

A guide for hearing healthcare providers to characteristics of traumatic brain injury

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Traumatic brain injury (TBI) is a ubiquitous injury that can give rise to hearing loss. By 2020, TBI is expected to affect 10 million people annually.¹ As TBI becomes more prevalent, more and more people with the condition will need hearing healthcare during both the acute and the chronic stages of injury. In the early stages of recovery, audiological services are usually provided in the context of specialized rehabilitation settings where hearing healthcare professionals work with inter-professional teams. Following discharge from such settings, individuals who have had a TBI may need continued services in the general community for the rest of their lives.

TBI is currently the leading cause of disability in North America in people under the age of 40. Patients

focusing on the more vulnerable *severely* brain-injured patients.

This background information will help hearing professionals communicate more effectively about TBI cases with other health professionals and will also help them work more effectively with patients who have suffered a TBI. We will start with a general overview of the classification of TBI. This will be followed by an outline of the general pathophysiology of TBI and the impact of TBI on the auditory system. We will end with a brief discussion of the clinical implications for assessment, diagnosis, and treatment of patients with TBI for practitioners in rehabilitation settings and in the community.

CLASSIFICATION OF TBI

TBI is caused by an externally inflicted trauma to the head (or brain) that can result in transient or permanent impairments.^{8,9} The cognitive functions most commonly affected are executive functions (i.e., a constellation of higher-order abilities, including self-monitoring, planning, problem-solving, initiation and follow-through, and emotion regulation), memory (both verbal and visual-spatial), attention and concentration, and speed of processing.¹⁰ As we will discuss later, impairments to these abilities can have a significant impact on a clinician's ability to identify and treat changes in hearing.

TBI can vary widely in severity, although it is typically differentiated both clinically and scientifically into the broad categories of "mild" and "moderate-to-severe." Classification of injury severity is most commonly carried out by using the Glasgow Coma Scale (GCS),¹¹ in which a score of 13 to 15 constitutes a mild injury, 9 to 12 moderate, and 3 to 8 severe. The GCS score can be obtained readily at the scene of the accident or in acute care, and is well correlated with early morbidity and mortality outcomes.^{12,13}

Mild and moderate-to-severe TBI differ markedly in their clinical presentation and outcomes and in their time course of recovery. For the former, visible damage to the brain is rarely detectable with conventional neuroimaging,¹⁴ a full clinical recovery is expected for most patients,¹⁵ and recovery typically occurs within 3 months of injury¹⁶ and often sooner.¹⁷ In the latter, permanent, readily visible damage is a cardinal feature;¹⁸ a full clinical recovery without persisting cognitive, motor, or personality change is rare;¹⁹ and an obvious plateau in recovery is not reached until at least 6 to 12 months

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endure decades of disability because of the young demographic affected,² and it impedes return to work and school,^{3,4} disrupts family and social networks, and causes increased social isolation, mood disturbance, and even suicide.⁵⁻⁷ These disabilities are the result of a range of cognitive, motor, sensory-perceptual, and emotional disturbances, and these various impairments can combine in complex ways, necessitating an interprofessional approach to rehabilitation in order to minimize their impact. For example, cognitive deficits may result in poorer insight into changes in a person's hearing; emotional changes may affect one's initiative regarding many forms of self-care; motor deficits may make travel to clinical appointments harder and less likely.

Therefore, patients with TBI represent a distinctive and challenging population for hearing healthcare practitioners. It is important for them to be aware of the vulnerability of people with TBI, including their diminished capacity to practice optimal healthcare and, in many cases, the absence of family and social networks to provide secondary healthcare support. The purpose of this paper is to provide increased understanding of TBI,

post-injury,²⁰ although slower and less perceptible recovery may continue thereafter for years.¹⁹

Because behavioral impairments can affect the hearing assessment, the hearing care provider should make use of information that may be available in reports or letters of referral regarding prior neuropsychological assessments or disability assessments (e.g., the Ranchos Los Amigos Scale,²¹ the Functional Independence Measure,²² the Disability Rating Scale²³ and the Glasgow Outcome Scale.¹¹) However, the clinician must also be mindful of the date of assessment. During the early months of rapid recovery, a report that is only a few weeks old will be out of date and potentially irrelevant. In the rehabilitation setting, hearing assessments are often requested by the treating therapists when their own assessments reveal suspected hearing changes. Thus, the timing of the hearing assessment is typically linked to these other impairment and disability assessments.

RISK FACTORS FOR TBI

The two leading causes of TBI are falls and motor vehicle accidents.²⁴ A number of factors have been identified as increasing the risk of sustaining a TBI. The age distribution of TBI is bimodal, with one peak between 15 and 24 years^{8,25,26} and a second peak in the elderly, largely attributable to falls. Impaired hearing is a risk factor for falls.²⁷ Thus, hearing impairments may play a causal role in TBIs in the elderly, given the commensurate increase in hearing impairment and fall-related TBIs in this age group. Males are 1.5–2 times more likely than females to suffer a TBI during the first peak,^{8,25,26} but among the elderly the incidence of TBI does not differ by gender.

There is also elevated risk in the very young (i.e., under 5 years).^{8,26} One may speculate that failure to respond to warning sounds plays a role. Other factors associated with increased risk for TBI include depression, anxiety, conduct disorders, lower socioeconomic status, unemployment, lower educational attainment, lower cognitive abilities, prior history of

TBI, and chronic alcohol and substance abuse.^{8,26} Those who are acutely intoxicated are at eight times the risk of sustaining a TBI than those who are not.²⁶

Poorer long-term clinical outcomes are related to the severity of the injury and demographic factors, some of which overlap with those above. Higher injury severity,²⁸ older age at the time of injury, lower premorbid educational level,^{29,30} history of previous injury,³¹ substance abuse,³² and premorbid psychiatric disorder³¹ all negatively affect clinical

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outcomes.¹¹ Cognitive impairments also predict outcome. For example, Green et al. recently found that the ability to remember organized, structured verbal information (e.g., a story or conversation as opposed to a list of unrelated words) and intact executive functioning are good early prognostic indicators for return to a productive lifestyle at one year.³³

MECHANISMS OF TBI

There are numerous mechanisms of injury to the brain, and injury to hearing structures after TBI may result from various causes, including mechanical injury, lost blood/oxygen supply to tissue, disconnection of one structure from another due to “diffuse axonal injury” (i.e., widespread damage to the axons of neurons), and excitotoxic damage or transneuronal degeneration, whereby an intact structure loses afferent projections from a damaged one.³⁴ Thus, injury mechanisms and neuropathology of TBI can be classified on a number of dimensions as follows:

Penetrating/Non-penetrating injury

Injuries may be penetrating (open-head injury) or non-penetrating (closed-head injury). A penetrating TBI occurs when an object or bone from the skull penetrates

the brain. This typically results from gunshot and knife injuries⁹ as well as workplace accidents. Non-penetrating injury refers to injury to the brain without opening of the skull.

Focal (rapid impact vs. slow crush)/Diffuse Injury

Both penetrating and non-penetrating injuries may occur when the head collides with an object. This may happen suddenly, as in the case of a fall, assault, sports injury, or the head striking the dashboard in a moving vehicle accident, or slowly, as in a crush injury.³⁵

It is important for clinicians to be aware that non-penetrating injuries may occur without a direct blow to the head,⁹ as in the case of a seat-belt-wearing passenger in an auto accident or a soldier within the blast field of a

bomb.³⁵⁻³⁷ Here, injury to the brain is caused by the brain bouncing inside the head against the bony calvarium resulting in contusions and hemorrhages and, more importantly, by acceleration, deceleration, and rotational forces that compromise the axons and microvasculature.

These forces, which are generally associated with the inertial forces common to motor vehicle accidents and falls from greater than one's own height,³⁸ cause stretching and distortion of axons (of neurons)^{39,40} known as diffuse axonal injury (DAI) as well as widespread petechial hemorrhage.⁴⁰ Thus, they give rise to micro-injuries distributed over broad areas of the brain.⁴¹ In a serious brain injury, diffuse injury typically occurs with concomitant focal injuries.^{40,41} Focal injury is used to refer to discrete, larger lesions greater than 1.6 mm³.⁴² Focal injury may be caused by a penetrating injury to a circumscribed area or by blunt force causing injuries near the surface of the brain.^{41,43}

Acute primary/Acute secondary/Sub-acute

Initial impact injury is usually referred to as “primary injury.” So-called “secondary injury” refers to the evolution of these injuries over time, involving hypometabolism, white matter degeneration,

or “Wallerian degeneration,” whereby damaged axons and myelin slowly degenerate and are removed^{44,45} and in some cases result in total neuronal death.

Secondary injury is triggered by biochemical mechanisms^{44,46-48} as well as by mechanical axonal injury that can lead to disruption of axonal transport, accumulation of proteins, axonal swelling, and eventual axotomy.^{41,49} The effects of secondary injury on neuroimaging tend to take longer to manifest and are eventually observed on CT and conventional MRI as encephalomalacia.¹⁸

There is also accumulating evidence that a second wave of damage occurs over the longer term, after the acute neurological events have resolved.⁵⁰⁻⁵²

TBI-RELATED HEARING LOSS

Overview of auditory pathways

In the auditory system, sound waves travel initially through the external and middle ear. Hearing loss associated with injury to the middle ear is referred to as conductive hearing loss. When sound waves reach the cochlea, they are converted to electrical signals within the hair cells of the organ of Corti. Hearing loss from the cochlea onwards is referred to as sensorineural hearing loss. From the cochlea, sound information is transmitted from the hair cells to the auditory nerve to cranial nerve VIII (the vestibulocochlear nerve) and into the brainstem of the central nervous system (CNS).

Auditory structures within the brainstem and between the brainstem and midbrain are connected by the lateral lemniscus, a major white matter tract. Via this tract sound information travels from the brainstem to the midbrain (i.e., the inferior colliculi). From the midbrain, sound information is transmitted up to the auditory cortex of the temporal lobes via the thalamus.

All of these CNS structures and pathways are vulnerable to penetrating and non-penetrating injuries. The auditory cortex of the temporal lobes is particularly vulnerable to impact injury. More deeply buried structures in the brainstem and midbrain are vulnerable to

functional disconnection due to diffuse axonal injury (particularly of the lateral lemniscus) as well as excitotoxic injury and transneuronal degeneration.⁵³

Injury to the middle and external ear

Trauma and infection associated with TBI can give rise to disorders of the external and middle ear, which usually

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produce a conductive hearing loss by interfering with mechanical transmission,⁵⁴ for example through thickening or perforation of the tympanic membrane. Head injury and blast injury can also cause otitis media by damaging the otic capsule or the tissues separating the perilymph from the middle ear.⁵⁵

Rupture of the membranous barrier between the middle ear and inner ear at the oval or round windows may result in leakage of fluid from the inner ear. Such a perilymphatic fistula may cause progressive or fluctuating sensorineural loss as well as vestibular symptoms. Non-penetrating head injuries may cause more damage to the membranes than penetrating head injuries and TBIs since the accelerating forces may not be readily dissipated, as they are when the skull is fractured or the brain is actually damaged.⁵⁵

Injury to the cochlea and cranial nerve VIII

The hair cells are the most vulnerable elements in the cochlea,⁵⁶ being subject to damage by necrotic and apoptotic mechanisms of TBI⁵⁷ as well as excitotoxic injury.⁵⁸ Cranial nerve VIII is also vulnerable to the consequences of TBI, particularly pressure due to edema or hematoma. Pressure may give rise to tinnitus due to imbalanced firing patterns in the auditory nerve.⁵⁹ Hearing loss and decreased speech discrimination may also arise due to lost temporal coherence.⁶⁰ The hearing loss here can be identified by loss in the mid-frequency range.⁶¹

Central nervous system injury

A range of hearing symptoms may arise from lesions of the CNS. Auditory hallucinations, whereby meaningful sounds such as music or speech are hallucinated, can be caused by damage to the auditory cortex of the temporal lobe (as well as damage to the medial geniculate body of the thalamus). In addition, temporal lobe insults generally result in auditory agnosia, which involves aberrant interpretation of sound.⁶²

Damage to deeper structures also gives rise to distinctive behavioral effects. For example, the ventral medial geniculate body is associated with the relay of frequency, intensity, and binaural information up to the cortex. Thus, damage to this region would be suspected with loss of this information following TBI.

Damage to the inferior colliculus may cause changes in hearing by affecting the frequency tuning, which may not cause actual hearing loss, but rather increased frequency selectivity and heightened sensitivity to soft sounds.^{61,62} Compromised integration of information from both ears and compromised directional hearing may be caused by damage to the superior olivary complex of the brainstem, which sub-serves these functions by comparing the arrival time and intensity differences of neural activity from both ears.

CLINICAL CONSIDERATIONS

The challenges for healthcare practitioners working with patients with moderate/severe TBI are numerous. First, there is the difficulty of diagnosing the cause of hearing deficits. While conventional neuroimaging can help identify focal cortical and sub-cortical lesions, DAI or atrophy of sub-cortical structures secondary to excitotoxic injury or transneuronal degeneration cannot be readily visualized. Therefore, diagnosis must be made on the basis of behavioral findings. However, given the confounding effects of cognitive deficits that may affect test taking, this is a complex challenge requiring the use of information from many sources.

Here, the rehabilitation setting offers distinct advantages. First, a comprehensive

neuropsychological assessment (or an assessment of some aspects of cognition or cognitive communication by occupational therapy or speech-language pathology) may be available to shed light on cognitive deficits that could confound the hearing assessment or be confounded by hearing loss. In the absence of formal

earlier. Tests that rely less heavily on cooperation from patients, such as event-related potentials, would likely be much less vulnerable to these confounds.

Cognitive impairments may also affect treatment. A person with TBI may require interventions for hearing

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assessment, the hearing clinician also has the advantage of informal communication with therapists working with the TBI patient.

Relevant deficits include attentional disorders and speed of processing deficits, which may affect response to hearing tests. It may be difficult to determine whether the absence of behavioral responses is due to test-taking difficulties or to hearing loss. Language comprehension deficits can similarly affect results of assessment. Memory or tracking deficits—whereby a patient understands and initiates a test correctly, but fails to remain on task over time—is another source of potential complications during testing.

In the community, the practitioner will ideally have reports from acute care and in-patient care. However, these may be less informative and, moreover, out of date if they were written while the patient was actively recovering, as discussed

loss, infection, tinnitus, or ear pain. However, TBI may compromise the patient’s ability to understand and remember the procedures required for the intervention. Simple language, repetition, written instructions (when a patient has no reading deficits), and demonstration of any technique by the practitioner followed by hands-on practice by the patient will be of value. Also, family support is always critical. Again, in the rehabilitation setting, there is a distinct advantage. Nurses and therapists can work with the patient to help them to learn and remember the intervention. They can also report to the hearing practitioner on the efficacy of the intervention.

IN CONCLUSION

Traumatic brain injury is a common neurological disorder, and compromise to hearing may be a consequence. Increased awareness and understanding of TBI can improve hearing healthcare for TBI patients and thereby improve their quality of life.

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