causing profound sensorineural deafness, dizziness and related severe head injury.

Figure 10: Longitudinal fracture of the temporal bone. A substantial linear defect is seen in the roof of the canal, leading into the attic. Drum intact.

On inspection, blood and possible CSF may be present in the EAC, with a haemotympanum evident. Suction toilet may reveal a tear along the rear of the canal and/or drum rupture.

Figure 11: Transverse fracture of the temporal bone, with a concurrent occipital fracture. Severe frontal trauma.
Upon recovery of consciousness, gross disequilibrium, severe tinnitus and profound sensorineural deafness are noted.

The unsteadiness may be compounded by cerebellar or brainstem injury, causing gross long term disability. The facial nerve palsy may go unnoticed until recovery of consciousness. This may require formal nerve grafting procedures as a result or physical division, or delayed treatment of a milder compressive situation.

CT radiology at admission of head injuries will identify the fracture and its pattern. Early assessment for VII damage is strongly recommended and operative management if needed should be prompt. Later audiology will clarify the hearing situation. Regrettably, some transverse fracture cases will transect the VIII nerve or inflict severe cochlear damage such that cochlear implantation of such cases will be prevented. Progressive VII assessments may help guide the management of injury to this nerve.

6. BAROTRAUMA

Barotrauma occurs when an individual cannot open the Eustachian tube in conditions of increasing external pressure, as in aircraft descent or diving. The trauma is incurred when the pressure differential reaches approximately 80-90 mm.

The problem may be an idiopathic lesser tubal insufficiency, triggered only in acute circumstances or secondary to often minor URTI.

The clinical event appears during or shortly after the trigger event. In more severe episodes, e.g. diving accidents, pain may be severe, followed by immediate deafness or even bleeding from the ear.

Figure 12: Acute Barotrauma. A bloodstained effusion has formed secondary to a diving episode.

Dizziness/unsteadiness is common. In lesser events, subtle stuffiness may emerge with loss of hearing being experienced in the hours after the event.

Severe episodes may incur a ruptured and bleeding drum similar to blast injuries. Milder patterns are more common. Subtle bruising, especially along the handle of the malleus may be the only finding. In other cases, bubbles or fluid levels are visible, heavily bloodstained in some cases. The effusion may persist for weeks, especially if a causative URTI is present. Tuning fork tests show a conductive loss (although sensorineural losses are also well noted).

Management depends on the individual case. Lesser episodes will clear spontaneously, but if effusions persist, a temporary mini-grommet will expedite recovery, inserted under a simple topical anaesthesia. Vent tubes are also used widely as preventative measures for those with a demonstrated susceptibility to barotrauma, when aircraft travel is essential. Perforated drums normally heal, but some will require drum repair.
Sensorineural deafness may respond to aiding, if not too severe. Decongestants or other topical/systemic medications are of uncertain therapeutic or preventive value. “Ear plane” earplugs are ineffective. INNER EAR TRAUMA

Many of the above traumatic events may also incur inner ear trauma.

Patterns of Inner Ear Trauma

a) Penetrating injury may produce damage by stapes avulsion.
b) Blast may cause concussive sensorineural effects without other pathology, or may cause a perilymph fistula
c) Barotrauma may also cause fistula
d) Diving may cause the “bends”: nitrogen bubbles in the otic capsule with subsequent severe losses.
e) Head injury may cause concussive sensorineural losses, or a fracture may rupture the otic capsule, with total loss resulting.

Perilymph fistula

A range of factors (blast injuries [e.g. bombs], severe barotrauma, other severe noise injuries, or stapedial avulsion/surgery) may cause persistent loss of perilymph from either the oval or round windows of the inner ear. Generally there is a history of a trigger incident. Followed by sensorineural loss, perhaps slight or fluctuant at the onset, then worsening, with disequilibrium or vertigo on movement.

A fistula test, exerting pressure on the closed EAC with the finger or a pneumatic otoscope, will produce rotatory vertigo and nystagmus.

The problem may resolve with strict bed rest, head raised, and avoidance of exertion or postural situations that raise intracranial pressure. If symptoms persist or are severe, exploration of the middle ear, and sealing the site with soft tissue is undertaken. Relief from vertigo and improved hearing may be achieved.

8. IATROGENIC DAMAGE

Regrettably, all aspects of the ear may be damaged in the course of treatment.

Pinna

Unsatisfactory outcomes of pinna surgery may result from scarring, defects, or disfigurement. Correction of protruding auricles, if done poorly, causes irregular contours or atypical shapes. Trauma repair needs an expert touch to avoid unsightliness, particularly in haematoma auris or similar severe injury. Excisional surgery for malignancy in particular may demand skilled reconstruction to avoid poor outcomes.
External Canal

External canal and drum damage arises from several procedures.

Figure 13: Post-tympanoplasty mid-canal stenosis. Unsatisfactory EAC tissue management after the middle ear reconstruction.

Removal of exostoses is notorious for skin loss that may prolong healing and predispose to infection. This surgery and a range of tympanoplasty procedures may produce a canal stenosis, or blunting (thick scarring) of the anterior angle between the drum and EAC wall.

Careless drill technique during bony canal widening may damage the drum if not protected during the procedure.

Ossicular Chain

Chain trauma is less common as the surgeon is aware of the delicacy concerned. However incus dislocations are reported during stapedectomy, and stapes footplate subluxation or fracture is risked during cholesteatoma removal off this site.

Clearance of cholesteatoma off a fistula may tear the labyrinthine endosteum, exposing the perilymph to infection. Stapedectomy footplate surgery is legendary for associated inner ear trauma. Also deliberate trauma is incurred during labyrinthectomy or surgery for VIII nerve tumours.

9. RADIATION TRAUMA

Because of its relationship to structures such as the parotid or the postnasal space, the ear may be included in fields of radiotherapy. Regrettably, several severe side effects may result:

Irradiation of delicate EAC skin may devitalise this tissue rendering the relatively humid canal prone to persistent otitis externa. Meticulous serial suction toilet, wet mopping and gentamicin/ciprofloxacin drops are generally beneficial, but recurrent care may be needed.

Breakdown of the canal skin may render the canal denuded of epithelium over extended areas. Osteitis of the tympanic plate may result. This is difficult to manage, as necrotic bone spicules act as foreign bodies until extruded, which may take months, or linger indefinitely. Grafting the site is often ineffectual, given the devitalised vascular supply. Use of a non-irradiated vascular flap may be needed.

Inner Ear

Inner ear damage is a concern during cholesteatoma or mastoid surgery. Care must be taken to avoid drill contact with an intact chain.
Figure 14: Radiation-induced skin necrosis and exposure of the mastoid bone. Chronic and progressive necrosis of ear structures after radiation is notoriously difficult to correct.

Conductive deafness is frequent, due to effusions, chronic scarring, or infection. Drum breakdown is common, and subsequent recurrent infection is frequent. Sensorineural deafness commonly progresses after cessation of treatment, or accompanies concurrent chemotherapy. Aminoglycoside antibiotic treatment of immunosuppressed cases may contribute.