Chapter 14

HEARING IMPAIRMENT AMONG SOLDIERS: SPECIAL CONSIDERATIONS FOR AMPUTEES

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INTRODUCTION

Injuries to the ear were the single most common injury type reported among Marines during Operation Iraqi Freedom (OIF) through 2004, accounting for 23% of all injuries.[1] One third of soldiers returning from Operation Enduring Freedom (OEF) and OIF were referred to audiologists for hearing evaluations because of exposure to acute acoustic blasts; 72% of those sustained hearing loss. Auditory disabilities represent one of the most prevalent service-connected disabilities, resulting in over one billion dollars in annual compensation. Acute blast trauma and resultant traumatic brain injury (TBI) make up 90% of the injuries seen at the Department of Veterans Affairs (VA) polytrauma rehabilitation centers, and an additional 11% to 28% of OEF and OIF troops may have mild TBI from blast exposure.[2] Polytrauma patients require care for hearing impairment, vestibular pathology, vision loss, nerve damage, multiple bone fractures, wounds, psychological problems, and amputations.[3] Soldiers with amputations and hearing impairment present significant challenges because their injuries affect multiple systems, often impairing communication and cognition. In addition, each patient has distinctive psychosocial requirements that greatly impact pain management, adjustment to disability, body image perception, movement through the military disability system, and reintegration into the community or back to active-duty status.[4] Furthermore, sensorineural hearing loss, unlike many other injuries, will continue to progress, creating greater impairment for the injured soldier. Early identification and treatment of hearing loss and vestibular impairment is essential to developing treatment strategies, ultimately leading to reduced cognitive deficits and improved rehabilitation outcomes and quality of life.[5]

TYPES OF HEARING LOSS

For service members with limb loss, the types of hearing damage sustained are not fundamentally different from those that could accompany any other type of injury. Hearing loss can result from a blast injury, from explosion or weapon firing (acoustic trauma), or from ototoxic medications used to treat injuries. The combined effect of trauma, noise exposure, and ototoxic agents (such as solvents or medications) can also result in hearing loss or vestibular impairment. Complex polytrauma patients commonly have hearing and balance deficits, which are often initially overlooked. TBI patients with hearing loss can also be misdiagnosed as unresponsive.[6] Clinical complaints from hearing-loss patients rarely occur until a communication problem becomes significant, indicating reduced audibility within the frequency range important for speech understanding (less than 8 kHz). Also, vestibular problems can be misdiagnosed in patients with lower limb amputation because impaired balance may be attributed to the limb loss. It is essential that auditory and vestibular deficits be identified early for possible intervention and so medical care and rehabilitation can be facilitated. Otologic consultation or, at the minimum, an audiometric hearing evaluation should be performed on the basis of exposure, regardless of symptoms. Damage to the outer or middle ear results in a conductive hearing loss, which can usually be corrected medically or surgically. Inner ear damage can result in permanent sensorineural hearing loss. Some otologic symptoms, such as tinnitus and hearing loss, may be permanent consequences of a blast injury, and their effects on quality of life may be substantial.[7]

Noise-Induced Hearing Loss

Sounds of a sufficient intensity and duration can cause temporary or permanent threshold shifts due to vascular, metabolic, and chemical changes of the hair cells of the cochlea and swelling of the auditory nerve endings. The stiffness of hair cell stereocilia can also decrease, which reduces the coupling of sound energy.[8] Recovery from temporary threshold shifts can occur if the noise exposure is not prolonged, although short exposures to sounds of sufficient intensity (such as explosions) can cause immediate, permanent hearing loss. Harmful noise exposure results in cell death by necrosis and apoptosis (programmed cell death), and eventually leads to the degeneration of the corresponding nerve fiber. Acoustic trauma typically produces a unilateral hearing loss without associated middle ear damage. The susceptibility to noise-induced hearing loss between individuals varies as much as 30 to 50 dB, possibly due to differences in ear anatomy and physiology, prior exposure to noise, and interactions with medications.[8]

Noise-induced hearing loss can occur as a result of steady-state or intermittent exposure to loud noise or from a single, impulsive exposure to a loud sound, such as an explosion (acoustic trauma). Steady-state exposure is usually symmetric, leading to bilateral sensorineural hearing loss, while some impulse noises,

such as gunshots, can produce an asymmetric loss. Both steady-state and impulse noise exposure cause excessive oxidative stress and the production of free radicals (molecules that exist independently in an unstable state). To stabilize itself, a free radical takes available electrons from adjacent molecules, leaving them oxidized and therefore damaged. These changes lead to permanent cell damage in the inner ear. In addition, concurrent exposure to other agents, such as tobacco, solvents, or heavy metals, may have a synergistic effect on cell damage.[9] Soldiers in tactical situations who sustain acute noise-induced hearing loss may initially be unaware of it or delay reporting it because of the complexity of other injuries they have sustained, stress, fatigue, or the shock of the explosion.

Ototoxicity

Therapeutic drug regimens for infectious diseases and cancer can be ototoxic; nearly 200 prescription and over-the-counter drugs are recognized as having ototoxic potential and result in auditory or vestibular dysfunction. Ototoxicity causes irreversible cochlear damage and can occur in 60% to 70% of patients, often exacerbating preexisting hearing loss.[10] Clinical symptoms of ototoxicity are those commonly associated with hearing loss, such as tinnitus and difficulty understanding speech in noise. Hearing loss due to ototoxic medications is sensorineural, bilateral, and usually initially presents at high frequencies, subsequently progressing to the lower frequencies.[11–14] In addition, the effects of ototoxic drugs can continue well after cessation of therapeutic treatment. Previous noise exposure, almost universal in patients with blast injuries, increases the risk of ototoxicity.[15] Some ototoxic drugs commonly used to treat amputees for drug-resistant organisms (eg, gentamicin or vancomycin) can damage the vestibular system, causing instability and dizziness (dizziness, which includes vertigo, imbalance, and lightheadedness, can manifest with nausea, vomiting, a recurrent tendency to stumble or fall, floating feeling, mild unsteadiness, or faintness).[16]

Variations in the behavioral hearing threshold and number of frequencies affected suggest that individual susceptibility to ototoxicity is determined by multiple biochemical, physiologic, and genetic factors.[17] This individual susceptibility makes it difficult to determine which patients will experience ototoxic hearing changes and when the changes will occur. Therefore, high-frequency audiometry is essential to accurately identify and monitor ototoxic-induced hearing changes.

Audiologic Injury from Blast Exposure

Between 2003 and 2005, 68% of combat injuries in Afghanistan and Iraq were caused by explosions.[6] When exposed to the blast wave of an explosion, the auditory system is vulnerable to both peripheral and central damage from the pressure wave itself, and from the secondary and tertiary effects of blown objects impacting the body and the blown body impacting stationary objects (Figure 14-1). Blast trauma usually lasts longer and is more intense than acoustic trauma and typically affects both ears. The extent of damage from the primary blast injury is usually inversely proportional to the distance away from the blast. Due to the physics of blast waves, location within an environment may change the severity of injury, regardless of distance. Gases heated within a confined space may also result in a sustained period of positive pressure, and therefore prolonged injury. In general, closed-space blast injuries result in a higher incidence of primary blast injury, greater mortality, and greater injury severity.[18] Also, blast waves that are directed laterally to the head impact the auditory ear canal with a greater amount of energy, increasing the likelihood of ear injury. Permanent, pure sensorineural hearing loss is the most prevalent type of auditory impairment, occurring in 35% to 54% of blast injuries.[19]

One of the hallmark symptoms of unprotected blast exposure is injury to the tympanic membrane (TM) and, less frequently, to the ossicular chain. A ruptured TM indicates that a patient has undergone significant blast exposure and that further examination is warranted. The most common otologic complaints immediately following a blast are otalgia, tinnitus, aural fullness, dizziness, loudness sensitivity, distorted hearing, and hearing impairment. These complaints may last several minutes to several days. Hearing-related complaints may be due to a temporary threshold shift if present less than a month, or considered a permanent threshold shift if present over a month. Peripheral and central auditory system injury can combine to produce complex symptoms. The type of acoustic trauma, proximity, orientation of the ear to the blast, environment (open or closed space), and degree of ear shielding should be noted, when possible, in a patient's audiological case history.

Primary blast waves can cause concussions or mild TBI without a direct blow to the head by producing shock wave effects on the brain.[20] Concussions can cause audiologic symptoms, such as hearing loss, dizziness, and central deficits. Computed tomography scans and magnetic resonance imaging can detect pathology in the internal auditory canal, membranous

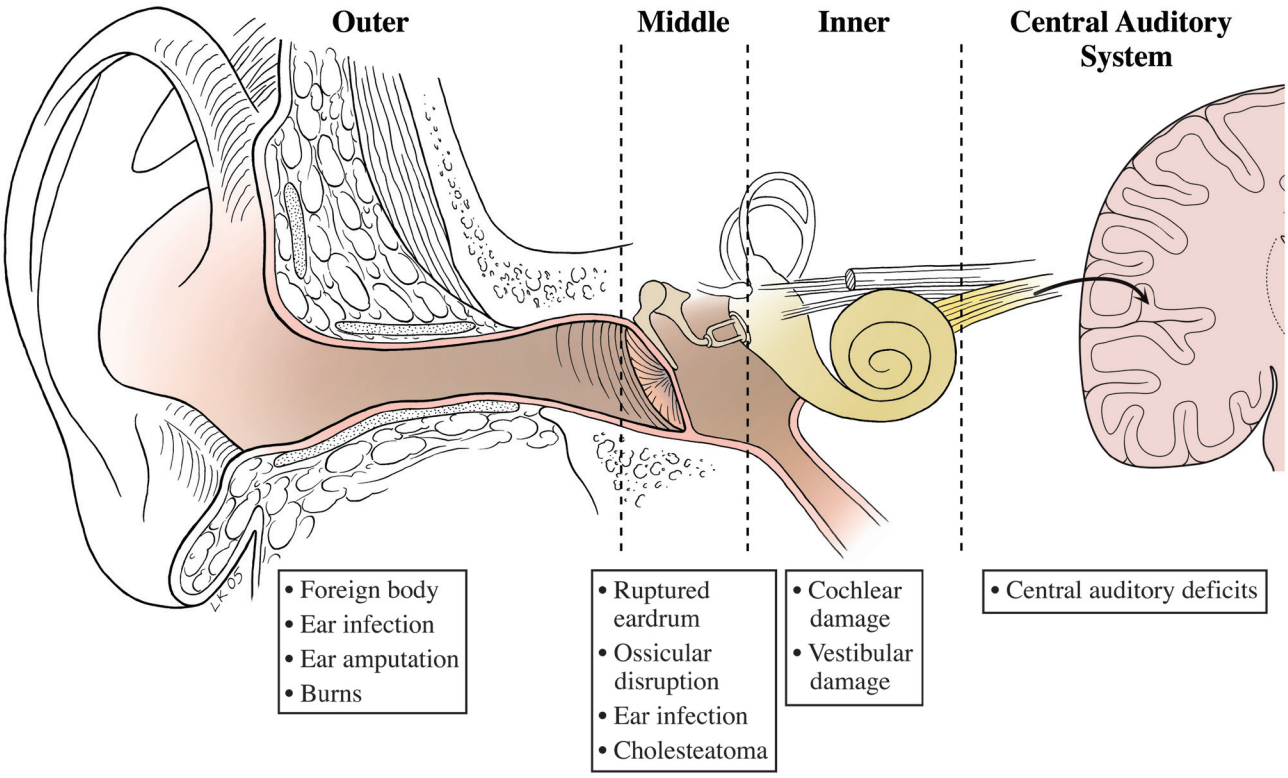

Figure 14-1. Blast injuries to the ear.
Drawing: Courtesy of Lynn H. Kitagawa, Veterans Affairs Medical Center, Portland, Oregon.

labyrinth, and bony labyrinth, and can characterize petrous apex lesions. Temporal bone fractures often cause loss of audiovestibular function. Auditory brain areas in the temporal lobe, corpus collosum, and thalamus are vulnerable to damage from exposure to the mechanisms of blast injury. Dizziness and sensitivity to sound can be symptoms of vestibular, peripheral, and central abnormality; posttraumatic stress disorder; concussion; and mild TBI. A thorough, multidisciplinary evaluation by specialists in neurology, otolaryngology, audiology, neuropsychology, physical therapy, and other related areas can facilitate appropriate diagnosis and treatment for patients with blast injuries and amputations. Long-term observation is important in all cases of blast-related auditory injuries because symptoms may be delayed. Postural instability and inner ear dysfunction may be evident up to 6 months or longer after blast trauma.[21]

Outer Ear Injury

The pinna can be damaged from flying debris and bomb fragments. Burns, contaminated debris, and purulent otorrhea in the ear canal are also frequently seen after exposure to blasts.[22,23] In addition, blasts can cause inner ear damage because of the impulse sound, location, and environment, leaving a patient with conductive, mixed, or sensorineural hearing loss. Alternatively, when the pinna is burned without associated noise-induced hearing loss or penetrating trauma, normal hearing sensitivity is often preserved. Burn patients, who are usually treated with antibiotics, are at risk for ototoxic sensorineural hearing loss and should be monitored accordingly.

Middle Ear Injury

Tympanic Membrane Perforation

When the primary shock wave reaches the end of the external auditory canal, it stretches and displaces the TM medially. Ruptured eardrums indicate that a patient has been exposed to peak pressure levels that far exceed those needed to produce damage to the inner ear, resulting in extensive hearing loss. The ear is much more sensitive to rupture pressure than other organs, with ruptures occurring in 50% of adults at 5 lb/in.^{2} (approximately 185 dB peak pressure level)

depending on the noise spectra and duration.[2,24,25] In contrast, pressure gradients of 56 to 76 lb/in^2 are needed to cause damage to other organs.[26] The improvised explosive devices causing injury in OIF and OEF produce pressures exceeding 60 lb/in^2, reaching peak pressure in about 2.5 to 50 milliseconds.

TM perforation is the most common injury to the middle ear, occurring during the positive phase of a blast. Typically, TM ruptures occur in the pars tensa, vary in appearance from linear tears to subtotal defects, and may have a protective effect because they reduce sound energy transmission to the inner ear.[23] TM rupture is also a clinical indicator that a patient has undergone a significant exposure, and it has been recommended as a diagnostic tool for determining whether or not a blast survivor has sustained life-threatening injuries. It may also be used as a potential marker for concussive injury.[22] However, the absence of TBI should not be assumed when TMs are intact.[27]

Clinical reports of TM rupture associated with ear infections range from 4% to 79% for explosion casualties.[1,28–30] Most TMs heal without surgical intervention, except in cases of resultant middle ear infection or when blast causes total TM perforation.

When the overpressure of a primary blast wave ruptures the eardrum, it sends small fragments of the squamous epithelium into the middle ear cavity. These cells may still be viable and grow into cholesteatomas that can infect or erode ossicles. Cholesteatomas usually present with conductive hearing loss and dizziness. Muscle weakness on the affected side can also occur. Careful surgical debridement is recommended for treatment, as is close follow up (10%–20% of cholesteatomas recur). Complications of cholesteatoma can include nerve damage and deafness.

Ossicular Disruption

The ossicles attach to the TM and transmit its vibrations to the cochlea, where the vibrations are converted into neural impulses via tiny hair cells. Overpressure can cause ossicle distortion and fracture. Ossicular damage is rare, usually occurring only with more severe blast trauma. Blasts that cause inner ear damage but spare the ossicular bones have been reported. A study of individuals exposed to blasts by explosive devices reported that ossicular damage occurred in 16% of patients, while 79% experienced TM perforations.[31] Injury to the middle ear structures usually results in relatively minor conductive hearing loss, compared to the extensive sensorineural hearing loss that can accompany exposure to impulsive pressure waves. In addition, ossicular-chain disruption may absorb some of the incoming energy of the blast wave, sparing the sensory structures of the inner ear. In a small study of patients with ossicular-chain disruptions, subjects regained much of their hearing at the lower frequencies following surgical repair of the ossicles.[32]

Inner Ear Injury

Cochlear Injury

The cochlea at the Organ of Corti is less resilient than the ossicles. This delicate system is overwhelmed by the amplification of blast and sound waves, causing loss of structural integrity with damage of the inner and outer hair cells. The result is the development of conductive and sensorineural hearing loss[33] because the basilar membrane displacement tears and mechanically injures the sensory cells in the cochlea. Inner ear damage following explosions results from the combination of the blast wave and the subsequent impulse sound. Signs of otologic injury are usually present at the time of the blast and should be suspected in anyone presenting with hearing loss, tinnitus, otalgia, vertigo, bleeding from the external ear canal, TM rupture, or mucopurulent otorrhea. Immediately following a blast, casualties may experience temporary hearing loss and tinnitus; some may sustain permanent damage to the Organ of Corti.[7] Disruption of the oval or round window can cause permanent hearing loss.[22]

The risk of acoustic trauma exists at sound pressure levels exceeding 140 dB. In a blast injury, pressure levels may exceed 160 dB, subjecting the ear to both the pressure effect and the acoustic wave. The structures of the inner ear are exposed to mechanical forces exceeding the elastic compliance of the tissue. At lower sound pressure levels, the effects are thought to be conveyed via metabolic disturbances in the inner ear, which lead to subsequent sensory and neuronal damage.[34] Exposure to blasts in excess of 200 dB results in substantial TM and ossicular damage, but sensorineural hearing loss can be variable, with most losses having a recoverable temporary threshold shift. In general, sustained high-intensity noise causes more sensorineural damage than a single high-intensity blast.[32]

Pure sensorineural hearing loss is the most prevalent type of auditory impairment following blast injury.[19] Recent literature suggests that 35% to 100% of blast victims present with hearing loss.[7,33,35–37] Walter Reed Army Medical Center reported that 64% of blast injured patients seen in 2005 had ongoing hearing loss.[6] The audiometric configuration and degree of

the hearing loss varied from normal hearing (typical of those who had shielding during the blast, such as headphones or earplugs), to high-frequency hearing loss at one or more frequencies (most likely presentation), to flat configurations from mild to severe hearing loss (primarily conductive or sensorineural), and in rare cases, profound deafness. Initial presentation can be mixed hearing loss (conductive element of TM perforation and mucosal lacerations of the middle ear and the sensorineural element due to mechanical injury). Although patients who have noise-induced hearing loss usually present with a 4 kHz "noise notch," or a decrease in hearing sensitivity around 4 kHz, those with blast injuries have a sloping high-frequency hearing loss that often affects frequencies below 8 kHz.

Vestibular Injury

Labyrinthine Damage

To maintain balance and postural stability, the vestibular system in the inner ear provides the brain with information about head movement via communication with the sensory organs (semicircular canals and otolith organs) and the eye, neck, arm, and leg muscles. Nerve connections from the semicircular canals and otoliths send information about head movement to the brain, resulting in eye movements that keep vision stable when the head moves. Similarly, nerve connections from the vestibular sensory organs to the brain send information to the neck, trunk, arms, and legs, resulting in posture changes that maintain balance. For the soldier with blast injury and amputation, it is possible that vestibular pathology, visual impairment, central pathology, certain medications, and proprioceptive changes all contribute to dizziness. The incidence of dizziness with mild head injuries ranges from 15% to 78%,[38,39] and is most often due to pathologies affecting the peripheral vestibular system, central nervous system (CNS), or cervical structures. In one of the few long-term studies on untreated patients with mild head trauma, vertigo persisted in 59% of patients after 5 years of recovery.[40] Compensation results from changes that occur within the CNS, rather than from changes in the peripheral vestibular system.[41] Following blast trauma, 27% of soldiers with amputation reported experiencing dizziness, and 18% reported experiencing vertigo.[16]

A primary care physician should rule out nonvestibular causes (orthostatic hypotension, postconcussive syndrome, TBI, ototoxic drugs, and visual impairment) of dizziness and an audiologist, otolaryngologist, and physical therapist can determine if dizziness is related to otovestibular pathology, such as peripheral disequilibrium, vertigo, or posttraumatic Ménière's disease. TM injury can be complicated by a perilymph fistula, an often under-diagnosed condition in which a tear between the membranes of the inner and middle ear causes a perilymph leak that produces vertiginous symptoms.[39] Labyrinthine damage may also occur after blast injuries, resulting in complaints of vertigo.

Although blast injury often affects vestibular function, it is less common and the symptoms less defined than other auditory damage sustained during an explosion. Vestibular testing can typically determine the presence of a vestibular pathology, yet the cause of the vestibular disorder may remain unknown. In general, vestibular damage can occur to one or both ears, and may affect the sensory organs (semicircular canals or otoliths), the vestibular nerve, or its connections. The most common causes of vestibular disorders include benign paroxysmal positioning vertigo (BPPV), Ménière's disease, labyrinthitis, vestibular neuritis, and ototoxicity.

Head trauma as a result of blast injury is one of the most frequent causes of BPPV.[21] BPPV is characterized by brief (a few seconds to a minute), severe vertigo associated with changing head positions, such as looking up or rolling over in bed. Ménière's disease (or endolymphatic hydrops) results from an increase in the fluid of the inner ear and can be traumatically induced. It is characterized by attacks of vertigo, ringing or roaring in the ears, a feeling of pressure or fullness in the ear, and hearing loss. Attacks typically last several hours, but can vary in length and frequency.

Some acoustic trauma and dizziness studies also suggest that noise exposure may result in vestibular disturbance.[42] Asymmetric exposure to extremely intense (140 dB or above) sounds leads to a greater likelihood of producing vertigo. Finally, ototoxicity can cause temporary or permanent balance loss, hearing loss, or both. Individuals who lose unilateral peripheral vestibular information must depend on other components of the sensory system (visual and proprioception), further complicating the rehabilitation process for patients with visual field deficits and lower limb loss, particularly if they have uncompensated unilateral peripheral vestibular loss or bilateral vestibular loss.[41]

Central Vestibular Damage

Otologic effects of blast injuries can include centrally mediated symptoms. TBI as a result of the primary blast wave or subsequent blow to the head may lead to central vestibular pathology, peripheral vestibular pathology, or both. Central vestibular trauma may be due to postconcussive syndrome or cerebral or brainstem injuries.[43,44] Central vestibular pathology is less common than peripheral pathology in patients follow-

ing head injury[45] and blast exposure.[37] In some cases, there may be evidence of both central and peripheral vestibular dysfunction.[45]

Central Auditory System Damage

While the presence of a ruptured TM is an obvious physical finding of blast exposure, the most detrimental effects of a blast may be less obvious and harder to treat. It is likely that patients with amputations resulting from blast exposure were either hit by flying objects (secondary damage) or were picked up by the blast wave and thrown into a stationary object, such as a vehicle or a wall (tertiary damage). In either case, there is a significant chance that the brain was also impacted. It is not known what proportion of those exposed to blasts sustained damage to the central auditory system; however, it has been well established that when an impact causes the brain to move to the extent that it impacts bone, contusions (hemorrhage and edema) occur, and the auditory processing areas of the temporal lobe are most commonly impacted.[46] Additionally, shearing, stretching, and angular forces of blast waves cause swelling and disconnection of axons in the central auditory system.[46] Damage to the CNS after an explosion has been increasingly attributed to the direct effects of the blast.[22] Inhalation of toxic chemicals (including materials such as paint and some industrial organic chemicals) may also affect the CNS and may present as vertigo or unsteadiness.[42]

There are a significant number of people who have normal hearing sensitivity following blast exposure, yet still complain of hearing difficulty. Central auditory dysfunction may contribute to these complaints, but diagnosis and treatment is challenging because the link between neural structures and function in central auditory processing (CAP) disorders has not been fully discerned. The lower brainstem nuclei, such as the inferior colliculus, are involved with comparing stimuli arriving from the ears ("binaural" processing). For this reason, only sensitivity to binaural relationships is affected by unilateral brainstem damage (bilateral lower brainstem damage is generally fatal). The thalamic nuclei in the upper brainstem can be damaged by shearing or stretching forces, resulting in deficits in determining the location or duration of sounds. Binaural "unmasking" refers to the ability to determine time and level differences at each ear to improve detection of speech and other important sounds in noisy environments. Essentially, listeners can "cancel out" noise coming from a location that differs from that of the signal to be detected.[47] Although exactly where such binaural unmasking occurs is still a matter of debate, it is possible that the binaural information extracted by the early brainstem is used for binaural unmasking operations that actually occur at the level of the auditory cortex.

At the cortical level, bilateral damage to the superior temporal gyrus (primary auditory cortex) can result in immediate and ongoing insensitivity to all sounds. The extent to which attentional and behavioral processes underlie these deficits is not yet well understood.[48] In addition to "cortical deafness," which often recovers over time, there are a number of additional processing deficits associated with damage to auditory cortical areas that may impact speech comprehension, appreciation and discrimination of music, and identification and discrimination of environmental sounds. These diverse "agnosias" may stem from difficulties analyzing spectrotemporal patterns in sounds.[49]

Providing clinical data that relates specific damage to discrete behavioral deficits will provide a better understanding for a clinician and corroborative evidence for the spectrotemporal processing pathways that are being uncovered in animal studies. The relevance of diagnosing clinical disorders using animal models of cortical processing will likely only be appreciated when clinical and animal data are merged. Current trends suggest that the same basic units of analysis that exist in the mammalian cortices of cats and primates correlate with human perception of speech, music, and environmental sounds. If such a relationship could be clearly established, it would be possible to develop a set of standardized diagnostic tests that specifically target the perception of patterns in frequency, time, level, and spatial position. A more detailed discussion of diagnostic testing has been provided below.

Tinnitus

Damage to hearing due to excessive noise exposure in acoustic trauma is frequently accompanied by tinnitus. Noise-induced tinnitus can be characterized by a high-pitched buzzing or ringing sound that lasts longer than 5 minutes and may produce annoyance, behavioral changes, and depression.[50,51] Damage to neural networks, association pathways, associated cortical regions, the limbic system, and the prefrontal cortex may be involved in the generation of tinnitus.[52] Tinnitus symptoms resulting from blast or impulse noise exposure often resolve over time. Persistent symptoms can occur in some tinnitus patients, many of whom report disability equal to or greater than that associated with hearing loss.[50] Although tinnitus is a symptom associated with many forms of hearing loss, it can also be a symptom of other health problems, such as stress, use of certain medications, allergies, tumors, metabolic factors, and vascular disease (especially that involving vessels in the jaw and neck).

DIAGNOSTIC TESTS

It is often difficult to assess for otovestibular injury in a polytrauma patient in a timely manner because of consciousness level, contraindications to testing (c-spine precaution, ventilator use, bed rest orders, pain), and multiple priority appointments at other clinics. Testing protocols are commonly modified based on the complexity of an injury. For example, many polytrauma patients have burns or severe injuries to their skulls, so some testing equipment, such as earphones or bone conduction headsets, is contraindicated. There is currently no standard method for diagnosing otologic, otovestibular, and CAP disorders in patients with blast injury and amputation. When possible, the following protocol is recommended until ongoing clinical research identifies factors to aid in the development of more sensitive tests to assess blast-injury amputees for peripheral, central, and otovestibular pathology.

Patient Questionnaires

Polytrauma patients should complete case history and standardized questionnaires that assess the impact of hearing loss, tinnitus, and dizziness. The audiologic case history questionnaire should elicit information about a patient's medical history, including cognitive functioning, visual history before and after the blast, audiologic history before and after the blast (including exposure to potentially damaging noise), and tinnitus history (if applicable). Patients should also be asked to provide detailed information about the blast exposure, describing the nature of the blast, proximity to the blast, use of hearing protection, and loss of consciousness at the time of injury. The Hearing Handicap Inventory for Adults consists of two subscales and assesses the emotional and social consequences of hearing loss.[53] The Tinnitus Handicap Inventory is used to assess self-perceived handicap related to tinnitus, and the Dizziness Handicap Inventory assesses handicap by quantifying the functional, emotional, and physical effects of dizziness and unsteadiness.[54,55] Screenings for posttraumatic stress disorder and mild TBI should be administered when appropriate. TBI screening was created for VA clinicians to screen all OEF and OIF veterans receiving medical care within the Veterans Health Administration. The screening allows clinicians to offer further evaluation and treatment to those who test positive.

Comprehensive Audiometric Evaluation

Otoscopy should be performed prior to audiometric evaluation. If otoscopy reveals occluding or impacted cerumen or otherwise abnormal pathology, patients should be referred to an otolaryngologist. Monaural air- and bone-conduction thresholds should be measured in each ear to determine the type and degree of peripheral hearing impairment. Because many patients with blast-related limb loss may be taking ototoxic medications, air-conduction hearing thresholds should be measured at frequencies up to 12,000 Hz, or to the frequency limits of the individual's hearing.[56] Monaural speech-recognition testing, including reception thresholds for speech and speech intelligibility for one-syllable words, should be conducted in quiet. To further rule out or confirm the presence of conductive or retrocochlear pathology, immittance audiometry, including tympanometry, ipsilateral and contralateral acoustic reflex thresholds, and contralateral acoustic reflex decay should be completed in each ear. If pure tone testing reveals conductive or mixed hearing loss or if tympanometry results are abnormal, the patient should be referred to an otolaryngologist for evaluation. Otoacoustic emissions should be used to confirm other test findings. Additionally, auditory brainstem response testing and auditory steady-state response testing should be considered when a patient is unable to undergo conventional behavioral audiometry.

Ototoxic Monitoring Protocol

For patients on ototoxic medications, comprehensive baseline testing should supply information about pretreatment hearing levels. Baseline measures will provide a reference for comparison to determine if hearing levels have changed. This testing should be done as soon as possible after patients have been identified as needing ototoxic medication. For patients treated with aminoglycoside antibiotics, baseline testing should occur prior to the start of medication or within 72 hours of the first dose administered. Patients receiving loop diuretics should be seen sooner than 72 hours after the first dose. For patients receiving cisplatin or carboplatin, baseline testing should be evaluated within 24 hours of the first dose.

Minimum baseline evaluation should consist of bilateral pure tone air-conduction thresholds from 250 to 8,000 Hz. This should include the half octaves of 3,000 and 6,000 Hz. Whenever possible, high-frequency thresholds above 8,000 Hz should be tested (9,000, 10,000, 11,200, 12,500, 14,000, 16,000, 18,000, and 20,000 Hz). Intrasession reliability should also be evaluated by retesting some frequencies to measure for consistent results. A reliable test should show responses that are within or equal to 5 dB HL. If hearing loss is present from 250 to 4,000 Hz, bone conduction should also be

performed. Testing should be as thorough as possible; however, if a patient is too sick for a comprehensive evaluation, testing may need to be modified or may need to occur over multiple sessions. If a patient is unable to complete behavioral testing, electrophysiological testing may be performed.

Ototoxicity monitoring is based on a patient's drug therapy schedules. For patients receiving aminoglycosides, otoscopy and pure tone air conduction tests in the normal and high-frequency ranges should occur at least once a week. Cisplatin patients should be monitored within 24 hours preceding each dose. For ototoxic medication monitoring, speech audiometry, bone conduction, and immittance testing should be performed if there is a change in hearing. If a change in hearing occurs, a retest should take place within 24 hours to confirm threshold change. Reevaluation should occur at approximately 3 and 6 months after treatment to assess any residual effects of drug treatment. If a decrease in hearing threshold is noted, weekly tests should take place until changes cease.

Ototoxic monitoring protocols at military facilities and VA hospitals must institute best practice policies for situations in which baseline measures are not available, concomitant medications are being used, septic patients require immediate therapy, and when bedside testing must be made available for patients that cannot be transferred for testing. In addition, ototoxicity monitoring should include patient counseling and education.

Assessment of the Vestibular System

If a patient reports problems with dizziness or unsteadiness, a multisensory balance evaluation is indicatied. The comprehensive balance evaluation should include a vestibular, visual, somatosensory, and brain integration system assesment. Clinicians working with soldiers with blast trauma and amputation need to consider several causes of postural instability, including TBI, orthostatic hypotension, cervical vertigo, visual deficits, possible side effects of ototoxic drugs, and vestibular pathology. Vertigo is often related to a vestibular balance disorder; however, imbalance and lightheadedness may or may not be related to inner ear damage. Some other causes of imbalance or lightheadedness include neurological impairments, vascular flow or pressure changes, cardiac problems, certain medications, or systemic disorders, such as diabetes. Screening tests typically administered by physical therapists during the vestibular portion of the blast evaluation include a cervical range-of-motion and cervicalgia assessment, oculomotor evaluation, postural stability tests, gait assessments, and vertebral artery tests 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 these screenings include cervical instability, fractures preventing cervical range-of-motion assessments, and use of medical equipment that precludes communication, such as a jugular line or ventilator.[16] Screening tests are not sensitive enough to detect all vestibular abnormalities and do not provide quantitative data to determine the presence or absence of vestibular lesions.

Patients showing signs of vestibular pathology should be further evaluated to determine if dizziness or balance problems are related to a vestibular disorder. Vestibular tests can help characterize the deficits a patient has and guide the development of appropriate rehabilitative techniques. Videonystagmography is a battery of tests that evaluates the horizontal semicircular canals (one of the inner ear balance organs), its nerve connections, and the eye movement systems. Because the vestibular system is connected to the eye muscles, vestibular function is measured by recording eye movement. If electrodes are used, the test battery is typically called electronystagmography. The videonystagmography test can identify an inner ear balance disorder that can occur in one or both ears and is related to the horizontal semicircular canal and its nerve. The Dix-Hallpike maneuver, often performed as part of the videonystagmography/electronystagmography test battery, can determine if the symptoms of vertigo are related to BPPV and can identify which semicircular canal is involved.

Vestibular-evoked myogenic potentials assess the saccule (one of the otolith organs) and its nerve connections to the neck muscles to evaluate vestibular function. The rotary chair test evaluates the horizontal semicircular canals and their connection to the eye muscles and is used to monitor vestibular loss in patients on ototoxic medications. Finally, computerized dynamic posturography evaluates a patient's ability to integrate vestibular, visual, and somatosensory cues to maintain balance. This test is helpful for patients with lower extremity issues (eg, amputation, paralysis, etc) to monitor their risk of falls and retrain them for better stability and balance.

More recent best practices have begun to incorporate the computerized dynamic visual acuity test and the subjective visual vertical test during off-axis rotation to assess utricle function in the blast-injured population. The computerized dynamic visual acuity test measures visual acuity during head movement to assess vestibular deficits.[57,58] Monitoring an amputee with vestibular symptoms is essential and can

be accomplished by outcome questionnaires, physical screens, repeating tests that had initial positive findings, and assessing and modifying management strategies on an ongoing basis.

Assessment of Central Auditory Processing Disorders

Deficits in CAP refer to "difficulties in the perceptual processing of auditory information in the CNS," as demonstrated by poor performance in one or more of the following skills: (*a*) auditory performance in the presence of competing acoustic signals (eg, dichotic listening); (*b*) temporal aspects of audition (eg, temporal integration, temporal discrimination, temporal ordering, temporal masking); (*c*) auditory pattern recognition; (*d*) auditory discrimination; (*e*) auditory performance with degraded acoustic signals; and (*f*) sound localization and lateralization.[10] Unfortunately, central deficits can be mistaken for posttraumatic stress disorder, mental health issues, and cognitive deficits, and therefore may be overlooked. With the increased incidence of TBI, CAP function must be assessed to ensure that remediation strategies can be devised and implemented. Currently, no standard CAP assessment protocol exists. A CAP test battery should be chosen for a given patient based on specificity, reliability, and validity. Tests should be selected that measure different central processes, and both verbal and nonverbal stimuli should be used. Some potential test measures an audiologist may include in a CAP assessment battery are provided below.

Auditory Temporal Processing and Patterning Tests

Auditory temporal processing and patterning tests are designed to assess a patient's ability to analyze acoustic events over time. An example of such a test is the Gaps-in-Noise Test.[59]

Dichotic Speech Tests

Dichotic speech tests are designed to assess a patient's ability to either separate or integrate different auditory stimuli presented to the ears simultaneously. One such test, the Dichotic Digits Test, requires a patient to listen to numbers presented to both ears simultaneously and indicate what was heard in each ear.[60] This test has demonstrated sensitivity to CNS pathology while remaining relatively resistant to mild-to-moderate high-frequency cochlear hearing loss.[61]

Monaural Low-Redundancy Speech Tests

Monaural low-redundancy speech tests assess a patient's ability to recognize degraded speech stimuli. Many audiologists use a speech-in-noise test to assess this component of central auditory processing. An example of such an assessment is the Quick Speech-in-Noise Test.[62] This test can be used with patients that have either normal or impaired hearing.

Binaural Interaction Tests

Binaural interaction assessments, such as the masking-level difference test, evaluate binaural processes depending on intensity or time difference of acoustic stimuli. In the masking-level difference test, binaural thresholds for pure tones (typically 500 Hz) or speech are determined in the presence of binaural masking noise. The tone or speech signal is presented in-phase to the ears and the noise is presented either in-phase or out-of-phase between the ears. Normal binaural processing is associated with an improved detection threshold for the out-of-phase noise. A 500-Hz masking-level difference test is clinically available and can be used for this assessment.[63]

Electrophysiologic Tests

Electrophysiologic tests measure electrical potentials that reflect activity generated by the CNS in response to auditory stimulation. The middle-latency response and the late-latency response have been shown to assess central auditory dysfunction. Putative neural generators for the middle-latency response include the auditory thalamocortical pathways, the mesencephalic reticular formation, and the inferior colliculus. The middle-latency response consists of a series of positive and negative peaks that fall in the latency range of 10 to 80 milliseconds after onset of the stimulus. Putative neural generators for the late-latency response, on the other hand, include thalamic projections into the auditory cortex, the primary auditory cortex, the supratemporal plane, the tempoparietal association cortex, and the lateral frontal cortex. In general, an averaged late-latency response wave is composed of contributions from multiple structures and can reflect brain functions such as attention, cognition, discrimination, and integration. The recorded wave consists of a series of positive and negative peaks that occur from 70 to 500 milliseconds after onset of the stimulus. These electrophysiologic tests, in conjunction with the auditory brainstem response, can help pinpoint where in the auditory system deficits might exist.

CAP assessment is controversial and complex,

especially with the polytrauma population, because variables such as motivation, attention, cooperation, cognition, neuronal loss, noise toxicity, metabolic and circulatory changes, working memory, and other comorbid factors can confound test interpretations and cause misdiagnosis. Because CAP deficits can be mistaken for myriad other disorders, it is essential that this assessment be done as part of a comprehensive team approach. An audiologist should work closely with professionals in mental health, neuropsychiatry, speech-language pathology, physical and occupational therapy, and other related specialties.

Tinnitus Evaluation

Patients presenting with complaints of tinnitus should undergo evaluation to assess symptoms and to identify treatment options. The Tinnitus-Impact Screening Interview and the Tinnitus Handicap Inventory questionnaire should be completed to determine the severity of symptoms. Sound tolerance measures can determine the presence of hyperacusis, or reduced loudness tolerance.[64] In addition, psychoacoustic measures, such as loudness and pitch matching,[65] allow for full characterization of a patient's tinnitus if it is problematic and requires treatment. An assessment of tinnitus maskability, or the minimum masking level necessary to render the patient's tinnitus inaudible, has been shown to correlate with treatment efficacy.[66] Finally, if measures of otoacoustic emissions have not already been performed, they should be done at this time to provide objective evidence of cochlear dysfunction (even if normal pure tone thresholds), and to provide frequency-specific information, which may be associated with the frequency region of the tinnitus.[67]

Reevaluation

The long-term effect of blast exposure on peripheral and central auditory functioning is not yet known and needs to be investigated more fully. The current recommendation, therefore, is to reassess patients in 6 months (or sooner if indicated) and annually thereafter.

TREATMENT AND REHABILITATION

Treatment and rehabilitation of auditory and vestibular injury in the amputee often require a multidisciplinary approach, focusing on otolaryngology, neurology, audiology, occupational therapy, and physical therapy. The type of auditory deficit present in a patient will determine treatment options, including medical and surgical interventions, technological considerations, and auditory training and counseling. Audiologists must make special considerations for service members with severe injuries to their extremities as a result of blast exposure. Those with concurrent hearing impairment must adjust to life with prosthetic limbs as well as prosthetic hearing aids and assistive listening devices.[68] Additionally, many soldiers with amputation have concurrent TBI and require accommodating rehabilitative approaches. Providers may need to give examples to go with new ideas and concepts, reduce visual and auditory distractions, and give step-by-step directions to reduce confusion. It is also important to demonstrate and explain information in more than one modality. Brain injury can cause cognitive impairments, such as problems with orientation, attention, concentration, perception, comprehension, learning, thought organization, executive function, problem solving, and memory. Additionally, up to 28% of all blast-exposed soldiers have significant eye injuries. Visual field deficits must be taken into consideration when providing aural rehabilitation and amplification device instructions. Furthermore, behavioral issues, including aggression, agitation, mental trauma, and adjustment to disabilities may interfere with treatment plans at early stages of recovery.[3]

Audiologists must be knowledgeable about managing problems specific to a patient's amputation and work with the treatment team to reduce this impact. For example, upper limb amputees that require hearing aids need to learn how to independently place an aid using a prosthesis. These challenges become compounded if an individual also has a TBI, vision loss, or loss of more than one limb. It is imperative that an audiologist actively participates as part of the rehabilitation team.

Peripheral Hearing Loss

Hearing aids deliver an amplified signal to the ear and are recommended when their use can significantly improve a patient's ability to communicate. When effective medical treatment can be implemented to restore normal hearing, or when hearing-aid use would exacerbate a disease or interfere with treatment, amplification is not used. Hearing aids are classified by their style, size and placement, or technological features. The most important developments in hearing aids in the past decade have been the introduction of digital programmable hearing aids, ranging

in size from completely in the ear canal to behind the ear, and the increasing popularity of directional microphones. Directional microphones provide the greatest amount of amplification for signals arriving from the front, where speech typically emanates, and places nulls (areas of reduced amplification) for signals from the sides and back, which are the usual locations for noise sources.

When selecting a hearing aid for today's polytrauma patient, there are multiple factors to consider. For instance, some returning soldiers with hearing impairment have body-image issues stemming from multiple scars, burns, and amputation requiring prosthetic limbs, and may prefer a completely in-the-canal hearing aid versus a larger style. Additionally, it is important to consider possible alternatives when it is difficult or impossible for the patient to manually insert the hearing aid. This is especially true if the patient has lost or significantly impaired one or both upper limbs. The use of remote control options to adjust the volume and program settings (for quiet and noisy environments, telecoil or frequency modulation [FM] use) is likely to be a good option for these patients. Automatic hearing aids that adjust to different acoustic environments without human intervention are also available as needed, especially for those with significant cognitive impairment.

Unfortunately, traditional hearing aids, even those equipped with directional-microphone technology, may provide only limited improvement in speech perception in everyday listening environments where there is noise and reverberation. One well-recognized technology to improve speech perception in listeners with hearing loss is FM systems. With an FM system, the speaker's voice is picked up by a wireless microphone and the signal is transmitted via FM radio waves to the receiver. The close proximity of the FM microphone minimizes the negative effects of reverberation, distance, and noise on speech perception.[69]

In addition to FM technology, a host of other assistive listening devices could be considered for this patient population. Some of these options include captioning for television viewing, teletext or volume-control telephones, and alerting devices that employ flash-visual or vibrotactile signals to warn of acoustical environmental events. Many of these devices are available at no cost to the veteran through the VA or the state in which the veteran resides.

Vestibular Problems

Treatment of vestibular problems should be specific to the patient and to the deficit, and physicians and physical therapists should collaborate on patient treatment. Depending on diagnostic test results, vestibular treatment options can include vestibular rehabilitation therapy; physical therapy; canalith repositioning therapy; change in activity levels, medication, and diet; treatment for underlying disease that may be contributing to the balance disorder; and surgery.

Vestibular rehabilitation therapy is the treatment of choice for many types of vestibular disorders. Vestibular rehabilitation therapy is exercise therapy that includes head and eye movement and balance and walking tasks that are designed to help the brain compensate for vestibular problems. Vestibular rehabilitation therapy, postural stability training, and gait training are usually provided by a physical therapist.

Canalith repositioning therapy is the preferred treatment for BPPV. The goal of canalith repositioning therapy is to move the displaced particles (otoconia) through the semicircular canal and back to the vestibule that houses the otolith organs. This is achieved by placing the patient in a series of head positions and observing eye movement. It may be performed by an audiologist, otolaryngologist, or physical therapist.

Central Auditory Processing Deficits

Treatment options for CAP deficits are currently under investigation by the research community. Current clinical guidelines recommend a two-step approach that includes auditory training and general management.[10] Auditory training is designed to capitalize on the plasticity of the auditory system by altering the neural encoding of sound and subsequent timing of brainstem responses. Studies have linked the neurophysiologic changes seen after training to perceptual changes.[70–72] Subjects can learn to interpret sounds as speech that could not be discriminated before training. It is recommended that training occur soon after injury to maximize the plasticity of the brain, and training should be patient specific and focused on the deficit areas noted on the CAP assessment. General management strategies include environmental tactics, like the use of an FM system, and teaching compensatory strategies. These treatment options may be provided by a speech-language pathologist, an audiologist, and an occupational and physical therapist. Specific remediation activities (deficit specific), such as phonological awareness and discrimination training (speech-to-print skills), auditory closure, and prosody training, can be provided by a speech-language pathologist. Speechreading and auditory training exercises can be provided by an audiologist, and occupational and physical

therapists can use cross-modality activities to improve interhemispheric transfer of information.

Tinnitus

The majority of patients who present with tinnitus following blast exposure experience a resolution of symptoms over time and do not require any audiologic intervention.[19] Of the approximately 20% of tinnitus patients that have clinically significant symptoms, rehabilitation needs vary from simple education about the condition to counseling and treatment.[19] Because no treatment has been effective in directly reducing tinnitus symptoms, the goal of treatment is to lessen their impact.[9] Tinnitus treatments include medication, masking therapy, Tinnitus Retraining Therapy, Neuromonics Tinnitus Treatment and other sound therapy and directed counseling, psychological therapy (such as cognitive-behavioral therapy), and hearing aids.[50] The type of intervention for tinnitus patients can vary significantly, and clinical management methods do not adhere to any standards at this time.[67]

PREVENTION

Because of the many types of ear injuries incurred by soldiers, multiple strategies must be employed to prevent hearing loss in this population. Physical barriers to sound, pressure, and debris, such as headphones and earplugs, prevent injury to the external and middle ear. In addition, monitoring hearing thresholds while patients are receiving ototoxic medications provides early identification of hearing loss, allowing for possible changes in treatment. Finally, systemic compounds for otoprotection may prevent damage at the cellular level. The optimal treatment for soldiers may be a combination of all these approaches to ultimately minimize hearing impairment.

Hearing Conservation

Headphones and hearing protectors offer significant shielding and protection from otologic injury by protecting the ear from excessive noise, flying debris, and the pressure wave associated with a blast. Technological advances have enabled hearing protectants to be equipped with microphones so users can maintain hearing sensitivity. For those wearing hearing protection, the risk of hearing loss is minimized and the incidence of ruptured TM is significantly reduced. In several blast incidents, soldiers using hearing protection did not sustain ear injuries, whereas those without ear protection suffered ear damage and hearing loss.[73,74] Military personnel assigned to light armored vehicles as commanders, gunners, and drivers wear protective helmets, but personnel in the rear of these vehicles do not generally wear protection and are therefore most susceptible to ear blast injuries.[1] Greater than 20% of the blast-injured soldiers traveling in light armored vehicles in various military conflicts presented with ear injury, mostly ruptured TMs.[1] In contrast, eye injuries are extremely uncommon for these soldiers, presumably due to the ballistic eye protection soldiers wear when traveling in light armored vehicles.[1] It is essential to adopt hearing protection standards to shield service members at risk for blast or excessive noise exposure.

Otoprotectants

Otoprotective agents may be capable of preventing noise-induced and ototoxic hearing loss by reducing the damaging effects of noise or medications on the hair cells of the cochlea. In addition, some otoprotectants may be able to rescue damaged cells once injury has occurred, thereby minimizing hearing loss. There have been numerous studies of various compounds used to protect hearing, including antioxidants that scavenge free radicals, agents that increase blood flow, and drugs that block cell-death- pathway–signaling factors. Despite the potential of these compounds, the translation of animal studies of otoprotectants to clinical treatment has been limited. Clinical trials are needed to delineate the effects of these protective compounds. Effective preventative therapies may ultimately require a combination of compounds that employ multiple mechanisms to combat cell damage in the inner ear.

SUMMARY

The escalating use of explosive devices in warfare, combined with the excessive noise of ballistic weapons, has created an unprecedented number of hearing impairments among soldiers, many of whom also sustain amputations, further complicating rehabilitation efforts. A comprehensive interdisciplinary evaluation of peripheral, central, and vestibular components of the auditory system must be completed in polytrauma patients to ensure that all injury is accurately diagnosed and appropriate rehabilitation can be devised. Furthermore, the implementation of hearing conservation strategies—including physical ear protection, preventative therapy with otoprotectants, and implementation of early detection and monitoring programs—may sig-

nificantly reduce the number of patients with disabling hearing impairments. Effective treatment and hearing loss protection programs can also reduce the potential for the medical, legal, and socioeconomic consequences of hearing loss, and ultimately allow patients to retain a better posttreatment quality of life.

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