Tinnitus after head injury: evidence from otoacoustic emissions

Borka J Ceranic, Deepak K Prasher, Ewa Raglan, Linda M Luxon

Abstract

Objective—Tinnitus may be caused by a lesion or dysfunction at any level of the auditory system. This study explores cochlear mechanics using otoacoustic emissions in patients with tinnitus after head injury, in whom there seems to be evidence to support dysfunction within the CNS.

Methods—The study included 20 patients with tinnitus and other auditory symptoms, such as hyperacusis and difficulty in listening in background noise, after head injury, in the presence of an “intact” auditory periphery (normal or near normal audiometric thresholds). They were compared with 20 normal subjects and 12 subjects with head injury, but without tinnitus, who had similar audiometric thresholds. In all subjects otoacoustic emissions, including transient click-evoked (TEOAEs) and spontaneous otoacoustic emissions (SOAEs), were recorded, and a test of efferent medial olivocochlear suppression, consisting of recording of TEOAEs under contralateral stimulation, was performed.

Results—A significantly higher prevalence of SOAEs (100%), higher TEOAE response amplitudes, and reduced medial olivocochlear suppression in patients with tinnitus in comparison with subjects without tinnitus have been found.

Conclusion—These findings have been interpreted to be an extracochlear phenomenon, in which the reduction in central efferent suppression of cochlear mechanics, leading to an increase in cochlear amplifier gain, was subsequent to head injury. Auditory symptoms in these patients seemed to constitute the “disinhibition syndrome”.

Keywords: tinnitus; head injury; cochlear mechanics; efferent suppression

Tinnitus is an auditory perception that is not caused by externally applied stimulation. It is assumed that tinnitus is a consequence of altered neural activity and may result from a lesion or dysfunction at any level of the auditory system. Therefore, the source of “tinnitogenic” activity could be anywhere in the auditory system, although it is thought to be most often located in the auditory periphery.

In some patients with tinnitus, there is a strong indication that the “tinnitogenic” signal, leading to the perception of tinnitus, results from a lesion within the CNS, and this presumption would be supported by the finding of “intact” peripheral auditory structures. Such patients may be found among those whose tinnitus occurred after head injury, and their evaluation, using otoacoustic emissions, is the objective of this study.

There is a paucity of literature concerning tinnitus induced by head injury, and tinnitus in these cases is probably largely unreported. According to Vernon and Press, 8% of the patients from their tinnitus data registry (n=1240) represent the group with head injury.

Otoacoustic emissions represent unique tools for examining the cochlea, and have revolutionised clinical audiology by allowing a direct communication with the sensory cells. Substantial evidence exists that otoacoustic emissions result from the non-linear motile characteristics of outer hair cells, through the action of the contractile elements, myosin and actin, contained in the infrastructure of outer hair cells, and, presumably, outer hair cell active motility is the basis for the high hearing sensitivity and frequency selectivity. As otoacoustic emissions are invariably associated with functioning outer hair cells, their presence is a reliable indicator of cochlear (OHC) structural integrity, and their absence may indicate a cochlear lesion. Furthermore, by recording otoacoustic emissions, a subclinical cochlear lesion may be detected, as up to 30% of the OHC population may be damaged before any audiometric evidence in the quarter octaves pure tone audiometry from 0.125 kHz to 16 kHz.

However, besides being an expression of the cochlear structural status, otoacoustic emission may also give an indication of functional integrity of the mechanisms which control the cochlea. Cochlear activity is regulated or modulated by the CNS. The olivocochlear efferent system is a part of that regulatory complex, and its medial division, widely known as the medial olivocochlear system, seems to be of particular importance in the modulation of cochlear activity. The high density of the medial olivocochlear innervation, with about 95% of its fibres targeting the outer hair cells, and large direct synaptic contact with the soma of the outer hair cells, in comparison with the indirect innervation of the inner hair cells (IHCs), reflects the potential influence of this system on the control of cochlear mechanics.

The effect of the medial olivocochlear system on the cochlea can be assessed by contralateral acoustic stimulation, which presumably activates, predominantly crossed, fibres of the
medial olivocochlear system bundle and alters cochlear mechanics, and thus otoacoustic emission.

Although the medial olivocochlear system has been the most studied part of the efferent system, its functional aspects are still not well understood. The system is classically considered to be inhibitory,\(^\text{12}\) the effect being shown in many studies as reduced otoacoustic emission response amplitude, obtained by presenting the stimuli against silent background, and simultaneous contralateral acoustic stimulation.\(^{\text{10}}\)\(^\text{13}\)

However, there is evidence to suggest that the medial olivocochlear system enhances transient stimuli if they are presented against a continuous background noise.\(^{\text{14}}\)

The higher structures, above the superior olivary complex (medulla oblongata), can modulate the excitability of olivocochlear neurons—for example, inferior colliculi—\(^{\text{15}}\) or cortical and subcortical pathways,\(^{\text{16}}\) and, indirectly, exert their influence on cochlear mechanics.

The suppressive effect of the medial olivocochlear system in tinnitus and other auditory dysfunction has already been investigated. Preliminary studies of recording otoacoustic emission under contralateral acoustic stimulation in patients with unilateral tinnitus (comparing findings in the ears with and without tinnitus in the same subject), have reported that contralateral acoustic stimulation is less effective in ears with tinnitus.\(^{\text{17}}\)\(^\text{18}\)

However, subsequent studies\(^{\text{19}}\)\(^\text{20}\) showed a significant intersubject variability, and a diversity in results between studies, so that no general rule could have been drawn. In another study,\(^{\text{21}}\) medial olivocochlear system function was compared in subjects with noise induced hearing loss, with and without tinnitus, and with normal hearing, with and without tinnitus. Otoacoustic emission responses up to 1.5 kHz only, due to the absent responses in higher frequency bands in noise induced hearing loss, were considered. A significant difference (an enhancement of otoacoustic emission in the presence of contralateral stimulation) was found and attributed to a global efferent dysfunction. By contrast, a study comparing the efferent effect between the normal hearing (pure tone audiometry (PTA), 125–8000 Hz) patients with tinnitus and normal control subjects,\(^{\text{22}}\) suggested “a likely impaired functioning of the medial olivocochlear system”, with less pronounced medial olivocochlear system effect in patients with tinnitus.

**Table 1** Subjects included in the study

<table>
<thead>
<tr>
<th>Subjects (n)</th>
<th>Sex (f/m)</th>
<th>Ears (n)</th>
<th>Age (mean (SD), range)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients with head injury and tinnitus</td>
<td>20</td>
<td>12/8</td>
<td>37</td>
</tr>
<tr>
<td>Normal subjects</td>
<td>20</td>
<td>12/8</td>
<td>40</td>
</tr>
<tr>
<td>Patients with head injury without tinnitus</td>
<td>12</td>
<td>7/5</td>
<td>23</td>
</tr>
</tbody>
</table>

**Table 2** Patients according to the severity of head injury (n (%))

<table>
<thead>
<tr>
<th>Minor</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients with head injury and tinnitus</td>
<td>6/20 (30)</td>
<td>11/20 (55)</td>
</tr>
<tr>
<td>Patients with head injury without tinnitus</td>
<td>2/12 (16.7)</td>
<td>8/12 (66.6)</td>
</tr>
</tbody>
</table>

The diversity in these findings, some of them conflicting, is probably a result of different evaluation methods applied and the heterogeneity of tinnitus groups (for example, aetiological, audiometrical, or age related).

In addition to tinnitus, the medial efferent effect seemed to be altered in hyperacusis, with an increase in emissions under contralateral stimulation reported in a single case,\(^{\text{23}}\) and reduced in obscure auditory dysfunction.\(^{\text{24}}\)

**Materials and methods**

**SUBJECTS**

All subjects included in the study (table 1) had normal or nearly normal hearing, defined as hearing thresholds equal to or better than 25 dB HL at octave step frequencies from 0.25 to 4 kHz and up to 30 dB HL at 6–8 kHz, together with normal middle ear function, defined as the ear drum compliance from 0.3–1.7 cm\(^{\text{3}}\) and the peak middle ear pressure ± 50 daPa. This criterion was essential in view of the known influence of the transmission properties of the middle ear on otoacoustic emission.\(^{\text{25}}\)\(^\text{26}\)

**Patients with tinnitus after head injury**

Twenty consecutive patients attended a neuro-otology clinic, with tinnitus lasting at least 1 year after head injury due to a road traffic accident (13/20, 65%), sport injury (3/20, 15%), blow to the head (2/20, 10%), or a fall (2/20, 10%). According to the classification of head injuries for severity,\(^{\text{27}}\)\(^\text{28}\) six (30%) patients sustained minor (post-traumatic amnesia lasted less than 3 hours and no skull fracture), 11 (55%) moderate (a skull fracture was identified, or post-traumatic amnesia lasted between 3 hours to 7 days), and three (15%) severe head injury (post-traumatic amnesia lasted more than 7 days) (table 2).

**Figure 1** Mean pure tone audiometric thresholds for all groups.
All patients complained of tinnitus of complex composition, which included different sounds, variable in pitch and volume and aggravated by environmental noise. Other auditory complaints included hyperacusis and difficulty in listening in background noise.

Apart from auditory disorders, other aspects of head injury in these patients have not been taken into consideration in this study.

**Patients with head injury, but without auditory complaints**

Twelve consecutive patients who sustained head injury, due to a road traffic accident (seven, 58.3%), blow to the head (two, 16.7%), sports injury (two, 16.7%), or due to a fall (one, 8.3%), were included in the study. Two of them (16.7%) had minor, eight (66.6%) had moderate, and two (16.7%) had severe head injury.

**Normal subjects without auditory complaints**

Twenty consecutive volunteers were selected to match the group with tinnitus for age and sex. This control group was introduced at a later stage of the study, after the finding of significantly reduced otoacoustic emission responses in the group without tinnitus in comparison with the group with tinnitus. The lower level of otoacoustic emission responses might result from a lower gain of the cochlear amplifier, but might also be a consequence of outer hair cell damage. As otoacoustic emissions represent the response based on the outer hair cell population, a scattered lesion of the outer hair cells could lead to a reduction in amplitude, without significant change in the microstructure of the response pattern.

Therefore, by examining the spectral bands of the otoacoustic emission response, this type of outer hair cell lesion would not be identified.

The introduction of the group of normal subjects as another control group was considered to be adequate, as there is no reason to suspect that the group with tinnitus after head injury was not a part of the normal population before head injury.

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### Table 3 Tympanometric measures (mean (SD)) in patients with tinnitus and control groups

<table>
<thead>
<tr>
<th></th>
<th>Ear drum compliance (cm³)</th>
<th>Peak pressure (dPa)</th>
<th>Ear canal volume (cm³)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients with head injury and tinnitus</td>
<td>0.7 (0.3)</td>
<td>5.6 (15)</td>
<td>1.2 (0.4)</td>
</tr>
<tr>
<td>Normal subjects</td>
<td>0.8 (0.3)</td>
<td>−0.7 (9)</td>
<td>1.4 (0.4)</td>
</tr>
<tr>
<td>Patients with head injury without tinnitus</td>
<td>0.6 (0.2)</td>
<td>−0.4 (9)</td>
<td>1.3 (0.4)</td>
</tr>
</tbody>
</table>

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### TEOAE recording

The stimulus presentation, data recording, averaging, and spectrum analysis have been carried out as described by Kemp et al. Briefly, the stimuli were unfiltered rectangular clicks (bandwidth 5 kHz), duration of 80 μs, presented at repetition rate 50/s, with the peak receptor level at 80 (SD 3) dB sound pressure level (SPL). They were presented in non-linear mode (4 clicks with every fourth click of reversed polarity and 10 dB increase in amplitude), which cancels the linear portion of the stimulus and response, so that non-linear cochlear emissions can be extracted. The number of sweeps during the period of collection was 260 and the poststimulus analysis was 2.5–20 ms.

The random noise contamination, the main sources of which are low frequency biological noise from the subject and ambient noise, was controlled by setting the rejection threshold at 47.3 dB SPL.

The following data, supplied by the ILO92 software, have been considered:

- The overall TEOAE response.
- The difference (A-B) of the two averaged waveforms, which is a good estimate of the noise level, to ensure comparable noise levels between the groups, preventing bias in TEOAE responses.
- The signal to noise (S/N) ratio in the bands (about 1 kHz width) centred at 1, 2, 3, 4, and 5 kHz, as the simplest form of TEOAE spectral analysis.

This analysis was performed to identify subclinical cochlear lesions (absence of the response in a spectral band) which may influence the TEOAE response.

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### Table 4 Mean (SD) TEOAE and noise levels in patients with tinnitus and control groups

<table>
<thead>
<tr>
<th></th>
<th>TEOAE responses (dB SPL)</th>
<th>Noise (dB SPL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients with head injury and tinnitus</td>
<td>12.8 (4)*</td>
<td>−1.0 (1)</td>
</tr>
<tr>
<td>Normal subjects</td>
<td>8.0 (3)</td>
<td>−1.5 (1)</td>
</tr>
<tr>
<td>Patients with head injury without tinnitus</td>
<td>6.2 (4)</td>
<td>−0.9 (1)</td>
</tr>
</tbody>
</table>

*P<0.05.

TEOAE=transient click evoked otoacoustic emission.
Synchronised SOAEs were recorded using a single 80 µs click at around 75 dB SPL, presented in 80 ms intervals. As most of the subsequent response lasts less than 20 ms, the microphone signal, averaged over a 20–80 ms poststimulus period, represents mostly spontaneous cochlear activity. Typically, 260 responses were averaged and Fast Fourier transformation (FFT) analysis was performed in the spectral band from 0 to 6250 Hz, with a resolution of 12.3 Hz. The presence of SOAEs was seen as spectral peaks of amplitude of at least 5 dB above the noise floor in the frequency range from 500 to 6250 Hz. SOAEs at frequencies <500 Hz were not considered due to the higher susceptibility to noise contamination in this frequency region.

SOAE recording

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Table 5 Results of SOAEs recorded in patients with tinnitus and in control groups

| Patients with head injury and tinnitus | 92% (100%) | 4.4% |
| Normal subjects                        | 38 (50)    | 1.7  |
| Patients with head injury without tinnitus | 17 (17)  | 0.25 |

*p<0.05.
SOAE=spontaneous otoacoustic emission.

Table 6 Suppression of TEOAEs by contralateral noise

<table>
<thead>
<tr>
<th>Suppression &gt;1dB</th>
<th>Mean (SD, range)</th>
</tr>
</thead>
<tbody>
<tr>
<td>%</td>
<td>(dB)</td>
</tr>
<tr>
<td>------</td>
<td>------------------</td>
</tr>
<tr>
<td>Patients with head injury and tinnitus</td>
<td>57/35*</td>
</tr>
<tr>
<td>Normal subjects</td>
<td>100</td>
</tr>
<tr>
<td>Patients with head injury without tinnitus</td>
<td>100</td>
</tr>
</tbody>
</table>

*p<0.05.
TEOAE=transient click-evoked otoacoustic emission.
illustrated in fig 2, showing 95% confidence intervals (95% CIs) for the means for all three groups. This difference was seen in the presence of comparable noise level in all three groups (table 4).

TEOAE spectral analysis showed a similar distribution of the presence of TEOAE responses in 1, 2, 3, 4, and 5 kHz bands (fig 3 A). However, in patients with tinnitus, the mean TEOAE responses in all frequency bands were significantly higher than those in the control groups (fig 3 B).

SOAEs
The analysis of SOAE spectra showed a 100% prevalence of SOAEs in the group of 20 subjects with tinnitus. This was significantly higher than in the normal subjects (10, 50%) and the patients with head injury without tinnitus (two, 17%). Similarly, the number of SOAE peaks were significantly higher in tinnitus (4.4/ear) than in the normal (1.7) and head injury without tinnitus (25/ear) groups. The summarised results of SOAEs, including the prevalence of SOAE/ear, to indicate the presence of SOAEs in both ears, are shown in table 5.

Medial olivocochlear suppression test
The suppression effect of the medial olivocochlear system was obtained by subtraction of the TEOAE responses under contralateral stimulation from those without contralateral stimulation. In all normal subjects this suppression effect was >1 dB. Therefore, 1 dB was considered to be a cut off point, below which the medial olivocochlear suppression was labelled as reduced (<1 dB), or absent (0). Accordingly, all subjects in the control groups (20 normal subjects and 12 patients with head injury but without tinnitus) had normal efferent suppression, whereas in patients with tinnitus, normal suppression was found in only seven out of 20 (35%) subjects, or in the other words, in a significant number of subjects (65%), medial olivocochlear suppression was found to be reduced or absent in one or both ears (table 6).

Figure 4 illustrates the findings in a patient with auditory complaints, including tinnitus, after severe head injury.

Discussion
Patients with tinnitus and other auditory complaints, including hyperacusis and difficulty in listening in background noise after head injury, showed significantly higher TEOAE amplitudes (12 (SD 4) dB SPL) than patients who had no auditory complaints, but who also sustained head injury (6.2 (SD 4) dB SPL). They were matched for sex and age, and had similar audiometric patterns. To exclude subclinical cochlear lesions, with the subsequent reduction of TEOAE amplitudes, and therefore to avoid potential bias in favour of the group with tinnitus, a simple spectral analysis, signal to noise ratio in 1, 2, 3, 4, and 5 kHz centred bands, was performed. This analysis, which was considered to be, together with audiometric thresholds, an additional clinical criterion for normal hearing, did not show a significant difference in the distribution of the presence of TEOAE responses in frequency bands between the tinnitus and non-tinnitus head injury groups.

However, this procedure could not exclude scattered outer hair cell lesions, with a reduced number of outer hair cells contributing to the TEOAE response, leading to a reduction of amplitude, but without obvious change in the pattern of frequency dispersion.

Therefore, TEOAE responses, overall and in frequency bands, in patients with tinnitus after head injury were additionally compared with
those in normal subjects who had neither tinnitus nor head injury. As expected, no subclinical cochlear lesions were detected in normal subjects, but TEOAE amplitude in patients with tinnitus still remained significantly higher than in this control group (8.0 (SD) 3 dB SPL).

A striking 100% prevalence of recordable SOAEs, as well as the largest number of SOAE spectral peaks/ear (4.4), in subjects with tinnitus was found, significantly higher than in both control groups; in normal subjects the prevalence of SOAE was 50%, with 1.7 SOAE peaks/ear and in subjects with head injury without auditory complaints, SOAE prevalence was 17%, with 0.25 SOAE peaks/ear. The SOAE prevalence in normal subjects was in agreement with previously reported results for such subjects.8

The magnitude of relative TEOAE reduction under contralateral acoustic stimulation, in comparison with TEOAE amplitude without contralateral stimulation, was found to be ≥1 dB in all control subjects and these subjects were considered to have normal medial olivocochlear suppression (in this study 1 dB TEOAE reduction was a cut off point to separate subjects with normal and abnormal medial olivocochlear suppression). By contrast, in a significant number of patients with tinnitus (65%), medial olivocochlear suppression was reduced (<1 dB) or absent (0 dB).

The effect of a lesion in the CNS (for example, at the brainstem level) on medial olivocochlear function leading to the absence or reduction of medial olivocochlear suppression has already been documented.33-34 Whether the lesion affects the afferent, or efferent, or both parts of the olivocochlear reflex, is open to question.

The integrity of the afferent input and access to the efferent pathways was judged on the basis of normal stapedial reflexes and auditory brainstem responses in all subjects with the exception of four patients with tinnitus, one of whom had abnormal acoustic reflexes, and in three in whom auditory brainstem responses were not recorded because they could not tolerate the necessary acoustic stimulation. As the acoustic reflex, auditory brainstem responses, and olivocochlear reflex share the same ascending pathways, it is assumed that reduced or absent suppression of TEOAEs in these patients results from dysfunction of descending auditory pathway. However, for abnormal acoustic reflex and auditory brainstem evoked responses, the dysfunction could be in either the ascending or descending pathways.

In this study there is an indication of auditory efferent dysfunction involving the medial olivocochlear system in 65% of patients with tinnitus. In the remaining 35% of the patients, who exhibited similar characteristics with respect to the auditory symptoms and otoacoustic emission findings, the suppression test showed TEOAE reduction values >1 dB, implying normal medial olivocochlear function. As this test activates the medial olivocochlear system, the functioning of the rest of the efferent system, above the superior olivary complex (medulla oblongata), remains obscure. In view of the known multisynaptic connections of the medial olivocochlear system, within the inferior colliculi and via the inferior colliculi to the auditory cortex, suggesting a descending trisynaptic pathway from the cortex to the cochlea, it could be speculated that higher auditory pathways may be implicated in a more global efferent dysfunction, with the above described consequences.

The presence of significantly higher TEOAE responses, high prevalence of SOAEs with many SOAE spectral peaks, and reduced or absent medial olivocochlear efferent suppression in patients with tinnitus was interpreted to be due to an increase in the cochlear amplifier gain secondary to the dysfunction in the efferent control of cochlear mechanics (disinhibition of suppressive effect), subsequent to head injury.

There have already been suggestions that the size of emissions is related to the status of the medial olivocochlear system,33 which may be abnormal structurally—that is, with a demonstrable morphological lesion—or functionally—for example, with an imbalance of central neurotransmitters. This was illustrated in the experiment by Salonna et al.,35 in which intravenous injection of acetylcholine, the principle neurotransmitter of the medial olivocochlear system, in 10 healthy human subjects led to a marked increase in emissions.

In normal conditions the central auditory system exerts its effect on the cochlea through efferently induced mechanisms of electromechanical transduction (outer hair cell electromotility), thus extending the dynamic range of the cochlea. The resulting cochlear amplification or attenuation is presumably a basis for the high sensitivity and frequency selectivity. However, reduced or absent medial olivocochlear suppression, as has been shown in this study in patients with head injury, may lead to a reduction in the dynamic range of the cochlea, leading further to a reduced ability of fine tuning and to difficulty in extracting transient stimuli in background noise. This could be a possible explanation for the symptom of difficulty in listening in background noise.

At the same time, the loss of a damping effect (attenuation) on afferent cochlear activity, normally produced by stimulation of the medial olivocochlear bundle, and an increase in amplifier gain caused in increase in the cochlear partition displacement as a response to auditory stimuli. This could be responsible for abnormal sensitivity to ordinary environmental sounds (hyperacusis). This increased auditory gain may also result in abnormal neural excitation, abnormal central sound processing, and, consequently, tinnitus.

It is apparent that tinnitus and other auditory complaints—hyperacusis and difficulty in listening in background noise—attributed to head injury and associated with normal peripheral auditory function (normal or nearly normal audiometric thresholds) and undamped otoacoustic emission, robust TEOAEs, and almost invariably recordable
SOAE—are the consequences of an extracochlear phenomenon and constitute a clinical presentation which may be termed “disinhibition syndrome”, subsequent to central efferent auditory dysfunction.

To summarise, this study has shown the potential value of otoacoustic emission in the assessment of tinnitus and other auditory complaints, providing information on structural integrity of the cochlea. It also gives insight into the functioning of the central control mechanisms. This information may contribute to a better understanding of the origin and mechanisms underlying tinnitus resulting from a lesion or dysfunction in the CNS.

Additionally, the assessment of medial olivocochlear system by recording otoacoustic emission under contralateral acoustic stimulation in a suspected lesion of the CNS could contribute to neuro-otological topographic diagnostics.

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