

# Noise-Induced Cochlear Synaptopathy in Dental Prosthesis Students

✉ Bünyamin Çıldır<sup>1</sup>, ✉ Suna Tokgöz-Yılmaz<sup>2</sup>

<sup>1</sup>Department of Language and Speech Therapy, Ankara Yıldırım Beyazıt University Faculty of Health Sciences, Ankara, Türkiye

<sup>2</sup>Department of Audiology, Ankara University Faculty of Health Sciences, Ankara, Türkiye

## Abstract

**BACKGROUND/AIMS:** Noise causes damage to cochlear hair cells and loss of sensitivity for low volume sounds. Hidden hearing loss is a functional disorder which can be seen in individuals with noise exposure history but no permanent threshold loss. We aimed to determine which tests can be used to diagnose hidden hearing loss in dental prosthesis associate degree students with normal hearing who are exposed to noise.

**MATERIALS AND METHODS:** Ninety individuals between the ages of 19-35 whose pure tone average was within normal limits were included in our study. These individuals were divided into two groups according to their noise exposure score as the high-risk group (n=45) and the low-risk group (n=45). Auditory brainstem response (ABR) and amplitude modulation detection tests were performed with and without background noise after standard audiometric tests and otoacoustic emission suppression measurement.

**RESULTS:** The otoacoustic emission suppression values of those individuals in the high-risk group were found to be significantly lower than those in the low-risk group. As the stimulus level increased, the differentiations (amplitude increase and latency decrease) in the first wave of ABR without background noise were observed in those individuals in the low-risk group. The recognition threshold score which was modulated to the amplitude was found to be lower in the presence of background noise than in the absence of background noise for all participants.

**CONCLUSION:** Although noise exposure does not result in any permanent differences in hearing thresholds, the otoacoustic emission suppression values, the differentiations of the first wave and the amplitude modulation detection values can provide useful information in the diagnosis of hidden hearing loss in individuals with normal hearing.

**Keywords:** Hidden hearing loss, auditory brainstem response, otoacoustic emission suppression, amplitude modulation detection

## INTRODUCTION

Exposure to high intensity noise may cause unwanted sounds which mask speech and communication, as well as the long-term exposure to such noises causing physical (such as temporary hearing loss, increased blood pressure) and mental effects (such as stress, anxiety, mental difficulty).<sup>1-3</sup> These effects may vary depending on the duration of exposure to noise, the distance to the source and the sensitivity of the person.<sup>1,3</sup>

The loss of the synaptic connection between the inner hair cell and spiral ganglion cells after noise exposure is the first pathological finding of temporary hearing loss.<sup>4</sup> This deterioration is called "synaptopathy" due to the connection loss between the lesion area inner hair cell ribbon synapses and afferent hearing nerves. It is also known as "hidden hearing loss" since it does not reach hearing thresholds.<sup>4</sup>

Hidden hearing loss is a functional disorder which can be seen in individuals with noise exposure history but no permanent threshold

**To cite this article:** Çıldır B, Tokgöz-Yılmaz S. Noise-Induced Cochlear Synaptopathy in Dental Prosthesis Students. Cyprus J Med Sci 2024;9(1):51-57

**ORCID IDs of the authors:** B.Ç. 0000-0002-5632-1650; S.T.Y. 0000-0002-4656-099X.



**Address for Correspondence:** Bünyamin Çıldır

**E-mail:** bunyamin.cildir@gmail.com

**ORCID ID:** orcid.org/0000-0002-5632-1650

**Received:** 18.02.2021

**Accepted:** 13.06.2021



Copyright© 2024 The Author. Published by Galenos Publishing House on behalf of Cyprus Turkish Medical Association.

This is an open access article under the Creative Commons AttributionNonCommercial 4.0 International (CC BY-NC 4.0) License.

loss.<sup>5</sup> We can investigate functional distortions in outer hair cells (OHC) by determining the lowest discernible hearing threshold levels in a given frequency region with the use of pure tone audiometry.<sup>6</sup> Hidden hearing loss is assumed to affect low-spontaneous rate fibres with high thresholds, which are responsible for encoding moderate to high noise intensity.<sup>7</sup> In this pathology which occurs after noise exposure, due to the peripheral loss of hearing nerve fibres with a high threshold and low spontaneous rate, no deterioration occurs in low-intensity sounds.<sup>7</sup> High-intensity sounds, consistent with low SR fibre loss, are associated with non-abnormal results.<sup>8</sup> It has been indicated that hidden hearing loss cannot be determined by audiometric results but there is a decrease in the amplitude of wave I of the auditory brainstem responses (ABR) to moderate-to-high intensity stimuli,<sup>8</sup> and amplitude modulation detection values.<sup>9</sup> In the study by Liberman et al.<sup>10</sup>, normal hearing individuals with low-risk of noise exposure and normal hearing individuals with high-risk were compared in terms of cochlear synaptopathy, and the amplitude of wave I of the ABR responses was not found to be decreased in the high-risk group. In another study conducted on individuals with and without tinnitus, it was reported that the amplitudes of individuals with tinnitus decreased more with wave I of the ABR compared to the group without tinnitus.<sup>11</sup> Dentists or dental assistants can be exposed to different noise levels and types while working in dental offices or laboratories.<sup>12</sup>

In our study, we aimed to determine whether the ABR and distortion suppression of otoacoustic emissions and amplitude modulation detection (AMDT) tests and speech in noise tests would be useful in determining whether there is latent hearing loss in individuals with dental prosthesis associate degrees with normal hearing. At the same time, the results of dental prosthesis associate degree students (dental auxiliaries) were compared with the results of individuals with normal hearing who were not exposed to noise, and the damage caused by noise exposure to the hearing system was evaluated.

## MATERIALS AND METHODS

Our study was approved by the Ethics Committee of Ankara Yıldırım Beyazıt University and implemented according to the Helsinki Declaration (approval number: 56/15). The individuals who applied for occupational health check-ups were informed about the aims of this study and consent was obtained from those who agreed to participate.

**Participants:** A total of 90 individuals, who were aged between 19-35 years ( $23.45 \pm 3.67$ ) took part in this study. Dental prosthesis associate degree individuals with high-risk ( $n=45$ ; noise exposure score=5 or above) exposed to noise between the ages of 19-35 were compared with low-risk normal hearing low noise exposure ( $n=45$ ; noise exposure score=4 or below) according to their one-minute noise questionnaire score. Our study was conducted with Ankara University, Vocational High School, dental prosthesis associate degree students and undergraduate students with normal hearing and no history of noise exposure. This study was conducted in Ankara University Faculty of Health Sciences, Department of Audiology between 2016-2018. Individuals who were over 18 years and with no hearing loss history, neurological disorders, or tinnitus were included in this study (hearing thresholds  $\leq 20$  dB HL at 125 to 12,000 Hz). Four participants with hearing thresholds higher than 20 dB HL were excluded from this study. After otoscopy, all participants were tested by pure tone audiometry, immittance, speech in noise, Distortion Product Otoacoustic Emission (DPOAE) suppression measurement, auditory brain stem response, and

AMDT with and without background noise. The measurements of all participants were made when the schools entered the semester break. None of the participants entered the laboratory within this time period (approximately 3 weeks).

**Evaluation of noise exposure and sensitivity:** The participants' noise exposure scores were determined by a 1-minute noise screen questionnaire.<sup>13</sup> Those with a score of 5 or above were evaluated as being high-risk individuals and those with a score of 4 or below were evaluated as being low-risk individuals according to their questionnaire responses. In addition, in order to evaluate the participants' noise sensitivity, we used the Turkish version of the Weinstein noise sensitivity scale which had been tested for its validity and reliability. The total score is calculated by giving 1 to 6 points (agree/disagree) to 21 questions in this scale (the highest possible score is 126).<sup>14</sup> At the same time, the noise level of the dental prosthesis laboratory was measured using the Larson Davis system 824 sound level meter. The noise levels of the laboratory were measured every 10 minutes (10.00-12.00 and 13.00-14.00) during a three-month period (excluding weekends) from the same point in the middle of the laboratory while all devices in the laboratory were working. Measurements were made a total of 18 times every day.

## Speech in Noise Test

This test is carried out using monosyllabic word lists in background noise such as narrowband and white noise.<sup>15,16</sup> This test can be performed at different signal to noise ratios as well as at fixed signal to noise ratios.<sup>17</sup> In our study, 50-word monosyllabic word lists were given at 40 dB SL, with a constant +10 dB signal/noise ratio to the ear to be tested for both noise and speech stimuli. Each ear was evaluated separately.

**Pure tone audiometry and immittance measurements:** Pure tone audiometry were performed with Sennheiser TDH 49 P supra-aural headset in the range of 0.125 to 8 kHz and with Sennheiser HDA 200 circumaural headphones in the range of 8 to 12 kHz according to the British Society of Audiology (2011). The participants' middle ear pressure values were between  $\pm 50$  daPa and performed by GSI TympStar device.

**Contralateral suppression of distortion product otoacoustic emissions:** DPOAE is an electro-acoustic measurement which reflects the mechanical properties of the OHCs. The DPOAE test is performed by simultaneously transmitting two pure tone sounds (to the ear at different frequencies  $f_1$  and  $f_2$  using an  $f_2/f_1$  ratio of 1.2).<sup>18</sup> The DPOAE amplitude was chosen when L1 (75 dB SPL) was 10 dB higher than L2 (65 dB SPL). The contralateral DPOAE suppression test causes suppression of OHCs with contralateral noise. The contralateral DPOAE suppression test can either be performed by decreasing the amplitude (Input-Output-I/O function) or by changing the frequency values of the constant amplitude (DP-Gram).<sup>19</sup> We used the Otometrics (Denmark, Taastrup) Capella DPOAE. DP-gram was obtained at octave frequencies of 200, 2,383, 3,359 and 4,004 Hz at 60 dB SPL broadband-contralateral white noise Hz using contralateral TDH39 headphones.

**Auditory brainstem response:** ABR measurements of the participants were performed in a natural sleep state in a Faraday cage test room using Vivasonic (Canada, Toronto) Integrety™ V500 Auditory Diagnostic System in two channels. The active electrode ( $F_z$ ) was placed on the upper part of the forehead, the ground electrode ( $F_{pz}$ ) was placed on the lower part of the forehead, one of the reference electrodes was placed on the left (M1) and the other was placed on the right mastoid (M2) region (electrode skin impedance below 3 k $\Omega$ ). The click stimuli

(with 1,024 repeats) at alternative polarity were transmitted through ER-3A insert headphones, using a 30 Hz high-pass filter (high-pass). ABR recordings were kept by using a 100  $\mu$ s stimulus at a rate of 9.1 Hz (rate) at 70, 80, 90 and 99 dB nHL levels to the opposite ear, for both cases of a 55 dB nHL broadband mask and without mask.

**Amplitude modulation detection test:** The AMDT is used to detect amplitude modulation sounds which are assumed to decrease after noise exposure because of impairment in individual's over-threshold responses. AMDT was performed with and without contralateral narrowband noise (40 dB SPL).<sup>20</sup> The bandwidth of narrowband noise was set to 1/3 of an octave in this study. A sinusoidal sound modulated to 19 Hz with a carrier frequency of 5 kHz at 75 dB SPL level was used. The AMDT test was performed using the Parameter Estimation by Sequential Testing (3 interval-3 alternative selection methods) and the p-value was taken as 0.75. One of three randomly transmitted sounds was modulated to stimulus amplitude and the other two were not modulated. While the target tone was being transmitted, the AM depth was initially set as 6 dB (50%) and was adaptively randomized until the final modulation change size reached 0.45 dB; the mean value of the last two steps was accepted as the threshold.<sup>21</sup> We used AMDT software with the Matlab 2019.b program.

### Statistical Analysis

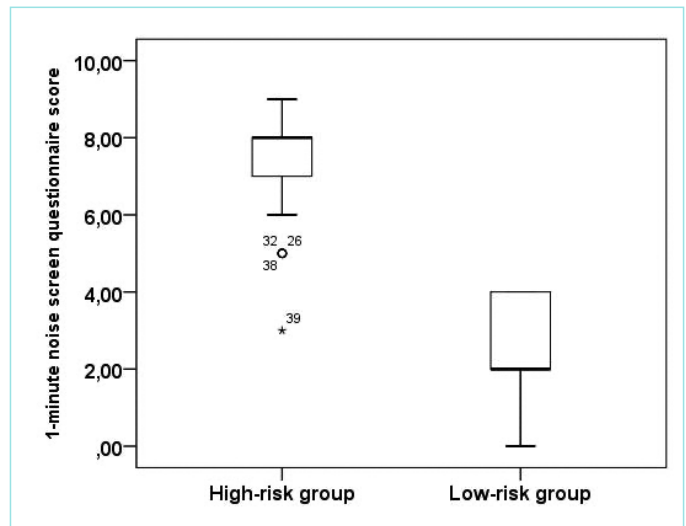
Data analysis was performed using SPSS v.23.0 (Statistical Package for Social Sciences) (SPSS Inc., Chicago, IL, US). The normal distribution of our study was examined with the Shapiro-Wilk test. A chi-square test was used to compare the demographic findings. The non-parametric Mann-Whitney U test was used for comparisons between the high-risk and low-risk groups. The Wilcoxon test was used for in-group comparison and the Spearman test was used for the relationships between two variables. The p-value for statistical significance was accepted as  $<0.05$ .

## RESULTS

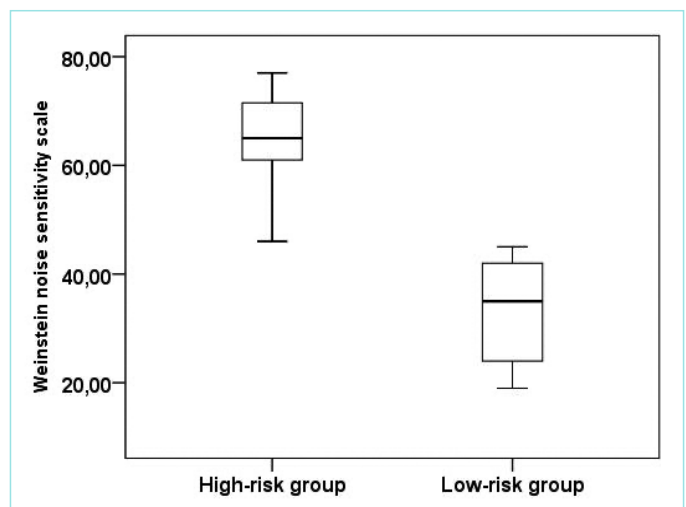
**Noise exposure and sensitivity:** The average noise level of the dental laboratory was  $97 \pm 11.3$  dB (A) at 10.00 a.m. and  $102 \pm 9.3$  dB (A) at 2 p.m. These hours are when the laboratory was very busy. Of the 45 participants in the high-risk group (dental prosthesis associate degree students), 41 reported that they were exposed to sounds such as wedding halls, ambulances, and traffic noise during the day. 85% of the individuals in the low-risk group stated that they were exposed to traffic noise. As shown in Figure 1, the 1-minute noise screen scores of the individuals in the high-risk group ( $7.29 \pm 1.23$ ) were found to be statistically higher than the low-risk group's scores ( $2.81 \pm 1.35$ ) ( $p=0.001$ ). The Weinstein's noise sensitivity scale scores of the individuals in the high-risk group ( $65.44 \pm 7.53$ ) were statistically higher than the scores of the low-risk group ( $34.16 \pm 8.14$ ) ( $p=0.011$ ) (Figure 2). A positive and high correlation was observed between the 1-minute noise screen and Weinstein's noise sensitivity scale of all individuals ( $r=0.822$ ,  $p=0.001$ ).

**Pure tone audiometry:** All participants had normal hearing thresholds (equal to or less than 20 dB HL at 125-8,000 Hz octave frequencies). No statistical difference was observed between the hearing thresholds of the groups ( $p>0.05$ ).

**Speech in noise test:** Although no statistically significant difference was found between the scores of speech understanding in noise of the high and low-risk groups for both ears, the scores of the high-risk group were lower than the scores of the low-risk group.



**Figure 1.** One-minute noise screen questionnaire findings of both groups, error bars show the standard deviation.



**Figure 2.** Weinstein noise sensitivity scale findings of both groups, error bars show the standard deviation.

**Contralateral suppression of distortion product otoacoustic emissions:** Contralateral DPOAE suppression amounts were calculated at all frequencies and were found to be 0.84 dB SPL for individuals at high-risk, and 2.02 dB SPL for individuals with low-risk. The difference in the contralateral DPOAE suppression amounts between the two groups was statistically significant ( $p=0.014$ ).

**Auditory brainstem responses:** ABR records of all participants were made on the right ear of 68 patients and the left ear of 16 patients. While the amplitude of wave I of ABR with mask was statistically smaller than the amplitude value obtained without mask at 99 dB nHL in the high-risk group ( $z=-3.087$ ,  $p=0.002$ ), the amplitude was found to be bigger at 80 dB nHL without mask ( $z=-3.155$ ,  $p=0.002$ ) (Table 1). A negative value was found in the amplitude of wave I of ABR in 10 individuals in the low-risk group and 8 individuals in the high-risk group. The latency of wave I obtained with mask was statistically significantly shorter

than the latency without mask at 90 dB nHL ( $z=-2.178$ ,  $p=0.029$ ) and 99 dB nHL ( $z=-2.449$ ,  $p=0.014$ ) in the high-risk group. There was no statistically significant prolongation of the ABR wave V latency with mask in the high-risk group ( $p>0.05$ ).

As shown in Table 2, for the low-risk group, the wave I amplitude ( $z=-2.587$ ,  $p=0.010$ ) at 70 dB nHL and the wave III amplitude at 90 dB nHL ( $z=-2.807$ ,  $p=0.005$ ) values were found to be statistically lower with mask. There was no statistical difference between the amplitude and latency values of the wave I, III and V with mask and without mask at other (80 and 99 dB nHL) intensity levels ( $p>0.05$ ). Although there was no statistical difference, it was observed that the amplitude increased as the stimulus intensity level rose.

For the high-risk group, the wave V amplitude was found to be smaller with mask than without mask at 99 dB nHL ( $z=-2.562$ ,  $p=0.010$ ) and 90 dB nHL ( $z=-1.999$ ,  $p=0.046$ ), but it was found to be bigger at 80 dB nHL ( $z=-3.905$ ,  $p=0.001$ ) (Table 1).

There were statistically significant differences in the wave I amplitude at 70 dB nHL ( $U=288$ ,  $p=0.001$ ), 80 dB nHL ( $U=586.5$ ,  $p=0.011$ ) and 99 dB nHL ( $U=635.5$ ,  $p=0.035$ ) with mask between the two groups. However,

there was no difference at all stimulus levels without mask (Table 1, 2) ( $p>0.05$ ).

**Amplitude modulation detection threshold:** For both groups, the AMDT scores were evaluated both with mask and without mask (Table 3). AMDT values with mask were statistically higher (worse) than the AMDT values without mask in all individuals ( $p=0.001$  for the high-risk group;  $p=0.035$  for the low-risk group). The AMDT values in the high-risk group were lower (better) than the AMDT values in the low-risk group ( $p<0.001$ ).

## DISCUSSION

Noise-related hidden hearing loss is a functional disorder which is seen in individuals who suffer from noise exposure without hearing loss<sup>5</sup> and it is also known as cochlear synaptopathy.<sup>8</sup> In a cochlear synaptopathy study on mice, no difference was observed in the hearing thresholds which were measured with ABR, although half of the synapses between the inner hair cell and spiral ganglion neurons were lost due to noise exposure.<sup>22,23</sup> In synapse loss after noise exposure, while otoacoustic emissions were obtained, the slope of the ABR wave I amplitude was observed at high stimulus levels.<sup>1</sup>

**Table 1. Auditory brainstem responses amplitude with/without mask at different stimulus level for the high-risk group**

Mask	Stimulus level	Wave I, Mean $\pm$ SD	p	Wave III, Mean $\pm$ SD	p	Wave V, Mean $\pm$ SD	p
With mask	99 dB nHL	0.12 $\pm$ 0.28	0.002*	0.33 $\pm$ 0.25	0.169	0.45 $\pm$ 0.22	0.010*
Without mask		0.21 $\pm$ 0.25		0.37 $\pm$ 0.27		0.75 $\pm$ 0.96	
With mask	90 dB nHL	0.17 $\pm$ 0.25	0.608	0.34 $\pm$ 0.22	0.797	0.56 $\pm$ 0.17	0.046*
Without mask		0.17 $\pm$ 0.19		0.31 $\pm$ 0.16		0.49 $\pm$ 0.17	
With mask	80 dB nHL	0.22 $\pm$ 0.23	0.002*	0.31 $\pm$ 0.19	0.103	0.50 $\pm$ 0.17	0.001*
Without mask		0.11 $\pm$ 0.20		0.25 $\pm$ 0.20		0.36 $\pm$ 0.17	
With mask	70 dB nHL	0.19 $\pm$ 0.22	0.063	0.21 $\pm$ 0.18	0.395	0.35 $\pm$ 0.22	0.331
Without mask		0.16 $\pm$ 0.36		0.25 $\pm$ 0.22		0.33 $\pm$ 0.19	

\* $p<0.05$ , SD: Standard deviation.

**Table 2. Auditory brainstem responses amplitude with/without mask at different stimulus level for the low-risk group**

Mask	Stimulus level	Wave I, Mean $\pm$ SD	p	Wave III, mean $\pm$ SD	p	Wave V, Mean $\pm$ SD	p
With mask	99 dB nHL	0.15 $\pm$ 0.14	0.786	0.29 $\pm$ 0.16	0.678	0.53 $\pm$ 0.22	0.120
Without mask		0.14 $\pm$ 0.17		0.27 $\pm$ 0.19		0.46 $\pm$ 0.25	
With mask	90 dB nHL	0.10 $\pm$ 0.16	0.223	0.25 $\pm$ 0.15	0.005*	0.38 $\pm$ 0.24	0.275
Without mask		0.18 $\pm$ 0.22		0.35 $\pm$ 0.18		0.44 $\pm$ 0.19	
With mask	80 dB nHL	0.77 $\pm$ 0.23	0.488	0.23 $\pm$ 0.26	0.771	0.36 $\pm$ 0.15	0.322
Without mask		0.12 $\pm$ 0.17		0.30 $\pm$ 0.63		0.40 $\pm$ 0.17	
With mask	70 dB nHL	-0.1 $\pm$ 0.21	0.010*	0.16 $\pm$ 0.30	0.662	0.35 $\pm$ 0.27	0.411
Without mask		0.05 $\pm$ 0.17		0.19 $\pm$ 0.16		0.30 $\pm$ 0.14	

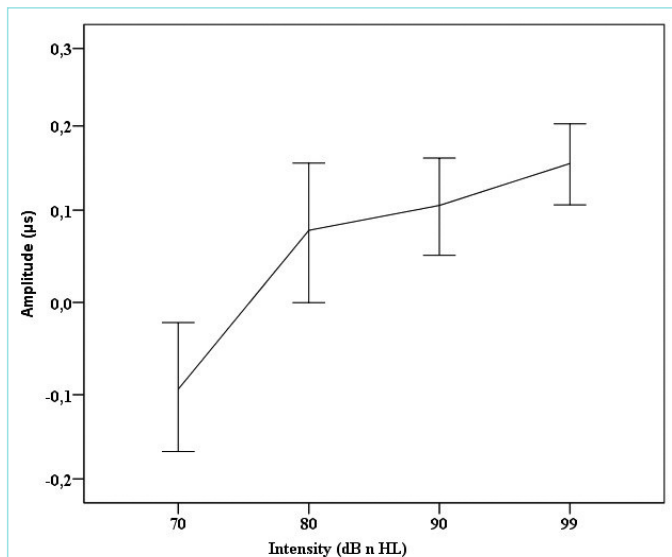
\* $p<0.05$ , SD: Standard deviation.

**Table 3. Amplitude modulation detection test findings with/without mask for two groups**

Groups	AMDT with mask		AMDT without mask		p
	Median (min.-max.)	Mean $\pm$ SD	Median (min.-max.)	Mean $\pm$ SD	
High-risk	-40.1 (-46.1, -32.6)	-39.9 $\pm$ 3.16	-40.8 (-49.8, -28.8)	-41.2 $\pm$ 4.57	0.035*
Low-risk	-32.2 (-41.5, -26.3)	-32.7 $\pm$ 3.58	-37.6 (-43.1, -31.1)	-37.5 $\pm$ 3.19	0.001*
p-value	0.001*		0.001*		

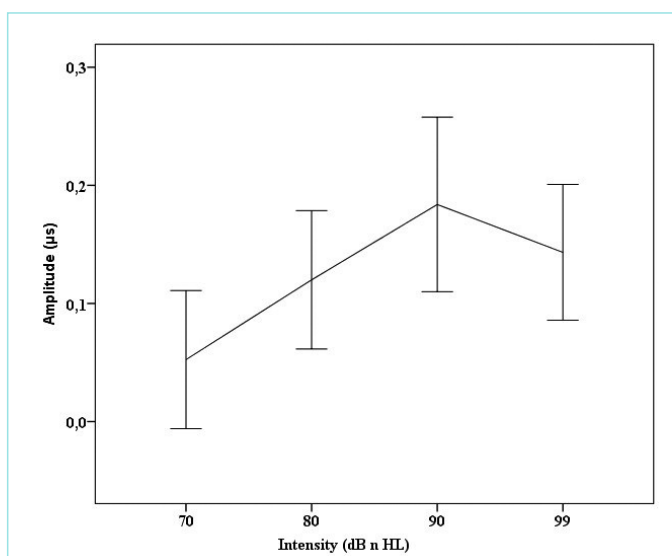
AMDT: Amplitude modulation detection test, Min.: Minimum, Max.: Maximum, SD: Standard deviation, \* $p<0.05$ .

As shown in Figure 3, 4, we found a decrease in the suprathreshold ABR wave I amplitude with mask in the high-risk group compared to the low-risk group. This decrease in amplitude value was highlighted in another study about mild and high spontaneous rate fibre loss-induced hidden hearing loss.<sup>4</sup> In animal studies, it has been reported that decreases in wave I amplitude are associated with synaptic loss with long-term exposure to noise, but the measurement of the wave I amplitude in humans does not provide precise information on the evaluation of synaptopathy.<sup>8</sup> Studies have indicated that individuals with noise exposure may experience difficulties in terms of supra-threshold processing skills, such as poor understanding and impaired



**Figure 3.** Amplitude findings of wave I of ABR with mask in individuals in the high-risk group.

ABR: Auditory brainstem response.



**Figure 4.** Amplitude findings of wave I of ABR without mask in individuals in the high-risk group.

ABR: Auditory brainstem response.

attention.<sup>24</sup> However, exposure to noise did not cause any hearing loss in the high-risk group in our study.

We observed an increase in the amplitude of wave I without mask and a decrease with mask as the stimulus level increased ( $p < 0.05$ ). There was a minimum prolongation in the latency of wave V with mask in comparison to without mask in the high-risk group, which was found to be compatible with previous studies.<sup>25,26</sup> As a finding different from the literature, we found that the masked wave I latency was shorter than the unmasked wave I latency at suprathreshold levels in the high-risk group. In our study, the change in the wave V latency was accompanied by a decrease in the wave I amplitude in the high-risk group, suggesting that there may be hidden hearing loss in these individuals. This change mostly reflects the activities of low spontaneous fibre in the presence of background noise.<sup>27</sup>

The AMDT was performed with and without mask and was lower in the high-risk group than for those in the low-risk group with mask ( $p < 0.05$ ). This finding suggests that the temporal modulation of the sound may impair auditory sensitivity and may be useful in diagnosing hidden hearing loss.

The finding that individuals in the high-risk group with mask had lower AMDT scores than those in the low-risk group may be useful in the diagnosis of hidden hearing loss (Table 3). In studies which were previously conducted, it was stated that ABR with mask may reflect the efferent auditory system.<sup>28</sup> In our study, there was no change in the wave I amplitude at stimulus levels of 70 and 80 dB nHL with mask for both groups. This finding is consistent with the study conducted by Matas et al.<sup>29</sup>

In our study, individuals in the high-risk group had lower suppression values with contralateral noise than those individuals in the low-risk group ( $p < 0.05$ ). This finding is thought to be due to the fact that contralateral noise results in DPOAE suppression by activating efferent neurons as well as a decrease in afferent electric activity.<sup>30</sup> Marques and da Costa<sup>31</sup> highlighted that especially DPOAE data would be beneficial in the early diagnosis of cochlear impairments before noise induced hearing loss occurs due to noise exposure.

We thought that a decrease in suppression, I-wave amplitude and the AMDT score may indicate hidden hearing loss which leads to a decrease in communication skills. The background noise (40dB SPL) which was used in our study can be used to detect low spontaneous fibre loss, especially since it causes suppression of high spontaneous fibre.

Although the ABR test is the gold standard in the diagnosis of occult hearing loss in our study and in the literature, the diagnosis of occult hearing loss with only the ABR test is insufficient in revealing all the signs of the disease in real terms. Therefore, when diagnosing latent hearing loss, all objective and subjective tests such as AMDT, otoacoustic emission tests, adaptation test, and the ABR test should be used together.

## CONCLUSION

The control of noise in high-noise environments such as dental prosthesis laboratories is important for the health of employees due to hearing loss which can be seen in groups with high exposure to noise. In our study, it was observed that ABR wave I amplitude decreased as the intensity increased in the high-risk group. In the low-risk group, it was

observed that, as the stimulus intensity increased, ABR wave I amplitude wave increased. This is a finding that should be taken into consideration in the diagnosis of occult hearing loss. At the same time, periodically performing audiometric tests on technicians working in these locations and using earplugs to minimize the noise exposure of these individuals can help prevent hearing loss. In the process of diagnosing individuals with hidden hearing loss, differentiations in the ABR wave I amplitude, the AMDT and suppression otoacoustic emission (especially DPOAE) may be useful in the early diagnosis of hidden hearing loss.

## MAIN POINTS

- Noise exposure can cause wave differences in auditory brainstem responses without causing permanent loss of hearing thresholds.
- Continuous exposure to high intensity noise in dental prosthesis associate degree students can cause permanent loss of auditory nerve cells.
- The combined use of amplitude modulation detection tests, otoacoustic emission tests, auditory brainstem response and speech in noise tests provides useful information in the diagnosis of occult hearing loss.

## ETHICS

**Ethics Committee Approval:** Our study was approved by the Ankara Yıldırım Beyazıt University Ethics Committee and implemented according to the Helsinki Declaration (approval number: 56/15).

**Informed Consent:** The individuals who applied for occupational health check-ups were informed about the aims of this study and consent was obtained from those who agreed to participate.

## Authorship Contributions

Concept: B.Ç., Design: B.Ç., Supervision: B.Ç., Fundings: B.Ç., Data Collection and/or Processing: B.Ç., Analysis and/or Interpretation: B.Ç., Literature Search: B.Ç., Writing: B.Ç., Critical Review: S.T.Y.

## DISCLOSURES

**Conflict of Interest:** No conflict of interest was declared by the authors.

**Financial Disclosure:** The authors declared that this study had received no financial support.

## REFERENCES

1. Mojarad F, Massum T, Samavat H. Noise levels in dental offices and laboratories in Hamedan, Iran. 2009; 181-6.
2. Setcos JC, Mahyuddin A. Noise levels encountered in dental clinical and laboratory practice. *Int J Prosthodont*. 1998; 11(2): 150-7.
3. Sampaio Fernandes JC, Carvalho AP, Gallas M, Vaz P, Matos PA. Noise levels in dental schools. *Eur J Dent Educ*. 2006; 10(1): 32-7.
4. Kujawa SG, Liberman MC. Synaptopathy in the noise-exposed and aging cochlea: Primary neural degeneration in acquired sensorineural hearing loss. *Hear Res*. 2015; 330(Pt B): 191-9.
5. Borg E, Canlon B, Engström B. Noise-induced hearing loss. Literature review and experiments in rabbits. Morphological and electrophysiological features, exposure parameters and temporal factors, variability and interactions. *Scand Audiol Suppl*. 1995; 40: 1-147.
6. Plack CJ, Léger A, Prendergast G, Kluk K, Guest H, Munro KJ. Toward a Diagnostic Test for Hidden Hearing Loss. *Trends Hear*. 2016; 20: 2331216516657466.
7. Furman AC, Kujawa SG, Liberman MC. Noise-induced cochlear neuropathy is selective for fibers with low spontaneous rates. *J Neurophysiol*. 2013; 110(3): 577-86.
8. Kujawa SG, Liberman MC. Adding insult to injury: cochlear nerve degeneration after “temporary” noise-induced hearing loss. *J Neurosci*. 2009; 29(45): 14077-85.
9. Kumar UA, Ameenudin S, Sangamanatha AV. Temporal and speech processing skills in normal hearing individuals exposed to occupational noise. *Noise Health*. 2012; 14(58): 100-5.
10. Liberman MC, Epstein MJ, Cleveland SS, Wang H, Maison SF. Toward a Differential Diagnosis of Hidden Hearing Loss in Humans. *PLoS One*. 2016; 11(9): e0162726.
11. Schaette R, McAlpine D. Tinnitus with a normal audiogram: physiological evidence for hidden hearing loss and computational model. *J Neurosci*. 2011; 31(38): 13452-7.
12. Morăraşu C, Burlui V, Bortă C, Ignat L, Bortă B, Morăraşu G. Evaluarea nivelului zgomotului în cabinetul stomatologic [The evaluation of sound level in dental practice]. *Rev Med Chir Soc Med Nat Iasi*. 2001; 105(4): 785-9.
13. Johnson TA, Cooper S, Stamper GC, Chertoff M. Noise Exposure Questionnaire: A Tool for Quantifying Annual Noise Exposure. *J Am Acad Audiol*. 2017; 28(1): 14-35.
14. Keskin M. Comparison of the Subjects with and Without Noise Sensitivity, As Determined by the Turkish Version of Weinstein Noise Sensitivity Scale (Wnss) Following Its Adaptation into Turkish. *Gazi University Institute of Health Sciences*. 2015.
15. Willeford J. Sentence tests of central auditory dysfunction. *Handbook of clinical audiology*. 1985; 3: 404-20.
16. Durankaya SM, Serbetçioğlu B, Dalkılıç G, Gürkan S, Kirkim G. Development of a Turkish monosyllabic word recognition test for adults. *The Journal of International Advanced Otology*. 2014; 10(2): 172.
17. Beck D, Benite L. A two-minute speech-in-noise test Protocol and Pilot Data. *Audiology Today*. 2019; 31(3): 28-34.
18. Gorga MP, Neely ST, Dorn PA, Hoover BM. Further efforts to predict pure-tone thresholds from distortion product otoacoustic emission input/output functions. *J Acoust Soc Am*. 2003; 113(6): 3275-84.
19. Ramos JA, Kristensen S, Beck DL. An overview of OAEs and normative data for DPOAEs. *Hear Rev*. 2013; 20(11): 30-3.
20. Zilany MS, Bruce IC, Carney LH. Updated parameters and expanded simulation options for a model of the auditory periphery. *J Acoust Soc Am*. 2014; 135(1): 283-6.
21. Taylor M, Creelman CD. PEST: Efficient estimates on probability functions. *The Journal of the Acoustical Society of America*. 1967; 41(4A): 782-7.
22. Hickox AE, Liberman MC. Is noise-induced cochlear neuropathy key to the generation of hyperacusis or tinnitus? *J Neurophysiol*. 2014; 111(3): 552-64.
23. Fernandez KA, Jeffers PW, Lall K, Liberman MC, Kujawa SG. Aging after noise exposure: acceleration of cochlear synaptopathy in “recovered” ears. *J Neurosci*. 2015; 35(19): 7509-20.
24. Brattico E, Kujala T, Tervaniemi M, Alku P, Ambrosi L, Monitillo V. Long-term exposure to occupational noise alters the cortical organization of sound processing. *Clin Neurophysiol*. 2005; 116(1): 190-203.
25. Dau T. The importance of cochlear processing for the formation of auditory brainstem and frequency following responses. *J Acoust Soc Am*. 2003; 113(2): 936-50.

26. Mitchell C, Phillips DS, Trune DR. Variables affecting the auditory brainstem response: audiogram, age, gender and head size. *Hear Res.* 1989; 40(1-2): 75-85.
27. Bourien J, Tang Y, Batrel C, Huet A, Lenoir M, Ladrech S, et al. Contribution of auditory nerve fibers to compound action potential of the auditory nerve. *J Neurophysiol.* 2014; 112(5): 1025-39.
28. Salo SK, Lang AH, Salmivalli AJ, Johansson RK, Peltola MS. Contralateral white noise masking affects auditory N1 and P2 waves differently. *Journal of Psychophysiology.* 2003; 17(4): 189-94.
29. Matas CG, Silva FN, Leite RA, Samelli AG. Study of suppression effect in the brainstem auditory evoked potential. *Pro Fono.* 2010; 22(3): 281-6.
30. Tomchik SM, Lu Z. Modulation of auditory signal-to-noise ratios by efferent stimulation. *J Neurophysiol.* 2006; 95(6): 3562-70.
31. Marques FP, da Costa EA. Exposure to occupational noise: otoacoustic emissions test alterations. *Braz J Otorhinolaryngol.* 2006; 72(3): 362-6.