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Original Article

# Effects of Aging Versus Noise Exposure on Auditory System in Individuals With Normal Audiometric Thresholds

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**BACKGROUND:** The study aims at investigating the effect of aging and noise exposure on the auditory system using auditory brainstem responses (ABRs), distortion product otoacoustic emissions (DPOAEs), and contralateral suppression of OAEs (CSOAEs). The objective was to compare DPOAEs, CSOAEs, and ABR in aged and noise-exposed individuals with the normal, to find an indicator for early diagnosis of auditory damage.

**METHODS:** Sixty adult male participants were divided into 3 groups. Group 1 included individuals not exposed to occupational noise and group 3 included individuals exposed to occupational noise who were <35 years of age. Group 2 consisted of individuals with an age range of 45-65 years without any occupational noise exposure. DPOAE fine structure was studied at 8 points per octave at different F2 frequencies. Transient evoked otoacoustic emissions (TEOAEs) were measured with and without contralateral broad band noise (BBN) at 30 dB SL (CSOAEs). ABR was recorded using click stimuli at different levels, from 90 dB nHL down to 50 dB nHL. The absolute amplitude and peak latencies for peaks I, III, and V; and the wave V/I amplitude ratio were analyzed.

**RESULTS:** In CSOAEs, group 1 showed greater contralateral suppression when compared to group 2 and group 3. The amplitude of ABR wave I and the wave V/I ratio showed a significant difference between the 3 groups, and there was a reduction in amplitude of wave I for groups 2 and 3.

**CONCLUSION:** The findings indicate that the functioning of the auditory system is affected by occupational noise exposure and aging. CSOAEs, ABR wave I amplitude, and wave V/I amplitude ratio serve as reliable markers in the identification of hidden hearing loss.

**KEYWORDS:** Hidden hearing loss, efferent auditory system, cochlear synaptopathy, aging, noise exposure

## INTRODUCTION

The sounds we hear are of different types and sources, and can damage the auditory system and cause hearing loss of varying degrees, if not at safe levels.[1] The protocols used clinically for evaluating noise-induced hearing loss (NIHL) rely strongly on behavioral measures like pure-tone audiometry, where the hallmark of NIHL is a high-frequency notching in the audiogram seen at the 3000-6000 Hz region. It is accepted widely that the permanent threshold shift following noise exposure is caused due to permanent damage to the auditory structures. Recent animal studies have revealed that exposure to noise causes not just temporary threshold shifts (TTSs), but can also produce permanent damage to the synapses in the cochlea, termed as "cochlear synaptopathy." Cochlear synaptopathy is the loss of synapses and cochlear nerve terminals innervating the inner hair cells (IHCs).[2-4] There are also evidences to show that aging also has the same mechanism, with the loss of synaptic connections, which is independent of hair cell loss.[5-7] As the problem persists in individuals who have their thresholds within normal range of <20 dB HL, the term "hidden hearing loss" was coined.[8] The possible cause for such a condition could be many. Findings have shown that both in individuals with noise exposure and in individuals who are aged above 40 years,[9,10] there is a permanent destruction of synapses between the IHCs and type I auditory nerve fibers (ANFs), thus leading to a slow degeneration of the ANFs. However, the hair cells are not affected, leaving the hearing sensitivity normal.[11]

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The classic outlook of sensorineural hearing loss (SNHL) is that the hair cells are the primary targets, and deafferentation or cochlear nerve loss is secondary to hair cell loss, which is the secondary target. However, this outlook toward SNHL has been challenged by many recent studies. One such study was by Kujawa and Liberman.[3] They induced a temporary NIHL of up to 40 dB in guinea pigs and mice; following the recovery from TTS, the auditory system was assessed using distortion product otoacoustic emissions (DPOAEs) and auditory brainstem responses (ABRs). The results indicated permanent damage at a frequency region corresponding to maximum TTS in the afferent nerve ending that is between the IHCs and the ANFs. However, there was no significant effect of deafferentation evident in ABR. Therefore, it is said that the remaining afferent connections that are undamaged would take up the work and help in preserving the threshold within the normal range.[2,3,12]

Further, a study by Furman et al[2] has shown a reduction in the amplitude of wave I after significant exposure to noise at supra-threshold levels (>40 dB SPL), when the same set of animals were assessed both before and after the noise exposure. It was also reported that there is damage to the low spontaneous-rate ANFs (low SR ANFs) and concluded that temporary NIHL further leading to hidden hearing loss affects responses to high levels more than at low levels of sound. Therefore, supra-threshold responses of ABR are demonstrated to have better sensitivity in identifying the damage to the auditory structures. Besides, the responses to DPOAEs were not affected, suggesting normally functioning OHCs.[2,3,12] Further, immunostaining techniques were developed to study the age-graded succession of mice to compare synaptic and the hair cell counts as a measure of cochlear function (ABRs and OAEs).[7] The results obtained in this study were similar to those obtained by Kujawa and Liberman,[3] showing that the afferent connections between the IHC and the ANF were affected. The synaptic ribbon counts near the IHC reduced monotonically with age, but, the hair cell loss was minimal even at a later age.[7]

However, Norena, Tomita, and Eggermont[13] reported that when a cat was exposed to a 76 dB (A) noise for almost 4 months at 24 hours per day, the results showed no changes in ABR thresholds, but the central auditory responses were affected. However, it is not clear what effect long exposure times can have on the central auditory system in individuals with normal audiometric thresholds. In the same vein, Collet et al.[14] observed that otoacoustic emissions (OAE) in humans could be suppressed by contralateral white noise. They also reported that the suppression of OAEs after contralateral auditory stimulation seems to be the only objective and non-invasive method to evaluate the functional integrity of the medial efferent system and the structures lying on its course. In the natural environment, the system could function as a mechanism for "unmasking" biologically significant acoustic stimuli by reducing the response of the cochlea to simultaneous low-level noise.[15] The contralateral suppression of OAEs (CSOAEs) are absent or reduced in cases with auditory dyssynchrony,[16] retrocochlear pathology,[17] and auditory processing disorder.[18] Additionally, an improvement in scores for speech identification in a noisy environment was reported when the efferent system was activated by contralateral noise.[19] Further, these results had a positive correlation with CSOAEs.

In human cochlear tissues, the most direct evidence noted for synaptopathy comes from the assessing the changes in synaptic integrity due to aging,[20] when the number of synapses were counted in 5 temporal bones. Until recently, based on the animal work, it has been hypothesized that few measures of amplitude at supra-threshold levels in ABR are useful in the non-invasive diagnosis of cochlear synaptopathy. In the same vein, a non-invasive test to reveal the initial damage in human ears is important to prevent further damage, as there are very few studies to outline such tests for delineating the changes in the auditory system. ABRs are routinely used in clinics as a test to provide a non-invasive correlate on hearing sensitivity. Moreover, ABR responses to transient stimuli are able to locate hearing deficits along the auditory pathway, as the abnormalities seen at different waveform peaks stem from aggregate responses of the population of neurons at different ascending processing stages.[21] Cochlear synaptopathy predominantly affects supra-threshold processing and has been associated with shallower ABR amplitude versus intensity growth in the presence of normal ABR threshold.[2] For humans, a significant correlation between high-intensity ABR wave I amplitude and noise exposure history was recently reported when the recording was obtained with click stimulus at 90 dB nHL using a mastoid recording electrode. It was reported that ABR wave I amplitudes decreased as a function of noise exposure backgrounds.[22] It is said that low SR ANFs are responsible for perceiving high-intensity sounds, and these fibers are affected in both individuals with noise exposure and in the older age group. Further, a comparison of the effects of synaptopathy at supra-threshold levels between the noise exposure and aged individuals is required, as it has been said that the same mechanisms are affected in both.

Hence, by reviewing the literature, the need to find the effects of both noise exposure and aging on the auditory system is clear, as there is heterogeneity in the results of previous studies. Moreover, a detailed audiological assessment tapping the auditory pathway is necessary to reveal the effects.

## METHODS

The study was carried out to compare DPOAEs, CSOAEs, and ABR in individuals with noise exposure and in aged individuals, with normal hearing. Sixty adult male participants were divided into 3 groups. Group 1 included individuals not exposed to occupational noise, who were <35 years of age ($N=20$) and also served as the control group. Individuals with age ranging between 45 and 60 years, and without any occupational noise exposure, formed group 2 ($N=21$). Group 3 included individuals exposed to noise greater than 80 dB (A) for 8 hours per day in their workplace, aged <35 years ($N=19$). The mean age and age range of the individuals considered for the study is provided in Table 1. The individuals considered for the study had a flat audiometric configuration, that is, a less than 5 dB rise or fall per

**Table 1.** Mean Pure-Tone Average, Mean Age, and Age Range of Participants

| | Number of subjects (*N*) | Age (in years) | |
|---|---|---|---|
| | | Mean | Range |
| Control group (group 1) | 20 | 31.2 | 28-33 |
| Clinical group | | | |
| Aged individuals (group 2) | 21 | 54.5 | 45-65 |
| Noise-exposed individuals (group 3) | 19 | 30.4 | 28-35 |

octave.[21] A written consent was procured from all the subjects before the evaluations.

As a criterion for selection, the hearing threshold of the subjects in each of the groups was within the normal range of <25 dB HL at all 4 octave frequencies (500 Hz, 1000 Hz, 2000 Hz, and 4000 Hz). However, the testing was done from 250 Hz to 8000 Hz, and if the threshold was >25 dB HL at any of the tested frequencies, they were not considered for the study. Participants who presented with any history or presence of middle ear disorders; psychological or neurological dysfunction; the presence of tinnitus; exposed to loud music/use earphones for a longer duration daily were excluded from the study. All the participants were subjected to tests in an acoustically treated room where the ambient noise level was within the permissible limits as specified by ANSI S3.11999 (R 2008).

**Procedure**

As a first step, a detailed case history was taken from all the participants to rule out any pathological conditions of the auditory system and to procure information about their working environment and work experience. All participants were subjected to pure-tone audiometry using Inventis Piano, a dual-channel audiometer coupled to TDH 39 earphones with MX-41/AR ear cushions for octave frequencies between 250 and 8000 Hz to estimate the air conduction threshold, and a bone vibrator (Radio ear B-71) for testing the bone conduction thresholds. The threshold was estimated using the modified Hughson and Westlake procedure[23] in a sound-treated room. The 25 dB HL threshold criteria were fixed in order to rule out any peripheral hearing loss in the participants. The mean pure-tone averages for all the 3 groups are provided in Table 1. Speech recognition thresholds were obtained using Kannada-paired words and Speech Identification Scores using phonetically balanced word lists in the Kannada language.[24] Immittance evaluation, which includes both tympanometry and acoustic reflexes, was done to rule out any middle ear dysfunction. Acoustic reflex using a 226 Hz probe tone at 500 Hz, 1000 Hz, 2000 Hz, and 4000 Hz was assessed using a GSI-Tympstar middle ear analyzer. Individuals who had normal acoustic reflexes at the above-mentioned frequencies were considered for the study. Participants satisfying the selection criteria mentioned above were included for further evaluations.

To measure CSOAEs, transient evoked otoacoustic emissions (TEOAEs) were recorded using ILO V6, Otodynamics OAE equipment. The TEOAEs were recorded at 70 dB SPL using click stimuli. A probe was placed in the external ear canal and was adjusted to obtain a flat spectrum of stimulus across the frequencies. The TEOAE amplitudes were measured using this procedure. The procedure was also repeated using a 30 dB SL contralateral broadband noise (BBN; i.e., threshold noise) delivered through the insert earphone. The amount of suppression induced by contralateral acoustic stimuli was calculated by measuring the difference of TEOAE amplitude with and without contralateral acoustic stimulation. DPOAE fine structure was studied at 8 points per octave to assess the functioning of the outer hair cells. DPOAEs were carried out at different F2 frequencies—500 Hz, 1000 Hz, 2000 Hz, 4000 Hz, and 8000 Hz—with 1.22 as F2/F1 ratio, since it provides optimal DPOAE amplitude. The F1 and F2 primaries were presented at 65 dB SPL and 55 dB SPL respectively, as they provide fewer artifacts and optimum results[25] The DPOAEs were evaluated for the amplitude parameter at various DP frequencies and the signal to noise ratio (SNR) was recorded. The responses were considered to be present if the SNR exceeded 6 dB.[26]

The ABR was measured in a sound-treated room using Biologic Navigator Pro system (Version 7.2.0.). The potentials were obtained with electrodes placed at Fz, M1, and M2; and ground at Fpz position (vertical montage). The electrode impedance considered was below 5 kΩ at all the electrodes. The stimulus was presented through ER-3A insert earphones. The stimulus used for assessment was a click, and the level was decreased in 10 dB steps from 90 dB nHL to 50 dB nHL. The level was not reduced further, because wave I is absent in most of the individuals as it nears the threshold.[27] A repetition rate of 7.1/sec was considered as it provides good representation/morphology of the waveform at lower levels of stimulus presentation.[28,29] A bandpass filter of 100-3000 Hz was used and collected in a 12-millisecond time window. Two thousand sweeps were averaged at each presentation for 2 replications, and the average was taken. The absolute amplitude and absolute peak latencies for wave I, III and V; and wave V/I ratio were analyzed for all the groups at only high levels of presentation (90, 80, and 70 dB nHL), since wave I is not prominent for all at lower levels of presentation. However, at lower levels (60 dB nHL and 50 dB nHL), only wave V latency was analyzed. The analyses of the waveforms were performed for all the participants; the peak identification and morphology rating were done by 2 experienced audiologists in waveform analysis. The ABR measures considered for the analysis were absolute latency, absolute amplitude, and the peak V/I amplitude ratio. The ABR amplitude was measured from peak to baseline. The peaks considered were marked as I, III, and V.

**RESULTS**

The Shapiro–Wilks test of normality was administered to check whether the data followed normal distribution for ABR, DPOAE, and CSOAE measures. It was found that the ABR and CSOAE parameters studied did not follow a normal distribution ($P < .05$) and hence nonparametric tests were administered, whereas the DPOAEs data did follow the normal distribution ($P > .05$) and therefore, parametric tests were administered. The variability is attributed to the heterogeneity among the participants of the study.

**Comparison of Amplitude of DPOAEs Between the Groups**

Descriptive statistics for DPOAEs indicated similar amplitudes at almost all the frequencies tested in all the groups. Multivariate Analysis of Variance (MANOVA) test revealed a significant difference among the 3 groups compared at only 3 frequencies, which were 4358 Hz, 6165 Hz, and 6726 Hz. It was observed that the amplitude of DPOAEs for group 2 and group 3 was reduced in comparison to group 1; however, there was no significant difference observed between the groups. A post-hoc analysis was carried out using Duncan's test for DPOAEs amplitude for the frequencies that showed a significant difference in MANOVA. Among all the frequencies that showed a significant difference, that is, at 4358 Hz, 6165 Hz, and 6726 Hz, there was no significant difference found between group 2 and group 3 ($P > .05$), whereas a significant difference was present for group 1 when compared with groups 2 and 3 ($P < .05$). In summary, the DPOAEs amplitude showed a significant difference only at 4358 Hz, 6165 Hz, and 6726 Hz, and no significant difference at the other frequencies tested.

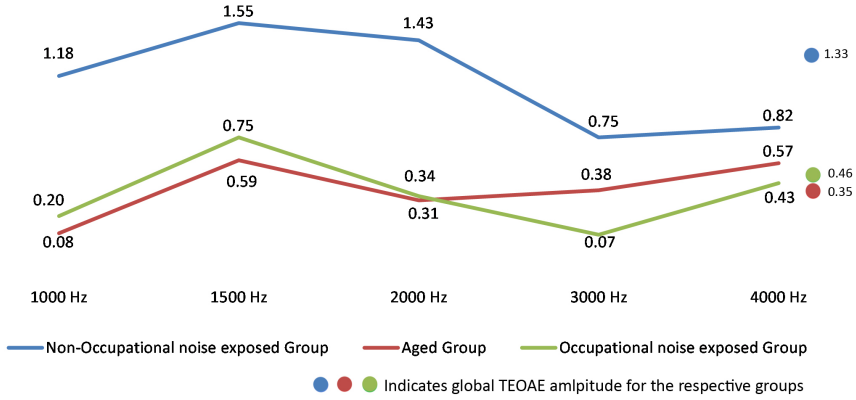


**Figure 1.** Representation of median average frequency-specific and global amplitude for contralateral suppression of OAEs across the 3 groups.

### Comparison of CSOAEs Between Groups

The results showed decreased mean values of contralateral suppression of TEOAEs across all frequencies in groups 2 and 3 when compared to group 1. The median amplitude of CSOAEs at different frequencies along with the global amplitude is provided in Figure 1. The Kruskal–Wallis Test revealed a significant difference among the 3 groups compared for the amount of suppression. It was observed that the suppression for group 2 and group 3 was reduced in the comparison to group 1. The test results are depicted in Table 2. In summary, the CSOAEs showed a significant difference at all the tested frequencies, when group 1 was compared with groups 2 and 3.

Further, the Mann–Whitney *U*-test was administered to check for the difference between 2 independent groups for the parameters that showed a significant difference in the Kruskal–Wallis test. It was observed that there was a difference seen between groups 1 and 2; and groups 1 and 3 for global amplitude. However, there was no significant difference observed when individual frequencies were considered. Also, between group 2 and group 3, there was no significant difference seen among any of the frequencies as well as for global amplitude. The Mann–Whitney *U*-test results are depicted in Table 2.

### Comparison of ABR Latency Between the Groups

The descriptive statistics of latency parameters indicate that there was an increase in the mean latency of different ABR waves including I, III, and V at all the tested intensity levels for both group 2 and group 3 when compared with group 1. It was observed that the prolongation of waves I, III and V were slightly more in group 2 compared to group 3, when mean latencies were analyzed using descriptive statistics. The averaged waveform across the 3 groups at different intensities is represented in Figure 2. The Kruskal–Wallis Test was administered to compare the 3 independent groups for latency parameters at different intensities. The test indicated a significant effect, $\chi^2(2) = 18.79$, $P = .00$ at 90 Db nHL for wave I; $\chi^2(2) = 10.06$, $P = .01$ at 60 Db nHL for wave V; $\chi^2(2) = 13.75$, $P = .01$ and $\chi^2(2) = 7.52$, $P = .01$ at 50 Db nHL for wave V and wave III respectively.

**Table 2.** The MANOVA Test Results for Frequencies that Exhibited a Significant Difference in Group 1, Group 2, and Group 3

| Frequency (Hz) | *F* Value |
|---|---|
| 4358 | $F_{2.48} = 9.97$, $P < .05$ |
| 6165 | $F_{2.48} = 6.13$, $P < .05$ |
| 6726 | $F_{2.48} = 4.99$, $P < .05$ |

Further, the Mann–Whitney *U*-test was administered to check for the difference between 2 independent groups, for which parameters that showed a significant difference in Kruskal–Wallis test were analyzed. It was observed that there was a significant difference in latency, at 50 Db nHL for wave V and wave III respectively, between group 1 and group 2, whereas the difference, $|Z| = 3.57$, $P = .03$ was present only for latency of wave I at 90 dB nHL, between group 1 and group 3. When group 2 and group 3 were compared, the difference was evident only for wave V, $|Z| = 2.01$, $P = .04$ at 50 dB nHL; and $|Z| = 2.67$, $P = .04$ at 60 dB nHL for the latency parameter. The wave I latency at 50 dB nHL and 60 dB nHL was excluded from the statistical analyses, since the number of subjects who demonstrated a response at that intensity were very few ($N < 3$ in each group). Hence, it is difficult to compare wave I latency measures between groups at such low intensity levels, even though it is considered as a supra-threshold level.

### Comparison of Absolute Amplitude of ABR Waves and Wave V/I Ratio Between the Groups

The descriptive statistics of amplitude parameters indicate that there was a decrease in the mean amplitude of different ABR waves including I, III, and V; and an increase in wave V/I amplitude ratio at most of the tested intensity levels for both group 2 and group 3 when compared with group 1. It was observed that the reduction in amplitude of waves I, III, and V was more in group 2 compared to group 3, when mean amplitudes of different waves were analyzed using descriptive statistics. It was also noted that the mean amplitude of wave V for group 3 was similar to group 1, especially at higher stimulation levels of 90, 80, and 70 dB nHL. In other words, a more pronounced difference was seen for wave I amplitude when compared to wave III and wave V amplitudes. The averaged waveform across the 3 groups at different intensities is represented in Figure 2.

The Kruskal–Wallis test was administered to compare the 3 independent groups for amplitude parameters at different intensities. This test indicated a significant effect for wave I amplitude and wave V/I amplitude ratio at higher intensity levels. The number of subjects who exhibited wave I response and wave V/I amplitude ratio were very low ($N < 3$ in each group) at 50 dB nHL and 60 dB nHL and hence, were excluded from the statistical analyses. However, the difference was significant at only very few selected intensities for wave III and V, which did not follow any trend. Further, the Mann–Whitney *U*-test was administered to check for the difference between 2 independent

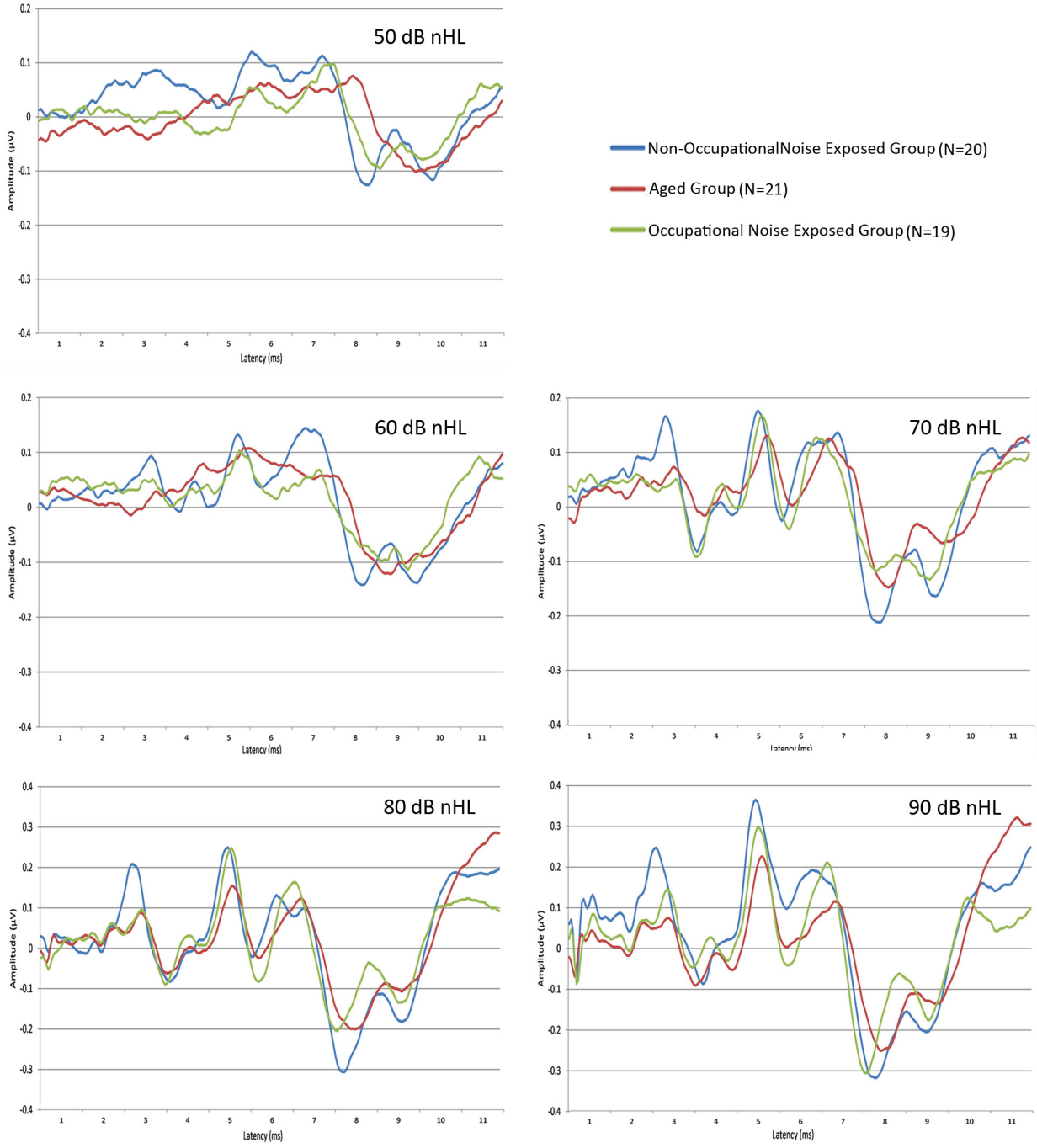


**Figure 2.** Averaged waveforms of the 3 groups at 90, 80, 70, 60, and 50 dB nHL respectively.

groups for the parameters that showed a significant difference in the Kruskal–Wallis test. It was observed that there was a difference seen between groups 1 and 2, and groups 1 and 3 in most of the parameters that exhibited a significant difference in the Kruskal–Wallis test. However, there was no significant difference observed between group 2 and group 3.

To summarize the findings obtained, there was a significant difference observed when the comparisons were made between group 1 and group 2; and group 1 and group 3, in terms of ABR parameters. The differences were evident for wave I and wave V/I amplitude ratio at supra-threshold levels. Also, there was a significant difference in CSOAEs for the group mentioned above in the comparisons, because the amount of suppression was reduced in group 2 and group 3 when compared to group 1. However, DPOAEs did not exhibit any significant difference at any of the frequencies tested.

## DISCUSSION

The results of this indicated a significant difference observed in the amplitude parameters for wave I and wave V/I ratio compared to the amplitude of other waves, and no significant difference in terms of latency parameters was noted. Moreover, the difference was not evident for the amplitude of DPOAEs between the groups compared. However, it was observed that there was reduced suppression in aged individuals and those who were exposed to occupational noise, compared to the control group.

The responses of ABR showed a clear increase in latency of wave I, III, and V with a decrease in intensity level, similar to a previous report.[30] Although the latencies of group 2 and 3 were slightly higher compared to group 1, there was no significant difference observed between the groups. Literature has shown that approximately 40% of the auditory nerve cochlear synapses could be destroyed permanently without any permanent threshold elevation for the ABR, which is reflected by the summed activity of the auditory nerve fibers in its first wave.[31] It is clear from the present study that there was no significant difference observed for the latency parameter in all the intensities tested. This could be because there is a reduction in the number of fibers firing which is evident as reduced wave I amplitude and not with the speed of transmission of the signal which is characterized by the latency.

The wave I amplitude at higher intensities to click stimuli were significantly smaller in ears with noise exposure and aging when compared to the normals of age <35 years without occupational noise exposure, referred to as normals from here on. At higher testing levels (>70 dB nHL), there was a systematic trend for wave I amplitude to decrease in aged and noise-exposed individuals. This trend of reduced amplitude was not well established at lower intensity levels. A possible reason could be that even in individuals with a normal hearing without occupational noise exposure, the presence of wave I and III reduces at lower intensity levels and hence it is difficult to use this as an indicator at low intensities. In contrast to the results obtained for wave I, there was no decrement in the wave V amplitude at supra-threshold levels. Similar results were obtained by Stamper and Johnson (2015),[22] where the supra-threshold wave I amplitude was reduced in non-occupational noise-exposed group when compared to occupational noise-exposed group. It was stated by Furman et al (2013)[2] that the neural degeneration is to begin with loss of synapses on the IHCs especially at the basal half of the cochlea, and later, as a loss of central projections of spiral ganglion cells. Therefore, a decrement of amplitude is observed in wave I and not in wave V. The results of the present study support the idea that noise-induced synaptopathy is selective to low SR fibers, which is indicated by a reduction in amplitude at higher compared to lower intensities. The low SR ANFs with high thresholds are more defiant to masking,[31, 32] therefore the virtual contribution to the entire neural responses would increase with noise. There could be several possible reasons for the reduction in wave I amplitude and not in wave V. It is an established fact that wave I originates from the distal portion of the auditory nerve, [21] while the auditory midbrain would act as the generator of wave V.[33, 34] Due to dissimilar sites of generation, the auditory nerve and the auditory midbrain have a mechanism that might compensate for the decline in output from the auditory nerve. The hyperactivity in the central auditory pathways was observed in mice with synaptic loss induced by noise exposure.[35]

Studies on tinnitus also support this idea. Schaette and McAlpine (2011)[8] did a study in individuals with tinnitus and found that the ABR wave I amplitude was reduced compared to the non-tinnitus group; however, wave V did not exhibit any difference between the groups. The authors concluded the existence of a homeostatic gain control mechanism in which there is an overshoot in the spontaneous firing rate of neurons at the inferior colliculus in the animals subjected to noise exposure. Don and Eggermont (1978)[36] came up with another explanation for the distinctive results of wave I and V amplitudes. A high pass masker was used to find the various frequencies contributing to the generation of waves using clicks. The authors suggested that the generation of wave I is mainly contributed by neurons with characteristic frequencies above 2000 Hz, whereas the entire cochlear partition contributes to the generation of wave V. Hence, a damage to structures encoding higher frequency regions (3000 Hz-6000 Hz) would lead to reduced wave I amplitude as the number of contributing neurons are less at this frequency. However, this is not the case for wave V amplitude, as the structures responsible for encoding lower frequencies remain unaffected, and hence would be unaltered even if the higher frequency neurons are compromised.[36] These results also explain why ABR wave V/I amplitude ratio is increased in aged and noised-exposed ears.

The DPOAEs amplitude was found to be similar in all the 3 groups examined. There was a significant difference seen only at 3 frequencies (4358 Hz, 6165 Hz, and 6726 Hz) which did not follow any trend. The possible reason for this could be due to damage in the high-frequency region, which is in parallel with a decrease in ABR wave I amplitude that arises from the higher frequency. Hence, a difference at 4358 Hz, 6165 Hz, and 6726 Hz in DPOAEs and for ABR wave I amplitude indicates that the damage at the level of cochlea could be succeeding the damage at the synaptic level.

Contralateral suppression of TEOAEs was observed more in non-exposed individuals compared to aged and noise-exposed individuals. Amplitudes of TEOAEs were also reduced in noise-exposed and aged individuals compared to non-exposed individuals. This could be attributed to an efferent auditory system damaged due to occupational noise exposure which in turn failed to suppress. Kotylo (2002)[37] reported reduced suppression of OAEs in individuals with occupational exposure to noise. Prasher et al (1994)[17] reported a significant reduction in contralateral suppression of TEOAEs and suggested that contralateral sound-activated efferent suppression may provide an early indication of auditory damage after exposure to noise. The current findings also suggest that the physiological test is a reliable measure for the early detection of central dysfunction due to noise exposure. Therefore, it is difficult to diagnose an individual's damage due to aging and noise exposure at an earlier stage by using DPOAEs as a measure.

We can infer from the present study that the reduction in ABR wave I amplitude and the increment in wave V/I amplitude ratio (which is due to the lessening of wave I amplitude and not because of wave V

**Table 3.** Test Values Having Significant Differences in the Kruskal–Wallis Test and Mann–Whitney *U*-Test Between the 3 Groups for CSOAEs

| Frequency | Kruskal–Wallis H test | Mann–Whitney *U*-test | |
|---|---|---|---|
| | | Group 1 & Group 2 | Group 1 & Group 3 |
| 1 kHz | $\chi^2(2) = 6.35$ $P = .00^*$ | $\|Z\| = 2.79$ $P = .04^*$ | $\|Z\| = 1.66$ $P = .96$ |
| 1.5 kHz | $\chi^2(2) = 5.22$ $P = .00^*$ | $\|Z\| = 2.18$ $P = .29$ | $\|Z\| = 1.44$ $P = .15$ |
| 2 kHz | $\chi^2(2) = 5.20$ $P = .00^*$ | $\|Z\| = 3.56$ $P = .00^*$ | $\|Z\| = 2.77$ $P = .00^*$ |
| 3 kHz | $\chi^2(2) = 5.55$ $P = .00^*$ | $\|Z\| = 0.94$ $P = .34$ | $\|Z\| = 2.05$ $P = .40$ |
| 4 kHz | $\chi^2(2) = 2.67$ $P = .00^*$ | $\|Z\| = 0.32$ $P = .74$ | $\|Z\| = 0.64$ $P = .51$ |
| Global | $\chi^2(2) = 11.63$ $P = .00^*$ | $\|Z\| = 3.73$ $P = .00^*$ | $\|Z\| = 2.96$ $P = .00^*$ |

*indicates a significant difference
*kHz: Kilohertz

changes), along with CSOAEs could act as first-level clinical indicators when compared to DPOAEs alone, suggesting that prior to hair cell damage, there is a damage at the synaptic level.

## CONCLUSION

Regular exposure of cochlear amplifiers to high-level noise, and aging-related changes in humans may lead to irreversible damage in hearing. The role of the efferent system presumably is to enhance signals in the presence of noise, and an ideal test used for identifying the shifts observed in cochlear functioning would be OAEs. OAEs are preferred over pure-tone audiometry for early identification of NIHL because they are sensitive to minor damage to outer hair cells and also can be monitored easily due to their objectivity and speed. However, in early stages, there may not be any evident threshold shift even in the presence of underlying efferent system damage, seen in OAEs. CSOAEs are more preferred as they tap the efferent auditory pathway more robustly. Previous studies have reported neural degeneration in ears with noise-induced threshold shifts and aging, suggesting that normal hearing thresholds can be accompanied by impaired function of efferent fibers that project from the brainstem to the cochlea. Hence, assessment at the brainstem level provides valuable information on early identification of such conditions.

It is noted that clinically relevant hearing loss may not serve as the standard diagnosis in identifying hidden hearing loss. As this study included individuals in both low-risk (without any significant noise exposure) and high-risk (with occupational noise exposure) groups, the differences were evident in the results. However, the study has to be performed with a larger group for generalization and usage in routine clinical evaluations.

**Ethics Committee Approval:** All procedures performed in the study involving humans were in accordance with the ethical standards of the institutional review board**.**

**Informed Consent:** Prior to the evaluation, informed consent was obtained from the participants after briefing them about the study.

**Peer Review:** Externally peer-reviewed.

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