Chapter 22

TEMPORAL BONE FRACTURES

CARLOS R. ESQUIVEL, MD,* AND NICHOLAS J. SCALZITTI, MD†

**Lieutenant Colonel (Retired), Medical Corps, US Air Force; Clinical Director, Department of Defense, Hearing Center of Excellence, Wilford Hall Ambulatory Surgical Center, 59th Medical Wing, Lackland Air Force Base, Texas 78236*

†Captain, Medical Corps, US Air Force; Resident Otolaryngologist, San Antonio Military Medical Center, 3551 Roger Brooke Drive, Joint Base San Antonio, Fort Sam Houston, Texas 78234

INTRODUCTION

The conflicts in Iraq and Afghanistan have resulted in large numbers of head and neck injuries to NATO (North Atlantic Treaty Organization) and Afghan service members. Improvised explosive devices, mortars, and suicide bombs are the weapons of choice during this conflict. The resultant injuries are high-velocity projectile injuries. Relatively little is known about the precise incidence and prevalence of isolated closed temporal bone fractures in theater. During the senior author's (CRE) last deployment (>500 clinical and surgical encounters), there were five cases of closed-skull base fractures. The current International Classification of Diseases (ICD)-9 coding system does not allow for documentation of isolated temporal bone fractures. A recent Department of Defense trauma theater registry data extraction using ICD-9 trauma codes for cranial/skull injury revealed a large number of service members sustaining both open and closed cranial injuries. In fact, during the last decade of war, service members suffered more than 1,065 closed-skull base fractures. The reported number of open skull base fractures was 490 injuries over the last decade. This data represent individuals who were transported from theater.

An innate instinct in humans leads to turning the eyes and face away from incoming impact or projectiles, which can often lead to trauma to the ear and side of the head. The outer ear and temporal bone can absorb the majority of these forces and limit damage to the deeper structures of the middle and inner ear, as well as the brain. Special arrangement of the outer and middle ear is essential for efficient capture and transduction of sound energy to the inner ear in order for hearing to take place. This efficiency relies on a functional anatomical relationship of a taut tympanic membrane connected to a mobile, intact ossicular chain. Traumatic forces that disrupt this relationship will alter the handling of sound energy along its pathway to the inner ear. This breach also allows for potential damage to the deeper, closed structures of the inner ear.

Consequences of trauma to the ear and temporal bone are not limited to functional deficits related to hearing, balance, and facial nerve function. An estimated lateral force of greater than 1,875 lbs is required to cause temporal bone fracture.[1] Obviously, this amount of force can often lead to injury to surrounding tissues. Damage to underlying intracranial structures can result in serious or life-threatening injuries that take precedence in initial management of the patient. Large series have shown that temporal bone fractures were associated with intracranial injuries in 56% of cases,[2] and death from related injuries was as high as 18%.[3] This chapter will discuss the evaluation and management decisions that such injuries pose. In addition, special considerations for war-related trauma will be addressed.

INITIAL EVALUATION

The initial evaluation of any trauma patient will require an assessment of the airway, breathing, and circulation, followed by appropriate trauma surveys. For this population of head trauma patients, the assessment of mental status and stabilization of the cervical spine will be paramount. Intracranial hemorrhage leading to elevated pressure will need to be excluded or managed appropriately. Observational studies have estimated that basilar skull fractures occur in 4% to 30% of head trauma patients, with 18% to 40% of these involving the temporal bone.[4]

For the otolaryngology consultant, evaluation will likely take place after completion of initial trauma survey and patient stabilization. A history of the traumatic event and a complete head and neck examination are the most important steps in the evaluation. Special attention should be given to the otoscopic examination. Debridement of the external ear canal may be necessary to establish the need for microwick placement and removal of possible debris. After examination, the tympanic membrane can be evaluated for perforation or drainage. When the patient's condition allows it, hearing evaluation with tuning forks is essential to determine the presence and type of hearing loss. The presence of clear fluid in the external canal in conjunction with a tympanic membrane perforation suggests a cerebrospinal fluid (CSF) leak. This can be confirmed by laboratory testing of the fluid. Particular attention to facial nerve function is essential during the initial evaluation of these patients. Keys to the appropriate management are the degree and timing of facial nerve dysfunction, which should be documented as soon as possible.

Certain physical findings should alert the physician to the possibility of temporal bone or otological trauma:

- otorrhea (bloody or clear),
- hemotympanum,
- tympanic membrane perforation,
- external auditory canal (EAC) laceration,
- facial weakness,
- acute vertigo, or
- hearing abnormality.

The presence of hemotympanum (Figure 22-1) has been found to have a positive predictive value of 86% for ipsilateral temporal bone fracture in one study.[2] Suspected or audiometrically confirmed sensorineural hearing loss (SNHL), vertigo, or nystagmus each had 100% positive predictive value in this series of 113 cases of suspected temporal bone fractures. Facial weakness was less predictive, with a positive predictive value of 72%. Traditionally, ecchymosis overlying the mastoid process, known as Battle's sign, has long been taught as a clinical marker of basilar skull fracture. One or a combination of these signs should lead to radiographic examination.

FRACTURE CLASSIFICATION

Historically, fractures of the temporal bone have been classified as longitudinal, transverse, or mixed in relation to the long axis of the petrous ridge. This classification system was developed based on postmortem and animal studies.[2,3,5] Longitudinal fractures are more common, accounting for 70% to 90% of fractures, whereas transverse fractures occur 10% to 30% of the time. Previous studies have documented the rate of facial nerve injury to be 10% to 25% in longitudinal fractures and 38% to 50% in transverse fractures.[3] Traditional teaching is that longitudinal fractures are the result of a blow to the head from a lateral or temporoparietal direction (Figure 22-2). They generally affect the EAC and the middle ear space. Alternatively, transverse fractures occur after a frontal or occipital blow to the head, and originate at the vestibular aqueduct (Figure 22-3). In the practical setting, categorizing a fracture as longitudinal or transverse is not always straightforward, and recent data have shown that this classification system does not accurately predict clinical outcomes.[3,5,6]

Several studies in the past two decades have developed and tested alternate methods for categorizing temporal bone fractures with respect to clinical significance.[3,5,6] In a study of 155 fractures in 132 patients, Ishman and Friedland[3] found that a classification system of petrous and nonpetrous fractures showed a statistically significant difference in the presence of CSF leak, facial nerve injury, and conductive hearing loss. Petrous fractures had a 10-fold greater risk of CSF leak (33.3% vs 3.6%) and a 3-fold greater risk of facial nerve injury (22.2% vs 7.2%) compared with nonpetrous fractures. However, nonpetrous fractures had a 55.6% incidence of conductive hearing loss (CHL) compared with 20% for petrous fractures. Comparatively, differences in these measures between longitudinal and transverse fracture patterns did not show statistical significance. Only the disparity between the groups

Figure 22-1. Hemotympanum sustained after a longitudinal temporal bone fracture.

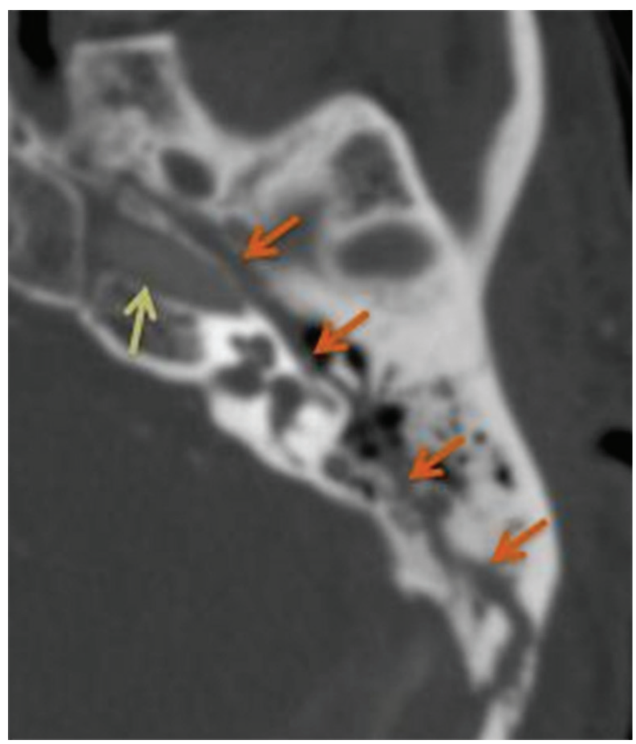

Figure 22-2. Longitudinal otic capsule-sparing temporal bone fracture. Longitudinal fracture line indicated by *orange arrows*. Carotid canal indicated by *yellow arrow*.

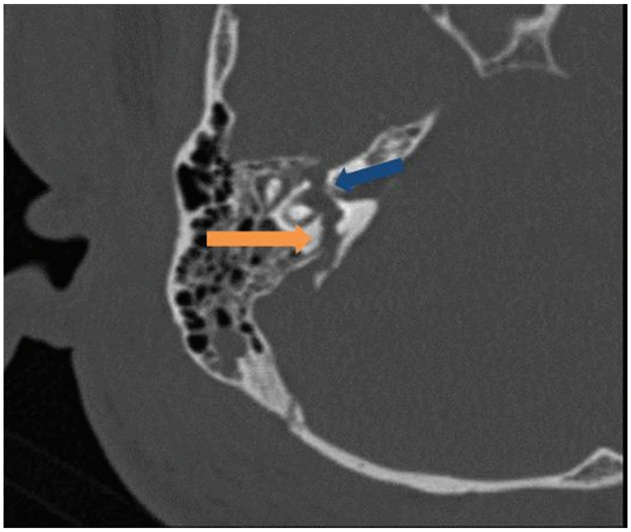

Figure 22-3. Right transverse fracture involving the otic capsule. First genu of the facial canal indicated by *blue arrow*. Transverse fracture line through the otic capsule indicated by the *orange arrow*.

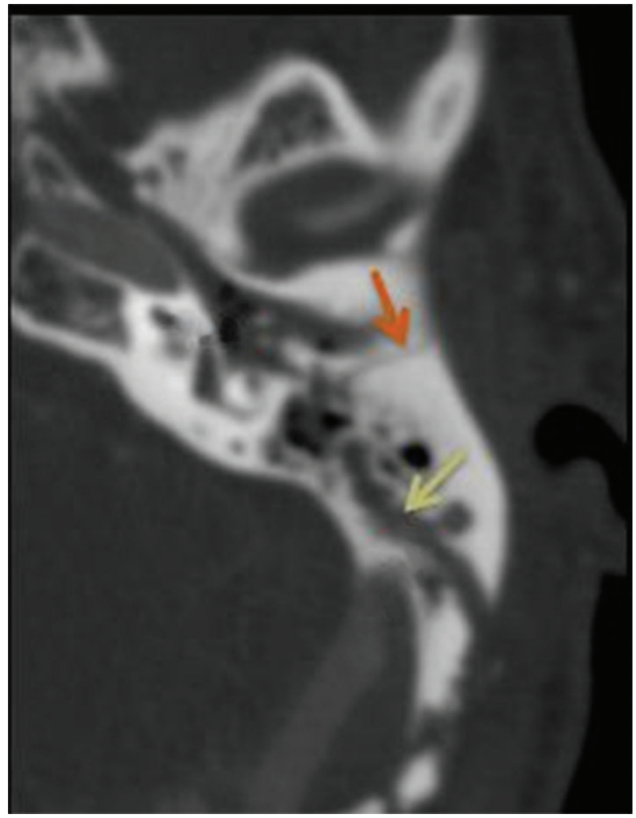

Figure 22-4. Mixed temporal bone fracture. Longitudinal fracture line indicated by the *yellow arrow*. Transverse fracture line indicated by the *orange arrow*.

with respect to SNHL was significant (50% of longitudinal vs 15.4% of transverse), which contradicted previous teachings that transverse fractures had a higher association with SNHL.

Another classification system, studied by Dahiya et al,[5] separated the fractures into otic capsule-sparing and otic capsule-violating fractures. This retrospective review of 82 temporal bone fractures showed a significant difference in complications when clinical and radiographic findings were correlated. Otic capsule-violating fractures were far less common, comprising only 5.6% of fractures. However, these fractures had a higher correlation with clinical complications, showing more than double the rate of facial nerve paralysis, four times the rate of CSF leak, and seven times the likelihood of severe SNHL compared with patients having otic capsule-sparing fractures. Little and Kesser[6] investigated this classification system again and compared it with the traditional system. They found a 5-fold increased rate of facial nerve paralysis in the otic capsule-violating fractures (67% vs 12%) and an 8-fold increase in the rate of the CSF leak. In their study, 100% of patients with an otic capsule-violating fracture had an SNHL compared with 4% in the otic capsule-sparing group. All of these differences proved to be statistically significant, whereas there was no finding of difference when the groups where classified using the traditional system of transverse, longitudinal, and oblique fractures.

Recent data suggest that newer classification schemes provide better correlation to clinical sequelae than the traditional system. Fractures of the temporal bone often have a more complex orientation than the two-dimensional system of longitudinal and transverse fractures can account for (Figure 22-4). A more useful classification is one that relates to involvement of the petrous portion of the bone or violation of the otic capsule. These features correlate with a much greater risk of facial nerve injury, CSF leak, and hearing loss.

RADIOGRAPHIC EVALUATION

Initial survey in these patients will undoubtedly lead to computed tomography (CT) of the head to assess for acute hemorrhage and displacement of the brain due to increased intracranial pressure. At many trauma centers, further examination is performed by obtaining a high-resolution CT (HRCT) of the temporal bones when physical examination or head CT is consistent with fracture of the temporal bone. Whether this HRCT is useful or necessary is a source of debate.

The temporal bone has a complex anatomy, with several intrinsic suture lines and channels that can often be misread as fractures on imaging studies. These "pseudofractures" represent fissures separating the five portions of the temporal bone from each other, as well as separations of the temporal bone from the sphenoid, occipital, and parietal bones.[7] Knowledge of these normal divisions is necessary for accurate interpretation of CT imaging after trauma. The tympanosquamous and tympanomastoid sutures parallel the anterior and posterior walls of the EAC, respectively. The tympanosquamous fissure divides into the petrosquamosal and the petrotympanic sutures as it traverses medially. Four suture lines separate the temporal bone from the surrounding bones of the skull. The squamosal suture separates the squamous portion from the parietal and sphenoid bones. The petrosal part of the temporal bone is divided from its neighbors by the sphenopetrosal and the petrooccipital sutures. The occipitomastoid suture, a continuation of the lambdoid suture, separates the mastoid process from the occipital bone.

Aside from suture lines, several channels in the temporal bone allowing for passage of various structures can also be confused with fractures. Also, these foramina and canals create natural areas of weakness for fractures to run to and through. The vestibular aqueduct courses from the posterior aspect of the vestibule inferomedially to the posterior aspect of the petrous bone. It contains the endolymphatic duct and terminates at the endolymphatic sac. The perilymphatic counterpart to this is the cochlear aqueduct that courses medially from the basal turn of the cochlea to empty into the subarachnoid CSF space. The most prominent nerve coursing through the temporal bone is the facial nerve. Its path is termed the Fallopian Canal, which is divided into the labyrinthine, tympanic, and mastoid segments from medially to laterally. The nerve enters the temporal bone from the anterosuperior quadrant of the internal auditory canal, lying above the cochlear nerve.

The question of when to obtain dedicated temporal bone HRCT continues to be debated. Kahn et al[2] reported from a survey at a Level 1 trauma center that two-thirds of otolaryngologists consulted for suspected temporal bone fracture either always or almost always obtained HRCT. One-third reported only obtaining the HRCT if certain sequelae occur or if the imaging is expected to change the management of the patient. This retrospective study of 105 patients with suspected temporal bone fractures explored the effect that HRCT had on the patients' management. Overall, the HRCT scan confirmed fractures in 79% of cases that had suspected fractures from either clinical examination or standard head CT imaging. Because most of these injuries were managed expectantly, they reported that HRCT scanning only complemented the decision-making process in 10% of cases. The HRCT was found to be necessary to identify the cause or site of facial nerve injury, prolonged CSF leak, or possible perilymphatic fistula in patients with the appropriate symptoms. This information was used in surgical planning for these cases. They concluded that HRCT showed utility in cases with an unexpected clinical course, when the clinical examination was unreliable, or for surgical planning in operative cases. Otherwise, a reliable clinical examination, coupled with standard head CT imaging, provided equivalent information in treatment decisions.

MANAGEMENT OF INJURIES

Facial Nerve Injury

An estimated 7% to 10% of patients with temporal bone trauma will display some level of facial nerve weakness (Figure 22-5).[8] For penetrating trauma, the rate of facial nerve injury is as high as 50%, and the degree of injury is often more severe than in blunt trauma.[9] The most revealing pieces of information regarding likelihood of recovery are the degree and timing of the dysfunction. Therefore, a reliable examination of facial nerve function is essential at the earliest possible time after injury. Unfortunately, this can often be delayed due to the altered mental status of the patient or the precedence of management being given to life-threatening injuries.

In general, if facial weakness (House-Brackmann [HB] grades II–V) does not progress to paralysis (HB grade VI), prognosis of recovery is good (Table 22-1). Care is usually supportive and immediately concerned with protection of the cornea and globe. These injuries represent neurapraxia (conduction block) or axonotmesis (disruption of axons), with an intact supporting sheath of the nerve fiber. On one hand, the scenario of axonotmesis allows for Wallerian degeneration distal to the injury followed by regeneration from the proximal site at a rate of 1 to 2 mm/day. On the other hand, immediate complete paralysis is consistent with neurotmesis (complete transaction of the nerve fiber), and surgical reanastomosis is warranted. Occasionally, this complete paralysis will develop progressively over the first week after injury, and the management decision becomes quite controversial.

A systematic review by Nash et al[10] of 612 cases of facial nerve injury following temporal bone trauma investigated how severity and timing of injury related to final facial nerve function. They determined that the

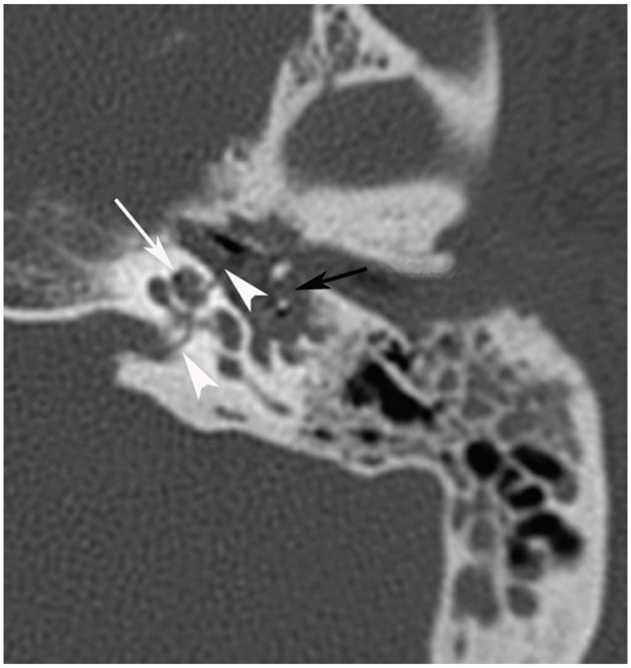

Figure 22-5. Transverse fracture traversing the bony facial canal. Air in the cochlea indicated by the *white arrow*. Transverse fracture through the cochlea indicated by the *white arrowheads*. Incudomalleolar joint dislocation indicated by the *black arrow*.

TABLE 22-1

HOUSE-BRACKMANN SCALE

Grade	Description	Characteristics
I	Normal	Normal facial function
II	Mild	Slight synkinesis / weakness
III	Moderate	Complete eye closure, noticeable synkinesis, slight forehead movement
IV	Moderately severe	Incomplete eye closure, symmetry at rest, no forehead movement, disfiguring synkinesis
V	Severe	Asymmetry at rest, barely noticeable motion
VI	Total paralysis	No movement

aggregate evidence for deciding for or against facial nerve decompression in blunt temporal bone injury was grade C, since data are comprised mostly of uncontrolled studies. Their results demonstrated that 66% of all patients returned to full facial function (HB grade I), regardless of timing (immediate or delayed) or initial severity (partial or complete). The group receiving steroids had a similar rate of full recovery (67%), whereas patients undergoing surgical decompression had a rate of 23%. To account for selection bias in the group undergoing surgery, subgroup analyses were performed.

For all patients who had complete paralysis, either immediate or delayed, the chance of full recovery decreased. Fifty-seven percent of the observation group had full recovery in this scenario, compared with 44% for those receiving steroids and 21% for those undergoing surgery. When the nerve showed only partial weakness (HB grades II–V), the chance of return to HB grade I status was higher for all treatment groups: 82% for the observation group, 95% for steroid treatment, and 57% for surgical decompression. Given the heterogeneity of the data, it was not possible to determine how these patients were selected for surgery.

Timing of complete paralysis has traditionally been considered paramount to the management decision, with immediate complete paralysis being an indication for surgical intervention. In this systematic review, complete paralysis was observed in 25 patients, with most having a final result of partial weakness, but no specific details were available. Most patients with immediate paralysis went on to surgery, resulting in 16% having complete recovery, 71% partial recovery, and 6% no recovery. The remaining patients had incomplete data sets to determine final outcome. For delayed complete paralysis, observation provided the highest rate of return (80%) to full recovery.

For incomplete or delayed paralysis, electrophysiological testing can provide useful prognostic information that may guide the management decision. The use of electroneuronography (ENoG) of the facial nerve has been extensively studied by Fisch.[11] Serial ENoGs have shown that after deliberate sectioning of the facial nerve, denervation of the muscles of facial expression is complete within 3 to 5 days. Alternatively, serial ENoGs following delayed facial paralysis from nonsectioning injury to the facial nerve demonstrated that Wallerian degeneration took between 14 and 21 days to develop. This data suggest that the rate of denervation is proportional to the severity of injury to the nerve. For these reasons, the timing of ENoG should be between 3 and 21 days of injury, with the goal of determining the degree of maximal denervation. When maximal denervation is less than 90% or if at least 10% of the facial muscles remain innervated, there is an 80% to 100% rate of recovery of function in 1 year. However, when denervation exceeds 90%, the rate of recovery is poor (60%–70%).

Electromyography (EMG) can also add information regarding the state of the nerve and motor endplate. Spontaneous and volitional EMGs provide useful information regarding nerve status. EMGs are of greatest prognostic value in patients with delayed paralysis and can be used for cases with bilateral paralysis. They require more sophisticated equipment, technical support, and training to read. Voluntary potentials equate to neural integrity, which means no transection and an intact motor endplate. Reassuring polyphasic regeneration potentials may be seen on spontaneous EMG after 21 days from injury and can be seen 8 to 12 weeks prior to clinical movement. Positive waves and fibrillation potentials are more ominous. EMG can be used to rule out fibrosis of the nerve–muscle interface that precludes nerve repair benefit in long-standing paralysis.

EMG is most useful in delayed paralysis because the different action potentials seen can provide insight. Voluntary potentials require an intact nerve and motor endplate unit. Polyphasic potentials are a sign of reinnervation and can precede clinical improvement by several weeks. Fibrillation potentials suggest denervation of the motor endplate and a poor recovery. EMG also has the advantage of utility in bilateral injury, while ENoG requires a comparison to the normal side.

Hato et al[12] performed a review of 66 patients who underwent surgical decompression after sustaining posttraumatic facial nerve injury. Patients were selected for surgery if they met the criteria of severe-to-complete paralysis (HB grades IV–VI), immediate onset, and more than 90% weakness shown on ENoG. Their results indicated that the ideal timing for decompression surgery was the first 2 weeks because this group had an 85.7% rate of complete recovery and a 92.9% rate of good recovery (HB grade I or II with minimal sequelae). For patients who could not undergo surgery in the first 2 weeks for whatever reason, there was also a significant advantage to performing surgery in the first 2 months (58.3% complete recovery, 77.1% good) compared with performing surgery after 2 months (16.7% complete recovery, 50.0% good).

Hearing Loss

Although hearing loss is likely the most common major complication of temporal bone fracture, it is safe to assume that the incidence of hearing loss is underestimated by published reports. Because it is a subjective symptom, its presence may not be discovered because of altered mental status. Also, hearing assessment is often deferred until more pressing issues are handled.

In a review of temporal bone fractures in 699 patients, Brodie and Thompson[13] found a 24% incidence

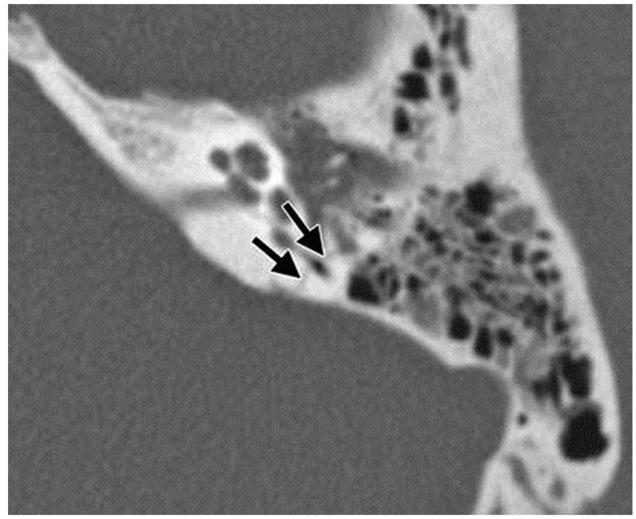

Figure 22-6. Axial CT (computed tomography) shows a transverse temporal bone fracture extending into the semicircular canals/inner ear. Transverse fracture through the labyrinth (*black arrows*).

of subjective hearing loss. For patients with audiometric data to classify the deficit, the loss was conductive in 21%, sensorineural in 57%, and mixed in the remaining 22%. Only 3% of patients developed a profound SNHL (Figure 22-6), which is consistent with other published data that have a range from 0% to 14%.[14] They reported that most patients with CHL either had spontaneous resolution or were lost to follow-up, with only five patients proceeding to ossicular chain reconstruction. In a different report, Yalciner et al[14] found that an initial incidence of CHL was 65%, in large part because of a high rate of hemotympanum (58.5%) and tympanic membrane perforation (25.6%). After routine 4- to 6-week follow-up, the rate of CHL was down to 12.3%, which likely represents timing of resolution of hemotympanum. If tympanic membrane perforation resolves, but CHL persists beyond 3 months, middle ear exploration with ossiculoplasty may be indicated. The most common finding in this case will be incudostapedial dislocation (11% to 14%), followed by incudomalleolar joint dislocation (Figure 22-7) and stapes fracture.[4]

SNHL does not lend itself to operative repair. Fractures of the otic capsule are the prevailing cause of SNHL after temporal bone trauma. Often, these fractures escape detection on clinical imaging, and postmortem histopathological examination has demonstrated microfractures of the otic capsule following temporal bone trauma.[15] This phenomenon, known as "cochlear concussion," may explain SNHL where no disruption of the otic capsule is seen on CT imag-

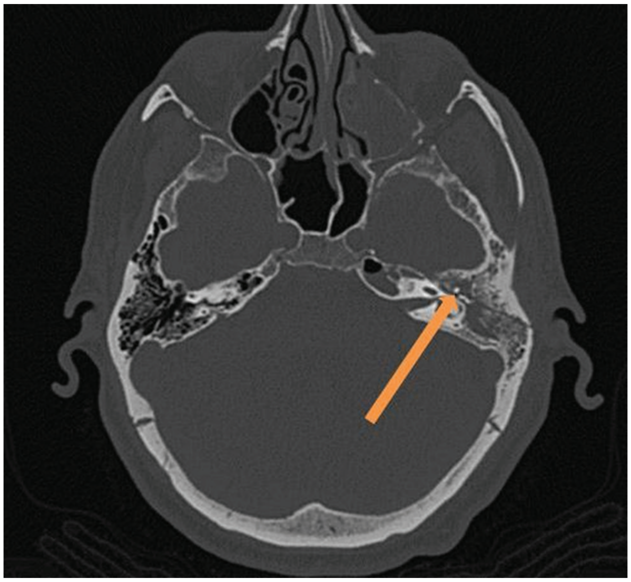

Figure 22-7. Axial CT (computed tomography) shows a longitudinal temporal bone fracture with incudomalleolar joint dislocation. Ossicular chain dislocation indicated by *orange arrow*.

ing. Auditory rehabilitation options are similar for patients with other causes of SNHL. Conventional hearing aids are the primary option when the residual hearing allows. For unilateral deafness, contralateral routing of sound and bone-anchored hearing aids provide the user with sound direction discrimination and elimination of the head shadow effect. Several authors have also investigated cochlear implantation in cases of bilateral severe-to-profound SNHL following temporal bone trauma. Although limited to small case series, these reports have shown success in auditory rehabilitation for patients when the fractures have not significantly disrupted the anatomy of the inner ear.[4]

Cerebrospinal Fluid Leak and Use of Antibiotic Therapy

The role of prophylactic antibiotics in the case of basilar skull fractures has long been debated. These fractures violate the bony barrier that exists between the middle ear space or the paranasal sinuses and the intracranial space. Often, the fracture can lacerate the dura mater layer of the meninges, creating a direct connection to the subarachnoid space and leading to a CSF fistula. This has been shown to occur in 15% to 20% of basilar skull fractures.[16] The fistula may not always be immediately obvious and can take days to manifest, often clear fluid drainage from the nose or ear. This leakage of CSF has previously been shown to increase the risk of meningitis after fractures of the skull base.[17] Certain circumstances do increase the risk of meningitis when a CSF leak is present. A well-defined factor is the duration of the leak, with a cutoff of 7 days having been studied in detail. Reported meningitis rates in patients with a leak for fewer than 7 days range from 5% to 11%, whereas leaks lasting longer than 7 days correlated with meningitis rates from 55% to 88%.[16] Other factors (eg, site of leak, delayed onset, and concurrent infection) have not been as well studied, and their role is largely unknown.

In any case, the benefit of antibiotics has not been clearly shown, even in the subset of patients that do leak CSF. In fact, certain studies have even shown an increased risk of meningitis in subjects receiving prophylactic antibiotics.[18] Two separate metaanalyses in the late 1990s produced conflicting data on this topic. In one study, Brodie[16] pooled six studies with a combined 324 patients and found a significant difference in meningitis occurrence in patients receiving antibiotics (2.5%) from those who did not (10%). A separate analysis by Villalobos et al[19] failed to show statistical significance for the risk of meningitis in the groups, independent of whether a CSF leak was present. Both of these analyses depended in large part on retrospective studies. More recently, a Cochrane review analyzed 208 patients from four randomized controlled trials.[20] The reviewers did not identify a difference in meningitis rates between the antibiotic treatment group and the control group. Interestingly, subgroup analysis for patients with and without CSF leakage also demonstrated no significant difference in the frequency of meningitis, although there was a tendency toward decreased rates of meningeal infection in the CSF leak group who received antibiotics. In their metaanalysis, antibiotic therapy did not alter the all-cause mortality or meningitis-related mortality for patients with basilar skull fractures. It should be noted that these studies represent only a small number of patients and that the outcome measure of meningitis is rare, making a statistical difference difficult to demonstrate. However, the current highest level of evidence does not support a clear recommendation for prophylactic antibiotics in basilar skull fracture, regardless of the presence or absence of a CSF fistula.

When CSF leakage does occur, the initial management should include conservative therapy with head-of-bed elevation, bed rest, stool softeners, avoidance of nose blowing, and straining. Placement of an indwelling lumbar drain provides a diverting drainage pathway to allow for spontaneous closure of the traumatic fistula. In published reports, these conservative measures have led to spontaneous resolution of the

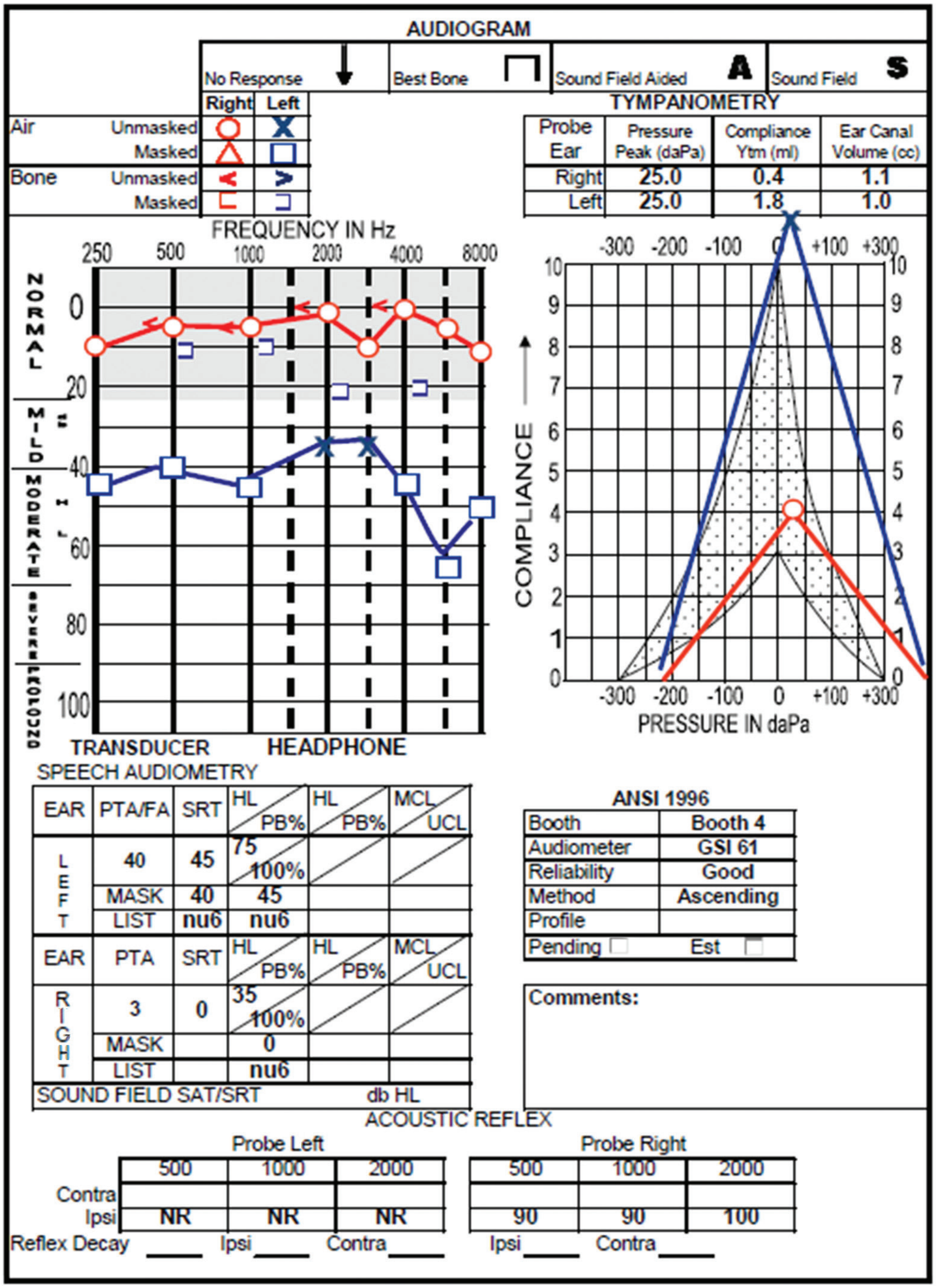

Figure 22-8. Audiogram displaying conductive hearing loss in left ear. Speech recognition threshold on left = 45 dB and on right = 0 dB.

ANSI: American National Standards Institute; Contra: contralateral acoustic reflex; daPa: dekapascal; db: decibel; Est: inconsequential abbreviation on this audiogram; GSI 61: refers to the Grason-Stadler audiometer used for this audiogram; HL: hearing level; Ipsi: ipsilateral acoustic reflex; MCL: most comfortable listening level; NR: no response; nu6: Northwestern University Auditory Test Number 6 word list; PB%: phonetically balanced percentage correct; PTA/FA: pure tone average; SAT: speech awareness threshold; SRT: speech reception threshold; UCL: uncomfortable listening level; Ytm: peak static acoustic admittance

CSF leak in more than 95% of cases.[4] In a large series, Brodie and Thompson[13] reported that 78% of leaks closed in the first 7 days and another 17% closed in the next 7 days. When the leak persisted beyond 7 days, the risk of meningitis did increase from 3% to 23%. Based on published data, surgical closure is indicated when a leak lasts longer than 7 to 10 days, and antibiotic prophylaxis for meningitis should be considered in the presence of CSF leak, especially if resolution does not occur in the first 7 days.

SPECIAL CONSIDERATIONS FOR WARTIME INJURIES

Acute Management

In the deployed setting, triage considerations guide management when many patients arrive critically wounded. Rarely will temporal bone trauma be an isolated injury, so the patient's prognosis will be determined by coexisting wounds. The initial focus will be on the principles of Advanced Trauma Life Support (ATLS). When the patient's stability is established, assessment of otological and temporal bone trauma can be made. For temporal bone injury, most treatment is not emergent and will be done outside of theater. These injuries should not delay a patient's transport to a higher level of care outside of the war zone. However, early evaluation should be performed to document deficits that may guide later treatment.

The initial evaluation should include a thorough facial nerve examination and evaluation with tuning forks to establish SNHL or CHL, if the patient is able. Attention should be given to the ear canal, which will often sustain a laceration from the shearing force of a fracture. Placing an expandable wick in the canal can stent it open and prevent stenosis. If no ear wicks are available, cotton fashioned into a long solid tube can also be placed. When at all possible, these should be done under clean conditions. If otorrhea exists, a sample can be collected to determine the presence of CSF by using a Beta-2 transferrin test (Quest Diagnostics, Madison, NJ). Open, contaminated wounds may require washout and debridement of devitalized tissue to prevent delayed infection. During cases of facial paralysis, attention should be given to protection of the cornea during transportation. Treatment of facial paralysis, CSF leak, hearing loss, and tissue loss or deformity can occur after evacuation from theater. Even in complete facial paralysis, published data suggest that decompression surgery can be delayed up to several weeks with excellent results.[12]

CASE PRESENTATIONS

Case Study 22-1

A service member sustained a blast injury and was ejected from the vehicle, resulting in soft-tissue injuries to the lower extremities and closed-head injury. He was evaluated in the ENT (ear-nose-throat) clinic after air evacuation. Initial assessment was an HB grade I on facial function. Ear canals were clear, and hearing was assessed with tuning forks. The Weber test lateralized to the left, and the Rinne test was negative at 512 Hz and 1,024 Hz on the left. An audiogram was obtained (Figure 22-8) that confirmed the tuning fork test, with a CHL of 35 to 40 dB in his left ear. CT temporal bones showed malleus and incus disruption (Figure 22-9). The service member underwent middle ear exploration with ossiculoplasty with good results (Figure 22-10).

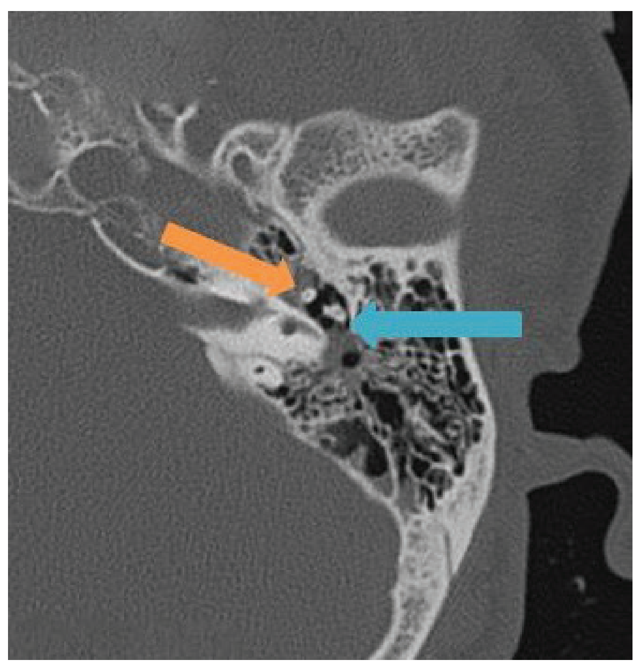

Figure 22-9. Axial CT (computed tomography) depicting incudomalleolar dislocation. The incus is indicated by the *orange arrow*. The malleus is indicated by the *blue arrow*.

Case Study 22-2

A service member was evaluated in the ENT clinic for hearing loss after a prolonged hospital stay (18 months). On initial evaluation, patient complaints were of mild intermittent drainage from the right ear, poor-to-no hearing in the right ear, and constant unsteadiness.

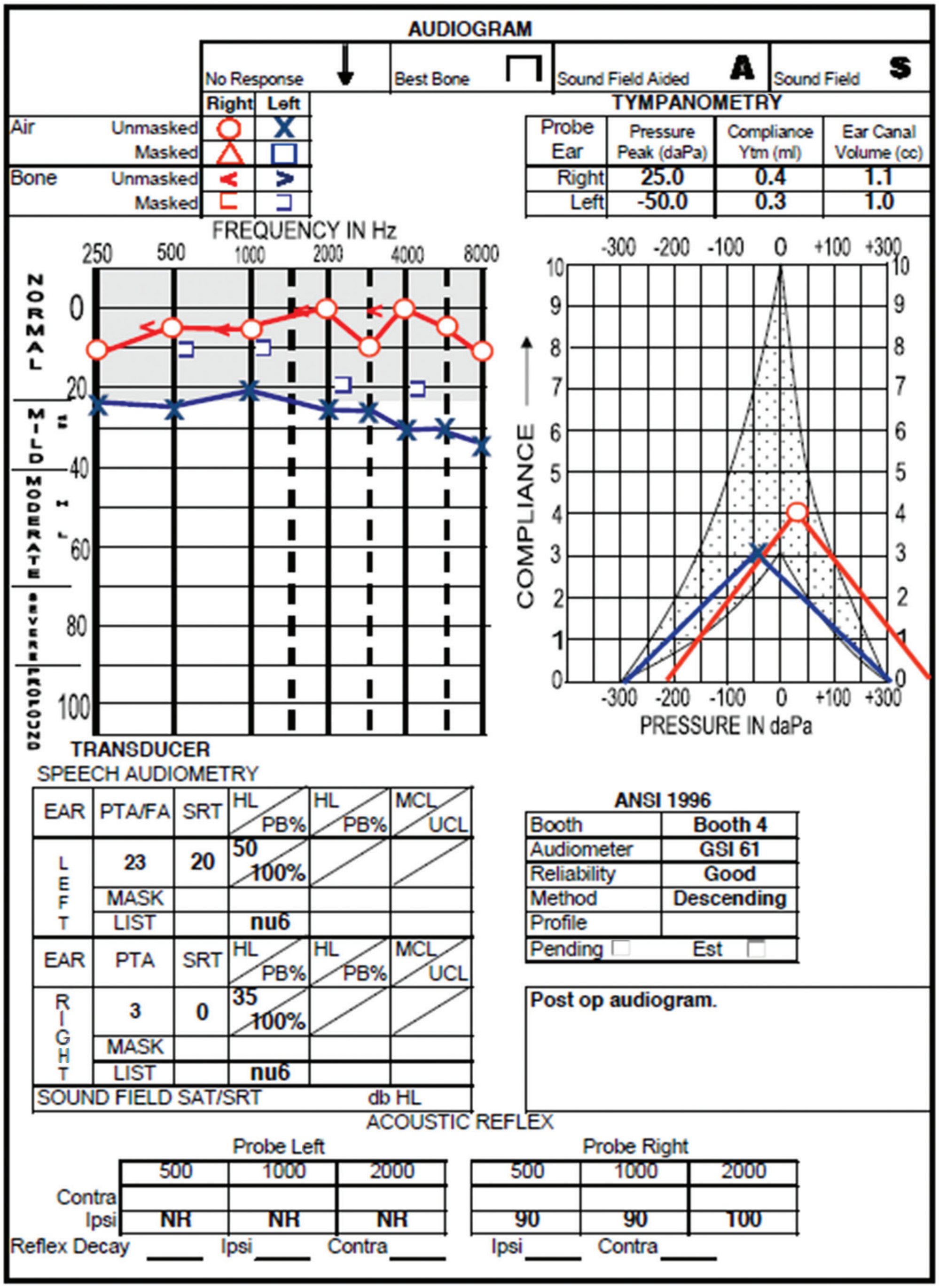

Figure 22-10. Postoperative audiogram. (*Left*) Note closure of air bone gap with improvement of speech recognition threshold to 20 dB.
ANSI: American National Standards Institute; Contra: contralateral acoustic reflex; daPa: dekapascal; db: decibel; Est: inconsequential abbreviation on this audiogram; GSI 61: refers to the Grason-Stadler audiometer used for this audiogram; HL: hearing level; Ipsi: ipsilateral acoustic reflex; MCL: most comfortable listening level; NR: no response; nu6: Northwestern University Auditory Test Number 6 word list; PB%: phonetically balanced percentage correct; Post op: postoperative; PTA/FA: pure tone average; SAT: speech awareness threshold; SRT: speech reception threshold; UCL: uncomfortable listening level; Ytm: peak static acoustic admittance

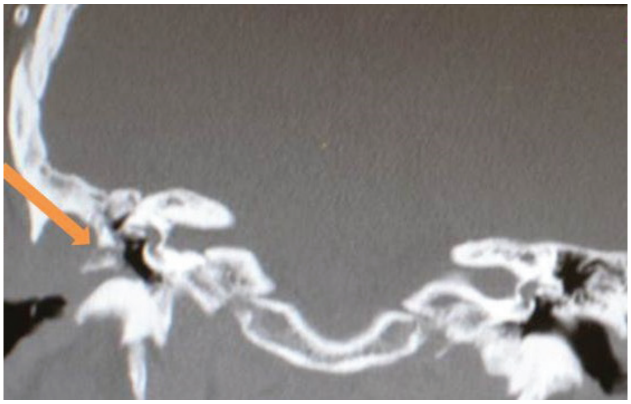

Figure 22-11. Comminuted right skull and bilateral temporal bone fracture resulting in right anacusis and posttraumatic cholesteatoma. Right temporal bone fracture line indicated by the *orange arrow*.

Examination of the right ear under microscopic view showed stenosis of the ear canal and a mass in the posterior-superior aspect of the tympanic membrane. An audiogram displayed profound SNHL in the right ear with poor speech discrimination. An HRCT was obtained (Figure 22-11), with the finding of a comminuted right skull base fracture and cholesteatoma. The patient elected to undergo a tympanomastoidectomy with placement of a bone-anchored hearing aide.

REFERENCES

1. Erbele ID, Sorensen MP, Rivera A. Otologic and temporal bone injuries, triage, and management. *Atlas Oral Maxillofac Surg Clin North Am*. 2013;21:117–125.

2. Kahn JB, Stewart MG, Diaz-Marchan PJ. Acute temporal bone trauma: utility of high-resolution computed tomography. *Am J Otol*. 2000;21:743–752.

3. Ishman SL, Friedland DR. Temporal bone fractures: traditional classification and clinical relevance. *Laryngoscope*. 2004;114:1734–1741.

4. Johnson F, Semaan MT, Megerian CA. Temporal bone fracture: evaluation and management in the modern era. *Otolaryngol Clin North Am*. 2008;41:587–618.

5. Dahiya R, Keller JD, Litofsky NS, et al. Temporal bone fractures: otic capsule sparing versus otic capsule violating clinical and radiographic considerations. *J Trauma*. 1999;47:1079–1083.

6. Little SC, Kesser BW. Radiographic classification of temporal bone fractures: clinical predictability using a new system. *Arch Otolaryngol Head Neck Surg*. 2006;132:1300–1304.

7. Collins JM, Krishnamoorthy AK, Kubal WS, Johnson MH, Poon CS. Multidetector CT of temporal bone fractures. *Semin Ultrasound CT MR*. 2012;33:418–431.

8. Chang CYJ, Cass SP. Management of facial nerve injury due to temporal bone trauma. *Am J Otol*. 1999;20:96–114.

9. Kilty S, Murphy PG. Penetrating temporal bone trauma. *J Trauma*. 2009;66:E39–E41.

10. Nash JJ, Friedland DR, Boorsma KJ, Rhee JS. Management and outcomes of facial paralysis from intratemporal blunt trauma: a systematic review. *Laryngoscope*. 2010;120:1397–1404.

11. Fisch U. Prognostic value of electrical tests in acute facial paralysis. *Am J Otol*. 1984;5:494–498.

12. Hato N, Nota J, Hakuba N, Gyo K, Yanagihara N. Facial nerve decompression surgery in patients with temporal bone trauma: analysis of 66 cases. *J Trauma*. 2011;71:1789–1792.

13. Brodie HA, Thompson HC. Management of complications from 820 temporal bone fractures. *Am J Otol*. 1997;18:188–197.

14. Yalciner G, Kutluhan A, Bozdemir K, Cetin H, Tarlak B, Bilgen AS. Temporal bone fractures: evaluation of 77 patients and a management algorithm. *Ulus Travma Acil Cerrahi Derg*. 2012;18:424–428.

15. Ohlrogge M, Francis HW. Temporal bone fracture. *Otol Neurotol*. 2004;25:195–196.

16. Brodie HA. Prophylactic antibiotics for posttraumatic cerebrospinal fluid fistulae. A meta-analysis. *Arch Otolaryngol Head Neck Surg*. 1997;123:749–752.

17. Leech PJ, Paterson A. Conservative and operative management for cerebrospinal-fluid leakage after closed head injury. *Lancet*. 1973;1:1013–1016.

18. Choi D, Spann R. Traumatic cerebrospinal fluid leakage: risk factors and the use of prophylactic antibiotics. *Br J Neurosurg*. 1996;10:571–575.

19. Villalobos T, Arango C, Kubilis P, Rathore M. Antibiotic prophylaxis after basilar skull fractures: a meta-analysis. *Clin Infect Dis*. 1998;27:364–369.

20. Ratilal BO, Costa J, Sampaio C, Pappamikail L. Antibiotic prophylaxis for preventing meningitis in patients with basilar skull fractures. *Cochrane Database Syst Rev*. 2011;August 10(8):CD004884.