Unilateral Tinnitus Caused by Cerumen Impaction and Stochastic Resonance
O Vysata, M Kucera, A Prochazka, J Kukal, L Pazdera

Citation

Abstract
Objectives: The objectives of this study were: (i) To investigate hearing lost in patients with tinnitus caused by cerumen impaction before and after cerumen removal and its relationship to the tinnitus. (ii) To discuss possible role of synaptic noise by mechanism of stochastic resonance.

Methods: Nine patients, who had unilateral tinnitus and cerumen impaction, were selected for the study. Standard audiometric evaluation was performed, including case history, and measures of hearing sensitivity at 0.25-8 kHz. The testing protocol had three response tasks: threshold testing, loudness matching, and pitch matching. The time after cerumen removal to the end of tinnitus was measured.

Results: In all patients the tinnitus disappeared in 20 minutes after cerumen removal. Control audiometry five minutes after cerumen removal showed recovery of normal hearing.

Conclusion: Fast dissipation of tinnitus after cerumen removal and recovery of normal hearing shows possible connection between mechanism of weak signal enhancement for example by mechanism of stochastic resonance and tinnitus. Such a mechanism may also explain presence of tinnitus in individuals with intact auditory perception.

Study conducted at Otorhinolaryngology Outpatient Clinic, Rychnov nad Kneznou
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INTRODUCTION
Chronic tinnitus is a frequent symptom in the current medical practice. It is a common disorder with many possible causes and affects about 10% of the general population. In many people activities of daily living are affected in proportion to the intensity of the tinnitus. Most cases of tinnitus are subjective. Objective tinnitus is rare. It is usually caused by vascular abnormalities of the carotid artery or jugular venous systems. The mechanism that produces subjective tinnitus remains poorly understood. Tinnitus may originate at any location along the auditory pathway. Possible pathogenetic mechanisms include injured cochlear hair cells that discharge repetitively and stimulate auditory nerve fibers, spontaneous activity in individual auditory nerve fibers, hyperactivity of the auditory nuclei in the brain stem, or a reduction in the usual suppressive activity of the central auditory cortex on peripheral auditory nerve activity. The last two theories may be explained in terms of the nonlinear effect called stochastic resonance. This counterintuitive effect relies on “noise” - any random, or stochastic, background fluctuation to make a system sensitive to an otherwise undetectable signal. In biological neural networks electrical signals are transmitted through synapse where synaptic noise is present. Increase in synaptic noise may increase utilization of weak signal. If the level of synaptic noise exceeds the threshold of the postsynaptic membrane, useful signal may be compromised by the noise. Such a regulatory mechanism is not structurally conditioned and may quickly react to the changing intensity of the useful signal.

The aim of this study was to investigate hearing lost in patients with tinnitus caused by cerumen impaction before and after cerumen removal and its relationship to the tinnitus.
MATERIALS & METHODS

Nine subjects participated in this study, including 5 females and 4 males, with a mean age of 58.11 y (range, 34-81; SD, 12.8). Subjects were selected based on having unilateral tinnitus caused by cerumen impaction. All subjects were naive to any form of tinnitus assessment. At the start of the session, standard audiometric evaluation was performed, including case history, and measures of hearing sensitivity at 0.25-8 kHz, loudness matching, and pitch matching. The standard audiometry was performed also 5 minutes after cerumen removal.

RESULTS

All of the evaluated subjects had constant unilateral tinnitus. It developed gradually during last week before examination (2-7 days). Comparing other side on the affected side the hearing threshold was decreased in all frequencies (Fig. 1).

Figure 1
Figure 1: Difference between affected and non-affected ear (standard audiometry)

Differences between auditory threshold before and 5 minutes after cerumen removal on affected ear are similar. (Fig. 2).

In all patients tinnitus has gradually disappeared within 20 minutes. Exact time point was difficult to establish. Tinnitus frequency covers whole frequency spectrum (Tab. 1).

Figure 2
Figure 2: Auditory threshold improvement 5 minutes after cerumen removal

<table>
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<tr>
<th>Patient No.</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
</tr>
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<tbody>
<tr>
<td>Pitch matching (Hz)</td>
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<td>4000</td>
<td>500</td>
<td>500</td>
<td>6000</td>
<td>1000</td>
<td>1000</td>
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<tr>
<td>Loudness matching (dB)</td>
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<td>10</td>
<td>25</td>
<td>15</td>
<td>20</td>
<td>70</td>
<td>40</td>
<td>30</td>
</tr>
</tbody>
</table>

DISCUSSION

Measured data suggest fast recovery of hearing threshold and tinnitus disappearance. Those data shows rather functional then anatomic background of tinnitus mechanism. One possible explanation is neural feedback loop to cochlear hair cells, whose gain is regulated by the brain. This loop is normally adjusted just below onset of self-oscillation. If hearing threshold decreases, self-oscillation with spontaneous otoacoustic emission may appear. However, tinnitus has been found to correlate to otoacoustic emissions in only 5% of cases. Another mechanism, which increases sensitivity of central nerve system is stochastic resonance. It doesn't require self-oscillation of some neural population. Intracellular recordings of cortical neurons in vivo consistently display a highly complex and irregular activity (1) which results from an intense and sustained discharge of presynaptic neurons in the cortical network. Computational models suggest that this synaptic activity, or synaptic “noise” (2), may have important consequences on the integrative properties of these neurons (3), as well as on the subthreshold behavior (4). Synaptic noise improves detection...
of subthreshold signals of the neurons in central nerve system (6). Such a mechanism may compensate hearing lost on different levels of auditory pathway (Fig. 3 a-d).

Figure 4
Figure 3a: Signal in auditory pathway exceeds threshold of postsynaptic membrane in the presence of low synaptic noise.

Figure 5
Figure 3b: Weak signal doesn't reach threshold of postsynaptic membrane, synaptic noise is low.

The frequency of tinnitus probably doesn't reflect entire frequency of noise but affected region of tonotopic organized auditory pathway. Tinnitus is not necessarily an ear-related symptom. Level of synaptic noise may be probably influenced by many different physiological and pathological causes. For example there is strong evidence that the level of an individual's awareness of their tinnitus can be stress-related. Stress activation of the hypothalamic–pituitary–adrenal axis can directly modulate hearing sensitivity (1). Stochastic resonance may give explanation of tinnitus by non-specific increase of synaptic noise in many different conditions.

CONCLUSIONS
Mechanism of subjective tinnitus is poorly understood. Fast dissipation of tinnitus after cerumen removal shows possible

Figure 6
Figure 3c: Superposition of higher intensity of synaptic noise with weak signal increases probability of reaching threshold.

Figure 7
Figure 3d: Further decreasing of signal intensity and increasing of noise energy. Noise reach threshold of postsynaptic membrane, tinnitus appears.
connection between mechanism of weak signal enhancement for example by mechanism of stochastic resonance and tinnitus. Such a mechanism may also explain presence of tinnitus in individuals with intact auditory perception.

CORRESPONDENCE TO
Mr O. Vysata, Jiraskova 1389 51601 Rychnov nad Kneznou Czech Rep. Tel. Number: +420 775575460 Fax Number: +420 494531544 Email: vysata@neurol.cz

References
Author Information

Oldrich Vysata, MD
Neurocenter Caregroup, Rychnov nad Kneznou

Martin Kucera, MD
Otorhinolaryngology Outpatient Clinic, Rychnov nad Kneznou

Ales Prochazka, PhD
Department of Computing and Control Engineering, Institute of Chemical Technology in Prague

Jaromir Kukal, PhD
Department of Computing and Control Engineering, Institute of Chemical Technology in Prague

Ladislav Pazdera, MD
Neurocenter Caregroup, Rychnov nad Kneznou