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REVIEW

# Mild Traumatic Brain Injury and the Auditory System: An Overview of the Mechanisms, Clinical Presentations, and Current Diagnostic Modalities

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**Abstract**

The acute and long-term consequences of mild traumatic brain injury (mTBI) are far reaching. Though it may often be overlooked due to the now expansive field of research dedicated to understanding the consequences of mTBI on the brain, recent work has revealed that substantial changes in the vestibulo-auditory system can also occur due to mTBI. These changes, termed "labyrinthine" or "cochlear concussion," include hearing loss, vertigo, and tinnitus that develop after mTBI in the setting of an intact bony labyrinthine capsule (as detected on imaging). In the review that follows, we focus our discussion on the effects of mTBI on the peripheral structures and pathways of the auditory and vestibular systems. Although the effects of indirect trauma (e.g., noise and blast trauma) have been well-investigated, there exists a profound need to improve our understanding of the effects of direct head injury (such as mTBI) on the auditory and vestibular systems. Our aim is to summarize the current evidentiary foundation upon which labyrinthine and/or cochlear concussion are based to shed light on the ways in which clinicians can refine the existing modalities used to diagnose and treat patients experiencing mTBI as it relates to hearing and balance.

**Keywords:** cochlear concussion; hearing loss; mild traumatic brain injury; tinnitus; vertigo

## Introduction

Approximately 4.8 million Americans are evaluated for traumatic brain injury (TBI) every year, with 85% of these individuals experiencing mild TBI (mTBI).[1,2] After sustaining mTBI, patients can have continued impairment including headache, anxiety, depression, anger, substance use disorder, dementia, and suicidal ideation.[1] It is estimated that 53% of those who experience mTBI report continued impairment at 1 year post-injury.[3] The lasting impact of mTBI and the myriad neuropsychiatric effects it can induce make it an important area of research. For example, mTBI has been studied for its association with neurodegenerative disease and chronic cognitive impairment as well as persistent post-concussion syndrome. As a result of the ongoing Transforming Research and Clinical Knowledge in Traumatic Brain Injury (Track-TBI) study, we are now on the forefront of major advances in terms of leveraging the compromised blood–brain barrier in mTBI to identify blood biomarkers that can help distinguish concussion from

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sub-concussive head injuries, and overall degree of mTBI severity, and predict recovery following mTBI. Although these have been major areas of focus in recent years, emerging evidence suggests that the otological system—namely the vestibular apparatus and cochlea—may play a role in symptomatic mTBI, and it has already been shown that alterations in auditory function are associated with severity of clinical course in patients with mTBI.[4]

Hearing loss, tinnitus, and hyperacusis are all common side effects of mTBI yet can be difficult for clinicians who are managing patients with mTBI to detect.[4] To date, most studies investigating auditory dysfunction in the setting of mTBI have focused on indirect noise-related blast injuries, particularly among current members and veterans of the U.S. military.[5–8] However, direct head-trauma-induced mTBI may also cause auditory dysfunction. The incidence of hearing loss post-TBI reportedly ranges between 7% and 50%, with the authors reporting high-frequency sensorineural hearing loss (SNHL) in 10–24% of cases.[9,10] These results are concordant with previous studies such as Griffiths' investigation, which found a 56% incidence of hearing loss in patients following TBI.[11] The clinical signs of mTBI-associated auditory effects can be difficult to detect in mTBI due to the complexities of the functional alterations in the auditory pathway. Further, auditory disturbances are not the only effects related to mTBI-induced inner ear dysfunction, as vestibular manifestations can add an additional layer of complexity to this pathological phenomenon. Unfortunately, it is still unclear how often mTBI leads to vestibular dysfunction, as Fausti and associate reported presence of vertigo in only 15% of patients, which is markedly lower than the 95% incidence reported by Davies and Luxon.[12,13]

In this review, we investigate changes that occur within the vestibular and auditory systems in the setting of mTBI-associated cochlear concussion, a phenomenon characterized by functional impairment that is not mirrored by any detectable damage to the structures of the inner ear. Notably, deficiencies in the auditory or vestibular systems are clinically important and can significantly alter patient quality of life and ability to perform activities of daily living. Ultimately, this review aims to promote awareness of cochlear (or labyrinthine) concussion among clinician-scientists so that they may seek ways to refine existing diagnostic tools and treatments for patients who sustain mTBI and subsequently experience symptoms of vestibular and/or auditory impairment.

## Methods

To identify studies describing the associated mechanisms, clinical signs and symptoms, and diagnostic techniques and/or modalities used to diagnose cochlear concussion and/or auditory mTBI, we first performed a search of the PubMed, Scopus, and Web of Science databases using the following Boolean search term: [(mild TBI OR mTBI OR mild traumatic brain injury OR blast trauma OR cochlear concussion OR labyrinthine concussion) AND (vestibulo-auditory OR vestibular OR vestibule OR auditory OR cochlear OR hearing OR tinnitus OR vertigo)]. Following removal of duplicates, remaining studies were subject to title and abstract screen, which was performed by two authors (R.S., N.J.B.). Remaining articles underwent full text screen to determine their suitability for incorporation into the present review. References for each study were screened to identify any studies that might not have been identified during our initial query. Ultimately, reference screen did not yield any additional studies. From the final list of included studies, we highlighted the main points from each to synthesize a concise narrative such that it may serve as a useful reference for clinicians who might otherwise not be familiar with the clinical syndrome involving vestibular and/or auditory impairment in the setting of mTBI. Although it is likely under-diagnosed, we hope to increase awareness of this disorder and equip clinicians with the baseline knowledge necessary to detect mTBI with concurrent vestibulo-auditory symptoms so that it may be properly recognized and diagnosed by a wide breadth of health care providers.

## Mechanism of Noise and Blast Injury Mediated Effects on the Auditory System

### Anatomy and function of the auditory system: A brief introduction

Following movement through the outer ear, sound waves reach the tympanic membrane, commonly known as the eardrum, at which point they are transmitted to the middle ear. Vibrations from the tympanic membrane induce movement of the malleus, incus, and stapes, the three small bones within the middle ear that amplify vibrational sound energy and transfer it to the cochlea (inner ear) via the oval window (Fig. 1). The oval window serves as a fluid cushion that transmits the sound energy to the cochlear fluid, and this energy is dissipated as necessary through decompression at the round window (a small cochlear aperture that opens and closes as sound waves strike the oval window to keep inner ear pressure within normal limits).

The cochlea ultimately contains special mechanoreceptors called hair cells that convert sound energy to electrical energy, which is then relayed to the central nervous system by peripheral nerves. Specifically, inner hair cells (IHCs) overlying the basilar membrane within the organ of Corti contain stretch mechanoreceptors that are activated when sound waves induce vibration of the basilar membrane, bringing them into contact with the overlying tectorial membrane. Activation occurs any

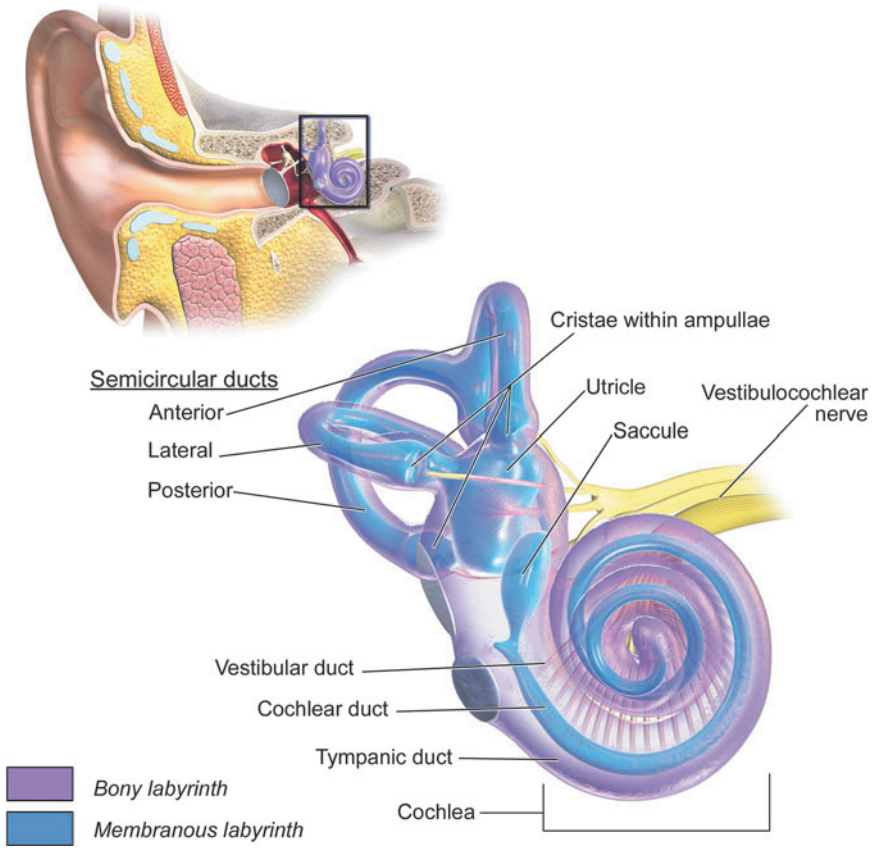


**FIG. 1.** The inner ear. Reprinted from: Blausen.com staff (2014). Medical gallery of Blausen Medical 2014. Wikidata Q44276831. ISSN 2002-4436. WikiJournal Med 1(2); doi: 10.15347/wjm/2014.010. Color image is available online.

time the hair cells brush up against the tectorial membrane. The electrical signal generated is transferred first to the spiral ganglion followed by auditory nerve fibers, which conduct the signal to the cochlear division of cranial nerve (CN) VIII, and ultimately to the cochlear nucleus within the ipsilateral brainstem. From the cochlear nucleus, many neurons cross over to the contralateral superior olivary nucleus, whereas some synapse within the ipsilateral superior olivary nucleus. From the superior olives, ascending tracts make their way to the inferior colliculus and ultimately to the medial geniculate nucleus of the thalamus, from which point they are relayed to the primary auditory cortex of the temporal lobe.

Other important structures of the inner ear include the utricle and saccule, which are components of the vestibular apparatus. These otolith organs contain small crystalline calcium carbonate stones referred to as otoconia that lie in suspension within a viscous fluid termed ''endolymph.'' Linear acceleration of the head causes movement of the stones, which puts pressure on the cilia of the hair cells overlying the utricle and saccule, inducing mechanotransmission of signals to the vestibular branch of CN VIII. The utricle and saccule work in tandem with the three semicircular canals—the components of the vestibular system responsible for detecting angular acceleration—to maintain the body's sense of equilibrium (balance) through coordination of eye, head, and body movements.

## Direct neurodegeneration from high-intensity signals

Recent animal studies have revealed that mild noise exposure/injury can result in reversible threshold shifts that are associated with auditory neurodegeneration. Hidden hearing loss may also occur and is characterized by synaptopathy, followed by neural axonal loss and neuronal degeneration in the setting of full recovery of cochlear thresholds.[14–16] The hidden hearing loss paradigm has been demonstrated in mouse and guinea pig models, which show dramatically diminished auditory brainstem response (ABR) wave-I outputs in animals subjected to noise-induced; these responses involved temporary threshold shifts.[15] Following intense noise

exposure with reversible threshold shifts in the animal model, there is an immediate and irreversible loss of approximately 50% of the afferent synapses of IHCs, despite no loss of the IHCs themselves. Although the hidden hearing loss paradigm has yet to be validated in humans directly, a similar phenomenon may underlie tinnitus, hyperacusis, and other forms of anomalous auditory signal processing. Recent translational and clinical studies have also demonstrated measurable reductions in cochlear nerve output in humans who have a history of pathological noise exposure and chronic tinnitus.[17,18]

Further, experimentation in human subjects has suggested that normal audiometric testing and the presence of intact IHCs does not rule out the possibility that noise-induced damage has occurred within the auditory nervous system. However, in a study that investigated individuals suspected to be at high-risk for auditory damage due to an extensive history of sustained noise exposure, it was found that these high-risk subjects had no difference in audiometric thresholds at standard frequencies compared with low-risk individuals.[19] Despite this, after pre-synaptic summation potentials (SPs) and action potentials (APs) of the cochlear nerve fibers were measured via ABR wave-I outputs, it was found that the SP/AP ratio was significantly higher within the high-risk ear damage group, consistent with the presence of cochlear nerve degeneration within these subjects. This corroborates results from previous studies that have displayed reduced auditory nerve output in patients with histories of pathological noise exposure and clinically diagnosed hearing disorders. Previous ABR wave-I studies have also demonstrated reduced auditory nerve output in tinnitus subjects relative to control subjects; the tinnitus subjects reportedly displayed normal audiometric threshold measurements despite the presence of tinnitus.[20] As these studies suggest, noise exposure and injury may induce lasting pathological effects on the human auditory system, despite the fact that audiometric threshold testing is otherwise normal. Because this pathological picture is very unclear, it should be investigated to a greater extent in populations that have been subjected to potentially damaging noise exposures.

### Blast trauma

Although it differs from auditory mTBI, the mechanism of blast trauma injury mirrors that of mTBI and may help elucidate information on mTBI pathogenesis. Authors have reported that significant threshold shifts on audiometry, in addition to the symptoms of tinnitus, are associated with blast-induced TBI in military personnel.[21] Additional investigations into ABR threshold changes post-blast exposure in rats have found temporary threshold shifts leading to dramatic ABR reduction indicative of cochlear dysfunction resulting from singular insults.[22–24] These insults were found to have the largest ABR threshold shift on post-insult day 1, with a gradual return to baseline at post-insult day 14. By contrast, blast-exposed veterans with clinically normal hearing have been shown to have auditory problems.[25]

Further, structural deformities and extreme acoustic trauma have been reported in a variety of animal models exposed to blast trauma. For example, shock pulses transmitted through the labyrinthine fluid in cats resulted in spiral ganglion cell (SGC) and IHC loss in the middle cochlear turns.[26] Similar damage was noted by Cho and co-workers from the effects of high-pressure waves on outer hair cells (OHCs) at the basilar end of the cochlea in mice.[27] Additionally, membrane leakage of OHCs secondary to nuclear condensation, directly caused by mechanical damage, has been observed in guinea pig models.[28] Experiments have also been conducted highlighting the effect of blast trauma versus non-blast acoustic trauma in rats, finding that blast-injured subjects presented with more significant and prolonged central and peripheral auditory deficits.[29] These findings elucidate potential mechanisms for trauma-induced SNHL in humans. Although most of the literature examines the effects of indirect trauma on the auditory system, recent studies investigating the effects of head trauma on cochlear function have yielded robust animal and human findings. We provide an overview of these findings in the sections that follow.

## Effects of mTBI on the Auditory System

### Demographics of auditory issues in mTBI

mTBI may contribute to hearing loss, tinnitus, and difficulty with understanding speech in noisy environments that is independent of hearing loss from high signal intensity exposure or blast trauma exposure.[30] mTBI commonly results from motor vehicle accidents, sport injuries, work-related incidents, falls, and blast-related head injury.[12,31,32] A recent study observed a strong correlation between auditory dysfunction and post-concussion symptoms irrespective of cause.[33] However, the precise mechanisms by which mTBI affects the auditory system are poorly understood. Damage to the central and peripheral auditory pathways could occur through overt physical trauma via shearing of CN VIII, endolymphatic duct disruption, or labyrinthine fluid shock pulses that injure the sensory epithelium of the vestibulocochlear apparatus.[9] Permanent hearing loss may also result from the rupture of the oval or round windows secondary to trauma.[34] Authors have estimated that approximately 34–50% of patients experiencing mTBI develop SNHL, tinnitus, or vestibular symptoms of vertigo or postural imbalance.[35–37] Patients may experience symptoms for variable amounts of time with some reports describing cases of persistent vertigo for 5 years and hearing loss for 18 years post-injury.[9,11,36,38]

### Histomorphological changes

Histological changes following cochlear concussion may include hair cell damage, organ of Corti degeneration, basilar membrane shearing, and auditory nerve fiber avulsion, which would explain the absence of clinically detectable damage to the organs of the inner ear on imaging.[9] Further, experiments measuring the effects of intense noise exposure on cochlear rat tissue have shown a significant correlation between these exposures and ICH auditory nerve fiber counts. The differences in the immunohistological intensity of synaptic ribbons (LH1) between noise-exposed versus non-exposed rats were quantified and demonstrated markedly decreased counts in the exposed subjects.[39] Overall, these observations were consistent with previous animal models and suggest that blast-trauma-mediated auditory effects evoke a noise-exposure-induced response that occurs primarily at the neuronal level.[40]

Additionally, in the previously mentioned studies regarding blast-trauma-induced injuries in the animal model, experiments also revealed changes in auditory function when direct head trauma was the source of mTBI. In an experimental group of cats that had sustained mild head injuries, a microscopic view of hematoxylin and eosin (H&E) stained vestibular tissue displayed structural changes. Specifically, a 30% decrease in hair cells, reduction in cochlear ganglion neurons, and deterioration of the saccular wall were observed. A view of the basilar membrane exhibited a decrease in ganglion cells. Depending on the severity of the concussion, the effects on auditory cell structures varied from subtle alteration of shape (e.g., short and wider hair cells, and smaller nuclei than normal) to complete disappearance of hair cells.[41,42] Interestingly, there was not always a direct correlation between the severity of the induced blow, hearing loss intensity, or severity of histological alterations.

### Auditory brainstem responses

In humans who have previously sustained mTBI, such as current and former military service members,[43] delayed latencies for ABR waves I, III, and V have been observed alongside reductions in peak amplitudes and latencies of speech ABR.[44,45] However, Nolle and colleagues,[46] Fausti and associates,[12] and Vander Werff and Rieger[44] reported no difference in ABR measurement of blast-induced or physical mTBI subjects compared with normal patients. Nonetheless, select mTBI individuals who suffered from central auditory processing deficiencies demonstrated a deviation (LH2) from normal ABR measurements, indicative of central auditory processing deficits. This included delayed ABR latencies and reduction in various peak amplitudes, particularly in speech-evoked ABR waves among mTBI subjects.[12,44,46] Moreover, the sub-category of patients with mTBI with abnormal behavioral auditory results showed differences in speech ABR onset.[44]

An observed increase in peak latency in ABR may be utilized to assess brainstem conduction time and could indicate the presence of a brainstem lesion. In a high-level fluid-percussion injury feline model, brainstem dysfunction was measured using brainstem auditory evoked responses (BAERs) in sub-groups based on the magnitude of the neuropathological injury. Animals with mild intraparenchymal and subarachnoid hemorrhages experienced augmented latencies for waves II, III, and IV between 60 and 150 min post-injury, but returned to baseline at 8 h. However, animal models of chronic intraparenchymal and subarachnoid hemorrhages exhibited poor recovery to baseline assessment,[47] indicating the severity of brain injury was associated with the severity of auditory deficit. Additionally, at chronic levels of injury, differences in BAERs were observed in a rodent TBI-induced lateral fluid-percussion model with wave suppression correlating with greater post-injury hemorrhage, vascular disruption, cavitation, and neurological deficit.[48,49] In another study, mice that received a single impact directly to the cortex experienced abnormal ABRs 2 weeks after injury with deficits lasting for up to 14 weeks post-injury. These mice exhibited a significant reduction in ABR amplitude of waves I and IV, coupled with significantly increased latency responses measured at 2, 6, and 14 weeks post-mTBI compared with controls.[50,51] This pioneering study demonstrated that mTBI can result in deficits in hearing and temporal processing when tested at high frequencies.[50,51]

### Late latency responses

In addition to an ABR, late latency responses (LLRs), which represent the activity at higher levels of processing within the auditory system, may be useful in assessing patients with mTBI. This primary auditory cortex response is significantly inhibited in repeatedly concussed athletes.[52] Consequently, future prospective studies of electrophysiological measurements such as ABR and LLR can delineate objective ranges for diagnosis and management of mTBI to identify the nature and extent of auditory damage.[46]

### Speech comprehension

Individuals with mTBI can also experience deficits in speech comprehension. These patients are more likely to present with speech-in-noise abnormalities than control patients matched for age and pure-tone threshold.[53,54] Lew and colleagues described the case of a 55-year-old male with speech comprehension difficulties post-TBI and found that behavioral audiometry and Weber/Rinne tests did not provide an accurate assessment of function. Instead, brainstem auditory-evoked potentials revealed bilateral deafness, confirmed with a computed tomography (CT) scan of the bilateral temporal lobes indicating bilateral temporal transverse bone fractures.[55] However, there is no consensus on an objective test to confidently

identify auditory issues in mTBI despite the promise of tests of frontal lobe function such as the Controlled Oral Word Association Test and Paced Auditory Serial Addition Task.[56]

### Fluid biomarkers

With the recent approval of glial fibrillary acidic protein (GFAP) and ubiquitin carboxy-terminal hydrolase L1 (UCH-L1) plasma levels by the U.S. Food and Drug Administration (FDA) for determining the need for brain CT in patients with TBI, the field of biomarkers in TBI is increasing in importance.[57] Although most biomarkers focus on blood-based and cerebrospinal fluid (CSF) identification tools, Kraus and colleagues used the frequency following response (FFR) as a novel marker for concussion.[58] The FFR, also described in literature as the auditory brainstem response to complex sounds, is generated by the auditory midbrain and reflects the encoding efficiency of sound. Kraus and her research team sought to understand whether processing of the fundamental frequency, a common acoustic cue measured by the FFR, could be used to identify cases of concussion among children. Notably, they found that children diagnosed with a concussion had unique neural signatures with lesser representation of the fundamental frequency and deemed this investigation a successful deployment of a clinical model for concussion diagnosis with a 90% sensitivity and 95% specificity. The FFR presents a unique paradigm for mTBI biomarker research due to its relative objectivity and ability to concurrently track recovery, potentially holding immense clinical applications in management planning.[58] Kraus and colleagues noted that future efforts may look to integrate an understanding of behavioral consequences, such as disruptions to memory and attention, into clinical FFR applications. In this way, the FFR serves as a practical, quantitative method of indicating both the presence and severity of an mTBI.

### Clinical presentations

Patients with mTBI exhibit clinically significant heterogeneity, with variable presentation and severity of symptoms related to auditory issues.[59] Some patients may immediately exhibit deterioration of hearing and vestibular function after head injury that may become either transient or permanent.[9] Other symptoms, such as tinnitus, may be delayed and increased in severity with repeated mTBI.[41,60] Hearing loss following head trauma can occur in the absence of bony labyrinth injury and hearing defects are most noticeable at higher frequencies.[61]

Because the inner ear houses a small volume of fluid, errant blood and debris can easily cause obstructive hydrops. Head trauma can often lead to signs characteristic of Ménière's disease, with symptoms such as vertigo, tinnitus, and hearing loss.[62,63] Clinical studies have demonstrated that damage to the endolymphatic duct can cause endolymphatic hydrops and emulate the pathogenesis of Ménière's disease.[64] This traumatic Ménière's syndrome often occurs with delayed presentation and the symptom-free period could last up to several years.[9] Additionally, vestibular deficiencies are more common and pronounced than auditory deficits in post-traumatic Ménière's syndrome when compared with typical Ménière's disease.[62,63,65]

A traumatic fracture in the temporal bone can also lead to a perilymph fistula (PLF), which is difficult to differentiate from Ménière's disease. PLFs are believed to result from tears in the round window membrane or stapes footplate attachment, causing spillage of perilymph fluid into the middle ear. Trauma-induced PLF is more likely to cause hearing-loss symptoms than vestibular symptoms and often presents as a fistula of the round window rather than the oval window.[9] The precipitating injury is either explosive or implosive. In the former, head trauma abruptly increases CSF pressure, increasing perilymph pressure and rupturing the round or oval windows. In the latter, direct external trauma to the ear causes tympanic membrane implosion and medial displacement of the stapes footplate, resulting in oval or round window rupture.[66] Although symptoms of PLFs typically begin between 24 and 70 h following the injury, in some cases symptoms may not manifest until long after the inciting event.[9]

Additionally, patients with mTBI may present with a variety of central vestibular disorders ranging from oculomotor abnormalities to migraines. Following mild head injury, oculomotor abnormalities may impact up to 90% of patients.[67] However, many of these oculomotor findings resolve within 6 months.[68,69] In addition, migraine disorders can present after mild head injury and can often be complicated by post-concussive symptoms. Another common consequence of mild head injury is vestibular migraine, a diagnosis distinct from primary migraine disorders. Although treated similarly, the former typically entails episodes of dizziness described as a rocking sensation. Shared symptoms, particularly headache, do not always manifest in conjunction with vertigo. Further, exacerbation of symptom frequency has been reported in up to 30% of patients over a decade. The nuanced presentation in addition to the concerning progression necessitates careful consideration of symptoms signaling vestibular migraine in patients with mTBI.[67,70]

### The current state of imaging modalities

In the clinical setting, observation and diagnosis of post-TBI auditory damage is aided by a variety of imaging modalities including CT, magnetic resonance imaging (MRI), and recently, optical coherence tomography (OCT).[71] Multi-detector CT (MDCT) may identify injury to crucial structures such as the petrous portion of the temporal bone, otic capsule, ossicles, and vasculature

by imaging multiple tissue slices simultaneously with higher speed and resolution than standard CT. Injury to these structures may lead to SNHL, conductive hearing loss (CHL), vertigo, PLF, CSF leaks, facial nerve palsy, and vascular injury.[71] Dahyia and associates and Jani and co-workers demonstrated that fractures that damaged the otic capsule doubled the risk of facial paralysis, quadrupled the risk of CSF leak, and resulted in a seven times increased risk for hearing loss.[72,73] Recently, Yuh and colleagues demonstrated CT pathology for mTBI carries different prognostic implications at 1 year post-injury.[74] Further analysis should be conducted into neuro-imaging to determine more clinical features that may be indicative of auditory issues and recovery.

MRIs have utility for certain presentations of TBI.[73] MRIs can show lesions indicative of post-TBI hearing loss, as hemorrhage in the inferior colliculus and the pons correlate with deafness.[75] Enhanced MRI can also detect PLFs by demonstrating middle ear bleeding after round window perforation. Contrast-enhanced MRI with gadolinium allows identification of inflammation in the basilar membrane commonly seen after PLF.[9] Similarly, functional MRI (fMRI) changes include hypoactivation of regions such as the bilateral posterior cingulate gyrus, thalamus, striatum, midbrain nuclei, and cerebellum that are associated with behavioral auditory deficits.[76.77] Repeat concussions are associated with alterations in the verbal encoding system that may damage higher-order semantic networks that have been demonstrated on fMRI.[78] Moreover, hypoactivation on fMRI of the auditory cortex has been observed in patients with mTBI when they have been observed performing a listening task.[79]

Recent studies in biomedical optics have investigated the utility of OCT in assessing cochlear damage. OCT is a broadband laser traditionally used in ophthalmology that analyzes interference fringes created by backscattering electromagnetic waves with high, near histopathological resolution and low optical penetration depth. These advantages allow imaging of subepithelial structures. OCT may potentially provide a simple and minimally invasive procedure to image internal microstructures of the cochlea and diagnose inner ear pathology in the clinical setting.[80] Imaging has helped clinicians better understand the underlying mechanisms related to hearing deficits, but more research is required to understand the association between mTBI, specific hearing deficiencies, and specific imaging findings.

### Why is it important for clinicians to be familiar with auditory mTBI?

It is the authors' hope that in the near future auditory mTBI will be taught in didactic sessions throughout medical school and residency because it is a comorbidity that is ''hidden,'' unlikely to be reported, and not talked about, despite the fact that it can have a tremendous impact on patient quality of life. In reflecting on the implications that our lack of understanding about this disease process might have, we can imagine the case of a patient with concurrent psychiatric comorbidities and SNHL or tinnitus due to auditory mTBI. If such an individual is a veteran of the U.S. military, there is a chance that they might have concurrent post-traumatic stress disorder (PTSD), related psychiatric disorders, and auditory dysfunction. If auditory responses in mTBI remain incompletely understood, clinicians will not likely be able to make an accurate diagnosis. This could prove very frustrating to the military veteran with PTSD or the athlete post hundreds of concussions, each of whom may be unlikely to describe the hearing symptoms (or lack thereof) they are experiencing. There is a reason that such great care and detail is taken to preserving hearing in surgeries involving surgical resection and/or stereotactic radioherapy—the ability to hear the people and the world around us is both a right and a privilege that must not be taken for granted.

## Conclusions

A variety of auditory changes occur in response to mTBI, which may include peripheral, central, and vestibular components of the auditory system, with subsequent histomorphological and functional sequelae. Patients with mTBI present with significant clinical heterogeneity and auditory deficits can be difficult to detect due to the many potential types of injury. Hearing loss, tinnitus, or hyperacusis can arise from central or peripheral vestibular and/or auditory deficiencies and do not always arise immediately with injury. Although imaging is helpful, patients frequently only receive brain CTs at the time of injury, which are not as precise at picking up injury, and over half of these patients do not see a health care provider within 3 months of injury.[81] There is a limited understanding of the effects of mTBI on the auditory system and further investigations are necessary to elucidate the precise mechanisms involved in ''auditory TBI.''

## Transparency, Rigor, and Reproducibility Summary

The nature of the study as a literature review was not conducive to pre-registration, or any ethical board approval. There were no statistical methods utilized in the study. This study's rigor can be verified through examination of the content in the references. The study is completely reproducible through thorough knowledge and use of the references.

## Authors' Contributions

Authors' contributions are as follows. Mark Harris: conceptualization, investigation, writing original draft,

writing review and editing, project administration; Andrew Nguyen: writing original draft, writing review and editing; Nolan J. Brown: conceptualization, investigation, writing original draft, writing review and editing, project administration; Bryce Picton: writing original draft, writing review and editing; Julian Gendreau: writing original draft, writing review and editing; Nicholas Bui: writing review and editing; Ronald Sahyouni: writing original draft, writing review and editing, project administration; Harrison W. Lin: writing original draft, writing review and editing, project administration, supervision.

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## Author Disclosure Statement

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