

Hearing Impairment and Traumatic Brain Injury among Soldiers: Special Considerations for the Audiologist

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ABSTRACT

The increased use of explosive devices and mines in warfare and excessive noise of weapons has created an unprecedented amount of auditory dysfunction among soldiers. Blast-related injuries may damage the auditory processing and/or balance centers resulting in hearing loss, dizziness, tinnitus, and/or central auditory processing disorders. Some also lead to traumatic brain injury (TBI), postconcussive syndrome (PCS), and/or posttraumatic stress disorder. Some PCS symptoms such as dizziness, loss of balance, hearing difficulty, and noise sensitivity also can signify auditory or vestibular dysfunction and should not be obscured with the PCS package. This article provides information about the mechanisms of blast injury with emphasis on auditory dysfunction and TBI. Audiologists must be prepared to identify those at risk for TBI or mental health problems and adapt audiologic clinical practices to this population. An interdisciplinary comprehensive evaluation of peripheral, central, and vestibular components of the auditory system should be employed in patients with TBI to ensure that auditory dysfunction is accurately diagnosed and that appropriate rehabilitation can be performed.

KEYWORDS: Traumatic brain injury, auditory dysfunction, vestibular dysfunction, tinnitus, central auditory processing disorders, polytrauma

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Learning Outcomes: As a result of this activity, the participant will be able to (1) explain how blast injury can affect the auditory peripheral, central, and vestibular system, (2) describe common cognitive, emotional, behavioral, and physical problems associated with traumatic brain injury, and (3) describe resources for information regarding the therapeutic management of persons with traumatic brain injury and polytrauma.

The Global War on Terror (GWOT) has resulted in a new generation of combat service members with complex physical injuries, emotional trauma, and traumatic brain injury (TBI). TBI has become the signature wound of the war in Iraq, afflicting thousands of soldiers exposed to bomb blasts.¹ TBI and blast-related injuries may damage the auditory peripheral system, auditory cortex, and/or pathways that connect the ears to auditory processing and/or balance centers. These injuries may, therefore, result in hearing impairment, deafness, dizziness, vertigo, tinnitus, and/or central auditory processing disorders. Other areas of the brain also may be injured, such as sensory and motor areas and areas governing cognition, memory, executive function, self-awareness, and emotion. Brain injuries, even involving these nonauditory areas, could impact communicative capabilities. The high prevalence of hearing loss, tinnitus, and balance problems caused by acoustic trauma, blast trauma, and/or TBI in this growing population of returning soldiers stresses the need to develop and implement efficient strategies for their evaluation and management.²⁻⁴ Additionally, audiologists must gain new information to identify those with or at risk for TBI or mental health problems, work as an active team member with the patient with blast injury, and adapt audiologic clinical practices to this population.

As U.S. soldiers who have sustained blast injuries from explosive devices continue to return to military health care facilities and Veterans Affairs (VA) hospitals, clinicians across medical disciplines are learning to treat the patient as a whole by treating the mechanism of blast injury, not just the singular symptoms.⁵ Scott et al⁴ proposed a mechanism of injury as the preferred model for assessing and treating blast-related cases. An advantage of this model is its emphasis on the typical affected type of injuries given the mechanism of injury, which increases the likelihood that

related conditions to include auditory dysfunction will be identified. Undetected hearing loss can be masked by other conditions such as cognitive problems, communication problems, or posttraumatic stress disorder (PTSD). Audiologists need to learn about the mechanisms of blast injury with emphasis on auditory dysfunction and TBI and apply this information into their clinical practice by treating the whole patient, not just the singular symptoms of auditory dysfunction.

BLAST INJURIES

The current ongoing conflicts have seen a rapid increase in the incidence of blast injury, with 68% of combat injuries between 2003 and 2005 caused by explosions.⁶ In an explosion, solid or liquids are rapidly converted to gas form with a resultant release of energy. The detonation causes an extremely rapid buildup of pressure causing a blast wave that expands in all directions. A shock wave of overpressure travels through the air faster than the speed of sound, followed by a region of gas flow. This results in a short positive-pressure phase and a relatively long negative-pressure phase. Finally, air movement occurs, creating a blast wind. Surrounding structures can affect the pressure phases, either attenuating or, in a confined space, increasing the amount of damage.⁷

As the initial blast wave expands, surrounding air is compressed faster than thermal motion is able to disintegrate its individual molecules. As the distance from the center of the explosion increases, the blast wave gradually loses energy and diminishes into acoustic waves. The essential difference between a blast wave and an acoustic wave is in the shape, velocity, and magnitude of the pressure impulse. An acoustic wave usually has a sine-wave pattern and its wavelength is measured in decimeters, whereas the thickness of a blast wave is typically measured in millimeters. As the distance from the point of the explosion

increases, so does the proportional damage caused by the acoustic wave.⁷

Table 1 relates the mechanisms of blast injury to particular wounds. Injuries caused by explosion commonly fall into four categories: primary, secondary, tertiary, and quaternary.⁸ A fifth effect, quinary, has been recently added.⁹ Primary blast injuries are caused by barotrauma due to the blast wave itself. Secondary blast injuries are due to debris and bomb fragments being propelled by blast force. Tertiary blast injuries are due to impact of the body with another object. Quaternary blast injuries are blast-related injuries not due to primary, secondary, and tertiary mechanisms. Quinary blast injuries are from deliberately added agents such as chemical, biological, or nuclear agents.⁹ All four categories of blast injury can cause TBI and auditory and/or vestibular dysfunction.

TRAUMATIC BRAIN INJURY

TBI can result from physical damage by external blunt or penetrating trauma to the head, skull, dura, or brain. TBI also can result from acceleration-deceleration movement such as whip-lash or coup-contrecoup, resulting in tearing or shearing of nerve fibers and contusion of the brain against opposite sides of the skull. Scraping of the brain across the rough bony base of the skull can cause bruising and also can affect the olfactory, oculomotor, optic, and acoustic nerves leading to sensory problems. These symptoms may resolve over time and grow back to reinnervate the sensory receptors or muscles.¹⁰

TBI can be categorized by severity in three levels: mild, moderate, and severe. Mild TBI is defined as including associated diminished or altered state of or loss of consciousness (LOC), posttraumatic amnesia (PTA) for less than 24 hours, and a Glasgow Coma Scale (GCS) score* of 13 or greater quantifying level of consciousness.¹¹ With most TBIs, a set of postconcussive symptoms occur right after brain injury and can include cognitive deficits in memory and attention, fatigue, dizziness, headaches, irritability, anxiety, and depression.¹⁰

Mild TBI can cause cognitive deficits in speed of information processing, attention, and memory in the immediate postinjury period and in motor skills and new problem solving and general intellectual skills. Good recovery of postconcussive deficits can be expected over time from 1 to 3 months for most patients with mild TBI, although some patients may have symptoms for years or even develop postconcussive syndrome (PCS). Some post-TBI symptoms are similar to characteristic PTSD symptoms and can be a problematic overlap in considering PTSD incidence rates in those persons with mild TBI.^{10,12}

Moderate TBI is characterized by longer periods of LOC and PTA that follow the traumatic event. Patients with moderate TBI may experience a variety of symptoms, including physical, cognitive, and emotional problems that may persist for months or longer. Moderate TBI requires increased clinical management and rehabilitation but usually responds to medication, psychotherapy, and compensatory strategies.¹³

In severe TBI, a coma that lasts longer than 24 hours or PTA for longer than 7 days may follow the traumatic event. The GCS score can be used to describe the severity of TBI, with patients with severe TBI falling between 3 and 8 on the scale. Patients with severe TBI may remain in a vegetative or minimally responsive state for an extended length of time.¹¹ Severe TBI most often results in long-term problems with independent functioning and requires comprehensive medical care and ongoing rehabilitation.^{14,15}

The unique occurrence of the physical, cognitive, behavioral, and emotional sequelae involved with TBI requires audiologists to work in cooperation with many services. Severe TBI often occurs with other polytraumatic injuries that can complicate audiologic evaluation and management. Mild and moderate TBI as well as PTSD symptoms may not be immediately obvious. Dizziness, loss of balance, hearing complaints, and sensitivity to sound are potential symptoms of PCS, but they are also symptoms of otologic pathology. Thus, the audiologist is a

*The GCS is a neurological scale (scored between 3, indicating deep unconsciousness, to 15, indicating very minor injury) to record the conscious state of a person for initial as well as continuing assessment.

Table 1 Mechanisms of Blast Injury

Category	Characteristics	Body Part	Types of Injuries
Primary	Results from the impact of the overpressure wave with body surfaces	Gas-filled structures are most susceptible: lungs, gastrointestinal tract, middle ear	Blast lung, tympanic membrane rupture, middle ear damage, abdominal hemorrhage and perforation, concussion (TBI without physical signs of head injury)
Secondary	Results from flying debris and bomb fragments	Any body part	Penetrating ballistic or blunt injuries, eye penetration, fracture and traumatic amputation, closed and open brain injury
Tertiary	Results when bodies are thrown by blast wind	Any body part	Fracture and traumatic amputation, closed and open brain injury
Quaternary	Results from all explosion-related injuries not due to primary, secondary, and tertiary mechanisms	Any body part	Burns, crush injuries, closed and open brain injury, asthma, chronic obstructive pulmonary disease (from inhalation of dust, smoke, or toxic fumes), angina, hyperglycemia, hypertension
Quinary	Results from deliberately added toxic agents (e.g., chemical, biological, or nuclear agents) that are released in the explosion	Any body part	Range of injuries depends on particular agent that was added

Source: Adapted from Explosions and Blast Injuries. A Primer for Clinicians. Available at <http://www.bt.cdc.gov/masscasualties/explosions.asp>. Accessed December 2007.

vital team member of the interdisciplinary approach to management of the soldier with blast injury and TBI.

AUDITORY DYSFUNCTION IN SOLDIERS

Injuries sustained as a result of blast exposure, head trauma, or exposure to loud noises can result in hearing loss, tinnitus, and balance problems. Similar problems can be caused by the use of ototoxic medications during treatment of injuries, and there can even be a synergistic interaction between trauma and effects of noise exposure and ototoxic agents such as solvents and medication (see the article by Steyger,¹⁶ this issue). Injuries to the ear were the most common single injury type among Marines during Operation Iraqi Freedom (OIF) through 2004, accounting for 23% of all injuries.¹⁷ One third of soldiers returning from Operation Enduring Freedom (OEF)/OIF were referred to audiologists for hearing

assessments due to blast exposure, and of those referred, 72% were found to have hearing loss. Commonly, complex polytrauma patients have ear and balance deficits, which often are initially overlooked.^{2,6}

Forty-four percent of mild TBI referrals to a VA Polytrauma Network Support Clinic (described later in the VA Polytrauma Care Coordination section) complained of hearing difficulty but only 35% failed the hearing screening test. It is tempting to speculate that some patients who passed the hearing test yet complained of hearing difficulty had a central auditory processing disorder, which would not have been obvious through hearing screening.² It is also a possibility that some patients had a slight threshold shift compared with their enlistment audiogram. Patients with mild TBI often exhibit a hyperacute awareness of their problems. Conversely, patients with moderate and severe TBI may lack self-awareness and self-assessment and may deny a sensory deficit when one exists.¹⁸

It is therefore essential that peripheral and central auditory and vestibular function be examined in patients with TBI early on for possible therapeutic intervention and to facilitate any subsequent medical care and rehabilitation. This examination should be performed regardless of symptoms on the basis of blast exposure and/or TBI. Early identification and management of hearing loss, tinnitus, and vestibular impairment in these patients is essential to developing treatment strategies, ultimately leading to improved rehabilitative outcomes, reduced cognitive deficits, and improved quality of life.^{7,19}

Auditory Dysfunction from Blast Exposure

Explosive forces produce blast waves that endanger the auditory and vestibular systems both from exposure to the pressure wave itself and from the secondary and tertiary effects (see Table 1). Such forces and impacts are increased if the soldier was in a confined space without ear shielding. The most common otologic complaints immediately after a blast are otalgia, tinnitus, aural fullness, dizziness, loudness sensitivity, distorted hearing, and hearing impairment. These symptoms may last several minutes to several days or result in permanent deficits.²⁰

Even without a direct blow to the head, primary blast waves can have strong effects on the brain, resulting in concussions or mild TBI.²¹ Audiologic consequences of concussions include problems such as hearing loss, dizziness, and central deficits. Damage to the internal auditory canal, membranous labyrinth, and bony labyrinth, as well as temporal bone fractures can all cause loss of auditory or vestibular function. These injuries, along with petrous apex lesions, can be assessed through computed tomography scans and magnetic resonance imaging. All mechanisms of blast injury may also endanger auditory brain areas in the temporal lobe, corpus callosum, and thalamus.

To determine the degree of damage and the appropriate rehabilitation, it is important for patients with TBI and blast injury to undergo thorough evaluation by physicians, neuropsychologists, audiologists, otolaryngologists, physical therapists, speech language pathologists, and other related services. Because of

the likelihood of delayed onset of symptoms, long-term observation is important in all cases of blast-related auditory injuries.²⁰

Outer Ear Injury

Secondary blast effects are the most common cause of outer ear damage after exposure to bomb blasts. Burns, contaminated debris, and purulent otorrhea in the ear canal also have been observed.^{22,23} Many patients from the Kenya Embassy bombing in 1998 still had significant shrapnel extrusion from their facial wounds and debris (glass and concrete dust) in their ear canals at 5 months after injury.²³ Patients with burns are often treated with infection-controlling antibiotics and are at risk for ototoxic sensorineural hearing loss (SNHL). When the ear canal is full of contaminated debris, as may occur in those exposed to bomb blasts, a course of antibiotic eardrops should be started.²² Earlobe amputations and upper thoracic spine fractures were markers of critical injuries in severely wounded casualties of the March 11, 2004, Madrid terrorist bombing presenting to hospitals.²⁴

Middle Ear Injury

TYMPANIC MEMBRANE PERFORATION

Tympanic membrane (TM) perforation is the most common blast injury and accounts for 48% to 90% of all blast-related ear injuries.^{25,26} Clinical reports of incidence of TM rupture and ear infections after explosions vary dramatically, from 4 to 79%^{17,27,28} depending on factors such as type of explosive, distance from blast, confined or open environment, orientation of the ear to the blast, and if hearing protection was used. Depending on the frequency content and duration of the noise, TM ruptures can occur in up to 50% of adults at 5 lb/in.² (~185 dB peak pressure level [PPL]).^{29,30} Most other organs can withstand pressure gradients of up to 56 to 76 lb/in.²³¹ The improvised explosive devices (IEDs) causing injury in OEF/OIF produce pressures exceeding 60 lb/in.² resulting in a high likelihood of TM perforation in soldiers not wearing ear protection.⁶ The damage produced typically

occurs in the pars tensa and varies in appearance from linear tears to total defects.

Most TMs heal without surgical intervention, except in cases of resultant middle ear infection or when the blast causes total TM perforation. Treatment of TM perforation involves having the soldier avoid probing or irrigating the external auditory canal and submerging the head under water. Additionally, soldiers are prescribed ototopical antibiotics as indicated and referred for potential tympanoplasty if spontaneous healing does not occur. Small perforations usually heal within a few weeks.²² Table 2 summarizes some of the published reports of TM rupture, ear infection, and other auditory dysfunction due to explosions.^{2,7,17,23,27,28,32-39}

Blast effects on the TM such as radial laceration and often intratympanic hemorrhage⁷ are a clear indication that the patient has been exposed to PPLs far beyond those needed to damage the inner ear. TM rupture may protect the inner ear by reducing transmission of the potentially damaging sound energy.²³ Of 210 troops evaluated for TM perforation and LOC, those with TM perforations had a nearly 3-fold greater risk of concussive injury. The presence of a ruptured TM is sometimes used clinically as evidence that a patient has undergone a significant blast exposure, in which case further examination is indicated.²² However, an intact TM does not rule out serious injury.^{39,40}

CHOLESTEATOMA

TM rupture from blast is likely to result in small pieces of the squamous epithelium being blown into the middle ear cavity. If such fragments remain viable, they can grow into cholesteatomas, leading to infection, the erosion of ossicles, or even intracranial growths.²³ Kronenberg et al⁴¹ reported an 8% incidence of cholesteatomas from blast-related ear injuries. Of those, 3% were epithelial pearls found during tympanoplasty for persistent TM rupture at 11 to 18 months after injury, and 5% were invasive middle ear and mastoid cholesteatomas diagnosed 12 to 48 months after injury. Removal through careful surgical debridement by an otolaryngologist and close follow-up is important as 10 to 20% of chole-

teatomas recur. Conductive hearing loss and dizziness are two common symptoms of cholesteatomas. Cholesteatomas also can cause muscle weakness on the affected side, nerve damage, and deafness.

OSSICULAR DISRUPTION

If dynamic overpressures of the blast are high enough, the ossicles of the middle ear can be dislocated, distorted, fractured²² or can deteriorate.⁴² Depending on the force of the blast, ossicular damage can occur in 4 to 33% of cases where TM perforation is present.⁴³ Ossicular disruption may absorb some of the blast wave sparing the sensory structures of the cochlea.

Surgical repair of the ossicles restores much of the hearing lost at lower frequencies.⁴²

Inner Ear Injury

COCHLEAR INJURY

Blast exposure can subject the cochlea to such high pressure levels that the structural integrity of the inner and outer hair cells is disrupted.⁴⁰ Damage is then compounded by the following impulse sound.⁴³ When levels exceed 140 dB, metabolic disturbances in the inner ear are created that can severely impact the sensory and neuronal systems,⁴⁴ and mechanical forces begin to exceed the elastic compliance of the tissue as well. Extreme displacement of the basilar membrane damages the membrane and can tear sensory cells from support structures. Disruption of the oval or round window also can occur.²² Generally, sustained high-intensity noise causes more SNHL damage than does a single, very-high-intensity blast.⁴² Immediately after exposure to blast, soldiers may experience temporary hearing loss and tinnitus. For some, these symptoms are permanent deficits.^{7,40}

Different blasts produce different degrees and types of hearing loss, with prevalence estimates varying from 35 to 100% due to type of explosive, distance from blast, orientation of ear to blast, environment, and if ear protection was utilized.^{7,36,43,45,46} SNHL is most common,² occurring in 35 to 54% of blast injuries.^{7,23,33,47,48} For the current conflicts, 64% of the blast-injured patients seen at Walter

Table 2 Published Reports of Audiologic Dysfunction for Blast-Exposed Persons

Author	Blast-Exposed Persons	Blast-Related Auditory Dysfunction
Roth et al (1989) ³²	147 Israeli soldiers with TM perforations after blast; retrospective study between 1967 and 1986	74% SNHL or mixed hearing loss 60% tinnitus and/or vertigo 13% purulent discharge 8% cholesteatoma developed
Lucic (1995) ²⁷	49 wounded with explosive injury to the middle ear	79% TM perforation 16% fractured ossicle chain 5% paralysis of seventh cranial nerve 18% secondary infection 10% chronic otitis
Persaud et al (2003) ³³	17 patients from the Soho Nail Bomb in London, April 1999	62% TM perforation 100% mixed hearing loss (mainly SNHL) 88% tinnitus
Davis et al (2003) ³⁴	USS <i>Cole</i> explosion	14% TM perforation
Mrena et al (2004) ⁷	29 patients treated for ear trauma after suicide explosion at Finnish Shopping Mall, March 11, 2004	28% TM perforation 55% hearing loss (64% SNHL, 18% conductive, 18% mixed) 66% tinnitus 41% hearing loss and tinnitus 41% ear pain 18% dizziness 28% sound distortion or hyperacusis
Helling (2004) ²³	132 patients from the truck bombing next to the U.S. Embassy in Nairobi, Kenya, 1998	36% SNHL 23% TM perforation 6 of the 10 patients treated for TM perforation reported tinnitus
Teufert-Autry (2004) ³⁵	364 troops from OIF/OEF seen by ENT in 2003–2004 who experienced blast; seen at Landstuhl Regional Medical Center, West Germany	29% TM perforation 57% hearing loss and/or tinnitus 34% with hearing loss, majority with preexisting loss that had worsened since deployment 8% ear infections and conductive hearing loss 6% referred for complete balance evaluation 17% nonorganic hearing loss
Peral-Gutierrez de Ceballos et al (2005) ²⁸	243 patients from the terrorist bomb explosions in Madrid, Spain, 2004	67% of critically ill presented with ear-related blast injuries 41% TM perforation 1% ear amputation
Gondusky and Reiter (2005) ¹⁷	120 Marines injured in 32 IED attacks, March–August 2004	Ear injury was the most common single injury type (23%) 7% wounded in action had TM perforation
Xydakis et al (2005) ³⁶	980 WIA evacuated soldiers seen by ENT in Landstuhl during combat operations, 2003	TM perforation was the single most common otologic injury, accounting for 57% of ENT patients seen with head and neck injuries
Almogly et al (2006) ³⁷	154 patients injured as a result of 17 suicide attacks in Jerusalem from August 2001 to August 2004	22% TM perforation
Cave et al (2007) ³⁸	257 patients with blast-related injury seen at Walter Reed Army Audiology and Speech Center, April 2003 to August 2005	32% TM perforation 66% hearing loss (50% SNHL) 49% tinnitus 26% otalgia 15% dizziness

Table 2 (Continued)

Author	Blast-Exposed Persons	Blast-Related Auditory Dysfunction
Xydakis et al (2007) ³⁹	210 soldiers with blast injury who were evaluated consecutively for TM perforation and LOC October 1, 2005 to December 31, 2005 at Air Force Theater Hospital Iraq	35.2% TM perforation 35.7% had LOC 77.9% with no TM perforation had no LOC 60.8% with TM perforation had LOC
Lew et al (2007) ²	Record review of 42 inpatients with blast-related TBI seen at a PRC April 1, 2003 to July 31, 2006	62% hearing loss (SNHL most prevalent) 38% tinnitus

ENT, ear nose throat [doctor]; WIA, wounded in action.

Reed Army Medical Center in 2005 presented with hearing loss.⁶ For those who had ear shielding during the blast, such as headphones or earplugs, hearing was frequently normal. For those unprotected, however, hearing was often damaged. The audiometric configuration of blast exposure is quite different from the 4 kHz “noise notch” typical of noise exposure. Rather than a decrease in hearing sensitivity around 4 kHz, the signature audiogram of blast exposure is a sloping high-frequency hearing loss that often affects frequencies below 8 kHz.² Losses could be characterized as high-frequency hearing losses at one or more frequencies (most likely presentation) or flat configurations from mild to severe hearing loss. In rare cases, profound deafness occurred. A mixed hearing loss is often the initial diagnosis due to the combined effects of TM perforation and/or mucosal lacerations of the middle ear and mechanical injury to the sensory system. SNHL after a blast may respond to a brief course of steroids if there are no contraindications.²²

Drugs such as gentamicin or vancomycin are commonly used to treat soldiers with amputation and polytrauma to combat multidrug-resistant organisms. These drugs can be vestibulotoxic and/or cochleotoxic, causing instability and/or dizziness and hearing impairment.⁴⁹ Previous noise exposure appears to increase the risk of cochleotoxicity,⁵⁰ and noise exposure during ototoxic exposure amplifies the ototoxic effect (see the article by Steyger, this issue). High-frequency audiometry and otoacoustic emission testing are essential as an early detection and monitoring tool to accurately identify ototoxic-induced hearing changes.

Central Auditory System Damage

Whereas the presence of a ruptured TM is an obvious visible symptom of blast exposure, the most detrimental effects of the blast may be less visible and harder to manage. When an impact causes the brain to move within the skull to the extent that it impacts bone, contusions occur. Auditory processing areas of the temporal lobe are among the most commonly affected brain regions.⁵¹ The shearing, stretching, and/or angular forces that can be exerted on axons and small blood vessels by the blast wave or the motion of the brain within the skull can result in swelling and disconnection of axons.⁵¹ Once an axonal injury occurs, by whatever mechanism, there are fewer axonal connections throughout the brain, neurochemical insufficiencies, and/or less efficient electrophysiologic function of at least some of the axonal connections that are present.¹⁸

Although these factors encourage the idea that blast exposure may lead to central auditory damage, the prevalence of central auditory processing (CAP) disorders in TBI patients is not yet known.² The ability of clinicians to diagnose CAP disorders in the blast-exposed population is limited by a lack of relevant research findings in the literature. Most of the existing TBI literature pertains to injuries caused by weaker forces than those produced by explosive blasts. Compared with other causes of TBI, blast-related TBI may involve more extensive microinjuries and diffuse axonal injuries, and these injuries may result in diagnostic patterns and long-term prognoses that are distinct from other causes of TBI.

Tinnitus

Tinnitus can be defined as head or ear noise lasting at least 5 minutes.⁵²⁻⁵⁴ However, the typical tinnitus patient experiences tinnitus as a continuous or near-continuous symptom.⁴⁵ Tinnitus can be caused by anything that causes auditory damage.^{46,55} Excessive noise exposure is the most common cause of tinnitus,^{56,57} and when caused by noise, tinnitus is typically perceived as a high-pitched tonal or hissing sound.⁴⁵ However, because tinnitus has many causes, the audiologic approach to tinnitus should be interdisciplinary.

Blast-related tinnitus is emerging as a major problem among returning OEF/OIF veterans.⁶ For example, 58% of patients with blast injury at a Polytrauma Network Site complained of high-pitched tinnitus.² Blast-induced tinnitus often resolves. However, tinnitus persists in many patients, sometimes resulting in greater disability from the tinnitus than from the associated hearing loss.⁵⁸ Tinnitus and hearing loss due to blast or acoustic trauma are frequently developed under intense and stressful events with potential emotional sequelae.⁵⁹ According to Cave et al³⁸ the onset of tinnitus is pronounced to the sufferer when secondary to blast injury as compared with the gradual onset of tinnitus that accompanies progressive hearing loss. Tinnitus associated with a traumatic event might trigger traumatic memories.⁶⁰

A chart review of veterans seen over a 4-year period for tinnitus services at a VA medical center tinnitus clinic revealed that 34% of 300 patients also carried a diagnosis of PTSD. Reports of severe tinnitus and sound tolerance problems were more common for patients with a PTSD diagnosis than for patients with tinnitus only.⁶¹ Although tinnitus and PTSD involve distinct perceptual events, they may share many CNS mechanisms, particularly those composing the limbic system and auditory subcortical pathways. When present concurrently in an individual, their effects can be staggering, reducing the range of sounds that can be comfortably heard, exacerbating startle responses, and producing aversive and uncontrollable physical and emotional responses to sound.⁶¹ Therefore, it is critical that the severity and significance of

tinnitus is evaluated and effective management of tinnitus is provided for patients with blast injury, TBI, and/or PTSD.

Vestibular Injury

PERIPHERAL AND CENTRAL VESTIBULAR SYSTEM DAMAGE

Although blast injury often affects vestibular function, vestibular deficits are less common and the symptoms less defined than are auditory deficits sustained during an explosion. Some symptoms related to blasts and/or TBI are vertigo, dizziness, and balance disorders with benign paroxysmal positioning vertigo being the most common vestibular disorder associated with head trauma and/or blast injury. Vertigo as a result of blast injury is often associated with head trauma and PCS.²⁰ For the soldier with blast injury and TBI, it is possible that peripheral vestibular pathology, visual impairment, central pathology, peripheral neuropathies, musculoskeletal injuries, vascular disorders, certain medications, and proprioceptive changes all contribute to dizziness.^{62,63} With mild head injuries, this collection of factors results in rates of dizziness that range from 15 to 78% and is most often due to pathologies affecting the peripheral vestibular system, CNS, or cervical structures.^{64,65} In one of the few long-term studies on untreated patients with mild head trauma injury, vertigo persisted in 59% of patients after 5 years of recovery.⁶⁶ In soldiers with amputation, self-reported dizziness rates are 27% and self-reported vertigo rates are 18% after blast trauma.⁴⁹ Similar to findings in mild TBI, vestibular dysfunction appears to underlie postural instability after severe TBI.³

The pathophysiologic mechanism of trauma to the vestibular end organ is not fully understood. There are several theoretical mechanisms of pathophysiology that all accept the vulnerability of the semicircular canal epithelium to trauma.⁶⁴ Careful evaluation by the otolaryngologist is indicated to rule out perilymph leak as the cause of vertiginous symptoms.⁶⁵ TM injury can be complicated by perilymph fistula, which may be one of the most underdiagnosed

conditions in soldiers exposed to explosive blasts. Only with the knowledge of health care providers that they exist and that their symptoms can be obscured in a PCS package can patients be helped.⁶⁴

Evidence from acoustic trauma and vertigo/dizziness studies suggests that extremely intense noise exposure may cause vestibular disturbance⁶⁷ characterized by a momentary imbalance lasting for seconds or a minute or two. Noise exposure must be extremely intense, 140 dB or above, to have a major effect on vestibular function. Vestibular damage may be more likely to result from noise exposure for exposures that affect one ear to a greater extent than the other ear.

Effects of otologic blast injuries can include centrally mediated complaints that are less common than peripheral pathology in patients with head injury, including central vestibular pathology, peripheral vestibular pathology, or both, as a result of PCS or cerebral or brain-stem injuries.^{62,63} Head trauma to the brain stem and eighth nerve complex can lead to hemorrhages, often in the brain stem, with symptoms of acute vertigo, unsteadiness that is worse in the dark, and motion intolerance.⁶⁴ Toxic chemical inhalation (including paint and some industrial organic chemicals) also may have effects on the central nervous system, which may present as vertigo or unsteadiness.⁶⁷

For patients with blast injuries and TBI, vestibular pathology should always be considered. Loss of peripheral vestibular information creates greater dependence on visual and proprioceptive components of the sensory system. For the polytrauma patient with a visual field deficit and lower limb loss, this complicates the rehabilitation process particularly if they have uncompensated unilateral peripheral vestibular loss or bilateral vestibular loss. Unfortunately, patients with lower limb amputations may have vestibular problems that are overlooked.⁶

Although vestibular testing typically can determine the presence of a vestibular pathology, the cause of the vestibular disorder may remain unknown. In determining the cause of dizziness, it is important to work closely with the primary care physician to rule out non-vestibular causes of dizziness. Information

about orthostatic hypotension, cervical vertigo, PCS, TBI, ototoxic drug exposure, and visual impairment is needed to allow the audiologist, otolaryngologist, and physical therapist to more successfully determine if the dizziness is related to vestibular pathology. In addition, other common causes of vestibular disorders must be ruled out, including benign paroxysmal positioning vertigo, post-traumatic endolymphatic hydrops, labyrinthitis/vestibular neuritis and emotional factors.

AUDIOLOGIC ASSESSMENT AND MANAGEMENT

General Considerations

Often as a result of blast exposure and TBI, soldiers are increasingly suffering physical, cognitive, and mental injuries. Audiologists need to be mindful that for persons with TBI, auditory, vestibular, and other sensory stimuli must be processed by potentially damaged sensory systems and the distorted stimuli then perceived by the injured brain. The injured brain influences the way the patient thinks, feels, moves, speaks, behaves, perceives, and responds. Assessment and management of auditory and vestibular injury of soldiers with TBI, therefore, requires a multidisciplinary approach. The patient's type of auditory deficit and severity of TBI will largely determine diagnostic, treatment, and management options. Blast-exposed soldiers with hearing impairment may ultimately need to adjust to life with TBI, PTSD, prosthetic devices,⁶⁸ hearing aid(s), and/or assistive listening devices. The therapeutic care of soldiers exposed to blasts must consider the individual needs related to physical, cognitive, mental, or emotional injuries with common associated cognitive, behavioral, and mental problems discussed next.

Impairment in Memory, Attention, and Executive Function

The most fundamental cognitive problem seen in virtually all persons with TBI as a direct consequence of the diffuse axonal injury is an alteration in arousal and slowed processing

speed, difficulty with multitasking, and reduced cognitive stamina.¹⁸ Memory is considered a cornerstone of good cognitive function, and attention is the foundation of memory.⁶⁹ TBI often affects memory and attention systems, particularly in more severe injuries in which greater frontal and temporal damage exists. In cases of moderate and severe TBI with frontal injury present, deficits in executive function also are likely. The critical components of executive function include time management, anticipation, goal setting, planning, initiation, sequencing, error detection, and self-correction.^{69,70} Patients who have sustained a significant TBI may appear strikingly normal in a quiet, structured clinic or even a neuropsychology laboratory yet break down cognitively or behaviorally once they have to interact with the uncertainties and distractions of the real world.¹⁸

Because the ability to function in the real world and executive function may be impaired in patients with TBI, audiologists should provide cues to these patients, give real-world examples to go with new ideas and concepts, reduce visual and auditory distractions, give directions one step at a time, and provide rest breaks to these patients. The audiologist may need to frequently direct the patient's attention to task. It is also important to demonstrate and explain complex information in more than one modality and provide supplemental written information. It is helpful to write down appointments and important information on a calendar for the patient.

Behavioral Problems

Behavioral problems are common in persons with TBI, particularly when damage includes ventromedial and orbital frontal lesions.⁷¹ Behavioral problems such as impaired social judgment, agitation, and aggression may interfere with treatment plans at early stages of recovery.^{18,72} Additional chronic behavioral and neurologic conditions that can develop after TBI, particularly in persons with temporal lobe lesions, are intermittent explosive disorder and epilepsy.⁷³ Audiologists need to provide a calm, comfortable, and structured supportive environment to minimize behavioral outbursts.

Disorders of Self-awareness and Self-assessment

Persons with moderate and severe TBI typically lack insight of their own deficits; however, unawareness may not be universal after TBI, nor is it evidenced in a homogeneous manner across behavioral domains. Patients with TBI are generally good historians regarding their preinjury functioning, but collateral sources provide valuable information during assessment of postinjury functioning.⁷⁴ Neurologically based unawareness typically involves frontal and/or parietal lesions. This prevents the person with TBI from using strategies to compensate for their impaired functions.¹⁸ The audiologist thus may find discrepancies between the patient's behavioral postinjury report and objective audiologic test findings. It is important for the audiologist to include the family and medical team in the audiology case history taking and observations in real-world settings. The patient should be closely supervised to protect him or her from any consequences of lack of awareness (such as falling if vestibular dysfunction is present). In contrast with persons with moderate and severe TBI, persons with mild TBI usually exhibit a hyperacute awareness of their problems.¹⁸

Mental Disorders

Mood disorders and psychiatric disorders are common after TBI and may include depression, mania, bipolar disorder, psychosis, obsessive-compulsive disorder, and panic. Supportive psychotherapy, in addition to medication, can be used in patients whose cognitive status does not prevent it.^{18,75}

PTSD and PTSD-like symptoms can occur along with mild TBI and concussion.^{10,12} PTSD is an anxiety disorder comprising four major criteria: exposure to or witnessing a traumatic event; symptoms of reexperiencing or distress when reminded of aspects of the trauma; avoidance of thoughts, feelings, or reminders of the trauma, withdrawal, and emotional numbing; and an increase in arousal. To make a diagnosis of PTSD, these symptoms must cause marked impairment in functioning and persist for at least 1 month after the trauma.⁷⁶ Estimates of the prevalence rates of

PTSD after TBI vary dramatically, from 0 to 50%, depending on the study.⁷⁷

It is important for audiologists to be aware of the associations between TBI and mental health problems for a variety of reasons: to improve the recognition of problems when they occur, to enhance understanding and empathy for difficult patients, to identify reasons for noncompliance, and to justify screening and/or clinical referral for further evaluation and treatment of PTSD and/or other emotional sequelae.¹⁰ It is helpful to display posters and have handouts on PTSD and signs of depression in the audiology clinic to increase awareness of mental health issues of veterans among patients and their families and to provide information about how to seek help.

Dual Sensory Disorders

The senses are the first step in cognition and allow one to monitor and process data in the environment. TBI often results in visuospatial processing problems.⁶⁹ The rate of visual impairment in blast-related injury at a VA Polytrauma Rehabilitation Center (PRC) was 52% compared with 20% for all other sources of injury.⁷⁸

TBI may damage the visual cortex and/or visual pathways that connect the eyes to the cortical visual centers. Lew et al⁷⁹ performed electrophysiologic P300 testing in the auditory and visual modalities of 11 patients with TBI and 11 control subjects and found that TBI patients had lower P300 amplitude in the auditory and visual domains and longer P300 latencies in the auditory and visual domains. The diminished amplitudes and prolonged latencies of the patients with TBI suggested impaired organization and categorization of incoming stimuli. The patients with TBI had longer reaction times in both the auditory and visual domains suggestive of slowing in the response executive process.

Vision plays a key role in audiologic rehabilitation because many related activities (e.g., ambulation, orientation, reading, speechreading, watching facial expressions and gestures, taking care of hearing aids and assistive devices) depend on sensory input from the visual system.⁷⁸ Visual impairment and visual-field

deficits must be taken into consideration when providing aural rehabilitation and amplification device instructions and vestibular evaluation and management. Specifically, the audiologist must be aware of the patient's field of vision and use of glasses, magnifiers, or other visual aids. Additionally, patient education materials should be written in large font and accompanied by audiotapes when appropriate. Audiologists can assist these patients by working with the vision team and being mindful about audiologic clinical practices in which vision plays a role.

AUDIOLOGIC DIAGNOSIS

Audiologic diagnosis of a polytrauma patient is often difficult to perform in a timely manner due to variations in level of consciousness, contraindications to testing (such as c-spine precaution, isolation, ventilator, bed rest orders, pain), and multiple, higher priority appointments at other clinics.⁶ Most polytrauma patients are seen for an audiologic evaluation within 48 hours of consult at the VA PRCs. Audiologic evaluation protocols are commonly modified based on the complexity of the injury. Consequently, the diagnosis of auditory and vestibular disorders with patients with blast injury and polytraumatic brain injury requires innovation and extrapolation from the standard methods described in the literature. The diagnostic protocol described in this section is recommended with the understanding that ongoing clinical research is still needed to identify the most sensitive tests to assess persons with blast injury and TBI. Appendix A contains an audiologic report template highlighting a clinic examination protocol developed at a VA PRC. Researchers and clinical audiologists should use existing evidence-based literature on TBI and CAP disorders and expand and refine this PRC protocol to tailor audiologic assessment and management protocols for persons with blast-related TBI.

Patient Questionnaires

Polytrauma patients should complete an audiologic case-history questionnaire and standardized questionnaires that assess the impact of hearing loss, tinnitus, and/or dizziness. Some appropriate questionnaires are the Hearing

Handicap Inventory for Adults (HHIA),⁸⁰ the Tinnitus Handicap Inventory (THI),⁸¹ the Dizziness Handicap Inventory (DHI),⁸² and/or the Vestibular Disorders Activities of Daily Living Scale (VDAL).⁸³ Collateral information from significant others after injury should be obtained because (as described above) patients with TBI may lack self-awareness and self-assessment of their problems or exhibit a hyperacute awareness of their problems.¹⁸ Further, patients with TBI are generally good historians regarding their preinjury functioning, but collateral sources provide valuable information during assessment of postinjury functioning.⁷⁴

Patient case-history questionnaires should elicit both medical history and detailed information about the nature of the blast. Items should include preblast and postblast cognitive functioning and visual, audiologic, dizziness, and tinnitus history. Information about the blast exposure should focus on the details such as the nature of the blast, the proximity to the blast, and the use of helmets, hearing and eye protection, and LOC at time of injury.

Screenings for PTSD and mild TBI should be administered when appropriate. A national clinical reminder, VA-TBI screening was built for VA hospital clinicians to screen all OEF and OIF veterans receiving medical care within the Veterans Health Administration and to offer further evaluation and treatment to those who screen positive.⁸⁴

Comprehensive Audiometric Evaluation

If otoscopy reveals occluding cerumen or abnormal pathology, then a referral to otolaryngology is required. When possible, monaural air- and bone-conduction threshold measurements should be obtained to determine the type and degree of peripheral hearing impairment. Because of the possibility of hearing impairment from ototoxic medications, which are frequently used in blast-injury cases, hearing thresholds should be measured at frequencies up to 12,000 Hz or to the frequency limits of the individual's hearing.⁸⁵ Speech recognition should be tested monaurally in quiet and in noise, using sentences and one-syllable words. Immittance audiometry and acoustic reflex decay should be completed in each ear. If

pure tone testing reveals a conductive or mixed hearing loss and/or tympanometry results are abnormal, then the patient should be referred to otolaryngology for evaluation. The use of otoacoustic emissions should be conducted to confirm other test findings. When conventional behavioral audiometry is not possible, objective auditory brain-stem response (ABR) and auditory steady-state response (ASSR) testing should be performed.

Ototoxicity Monitoring

For patients on ototoxic medications, baseline testing should be comprehensive to provide information about pretreatment hearing levels using conventional and high frequency audiometry upon initial exam and be monitored serially for change in auditory sensitivity on a five-day cycle throughout antibiotic therapy. These patients should be educated to avoid loud noises and/or to use ear protection when exposed to loud noises. In setting up ototoxicity monitoring protocols at military treatment facilities and VA hospitals, there are questions brought up by physicians, pharmacy, infection control, and audiology service that should be addressed at each site with the medical team. For example, issues discussed at a PRC have included: What if there is no baseline test to compare with? What if the patient is on other concomitant medications or antibiotics? It is not uncommon to have patients on an aminoglycoside, vancomycin, metronidazole, a diuretic, and a nonsteroidal anti-inflammatory drug, so is there a validated evidence for a threshold at which point you would absolutely not use the agent (stop it and never use it again)? In many situations, patients are septic (requiring immediate therapy), are in the unit and/or in isolation (and unable to be transferred to audiology for testing). Ototoxicity monitoring protocols will need to be modified as warranted via a team approach. Detailed guidelines on ototoxicity monitoring are available elsewhere.⁸⁶

Assessment and Treatment of the Vestibular System

If the patient reports any problems with dizziness or unsteadiness, then vestibular functioning

should be assessed. There is no universal accepted approach for assessing balance in patients with mild TBI.³ The assessment should be done using an interdisciplinary team approach given the number of different causes that may be contributing to the symptoms. Balance is a multisensory function, thus, evaluations are not sensitive site-of-lesion tests. Dizziness can be difficult to diagnose because there are many causes of TBI that can likely involve peripheral, proprioceptive, central visual/vestibular, oculomotor, and multimodality processing.

Clinicians working with soldiers with blast trauma and TBI need to consider possible causes of postural instability previously described. Vestibular screening by physical therapists in the blast evaluation usually includes a cervical range-of-motion and cervicalgia assessment, oculomotor evaluation, postural stability, gait assessments, and vertebral artery test to assess for potential vertebral insufficiency and vascular causes of dizziness. The passive dynamic visual acuity test and head thrust test performed bilaterally assess the vestibular ocular reflex. Contraindications to this screening include cervical instability, fractures precluding cervical range-of-motion assessment, and medical equipment that precludes communication such as jugular line or ventilator in place.⁴⁹ Screening tests are not sensitive enough to detect all vestibular abnormalities nor do they provide quantitative data to determine the presence or absence of vestibular lesions. If signs of possible vestibular pathology are present, further evaluation is recommended, provided that there are no medical contraindications. A patient may have to undergo one or more of the following inner ear balance tests to include videonystagmography, electronystagmography including the Dix-Hallpike maneuver, rotational test, vestibular evoked myogenic potentials, computerized dynamic posturography, computerized dynamic visual acuity test, subjective visual vertical test, and other physical screens in the audiology and/or physical therapy clinic.^{3,49,87,88} These tests can help to characterize the deficits a patient might have and help with management.

Treatment of vestibular dysfunction should be patient and deficit specific and provided in collaboration with both physicians and phys-

ical therapists. Depending on diagnostic test results, a variety of vestibular treatment options may be used including vestibular rehabilitation therapy, physical therapy, postural stability training, gait training, canalith repositioning therapy, change in activity levels, medication, change in diet, treatment for any underlying disease that may be contributing to the balance disorder (such as eye glasses, infection, etc.), patient centered and directed counseling, lifestyle and compensatory strategies counseling, or surgery. Otolith disorders are more difficult to treat as little is known regarding the adaptation process that occurs following otolith damage. Finally, monitoring the patient with TBI and vestibular symptoms is essential via incorporating outcome questionnaires, physical screens, repeating tests that had initial positive findings, and assessing and modifying management strategies on an ongoing basis.

Assessment and Management of CAP Disorders

Deficits in CAP refer to difficulties in “the perceptual processing of auditory information in the central nervous system”⁸⁹ as demonstrated by poor performance in one or more of the following skills: (1) auditory performance in the presence of competing acoustic signals (e.g., dichotic listening); (2) temporal aspects of audition (e.g., temporal integration, temporal discrimination, temporal ordering, temporal masking); (3) auditory pattern recognition; (4) auditory discrimination; (5) auditory performance with degraded acoustic signals; and (6) sound localization and lateralization.

Currently, there are no standardized CAP assessment and management tools that have been developed for the polytrauma patient. This leaves test battery choices to the individual clinician, requiring detailed knowledge both of the patient’s complaints and of the specificity, reliability, and validity of the available tests. Ongoing clinical research is moving in the direction of standardizing these choices, but much work still remains to be done. Clearly, the tests chosen should measure different central processes by means of both verbal and nonverbal stimuli. CAP assessment is complex

with polytrauma patients because there are so many variables that can confound CAP test interpretations causing misdiagnosis. As described throughout this article, blast-injured patients with TBI may suffer from problems related to motivation, memory, attention, executive function, cooperation, cognition, neuronal loss, noise toxicity, metabolic and circulatory changes, working memory, hearing loss, vision loss, awareness issues, medication effects, PTSD, and other comorbid factors. In many cases, however, audiologists can employ sensitive behavioral tests and electrophysiologic procedures to gain insight into the integrity of the central auditory system and be mindful that whereas these patients have auditory complaints, the symptoms may not be entirely auditory modality specific. Some potential test measures for inclusion in a CAP assessment battery for patients with TBI include auditory temporal processing and patterning tests, dichotic speech tests, dichotic digit tests, monaural low redundancy speech tests, binaural interaction tests, and electrophysiologic tests.

Central deficits can be mistaken for PTSD or other emotional sequelae and cognitive deficits and therefore may be overlooked. With the increased incidence of TBI and PTSD,¹² it is imperative that CAP function is assessed to ensure that remediation strategies can be devised and implemented. In this population, a multidisciplinary approach is critical given the number of different causes that may be causing or contributing to the symptoms.

Treatment options for CAP deficits currently are under investigation by the research community. Current clinical guidelines⁸⁹ recommend a two-step approach that includes auditory training as well as general management options. Auditory training is designed to capitalize on the plasticity of the auditory system by altering the neural encoding of sound and subsequent timing of brain-stem responses. Studies have linked the neurophysiologic changes seen after training to perceptual changes.⁹⁰⁻⁹⁴ Patient-specific training focused on the deficit area(s) noted on the CAP assessment should occur soon after injury to maximize the plasticity of the brain. General management strategies include the implementation of environmental strategies, like the use

of an FM system, and the teaching of compensatory strategies. These treatment options may be provided by a speech-language pathologist, an audiologist, and an occupational and/or physical therapist. For example, specific remediation activities (deficit specific) such as phonologic awareness and discrimination training (speech to print skills), auditory closure, and prosody training can be provided by the speech-language pathologist; speechreading, communication strategy, clear speech family education, issuance of assistive listening device, and auditory training exercises can be provided by the audiologist; and exercises to improve interhemispheric transfer of information via cross-modality activities can be provided by the occupational and physical therapist.^{89,93,94}

The relationship between CAP test results and specific dysfunction is tenuous for the current CAP measures that exist. The collaboration of auditory researchers with cognitive psychologists, speech-language pathologists, neurologists, and other disciplines within the VA and the Department of Defense (DoD) and from the general academic, scientific, and medical community will significantly benefit the patient with TBI. Detailed guidelines on CAP evaluation and management are available elsewhere.^{93,94}

Tinnitus Evaluation and Management

Patients with complaints of tinnitus should undergo evaluation to assess symptoms and to identify management options. These patients have widely varying needs with respect to managing their tinnitus symptoms.⁵⁵ To accommodate the different levels of need, a hierarchical program of tinnitus management has been developed.⁹⁵ This comprehensive progressive intervention approach to tinnitus management includes Web-based training for audiologists and a patient self-management tinnitus workbook.⁹⁶ The program is evidence-based and is currently being piloted at a VA PRC. The five progressive levels of management include (1) triage (clinical referral at initial point of contact—normally to audiology, otolaryngology, and/or mental health); (2) audiologic evaluation (assessment of auditory function and tinnitus screening); (3) group education (classes that teach self-help strategies

and lifestyle factors to minimize tinnitus symptoms); (4) tinnitus evaluation (comprehensive evaluation of tinnitus that includes tinnitus psychoacoustic assessment, tinnitus intake interview, and assessment for any loudness tolerance complaints); and (5) individualized management (provision of one-on-one intensive care using a behavioral intervention).^{97,98}

At the Level 2 Audiologic Evaluation, an audiologist evaluates hearing function and administers written questionnaires. The questionnaires include the Tinnitus and Hearing Survey,⁹⁹ Tinnitus Handicap Inventory,⁸¹ and Hearing Handicap Inventory.⁸⁰ The purpose of the Level 2 Audiologic Evaluation is to determine if the patient needs medical, audiologic, and/or mental health intervention for hearing or tinnitus problems. The Tinnitus-Impact Screening Interview (TISI) is available as an efficient tool to assess tinnitus severity.^{100,101} Mental health screening questionnaires can be administered with appropriate referrals if deemed appropriate.⁹⁷

Management of hearing loss (with fitting of hearing aids as necessary) at Level 2 often addresses most or all tinnitus-related complaints. If tinnitus remains a problem, then patients are advised to attend the Level 3 Group Education classes. Relatively few patients require services beyond Level 3. The Level 4 Tinnitus Evaluation relies mainly on conducting an in-depth interview to determine the potential need for long-term individualized management.⁹⁷ At Level 4, tinnitus psychoacoustic measures (primarily tinnitus loudness and pitch matching and tinnitus maskability) are obtained. If the patient reports a reduced sound tolerance problem (hyperacusis), then a special evaluation is conducted to determine the characteristics and extent of the problem. Techniques are available to manage hyperacusis.^{60,98} Patients with PTSD and severe tinnitus may require test protocol modifications and referrals that address powerful limbic system responses.⁶¹ It is recommended that all patients who reach Level 4 should be screened for depression, anxiety, and PTSD.

At Level 4, a plan for Level 5 Individualized Management can be formulated and implemented if necessary. The type of intervention for tinnitus patients can vary signifi-

cantly, and clinical management methods do not adhere to any standards at this time.⁹⁷ The goal of individualized management is to lessen the impact of tinnitus symptoms on the patient's daily life, as there is no treatment that has been effective in reducing the symptoms directly.¹⁰² Patients who require this level of tinnitus management can receive any of several behavioral interventions, including Progressive Audiologic Tinnitus Management,⁹⁵ Cognitive-Behavioral Therapy (CBT),¹⁰³ Neurotonics Tinnitus Treatment,¹⁰⁴ Tinnitus Masking,¹⁰⁵ and Tinnitus Retraining Therapy.^{101,106} Each of these techniques requires special training. Any of the techniques can be effective, and research has not proved one method any more effective than any other. Normally, CBT is administered by psychologists, whereas the other methods are practiced by audiologists. However, CBT can be administered competently by nonpsychologists who have received the proper training.^{107,108}

Reevaluation

Research is focusing on the long-term effects of blast injury to the peripheral and central auditory system, but the likely time course of such injuries is not yet known. The current recommendation, therefore, is to reassess patients in 6 months (or sooner if indicated) and annually thereafter.

PREVENTION

Because of the many types of ear injuries incurred by soldiers, multiple strategies to prevent hearing loss are a high priority for DoD and VA. Physical barriers to sound, pressure, and debris in the form of headphones and earplugs prevent injury to the outer, middle, and inner ear. For those wearing hearing protection, the risk of hearing loss is minimized, and the incidence of ruptured TM is significantly reduced. In many incidents where troops used hearing protection when exposed to blasts, they did not sustain ear injuries, whereas soldiers without ear protection suffered ear damage and hearing loss (see the article by Jordan et al in this issue).^{23,109,110} In addition, monitoring of hearing thresholds in conjunction

with modern hearing conservation education has been shown to increase ability to correctly use hearing protection and to remove barriers to their use (see the article by Stephenson in this issue). Monitoring of hearing thresholds and education about synergistic effects of toxins and noise (see the article by Steyger in this issue) while patients are receiving ototoxic medications will provide early identification of hearing loss and possibly minimize any further hearing loss. Finally, preventative therapy with otoprotectants is under study (see the article by Lynch and Kil in this issue). The optimal treatment for soldiers may be a combination of all these approaches to ultimately minimize hearing impairment and auditory disabilities.

VA POLYTRAUMA CARE COORDINATION

The context of injury and treatment affects the rehabilitation care of the injured soldiers. Active-duty patients who are injured in combat have high visibility and are a political, military, and media focus. These patients are also a focus for DoD and VA leadership, which has responded by creating new systems of care at the local and national levels. There are currently four regional VA PRCs and a fifth under construction that provide acute inpatient medical and rehabilitation care for complex and severe polytraumatic injuries and 21 regional Polytrauma Network Sites to manage the subacute rehabilitation and care throughout the 21 Veterans Integrated Service Networks across the country. There are also more than 130 Polytrauma Support Clinic Teams and Polytrauma Points of Contact that manage patients with stable treatment plans. Transitions to these facilities allow patients and their families to be closer to home. Each PRC is currently developing or expanding its transitional community reentry program known as the Polytrauma Transitional Rehabilitation Program.⁵ The Veterans Health Administration clinical directive defines polytrauma as injury to the brain concurrently with injury to several other body or organ systems that results in physical, cognitive, and psychosocial impairments and disability.¹¹¹ The most common source of injury

of polytrauma patients is explosive blast. The resulting new clinical complexities not seen in previous conflicts are due to enhanced body armor and lifesaving medical care. The blast injured/polytrauma patients seen at the PRCs present with severe TBI, amputations, fractures, burns, vision loss, hearing loss, dizziness, tinnitus, PTSD and/or other psychiatric problems, pain, and/or infection.^{15,72} Admissions for TBI increased 47% at a PRC since onset of OIF.² Acute blast trauma and the TBI make up 90% of the injuries seen at the VA PRCs, and an additional 11 to 28% of OEF and OIF troops may have mild TBI from blast exposure.¹¹²

The patients seen at the PRCs are demographically different than the patients typically treated at VA hospitals prior to the GWOT. They are younger, technologically savvy, and have active family participation in all aspects of care.¹⁵ The severe combination of injuries presented by polytrauma patients is an unprecedented challenge for PRCs. These complex patterns have meant that audiologists need to screen for multiple conditions, adapt standard audiologic treatment protocols, incorporate new disciplines in the rehabilitation process, include more frequent use of cotreatment, continue to learn new skills about TBI and emotional sequelae, and involve more consultative services and the patient's family in the care of the injured returning service member.

CONCLUSION

The escalating use of explosive devices and mines in warfare combined with the excessive noise of ballistic weapons has created an unprecedented amount of auditory dysfunction among soldiers. Some of these patients also endure TBI and/or PTSD, further complicating audiologic rehabilitation efforts. An interdisciplinary team approach to this complex patient management is essential using the mechanism of blast injury model.⁴ Web sites to learn more about polytrauma and TBI are provided in Appendix B. An interdisciplinary comprehensive evaluation of peripheral, central, and vestibular components of the auditory system should be employed in patients with TBI to ensure that auditory dysfunction is

accurately diagnosed and appropriate rehabilitation can be performed.

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APPENDIX A: An Audiology Polytrauma Rehabilitation Center Report Template

AUDIOLOGY POLYTRAUMA REHABILITATION CENTER TEMPLATE

SUBJECTIVE:

CC: Evaluate Hearing Status

Patient complains/does not complain of hearing impairment, tinnitus, and/or vestibular problems since injury.

Patient reports/does not report sustaining a TM perforation. Patient reports/does not report losing consciousness at time of injury.

HPI: ___ year old (f)male polytrauma patient with penetrating/nonpenetrating TBI due to:

Review of Medical Record:

Review of Audiology Function:

SINCE YOUR INJURY

1. Have you had hearing problems?
2. Have you had ringing in your ears?
3. Have you had vertigo, dizziness, or loss of balance?
4. Have you had pain or fullness in your ears?
5. Have you had sensitivity to noises?
6. Have you had hearing problems in background noise?

BEFORE YOUR INJURY

1. Did you wear hearing aids?
2. Did you have hearing problems?
3. Did you have ringing in your ears?
4. Did you have vertigo, dizziness, or loss of balance problems?
5. Did you have a history of noise exposure (protected/unprotected)?

Observation of Physical Status:

- Bedridden
- Wheelchair
- Support cane/walker
- Able to transfer
- Helmet
- Other:
- Vision
- Communication Status (SLP report)

Hearing Handicap Inventory for Adults:

Tinnitus Handicap Inventory (if applicable):

Dizziness Handicap Inventory (if applicable):

Vestibular Disorders Activities of Daily Living Scale (if applicable):

Current Medicines:

OBJECTIVE:

Otoscopy:

Speech Reception Threshold:

Pure Tone Audiometry:

Speech Recognition in Quiet:

Word in Noise/Speech in Noise:

Otoacoustic Emissions:

Dichotic Digits:

GAP in Noise Test:

Other Tests:

Assessment:

Polytrauma patient has/does not have auditory symptoms related to injury.

Polytrauma patient has objective auditory findings possibly related to injury to include:

Polytrauma patient is/is not a hearing aid candidate.

PLAN:

Diagnosis:

Rx- To maximize communication function with patient: Face patient, talk slowly, and reduce ambient noise levels to maximize communication function.

Return for hearing aid and/or assistive listening device education with patient and significant other and/or caregiver.

Auditory training exercises

Aural rehabilitation (communication strategies)

Tinnitus management

Schedule for further audiologic assessment

- Completion of audiogram due to rest breaks/inattention/patient fatigue/illness
 - Auditory Brain-stem Response Evaluation
 - Auditory Steady-State Response Evaluation
 - Vestibular Evaluation
 - Tinnitus Evaluation
 - Central Auditory Processing Evaluation
 - Evoked Potential Testing-Middle and Late Latency Response Evaluation
 - Other:
-

APPENDIX B: Web Sites for the Audiologist to Learn More About Polytrauma and TBI

<http://www.polytrauma.va.gov/>

<http://www.hsrd.minneapolis.med.va.gov/PTqueri>

<http://www.dvbic.org/> (home page for the Defense & Brain Injury Centers)

<http://vaww1.va.gov/rehab4veterans/> (contains the 22-item NSI)

<http://vaww1.va.gov/VHI/page.cfm?pg=33> (EES TBI Veteran Health Initiative)

<http://vaww.ees.lrn.va.gov/Site/Templates/SearchResultDetails.aspx?pid=526&query=vhi&catalogId=22836>

<http://www.myvitalconnections.org/MVCHomepage.nsf> (Brain Injury Web Modules in English and Spanish from the Shepherd Rehabilitation Center in Atlanta, GA)

<http://vaww.va.gov/webcom/guidance0307.asp> (fact sheets on TBI and polytrauma)

www.wri.med.va.gov (War Related Illness and Injury Study Center Experts in Post-Deployment Health)

<http://vaww1.va.gov/audiospeech/> (National Audiology and Speech Pathology Service)

<http://www.seamlesstransition.va.gov>

http://vaww.sites.lrn.va.gov/vacatalog/cu_detail.asp?id=23155 (disseminates information to VA and DoD health care providers on unique and challenging health care needs for transitioning veterans with war wounds)

<http://www.ncptsd.va.gov>

<http://www.battlemind.or>

<http://www.oefoif.va.gov>

General Information Web Sites

www.biausa.org Brain Injury Association (BIA) of America

<http://www.tbinet.org> The Brain Injury Information Network

www.neuroskills.com The Center of Neuroskills Web site

www.waiting.com The Coma Waiting Page

www.dvbic.org Defense and Veterans Head Injury Program (DVHIP)

www.headinjury.com Head Injury Hotline

www.tbincd.org NIDRR TBI Homepage
