Injuries resulting from trauma to the temporal bone can cause minor temporary symptoms or permanent debilitation. Knowledge of the anatomy of the many fragile sensory structures within the temporal bone 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.

**Incidence and Epidemiology**

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. 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.

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 barotraumas. 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. Perilymphatic fistulas are thought to occur during the descent usually as a sequelae of Eustachian tube 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. Immobilizations of any 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.

HRCT is the most commonly used radiographic modality in evaluating the patient with temporal bone trauma. It has the advantage of being very fast and conveniently located in most E.R’s. Axial images can be processed immediately during the initial evaluation of these patients along with reformatted coronal images. Dedicated coronal views can be possible when it is known that the cervical spine is stable. 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.

Classification

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.

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 occur 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

The most common cause of CSF otorrhea is fracture of the temporal bone. Blunt trauma to the skull may produce fractures in the temporal bone with tearing of dura and foramina causing acute leakage. Fractures may also produce defects in the bony tegmen plate, predisposing one to
encephaloceles or meningoceles with resultant delayed CSF leakage. Temporal bone fractures have been traditionally divided into transverse or longitudinal, based on the relationship of the fracture line to the otic capsule and axis of the petrous ridge. In reality, however, most fractures are actually oblique in nature. An important factor in temporal bone fracture classification is whether the fracture passes through the otic capsule. Indications for surgical repair of and the approach for CSF fistula is significantly influenced by otic capsule violation. Tympanic membrane or EAC lacerations are frequently seen in longitudinal fractures which allows for the egress of CSF from the ear. However, with transverse fractures, the tympanic membrane is typically intact and the fluid may build within the middle ear and mastoid and eventually drain through the eustachian tube producing CSF rhinorrhea. CSF otorrhea in temporal bone fractures usually occurs within minutes of the accident but may be delayed in its presentation if it is draining through the nasopharynx. A high resolution CT scan can demonstrate the course of the fracture line and give information as to the likely site of CSF fistula. Accurate identification of CSF is important. After trauma, CSF otorrhea is typically serosanginous and can be mistaken for blood byproducts. The fluid should be sent for beta-2-transferrin, as this protein is highly specific to the CSF. Measurements of glucose and protein in the fluid have fallen out of favor for CSF identification. Bed rest with head elevation, stool softeners, and occasionally the use of lumbar drains is indicated. Sterile cotton should be used to prevent contamination of the ear. Antimicrobial ear drops are unnecessary and may actually confuse the clinician in regards to cessation of CSF flow.

In a study by Brodie and Thompson, 820 temporal bone fractures were treated over a 5 year period. There were 122 patients with CSF fistulae (97 with otorrhea, 16 with rhinorrhea, and 8 with both). Ninety-five of the patients had the fistulae close spontaneously within 7 days, 21 closed within 2 weeks, and only 5 had persistent drainage over 14 days. Only seven patients underwent surgery for repair of the CSF leak (middle cranial fossa, transmastoid, or combined). Nine of the 121 developed meningitis (7%). The use of prophylactic antibiotics was not statistically correlated with the development or prevention of meningitis in this study. A later meta-analysis by the same author, however, did reveal a statistically significant reduction in the incidence of meningitis with the use of prophylactic antibiotics.

The overall incidence of pediatric basilar skull fractures is lower than that of adults. The adult to child ratio is 10:1. This lower incidence is thought to be because of the increased skull flexibility and underdeveloped sinuses. The incidence of otorrhea is greater than rhinorrhea. Despite having a small number of cases, a meta-analysis showed that the use of prophylactic antibiotics did not influence the development of meningitis.

**Testing of Secretions**

Testing the fluid for the beta-2 transferrin is highly sensitive and specific for CSF. The amount of CSF required is extremely small. In an undiluted sample, as little as 1/50th of a drop is sufficient for analysis. In the past, the diagnosis was aided by the use of glucose and protein determination. The guidelines used in the past was that a definitive CSF leak exists when the glucose content exceeds 0.4 g/l and the protein content is from less than 1g/l up to a maximum of 2 g/l, but contamination of blood or wound secretions can confound the results.

The electronic nose is new technology that may be able to distinguish CSF from serum. Proponents of the electronic nose argue that it is faster and requires a smaller sample for analysis.
Further studies are needed to confirm these findings.

**Surgical Repair**

Surgical repair is recommended for those cases that do not resolve in order to prevent the morbidity and mortality associated with meningitis. Tegmen defects may be multiple rather than single, and identifying only one defect may not be sufficient for achieving definitive repair. Because surgical repair by way of a mastoidectomy approach alone can be inadequate if there are multiple tegmen defects, a middle fossa approach alone or in combination with a transmastoid approach should be considered in most cases.

The success rate is significantly higher for those patients who undergo primary closure with a multi-layer technique (using bone wax plus 2 additional materials, such as Oxycel cotton, muscle, fascia, or abdominal fat) versus those patients who underwent primary closure with a single-layer technique. The surgeon should also be prepared to deal with an encephalocele encountered incidentally during surgery. This herniated, pedunculated tissue is functionless and can be amputated. Closing off the external auditory canal and obliteration of the Eustachian tube and mastoid cavity may be indicated when CSF otorrhea has been refractory to medical and surgical interventions.

**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 labyrinthise 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.
<table>
<thead>
<tr>
<th>Grade</th>
<th>Characteristics</th>
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<tbody>
<tr>
<td>I</td>
<td>Normal facial function</td>
</tr>
<tr>
<td>II</td>
<td>Slight synkinesis, no asymmetry, slight weakness</td>
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<tr>
<td>III</td>
<td>Complete eye closure, noticeable synkinesis, no asymmetry at rest, obvious weakness, slight forehead movement</td>
</tr>
<tr>
<td>IV</td>
<td>Incomplete eye closure, no asymmetry at rest, no forehead movement</td>
</tr>
<tr>
<td>V</td>
<td>Asymmetry at rest, barely perceptible motion</td>
</tr>
<tr>
<td>VI</td>
<td>No movement</td>
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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. 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.

Electrophysiologic testing of the facial nerve includes the Nerve Excitability Test (NET), Maximal Stimulation Test (MST), and 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 reinervation, 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. Fractures can be identified laterally upon visualization of the mastoid cortex. Theses 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-supralabrinithine 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.
Bibliography


