

Narrow-band chirp and tone burst auditory brainstem response as an early indicator of synaptopathy in industrial workers exposed to occupational noise

Kondli Nagaraj Megha, Koratagere Narayanaswamy Divyashree, Aishwarya Lakshmi, Sugathan Adithya, Kunnupurath Puthenveedu Keerthana, Zeena Venkatcheluviah Pushpalatha, Sreeraj Konadath*

All India Institute of Speech and Hearing, Manasagangothri, Mysuru, India.

Summary

This study aims at characterizing and comparing the findings of auditory brainstem response (ABR) using narrow-band chirp (NB-chirp) and tone burst (TB) for both latency and amplitude parameters among those exposed to occupational noise and to determine which among the two serves as a better indicator of noise-induced cochlear neuropathy. Forty adult males in the age range of 20-35 years were considered, wherein 20 of them were exposed to noise > 80 dB (A) for 8 hours per day constituting Noise-exposed group; and Control group consisted of 20 individuals without occupational noise exposure. ABR was recorded using NB-chirp and TB for four frequencies at 80 dB nHL through Etymotic Research – 3A (ER-3A) Insert phones using Interacoustics Eclipse EP-25 in individuals with and without noise exposure. MANOVA was performed to compare between TB ABR and NB-chirp ABR between the two groups. Statistical analysis revealed a notable difference for NB-chirp comparisons between the two groups at three frequencies: 500 Hz, $F(1, 38) = 10.6$; 1000 Hz, $F(1, 38) = 7.91$; and 2000 Hz, $F(1, 38) = 6.64$. Whereas, the difference was evident at only 500 Hz: $F(1, 38) = 4.98$ in case of TB ABR. However, there was no significant difference seen at any of the frequencies for amplitude parameters in both TB and NB-chirp ABR. Latency of wave V using NB-chirp was considered to be a better indicator compared to TB, acting as a better clinical tool in early identification, diagnosis, and monitoring of noise induced hearing loss (NIHL).

Keywords: Auditory brainstem response, narrow band-chirp, tone burst, noise induced hearing loss, cochlear synaptopathy

1. Introduction

We all experience sound in our environment through different sources which are at safe levels of hearing and do not cause any discomfort. But, there are sounds, which are loud enough to cause damage to the hearing structures and hence cause hearing loss depending on

the exposure duration (I). The effects of noise-induced hearing loss (NIHL) include auditory and non-auditory effects. The two sets of auditory effects include the effects that are noticeable after a certain duration of exposure to noise and the effects that are seen during the course of noise exposure. The after effects of noise can lead to temporary or permanent hearing loss, which arises due to damage in peripheral or higher auditory centers.

Chronic exposure to noise in industrial workers that affect bilateral cochlea causes high-frequency sensorineural hearing loss (SNHL) with 4000 Hz notch (2). Further, they observed that around 39% of industrial workers who were exposed to noise levels > 87.3 dB (A), for 8-12 hours per day suffered from SNHL (3).

Released online in J-STAGE as advance publication August 20, 2019, 2019.

*Address correspondence to:

Dr. Sreeraj Konadath, Assistant professor in Audiology, All India Institute of Speech and Hearing, Manasagangothri, Mysuru 570006, India.
E-mail: sreerajkonadath@aiishmysore.in

Regular exposure of cochlear amplifiers to high-level noise may yield irreversible damage to them (3). An ideal test used for identifying the shifts observed in cochlear functioning would be otoacoustic emissions (OAEs). OAEs are preferred over pure tone audiometry for early identification of NIHL because, they are sensitive to minor damage and also can be monitored easily due to their objectivity and speed (4). However, in early stages, there may not be any evident threshold shifts even in the presence of underlying efferent system damage. There is evidence from animal as well as human studies which suggest that, even moderate exposure to acoustic stimulus, which can cause temporary threshold shift can destroy the connections between the auditory nerve fibers (ANFs) and cochlear hair cells causing synaptopathy (5-7). This type of damage to the synapse, which causes no permanent threshold elevation, is termed hidden hearing loss (8). It was reported that neural degeneration in ears with noise-induced threshold shifts in mice subjected to mild acoustic trauma suggested that normal hearing thresholds can be accompanied by impaired function of efferent fibers that project from the brainstem to the cochlea (5). Hence, assessment at the brainstem level provides valuable information on early identification of NIHL. There is also reduction seen in compound action potentials and spontaneous neural activity induced by noise exposure in electrophysiological tests (9).

Auditory brainstem response (ABR) is an electrophysiological test to measure the functional integrity of brainstem auditory structures (10). ABR is considered to be a valuable tool in evaluating auditory functioning, including difficult to test populations. The brainstem auditory evoked potential or short latency potential represents a series of neuro-electric potentials recorded from electrodes placed on the scalp. In order to assess different frequency regions within the cochlea, various stimuli have been employed in ABR measurements such as click, tone burst (TB) and speech stimuli. Out of these stimuli types, a brief tonal stimulus gives a better representation (11). Chirp stimuli are brief tonal stimuli designed to compensate for the delay in time for basilar membrane travelling wave in order to improve the temporal synchrony between the neural elements that usually are asynchronously activated by a brief stimulus such as a click (12). It is said that such compensation yields higher temporal synchronization of the neural structures that contribute to elicitate ABR, and also produces extremely large response amplitudes (12). A study done on individuals with and without noise exposure using click and Claus Elberling chirp stimuli (CE-chirp) indicated that there was a significant delay in latency as well as reduced amplitude in individuals exposed to noise. However, this was not seen when clicks were used. Hence, it was concluded that responses obtained with CE-chirp stimuli is an effective tool in identifying the early

pathological changes caused due to occupational noise exposure when compared to click-evoked ABR (13). NB-chirps are constructed with an octave bandwidth wherein there is super position of four one-octave-wide chirps centered at 500 Hz, 1000 Hz, 2000 Hz, and 4000Hz which are capable of improving the neural synchronization and also provide frequency-specific information (12,14). A complete evaluation should contain frequency-specific information because it provides better detail about the configuration of the hearing loss. TB is a short-time signal consisting of a single tone, which is utilized for testing, measurement, and/or calibration. TB is a spectrally narrow stimulus. Studies have shown that as the frequency increases, the peak V latency decreases (12). The responses from these stimuli are also frequency specific with consideration that, with the use of frequency specific stimuli like TB and NB-chirps, the spectral splatter is reduced to some extent and reduces the participation of other regions of the cochlea. According to the literature, frequency specific TB can be a better predictor of pure tone thresholds than click evoked ABR (15,16). Also, since NB-chirp is constructed with an octave bandwidth centered at 500 Hz, 1000 Hz, 2000 Hz, and 4000Hz, it would also yield frequency-specific information (12-14).

The aim of the present study is to show the effectiveness of two different frequency specific stimuli available namely, NB-chirp and TB in ABR, and to show which of the two are ideal for ascertaining the auditory system changes that arise due to NIHL, with normal peripheral hearing sensitivity, in turn helping early identification of cochlear neuropathy resulting from NIHL. Two groups consisting of individuals with and without noise exposure were involved and the effect of two different stimuli, NB-chirp and TB on the auditory system were compared in this study. Between-group comparisons were made wherein, the same stimuli were compared across two groups, that is, TB of Control group was compared with TB of Noise-exposed group and the same for NB-chirp ABR.

2. Materials and Methods

2.1. Participants

Forty adult male participants were selected randomly from a single work place and were divided into two groups of twenty individuals each. "Control group" included individuals who were not exposed to occupational noise (age range = 20 to 35years, mean = 23.5 years) and the "Noise-exposed group" included individuals who were exposed to noise greater than 80 dB(A) [mean = 87.5 dB(A)] for a duration of 8 hours per day in their workplace (age range = 20 to 35years, mean = 27.75 years) for a minimum time period of 3 years (range = 3 to 5.6 years). The noise measurement

at the working place was performed using a calibrated SLM (B & K model 2270) with windshield for a duration of 5 minutes at each site. The microphone was placed at the ear level within a diameter of 1 meter. For measuring the amount of noise exposure, the tripod stand with the microphone was placed behind the individual's ear with approximately 180° azimuth within a distance of 1 meter. The subjects considered were non-smokers and non-alcoholics. None of them were under any medications for other ailments or using any type of hearing protective devices. Not all participants expressed difficulties during communication. However, the major communication disability reported by these individuals was difficulty in listening in the presence of background noise and difficulty in talking over the phone. Each subject gave written informed consent at the outset.

2.2. 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, work experience and listening difficulties faced by them. All participants from control group and noise-exposed group were subjected to pure tone audiometry, immittance and ABR. Pure tone audiometry for octave frequencies between 250 to 8000 Hz were tested using a dual channel diagnostic audiometer (calibrated as per ANSI S3.6, 1996). Only those participants whose hearing sensitivity was < 20 dB HL at each frequency in the aforementioned frequency range (in both groups) without any otologic, psychological or neurological dysfunction were selected for the study. The 20 dB HL threshold criteria were fixed in order to rule out any peripheral hearing loss in the participants. The mean pure tone average at four frequencies (500Hz, 1000Hz, 2000Hz and 4000Hz) was 8.50 dB for control group and 9.25 dB for noise-exposed group participants. Speech recognition thresholds were obtained using Kannada paired words and Speech Identification Scores (SIS) using Phonetically Balanced (PB) word lists in Kannada language (17). The mean SIS scores were 7.25 for control group and 7.75 for the occupational noise exposed group. Immittance evaluation, which includes both tympanometry and acoustic reflexes was done to rule out any middle ear dysfunction.

The participants were also tested with distortion product otoacoustic emissions (DPOAEs) at 8-points per octave from 1000Hz to 8000Hz at 80 dB to assess the outer hair cell (OHC) functioning. They were recorded with a frequency ratio of 1.22 for the primary tones and the level of f2 primary was kept 10 dB less than f1 level. The ABR assessment was carried out in a sound treated room using the Interacoustics Eclipse EP-25 system. The electrical potentials were obtained

with electrodes placed at Fz, M1, M2; and ground at Fpz position. The measured potentials were recorded with impedance below 5kΩ at all electrodes and the stimulus was presented through ER-3A insert phones. The assessment was done with two stimuli namely, narrowband-chirp (NB-chirp), and TB of 2-0-2 cycle at four different frequencies 500 Hz, 1000 Hz, 2000 Hz and 4000 Hz. The stimulus level was kept constant at 80 dB nHL at a repetition rate of 11.1/sec. A band pass filter of 100-3000 Hz was used and the data were collected in a 12 ms time window for NB-chirp and 14 ms for TB. One thousand five hundred sweeps were averaged at each presentation for two replications and the average was taken. The absolute amplitude and absolute peak latencies were recorded for peak V in all four frequencies between the groups. The peaks were marked by two experienced audiologists for reliable measures. The data analysis was done using SPSS, software version 21 for 40 ears. Shapiro-Wilk's test for normality was administered. The amplitude of DPOAEs followed a non-normal distribution and hence, a non-parametric test was administered. For ABR, the latency parameter was observed to be within normal distribution, and hence a parametric test was administered. Whereas, a non-parametric test was administered for amplitude parameters because the data did not follow normal distribution. $p < 0.05$ was used to verify the level of significance during statistical analysis.

3. Results

3.1. Comparison of DPOAEs amplitude between Control group and Noise-exposed group

Because the data followed non-normal distribution, Mann-Whitney U test was administered. DPOAEs were present for both groups wherein, the criteria of amplitude greater than 6 dB were considered for OAEs to be present. It was evident that the DPOAEs of the participants from both groups had no significant difference across most of the frequencies. There was a significant difference observed only at frequencies of 1001Hz with $Z = -2.33$, $p < 0.05$, and $r' = 0.03$; 3369 Hz with $Z = -2.08$, $p < 0.05$, and $r' = 0.03$; and 7336 Hz with $Z = -2.09$, $p < 0.05$, and $r' = 0.03$. However, there was no trend observed in amplitude difference across frequencies between the two groups. The median amplitude of DPOAEs across frequencies between control group and noise-exposed group is presented in Figure 1.

3.2. Comparison of the absolute latency and amplitude of peak V between Control group and Noise-exposed group for TB ABR

Latency Comparisons: Descriptive statistics were

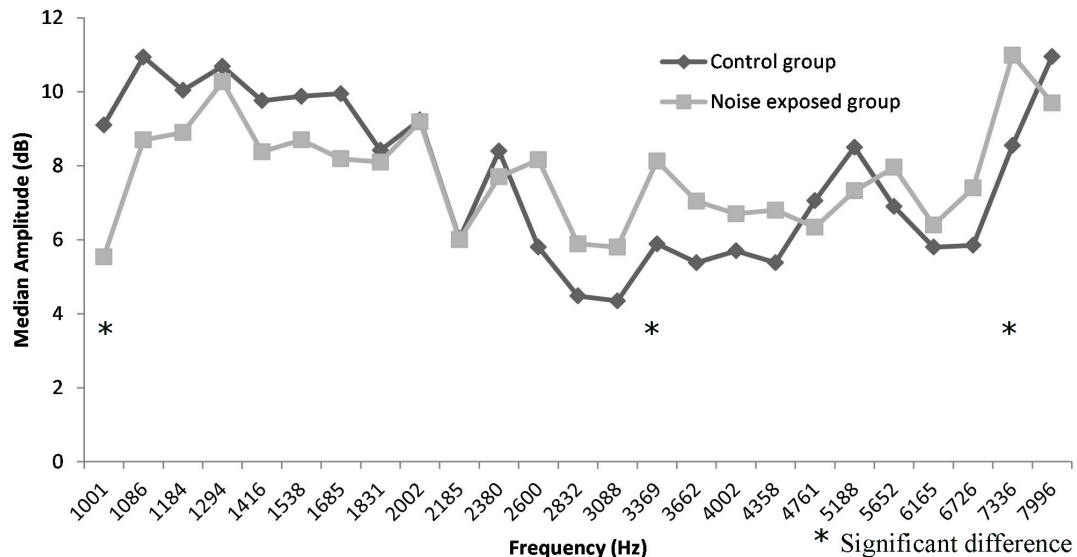


Figure 1. Median amplitude of DPOAEs in Control group and Noise-exposed group across different frequencies. DPOAEs, distortion product otoacoustic emissions.

carried out to find the mean and standard deviation of peak V between control group and noise-exposed group for TB. The mean latency for 500 Hz was found to be 7.92 ms (SD = 0.78) for Control group and 7.45 ms (SD = 0.53) for Noise-exposed group; at 1000 Hz the mean latency was 7.03 ms (SD = 0.65) for Control group and 6.88 ms (SD = 0.38) for Noise-exposed group; at 2000 Hz the mean latency was 6.36 ms (SD = 0.65) for Control group and 6.18 ms (SD = 0.24) for Noise-exposed group; and at 4000 Hz the mean latency was found to be 5.77 ms (SD = 0.22) for Control group and 5.85 ms (SD = 0.25) for Noise-exposed group. To compare the absolute latency of peak V for TB ABR between Control group and Noise-exposed group, MANOVA was administered. A statistically significant difference ($p < 0.05$) was exhibited at only 500 Hz, $F(1, 38) = 4.98$, $\eta^2_p = 0.17$. But, there was no statistically significant difference ($p > 0.05$) evident at 1000 Hz, $F(1, 38) = 0.78$, $\eta^2_p = 0.02$; 2000 Hz, $F(1, 38) = 1.28$, $\eta^2_p = 0.33$; and 4000 Hz, $F(1, 38) = 0.97$, $\eta^2_p = 0.03$.

Amplitude Comparisons: The median amplitude for TB ABR at 500 Hz was found to be 0.27 μV for both control group and noise-exposed group; at 1000 Hz the median amplitude was 0.19 μV for Control group and 0.25 μV for Noise-exposed group; at 2000 Hz the amplitude was 0.22 μV for Control group and 0.21 μV for Noise-exposed group; and at 4000 Hz the amplitude was found to be 0.2 μV for Control group and 0.19 μV for Noise-exposed group. Mann-Whitney test was carried out to compare the absolute amplitude of peak V for TB between the groups. There was no statistically significant difference ($p > 0.05$) observed at all four frequencies.

3.3. Comparison of the absolute and the absolute latency of peak V between Control group and Noise-exposed group for NB-chirp ABR

Latency Comparisons: Descriptive statistics was carried out to find the mean and standard deviation of peak V between control group and noise-exposed group for NB-chirp. The mean latency for 500 Hz was found to be 2.45 ms (SD = 0.68) for Control group and 3.13 ms (SD = 0.63) for Noise-exposed group; at 1000 Hz the mean latency was 3.49 ms (SD = 0.72) for Control group and 4.06 ms (SD = 0.53) for Noise-exposed group; at 2000 Hz the mean latency was 4.54 ms (SD = 0.59) for Control group and 4.99 ms (SD = 0.51) for Noise-exposed group; and at 4000 Hz the mean latency was found to be 5.38 ms (SD = 0.32) for Control group and 5.68 ms (SD = 0.62) for Noise-exposed group. To compare the absolute latency of peak V for NB ABR between Control group and Noise-exposed group, MANOVA was administered. A statistically significant difference was exhibited at 500 Hz, $F(1, 38) = 10.61$, $\eta^2_p = 0.21$; 1000 Hz, $F(1, 38) = 7.91$, $\eta^2_p = 0.17$; and 2000 Hz, $F(1, 38) = 6.64$, $\eta^2_p = 0.14$. However, there was no statistically significant difference at 4000 Hz, wherein, $F(1, 38) = 3.5$, $\eta^2_p = 0.08$.

Amplitude Comparisons: The median amplitude for NB-chirp at 500 Hz was found to be 0.12 μV for Control group and 0.19 μV for Noise-exposed group; at 1000 Hz the median amplitude was 0.07 μV for both Control group and Noise-exposed group; at 2000 Hz the amplitude was 0.05 μV for Control group and 0.07 μV for Noise-exposed group; and at 4000 Hz the amplitude was found to be 0.09 μV for Control group and 0.11 μV for Noise-exposed group. Mann-Whitney test was carried out to compare the absolute amplitude of peak V for NB-chirp between the groups. There was no statistically significant difference ($p > 0.05$) observed at all four frequencies.

The overall results indicate that, there was a significant difference observed for latency parameter at only 500Hz for TB ABR, whereas, the difference was

evident at 500Hz, 1000Hz and 2000Hz for NB-chirp ABR. However, there was no significant difference seen at any of the frequencies for amplitude parameter in both TB and NB-chirp ABR. The graphs representing the comparisons are shown in Figure 2 and Figure 3 for latency and amplitude parameters respectively.

4. Discussion

The study aimed to compare the absolute peak latency and absolute amplitude using TB and NB-chirp, across two groups: Control group (without occupational noise exposure) and Noise-exposed group (with occupational noise exposure). Results indicated characteristic

differences in NB-chirp between the two groups in ABR recordings. Cochlear synaptopathy is a condition where there is no evident loss in the hair cells but an irreversible loss of synapses between the inner hair cells (IHCs) and the ANFs is seen (5). OHC dysfunctioning leads to a loss of sensitivity and a reduction in frequency selectivity. When assessed audiometrically in quiet, the thresholds are in normal limits as the OHCs are intact in spite of up to 80% of synaptic loss corresponding to IHC damage (18). Hence, the presence of DPOAEs in both the groups showing no significant difference in the amplitude indicates damage at the IHC or at the synaptic level. To assess the functioning of IHC/auditory nerve synapse ABR would be a better tool, and more particularly a frequency specific stimuli would tell us the functioning characteristics at different regions. For the TB stimulus, latency values decreased as frequency increased which followed the expected trend. Supported by several studies, which have shown that at lower frequencies, the latencies are prolonged due to responses that arise from the apical region of the cochlea (19-22). For NB-chirp stimulus, the ABR latency values decreased with decrease in frequency. This pattern was the opposite of that which occurred for the TB stimulus (23,24). This can be explained by the fact that, there are shorter response latencies in the NB-chirp ABR due to the temporal references [0 ms] (24). The arrival time at the eardrum is the 8000 Hz component, which compensates for frequency delay characteristics of the basilar membrane (22). The NB-chirp 500 Hz, 1000 Hz, 2000 Hz and 4000 Hz can be considered as a subset of the chirp evoked ABR, thus

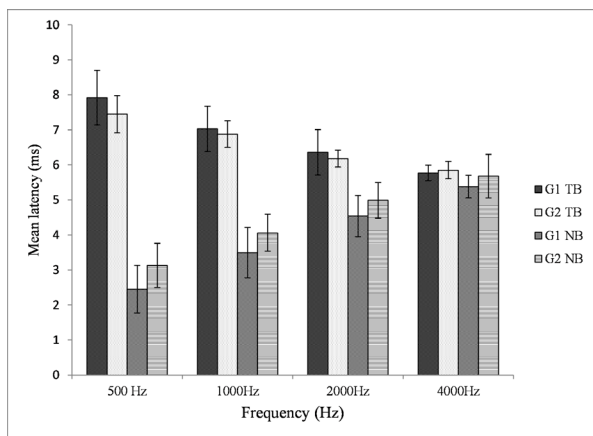


Figure 2. Mean latency (ms) of peak V for TB and NB-chirp ABR in Control group and Noise-exposed group across different frequencies. TB, tone burst; NB-chirp, narrow-band chirp; ABR, auditory brainstem response.

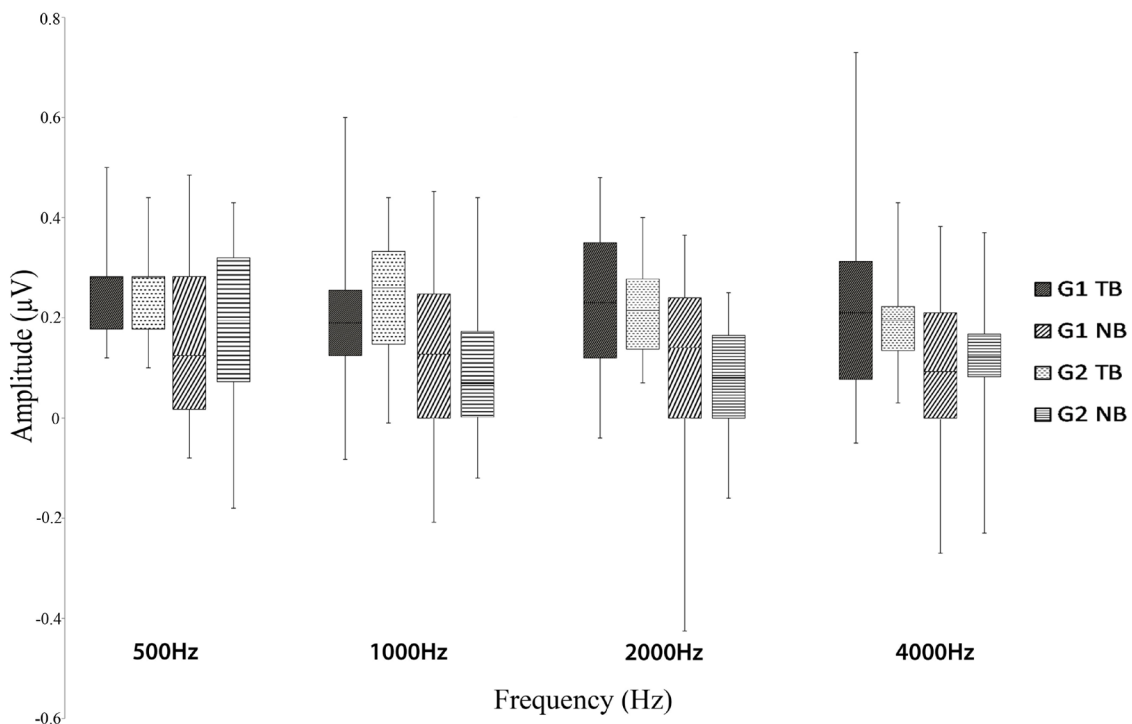


Figure 3. Amplitude (µV) of peak V for TB and NB-chirp ABR in Control group and Noise-exposed group across. TB, tone burst; NB-chirp, narrow-band chirp; ABR, auditory brainstem response.

exhibiting shorter latencies compared to TBs for low frequency stimuli. Also, because NB-chirp provides maximum stimulation to the cochlea, the delay in the cochlear travelling wave will be compensated by attaining different timing across each of the frequencies (24,25). That is, the regions centered at 500 Hz and 1000 Hz receives the stimulus earlier, giving shorter peak latency. This trend of latency change with frequency of the stimuli was consistently observed in both groups.

In the current study, there was a significant difference observed in terms of latency at 500Hz, 1000Hz, and 2000Hz for NB-chirp stimuli. But, there was no significant difference observed between the two groups at 4000Hz, although the latency was prolonged in the noise exposed group. A cochlear histopathological study conducted on mice by Sergeyanko, Lall, Liberman, & Kujawa in 2013 (26), revealed similar results *i.e.* IHC ribbon losses were initially greater in the apical region when compared to base, but with increasing age, the synaptopathy spread throughout the cochlear spiral, whereas, OHC ribbons were well preserved, which approximated to < 10% of loss across all frequencies. The reason for the discrepancy is unclear, but one possibility could be that the surviving hair cells in the region of the cochlea corresponding to very high frequencies *i.e.* 16-40 kHz may be present, but functioning would be abnormal (27,28) and thus, we hypothesize that this delay in latency values, which is more evident in lower frequencies could be due to abnormal functioning of the hair cells in the higher frequency regions which might hamper further signal conduction along the basilar membrane to the low frequency regions in individuals with occupational noise exposure.

The amplitude of the TB was more than the amplitude of the NB-chirp at all four tested frequencies. This is because the chirp evoked ABRs exhibit a non-monotonic level-dependent behavior, which is caused by the broadening of neural excitation as the level increases (25). At low intensity levels, each frequency corresponds to a narrow frequency region of the basilar membrane and hence, each component adds up in phase (29). At high levels, there will be a broader excitation on the basilar membrane, which results in desynchronization and causes the peak V amplitude to reduce (23). Also, the higher ABR amplitude for TB can be explained in terms of shaping of a pure tone with its additional sidebands due to its low frequency specificity. This effect can become evident in normal hearing subjects, especially at high stimulation levels. Some of the earlier studies, which compared ABR for the TB and the narrow band CE-chirp stimuli at various levels explained that, the narrow band CE chirps have greater amplitude than the TBs as a result of simultaneous depolarization at the specific frequency region of the cochlea accounting for the design of the

narrow band CE-chirps (24,25). However, this pattern is not observed for higher stimulus levels [80 dB nHL], where the TBs may have relatively better amplitude [for 500 Hz], and/or there may not be a significant difference between the stimuli across the frequencies 1000 Hz, 2000 Hz, and 4000 Hz. The observed pattern was explained by the upward spread of the excitation at low levels where, each frequency component excites specific area in the cochlea. But, at higher levels the excitation is present for the broader area around the specific frequency region, which is represented as reduced amplitude (24,25). In line with these studies, there was no significant difference observed in the amplitude of peak V between the TB and the narrow band CE chirp stimuli at 80 dB nHL.

It is evident that although the participants from both groups had good pure tone thresholds, individuals with occupational noise exposure showed prolonged latencies in comparison with the normal population when NB-chirp stimuli was used and no difference in the amplitude parameter for NB-chirp, whereas, there was no significant difference observed for both latency as well as amplitude parameters in TB stimuli. Increased noise exposure results in reduced amplitude of the ANF-generated ABR wave-I (30), which is consistent with the effects of cochlear synaptopathy on ABR wave-I generated in animals. Even though ABR wave-I amplitude offers an objective measure of loss of ANFs in animals, it is difficult to measure robustly across different intensities in humans. However, wave-V of ABR, which is generated in the lateral lemniscus and inferior colliculus (31) is robustly represented in humans and can be recorded even at low levels of stimulation and in the presence of background noise. Unfortunately, ABR wave-V amplitude is not reduced by cochlear synaptopathy (26). This shows that latency would be a better measure than amplitude at supra-threshold (80 dB nHL) level stimulation for the early identification of cochlear synaptopathy. Kujawa and Liberman (2009) (5) studied the effects of noise exposure on mice, and their findings show that even if the thresholds had resumed to normal, with intact cochlear cells, there was some loss of afferent nerve terminals and delayed degeneration of the cochlear nerve. Thus, the significant difference seen can be evidence of the early neuro-physiological changes that are underlying even when the thresholds indicate normal hearing sensitivity. These changes may further increase and gradually lead to a greater effect on the auditory system (5). Hence, even when the thresholds are clinically normal, the prolonged ABR latency results obtained using NB-chirp stimuli at higher intensities helps in determining cochlear synaptopathy. The major limitation of the study was the number of participants in the study because it was limited to 30. To generalize the findings a larger sample size would have been appropriate.

5. Conclusion

The study evaluated changes in both amplitude and latency parameter of wave V of TB and NB-chirp ABR in individuals exposed to occupational noise. There was no change seen between the groups when the comparison was made in terms of amplitude parameters. The significant difference observed for latency parameters at 500Hz, 1000Hz and 2000Hz for NB-chirp ABR, whereas at only 500Hz for TB ABR suggests that NB-chirp ABR is a better clinical tool in identifying damage at a higher level of the auditory system. This difference can be evidence of the early neuro-physiological changes happening at the core level even when the thresholds indicate normal hearing sensitivity.

Acknowledgements

Authors acknowledge the Director, All India Institute of Speech and Hearing, Mysuru and HOD-Audiology, All India Institute of Speech and Hearing, Mysuru for permitting to publish this research work.

References

1. Wheeler D. Physical and physiological variables in noise induced hearing loss. *AMA Arch Otolaryngol.* 1951; 54:267-272.
2. Ranga RK, Yadav S, Yadav A, Yadav N, Ranga SB. Prevalence of occupational noise induced hearing loss in industrial workers. *Indian Journal of Otolaryngology.* 2014; 20:115-118.
3. Müller J, Janssen T. Impact of occupational noise on pure-tone threshold and distortion product otoacoustic emissions after one workday. *Hear Res.* 2008; 246:9-22.
4. Hall AJ, Lutman ME. Methods for early identification of noise-induced hearing loss. *Audiology.* 1999; 38:277-280.
5. Kujawa SG, Liberman MC. Adding insult to injury: Cochlear nerve degeneration after "temporary" noise-induced hearing loss. *J Neurosci.* 2009; 29:14077-14085.
6. Furman AC, Kujawa SG, Liberman MC. Noise-induced cochlear neuropathy is selective for fibers with low spontaneous rates. *J Neurophysiol.* 2013; 110:577-586.
7. Liberman LD, Suzuki J, Liberman MC. Dynamics of cochlear synaptopathy after acoustic overexposure. *J Assoc Res Otolaryngol.* 2015; 16:205-219.
8. Mehraei G, Hickox AE, Bharadwaj HM, Goldberg H, Verhulst S, Liberman MC, Shinn-Cunningham BG. Auditory brainstem response latency in noise as a marker of cochlear synaptopathy. *J Neurosci.* 2016; 36:3755-3764.
9. Dallos P, Harris D, Özdamar Ö, Ryan A. Behavioral, compound action potential, and single unit thresholds: Relationship in normal and abnormal ears. *J Acoust Soc Am.* 1978; 64:151-157.
10. Telian SA, Kileny PR, Niparko JK, Kemink JL, Graham MD. Normal auditory brainstem response in patients with acoustic neuroma. *Laryngoscope.* 1989; 99:10-14.
11. Gorga MP, Kaminski JR, Beauchaine KA, Jesteadt W. Auditory brainstem responses to tone bursts in normally hearing subjects. *J Speech Lang Hear.* 1988; 31:87-97.
12. Elberling C, Don M. A direct approach for the design of chirp stimuli used for the recording of auditory brainstem responses. *J Acoust Soc Am.* 2010; 128:2955-2964.
13. Pushpalatha ZV, Konadath S. Auditory brainstem responses for click and CE-chirp stimuli in individuals with and without occupational noise exposure. *Noise Health.* 2016; 18:260-265.
14. Elberling C, Don M, Cebulla M, Stürzebecher E. Auditory steady-state responses to chirp stimuli based on cochlear traveling wave delay. *J Acoust Soc Am.* 2007; 122:2772-2785.
15. Gorga MP, Johnson TA, Kaminski JR, Beauchaine KL, Garner CA, Neely ST. Using a combination of click- and tone burst-evoked auditory brain stem response measurements to estimate pure-tone thresholds. *Ear Hear.* 2006; 27:60-74.
16. Stapells DR, Oates P. Estimation of the pure-tone audiogram by the auditory brainstem response: A review. *Audiol Neurootol.* 1997; 2:257-280.
17. Yathiraj A, Vijayalakshmi CS. Phonemically Balanced Word List in Kannada: Developed in Department of Audiology. Mysore: AIISH, 2005.
18. Lobarinas E, Salvi R, Ding D. Insensitivity of the audiogram to carboplatin induced inner hair cell loss in chinchillas. *Hear Res.* 2013; 302:113-120.
19. Yamada O, Ashikawa H, Kodera K, Yamane H. Frequency-selective auditory brain-stem response in newborns and infants. *Arch Otolaryngol.* 1983; 109:79-82.
20. Sininger YS, Abdala C, Cone-Wesson B. Auditory threshold sensitivity of the human neonate as measured by the auditory brainstem response. *Hear Res.* 1997; 104:27-38.
21. Ribeiro FM, Carvallo RM. Tone-evoked ABR in full-term and preterm neonates with normal hearing. *Int J Audiol.* 2008; 47:21-29.
22. Rodrigues GR, Ramos N, Lewis DR. Comparing auditory brainstem responses (ABRs) to toneburst and narrow band CE-chirp® in young infants. *Int J Pediatr Otorhinolaryngol.* 2013; 77:1555-1560.
23. Cebulla M, Elberling. Auditory brain stem responses evoked by different chirps based on different delay models. *J Am Acad Audiol.* 2010; 21:452-460.
24. Rodrigues GRI, Lewis DR. Comparison of click and CE-chirp® stimuli on brainstem auditory evoked potential recording. *Revista da Sociedade Brasileira de Fonoaudiologia.* 2012; 17:412-416.
25. Dau T, Wegner O, Mellert V, Kollmeier B. Auditory brainstem responses with optimized chirp signals compensating basilar-membrane dispersion. *J Acoust Soc Am.* 2000; 107:1530-1540.
26. Sergeenko Y, Lall K, Liberman MC, Kujawa SG. Age-related cochlear synaptopathy: An early-onset contributor to auditory functional decline. *J Neurosci.* 2013; 33:13686-13694.
27. Liberman MC, Kiang NY. Acoustic trauma in cats. Cochlear pathology and auditory-nerve activity. *Acta Otolaryngol Suppl.* 1978; 358:1-63.
28. Salvi RJ, Ahroon WA, Perry JW, Gunnarson AD, Henderson D. (1982). Comparison of psychophysical and evoked-potential tuning curves in the chinchilla. *Am J Otolaryngol.* 1982; 3:408-416.
29. Mühler R, Mentzel K, Verhey J. Fast hearing-threshold

- estimation using multiple auditory steady-state responses with narrow-band chirps and adaptive stimulus patterns. *ScientificWorldJournal*. 2012; 2012:192178.
30. Stamper GC, Johnson TA. Auditory function in normal-hearing, noise-exposed human ears. *Ear Hear*. 2015; 36:172-184.
31. Miller AR, Jannetta P. Neural generators of the ABR. In: Jacobson JT, ed. *The auditory brainstem response*. California: College-Hill, 1985:13-31.

(Received June 4, 2019; Revised August 2, 2019; Accepted August 10, 2019)