

TITLE: Trauma to the Middle and Inner Ear

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Injuries resulting from trauma to the middle and inner ear can cause minor temporary symptoms or permanent debilitation. Knowledge of the anatomy of the many fragile sensory structures within the middle and inner ear is vital to proper diagnosis and appropriate management of such injuries. Appropriate evaluation takes into account the spectrum of severity and the sometimes-unappreciated subtle symptoms of otologic trauma.

Anatomy

The tympanic membrane is an elliptically shaped structure creating the lateral border of the middle ear. It consists of three germinal layers: 1) outer keratinizing squamous epithelium from ectoderm, 2) middle fibrous layer from mesoderm, and 3) inner cuboidal mucosa from endoderm. It is composed of the large inferiorly located sound conducting pars tensa, and the smaller superiorly located pars flaccida. These two areas differ in structure and in function. The pars flaccida has a thicker epithelial layer and contains collagen within the middle layer, which allows for more flexibility, making it less susceptible to barotrauma. The pars tensa is taut compared to the pars flaccida. The thickened periphery sits firmly within the tympanic sulcus of the temporal bone to create the tympanic annulus. The average surface area of the tympanic membrane is 90mm², the effective vibratory portion consisting of the pars tensa averages 55mm².

The three ossicles of the middle ear (the malleus, incus, and stapes) bridge the gap between the tympanic membrane and the oval window while conducting sound as a lever system. The average area of the oval window being 3.2mm² gives a 17:1 (55/3.2) magnification. Likewise the handle of the malleus being 1.3 times the length of the incus contributes additional amplification of sound energy to a final amplification of 22:1. The ossicles are supported as a chain by the walls of the tympanic cavity. The malleus is firmly embedded with the tympanic membrane as well as being suspended by three suspensory ligaments, and a tendinous attachment. The stapes sits firmly within the oval window by the annular ligament and is further secured by the stapedial tendon. The incus, supported by two ligaments, is the weakest part and is the most likely to be sheared by external forces. The chorda tympani branch of the facial nerve traverses the middle ear between the malleus and incus.

The delicate sensory organs of the inner ear consist of the cochlea, vestibule, and semicircular canals housed within the petrous portion of the temporal bone. The semicircular canals are well hidden within the bony labyrinth, and their injury requires extreme disruption of the temporal bone. On the other hand, the vestibule and cochlea lie immediately medial to the middle ear, behind the TM. Penetrating trauma to the TM jeopardizes the auditory labyrinth by possible violation of the oval and round windows or the cochlear promontory.

Epidemiology

The TM is much more frequently traumatized than the middle or inner ear, but usually to a less serious degree. Annual incidence rates of traumatic perforations vary between 1.4 and 8.6 per 100,000. It occurs in all age groups with a predisposition for children likely due to their inquisitive nature and habit of placing foreign bodies into the external ear canal. Young men are more commonly found to have perforation injuries. Due to the rise in domestic violence, women have increasingly become victims of open-handed slap injuries with subsequent TM perforations.

Reliance on motorized transportation has greatly increased the risk of head injury. It has been estimated that 30-75% of blunt head trauma had associated temporal bone lesions. Gunshot wounds are an increasing source of temporal bone trauma. Increased mortality is more frequently seen in this group due to the high association with intracranial trauma. New laws governing the use of safety restraints and child seats for MVAs and head protection for motorcyclists have been very beneficial as forms of secondary prevention.

Etiology

TM perforations occur by various mechanisms and sources of energy, and therefore can be of many shapes and sizes. They are described in relation to the four quadrants of the TM as determined by the handle of the malleus. Size is normally described as a percentage perforation (ex. 40% perforation) or directly for smaller perforations (ex 2, 3, or 4mm perforation). Further classification of central versus marginal perforations is important for management.

1. Compression injuries:

Sudden changes in air pressure (blast or slap injuries) as well as gradual changes (barotrauma) can lead to significant TM damage. Blast injuries are more severe when there is less reflection or obstruction of the blast energy wave en route to the TM. Water skiing accidents are frequently seen during summer months. Changes in water pressure during the descent of SCUBA divers can lead to compression type injuries as well.

2. Penetrating injuries:

The second most common cause of TM perforations include Q-tips, bobby pins, keys, and paper clips often used in an attempt to clean the external ear canal.

3. Thermal injuries:

In industrial communities such as ours, hot welding slag is occasionally encountered as the culprit for TM injuries. Because of the tissue damage and

associated risk of infection, these are felt to be less amenable to observational treatment.

4. Lightning injuries:

The instantaneous electrical conduction of lightning strikes is thought to cause damage to the TM by either compression or rarefaction pressure changes. These injuries are also less likely to heal spontaneously.

Temporal bone trauma can be classified into blunt, penetrating, blast, and barotrauma. Blunt trauma to the skull occurs when there is a rapid collision between the head and a solid or semisolid object. The most common temporal bone fracture occurring from blunt trauma is the longitudinal fracture (80%). Directly applied lateral forces travel through the path of least resistance along the petrosquamous suture line and continues anterior to the otic capsule. This path usually involves major foramina in the skull base. The most common being the carotid artery and jugular bulb. The anterior extension may also include the temporomandibular joint. The most frequent structures involved are the tympanic membrane, the roof of the middle ear, and the anterior portion of the petrous apex. 15-20% will have involvement of the facial nerve, and injury occurs near the geniculate ganglion or in the horizontal portion. The facial paralysis is often delayed in onset, attributed to edema rather than direct interruption of the nerve. Vestibular involvement and sensorineural deficits are relatively uncommon and are attributed to concussive effects rather than direct trauma on the vestibular labyrinth and cochlea.

Twenty percent of temporal bone fractures are transverse in nature generated by forces in the anterior-posterior axis. These fractures often require much greater energy and are more commonly associated with more serious or even fatal head injuries. The facial nerve is involved in 50% of cases. The otic capsule and internal auditory canal are frequently involved as well.

Penetrating trauma usually resulting from firearms and violence is becoming more prominent. Unfortunately, their outcomes are more dismal. Victims may present with destructive lesions to the facial nerve, conductive hearing loss, TM perforation, ossicular disruption, labyrinthine fractures, and cochlear nerve transection. Intracranial injuries include laceration of the internal carotid artery, dura, or brain.

Barotrauma principally results from air travel or scuba diving. Rapid transient pressure fluctuations may jar the ossicular chain, and cause displacement of the stapes footplate, resulting in sensorineural hearing loss and vertigo. These same pressure events occurring on the oval or round window can lead to fluctuating auditory and vestibular symptoms known as inner ear barotraumata. Scuba divers descending beyond 30 feet must undergo decompression stages during ascent. Too rapid an ascent can cause percolation of nitrogen bubbles known as “the bends”, causing severe CNS or musculoskeletal dysfunction. Blast injuries occur by similar rapid pressure fluctuations yet cause more mechanical injuries.

Evaluation and Management

Along with an adequate history of the patient and the events of the injury, a thorough head and neck examination is necessary. The patient with multiple systems trauma must proceed

according to the ATLS protocol of emergency resuscitation. Immobilization or clearance of cervical spine injuries are immediately performed. The signs of middle and inner trauma can be very prominent. Evidence of a basilar skull fracture includes hemotympanum, Battle's sign, and periorbital ecchymosis. Blood in the external ear canal may be more representative of longitudinal versus transverse fractures of the temporal bone. Foreign bodies are more accountable for injuries to the TM, ossicles, facial nerve, or labyrinth without temporal bone fractures. The external ear canal may be lacerated in longitudinal fractures, whereas the transverse fracture will reveal hemotympanum. Pneumatic otoscopy may initiate the nystagmus and vertiginous symptoms of a perilymphatic fistula, or reveal a subtle fracture of the malleus.

Hearing Loss

Hearing loss is a common complaint after middle and inner ear trauma. Evaluation of hearing should be done with formal audiometry, however in the emergency room setting a tuning fork test should be enough preliminary data. Autophony may indicate a conductive hearing loss. 71% of patients with temporal bone trauma relate hearing loss. The type and degree of deficit is related to the force of injury and location of the fracture.

Transverse fractures involving the otic capsule and internal auditory canal frequently cause severe sensorineural hearing loss. Longitudinal fractures are more likely to cause conductive or mixed hearing loss. Even without temporal bone fractures, concussive injuries to the cochlea or labyrinth can cause hearing loss. In a review by Tos on the prognosis of hearing loss in temporal bone fractures, he found that 80% of conductive hearing losses from longitudinal fractures resolved spontaneously, while cases of sensorineural hearing loss due to transverse fractures showed no improvement.

TM perforations result in conductive hearing loss due to the loss of the effective vibratory portion of the TM and by the cancellation effects of sound waves reaching the remnant TM and oval window at nearly the same time. CHL greater than 40dB should alert the physician to the possibility of ossicular discontinuity.

Dizziness

The vestibular symptoms of vertigo or nausea and vomiting may be the result of a fracture through the otic capsule or a labyrinthine concussion. It is often times a late presentation due to the sedation, bed rest, and obtundation of this class of patients. In the majority of cases, the symptoms are temporary, and in injuries in which the lesion is permanent, recovery usually occurs as a result of compensation. Vestibular suppressants can be employed transiently for patients with extreme symptoms, however, they should be reduced or discontinued as soon as possible to minimize the suppression of central compensation. Benign Paroxysmal Positional Vertigo can often follow an episode of head trauma and can occur at any time following the injury.

Perilymphatic fistulas may present as fluctuating episodes of dizziness/vertigo with or without hearing loss lasting a few seconds. Tullio's phenomenon may be present. PLFs are initially treated conservatively as up to 40% should heal spontaneously. In certain cases in

which there is progressive hearing loss or persistent vertigo beyond an observation period of 10-14 days, surgical options may be considered. There is controversy about whether the presence of a perilymph fistula represents an emergent situation requiring surgical exploration because of the risk of severe sensorineural hearing loss, labyrinthitis, or meningitis.

The most common areas of fistulization are the oval and round windows, and therefore require elevation of a tympanomeatal flap and visualization. Suspected defects are repaired then plugged with fascia, muscle, or fat. Regardless of visualization of a specific leak site, the majority of patients achieve resolution of their symptoms.

CSF Otorrhea and Rhinorrhea

Temporal bone fractures account for the most common cause of CSF otorrhea. TM or external canal lacerations associated with longitudinal temporal bone fractures will allow CSF otorrhea, whereas in transverse fractures, the CSF may build behind an intact TM and eventually drain via the Eustachian tube. Because of the associated hemorrhage with traumatic lesions, the fluid is often not overtly characteristic of CSF. The test of choice for identification of CSF is confirmation of the beta-2-transferrin protein, and should be analyzed in any suspicious case. High resolution CT scan can help identify the site of CSF fistula. Severe fractures may also produce defects in the tegmen plate, predisposing the patient to meningocele or encephalocele development and delayed CSF leakage.

Sterile cotton should be placed within the external canal to prevent contamination. Measures to reduce intracranial pressure such as bed rest with head of bed elevation, stool softeners, no nose blowing, and lumbar drains are used. The use of prophylactic antibiotics is controversial. Otic antimicrobial solutions may only cause confusion in monitoring the CSF flow. The use of prophylactic antibiotics in CSF fistula is controversial, however, a meta-analysis by Brodie did reveal a significant reduction in meningitis when prophylactic antibiotics were applied compared to no antibiotics. The most common infecting organisms are Pneumococcus, Streptococcus, and Haemophilus influenza.

Conservative therapy is generally attempted for 7-10 days before surgical options are considered. The approach for surgical closure of CSF fistulas depend mainly on the status of hearing, presence of meningocele or encephalocele, and location of the fistula. Defects of the mastoid tegmen may require a transmastoid approach and plugging with fascial grafts. If brain herniation or significant defects are found, the middle cranial fossa approach may be justified. Tegmen tympani defects in a functional ear should be repaired through a middle fossa approach to preserve hearing.

Facial Nerve injuries

The facial nerve enters the temporal bone via the internal acoustic meatus. The nerve then travels 8-10mm within the anterosuperior quadrant of the internal auditory canal to the meatal foramen where the canal reaches its most narrow point (0.68mm). The labyrinthine segment then runs 2-4 mm to the geniculate ganglion where the greater superficial petrosal nerve exits to carry parasympathetic secretomotor fibers to the vidian nerve. The tympanic segment begins just distal to the geniculate ganglion where the nerve turns 40 to 80 degrees at the first

genu and runs posteroinferiorly across the tympanic cavity to the second genu. Here the stapedial muscle branch exits. The nerve then turns 90 degrees inferiorly where the mastoid segment travels for 12-14mm in the anterior mastoid to exit the stylomastoid foramen.

Early evaluation and a careful and thorough history when evaluating the status of the facial nerve is crucial. Particular attention should be given to the time and characteristics of onset of facial weakness, whether sudden or delayed, and determination of complete versus incomplete paralysis. The previous status of the nerve should also be documented. It is also important to determine if the paralysis is central or peripheral. Supranuclear or central lesions produce contralateral voluntary lower facial paralysis. The frontalis is spared due to the bilateral innervation. An incomplete paralysis is termed a paresis, and if there is no movement in the facial musculature, the paralysis is described as complete. Care must be taken not to misdiagnose a facial nerve paralysis as a paresis by attributing movement of the levator palpebrae superioris muscle of CN III. The House- Brackman grading system was designed to classify the long term degree of facial nerve deficit but is also useful to describe acute facial weakness.

	Grade	Characteristics
I	Normal	Normal facial function
II	Mild	Slight synkinesis, no asymmetry, slight weakness
III	Moderate	Complete eye closure, noticeable synkinesis, no asymmetry at rest, obvious weakness, slight forehead movement
IV	Moderately Severe	Incomplete eye closure, no asymmetry at rest, no forehead movement
V	Severe	Asymmetry at rest, barely perceptible motion
VI	Total	No movement

Temporal bone fractures are the most common cause of traumatic injury to the facial nerve. Fortunately, the facial nerve is robust and has shown a remarkable regenerative response to mechanical injury. As previously mentioned, the facial nerve is involved in 15-20% of longitudinal fractures and 50% of transverse fractures. High resolution CT scanning with axial and coronal images is the diagnostic tool of choice for evaluating the facial nerve in temporal bone trauma. Chang and Cass's review suggests that of longitudinal fractures; 43% had intraneural hematoma or contusion, 33% had bony impingement, 15% had transaction, and 12% had no identifiable pathology. In contrast, in transverse fractures, 92% had transection and 8% had bony impingement.

In a study by Haberkamp, Gadolinium enhanced MRI was found to be helpful in accurately predicting the site of facial nerve injury as a result of trauma. Likewise, MRI may play an important role in diagnosis and documentation of subclinical temporal lobe injuries, and other preexisting CNS injuries.

Electrophysiologic testing of the facial nerve includes the Nerve Excitability Test (NET), Maximal Stimulation Test (MST), Electroneuronography. These tests can only be used for unilateral paralysis because all three involve comparison to the contralateral side, which must be normal for valid results. Also, each will give normal results during the first 72 hours. The NET involves placement of a stimulating electrode over the stylomastoid foramen and measuring the lowest current necessary to produce a twitch on the affected side, which is then compared to the contralateral, normal side. A difference greater than 3.5mA indicates a poor prognosis for return of facial function. The MST is a modified NET. A maximal stimulus is used to depolarize all facial nerve branches and is compared to the contralateral side. ENoG is considered to be the most accurate prognostic test because it provides quantitative, objective measurement of neural degeneration. An electrode is placed near the stylomastoid foramen and a transcutaneous stimulus is applied. The muscular response is then measured using bipolar electrodes placed near the nasolabial groove. The peak-to-peak amplitude wave is then measured and compared to the contralateral side. A reduction of greater than 90% amplitude correlates with a poor prognosis for spontaneous recovery. A reduction of less than 90% gives an expected spontaneous rate of recovery of 80-100%. It should be noted that ENoG data is very well known for Bell's Palsy, however there is limited definitive ENoG data for facial nerve injuries due to trauma. Development of muscular degeneration fibrillations does not develop for 10-14 days, therefore making EMG of limited value in the early detection. However, diphasic or triphasic potentials indicate normal voluntary contraction. Polyphasic potentials indicate reinnervation, which develop 6-12 weeks before clinical return of function, which is useful in the evaluation of patients seen in the late post-traumatic period.

There is general consensus supporting the conservative treatment of patients with an incomplete paralysis. In an overview by Chang and Cass, it was concluded that surgical treatment was not required in patients who had 1) documented normal facial nerve function after injury regardless of its progression, 2) incomplete paralysis as long as there was no progression to complete paralysis, and 3) less than 95% degeneration by ENoG. Treatment of a complete paralysis is much more controversial, however. In 1974, Fisch recommended basing the decision for surgery on the time of onset of paresis, the degree of paresis, the degree and evolution of degeneration as measured by electroneurography, and the degree and evolution of regeneration. He noted a poor functional outcome in patients presenting with greater than 90% nerve degeneration by ENoG within 6 days of onset of palsy. Chang and Cass suggest that if decompression surgery is anticipated it should be done within a 14 day window from the time of injury based on animal studies by Yamamoto and Fisch. Despite the controversy, three general guidelines can be followed to select patients as surgical candidates:

- 1) Immediate paralysis with no evidence of clinical return after 1 week and absent electrical responses.
- 2) Immediate paralysis with significant disruption of the temporal bone on CT scan.
- 3) Immediate paralysis with progressive decline of electrical responses to less than 10% of responses on the normal side.

After deciding on facial nerve exploration, the suspect location of neural injury and hearing status are the two key factors in determining an appropriate approach. Injuries of the facial nerve at or distal to the geniculate ganglion can be approached via the transmastoid

procedure. Patients with transverse fractures are not candidates for this approach. Fractures can be identified laterally upon visualization of the mastoid cortex. These fractures can be chased medially to the point of injury. If there is no obvious fracture, a facial recess approach will help provide examination of the nerve from the geniculate ganglion to the second genu. Partial transections of less than 50% may be repaired with onlay nerve grafts. If transection exceeds 50%, an interposition nerve graft, such as the greater auricular nerve, should be used in approximation after the epineurium is trimmed and the nerve fascicles optimized. Of patients who undergo direct anastomosis or cable graft repair, the majority of patients (82%) will recover to a HB 3 or 4, and none have shown to recover to HB 1 or 2. It has not been shown whether early versus delayed repair leads to better functional outcome. If the nerve is found to be intact, decompression of the epineural sheath is performed in proximal and distal fashion until normal nerve is encountered. In Chang and Cass's review, about 50% of patients undergoing facial nerve decompression obtain excellent functional outcomes. Histopathological analysis of patients with severe facial nerve injuries has shown that retrograde axonal degeneration takes place to the level of the labyrinthine segment and possibly the meatal segment. If this stands true, lesions that are distal to the geniculate ganglion may not adequately be addressed by a transmastoid approach alone.

Injuries medial to the geniculate ganglion may be approached in several ways, depending on the status of hearing. For patients in whom hearing is not useful, a transmastoid-translabyrinthine approach is reserved. The entire intratemporal course of the facial nerve can be seen after translabyrinthine skeletonization of the internal auditory canal.

For patients with intact hearing, a transmastoid-supralabyrinthine approach or a middle cranial fossa approach is considered. Following complete mastoidectomy, the superior semicircular canal is skeletonized, thus allowing exposure of the labyrinthine portion of the facial nerve. If there are any concerns regarding adequate exposure or if grafting of the meatal portion is anticipated, the middle fossa approach is more suitable. The middle fossa approach is usually preceded by a mastoidectomy to aid in the identification of the internal auditory canal. The superior portion of the temporal bone is then exposed via an extradural craniotomy approach.

In summary, most lesions are of the perigeniculate and labyrinthine segments, and serious facial nerve injury may occur proximal to the fracture site. Therefore, during preoperative planning complete decompression of the nerve must be considered.

Iatrogenic Facial Nerve Injuries

Iatrogenic facial nerve injuries are rare but devastating complications of otologic surgery. The most common procedure resulting in facial nerve injury is Mastoidectomy (55%), followed by Tympanoplasty (14%), and removal of exostoses (14%). The region that is most commonly injured is the lower tympanic segment. Green found that 79% of injuries were not identified at the time of surgery. In patients with less than 50% transection, decompression was performed and 75% of the patients had a HB 3 or better. For lesions greater than 50%, direct anastomosis or cable graft was performed and no patients had better than a HB 3. Along with facial nerve injuries in temporal bone trauma, there is still much controversy regarding the management of

iatrogenic facial nerve injuries. However, it is generally agreed upon by otologic surgeons that an acute, complete, postoperative facial nerve paralysis should be surgically explored as soon as possible. It should be kept in mind that local anesthetic effects may mimic a mechanical surgical injury. For postoperative delayed onset weakness, serial electrophysiologic testing should be performed. If there is greater than 90% degeneration within one week, exploration is necessary.

Emergencies

Brain herniation and massive hemorrhage are two consequences of middle and inner ear trauma that are considered emergent and require rapid intervention. Brain herniation into the middle ear or the external canal requires patient stabilization and high resolution CT scan. Surgical repair of the defect as soon as possible is usually needed. If massive bleeding from the external auditory canal occurs, it should be immediately packed and carotid arteriography performed to determine the bleeding site. Embolization of the bleeding artery is usually the treatment of choice.

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