Auditory dysfunction after Traumatic Brain Injury

Cause, evaluation and treatment

Kristina Sargénius Landahl
Specialistarbetsterapeut neurologisk utredning och intervention
Filosofi magister klinisk neurologi
Filosofi kandidat psykologi

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Preface

Patients with brain injury often complain about hyperacusis, hearing impairment, difficulty in listening especially in mild noisy environment and tinnitus. This literature review aim to providing a summary of current research in the field of auditory dysfunction after TBI. How can clinicians and other healthcare professional's best help these patients? What evaluations should be done? And what treatment should be proposed or offered?

Auditory dysfunction after TBI

Auditory dysfunction, which typically is a component of temporal bone fractures and basilar skull fractures, also occur in subjects without evidence of fracture. The otologic changes after TBI are varied, and depend on the type, magnitude and direction of trauma (1, 2). Studies of severely brain injured patients in intensive care settings show that about 70% of these patients has an otologic abnormality in one or both ears (3). Mechanical, pressure-related, and noise-related trauma can cause tinnitus (4) and hearing and labyrinthine dysfunction can occur as a consequences of damage to the auditory nerve, the cochlea, or middle ear conducting elements, including the tympanic membrane and ossicular chain (5).

Brain injury and injuries to the auditory nerve are well known risk factors for tinnitus, hyperacusis and difficulty in listening especially in mild noisy environment (2, 6-8). A majority of subjects with head trauma have resultant hearing impairment (10). About 50% develop tinnitus (3, 4), a symptom that can persist for a lifetime (5). The prevalence of sensorineural loss after TBI is about 25% (3) and approximately 75% has lowered loudness discomfort levels (LDL) (2). Other common symptom is abnormal facial sensory symptoms and aversion to loud/sudden noises due to cochlea damage (3).

Even relatively mild injuries can produce subtle impairments, which may go undiagnosed, especially in the early phase when more vital problems are in focus (1, 4, 9, 10). Patients are often unable to report their hearing deficits until they reach a certain level of cognitive awareness, and hence the diagnosis of this silent handicap may be delayed until late in the recovery phase (1). Even after minor head injuries there is a relatively high incidence of hearing impairment, especially in the higher frequencies, but the whole frequency range can be affected. According to Munjal (2010) 10–24% of patients with TBI have a high frequency sensory neural hearing loss. The harder the head blow and the more directly it hits the temporal bone, the more severe the damage and the more likely it is to involve the sensory neural mechanism (1).

Brain injury is also significantly associated with an increase in the risk of severe tinnitus (7). Direct effects of severe tinnitus include cognitive, emotional, and sleeping disorders— all of which can impact performance of everyday activities (11). Auditory dysfunction is also likely to impinge negatively on outcomes of therapy delivered by health professionals and hinder successful return to the community and re-entry to the workforce (3). Auditory dysfunction after TBI has become the most prevalent individual service-connected disability, with compensations totaling more than $1 billion annually in US Armed Forces (4).

The neural activity that causes most forms of tinnitus is generated in the nervous system with or without the involvement of the ear. Hypothesis on the pathophysiology of tinnitus generally concern neuroplastic changes in the central auditory system, probably initiated by some form of injuries to the cochlear sensory cells or to auditory nerve fibers (5, 6, 10). Inhibitory synapses seem to be more affected than excitatory synapses thus creating the basis for hypersensitivity and hyperactivity. Moreover, rerouting of information may cause structures of the CNS that are normally not involved in processing auditory information, to become activated by sound stimulation (6).

Evaluation of auditory dysfunction after TBI

Objective audiological assessment is essential to localize the site and extent of auditory impairment (2). Current rehabilitation assessment and follow-up procedures can fail to adequately identify the possible neuro-otologic sequelae of TBI. If only pure-tone hearing assessment is conducted it would be easy to dismiss the findings as relatively insignificant given that the hearing loss may be mild in the majority of TBI patients (3).

In research literature common audiological assessment after TBI consist of:

- Otoscopy (3, 8, 9, 11)
- Pure tone audiometry: determination of the degree, type and configuration of hearing loss (conductive, sensorineural and mixed) (1, 3, 7-9, 11, 12)
- Speech audiometry: measures the sensitivity for speech stimuli but also indicates the retro cochlear lesion (1, 9, 11)
- Tympanometry: a middle ear function test and measure of integrity of middle ear conduction mechanisms (1, 8, 9, 12)
- Acoustic reflex testing: aids in site of lesion information, especially at auditory crossover pathways (1, 9)
- Middle latency response audiometry (MLR): provide information regarding lesions at the primary auditory cortex (1)
- Transient optoacoustic emissions (TEOAEs), medial-cochlear suppression test (MOSE), spontaneous optoacoustic emissions (SOAEs), and auditory brain stem responses (ABR) (1, 2, 9)
- The loudness discomfort level (LDL): verify the level of hyperacusis (2, 11, 12)
- Immittance measures (11)
- Tinnitus evaluation consisting of tinnitus loudness, pitch matching, minimum masking levels and residual inhibition testing (11)

Cause of auditory dysfunction after TBI

Research shows altered neural activity in the central nervous system following TBI. Current assessment procedures therefor must incorporate understanding of auditory-cognitive disability to ensure optimal functional outcomes. Specific auditory evoked potential measures is needed to identify the exact site of lesion (3). In TBI-associated tinnitus, exclusion of treatable causes such as ossicular chain disruption and perilymphatic fistula also is recommended (4).

In research literature, otoscopy (2), tympanograms (1, 2, 9) and MRI scans of the brain, brainstem and medulla oblongata (2) commonly is normal after TBI. Puretone audiograms and auditory brainstem responses (ABR) also often are normal (2, 9) but an association has been found between the severity of TBI and high frequency hearing abnormalities and extent of damage at the central level (1).

Significant alterations between TBI patients with and without tinnitus have been found in auditory efferent activity and auditory and visual brain event related potentials. These findings strongly support the involvement of central auditory dysfunction in tinnitus generation after TBI (8). Research of otoemission suppression and stapedial reflex recordings (an acoustico-facial reflex that can be elicited in normally hearing subjects without central pathology (2)) reveal that the central auditory processing may be impaired by blunt trauma to the head as a result of diffuse axonal injury of the central auditory pathway (2, 9).

TEOAE response has been found to be altered in TBI patients with auditory disturbances (8, 9). Results from SOAEs indicate that for many TBI patients with tinnitus, medial olivocochlear suppression is reduced or absent in one or both ears. Dysfunction in the efferent control of cochlear mechanics (disinhibition of suppressive effect) is assumed to be the cause of higher TEOAE responses, high prevalence of SOAEs with many SOAE spectral peaks, and reduced or absent medial olivocochlear efferent suppression in TBI patients with tinnitus. This deficit may also lead to a reduction in the dynamic range of the cochlea, leading further to a reduced ability of fine tuning and to difficulty to extracting transient stimuli in background noise. This could be a possible explanation for the difficulty in listening in background noise (9).

Severity of TBI does not seem to affect the peripheral auditory system (middle ear transmission mechanism) to the same extent as the central (1). Trauma to the brain instead results in dysfunction of hippocampal-cholinergic systems, therefore causing MLR abnormalities. In TBI the lesions therefor may be present more frequently at the level above the brainstem (10). Research indicates that the major site of pathology in most head injury subjects is in the olivocochlear bundle and the auditory midbrain, brain parts essential for the processing of acoustic information (2, 9).

Tinnitus, hyperacusis and difficulty in listening in background noise—attributed to head injury and associated with normal peripheral auditory function and undamped otoacoustic emission, robust TEOAEs, and almost invariably recordable SOAE—constitute a clinical presentation which may be termed “disinhibition syndrome”, subsequent to central efferent auditory dysfunction (9). As in every other efferent sensory system, the efferent auditory system is thought to participate in regulation and feedback of activity in the central auditory system (1, 2, 8, 9, 12, 13). In humans, only the medial olivocochlear efferent system, a small part of the entire efferent auditory system, is routinely probed for its role in contralateral suppression of otoacoustic emissions (OAEs) (13). The medial olivocochlear system is classically considered to be inhibitory but evidence suggest that the system also enhances transient stimuli if they are presented against a continuous background noise (9). Through

control of the cochlea the system is hypothesized to aid in better detection of signals in noisy environments, to provide protection against acoustic trauma, and to be involved in selective attention (13).

The auditory efferent system can be assessed by recording the suppression effect on the transient OAE (TEOAE) (1, 2, 8, 12). Otoacoustic emissions represent unique tools for examining the cochlea, and have revolutionized clinical audiology by allowing a direct communication with the sensory cells. Otoemissions are typically found in subjects with normal hearing (a normal pure-tone audiogram), without central pathology (9) and reflects the protective mechanism against acoustic overstimulation that the olivocochlear bundle transmits and mediates in normal hearing (2). At the same time, the loss of a damping effect (attenuation) on afferent cochlear activity causes an increased response to auditory stimuli. This could be responsible for abnormal sensitivity to ordinary environmental sounds (hyperacusis). Increased auditory gain may also result in abnormal neural excitation, abnormal central sound processing, and tinnitus (9).

Treatment of tinnitus

Different forms of treatment have been shown to be beneficial to individuals with some forms of tinnitus. Numerous treatment are in clinical use such as tinnitus retraining therapy (TRT), tinnitus masking therapy (TM), tinnitus habituation therapy, tinnitus activities treatment and other forms of therapies that use counseling together with sound stimulation (4, 6). Significant improvements for both TRT and TM have been found when the interventions recently was compared (11). Moreover, electrical stimulation of the cerebral cortex has shown ability to alleviate some forms of tinnitus (4, 6)

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