Original Article
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Medical Journal of the Islamic Republic of Iran (MJIRI)
Iran University of Medical Sciences

The relationship between ultra-high frequency thresholds and transient evoked otoacoustic emissions in adults with tinnitus

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Received: 29 June 2016 Accepted: 16 July 2016 Published: 26 November 2016

Abstract

Background: The possible role of cochlear function in tinnitus generation is still a matter of debate. To assess the role of outer hair cell dysfunction in tinnitus and its possible relationship with ultra-high frequency (UHF) hearing sensitivity, transient evoked otoacoustic emissions (TEOAE) and UHF hearing thresholds were investigated in normal hearing individuals with and without tinnitus.

Methods: Eighteen individuals with tinnitus and 22 without tinnitus participated in this study. TEOAE was recorded with click stimulus at 80 dBpeSPL. UHF pure tone audiometry was performed at 10, 12.5, 16, and 18 kHz.

Results: TEOAE was significantly abnormal in 72.2% of the tinnitus, and 18.2% of the control groups ($p=0.001$). The individuals with tinnitus had significantly poorer UHF hearing sensitivity compared to the control group at 12.5 and 18 kHz ($p\leq0.048$). There was a stronger correlation between increasing UHFs hearing threshold and decreasing SNRs of TEOAEs in the tinnitus group compared to the controls.

Conclusion: Our study revealed poorer UHF hearing thresholds and more TEOAE abnormalities in normal hearing individuals with tinnitus compared to the controls. Perhaps the alterations in the basal cochlea, following a decrease in UHF hearing sensitivity, affect OAEs that are originated from more apical cochlear parts in tinnitus ears more than non-tinnitus ears.

Keywords: Tinnitus, Otoacoustic Emissions, Ultra-High Frequency Thresholds.

Cite this article as: Omidvar Sh, Jafari Z, Mahmoudian S, Khabazkhoob M, Ahadi M, Yazdani N. The relationship between ultra-high frequency thresholds and transient evoked otoacoustic emissions in adults with tinnitus. *Med J Islam Repub Iran* 2016 (26 November). Vol. 30:449.

Introduction

Tinnitus is a phantom perception of sound (1), influencing 10–20% of the general population (2). However, the high prevalence and the harmful effect of tinnitus on patients' life, and the pathophysiology of tinnitus, especially the possible role of cochlear function, is still the matter of debate (3). It seems that impairment to the peripheral auditory system is essential for tinnitus generation (4,5), because 85% of the individuals with tinnitus also suffer from hearing loss, and 35% of them have moderate to severe hearing losses. However, 10–15% of the patients with tinnitus have normal hearing thresholds in the 250–8000 Hz frequency range (6). Thus, the loss of hearing threshold may not be a necessary condition for tinnitus, but audiograms cannot always indicate peripheral damage (7).

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There is no consensus on what mechanisms might cause tinnitus in individuals with normal hearing sensitivity. Hypotheses and theories vary, but most consider tinnitus a result of a minor damage (i.e., subclinical) in peripheral auditory system. According to discordant damage hypothesis, in normal hearing individuals tinnitus might be the consequence of minor dysfunction of outer hair cells (OHC) where inner hair cells (IHC) retain normal function (8). This disproportionately stimulates cells in the dorsal cochlear nucleus (DCN) and leads to tinnitus-related neuronal activity (1). This might explain tinnitus in normal hearing cases, as remote damage of OHCs can occur up to 30% without any related detectable hearing loss (9).

One of the most important methods to investigate cochlear function in humans is otoacoustic emissions (OAEs). Whereas the presence of OAEs is a reliable indicator of structural integrity of OHCs, their absence might reveal a subclinical cochlear lesion before any relevant evidence in pure tone audiometry (10). OAE is probably a population-based response whose amplitude shows summed activity of a significant number of OHCs. In contrast, normal hearing sensitivity to pure tone stimuli might depend on the optimal activity of a few OHCs, IHCs, and their associated neural fibers. By accumulating scattered OHC loss, OAE amplitude may decrease before any detectable changes in pure tone behavioral measurements. In these cases, it seems that minimal amounts of cochlear damage might cause measurable changes in OAE responses not great enough to influence clinical audiogram (11). One type of evoked OAEs is transient-evoked otoacoustic emissions (TEOAEs), which are emitted after presenting a short-duration acoustic stimulus (clicks or tone bursts), and therefore lead to wide stimulation of the cochlea (12).

Different studies investigated the role of minor OHC dysfunction in the generation of tinnitus by measuring OAEs in tinnitus patients. These studies reported diverse, sometimes conflicting results such as decreasing (13-19) or increasing (20-23) OAEs in tinnitus versus non-tinnitus ears. The diversity of findings might be due to the differences in protocols, measurement methods, and particularly etiologic conditions in tinnitus group or diversity of audiometric conditions between tinnitus and control groups. Moreover, most studies reporting tinnitus in normal hearing were based on classical pure tone audiometry, with the highest test frequency as 8 kHz (7, 10,14,18,24-25). It seems that ultra-high frequency (UHF) hearing loss can affect OAEs at much lower frequencies. Two possible reasons include OAEs sensitivity to subtle alterations in OHCs not yet detected by pure tone audiometry, or changes in basal portion of cochlea that influence the generation of lower frequencies OAEs, originating from more apical cochlear portions (11). A study indicated that poorer UHF hearing loss (8–16 kHz) correlated with diminished TEOAE amplitudes at significantly lower frequencies (1–5 kHz) (26). Accordingly, TEOAEs, which investigate much lower frequencies, can be influenced by UHF hearing loss.

The findings of this study are in line with the hypothesis that tinnitus and hearing loss are related. In neuroscientific approaches of tinnitus, hearing loss assumes a dominant role as an initiating event that induces neurophysiologic mechanisms, eventually perceived as tinnitus. Therefore, investigating UHF thresholds and TEOAEs is interesting and might have clinical implications, as UHF audiometry is not yet a routine clinical measure.

Different studies have investigated the cause of tinnitus in normal hearing individuals, and most of them tried to show hearing impairment in these patients by OAEs or UHFs. However, due to the heterogeneity of tinnitus groups compared to the controls, or diversity in audiometric conditions between groups, the findings were ambiguous, either as higher (20-23) or lower (13-19) OAE amplitudes, or weaker (27,28) or equal (29) UHF hearing thresholds in indi-

viduals with tinnitus than in the control group. Moreover, some studies reported a correlation between UHF assessments and TEOAEs (11,26), but this correlation has not yet been investigated in patients with tinnitus. It seems that alterations in basal portions of cochlea following UHF hearing loss affect OAEs of tinnitus patients more that non-tinnitus patients, particularly when the following possible reasons are considered: An influence of an unmeasurable damage basal to the site of OAE response in generation of more apically OAEs; and a role of UHF hearing impairments in triggering tinnitus in normal hearing subjects. Therefore, the main objectives of this study were to investigate any detectable hearing impairment by UHF hearing assessments and TEOAEs in normal hearing individuals with tinnitus and to detect any relationship between these measurements in the participants.

Methods

Participants

This cross-sectional analytic study was conducted on 18 normal hearing individuals with tinnitus (mean ± SD= 38.11±8.80 years; 9 males), and 22 controls without tinnitus (mean ± SD= 35.36±7.98 years; 9 males). The individuals with tinnitus were selected among patients referred to Emam Khomeini hospital, and the control group was selected from the employees and students of the Rehabilitation School of Iran University of Medical Sciences (IUMS), and were matched based on age and gender. This study was conducted in the audiology department of the school of rehabilitation sciences of IUMs. The overall inclusion criteria for the participants were as follows: Normal hearing sensitivity in both ears (≤ 25-dB hearing level [HL] at frequencies of 250 and 500 Hz and 1, 2, 4, and 8 kHz (30); threshold differences lower than 10 dB between ears at mentioned frequencies; no precedent of noise exposure or ototoxic drug consumption; and no precedent of audiological, psychological, or medical complications. To eliminate the possible effects of middle ear functionality on TEOAEs recording of the participants, middle ear function (ear canal volume: 0.9–2cm^3, static compliance: 0.3–1.5mmho, sound pressure level: ±50dapa (31)) and acoustic reflexes were tested to be normal. Otoscopic evaluations were normal, and the possibility of retrocochlear pathology was removed (wave V<6.8ms) (32), using auditory brainstem responses (ABR) evaluated by presenting a 100ms click stimulus at 80dBSPL (peak equivalent) at a rate of 13.3Hz. In addition to the above criteria, tinnitus perception for at least six months (33) in one or both ears was considered an inclusion criterion. We excluded those patients with somatosensory tinnitus, vascular abnormalities, temporo-mandibular joint syndrome or any other etiology known to cause tinnitus through mechanisms external to the auditory system.

The tinnitus handicap inventory (THI) questionnaire (34) was filled for all patients with tinnitus. According to THI, tinnitus severity was slight in nine persons, mild in seven, moderate in one, and severe in one. The study followed the Declaration of Helsinki, and its protocol was approved by the Ethics Committee of Iran University of Medical Sciences (IUMS) (the approval protocol number: 93/D/105/4847). Participants were informed about the aim of the study and provided written consent before participation.

Statistical analysis was performed by considering the results in just one ear for each individual. Because all the ears tested had thresholds of less than 25 dB at each frequency over the range of 250-8000 Hz, only the data of tinnitus ears were analyzed in individuals with unilateral tinnitus, and in those with bilateral tinnitus and in the controls the data of the right ears were examined (personal communication with Alessia Paglialonga).

Instrumentation

Pure tones were presented through a two-channel audiometer (Madsen Orbiter 922; GN Otometrics, Copenhagen, Denmark)

through a specialized headphone (HAD-200; Sennheiser, Wedemark, Germany) to obtain hearing thresholds for frequencies of 10, 12.5, 16, and 18 kHz at a dB sound pressure level (dBSPL). Measurements were performed, using an ascending-descending method in 5 dB steps at all frequencies. If a patient made two responses to a set of three stimuli at a hearing level, the level was considered as his/her hearing threshold at that frequency (28).

TEOAEs were recorded, using the Otodynamics ILO88 system in a double-walled soundproof cabin with dimmed lights and a standard noise level (35). Participants were asked to sit on a comfortable chair in a relaxed position, and breathe normally without any effort to produce the least possible additional noise during the recording session. Only one ear was considered for each participant: In patients with unilateral tinnitus, TEOAEs were assessed in the tinnitus ear; in patients with bilateral tinnitus and in the controls, TEOAEs were measured in the ear with better hearing thresholds (10, 16).

TEOAEs were recorded, using a standard protocol (36). Click stimuli were presented at 80 dBpeSPL. Number of sweeps was 260 for each ear. The rejection threshold was set at 4.6 millipascals. During the test, the responses were stored and averaged alternatively in two separate buffers, A and B, resulting in two averaged traces. Comparison of these two average traces allows the software to determine TEOAE parameters, including the TEOAE response, AB difference, reproducibility, and signal-to-noise ratio (SNR). Response is the overall level of the correlated parts of the A and B response traces, and AB difference is the average difference between the A and B traces and refers to the noise contained within the response. The reproducibility refers to how well two mean traces correlate with one another in five frequency bands, including 1, 2, 3, 4 and 5 kHz and are expressed as percentage. SNR is a ratio of the level of the TEOAE (the signal) to the level of the noise expressed in dB. The software calculates SNRs for the mentioned frequency bands (36). The criteria for considering a response as a presence of each frequency band were SNR≥6dB, and the reproducibility of 70% or more (36), and for whole frequency bands (whole response) they were SNRs≥6dB and the reproducibility of 70% or more in at least four of the five test frequencies (1, 2, 3, 4, and 5kHz) (24). Therefore, according to the mentioned criteria, TEOAE was considered normal or abnormal for each frequency band, and for the whole frequency bands. Measurements were performed by an audiologist who was blinded to the participants' membership in the investigated groups. The proper fitting of the ear probe was continuously monitored by the tester during TEOAE recording.

Statistical Analysis

The normal distributions of the data were measured, using the Kolmogorov–Smirnov test ($p<0.05$). Accordingly, the distributions of all quantitative parameters were normal except hearing thresholds at 0.5 and 18 kHz, and the AB difference of TEOAE. Therefore, an independent samples t-test was administered to compare hearing thresholds for frequencies of 0.25, 1, 2, 4, 8, 10, 12.5, and 16kHz; SNRs for frequencies of 1, 2, 3, 4, and 5kHz; and the response of TEOAEs between the tinnitus versus non-tinnitus ears. A non-parametric Mann-Whitney test was applied to compare hearing thresholds for frequencies of 0.5 and 18kHz and the AB difference of TEOAE between the two investigated groups; χ^2 test was used to compare tinnitus ears and control ears with respect to abnormal results according to the above criteria for each frequency band and the whole frequency bands (whole response). Moreover, Pearson rank-order correlations were used to determine the correlation between hearing thresholds at 10, 12.5, 16 and 18kHz, and SNRs at 1, 2, 3, 4, and 5kHz in tinnitus and control groups. It was also applied to determine the correlation between tinnitus severity based on THI questionnaire and

UHF hearing thresholds and SNRs of TE-OAE response in mentioned frequencies. P values and estimations of effect size (partial η^2) are reported in the annexed tables for these statistical analyses. All statistical analyses were conducted, using SPSS Statistics 22.0 at a significance level of 0.05.

Results

The means and SDs of hearing thresholds at conventional audiometric frequencies are shown in Table 1. Figure 1 displays the means and SDs of hearing thresholds (dBSPL) at 10, 12.5, 16, and 18kHz in tinnitus and control groups. The difference was significant at 12.5kHz (p=0.043, Partial η^2=0.653), 16kHz (p=0.048, Partial η^2=0.643), and 18kHz (p=0.006, Partial η^2=0.794).

In TEOAE measurements, the mean ± SD of response in individuals with and without tinnitus were 10.43± 5.37 dBSPL and 14.43±4.43 dBSPL, respectively, and the difference was significant (p=0.018). Moreover, the mean ± SD of the AB difference in individuals with and without tinnitus were 3.18±2.03 dBSPL and 2.29±1.25 dBSPL, respectively, but the difference was not significant (p=0.112). The means of SNRs of TEOAE from 1–5 kHz in those with tinnitus and in controls are demonstrated in Figure 2; the difference was significant at 2kHz (p=0.048, Partial η^2= 0.628), 3kHz (p=0.013, Partial η^2=0.834), 4kHz (p=0.029, Partial η^2=0.716), and 5kHz (p=0.048, Partial η^2=0.639).

Table 1. Mean and SD Values for Hearing Thresholds at Conventional Audiometric Frequencies in Tinnitus and Control Groups (Statistically Significant at p<0.05)

Frequency (Hz)	Tinnitus Group (N = 18)		Control Group (N = 22)		Statistical Test Results	
	Mean	SD	Mean	SD	p	Partial η^2
250	11.11	7.39	12.27	5.72	0.578*	0.175
500	7.50	5.75	9.09	4.26	0.229**	0.314
1000	8.61	4.79	10.68	4.95	0.190*	0.425
2000	5.28	6.75	5.45	7.22	0.937*	0.024
4000	7.22	9.27	7.27	8.83	0.986*	0.005
8000	10.56	9.98	9.09	10.65	0.659*	0.142

* Independent Samples t-test, **Mann-Whitney test, SD = Standard Deviation, N = Number, Partial η^2 = Effect Size

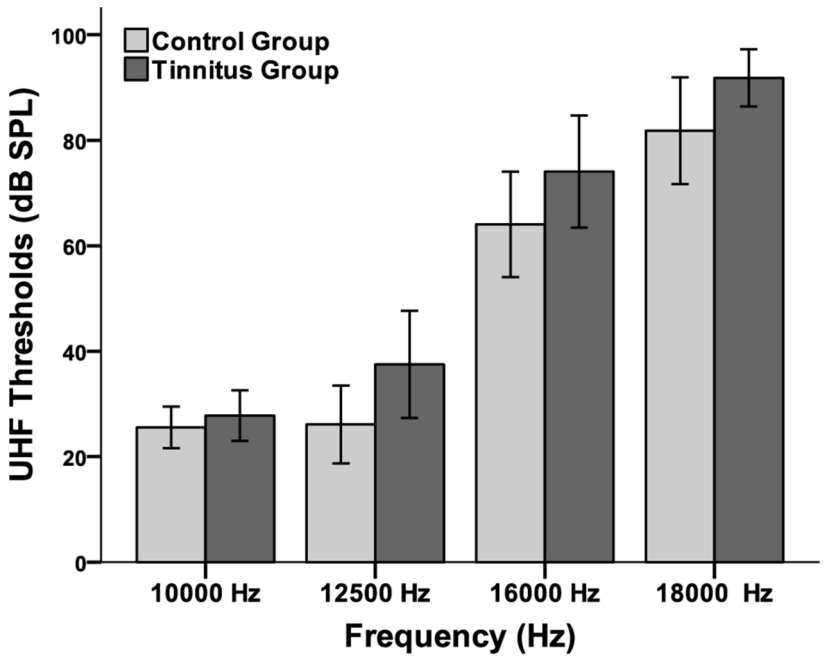

Fig. 1. The Mean Values for UHF (ultra-high frequency) Hearing Thresholds (dBSPL) in Tinnitus and Control Groups (* Indicates p<0.05, and Error Bar Represents Mean ± 2 Standard Error of the Mean (SEM))

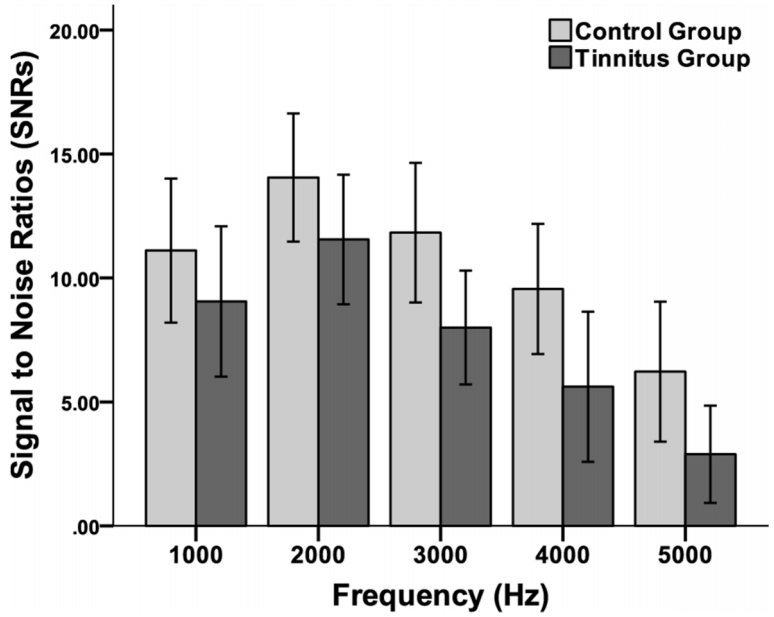

Fig. 2. The Means of SNRs (signal to noise ratios) of TEOAEs According to the Test Frequencies in Tinnitus and Control Groups (* Indicates $p<0.05$, and Error Bar Represents Mean ± 2 Standard Error of the Mean (SEM))

Table 2 demonstrates the percentages of normal and abnormal TEOAE tests by considering SNR and the reproducibility of all frequency bands and separate frequency bands in each group. Table 3 displays the correlation between UHF hearing thresholds and SNRs of TEOAE at 1–5 kHz in each group. No significant correlation was observed between tinnitus severity and SNRs of TEOAE at any of test frequencies, as well as UHF hearing thresholds ($p>0.05$).

Table 2. Percentage of Normal and Abnormal TEOAE Tests by Considering SNR and the Reproducibility of All Frequency Bands and Separate Frequency Bands Evaluated in Each Group (Statistically Significant at $p<0.05$)

	Tinnitus Group (N = 18)		Control Group (N = 44)		p*
Frequency (Hz)	Normal (%)	Abnormal (%)	Normal (%)	Abnormal (%)	
1000 to 5000	27.8	72.2	81.8	18.2	0.001
1000	72.2	27.8	86.4	13.6	0.266
2000	88.9	11.1	90.9	9.1	0.832
3000	66.7	33.3	90.9	9.1	0.057
4000	38.9	61.1	86.4	13.6	0.002
5000	22.2	77.8	54.5	45.5	0.038

*χ2test, SNR = Signal to Noise Ratio, TEOAE = Transient-Evoked Otoacoustic Emissions, N = Number

Table 3. Pearson Correlation (r) between UHF Hearing Thresholds and SNRs of TEOAEs According to the Investigated Frequencies (Statistically Significant at $p<0.05$)

	SNRs (dB)									
Hearing Threshold (dBnHL)	1000 Hz		2000 Hz		3000 Hz		4000 Hz		5000 Hz	
	r	p	r	p	r	p	r	p	r	p
Tinnitus Group										
10000 Hz	-0.173	0.492	-0.039	0.877	0.107	0.673	-0.374	0.126	-0.354	0.150
12500 Hz	-0.149	0.554	-0.108	0.669	0.078	0.757	-0.224	0.371	-0.269	0.281
16000 Hz	-0.229	0.339	-0.550	0.027	-0.302	0.256	-0.725	0.001	-0.393	0.132
18000 Hz	-0.408	0.213	-0.747	0.008	-0.707	0.015	-0.800	0.003	-0.012	0.973
Control Group										
10000 Hz	-0.019	0.933	0.229	0.305	0.256	0.251	-0.022	0.921	0.022	0.924
12000 Hz	-0.306	0.166	-0.135	0.550	-0.222	0.321	-0.257	0.249	-0.437	0.058
16000 Hz	-0.127	0.574	-0.076	0.738	-0.439	0.041	-0.337	0.125	-0.316	0.152
18000 Hz	-0.138	0.539	-0.078	0.730	-0.434	0.044	-0.507	0.016	-0.517	0.014

UHF = ultra-High Frequency, SNRs = Signal to Noise Ratios, TEOAE = Transient-Evoked Otoacoustic Emissions

Discussion

In this study, those individuals, who suffered from tinnitus with normal hearing thresholds according to classical audiometry, had weaker UHF hearing sensitivities that were significant at 12.5 and 18 kHz, and close to significant at 16 kHz. This finding was in accordance with the previous studies (27,28). The results support the previous tinnitus models, including the deafferentation hypothesis, in which tinnitus is considered a failure of the brain to adapt to deprived peripheral input (1). This hypothesis suggests that the cochlear damage triggers tinnitus incidence, even in patients with normal hearing sensitivity on conventional audiometry, and it considers a deafferentation as the underlying cause that elicits central reorganization and eventually leads to tinnitus (7). The deafferentation hypothesis provides a rationale for performing UHF threshold assessments in subjectively normal hearing patients with tinnitus (27). The SDs of UHF hearing thresholds were large in both groups, which were in accordance with the previous studies (28, 37), and might be attributed to the greater impressibility of the anatomical structure of the external auditory canal from higher frequencies (28,38).

The findings indicated lower SNRs at investigated frequencies in the tinnitus ears compared with the control ears, and these differences were significant from 2–5kHz. This finding confirms the results of the previous studies and it almost certainly indicates that a dysfunction of the cochlear active mechanisms, involved in the generation of OAEs, causes frequency components with lower SNRs in individuals with tinnitus. It seems that a subclinical OHC dysfunction, especially in high frequency cochlear regions, triggers tinnitus in normal hearing patients (11). However, some studies also reported higher OAEs amplitude. For instance, in a study (22), abnormally high DPOAEs were observed and attributed to amplification of mechanical distortion produced by cochlear hyperactivity (in particular OHCs). In another study (21), a significant increase was found in DPOAE amplitudes in a group of patients with acute tinnitus. The authors argued that a cochlear impairment (with a loss of IHCs) may reduce tonic efferent activity to the OHCs by decreasing afferent inputs to the central nervous system, which follows by decreasing tonic suppression of OHC electromotility, eventually making OHCs hyperactive. Hence, although the number of studies investigating tinnitus in normal hearing patients using OAEs has increased in recent years, the results are still inconclusive and further research is required in this field.

Considering the criteria of normality, which included both SNR and reproducibility, TEOAE was abnormal in 72.2% of the tinnitus ears compared with 18.2% in control ears; this difference was significant. This finding was in accordance with two previous studies (18, 24), but differed from Lonsbury-Martin et al. (39), who reported normal TEOAE in 97–100% of normal hearing individuals with tinnitus. This high percentage might result from considering just SNR as a normality criterion. Moreover, a significant difference at 4 and 5 kHz and a close to significant difference at 3 kHz were observed by comparing two groups (tinnitus vs. control groups) in each test frequency. This finding was similar to previous studies that reported abnormal TEOAEs in patients with hearing loss, mainly at higher frequencies than 2 kHz (40-41). Higher percentage of abnormal TEOAE in normal hearing patients with tinnitus, compared to normal hearing individuals without tinnitus, might suggest the role of OHC dysfunction in the production of tinnitus and in the support of deafferentation hypothesis. Jasterboff (42) postulated that all levels of auditory pathways may play a role in tinnitus generation, but most likely, the main trigger is OHCs.

In this study, a significant negative correlation was detected between the hearing threshold at 16 kHz and SNRs of 3kHz as well as hearing thresholds at 18 kHz and SNRs of 3, 4, and 5kHz in control ears.

However, this significant negative correlation with a higher correlation coefficient factor was observed between hearing thresholds at 16 kHz and SNRs of 2 and 4 kHz, and hearing thresholds at 18 kHz and SNRs of 2, 3, and 4kHz in tinnitus ears. It seems that poor hearing sensitivity at UHFs might be associated with a decreased number of functioning OHCs. In other words, damage basal to the site of OAE measurements may affect the production of lower-frequency OAEs originated from more apical cochlear parts (11). A study on guinea pigs (43) reported reduction in low frequency TEOAE by noise damage to the basal cochlea. Two human studies (11,26) indicated that hearing thresholds at 16 and 18 kHz affected TEOAE and DPOAE at very lower frequencies. In this study, lower SNRs were associated with reduced hearing sensitivity at more UHFs in tinnitus ears compared to control ears. Experimental studies suggested that the evoked OAEs seems to be affected by remote changes in hearing sensitivity, but the exact interpretation of these reductions in evoked-emissions levels is still unclear (11). It seems that OAEs might be beneficial in detecting the early abnormalities. Therefore, reduced OAEs in the presence of apparently normal hearing sensitivity might indicate UHF hearing loss, and explain the possible triggering of tinnitus in the high percent of patients with tinnitus that have normal hearing sensitivity at conventional audiometric frequencies. However, a greater increase in the sample size might indicate the higher relationship between poorer UHF hearing sensitivity and OHC function alteration in those suffering from tinnitus more apparently.

No relationship was found between tinnitus severity (based on THI scores) and UHF hearing thresholds and SNRs at the measured frequencies. Many studies have tried to indicate the possible relationship between tinnitus severity and decreasing hearing sensitivity level, but the results were different and sometimes contradictory (44-50). For instance, in a study (46), no significant difference was indicated in THI scores between normal-hearing and hearing-impaired individuals with tinnitus. Similarly, in another study (50), no relationship was detected between the percentage of hearing loss and the participants' assessment of tinnitus intensity. Whereas a number of studies indicated higher THI scores in the individuals with poorer pure tone thresholds (44,45,47,49), Savastano (48) reported an opposite finding: Higher THI scores in patients with normal hearing sensitivity. According to Jasterboff's neurophysiological model (42), tinnitus is due to impairment to the auditory pathways. However, its severity, in particular at cortical level, is defined by the processing of the signal (non-auditory factor). Therefore, decreasing hearing sensitivity might not be simply related to increasing tinnitus severity (49). Nevertheless, more research needs to clarify any relationship between tinnitus severity and hearing sensitivity.

This study used TEOAE because it is the most popular OAE measurement in clinical practice, and has a more standardized methodology (18). In our opinion, the TEOAE evaluation might be advantageous in investigating individuals with tinnitus, particularly when it is of cochlear origin. We believe that the application of the TEOAE test and UHF hearing thresholds together might be a tool for otorhinolaryngologic and audiological diagnosis that is able to objectively characterize a symptom that is usually subjective and multifactorial. Although early variations in TEOAE and UHF thresholds were detected prior to any alternations in conventional pure tone thresholds, these examinations alone cannot elucidate all tinnitus cases in an adequate manner, due to the complexity and diversity of its origin, from the peripheral auditory pathway to the central nervous system. Performing other assessments associated with cochlear function such as DPOAE, threshold-equalizing-noise (TEN) test (a test for the diagnosis of dead regions in the cochlea), and psychophysical tuning curves (PTCs) in combination with TEOAE could

provide more complete and precise findings to discuss. Such detailed evaluations of the cochlear function in normal hearing participants with tinnitus could be useful to comprehend the possible mechanisms involved in tinnitus generation more deeply as well as provide precious indications to improve the clinical examinations and standards of care for those suffering from tinnitus.

Conclusion

The findings revealed more decreased hearing sensitivity at UHFs and higher prevalence of TEOAE abnormalities in normal hearing individuals with tinnitus compared to the control group. Moreover, stronger correlation was observed between increasing UHFs hearing threshold and decreasing SNRs of TEOAEs in tinnitus group compared with the controls. Tinnitus severity was not related to UHF hearing thresholds or SNRs at all investigated frequencies. Further research is needed to understand the value of measuring OAEs and UHF hearing sensitivity in normal hearing individuals with tinnitus, and their potential advantages on clinical outcome of these patients. Furthermore, the combined use of different tests investigating cochlear function can help comprehend the role of cochlea and hair cells in tinnitus generation better.

Acknowledgements

This study was part of a Ph.D. dissertation project in audiology that was approved by Iran University of Medical Sciences (grant #93.D.3303.320). We are grateful to Fariba Nassaj and Dr. Masoud Motasadi Zarandi for technical support.

Conflict of Interest

The authors declare that they have no competing interests.

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