Hearing disorder from music; a neglected dysfunction

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Background
Exposure to loud music is one of the most common causes of acute acoustic trauma, which adolescents and teenagers experience by voluntary exposure to loud music of sound levels up to 110 dB(A).

Methods
The clinical and psychophysical data of 104 consecutive patients with music-induced hearing disorder (MIHD) were analyzed to construct individual hearing and tinnitus profiles. In all cases, tinnitus was the presenting symptom.

Results
Hearing abilities were normal in about two-thirds of the tinnitus patients but two patients showed temporary thresholds shifts immediately after the insult. Tinnitus was experienced most often as a high frequency tone (83%) and in two-third of the cases the level of tinnitus-matched tones were below 30 dB HL. The Tinnitus Handicap Inventory (THI) scores ranged from 0-94 with an average score of 43.1 and most patients were classified as mild or moderate (60%). Visual Analogue Scales (VAS) were used to assess tinnitus loudness (average 42.4) and annoyance (average 54.2), and tinnitus awareness was estimated (average 60.3). All VAS values correlated strongly with the THI. Hyperacusis was present in 65% and 79% of the patients reported sleeping disorders.

Conclusion
This study gives evidence that music-induced acute acoustic trauma is not inevitably linked to hearing dysfunction as validated by conventional pure tone audiometry. In contrast, the main symptom is tinnitus often in combination with hyperacusis. Our results point at “silent hearing loss” as the underlying pathology, having afferent nerve terminal damage rather than hair cell loss as the structural correlate.

Keywords: MIHD, music-induced hearing disorder; CS, cochlear synaptopathy; HL, hearing loss; NIHD, noise-induced hearing disorder; NIHL, noise-induced hearing loss; THI, tinnitus handicap inventory; VAS, visual analogue scales

Introduction

Noise-induced hearing loss (NIHL) is an increasingly prevalent disorder, especially when in addition to hearing loss all noise-triggered auditory dysfunctions such as tinnitus and hyperacusis are included. In the past, NIHL was considered to be an occupational health problem in environments where workers are exposed to very hazardous noise levels (such as military personnel) [1]. Today, effective occupational hearing protection programs have practically eliminated severe occupational noise traumas and, thus, occupational NIHL affecting speech frequencies is rarely seen. However, the occurrence of milder forms of occupational noise-induced hearing disorders (NIHD), that includes tinnitus and hyperacusis and can include high tone hearing dysfunction, is becoming more common. In most cases of NIHD tinnitus and hyperacusis are present, but hearing is normal as revealed by conventional pure-tone audiometry (PTA). Nevertheless, these individuals may have ”hidden hearing loss” [2] associated with cochlear synaptopathy (CS) [3,4]. CS seems to arise from exposure to sound pressure levels lower than those causing hair cell damage. The current hearing conservation guidelines and strategies (OSHA; NIOSH; ISO) are targeted to prevent hair cell damage and subsequent hearing dysfunction. Therefore, the protective strategies should be revised.

Leisure time noise exposures earlier considered to play only a minor role as a health hazard are now recognized as a major risk factor of NIHD. Adolescents and teenagers often expose themselves to loud music and excessive noise levels during social and music events: noise levels between 104 and 112 dB (A) can be measured in nightclubs and discos [5]. Furthermore, sound surveys have indicated that orchestral musicians are exposed to sound levels ranging from 83 to 97 dB(A) Leq, and rock/jazz musicians from 91 dB(A) to as high as 115 dB(A). Furthermore, in a previous study 74% of rock and jazz musicians exhibited

symptoms of music-induced hearing disorder (MIHD) and more than 50% of all musicians have very distressing combinations of MIHDs [6]. Sound pressure levels in these types of exposures are not as high as in exposures leading to typical NIHL in which HL is caused by direct destruction of cochlear sensory cells (first outer hair cells, OHCs). However, sound pressure levels of acoustic overstimulation from music are high enough to cause such auditory dysfunctions as tinnitus and hyperacusis. These symptoms often cause much greater distress to affected individuals than what high-tone hearing loss would ever do.

The aim of this study was to record clinical and psychophysical characteristics of MIHD from individuals after a single trial of acoustic overstimulation from music. Our data show that the occurrence of HL and tinnitus can indeed occur fully detached from each other. It remains to be tested if HL is the ultimate fate of all tinnitus patients and thus, tinnitus being the first symptom of an initiated CS.

Methods

Patients

Data on clinical and psychophysical characteristics of 104 consecutive outpatients presenting to the Helsinki Tinnitus Clinic from Jan 1, 2010 to Mar 31, 2014 were analyzed retrospectively. The patient population consists of 71 males and 33 females with an age ranging from 14 to 62 years (average±SD 31.3±9.3) (Figure 1). All patients suffered from tinnitus triggered by a music-induced acoustic trauma. The major study inclusion criterion was tinnitus triggered by an exposure to loud music within three months before the first visit. Specific exclusion criteria were strictly applied, i.e. (1) no time delay more than 100 days from the traumatic exposure; (2) no presence of otological or neurological diseases prior to

the noise trauma; and (3) no history of vertigo or dizziness. No segregation of cases was made on the basis of audiometric results or socio-demographic variables. All parts of the study were performed in accordance to the principles outlined by the Declaration of Helsinki. Informed consent was obtained from each participant before study inclusion.

Audiological examinations

All patients underwent standard audiological examination including tone-audiometry for frequencies ranging from 125 Hz to 12.0 kHz. Psychoacoustic pitch and loudness matches were determined for each patient. Clinical audiometers and earphones (Interacoustic AC 40 and Telephonics TDH 39 P) generated and reproduced the acoustic signals in a sound proof chamber.

Psychophysical evaluations

Tinnitus was evaluated using a validated questionnaire, namely the tinnitus handicap inventory (THI) [7]. Briefly, THI is a 25-item questionnaire with 3 answer possibilities (no; yes; sometimes) that yields highly reliable scores ranging from 0 to 100. It is one of the most commonly used questionnaires to measure the degree of disability caused by tinnitus, and to select patients who are in need of an intervention. Tinnitus loudness and annoyance were characterized using the visual analogue scale (VAS) [8], which is a measurement instrument for purely subjective symptoms. Awareness of tinnitus is given in percentage (0 to 100%).

Results

Most of the patients (87.5%) presented within 30 days (range 5 to 91 days) from the exposure, and two thirds of the affected were male. The traumatic exposure had occurred in

concerts (41%) or nightclubs (31%), during band playing (21%), using headphone (4%), or during studio work (2%). Only one percent of the patients was not able to identify the actual traumatic event (Figure 2). Tinnitus was experienced most often (78%) as a high frequency tone such as whining, ringing, beeping, whistling or TV-tuning sound at 6.0 kHz (n=13), 8.0 kHz (n=26), 10.0 kHz (n=25) and 12.0 kHz (n=17) (Figure 3(A)). The sound level of tinnitus-matched tones (n=90) was relatively low. In 21% of the cases, tinnitus was matched to a tone higher than 30 dB (HL). To classify the severity of the tinnitus, patients were scored using THI. The scores ranged from 0-94 (average±SD, 43.1±22.8). The majority reported of mild (26%) or moderate (29%) symptoms, and all others complained of slight (13%), severe (15%) or catastrophic tinnitus (9%). Occasionally, the THI score could not be determined (8%) (Figure 3(B)).

Relevant comorbidities were sleeping disorders (71%), concentration disorders (40%) and anxiety disturbances (40%). It was one of the major aims of this study to relate subjective hearing dysfunctions with detectable loss of hearing thresholds. Unexpectedly, hearing as measured by tone-audiometry was normal in 60% of the patients, 31% had a (chronic) high frequency and 9% a mild low frequency HL. None of the patients reported acute HL at the time of the examination but two had been diagnosed with temporary threshold shift immediately after the music trauma. In order to correlate the THI scores with subjective perception, tinnitus was rated similar to VAS (from 10 to 100). The values (average±SD) for loudness, annoyance and awareness were 42.4±19.4, 54.2±23.6, and 60.3±24.7, respectively. VAS values and awareness correlated strongly with THI scores ($p<0.001$ each). However, no clear correlation was found when the THI scores were correlated with HL (Figure 4(A)). The finding that 68 (65%) patients reported symptoms of hyperacusis (HA) prompted us to quantify HA occurrence in the different severity classes of tinnitus. As expected, higher THI

scores increased the probability of suffering from HA dramatically. With THI scores describing slight, mild or moderate tinnitus it is 1.3-2.5 times more likely to develop HA. With severe or catastrophic tinnitus, the ratio increases to 8-13 (Figure 4(B)).

Discussion

The present study investigated 104 consecutive tinnitus patients who had experienced loud music exposure and consequently developed a hearing disorder. The results reveal that music can cause an acute acoustic trauma with subsequent various degrees of hearing dysfunction, above all tinnitus. Severity of tinnitus (THI) and its subjective ratings correlated strongly with each other but not with HL as recorded by PTA.

Tinnitus and hyperacusis are the most common sequelae of acoustic overexposure. They are considered to be perceptual indicators of an abnormal increase in the “gain” of the central auditory system, resulting from the loss of normal peripheral input. Animal models of acoustic injury support a link between neural hyperactivity in the central auditory system and hearing loss-causing cochlear damage. From that follows the hypothesis that tinnitus is tightly connected to hearing loss. This study suggests that this is not the case. It is suggested that loud music, the most common trigger of tinnitus/hyperacusis, does not usually trigger permanent threshold shift, although it can cause severe tinnitus.

In conventional NIHL, the pathophysiological basis is OHC-damage explaining hearing loss. This functional deficit in conjunction with structural damage has qualified NIHL as a real medical entity. In ”hidden hearing loss” [2] (tinnitus/hyperacusis without change in PTA) this foundation has not existed and the NIHD has been largely ignored. If the novel theories of cochlear synaptopathy, explaining NIHD in animal models [3], are valid also in humans, it is

possible that in NIHD the selective damage of cochlear high-threshold afferent fibers (synapses), undetected by clinical audiometry, trigger neural hyperactivity that underlies tinnitus and/or hyperacusis [4]. Furthermore, if the synaptopathy theory holds true in humans, it seems that this may ruin the for more than a decennium dominated theory of pathophysiologic basis of noise trauma which states that those are the auditory hair cells which are most sensitive to noise trauma and are damaged first.

Nevertheless, leisure time noise exposures, which earlier were considered to play only a minor role as a health hazard, are now recognized as a major health risk. A recent study carried out on students at British universities showed that 88% of students experienced tinnitus after leaving a nightclub and 66% suffered from MIHD in the following morning. It was also reported that the use of hearing protection, and knowledge about the risks of loud music were negligible [9]. Proper risk recognition by organizers and visitors should lead both to down tuning of on stage loudspeakers and improved availability and use of ear protectors.

Additionally, not only are the participants and visitor of music events prone to contract MIHD, but they may also risk of developing more severe hearing deterioration. There is now evidence from noise injury animal models showing that prior noise exposures can exacerbate and accelerate age-related hearing loss (presbyacusis) [10]. It may be that a noise exposure previously believed to be “benign” still can cause irreversible neural damages in the absence of elevated hearing thresholds [3].This also raises the question as to whether cochlear aging may, at least partly, be a consequence of cumulative prior acoustic overexposures.
The problem of MIHD is widespread, particularly in musicians and music lovers but also in the general public and it is under-perceived since dysfunctions often accumulate over time, also from infra-pathogenic noise exposures. Concerted information campaigns using social

networks are needed to reach the populations at risk. Only a better understanding of MIHD combined with public awareness will allow music enjoyment without exposure to hazardous noise.

Conflict of Interest: Lehtimäki J, Ylikoski J, Ylikoski M and are board members Vagus Medical Inc

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Figure legend

Figure 1 Diagram Age distribution of tinnitus patients

Figure 2 Diagram Causes distribution of the music induced tinnitus

Figure 3 Diagram Tinnitus frequencies (A) and severity (B) distribution

Figure 4 Diagram THI distribution depending on (A) presence of hearing loss (HL, n=104) and (B) of hyperacusis (HA, n=96).

Figure 1

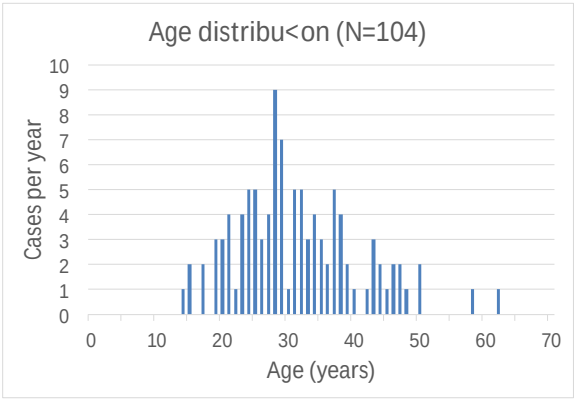


Figure 2

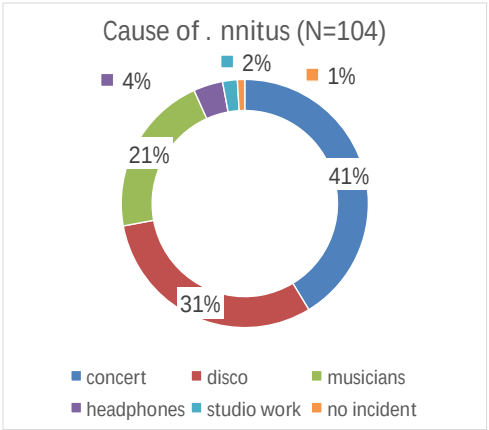
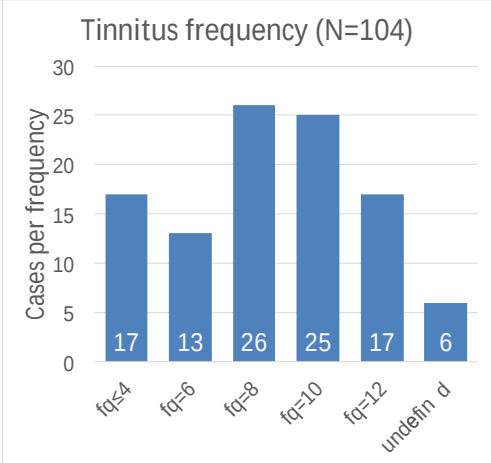


Figure 3

A



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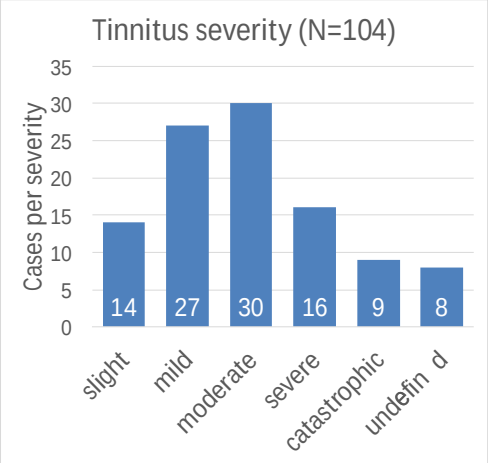
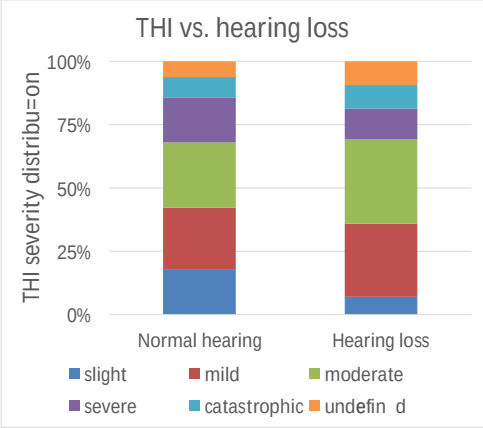


Figure 4

A



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