ISSN: 2083-389X
eISSN: 2084-3127

J Hear Sci, 2024; 14(2): 39–45
DOI: 10.17430/jhs/190258

ELECTROPHYSIOLOGICAL MEASURES OF AUDITORY IMPAIRMENT IN NOISE-EXPOSED, NORMAL-HEARING SOLDIERS

Contributions:
A Study design/planning
B Data collection/entry
C Data analysis/statistics
D Data interpretation
E Preparation of manuscript
F Literature analysis/search
G Funds collection

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Abstract

Introduction: Some electrophysiological changes can occur in the auditory system in response to noise exposure with or without any permanent auditory threshold shift. The purpose of this study was to identify and measure cochlear function after noise exposure in individuals with normal hearing according to standard audiometric thresholds.

Material and methods: Pure tone audiometry (PTA) over the standard 0.250–8 kHz range and at 12 kHz, as well as distortion product otoacoustic emission (DPOAE) and auditory brainstem response (ABR) testing, were performed on 42 soldiers who had participated in combat. A control group of 40 participants underwent the same tests.

Results: In the noise-exposed group, significantly poorer PTA thresholds were recorded at 12 kHz. DPOAE levels were significantly low only at 4 kHz. On ABR testing, both wave I and wave V demonstrated a significant decrease in amplitude and a significant increase in latency for the noise-exposed group.

Conclusions: Our findings reveal that high levels of noise can not only damage outer hair cells but also cause changes at the level of the synapses (synaptopathy) which are not evident using standard PTA tests. However, electrophysiological methods can detect some changes in cochlear function.

Keywords: DPOAE • ABR • noise exposure • cochlear synaptopathy

ELEKTROFIZJOLOGICZNE POMIARY UPOŚLEDZENIA SŁUCHU U NARAŻONYCH NA HAŁAS ŻOŁNIERZY ZE SŁUCHEM W NORMIE

Streszczenie

Wstęp: W układzie słuchowym mogą wystąpić pewne zmiany elektrofizjologiczne w reakcji na hałas skutkujące lub nie stałym przesunięciem progu słyszenia. Celem tego badania było zidentyfikowanie i zmierzenie funkcji ślimaka po narażeniu na hałas u osób ze słuchem w normie zgodnie ze standardowym progami audiometrycznymi.

Materiał i metody: W grupie 42 żołnierzy, którzy uczestniczyli w walce, wykonano następujące badania: audiometrię tonalną (PTA) w standardowym zakresie 0.250–8 kHz oraz dla 12 kHz, pomiar emisji otoakustycznych produktów zniekształceń nieliniowych (DPOAE), a także słuchowych potencjałów wywołanych pnia mózgu (ABR). Grupa kontrolna złożona z 40 osób przeszła te same testy.

Wyniki: W grupie narażonej na hałas zanotowano: statystycznie istotne pogorszenie progów PTA dla 12 kHz, poziomy DPOAE były istotnie obniżone tylko dla 4 kHz, w teście ABR zarówno fala I jak i fala V miały istotnie obniżoną amplitudę i istotnie opóźnioną latencję.

Wnioski: Nasze wyniki pokazują, że duży poziom hałasu może nie tylko uszkadzać zewnętrzne komórki rzęsate, lecz także powodować na poziomie synaps (synaptopatia) szkody, które nie są widoczne w standardowym badaniu PTA. Jednak metody elektrofizjologiczne mogą wykryć pewne zmiany w funkcjonowaniu ślimaka.

Słowa kluczowe: DPOAE • ABR • narażenie na hałas • synaptopatia ślimakowa

Introduction

Prolonged exposure to loud noise can cause tinnitus and hearing loss. It has long been assumed that the key indicator of noise-induced hearing loss was outer hair cell death [1]. Work in animal models has demonstrated that in noise-induced hearing loss such exposures cause only reversible threshold shifts (and no hair cell loss); however, they did result in the permanent loss of > 50% of cochlear nerve/hair cell synapses [2]. In humans, on study found that after a high level of voluntary noise exposure background (NEB), wave I of the auditory brainstem response (ABR) had reduced amplitude but there was normal hearing in response to suprathreshold clicks and 4 kHz tone bursts [3]. However, damage was only apparent when ABR wave I amplitude was examined. In contrast, distortion product otoacoustic emission (DPOAE) levels were found not to be significantly changed in the context of NEBs [3]. The suprathreshold ABR wave I amplitude was also lower in veterans reporting high levels of military noise exposure and in nonveterans reporting a history of firearm use than in veterans and nonveterans with lower levels of reported exposure [1]. Otoacoustic emissions (OAEs) have been described in some studies as early indicators of noise-induced damage or as a method to determine potential risks for developing noise-induced hearing loss [4–6]. Some reports, however, have not supported a significant role for OAEs in this regard [7].

Noise-induced cochlear synaptopathic injury cannot be detected by conventional audiometric assessment of threshold sensitivity. Thus, potential damage to auditory health and the performance consequences of noise-induced cochlear synaptopathic injury can be easily overlooked, especially if loss of threshold is the major concern [8]. When compared with behavioural threshold assessment, the use of DPOAEs in ears with normal thresholds (≤ 20 dB nHL) is not strongly supported as a way of detecting noise damage at an early stage [5–7]. In general, it seems that factors like ear side and gender have only minor effects on both DPOAEs and hearing thresholds in both the standard and extended high frequency ranges [9]. They confirm that hearing thresholds and DPOAEs in the extended frequency audiometry band seem to show promise for identifying early signs of hearing loss. Both EHFA and DPOAEs provide early evidence of noise-induced hearing loss in young recreational firearm users [10].

The purpose of our study was to assess cochlear function in normal hearing subjects after noise exposure in combat, giving insight into outer hair cell function and synaptopathy.

Material and methods

After the approval of the YSMU Science Coordination Council (19/02/2021, Nr. 1), 42 male subjects (84 ears) aged 20 to 39 years (mean age = 22.7 years) who had participated in combat in 2020 were involved in our study. The total duration of the military action lasted for 44 days, and the subjects had not previously participated in such events. Our study also excluded any acoustic trauma incidents prior to the combat. During combat they were constantly exposed to different forms of potentially harmful noise. All participants gave permission to participate. The study inclusion criteria were as follows: hearing threshold at in the range 0.25–8 kHz ≤ 20 dB hearing level (HL) (normal range according to BIAP) [11], normal middle ear function, absence of any contemporary hearing reduction during or after the war, and absence of any subjective hearing loss. The middle ear was evaluated by otoscopy and tympanometry (GSA Tympstar Pro), and was considered within normal limits if categorised as Type A by the Jerger classification [12]. As a control group, 40 male peers (79 ears, because one participant had unilateral otitis media at the time of the study) aged 19–41 years (mean age = 22.9 years old) were chosen. The inclusion criteria for the control group were a hearing threshold of ≤ 20 dB HL from 0.25 to 8 kHz, normal middle ear function, and no previous long-term, high-level noise exposure.

The participants of our study underwent the following audiological testing for cochlear function in Nairi MC:

- Pure tone audiometry (PTA) over the conventional 0.25–8 kHz range and at one extended high-frequency only at 12 kHz (examination at higher frequencies was not possible due to limitations of the audiometer). Hearing thresholds were determined for air conduction (GSI Audiostar Pro) and measurements were made in a sound proof cabin.
- Recording of the discomfort threshold in the 0.25–12 kHz range. Normal-hearing individuals have loudness discomfort level (LDL) between 86 and 98 dB HL for 0.5–8 kHz stimuli [13,14]. Thus discomfort threshold was evaluated at every frequency starting from 80 dB HL and increasing the intensity until the patient reported discomfort (GSI Audiostar Pro).
- DPOAE testing (2f1–f2 DP-gram, L1 = 65 dB sound pressure level (SPL), L2 = 55 dB SPL, f2/f1 = 1.22, SNR ≥ 6 dB, DP stability ± 2 dB); DP-gram responses were analyzed at 1, 2, 4, 6, and 8 kHz. Measurements were made in a sound proof cabin. If needed, testing was repeated several times until there was no difference among the two groups in terms of noise level at all frequencies. Thus objective comparisons were achieved in DP-level. All test subjects were evaluated using the Interacoustics Eclipse device.

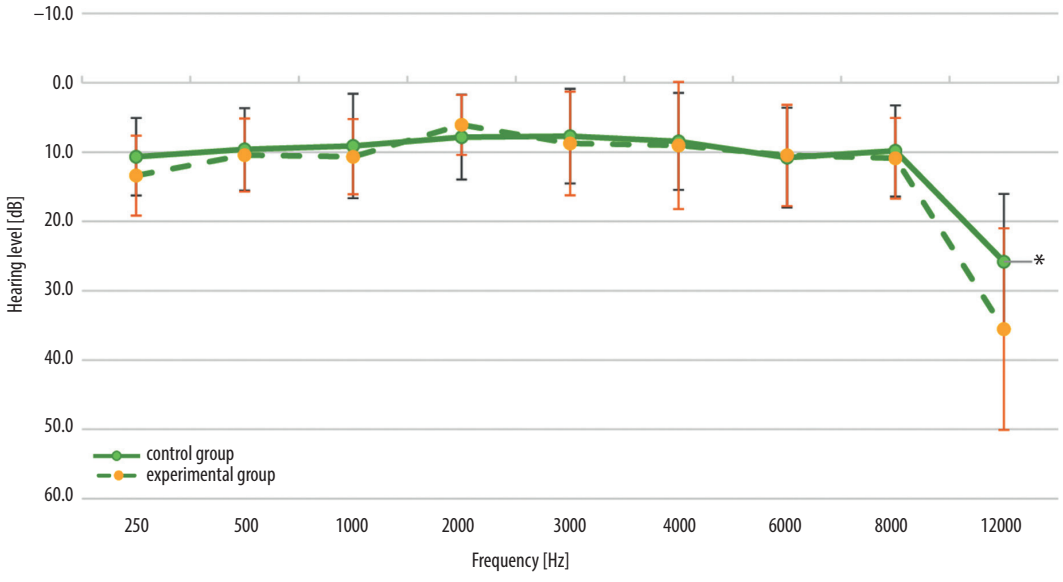

Figure 1. Hearing thresholds for the noise-exposed experimental group and the control group (means and *SD*). Conventional frequencies plus 12 kHz. * = significant difference

- ABR testing at a suprathreshold intensity of 80 dB normal hearing level (nHL) using a click (with pulses of alternating polarity at a rate of 13.1 per second and filtered from 0.1 to 1.5 kHz). This testing assessed the amplitude and latency of ABR waves I and V, as well as the time interval between waves I and V. All test subjects were evaluated by the Interacoustics Eclipse device.

Statistical analysis was done using IBM SPSS Statistics 26. Descriptive statistics, bivariant correlations, and independent samples tests were calculated. Correlations were considered significant at the 0.05 level. For statistical analysis the results for each ear were analyzed separately. In the experimental group $n = 84$ and in the control group $n = 79$.

Results

According to the PTA data in the standard 0.25–8 kHz range, no significant differences were found between the soldier group and the control group. At all frequencies, air conduction thresholds of soldiers and the control group were all under 20 dB HL. However, as shown in **Figure 1**, there was a significant difference between thresholds at 12 kHz ($p < 0.05$). Specifically, the mean PTA result at 12 kHz of the soldiers was 35.5 ± 14.5 dB, whereas that of the control group was 25.8 ± 9.8 dB. As per ISO 7029 standards, hearing at 12 kHz should be less than 20 dB in both our age groups, but in our control group there was still a mild hearing loss at this frequency [15]. The difference between the ISO 7029 data and hearing threshold data for other countries [16] tends to be more pronounced for male adults than for female adults and for higher frequencies than for lower frequencies [16]. The authors consider that the next revision of ISO 7029 will need to be based on data from various countries with uniform ages, frequency ranges, and threshold calculation methods in order to more accurately reflect hearing threshold data.

The discomfort thresholds between the two groups were not significantly different (**Figure 2**). In both groups, the threshold of discomfort was 90 dB HL or greater at all frequencies.

The DP-grams were recorded at frequencies of 1, 2, 4, 6, and 8 kHz. DPOAE testing revealed DP-level differences at some of the tested frequencies between the two groups, but the only significant difference was at 4 kHz ($p = 0.008$). Noise floor levels at all frequencies were not significantly different (**Figure 3**). In the control group there were low values of DP level at high frequencies, and these may be related to the mild hearing loss at 12 kHz, given that DPOAEs are more sensitive for detecting high-frequency hearing loss [17].

Statistical analysis of the ABR results showed significant differences in ABR wave I amplitude and latency between the two groups ($p < 0.0005$ and $p = 0.007$, respectively). The differences in the ABR wave V amplitude and latency were also significant ($p = 0.023$ and 0.044 respectively), but less so than for wave I. Wave I–V interpeak latencies (IPLs) were not different between the two groups ($p = 0.7$).

Discussion

High-level noise exposure can result in some hearing impairment, but the related changes in the hearing system emerge long after the exposure events. However, it is possible to detect these changes at the earlier, nonsymptomic stage with electrophysiological audiological examinations.

Our study showed that the ABR wave I amplitude was lower in the soldiers than in the control group participants, corroborating findings in other studies [1,3]. Studies in animal models with noise-induced and age-related synaptopathies have also shown reductions in the ABR wave I amplitude [18–1]. Such interrelations between the ABR

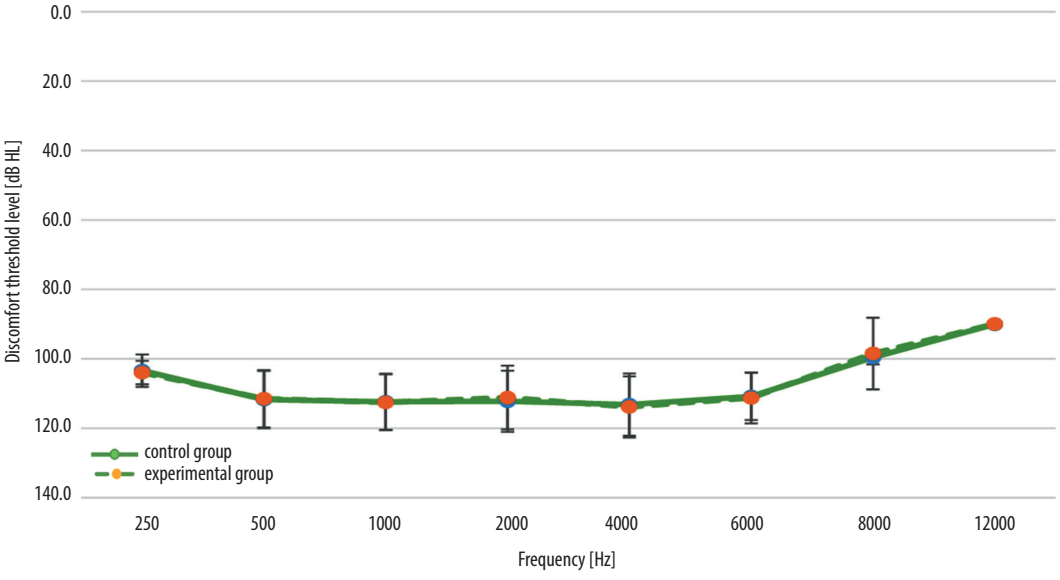

Figure 2. Discomfort thresholds at each frequency for the noise-exposed experimental group and the control group (means and *SD*). Conventional frequencies plus 12 kHz

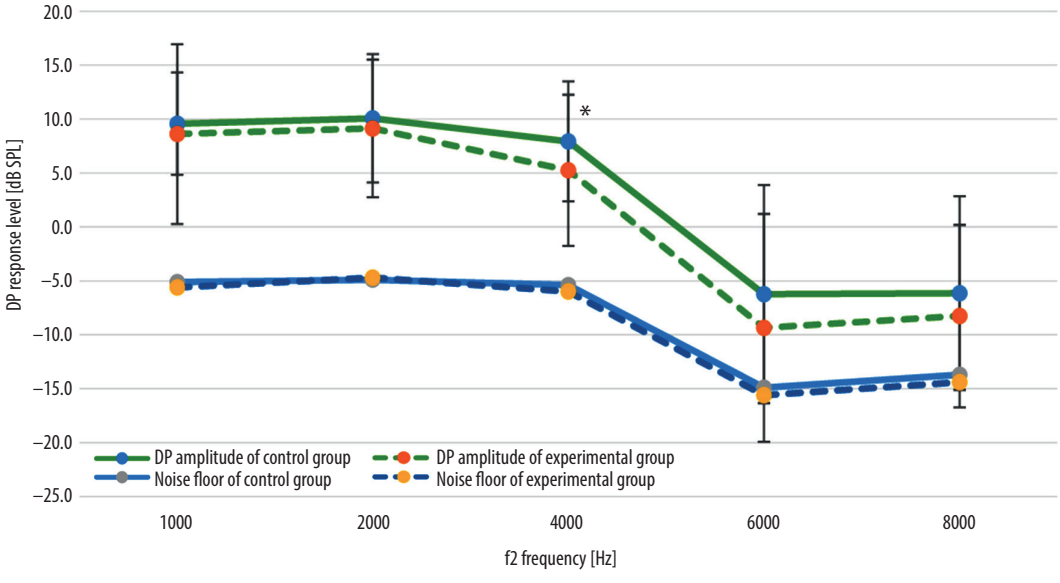

Figure 3. DPOAE amplitude as a function of f2 frequency for the noise-exposed experimental group and the control group (means, *SD*, and average noise floors). * = significant difference

wave I amplitude and synaptopathic processes are also noticeable in humans. Our study could not confirm that a reduced ABR wave I amplitude is conditioned by the synapse loss alone, as we also recorded decreased DPOAE threshold levels at 4 kHz, which reflects outer hair cell damage. Some studies do not support the use of DPOAE assessment as a method for the early detection of noise-induced damage before the behavioural threshold is changed [3]. But a previous study of firearm users revealed significantly lower DPOAEs than predicted from hearing thresholds [22]. Nadon and others demonstrated that the monitoring of an individual's OAEs could be useful in monitoring temporary changes in hearing status induced by exposure to ambient noise [23].

Of course, duration of noise exposure is important too. Trzaskowski and colleagues found that 30-min exposure to amplified music at 87 dBA did not cause measurable PTA threshold shift or significant changes in TEOAE and DPOAE parameters [24]. Another group of researchers demonstrated that a duration of noise from 30 to 60 min changed temporary threshold shifts at several frequencies,

both by conventional and extended high-frequency (EHF) audiometry, but they were minor [25]. Our participants were exposed to loud noise for 44 days.

Research about immediate and long-term impacts of military aircraft noise exposure on noise-induced hearing loss concluded, that EHF is more sensitive in detecting potentially lasting noise-induced hearing loss, whereas DPOAEs are more able to reveal the immediate noise impact on hearing [26].

As per our results there was difference in 10 dB among two groups in 12 kHz frequencies. Similar results have been reported by other authors. Konopka and colleagues showed significant deterioration of hearing, on average by 6 dB, exclusively at frequencies of 10 and 12 kHz after military service [27]. Another group of researchers reported that in adults subjected to steady-state noise mean thresholds from 8–12 kHz were up to 20 dB poorer than in a sample of young normal adults [28]. Some studies do not support the use of EHF audiometry in assessing and monitoring noise-induced hearing loss [29]. In another study similar to ours done on civilian pilots 20–39 years old, a 7.8–9.9 dB decrease in EHF audiometry was reported [30]. Although some authors have documented decreases at standard frequencies in noise exposure groups, it is generally considered that EHF audiometry is more sensitive than conventional audiometry.

Büchler and colleagues believe that PTA remains the most important measurement to monitor acute acoustic trauma, while it may be useful to complement it with EHFA, focusing on the 11–14 kHz range; OAEs are best analysed in the 3–6 kHz range [31]. Based on our results, we come to same conclusion, as we found a significant difference in our groups at 12 kHz, while DPOAE testing revealed a significant difference at 4 kHz.

Our study also confirms an abnormal increase in ABR wave I latencies. A similar result has also been shown by other authors in normal hearing patients with tinnitus. In patients with hearing loss there is an abnormal prolongation of ABR wave I latency, with similar increases in the latencies of later ABR waves [32]. Our study also supports this finding, with a significant hearing threshold reduction at 12 kHz in the high-level noise-exposed patient group relative to the control group. Other authors have stated that, in normal hearing people with tinnitus, prolonged IPLs of ABR waves III–V point to an increased neural conduction time in the upper brainstem, which can be attributed to impaired neural synchronization and transmission in the auditory pathways [33,34]. Our study did not find any evidence of prolonged ABR wave I–V latencies, which suggests that, in high-level noise-exposed individuals with normal hearing, such changes had not yet developed.

According to Kaf and colleagues, noise exposure can result in decreases in the amplitudes of ABR waves I and V [35]. Our study showed such decreases and that the amplitude of wave I had decreased significantly ($p < 0.0005$). In contrast, Suresh and Krishnan saw a smaller ABR wave I amplitude in the noise exposure group than in the low-risk group, alongside similar amplitudes of ABR waves III and V [36]. In contrast to these findings, according to data from Stamper and Johnson [3], the amplitudes of ABR waves I and V were not significantly related to the subjects' NEBs.

We found no significant difference in Loudness Discomfort Level (LDL) between the two groups ($p > 0.05$). Liberman and colleagues found that their high noise exposure group was more likely to report irritation caused by everyday sounds and to avoid noisy environments than did their low noise exposure group [37]. We checked LDL assuming that hyperacusis might be revealed, but it appears that LDL alone is not a good indicator of this condition [38,39].

The significant DPOAE level reduction at 4 kHz, together with the significant reduction in ABR wave I amplitude, in the high noise exposure group may be indicative of simultaneous outer hair cell damage and synaptopathic impairment. Our study confirms the necessity of assessing hearing function in vulnerable patient groups. It appears that the techniques we used may be useful in predicting the development of hearing loss later on.

Conclusion

The results of our study have confirmed that standard audiometric tests are not sufficient to evaluate the effect of gunfire exposure on hearing. Combat soldiers require EHF audiometry, DPOAE testing, and electrophysiological testing. Our findings show the importance of follow-up monitoring of auditory function in noise-exposed individuals. Future studies are needed to determine whether hearing loss develops later in these patients and whether there are ways to prevent this impairment.

Acknowledgements

This work was supported by the Science Committee of the Republic of Armenia in the frames of research project No 21T-3B161.

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